

# Health and Mortality

## Issues of Global Concern



United Nations



Department of Economic and Social Affairs  
Population Division

in collaboration with

Population and Family Study Centre (CBGS)  
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## PREFACE

This volume presents the collected set of papers presented at the Symposium on Health and Mortality organized by the United Nations Population Division in collaboration with the Population and Family Study Centre (CBGS) of the Government of Flanders, Federal Government of Belgium, that was held in Brussels, Belgium, from 19 to 22 November 1997. The Symposium brought together experts in the study of health and mortality to review the state of knowledge in the field, analyse recent trends and discuss prospects for the improvement of the health status in different regions of the world. The Symposium focused mostly on issues related to the health and mortality of persons aged 15 or over.

The sessions of the Symposium were organized along three major issues: (a) the measurement of mortality and health status; (b) the state of current knowledge about the evolution of mortality and health in developed market-economy countries, countries with economies in transition, and developing countries; and (c) the analysis of risk factors associated with specific behaviours that account or may potentially account for large or growing proportions of deaths. A discussion of the meaning and relevance of the mortality, epidemiological and health transitions set the framework for the discussion of the issues addressed by the Symposium. This collection of papers together with a report of the Symposium's deliberations and conclusions served as background material for the thirty-first session of the United Nations Commission on Population and Development, which was held in February 1998 and focused on health and mortality issues.

The Symposium's deliberations highlighted the truly remarkable progress made during the twentieth century in postponing death and increasing the span of healthy life for all the world's inhabitants. However, they also underscored the large disparities that still exist between the average expected lifespan in developed countries and that typical of the developing world. Although major headway has been made in combatting infectious and parasitic diseases, the latter are still major causes of death in most developing countries. The emergence of new infectious diseases and the re-emergence of others, as the methods used to control them lose effectiveness, are a cause of concern. Furthermore, although considerable advances have been made in reducing mortality due to non-communicable diseases in developed countries, mortality due to cardiovascular diseases and cancer has not declined in countries with economies in transition and, combined with high mortality rates because of accidents or violence, has been at the root of the stagnation or even the decline of life expectancy that some of those countries experienced over the past decade or two. The analysis of selected behavioural factors associated with higher risks of morbidity and mortality suggests that increasing levels of smoking or alcohol intake, for instance, contribute or have the potential to contribute to a considerable proportion of premature mortality. The importance of traffic accidents and violence as causes of health impairment and death is also rising; and the evidence linking certain nutritional patterns to higher incidence of degenerative diseases is growing. The challenge is therefore to devise effective means to influence individual behaviour so as to reduce the prevalence of risk factors contributing to health impairment and early death. These conclusions are based on the wealth of material presented in this volume.

The Population Division and the Population and Family Study Centre (CBGS) wish to thank the following officials for providing the funding that made the Symposium possible: Mr. Luc Van den Brande, Prime Minister of the Government of Flanders; Mr. Erik Derycke, Federal Minister of Foreign Affairs; Mr. Reginald Moreels, State Secretary for Development Cooperation; Mrs. Wivina Demeester, Minister of the Government of Flanders for Finance,



Budget and Health; and Mr. Luc Martens, Minister of the Government of Flanders for Culture, Family and Welfare. In addition, full recognition is due to the experts who prepared invited papers and to other participants at the Symposium who contributed significantly and constructively to the deliberations.

This volume was also published in Belgium by the Population and Family Study Centre (CBGS) in March 1999 as *Health and Mortality: Issues of Global Concern, Proceedings of the symposium on Health and Mortality, Brussels, 19-22 November 1997*.

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## Explanatory notes

The following symbols have been used in the tables throughout this report:

Two dots (..) indicate that data are not available or are not separately reported.

An em dash (—) indicates that the amount is nil or negligible.

A hyphen (-) indicates that the item is not applicable.

A minus sign (-) before a figure indicates a decrease.

A point (.) is used to indicate decimals.

A slash (/) indicates a crop year or financial year, for example, 1994/95.

Use of a hyphen (-) between dates representing years, for example, 1994-1995, signifies the full period involved, including the beginning and end years.

Details and percentages in tables do not necessarily add to totals because of rounding.

Reference to "dollars" (\$) indicates United States dollars, unless otherwise stated.

The term "billion" signifies a thousand million.

The group of least developed countries currently comprises 48 countries: Afghanistan, Angola, Bangladesh, Benin, Bhutan, Burkina Faso, Burundi, Cambodia, Cape Verde, Central African Republic, Chad, Comoros, Democratic Republic of the Congo, Djibouti, Equatorial Guinea, Eritrea, Ethiopia, Gambia, Guinea, Guinea-Bissau, Haiti, Kiribati, Lao People's Democratic Republic, Lesotho, Liberia, Madagascar, Malawi, Maldives, Mali, Mauritania, Mozambique, Myanmar, Nepal, Niger, Rwanda, Samoa, Sao Tome and Principe, Sierra Leone, Solomon Islands, Somalia, Sudan, Togo, Tuvalu, Uganda, United Republic of Tanzania, Vanuatu, Yemen and Zambia.

The following abbreviations have been used:

AD	Alzheimer's disease
AIDS	acquired immunodeficiency syndrome
APC	age-period-cohort
APOE	Apolipoprotein-E
BAC	blood-alcohol concentration
CBGS	Population and Family Study Centre
CCEE	countries of central and eastern Europe
CDC	Centers for Disease Control
CDPO	European Population Committee
CELADE	Latin American Demographic Centre
CHD	coronary heart disease
CPS	Current Population Surveys
CPS-II	second American Cancer Society Cancer Prevention Study
CVD	cardiovascular disease
DALYs	Disability Adjusted Life Years
DHS	Demographic and Health Surveys
DMEs	drug metabolizing enzymes
DPT	diphtheria, pertussis, tetanus
ECOHST	European Centre on Health of Societies in Transition
ELISA	enzyme-based immunosorbent assay
EME	Established Market Economies
EPI	Expanded Programme on Immunisation
ESPO	European Studies of Population
FFS	Fertility and Family Survey
FSE	Former Socialist Economies



FSU	former Soviet Union
GBD	Global Burden of Diseases
GDP	gross domestic product
GDR	German Democratic Republic
GFHP	Gulf Family Health Project
GFHS	Gulf Family Health Survey
GNP	gross national product
GRP	glucose-regulated proteins
HDL	high density lipo-protein
HIV	human immunodeficiency virus
HSP	heat shock proteins
ICD	International Classification of Diseases, Injuries and Causes of Death
IHD	ischaemic heart disease
IMIAL	Research on Infant Mortality in Latin America
INEGI	Instituto Nacional de Estadística e Informática
INSEE	National Institute of Statistics and Economic Studies
IUSSP	International Union for the Scientific Study of Population
LAC	Latin America and the Caribbean
LDL	low-density lipoprotein
LMICs	low and middle-income countries
MEC	Middle Eastern Crescent
MEC	mobile examination center
MOHCW	Ministry of Health and Child Welfare
MPR	mortality profile ratio
MRFIT	Multiple Risk Factor Intervention Trial
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
NIDI	Netherlands Interdisciplinary Demographic Institute
OAI	Other Asia and Islands
OECD	Organization for Economic Co-operation and Development
OPCS	Office of Population Censuses and Surveys
ORT	oral rehydration therapy
PAHO	Pan American Health Organization
PAPCHILD	Pan Arab Project for Child Development
PAPFAM	Pan Arab Project for Family Health
PPA	Population and Policy Acceptance
PPPS	purchasing power parity
RR	relative risk
RTAs	road traffic accidents
SLAITS	State and Local Area Integrated Telephone Survey
SRS	Sample Registration System
SSA	sub-Saharan Africa
STDs	sexually transmitted diseases
TB	tuberculosis
UNAIDS	Joint United Nations Program on HIV/AIDS
UNHSCP	United Nations Household Survey Capability Programme
UNICEF	United Nations Children's Fund
US DHHS	United States of America, Department of Health and Human Services
WFS	World Fertility Survey
WHO	World Health Organization
WPPA	World Population Plan of Action
WRN	Warner's Syndrome gene
ZAGS	Zapis' aktov grazhdanskogo sostoiianiia [Registry of Acts of Civil Status]



**Statement by Mr. Joseph Chamie, Director  
Population Division, United Nations  
to the Symposium on Health and Mortality  
Brussels, Belgium  
19-22 November 1997**

Representatives of the Government,  
Distinguished Experts and Guests,  
Ladies and Gentlemen,

I would like to welcome all of you to this Symposium on Health and Mortality. And on behalf of the United Nations, I wish to express our sincere appreciation and thanks to both the Federal Government of Belgium, the Government of Flanders, and especially the Population and Family Study Centre (CBGS) for their collaboration, cooperation and support for this Symposium.

In addition to this Symposium, we in the United Nations Population Division have had the fortunate opportunity to work closely with Professor Robert Cliquet and the capable staff of CBGS on other occasions. I also wish to emphasize that the Population Division looks forward to continued collaboration with Belgium, and in particular the Population and Family Study Centre, in the coming years.

Personally, it is a pleasure for me to address you this morning. This Symposium had a long gestation period and I am pleased that its now being delivered. I am also pleased to see many familiar faces among the participants. In addition, I am looking forward to getting to know those of you whom I have not met previously.

The purpose of my brief opening remarks this morning is neither to present detailed demographic statistics nor to summarize the important results of the many excellent papers that have been prepared for this Symposium. I leave these tasks to you, the experts on health and mortality. In addition to wishing to extend to you a warm welcome, the purpose of my introductory comments is to bring to your attention several points that may be useful to keep in mind during this Symposium.

First, it is important for you to know that the impact of this Symposium goes well beyond the next four days. As usual, the papers presented at this Symposium will be revised, edited and published. In addition, however, the results of this meeting will be made available in a report to the United Nations Commission on Population and Development, which will meet in February of next year. The theme of the Commission for that session is the same as this Symposium, namely, health and mortality. Therefore, your deliberations and conclusions are highly relevant.

Second, during this decade the international community has convened a variety of intergovernmental conferences and summits. Although the themes of these conferences differed, a central concern for all of them has been development. And while the term "development" has varying definitions and a multitude of interpretations, few would disagree with the statement that good health and long life are at the heart of development. This Symposium is therefore not an academic meeting discussing a marginal topic. The theme of this meeting is a fundamental and integral component of national, regional and international development efforts.

Third, it is important to acknowledge that considerable progress has been achieved in the area of health and mortality. In the twentieth century, for example, mortality rates have declined in virtually every corner of the world. By and large, people are healthier and living longer than ever before.

These transitions in mortality and health are certainly good news and need to be widely recognized, especially by policy makers and those in charge of government resources. However, the other vital part of this message is that more can and must be done.

Moreover, we need to emphasize that improvements in health and mortality are not guaranteed. The health and mortality transitions are not "one-way" paths. As has been reported in various parts of the world, faltering economic, social and political conditions have lead to worsening health and mortality levels, and in some cases rather significant setbacks. This Symposium can be useful in highlighting those activities and policies that need to be done to maintain and to improve the health and mortality conditions for every segment of society.

My fourth and final point this morning is this. The world is a highly diverse place. There are different languages, religions, economies, political systems, customs and life styles among the world's 5.8 billion people. These differences attract the public's attention, and are evident in newspapers, radio, television and other forms of the popular media. Despite all

these differences, however, one thing is common to all peoples and cultures, common to the rich and the poor, to conservative and liberal, and to men and women. And that is health and mortality.

Long life and good health are universal goals and the concerns of all societies. This Symposium is an attempt to address these concerns, to further our knowledge base and, hopefully, to contribute to better health and longer lives of people.

In closing, I would like to say that I am looking forward to the next four days. I am confident that the experts and participants gathered here will be able to combine theoretical insight, sound analysis and practical experience, yielding a successful Symposium.

Thank you.

**Statement by Professor Robert Cliquet**  
**General Director of the**  
**Population and Family Study Centre (CBGS)**  
**to the Symposium on Health and Mortality**  
**Brussels, Belgium**  
**19-22 November 1997**

Dear Colleagues,

It is a great honour and pleasure for my Staff and for me personally to welcome the Population Division, the invited speakers and discussants, and the observers at this Technical Symposium organised by the United Nations Population Division in co-operation with the Population and Family Study Centre (CBGS).

First of all I must apologize on behalf of the Prime Minister of the Government of Flanders as well as the Flemish Minister for Culture, Family and Welfare who is in charge of the Population and Family Study Centre. Being committed to other urgent obligations in this politically busy period of the year, both the Prime Minister of the Government of Flanders and the Flemish Minister for Culture, Family and Welfare asked me to transmit to all of you their best wishes for a fruitful and successful meeting.

The scientific, societal and policy relevance of this Technical Symposium, as a preparation for the forthcoming thirty-first Session of the United Nations' Commission on Population and Development, is fully appreciated by the authorities in this country. As you have seen from the invitation and programme, this Technical Symposium not only enjoys the auspices of several ministers, both of the Federal Government of Belgium and the Government of Flanders, but is made possible by the financial support of several federal and Flemish departments. From the federal government, the Technical Symposium has the support of the Minister of Foreign Affairs, Mr. Erik de Rycke, and the State Secretary for Development Aid, Mr. Reginald Moreels. From the Government of Flanders, in addition to the Prime Minister, Mr. Luc Van den Brande, and the Minister for

Culture, Family and Welfare, Mr. Luc Martens, whom I mentioned already, we have also the support of the Minister for Finance, the Budget and Health, Mrs. Wivina Demeester.

At this opening session, I am, moreover, happy to see and welcome several high-ranking officials, among others from the federal General Administration for Development Aid, from the Flemish Foreign Relations Administration, and last but not least, from the Flemish Administration for the Family and Public Welfare to whom the CBGS is administratively affiliated.

Please first allow me to say a few words about your host, the Population and Family Study Centre, which is the English translation of the Dutch-language name of the institute: *Centrum voor Bevolkings- en Gezinsstudie*, abbreviated as CBGS. This is a Flemish scientific institute, belonging to the services of the Government of Flanders. Formerly, the CBGS was part of the national, bilingual Population and Family Study Centre which was created in 1962 with a view of undertaking policy relevant population and family research. With the federal state reform of the 1980s, the two language branches of the former national institute became autonomous institutes, henceforth working under the authority respectively of the Dutch-speaking and French-speaking community governments in Belgium. Whereas - unfortunately - the French-language institute "Centre d'Etudes de la Population et de la Famille" disappeared, the institutional status of the Dutch-language centre was not only consolidated, but even strengthened. In 1991, the CBGS staff was substantially enlarged and the institute reorganised in four scientific sections. The CBGS has now a permanent staff of 17 researchers and 9 administrative officers.



The CBGS has a relatively broad and ambitious research programme; you will find a brief presentation in the yearly "CBGS Progress Reports". The CBGS presents the results of its investigations in several Dutch-language publication series, - a scientific journal, a series of monographs, and a series of working papers. With a view of disseminating its work also at the international level, the CBGS publishes, in co-operation with its Dutch sister institute, the NIDI, an English language series, the NIDI-CBGS publications. Recently, an offshoot was established as the "European Studies of Population" (ESPO), edited by several population institutes and organisations in Europe. Copies of all of these publication series are on display in this room.

For many years, one of the CBGS policy goals has been to contribute in a modest, but systematic way to international and intergovernmental activities. This is done in two ways. Firstly, by encouraging the active participation of CBGS members in international bodies such as the European Population Committee (CDPO) of the Council of Europe, and the Commission on Population and Development of the United Nations, or in United Nations co-ordinated initiatives such as the World Fertility Survey (WFS), the Fertility and Family Survey (FFS) and the Population Policy Acceptance (PPA) projects. Secondly, the Centre organises workshops or other meetings with an international or intergovernmental character. Since the CBGS is a small institute, our internationally oriented activities concentrate on the organisation or hosting of small-scale activities which, however, may have a multiplier effect.

The CBGS is particularly eager to provide, in the fields of population and family problems, assistance to developing countries. A number of years ago, it contributed substantially to the Flemish Interuniversity Programme in Demography which was largely oriented towards developing countries. In the future, it hopes, as an implementation of one of the recommendations of the Cairo Programme of Action, to

make available its staff with a view to systematically providing advice and services to population researchers and policy makers in developing regions. With this in mind, I recruited some years ago an eminent expert, well known to you, who is now one of the scientific directors within the Centre.

The present Technical Symposium is a typical example of what the CBGS wants to host and support: it is a small sized meeting of highly qualified experts - I already have the opportunity to read the papers! - whose work will form an essential input to the thirty-first Session of the United Nations Commission on Population and Development.

The fact that an intergovernmental body such as the United Nations Commission on Population and Development wants to devote one of its sessions to "health and mortality" means that the international community perceives these issues as highly relevant. Moreover, "Health and Mortality" are issues that already occupy a major place in population related policy documents such as the World Population Plan of Action (WPPA) and more particularly the Cairo Programme of Action. The latter dwells at great length on reproductive health.

In this Technical Symposium we shall study the mortality, epidemiological and health transitions, their mutual relations, and their demographic, societal and policy implications, not only for the present but also for the future generations. Special attention will, obviously, go to those regions in the world or socioeconomic groups which lag behind in the transition processes or have experienced reverse trends.

In this introductory statement I can only express the hope that our discussions will lead to a report that will be of such a nature and quality that it will fuel the debate and the decision making process in the Commission on Population and Development and other United Nations bodies.

## REPORT OF THE SYMPOSIUM ON HEALTH AND MORTALITY

The United Nations Population Division in collaboration with the Population and Family Study Centre (CBGS), Flemish Scientific Institute, and with the financial support of the Government of Flanders and the Federal Government of Belgium organized a Symposium on Health and Mortality that took place in Brussels, Belgium, from 19 to 22 November 1997. The Symposium was attended by invited experts; representatives of the United Nations Children's Fund, the United Nations Population Fund and the World Health Organization; representatives of Flanders and the Federal Government of Belgium; and scholars from Belgium and other countries as well as government officials from other countries participating as observers in their personal capacity.

Robert Cliquet, General Director of CBGS and Joseph Chamie, Director of the United Nations Population Division made opening statements emphasizing the importance of the Symposium in the substantive preparations for the thirty-first session of the United Nations Commission on Population and Development which was to be held in February 1998 and would be devoted to a discussion of issues of health and mortality. The role played by CBGS in ensuring that the inter-governmental bodies in which it participated had at their disposal the best and most up-to-date substantive and technical information on which to base their deliberations was underscored. Thus, the conclusions reached by the Symposium would be transmitted to the Commission and were expected to be highly relevant in guiding the discussion of policies and programmes directed to improve the health and lengthen the life span of the peoples of the world, efforts that were considered to be a fundamental and integral part of development programmes.

The participants of the Symposium proceeded to elect its bureau, which consisted of Robert Cliquet as Chairperson, Juan Chackiel, Samir Farid and Gigi Santow as Vice-chairpersons, and Shiro Horiuchi as Rapporteur.

The Symposium was to focus attention on issues related to the health and mortality of persons aged 15 or over. Indeed, as it was noted several times during the deliberations, significant advances had been made in reducing mortality in childhood, even in countries where the mortality of adults had declined only moderately or not at all. Furthermore, both in terms of measurement issues and in terms of its determinants, more was known about mortality in childhood than about mortality at older ages. For that reason, it was appropriate to concentrate on the latter.

The sessions of the Symposium were organized along three major issues: (a) the measurement of mortality and health status; (b) the state of current knowledge about the evolution of mortality and health in developed market-economy countries, countries with economies in transition, and developing countries; and (c) the analysis of risk factors associated with specific behaviours that account or may potentially account for large or growing proportions of deaths. During the Symposium, each session consisted of the presentation of solicited papers and a general discussion on the issues they raised. The following sections summarize the presentations and discussion made during each session. They reflect the order actually followed during the Symposium.<sup>1</sup> The first session, which focused on the meaning and relevance of the mortality, epidemiological and health transitions, set the framework used to address the major issues considered by the Symposium.

### A. THE MORTALITY, EPIDEMIOLOGICAL AND HEALTH TRANSITIONS

In discussing the three transitions, Gigi Santow began by underscoring that the global mortality decline that had occurred during the twentieth century had been a striking achievement. The almost universal decline in death rates that had been registered by the 1960s led demographers to coin the term "mortality tran-

sition" to refer to the passage from high mortality, associated in large part with the high prevalence of infectious and parasitic diseases, to lower mortality, stemming from the control of such communicable diseases. Since the distribution of deaths changed in terms of both causation and age structure as the expectation of life at birth moved from about 40 to about 60 years, the mortality transition was said to have been accompanied by an "epidemiological transition", a term that focused attention on the changing relevance of different causes of death and, especially at the early stages of the transition from high to low mortality, put particular emphasis on the declining role of communicable diseases and on the growing share of mortality due to non-communicable diseases, primarily cardiovascular disease and cancer. In recent years, analysis of the experience of different groups of countries from the perspective of the epidemiological transition revealed that still further declines in mortality had been possible because of a reduction of mortality caused by cardiovascular disease and, in particular, ischaemic heart disease. Partly in recognition of the importance of behavioural changes in achieving reductions of mortality due to non-communicable diseases, the term "health transition" was coined to draw attention to the social and behavioural factors underpinning the mortality and epidemiological transitions. Santow noted that such a term was sometimes used as a synonym of the "mortality transition" but that it differed from the "epidemiological transition" in laying greater emphasis on the social, cultural and behavioural factors affecting health rather than on medical interventions.

Analysing the experience of different groups of countries from the perspective of the different transitions, Santow remarked that the developed market-economy countries were the first to undergo the mortality and epidemiological transitions and that, although there was considerable evidence about the paths that those countries had followed to reach the very low mortality levels that characterized them today, there was still some debate about the factors responsible for the first phase of their mortality decline which began long before the discovery of antibiotics and the introduction of other effective medical interventions. In contrast, in the case of developing countries it was generally agreed that it was precisely the availability

of medical tools to combat or prevent infectious and parasitic diseases and of insecticides to reduce their spread that was largely responsible for the very rapid declines of mortality that most developing countries had experienced by 1980. Similarly, the countries with economies in transition had been the beneficiaries of medical and other public health interventions that had reduced mortality due to communicable diseases to very low levels. However, the countries with economies in transition had not yet managed to reduce mortality due to non-communicable diseases and, in some cases, even increases in mortality due to such causes had been recorded. The experience of these countries indicated that there was no guarantee that, once started, the mortality and epidemiological transitions would necessarily lead to ever decreasing mortality levels. Reversals were possible and likely, especially in contexts where key factors underpinning the health transition were overlooked, including social and individual behaviours associated with increased risk of morbidity or death.

According to Santow, the three transitions provided a general framework to establish priorities for future policy-relevant research. They implied, for instance, that useful lessons could be learned from past experience, by drawing parallels between the experience of today's developed countries at early stages of the mortality transition and the state of developing countries today or, alternatively, by documenting contrasting experiences. Consideration of the three transitions also implied that more emphasis had to be put on the analysis of health status rather than on the terminal outcome, death, and that key research issues were the study of risk factors, the analysis of differentials among population subgroups, and the reasons for the continued existence of inequality in health status. Santow remarked that considerable effort had been devoted to the study of mortality at the extremes of the age range—in childhood and at advanced ages—where the risks of dying had been or were still high. Yet a focus on health status rather than death demanded that more attention be paid to the middle of the age range, especially between ages 15 and 60.

The discussion expanded on Santow's description of the transitions and their utility in

guiding research and policy action. It was noted that the mortality transition helped describe what had happened, the epidemiological transition focused on the proximate determinants of the changes observed, and the health transition called attention to the less proximate determinants of change, including social and behavioural factors. There was a need, however, to consider yet other factors underlying and shaping the mortality transition, such as the effects of nutrition, or of the increasing exposure to pollution, particularly of air and water, and also the genetic make-up of populations. By expanding the horizon of interest, the health transition not only highlighted the importance of considering morbidity, disability and health status in their own right but it also indicated that societal conditions played a key role in determining the well-being of individuals. Thus, the high mortality levels over the middle age range that had prevailed in countries with economies in transition since the 1960s were symptomatic of a dysfunctional social system that fostered self-destructive behaviours and risk-taking among large segments of the population. Another cause of concern was the persistence of inequality in developed market-economy countries, where significant population subgroups continued to experience relatively high morbidity and mortality levels. Several participants suggested that a more thorough analysis of the causes of persistent inequalities within countries and even between countries was likely to shed light on the mechanisms leading to both improvements in health status and reductions in mortality. Although it was known that the causes of mortality decline were multiple and varied, a better understanding of how the different factors contributed to such a decline and of their relative importance was still needed, especially in contexts where the risk factors themselves were changing as society was transformed.

It was noted that such a comprehensive approach to the study of health and mortality was in agreement with the WHO definition of health, which emphasized three different dimensions of well-being: the physical, mental and social. Several participants noted that, although it had been argued that extending life tended to result in longer periods of illness or disability, recent studies by Manton's group as well as those by Crimmins revealed that the health

status of the elderly had actually improved significantly during the 1980s. There was also evidence to the effect that persons dying in their 60s or 70s, whose deaths were generally caused by non-communicable diseases such as cardiovascular disease or cancer, tended to incur higher medical costs at the end of life than those dying at a later age, whose terminal illnesses tended to be of shorter duration. The existence of such differences underscored the need to take into account the heterogeneity of populations in studying either the determinants or the consequences of ill health. Furthermore, the effects of biological, demographic, social or economic selectivity needed to be disentangled from those of socio-economic inequality, and it was important to make explicit the causal relations or pathways through which social or economic factors affected health so that health interventions could be designed to cope with those factors and the health sector was not marginalized or left impotent to make a difference.

Some participants underscored the fact that the continuous decline of mortality or the success registered so far in combatting an ever wider range of diseases was not irreversible. The emergence of new infectious diseases, such as HIV infection, or the growing virulence of well-known ones, such as tuberculosis or malaria, did not leave much room for complacency. The increasing levels of trade and the growing spatial mobility of people contributed to accelerate and expand the geographical diffusion of communicable diseases. Furthermore, the prospect of continued ecological change, as man's habitat was extended to previously uninhabited areas, such as tropical forests, or the destruction of ecological systems because of human activity could potentially change the epidemiology of infectious disease or bring human populations in contact with rare or unknown pathogens. Concern was also expressed about the increasing levels of interpersonal violence within societies, as outright conflicts multiplied and social disparities increased the potential for chronic strife.

In considering the role of the individual in the pursuit of better health, the issue of individual choice versus societal responsibility was debated. One participant noted that in today's developed market economies, although infant mortality had reached very low levels, the issue

of the impact that parents' decisions could have on the health of their children needed to be addressed. Thus, children whose parents smoked were exposed to passive smoking that could potentially damage their health. Postponing reproduction to older ages increased the probability of conceiving a child with Down syndrome and, although a number of birth defects could be detected *in utero*, parents did not always opt for abortion when no treatment was possible. Choice was also an issue in the case of individuals adopting behaviours or practices that endangered their own health or that of others. Smoking and alcohol abuse, for instance, had proven negative effects on health. Yet most countries had minimal constraints on the accessibility of adults to those products and many did not enforce strictly whatever provisions they had to prevent under-age persons from having access to them. Participants agreed that society had the responsibility of protecting children and adolescents from making choices that would endanger their health. Furthermore, Governments had the obligation of providing their citizens with the information needed to make informed choices. In many instances, the inaction of Government constrained the choices that individuals had, as when non-smokers had few means of avoiding exposure to passive smoking because regulations to protect them did not exist. Yet, it was recognized that even when Governments took measures to modify undesirable and risky behaviours, achieving that goal was by no means straightforward.

#### B. THE MEASUREMENT OF MORTALITY AND CAUSES OF DEATH

Kenneth Hill reviewed several aspects of the measurement of mortality in developing countries. The main sources of data allowing the estimation of mortality over age 15 included: (a) the distribution of deaths by age and sex obtained from a civil registration system together with the distribution of the total population classified by age and sex, usually obtained from a census; (b) similar information on deaths and the population exposed to the risk of dying obtained from a sample registration system; (c) the availability of complete enumerations of a population from successive censuses (which permitted the derivation of probabilities of survival during the intercensal period); and (d)

the availability of data on the survivorship of kin (mothers, fathers, siblings) or on deaths over a particular period obtained from censuses or from moderate to large-scale household surveys.

Because information on deaths by age derived from a civil registration system had the greatest potential of permitting an in-depth analysis of mortality trends and characteristics, it was important to assess the extent to which such data were available. Using information compiled by the United Nations Statistics Division on the countries reporting that their civil registration system attained at least a 90 per cent completeness in the coverage of deaths, Hill concluded that the availability of such information in terms of number of countries had shown only a modest improvement from the early 1970s to the early 1990s (the proportion of countries with complete reporting of deaths increased from 28 to 35 per cent). However, when countries were weighted by their population size, the improvement disappeared: in terms of total population covered by an adequate system of death registration, the percentage declined from 17 to 16 per cent over the two decades considered. There were also significant differences between the developing regions. Thus, whereas the population covered by adequate death registration amounted to 81 per cent in Oceania, 60 per cent in the Caribbean, 49 per cent in Northern Africa and 41 per cent in Latin America, it was less than 5 per cent in Asia and virtually nil in sub-Saharan Africa.

A second way of assessing the availability of information allowing the estimation of mortality over age 15 was to resort to the information published by the United Nations Population Division regarding the type of method used to derive estimates of adult mortality in preparing projections for all countries of the world. The information available referred to a period preceding 1994 and indicated that registered deaths, which included those obtained via the sample registration system operating in India, served as the basis for the estimation of adult mortality for about 48 per cent of the developing world's population. The second most important source of information was data on deaths over a specific reference period obtained from censuses or surveys, which covered 32 per cent of the developing world's population and included China, whose 1991 census gathered

the required information. Among the remaining 20 per cent of the population in developing countries, estimates derived via models from information pertaining exclusively to mortality in childhood were used in most cases (for 13 per cent of the population) and data on adjusted registered deaths were used for another 3 per cent. That is, for 83 per cent of the population of the developing world there was some information about the distribution of deaths by age and their mortality level. However, the proportion covered by region varied considerably and was particularly low in Africa, where it reached only 11 per cent.

Given that over a third of all countries in the developing world lacked adequate data allowing the estimation of adult mortality, it was important to suggest strategies for the improvement of that situation. Since the establishment of a fully functional and reliable system of death registration was judged beyond the means of most of those countries, attention was focused instead on the collection of data allowing the indirect estimation of adult mortality. Information on the number of deaths occurring in a household over a 12 month period together with the age at death and sex of the deceased could be obtained via censuses or large-scale household surveys. Estimates derived from such information would be more likely to be reliable for populations with moderate to high average levels of educational attainment and for which the dating of events was meaningful. Because the death of an adult was a rare event, large sample sizes were required to obtain reliable estimates of mortality by age and sex.

In countries where the reporting of deaths occurring over a specific reference period was not expected to be sufficiently reliable, it was suggested that gathering information on the survival of close relatives was the next option. The relatively new approach of obtaining information on the survival of siblings had potential advantages because it provided a more recent estimate of mortality than methods based on the survivorship of parents. However, the collection of the data required to estimate mortality from sibling survival was more demanding than that involved in gathering information on the survival of parents. Furthermore, to obtain recent estimates of mortality based on information on the survival of mothers or fathers, that

information should be complemented by a question on whether the dead parent had survived to some key event in the life of the son or daughter to which the information referred, such as that person's marriage or the birth of that person's first child.

Eduardo Arriaga addressed the issue of measuring causes of death in developing countries. He remarked that few developing countries gathered and published data on deaths classified by cause and those data often suffered from lack of completeness and errors in the reporting of cause of death. An important indicator of the quality of data on cause of death was the proportion of deaths attributed to ill-defined or unknown causes. Usually, the proportion of deaths with cause unknown was higher at very young ages and among persons belonging to lower socio-economic groups. Furthermore, in countries where persons with certain illnesses were likely to be stigmatized, there was reluctance on the part of both physicians and relatives to declare the true cause of death. Despite these problems, information on the distribution of deaths by cause had proven useful in assessing trends and for the detection of changing patterns, especially in regard to deaths attributable to "external causes". Thus, the available data on deaths classified by cause indicated that rates of suicide were particularly high among Chinese women and that in Colombia in 1986 violent deaths among males aged 15-44 were about nine times higher than those among females in the same age group.

Arriaga noted that in countries where the proportion of deaths with ill-defined or unknown causes was high, there was often no basis on which to adjust the observed distribution of deaths by cause, since it was unlikely that the deaths included in the ill-defined and unknown categories were randomly assigned to it. The possibility of selectivity meant that proportional redistribution would result in biases. Although other methods of redistribution were available, there was usually no basis on which to judge whether the resulting distribution was closer to the true one than the unadjusted one.

An even more serious problem arose when a country lacked information on cause of death altogether. Arriaga suggested that current knowledge about the typical distribution of



deaths by cause characterizing the different stages of the epidemiological transition could be used to impute a distribution by cause to the estimated deaths of developing countries on the basis of their level of mortality (which was an indicator of the stage reached in the epidemiological transition). Such imputation was justified by the empirical finding that, among deaths under age 10 and those over age 45, the distribution by cause was fairly similar among countries having similar levels of mortality over those age ranges. Multivariate analysis could be used to derive model patterns and make the necessary imputations. However, there was greater variability in the distribution of deaths by cause over the middle age range, especially because there was not a close correspondence between level of mortality at those ages and the relative weight of mortality due to external causes and to causes related to pregnancy and delivery. To address that problem, Arriaga suggested that censuses or surveys collecting information on deaths over a specific period should also include a question designed to determine whether the death was caused by external causes. In particular, Arriaga proposed that the respondent be asked whether a reported death occurred as the result of an accident, homicide or suicide. If the deceased person was a woman, a further question on whether her death occurred because of pregnancy or delivery could be added.

Arriaga reviewed briefly the use of surveys and "verbal autopsy" methods to establish the cause of death of persons deceased within a household. He concluded that although such methods had proven useful in investigating the incidence of specific causes of death among small groups of children and women of reproductive age, it was unlikely that similar methods could be extended to gather information on a wider range of causes of death for a whole population. Consequently, barring the substantial improvement of systems of death registration in developing countries, the gathering of information such as that suggested above that would allow the indirect estimation of the distribution of deaths by cause seemed the most cost-efficient approach over the near future.

The presentation by Rosario Cárdenas focused on the changing causes of death in Mexico, as measured by the death registration sys-

tem of that country. The coverage of that system had generally been high and by the 1990s about 95 per cent of all deaths were medically certified. However, the data were not considered to be free from error and misreporting of age at death was still considered to be a problem. Comparison of the standardized mortality rates estimated for 1979 and 1996 showed that mortality levels had declined substantially, with the reduction being larger for males than for females, so that the sex differentials diminished. Three mutually exclusive groups of causes of death were considered: (a) communicable diseases plus maternal and perinatal causes of death; (b) non-communicable diseases; and (c) injuries. In accordance with the epidemiological transition, a large proportion of the gains in life expectancy registered between 1979 and 1996 was attributable to the reduction of mortality from infectious and parasitic diseases among both sexes. In addition, mortality due to injuries declined significantly among men. Already by 1979, non-communicable diseases accounted for most of the mortality among men and women in Mexico and, among men, injuries accounted for a larger share of mortality than communicable diseases. By 1995 the estimated number of years lost because of non-communicable diseases were 9.8 for men and 8.4 for women. In addition men lost over 4 years because of deaths caused by injuries and women lost about half that amount.

Analysis of specific causes of death in Mexico indicated that mortality due to diabetes was rising but that there was lower mortality caused by peptic ulcer and stomach cancer. Among men, mortality due to lung and prostate cancer was rising, whereas among women, mortality from cervical cancer and, to a lesser extent, from breast cancer was increasing. An analysis of mortality at the state level indicated the existence of large differentials in survivorship, but a trend towards convergence was noticeable.

Despite the severe economic recession that Mexico experienced during most of the 1980s, mortality had continued to decline. Several factors might have contributed to maintain such decline, especially the expansion of interventions aimed at improving the health status of children. However, much remained to be done to understand fully the processes leading to the reduction of mortality among adults. Policies

aimed at combatting the known causes of non-communicable diseases had yet to be put in place, especially regarding the prevention of automobile accidents, and the reduction of smoking and alcohol abuse.

In the discussion that followed it was noted that different types of data or estimates of mortality were needed to fulfill different needs. Thus, a general level of mortality was needed to estimate population growth rates. Complete life tables were necessary to project a population but more detailed information was needed to address issues of equity, quality of life or to track emerging causes of death. It was suggested that different sources of data be combined to obtain a more comprehensive view and that non-traditional sources, such as hospital records or reports by physicians, be used to obtain information on morbidity and causes of death. It was pointed out that the deficiencies or shortcomings of one source of data could sometimes be palliated or compensated by information obtained from other sources. Thus, although the estimates derived from reports of deaths occurring over a specified period might not be perfect, they could provide a basis for comparison with estimates obtained from the survivorship of close relatives in countries where other sources of data were deficient or non-existent.

The strengths and limitations of traditional sources of data for demographic estimation were reviewed in terms of the potential of the different sources to yield data classified by age, sex, socio-economic status, region, and reference period (recent vs. further in the past). The problem of deciding which source of data to promote in countries where resources were scarce and registration systems non-existent was judged to depend on a consideration of cost vs. accuracy. From that perspective surveys could be considerably less costly than censuses but their effectiveness in producing reliable estimates of mortality depended on ensuring that they were truly representative of the population under study and that adequate sample sizes were used. It was noted that, to estimate with a 5 per cent error a mortality rate over ages 15 to 60 that was expected to be in the range of 3 to 8 deaths per 1,000, a sample of 103,000 households was needed. Although the sample size could be considerably smaller if the method

based on the survivorship of siblings was used (provided total fertility had been moderate to high in the past), the numbers involved were still four or five times larger than the typical sample size of the demographic and health surveys normally undertaken (about 4,000 respondents).

Several participants expressed concern about the quality of the data available. Age misreporting was recognized as a major problem affecting the reliability of estimated age-specific mortality rates. The problem was especially serious in cases where the patterns of misreporting differed between the source of deaths by age and that of population by age. Age misreporting was expected to be particularly acute among populations characterized by low levels of educational attainment for whom dates had little meaning. Although it was suggested that the use of historical calendars could improve the quality of information on age and on dates of events in such populations, studies on the use of such devices were said to have shown that their use improved only the smoothness but not the quality of age reporting.

Regarding information on cause of death, it was noted that even in developed countries, where most deaths were medically certified, that information was not totally reliable. Furthermore, although a high percentage of deaths reported as having ill-defined or unknown causes signaled that the information was probably deficient, it was not necessarily the case that information on cause of death was better when the proportion of deaths in such categories was low. In some of the successor States of the former USSR, for instance, causes were assigned to most deaths, but erroneous reporting of such causes was high.

With respect to developing countries lacking vital registration systems, several participants cast doubts about the possibility of obtaining useful information on causes of death from a few questions added to censuses or large-scale surveys. Thus, it was thought that respondents would be reluctant to report that the death of a household member had resulted from violence. Distinguishing deaths due to suicide from those due to homicide might also prove to be problematic in practice. It was further argued that even the usefulness of information obtained

from "verbal autopsies" was generally limited because different diseases could present similar symptoms and the respondent might not recall or might never have been aware of the full array of symptoms experienced by the deceased. Yet, although epidemiological research required detailed and reliable information on causes of death, for the purpose of setting priorities for public health interventions in contexts where other information was lacking, the rough data yielded by verbal autopsies could be helpful. In addition, given that high mortality countries were also the most likely to lack information on both mortality levels and causes of death, it was important to use any suitable opportunity to try to fill such information gap. The preparations for the 2000 round of censuses presented such opportunity and, although the tendency was to reduce the number of questions included in censuses, it was noted that the recommendations for the 2000 round of censuses about to be issued by the United Nations included a question on deaths in the household over the 12 months preceding enumeration. The prospects regarding the introduction of other questions in the upcoming round of censuses were judged to be slim, but in order to enhance them it was suggested that efforts be made to test the proposed questions in pilot surveys or pilot censuses, since evidence validating their adequate performance in the field was a pre-requisite for their inclusion in censuses or large-scale surveys.

Some participants suggested that another way of improving data availability would be through the use of surveillance systems in multiple sites so that certain representativeness could be achieved. The Sample Registration System of India was provided as an example of such surveillance systems, but it was noted that its operation was costly and that, in order to ensure the representativeness of the system, the areas covered had to be changed periodically. In the case of India, the Sample Registration System had last been "re-based" using the results of the 1991 Census. Other surveillance systems, particularly those used to gauge the effects of particular health interventions, had soon lost their representativeness and could no longer be used to produce data reflecting national conditions.

There was some discussion about the problems faced in collecting the data needed for the application of the estimation method based on the survivorship of siblings. It was noted that when such questions were included in a household questionnaire where only the head of household provided information about household members, requesting information about siblings from every person listed led to needless repetition and did not elicit the cooperation of the respondent. There were also problems about the performance of the method, since in a number of countries it had produced estimates of mortality that suggested increasing rather than decreasing trends over time, but the general conclusion was that this method was sufficiently promising as to deserve further use and testing. To make such testing possible, it was recommended that information allowing the application of multiple indirect methods of mortality estimation be collected.

### C. THE MEASUREMENT OF HEALTH STATUS

Samir Farid spoke about the use of surveys to gather information on health status in developing countries. He noted that health indicators were necessary, among other things, to assess performance in the attainment of goals set by the Health for All Programme of the World Health Organization (WHO). However, many of the countries which had to make significant strides in improving the health status of their populations were precisely those lacking adequate sources of information on health. To fill that gap, some countries were using surveys to measure a variety of health indicators. There were, however, no established guidelines for the design and implementation of health surveys in developing countries and practices could vary widely from one survey programme to another. If cross-country comparability was to be attained and if comparability was to be maintained over time within countries it was important to establish internationally validated guidelines for the design and implementation of such surveys as well as standard concepts and procedures for the measurement of health status using survey data. Ideally, an international effort in that regard would produce model questionnaires containing modules addressing

specific priority areas, such as the prevalence of morbidity or long-term disability or the utilization of health services. In addition, guidelines were needed about sampling design, the training of interviewers, the adaptation of questionnaires to specific contexts, and the effects that seasonality might have on survey results.

Farid noted that over the past ten years three survey programmes had conducted comparable surveys in countries of Northern Africa and Western Asia: the Gulf Child Health Survey Programme (1987-1989); the Pan Arab Project for Child Development (PAPCHILD, 1989-1996); and the Gulf Family Health Survey (GFHS, started in 1995). A more ambitious health survey programme was being developed under the title Pan Arab Project for Family Health (PAPFAM). The first two had focused mostly on children, but the GFHS had gathered information on the health status of persons of all ages. It included four questionnaires recording: (a) the health status of household members; (b) the socio-economic status and environmental conditions of the household; (c) the reproductive health of all women aged 15 to 49; and (d) the health status of all children under age 5. Model questionnaires had been developed for each of these purposes and the experience gained in their use would be helpful in improving their design and effectiveness.

The first questionnaire, focusing on health status, was meant to be answered by the head of household who provided information about the health of each household member. The questionnaire included a module on chronic conditions, including high blood pressure, heart disease, asthma, diabetes, ulcers, kidney disease, liver disease, stroke, epilepsy and mental problems. For each individual who was reported as having one of those conditions, it was ascertained whether a medical doctor had diagnosed the condition, the age at diagnosis, and the current use of medication. Another part of the questionnaire recorded current morbidity by inquiring about perceived health status, and illness or injury during the two weeks preceding interview. Those reporting illness or injury were asked about the utilization of health services, the use of medication and the duration of the illness. The health status questionnaire also included questions on the survivorship of close relatives to permit the derivation of indirect

estimates of mortality levels. The information recorded permitted the calculation of a wide array of health indicators, some of which were presented for the Gulf countries. Farid also presented in some detail the information on reproductive health for Gulf countries obtained from the third questionnaire used in the GFHS.

Jennifer Madans provided an overview of the official sources of information on health status and morbidity in the United States. Although the National Center for Health Statistics (NCHS) was described as the principal and official agency producing, analysing and disseminating health statistics in the United States, health information was also collected by other departments of the Federal Government and by the research community. The main role of NCHS was to provide consistent time series data that fulfilled multiple purposes and could serve as basis for both demographic and epidemiological applications. A key use of NCHS data was the monitoring of trends. All data systems managed by NCHS tried to cover different aspects of four health dimensions: (a) health status; (b) the use of health services; (c) environment and behaviour; and (d) insurance coverage and use. In relation to health status, several dimensions were of potential interest, including physical and mental health status *per se*; functional ability and disability; biological and physiological attributes; and genetic make-up and risk factors. Not all were covered as yet by the data collection systems of NCHS.

Madans reported that NCHS operated four major data collection systems. The first was the National Vital Statistics System that produced data on births, deaths and foetal deaths for the whole country. NCHS compiled the data gathered at the state or county levels, where the basic data were collected according to state or county rules. The data had achieved complete coverage but were not free from error. A problem faced in deriving rates was the lack of consistency between numerators obtained from the Vital Statistics System and the denominators obtained from census data, especially in the case of ethnic minorities.

The second system operated by NCHS was the National Health Care Survey, an integrated family of seven record-based surveys that was used to monitor the utilization and content of

medical care in the United States. The surveys collected data on the characteristics of providers and patients, as well as on services provided, diagnoses, payment sources and outcomes. The data gathered were used to enhance and expand measures of the prevalence of disease. Madans noted that there were two other sources of the incidence of disease that were not operated by NCHS: the cancer registers maintained by the National Cancer Institute and the reporting system of specific conditions operated by the Centers for Disease Control (CDC). The cancer registers were not representative of the whole population. The CDC system focused on rare conditions and its completeness depended on physicians abiding by the reporting rules established by the Department of Health and Human Services.

NCHS also operated two major population-based surveys: the National Health Interview Survey (NHIS), which was the principal source of information on the health of the non-institutionalized population in the United States, and the National Health and Nutrition Examination Survey (NHANES) which gathered information on the health status of the population by conducting standardized medical examinations as well as blood and urine tests. Together these surveys produced a good picture of the health status of the population of the United States.

The NHIS had been conducted annually since 1957 and every year it gathered information from some 40,000 households covering about 100,000 persons. African-Americans and Hispanics were oversampled. Until 1996 the approach used to determine health status was based on a subject's self-assessment of health status; information on the prevalence of selected conditions, including heart disease, diabetes, cancer, pulmonary diseases, depression and arthritis; and information on limitation of activity, that is, the subject's ability to perform age and sex appropriate roles and activities of daily living. Focusing on the limitation of activity because of ill health was thought to provide a more "objective" indication of health status than a self-assessment of that status. Such an approach was appropriate as long as ill health was primarily the result of acute conditions where the onset was clear and there was a direct association between the conditions and behaviour.

However, as a result of the epidemiological transition, acute conditions were being replaced by chronic conditions. Given the greater difficulty in diagnosing chronic conditions, longer periods were being spent in the disease state both prior and after diagnosis, making the traditional approach used by NHIS problematic. For that reason, in 1996, investigation of the prevalence of chronic conditions changed: respondents were asked first if they had ever been diagnosed with a condition or if they had symptoms associated with specific conditions and, if they had, follow-up questions were asked about the date of onset, use of medical services and medications. Since data on functioning were still gathered as before, the availability of more complete information on chronic conditions could potentially allow a better assessment of the interrelations between the social and biological aspects of health. Madans thus underscored that health status was much more than the causes and progression of disease; social, cultural and economic characteristics conditioned the perception of symptoms, the adoption of health-related behaviours, and the use of health care, and thus determined self-perceived health status. Surveys such as the NHIS were considered the best suited tools to obtain information about both the social and the biological aspects of health status.

In comparison to the NHIS, the NHANES focused on the biological aspects of disease by incorporating standardized physical and medical examinations, laboratory tests, nutritional assessments and an in-depth questionnaire. The NHANES was described as costly and demanding. It required the use of mobile testing units, covered 5,000 persons annually and took 6 years to be completed. It was useful for the study of trends in the prevalence, treatment and control of selected diseases; for the investigation of risk factors associated with selected diseases; for the analysis of trends in risk factors; and for the study of the relation between diet, nutrition and health.

To enhance the usefulness of the data sources it operated, NCHS had been engaged in creating linkages between different data sets and exploiting the potential for longitudinal coverage. For instance, the results of cross-sectional surveys were being linked with mortality records to obtain some sort of "longitudinal" cov-

erage. Similarly, sub-samples of the populations covered by certain surveys were being traced and re-interviewed. Lastly, attempts were being made to carry out "statistical matching" of persons covered by the NCHS surveys and those covered by Census Bureau surveys or censuses, so that more detailed socio-economic characteristics could be studied in relation to health. However, the protection of confidentiality was hindering the use of such matching.

The discussion focused on a number of ethical issues related to the investigation of health status. When surveys involving medical examinations were carried out, the question of what to do if a serious condition was discovered was raised. In the case of the NHANES, written guidelines existed. Interviewers were instructed to contact the persons tested and to refer them to appropriate health care providers if certain conditions were detected, but not all conditions were disclosed to the persons tested. NCHS was considering the possible use of genetic testing as part of the health surveys it conducted but discussions were going on regarding the ethical issues involved and the need to ensure informed consent. Because such testing, if done, would cover nationally representative samples, it was not expected to yield useful results regarding rare gene mutations. Instead, it would provide information about the relative frequency of commonly occurring polymorphisms, some of which were already known to have specific implications for health prospects. The issue of whether to inform the individuals tested about the results of genetic testing was far from resolved.

It was noted that, although the population-based surveys of NCHS had large sample sizes, they were insufficient to cover certain minorities in the United States, such as American Indians or the populations of Asian origin. NCHS considered that over-sampling such populations would be too costly. A possible alternative and more cost-effective approach could be the use of regional surveys that took advantage of the fact that such minorities tended to be concentrated in certain geographical areas.

Regarding the use of surveys to measure health status in developing countries, questions were raised about the accuracy of information

provided by the head of household on the health status of other members of the household. It was also noted that, in the Gulf countries, most nationals had access to medical care so that questions focusing on medical diagnoses and utilization of health services were appropriate. However, in countries lacking similar health services, there were doubts that such questions would yield usable information.

#### D. HEALTH AND MORTALITY IN DEVELOPED COUNTRIES

Shiro Horiuchi provided a general framework for the understanding of the processes leading to significant declines of mortality over the history of humanity. Five epidemiological transitions were identified, three that characterized past experience and two that were still to take place. Each transition was associated with a change in the major causes of death. The first occurred when external injuries gave way to infectious and parasitic diseases as the most important causes of death. Horiuchi indicated that such transition was associated with the sedentarization of the human population which, by increasing population concentration and reducing the dependence on hunting reduced the likelihood of injuries and favoured the transmission of infections. The second transition was more recent, having occurred mostly during the nineteenth and twentieth centuries, and consisted of the decline of infectious and parasitic diseases as causes of death and the increase of degenerative disease. The third transition involved the decline of cardiovascular disease as a cause of death and was the one experienced since the 1970s by developed market-economy countries. The next transitions expected would involve, respectively, the reduction of cancer as a cause of death and the slowing of senescence. The very recent reductions in mortality rates due to cancer recorded in some developed countries (e.g., the United States) seemed to indicate that the fourth transition might be starting.

Horiuchi noted that such a framework did not imply that there had necessarily been a continuous and progressive reduction of mortality through human history. Reversals were possible and had occurred. Thus, the concentration of population in rapidly growing cities caused by the industrial revolution led to higher risks of



contracting an infectious disease and, consequently, to higher mortality. More recently, the high prevalence of smoking and excessive alcohol intake combined with a high-calorie, high-fat diet and sedentary lifestyle, especially among the inhabitants of countries with economies in transition, had not only prevented the reduction of mortality due to cardiovascular disease but had even led to increasing mortality caused by cancer and injuries. Lastly, the emergence of new infectious diseases, such as HIV/AIDS, or the re-emergence of well-known ones also threatened to reverse the transitions already experienced by some populations.

In developed market-economy countries, the number of persons surviving to very advanced ages had risen markedly. Among the oldest-old (persons over age 80 or 85), the proportion of deaths due to cardiovascular disease and cancer declined with age whereas the proportion of deaths due to senescence, that is, to the deterioration of normal bodily functions and especially of the immune system, increased. Thus, whereas middle-aged and older individuals tended to die of cardiovascular disease or cancer, the oldest-old were more likely to succumb to influenza, acute bronchitis, pneumonia, acute digestive disorders or congestive heart failure.

In presenting the paper by Manton, Stallard and Corder, Larry Corder focused on the problem of establishing the limits of longevity. Reviewing the different approaches that had been suggested to establish such limits, Corder noted that the distinction made between endogenous (i.e., genetical) and exogenous (i.e., environmental) factors as determinants of the length of life was not useful to establish the possible limits of longevity because recent biomedical research had shown that the interaction of both types of factors was necessary to prolong life. The study of the mechanisms of cell growth and the preservation of its functions had shown that, because of the time lag in the response of complex systems to environmental stresses, such response usually involved an excess expenditure of energy to counterbalance the anticipated change in the environment during the lag between perception and response. Such augmented response not only increased the probability of survival of the organism but also its ability to meet, over the long run, greater environmental stress. That is, the organism's

"fitness" was enhanced. In the case of human populations, such mechanisms might be at the root of the observation that the rate of loss of certain types of biological functions, such as cardiovascular function or the ability to improve voluntary muscle function through training, was lower in elderly persons with no manifest chronic diseases that previously observed.

Mention was also made of experiments with mice showing that restriction of caloric intake increased life span. The most frequently cited explanation for this finding was considered to be that caloric restriction reduced oxidative stress. However, in human populations, the epidemiological evidence relating caloric intake with life span did not show consistently the expected inverse relationship.

Consideration of two basic mechanisms involved in the maintenance of cellular function when subject to stress, heat shock proteins and drug metabolizing enzymes such as the P450 group, indicated that under certain circumstances exogenous factors could mimic endogenous cell regulators, so that both endogenous and exogenous chemicals could affect the genetic regulation of cell function. It was therefore incorrect to assume that a person's genetic mechanisms were fixed and not alterable by external intervention. Similarly, the immune system could be altered by exposure to external factors and there was growing evidence about the fact that viral or bacterial infections could be the underlying cause of certain chronic diseases.

In view of the above it was concluded that, in order to adapt successfully to the external environment, exposure to small stresses early in life was necessary to ensure that appropriate cellular defenses developed. Because of the complexity of the systems involved, the risk of death from environmental challenges would have to act over various dimensions and, since the effects of past exposure to different risks would be embedded in the individual, that individual's state at time  $t$  would depend on his state at time  $t-1$ . These considerations led to a model of human aging expressed in terms of a  $J$ -dimensional auto-regressive stochastic process. Such a model reflected the conclusion that, because of the complexity of the human body, the limits of longevity could not be established deterministically but were rather the result of

stochastic processes. As more persons survived to more advanced ages, the likelihood that an individual would live more than anyone else before increased stochastically.

Tapani Valkonen spoke next about the widening mortality differentials by socio-economic status in developed countries. He reviewed the various methodological problems faced in measuring those differentials adequately, particularly because the variables used to determine socio-economic status might change over time and be correlated to health status (e.g., persons who were ill or disabled were more likely to have lower incomes or to be out of the labour force than persons who were fit) and because the traditional data sources for deaths by cause only recorded a few indicators of socio-economic status. Furthermore, there was often a lack of consistency in the recording of socio-economic status between the vital registration system used to obtain the numerators of mortality rates and the census used to obtain the denominators. In the Nordic countries the last problem was avoided by linking the death registration system to the population register and thus ensuring the consistency of both.

Focusing on mortality differentials by occupation, which had the disadvantage of leaving out those who did not work, Valkonen reviewed their trends for a number of developed countries. In Denmark, England and Wales, Finland, Norway and Sweden the mortality differentials between manual and non-manual male workers tended to increase between the late 1960s or early 1970s and the 1980s, mainly because mortality due to cardiovascular disease declined faster among non-manual workers than among manual workers. In Finland, a reduction of those differentials was recorded between the late 1980s and the early 1990s, as manual workers experienced a more rapid reduction of mortality due to cardiovascular disease than non-manual workers. In some countries, changes in the relative impact of other causes of death, such as those related to alcohol abuse (Finland) or cigarette smoking (Norway), were also responsible for the trends in the observed differentials, with men in the lower-status occupational groups showing higher mortality caused by the adoption of such high-risk behaviours.

Reasonably reliable data on trends in mortality differentials by socio-economic status for groups other than working-age men were said to be limited. In the cases of England and Wales and Finland, mortality differentials by socio-economic status had increased significantly since the 1970s for both men and women and it seemed quite plausible that diverging trends in mortality due to cardiovascular disease between the higher and lower socio-economic groups were at the root of such increases for both sexes and at all adult ages. Since WHO had established as an important goal the reduction of socio-economic differentials in mortality, it seemed pertinent to underscore the need to understand better the factors and processes leading to a widening of the differentials so that adequate policy interventions could be devised.

Douglas Ewbank considered the effects of the genetic make-up of populations on mortality by cause of death by focusing specifically on the fact that certain commonly occurring alleles of the APOE gene had been associated with a higher incidence of ischaemic heart disease (IHD) and Alzheimer's disease. Using information on the relative frequency or the different alleles of APOE in different populations, Ewbank proposed a method for estimating the "excess" mortality due to ischaemic heart disease because of the frequency of the high-risk alleles of APOE in one population relative to that of a standard population. Such estimates showed that substantial proportions of the difference in mortality due to ischaemic heart disease in different populations could be explained by differences in the frequencies of the e4/3 and e4/4 alleles of the APOE gene between the two populations.

The estimates of excess mortality were made only for developed countries, where populations with high frequencies of the key alleles were concentrated in Northern Europe (Denmark, Finland and Sweden) and in the United States (among blacks) and where high-fat diets that were known to increase the risk of ischaemic heart disease were common. In developing countries, the frequency of e3/4 and e4/4 was known to be particularly high in Africa (e.g., Nigeria) and in Papua New Guinea, where mortality due to ischaemic heart disease and

Alzheimer's disease was low. A possible explanation for this outcome was that low-fat diets helped prevent the detrimental effects of the  $\epsilon 4/3$  and  $\epsilon 4/4$  alleles. In that case, adoption of a high-fat diet by the populations of Africa and Papua New Guinea would likely result in high mortality due to ischaemic heart disease. In contrast, Chinese populations, which were characterized by low frequencies of  $\epsilon 4/3$  and  $\epsilon 4/4$  alleles, might adopt a high-fat diet with few detrimental effects in terms of increased incidence of ischaemic heart disease or Alzheimer's disease. An important implication of these observations was that, in studying the relation between diet and morbidity or mortality across populations, it was necessary to control for variations in their genetic make-up, otherwise misleading associations might result.

The discussion noted that, given the major advances made in reducing mortality in developed market-economy countries, it was pertinent to ask what were the limits of human life span and whether they could be realized at the population level. Current biomedical research certainly raised interesting prospects about the possibility of increasing longevity, but the papers presented did not offer clear guidelines for action. For instance, it was not clear whether genetic factors played a greater role in survival up to age 65 or beyond. The model proposed by Manton, Stallard and Corder seemed to say little about what the limits of a population's average life expectancy could actually be and the relative risks of dying because of specific causes that were derived from the model did not seem to have immediate relevance for the formulation of health interventions. Besides, it was not clear whether the model was meant to be descriptive or predictive. Estimation of the parameters it required seemed less than straightforward given the current state of mortality and health statistics.

There was considerable discussion about the framework proposed by Horiuchi to analyse the various epidemiological transitions. His attempt to relate the different transitions to a specific type of society was judged to be too simplistic and possibly misleading. It was noted that the factors determining the transition from one set of causes of death to another varied from country to country, and that the transition from high to lower mortality often occurred in different

groups of society at different times and depended on various factors that were subject to greater or lesser control by the individual. It was suggested that a more complex type of framework was necessary, one that allowed for feedback mechanisms, advances and reversals.

Any type of framework should recognize the separate relevance of factors such as: control over the environment and sanitation; availability of health services and access to them; the type of political and social organization; the impact of nutrition. Several participants underscored the importance of the latter in understanding why certain transitions had happened. It was mentioned, for instance, that measles killed very few children in developed countries while it was a major cause of death in developing countries, mainly because of the malnourished status of children in the latter. The reduction of mortality caused by infectious diseases in today's developed countries began when the food supply could be assured and the prevalence of malnutrition declined. Despite these observations, it was considered that Horiuchi's framework provided a useful "cognitive map" that put in stark focus the major turning points for humanity in terms of health and mortality and that highlighted the fact that significant improvements were occurring at an ever faster pace.

Noting the generally optimistic tone of the papers regarding the expectation that mortality would continue to decline in the future, a number of participants mentioned possible causes of concern, including the re-emergence of certain infectious diseases that were either resistant to available treatments or that could only be controlled by a concerted public health effort that had been dismantled and might not be undertaken rapidly enough; the emergence of new infectious diseases; the rise of mortality due to violence, outright conflict or war; the potentially negative effects of rising pollution on health; and reversals in the decline of high-risk behaviours, such as smoking, drug addition or excessive alcohol consumption.

Considerable attention was focused on the discussion of social inequality and its implications for health and mortality. Although it was recognized that striving to eliminate such inequality was laudable, concerns were expressed about its feasibility, especially in today's developed market-economy countries where indi-

vidualism was highly valued. Because of the stress on individualism, an increasing interpersonal variation in life choices and lifestyles was to be expected, some of which might involve adherence to high-risk behaviours having detrimental impacts on health. Furthermore, it was likely that such variability would result in the persistence of differences according to status. It was noted, however, that status did not necessarily depend on income. In the former USSR, for instance, those with lower educational attainment had higher mortality than those with higher educational attainment although the incomes of both groups were very similar. Access to health services also seemed not to be the major determinant of mortality differentials by socio-economic status in developed countries, since the evidence suggested that persons with lower socio-economic status made more use of health services than those with higher status. Furthermore, in most of the market-economy countries of Europe, socialized medicine provided similar access to health care to all groups. The likely causes of socio-economic differentials in mortality appeared to lie instead on behavioural and social factors. The adoption of healthier lifestyles and diets or the maintenance of more stable primary relations among those having higher socio-economic status probably made a significant contribution to lowering their risks of dying. Being single, widowed or divorced, for instance, was associated with higher mortality rates among both men and women, but especially among men. Access to social resources was likely to be a major determinant of a healthy life.

Lastly, with regard to the importance of genetic factors, the discussion emphasized the many questions that still remained regarding their relevance. Furthermore, it was underscored that the genetic composition of populations was inferred from the study of relatively small groups that were not necessarily representative of whole populations. Thus, the assertion that certain alleles of the APOE gene were more common among African populations than among those of Chinese origin was based on a number of studies of small populations that might not appropriately reflect the full variability within either of those groups. Nevertheless, the evidence suggested that significant differences existed between population groups and

that they should not be ignored when doing epidemiological analyses regarding the possible factors leading to the high prevalence of certain diseases.

#### E. HEALTH AND MORTALITY IN COUNTRIES WITH ECONOMIES IN TRANSITION

Martin Bobak reviewed health and mortality trends in countries with economies in transition, noting that whereas until the mid-1960s those countries had experienced declines of mortality similar to the ones recorded by the developed market-economy countries, since about 1965 life expectancy at age 15 in most of the countries of Central and Eastern Europe (including the former USSR) had been declining, especially among males. In recent years, however, some Central European countries had begun to experience a reversal of that trend. Thus, since 1990 life expectancy at age 15 had begun to rise in the former German Democratic Republic and the former Czechoslovakia, since 1992 in Poland and since 1994 in Hungary. The former USSR had also experienced a rise in life expectancy at age 15 between 1984 and 1987 but declines had set in thereafter. In the Russian Federation male life expectancy at age 15 had dropped by more than 7 years between 1989 and 1994, a decline of unprecedented magnitude in non-war situations. Reductions in life expectancy at age 15 were also recorded among women, though they were smaller in magnitude, and were also common among adults of both sexes in other successor States of the former USSR. In general, increased mortality due to cardiovascular disease and external causes was responsible for most of the increase recorded in the Russian Federation.

Bobak noted that the results of surveys using questions on self-rated health status corroborated the existence of poorer health in the former USSR than in the developed market-economy countries. Thus, whereas in Germany or Italy a low 5 per cent of the population reported having very poor health, in the Baltic States and the Russian Federation the equivalent proportion was 25 per cent. Bobak discussed the validity of the various reasons advanced to explain the stark differences in health status observed between the former USSR and other countries with economies in transition on the

one hand and developed market-economy countries. He noted that differences in the health care system accounted for only a small fraction of the disparity. Higher levels of environmental pollution were also not a major contributing factor. Differences in lifestyles, the high prevalence of high-risk behaviours (including smoking and excessive alcohol intake), and the high fat content of the diet were the major factors underlying the diverging trends observed. Such proximate determinants, however, had a variety of psycho-social factors as root causes, factors that were intrinsically related to the major socio-economic transformation that the populations of the countries with economies in transition and particularly the CIS countries were undergoing. A useful concept in trying to understand the effects of such transformation was that of "social capital", which encompassed all the trustworthy relationships that an individual could rely on. The erosion of trust that accompanied the transformation of society led to greater stress at the individual level and conditioned individual choice in ways that made high-risk behaviours appealing. Thus, individuals having lower "social capital" were expected to be subject to higher mortality risks. That seemed to be the case of men who were not currently married and had low levels of educational attainment, whose mortality rates were considerably higher than those of other groups. From the "social capital" perspective, sharp increases in mortality differentials by socio-economic status were expected.

Citing Powles, Bobak pointed out that several paths could be followed to effect the transition from high to low mortality. In the cases of some Northern European countries, such as England and Wales, the reduction of infectious and parasitic diseases had resulted in low mortality among children but had not reduced much mortality among adults, especially among men, who had remained subject to high risks of death from cardiovascular disease until the 1970s. According to Powles most developed market-economy countries had followed such a "central path", passing from high levels of mortality due to infectious diseases to initially intermediate levels of mortality due to non-communicable diseases among adults. Southern European countries (including Albania and the former Yugoslavia) had followed a fast track, benefiting from the reduction of mortality due to

infectious and parasitic diseases but also maintaining their traditional low levels of mortality caused by non-communicable disease, especially cardiovascular disease. Central and Eastern European countries had taken the slow route, controlling mortality due to infectious diseases but failing to reduce mortality from non-communicable diseases. Since the proximate determinants of high levels of mortality caused by non-communicable disease were known, the challenge was to establish which societal mechanisms could be used to modify behaviour.

Witold Zatonski focused on the path taken by the epidemiological transition in Poland. He remarked that in the early 1930s, life expectancy in Poland was about 12 years lower than in Germany. After the Second World War, with the introduction of antibiotics, mortality due to infectious disease declined rapidly so that by 1965 Poland and Western Germany had similar expectations of life at birth. However, from 1965 to 1990, the expectation of life in Poland changed little because, although mortality continued to decline among infants and children, the mortality of adult males increased. Thus, the age-standardized mortality rate of Polish men aged 45-64 rose markedly between 1965 and 1988. This increase was dominated by deaths caused by cardiovascular disease, cancer and external causes. Among women aged 45-64, mortality stagnated over the same period. Female mortality from cancer and external causes remained stable while that from cardiovascular disease showed a slight increase.

Special attention was given to changes in male mortality over the period 1980-1996, when Polish society underwent major social and economic transformations. Mortality due to external causes dropped during 1980-1982, partly because the imposition of martial law reduced traffic and imposed restrictions on alcohol sales. During 1989-1991 there was a significant increase of overall mortality among men aged 15-64, stemming primarily from a higher mortality rate because of external causes. During the period, lax border controls facilitated the importation of vodka from the former USSR and, as standards of living rose, car ownership and road traffic increased. Both factors probably contributed to the rise of deaths caused by external causes. Lastly, since 1991

life expectancy in Poland began to rise again, with men posting larger gains than women. Mortality rates fell at all ages but particularly among adults aged 20-64. Reductions of mortality due to cardiovascular disease were the major contributors to such trend but, among men, a decline in mortality from lung cancer and external causes also had an effect.

Zatonski argued that the recent decline of mortality caused by cardiovascular disease could be attributed to changes in lifestyles brought about by the social and economic changes that Poland had undergone. During 1980-1982, many food products became scarce, particularly meat, animal fats, eggs and sugar. Average calorie intake fell by about 10 per cent. Later in the decade, as wages fell and government subsidies for food were discontinued, animal products became less accessible than plant products for many people. Furthermore, with the opening of the market, new low-fat products or those having less saturated fat became available. During the 1980s, although overall fat consumption remained almost constant, animal fats were replaced by vegetable fats, mainly because the latter were cheaper and because, being imported products, they were the subject of intensive advertisement campaigns. In addition, fresh fruits and vegetables became more accessible all year round both as a result of changes in national production and because of imports. Such changes led to a transformation of the Polish diet that probably played a major role in reducing mortality due to cardiovascular disease during the 1990s.

Changes in tobacco consumption also appear to have played a role in the reduction of mortality in Poland. Tobacco consumption among males in Poland increased steadily until the late 1970s and stabilized during the 1980s. Data on the prevalence of daily smoking among men suggested that it decreased during the 1980s. In the 1990s the organized health lobby in Poland prompted the first democratically appointed Senate to draft a law to limit the detrimental effects of tobacco smoking in the country. The law came into effect in May 1996 and was expected to further spur the avoidance of tobacco smoking among the Polish population.

Evgueni Andreev discussed the dynamics of mortality change in the Russian Federation. He

noted that during the Soviet period of Russia's history there were only two intervals over which life expectancy had increased steadily: from 1922 to 1928 and from 1948 to 1965. Since 1965 life expectancy had been declining, except during 1985-1987 when an anti-alcohol campaign seems to have reduced mortality rates somewhat. Increases of mortality were particularly marked during 1989-1994 so that, by 1994, males in all age groups above 10-14 were displaying the highest mortality rates observed since 1947.

Data on mortality by cause of death in the Russian Federation were only available since 1959 and only for urban areas at that time. They indicated that up to 1965 mortality caused by infectious diseases had been declining, especially that caused by tuberculosis in adults and by diarrhoea in children. More recently, during the rise of life expectancy in 1985-1987, data on cause of death indicated that mortality due to external causes (accidents and injuries) had been significantly reduced. Analysis of the components of change of life expectancy between 1959 and 1996 showed that, for males, 3 years of life had been lost because of cardiovascular disease and another 3 because of external causes (accidents, poisonings and violence). For females the equivalent estimates were 1.7 and 0.9 years. The life expectancy of both men and women also increased because of reductions in mortality due to other causes.

In an effort to explain the dynamics of mortality change in the Russian Federation in relation to changing causes of death, Andreev used principal component analysis to determine which were the main clusters of causes of death associated with increases or decreases of mortality. Such analysis suggested that the factors leading to mortality increase were global in nature since the principal components identified included causes of death of a diverse etiological character. Furthermore, when the relevance of the components identified was measured with respect to the decline of mortality registered during 1985-1987, it was found that the clusters of causes explaining such decline were different from those related to the increase of mortality. These findings plus the corroboration that the rise in mortality was larger among men with lower levels of educational attainment than among the better educated suggested that the

factors ultimately responsible for the mortality trends observed in the Russian Federation operated at the societal level.

Lastly, Andreev noted the wide regional disparities in survival that existed within the Russian Federation, with the expectation of life at birth in 1996 ranging from 50 to 65 years for males and from 62 to 75 years for females. In general, life expectancy tended to be higher in the western and southern regions of European Russia, where population density was higher and the rural population was substantial, and lower in the more sparsely settled northern and far eastern parts of the country.

The discussion underscored the similarity of mortality trends experienced by the countries of Central and Eastern Europe until 1990. However, although it was accepted that the general traits of those trends were known, some questions were raised about possible biases in the estimates available. The quality of information on infant mortality in the former USSR, for instance, was questioned since there were different assessments of the impact that a change of definition introduced in 1993 would have. Yet, it was recognized that even if mortality in childhood had not declined as much as estimated, its impact on the overall change of life expectancy would not be large. There were also questions raised about the reasons for the stagnation or increase of adult mortality in the different countries. It was noted, in particular, that the reporting of causes of death in the former USSR lacked accuracy and that in 1987, at the initiative of the Ministry of Health, deaths of unknown cause that were formerly reported as caused by cardiovascular disease began to be reported in the ill-defined category. Hence, a reduction of mortality due to cardiovascular disease since that date would be spurious. There were also questions raised about the rapid rise of mortality caused by lung cancer and cirrhosis of the liver in Hungary. Several participants noted that information on the level of alcohol consumption did not always correlate well with the level of mortality due to cirrhosis. In some cases, as in certain regions of the Russian Federation, cirrhosis was also caused by exposure to pesticides.

Although it was recognized that care should be taken to ensure that the estimates used were

as free of bias as possible, there was general agreement that in most Central and Eastern European countries the quality of the data on mortality in general and on mortality by cause of death were adequate. There were, however, severe limitations in the availability of data on key factors affecting health and mortality. In the Russian Federation, for instance, there was no information on smoking or on alcohol consumption at the individual level. Only rough estimates based on overall consumption could be made.

Participants noted that it was important to bear in mind that risk factors often acted in a multiplicative fashion, so that the interaction between them could not be discounted. The inadequacy of data and limitations in analytical methods often precluded the identification of the risks related to each factor separately as, for example, environmental pollution vs. behavioural risk-factors. The practice of comparing similar populations in different geographical locations to deduce the relative importance of various factors in determining mortality differentials between them could be misleading because it was unlikely that all possible determinants of those differentials could be controlled for.

There was some discussion of societal factors that might be at the root of the mortality increases observed in Central and Eastern European countries. Mention was made of the sense of hopelessness that many people might have felt under a regime that offered few prospects for social or economic mobility. Especially in periods of transformation, the situation of many people was likely to have worsened. Under those circumstances, the weakest members of society might be persons that had low "social capital", that is, few meaningful relationships with other persons. Such considerations would explain the high mortality rates observed among men who were not currently married. However, several participants pointed out the dangers of focusing on such a group, since selectivity effects might be involved. That is, men who remained single or those who were divorced might have done so because their health was worse to begin with than that of other men.

Lastly, the experience of Finland was cited as a good example of what Central and Eastern



Europe could learn from the experience of developed market-economy countries. In the 1960s, Finland was facing the same prospects of continued high mortality in adult ages as Central and Eastern European countries today but, because Finland had good data on mortality by cause of death, the immediate reasons for the stagnation of mortality could be detected early on. Furthermore, epidemiologists soon became engaged in finding factors that correlated with increased survivorship and were vocal in disseminating their findings. Lastly, there was the political and societal will to implement measures or undertake campaigns that would change the behaviour of individuals. In Finland both political parties and non-governmental organizations collaborated in achieving that goal.

#### F. MORTALITY IN DEVELOPING COUNTRIES

Ian Timæus reviewed existing evidence on mortality trends in sub-Saharan Africa, a region characterized by the highest mortality levels in the world. There were several reasons to be pessimistic about the future evolution of mortality in the region, including the economic difficulties that many countries in the region were experiencing, the negative effects of structural adjustment programmes on social development and on the functioning of the health sector, the spread of the AIDS epidemic, and the prevalence of internal conflict or outright war. However, there were also some reasons for optimism, including the fact that some countries were experiencing economic growth; that the democratization of South Africa was contributing to the stability of the Southern part of the continent; that conflicts had been or were being resolved in Angola, Ethiopia and Mozambique; and that considerable advances had been made in combatting childhood diseases through, among other things, immunization campaigns such as those promoted by UNICEF.

Timæus noted that the availability of data allowing the estimation of mortality in sub-Saharan Africa was dismal. Among mainland countries, none had an adequate vital registration system and only in South Africa were a sufficient number of deaths registered at the national level so as to allow the indirect estimation of mortality. Consequently, for most

countries of sub-Saharan Africa mortality had to be estimated on the basis of information obtained through censuses or sample surveys, which were more likely to record data allowing the estimation of mortality in childhood than those needed to estimate adult mortality. Essentially, only three types of data were useful for the indirect estimation of adult mortality: the number of deaths occurring over a specified period (usually a year); information on the survival of parents; or information on the survival of siblings. All could be obtained through large-scale sample surveys and the first two could also be gathered through censuses.

Timæus reviewed the evidence regarding levels and trends of child mortality in sub-Saharan Africa, paying particular attention to countries that had carried out Demographic and Health Surveys. He concluded that under-five mortality in the region tended to be high, with only a few countries having an under-five mortality below 100 deaths per 1,000 births. Although in most countries with data available under-five mortality had tended to decline during the 1980s, improvements of child survival appeared to have tapered off in Botswana, the Central African Republic, Côte d'Ivoire, Kenya, Malawi, Rwanda and Zimbabwe, and mortality had stagnated or increased in Nigeria and Zambia. Timæus argued that the AIDS epidemic was an important factor leading to a slowdown in the mortality decline experienced by a number of African countries but by no means in all, since some showed signs of stagnation before the epidemic took hold. Other factors, therefore, had to be considered in explaining the adverse trends experienced by some countries.

Estimates of adult mortality could be obtained for 22 out of the 43 countries in sub-Saharan Africa. A quarter of the estimates referred to the first half of the 1980s, half to the rest of the decade and a quarter to the period 1990-1992. The indicator used to measure mortality levels was the probability of surviving between exact ages 15 and 60, and it was derived using indirect estimation techniques applied to data on deaths over a period preceding a census, data on the prevalence of orphanhood, or that on the survival of siblings. According to the estimates obtained, adult mortality levels varied widely among the countries of sub-Saharan Africa. Thus, in the Central African Republic, Mali

(towards the mid-1980s) and Uganda, about half of all persons aged 15 were expected to die before age 60, whereas in Ghana, Kenya and, by the end of the 1980s, Senegal the equivalent proportion was 20 per cent. In most of sub-Saharan Africa, adult men had higher mortality than women, but in Malawi women's mortality was higher than that of men. Data allowing an assessment of trends in adult women's mortality showed that it declined rapidly in Cameroon, Niger and Senegal, but that the decline slowed down or that mortality increased in Botswana, the Central African Republic, Lesotho, Malawi, Uganda, the United Republic of Tanzania and Zimbabwe. Estimates derived from the survival of siblings showed that both male and female adult mortality increased markedly in Uganda and Zimbabwe and slightly less so in the Central African Republic and Malawi, mostly during the 1990s. The onset of the AIDS epidemic was considered a major factor underlying such increases. Thus, the evolution of mortality in sub-Saharan Africa showed that the increase of survivorship was not an irreversible process, though the full set of factors leading to the reversals observed remained to be explored. In particular, the role that economic difficulties had had in slowing down reductions in mortality demanded further attention.

Juan Chackiel discussed mortality trends in Latin America and the Caribbean. He noted that censuses had been carried out systematically in most countries of the region since the 1950s, but that there had been a deterioration of their quality and completeness of coverage. Furthermore, five countries in the region had not carried out a census during the 1990 round. With regard to data on deaths, most countries of the region had an operating system of death registration, but its completeness of coverage varied widely, with Chile, Costa Rica and Cuba having virtually complete registration of deaths and the Dominican Republic, Nicaragua, Paraguay and Peru registering less than 60 per cent. Furthermore, even in countries where the completeness of death registration was judged to be high, the data were not free from other errors, including age misreporting, changes in the completeness of registration by age, and problems in the reporting of cause of death.

Estimates for 1990-1995 showed that, among all developing regions, Latin America and the

Caribbean as a whole had the highest life expectancy (69 years), having experienced an increase of 18 years since 1950-1955. Given the constraints imposed by data availability, mortality trends were analysed in detail for three countries with very low mortality (Chile, Costa Rica, Cuba), four with low to intermediate mortality (Argentina, Mexico, Uruguay and Venezuela) and one with high mortality (Guatemala). Among those countries, Chile, Costa Rica and Cuba had a life expectancy of about 75 years, similar to that of developed market-economy countries, whereas Guatemala's life expectancy was 63 years. Mortality was higher among men than among women and, especially in countries with intermediate or high mortality levels, the sex differentials in mortality were increasing. Focusing on mortality rates over the age range 15-44, male mortality was found to be considerably higher than female mortality (in some cases twice as high) and the differentials between the two showed a tendency to increase over time. The major causes of death in that age range were external causes (injuries and violence) among men and neoplasms among women. Mortality among those aged 45-64 ranged mostly between 65 and 80 deaths per 1,000 among women and between 120 and 145 deaths per 1,000 among men, but in Guatemala the equivalent rates were 126 and 172 deaths per 1,000 for men and women, respectively. The main causes of death for both men and women in that age group were cardiovascular disease and cancer. The same causes predominated among older persons (aged 65 or over), whose mortality levels had nevertheless been declining steadily since 1950. Gains in survivorship had tended to be higher among elderly women than among elderly men.

The data for the 8 Latin American countries considered confirmed that they had all experienced a decline of mortality due to communicable diseases. However, Chackiel noted that the data available did not allow an assessment of the likely impact of the AIDS epidemic in the region (countries with high prevalence of HIV infection had deficient mortality data) and that the re-emergence of certain infectious diseases, such as cholera, seemed to have increased morbidity rather than mortality. He emphasized the need for morbidity statistics or information on the health status of the population to assess the stage of the epidemiological transition in

which different populations found themselves. He also discussed the relevance of considering the health and mortality experience of different socio-economic groups since, given the marked inequalities that characterized Latin American societies, differentials in mortality by socio-economic status were expected to be revealing and useful tools for planners.

The discussion contrasted the experiences of sub-Saharan Africa and Latin America and the Caribbean, underscoring the fact that data availability in the latter regions was considerably better than in Africa. However, data deficiencies were also common among Caribbean countries, since only Cuba could be included in the in-depth study undertaken. Particular emphasis was put on the lack of adequate data on adult mortality trends in sub-Saharan Africa and on the problems involved in trying to estimate trends from data on the survival of siblings which, being obtained through surveys, were subject to sampling variability and might moreover be affected by reporting errors. Despite data deficiencies, it was agreed that countries in Latin America and the Caribbean had been considerably more successful in reducing mortality than those in sub-Saharan Africa. Yet, the two regions showed certain similarities. Thus, countries that had embarked earlier in the epidemiological transition, such as Argentina and Uruguay in Latin America or Ghana and Kenya in sub-Saharan Africa, had been experiencing a reduction of the rate of mortality decline in recent periods (since the 1970s). Cuba was another country where the pace of mortality decline had slowed since the mid-1970s. Senegal, however, was an exception, since it had maintained a rapid rate of improvement of the survivorship chances of its population since the 1970s.

The discussion noted that there were sufficient elements to infer that the decline of mortality had stagnated or even been reversed among both children and adults in several of the sub-Saharan countries affected by the AIDS epidemic. The fact that the estimates of child mortality for those countries did not always show clearly increasing trends might be due to the underestimation of mortality levels stemming from respondent bias: HIV-positive mothers who had died would not have been able to report their dead children. Nevertheless, some

scepticism was expressed about the high prevalence levels of HIV infection reported for certain countries, especially if they were derived from testing women who sought pre-natal care. Being pregnant, those women were more likely to have been exposed to infection than women who were not pregnant, and pregnant women seeking pre-natal care were more likely to be sick than other pregnant women who did not seek medical attention. Consequently, prevalence levels of HIV infection among pregnant women attending pre-natal care programmes probably overestimated the prevalence among the whole female population. It was argued, however, that a counterbalancing mechanism might also be in operation if HIV-positive women were less likely to become pregnant than other women did. Despite these concerns about the accuracy of existing indicators, it was agreed that HIV infection was almost certainly a major contributor to the increases of adult mortality experienced by some countries of sub-Saharan Africa, especially since in the African context the progression between HIV infection, full-blown AIDS and death was more rapid than in developed market-economy countries where complex drug therapy was available. Given the rapid spread of the disease in some contexts, the expectation was that the chances of survival in several countries would keep on deteriorating for some time. However, there were some hopeful signs, such as reports suggesting that the prevalence of HIV infection had been declining in Uganda.

The possible contribution of deteriorating economic conditions to the stagnation or increase of mortality was discussed but no firm conclusions were reached. It was argued that the mortality of adults (persons aged 15 to 64) was more likely to be affected by economic conditions than that of children since studies of the impact of severe recessions in Latin American countries, Eastern and Central European countries, and England and Wales during the 1930s had shown that they did not necessarily lead to stagnant or rising mortality in childhood. However, it was noted that the work of Murphy and Dyson had shown that, over the long-term, changes in GDP had an impact on child mortality. In sub-Saharan Africa, as a result of structural adjustment programmes, user-fees had been introduced by the health services and less people were resorting to such services. Fur-

thermore, because of the stringent economic conditions, the nutritional status of both adults and children had been deteriorating. However, the impact of such changes on mortality was not expected to be immediate. An intensive study in a small region of Zambia, for instance, had shown that there was at least a two-year lag between the deterioration of economic conditions and its measurable impact on mortality. The need for more studies of that type was stressed.

It was suggested that one way of establishing the causes of mortality stagnation or its increase was by a process of elimination. If a country was not highly affected by the AIDS epidemic, if it had programmes in place to combat mortality in childhood, and if it had not experienced internal conflict or war, the most likely cause left would be a stagnating or deteriorating economy. However, in a country such as Botswana, where the economy was growing, other factors had to account for the stagnation observed since the 1970s and it was not clear which. In fact, it was argued that the exact factors leading to a mortality decline were also difficult to identify. Thus, in the case of mortality in childhood, evaluation of the interventions generally credited for reducing it showed that they were not as efficacious as thought. Oral rehydration therapy, for instance, did not seem to have a major impact in Africa. As for the vaccination programmes adopted in most countries of the region, only the immunization against measles was geared to combatting a major cause of death in childhood. There were, however, some interventions that had been successful in reducing overall morbidity, including those geared to combat sleeping sickness, to eradicate smallpox, to provide vitamin A supplementation, or to foster the use of nets impregnated with insecticide so as to combat malaria. But it was agreed that a multifaceted approach was needed to sustain and improve the gains in survivorship recorded in sub-Saharan Africa and that the evidence suggested that once a moderate mortality level was achieved, the strategy needed to achieve further reductions was not always clear.

There were several attempts to draw parallels between the situation in Latin America and the Caribbean or sub-Saharan Africa with that in

Eastern and Central European countries. In particular, the issue of whether developing countries that were winning the battle against communicable diseases would be faced by stagnating or increasing mortality due to non-communicable diseases was raised. With respect to most of sub-Saharan Africa, such situation was considered unlikely since communicable diseases were still major causes of death and detrimental changes in lifestyles were yet to take hold. Most inhabitants of sub-Saharan Africa were still consuming relatively small amounts of meat and other sources of saturated fats, they engaged in sufficient physical exercise (walking, working etc.) and, although the level of smoking and alcohol consumption was rising, it was still at relatively low levels. Nevertheless, in some low-mortality countries of the region, there were worrying signs regarding the evolution of mortality among the male population. Thus, in the Republic of South Africa, adult men were experiencing mortality rates similar to those of their Russian counterparts and the incidence of cardiovascular disease was high. In Latin America and the Caribbean, the case of Cuba was not considered to be similar to that of Eastern and Central European countries since, although there was in Cuba a very low decline in the mortality of men aged 15-64 between 1970-1975 and 1985-1990, that slowdown seemed to be caused more by the very low levels already achieved than by a deterioration of male health. However, it was recognized that the country was facing a health care crisis because its health services were not sustainable and the country's poor economic performance during the 1990s was affecting the nutritional status of the population. As for the causes of the relatively small advances made in reducing mortality in Argentina and Uruguay, it was suggested that resistance to change within the health system and the maintenance of risky behaviours, such as excessive fat intake and the prevalence of smoking, contributed to such trends. In general, Latin American countries characterized by moderate to low mortality levels needed to put more emphasis on the prevention of non-communicable disease by, among other things, promoting the dissemination of information and undertaking concerted campaigns designed to reduce high-risk behaviours, if further advances in increasing life expectancy were to be made.

Lastly, the importance of considering not only indicators of mortality but also those related to health status was stressed. Data on the prevalence of specific diseases would probably provide better indicators of the advances being made in Africa, especially as immunization campaigns continued. More detailed information about morbidity patterns was necessary to assess whether countries were advancing in the epidemiological transition and what directions they might be taking. Important findings might be possible even if the data available did not achieve national coverage. It was suggested, for instance, that efforts be made to obtain the publication of data on deaths from the registration systems operating in some of the capital cities of Africa. Similarly, the study of the health situation of particular sub-populations was recommended, especially of slum dwellers in the cities of developing countries. Because the populations of most countries were heterogeneous, the development of adequate programmes for different segments of the population required information about their particular health needs. Use of diverse sources of data, some of which have not been traditionally exploited to study the health status of a population, was recommended as a first step to identify such needs.

#### G. RISK FACTORS UNDERLYING MAJOR CAUSES OF DEATH

Alan Lopez focused on two types of behaviour that placed people at a greater risk of early death: tobacco smoking and excessive alcohol intake. Epidemiological research had shown that smoking increased the risk of contracting cancer and cardiovascular disease, whereas excessive alcohol consumption increased the risk of cardiovascular disease, cirrhosis of the liver and injuries. Based on a number of studies that attempted to quantify the hazards associated with smoking, Lopez estimated that, in Western countries, smoking among males was associated with a 70 to 80 per cent excess mortality from all causes. At the individual level, smoking had the largest effect in increasing the relative risk of contracting cancer of the lung and of the upper-aerodigestive track and a smaller effect in increasing the risk of cardiovascular disease. However, because cardiovascular disease was a more common cause of death than the other

causes related to smoking, at the population level the impact of smoking was greatest in increasing the number of deaths due to cardiovascular disease. Thus, in 70 to 80 per cent of the smokers dying because of cardiovascular disease before age 50, the disease had been caused by smoking, making it the factor responsible for a three-fold increase in mortality during middle age. With respect to lung cancer, smokers were 10 to 12 times more likely to die of the disease than non-smokers and the excess risk associated with smoking increased with the duration and intensity of smoking. On average, a smoker who had begun smoking as a young adult and continued to do so for the rest of his or her life had at least a 50 per cent chance of eventually dying because of a smoking-related illness.

Lopez estimated that in 1995 tobacco smoking was responsible for 1.9 million deaths in developed countries, 1.2 million of which occurred in the developed market-economy countries and the rest in countries with economies in transition. In developing countries, smoking-related deaths amounted to 1.6 million, half of which occurred in China. This distribution of smoking-related deaths by region was changing rapidly. By 2020, smoking was expected to cause 8.4 million deaths annually, 6 million of which would occur in the developing world. That year, smoking would be by far the leading cause of death in the world. The expected trends in smoking-related mortality were the result of changes in the prevalence of smoking in different regions. WHO estimated that by the mid-1990s there were about 1.1 billion daily smokers in the world or about one-third of the world's population aged 15 years or over. Eight hundred million of those smokers were male and 700 million lived in developing countries. Globally, almost half (47 per cent) of all men aged 15 or over smoked, but considerably fewer women did so (12 per cent). There were, however, marked regional variations. Thus, in developed market-economy countries, 37 per cent of men were regular smokers, compared with 60 per cent in the countries with economies in transition and in China. The prevalence of smoking was also estimated to be high in other developing regions: 40 per cent among men in India, Latin America, North Africa and Western Asia, and 54 per cent in other parts of Asia. Between 1980-1982 and 1990-1992, cigarette

consumption declined by 1.5 per cent per year in developed market-economy countries, remained relatively constant in countries with economies in transition, and rose by 1.4 per cent annually in developing countries, with the result that global consumption remained relatively constant at around 1,650 cigarettes per adult per year.

In contrast with smoking, Lopez noted that the effect of alcohol consumption on survivorship was not necessarily negative. When consumed at low levels, alcohol provided some protection against ischaemic heart disease. However, moderate to high levels of alcohol intake increased the incidence of cirrhosis of the liver, certain cancers, certain types of cardiovascular disease and traumatic or violent deaths mostly due to accidents. According to estimates by Murray and Lopez, 1.25 million deaths were caused annually by alcohol, 625,000 because of injuries and 620,000 because of disease. However, alcohol intake was also responsible for preventing 470,000 deaths that would have been due to ischaemic heart disease. Therefore, the net annual contribution of alcohol consumption to mortality amounted to 775,000 deaths, 640,000 of which occurred in developing countries and 710,000 of which were deaths of men. Data on alcohol consumption compiled by WHO confirmed that alcohol consumption was rising rapidly in many developing countries and that hazardous consumption of alcohol was prevalent in many developed countries although some of them had been successful in reducing hazardous drinking (e.g., France).

Hugo Kesteloot focused on another set of behavioural factors considered to affect the risk of morbidity and mortality: diet and exercise. With regard to diet, Kesteloot reviewed the strong evidence linking high levels of saturated fat intake to the incidence of cardiovascular disease, especially ischaemic heart disease and stroke. The effect of high saturated fat intake was said to be modulated by genetic factors, smoking and obesity, and the level of serum cholesterol was used as a biomarker that had an important effect on the incidence of cardiovascular disease. Epidemiological evidence also suggested that high levels of saturated fat intake increased the risk of developing certain types of cancer (e.g., of the lung, breast, rectum and prostate) and that a high salt intake was related

to an increased incidence of stomach cancer. A diet high in sodium was also associated with increased blood pressure which was a major predisposing factor for stroke.

Kesteloot argued that nutrition and dietary factors were likely to have a more important influence on survivorship than genetic factors, since the genetic composition of the population had not changed much but countries that had adopted diets richer in fruits and vegetables and with a lower content of meat products and other sources of saturated fats had recorded marked reductions in mortality. He also suggested that a "healthy" diet, low in saturated fats and sodium while rich in plant products, might counterbalance or reduce the negative effects of factors such as stress, environmental pollution or even tobacco smoking. Kesteloot underscored that, if the life span and health status of a population were to keep on improving, behavioural changes in regard to nutrition were necessary.

With regard to the influence of physical exercise in improving survivorship, the evidence was more mixed. Kesteloot noted that, as discussed in the case of mortality differentials by socio-economic status in developed market-economy countries, male manual workers whose level of physical activity was higher than that of non-manual workers had higher levels of mortality than the latter. In that case, dietary and other behavioural factors were probably responsible for the bulk of the differentials observed but, unless such confounding factors were controlled for, linking physical activity to risks of death at the population level was less than straightforward. Furthermore, in most developed market-economy countries physical activity at the population level had been declining just as mortality had been falling, suggesting that physical activity was not a major determinant of a longer life. Yet, studies linking physical activity, especially during leisure time, to all-cause mortality, to mortality due to cardiovascular disease, and to morbidity all indicated that risks of all three were lower among persons who exercised regularly. However, the results of such studies could not be taken as definitive because they failed to control for interactions between different risk factors, interactions that might be large because persons who exercised regularly were a selected group

that was generally health-conscious and tended to have other behaviours that were known to reduce risks of morbidity and mortality. Kesteloot concluded therefore that, whereas the evidence pointing to the important effects that nutritional factors could have on survivorship was strong, the case for the relevance of physical exercise was less so.

Dinesh Sethi and Anthony Zwi considered yet another aspect of behaviour whose influence on the morbidity and mortality of populations was increasing: the frequency of accidents and violent events leading to injury or death. It was expected that, as mortality due to natural causes declined, the relative importance of mortality from external causes (injuries and violence) would rise. By 1990 already about one in every 10 deaths in the world was attributable to such causes. Accidents and injuries accounted for 12.5 per cent of all deaths among males and 7.4 per cent among females. In the developed market-economy countries, homicides caused 6 per cent of all deaths compared to 12 to 13 per cent in Latin America and the Caribbean and in sub-Saharan Africa. In the latter two regions, homicides accounted for one sixth of all male deaths. But the major cause of injuries and death by external causes was road traffic accidents. By 1990 they were estimated to be the ninth most important cause of death worldwide. Between 1968 and 1985, road traffic fatalities (fatalities per 10,000 registered vehicles) increased by 150 per cent in Asian countries and by 300 per cent in African countries, whereas in market-economy European countries they declined by 25 per cent. Such changes took place even though African and Asian countries had lower vehicle densities than developed market-economy countries. Furthermore, the statistics cited did not reflect the major impact that accidents and violence had on morbidity and on health expenditures. In the United States, for instance, US\$ 400 billion were spent in 1993 to treat injured persons, four times the cost of treating cardiovascular disease.

Dinesh and Zwi remarked that, traditionally, injuries had been a neglected public health problem because both authorities and donors tended not to perceive them as amenable to intervention. Although that perception was changing in developed market-economy countries, it was important to stress that most injuries were

preventable and there was a growing body of community-based experience on ways of reducing road traffic accidents, lowering the risk of injury or death when they occurred, and on strategies to prevent or reduce inter-personal violence. Efforts were needed to transmit such information to low and middle-income countries which were only beginning to consider the possibility of intervention. Little was known about the transferability of experience between developed market-economy countries and the developing world. Regional and multi-sectoral exploration of solutions was desirable, especially to establish an exchange of information and approaches among developing countries.

Lastly, Jacob Adetunji discussed another of the growing threats to human well-being: the AIDS epidemic caused by the human immunodeficiency virus (HIV). Because sexual intercourse was the main form of transmission of HIV, behavioural factors were at the root of the epidemic and needed to be addressed if the epidemic was to be brought under control. Adetunji noted that the basic information on the prevalence of HIV infection and AIDS was generally poor and that, consequently, most estimates for the world as whole and for sub-Saharan Africa in particular were derived from models. In 1997, an estimated 22.6 million people in the world were HIV-positive and 14 million (63 per cent) of them lived in sub-Saharan Africa. Between 1992 and 1995, another 1.5 million persons had become infected with HIV in Africa. African countries with the highest prevalence rates of HIV infection included Botswana, Burundi, the Central African Republic, Kenya, Malawi, Rwanda, Uganda, the United Republic of Tanzania, Zambia and Zimbabwe, whose geographical location produced the so-called "AIDS belt of sub-Saharan Africa". As discussed in the session focusing on mortality trends in developing countries, the high prevalence of HIV infection had already had deleterious effects on the survival chances of the population of those countries. Furthermore, because the persons most likely to contract HIV were those in the most productive ages, the epidemic was having a strong negative impact on the human capital of the countries affected. From the social perspective, the AIDS epidemic was undermining the levirate system, whereby a man was obliged to marry his brother's widow, as well as the ten-

dency to adopt or take care of a dead brother's children. The growing number of orphaned children whose parents had died of AIDS was putting undue stress on the extended family. The policy options available to countries with high HIV prevalence rates included promoting low-risk behaviours, such as the use of condoms and the avoidance of sex with multiple partners; reducing the likelihood of transmission by treating and preventing sexually transmitted diseases; providing treatment for co-infections, such as tuberculosis, among those already suffering from HIV infection; and providing some assistance to families with AIDS patients or children left orphan because of AIDS.

The discussion acknowledged the importance of behavioural factors in determining risks of morbidity and mortality, but it emphasized that such factors generally did not operate by themselves. Rather, interactions between the behavioural factors themselves and those with the context in which they were rooted were more important. In Eastern and Central Europe, for instance, there was currently a clustering of unfavourable social and economic factors that favoured high-risk behaviour and increased the risk of morbidity and mortality. Consequently, any intervention aimed at reducing high-risk behaviour would have to take account of the contextual factors that might be promoting such behaviour. To be successful, interventions to change behaviour should be rooted in a firm understanding of the influence that societal forces had on individual actions. Although legislation could be used to influence behavioural change, it had better chances of being successful if its formulation was based on sufficient knowledge about the characteristics of the persons most inclined to adopt risky behaviour, on people's perceptions about what constituted risky behaviour, and on their responses to risk. In the United States, for instance, the effectiveness of anti-smoking legislation depended in large part from the stance taken by non-smokers to prevent their own exposure to passive smoking. The need for political commitment to the reduction of high-risk behaviours was also stressed.

Although several participants noted the potential conflict between individual rights regarding freedom of choice and societal or governmental action to influence individual

behaviour, it was recognized that people required assistance to make informed choices and that, especially in the case of minors, public authorities had the responsibility of preventing their being exposed to harmful influences. Thus, it was appropriate for Governments to prevent tobacco companies from undertaking advertisement campaigns geared mostly to minors. Furthermore, as far as the rights of individuals were concerned, one could raise the issue of whose rights were at stake, since those engaging in high-risk behaviours often subjected to risk persons who did not choose to follow similar behaviours. That was the case of non-smokers subjected to passive smoking or of persons injured by accidents caused by those acting under the influence of alcohol.

Several participants raised issues related to the possible existence of "social causation", that is, of social factors that determined the adoption of different behaviours among distinct population groups. It was thus noted that women tended to engage less in high-risk behaviours than men (women smoke and drank less than men, for instance) and that such "risk avoidance" might be socially determined. Similarly, the fact that the better educated segments of society tended to adopt healthier lifestyles than other groups suggested that socio-economic status influenced behaviour. However, it was not just a matter of access to resources since, in Eastern and Central European countries during the communist period the better educated were not necessarily better-off economically than the rest of society. The processes leading to such social differentiation were not yet sufficiently understood.

Despite such gaps in knowledge, participants stressed that much was known about which interventions were successful in influencing behaviour. In the case of smoking or alcohol consumption, for instance, raising the price of tobacco or alcohol products would almost surely have a dampening effect on their consumption, especially by the less well-off groups in society. It would also make those products less accessible to adolescents. Examples were also cited of campaigns that had been successful in reducing drunken driving or violence within the home. Since it was clear that major improvements in health status and gains in survival could be achieved by reducing the prevalence of smoking



and preventing the abuse of alcohol, there was agreement that action in that front was urgently needed and did not have to wait for better or more information to be available.

#### H. CONCLUDING PANEL DISCUSSION

A concluding panel discussion attempted to summarize the main implications of the deliberations carried out during the Symposium. Panelists stressed the importance of doing comparative research and of disseminating the findings of such research so that countries could learn from the experience of one another. The framework provided by the mortality, epidemiological and health transitions was considered a useful point of reference for such research. However, it was recognized that since general overviews of trends in a group of countries tended to focus on the similarities between them, there was the danger of disregarding the distinctive experiences of particular countries. In Central and Eastern Europe, for instance, the experience of different countries had been far from uniform although the main trait was shared by all: they had all experienced a long hiatus in the transition between high and low mortality. Developing countries might derive some fruitful lessons from such experience. The importance of social conditions in fostering or hindering change was one of them. However, although the full set of mechanisms leading to the stagnation of mortality was not yet well understood, action should not be delayed. Enough was known about high-risk behaviours, such as cigarette smoking, to validate concerted action aimed at modifying them.

It was also recognized that, although general frameworks or theories regarding changes in mortality and morbidity helped to explain overall trends, they often did not fit well the experience of specific countries. Furthermore, trends in period measures might be "reversed" because of changing cohort experience. It was suggested that projections about likely mortality levels should take into account the prevalence of high-risk factors among different cohorts. Thus, the fact that in certain developed countries cigarette smoking was more common among younger cohorts of women than among older cohorts suggested that female mortality might stagnate or even rise in the future. Simi-

larly, in countries where cigarette smoking was increasing among younger adults, whether male or female, mortality declines would be less likely in the future. That was the case of China, where smoking had increased markedly, and it might also affect Japan, where mortality from lung cancer was rising.

The need to consider cohort experience was further validated by current biomedical evidence about the influence that exposure to certain conditions early in life might have on morbidity and mortality at a later stage. In this respect, the distinction between some infectious and chronic diseases was becoming somewhat blurred, as it was found that exposure to certain infectious pathogens could trigger a chronic condition. Similarly, the effects of diet and nutritional status acted over the long-run and, because of changing conditions, might affect differently the various cohorts. Behavioural changes were also likely to be more common among certain cohorts than among others, since the diffusion process would tend to reach peers first. Furthermore, interventions were often more successful if aimed at particular age groups. Children, for instance, should be a key target of programmes aimed at reducing smoking because it was easier to prevent a habit-forming behaviour than to stop it once dependence had taken hold.

Considerable emphasis was put on the scarcity of funds to improve health. In sub-Saharan Africa, for instance, one of the effects of structural adjustment had been the reduction of subsidies for health care and education that had forced many people to do without both, thus limiting their chances to improve their health and living conditions over the long run. In Latin America, the reduction of birth rates was leading to an aging population that was more likely to experience non-communicable diseases. Yet the health systems of countries in the region were not geared to provide long-term care for a larger number of elderly persons. Given the scarcity of resources, priorities needed to be set. However, priorities could not be set on the basis of objective criteria when information on health status and morbidity was mostly lacking. The importance of raising awareness about the urgent need for more data collection efforts and analysis in developing countries was underscored.

It was agreed that a key element to improve health and reduce mortality was the mobilization of social resources by empowering people to opt for behaviours that could reduce the risk of illness and death. Providing education was a major component of such mobilization, since it was a means of ensuring people's commitment to live longer and better lives by adopting health-promoting behaviours. It was also necessary to provide the means of enacting such behaviours and to avoid inconsistent policies.

Proclaiming the deleterious effects of smoking, for instance, while at the same time reducing the price of cigarettes was not likely to reduce smoking.

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NOTE

<sup>1</sup>It should be noted that in this volume, the papers are presented in a slightly different order.

## ANNEXES

### ANNEX I

#### Agenda

1. Opening statements.

2. Election of officers and adoption of the agenda and the organization of work.

3. The mortality, epidemiological and health transitions:

The mortality, epidemiological and health transitions: their relevance for the study of health and mortality.

4. The measurement of adult mortality and mortality by cause of death:

(a) The measurement of adult mortality: an assessment of data availability, data quality and estimation methods;

(b) Causes of death in developing countries and in countries with economies in transition: an overview of current knowledge, data availability and novel methods of data collection;

(c) The epidemiological transition in Mexico: what the data on cause of death reveal.

5. The measurement of health status:

(a) The measurement of health: assessment of various indicators of the overall health status of a population and of the availability of data to estimate them;

(b) The use of surveys to gather information on health status (developing countries);

(c) The measurement of health status in the United States.

6. Health and mortality in developed countries:

(a) Epidemiological transitions in developed countries: past, present and future;

(b) The widening differentials in mortality by socio-economic status and their causes;

(c) The genetic make-up of population and its implications for mortality by cause of death: links between Alzheimer's and ischemic heart disease;

(d) The limits of longevity and their implications for health and mortality in developed countries.

7. Health and mortality in countries with economies in transition:

(a) Health and mortality trends in countries with economies in transition;

(b) The dynamics of mortality in Poland;

(c) The dynamics of mortality in the Russian Federation.

8. Mortality in developing countries:

(a) Mortality in Sub-Saharan Africa;

(b) Mortality in Latin America;

(c) Mortality in Asia, with particular emphasis on China and the Indian sub-continent;

9. Risk factors underlying major causes of death:

(a) Alcohol and smoking as risk factors;

(b) Nutrition, all-cause and cardiovascular mortality: its possible modulation by other factors, especially physical exercise;

(c) Accidents and other injuries;

(d) HIV-infection and AIDS-related deaths in Africa.

10. Panel discussion on prospects for health and mortality in the twenty-first century.

11. Closing session.

## Annex II

### List of participants

#### EXPERTS

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### Annex III

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ESA/POP/1997/SYMP.1/2.1	4	The measurement of adult mortality: an assessment of data availability, data quality and estimation methods Kenneth Hill
ESA/POP/1997/SYMP.1/2.2	4	Causes of death in developing countries and in countries with economies in transition: an overview of current knowledge, data availability and novel methods of data collection Eduardo Arriaga
ESA/POP/1997/SYMP.1/2.3	4	The epidemiological transition in Mexico: what the data on cause of death reveal Rosario Cárdenas
ESA/POP/1997/SYMP.1/3.2	5	The use of surveys to gather information on health status (developing countries) Samir Farid
ESA/POP/1997/SYMP.1/3.3	5	The measurement of health status in the United States Jennifer Madans
ESA/POP/1997/SYMP.1/4.1	6	The limits of longevity and their implications for health and mortality in developed countries Kenneth Manton, Eric Stallard and Larry Corder
ESA/POP/1997/SYMP.1/4.2	6	The widening differentials in mortality by socio-economic status and their causes Tapani Valkonen

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ESA/POP/1997/SYMP.1/4.3	6	The genetic make-up of population and its implications for mortality by cause of death: links between Alzheimer's and ischemic heart disease Douglas Ewbank
ESA/POP/1997/SYMP.1/4.4	6	Epidemiological transitions in developed countries: past, present and future Shiro Horiuchi
ESA/POP/1997/SYMP.1/5.1	7	Health and mortality trends in countries with economies in transition Martin Bobak
ESA/POP/1997/SYMP.1/5.2	7	The dynamics of mortality in Poland Witold Zatoński
ESA/POP/1997/SYMP.1/5.3	7	The dynamics of mortality in the Russian Federation Evgueni Andreev
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# I. THE MORTALITY, EPIDEMIOLOGICAL AND HEALTH TRANSITIONS: THEIR RELEVANCE FOR THE STUDY OF HEALTH AND MORTALITY

*Gigi Santow\**

## A. INTRODUCTION

Many authors of summary accounts of recent developments in human well-being observe that, of all the advances of the twentieth century, the global decline in mortality rates is one of the most impressive. There is considerable justification for such an observation. As I shall describe, levels of mortality at the turn of the century were everywhere higher than they are today, and in many places considerably higher. The gains have been particularly great as they affect infant and child mortality, and nowhere so much as in the West. At the beginning of this century few of today's developed market-economy countries were fortunate enough to enjoy levels of mortality that implied that no more than one in ten newborns would fail to reach their first birthday. Today's leaders in the mortality revolution vie with one another to see how much below the unprecedentedly low ratio of one in one hundred they can go—or even how close they can get to the magical one in two hundred.

Nevertheless, the progress is by no means universal. The populations of many countries do not yet enjoy low mortality, some countries have recently experienced reversals of previous mortality gains, and whatever the overall level of mortality in a society there are pockets—sometimes disturbingly large ones—of comparative or absolute disadvantage. Thus there are laggards, reversals, and inequities.

The task of formulating effective health interventions to combat such situations is both important and difficult. It presents perhaps a different degree of difficulty than in the past because for the vast majority of diseases—acquired immunodeficiency syndrome (AIDS) remains an important exception—there is now at least a technical (either preventative or curative) solution, or partial solution. In the past this was not always the case, and the progressive developments of vaccines and drugs such as antibiotics and new anti-malarials throughout the course of the century have each seemed to hold out the promise of the control or even the eradication of former scourges. In some cases—smallpox is a notable example—the promise has been fulfilled.

The situation today, however, seems to be one of decelerating gains. It has become increasingly plain that many barriers to good health still exist, even though we now have the technical means to dismantle them.

In this brief account I sketch out first the nature of the three “transitions”—the mortality transition, the epidemiological transition, and the health transition—that provide the frameworks within which demographers attempt to explain our past, understand our present, and improve our future. Next, I describe the recent health experiences of countries with developed market economies, of countries with economies in transition, and of the developing countries. Finally, I make some observations concerning the nature of contemporary research approaches, highlight some areas in which a change of emphasis might be rewarding, and identify some directions that might be profitable for future research.

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## B. TRANSITIONS

### 1. *The mortality transition*

The mortality transition and the fertility transition together make up the so-called demographic transition. This term is used to describe the historical process whereby fertility and mortality rates declined from the high and approximately compensating levels that they exhibited in past times to the low and approximately compensating levels that they exhibit in rich countries today. The intervening period generally exhibited higher rates of population growth than occurred in either the earlier or later period because mortality rates tended to fall before fertility rates. The intermediate period has tended therefore to be one of marked population increase. In the earliest formulations of the demographic transition, in the 1940s and 1950s, the driving force behind the declines in both mortality and fertility was identified as modernisation although this term, apparently in the belief that it was self-evident, was not defined. In addition, links were suggested between these declines: people were said to begin to limit their fertility after they recognised that mortality was falling and hence that they did not need to produce so many children in order to ensure a sufficient number of descendants.

With the growth of demography as a discipline and with the proliferation of analytical methods and of demographic data to which to apply them, it has become clear that the reality is far more complex. Mortality decline has not always preceded fertility decline, nor has fertility always declined after mortality did, and both declines have been observed in the absence of social and economic modernisation. Thus, even if demographic transition remains "one of the best-documented generalisations in the social sciences" (Kirk, 1996, p. 361), it is also true that the demographic transition is both "less a theory than a body of observations and explanations" (Caldwell, 1996a, p. 175).

We are perhaps on safer ground when we concentrate on just one of the transitions. In the present case this is the mortality transition, which we define simply as the transition from high to low mortality rates or, equivalently, as the transition from short to long life expectancies. I shall use the term in a purely descriptive

way, carrying no explanation or "theory" of how the transition may have come about.

### 2. *The epidemiological transition*

Although proposed as a theory, the heart of Omran's (1971) epidemiological transition is probably also, like the mortality transition, best characterised as an empirical generalisation. The core proposition is that when mortality is high during "the age of pestilence and famine" (p. 516), causes of death are dominated by infectious and communicable diseases; then, after an intervening "age of receding pandemics", low mortality brings "the age of degenerative and man-made diseases" (p. 517). Analyses of more recent developments in mortality in the United States have led some observers to posit a fourth period, "the age of delayed degenerative diseases", where the age at which degenerative diseases become lethal is postponed to such an extent that life expectancy is propelled into or even beyond the eighth decade of life (Olshansky and Ault, 1986).

While the epidemiological transition is generally described in terms of declining mortality being associated with a shift in the disease burden, one can reverse the description and say that as the burden shifts from infectious to degenerative diseases, mortality falls. This restatement highlights a number of important considerations. One is that death rates from infectious diseases are capable of reaching levels far higher than those ever achieved by degenerative diseases and neoplasms. Although extremely high levels of mortality are not sustainable for extended periods, since survivors of infectious diseases may be better equipped to withstand the next onslaught and since diseases themselves die out if they kill all their hosts, the mortality rates experienced by pre-transitional populations, particularly in years of severe epidemics, are strikingly high. Indeed, one of the features of the epidemiologic transition as well as of the mortality transition is a pronounced reduction in the variability of death rates from year to year (Schofield and Reher, 1991; Fogel, 1997).

Another consideration is that although infectious diseases can be lethal at any age, their effects tend to be particularly virulent among the young. In contrast, the major targets of

degenerative diseases are older people. As a result, reducing the mortality rate from an infectious disease is likely to have a much more pronounced effect on overall mortality than would reducing the mortality rate from a degenerative disease. Given the characteristic shape of the population pyramid, there are more people at risk of death from an infectious than from a degenerative disease. Moreover, the effects of such a disease shift are not confined to mortality rates, since infectious diseases eliminate a proportion of the very young who would otherwise go on to bear their own children, but degenerative diseases affect those who have started, or even completed, their own families. In illustration, Keyfitz (1977) has shown that even when approximately the same number of deaths occur from malaria and from heart disease, eliminating malaria has four times the effect on subsequent population increase as eliminating heart disease.

### 3. *The health transition*

Dissatisfaction with the gains from conventional demographic investigations of mortality characteristics and trends led some ten years ago to the coining of a new term, health transition. The deliberate use of the word "health" signalled a commitment to focus not just narrowly on death and illness but more broadly on the positive condition of health and well-being, an altogether larger condition than survival or even the absence of ill-health. The use of the word "transition" was intended to carry echoes of demographic transition, mortality transition and epidemiological transition. The term "health transition"—"changes over time in a society's health" (Caldwell, 1996b, p. 356)—might seem self-explanatory and indeed it is used by some researchers as a virtual synonym for the mortality transition. Nevertheless, as originally formulated, the term was meant to refer also to the determinants of changing health and, in particular, to its cultural, social and behavioural determinants. Health-transition research therefore does not necessarily have at its core a collection of empirical data (although, this being said, most health transition analysts support at least some of their arguments with standard demographic measures). Rather, the research seeks to explain how cultural, social or behavioural factors have contributed to a particular health situation, whether static or

dynamic, and frequently concentrates on a search for determinants of health improvement that may be susceptible to intervention. The reasons for looking beyond strictly medical and economic conditions are two-fold. First, these two areas have already received a great deal of research attention. Secondly, the policy implications of research that finds a beneficial effect on health of an increase in a country's health budget or in its industrial or tertiary sector is likely to be of limited practical importance in many countries.

A major stimulus to the growth of health-transition research was Caldwell's (1986) article that attempted to explain the mortality transition that had occurred in Sri Lanka, Costa Rica and the Indian state of Kerala (which has a population as large as many countries). Each population remained poor, nor was public expenditure on the provision of medical services such as to preclude emulation by other societies at a similar level of economic development. What these very different populations shared was a respect—indeed a demand—for education, a relatively high degree of female autonomy, a fairly open society, and a tradition of government intervention to improve both health and nutrition. In suggesting the possibility of improved health and lower mortality in societies that have not passed through the economic and social transformations experienced by contemporary low-mortality countries, this and other key pieces of research have directed the attention of a sizeable group of researchers back to the beliefs, attitudes and behaviour of the individuals whose collective experience is ultimately reflected in morbidity and mortality statistics.

## C. REGIONAL PATTERNS OF MORTALITY DECLINE

### 1. *Developed market-economy countries*

Countries grouped together under this heading (the Western-bloc countries of Europe, the United States and Canada, Australia and New Zealand, and Japan) exhibit what Omran (1971) called the "classical or western model" of the epidemiological transition and now enjoy the longest life expectancies ever recorded. Japan, which was Omran's prime example of an "ac-

celerated" epidemiological transition, appears currently to be leading the field, with the most recent official statistics (for 1993) suggesting a life expectancy of 82.51 years for women and 76.25 for men (United Nations, 1996). Apart from Japan, countries with life expectancy at birth for females of 80 years or more include (in descending order) France, Australia, Sweden, Norway, and the Netherlands; those with life expectancies for men of 74 years or more include Sweden, Australia, Norway, the Netherlands, and Singapore (*ibid.*). These figures represent increases in life expectancy of about 25 years over the last century (Preston and Haines, 1991a).

One interesting aspect of the historical mortality transition in the West is that its occurrence was not recognised at the time. Not until the beginning of the present century, for example, was it recognised that mortality rates in Britain were really declining rather than just fluctuating, but we now place the onset of decline a century and a half earlier, in the middle of the eighteenth century (Fogel, 1997). Indeed, by the middle of the eighteenth century mortality was declining not just in Britain but throughout northwestern Europe. Thus life expectancy in Sweden rose from about 37 years in 1780 to around 45 years in 1870 and, the rate of increase accelerating, to around 55 years in 1900, 71 years in 1950 and 78 years today (Keyfitz and Flieger, 1968; United Nations, 1996).

Past levels of infant and child mortality in today's low-mortality countries were brutally high. It has been estimated, for example, that not until the early eighteenth century did fewer than 30 per cent of children of the Genevan bourgeoisie die within the first five years of life, and not until the middle of that century did fewer than 20 per cent die (Hill, 1995). And at the same time in France the comparable proportion dying was close to one-half (Vallin, 1991). In 1780 in Sweden almost one-third of children died before they reached their fifth birthday; this proportion had fallen to about 23 per cent by 1870, about 15 per cent by 1900 and less than three per cent by 1950 (Keyfitz and Flieger, 1968). Today it is well under one per cent (Keyfitz and Flieger, 1990).

The task of explaining the mortality transition in these countries has been complicated by the

discovery, as already mentioned, that twentieth-century mortality decline is a more rapid continuation of a process that had started considerably earlier. Various studies attributed the decline to public-health reforms, advances in medical treatment, improved hygiene and rising standards of living, or a combination of these, but none of these explanations has escaped criticism (Fogel, 1997). McKeown (1976), perhaps the best known of the critics, reacted in particular against claims that what had been most important were advances in medical treatment and improvements in public health. McKeown shows that death rates from respiratory tuberculosis, for example, declined steadily in England and Wales at least from the middle of the nineteenth century, although the bacillus itself was identified only in 1882, effective chemotherapy was introduced only in 1947, and BCG vaccination used on a substantial scale only from 1954. The decline in tuberculosis mortality started a little later in Scotland, Ireland, Denmark, Norway, Sweden, Paris and Massachusetts (Springett, 1950), but in each place still ante-dated by decades the development of effective chemotherapy. McKeown makes similar arguments for other infectious diseases as well, showing that in England and Wales rates of death from pulmonary infections (bronchitis, pneumonia and influenza) and, for children under 15, from whooping cough, measles, scarlet fever and diphtheria were all falling before the causal organism was identified, and certainly before effective treatment or immunisation was introduced. McKeown's alternative suggestion was that the primary determinant of the mortality transition must have been increasing affluence which brought with it improved nutrition, but as Preston and Haines (1991a) and Fogel (1997) have noted, and as McKeown (1978) himself admits, his arguments are both circumstantial and based more on the elimination of other factors that may have been important—through what Schofield and Reher (1991, p. 8) term a "rather liberal use of reductive logic"—than on demonstrations of improving nutrition and of a link between better nutrition and lower mortality.

Perhaps McKeown's greatest contribution has been in stimulating closer examination of this issue. Interesting and persuasive cases have been made for the importance of improvements in water supply and sewerage (Preston and van

de Walle, 1978) and more generally of public-health measures designed to ameliorate living conditions in crowded urban areas (Szreter, 1988), of the control of disease-spreading insects (Riley, 1986), of improvements in housing (Burnett, 1991), and, around the turn of the present century, of improvements in domestic childcare. Preston and Haines (1991a, 1991b), for example, argue that although physicians had few means at their disposal to combat an infectious childhood disease once a child had fallen sick, there were increasingly many precautions that parents could take to ward off sickness in the first place; they observe that the advice given in popular American books about how to protect children from the common childhood diseases became increasingly sensible between the 1890s and 1930s. Finally, the importance of improved nutrition has at last been demonstrated more directly. Fogel (1997), for example, finds that in England and France, outright famines accounted for only a small share of total mortality before 1800. These famines were man-made in the sense that they could have been avoided by the implementation of efficient policies of food distribution. However, what governments could not do at that earlier time was to eliminate pervasive chronic malnutrition. When improvements in nutritional status began to appear, in France and England between the last quarter of the eighteenth century and the third quarter of the nineteenth, they explain about nine-tenths of the mortality decline. They explain only about half of the decline between the third quarter of the nineteenth century and the third quarter of the twentieth. "Only half", however, is an impressive proportion, particularly when one considers the progress that medicine made during this century, especially with regard to the development of antibiotics and of vaccines for major childhood and other diseases.

## 2. *Countries with economies in transition*

The experience of this group of countries (the Eastern-bloc countries of Europe and the successor states of the former USSR) has been somewhat different. Their mortality transition came later than did those of Western Europe and the countries of overseas European settlement. For example, life expectancy at the turn of the century was only 40 years in Bulgaria and Czechoslovakia (Bohemia and Slovakia), 37.5

years in Hungary and a mere 32 years in European Russia. The probabilities of dying within the first five years of life at that time were 29 per cent in Bulgaria and a massive 42 per cent in European Russia, a level scarcely less than that of France in the mid-eighteenth century (Preston and Haines, 1991a; Vallin, 1991). The traditional scourges of famine and pestilence, often associated with political upheaval, remained of significance in some of these countries until well into the present century.

Even if these countries did exhibit mortality decline in the twentieth century, they are probably best described as laggards. In various years between 1950 and 1985, the lowest expectations of life at birth in 26 European countries were recorded by the Eastern-bloc countries and the former USSR (Meslé, 1991, pp. 600-601). Thus, for example, in 1950, when life expectancy for males ranged across Europe from 53 to 70 years, no Eastern-bloc country had a life expectancy of more than 62 years and their median value was 57 years whereas the median for the other countries was 63. In 1970, when life expectancy had risen everywhere and the range had contracted to between 61 and 71 years, no Eastern-bloc country enjoyed a life expectancy exceeding 68 years and their median was 66. The other European countries made continuing gains to 1985, but some of those of the Eastern bloc remained where they were, and others slipped backward. The picture is similar for women, although at higher levels of life expectancy.

A clue to what was happening, at least in a demographic sense, is to be found in an examination of more detailed statistics (Meslé, 1991). For both sexes, infant mortality and mortality at ages 1-14 show continued decline. At ages 15-29, mortality rates for men levelled off in the early 1960s, but those for women continued to decline. At older ages the pattern is one of levelling off or even reversal, more strongly for men than women. In the 1980s in Hungary, the former Czechoslovakia and Romania, the death rates of men of working age in some age groups were actually higher than they had been during the late 1940s or 1950s (that is, in the first life table calculated after the end of the Second World War); in Poland and the former East Germany death rates were much the same for age 50 and older. It seems that gains that were

being made during the 1950s were eroded thereafter or reversed. Hungary provides perhaps the most dramatic case among those discussed by Meslé: there were negligible gains for both men and women after the early 1960s, and adult mortality for men increased after the early 1970s. The Polish picture is similar, although male mortality did not rise above post-war levels in the mid-1980s. The picture was better in the former Yugoslavia, with adult male mortality merely stagnating and female mortality actually showing some improvement.

More recent studies demonstrate not only that mortality is higher in Russia than in the other countries in this group (Meslé and Hertrich, 1997), but that there has been a marked deterioration in the health of Russians since the collapse of the Soviet Union in 1991 (Leon, Chenet, Shkolnikov and others, 1997). Between 1990 and 1994, life expectancy fell by six years for men and three years for women. Between 1984 and 1987, death rates of adults (especially young adults and the middle-aged) from alcohol-related diseases, accidents and violence, pneumonia, other respiratory diseases, circulatory diseases, and infectious and parasitic diseases fell for both men and women; but between 1987 and 1994 death rates rose, and by more than they had previously fallen. Death rates from neoplasms showed no change, from which the authors conclude that the movements in the other death rates are not artefactual.

The agent identified by Leon and colleagues (1997) is alcohol. The period of declining death rates coincides with falling consumption because of increased prices during Gorbachev's anti-alcohol campaign, while the period of rising rates coincides with falling prices and rising consumption. All the causes of death that exhibit an increase in the latter period are consistent, the authors argue, with overconsumption of alcohol, alcoholic poisoning and binge drinking: even the rise in deaths from infectious and parasitic diseases may reflect this association since alcohol depresses the immune function.

Although the Hungarian case attracted attention as early as the late 1980s the underlying causes of the mortality increase are not yet well understood. By that I do not mean that the cause-specific components are not understood:

we know, indeed, that over three-quarters of the male mortality increase between 1964 and 1987 is attributable to an increase in ischaemic heart disease and cerebrovascular disease, and that deaths from cirrhosis of the liver, lung cancer and suicide increased dramatically (Józan, 1991). Rather, I mean that the root causes of these changes are not understood: if alcohol is implicated, for example, then how is it that patterns of consumption have changed in such a lethal way?

A promising approach to the investigation of such questions is provided by Watson's (1995) wide-ranging essay that attempts to uncover the forces that have led to the divergence between the experiences of Eastern and Western Europe. She discounts environmental pollution, and "life-style factors" such as smoking, drinking, and consumption of fats. Instead, she implicates "the hidden injuries of State socialism" whereby the stresses and frustrations of living in a socialist society within a "globalizing" world have adverse emotional and motivational effects, which themselves have implications for health. A strength of Watson's argument is that she shows that in Poland during the 1970s and 1980s, the married, and especially married men, were largely quarantined from rising mortality rates, and that rising mortality was confined to the divorced population. Citing similar results for non-married men in East Germany and Hungary, Watson argues that marriage is particularly protective in these societies because "State socialism... fosters a pattern of fixed coping strategies based on a traditional gender identity which is (within limits) adaptive for women and their families, but ultimately maladaptive for men as men" (p. 932). Men who live outside the physically and emotionally protective shelter of the family may thus be at particular risk. A weakness of Watson's argument, however, is that she does not identify precise pathways to ill health and premature death among these unmarried sub-populations: if not "life-style factors", then what?

### 3. *Developing countries*

A quarter of a century ago Omran (1971) designated the epidemiological transition of the developing countries as "contemporary" or "delayed", but in many instances their subsequent mortality declines could also be termed

“accelerated”: mortality decline may have been delayed relative to that in the West, but in many regions, once begun, it progressed extremely rapidly. Some of the greatest success stories, as well as some of the greatest failures, have been registered in the developing countries. Preston (1995) places average life expectancy at the turn of the century for these countries as a group, at less than 30 years, which is about the level that is assumed to have applied in prehistoric populations, and at least 20 years lower than the level then enjoyed by the countries of the Western bloc. Thus China’s life expectancy was around 24 years in 1930, but it has now reached 69 years; India’s life expectancy has risen from around 25 years during 1901-1911 to 58 years; and Chile’s has risen from 31 years in 1909 to 72 years (Preston, 1995).

A major component of the gain has, of course, been a massive decline in infant and child mortality. Two out of five children failed to survive to their fifth birthday in the China of the 1920s, in Chile before 1920, and in rural Gambia in the 1950s, but all regions of the developing world have since experienced declines in infant and child mortality (Hill, 1995). Africa, south Asia and Latin America exhibited declines in child mortality of at least 100 points between 1950 and 1980 but retained their relative ranking, Africa having the highest mortality of the three and Latin America the lowest. East Asia, which is dominated by the Chinese experience, has managed particularly rapid decline, with higher mortality than Latin America in 1950, similar mortality in the early 1960s, and lower mortality thereafter.

Although no country in sub-Saharan Africa has failed to achieve at least some reduction in infant and child mortality, this region remains the most resistant to intervention. The few apparent successes (Botswana is an obvious example, with the probability of child death falling from around 175 per 1,000 in 1960 to 60 in 1985) are counter-balanced by the experience of a large group of countries that have experienced disappointingly little decline at all. In the former Zaire, for example, the probability of child death, estimated as 285 per 1,000 before 1960, fell only from 235 to 200 between 1975 and 1985 (Ewbank and Gribble, 1993). Of 15 countries for which child-mortality estimates were available in 1985—this region is notorious

not just for high mortality but deficient data—four had child mortality of 200 or more per 1,000, six were in the range 150-199, three were in the range 100-149, and only two fell below 100.

There are several ways to view these levels, and the life expectancies they imply. One way emphasises progress: thus, Preston (1995) observes that Africa’s regional life expectancy of 52 years “would have been the envy of Europe at the turn of the century” (p. 34). Another emphasises regional variation, as I have done above. Yet another might point out that, except for smallpox, for which vaccination was available already in the nineteenth century, Europe exhibited her “African” levels of child mortality when there were neither vaccines to prevent the major childhood diseases nor modern drugs, most importantly antibiotics, with which to combat them. Where child mortality has fallen in sub-Saharan Africa it is largely because of public-health programmes, notably the World Health Organisation’s Expanded Programme on Immunisation (EPI). No parallel activity existed in Europe at the turn of the century.

Vaccines exist for measles, diphtheria, pertussis (whooping cough), tetanus (the latter three being combated with DPT vaccine), and tuberculosis, but these diseases, and especially measles, still remain major killers of Africa’s children (as does tuberculosis of her adults) (Ewbank and Gribble, 1993). Even where immunisation programmes exist they face many problems: vaccines improperly stored; poor coverage; injections given at the wrong age; or the full cycle of injections not completed. For example, to be effective the pertussis vaccine requires that three doses be given, and pregnant women require two injections of tetanus toxoid to protect their new-borns from neonatal tetanus. Moreover, failure to immunise effectively may have wide repercussions. Children who are unprotected against measles may die not only from this cause but also from complications associated with severe diarrhoea or respiratory infections.

Other major killers are diarrhoeal disease and malaria. Treatments exist for both—largely oral rehydration therapy (ORT) for the former, and anti-malarial drugs for the latter—but both

remain serious problems. Oral rehydration therapy is a desperate stopgap measure that does not always manage to compensate for a lack of clean water and clean living conditions; in many places these appear to remain unattainable goals. As for malaria, the spread of chloroquine-resistant strains and the expense and managerial complexity of regimens of prophylaxis and treatment continue to impede the public-health effort (Ewbank and Gribble, 1993).

It would be wrong to give the impression that among the developing regions it is primarily sub-Saharan Africa where these diseases remain to a greater or lesser extent resistant to attempts at intervention, and where reduction of mortality continues to pose a difficult problem. High levels of infant and child mortality still prevail in much of south Asia, especially Bangladesh, and also in Southeast Asia, especially Indonesia.

It would also be wrong to give the impression that the only major problem in these regions is early mortality. Rather, these are regions that suffer not just from high mortality but from poor data, especially on adults. Demographic surveys, as they have developed since the 1970s through the experience of the World Fertility Survey, and from the mid-1980s to the present of the Demographic and Health Surveys, have become increasingly good at measuring the mortality of children from information supplied by their mothers. But in the absence of good systems of vital registration in the vast majority of these countries, or of reliable ways of indirectly estimating adult mortality, we simply do not know enough about the mortality of the adult members of their population. We only know that it is too high. In many countries the evidence for this is that causes of adult death are dominated by infectious and communicable diseases—that people have not had the luxury of surviving such diseases for long enough to fall prey to the degenerative ones.

But this is a gloomy way to conclude the present summary of mortality trends and determinants in developing countries. On a brighter note it must be said that the categorisation of countries as “developing” is rapidly becoming more difficult. A number of regions have exhibited such progress over recent decades in both economic and demographic terms that the

designation seems ill placed. Perhaps the most notable examples are the so-called Asian Tigers—Singapore, South Korea, Taiwan—which have experienced both rapid industrialisation and rapid and convincing declines in both mortality and fertility rates.

This is the first time that fertility, the other component of the demographic transition, has been mentioned. It is perhaps worth observing that sub-Saharan Africa, the region where mortality decline has been the most difficult to achieve, is also the region that has been the most resistant to efforts to reduce fertility.

#### D. FUTURE RESEARCH: DIRECTIONS, EMPHASES AND CONSIDERATIONS

##### 1. *The emphasis on death*

One area of potential improvement in demographic research on health and mortality concerns our preoccupation with death. This focus is easily justified: mortality is one of the principal subjects of demographic investigation and a critically important component of population growth; death is more accurately observed, recorded and reported than any other health-related transition; and the calculation of a life table permits us to assess where a population stands, in terms of its mortality, in relation both to others, and to its own past experience.

All that being said, it is disappointing that despite Omran's emphasis on shifts in the primary causes of death and despite the attempts of Caldwell and others to direct attention to health, there is precious little in the demographic literature on either. Enormous advances in the collection of demographic data, originally pioneered by the World Fertility Survey and now represented by the endeavour of the Demographic and Health Surveys, have greatly improved our ability to quantify the incidence of infant and child death. Charting the levels and trends in infant and child mortality and identifying differentials was amongst the greatest contributions of the World Fertility Survey (WFS) (Chidambaram, McDonald and Bracher, 1987). The Demographic and Health Surveys have extended the WFS approach by collecting information on immunisation and recent episodes of illness among the young children of



survey respondents. Nevertheless, sufficiently accurate ascertainment of causes of death continues to elude the survey-taker, despite attempts to develop such techniques as the verbal autopsy. Reliable information on morbidity remains notoriously difficult to collect, certainly in a census (as was optimistically attempted in the Indonesian Census of 1980), and also in a retrospective survey. In the bygone age of more generous funding for demographic research one might have called at this point for more prospective studies, but in the present economic climate their expense would seem to render such a call impractical.

Thus it seems that we must make do with the information we already have, and perhaps do better with it. In countries with efficient censuses and systems of vital (and perhaps other) registration, one feasible avenue is to link cause-specific deaths with population classified according not only to sex and age but also to such characteristics as occupation (Vågerö and Lundberg, 1995), employment history (Valonen and Martikainen, 1995) or marital status (Goldman, Takahashi and Hu, 1995). There are particular gains if the linking can be done at the individual level, as in the first two of these studies, but as the third study demonstrates, this capability is not essential. Alternatively, there are the small-scale, intensive, cost-effective studies of the sort reported in the pages of *Health Transition Review* or *Social Science and Medicine*. Such studies tend to be poor at quantification but rich in throwing up hypotheses or in providing interpretational material that throws light on the findings of larger-scale studies, and are to be recommended for those reasons.

One reason for regretting the emphasis we place on death is that it draws attention away from the quality of life. Riley (1989) inclines to the view that as life expectancy has increased, so has morbidity, that the transition between 1850 and 1900 can usefully be seen as a transition from an age of death to an age of sickness, and that this trend has only been exaggerated in the twentieth century. When mortality rates were high, he argues, so were rates of falling sick, but most sicknesses were resolved quickly and morbidity rates were comparatively low: to paraphrase Riley (1989, p. 156), the sicknesses of people who die do not show that ill health

prevails among the people who survive. Thereafter, however, the rise in life expectancy was accompanied by a rise in rates of ill health and in public and private spending on health. In some sense, his argument is a gloss on Omran's (1971) old formulation, with the subduing of infectious diseases leading not to the disappearance of ill health but to its becoming chronic and degenerative. It has particular and growing relevance at the older ages, a point to which I return in a moment, when I consider the current emphasis on old-age survival.

Another reason to regret the almost exclusive focus on death is that it ignores the process leading to death. The rate of death from a particular cause may vary both because of variation in the rate at which people fall ill, and because of variation in the rate at which they succumb, and it may be extremely important to distinguish between the two. Since this opposition is essentially that between a preventative solution and a curative one, distinguishing which has had the greater influence over the death rate is critically important in designing an effective health policy.

A final, and related reason for wishing to move beyond the emphasis on the dichotomous outcome of death versus survival is that there is mounting evidence that adverse conditions in childhood may be reflected decades later in elevated rates of adult mortality (Elo and Preston, 1992; Fogel, 1997). Research on this connection is still in its infancy, but if even some of the postulated linkages can be demonstrated then it becomes clear that a simple reduction of infant and child mortality, without a corresponding reduction in infant and child morbidity, gives less cause for congratulation than one might initially think.

## 2. *The emphasis on the extreme ages*

### *Infants and children*

Infant, and to a lesser extent child, mortality attracts more attention from demographers than mortality at any other ages. One reason is that infant mortality is easier to measure than adult mortality, but of course this is not the whole story. Rather, we pay particular attention to early mortality because in past times and in poor countries today, especially those of sub-Saharan

Africa, there is simply so much of it; absolute levels are high, and in high-fertility countries with a large population base, such deaths are a large proportion of all deaths. Moreover, in countries where the mortality transition is underway or essentially complete a decline in early mortality is its most dramatic feature, and has the most immediate impact on age structure, population growth, and life expectancy. Note, for example, that eliminating France's horrendous mid-eighteenth century probability of child death of 0.474 would have instantly raised the expectation of life at birth from 24 to 45 years (Vallin, 1991).

Nevertheless, it is not clear that the demographer's emphasis is justified in every situation. Despite the fact that the developed market-economy countries now enjoy levels of infant mortality that fall clearly below the previously unimaginable threshold of ten per thousand—a level less than one-tenth that of the success stories of sub-Saharan Africa—considerable amounts of research money are devoted to investigating how it is that some infant deaths (formerly "cot deaths", now "sudden infant deaths") do not have an obvious cause. Such deaths may represent as many as one-quarter of infant deaths or one-half of post-neonatal deaths, but their occurrence would not even register in the mortality experience of developing countries. Perhaps it is simply that in the West we have been led to expect that our babies will not die, and we extrapolate from this to societies elsewhere. One begins to suspect even that very low infant mortality is interpreted as some sort of status symbol. This interpretation is borne out by the concern for levels of infant mortality shown by demographers in our second group of countries, those with economies in transition. In summarising the main features of Hungary's Health Promotion Programme, for example, Józán (1991) lists in his text only three items, and the first two deal respectively with infant mortality and the prevalence of low birth-weight babies. Yet, Hungary has been experiencing a crisis of adult mortality, not of infant mortality (which stands at around two per cent), and it is surprising that that is not given more particular emphasis.

Perhaps one reason for our particular stress on infant mortality is that this is an area that is now particularly amenable to intervention. Even if

implementation remains a problem we now have the technical solutions to eliminate a great deal of infant mortality. One of the best illustrations is the reduction in infant mortality that has been achieved in the so-called Fourth World, which is made up of indigenous peoples "submerged by an invading society" (Kunitz, 1994, p. 22). Such populations, whether American or Canadian Indian, aboriginal Australian, New Zealand Maori, Polynesian or Micronesian, now exhibit levels of infant mortality that in comparison with their levels of adult mortality are unnaturally low. Despite the often deplorable living conditions of many infants, which is reflected in growth faltering and frequent episodes of sickness, their levels of infant and child mortality are held down to levels only several times higher than those of the surrounding population by means of aggressive programmes of maternal care, child immunisation, and repeated hospitalisations to combat respiratory infections and bouts of diarrhoea. Thus patched up, the infants are returned to their own communities until their next bout of ill health.

Nevertheless, while such intervention is demonstrably successful for the very young, there is now an epidemic of adult mortality among these populations. Major contributors are suicide, violence, and diseases of the circulatory system, the relative importance of each of these varying according to the particular sub-population under examination. Whether or not elevated rates of diseases of the circulatory system are related to nutritional insults during the developmental stage, a possibility mentioned earlier, it seems clear that public-health services have been much more successful at dealing with the infectious diseases than the non-infectious and man-made ones (Kunitz, 1994).

### *The aged*

The demography of ageing has become a sub-field in its own right especially in the developed market-economy countries, where old-age mortality rates continue only to decline, and where the biggest gains in life expectancy are being made at the oldest ages (Olshansky and Ault, 1986). A major stimulus of such work has been a well-founded concern for the solvency of pension funds and health-insurance systems if,

given concomitant fertility decline, an ever-increasing proportion of their users are both heavy users, and no longer active contributors. Another stimulus has undoubtedly been simple wonderment that over so few generations it has been possible to increase the human life span to such an extent.

Neither of these considerations should be allowed to obscure the major consideration that survival is not necessarily an unalloyed good. Rather, what we should be aiming for is survival in reasonable health so that the capacity to enjoy life, however restricted it may inevitably be, still remains. This is a far more difficult quest than the rectangularisation of the survival curve, but deserves attention no less.

Responses to the increasing longevity of the populations of the industrialised countries are varied. In a characteristically pessimistic mode, Smith (1995, p. 28) believes that the intractability to prevention and palliation of "terrible disablers like mental illness and arthritis... leaves health policy vulnerable to political decisions dictated by small claimant groups, and protagonists of expensive machines and techniques and screening surveys whose value for money and impacts on the general wellbeing are dubious". On a more sanguine note, Riley (1989, p. 248) concludes that "If the bad news is that our society faces a mounting aggregate burden of ill health, the good news is that this is the outcome of a long and only recently successful campaign to extend survivorship, and that the campaign has begun to be won at a time when material prosperity is great enough to afford the financial costs of ill health". The sad fact, however, is that whatever the truth of this statement, it applies only to the developed market-economy countries. Extended old-age survival is a problem to which the developing countries can merely aspire.

### 3. *Learning from the past*

Although it is probably harder to attract funding for research into population history than for any other area of demography, fine examples of historical work are sufficiently numerous to justify our continuing attention. In some cases, such as Fogel's work on nutrition and mortality, historical data are actually superior to data from contemporary developing

countries, and the research findings have important consequences for future population developments in developing countries as well as in the industrialised countries. In other cases, parallels can be drawn between the determinants of mortality in past times and those in the developing countries today. A fine example, following the seminal work of Peter Aaby, is Burström's (1996) demonstration of the importance of crowding for measles mortality, not just in contemporary Kenya but in nineteenth-century Stockholm, as well.

Other cases illustrate the dangers of extrapolating automatically from the developed-country past to the developing country present, but in so doing raise issues of great importance that might otherwise have remained hidden. One example of this is the discovery that, unlike the almost universally observed experience of the contemporary developing countries, child mortality in the United States at the beginning of the present century exhibited no sharp gradient according to maternal education (Preston and Haines, 1991a). This discovery, and the ensuing discussion, raise issues concerning not only McKeeown's original hypothesis but also the differences between the public-health environments of turn-of-the-century America and the developing world today, between the meaning and significance of education, between the interpretation of illness and health, and between the roles and capabilities of the health systems (Caldwell, 1991; Preston and Haines, 1991b). One hopes such work will be enabled to continue.

### 4. *Risk factors, differentials and inequality*

Whether we write about "risk factors", as do the epidemiologists, or about "differentials", as is more common among demographers, or about "inequality", as is becoming especially common among researchers investigating population health in the developed market-economy countries, following the lead of the World Health Organization, we are actually all engaged in a task that is central to demography, namely, comparison. Indeed, the very task I was set of describing the recent evolution of mortality in the developed market-economy countries, the countries with economies in transition, and the developing countries, is one of comparison. If adult mortality had not fallen so far in the first

group of countries, and had not recently risen in the second, then we would not be talking about the latter's crisis of adult mortality; had infant mortality not fallen so definitively not only in the first group of countries but also in the second, and in many of the developing countries too, then we would not be deploring the stagnation of infant mortality in the primarily sub-Saharan-African remainder.

Whatever our vocabulary, the primary reason for making comparisons is that we wish to identify groups with unacceptably high mortality, and from this identification to be able to devise ways in which this apparently excess mortality might be reduced. Especially in the context of infant survival, the search for high-risk groups used to be justified on the basis that particular attention could be focused on individuals with characteristics identified as life-threatening—for example, the low birth-weight children of mothers with deficient education, without access to a clean water supply, and with a previous history of child loss and rapid child-bearing. Research that has identified such differentials has been valuable in itself, although it is doubtful, given the managerial demands of such an intervention system, that any developing-country health system has been capable of such focused action. Rather, such research has highlighted particular areas that should be amenable to intervention; birth intervals can be lengthened by the use of contraception, with an added benefit to maternal health; better water can be provided by digging tube wells.

Before we can make comparisons, however, we must have something to compare, and hence our first task is one of quantification and description. Such work is exemplified by the body of excellent quantitative analyses by Meslé and her colleagues on mortality trends in the countries with economies in transition. The most recent example was the paper that introduced the lively session devoted to this topic in the recent General Population Conference of the International Union for the Scientific Study of Population (Meslé and Hertrich, 1997). The paper's task was to "anatomise" recent developments, and it attempts no explanations. Indeed, it is difficult to see how explanation could be attempted from age, sex and cause-specific mortality statistics alone.

One notable omission from that conference session was any mention, in any of the published papers, oral presentations, or discussion from the floor, of the possible role of marital status. Yet this may be fertile ground for exploration if Watson's (1995) demonstration that rising death rates in Poland, Hungary and the former East Germany were largely confined to the unmarried population has broader applicability throughout the region. Goldman and her colleagues have documented in a series of important articles that the married tend to have lower mortality rates than the single and the previously married. They also address the question of how this may come about, through a combination of selection (those who marry are the healthy) and protection (those who are married live healthier lives) (Goldman, Takahashi and Hu, 1995; Hu and Goldman, 1990).

If the supposition of a link with marital status turns out to be correct, then we shall have opened up a whole new area for investigation. Questions that may reward attention include the roles of declining rates of marriage and marital stability in the overall mortality shift; of changing health-related behaviours among the unmarried, especially men; and of unemployment. In an analysis of the effect of unemployment on mortality in Finland, Valkonen and Martikainen (1995, p. 221) conclude that the effect mainly operates not causally but through selection, and that although unemployment is a serious social problem that acts to the detriment of individual well-being, "it may be only very exceptionally a catastrophe that kills". The case of Eastern Europe may be just such an exception. Another question concerns the movement in marital-status and cause-specific death rates; suicide, for example, is far more common among the non-married, especially the divorced, than among the married (Ruzicka, 1995).

A better understanding of the causes of the mortality reversal is likely to lead to more effective intervention, if the political will exists. It should certainly allow us to do better than merely to recommend, as we could have done without prior statistical analysis, that alcohol abuse be prevented, smoking discouraged, and a healthy diet promoted (Kromhout, Bloemberg and Doornbos, 1997). After all, although Gorbachev's anti-alcohol campaign is widely credited with successfully reducing Russian death

rates during the mid-1980s, subsequent developments have demonstrated just how little momentum this reduction had: once alcohol became cheaper and more available, death rates rose. Indeed, it may be that a broad programme of health education may increase existing health differentials since it is likely to have more effect on people who are already in a comparatively advantageous position than on people who are marginalised. Valkonen (1992) believes, for example, that well-educated Finns have benefited most from efforts to improve public health, and unskilled workers the least, or not at all.

Whether research is motivated by a desire to understand the determinants of elevated mortality, or to remove social inequalities that are correlated with poor health, the identification of heterogeneity remains a primary goal. Nevertheless, such heterogeneity must be defined on more than demographic characteristics: if married people have lower mortality than others, then how is this brought about? Neither does the identification of a social correlate of elevated mortality necessarily mean that we are much closer to real understanding: if the better educated have lower mortality than other people, and are more successful at raising their children to adulthood, then how does this come about? I return to the issue of the interpretation of heterogeneity in the following, concluding section.

### 5. *One approach or three?*

In discussing what we mean by the mortality transition, the epidemiological transition, and the health transition we are led naturally to question whether the approaches they imply are best viewed as distinct or as complementary. The question is important because whether we view past and contemporary developments in population health, ill-health and mortality within the framework of the mortality transition, or within that of the epidemiological transition, or that of the health transition, may lead to rather different types of analysis, and hence to rather different conclusions. The framework of the mortality transition is likely to emphasise the identification of trends in mortality rates, of differences between rates that obtain in different populations, or of heterogeneity in rates within a population. That of the epidemiological transition is likely to stress the contributions of

shifts in causes of death to changes in overall mortality, or even to concentrate solely on the evolution of rates of death according to a particular cause. Finally, the health-transition approach is likely to focus more closely on the role of individual agency, and to broaden the field of investigation from death, disease or ill-health to beliefs about health and illness, attitudes to modern preventative and curative health services, and social or cultural barriers or enabling factors that variously inhibit or promote the use of such services.

Despite the fact that, as I have described them, the three approaches appear to be distinct, it can be difficult to state categorically which one of the three is best suited to the investigation of a particular research issue. Take, for example, work on within-population heterogeneity in the risk of death. Well before the advocacy of the health-transition approach, researchers had recognised that mortality rates varied according to socio-economic characteristics: of the individuals themselves in the case of adult mortality, or of their parents in the case of infants and children. Indeed, even Omran (1971, p. 527), whose description of the epidemiological transition is usually interpreted as referring merely to a shift in causes of death as mortality rates fall, proposed that the shifts in patterns of health and disease that characterise the epidemiological transition are "closely associated with the demographic and socio-economic transition that constitute the modernisation complex". More recently, in their investigation of differentials in child mortality in 15 developing countries Mensch, Lentzner and Preston (1985, p. 286) conclude that "membership in a social group that is more advantaged or more modern is associated with lower child mortality, whether the variable of interest is education, income, urban residence, housing type, father's occupation, or one of the many other variables examined". Many of the characteristics that were identified as associated with lower child mortality (the ones mentioned above, with particular attention paid to the distinction between the effects of paternal and maternal education, as well as ethnicity, and access to and use of health services) have been the focus of subsequent research that can probably best be described as falling within the health-transition framework. The difficulty is that a large-scale demographic sample survey or

census is a good vehicle through which to identify the existence of heterogeneity in the chance of survival, but it is ill-suited to explaining how such heterogeneity has been created. Conversely, a smaller investigation that employs a variety of data-collection techniques, probably involving considerable direct participation by the principal investigators, is a good vehicle through which to understand the immediate determinants of individual health-seeking behaviour, but is ill-suited to quantifying the extent to which different forms of individual behaviour lead to different health outcomes.

For people who would wish to apply research findings to health policy this distinction is of more than academic significance. A study of the former type, involving quantitative analysis of a large-scale demographic survey, perhaps identifies maternal schooling as an important correlate of child survival, and reports that ten years of schooling is associated with an average reduction of child mortality of one-third. But what is the policy implication? A huge injection of (possibly scarce) government funds into mass education, especially for girls? Conversely, although a study of the latter type, involving close analysis of the pathways that individuals follow in the quest for health, can result in no such dramatic statistic, it may reveal something about how maternal education operates to improve child survival. It may tell us, for example, whether what is most important is having grown up in a family that bothered to send its daughters to school, or the actual experience of having gone to school, or the confidence instilled by the experience of schooling, or the content of what was taught, or simple literacy. The implications for health interventions of each of these would be very different.

It is likely, then, that the studies that are going to be the most revealing and certainly the most useful to health policy makers are those that combine the different approaches (see Cleland and van Ginneken, 1988). Moreover, while most research that explicitly falls under the health-transition rubric is concerned with health in the developing world, the quest for a better understanding of the behavioural and social determinants of health is a worthwhile endeavour in any population, whatever its mortality statistics. An obvious candidate for such inves-

tigation is that of the mortality reversal in the countries with economies in transition. Another candidate is that of the widening gap between the death rates of manual and non-manual workers such as observed in Sweden, a country where it was believed until recently that social-class differentials in mortality probably no longer existed (Vågerö and Lundberg, 1995).

Quantification of the extent of a health problem is critically important for the allocation of resources to combat it, and so is quantification of the extent to which the problem can be reduced by particular interventions. In certain applications it will be important also to focus not just on death, but on death from particular causes and morbidity from those causes: after all, the prevention of death from one cause does not mean that death has been definitively prevented but merely that it has been delayed, the individual then becoming at risk of dying from something else. Finally, unless we have information from closer observation of study participants than is usual in a standard social or demographic survey, it is possible that we shall not be sufficiently equipped to formulate an intervention that is likely to succeed. All we shall have done is to identify a problem and quantify its extent. These are critical first steps, but do not automatically carry within themselves the germ of a solution.

#### REFERENCES

- Burnett, John (1991). Housing and the decline of mortality. In *The Decline of Mortality in Europe*, R. Schofield, D. Reher and A. Bideau, eds. Oxford: Clarendon Press, pp. 158-176.
- Burström, Bo (1996). Risk Factors for Measles Mortality: Studies from Kenya and 19th century Stockholm. Doctoral thesis. Sundbyberg: Department of Public Health Sciences, Karolinska Institutet.
- Caldwell, John C. (1986). Routes to low mortality in poor countries. *Population and Development Review*, vol. 12, pp. 171-220.
- \_\_\_\_\_. (1991). Major new evidence on health transition and its interpretation. *Health Transition Review*, vol. 1, pp. 221-229.
- \_\_\_\_\_. (1996a). Demographic transition. In *The Social Science Encyclopedia*, Adam Kuper and Jessica Kuper, eds. London: Routledge, pp. 174-175.
- \_\_\_\_\_. (1996b). Health transition. In *The Social Science Encyclopedia*, Adam Kuper and Jessica Kuper, eds. London: Routledge, pp. 357-358.
- Chidambaram, V. C., John W. McDonald and Michael D. Bracher (1987). Childhood mortality. In *The World Fertility Survey: An Assessment*, John Cleland and Chris Scott, eds. Oxford: Oxford University Press, pp. 862-881.

- Cleland, John G., and Jerome K. van Ginneken (1988). Maternal education and child survival in developing countries: The search for pathways of influence. *Social Science and Medicine*, vol. 27, pp. 1357-1368.
- Elo, Irma T., and Samuel H. Preston (1992). Effects of early-life conditions on adult mortality: A review. *Population Index*, vol. 58, pp. 186-212.
- Ewbank, Douglas C., and James N. Gribble, eds. (1993). *Effects of Health Programs on Child Mortality in Sub-Saharan Africa*. Washington, D.C.: National Academy Press.
- Fogel, Robert William (1997). New findings on secular trends in nutrition and mortality: Some implications for population theory. In *Handbook of Population and Family Economics*, vol. 1A, Mark R. Rozenzweig and Oded Stark, eds. Amsterdam: Elsevier, pp. 434-481.
- Goldman, Noreen, Shigesato Takahashi and Yuanreng Hu (1995). Mortality among Japanese singles: A re-investigation. *Population Studies*, vol. 49, pp. 227-239.
- Hill, Kenneth (1995). The decline of childhood mortality. In *The State of Humanity*, Julian L. Simon, ed. Oxford: Blackwell, pp. 37-50.
- Hu, Yuanreng, and Noreen Goldman (1990). Mortality differentials by marital status: An international comparison. *Demography*, vol. 27, pp. 233-250.
- Józán, Péter (1991). Changes in Hungarian mortality and the role of the national health promotion program. In *Future Demographic Trends in Europe and North America. What Can we Assume Today?*, Wolfgang Lutz, ed. London: Academic Press, pp. 55-69.
- Keyfitz, Nathan (1977). *Applied Mathematical Demography*. New York: Wiley.
- \_\_\_\_\_, and Wilhelm Flieger (1968). *World Population: An Analysis of Vital Data*. Chicago: University of Chicago Press.
- \_\_\_\_\_, (1990). *World Population Growth and Aging. Demographic Trends in the Late Twentieth Century*. Chicago: University of Chicago Press.
- Kirk, Dudley (1996). Demographic transition theory. *Population Studies*, vol. 50, pp. 361-387.
- Kromhout, Daan, Bennie Bloemberg, and Gerda Doornbos (1997). Reversibility of rise in Russian mortality rates. *Lancet*, vol. 350, No. 9075, pp. 379-380.
- Kunitz, Stephen J. (1994). *Disease and Social Diversity: The European impact on the health of non-Europeans*. Oxford: Oxford University Press.
- Leon, David A., Laurent Chenet, Vladimir M. Shkolnikov and others (1997). Huge variation in Russian mortality rates 1984-94: Artefact, alcohol, or what? *Lancet*, vol. 350, No. 9075, pp. 383-388.
- McKeown, Thomas (1976). *The Modern Rise of Population*. London: Edward Arnold.
- \_\_\_\_\_, (1978). Fertility, mortality and causes of death. An examination of issues related to the modern rise of population. *Population Studies*, vol. 32, pp. 535-542.
- Mensch, Barbara, Harold Lentzner and Samuel Preston (1985). *Socio-economic Differentials in Child Mortality in Developing Countries*. New York: United Nations. Sales No. E.85.XIII.7.
- Meslé, France (1991). La mortalité dans les pays d'Europe de l'Est. *Population*, vol. 46, pp. 599-648.
- \_\_\_\_\_, and Véronique Hertrich (1997). Évolution de la mortalité en Europe: La divergence s'accroît entre l'Est et l'Ouest. *International Population Conference Beijing*, vol. 2, pp. 479-508. Liège: International Union for the Scientific Study of Population.
- Olshansky, S. J., and A. B. Ault (1986). The fourth stage of the epidemiologic transition: The age of delayed degenerative diseases. *The Milbank Quarterly*, vol. 64, pp. 355-391.
- Omran, Abdel R. (1971). The epidemiologic transition: A theory of the epidemiology of population change. *Milbank Memorial Fund Quarterly*, vol. 49, pp. 509-538.
- Preston, Samuel H. (1995). Human mortality throughout history and prehistory. In *The State of Humanity*, Julian L. Simon, ed. Oxford: Blackwell, pp. 30-35.
- \_\_\_\_\_, and Michael R. Haines (1991a). *Fatal Years. Child Mortality in Late Nineteenth-Century America*. Princeton: Princeton University Press.
- \_\_\_\_\_, and Michael R. Haines (1991b). Response to comments on Fatal Years. *Health Transition Review*, vol. 1, pp. 240-244.
- Preston, Samuel H., and Etienne van de Walle (1978). Urban French mortality in the nineteenth century. *Population Studies*, vol. 32, pp. 275-297.
- Riley, James C. (1986). Insects and the European mortality decline. *The American Historical Review*, vol. 91, pp. 833-858.
- \_\_\_\_\_, (1989). *Sickness, Recovery and Death: A History and Forecast of Ill Health*. Iowa City: University of Iowa Press.
- Ruzicka, Lado T. (1995). Suicide mortality in developed countries. In *Adult Mortality in Developed Countries: From description to explanation*, Alan Lopez, Graziella Caselli, and Tapani Valkonen, eds. Oxford: Clarendon Press, pp. 83-110.
- Schofield, Roger, and David Reher (1991). The decline of mortality in Europe. In *The Decline of Mortality in Europe*, R. Schofield, D. Reher and A. Bideau, eds. Oxford: Clarendon Press, pp. 1-17.
- Smith, F. Barry (1995). The first health transition in Australia 1880-1910: An exploratory essay. Paper presented at The John C. Caldwell Seminar on the Continuing Demographic Transition, organised by the National Centre for Epidemiology and Population Health, The Australian National University, Canberra, 14-17 August.
- Springett, V. H. (1950). A comparative study of tuberculosis mortality rates. *The Journal of Hygiene*, vol. 48, pp. 361-395.
- Szreter, Simon (1988). The importance of social intervention in Britain's mortality decline c.1850-1914: A re-interpretation of the role of public health. *Social History of Medicine*, vol. 1, pp. 1-37.
- United Nations (1996). *1994 Demographic Yearbook*. Sales No. E/F.96.XIII.1.
- Vågerö, Denny, and Olle Lundberg (1995). Socio-economic mortality differentials among adults in Sweden. In *Adult Mortality in Developed Countries: From description to explanation*, Alan Lopez, Graziella Caselli, and Tapani Valkonen, eds. Oxford: Clarendon Press, pp. 223-242.
- Valkonen, Tapani (1992). Trends in regional and socio-economic mortality differentials in Finland. *International Journal of Health Sciences*, vol. 3, pp. 157-166.
- \_\_\_\_\_, and Pekka Martikainen (1995). The association between unemployment and mortality: Causation or selection? In *Adult Mortality in Developed Countries: From description to explanation*, Alan Lopez, Graziella Caselli, and Tapani Valkonen, eds. Oxford: Clarendon Press, pp. 201-222.
- Vallin, Jacques (1991). Mortality in Europe from 1720 to 1914: Long-term trends and changes in patterns by age and sex. In *The Decline of Mortality in Europe*, R. Schofield, D. Reher and A. Bideau, eds. Oxford: Clarendon Press, pp. 38-67.
- Watson, Peggy (1995). Explaining rising mortality among men in Eastern Europe. *Social Science and Medicine*, vol. 41, pp. 923-934.

## II. EPIDEMIOLOGICAL TRANSITIONS IN HUMAN HISTORY

Shiro Horiuchi\*

### A. INTRODUCTION

Many aspects of human life have changed entirely from prehistoric, tribal societies to today's global high-technology world. Probably our genetic characteristics have not changed substantially, but the length of human life has. The expectation of life at birth has increased from about 20 years for early man (Acsadi and Nemeskeri, 1970; Howell, 1979; Kaplan, 1997; Preston, 1995) to nearly 80 years in the countries with the lowest mortality levels today.

Underlying this spectacular fall in the *level* of mortality were fundamental shifts in the *pattern* of mortality. The path from 20 years to 80 years of life, and to an even longer life expectancy in the future, can be viewed as a sequence of mortality regimes, each of which has a distinct cause-of-death profile. A shift between two different regimes may be called an "epidemiological transition."

Originally, the concept of "epidemiological transition" was proposed to indicate a particular set of changes in the pattern of mortality and morbidity (Omran, 1971). In this paper, however, the term will be defined more broadly and Omran's epidemiological transition will be considered the second in time among five epidemiological transitions in the human history. Since epidemiology is the study of distribution of diseases, injuries, and their risk factors, an "epidemiological transi-

tion" means a long-term change in the overall distribution of diseases, injuries, and their risk factors. In particular, the focus of this paper will be on transitions that significantly alter the distribution of deaths by cause and/or by age.

Major cause-of-death categories used in this paper are "infectious diseases," "degenerative diseases," and "external injuries," together with two main subcategories of degenerative diseases, "cardiovascular diseases" and "cancers." The first three categories essentially represent the three "level-one categories" in the Global Burden of Diseases (GBD) study (Murray and Lopez, 1997): Group 1 includes infectious and parasitic diseases, maternal and perinatal disorders, and nutritional deficiencies; Group 2 comprises non-communicable diseases; and Group 3 consists of injuries, poisoning, toxic effects, and other external causes, whether intentional or accidental.

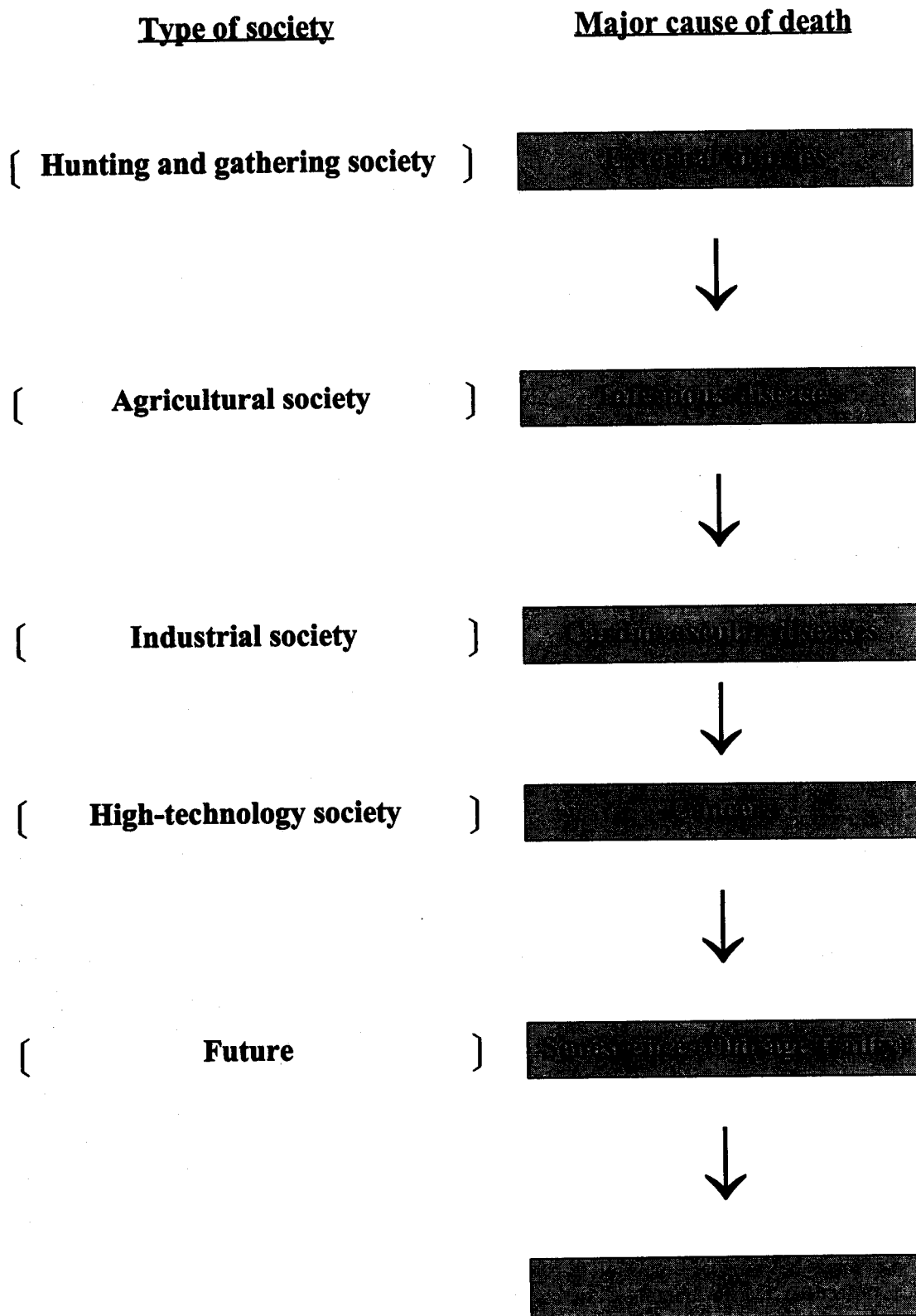
This paper presents a conceptual framework, in which historical changes of the human mortality pattern in the past and future are summarised as five epidemiological transitions. Three transitions occurred in the past: from external injuries to infectious diseases, from infectious diseases to degenerative diseases, and most recently, the decline of cardiovascular disease mortality. Two more transitions are expected to take place in the near future: the decline of cancer mortality, and slowing of senescence. The typical order of these transitions is shown in figure 1. (Note that the "major cause of death" in figure 1 does not necessarily mean the most frequent cause of death in the population, but rather indicates a cause of death that is considerably more prevalent in the era than in the other eras. Thus the cause of death *characterizes* the mortality pattern of the society).

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Figure 1. Epidemiological transitions in the human history



## B. EPIDEMIOLOGICAL TRANSITIONS

### 1. *First transition: external injuries to infectious diseases*

A number of communicable diseases were endemic or occasionally became epidemic in pre-industrial agricultural societies. Many of them, however, were rare or less serious in small, isolated tribes of early humans, who engaged mainly in hunting and gathering (Black, 1980). It is suspected that a high proportion of the early human population died from external injuries, comprising various kinds of accidents and homicide (Lancaster, 1990, chapter 1; also see Brothwell, 1967). Causes of death that are rare now but probably were not uncommon in those days include attacks by carnivores, drowning, intertribal war (Keeley, 1997), and infanticide. Natural disasters, starvation, and complications of pregnancy and childbirth are also considered to have been prevalent causes of death.

Agriculture started some 10,000 to 12,000 years ago. It diffused widely in the period between 8,000 BC and 4,000 BC and was accompanied by various changes in human life, including diet, dwelling and habitat, social structures, and population size (Cohen, 1995, chapter 3). A fundamental shift in the pattern of health and mortality took place as well (Austad, 1997, chapter 3). The change in the major means of survival from hunting and gathering to agriculture reduced risks of various accidents and risks of homicide by other animals. However, some characteristics of agricultural societies made it easier for various pathogens to remain continuously or diffuse effectively in human populations. These factors include greater population size of communities, higher population density in broad geographical areas that have multiple communities, longer periods of residence at the same locations (often with domesticated animals), storage of foods, domestication of some animals, and extended contacts with other communities. The emergence of urban communities must have amplified these risks. In addition, nutritional deficiencies due to decreased dietary diversity in agricultural societies might have made humans more vulnerable to infections than in hunting-gathering tribes. Infectious and parasitic diseases had become the dominant cause of human mortality. This

pattern of mortality then predominated until the latter part of the present millennium.

### 2. *Second transition: infectious diseases to degenerative diseases*

Limited historical data on mortality in the eighteenth century suggest that the expectation of life at birth in European countries was usually in the range of about 25 to 40 years, with substantial regional variations. By the mid-twentieth century, the life expectancy for all European countries combined reached the neighbourhood of 65 years. In particular, the gain in the life expectancy during the first half of the twentieth century was spectacular (United Nations Secretariat, 1962, table III.4).

This gain was largely due to reduction in mortality from infectious and parasitic diseases, as well as maternal, perinatal, and nutritional disorders (Omran, 1971; Preston, 1976; United Nations Secretariat, 1962, chapter 5). In France, the age-standardised death rate due to infectious and parasitic diseases (most notably, tuberculosis) declined between 1925 and 1955<sup>1</sup> by 79 per cent for males and 87 per cent for females, and the age-standardised death rate due to complications of pregnancy and childbirth fell by 71 per cent for females (Vallin and Meslé, 1988).<sup>2</sup>

This transition can be divided into two phases. The first phase is the reduction of crisis mortality. Demographic crises occurred irregularly but not necessarily infrequently in the nineteenth century and before. The mortality level rose suddenly due to disasters such as harvest failures or pandemics. In Europe, episodes of the crisis mortality became less frequent toward the late nineteenth century (Perrenoud, 1991). This reduction might not have had significant impacts on the mortality level in ordinary (non-crisis) years, but lowered the average level of mortality for relatively long periods. The Spanish Influenza epidemic of 1918 may be the last episode of the traditional type of mortality crisis in European countries.

In the second phase, the mortality level of regular years showed a gradual decline. This shift occurred mainly in the second half of the nineteenth century and the first half of the twentieth century, although the exact timing

varied greatly among countries. As discussed later, this phase can be split further into two periods that have different age patterns of mortality reduction. The reason for the secular mortality decline was multi-factorial. Some researchers emphasise the impact of the improved standard of living, in particular, nutrition (Fogel, 1994; Fogel and Costa, 1997; McKeown, 1979), whereas others focus on the importance of public health measures and personal hygiene (Morel, 1991; Preston, 1990; Preston and van de Walle, 1978).

The reduction of infectious disease mortality shifted major causes of death from infectious to degenerative diseases including heart diseases, strokes, cancers, diabetes mellitus, chronic liver diseases, and chronic kidney diseases. Generally, death rates due to these degenerative diseases rise steeply with age.

The age-standardised death rate due to all cardiovascular diseases combined declined in France between 1925 and 1955 by 27 per cent for males and 34 per cent for females. This decrease was significantly slower than the reduction of infectious disease mortality. As for neoplasms, the death rate increased by 52 per cent for males, and decreased slightly, only by 4 per cent, for females in the same period. (These cancer mortality trends may be partly attributable to improved diagnosis and partly to increased cigarette consumption.) The growing proportion of population at older ages also helps to increase the proportion of all deaths that are attributable to degenerative diseases.

### *3. Third transition: decline of cardiovascular disease mortality*

By the mid-twentieth century, considerable reduction in infectious disease mortality, in particular, among children and adults at reproductive ages, had been achieved. On the other hand, the relative reduction of mortality at old ages was modest. This gave rise to a view that, although mortality at young and middle ages can be reduced significantly, mortality at old ages cannot, because senescence is the inevitable fate of every human organism. It was argued that since mortality rates at young and middle-ages had already fallen to very low levels, the potential of human life prolongation had largely been exhausted. Thus, life

expectancy was close to its upper limit (Bourgeois-Pichat, 1978; Fries, 1980; Gavrilov and Gavrilova, 1991).

An unexpected breakthrough occurred, however, in the late twentieth century. Economically developed countries entered a "new stage of epidemiological transition" (Olshansky and Ault, 1986; Rogers and Hackenberg, 1987). The transition is characterised by reduction of mortality due to degenerative diseases, in particular, cardiovascular diseases. The decline of cardiovascular disease mortality was generally slow in the 1950s and 1960s, but accelerated in the 1970s (Uemura and Pisa, 1988). Total (all-cause) death rates at oldest ages (80 and over), which had previously been stagnant (probably for centuries and possibly for millenniums), also commenced a considerable decline. The onset of the oldest-age mortality reduction occurred between the late 1950s and the early 1980s, earlier for females than males (Kannisto, 1994, 1996; Kannisto and others, 1994).

This was not only due to an extension of life of the morbid or disabled elderly, but also due to improvements of health conditions of the elderly. The increase of life expectancy in the United States during the 1980s was mainly due to longer years of life without disability (Crimmins, Saito, and Ingegneri, 1997). The prevalence of disability decreased among the elderly in the United States in the 1980s and the early 1990s (Manton, Corder, and Stallard, 1997).

Reasons for this health improvement are not limited to technological advancements in curative medicine. Publicly supported health care systems have developed. Hypertension became less prevalent partly because of anti-hypertensive drugs but partly because greater availability of fresh foods and diffusion of refrigerators changed diet patterns and reduced salt intake. Regular medical check-ups help detect hypertension and high cholesterol level in early stages. Other relevant health trends with long-term impacts include vitamin D supplementation, thermal inactivation of atherogenic viruses in commercial food processing, and diminished prevalence of virulent streptococcal strains that cause chronic rheumatic heart diseases (Manton, Stallard, and Corder, 1997).

For most developed countries, the period of the second epidemiological transition and that of the third epidemiological transition are fairly distinguishable. In Sweden, the cut-off point of the two transitions was in the late 1950s (Horiuchi and Wilmoth, 1998). In the United States, the second transition virtually ended around 1954 and the third transition started around 1968. During the inter-transition period of 1954-1968, the mortality decline was stalled (Crimmins, 1981). In contrast, the timing of the two epidemiological transitions heavily overlapped in Japan after the Second World War, thereby raising the life expectancy very rapidly (Horiuchi and Wilmoth, 1998). Similar patterns of two *simultaneous* epidemiological transitions probably characterise the fast mortality reductions in the newly industrialised countries in Eastern and Southeastern Asia during the recent decades.

#### 4. Fourth transition: decline of cancer mortality

How far can the length of human life be extended in the next century? Will it soon face the practical limit of increase, perhaps around 85 years of the life expectancy, or will it reach 90 years and even approach 100 years? The prospect varies, depending on theories and interpretations of empirical evidence (Manton and Stallard, 1996; Manton, Stallard, and Tolley, 1991; Olshansky and Carnes, 1996; Olshansky, Carnes, and Cassel, 1990; Vaupel, 1997; Wilmoth, 1998). For life expectancy to be lengthened significantly in the future, a continuation of the current transition may not be sufficient. New types of epidemiological transitions need to take place. Cancer's turn should be next.

In contrast to the decline in mortality due to cardiovascular diseases, mortality due to cancers did not show a substantial reduction in developed countries during the last few decades. The trend varies considerably by the site of cancer, among countries, and between sexes (Coleman and others, 1993). For some types of cancers, incidence trends and mortality trends exhibit notable discrepancies, and period data and cohort data show different aspects of cancer trends. In general, downward trends seem dominant for cancers of the

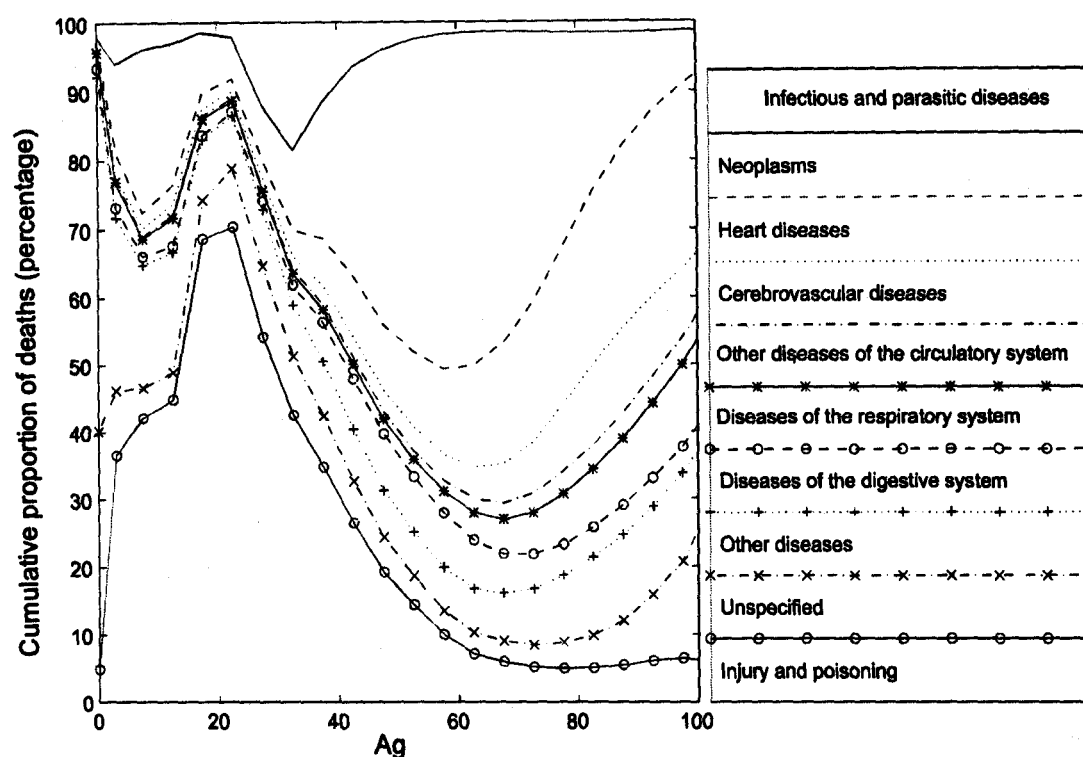
colon, rectum, bone, cervix uteri, testis, bladder, and thyroid, as well as Hodgkin's disease. The reduction of stomach cancer has contributed significantly to prolongation of the life expectancy. However, trends in cancers of the larynx, lip, tongue, oesophagus, pancreas, and ovary, are either flat, unclear, or divergent among countries. Upward trends prevail among cancers of the mouth, pharynx, skin (melanoma), breast, prostate, and kidney, as well as Non-Hodgkin's lymphoma and multiple myeloma. Lung cancer mortality is still on the rise in many countries, although the trend appears to be changing in countries where smoking is becoming less prevalent.

When causes of death are grouped into very broad categories, cancer is the second most prevalent cause of death, next to heart disease, in the majority of developed countries, and the dominant cause of death in several developed countries. Furthermore, cancer deaths are common at *younger* old ages, as shown later (figure 2). Thus, if the reduction of cancer mortality is substantial, so will be the increase of life expectancy.

A long-awaited decline in total cancer mortality has finally started around 1990 in Canada (McLaughlin and others, 1997), the United States (Cole and Rodu, 1996) and the European Union (Levi and others, 1997). The downward trend is notable for both cancers that can be caused by smoking and cancers that are not closely related to smoking. Whether this is a temporary phenomenon or marks the beginning of a long-term decline remains to be seen.

Breakthroughs may occur when the progress in basic research on cancer (for example, studies on such subjects as telomere shortening, angiogenesis around tumor cells, DNA damage, and oncogenes and tumor suppresser genes), which is greatly enhancing our understanding of the cancer mechanisms on the cellular and molecular levels, leads to new medical technologies. The challenge of cancers, however, seems biologically more fundamental than the challenge of cardiovascular diseases. Although coronary heart diseases are peculiar to humans and rare in other animals, cancers at old ages are found widely among mammalian species.

Figure 2. Age-related changes in the distribution of death by cause in France, both sexes combined, 1990-1994



Source: Institut National d'Études Démographiques mortality database

### 5. Fifth transition: slowing of senescence

What will become the most frequent causes of death if we succeed in reducing both mortality from cardiovascular diseases and from cancers to fairly low levels? The list of likely candidates includes acute pneumonia and bronchitis, influenza, acute gastro-enteritis, and congestive heart failure. Deaths that are difficult to classify by specific medical cause may also increase. Such deaths will occur mostly at very old ages.

The distribution of deaths by cause varies with age for both sexes in France, 1990-1994, as shown in figure 2. The age pattern helps us to reflect on major causes of death after the third and fourth epidemiological transitions. Around age 20, some 70 per cent of deaths are due to external injuries (including poisoning) in accidents, homicide, and suicide. The relatively high proportion of deaths due to infectious diseases in the late 20's and 30's suggests substantial impacts of acquired immuno-

deficiency syndrome (AIDS) on mortality in this age group. Cancers dominate the "younger old ages". Nearly half of deaths in the 50's and 60's are due to neoplasms. The most frequent cause shifts during the 70's from cancers to cardiovascular diseases, which dominate "older old ages." Indeed, it has been observed that cancers are quite rare among centenarians (Allard and others, 1996). More than 40 per cent of deaths in the 80's and 90's are attributed to cardiovascular diseases.<sup>3</sup>

Figure 2 indicates that the proportion of deaths without specifically identified causes, including "senility without mention of psychosis" (797 in ICD9), increases steeply with age from 3 per cent in 65-69 to 14 per cent in 95-99. This may be partly due to the tendency for persons at older ages to die from multiple diseases, which makes it difficult to select the primary cause of death. However, it also seems to indicate that a number of deaths at very old ages may occur without clear mani-

festations of any specific diseases. In a review of autopsy findings for two-hundred persons above age 85, no specific cause of death could be identified for more than 30 per cent of the cases (Kohn, 1982). It should be noted that deaths without identifiable causes in autopsy are not necessarily "natural" deaths without diseases. Schneider and Brody (1983) argue that for even a very old person to die there must be "a specific pathologic insult". At very old ages, the insult could be too small for the physician to detect.

Figure 2 also shows that the proportion of deaths due to diseases of the respiratory system rises steeply with age, from 4 per cent in 60-64 to 12 per cent in 95-99. Included in this category are pneumonia, bronchitis, and influenza. This is consistent with the finding of a recent study on age-patterns of cause-specific mortality (Horiuchi and Wilmoth, 1997). The study revealed that although death rates due to most diseases rise with age, the pattern of age-related increase differs considerably between diseases. Three major types of age pattern are identified. First, for most cancers, the death rate rises with age very steeply in the middle years, but slows down markedly at older ages. Second, death rates due to ischemic heart disease and infarctive stroke tend to increase with age at relatively constant high rates, though the rises eventually slow down at very old ages. Third, death rates due to some diseases rise slowly in the middle years, but markedly accelerate at later ages. These diseases include pneumonia, bronchitis, influenza, gastro-enteritis, and heart failure. The increased importance of these diseases is consistent with the observation that the hospitalisation cost declines with age after the peak in the 70s (Perls and Wood, 1996). Treatment of these diseases is generally less expensive than that of coronary heart disease or cancer. At the oldest ages, the rate of relative mortality increase with age ("life table ageing rate") is higher for these diseases than most others.<sup>4</sup>

Deaths at very old ages due to these diseases or without specified causes may be considered direct manifestations of senescence, or the "state of non-specific vulnerability" of old age (Gavrilov and Gavrilova, 1991). Persons with favourable endowments and healthy life styles have better chances to survive the risks of atherosclerosis, hypertension, and malignancy. However, as "normal ageing" proceeds, some

immune functions deteriorate, thereby making the persons more vulnerable to infections that they would control easily at younger ages (Miller, 1995). The ability to pump the sufficient amount of blood out of the heart weakens with age, leading to congestive heart failure. Many other physiological functions also deteriorate with age (Masoro, 1995). If these declines result in a death without clear manifestation of any disease, the cause of the death is difficult to specify.

Can the senescent processes be slowed and deaths due to "old-age frailty" be delayed? Although some data suggest health conditions of the elderly have markedly improved in recent decades, this transition is considered to be in a very early stage, if it has indeed even started. The proportional mortality decline during the last few decades has been substantial at old ages in general, but relatively small in the 90s. For ages 75 and above, there has been a notable tendency for the decline to be smaller at older ages (Kannisto, 1996, figure 5; Wilmoth, 1997, figure 3.9).

Nevertheless, there are at least three reasons for optimism. First, the slowing-down may be achieved, though probably to a more limited extent, by adopting and keeping healthy life styles. There is much direct and indirect epidemiological evidence that exercise, a low-calorie and low-fat diet that includes a variety of vegetables and fruits, no smoking and moderate drinking, avoidance of excessive mental stress, and active participation in social interactions help to slow the age-related physiological deterioration and/or keep the person free of diseases and disabilities of old ages. Second, benefits are expected from further development, wider availability, and more efficient use of medical technologies for preventing incidence and progression of degenerative diseases (Butler and Brody, 1995). Thirdly, gerontological research is exploring the fundamental biological mechanisms of senescence (for example, free-radical damages, replicative senescence, cross-linking of collagen, and senescence-related genes). Findings of the research may eventually lead to entirely new types of medical technologies that slow senescent processes (Banks and Fossil, 1997; Miller, 1997).

In the traditional evolutionary biology of ageing, it has been theorised that genes that

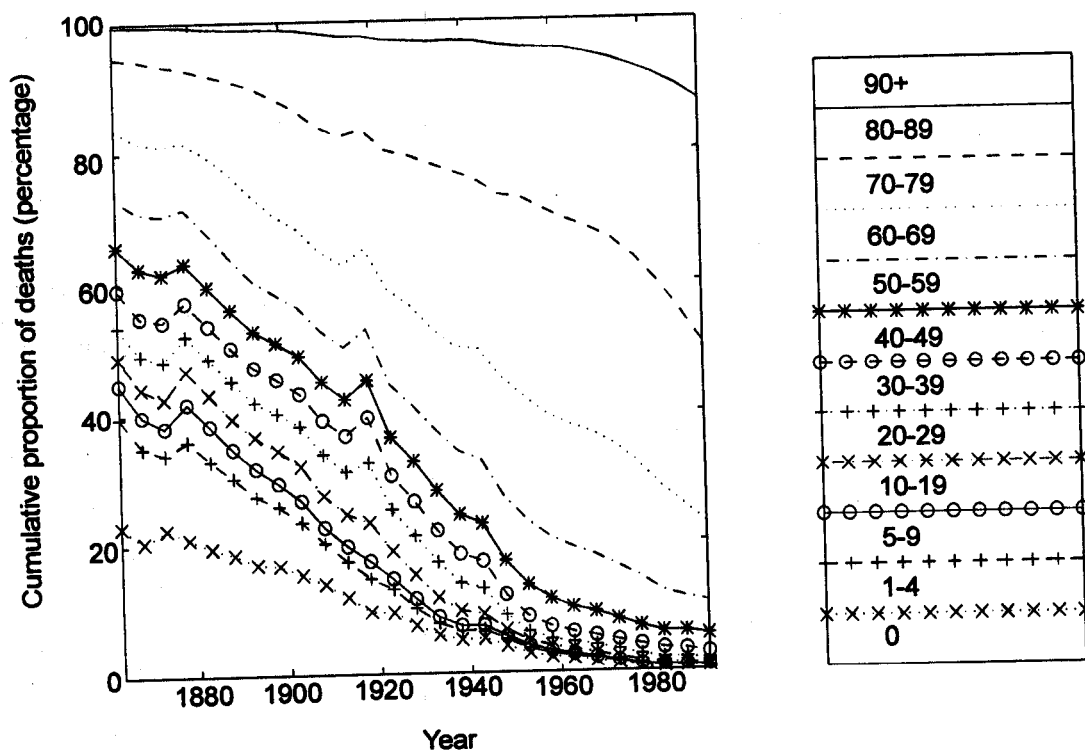
have deleterious effects at old ages may not be eliminated by the force of natural selection (Medawar, 1952), particularly if those genes contribute to reproductive success at younger ages (Williams, 1957). These theories imply the inevitability of senescence. However, recent laboratory studies using nematodes, fruit flies, and rodents show that the length of life of these animals can be extended significantly by experimental manipulations, including caloric restriction, genetic intervention, and temporary exposure to non-lethal levels of radiation, heat, and toxic substance. These results may indicate that in the course of evolution, animals have developed certain mechanisms to raise the ability to resist stress and/or repair damages under special circumstances (Masoro and Austad, 1996; Johnson, Lithgow, and Murakami, 1996; Richardson and Pahlavani, 1994). These mechanisms are

inactive in the usual life course, but are activated by some stimuli in the above-mentioned experimental manipulations. Although these laboratory results are not directly applicable to humans, this emerging theory suggests that senescent processes in the human organism may be slowed by activating a latent capacity that is already present in the human body.

### C. DEMOGRAPHIC PATTERNS OF THE EPIDEMIOLOGICAL TRANSITIONS

The mortality decline due to the second and third epidemiological transitions shifted the age distribution of deaths to older ages. The reduction in the proportion of children due to fertility decline added to this shift. Figure 3 shows that the age composition of deaths in Sweden changed markedly between 1861 and

Figure 3. Trends in the age distribution of deaths, Sweden, both sexes combined, 1861-199



Source: University of California, Berkeley, mortality database.

1995. In 1861-1865, nearly half of all deaths occurred at ages under 20, but in 1991-1995, almost half of all deaths were at ages 80 and over.

The age pattern of mortality decline differs markedly between the second and third epidemiological transitions (Wilmoth, 1997, figure 3.9). This is reflected in the trends of life expectancies at different ages, as shown in figure 4. The expectation of life at birth in Sweden increased progressively from about 46 years in 1861-1865 to 81 years in 1991-1995 for females and 44 years to 76 years in the same period for males, with the exception of the sudden fall due to the pandemic of Spanish Influenza in 1918 (figure 4A). Life expectancies at other ages, however, did not necessarily follow similar trajectories.

For the following analysis, it is useful to consider the temporary expectation of life, which is the number of years between any two ages,  $x$  and  $y$ , that are expected to lived by a person at exact age  $x$ . Its maximum value is  $x - y$ .

Mortality of young children continued to fall, as reflected in the trend of temporary life expectancy between age 0 and 10, as shown in figure 4B. The apparent deceleration of the rate of increase in the second half of the twentieth century is simply due to the fact that age-specific mortality rates of young children were approaching zero, leaving little room for further significant improvements.

The trend of the temporary life expectancy between age 10 and 60 (figure 4C) was very different from that between age 0 and 10. It increased slowly in the late nineteenth century and early twentieth century, then rose very steeply during the second quarter of the twentieth century, reflecting the substantial reduction of tuberculosis mortality among young adults. After the mid-century, the progress slowed down. The temporary life expectancy between age 10 and 60 even decreased slightly for males in the 1960s and 1970s.

The expectation of life at age 60 followed another, entirely different trend (figure 4D). It was relatively constant, around 14 years for females and 13 years for males, during the first half of the twentieth century. In females, it suddenly began to increase in the 1950s.

The steep rise continued during the second half of the twentieth century, the era of the third epidemiological transition. Now it exceeds 19 years. In contrast, the life expectancy of males at age 60 remained nearly constant around 14 years during the 1950s, 1960s, and 1970s, then began to ascend notably in the 1980s.

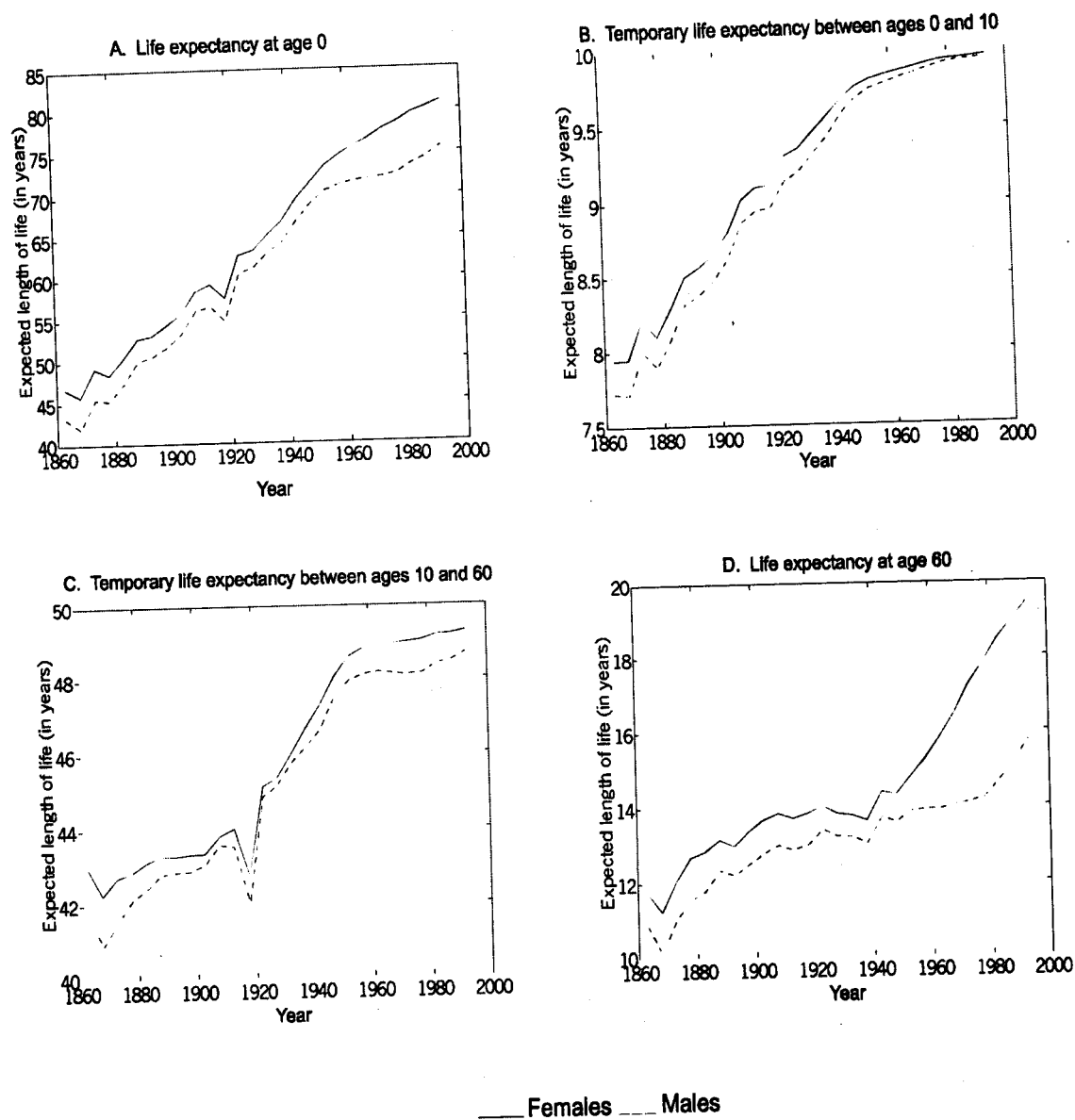
In summary, the differential time trends of age-specific life expectancies in Sweden suggest that: (1) in early stages of the second epidemiologic transition, mortality reduction was pronounced for infants and young children, but not necessarily for young and old adults; (2) in later stages of the second transition, the mortality level decreased substantially among adults at reproductive ages, mainly due to the reduction of tuberculosis mortality; (3) in the third epidemiological transition, a marked mortality decline was seen among the elderly, mainly due to the reduction of cardiovascular disease mortality, though with a notable difference between females and males.

Why did this sex differential emerge? International time-series data show that the excess of male over female mortality has increased substantially in the twentieth century. The sex difference (female minus male) in the expectation of life at birth in developed countries was typically about 2 to 3 years around the beginning of the century, but 5 to 8 years in recent decades (United Nations Secretariat, 1988). The discrepancy expanded rapidly after 1960, in particular, during the 1970s (Vallin, 1983). Figure 5 indicates that sex ratios (male over female) of age-specific death rates in Sweden increased steeply between 1931-1935 and 1981-1985.

Life table ageing rate analysis reveals that the age pattern of old-age mortality differs considerably between males and females in industrialised countries during the late twentieth century (Horiuchi, 1997, figures 1-3). In the model schedules of old aged mortality for industrialised countries constructed by Himes, Preston and Condran (1994), notable differences are found between the male and female patterns. Swedish data indicate, however, that females and males had similar age patterns of mortality in the nineteenth century. The sex difference evolved as the age pattern of male mortality changed appreciably (Horiuchi and Wilmoth, 1998, figure 4D).

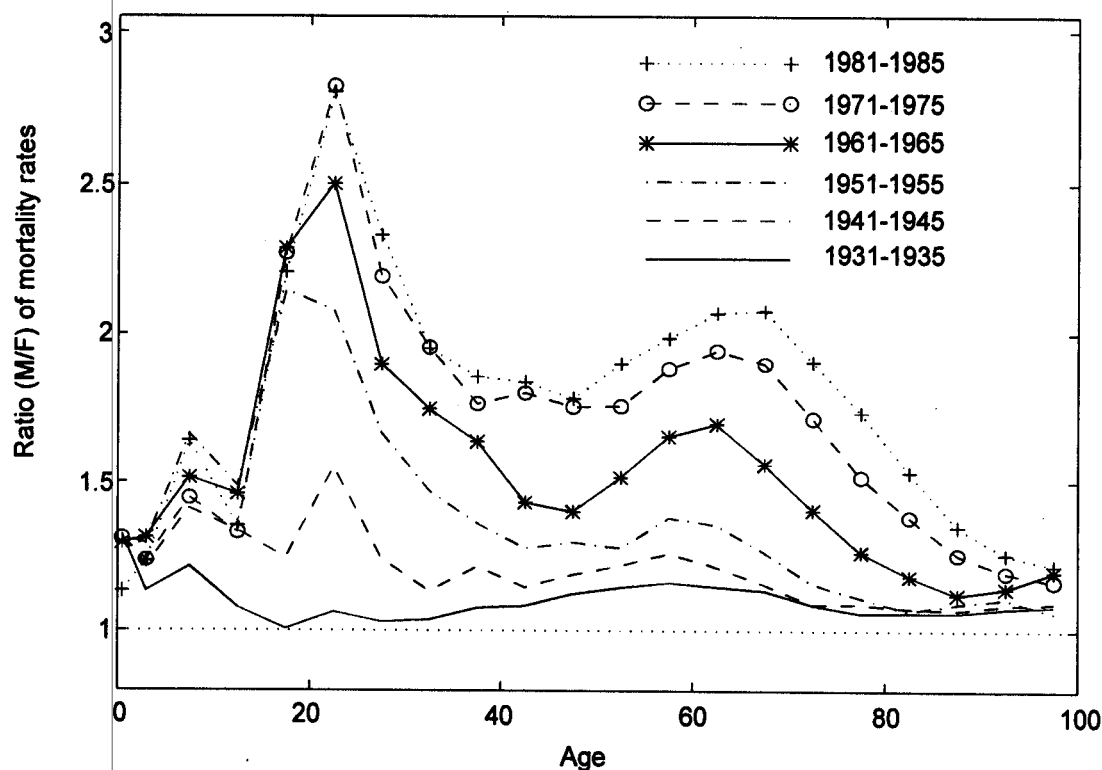


Figure 4. Trends in the expectation of life in Sweden, for five-year periods from 1861-1865 to 1991-1995



Source: University of California, Berkeley, mortality database.

Figure 5. Trends in the sex ratio (male/female) of age-specific death rate, Sweden, selected year



Source: University of California, Berkeley, mortality database.

The contribution of specific causes of death to the increasing sex mortality differential varies among countries, but typically the divergence is due mainly to cardiovascular diseases and cancers (most notably, lung cancers), and to lesser extents, liver cirrhosis and diabetes mellitus, as well as external injuries (Lopez, 1983; United Nations Secretariat, 1988; Wingard and Cohn, 1990). Mortality trends by sex, age, and cause, together with epidemiological data on sex differences in risk factors, suggest that a major reason for the increasing sex mortality differentials is less healthy life styles (in particular, smoking, and possibly to a lesser extent excessive drinking) of males than those of females (Lopez, 1983; Waldron, 1985, 1986).

#### D. REVERSE TRANSITIONS

The epidemiological transitions, which generally lower total mortality, may meet counteracting trends that keep the mortality level from declining or even raise it. Such health trends are referred to as "reverse transitions." Two major reverse transitions in the recent history and three potential reverse transitions in the near future merit attention.

##### 1. *Early stages of the industrial revolution*

In the early stages of industrialisation, the poor and sometimes dangerous working conditions of factories and mines and the low standard of living among industrial workers

posed new types of health risks (Szreter, 1997). Industrialisation induced further urbanisation and expanded urban slums, which were not necessarily followed by measures to deal with the health problems arising there (Wrigley, 1969, chapter 5). "The positive results which were achieved during the second half of the eighteenth and the beginning of the nineteenth century in reducing the mortality from traditional diseases were cancelled by the rise in mortality from certain diseases associated with the laissez-faire policies of the industrial revolution" (Caselli, 1991). In France, the crude death rate declined appreciably between 1750 and 1845, but the decline stagnated in the following 40 years, the most intense period of early industrialisation (Valin, 1991).

Although the spectacular decline of tuberculosis mortality in the twentieth century was a major component of the second epidemiologic transition, data from Sweden and Finland suggest that death rates from tuberculosis were relatively low in the eighteenth century and then increased markedly in the nineteenth century (Puranen, 1991). Tuberculosis mortality among adults increased in Norway in the late nineteenth century, and geographical variations of tuberculosis mortality in Sweden during the nineteenth century indicate its strong linkage with urbanisation (Lancaster, 1990).

## 2. *Unhealthy life styles in the wealthy society*

Economic development in the twentieth century improved the health conditions of the population through various direct and indirect pathways. The increased productivity and the wider and faster distribution of products, however, had some adverse "side effects" as well. Alcoholic beverages and cigarettes are produced in large amounts, distributed widely, and affordable to most people. This makes excessive drinking and habitual smoking possible. The reduction of intense manual labour encourages more sedentary life styles. The high-calorie and high-fat diet that was previously a luxury, has become common. A number of epidemiological studies report that these factors raise risks of a variety of degenerative diseases (for example, Butler and Snowdon, 1996; Cockerham, 1997).

Among the adverse health effects of the modern life styles, the most evident and most detrimental seem to be those of cigarette smoking. It is estimated that 24 per cent of all male deaths and 7 per cent of female deaths in developed countries during the year of 1990 are due to smoking (Peto and others, 1994). Lung cancer, which was relatively rare in the 1930s, has now become one of the leading causes of death. In developed countries, the age-standardised death rate from lung cancer has risen by 170 per cent on average between 1950-1954 and 1988-1990 for men and by 230 per cent for women (Lopez, 1995). Currently, tobacco consumption per adult is declining in developing countries, but increasing rapidly in many developing countries (World Health Organization, 1997).

## 3. *Emergence and re-emergence of infectious diseases*

The reduction of mortality due to infectious diseases has lengthened human life expectancy considerably. Recent decades, however, witnessed increasing episodes of sudden and unexpected new infectious diseases (Levine and Thacker, 1996; U.S. Center for Disease Control and Prevention, 1994). The most threatening was AIDS; other recent examples include hantavirus pulmonary syndrome, Lyme disease, Legionnaire's disease, haemorrhagic colitis, haemolytic uremic syndrome, and Ebola haemorrhagic fever. In addition, some old infectious diseases, including tuberculosis and malaria, have become more virulent, more prevalent, or less controllable.

There are a number of reasons for this emergence or re-emergence of infectious diseases (Morse, 1995; Olshansky and others, 1997). First, a number of infectious diseases were once highly prevalent but then brought under control by effective drugs. For some of these old diseases, new strains of the pathogens that are resistant to those drugs have evolved (Levy, 1998). Some vectors of these pathogens also have developed resistance to drugs for controlling them (typically, insecticides).

Second, technological developments and socio-economic changes make it possible for infectious diseases to spread more quickly than before. Some infectious diseases, if they

had emerged previously, would have been contained in a local area for many decades or would have expanded their reach only gradually. However, now they are able to become global epidemics in a relatively short period of time. The major reason for the more rapid disease diffusion is the increased, faster, and wider range of trade, tourism, and migration. More frequent sexual contacts between persons from diverse geographical areas as well as some medical technologies (for example, blood transfusion) may contribute to the disease diffusion.

Thirdly, such rising rates of disease transmission may help the pathogens to evolve to more virulent forms (Ewald, 1994). Only rapidly diffusing diseases can be deadly and still continue to expand at the same time. (Otherwise, the host population of the virulent disease will diminish.)

Lastly, ecological disruptions accompanying economic developments may help new diseases to appear. Economic activities in wild areas (for example, forestry in tropical rain forests) expose humans to the risk of contact with pathogens that are rare outside the areas. Construction of roads, bridges, dams, etc. may transform the geographical distribution of animals that carry infective agents and parasites, thereby creating new chances of their close contacts with humans.

Thus, the recent emergence and re-emergence of infectious diseases are rooted in the interactions among technological advancement, economic development, and biological evolution. In this sense, AIDS may not be an isolated episode of unforeseeable disaster. The risk of a second AIDS-type outbreak seems worthy of serious consideration. Whether this new trend will significantly reverse the second epidemiological transition remains to be seen.

#### *4. Pollution*

Pollutants from industry, agriculture, transportation, and household activities may contaminate air, water, and soil, and accumulate in edible animals and plants. In houses and other buildings, humans may be exposed to radon, lead, asbestos, and possibly toxic materials in pesticides, paints, and other chemicals used for building maintenance (Ott and Rob-

erts, 1998). The depletion of atmospheric ozone ("ozone hole") increases the danger of exposure to intense ultraviolet radiation.

Effects of pollution on mortality have been documented in a number of situations: examples include some seriously polluted areas such as Minamata and Chernobyl (Harada, 1995; Hubert, 1997), occupational exposure to certain chemicals (Johanson and Olsen, 1998), and daily variations in air pollution (Kelsall and others, 1997). Nevertheless, evidence of significant effects of pollution on national mortality trends of populous countries has been far from solid. The geographical correlation between all-cause mortality and environmental contamination is usually insignificant. For example, although it was suspected that the recent rise of mortality in Russia had been partly attributable to pollution, geographical mortality data failed to support this conjecture (Chen, Wittgenstein, and McKeon, 1996). Nevertheless, notable large-scale mortality effects of pollution may occur in the future if long-term accumulation of environmental contamination exceeds some threshold.

In the United States, the rate of cancer incidence for children under age 15 has been increasing since the early 1970s. This upward trend is difficult to attribute to improved diagnosis and more complete reporting alone. It has been conjectured that the rise may be due to toxic chemicals in the environment (United States Environmental Protection Agency, 1997).

#### *5. Social alienation*

Various social, economic, and political problems arise in the course of industrial and technological development. These problems sometimes lead to the emergence of socially alienated population segments. A "socially alienated" individual is here defined to be someone lacking (1) firm feelings of belonging to the community and the larger society (other than to criminally oriented groups), (2) values to which he/she strongly commits him/herself, and (3) goals that guide his/her life in constructive manners. The causes of social alienation are complicated and beyond the scope of this paper.

Social alienation might raise the level of mortality through at least four mechanisms.

First, lack of ethical values, lower levels of self-control, depression, self-destructive attitudes, and lack of long-term life plans may increase the incidence of external injuries including homicide, suicide, and accidents. Second low concerns about health care may result in under-usage of medical services and unhealthy life styles, including substance addiction, alcoholism, heavy smoking, nutritional imbalance, and poor personal hygiene. Third, unhealthy life styles during pregnancy and improper health care of young children may raise infant and child mortality, and leave some long-term adverse effects on the health of the surviving children. Finally, any long-term positive effects on health associated with positive-mindedness and constructive attitudes that could enhance neuro-endocrine and neuro-immune functions may be reduced.<sup>5</sup> It may also be noted that the mortality effects of social alienation may not at all be limited to urban slum populations.

These effects of social alienation on the mortality level are not necessarily small. Some cases of unexpectedly high mortality seem to be partly attributable to social alienation: homicide rates are high in some developed countries, in spite of their economic wealth. The age-standardised death rate due to homicide among males in the United States doubled between 1960 and 1974. The level of total mortality is high in some urban slums. A recent study shows that a boy in Harlem, New York City, who reaches the age of 15 has only a 37 per cent chance of surviving to the age of 65 (Geronimus and others, 1996; McCord and Freeman, 1990). The mortality decline stagnated in a number of countries in Eastern Europe and the former Union of Soviet Socialist Republics during the 1970s and 1980s, when various socio-economic problems accumulated, leading to a delay of political reforms (Jozan, 1996; Rychtarikova, Vallin, and Meslé, 1989; Watson, 1995). Mortality due to suicide, homicide, alcoholism, and poisoning in some countries of the former Union of Soviet Socialist Republics rose steeply during the transition period of the early 1990s, when rapid socio-economic changes might have loosened the existing systems of social integration (Shkolnikov, Meslé, and Vallin, 1996).

In the relation between social alienation and mortality, of particular importance seems to be

the extent to which members in relatively lower positions in the socio-economic hierarchy find firm, positive "meanings" of their lives.<sup>6</sup> Although this factor is difficult to measure in internationally comparable ways, its effects on the average mortality level of the entire population through various indirect pathways may be significant. In general, more gain in the life expectancy of the population can be achieved by improving the mortality of population segments that have relatively high mortality, rather than by lowering further the mortality level of population segments that already have lowest death rates.

It is not easy to achieve a very high level of life expectancy if members of significantly large segments of the society have difficulties in finding firm, positive meanings of their lives. For a life to be fully lived, it should be worthy of living.

## E. CONCLUSION

This paper has presented a five-stage model of the epidemiological transition of human mortality, and also described some possible reverse transitions. The model is a combination of a generalisation of past patterns and a prospect of future changes. Since the actual pattern of historical mortality change varies among countries and regions, it may not be difficult to find specific cases that do not fit the model well. The main purpose of this model, however, is to provide a bird-eye view of a *typical* course of mortality history in human societies. The model can be used as a conceptual framework for describing, analysing, and predicting actual mortality trends in the past and future.

## NOTES

<sup>1</sup>1925 is the earliest year for which the time-series of cause-specific mortality estimates in France are available. 1955 is the year of the onset of significant decline in old-age mortality in France (Kannisto, 1994, chapter 9), thus may be considered the beginning of the next transition.

<sup>2</sup>Long-term trends in cause-specific mortality are difficult to estimate, because of relatively frequent changes in the classification of diseases. A notable exception is France, where a considerable amount of research has been done on the adjustment of vital statistics for coding changes (Vallin and Mesle, 1990; Mesle and Vallin, 1996).

<sup>3</sup>Because figure 2 does not reflect the age distribution of deaths, it does not show the actual importance of cardiovas-

cular diseases for total mortality at all ages. Almost half (49 per cent) of all deaths in this population occur at age 80 or above, and more than half (57 per cent) of deaths at all ages are due to cardiovascular diseases.

<sup>4</sup>In all of the three patterns, the age-related relative increase in mortality eventually slows down at very old ages. This deceleration is probably due to selective survival (Vaupe, Manton, and Stallard, 1979; Manton and others, 1994; Horiuchi and Wilmoth, 1998).

<sup>5</sup>Positive effects on health and/or longevity have been found for such factors as religious service attendance (Strawbridge and others, 1997), positive thinking (Minozaki, 1995), social ties (reviewed in Grundy, Bowling, and Farquhar, 1995), community involvement (Strawbridge and others, 1996), and decision latitude at workplace (Bobak and others, 1998), as well as negative effects of hopelessness (Anda and others, 1993), depression (Pratt and others, 1996), and stress in general (Stein-Behrens and Sapolsky, 1992). These effects may work through psycho-neuro-immune and psycho-neuro-endocrine pathways. For example, religious service attendance is positively associated with increased immune functions (Koenig and others, 1997). For reviews of the emerging fields of psychoneuroendocrinology and psychoneuroimmunology, see: Biondi and Zannino, 1997; Cohen and Herbert, 1996; and Kiecolt-Glaser and Glaser, 1995.

<sup>6</sup>It should be noted that in Japan, where an unprecedented rapid rise of life expectancy was achieved during the last few decades, great importance is given to communication and interdependence between upper and lower positions in the social hierarchy (Nakane, 1986).

#### REFERENCES

- Acasadi, George, and J. Nemeskeri (1970). *History of Human Life Span and Mortality*. Budapest: Akademiai Kiado.
- Allard, Michel, Jacques Vallin, Jean-Michel Andrieux and Jean-Marie Robine (1996). In search of the secret of Centenarians: A demographic and medical survey about Centenarians in France. In *Health and Mortality among Elderly Populations*, Graziella Caselli and Alan D. Lopez, eds. Oxford: Clarendon Press, pp. 61-86.
- Anda R., D. Williamson, D. Jones, C. Macera, E. Eaker, A. Glassman, and J. Marks (1993). Depressed affect, hopelessness, and the risk of ischemic heart disease in a cohort of U.S. adults. *Epidemiology*, vol. 4, pp. 285-94.
- Angel, J. Lawrence (1975). Paleoeology, paleodemography and health. In *Population, Ecology, and Social Evolution*, Steven Polgar, ed. The Hague: Mouton, pp. 167-190.
- Austad, Steven N. (1997). *Why We Age*. New York: John Wiley & Sons.
- Banks, D. A., and M. Fossel (1997). Telomeres, cancer, and aging - altering the human life span. *Journal of the American Medical Association*, vol. 278, pp. 1345-1348.
- Black, F. L. (1980). Modern isolated pre-agricultural populations as a source of information on prehistoric epidemic patterns. In *Changing Disease Patterns and Human Behaviour*, N. F. Stanley and R. A. Joske, eds. New York: Academic Press, pp. 37-54.
- Biondi, M., and L. G. Zannino (1997). Psychological stress, neuroimmunomodulation, and susceptibility to infectious diseases in animals and man: a review. *Psychotherapy & Psychosomatics*, vol. 66, pp. 3-26.
- Bobak, M., C. Hertzman, Z. Skodova and M. Marmot (1998). Association between psychosocial factors at work and nonfatal myocardial infarction in a population-based case-control study in Czech men. *Epidemiology*, vol. 9, pp. 43-47.
- Brothwell, Don (1967). The bio-cultural background to disease. In *Disease in Antiquity: A Survey of the Diseases, Injuries and Surgery of Early Populations*, D. Brothwell and A. T. Sandison, eds. Springfield, IL: C.C. Thomas, pp. 56-68.
- Bourgeois-Pichat, J. (1978). Future outlook for mortality decline in the world. *Population Bulletin of the United Nations*, vol. 11, pp. 12-41.
- Butler, Robert N. and Jacob A. Brody, eds. (1995). *Delaying Onset of Late-Life Dysfunction*. New York: Springer.
- Butler, Steven M., and David A. Snowdon (1996). Trends in mortality in older women: findings from the nun study. *Journal of Gerontology: SOCIAL SCIENCES*, vol. 51B, pp. S201-S208.
- Caselli, Graziella (1991). Health transition and cause-specific mortality. In *The Decline of Mortality in Europe*, Roger Schofield, David Reher and Alain Bideau, eds. Oxford: Clarendon Press, pp. 68-96.
- Chen, Lincoln, Friederike Wittgenstein and Elizabeth McKeon (1996). The upsurge of mortality in Russia: Causes and policy implications. *Population and Development Review*, vol. 22, No. 3 (September), pp. 517-530.
- Chollat-Traquet, C. (1992). *Women and Tobacco*. Geneva: World Health Organization.
- Cockerham, William C. (1997). The social determinants of the decline of life expectancy in Russia and Eastern Europe: a lifestyle explanation. *Journal of Health and Social Behavior*, vol. 38, No. 2, pp. 117-130.
- Cohen, Joel E. (1995). *How Many People Can the Earth Support?* New York: W. W. Norton.
- Cohen, S., and T. B. Herbert (1996). Health psychology: psychological factors and physical disease from the perspective of human psychoneuroimmunology. *Annual Review of Psychology*, vol. 47, pp. 113-142.
- Cole, Philip and Brad Rodu (1996). Declining cancer mortality in the United States. *Cancer*, vol. 78, No. 10 (November 15), pp. 2045-2048.
- Coleman, Michel P., Jacques Estève, Philippe Darniecki, Annie Arslan and Hélène Renard (1993). Trends in Cancer Incidence and Mortality. IARC Scientific Publications No. 121. Lyon: WHO/International Agency for Research on Cancer.
- Crimmins, Eileen. M. (1981). The changing pattern of American mortality decline, 1940-77, and its implications for the future. *Population and Development Review*, vol. 7, no. 2, pp. 229-254.
- \_\_\_\_\_, Yasuhiko Saito and Dominique Ingegneri (1997). Trends in disability-free life expectancy in the United States, 1970-90. *Population and Development Review*, vol. 23, No. 3 (September), pp. 555-572.
- Ewald, Paul W. (1994). *Evolution of Infectious Disease*. New York and Oxford: Oxford University Press.
- Fogel, R. W. (1994). Economic growth, population theory, and physiology: the bearing of long-term processes on the making of economic policy. *American Economic Review*, vol. 84, pp. 369-95.
- \_\_\_\_\_, and D. L. Costa (1997). A theory of technophysio evolution, with some implications for forecasting population, health care costs, and pension costs. *Demography*, vol. 34, pp. 49-66.
- Fries, J. F. (1980). Aging, natural death, and the compression of morbidity, *New England Journal of Medicine*, vol. 303, pp. 130-135.
- Gavrilov, L. A., and N. S. Gavrilova (1991). *The Biology of Life Span: A Quantitative Approach*. New York: Harwood.
- Geronimus, A. T., J. Bound, T. A. Waidmann, M. M. Hillemeier and P. B. Burns (1996). Excess mortality among blacks and whites in the United States. *New England Journal of Medicine*, vol. 335, No. 21 (November 21), pp. 1552-1558.

- Grundy, Emily, Ann Bowling and Morag Farquhar (1996). Social support, satisfaction, and survival at old ages. In *Health and Mortality among Elderly Populations*, Graziella Caselli and Alan D. Lopez, eds. Oxford: Clarendon Press, pp. 135-156.
- Harada, M. (1995). Minamata disease: methylmercury poisoning in Japan caused by environmental pollution. *Critical Reviews in Toxicology*, vol. 25, pp. 1-24.
- Himes, Christine L., Samuel H. Preston and Gretchen A. Condran (1994). A relational model of mortality at older ages in low mortality countries. *Population Studies*, vol. 48, No. 2 (July), pp. 269-291.
- Horiuchi, Shiro (1997). Postmenopausal acceleration of age-related mortality increase. *Journal of Gerontology: BIOLOGICAL SCIENCES*, vol. 52A, No. 1, pp. B78-B92.
- \_\_\_\_\_, and John R. Wilmoth (1997). Age patterns of the life-table aging rate for major causes of death in Japan, 1951-1990. *Journal of Gerontology: BIOLOGICAL SCIENCES*, vol. 52A, pp. B67-B77.
- \_\_\_\_\_. (1998). Deceleration in the age pattern of mortality at older ages. *Demography*, in press.
- Hubert, Philippe (1997). Chernobyl, 10 years after: health consequences. *Epidemiology*, vol. 19, No. 2, pp. 187-204.
- Howell, Nancy (1979). *Demography of the Dobe !Kung*. New York: Academic Press.
- Johanson, Christoffer, and Jorgen H. Olsen (1998). Risk of cancer among Danish utility workers - a nationwide cohort study. *American Journal of Epidemiology*, vol. 147, pp. 574-580.
- Johnson, Thomas E., Gordon J. Lithgow and Shin Murakami (1996). Hypothesis: Interventions that increase the response to stress offer the potential for effective life prolongation and increased health. *Journal of Gerontology: BIOLOGICAL SCIENCES*, vol. 51A, No. 6, pp. B393-B395.
- Jozan, Peter (1996). Change in mortality in Hungary between 1980 and 1994. In *Demography of Contemporary Hungarian Society*, Pál P. Tóth and Emil Valkovics, eds. New York: Columbia University Press, pp. 111-138.
- Kannisto, V. (1994). Development of oldest-old mortality, 1950-1990: Evidence from 28 developed countries. *Monographs on Population Aging*, 1. Odense: Odense University Press.
- \_\_\_\_\_. (1996). *The Advancing Frontier of Survival: Life Tables for Old Age*. Odense, Denmark: Odense University Press.
- \_\_\_\_\_, J. Lauristen, A. R. Thatcher and J. W. Vaupel (1994). Reduction in mortality at advanced ages. *Population and Development Review*, vol. 20, pp. 793-810.
- Kaplan, Hillard (1979). The evolution of the human life course. In *Between Zeus and the Salmon: The Biodemography of Longevity*, K. E. Wachter and C. E. Finch, eds. Washington, D.C.: National Academy Press, pp. 175-211.
- Keeley, Lawrence H. (1997). *War Before Civilization*. New York: Oxford University Press.
- Kelsall, J. E., J. M. Samet, S. L. Zeger and J. Xu (1997). Air pollution and mortality in Philadelphia, 1974-1988. *American Journal of Epidemiology*, vol. 146, pp. 750-762.
- Kiecolt-Glaser, J. K., and R. Glaser (1995). Psychoneuroimmunology and health consequences: data and shared mechanisms. *Psychosomatic Medicine*, vol. 57, pp. 269-274.
- Koenig, H. G., H. J. Cohen, L. K. George, J. C. Hays, D. B. Larson and D. G. Blazer (1997). Attendance at religious services, interleukin-6, and other biological parameters of immune function in older adults. *International Journal of Psychiatry in Medicine*, vol. 27, pp. 233-250.
- Kohn, Robert R. (1982). Cause of death in very old people. *Journal of the American Medical Association*, vol. 247, No. 20 (May), pp. 2793-2797.
- Lancaster, H. O. (1990). *Expectations of Life: A Study in the Demography, Statistics, and History of World Mortality*. New York: Springer-Verlag.
- Levi, F., C. Lavecchia, E. Negri and F. Lucchini (1997). Declining cancer mortality in European Union. *Lancet*, vol. 349, pp. 508-509.
- Levine, Myron M., and Stephen B. Thacker, eds. (1996). *Epidemiologic Reviews: Emerging and Reemerging Infections*, vol. 18, No. 1.
- Levy, Stuart B. (1998). The challenge of antibiotic resistance. *Scientific American* (March), pp. 46-53.
- Lopez, Alan D. (1983). The sex mortality differentials in developed countries. In *Sex Differentials in Mortality: Trends, Determinants and Consequences*, A. D. Lopez, and L. T. Ruzicka, eds. Canberra: Australian National University, pp. 53-120.
- \_\_\_\_\_. (1995). The lung cancer epidemic in developed countries. In *Adult Mortality in Developed Countries: From Description to Explanation*, Alan D. Lopez, Graziella Caselli and Tapani Valkonen, eds. Oxford: Clarendon Press, pp. 111-134.
- Manton, Kenneth G., and Eric Stallard (1984). *Recent Trends in Mortality Analysis*. Orlando, Florida: Academic Press.
- \_\_\_\_\_. (1996). Longevity in the United States: age and sex-specific evidence on life span limits from mortality patterns 1960-1990. *Journal of Gerontology: BIOLOGICAL SCIENCES*, vol. 51A, No. 5, pp. B362-B375.
- Manton, Kenneth G., Larry S. Corder and Eric Stallard (1997). Chronic disability trends in elderly United States populations: 1982-1994. *Proceedings of the National Academy of Sciences*, vol. 94, pp. 2593-2598.
- Manton, Kenneth G., Eric Stallard and Larry S. Corder (1997). Changes in the age dependence of mortality and disability: cohort and other determinants. *Demography*, vol. 34, pp. 135-158.
- Manton, Kenneth G., Eric Stallard and H. Dennis Tolley (1991). Limits to human life expectancy: evidence, prospects, and implications. *Population and Development Review*, vol. 17, No. 4 (December), pp. 603-637.
- Manton, Kenneth G., Eric Stallard, Max A. Woodbury and J. Ed Dowd (1994). Time-varying covariates in models of human mortality and aging: multidimensional generalizations of the Gompertz. *Journal of Gerontology: BIOLOGICAL SCIENCES*, vol. 49, No. 4 (July), pp. B169-B190.
- Masoro, Edward J., ed. (1995). *Handbook of Physiology. Section II: Aging*. New York: Oxford University Press.
- \_\_\_\_\_, and Steven N. Austad (1996). The evolution of the antiaging action of dietary restriction: A hypothesis. *Journal of Gerontology: BIOLOGICAL SCIENCES*, vol. 51A, No. 6, pp. B387-B391.
- McCord, C., and H. P. Freeman (1990). Excess mortality in Harlem. *New England Journal of Medicine*, vol. 322, pp. 173-177.
- McKeown, T. (1979). *The Role of Medicine: Dream, Mirage, or Nemesis*. Princeton: Princeton University Press.
- McLaughlin, John R., Anthony L. A. Fields, Jane F. Gentleman, Isra Levy, Barbara Whyllie, Heather Whittaker, Rod Riley, Judy Lee, B. Ann Coombs and Leslie A. Guadette (1997). Cancer incidence and mortality, 1997. *Health Reports* (Ottawa, Canada), vol. 8, No. 4, pp. 41-51 and 43-54.
- Medawar, P. B. (1952). *Unsolved Problems of Biology*. London: H. K. Lewis.
- Meslé, France, and Jacques Vallin (1996). Reconstructing long-term series of causes of death. *Historical Methods*, vol. 29, No. 2 (Spring), pp. 72-87.
- Miller, Richard A. (1995). Aging and the immune response. In *Handbook of the Biology of Aging* (4th edition), Edward L. Schneider and John W. Rowe, eds. San Diego: Academic Press.

- (1997). When will the biology of aging become useful?: Future landmarks in biomedical gerontology, *Journal of the American Geriatrics Society*, vol. 45, No. 10, pp. 1258-1267.
- Mizokami, S. (1995). Some basic factors of future-life perspective from "Why is it" test. *Japanese Journal of Psychology*, vol. 66, pp. 367-72.
- Morel, Marie-France (1991). The care of children: the influence of medical innovation and medical institutions on infant mortality 1750-1914. In *The Decline of Mortality in Europe*, Roger Schofield, David Reher and Alain Bideau, eds. Oxford: Clarendon Press, pp. 196-219.
- Morse, Stephen S. (1995). Factors in the emergence of infectious diseases. *Emerging Infectious Diseases*, vol. 1, No. 1 (January-March), pp. 7-15.
- Murray, Christopher J. L., and Alan D. Lopez (1997). Mortality by cause for eight regions of the world: Global burden of disease study. *Lancet*, vol. 349 (May 3), pp. 1269-1276.
- Nakane, Chie (1986). *Japanese Society*. Berkeley: University of California Press.
- Olshansky, S. Jay, and A. B. Ault (1986). The fourth stage of the epidemiologic transition: the age of delayed degenerative diseases. *Milbank Memorial Fund Quarterly*, vol. 64, pp. 355-391.
- Olshansky, S. Jay, B. A. Carnes and C. Cassel (1990). In search of Methuselah: estimating the upper limits of human longevity. *Science*, vol. 250, pp. 634-640.
- Olshansky, S. Jay, and Bruce A. Carnes (1996). Prospect for extended survival: A critical review of the biological evidence. In *Health and Mortality among Elderly Populations*, Graziella Caselli and Alan D. Lopez, eds. Oxford: Clarendon Press, pp. 39-58.
- Olshansky, S. Jay, Bruce A. Carnes, Richard G. Rogers and Len Smith (1997). Infectious diseases—new and ancient threats to world health. *Population Bulletin*, vol. 52, pp. 1-52.
- Omran, A. R. (1971). The epidemiological transition: a theory of the epidemiology of population change. *Milbank Memorial Fund Quarterly*, vol. 49, pp. 509-538.
- Ott, Wayne R., and John W. Roberts (1998). Everyday exposure to toxic pollutants. *Scientific American* (February), pp. 86-91.
- Perls, T. T., and E. R. Wood (1996). Acute care costs of the oldest old: they cost less, their care intensity is less, and they go to nonteaching hospitals. *Archives of Internal Medicine*, vol. 156, No. 7 (April 8), pp. 754-760.
- Perrenoud, Alfred (1991). The attenuation of mortality crises and the decline of mortality. In *The Decline of Mortality in Europe*, Roger Schofield, David Reher and Alain Bideau, eds. Oxford: Clarendon Press, pp. 18-37.
- Peto, R., A. D. Lopez, J. Boreham, M. Thun and C. Heath, Jr. (1994). *Mortality from Smoking in Developed Countries, 1950-2000*. New York: Oxford University Press.
- Pratt, L. A., D. E. Ford, R. M. Crum, H. K. Armenian, J. J. Gallo and W. W. Eaton. (1996). Depression, psychotropic medication, and risk of myocardial infarction: Prospective data from the Baltimore ECA follow-up. *Circulation*, vol. 94, pp. 3123-3129.
- Preston, Samuel (1976). *Mortality Patterns in National Populations; with Special References to Recorded Causes of Death*. Studies in Population. New York: Academic Press.
- \_\_\_\_\_. (1990). Sources of variation in vital rates: an overview. In *Convergent issues in genetics and demography*, Julian Adams, David A. Lam, Albert I. Hermalin, and Peter E. Smouse, eds. New York: Oxford University Press, pp. 335-350.
- \_\_\_\_\_. (1995). Human mortality throughout history and prehistory. In *The State of Humanity*, Julian L. Simon, ed. Oxford: Blackwell, pp. 30-36.
- \_\_\_\_\_, and Etienne van de Walle (1978). Urban French mortality in the nineteenth century. *Population Studies*, vol. 32, pp. 275-297.
- Puranen, Bi (1991). Tuberculosis and the decline of mortality in Sweden. In *The Decline of Mortality in Europe*, Roger Schofield, David Reher and Alain Bideau, eds. Oxford: Clarendon Press, pp. 97-117.
- Richardson, Arlan, and Mohammad A. Pahlavani (1994). Thoughts on the evolutionary basis of dietary restriction. In *Genetics and Evolution of Aging*, Michael R. Rose and Caleb E. Finch, eds. Dordrecht, the Netherlands: Kluwer Academic Publishers.
- Rogers, Richard G., and R. Hackenberg (1987). Extending epidemiologic transition theory: a new stage. *Social Biology*, vol. 34, pp. 234-43.
- Rychtaríková, Jitka, Jacques Vallin and France Meslé (1989). Comparative study of mortality trends in France and the Czech Republic since 1950. *Population* (English edition), vol. 1, pp. 293-321.
- Schneider, Edward L., and Jacob A. Brody (1983). Aging, natural death, and the compression of morbidity: another view. *New England Journal of Medicine*, vol. 309, pp. 854-855.
- Shkolnikov, Vladimir, France Meslé and Jacques Vallin (1996). Health crisis in Russia. *Population: An English Selection*, vol. 8, pp. 123-154.
- Stein-Beihrens, B. A., and R. M. Sapolsky (1992). Stress, glucocorticoids, and aging. *Aging*, vol. 4, pp. 197-210.
- Strawbridge, W. J., R. D. Cohen, S. J. Shema and G. A. Kaplan (1996). Successful aging: predictors and associated activities. *American Journal of Epidemiology*, vol. 144, pp. 135-141.
- \_\_\_\_\_. (1997). Frequent attendance at religious services and mortality over 28 years. *American Journal of Public Health*, vol. 87, pp. 957-961.
- Szreter, Simon (1997). Economic growth, disruption, deprivation, disease, and death: On the importance of the politics of public health for development. *Population and Development Review*, vol. 23, pp. 693-728.
- Uemura, Kazuo, and Zbynek Piza (1988). Trends in cardiovascular disease mortality in industrialized countries since 1950. *World Health Statistics Quarterly*, vol. 41, No. 3/4, pp. 155-178.
- United Nations Secretariat (1962). The situation and recent trends of mortality in the world. *Population Bulletin of the United Nations*, No. 6, pp. 3-145.
- \_\_\_\_\_. (1988). Sex differentials in life expectancy and mortality in developed countries: an analysis by age groups and causes of death from recent and historical data. *Population Bulletin of the United Nations*, No. 25, pp. 65-106.
- United States Center for Disease Control and Prevention (1994). *Addressing Emerging Infectious Disease Threats: A Prevention Strategy for the United States*, Atlanta.
- United States Environment Protection Agency (1997). *Children's Health Experts Call for National Strategy to Prevent Childhood Cancer* (Press Release, 09/17/97).
- Vallin, Jacques (1983). Sex patterns of mortality: a comparative study of model life tables and actual situations with special reference to the cases of Algeria and France. In *Sex Differentials in Mortality: Trends, Determinants and Consequences*, A. D. Lopez, and L. T. Ruzicka, eds. Canberra: Australian National University, pp. 443-476.
- \_\_\_\_\_. (1991). Mortality in Europe from 1720 to 1914: Long-term trends and changes in patterns by age and sex. In *The Decline of Mortality in Europe*, Roger Schofield, David Reher and Alain Bideau, eds. Oxford: Clarendon Press, pp. 38-67.
- \_\_\_\_\_, and France Meslé (1988). *Les Causes de Décès en France de 1925 à 1978*. Paris: Institut National d'Études Démographiques Presses Universitaires de France.



- \_\_\_\_\_ (1990). The causes of death in France, 1925-1978: Reclassification according to the eighth revision of the international classification of diseases. In *Measurement and Analysis of Mortality: New Approaches*, J. Vallin, S. D'Souza, and A. Palloni, eds. Oxford: Clarendon Press, pp. 295-327.
- Vaupel, James W. (1997). The average French baby may live 95 or 100 years. In *Longevity: To the Limits and Beyond*, J.-M. Robine, J. W. Vaupel, B. Jeune, and M. Allard, eds. New York: Springer-Verlag, pp. 11-27.
- \_\_\_\_\_, Kenneth G. Manton and Eric Stallard (1979). The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography*, vol. 16, No. 3 (August), pp. 439-454.
- Waldron, I. (1985). What do we know about causes of sex differences in mortality?: a review of the literature. *Population Bulletin of the United Nations*, vol. 18, pp. 59-76.
- \_\_\_\_\_ (1986). The contribution of smoking to sex differences in longevity. *Public Health Reports*, vol. 101, pp. 163-173.
- Watson, P. (1995). Explaining rising mortality among men in eastern Europe. *Social Science & Medicine*, vol. 41, No. 7, pp. 923-34.
- Williams, G. C. (1957). Pleiotropy, natural selection and the evolution of senescence. *Evolution*, vol. 11, pp. 398-411.
- Wilmoth, John R. (1997). In search of limits: What do demographic trends suggest about the future of human longevity? In *Between Zeus and the Salmon: The Biodemography of Longevity*, K. E. Wachter and C. E. Finch, eds. Washington, D. C.: National Academy Press, pp. 38-64.
- \_\_\_\_\_ (1998). The future of human longevity: A demographer's perspective. *Science*, vol. 280, pp. 395-397.
- Wingard, D. L., and B. A. Cohn (1990). Variations in disease-specific sex morbidity and mortality ratios in the United States. In *Gender, Health and Longevity: Multidisciplinary Perspectives*, M. G. Ory and H. R. Warner, eds. New York: Springer, pp. 25-37.
- World Health Organization (1997). *Tobacco or Health: A Global Status Report*. Geneva.
- Wrigley, E. A. (1969). *Population and History*. New York and Toronto: McGraw-Hill.

### III. THE MEASUREMENT OF ADULT MORTALITY: AN ASSESSMENT OF DATA AVAILABILITY, DATA QUALITY AND ESTIMATION METHODS

Kenneth Hill\*

#### A. INTRODUCTION

Both fertility and child mortality have proved to be measurable with reasonable accuracy by sample surveys in a wide range of data availability and data quality settings, but the two remaining components of demographic change, adult mortality and (particularly) migration, have proved more problematic. This paper is concerned with the problems posed by the measurement of adult mortality. In market economies and the formerly socialist countries of Europe, death registration is essentially complete and adult mortality rates can for the most part be calculated directly from registered deaths and population denominators. Only at the oldest ages, where age misreporting patterns in either data source may lead to bias, is there any significant degree of uncertainty. In many developing countries, on the other hand, death registration is often significantly incomplete. Although the registration of adult deaths is often more complete than that of child deaths, the alternative data sources and analytical methods that have been developed to measure child mortality (such as birth histories and indirect methods based on summary birth histories) have been more successful than those developed to measure adult mortality. This paper first reviews data sources relevant to the measurement of adult mortality, then data availability from each source, and finally the analytical methods available to use the data from these sources. The paper concludes with a discussion of appropriate strategies for countries in order to improve their adult mortality estimates.

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#### B. RELEVANT DATA SOURCES

##### 1. *Conventional civil registration and population census*

The conventional sources of information on which life tables for adults are based are civil registration providing counts of deaths by age and sex (the numerators of age-specific mortality rates) and population estimates by age and sex usually based upon one or more population census (the denominators of the rates). For the rates to be accurate, the coverage of deaths and population has to be similar and age reporting has to be of good quality. It is not sufficient for age reporting errors to be similar in the two data sets.

##### 2. *Sample civil registration*

Information on deaths by age and sex is collected for a sample of geographic areas on a continuous basis. Regular independent retrospective surveys provide population denominators and may also record vital events that can then be matched against registered events. With matching of events between sources, field reconciliation can in theory be used to obtain a complete and unduplicated count of events. The resulting rates will be accurate if coverage of deaths and population is the same and if age reporting is of good quality.

##### 3. *Sequential population censuses*

A sequence of population censuses provides information about attrition of cohorts from one census to the next. If the censuses are of similar accuracy, age reporting is of good quality and net migration negligible, the attrition can be interpreted as mortality, and intercensal prob-

abilities of dying for cohorts calculated. In practice, none of these conditions is likely to hold.

#### 4. *Census or survey questions on recent deaths*

Censuses or surveys can include questions on deaths by age and sex in some reference period, usually the preceding year or since a recent widely-known event or festival, as well as collecting information on the population by age and sex. For the resulting mortality rates to be accurate, reporting of deaths must be as complete as of population, and age reporting must be of good quality. Once again, it is not sufficient that age errors be similar in both sets of data.

#### 5. *Census or survey questions on survival of kin*

Census and survey questions on survival of mother, of father, of first spouse and of siblings have all been proposed as ways of estimating adult mortality in situations where other data are lacking. Age of the respondent is typically used as a proxy for exposure to risk of dying, and models of nuptiality, fertility and mortality are used to convert proportions with surviving kin into life table measures. Results are accurate to the extent that survival status is correctly reported and the underlying models used are approximately valid for the population being studied. The estimates obtained do not have a precise time reference, however, since the events accumulate over often extended periods of time. The estimates reflect a rather complex average of the rates in operation over the period.

### C. DATA AVAILABILITY

#### 1. *Civil registration*

Countries are encouraged to report to the United Nations the completeness of the national civil registration system. The system is coded as "C" for complete if the country reports that at least 90 per cent of deaths are recorded, and "U" for incomplete if less than 90 per cent of deaths are recorded. The United Nations also codes countries if no data are available on

completeness, or if the country does not report completeness to the United Nations.

Table 1 shows the percentage distribution of countries by region and by reported completeness of death registration for the early 1970s and the early 1990s. Part (a) of the table uses country as the unit, whereas part (b) weights countries by their approximate population at the time.

On a country basis, coverage of civil registration appears to have improved somewhat from the early 1970s to the early 1990s. The proportion of all developing countries (excluding the Asian republics of the former Soviet Union) that reported complete coverage increased from 28 per cent to 35 per cent. The percentage of countries reporting incomplete registration dropped from 28 to 21 per cent, with the largest single category, information unavailable, staying constant at 44 per cent. By region, the Caribbean is easily the best, followed by Latin America and Oceania. Sub-Saharan Africa and West Asia are the regions with the poorest civil registration systems.

When weighted by population, the improvement of civil registration data availability disappears. In the early 1990s, only 16 per cent of the populations of the countries included had death registers reported as complete, down from 17 per cent in the early 1970s. The proportion of population with incomplete registration of deaths also fell, from 17 to 15 per cent, but the proportion not reporting rose from 66 to 69 per cent. On the population-weighted basis, Oceania has the highest (81 per cent) level of complete registration, followed by the Caribbean, North Africa and Latin America. At the other end of the scale, less than 1 per cent of the population of sub-Saharan Africa is covered by reportedly complete death registration, and in both West Asia and Other Asia the comparable figure is 5 per cent or less. Only the Caribbean shows a strong improvement from the early 1970s to the early 1990s.

In summary, civil registration seems to be providing adult mortality information about an increasing number of countries, but not about an increasing proportion of the population of the developing world. It provides us with effec-

TABLE 1. REPORTED COMPLETENESS OF DEATH REGISTRATION, DEVELOPING COUNTRIES

<i>Region</i>	<i>Time period</i>	<i>"Complete"</i>	<i>"Incomplete"</i>	<i>Other<sup>a</sup></i>	<i>Number of countries</i>
<i>A. Percentages of countries of region</i>					
Latin America	Early 1970s	41	50	9	22
	Early 1990s	50	41	9	22
Caribbean	Early 1970s	75	17	8	24
	Early 1990's	84	8	8	25
Sub-Saharan Africa	Early 1970s	8	27	65	51
	Early 1990s	12	16	73	51
Northern Africa	Early 1970s	20	60	20	5
	Early 1990s	20	80	0	5
West Asia	Early 1970s	6	35	59	17
	Early 1990s	18	24	59	17
Other Asia <sup>b</sup>	Early 1970s	20	28	52	25
	Early 1990's	24	16	60	25
Oceania	Early 1970s	29	13	58	24
	Early 1990s	36	24	40	25
Total	Early 1970s	28	28	44	171
	Early 1990s	35	21	44	173
<i>B. Percentages of regional population</i>					
Latin America	Early 1970s	38	25	37	22
	Early 1990s	41	53	7	22
Caribbean	Early 1970s	29	51	20	24
	Early 1990s	60	21	19	25
Sub-Saharan Africa	Early 1970s	1	24	75	51
	Early 1990s	0	11	88	51
Northern Africa	Early 1970s	47	31	22	5
	Early 1990s	49	51	0	5
West Asia	Early 1970s	2	43	55	17
	Early 1990s	4	42	54	17
Other Asia <sup>b</sup>	Early 1970s	6	14	79	25
	Early 1990s	5	9	86	25
Oceania	Early 1970s	80	4	16	24
	Early 1990s	81	2	17	25
Total	Early 1970s	17	17	66	171
	Early 1990s	16	15	69	173

Sources: United Nations, *Demographic Yearbook* (New York, various years).

NOTE: Row percents may not sum to 100 because of rounding.

<sup>a</sup>Combines "Data regarding completeness not available" and "Category not applicable" categories.

<sup>b</sup>Excludes Asian republics of former Soviet Union.

tively no information about sub-Saharan Africa and Asia.

## *2. Sample registration*

Sample registration systems have a mixed record. A number of Andean countries of Latin America introduced sample registration systems in the 1960s, generally with the objective of gradually expanding the sample to become national. However, these experiments have not survived. In the early 1970s, India introduced a dual system (continuous registration combined with retrospective surveys at 6-monthly intervals) on a sample basis, with a sample size of about 4,000 villages or urban blocks. The Sample Registration System (SRS) as it is known has become the main source of fertility and mortality estimates for states and India as a whole, and appears to have achieved good coverage of events. The SRS sample is updated every decade to reflect changes in population distribution. Rather similar systems introduced in Bangladesh and Pakistan have yet to be adequately evaluated.

## *3. Sequential population censuses*

Most countries of the world have conducted multiple population censuses, providing age distributions for two or more points in time. Thus in theory, most countries could estimate adult mortality from intercensal survival methods, particularly given the flexibility of recent developments in such methods (see section E below). However, in practice, the requirements of negligible or known migration, negligible or known changes in census coverage, and negligible age misreporting prove unattainable.

## *4. Census or survey questions on recent deaths*

Household deaths by age and sex in some reference period (typically the 12 months preceding the date of interview, but sometimes in the period since a well-known event or religious festival) have been included in a number of censuses in the 1990 round, though there are wide differences between regions. In sub-Saharan Africa, 60 per cent of censuses for which census questionnaires could be inspected included a question on household deaths, compared to only 29 per cent in Other Asia and only 10 per cent in Latin America. From a popula-

tion perspective, this source of data is particularly important in the Other Asia region; China included appropriate questions in the 1990 census.

## *5. Prospective sample surveys*

Prospective sample surveys have provided information on adult deaths in some countries, often of high quality, but such surveys have become rare because they are expensive.

## *6. Census or survey questions on survival of kin*

Questions on survival of parents have been included in a number of censuses in the 1990 round, though once again there are wide differences between regions. In sub-Saharan Africa, 67 per cent of countries whose census questionnaires were inspected included questions on both survival of mother and of father. In Latin America, the comparable figures were 60 per cent for survival of mother but zero per cent for survival of father. No other region except Oceania had any census data on parental survival. Questions on survival of parents have also been included in a number of Demographic and Health Surveys (DHS), though in an unusual format, and in other household surveys.

Questions on survival of siblings have also been included in a number of DHS surveys as part of the module aimed at measuring maternal mortality. Although the main purpose of this module is to identify maternal deaths, the module asks about all siblings born of the same mother, collecting sex, age (for those still alive), or age at death and years ago of death (for those who have died). These data have been collected in 18 countries in sub-Saharan Africa, 7 countries in Asia and North Africa, and 5 countries in Latin America (twice in Bolivia).

## **D. BASIS OF UNITED NATIONS ESTIMATES**

Data availability is only one dimension of data use: what really matters is on what data adult mortality estimates are really based. The United Nations updates its World Population Prospects every two years. The 1996 Revision (United Nations, 1997) classifies the information on which estimates of life expectancy are based. Table 2 summarises the basis for the estimates,

TABLE 2. DISTRIBUTION OF DEVELOPING COUNTRIES BY REGION AND BASIS OF UNITED NATIONS ADULT MORTALITY ESTIMATES, 1996 REVISION

Region	Basis	Source of estimates (percent)							Number of countries
		Registered deaths <sup>a</sup>	Adjusted Registered deaths <sup>b</sup>	Deaths in reference period <sup>c</sup>	Survival of parents	Infant mortality and models <sup>d</sup>	Neighbouring countries <sup>e</sup>	Not specified	
Latin America	Countries	32	45	9	0	5	0	9	22
	Population	36	62	2	0	0	0	0	
Caribbean	Countries	36	8	0	0	0	0	54	24
	Population	56	42	0	0	0	0	2	
Sub-Saharan Africa	Countries	6	0	10	4	58	16	6	50
	Population	0	0	6	12	64	17	0	
Northern Africa	Countries	33	0	17	0	33	17	0	6
	Population	26	0	7	0	67	0	0	
West Asia	Countries	44	0	0	0	50	0	6	18
	Population	17	0	0	0	83	0	0	
Other Asia	Countries	55	0	13	0	13	19	0	31
	Population	47	0	43	0	2	8	0	
Oceania	Countries	14	0	0	9	5	9	64	22
	Population	8	0	0	74	6	5	8	
Total	Countries	29	7	6	4	29	7	19	173
	Population	38	6	31	2	15	7	0	

Source: United Nations, *World Population Prospects: The 1996 Revision* (New York, United Nations, Sales No. E.97.XIII.16).

NOTES: Percents may not add to 100 because of rounding. In cases where more than one source was used, the country was classified by the closest to the conventional method.

<sup>a</sup>Includes estimates based on registered deaths, deaths registered by sample registration, and national life tables for a specific period.

<sup>b</sup>Registered deaths adjusted by growth balance or Courbage-Fargues methods.

<sup>c</sup>Generally collected by retrospective questions in a population census; length of reference period generally about 12 months. Sometimes collected by prospective, multi-round sample surveys.

<sup>d</sup>Estimates of adult mortality obtained from estimates of infant mortality combined with a model life table system.

both in the form of a numerical count of countries and on a population-weighted basis.

Contrasts between regions are sharp. For countries of Latin America, 77 per cent of life expectancy estimates are derived from registered deaths (32 per cent) or adjusted registered deaths (45 per cent), and for countries of the Caribbean, the corresponding figures are 98 per cent, 36 per cent and 62 per cent; for less than 5 per cent of countries and less than 1 per cent of populations of these two regions are estimates of adult mortality derived from estimates of infant mortality in combination with a model life table system or extrapolated from estimates for neighbouring countries. However, since sources are not described for very small countries, and the Caribbean has many small island nations, source is not specified for 54 per cent of the countries (and 2 per cent of the population) of this region. In sub-Saharan Africa, in contrast, registered deaths provide a basis for estimates in only 6 per cent of countries (less than 1 per cent of the population), whereas estimates of adult mortality are derived from estimates of infant mortality (58 per cent) or extrapolated from estimates for neighbouring countries (16 per cent) in nearly three-quarters of countries and over 80 per cent of the population of the region. Other regions fall in between these two extremes, with North Africa and West Asia relying more heavily on extrapolation from child mortality estimates, and Other Asia making more use of registered deaths.

For populations of developing countries as a whole, close to 40 per cent of estimates of adult mortality are based on registered deaths, over 30 per cent on deaths in the household in a reference period prior to a census or during a prospective survey, and 15 per cent on estimates of infant mortality in combination with model life tables. Clearly, these population percentages are strongly influenced by India (registration by virtue of the SRS) and China (deaths in the 6 months before the 1990 census).

In summary, tables 1 and 2 suggest that the quality of vital registration data has been improving over the last two decades when looked at on the basis of counts of countries, but has remained almost unchanged on the basis of populations. In the early 1990s, adult mortality estimates for some 44 per cent of the population of developing countries (and 36 per cent of

countries) came from registered deaths (in a few cases after adjustment), and for only 22 per cent of total developing country population (but 40 per cent of countries) did the estimates come from survival of parents data, infant mortality estimates, or adoption of information from neighbouring countries.

## E. ESTIMATION METHODOLOGIES

A thorough review of estimation methodologies is beyond the scope of this paper. Timæus (1991a) provides an excellent review up to that date, and this topic will be the subject of a manual in the near future. However, before discussing strategies, it is important at least to outline the major methods available.

### 1. *Methods for adjusting registered deaths*

Early methods proposed by Carrier (1958) and Fargues and Courbage (1972) essentially used the age distribution of deaths as a basis for estimating coverage. The most widely used methods for estimating an adjustment factor for the completeness of registration of adult deaths compare the age distribution of registered deaths to the age distribution of the population. The first such method developed, the Growth Balance Equation (Brass, 1975), compared entry rates into the open-ended population segments age  $a$  and over to death rates in the same age segments. By assuming a stable population, and thus a constant growth rate for all age segments, and a constant omission of deaths relative to population across all ages, the coverage of death registration was estimated as the reciprocal of the slope of the relationship between the entry rates and the observed death rates across age segments. This method was generalised by Hill (1987). Using observed intercensal growth rates for the population segments, the assumption of stability could be relaxed. The slope of the relationship across age segments between entry rate minus growth rate on the one hand and observed death rate on the other could be interpreted as the reciprocal of the completeness of registration relative to an average of census coverage, while the intercept could be interpreted as an indicator of the proportionate change in census coverage from one census to the other (appearing as a constant error in the growth rates). Bennett and Horiuchi (1981) proposed another approach, essentially

a generalisation of Carrier's (1958) method, that does not require an assumption of stability. Age-specific growth rates above any age  $a$  are used to convert the actual age distribution of deaths to that of a stationary population. The ratio of the sum of deaths estimated for the stationary population above age  $a$  to the population of the same age estimates the completeness of death registration above that age relative to population coverage. The method is sensitive to errors in observed growth rates, and these errors may be substantial if census coverage changes from one census to the next.

It appears that the original Growth Balance Equation has been much more widely used than either the Generalised Growth Balance Equation or the Bennett-Horiuchi method, despite the fact that populations are now substantially destabilised in almost all parts of the world. The methods that do not require the assumption of stability still require the assumption of zero net migration (or known net migration) and are sensitive to age reporting errors, particularly if these errors are different between data sources; the Bennett-Horiuchi method is also sensitive to changes in census coverage. However, these evaluation methods can usefully be applied not only to registered deaths but also to deaths recorded retrospectively by a census or large survey. It should also be noted that these methods all assess coverage of registered deaths relative to the coverage of the population or populations being used. The problem of census coverage is thus automatically accounted for.

One additional approach to the adjustment of registered deaths is a field-based approach through multiple collection systems and the matching and reconciliation of events between systems. Such a multiple record system is the basis of the Indian SRS, which is believed to give valid results.

## 2. *Estimation methods based on survey data on survival of parents*

Data on survival of mother or of father provide information on survival for a defined period of time (the respondent's age or age plus gestation) but not from a defined age. Models of parental survival have been developed to allow for the distributional effects of the range of ages of childbearing (Brass and Hill, 1973; Hill and Trussell, 1977; Timæus, 1991b; 1992)

in order to develop methods to convert proportions with parents surviving into life table survival probabilities, and to provide estimates of an approximate reference period for the estimates (Brass and Bamgboye, 1981). The methods have been summarised in United Nations (1983), though Timæus (1992) has shown that Hill's original calculations of multiplying factors for survival of fathers given in United Nations (1983) were incorrect; he provides a revised methodology. Survival of parents methods are sensitive to systematic age misreporting and to the "adoption effect", whereby the survival of adoptive rather than natural parents is reported for dependent children.

Various developments of the parental survival methods have been suggested. Changes in proportions with surviving mothers or surviving fathers from one census to another can be interpreted in terms of life table survivorship for the intercensal period (Preston and Chen, 1984; Timæus, 1986). Additional information on time period of death has been proposed as a way of improving the timeliness of estimates (Chackiel and Orellana, 1985). Use of additional information on survival to some specified life event of the respondent, such as first marriage, has been proposed both to improve the timeliness of estimates and to reduce the effects of the "adoption effect" (Timæus, 1991b). Despite these developments, and despite wide availability of data for sub-Saharan Africa and Latin America, relatively few country estimates of adult mortality in table 2 are based on parental survival data.

In the parental survival methods, proportions of respondents with a surviving mother or father are converted into life table probabilities of surviving by use of methods based on models of fertility and mortality. Exposure to risk is determined by the age of the respondent, but the distribution of starting points for that risk is given by fertility models, and the pattern of subsequent risk is given by model life tables. However, the HIV/AIDS epidemic has given rise to actual life tables that are very different from existing models: typically, mortality rates in populations with high HIV prevalence are very high for young adults, may drop slightly in early middle age, and then rise in a more or less standard pattern. Such a non-standard age pattern of mortality will have no major impact on the average level of mortality estimated from



parental survival data, though of course a model life table fitted to the estimated survivorship ratios will not represent the true age pattern of mortality. More seriously, however, the time reference of orphanhood-based estimates will be incorrect in situations of high AIDS mortality (assuming that excess mortality is a recent phenomenon); the time reference will not reflect the recent upsurge in mortality, and will give the impression of higher mortality in the more distant past than was actually the case. The availability of data from two or more surveys would alleviate this problem. AIDS may also increase the severity of selection effects on parental survival estimates. Since the children of HIV positive mothers are at high risk of dying in childhood, mortality risks for mothers and children will be correlated, reducing the reported prevalence of dead mothers. Behavioural factors might also contribute to selection, if family traits affect the risk of HIV infection.

### 3. *Estimation methods based on survey data on survival of siblings*

Hill and Trussell (1977) first proposed a method for estimating mortality from data on survival of siblings. The basic idea of the method is that, on average, the siblings of a respondent are about the same age as the respondent him- or her- self, and thus that the siblings have been exposed to the risk of dying for approximately the same length of time as the respondent. The proportion alive of a respondent's siblings can then be interpreted as an estimate of the cohort probability of surviving to the respondent's age. Timæus and others (1997) have recently improved on the original formulation, recalculating adjustment factors to take account of the fact that the age range of children within a family is smaller than the age range of the population age specific fertility distribution.

Methods of adult mortality estimation based on sibling survival have recently become important because sibling survival data have been widely collected as a way of estimating maternal mortality (Graham and others, 1989). The Demographic and Health Surveys (DHS) program has incorporated a maternal mortality module in some 30 surveys, collecting not only information on survivorship but also sibling age (for those still alive) or age at death and time period of death (for those that have died).

Such data permit the direct calculation of age-specific mortality rates for specified time periods. Preliminary assessments of such data suggest that they may underestimate adult mortality (Stanton and others, 1997), particularly for time periods more than seven years before interview.

Mortality estimates based on sibling survival are reasonably robust to the appearance of abnormal age patterns of mortality resulting from the HIV/AIDS epidemic. Sibling data are also less likely to be distorted by selection effects than parental survival. However, the time reference of indirect estimates of adult mortality based on sibling survival data will be distorted by any recent upsurge in mortality related to the epidemic. Sibling survival data of the DHS type (with current age of survivors, and both age at and time period of death, for those that have died) could be evaluated using growth balance methods if the data were collected for all adults.

### 4. *Intercensal survival methods*

Methods of adult mortality estimation based on intercensal survival have proved to be very sensitive to variations in census coverage from one census to the next and to age reporting errors. Preston and Bennett (1983) propose a method that uses age-specific growth rates in its computation, smoothing out some of the effects on cohort survival of repeated patterns of age reporting errors, but the method still proves sensitive to systematic age exaggeration, for example.

## F. STRATEGIES FOR IMPROVING ADULT MORTALITY ESTIMATES

### 1. *Data collection strategies for countries lacking data*

Choice of a strategy depends on a number of characteristics of the country in terms of educational level, statistical infrastructure and culture. The first categorisation is into those countries in which ages and dates of events are generally reported with considerable accuracy and those in which this is not the case. Typically, quality of age reporting and quality of date of event reporting are closely linked, so they can be considered together.

### *Countries with good age and date reporting*

Countries with high educational levels and countries of East and South-east Asia typically have good age reporting and good date of event reporting. In these settings, a census question on household deaths by age and sex in some recent period such as the last 12 months or since some widely observed festival such as Easter or the end of Ramadan can provide the basis for estimates of adult mortality. However, even in such countries, household deaths in the last 12 months are likely to underestimate total mortality to some extent because of deaths to single person households or more generally the dissolution of households as a result of a death. Evaluation and possible adjustment of the data is therefore necessary. Such evaluation is most effective when data from two censuses are available, to allow the application of techniques that do not assume population stability. These techniques work much better when age reporting is accurate, so these countries are well suited for both the collection of reasonably accurate data and the successful application of evaluation techniques.

Questions on deaths in some recent reference period cannot usefully be included in household sample surveys unless the samples are very large. Adult deaths are rare events, even at the oldest ages, and small samples cannot provide the precision required for rate estimates. As a rough rule of thumb (and mortality level is an important variable) Blacker and Scott (1974) suggest a minimum sample of 20,000 households if a short reference period of 12 months or less is to be used, though if estimating the age pattern of adult mortality is an important goal, a substantially larger sample will be required. Sample size can be reduced by increasing the length of the reference period, but recall bias and structural bias become potentially larger the longer the reference period.

### *Countries in which ages and dates are poorly reported*

In countries with low educational levels, with the exception of those in East and south-east Asia, ages are reported with considerable error (particularly at higher ages, and particularly for proxy reports on ages of the dead) and dates of events are generally poorly reported also (unless

some very well-known event or religious festival can be used to define a reference period). The combination of these errors means that reporting of deaths in some reference period is likely to be incorrect (possibly too high or too low), the ages of the deceased persons are likely to be wrong (generally with bias as well as random error) and the ages of the living are likely to be wrong (also generally with bias). Under such circumstances, a census question on deaths in a reference period is unlikely to produce accurate data and techniques for evaluation and adjustment of the data are unlikely to work well. A country in this category is encouraged to include such a question in a census or large sample survey, particularly if a well-defined event or festival can be identified in the preceding year, an age-sex distribution from a previous census in the last 15 years exists and international migration can be regarded as negligible. However, the country should not rely exclusively for adult mortality information on the data from such a question. An example of a census that appears to have collected usable information with such a question even in a setting of poor age and date reporting is the 1988 census of Senegal (Pison and others, 1995).

Questions on survival of mother and survival of father are also recommended for inclusion in a census or large sample survey in countries in this category. Although the accuracy of the survivorship information is uncertain, particularly for young respondents, and the time reference of the estimates is often quite far in the past (and may be misestimated if trends have not been approximately linear), data on parental survival provide a useful comparator for estimates derived from deaths in a reference period, and become particularly valuable once data from successive censuses become available. For a more complete discussion of the merits of parental survival data, see Timæus (1991a).

For a country that is not planning a census or large sample survey in the short or medium term, some other approach is needed. The remaining possibilities include retrospective questions on survival of relatives in a small sample household survey, a prospective survey, or the establishment of a sample registration system. The sample registration system has proved successful in India, but may have been a success there because of the relatively strong

statistical infrastructure that already existed in the country, including a long history of population censuses. It is doubtful whether the technique can be generalised to other settings with a weaker statistical infrastructure and less motivation. It is also questionable whether the statistical value of the information collected justifies the cost of data collection. Prospective surveys have also proved to be expensive, to require intensive supervision, and to be very sensitive to social upheaval. A special sort of prospective survey, the surveillance system, such as the Matlab Thana data collection system in Bangladesh, generally does not provide nationally representative estimates, though the results can none-the-less be very interesting for the time detail they provide and their potential for further analysis.

The short-term fix that remains is the inclusion in a household survey of retrospective questions about the survival of relatives. Methods have been developed for three types of relative: mothers and fathers, first spouses and siblings. No further discussion will be devoted to spouses, since the data appear always to be suspect. Data on survival of mothers and fathers have been discussed above. Such questions can be included in small sample surveys because of the long exposure period over which events accumulate, and hence a large number of events for a given sample size. However, this advantage comes at the cost of having a long and poorly-defined reference period. Attempts to reduce the reference period by asking for additional timing information, such as the year of death (Chackiel and Orellana, 1985), increase sample size requirements and also the sensitivity of the results to dating errors. No attempt has been made to turn parental survival into a direct method through collecting a parental history (year of birth and date of death if dead) analogous to a birth history. Such a history would require larger samples than those usually collected by birth history surveys, but might be worth exploring in countries with reasonably good age and date information since it would provide information on both temporal variation and age patterns of mortality, as well as making data quality easier to evaluate.

Initial experience with survey questions on survival of siblings suggested serious data quality problems. Respondents could not be expected to report siblings who died before they

were born, so overall survivorship tended to be overestimated. Summary questions also tended to lead to confusions about whether the respondent should or should not be included among surviving siblings. The method was revived in the late 1980s as a way of estimating maternal mortality (Graham and others, 1989), limiting questions to sisters known to have reached age 15 or to have married, and thus getting round the problem of omission of very early events. The method has since been further developed by the Demographic and Health Surveys (DHS) project, which has collected entire sibling histories including age at survey for surviving siblings and calendar year and age at death for dead siblings (Stanton and others, 1997). In high fertility populations, questions about siblings tend to expand sample size substantially (though sampling errors are still large in samples of the size typical of the DHS project), and the detailed sibling histories provide a basis for examining time trends and quality issues. However, assessments of data quality suggest that reports of events more than seven years before the survey are affected by recall bias, and that even recent events may be subject to some omission (Stanton and Hill, 1996). It should be noted that sibling-based approaches do not offer sample expansion advantages in low fertility populations.

Final recommendations on what information to collect to measure adult mortality must await a more complete evaluation of both parental survival and sibling survival data. It should also be pointed out that the choice of a data collection strategy depends to some extent on the use to which the data are to be put. For population forecasts, estimates of high precision and specific time reference are not required (unless there is evidence of recent and dramatic changes in adult mortality such as those associated with HIV/AIDS). For studies of program impact, epidemiological context or mortality links to social or economic change, much more accurate and detailed information is required.

## 2. *Data collection and analysis strategies for countries with deficient registration*

Countries with deficient death registration can be divided into those where death registration is so incomplete that adjustment is unlikely to be possible and those where a registration system provides a useful source of mortality informa-

tion (the conventional wisdom is that this cut-off point is at around 60 per cent coverage (Preston, 1984)). Countries in the former category can be treated as described in section F.1; there is probably no easy fix for the registration system, and alternative approaches are necessary. This section is concerned with the countries with registration systems that are believed to be 60 per cent complete or more.

The essential data to collect are the age and sex structure of the population. It is this structure against which the age pattern of mortality is compared in all the indirect evaluation methods. Two censuses greatly increase flexibility, allowing for applications to non-stable populations.

The second most useful type of information concerns migration, either to reassure the analyst that migration at the national level is indeed negligible or to provide a basis for an analysis that takes migration into account. Experience on how to deal with migration is limited, but information on birthplace by age and sex, particularly if available for two censuses, and birthplace information for those who die provides a partial solution for problems resulting from immigration (compare deaths of persons born in the country with age structure of the population born in the country). Some information about persons born in the country but living in other countries, as reported by censuses in major destination countries, by age and sex, makes possible an assessment of the magnitude of the problem posed by emigration, and may make possible some rough allowance in the data evaluation. However, collection of data about natives living in other countries is beyond the authority of the country itself, so there is not much that can be done to remedy the lack of such data.

A number of methods have been outlined above for the evaluation of registered deaths and the estimation of adjustment factors for coverage errors. If only one census age distribution is available, the Brass Growth Balance method (Brass, 1975) is to be preferred, since it tends to be more revealing of data errors than alternative methods. If two census age distributions are available, the General Growth Balance (Hill, 1987) method is preferred. The method allows for non-stability, explicitly estimates changes in census coverage, can incorporate

information on migration easily and intuitively, and can be truncated at any upper age to avoid problems arising from increasing data errors at old ages. The graphical basis of the method also provides clues as to whether the method's assumptions (the most important of which is constant levels of coverage at all ages) are violated.

#### G. ESTIMATES OF ADULT MORTALITY FOR POPULATION SUBGROUPS

Survival of relative based methods are not convenient for estimating adult mortality differentials for population subgroups because the residence of the respondent is not necessarily the same as the place of exposure of the relative being reported on. It might be argued that the parents of respondents who live in the area in which they were born are likely also to have lived their lives in the same area, but estimates based on this subgroup will potentially be affected by selection bias: the parents of those who stay may have higher or lower mortality than the parents of those who leave. Methods based on adjustments of registered deaths or deaths reported in a reference period prior to a census are somewhat better, though information on birthplace of the deceased will be useful. Coverage of deaths for those born and dying in a region can be estimated by comparisons with age distributions of those born in the region and still resident in the region, a group that will normally be a substantial majority of the region's population. Such an approach is not likely to work well for urban areas, since a high proportion of the population may be migrants, nor for socio-economic groups, between which migration may be even more common. Evaluation techniques can still be tried in such circumstances (and with a technique such as the General Growth Balance method problems with the application should be evident in the graphical display) as long as deaths can be tabulated by the appropriate characteristic, such as birthplace or education.

The Sample Registration System in India provides adult mortality estimates for population subgroups (states, urban and rural areas, educational levels), so a sample registration system is another possible strategy. However, the Indian SRS is able to provide such estimates because of its huge sample size, and such a strategy may

be beyond the reach of the majority of countries.

## H. CONCLUSION

Our knowledge of levels and patterns of adult mortality in the developing world is improving. Civil registration systems are only holding their own in terms of the proportion of population covered by "complete" registration. Alternative sources of data, particularly the Indian Sample Registration System and a question on deaths in the 12 months before the Chinese 1990 census, have greatly increased since the early 1970s the proportion of the population for which we have reasonable estimates. There are still a large number of countries, however, particularly in sub-Saharan Africa, North Africa and the Middle East, for which we do not have good estimates. In such circumstances, a series of fairly regular censuses combined with questions on deaths by age and sex in some reference period in the most recent census or in a large household survey, with additional questions on parental survival, will often prove sufficient to get reasonable estimates of levels, though not of trends or fluctuations, of adult mortality. If such questions cannot be included in a census or large household survey, and a sample registration system is not feasible, the use of parental survival questions that include some time frame (such as whether the parent died before or after the respondent reached age 15) and a sibling survival history in a smaller household survey may be a satisfactory approach, but results need to be interpreted cautiously.

## REFERENCES

- Bennett, N. G., and S. Horiuchi (1981). Estimating the completeness of death registration in a closed population. *Population Index*, vol. 47, No. 2, pp. 207-221.
- Blacker, J. G. C., and C. Scott (1974). *Manual on Demographic Sample Surveys in Africa*. Addis Ababa: United Nations Economic Commission for Africa; United Nations Educational, Scientific and Cultural Organisation.
- Brass, W. (1975). *Methods for estimating Fertility and Mortality from Limited and Defective Data*. Chapel Hill, NC: University of North Carolina, Laboratories for Population Statistics.
- \_\_\_\_\_, and E. Bamgboye (1981). The time location of reports of survivorship: Estimates for maternal and paternal orphanhood and the ever-widowed. Centre for Population Studies Working Paper No. 81-1. London: London School of Hygiene and Tropical Medicine.
- Brass, W., and K. Hill (1973). Estimating adult mortality from orphanhood. In *International Population Conference, Liège*, vol. 3, pp. 111-123. Liège: International Union for the Scientific Study of Population.
- Carrier, N. (1958). A note on the estimation of mortality and other population characteristics, given deaths by age. *Population Studies*, vol. 12, No. 2, pp. 149-163.
- Chackiel, J., and H. Orellana (1985). Adult female mortality trends from retrospective questions about maternal orphanhood included in censuses and surveys. *International Population Conference, Florence, 1985*, vol. 4, pp. 38-52. Liège: International Union for the Scientific Study of Population.
- Fargues, P., and Y. Courbage (1972). Working paper for UNESOB Expert Group Meeting on Mortality, Beirut. Unpublished manuscript.
- Graham, W. J., W. Brass and R. W. Snow (1989). Estimating maternal mortality: The sisterhood method. *Studies in Family Planning*, vol. 20, No. 3, pp. 125-135.
- Hill, K. (1987). Estimating census and death registration completeness. *Asian and Pacific Population Forum*, vol. 1, No. 3, pp. 8-13, 23-24.
- \_\_\_\_\_, and T. J. Trussell (1977). Further developments in indirect mortality estimation. *Population Studies*, vol. 31, No. 2, pp. 313-33.
- Pison, G., K. Hill, B. Cohen and K. Foote, eds. (1995). *Population Dynamics of Senegal*. Washington, D. C.: National Academy Press.
- Preston, S. H. (1984). Use of direct and indirect techniques for estimating the completeness of death registration systems. In *Data Bases for Mortality Measurement*, pp. 66-76. New York: United Nations, Sales No. E.83.XIII.3.
- \_\_\_\_\_, and N. Chen (1984). Two census orphanhood methods for estimating adult mortality, with applications to Latin America. Unpublished manuscript, University of Pennsylvania.
- Preston, S. H., and N. G. Bennett (1983). A census-based method for estimating adult mortality. *Population Studies*, vol. 37, No. 1, pp. 91-104.
- Stanton, C., and K. Hill (1996). Modelling Maternal Mortality in the Developing World. Paper presented at the 1996 Annual Meeting of the Population Association of America, New Orleans, Los Angeles, 1996.
- Stanton, C., N. Abderrahim and K. Hill (1997). *DHS Maternal Mortality Indicators: An Assessment of Data Quality and Implications for Data Use*. Demographic and Health Surveys Analytical Report No. 4. Calverton, Maryland: Macro International.
- Timæus, I. (1986). An Assessment of methods for estimating mortality from two sets of data on maternal orphanhood. *Demography*, vol. 23, No. 3, pp. 435-450.
- \_\_\_\_\_, (1991a). Measurement of Adult Mortality in Less Developed Countries: A Comparative Review. *Population Index*, vol. 57, No. 4, pp. 552-568.
- \_\_\_\_\_, (1991b). Estimation of adult mortality from orphanhood before and since marriage. *Population Studies*, vol. 45, No. 3, pp. 455-472.
- \_\_\_\_\_, (1992). Estimation of adult mortality from paternal orphanhood: A reassessment and a new approach. *Population Bulletin of the United Nations* 33.
- \_\_\_\_\_, B. Zaba and M. Ali (1997). Estimation of Adult Mortality from Data on Adult Siblings. Paper presented to British Society for Population Studies Annual Conference, Exeter, United Kingdom, September 1997.
- United Nations (1983). *Manual X: Indirect Techniques for Demographic Estimation*. New York: United Nations, Sales No. E.83.XIII.2.
- \_\_\_\_\_, (1997). *World Population Prospects: The 1996 Revision*. New York: United Nations, Sales No. E.97.XIII.16.
- \_\_\_\_\_, (various years). *Demographic Yearbook*. New York: United Nations.

# IV. CAUSES OF DEATH IN DEVELOPING COUNTRIES AND IN COUNTRIES WITH ECONOMIES IN TRANSITION: AN OVERVIEW OF CURRENT KNOWLEDGE, DATA AVAILABILITY AND NOVEL METHODS OF DATA COLLECTION

*Eduardo Arriaga\**

## A. INTRODUCTION

Information on causes of death is a fundamental tool for public health policies, since it indicates from which diseases the population is dying. To know the level of mortality in a country is one of the first steps in mortality analysis, but to make such analysis policy-relevant it is necessary to consider causes of death. Unfortunately, most of the countries of the World do not collect information on causes of death. As a consequence, public health measures are based on guesses about the prevalence of the leading diseases in a country.

Information on causes of death is collected through a registration system that has to be related to hospitals and that requires reporting the causes of death, if possible certified by a medical professional. To establish an effective death registration system requires a long administrative process and co-ordination of the various agencies and geographic areas of the country. One of the few current advantages for improving or creating such a registration systems is the great development in communication systems during recent years as a consequence of advances in the electronic and computer sciences. Countries should take measures to create or improve vital registration system because the great potential for planning.

This article presents first, an overview of the completeness of the registration of deaths and causes of death in a range of countries; second it deals with the most frequently encountered problems in collecting cause of death statistics; finally it considers some possibilities

of obtaining information of causes of death for those countries where they are not now available.

## B. AVAILABILITY OF DATA AND COMPLETENESS OF DEATH REGISTRATION

Developed countries, with high levels of education, high degrees of urbanisation and an advanced communications network also have a complete death registration system. In other countries, the situation is different. Completeness may vary greatly from country to country, depending on the level of development that they have achieved. North America, Europe, Australia and New Zealand, and some countries of Asia have achieved complete death registration, although a few of them have problems in accurately registering infant deaths. Most of the under reporting of infant deaths occurs at very young ages, from birth to 3 or 4 months of age, as is the case of some countries that were previously part of the Soviet Union (Kinkade and Arriaga, 1997).

In other regions of the world, the situation is different. For instance, around half of the countries of Latin America have an acceptable degree of completeness of death registration. However, Brazil, with the largest population in the region, is among those countries with rather incomplete registration.

The United Nations publishes an assessment of death registration coverage in each country of the world. The classification is not *complete* or *incomplete*, but whether or not the country has a registration system with at least 90 per cent of all deaths registered. Findings for most recent dates are reported in table 3.

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\*Population Division, Bureau of the Census, United States Department of Commerce, Washington, D.C.

TABLE 3. COMPLETENESS OF DEATH REGISTRATION, MAJOR REGIONS, MID-1990s

Region	Countries with Death Registration with a completeness of	
	90 per cent or more	Less than 90 per cent
Europe	50	0
North America, Central America and the Caribbean	28	9
South America	8	6
Africa	9	51
Asia	19	33
Oceania	12	13

Source: United Nations, *Population and Vital Statistics Report, Series A, Vol. XLIX* (New York, United Nations, 1997).

The United Nations classification refers to registration of all deaths, without reference to information on causes of death.

The World Health Organization has been publishing reports on deaths by sex, age and cause for the last six decades. Most of the countries that provide such information are found in Europe and the other developed regions. More recently, the Organization's *Statistical Annual* has started to publish information on deaths by cause for a few developing countries that have more complete registration. They are:

Argentina	Paraguay
Chile	Puerto Rico
Colombia	Republic of Korea
Costa Rica	Singapore
Cuba	Trinidad and Tobago
Mauritius	Uruguay
Mexico	Venezuela

In addition, the World Health Organization has also published information for other developing countries, but only for selected years and with some acknowledged degree of incompleteness. These countries collect information on causes of death, but they do not tabulate them every year. They are:

Barbados	Jamaica
Brazil	Nicaragua
Ecuador	Panama
Egypt	Peru
El Salvador	Saint Lucia
Guatemala	

Hong Kong routinely collected and publishing information of cause of death, but since once again becoming part of China it is not known

whether the information will be published separately or will be included in the information that China publishes for selected urban and rural areas.

The World Health Organization gives information on the completeness of the registration of deaths for a few countries with data on causes of death. Unfortunately, the estimation of the completeness is not a result of an analysis of the registration system, but it is estimated by comparing of the number of registered deaths with an expected number of deaths from population projections made by the United Nations. Although this information about the completeness of the death registration is not perfectly accurate it is very useful.

For most countries of the World, there is not an official or acceptable estimate of the completeness of death registration. However, it is possible to estimate the completeness of death registration (Bennett and Horiuchi, 1981; Brass, 1975; Preston, 1983; Preston and Bennett, 1983). These techniques do not offer a perfect solution; their results are often biased because the actual populations to which they are applied do not meet the assumptions required by the models. In addition most of the techniques do not evaluate deaths under age 5. Infant and child mortality in countries with incomplete vital statistics are estimated using indirect techniques (Brass and others, 1968; Feeney, 1980; Palloni and Heligman, 1985; Sullivan, 1972; United Nations, 1983) or direct estimates based on retrospective surveys such as the Demographic and Health Surveys.

In Latin America and the Caribbean there are several countries that may be considered as having acceptable statistics on causes of death:

Argentina, Costa Rica, Chile, Cuba, Uruguay, Mexico, Trinidad and Tobago, Puerto Rico, and Barbados. The other countries of the region may have problems in registering cause of death.

In Africa, some of the North Africa countries have complete or nearly complete registration of deaths and, as Egypt, may also collect information on causes of death. However, only the island of Mauritius regularly collects and publishes cause of death statistics.

In Asia, few countries publish cause of death statistics. Hong Kong published cause of death reports before becoming a part of China. Singapore has good information on cause of death; recently, the Republic of Korea also has begun to publish cause of death statistics, but the quality of the information is unknown. Other countries in Asia, such as Malaysia, Thailand, Indonesia, Philippines and others do not publish cause of death, but are making efforts to improve their vital registration systems. In Malaysia the registration of vital events is now considered complete.

In some countries, the information is complete for special areas, but not for the whole country. Thus, the two largest countries, China and India, do not have complete death registration systems, but both publish some information on causes of deaths derived from their sample registration systems. The classification of causes used by India differs from the World Health Organization system and hence is not included in the *Statistical Annual*. China is included, although only for selected rural and urban areas. Brazil is another case where three large regions of the country (South, SouthEast and Central West) have acceptable information, but two others (North and NorthEast) do not.

The overview of the conditions and completeness of the cause of death statistics in developing countries shows that the situation is not encouraging: few countries collect and report causes of deaths, and those countries are the most developed of the developing countries. The mortality levels in most developing countries indicate a substantial excess of mortality or premature deaths in young adult ages, but there are no acceptable statistics to guide planners in formulating adequate public health policies.

### C. QUALITY OF INFORMATION ON CAUSES OF DEATH

There is another dimension of the problem that has to be discussed before attempting to present partial solutions. There are countries where death registration is complete, and where cause of death statistics are collected and published. However, their classification of deaths by cause shows a substantial proportion of deaths registered with unknown causes. Figure 6 presents the proportion of all deaths that were classified as "Signs, symptoms and other ill-defined conditions" together with "senility without mention of psychosis" in selected developing countries or in countries with economies in transition.

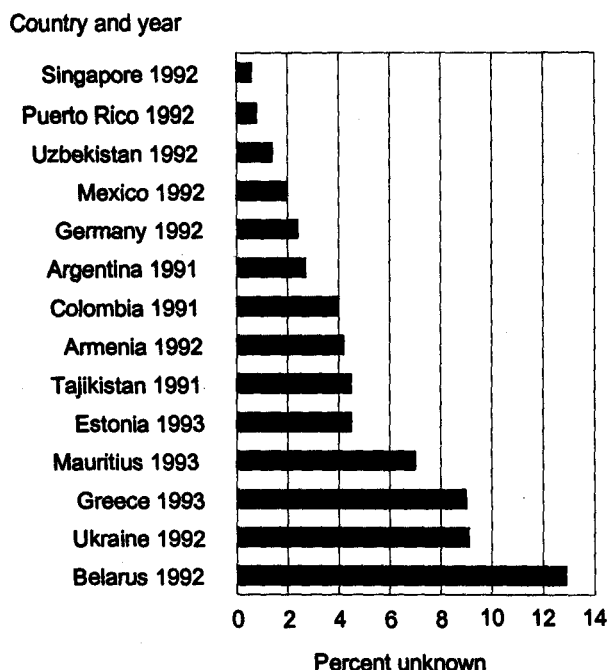
The proportion of unknown cause of death is not the same in all ages. Apparently the proportion due to unknown causes increases with age. It is also higher among the very young than among young adults, as shown in figures 7 to 10. The proportion of unknown causes of death is lower among young adult males than among females of the same ages. This does not reflect fewer male deaths of unknown cause, but rather the much larger number of male violent deaths (accidents, suicides, and homicides) increases the total number of deaths and makes the proportion of unknown deaths smaller.

The problem of unknown cause of death is not the only one. The reported cause of death can also be erroneous, if the reporting person does not know the cause of death. In poor and uneducated populations there is evidence that family members often do not know the cause of death of their relative (Snow and others, 1992).

Another source of error in reported cause of death is incorrect medical certification. If the deceased has been under medical treatment, there is a good chance that the physician will properly report the cause. But if the physician has not treated the person who died, the reported cause may not be accurate (Moriyama, Loy and Robb-Smith, 1994). There may be a tendency to over-use one or another cause such as "cardiac arrest", or to misclassify deaths due to a specific cause such as "motor vehicle accident" into a less specific category such as "other violence".



**Figure 6. Percent of deaths of unknown cause, selected countries**



Source: World Health Organization, 1994 *World Statistics Annual* (Geneva, WHO, 1995).

Finally, another source of error could be that some countries have not classified certain causes of death properly because new diseases may not yet have a proper code, or because families of the deceased person do not want the cause to be known. The case of deaths from AIDS is a good example. Based on a detailed analysis of reported single causes of deaths, it is almost certain that some male deaths from AIDS in Buenos Aires were classified as Cause 279, Disorder of the Immune System in 1991 (Arriaga, 1995b).

### *1. Unknown causes of death*

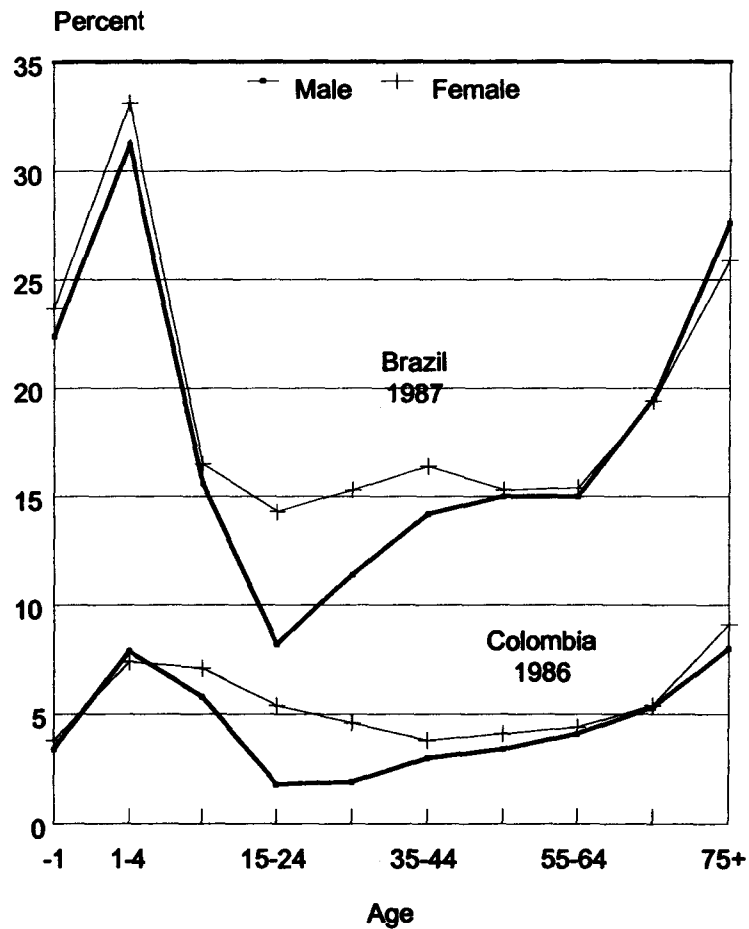
The number of deaths without a certification of the cause also affects the quality of the information on causes of death. Deaths for which the cause is unknown are likely to be concentrated in social groups with less education and income, and to live under poorer conditions than the rest of the population. These characteristics

are associated with higher mortality (Vallin, 1979), and hence with different causes of death. Hence, the unknown causes of death are not only likely to be concentrated in groups with higher mortality, but also with a distribution of deaths by cause different from that reported for the general population.

Under-registration of deaths may further distort the reported distribution of causes of death. Unregistered deaths are likely to have a different distribution of causes than those that were registered with the cause specified. As in the case of deaths of unknown cause, most of these unregistered deaths may be experienced by social groups with distributions of causes that are different from those of registered deaths.

Facing the fact that information on causes of death is imperfect, the question is whether or not they should be used at all. The answer is yes. The information should be used but with

Figure 7. Percent of registered deaths of unknown cause by age and sex, Brazil, 1987 and Colombia, 1986



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).

Figure 8. Percent of registered deaths of unknown cause by age and sex, Chile, 1962 and 1987

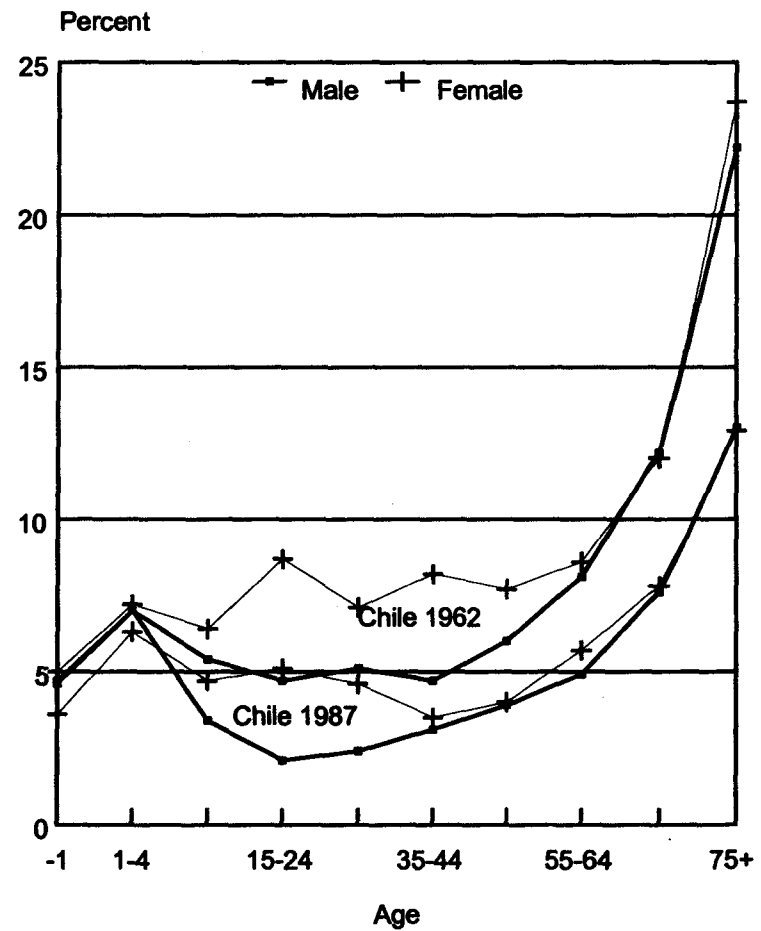
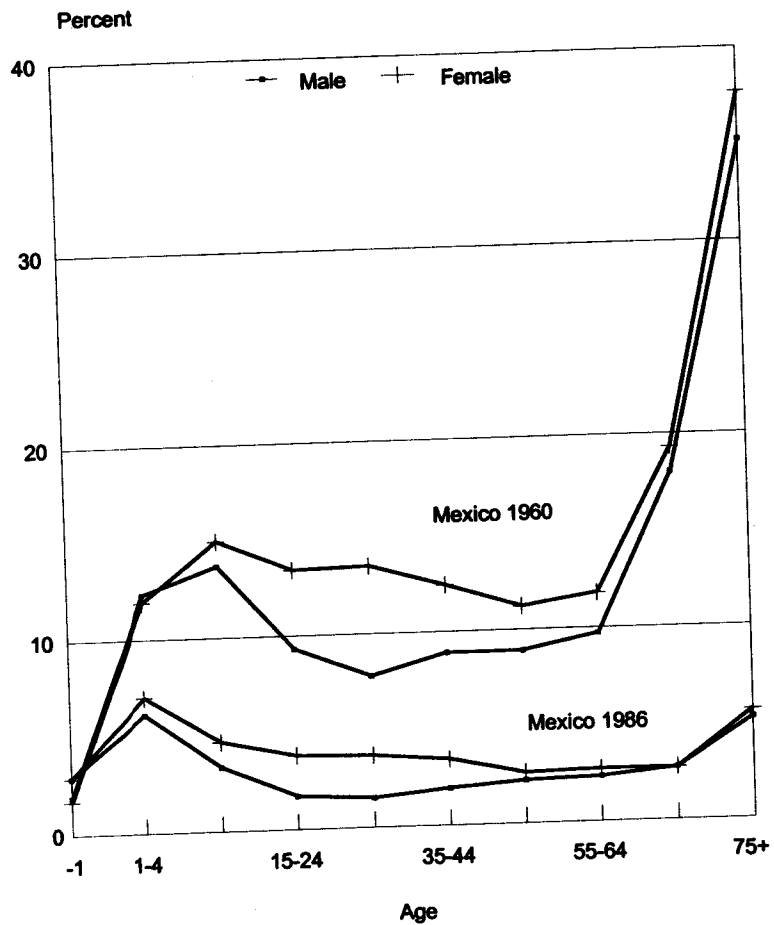
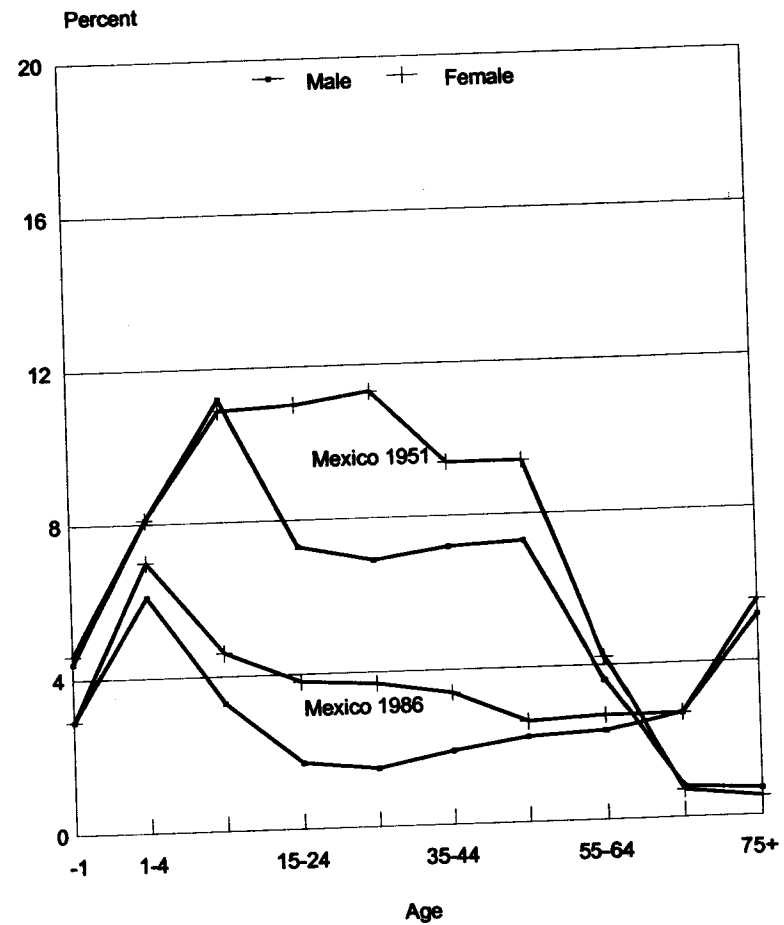


Figure 9. Percent of registered deaths of unknown cause by age and sex, Mexico, 1960 and 1986



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).

Figure 10. Percent of registered deaths of unknown cause by age and sex, Mexico, 1951 and 1986



the understanding that it is not 100 per cent accurate, and that the results of the analysis should be taken with caution.

For example, Colombia has an incomplete death register, but the information is still useful, particularly for identifying large differences of mortality between sexes for certain causes of death. Large differences of deaths by sex could indicate that one of the sexes is behaving differently than the other. This is specifically the case of violent deaths (including accidents, homicides and suicides) in Colombia in 1986. The numbers of deaths for 1986 for selected ages were:

TABLE 4. NUMBERS OF DEATHS BY SEX AND SELECTED AGE GROUPS, COLOMBIA, 1986

	15-24	25-34	35-44	45-54
Males	6 795	7 641	4 027	2 227
Females	937	679	409	304

The differences between men and women on the number of accidental deaths are too large to be "normal" and they indicated a situation that requires attention in that country.

Similarly, although death registration in China is not complete, the information provided by a Sample Registration System for selected urban and rural areas shows a very high number of suicides among young women. This is not expected on the basis of experience of other countries with information on causes of death. In most countries, the mortality rates for suicide in the young adult ages is at least twice as high among men as for females. But the information for China shows a completely different situation, mainly in rural areas (table 5).

The large number of female suicides in relation to males in both areas, particularly in rural areas, indicates a unique and undesirable situation in China

Incomplete classification of deaths was also found in Brazil. Among the country's five Geographic Regions, three have fairly complete death registration and reliable information on causes of death, whereas the two regions in the

TABLE 5. NUMBER AND MORTALITY RATES OF SUICIDES PER 100,000 POPULATION BY SEX, SELECTED AGES, CHINA, SELECTED RURAL AND URBAN AREAS, 1990

	15-24	25-34	35-44	45-54
Selected rural areas				
Males	731	707	565	504
Females	1 538	1 161	605	453
Mortality rates				
Males	16.2	18.8	19.1	25.2
Females	35.1	32.0	21.7	24.5
Selected urban areas				
Males	326	483	380	264
Females	492	547	316	266
Mortality rates				
Males	6.1	7.6	8.6	8.5
Females	10.3	9.3	7.7	8.9

Source: World Health Organization, 1993 *World Health Statistics Annual* (Geneva, WHO, 1993).

northern part of the country do not. In addition, the two northern regions report large proportions of causes of death as "unknown". How do the large number of unregistered deaths and the large proportion of registered deaths of unknown cause affect the distribution of deaths by cause? Are there differences *between the complete and incomplete* distribution of deaths by cause? And if they are different, are the differences consistent with the different mortality rates in each of the areas?

The implications of these questions are evident in figure 11. The section of the country with more complete information and lower mortality has only 10 per cent of registered death classified as due to unknown cause. On the other hand, 40 per cent of the registered deaths are classified as of unknown cause in the area of higher mortality and incomplete registration of deaths. Thus, while an analysis can be conducted in the areas of acceptable registration of deaths, analysis is not possible in the area of incomplete registration of deaths; the proportion of unknown causes is too high. For the section with incomplete registration of cause of death it will be necessary to resolve the problem of deaths due to unknown cause before attempting to analyse the information.

The problem is how to distribute deaths of unknown cause. If the proportions are small,

any procedure will give acceptable results. The problem emerges when the proportion of unknown cause is large, as in the case of the two northern regions in Brazil.

Distributing a large number of deaths of unknown cause proportional to the distribution of reported cause of death is easy, but may create biases. If the number of deaths of unknown cause represents a high proportion of all deaths, other possible procedures for distributing them may be considered (Palloni, 1981).

As a case study, deaths of unknown cause from the north and north east regions of Brazil were distributed proportionally in order to see if the procedure produces something obviously wrong (figure 11). Are there any obvious irregularities when the areas with acceptable statistics are compared with the area of high degree of incompleteness and unknown cause of death? The proportion of infectious diseases is higher in the two northern regions than in the three with acceptable data, as expected, since the two "bad" regions have higher mortality. Similarly, it is to be expected that the proportion of deaths from chronic diseases will be higher in the areas of lower mortality than in the northern part of Brazil. It also seems plausible that the proportions for the other three groups of causes: violent deaths, other causes and diseases of the respiratory system. But the question remains, is the *adjusted* distribution of causes of death of the two northern part of Brazil accurate? Could such a distribution be used for planning purposes?

Based on the previous examples, we could be tempted to say that it is better to have an incomplete distribution of deaths by cause with large numbers of deaths of unknown cause than to have no information at all. There is at least always the possibility of separately analysing those areas that have acceptable levels of registration and classification of deaths.

However, there are too many countries with no death registration or with no information whatever on causes of death. The crucial question is what can be done in those countries?

#### D. COUNTRIES WITHOUT INFORMATION OF CAUSES OF DEATH

Is there any possibility of estimating cause of death patterns in countries that do not collect such information? What degree of accuracy on cause of death is sufficient for the formulation of public health programs?

Countries without death registration or without cause of death statistics cannot simply wait to have a good death register in order to have a health policy. To take surveys is an option, but a more plausible solution would be to estimate the causes of death, given the fact that there is so close a relationship between cause of death and level of mortality.

#### E. STUDIES TO DETECT PARTICULAR CAUSES OF DEATH

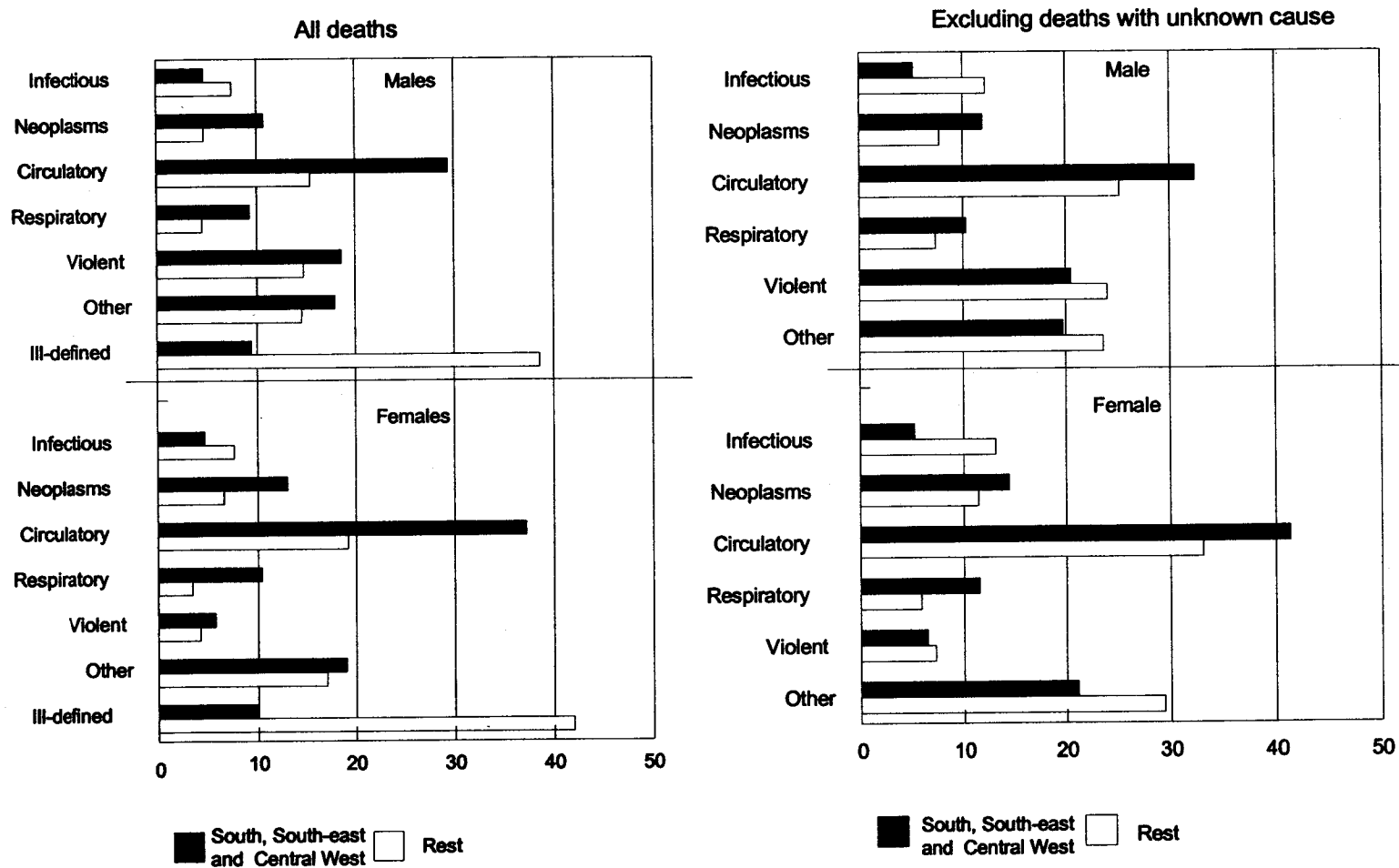
As mentioned, one possibility to know the causes of death in a population is to take a survey. However, there are two limitations to the use of surveys for investigating causes of death:

- the sample has to be large enough to permit a reliable classification of all deaths by sex, age and cause;
- the questionnaire has to be well designed, and the respondent has to know some of the principal symptoms associated with causes of death.

These two requirements, together with the inevitable limitation of resources, make surveys unsuitable as a means to encounter enough deaths so that causes by age, sex and cause can be obtained for the whole population. Given these requirements, most cause of death surveys have concentrated on specific ages.

Surveys to investigate cause of death have to use special questionnaires that determine cause on the basis of reports by a member of the household. The procedure is called "verbal autopsy", and most of these surveys have been concentrated on children and/or females in reproductive ages. A majority of the surveys investigate infant and child mortality in coun-

Figure 11. Percent distribution of deaths by cause, regions of Brazil, 1989



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).

tries where mortality is believed to be high. The purpose has been to assess specific infectious disease prevalence, to measure vaccination coverage among the young, and to check the knowledge and/or availability of health services (Macro International, 1994; Broeck, Eeckels and Vuylsteke, 1993; Mobley and others, 1996; Islam and others, 1996; Snow, 1995; Sadruddin, 1994; Khan and others, 1993; Marshal and others, 1993; Fauveau and others, 1992). Practically all the studies have found that a high proportion of child deaths involve diarrhoea.

For women in reproductive ages, the main purpose has been to estimate maternal mortality rates (Fauveau, 1995; Campbell and Ronsmans, 1995; Fikree, 1994; Kane and others, 1992) and to measure the impact of maternal HIV on infant and child mortality (Dowell and others, 1993).

In most cases, the surveys produced the expected result, that the high mortality of very young children was due to infectious and parasitic diseases, particularly diarrhoea. Other surveys have investigated the impact of malaria on infant and child mortality; they found that this disease is still responsible for a high proportion of child deaths in countries where malaria is endemic.

The survey analysts have identified some key problems with the method. Thus, although they were able to measure levels of mortality as well as groups of causes producing high mortality, the surveys could not clearly assess the mortality impact of specific infectious diseases. Various infectious diseases have similar symptoms, and hence the verbal autopsies failed to distinguish the disease. An additional reporting problem was that the respondents were not able to differentiate the symptoms asked in the questionnaire (Chandramohan and others, 1994; Martinez and others, 1993; Snow and others, 1992; Gray, 1991).

#### *1. Other possibilities*

In the long run, the best hope that we have is to improve vital registration systems so that all deaths are reported, with physicians properly certifying the final cause of death as well as underlying causes and other possibly

related conditions. But can we wait for such improvements in countries where there still is not a registration system? We should not wait.

There are several countries in Asia and Latin America, where registration systems exist, but need to be improved. The improvement cannot be made in a year; it will inevitably be a long-term project. There are some countries where the improvement is being achieved slowly but continuously, such as India and China through their Sample Registration Systems (World Health Organization, 1993).

#### *Relationship of causes of death and level of mortality*

There is a strong relationship between cause of death and level of mortality, since mortality does not decline equally for all causes at the same time. The high mortality of populations during the last century due to infectious and parasitic diseases posed a challenge to the society. The purification of drinking water, the discovery of drugs and vaccinations, improved public and personal hygiene and the elimination of some vectors largely reduced mortality from infectious and parasitic diseases. Because infectious and parasitic diseases were eliminated, general mortality was reduced (McKeown, 1976).

During this century, the elimination of certain causes of death has continued, not only of the infectious and parasitic diseases, but also of other threat to human life. This process of reducing diseases, and hence mortality, leads us to expect a strong relationship between cause of death and mortality level.

If such a relationship holds among different countries at different times, it may be useful in predicting causes of death in countries where no direct measurement is available. Of course, the relationship of causes of death and level of mortality cannot be exactly the same between countries with information from the end of the last century (or early decades of this century) and countries currently with the same level of mortality. The possibilities for reducing certain infectious and parasitic diseases and the speed of reduction is quite different now from what it was 100 years ago.

This article *does not try to prove* that such relationship holds, *but rather tries to suggest possibilities* that should be explored for use in countries without statistics on causes of death. The idea is not new (Palloni, 1981) but it should be revitalised now that we have more information and better prospects for improved methodologies.

Several countries were selected to analyse their distribution of deaths by cause at different levels of mortality and different years, shown in figure 12. As well known and expected at high levels of mortality, the majority of deaths are due to infectious and parasitic diseases. Once these diseases are eliminated, chronic diseases are left, and hence they are responsible for most of the deaths in a population.

But the distribution of causes of death for the whole country is affected by the age structure of each sub-population. Therefore, rather than using the population, it is better to examine the distribution of causes of death within each particular age group of the population. The information is taken from data published by the World Health Organization; they are by 10-year age groups, except for infant deaths and deaths in the ages 1 to 4 years.

The distribution of the causes of death within each age group also is related to the level of mortality. The relationship persists even when the examples are taken from a long period of time, from the early 1950's to the late 1980's. During that period, there were changes in public health programs and changes in the treatment of each disease. Nevertheless, the relationship between level of mortality and cause of death seems to hold.

There are some particular relationships of mortality and cause of death. Infant and child mortality from ages 1 to 14 tends to remain with a high proportion of deaths from infectious and parasitic disease. Only when mortality is very low do chronic diseases start to account for a high proportion of deaths (figures 13 through 15). From ages 15 to 45 years, a high proportion of male deaths are due to violence, while for females, with fewer violent deaths, chronic diseases account for a high percentage of the deaths. However, still in this age range, if mortality is high it reflects a high level of infec-

tious and parasitic diseases (figures 16 through 18). Over age 45, the main causes of death in countries with low mortality are chronic diseases. But again, if mortality is high, infectious and parasitic diseases will also cause a high proportion of deaths (figures 19 to 22).

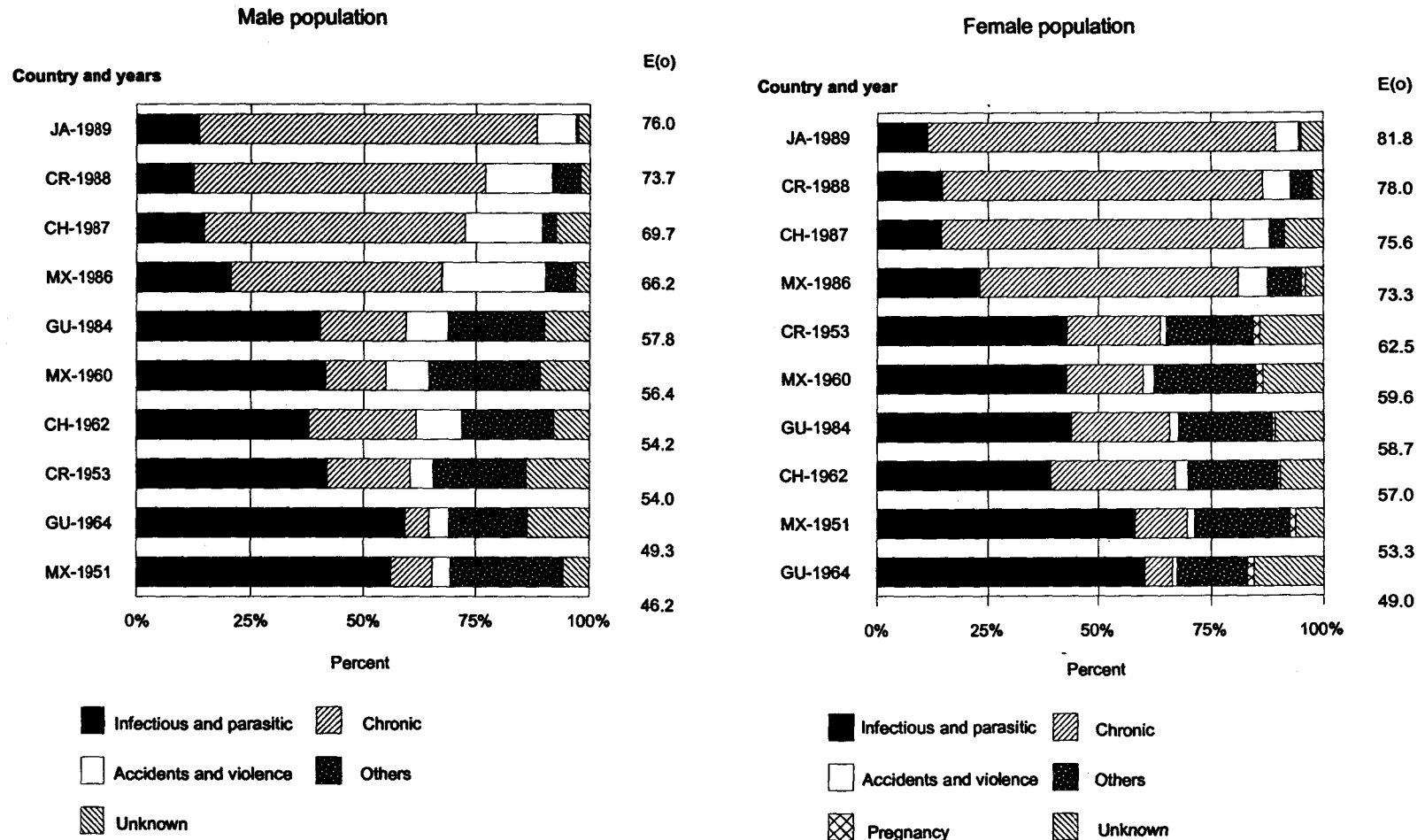
This strong relationship between cause of death and the level of mortality may help in determining procedures for estimating the cause of death. The problem is how to estimate the level of mortality in countries where there is not a death register, or where the data are not reliable. The procedures for estimating mortality and/or evaluating deaths in relation to the population by age may have biases due to errors in the data, or because they were developed under assumptions that are not present in the actual populations. However, we have mortality estimates for most of the countries of the world (United Nations, 1996; US Bureau of the Census, 1996). The estimates may contain some errors, but they cannot be too far from actuality. Therefore, since we have estimates of the level of mortality, there is a good possibility for estimating approximately the main groups of causes of death in such countries, if multivariate correlation techniques are used to relate level of mortality and groups of causes of death in each age group.

#### *The year 2000 census round and possibilities for mortality research*

In general, levels of infant and child mortality can be estimated by applying certain technique to special census questions. Techniques developed in the late 1960s and the 1970s have been widely used (Brass, 1968; Sullivan, 1972; Feeney, 1980; Johnson, 1982). It can be said that thanks to information collected in censuses and surveys, we now have reasonable estimates of infant and child mortality for most countries of the world. Unfortunately we cannot say the same regarding adult mortality. Techniques designed for estimating adult mortality based on orphanhood and widowhood data have provided some estimates of adult mortality, but such estimates are not very reliable. The information collected for use with such techniques seems to have errors that strongly affect the estimates. One of the disadvantages of the orphanhood and widowhood techniques is that the estimates of the pattern of mortality for adult ages (males



Figure 12. Percent distribution of total deaths, by cause of death



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).

Note: E(o) represents the life expectancy at birth.

and females) is nearly a model pattern, and these model patterns may often differ from the actual pattern (Arriaga, 1994).

Questions on censuses about deaths during the last 12 months prior to the census date have proved to be useful for detecting the mortality patterns and differentials between males and females at young adult ages. This census information on deaths has to be evaluated in order to obtain an estimate of the level of mortality. The information has been useful for detecting excess male mortality in relation to females, generally due to accidental death. Similarly, among women, the mortality pattern given by the information on deaths during the last 12 months shows the impact of maternal mortality on total deaths, mainly in countries of high fertility.

Questions for detecting maternal mortality with the application of the sisterhood technique have been tested with satisfactory results. The questions, put to all women over age 15, request information about whether or not the death of a sister was due to a pregnancy. The questions are used in order to estimate the mortality of young adult females in the reproductive ages, and hence maternal mortality. The technique was developed several years ago (Graham and others, 1989) and it is based on a method of sibling survivorship (Hill and Trussell, 1977). The results indicate surprisingly high levels of maternal mortality in those countries where applied (such as Niger, Bangladesh, Argentina, Tanzania). The inclusion of such questions in censuses would give us another means to estimate maternal mortality in young adult ages. The questions, to be asked of the population 15 years of age and older, are:

Do you have sisters that have reached reproductive ages?

Yes No

If Yes:

How many of them are:

Alive

Dead

If dead:

Did they die during pregnancy or delivery or during the month after delivery?

Yes No

It has also been proposed recently that the census should investigate male mortality in young adult ages (Arriaga, 1997). Thus, after asking whether or not a person has died in a household, the next question for males and females should be whether or not the death was due to an accident, homicide or suicide. The subsequent questions, only for females, ask whether the deceased person was pregnant and if she was, whether the death was related to pregnancy. For instance, after the question about deaths in the household during 12 month (or longer period), if there were any death, and after asking about the age and sex, the following questions should be ask:

Was it an accident, homicide or suicide?

Yes No

and if the deceased person was a woman and the answer to the previous question was "No",

Did she die during a pregnancy, delivery, or during the month after a delivery?

Yes No

It is known that if maternal mortality and accidental deaths are eliminated from the total deaths in young adult ages 15 to 44, the remaining mortality will be quite low. If the remaining deaths still indicate very high mortality, the most probable cause of death would be infectious and parasitic diseases and/or AIDS.

For ages 45 and over, the relationship between the level of mortality and the groups of causes of death is also strong. The fluctuations of the proportions of the groups of causes of death (infectious, chronic, violence and others) and the level of mortality are rather small, and hence there are better chances of estimating the distribution of deaths by large groups of causes.

## F. CONCLUSIONS

The preceding overview of the completeness of death registers and availability of information indicates that most of the countries of the world do not have information on causes of death. Facing this problem, what are the best possible solutions for increasing the number of countries with such information?

Complete death registers is the best solution, but in the short-term it is almost impossible to achieve because of the high level of resources required. Therefore, while countries should be encouraged and supported in their efforts to improve or create complete and reliable death registration systems, we need another solution for the near and medium terms.

Surveys conducting verbal autopsies have given good results for analysing very specific causes of death among children in selected age groups or among women in reproductive ages. The use of these kinds of surveys for detecting causes of death for the whole population is somewhat Utopian because they too will be very costly. In addition, for the reasons described, the results of these surveys are not so very different from what is expected, and hence, could be estimated with some margin of error without taking the surveys.

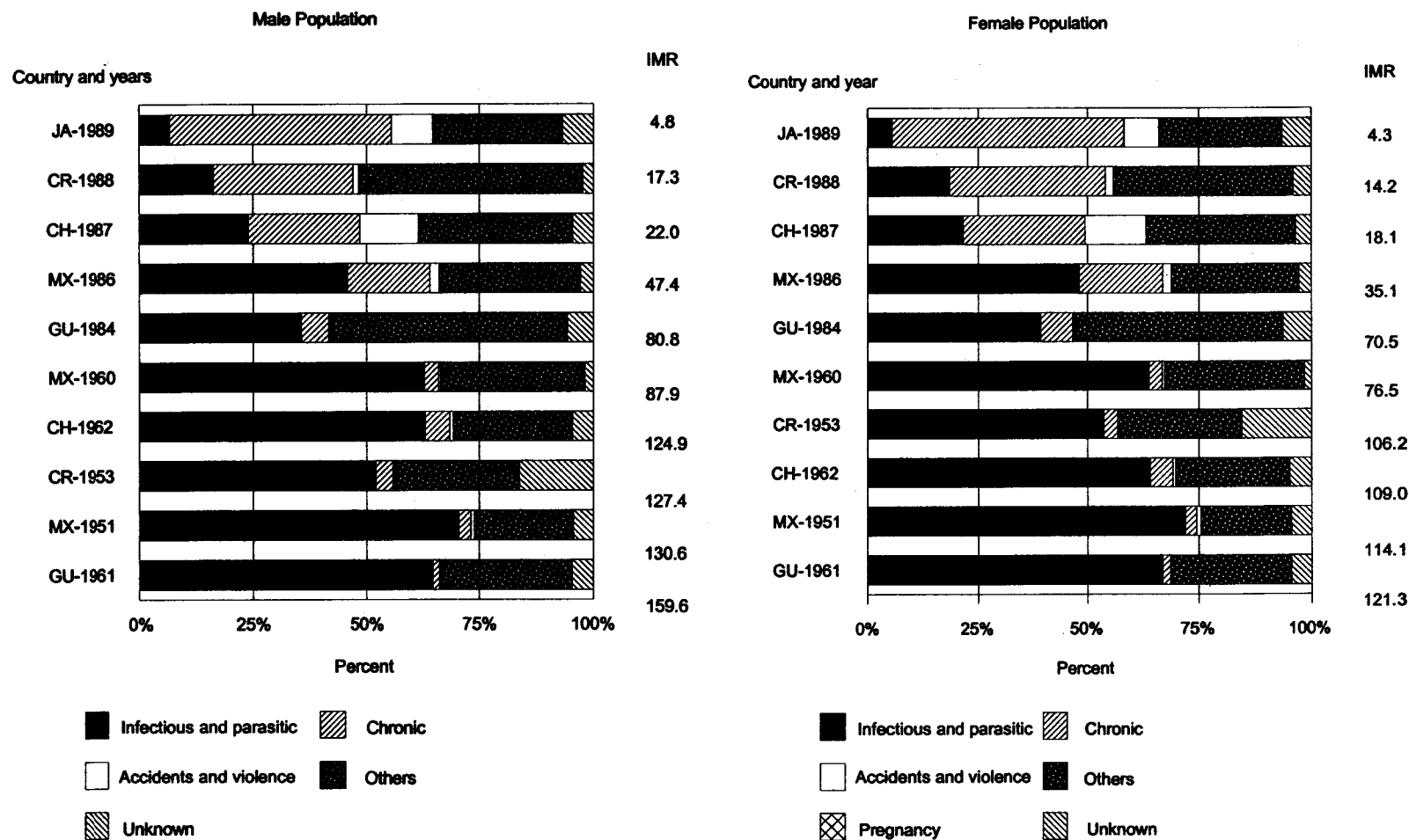
The close relationship of cause of death and level of mortality is something that should be taken into account, particularly for estimating causes of death among adults. There are two inter-related possibilities for the process of estimation. For adult mortality, the main estimation problem is in the young adult ages. The population in these ages can have quite different patterns of mortality. Men and women can have quite distinctive patterns of cause of death at the same level of mortality. Females may have very high maternal mortality, or high mortality may be due to violence or AIDS instead of maternity. For males, violent deaths could be high, or the excess could be due to infectious diseases or also AIDS. The only certainty that we have in these young adult ages is that mortality from chronic diseases is low.

Hence, if mortality is high in these ages, the process of multi-variate correlation would not necessarily give good results, because countries may have large differences in the extent of mortality due to violent deaths or maternity. Additional information is needed, and such information could, and should, be obtained from the coming year 2000 census round. We should not let pass this unique opportunity for introducing additional questions in the census for developing countries without good registration systems. The questions on deaths during the last 12 month (or larger period of time) and additional questions for detecting violent deaths or maternal deaths should be included in the coming censuses.

For ages 45 and above, the relationship between mortality level and cause of death is more certain, and hence the process of estimating causes of death based on the level of mortality would be feasible. In addition to the questions of deaths in a household during the last 12 months, the question about violent death should be asked for these ages too. We know that even when the proportion of accidental deaths is rather low in these ages, the mortality rates from those causes is not necessary lower than at young adult ages.

There are not too many opportunities in the immediate future for improving the estimates of causes of death in adult populations. After considering the limited resources available, and the possibilities of obtaining estimates from other sources, we can see that the year 2000 census round offer us an excellent opportunity. If we do not use the coming round of censuses, we will face another decade with only a few developing countries having information on causes of death.

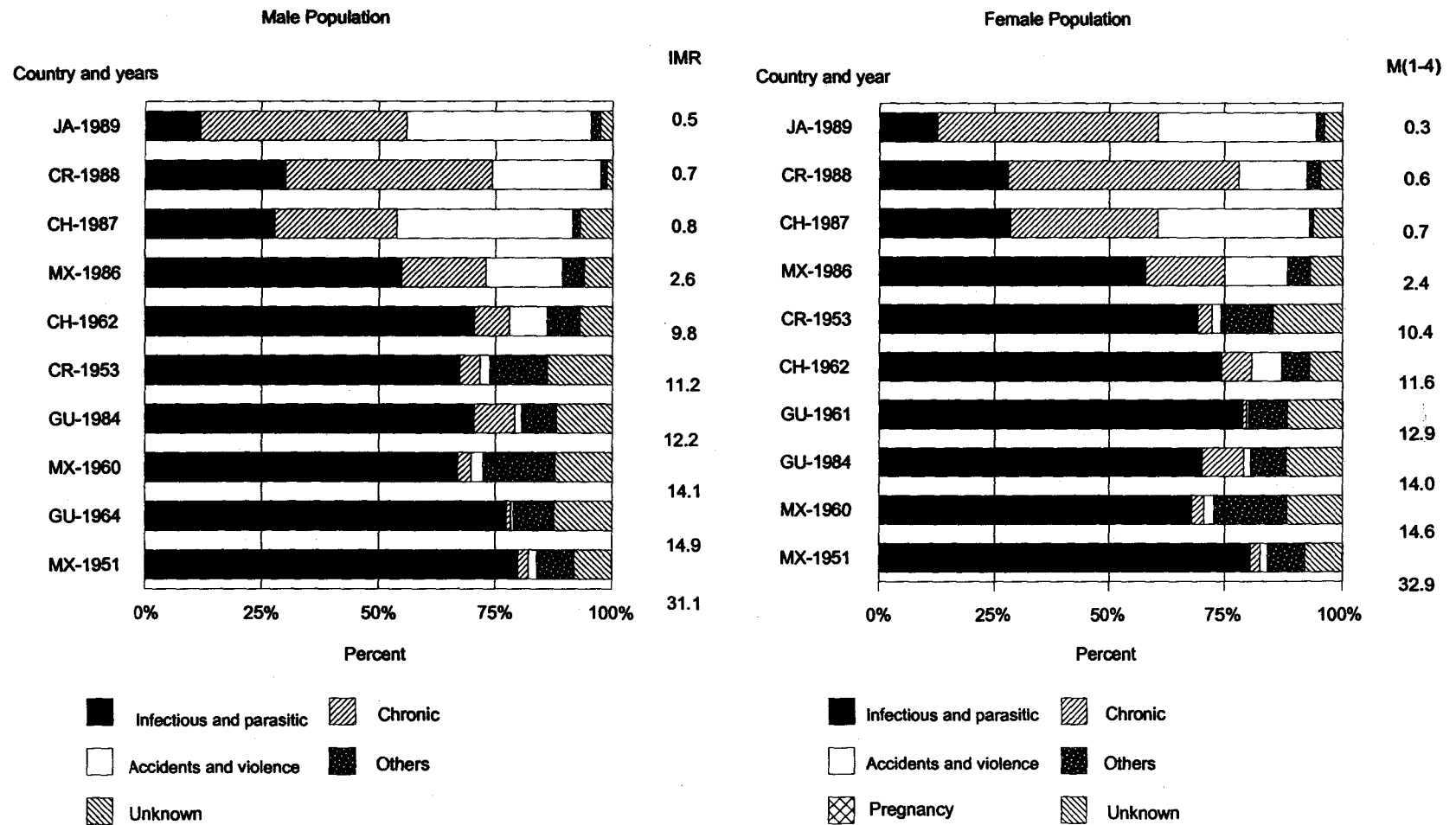
Figure 13. Percent distribution of infant deaths, by cause of death



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).

Note: IMR represents Infant Mortality Rate per thousand.

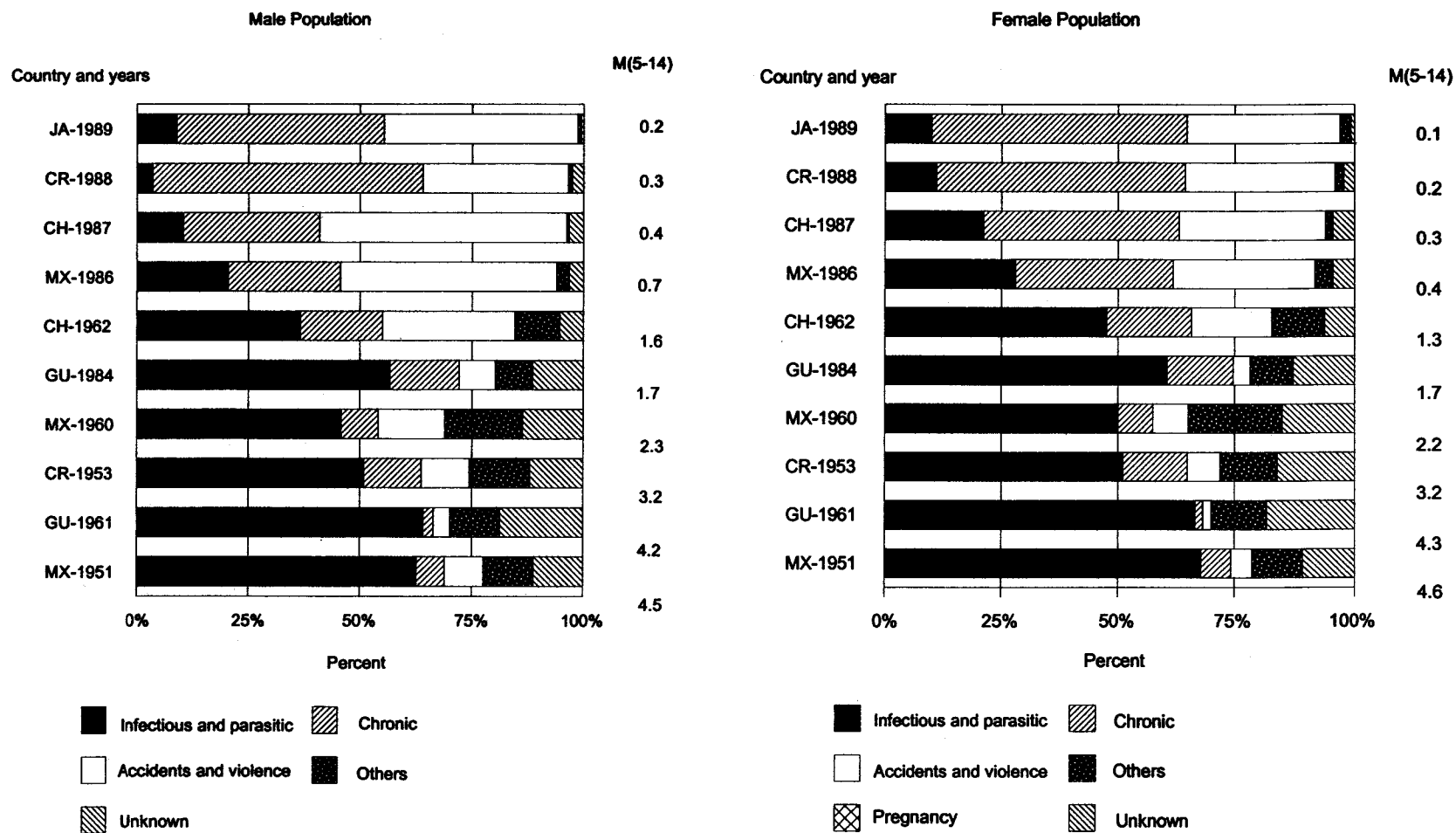
Figure 14. Percent distribution of deaths at age 1-4, by cause of death



Source: World Health Organization, 1994 *World Statistics Annual* (Geneva, WHO, 1995).

Note: IMR represents Infant Mortality Rate per thousand.

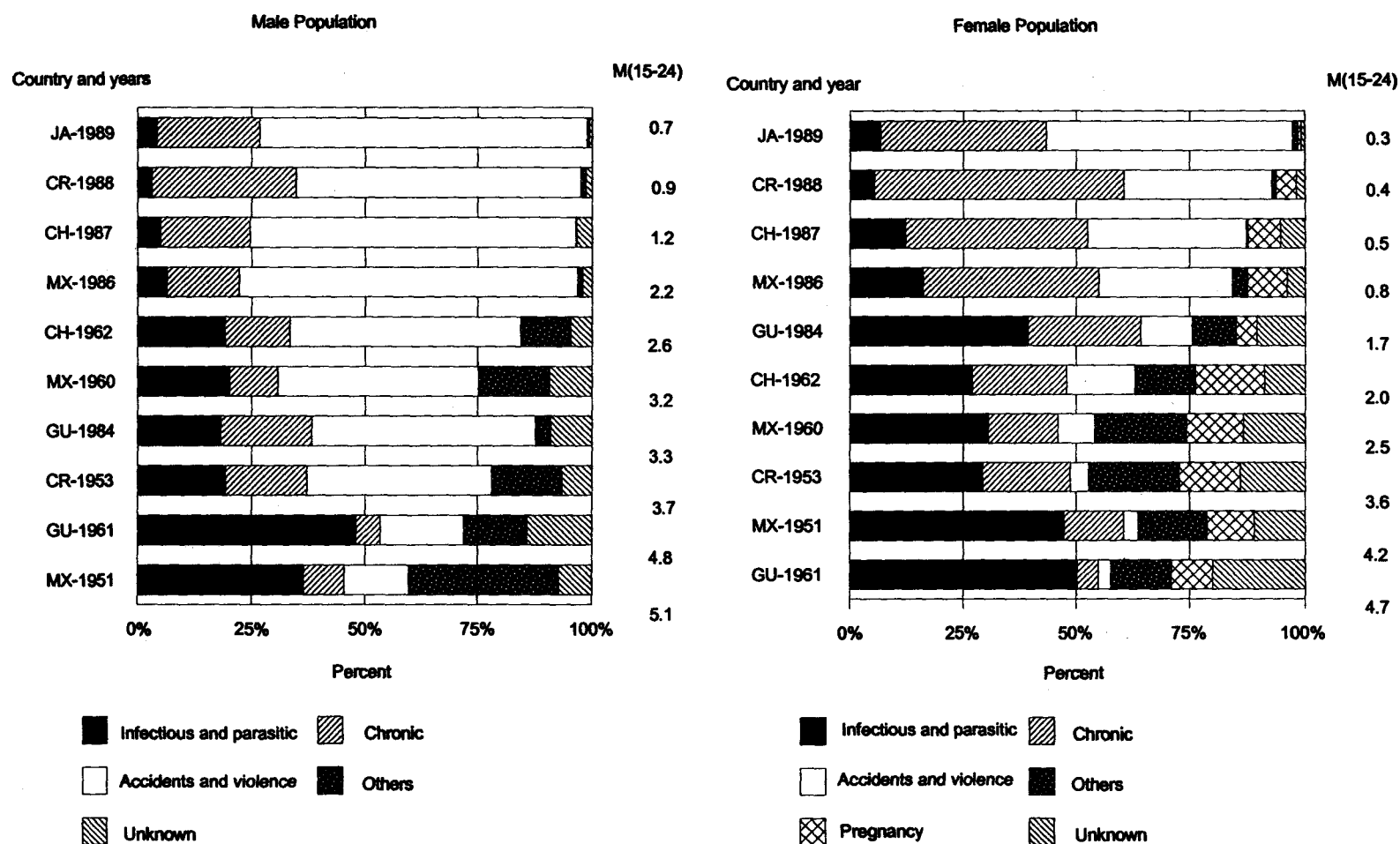
Figure 15. Percent distribution of deaths at age 5-14, by cause of death



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).

Note: M(5-14) represents the mortality rate per thousand.

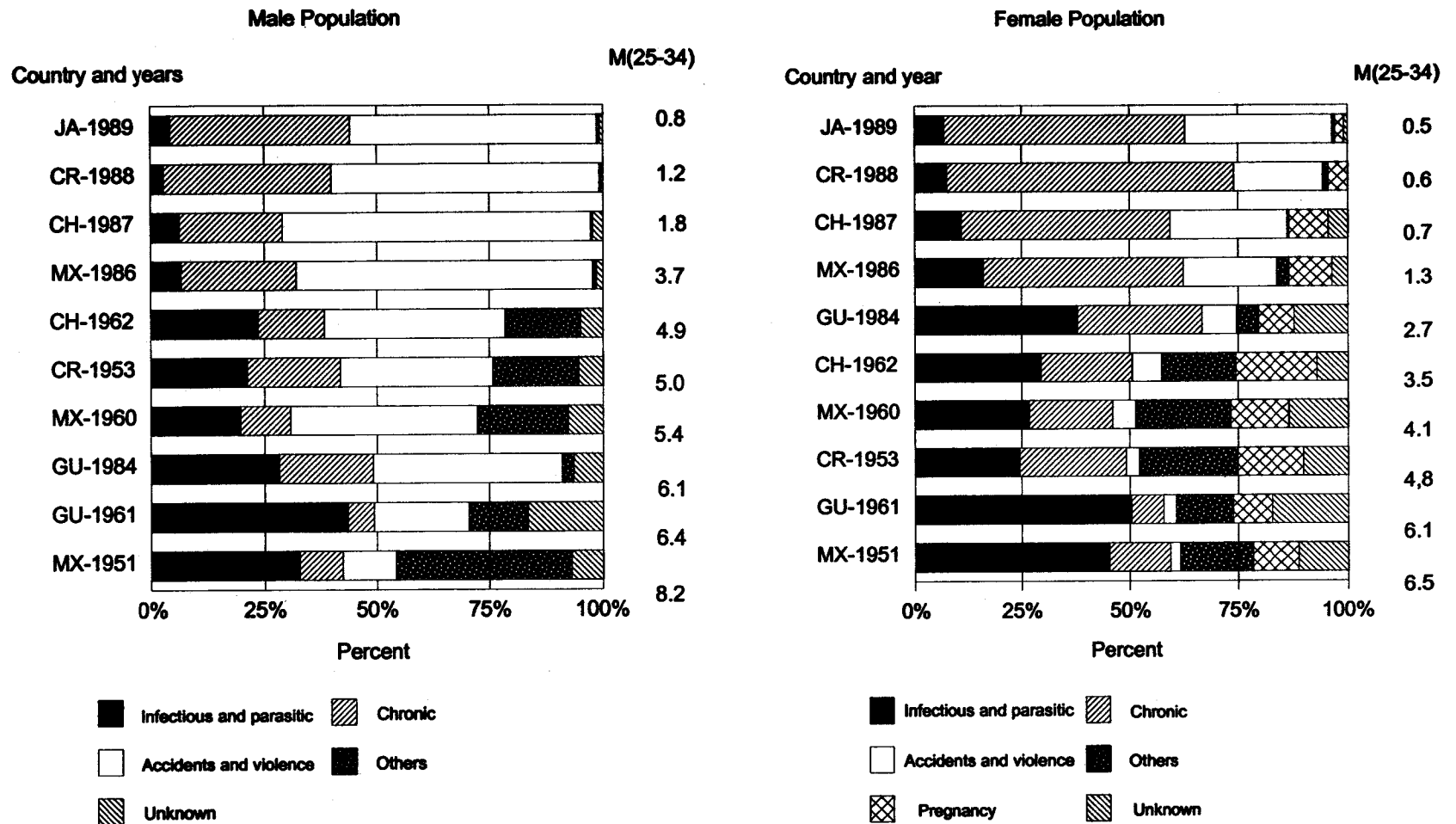
Figure 16. Percent distribution of deaths, ages 15-24, by cause of death



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).

Note: M(15-24) represents the mortality rate per thousand.

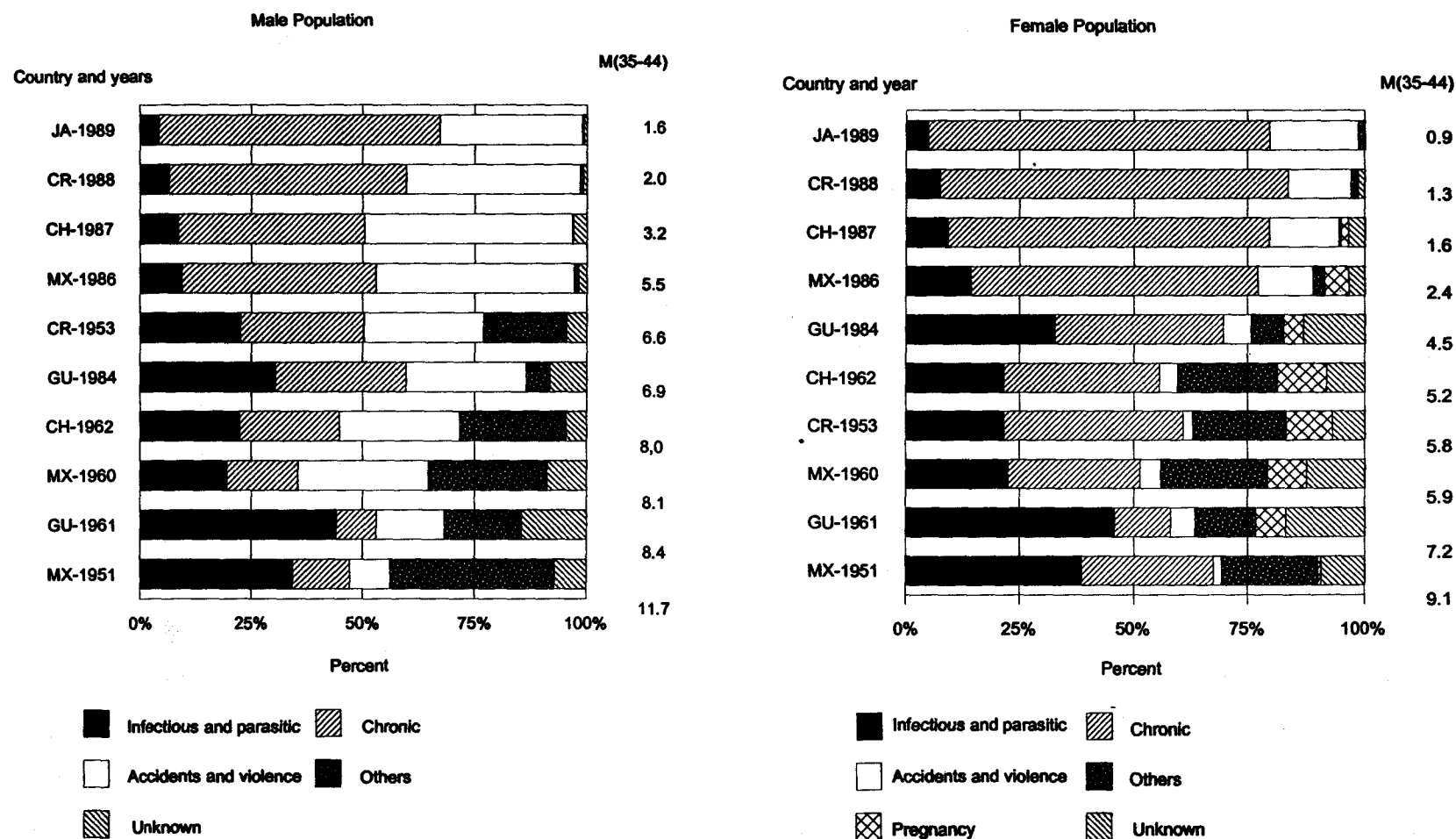
Figure 17. Percent distribution of deaths, ages 25-34, by cause of death



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).  
 Note: M(25-34) represents the mortality rate per thousand.



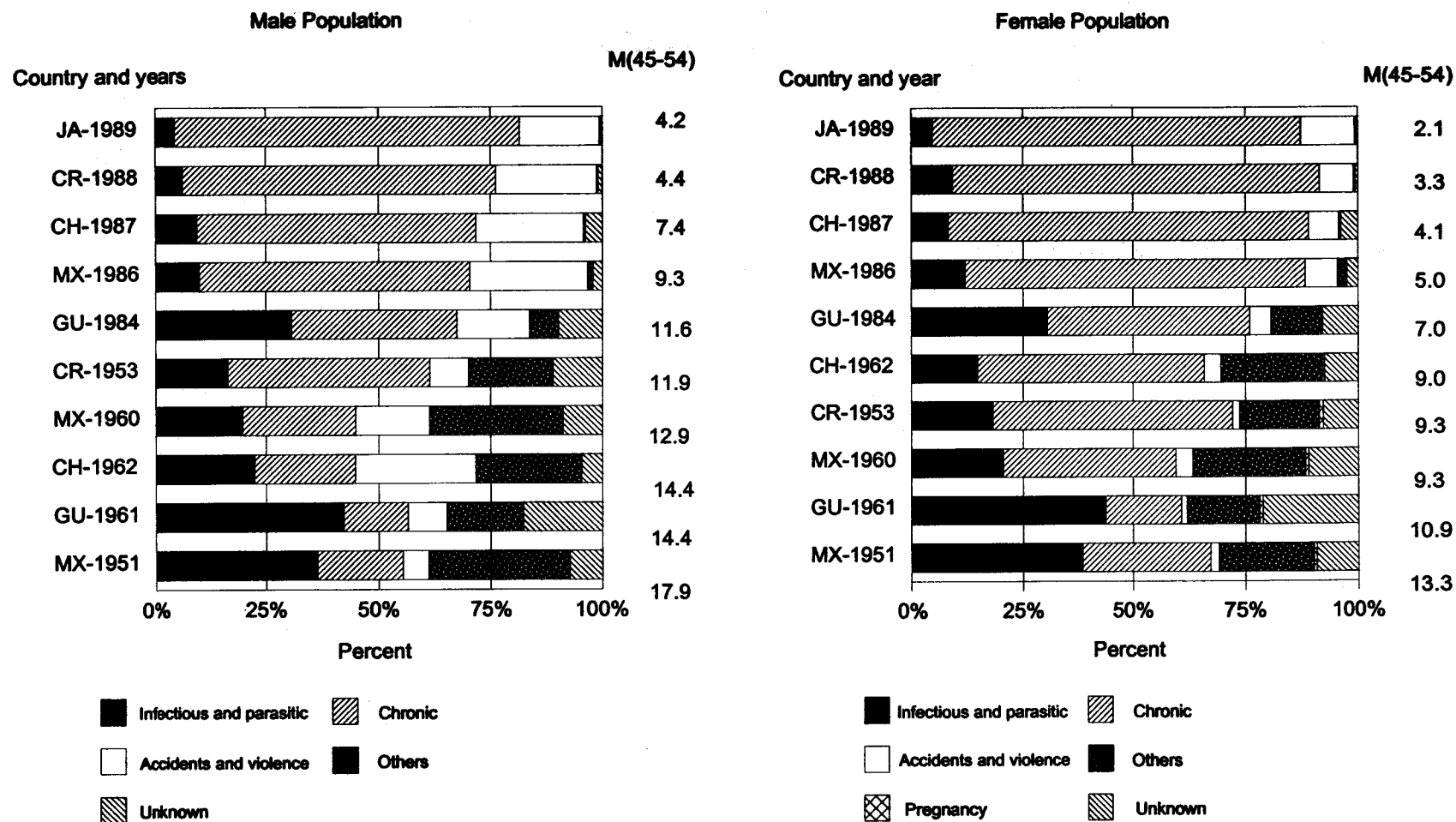
Figure 18. Percent distribution of deaths, ages 35-44, by cause of death



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).

Note: M(35-44) represents the mortality rate per thousand.

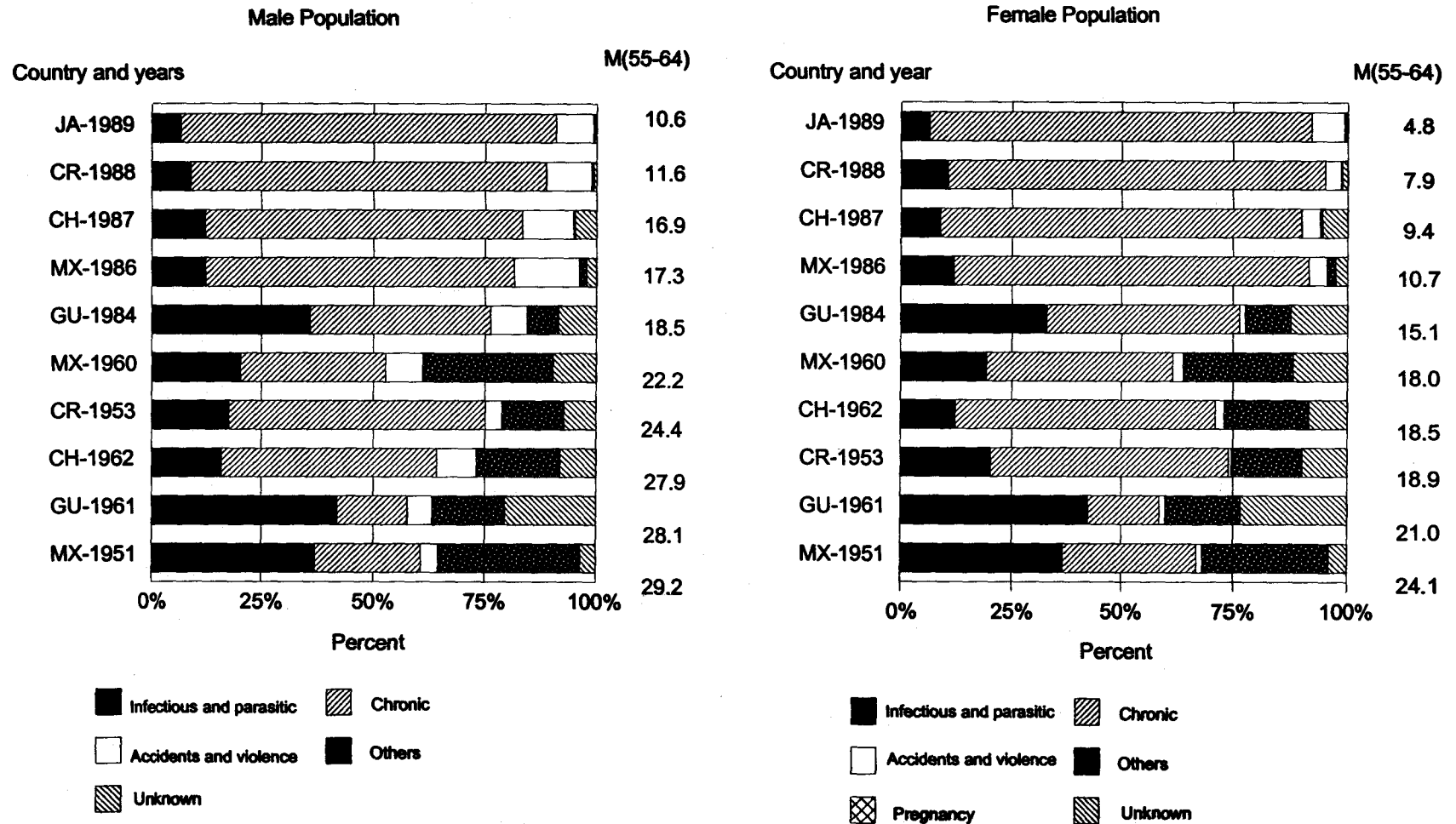
Figure 19. Percent distribution of deaths, ages 45-54, by cause of death



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).

Note: M(45-54) represents the mortality rate per thousand.

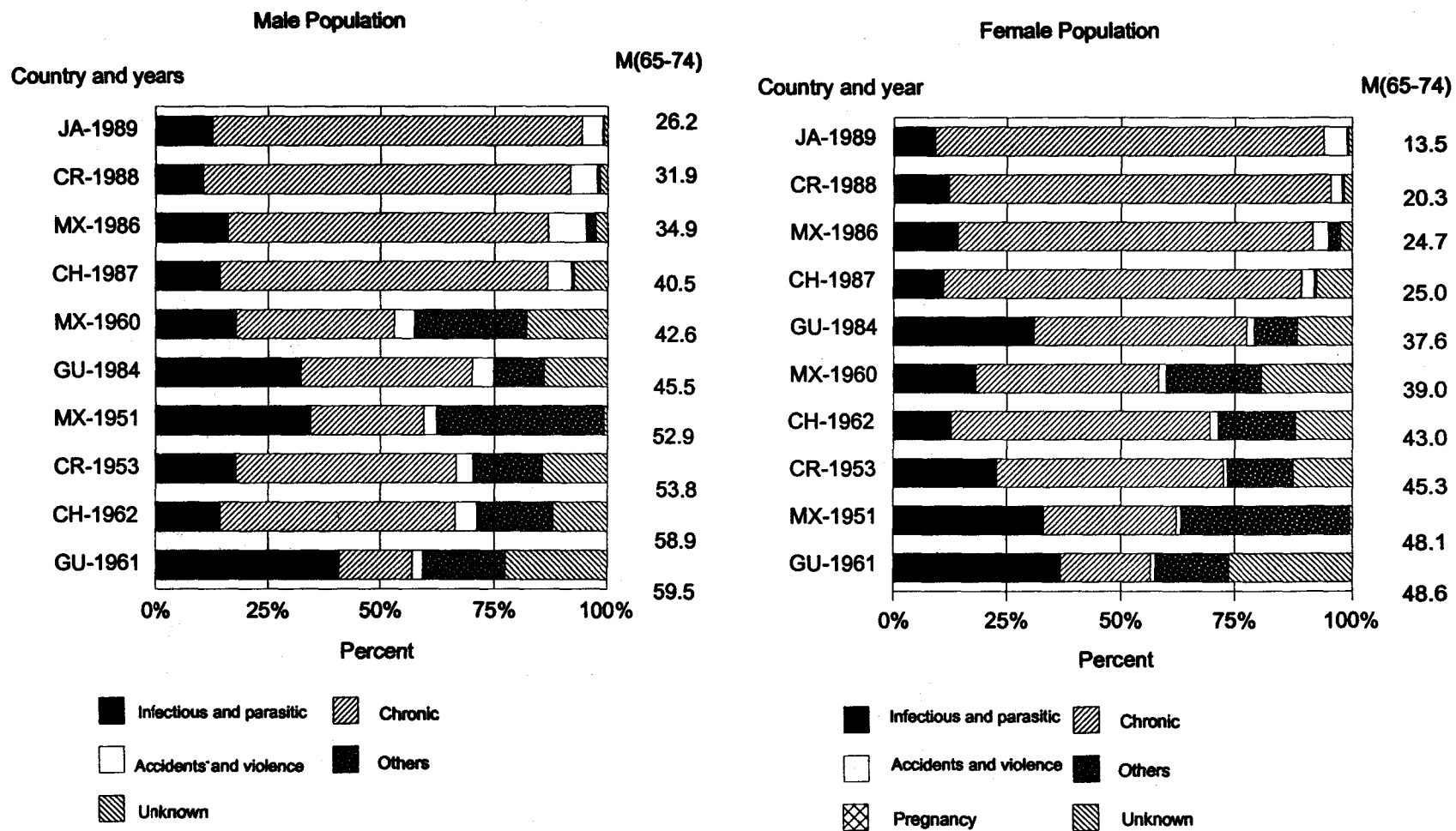
Figure 20. Percent distribution of deaths, ages 55-64, by cause of death



Source: World Health Organization, 1994 *World Statistics Annual* (Geneva, WHO, 1995).

Note: M(55-64) represents the mortality rate per thousand.

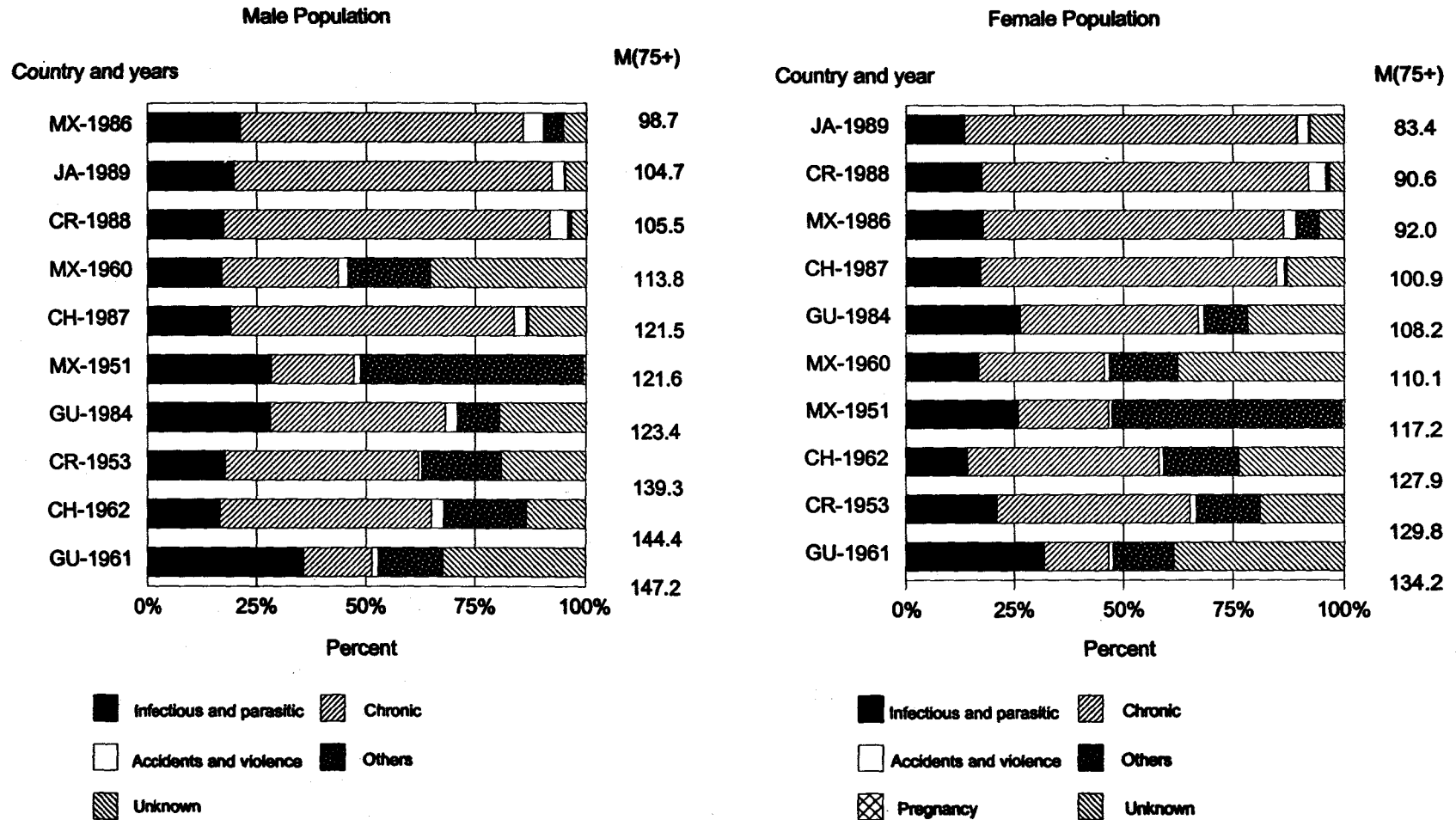
Figure 21. Percent distribution of deaths, ages 65-74, by cause of death



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).

Note: M(65-74) represents the mortality rate per thousand.

Figure 22. Percent distribution of deaths, ages 75 and over, by cause of death



Source: World Health Organization, 1994 World Statistics Annual (Geneva, WHO, 1995).

Note: M(75+) represents the mortality rate per thousand.

## REFERENCES

- Arriaga, Eduardo (1995). La mortalidad en la Capital Federal y en tres provincias nortenas. To be published in the Proceedings of the V Population Conference of the Argentinian Population Studies Association
- \_\_\_\_\_ (1997). The 2000 round of censuses and large surveys: data collection proposals. In *Proceedings of the Expert Group Meeting on Innovative Techniques for Population Censuses and Large-Scale Demographic Surveys*, Netherlands Interdisciplinary Demographic Institute and United Nations Population Fund (The Hague), pp. 269-274.
- Bennett, N. G., and S. Horiuchi (1981). Estimating the completeness of death registration in a closed population. *Population Index* (Princeton), vol. 47, No. 2, pp. 207-222.
- Brass, William (1975). *Methods for Estimating Fertility and Mortality from Limited and Defective Data*. Chapel Hill: University of North Carolina, Laboratories for Population Statistics.
- \_\_\_\_\_, and others (1968). *The Demography of Tropical Africa*. Princeton: Princeton University Press.
- Broek, J. Van den, R. Eeckels and J. Vuylsteke (1993). Influence of nutritional status on child mortality in rural Zaire. *Lancet*, vol. 341, No. 8859, pp. 1491-1491.
- Campbell, O., and C. Ronsmans (1995). Verbal autopsies for maternal deaths. World Health Organization Workshop, January 10-13, London. W.H.O./FHE.MSM/95.15.
- Chandramohan, D., G. H. Maude, L. C. Rodriguez and R. J. Hayes (1994) Verbal autopsies for adult deaths: issues in their development and validation. *International Journal of Epidemiology*, vol. 23, No. 2 (April), pp. 213-322.
- Dowel, S. F., H. L. Davis, E. A. Holt, A. J. Ruff, P. J. Kissinger, J. Bijoux, R. Boulos, C. Boulos and N. A. Halsey, (1993). The utility of verbal autopsies for identifying HIV-1-related deaths in Haitian children. *AIDS*, vol. 7, No. 9 (September), pp. 1255-1259.
- Fauveau, V., F. J. Henry, A. Briend, M. Yunus and J. Chakraborty (1992). Persistent diarrhoea as a cause of childhood mortality in rural Bangladesh. *Acta Paediatrica*, vol. 81, Supp. 381 (September), pp. 12-14.
- Fauveau, V. A. (1995). The Lao People's Democratic Republic: maternal mortality and female mortality: determining causes of death. *World Health Statistics Quarterly*, vol. 48, No. 1, pp. 44-6
- Feeney, Griffith (1980). Estimating infant mortality trends from child survivorship data. *Population Studies* (London), vol. 34, No. 1, pp. 109-129.
- Fikree, F. F., R. H. Gray, H. W. Berendes and M. S. Karim (1994). A community-based nested case-control study of maternal mortality. *International Journal of Gynaecology and Obstetrics*, vol. 43, No. 3 (December), pp. 247-255.
- Graham, W., W. Brass and R. V. Snow (1989). Indirect estimation of maternal mortality: the sisterhood method. *Studies in Family Planning*, vol. 20, No. 3, pp. 125-135.
- Gray, R. H. (1991). Verbal autopsy: using interviews to determine causes of death in children. Institute of International Programs, School of Hygiene and Public Health, John Hopkins University, Occasional Paper No. 14.
- \_\_\_\_\_, and J. Trussell (1997). Further developments in indirect mortality estimation. *Population Studies* (London), vol. 31, No. 1, pp. 313-334.
- Islam, M. A., M. M. Rahman, D. Mahalanabis and A. K. Rahman (1996). Death in a diarrhoeal cohort of infant in young children soon after discharge from hospital: risk factors and causes by verbal autopsy. *Journal of Tropical Pediatrics*, vol. 42, No. 6 (December), pp. 342-347.
- Johnson, P. (1982). Estimating infant mortality using information on the survival status of births in the year prior to a census or survey. In *Techniques for Estimating Infant Mortality*, U. S. Bureau of the Census, International Research Document No. 8, Washington D.C.
- Kane, T. T., A. A. El-Kady, S. Saleh, M. Hage, J. Stanback and L. Potter (1992). Maternal mortality in Giza, Egypt: magnitude, causes, and prevention. *Studies in Family Planning*, vol. 23, No. 1 (January-February), pp. 45-57.
- Khan, S. R., F. Jalil, S. Zaman, B. S. Lindblad and J. Karlberg (1993). Early child health in Lahore, Pakistan: X. Mortality. *Acta Paediatrica*. Vol. 390, Supplement (August), pp. 109-117.
- Kingkade, W. W., and E. E. Arriaga (1997). Mortality in the New Independent States: patterns and impacts. In *Premature Deaths in the New Independent States*, Washington, D.C. National Academy Press, pp. 156-183.
- Macro International (1994). *Methodological Reports 2. An Assessment of the Quality of Health Data in DHS-I Surveys*. Calverton, Maryland: Demographic and Health Surveys.
- Marshall, D., N. Majid, Z. Rasmussen, K. Mateen and A. A. Khan (1993). Cause-specific child mortality in a mountainous community in Pakistan by verbal autopsy. *Journal of the Pakistan Medical Association*, vol. 43, No. 11 (November), pp. 226-229.
- Martinez, H., H. Reyes, P. Tome, H. Guiscafre and G. Gutierrez (1993). La autopsia Verbal: Una herramienta para el estudio de la mortalidad en niños. *Boletín Médico del Hospital Infantil de México*, vol. 50, No. 1 (January), pp. 57-63.
- McKeown, T. (1976). *The Modern Rise of Population*. New York: Academic Press.
- Mobley, C. C., J. T. Boerma, S. Titus, B. Lohrke, K. Shangula and R. E. Black (1996). Validation study of Verbal autopsy method for causes of childhood mortality in Namibia. In *Child Survival in Developing Countries. Can Demographic and Health Surveys Help to Understand the Determinants?* Jan Ties Boerma ed. Amsterdam, the Netherlands: Royal Tropical Institute, pp. 99-110.
- Moriyama, I. M., R. M. Roy and A. H. T. Robb-Smith (1994). *History of Nomenclature of Diseases, the International Classification of Diseases, and the Classification of Causes of Death*. International Institute for Vital Registration and Statistics, Washington D.C.
- Palloni, Alberto (1981). Mortality patterns in Latin America. *Population and Development Review* (New York), vol. 7, No. 4, pp. 623-650.
- \_\_\_\_\_, and L. Heligman (1985). Re-estimation of structural parameters to obtain estimates of mortality in developing countries. *Population Bulletin of the United Nations* (New York), No. 18, pp. 1-33.
- Preston, Samuel (1983). An Integrated System for Demographic Estimation from Two Age Distributions. *Demography* (Washington, D. C.), vol. 20, No. 2, pp. 213-226.
- \_\_\_\_\_, and N. Bennett (1983). A census-based method for estimating adult mortality. *Population Studies* (London), vol. 37, No. 1, pp. 91-104.
- Sadruddin, S. (1994). Infant mortality rates and clinical causes of death in rural Balochistan and the Hazara division of NWFP. In *Maternal and Infant Mortality Policy and Interventions: Report of an International Workshop at the Aga Khan University*, February 7-9, Aga Khan University, Karachi.
- Snow, R. W., J. R. Armstrong, D. Forest, M. T. Winstanley, V. M. Marsh, C. R. Newton, C. Wuruiru, I. Mwangi, P. A. Winstanley and K. Marsh (1992). Childhood deaths in Africa: uses and limitations of verbal autopsies. *Lancet*, vol. 340, No. 8815 (8 August), pp. 351-355.
- Snow, R. W. (1995). An assessment of the impact of malaria interventions: methodological issues and the use of verbal autopsies. In *Evaluation of the Impact of Health Interventions*

- tions, Hoda Rashad, Ronald Gray, and Ties Boerma, Eds. International Union for the Scientific Study of Population, Liege, Belgium, pp. 297-322.
- Sullivan, James (1972). Models for the estimation of the probability of dying between birth and exact ages of early childhood. *Population Studies* (London), vol. 26, No. 1, pp. 77-97.
- United Nations (1983). *Indirect Techniques for Demographic Estimation*. Manual X. New York: The United Nations. Sales No. E.83.XIII.2.
- \_\_\_\_\_. (1996). *World Population Prospects: the 1996 Revision*. Annex II and III Demographic Indicators by Major Area, Region and Country. New York: United Nations. Sales No. E.97.XIII.5.
- \_\_\_\_\_. (1997). *Population and Vital Statistics Report Series A Vol. XLIX*. No.3, New York.
- U. S. Bureau of the Census (1996). *World Population Profile: 1996 Report*, by T. M. McDevitt, U.S. Government Printing Office, Washington D.C.
- Vallin, Jacques (1979). Socio-economic determinants of mortality in industrialised countries. In *Proceedings of the Meeting on Socio-economic Determinants and Consequences of Mortality, Mexico City, June 19-25, 1979*, New York, United Nations, pp. 266-302.
- World Health Organization (1993). *1993 World Health Statistics Annual*. Geneva

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## V. MORTALITY IN SUB-SAHARAN AFRICA

*Ian M. Timæus\**

### A. INTRODUCTION

Mortality in sub-Saharan Africa is higher than in the other major regions of the world. Moreover, among infants and children at least, the rate of decline in mortality has been slower in sub-Saharan Africa than elsewhere (Hill and Pebley, 1989; United Nations, 1988). The level of mortality varies markedly across Africa, both between countries and between regions and social groups within particular countries. Until the 1960s, most of the highest mortality countries lay in the far west of the continent and a gradient existed in the level of mortality across Africa, with the lowest mortality countries being found to the south and east (Hill, 1991a; Timæus, 1991a). During the 1970s and early 1980s, however, this pattern became less clear: mortality fell rapidly in some Western African countries but stagnated at a rather high level in parts of Eastern and Southern Africa (Hill, 1993; Timæus, 1993).

Several developments suggest that mortality decline in Africa may have slowed further or been reversed in recent years. The first reason for pessimism is the economic difficulties that have afflicted most African countries since the 1970s. Output per head in the region as a whole fell between the end of the 1970s and mid-1990s and only a handful of countries, notably Botswana, experienced significant growth. Economic difficulties have been shown to have had a short-run adverse impact on infant and child mortality in several African countries, notably Ghana and Nigeria (Working Group on Demographic Effects of Economic and Social Reversals, 1993). Moreover, economic decline and adjustment programmes designed to reduce inflation and

budgetary deficits have had a longer-term adverse impact on social development programmes (Barbieri and Vallin, 1996). In particular, while the proportions of girls attending both primary and secondary school have continued to rise in most African countries, they grew more slowly in the 1980s than the 1970s. Thus, growth in the proportion of mothers who have attended school has now begun to slacken in much of Africa, suggesting that the pace of decline in childhood mortality may also slow. The health sector has been affected even more severely. Government expenditure on health services has stagnated since the beginning of the 1980s in many African countries, leading to declines in the resources per head of the population devoted to health.

Another reason for pessimism about mortality trends in parts of Africa is civil warfare and other conflicts. A long history of military conflict has left countries and areas such as Eritrea, Ethiopia, Mozambique, Somalia and southern Sudan among the poorest parts of Africa. The murder of approaching one million Rwandans in 1994 was of major demographic significance. Other recent conflicts, such as those in Liberia and Sierra Leone, have involved fewer violent deaths. Warfare in Africa, however, as elsewhere, often brings in its wake substantial mortality from famine and disease.

Despite these problems, some reasons exist for a degree of optimism about the evolution of mortality in Africa. In at least some countries, economic reform has met with a degree of success. Ghana was the first African country to experience a major economic crisis. Equally, standards of living began to rise again in Ghana as early as the mid-1980s. During the last couple of years, average output per head has probably grown in sub-Saharan Africa as a whole. Most notably, Uganda's economy has grown rapidly since

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civil order was re-established in most of the country at the end of the 1980s. Favourable political developments have also occurred, notably in Southern Africa. The establishment of a democratic political system in South Africa has helped to stabilise this area of Africa as a whole. Conflict has ended in Mozambique and, to a large extent, Angola. Other wars, such as that in Ethiopia, have also ended. In addition, despite financial constraints, some important advances in health care have been achieved during the past 20 years. The most notable of these is the rapid spread of immunisation against the common childhood diseases. While the record of certain countries (including some of the most populous) is lamentable, in much of Africa the majority of children now receive all the EPI vaccines.

The influence on mortality of recent changes in infectious disease ecology in sub-Saharan Africa is likely to be wholly negative. One worrying development is the spread of chloroquine-resistant malaria across the continent between the early 1980s and early 1990s. Without doubt, malaria is a major killer in Africa. Treatment and self-treatment of fevers with chloroquine have been very common and are thought to have had a substantial impact on child mortality. By implication, chloroquine-resistant malaria must now be exerting a significant upward influence on mortality (Bradley, 1991; Ewbank and Gribble, 1993; Nájera and others, 1993). However, too little is known about the scale of mortality from malaria in Africa, about how many deaths are averted by treatment of fevers with antimalarials and about the epidemiology of drug-resistant malaria to attempt to quantify this impact.

A development in relation to which it is even harder to find any grounds for optimism is the epidemic spread of HIV in Africa. Reconstructing the growth of the HIV epidemic in much of Africa is extremely difficult. Nearly all the available data on the prevalence of HIV infection are based on the testing of women attending antenatal clinics. Such women are unlikely to be representative of all women and provide no information on the severity of the epidemic among men. In addition,

most of these seroprevalence data refer to urban populations. To obtain national estimates usually involves the assumptions that data from a few rural surveillance sites are representative of other rural areas and that the ratio of rural-to-urban seroprevalence at one date can be extrapolated to other years (Stanecki and Way, 1997).

Such calculations suggest that more than 10 per cent of the adult population were infected with HIV by the early 1990s across a belt of Africa extending from Botswana to Kenya (Bongaarts, 1996; Stanecki and Way, 1997). Côte d'Ivoire and the Central African Republic are also thought to be affected severely but the infection remains relatively rare in much of Western Africa. By contrast, the prevalence of HIV infection has risen very rapidly in Zimbabwe and much of Southern Africa since the end of the 1980s.

AIDS in Africa is primarily a heterosexually transmitted disease. Its impact extends to both men and women but is concentrated among adults in the age range that typically forms new sexual partnerships. Death rates in early adulthood and early middle age are expected to rise several fold. While appreciable increases in mortality in the first few years of life also occur due to vertical transmission (Nicoll and others, 1994), the mortality of older children and older adults will rise by less. Thus, the HIV epidemic is likely to have a dramatic effect on the age pattern of mortality in Africa as well as on its overall level.

## B. AVAILABILITY OF DATA

No mainland country in sub-Saharan Africa has an adequate vital registration system. South Africa is the only country where sufficient deaths are registered routinely to attempt to produce national estimates of mortality from this source. Even in this country, coverage is far from complete. Thus, apart from research studies of localised populations, the main sources of information on mortality in Africa are national censuses and household surveys. Table 6 provides an inventory of the data on mortality collected in censuses and Demographic and Health Survey (DHS) in-

TABLE 6. MORTALITY DATA COLLECTED IN SUB-SAHARAN AFRICAN POPULATION CENSUSES, 1985-1994 AND DHS SURVEYS

Country	Date	Census data				DHS data <sup>a</sup>	
		CEB/CS	Mother alive?	Father alive?	Recent deaths	Survey date(s)	Data on adults <sup>b</sup>
<b>Eastern Africa</b>							
Burundi	1990	✓ <sup>c</sup>	✓	✓	(✓)	1987	O <sup>d</sup>
Djibouti	no census						
Eritrea	no census					1995	(S)
Ethiopia	1994	✓ <sup>c</sup>					
Kenya	1989	✓	✓	✓		1988, 1993, 1998	-, -, (S)
Madagascar	1993	(✓)	(✓)	(✓)	(✓)	1992, 1997	-, -
Malawi	1987	✓			(✓)	1992	S
Mozambique	no census					1996-1997	(S)
Rwanda	1991	(✓)	(✓)	(✓)		1992	-
Somalia	1987	(✓)					
Uganda	1991	✓	✓	✓		1988-1989, 1995	-, S
United Republic of Tanzania	1988	✓ <sup>c</sup>	✓		✓	1991-1992, 1996	-, S
Zambia	1990	✓			(✓)	1992, 1996-1997	-, S
Zimbabwe	1992	✓	(✓)	(✓)	✓	1988-1989, 1994	-, S
<b>Middle Africa</b>							
Angola	no census						
Cameroon	1987	✓ <sup>c</sup>	✓	✓	✓	1991, 1997	-, (S)
Central African Republic	1988	✓ <sup>c</sup>			✓	1994-1995	S
Chad	1993	(✓)			(✓)	1997	(S)
Congo <sup>e</sup>	1994					1997	-
Congo, D. R.	no census						
Equatorial Guinea <sup>e</sup>	1994						
Gabon <sup>e</sup>	1993						
<b>Northern Africa</b>							
Sudan (Northern)	1993	✓ <sup>c</sup>	✓		✓	1989-1990	S <sup>c</sup>
<b>Southern Africa</b>							
Botswana	1991	✓			✓	1988	-
Lesotho	1986	✓	✓	✓	✓		
Namibia	1991	✓ <sup>c</sup>				1992	S
South Africa <sup>e</sup>	1985, 1991					1997-1998	(S)
Swaziland	1986	✓ <sup>c</sup>	✓	✓			
<b>Western Africa</b>							
Bénin	1992				(✓)	1996	S
Burkina Faso	1985	✓			(✓)	1993, 1998	-, -
Côte d'Ivoire	1988	✓				1994, 1997	-, -
The Gambia	1993	(✓)	(✓)	(✓)			
Ghana	no census					1988, 1993	O, -
Guinea	no census					1992	-
Guinea-Bissau	1991	(✓)					
Liberia	no census					1986	-
Mali	1987	✓	✓	✓		1987, 1995-1996	-, S
Mauritania	1988	✓ <sup>c</sup>					
Niger	1988	✓ <sup>c</sup>	✓	✓		1992, 1998	S, -
Nigeria	1991	(✓)			(✓)	1990	
Senegal	1988	✓ <sup>c</sup>	✓	✓	✓	1986, 1992-1993	O, S
Sierra Leone	1985	✓	✓				
Togo	1993	(✓)	(✓)	(✓)	(✓)	1988	

Source: See note 1.

NOTES: O Data collected from ever-married adult women about orphanhood before and since first marriage.

S Sibling histories collected.

( ) Data not known to have been published by 1997 (see note 1).

<sup>a</sup> All these DHS surveys collected birth history data that can be used to estimate infant and child mortality.<sup>b</sup> Except in Guinea, the household schedule for all DHS surveys in sub-Saharan Africa since 1991 has asked about the orphanhood of children aged less than 15.<sup>c</sup> Information not obtained for this study.<sup>d</sup> More detailed questions about adult mortality were also asked in this survey (Makinson, 1993).<sup>e</sup> Census schedule not seen.

quiries since 1985.<sup>1</sup> In addition, a few African countries such as South Africa have collected data on mortality in nationally representative sample surveys conducted outside the DHS programme.

Most of the information available on child mortality comes from one of two sources (Hill, 1991b). The first is questions put to adult women about how many children they have ever borne and how many of these children remain alive. These questions have been asked in numerous censuses and other inquiries in Africa. Methods were developed over 30 years ago for converting such data into life table indices of mortality (United Nations, 1983). Data on different age groups can provide a time series of estimates of overall child mortality. The approach cannot be used, however, to partition precisely the under-five mortality rate into infant mortality and mortality between ages one and four.

The second main source of information on child mortality is the detailed birth histories collected retrospectively from women in fertility surveys. In a fertility survey, women are asked about the date of birth of each of their children and the ages at death of those of their children who have died. From these data, one can calculate age and period-specific mortality directly (Somoza, 1980) and can investigate mortality differentials according to a wide range of characteristics of the mother and household. Between 1977 and 1982, such surveys were conducted in ten African countries as part of the World Fertility Survey (WFS). Since 1986, 32 more national fertility surveys have been completed in sub-Saharan Africa under the auspices of the DHS programme and 12 more are underway.

Table 6 reveals that almost every African country has collected data on child mortality since 1985 in either the population census or a DHS survey. Not all of these data are yet published. Nevertheless, the only major African country in which no prospect exists currently of obtaining estimates of mortality under age five is the Democratic Republic of Congo (ex-Zaire), together perhaps with some of the Middle African countries where it is

unclear what information was collected in the 1990-round census.

Unfortunately, both forms of retrospective data on child mortality are vulnerable to bias resulting from reporting errors. Failure to mention dead children can be a problem and direct measures based on birth histories only represent an improvement over the indirect approach if reasonably accurate data can be collected on ages and dates. In countries affected by AIDS, a major selection bias also afflicts data on child survival. Infected women transmit the HIV virus to 25 to 35 per cent of their children. Both these children and their mothers suffer extremely high mortality. Thus, no report is made on many of the children with the highest mortality.

In a major review of levels and trends in child mortality, Hill (1991a; 1992) presents national estimates of under-five mortality in sub-Saharan Africa for the 1920s to 1970s based on census and WFS and other survey data. In a later paper (Hill, 1993), she presents updated estimates for 16 African countries that had published the results of their 1980 round census or Phase 1 DHS survey by 1992. This extends the temporal scope of her analysis up to the mid-1980s.

During the last five years, the results have been published of some 19 African censuses conducted during the period 1986 to 1992, together with the results of Phase 2 and 3 DHS surveys for 14 new countries, and for six of the countries that also participated in Phase 1 of the programme (see table 6). These data can be used to update Hill's (1991a; 1993) earlier work and track trends in child mortality in a number of countries into the early-1990s.

Collecting data on adult mortality retrospectively in censuses and surveys has proved more difficult than collecting data on child mortality (Timæus, 1991d). Several questions have proved useful but none of them is reliable. Moreover, retrospective data are of limited value for the study of socio-economic differentials in adult mortality as information is seldom collected on the characteristics of dead individuals.

Questions about recent deaths in the household have been asked in many African censuses and some large surveys. Most fertility surveys, however, visit too few households to attempt to measure mortality by this approach. On occasion, these questions have worked well. In other inquiries, only a small fraction of the expected number of deaths has been reported. A range of methods has been developed for evaluating the completeness of data on recent adult deaths (Preston, 1984). In favourable circumstances these methods allow one to rehabilitate incomplete data. In Africa, however, they have often yielded inconclusive findings.

The second main source of information about adult mortality in sub-Saharan Africa is questions in surveys and censuses about orphanhood. As with data on children ever-born and surviving, methods exist for estimating time series of life table indices from information by age on the proportions of respondents with living mothers and fathers (Timæus, 1992). Unfortunately, individuals who are orphaned at a young age are often not reported as such in parts of Africa. A large downward bias can result in mortality estimates made from orphanhood data for children. Mortality estimates based on data supplied by adults are affected less. They usually reflect conditions a decade or more before their collection. However, more up-to-date estimates of mortality can be obtained if data are available on whether parents died before or after the respondent first married (Timæus, 1991b). This information was collected in several Phase 1 DHS surveys.

Like reports by mothers on the deaths of their children, reports by children on their parents' survival, particularly that of their mothers, are subject to bias as a result of vertical transmission of HIV. Only a minority of children of infected mothers become infected themselves but, because deaths of their mothers account for a substantial proportion of all orphanhood, the bias in the mortality estimates is again large. Nevertheless, if one can estimate the proportion of the mothers who were infected at the time that their children were born, one can adjust data on orphanhood

to correct for this bias (Timæus and Nunn, 1997).

A third source of information on the adult mortality in Africa has become available recently. It is the sibling histories collected as part of the maternal mortality module of some Phase 2 and Phase 3 DHS surveys (Rutenberg and Sullivan, 1991). These surveys asked women about the date of birth of each of their brothers and sisters and the ages at death of any of their siblings who have died. Thus, like birth histories, sibling histories provide the information needed to calculate age and period-specific mortality directly. Most respondents have several siblings and one can analyse data on a reference period of several years. Thus, these histories can provide useful estimates of adult mortality in surveys with samples of the size interviewed in DHS inquiries.

Little experience in the analysis of sibling history data has been accumulated. It remains unclear how complete and accurate are the reporting of deaths. An initial investigation of these issues suggests that, while respondents can report the ages and ages at death of their siblings, reporting of when siblings died is both less complete and subject to rounding errors (Stanton and others, 1997). Throughout the world, the sibling histories tend to yield lower adult mortality estimates for the period 7 to 13 years ago than for 0 to 6 years before the data were collected. This is implausible and suggests that such data are of little value for study of medium-term trends in mortality. Moreover, on the basis of comparison of the more recent estimates with other estimates 'of good quality', the report suggests that even the recent sibling history estimates may underestimate adult mortality, especially for women. For Africa, however, the only comparison is with a set of estimates of mortality that may be too high: they were made using the 1988 Census data for Senegal (Pison and others, 1995) and these can be interpreted in several ways.

In contrast to the situation with regard to data on child mortality, in at least a dozen African countries no data at all have been col-

lected that can be used to estimate adult mortality at any point in the 1980s or 1990s (see table 6). This group includes the most populous countries in the region: Nigeria, Ethiopia, and the Democratic Republic of Congo. In much of the rest of the continent, moreover, the data on adult mortality are more limited and less reliable than those on children. Timæus (1991a) presents estimates of life expectancy at age 15 by sex in 20 sub-Saharan African countries for some point in the period 1965 to 1981. This analysis was subsequently updated to take into account the census and DHS survey results published by 1992 (Timæus, 1993). The later paper presents estimates of the probability of surviving from age 15 to 60 at some point between 1970 and 1986 for 24 countries. It also examines trends in adult women's mortality in a subset of 15 countries.

Nearly all the African censuses published since Timæus (1993) produced his review paper have collected information on either recent deaths or orphanhood (see table 6). However, the most up-to-date of these data were collected in 1993. The DHS sibling history data for 11 sub-Saharan African countries were analysed in time for them to be presented here (Timæus, 1998 in press). Some of these data were collected back in 1992 but some as recently as the end of 1996. In addition, adult mortality can be estimated from information on orphanhood of children aged less than 15 years collected on the household schedule used in most DHS surveys conducted in Africa during the 1990s (Timæus, 1998 in press).

The underdevelopment of vital statistics and low coverage of the health services in sub-Saharan Africa combine to obstruct the investigation of causes of death. Incomplete data are available for South Africa. In addition, something has been learnt about the diseases that kill infants and children from prospective population-based research studies conducting some form of verbal autopsy (Feachem and Jamison, 1991; Garenne and Fontaine, 1990). Unfortunately, both the accuracy and detail of verbal autopsy data remain limited. Too little such information exists to develop even a crude map of how patterns of cause of death vary across Africa. In addition, only very few

prospective studies have been conducted on a large enough scale to yield useful information on causes of death in adulthood (Chandramohan and others, 1994; Kitange and others, 1996).

As part of their study of the global burden of disease, Murray and Lopez (1996; 1997) synthesised the fragmentary data available on causes of death in sub-Saharan Africa with models based on other high-mortality populations to produce a comprehensive series of cause-of-death estimates for the region. As in any such exercise in systematic guesswork, some of their results will be misleading. Nevertheless, the estimation process was designed to ensure that the results remain epidemiologically plausible and consistent with what is known of the demography of Africa. The main findings refer to 1990, when AIDS deaths were far less common than today. In other ways, however, they are the best indication available of the main public health problems in sub-Saharan Africa. No attempt is made to improve on those estimates here.

### C. INFANT AND CHILD MORTALITY

Table 7 compiles the direct estimates of the under-five mortality rate, that is, the probability of dying before age five, obtained in 32 DHS surveys conducted in sub-Saharan Africa between the mid-1980s and mid-1990s. Estimates are presented for the three quinquennia preceding each survey. Figure 23 presents these data graphically and compares them with earlier estimates produced by Hill (1993). Figure 23 also includes indirect estimates of under-five mortality for several countries based on children ever-born and surviving data collected in the 1990-round census (see table 6). No attempt has been made to reconcile the various sets of measures and produce best estimates of child mortality.

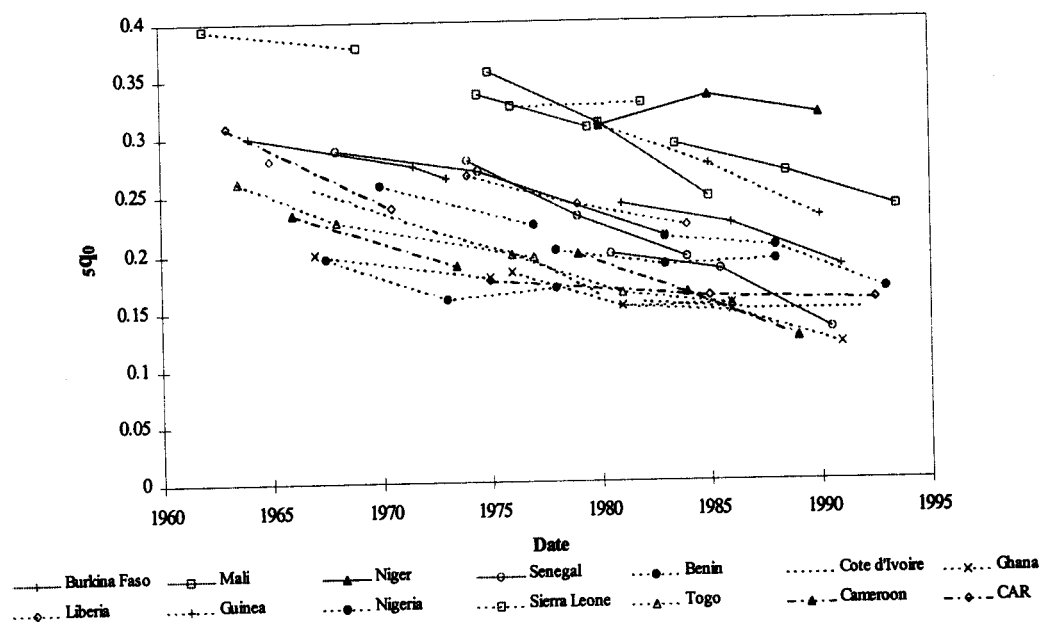
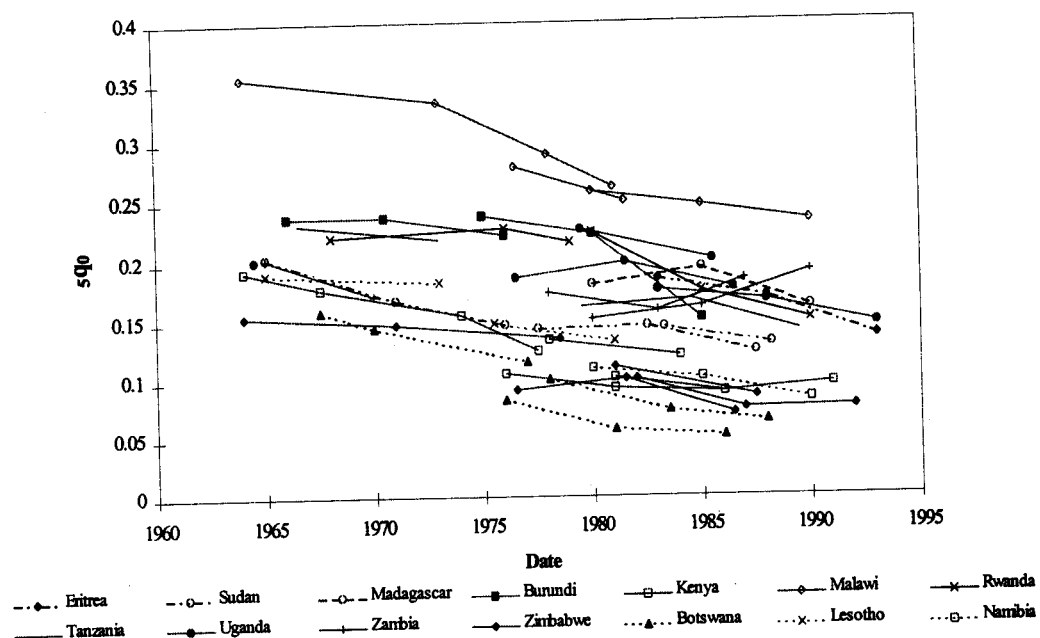
In general, the data collected by DHS surveys indicate levels and trends in mortality that are broadly consistent with earlier estimates. In addition, in most of the countries that have conducted two DHS surveys the estimates from them agree quite well. However, the majority of the triplets of estimates

TABLE 7. UNDER-FIVE MORTALITY RATE ( $_5q_0$ ), DEMOGRAPHIC AND HEALTH SURVEYS, BIRTH HISTORIES,  
COUNTRIES OF SUB-SAHARAN AFRICA, VARIOUS DATES

Country	Date of fieldwork	Years before survey			Reduction in mortality (%)	
		10-14	5-9	0-4	10-14 to 5-9 years	5-9 to 0-4 years
Eastern Africa						
Burundi	1987	0.233	0.224	0.152	4	32
Eritrea	1995	0.185	0.170	0.136	8	20
Kenya	1988	0.106	0.093	0.090	12	3
	1993	0.102	0.090	0.096	12	-7
Madagascar	1992	0.181	0.195	0.162	-8	17
Malawi	1992	0.259	0.247	0.234	5	5
Rwanda	1992	0.225	0.176	0.151	22	14
Uganda	1988-1989	0.187	0.200	0.177	-3	16
	1995	0.176	0.167	0.147	7	11
U. R. of Tanzania	1991-1992	0.163	0.168	0.141	-7	12
	1996	0.166	0.154	0.137	5	12
Zambia	1992	0.152	0.162	0.191	-7	-18
	1996-1997	0.174	0.187	0.197	-7	-5
Zimbabwe	1988-1998	0.092	0.101	0.071	-10	30
	1994	0.101	0.075	0.077	26	-3
Middle Africa						
Cameroon	1991	0.198	0.165	0.125	17	24
Central African Republic	1994-1995	← 0.161 →		0.157		2
Northern Africa						
Sudan (Northern)	1989-1990	0.144	0.146	0.124	-1	15
Southern Africa						
Botswana	1988	0.083	0.058	0.053	30	9
Namibia	1992	0.110	0.102	0.084	7	18
Western Africa						
Bénin	1996	0.212	0.203	0.167	4	18
Burkina Faso	1993	0.242	0.224	0.187	7	17
Côte d'Ivoire	1994	0.157	0.150	0.150	4	0
Ghana	1988	0.183	0.153	0.155	16	-1
	1993	0.153	0.148	0.119	3	20
Guinea	1992	0.310	0.275	0.229	11	17
Liberia	1986	0.266	0.241	0.223	9	7
Mali	1987	0.356	0.310	0.247	13	20
	1995-1996	0.292	0.268	0.238	8	11
Niger	1992	0.308	0.334	0.318	-8	5
Nigeria	1990	0.201	0.189	0.193	6	-2
Senegal	1986	0.280	0.231	0.195	18	16
	1992-1993	0.198	0.185	0.132	7	29
Togo	1988	0.198	0.165	0.155	17	6

Sources: Bicego, G. and O. B. Ahmad, *Infant and Child Mortality*, DHS Comparative Studies, 20 (Calverton, Maryland, Macro International, 1996); Sullivan, J. M., S. O. Rutstein and G. T. Bicego, *Infant and Child Mortality*, DHS Comparative Studies, 15 (Calverton, Maryland, Macro International, 1994); Demographic and Health Surveys country reports.

Figure 23. Trends in under-five mortality, Eastern and Southern Africa and Middle and Western Africa



Sources: Table 7; Althea L. Hill, "Trends in childhood mortality", in *Demographic Change in Sub-Saharan Africa*, Karen A. Foote, Kenneth H. Hill and Linda G. Martin, eds. (Washington, D. C., Population Dynamics of Sub-Saharan Africa, National Academy Press, 1993); national censuses.

obtained from DHS surveys follow a convex curve, indicating an accelerating decline in mortality. In instances where the earliest of the three points can be checked against an estimate from an earlier inquiry for about the same time, it tends to be lower. Thus, it seems likely that in Africa many of the DHS estimates for 10-14 years before the data were collected are biased downward by omission of deaths.

In a few countries more serious inconsistencies exist between the estimates of under-five mortality from different sources. In most such countries, the DHS surveys indicate lower mortality than other data. In Botswana, for example, the three estimates based on the 1988 DHS survey are substantially lower than the slightly more up-to-date triplet of estimates based on the 1991 Census (see figure 23). One explanation could be that the DHS survey interviewed an unrepresentative sample of women with children who have low mortality (Blanc and Rutstein, 1994; Thomas and Muvandi, 1994a; Thomas and Muvandi, 1994b). Similar discrepancies exist in Uganda, where both the DHS surveys yield lower mortality estimates than the 1991 Census, and Kenya, where about 30 per cent of all but the most recent child deaths were not reported in the 1988 DHS survey (Brass and Jolly, 1993).

The under-five mortality rate reported in 1990 for these 26 sub-Saharan African countries ranges from nearly one third in Niger down to about 70 per 1,000 in Botswana and Zimbabwe. The level of mortality in most countries, however, lies in a somewhat more restricted range. Namibia and Kenya are probably the only mainland sub-Saharan African countries other than Botswana and Zimbabwe in which under 10 per cent of children die in the first five years of life. Extrapolating forward from the earlier census-based estimates, it seems likely that in 1990 the under-five mortality rate remained above 30 per cent in Sierra Leone as well as Niger (see figure 23). Though no nationally representative data exist, mortality may also have been this high in Angola, Mozambique, and southern Sudan. In contrast to Niger, however, mortality has fallen substantially since the 1960s in the western Sahel. Thus, in all sub-Saharan Afri-

can countries except those just mentioned, the under-five mortality rate in 1990 was probably somewhere in the range 125 to 250 per 1000.

Only limited evidence exists of any regional patterning in infant and child mortality at the end of the 1980s. Mortality in Southern Africa is relatively low. Many of the countries in the eastern half of Africa had an under-five mortality rate of between 140 and 190 per 1000. In Western and Middle Africa, however, the situation was more varied. Even after making considerable progress during the 1970s and 1980s, many Sahelian countries still had comparatively high mortality. In contrast, sustained and rapid mortality decline during the 1970s and 1980s left Senegal with low infant and child mortality for the region. By 1990, it ranked alongside Ghana and Cameroon with an under-five mortality rate of around 125 per 1,000.

The overall impression that one gains from figure 23 is that under-five mortality continued to decline in Africa during the 1980s. After stagnating in the early 1980s, under-five mortality in Ghana began to fall again in the second half of the decade. Moreover, mortality may have fallen particularly rapidly in some of the higher mortality countries. Nevertheless, quite widespread evidence exists of a slowdown in the rate of mortality decline in recent years. First, improvements in child survival seem to have tapered off at some point during the 1980s in a number of the lower mortality countries including Botswana, Central African Republic, Côte d'Ivoire, Kenya, Togo, and Zimbabwe. The rate of decline in mortality was also slow in several high mortality countries, namely Malawi, Liberia, and Niger. In Nigeria, no improvement occurred in under-five mortality between the late-1970s and the late-1980s. The most worrying development, however, is in Zambia, where both DHS and census data suggest that infant and child mortality rose substantially during the 1980s.

Can these adverse trends in the under-five mortality rate be accounted for by the HIV epidemic or are other factors also important? One can attempt to answer this question because estimation of the impact of the HIV epi-



demic on infant and child mortality is simpler than estimation of its impact on adult mortality. First, data on the prevalence of HIV infection collected at antenatal clinics measure directly the proportion of births at risk of infection through vertical transmission of the virus from their mothers. Second, most African infants who acquire HIV infection from their mothers will die in the first five years of their life. Thus, the impact on mortality of a rise in the prevalence of HIV infection is lagged by only a couple of years.

If about one-third of infected women giving birth transmit the HIV virus to their child, one would expect each 1 per cent rise in seroprevalence among women attending antenatal clinics to raise the under-five mortality rate by about 3 per 1,000. More sophisticated modelling of the impact of the HIV epidemic on infant and child mortality indicates that this is a robust approximation (Nicoll and others, 1994). Thus, in countries where the seroprevalence rate among pregnant women is 6.5 per cent, the under-five mortality rate could rise by about 20 per 1,000. If the seroprevalence rate rises to 20 per cent, an increase in under-five mortality of nearly 60 per 1,000 is likely to follow.

Such calculations suggest that the HIV epidemic may be responsible for the slowdown or reversal of the decline in infant and child mortality in some African countries. In Malawi, Rwanda, Uganda, Zambia and Zimbabwe, HIV may have become sufficiently prevalent by the mid-1980s to account for rises in the under-five mortality rate of 15 to 20 per 1,000 by the late 1980s. By the early 1990s, paediatric AIDS mortality could have been 25 to 30 per 1000 births. HIV infection could also have driven up the most recent mortality estimates available for Burkina Faso, Central African Republic, Côte d'Ivoire, and Kenya by 10 to 20 per 1,000. In all these countries, a proportion of these additional deaths will not be reflected in the birth history data because the children's mothers have also died of HIV-related disease.

Thus, the impact of HIV could account for the adverse trends in under-five mortality in Kenya and Zimbabwe during the early 1990s

revealed by the most recent DHS surveys. Moreover, the HIV epidemic might also account for the slow decline in early-age mortality in Malawi in the 1980s and the large rise in mortality in Zambia. In Zambia at least, however, it seems likely that the overall under-five mortality rate rose by so much only because other factors were not exerting a strong downward influence on infant and child mortality. It seems unlikely that infant and child mortality would have improved much in Zambia during the 1980s and early 1990s even without the HIV epidemic.

Elsewhere, it is unlikely that the HIV epidemic is responsible and the explanation for the adverse trend in the under-five mortality rate must be sought among other causes. For example, AIDS cannot explain the stagnation or rise in early-age mortality in Nigeria since the 1970s. Similarly, it is unlikely that the slowdown in the early 1980s of mortality decline in Botswana, Côte d'Ivoire and the Central African Republic is related to HIV. However, the epidemic might be implicated in the continuing lack of improvement in child survival in these countries during the early 1990s.

It is worth emphasising that infant and child mortality fell in Uganda in the early 1990s despite the severity of the HIV epidemic in this country. Presumably developments acting to improve child health have outweighed the impact on mortality of the spread of HIV. Thus, these estimates suggest that the HIV epidemic exerts only an important, not a decisive, influence on trends in infant and child mortality.

#### D. ADULT MORTALITY

Table 8 presents estimates of adult survivorship by sex for 24 sub-Saharan African countries. The estimates are the most up-to-date available and refer to a range of dates between 1981 and 1992. In the other 19 mainland sub-Saharan countries, data on adult mortality are non-existent, could not be obtained for this study, or reflect the death rates of the 1970s. A quarter of the estimates refers to the first half of the 1980s, half to the later 1980s, and a quarter to the period 1990 to 1993. The index

TABLE 8. SURVIVORSHIP FROM AGE 15 TO AGE 60 ( $_{45}P_{15}$ ) BY SEX,  
COUNTRIES IN SUB-SAHARAN AFRICA, 1980S AND 1990S

Country	Date	Women	Men	Both sexes	Source
<b>Eastern Africa</b>					
Burundi	1981	0.699	0.622	0.660	1987 DHS and 1990 Census: orphanhood
Kenya	1984	0.854	0.773	0.813	1979 & 1989 Censuses: intercensal orphanhood
Madagascar	1987	0.650	0.691	0.670	1992 DHS: orphanhood
Malawi	1989	0.649	0.662	0.656	1992 DHS: sibling histories
Uganda	1992	0.560	0.530	0.545	1995 DHS: sibling histories
U. R. of Tanzania	1988	0.675	0.656	0.665	1988 Census: recent deaths and orphanhood
Zambia	1993	0.546	0.430	0.488	1996 DHS: sibling histories
Zimbabwe	1992	0.675	0.581	0.628	1992 Census: recent deaths
<b>Middle Africa</b>					
Cameroon	1987	0.710	0.651	0.680	1987 Census: recent deaths and orphanhood
Central African Rep.	1991	0.550	0.477	0.513	1994-5 DHS: sibling histories
Congo	1984	0.703	0.656	0.680	1984 Census: recent deaths and orphanhood
<b>Northern Africa</b>					
Sudan (Northern)	1992	0.797	0.773	0.785	1993 Census: recent deaths and orphanhood
<b>Southern Africa</b>					
Botswana	1990	0.711	0.625	0.668	1991 Census: recent deaths
Lesotho	1985	0.741	0.576	0.658	1986 Census: recent deaths and orphanhood
Namibia	1989	0.813	0.670	0.742	1992 DHS: sibling histories
South Africa	1985	0.766	0.638	0.702	1985: adjusted vital registration
Swaziland	1981	0.773	0.569	0.670	1976 & 1986 Censuses: intercensal orphanhood
<b>Western Africa</b>					
Bénin	1993	0.796	0.747	0.771	1996 DHS: sibling histories
Ghana	1982	0.880	0.778	0.828	1988 DHS: orphanhood since marriage
Mali	1986	0.541	0.579	0.560	1986 Census: recent deaths and orphanhood
Mauritania	1980	0.823	0.782	0.802	1980 WFS: orphanhood and recent deaths
Niger	1988	0.766	0.782	0.774	1992 DHS: sibling histories
Senegal	1989	0.841	0.766	0.803	1992-3 DHS: sibling histories
Togo	1981	0.760	0.704	0.732	1981 Census: recent deaths

Sources: Ian M. Timæus, "Adult mortality", in *Demographic Change in Sub-Saharan Africa* (Washington, D.C., National Academy Press, 1993); original analyses of published and unpublished census and survey data.

of adult mortality used is the life table probability of surviving from age 15 to age 60. Recent deaths data usually underestimate old age mortality and neither the DHS sibling histories nor orphanhood data reflect the mortality of older children or of the elderly population. Moreover, because AIDS is expected to reshape age patterns of mortality in Africa, to use existing model life tables to extrapolate from mortality in the central adult ages to a wider age range is inappropriate. Thus, no basis exists in most of these coun-

tries for the calculation of such summary indices of mortality as life expectancy at age five.

Table 8 contains estimates based on a range of sources, including various forms of orphanhood data, census questions about recent deaths in the household, and the DHS sibling histories. The orphanhood estimates were calculated using methods proposed by Timæus (1991b; 1991c; 1992). The completeness of retrospective reports of recent deaths and of the civil registration statistics for

South Africa have been evaluated using the growth balance equation (Brass, 1975) and Preston-Coale (1980) method. Recent deaths data for women in Botswana and for Cameroon, Tanzania, and Zimbabwe are treated as complete. Those for the Central African Republic, Congo, Lesotho, Mali, South Africa, and Togo have been adjusted for underreporting, while those for men in Botswana and for Senegal and Sudan are adjusted for overreporting.

All the estimates calculated from sibling history data are based on deaths in the period 0 to 6 completed years before the survey. This seven-year reference period was chosen to reduce the impact of heaping of reported times of death on five completed years before the survey (Stanton and others, 1997). The mortality rates have been smoothed against a reference standard in seven countries and by fitting a fourth-order polynomial of age to the log death rates in Uganda, Tanzania, Zambia and Zimbabwe, where the age pattern of mortality differs markedly from that in existing model life tables. The models were fitted using Poisson regression and allow for the strong skew of the exposure of siblings toward younger ages.

The number of African countries in which one can measure adult mortality has not grown since the publication of Timæus' (1993) paper. However, the amount of information available has tended to increase for those countries where one can estimate adult mortality at all. Table 8 indicates the primary source of each of the pairs of estimates. In most countries, however, this information has been supplemented with data obtained in earlier inquiries or by using other questions. In Central African Republic, Senegal, and Zimbabwe, for example, broadly consistent estimates exist based on the DHS sibling histories and on recent deaths data collected in a recent census. Similarly, in Niger and Uganda the sibling histories have been checked against orphanhood data from a recent census. Only in Bénin, Malawi, Namibia and Zambia are the estimates based on sibling history data that cannot be checked against other up-to-date sources. The estimates for Botswana, Madagascar, and Togo are also based on single

questions that cannot be checked against other information. In addition, in Congo, Mali and Tanzania such crosschecks suggest that considerable uncertainty exists as to the actual level of mortality in the country. One can have somewhat more confidence in the estimates for the other 14 countries.

The results in table 8 reveal that adult mortality continues to vary markedly across sub-Saharan Africa. At the level of mortality prevailing in the Central African Republic and Zambia in the early 1990s, about half of those who live to their fifteenth birthday die before age 60. The mortality of adults in Uganda also approaches this level. So did adult mortality in Mali in the mid-1980s. Moreover, in over half the African countries where one can estimate adult mortality, the probability of dying between ages 15 and 60 exceeds 30 per cent. Few countries outside this region have such elevated adult mortality. On the other hand, by the 1980s the probability of dying between ages 15 and 60 had fallen below 20 per cent in several African countries. They include Ghana, Kenya and, by the end of the decade, Senegal. This represents a moderate level of mortality by world standards.

No clear regional patterning exists in these estimates of adult mortality. Assessment of this is complicated because the estimates for Western Africa are, on average, slightly more out of date than those for other parts of Africa. Nevertheless, it is clear that any tendency for Southern and Eastern African countries to have relatively low adult mortality had disappeared completely by the 1980s. In contrast, many of the countries with the lowest adult mortality are now to be found in Western Africa.

In most of Africa, adult men have higher mortality than adult women. The excess in men's mortality is particularly large in Southern Africa and the updated estimates in table 8 reveal that this pattern persisted into the second half of the 1980s. Nevertheless, comparison of the estimates for Botswana and Lesotho with those for a decade earlier (Timæus, 1993) suggests that men's mortality is improving slowly in these countries but that the survival of women is not. Thus, sex differen-

tials in mortality in Southern Africa probably narrowed somewhat between the 1970s and 1980s.

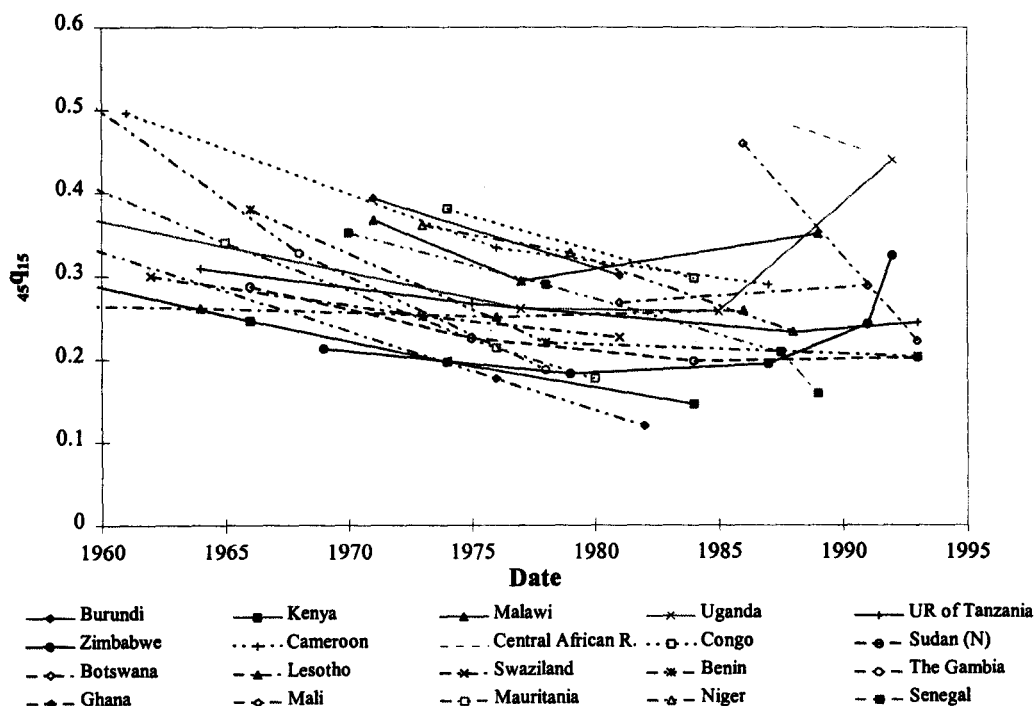
The DHS sibling histories suggest that adult women's mortality in Malawi is higher than that of adult men. A series of inquiries since the early 1970s, measuring adult mortality by a wide range of methods, have all found the same thing (Timæus, 1993). Thus, Malawi almost certainly is anomalous in this respect, although the reasons for this are unknown. The sibling history data also suggest that women have higher mortality than men in Niger. The same pattern is reported in the neighbouring country of Mali. While this is suggestive, these reversed differentials may reflect no more than errors in the data.

Figure 24 examines trends in women's survivorship from age 15 to 60 for the 18 countries in table 8 in which survivorship can be estimated at more than one point in time. The figure also presents estimates for the 1960s and 1970s for The Gambia and Mauritania. During the 1970s, adult women's mortality fell fairly rapidly in many African countries,

especially those in Western Africa. In contrast, progress was slow in Southern Africa and in some eastern African countries, such as Tanzania and Zimbabwe, but not others, such as Burundi and Kenya.

Adult women's mortality can be tracked into the late 1980s or the early 1990s in 13 countries. In Central African Republic, however, the estimates convey little more than that mortality remains high while, in Mali, the apparent drop in mortality is implausibly rapid, suggesting that at least one of the sets of estimates is biased severely. The other results suggest that the pace of mortality decline has remained rapid in Cameroon, Niger, and Senegal but has slowed in Bénin and northern Sudan. In Botswana, Lesotho, and Tanzania, progress has continued to be limited and, in Botswana and Tanzania at least, women's mortality may have risen over the decade. Mortality rose more markedly in the 1980s in Malawi, although the data are insufficiently detailed to indicate exactly when the previous trend was reversed. In Uganda and Zimbabwe, however, mortality began to rise sharply in the second half of the 1980s. The

Figure 24. Trends in women's mortality between ages 15 and 60



Source: Table 8.

adverse trend extends into the 1990s and, in Zimbabwe at least, appears to be accelerating.

Two obvious questions arise about these recent increases in adult mortality: are they genuine and can they be accounted for by the spread of the HIV epidemic? Because adults infected with HIV tend to survive longer than infected children, the delay between a rise in the prevalence of HIV infection and the subsequent rise in mortality is longer for adults than children. The lag may be four to five years. Table 8 includes estimates of adult mortality for four of the countries that are believed to have been affected most severely by the HIV epidemic in the 1980s: Malawi, Uganda, Zambia and Zimbabwe (Stanecki and Way, 1997). As the mortality estimates for Malawi are three or four years more out-of-date than those for the other countries, one would expect them to be affected less by AIDS deaths. In addition, some 2 to 3 per cent of women in the Central African Republic in the mid-1980s and Tanzania in the early 1980s may have been infected with HIV. Even prevalence rates this low could raise adult mortality by a significant amount. In the other countries in table 8, however, including Botswana, the prevalence of HIV infection probably rose too late to have had measurable effect on these estimates of adult mortality. Thus, the adult mortality estimates in table 8

and figure 24 are consistent with what is known about the spread of the HIV epidemic in Africa.

Data have been released so far for 11 African countries that conducted a DHS survey that collected sibling histories. HIV was prevalent in six of these countries by the time that these data were collected. The histories are analysed further, therefore, to see whether they can document the trend in adult mortality during the seven-year period prior to their collection (Timæus, 1998 in press). To do this, the Poisson regression models used to smooth the data were expanded to include a term measuring the log-linear trend in mortality over the seven years. Fitted estimates of survivorship from age 15 to 60 by sex for three points in time are shown in table 9. Because reports of when siblings died are affected by digital preference and have been imputed in some instances, considerable uncertainty surrounds the estimated trends in adult mortality indicated by table 9. However, the overall direction and magnitude of the changes are probably meaningful.

The results of this analysis are striking. A clear split exists between those countries where one would expect significant HIV mortality by the time that the data were collected and those where one would not. Ac-

TABLE 9. RECENT TRENDS IN THE PROBABILITY OF DYING BETWEEN AGE 15 AND 60 ( $_{45}Q_{15}$ ) BY SEX, DEMOGRAPHIC AND HEALTH SURVEYS, SIBLING HISTORIES, COUNTRIES IN SUB-SAHARAN AFRICA, VARIOUS DATES

Country	Date of fieldwork	Women's mortality			Men's mortality		
		6 years before survey	3 years before survey	Year before survey	6 years before survey	3 years before survey	Year before survey
Eastern Africa							
Malawi	1992	0.328	0.348	0.369	0.270	0.326	0.389
Tanzania	1996	0.229	0.243	0.257	0.232	0.296	0.373
Uganda	1995	0.282	0.404	0.556	0.365	0.451	0.547
Zambia	1996-1997	0.355	0.437	0.530	0.386	0.525	0.678
Zimbabwe	1994	0.146	0.220	0.324	0.176	0.306	0.498
Middle Africa							
Central African Republic	1994-1995	0.391	0.438	0.488	0.481	0.514	0.547
Southern Africa							
Namibia	1992	0.199	0.189	0.179	0.346	0.333	0.320
Western Africa							
Bénin	1996	0.215	0.205	0.196	0.249	0.253	0.257
Mali	1995-1996	0.270	0.230	0.194	0.301	0.310	0.319
Niger	1992	0.333	0.240	0.170	0.236	0.221	0.206
Senegal	1992-1993	0.207	0.163	0.127	0.273	0.239	0.208

Source: Ian M. Timæus, "Impact of the HIV epidemic on mortality in sub-Saharan Africa: evidence from national surveys and censuses", *AIDS Supplement*, vol. 12 (1998 in press).

*Italics indicate that the estimated trend in mortality is statistically insignificant.*

According to the sibling histories, huge increases occurred in adult mortality in Uganda, Zambia and Zimbabwe between the late 1980s and mid-1990s. In just six years, adult mortality in Uganda rose from a moderate level to the highest level documented in Africa since the 1960s. Zambia already had fairly high adult mortality in 1990. Nevertheless, during the next six years adult death rates doubled and, by 1996, men's probability of surviving from age 15 to 60 may have dropped to below one third. Zimbabwe had low adult mortality in the late-1980s. By 1994, men's death rates were about three and a half times higher and women's death rates two and a half times higher.

The sibling history data also suggest that mortality rose in the other countries in the high HIV-prevalence belt of Africa. Adult mortality in the Central African Republic and Malawi remained fairly high in the 1980s but this has not stopped it rising further. Over a six-year period, men's death rates rose by about 55 per cent in Malawi and women's death rates by about 35 per cent in the Central African Republic. Similarly, although reported adult mortality is surprisingly, and perhaps implausibly, low in Tanzania, men's death rates apparently rose about 80 per cent during the first half of the 1990s.

It is worth noting that, according to these sibling history data, large rises have occurred in the mortality of both men and women. In two of the higher mortality countries, the Central African Republic and Uganda, women's mortality rose faster than men's mortality during this seven-year period. However, in Zambia and in the lower mortality countries, Malawi, Tanzania and Zimbabwe, men's mortality rose faster than that of women. These data offer no support for the suggestion that more women than men in Africa have become infected with HIV (Berkley and others, 1990; Gregson and others, 1994).

The sibling history data for Eastern and Middle Africa therefore provide evidence in support of the pattern of mortality increase by country revealed in figure 24. They suggest further that very dramatic rises in adult mor-

talities have occurred in some countries during the 1990s that no other data are up-to-date enough to reveal. If respondents' reporting of dead siblings was less complete toward the beginning of the seven-year reference period, the apparent rise in adult mortality in countries in the high HIV prevalence belt could be partly or wholly spurious. The sibling history estimates for the Sahel, however, suggest that the decline in adult mortality in this region apparent in figure 24 continued into the early 1990s. They also confirm that adult mortality is stagnant in Bénin and suggest that the same is true of Namibia, which is what the experience of other Southern African countries would lead one to expect. Reporting errors have not occurred on a sufficient scale to suggest that mortality is rising rapidly in Western Africa. No obvious reason exists for suspecting the data on Eastern Africa to be of worse quality.

The other data on adult mortality collected in Phase 2 and 3 DHS inquiries in Africa are the questions on the household schedule about the orphanhood of children aged less than 15 years. Data on the survival of the mothers of children aged 5-9 and 10-14 years yield a pair of estimates of adult women's mortality, while data on the survival of fathers of children in these two age groups can be combined to produce a single estimate of men's mortality (Timæus, 1992).

These orphanhood data are potentially very valuable for examining the impact of AIDS on all-cause mortality because they largely reflect the mortality of women in their late twenties and thirties and of men in their late thirties and forties. These are probably the age groups in which AIDS deaths are most common. Thus, in populations where HIV is prevalent, mortality in this age range should be much higher, relative to mortality across the age range 15 to 60 years, than in existing model life tables. Unfortunately, as section B points out, orphanhood of children has often been underreported severely. If this is a problem in these surveys, one might expect the mortality estimates to be lower, relative to mortality between ages 15 and 60, than in existing model life tables.

In populations affected by HIV, mortality estimates based on maternal orphanhood data collected in surveys are biased because infected women tend to have low fertility and because a proportion of the potential respondents are infected by their mothers and die at an early age. The maternal orphanhood data for Eastern African countries (excluding Madagascar), Central African Republic, and Côte d'Ivoire have been corrected for these selection biases using estimates of the prevalence of infection among mothers at the time the children were born and a recently developed adjustment procedure (Timæus and Nunn, 1997). In these countries, life table survivorship was estimated from data on children aged 5-9 using regression models that embody the unusual age pattern of mortality in populations with significant numbers of HIV-related deaths (Timæus and Nunn, 1997). By contrast, data on children aged 10-14 in these countries (which reflect more distant mortality), and on both age groups in other parts of Africa, were analysed using methods developed for populations with more usual age patterns of mortality (Timæus, 1992). Men's survivorship was

estimated from age 35 to 50 using an unpublished regression model fitted to the simulations described in Timæus (1992). For this first pair of age groups, this model yields more robust estimates than the published model, which estimates fathers' survivorship over the average of ten years that the respondents have been alive.

Mortality estimates obtained from the DHS household schedule data using these methods are presented in table 10. The most up-to-date of the three sets of estimates are those of women's mortality between ages 25 and 35. These estimates suggest that early adult mortality is higher throughout Eastern Africa, in the Central African Republic, and in Côte d'Ivoire than in countries where HIV was less prevalent at the time that the data were collected. The proportion of women dying between ages 25 and 35 is 9 per cent or more in seven countries, all of which developed a severe epidemic in the 1980s. It was 13 per cent in Uganda and Zambia by the early 1990s.

The estimates for women across the age range 25 to 40 years and those for men reflect

TABLE 10. PROBABILITIES OF DYING IN ADULTHOOD PER 1,000 ESTIMATED FROM ORPHANHOOD, DEMOGRAPHIC AND HEALTH SURVEYS, HOUSEHOLD DATA, COUNTRIES IN SUB-SAHARAN AFRICA, VARIOUS DATES

Country	Date of fieldwork	Women's mortality		Men's mortality
		19 <sub>25</sub>	19 <sub>25</sub>	19 <sub>35</sub>
Eastern Africa				
Kenya	1993	61	36	95
Madagascar	1992	61	90	110
Malawi	1992	111	91	98
Rwanda	1992	88	80	123
Uganda	1995	134	137	175
U. R. of Tanzania	1991-1992	74	61	92
	1996	89	80	108
Zambia	1992	96	73	94
	1996-1997	130	119	155
Zimbabwe	1994	90	72	117
Middle Africa				
Cameroon	1991	29	49	80
Central African Republic	1994-1995	100	79	133
Southern Africa				
Namibia	1992	27	43	93
Western Africa				
Bénin	1996	37	49	71
Burkina Faso	1992-1993	46	63	97
Côte d'Ivoire	1994	74	57	74
Ghana	1993	38	54	86
Mali	1995-1996	31	43	63
Niger	1992	38	73	70
Senegal	1992-1993	27	46	75

Source: Ian M. Timæus, "Impact of the HIV epidemic on mortality in sub-Saharan Africa: evidence from national surveys and censuses", *AIDS Supplement*, vol. 12 (1998 in press).

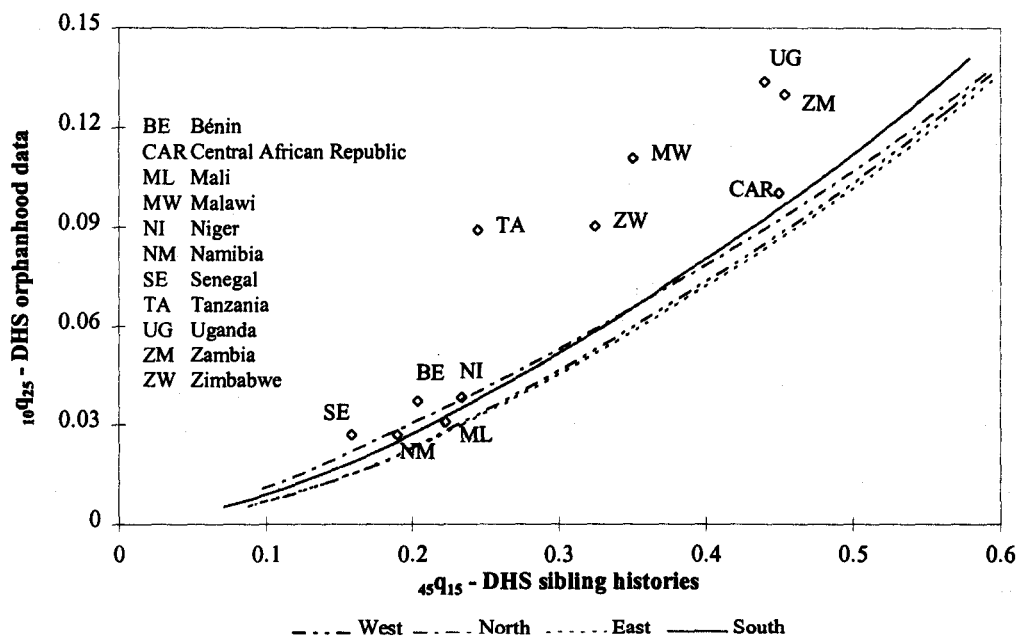
mortality over a longer period before their collection. Again, nearly all the estimates for countries in which HIV is believed to be prevalent are higher than those for other countries. Madagascar also shows up as a high mortality country, however, while Kenya has fairly low mortality. In Côte d'Ivoire and in all the Eastern and Middle African countries except Cameroon and Uganda, more mothers of children aged 5-9 years died between ages 25 and 35 than mothers of children aged 10-14 years died between ages 25 and 40. This suggests that the mortality of young women has risen sharply in recent years.<sup>2</sup>

Because of the profound but poorly understood effect that AIDS has on the overall age pattern of mortality, it is impossible to extrapolate from the measures in table 10 to mortality over a wider range of ages. However, the estimates of women's mortality between ages 25 and 35 reflect mortality in the eight or so years before they were collected.

This is approximately the period referred to by the sibling history estimates. Figure 25 compares these two sets of data. The relationship between  ${}_{45}q_{15}$  and  ${}_{10}q_{25}$  in the four families of Princeton model life tables is also shown (Coale and Demeny, 1983).

The orphanhood and sibling history estimates correlate closely: two very different methods of estimation agree as to the severity of adult mortality in these countries. In the five low mortality countries, the relationship between the mortality of young women and overall adult mortality is very close to that in existing systems of model life tables. In the Central African Republic, the mortality of young women is somewhat higher than expected. In the other five countries, young women have exceptionally high mortality relative to mortality in middle age. The size of the excess could be exaggerated in Tanzania, where the sibling histories may underestimate mortality. Malawi, Uganda, Zambia,

**Figure 25. Relationship between women's mortality between ages 25 and 35, according to orphanhood data, and women's mortality between ages 15 and 60 according to sibling history data**



Source: Table 8.



and Zimbabwe, however, are the countries in which the sibling history data indicate that mortality has risen most.

Similar plots (not shown) reveal that none of the maternal orphanhood estimates based on 10-14 year old respondents or paternal orphanhood estimates represent much higher early adult mortality than one would expect. These estimates reflect the mortality of slightly longer ago than the maternal orphanhood data for children aged 5-9. Thus, the unusual age pattern of mortality found in Eastern Africa results from a very recent rise in the mortality of young women. The conclusion that mortality increase in this part of the continent has been concentrated in early adulthood adds weight to the view that the cause of the rise in mortality is HIV.

#### E. DISCUSSION

The availability of data for the study of mortality in sub-Saharan Africa has improved during the last five years. This reflects the large number of DHS surveys conducted in the region. Unfortunately, no DHS survey has been undertaken in two of the most populous countries in Africa, the Democratic Republic of Congo and Ethiopia. Few other data are available for either of these countries and nationally representative data on mortality in Nigeria are also rare. Moreover, the information that is available on countries such as Botswana and Burundi is too out of date to provide anything more than a baseline against which the impact of HIV could be measured in future.

The DHS programme only collected information that can be used to measure adult mortality in a subset of the countries where it conducted surveys. Thus, no adult mortality data exist for many African countries in which levels and trends in child mortality are known. Even in countries where some data on adults exist, they have often been collected in just one inquiry using a single set of questions, thereby limiting the confidence with which estimates can be made from them. One reason for the shortage of data on adult mortality is that measuring it is more difficult than meas-

uring child mortality. Arguably, however, against the background of the AIDS epidemic, this means that disproportionate resources should be put into the collection of data on adults, using the entire range of methods at our disposal.

Few population-based data on causes of death in Africa exist for either children or adults. Just about enough information exists to identify the main public health problems of the region. It is impossible however, to map variation in the profile of causes of death across Africa or to identify the progress of individual countries through the epidemiologic transition. In the least developed countries of Africa this is not a major problem: the priorities for action are clear. In those countries that have made more progress against infectious disease and in which administrative capacity is greater, however, the lack of such data is becoming a significant obstacle to the rational allocation of resources.

Africa remains a high mortality region. It also continues to be characterised by very diverse mortality conditions, making it difficult to summarise the situation in the continent. By the middle to late 1980s, Western Africa contained some of the lowest mortality countries in Africa, such as Ghana, but also some of those with the highest mortality, such as Niger. Both child and adult mortality fell rapidly in most of Western Africa during the 1970s and 1980s. However, this trend tapered off in some of the lower mortality countries during the 1980s.

All three Middle African countries on which we have data are characterised by high adult mortality but relatively low child mortality. At least until the onset of the AIDS epidemic, they were experiencing rapid mortality decline. Northern Sudan is characterised by low mortality in both childhood and adulthood, but by a slow rate of mortality decline. Southern Africa is characterised by some of the lowest levels of child mortality in Africa, by moderate mortality among adult women, but by quite high mortality among adult men. The rate of decline in mortality has been slow in this part of Africa, especially for adults.

By the mid-1980s, the level of mortality varied markedly across Eastern Africa. The area included both high mortality countries, such as Burundi and Malawi, and low mortality countries, such as Kenya and Zimbabwe. Child mortality fell steadily in most of Eastern Africa during the 1970s and early-1980s, although not as rapidly as in Western Africa, but typically adult mortality fell rather slowly. Since the late 1980s, adult mortality has risen substantially in several Eastern African countries. Child mortality in these countries has followed diverse paths: it has continued to fall slowly in the higher mortality countries, stagnated in Zimbabwe, and risen markedly in Zambia.

The estimates presented here of African mortality in the second half of the 1980s document the closing chapter of a history outlined in previous reviews of the subject (Hill, 1993; Timæus, 1993). By then, the continuing decline in mortality in the higher mortality Western African countries and Malawi, combined with limited decline in Southern Africa and the slowing of decline in some other low mortality countries, had almost obliterated the regional patterning of mortality that was so clear in the 1960s. Excepting those countries held back by civil war or particularly incompetent and corrupt governments, the level of mortality in different African countries was showing signs of partial convergence.

Earlier research suggested that, except in countries disrupted by civil war, mortality was in continuous decline throughout the African region. The estimates presented here dispel any illusion that this is an inevitable process. Mortality decline slowed or stopped in a number of African countries during the 1980s, albeit mainly those with fairly low mortality. War was not a factor in most of these countries and adverse trends developed too early in several of them for AIDS deaths to be the full explanation but what mechanisms were involved is unclear. While the stalling of the mortality transition must be rooted in the economic difficulties of much of Africa, the long-term failure to develop human resources is probably a more serious issue than the immediate impact of fluctuations in disposable income. While the history of Ghana in the

1980s reveals that renewed mortality decline remains a possibility, it is one that may not be realised elsewhere.

This study has documents the initial impact of the HIV epidemic on mortality in a few sub-Saharan African countries. All the data available for analysis here were collected more than two years before this chapter was written. In general, they can only document mortality up till three or four years before they were collected. Moreover, the prevalence of HIV infection has been rising rapidly and the number of AIDS deaths depends on how many people were infected several years earlier. Thus, many of the mortality data presented here reflect the state of HIV epidemic over a decade ago. It is unlikely that more than about 8 per cent of adult women were infected at that time in even the most severely affected countries. Today, this proportion is at least three times higher.

Even in its initial stages, the HIV epidemic had a dramatic impact on mortality in several African countries. For example, the probability of dying between ages 15 and 60 in Zimbabwe in the mid-1980s was less than 20 per cent; ten years later it was more than 40 per cent. In the space of a decade, one of the lowest mortality countries in Africa became one of those with the highest mortality. Even in the early stages of the HIV epidemic, adult mortality in affected countries rose to levels more characteristic of the 1950s than the 1990s. Such statistics emphasise, if it needs emphasising, that HIV is the most important public health problem facing Africa. By now, mortality will have begun to rise sharply not just in a few countries but across much of the continent. This huge increase in mortality represents a major setback to development. In many countries, AIDS mortality will overwhelm the progress made in the control of infectious disease mortality during the last half century. The primary determinant of life expectancy in the African region in future will be severity of the AIDS epidemic in the country concerned.

Reporting both of orphanhood and of the ages and dates of death of respondents' siblings are subject to errors and biases. Except

in Tanzania, however, the agreement between the two sources as to the relative severity of adult mortality is close. Furthermore, the evidence from the sibling histories as to where, and how rapidly, mortality has risen is supported by the comparisons with data collected in earlier inquiries in figure 24. Crosschecks such as these leave scope for considerable error in sibling-history estimates of both the level and trend in mortality. However, they suggest strongly that the approach can reveal broadly what is happening to adult mortality in Africa.

Failure to report deaths and other likely biases in the orphanhood and sibling data result in underestimation rather than overestimation of mortality. Equally, because these problems are more serious for distant events than recent ones, the results are more likely to exaggerate than underestimate the rate of increase in mortality. Thus, if the retrospective data are biased, mortality in Eastern Africa was probably even higher in the early 1990s than this chapter suggests but this situation may have developed more gradually than indicated.

Though retrospective data on adult mortality suffer from deficiencies, to try to put them to good use seems imperative. It is impossible to obtain the information that they can provide about the HIV epidemic in any other way. Knowledge of the epidemiology and natural history of HIV infection in Africa exists only for a few small populations. One cannot estimate levels and trends in mortality in particular African countries from mathematical models of the epidemic when few of the crucial parameters involved are known. To neglect the demographic data available for national populations in Africa leaves only the data from a few cities and longitudinal studies of small populations to provide direct documentation of this demographic catastrophe.

It is regrettable, therefore, that, more than a decade after the possible future impact on adult mortality of AIDS in Africa became clear (Anderson and others, 1988), little has been done to improve the scope and quality of the data collected on adult deaths. A first priority is to promote interest in existing data and the capacity to analyse them and to integrate

the findings of such analyses into the policy and planning process at the national level. Second, a great deal would be learnt if the questions needed to apply existing methods were included in more of the 2000 round of censuses in Africa and in more of the national surveys that are to be conducted anyway. Third, research into improved methods is required.

The sibling histories collected by the DHS programme of surveys are one of the most useful sources available for study of the mortality impact of AIDS. In addition to countries examined here, such data have been collected in six further African countries. They include surveys of several other countries thought to be affected severely by the HIV epidemic. Nevertheless, it seems an ironic comment on the priorities of donors and governments that these crucial data have been generated as a by-product of efforts to measure maternal mortality. Moreover, the decision whether to continue to collect them is likely to ignore their value for monitoring the impact of the AIDS epidemic. Maternal causes kill between 100,000 and 200,000 women in Africa annually (Murray and Lopez, 1994; 1997). AIDS almost certainly already kills more Africans than this each year. Moreover, the number of AIDS deaths will rise sharply during the next decade.

One reason why so few data have been collected on adult mortality in the 1990s is simply inertia. Adult mortality has not been a priority for either health programmes or data collection for at least 25 years. Fertility surveys have become established as the main form of demographic inquiry in developing countries and have seldom included questions designed to measure adult mortality. Both officials in national statistical offices in Africa and their advisers and consultants have failed to rise to the challenge posed by the HIV epidemic. Equally, little impetus to change has come from donors. The DHS programme is funded by USAID. The only other donor with a major commitment to funding the collection of demographic data in the developing world is UNFPA. In recent years, both agencies have expanded their agenda from family planning to integrate other aspects of repro-

ductive health into their activities, including the prevention of HIV infection. However, AIDS mortality and adult mortality more generally do not fit well with their traditional concerns and have not been addressed in the same way.

It is also unfortunate that, while the US National Academy of Sciences' report, *Preventing and Mitigating AIDS in Sub-Saharan Africa* (Cohen and Trussell, 1996), emphasises the need to collect more data on the epidemiology of HIV, it does not recommend the collection of data on the mortality impact of AIDS. As a discipline, demography has not only failed in its intellectual and moral responsibility to describe one of the more important contemporary events in its field of study but has been remarkably unconcerned about this failure. While a great deal of intellectual energy has been devoted to modelling AIDS mortality in Africa, far less scientific effort has gone into the development, assessment and application of methods for the measurement of mortality impact. If demographic researchers fail to concern themselves with this issue, who else will?

#### NOTES

<sup>1</sup>The inventory of questions asked is based on published census reports and census schedules available in London or to the UN Population Division. Census data are classified in Table 6 as 'not known to have been published' if they could not be found by the author in London, by the UN Population Division in New York, or in the index of the International Census Collection maintained by the Population Research Center of the University of Texas at Austin. Some of these data will probably be published in the future - particularly those collected in 1993 and 1994. It is also likely that some mortality data have been collected and/or published that could not be traced for inclusion in table 6.

<sup>2</sup>In some countries, the finding that  $_{10}q_{25}$  is greater than  $_{15}q_{25}$  may be an artefact of the decision to use revised regression equations to estimate mortality from data on 5-9 year old children but not from those on children aged 10-14 years. However, both the conclusion that the six countries where HIV is thought to be most prevalent have exceptionally high early adult mortality and the conclusion that women's mortality has risen sharply would hold, albeit somewhat less dramatically, if the data on women in Eastern and Southern Africa were analysed using the methods presented in Timæus (1992).

#### REFERENCES

- Anderson, Roy M., Robert M. May and Angela R. McLean (1988). Possible demographic consequence of AIDS in developing countries. *Nature*, vol. 332, No. 6161, pp. 228-234.
- Barbieri, Magali, and Jacques Vallin (1996). Les conséquences de la crise économique africaine sur l'évolution de la mortalité. In *Crise et population en Afrique*, Jean Coussey and Jacques Vallin, eds. Les Etudes des CEPED, No. 13. Paris: Centre français sur la population et le développement, pp. 319-343.
- Berkley, Seth, Warren Naamara, Samuel Okware, Robert Downing, Joseph Konde-Lule, Maria Wawer and others (1990). AIDS and HIV infection in Uganda - are more women infected than men?, *AIDS*, vol. 4, No. 12, pp. 1237-1242.
- Blanc, Ann K., and Shea O. Rutstein (1994). The demographic transition in Southern Africa: yet another look at the evidence from Botswana and Zimbabwe. *Demography*, vol. 31, No. 2, pp. 209-215.
- Bongaarts, John (1996). Global trends in AIDS mortality. *Population and Development Review*, vol. 22, No. 1, pp. 21-45.
- Bradley, David J. (1991). Malaria. In *Disease and Mortality in Sub-Saharan Africa*, R. G. Feachem and D. T. Jamison, eds. Oxford: Oxford University Press, pp. 190-202.
- Brass, William (1975). *Methods for Estimating Fertility and Mortality from Limited and Defective Data*. Chapel Hill: University of North Carolina, Laboratories for Population Statistics.
- \_\_\_\_\_, and Carole L. Jolly (1993). *Population Dynamics of Kenya*. Population Dynamics of Sub-Saharan Africa. Washington, D.C.: National Academy Press.
- Chandramohan, Daniel, Gillian H. Maude, Laura C. Rodrigues and Richard J. Hayes (1994). Verbal autopsies for adult deaths: issues in their development and validation. *International Journal of Epidemiology*, vol. 23, No. 2, pp. 213-222.
- Coale, Ansley J., and Paul Demeny (1983). *Regional Model Life Tables and Stable Populations*. London: Academic Press.
- Cohen, Barney, and James Trussell, eds. (1996). *Preventing and Mitigating AIDS in Sub-Saharan Africa*. Washington, D.C.: National Academy Press.
- Ewbank, Douglas C., and James N. Gribble (1993). *Effects of Health Programs on Child Mortality in Sub-Saharan Africa*. Population Dynamics of Sub-Saharan Africa. Washington: National Academy Press.
- Feachem, Richard G., and Dean T. Jamison, eds. (1991). *Disease and Mortality in Sub-Saharan Africa*. Oxford: Oxford University Press.
- Garenne, Michel, and Oliver Fontaine (1990). Assessing probable causes of death using a standardised questionnaire: a study in rural Senegal. In *Measurement and Analysis of Mortality: New Approaches*, Jacques Vallin, Stan D'Souza and Alberto Palloni, eds. International Studies in Demography. Oxford: Clarendon Press, pp. 123-142.
- Gregson, Simon, Geoffrey P. Garnett and Roy M. Anderson (1994). Assessing the potential impact of the HIV-1 epidemic on orphanhood and the demographic structure of populations in sub-Saharan Africa. *Population Studies*, vol. 48, No. 3, pp. 435-458.
- Hill, Althea L. (1991a). Infant and child mortality: levels, trends, and data deficiencies. In *Disease and Mortality in Sub-Saharan Africa*, Richard G. Feachem and Dean T. Jamison, eds. Oxford: Oxford University Press, pp. 37-74.
- \_\_\_\_\_. (1992). Trends in childhood mortality in sub-Saharan mainland Africa. In *Mortality and Society in Sub-Saharan Africa*, Etienne van de Walle, Gilles Pison and Mpermbele Sala-Diakanda, eds. International Studies in Demography. Oxford: Clarendon, pp. 10-31.

- \_\_\_\_\_. (1993). Trends in childhood mortality. In *Demographic Change in Sub-Saharan Africa*, Karen A. Foote, Kenneth H. Hill and Linda G. Martin, eds. Population Dynamics of Sub-Saharan Africa. Washington, D. C.: National Academy Press, pp. 153-217.
- Hill, Kenneth (1991b). Approaches to the measurement of childhood mortality: a comparative review. *Population Index*, vol. 57, No. 3, pp. 368-382.
- \_\_\_\_\_, and Anne R. Pebley (1989). Child mortality in the developing world. *Population and Development Review*, vol. 15, No. 4, pp. 657-687.
- Kitange, Henry M., Harun Machibya, Jim Black, Deo M. Mtasiwa, Gabriel Masuki, David Whiting and others (1996). Outlook for survivors of childhood in sub-Saharan Africa: adult mortality in Tanzania. *British Medical Journal*, vol. 312, No. 7025, pp. 216-220.
- Makinson, Caroline (1993). Estimates of adult mortality in Burundi. *Journal of Biosocial Science*, vol. 25, No. 2, pp. 169-186.
- Murray, Christopher J. L., and Alan D. Lopez (1994). Global and regional cause-of-death patterns in 1990. *Bulletin of the World Health Organisation*, vol. 72, No. 3, pp. 447-480.
- \_\_\_\_\_, eds. (1996). *The Global Burden of Disease: a Comprehensive Assessment of Mortality and Disability from Diseases, Injuries, and Risk Factors in 1990 and projected to 2020*. Global Burden of Disease and Injury Series, No. 1. Cambridge: Harvard University Press.
- \_\_\_\_\_. (1997). Mortality by cause for eight regions of the world: Global Burden of Disease Study. *Lancet*, vol. 342, pp. 1269-1276.
- Nájera, José A., Bernhard H. Liese and Jeffrey Hammer (1993). Malaria. In *Disease Control Priorities in Developing Countries*, Dean T. Jamison, W. Henry Mosley, Anthony R. Measham and José L. Bobadilla, eds. New York: Oxford University Press, pp. 281-302.
- Nicoll, Angus, Ian Timæus, Rose-Mary Kigadye, Gijs Walraven and Japhet Killewo (1994). The impact of HIV-1 infection on mortality in children under 5 years of age in sub-Saharan Africa: a demographic and epidemiologic analysis. *AIDS*, vol. 8, No. 7, pp. 995-1005.
- Pison, Gilles, Kenneth H. Hill, Barney Cohen and Karen A. Foote, eds. (1995). *Population Dynamics of Senegal*. Population Dynamics of Sub-Saharan Africa. Washington, D.C.: National Academy Press.
- Preston, Samuel, Ansley J. Coale, James Trussell and Maxine Weinstein (1980). Estimating the completeness of reporting of adult deaths in populations that are approximately stable. *Population Index*, vol. 46, No. 2, pp. 179-202.
- Preston, Samuel H. (1984). Use of direct and indirect techniques for estimating the completeness of death registration systems. In *Data Bases for Mortality Measurement*. Population Studies, No. 84. New York: United Nations, pp. 66-76.
- Rutenberg, Naomi, and Jeremiah M. Sullivan (1991). Direct and indirect estimates of maternal mortality from the sisterhood method. In *Demographic and Health Surveys World Conference, August 5-7, 1991, Washington, D.C.* vol. 3. Columbia: Macro International, pp. 1669-1696.
- Somoza, Jorge L. (1980). *Illustrative Analysis: Infant and Child Mortality in Colombia*. World Fertility Survey Scientific Reports, 10. Voorburg, Netherlands: International Statistical Institute.
- Stanecki, Karen A., and Peter O. Way (1997). *The Demographic Impacts of HIV/AIDS, Perspectives from the World Population Profile: 1996*. International Programs Center Staff Paper, 86. Washington, D.C.: Population Division, U.S. Bureau of the Census.
- Stanton, Cynthia, Nouredine Abderrahim and Kenneth Hill (1997). *DHS Maternal Mortality Indicators: An Assessment of Data Quality and Implications for Data Use*. DHS Analytical Reports, No. 4. Calverton: Macro International.
- Thomas, Duncan, and Ityai Muvandi (1994a). The demographic transition in Southern Africa: another look at the evidence from Botswana and Zimbabwe. *Demography*, vol. 31, No. 2, pp. 185-207.
- \_\_\_\_\_. (1994b). The demographic transition in Southern Africa: reviewing the evidence from Botswana and Zimbabwe. *Demography*, vol. 31, No. 2, pp. 217-227.
- Timæus, Ian M. (1991a). Adult mortality: levels, trends and data sources. In *Disease and Mortality in Sub-Saharan Africa*, Richard G. Feachem and Dean T. Jamison, eds. New York: Oxford University Press, pp. 87-100.
- \_\_\_\_\_. (1991b). Estimation of adult mortality from orphanhood before and since marriage. *Population Studies*, vol. 45, No. 3, pp. 455-472.
- \_\_\_\_\_. (1991c). Estimation of mortality from orphanhood in adulthood. *Demography*, vol. 28, No. 2, pp. 213-227.
- \_\_\_\_\_. (1991d). Measurement of adult mortality in less developed countries: a comparative review. *Population Index*, vol. 57, No. 4, pp. 552-568.
- \_\_\_\_\_. (1992). Estimation of adult mortality from paternal orphanhood: a reassessment and a new approach. *Population Bulletin of The United Nations*, vol. 33, pp. 47-63.
- \_\_\_\_\_. (1993). Adult mortality. In *Demographic Change in Sub-Saharan Africa*, Karen A. Foote, Kenneth H. Hill and Linda G. Martin, eds. Population Dynamics of Sub-Saharan Africa. Washington, D. C.: National Academy Press, pp. 218-255.
- \_\_\_\_\_. (1998 in press). The impact of HIV/AIDS on mortality in sub-Saharan Africa: evidence from national surveys and censuses. *AIDS Supplement*, vol. 12.
- \_\_\_\_\_, and Andrew J. Nunn (1997). Measurement of adult mortality in populations affected by AIDS: an assessment of the orphanhood method. *Health Transition Review*, vol. 7, No. S2, pp. 23-43.
- United Nations (1983). *Manual X: Indirect Techniques for Demographic Estimation*. New York: United Nations Sales No. E.83.XIII.2.
- \_\_\_\_\_. (1988). *Mortality of Children under Age 5*. New York: United Nations. Sales No. E.88.XIII.4.
- Working Group on Demographic Effects of Economic and Social Reversals (1993). *Demographic Effects of Economic Reversals in Sub-Saharan Africa*. Population Dynamics of Sub-Saharan Africa. Washington, D.C.: National Academy Press.

## VI. MORTALITY IN LATIN AMERICA

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### A. INTRODUCTION

In Latin America, as in the developed countries, the demographic transition began with a drop in mortality rates, particularly in the first years of life. Within the region, the mortality transition has been very uneven: in a number of countries it has in fact been quite rapid, and these countries are now in a situation similar to that of the developed countries. Further gains in life expectancy at birth, particularly over the next few decades, are likely to be mostly the result of reductions in adult mortality. However, the health and mortality status of another large section of the population of Latin America is still highly unfavourable, and high rates of infant mortality persist.

With this in mind, and because numerous studies already exist on infant mortality, the emphasis in this paper is on developments in adult mortality: rates, trends and causes are examined, and the epidemiological transition now in progress in the countries of the region is discussed.

Given the problems of availability and quality of basic data, eight countries were selected to illustrate the various situations: Argentina, Chile, Costa Rica, Cuba, Guatemala, Mexico, Uruguay and Venezuela. The information comes chiefly from data bases in the Latin American Demographic Centre (CELADE) (life tables and population by sex and age) and the Pan American Health Organization (PAHO) (deaths by sex, age and cause of death). In addition, particularly valuable information was provided by the papers presented at the Seminar on the Causes and Prevention of Adult Mortality in Developing Countries organized by the International Union for the Scientific Study of Population (IUSSP), CELADE and PAHO, and held in Santiago, Chile from 7 to 11 October 1991.

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### B. AVAILABILITY AND QUALITY OF DATA

The data from which mortality trends are calculated should be a combination of vital statistics records and population censuses. Vital statistics show the number of deaths by sex, age and cause, while censuses indicate the population at risk of falling ill and dying, by sex and age. Tables 11 and 12 review the availability of data and the quality of sources, according to current level of life expectancy at birth (CELADE, 1997). Census and registration coverage was measured by comparing the unadjusted figures with those implied in the population estimates and projections produced by CELADE in conjunction with the relevant national agencies and in continuing exchange with the United Nations Population Division.

In the majority of the countries of Latin America, population censuses have been carried out systematically since the 1950s. In the 1990 round, only three countries (Costa Rica, Cuba and Haiti) did not conduct one. As can be seen from table 11, census coverage is reasonably good, although it varies from country to country. The omission rate in all but two countries is currently below 10 per cent and in more than half the countries it is below 5 per cent. Nevertheless, there is a striking lack of real improvement over time, and in a number of countries, data quality has actually deteriorated.

The picture as regards death registration is less encouraging. Registration varies far more than census coverage: in some countries registration is nearly complete, while in others it is almost non-existent or of little value owing to high rates of under-registration. Trends in under-registration have not improved substantially over time, except in Cuba and Venezuela. In recent years, the low and moderate mortality countries (Argentina, Costa Rica, Cuba, Chile, Mexico, Uruguay and Venezuela) have generally presented what has been regarded as low under-registration, with less than 10 per cent of deaths unrecorded. In the majority of the countries, however, omission rates exceed

TABLE 11. INDICATORS SHOWING QUALITY OF SOURCES OF DEMOGRAPHIC INFORMATION,  
COUNTRIES OF LATIN AMERICA, SELECTED DATES

Country	Percent omission in census			Percent under-registration of deaths		Percent causes of death ill-defined		
	Circa 1970	Circa 1980	Circa 1990	1960-1965	1975-1980	Circa 1993	Circa 1965	Circa 1992
Costa Rica	-0.5	5.4	<sup>b</sup>	11.7	13.7	0.0	9.2	2.1
Cuba	-0.3	0.8	<sup>b</sup>	26.4	4.3	0.0	1.6	0.4
Chile	6.1	1.2	1.9	3.7	6.3	0.0	7.7	5.3
Panama	4.8	5.8	2.6	25.2	25.2	26.4	18.6	7.7
Uruguay	1.4	1.9	<sup>a</sup>	6.0	2.9	2.6	6.7	6.4
Argentina	2.8	1.0	0.9	5.8	2.1	4.5	12.4	2.8
Venezuela	4.5	7.0	7.8	24.8	9.4	3.7	25.0	12.5
Mexico	3.4	0.9	1.8	11.3	9.3	7.5	18.6	1.7
Colombia	14.9	13.0	11.4	10.1	26.5	14.5	13.5	6.5
Dominican Republic	8.2	5.8	6.3	54.6	44.8	43.9	32.1	15.0
Ecuador	2.6	5.1	6.8	12.8	18.9	24.8	21.7	15.9
Paraguay	4.6	8.4	7.8	58.5	31.1	43.7	25.8	17.4
El Salvador	3.8	<sup>b</sup>	3.5	28.3	31.4	22.5	34.4	17.8
Honduras	8.3	<sup>b</sup>	7.2	49.8	51.0	52.7	41.0	48.1
Peru	2.7	4.3	2.7	42.6	35.7	46.2	13.7	31.7
Brazil	3.4	2.5	2.5	34.4	19.6	22.4	39.7	17.8
Nicaragua	11.0	<sup>b</sup>	<sup>a</sup>	56.4	39.0	44.8	19.9	4.7
Guatemala	8.8	14.2	13.8	12.7	4.9	2.5	16.0	10.0
Bolivia	5.9	<sup>b</sup>	6.7	<sup>c</sup>	<sup>c</sup>	<sup>c</sup>	<sup>c</sup>	<sup>c</sup>
Haiti	6.1	9.3	<sup>b</sup>	<sup>c</sup>	<sup>c</sup>	<sup>c</sup>	<sup>c</sup>	<sup>c</sup>

Sources: Prepared by the author, on the basis of published censuses and registers, and of estimates; Pan American Health Organization (PAHO), *Health Statistics from the Americas*, Scientific publication Nos. 542, 556 and in press (Washington, D.C., 1992, 1995 and 1997); Latin American Demographic Centre (CELADE), "Latin America: Population projections 1950-2050", *Demographic Bulletin No. 59* (Santiago, Chile, CELADE, 1997); F. Jaspers and H. Orellana, "Evaluation of vital statistics for the study of causes of death, in *Adult Mortality in Latin America*, I. Timæus, J. Chackiel and L. Ruzicka, eds. (Clarendon Oxford Press, IUSSP, 1996).

<sup>a</sup>Census not yet evaluated.

<sup>b</sup>No census taken.

<sup>c</sup>No information available.

20 per cent and in some they are higher than 50 per cent. The most notable exceptions are Panama, with low mortality and high under-registration of deaths and, at the other extreme, Guatemala, with high mortality but with data of reasonable quality.

Registration coverage problems have led in past decades to the widespread use of indirect techniques to estimate both mortality and fertility in the region. Table 12 shows the sources and methods on which the current child and adult mortality estimates for each country are based. As expected, countries with low mortality base their estimates on their registers, which are generally of high quality, but countries with moderate or high mortality must resort to indirect techniques based on retrospective questions in censuses and surveys and certain information from their vital statistics records. Table 12 shows the sources and methods used in obtaining the estimate most acceptable to

CELADE and the government institution of countries in the preparation of demographic estimates. In a number of cases, other estimates were available but were not reliable enough to be considered. Moreover, in cases where vital statistics are indicated as the selected source, they may have required correction, since the evaluation process has revealed varying degrees of under-registration (table 11).

For child mortality, the most widely used indirect technique is estimation based on information on children ever born and children surviving (Brass, 1975; United Nations, 1983), obtained by means of retrospective questions in censuses and surveys. The most widely used surveys are those that include birth histories, such as the World Fertility Survey (WFS) and, more recently, the Demographic and Health Surveys (DHS). Such surveys also provide direct estimates, by calculating death probabilities from the number of deaths and the length

TABLE 12. SOURCES AND TECHNIQUES USED IN ESTIMATING MORTALITY,  
COUNTRIES OF LATIN AMERICA, 1965-1970 AND 1985-1990

Country	1965-1970							1985-1990						
	Child mortality			Adult mortality				Child mortality			Adult mortality			
	VS	RQC	RQS	VS	GBE	RQC	RQS	VS	RQC	RQS	VS	GBE	RQC	RQS
Costa Rica	x	x		x				x			x	x		
Cuba	x			x				x			x			
Chile	x			x				x			x			
Panama	x				x				x			x		
Uruguay	x			x				x			x			
Argentina	x			x				x	x		x			
Venezuela	x				x			x	x			x		
Mexico	x			x				x			x			
Colombia <sup>a</sup>		x	x		x				x	x				
Dominican Republic <sup>a</sup>		x		x						x				
Ecuador		x			x				x			x		
Paraguay		x	x			x			x	x		x	x	
El Salvador	x	x			x			x		x	x			
Honduras		x	x			x	x		x			x		
Peru		x	x			x	x		x	x		x	x	
Brazil		x			x				x	x		x		
Nicaragua		x	x		x	x			x	x		x		
Guatemala	x	x			x	x				x		x		
Bolivia			x			x			x	x				x
Haiti <sup>a</sup>			x		x					x				

Source: Latin American Demographic Centre (CELADE), reports prepared for the United Nations Population Division.

<sup>a</sup> Adult mortality 1985-90 is projected.

VS- vital statistics

GBE- growth balance equation

RQC- retrospective questions in censuses

RQS- retrospective questions in surveys

of exposure to the risk of dying of children ever born reported by women of child-bearing age.

In estimating adult mortality, particularly mortality among the elderly, the data situation is more precarious than in child mortality. Even in countries with good vital statistics there are grave doubts as to the quality of mortality estimates for the elderly, as they may be biased by age misreporting in censuses and even in death registration (Dechter and Preston, 1991; Grushka, 1996). In the low-mortality countries, not surprisingly, calculations of mortality rates after the age of 5 are based on death registration. In countries with moderate or high mortality, the "growth balance equation", or some similar indirect technique, is most widely used (United Nations, 1983). Methods of this kind are based mainly on the age structure of deaths registered in vital statistics records and the population by age, as pro-

vided by censuses. In only very few cases is the age structure of deaths obtained by means of special questions in censuses or surveys. Such techniques are used to adjust mortality rates by age calculated in the traditional way (deaths registered/census population). One study that set out to evaluate indirect techniques for estimating mortality, particularly adult mortality, concluded that "the most satisfactory basis seems to be registered deaths by age and sex, at least if coverage is 75 per cent or more, and adjustment by comparison with the age distribution" (referring to the growth balance equation technique) (Hill, 1984).

It is interesting to observe the decline in the use of techniques for estimating adult mortality based on census and survey questions on the survival of relatives—maternal and paternal orphanhood, for example, or widowhood. In the period 1965-1970, six countries based their



estimates on information of this kind, but in the period 1985-1990 only three did so. This fall from favour may be due to growing doubt on the part of the experts—including the techniques' own designers—as to the effectiveness of such methods (Hill, 1984). Problems arise from informants' selectivity and bias in their responses, and from the fact that the estimates relate to a time several years before the survey or census takes place. Despite efforts to rehabilitate them (Chackiel and Orellana, 1985; Timæus, 1996), such techniques are not in widespread use.

Jaspers and Orellana (1996) have analysed the under-registration of deaths in the period 1980-1985 in more detail, giving a breakdown by sex and broad age groups. Under-registration was found to be systematically lower in adults (persons aged 15 and over), and particularly in the older age groups (open-ended group). This may be due in large part to the high level of under-registration of very early deaths, which are concealed under the heading of late foetal deaths. However, the estimates used as a basis for comparison when assessing the level of under-registration of adult deaths are not very reliable since, as previously mentioned, no adequate indirect techniques exist. In a large number of countries, patterns of adult mortality by age, particularly among the elderly, are based on standard models.

As regards under-registration by sex, the majority of countries were found to have greater coverage of male deaths, chiefly among young adults. This result is surprising: one would expect the opposite to be the case, since there is a greater likelihood of male deaths associated with social, political or criminal conflict being concealed. One explanation may again be the problem of indirect techniques, which yield less reliable results for men and could quite well lead to an underestimation of male mortality.

It is less easy to investigate causes of death, for in addition to the problems of under-registration already discussed, there are difficulties in correctly diagnosing morbidity leading to death. Table 11 shows the proportion of ill-defined causes of death, which is high in a large number of countries in the region. Surprisingly, some countries with high under-registration, such as Colombia and Nicaragua, present a low percentage of ill-defined causes. Jaspers and Orellana (1996)

attribute this situation to the fact that the majority of those whose deaths are recorded belong to above-average socio-economic strata and to a generally urban population, and are therefore likely to receive medical attention prior to death. On analyzing the percentages of ill-defined causes by sex and broad age groups, they also found that the proportion was slightly higher among women and gradually increased with age. The highest percentages of ill-defined causes among women are to be found mainly in the fertile ages and are probably associated with deaths from clandestine abortions and/or because a greater number of male death are due to more readily known external causes (violence) (Arriaga, 1997). Given the levels of under-registration and the percentage of ill-defined causes, analyses of mortality by cause of death would have to ignore a high percentage of the deaths in these countries. Extreme cases are Honduras and Peru, where the two indicators combined give a total of more than 80 per cent of deaths where the cause is unknown. Interestingly, although levels of under-registration do not appear to decline over time, an improvement can be seen in the definition of cause of death, which is consistent with an increase in the percentage of medically-certified deaths.

Bearing in mind data availability and quality, the remainder of this paper examines selected countries representing different mortality levels and trends, as follows:

- Countries with very low mortality - recent transition: Chile, Costa Rica and Cuba
- Countries with low mortality - early transition: Argentina and Uruguay
- Countries with low and moderate mortality - recent transition: Mexico and Venezuela
- Countries with high mortality: Guatemala.

The analysis that follows is based on information taken from the life tables by sex prepared by CELADE in conjunction with the relevant national agencies (CELADE, 1997). These tables, which were drawn up as input for population estimates and projections, cover the historical period 1950-1995 and are projected to 2050. Generally speaking, the most reliable estimates are those for the years prior to 1990, in some cases because the last census was carried out in 1990 or before, and in others because the information from the registers is not up-to-date. Costa Rica and Cuba have not taken censuses this decade (the latest were in

1980 and 1984, respectively) and the data for Uruguay's 1996 census are not yet available (the previous one was in 1985).

### C. MORTALITY TRENDS

Average life expectancy at birth ( $e_0$ ) in Latin America in the period 1990-1995 stood at around 69 years, and it should by now have reached 70, the target set for the end of the century by the World Health Organization (WHO), and for the year 2005 by the International Conference on Population and Development, held in Cairo in 1994 (United Nations, 1995). Latin America has the lowest mortality of all the developing regions, and its average life-span has increased by 18 years in four decades (United Nations, 1996). Only East Asia has done better, having gained 26 years of life over a similar period, to now have reached similar levels of life expectancy at birth.

There is, however, great diversity between countries. In Chile, Costa Rica and Cuba, on the one hand, life expectancy at birth is around 75 years, a figure that exceeds the average for the most developed regions in the world (74.2 in 1990-1995; United Nations, 1996), while in Haiti it is less than 60 and in Bolivia it is only just above 60. Thus there is a difference of more than 15 years between the region's two extremes, which, in terms of average regional gains in a period of time, implies a lag of some 45 years. Of the countries studied in this paper, Guatemala has the highest mortality, with an  $e_0$  of 63 years. Although the average life-span in Guatemala has increased by somewhat more than 20 years since 1950-1955, its initial level of  $e_0$  was very low (42 years). Uruguay and Argentina are exceptional cases since their mortality rates fell in the first half of the century, and their more recent gains in life expectancy have therefore been smaller. Half of the twenty countries of the region have already met the life-expectancy target set at the Cairo conference (70 years by 2005) and, according to projections, only three countries are unlikely to do so.

Generally speaking, as might be expected from what is known about the patterns of mortality by sex, women's average life-span is longer than men's (table 13). Nevertheless, the sex differential in life expectancy at birth, and the changes in the sex differential, vary from country to country, even among countries with similar mortality levels. Argentina, for exam-

ple, presents the greatest difference between women and men in terms of life expectancy at birth (7 years in 1985-90), while the difference in Cuba is half that figure. Interestingly, Cuba and Costa Rica, which have the lowest mortality, show the narrowest gaps between the sexes, and Cuba's differential of 3.5 years has remained relatively stable for the last 45 years. At current average mortality levels, the differential is normally around 6 years.

Generally speaking, the sex differential has tended to widen yet further in women's favour, increasing from 2.5-3 years at life expectancy at birth of 60 years, to 6 years at life expectancy at birth of over 70. On the other hand, men and women have tended to make similar gains in terms of years of life, which means that the differentials should stabilize in the future. Moreover, in certain countries men are beginning to make greater gains than women, which is narrowing the gap between the sexes. This is already happening in Chile, Mexico, Uruguay and Venezuela, where life expectancy at birth for both sexes reaches 68-70 years. Are we seeing a reversal of the tendency for women's advantage to increase? If so, we may expect in the future to see a converging trend in men's and women's life expectancies at birth, or at least a stabilization of the gap.

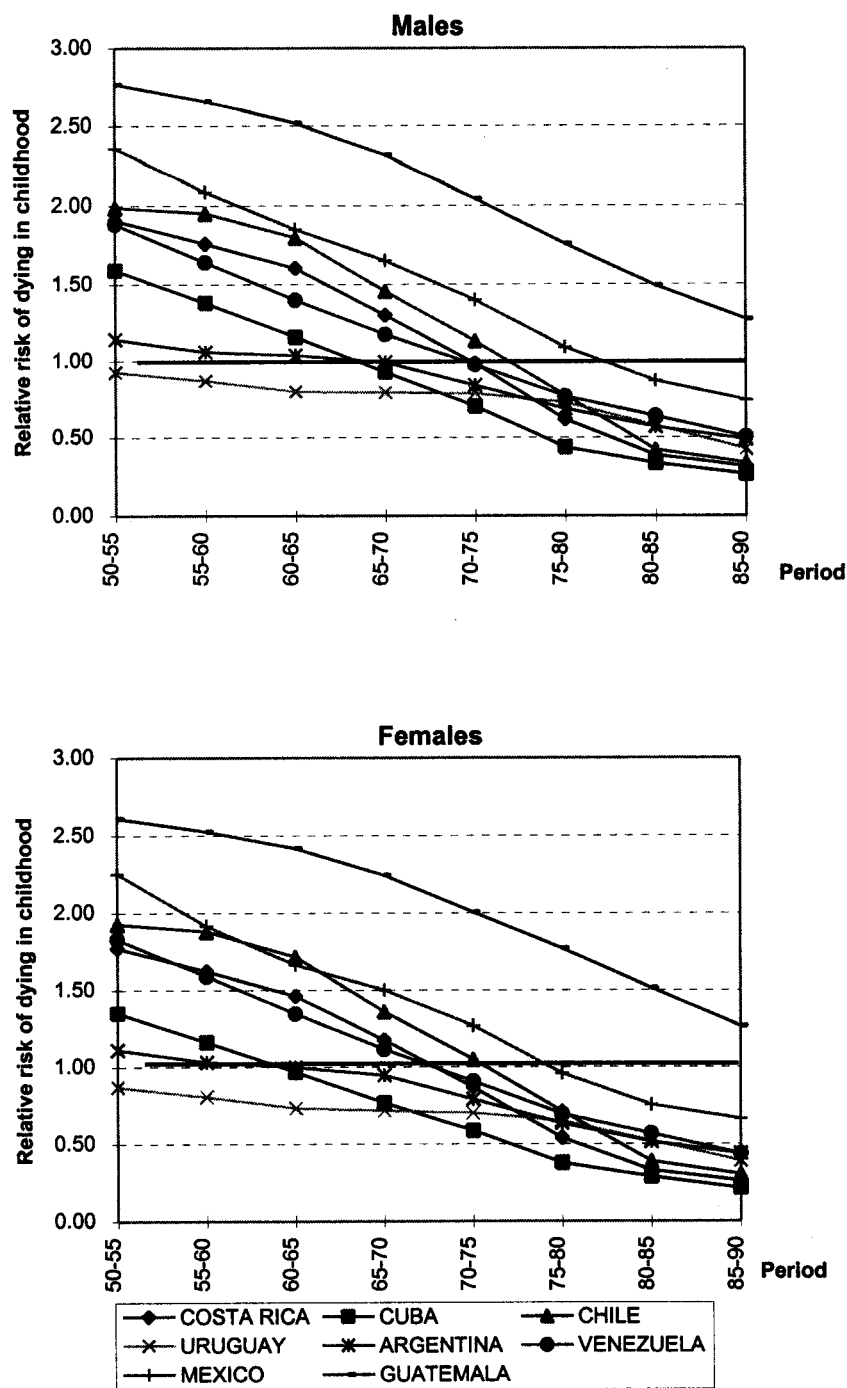
The decline in both male and female mortality is mainly a result of the successful reduction of mortality at early ages, particularly infant mortality (table 13). In order to analyse the effects of the change in mortality in the first five years of life, an indicator has been adapted from Vallin (1996). Life expectancy at birth is multiplied by the life table mortality rate for children aged 0-4 ( $m_{0-4} \cdot e_0$ ) to obtain the ratio of the mortality rate at ages 0-4 to the overall mortality rate, which expresses the relative risk of dying in childhood. If this indicator equals 1, the risk of dying is similar for under-fives and over-fives. When mortality is high, the ratio is greater than 1: during the 1950s it was 2.8 in Guatemala, 2.3 in Mexico and 2.0 in Chile, indicating high excess mortality among children in relation to adults (figure 26). The ratio in low-mortality countries today is between 0.2 and 0.4, which means that the decline in mortality has been brought about through a substantial reduction of the relative and absolute risk of dying at the beginning of life. The countries that have made the biggest reductions in the ratio of child to adult mortality (an eight-fold decrease in 45 years) are those that have

TABLE 13. LIFE EXPECTANCY AT BIRTH, AT 5 YEARS AND AT 65 YEARS, AND INFANT MORTALITY RATE (PER 1,000),  
BY SEX, SELECTED COUNTRIES AND PERIODS

Indicators	Country and period							
	Costa Rica	Cuba	Chile	Uruguay	Argentina	Venezuela	Mexico	Guatemala
1950-1955								
Life expectancy at birth								
Both sexes	57.3	59.5	54.8	66.3	62.7	55.2	50.7	42.0
Male	56.0	57.8	52.9	63.3	60.4	53.8	48.9	41.8
Female	58.6	61.3	56.8	69.4	65.1	56.6	52.5	42.3
Life expectancy at age 5								
Both sexes	61.6	61.9	59.7	65.8	63.4	59.6	57.6	51.3
Male	60.7	60.8	58.0	62.9	61.1	58.4	56.1	51.4
Female	62.5	63.1	61.5	68.7	65.7	60.9	59.1	51.1
Life expectancy at age 65								
Both sexes	12.6	12.6	12.7	13.7	13.3	12.9	12.6	11.6
Male	12.2	12.4	11.8	12.3	12.1	12.3	12.2	11.4
Female	13.1	12.7	13.7	15.2	14.5	13.5	13.0	11.8
Infant mortality								
Both sexes	93.8	80.6	120.4	57.4	65.9	106.4	121.2	140.8
Male	101.0	90.8	128.0	62.1	69.9	110.9	133.8	150.3
Female	86.2	69.9	112.4	52.5	61.7	101.7	107.9	130.9
1970-1975								
Life expectancy at birth								
Both sexes	68.1	71.0	63.6	68.8	67.4	66.1	62.6	53.9
Male	66.1	69.4	60.5	65.6	64.1	63.3	60.1	52.4
Female	70.2	72.7	66.8	72.2	70.8	68.9	65.2	55.4
Life expectancy at age 5								
Both sexes	67.7	69.3	64.0	67.5	66.4	65.8	64.3	59.2
Male	66.0	67.9	61.1	64.5	63.3	63.2	62.1	57.8
Female	69.6	70.7	67.0	70.7	69.7	68.5	66.5	60.7
Life expectancy at age 65								
Both sexes	14.7	15.4	13.9	14.5	14.0	14.7	15.0	13.3
Male	14.0	14.6	12.8	12.9	12.5	13.5	14.5	12.8
Female	15.3	16.2	15.0	16.1	15.6	15.9	15.6	13.8
Infant mortality								
Both sexes	52.5	38.4	68.6	46.3	48.1	48.7	69.0	102.4
Male	58.5	43.0	74.3	51.3	52.2	53.4	77.2	108.4
Female	46.3	33.6	62.6	41.1	43.8	43.7	60.3	96.2
1985-1990								
Life expectancy at birth								
Both sexes	75.3	74.6	72.7	72.0	71.0	70.5	69.8	59.7
Male	73.1	72.8	69.6	68.9	67.6	67.7	66.8	57.3
Female	77.7	76.5	75.9	75.3	74.6	73.5	73.0	62.2
Life expectancy at age 5								
Both sexes	71.8	70.8	69.3	69.0	68.3	67.9	68.3	61.1
Male	69.7	69.1	66.3	66.0	65.0	65.2	65.5	58.7
Female	74.0	72.5	72.4	72.3	71.8	70.7	71.3	63.5
Life expectancy at age 65								
Both sexes	16.6	16.9	15.6	15.4	14.9	15.0	16.4	13.8
Male	15.4	16.0	14.2	13.5	13.1	13.9	15.5	13.4
Female	17.8	17.8	17.1	17.4	16.7	16.1	17.3	14.2
Infant mortality								
Both sexes	16.0	12.9	18.3	24.4	27.1	26.9	39.5	65.0
Male	17.9	14.6	19.9	26.7	30.0	30.3	43.0	69.6
Female	14.0	11.1	16.7	22.0	24.1	23.4	35.9	60.2

Source: Latin American Demographic Centre (CELADE), "Population projections: 1950-2050", *Demographic Bulletin No. 59* (Santiago, Chile, CELADE, 1997).

Figure 26. Relative risk of dying in childhood, by sex, selected countries of Latin America, 1950-1990



Source: Latin American Demographic Centre (CELADE), "Latin America: Life table 1950-2050", *Demographic Bulletin No. 61* (Santiago, Chile, 1997, in press).

achieved the region's highest life expectancies at birth. Once the ratio equals 1, it is mainly in adult ages that mortality gains will be made. Thus in Uruguay, where mortality has been relatively low since the first half of the century, declining values of less than 1 can be observed during the period in question, while in Guatemala, where mortality is still high, the indicator has only just begun to approach unity.

Analysis by sex shows that women consistently run less risk of dying in childhood than men, and that women's greater gains in life expectancy at birth are the result of bigger reductions in mortality at the start of life. The exception would appear to be Guatemala, where in recent years the indicator has not been noticeably higher for men. While the lag between the sexes in attaining the value 1 is roughly five years in countries where mortality has been moderately low for some time, it is less (around 2.5 years) in countries where mortality has fallen markedly in recent years. The difference may have to do with recent advances in averting premature adult death from causes affecting principally men, which could also explain the incipient narrowing of the sex differential in life expectancy.

The difference between child and adult mortality patterns is reflected in trends in life expectancy at birth and life expectancy at age 5 ( $e_5$ ). In the initial stages of the mortality transition, when the risk of dying in infancy is still very high, values for  $e_0$  are lower than values for  $e_5$ , that is to say, those who survive the first five, most risky, years of life are likely to live a greater number of years. Thus, at mid-century in Guatemala, for example,  $e_5$  was nearly 10 years higher than  $e_0$  (51 versus 42), which means that those who survived their early years had, partly through natural selection, a far greater life expectancy than those newly born. At that time, as table 13 shows, in other countries of the region where mortality was lower than in Guatemala but nevertheless relatively high ( $e_0$  around 55 and  $e_5$  of around 60),  $e_5$  was still from 4 to 7 years higher than  $e_0$  (figure 27). The only country whose  $e_0$  had already moved slightly ahead by 1950-1955 was Uruguay, where mortality rates declined earlier than those of the other countries of the region ( $e_0$  and  $e_5$  of around 66 years).

As infant mortality declines, the trend over time is for both life expectancies to converge and, in some cases, the difference should invert

itself (figure 27). Generally speaking, they converge at around 65 years, which is why convergence took place before the period under consideration in Uruguay and after it in Guatemala. In the other countries analysed,  $e_0$  was from 2 to 4 years higher than  $e_5$  by 1985-1990, with  $e_5$  standing at just over 70 years in Costa Rica, Cuba and Chile, just under 70 in Argentina, Uruguay, Mexico and Venezuela, and 61 in Guatemala. Since mortality at early ages is the chief determinant of the downward trend in mortality, the differences between countries are less pronounced in  $e_5$  than in  $e_0$ . Thus a comparison of the extremes shows that, while Costa Rica's  $e_0$  is 26 per cent higher than Guatemala's, its  $e_5$  is only 18 per cent higher. At present, at least in the low- and moderate-mortality countries, gains in  $e_5$  are diminishing, but are similar to gains in  $e_0$  (figure 27).

It follows from the analysis thus far that, in order to reduce mortality further, a large number of countries of the region must now target adult mortality, although in a sizeable group of countries and sectors infant and child mortality remain high.

#### D. MORTALITY BY SEX, AGE AND CAUSE OF DEATH

##### 1. *Child mortality (0-14 years)*

An analysis of mortality rates by age<sup>1</sup> for each sex shows that, as demonstrated in the preceding section, it is in child mortality that the greatest decline has taken place. Table 14 gives mortality rates by age and sex, from life tables, including those for the 0-14 age group, for the periods 1955-1960, 1970-1975 and 1985-1990. The variations in these rates fall into two periods, one between 1955-1960 and 1970-1975 and the other between 1970-1975 and 1985-1990 (table 15). In Uruguay and, to a lesser extent, Argentina, child mortality rates stalled or fell relatively slowly between 1955-1960 and 1970-1975, partly because rates were lower in these two countries than elsewhere. The biggest gains during this period were made in Cuba and Costa Rica (roughly 55 per cent), followed by Chile and Venezuela and, just behind them, Mexico. Reductions in Guatemala were substantial (around 40 per cent), but not sufficient given the country's mortality level. Mortality fell more sharply during the next 15 years, with reductions of between 50 per cent and 73 per cent in the recent-transition,

Figure 27. Difference between life expectancy at birth and life expectancy at age 5, and gains in years per five-year period for both indicators, 1950-1990

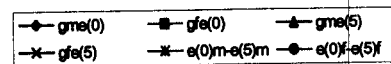
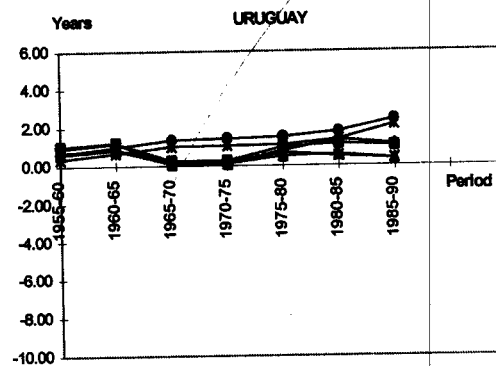
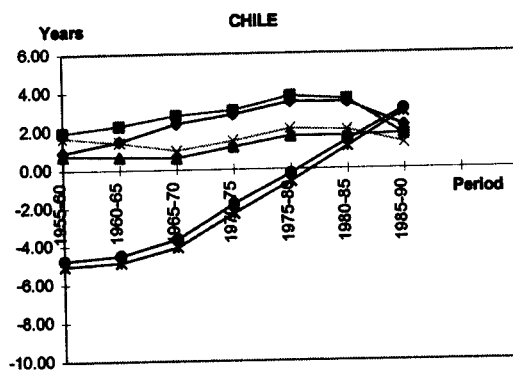
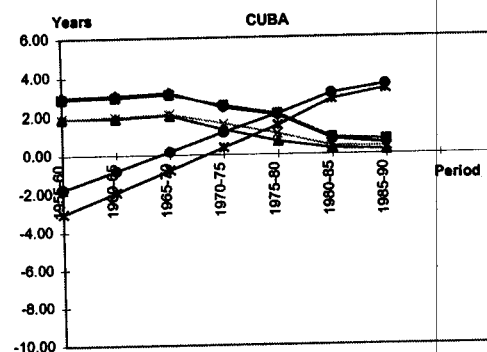
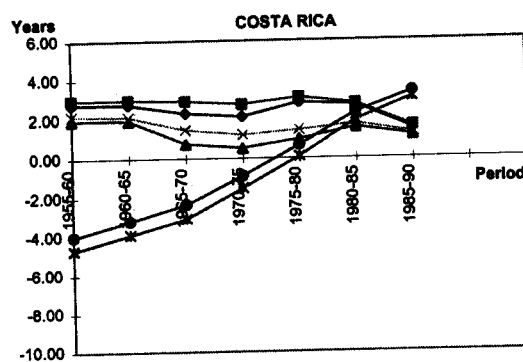
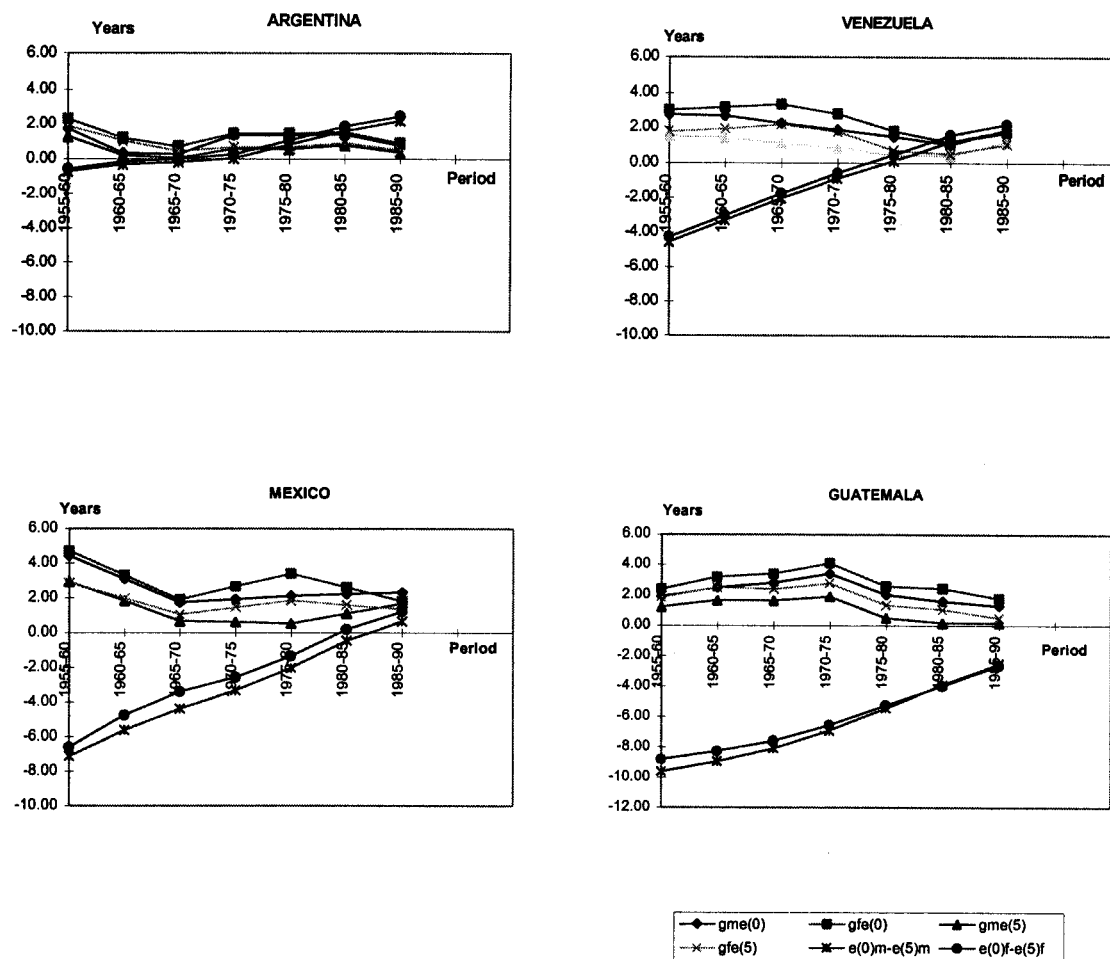


Figure 27 (continued)



gme(0), gains for males in e(0).

gfe(0), gains for females in e(0).

gme(5), gains for males in e(5).

gfe(5), gains for females in e(5).

e(0)m-e(5)m, difference in years between male life expectancy at birth and male life expectancy at 5 years old.

e(0)f-e(5)f, difference in years between female life expectancy at birth and female life expectancy at 5 years old.

Source: Latin American Demographic Centre (CELADE), "Latin America: Life table 1950-2050", *Demographic Bulletin*, No. 61 (Santiago, Chile, 1997, in press).

TABLE 14. LIFE TABLES MORTALITY RATES BY SEX AND AGE (PER 10,000),  
SELECTED COUNTRIES, 1950-1955, 1970-1975 AND 1985-1990

Sex and age	Country and period							
	Costa Rica	Cuba	Chile	Uruguay	Argentina	Venezuela	Mexico	Guatemala
1955-1960								
Male								
0-14	112.92	85.97	134.32	49.22	63.65	109.18	151.08	258.45
15-44	33.19	37.28	52.53	24.96	29.86	46.49	58.31	80.27
45-64	143.81	132.61	200.98	161.82	172.66	192.03	174.34	228.94
65 y +	777.24	777.79	822.53	798.81	796.56	789.35	743.72	855.20
Female								
0-14	98.96	69.59	117.91	41.08	56.36	99.52	130.61	244.16
15-44	29.24	30.46	39.18	15.54	22.43	34.76	44.44	83.09
45-64	121.86	101.16	139.37	88.23	99.20	171.32	138.39	209.93
65 y +	722.61	740.56	704.78	644.02	660.89	716.03	707.13	821.41
1970-1975								
Male								
0-14	55.14	37.66	68.41	43.45	48.49	59.01	88.56	158.31
15-44	24.62	18.99	41.78	22.31	26.56	34.03	45.57	57.78
45-64	114.82	93.27	183.76	147.89	163.26	151.19	154.33	184.61
65 y +	713.84	686.44	779.52	777.41	797.56	738.38	692.17	780.32
Female								
0-14	45.37	29.91	56.80	34.90	40.72	49.01	73.94	147.01
15-44	15.35	14.88	23.15	13.36	16.33	19.05	28.51	47.36
45-64	79.74	73.22	113.27	77.18	79.11	105.52	111.30	147.74
65 y +	652.30	616.08	667.82	619.63	639.75	629.10	639.23	726.80
1985-1990								
Male								
0-14	16.87	15.61	19.10	23.61	27.29	29.06	42.01	88.80
15-44	15.44	18.65	23.24	17.87	21.21	27.67	35.01	63.52
45-64	81.28	93.02	121.58	135.33	143.22	125.34	124.56	172.44
65 y +	648.98	624.99	703.61	739.99	762.34	719.73	643.29	748.21
Female								
0-14	12.99	11.57	15.44	19.20	21.72	22.69	33.76	81.91
15-44	7.92	12.87	9.90	10.15	12.22	12.73	14.40	37.46
45-64	52.68	67.39	68.59	76.40	66.28	77.61	80.14	125.51
65 y +	560.69	562.13	583.49	575.80	598.25	621.47	578.04	702.99

Source: Latin American Demographic Centre (CELADE), "Latin America: Life table 1950-2050", *Demographic Bulletin*, No. 61 (Santiago, Chile, 1997, in press).

low- and moderate-mortality countries, and around 45 per cent in Uruguay and Argentina, where the process began to gather pace once again. Even in Guatemala, a country where the variations were not as great as elsewhere in the region, mortality fell by more than 40 per cent during the second period. In all the countries analysed, female rates are lower than male rates and have also fallen further, particularly in low-mortality countries where the ratio of male to female rates is between 1.25 and 1.30 (figure 28).

The increase in child survival is due chiefly to success in combating communicable dis-

eases and, to a lesser extent, conditions originating in the perinatal period.<sup>2</sup> Where mortality is high, as in Costa Rica and Chile 30 years ago, or in Guatemala today, the prime causes of death are communicable diseases, followed by conditions originating in the perinatal period. Although there has been a considerable decline in mortality from these causes, they have retained their primacy but the order is reversed (table 16). An analysis of mortality rates by cause shows clearly that the pattern of change and the ratio between the sexes in the 10-14 age group reflects trends in these two main cause groups. The rates for the other causes show minor variations, with a moderate



TABLE 15. PERCENTAGE REDUCTION OF LIFE TABLES MORTALITY RATES BY SEX AND AGE,  
1955-1960/1970-1975 AND 1970-1975/1985-1990

Sex and age	Country and period							
	Costa Rica	Cuba	Chile	Uruguay	Argentina	Venezuela	Mexico	Guatemala
<i>1955-1960/1970-1975</i>								
Male								
0-14	51.16	56.19	49.07	11.72	23.82	45.95	41.38	38.75
15-44	25.83	49.07	20.46	10.61	11.05	26.81	21.85	28.03
45-64	20.16	29.67	8.57	8.60	5.44	21.27	11.48	19.36
65 y +	8.16	11.75	5.23	2.68	-0.13	6.46	6.93	8.76
Female								
0-14	54.15	57.01	51.83	15.04	27.76	50.76	43.39	39.79
15-44	47.51	51.16	40.91	14.00	27.22	45.21	35.85	43.00
45-64	34.57	27.62	18.72	12.52	20.25	38.41	19.57	29.62
65 y +	9.73	16.81	5.24	3.79	3.20	12.14	9.60	11.52
<i>1970-1975/1985-1990</i>								
Male								
0-14	69.40	58.56	72.07	45.66	43.71	50.76	52.56	43.91
15-44	37.28	1.80	44.38	19.93	20.13	18.69	23.16	-9.93
45-64	29.21	0.27	33.84	8.50	12.28	17.10	19.29	6.59
65 y +	9.08	8.95	9.74	4.81	4.42	2.53	7.06	4.12
Female								
0-14	71.38	61.34	72.82	44.98	46.66	53.71	54.34	44.28
15-44	48.37	13.49	57.23	24.02	25.17	33.16	49.47	20.90
45-64	33.94	7.95	39.45	1.01	16.22	26.45	28.00	15.05
65 y +	14.04	8.76	12.63	7.07	6.49	1.21	9.57	3.28

Source: Latin American Demographic Centre (CELADE), "Latin America: Life table 1950-2050", *Demographic Bulletin*, No. 61 (Santiago, Chile, 1997, in press).

decline in some countries and a slight rise or stability in others. Child deaths in recent years, therefore, are mainly due to conditions originating in the perinatal period, because of certain persistent endogenous conditions where fatalities are hard to avoid.

## 2. Mortality among young adults (15-44 years)

The pattern from the age of 15 onward is generally that mortality rates increase progressively with age, while reductions diminish. Thus the lowest rates correspond to the 15-44 age group and, in the most recent period analysed, range between 15.4 per 10,000 (Costa Rica) and 63.5 per 10,000 (Guatemala) for men and between 7.9 per 10,000 and 37.5 per 10,000 respectively for women. In this age group, the biggest reductions in mortality rates have occurred in the most recent period, except in Cuba, Venezuela and Guatemala (table 15). The countries where the downward trend has been most pronounced in recent years are recent-transition countries, some of which have achieved lower rates than the early-transition countries. Male mortality is invari-

bly higher than female and the gap widens over time. In certain cases, male rates are more than twice as high as female rates, and indeed sex differentials in mortality are wider in this age group than in any other at present (figure 28).

In terms of causes of death, the chief characteristic of mortality in this age group is the prominence among men of external causes of death, such as violence and injuries. The most important of these are motor vehicle traffic accidents, suicide, homicide and, in some countries, drowning and submersion. External causes far outweigh other causes of male mortality, which is why the highest levels of male excess mortality occur in this age group. For women, the main causes are neoplasms and, in second place, diseases of the circulatory system and external causes. The biggest inroads have been made on the rates for communicable diseases, which are high when mortality is high, followed by diseases of the circulatory system. Generally speaking, mortality from external causes in both sexes is relatively stable or exhibits a moderate decline in all the countries, with the exception of Guatemala, where there

Figure 28. Male excess mortality<sup>a</sup>, by age, selected countries of Latin America, 1950-1990

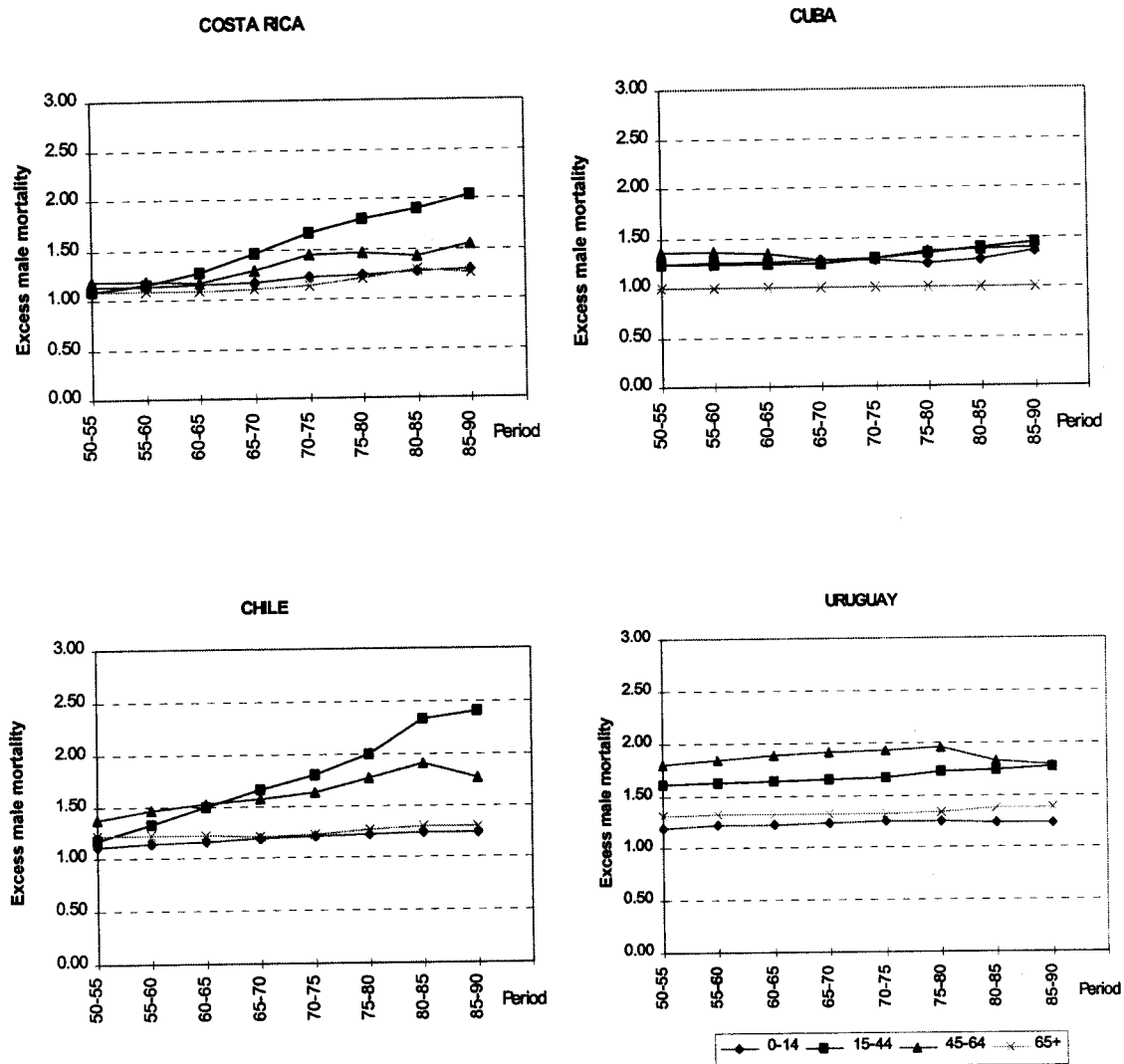
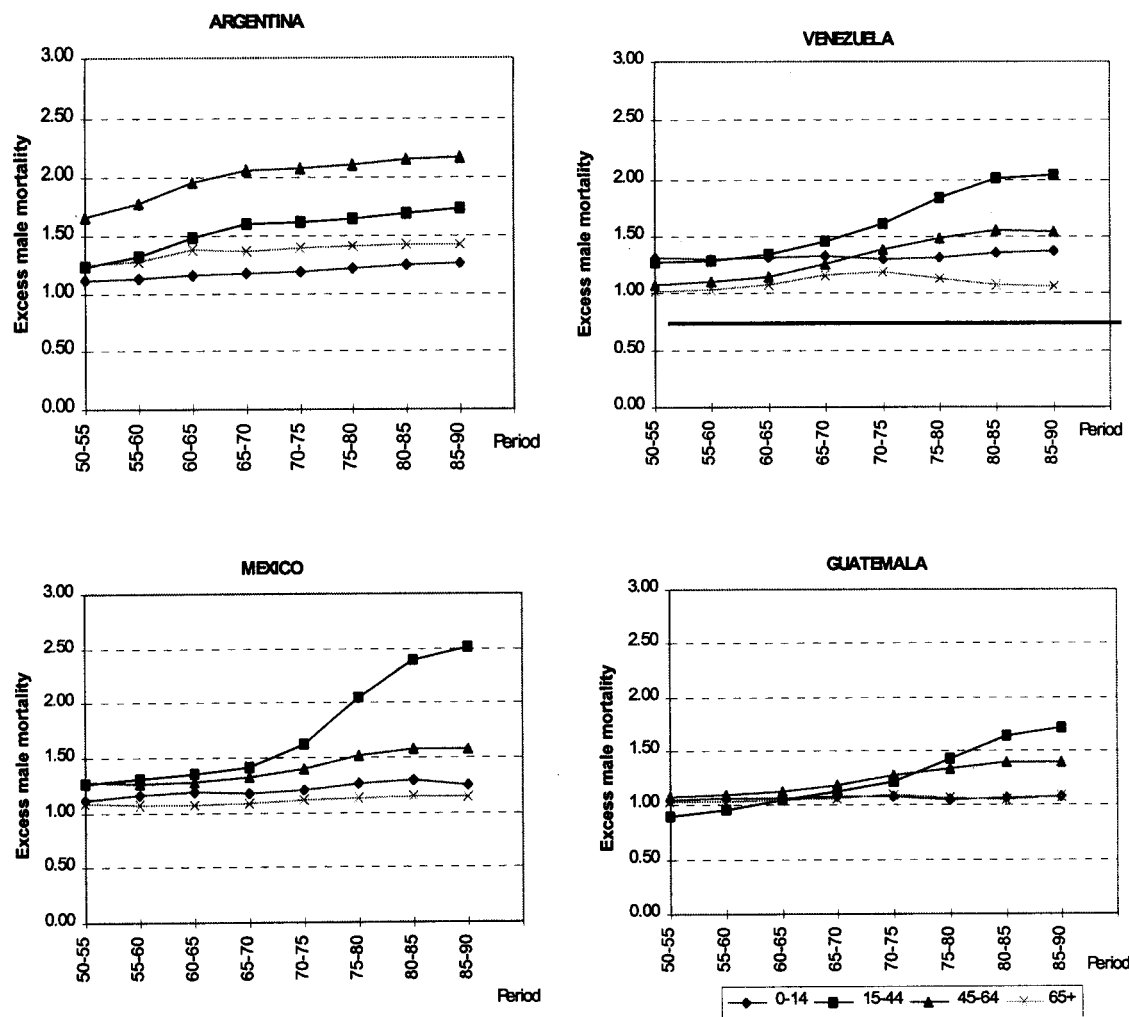


Figure 28 (continued)



Source: Latin American Demographic Centre (CELADE), "Latin America: Life table 1950-2050", *Demographic Bulletin*, No. 61 (Santiago, Chile, 1997, in press).

\*Ratio of male mortality rate to female mortality rate.

was a steady rise for both sexes, albeit at very different levels (table 16).

For women in this age group, another cause of death is complications in pregnancy, childbirth and the puerperium, which fall into the "all other causes" group. Apart from the data problems already discussed, it is difficult to obtain reliable estimates of the maternal mortality rate because of the large number of deaths that are associated with clandestine abortions.

It has been possible, nevertheless, to establish, on the basis of existing information, that maternal mortality rates in Latin America are many times higher than rates in the developed countries (Rajs, 1996). Even according to uncorrected vital statistics, maternal mortality rates are at present around 4.5 per 10,000 births in the low-mortality Latin American countries and around 10 per 10,000 in the high-mortality countries, whereas in Canada, for example, they are 0.4 per 10,000. While this cause of

TABLE 16. MORTALITY RATES BY SEX, AGE AND CAUSE<sup>a,b</sup> (PER 100,000), SELECTED COUNTRIES AND PERIODS

Age and cause	MALES															
	Costa Rica		Cuba		Chile		Uruguay		Argentina		Venezuela		Mexico		Guatemala	
	1960- 1965	1985- 1990	1965- 1970	1985- 1990	1960- 1965	1985- 1990	1960- 1965	1985- 1990	1960- 1965	1985- 1990	1970- 1975	1985- 1990	1960- 1965	1985- 1990	1960- 1965	1980- 1985
0-14																
Communicable	565.1	30.6	182.8	31.6	633.2	45.1	116.4	34.9	225.2	43.3	440.1	82.8	743.6	170.6	1 567.5	661.8
Neoplasms	10.5	5.4	8.7	5.5	8.8	7.0	11.3	8.5	11.6	7.8	11.2	7.7	4.7	7.1	3.9	6.0
Circulatory system	11.5	1.9	5.5	3.3	7.6	1.8	2.5	6.1	10.4	13.3	5.1	4.1	7.2	9.2	7.1	2.3
Originating in the																
Perinatal period	222.2	64.3	109.0	48.4	406.0	48.8	224.6	90.1	218.2	115.0	276.9	107.6	342.5	101.2	522.0	233.3
External	25.3	14.1	23.4	25.7	54.5	44.3	18.6	30.7	27.7	30.0	39.8	34.0	38.3	40.0	19.1	19.7
Other causes	137.8	52.3	181.6	41.7	84.0	44.0	70.4	65.7	120.1	63.5	117.3	54.3	118.4	92.1	181.2	137.2
15-44																
Communicable	37.0	4.3	15.4	6.0	123.6	17.7	27.9	8.8	37.9	12.8	64.4	17.9	128.9	24.9	395.9	140.0
Neoplasms	36.9	19.4	17.9	18.1	31.2	22.2	32.8	31.2	36.9	27.4	32.7	19.3	15.6	15.3	14.1	14.3
Circulatory system	38.0	16.7	24.3	26.4	40.0	17.5	39.4	29.6	47.5	49.6	60.9	30.8	48.5	22.3	28.8	28.2
External	110.4	86.7	80.2	109.5	194.3	136.6	87.1	82.4	100.3	88.0	186.6	166.7	196.1	207.6	133.3	361.8
Other causes	52.7	27.2	85.4	26.5	110.0	38.5	40.3	26.7	54.9	34.3	64.1	41.8	121.4	79.9	143.8	98.9
45-64																
Communicable	138.4	30.1	60.0	32.9	416.8	113.1	109.4	40.7	113.3	64.6	261.1	97.3	411.6	108.5	1 133.7	573.2
Neoplasms	351.8	234.4	226.9	231.3	334.4	275.2	459.3	481.0	536.7	364.1	349.7	225.1	132.6	141.2	125.3	127.0
Circulatory system	342.2	240.0	346.2	400.6	471.6	292.4	563.2	482.0	564.2	635.0	644.6	482.6	321.0	244.4	196.7	240.7
External	130.8	123.7	77.7	130.1	246.3	203.2	106.5	110.4	123.3	111.1	176.5	164.3	194.2	232.4	135.2	298.6
Other causes	271.7	184.6	280.0	135.4	519.4	332.0	263.5	239.3	356.0	257.4	313.9	284.2	553.9	519.0	547.2	492.6
65 y +																
Communicable	964.3	382.2	478.1	498.3	1 732.2	902.1	450.0	312.9	438.0	413.2	920.0	719.4	1 573.5	718.4	4 327.8	2 483.1
Neoplasms	1 730.8	1 684.5	1 300.8	1 400.8	1 512.5	1 507.9	1 968.9	1 953.1	2 192.8	1 492.0	1 389.7	1 217.4	512.0	824.1	530.1	714.1
Circulatory system	2 834.9	2 698.2	3 650.0	3 206.5	3 031.6	2 805.1	3 974.9	3 458.3	3 364.7	4 145.6	3 744.2	3 479.4	1 493.7	2 079.0	1 193.7	1 879.5
External	231.4	277.6	208.2	286.6	321.1	311.1	213.8	212.1	236.0	227.3	244.5	299.7	182.5	371.9	191.5	331.8
Other causes	1 613.8	1 447.3	1 610.9	857.7	1 439.8	1 509.9	1 202.7	1 463.5	1 972.7	1 345.2	1 378.5	1 481.4	3 310.5	2 439.6	1 995.5	2 000.8

TABLE 16 (continued)

Age and cause	FEMALES															
	Costa Rica		Cuba		Chile		Uruguay		Argentina		Venezuela		Mexico		Guatemala	
	1960- 1965	1985- 1990	1960- 1965	1985- 1990	1960- 1965	1985- 1990	1960- 1965	1985- 1990	1960- 1965	1985- 1990	1970- 1975	1985- 1990	1960- 1965	1985- 1990	1960- 1965	1980- 1985
0-14																
Communicable	524.3	25.6	152.2	25.1	577.7	39.0	109.6	33.3	213.8	37.1	423.2	72.1	667.1	150.3	1 546.3	660.4
Neoplasms	7.2	4.1	7.5	4.9	6.7	5.9	7.9	6.7	8.5	6.9	10.0	5.5	3.6	5.7	2.7	3.2
Circulatory system	10.5	2.2	7.0	3.2	5.9	1.8	2.5	4.2	10.0	11.4	4.4	3.5	6.4	7.7	5.9	2.5
Originating in the perinatal period	155.4	45.5	75.1	32.5	330.5	37.8	176.1	70.2	179.0	86.8	227.0	80.3	253.9	70.2	412.7	184.1
External	13.9	7.8	15.4	15.2	33.2	29.3	10.4	19.4	16.0	19.7	24.1	18.5	23.6	22.4	11.4	14.2
Other causes	126.7	44.7	150.2	34.6	70.6	40.4	61.4	58.2	103.5	55.3	110.0	47.1	104.6	81.2	177.2	137.5
15-44																
Communicable	35.9	3.7	13.2	4.7	93.9	10.3	19.7	6.0	35.0	10.2	59.4	12.4	120.3	19.9	419.9	156.8
Neoplasms	41.1	24.7	24.0	23.0	42.1	28.4	36.3	32.1	38.5	30.1	55.6	28.2	30.3	23.2	27.4	26.9
Circulatory system	36.6	11.5	23.1	20.7	34.5	11.4	25.0	18.1	28.1	30.2	43.3	21.5	54.4	19.2	31.3	37.7
External	14.4	15.2	37.8	52.0	32.3	21.8	20.2	21.3	21.4	21.1	32.2	24.9	23.1	28.8	19.1	54.6
Other causes	92.9	24.1	84.8	28.3	127.5	27.1	39.1	24.0	63.9	30.6	98.8	40.4	146.8	52.9	182.0	122.4
45-64																
Communicable	119.8	16.6	50.6	24.0	215.6	46.2	38.2	26.9	49.5	31.0	187.3	56.2	309.8	69.6	1 061.0	435.8
Neoplasms	367.5	210.4	223.3	216.7	377.0	265.2	312.7	324.9	340.2	230.0	473.8	240.3	229.3	195.9	205.1	213.9
Circulatory system	307.1	142.3	271.4	265.4	362.0	173.3	292.4	240.9	279.4	250.0	520.7	279.0	288.5	174.9	168.5	198.7
External	18.9	20.2	36.5	41.5	44.1	35.8	23.0	36.5	26.7	31.5	43.2	33.1	37.4	41.4	21.0	50.7
Other causes	243.4	137.3	214.7	126.4	317.0	165.3	140.0	135.0	191.2	120.2	259.9	167.5	399.1	319.7	454.8	369.5
65 y +																
Communicable	1 039.7	332.7	408.4	431.0	1 454.6	776.1	333.5	283.7	319.8	311.5	798.6	608.1	1 957.6	617.8	4 096.6	2 366.6
Neoplasms	1 416.6	1 211.4	789.9	901.1	1 367.2	1 187.6	1 345.9	1 132.1	1 517.2	958.2	1 303.9	962.7	775.4	700.0	707.6	836.9
Circulatory system	2 768.6	2 444.8	3 494.0	3 145.2	2 980.9	2 614.8	3 633.2	3 243.8	3 045.1	3 601.5	3 491.3	3 210.5	2 148.2	2 131.6	1 382.0	1 901.2
External	122.6	186.4	144.8	239.2	117.6	139.3	126.5	123.3	109.1	128.4	136.2	144.5	99.8	169.2	71.7	117.6
Other causes	1 509.0	1 431.5	796.1	904.8	965.3	1 117.1	835.4	975.1	1 583.5	982.9	1 134.5	1 288.9	1 735.4	2 162.0	1 647.2	1 831.7

Sources: Prepared on the basis of Pan American Health Organization (PAHO), *Health Statistics from the Americas*, Scientific publication No. 542 (Washington, D.C., PAHO, 1992); Latin American Demographic Centre (CELADE), "Latin America: Population projections 1950-2050", *Demographic Bulletin*, No. 59 (Santiago, Chile, 1997); "Latin America: Life table 1950-2050", *Demographic Bulletin*, No. 61 (Santiago, Chile, 1997, in press).

\*Causes included under "ill-defined causes" have been distributed proportionally among the remaining cause groups, with the exception of "external causes".

<sup>b</sup>Life table mortality rates for all deaths were simply disaggregated according to the relative share of deaths registered per cause.

death is not a decisive component of overall mortality and is one of those where the greatest reductions have been made, thanks to mother-and-child health care programmes, it is considered to be a major indicator of health status, since such deaths are perfectly avoidable if pregnancies develop under favourable conditions.

### 3. *Mortality among adults (45-64 years)*

Before looking at the remaining age groups, mention should be made of the widespread belief that mortality among old people in the Latin American countries, as calculated on the basis of death registration and population censuses, is underestimated. Such doubts spring from the fact that the uncorrected data yield levels of mortality that are low compared with those of developed countries. Indeed, a methodology has been devised to demonstrate the existence of errors in old people's age reporting which influences mortality estimates (Dechter and Preston, 1991; and Grushka, 1996). The first of these studies concludes that "pervasive misstatement of age among the elderly in Latin America is clearly demonstrated by these results, even though two of the countries, Costa Rica and Chile, are among the most literate and affluent of Latin American populations", and that "age overstatement is a significant problem in both death and population enumerations". Account is taken of these problems in the rates given here, which have been adjusted, depending on the consistency of the information, during preparation of population estimates and projections. Nevertheless, further investigation is still needed in order to establish whether mortality patterns among older adults reflect errors in the data or trends peculiar to the region, or a combination of both.

Guatemala's rates for the 45-64 age group are higher than those of the other countries, and mortality in Chile was also high during the period 1955-1960, especially among men (table 14). Trends in recent years show some convergence among the low-mortality countries, particularly in the female rates. Wider variations persist in the male rates, according to the most recent estimates, which show Costa Rica (81.3 per 10,000) and Cuba (93 per 10,000) with rates far below those of the rest of the region (120-172 per 10,000). These differences reflect the great unevenness of reduction in the different countries. For example, whereas reductions in the early-transition countries were moderate,

Cuba made significant improvements during the first period analysed, Costa Rica's rates dropped steadily during the entire thirty-year period, while Chile's reductions are more recent (table 15). Arriaga (1995) has obtained similar results in the analyses of temporary life expectancies for adults. This age group (45-64) also presents high, and increasing, excess male mortality (figure 28), coming second after the young adults group (15-44) with a male-female ratio of between 1.4 and 2.

In low- and moderate-mortality countries, the major causes of death in this age group are, for men, diseases of the circulatory system, followed by neoplasms, and for women—in some countries—the reverse (table 16). In Chile and Mexico in the early 1960s, deaths from infectious and parasitic diseases still tended to predominate somewhat, a tendency characteristic of high-mortality countries, and one that can be clearly seen today in Guatemala, which explains why current mortality rates from cardiovascular diseases and cancer are so much lower there than elsewhere in the region.

The rates that have fallen most are those of deaths from communicable diseases, which in Costa Rica, Chile and Mexico have declined by more than 70 per cent in men and 80 per cent in women. Cuba does not present large gains, since mortality rates from these causes there have been low for decades. Trends in the other two major cause groups for these ages, neoplasms and cardiovascular diseases, are more uneven. The biggest reductions in male rates range between 30 per cent and 40 per cent, and occur in both these cause groups in Costa Rica, for example, in cardiovascular diseases in Chile and in neoplasms in Argentina and Venezuela. The situation is similar for women, but with bigger decreases than for men, ranging between 40 per cent and 50 per cent. It is women's greater gains in mortality from conditions that are becoming less prevalent, such as communicable diseases, and from other causes that are responding to new medical advances, such as neoplasms and cardiovascular diseases, that explain the widening sex differential.

### 4. *Mortality among the elderly (65+ years)*

In the 65 and over age group, mortality rates differ little from country to country. In Guatemala they are somewhat higher than in the other countries, although this was not always

the case, as for example in 1985-1990, when male rates in Argentina overtook those of Guatemala (762.3 versus 748.2 per 10,000). Percentage gains are generally moderate, which is to be expected since, in Latin America, rates are low for advanced ages. Excess male mortality is also moderate. Mortality in this open-ended final age group is directly related to life expectancy at 65 years of age, an indicator of the greatest relevance in allocating resources to health and social security. In 1985-1990, life expectancy at 65 ( $e_{65}$ ) in the selected countries varied between 13.5 and 15.5 in men and between 14.2 and 17.8 in women (table 13). This indicator has shown significant growth since mid-century, especially among women, whose  $e_{65}$  in a number of countries of recent mortality transition has lengthened by between 3.5 and 5 years in three decades.

As regards male rates, it is interesting that Guatemala's  $e_{65}$  is higher than Argentina's and similar to Uruguay's (around 13.5), a situation that may be attributable to errors in the basic data or to corrections to the data. It may also indicate a special low mortality in Guatemala from a natural selection due to high mortality at younger ages.

Except in Guatemala, cardiovascular diseases are the prime cause of death, followed by neoplasms. The different cause groups show little variation over time, but the greatest decline is in mortality rates from communicable diseases. External causes, which present the widest sex differentials (higher for men), show increases in a number of countries. In Guatemala, a high-mortality country, the highest rates correspond to infectious and parasitic diseases, although these have declined substantially during the period whereas rates for the other causes have increased.

#### E. MORTALITY TRANSITION AND EPIDEMIOLOGICAL TRANSITION

##### 1. *Changes in the patterns of mortality by cause and age*

Changes in mortality rates by age and cause of death, and population ageing due to the demographic transition, lead to changes in epidemiological profiles. This process of epidemiological transition involves such a shift in the patterns of morbidity and cause of death, and the preponderance of communicable and

early childhood diseases gives way to chronic and degenerative diseases and external causes. There is also a fundamental change in the age structure of mortality, with the burden of death shifting into older age groups.

Table 17 shows the structure of mortality by sex and cause in the eight countries under analysis. The epidemiological transition has progressed furthest in Uruguay, where mortality rates from communicable diseases and conditions originating in the perinatal period have fallen and the population is ageing. Around 6 per cent of deaths in Uruguay are from diseases of this type, while nearly 70 per cent are due to cardiovascular diseases, cancer and external causes. A diametrically opposite pattern can be seen in Guatemala, where around 60 per cent of deaths are from communicable and early childhood diseases and only 13 per cent are due to chronic or degenerative diseases or injuries. In countries that recently experienced a mortality transition, the shift in the epidemiological structure has been dramatic: in Chile, for example, in the last thirty years, death rates from communicable diseases and conditions originating in the perinatal period have fallen from more than 60 per cent to less than 15 per cent, while the converse increase has occurred with deaths from chronic illness and violence.

In order to analyse the situation in the Latin American countries, an indicator known as the "mortality profile ratio" (MPR) (Frenk, Bobadilla and Lozano, 1996) has been used. The MPR is defined here as the ratio of deaths from communicable diseases and conditions originating in the perinatal period to deaths from neoplasms, cardiovascular diseases and external causes. In the 1960s, the MPR had already fallen below 1 in the early-transition countries and Cuba, showing that the latter cause group dominated, whereas in the other countries it varied between 1.5 (Venezuela) and 10 (Guatemala). Today the MPR is between 0.1 and 0.3 in the low-mortality countries and in the moderate-mortality countries. In Guatemala, where the situation is still extreme, the MPR is 3, i.e., deaths from communicable and early childhood diseases are three times as common as deaths from non-communicable diseases, a level that in Mexico was seen more than 30 years ago. The greatest advances in the epidemiological transition during the last twenty years have been made in Chile, followed by Costa Rica.

TABLE 17. RELATIVE DISTRIBUTION OF DEATHS BY SEX AND CAUSE, SELECTED COUNTRIES AND PERIODS

Country, period and sex		Total	Communicable diseases	Neoplasms	Circulatory system	Originating in the perinatal period	External causes	Other causes	MPR <sup>a</sup>
Costa Rica									
Male	1960-1965	100.0	38.5	10.3	13.4	13.0	7.6	17.2	1.6
	1995	100.0	6.9	19.2	28.4	3.7	16.3	25.4	0.2
Female	1960-1965	100.0	40.8	11.3	11.3	15.4	10.2	2.1	1.8
	1995	100.0	7.5	22.3	33.1	3.9	5.9	27.3	0.2
Cuba									
Male	1970-1975	100.0	14.8	13.1	29.0	6.1	8.1	29.0	0.4
	1995	100.0	7.0	20.2	41.4	0.8	13.9	16.6	0.1
Female	1970-1975	100.0	15.9	11.8	29.0	5.6	5.5	32.2	0.5
	1995	100.0	6.6	18.3	45.6	0.7	9.4	19.4	0.1
Chile									
Male	1960-1965	100.0	34.8	9.2	15.3	13.9	11.1	15.8	1.4
	1994	100.0	10.5	21.0	27.2	1.7	17.1	22.5	0.2
Female	1960-1965	100.0	35.6	12.7	19.1	13.2	3.6	15.8	1.4
	1994	100.0	12.1	25.1	32.3	1.7	5.4	23.4	0.2
Uruguay									
Male	1960-1965	100.0	9.3	22.8	38.0	6.3	7.4	16.2	0.2
	1990	100.0	4.3	27.0	37.8	1.8	8.3	20.8	0.1
Female	1960-1965	100.0	9.0	22.4	43.8	6.2	3.2	15.5	0.2
	1990	100.0	5.5	22.8	46.1	1.6	3.9	20.1	0.1
Argentina									
Male	1960-1965	100.0	12.6	22.0	28.5	6.6	8.5	21.8	0.3
	1993	100.0	6.6	19.1	42.8	3.0	9.2	19.3	0.1
Female	1960-1965	100.0	14.7	21.1	29.4	7.7	3.4	23.6	0.4
	1993	100.0	7.0	19.5	47.5	2.8	4.3	18.9	0.1
Venezuela									
Male	1960-1965	100.0	31.9	8.4	17.0	15.6	11.9	15.2	1.3
	1994	100.0	11.1	11.9	28.9	6.4	23.0	18.8	0.3
Female	1960-1965	100.0	34.3	12.2	17.5	14.7	3.8	79.2	1.5
	1994	100.0	12.5	16.9	35.9	6.5	6.2	22.0	0.3
Mexico									
Male	1960-1965	100.0	40.5	3.6	9.7	12.8	8.9	24.5	2.4
	1994	100.0	9.9	9.9	19.7	5.2	20.2	35.1	0.3
Female	1960-1965	100.0	45.6	6.4	13.3	11.8	2.6	20.3	2.6
	1994	100.0	10.9	14.2	27.0	4.7	5.8	37.3	0.3
Guatemala									
Male	1960-1965	100.0	62.8	1.8	3.4	14.2	4.3	13.5	8.1
	1980-1985	100.0	45.7	3.2	7.1	11.0	15.0	17.9	2.2
Female	1960-1965	100.0	66.2	2.9	3.8	12.0	1.0	14.2	10.2
	1980-1985	100.0	52.1	5.5	8.5	9.9	3.6	20.5	3.5

Sources: Prepared on the basis of Pan American Health Organization (PAHO), *Health Statistics from the Americas*, Scientific publication No. 542 (Washington, D.C., PAHO, 1992); Latin American Demographic Centre (CELADE), "Latin America: Population projections 1950-2050", *Demographic Bulletin*, No. 59 (Santiago, Chile, 1997); "Latin America: Life table 1950-2050", *Demographic Bulletin*, No. 61 (Santiago, Chile, 1997, in press).

<sup>a</sup>MPR, the mortality profile ratio, is the ratio of deaths from communicable diseases and conditions originating in the perinatal period to deaths from neoplasms, cardiovascular diseases and external causes.

This transition also involves a fundamental change in the age structure of deaths. Today, more than 60 per cent of deaths in the low-mortality countries take place in the 65 and over age group, whereas 35 years ago, in the recent-transition countries, the proportion was some 20 to 25 per cent. In Guatemala, at the other extreme, 25 per cent of deaths are now in the elderly age group, whereas in the 1950s the figure was below 10 per cent (table 18). This shift of the burden of death into the older age groups far outweighs the change in the age distribution of the population, showing that the epidemiological transition is best explained by the reduction of mortality from specific causes affecting mainly children and young people. The fact that the age structure of mortality

reflects a country's progress through the epidemiological transition means that, in countries where there is a lack of reliable information on causes of death, deaths by age can be used as an indicator of the stage they have reached. The last column of table 18 shows the ratio of deaths among children to those of elderly adults, analysis of which leads to the same conclusions as analysis based on the MPR (table 17).

Strictly speaking, study of the epidemiological transition as an indicator of a population's health status ought to be based on morbidity statistics showing the prevalence of the different diseases. However, given the great difficulty of obtaining reliable morbidity data, it has



TABLE 18. RELATIVE DISTRIBUTION OF DEATHS BY BROAD AGE GROUPS, SELECTED COUNTRIES AND PERIODS

Country and period	Age groups					0-14/65+
	Total	0-14	15-44	45-64	65+	
Costa Rica						
1950-1955	100.0	53.6	11.8	12.5	22.1	2.42
1995	100.0	10.5	14.5	18.8	56.2	0.19
Cuba						
1950-1955	100.0	33.1	15.9	18.0	33.0	1.00
1995	100.0	3.1	10.4	19.1	67.4	0.05
Chile						
1950-1955	100.0	42.3	15.7	19.0	22.9	1.84
1994	100.0	6.4	11.2	21.6	60.8	0.11
Uruguay						
1950-1955	100.0	13.6	9.1	22.7	54.6	0.25
1990	100.0	4.9	5.6	20.7	68.8	0.07
Argentina						
1950-1955	100.0	24.8	16.0	27.2	32.0	0.77
1993	100.0	7.6	8.0	21.2	63.2	0.12
Venezuela						
1950-1955	100.0	36.3	16.1	23.6	23.9	1.52
1994	100.0	18.8	19.8	20.1	41.3	0.45
Mexico						
1950-1955	100.0	53.3	13.9	13.9	19.0	2.81
1994	100.0	16.3	18.1	20.7	44.9	0.36
Guatemala						
1950-1955	100.0	63.7	16.6	10.6	9.2	6.91
1980-1985	100.0	50.5	17.8	14.1	17.7	2.86

Source: Prepared on the basis of Pan American Health Organization (PAHO), *Health Statistics from the Americas*, Scientific publication No. 542 (Washington, D.C., PAHO, 1992); Latin American Demographic Centre (CELADE), "Latin America: Population projections 1950-2050", *Demographic Bulletin*, No. 59 (Santiago, Chile, 1997); "Latin America: Life table 1950-2050", *Demographic Bulletin*, No. 61 (Santiago, Chile, 1997, in press).

often been necessary to look instead at the causes of death. Increases in survival are frequently achieved without a clear picture of individual health status, and in these cases mortality indicators are not enough to describe the health conditions of a population. Attempts have recently been made to create a synthetic indicator incorporating the disabilities that affect the population, mainly a result of morbidity. Two developments in this regard concern "the global burden of disease" (Murray and Lopez, 1996) and "health expectancies" (Robine and others, 1997). Both indicators require relatively specialized information that is not readily available in the region. However, the first has been applied in a number of countries of Latin America (Mexico, Chile and Uruguay), with a view to its use in health-sector resource allocation.

## 2. Overlap between stages of epidemiological transition

The overall pattern of epidemiological transition does not develop in linear fashion, but rather in a series of advances and setbacks, and

unexpected situations arise. In a number of countries, or regions within countries, certain communicable diseases have made a comeback, or new ones have emerged. Little statistical information exists on this point, but there has been a major resurgence of cholera, chiefly in Peru, and malaria and dengue have reappeared elsewhere. In addition, pollution, which affects a number of the region's largest cities, leads to an increase in respiratory diseases and others. In the south of the continent, at the time of writing, Argentina and Chile are fighting an outbreak of the "hanta" virus, which, in Chile, has claimed 12 victims in a very short space of time.

The numbers of deaths caused by such outbreaks are normally unlikely to make a significant difference in life expectancy at birth or in the overall pattern of causes of death. However, they do require ongoing epidemiological surveillance among the poor sectors of the population, at a high cost to the national health budget. During the cholera epidemic in Peru in 1991 and 1992, for example, 540,000 cases were reported, yet the fatality rate in 1991 was

1 per cent and in 1992, 0.3 per cent. This low rate was achieved thanks to early diagnosis of the disease, public information campaigns and the high concentration of cases in urban areas (PAHO, 1997a). One of the reasons for outbreaks of this kind is undoubtedly the continued existence of poor sectors of the population who live in inadequate sanitary conditions, but another important factor is viral resistance and adaptation to medicines, particularly antibiotics, abilities that are reinforced through abuse of such medication.

The most dramatic instance of a new disease emerging is that of HIV/AIDS, which in Latin America affects chiefly Haiti and Brazil. According to information from The Joint United Nations programme on HIV/AIDS (UNAIDS), 22.6 million persons worldwide were living with HIV/AIDS in December 1996. Of these, 21.8 million were adults, of whom 42 per cent were women. Latin America, with 8 per cent of the world population, accounted for 5.8 per cent of the cases, and 20 per cent of those were women (UNAIDS, 1997).

### *3. Social and economic factors relating to the health and mortality transition*

The epidemiological transition is one component of a broader concept known as the health transition. In addition to the health conditions of the population as expressed in mortality levels and epidemiological profiles, this concept includes society's changing response to those conditions.

Although it is no surprise to find a connection between health status and the steps taken to improve it, and levels of socio-economic development, our understanding of the mechanism underlying this relationship in Latin America is somewhat confused. Clearly, there is some correspondence between the stage countries have reached in the epidemiological transition and their level of development. According to Frenk, Bobadilla and Lozano (1996), the countries described in this study as low-mortality (advanced transition) countries belong to a group that some decades ago attained an advanced level of "economic modernization" some decades ago. The moderate-mortality countries such as Venezuela and Mexico belong to a group of rapidly modernizing countries; the high-mortality countries such as Guatemala are at a level of incipient modernization. One World Bank study finds

a negative correlation between economic development and average adult mortality, in cross-sectional analyses comparing countries and also in longitudinal analyses (Kjellstrom, Koplan and Rothenberg, 1992). The study finds a clear relationship in terms of the reduction of mortality from communicable diseases, but it is less clear for non-communicable diseases.

It is less easy to establish a correlation between economic development and mortality trends in a given country. Rosero-Bixby (1996) shows that in Costa Rica's case it is hard to find a connection between the pace of adult mortality decline and trends in socio-economic development and health interventions. He points out that the 1960s, a period of rapid economic growth and expanding health expenditure in Costa Rica, coincided with stalled adult mortality levels, while in the 1980s the converse happened, with economic stagnation and a decline in older adult mortality. He concludes that "The existence of cohort effects, long latency periods, and complex lagged reactions might be the cause for these puzzling temporal relationships".

No conclusive studies have been done on the effects on mortality trends of the crisis that hit the majority of the countries of the region during the 1980s. Palloni and Hill (1997), in a very detailed study using data from Latin American countries, can find no strong association between the short-term economic changes and variations in mortality. It is generally accepted that the crisis had little effect on the downward trend in child mortality. That trend was influenced rather by socio-economic factors such as primary health care, improved sanitary conditions, extended educational coverage and the urbanization process, which were the results of the rapid economic growth of earlier decades and specific social policies and programmes (Bravo, 1997). It could also be the case that, while the crisis had no appreciable effect on mortality, it did influence morbidity, although, as mentioned earlier, there is no reliable information available for systematic studies on this question.

### *F. MORTALITY DIFFERENTIALS ACCORDING TO SOCIO-ECONOMIC VARIABLES*

Mortality rates in the region vary widely from country to country, but there is also grave

concern over the disparities to be found within countries. Given the region's poverty levels—around 40 per cent of the population of Latin America have an income below that required for a life of dignity—the death rates of the poor and their health status are likely to show high levels of mortality and a lag in the epidemiological transition, with a high preponderance of communicable diseases and conditions originating in the perinatal period.

However, when an attempt is made to analyse mortality differentials by disaggregating data according to socio-economic variables, even greater problems of information availability and reliability are encountered, particularly as regards lower-income sub-populations. The problems associated with the use of vital statistics, discussed in section A of this paper, are compounded by the difficulty of achieving consistency between mortality-rate numerators and denominators. The disaggregation of births, deaths or population by socio-economic variables such as level of education, or mother's level of education raise problems of comparability. Moreover, the demographic and the socio-economic variables are produced by different sources and are likely to be influenced by different reporting errors.

As in the case of mortality estimates at the national level, attempts have been made to deal with these problems by using indirect techniques at the sub-population level. Such techniques have been applied with some success in studies of infant and child mortality, and it has been possible to establish orders of magnitude for the differentials and, using explanatory models, to improve understanding of the determining factors. The chief sources used in such studies are population censuses based on questions on children ever born and children surviving, and WFS and DHS surveys based on birth histories, all of which have the advantage that both numerator and denominator belong to the same data-collection operation. The best-known studies in the region include the CELADE programme, "Research on Infant Mortality in Latin America" (IMIAL) (Behm and Primante, 1978); the case studies published by the United Nations Population Division (United Nations, 1991), three of which relate to countries of the region; and, more recently, a database of the various sources that exist (CELADE and UNICEF, 1995). These studies, and others based on multivariate analysis (Trussell and Preston, 1981; Chackiel, 1982),

find great importance in the discriminating power of the mother's level of education, which reflects both social differences and differences in the degree of access to information.

There are few studies of adult mortality differentials, and even fewer that examine the situation in the different countries of the region. As mentioned earlier, the procedures based on the survival of relatives have not yielded reliable results in estimates of adult mortality. This unreliability is compounded in the current context by the fact that, while the socio-economic situation of the informant may be known, that of the deceased or surviving relative may not be; in addition, greater problems of misreporting and bias are encountered. As a result, only isolated efforts have been made to know disparities in adult mortality, and the majority of these have focused on differentials according to geographical area or area of residence.

Given the trends discussed in the preceding sections of this paper, in addition to higher mortality rates among the more deprived sectors of society, one would also expect to find that differentials were now wider among adults than among children. If that were indeed the case, it would be due to the success of the fight against infectious and parasitic diseases, which affect mainly children, through the extension of primary health care coverage to the majority of the population. The chances of avoiding death in adulthood, on the other hand, depend on the population's access to more expensive medication and longer treatments, and on changes in lifestyle.

A recent study has attempted to estimate mortality differentials for adult women by ethnic group in Bolivia and Guatemala (Robles, 1996). The indigenous and non-indigenous populations were distinguished by language in Bolivia, and by self-identification in Guatemala. Adult mortality was estimated on the basis of questions on maternal orphanhood in censuses and surveys, and child mortality estimates were included in order to analyse the differential patterns in mortality by age. Table 19 shows selected results for Guatemala, since, unlike the results for Bolivia, they appear to be consistent. Life expectancy at 20 was taken as the indicator of adult mortality, and the infant mortality rate was used as the indicator of child mortality, together with, for each of these indicators, the corresponding level in the Coale-

TABLE 19. MORTALITY DIFFERENTIALS FOR THE FEMALE ADULT POPULATION,  
GUATEMALA AND ARGENTINA, SELECTED DATES

Source and indicators	Guatemala			
	Mortality		Coale-Demeny West level	
	non-indigenous	indigenous	non-indigenous	indigenous
1973 census				
Life expectancy at age 20	46.8	42.5	15.7	12.3
Infant mortality rate	0.113	0.156	14.2	11.2
1981(a) census				
Life expectancy at age 20	47.6	43.3	16.4	12.8
Infant mortality rate	0.089	0.108	16.1	14.6
1987 DHS				
Life expectancy at age 20	49.4	46.1	17.9	15.2
Infant mortality rate	0.076	0.085	17.2	16.5
<i>Argentina 1988-1992</i>				
Model 3	<i>Social security contributors, by fund</i>			
	<i>State</i>	<i>Industry</i>	<i>Independent</i>	
Life expectancy at age 20	50.8	44.9	56.3	
at age 65	13.4	11.3	15.2	
Model 4	<i>Social security contributors, by income</i>			
	<i>Low</i>	<i>Middle</i>	<i>High</i>	
Life expectancy at age 20	51.2	53.9	62.8	
at age 65	15.5	16.0	19.9	

Sources: A. Robles, "Mortalidad adulta entre poblaciones indígenas de Guatemala y Bolivia", Notas de Población No. 64 (Santiago, Chile, CELADE, 1996); R. Rofman, "Diferenciales de mortalidad adulta en la Argentina", Notas de Población No. 59 (Santiago, Chile, CELADE, 1994).

Demeny West model life tables. The study confirms that mortality among indigenous populations is higher for all ages and that the greatest decline is in child mortality. Differentials between ethnic groups are therefore narrowing in terms of infant mortality, but stable for adult mortality. As a result, different patterns of mortality by age are observable among indigenous and non-indigenous populations, reflecting the different frequency of occurrence of causes of death in each age group.

Robles found that the differences between ethnic groups were relatively moderate (around 5 years for  $e_{20}$ ). However, according to an Argentine study, based on non-traditional data from the National Social Security System and taking into account area of residence, economic activity and income, the widest differentials for the same adult mortality indicator were 11 years in a univariate analysis and 24 years in a multivariate analysis covering sectors in extreme situations (table 19) (Rofman, 1994).

It is a more complex matter to study the causes of death that could explain differences

in mortality levels and patterns by sex and age in relation to socio-economic groups. It is only possible—although still by no means straightforward—in countries with very high-quality data, which, as shown earlier, are likely to have the lowest levels of mortality and social inequity. Taucher, Albala and Icaza (1996), in a study of adult mortality from chronic diseases in Chile, present differentials based on geographical region, urban or rural area and educational level, using vital statistics records. The authors find, *inter alia*, that mortality is higher in rural than in urban areas up to 54 years of age, when the situation reverses itself. This may be due to errors in the data, possibly connected with more frequent age misreporting among the elderly in rural areas, or it may be the actual situation, similar to the one found in Guatemala and possibly connected with natural selection. In terms of cause of death, the greatest difference between areas of residence is for malignant neoplasms of the respiratory organs, cirrhosis of the liver and other diseases that are more frequent in urban areas and are related to smoking habits, alcoholism, obesity, stress, sedentary habits and air pollution. On the other hand, mortality from cancer of the oesophagus

and stomach is higher in rural areas, possibly because of dietary habits.

As the authors point out, it is difficult to calculate mortality rates by educational level since the population is classified by this variable only at the time of the census, i.e., every 10 years in Chile. This information also appears on death certificates, however, and, in order to make some use of it, the authors analysed the proportion of deaths of persons with a low educational level (i.e., who have at most primary education) from each cause, by age group (between 25 and 64). Cirrhosis of the liver figures more prominently among the less well educated, probably owing to a greater prevalence of alcoholism. Causes attributable to the intake of low-fiber foods and a high proportion of saturated fats, such as cancer of the colon and breast cancer, chiefly affect the better educated, as does cancer of the respiratory apparatus due to smoking.

Clearly it is extremely difficult, given the information available, to reach any real understanding of mortality and its causes in precisely those sectors of the population where special policies to improve health conditions are most likely to be needed. Studies such as those cited here would be very useful in this respect, but they can be carried out only occasionally, and yield only limited results that are difficult to generalize.

#### F. CONCLUDING REMARKS

This analysis provides an overview of mortality in the region. It may give the impression that the majority of the Latin American countries have greatly reduced their mortality rates and are at a very advanced stage in the health transition. This is the danger of working with limited data and, paradoxically, it is in the countries that most need reliable information in order to implement their social programmes where it is lacking. There is no doubt that in a number of countries of the region mortality levels are very similar to those of the developed countries and the epidemiological profile is fairly advanced. This is true of Costa Rica, Cuba, Chile, Uruguay and Argentina, and, to a lesser extent, Mexico and Venezuela. Yet the situation found in Guatemala, a high-mortality country where a large percentage of the population dies in childhood, also prevails in many other countries of the region. Moreover, a number of countries have not been considered

in this study owing to data quality problems. Brazil, for example, with more than one third of the region's population, has large groups living in poverty, whose health and mortality conditions should not be ignored simply because they are not well understood.

The health, mortality and epidemiological transition can be divided into two main stages. In the first, mortality is high, infectious and parasitic diseases are very prevalent and there are problems related to nutrition and reproductive health, a situation closely associated with the level of poverty in the population. The second stage corresponds to low-mortality countries, where the predominant causes of death relate more to modern life: tobacco consumption, motor vehicle accidents, environmental pollution and a sedentary lifestyle. Risk factors such as these, which are found in modern societies, also mainly affect the poor, who lack access to adequate health care. At one extreme, then, the high-mortality countries still need to overcome problems relating to living conditions, while, at the other, a growing number of countries have to implement preventive measures appropriate to the second stage of transition, which will entail profound changes in lifestyle.

Nevertheless, all these risk factors, from both stages of the transition, play an important role in the majority of the countries of Latin America. It is essential to persevere with primary care and mother-and-child health programmes and to keep up epidemiological surveillance against the ever-present risk of outbreaks of communicable diseases. However, the health sector should also address itself to the risk factors arising from economic growth, through policies that aim to alter lifestyles by promoting a high-fiber, low saturated fat diet, appropriate exercise at all ages, to reduce tobacco and alcohol abuse, and also through preventive measures to reduce traffic and household accidents and combat environmental pollution. It is essential, and still possible, for the Latin American countries, whose populations are as yet relatively young, and where development-related "epidemics" have not yet taken hold, to keep one step ahead and thus to avoid the costly new burdens that are looming. Generally speaking, the spread of communicable diseases has been successfully controlled using preventive measures; something along this line may also possibly be done for non-communicable diseases, at least

through preventive measures against certain types of cancer, cardiovascular diseases and accidents.

#### NOTES

<sup>1</sup>The age groups selected for this analysis were 0-14, 15-44, 45-64 and 65 and over. These groups exhibit differences in mortality levels and in patterns of causes of death, and were considered adequate for the purposes of this study. More detailed groupings would hinder analysis and—especially in the case of the older age groups—would render the data less reliable.

<sup>2</sup>This study uses the list of 6/61 mortality cause groups as defined by PAHO on the basis of the 9th edition of the International Classification of Diseases. The list contains six major groups: "communicable diseases", "neoplasms", "diseases of the circulatory system", "certain conditions originating in the perinatal period", "external causes of injury and poisoning" and "all other diseases". The last, residual group represents a limitation for the analysis, as, generally speaking, it is quite large and includes high-frequency diseases affecting mainly adults, such as diabetes, certain respiratory conditions, and, in some countries, cirrhosis. In order to establish mortality trends by cause group in each age group, the life table mortality rates were simply disaggregated according to the relative share of deaths registered per cause.

#### REFERENCES

- Arriaga, Eduardo E. (1995). La mortalidad adulta en países en desarrollo. Una visión general. Notas de Población No. 61. Santiago, Chile: Latin American Demographic Centre (CELADE), pp.79-110.
- \_\_\_\_\_. (1997). Causes of death in developing countries and in countries with economies in transition: An overview of current knowledge, data availability and novel methods of data collection (chapter IV in this volume).
- Behm, Hugo, and Domingo Primante (1978). Mortalidad en los primeros años de vida en la América Latina. Notas de Población No. 16. Santiago, Chile: Latin American Demographic Centre (CELADE), pp. 23-44.
- Brass, William (1975). *Methods for estimating fertility and mortality from limited and defective data*. Chapel Hill, N.C.: University of North Carolina, Laboratories for Population Studies.
- Bravo, Jorge (1997). Demographic consequences of economic adjustment in Chile. In *Demographic Responses to Economic Adjustment in Latin America*. George Tapinos, Andrew Mason and Jorge Bravo, eds. Clarendon Press Oxford: International Union for the Scientific Study of Population, pp. 156-173.
- CELADE (1997). Latin America: Population projections 1950-2050. *Demographic Bulletin No. 59*, Santiago, Chile: Latin American Demographic Centre.
- \_\_\_\_\_. (1997a). Latin America: Life Tables 1950-2050. *Demographic Bulletin No. 61* (in press). Santiago, Chile: Latin American Demographic Centre.
- \_\_\_\_\_, and UNICEF (1995). América Latina: Mortalidad en la niñez. Una base de datos actualizada en 1995. LC/DEM/G. 157. Santiago, Chile: Latin American Demographic Centre and United Nations Children's Fund.
- Chackiel, Juan (1982). Factores que afectan a la mortalidad en la niñez. Notas de Población No. 2. Santiago, Chile: Latin American Demographic Centre (CELADE), pp. 66-120.
- \_\_\_\_\_, and Hernán Orellana (1985). Adult female mortality trends from retrospective questions about maternal orphanhood included in censuses and surveys. In *International Population Conference. Florence 1985*. Liège, Belgium: International Union for the Scientific Study of Population (IUSSP).
- Dechter, Aimée R., and Samuel H. Preston (1991). Age misreporting and its effects on adult mortality estimates in Latin America. *Population Bulletin of the United Nations* (New York), No. 31/32, pp. 1-16.
- Frenk, Julio, José L. Bobadilla and Rafael Lozano (1996). The epidemiological transition in Latin America. In *Adult Mortality in Latin America*, Ian M. Timaeus, Juan Chackiel, Lado Ruzicka, eds. Clarendon Press Oxford: International Union for the Scientific Study of Population, pp. 123-139.
- Grushka, Carlos O. (1996). Adult and old age mortality in Latin America: evaluation, adjustments and a debate over a distinct pattern. Philadelphia: University of Pennsylvania Press.
- Hill, Kenneth (1984). An evaluation of indirect methods for estimating mortality. In *Methodologies for the Collection and Analysis of Mortality Data*, Jacques Vallin, John H. Pollard and Larry Heligman, eds. Liège, Belgium: International Union for the Scientific Study of Population, pp. 145-177.
- Jaspers Faijer, Dirk, and Hernán Orellana (1996). Evaluation of vital statistics for the study of causes of death. In *Adult Mortality in Latin America*, Ian M. Timaeus, Juan Chackiel, Lado Ruzicka, eds. Clarendon Press Oxford: International Union for the Scientific Study of Population, pp. 45-68.
- Kjellstrom, Tord, Jeffrey P. Koplan and Richard B. Rothenberg (1992). Current and future determinants of adult ill-health. In *The Health of Adults in the Developing World*, Richard G. A. Feachem, Tord Kjellstrom, Christopher J. L. Murray, Mead Over and Margaret A. Phillips, eds. New York: World Bank, pp. 209-259.
- Murray, Christopher J. L., and Alan D. Lopez (1996). *The global burden of disease*. Harvard School of Public Health: World Health Organization/World Bank.
- Palloni, Alberto, and Kenneth Hill (1997). The effects of economic changes on mortality by age and cause: Latin America, 1950-1990. In *Demographic Responses to Economic Adjustment in Latin America*. George Tapinos, Andrew Mason and Jorge Bravo, eds. Ian M. Timaeus, Juan Chackiel, Lado Ruzicka, eds. Clarendon Press Oxford: International Union for the Scientific Study of Population (IUSSP), pp.75-128.
- PAHO (1992). *Health Statistics from the Americas*. Scientific publication No. 542. Washington, D.C.: Pan American Health Organization
- \_\_\_\_\_. (1995). *Health Statistics from the Americas*. Scientific publication No. 556. Washington, D.C.: Pan American Health Organization
- \_\_\_\_\_. (1997). *Health Statistics from the Americas*. Scientific publication, in press. Washington, D.C.: Pan American Health Organization
- \_\_\_\_\_. (1997a). Pan American Health Organization, Country Profiles. <http://www.paho.org>. Accessed 10 September, 1997.
- Rajs, Danuta (1996). Maternal mortality. In *Adult Mortality in Latin America*, Ian M. Timaeus, Juan Chackiel, Lado Ruzicka, eds. Clarendon Press Oxford: International Union for the Scientific Study of Population (IUSSP), pp.276-294.
- Robine, Jean M., Isabelle Romieu and Emmanuelle Cambois (1997). La estimación de los años vividos con discapacidad: una iniciativa universal. Notas de Población No. 64. Santiago, Chile: Latin American Demographic Centre (CELADE), pp. 7-32.
- Robles, Arodys (1996). Mortalidad adulta entre poblaciones indígenas y no indígenas de Guatemala y Bolivia. Notas de Población No. 64. Santiago, Chile: Latin American Demographic Centre (CELADE), pp. 33-61.
- Rofman, Rafael (1994). Diferenciales de mortalidad adulta en la Argentina. Notas de Población No. 59. Santiago, Chile: Latin American Demographic Centre (CELADE), pp. 73-91.
- Rosero-Bixby, Luis (1996). Adult mortality decline in Costa Rica. In *Adult Mortality in Latin America*, Ian M. Timaeus, Juan Chackiel, Lado Ruzicka, eds. Clarendon Press Ox-

- ford: International Union for the Scientific Study of Population, pp. 166-195.
- Taucher, Erica, Cecilia Albala and Gloria Icaza (1996). Adult mortality from chronic diseases in Chile, 1968-1990. In *Adult Mortality in Latin America*, Ian M. Timaeus, Juan Chackiel, Lado Ruzicka, eds. Clarendon Press Oxford: International Union for the Scientific Study of Population (IUSSP), pp. 253-275.
- Timaeus, Ian M. (1996). New estimates of the decline in adult mortality since 1950. In *Adult Mortality in Latin America*, Ian M. Timaeus, Juan Chackiel, Lado Ruzicka, eds. Clarendon Press Oxford: International Union for the Scientific Study of Population (IUSSP), pp. 87-107.
- Trussell, James, and Samuel Preston (1981). Estimating covariates of childhood mortality from retrospective reports of mothers. Presented at the Annual Meeting of the Population Association of America, Washington, D.C., March 1981.
- UNAIDS (1997). HIV/AIDS: The global epidemic (December 1996). <http://www.UNAIDS.org>.
- United Nations (1983). *Manual X. Indirect techniques for demographic estimation*. United Nations: New York, Sales No. E.83.XIII.2.
- \_\_\_\_\_. (1991). *Child mortality in developing countries*. United Nations: New York, Sales No. E.91.XIII.13.
- \_\_\_\_\_. (1995). *Population and development. Programme of action adopted at the International Conference on Population and Development, Cairo, 5-13 September 1994*. United Nations: New York, Sales No. E.95.XIII.7.
- \_\_\_\_\_. (1996). *World population prospects: The 1996 revision. Annex II & III: Demographic indicators by major area, region and country*. United Nations: New York (in press).
- Vallin, Jacques (1996). Causes of death in low-mortality developing and developed countries. In *Adult Mortality in Latin America*, Ian M. Timaeus, Juan Chackiel, Lado Ruzicka, eds. Clarendon Press Oxford: International Union for the Scientific Study of Population (IUSSP), pp. 140-165.

## VII. THE EPIDEMIOLOGICAL TRANSITION IN MEXICO: WHAT THE DATA ON CAUSE OF DEATH REVEAL

*Rosario Cárdenas\**

### A. INTRODUCTION

The epidemiological transition has been described as a framework for analysing the shifts in disease patterns that take place in response to demographic, economic and social changes (Omran, 1971). In his original paper, Omran identifies three stages of the transition: the age of pestilence and famine, characterised by high and fluctuating mortality, and low levels of life expectancy; the age of receding pandemics, during which mortality started to decline and life expectancy to improve; and the age of degenerative and man-made diseases, in which further reductions in mortality are observed, eventually reaching a stable relatively low level. More recently, Olshansky and Ault (1986) have proposed a fourth stage, the era of delayed chronic and degenerative diseases, designed to account for the lengthening of the life span and its associated medical conditions.

Three distinct time paths that societies have followed in the course of their epidemiologic transition were also identified by Omran in his initial description (1971). They were the classical or Western model, experienced by the European countries and the United States; the accelerated model, as in the Japanese case; and the contemporary or delayed model, typical of the transition in most developing countries.

In the case of the classical or Western model, the social and economic changes that improved living conditions, particularly during the nineteenth century, are held responsi-

ble for much of the decline. Similar factors are identified for the accelerated model, except that the transition took place in a shorter period of time. The main difference between the classical and accelerated models and the contemporary model is the leading role played in the latter by public health measures.

Frenk and others (1989) have proposed another epidemiological transition model to better describe the experience of middle income countries.<sup>1</sup> According to the authors, the transition in this group of countries is characterised by: a) overlap of eras as a result of the reduction of infectious diseases but not their eradication or full control; b) a counter-transition, as previously controlled diseases re-emerge; c) a protracted transition, with the prolonged co-existence of pathologies typical of two different eras (infectious and chronic); and d) a pattern of epidemiological polarisation, with marked health differences between social groups or regions within a population.

With respect to epidemiological polarisation, indicators of increasing income inequalities, combined with the impact of the economic recession, has given rise to concerns about possible widening of mortality differentials. Studies showing a deceleration of the pace of mortality decline in developing countries during the 70s and 80s (Adlakha and Suchindran, 1985; Arriaga, 1981; Hill, 1985; Kunitz, 1990; Lee, 1985; Meegama, 1981; Palloni 1981a, 1981b, 1989; Ruzicka and Hansluwka, 1982; Sivamurthy, 1981) have suggested that the deterioration of living standards is at the base of it. For example, according to Palloni (1981a) this slowdown was the result of the shortage of the social and economic transformations needed to continue the mortality reduction momentum achieved after World War II; for Arriaga (1981) the

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slowdown was a consequence of the fact that not all population subgroups had achieved full access to health and other services. Similar reasons were suggested in the case of South and East Asian countries where mortality stagnation was seen as reflecting within-country widening differentials, and the pace of mortality decline as an indicator of the slowdown in economic growth (Ruzicka and Hansluwka 1982).

In the case of Mexico, a middle income country, there are reasons to believe that health conditions have recently deteriorated. In 1982 Mexico entered a period of economic recession, characterised by several currency devaluations, the most recent in 1994. From the onset of the economic downfall, people's purchasing power and other economic indicators have never recovered. For example, between 1982 and 1990 the official minimum wage declined 51 per cent (Comisión Nacional de Salarios Mínimos, n.d.). The potential impact of this reduction can be judged from the fact that in 1990 26.5 per cent of the economically active population in the country earned less than the minimum wage (INEGI, 1992).

By analysing cause of death data, this paper aims at identifying: a) the main characteristics of the epidemiological profile of Mexico; b) causes for changes in life expectancy; and c) the main causes of premature mortality.

## B. MATERIALS AND METHODS

Two sources of data are used. Population estimates are based on censuses and mortality data come from the Vital Registration System.<sup>2</sup> Information analysed covers the period between 1979 and 1996.

During the last few years the quality of Mexico's mortality data has greatly improved. Registration coverage as well as the proportion of deaths medically certified have increased. More than 95 per cent of deaths currently are medically certified, and the pro-

portion of deaths coded as ill defined has steadily declined. Training of administrative personnel responsible for death registration, together with collaboration between various concerned branches of the government have played a major role in the improvement of mortality data. The fact that 1996 mortality data files were made available to the public in October of 1997 is evidence of the advances achieved in the organisation, processing and compilation of health information in the country. Nevertheless, some problems affecting specific population subgroups have been detected. For example, an inspection of mortality information pertaining to areas considered predominantly inhabited by indigenous groups shows age reporting errors. In these regions, an exaggeration of the age at death seems to be the main problem. Information on cause of death may also be subject to errors in these areas, given the cultural and sometimes also language differences between registrar officials and the population.

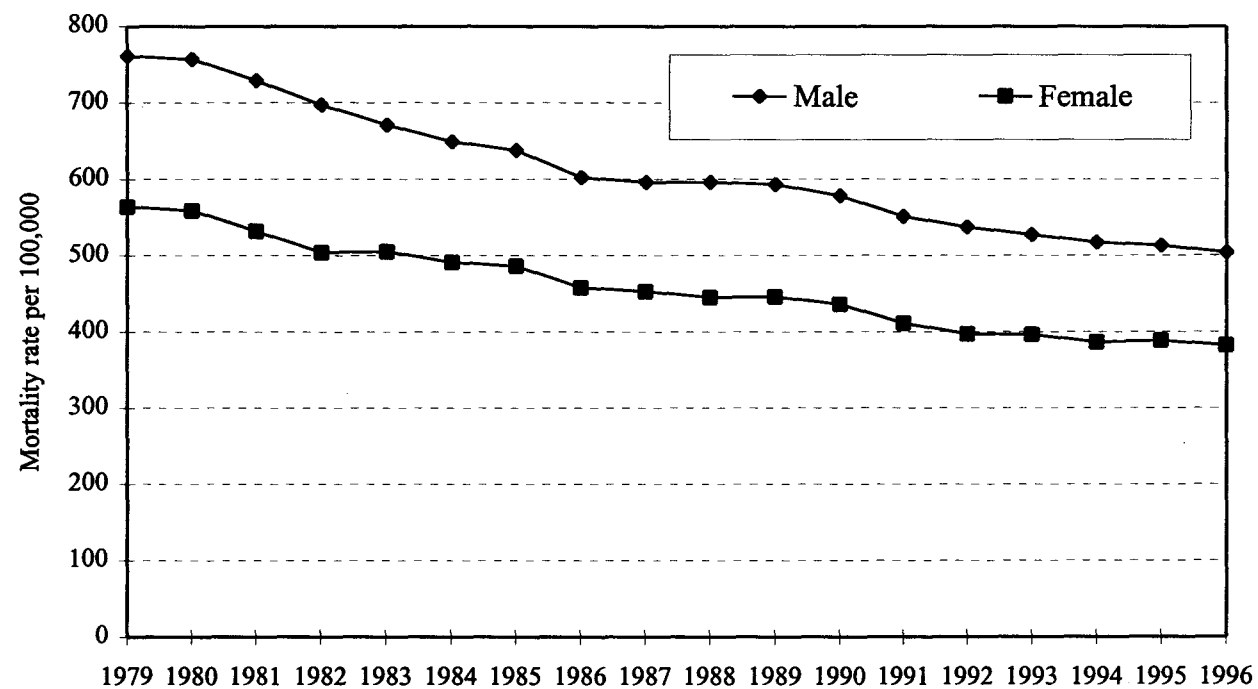
In the discussion that follows, standardised mortality rates by age, sex, cause and state, are used to study mortality trends, as well as to depict changes in the epidemiological profile. The technique developed by Arriaga (1984) for decomposing changes in life expectancy is used to assess the impact of shifts in cause of death. Premature mortality is measured in terms of years of life lost (Arriaga, 1994).

Causes of death have been grouped following the classification proposed by Murray and others (1992). The three groups of causes originally proposed are used for most of the analysis: a) communicable, maternal and perinatal causes<sup>3</sup>; b) non-communicable causes; and c) injuries. Information on specific causes is also presented, to highlight major current health problems.

## C. MORTALITY DIFFERENTIALS

Between 1979 and 1996 mortality declined steadily in Mexico (figure 29). Standardised

Figure 29. Standardized mortality rates by sex, Mexico, 1979-1996



Source: Author's estimates based on national vital statistics.

mortality rates changed from 761 per 100,000 inhabitants in 1979 to 504 in the case of males, and from 564 to 382 for females. As expected, male mortality levels were higher. However, the differential between sexes tended to diminish.

Mortality levels for each group of causes declined during the period analysed (figure 30). The reductions in mortality due to communicable causes in both sexes, together with the decline in injuries among men were the outstanding features. In 1979 non-communicable causes already were predominant; in 1996 its relative presence is even larger (figure 30). Among men, injuries explain a larger proportion of mortality than communicable causes, a pattern not observed for women. During this period, sex differentials in every group of causes diminished.

Cause-of-death by age follows the expected pattern. Most deaths of children under one year of age are caused by infectious and parasitic diseases (figure 31). As age advances, these conditions are replaced by non-communicable causes. Between 1979 and 1996 the relative contribution of non-communicable diseases increased for both sexes. However, for male teenagers and adults between 15 and 45 years of age, injuries are responsible for a large proportion of the mortality. This is particularly the case among men 15 through 29 years of age. Nevertheless, during this period the relative contribution of injury-related mortality declined for most ages except for children under 5 years (both sexes) and women 15-29 years of age.

The analysis of state level mortality data shows a reduction in regional differentials. The comparison of 1979 with 1996 rates shows that, independent of cause and sex, the largest reductions in mortality took place in states where the highest figures were registered at the beginning of the period (figures 32-34). Regarding mortality by cause, rates due to communicable, maternal and perinatal causes declined the most.

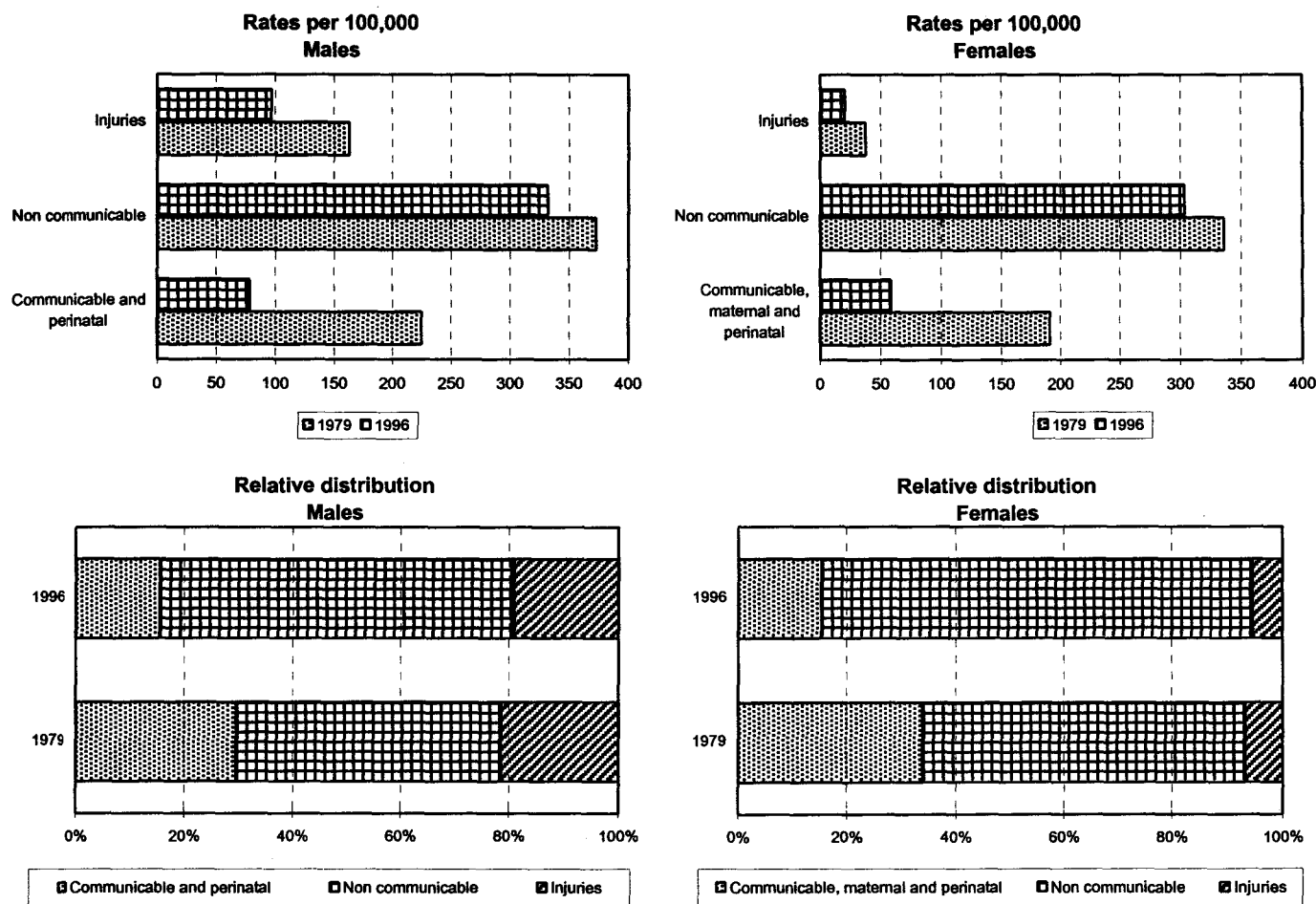
#### D. MORTALITY CHANGES

Gains in life expectancy between 1979 and 1996 tended to be consistently larger among males, except for the age groups 75 and older (figure 35). Decomposition of changes in life expectancy by groups of causes highlights the important role of reductions in injury-related mortality (figure 36). Most of life expectancy gains among males between 15 and 45 years of age are explained by reductions in mortality due to injuries. In contrast, gains among women are due to non-communicable mortality decline.

The analysis of specific communicable conditions shows that most of the gains in life expectancy associated with declines in non-communicable diseases are due to reductions in intestinal infections and parasitic mortality (figure 37). It is surprising that, even among women of reproductive age, these conditions explained more of the improvement than maternity related causes. It is worth noting that not only were reductions in infectious and parasitic diseases greater among male children under 1 year of age, but also those due to declines in perinatal conditions. Among children under one year of age, gains tended to be higher for males than for females, while the opposite holds among the 1-4 age group. Infectious and parasitic diseases decline faster among women in all other age groups. As expected, most gains associated with respiratory infections are observed among groups at extreme ages (under 1 year and 60 or over).

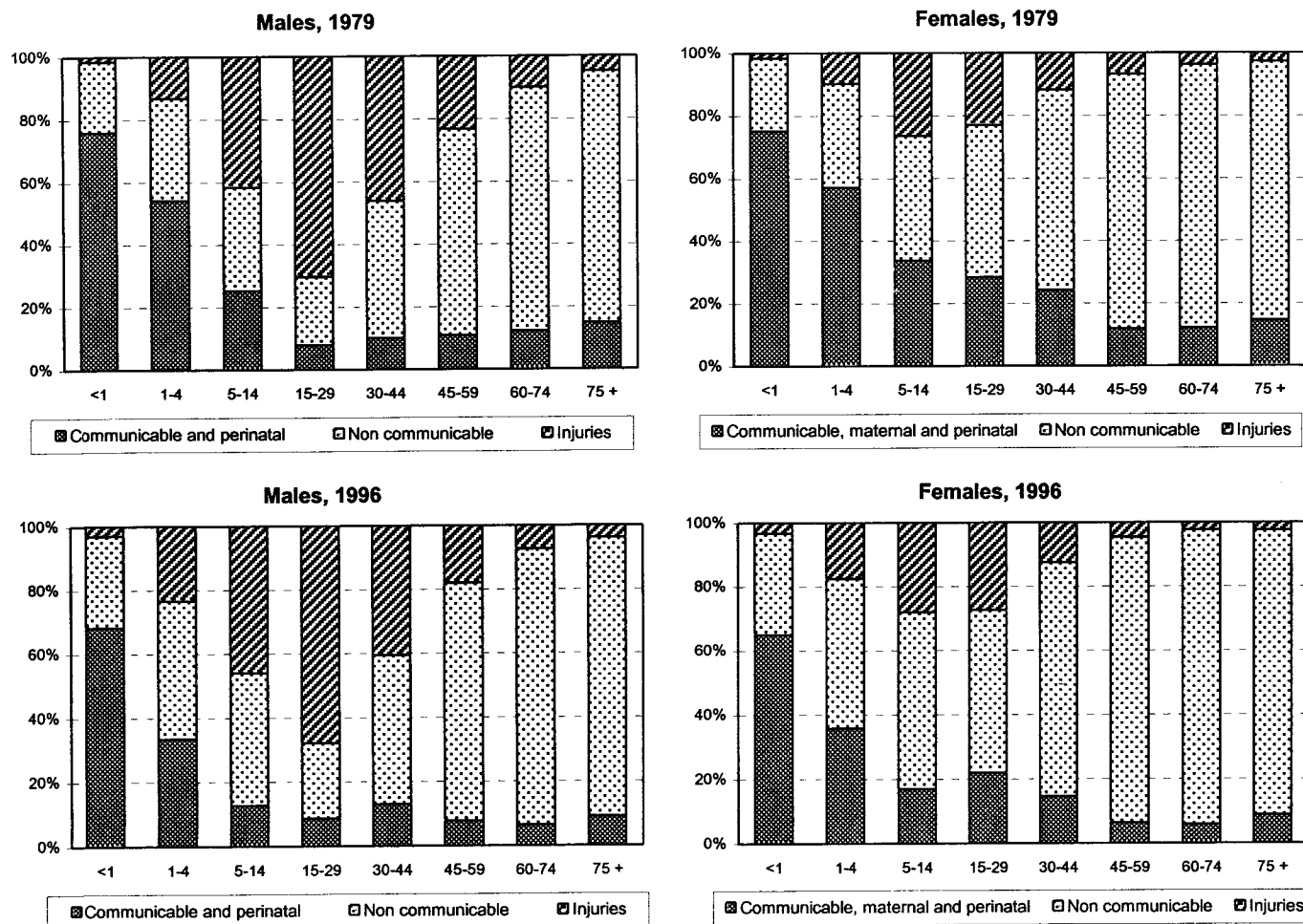
Regarding non-communicable causes, the reduction in respiratory and digestive pathologies was responsible for the largest gains in life expectancy, albeit with different age patterns. Respiratory conditions were more important among younger age groups, and digestive among the older, irrespective of sex (figure 38). Between the period 1979 to 1996, mortality due to malignant neoplasms increased among men, acting as an obstacle for further improvements in life expectancy. A slight mortality increase due to these causes was also observed among women 75 and

Figure 30. Standardized mortality rates and relative mortality distribution by groups of causes and sex, Mexico, 1979 and 1996



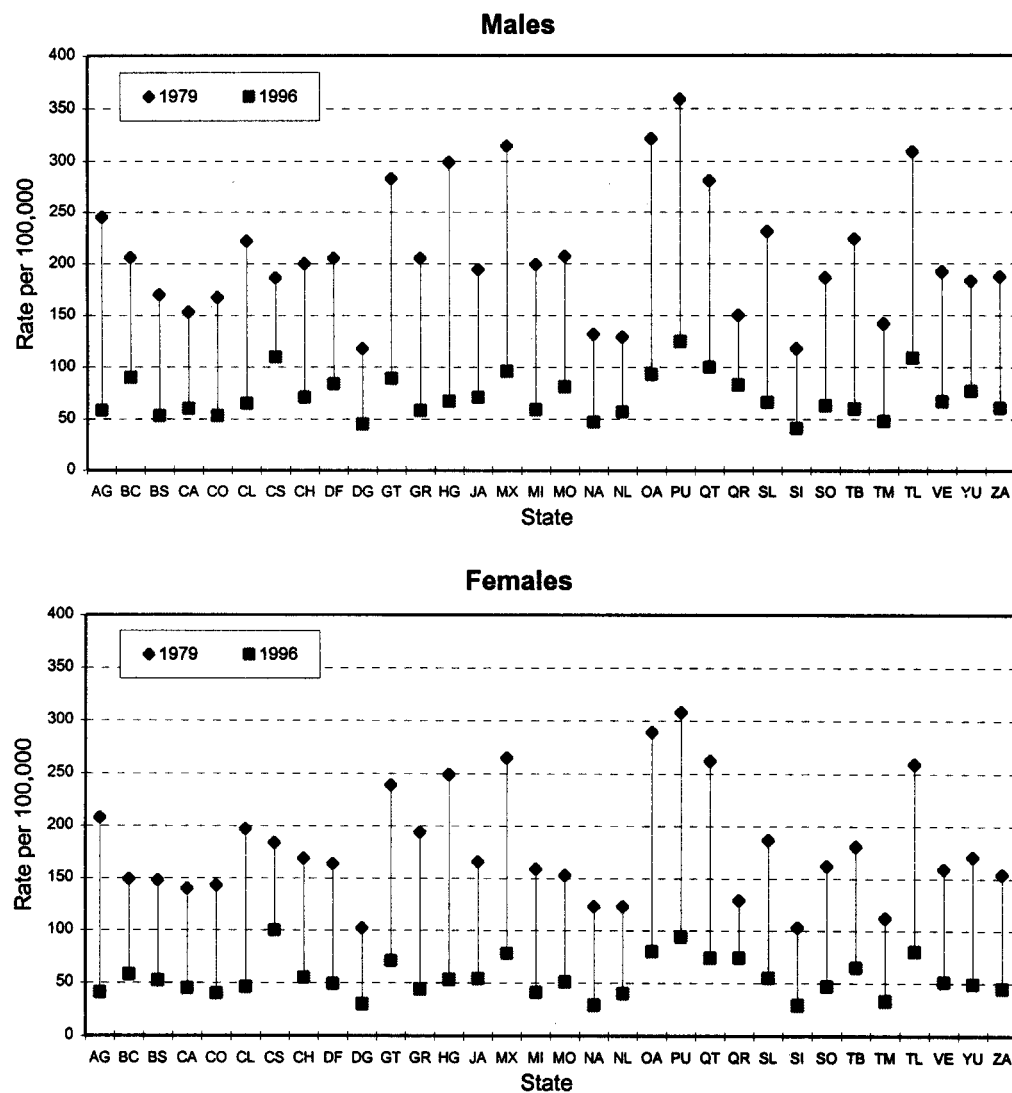
Source: Author's estimates based on national vital statistics.

Figure 31. Relative mortality distribution by age, sex, and groups of causes, Mexico, 1979 and 1996



Source: Author's estimates based on national vital statistics.

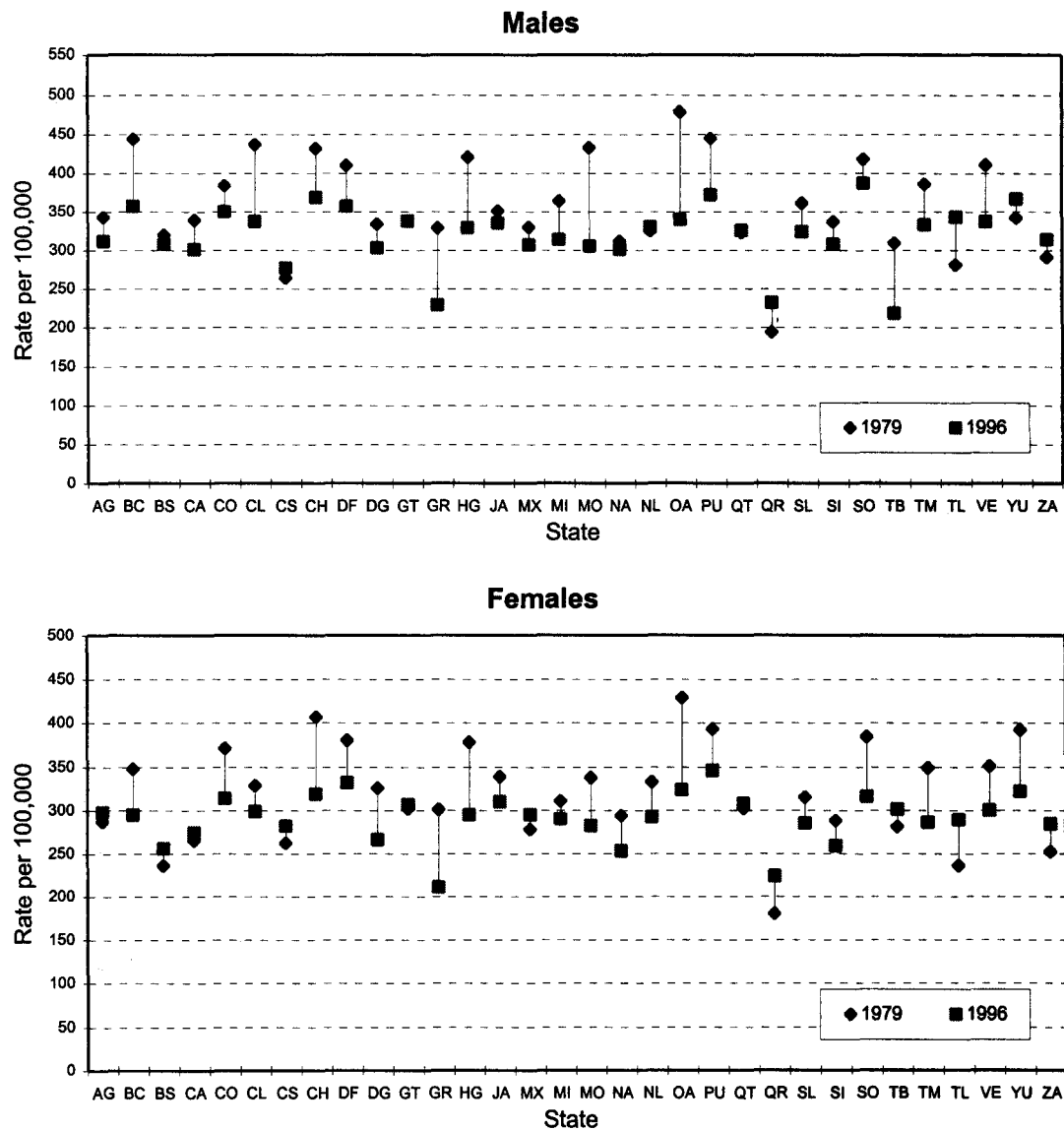
Figure 32. Standardized mortality rates due to communicable, maternal and perinatal causes, by sex and state, Mexico, 1979 and 1996



AG = Aguascalientes; BC = Baja California; BS = Baja California Sur; CA = Campeche; CO = Coahuila;  
 CL = Colima; CS = Chiapas; CH = Chihuahua; DF = Distrito Federal; DG = Durango; GT = Guanajuato;  
 GR = Guerrero; HG = Hidalgo; JA = Jalisco; MX = Estado de México; MI = Michoacán; MO = Morelos;  
 NA = Nayarit; NL = Nuevo León; OA = Oaxaca; PU = Puebla; QT = Querétaro; QR = Quintana Roo;  
 SL = San Luis Potosí; SI = Sinaloa; SO = Sonora; TB = Tabasco; TM = Tamaulipas; TL = Tlaxcala;  
 VE = Veracruz; YU = Yucatán; ZA = Zacatecas.

Source: Author's estimates based on national vital statistics.

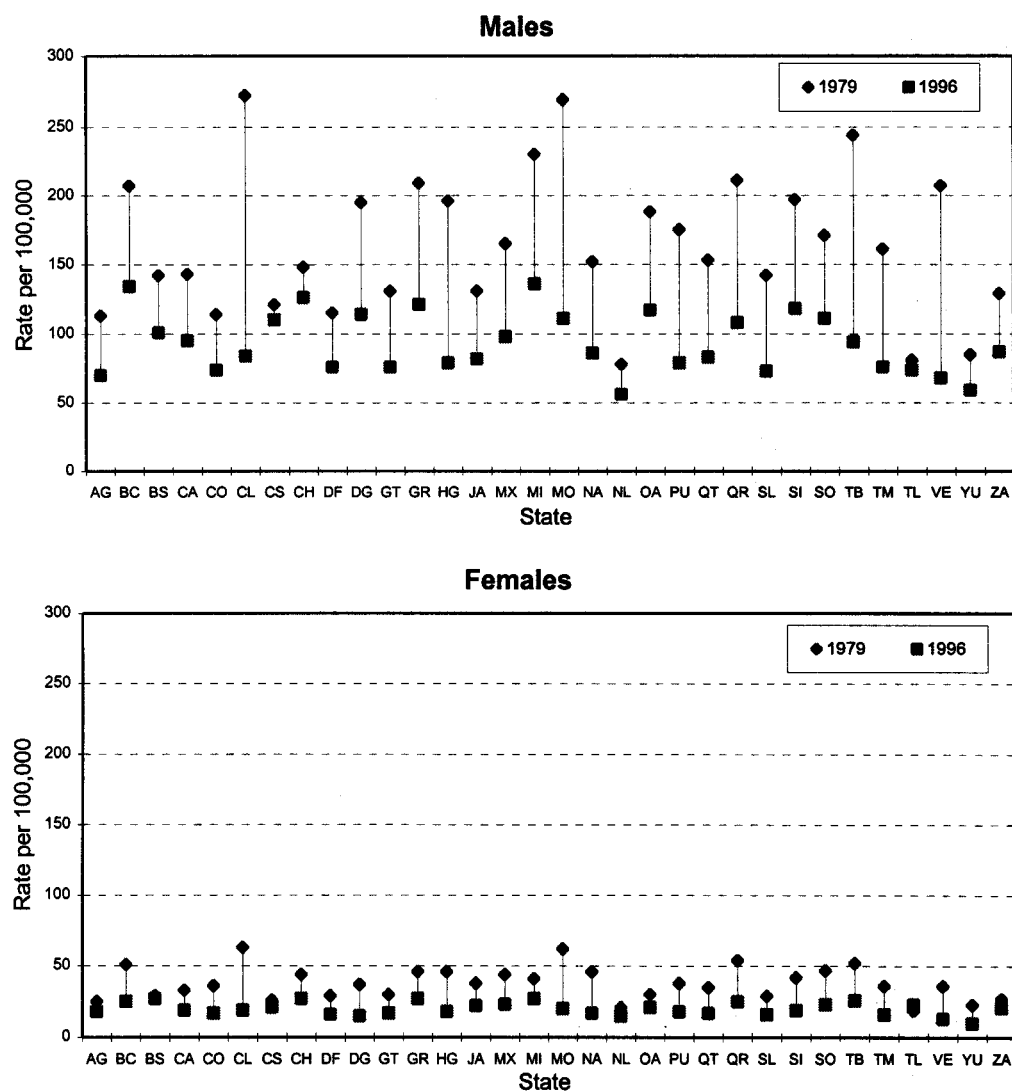
Figure 33. Standardized mortality rates due to non communicable causes, by state and sex, Mexico, 1979 and 1996



AG = Aguascalientes; BC = Baja California; BS = Baja California Sur; CA = Campeche; CO = Coahuila;  
 CL = Colima; CS = Chiapas; CH = Chihuahua; DF = Distrito Federal; DG = Durango; GT = Guanajuato;  
 GR = Guerrero; HG = Hidalgo; JA = Jalisco; MX = Estado de México; MI = Michoacán; MO = Morelos;  
 NA = Nayarit; NL = Nuevo León; OA = Oaxaca; PU = Puebla; QT = Querétaro; QR = Quintana Roo;  
 SL = San Luis Potosí; SI = Sinaloa; SO = Sonora; TB = Tabasco; TM = Tamaulipas; TL = Tlaxcala;  
 VE = Veracruz; YU = Yucatán; ZA = Zacatecas.

Source: Author's estimates based on national vital statistics.

Figure 34. Standardized mortality rates due to injuries, by state and sex, Mexico, 1979 and 1996

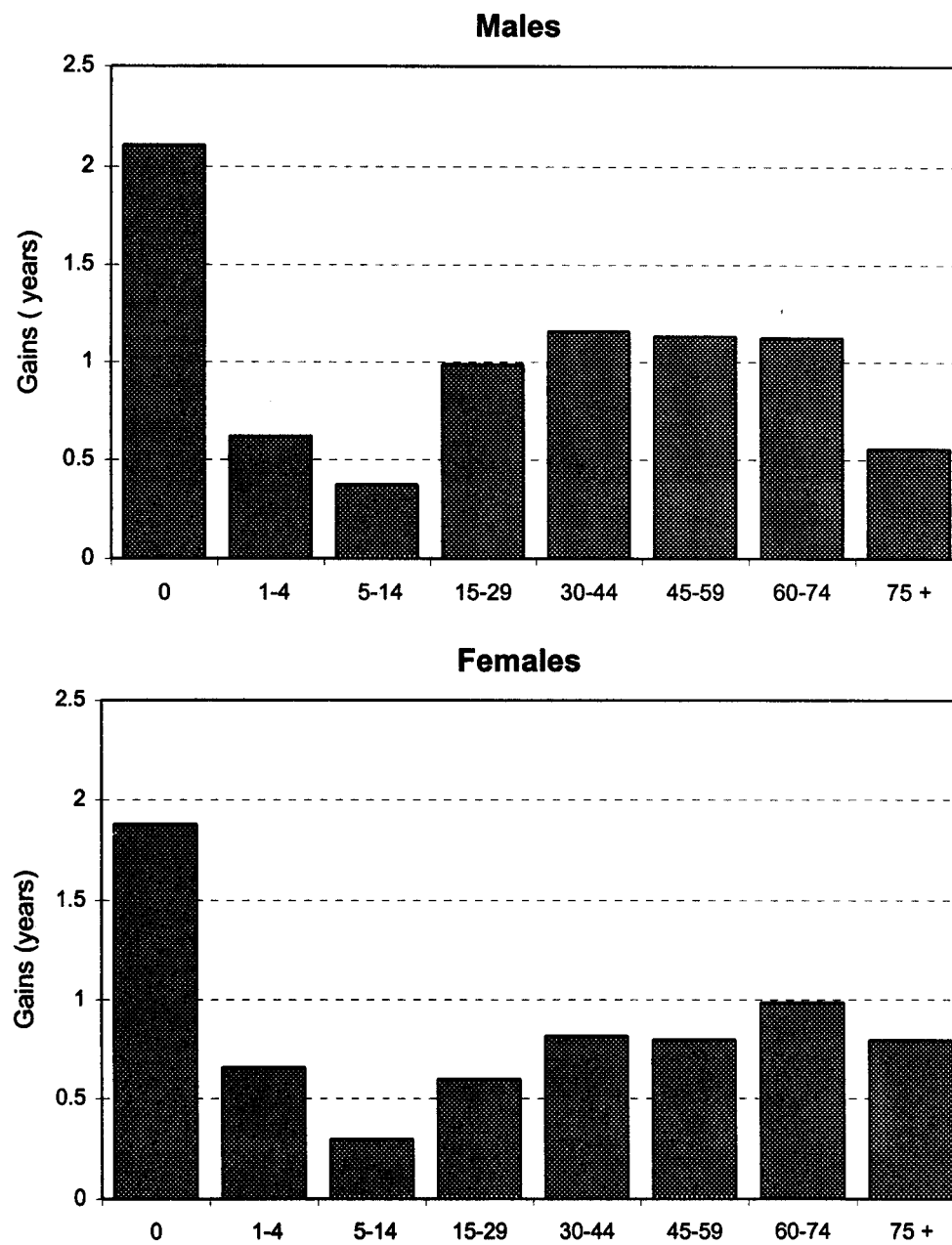


AG = Aguascalientes; BC = Baja California; BS = Baja California Sur; CA = Campeche; CO = Coahuila; CL = Colima; CS = Chiapas; CH = Chihuahua; DF = Distrito Federal; DG = Durango; GT = Guanajuato; GR = Guerrero; HG = Hidalgo; JA = Jalisco; MX = Estado de México; MI = Michoacán; MO = Morelos; NA = Nayarit; NL = Nuevo León; OA = Oaxaca; PU = Puebla; QT = Querétaro; QR = Quintana Roo; SL = San Luis Potosí; SI = Sinaloa; SO = Sonora; TB = Tabasco; TM = Tamaulipas; TL = Tlaxcala; VE = Veracruz; YU = Yucatán; ZA = Zacatecas.

Source: Author's estimates based on national vital statistics.

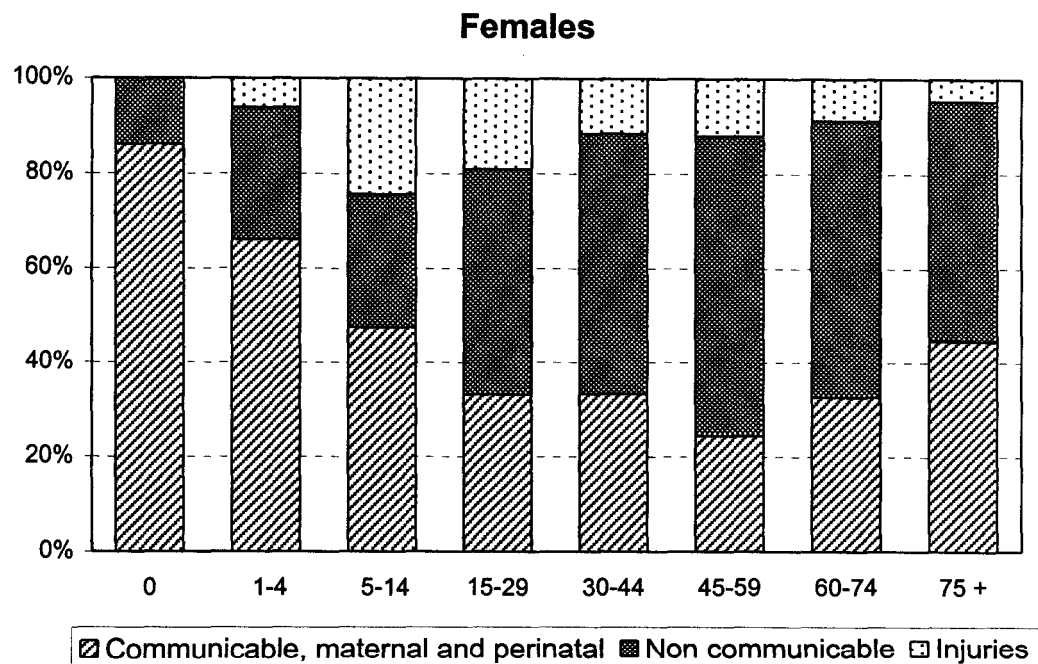
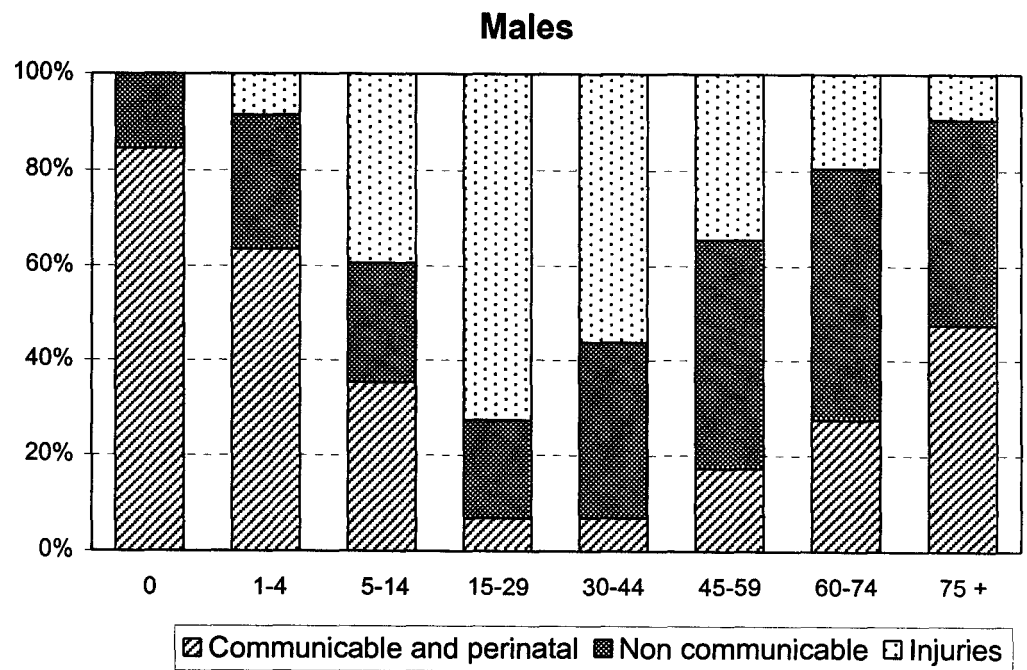


Figure 35. Gains in life expectancy by sex and age group, Mexico, 1979-1996



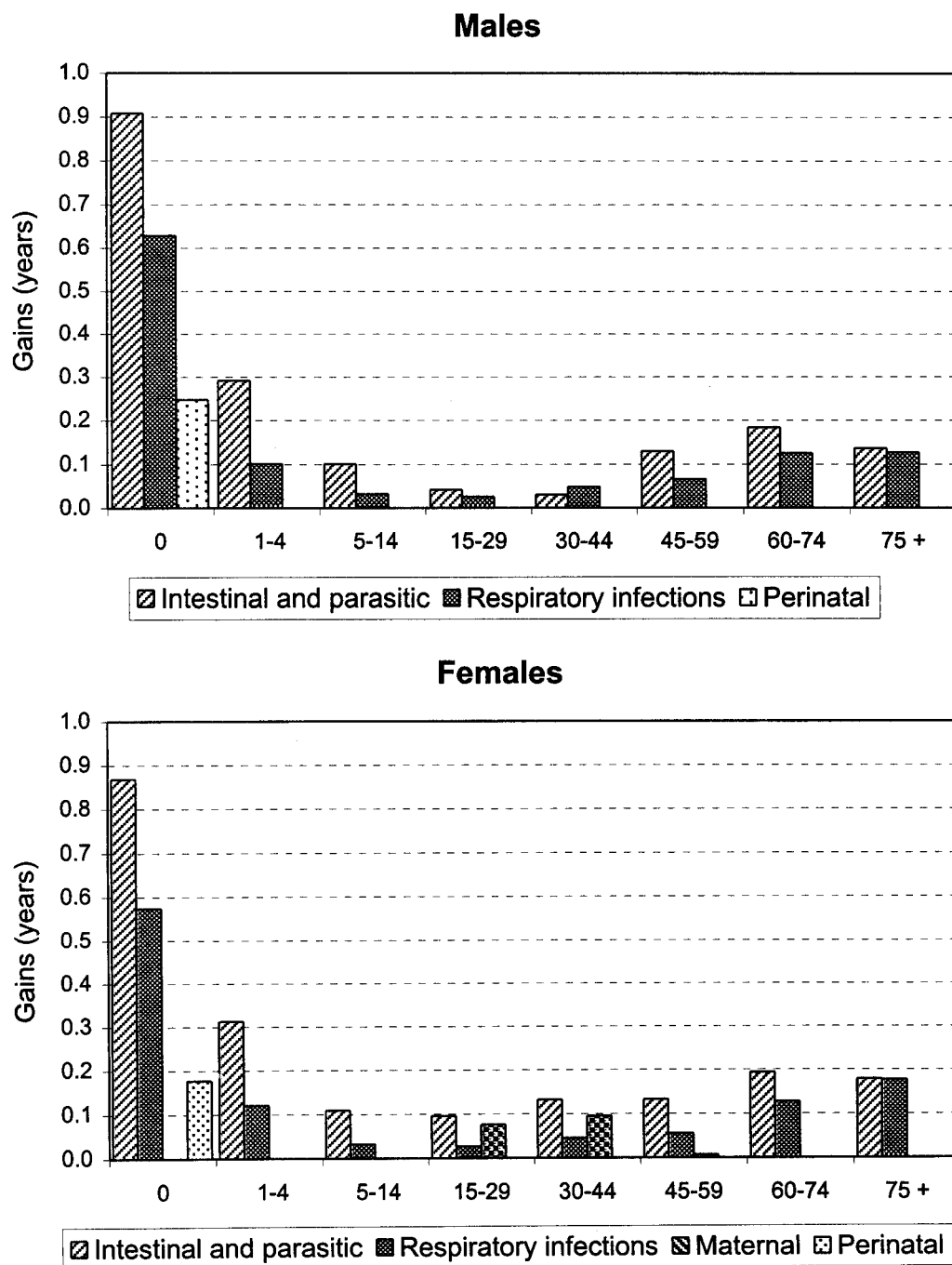
Source: Author's estimates based on national vital statistics.

**Figure 36. Relative distribution of life expectancy gains by sex, age, and groups of causes, Mexico, 1979-1996**



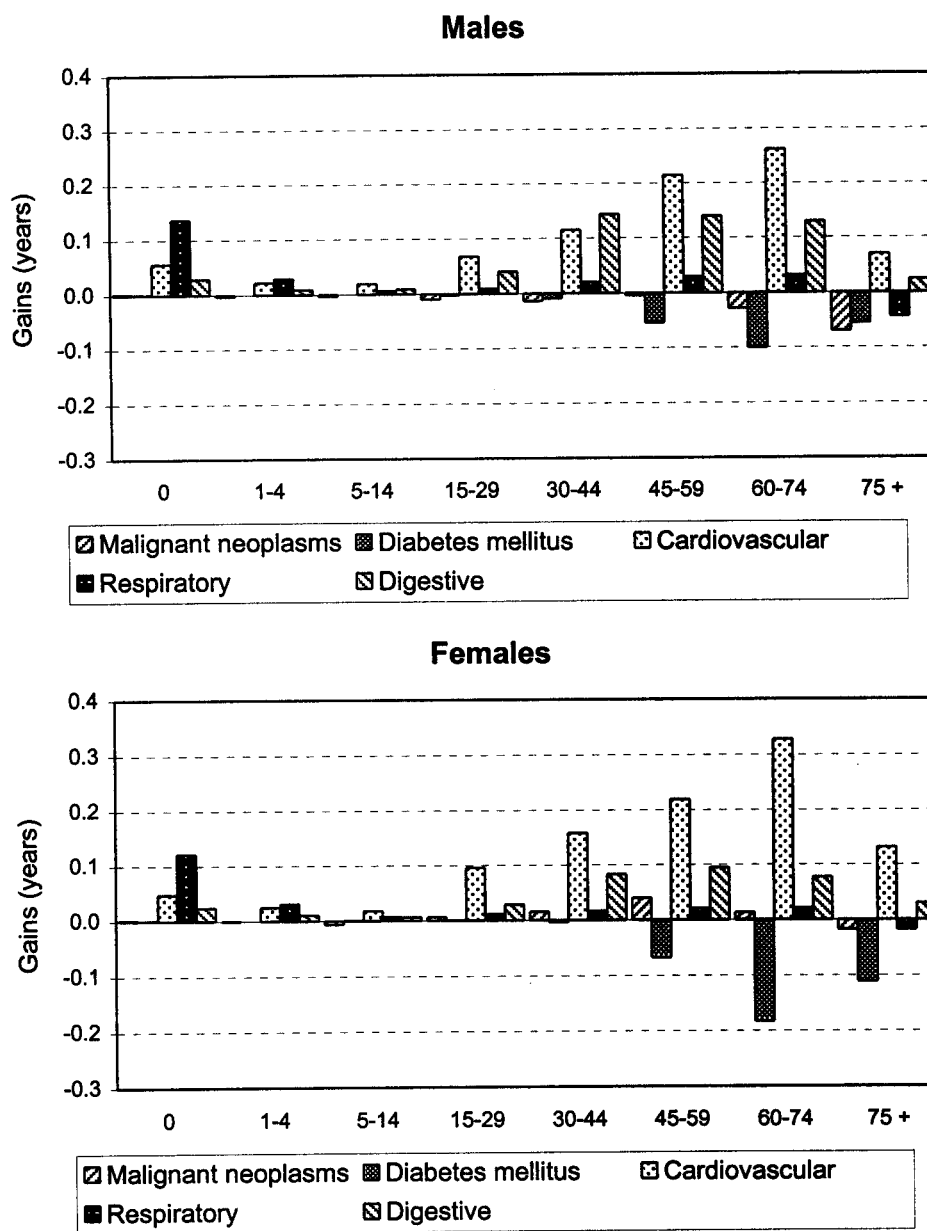
Source: Author's estimates based on national vital statistics.

**Figure 37. Life expectancy gains due to reductions in specific communicable, maternal and perinatal causes, by sex and age, Mexico, 1979-1996**



Source: Author's estimates based on national vital statistics.

Figure 38. Life expectancy gains due to reductions in specific non-communicable causes, by sex and age, Mexico, 1979-1996



Source: Author's estimates based on national vital statistics.

older. Nevertheless, diabetes mellitus is the non-communicable cause with the largest negative impact on life expectancy. Although this is the case for both men and women, the impact among women is larger, exceeding even the gains associated with reductions in digestive disease mortality.

The reduction in injury-related mortality mentioned above is reflected in the gains in life expectancy due to traffic accidents and homicide (figure 39). However, not all causes included in this group have had an impact in the same direction. Mortality due to suicide had a negative impact on life expectancy. For males, its effects are observed in all the age groups, starting in the teens. In the case of women, the impact of suicide is less. Nevertheless, it is also found among female teenagers as well as among women 45 to 75 years. Although very small, it is striking to observe a negative impact of homicide among very young women.

In general, premature mortality due to injuries is far more important among men than women (figure 44). Although the number of years of life lost due to injuries is smaller than for other groups of causes, their preventability should be stressed.

Estimates of the number of years of life lost identify the age group 60-74 years as having the largest premature mortality (figure 40). As with other indicators, the number of years of life lost is higher among men than women, irrespective of age. Non-communicable pathologies are the causes of most of the years lost due to premature mortality (figure 41). The number of years of life lost due to non-communicable causes among men is 9.3 years, and for women 8.4. The years of life lost due to injuries are notably higher among men.

Analysis of years of life lost due to specific communicable cause shows the comparatively small impact of maternity-related causes compared to intestinal infections and parasitic conditions (figure 42). Perinatal conditions are

the most important cause of premature mortality in this group of causes, its impact being greater among males, 1.2 years in comparison with 0.90 years of life lost among females.

The toll taken by premature mortality due to malignant neoplasms and diabetes mellitus is greater among women than men (figure 43). The opposite is observed regarding cardiovascular, respiratory, and digestive diseases. It is worth noting that despite reductions in digestive disease mortality, more than 1.6 years of life among men and 0.8 among women are lost due to these causes.

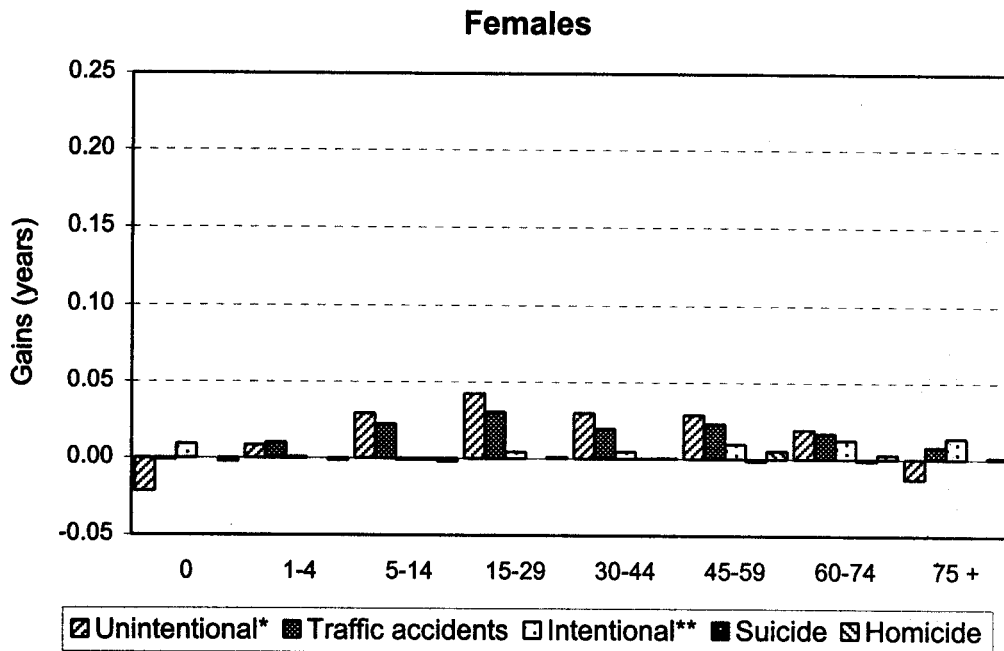
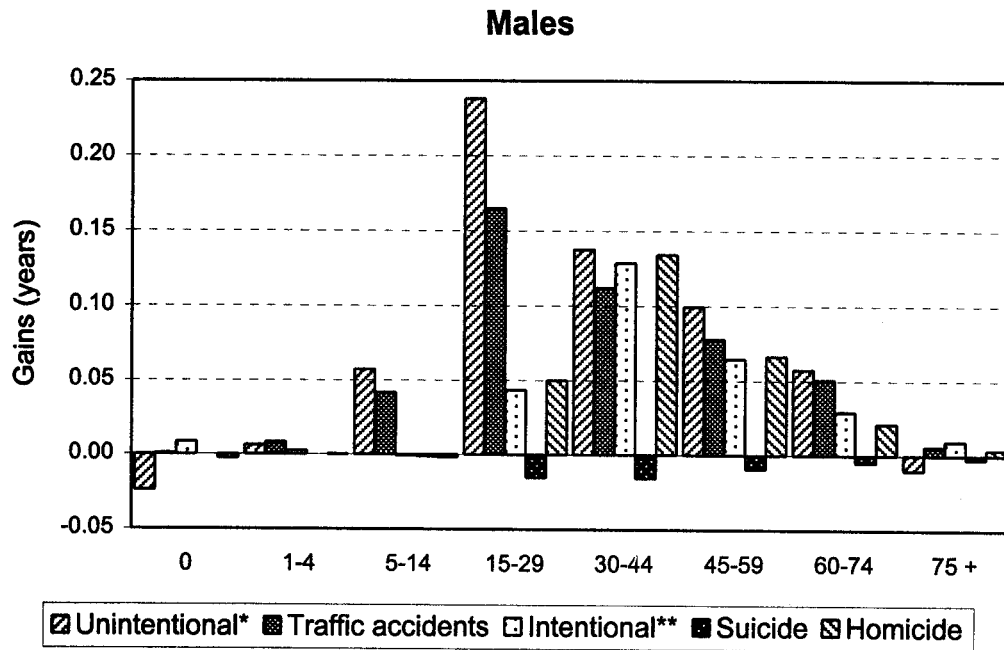
## E. CONCLUSIONS

State-level data shows a predominance of non-communicable causes in the epidemiological profile of Mexico. Thus, it is reasonable to conclude that the country, as a whole, has advanced in its epidemiological transition. Nevertheless, it is important to emphasize that Mexico is socio-economically heterogeneous. It is possible that studies using different units of analysis would reveal very different epidemiological profiles from the one presented here. Indeed, preliminary analyses of mortality data for the states with predominantly indigenous population in Mexico, reveal a diversity of cause-of-death structures.

The fact that mortality has steadily declined during the period under study, in spite of the economic recession that began in 1982, calls attention to aspects that might help explained this pattern. The observation that the greatest mortality reduction was in the states with the highest rates at the beginning of the period is an important point.

Several elements might account for these trends. Health interventions such as the expansion of immunization coverage and the promotion of oral rehydration therapy played a key role in reducing mortality in children under five years of age. However, the observed sex differences in gains in life expectancy due to perinatal conditions and infec-

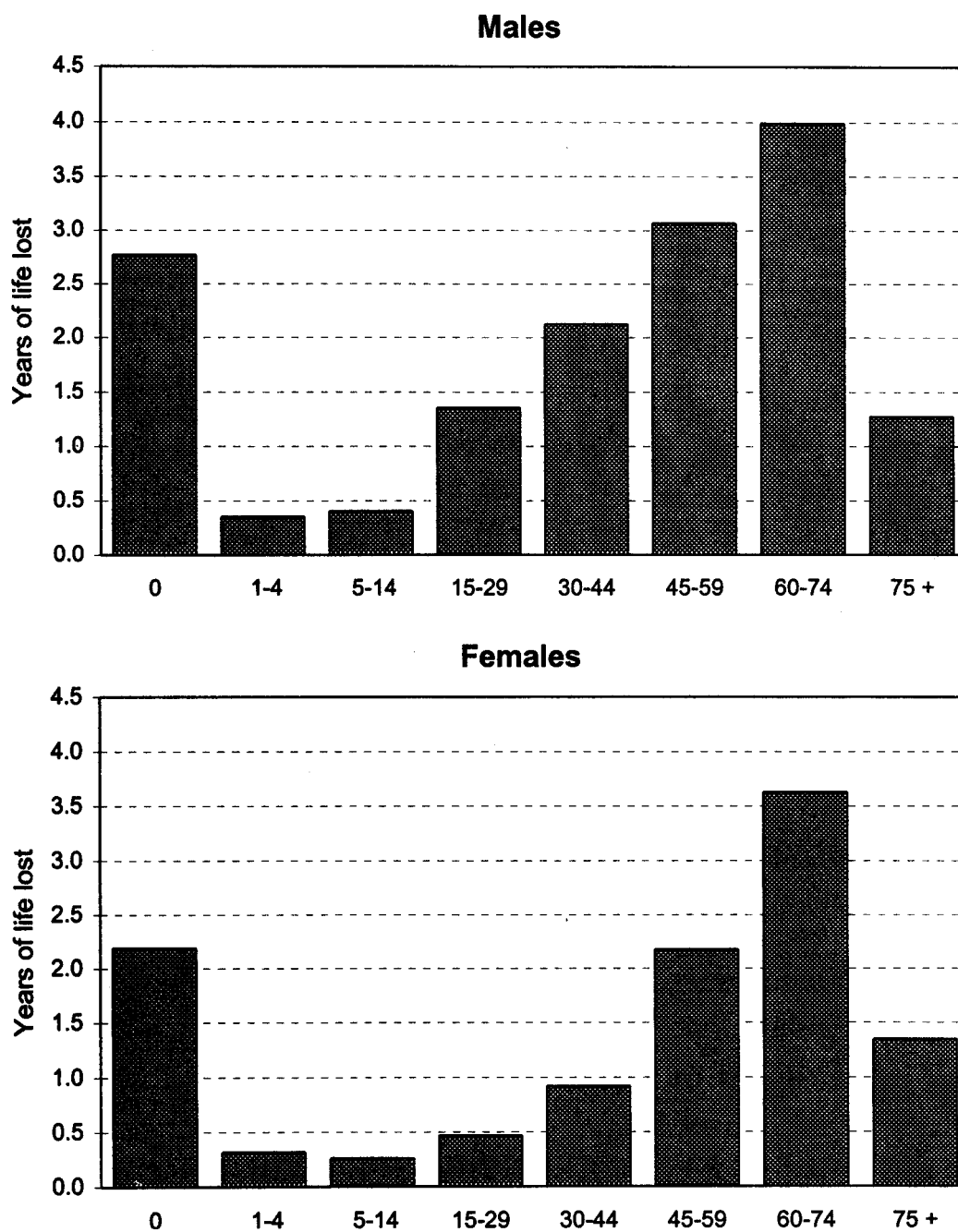
**Figure 39. Life expectancy gains due to reductions in specific types of injuries, by sex and age, Mexico, 1979-1996**



\*Unintentional injuries includes, in addition to traffic accidents, deaths due to poisoning, falls, fire, asphyxia or labor related. \*\* Intentional injuries includes, besides homicide and suicide, war related deaths.

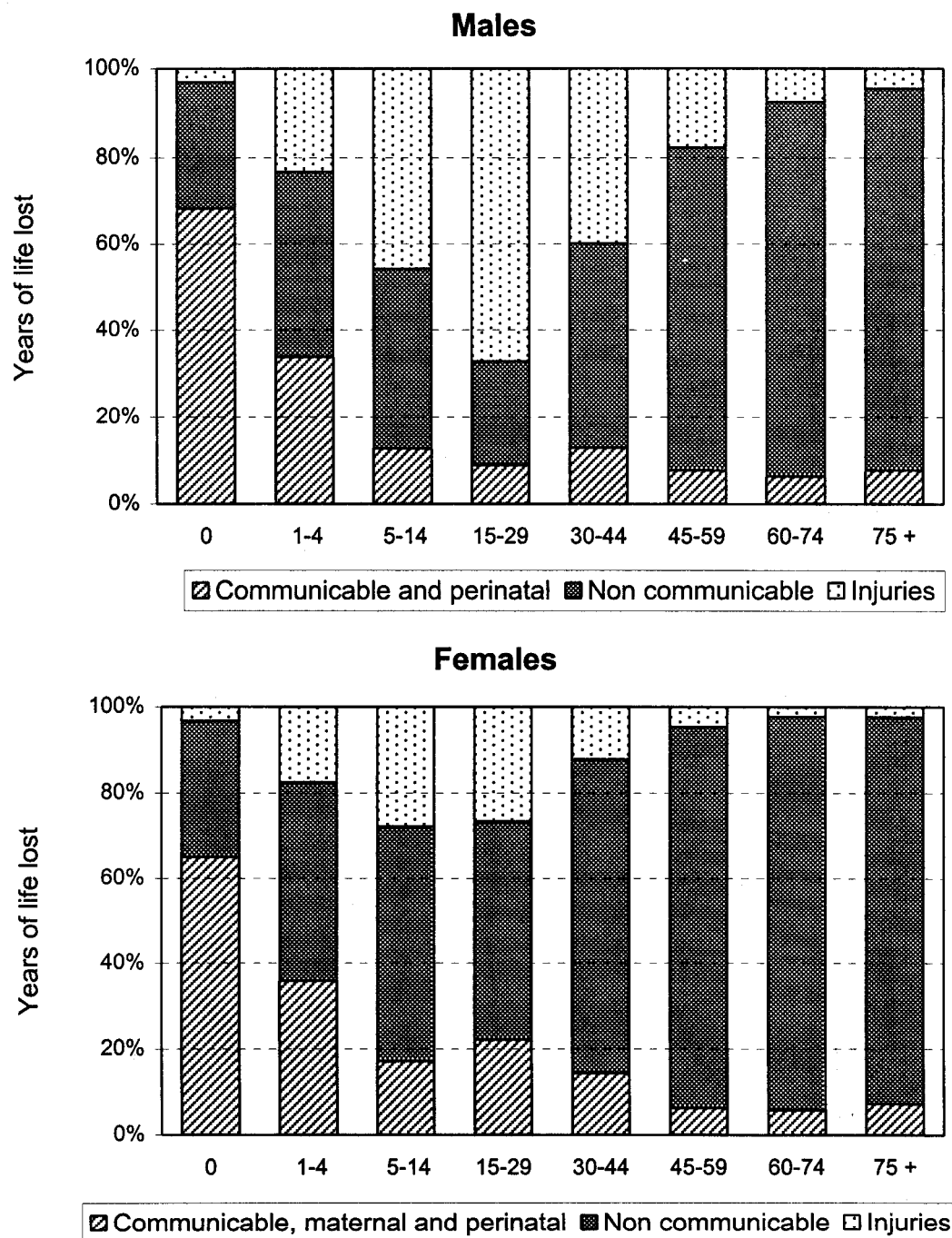
Source: Author's estimates based on national vital statistics.

**Figure 40. Years of life lost, by sex and age group, Mexico, 1996**



*Source:* Author's estimates based on national vital statistics.

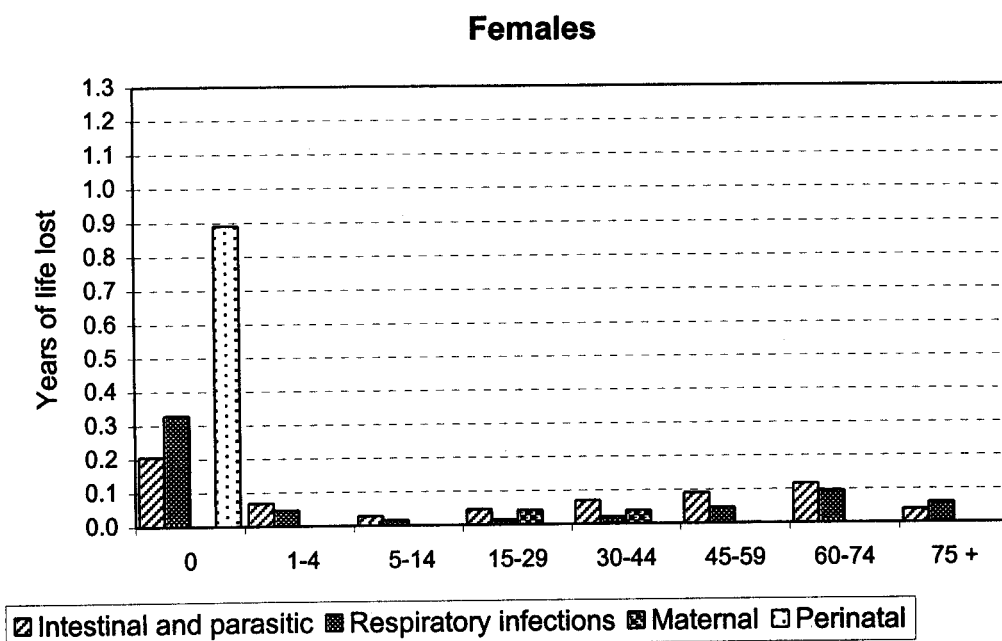
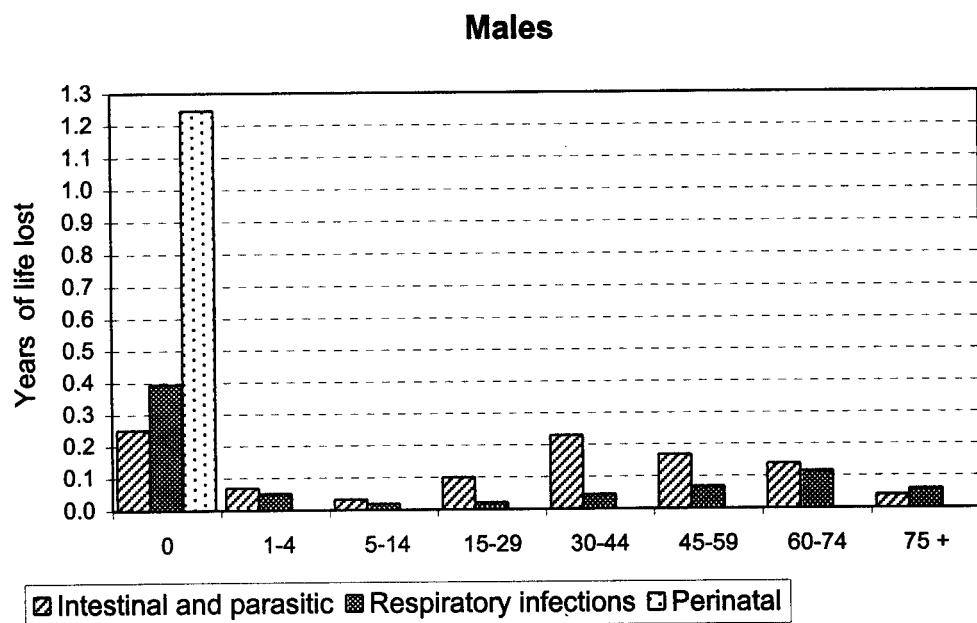
Figure 41. Years of life lost by sex, age, and groups of causes, Mexico, 1996



Source: Author's estimates based on national vital statistics.

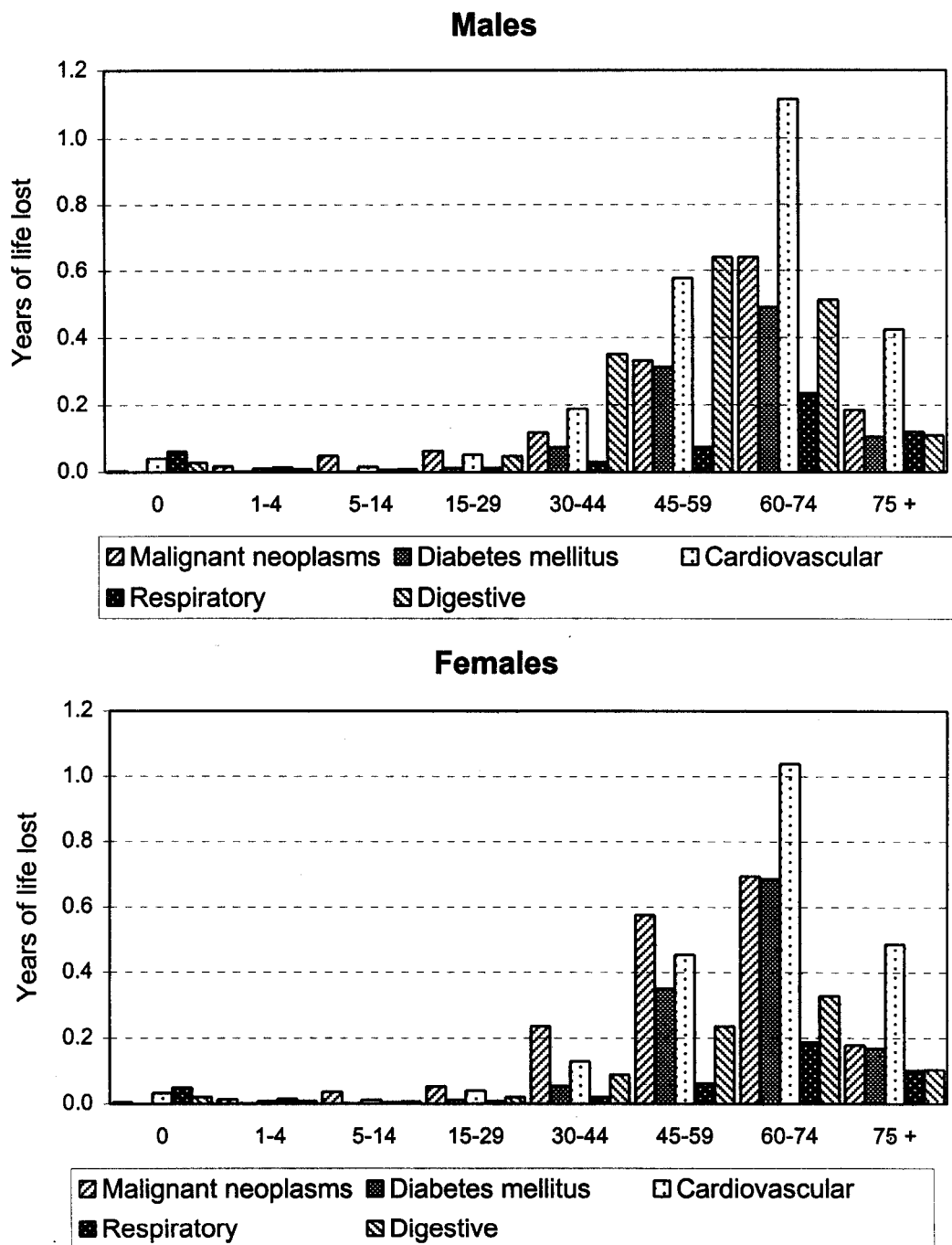


**Figure 42. Years of life lost due to specific communicable, maternal and perinatal causes, by sex and age, Mexico, 1996**



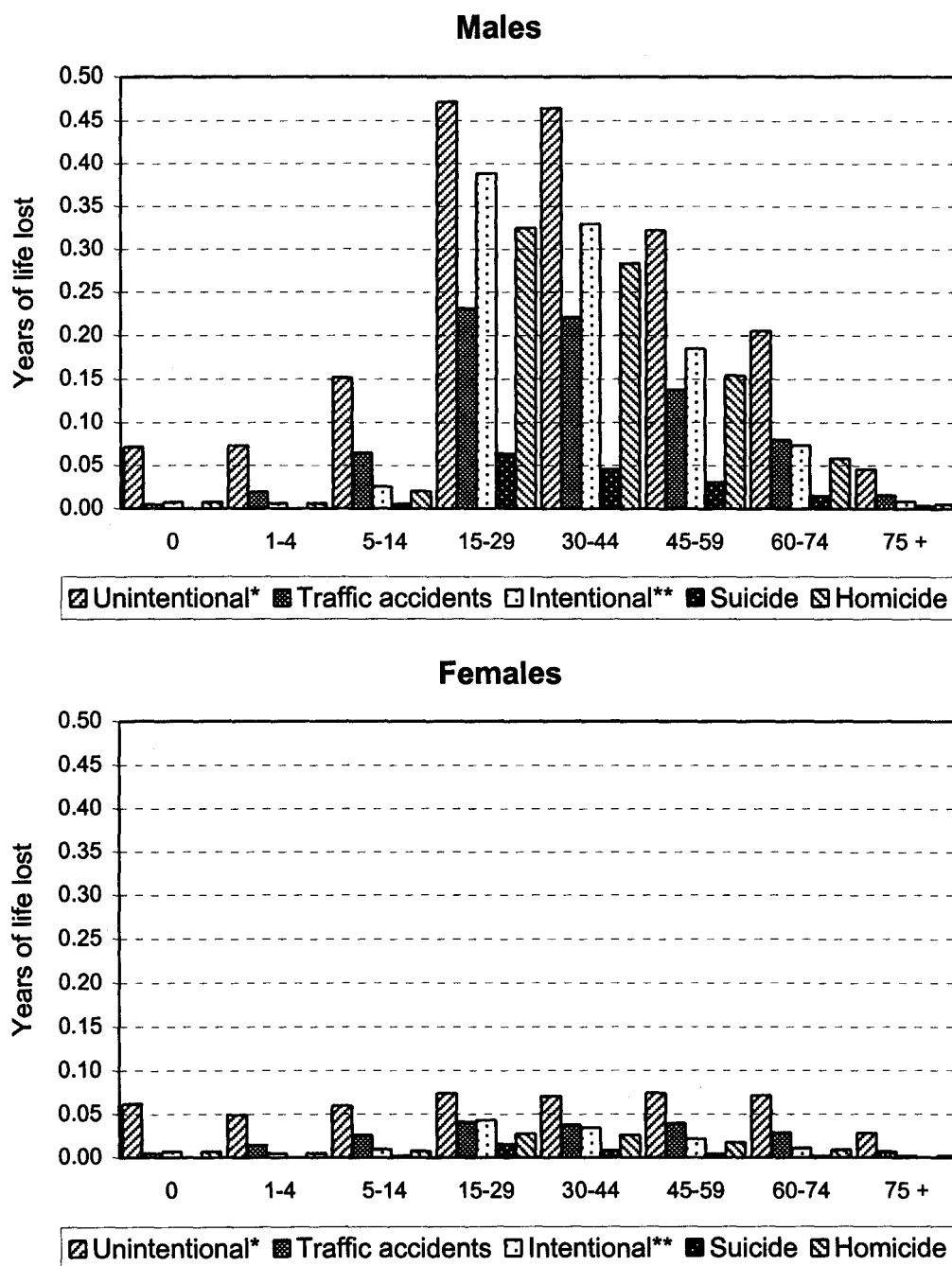
Source: Author's estimates based on national vital statistics.

Figure 43. Years of life lost due to specific non-communicable causes, by sex and age, Mexico 1996



Source: Author's estimates based on national vital statistics.

Figure 44. Years of life lost due to specific types of injuries, by sex and age, Mexico, 1996



\*In addition to traffic accidents, includes deaths due to poisoning, falls, fire, asphyxia or labor related. \*\*In addition to homicide and suicide, includes war related deaths.

Source: Author's estimates based on national vital statistics.

tious and parasitic causes among children under one, raises the question of possible discrimination against girls. The nature of the medical problems involved, together with the availability and type of medical technology needed to control them, make it difficult to consider these differences an expression of underlying biological conditions. It appears rather to be a matter of social causes. A similar set of factors might be at the root of the incipient increase of violent deaths observed among young women.

The specific program aimed at improving health of mothers and children might have lowered maternal mortality from the levels observed at the beginning of the period. However, the most recent data show some tendency for maternal mortality to persist at a level higher than that observed in other countries. It is likely that women at greater risk of dying of pregnancy related conditions are those located in remote rural areas, with limited access to health services. Hence health strategies different from those that reduced maternal mortality in the past need to be designed and implemented.

The decomposition of changes in life expectancy showed that children enjoyed the largest mortality reductions during the years after 1979. Adult men, particularly the younger, also experienced a considerable mortality decline, particularly in deaths due to injuries. Nevertheless, violent mortality continues to be a major health problem in Mexico.

During the period under study, women continue to have lower mortality than men. However, the reduction in male mortality exceeded that of females, except for those 70 years of age or older.

Although in general violent mortality has shown a decline, the increasing levels of suicide and homicides make it urgent to develop more effective measures to deal with these problems. At present, there are no formal programs aimed at curbing these problems.

Measures against consumption of illegal drugs are the only ones that might be having a slight impact upon violence. However, a broad social debate on gun control, as well as more psychological and medical support services for suicide attempts are among the steps needed.

The results reported in this paper have been brought about mainly by a combination of factors. They include a sustained public health efforts such as the immunization campaigns which resulted in the eradication of polio; a demographic change in the age-structure, which is a consequence in turn of the increase in life expectancy; and to certain extent, the wider availability of medical services.

Health policies should take into account the fact that the majority of the Mexico's medical problems are now of the non-communicable type. This means that programs should emphasize behavioural changes: better diet, more exercise, less use of tobacco, and alcohol abuse. At the same time the country must continue the expansion of vaccination coverage and the provision of primary health care.

The growing negative impact of diabetes mellitus, calls for further emphasising the adaptation of health services to the current features of the epidemiological profile. Health sector efforts should be directed both to reduction of the disease complications that particularly associated with diabetes mellitus, and health education leading to behavioural changes in the population. Several studies have pointed out the importance of family and social network support in helping patients control diabetes mellitus. This is a new aspect of the type of strategies that are needed to face the main health problems of an epidemiological profile such as that of Mexico: collaboration between families and/or social networks and health services.

Another finding is the fact that maternal mortality is not the most frequent cause of death among reproductive age women. Cervi-

cal and breast cancer are among the main problems of this population group. A new strategy to curb and reduce cervical cancer was launched by the health sector in October of 1997. However, breast cancer is not being addressed in the same way. Because of changes in age structure, as well as changing risk factors, it is likely that breast cancer incidence will increase in the near future.

The fact that cervical and breast cancer are mentioned as main health problems among reproductive age women should not lead to reduced efforts to reduce maternal mortality. There remains a wide gap between the levels observed in Mexico and in many other countries. With the eradication of polio and the control of childhood infectious diseases, Mexico may well be in a position to further reduce maternal mortality.

Finally, it is important to keep in mind that we are dealing with mortality data, the most extreme outcome of the health process. It is possible that in order to monitor for evidence of any possible deterioration of health conditions, we should also look into morbidity data. However, morbidity data are scanty and their coverage incomplete. Nevertheless, as the country moves further through its epidemiological transition, morbidity information will become more important for assessing health conditions. Efforts are being placed upon getting such information through the health statistics system. In the near future we may be able to start looking into epidemiological changes from an additional perspective. In the meantime, the analysis of available morbidity data should be started, so that the impact of diseases such as the sexually transmitted, or mental conditions such as depression, can be fully estimated.

#### NOTES

<sup>1</sup>Middle income refers to the classification used by The World Bank for ranking countries. It is based on Gross National Product per capita levels, measured in US dollars.

<sup>2</sup>Mortality data used in the analysis have been provided by the Directorate of Statistics and Informatics of the Ministry of Health. The estimated were produced by the author,

who is solely responsible for the views and opinions expressed.

<sup>3</sup>Mortality due to communicable, maternal and perinatal causes will be referred to hereinafter as communicable, although it will include the three types of causes.

#### REFERENCES

- Adlakha, A. L., and C. M. Suchindran (1985). Infant and child mortality in Middle Eastern countries. *International Population Conference, Florence*. International Union for the Scientific Study of Population, vol. 2, pp. 367-375.
- Arriaga, E. E. (1981). The deceleration of the decline of mortality in LDCs: the case of Latin America. In *International Population Conference, Manila*. Liege, Ordina Editions, vol. 2, pp. 21-50.
- \_\_\_\_\_. (1984). Measuring and explaining the change in life expectancies. *Demography*, vol. 21, No. 1, pp. 83-96.
- \_\_\_\_\_. (1994). Measuring the level and change of mortality by causes of deaths. The use of Years of Life Lost. Paper presented at the 1994 Population Association of America Meeting, Miami, Florida, 5-7 May.
- Comisión Nacional de Salarios Mínimos (n.d.) *Boletín*.
- Frenk, J., T. Frejka, J. L. Bobadilla, C. Stern, J. Sepúlveda, and M. José (1989). The epidemiologic transition in Latin America. *International Population Conference, New Delhi, India, 20-27 September*.
- Hill, K. (1985). The pace of mortality decline since 1950. In *Quantitative Studies of mortality decline in the developing world*, Julie DaVanzo, Jean-Pierre Habicht, Ken Hill and Samuel Preston, eds. Washington, D.C.: The World Bank (World Bank Staff Working Papers No. 683), pp. 55-95.
- Instituto Nacional de Estadística e Informática (INEGI) (1992). *Estados Unidos Mexicanos. Resumen General. XI Censo de Población y Vivienda, 1990*. INEGI.
- Kunitz, S. J. (1990). The value of particularism in the study of the cultural, social, and behavioural determinants of mortality. In *What We Know about Health Transition: The Cultural, Social and Behavioural Determinants of Health*, John Caldwell, Sally Findley, Pat Caldwell, Gigi Santow, Wendy Cosford, Jennifer Braid, Daphne Broers-Freeman, eds. Canberra, Australia: Health Transition Centre, The Australian National University, vol. 1, pp. 92-109.
- Lee, E. (1985). Epidemiological transition in Korea: a new perspective in population and development studies. *Bulletin of the Population and Development Studies Center*, vol. 14, pp. 1-14.
- Meegama, S. A. (1981). The decline in mortality in Sri Lanka in historical perspective. *International Population Conference, Manila*. Liege, Ordina Editions, vol. 2, pp. 143-164.
- Murray, C. J. L., Y. Gonghuan and Q. Xinjian (1992). Adult mortality: levels, patterns and causes. In *The Health of Adults in the Developing World*, Richard G. A. Feachem, Tord Kjellstrom, Christopher J. L. Murray, Mead Over, and Margaret Phillips, eds. New York: Oxford University Press for the World Bank, chapter 2, pp. 23-111.
- Olshansky, S. J., and A. B. Ault (1986). The fourth stage of the epidemiologic transition: the age of delayed degenerative diseases. *Milbank Memorial Fund Quarterly*, vol. 64, pp. 355-391.

- Omran, A. (1971). The epidemiologic transition. A theory of the epidemiology of population change. *Milbank Memorial Fund Quarterly*, vol. 49, pp. 509-538.
- Palloni, A. (1981a). Mortality in Latin America: emerging patterns. *Population and Development Review*, vol. 7, No. 4, pp. 623-649.
- \_\_\_\_\_ (1981b). Current mortality conditions in Latin America with emphasis on infancy and early childhood. *Statistical Bulletin of the OAS*, vol. 3, Nos. 3-4, pp. 1-26.
- \_\_\_\_\_ (1989). Population trends and economic crises in Latin America: Is there any evidence of a relation? Paper presented at the Annual Meeting of the Population Association of America, Baltimore, 30 March-1 April.
- Ruzicka, L. T., and H. Hansluwka (1982). Mortality transition in South and East Asia: technology confronts poverty. *Population and Development Review*, vol. 8, No. 30, pp. 567-588.
- Sivamurthy, M. (1981). The deceleration of mortality decline in Asian Countries. In: International Population Conference, Manila. Liege, Ordina Editions, vol. 2, pp. 51-76.

## VIII. THE USE OF SURVEYS TO GATHER INFORMATION ON HEALTH STATUS (DEVELOPING COUNTRIES)

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### A. INTRODUCTION

The need and demand for health status indicators has grown in recent years with the recognition of the importance of monitoring and evaluating of progress towards the achievement of "health for all". Recent international conferences and summits, particularly the 1992 World Summit for Children, the 1994 International Conference on Population and Development, and the 1995 Fourth World Conference on Women, have affirmed the primacy of health, education, and the status of women for social and economic development. The World Health Organization (WHO) has also embarked on a global initiative to promote practical policies and programmes to reduce avoidable social gaps in health and health care (World Health Organization, 1996).

According to WHO constitution, health is defined as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity; it should enable people to lead socially and economically productive lives. Health status thus is a dynamic, multi-dimensional concept, varying on a continuum from one extreme of ill-health (death) to the other of perfect health (Hansluwka, 1987). An abundant literature discusses the many concepts of health, the multitude of indicators available to measure health status, and criteria to be used to select appropriate measures in particular applications (Caplan, Engelhardt and McCartney, 1981; Carlson, 1985; Feachem, Graham and Timæus, 1989; Hansluwka, 1987; McDowell and Newell, 1987; Murnaghan, 1981; Ware, 1981; Wilson and Drury, 1984; World Health Organization, 1981).

Traditionally, health indicators have concentrated on mortality based measures; little atten-

tion was paid to measures of the other components of the health spectrum. In recent years, increased attention has been given to measuring "wellness" rather than illness. However, experimentation with indicators of positive health has given rise to concern that a concentration on positive health may deflect attention from the issues of practical relevance to health policy in developing countries (Hansluwka, 1987). While indicators on the extremes of health status are of interest, information on the departure from health is of greater interest, and it seems practically more relevant to focus on the in-between states, on indicators of illness or morbidity.

A large number of the "health for all" indicators can best be estimated using data collected by sample surveys of the population (World Health Organization, 1981, 1996). This applies mainly to indicators of self-perceived health status, longstanding illness or disability, use and non-use of health services, reproductive health, child health, lifestyle, health promotion, and environmental health.

### B. RELEVANCE OF HEALTH SURVEYS AND THE NEED FOR STANDARDISED GUIDELINES

Household health surveys provide data on health status and on equity in health that cannot sufficiently or efficiently be collected by other methods. Well known examples include the health interview survey in the United States, undertaken continuously since 1957, health interview surveys in Japan (since 1953), the Netherlands (since 1981), and England (since 1991), and the disability surveys conducted in Canada since 1983.

Since the mid-1980s, more than 40 developing countries have collected data on maternal and child health as part of the Demographic and Health Surveys programme, sponsored by USAID. Maternal and child health data have

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also been collected in seven Arab Gulf states as part of the Gulf Child Health Survey programme, conducted in 1987-89, and in ten Arab countries under the auspices of the Pan Arab Project for Child Development (PAPCHILD), conducted during 1989-96. A household health interview survey in six Arab Gulf states, known as the Gulf Family Health Survey, has been in progress since 1995. The League of Arab States is currently developing an ambitious health survey programme to be known as the Pan Arab Project for Family Health (PAPFAM), with the collaboration of the Arab Gulf Programme for the United Nations Development Programme, the United Nations, and the European Union. PAPFAM will be implemented in 16 Arab countries and will include household health interview surveys, health examinations, qualitative surveys of correlates of reproductive health, and situation analyses.

Many developing countries have also implemented general morbidity and disability surveys within the framework of the United Nations Household Survey Capability Programme (UNHSCP). Most of these surveys were one-time or ad hoc affairs used to obtain baseline information for determining the presence or magnitude of a disease or health problem (White, 1985).

The diversity in the methods and instruments used in health surveys in developing countries has imposed limits on the use of the data for any comparative analysis. Some of the main reasons for the diversity are narrow conceptualization, inappropriate outcome indicators, limited measurement techniques and lack of trained and experienced health data analysts. At this point, there is still no standardized and well-tested model household health interview survey available for use in developing countries.

The need to improve this situation is becoming more urgent with the growing emphasis on equity in health and health care, and on the reproductive health of women which is a key component of overall general health.

Many developing countries have already gained experience in conducting demographic surveys through participation in the World Fertility Survey and the Demographic and Health

Surveys. A comparable international initiative is urgently needed to produce guidelines for household health surveys in two broad areas: survey strategy and survey operations. Such an initiative would bring together specialists in "social" health, bio-medical scientists, programme managers and policy makers, women's health advocates, and other concerned agencies and persons, each contributing from their particular perspectives.

Barnum (1987) has called for the establishment of a World Health Survey to collect comparable and accurate epidemiological information. Timæus and others (1988), examining Barnum's suggestion, concluded that a multi-country single-round household health survey could be designed. A similar conclusion could be drawn from the successful implementation of the first round of the Gulf Family Health Survey.

The most urgent need is for the development of standardised guidelines that would discuss and clarify alternative survey designs, and provide a list of internationally comparable health indicators that are feasible, objective and useful to health policy in developing countries. The need for standardised guidelines is urgent. Many of the concepts and definitions are still vague and evolving, measurement tools are either cumbersome or unavailable, and data collection procedures are not delineated, especially because they must include a method mix of both quantitative and qualitative techniques.

The guidelines should propose a series of modules for priority areas, morbidity, long term disability, reproductive health, and associated use of services. They should discuss the many problems associated with measuring ill health. In particular, they should:

- Suggest a logical ordering of questions and the use of optimum recall periods;
- Recommend procedures for the design of national probability samples and the determination of the sample size;
- Discuss alternative modalities of data collection, particularly with regard to the period of field operations and the effect of seasonality;



- Consider how best to use lay interviewers and proxy informants;
- Provide instructional materials to help in training of staff at various levels;
- Provide state-of-the-art data processing and analysis software tailored to the needs of health and bio-demographic survey data analysis.
- *General morbidity module:* perceived health, illness or injury in the last two weeks, number of days of illness during the last two weeks, whether the person was kept from his/her normal activities because of this illness, utilization of health services, and medication.
- *Long-standing illness module:* in this module information is gathered on a number of chronic conditions, e.g. high blood pressure, heart disease and whether the person ever had myocardial infarction, asthma, diabetes, joint disease, ulcers, kidney disease, liver disease, severe headache, epilepsy, stroke, and mental conditions.

### C. INSTRUMENTS FOR HOUSEHOLD HEALTH SURVEYS

The main aim of a household health survey is to obtain information on self-perceived morbidity, use and non-use of health services, and health-related behaviour. Information on socio-economic status and environmental conditions is also gathered to permit an analysis of health and health care differentials.

The instruments for the survey may be grouped into the following four questionnaires:

- household health status;
- household socio-economic status and environmental conditions;
- reproductive health;
- child health.

Each questionnaire contains a set of self-contained modules that deal with particular health or health-related topics.

#### 1. Household health status questionnaire

This family-oriented questionnaire is used to gather information on the general health of all members of the household. The questionnaire is administered to the head of the household, who is asked to respond for all members.

The recommended household health status questionnaire consists of five modules:

- *Demographic and socio-economic module:* age, sex, relation to head, marital status, educational status, and economic activity.
- *Other indicators of health status:* this module is concerned with other aspects of health status: e.g. psychiatric care, dental care, use of glasses or contact lenses, use of hearing aids, smoking, and alcohol consumption.
- *Adult mortality module:* in this module information is gathered on the survival of close relatives. The most widely tested and applied method, the so-called "orphanhood" technique, is based on two simple questions put to all members of the household on the survival of the biological parents. An alternative procedure, known as the "sibling survival" method, is to ask all adult respondents over the age of 14 years about the survival of their brothers and sisters. Supplementary information which

For any given disease, the head of household is asked "Does anyone in this household now have or has anyone ever had (NAME OF DISEASE)?" For each individual with a given disease the following information is ascertained: whether the person was told by a doctor that he/she had the given condition, age at diagnosis, and current medication.

The long-standing illness module should also include a section on disability with questions on type of disability, cause of disability, age at which the individual had the condition, and whether he/she currently receives socio-medical care.

might be collected in addition to the questions on the survival of parents and of siblings, ask about the survival of first spouses (the "widowhood" method) (United Nations, 1983; 1995).

## 2. *Household socio-economic status and environmental conditions questionnaire*

This questionnaire includes two modules:

- *Environmental conditions:* housing, cooking, drinking water, lighting, sanitation, waste disposal, and drainage.
- *Ownership of objects and assets.*

## 3. *Reproductive health questionnaire*

This questionnaire is administered to all women under 50 years of age. It consists of a series of modules covering the following topics:

- *Woman's resources:* date of birth, current age, place of residence (current, prior, as a child) and duration of residence, literacy and level of education, radio and television exposure, work status, occupation, desire to work for cash in the future.
- *Marriage:* current marital status, number of times married, date of first marriage, blood relation to husband, number of other wives, and age at menarché.
- *Reproduction:* number of sons, daughters ever born/now living, full or short pregnancy or birth history, number of still births, number of miscarriages and abortions, current pregnancy status and preference for the sex of expected baby, persons helping with daily chores during current pregnancy, preference for place of delivery, and expected duration of breastfeeding.
- *Maternal health in the last three years:* maternal health of women who had at least one pregnancy in the three years preceding the survey. For each pregnancy, the respondent is asked three sets of questions: on ante-natal care and

health status during pregnancy, care at the delivery, and post-natal care.

The first set covers questions on ante-natal care attendance: timing of first check-up, reason for first check-up, number of pregnancy checks, duration since last visit, nature of care, type of health facility providing care, accessibility of ante-natal care, waiting time at facility, satisfaction with service provided, reason for not having pregnancy check, medication during pregnancy, number of tetanus toxoid injections, bleeding during pregnancy, differential diagnosis of bleeding during pregnancy, symptoms of pre-eclampsia, differential diagnosis of eclampsia, fever in pregnancy, pregnancy diabetes, and symptoms of anaemia.

The second set, on delivery, covers: place of delivery, attendance at delivery, length of labour, assisted delivery, reason for caesarean-section, bleeding during delivery, convulsions during labour or delivery, reason for delivery at health facility/at home, whether delivery was at expected time, and whether the birth weight was low, normal or high.

The third set, on post-natal care, covers: post-natal care attendance, type of care, reason for no check-up, bleeding during the postpartum period, differential diagnosis of eclampsia, fever in the post-natal period, and differential diagnosis of fever in puerperium.

- *Child feeding (asked about all live births in past three years):* prevalence, initiation, duration, and frequency of breastfeeding, demand versus schedule breastfeeding, breastfeeding during diarrhoeal episodes, reason for not breastfeeding, bottle feeding, age at introduction of the bottle, supplementation with solids, first liquid/solid given to baby other than milk, weaning foods, reason for early weaning, reason for weaning, and weaning practices.
- *Family planning:* knowledge of various methods, past and current use of meth-

ods, parity at first use, sources for modern methods, reasons for discontinuation of contraceptive use, intention to use in the future, and reasons for non-use.

- *Fertility preferences:* desire for more children, ideal number of children, family planning and family size discussions, and unplanned fertility.
- *Maternal anthropometric status.*
- *Women's status and aspirations for children.*
- *Husband's background.*

#### 4. Child health questionnaire

This questionnaire is used to gather information on all children, under five years of age who were enumerated in the household survey. The recommended modules may be grouped into the following four sections:

- *Child care:* age of child, prime responsibility for the care of child (usually and when mother leaves home for work/visiting, etc.), place of care, whether child is enrolled in a kindergarten, whether father plays regularly with child, who prepared food for the child, who feeds the child with liquids/solids, who puts baby to sleep, whether child is struck to discipline and if yes how often.
- *Child health status:* recent illness episodes, specific past morbidities, injury risks, and curative practices.

On recent episodes of four acute infectious disease conditions:

- diarrhoea: 24-hour and 2-week recall, duration of last episode, patterns of feeding during last episode (breast-milk, liquids, solids), treatment by type, use of health services, and reason for not seeking treatment;
- respiratory infections: 2-week recall, severity, duration of last episode, treatment by type and use of health services;
- ear infections: whether frequent, and 2-week recall; and

- eye infections: whether frequent, and 2-week recall.

On specific past morbidities and cumulative consequences of health risks:

- whether child now has/ever had urinary tract infection, skin allergy, any kind of food allergy, deafness or trouble hearing, blindness or trouble seeing, crossed eyes, stammering or stuttering, rickets;
- whether child had congenital hip dislocation at birth;
- whether child ever had measles/mumps/German measles/chickenpox, and age at infection;
- injuries: ever and past 12 months, cause, type, place and long-term implications.
- *Immunization:* health card coverage and availability, BCG, DPT, polio, measles, MMR, hepatitis B, and reason for not immunizing the child.
- *Nutritional status of children under five years of age:* birth weight, weight and height (or recumbent length), and vitamin A deficiency.

#### D. PRELIMINARY FINDINGS FROM THE GULF FAMILY HEALTH SURVEY

##### 1. Survey design

The Gulf Family Health Project (GFHP) is a multi-component research programme of the Council of Health Ministers of the Gulf Cooperation Council States. The main objective of the GFHP is to provide policy-makers, programme managers and researchers with a timely and integrated flow of reliable information suitable for formulating, implementing, monitoring and evaluating family health and related development policies and programmes in a cost-effective manner.

The first component of the GFHP involved the development of data collection tools to measure health status, service use, content, quality and coverage of health services, through conducting large, population-based surveys. A household survey programme, known as the

Gulf Family Health Survey (GFHS) has already been conducted in Bahrain, Oman and United Arab Emirates in 1995, in Kuwait and Saudi Arabia in 1996, and will be carried out in Qatar during 1998.

Four questionnaires were used in the GFHS: the household health status questionnaire, the household socio-economic status questionnaire, the reproductive health questionnaire, and the child health questionnaire.

The remainder of this paper will be devoted to a brief description of main findings from GFHS on selected demographic and health topics. The figures presented here were derived from the Preliminary Reports on the family health surveys in Bahrain (Yacoub, Naseeb and Farid, 1996), Kuwait (Al-Rashoud and Farid, 1997), Oman (Sulaiman, Al-Riyami and Farid, 1996), Saudi Arabia (Khoja and Farid, 1997), and the United Arab Emirates (Al-Qassimi, Fikri and Farid, 1996). It should be pointed out that the indicators shown here refer only to *nationals*.

## 2. General health of the adult population

### *Characteristics of the household population*

Table 20 gives information on the numbers of households, individuals, ever-married women and children who were recorded in GFHS 1995-1997. Most households are quite large. The average household size ranges

between 6.5 in Bahrain and 8.5 in Oman. Data on the age distribution shows a very young population for the Arab Gulf States and conforms to the pattern observed in high-fertility societies, with a higher proportion of the population in the younger age groups (table 21).

Broadly speaking, the education system in the region has four tiers: primary (6 years), preparatory (3 years), secondary (3 years) and higher education. Among the male population aged 10 years or more, the level of literacy ranges between 84 per cent in Oman and 96 per cent in Kuwait. Women in the region were traditionally less educated than men. Among women at ages 10 and over the percentage literate ranges between 65 in Oman to around 85 in Bahrain and Kuwait (table 22). A recent substantial improvement in educational attainment can be seen in the fact that younger males and females have attained much higher levels than older men. The proportion of men and women with no education declines sharply as we approach the more recent and younger cohorts and is virtually nil among those at ages 10-14 years.

### *Longstanding illness or disability*

The GFHS household health status questionnaire included a module on longstanding illness which was administered to the head of the household, who was asked to respond for all members. From the detailed information gathered in this module on the presence of chronic conditions, an indicator of longstanding illness

TABLE 20. SAMPLE SIZE: NUMBER OF HOUSEHOLDS, WOMEN AND CHILDREN SUCCESSFULLY INTERVIEWED, GFHS-1995-1997

Country	Year of survey	Number of households	Number of persons in household health status interview	Number of ever married women in reproductive health interview	Number of children under 5 years in child health interview
Bahrain	1995	4 122	26 742	3 725	3 169
Kuwait	1996	3 673	25 648	3 453	3 514
Oman	1995	6 103	51 653	6 418	9 033
Saudi Arabia	1996-1997	10 510	73 462	8 894	10 831
United Arab Emirates	1995	5 822	45 830	5 745	6 285

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

TABLE 21. POPULATION COMPOSITION: PER CENT DISTRIBUTION OF THE *DE JURE*  
POPULATION BY BROAD AGE GROUPS, GFHS-1995-1997

Country	Age				Total	Among all females, the per cent aged 15-49	Average household size	Dependency ratio
	<15	15-49	50-64	65+				
Bahrain	37.7	50.6	7.4	4.3	100.0	51.3	6.5	72.4
Kuwait	40.5	48.1	6.3	5.1	100.0	48.6	7.0	83.8
Oman	50.4	39.5	6.9	3.2	100.0	39.9	8.5	115.5
Saudi Arabia	46.6	42.7	7.0	3.7	100.0	44.2	7.0	101.0
United Arab Emirates	45.3	44.6	6.8	3.3	100.0	46.1	7.9	94.5

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

TABLE 22. EDUCATIONAL ATTAINMENT ACCORDING TO SEX, GFHS-1995-1997

Country	Sex	Per cent literate among persons aged 10 or more years	Among persons aged 20-24 the percentage with:	
			Post primary education	Secondary or higher education
Bahrain	Female	84	88	72
	Male	93	82	59
Kuwait	Female	85	98	67
	Male	96	82	59
Oman	Female	65	49	37
	Male	84	64	42
Saudi Arabia	Female	70	80	44
	Male	87	91	42
United Arab Emirates	Female	78	79	59
	Male	88	73	39

Source: See table 21.

or disability was constructed. A person was considered as having a longstanding illness or disability if he/she was reported to have had any of the following doctor-diagnosed conditions: high blood pressure, heart disease, diabetes, stroke, asthma, joint disease, peptic disease, renal disease, liver disease, nervous disease, cancer, or any longstanding condition which prevents or limits his/her participation in activities normal for a person of his/her age.

Around 25 per cent of women and 18 per cent of men, 15 years of age and over, were reported as having a doctor-diagnosed longstanding illness or disability. For both men and

women, the likelihood of having a longstanding illness increased with age. At all ages, women were more likely to be reported as having a longstanding illness than men (table 23). The most frequently reported chronic conditions for both men and women were high blood pressure, diabetes and joint disease. The prevalence of diabetes was slightly higher among women than men (e.g. 35 per cent and 33 per cent among Kuwaiti women and men aged 50 and over). The gender gap in the prevalence of high blood pressure was wider (e.g., 37 per cent and 21 per cent among women and men aged 50 and over), and it increased further in the case of joint disease (e.g., 29 per cent and 10 per

TABLE 23. LONGSTANDING ILLNESS: AMONG PERSONS AGED 15 YEARS AND OVER, THE PERCENTAGE REPORTED TO HAVE DOCTOR-DIAGNOSED LONGSTANDING ILLNESS, BY AGE AND SEX, GFHS-1995-1997

Country	Age							Total
	15-19	20-29	30-39	40-49	50-59	60-69	70+	
Females								
Bahrain	8.1	10.9	25.4	37.5	54.6	58.0	53.4	25.4
Kuwait	7.4	9.9	22.3	41.4	64.3	75.7	66.7	27.1
Oman	8.9	12.0	20.2	31.2	42.7	47.2	51.7	21.4
Saudi Arabia	8.7	13.7	25.8	41.1	56.8	58.8	58.9	25.4
United Arab Emirates	7.7	10.4	23.4	37.3	53.7	58.8	55.8	22.5
Males								
Bahrain	6.8	7.7	16.6	24.7	36.1	42.5	38.9	17.7
Kuwait	8.7	7.9	16.0	28.0	47.5	57.1	62.3	20.1
Oman	5.9	6.0	7.6	15.1	24.8	35.6	46.8	13.1
Saudi Arabia	6.9	7.4	14.9	24.7	36.8	48.2	51.9	18.6
United Arab Emirates	5.5	6.4	13.7	24.0	37.0	44.0	53.8	16.3
Total								
Bahrain	7.4	9.3	21.5	31.9	46.2	50.1	45.3	21.6
Kuwait	8.1	8.9	19.3	35.7	57.8	65.4	64.0	23.7
Oman	7.4	9.0	14.3	22.9	34.2	40.6	49.0	17.2
Saudi Arabia	7.8	10.7	20.8	32.2	47.8	52.1	54.7	22.0
United Arab Emirates	6.6	8.5	19.5	30.5	46.1	49.8	54.7	19.5

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and the **United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

cent among Kuwaiti women and men aged 50 and over, respectively).

#### *Cardiovascular disorder*

Table 24 shows the proportion of persons reported to have ever had a doctor-diagnosed cardiovascular disorder, by age and sex. In this study, a person was classified as having a cardiovascular disorder if he/she was reported in the household health interview to have ever had any of the following conditions confirmed by a doctor: cardiac disease, other heart trouble, stroke, high blood pressure or diabetes. High blood pressure and diabetes were considered to be cardiovascular disorders although these are predisposing conditions rather than cardiovascular disorders *per se*.

Among those aged 15 years and over, the prevalence of cardiovascular disorder was, on average, 10 per cent for men and 12 per cent

for women. At all ages, women were more likely to be reported to have ever had a cardiovascular disorder.

#### *Acute sickness and use of health services*

In the GFHS, a general morbidity module was used to collect information on acute sickness and use of health services during the two weeks before interview. For each member of the household, it was asked whether he/she had had to cut down, in the two weeks preceding the interview, on anything usually done at home, at work, or in free time, because of illness or injury. This could include recurrence or exacerbation of a long-standing illness. For each person reported to have had an acute condition, during the reference period, questions were asked for the number of days of limited activity, use of health services, and use of medication, caused by this condition.

TABLE 24. CARDIOVASCULAR DISEASE: AMONG PERSONS 15 YEARS OF AGE AND OVER,  
THE PERCENTAGE REPORTED TO HAVE EVER HAD A DOCTOR DIAGNOSED  
CARDIOVASCULAR DISORDER, BY AGE AND SEX, GFHS-1995-1997

CARDIOVASCULAR DISORDER, BY AGE AND SEX, GPAS 1995-1997								
Country	Age							Total
	15-19	20-29	30-39	40-49	50-59	60-69	70+	
Females								
Bahrain	1.2	1.8	7.4	18.4	40.4	40.4	37.1	12.1
Kuwait	1.1	2.0	7.8	25.9	51.6	64.9	55.1	16.5
Oman	0.7	3.4	6.0	19.4	28.0	28.0	29.1	9.9
Saudi Arabia	1.0	2.1	7.5	19.4	35.4	37.4	36.1	10.7
United Arab Emirates	0.9	2.3	11.2	23.6	37.5	39.4	39.6	11.9
Males								
Bahrain	0.9	1.3	6.6	15.5	26.2	32.6	23.1	9.3
Kuwait	0.6	1.7	8.4	22.0	41.9	50.3	54.1	13.2
Oman	0.5	0.8	3.2	11.6	18.7	23.9	24.4	6.3
Saudi Arabia	0.4	1.2	5.6	15.8	26.3	35.5	32.9	9.9
United Arab Emirates	0.6	1.4	6.3	18.9	29.8	32.7	34.0	9.7
Total								
Bahrain	1.0	1.5	7.0	16.9	33.9	36.4	29.3	10.7
Kuwait	0.9	1.8	8.1	24.2	47.8	56.8	54.5	14.9
Oman	0.6	2.1	5.7	15.3	23.5	25.7	26.6	8.1
Saudi Arabia	0.7	1.7	6.6	17.4	31.3	36.2	34.2	10.3
United Arab Emirates	0.8	1.9	9.2	21.2	34.0	35.4	36.6	10.8

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

Table 25 shows the proportion of persons reported to have had an acute sickness during the two weeks preceding the interview, and the mean number of days on which activities had been limited for those reported to have had a bout of acute sickness. Overall, 11 per cent of women and 8 per cent of men aged 15 or more years were reported to have had an acute sickness during the past two weeks. Reported prevalence of acute sickness increased only slightly with age up to age 35, and thereafter it increased to higher levels among persons at 35-59 years. At all ages a higher proportion of women than men were reported to have had an acute sickness. The mean number of days of restricted activity associated with acute conditions increased with age for both men and women. The overall mean number of days of acute sickness (5.2 days) was similar for men and women.

For persons who had had an acute sickness in the past two weeks, information was gathered on whether they have consulted with a government doctor, a private doctor, a trained nurse, a pharmacist, a traditional healer, or anyone else. The results are summarized in table 26. It should be noted that multiple response was allowed so that the percentages reporting contact with various health care providers in table 26 do not add to 100. Overall, among persons who had had an acute condition, around two-thirds had consulted a government doctor. The likelihood of consulting a private doctor about an acute condition was lowest in Kuwait (10 per cent) and highest in Oman (24 per cent).

Table 27 shows the percentage of persons with an acute sickness during the two weeks preceding the survey who had taken medicines.

TABLE 25. ACUTE SICKNESS: AMONG PERSONS 15 YEARS OF AGE AND OVER, THE PERCENTAGE REPORTED TO HAVE HAD AN ACUTE SICKNESS, DURING THE TWO WEEKS PRECEDING THE INTERVIEW, GFHS-1995-1997

Country	Percentage of persons 15 years and over reported to have had an acute sickness during the two weeks preceding the survey			Mean number of days of restricted activity among those with acute sickness in past two weeks		
	Men	Women	Total	Men	Women	Total
Bahrain	9.3	12.9	11.1	5.0	5.1	5.1
Kuwait	11.7	15.8	13.8	4.8	5.0	4.9
Oman	6.7	10.2	8.4	5.0	5.2	5.1
Saudi Arabia	6.5	10.2	8.4	5.7	5.3	5.5
United Arab Emirates	4.6	7.3	6.0	5.6	5.5	5.5

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

TABLE 26. USE OF HEALTH SERVICES: AMONG PERSONS 15 YEARS OF AGE AND OVER WHO HAVE HAD AN ACUTE SICKNESS DURING THE TWO WEEKS PRECEDING THE SURVEY, THE PERCENTAGE REPORTED TO HAVE CONSULTED VARIOUS HEALTH CARE PROVIDERS, GFHS-1995-1997

Country	Government doctor	Private doctor	Trained nurse	Pharmacist	Traditional healer	Others
<i>Women</i>						
Bahrain	58.6	13.6	2.3	8.3	3.7	1.7
Kuwait	76.0	11.4	0.2	4.0	2.5	2.4
Oman	65.6	23.4	2.3	1.8	1.2	3.1
Saudi Arabia	64.4	17.9	3.0	7.8	1.7	1.6
United Arab Emirates	61.6	19.1	1.0	3.8	1.0	1.0
<i>Men</i>						
Bahrain	60.0	15.1	2.6	10.1	3.4	2.0
Kuwait	79.3	9.0	0.5	3.7	2.5	2.1
Oman	69.2	24.0	1.9	1.6	1.4	2.5
Saudi Arabia	66.9	17.9	2.4	7.7	1.6	1.3
United Arab Emirates	74.5	19.5	1.2	2.9	1.4	1.6
<i>Total</i>						
Bahrain	59.2	14.2	2.4	9.0	3.6	1.8
Kuwait	77.4	10.4	0.3	3.9	2.5	2.3
Oman	67.1	23.6	2.1	1.7	1.2	2.8
Saudi Arabia	65.4	17.9	2.8	7.8	1.6	1.5
United Arab Emirates	66.4	19.2	1.1	3.5	1.1	1.2

Source: See table 25.

Overall, nine in ten of persons 15 years and over who had had an acute sickness in the past two weeks had taken medication for the condition. Men were slightly more likely than women to use medication.

Table 28 shows the percentage of persons 15 years of age and over reported to have gone to

the dentist at least once in the past 12 months. The results indicate a very low prevalence of dental care. Among persons 15 years or more only one in ten in Oman had gone to the dentist in the last 12 months, compared with one in six in Saudi Arabia and United Arab Emirates, one in five in Bahrain and one in four in Kuwait. Women were more likely to visit the dentist



TABLE 27. MEDICATION: AMONG PERSONS 15 YEARS OF AGE AND OVER WHO HAVE HAD AN ACUTE SICKNESS DURING THE TWO WEEKS PRECEDING THE INTERVIEW, THE PERCENTAGE REPORTED TO HAVE TAKEN MEDICINES, GFHS-1995-1997

Country	Men	Women	Total
Bahrain	88.6	86.1	87.1
Kuwait	94.1	91.2	92.8
Oman	90.3	88.2	89.0
Saudi Arabia	88.3	85.2	86.4
United Arab Emirates	89.3	84.4	86.3

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

TABLE 28. DENTAL CARE: PERCENTAGE OF PERSONS 15 YEARS OF AGE AND OVER WHO HAD GONE TO THE DENTIST AT LEAST ONCE IN THE PAST 12 MONTHS, GFHS-1995-1997

Country	Men	Women	Total
Bahrain	14.0	25.0	19.6
Kuwait	17.6	28.9	23.4
Oman	6.3	14.4	10.3
Saudi Arabia	11.0	21.7	16.4
United Arab Emirates	10.9	21.4	16.3

Source: See table 27.

(22 per cent) than men (12 per cent), with the greatest gender difference occurring during the childbearing years.

In the GFHS, information on smoking was collected from the household head, who was asked a series of questions about smoking habits and behaviour of members of the household. This proxy information is likely to underestimate smoking prevalence, particularly among young adults and women, because of either a lack of knowledge or a reluctance to give information.

The prevalence of smoking is highest in Kuwait and lowest in Oman (table 29). In Kuwait, the reported proportion ever smoking cigarettes was 35 per cent for men and only 2 per cent for women. Men aged 30-49 years showed the highest prevalence of ever smoking cigarettes, the only exception being shown for Bahrain where men aged 50-69 had the highest prevalence of ever smoking.

The reported proportion of men who currently smoked cigarettes increases from 13 per cent in Oman to around 18 per cent in Saudi Arabia and United Arab Emirates, 22 per cent in Bahrain and 30 per cent in Kuwait. The prevalence of current cigarette smoking among men is highest at ages 30-49. About six per cent of men were reported to have stopped smoking cigarettes. The proportion of men who were former regular cigarette smokers increased steadily with age.

### 3. Reproductive health

#### *Marriage patterns*

In the Arab Gulf states, the family is the unit in which reproduction is authorized and expected, and to which the responsibility for child care is assigned. Marriage and fertility are, therefore, viewed as interrelated and as sequential phases in the life cycles of women.

TABLE 29. SMOKING: AMONG PERSONS 15 YEARS OF AGE AND OVER, THE PERCENTAGE WHO EVER SMOKED CIGARETTES, AND THE PERCENTAGE CURRENTLY SMOKING BY AGE AND SEX, GFHS-1995-1997

Country	Ever smoked cigarettes					Currently smoking cigarettes				
	15-19	20-29	30-49	50+	All	15-19	20-29	30-49	50+	All
<i>Men</i>										
Bahrain	8.2	26.9	38.9	47.5	31.2	7.6	24.3	27.3	27.6	22.4
Kuwait	12.1	41.2	44.4	37.2	35.2	11.6	37.2	37.3	25.5	29.6
Oman	4.8	19.1	32.7	18.7	19.0	4.1	16.0	21.8	9.5	13.2
Saudi Arabia	6.0	25.8	34.0	19.7	21.8	5.5	23.6	27.1	12.3	17.5
United Arab Emirates	6.7	27.2	38.1	27.9	24.2	6.2	23.6	28.3	16.0	18.3
<i>Women</i>										
Bahrain	0.6	0.8	2.6	4.9	2.2	0.5	0.6	1.2	1.2	0.9
Kuwait	0.1	0.9	2.2	4.0	1.8	0.1	0.7	1.9	3.0	1.5
Oman	0.1	0.2	0.5	0.9	0.4	0.1	0.2	0.3	0.5	0.2
Saudi Arabia	0.3	0.8	1.3	0.8	0.9	0.2	0.7	1.0	1.0	0.7
United Arab Emirates	0.1	0.4	0.8	1.4	0.6	0.1	0.3	0.6	1.0	0.4
<i>Total</i>										
Bahrain	4.4	13.8	19.9	34.1	16.4	4.1	12.4	13.7	13.8	11.5
Kuwait	6.1	21.5	21.6	19.7	18.0	5.9	19.3	18.1	13.6	15.1
Oman	2.4	9.8	16.2	10.2	9.7	2.1	8.2	10.7	5.2	6.7
Saudi Arabia	3.1	12.6	17.2	11.1	11.3	2.8	11.5	13.5	7.1	9.0
United Arab Emirates	3.4	13.3	17.3	15.2	12.0	3.1	11.6	12.8	8.7	9.1

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

Marriage is almost universal in the Gulf states. Detailed tabulations, however, show substantial decreases over time in the proportions of young marriages and a concomitant tendency for first marriages to become spread over a wider age range. The youngest median age at first marriage is found in Oman and the oldest in Kuwait, a country that seems to have had the oldest marriage pattern even among the oldest cohorts covered.

This important transformation in the tempo of female nuptiality in the Arab Gulf states reflects, of course, an upward shift in age at first marriage. This may be illustrated by an examination of trends in the median age at first marriage, i.e. the age by which 50 per cent of the women of any given cohort had entered into a first marriage. Table 30 shows that the median age at first marriage was achieved before age 20 in the five countries considered

among the generations of women currently aged 40-49, but in only Oman and Saudi Arabia among women currently aged 25-29. Among the younger cohort of women aged 20-24, the median age at first marriage has already increased to above 20 years in all the states covered, with the exception of Oman. It is also of interest to note that in all the countries studied, women currently aged 15-19 years had not attained the level of 50 per cent ever married.

Detailed tabulations from the GFHS (not given here) show substantial differentials in the age at first marriage. These differentials tend to be quite large among younger women, reflecting differences in the delay of marriage among the various subgroups. Marriage is later among women with secondary or higher education, urban residence and those who marry a non-relative.

TABLE 30. AGE AT FIRST MARRIAGE: MEDIAN AGE AT FIRST MARRIAGE FOR SUCCESSIVE FIVE-YEAR AGE COHORTS OF WOMEN UNDER 50 YEARS OF AGE, GFHS-1995-1997

Country	Age of cohort (as of survey date)				
	25-29	30-34	35-39	40-44	45-49
Bahrain	22.9	20.8	18.4	17.7	15.1
Kuwait	21.2	20.5	20.1	18.8	18.2
Oman	14.8	13.9	13.6	13.7	13.5
Saudi Arabia	17.3	15.8	15.1	15.0	15.0
United Arab Emirates	20.0	15.7	15.1	14.7	14.4

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

NOTE: The median age at marriage for women aged 15-19 and 20-24 could not be determined because less than 50 per cent of the women in each cohort have been married, the only exception being women aged 20-24 in Oman who have a median age at marriage of 18.6 years.

The results suggest that the decisions of educated women about when to marry and whom to marry are becoming less influenced by traditional forces that have favoured marriage early and to a relative. Since the mid-1970s, the trend has been towards more schooling for women as well as increased opportunities for employment in the modern sector of the economy. The process of modernization in the Arab Gulf region, thus, seems to be affecting age at marriage not only by increasing the time spent in school but also by changing values about the proper age to enter a marital union. These changes in marriage patterns have decreased marital exposure to childbearing by reducing the proportion of women who are potential teen age mothers. This trend has almost certainly altered the age pattern of births in the Arab Gulf region.

#### *Fertility patterns*

As in most developing countries, fertility in the Arab Gulf region is the most important determinant of changes in the rates of population growth. While information on fertility patterns and levels, for most countries in the region, has been sparse until recently, the general impression has been one of pro-natalist societies in which the average number of births per woman has been and remains high. Results from surveys conducted since the late 1980s, however, convey a picture of declining fertility throughout the region, but also of a striking contrast between the rural and the urban family forma-

tion patterns. Not only is urban residence distinguished from rural residence, but within the urban sector women's education is rapidly becoming associated with the emergence of a new pattern of reproductive preference and practice.

The level and age pattern of current fertility may be compared across the five Gulf states in using age-specific fertility rates and total fertility rates (table 31). The region shows quite a wide range in current fertility with Bahrain showing the lowest rate (3.2 births) and Oman the highest (7.1). The total fertility rate is between four and five in Kuwait and United Arab Emirates, and it rises to 5.7 births in Saudi Arabia.

Most of the countries included show high fertility rates over a broad peak extending over ages from 20 to 34, with maximum fertility occurring at ages 25-29, and with fertility rates in age group 30-34 ranking second to those in the peak group. The exception to this pattern is shown by Bahrain with maximum fertility rates occurring at ages 25-34, and with a fertility rate in age group 35-39 exceeding that in age group 20-24. Fertility rates among adolescents are not as high as they were a decade ago or so, being below 50 births per 1,000 women in three of five countries, and above 80 births per 1,000 women in only Oman.

Trends in fertility can be inferred by comparing the completed fertility of women aged 40-49 at the time of the survey with the level of

TABLE 31. CURRENT FERTILITY: AGE-SPECIFIC FERTILITY RATES (PER 1,000 WOMEN), TOTAL FERTILITY RATE, GENERAL FERTILITY RATE AND CRUDE BIRTH RATE, FOR THE THREE YEARS PRECEDING THE SURVEY, GFHS-1995-1997

Country	Age						
	15-19	20-24	25-29	30-34	35-39	40-44	45-49
Bahrain	16	115	167	157	125	49	20
Kuwait	28	174	223	191	142	56	11
Oman	86	270	332	300	222	114	86
Saudi Arabia	50	201	270	264	191	103	68
United Arab Emirates	41	183	249	222	161	80	42

Country	Total fertility rate (TFR)	General fertility rate (GFR)	Crude birth rate (CBR)
Bahrain	3.24	100	24.0
Kuwait	4.12	116	28.6
Oman	7.05	194	38.8
Saudi Arabia	5.74	157	34.8
United Arab Emirates	4.89	137	31.4

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

NOTE: Rates are for the three-year period preceding the survey, a period covering approximately the calendar years 1992-1995 for the United Arab Emirates, 1993-1995 for Bahrain and Oman, and 1994-1996 for Kuwait and Saudi Arabia

current fertility as measured by the total fertility rate. When fertility remains stable over time, these two indicators will be very similar.

The GFHS results indicate that there has been a significant decline in fertility levels during the past two decades or so in the five countries considered. Declines in the order of from 1.5 to 1.6 children per woman were implied by the data for Kuwait and Oman, and larger declines in the order of 2.1 to 2.5 children per woman were implied by the data for Bahrain and Saudi Arabia. The steepest decline, in the order of 3 children per woman, was estimated for the United Arab Emirates (table 32).

An assessment of recent trends in fertility suggests that the decline in fertility in almost all cases was caused by an increase in age at first marriage, a decline in the propensity of marriage at young ages and a decline in marital fertility at higher ages and longer durations of marriage.

Results from recent surveys in the countries included here all show, to varying degrees, evidence of the existence of certain socio-economic differentials in fertility. The most significant factor is women's educational status. Other social and economic factors are also associated with differentials in marital fertility, although none is as powerful as residence and education.

Urban residence is consistently related to both the tempo and the level of fertility in Oman, Saudi Arabia and United Arab Emirates. The age-specific fertility schedules by current residence (not shown here) are all broadly similar in shape, with a flatter peak, or later tapering-off, among rural women; and the sharpest peak and greatest concentration of childbearing at young ages among women living in urban areas. Detailed tabulations also indicate that in urban areas, later marriage rather than a longer first birth interval accounts for any delay in the onset of childbearing, and lower fertility is produced through a lower

TABLE 32. FERTILITY BY RESIDENCE AND EDUCATION: TOTAL FERTILITY RATE (TFR) FOR THE THREE YEARS PRECEDING THE SURVEY AND THE MEAN NUMBER OF CHILDREN EVER BORN (CEB) TO ALL WOMEN AT AGES 40-49, BY TYPE OF PLACE OF RESIDENCE AND EDUCATION, GFHS-1995-1997

Country	Fertility indicator	Residence	
		Urban	Rural
Bahrain	TFR	3.24	--
	CEB	5.70	--
Kuwait	TFR	4.12	--
	CEB	5.71	--
Oman	TFR	6.60	7.95
	CEB	8.68	8.38
Saudi Arabia	TFR	5.39	6.50
	CEB	7.42	8.65
United Arab Emirates	TFR	4.24	6.57
	CEB	7.56	8.27

Country	Fertility indicator	Education					Total
		Illiterate	Less than primary	Primary	Preparatory	Secondary or more	
Bahrain	TFR	6.92	4.65	3.61	3.12	2.99	3.24
	CEB	7.09	7.49	5.98	4.90	3.50	5.70
Kuwait	TFR	6.13		5.14	4.50	3.48	4.12
	CEB	7.83	6.90	6.03	5.24	4.07	5.71
Oman	TFR	8.61	7.59	7.51	4.75	3.81	7.05
	CEB	8.75	8.39	8.14	5.25	3.95	8.57
Saudi Arabia	TFR	7.38	6.08	5.62	5.94	4.56	5.74
	CEB	8.55	7.71	6.60	5.36	4.51	7.85
United Arab Emirates	TFR	7.34	5.71	5.29	4.54	3.29	4.89
	CEB	8.36	7.53	7.25	6.02	5.11	7.76

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

tempo of fertility after the first few years of marriage and an earlier completion of childbearing.

A uniformly inverse relationship between current levels of fertility and women's educational status is also shown by detailed tabulations for all the countries included, although the size of the differential is greater where substantial decline in urban fertility has occurred.

The extent that variations in fertility were a function of rural or urban residence *per se*, or could equally well be explained by other socioeconomic factors, notably mother's education,

was investigated in a number of studies which confirmed the significance of both residence and mother's education (Omran and El-Khorazaty, 1996; Al-Mazrou, Callum and Farid, 1993). The figures thus indicate earlier completion of childbearing to be a major factor in formation of the smaller "urban/literate" family.

#### Family planning

Three aspects of childbearing have an important effect on maternal health and child survival: the mother's age at maternity, the number of children she has previously born, and the

length of time between births. Of these factors, the birth interval appears to have the greatest impact on both maternal health and child survival. Increasing women's control over their own fertility, therefore, could clearly have a major impact on the health of mothers and the growth and survival of their children. Individual choices about family size, however, are made effective through fertility regulation.

The percentage of ever-married women who had ever used contraception ranged from 35 per cent in Oman to 83 per cent in Bahrain. In two of the five countries shown in table 33, about three quarters or more of women have ever-used contraception. Ever use of contraceptives, in contrast to knowledge, varied substantially between socio-economic groups. Urban residence and higher levels of education are the characteristics most associated with ever use.

Current contraceptive use among currently married women was highest in Bahrain and Kuwait (62 and 50 per cent, respectively), followed by Saudi Arabia and United Arab Emirates with prevalence rates in the range 27-32 per cent, and Oman which shows the lowest prevalence rate of 24 per cent.

The position of Bahrain and Kuwait, however, changes when the level of current use of only modern methods is examined. In Bahrain, contraceptive prevalence drops from 62 per cent for "any" method to only 31 per cent for

"any modern" method. Withdrawal is by far the most commonly used method in Bahrain, and it accounts for 43 per cent of total contraceptive prevalence. As may be seen from table 33, Kuwait shows the highest rate of use of modern methods (41 per cent), following by Bahrain and Saudi Arabia with rates in the range 29-32 per cent, and by Oman and United Arab Emirates with rates in the range 18-24 per cent.

In the five countries considered, the pill is by far the most widely used method, followed by the IUD. Use of other modern methods is negligible.

Detailed tabulations also show that education was closely associated with contraceptive use. Thus, contraceptive prevalence increased dramatically as women's education rose. In Saudi Arabia, for example, contraceptive use increases from 19 per cent among illiterate women, to 30 and 34 per cent for women with incomplete primary and primary education, respectively, and to 39 per cent for women with secondary or higher education. The results also indicate that for women of higher educational attainment and women residing in major urban areas, contraception now appears to be a means of birth-spacing as well as family limitation. In fact, there are indications that the "urban/literate" women are leading the transition to a new reproductive pattern in the region.

TABLE 33. KNOWLEDGE, EVER USE AND CURRENT USE OF FAMILY PLANNING: AMONG WOMEN UNDER 50 YEARS OF AGE, THE PERCENTAGE OF EVER-MARRIED WOMEN WHO KNOW A FAMILY PLANNING METHOD, AND THE PERCENTAGE OF CURRENTLY MARRIED WOMEN WHO CURRENTLY USE A METHOD, GFHS-1995-1997

Country	Per cent who know a method	Per cent who ever used a method		Per cent who currently use a method	
		Any method	Any modern method	Any method	Any modern method
Bahrain	99.6	83.2	64.1	62.1	31.4
Kuwait	96.7	72.9	67.6	50.3	40.9
Oman	96.8	35.3	28.0	23.8	18.3
Saudi Arabia	91.3	54.3	51.3	31.7	28.5
United Arab Emirates	86.1	47.3	44.5	27.6	23.6

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

## Maternity care

Antenatal coverage is almost universal in the region (table 34). Almost all of the mothers who received antenatal care in the past three years had at least two check-ups, and between 80 and 95 per cent had five or more check-ups. Virtually all mothers who received antenatal care were seen by a doctor or trained nurse. Public health facilities were the principal provider of care. The proportion of mothers who sought antenatal care because of health problems associated with the pregnancy ranged between 24 per cent in Bahrain and United Arab Emirates and around 30 per cent in Kuwait and Saudi Arabia. This proportion was somewhat higher among mothers with some education than among those with no education. This pattern may denote a greater recognition of the risks associated with pregnancy on the part of the better educated women, and greater ability to make full use of the maternal health care services available.

Almost all children in Bahrain and Kuwait, and 9 in 10 children in Oman, Saudi Arabia

and United Arab Emirates are born at a health facility. A greater utilization of health care facilities for pregnancy check-ups than for deliveries in Oman and United Arab Emirates is thus indicated. However, almost all deliveries in the three-year period preceding the survey were assisted by a doctor or trained nurse.

The level of postnatal care in the region is rather low. Among women who had given birth in the past three years, the proportion receiving a postnatal check-up was above the 50 per cent level in only two countries, Bahrain (62 per cent) and Oman (74 per cent). It would thus seem that the attention given to prenatal care and delivery, particularly by educated mothers, is not followed through in respect of the mother's complete physical and nutritional recovery.

## Pregnancy complications

One of the major objectives of the GFHS was to quantify how frequently women experience danger signs and symptoms of major complica-

TABLE 34. MATERNITY CARE INDICATORS: FOR ALL BIRTHS IN THE THREE YEARS PRECEDING THE SURVEY, THE PERCENTAGE OF RESPONDENTS WHO REPORTED HAVING RECEIVED ANTENATAL, DELIVERY AND POSTNATAL CARE, GFHS-1995-1997

Country	Antenatal				
	Per cent receiving antenatal care	Per cent receiving antenatal care from a public health facility	Per cent receiving less than 5 checkups	Per cent seeking care because of pregnancy problems	Per cent receiving tetanus toxoid injection
Bahrain	97.1	86.4	8.9	23.5	53.1
Kuwait	95.0	69.8	11.5	29.0	33.4
Oman	98.2	96.4	20.6	26.3	73.6
Saudi Arabia	90.0	81.9	20.9	29.7	53.8
United Arab Emirates	96.8	87.3	14.6	24.4	N.A.

Country	Delivery		Postnatal	
	Per cent of deliveries taking place in a health facility	Per cent of medically assisted deliveries	Per cent receiving postnatal care	Number of births
Bahrain	98.3	98.4	61.9	1,940
Kuwait	97.5	98.2	34.5	2,132
Oman	88.7	91.1	73.8	5,452
Saudi Arabia	91.0	91.4	45.0	6,665
United Arab Emirates	88.6	99.2	42.4	4,317

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

tions in the antenatal, intrapartum, and postpartum periods. Danger signs include any vaginal bleeding, severe headache, dizziness or blurred vision, generalized oedema, convulsions, breathlessness and tiredness, labour pains for more than 12 hours, excessive bleeding in labour or after delivery, ruptured membranes without labour for more than twelve hours, and fever with or without vaginal discharge after delivery. Such information is vital in planning for the services necessary for reducing risk factors and complications during pregnancy, labour, delivery, and after delivery, and for improving the health of the mother and of her child.

Vaginal bleeding in pregnancy is usually an indication of a problem. However, causes of bleeding during pregnancy vary depending on length of gestation. In early pregnancy, bleeding may be due to a threatened or inevitable miscarriage. Vaginal bleeding in the third trimester may be an indication of life-threatening placental abruption or placental praevia. The question asked in the survey referred to "heavy" vaginal bleeding but did not differentiate the above-mentioned types of bleeding.

Table 35 shows that in 6 to 10 per cent of the live births during the past three years, the respondent said she experienced heavy vaginal bleeding. Detailed tabulations show a steady increase in the reported level of heavy bleeding as maternal education rises. Women with secondary or higher education were twice as likely as illiterate women to report heavy bleeding during pregnancy.

Pre-eclampsia/eclampsia is a major cause of maternal and perinatal mortality. Elevated blood pressure in the last trimester is a major sign of pre-eclampsia. High blood pressure with any of the symptoms of severe headache, generalized oedema or pain in the upper abdomen would strongly indicate pre-eclampsia.

The percentage of the live births in the past three years for which the respondent said she experienced high blood pressure during pregnancy increased from around 6 per cent in Oman and Saudi Arabia, to 8 per cent in Bahrain and United Arab Emirates, and 11 per cent in Kuwait. Respondents under 20, or 35 years of age and older, were more likely to report elevated blood pressure during pregnancy.

In 9 to 20 per cent of the live births in the past three years the mother reported the presence of swelling and/or severe headache, whereas upper abdominal pain was reported in nearly one in six cases. These symptoms of pre-eclampsia, particularly upper abdominal pain, were more likely to be reported by mothers under 35 years of age than by older mothers.

Convulsions or fits during pregnancy may indicate eclampsia; if they have also occurred outside pregnancy it may indicate epilepsy. More than three per cent of mothers reported having had convulsions during pregnancy with one or more of the live births they had in the past three years. Younger mothers with some education were more likely to report convulsions than older women with no education.

TABLE 35. SYMPTOMS OF COMPLICATIONS DURING PREGNANCY: FOR LIVE BIRTHS IN THE LAST THREE YEARS, THE PERCENTAGE OF RESPONDENTS WHO REPORTED PRESENCE OF SPECIFIED SYMPTOMS DURING PREGNANCY, GFHS-1995-1997

Country	Vaginal bleeding	High blood pressure	Swelling	Severe headache	Convulsions	Pain in upper abdomen	Breathlessness	Number of births
Bahrain	9.8	8.2	15.6	14.2	3.5	18.7	27.1	1 940
Kuwait	8.1	10.7	20.3	16.3	3.6	20.7	30.9	2 132
Oman	5.4	5.4	11.3	11.2	2.7	13.4	23.7	5 452
Saudi Arabia	5.5	5.7	9.0	15.0	0.9	14.1	30.3	6 665
United Arab Emirates	6.0	7.6	11.3	12.9	4.1	17.7	28.8	4 317

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).



Anaemia is a common and serious problem in pregnancy; its prevention at the time of delivery is one of the major goals of antenatal care. The figures in table 35 indicate that anaemia was the most common problem reported in the antenatal period. In a quarter to one-third of the live births in the past three years, the respondent said she experienced severe breathlessness, during pregnancy, such that she was only able to walk a few steps before stopping. There is little variation in the prevalence of this symptom of severe anaemia according to background characteristics.

#### *Delivery complications*

It has been shown that between 91 and 98 per cent of all deliveries in the past three years were medically assisted. With such an impressive level of care, it is to be expected that the majority of women experience no problems or complications once labour has started. Sometimes, however, problems do arise that require skilled management and care. In the GFHS, information was gathered on the presence of complications during delivery, the frequency of caesarean sections, and the frequency of preterm deliveries.

Between 74 and 89 per cent of respondents giving birth in the three years preceding the survey reported that the delivery was normal, while the remaining 11-27 per cent experienced problems/complications with delivery (table 36).

The proportion of deliveries associated with complications varied from around 18 per cent for women under 35 years of age, to 26 per cent for women in their forties. The reported presence of delivery complications was higher for urban mothers than rural mothers and higher for women with secondary or higher education than for those with less than primary education.

The proportion of deliveries in the three years preceding the survey that were performed by caesarean section varied from 7 per cent in Oman to 16 per cent in Bahrain. The frequency of caesarean sections was higher among women aged 35 or older. Surgical delivery was more common among the most educated than among women with secondary or less education, indicating the importance of service access.

Around 12 per cent of all live births in Bahrain and Kuwait in the past three years were reported to have been born before their due dates. The prevalence of preterm delivery was lower in Oman, Saudi Arabia and United Arab Emirates, ranging between 5 and 8 per cent.

#### *Postpartum complications*

In the GFHS, information was gathered on three serious complications that can develop in the postpartum period: haemorrhage, eclampsia, and infection.

TABLE 36. DELIVERY COMPLICATIONS: FOR LIVE BIRTHS IN THE THREE YEARS PRECEDING THE SURVEY, THE PERCENTAGE OF RESPONDENTS WHO REPORTED DELIVERY COMPLICATIONS, THE PERCENTAGE OF DELIVERIES BY CAESAREAN SECTION, AND THE PERCENTAGE WHO REPORTED PRETERM LABOR, GFHS-1995-1997

Country	Delivery characteristic			Number of births
	Delivery complications	Delivery by C-section	Preterm delivery	
Bahrain	20.4	16.1	12.7	1 940
Kuwait	23.6	11.2	11.5	2 132
Oman	11.6	6.6	4.5	5 452
Saudi Arabia	26.7	8.1	6.2	6 665
United Arab Emirates	15.8	9.6	8.3	4 317

Source: Bahrain: Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); Kuwait: Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); Oman: Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); Saudi Arabia: Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and the United Arab Emirates: Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

Postpartum haemorrhage is always a serious problem. It can be due to a number of causes: retained placenta, weak uterus, rupture of the uterus, or perineal tear. Between 4 per cent (in Kuwait) and 9 per cent (in Oman), of the respondents giving birth in the past three years reported having had severe bleeding after the delivery of at least one of those births (table 37). The frequency of heavy bleeding immediately following delivery was highest among women under 20 or above 40 years, whereas the likelihood of haemorrhage happening as late as more than 7 days postpartum was highest among teenage mothers. The frequency of postpartum haemorrhage showed little variation by background characteristics.

Eclampsia, in which a woman has fits or convulsions and loses consciousness, is a life-threatening complication for both mother and child. It can occur late in pregnancy, during delivery, or shortly after the baby is born. As indicated earlier, around three per cent of respondents reported having had convulsions during pregnancy. The figures in table 37 show that between 2 and 4 per cent of respondents giving birth in the past three years reported having had convulsions during the puerperium in at least one of those births. The frequency of

postpartum convulsions was highest in young women.

Infection is one of the most serious complications that can develop in the postpartum period. Fever is the most easily diagnosable sign of infection. In 12 per cent of the births in the past three years in Bahrain and Oman, 9 per cent in United Arab Emirates, and 6 per cent in Kuwait and Saudi Arabia, the respondent said she experienced a very high fever in the postpartum period (table 38).

On average, fever was associated with severe lower back pain in five per cent of cases, with severe upper back pain in three per cent of cases, and with pain on passing urine in nearly two per cent of cases, indicating possible inflammation of kidney and renal pelvis (pyelonephritis). In more than four per cent of cases, the respondent said the fever was associated with severe lower abdominal pain, indicating possible sepsis. Finally, fever was associated with a painful breast in around four per cent of cases, indicating possible abscess or mastitis. The reported frequency of each of these symptoms was higher among literate than illiterate respondents. Other background characteristics showed little variation.

TABLE 37. POSTPARTUM HAEMORRHAGE AND CONVULSIONS: FOR LIVE BIRTHS IN THE THREE YEARS PRECEDING THE SURVEY, THE PER CENT DISTRIBUTION BY REPORTED PATTERN OF POSTPARTUM HAEMORRHAGE, AND THE PERCENTAGE OF RESPONDENTS WHO REPORTED HAVING HAD POSTPARTUM CONVULSIONS, GFHS-1995-1997

Country	Symptom					Post-partum convulsions	Number of births
	Excessive bleeding following delivery						
	within 24 hours	between 1-7 days	more than 7 days	no bleeding	Total		
Bahrain	3.6	1.6	1.6	93.2	100.0	3.8	1 940
Kuwait	2.4	1.1	0.8	95.7	100.0	2.1	2 132
Oman	4.1	1.7	3.3	90.9	100.0	2.8	5 452
Saudi Arabia	3.6	2.4	1.3	92.7	100.0	1.5	6 665
United Arab Emirates	2.5	1.4	2.3	93.8	100.0	2.7	4 317

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

TABLE 38. POSTPARTUM SYMPTOMS OF INFECTION: FOR LIVE BIRTHS IN THE LAST THREE YEARS, THE PERCENTAGE OF RESPONDENTS WHO REPORTED HAVING HAD VERY HIGH FEVER IN THE POSTPARTUM PERIOD, ACCORDING TO SPECIFIED SYMPTOMS OF COMPLICATIONS, GFHS-1995-1997

Country	Very high fever associated with:						Number of births
	Very high fever	Severe lower back pain	Severe upper back pain	Lower abdominal pain	Painful urination	Swollen, painful breasts	
Bahrain	12.1	5.1	2.8	4.2	2.6	7.9	1 940
Kuwait	5.4	2.0	1.0	2.6	1.2	3.6	2 132
Oman	12.1	7.2	4.8	5.4	2.2	3.4	5 452
Saudi Arabia	5.8	2.9	1.6	2.9	1.3	2.9	6 665
United Arab Emirates	8.9	4.6	3.1	4.6	1.5	4.4	4 317

Source: **Bahrain:** Yacoub, I., T. Naseeb and S. Farid, eds., *Bahrain Family Health Survey 1995, Preliminary Report* (Manama, Ministry of Health, 1996); **Kuwait:** Al-Rashoud, R. and S. Farid, eds., *Kuwait Family Health Survey 1996, Preliminary Report* (Kuwait, Ministry of Health, 1997); **Oman:** Sulaiman, A. J. A. Al-Riyami and S. Farid, eds., *Oman Family Health Survey, Preliminary Report* (Muscat, Ministry of Health, 1996); **Saudi Arabia:** Khoja, T. and S. Farid, eds., *Saudi Arabia Family Health Survey, Preliminary Report* (Riyadh, Ministry of Health, 1997); and **the United Arab Emirates:** Al-Qassimi, S., M. Fikri and S. Farid, eds., *United Arab Emirates Family Health Survey, Preliminary Report* (Abu Dhabi, Ministry of Health, 1996).

## E. CONCLUDING REMARKS

Household health interview surveys are the most important tool for assessing certain dimensions of health. They are population-based and, therefore, represent all subgroups of the population, including the non-users of health services. The many other advantages of household surveys render them indispensable for health monitoring. It is encouraging to see that relatively small national sample surveys in the Arab Gulf states can provide such a depth as well a breadth of health information in these countries. An international effort to produce structured information on household health survey methodology and recommended measurement instruments would enhance the value of survey results at the national level and contribute to better international comparability.

## REFERENCES

- Al-Mazrou, Y., C. Callum and S. Farid (1993). Differentials in marital fertility. In *Reproductive Patterns and Child Survival in Saudi Arabia*, Y. Al-Mazrou and S. Farid, eds. Riyadh: Ministry of Health, pp. 53-96.
- Al-Rashoud, R., and S. Farid, eds. (1997). *Kuwait Family Health Survey 1996, Preliminary Report*. Kuwait: Ministry of Health.
- Al-Qassimi, S., M. Fikri and S. Farid, eds. (1996). *United Arab Emirates Family Health Survey 1995, Preliminary Report*. Abu Dhabi: Ministry of Health.
- Barnum, H. (1987). Evaluating healthy days of life gained from health projects. *Social Science and Medicine* (Oxford, United Kingdom), vol. 24, pp. 833-841.
- Caplan, A. L., H. T. Engelhardt and J. McCartney, eds. (1981). *Concepts of Health and Disease: Interdisciplinary Perspectives*. Reading, Mass: Addison-Wesley.
- Carlson, B. A. (1985). The potential of national household survey programmes for monitoring and evaluating primary health care in developing countries. *World Health Statistics Quarterly* (Geneva), vol. 38, pp. 38-64.
- Feachem, R., W. Graham and I. Timæus (1989). Identifying health problems and health research priorities in developing countries. *Journal of Tropical Medicine and Hygiene* (London), vol. 92, pp. 133-191.
- Hansluwka, H.E. (1987). Measuring the health status of a population: current state of the art. *Population Bulletin of the United Nations* (New York), Nos. 23/24, pp. 56-75.
- Khoja, T., and S. Farid, eds. (1997). *Saudi Arabia Family Health Survey, Preliminary Report*. Riyadh: Ministry of Health.
- McDowell, I., and C. Newell (1987). *Measuring Health: A Guide to Rating Scales and Questionnaires*. New York: Oxford University Press.
- Mumaghan, J. H. (1981). Health indicators and information systems for the year 2000. *Annual Review of Public Health* (Palo Alto, California), vol. 2, pp. 299-361.
- Omran, M., and N. El-Khorazaty (1996). Socio-economic correlates of fertility. In *Reproductive Patterns and Child Survival in the United Arab Emirates*, S. Al-Qassimi and S. Farid, eds. Abu Dhabi: Ministry of Health, pp. 52-71.
- Sulaiman, A. J., A. Al-Riyami and S. Farid, eds. (1996). *Oman Family Health Survey, Preliminary Report*. Muscat: Ministry of Health.
- Timæus, I., T. Harpham, M. Price and L. Gilson (1988). Health surveys in developing countries: the objectives and design of an international programme. *Social Science and Medicine* (Oxford, United Kingdom), vol. 27, pp. 359-368.
- United Nations (1983). *Manual X. Indirect Techniques for Demographic Estimation*. United Nations: Sales No.E.83.XIII.2.
- \_\_\_\_\_. (1995). *Guidelines for Household Surveys on Health*. Statistical Division, New York: United Nations.
- Ware, J. E., R. H. Brook, A. Davies and K. N. Lohr (1981). Choosing measures of health status for individuals in gen-

- eral populations. *American Journal of Public Health* (Washington D.C.), vol. 71, pp. 620-625.
- White, K. L. (1985). Health surveys: who, why and what? *World Health Statistics Quarterly* (Geneva), vol. 38, pp. 2-14.
- Wilson, R., and T. F. Drury (1984). Interpreting trends in illness and disability: health statistics and health status. *American Journal of Public Health* (Washington D.C.), vol. 5, pp. 83-106.
- World Health Organization (1981). *Development of Indicators for Monitoring Progress Towards Health for All by the Year 2000*. Geneva: World Health Organization.
- \_\_\_\_\_ (1996). *Equity in Health and Health Care*. Geneva: World Health Organization.
- Yacoub, I., T. Naseeb and S. Farid, eds. (1996). *Bahrain Family Health Survey 1995, Preliminary report*. Manama: Ministry of Health.

## IX. HEALTH AND MORTALITY TRENDS IN COUNTRIES WITH ECONOMIES IN TRANSITION

*Martin Bobak\**

### A. MORTALITY IN COUNTRIES WITH ECONOMIES IN TRANSITION

After the political changes in 1989, the countries of central and Eastern Europe (CCEE) and the former Soviet Union (FSU) entered a period of economic transition. The transition was accompanied by declines in living standards that were documented by a wide range of social and economic indicators (United Nations Children's Fund, 1997). Mortality increased in several of these countries. The deterioration of mortality was most pronounced in Russia where life expectancy declined by more than 6.7 years in men and by 3.4 years in women between 1989 and 1994 (Shkolnikov, 1997). Some authors have called the post-1989 mortality developments acute mortality, population, or demographic crises, mostly ascribed to the political, social and economic changes (United Nations Children's Fund, 1994; Heleniak, 1995; Eberstadt, 1994; Eberstadt, 1993).

The beginning of the economic transition in the region may indeed have produced an economic shock that affected mortality. Many macroeconomic or social indicators indeed deteriorated after 1989. We would argue, however, that this process needs to be analysed in a broader context.

During the 1950s and 1960s, health, as measured by life expectancy, improved throughout much of Europe. This improvement had little respect for political boundaries or ideologies. However, in the late 1960s and 1970s this situation began to change. For the most part, life expectancy continued to improve in the countries of Western Europe, but it failed to improve

or even declined, in the CCEE and the FSU. By 1989, there already was a striking gap in life expectancy between East and West. What was notable about this trend was the similarity within the CCEE, except for Yugoslavia and Albania. After 1989, differences between individual countries in the region became more evident.

This paper has two objectives. First, it will present the most recent mortality figures and will place the recent trends in their longer-term context. Secondly, it will briefly discuss the causes for the poor health status in the region. When not stated otherwise, data used are those reported by the countries to the World Health Organization and made available through WHO Health for All database (WHO Regional Office for Europe, 1997).

#### 1. *Mortality trends pre- and post-1989*

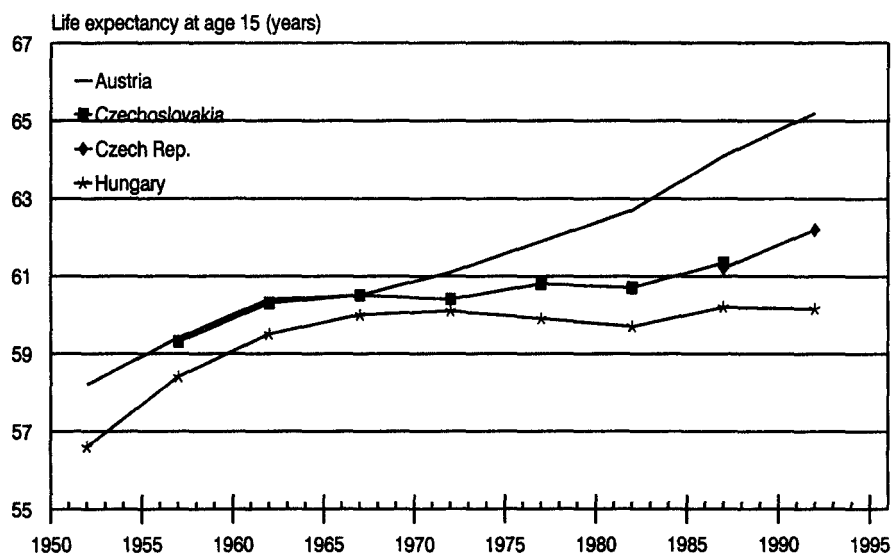
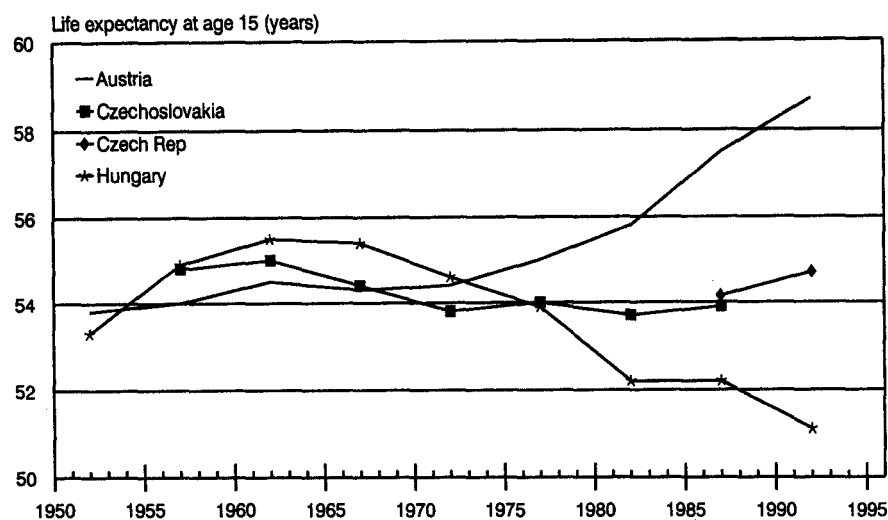
Figure 45 shows "smoothed" trends (in 5-year averages) of male and female life expectancy at age 15 in Austria, Czechoslovakia and Hungary, 3 countries, which until 1918 were part of one state, the Austro-Hungarian Empire. Life expectancy at age 15 is shown here because it better reflects mortality at middle ages, which contributes most the East-West gap (Bobak and Marmot, 1996a). However, trends in life expectancy at birth are similar.

During the 1950s and early 1960s, life expectancy at age 15 was similar in all 3 countries. In the mid 1960s, the trends began to diverge. Life expectancy for both sexes improved in Austria (as it did elsewhere in Western Europe), but declined in men and failed to improve in women in the socialist countries. The fall in life expectancy was particularly dramatic for Hungarian men. This divergence is representative of the overall East-West mortality gap.

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\*Department of Epidemiology and Public Health, University College London Medical School, London, United Kingdom.

Figure 45. Life expectancy at age 15 in Austria, Hungary and Czechoslovakia 1950-1994



Source: World Health Organization, Regional Office for Europe, *Health for All Database* (Geneva, WHO, 1997).

Figure 46 shows life expectancy at age 15 since 1970 for a wider range of countries and allows a more detailed examination of the changes after 1989. In this period, 1970-1989, life expectancy declined in a broadly similar way in most CCEE, with the exception of stagnating trends in the former German Democratic Republic (GDR) and the former Czechoslovakia. In this perspective, the year 1990, the first "post-communist" year, does not show a major change. In fact, life expectancy dropped only slightly in this year in the former GDR and Czechoslovakia. In Bulgaria, Hungary and Romania, life expectancy continued its long-term decline with no clear break in the trend in 1989. It is also interesting to observe that soon thereafter life expectancy started the rise in some countries: first, in 1991, in GDR and Czechoslovakia, in 1992 in Poland, and in 1994 in Hungary.

Trends in life expectancy at age 15 in the FSU share with the CCEE the prolonged decline since the 1970s (figure 47). Estimates of Russian life expectancy at birth, recently reconstructed by Shkolnikov and others (Shkolnikov, Mesle, and Vallin, 1995), show that the decline actually started in the mid-1960s. Since the mid-1980s, however, levels in the FSU differ dramatically from those in the CCEE: there was a sharp rise in life expectancy between 1984 and 1987, followed by a steep fall after 1989. Between 1989 and 1994, Russian male life expectancy at age 15 dropped by more than 7 years. Most of the decline was due to external causes and cardiovascular deaths. It has been estimated that the increase in mortality in 1990-1994 is equivalent to more than 2 millions extra deaths above the expected number if the trends since 1970 had continued (Shkolnikov, 1997). An increase of this magnitude is unprecedented in countries that collect mortality statistics. These trends were shared, to a varying extent, by all countries of the FSU for which reliable data exist, and occurred in both genders.

In all comparisons, differences in life expectancy (and mortality) between Eastern and Western Europe are substantially larger in males than in females. It is interesting that the difference between female and male life expectancy in individual countries is strongly related to overall life expectancy - the longer the life

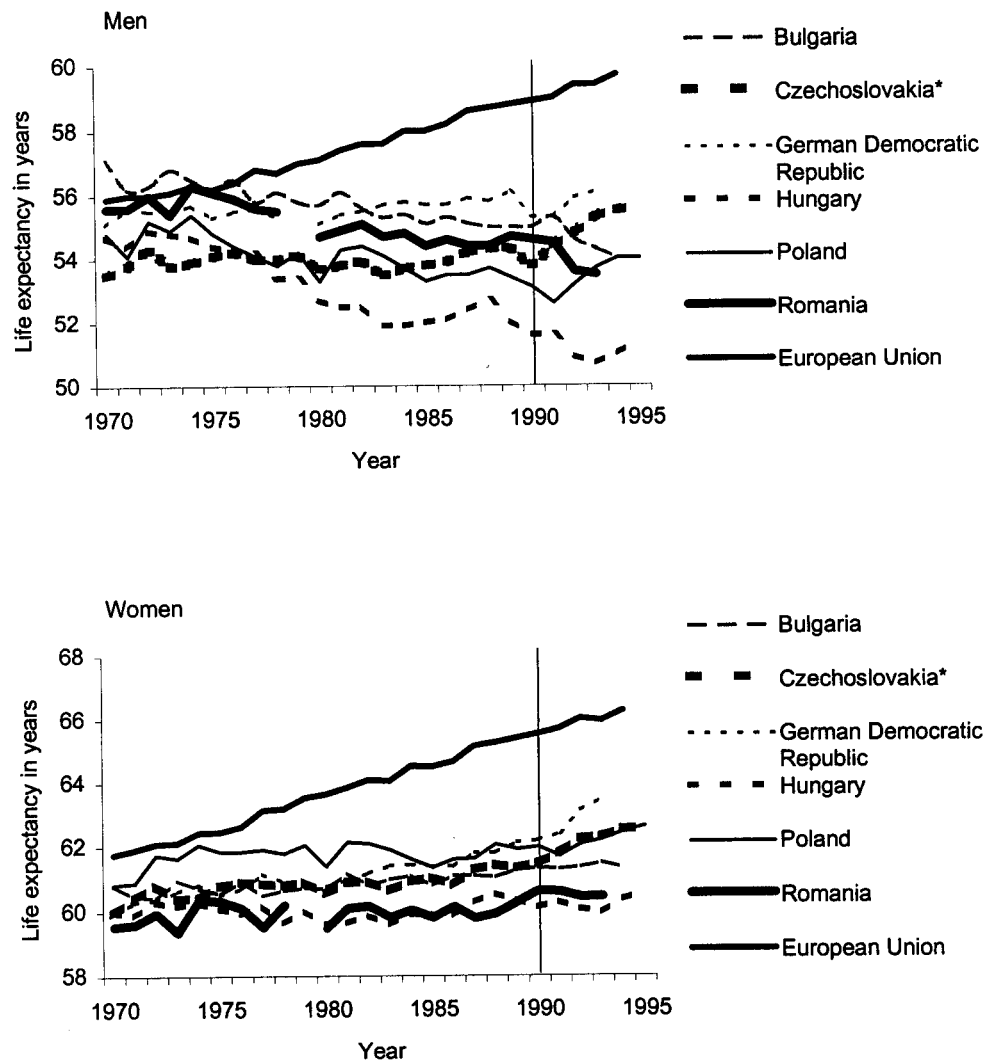
expectancy, the smaller the sex difference; again, the East-West difference is evident (figure 48) (Bobak and Marmot, 1996b). In Russia, with the shortest overall life expectancy, life expectancy gap between women and men was an astonishing 13.6 years in 1994, the largest in the world (Heleniak, 1995). The explanation for this difference is not obvious but it suggests that women were less affected by whatever were the causes of high mortality in the CCEE.

Examination of trends in infant mortality can help to assess the impact of the changing socio-economic circumstances in the CCEE and the FSU. As infant mortality has been shown to be sensitive to environmental factors, particularly to material deprivation (Margolis, Greenberg, Keyes and others, 1992; Leon, Vagero, and Otterblad Olausson, 1992), one would expect that this indicator would reflect best any effects of socio-economic changes. In a longer-term perspective, infant mortality declined in all countries in roughly similar degree (figure 49).

Figures 50 and 51 show the recent trends in infant mortality in more detail. In the CCEE, infant mortality, with the possible exception of Bulgaria, did not show a major increase after the political changes (figure 50). In the FSU, on the other hand, available data do suggest an increase over the last few years (figure 51). Trends in the individual components of infant mortality, early neonatal, neonatal and post-neonatal mortality, closely resemble total infant mortality. They are only available for the CCEE and are not shown here. Although the data on infant mortality from the FSU may not be fully reliable (Anderson and Silver, 1986), the increases they show in the early 1990s are consistent with the adult mortality shown above.

This overview of mortality trends suggests that if there was a mortality crisis related to post 1989 developments, it occurred only (or predominantly) in the FSU, over and above the long-term but slower decline in life expectancy in the pre-1990 period. In the CCEE the mortality crisis is not new but is rather a continuation of the unfavourable trends of the previous two or three decades. Ideally, therefore, in the CCEE we should seek explanations for the medium term (say, post 1970) decline in life

**Figure 46. Trends in life expectancy by sex at age 15 in central and eastern Europe since 1970**

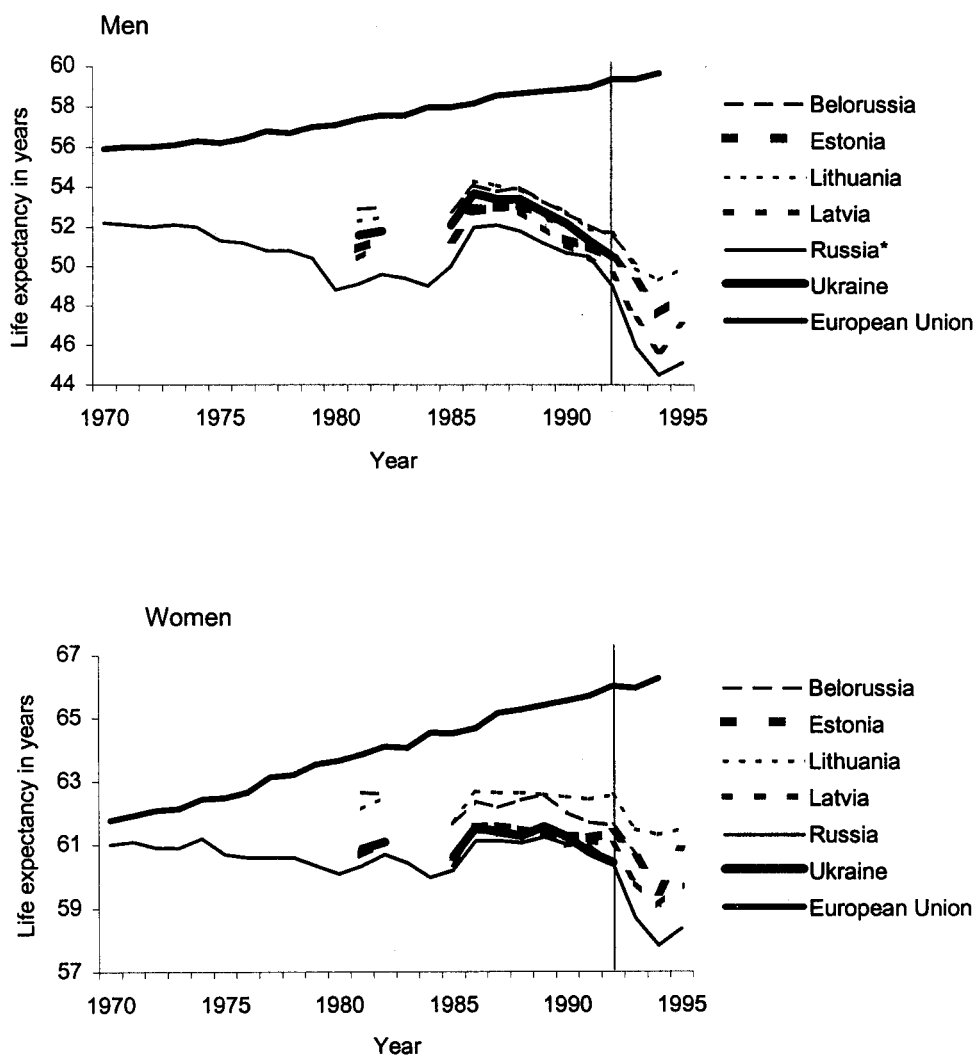


\*Czechoslovakia until 1985, Czech Republic since 1986

Source: World Health Organization, Region Office for Europe, *Health for All Database* (Geneva, WHO, 1997).



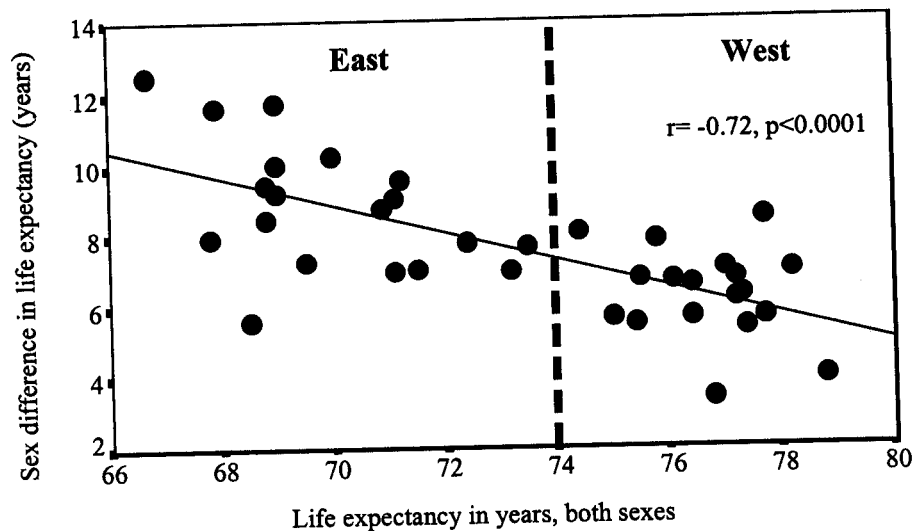
Figure 47. Trends in life expectancy by sex at age 15 in post-Soviet countries since 1970



\*Soviet Union until 1979, Russia since 1980

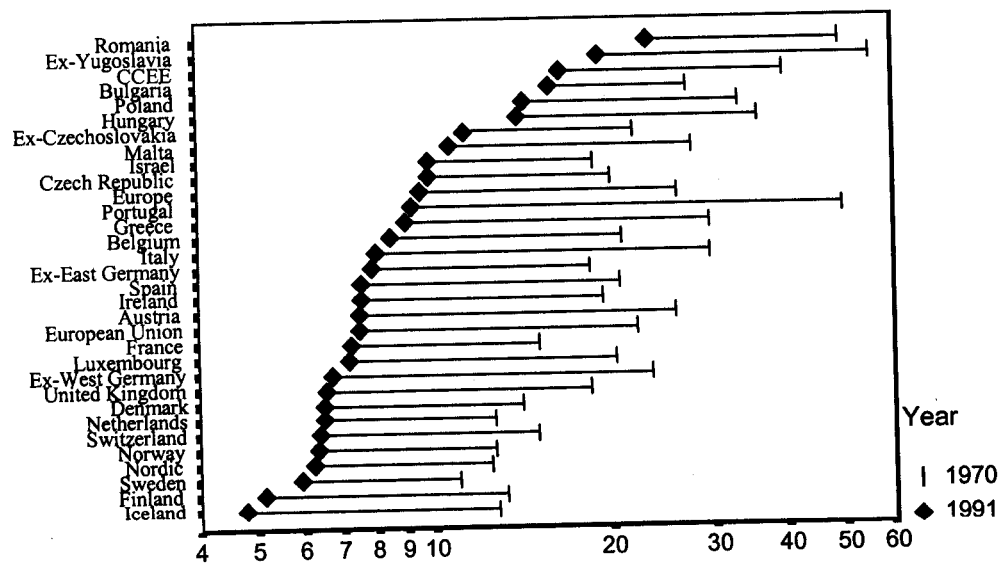
Source: World Health Organization, Region Office for Europe, *Health for All Database* (Geneva, WHO, 1997).

**Figure 48. Sex difference in life expectancy at birth by the overall life expectancy, Europe, 1990-1992**



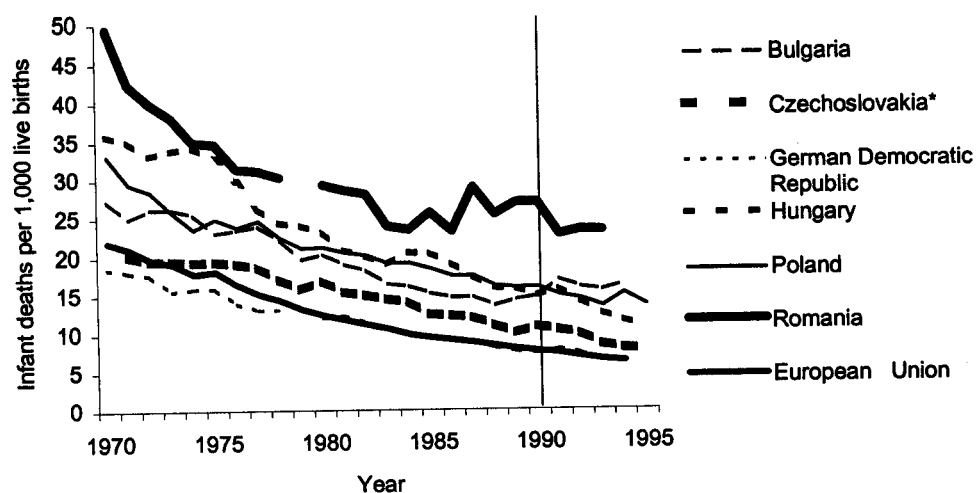
Source: World Health Organization, Regional Office for Europe, *Health for All Database* (Geneva, WHO, 1997).

**Figure 49. Infant mortality in 1970 and 1991, per 1,000 live births (log scale)**



Source: See figure 48.

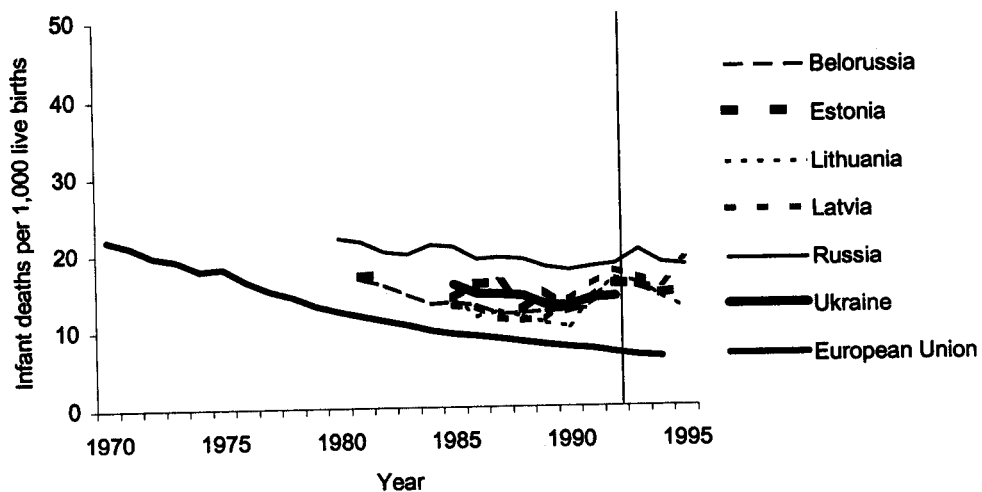
**Figure 50. Infant mortality in central and eastern Europe**



\*Czechoslovakia until 1985, Czech Republic since 1986

Source: World Health Organization, Region Office for Europe, *Health for All Database* (Geneva, WHO, 1997).

**Figure 51. Infant mortality in post-Soviet countries**



Source: See figure 50.

expectancy, not simply for the post-1989 changes, while in the FSU explanations are needed for both the long term and the short term changes, recognising that they may have partly similar causes.

## *2. Causes of death responsible for the high mortality in Central and Eastern Europe*

Calculations by WHO have shown that of the 6.06 year gap in life expectancy between CCEE/FSU and Western Europe, nearly 3.3 years were due to cardiovascular diseases and 1.4 years to external causes of death (Bobak and Marmot, 1996a). The largest part of the gap appears in the age group 35-64 (43 per cent) but almost a quarter (23 per cent) in those aged 65 and more. Mortality in infancy, on the other hand, contributed only 15 per cent of the gap. Virtually identical results were obtained when death rates were compared between the former Soviet Union and the United States (Kingade and Boyle Torrey, 1992), former Eastern and Western Germany (Chenet, McKee, Hort, Brand, and Bojan, 1995) or several central European countries (Chenet, McKee, Fulop, Bojan, Brand, and Kalbarczyk, 1995).

### **B. HEALTH STATUS IN COUNTRIES WITH ECONOMIES IN TRANSITION**

Thus far, only data on mortality have been considered. However, there is abundant evidence that rates of ill health that did not necessarily lead to death are also high in CCEE and FSU. Only a few examples will be given here.

#### *1. Cardiovascular morbidity*

Cardiovascular diseases constitute a major part of the East-West mortality difference (Bobak and Marmot, 1996a). The question arises whether the high mortality is due to high incidence or high case-fatality. The WHO MONICA Project, an international study on cardiovascular disease and its risk factors, provides an answer. That Project has assembled data on cardiovascular mortality and morbidity and surveyed risk factors in a wide range of countries. Routine death registration was checked against hospital records, and registers

of myocardial infarctions (and strokes in a subsample of centres) were set up. Analysis of data on myocardial infarctions from 37 centres in 27 countries (in both East and West) in 1985-87 revealed that differences in mortality rates between centres were not due to differences in reporting or treatment, but that mortality generally reflects incidence of infarctions (WHO MONICA Project, Tunstall-Pedoe, Kuulasmaa, Amouyel, Arveiler, Rajakangas, and Pajak, 1994). The correlations between nonfatal definite myocardial infarction and fatal infarctions were 0.67 for men and 0.59 for women; correlations between official national death rates from coronary heart disease and nonfatal definite myocardial infarction rates were 0.77 in men and 0.75 in women. Countries that ranked high in coronary mortality also rank high in coronary morbidity.

#### *2. Birth weight*

Birth weight is considered to be one of the most important measures of health status of a population; it is a strong predictor of both mortality and morbidity, and reflects nutritional status and growth rates (Alberman, 1991). An analysis of national birth registers in the Czech Republic and Sweden for the years 1989-1991 showed that mean birth weight was substantially lower in Czech babies (table 39), and that large differences in birth weight were present at each level of education or marital status (Koupilova, Vagero, Leon, Pikhart, Prikazsky, Holcik and Bobak, 1997).

#### *3. Self-rated health*

The last example of a non-fatal health indicator is self-rated health. It is a useful indicator for the study of the health of populations. It has been shown to predict mortality in prospective studies (Mossey and Shapiro, 1982; Idler and Angel, 1990; Pijls, Feskens, and Kromhout, 1993; Appels, Bosma, Grabauskas, Gostautas, and Sturmans, 1996), even after controlling for physician's evaluation of health status (Pijls, Feskens, and Kromhout, 1993). This predictive power of self-rated health has also been demonstrated in Lithuania, which was part of the former Soviet Union (Appels, Bosma, Grabauskas, Gostautas, and Sturmans, 1996).

TABLE 39. MEAN BIRTH WEIGHT AND DIFFERENTIALS BY MATERNAL EDUCATION AND MARITAL STATUS; BIRTHS REGISTERED IN THE CZECH REPUBLIC AND SWEDEN, 1989-1991

	Czech Republic (N=380,633)			Sweden (N=351,268)		
	Birth weight (g)	Difference from baseline (within country) (g)		Birth weight (g)	Difference from baseline (within country) (g)	
		Crude	Adjusted*		Crude	Adjusted*
All births	3 310	-	-	3 522	-	-
Education						
Primary	3 165	0	0	3 458	0	0
Vocational	3 308	143	151	3 526	64	74
Secondary	3 350	185	190	3 545	83	107
University	3 371	206	197	3 570	109	136
Marital status						
Non-married	3 327	0	0	3 408	0	0
Married	3 133	194	167	3 476	123	86

Source: Koupilova and others, "Social variation in size at birth and preterm delivery in the Czech Republic and Sweden, 1989-1991", *Paediatric and Perinatal Epidemiology* (1997).

\*Adjusted for maternal age and parity, and sex of the child.

All p-values for trends and differences less than 0.001.

We have conducted random sample surveys in several populations in CCEE and FSU. Consistently with the high overall mortality in the region, the proportion of respondents rating their health as poor or very poor was also higher than in Western countries (figure 52).

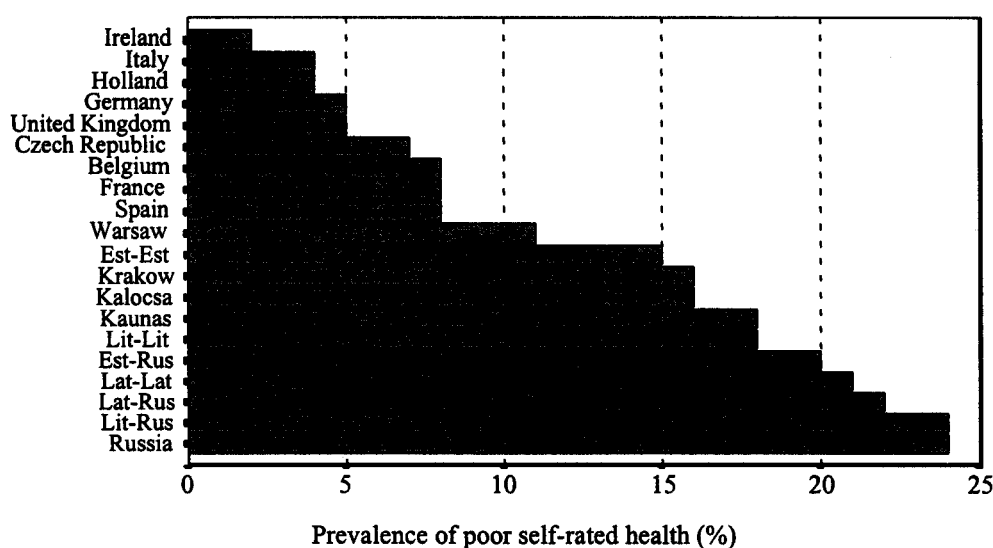
In summary, available data show that levels of health and well being are lower in CCEE and FSU than in Western populations. This finding leads to two conclusions. First, the higher mortality in CCEE and FSU is due to high incidence, rather than to high fatality as a result of poor medical care. Second, mortality reflects the poor health status in general in CCEE and FSU.

#### C. AN EPIDEMIOLOGICAL TRANSITION GONE WRONG?

We have come to think of the epidemiological transition as the shift from infectious to non-infectious diseases that occurred when overall mortality declined and life expectancy improved. This is the pattern that was observed in Western Europe (Omran, 1971). However, the picture is more complex.

The example of England and Wales is instructive (table 40). In the 1930s, life expectancy at birth improved, almost completely as a result of the decline in infant mortality rates. For men in their middle years, there was no improvement in life expectancy because of the increased mortality from cardiovascular disease and cancer. In fact, between 1921 and 1971, life expectancy for men aged 45 increased by only 2 years. The decline in infectious disease mortality was more or less balanced by the increase in mortality from lung cancer and cardiovascular diseases (Davey Smith and Marmot, 1991). Because these causes of death affected men more than women, life expectancy for women aged 45 increased more, by about 5 years, during the same 50-year period. The epidemiological transition was even less successful in the United States where life expectancy at birth declined from 63.3 to 58.5 years between 1933 and 1936 (Heleniak, 1995). Then, in the 23 years after 1971, life expectancy of men aged 45 in England and Wales increased by more than it had in the previous 50 years. This was in large measure due to the decline in cardiovascular diseases mortality and the levelling off of the lung cancer epidemic.

Figure 52. Self-rated health in selected populations



Source: Author's database; A. Kunst, *Cross-national comparisons of socio-economic differences in mortality* (Rotterdam, Erasmus University, 1997).

TABLE 40. LIFE EXPECTANCY AT BIRTH IN ENGLAND AND WALES IN THIS CENTURY

Year	Life expectancy at birth (years)		Infant mortality (per 1,000 live births)	Life expectancy at 45m (years)	
	Men	Women		Men	Women
1901-1905	-	-	138	-	-
1921	55.9	59.9	83	25.5	28.0
1931	58.2	62.4	66	25.3	28.0
1940	59.4	63.9	60	24.5	28.0
1951	65.8	70.9	30	25.9	30.2
1960	68.2	74.1	22	27.3	32.2
1971	69.2	75.5	17.5	27.7	33.2
1981	71.2	77.2	11.1	28.9	34.2
1991	73.5	79.0	7.4	30.8	35.5
1994	74.3	79.7	6.2	31.5	36.1

Sources: 1920 to 1960: Ministry of Health, *On the state of public health. Annual reports of the Chief Medical Officer to the Ministry of Health (years 1920 to 1961)* (London, HMSO, 1961); 1960-1994: WHO Regional Office for Europe, *Health for All Database*, Internet: <http://www.who.dk/country/country.htm> (1997).

Data from Hungary, as an example of the CCEE, shows both similarities and differences from the West (table 41). Life expectancy at birth for men failed to increase after the mid-1960s despite the decline in infant mortality. This may appear surprising, given that infant

mortality has a large effect on life expectancy at birth. The reason lies in decline in life expectancy at age 15 (as shown in figure 45). The probability of survival between 35 and 65 in Hungary had increased steadily from 1900 to 1960 when it reached its peak of 73 per cent

surviving. It then began to decline steadily. By 1994, the corresponding probability was only 58 per cent. This contrasts markedly with Austria where the probability of a 35 year old man surviving until 65 in 1994 was 79 per cent (Division of Population and Health Statistics, 1996). What we are witnessing in CCEE is an adverse mortality pattern that affects particularly middle-aged men. While the decline in adult life expectancy itself may not be entirely unique, what is unprecedented is its long-term character: it has lasted for several decades!

This pattern is consistent with the conceptual framework developed by John Powles (Powles, 1992). He has argued that there are several paths from high to low mortality (figure 53). According to his model, most Western countries followed a "central path" to initially intermediate levels of non-communicable diseases; European Mediterranean countries experienced a fast decline in infectious disease while preserving their low levels of non-communicable disease; and Eastern Europe experienced high levels of non-communicable diseases.

In figures 54-56, real data for the period 1950-1990 were plotted according to the model. Infant mortality was taken as a proxy for mortality from infections, and male life expectancy at age 15 was taken as a proxy indicator for

mortality from non-communicable diseases. Figure 54 again uses data on Austria, Czechoslovakia and Hungary (one country until 1918). The difference in mortality patterns is obvious. While in Austria infant mortality declined and adult life expectancy increased, Hungary and Czechoslovakia experienced decline in both infant mortality *and* adult life expectancy.

In figures 55 and 56, countries were grouped by mortality pattern. CCEE confirm the observation from the previous figure - none of them recorded an increase in adult male life expectancy (figure 55). Non-Mediterranean CCEE experienced substantial decline in infant mortality but also in adult male life expectancy. Western European and Mediterranean countries, in contrast, experienced both decline in infant mortality and increase in adult life expectancy (figure 56). The development in the Mediterranean CCEE is extremely interesting. Albania and Yugoslavia differ substantially from other CCEE: reported infant mortality that was still relatively high in 1990, but adult male life expectancy that increased by more than 5 years after 1950 (Gjonca, 1997).

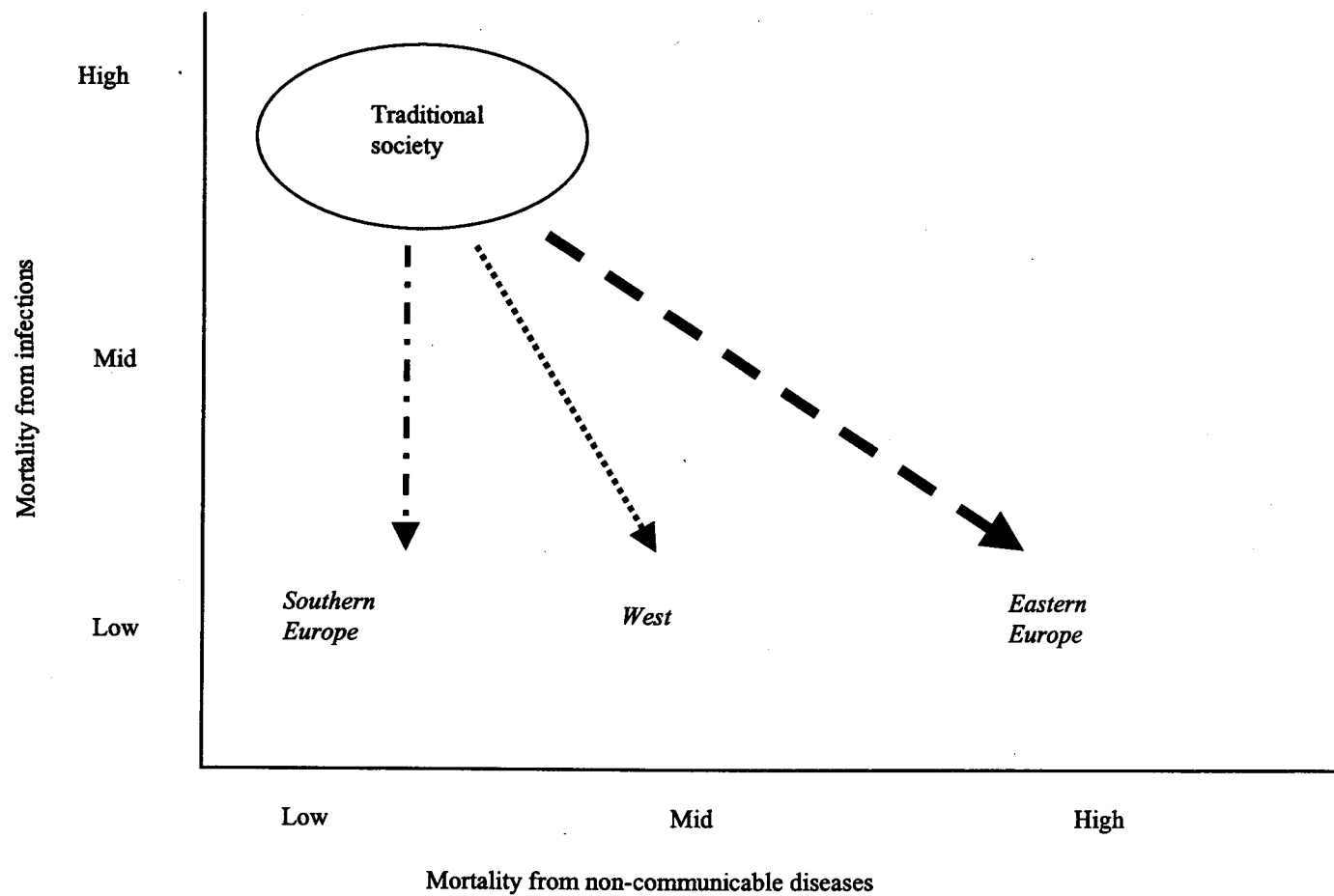
It is worth noting that when comparing 1950 with 1990, Mediterranean countries do not seem to have the advantage over Western populations predicted by Powles (1992). This is due to the

TABLE 41. LIFE EXPECTANCY AT BIRTH, INFANT MORTALITY, AND PROBABILITY OF SURVIVAL BETWEEN AGES 35 AND 65, HUNGARY, 1900-1994

Year (annual average)	Life expectancy at birth		Infant mortality (per 1,000 live births)	Probability of survival between ages 35 and 65	
	Men	Women		Men	Women
1900-1901	36.6	38.2	218.2	53.8	54.5
1920-1921	41.0	43.1	192.6	58.7	62.6
1930-1931	48.7	51.8	157.0	63.7	68.5
1941	55.0	58.2	115.6		
1949	59.3	63.4	91.0		
1960	65.9	70.1	47.6	72.9	81.9
1966	67.5	72.2	38.4		
1970	66.3	72.1	35.9		
1980	65.5	72.7	23.2	65.3	81.6
1990	65.1	73.7	14.8		
1994	64.8	74.2	11.5	58.3	81.5
Austria 1993/94	73.5	79.9	6.3	79.1	90.7

Source: Division of Population and Health Statistics, (1996). *Main features of the Hungarian demographic situation in the early nineties*. Budapest: Hungarian Central Statistical Office.

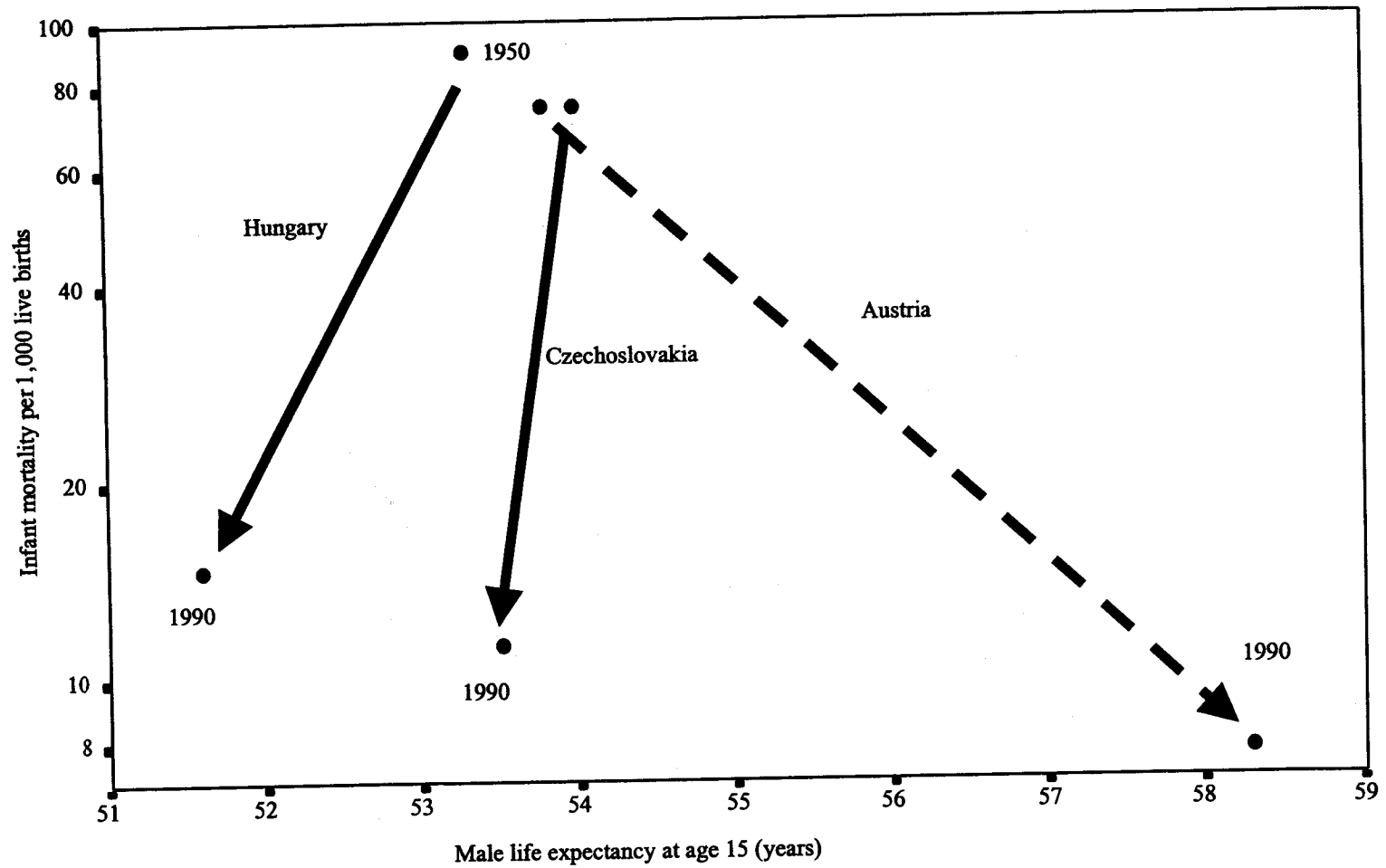
Figure 53. Paths from high to low mortality



Source: J. Powles, "Changes in disease patterns and related social trends", *Social Science in Medicine*, vol. 35, pp. 377-387 (1992).

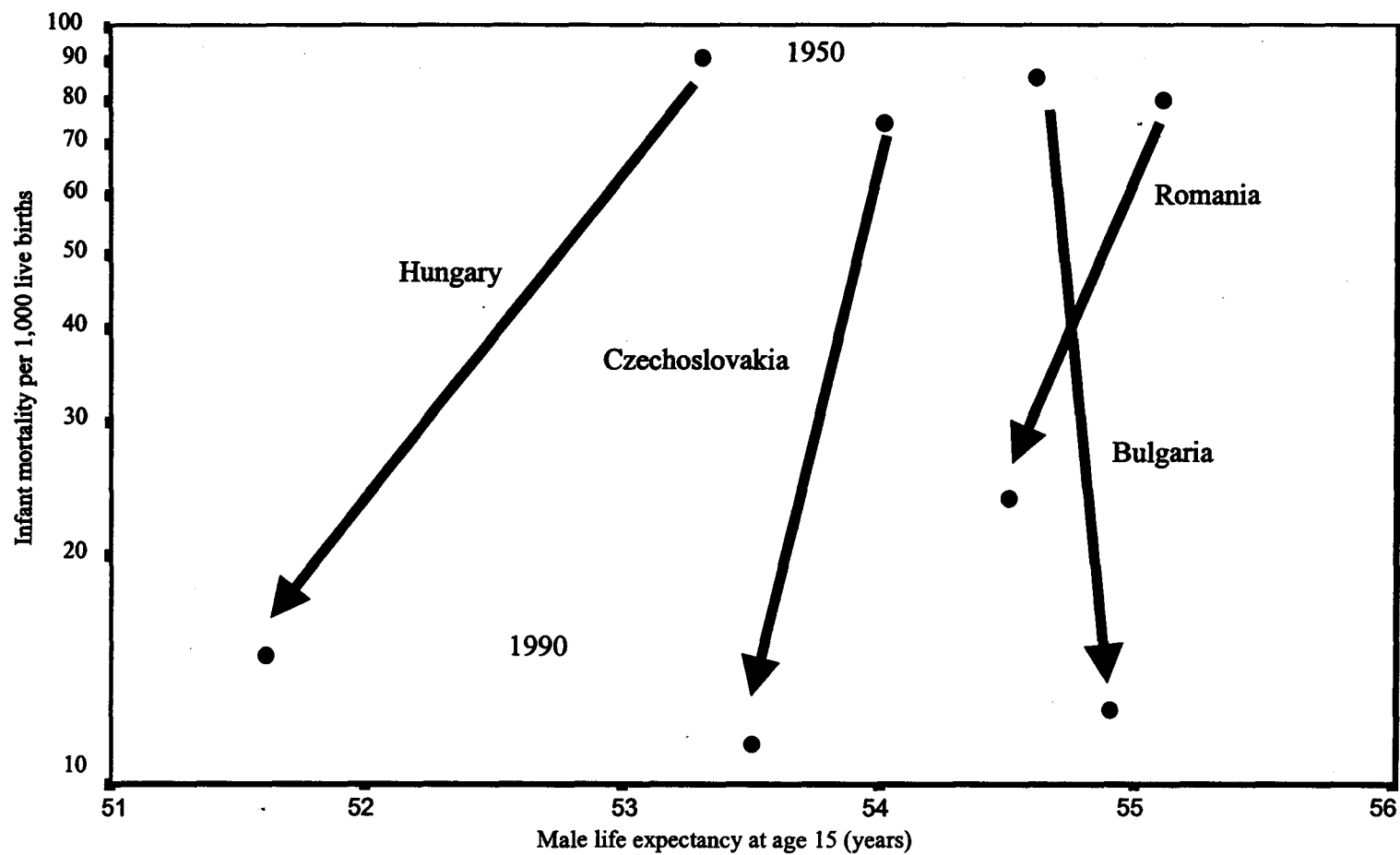


Figure 54. Changes in infant mortality and life expectancy of men at age 15, 1950-1990: Austria, Czechoslovakia and Hungary



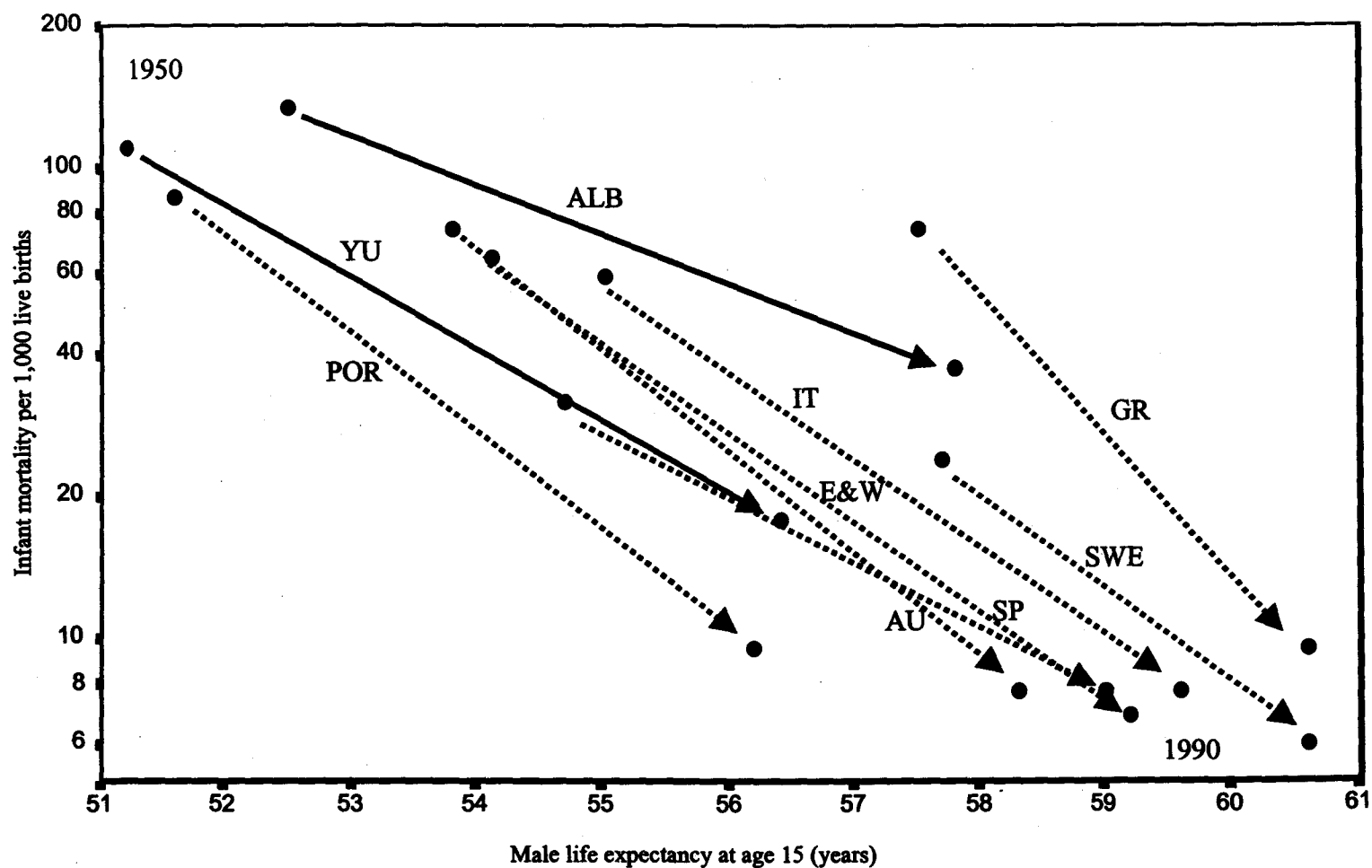
Source: World Health Organization, Regional Office for Europe, *Health for All Database* (1997); figure 50 in this chapter.

Figure 55. Changes in infant mortality and life expectancy of men at age 15, 1950-1990: Hungary, Czechoslovakia, Romania and Bulgaria



Source: World Health Organization, Regional Office for Europe, *Health for All Database* (1997); figure 50 in this chapter.

Figure 56. Changes in infant mortality and male life expectancy at age 15, 1950-1990: Western Europe and Mediterranean countries



Source: World Health Organization, Regional Office for Europe, *Health for All Database* (Geneva, WHO, 1997).

decline in cardiovascular mortality in Northwest Europe since the late 1960s. The pattern observed in the Mediterranean CCEE points to diet as the key factor determining mortality.

The current unfavourable mortality and health status of CCEE and FSU indeed resembles an unsuccessful epidemiological transition, at least its fourth stage (the "age of delayed degenerative diseases" (Olshansky and Ault, 1986)). While the CCEE and the FSU successfully reduced mortality from infections, they were very soon caught by an epidemic of non-communicable diseases. However, this cannot be simply considered a natural cause of epidemiological transition. While mortality in the region has traditionally been higher than in the West, this was not a universal phenomenon. Hungary, Czechoslovakia and Austria had similar rates of both infant and adult (including cardiovascular) mortality after WWII (Bobak and Feachem, 1992); the Baltic states and Finland also had similar mortality rates in the 1950s (Hertzman, 1995); Eastern and Western Germany also diverged (Chenet, McKee, Hort, Brand, and Bojan, 1995). At least in these countries, the roots to the post-war trends are related to political developments in individual countries. The crucial questions then become: Why was the epidemic of non-communicable diseases controlled so rapidly in the West? Why was it not so in the East?

#### D. AN EXPLANATORY MODEL FOR DEVELOPMENT OF THE MORTALITY GAP

##### 1. *Post-war period*

##### *General model*

Attempts to explain the long-term unfavourable mortality trends in the CCEE and the FSU are limited by the absence of relevant data. However, we can offer a formulation that may, at least in part, be testable. We present this formulation in relation to Czechoslovakia but it may well apply to other central European countries as well. It runs as follows. After the war, the political and economic changes that accompanied the coming of communism to Czechoslovakia may well have improved basic conditions for a large proportion of the population in

the form of better housing, nutrition, sanitation, and general welfare services. These may have reduced absolute deprivation and insecurity. During this period, mortality in Czechoslovakia was similar to that of West Germany and Austria, neighbouring countries in Central Europe.

From the 1960s on, the Western countries enjoyed the benefits of the growing economic prosperity seen in most established market economies. On the other hand, it is possible to argue that once the basic prerequisites for freedom from absolute deprivation were in place in the communist countries, other aspects of quality of life became more important. In this respect, Czechoslovakia and other countries lagged behind the West (Bobak and Feachem, 1992).

The failure of the economic and political system to deliver increasing quality of life led to growing dissatisfaction in the population. Any movement toward economic and political liberalisation led to repression that must have led to a heightened sense of hopelessness and deprivation compared to the populations of neighbouring Western states. Our hypothesis would be that this situation in Central and Eastern European countries had profound effects on the nature of social relationships, the organisation of the work place, and a personal sense of control and psychological well-being that may, in turn, have had profound effects on the risk of death. There may have been differences in detail that were vitally important, but we speculate that such changes affected the majority of countries in the region.

In all Western societies, there are socio-economic gradients in most diseases. The reasons for these gradients are not entirely clear, but a similar set of explanations have been suggested as those given above for the East-West gap. We have speculated that the East-West gradient may in fact be analogous to the socio-economic gradient within Western societies (Bobak and Marmot, 1996a). Figure 57 provide some support for this hypothesis. The figures are based on the observation that various diseases show different relationships to socio-economic status. For example, breast cancer within Western societies is usually more common among women from higher social groups,

while lung cancer is more common among lower social groups. Figure 57 shows that for death rates from various causes the East-West ratios and social class gradient within England and Wales are markedly correlated.

### *Mechanisms*

The mechanisms through which social and political conditions affect the health of populations are complex. The broad determinants of health include medical care, life style and diet, environmental pollution, socio-economic environment and psychosocial factors. Each one has contributed, although in differing amounts, to the patterns of mortality and health status in CCEE and FSU; their contribution has been extensively reviewed elsewhere (Bobak and Marmot, 1996a; Hertzman, Kelly and Bobak, 1996). Briefly, environmental pollution and medical care probably played only a modest role in the mortality trends. Life style and diet probably had a more substantial impact, and smoking and alcohol seem to have the largest identifiable impact. For example, Peto and others estimated that about half of the East-West gap was caused by smoking (Peto, Lopez, Boreham, Thun, and Heath, 1992); Leon and colleagues believe that much of Russian mortality is due to alcohol (Leon, Chenet, Shkolnikov, Zakharov, Shapiro, Rakhmanova, Vassin, and McKee, 1997). However, even if these views are correct (although they may be over-estimates), the next question is: why have people in CCEE and FSU been smoking, drinking to such an excessive level and why have they been eating such an unhealthy diet?

We have speculated that if the social environment existing in these countries had a strong influence on psychosocial well being, this would result in poor life style and diet (Bobak and Marmot, 1996b). Psychosocial factors have been shown to have a direct effect on health via neuroendocrine pathways (Steptoe, 1997); part of the poor health in CCEE and FSU could be due to this direct effect. Psychosocial factors also influence health behaviours and diet; these may "mediate" the rest of the effect of the political, social and economic environment on health. While not important on the biological level, clarification of the hierarchy of causes is essential in designing and promoting policies to

improve the currently unfavourable health status.

### *2. Social and political changes after 1989*

After 1989, inter-country differences may have become more important. All countries in the region experienced the "shocks" of the economic changes but they took different forms. It is possible that in the former Eastern Germany, the Republic of Czechoslovakia and Poland, for example, society adapted to the new rules after the initial shock of transition. We have no evidence, but there may have been some re-discovery of forms of social relationship that antedated the communist period. Such social mechanism would not have been available to the same extent in the former Soviet Union. The studies by Rose have demonstrated that in Russia the new political and economic regime has substantially less support than have those of the CCEE. In 1995, for example, less than 40 per cent of all Russians approved the current political system, and only 13 per cent rated the new economic system as better than the old (World Bank, 1996; Rose, 1996).

Further support for growing differences between individual countries after 1989 is provided in table 42. Real wages declined to a greater extent in the countries of the former Soviet Union than in the Czech Republic or in Poland. Similarly, the measures of poverty are greater in the FSU. Income inequalities increased in all countries in the region, but increased more rapidly in the FSU. Table 42 shows, among other indicators, Gini coefficient, a measure of income inequality, in selected countries of the region. (The larger the values of Gini coefficient, the larger the income inequality). According to World Bank estimates, income inequalities in Russia (and probably other countries of the FSU) are approaching Latin American levels (World Bank, 1996). In addition, unemployment, previously unknown in the region, appeared and rose substantially in some countries.

Although these figures show worsening of the overall socio-economic circumstances in all countries in the region, the trends in life expectancy suggest that only in FSU did an acute mortality crisis occur after 1990, above the

Figure 57. Mortality ratios: east/west and low/high social class in the United Kingdom

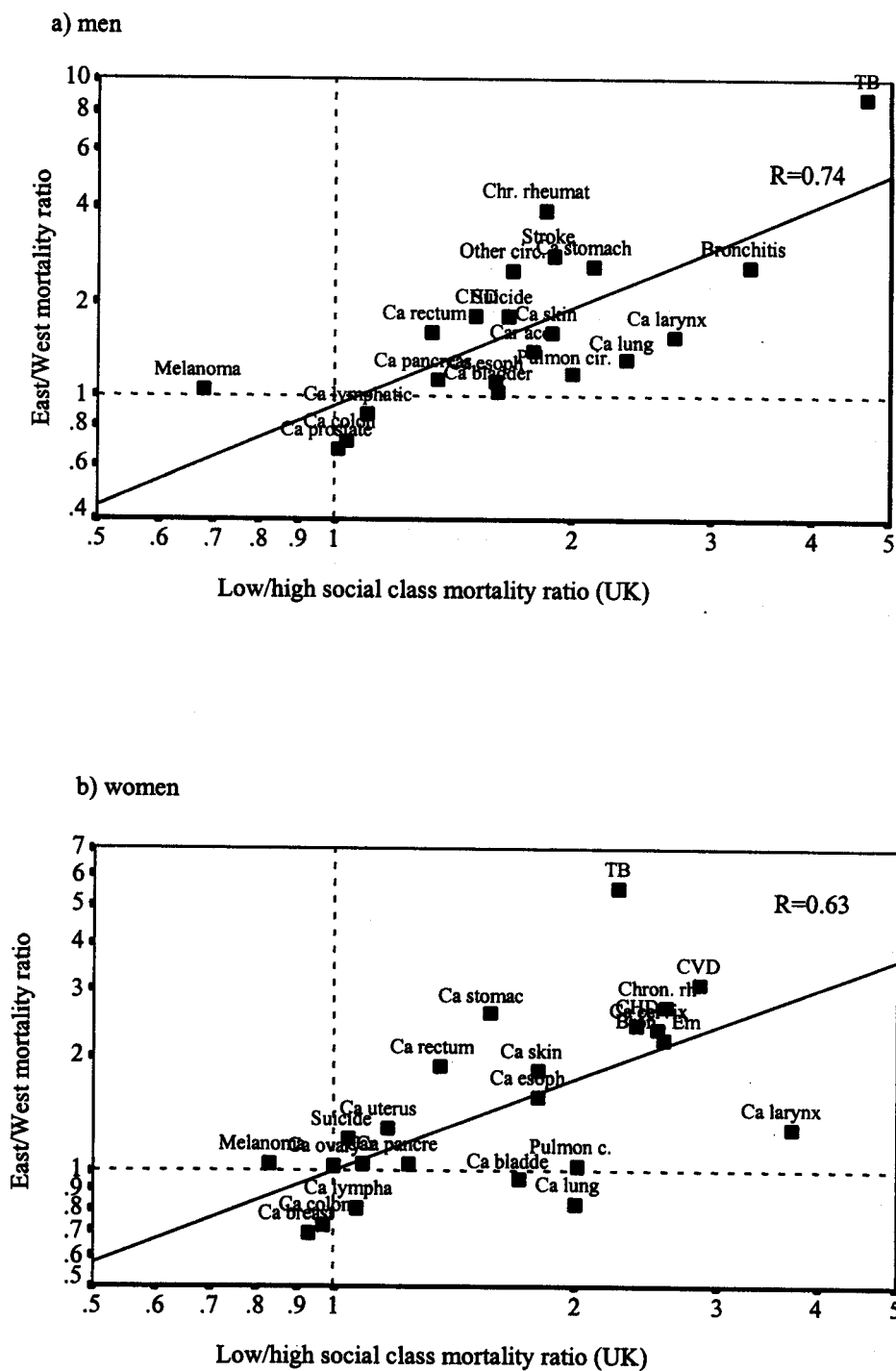


TABLE 42. CHANGES IN SOCIAL INDICATORS IN CCEE AND FSU BETWEEN 1989 AND 1994

	Real wages (per cent decline between 1989 and 1994)	Real income (per cent decline between 1989 and 1994)	Food share (per cent of in- come)		Income inequality (Gini coeff.)		Percentage of earners below 50 per cent of average	
			1989	1994	1989	1994	1989	1994
Czech Republic	-14	-18	33	32	19	27	6	9
Hungary	-12	-10	37	38	21	23	13	15
Poland	-27	-9	49	43	25	30	4	12
Bulgaria	-38	-44	43	49	25	37	5	10
Romania	-47	---	52	62	23	28	---	---
Estonia	---	---	---	---	28	39	---	---
Latvia	-47	---	36	52	---	---	10	21
Lithuania	-67	---	35	57	28	37	12	20
Russia	-36	-21	34	47	26	41	12	33

Source: United Nations Children's Fund, *Poverty, children and policy: responses for a brighter future. Central and Eastern Europe in Transition. Public Policy and Social Conditions. Economies in Transition Studies. Regional Monitoring Report No. 3* (Florence, UNICEF, 1995).

long-term unfavourable trend. In most CCEE, today's crisis is, in principle, simply a legacy of a longstanding situation.

Even in the FSU, however, the link between the economic transition and mortality is not simple. The change in the political situation (the break-up of the Soviet Union) and the substantial deterioration of the social and economic circumstances of the population began in 1992, but the increase in mortality started in 1990 (United Nations Children's Fund, 1997). Although it is possible that the official macro-economic indicators do not sufficiently reflect the living conditions of the population, the available information on trends over time do not provide clear evidence. On the other hand, the life expectancy decline in Russia accelerated after 1992, consistent with trends in socio-economic situation. Cornia found that in geographical areas of Russian Federation, changes in social indicators (unemployment, labour

turnover and shifts in marital status) between 1989 and 1993 were markedly correlated with changes in life expectancy over the same period (Cornia, 1997).

Other factors may also be related to the recent mortality changes in Russia. Although direct evidence is sparse, it has been widely accepted that the increase in life expectancy in the FSU in the late 1980s was related to the antialcohol campaign, while the subsequent fall in life expectancy was at least partly due to the collapse of the antialcohol measures (Shkolnikov, 1997; McKee and Leon, 1994). Analyses of reconstructed Russia mortality data indirectly support this hypothesis (Leon, Chenet, Shkolnikov, Zakharov, Shapiro, Rakhmanova, Vassin, and McKee, 1997). Anecdotal evidence (Gorbachev, 1996) and discussions with Russian colleagues suggest that the antialcohol campaign and its collapse were accompanied by a rise and fall of popular hope in change

to the better. Disillusionment, perceived lack of control, and other related psychosocial factors may have produced this mortality pattern.

The role of such factors is supported by the fact that the changes in mortality were accompanied by no less dramatic changes in fertility on Russia - the crude birth rate declined by 36 per cent between 1989 and 1994, from 14.7 to 9.4 per thousand (Heleniak, 1995). The psychosocial factors may have acted indirectly, through diet or health-related behaviour (including alcohol and violence), and/or directly, via neuroendocrine pathways (Steptoe, 1997).

#### E. SOCIO-ECONOMIC DIFFERENCES IN HEALTH WITHIN CCEE AND FSU

Socio-economic factors are perhaps the most powerful predictors of health outcomes in all populations for which data are available. The same applies for studying differences between populations; for example, per capita gross domestic product effectively predicts national mortality on the global scale. The links between socio-economic factors and health indicators in former communist countries were not made public until recently, but some information became available after 1990. This information is highly relevant for studying the determinants of health in CCEE and FSU.

##### 1. *The magnitude of health inequalities in CEE*

Recent research is remarkably consistent in the finding that socio-economic inequality in health in CEE is as large as, or larger than, in Western countries. Several examples will illustrate this observation with respect to mortality, self-rated health, and cardiovascular risk factors.

Kunst (1997) has analysed mortality by occupation type (manual vs. non-manual) and by education (upper secondary and higher versus lower than upper secondary) in the Czech Republic, Hungary and Estonia and compared the results with several Western countries. The results show that the relative differences in CCEE are larger than in Western Europe.

In our study of cardiovascular diseases risk factors in the Czech Republic, we were able to examine the educational gradient in cardiovascular diseases risk factors, and to compare it with the Western European pattern, as typified by the Finnish data. The comparisons covered the gradients in mean total cholesterol, diastolic blood pressure, body mass index and smoking prevalence in Czech and Finnish men and women. For cholesterol, smoking and body mass index (women only) the educational differences were larger among the Czechs. When gradients in other risk factors were compared with the British civil servant, similar findings were obtained (Bobak, 1996).

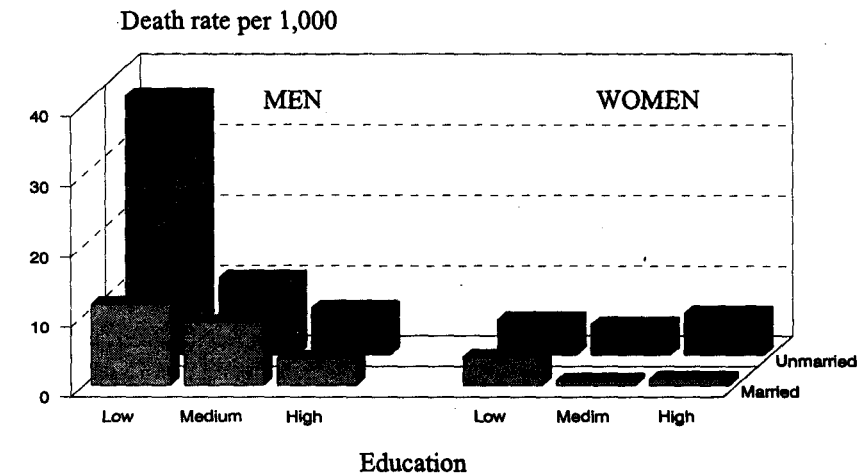
Koupilova and others (Koupilova, Vagero, Leon, Pikhart, Prikazsky, Holcik, and Bobak, 1997) compared socio-economic difference in birth weight in Czech Republic and Sweden in the period 1989-1991 (table 39). Again, social variations were larger in the Czech Republic.

The final example relates to self-rated health. We compared social gradients in Eastern European populations, shown in figure 52, with those found in national samples of Western countries (Kunst, 1997). With the exception of both Polish samples, the results are broadly similar between the CCEE and Western Europe, indicating a considerable gradient in self-rated health. In an unpublished preliminary ecological analysis, measures of material inequality were also found to be related to self-rated health, beyond the individual measures of material deprivation.

Analysing routine mortality data from Hungary and Poland, Hajdu, McKee and Bojan (1995) and Watson (1995), both observed that unmarried men are at particularly increased risk of death. This was also found in Czech data (Blazek and Dzurova, 1997). Subsequent cohort analysis of the Warsaw MONICA sample has not confirmed these observations, but has also suggested that unmarried men with low education are the most vulnerable group (figure 58). Whether this is due to their material circumstances, as suggested by Hajdu, McKee, and Bojan (1995), or to their social isolation and absence of vital social networks, as speculated by Watson (Watson, 1995), remains an interesting topic for future research.



**Figure 58. Cardiovascular mortality by education and marital status in Warsaw, 10-year follow-up of the MONICA sample**



From Source: Rywik, Broda and others, unpublished.

The existence and magnitude of health inequalities in the CCEE raises an important issue: even decades of focused and often ruthless pursuit of material equality did not reduce health inequalities. Possibly, the material inequalities have diminished, but the educational inequalities appeared even stronger. As health inequalities in the CCEE become better understood, it is possible that other important dimensions will be identified such as, for example, in ethnic minorities (Mann, 1992).

## 2. Trends in health inequalities

We have seen that health inequalities in the CCEE are as large as in Western countries, or even larger. A question arises on the trends in inequalities during the social and economic transformation.

It has been documented that the transition has had a heavy impact on living standards (table 42), and that the impact is likely to be divided unequally in the society. Data from UNICEF and the World Bank suggest that there

was a relatively rapid increase in income inequalities since 1989 (World Bank, 1996; United Nations Children's Fund, 1997). These macro-economic data are supported by a series of micro censuses conducted by a Czech team to study the impact of the transformation on the society (but not including health). The team found a remarkable divergence in income by educational group (Machonin and Tucek, 1996) whereas until the end of 1990s, education had been only weakly correlated with income. In this context, it is not surprising that the few available data all suggest a divergence in health status between social groups.

National data from the Czech Republic revealed that the gradient in mortality became steeper between 1990-91 and 1995 for marital status, and for education between 1980-81 and 1995 (Blazek and Dzurova, 1997). In the case of mortality, the widening of inequalities was related to both improvement in the advantaged groups and worsening in the disadvantaged groups (table 43). The dramatic increase in mortality of least educated men (by 77 per cent)

TABLE 43. DIFFERENCE IN MORTALITY AND MORTALITY RATIOS (RR) BY MARITAL STATUS IN 1990-1991 AND 1995, AND BY EDUCATION IN 1980-1981 AND 1995

	Men 40-64			Women 40-59		
	<i>Abs. change</i>	<i>RR 1990</i>	<i>RR 1995</i>	<i>Abs. change</i>	<i>RR 1990</i>	<i>RR 1995</i>
Marital status						
Married	-5%	1.0	1.0	-11%	1.0	1.0
Single	-5%	1.88	1.85	+3%	1.54	1.78
Divorced	+6%	2.03	2.27	+8%	1.52	1.84
Widowed	+12%	1.42	1.67	+2%	1.25	1.42
	<i>Abs. change</i>	<i>RR 1980</i>	<i>RR 1995</i>	<i>Abs. change</i>	<i>RR 1980</i>	<i>RR 1995</i>
Education						
Primary	+77%	2.42	4.46	+4%	1.44	2.04
Vocational	-17%	1.67	1.44	+33%	1.07	1.93
Secondary	+8%	1.41	1.58	-16%	1.33	1.53
University	-4%	1.0	1.0	-27%	1.0	1.0

Source: J. Blazek and D. Dzurova, "The case of the Czech Republic", Part of the United Nations University/World Institute for Development Economics Research (UNU/WIDER) Project, *Economic shocks, social stress and the demographic impact*, (Unpublished manuscript, 1997).

is alarming, and may indicate that a relatively large segment of the society is highly vulnerable to the social changes.

This widening of inequalities has been also observed in other countries. Forster (unpublished) examined social differences in mortality between Budapest districts between 1980-1983 and 1990-1993. He found that the while mortality from most causes declined in the most affluent districts over the last decade, they increased in the most deprived. Consequently, the mortality gap between the most and least affluent areas increased by some 13 per cent.

Recent data from Russia also showed an increase in mortality differences by education between 1988-1989 and 1993-1994. Mortality in men with better education increased by 35 per cent but among men with lower education the increase was 57 per cent. Among women, the corresponding mortality increases were 8 per cent and 30 per cent (Shkolnikov, 1997).

Data on trends in outcomes other than mortality are sparse but it seems that the gradient in cardiovascular diseases risk factors and behaviours is also increasing. The Czech MONICA study found that the educational gradient in most cardiovascular diseases risk factors increased between 1985 and 1992 (Bobak, Sko-

dova, Pisa, Poledne, and Marmot, 1997). In contrast to the mortality analyses shown above, however, the widening of inequalities in risk factors was caused mainly by improvement in the higher educational groups, not by worsening in the lower groups. One exception was found in with respect to smoking by women; the increase in inequalities was due to a rise in smoking among women with lower education.

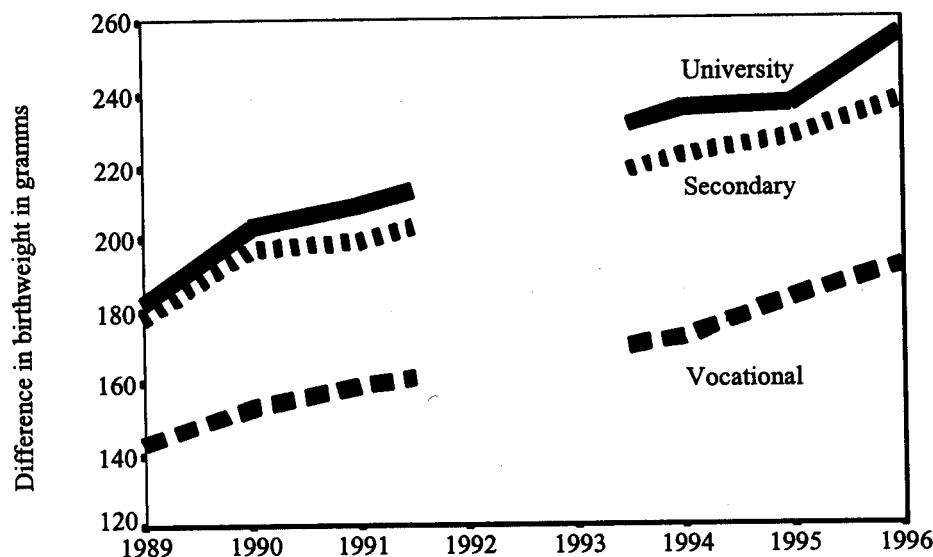
A recent analysis of birth weight and infant mortality in the Czech Republic also shows a widening of the social differentials (figure 59). As with the cardiovascular diseases risk factors in the Czech Republic, this seems to be related more to improvement in the higher social groups than to worsening in the lower ones.

These trends toward greater social inequalities in CCEE and FSU are worrying and require not only more research but also a policy response.

#### D. FINAL REMARK

Mortality is now improving in several CCEE. Life expectancy in Russia improved for the first time in 1995, and preliminary data suggest that it continued improving in 1996 (Shkolnikov, personal communication). These improvements

Figure 59. Trends in birthweight differences by maternal education in the Czech Republic, 1989-1996 (differences from primary educated)



at times of the often difficult social and economic transition provide hope for the countries in the region. At the same time, it challenges the premise that mortality increase is the consequence and the inevitable cost of the transformation in the former socialist countries.

#### REFERENCES

- Alberman, E. (1991). Are our babies becoming bigger? *Journal of the Royal Society of Medicine*, vol. 84, pp. 257-260.
- Anderson, B.A., and B. D. Silver (1986). Infant mortality in the Soviet Union: regional differences and measurement issues. *Population and Development Review*, vol. 12, pp. 705-738.
- Appels, A., H. Bosma, V. Grabauskas, A. Gostautas, and F. Sturmans (1996). Self-rated health and mortality in a Lithuanian and Dutch population. *Social Science in Medicine*, vol. 42, pp. 681-689.
- Blazek, J., and D. Dzurova (1997). The case of the Czech Republic. Part of the United Nations University/World Institute for Development Economics Research (UNU/WIDER) Project "Economic shocks, social stress and the demographic impact". Unpublished manuscript.
- Bobak, M. (1996). *Determinants of the epidemic of coronary heart disease in the Czech Republic*. Doctoral dissertation. London School of Hygiene and Tropical Medicine. London: University of London.
- \_\_\_\_\_, and R. G. A. Feachem (1992). Health status in the Czech and Slovak Federal Republic. *Health Policy and Planning*, vol. 7, pp. 234-242.
- Bobak, M., and M. Marmot (1996a). East-West mortality divide and its potential explanations: proposed research agenda. *British Medical Journal*, vol. 312, pp. 421-425.
- \_\_\_\_\_. (1996b). East-West health divide and potential explanations. In *East-West life expectancy gap in Europe. Environmental and non-environmental determinants*, C. Hertzman, S. Kelly, and M. Bobak, eds. Dordrecht: Kluwer Academic Publishers, pp. 17-44.
- Bobak, M., Z. Skodova, Z. Pisa, R. Poledne, and M. Marmot (1997). Political changes and trends in cardiovascular risk factors in the Czech Republic 1985-1992. *Journal of Epidemiology and Community Health*, vol. 51, pp. 272-277.
- Chenet, L., M. McKee, N. Fulop, F. Bojan, H. Brand, and P. Kalbarczyk (1995). Changing life expectancy in central Europe: is there a single reason? *Journal of Public Health*, in press.
- Chenet, L., M. McKee, A. Hort, H. Brand, and F. Bojan (1995). Explaining the health divide in Germany: contribution of major causes of death to the difference in life expectancy at birth between East and West. Unpublished manuscript.
- Cornia, G.A. (1997). Labour market shocks, psychosocial stress and the transition's mortality crisis. A paper presented at the United Nations University/World Institute for Development Economics Research (UNU/WIDER) Project Meeting on Economic Shocks, Social Stress and the Demographic Impact, Helsinki, April 17-19, 1997.
- Davey Smith, G., and M. G. Marmot (1991). Trends in mortality in Britain: 1920-1986. *Annals of Nutrition and Metabolism*, vol. 35 (Supplement 1), pp. 53-63.
- Division of Population and Health Statistics, (1996). *Main features of the Hungarian demographic situation in the early nineties*. Budapest: Hungarian Central Statistical Office.
- Eberstadt, N. (1993). Eastern Europe's Disturbing Health Crisis. *Wall Street Journal Europe*, 30 September.

- \_\_\_\_\_ (1994). Demographic disaster. The Soviet legacy. *The National Interest* (Summer), pp. 53-57.
- Gjonca, A. (1997). The limits to "good health at low cost": mortality transition in Albania, 1950-1990. A paper presented at the annual conference of Population Association of America, Washington, D.C., 27-29 March 1997.
- Gorbachev, M. (1996). *Memoirs*. London: Doubleday.
- Hajdu, P., M. McKee and F. Bojan (1995). Changes in premature mortality differentials by marital status in Hungary and in England and Wales. *European Journal of Public Health*, vol. 5, pp. 259-264.
- Heleniak, T. (1995). Economic transition and demographic change in Russia, 1989-1995. *Post-Soviet Geography*, vol. 36, pp. 446-458.
- Hertzman, C. (1995). *Environment and health in Central and Eastern Europe. A report for the Environmental Action Programme for Central and Eastern Europe*. Washington, D.C.: World Bank.
- Hertzman, C., S. Kelly, M. Bobak, eds. (1996). *East-West life expectancy gap in Europe. Environmental and non-environmental determinants*. Dordrecht: Kluwer Academic Publishers.
- Idler, E.L., and R. J. Angel (1990). Self-rated health and mortality in the NHANES-I Epidemiologic Follow-up Study. *American Journal of Public Health*, vol. 80, pp. 446-452.
- Kingkade, W. W., and B. Boyle Torrey (1992). The evolving demography of ageing in the United States of America and the former USSR. *World Health Statistics Quarterly*, vol. 45, pp. 15-28.
- Koupilova, I., D. Vagero, D. A. Leon, H. Pikhart, V. Prikazsky, J. Holcik, and M. Bobak (1997). Social variation in size at birth and preterm delivery in the Czech Republic and Sweden, 1989-1991. *Paediatric and Perinatal Epidemiology*.
- Kunst, A. (1997). *Cross-national comparisons of socio-economic differences in mortality*. Rotterdam: Erasmus University.
- Leon, D. A., L. Chenet, V. Shkolnikov, S. Zakharov, J. Shapiro, G. Rakhmanova, S. Vassin, and M. McKee (1997). Huge variation in Russian mortality rates 1984-94: artefact, alcohol, or what? *Lancet*, vol. 350, pp. 383-388.
- Leon, D.A., D. Vagero and P. Otterblad Olausson (1992). Social class differences in infant mortality in Sweden: comparison with England and Wales. *British Medical Journal*, vol. 305, pp. 687-691.
- Machonin, P., and M. Tucek (1996). *Ceska společnost v transformaci (Czech society in transformation) [in Czech]*. Prague: Sociologické nakladatelství.
- Mann, A.B. (1992). Vyvoj romskej rodiny na priklade troch spisskych obci (Development of gypsy family on the example of three villages) [in Slovak] *Demografie*, vol. 34, pp. 118-129.
- Margolis, P.A., R. A. Greenberg, L. L. Keyes and others (1992). Lower respiratory illness in infants and low socio-economic status. *American Journal of Public Health*, vol. 82, pp. 1119-1126.
- McKee, M., and D. Leon (1994). Deaths in Russia. *Lancet*, vol. 344, p. 1698.
- Ministry of Health (1961). *On the state of public health. Annual reports of the Chief Medical Officer to the Ministry of Health (years 1920 to 1961)*. London: HMSO.
- Mossey, J. M., and E. Shapiro (1982). Self-rated health: a predictor of mortality among the elderly. *American Journal of Public Health*, vol. 72, pp. 800-808.
- Olshansky, S. J., and A. B. Ault (1986). The fourth stage of epidemiologic transition: the age of delayed degenerative diseases. *Milbank Memorial Fund Quarterly*, vol. 64, pp. 355-391.
- Omran, A. R. (1971). The epidemiologic transition. A theory of the epidemiology of population change. *Milbank Memorial Fund Quarterly*, vol. 49, pp. 509-538.
- Peto, R., A. D. Lopez, J. Boreham, M. Thun, and C. Heath, JR. (1992). Mortality from tobacco in developed countries: indirect estimation from national vital statistics. *Lancet*, vol. 339, pp. 1268-1278.
- Pijls, L.T., E. J. Feskens, and D. Kromhout (1993). Self-rated health, mortality and chronic diseases in elderly men. The Zutphen Study, 1985-1990. *American Journal of Epidemiology*, vol. 138, pp. 840-848.
- Powles, J. (1992). Changes in disease patterns and related social trends. *Social Science in Medicine*, vol. 35, pp. 377-387.
- Rose, R. (1996). *New Russia Barometer VI: After the presidential election. Centre for the Study of Public Policy: Studies in Public Policy, No. 272*. Glasgow: University of Strathclyde.
- Shkolnikov, V. (1997). The population crisis and rising mortality in transitional Russia. Contribution for the United Nations University/World Institute for Development Economics Research Project "Economic shocks, social stress and the demographic impact". Unpublished manuscript.
- \_\_\_\_\_, F. Mesle and J. Vallin (1995). Health crisis in Russia: I. recent trends in life expectancy and causes of death from 1970 to 1993. *Population*, vols. 4-5, pp. 907-944.
- Stephens, A. (1997). Psychophysiological bases of disease. In *Comprehensive clinical psychology. Volume 8: Health Psychology*, M. Johnston and D. Johnston, eds. Elsevier Science.
- United Nations Children's Fund (UNICEF) (1994). *Crisis in mortality, health and nutrition. Central and Eastern Europe in transition. Public policy and social conditions. Regional Monitoring Report No. 2*. Florence: UNICEF.
- \_\_\_\_\_. (1995). *Poverty, children and policy: responses for a brighter future. Central and Eastern Europe in Transition. Public Policy and Social Conditions. Economies in Transition Studies. Regional Monitoring Report No. 3*. Florence: UNICEF.
- \_\_\_\_\_. (1997). *Children at risk in Central and Eastern Europe: perils and promises. Central and Eastern Europe in transition. Public policy and social conditions. Regional Monitoring Report No. 4*. Florence: UNICEF.
- Watson, P. (1995). Explaining rising mortality among men in Eastern Europe. *Social Science in Medicine*, vol. 41, pp. 923-934.
- World Health Organization MONICA Project, H. Tunstall-Pedoe, K. Kuulasmaa, P. Amouyel, D. Arveiler, A.-M. Rajakangas and A. Pajak (1994). Myocardial infarction and coronary deaths in the World Health Organization MONICA Project. Registration procedures, event rates, and case-fatality rates in 38 populations from 21 countries in four continents. *Circulation*, vol. 90, pp. 583-612.
- World Health Organization Regional Office for Europe (1997). *Health for All Database*. Internet: <http://www.who.dk/country/country.htm>.
- World Bank (1996). *World Development Report 1996. From plan to market*. New York: Oxford University Press.

## X. THE DYNAMICS OF MORTALITY IN POLAND

*Witold Zatoński\**

### A. INTRODUCTION

Premature mortality among Polish people from a cause other than an infectious disease is shaped primarily by so-called "man-made" diseases—those resulting directly or indirectly from human activity or life style. Between 1965 and 1991, the unusual phenomenon of increasing rates of premature death was observed in Polish men. As a result, at the death rates of the early 1990s, the probability that a 15-year-old boy living in Poland would reach the age of 60 is not only worse than in Western Europe, but also worse than in Latin America or even India (Murray and Lopez, 1994). Poland entered the 1990s in a catastrophic state of adult health (Zatoński, 1995).

The main causes of the increasing death rate, especially in the middle-aged Polish population, seem to involve the Polish life-style. There are more than 10 million smokers in Poland, with an annual cigarette consumption of almost 100 billion cigarettes; Polish men have been the world's greatest consumers of tobacco for more than 20 years. Three to four million Poles drink distilled alcohol daily, or almost daily, often to a state of intoxication. The Polish diet has long been characterised by a high intake of animal fat and a low intake of fruits and vegetables, especially during the winter and spring. Behaviour on the roads (by both pedestrians and drivers) and in the workplace does not meet West European norms of safety. Physical activity is still undiscovered as a method for maintaining good health and self-esteem.

Since 1991, however, after the sudden socio-economic changes of the late 1980s, there has been a sudden drop in adult mortality rates. The main contribution to this change was a sharp decrease in cardio-

vascular disease (CVD) mortality, with a rate of decline unprecedented in any developed country in the world (Zatoński, Boyle, 1996). The decrease in premature mortality in middle age and constant post-war decrease in infant mortality (which accelerate after 1993) led to an increase in life expectancy between 1991-1996 of 2 years in males and 1.5 years in females (Central Statistical Office, 1997). In 1996 life expectancy reached its highest value ever (Central Statistical Office, 1997). These positive health developments are also noted in other Central and Eastern European (CEE) countries (the Czech Republic, Slovakia) (Bobak and Marmot, 1996). During the same period in Russia, however, life expectancy decreased by 8 years and premature death in young adults and in middle age increased sharply (figure 78).

### B. BACKGROUND MATERIALS AND METHOD OF ANALYSIS

This analysis is based on mortality statistics, as these are the only nation-wide health data set that have been gathered in a fairly standardised way over a long period. Data were obtained on Poland from publications of the Central Statistical Office, and on neighbouring countries (Germany, Sweden and Finland) from the World Health Organization (WHO) in Geneva (World Health Organization, 1997). Since the early 1960s (Holzer, 1970) mortality statistics for Poland have been published according to WHO standards. Data for Germany before the war refer to the entire country, whereas those after it relate only to Western Germany. Comparisons with other countries are based on estimates from the World Bank and WHO (Feachem and others, 1992; Murray and Lopez, 1994).

To increase the comparability of death statistics in different countries or in different periods, wide disease groups were often used—e.g. all cardiovascular diseases (ICD-9 390-459), all neoplasms (ICD-9 140-208) or all deaths from external causes (ICD-9 800-

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989). The main epidemiological measures used in our analysis include infant mortality rates, standardised mortality rates and average life expectancy.

Mortality rates for children below the age of one were calculated as the quotient of the number of infant deaths in a given year over the number of live births in that year, calculated per 1,000 live births. Standardised mortality rates are given per 100,000 people, assuming a constant (standard) population structure, which was usually the world standard population. Standardisation of mortality rates according to age per 100,000 people allows international comparisons and comparisons over time. When death rates in broad age ranges (e.g. 20-44, 45-64, 65 and over) are cited, these are themselves standardised to the relevant part of the world population. Average life expectancy is the average number of years that a person at a given age has yet to live. Thus, "average life expectancy at age 0" is the expected number of years a person will live at the time of birth.

#### C. TO WHAT DEGREE CAN HEALTH STATUS CHANGE?

In most countries of the world, developed as well as developing, there has been a constant decrease in mortality rates, except in times of war, famine or epidemic. In the United Kingdom, with the death rates of 1880, about 50 per cent of new-born infants could be expected to die before the age of 40 (Peto and others, 1992; table 44), but at the death rates of 1950, only 10 per cent would be expected to do so. This decline in United Kingdom death rates continued through the 1970s and 1980s, and at the death rates of the 1990s only 2-3 per cent of female and 4-5 per cent of male infants would expect to die before 40 (figure 60), and there has been a continuous increase in the fraction of the population that can expect to reach the age of 65 or 70 years. At United Kingdom 1880 death rates, 75 per cent of the population died before they reached the age of 70; but at United Kingdom 1990 death rates, this percentage had fallen to 30 per cent (Peto and others, 1992; table 44). In many developed countries like the United Kingdom, however, the decline was slower in the 1950s or 1960s than in the 1970s and 1980s (figure 60).

TABLE 44. PERCENTAGE OF DEATHS BEFORE A GIVEN AGE, UNITED KINGDOM, 1880 AND 1980

Age (years)	Year	
	1880	1980
< 40	50	3
< 70	75	30
< 100	~100	~100

In part, it was effective prevention of cardiovascular diseases and to some extent cancer that restored the decline in mortality in middle age in many western countries in the 1970s and 1980s. Such health transformations allow a progressively larger part of the new-born population to fulfil its biological potential and death before the age of 40—and in more and more West European countries, death before the age of 65 or 70—is no longer the norm but the exception. A look at medical statistics also shows, however, that biological limits to such progress exist: living for more than 100 years has always been exceptional, and so it remains (table 44).

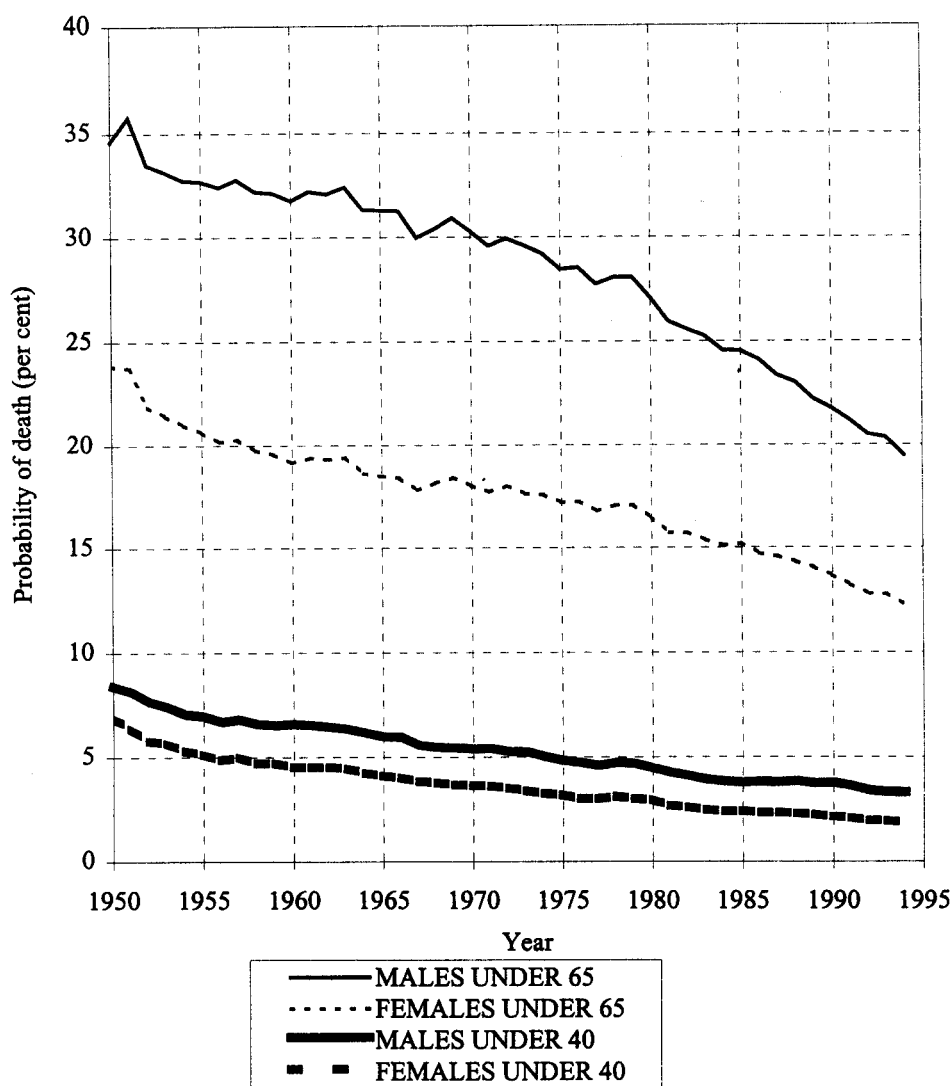
These historical epidemiological observations indicate that, independently of the complicated and variable influences of genetic structures, external factors can determine premature mortality on a population scale.

#### D. THE DEVELOPING HEALTH SITUATION IN POLAND

Health changes in Poland after the Second World War were marked by two important trends. First, there was a constant decrease in infant mortality, maternal mortality, and infectious disease mortality. Second, there was a constant increase, until the late 1980s, of manmade diseases (lung cancer and other tobacco-related diseases, cirrhosis of the liver, external death, i.e., due to accidental and other non-medical causes etc.).

When the Polish nation regained independence after the First World War, infant mortality rates remained high (130-150 per 1,000) and showed only slight improvement up to 1945 (figure 61). Infectious diseases were common. Life expectancy in the early 1930s was about 12 years shorter in Poland than in neighbouring Germany (Zatoński, 1995; figure 62).

**Figure 60. Probability of death under 40 and 65 years of age,  
Great Britain, 1950-1994**

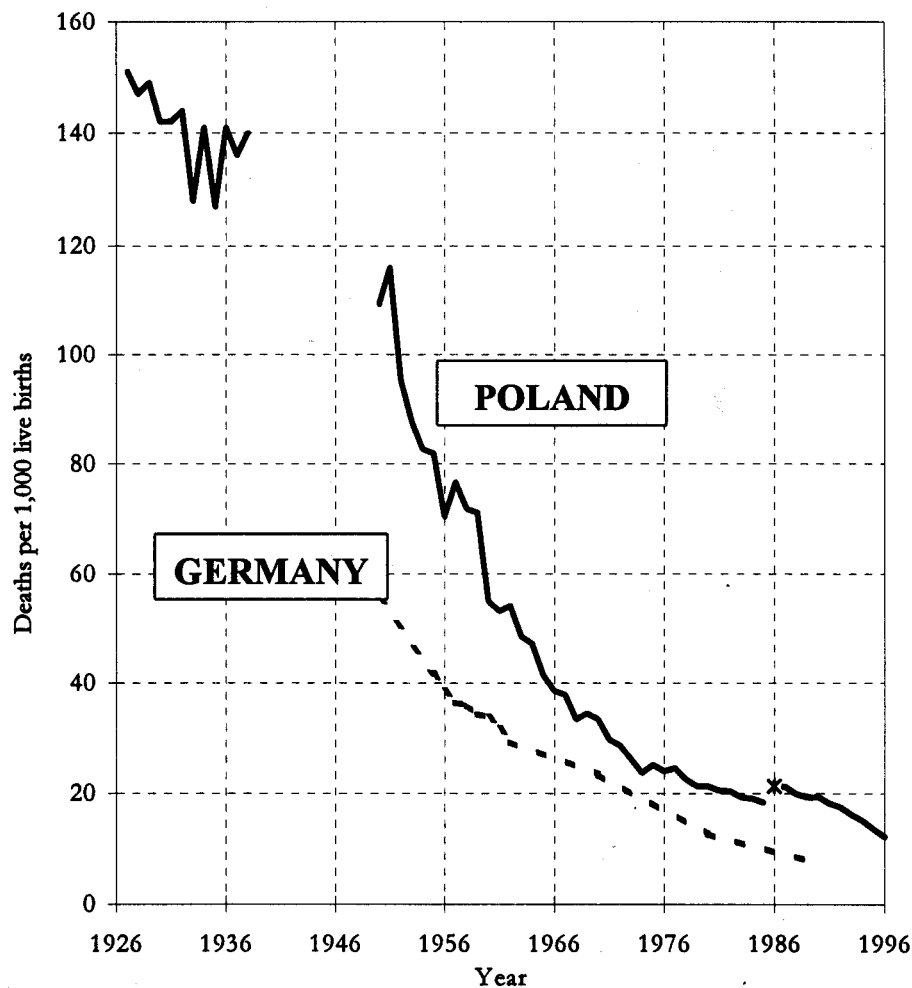


Source: World Health Organization, *WHO Mortality Data Bank* (Geneva, WHO, 1997).

In the first decade or two after the Second World War, the health transformation in Poland was proceeding relatively well. Significant success was achieved in the control of infectious diseases and in reducing infant mortality (Brzezinski and Szamotulska, 1996; figure 61) and maternal mortality (Troszynski and others, 1996). Various infectious diseases in children were reduced through the introduction of effective immunisation. Public health services were created, and health care for mothers and children was expanded. Epi-

demiological health indicators showed significant improvement until the mid-1960s, when the infant mortality rate was about 40 per 1,000, compared to 110 in 1950 (figure 61). Mortality rates from infectious diseases, including tuberculosis, fell drastically in all age groups (Magdzik and Czarkowski, 1994; figure 63). The health transformation came about faster in Poland and other countries of Eastern Europe between 1945 and 1965 than it had previously in many countries of Western Europe (Zatoński, 1995; Hertzmann, 1995),

Figure 61. Infant mortality (per 1,000 live-born), Poland and Germany, 1927-1996



\* since 1986, rates based upon WHO live-birth

Source: **Poland:** Central Statistical Office, *Rocznik Statystyczny* [Statistical Yearbook] (Warsaw, GUS, various years); **Germany:** World Health Organization, *WHO Mortality Data Bank* (Geneva, WHO, 1997).

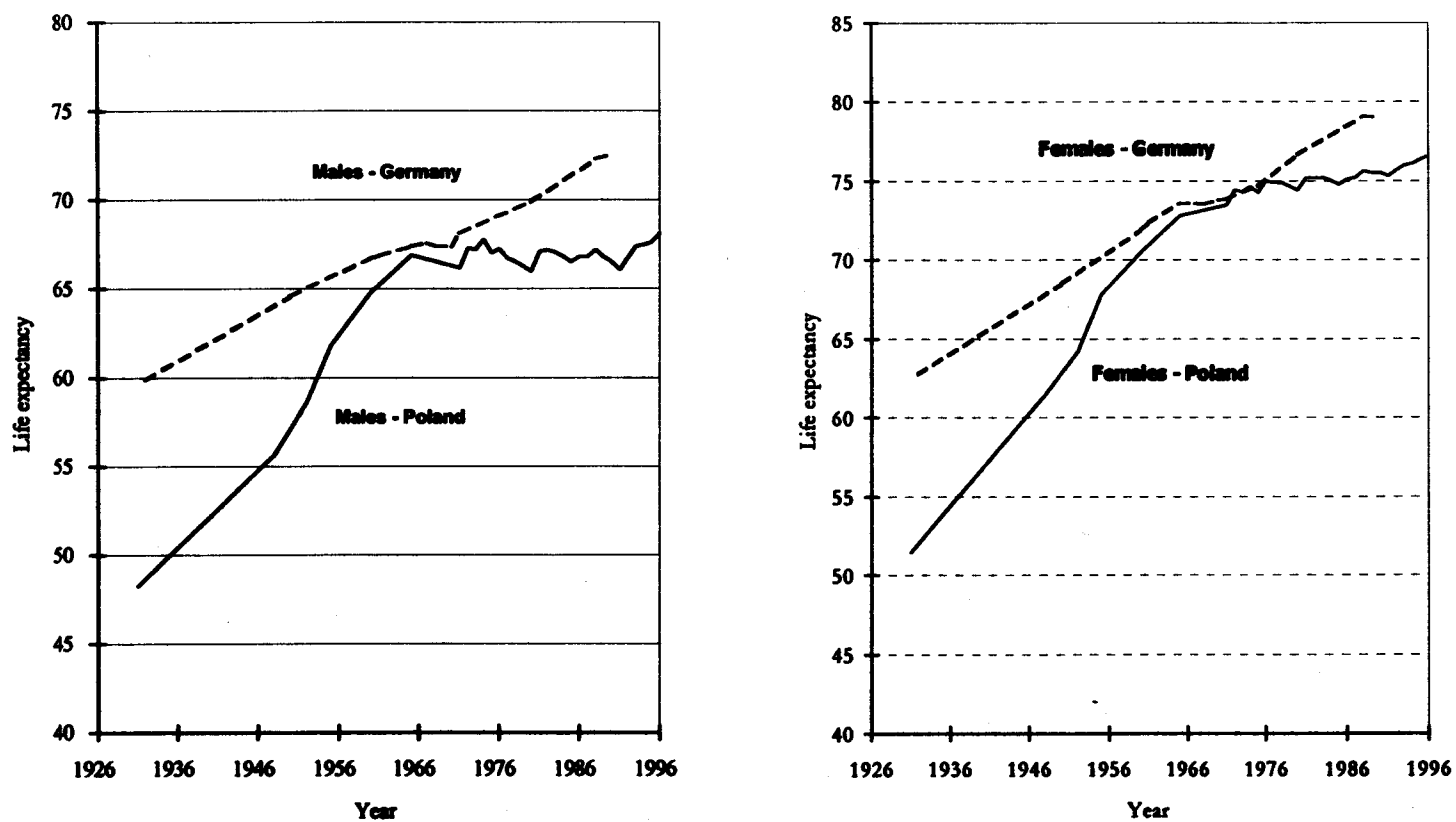
and in 1965 life expectancy was about the same in Poland as in Western Germany (Zatoński, 1995; figure 62).

Over the past few decades infant and child mortality has continued to decline and is now comparable to levels in many developed countries of the world, including neighbouring Germany (figure 61). Similarly, deaths from infectious diseases continued to fall in all age groups (figure 63) from the end of World War

II until the mid-1990s, and death from these causes is now rare (Magdzik, 1996). The corresponding morbidity and mortality rates are close to those in western developed countries (Murray and others, 1993). By the mid-1990s, infectious diseases account for only 0.5 per cent of all deaths (Magdzik and Czarkowski, 1994), of which slightly over one-half are due to tuberculosis (Leowski and Miller, 1993). In 1994, 50 deaths from AIDS were recorded in Poland, with 12 deaths in

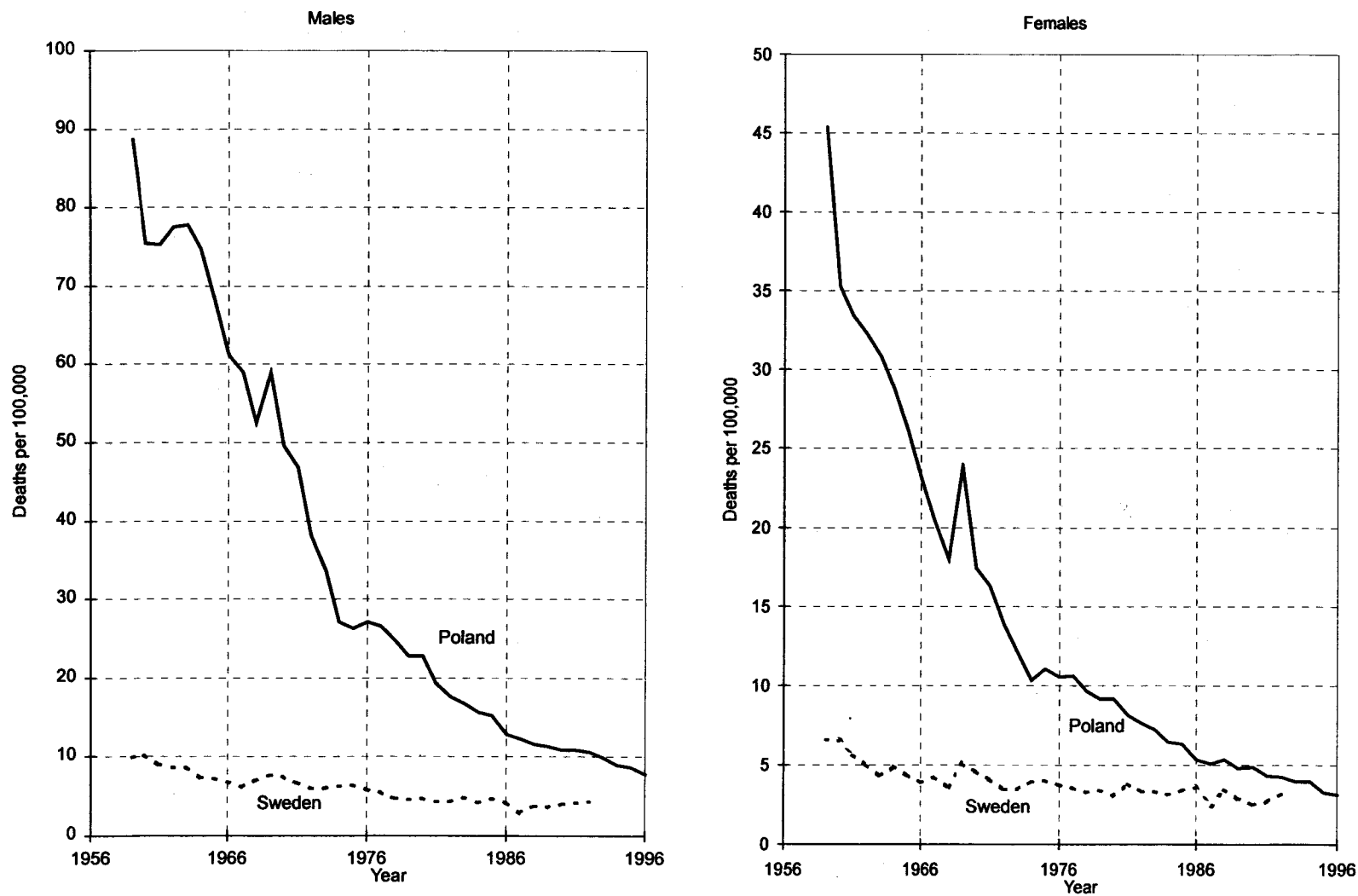


Figure 62. Average expectancy of life at birth, Poland and Germany, 1925-1996



Source: Poland: Central Statistical Office, *Rocznik Statystyczny* [Statistical Yearbook] (Warsaw, GUS, various years); Germany: World Health Organization, *WHO Mortality Data Bank* (Geneva, WHO, 1997).

Figure 63. Mortality trends from infectious and parasitic diseases, all age groups, Poland and Sweden, 1959-1996



Source: **Poland:** Central Statistical Office, *Rocznik Statystyczny* [Statistical Yearbook] (Warsaw, GUS, various years); **Sweden:** World Health Organization, *WHO Mortality Data Bank* (Geneva, WHO, 1997).

1991 and 24 in 1992 (Wojtyniak and others, 1996).

From the mid-1960s until the 1990s, the mortality trend among infants and children diverged from that among adults. The two trends cancelled each other out and produced a misleading "stabilisation" in life expectancy in Poland (figure 64). We must therefore consider the two conflicting trends separately in order to understand the true health situation.

While infant and child mortality has steadily declined (figure 64), as has mortality from infectious diseases in all age groups, chronic disease mortality in adults began to outweigh these gains. Since the late 1960s, the health transformation has clearly differentiated between people under 15 and those aged 15-59 (young and middle-aged adults). While a constant drop in death rates can still be seen in the first age category, as in other countries of Europe—at 1994 death rates, only 1 per cent of female and 2 per cent of male infants could expect to die before reaching 15 (figure 65).

In the early 1990s, the probability of death of boys and girls in the 0-14 age group in Poland (as in other CEE countries) was comparable to that in most developed countries of the world (Murray, Lopez, 1994) (figure 65). According to the World Bank, "infant mortality rates in the former socialist (FS) countries are lower than would be predicted given their income levels. However, this is consistent with global patterns; the infant mortality rate is negatively correlated with GDP per capita. This favourable outcome is largely attributable to superior achievements in primary school enrolment among females, childhood immunisation coverage (>90 per cent) and environmental sanitation in the FS countries" (World Bank, 1996).

#### *Increase in premature deaths among middle-aged Poles, 1965-1988*

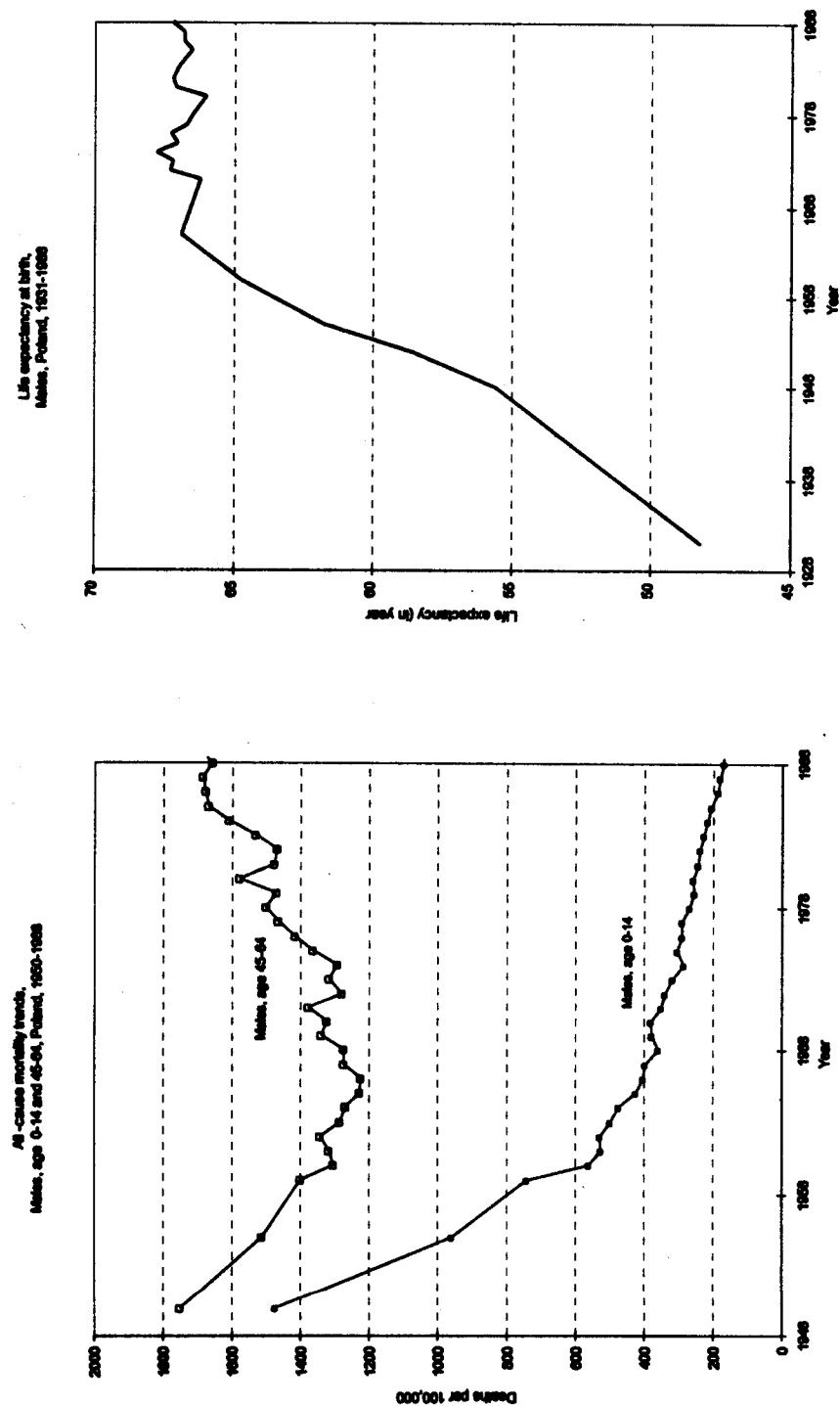
However, a second countervailing phenomenon also occurred in the post-war period. There was an increase in manmade diseases. Lung cancer and other cancers related to smoking, mainly among men in middle age, reached one of the highest levels in Europe. Similarly, external deaths were very common (Zatoński, 1995). Cirrhosis of the liver rose

steadily from the end of WW II, especially among young adults (20-44), male as well as female (Zatoński, 1995). Cardiovascular disease mortality has been rising since the beginning of the 1970s, but only among men; among women, this measure remains stable (figure 68).

The standardised mortality rate for men aged 45-64 rose from 1230 in 1965 to 1660 in 1988 (figure 66). This extraordinary increase was dominated by cancer, external causes and cardiovascular disease. In the late 1980s, mortality rates from these causes in middle age were some of the highest ever recorded world-wide; similar conditions were observed in other countries of Eastern Europe (Zatoński and others, 1996a): about 40 per cent of the deaths in middle age were due to cardiovascular disease, 30 per cent to cancer and almost 10 per cent to external causes. Poland entered the 1990s in a catastrophically bad state of adult health; for Polish men it was worse than immediately after the War (figure 66). When compared with the situation in other nations of the world, it was about the same as in India or sub-Saharan Africa (figure 67; Murray and Lopez, 1994; Zatoński, 1995) and was much worse than in other less developed regions. Likewise, based on the work of Murray and Lopez, at the death rates of 1990 the chances of a 15-year-old boy in Poland (and most former socialist countries) living to the age of 60 were worse than those of a boy of the same age in China or Latin America, similar to those of a 15-year-old boy in India, and better only than those of a boy in sub-Saharan Africa (figure 67). In Poland, more than 25 per cent could expect to die at ages 15-59, whereas in Sweden only 12 per cent do so (Feachem and others, 1992). This turnabout of the health situation stemmed from man-made causes, acting either directly (external causes) or indirectly (chronic degenerative diseases resulting from lifestyle).

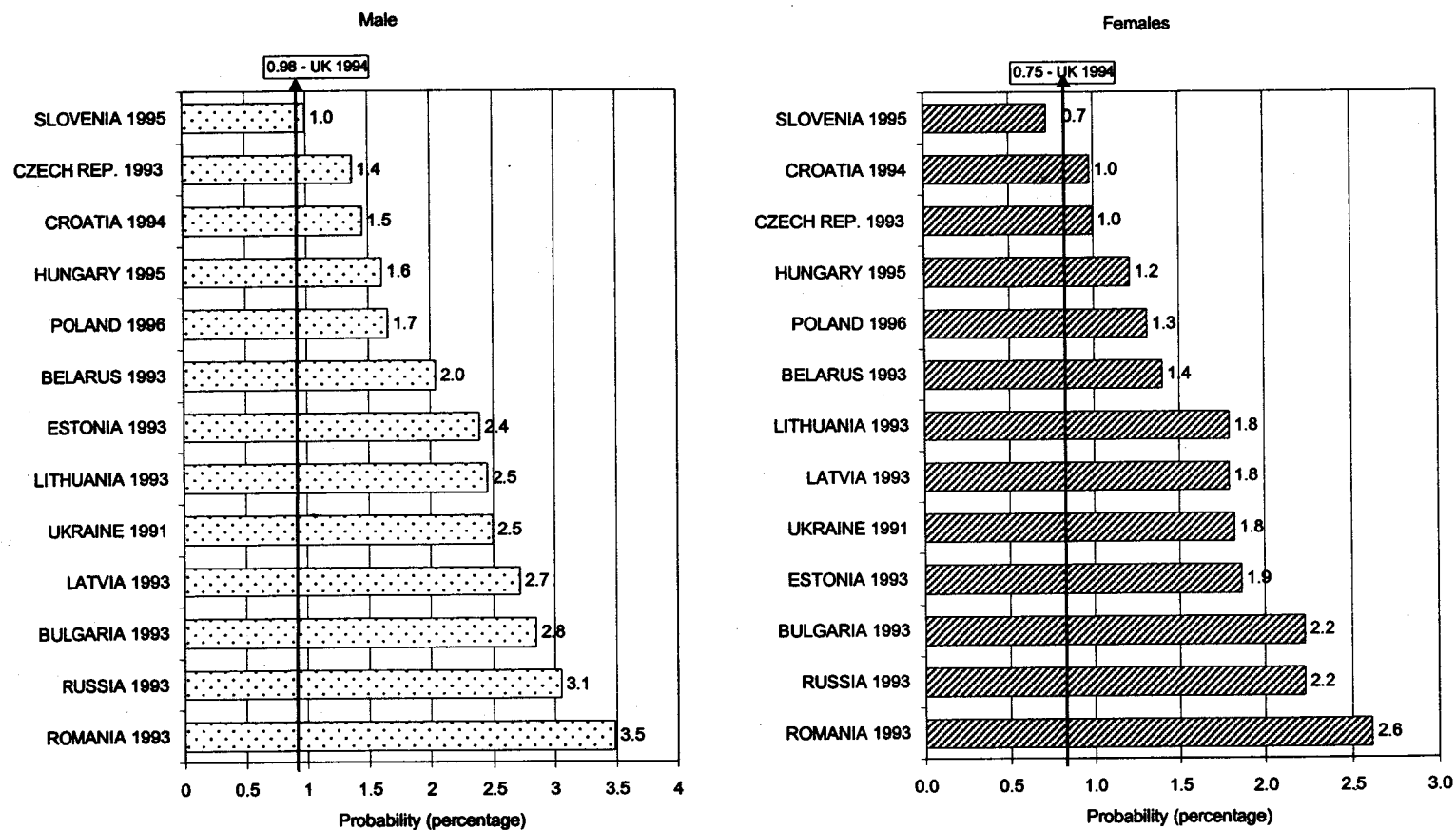
The increase in mortality from cardiovascular diseases and cancer in middle-aged men continued to the end of the 1980s. By then, the rate of mortality from cancer among men was significantly higher than any rate ever observed in Western Europe (Zatoński and others, 1996a; figure 68). At the same time that these recent changes were occurring in Poland, mortality from cardiovascular disease and cancer among both men and women in

Figure 64. Trends in all-cause mortality, at selected ages and expectation of life at birth, males, Poland, 1950-1998



Source: Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years).

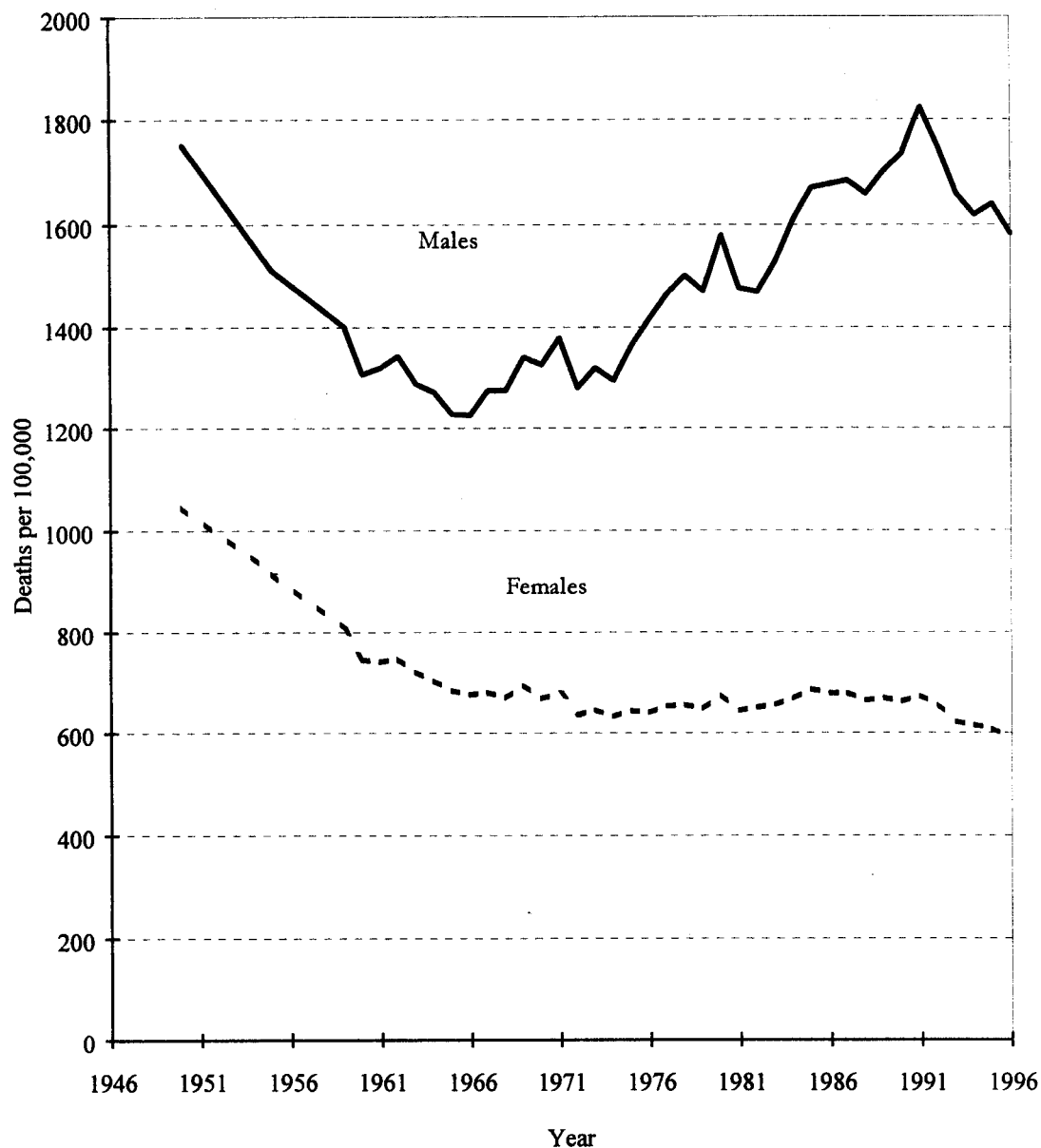
Figure 65. Probability of death in central and eastern Europe\*, by sex, age 0-14



\*The last year for which data is available

Source: World Health Organization, *WHO Mortality Data Bank* (Geneva, WHO, 1997).

Figure 66. Trends in mortality from all causes, age 45-64, Poland, 1950-1996



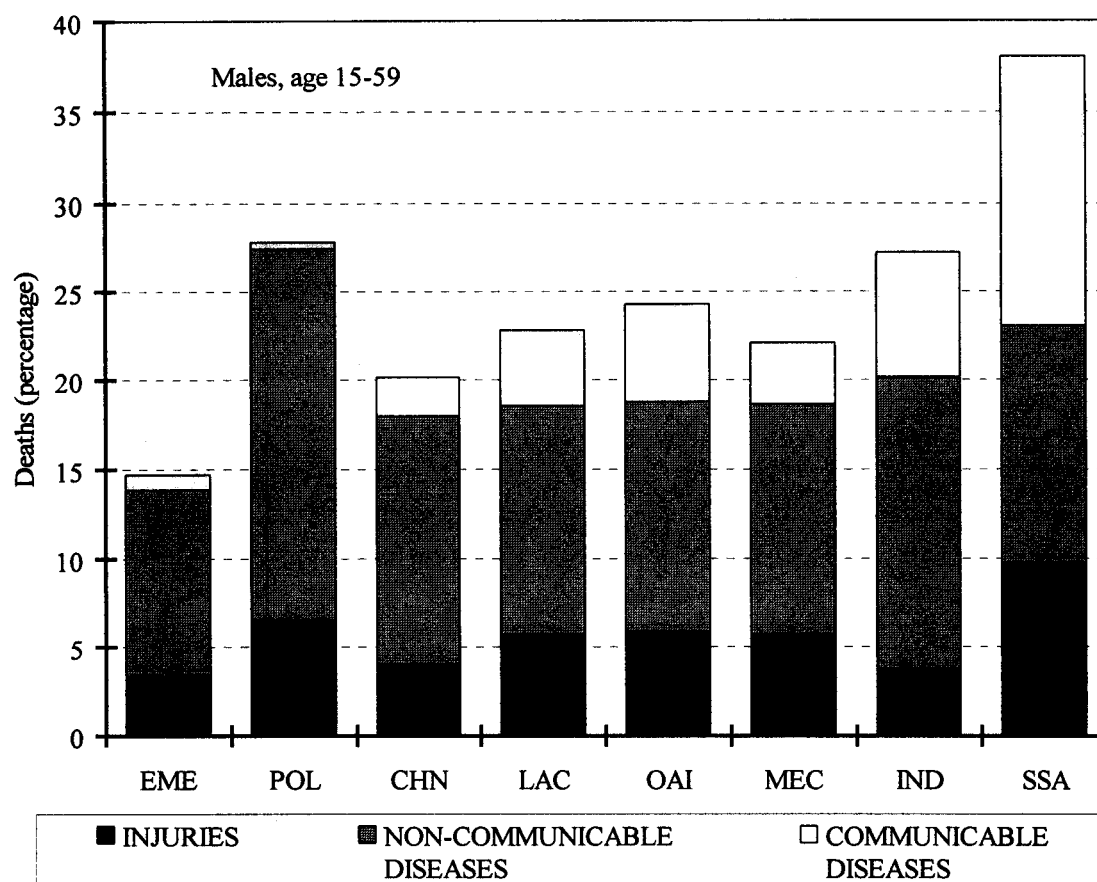
Source: Central Statistical office, *Rocznik Statystyczny* [Statistical Yearbook] (Warsaw, GUS, various years).

middle age in many Western European countries was decreasing (Thom and others, 1992; Rywik and Broda, 1996; figure 68).

Mortality from external causes rose steadily as well (figure 69). At the beginning of the 1980s, there was a sudden drop; then, a temporary rise was followed by stabilisation. The sudden drop observed in 1980-1982 seems to have resulted from the politico-socio-

economic crisis of that time, which led, especially during the years of martial law, to a social inertia. There were restrictions on travel, traffic lessened considerably, people worked more slowly, alcohol sales were regulated, and social life was under the strict control of the police and the army. With the lifting of martial law, mortality rates from external causes began returning to "normal" (figure 69).

Figure 67. Probability of dying, by region, 1990



EME - Established Market Economies

POL - Poland

CHN - China

LAC - Latin America and the Caribbean

OAI - Other Asia and Islands

MEC - Middle Eastern Crescent

IND - India

SSA - Sub-Saharan Africa

Source: Adapted from C. Murray and A. Lopez, "Global and regional cause-of-death patterns in 1990", *Bulletin of the World Health Organization* (Geneva, vol. 72/3, pp. 447-480, 1994).

Mortality rates for middle-aged women have stagnated in the last two decades, as their health transformation has merely been checked (figure 66). Mortality rates for cancer (figure 68) and externally-caused death (figure 69) and CVD (figure 68) remained stable. If Poland's situation is compared with that of the countries of Western Europe, where the death rates of middle-aged women are declining consistently, the observed stagnation

must be recognised as an indicator of unfavourable health processes (figure 68), especially since for certain diseases such as lung cancer and cirrhosis of the liver, there are substantial increases (Zatoński and others, 1996a).

Cirrhosis of the liver is another manmade disease that reached extraordinarily high levels in countries of CEE. In Poland this disease

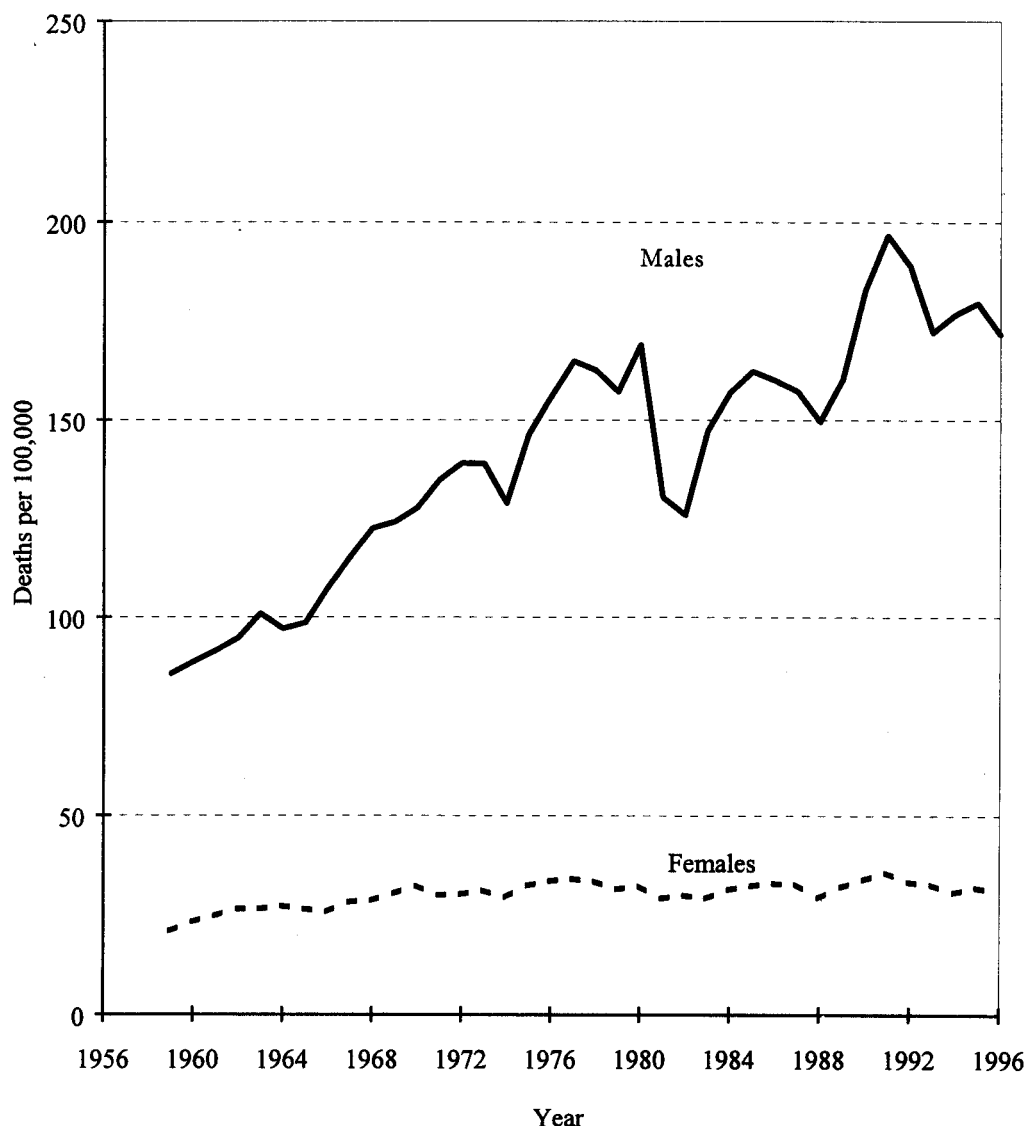
**Figure 68. Mortality rates from cardiovascular diseases and from all cancers, by sex, age 45-64, Poland, Finland and the United Kingdom, 1956-1996**



Sources: Poland: Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years); Finland and the United Kingdom: World Health Organization, *WHO Mortality Data Bank* (Geneva, WHO, 1997).



Figure 69. Sudden deaths from external causes, age 45-64, Poland, 1959-1996



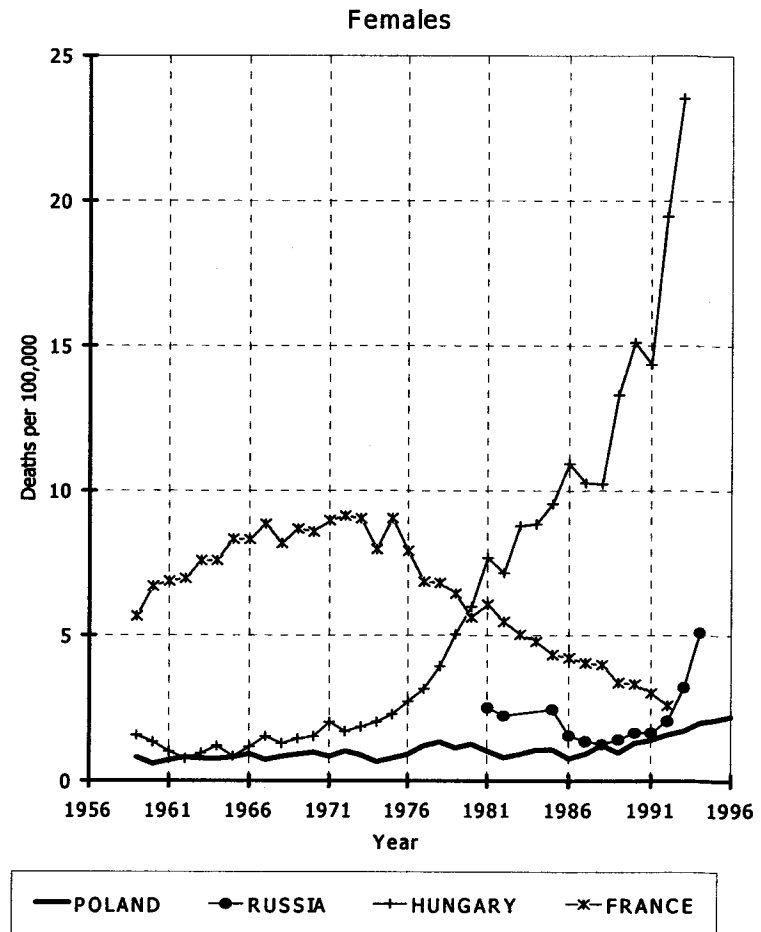
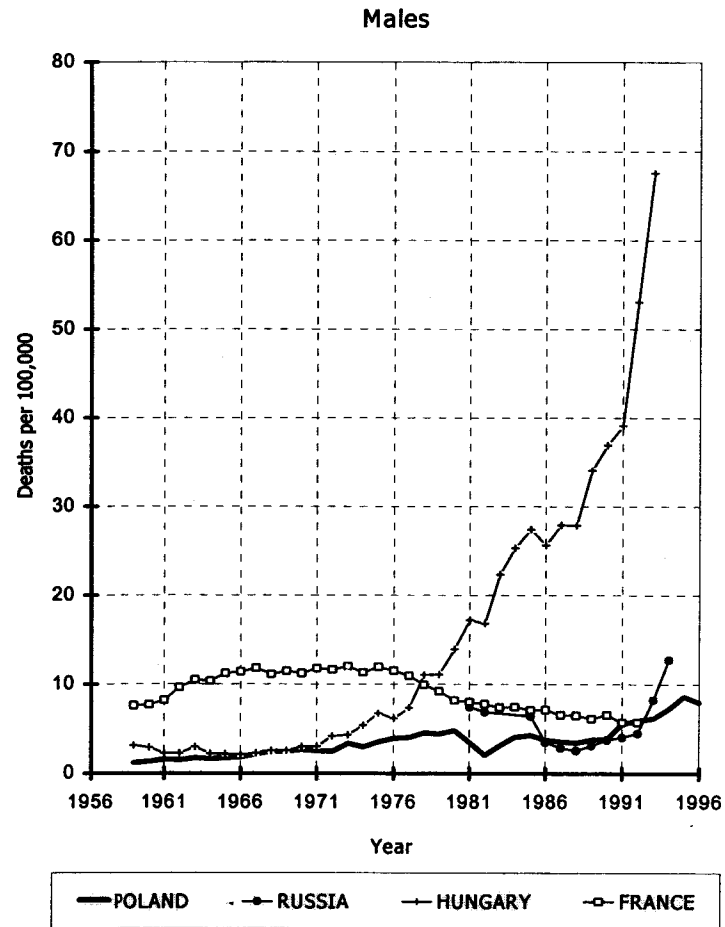
Source: Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years).

has been steadily increasing especially among young adults since WW II (Zatoński and others, 1996a). In Hungary, the disease has reached levels never before observed anywhere in the world. In young Hungarian adults (aged 20-44) cirrhosis of the liver accounts for 20 per cent of total mortality (Zatoński and others, 1996a) (figure 70).

What were the chief causes of this increase in premature mortality among men and the

stagnation of rates among women in middle age in Poland? Let us first try to eliminate all of the factors that did not determine the observed changes. No breakdown in health care services appears to have taken place (Zatoński and others, 1996a; Bojan and others, 1991). Medical care in Poland is of a standard appropriate to the socio-economic development of the country, and the improvement after the Second World War was commensurate with its possibilities. The number of doctors, the

Figure 70. Cirrhosis mortality, selected countries, by sex, age 20-44, 1959-1996



Sources: **Poland:** Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years); **Russia, Hungary and France:** World Health Organization, *WHO Mortality Data Bank* (Geneva, WHO, 1997).

level of their education and the numbers of available beds in health care facilities all increased substantially, and access to modern medicine improved. Thus, the numbers of doctors and hospital beds per 1,000 people in Poland are at the same level as (or higher than) in many countries of Western Europe (Feachem, 1994). The results of the work of the health services are also substantial, resulting in falling rates for infant, child and maternal mortality, fewer deaths before the age of 40, improved infectious diseases control and widespread child immunisation (Zatoński, 1995; Zatoński and others, 1996a).

Many factors can cause premature death. Epidemiological observations for recent decades seem to indicate that lifestyle factors might be particularly significant determinants of premature mortality among adults (Doll and Peto, 1981; Murray and others, 1993; table 45). Lifestyle, is, however, greatly underestimated as an avoidable cause of ill health. Meanwhile, the importance of genetic and iatrogenic factors, and environmental pollution are often considerably overestimated (table 46). Lifestyle factors include tobacco and alcohol consumption, dietary habits, exposure to harmful substances in the workplace and external determinants of internal factors such as obesity, high blood pressure, and blood biochemistry.

TABLE 45. ESTIMATED RISKS OF VARIOUS ACTIVITIES

<i>Activity or cause</i>	<i>Annual number of deaths per 1 million persons exposed</i>
Smoking	7 000 <sup>a</sup>
Alcohol	541
Accident	275
Disease	266
Motor vehicles	187
Alcohol-involved	95
Non-alcohol-involved	92
Work	113
Swimming	22
Passive smoking <sup>b</sup>	19
All other air pollutants <sup>b</sup>	6
Football	6
Electrocution	2

Source: Active smoking, CPS-II; NHISs 1965, 1985; U.S. Bureau of the Census (1974, 1986).

<sup>a</sup>Number of deaths per million smokers who began smoking before 1965.

<sup>b</sup>Cancer deaths only.

The work of Peto and collaborators (1992, 1994; Doll and others, 1993) suggests that

TABLE 46. THE MOST IMPORTANT FACTORS INFLUENCING HUMAN HEALTH IN THE OPINION OF ADULT POLES IN 1995<sup>a</sup>  
(percentage)

	<i>Men</i>	<i>Women</i>
Environment	62.5	61.8
Dietary habits	52.2	56.8
Stress, hectic lifestyle	41.4	49.5
Tobacco smoking	26.5	26.9
Genetics	25.0	25.1
Sport, physical activity	29.2	18.2

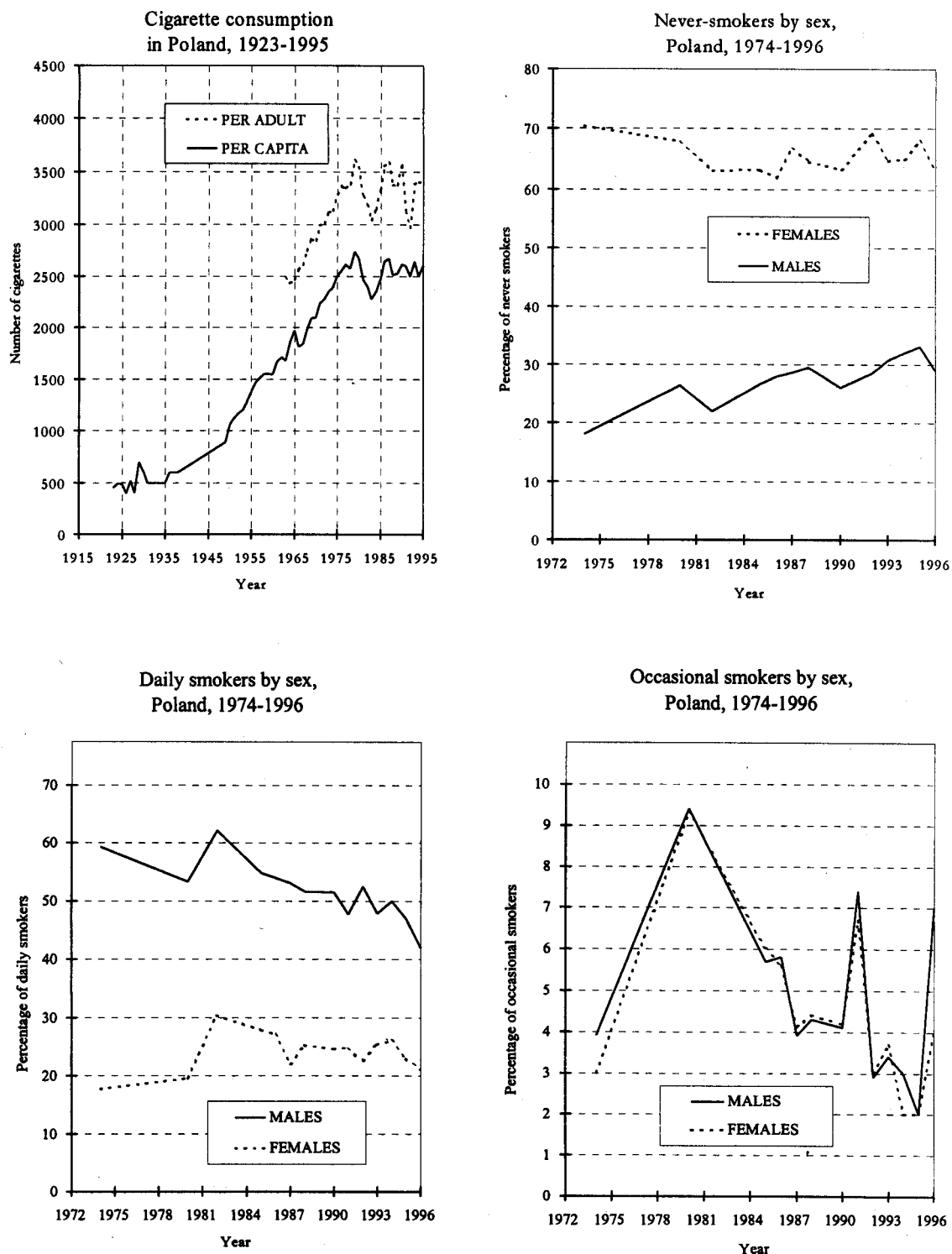
<sup>a</sup>Based on sociological studies conducted on a representative sample of the Polish population aged 15 years and above (N=1391) by RUN at the request of the Centre of Oncology and the Gazeta Wyborcza newspaper on 25-31 November 1995.

about 40 per cent of the male Polish deaths in middle age in the late 1980s were due to tobacco smoking. Tobacco consumption had risen dramatically and consistently up to the end of the 1970s, and had stabilised at the beginning of the 1980s at one of the highest levels in the world (Zatoński, 1996). In the mid-1970s, only 18 per cent of adult men had never smoked tobacco (figure 71). Cigarettes sold in Poland contained relatively high levels of tar and nicotine (Zatoński and Przewozniak, 1996; Przewozniak and Zatoński, 1996; Zatoński and others, 1996b).

Alcohol is another important determinant of risk. It is estimated that 3-4 million Poles drink vodka every day or almost every day, often reaching a state of intoxication (Sieroslawski 1992, 1995; Habrat and Puzynski, 1996). Apart from the role alcohol plays in liver disease and upper aerodigestive cancers, its consumption is probably also an important indirect cause of death from external causes (Mlekodaj and others, 1994; Kopczynski and Gorynski 1996). During the entire post-war period, Poland had a promotional policy regarding tobacco and alcohol, considering these products good sources of revenue. Smoking tobacco, drinking vodka, driving while intoxicated and the presence of intoxicated people on the road are socially accepted. The anti-alcohol act, introduced during the time of political crisis in the early 1980s, was generally not obeyed.

Another danger is the unhealthy diet, typified by a high intake of animal fat and low consumption of fresh fruits and vegetables, especially in winter and spring (Sekula and others, 1992; Sekula, 1995; Sekula and others,

Figure 71. Evolution of cigarette smoking in Poland according to several indicators, 1923-1996



Source: Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years).

1996). The closed market manipulated by the government and the restricted availability of products precluded much change in this dietary structure.

Other potential causes are poor workplace hygiene, and general environmental pollution, particularly air pollution (McMichael, 1996). Despite the development of occupational medicine, prompted by political doctrine, technological delay and lack of capital to introduce "clean" technologies make industrial hazards more of a threat than in Western Europe (Indulski and Szeszenia-Dabrowska, 1996). The role of environmental pollution in the risk of cancer has, however, often been overestimated, principally by the public. As in other countries, pollution in Poland is probably a cause of only a few percent of tumours (Doll and Peto, 1981; Zatoński and others, 1993, McMichael and Zatoński 1996), as confirmed by an epidemiological study in regions of heavy air pollution in Poland (Jedrychowski and others, 1989).

## E. MORTALITY RATES AFTER 1988

### 1. Phase I

In 1989-1991, another significant increase in overall mortality rates was noted, mainly among men aged 15-64. The increase was due primarily to a higher rate of externally-caused deaths (but cirrhosis of the liver and CVD as well), with an increase of almost 25 per cent between 1988 and 1991. Among women aged 15-64 the corresponding increase in deaths from non-medical causes (ICD 800-999, which includes traffic and other accidents) was almost 18 per cent, but as the absolute rate was small, the absolute increase was also small. Hence, the absolute increase in non-medical deaths affected mainly men and resulted in almost 5000 additional deaths per year (Zatoński and others, 1996a).

The factors that led to the increase in premature deaths among young and middle-aged adults, particularly men, may well involve diffuse causes such as the exceptional animation of society during 1989-1991, with increased social mobility, a sudden increase in the number of cars, a feeling of unrestricted freedom (but without the skills or culture to make proper use of it), and the availability of

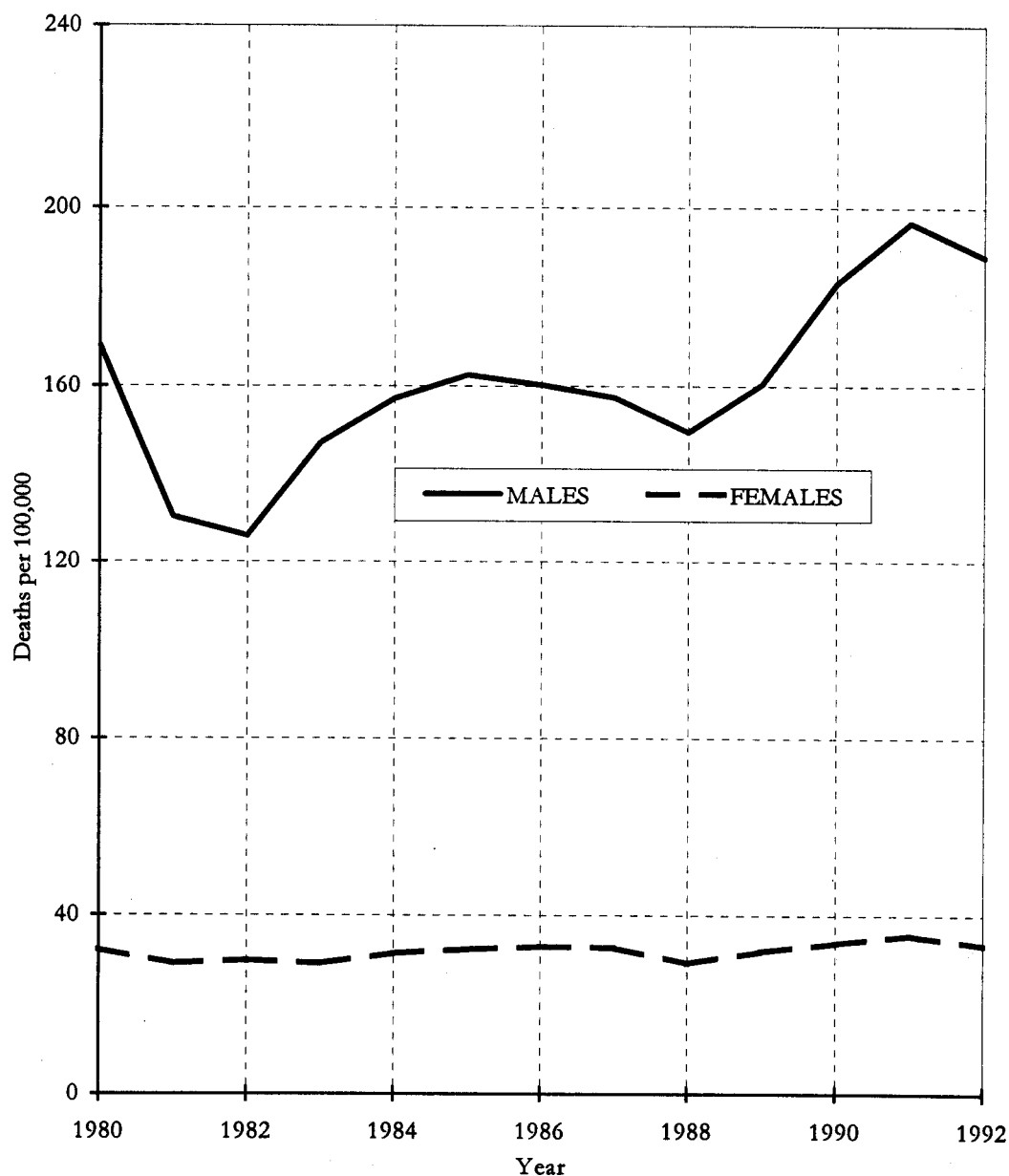
rapid forms of transport, together with a lack of police authority and traffic control during the country's reorganisation (figure 72).

Another important cause, however, was easier access to cheap alcohol. The fall of the previous government structure in 1988-1989 and lack of control by the new political leaders contributed to the inflow of millions of litres of cheap, duty-free, concentrated alcohol. Taking into account indirect indicators such as first-time alcohol psychoses, Sieroslawski (1992) estimated that alcohol consumption during this time leapt by about 75 per cent from 6 litres to 10-11 litres of 100 per cent alcohol per capita annually. After 1991, the increase stopped, and by 1995 alcohol consumption had fallen somewhat, to 8.5-9 litres per capita (Sieroslawski, 1995; Habrat and Puzynski, 1996).

A similar increase in premature mortality in young and middle-aged adults was observed in all the countries of Eastern Europe during the period of political and economic transformation. In Eastern Germany, the effect was much less marked and lasted for only two years, 1989 and 1990. In the Czech Republic, the increase was associated mainly with deaths from traffic accidents among young men (WHO, 1995). In the countries of the former Soviet Union, the increase in premature mortality between 1991 and 1994 owing to deaths from cirrhosis of the liver (120 per cent), external death (100 per cent for males, 70 per cent for females) and CVD (70 per cent for males, 40 per cent for females) follows a decidedly more dramatic course (figure 73). In the Russian Federation between 1991 and 1994, a huge increase in premature mortality was noted, producing a sudden decrease of 7 years in male life expectancy (World Health Organization, 1995; Chenet and others, 1996).

Alcohol consumption in Poland, as in other countries of Eastern Europe, has a particular negative effect on public health. While in the Mediterranean and Anglo-Saxon countries alcohol consumption is characteristically spread out over time, in Poland the same total consumption is often associated with isolated episodes of binge-drinking of hard liquor, mostly vodka, which leads to severe alcoholic intoxication. Of course, the protective influence of drinking moderate amounts of alcohol every day on cardiovascular mortality, which

Figure 72. Sudden deaths from external causes, age 45-64, Poland, 1980-1992



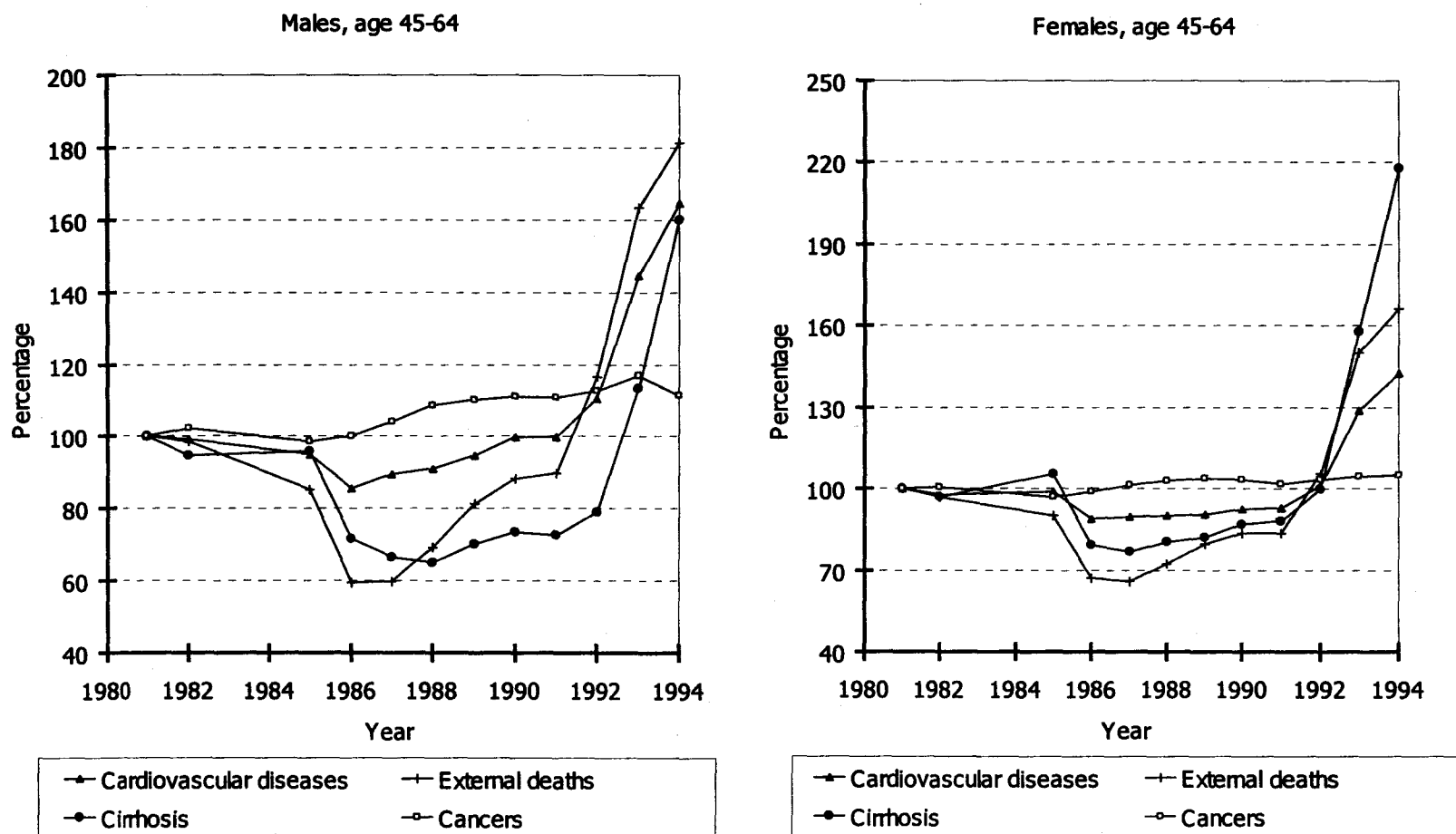
Source: Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years).

has been reported in many epidemiological studies, does not exclude Eastern Europeans.

It seems however that in the CEE countries, especially those where vodka is the main alcohol consumed (Poland, former USSR countries, etc.) the style of drinking is just as important a factor as the amount of alcohol consumed. WHO estimates the amount of alcohol consumed in Latvia in the mid-1990s at 22 litres, including 16 litres unrecorded

consumption (WHO, 1997). Apart from diseases whose relationship to alcohol has long been acknowledged (cirrhosis of the liver, external deaths) it appears that binge drinking is a risk factor in lethal cardiac death. Observations in Poland and Russia appear to confirm this. Huge fluctuations in alcohol consumption in these countries lead to fluctuations in deaths from cirrhosis of the liver and external deaths, but also to unusual jumps in CVD mortality (increases as well as

Figure 73. Changes in standardized mortality rates from selected causes (expressed as a percentage, 1981=100%), Russia, 1981-1994



Source: World Health Organization, *WHO Mortality Data Bank* (Geneva, WHO, 1997).

decreases), especially in Russia (figure 73). The extent of this increase in CVD deaths observed in Russia (and other countries of the former USSR) in 1987-1994 is a world record. CVD deaths in middle-aged men in Russia rose from 737/100,000 in 1987 to 1356/100,000 in 1994 (World Health Organization, 1997). The interpretation of the Russian situation (and that of other countries) can usefully be approached in light of a prospective cohort study done on alcohol-drinking men in Finland. This study demonstrated that after adjusting for the amount of alcohol consumed, the risk of lethal myocardial infarction was greatly influenced by the style of drinking. Drinking large quantities of alcohol in one session (6 or more bottles of beer as opposed to 3 or fewer) increased risk of lethal myocardial infarction 6.5 times ( $RR=6.5$ ) (Kauhanen and others, 1997).

## 2. Phase II

Unexpected changes in adult health occurred in Poland after 1991. All health indicators then began to signal a return to the normal health transformation (decreases in infant and child mortality, but also in premature adult mortality). Premature adult mortality decreased considerably, mainly as a result of decreasing CVD mortality (but also external death and cancer). This phenomenon also gained full momentum in about 1990 in the Czech Republic (World Health Organization, 1997).

One of the best and clearest illustrations of this phenomenon has been a consistent decline in overall annual mortality since 1991 in Poland. The combined total for the years 1991-96 was 77,000 fewer deaths than expected, based upon 1991 projections (Central Statistical Office, 1997). For the first time in nearly twenty years, life expectancy began to increase. The trend in adult mortality turned downward to once again parallel the continuing decline in infant and child mortality. In this period life expectancy among men rose by 2 years and among women by 1.3 years. For the first time since World War II this indicator improved faster among men than women. It is now at its highest level in Polish history among both genders (Central Statistical Office, 1997). In 1991-1996, the overall mortality rate decreased from 1062 to 971 per 100,000 for men and from 550 to 519 per

100,000 for women, with particularly striking effects in certain age groups. The mortality rates fell in all age groups but especially for people who were under 65 years of age. The decrease was due primarily to a fall in deaths from cardiovascular diseases: by 30-35 per cent in the 20-44 age group and by almost 20 per cent in the 45-64 age group; for people aged over 65, however, the decrease was only 10 per cent (figure 74; Zatoński and others, 1996a). Similarly, CVD mortality has been falling dramatically in the Czech Republic and Slovakia (figure 75).

The two remaining main causes of premature mortality, cancer and accidents, played less significant roles in the decrease in mortality rates in 1991-1996. The steady increase during the past few decades in male lung cancer deaths halted and, among men aged 20-44, lung cancer mortality has at last begun to decline (figure 76). In the 45-65 age group, the increase was simply checked however, and lung cancer mortality rates are still rising in men over 65. In women of all ages, the number of deaths from lung cancer rose during the same period (Zatoński and others, 1996a). Similarly, sudden deaths from non-medical causes, having reached a maximum in 1991, tended to decline slightly in men and women of all ages over the next few years (Zatoński and others, 1996a) (figure 69).

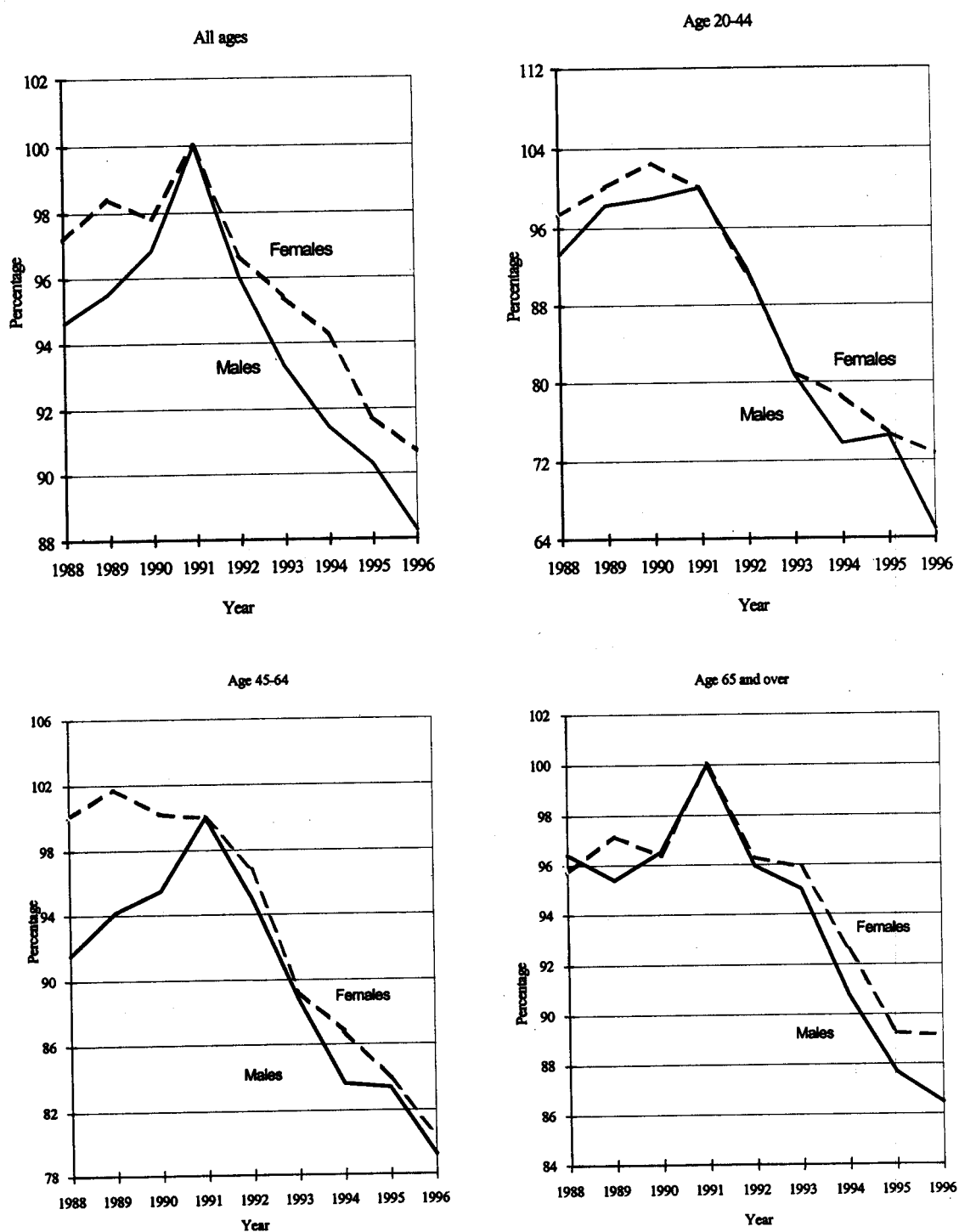
It is worth stressing that in the period of health improvement (1991-1996), mortality rates among infants (figure 61) and children (Central Statistical Office, 1997), and from infectious diseases (figure 63), all showed declines.

A marked decline in the birth-rate (Central Statistical Office, 1997) has occurred during Poland's economic transformation. At the same time the rate of low-weight births (<2,500g) also declined sharply, as did infant mortality.

Until the end of the 1980s, the development of the health situation was similar in Poland and its neighbouring countries. The rates of death in middle age, particularly among men, were very high throughout the region (Murray and Lopez, 1994). After 1990, however, health indicators in these countries began to move in various directions. In Poland, the Czech Republic and Slovakia, after brief tran-

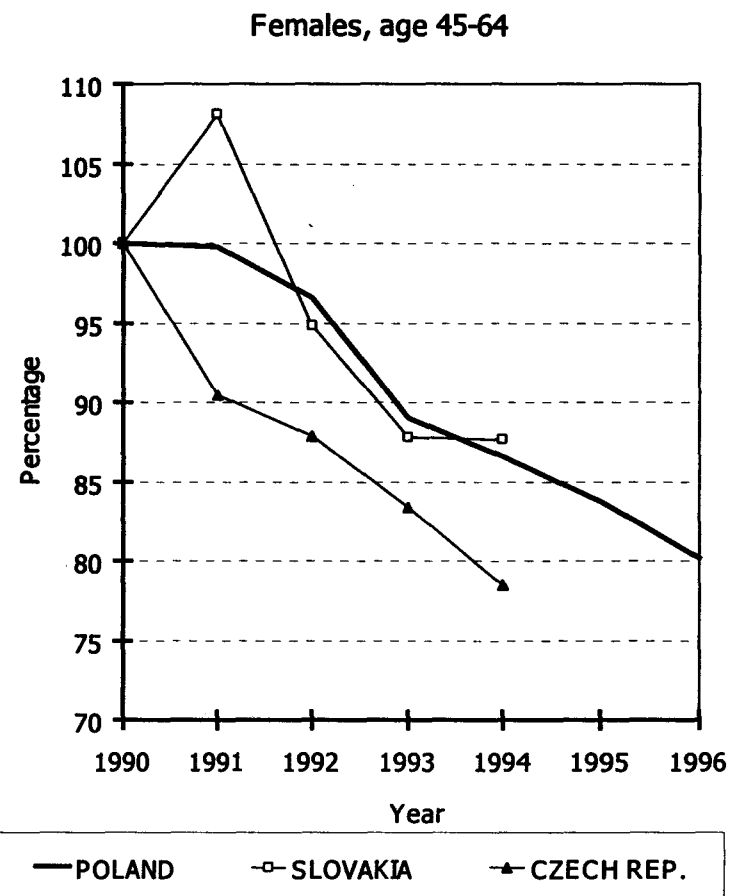
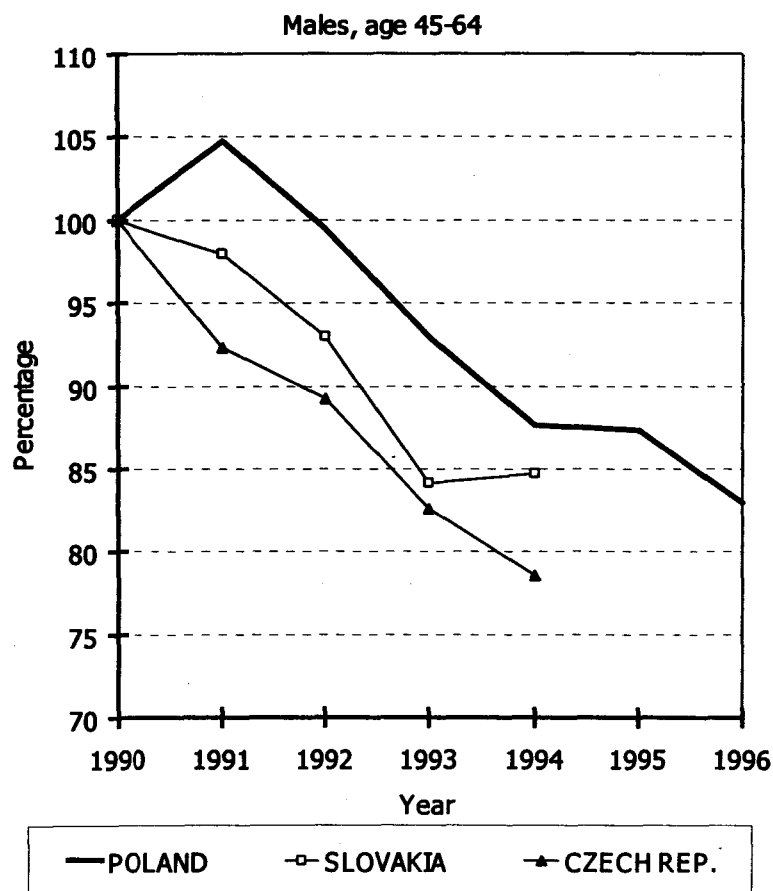


**Figure 74. Changes in mortality from cardiovascular diseases (expressed as a percentage, 1991=100%), by sex, Poland, 1988-1996**



Source: Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years).

**Figure 75. Changes in standardized mortality rates from cardiovascular diseases (expressed as a percentage, 1990=100%), Poland, Czech Republic and Slovakia**



Source: **Poland:** Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years); **Czech Republic and Slovakia:** World Health Organization, *WHO Mortality Data Bank* (Geneva, WHO, 1997).

Figure 76. Trends in mortality from lung cancer, age 20-44, Poland, 1963-1996



Source: Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years).

sition periods, premature deaths began to decline. At the same time, in most countries of the former Soviet Union, the corresponding figures increased rapidly in both sexes. For example, life expectancy figures among men, which had been at similar levels in Russia and Poland as recently as 1988, diverged so quickly that in 1994 they already differed by 10 years. Large divergences also appeared in rates of premature death in young adults and in middle age (figure 77).

## B. CAUSES OF THE CHANGES IN MORTALITY RATES AFTER 1991

The significant decrease in mortality rates, especially the large decrease in premature deaths from cardiovascular diseases, was unexpected.

### 1. *Medical and demographic statistics*

The quality and completeness of data on causes of death might first be questioned. In principle, the decrease in overall mortality could be due to incomplete data, but there has been no indication of drastic changes in the quality, completeness or accuracy of the documentation of deaths. Regulations in force in Poland since 1962 prohibit disposal of dead bodies in the absence of a written death certificate registered by the Office of Civil Status (Holzer, 1970), and there has been no change in death registration regulations. The method used to code causes of death has not changed since 1980; the Ninth Revision of the International Classification of Diseases, Injury, and Causes of Death is still in use. This allows comparisons over the period 1980 to 1994 for groups of illnesses as well as for specific causes of death. Although changes may have occurred between the vascular categories 410-429 and 440-459, changes in mortality attributed to cardiovascular diseases as a whole (390-459) cannot be due to diagnostic or coding errors, as there is no other category of causes into which such a large number of vascular deaths could be transferred, and there is no evidence of any such transfer. The number of deaths attributed to the "cause unknown" (ICD-9, 797-799) shows no new trends. Likewise, the differentially changing rates for lung cancer, falling for young men and rising for women, do not indicate any systematic change in the quality of coding of

causes of death in Poland, and the rates for other cancers are generally constant (Zatoński and Tyczynski, 1998).

Since the 1980s, the population of Poland has been progressively overestimated by the Central Statistical Office because illegal emigration was not taken into account in the statistics, i.e. people who emigrated from Poland but did not register their departure were not discounted. This overestimation concerns principally people under 55 years of age. Okolski (1994) estimated that in 1989 the Polish population was overestimated by 2.2-2.9 per cent. Such overestimation would result in underestimated mortality rates calculated on the basis of the population, but the errors involved are small in comparison with the main contrasting changes in the cause-specific mortality rates.

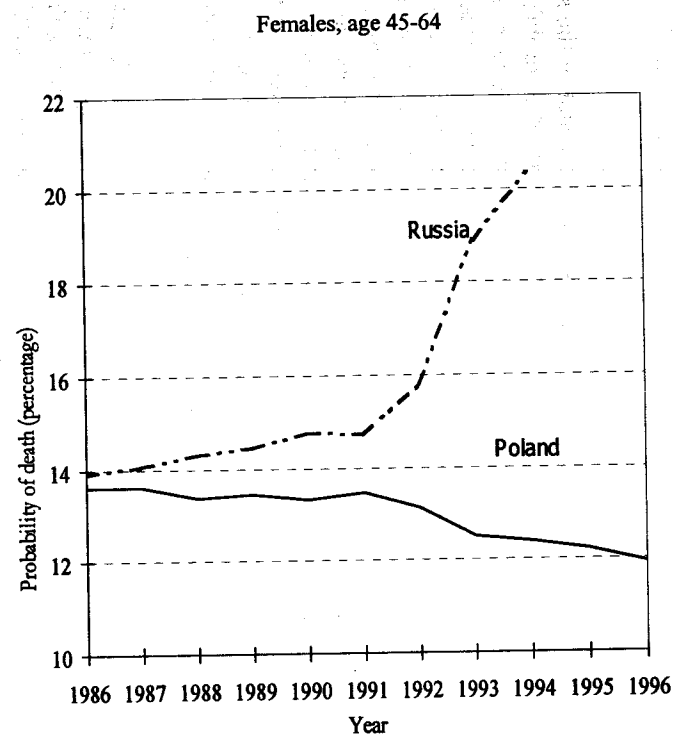
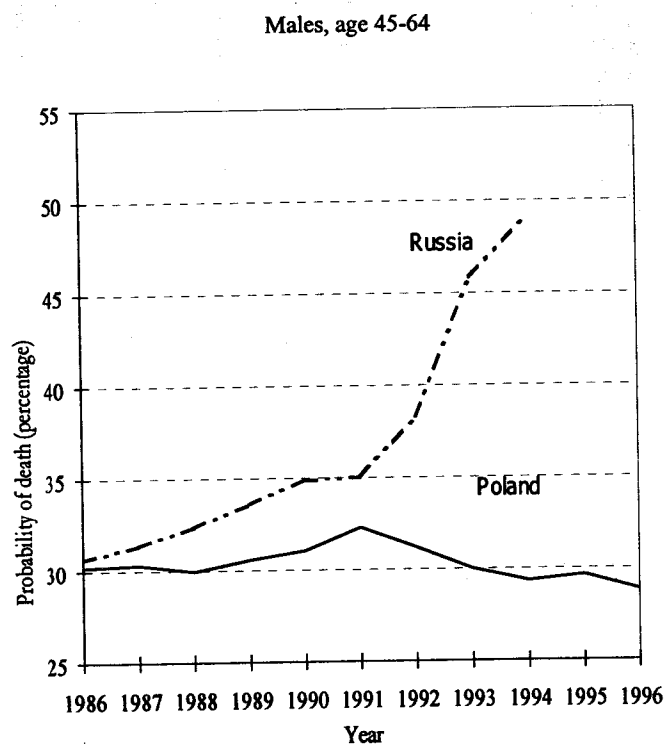
Thus, no important changes have taken place in recent years in the completeness or quality of statistics of death that could explain the main patterns of change in Polish mortality rates over the past few years.

### 2. *Changes in lifestyle*

If it is accepted that premature mortality, especially from cardiovascular disease in young and middle-aged adults, is really decreasing, what are the main causes? Is it only a temporary stalling of the process of increasing premature mortality, or is it a return to the health transition, discontinued in Poland in the 1960s and 1970s? Can the newly favourable trends be continued?

Changes in health status are determined by many factors, with complicated interactions. Scientific literature tends to agree that the poor health status of the Poles (and the inhabitants of other countries of Eastern Europe) results primarily from factors associated with lifestyle (Zatoński and others, 1988; Peto and others, 1992; Feachem, 1994; Zatoński, 1995; Bobak and Marmot, 1996; Chenet and others, 1996; McMichael and Zatoński, 1996). Important changes in health risk factors appeared with the introduction of a market economy after 1989, primarily associated with diet. Paradoxically, tobacco and alcohol remained (at least in theory) under the jurisdiction of the Government, although it was now a new, democratically elected one.

**Figure 77. Probability of death, Poland and Russia, 1986-1996**



**Source:** **Poland:** Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years); **Russia:** World Health Organization, *WHO Mortality Data Bank* (Geneva, WHO, 1997).

Both the Government and society passively tolerated an incredible degree of alcohol smuggling. The democratically elected Parliament tried to weaken the first positive effects of anti-alcohol legislation from the early 1980s, and the politico-social Friends of Beer Party was surprisingly successful. The law that bans alcohol advertising in Poland was transgressed openly, in full view of society and constitutionally appointed institutions such as the Ministry of Justice and the Citizens' Rights Advocate. This situation led to a drastic increase in alcohol consumption (figure 78) and to the availability of alcohol to children and the young.

Exposure to tobacco has followed a different path from that of alcohol in Poland. In contrast to the case of alcohol, positive changes had gradually begun to emerge over the past 20 years in society's attitude towards smoking. The steady increase in tobacco consumption after the end of the Second World War stopped in the late 1970s (figure 71), and tobacco consumption has since stabilised at sales of about 95 billion cigarettes per year. In terms of consumption per adult inhabitant, this figure still leaves Poland at or near the top of the international list, since consumption is falling in most countries (Przewozniak and Zatoński 1996; Zatoński and others, 1996b). However, some favourable trends are observed in the smoking patterns of men: most importantly the highest smoking prevalence of daily smoking (62 per cent) was in 1982, but the percentage has since decreased to about 45 per cent. (The figure of 62 per cent was, however, somewhat inflated by governmental use of tobacco coupons as pay in the early 1980s) (figure 71).

The pattern of tobacco consumption over the past two decades is reflected in the pattern of mortality from tobacco-attributable diseases. The decreases in smoking prevalence among men have now been followed by a slight decrease in lung cancer mortality in the 20-44 age group that began in the early 1980s (figure 76). Lung cancer rates in this age group should be the earliest indicators of changes in the population's exposure to the cancer-causing effects of tobacco (Peto, 1986). These findings in Poland are even more significant when compared with the continuing increase in mortality rates from lung cancer among men aged 20-44 in Hun-

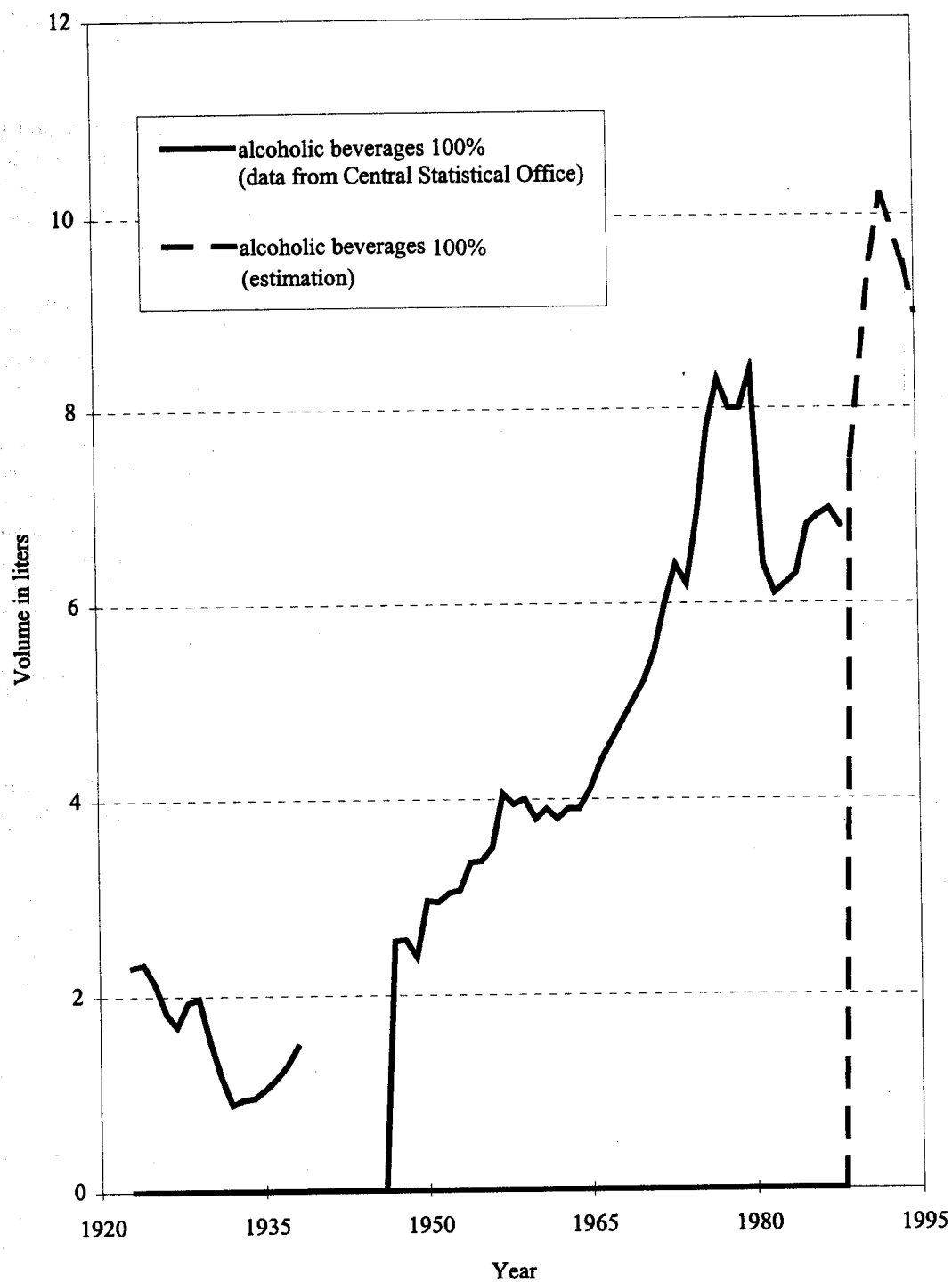
gary (figure 79), where the consumption of tobacco has not slowed (Kubik and others, 1995; Zatoński and others, 1996a). Hungary can therefore be used as a control for appraising changes in tobacco-attributable disease risk in the Polish population. Comparisons show a divergence of the trends for mortality from cancer among men in Hungary and Poland since about 1980. Mortality from lung cancer in Hungarian men aged 20-64 years has now reached a level never previously seen in any country (Zatoński, 1996a).

Negative trends in cigarette smoking are, however, being observed among Polish women. Although the percentage of women who have never smoked remains above 60 per cent, the prevalence rose substantially during the 1970s, from 18 per cent in 1974 to 30 per cent in 1982 (figure 71), and this increase was particularly marked in younger women. Since the early 1980s, the level of smoking among Polish women has stabilised rather than declined (figure 71). The previous increase in smoking is reflected in the currently increasing female mortality from lung cancer, the rates having doubled in the 20-44 (figure 76) and 45-64 age groups between 1974 and 1994 (Zatoński, 1996b).

The health consequences of smoking were also influenced by the introduction in 1991 of Governmental norms for permissible levels of tar (20 mg per cigarette) and nicotine (1.8 mg per cigarette) in cigarettes sold on the Polish market. These limits are still, however, much higher than those in the European Union, where the permissible tar content is no more than 12 mg per cigarette.

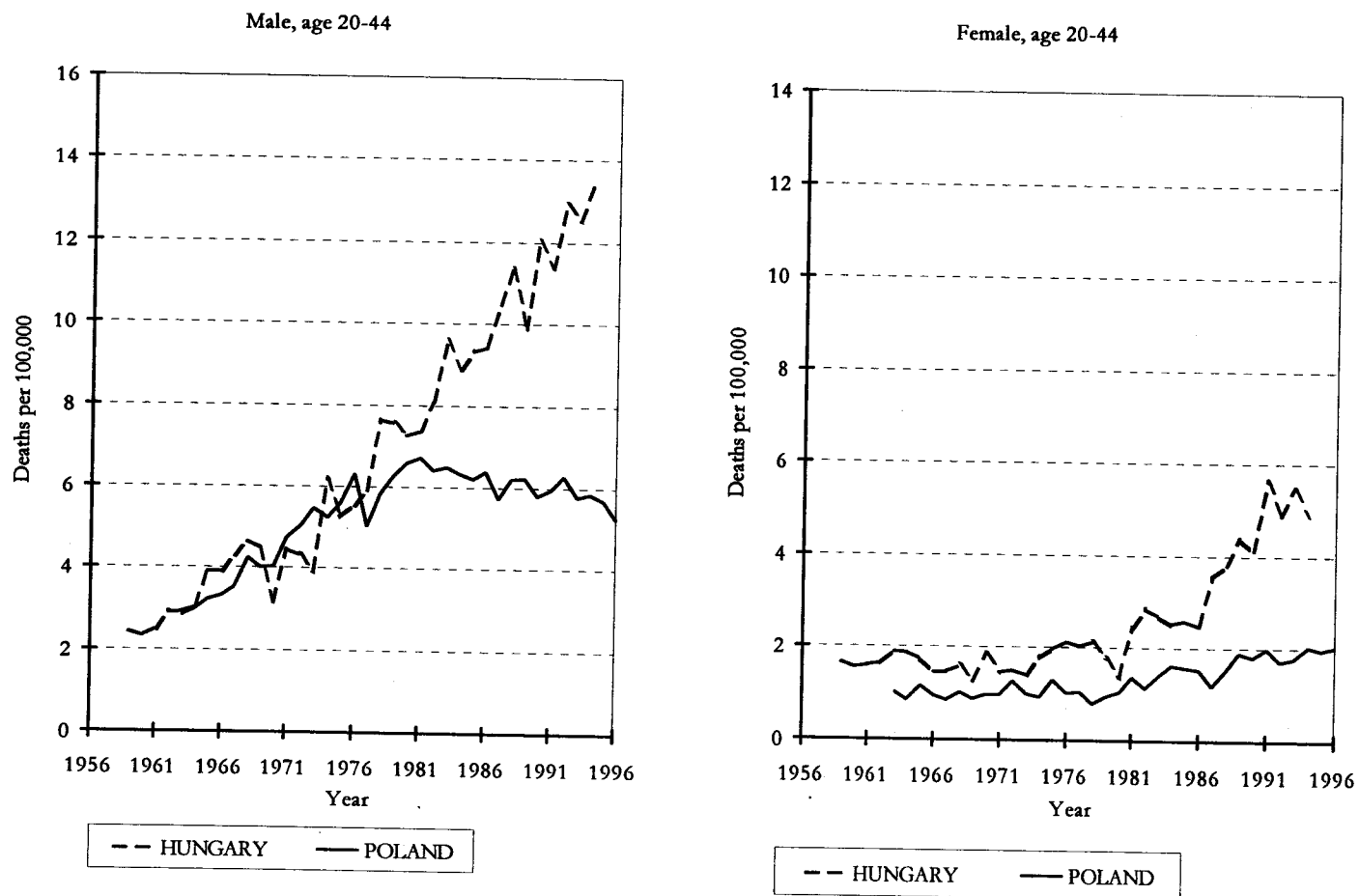
Most of the changes in smoking and the resulting changes in health began before 1989. Unfortunately, the economic reforms since 1989 have left tobacco products at a very low price, and a packet of the most commonly smoked cigarettes is still cheaper than a loaf of bread. Foreign cigarettes were introduced onto the market immediately after the borders were opened, mostly through smuggling, and are also relatively cheap. These cigarettes have attractive, colourful packaging and aromatized tobacco; they were also promoted as "healthier" than local brands. Contrary to popular opinion, however, the cigarettes produced by foreign companies do not necessarily have lower levels of carcinogenic sub-

**Figure 78. Consumption of alcoholic beverages in Poland, 1923-1995**  
(in liters of 100% alcohol per capita)



Source: Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years).

**Figure 79. Mortality rates from lung cancer, Poland and Hungary, 1959-1996**



Source: **Poland:** Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years);  
**Hungary:** World Health Organization, *WHO Mortality Data Bank* (Geneva, WHO, 1997).



stances—indeed, they sometimes deliver more tar and nicotine than those produced locally (Zatoński, 1996).

Since 1989, stylish cigarette advertisements have been introduced on Polish streets and in the press on a huge scale, creating a positive image for tobacco. Rapidly, most advertising agencies and newspaper publishers and also some politicians became strongly “dependent” on the tobacco industry, which has also started organising rock music concerts and other activities targeted at the young. There is also, however, an organised health lobby in Poland, mainly within the medical community, which has created a counterweight to these activities, and the first democratically appointed Senate (upper Chamber of Polish Parliament) drafted a law aimed at controlling smoking in Poland. After five years of well-financed opposition the act was eventually brought into force in May 1996, but it will be some years before it can have much effect on Polish death rates (Zatoński and others, 1996b).

Effective legislation against tobacco promotion, a total ban on advertising, appropriate pricing policies (with labelling of cigarettes that are to be sold in Poland in ways that discourage smuggling), and much clearer health warnings on cigarette packages (to cover 30 per cent of both of the two largest sides, from mid-June 1998) could lead to a significant decrease in the risk of death from tobacco-attributable diseases in the Polish population. This is especially true for young people and children, who are the subject of the tobacco industry's special attention and who more and more often become physiologically addicted to nicotine before they reach adulthood (Szymborski and Zatoński, 1996; Szymborski and others, 1996).

After the Second World War, dietary habits in Poland became progressively more and more “westernised”, and the calorie content of daily food and the consumption of animal fats rose steadily up to the late 1970s. Consumption of fruits and vegetables also rose during this period, although it was limited to local products and thus showed distinct seasonal variation, with substantial intake only in the autumn (Sekula and others, 1996). The changes in dietary patterns in Poland since the early 1960s are in contrast to those in the countries of Western Europe and North

America. For example, by the end of the 1970s, Poland was among the world's largest consumers of butter, whereas in many countries there was a decided decrease in consumption: e.g. in Finland, annual butter consumption fell from 19 kg per capita in 1960 to 5 kg in 1990 (WHO, 1995).

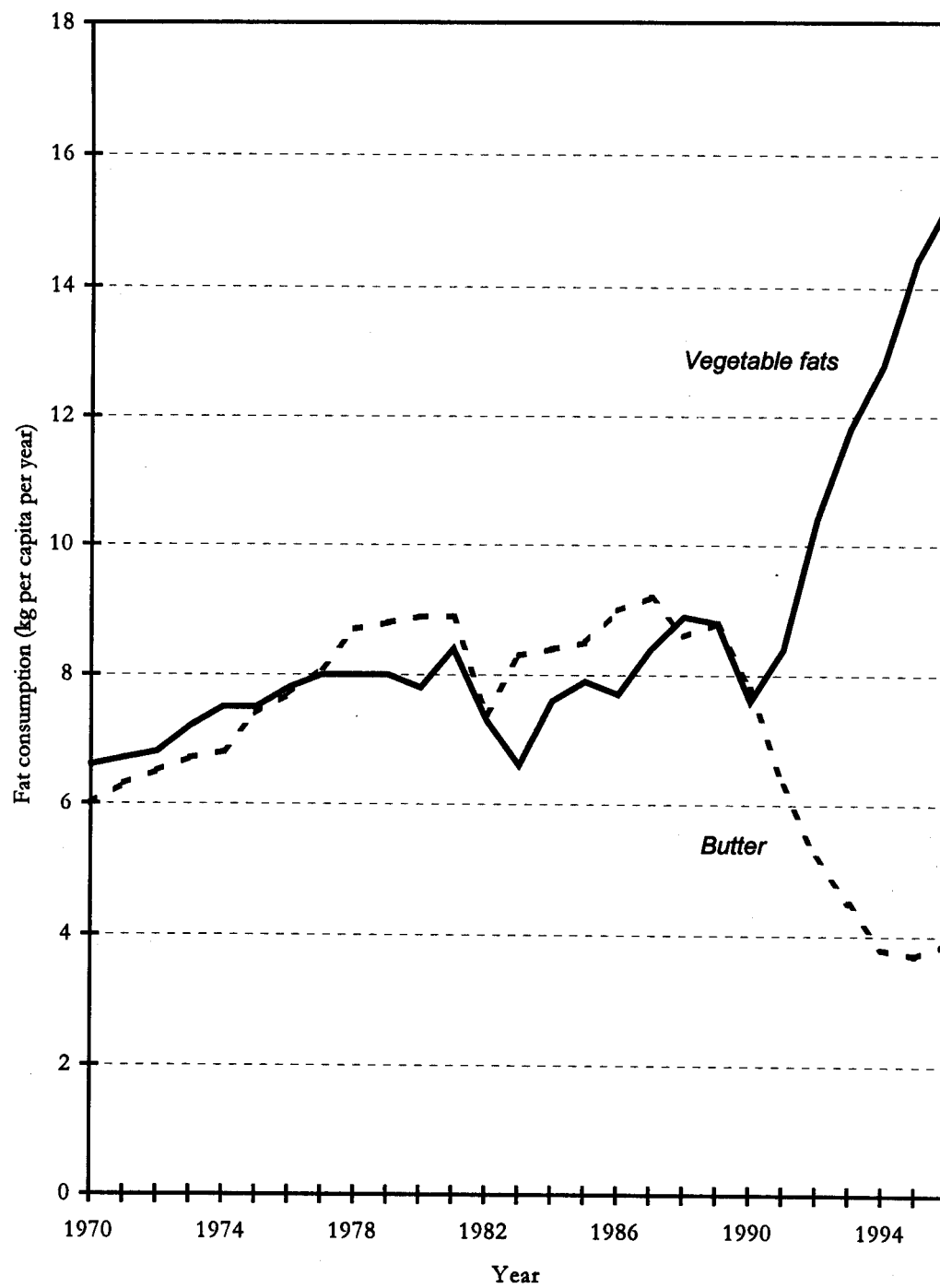
At the beginning of the 1980s, however, the food market in Poland broke down. The availability of food products in 1980-1982, especially meat, animal fats (including butter), eggs and sugar was very limited. Certain food products were rationed, and the average daily energy intake fell from 3,600 kcal in 1980 to 3300 kcal in 1982 (Sekula and others, 1992, 1996), after which it rose again to 3,500 calories.

In 1989-1990, the market suddenly changed again. Wages fell, government subsidies were discontinued, food products of animal origin were priced much higher, while food products of plant origin were priced relatively lower. Foreign products began to appear on the Polish food market and new techniques were introduced for local food production. The price revolution, the introduction of new products and the availability of a wide variety of foods all contributed to a sudden, profound change in dietary patterns. The average food energy intake dropped from 3,489 kcal in 1989 to 3,248 kcal in 1994. Daily animal fat consumption fell from 101 to 77 g (Central Statistical Office, 1995), due mainly to a sudden drop in butter consumption, from 8.8 kg in 1989 to 3.8 kg in 1994 (figure 80), and in meat and milk consumption (Sekula and others, 1996). Overall fat consumption remained essentially the same. Animal fats were generally replaced by vegetable fats, the consumption of which increased from about 8 kg in 1989 to almost 16 kg in 1996 per capita (figure 80), mainly because of the replacement of butter by cheaper, “easy-spread” foreign margarines, which were introduced by intensive advertising campaigns (Central Statistical Office, 1987, 1993, 1995).

Another change in consumption concerned “southern” fruits (which in Poland are oranges, grapefruits, tangerines, bananas, kiwis, etc.) (figure 77). The convertibility of Polish currency and the opening of borders meant that such foreign products were now available everywhere, all year round. This market ex-

**Figure 80. Consumption of fat in Poland, 1970-1996**

(per capita/year)



Source: Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks]  
(Warsaw, GUS, various years).

pansion also prompted the increased production and consumption of locally produced fresh fruit and vegetables, with some availability (in the form of juices) even out of season; however, Poland does still remain a country with wide seasonal fluctuations in fruit and vegetable consumption (figure 81; Sekula and others, 1996; Sekula, 1995).

The changes in Poland during the late 1980s and early 1990s reflect one of the largest natural experiments on diet in developed countries over the past few decades. These changes seem to be leading to sudden, unexpected, positive changes in the health status of the Polish population. How are the rapid changes in mortality best explained?

The decrease in premature mortality from cardiovascular diseases in Poland is of a magnitude that is apparently without precedent in peacetime. A similar, well-documented phenomenon was, however, observed during the Second World War in Norway. During the German occupation of that country, mortality from vascular causes fell by around 20 per cent within two to three years (Strom and Jensen, 1951). This was associated with the enforced changes in the national diet, notably a radical decrease in the consumption of meat and dairy products and considerable increases in fish and fresh vegetables (Strom, 1948).

During the 1960s and 1970s, mortality from vascular diseases, particularly among males, changed sharply in several western countries, including Australia, New Zealand, the United States, Finland and Belgium (Thom and others, 1992). In Australia, however, where the change was perhaps the most marked, vascular mortality among men aged 45-64 still fell by only about 4 per cent per year - well below the corresponding Polish figure for 1991-1996 (figure 74; Zatoński and others, 1998). These reductions in vascular mortality in western countries are attributed to a variety of determinants, not all of which are understood.

There is little evidence that, in Poland, improved access to medical services (Zatoński, 1996; Bojan and others, 1991) or increases in their effectiveness could account for much of the improvement in mortality in 1988-1994. Indeed, it appears that despite progress in cardiology in Poland, financial barriers continue to prevent access to new cardiological

technologies on a population scale, as the annual state expenditure on health services per capita is currently only approximately US\$150. Progress in treatment, its rationalisation, preventive care and hypertension treatment on a population scale are still challenges rather than successes of medicine in Poland.

Smoking is clearly an important determinant of vascular disease (Rywik and Broda, 1996); however, the acuteness of the change in mortality, its appearance in all age groups and the lack of a sex difference speak against a dominant role for reduced effects of tobacco (figure 71).

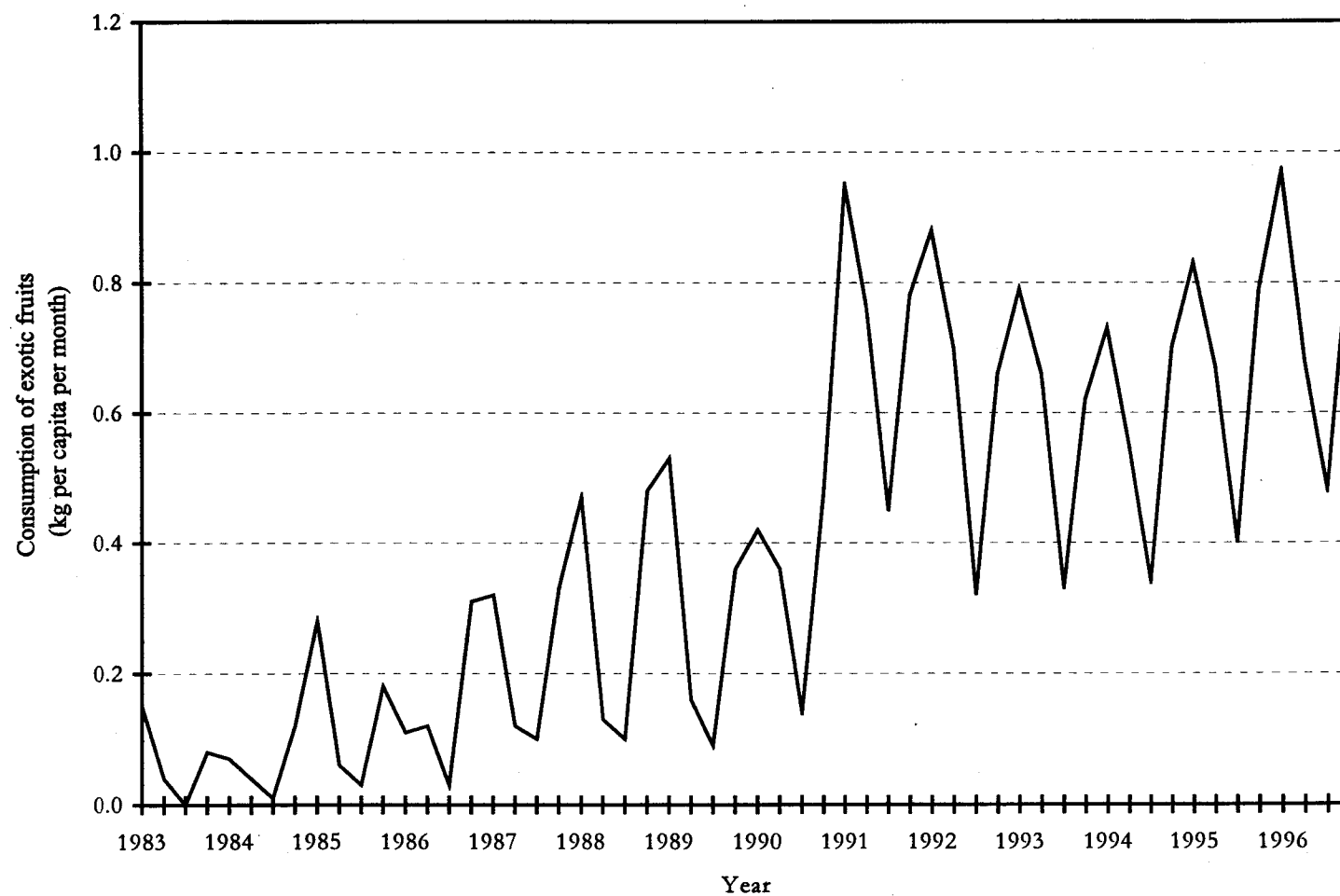
Although the political and economic transformation that began in Poland in 1989 resulted in marked changes in everyday life, it is certainly not evident that stress has generally been reduced. Unemployment, currently affecting about 13.5 per cent of the potential workforce, contrasts with the preceding era of state-assured job security, and suicide rates have not fallen (Central Statistical Office, 1995).

By exclusion, and taking into account biological and epidemiological plausibility, explanations for the changing epidemiological situation should thus be sought principally in the dietary changes that have occurred throughout Poland, although most markedly in urban districts (Sekula and others, 1996; Central Statistical Office, 1987, 1995; Zatoński, 1996).

Randomised controlled trials clearly show that lowering blood cholesterol levels by drugs can reduce the risk of death from coronary disease within two or three years. However there are no reliable studies on the whole Polish population concerning the blood cholesterol concentrations in years 1988-1995.

Consumption of fresh fruits and vegetables is generally associated with a reduced risk of mortality from vascular diseases (Ness and Powles, in press). A close temporal association has been also suggested between low consumption of vitamin C in winter and excess mortality from vascular diseases (Khaw and Woodhouse, 1995). In Poland, this increase of mortality during the first 3 months of the year has also been observed, but its rela-

Figure 81. Consumption of exotic fruits in Poland, 1983-1996



Source: Central Statistical Office, *Rocznik Statystyczny* [Statistical yearbooks] (Warsaw, GUS, various years).

tion to vitamin levels needs future investigation (Zatoński and others, 1996a).

The sudden, unexpected changes in Polish mortality trends since 1988 are only partially understood and need much further exploration.

## G. CONCLUSION

The political-economic transformation in Poland after 1988 led to substantial changes in factors that can influence the health of the population. Over the past few years, a great natural experiment has been occurring in Poland. The changes, although similar to those in some other countries of central Europe, were larger.

The changes in health status in 1988-1994 went through two phases. The increase in mortality from non-medical causes in 1988-1991, especially among young men, in seems to have been chiefly the result of a sudden increase in alcohol consumption and its consequences. More impressive and positive is the second phase, involving the large decline in mortality in 1991-1996, principally involving premature deaths from cardiovascular diseases. This process, although difficult to interpret definitively, is probably chiefly due to the changes in the dietary patterns of the Polish population that followed the introduction of a market economy. The important changes include decreasing consumption of animal (saturated) fat and increasing consumption of vegetable (unsaturated) fat, while maintaining a stable overall fat intake. Another important element may well be the increased consumption of fresh fruits and vegetables (with fewer seasonal fluctuations, particularly in winter and spring).

Many recent epidemiological studies indicate that a "Mediterranean" diet (Willett and others, 1995) is generally associated with low vascular death rates. This diet differs in many ways from the traditional, climate-dependent diet of Central Europe.

A good example of health changes resulting from dietary changes is found in Finland. In the early 1960s, that country had the highest animal fat consumption in Europe, low fruit and vegetable consumption and also the high-

est morbidity and mortality rates from cardiovascular disease among European countries (figure 68). Finnish males also had one of Europe's highest incidence rates of lung cancer, attributed to cigarette smoking (Zatoński and others, 1995). The Finnish government, with broad support in the society, developed a public health policy with regard to tobacco, alcohol and diet, that was rapidly followed by a decrease in premature mortality, mainly from cardiovascular diseases and cancer (figure 68). The experience of Finland might provide the basis for a successful health intervention strategy in Poland.

## REFERENCES

- Bobak, M., and M. Marmot (1996). East-West mortality divide and its potential explanations: proposed research agenda. *British Medical Journal*, vol. 312, pp. 421-425.
- Bojan, F., P. Hajdu and E. Belicza (1991). Avoidable mortality: is it an indicator of quality of medical care in Eastern European countries? *Quality Assurance Health Care*, vol. 3, pp. 191-203.
- Brzezinski, Z., and K. Szamotulska (1996). Stan zdrowia matek, dzieci i młodzieży w Polsce [The health state of mothers, children, and youth in Poland]. In *Stan zdrowia Polaków [The health status of the Poles]*, W. Zatoński, B. Hulanicka and J. Tyczynski, eds. Wrocław: Monografie Zakładu Antropologii Polskiej Akademii Nauk.
- Central Statistical Office (1987). *Budżety Gospodarstw Domowych w 1986 r.* [The 1986 household budget surveys]. Warsaw: GUS.
- \_\_\_\_\_. (1993). *Instrukcja dotycząca organizacji i sprawozdawczości z badań budżetów gospodarstw domowych metoda rotacji miesięcznej w latach 1994-1996* [Instructions concerning organization and reporting from household budget surveys using the monthly rotation method]. Warsaw: GUS.
- \_\_\_\_\_. (1945-1993). *Rocznik Demograficzny* [Demographic yearbooks]. Warsaw: GUS.
- \_\_\_\_\_. (various years). *Rocznik Statystyczny* [Statistical yearbooks]. Warsaw: GUS.
- \_\_\_\_\_. (1995). *Budżety Gospodarstw Domowych w 1994 r.* [The 1994 household budget surveys]. Warsaw: GUS.
- Chenet, L., M. McKee, N. Fulop, F. Bojan, H. Brand, A. Hort and P. Kalbarczyk (1996). Changing life expectancy in Central Europe: is there a single reason? *Journal of Public Health Medicine*, vol. 18, pp. 329-336.
- Doll, R., and R. Peto (1981). *The causes of cancer*. Oxford Medical Publications. Oxford: Oxford University Press.
- Doll, R., R. Peto, K. Bjartveit, L. Chazova, G. Chlomo-gorova, A. Hirsch, V. Levshin, D. Simpson and W. Zatoński (1993). Tobacco and death in Eastern Europe. In *Cancer prevention in Europe*, W. Bodmer and D. Zaridze, eds. London: Organization of European Cancer Institutes, pp. 71-97.
- Feachem, R., T. Kjellstrom, C. Murray, M. Over and M. Phillips, Eds. (1992). *The health of adults in the developing world*. A World Bank Book. Oxford: Oxford University Press.
- Feachem, R. (1994). Health decline in Eastern Europe. *Nature*, vol. 367, pp. 313-314.

- Habrat, B., and S. Puzynski (1996). *Szkody zdrowotne spowodowane piciem alkoholu w Polsce* [Health damage caused by drinking alcohol in Poland]. In *Stan zdrowia Polaków* [The health status of the Poles], W. Zatoński, B. Hulanicka and J. Tyczynski, Eds. Wrocław: Monografie Zakładu Antropologii Polskiej Akademii Nauk.
- Hertzmann, C. (1995). *Environment and health in Central and Eastern Europe*. Washington, D.C.: World Bank.
- Holzer, J. Z. (1970). *Demografia* [Demography], Warsaw: PWE.
- Indulski, J., and N. Szeszenia-Dabrowska (1996). *Srodowisko pracy a stan zdrowia* [Workplace environment and health state]. In *Stan zdrowia Polaków* [The health status of the Poles], W. Zatoński, B. Hulanicka and J. Tyczynski, eds. Wrocław: Monografie Zakładu Antropologii Polskiej Akademii Nauk.
- Jedrychowski, W., H. Becher, J. Wahrendorf, E. Flak and Z. Basa-Cierpalek (1989). *Roczny wpływ palenia tytoniu, czynników zawodowych i zanieczyszczenia powietrza atmosferycznego na ryzyko raka oskrzela, wyniki badań retrospektywnych* [The joint influence of smoking tobacco, worksite factors, and environmental air pollution on bronchial cancer, results of retrospective studies]. *Nowotwory*, vol. 39, pp. 65-75.
- Kauhanen, J., G. A. Kaplan, D. E. Goldberg and J. T. Salonen (1997). Beer drinking and mortality: results from the Kuopio ischaemic heart disease risk factor study, a prospective population based study. *British Medical Journal*, vol. 315, pp. 846-851.
- Khaw, K. T., and P. Woodhouse (1995). Interrelation of vitamin C, infection, haemostatic and cardiovascular disease. *British Medical Journal*, vol. 310, pp. 1559-1563.
- Kopczynski, J., and P. Gorynski (1996). Epidemiologia zgonów z przyczyn zewnętrznych w Polsce [Epidemiology of death from external causes in Poland]. In *Stan zdrowia Polaków* [The health status of the Poles], W. Zatoński, B. Hulanicka and J. Tyczynski, eds. Wrocław: Monografie Zakładu Antropologii Polskiej Akademii Nauk.
- Kubik, A. K., D. M. Parkin, I. Plesko, W. Zatoński, E. Kramarowa, M. Möhner, H. P. Friedl, L. Juhasz, CH. G. Tzvetansky and J. Reissigova (1995). Patterns of cigarette sales and lung cancer mortality in some Central and Eastern European countries, 1960-1989. *Cancer*, vol. 75, pp. 2452-2460.
- Leowski, J., and M. Miller (1993). Ocena sytuacji epidemiologicznej gruźlicy w Polsce i w świecie [An evaluation of the epidemiological situation of tuberculosis in Poland and the world]. *Przegl. Epidemiol.*, vol. 3, pp. 197-207.
- Magdzik, W. (1996). Epidemiologia chorób zakaźnych w Polsce [Epidemiology of infectious diseases in Poland]. In *Stan zdrowia Polaków* [The health status of the Poles], W. Zatoński, B. Hulanicka and J. Tyczynski, eds. Wrocław: Monografie Zakładu Antropologii Polskiej Akademii Nauk.
- \_\_\_\_\_, and M. Czarkowski (1994). Choroby zakaźne w Polsce w 1992 roku [Infectious diseases in Poland in 1992]. *Przegl. Epidemiol.*, vol. 47, pp. 45-48.
- McMichael, A. J. (1996). Environmental epidemiology: issues, difficulties, challenges and opportunities. In *Stan zdrowia Polaków* [The health status of the Poles], W. Zatoński, B. Hulanicka and J. Tyczynski, Eds. Wrocław: Monografie Zakładu Antropologii Polskiej Akademii Nauk.
- \_\_\_\_\_, and W. Zatoński (1996). Environmental, behavioural, and socio-economic influences: tackling the historical jigsaw puzzle of health in Central and Eastern Europe. *International Journal of Occupational and Environmental Health*, vol. 2, pp. 161-163.
- Mlekoć, S., Z. Piasecki and T. Olakowski (1994). Urazy i zatrucia [Traumas and poisoning]. In *Promocja zdrowia*, J. Karski and others, eds. Sanmedia, pp. 79-105.
- Murray, C., and A. Lopez (1994). Global and regional cause-of-death patterns in 1990. *Bulletin of the World Health Organization*, vol. 72/3, pp. 447-480.
- Murray, C. J., G. Yang and X. Qiao (1993). Adult mortality: levels, patterns and causes. In *World Development Report: Investing in Health*. World development indicators. A World Bank Publication. Oxford: Oxford University Press, pp. 23-111.
- Ness, A.R., and J. W. Powles (in press). Fruit and vegetables and cardiovascular disease: a review. *International Journal of Epidemiology*.
- Okolski, M. (1994). Migracje zagraniczne w Polsce w latach 1980-1989 [Foreign migrations in Poland in 1980-1989]. *Zarys problematyki badawczej. Stud. Demograf.*, vol. 3/117, pp. 3-59.
- Peto, R., A. Lopez, J. Boreham, M. Thun and C. Heath, Jr. (1992). Mortality from tobacco in developed countries: indirect estimates from national vital statistics. *Lancet*, vol. 339, pp. 1268-1278.
- \_\_\_\_\_, (1994). *Mortality from smoking in developed countries 1950-2000*. Oxford: Oxford University Press.
- Peto, R. (1986). *Influence of dose and duration of smoking on lung cancer rates*. In: Peto R., Zaridze D. G. Tobacco: a major international health hazard. Lyon: IARC Scientific Publications; 74: 23-33.
- Przewozniak, K., and W. Zatoński (1996). Trendy, geografia i społeczno-behavioralna charakterystyka palenia tytoniu w Polsce [Trends, geography, and socio-behavioural characteristics of tobacco smoking in Poland]. In *Stan zdrowia Polaków* [The health status of the Poles], W. Zatoński, B. Hulanicka and J. Tyczynski, eds. Wrocław: Monografie Zakładu Antropologii Polskiej Akademii Nauk.
- Rywik, S., and G. Broda (1996). Epidemiologia chorób układu krążenia - program Pol-MONICA w Warszawie [Epidemiology of cardiovascular diseases - the Pol-MONICA program in Warsaw]. In *Stan zdrowia Polaków* [The health status of the Poles], W. Zatoński, B. Hulanicka and J. Tyczynski, eds. Wrocław: Monografie Zakładu Antropologii Polskiej Akademii Nauk.
- Sekula, W. (1995). *Spożycie ilościowe artykułów żywnościowych*. [Quantitative consumption of food products]. In: Warunki życia ludności w 1994 r. Studia i analizy statystyczne, Warsaw: GUS.
- \_\_\_\_\_, Z. Niedzialek, K. Figurska, M. Morawska and T. Boruc (1992). *Spożycie żywności w Polsce w latach 1950-1991 w przeliczeniu na energię i składniki odżywcze* [Food consumption in Poland converted into energy and nutrients, 1950-1991]. Warsaw: Instytut Żywności i Żywnienia.
- \_\_\_\_\_, (1996). Food consumption in Poland converted into energy and nutrients, 1950-1995. Warsaw: Instytut Żywności i Żywnienia.
- Sierosławski, J. (1992). *Spożycie alkoholu i polityka wobec alkoholu w ocenie społecznej - raport z badań ankietowych* [Alcohol consumption and policy in social view - a report from population surveys]. Warsaw: Instytut Psychiatrii i Neurologii.
- \_\_\_\_\_, (1995). *Spoleczno-demograficzne korelaty picia alkoholu w Polsce*. [Socio-demographic correlates of drinking alcohol in Poland]. *Alkoholizm i Narkomania* (Warsaw: Instytut Psychiatrii i Neurologii), vol. 3, pp. 57-77.
- Strom, A. (1948). Examination into the diet of Norwegian families during the war-years 1942-45. *Acta Medica Scand.*, vol. 214, p. 22.
- Strom, A., and R. A. Jensen (1951). Mortality from circulatory diseases in Norway 1940-45. *Lancet*, pp. 126-129.
- Szymborski, J., and W. Zatoński (1996). *Szkolne programy zapobiegania paleniu tytoniu* [School programs for preventing tobacco smoking]. Warsaw: Państwowy Zakład Higieny.

- \_\_\_\_\_, Z. Juczynski and N. Oginska-Bulik (1996). *Program zapobiegania paleniu tytoniu dla uczniow starszych klas szkół podstawowych* [Smoking prevention program for older elementary school children]. Warsaw: Instytut Matki i Dziecka.
- Thom, T. J., F. H. Epstein, J. J. Feldman, P. E. Leaverton and M. Wolz (1992). *Total mortality and mortality from heart disease, cancer and stroke from 1950 to 1987 in 27 countries; highlights of trends and their interrelationships among causes of death*. NIH Publication No. 92-3088. Bethesda: National Institutes of Health.
- Troszynski, M., A. Raczynski, J. Leibschang, E. Filipp, E. Swiatek and B. Chazan (1996). Zgony matek w Polsce w okresie powojennym [Maternal deaths in Poland in the post-war period]. In: Slomko Z., Breborowicz G.H., Gadziowski J. (Eds.) *Poznan: Kliniczna Perinatologia i Ginekologia*. Polsk. Tow. Med. Perinatalnej. 15: 85-94
- World Bank (1996). *The Transition in Central and Eastern Europe*. Technical Paper No. 341, Washington: The World Bank.
- World Health Organization (1988). WHO MONICA Project. Geographical variation in the major risk factors of coronary heart disease in men and women, aged 35-64 years. *World Health Quarterly*, vol. 41: pp. 130-131
- \_\_\_\_\_. (1995). *Food and health indicators 1961-1990*. Copenhagen: WHO Regional Office for Europe, Nutrition Unit.
- \_\_\_\_\_. (1997). *WHO Mortality Data Bank*. Geneva: World Health Organization.
- Willett W. C., Sacks F., Trichopoulou A., Drescher G., Ferro-Luzzi A., Helsing E., Trichopoulos D. (1995). Mediterranean diet pyramid: A cultural model for healthy eating. *American Journal of Clinical Nutrition*, vol. 6, pp. 1402-1406.
- Wojtyniak B., Chanska M., Seroka W., Szata W., Chojecka E. (1996). *Sytuacja zdrowotna ludnosci Polski w 1994 roku* [The health situation of Poles in 1994]. Warsaw: PZH Zaklad Statystyki Medycznej.
- Zatoński, W. A. (1995). The health of the Polish population. *Public Health Review (Israel)*, vol. 23, pp. 139-156.
- \_\_\_\_\_. (1996). *Evolution of health in Poland since 1988*. Warsaw: Cancer Centre.
- \_\_\_\_\_, ed. (1996). *Stan zdrowia mieszkancow Warszawy* [The health status of Warsaw inhabitants]. Warsaw: Health Promotion Foundation Expertise.
- Zatoński, W. A., and P. Boyle (1996). Commentary: Health transformations in Poland after 1988. *Journal of Epidemiology and Biostatistics*, vol. 1, No. 4, pp. 183-197.
- Zatoński, W., and K. Przewozniak (1996). *Tobacco smoking in Poland: attitudes, health effects and prevention*. [The health effects of smoking in Poland]. Warsaw: Centrum Onkologii.
- Zatoński, W., N. Becker, K. Gottesman, A. Mykowiecka and J. Tyczynski (1988). *Atlas of Cancer Mortality in Poland 1975-1979*. Berlin: Springer-Verlag.
- Zatoński, W., J. Didkowska, J. Tyczynski, E. Pukkala and N. Gustavsson (1993). *Nowotwory zlosliwe w Polsce* [Cancer in Poland]. Warsaw: Centrum Onkologii.
- Zatoński, W., M. Smans, J. Tyczynski, P. Boyle, eds. (1995). *Atlas of Cancer Mortality in Central Europe*. International Agency for Research on Cancer, Lyon: IARC Scientific Publications No. 134.
- Zatoński, W., J. Didkowska, K. Przewozniak, W. Tarkowski, J. Tyczynski and U. Wojciechowska (1996a). *Stan zdrowia Polakow* [The health status of the Poles]. Expert appraisal, Warsaw Centrum: Onkologii.
- Zatoński, W., Z. Brzezinski, J. Didkowska, J. Engel, E. Florek, J. M. Jaworski, J. Lissowska, Z. Mielecka-Kubien, T. Parchimowicz, M. Przetakiewicz-Koziej, K. Przewozniak, H. Szczecinska, J. Szymborski, W. Tarkowski, J. Tyczynski and U. Wojciechowska (1996b). Ekspozycja populacji polskiej na tyton: palenie tytoniu, substancje toksyczne, nastepstwa zdrowotne [Exposure of the Polish population to tobacco: tobacco smoking, toxic substances, health effects]. *Magazyn Med.*, vol. 7, pp. 1-12.
- Zatoński, W., and J. Tyczynski, eds. (1998). *Cancer in Poland in 1995*. Warsaw: Centrum Onkologii.
- Zatoński, W. A., A. B. Lowenfels, P. Boyle, P. Maisonneuve, H. B. Bueno de Mesquita, P. Ghadirian, M. Jain, K. Przewozniak, Baghurst P., Moerman C. J., Simard A., Howe G.R., McMichael A.J., Hsieh C.C., Walker A.M. (1997). Epidemiologic aspects of gallbladder cancer: a case-control study of the SEARCH program of the International Agency for Research on Cancer. *Journal of the National Cancer Institute*, vol. 89, No. 15, pp. 1132-1138.
- Zatoński, W. A., McMichael A. J., Powles J. W. (1998). Ecological study of reasons for sharp decline in mortality from ischaemic heart disease in Poland since 1991. *British Medical Journal*, vol. 316, pp. 1047-1051.

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## XI. THE DYNAMICS OF MORTALITY IN THE RUSSIAN FEDERATION

*Evgueni Andreev\**

### A. INTRODUCTION

During the Soviet period of Russia's history there were just two comparatively short intervals when life expectancy increased—from 1922 to 1928 and from 1948 to 1965.

The post-World War II mortality decline did not start immediately after the end of hostilities, it was delayed by the 1947 famine (Andreev, Darsky, Khar'kova, 1997). Then, from 1948, life expectancy rose steadily until the mid-1960s (figure 82). At that time, mortality began to show negative tendencies: there was an increase in mortality due to diseases of the circulatory system at younger ages and in mortality from accidents, poisonings and violence. There was also a stabilisation at a relatively high level of infant mortality caused by infectious and respiratory diseases. A decline of expectation of life at birth for Russia's population was experienced throughout the period 1965-1994, except for a short interruption during the anti-alcohol campaign (1985-1987). In 1995-1996 a new and significant increase of life expectancy occurred comparable to the increase that took place during the anti-alcohol campaign.

At the beginning of the 20th century, the difference in life expectancy between Russia and 22 developed countries<sup>1</sup> was about 17 years less for males and 15 years less for females (table 47). By 1930 the gap had increased to 20 and 18 years respectively. In the mid-1960s the difference was smaller than ever before or after. At present, it is about 16 years for male and 8 for females.

Between 1992 and 1993, the annual number of deaths in Russia increased by 18 per cent, and expectation of life at birth decreased by 3.1 years for males and 1.9 years for females. Such changes during peacetime are quite unprece-

ented. The sharp decline in the expectation of life at birth aroused deep concern on the part of many demographers (Ellman, 1994; Andreev, 1995b; Mesle and others, 1994; Shkolnikov and others, 1995). The more recent mortality decline makes it possible to reappraise the situation.

The purposes of this paper are to:

- describe mortality trends in post-war Russia, with particular attention to the latest period;
- analyse the probable factors that explain the gap in mortality between Russia and the West;
- assess the prospects for further mortality decline in Russia.

A widely used conceptual framework for the study of mortality history is the so-called epidemiological transition model (Andreev, 1990). Four major phases in the transition are usually identified.

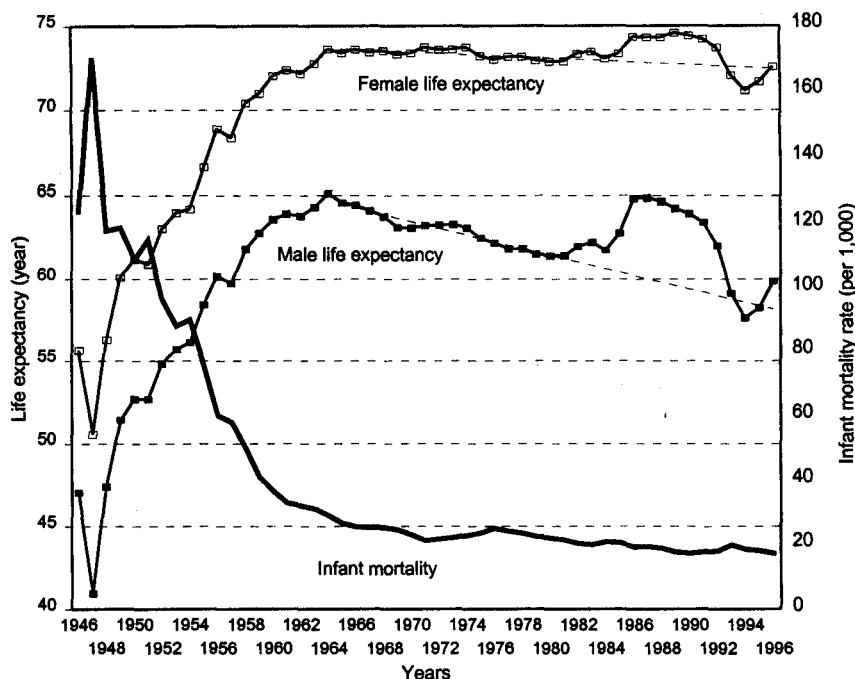
The first phase was characterised by extraordinary periodic mortality peaks due to famine and to pandemics (for example, of plague, cholera and small pox). During the second phase, there was an overall reduction of mortality. The decline was brought about by means of increased control over major infectious diseases such as childhood and gastro-intestinal infections and tuberculosis. The control came as a result of industrialisation and economic growth. However, the industrial revolution also led to environmental pollution and increased stress. During this second phase, mortality from cardiovascular diseases and neoplasms at younger ages also increased.

In the third phase, the negative effects of industrialisation were overcome through measures to improve the human environment and living conditions, the workplace, and health education. As a result, deaths from most diseases shifted toward the older age. In countries with the most

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Figure 82. Dynamics of expectation of life and infant mortality rate in Russia, 1946-1996



Source: Author's calculations based on national statistics.

TABLE 47. DYNAMICS OF EXPECTATION OF LIFE AT BIRTH IN RUSSIA AND IN 22 DEVELOPED COUNTRIES<sup>a</sup>, 1900 - 1995

Years	Russia		22 countries		Differences	
	Males	Females	Males	Females	Males	Females
1900	29.4	31.4	46.0	46.4	16.6	15.0
1930	33.7	38.2	53.8	57.0	20.1	18.8
1940 <sup>b</sup>	35.7	41.9	57.9	61.6	22.2	19.7
1950	52.7	61.2	64.6	68.0	11.9	6.7
1960	63.6	72.0	67.6	72.7	4.1	0.7
1970	63.1	73.4	68.9	74.9	5.8	1.6
1980	61.4	72.9	71.0	77.5	9.6	4.7
1985	62.7	73.2	71.8	79.0	9.1	5.7
1990	63.9	74.4	73.2	79.5	9.2	5.1
1995	58.3	71.7	74.1	80.1	15.8	8.4

Source: L. A. Dublin, Lotka and M. Spiegelman, *Length of life* (New York, Ronald Press, 1949; N. Keyfitz, and W. Flieger, *World population. Analysis of vital data* (Chicago, University of Chicago Press, 1968); United Nations, *Demographic Yearbook* (New York, United Nations, 1949-1994); Council of Europe, *Recent demographic developments in Europe* (Strasbourg, Council of Europe, 1992-1995); S. A. Novosel'sky, and V. V. Paevsky, *Mortality and expectation of life in the USSR, 1926-1927* (Life tables) (Moscow-Leningrad, 1930, in Russian); E. M. Andreev, L. E. Darsky and T. L. Kharkova, *Demographic history of Russia: 1927-1959* (Moscow, in press, 1997, in Russian); Goskomstat of Russia, *Demographic yearbook of Russia. Statistical handbook* (Moscow, 1997).

<sup>a</sup>Countries: Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Italy, Japan, Netherlands, New Zealand, Norway, Portugal, Spain, Sweden, Switzerland, United Kingdom (England and Wales, Scotland, Northern Ireland), United States of America.

<sup>b</sup>Last before World War II year.

fully developed and highly effective health care system, a fourth phase has emerged. There are further reductions in the infant and child mortality caused by congenital diseases, more

prematurely born children are saved, and there is a reduction of the level of mortality among aged persons. Expectation of life approaches its maximum.

In this paper we present results from our project "Demographic prospects of Russia" (INTAS-93-1732).

## B. SOURCES OF INFORMATION AND METHODOLOGICAL ISSUES

### 1. *Vital statistics*

Russia's modern vital statistics system was established at 1933. It is based on the registration of vital events by the local civil authorities ZAGS (Zapis' aktov grazhdanskogo sostoiianiia [Registry of Acts of Civil Status]). The system is governed by the Act of Civil Status (record of civil registration of birth, death, marriage or divorce), issued by the Ministry of Justice.

During the post-war period the content of Acts of Civil Status was revised several times. The modern form of the Acts of Civil Status was adopted by Russia's government on October 11, 1992. The civil death registration includes the following social-demographic items:

- date and place of birth;
- sex;
- ethnicity;
- occupation;
- marital status;
- education;
- place of permanent residence and length of time lived there;
- main cause of death (from medical death certificate).

In case of the death of an infant less than one year of age, the major socio-demographic characteristics of mother are also recorded.

Upon a death, a certificate is issued by the doctor or medical assistant to the relatives of the deceased or to some other person close to the deceased. That relative or other person registers the death at ZAGS. ZAGS in turn completes two copies of the "Record of civil registration of death" and sends the second copy and the medical death certificate to a regional statistical office for tabulation. Standard tabulations for each region of Russia include deaths by sex, age and cause of death. The program of tabulation is more extended in census years.

Between 1959 and 1964 Russia's vital statistics used the list of causes of deaths based on the 7th revision of the International Classification of Diseases, Injuries and Causes of Death (ICD). During this period data on causes of death were tabulated for irregular age groups. In 1965, Russia's vital statistics were switched to the 8th ICD revision, which was used until 1982. After 1982 mortality by cause was officially classified using the 9th ICD revision. In Russia, the shift from the 8th to the 9th revision was something of a formality, and the list of causes of death that is used today in Russia mostly remains close to the "A" list of ICD 8.

In 1996, age was absent from only 0.5 per cent of death certificates. Cause of death was classified as "senility without mention of psychosis" or "symptoms and other unspecified conditions" on just 4.4 per cent of medical death certificates. On the other hand, the quality of diagnosis in Russia is not always high. In 1996 in Russia, autopsies were performed on 33.2 per cent of all deaths, and of persons who died from unknown causes at ages over 1 year of age on less than 22 per cent.

We believe that the problem of *unregistered* deaths is not serious for Russia as whole. However, relatively high levels of under-registration of deaths were observed in the North Caucasus Republic and in the Republic of Tuva. No vital statistics are available for the Ingush Republic for 1993-1994 and for the Chechen Republic from 1993 onwards.

One must also keep in mind that in Russia before 1993, the definition of a live birth (adopted well before World War II) was operational. A live birth was defined as the complete expulsion of the foetus from the mother's womb, with a period of gestation of 28 weeks or more, the infant at least 35 cm in length and 1,000 grams in weight, and who after expulsion had taken at least one breath. If the infant was born after less than 28 weeks of gestation, it was considered a live birth only if it survived for over 168 hours.

Starting from 1993, Russia formally announced the adoption of the WHO definition of a live birth and a stillbirth. However, as noted with respect to the newly issued instructions on

death registration, the shift to the WHO definition was adopted only for departmental statistics. A delivery is registered as a birth by ZAGS only if the newborn child weighs 1,000 grams or more (or, when weight at birth is unknown, if the infant is 35 or more cm head to toe) and with a period of gestation of 28 weeks and over. Newborn children who weigh between 500 to 999 grams are considered live births only if they survive more than 168 hours.

However, in the case of multiple deliveries, the infant may be classified as a live birth even if it weighs less than 1,000 grams. Because official state statistics are based on ZAGS data, the real change consists in broadening the signs of life, separating live-born who died during the first week of life from still-births, and including in the number of live-births those new-born in multiple deliveries who weigh less than 1,000 grams (Goskomstat, 1995, p. 488).

From a statistical point of view, for registration based on the Acts of Civil Status the 1993 changes in the definition of a live birth and a stillbirth only increased the number of criteria that separate live born children from the still-born. Before 1993, a live birth required at least one breath; now there are the various criteria of the WHO definition. In addition, the decree abandoned the criteria of weight plus length. It is estimated (Andreev, 1995a) that these changes increase the infant mortality rate by not more than 2 deaths per thousand live births. However, according to Russian and Western researchers, a complete shift to the WHO definition of live births would have increased the infant mortality rate by some 22 to 25 per cent (Anderson and Silver, 1990).

A detailed description of sources of information on the population of Russia can be found in Andreev, Scherbov and Willekens (1995).

## 2. *Population estimates*

For the calculation of mortality rates, one normally uses estimates of population size by age and sex based on censuses or surveys. In the post-war period there were four population censuses in Russia, in 1959, 1970, 1979 and 1989.

Current estimates of population size by age and sex are based on the most recent census

returns, adjusted on the basis of vital rates and migration estimates. The standard balancing procedure is used: the population at the end of a year is equal to population at the beginning of the year plus births, minus deaths, plus immigrants, minus out-migrants during the year. Migration statistics are adjusted before they are used in the estimation of population. Preliminary estimates of the number of births and deaths were used to produce total population estimates; these may differ from the final estimates used for analysis of birth and death processes.

Official estimates of population size of Russia as a whole by age and sex are available for the period after 1970. Comparable data for the regions of the Russian Federation are available for the period after the 1979 censuses. In this paper we use our previously published population estimates for the period 1946-1958 (Andreev, Darsky, Khar'kova, 1997), and the results of our calculation for the period 1960-1969. One should keep in mind that the State Committee of the USSR on Statistics verified the current estimates of population size for the periods 1971-1978 and 1980-1988 after the 1979 and 1989 censuses, and has revised the current estimates. Thus, there are two version of Russia's population size by age and sex for these periods and two versions of age specific mortality rates.

Most of the mortality rates that are used in this paper are the result of our calculations, based on the official vital statistics. For the period 1965-1994, we also used the annual series of age specific death rates by sex based on the 1981 Soviet causes of death classification, as calculated by Mille, Shkolnikov and Vallin (1996).

## C. TRENDS IN AGE-SPECIFIC MORTALITY

Trends in age-specific mortality in the immediate post-war period may be considered as generally typical of the second phase of the epidemiological transition, at least as it is described by the model life tables (Coale and Demeny, 1966). The relative decline of female mortality in 1946-1950 was almost independent of age; only later was there linear dependency between mortality decline and age, with the

maximum decline in child ages (table 48). Mortality declines in childhood were almost equal for males and females. And the mortality of males in the age group 40-50 declined between 1946 and 1950 even more than in younger and older ages. Possibly this was due to the fact that by 1950, the higher mortality of disabled veterans no longer had any perceptible influence on male mortality levels. However, from 1950 to 1960 the decline of mortality in the middle ages progressed more slowly for males than for females.

The considerable decline of infant mortality during 1944 and 1945—the final 16 months of World War II and the first 8 months of peace—was unexpected. The mortality decline in this period was a result mainly on the introduction and widespread use of sulfa drugs (Sifman, 1979).

Unfortunately, cause of death data for the whole population of Russia is available only for the years after 1959. And the analysis of indices for the urban population over the period 1946-1958 is complicated by the lack of reliable age distributions. However, even the analysis of the absolute numbers of deaths shows a decline of adult mortality from tuberculosis. The adult mortality decline during this period probably also resulted from the use of new drugs to cure of infectious diseases, particularly tuberculosis and other diseases of the respiratory system.

After 1960 the decline of mortality for all females and for male children slowed down, and mortality levels of adult males were practically stationary up to 1965. The increase of mortality in the adult ages began in the middle of the 1960s. From 1964 till 1981, the probability of death for males in the age group 20-59 grew practically linearly over time (figure 83). The coefficient of determination ( $r^2$ ) for mortality by year was equal to 0.973. The minimum value was 0.705 for the age group 25-29. The increase in adult female mortality was not so dramatic. Mortality levels at age groups 15-34 were stable or slowly declining up to mid-1980s.

The decline in infant mortality only continued up until 1971. There were many controversies in connection with the increase in infant mortal-

ity in 1971-1976. Just at the beginning of that period a new medical certificate for perinatal death was introduced. That evidently increased the completeness of the registration of deaths during the first week of life, and thus also the reported rate of infant mortality. However, it is not yet clear to what degree the trend in infant mortality of 1971-1976 was a statistical artefact as opposed to a real process.

In the period of the anti-alcohol campaign (1985-1987) a significant drop in adult mortality was observed. The resumed increase of age specific mortality rates in 1989-1994 was not symmetric with the decline of 1985-1987, but has moved to the older ages. But in the real cohorts, the mortality decline of 1985-1989 was comparable to its rise in 1989-1994 (Avdeev and others, 1997).

None of these trends touched infants and children. For infants and children, the period 1980-1990 was one of slowly declining mortality. A small increase in 1990-1994 was replaced a similar decrease in 1995-1996.

It is useful to analyse trends in expectation of life at birth in terms of the impact of mortality changes in the various age groups. We use Korchak-Chepurkovskiy's method (1987) for such an analysis (table 49). Broadly speaking, we observe that from 1946 until 1965 life expectancy of males increased by 11.5 years due to mortality declines at ages 0-14 and by 6.0 years to declines at ages 15 and over. The corresponding indicators for females were 12.1 and 5.6 years. The mortality trends for ages 0-14 from 1965 to 1996 increased life expectancy of males by 0.8 years and of females by 0.9 years. The increase of mortality at ages 15 and over was the reason why life expectancy decreased by 6.0 years for males and 1.8 years and females. Thus, for males the gains between 1946 and 1965 were offset by the losses between 1965 and 1996. Over the whole post World War II period, changes in adult mortality contributed nothing to improved expectation of life at birth for males.

The other commonly used approach to study age specific mortality trends is age-period-cohort (APC) analysis. Variations in mortality are attributed to two sets of factors: contemporary and historical. Contemporary factors are

TABLE 48. AGE-SEX SPECIFIC DEATH RATES PER 1,000, RUSSIA, 1946-1996

Years	<i>Males</i>							
	<i>Age group</i>							
	0	1-4	5-14	15-24	25-44	45-64	65-84	85 and over
1946	146.2	21.6	3.6	4.3	8.9	26.8	81.6	184.6
1947	204.0	35.0	4.0	5.0	11.2	36.9	112.0	249.5
1948	139.3	31.9	3.2	4.5	8.7	25.6	76.5	169.6
1949	141.9	17.5	2.6	3.5	7.1	22.1	68.3	157.8
1950	129.3	16.0	2.3	3.2	6.5	21.8	68.9	159.1
1951	135.8	16.3	2.3	3.2	6.1	21.2	68.3	158.5
1952	113.5	11.7	2.0	2.9	5.8	20.9	70.7	179.4
1953	102.8	10.1	1.9	2.9	5.8	20.6	72.2	179.4
1954	104.8	10.1	1.8	2.7	5.4	19.8	71.8	182.7
1955	87.8	8.0	1.6	2.4	5.0	18.6	66.5	182.0
1956	68.9	6.4	1.4	2.3	4.8	18.1	65.2	178.5
1957	66.5	5.5	1.4	2.4	5.2	19.3	69.9	191.5
1958	57.6	4.2	1.2	2.2	4.7	17.4	63.4	180.5
1959	46.7	3.2	1.1	2.1	4.5	17.3	63.9	192.6
1960	41.6	2.7	1.0	2.2	4.5	16.7	60.7	174.7
1961	37.3	2.4	1.0	2.1	4.5	16.8	61.5	163.4
1962	36.2	2.3	0.9	2.0	4.5	17.2	64.8	177.9
1963	35.1	2.0	0.9	2.0	4.5	16.8	62.0	153.6
1964	32.1	1.7	0.8	1.9	4.4	16.0	59.4	137.8
1965	30.1	1.7	0.8	2.0	4.7	16.5	63.8	148.2
1966	29.2	1.6	0.8	1.9	4.9	16.6	64.1	149.1
1967	28.6	1.6	0.8	1.9	5.1	17.1	66.0	158.3
1968	29.1	1.6	0.8	2.0	5.3	17.5	66.2	166.0
1969	28.8	1.5	0.8	2.1	5.7	18.3	69.7	205.9
1970	26.4	1.6	0.8	2.1	5.8	18.4	70.2	233.8
1971	23.9	1.5	0.8	2.2	5.8	18.4	69.8	226.4
1972	24.1	1.6	0.8	2.2	5.7	18.2	70.7	229.7
1973	24.4	1.6	0.7	2.2	5.5	18.4	71.1	229.4
1974	25.5	1.6	0.7	2.3	5.8	18.5	69.3	211.9
1975	26.4	1.6	0.7	2.4	6.0	19.4	72.2	228.2
1976	28.0	1.6	0.7	2.4	6.1	20.0	71.9	225.1
1977	27.6	1.6	0.8	2.5	6.4	20.6	72.0	225.5
1978	27.3	1.6	0.7	2.4	6.4	21.0	71.5	223.2
1979	26.2	1.6	0.8	2.5	6.6	21.5	73.5	240.2
1980	25.6	1.7	0.7	2.5	6.7	21.8	73.6	247.2
1981	25.4	1.7	0.8	2.4	6.6	22.1	72.8	239.3
1982	24.5	1.5	0.7	2.3	6.2	22.2	70.2	230.6
1983	23.6	1.5	0.7	2.3	6.2	21.4	72.0	241.2
1984	23.9	1.6	0.7	2.3	6.4	22.1	74.3	255.0
1985	24.0	1.5	0.7	2.0	5.5	20.8	73.6	253.0
1986	22.7	1.4	0.6	1.7	4.1	18.0	68.4	240.0
1987	22.9	1.2	0.6	1.6	4.0	18.1	69.1	241.7
1988	21.6	1.3	0.7	1.8	4.2	18.3	69.0	243.9
1989	20.2	1.2	0.7	2.0	4.8	19.1	67.7	232.9
1990	19.9	1.1	0.6	2.1	5.0	20.1	66.7	208.1
1991	20.6	1.1	0.7	2.2	5.4	20.2	69.1	223.0
1992	20.8	1.1	0.7	2.5	6.5	22.5	70.9	224.8
1993	21.3	1.2	0.7	2.9	8.4	28.1	80.0	292.0
1994	21.5	1.2	0.7	3.0	9.4	32.3	83.9	315.0
1995	20.5	1.2	0.7	3.3	8.9	30.3	81.0	225.2
1996	20.2	1.1	0.6	3.1	7.8	26.6	75.5	234.4

TABLE 48 (continued)

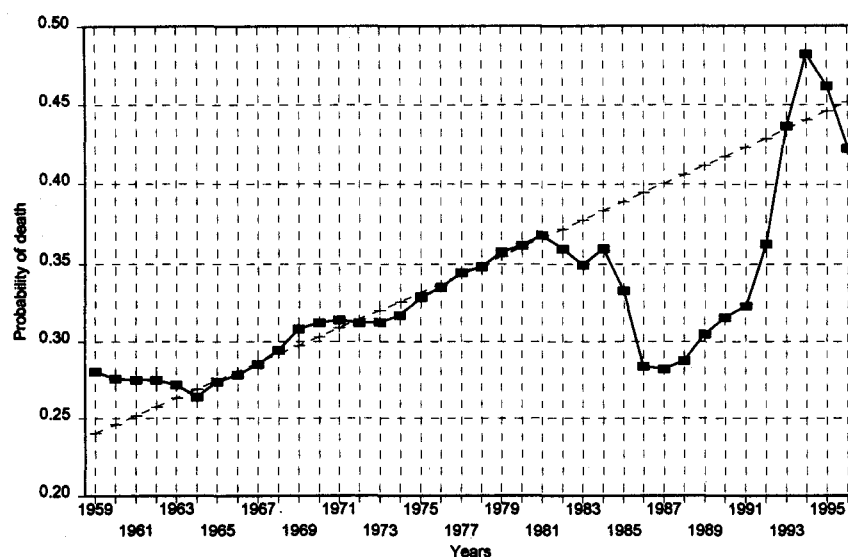
## Females

Years	Age group							
	0	1-4	5-14	15-24	25-44	45-64	65-84	85 and over
1946	124.3	19.9	2.8	3.0	4.4	11.4	53.4	159.1
1947	175.1	32.0	3.1	3.2	4.9	13.6	64.0	191.1
1948	117.4	29.6	2.5	2.8	4.1	10.7	50.4	144.7
1949	117.1	16.0	2.0	2.5	3.6	9.5	44.7	127.1
1950	107.1	14.7	1.8	2.3	3.4	9.1	44.4	134.9
1951	114.8	15.4	1.9	2.2	3.1	9.0	43.6	135.8
1952	94.6	11.1	1.6	1.9	2.9	8.9	45.6	150.4
1953	86.0	9.7	1.5	1.8	2.8	8.7	46.0	158.1
1954	88.5	9.6	1.4	1.6	2.6	8.4	45.9	159.4
1955	73.7	7.6	1.2	1.4	2.4	7.9	41.3	153.8
1956	57.7	5.9	1.0	1.2	2.2	7.4	39.2	147.7
1957	55.5	5.3	0.9	1.2	2.3	8.1	42.7	162.7
1958	47.2	3.9	0.8	1.1	2.1	7.5	38.1	148.1
1959	37.6	2.9	0.8	1.1	2.0	7.5	40.3	163.9
1960	33.5	2.4	0.7	1.0	1.9	7.2	37.9	155.5
1961	29.2	2.1	0.6	0.9	1.8	7.2	39.0	157.8
1962	28.2	2.0	0.6	0.9	1.8	7.4	41.3	183.8
1963	27.2	1.7	0.6	0.9	1.8	7.2	39.5	169.8
1964	25.3	1.5	0.5	0.8	1.7	6.9	36.9	157.9
1965	23.2	1.5	0.5	0.8	1.7	6.9	39.6	173.7
1966	22.4	1.3	0.5	0.8	1.7	6.9	38.8	169.9
1967	22.0	1.4	0.5	0.7	1.6	7.0	39.8	178.6
1968	22.3	1.3	0.5	0.7	1.6	7.0	39.1	173.4
1969	21.2	1.3	0.4	0.7	1.7	7.2	40.7	180.7
1970	19.9	1.4	0.5	0.7	1.7	7.2	40.6	181.9
1971	17.6	1.2	0.4	0.7	1.6	7.2	39.9	176.8
1972	18.0	1.2	0.5	0.7	1.6	7.2	40.5	181.7
1973	18.0	1.3	0.4	0.7	1.6	7.2	40.7	188.3
1974	18.9	1.2	0.5	0.7	1.6	7.2	39.2	170.3
1975	19.8	1.3	0.4	0.7	1.7	7.5	40.9	183.2
1976	21.1	1.3	0.4	0.7	1.7	7.7	40.9	187.8
1977	20.4	1.3	0.5	0.7	1.7	7.7	40.0	186.8
1978	20.1	1.3	0.4	0.7	1.7	7.9	39.7	182.4
1979	19.4	1.3	0.4	0.7	1.7	8.0	41.0	195.7
1980	19.0	1.3	0.4	0.7	1.8	8.1	41.0	199.5
1981	18.7	1.4	0.5	0.7	1.7	8.0	40.9	199.1
1982	18.0	1.2	0.4	0.6	1.6	8.0	39.5	191.9
1983	17.5	1.2	0.4	0.6	1.6	8.0	40.1	195.0
1984	17.8	1.3	0.4	0.7	1.7	8.1	41.7	211.4
1985	17.5	1.2	0.4	0.6	1.6	7.9	41.6	215.9
1986	16.6	1.1	0.4	0.6	1.3	7.1	39.5	199.8
1987	16.5	1.0	0.3	0.6	1.3	7.0	40.3	198.9
1988	15.8	1.0	0.4	0.6	1.3	7.0	40.1	204.4
1989	14.7	0.9	0.4	0.7	1.3	7.1	38.6	193.0
1990	14.5	0.9	0.3	0.7	1.4	7.3	38.9	181.9
1991	15.1	0.9	0.4	0.7	1.5	7.3	39.0	192.4
1992	15.2	0.9	0.4	0.8	1.7	7.9	39.7	194.1
1993	16.3	1.0	0.4	0.9	2.1	9.5	43.7	216.6
1994	15.9	0.9	0.4	0.9	2.4	10.7	45.8	220.9
1995	15.5	1.0	0.4	0.9	2.3	10.1	43.9	212.0
1996	14.9	0.8	0.4	0.9	2.0	9.1	42.5	197.8

Source: E. M. Andreev, L. E. Darsky and T. L. Kharkova, *Demographic history of Russia: 1927-1959* (Moscow, 1997, in press, in Russian); author's calculations based on national statistics.

NOTE: Death rates for age groups 15-24; 25-44; 45-64 and 65-84 were standardized by age. World standard of World Health Organization was used.

Figure 83. Dynamics of death probability of men age 20-59 in Russia, 1959-1996



Source: Author's calculations based on national statistics.

TABLE 49. THE COMPONENT ANALYSIS OF LIFE EXPECTANCY, SELECTED AGE GROUPS, RUSSIA, 1946 – 1996

Period	Life expectancy		Changes of life expectancy during the period as result of changes in mortality in the specific age groups					
	in the beginning of period	in the end of period	Total	0-4	5-24	25-44	45-64	65 and over
<b>Males</b>								
1946 - 1959	47.0	62.8	15.8	8.93	1.91	0.81	2.05	0.74
1959 - 1965	62.8	64.6	1.8	1.33	0.21	0.05	0.21	0.18
1965 - 1980	64.6	61.4	-3.1	0.28	-0.19	-0.23	-1.49	-0.84
1980 - 1984	61.4	61.7	0.3	0.13	0.11	0.09	-0.07	-0.05
1984 - 1987	61.7	64.9	3.2	0.15	0.31	0.28	1.05	0.28
1987 - 1994	64.9	57.6	-7.2	0.10	-0.59	-0.58	-3.19	-0.71
1994 - 1996	57.6	59.8	2.1	0.09	0.01	-0.01	1.00	0.32
<b>Females</b>								
1946 - 1959	55.7	71.0	15.3	9.37	1.94	0.86	1.27	1.19
1959 - 1965	71.0	73.4	2.4	1.52	0.35	0.18	0.25	0.01
1965 - 1980	73.4	72.9	-0.5	0.33	0.09	0.05	-0.46	-0.36
1980 - 1984	72.9	73.0	0.2	0.18	0.05	0.02	-0.02	-0.14
1984 - 1987	73.0	74.3	1.3	0.18	0.09	0.06	0.45	0.24
1987 - 1994	74.3	71.2	-3.2	0.06	-0.21	-0.19	-1.49	-0.73
1994 - 1996	71.2	72.5	1.3	0.08	0.02	0.01	0.56	0.42

Source: Author's calculations based on national statistics.

NOTE: Methods of calculation are presented in: Y. A. Korchak-Chepurkovskiy Y. A., "The influence of mortality in different ages on expectation of life expectancy", in *Soviet demography for 70 years*, T. V. Ryabushkin, ed. (Moscow, Nauka, 1987, pp. 263-280, in Russian).

usually referred to as "period effects" and they are approximated for a calendar year. The historical factors represent the influence of the past on current mortality and are usually referred to as "cohort effects". Cohort effects occur whenever events in the past have a lasting impact, felt by most people of the same age group or generation. In this paper we used the

APC model with age and sex for period analysis and with sex for cohorts. We used the estimation algorithm based on maximum likelihood with a Poisson distribution, the same method used by Willekens and Scherbov (1991, 1992). (We would like to thank Dr. V. Golubkov who made available to us his program for APC analysis).

The subject of analysis was age and sex specific morality rates for ages 10-84 years from 1946 to 1996. Such a global model can not be very precise, but for the age interval 20-69 and the period 1965-1980 the quality of the model's approximation to the empirical data is rather high. To have a general evaluation of the APC model for each sex we calculated the coefficient of determination,  $r^2$ , the proportion of the variance in the original data that is accounted for by the APC model. This indicator reached 0.997 for males and 0.995 for females. The relative error of the estimate of probability of dying at ages 15-69 was 1.4 per cent for males and 1.9 per cent for females. This indicator reached a maximum value of 3 per cent for males (in 1980) and 2 per cent for females (in 1973).

The main results of the APC analysis are presented in figures 84 and 85. To enhance the visual presentation, exponential functions of the mortality effects are shown. The lines of "period effects" have practically the same configuration as the expectation of life (figure 82). The trends in cohort effects are particularly interesting. The cohort effect was nearly stable for the generation born before World War I. The unfavourable impact of cohort effects on mortality grew with year of birth for all cohorts from 1914 to 1941. It changes very little for generations born during the World War II years. The cohort effect declines for the birth cohorts of 1948 to 1966, and stabilises thereafter. The data are not sufficient to estimate cohort effects for those born before 1900 and after 1970.

#### D. MORTALITY BY CAUSES OF DEATH

As noted earlier, we have cause of death data for the years after 1959 (table 50). In this table death rates are age standardised using the World Health Organization World Standard.

The standardised death rates from two classes of causes of death "diseases of circulatory system" and "accidents, poisonings and violence" in general increased (table 50). The growth was quite steady from 1959 to 1984. The increase of mortality from the specified causes began in 1956, when the decline of mortality for men in ages 20-59 notably slowed down. The drop during the period of the anti-alcohol campaign (1985-1987) was replaced by

a significant new increase in 1992-1994. The decline in 1995-1996 partly recovered from this last increase. It should be noted that male and female mortality trends from these causes of death were different in scale but not in direction.

The standardised death rates due to neoplasms for males and for females declined from the beginning of the 1960s until the end of 1970s and then increased from 1980 to around 1995. However, the overall change in these rates was not significant. The minimum for males (in 1978) was 199 per 100,000 and the maximum (in 1993) 226. The minimum for female was 97 (in 1980) and the maximum (in 1962) was 123. One can see that the maximum female mortality from neoplasms was at the beginning but for males at the end of the period.

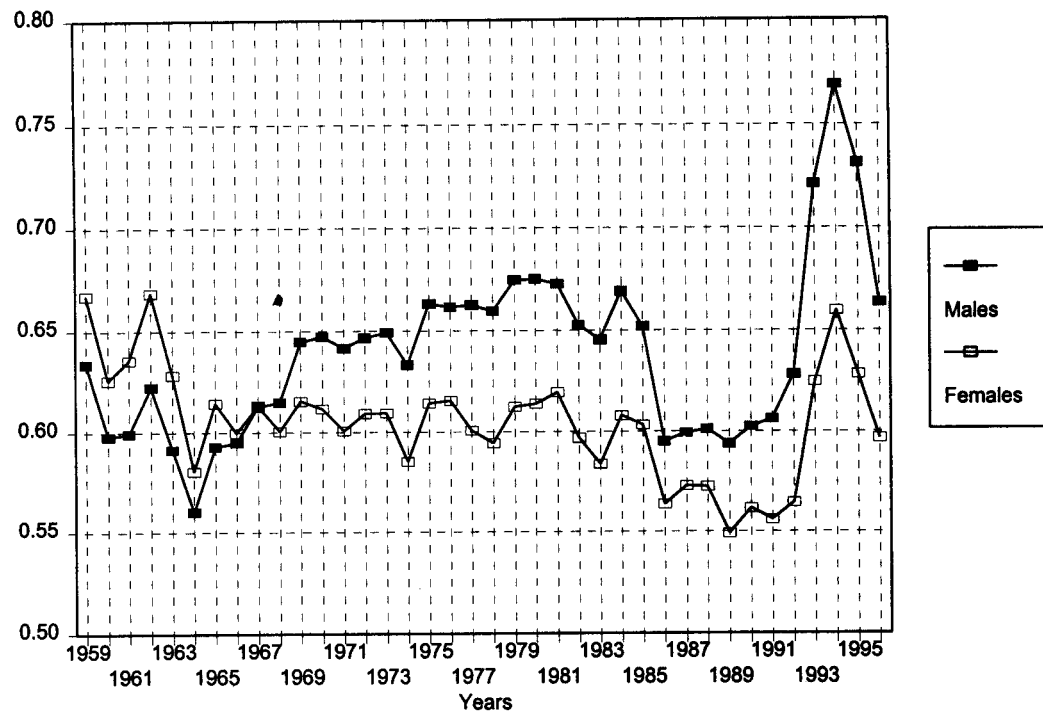
The general trends in mortality from infectious diseases, diseases of respiratory system and diseases of digestive systems for males and for females were very close. The standardised death rates from infectious diseases declined until 1993 but its reduction was slowing down. From 1959 to 1993 the standardised death rate from infectious diseases for males decreased by 76 per cent, and for females by 88 per cent. A small increase was observed in 1993-1996, which reflected a rise in mortality from tuberculosis.

Mortality from diseases of the respiratory systems was virtually stationary until 1980. A significant decrease of rates from this cause was observed from 1980 to 1991, for males by 44 per cent and for females, by 54 per cent. This trend was not connected with the anti-alcohol campaign, which was started later. One may suppose that the low mortality level in 1986-1991 was to a great extent due to an absence of epidemic influenza. The mortality level jumped in 1993-1994 and then fell again in 1995-1996.

The trend in mortality from diseases of the digestive system was irregular. It is possible that declines in mortality from these causes between 1959 and 1995 were in part an artefact of the reclassification of deaths by cause between digestive system and infectious diseases. They had originally been classified in accordance with the 1981 Soviet system. Changes in

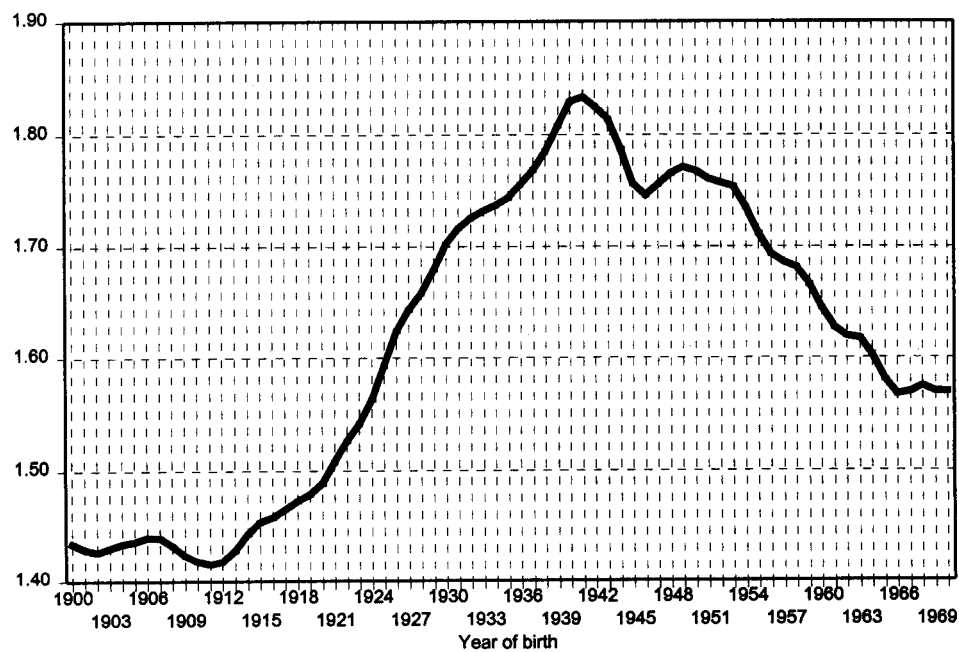


**Figure 84. APC analysis of Russia's mortality data. Period component of mortality dynamics, 1959-1995**



Source: Author's calculations based on national statistics, see text.

**Figure 85. APC analysis of Russia's mortality data. Cohort component of mortality dynamics, 1900-1969**



Source: Author's calculation based on national statistics, see text.

TABLE 50. MORTALITY BY CAUSE OF DEATH AND SEX  
(STANDARDISED DEATH RATES PER 100,000), RUSSIA, 1959-1996

*Males*

<i>Years</i>	<i>All causes</i>	<i>Diseases of circulatory system</i>	<i>Neoplasms</i>	<i>Diseases of respiratory system</i>	<i>Infectious and parasitic diseases</i>	<i>Diseases of digestive system</i>	<i>Other diseases</i>	<i>Accidents, poisoning and violence</i>
1959	1 156	354	215	139	114	43	156	135
1960	1 099	348	216	114	89	41	144	147
1961	1 086	378	214	114	81	37	117	146
1962	1 115	366	214	128	88	36	140	144
1963	1 070	391	210	113	73	34	109	140
1964	1 017	370	207	108	53	33	95	151
1965	1 062	406	208	105	51	32	98	162
1966	1 069	409	205	102	55	35	91	172
1967	1 096	421	205	120	54	33	85	179
1968	1 117	437	201	122	49	35	81	192
1969	1 184	476	203	136	46	36	83	205
1970	1 202	497	203	145	43	34	69	212
1971	1 191	502	204	131	42	34	65	213
1972	1 194	512	202	133	40	34	64	211
1973	1 195	514	202	139	37	33	64	207
1974	1 187	507	199	128	34	34	65	220
1975	1 242	539	199	139	37	35	65	228
1976	1 255	541	201	142	36	35	66	235
1977	1 277	553	202	138	36	35	66	247
1978	1 277	563	199	136	34	34	65	246
1979	1 314	586	201	137	35	36	67	254
1980	1 325	589	201	136	34	40	68	257
1981	1 318	584	205	125	33	38	75	257
1982	1 280	573	209	113	31	37	73	244
1983	1 277	580	207	112	28	38	73	238
1984	1 321	606	210	115	29	39	78	245
1985	1 262	590	210	112	28	37	78	207
1986	1 124	542	215	87	22	31	73	154
1987	1 126	547	217	84	21	31	76	150
1988	1 137	542	219	85	21	30	74	166
1989	1 151	532	221	80	24	31	72	191
1990	1 156	523	218	79	19	31	86	201
1991	1 196	536	223	76	19	31	97	213
1992	1 284	558	223	79	21	36	110	256
1993	1 542	673	226	103	28	42	127	342
1994	1 689	747	224	114	32	49	145	378
1995	1 591	689	217	103	33	51	141	356
1996	1 461	640	210	93	35	46	127	311

the quality of diagnosis also may also help explain this irregular mortality trend. On the other hand, the increase in the most recent years was clearly the result of real mortality growth from chronic diseases such as stomach ulcers and cirrhosis of the liver.

Components analysis (Andreev, 1982) permits the estimation of the cause-specific impact on life expectancy (table 51). One can see that from 1959 to 1996, male life expectancy decreased by 3 years due to growth in mortality from diseases of circulatory system. Mortality

TABLE 50 (continued)

*Females*

<i>Years</i>	<i>All causes</i>	<i>Diseases of circulatory system</i>	<i>Neoplasms</i>	<i>Diseases of respiratory system</i>	<i>Infectious and parasitic diseases</i>	<i>Diseases of digestive system</i>	<i>Other diseases</i>	<i>Accidents, poisoning and violence</i>
1959	684	253	122	78	59	25	113	35
1960	624	249	122	63	42	23	89	36
1961	629	272	121	62	36	20	83	35
1962	654	269	123	69	39	19	99	35
1963	624	289	120	60	31	19	72	34
1964	587	271	117	52	25	18	69	36
1965	608	299	115	52	19	17	69	38
1966	600	294	112	50	20	19	66	41
1967	611	304	110	62	18	17	59	41
1968	603	302	108	61	16	18	56	43
1969	619	317	106	65	14	18	55	45
1970	617	320	105	69	13	17	48	46
1971	600	320	105	60	13	16	41	46
1972	608	327	103	62	12	15	43	47
1973	612	330	102	65	11	16	42	47
1974	596	317	102	58	11	16	43	49
1975	624	337	101	64	12	15	44	51
1976	633	342	100	65	13	16	44	54
1977	626	340	98	60	13	16	43	56
1978	623	341	98	59	13	15	42	57
1979	640	356	98	57	13	15	44	58
1980	644	359	97	56	13	16	44	59
1981	640	358	98	51	13	16	46	58
1982	621	348	99	44	12	16	46	56
1983	623	353	99	43	12	16	47	55
1984	649	374	98	44	11	16	50	57
1985	640	372	98	42	10	16	51	52
1986	592	344	99	34	9	14	50	42
1987	592	347	100	32	8	15	51	40
1988	595	346	100	32	8	15	51	43
1989	579	330	100	29	9	14	50	46
1990	579	322	100	27	6	15	61	48
1991	591	323	101	26	6	15	68	51
1992	614	330	102	26	6	16	74	60
1993	698	378	102	30	7	18	84	79
1994	744	405	103	30	8	21	90	86
1995	712	382	101	28	8	22	89	82
1996	674	364	99	26	8	59	85	72

*Source:* Author's calculations based on national statistics. World standard of World Health Organization was used for age standardisation.

trends for accidents, poisonings and violence had a similar effect. Reductions in mortality from all other causes compensated for half of this decline. For females the negative effects of mortality from diseases of circulatory system and accidents, poisonings and violence were smaller (1.7 and 0.9 years, respectively)

and the net gain in life expectancy was 1.5 years.

Visual analysis can show the extent to which classes of causes of death are or are not homogeneous in their impact on standardised death rates. It is sometimes found that mortality

TABLE 51. COMPONENT ANALYSIS OF LIFE EXPECTANCY BY CAUSE OF DEATH AND SEX, RUSSIA, 1959-1996

Periods	Life expectancy		Changes of life expectancy during the period as result of changes in mortality from specific cause of death					
	in the beginning of period	in the end of period	Total	Diseases of circulatory system	Neoplasms	Infectious diseases and diseases of respiratory and digestive systems	All other diseases	Accidents, poisoning and violence
Males								
1959-1965	62.8	64.6	1.8	-0.53	0.03	1.84	0.99	-0.55
1965-1980	64.6	61.4	-3.1	-1.79	0.07	0.21	0.06	-1.69
1980-1984	61.4	61.7	0.3	-0.09	-0.07	0.29	-0.06	0.27
1984-1987	61.7	64.9	3.2	0.65	-0.05	0.66	0.12	1.77
1987-1994	64.9	57.6	-7.2	-2.12	-0.08	-0.42	-0.96	-3.67
1994-1996	57.6	59.8	2.1	0.81	0.13	0.11	0.23	0.85
Females								
1959-1965	71.0	73.4	2.4	-0.61	0.11	1.66	1.29	-0.05
1965-1980	73.4	72.9	-0.5	-1.09	0.36	0.41	0.30	-0.50
1980-1984	72.9	73.0	0.2	-0.08	-0.01	0.34	-0.15	0.05
1984-1987	73.0	74.3	1.3	0.49	-0.03	0.40	-0.01	0.45
1987-1994	74.3	71.2	-3.2	-1.11	-0.07	-0.01	-0.80	-1.16
1994-1996	71.2	72.5	1.3	0.70	0.07	0.09	0.15	0.30

Source: Author's calculations based on national statistics. Methods of calculation are presented in: E. M. Andreev, "Method of components in cause of death analysis", *Statistics Herald*, No. 9, pp. 42-47 (1982, in Russian).

trends are remarkably similar, even for causes that are medically very different. To explore and then to try to explain such differences and similarities for Russia from 1965 through 1996, we use a formal algorithm to identify clusters of causes of death on the basis of trend patterns. The subject of the analyses was standardised death rates at ages 20 and over. The technique used was the Generalised Agglomerative Hierarchical Clustering Algorithms with internal influence measurement (Milligan and Sokol, 1980).

Several specifications of the model were tested. The final model used was the Complete Linkage Method. The actual procedure used was Pearsonian correlation. As a result we obtained 21 clusters of causes of death. Six of the clusters account for some 88 per cent of all male and for 92 per cent of female deaths. We excluded from the model any cause of death that had a maximum standardised death rate in 1965-1996 at ages 20 and over that was less than 0.1 per 100,000. The results of our clustering exercise are presented in the annex. The clusters are ranked by average value of standardised death rates in 1965-1996. The mortality trends for clusters one through six are also shown in figure 85.

During the period 1965-1996, standardised death rates at age 20 and over increased by 43 per cent for males and by 15 per cent for females. The largest part of this growth was associated with the first cluster of causes of death. Thus, the first cluster of causes on average accounted for 32 per cent of male deaths for the period 1965-1996, and for 70 per cent of the mortality increase.

The existence of a common trend for the causes of death in such a cluster suggests that there are common underlying explanatory factors. This is despite the fact that each cluster includes diseases that are markedly different from an etiologic point of view. For example, the first cluster for females includes: cerebrovascular disorders and acute myocardial infarction without hypertensive disease, malignant neoplasm of breast, malignant neoplasm of the trachea, bronchus and lung, and motor vehicle traffic accident involving collision with pedestrian.

This observation leads to our first conclusion: The negative force of mortality in Russia has a global character because it produced mortality increases from etiologically different causes of death.

A second conclusion refers to the effect of the anti-alcohol campaign. We propose to measure the effect of the anti-alcohol campaign on mortality trends of some clusters of causes as follows:

$$CDR(1987,J)-[CDR(1984,J)+CDR(1990,J)]/2$$

where  $CDR_{(n,J)}$  are the standardised death rates at age 20 and over in period "n" from causes of cluster J. For all causes of death this indicator equalled 183 per 100,000 for males at age 20 and over and 34 for females. The effect for the first cluster was 15.1, or 8.3 per cent of the overall effect. About 80 per cent of the overall effect was related to a decline in mortality causes in clusters 5, 6 and 7. The full impact of this group on mortality increase was 22 per cent of the total increase in 1965-1984. This observation leads then to our second conclusion: The mortality increase in 1965-1985 and its decrease in 1985-1987 were related to different clusters of causes of death, rather than to any one single cause.

#### E. MORTALITY IN RUSSIA BY LEVEL OF EDUCATION

Unfortunately, we have at our disposal very limited data on social differences in mortality trends. For the period after the 1989 census we have only some indicators of mortality by level of education.

Table 52 presents indirectly standardised indices of mortality by level of education. The estimates are based on data from the 1989

census, the 1994 micro-census, and registered deaths at age 16 years and over by level of education. The age-specific death rates for the total population of Russia in 1988-1989 were taken as the standard.

As shown in the table, the largest increase of mortality was among people with secondary general education and the least was among those with higher and unfinished higher education. At the same time, given the considerable increase of mortality in Russia, the differential changes by level of education were not very great. Differences between those with secondary general education and secondary special education increased, and differences in mortality between males with secondary general and unfinished secondary education nearly disappeared.

Differential mortality trends between educational groups has two possible explanations which supplement one other:

First, the direct effect of anti-alcohol measures had a stronger impact on those with lower levels of education, where alcohol consumption had been greater. After the anti-alcohol campaign ended, the mortality increase in these groups was greater.

Second, lower levels of general culture and a less healthy life style may have created more obstacles to the adaptation to new socio-economic conditions in the transition to a market economy. This circumstance could not but lead to more rapid mortality growth. As seen from the results of public polls published in the

TABLE 52. MORTALITY BY LEVEL OF EDUCATION AND SEX, RUSSIA, 1988-1994  
(STANDARDISED MORTALITY INDEXES AT AGE 16 AND OVER)

Level of education	1988-1989		1993-1994		1993-1994, Total=1		Increase for 5 years (per cent)	
	Males	Females	Males	Females	Males	Females	Males	Females
Total	1.00	1.00	1.49	1.24	1.00	1.00	49	24
Higher and unfinished higher	0.61	0.68	0.77	0.70	0.52	0.56	26	2
Secondary special	0.73	0.74	1.06	0.81	0.71	0.66	45	10
Secondary general	1.13	1.11	1.95	1.53	1.31	1.23	73	38
Incomplete secondary	1.32	1.00	1.94	1.12	1.30	0.90	47	12
Primary and lower	0.99	1.04	1.46	1.40	0.98	1.13	48	34

Source: Author's calculations based on national statistics.

mass media, the negative attitude towards transformations in the country is most common among those with less education.

#### F. REGIONAL DIFFERENCES IN MORTALITY

The analysis of interregional mortality differences is based on data for the various local jurisdictional units of the Russian Federation.

In 1996, expectation of life at birth varied for males from 49.5 to 65.3 years, and for females from 62.4 to 75.1 years. The lowest of expectation of life at birth was observed in Tuva and the highest in Dagestan. Infant mortality rates ranged from 11.5 in Leningrad Oblast to 36.4 in the Jewish Autonomous Oblast.

We have detailed data on mortality in Russia by administrative divisions only for the period after 1978. Before that, life tables by regions of Russia were prepared only for the years of population censuses, and were not compiled for all territories.

Given the circumstances, we used indirectly standardised indices to analyse territorial differences in mortality, based on 1970 census data. (Andreev, 1979). The analysis clearly demonstrated that mortality levels in Russia rise on a gradient from Southwest to Northeast. This geographic pattern, which was also observed by Shkolnikov and Vasin (1994), has persisted up to the present (table 53).

There are other illustrations of the stability of inter-regional mortality differences. The correlation between expectations of life at birth in the territories of Russia at different time periods is very high. For example, the correlation coefficient of regional life expectancy in 1978-1979 with that of 1994 is 0.75 for males and 0.86 for females. Such high correlation coefficients indicate that the character of changes in expectation of life by territories is nearly simultaneous. The only exception occurred in the period of the anti-alcohol campaign, especially for males. The correlation coefficient for male life expectancy in 1982-1983 with 1986-1987 was 0.37. However, the mortality decline in 1994-1996 did not change the pattern of interregional differences in life expectancy. The correlation coefficient for levels of expectation of life in 1994 with 1995 was 0.92 for both males and females.

Similar regional patterns of mortality difference are found for both male and female life expectancy. Both sexes live longer in the southern regions of European Russia (including the North Caucasus), where there is a higher density of rural population, than they do in the poorly settled regions in the northern and the far-eastern parts of the country. The Central European and South Asian regions of Russia have intermediate levels of mortality.

It is useful to examine the territorial differentiation of mortality due to the first cluster of causes of death, which produced most of the

TABLE 53. LIFE EXPECTANCY IN THE GEOGRAPHICAL REGIONS OF RUSSIA, 1996

	Total population		Urban population		Rural population	
	Males	Females	Males	Females	Males	Females
Russian Federation	59.8	72.5	60.2	72.7	58.4	71.9
North Region	58.8	71.7	59.4	71.9	56.8	71.0
North East Region	60.6	72.9	61.3	73.2	56.4	70.5
Central Region	59.8	73.0	60.6	73.3	56.5	71.6
Volga-Vyatka region	60.7	73.4	61.4	73.6	58.8	72.7
Central European Russia Region	61.2	74.3	62.5	74.7	59.1	73.6
Volga region	61.0	73.8	61.2	73.8	60.2	73.4
North-Caucasus Region	61.5	73.4	61.9	73.5	61.1	73.2
Ural Region	59.6	72.5	60.0	72.6	58.3	72.1
West Siberian Region	59.0	71.7	59.1	71.9	58.7	71.2
East Siberian Region	56.9	70.1	57.1	70.4	56.4	69.2
Far East Region	58.0	70.0	58.1	70.2	57.5	69.3

Source: Author's calculations based on national statistics.

increase between 1965 and 1996. In 1995, the standardised male death rates at ages 20 and over due to this cluster of causes varied from 1578 per 100,000 in Magadan Oblast to 323 in the Republic of Dagestan. The five geographical areas with the next highest levels of mortality due to this cluster were Sakhalin Oblast (1405), Novgorod Oblast (1286), Sverdlovsk Oblast (1232), Tver Oblast (1209) and Khabarovsk Kray (1201). These geographical areas are typical of the Far East, Ural, NorthWest and Central regions.

The five geographical regions with the next-lowest levels of mortality due to this cause were: the Ingush republic (361), the Republic of Kalmykia (595), the Republic of Tuva (635), and Belgorod Oblast (645). Five of these six regions (excluding Belgorod Oblast) suffer from a low quality of cause of death statistics. If attention is redirected to the six territories with higher quality of cause of death statistics and with very low levels of mortality from these causes, we find Belgorod Oblast (645), Moscow City (701), Voronezh Oblast (724), the Republic of Tatarstan (735), St. Petersburg City (737) and Penza Oblast (750). This list includes two of Russia's largest cities and the Central European Russia and Volga regions that have favourable climatic conditions.

The pattern of regional differences in female mortality from causes of the first cluster is similar to that of males.

## G. DISCUSSION

### 1. *The peculiarity of the second phase of the epidemiological transition in Russia*

The second phase of the epidemiological transition was completed in Russia only in the 1960s. The investigations conducted by R. Sifman (1979) in 1945-1946 and published posthumously at the end of the 1970s demonstrated that the mortality decline in this period was based mainly on the introduction of sulfa drugs. It is known that sulfa drugs and penicillin were supplied to the USSR in limited amounts by the United States beginning in 1944-1945. Later, these drugs were manufactured in the USSR. Through use of sulfa drugs alone, the infant mortality rate in the post-war

year 1946 was reduced by a factor of 1.8 times lower than in 1940 (Andreev and others, 1993). Considering the difficult economic situation in the USSR in the immediate post-war period, one can conclude that the use of more effective medicines was the major factor in the adult mortality decline of 1947-1950.

Thus, the phase two mortality decline in Russia was much more like that of the developing than of the developed countries. The classic analysis of the data on England and Wales (McKeown and others, 1975) unambiguously indicates that in the developed countries, mortality decline due to reduction of infectious diseases took place before the availability of effective medical methods of prevention and cure. The similar timing of the mortality reduction in the other countries of Western Europe indicates that the same mechanism was at work. However, the contribution of modern medical technology was of much greater significance in the countries, where the principal mortality declines were achieved after World War II (United Nations, 1973, pp. 152-155).

After 1953, the end of the large-scale repression made a significant contribution to the improvement of life expectancy. The social reforms initiated by Nikita Krushchev played an important part. A leading factor was the construction of mass housing in the cities, which allowed millions of families to move out of basements, old and dilapidated structures, and overcrowded communal flats into more comfortable apartments.

The decline in expectation of life in Russia began in the mid-1960s. A slowing down of mortality reduction and even some increase were observed at the beginning of 1960s in many economically developed countries of the world. In nearly all of the more developed countries an increase of mortality from diseases of the circulatory system was observed among men at ages 45 and over (United Nations, 1973, p. 130).

In the Western countries, the alarming changes of mortality generated an effective social response. There were calls to strengthen environmental protection, reduce accidents, improve personal hygiene and to increase health education. The positive effect of these meas-

ures appeared in these countries by the mid-1970s, at which point a steady mortality decline was resumed. In the USSR, on the other hand, the government responded to the negative mortality trends by suppressing publication of all indices except the general mortality rates. It should be emphasised that, in the Russian Federation from 1931 to 1986, no mortality information was published except for general mortality rates and the absolute numbers of deaths.

The reasons for the increase of mortality in Russia in 1965-1980 and in the later period have been widely and thoroughly discussed (Anderson and Silver 1990, 1994; Andreev 1990, 1994; Avdeev and others, 1997; Biryukov, 1984; Blum, 1994; Blum and Monnier, 1989; Feshbach and Friendly, 1992; Okolski, 1993; Shkolnikov, Mesle and Vallin, 1995; Zakharov, 1997). Drawing on these and other authors, we have compiled a comprehensive schedule of factors, which *could* have negatively influenced Russia's mortality trends. Unfortunately, it is not possible to precisely weigh each of them, or even to rank them.

## *2. Historical factors*

In the 20th century, Russia went through World War I, civil war, famine in 1922 and 1933, a period of massive repression, and World War II. Each of these events certainly had negative effects on the health and mortality of Russians. In general, though, our APC model shows that the effect of historical factors is important but is not the main determinant of the mortality trends of the period 1965-1980.

Between 1964 and 1980, the probability of death for males of ages 20-69 increased from 479 to 564 or by 99 per 1,000 per year. The cohort effect explains 12 per cent of this increase. The period component accounts for 56 per cent of the increase, and the interaction effect for about another 30 per cent. For females the increase in the probability of death is smaller, 24 per 1,000; the cohort effects explain about 50 per cent and the period factors 50 per cent of the total increase.

## *3. Worsening of the ecological situation*

In general, efforts to modernise the Russian economy, including the numerous "great con-

structions", were ineffective. It is possible that precisely because of the ineffectiveness of the efforts, their negative demographic consequences were amplified. The attempt to construct modern industry was accompanied by a substantial worsening of environmental conditions in many areas and cities. There were radioactive catastrophes in the Urals, of the notorious chemical combine "Mayak" in 1957 and 1967 (Verbitskaya, 1997), and in Chernobyl in 1986. Industries were often built with insufficient safety measures. There was excessive and sometimes improper use of agricultural chemicals. All of these led to levels of environmental pollution that considerably exceeded the generally accepted limits. The number of studies objectively evaluating the state of the Russian environment has thus far been very limited. As a result, some authors (Feshbach and Friendly, 1992) have been obliged to depend heavily on press reports.

The period between 1959 and 1979 was one of intensive development of the chemical industry. During that time, the number of workers occupied in chemical production in Russia grew by a factor of 1.75 compared to overall growth of total employment by a factor of 1.32 (Central Statistical Board, 1973; Central Statistical Board, 1981).

At the beginning of the 1980s V. Biryukov (1984) uncovered notable differences in mortality among the cities of Russia and attempted to interpret them with environmental statistics. However, water quality and sanitary services were not found to be correlated with mortality. The author then simply ranked the cities according to their level of mortality from particular causes of death. Only later, mainly on the basis of the analysis of newspaper publications and foreign investigations (Feshbach and Friendly, 1992), has it emerged that almost all the cities with the highest mortality, also suffered from the worst environmental conditions.

We have compared Biryukov's data with estimates of their conditions made by Feshbach and Friendly (1992). The average decrease of expectation of life at birth for men between 1970 and 1979 in "clean" cities was 0.67 years, and in "dirty" ones 1.17 years.

However, the relative stability of mortality from neoplasms in the 1960s and through the



1980s raises doubts about the impact of environmental pollution on mortality. Possibly an increase of mortality from neoplasms does not result from a worsening of the environmental conditions, or possibly there is a substantial lag in the impact. Nevertheless, it has been indirectly substantiated that a stable level of mortality is accompanied by an increase of morbidity. Thus, in 1965 number of patients with newly diagnosed malignant neoplasms was 185 per 10,000 (Public health in the USSR, 1966, p. 298). In 1973 this number rose to 209.1, in 1985 it was 248 (Morbidity and Mortality of the population of the USSR, 1974, p. 98), and in 1994 it reached 280 (Public health in the Russian Federation, 1995, p. 15). Thus, a stable level of mortality from neoplasms was accompanied by a significant increase of mortality from malignant neoplasm of the trachea, bronchus and lung, all directly related not only to use of tobacco but also to atmospheric pollution.

Most recently, the decline in industrial activity has led to reduced industrial pollution. However, the residual effect of the pollution of previous years still remains.

#### *4. Psychological stress*

Another set of factors associated with mortality increase in the countries of Eastern Europe and in the former USSR has been proposed by Okolski (1993). He points to the widespread sense of disillusion with "real socialism". He writes, in particular, about the constant psychological stress arising from a comparison of standards of living in Eastern and Western Europe. Any such comparison was more glaring for citizens of the former German Democratic Republic than for the people of Russia. However, the absolute contradiction between the experience of everyday life and what was portrayed by the official propaganda—at a time of ever-increasing shortages of goods, the limitations on any personal initiative, the practical absence of any legal means to improve one's own circumstances—could not but lead to an acute sense of stress. After all, the beginning of the mortality increase was concurrent with Nikita Krushchev's dismissal and the public admission that the fruits of full communism could not be achieved in the USSR by 1980. Okolski argues that stress leads to an increase of mortality, primarily from diseases of the circu-

latory system. Moreover, such universally recognised factors as cigarette smoking, alcohol abuse, poor nutrition, hypertension, obesity, and diabetes all have more impact on mortality than complications in family life or at work, acute nervous tension, and neglect of one's own health.

The process of reforms, while perhaps diminishing some stress factors, at the same time has brought to Russian society many new ones. During the "era of stagnation", there was a state of social stability and the majority of the population felt assured that for the time, their life would not get better, but would also not get worse. After the reforms started, many people have begun to feel justified anxiety for their personal well-being. The USSR was a paternalist state. In the process of reform, the responsibility for personal well-being was shifted to a substantial degree to the individual and the family, which also caused stress. Finally, inter-ethnic conflicts, both in Russia and in the former USSR, has further split the society into supporters and antagonists of the reforms, and has also led to increased social tension and stresses.

#### *5. Health care*

The health care system of the USSR was primarily oriented to problems typical of the second phase of the epidemic transition and, as we can see, it solved them quite successfully. However, to move on to the third phase requires the effective treatment of heart diseases and chronic diseases. To do so demands large-scale investments and a restructuring of the health care system as a whole.

Under conditions of the economic crisis, a further worsening of the state health care system took place. Organisational conservatism of the public health care system together with the growing cost of drugs further reduced the effectiveness of medical care. A new feature in the dynamics of mortality as compared to the period 1966-1980, was the rapid increase of mortality from chronic diseases such as tuberculosis, epilepsy, diabetes, stomach ulcers, cirrhosis of the liver, diseases of the pancreas. These chronic diseases usually do not contribute heavily to the total number of deaths. However, the increase of mortality from these causes can be

explained only by the increase of mortality of chronic invalids, whose life depends directly on the functioning of the health care system and access to medication. Before prices were de-controlled, the shortage of medicines was a widespread disaster. Today, one may assume that people refuse to buy medicines not only because of their high cost, but also due to the strong conviction, cultivated over many decades, that medicines and medical services should be cheap.

#### *6. Life-style factors*

Many small-scale studies show that medically uninformed and at times harmful behaviour is widespread in Russia. These life-style problems include alcohol abuse, smoking, overeating and poor eating habits. There is often only belated resort to medical assistance even in the cities, where this assistance is generally more accessible (Andreev, 1988). It appears that such social-cultural factors alone contribute substantially to differences in mortality between groups with different levels of education (Andreev and Dobrovol'skaya, 1993).

It is quite evident that mass alcohol consumption at a time when the number of motor vehicles and other machines is increasing will lead to an increase of mortality from accidents. The rapid growth of life expectancy in 1985-1987 was mainly a result of strong anti-alcohol measures. The increase of mortality was resumed in 1988. Later, in 1992, after the anti-alcohol campaign had been abandoned, a drop in life expectancy was observed. Increase mortality caused by accidents was especially alarming (Shkolnikov and others, 1995), but it was a mirror reflection of the reduction that had occurred during the period of the anti-alcohol campaign.

However, we do not agree with the view that all of Russia's excess mortality is a result of drunkenness. There are three main reasons to doubt this hypothesis.

First, the data on territorial differences in alcohol consumption in the USSR (Trade in the USSR, Statistical abstract, Moscow, 1989, p. 50) shows that in 1980-1985, using sales as a proxy for consumption, Russia was not the republic with the highest per capita purchases.

The highest levels were observed in the Baltic republics.

Second, the causes of death that produced the rising mortality over the period 1965-1985 were not the same as the causes that were brought under control by the anti-alcohol campaign. This observation strongly supports the proposition that alcohol consumption was not the main factor behind the increase of mortality.

Third, the mortality decrease in 1995-1996 took place without any anti-alcohol measures. Nowadays, anyone can buy vodka anywhere, 24 hours a day, and at a relatively low price.

#### *7. Societal maladjustment*

As noted, the leading negative factors of mortality dynamics in Russia have a global character. Any change in living conditions and life-style may lead to societal maladjustment and to an increase in mortality. This view is presented in its most extreme form in the works of the Russian anatomist Davydovsky (1962). If such maladjustment continues or even increases, mortality may continue to rise.

It is evident that in the 1950s through the 1970s, the living conditions of Russians changed significantly. There was large-scale urbanisation, industrialisation, and mass migration to the newly developed areas. In 1957 the numbers of urban and rural inhabitants were equal; in 1975 the number of rural inhabitants fell to half that of the urban population. At present the rural is one-third the size of the urban population. From 1959 through 1979, the proportion of industrial workers in the labour force grew from 18.1 to 27.6 per cent (Central Statistical Board, 1973; Central Statistical Board, 1981).

The development of the extractive industries and of the areas in the North in general involved mass population movements to regions with harsh climatic conditions and higher levels of mortality. Thus, the population of the Hanty-Mansy Autonomous District increased between 1959 and 1979 by a factor of 3.6, in the Chukchi Autonomous District by 1.8, in the Yamalo-Nenets Autonomous District by 1.6. The population of Magadan Oblast grew by

76 per cent, the Republic of Yakutia by 72 per cent, Kamchatka Oblast by 71 per cent.

Unlike other economically developed countries, in the USSR and perhaps in the countries of Eastern Europe, the difficulties of the adaptation to changing conditions of life were not compensated by adequate improvement in the standard of living nor by improvements in the health care system. As a result, mortality rose.

It is useful to consider from this point of view the territorial differences in mortality from the causes of the first cluster. If one takes the mortality in Russia as whole as 100 per cent, then the level in urban area would be 105 for males and 107 for females, and in rural area 88 and 85 respectively. Levels 110 and higher were observed in five geographical regions: North (the Northeast part of European Russia), the Urals, West Siberian, East Siberian and the Far East. These are territories with high levels of urbanisation, high proportions of migrants, and high concentrations of industry. The high mortality from the first cluster of causes of death in these five regions supports the proposition that social maladjustment is a crucial factor.

It should be added that the transition to a market economy produces new kinds of maladjustment. Changes in the pattern of consumption caused by price decontrols may well impede further adjustment.

#### H. RUSSIAN MORTALITY IN A GLOBAL PERSPECTIVE

There is strong evidence to suggest that after its reduction in the later 1980s and its subsequent increase, mortality in Russia has in effect come back to the trajectory observed during the period 1964-1980. The following reasons support this proposition.

First, there are simply the trends in overall mortality (figures 82, 83, 84, and 85). Second, Avdeev and others, (1997) have shown that trends in age specific mortality after 1959 for ages 20-60 for every cohort may be projected on the basis of the trends over the period 1959-1980. The anti-alcohol campaign did not eliminate the trends, it only temporally interrupted

them. The mortality in each cohort is characterised by a short period of falling mortality in 1985-87, a brief stabilisation at a low level, and then a substantial increase and an evident tendency to return to the trajectory projected by the cohort's pre-1980 mortality.

Third, there is the obvious similarity of general changes of mortality by cause of death during the periods from 1965 to 1980 and from 1980 to 1994 (table 51). Thus, in 1965-1980 male life expectancy decrease was 3.1 and in 1980-1994 was 3.8 years. The decreases due to increases in diseases of the circulatory system were 1.8 and 1.6 years, respectively; those due to a rising numbers of accidents, poisoning and violence, 1.7 and 1.6 years respectively. The positive impacts of reductions in deaths due to infectious diseases and diseases of the respiratory and digestive systems in the first period were 0.2 years, and in the second 0.5. The impacts of neoplasms and all other diseases varied more significantly. In the first period, life expectancy rose due to reductions in deaths due to neoplasms by 0.1 year; in the second period it fell by 0.2. The corresponding indicators for "all other diseases" were 0.1 and -0.9.

Fourth, the analysis of mortality by local jurisdiction shows that the ranking by life expectancy before and after the anti-alcohol campaign remains very similar. With the exception of the period of the anti-alcohol campaign, inter-regional correlations of life expectancy over the various years is very high.

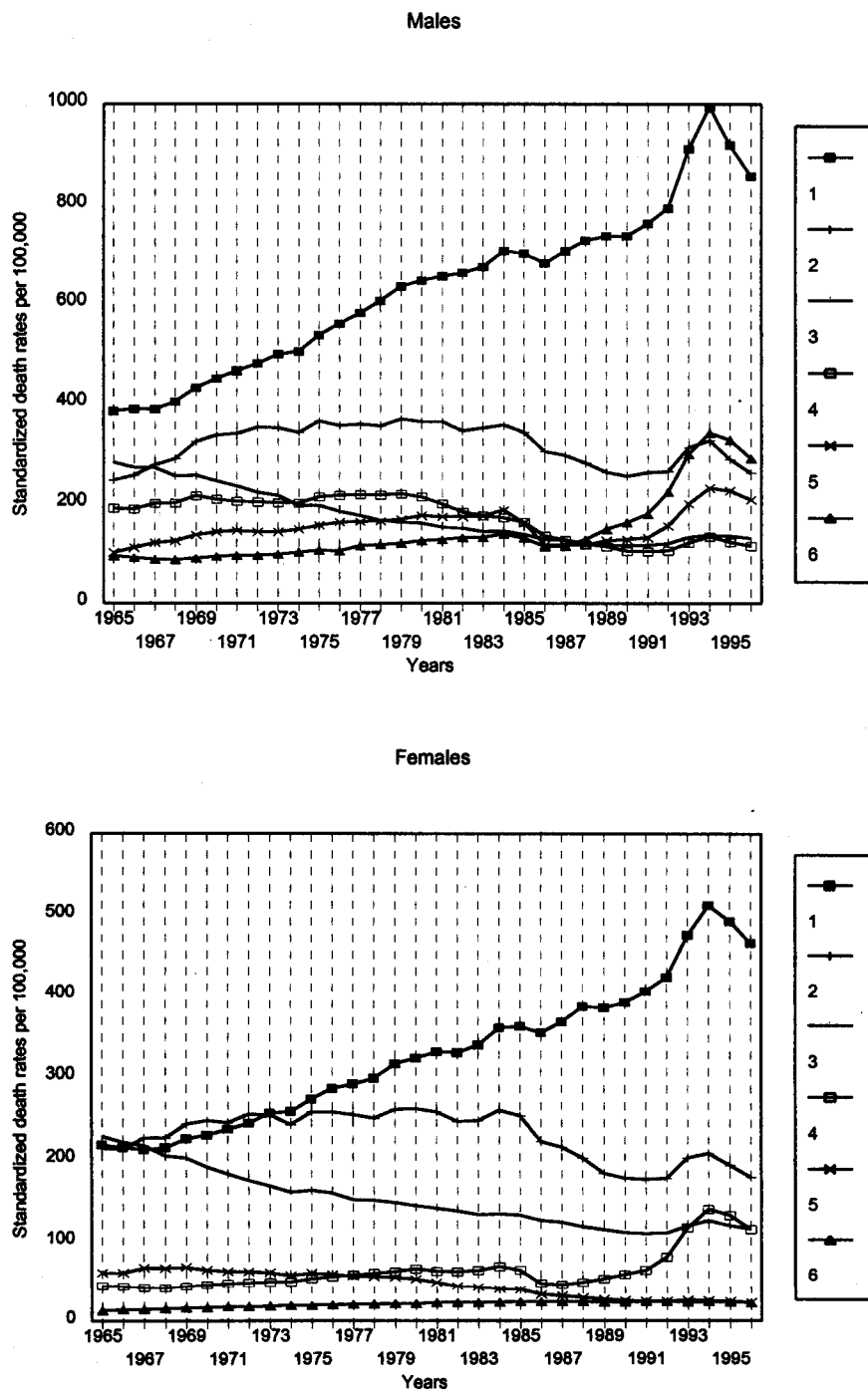
The decline of expectation of life in Russia in the period 1988 - 1994 was the result of an increase in adult mortality and a stagnation or very slow decrease of infant mortality. The increase of adult mortality had begun much earlier - in the mid-1960s. The socio-economic crisis of the 1990s could only strengthen the effect of those same factors that produced the rise in Russia's mortality over the years 1965-1980.

There are no grounds to believe that all of the cause of death clusters that produced the rising mortality of the 1965-1980 period have now lost their importance. The recent growth in life expectancy, which was comparable to the growth in the period of the anti-alcohol cam-

paign, raises many questions. We note that decomposition of the mortality trends by the various causes of death clusters makes the mortality decline in 1995 and 1996 more understandable. The significant decline was observed

in the same clusters where in 1993-1994 the significant increase took place: for males it was clusters 1, 2 and 5; for females 1, 2 and 4 (figure 86). These observations support the proposition that the decline was the result of excess

**Figure 86. Dynamics of mortality from six groups of causes of death, 1965-1995**  
(Standardized death rates per 100,000 at age 20 and over)



Source: Author's calculations based on national statistics, see text.

mortality in selected sub-populations in 1993-1994 and an adaptation of the population to the market economy, rather than that the changes are merely an artefact of a deteriorating vital statistics system. The mortality decline in Russia continues in 1997; according to our calculation, expectation of life at birth for males was 60.9 and was 72.7 for females. Nevertheless, we have no proof that this decline is more than a compensation for excess mortality a few years earlier.

A decline in the expectation of life of Russia's population has been observed for a period of more than 30 years. Under the circum-

stances, the pessimistic but most likely prospect is that the decline will continue.

In the mid-1960s the dynamics of mortality in Russia stalled in the second phase of the epidemic transition. The third phase has not yet started. Under the conditions of the current economic crisis, we can see only two significant new positive factors in the area of mortality. One is improved and more accessible vital statistic data, which may lead eventually to better public awareness of health issues. Secondly, there appears to be the beginning of some more constructive changes in the social response to the negative mortality trends.

# ANNEX

## RESULTS OF CLUSTERING OF CAUSES OF DEATH BY ITS DYNAMICS IN 1965-1996

### Grouping for males

#### Group 1

Cerebrovascular disorders without  
hypertensive disease  
Other ischaemic heart diseases without  
hypertensive disease  
Malignant neoplasm of trachea, bronchus and  
lung  
Acute myocardial infarction without  
hypertensive disease  
Diseases of arteries, arterioles and capillaries  
Malignant neoplasm of urinary organs  
Malignant neoplasm of rectum, rectosigmoid  
junction and anus  
Malignant neoplasm of larynx  
Malignant neoplasm of colon  
Malignant neoplasm of lip, oral cavity and  
pharynx  
Malignant neoplasm of prostate  
Other ischaemic heart diseases with  
hypertensive disease  
Leukaemia  
Infections of kidney  
Malignant neoplasm of bones, articular  
cartilage and connective tissue  
Non infective enteritis and colitis  
Malignant neoplasm of skin  
Malignant neoplasm of other respiratory and  
intrathoracic organs (thyroid gland, heart  
and mediastinum)  
Malignant neoplasm of other male genital  
organs  
Calculus of urinary tract  
Other congenital anomalies  
Nutritional and metabolic disorders and  
disorders involving the immune  
mechanism  
Congenital anomalies of heart  
Other bacterial infections excluding  
foodborne intoxications

#### Group 2

Atherosclerotic cardiosclerosis without  
hypertensive disease  
Motor vehicle traffic accident  
Septicaemia  
Other congenital anomalies of central  
nervous system

#### Group 3

Malignant neoplasm of stomach  
Respiratory tuberculosis  
Chronic rheumatic heart disease  
Acute myocardial infarction with  
hypertensive disease  
Hypertensive heart disease  
Benign and unspecified neoplasms  
Intestinal obstruction without mention of  
hernia  
Hernia of abdominal cavity  
Influenza  
Hypertensive renal disease  
Viral pneumonia  
Viral hepatitis  
Echinococcosis  
Hypertensive heart and renal disease  
Tuberculosis of bones and joints  
Other viral diseases  
Tuberculosis of nervous system  
Syphilis (all forms)  
Typhoid fever

#### Group 4

Cerebrovascular disorders with hypertensive  
disease  
Pulmonary congestion and hypostasis, and  
postinflammatory pulmonary fibrosis  
Bronchiectasis and other obstructive  
pulmonary diseases  
Accident caused by electric current  
Appendicitis  
Malignant neoplasm of small intestine,  
including duodenum  
Other diseases of circulatory system  
Pneumoconiosis and other lung diseases due  
to external agents  
Osteomyelitis and periostitis  
Other diseases of blood and blood-forming  
organs  
Tetanus

#### Group 5

Suicide and self-inflicted injury  
Other cirrhoses of liver  
Other accidental poisoning  
Accidental fall

Gastric ulcer  
 Accident caused by fire  
 Empyema, lung or mediastinal abscess  
 Diseases of pancreas  
 Alcoholic cirrhosis of liver  
 Meningitis, excluding infectious and parasitic  
 meningites  
 Diseases of the skin and subcutaneous tissue  
 Alcoholic psychosis

*Group 6*

Other heart diseases  
 Homicide and injury purposely inflicted by  
 other persons, including legal execution  
 Malignant neoplasm of other digestive organs  
 Injury undetermined whether accidentally or  
 purposely inflicted  
 Malignant neoplasm of other and unspecified  
 sites  
 Asthma  
 Diabetes mellitus  
 Other diseases of the nervous system and  
 sense organs  
 Duodenal ulcer and digestive ulcer of  
 unspecified site  
 Other psychoses  
 Multiple sclerosis  
 Mental retardation (oligophrenia)  
 Late effects of tuberculosis  
 Diphtheria

*Group 7*

Accidental poisoning by alcohol  
 Other accidents  
 Other acute pneumonias  
 Chronic alcoholism  
 Pneumococcal pneumonia  
 Other diseases of digestive system  
 Other diseases of liver and biliary tract  
 Shigellosis (dysentery)  
 Accidental drowning and submersion

*Group 8*

Atherosclerotic cardiosclerosis with  
 hypertensive disease  
 Other transport accidents  
 Accidental inhalation and ingestion causing  
 obstruction of respiratory tract, foreign  
 body entering other orifices  
 Other nephrites and nephroses, nephrotic  
 syndrome  
 Epilepsy  
 Other diseases of respiratory system  
 Acute intestinal infections due to unspecified

micro-organisms and ill-defined, including  
 toxical dyspepsia  
 Other diseases of genital organs

*Group 9*

Chronic bronchitis and emphysema  
 Motor vehicle traffic accident involving  
 collision with pedestrian  
 Other malignant neoplasms of lymphoid and  
 histiocytic tissue  
 Malignant neoplasm of breast  
 Misadventures to patients during medical  
 care

*Group 10*

Malignant neoplasm of oesophagus  
 Acute rheumatic fever  
 Acute nephritis and glomerulonephritis  
 Viral encephalitis  
 Other helminthiasis

*Group 11*

Other and unspecified hypertensive disease  
 Unspecified disorders of pericardium, mitral  
 and aortic valves  
 Accident caused by firearm missile  
 Tuberculosis of intestine, peritoneum and  
 mesenteric glands  
 Drug dependence

*Group 12*

Hyperplasia of prostate

*Group 13*

Gallstone diseases and cholecystitis  
 Bacterial foodborne intoxications excluding  
 salmonella infections  
 Other congenital anomalies of circulatory  
 system

*Group 14*

Other inflammatory diseases of the central  
 nervous system  
 Other tuberculosis excluding late effects  
 Meningococcal infection  
 Erysipelas  
 Intestinal infections due to other micro-  
 organisms including amoebiasis and other  
 protozoal intestinal diseases

*Group 15*

Phlebitis and thrombophlebitis, venous  
 embolism and thrombosis

*Group 16*  
 Other diseases of musculoskeletal system and  
 connective tissue  
 Gastritis and duodenitis  
 Other infectious diseases

*Group 17*  
 Other diseases of urinary system  
 Diseases of other endocrinous glands  
 Other parasitic diseases

*Group 18*  
 Anaemia

*Group 19*  
 Acute respiratory infections

*Group 20*  
 Inflammation of middle ear and mastoiditis

*Group 21*  
 Other salmonella infections

### Grouping for females

*Group 1*  
 Cerebrovascular disorders without  
 hypertensive disease  
 Other ischaemic heart diseases without  
 hypertensive disease  
 Diseases of arteries, arterioles and capillaries  
 Acute myocardial infarction without  
 hypertensive disease  
 Malignant neoplasm of breast  
 Malignant neoplasm of trachea, bronchus and  
 lung  
 Malignant neoplasm of colon  
 Malignant neoplasm of other and unspecified  
 sites  
 Diabetes mellitus  
 Motor vehicle traffic accident involving  
 collision with pedestrian  
 Other ischaemic heart diseases with  
 hypertensive disease  
 Leukaemia  
 Malignant neoplasm of urinary organs  
 Asthma  
 Infections of kidney  
 Accidental fall  
 Diseases of pancreas  
 Malignant neoplasm of bones, articular  
 cartilage and connective tissue  
 Malignant neoplasm of skin  
 Malignant neoplasm of lip, oral cavity and  
 pharynx  
 Other diseases of musculoskeletal system and  
 connective tissue  
 Non infective enteritis and colitis  
 Gastric ulcer  
 Other psychoses  
 Multiple sclerosis

Duodenal ulcer and digestive ulcer of  
 unspecified site  
 Malignant neoplasm of other respiratory and  
 intrathoracic organs (thyroid gland, heart  
 and mediastinum)  
 Congenital anomalies of heart  
 Mental retardation (oligophrenia)  
 Nutritional and metabolic disorders and  
 disorders involving the immune mechanism  
 Misadventures to patients during medical care

*Group 2*  
 Atherosclerotic cardiosclerosis without  
 hypertensive disease  
 Atherosclerotic cardiosclerosis with  
 hypertensive disease  
 Suicide and self-inflicted injury  
 Other transport accidents  
 Accidental drowning and submersion  
 Accidental inhalation and ingestion causing  
 obstruction of respiratory tract, foreign body  
 entering other orifices  
 Septicaemia  
 Other inflammatory diseases of the central  
 nervous system

*Group 3*  
 Cerebrovascular disorders with hypertensive  
 disease  
 Malignant neoplasm of stomach  
 Chronic rheumatic heart disease  
 Respiratory tuberculosis  
 Malignant neoplasm of oesophagus  
 Benign and unspecified neoplasms  
 Unspecified disorders of pericardium, mitral  
 and aortic valves



Pneumococcal pneumonia  
 Other complications of pregnancy, childbirth  
 and the puerperium  
 Diseases of other endocrinous glands  
 Viral hepatitis  
 Haemorrhage in pregnancy and childbirth  
 Ectopic pregnancy  
 Echinococcosis  
 Other tuberculosis excluding late effects  
 Tuberculosis of bones and joints  
 Accident caused by firearm missile

#### *Group 4*

Other heart diseases  
 Accidental poisoning by alcohol  
 Homicide and injury purposely inflicted by  
 other persons, including legal execution  
 Other cirrhoses of liver  
 Other accidents  
 Injury undetermined whether accidentally or  
 purposely inflicted  
 Other accidental poisoning  
 Accident caused by fire  
 Chronic alcoholism  
 Meningitis, excluding infectious and parasitic  
 meningites  
 Alcoholic cirrhosis of liver  
 Erysipelas  
 Diphtheria

#### *Group 5*

Pulmonary congestion and hypostasis, and  
 postinflammatory pulmonary fibrosis  
 Malignant neoplasm of cervix uteri  
 Acute myocardial infarction with hypertensive  
 disease  
 Epilepsy  
 Intestinal obstruction without mention of  
 hernia  
 Bronchiectasis and other obstructive  
 pulmonary diseases  
 Abortion started out of the hospital and  
 unspecified  
 Appendicitis  
 Hypertensive renal disease  
 Other diseases of respiratory system  
 Other diseases of circulatory system  
 Accident caused by electric current  
 Other diseases of blood and blood-forming  
 organs  
 Osteomyelitis and periostitis  
 Viral encephalitis  
 Hypertensive heart and renal disease  
 Pneumoconiosis and other lung diseases due

to external agents  
 Other viral diseases  
 Tuberculosis of nervous system  
 Tetanus  
 Syphilis (all forms)  
 Tuberculosis of intestine, peritoneum and  
 mesenteric glands

#### *Group 6*

Malignant neoplasm of other and unspecified  
 female genital organs  
 Malignant neoplasm of rectum, rectosigmoid  
 junction and anus  
 Calculus of urinary tract  
 Other congenital anomalies  
 Bacterial foodborne intoxications excluding  
 salmonella infections

#### *Group 7*

Chronic bronchitis and emphysema  
 Hernia of abdominal cavity  
 Influenza  
 Viral pneumonia

#### *Group 8*

Malignant neoplasm of other digestive organs  
 Other malignant neoplasms of lymphoid and  
 histiocytic tissue

#### *Group 9*

Malignant neoplasm of uterus  
 Other and unspecified hypertensive disease  
 Late effects of tuberculosis  
 Drug dependence  
 Other infectious diseases

#### *Group 10*

Hypertensive heart disease  
 Other diseases of the nervous system and  
 sense organs  
 Other diseases of digestive system  
 Other diseases of liver and biliary tract  
 Acute nephritis and glomerulonephritis  
 Acute intestinal infections due to unspecified  
 micro-organisms and ill-defined, including  
 toxical dyspepsia

#### *Group 11*

Other acute pneumonias  
 Other nephrites and nephroses, nephrotic  
 syndrome  
 Empyema, lung or mediastinal abscess  
 Meningococcal infection  
 Shigellosis (dysentery)

*Group 12*

Motor vehicle traffic accident  
Diseases of the skin and subcutaneous tissue

*Group 13*

Gallstone diseases and cholecystitis  
Septicaemia in childbirth and the puerperium

*Group 14*

Acute rheumatic fever  
Anaemia  
Gastritis and duodenitis

*Group 15*

Phlebitis and thrombophlebitis, venous  
embolism and thrombosis  
Toxaemia in pregnancy

*Group 16*

Malignant neoplasm of small intestine,  
including duodenum  
Legally induced abortion

*Group 17*

Other diseases of urinary system  
Other parasitic diseases

*Group 18*

Malignant neoplasm of larynx

*Group 19*

Other diseases of genital organs  
Other congenital anomalies of central nervous  
system

*Group 20*

Acute respiratory infections  
Alcoholic psychosis

*Group 21*

Inflammation of middle ear and mastoiditis

<sup>1</sup>Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Italy, Japan, Netherlands, New Zealand, Norway, Portugal, Spain, Sweden, Switzerland, United States of America, and the United Kingdom.

## REFERENCES

- Anderson, B. A. and B. D. Silver (1990). Trends in mortality of the Soviet population. *Soviet Economy* (Maryland), vol. 6, No. 3, pp. 191-251.
- \_\_\_\_\_. (1994). A comparison of Soviet mortality in the working ages: 1959-1988. In *Demographic trends and patterns in the Soviet Union before 1991*, W. Lutz, S. Scherbov and A. Volkov, eds. London: IIASA/Routledge, pp. 279-293.
- Andreev, E. M. (1979). Life expectancy in the USSR: differential analysis. In *Life expectancy: analysis and simulation*, E. Andreev, A. Vishnevsky, eds. Moscow, pp. 7-31.
- \_\_\_\_\_. (1982). Method of components in cause of death analysis. *Statistics herald* (Moscow), No. 9, pp. 42-47 (In Russian).
- \_\_\_\_\_. (1988). Social determination of mortality, demographic policy and expectation of life projections. In *Methodology of demographic projection*, A. Volkov, ed. Moscow, pp. 118-135 (In Russian).
- \_\_\_\_\_. (1990). Expectation of life and causes of death in the USSR. In *Demographic processes in the USSR*, A. Volkov, ed. Moscow, pp. 90-115 (In Russian).
- \_\_\_\_\_. (1994). Life expectancy and causes of death in the USSR. In *Demographic trends and patterns in the Soviet Union before 1991*, W. Lutz, S. Scherbov and A. Volkov, eds. London: IIASA/Routledge, pp. 279-293.
- \_\_\_\_\_. (1995a). Infant mortality in Russia. *Questions of statistics* (Moscow), No. 5, pp. 66-71 (In Russian).
- \_\_\_\_\_. (1995b). The last mortality trend in Russia. Paper presented at the 3rd European Population Conference, Milan, 4-8 September.
- \_\_\_\_\_, L. E. Darsky and T. L. Kharkova (1993). *Population of the Soviet Union: 1922-1991*. Moscow: Nauka (In Russian).
- \_\_\_\_\_. (1997). Demographic history of Russia: 1927-1959. Moscow (In press) (In Russian).
- Andreev, E. M. and V. M. Dobrovol'skaya. (1993). Social differentiation of mortality in Russia. *Health care in the Russian Federation* (Moscow), No. 9, pp. 18-21 (In Russian).
- Andreev, E., S. Scherbov, and F. Willekens (1995). Sources of information on the population of Russia. Demographic reports. Groningen.
- Avdeev, A., A. Blum, S. Zakharov and E. Andreev (1997). Réaction d'une population hétérogène à une perturbation. un modèle d'interprétation des évolutions de mortalité en Russie. *Population*, No. 1, pp. 7-44.
- Biryukov, V. A., (1984). *Statistical analysis of mortality of the urban population of the USSR*. Dissertation. Candidate degree in economics. Moscow (In Russian)
- Blum, Alain (1994). *Naître, vivre et mourir en URSS. 1917-1991*. Paris: Plon.
- \_\_\_\_\_, and Alain Monnier. (1989). Recent mortality trends in the U.S.S.R.: new evidence. *Population studies*, No. 43, pp. 211-241.
- Central Statistical Board of the USSR (1973). *The results of the 1970 All-Union census of population*, vol. VIII, Part 1. Moscow (In Russian).
- \_\_\_\_\_. (1981). *The results of the 1979 All-Union census of population*, vol. IX, Part 1. Moscow (In Russian).
- Coale, Ansley, and Paul Demeny (1966). *Regional model life table and stable population*. Princeton: Princeton University Press.
- Davydovsky, I. V. (1962). *Problems of causality in medicine (etiology)*. Moscow (In Russian).
- Ellman, M. (1994). The increase in death and disease under "katastroika". *Cambridge Journal of Economics*, vol. 18, No. 2, pp. 329-355.
- Goskomstat of Russia (1995). *Demographic yearbook of Russia. Statistical handbook*. Moscow.
- Dublin, L., A. Lotka and M. Spiegelman (1949). *Length of life*. New York: Ronald Press.
- Feshbach Murray, and Alfred Friendly. (1992). *Ecocide in the USSR: health and nature under siege*. New York: Harper Collins.
- Keyfitz, N., and W. Fliger (1968). *World population. Analysis of vital data*. Chicago: University of Chicago Press.
- Korchak-Chepurkovskiy, Yuriy A. (1987). The influence of mortality in different ages on expectation of life. In *Soviet demography for 70 years*, T. V. Ryabushkin, ed. Moscow, pp. 263-280 (In Russian).
- McKeown, Thomas, R. G. Record, and E. D. Turner (1975). An interpretation of the decline of mortality in England and Wales during the Twentieth Century. *Population Studies*, vol. 29, No. 3, pp. 391-422.
- Mesle, F., V. Shkolnikov and J. Vallin (1994). Brusque montée des morts violentes en Russie. *Population*, 49 année, No. 3, pp. 780-789.
- Mille, F., V. Shkolnikov, and J. Vallin (1996). *Recent trends in mortality by cause in Russia, 1965-1994*. Moscow-Paris (In Russian and French).
- Milligan, G. W. and L. Sokol (1980). A two stage clustering algorithm with robust recovery characteristics. *Educational and psychological measurement*, vol. 40, pp. 755-759.
- Morbidity and Mortality of the population of the USSR (1974). *Statistical handbook*. Moscow (In Russian).
- Novosel'sky, S. A. and V. V. Paevsky (1930). *Mortality and expectation of life in the USSR, 1926-1927 (Life tables)*. Moscow-Leningrad (In Russian).
- Okolski, M. (1993). Health and Mortality. Paper presented at the European Population Conference.
- Public health in the Russian Federation (1995). *Statistical handbook*. Moscow (In Russian).
- Public health in the USSR (1966). *Statistical handbook*. Moscow (In Russian).
- Shkolnikov, Vladimir, France Mesle and Jacque Vallin, (1995). La crise sanitaire en Russie. *Population*, Nos. 4-5, pp. 907-944 and 945-982.
- Shkolnikov, V. M., and S. A. Vasin, (1994). Spatial differences in life expectancy in European Russia. In *Demographic trends and patterns in the Soviet Union before 1991*, W. Lutz, S. Scherbov and A. Volkov, eds. London: IIASA/Routledge, pp. 57-69.
- Sifman, R. I. (1979). On the question of causes of child mortality decline in the days of the Great Patriotic War. In *Expectation of life: analysis and simulation*, E. Andreev and A. Vishnevsky, eds. Moscow, pp. 50-60.
- United Nations (1973). *Determinants and consequences of population trends*. New York: United Nations. Catalog no. 53.XIII.3.
- \_\_\_\_\_. (1982). *Levels and trends of mortality since 1950*. New York: United Nations. Sales no. E/F/S.81.XIII.3.
- United Nations (various years). *Demographic yearbook. 1949-1994*. New York: United Nations.
- Verbitskaya, O. M. (1997). Demographic consequences of radioactive catastrophe. In *Population: modern condition and prospects of develop of scientific knowledge*. Moscow, pp. 79-88.

Willekens, F., and S. Scherbov (1991). Age-period-cohort (APC) analysis of mortality with applications to Soviet data, WP 91 42. Laxenburg, Austria: International Institute for Applied Systems Analysis  
Willekens, F. and S. Scherbov. (1992). Analysis of mortality data from the former USSR: age-period-cohort analysis.

*World health statistics quarterly* (Geneva), vol. 45, No. 1, pp. 29-49.  
Zakharov, S. (1997). The demographic History of the USSR and Russia in a mirror of the history of generation. *Population and Society* (Moscow), No. 17 (In Russian).

## XII. THE WIDENING DIFFERENTIALS IN ADULT MORTALITY BY SOCIO-ECONOMIC STATUS AND THEIR CAUSES

*Tapani Valkonen\**

With the exception of the transitional economies of central and eastern Europe, developed countries have reported favourable overall trends in mortality since the 1970s. However, results from several countries suggest that not all segments of the population have benefitted equally from the decrease in premature mortality (Whitehead and Diderichsen, 1997, pp. 50-52). According to these results socioeconomic differentials in mortality have increased in contrast to the health policy goals set by the WHO and national governments.

The main purpose of this paper is to review and assess research results describing changes in socioeconomic mortality differentials since the 1970s. The paper focuses on adult mortality in developed countries, excluding transitional economies. Before assessing the existing evidence, it is necessary to discuss the methods and data sources used in analyzing changes in socioeconomic mortality differentials.

### A. DATA SOURCES AND THE MEASUREMENT OF SOCIOECONOMIC MORTALITY DIFFERENTIALS

Persons in lower socioeconomic positions die on average at a younger age than those in higher positions. This inequality in the face of death has been observed in almost all studies in different populations and using different indicators of socioeconomic position, such as social or occupational class, educational attainment, income and housing characteristics (see e.g. Antonovsky, 1967; Feinstein, 1993; Mackenbach and others, 1997). Although

there is no doubt about the existence of socioeconomic differentials in mortality, it is very difficult to produce exact measurements of these differentials so that valid comparisons can be made between countries and periods. This is because the results vary depending on several factors, such as the source and coverage of data, the socioeconomic indicators used and the way in which mortality and the size of mortality differentials is measured. Some of these problems have been discussed in great detail in a report prepared by Kunst and Mackenbach (1994a) for the WHO Regional Office of Europe. This section of the paper deals with the measurement issues that are most relevant in assessing the validity of results on changes in socioeconomic differences.

#### 1. Sources of data

Figures on mortality by socioeconomic category are not normally presented in regular statistics. The data for England and Wales published by the Registrar General since 1851 in the form of so called Decennial Supplements provide the only regular long-term series of mortality statistics by occupation and social class (Office of Population Censuses and Surveys (OPCS), 1978; Fox, 1979). The death rates given in these statistics are based on the independent classification of 'last occupation' recorded on death certificates and census forms. Deaths are usually grouped for a period of three or five years around the census. For example, in the latest Decennial Supplement the death rates by social class for England and Wales are based on two sources: deaths recorded in 1991-1993 by age and occupation-based social class of the deceased person provide the numerators, and a 10 per cent sample of the population recorded in the 1991 census the denominators by age and social class (Drever and Bunting, 1997).

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The "cross-sectional unlinked method" used in the Decennial Supplements has also been used elsewhere (Mielck and do Rosário Giraldes, 1993), but for many countries it is not an option because of the absence of valid socioeconomic information on the deceased in death certificates. Another serious limitation is that the results are likely to be biased because the information on the occupation of deceased and persons in the risk population comes from different sources. Even though the same question is asked in the census and on the death registration form, the accuracy of the information differs. There is evidence that this bias (called numerator/denominator bias) can be quite substantial (Fox, 1979; Kunst, 1996). Because of the increased bias, the results presented in the Registrar General's Decennial Supplement for mortality around the 1981 census (OPCS, 1986), were considered less reliable (Thatcher, 1986). The Decennial Supplement for mortality around the 1991 census examines some possible sources of bias, such as definitional issues, classification changes, and changing age structure, but does not discuss the possible effects of the numerator/denominator bias (Drever, 1997).

To avoid the numerator/denominator bias associated with the use of unlinked records, other methods are employed. These are usually based on linking death records to population records of individuals. As early as 1865, Chapin (see Antonovsky, 1967) compared death rates among taxpayers and non-taxpayers in Providence, Rhode Island using linked census, tax and death records. The method of linking death records back to census records was applied on a large scale in the Matched Records Study on differential mortality in the United States by Kitagawa and Hauser (1973). Some 340,000 deaths that occurred during the period from May to August 1960 were matched with the 1960 census records in order to obtain the social and economic characteristics of deceased as reported in the 1960 census. Death rates were calculated by occupation, level of education and income.

Another version of the linked record method is used in longitudinal studies, which have the same design as prospective epidemiological

studies. An early example of this approach is the study carried out by the National Institute of Statistics and Economic Studies (INSEE), in which a sample of almost 800 000 persons from the French census of 1954 was followed up for mortality until 1971 (Desplanques, 1976).

Another example of a longitudinal study based on linked records is the Longitudinal Study for England and Wales, based on a one per cent sample (about 500,000) from the census of 1971. Data on deaths as well as on some other demographic events occurring in this population have since been linked to the sample records. The results up to the end of 1992 are presented in the latest Decennial Supplement (Harding and others, 1997).

Studies based on linked records provide a more solid basis for analyzing socioeconomic differences in mortality than cross-sectional unlinked studies, but they still have their limitations. Manual linking of census and death records is time-consuming and very expensive. The costs involved mean that it is impossible to study large populations, and samples have to be used. Although the samples used in the three studies described above are large enough for a statistically satisfactory analysis of all-cause mortality, a detailed analysis by cause of death and in different population sub-groups is not possible. Moreover, the quality of data is not always very good because of problems in linking census and death records. For example, 23 per cent of the deceased included in the Kitagawa and Hauser study (1973, p. 187) could not be linked to the census records.

Longitudinal linked records studies are the most feasible option in countries that have a system of personal identity numbers, allowing for computerized linkage of records. Mortality studies using this method have indeed been carried out in the Nordic countries (Denmark, Finland, Norway and Sweden) (e.g. Ingerslev and others, 1994; Valkonen and others, 1993; Borgan, 1996; Vågerö and Lundberg, 1995). The whole population can easily be followed up for mortality in these countries, and practically all death records can be linked to census records. In Finland, for example, only 0.3 per

cent of all deaths in 1986-1990 could not be linked to the 1985 census records (Valkonen and others, 1993). An additional advantage of the system of personal identity numbers is that information from other registers, such as tax, hospital and pension registers, can also be used.

Different approaches have been used in countries in which, for legislative or other reasons, studies based on the linkage of records on individuals are not possible. One example is provided by ecological research where death rates in particular geographical areas are correlated with socio-economic characteristics of those areas (e.g. Wilkins and others, 1989; Quine and others, 1995). This method is inexpensive and generally feasible. However, the results of ecological studies are not comparable with those of studies in which data on individuals are used. The aggregate-level relationship between the average socio-economic status and mortality of the population in specific areas may be different from the individual-level association between the same variables. Changes in areal differentials in mortality may be influenced by changes in the socioeconomic composition of areas such as an increasing concentration of retired or disabled persons. Studies based on data on areas will not be discussed in this review.

## *2. Indicators of socioeconomic position*

The most common indicators of socioeconomic position in past research have been occupation-based social class or socioeconomic status, level of education and household or personal income. None of these indicators is a perfect indicator and their limitations must be taken into account in the assessment of the research results.

### *Occupation-based social class*

Occupation and groups of occupations were frequently used in mortality studies as early as the 19th century (Westergaard, 1901). British studies are still mostly based on the so-called Registrar General's classification of social classes, which has been used with some modifications since 1921. Occupation-based socioeconomic status or social class has also been

the most common indicator in other European countries, but not in North America. More or less valid results on mortality by occupation-based socioeconomic classifications are available for most European countries (Valkonen, 1987; Mielck and do Rosário Giraldes, 1993; Mackenbach and others, 1997; Kunst, 1997).

A major weakness of occupation-based classifications is that only the economically active population can usually be classified, whereas housewives, the unemployed, retired and persons on disability pension cannot be assigned to classes. This means that results for women and young and old age groups will often be missing altogether. In fact most of the published results on class differentials in mortality cover only a small part of all deaths - those among economically active middle-aged men.

Even for the working-age population results based on occupation-based class are often biased by the so-called healthy worker effect: in each class economically active persons have lower mortality than economically inactive persons. This bias can be avoided if information is available on the former occupation of retired, unemployed and disabled persons and on the occupation of the head of household for dependent persons. This is the case in Finland and the effects of the bias can be illustrated using Finnish data (Valkonen and Martikainen, 1997).

Table 54 shows the age-standardized death rates for men and women aged 35-64 by occupation-based social class on the basis of death records for 1981-1985 linked to the records of the 1980 census. For the purpose of calculating death rates by social class for the economically active population, only those who were employed in 1980 were assigned to social classes. All economically inactive persons and persons who were unemployed were excluded. For the purpose of calculating the death rates for the whole age groups, retired, disabled and unemployed persons were classified on the basis of their former occupation obtained by record linkage from the earlier censuses as far back as 1970. Non-working family members (mainly housewives) were classified on the basis of the class of the head

of the household. The column for "all" thus gives the mortality differences for the whole population, and not only for those who were employed in 1980.

The differences in mortality for the economically active and the whole population are clear. Mortality in each social class is lower for economically active persons because the mortality of retired and other non-employed persons is much higher than that of employed persons. More importantly, the absolute and relative differences between classes are clearly smaller in the economically active than in the whole population, because the propor-

tion of economically inactive persons is higher in the manual than in the non-manual classes.

Thus, findings on socio-economic differences by occupation-based social class for the economically active population only, underestimate the magnitude of socioeconomic mortality differences. The magnitude of underestimation is likely to be different for different periods and countries. The amount of bias diminishes with longer follow-up times, mortality differentials increase in proportion to the length of the follow-up time. The bias among economically active women is so large

TABLE 54. AGE-STANDARDIZED MORTALITY (PER 100,000) AND RELATIVE AGE-STANDARDIZED MORTALITY (UPPER WHITE-COLLAR EMPLOYEES=1.00) BY SOCIAL CLASS FOR THE ECONOMICALLY ACTIVE POPULATION IN 1980 AND FOR THE WHOLE POPULATION, AND THE DISTRIBUTIONS OF PERSON-YEARS BY SOCIAL CLASS, MEN AND WOMEN AGED 35-64 IN FINLAND, 1981-1985

Social class	Age-standardised mortality		Relative age-standardised mortality		Distribution of person-years (per cent)	
	Economically active	All	Economically active	All	Economically active	All
<b>Men</b>						
Upper white-collar employees	595	768	1.00	1.00	15.3	13.0
Lower white-collar employees	709	1 062	1.19	1.38	17.0	16.3
Skilled workers	781	1 229	1.31	1.60	40.1	39.8
Unskilled workers	944	1 744	1.59	2.27	7.1	9.8
Farmers	702	1 068	1.18	1.39	13.1	13.8
Other self-employed persons	843	898	1.42	1.17	7.4	6.2
Others	391	2 724	0.66	3.55	0.0	1.2
All	749	1 176	1.26	1.53	100.0	100.0
person-years in thousands					2 990	3 839
<b>Women</b>						
Upper white-collar employees	242	344	1.00	1.00	9.0	9.3
Lower white-collar employees	267	393	1.10	1.14	38.4	32.5
Skilled workers	280	444	1.16	1.29	24.6	27.0
Unskilled workers	268	490	1.11	1.42	12.0	12.8
Farmers	276	431	1.14	1.25	11.7	13.1
Other self-employed persons	300	310	1.24	0.90	4.3	4.1
Others	256	1 148	1.06	3.34	0.0	1.1
All	273	428	1.13	1.24	100.0	100.0
person-years in thousands					2 768	4 193

Source: Tapani Valkonen and Pekka Martikainen, "Problems of comparability resulting from the exclusion of economically inactive persons in studies on social class differences in mortality", in *Morbidity and Mortality Data: Problems of Comparability, Proceedings of the EAPS and Hacettepe Institute of Population Studies Workshop*, Attila Hancioglu and Guillaume Wunsch, eds. (Ankara, Hacettepe Institute of Population Studies, Population No. IPS-HU. 97-01, 1997).



and depends so strongly on the length of follow-up that results based on economically active women seem almost useless (Valkonen and Martikainen, 1997).

Another problem with the use of occupation-based socioeconomic classifications is that these classifications in different countries are not similar and comparable. A recent cross-national study (Mackenbach and others, 1997; Kunst, 1997) attempted to avoid this problem by applying the so-called EGP social class scheme (Erikson and Goldthorpe, 1992). Another strategy has been to use crude classifications, for example simple distinctions between manual and non-manual occupations. For the analysis of trends in inequalities, the main problem is presented by changes in the socioeconomic classifications. For example, the distinction between skilled and unskilled workers used in table 54 is no longer applicable in Finland because it has been omitted from the socioeconomic classification used by Statistics Finland. Differences in the level of detail in classifications are also a problem in comparisons: the larger the number of groups, the more homogenous they tend to be and the greater the differences.

#### *Level of education*

Kitagawa and Hauser (1973) considered education the most satisfactory of the several indexes of socioeconomic status available in their study on differential mortality in the United States. In later studies for the United States, level of education has been virtually the only indicator used. This measure of socioeconomic position does indeed have some important advantages. First of all, level of education can be defined on the same grounds for both the economically active and the economically inactive population. Secondly, level of education does not usually change after youth. It is therefore not influenced by deterioration of health, whereas a person's occupation and income level may change. Thirdly, information on the length of education is often more comparable than information on occupational class both internationally and over time.

Unfortunately, the information on level of education is, in general, not as useful in Europe as in the United States. In the study by Kitagawa and Hauser and in later studies in the United States, educational attainment is measured in terms of actual number of years of education completed, and there is much variation in this variable in the United States. In many European countries most of the population used to receive the same level of education as prescribed by compulsory schooling. The distribution of education is therefore quite skewed, particularly in older age groups, and meaningful analyses of differences in mortality are difficult because of insufficient variation in the level of education. However, since the average level of education is rising rapidly, level of education is becoming more and more useful.

#### *Income and living standard*

Income seems to be a good indicator of socioeconomic position for mortality studies and it has been used in some studies. However, as Kitagawa and Hauser (1973) point out, it does have one major handicap in that the association between income and mortality is likely to be influenced by the confounding effect of health status: poor health influences both income and risk of death. Results on the association between income and mortality will thus depend on such factors as the level of pensions or allowances for sick, permanently disabled and retired people.

In the absence of direct information on income, proxy measures such as house and car ownership have been used as indicators of economic resources (Goldblatt, 1990; Smith and Harding, 1997). These indicators have revealed marked mortality differences, but again it is difficult to assess the contribution of confounding by poor health status.

## *2. Measurement problems*

Even if valid data are available, it is not a straightforward task to tell whether and to what extent the socioeconomic mortality gap has widened or narrowed during a given pe-

riod. The difficulties stem from three main sources:

- The size of the differentials can be measured either in absolute or in relative terms. As the level of mortality has been declining in general, the sizes of changes in absolute differences are necessarily different from those in relative differences. Even the direction of changes may be different.
- Trends in mortality may be different for men and women and for different age groups, and the conclusions vary depending on the groups selected for the study and on the way in which the groups are weighted in the calculations.
- The distribution of the population into socioeconomic categories changes. This makes the comparison of results for different periods problematic.

#### *Absolute versus relative differences*

These difficulties are illustrated here by data on mortality by level of education for Finland during 1972-1995. The data are based on census records for the years 1970, 1975, 1980, 1985 and 1990, which have been linked with the death records for the five-year period following the census (Valkonen and others, 1990; Valkonen and others, 1993; Valkonen and Jalovaara, 1996). The information on level of education is based on the national register on degrees and certificates. Three levels of education are distinguished: primary education (9 years of less), secondary education (10-12 years), and tertiary education (at least 13 years).

Figure 87 shows the age-standardized death rates for males from 1972 to 1995. In the light of this figure it seems that the difference between the high and low educational categories is nearly the same at the beginning of the 1970s as at the beginning of the 1990s and was slightly smaller in the early 1980s. If, however, we use a logarithmic scale (figure 88) and thus examine trends in relative instead of absolute differences, the picture is different. The relative difference between men with ter-

tiary education and the other categories has increased.

The data shown illustrate one key problem in the measurement of socioeconomic inequalities: should we measure absolute differences or relative differences in mortality? From a public health perspective the absolute decrease in mortality differences is more important than the decrease in relative differences because the aim of health policies is to prevent as many premature deaths as possible.

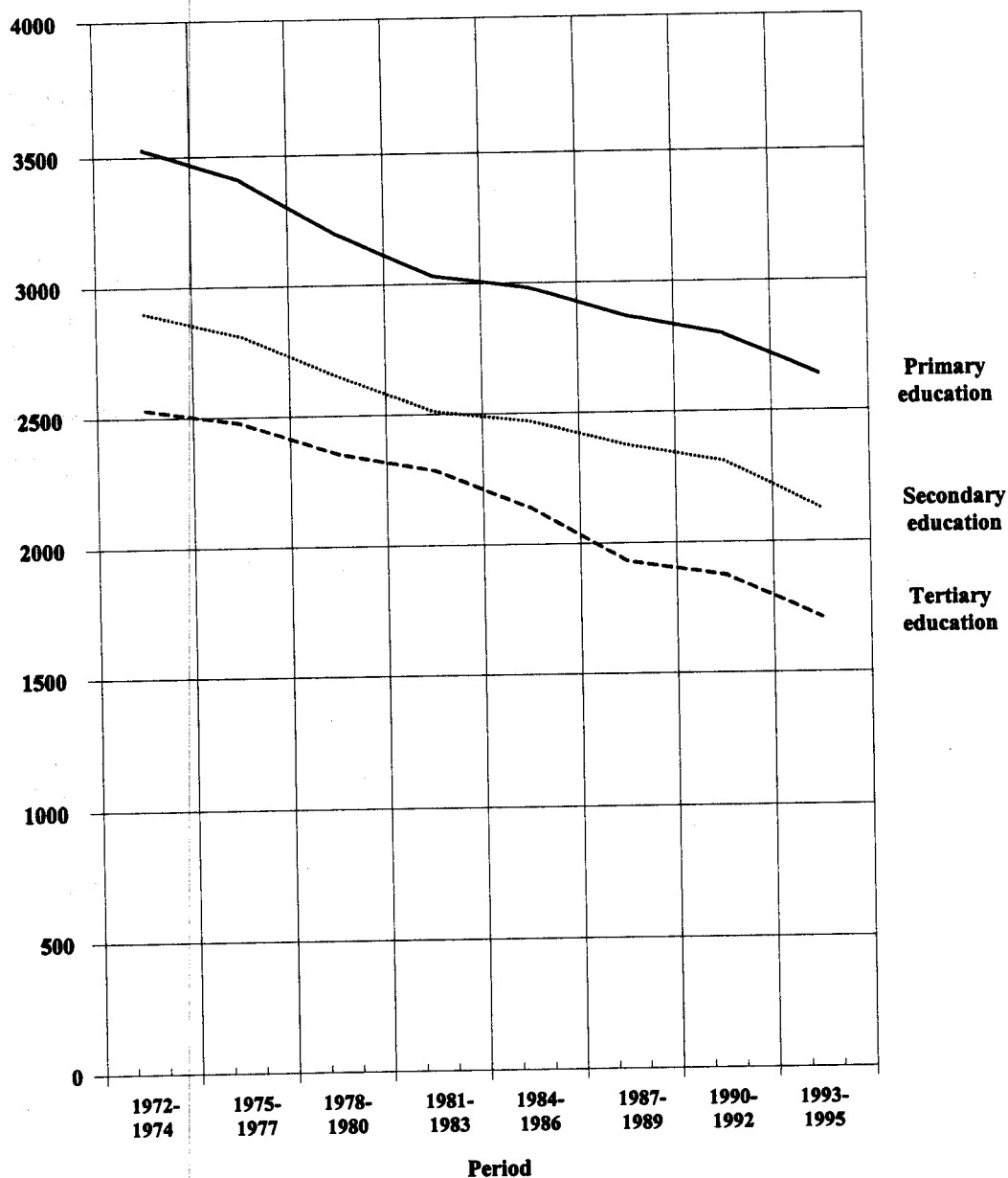
On the other hand, epidemiologists and other researchers usually base their analyses on relative differences, and conclusions on widening mortality differentials often concern relative differences, even though this is not explicitly stated. Recently, more attention has been paid to the significance of absolute differences (Mackenbach and others, 1997; Kunst, 1997).

#### *Variation according to age and sex*

The data shown in figures 87 and 88 have covered the male population aged 35 and over. These results are dominated by trends in the old age groups, where the number of deaths is highest. The trends are not necessarily the same in younger age groups. Figure 89 shows trends in age-standardized death rates by level of education among men aged 35-49 in Finland. In this age group trends are clearly different from those among all men aged 35 and over, because mortality has actually increased among men with basic education. The rapid widening of relative differentials can be seen from the standardized mortality ratios (figure 90). In 1972-1974 mortality in the low education group was 2.2 times the mortality in the high education group but in 1993-1995 this ratio was 3.4. Figures 89 and 90 also show that the trends are all but uniform during the 25-year period studied.

Table 55 illustrates how conclusions on the extent and direction of changes in educational differentials may vary depending on the age group and on the type of difference examined (absolute versus relative). The relative difference in age-standardized mortality between men with primary and tertiary education has clearly increased in all age groups from the

**Figure 87. Age-standardized mortality (per 100,000, European standard population)  
by level of education, men aged 35 and over in Finland, 1972-1995**

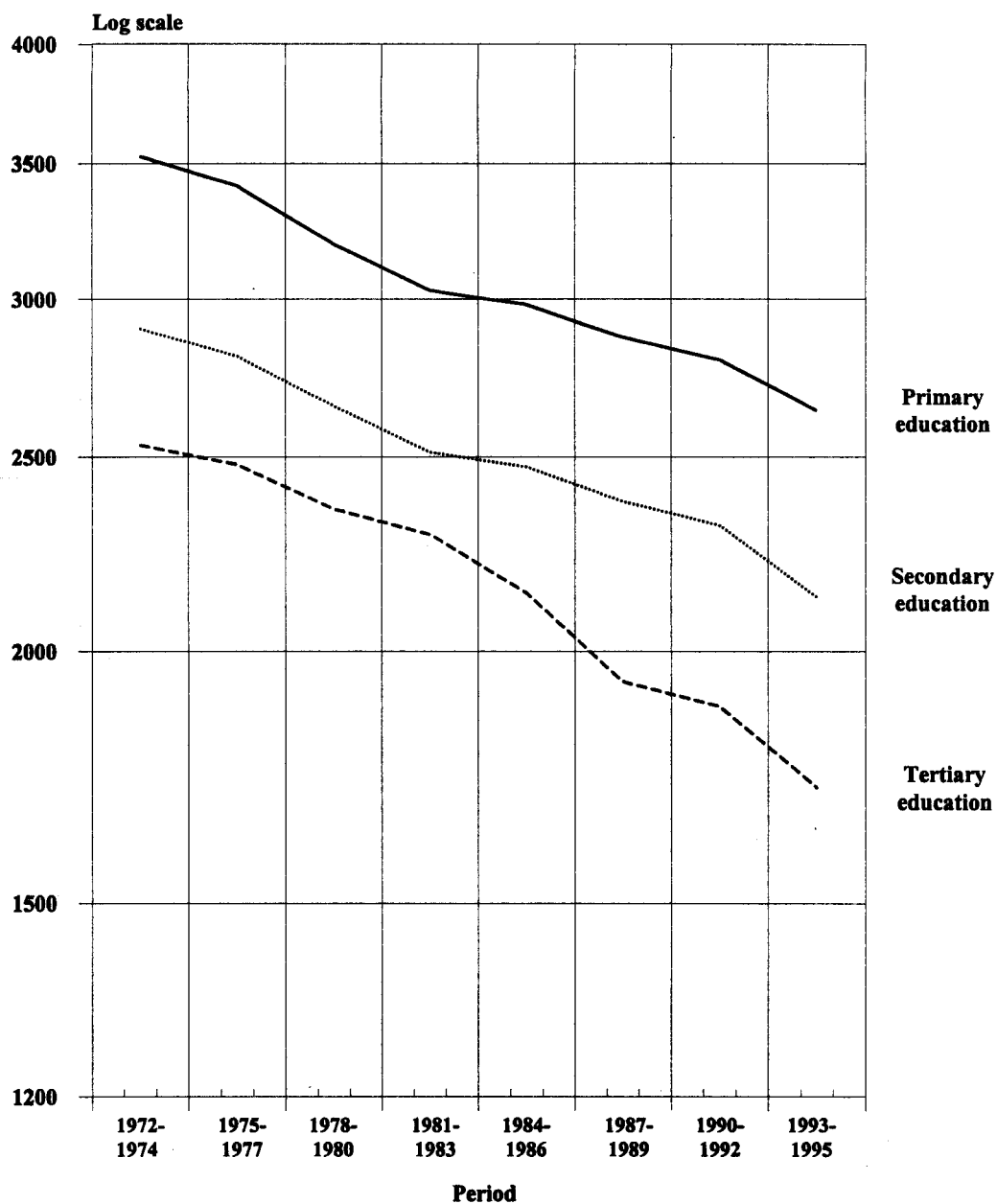


Source: Unpublished data from the Population Research Unit, University of Helsinki.

1970s to the 1990s in Finland, but the magnitude of the increase varies. The increase is greater in younger than older age groups. On the other hand, the absolute difference has increased in two age groups and decreased in two age groups resulting to a slight decrease in the total over 35 population.

Because of data shortages many studies on socioeconomic mortality differences do not cover all age groups. For example, most studies reviewed in this paper are limited to the working-age population. Results on limited age ranges cannot be generalized to the whole population.

**Figure 88. Age-standardized mortality (per 100,000, European standard population) by level of education, men aged 35 and over in Finland, 1972-1995 (log scale)**



Source: Unpublished data from the Population Research Unit, University of Helsinki.

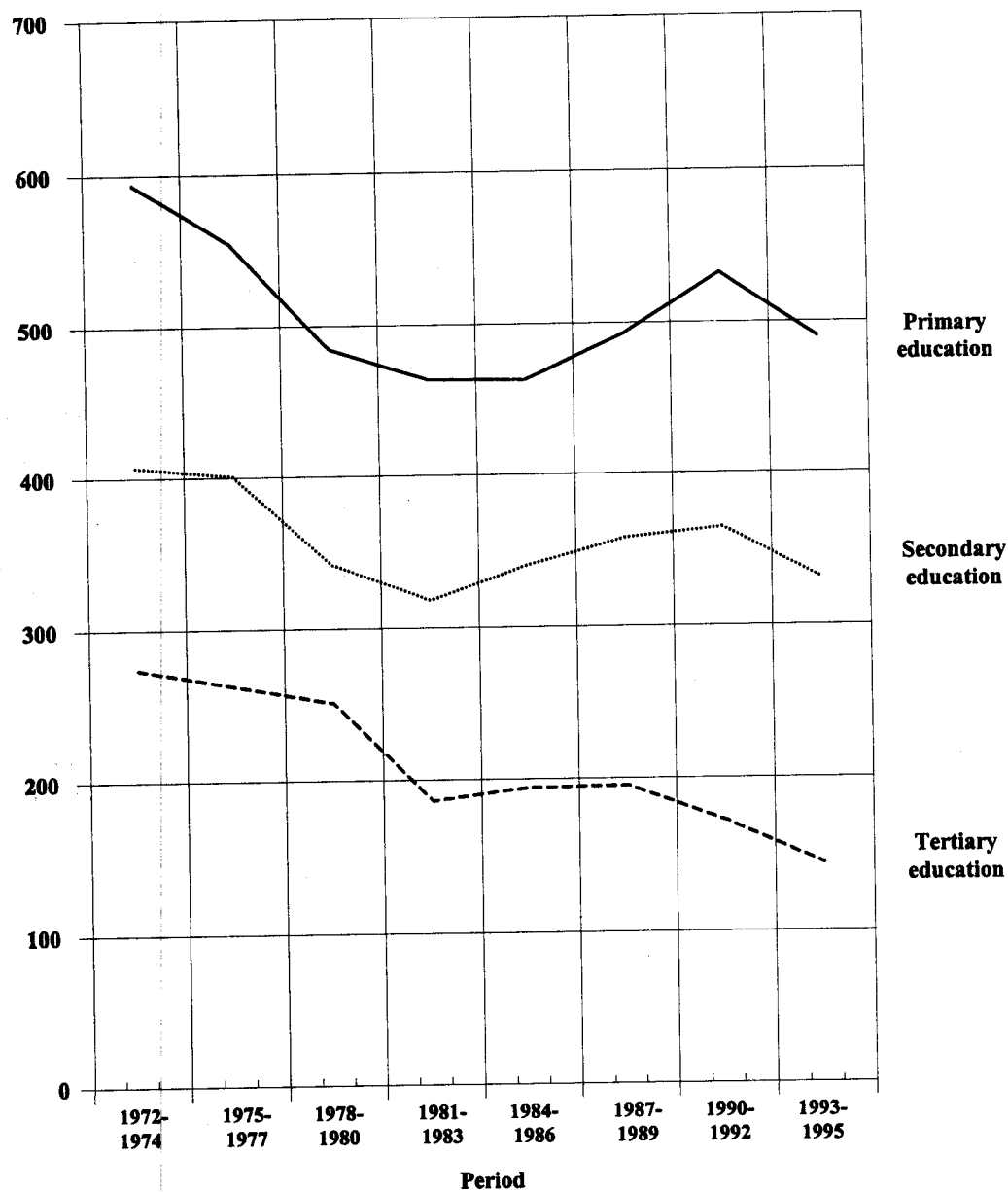
#### *Changes in socio-economic distributions*

There is yet another problem in the measurement of trends in inequalities: the influence of changes in how the population is divided into socioeconomic categories. It is intuitively clear that a difference of a given size in mor-

tality between two large population groups is more important than a difference of the same size between a large group and a small group.

Several summary measures that take into account the distribution of the population into socio-economic categories have been developed for the measurement of inequalities in

**Figure 89. Age-standardized mortality (per 100,000, European standardized population)  
by level of education, men aged 35-49 in Finland, 1972-1995**

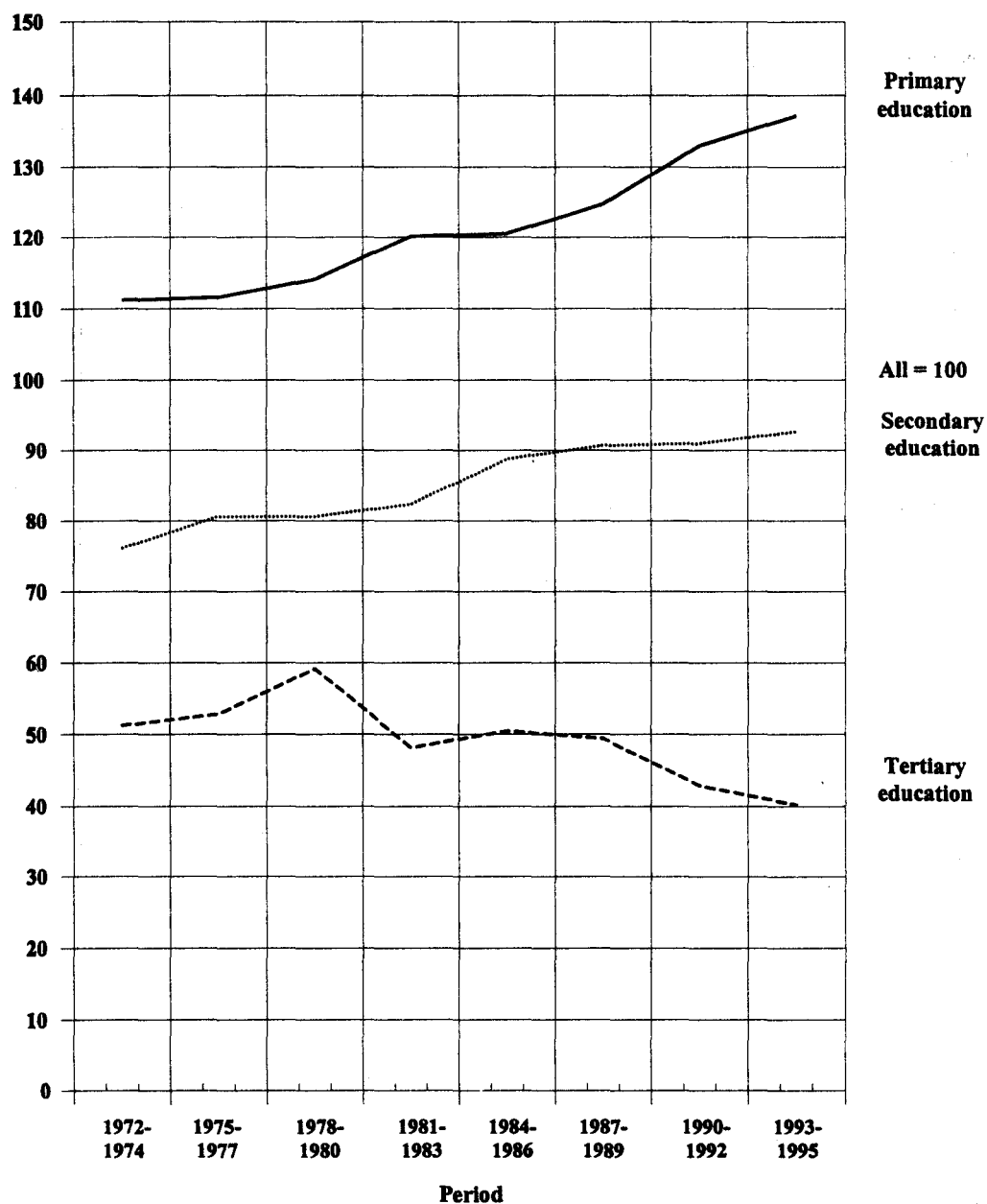


Source: Unpublished data from the Population Research Unit, University of Helsinki.

mortality. These include the index of dissimilarity and regression-based measures (see e.g. Wagstaff and others, 1991; Kunst and Mackenbach, 1994a; Mackenbach and Kunst, 1997).

It is not possible to discuss these measures in any detail here, although the measure chosen may have a major impact on the conclusions drawn. Kunst (1997, pp. 48-56) applied twelve different measures to examine data on

**Figure 90. Standardized mortality ratios by level of education, men aged 35-49 in Finland, 1972-1995**



Source: Unpublished data from the Population Research Unit, University of Helsinki.

educational differences in mortality in Finland from 1971-1975 to 1986-1990. Three of the indicators showed an increase in inequality and one a decrease in inequality for both sexes. The proportion of persons in the two

higher educational categories increased from 1971 to 1990 in Finland and the educational distributions became less skewed. When this takes place most measures that take into account changes in distribution would show an

TABLE 55. ABSOLUTE DIFFERENCES AND RATE RATIOS (PER 100,000) IN AGE-STANDARDIZED MORTALITY BY PRIMARY AND TERTIARY EDUCATION FOR MEN IN SELECTED AGE-GROUPS, FINLAND, 1972-1974 AND 1993-1995

<i>Difference in mortality (per 100,000) between primary and tertiary</i>	<i>Age</i>				
	<i>35-49</i>	<i>50-64</i>	<i>65-74</i>	<i>75+</i>	<i>All</i>
1972-1974	320	817	1 490	3 165	994
1993-1995	347	678	1 641	2 704	928
Change	+27	-139	+151	+461	-66
<i>Mortality ratio primary to tertiary</i>					
1972-1974	2.17	1.62	1.37	1.27	1.39
1993-1995	3.42	1.99	1.74	1.29	1.54
Change	+1.25	+0.37	+0.37	+0.02	+0.15

Source: Unpublished data from the Population Research Unit, University of Helsinki.

increase in inequality, even in the case when the death rates remain unchanged in all categories.

## B. CHANGES IN SOCIOECONOMIC DIFFERENTIALS IN MORTALITY: A REVIEW

### 1. Comparative studies

Several cross-country comparisons have been carried out on socioeconomic mortality differentials in the 1970s (Vågerö and Lundberg, 1989; Leclerc and others, 1990; Kunst and Mackenbach, 1994b and 1994c), but only one has also compared changes in inequalities (Valkonen, 1989). According to the findings, changes in mortality relative to educational inequality were not statistically significant in Finland, Sweden, Norway and Denmark in the 1970s, among men and women aged 35-54. In England and Wales inequality increased among both men and women. Among men this was mainly due to changes in cardiovascular mortality. The growth of relative mortality differentials among working-age men in England and Wales in the 1970s has also been observed using data on mortality by social class (Marmot and McDowall, 1986).

An international project headed by Johan Mackenbach (Mackenbach and others, 1997; Kunst, 1997) carried out a major comparative

study on cross-national differences in socioeconomic inequalities in mortality. The study also presents some data on changes in socioeconomic differentials in mortality in the 1980s (Kunst and Groenhof, 1996).

As a whole there is only very limited data on changes in these differentials and the quality of the data is far from perfect. This review covers those countries for which data on adult mortality, to the best of the author's knowledge, were available at least up to the mid-1980s. The main focus is on Denmark, England and Wales, Finland, Norway, Spain, Sweden and the United States, for which the data seem most reliable and relevant.

### 2. England and Wales

A recent report on Health Inequalities published by the Office for National Statistics (Drever and Whitehead, 1997) continues the tradition of decennial supplements that was started in England and Wales in the mid-nineteenth century. The data on socioeconomic differences are based on both sources discussed above in section A.1: the national death registry data on deaths in England and Wales and the Office for National Statistics Longitudinal Study, covering one per cent of the population. The results are mainly presented using the Registrar General's social classes. Since information on occupation is

partly missing for persons not economically active, the healthy worker bias discussed earlier may have some effect on the results. Housing tenure and access to cars are used as alternative indicators.

Using the Longitudinal Study data, the report shows that the gap in life expectancy between Classes I/II (professional and managerial and technical occupations) and the other classes widened between the late 1970s and the late 1980s (Hattersley, 1997). For men, for example, the gap in life expectancy at age 15 between Classes I/II and IV/V (partly skilled and unskilled workers) widened from four to five years over this period. Similarly, for women at age of 15, the gap widened from two to three years. It is not clear from the report how persons without an occupation were classified when calculating the life expectancies.

Both data sets used in the study point at a relative widening of class mortality differentials among men in working ages. For example, according to the Longitudinal Study the ratio of mortality in manual to non-manual men aged 35-64 increased from 1.2 in the late 1970s to 1.5 in the period 1986-1992. This widening was due to a more rapid relative decline of mortality in the non-manual group, particularly from the early 1980s to late 1980s (Harding and others, 1997). Among women aged 35-64 the Longitudinal Study data showed a narrowing and then a widening of the relative differential between manual and non-manual classes from the late 1970s to late 1980s.

The report does not discuss the causes of the widening of the mortality gap among men in any detail. However, the possible contribution of unemployment is analyzed. It is concluded that the increase in unemployment does not explain the widening of the mortality gap. The tables presented in the report show that mortality from heart disease (IHD) mortality was a major factor contributing to the increase in mortality differentials. In 1970-1972 the difference in mortality from IHD between unskilled workers (Class V) and professionals (Class I) was relatively small, 48 deaths per 100,000 or 25 per cent. From

1970-1972 to 1991-1993 mortality from IHD in Class I declined by 58 per cent and by 3 per cent in Class V. As a result the gap was 154 per 100,000 or 190 per cent in 1991-1993 (Drever and Bunting, 1997, table 8.6). Changes in other causes of death than IHD for which data are shown, namely lung cancer, stroke, accidents and suicide, also contributed to the relative widening of the socioeconomic mortality gap.

### *3. Finland*

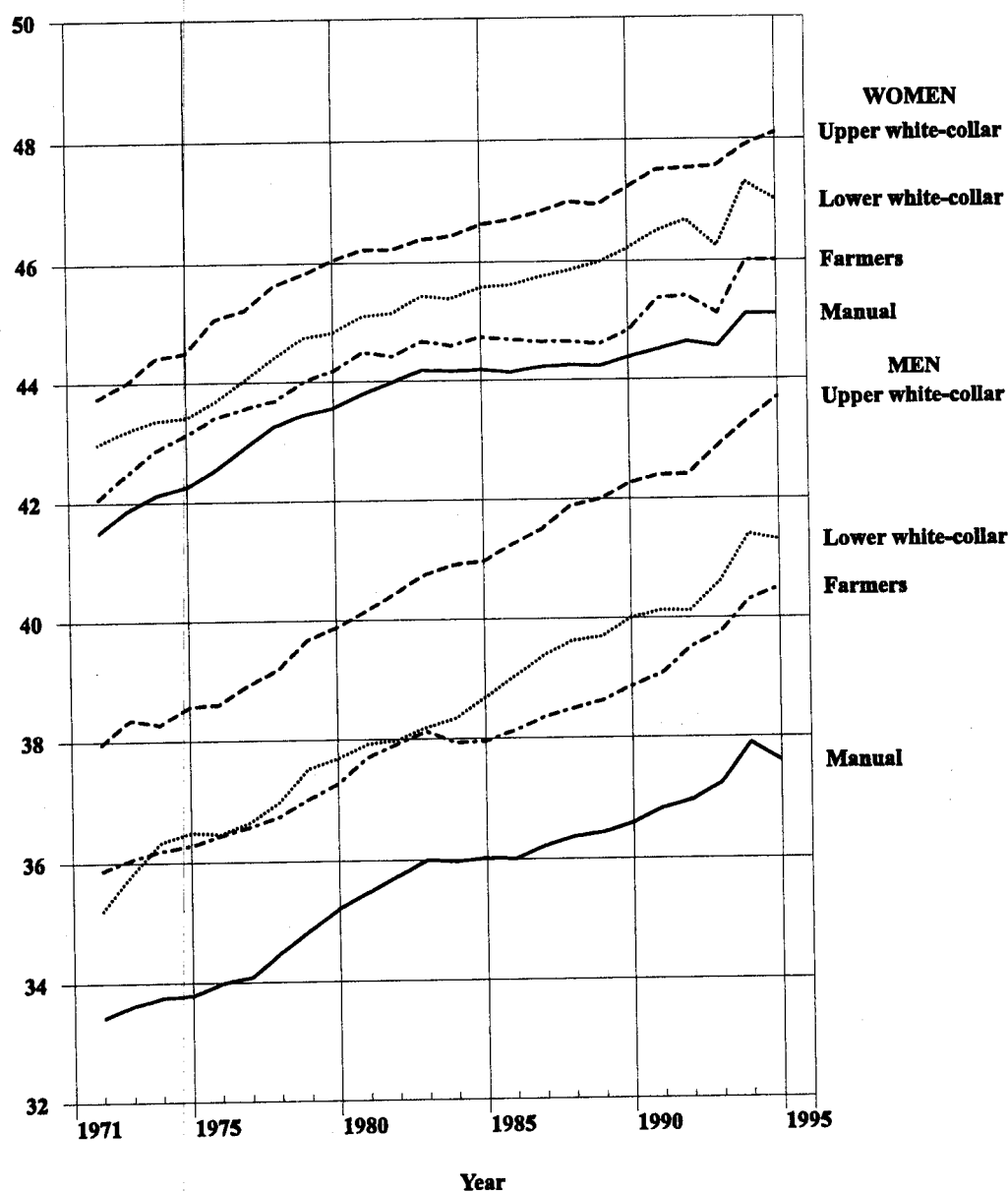
Scholars in Finland have made good use of the possibility of computerized linking of census and death records in socioeconomic mortality research and trends in social class and educational differentials have probably been analyzed more thoroughly than in any other country (e.g. Valkonen and others, 1990; Valkonen and others, 1993; Valkonen, 1992 and 1993; Valkonen and Jalovaara, 1997).

Finnish data on trends in male mortality by level of education in ages 35 and over were already shown in section A.2 above. The results indicate that relative differences in mortality by level of education increased in all age groups from the 1970s to the 1990s. Due to the decline in the overall level of mortality, changes in the absolute differences have been small, and among men aged 50-64 and 75 and over, the absolute difference diminished from 1972-1974 to 1993-1995.

The most common measure of level of mortality used in comparisons between countries or in describing trends is life expectancy. Life expectancy also seems to be the best summary measure for drawing conclusions about the possible widening of the socioeconomic mortality gap. Figure 91 shows the trends in life expectancy at age 35 for occupation-based social class in Finland. They point at a clear widening of differences which has mainly occurred after the 1970s. The difference between male manual workers and upper non-manual employees was 4.5 years in 1972-1974, but in 1993-1995 it was 6.1 years. The difference has also increased among women but less than among men, from 2.3 years to 3.0 years. The results are approximately the



Figure 91. Life expectancy at the age of 35 by occupation class, men and women, 1971-1995



Sources: Pekka Martikainen and Tapani Valkonen, *Lama ja ennenaikainen kuolleisuus* (Statistics Finland, Population 1995); unpublished data from the Population Research Unit, University of Helsinki.

same if level of education is used as the socio-economic indicator.

The trends in life expectancy and all-cause mortality are the result of the sum of trends in mortality from specific causes of death. It is

difficult to summarize the cause-specific results briefly because they vary according to age group, sex and period. To illustrate this complexity it is useful to look at changes in mortality from selected causes of death among manual workers and non-manual employees

aged 35-64 during 1981-1995 (figure 92) (Valkonen and others, 1998). The data are based on death records for 1981-1995 which have been linked to the census records for 1980, 1985 and 1990.

Both the relative and absolute difference in all-cause mortality between male manual and non-manual workers increased from 1981-1985 to 1986-1990. Lung cancer was the only cause of death identified for which the class difference decreased (figure 92). The increase was greatest in mortality from ischaemic heart disease, which diminished rapidly in both classes, but more in the non-manual class. The patterns of change were similar in mortality from cerebrovascular diseases and other cardiovascular diseases as in mortality from ischaemic heart disease. Altogether, the differences in the trends for cardiovascular diseases accounted for about 40 per cent of the total increase in the male mortality gap from 1981-1985 to 1986-1990.

More than half of the increase in the class difference in total mortality from 1981-1985 to 1986-1990 among men was due to four groups of causes of death: alcohol-associated causes, "other diseases" (diseases other than cancer, cardiovascular diseases or alcohol-associated diseases), suicides, and accidents and violence other than suicide and alcohol poisoning. Mortality from these causes increased in the manual class and, except for "other diseases", also in the non-manual class, but the increase was greater in the manual class (figure 92). Alcohol-associated deaths here include deaths for which the underlying cause is either a disease directly associated with alcohol use or accidental alcohol poisoning.

Both the relative and absolute class mortality gaps increased among women from 1981-1985 to 1986-1990. As in the case of men, cardiovascular mortality diminished in both classes, but more so in the non-manual class than in the manual class (figure 92). Since the proportion of cardiovascular deaths of all deaths is smaller among women than men in the age bracket studied here, the differences in trends in mortality from cardiovascular diseases only contributed about 20 per cent to the

widening of the mortality gap. Again, as among men, mortality from alcohol-associated causes, "other diseases", suicide, and accidents etc. increased among women in the manual class, but less so or not at all in the non-manual class. Altogether these causes accounted for most of the increase in the difference in total mortality.

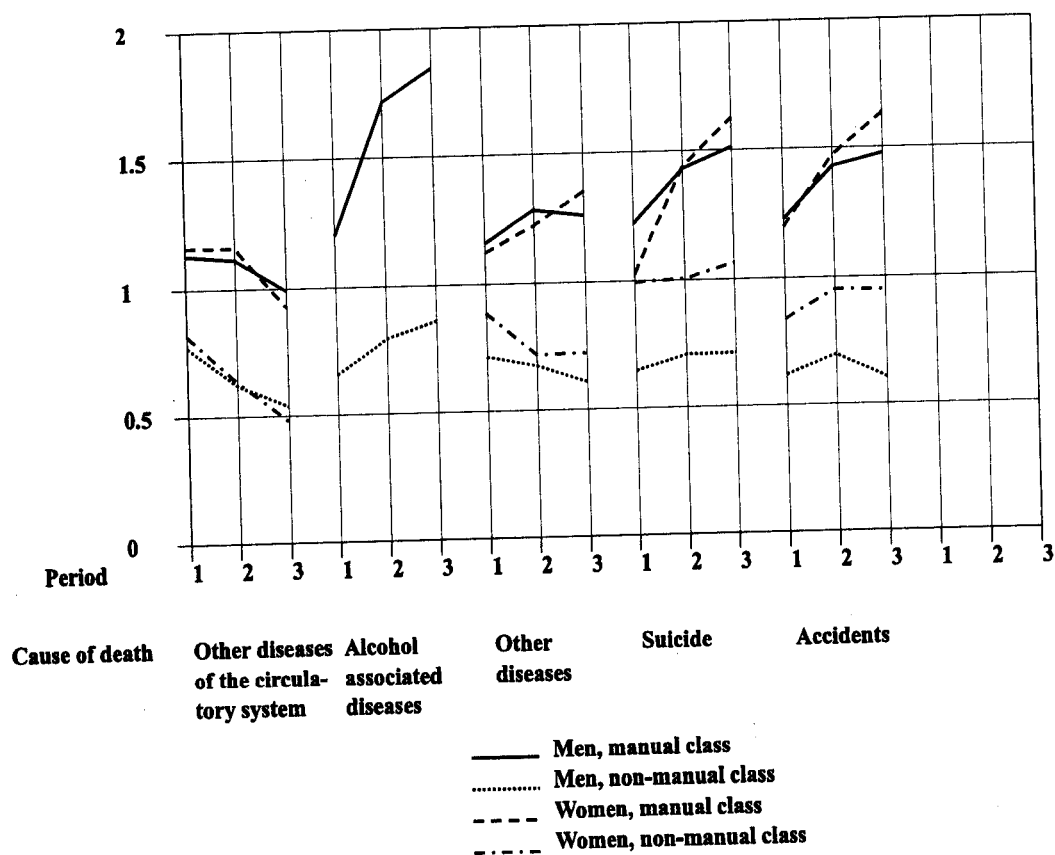
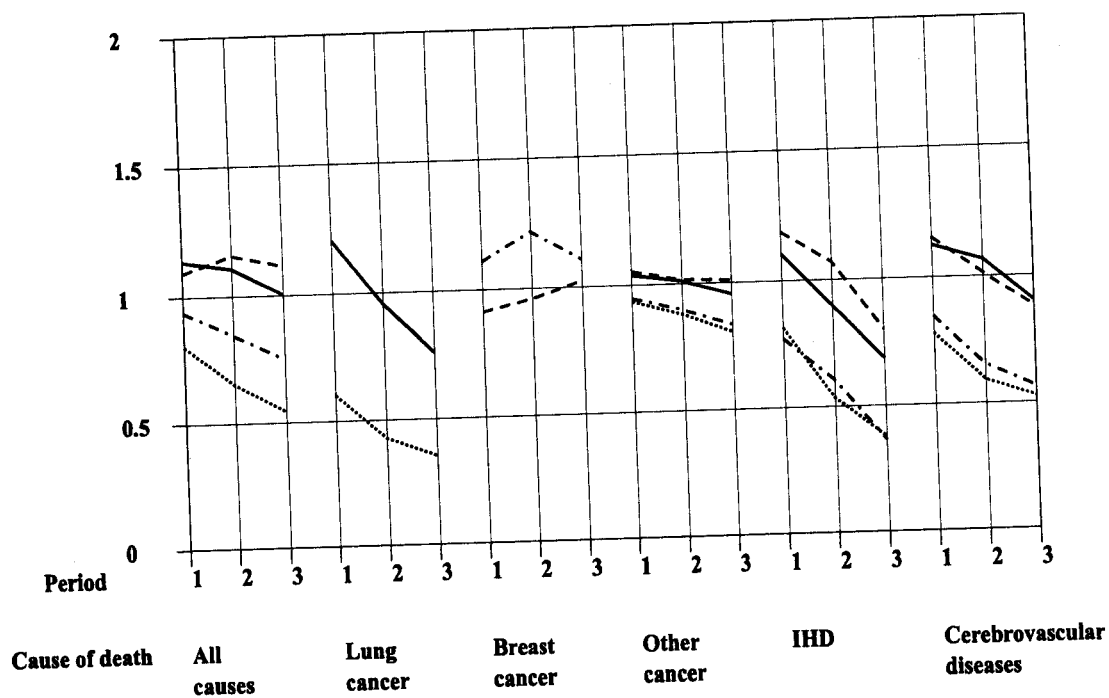
Mortality from breast cancer was somewhat higher in the non-manual class than in the manual class in 1981-1985. It increased slightly in both classes from 1981-1985 to 1986-1990, whereas mortality from other cancers diminished. There was practically no class difference in total cancer mortality among women.

Overall, mortality showed more favourable trends in development in the early 1990s than in the late 1980s: mortality among the men studied here dropped by 9.1 per cent from the period 1981-1985 to 1986-1990 but by 14.6 per cent from the period 1986-1990 to 1991-1995. Among women these figures were 3.6 per cent and 6.6 per cent, respectively.

The absolute class difference in male mortality in 1991-1995 was slightly smaller than in 1986-1990, although the relative difference still increased. The main factor responsible for the increase in the absolute mortality gap coming to an end was the development of mortality from cardiovascular diseases: unlike in the 1980s, cardiovascular mortality diminished more in the manual than in the non-manual class (figure 92). Another important factor was the development of mortality from alcohol-associated causes, "other diseases", suicide and accidents etc. Mortality from these causes (except for "other diseases") continued to increase in the manual class, but the increase was much smaller than in the 1980s. Although the class difference in mortality from these causes continued to increase, the decrease in the gap in mortality from cardiovascular diseases and lung cancer more than compensated for the increase.

Among women the class mortality difference continued to increase in the 1990s, but the absolute increase was only about half of that in the 1980s. As among men, the de-

**Figure 92. Relative age-standardized mortality from selected causes of death, by social class, men and women aged 35-64 in Finland, 1981-1985 (1), 1986-1990 (2) and 1991-1995 (3) (1.0 = all men/women, 1981-1985)**



Source: Unpublished data from the Population Research Unit, University of Helsinki.

crease in cardiovascular mortality was now greater in the manual than in the non-manual class. Due to the low level of female cardiovascular mortality this was not sufficient to stop the absolute mortality gap from widening, to which all causes other than cardiovascular diseases contributed.

#### 4. Sweden, Norway and Denmark

Sweden, Norway and Denmark have similar systems of personal identity numbers to Finland, which means that socioeconomic mortality differences can be studied by linking death records to census records. Most results from these countries are based on occupational class and cover only economically active persons.

*Swedish* results on mortality by socioeconomic group have been presented by Vågerö and Gullberg (1996) for the periods 1981-1985 and 1986-1990; a recent publication (Folkhälsorapport, 1997) includes a graph showing results also for 1991-1995. According to the results, all-cause mortality declined in all groups of economically active men aged 20-64. The absolute difference between the non-manual and manual groups seems to have remained approximately the same or to have increased slightly throughout the period 1981-1995. As the overall level of mortality decreased, relative differences increased. Among economically active women mortality differentials increased because the decline in mortality was smaller among medium and lower level non-manual employees and unskilled workers than among higher level non-manual employees. However, it is likely that the results for women are heavily biased due to the exclusion of economically inactive persons.

The sources mentioned above do not give cause-specific data. An earlier study by Vågerö and Lundberg (1995) shows that all-cause mortality among non-manual men decreased markedly from 1961-1965 to 1981-1986 as a result of reduced cardiovascular mortality. Both the absolute and relative mortality gaps grew, because there was almost no decline in all-cause mortality among manual men and cardiovascular mortality even

increased in this group. The central contribution of cardiovascular mortality to the growth of socioeconomic mortality differentials in Sweden from the 1960s to the early 1980s is also highlighted by results on mortality by occupational group categorized according to sector of the economy (Diderichsen and Hallqvist, 1997). Mortality from cardiovascular diseases (adjusted for the healthy worker effect) was 11 per cent *lower* among men in industrial occupations than in managerial, professional and commercial occupations in 1961-1965, but 37 per cent *higher* in 1981-1986. The reversal took place in the early 1970s. From 1981-1986 to 1986-1990 the absolute decline in cardiovascular mortality was approximately the same in both categories.

For *Norway* results on trends in socioeconomic mortality differentials are available on the basis of classes (Dahl and Kjaersgaard, 1993; Borgan, 1996). They only cover persons who were economically active at the time of the censuses. Because of the healthy worker bias the data for women seem too unreliable to be useful.

In the 1960s male mortality differences between classes were small: the mortality of unskilled workers was only 22 per cent higher than that of higher level salaried employees (Borgan, 1996). In 1985-1990 the relative difference was much greater at 73 per cent, because mortality from cardiovascular diseases diminished more rapidly in the manual than non-manual classes. Another factor contributing to the increase in the mortality gap was lung cancer mortality: the excess mortality of unskilled workers compared to skilled workers was 30 per cent in 1960-1965 but 129 per cent in 1980-1985.

In *Denmark* mortality diminished by almost 20 per cent among economically active male white-collar employees and skilled workers from 1971-1975 to 1986-1990, but remained almost unchanged among unskilled workers and farmers (Ingerslev and others, 1994). As elsewhere, the main factor contributing to the increase in the mortality gap was cardiovascular mortality. In 1971-1975 mortality from cardiovascular diseases was about the same

among white-collar employees, skilled workers and unskilled workers. By 1986-1990 mortality among white-collar employees and skilled workers had dropped by about 35 per cent but only by about 10 per cent among unskilled workers. In addition, mortality from cancer increased among skilled workers but decreased in the other groups.

### 5. Spain

No data are available on socioeconomic mortality for the whole country of Spain. The study by Redigor and others (1995) covers eight provinces, for which the data on occupation on the death certificate are the most reliable. The data set used in the analysis has many weaknesses: the data for the numerator and denominator of death rates come from different sources, information on occupation was not available for 9 per cent of the deceased, the study covered only economically active men aged 30-64. Only two groups are compared: professional and managerial occupations and production and transportation workers; the former group comprised less than 10 per cent of economically active men. Despite these weaknesses the main results on cause-specific mortality are worth reporting: the excess mortality for the manual labourers compared to professionals and managers increased from 27 per cent in 1980-1982 to 72 per cent in 1988-1990. Most causes of death contributed to this increase and the relative excess mortality of labourers increased for all causes except cancer of the colon and rectum. The most interesting finding, however, was that mortality from ischaemic heart disease was 20 per cent *lower* among labourers than among professionals and managers in 1980-1982, but 31 per cent *higher* in 1988-1990. In spite of the weaknesses of the data, it emerges clearly from the results that the reversal of class differences in male mortality from ischaemic heart disease occurred in the Spanish provinces studied as late as the 1980s. According to the results of Mackenbach and others (1997) there were almost no socioeconomic differences in mortality from ischaemic heart disease in Spain, Italy and Portugal in the early 1980s.

### 6. United States

Several studies have been carried out on changes in socioeconomic mortality differentials in the United States (Feldman and others, 1989; Pappas and others, 1993; Preston and Elo, 1995). However, the results of these studies are not up to date, because the most recent data are for 1986.

The study by Preston and Elo is the most thorough of the studies presenting results on trends. It is based on five Current Population Surveys (CPS) conducted between March 1979 and March 1981. Individuals enumerated in the CPS were followed up for a five-year period by linking the records to the National Death Index for the years 1979-1985. The number of deaths is relatively large (22,649) and there is no numerator/denominator bias. Educational attainment is used as the indicator of socioeconomic status. Both absolute and relative differences are examined and changes in educational distributions are taken into account by using appropriate inequality measures.

The authors conclude that compared to 1960, educational inequalities have widened for males but contracted for working-age females. For both sexes, inequality trends are more adverse for persons aged 65+ than for persons aged 25-64. However, it is difficult to draw firm conclusions about trends in inequalities particularly in the older age groups because of possible biases in the baseline data, which were obtained from the study by Kitagawa and Hauser (1973).

Feldman and others (1989) also found evidence of a widening mortality gap among males in the United States in their study, which compared mortality in 1960 with mortality during 1971-1984. They had cause-specific data and concluded that trends in educational differentials for heart disease mortality were responsible for much of the change in all cause-specific mortality. Feldman and others discuss the factors that may have influenced trends in heart disease mortality, such as changes in the distribution of cigarette

smoking (which is not found to be of major importance) and improvements in medical and surgical treatment of heart disease, which may have diffused more rapidly among better educated people. The final conclusion, however, was that "reasons for the observed educational differentials and their changes over time are not easily explained and are likely to be multifactorial."

According to the results presented by Kunst (1997) relative mortality differences both by education and occupational class increased in the United States from the period 1979-1984 to the period 1985-1989 among men aged 40-54. The results are based on data from the National Longitudinal Mortality Study and are thus not influenced by the possible bias in baseline data.

#### *7. Other countries*

There are some other relevant data, which cannot be discussed in detail here. For example, Bennet (1996) studied socioeconomic inequalities in coronary heart disease and stroke mortality among Australian men during 1979-1993. According to the results both men in manual and professional groups experienced reductions in coronary risk and mortality since 1979. Socioeconomic inequalities in coronary heart disease mortality widened during the early 1980s, stabilized thereafter and persisted into the 1990s. However, the study is based on data that may be biased: the data for deaths and the risk population come from different sources and only cover men in the workforce.

There are also data from Italy showing increases in socioeconomic differentials. Kunst (1997) has analysed data from Costa's linked records study for Turin, which show some increase in relative mortality differences by level of education and social class among men aged 50-59 years during the 1980s.

As mentioned earlier, longitudinal studies on mortality by occupational group and level of education have been carried out in France (Desplanques, 1976 and 1984). Early results from these studies show that mortality differ-

entials between socio-occupational groups increased from 1955-1959 to 1975-1980. The more recent data published in France do not make an unbiased study of trends possible. According to Kunst's (1997) calculations based on the French data, the excess mortality of 50-59 year old men with low education compared to men with high education was 54 per cent in 1981-1985 and 75 per cent in 1986-1990. A similar increase in the mortality gap was observed between men in manual and non-manual occupations.

#### C. CONCLUSIONS

The first part of this paper discussed some of the problems involved in measuring socioeconomic differences in mortality and, particularly, in drawing conclusions as to whether these differences are increasing or not. It is obvious that there exists no single valid method for measuring the size of socioeconomic differences in mortality. In 1985 The WHO Regional Office for Europe issued a document listing its Targets for Health for All. The first of these 33 targets is that "by the year 2000, the actual differences in health status between and within countries should be reduced by at least 25 per cent". The most common indicator for health status used in the document is mortality. The quantitative target of reducing socioeconomic differences in mortality by at least 25 per cent may be useful as an expression of good intentions, but it is problematic in the sense that it is impossible to establish how far it is reached. In practice, however, this hardly matters because, as we have seen in this review, the socioeconomic differences in adult mortality have in fact not diminished at all, but generally increased at least in relative terms.

Socio-economic differentials are substantial in all countries for which data are available and should be a major concern for health and social policies. In Finland, for example, the six-year difference in the life expectancy of males in manual and upper non-manual classes implies that the current level of mortality in the manual class is the same as it was in the non-manual class more than 30 years ago.

Most of the data available on changes in socioeconomic mortality differences cover working-age men. In all developed non-transitional countries, death rates in this age group have diminished. The main factor behind this decrease has been the decline in mortality from ischaemic heart and other cardiovascular diseases.

In all countries covered by this review, ischaemic heart disease mortality is currently higher in the lower socioeconomic groups than in the higher groups, but this has not always been the case. According to Kunst (1997, p. 168), until about 1950 studies on ischaemic heart disease mortality among men at working age in the United States observed no or even positive association with indicators of socioeconomic status. The first inverse gradients appeared in the 1950s. In England and Wales the first inverse gradients among men were observed around 1960 (Marmot and others, 1978), but weaknesses of data may have affected results for earlier periods (Davey Smith, 1997). According to the data reviewed here the pattern of higher mortality from ischaemic heart disease or cardiovascular diseases among working-age men emerged in Sweden, Norway and Denmark in the 1960s or 1970s and in Spain in the 1980s. In Finland mortality from ischaemic heart disease as well as from all cardiovascular diseases was clearly higher among manual workers and less educated men and women in all age-groups already in the early 1970s (Valkonen and others, 1990).

Whatever the pattern of differences was in earlier times, the decline of cardiovascular mortality has been more rapid in the upper than in the lower socio-economic men in all countries for which we have data. This difference in trends has been the most important single cause for the widening of the socioeconomic mortality gap.

The growth of the male mortality gap resulting from trends in cardiovascular diseases can be viewed from two perspectives, a pessimistic and an optimistic one. According to the pessimistic view there is no indication that the mortality gap is going to decrease and indeed may even grow further. According to

the optimistic view, the rapid decline in cardiovascular mortality in the higher social classes shows that cardiovascular mortality is preventable and that rapid progress in reducing its detrimental effects is possible. So far the upper socioeconomic classes have benefited more from this trend, probably because they have been quicker to adopt recommended health behaviours (concerning diet, smoking, physical activity etc.), and because they have had better access to new medical treatments, such as coronary artery by-pass grafting and angioplasty (Keskimäki and others, 1997). According to the optimistic view the lower socioeconomic classes are likely to adopt the same healthy habits and will benefit from the new medical techniques when they become routine treatments. They will thus reach the current levels of the higher classes sooner or later. It is likely that the decline of mortality in the higher classes will slow down in the future, because it cannot continue indefinitely. We can therefore perhaps expect to see a narrowing of the cardiovascular and possibly of the all-cause mortality gap. The data for Finland presented above seem to lend some support to the optimistic view: mortality from cardiovascular diseases diminished more in the manual class than in the non-manual class in the 1990s. There is also evidence from Sweden that points at a slowing down of the increase in the gap in mortality from cardiovascular diseases (Diderichsen and Hallqvist, 1997).

Other causes than cardiovascular diseases have also contributed to the growth of mortality differentials among working-age men. Data on the contribution of these other causes are not systematic and it is hard to make generalisations. It seems, however, that there is no such cross-country uniformity in patterns of change as in the case of cardiovascular diseases. For example, lung cancer mortality has contributed to the increase in the mortality gap in several countries, but not in Finland in the 1980s and 1990s. On the other hand alcohol-associated mortality was a major cause contributing to the growth of the mortality gap in the 1980s and to a lesser extent in the 1990s in Finland, but there is no information of such effects from other countries.

In the light of the cause-specific results for Finland, it seems that changes in differences in mortality result from a complex combination of different and even opposite trends for various causes of death, which may vary by sex, class and period. It is unlikely that any overall explanation for differences and trends can be valid. For example, the suggestion that an increase in income inequalities is a major cause of the widening of inequalities in mortality (e.g. Wilkinson, 1994; Davey Smith and Egger, 1993) does not fit in the case of Finland. Inequalities in disposable income measured by Gini-coefficient diminished in Finland rapidly from 1965 to 1975 and continued to diminish slightly until 1985. Since then they have been more or less constant (Uusitalo, 1997). Nevertheless socio-economic inequalities have increased since the 1970s.

Reasonably reliable data on changes in mortality by socioeconomic status for other groups than working-age men are limited. Results on changes in life expectancy by social class are available only for two countries, Finland and England and Wales. In both cases socioeconomic differences in male and female life expectancy have increased markedly since the 1970s. It seems likely that the widening of the socioeconomic gap in cardiovascular mortality observed in the working-age male population in several countries has also occurred in older male and female populations, contributing to the growth of the socioeconomic gap in all-cause mortality.

The widening of the difference in mortality between the less and the more privileged socioeconomic groups is a trend about which we still know too little. The first task for future research is to collect relevant and valid descriptive data. The second and more important task is to increase our understanding of the phenomenon. To what extent is the widening of the gap a temporary phenomenon caused mainly by the earlier decline in cardiovascular mortality in the higher socioeconomic groups? To what extent is it a consequence of deeper tendencies in societal development leading to increasing social polarization in the living conditions and health status of the population? To what extent is it

due to specific factors in different countries and periods?

#### REFERENCES

- Antonovsky, Aaron (1967). Social class, life expectancy and overall mortality. *Milbank Memorial Fund Quarterly*, vol. 45, pp. 31-73.
- Bennett, Stan (1996). Socioeconomic inequalities in coronary heart disease and stroke mortality among Australian men, 1979-1993. *International Journal of Epidemiology*, vol. 25, No. 2, pp. 266-275.
- Borgan, Jens-Kristian (1996). Socioeconomic trends in differential mortality among middle-aged males in Norway 1960-1990. *Yearbook of Population Research in Finland*, vol. 33, pp. 73-81.
- Dahl, Espen, and Pal Kjaersgaard (1993). Trends in socioeconomic mortality differentials in post-war Norway: evidence and interpretations. *Sociology of Health and Illness*, vol. 15, No. 5, pp. 587-611.
- Davey Smith, George (1997). Socioeconomic differentials. In *A Life Course Approach to Chronic Disease Epidemiology*, Diana Kuh and Yoav Ben-Shlomo, eds. Oxford Medical Publications, Oxford: Oxford University Press, pp. 248-273.
- \_\_\_\_\_, and Matthias Egger (1993). Socioeconomic differentials in wealth and health. Widening inequalities in health - the legacy of the Thatcher years. *British Medical Journal*, vol. 307 (30 October), pp. 1085-1086.
- Desplanques, Guy (1976). La mortalité des adultes suivant le milieu social 1955-1971. *No 195 des collections de l'INSEE*, série D, No. 44.
- Desplanques, Guy (1984). L'inégalité sociale devant la mort. *Économie et Statistique* no 162 (janvier), pp. 29-49.
- Diderichsen, Finn, and Johan Hallqvist (1997). Trends in occupational mortality among middle-aged men in Sweden 1961-1990. *International Journal of Epidemiology*, vol. 28, No. 4, pp. 782-787.
- Drever, Frances, and Margaret Whitehead, eds. (1997). *Health Inequalities. Decennial Supplement*. Office for National Statistics, Series DS No.15, London: The Stationery Office.
- Drever, Frances (1997). How were the census based mortality rates calculated? In *Health Inequalities. Decennial Supplement*, Frances Drever and Margaret Whitehead, eds. Office for National Statistics, Series DS No.15, London: The Stationery Office, pp. 241-251.
- \_\_\_\_\_, and Julia Bunting (1997). Patterns and trends in male mortality. In *Health Inequalities. Decennial Supplement*, Frances Drever and Margaret Whitehead, eds. Office for National Statistics, Series DS No. 15, London: The Stationery Office, pp. 95-107.
- Erikson, Robert, and John H. Goldthorpe (1992). *The Constant Flux. A Study of Class Mobility in Industrial Societies*. Oxford: Clarendon Press.
- Feinstein, Jonathan S. (1993). The relationship between socioeconomic status and health: A review of literature. *The Milbank Quarterly*, vol. 71, pp. 279-322.
- Feldman, Jacob J., Diane M. Makuc, Joel C. Kleinman and Joan Cornoni-Huntely (1989). National trends in educational differentials in mortality. *American Journal of Epidemiology*, vol. 129, No. 5, pp. 919-933.
- Folkhälsorapport 1997 (1997). National Board of Health and Welfare, Stockholm, SoS Rapport 1997, vol. 18.



- Fox, A. J. (1979). Prospects for change in differential mortality. *Proceedings of the meeting on socioeconomic determinants and consequences of mortality*, El Colegio de Mexico, Mexico City, 15-19 June 1979, New York/Geneva, pp. 515-561.
- Goldblatt, Peter, ed. (1990). *Longitudinal Study. Mortality and Social Organisation 1971-1981*. London: Office of Population Censuses and Surveys, Series LS no. 6.
- Harding, Seeromanie, Ann Bethune, Roy Maxell and Joanna Brown (1997). Mortality trends using the Longitudinal Study. In *Health Inequalities. Decennial Supplement*, Frances Drever and Margaret Whitehead, eds. Office for National Statistics, Series DS No. 15, London: The Stationery Office, pp. 143-155.
- Hattersley, Lin (1997). Expectation of life by social class. In *Health Inequalities. Decennial Supplement*. Frances Drever and Margaret Whitehead, eds. Office for National Statistics, Series DS No 15, London: The Stationery Office, pp. 73-82.
- Ingerslev, Olaf, Mette Madsen and Otto Andersen (1994). *Sociale forskelle i dødeligheden i Danmark*. Copenhagen: Sundhedsministeriets Middellevetidsudvalg.
- Keskimäki, Ilmo, Seppo Koskinen, M. Salinto and Seppo Aro (1997). Socioeconomic and gender inequalities in access to coronary artery bypass grafting in Finland. *European Journal of Public Health*, vol. 7, pp. 392-97.
- Kitagawa, Evelyn M., and Philip M. Hauser (1973). *Differential Mortality in the United States: a Study in Socio-economic Epidemiology*. Cambridge, Massachusetts: Harvard University Press.
- Kunst, Anton E. (1996). Potential sources of bias in 'unlinked' cross-sectional studies. In *Socioeconomic Inequalities in Morbidity and Mortality in Europe: A Comparative Study*. Anton Kunst, Adrienne Cavelaars, Feikje Groenhouf, José Geurts, Johan P. Mackenbach and EU Working Group on Socioeconomic Inequalities in Health, Volume 2: Working Documents, Department of Public Health, Erasmus University Rotterdam, pp. 147-162.
- \_\_\_\_\_ (1997). *Cross-national comparisons of socioeconomic differences in mortality*. Thesis, Erasmus University Rotterdam.
- \_\_\_\_\_, and Feikje Groenhouf (1996). Mortality differences by educational level and occupational class: the effect of excluding economically inactive men. In *Socioeconomic Inequalities in Morbidity and Mortality in Europe: A Comparative Study*. Anton Kunst, Adrienne Cavelaars, Feikje Groenhouf, José Geurts, Johan P. Mackenbach and EU Working Group on Socioeconomic Inequalities in Health, Volume 2: Working Documents, Department of Public Health, Erasmus University Rotterdam.
- Kunst, Anton E., and Johan P. Mackenbach (1994a). *Measuring Socioeconomic Inequalities in Health*. World Health Organization. Regional Office for Europe, Copenhagen, EUR/ICP/RPD 416, 12234.
- \_\_\_\_\_ (1994b). International variations in the size of mortality differences associated with occupational status. *International Journal of Epidemiology*, vol. 23, pp. 742-750.
- \_\_\_\_\_ (1994c). The size of mortality differences associated with educational level in 9 industrialised countries. *American Journal of Public Health*, vol. 84, pp. 932-937.
- Leclerc, A., F. Lert and C. Fabien (1990). Differential mortality: some comparisons between England and Wales, Finland and France, based on inequalities measures. *International Journal of Epidemiology*, vol. 19, pp. 1-10.
- Mackenbach, Johan P., and Anton E. Kunst (1997). Measuring the magnitude of socio-economic inequalities in health: an overview of available measures illustrated with two examples from Europe. *Social Science & Medicine*, vol. 44, No. 6, pp. 757-771.
- \_\_\_\_\_, Adrienne E.J.M. Cavelaars, Feikje Groenhouf, José J.M. Geurts and the EU Working Group on Socioeconomic Inequalities in Health (1997). Socioeconomic inequalities in morbidity and mortality in western Europe. *Lancet*, vol. 349 (June 7), pp. 1655-1659.
- Marmot, M. G., A. M. Adelstein, N. Robinson and G.A. Rose (1978). Changing social-class distribution of heart disease. *British Medical Journal*, vol. 2 (21 October), pp. 1109-1112.
- Marmot, M. G., and M. E. McDowall (1986). Mortality decline and widening social inequalities. *Lancet*, vol. 2, pp. 274-276.
- Martikainen, Pekka, and Tapani Valkonen (1995). *Lama ja ennenaikainen kuolleisuus*. Helsinki: Statistics Finland, Population 1995:11.
- Mielck, Andreas, and Maria do Rosário Giraldez, eds. (1993). *Inequalities in Health and Health Care. Review of Selected Publications from 18 Western European Countries*. Münster/New York: Waxmann.
- Office of Population Censuses and Surveys (OPCS) (1978). *Occupational Mortality. The Registrar General's Decennial Supplement for England and Wales, 1970-71*. London, Her Majesty's Stationery Office, Series DS No. 1.
- \_\_\_\_\_ (1986). *Occupational Mortality. The Registrar General's Decennial Supplement for Great Britain, 1979-80, 1982-83*. London, Her Majesty's Stationery Office, Series DS No. 6.
- Pappas, Gregory, Susan Queen, Wilbur Hadden and Gail Fisher (1993). The increasing disparity in mortality between socioeconomic groups in the United States, 1960 and 1986. *The New England Journal of Medicine*, vol. 329, No. 2 (July 8), pp. 103-109.
- Preston, Samuel H., and Irma T. Elo (1995). Are educational differentials in adult mortality increasing in the United States? *Journal of Aging and Health*, vol. 7, No. 4 (November), pp. 476-496.
- Quine, Susan, Richard Taylor and Lillian Hayes (1995). Australian trends in mortality by socioeconomic status using NSW small area data, 1970-89. *Journal of Biosocial Science*, vol. 27, pp. 409-419.
- Rigdor, Enrique, Juan L. Gutiérrez-Fisac and Carmen Rodriguez (1995). Increased socioeconomic differences in mortality in eight Spanish provinces. *Social Science & Medicine*, vol. 41, No. 6, pp. 801-807.
- Smith, Jillian, and Seeromanie Harding (1997). Mortality of women and men using alternative social classifications. In *Health Inequalities. Decennial Supplement*, Frances Drever and Margaret Whitehead, eds. Office for National Statistics, Series DS No. 15, London: The Stationery Office, pp. 168-183.
- Thatcher, A. Roger (1986). Preface. In *Office of Population Censuses and Surveys. Occupational Mortality 1979-80, 1982-83. Decennial Supplement. Part I Commentary*. Her Majesty's Stationery Office, Series DS No. 6.
- Uusitalo, Hannu (1997). Neljä laman vuotta: mitä on tapahtunut tulonjaossa? *Leikkausten hinta. Tutkimuksia sosiaaliturvan leikkauksista ja niiden vaikutuksista 1990-luvun Suomessa*, Matti Heikkilä and Hannu Uusitalo, eds. Stakes, Sosiaali- ja terveysalan tutkimus- ja kehittämiskeskus, Raportteja 208, pp. 135-151.
- Vågerö, Denny, and Anders Gullberg (1996). *Yrke och dödlighet under 1980-talet*. Stockholm: Socialstyrelsen, Rapport 1996, vol. 3.
- Vågerö, Denny, and Olle Lundberg (1989). Health inequalities in Britain and Sweden. *Lancet*, vol. ii, pp. 35-36.
- \_\_\_\_\_ (1995). Socio-economic mortality differentials among adults in Sweden. In *Adult Mortality in Devel-*

- oped Countries. *From Description to Explanation*, Alan Lopez, Graziella Caselli and Tapani Valkonen, eds. Oxford: Clarendon press, pp. 223-242.
- Valkonen, Tapani (1987). Social inequality in the face of death. *European Population Conference 1987. Plenaries*, Helsinki: Central Statistical Office of Finland, pp. 201-260.
- \_\_\_\_\_. (1989). Adult mortality and level of education: a comparison of six countries. In *Health Inequalities in European Countries*, John Fox, ed. Gower: Aldershot, pp. 142-162.
- \_\_\_\_\_. (1992). Trends in regional and socio-economic mortality differentials in Finland. *International Journal of Health Sciences*, vol. 3, No. 3/4, pp. 157-166.
- \_\_\_\_\_. (1993). Problems in the measurement and international comparisons of socio-economic differences in mortality. *Social Science and Medicine*, vol. 36, No. 4, pp. 409-418.
- \_\_\_\_\_, Tuija Martelin and Arja Rumble (1990). *Socio-economic Mortality Differences in Finland 1971-85*. Helsinki: Statistics Finland, Studies 176.
- Valkonen, Tapani, Tuija Martelin, Arja Rimpelä, Veijo Notkola and Soili Savola (1993). *Socio-economic Mortality Differences in Finland 1981-90*. Helsinki: Statistics Finland, Population 1993:1.
- Valkonen, Tapani, and Marika Jalovaara (1996). Kuolleisuus koulutusasteen mukaan 1987-95. (Mortality by level of education 1987-95). Helsinki: Statistics Finland, *Causes of Death 1995*, Health 1996:5, pp. 17-19, 50-55.
- Valkonen, Tapani, and Pekka Martikainen (1997). Problems of comparability resulting from the exclusion of economically inactive persons in studies on social class differences in mortality. In *Morbidity and Mortality Data: Problems of Comparability, Proceedings of the EAPS and Hacettepe Institute of Population Studies Workshop*, Attila Hanciogly and Guillaume Wunsch, eds. Hacettepe Institute of Population Studies, Population No. IPS-HU. 97-01, Ankara.
- \_\_\_\_\_, Marika Jalovaara, Seppo Koskinen, Tuija Martelin and Pia Mäkelä (1998). Changes in socio-economic inequalities in male mortality during an economic boom and recession in Finland. Unpublished manuscript.
- Wagstaff, A., P. Paci and E. van Doorslaer (1991). On the measurement of inequalities in health. *Social Science and Medicine*, vol. 33, pp. 545-557.
- Westergaard, Harald (1901). *Die Lehre von der Mortalität und Morbilität. Anthropologisch-statistische Untersuchungen*. Jena: Verlag von Gustav Fischer.
- Whitehead, Margaret, and Finn Diderichsen (1997). International evidence on social inequalities in health. In *Health Inequalities, Decennial Supplement*, Frances Drever and Margaret Whitehead, eds. Office for National Statistics Series DS No. 15. London: The Stationery Office.
- Wilkins, R., O. Adams and A. Brancner (1989). *Changes in Mortality by Income in Urban Canada*, Statistics Canada, Health Reports.
- Wilkinson, R. G. (1994). Divided we fall. The poor pay the price of increased social inequality with their health. *British Medical Journal*, vol. 308, pp. 1113-1114.
- World Health Organization, Regional Office for Europe (1985). *Targets for health for all*. Targets in support for the European strategy for health for all. Copenhagen.

### **XIII. THE MEASUREMENT OF HEALTH STATUS IN THE UNITED STATES**

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#### **A. INTRODUCTION**

The mission of the National Center for Health Statistics (NCHS) is to provide statistical information that will guide actions and policies to improve the health of the American people. As the Nation's principal health statistics agency, NCHS designs, develops and maintains more than a dozen data systems that cover the full spectrum of health concerns. NCHS data systems can be used to profile the health of Americans, to track demographic shifts and trends, to monitor change in health and health care, to show relationships between risk factors and health outcomes, and to provide information for prevention, evaluation, research, planning and policy (United States of America, Department of Health and Human Services (US DHHS), US NCHS, 1997; US DHHS, 89-1325). The definition and measurement of health status is central to the fulfilment of the Center's mission. While NCHS is the only Federal agency whose sole mission is the collection, analysis and dissemination of health data, the measurement and analysis of health status occurs throughout the Federal government, in academia and in the private sector. NCHS's collection systems should be viewed as providing a core set of information on health. Approaches to the measurement of health status adopted by other parts of the research community will vary from those taken by NCHS depending on the specific objectives of each project.

NCHS views health status as multi-dimensional, involving all aspects of health and health care. This complex construct requires multiple indicators and multiple methodologies for adequate measurement. NCHS data collection programs are designed to capture the multi-

ple aspects of health status using a variety of approaches. When taken together, the results of these data collection activities provide a more comprehensive and complete assessment of health status than would be possible from any single strategy. For example, the National Health and Nutrition Examination Survey provides objective data on health status that complements the self-reported information on the impact of ill health on health care utilisation and social functioning provided by the National Health Interview Study. Data from the National Health Care Surveys, a family of provider based surveys, enhance the data produced by the population-based studies by focusing on supply and use of health care services. NCHS data systems are also designed to be as efficient as possible. Whenever possible, data collection strategies should result in measures that can be used for many purposes. For example, measures of health status should be constructed so that they can be used in epidemiologic analyses of risk factors as well as for monitoring trends.

The indicators of health status included in NCHS data systems can be divided into two major interrelated categories: those relating to disease incidence and prevalence and those relating to functioning (physical, cognitive, emotional and social). All NCHS data systems are currently undergoing re-evaluation and restructuring to assure that the most appropriate measures are being collected, using state-of-the-art methodologies.

#### **B. NCHS DATA SYSTEMS**

The Center operates two major types of data systems: those based on populations, containing data collected through interviews or examinations; and those based on records, containing data collected from vital and medical records (US DHHS, 89-1325). Efforts are underway to make methodologies as comparable as possible

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both within and across the data systems. However since each of the data systems are designed to fill unique data needs, important differences remain. The measurement of health status in each data system will be described within the context of that particular system.

### *1. Population-Based Surveys*

#### *National Health Interview Survey*

The National Health Interview Survey (NHIS) is a principal source of information of the health of the civilian non-institutionalised U.S. population (US NCHS Vital Stat, 1975; Kovar and Poe, 1985; US DHHS, 90-1302; US DHHS, 93-1307). The survey, conducted annually since 1957, currently collects information from approximately 40,000 households covering 100,000 people through in-person interviews. Topics include health status, functioning, access to care and insurance, health services utilisation, health behaviours and risk factors. The NHIS questionnaire underwent a major redesign in 1996 including moving from a paper and pencil to a Computer Assisted Personal Interview mode of administration. The survey consists of a set of core data items, the Basic Questionnaire, which is repeated each year. An additional set of items, administered periodically, obtains more in-depth information on topics covered in the Basic Questionnaire. Topical modules that can change each year address current health issues. The Basic Questionnaire itself is composed of two parts: the Family Core and the Person Core. The Family Core is administered family-style using a household respondent, as has always been the case for the NHIS. Health status information collected on the Family Core includes self-perceived health status and information on important but relatively low prevalence conditions, such as injuries. The Person Core collects information on a sample adult and a sample child, using self rather than proxy response for the sample adult. The Person Core is designed to obtain information on aspects of health status, such as symptomatology and functioning level, that can only be or are best obtained from self-response.

The NHIS collects information on health status in a variety of ways. Separate question-

naires are used for adults and children to assure that questions are age appropriate. Question batteries in the child's questionnaire are further targeted according to age. Information is obtained on perceived health status, i.e., the assessment of the subject's health status as excellent, very good, good, fair or poor. This measure has been shown to be highly correlated with other measures of health status and is predictive of mortality and admission to long term care facilities (US DHHS, Working Paper, 1994). Information is also obtained on selected conditions including heart disease, diabetes, cancer, pulmonary diseases, depression and arthritis. The approach used to obtain information on conditions changed significantly in 1996 when the new questionnaire was phased in. Respondents are first asked if they have ever been diagnosed with any of the conditions or if they have symptoms of the conditions. Depending on the condition, follow-up questions are asked on date of onset and use of medications or health care services. The final set of indicators of health status concern functioning. Information is obtained on limitation of activity (i.e. the subject's ability to perform age/sex appropriate roles such as working, going to school, keeping house), activities of daily living and physical functioning (e.g. walking, bending, and standing). Information is also obtained on the conditions causing the limitation. Coding to the International Classification of Disease is not attempted. The range of indicators of health status available from the NHIS is broad. Some examples include the proportion of the population in excellent or very good health, the proportion of the population unable to work or limited in the kind or amount of work they can do, the proportion of the population unable to perform the activities of daily living, the prevalence of diagnosed hypertension, the proportion unable to walk, and the proportion reporting depressive symptoms.

The decision to make a significant alteration in the way condition information is collected in the NHIS provides an insight into how health status is conceptualised. The NHIS had traditionally collected information on conditions in two ways. A condition list or a set of condition lists was used to determine if the respondent had been diagnosed with a condition. Over the years, the number of conditions of interest

increased dramatically so that a set of six lists was adopted. One approach was to rotate the lists each year. Another was to pose each list to one sixth of the sample. Both approaches introduced serious analytic limitations, especially for the study of comorbidity. Information on conditions was also obtained by asking questions about behaviours related to ill health such as whether the respondent had visited a physician, stayed in bed, cut down on usual activities or was limited in his/her ability to carry out age/sex appropriate roles. Information was then obtained on the conditions that caused the behaviour. Whenever a condition was mentioned, additional information was obtained on that condition to allow for coding according to the International Classification of Diseases. This approach reflects the concern of the original designers of the NHIS with the limitations of obtaining health information by interviewing the population. For a variety of reasons, interview studies are not good ways of obtaining information on specific diseases. However, interview studies provide unique and essential information on other aspects of health. Health status is much more than the aetiology and progression of pathologies; it is a social as well as a biological characteristic. Socio-demographic characteristics condition the perception of symptoms, the adoption of health related behaviours and the use of preventative and curative health care. How an individual perceives his/her health status in turn affects how the individual functions in the society in terms of employment, income and social relationships. The interrelationships between the biological and social aspects of health can best be obtained through interview surveys.

Based on the belief that direct questioning concerning disease states would not yield valid and useful information, the original NHIS was designed to obtain information on health status indirectly, as a function of what people did as a result of perceived health problems. The NHIS questionnaire was structured around obtaining information on the use of medical care, limitation in one's ability to fulfil social roles and restriction of activity. Most of the information on disease states used to make prevalence estimates was ascertained through questions about what caused the individual to seek medical care, and/or restrict or limit activity. Focusing on

what one does because of ill health as opposed to the nature of one's health has the advantage of providing a somewhat more concrete referent for the questions being asked. This results in some improvement in data validity and reliability. It also has the effect of limiting the definition of health problems to those that cause some behaviour such as seeking care or restricting activity. This approach is quite appropriate when ill health is primarily the result of acute conditions where onset is clear and there is a recognised association between the condition and the behaviour. However, as a result of the "epidemiologic transition", acute conditions have given way to chronic conditions as the major sources of morbidity. The greater difficulty in diagnosing chronic conditions and the longer period spent in the disease state both prior to and after diagnosis make the traditional approach used by NHIS more problematic. The collection of information on symptoms takes on greater importance when investigating chronic conditions that may or may not have been diagnosed but which affect an individual's ability to function in the society.

Another limitation of the traditional approach results from the conditioning of an individual's response to health problems by his/her social characteristics. This makes it extremely difficult to investigate social correlates of health status. For example, the issue of differentials in health status by socio-economic status is of great importance. However, the differential in the prevalence of conditions across socio-economic (SES) groups as determined by the NHIS is not as great as would be expected from other health indicators. This is in part due to the fact that, holding symptomatology and disease status constant, persons of lower SES are not able to stay home from work, to limit their activities or to seek medical care. Their social and economic situation does not provide them with the access to sick leave or to easily accessible or affordable medical care. Since they do not demonstrate the required behaviours, their health conditions are less likely to be counted in the NHIS.

The traditional approach also produces event and condition data rather than information that allows one to characterise individuals. The medical or health condition, the doctor visit and

the hospital stay, rather than the individual, are the unit of analysis. NHIS counted the number of conditions rather than people with conditions. The structuring of the NHIS data into separate visit stay and condition files was a result of this approach.

In light of these limitations, a decision was made to expand the collection of information on health status beyond that which is linked to the behavioural sequels of health status. In the redesigned questionnaire, less reliance is placed on event data and more on characterising individuals. While there was no disagreement with the basic premise that objective, clinical health information could not be obtained by the NHIS, it was felt that more information on symptoms and functioning could be obtained in a standardised, unbiased way improving our ability to characterise the health status of individuals.

Moving away from the approach where health information is obtained indirectly through information on behaviours resulting from ill health affects the kind of data that is produced as well as the way the questionnaire can be structured. Organising the questionnaire around medical care, restricted activity and limitation in functioning and the conditions that cause them provides data on the full range of conditions in the population related to these outcomes. However, the collection of detailed information on each condition takes up a considerable amount of interview time; the validity of this information has been questioned and the detailed information has not been widely used in analyses. Modifying this approach has allowed for a change in the structure of the questionnaire and has provided the flexibility to add additional content areas of interest.

During the past few years, the NHIS has become a major part of the Department of Health and Human Services' plan to improve the quality, efficiency, and timeliness of data by integrating surveys. Because of the integration plan, it was essential that the NHIS be redesigned to put more emphasis on an individual's health characteristics, such as disability, and less emphasis on estimating the number of conditions or medical events occurring in the population. The data collected will be more useful for disease and risk factor surveillance

and will be better able to address emerging health issues.

#### *National Health and Nutrition Examination Survey*

Unlike any other national survey, the National Health and Examination Survey (NHANES) is based on detailed physical examinations, including the collection of blood and urine samples in addition to in-depth interview information (Engel and others, 1978; McDowell, Engel, Massey, 1981; US NCHS, Vital Stat; 1985; US DHHS, 94-1308). A nationally representative random sample of the US civilian, non-institutionalised population is interviewed at home and then invited to visit a mobile examination center (MEC) for a standardised physical examination including a physician's exam, physical measurements, blood and urine tests and nutritional assessments. The MECs are sets of four connected tractor-trailers, which travel around the country. In order to examine approximately 5,000 subjects a year, two sets of trailers are in operation at any given time and a third set is in transit. Each set of trailers is staffed with medical personnel and each exam takes approximately four hours. The goals of the survey are:

- To estimate the number and percent of persons in the U.S. population and designated subgroups with selected diseases and risk factors.
- To monitor trends in the prevalence, treatment and control of selected diseases.
- To investigate risk factors for selected diseases.
- To monitor trends in risk behaviours and environmental exposures.
- To study the relationship between diet, nutrition and health.
- To explore emerging public health issues.

Seven NHANES have been conducted since 1960. Three of these studies, the National Health Examination Surveys, were conducted in the 1960s. Beginning in 1970 a large nutrition component was added to the basic design, and the name was changed to NHANES. A special study of Hispanic populations in the United

States was conducted in 1982-1984. The most recently completed survey was the NHANES III, 1988-1994. More than 40,000 persons ages 2 months of age and older were selected for the NHANES III. Young children, seniors, African Americans and Mexican Americans were sampled in large numbers so that precise estimates could be made of their health status and risk factors. Selected topics covered by this survey include osteoporosis, iron deficiency anaemia, dietary consumption, overweight, high blood cholesterol, hypertension, hepatitis B and C, and HIV.

The NHANES program provides the objective measures of health status that cannot be provided by the NHIS. Given the logistic complications and high costs of fielding a survey such as NHANES, sample sizes are considerably smaller than those obtained from the NHIS. The surveys are now being more closely coordinated so that analytic capabilities can be enhanced. The NHANES is also moving to continuous rather than periodic administration. Each year's sample will be representative of the U.S. population. While the sample size in any given year will be insufficient to make any but the most basic estimates, the continuous fielding of the study will increase flexibility by allowing data to be added across years and by facilitating survey content changes.

A variety of measures of health status are available from NHANES. Self-reported information, similar to that collected on the NHIS, on self-perceived health status, disease symptomatology and physician diagnosis, and level of functioning is obtained from the in-home or MEC interviews. In addition, the results of the physical exam and blood and other tests can be used to measure health status. While the interview portion of the NHANES is based on that used in the NHIS, modifications are made so that the interview information relates directly to the exam components. The objective information is used in conjunction with the self-report to describe an individual's health status. For example, information on diagnosed diabetes is obtained on the interview and information on undiagnosed diabetes is obtained from blood tests. This information on disease status is used to make prevalence estimates for the total population and for subgroups of interest. In-

formation on functional limitations related to diabetes (e.g. vision and mobility problems) coupled with similar information available from the other components of the NHANES allows for a detailed characterisation of an individual's health status. The information is also used for the investigation of disease risk factors and for monitoring of functional outcomes.

### *Longitudinal components*

When appropriate, NCHS is attempting to transform its cross-sectional surveys into longitudinal data collection systems. The NHIS is now routinely linked to the National Death Index allowing for the identification of decedents and their date and cause of death. Subsets of the NHIS sample have also been actively followed to obtain more information on change in health status. For example, the Longitudinal Survey on Ageing followed elderly respondents from the 1984 NHIS for 6 years and a second cohort of the elderly selected from the 1994 NHIS is currently being followed (US DHHS, 92-1304). Mortality follow-up is also done for the NHANES surveys. A more extensive follow-up was conducted for NHANES I, the NHANES I Epidemiologic Follow-up Study (US DHHS, 92-1303). The NHANES I cohort (1971-1975) has been recontacted in 1982-1984, 1986 (elderly only), 1987 and 1992. Hospital records for the cohort have also been obtained. These longitudinal activities present extensive opportunities for understanding the relationship between risk factors and disease outcome as well as the natural history of disease.

## *2. Record-based surveys*

### *National Health Care Survey*

The National Health Care Survey is an integrated family of surveys that monitor the use and content of medical care provided in the United States. The surveys collect data on the characteristics of providers and patients as well as on services provided, diagnoses, payment sources and outcomes. This family of surveys is the source of a wide range of data on the health care field. It represents a significant resource for tracking the changes in health care use, the impact of medical technology and the

quality of care being provided to the American population. Samples of providers are selected from frames that cover various aspects of the health care system. Information is then collected on characteristics of the provider. A sample of contacts with that provider is drawn and information obtained on the specifics of that contact. The content of the information obtained varies from provider to provider depending on the nature of the contact. Given the rapid change that characterises the health care system in the U.S., it is essential that the National Health Care Survey frame expand to include the full range of providers. Information is currently collected from six provider types.

- The National Hospital Discharge Survey is the principal source of information on inpatient utilisation of hospitals. It has been conducted annually since 1965 and 274,000 records from 525 hospitals are currently abstracted (US DHHS, 95-1782).
- The National Ambulatory Medical Care Survey provides data on visits to physicians. The survey was conducted annually from 1974 - 81, in 1985 and annually since 1989. The sample includes 3,000 physicians and information is collected on 40,000 visits (US DHHS, 96-1250).
- The National Hospital Ambulatory Medical Care Survey focuses on hospital emergency departments and outpatient departments. Annual data collection began in 1992. Data are abstracted from 70,000 medical records for visits to 440 hospitals (US DHHS, 94-1310).
- The National Survey of Ambulatory Surgery provides data on the use of free standing and hospital based ambulatory surgery centers. The survey was initiated in 1994.

The long-term care component of the health care system is captured by the National Nursing Home Survey and the National Survey of Home and Hospice Care (US DHHS, 97-1250; US DHHS, 94-1309).

- The National Nursing Home Survey has been conducted periodically since 1963 and most recently in 1995 when the sample included 1,500 facilities.

- The National Home and Hospice Care Survey provides data on home health agencies and hospices and was started in 1992. The sample includes approximately 1,500 agencies.

The components of the National Health Care Survey are used to enhance and expand measures of the prevalence of disease. Population based surveys such as the NHIS and provider based surveys such as the National Health Care Surveys are each designed to obtain information to describe a particular portion of a complex construct. However, there is considerable overlap in the topics of interest. Disease prevalence is one such area. Neither the provider nor the patient can provide a true description of prevalence but the two sources of information taken together give a more accurate picture than either taken separately. The Health Care Surveys offer an independent assessment of disease prevalence by approaching the problem from the provider side rather than the patient side. Information is produced on the number of contacts with the health care system, measures of length of stay, surgical and diagnostic procedures, and medications used in treatment.

The Health Care Surveys are particularly important since they obtain information on rare conditions that would not be picked up in population based surveys. They also provide larger sample sizes than would be obtained in surveys such as the NHIS and the NHANES. Condition information is obtained from the medical records and coded according to the International Classification of Disease. Information is collected on all conditions for which health care is obtained. However, the nature of the information available is limited to that which would routinely be obtained from records. In addition, the event (for example, the discharge) rather than the person is the unit of analysis. For example, while it is possible to estimate the number of hospital stays associated with hip fracture, it is not possible to estimate the number of persons with hip fracture even if one could assume that all hip fractures were admitted to the hospital. A proposed enhancement to the Health Care Surveys is to follow patients after the health care encounter.



The National Vital Statistics System is the basis for the Nation's official statistics on births, deaths, marriages, and divorces (US DHHS, 96-1120). The data are provided to NCHS through vital registration systems maintained and operated by the individual States and territories by co-operative agreements. Mortality data, compiled from information reported on death certificates, include demographic characteristics of the decedent, and underlying and multiple causes of death. Natality data include data on demographic and health characteristics of the mother and characteristics of the birth. Data on foetal deaths are compiled from reports of all foetal deaths of 20 weeks or more. Demographic and health characteristics of the mother and foetal death collected are similar to those for natality, but also include conditions causing death. Other data files maintained within this system include the Linked File of Live Birth and Infant Deaths.

Since the National Vital Statistics System provides information on all events, it allows for the most detailed analysis of population subgroups defined by socio-demographic characteristics and geographic area. The mortality statistics system provides basic health status information on death rates, cause of death (coded according to the International Classification of Diseases) and life expectancy. The National Mortality Followback Survey expands on the information on the death certificate to facilitate the investigation of the effects of lifestyle on health and the extent of the burden of illness in the last year of life. Information is obtained from an informant about the socio-economic characteristics of the deceased person, the use of and payment for hospitals and institutional care in the last year of life and factors related to health status. The most recent survey was conducted based on 1993 deaths. Information from the natality system provides basic health status information on infants and their mothers. The National Maternal and Infant Health Survey, last conducted in 1988, expanded on information available from birth, foetal death, and infant death records through contacts with mothers and health care providers to study factors related to poor pregnancy outcomes.

### C. OVERALL MEASURES OF HEALTH STATUS

NCHS's data collection systems are not designed with a primary objective of providing overall or composite health status measures. The data systems must be able to provide data to monitor all aspects of health status and to be able to relate these various aspects to each other as well as to health care utilisation. Composite measures that do not allow for the collection of information needed to characterise each component of health must compete with multi-purpose measures for scarce time in national data collection systems. For example, it is not sufficient to collect information on a general measure such as "ability to get around" when more detailed information is needed on whether that difficulty is due to cognitive problems or lower extremity impairments. The more detailed information would be related to the results of objective tests and specific risk factors or used to plan targeted intervention programs. However, there also are uses where overall measures are necessary. Cost-benefit and cost-effectiveness analyses are examples of activities that use overall measures of health status and interest in this kind of analysis for policy purposes is growing.

There is also increasing interest in developing basic health status indicators comparable to the economic indicators currently in use. These indicators would be used to monitor the effects of public policy and to identify areas where interventions are needed. An overall health measure is very appealing in this context. One example of this is the Department of Health and Human Services' strategy for improving the health of Americans by the end of the century. *Healthy People 2000: National Health Promotion and Disease Prevention* contains 500 measurable objectives and sub-objectives grouped into 22 priority areas. Increasing the span of healthy life is one of the three broad goals of Healthy People 2000. The Years of Healthy Life measure, an overall health status measure that includes both mortality and morbidity, was selected to monitor progress toward this goal (US DHHS, 95-1237). The selection of this measure was constrained by availability of data for the entire time period 1990-2000. Measures of activity limitation and perceived

health status from the National Health Interview Survey were used to create a measure of health-related quality of life. This measure was then used to adjust life expectancy estimates from the National Vital Statistics System to calculate the Years of Healthy Life measure.

### *1. Uses at the state and local level*

A limited set of indicators or an overall indicator is also important for monitoring health at the state and local level. Most health planning occurs at the local level, but it is at this level that data are most limited. There is also a great deal of variability in the availability of data below the national level. While the cost of data collection makes it unlikely that more complex collection systems can be developed in all state and local areas, a basic core of data should be available to inform planning and policy development. There is also increasing interest in the use of such indicators as performance measures. The need for data to support state and local efforts is a major component of the Healthy People 2000 objectives. One of the 22 priority areas concerns Surveillance and Data Systems and addresses the development of an infrastructure to track the objectives and to identify emerging public health issues at the national, state and local levels. To a large extent, the indicators used to measure progress in Healthy People 2000 objectives at the national and state level were selected on the basis of data availability as opposed to scientific rigor. Efforts are underway to develop data systems that can meet the planning and monitoring needs inherent in the Healthy People 2000 program.

There are clear advantages to linking the data collected at the state and local level to that collected at the national level. NCHS is attempting to do this by redesigning the sample of the National Health Interview Survey (NHIS) so that more estimates at the state level can be made. NCHS is also developing a telephone survey, the State and Local Area Integrated Telephone Survey (SLAITS), which can produce estimates at the state and local area for selected components of the NHIS. Similarly, plans are underway to develop a new component of the National Health and Nutrition Examination Survey (NHANES). This component will use smaller vehicles, such as recreational

vehicles, to collect subsets of the NHANES survey at the local level or on selected demographic groups that are difficult to include in the national survey. While replicating national surveys at the state and local level has advantages for comparison across units, there is also a need to respond to the individual data needs of each locality.

The need for basic measures of health status in the U.S. has been intensified by the devolution of responsibility for major social programs from the Federal to the state level. While program development and implementation is taking place on the state level, there remains a concern for program accountability and performance measures at both the national and state level. The process of developing mechanisms to demonstrate program outcomes is just beginning. There is some disagreement as to whether overall measures should be used or if the measures should be more specific to the program. For example, should programs designed to increase access to health care for children be judged on whether overall health status measures improve, on whether access indicators improve or whether some measured aspect of quality of care, such as immunisation, improves. If the option focusing on specific indicators is chosen, it would be necessary to select a small subset of indicators, since it would not be possible to monitor all outcomes. There is fear that this will make "gaming the system" more likely. On the other hand, measures that are more global might not be sensitive enough to the improvements associated with a given program. It is likely that a combination of indicators at different levels will be chosen, with the global measures providing the context for the specific indicators.

### *2. Derivation of overall measures from NCHS data systems*

Although NCHS's data systems have not been designed with the goal of producing an overall health status measure, it is possible to create measures from the data provided or to select one measure to act as the overall measure. Life expectancy at various ages and self-perceived health status each has a long history of being used as an overall measure of health status (US DHHS, Working Paper, 1994). Both have

limitations for this purpose. While mortality might be the ultimate indicator, it deals with only one of the extremes of health status. Many attempts have been made to use a measure of morbidity or health status to transform life expectancy into "healthy" life expectancy. Examples include Disability Free Life Years, Healthy Life Expectancy, Disability Adjusted Life Years and Years of Healthy Life. Constructs such as Years of Healthy Life combine measures of the different aspects of health into a single number using a conceptual model that views health as a continuum. The health states along the continuum are assigned numbers that represent the values that either society as a whole or individuals place on that health state. Various methods have been used to determine the values of the different health states. Examples of such measures are EuroQOL, the HUI-I and the Quality of Well-Being Scale. These measures are used to modify duration of life (US DHHS, 95-1237). The measure used to monitor the Year 2000 objective on healthy life as discussed above is another example. While the questions needed to produce data for calculating years of healthy life measures using existing methods have not been adopted for regular use in NCHS surveys, analogues of these can and have been created (Erickson and others, 1988; Erickson and others, 1989; Erickson and others, 1993).

Using self-perceived health status as the overall indicator is attractive since it relies on the individual's own methods of summarising his/her health status rather than on an external measure. However, this measure has limitations when used to monitor change over time, one of the primary uses of health status indicators. The means by which individuals evaluate the various aspects of health have been shown to be affected by contextual parameters. Implicit in self-perceived health status is the individual's evaluation of his/her health status against some unstated standard. Societal norms act to define the standard but the norms, and the standards, change in response to a variety of conditions. Thus, observed changes in the indicator may not reflect changes in underlying health status.

This problem affects many measures of health status, particularly those associated with functioning or limitations of activities. The less

explicit the referent standard, the more likely that the response will be affected by external factors. No explicit standard is provided when asking subjects if they are limited in the kind or amount of work they can do. Responses to this question will be affected by the kind of work that the individual thinks he or she should be doing as defined by their own expectations as well as by more objective states of health. The reporting of limitation of activity has been shown to be affected by changes in such things as the criteria used by government agencies in determining eligibility for disability benefits (Wilson and Drury, 1984).

This paradox is less of a problem if one is interested in monitoring the impact of health on work, as was the primary interest of the original designers of the National Health Interview Survey. However, it is much more of a problem if the aim is to understand and monitor a more objective measure of health. Other measures of health status, such as the prevalence of diagnosed conditions, are also subject to some of the same kinds of problems. Increased access to health care should result in improved health status and a decline in the prevalence of disease. In the short run, however, the opposite occurs. As the population has more access to health care, undiagnosed conditions become diagnosed, leading to an increase in prevalence. Self-perceived health status and functioning may also decline as awareness of health problems increases. The interpretation of seemingly straightforward measures of health status are often not straightforward (Wilson and Drury, 1984).

Despite the difficulties in measurement, a major aspect of health status involves the individual's ability to function physically, cognitively, emotionally and socially. Any overall indicator of health status will have to include functioning, in some way. Methodological work continues to be needed in the basic measurement of functioning. Related to the issues mentioned above is the notion that functioning occurs at the intersection of innate capabilities, the environment, and modifications that have or could be made to either of the components. Measures of functioning need to be explicit in identifying what is being measured. For some objectives, it is appropriate to measure innate

capability even if functioning has or can be changed through modification of the environment. For other objectives, it is necessary to take the environment as given and describe current functioning within that environment.

#### D. FUTURE PLANS

NCHS continues to conduct methodological work to improve our ability to measure health status. While there are no plans to make any significant changes in our basic approach to measurement, modification and restructuring of each measurement component is always under consideration. The measurement of perceived health status will continue to be included in the population-based survey. Major efforts will also be directed to determining disease prevalence from both the population based data systems and the provider surveys. However, more attention is being paid to ascertaining disease severity. In the past, the number of health conditions was often used as an indicator of health status. While comorbidity is important and more work needs to be done in this area, it is equally, if not more important, to measure the severity of the disease. Severity is highly related to, but not synonymous, with functional limitation resulting from the disease state. Disease severity relates to prognosis, which might be unrelated to current functional status. However, advances in the measurement of functional ability will improve the ascertainment of disease severity. NCHS data systems will continue to obtain information of the various aspects of functioning. Methodological work needs to be conducted in this area.

The overall objective is to design a set of data systems that can respond to data needs in a variety of areas. In addition to providing overall measures of health status, information must be available on specific aspects of health and on their inter-relationships. While each of the NCHS data systems is designed for specific objectives and focuses on different aspects of the complex maze encompassed by the term health, fostering greater integration between these systems will enhance the overall understanding of health. This approach also allows for incorporating multiple data sources into the measurement systems. When appropriate and

with adequate protection of subject confidentiality, linkages across data sets are being fostered. For example, linking population-based surveys such as the NHIS with mortality data or administrative health records enhances data quality and analytic capabilities at relatively minimal cost. Statistical linkage is also being explored as a way to enhance analytic potential when direct linkage is not possible. Even as we develop systems that reflect the complexities of health, it will also be necessary to develop a limited set of indicators that can be used not only to monitor health status but to plan, develop and evaluate programs at the Federal, state and local level.

#### REFERENCES

- Engel, A., R. S. Murphy, K. Maurer and E. Collins (1978). Plan and Operation of the Hanes I Augmentation Survey of Adults 25-74 Years, United States, 1974-75. National Center for Health Statistics. *Vital and Health Statistics*, vol. 1, No. 14.
- Erickson, P., E. A. Kendall, M. P. Odle and G. W. Torrance (1993). Assessing Health-related Quality of Life in the NHANES I Epidemiologic Followup Survey. NCHS Working Paper Series.
- Erickson, P., E. A. Kendall, J. P. Anderson and R. M. Kaplan (1989). Using Composite Health Status Measures to assess the nation's Health. *Medical Care*, vol. 27, No. 3, pp. 566-77.
- Erickson, P., J. P. Anderson, E. A. Kendall, R. M. Kaplan and T. Ganiats (1988). Using Retrospective Data for Measuring Quality of Life: National Health Interview Survey Data and the Quality of Well-being Scale. *Quality of Life and Cardiovascular Care*, vol. 4, No. 4, pp. 179-84.
- Kovar, M. G., and G. S. Poe (1985). The National Health Interview Survey Design, 1973-84, and procedures, 1975-83. *Vital and Health Statistics*, vol. 1, No. 18.
- McDowell, A., A. Engel, J. T. Massey and K. Maurer (1981). Plan and Operation of the Second National Health and Nutrition Examination Survey, 1976-80. National Center for Health Statistics. *Vital and Health Statistics*, vol. 1, No. 15.
- United States of America, Department of Health and Human Services (US DHHS) (89-1325). Data Systems of the National Center for Health Statistics. United States of America, Department of Health and Human Services publication No. (PHS) 89-1325, Series 1, No. 23.
- \_\_\_\_\_. (90-1302). Questionnaires from the National Health Interview Survey, 1980-84. United States of America, Department of Health and Human Services Publication No. (PHS) 90-1302, Series 1, No. 24.
- \_\_\_\_\_. (92-1303). Plan and Operation of the NHANES I Epidemiologic Followup Study, 1987. United States of America, Department of Health and Human Services Publication No. (PHS) 92-1303, Series 1, No. 27.
- \_\_\_\_\_. (92-1304). The Longitudinal Study of Ageing: 1984-90. United States of America, Department of Health and Human Services Publication No. (PHS) 92-1304, Series 1, No. 28.

- \_\_\_\_\_. (93-1307). Questionnaires from the National Health Interview Survey, 1985-89. United States of America, Department of Health and Human Services Publication No. (PHS) 93-1307, Series 1, No. 31.
- \_\_\_\_\_. (94-1308). Plan and operation of the third National Health and Nutrition Examination Survey, 1988-94. United States of America, Department of Health and Human Services Publication No. (PHS) 94-1308, Series 1, No. 32.
- \_\_\_\_\_. (94-1309). Development of the National home and Hospice Care Survey. United States of America, Department of Health and Human Services Publication No. (PHS) 94-1309, Series 1, No. 33.
- \_\_\_\_\_. (94-1310). Plan and Operation of the National Hospital Ambulatory Medical Care Survey. United States of America, Department of Health and Human Services Publication No. (PHS) 94-1310, Series 1, No. 34.
- \_\_\_\_\_. (95-1237). Years of Healthy Life. Series 1, No. 23. Data Systems of the National Center for Health Statistics. United States of America, Department of Health and Human Services Publication No. (PHS) 95-1237, Statistical Notes.
- \_\_\_\_\_. (95-1782). National Hospital Discharge Survey: Annual Summary, 1993. United States of America, Department of Health and Human Services Publication No. (PHS) 95-1782, Series 1, No. 121.
- \_\_\_\_\_. (96-1120). Births and Deaths: United States, 1995. United States of America, Department of Health and Human Services Publication No. (PHS) 96-1120, Vol. 45, No. 3, Supplement 2.
- \_\_\_\_\_. (96-1250). National Ambulatory Medical Care Survey: 1994 Summary. United States of America, Department of Health and Human Services Publication No. (PHS) 96-1250, Series 1, No. 273.
- \_\_\_\_\_. (97-1250). An Overview of Nursing Homes and their Current Residents: Data from the 1995 National Nursing Home Survey. United States of America, Department of Health and Human Services Publication No. (PHS) 97-1250, No. 280, Advance Data.
- \_\_\_\_\_. Working Paper (1994). Proceedings of the 1993 NCHS Conference on the Cognitive Aspects of Self-Reported Health Status. Hyattsville, MD: United States of America, Department of Health and Human Services. Cognitive Methods Staff Working Paper Series No. 10 (1994).
- United States National Center for Health Statistics (NCHS) (1997). Organization and Activities. Hyattsville, MD: United States of America, Department of Health and Human Services, National Center for Health Statistics. Unpublished manuscript.
- \_\_\_\_\_. Vital Stat (1975). Health Interview Survey Procedures, 1957-1974. National Center for Health Statistics, *Vital and Health Statistics*, vol. 1, No. 11.
- \_\_\_\_\_. (1985). Plan and Operation of the Hispanic Health and Nutrition Examination Survey, 1982-84. National Center for Health Statistics, *Vital and Health Statistics*, vol. 1, No. 19.
- Wilson, Ronald W., and Thomas F. Drury (1984). Interpreting Trends in Illness and Disability: Health Statistics and Health Status. *Annual Review of Public Health*, vol. 5, pp. 83-106.

# XIV. THE LIMITS OF LONGEVITY AND THEIR IMPLICATIONS FOR HEALTH AND MORTALITY IN DEVELOPED COUNTRIES

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## A. INTRODUCTION

There is considerable confusion about the precise definitions of the concepts of longevity, its limits, life span, the maximum life span, life expectancy, and limits to life expectancy, because of their different uses by the various scientific and clinical disciplines including demography, gerontology, geriatrics, epidemiology, biostatistics, biology, and actuarial science (Manton, 1996). This confusion has made even more difficult the efforts to empirically determine an intrinsic limit to the human life span, the effects of those limits on the rate of population ageing, and the limits to future population life expectancy increases.

Life expectancy is the average age at death for a cohort that is only directly calculable after the last person in a cohort has died. Since the complete observation of the survival experience of a recent human birth cohort in the United States will require 125 or more years (e.g., Social Security Administration, 1992; Manton and others, 1996), usually an estimate is made from the observed risk of death (i.e., the mortality rate) for specific ages – often for deaths occurring at specific ages over a given, short period of time (e.g., one or three years; the latter to minimise isolated period effects such as influenza cycles).

If life expectancy is increasing across birth cohorts, then cross sectional, or “period”, life expectancy estimates may be significantly downwardly biased from the true “life expectancy” for the current birth cohort (Manton and others, 1997). Cohort life expectancy, on the other hand, is limited as an indicator of human longevity in that it does not represent health and environmental conditions as they currently exist – but rather as they existed over a wide range of past times and historical conditions. The early mortality experience of

a current oldest-old cohort (i.e., one aged 85 or more years) may reflect social and health conditions about the end of the nineteenth, and the beginning of the twentieth century (Fogel, 1994).

Thus, because of their temporal confounding of age, period, and cohort factors, neither period nor cohort life tables, nor the life expectancies calculated from them, are completely satisfactory for all analytic purposes for mortality data. Each one has limitations when trying to infer the limit to the human life span. As discussed below, other types of analytic models, and longitudinal data sets with more extensive health measures on individuals measured at multiple time points, are needed to investigate these issues and to make better estimates.

Life expectancy is the average value of a “realised” or observed variable (or an estimate of the mean of the distribution of ages at death derived from a period life table). In contrast, longevity and the life span, especially viewed as a biologically determined “intrinsic” limit to the length of life, are theoretical quantities—distributed biological traits of the population being studied (Pearl, 1925). In its purest sense, the limit to human longevity would be represented by the highest age at death observable in a human population living in a life-long “ideal” environment (Manton and others, 1991).

Definition of the “ideal” environment is a critical problem not often fully appreciated in studying human longevity. No human population, especially one composed of persons currently, e.g., age 75 or above, has ever experienced anything approximating an “ideal” environment – let alone lived in an ideal environment over their entire life time. Indeed, as we shall discuss below, the concept of an “ideal” environment is at best a theoretical construct for human populations because those populations are biologically very heterogeneous. Depending upon the collection of

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physiological traits of the individuals in a given population, different individuals may express their intrinsic maximum life span in different environments. The ideal environment can only be defined relative to the intrinsic physiological state of an individual. Furthermore, as we will show, an individual's state can vary considerably over time.

In animal experiments, an ideal environment may be more closely "approximated" (Carey and others, 1992; Curtsinger and others, 1992; Johnson and Lithgow, 1992). However, even in well-controlled animal and insect experiments questions can be raised, about the changing density of an experimental population as the individual insects or animals die off, the oxidation of foods, the longevity effects of controlled variables such as temperature. This is because while an environmental variable may be strictly controlled at a given level we do not know *a priori* if that level is the "optimal" one for observing the maximum longevity.

Thus, using only demographic data and analytic concepts, we have to infer longevity, the biological endowment controlling individual life spans, from an imperfect and incomplete set of demographic measures – the highest observed ages at death and, for the population, life expectancy. Another critical factor often missing in such discussions is that there may be other statistical moments of the completed population age at death distribution that are important in characterising longevity, i.e., there may be intrinsic heterogeneity in the biological endowment for longevity, so that there is an irreducible variance (and possibly significant higher order moments) of the "ideal" age at death distribution. Indeed, it may be that the ideal environment that produces the highest average life span may not produce the maximum heterogeneity (or life span variance) – thus the highest individual theoretical life span may not be expressed in conditions that maximise the average life span in a human population with a given distribution of physiological traits.

For example, the Japanese population has one of the highest life expectancies at birth in the world for a developed country, 77 years for males and 83 years for females. The highest estimate for a politically defined population aggregate is for the Chinese territory of

Hong Kong, 79 years for males and 86 years for females (table A10; U.S. Bureau of the Census, 1996). In contrast, though United States life expectancy at birth is only 73 years for males and 80 years for females, the United States survival above age 80 is higher than in Japan and in a number of other developed countries with higher life expectancies at birth such as France and Sweden (Manton and Vaupel, 1995). This is likely due to the greater homogeneity of the Japanese population and their generally healthier diet, their heavy consumption of soy and plant products (Adlercreutz and others, 1994). In the United States of America the population is socially, economically, and ethnically more heterogeneous, factors that could have a greater impact on mortality at birth and the younger ages. By age 80 the less long-lived components of the very heterogeneous United States population will have tended to have died off, leaving a more select, long-lived population at ages 80 and above (e.g., Marenberg and others, 1994). In addition, the health care system in the United States has proven to provide a much better mix and quality of health services to very old persons; in Japan a specialised long-term care system is relatively undeveloped and many elderly do not get appropriate chronic therapeutic services but instead get caught in the acute care system (Ikegami and Campbell, 1995).

These issues raise a series of statistical problems for efforts to estimate parameters characterising longevity and its limits using only observed life expectancy and the realised distribution of ages at death. In the most general terms this is a standard statistical problem in the analysis of multivariate distributions, inferring a "true" state variable from multiple observed measures made with some degree of error. Indeed, the statistical issues involved in jointly assessing the mean and variance from a non-normal distribution of ages at death is the classical so-called "Behrens-Fisher" problem. The difficulty here is that there may be no empirical situation where the maximum theoretical value of the true state variable can be observed. For example, multiple measures could be made of mental abilities, and factor analysis used to identify the consistencies among them, to identify the construct of "intelligence" (or the component dimensions of intelligence). The problem is one of inferring the maximum value of the state variable,

whether it be intelligence or life span – neither of which may be observed. More precisely, we have no way to estimate the probability of the maximum observed age being the “true” or theoretical maximum age at death except when it is exceeded. One knows that then the probability of the prior observed age being the theoretical maximum age at death is 0.0.

#### B. MOLECULAR MECHANISMS RELATED TO LIFE EXPECTANCY AND LIFE SPAN DIFFERENCES

As a consequence, extensive ancillary biological data are needed to constrain the statistical problem in order to make a plausible extrapolation from the distribution of observed life span values to the maximum life span for an individual, or to predict the maximum life expectancy for a human population. This ancillary data may help us understand why the age trajectory of mortality rates ceases to follow standard hazard functions, such as the Gompertz, at advanced ages. For example, what biological mechanism might explain the observation of actuaries that there is no credible evidence of an annual mortality rate for a human population exceeding 50 per cent (Society of Actuaries, 1994). Indeed, in one actuarial study of 11 insured populations, mortality rates above age 95 for both males and females averaged 25 per cent per annum (Manton, 1996). In a series of studies of centenarian populations, mortality rates from 100 to 110 increased between 0 and 3 per cent per annum (Manton and Stallard, 1996) – compared to the 5 to 6 per cent assumed above age 100 in Social Security Administration projections (Social Security Administration, 1997).

##### 1. *Fixed genetic effects on life span*

Efforts have been made to estimate the maximum life expectancy by theoretically discriminating between endogenous and exogenous “causes of death” (Bourgeois-Pichat, 1952 and 1978). The age of death from endogenous causes of death are presumably “intrinsic”—fixed in the genetic code of the individual—while the age at death for exogenous causes are presumably controlled by the environment. It is usually assumed that we currently do not have the knowledge to alter ge-

netic determinants of longevity (Olshansky and others, 1990; Wilmoth, 1997).

One biological model relating a fixed genetic endowment to longevity potential was originally due to Hayflick (1965). He suggested that mammalian cells were limited by genetic factors to replicating a maximum of 40 to 50 times. After the biologically endowed maximum number of cell replications was exceeded, the cell would presumably enter a senescent state and die.

Efforts to confirm this model have not met with complete success. Martin and others (1970) cultured fibroblasts from persons aged 30 to 80 to see how many replications were “used” per year of life over that age range. The experiment showed that, on average, one replication was used for every five years of life – suggesting that the theoretical life span limit for 40 to 50 replications for this cell type (human fibroblasts) might be 200 to 250 years. Of course, the situation has to be more complex in nature since the human organism is composed of many different cell types, and different types of tissue have different potential numbers of cellular replications—and times between replication. For example, cells in the central nervous system are thought to replicate little after maturity. Hemopoietic stem cells in the bone marrow have a much greater potential for replication.

Added to the basic notions of the Hayflick limit were recent results on the telomere of cells (Bacchetti, 1996). This is the terminal part of the chromosomal material that is thought to be involved in assuring that the genetic code is faithfully maintained through cell division. It was proposed that the telomere might be the basic molecular mechanism explaining the Hayflick limit. However, new results suggested that the biological mechanisms involved are more complicated. First, programmed cell death was argued to be intrinsic to preserving function as the organism aged (Warner and others, 1995). Programmed cell death, or apoptosis, was found to be largely regulated by the p53 gene – the so-called “guardian of the genome”. This gene, when operational, either halted cell division or caused a controlled death in cells where serious mutational defects in DNA had occurred (Harris, 1996a and 1996b). Thus, the p53



gene could cause cells to die before the telomere reached the length that would not allow cells to faithfully reproduce their genetic content. Another recently discovered gene potentially controlling replication is the WRN (Warner's Syndrome gene) that appears to affect the production of a helicase, i.e., an enzyme involved in the winding and unwinding of DNA strands.

Further complicating the issue was that in certain tissues telomerase, an enzyme, was expressed that could allow reconstruction of the telomere. Though not thought to be generally present in somatic cells, it may be expressed at low levels in bone marrow stem cells (Bacchetti, 1996). The gene for the production of telomerase is likely to exist in a quiescent state in most somatic cells since, in many types of neoplasia, the cell lines become immortal. Their immortality is due in part to the induction of the production of telomerase and, in part, due to the failure of the p53 gene to control the replication of cells with serious mutations by apoptosis.

The role of telomerase in delaying cell senescence was experimentally demonstrated in cell tissue culture using new techniques that allow telomerase activity to be "switched on" in otherwise inactive normal cells. The results were telomere lengthening, continued cell division beyond normal limits, and continued maintenance of normal karyotype and youthful morphology – all of which provide evidence for a causal relationship between telomere shortening and cellular senescence (Bodnar and others, 1998).

Thus, not only is there complexity in the way different tissues express limits to growth, there are multiple mechanisms affecting cell replication, and those mechanisms could change in expression due to mutations and other stochastic events. Furthermore, since some cells can function without replicating for quite a long period of time, and some cells thought not to replicate in adults (e.g., in the central nervous system) were recently found to express stem cells and to be capable of slow cell replication, linking such mechanisms to the number of cell replications does not seem sufficient to explain the biological complexity and stochasticity of human life span control. Thus, there is a need to examine, in greater detail, the molecular mechanics of cell growth

and function preservation and the relation of those mechanisms to overall organism survival and longevity.

### C. REGULATORY AND PROTECTIVE MECHANISMS INFLUENCING LIFE SPAN

#### 1. *Endogenous versus exogenous causes of death*

The arguments in the previous sections suggest that simply dividing causes of death into endogenous and exogenous sources is too simplistic a biological model to explain the human life span, i.e., the distinction is a "false" one for biologically complex, multi-cellular organisms like humans (Olshansky and others, 1990). This is because, through evolution, different sets of cells have become biologically specialised reflecting the fact that, in multi-cellular organisms, not all cells have direct contact with the external environment. Though lacking such direct environmental contact, all cells still have basic energy, respiration, regulatory, and other biological needs that still must be met. Thus, the endogenous state of the organism is the new, artificial "environmental" image purposively provided for those cells by the biological structure of the organism, to replace direct contact with the external world. It reflects an internal representation of the outside world, as presented to the organism, to specialised sets of cell systems (i.e., "tissues").

As such the internal representation of the external world must accurately reflect changes in conditions in that outside world so that a co-ordinated response of multiple tissue systems can be brought to bear to preserve the overall organism in its rapidly changing environment. This "signaling" function is accomplished by complex sets of hormonal factors that co-ordinate the functioning of the different tissue systems. This also suggests that the operating principle governing the survival of the organism may be more complex than the oft cited principle of "homeostasis", i.e., the maintenance of the current physiological state of the organism (e.g., Strehler and Mildvan, 1960; Sacher and Trucco, 1962). The problem with the principle of homeostasis is that, due to the biological tissue complexity of multiple organ systems, there are temporal lags in the

hormonal communication to the different tissue systems, even of major changes in the environment.

## 2. *Hormesis*

The existence of such lag functions in complex organisms suggests that the crucial system operational characteristic is actually that of "Hormesis" (Stebbing, 1987). Hormesis, which operates in most multi-cellular organisms, reflects the need for a time lag in complex systems with negative feedback to respond to environmental stresses. Because of the time lags in response between tissue systems, the organism can not make instantaneous, continuously graded responses to external stresses. The lagged response of the complex organism will always be in response to a temporally prior state of the environment. Thus, the cells in the organism must somehow "anticipate" or "project" the future state of the environment. They can do that either by responding in fixed quanta, or by ensuring that the tissue response to the external challenge is always an over-compensation, so that the likelihood that the response made is sufficient to adjust to the "current" environmental stress is maximised.

This also means that, in response to most environmental stresses, excess energy will be expended. If the organism survives, that energy is used to make micro or macro-structural changes that increase the "fitness" of the organism to meet even greater environmental stresses in the longer term (Calow, 1982). This need to anticipate adverse future environmental changes has the consequence that the hazard function used to relate human mortality to "risk factors", to be biologically valid, must be a U or J-shaped quadratic function with, or without, variable rescaling (Yashin and Manton, 1997).

A fundamental question justifying the quadratic hazard function is how the internal physiological changes in fitness are biologically manifested. At a macro-level, changes in fitness in response to environmental factors are relatively easy to identify and measure. Familiar examples are increased muscle mass as a response to physical activity demands; increased immune responses to infections; increases in pulmonary capacity in response to greater oxygen demand; increased hematocrit

to lower oxygen concentrations at high altitudes; increased bone density to weight bearing activity.

Of interest is the fact that many prior estimates of the age changes in the ability to increase or retain fitness (e.g., Shock, 1984) have been significantly upgraded as a result of improvements in the methodology and design of ageing studies. Specifically, in the past there was an attempt to identify representative samples of populations for studies of human ageing. However, such representative samples reflect the effects of the particular environmental conditions that produce a specific prevalence of chronic health problems in the population. The age trajectories of functional loss in those study populations were thus "contaminated" by environmental differences in exposure to chronic disease risks (Lakatta, 1985).

In the past 10 to 15 years there has been considerable effort to screen out persons with manifest chronic diseases from older study populations. In the studies of elderly persons with no manifest chronic diseases, the rate of loss of specific types of biological function is much slower than previously observed. However, they still probably do not reflect the theoretical lower limit to the age rate of specific functional losses because of the presence of latent preclinical disease. For example, in elderly persons who remain physically active the loss of cardiovascular function was half the rate estimated in prior population studies (Kasch and others, 1993). Some physiological functions have shown almost no decline over the age ranges examined. Other functions show considerable capacity to improve even at advanced ages, voluntary muscles show the capacity to greatly increase in strength due to resistance training, even in frail institutionalised persons aged 90 or more (Fiatarone and others, 1990, 1993, 1994). Other studies show that, by appropriate interventions (e.g., vitamin E supplementation) some age related physiological functions (e.g., select types of immunological responses) can be improved in elderly persons (Meydani and others, 1997).

## 3. *Oxidative stress and caloric restriction*

A common theory about strategies to increase longevity by altering the basic rate of ageing is that of caloric restriction. In a num-

ber of recent animal studies, It has been found that caloric restriction increases the life span of animals over that of control rats fed ad libitum diets. Furthermore, in certain murine experiments, caloric restriction showed a dose-response effect on survival. The question that emerges from those experimental observations is what plausible physiological mechanism produces the increase in survival.

The most frequent explanation, observed from a series of studies of mechanisms of ageing, is that the increased longevity resulting from caloric restriction is due to a decrease in oxidative stress. This is partly based on the observation that genes controlling anti-oxidative enzymes (e.g., glutathione or super-oxide dismutase) in select insects and nematode studies were associated with a doubling or tripling of life span (Weindruch and Sohal, 1997).

However, the epidemiological evidence for caloric restriction producing a slowing of physiological ageing changes, and thus increased survival in humans is not consistent. This may be due to the greater biological complexity of humans multiple factors control oxidative products. Consequently, the simple monitoring of body mass and caloric intake is not sufficient to explain human life expectancy changes. We consider three more fundamental physiological mechanisms that may be more directly involved in controlling human life span.

#### 4. *Heat shock proteins*

The molecular and genetic dimensions of physiological fitness are just beginning to be explored at the micro-level (Perera, 1996). In particular, there are cellular structures and physiology that increase in capacity in response to external stresses (Stebbing, 1987; Calow, 1982). One of the evolutionary most "primitive" of these adaptive physiological mechanisms is the production of the several classes of so-called heat shock proteins (HSP) (Welch, 1992). These proteins were so named because they were first identified as adaptive responses to thermal stress on the cell. What the heat shock proteins do is to insure that new proteins being produced by the cell do not become "denatured" by stress; they prevent the topological shape or "folding" of the new protein molecule from being altered in the

face of a thermic or other shock. Proteins undergoing construction in the cell are far more subject to denaturation by heat shock than already formed proteins. Heat shock proteins are found in single cell organisms as well as in humans. Indeed, there is a high degree of biological conservation of the structure of heat shock proteins, even for organisms as organisationally disparate as microbes and humans (Roma and Catapano, 1996).

Heat shock proteins are now often more generally referred to as "stress" proteins in that similar cellular protein conserving mechanisms turn out to prevent denaturing of newly produced proteins against many forms of stress including oxidation, heavy metals, ischemia, bacterial toxin infections (Roma and Catapano, 1996). Stress proteins have been subdivided by some authors into HSP and GRP (glucose-regulated proteins). The GRP showed increased expression in cells starved of glucose and also apparently in cells with disrupted calcium homeostasis, hypoxia, or agents that interfere with protein secretion.

There are several families of heat shock proteins, each characterised by their molecular weight (in kilodaltons). HSP8 is active in removing damaged proteins after the shock to the cell. HSP28 can increase 10 to 20 fold in mammalian cells after a shock. It appears to correlate with the growth status of the cell and may regulate the actin cytoskeleton, helping preserve the cell's structure integrity. HSP56 is part of the steroid hormone receptor. HSP90 is one of the most abundant proteins in mammalian cells. It appears to regulate the activity of a number of proteins including the steroid receptor. GRP94 is related to HSP90 and may regulate the passage of proteins through the endoplasmic reticulum. HSP110 may be necessary for the development of cellular thermoresistance.

The two most important families of heat shock proteins in humans performing roles in the production and protection of proteins are HSP60 and HSP70. HSP60 is found in the mitochondria and operates as a molecular "chaperone" of newly formed proteins there because it appears necessary to facilitate the correct folding of proteins into their mature structure. HSP60 dysfunction may play a major role in atherosclerosis (Wick and others, 1995) and immunological disease (Welch,

1992) because it has a high degree of homology to a heat shock protein (HSP65) found in bacteria. Thus, the immune system may mistakenly respond to the HSP60 and cause a major inflammatory autoimmune response that is often a crucial phase in the development of atherosclerotic plaques as well as in other autoimmune diseases.

Proteins in the HSP70 family are found in both the cell nucleus and cell cytoplasm. HSP73 is the constitutively expressed form of the protein. It appears to play a major role in the early stages of protein construction. It also may play a role in forming stable combinations with mature proteins that have become de-natured. Indeed, the cellular store of HSP70 may be depleted as it is coupled with abnormally folded mature proteins. Thus, though more HSP70 may be synthesised, it appears that the utilisation of HSP70 in forming complexes with denatured proteins may be involved in the process of cellular ageing – as various stresses cause more denatured mature proteins the ageing of the cells (indexed by the ability to generate HSP70) may be accelerated.

The HSP72 protein is the primary HSP70 family member produced in an inducible form and is a particularly simple protein expressing no introns – and, as a consequence, not requiring RNA in replication so that it can be produced more rapidly in response to stressful conditions on the cell. HSP72 may be produced to a greater degree than required by environmental stress in that its induction must be restricted by a negative feedback, as required by the principle of Hormesis. This may produce a greater response to stress than called for by the original exposure, so that the production of HSP72 overshoots the exact amount of the protein needed. Thus, the inducible heat shock proteins, after being produced, confer a level of resistance that preserves the cell in the face of some future shocks that before the induction would have sufficed to kill the cell. In addition, since the heat shock proteins function to conserve proteins at such a basic level, the cell is protected against stresses of other types, a phenomenon called cross-tolerance.

This HSP mechanism has expressions at the macro-level. It has been found, for example, that organisms exposed to thermic stress in-

ducing HSP72 have a greater resistance to cellular ischemia and hypoxia (Bongrazio and others, 1994). Thus, a higher expression of HSP70 may be cardioprotective in myocardial infarctions (Radford and others, 1996). This may also explain why humans can chronically adapt to oxygen deprivation and tissue ischemia induced over a long period of time, such as in dialysis patients who, because of renal failure, typically have hematocrits 35 per cent below normal. Thus, an adverse environment can induce basic cellular changes adapting the internal environment at a molecular level so the cells have greater survival ability in future stress situations (Yellon and others, 1993). The ability to produce HSP72 appears to decline with age (Bongrazio and others, 1994). Thus, limitations in the early production of HSP72 due to an early "favourable" environment may actually handicap the organism's ability to survive at late ages.

The expression of HSP72 is also related to hypertension. This relation is important in that it directly links HSP to immune system responses. That is, it was shown that thermosensitivity identified a locus involved in genetically determined hypertension (Malo and others, 1988). The genes controlling the expression of HSP72 are located in the major histocompatibility complex suggesting a linkage to immunological factors (Wurst and others, 1989) as well as to attenuated HSP72 induction with age (Liu and others, 1989). It is of interest that in many cases, baseline values of physiological parameters do not change with ageing, but that when stressed an age related decrease in response is noted (Shock and others, 1984), such as the elderly's clinically significant reduced tolerance of hyperthermia (Wollner and Spalding, 1978).

### 5. P450 Enzymes

The HSP protein system is not the only component of human physiology that maintains cellular function. An external stress induces the production of specific enzymes by genetic factors also in the case of the cytochrome P450 enzyme system. Again, this is a basic physiological mechanism that evolved very early; the first enzymes were produced by a common gene ancestor that came into existence about 3.5 billion years ago (Nelson and others, 1996). The mechanism is highly biologically conserved across both plant and

animal species. The originating gene for this family of 482 genes and 22 pseudogenes existed before the divergence of prokaryotes and eukaryotes (Nelson and others, 1996) and even before oxygen was persistent in the Earth's atmosphere. The CYP51 family of genes has been found in mammals, fungi and yeasts, and plants. One CYP51 gene and two pseudogenes have been mapped to three different human chromosomes. The P450 enzymes are basic to the metabolism of steroids, bile acids, fatty acids, prostaglandins, leukotrienes, biogenic amines, retinoids, lipid hydroperoxides, and phytoalexins (Nelson and others, 1996). Thus, they are crucial to multiple dimensions of metabolism.

In addition, P450 enzymes are important drug metabolising enzymes (though they evolved long before there was such a thing as a "drug") and are responsible for detoxifying many environmental "pollutants" – both natural and manmade. If this were not so, then accelerated life testing of drug metabolism in animal models would not provide useful information on human metabolic responses. The evolution of the cytochrome P450 system was accelerated as animal and plants competed in a common environment. Plants developed toxins to prevent them from being consumed by animals. Changes in the cytochrome P450 system was the animal's response to the evolving nature of plant toxins (Gonzalez and Nebert, 1990).

There are two major classes of drug metabolising enzymes responsible for the detoxification and excretion of foreign chemicals. Many foreign compounds (e.g., drugs, plant metabolites, and environmental pollutants) are non-polar and dissolve in lipids. The drug metabolising enzymes convert these chemicals into water-soluble products that can then be excreted. This is done in two phases. In Phase I (functionalisation) a functional group is added to the substance. P450 enzymes are Phase I enzymes. Phase II (conjugation reactions) uses the functional group to add conjugates creating a water-soluble product. The P450 enzyme family is the most extensive set of drug metabolising enzymes. Furthermore, a P450 enzyme might affect multiple different chemicals and multiple P450 enzymes may work on a single chemical. Thus P450 can respond to a wide range of chemicals (Nebert, 1994).

Phase-I functionalisation often translates molecules into biologically active chemical forms. It is ironic that the metabolism of many organically inert molecules to biologically active forms in Phase I reactions allow them to play major roles in forming DNA adducts and in carcinogenesis (Gonzalez and Nebert, 1990). The second phase converts those biologically active forms into water-soluble compounds that can be excreted by the kidneys.

As for HSP, certain P450 enzymes are genetically determined and constitutive. Others, though their potential for production is genetically determined, are not expressed until a suitable chemical challenge is made by the environment. The rate of expression of these enzymes can vary across individuals by 300-fold or more (Perera, 1996). The ability to produce certain of the crucial P450 enzymes may also decline with age. For example, the production of many liver enzymes in this family involved in animal protein metabolism may begin to decline by the eightieth year.

Since drugs were not developed until recently, one might ask what are the basic purposes of P450 and other drug metabolising enzymes (DMEs). These enzymes regulate the sub-cellular steady state concentration of ligands important in cell growth and development (Nebert, 1994). That is, the fact that they metabolise exogenous chemicals is a function of chance – no enzyme exists which does not metabolise an endogenous substance. The endogenous chemicals cause increases in cell type specific division, apoptosis, altered cell differentiation, homeostasis and neuroendocrine functions. The DME enzymes are induced in response to effector signals – e.g., foreign chemicals mimicking endogenous growth effectors activate specific DME genes. Furthermore, it appears that animal-plant interactions affected growth and proliferation where plant products mimic endogenous cellular function effectors.

Thus, the DMEs reflect the fact that exogenous factors can mimic endogenous cell regulators and that DMEs developed to control the internal environment. However, they also respond to exogenous cell effector analogues and as a consequence, both endogenous and exogenous chemicals can stimulate the genetic regulation of the DMEs. Thus, in this

case the distinction of endogenous and exogenous physiological effectors is completely confounded at the cellular level. This suggests that we already possess a number of compounds that affect how genotype is expressed and, as a consequence, plays a major role in longevity. It is thus incorrect to argue that there are fixed genetic mechanisms that are currently not alterable by human intervention (Olshansky and others, 1990) – many alterable genetic mechanisms have already been identified. The problem is one of understanding the complex interplay of such interventions in increasing survival. This also gives us an interesting perspective on why many drugs are derived from plants.

### 6. *The immunological system*

A third important endogenous, physiological system that is affected or altered by environmental exposures is the immune system. This system is clearly one for which early exposure to modulated bacterial and viral pathogens produces a physiological adaptation suited to survival. Some antigenic responses are long lived. Others are short term.

It is now becoming clear that these responses have survival consequences far beyond those directly linked to the immediate consequences of acute infections. A number of chronic diseases have been linked to early exposures to pathogens. The most clearly linked is that between *H. pylori*, a water borne infection that is highly prevalent in countries without improved water systems, and gastric ulcer (Hosking and others, 1994). The pathogen was first clearly identified in 1983. It has caused a total revision of the clinical management and treatment of gastric ulcers. At one time, this treatment was palliative – H<sub>2</sub> blockers were used to suppress the production of stomach acid. Now it has been shown in a number of randomised clinical trials that a two-week course of antibiotics can eradicate the *H. pylori* infection and cure the ulcer in 90 per cent or more of cases. Also of interest is the relation of *H. pylori* to gastric cancer (Hansson and others, 1996; Parsonnett, 1996). Gastric cancer was the number one ranked cause of cancer related deaths in the United States in the 1930s. Now it has dropped to sixth – possibly because of improved water quality and the reduction of exposure to

*H. pylori* – possibly due to the increased use of antibiotics for many other infectious diseases.

This is but one chronic disease where a major etiological factor turned out to be exogenous (Mozar and others, 1990). There are many others – with more being discovered. There is currently an immunisation program against hepatitis B in 85 countries with the intent of eliminating chronic liver diseases and liver cancer (Zuckerman, 1997). In addition, CMV, herpes virus, and recently Chlamydia pneumoniae have been found to be related to atherosclerosis (Grayston, 1993). A number of other retro-viral agents have been hypothesised to be part of the aetiology for solid tumours (e.g., breast and oesophageal cancer) and are clearly involved in various types of leukaemia and lymphomas (e.g., Epstein-Barr virus). Epstein-Barr virus is estimated to be nearly 95 per cent prevalent in adult populations.

If the initiating agent for a chronic disease is a viral or bacterial infection this suggests that the risk of many chronic diseases may be reduced in a cost effective fashion by existing methodologies used against infectious agents (Capron, 1996). As in the cases of the P450 and HSP physiological systems, immune function, especially of the cell mediated type, may decline with age – though certain antioxidant vitamins (e.g., vitamin E; Meydani and others, 1997) may stimulate some components of the immune system to late ages. It is also clear that HSP, the P450 system, and immunological function interact. For example, females appear to be more susceptible to cigarette induced lung cancer (Zang and Wynder, 1996) due to gender differences in the P450 enzyme system; females more efficiently conduct phase I metabolism of the chemicals found in tobacco smoke (e.g., the CYP2D6 gene). Certain HSPs involve production of steroid hormones.

### D. IMPLICATIONS OF HORMESIS AND THE PHYSIOLOGICAL COMPLEXITY OF HUMAN SYSTEMS FOR LIMITS TO LIFE SPAN

We have reviewed three basic physiological systems that operate as endogenous interfaces of basic cellular functions to a wide variety of

biochemical and microbial environmental challenges. That review stresses four points in analysing the nature of limits to human longevity. First, it is clear that small challenges are intrinsic to the ability of many human systems to successfully adapt to the external environment. Thus, an ideal environment is not one without hazards but rather is one where challenges occur relatively early in life, and at a low level. Late age environmental exposures to, say, infectious disease, often prove to have more dire consequences (e.g., secular changes in the influenza virus). Second, this first point suggests that the lowest risk of death does not occur at zero exposure but at relatively low levels of exposure – but exposures significant enough to challenge the organism to produce cellular defences. Third, the representation of the risk of death from environmental challenges must be of high dimensionality because of the complexity of the systems involved. Fourth, the past exposure history of an organism becomes embedded in complex ways in the physiology of the organism. This means that the “state” of the organism must be described in a model to reflect the

current physiological consequences of those past exposures.

This leads us to the formulation of a multi-dimensional stochastic process to describe human ageing and mortality (Woodbury and Manton, 1977; Manton, 1997). This stochastic process has two interacting components. The first represents the dynamics of physiological state variables as an autoregressive process, i.e., one where the current state of the organism is partly determined by its past states. The second component is a quadratic hazard function that relates mortality to the current values of the state variables where the values of the state variables are predicted from the autoregressive process. Since the prediction of future state variable values is imperfect, the process is stochastic, i.e., the prediction of an individual's state variable values is uncertain.

The first component represents the state dynamics of individuals and might be represented as a  $J$ -dimensional auto-regressive process of order 1, e.g.,

$$x_{it} = u_0 + \beta_1 Age_{it-1} + \beta_2 x_{it-1} + \beta_3 (Age_{it-1} \cdot x_{it-1}) + \beta_4 y_{it-1} + \beta_5 Y_{it-1} x_{it-1} + e_{it}. \quad (1)$$

In this equation we can represent the dependence of an individual's state at time  $t$ , defined by the  $J$ -element vector,  $x_{it}$ ; or his age,  $Age_{it}$ ; his prior state,  $x_{it-1}$ ; his environment,  $y_{it-1}$ ; and his state-environment interactions,  $Y_{it-1} x_{it-1}$ ; where  $Y_{it-1}$  obtains from

$y_{it-1}$  by multiplying each element of  $y_{it-1}$  by a  $J \times J$  identity matrix and stacking the results to form  $Y_{it-1}$ .

The mortality of the individual is a sum of  $K$  cause-specific quadratic functions of his or her state, as determined by the “physiological history” of the individual in equation (1),

$$\mu(x_{it}, Age_{it}, \theta) = \sum_{k=1}^K \left( \mu_{0k} + b_k^T x_{it} + \frac{1}{2} x_{it}^T B_k x_{it} \right) e^{\theta_k Age_{it}}. \quad (2)$$

In equation (2) the  $T$  superscript reflects the transpose operation. The exponential term represents the average age-effect of unobserved state variables,  $K$  represents the number of distinct failure processes, and  $b_k$  and  $B_k$  reflect the linear and quadratic dependence of mortality of the  $k^{\text{th}}$  type on the state variables. The state variables represent both the intrinsic

(genetic) endowment for longevity, the dependence of longevity on the state of the person, and the interaction of the current environment with the state of the individual. In practice, the state variables are physiological parameters of the individual such as serum cholesterol, hematocrit, blood pressure, and blood glucose level. Some state variables are

readily measured and others such as the degree of resistance to blood flow are not. However, it is not necessary to measure all state variables, but only a systematic sampling of them sufficient to identify the temporal trajectory of the major dimensions of the system. The type of hazard function generated by equation (2) is presented in figure 93.

In figure 93 we show the dependence of female mortality on a risk factor at ages 50 and 90 for cancer, circulatory diseases, all other causes, and total mortality. For all four mortality functions, the risk of death increases far more rapidly as a function of the risk factors at age 90 than at age 50. The "low" central mortality implies that, for the risk factors selected, mortality is lowest at some intermediate value. The fact that the  $\theta$  varies by each cause indicates that latent risk factors have a stronger age related interaction with the observed risk factors for some causes, i.e., the latent factors associated with age have a greater effect per year of life on other causes (12.5 per cent) than for cancer (6.5 per cent). If one were to evaluate these curves at later ages (e.g., 110) they would be even steeper. Thus, the  $\theta$  indicates the probabilistic limit to survival induced by latent, age related variables. The estimates of  $\theta$  used in figure 93 were generated conditional on six risk factors (i.e., smoking, diastolic blood pressure, pulse pressure, serum cholesterol, BMI, and blood glucose) assessed in the 34-year follow-up of the Framingham Heart Study. How the estimates of  $\theta$  were determined is indicated in table 56.

For males and females four different types of cause specific quadratic functions were estimated. The first function was the standard Gompertz hazard. For males the Gompertz shape parameters was 10.7 per cent; for females 9.8 per cent. The  $\chi^2$  associated with the fit of that base model to the data are indicated, for each of four causes of death (cancer, cardio-vascular disease, other and total) and as risk factors are added in three steps. In the first step cigarette smoking is added. We see that the  $\theta$  for total mortality declines from 10.7 per cent to 10.1 per cent for males and from 9.8 per cent to 9.3 per cent for females when smoking is added. Adding in five more risk factors (i.e., BMI, serum cholesterol, diastolic blood pressure, pulse pressure, blood

glucose) reduces the  $\theta$  from 10.1 to 8.3 per cent for males and from 9.3 per cent to 8.3 per cent for females. Adding four more risk factors (vital capacity, heart rate, LVH, and hematocrit) decreases  $\theta$  only a small amount but significantly adds to the overall  $\chi^2$ . This suggests these latter four factors explain individual differences in risk that are not strongly age dependent.

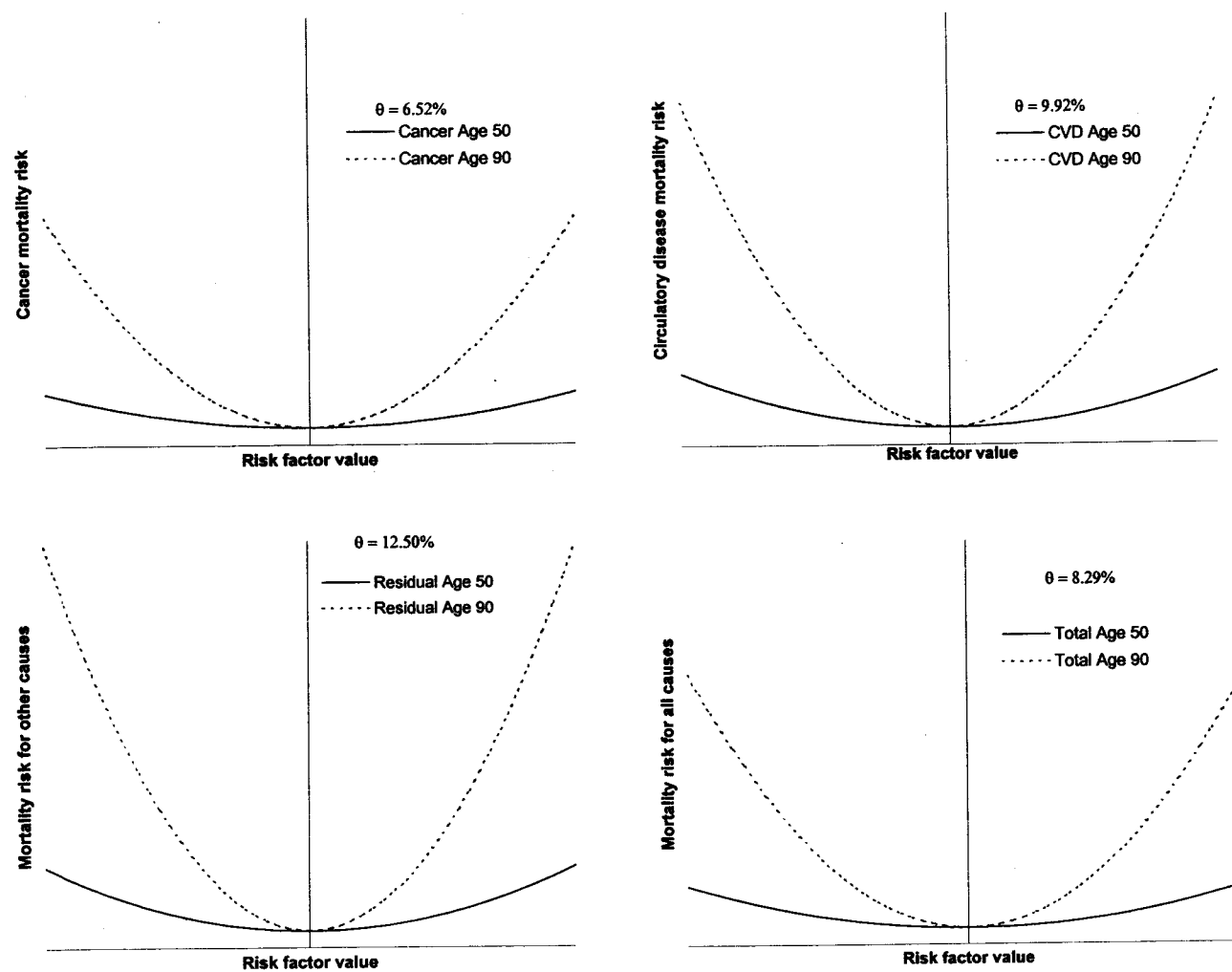
The equations reflect a stochastic limit to survival in the effects of  $e^{\theta_k \text{Age}_k}$  on mortality and in the structure of the auto-regressive process determining the age trajectory of state changes. This stochastic limit clearly varies strongly by the cause of death – even for chronic "endogenous" diseases (e.g., cancer,  $\theta_C = 6.52$  per cent; circulatory disease,  $\theta_{CD} = 9.92$  per cent).

One of the sources of stochasticity in the relations is the imprecision in measurements of the trajectories of state variables. This can be due to missing state variables or due to  $y_{it-1}$  being an exogenous process with stochasticity generated by independently operating mechanisms, e.g., ambient temperature. Thus, though  $\theta_k$  will decline as state variables and dynamics are better measured (see table 56), there will always be uncertainty in the endogenous state induced by chance environmental factors. The absolute values and percent changes in the  $\chi^2$  goodness-of-fit for total and cause specific mortality are given in table 56. There is a considerable increase, for both males and females, in the  $\chi^2$  goodness-of-fit of the model when entering risk factors, even taking into account age and cigarette smoking. This suggests that the risk factors tend to explain risk variation at each age better than they explain the age trajectory of latent variables, i.e.,  $\chi^2$  increases relatively more rapidly than  $\theta$  declines

Another way to examine the effects of the different model components is illustrated in figures 94a and 94b. These figures present age-specific life expectancies for different levels of smoking with the other risk factors controlled. For greater than 1 pack of cigarettes consumed per day, female survival drops faster (by more than 12 years at age 30) than for males (by less than 10 years at



Figure 93. Age dependence of cause specific and total mortality functions, model estimates



Source: See text.

TABLE 56. MODEL ESTIMATES FOR DIFFERENT RISK FACTOR SETS AND FOR CAUSE SPECIFIC AND TOTAL MORTALITY

	<i>Gompertz Parameter (<math>\theta</math>)</i>	<i>Per cent change in (<math>\theta</math>)</i>	<i>Total mortality <math>\chi^2</math></i>	<i>Per cent increase in <math>\chi^2</math></i>	<i>Cancer mortality <math>\chi^2</math></i>	<i>Per cent increase in <math>\chi^2</math></i>	<i>CVD mortality <math>\chi^2</math></i>	<i>Per cent increase in <math>\chi^2</math></i>	<i>Residual mortality <math>\chi^2</math></i>	<i>Per cent increase in <math>\chi^2</math></i>
<i>A. Males</i>										
Gompertz; Age Only	10.71		1 371		381		632		358	
Cigarettes + Age Only	10.12	-5.5	1 404	2.4	391	2.6	642	1.6	371	3.6
Six Risk Factors + Age	8.31	-17.9	1 706	21.5	415	6.1	780	21.5	511	37.7
Ten Risk Factors + Age	8.05	-3.1	2 175	27.5	503	21.2	1 034	32.6	638	24.9
<i>B. Females</i>										
Gompertz; Age Only	9.82		1 390		146		691		553	
Cigarettes + Age Only	9.26	-5.7	1 433	3.1	152	4.1	711	2.9	570	3.1
Six Risk Factors + Age	8.29	-10.5	1 680	17.2	187	23.0	854	20.1	639	12.1
Ten Risk Factors + Age	8.12	-2.1	2 048	21.9	218	16.6	1 073	25.6	756	18.3

*Source:* See text.

age 30). This is consistent with epidemiological data showing that the dose-response curve for smoking is steeper for females (Zang and Wynder, 1996; Ryberg and others, 1994; Risch and others, 1993). This is apparently due to sex differences in the inducibility of the P450 enzymes metabolising cigarette smoke chemicals into biologically active forms that can interact with DNA. That is, the inducibility of enzymes, which are under genetic control, can strongly influence the effect of an exogenous exposure factor on survival and this change can be illustrated at the population level.

The quadratic hazard function not only represents state uncertainty, but also the fact that the lowest mortality point will be a function of  $K$  failure processes, each influenced by  $J$  state variables whose changes are both endogenously and exogenously driven. The  $K$  individual cause-specific hazard functions may induce considerable uncertainty about longevity in that there may be a relatively large "flat" region over which the system may adapt successfully to changes in different state dimensions induced by environment over time.

This means that, not only will there be no unique limit to longevity, but that the currently observed maximum life span is a complex function of individual characteristics interacting with the environment over time. This suggests that there is a theoretical possibility of a very large longevity value – but that the multiplicity of factors operating will keep extreme theoretical limits to longevity from being reached with any realistic probability.

Clearly, since the factors limiting longevity are multidimensional, there are numerous ways in which incremental improvements/increases in life expectancy can be achieved – but, because of the competing effects of these factors, each increment in survival will tend to be small. There is a possibility that, if the  $K$  dimensions of failure are correlated, the production of a given number of improvements will lead, after a period of accumulation, to a large increase in life expectancy. Thus, because of the kinetics of the mechanisms involved, the rate at which life expectancy increases in a population is highly variable with slow increases punctuated by short periods of rapid gains during which

multiple correlated factors jointly change to produce a "surge" in life expectancy.

This model suggests that the more information available on both the physiological state of the individual, and his or her environment, the more precise the determination of the age at death distribution will be and the smaller  $\theta$  will be. For example, in table 56  $\theta$  declines from 10.7 per cent per annum for males to 8.0 per cent when 10 risk factors are added. If all relevant state variables were known then  $\theta = 0.0$  and mortality would be a function only of state variable dynamics. If the interaction terms ( $Y_{it-1} x_{it-1}$ ) in the auto-regressive process in (1) are significant this also suggests that the life span of individuals in the population cannot be determined without measures of specific environmental factors. Since environmental factors are impressed on the internal state of the organism, to the extent that the manifestation of genotype can be modulated, the maximum life span can be altered by interventions in the environment which alter the phenotypic expression of genotype at a molecular level.

To illustrate exactly how environment can affect genotype one needs to consider the molecular machinery involved in the expression of genotypes. Genotype cannot be manifested without transcription processes that allow the gene to be expressed in new proteins, or without induction of a signal to the gene produced by activating receptors in the cell membrane. There are a variety of ways that signaling and transcription can be affected. There is also the possibility of altering the genetic material directly by inducing mutation or creating adducts to the DNA. These more direct manipulations of genes are now being examined in clinical trials as potential therapies for cancer (Roth and Cristiano, 1997).

## E. CONCLUSION

The prior material allows us to make a number of specific conclusions about life span and life expectancy and the estimation of limits to those quantities.

Because of the complexity of the systems involved we cannot expect the limits to longevity to be deterministically fixed. As more

Figure 94. Life expectancy, model estimates

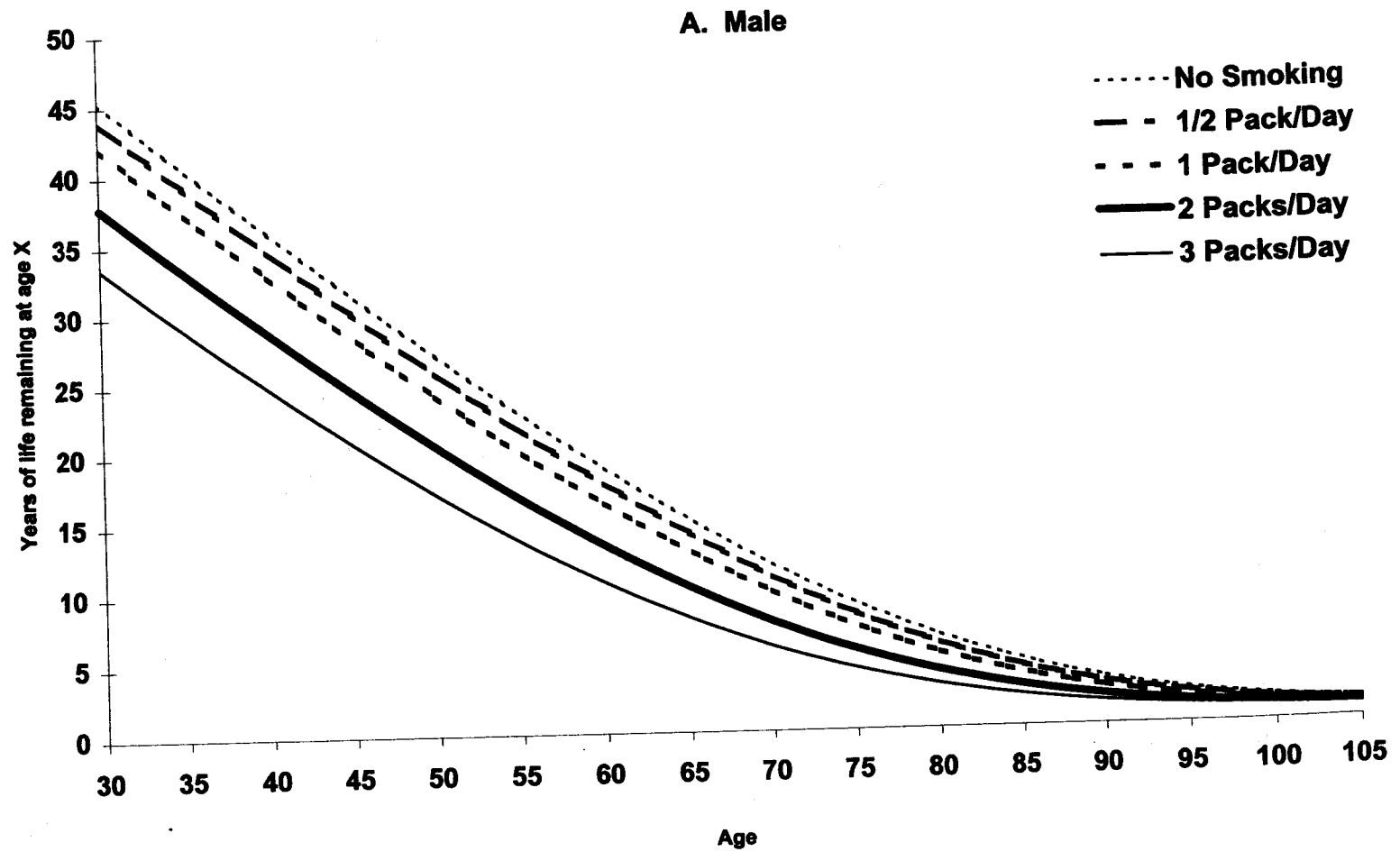
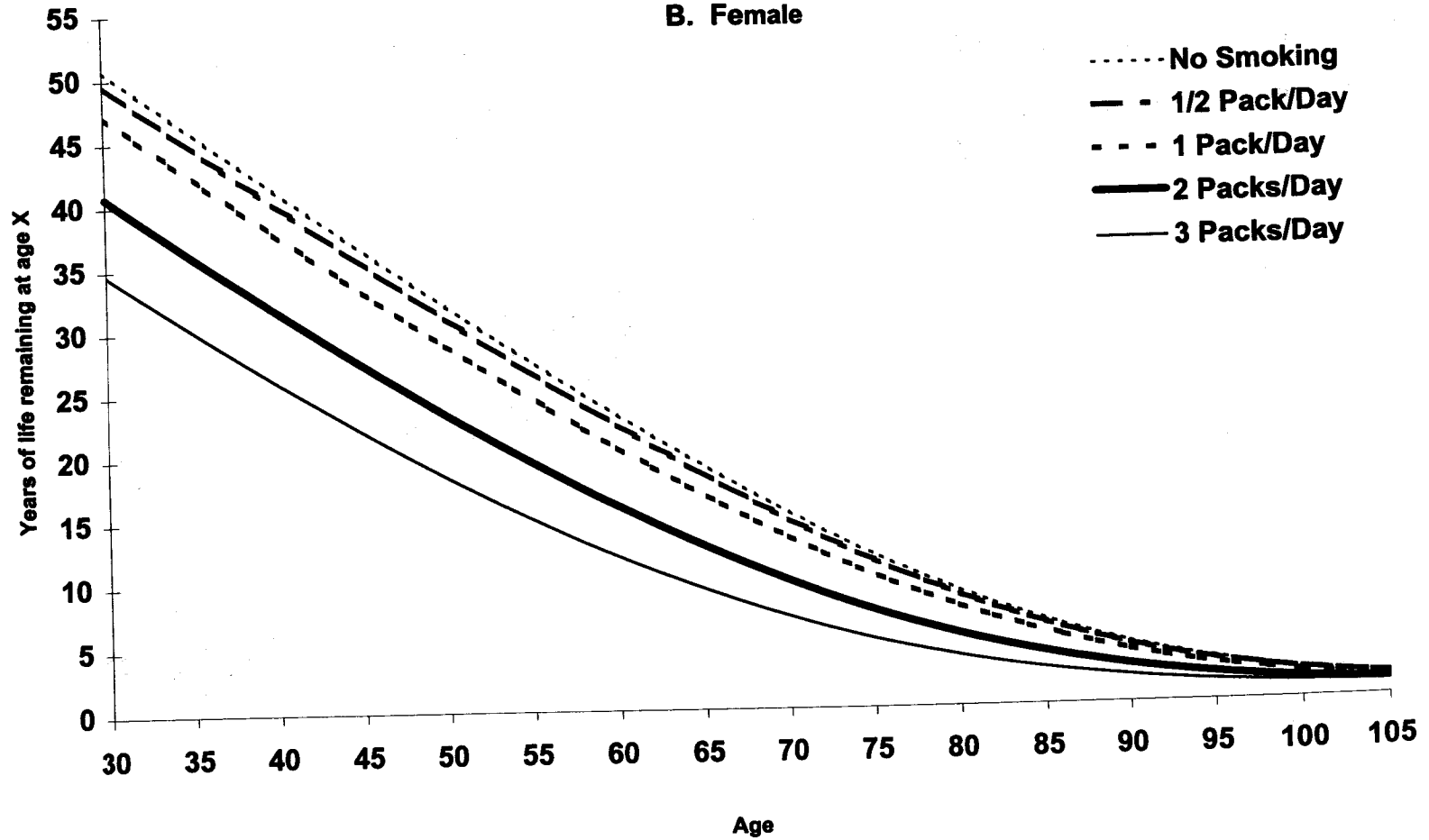


Figure 94 (continued)

B. Female



Source: See text.

persons survive to late ages the likelihood of an individual surviving to a greater age increases stochastically (Manton and Stallard, 1996).

It is also evident that biomedical science is beginning to unveil the basic cell mechanisms regulating longevity. These genetic mechanisms are alterable by appropriate molecular interventions. This is a result of the process of evolution by which they developed, in response to the effects of the changing environment on the cell's internal state. Thus, limits inferred today are likely to be altered in the future, both by molecular interventions and, possibly, by altering the genetic code; there are 101 clinical trials in progress testing different methods for altering the growth regulation of a cell in order to treat cancers (Roth and Cristiano, 1997).

Though the current limits to life expectancy have not been reached in any contemporary population, estimates of "current" limits with existing technology may be produced. To do so one must deal with data on the physiological state of individuals followed over time. One cannot infer changes in life span and longevity solely from age at death, the realised variables that are determined by past conditions. Instead, an understanding of changes in risk factors and functional status long prior to death are necessary to forecast future changes in life expectancy. This requires models of both the age trajectories of multiple state variables and functions describing the likelihood of failure conditional upon those trajectories. To the extent that "longevity" limits are reached, these will be limits determined by societal and economic constraints and conditional on the provision of health care.

The effects of changes in longevity on population age structure will be profound. This is because life expectancy, and the median age at death, are close to the ages that we use to define elderly (65) and oldest-old (85) populations and for which persons in many developed countries become entitled to pensions and national health services – including specialised services for the elderly.

As a consequence the derivative of the probability of survival is changing rapidly indicating that, as large birth cohorts pass those ages, large proportions of the population

may be "elderly". For example, in a Japanese study with an assumed limit to life expectancy of about 80 years, roughly 25 per cent of the Japanese population was projected to be aged 65+ by 2024 (Nihon University, 1982). If life expectancies of 95+ years are achieved in the next one to two hundred years with a standard deviation of ages at death roughly 5 years (the biologically "absolute" longevity might be 130 years, i.e., the maximum is 7 standard deviations above the mean), then it would be possible for one-third or more of the population to be aged 65 and over.

Any changes in longevity will be paralleled by changes in health. The diseases that cause most deaths from ages 50 to 70 are different than those causing death from ages 75 to 95. For example, the traits of heart disease change with age. Deaths from heart disease before age 15 are likely due to congenital anomalies. For deaths from 16 to 35 there are likely genetic predisposing factors to some type of accelerated atherosclerotic process or due to myocardial nerve conduction defects. From age 36 to 60 the classical stress related heart attack due to smoking, stress response, and arterial vasospasms kills many males prematurely. From ages 61 to 79 atherosclerotic heart disease becomes increasingly significant. From ages 80 to 94 persons have often already survived prior heart attacks so that degenerative processes affecting cardiac pump efficiency become significant. Above age 95 it appears that amyloid protein deposition, and other types of restrictive cardiomyopathies, may be significant (Jacobson and others, 1997). Similarly, for stroke, deaths up to age 30 or so are likely due to a circulatory defect (e.g., an AVM). At ages 31 to 49 haemorrhagic stroke may be more prevalent. For ages 50 to 74, hypertension and diabetes may be significant contributors to thromboembolic strokes. Above age 75, atherosclerosis in the cerebrovascular circulation and isolated systolic hypertension (due to decreased circulatory vasodilation capacity) may be important causes of stroke. Thus, for a person to survive to extreme ages he must be able to survive through the early expressions of multiple different chronic diseases.

If there is no rigid limit to life expectancy, and new biomedical technologies are emerging that increases our capacity to intervene in the molecular and genetic basis of disease,

then the prudent course may be to only estimate the changes in life expectancy expected to occur in the near future. A conservative estimate is that life expectancy at age 65 will increase by one year per decade. This suggests that, for the United States, in a hundred years male life expectancy at age 65 will increase from about 15.6 to 25.6 years and female life expectancy from 19.0 to 29.0 years. Such a rate is slower than was found in many scientific analyses (e.g., Lee and Carter, 1992) but faster than assumed in official United States forecasts. The Social Security Administration's intermediate alternative assumes about a three year increase in life expectancy in 75 years (Social Security Administration, 1997). The one year per decade gain assumes no major breakthrough in biomedical technology. Over the course of a hundred years, some breakthrough is a distinct possibility. In this case the increase, for example, might be 1.5 years per decade.

#### REFERENCES

- Adlercreutz, H., S. L. Gorbach, B. R. Goldin, M. N. Woods, J. T. Dwyer, and E. Hamalainen (1994). Oestrogen metabolism and excretion in Oriental and Caucasian women. *Journal of the National Cancer Institute* (Bethesda, MD), vol. 86, No. 14, pp. 1076-1082.
- Bacchetti, S. (1996). Telomere dynamics and telomerase activity in cell senescence and cancer. *Seminars in Cell & Developmental Biology* (Orlando, FL), vol. 7, pp. 31-39.
- Bodnar, A. G., M. Ouellette, M. Frolkis, S. E. Holt, C. P. Chiu, G. B. Morin, C. B. Harley, J. W. Shay, S. Lichtsteiner and E. W. Wright (1998). Extension of Life-Span by Introduction of Telomerase into Normal Human Cells. *Science* (Washington, D. C.), vol. 279, pp. 349-352.
- Bongrazio, M., L. Comini, G. Gaia, T. Bachetti and R. Ferrari (1994). Hypertension, ageing, and myocardial synthesis of heat-shock protein 72. *Hypertension* (Baltimore, MD), vol. 24, pp. 620-624.
- Bourgeois-Pichat, J. (1952). Essai sur la mortalité "biologique" de l'homme. *Population* (Paris), vol. 7, pp. 381-394.
- \_\_\_\_\_. (1978). Future outlook for mortality declines in the world. *Population Bulletin of the United Nations* (New York), No. 11, pp. 12-41.
- Calow, P. (1982). Homeostasis and fitness. *American Naturalist* (Chicago, IL), vol. 120, pp. 416-419.
- Capron, L. (1996). Chlamydia in coronary plaques—hidden culprit or harmless hobo? *Nature Medicine* (New York, NY), vol. 2, No. 8, pp. 856-857.
- Carey, J. R., P. Lliedo, D. Orozco and J. W. Vaupel (1992). Slowing of mortality rates at older ages in large medfly cohorts. *Science* (Washington, D. C.), vol. 258, pp. 457-460.
- Curtsinger, J. W., H. H. Fukui, D. R. Townsend and J. W. Vaupel (1992). Demography of genotypes: Failure of the limited-life span paradigm in *Drosophila melanogaster*. *Science* (Washington, D. C.), vol. 258, pp. 461-463.
- Fiatarone, M. A., E. C. Marks, N. D. Ryan, C. N. Meredith, L. A. Lipsitz and W. J. Evans (1990). High-intensity strength training in nonagenarians. *Journal of the American Medical Association* (Chicago, IL), vol. 263, pp. 3029-3034.
- Fiatarone, M. A., E. F. O'Neill, N. Doyle, K. M. Clements, S. B. Roberts, J. J. Kehayias, L. A. Lipsitz and W. J. Evans (1993). The Boston FICSIT study: The effects of resistance training and nutritional supplementation on physical frailty in the oldest old. *Journal of the American Geriatrics Society* (Washington, D. C.), vol. 41, No. 3, pp. 333-337.
- Fiatarone, M. A., E. F. O'Neill, N. D. Ryan, K. M. Clements, G. R. Solares, M. E. Nelson, S. B. Roberts, J. J. Kehayias, L. A. Lipsitz and W. J. Evans (1994). Exercise training and nutritional supplementation for physical frailty in very elderly people. *New England Journal of Medicine* (Boston, MA), vol. 330, No. 25, pp. 1769-1775.
- Fogel, R. W. (1994). Economic growth, population theory, and physiology: The bearing of long-term processes on the making of economic policy. *American Economic Review* (Nashville, TN), vol. 84, No. 3, pp. 369-395.
- Gonzalez, F. J., and D. W. Nebert (1990). Evolution of the P450 gene superfamily: animal-plant 'welfare' molecular drive, and human genetic differences in drug oxidation. *Trends in Genetics* (New York, NY), vol. 6, pp. 182-186.
- Grayston, J. T. (1993). Chlamydia in atherosclerosis. *Circulation* (Baltimore, MD), vol. 87, No. 4, pp. 1408-1409.
- Hansson, L. E., O. Nyren, A. W. Hsing, R. Bergstrom, S. Josefsson, W. H. Chow, J. F. Fraumeni and H. O. Adami (1996). The risk of stomach cancer in patients with gastric or duodenal ulcer disease. *New England Journal of Medicine* (Boston, MA), vol. 335, No. 4, pp. 242-249.
- Harris, C. C. (1996a). Structure and function of the p53 tumour suppressor gene: Clues for rational cancer therapeutic strategies. *Journal of the National Cancer Institute* (Bethesda, MD), vol. 88, No. 20, pp. 1442-1455.
- \_\_\_\_\_. (1996b). p53 Tumour suppressor gene: From the basic research laboratory to the clinic—an abridged historical perspective. *Carcinogenesis* (Fredrick, MD), vol. 17, pp. 1187-1198.
- Hayflick, L. (1965). The limited in vitro lifetime of human diploid cell strains. *Experimental Cell Research* (Orlando, FL), vol. 37, pp. 614-636.
- Hosking, S. W., T. K. W. Ling, S. C. S. Chung, M. Y. Yung, A. F. B. Cheng, J. J. Y. Sung and A. K. C. Li (1994). Duodenal ulcer healing by eradication of *heliobacter pylori* without anti-acid treatment: Randomised controlled trial. *Lancet* (New York, NY), vol. 343, pp. 508-510.
- Ikegami, N., and J. C. Campbell (1995). Medical care in Japan. *New England Journal of Medicine* (Boston, MA), vol. 333, No. 19, pp. 1295-1299.
- Jacobson, D. R., R. D. Pastore, R. Yaghubian, I. Kane, G. Gallo, F. S. Buck and J. N. Buxbaum (1997). Variant-sequence transthyretin (isoleucine 122) in late-onset cardiac amyloidosis in black Americans. *New England Journal of Medicine* (Boston, MA), vol. 336, No. 7, pp. 466-473.
- Johnson, T. E., and G. J. Lithgow (1992). The search for the genetic basis of ageing: The identification of gerontogenes in the nematode *caenorhabditis elegans*. *Journal of the American Geriatrics Society* (Washington, D. C.), vol. 40, pp. 936-945.
- Kasch, F. W., J. L. Boyer, S. P. Van Camp, L. S. Verity and J. P. Wallace (1993). Effect of exercise on cardiovascular ageing. *Age and Ageing* (Thousand Oaks, CA), vol. 22, pp. 5-10.
- Lakatta, E. G. (1985). Health, Disease, and Cardiovascular ageing. *America's Ageing: Health in an Older Society*. Washington, DC: National Academy Press, pp. 73-104.
- Lee, R. D., and L. R. Carter (1992). Modeling and forecasting U.S. mortality. *Journal of the American Statistical Association* (Alexandra, VA), vol. 87, No. 419, pp. 659-671.
- Liu, A. Y-C., Z. Lin, H. S. Choi, F. Sorhage and B. Li (1989). Attenuated induction of heat shock gene expres-

- sion in ageing diploid fibroblasts. *Journal of Biology and Chemistry* (Bethesda, MD), vol. 264, pp. 12037-12045.
- Malo, D., J. Tremblay and P. Harnet (1988). Genetic and molecular characteristics of thermosensitivity in hypertension. *Journal of Hypertension* (Baltimore, MD), vol. 6 (supplement 4), pp. S55-S57.
- Manton, K. G. (1996). The Demography of Ageing. In *Principles and Practice of Geriatric Medicine Third Edition*, M. S. J. Pathy, ed. England: John Wiley & Sons, Ltd.
- \_\_\_\_\_, and E. Stallard (1996). Longevity in the United States: Age and sex specific evidence on life span limits from mortality patterns: 1960-1990. *Journal of Gerontology: Biological Sciences* (Washington, D. C.), vol. 51A, No. 5, pp. B362-B375.
- \_\_\_\_\_, and L. Corder (1997). Changes in the age dependence of mortality and disability: Cohort and other determinants. *Demography* (Silver Springs, MD), vol. 34, No. 1, pp. 135-157.
- Manton, K. G., E. Stallard and H. D. Tolley (1991). Limits to human life expectancy: Evidence, prospects, and implications. *Population and Development Review* (New York), vol. 17, No. 4, pp. 603-637.
- Manton, K. G., and J. W. Vaupel (1995). Survival after the age of 80 in the United States, Sweden, France, England, and Japan. *New England Journal of Medicine* (Boston, MA), vol. 333, No. 18, pp. 1232-1235.
- Marenberg, M. E., N. Risch, L. F. Berkman, B. Floderus and U. de Faire (1994). Genetic susceptibility to death from coronary heart disease in a study of twins. *New England Journal of Medicine* (Boston, MA), vol. 330, No. 15, pp. 1041-1046.
- Martin, G. M., C. A. Spaque and C. J. Epstein (1970). Replicative life span of cultivated human cells: Effects of donor's age, tissue, and genotype. *Laboratory Investigation* (Baltimore, MD), vol. 23, pp. 86-92.
- Meydani, S. N., M. Meydani, J. B. Blumberg and others (1997). Vitamin E supplementation and in vivo immune response in healthy elderly subjects. *Journal of the American Medical Association* (Chicago, IL), vol. 277, No. 17, pp. 1380-1385.
- Mozar, H. N., D. G. Bal and S. A. Farag (1990). The Natural history of atherosclerosis: An ecological perspective. *Atherosclerosis* (Germany), vol. 82, pp. 157-164.
- Nebert, D. W. (1994). Drug-metabolising enzymes in ligand-modulated transcription. *Biochemical Pharmacology* (New York, NY), vol. 47, pp. 25-37.
- Nelson, D. R., L. Koymans, T. Kamataki, J. J. Stegeman, R. Flereisen, D. J. Waxman, M. R. Waterman, O. Gotoh, M. J. Moon, R. W. Estabrook, I. C. Gunsalus and D. W. Nebert (1996). P450 superfamily: Update on new sequences, gene mapping, accession numbers, and nomenclature. *Pharmacogenetics* (Philadelphia, PA), vol. 6, pp. 1-42.
- Nihon University (1982). *Population Ageing in Japan: Problems and Policy Issues in the 21st Century* (Kuroda, T., Ed.) Nihon University Population Research Institute: Nihon University, Japan.
- Olshansky, S. J., B. A. Carnes and C. Cassal (1990). In search of Methuselah: Estimating the upper limits to human longevity. *Science* (Washington, D. C.), vol. 250, No. 4981, pp. 634-640.
- Olshansky, S. J. (1991). Morbidity, Mortality, and Ageing. Paper presented at the Annual Meeting of the Population Association of America, Washington, DC, 1991.
- Parsonnet, J. (1996). *Helicobacter pylori* in the stomach-- A paradox unmasked. *New England Journal of Medicine* (Boston, MA), vol. 335, No. 4, pp. 278-280.
- Pearl, R. (1925). *The Biology of Population Growth*. New York: A. A. Knoff.
- Perera, F. P. (1996). Molecular epidemiology: Insights into cancer susceptibility, risk assessment, and prevention. *Journal of the National Cancer Institute* (Bethesda, MD), vol. 88, No. 8, pp. 496-509.
- Radford, N. B., M. Fina, I. J. Benjamin, R. W. Moreadith, K. H. Graves, P. Zhao, S. Gavva, A. Wiethoff, A. D. Sherry, G. R. Malloy and R. S. Williams (1996). Cardioprotective effects of 70-kDa heat shock protein in transgenic mice. *Proceedings of the National Academy of Sciences* (Washington, D. C.), vol. 93, pp. 2339-2342.
- Risch, H. A., G. R. Howe, M. Jain, J. D. Burch, E. J. Holowaty and A. B. Miller (1993). Are female smokers at higher risk for lung cancer than male smokers? A case-control analysis by histologic type. *American Journal of Epidemiology* (Baltimore, MD), vol. 138, No. 5, pp. 281-293.
- Roma, P., and L. Catapano (1996). Stress proteins and atherosclerosis. *Atherosclerosis* (Germany), vol. 127, pp. 147-154.
- Roth, J. A., and R. J. Cristiano (1997). Gene therapy for cancer: What have we done and where are we going? *Journal of the National Cancer Institute* (Bethesda, MD), vol. 89, No. 1, pp. 21-39.
- Ryberg, D., A. Hewer, D. H. Phillips and A. Haugen (1994). Different susceptibility to smoking-induced DNA damage among male and female lung cancer patients. *Cancer Research* (Philadelphia, PA), vol. 54, pp. 5801-5803.
- Sacher, G. A., and E. Trucco (1962). The stochastic theory of mortality. *Annals of the New York Academy of Sciences* (New York, NY), vol. 96, p. 985.
- Shock, N. W. (1984). The Baltimore Longitudinal Study of Ageing. *Normal Human Ageing*. Department of Health, Education, and Welfare publication, Bethesda, MD.
- \_\_\_\_\_, R. C. Greulich and R. Andres (1984). *Normal Human Ageing: The Baltimore Longitudinal Study on Ageing*. Pub No. 84-2480. Washington, D. C.: NIH.
- Social Security Administration (1992). Life Tables for the United States Social Security area 1900-2080 (Actuarial study 107). SSA Pub. No. 11-11536. Baltimore, MD: Social Security Administration.
- \_\_\_\_\_. (1997). *Social Security Area Population Projections: 1997*. Actuarial Study No. 112. SSA Pub. No. 11-11553. Baltimore, MD: Social Security Administration.
- Society of Actuaries (1994). 1994 Group annuity mortality table and 1994 group annuity reserving table. Schaumburg, IL: Society of Actuaries.
- Stebbing, A. R. D. (1987). Growth hormesis: A by-product of control. *Health Physics* (McLean, VA), vol. 52, No. 5, pp. 543-547.
- Strehler, B. L., and A. S. Mildvan (1960). General theory of mortality and ageing. *Science* (Washington, D. C.), vol. 132, pp. 14-21.
- U.S. Bureau of the Census (1996). *World Population Profile, 1996*. Report WP/96. Washington, D. C.: U.S. Government Printing Office.
- Warner, H. R., G. Fernandes and E. Wang (1995). A unifying hypothesis to explain the retardation of ageing and tumorigenesis by caloric restriction. *Journal of Gerontology: Bioscience* (Washington, D. C.), vol. 50, No. 3, pp. B107-B109.
- Weindruch, R., and R. S. Sohal (1997). Caloric intake and ageing. *New England Journal of Medicine* (Boston, MA), vol. 337, pp. 986-994.
- Welch, J. W. (1992). Mammalian stress response: cell physiology, structure/function of the stress proteins, and implications for medicine and disease. *Physiological Reviews* (Bethesda, MD), vol. 74, pp. 1063-1081.
- Wick, G., G. Schett, A. Amberger, R. Kliendienst and Q. Xu (1995). Is atherosclerosis an immunologically mediated disease. *Immunology Today* (New York, NY), vol. 16, pp. 27-33.
- Wilmoth, J. R. (1997). In Search of Limits. In *Between Zeus and the Salmon*, K. W. Wachter and C. E. Finch, eds. Washington, D. C.: National Academy Press, pp. 38-64.



- Wollner, L., and J. M. K. Spalding (1978). The autonomic nervous system. In *Textbook of Geriatric Medicine and Gerontology*, J. C. Brocklehurst, ed. Edinburgh, Scotland: Livingstone, pp. 245-267.
- Woodbury, M. A., and K. G. Manton (1977). A random walk model of human mortality and ageing. *Theoretical Population Biology* (Orlando, FL), vol. 11, pp. 37-48.
- Wurst, W., C. Benesch, B. Drabent, E. Rothermel, B. B. Benecke and E. Gunther (1989). Localization of heat shock protein 70 genes inside the rat major histocompatibility complex close to class III genes. *Immunogenetics* (Germany), vol. 30, pp. 46-49.
- Yashin, A. I., and K. G. Manton (1997). Effects of unobserved and partially observed covariate processes on system failure: A review of models and estimation strategies. *Statistical Science* (Iowa City, IA), vol. 12, No. 1, pp. 20-34.
- Yellon, D. M., A. M. Alkhulaifi and W. B. Pugsley (1993). Preconditioning the human myocardium. *Lancet* (New York, NY), vol. 342, pp. 276-277.
- Zang, E. A., and E. L. Wynder (1996). Differences in lung cancer risk between men and women: Examination of evidence. *Journal of the National Cancer Institute* (Bethesda, MD), vol. 88, Nos. 3/4, pp. 183-192.
- Zuckerman, A. J. (1997). Prevention of primary liver cancer by immunisation. *New England Journal of Medicine* (Boston, MA), vol. 336, No. 26, pp. 1906-1907.

## **XV. THE GENETIC MAKE-UP OF POPULATION AND ITS IMPLICATIONS FOR MORTALITY BY CAUSE OF DEATH: LINKS BETWEEN ALZHEIMER'S AND ISCHAEMIC HEART DISEASE**

*Douglas Ewbank\**

### **A. INTRODUCTION**

Few people believe that an individual's genes completely dictate their destiny or even their health and age at death. However, an increasing number of researchers are beginning to believe that the future of medical research will be dominated by genetics. Although it is clear that genes interact with the environment (e.g., pollution, diet, exercise, etc.), in some cases genetic research may provide the key to understanding the aetiology of disease. This might involve identifying genes associated with excess (or lowered) risk of disease, discovering the biochemical action of the gene, and creating drugs to counteract (or stimulate) those mechanisms. Gene therapy may even be the basis for treatment. This research strategy may uncover strategies that are effective for cases of the disease that are not associated with the particular gene type that led to the initial discovery.

Although there has been increased research on genetic epidemiology, there has been little discussion of the role played by genes in determining variations in mortality rates and the prevalence of morbidity. One reason for this is that much genetic research aims to discover rare genotypes that affect the risks of individual diseases. Many of these genotypes are either so rare or affect such uncommon diseases that they are associated with only a minuscule fraction of the overall burden of disease or mortality.

Another problem is that the effects of most genes are so heavily dependent on interactions with the environment or other genes that it is

difficult to identify single genes associated with the most common diseases. Interactions reduce the apparent effect of genes and make it more difficult to observe statistically significant differences. Most research strategies look for the marginal effect of one gene by comparing individuals with that gene to others. This is easier to do with families that include many cases of early or severe forms of the disease since it is likely that these cases share the same risk factors. However, common genes generally are not the cause of uncommon family clusters of disease.

Some researchers have used data on twins to estimate the extent to which genes affect survival. For example, studies of Danish twins suggest that about one-quarter of the variability among individuals in mortality can be ascribed to genetic variation within the population (Christensen and Vaupel, 1996). However, this does not tell us anything about the contribution of genetics to differences between populations. To date, there has been little effort to estimate what proportion of mortality differences between populations can be explained by genetic variation. It is not yet possible to estimate with any degree of precision the contribution of genetics to variations in health and mortality across populations.

There is one gene, however, that has been the object of extensive research that provides insights into the likely contribution of genetic factors to explaining morbidity and mortality differentials across populations. Variations in the Apolipoprotein-E (APOE) gene are associated with large variations in the risk of both Ischaemic Heart Disease (IHD) and Alzheimer's disease (AD). When we combine large differences in the frequency of the risky APOE genotypes with large relative risks of these two

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major diseases, the implications for cross-national comparisons are potentially quite large. Therefore, APOE provides a preliminary look at the contribution of genetic differences to mortality differentials.

There are three common forms of the APOE gene:  $\epsilon 2$ ,  $\epsilon 3$ , and  $\epsilon 4$ . Each individual inherits one copy of the gene from each parent. The most common combination is the  $\epsilon 3/3$  or homozygous  $\epsilon 3$  genotype. However, there is substantial variation across populations. The  $\epsilon 4/3$  and  $\epsilon 4/4$  genotypes, which are associated with an increased risk of both IHD and AD, make up 50 per cent of population samples in New Guinea and 48 per cent in Nigeria, but only 9 per cent in a sample of Chinese in Taiwan and 13 per cent of a sample of Chinese in Singapore. (Kamboh, Bhatia, and Ferrell, 1990; Sephernia and others, 1988; Kao and others, 1995; Heng, Saha, and Tay, 1995). Figure 95 presents a map of Europe shaded to reflect the proportion of the population that is APOE  $\epsilon 4/3$  or  $\epsilon 4/4$ . We do not have nationally representative data for any country and the estimates for some countries are based on data from a single town. However, it is clear that populations in Northern Europe have higher frequencies of 4 than southern populations. In Finland, 34 per cent of the population is either  $\epsilon 3/4$  or  $\epsilon 4/4$  whereas in Italy the proportion is only 17 per cent (Corbo and others, 1995).

This paper reviews the evidence linking APOE to the risks of IHD and AD and examines the implications of the variation in frequency of the  $\epsilon 4$  allele for differences in mortality and morbidity. This review reveals that differences in APOE gene frequencies explain a significant proportion of the differences in mortality in developed countries at the older ages. Although high levels of APOE  $\epsilon 4$  in many high mortality populations do not appear to be linked to high levels of IHD and AD, this gene might explain some of the rapid change in

mortality rates in response to higher fat diets and other changes in life styles.

The first section describes the methodology we will use to produce estimates of differences in morbidity and mortality attributable to APOE gene frequencies. The next section presents estimates of the extent to which differences in the incidence of heart attacks and mortality to IHD might be attributable to variations in APOE gene frequencies. The third section examines the likely contribution of variation in APOE gene frequencies to variations in the prevalence of AD. In the fifth section, we estimate the size of differentials in overall mortality at the oldest ages that might be attributable to APOE distributions. We then discuss implications for future mortality trends in developing countries. The final section of the paper discusses the implications of these findings for future research in demography and epidemiology and the significance for health planning.

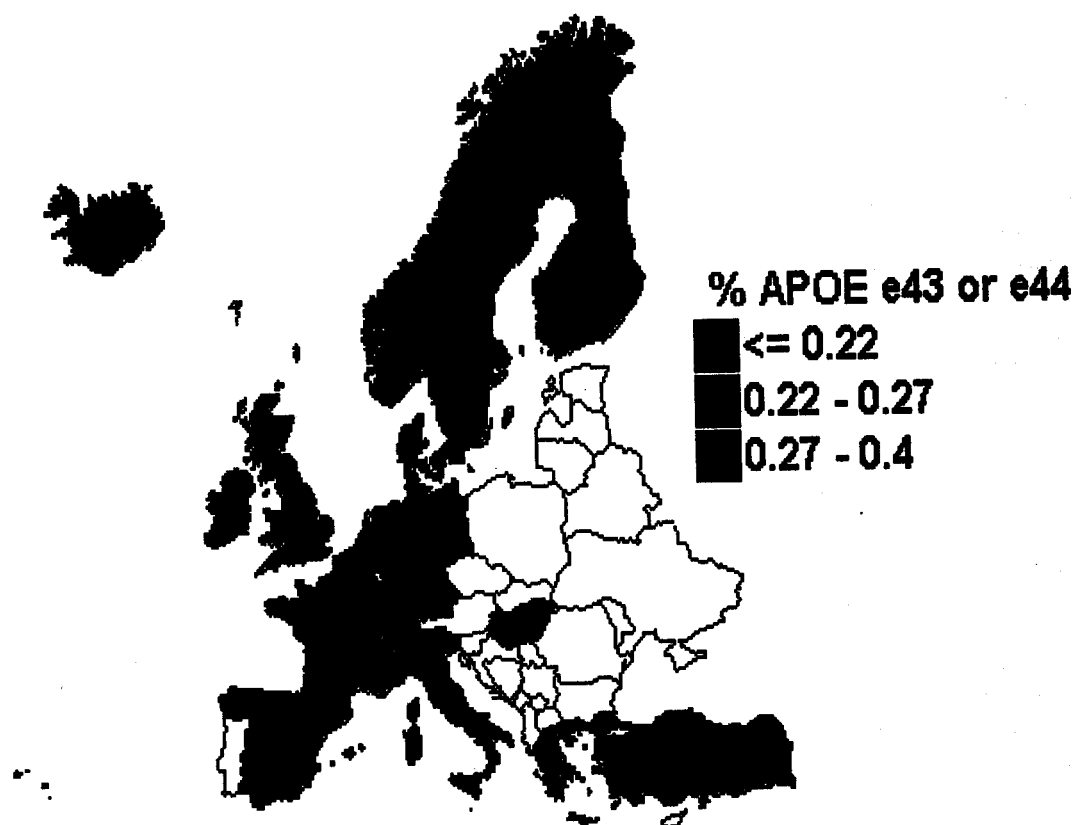
## B. METHODOLOGY

Several studies provide excellent estimates of the relative risks of AD, IHD or total mortality by APOE genotype in different populations. Our approach is to calculate what these estimates imply for differences in morbidity and mortality among a range of populations.

We use one country as the standard. The choice of a standard is arbitrary if we are interested in calculating the proportion of the difference between two countries that is attributable to APOE. However, we generally use as the standard the country for which the relative risks are available. If  $a_i$  is the proportion in the standard population with genotype  $i$  and  $m_x^1$  is the mortality rate for genotype 1 at age  $x$ , then the mortality rate for the standard population aged  $x$  is:

$$m_x = a_1 m_x^1 + a_2 m_x^2 R_2 + a_3 m_x^3 R_3 + \dots = m_x^1 (a_1 + a_2 R_2 + a_3 R_3 + \dots) \quad (1)$$

Figure 95. Proportion of the population of research studies areas that has APOE genotype  $\epsilon 4/3$  or  $\epsilon 4/4$



Source: R. M. Corbo and others, "Apolipoprotein E polymorphism in Italy investigated in native plasma by a simple polyacrylamide gel isoelectric focusing technique. Comparison with frequency data of other European populations", *Annals of Human Genetics*, vol. 59, pp. 197-209 (1995).

where  $R_i$  is the relative risk of mortality for genotype  $i$  relative to genotype 1. Since we know  $m_x$ , the  $R_i$ , and the  $a_i$ , we can solve this equation for  $m_x^1$ , the mortality rate for genotype 1. We then estimate the mortality rates for the other genotypes using the  $R_i$ .

We can now estimate what the IHD mortality rate would be in a different population if the only difference between the populations was the distribution of the populations by APOE genotype and if the effect of APOE genotypes is the same in both populations. We do this by simply changing the values of the  $a_i$  to match those in the other population. If the gene frequencies in two populations are labelled  $b_i$  and  $c_i$ , then the

ratio of the estimates for two mortality rates would be:

$$\frac{m_x^1 (b_1 R_1 + b_2 R_2 + \dots)}{m_x^1 (c_1 R_1 + c_2 R_2 + \dots)}$$

Since the  $m_x^1$  values cancel out, this ratio does not depend on the choice of a standard. Therefore, if we are only interested in the estimates of the relative risks in two populations, we do not even need to assume a rate for a standard population.

This method is based on the assumption that the relative risks associated with each genotype do not vary across populations. This is usually

a strong assumption given the importance of interactions among different genes and between genetic and environmental risk factors. However, in the case of APOE it appears that the risks associated with each genotype are quite similar across populations, at least among developed countries. Hallman and others (1991) have shown that the relationship between APOE genotype and mean serum cholesterol values are very similar in a sample of populations that includes Finland, Hungary, Japan, Sudan, and Chinese, Indians and Malays in Singapore. However, if the effect of serum cholesterol on the risk of heart attack is non-linear (as is likely) there may be differences in the relative risks of IHD across populations. Studies of Alzheimer's disease give very similar estimates of the risks of AD associated with APOE genotypes in Finland, the U.S., Japan and Chinese populations (Kuusisto and others 1994; Corder and others, 1993; Hong and others, 1996; Mak and others, 1996).

This approach to estimating the likely impact of differences in APOE genotypes is much more reliable than the frequently used alternative: regression analysis. Regression analysis would assign as much of the variance in mortality or morbidity as possible to the APOE distributions. Since there would inevitably be numerous variables that could not be included in the regressions (e.g., the gene frequencies for unidentified genes), the estimates for the differences attributable to APOE could be seriously in error. Regression estimates would also be subject to severe errors of aggregation. Showing that populations that have high frequencies of the APOE  $\epsilon 4$  allele have high mortality does not prove that individuals with  $\epsilon 4$  have higher risks. By starting with epidemiologic data comparing the APOE genotype and mortality/morbidity rates of individuals, we avoid all of these problems.

### C. APOE AND HEART DISEASE

Research has demonstrated that high levels of low-density lipoprotein (LDL) and cholesterol in the blood are associated with increased risks of heart attacks and total mortality. About 70 per cent of cholesterol in the plasma is carried by LDL particles that form LDL-C. About 50

per cent of inter-individual variation in LDL-C levels is genetic in origin. There has been extensive research designed to understand the particular genes involved in determining LDL-C levels. Several very rare genetic defects (including rare forms of Apolipoprotein-B) are known to produce very high or very low levels of LDL-C in the blood. However, APOE is the only gene that has been convincingly demonstrated to be associated with variations in serum levels of LDL-C in studies in a variety of populations. APOE is responsible for about 5-10 per cent of the inter-individual variation in plasma LDL-C levels. APOE genotype is also associated with variations in other risk factors for heart attack, including triglycerides (For a more complete discussions, see Cohen, and others, 1996).

#### 1. *The risk of heart attack*

There has been extensive research on the risks of heart attacks associated with the various APOE genotypes. The best of these studies were reviewed by Wilson and others (1996); they estimated the effects of APOE on various heart disease rates (including both mortality and morbidity) among men and women aged 40-70. They conclude that the APOE  $\epsilon 4/3$  genotype is associated with a probability of coronary heart disease among men that is 1.38 times the risk associated with the  $\epsilon 3/3$  genotype. Among women the relative risk is estimated to be 1.82. They estimate that the odds ratios for  $\epsilon 2/3$  are 0.94 among men and 1.07 among women.

We can use the methods described above to compare the risk of heart disease in two pairs of populations. The first pair is Finland and Italy; the second pair is U.S. blacks and whites. In each pair the first population has a very high frequency of the  $\epsilon 4$  allele and the second has a much lower frequency of  $\epsilon 4$ . We estimate that if there were no other differences between Finland and Italy, the differences in APOE gene frequencies would lead men in Finland to have a coronary heart disease rate 12 per cent higher than Italian men. Women in Finland would have a rate about 18 per cent higher than Italian women. Among both men and women, we would expect blacks to have a coronary heart disease rate about 16 per cent higher than among whites.

## 2. *Ischaemic heart disease mortality among men aged 45-64*

Eichner and others (1993) examined coronary heart disease (CHD) mortality rates by APOE genotype among men aged 35-57 using the Multiple Risk Factor Intervention Trial (MRFIT) study. They followed the survival of these men for 10.5 years (i.e., to ages 46-68). They found that after controlling for smoking, age, blood pressure, body mass index, and serum levels of low-density lipoproteins that men with the APOE  $\epsilon 3/4$  genotype had an IHD mortality rate that was 2.3 times the rate among those with genotype  $\epsilon 3/3$ . The men with genotype  $\epsilon 4/4$  had a rate that was 8.7 times as high and those with the  $\epsilon 3/2$  genotype had a rate 1.7 times as high.<sup>1</sup> We can use these data to estimate the extent to which differences in the APOE gene frequencies across populations could explain the observed differences in IHD mortality.

We use the U.S. white population as the basis for our estimates in order to match the MRFIT population. To apply this to the MRFIT data, we assume that the  $\epsilon 2/4$  and  $\epsilon 2/2$  (the two least frequent genotypes) have the same relative risk of CHD mortality as the  $\epsilon 2/3$  genotype. We then use equation (1) to estimate that the mortality rate to CHD mortality among the APOE  $\epsilon 3/3$  genotype is 0.642 per 1000 among U.S. white males aged 65-69. Then the estimated rate among the  $\epsilon 3/4$  is 2.3 times this or 1.475. We then apply the APOE gene frequencies from other populations and estimate the implications for differences in IHD mortality across populations.

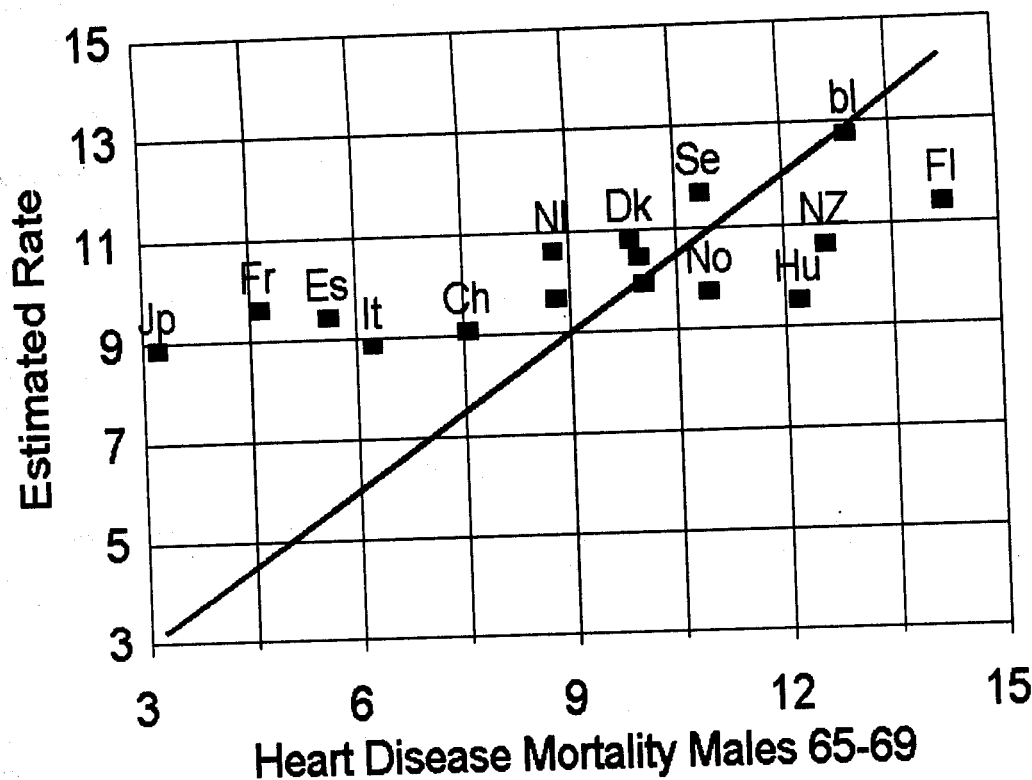
Figure 96 compares these estimated IHD mortality rates with the true rates. For example, the mortality rate among males aged 65-69 in Finland in 1989 was 14.2 per 1,000, 48 per cent higher than the rate among U.S. whites. However, if the only difference between these two populations were the frequency of the APOE genotypes, we would expect that men in Finland would have a rate 18 per cent greater than that among U.S. white males. Therefore about 14 per cent of the difference between Finnish and U.S. white males in IHD mortality rates is attributable to differences in APOE gene frequencies.

A useful way of summarising the role of APOE in explaining the differences between mortality rates to CHD is to compare the rates for Finland and Italy, which have very different APOE gene frequencies. The CHD mortality rate among males aged 65-69 in Finland is 129 per cent higher than the rate in Italy. If the two only differed in their APOE gene frequencies we would expect a difference of 29 per cent. Therefore, differences in their APOE gene frequencies "explain" about 22 per cent of the difference between them. If we assume that the relative risks reported by Eichner and others also apply at later ages, we estimate that 55 per cent of the excess IHD mortality at age 80-84 in Finland relative to Italy is attributable to APOE.

We can compare these results to an analysis of the relationship between the prevalence of high serum cholesterol levels and IHD mortality. The MONICA study provides data for a number of study areas on the proportion of the population that has high cholesterol (World Health Organization Monica Project, 1994). We can run a regression of these values on the IHD mortality rates for men aged 65-69 in ten European countries. Although there is some error added by comparing national level mortality rates to cholesterol rates for only a few locations in the country, this is no different than what we have done for APOE. The use of regression analysis may lead to biased estimates of the importance of cholesterol levels if those levels are correlated with other determinants of IHD mortality rates. However, the comparison is instructive.

The regression analysis suggests that differences in the percentage with high serum cholesterol would lead men in Finland to have an IHD mortality rate at age 65-69 that is 26 per cent higher than men in Italy. This is very similar to the 29 per cent difference ascribable to APOE. On the one hand, it is not surprising that APOE and cholesterol appear to have similar effects on mortality differences since an individual's serum cholesterol levels are affected by his APOE genotype. On the other hand, given the importance often ascribed to differences in cholesterol levels and the assumed implications for differences in dietary consumption of fats, it is surprising that APOE has not attracted equal attention.

Figure 96. Heart disease mortality rates for males 65-69 and rates estimated from APOE gene frequencies, 16 populations



Key:	bl	U.S. blacks	J	Japan
	CH	Switzerland	Hu	Hungary
	Dk	Denmark	NZ	New Zealand
	Es	Spain	No	Norway
	FI	Finland	Se	Sweden
	Fr	France	NI	Netherlands
	It	Italy		

Also included: Canada, German Federal Republic, and Iceland.

Source: United Nations, *Demographic Yearbook, Special Issue: Population Aging and the Situation of Elderly Persons* (New York, United Nations, ST/ESA/STAT/SER.R/22, 1993).

Although this analysis suggests that differences in the frequency of APOE genotypes might explain a significant fraction of the differences in IHD mortality among these countries, this result must be interpreted with caution. Since the APOE genotype appears to

mediate the relationship between dietary intake of fats and serum lipid concentrations, any correlation between APOE gene frequencies and population averages for dietary consumption of fats would greatly complicate this type of analysis.

It appears that individuals with different APOE genotypes have mean serum cholesterol levels that differ by about the same amount in a wide range of populations (Hallman and others, 1991). However, if the relationship between serum cholesterol and heart attack rates is non-linear, then differences in two APOE genotypes in the mean serum cholesterol level would have different effects on CHD in countries with a different mean serum level. It is likely that in countries with very high mean serum cholesterol levels there are larger differences in the incidence of heart attacks and CHD mortality (i.e., the relative risk associated with APOE  $\epsilon 4/3$  is larger). In addition, in populations with high consumption of cholesterol, the APOE  $\epsilon 3/3$  genotype may be associated with higher CHD mortality. Therefore, in populations that consume high levels of cholesterol, the base rate for APOE  $\epsilon 3/3$  might be higher and the relative risks for APOE  $\epsilon 4/3$  and  $\epsilon 4/4$  might be higher.

If countries with higher mean cholesterol levels have higher risks of heart attack associated with the APOE  $\epsilon 4/3$  and  $\epsilon 4/4$  genotypes, then our estimates will be biased. If Italians consume less fat or cholesterol than U.S. whites, the method used here will overestimate their CHD mortality rate. Similarly, if Finns eat more fats or cholesterol than U.S. whites their estimated CHD mortality rate will be too low. Therefore, it is possible that these calculations actually underestimate the true importance of APOE.

For example, if the CHD mortality rate among the APOE  $\epsilon 3/3$  genotype is 10 per cent higher in Finland than in Italy and the relative risks for APOE  $\epsilon 4/3$  and  $\epsilon 4/4$  are 2.5 and 9.1 in Finland (instead of 2.1 and 8.3), then the difference in APOE genotypes would explain 33 per cent of the difference between CHD mortality rates in Italy and Finland rather than 29 per cent.

Differences in the relative risks of CHD by APOE genotype might also explain why many populations that have a very high frequency of the APOE  $\epsilon 4$  allele do not have high mortality rates to CHD (for example, most of sub-Saharan Africa). Since cholesterol is needed by the body, and since the  $\epsilon 4$  allele is associated with

increased cholesterol absorption it may be that in a population that has a very low fat diet, the APOE  $\epsilon 4$  allele actually is advantageous. This might help explain why many populations have such a high frequency of a genotype that is associated with two major causes of death.

#### D. APOE AND THE RISK OF ALZHEIMER'S DISEASE

Alzheimer's disease (AD) is the most common cause of dementia in the elderly in most populations. In the U.S. and Europe it is responsible for about 65-70 per cent of all dementia. The Canadian Study of Health and Aging, which was based on a large random sample of the population, estimated that the prevalence of AD over age 85 was 26 per cent. It also estimated that 37 per cent of persons over age 65 living in institutions had AD. Individuals who first show symptoms of AD at age 75 live an average of about seven years (Diesfeldt, van Houte, and Moerkens, 1986), although there is substantial variation. In the U.S., AD is the third or fourth most common cause of death (Ewbank, forthcoming).

The variations in the risk of AD associated with APOE genotypes are probably the most thoroughly documented example of genetic effects on a chronic disease. Studies have demonstrated the excess risk of AD associated with the APOE  $\epsilon 4$  allele in populations as diverse as Finland, Italy, U.S. Blacks, Chinese and Japanese. The  $\epsilon 4$  allele is associated with the highest risk of AD and the  $\epsilon 2$  is associated with the lowest risk.<sup>2</sup> APOE does not cause AD in all cases. Individuals who are  $\epsilon 4/4$  can survive to age 100 without showing signs of dementia and individuals who are  $\epsilon 3/2$  can get AD. However, about 25 per cent of AD cases in the U.S. are attributable to the excess risk associated with the APOE  $\epsilon 4$  allele (Evans and others, 1997)

The risk of AD associated with the various APOE genotypes varies with age (Farrer and others, 1997). The highest excess risk for the  $\epsilon 4/3$  genotype around age 70. At younger ages there are other, rarer genes that cause AD. These reduce the differences between APOE



genotypes at the youngest ages (e.g., under 60). At the oldest ages we see the effects of other (mostly unknown) risk factors. Any individual carrying the  $\epsilon 4$  allele who survives to age 90 without becoming demented probably has a number of other genetic or environmental characteristics that are protective against AD. Therefore, their risks of AD are not as high as carriers of  $\epsilon 4$  at younger ages.

We can estimate the magnitude of differences in AD prevalence that might be associated with differences in APOE gene frequencies across populations. Three studies provide estimates of the relative odds of having AD around age 70. (Corder and others, 1995; Kukull and others, 1996; Bickbøller and others, 1997). Combining these data, we estimate that the relative odds of having AD are 3.8 for  $\epsilon 4/3$ , 20.3 for  $\epsilon 4/4$  and 0.30 for  $\epsilon 2/3$  and  $\epsilon 2/2$  compared to  $\epsilon 3/3$  genotype. We assume that the rare  $\epsilon 4/2$  genotype is associated with an odds ratio of 1.0. If we apply the odds ratios to the estimated prevalence at ages 65-74 in Canada, we estimate that the prevalence rate of AD among the  $\epsilon 3/3$  is 0.56 per cent. The estimated rates for the other groups are:  $\epsilon 4/3$ : 2.09 per cent,  $\epsilon 4/4$ : 11.11 per cent, and  $\epsilon 2/3$ : 0.17 per cent.

With these rates and the APOE gene frequencies for Italy and Finland, we estimate that AD prevalence at ages 65-74 in Finland would be about 65 per cent higher than the rates in Italy. Similarly, we estimate that the prevalence among U.S. blacks might be about 60 per cent higher than among U.S. whites if these populations only differed in their APOE distributions.

It is not possible to estimate the fraction of the total variation in AD prevalence that is attributable to APOE, since the data on AD are less common and less consistent than data on IHD mortality. Because of the large sample sizes required for comparing populations and because of differences in diagnostic criteria used in different studies, comparisons across prevalence studies are unreliable. However, it appears that U.S. blacks do have substantially higher rates of AD than whites (Hendrie and others, 1995b).

## E. OVERALL MORTALITY DIFFERENCES BY APOE GENOTYPE

There are two approaches to estimating differences in overall mortality by APOE genotype. The first utilises data provided by Corder and others (1996) on seven-year survival rates in the Kungsholmen Project in Sweden by APOE. The second approach uses data that show a much lower frequency of the  $\epsilon 4/3$  and  $\epsilon 4/4$  genotypes among centenarians than among middle-aged individuals from the same population.

### 1. Mortality estimates from the Kungsholmen Project

Corder and others (1996) provide estimates of variations by APOE genotype in the risk of dying over a seven-year period from the Kungsholmen Project in Stockholm. They report that among individuals aged 75-84 at a baseline survey, the odds of dying during the subsequent seven years were 1.3 times as high for the  $\epsilon 4/3$  genotype as for the  $\epsilon 3/3$  genotype, for sexes combined. At ages 85 and above, the relative odds increased to 2.25. The odds associated with the  $\epsilon 2/3$  genotype varied across sex and age group, probably because of the relatively small sample sizes. Overall the  $\epsilon 3/2$  genotype was associated with an odds ratio of 0.8 relative to the  $\epsilon 3/3$ .

We can assume that the  $\epsilon 4/4$  genotype is associated with an odds ratio that is the square of the odds ratio for  $\epsilon 4/3$ . This is consistent with the relationships observed for both IHD and AD in several studies. We can also assume that the  $\epsilon 2/2$  genotype is the same as the  $\epsilon 3/2$  and the  $\epsilon 4/2$  is the same as the  $\epsilon 3/3$  since these genotype are very rare.

Given these odds ratios, we can calculate what mortality rates we would expect for various countries. We use Sweden as the standard. We have calculated mortality rates standardised to the distribution by sex for ages 85 and over and rates standardised to the age and sex distribution of those 75-84. The standardised mortality rate at 75-84 in Finland is 10 per cent higher than the rate in Italy (United Nations,

1993). According to the APOE gene frequencies in the two countries and the odds ratios from Kungsholmen, we would expect Finland to have a rate that is 7 per cent higher than Italy. Over age 85, we observe a difference of 5 per cent, but would expect a difference of 11 per cent from the APOE distribution. The estimate for the older age group is probably biased upward since excess mortality among the  $\epsilon 4/3$  and  $\epsilon 4/4$  would reduce the differences between the APOE gene frequencies of the oldest old in Finland and Italy.

These calculations suggest that the different distributions by APOE can explain a substantial proportion of the differences in mortality at the oldest ages between Finland and Italy.

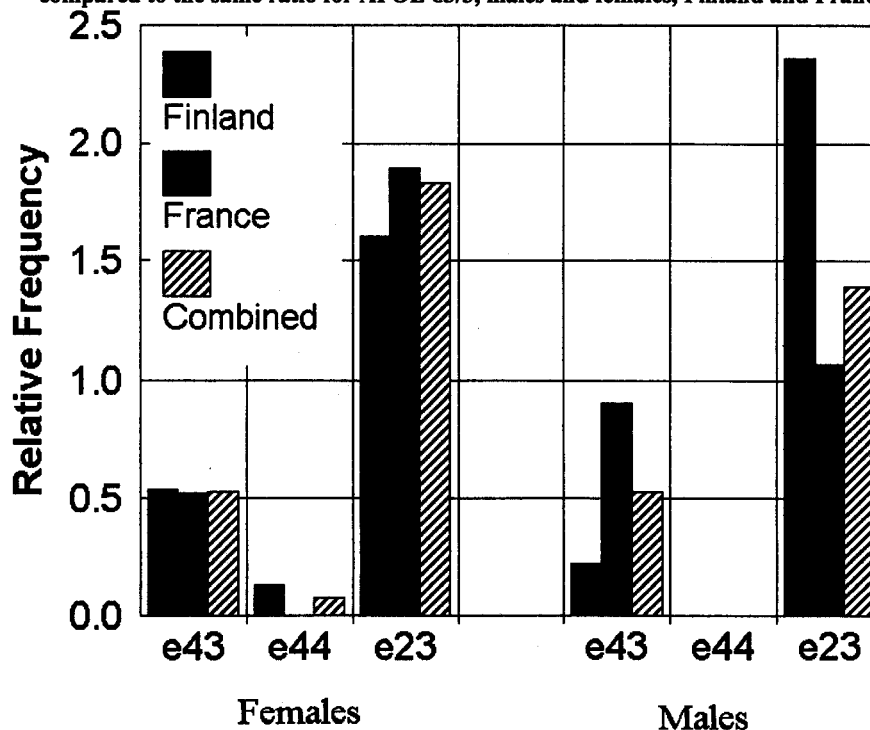
## 2. Estimates from APOE gene frequencies among centenarians

The two largest studies of APOE among centenarians cover populations in France

(Schächter and others, 1994) and Finland (Louhija and others, 1994). The French data show that among middle aged adults, 18.0 per cent were either  $\epsilon 4/3$  or  $\epsilon 4/4$  (figure 97). Among centenarians, this proportion had dropped to 14.6 per cent among men and 8.5 per cent among women. Similarly the Finnish data show a decline from 38.4 per cent to 7.4 per cent among men and 18.5 per cent among women. We can combine the data from the two countries by adding together the cases and weighting the data on controls by the number of cases in each study. This leads to an estimated 47 per cent decline in the proportion  $\epsilon 4/3$  or  $\epsilon 4/4$  among men and a 48 per cent decline among women.

These declines in the frequency of the  $\epsilon 4/3$  and  $\epsilon 4/4$  genotypes must be the result of excess mortality among the carriers of the  $\epsilon 4$  allele. We can estimate the implied excess mortality rate using the life table for U.S. whites which is reasonably accurate and available by single years of age up to 105. We assume that the

Figure 97. The frequency of APOE genotypes among centenarians relative to young adults compared to the same ratio for APOE  $\epsilon 3/3$ , males and females, Finland and France



Sources: France: F. Schächter, and others, "Genetic associations with human longevity at the APOE and ACE loci, *Nature Genetics*, vol. 6, No. 1, pp. 29-32 (1994); Finland: J. Louhija, and others, "Aging and genetic variation of plasma apolipoproteins. Relative loss of the apolipoprotein  $\epsilon 4$  phenotype in centenarians, *Arteriosclerosis & Thrombosis*, vol. 14, No. 7, pp. 1084-1089. NOTE: There were no APOE  $\epsilon 4/4$  among the males in either study.

declines are a result of excess mortality between ages 80 and 101. To estimate the relative risks of death among males implied by the observed changes in the APOE frequencies, assume that the gene frequencies change exponentially between ages 80 and 101. We also assume that the relative risk of death for each genotype relative to  $\epsilon 3/3$  is the same at every age. We then choose the relative risks associated with  $\epsilon 4/3$ ,  $\epsilon 4/4$ , and all other genotypes to minimise the sum of squared errors in the gene frequencies at age 101 implied by the French and Finnish data. The resulting relative risks are given in table 57. They are very similar to the values given by Corder and others for the seven-year survival rates.

These relative risks lead to a large difference in life expectancy at age 80 (table 57). If we apply the gene frequencies for other countries, we find that differences in the APOE gene frequencies can be responsible for noticeable differences in life expectancy at age 80. For example, from their APOE gene frequencies alone, we estimate that males in Italy would have a life expectancy at age 80 that is 0.17 years longer than males in Finland. This is substantially greater than the reported differences of 0.09 years. Similarly, the different APOE gene frequencies for whites and blacks in the U.S. implies a difference of 0.16 years for men compared to the estimated true difference of 0.33 years in 1985 (Preston and others, 1996). The differences for women are about the

same. The APOE gene frequencies imply a difference in life expectancy at age 80 of 0.27 years between Italy and Finland and 0.22 years between U.S. whites and blacks compared with reported differences of 0.37 years and 0.42 years.

#### F. IMPLICATIONS FOR DEVELOPING COUNTRIES

Data from developing countries suggest that populations that have high frequencies of the APOE  $\epsilon 4$  allele may not always have high rates of IHD and AD. In particular, it appears that African populations do not have high rates of IHD mortality despite very high frequencies of  $\epsilon 4$ . There are also some data that suggest that Nigerians have a very low rate of AD. This is in stark contrast with the rates among African-Americans that have very high IHD mortality and probably have very high rates of AD.

It is likely that there are environmental conditions in Africa that reduce or eliminate the excess risk associated with the  $\epsilon 4$  allele. Certainly the most likely candidate in the case of IHD is dietary consumption of fats. Furthermore, it is possible that as these populations (or parts of these populations) begin to adopt diets similar to those in Europe and the U.S. that their mortality rates to IHD and possibly their prevalence of AD, will increase substantially. This is not a new hypothesis. However, the evidence

TABLE 57. ESTIMATED RELATIVE RISKS OF DYING AND LIFE EXPECTANCY AT AGE 80, U.S. WHITES BY SEX AND APOE GENOTYPE

Genotype	Males		Females	
	Relative risks Ages 80-100	Life expectancy age 80	Relative risks ages 80-100	Life expectancy Age 80
APOE $\epsilon 3/3$	1.00	7.94	1.00	9.75
APOE $\epsilon 4/3$	1.18	6.98	1.21	8.76
APOE $\epsilon 4/4$	1.43	6.13	1.79	6.92
Other	0.95	8.01	0.84	10.77

Source: Author's calculation, see text.

about the role of APOE in IHD and AD suggests that many populations in developing countries might be even more sensitive to the risks associated with increased consumption of fats than some European populations.

If this is true, then we might expect that adoption of European-style diets would have very different effects in different developing countries. For example, in Papua New Guinea, Nigeria and the Sudan where the proportion of the population that is  $\epsilon 4/3$  or  $\epsilon 4/4$  reaches 44 per cent to 50 per cent, the effects of higher fat consumption might lead to IHD mortality rates that are among the highest in the world. On the other hand, studies in several Chinese populations suggest that only about 13 per cent of Chinese are  $\epsilon 4/3$  and  $\epsilon 4/4$  (Gerdes, 1992). Therefore, China may continue to have relatively low IHD mortality rates even in those parts of the population that adopt high fat diets. The same may also be true in parts of India, although the only available data cover Indians in Malaysia (Gerdes, 1992).

#### G. SUMMARY AND CONCLUSIONS

Differences in APOE gene frequencies do not fully explain differences in mortality among the elderly in developed countries. However, it is clear that this one gene is responsible for substantial differences in the incidence of IHD and AD, mortality to IHD and a small but significant amount of overall mortality. The differences in these rates attributable to variations in the APOE gene frequencies compare favourably with the differences attributable to major risk factors such as diet, serum cholesterol levels, and smoking and may be as large or larger than differences due to variations in health care systems.

These findings suggest several things for future studies of mortality differences between developing countries. First, it is dangerous to draw conclusions about the importance of dietary fats from the relationship between serum cholesterol and IHD morbidity and mortality. It is possible that much of the difference in mortality that is often ascribed to serum cholesterol is actually due to differences in APOE gene frequencies, not dietary patterns. This does not

imply that dietary patterns are not important contributors to differences in morbidity and mortality. At a minimum, it implies that any study of the relationship between diet and IHD, whether among individuals or across populations, must control for APOE genotypes.

Second, we cannot ignore the effects of differing gene frequencies as potentially important contributors to mortality differences. For the moment, APOE is unique. No other gene with several common alleles has been shown to have such large effects on one, let alone two, major causes of death. It is also unusual that these effects appear to be very similar across populations that are as different as Italy, Finland, Japan, Chinese, and U.S. blacks. However, it would be a mistake to assume that there are no other genes that have similar significance for differences among populations. Most genetic research has been designed to discover genetic risk factors that are responsible for very high rates of disease in small populations (often families). Therefore, it is not surprising that we have not identified common genotypes associated with relatively large difference in the risk of disease.

Third, genetic factors may provide clues to future trends in morbidity and mortality in developing countries. For example, many populations in developing countries have very high frequencies of  $\epsilon 4/3$  and  $\epsilon 4/4$ . For example, exceedingly high rates are found in parts of many countries in Africa (e.g., Sudan: 44 per cent are  $\epsilon 4/3$  or  $\epsilon 4/4$ , Nigeria: 48 per cent, and Zaire: 53 per cent; Zekraoui and others, 1997). Similarly, 50 per cent of adults in an area of Papua New Guinea were  $\epsilon 4/3$  or  $\epsilon 4/4$ . However, studies in other developing countries show more moderate levels, for example, 29 per cent in Indonesia and 13 per cent in China. Although these populations currently have relatively low rates of IHD morbidity and mortality, their APOE gene frequencies may affect how these populations respond to increasing consumption of fats. It may be that populations in Africa and Papua New Guinea would be much more seriously affected by increasing fat intake than Indonesian or Chinese populations.

Studying the effects of one gene cannot tell us much about the overall proportion of mortality

or morbidity differentials that are attributable to genetic factors in general. For example, it is theoretically possible that genotypes that are protective against cancer could be more common in Finland than in Italy, thereby counteracting some of the advantage the Italians enjoy from their APOE gene frequencies. However, the large effects associated with APOE raise the possibility that genetics may explain a substantial fraction of the differences among populations as well as among individuals within a population.

# NOTES

<sup>1</sup>Ideally we would like to see estimates that controlled for all of these factors except low-density lipoprotein. However, the relative risk without any controls did not differ from those reported here. The risk associated with the  $\epsilon 4/3$  is slightly lower (2.0) but the odds associated with  $\epsilon 4/4$  are slightly higher (11.8).

<sup>2</sup>There is still some uncertainty about the role of  $\epsilon 2$  in U.S. blacks (Tang and others, 1996; Hendrie and others, 1995a) and Nigerians (Osuntokun and others, 1995).

# REFERENCES

- Bickeb  ller, Heike, Dominique Campion, Alexis Brice, Philippe Amouyel, Didier Hannequin, Olivier Didierjean, Christiane Penet, Cosette Martin, Jordi P  rez-Tur and others (1997). Apolipoprotein E and Alzheimer's disease: genotype-specific risks by age and sex. *American Journal of Human Genetics*, vol. 60, pp. 439-446.
- Christensen, K. and J. W. Vaupel (1996). Determinants of longevity: genetic, environmental and medical factors. *Journal of Internal Medicine*, vol. 240, pp. 333-341.
- Cohen, Jonathan, Allan Gaw, Robert I. Barnes, Katherine T. Landschulz and Helen H. Hobbs (1996). Genetic factors that contribute to inter-individual variations in plasma low density lipoprotein-cholesterol levels. *1996 Variation in the Human Genome*. pp. 194-210. Ciba Foundation Symposium, 197. Chichester: Wiley.
- Corbo, R. M., R. Scacchi, L. Mureddu, G. Mulas and G. Alfano (1995). Apolipoprotein E polymorphism in Italy investigated in native plasma by a simple polyacrylamide gel isoelectric focusing technique. Comparison with frequency data of other European populations. *Annals of Human Genetics*, vol. 59, pp. 197-209.
- Corder, E. H., A. M. Saunders, W. J. Strittmatter, D. E. Schmechel, P. C. Gaskell, G. W. Small, A. D. Roses, J. L. Haines and M. A. Pericak-Vance (1993). Gene dose of apolipoprotein E type 4 allele and the risk of Alzheimer's disease in late onset families. *Science*, vol. 261, No. 5123, pp. 921-923.
- Corder, E. H., A. M. Saunders, W. J. Strittmatter, D. E. Schmechel, P. C. Gaskell JR., J. B. Rimmler, P. A. Locke, P. M. Conneally, K. E. Schmechel, R. E. Tanzi, J. F. Gusella, G. W. Small, A. D. Roses, M. A. Pericak-Vance and J. L. Haines (1995). Apolipoprotein E, survival in Alzheimer's disease patients, and the competing risks of death and Alzheimer's disease. *Neurology*, vol. 45, No. 7, pp. 1323-1328.
- Corder, Elizabeth H., Lars Lannfelt, Matti Viitanen, Larry S. Corder, Kenneth G. Manton, Bengt Winblad and Hans Basun (1996). Apolipoprotein e genotype determines survival in the oldest old (85 years or older) who have good cognition. *Archives of Neurology*, vol. 53, No. 5, pp. 418-422.
- Diesfeldt, H. F., L. R. van Houten and R. M. Moerkens (1986). Duration of survival in senile dementia. *Acta Psychiatrica Scandinavica*, vol. 73, pp. 366-371.
- Eichner, June E., Lewis H. Kuller, Trevor J. Orchard, Gregory A. Grandits, Lisa M. McCallum, Robert E. Ferrell and James D. Neaton (1993). Relation of apolipoprotein E phenotype to myocardial infarction and mortality from coronary artery disease. *American Journal of Cardiology*, vol. 71, pp. 160-165.
- Evans, Denis A., Laurel A. Beckett, Terry S. Field, Lin Feng, Marilyn Albert, David A. Bennett, Benjamin Tycko and Richard Mayeux (1997). Apolipoprotein E  $\epsilon 4$  and incidence of Alzheimer's disease in a community population of older persons. *Journal of the American Medical Association*, vol. 277, pp. 822-824.
- Farrer, Lindsay A., L. Adrienne Cupples, Jonathan L. Haines, Bradley Hyman, Walter A. Kukull, Richard Mayeux, Richard H. Myers, Margaret A. Pericak-Vance, Neil Risch, Cornelia M. van Duijn and APOE and Alzheimer's Disease Meta Analysis Consortium (1997). Effects of age, sex, and ethnicity on the association between apolipoprotein E genotype and Alzheimer's disease. *Journal of the American Medical Association*, vol. 287, pp. 1349-1356.
- Gerdes, Lars Ulrick, Ib Christian Klausen, Inger S  hm and Ole Faergeman (1992). Apolipoprotein E Polymorphism in a Danish population compared to findings in 45 other study populations around the world. *Genetic Epidemiology*, vol. 9, pp. 155-167.
- Hallman, D. M., E. Boerwinkle, N. Saha, C. Sandholzer, H. J. Menzel, A. Csazar and G. Utermann (1991). The apolipoprotein E polymorphism: a comparison of allele frequencies and effects in nine populations. *American Journal of Human Genetics*, vol. 49, No. 2, pp. 338-349.
- Hendrie, H. C., K. S. Hall, S. Hui, F. W. Unverzagt, C. E. Yu, D. K. Lahiri, A. Sahota, M. Farlow, B. Musick, C. A. Class, A. Brashear, V. E. Burdine, B. O. Osuntokun, A. O. Ogunniyi, O. Gureje, W. Baiyewu and G. D. Schellenberg (1995a). Apolipoprotein E genotypes and Alzheimer's disease in a community study of elderly African Americans. *Annals of Neurology*, vol. 37, pp. 118-120.
- Hendrie, Hugh C., Benjamin O. Osuntokun, Kathleen S. Hall, Adesola O. Ogunniyi, Siu L. Hui, Frederick W. Unverzagt and others (1995b). Prevalence of Alzheimer's disease and dementia in two communities: Nigerian Africans and African Americans. *American Journal of Psychiatry*, vol. 152, No. 10, pp. 1485-1492.
- Heng, C. K., N. Saha and J. S. Tay (1995). Lack of association of apolipoprotein E polymorphism with plasma lip(a) levels in the Chinese. *Clinical Genetics*, vol. 48, No. 3, pp. 113-119.
- Hong, C. J., T. Y. Liu, H. C. Liu, S. J. Wang, J. L. Fuh, C. W. Chi, K. Y. Lee and C. B. Sim (1996).  $\epsilon 4$  allele of apolipoprotein E increases risk of Alzheimer's disease in a Chinese population. *Neurology*, vol. 46, No. 6, pp. 1749-1751.
- Kamboh, K. I., K. K. Bhatia and R. E. Ferrell (1990). Genetic studies of human apolipoproteins. XII. Population genetics of apolipoprotein polymorphisms in Papua New Guinea. *American Journal of Human Biology*, vol. 2, pp. 17-23.
- Kao, J. T., K. S. Tsai, C. J. Chang and P. C. Huang (1995). The effects of apolipoprotein E polymorphism on the distribution of lipids and lipoproteins in the Chinese population. *Atherosclerosis*, vol. 114, No. 1, pp. 55-59.

- Kukull, Walter A., Gerard D. Schellenberg, James D. Bowen, Wayne C. McCormick, Chang-En Yu, Linda Teri, Jill D. Thompson, Ellen S. O'Meara and Eric B. Larson (1996). Apolipoprotein E in Alzheimer's disease risk and case detection: a case-control study. *Journal of Clinical Epidemiology*, vol. 49, No. 10, pp. 1143-1148.
- Kuusisto, Johanna, Keijo Koivisto, Kari Kervinen, Leena Mykkänen, Eeva-Liisa Helkala, Matti Vanhanen, Tuomo Hänninen, Kalevi Pyörälä, Y. Antero Kesäniemi, Paavo Riekkinen and others (1994). Association of apolipoprotein E phenotypes with late onset Alzheimer's disease: population based study. *British Medical Journal*, vol. 309, No. 6955, pp. 636-638.
- Louhija, J., H. E. Miettinen, K. Kontula, M. J. Tikkanen, T. A. Miettinen and R. S. Tilvis (1994). Aging and genetic variation of plasma apolipoproteins. Relative loss of the apolipoprotein e4 phenotype in centenarians. *Arteriosclerosis & Thrombosis*, vol. 14, No. 7, pp. 1084-1089.
- Mak, Y. T., H. Chiu, J. Woo, R. Kay, Y. S. Chan, E. Hui, K. H. Sze, C. Lum, T. Kwok and C. P. Pang (1996). Apolipoprotein E genotype and Alzheimer's disease in Hong Kong elderly Chinese. *Neurology*, vol. 46, No. 1, pp. 146-149.
- Osuntokun, B. O., A. Sahota, A. O. Ogunniyi, O. Gureje, O. Baiyewu, A. Adeyinka, S. O. Oluwale, O. Komolafe, K. S. Hall, F. W. Unverzagt and others (1995). Lack of an association between apolipoprotein E epsilon 4 and Alzheimer's disease in elderly Nigerians. *Annals of Neurology*, vol. 38, No. 3, pp. 463-465.
- Preston, Samuel H., Irma T. Elo, Ira Rosenwaike and Mark Hill (1996). African-American mortality at older ages: results of a matching study. *Demography*, vol. 33, No. 2, pp. 193-209.
- Schächter, François, Laurence Faure-Delanef, Frédérique Guénou Guénou, Hervé Rouger, Philippe Froguel, Laurence Lesueur-Ginot and Daniel Cohen (1994). Genetic associations with human longevity at the APOE and ACE loci. *Nature Genetics*, vol. 6, No. 1, pp. 29-32.
- Septhemia, B., M. I. Kamboh, L. L. Adams-Campbell, M. Nwankwo and R. E. Ferrell (1988). Genetic studies of human apolipoproteins. VII. Population distribution of polymorphisms of apolipoproteins A-I, A-II, A-IV, C-II, E, and H in Nigeria. *American Journal of Human Genetics*, vol. 43, pp. 847-853.
- Stengård, Jari H., Kim E. Zerba, Juha Pekkanen, Christian Ehnholm, Aulikki Nissinen and Charles F. Sing (1995). Apolipoprotein E polymorphism predicts death from coronary heart disease in a longitudinal study of elderly Finnish men. *Circulation*, vol. 91, pp. 265-269.
- Tang, M. X., G. Maestre, W. Y. Tsai, X. H. Liu, L. Feng, W. Y. Chung, M. Chun, P. Schofield, Y. Stern, B. Tycko and R. Mayeux (1996). Relative risk of Alzheimer's disease and age-at-onset distributions, based on APOE genotypes among elderly African Americans, Caucasians, and Hispanics in New York City. *American Journal of Human Genetics*, vol. 58, No. 3, pp. 574-584.
- United Nations, Department for Economic and Social Information and Policy Analysis, Statistical Division (1993). *Demographic Yearbook. Special Issue: Population Aging and the Situation of Elderly Persons*. New York: United Nations. (ST/ESA/STAT/SER.R./22).
- Wilson, Peter W. F., Ernst J. Schaefer, Martin G. Larson and Jose M. Ordovas (1996). Apolipoprotein E alleles and risk of coronary disease: A meta-analysis. *Arteriosclerosis Thrombosis and Vascular Biology*, vol. 16, pp. 1250-1255.
- World Health Organization Monica Project (1994). Ecological analysis of the association between mortality and major risk factors of cardiovascular disease. *International Journal of Epidemiology*, vol. 23, No. 3, pp. 505-516.
- Zekraoui, L., J. P. Lagarde, A. Raisonniere, N. Gérard, A. Aouizerate and G. Lucotte (1997). High frequency of the apolipoprotein E \*4 allele in African Pygmies and most of the African populations in sub-Saharan Africa. *Human Biology*, vol. 69, No. 4, pp. 575-581.

## **XVI. NUTRITION, ALL-CAUSE AND CARDIOVASCULAR MORTALITY ITS POSSIBLE MODULATION BY OTHER FACTORS, ESPECIALLY PHYSICAL EXERCISE**

*H. Kesteloot\**

Important differences in mortality levels exist world-wide in countries at all levels of development, including the more industrialised. These countries are characterised by low levels of mortality due to infectious diseases and cancer. Important changes in mortality and in mortality patterns have occurred during the last 50 years but the direction of the change and its magnitude vary between countries. Much can be learned by comparing mortality levels and patterns between countries and relating them to their different life styles.

### **A. MORTALITY RATES OF VARIOUS POPULATIONS**

The mortality rates of 42 populations for both sexes in the age classes 55-64 years and 65-74 years are given for all causes in table 58 and for cardiovascular mortality in table 59. The countries are ranked according to the male mortality rate in the age class 55-64 years. The sex ratio (M/F) of mortality is also given. The means, standard deviations and the correlation between the sexes are given in table 60. The important differences between countries are readily apparent. Mortality in the Eastern European countries and the former Soviet-Union is very high. In Western Europe mortality is highest in Denmark, Northern Ireland and Scotland. Within China (selected regions) mortality is higher in rural compared to urban regions. A high degree of correlation for all-cause and total cardiovascular mortality exists between the sexes, although female mortality is always markedly lower. This demonstrates that essentially the same factors are influencing mortality in men and women,

but at a lower level in women. Total cardiovascular mortality accounts in the age class 55-64 years for 41 per cent and 37 per cent of all-cause mortality in men and women respectively, and for 47 per cent and 49 per cent respectively in the age class 65-74 years.

### **B. MORTALITY TRENDS IN THE PERIOD 1950-1994**

These trends will be discussed in a limited number of countries, chosen as representative of their respective regions. Only all-cause and total cardiovascular mortality data are presented. Death is the only mortality parameter that does not need standardisation. Total cardiovascular mortality is preferred to ischaemic heart disease mortality as the data on the latter are not completely reliable. Data for the sexes are given separately as the trends may differ markedly between the sexes. The data are age-adjusted 45-74 years according to the Standard European population, and smoothed over a period of 3 years.

The trends in all-cause mortality for men are shown in figure 98 and for women in figure 99. In men the different trends can be observed: an increase in Hungary; a decrease in Japan and from 1970 onwards on in the United States of America, Belgium and England and Wales; a relative status quo in Sweden and the Netherlands. For women the downward trend is almost universal except for Hungary. Up to the early 1960s Hungary had a lower mortality for both sexes than Japan but in 1994 they are at opposite extremes of the spectrum.

The trends in total cardiovascular mortality are similar to those of all-cause mortality for both sexes. A marked decrease occurred in men in the United States of America from

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TABLE 58. ALL-CAUSE MORTALITY RATES PER 100,000, RANKED ACCORDING TO MALE MORTALITY IN THE AGE CLASS 55-64, 42 SELECTED COUNTRIES, 1992-1994

Country	Year	Men 55-64	Women 55-64	M/F 55-64	Men 65-74	Women 65-74	M/F 65-74
Switzerland	1994	988	516	1.91	2 833	1 338	2.12
Japan	1994	1 039	440	2.36	2 387	1 133	2.11
Sweden	1993	1 060	592	1.79	2 967	1 608	1.85
Australia	1993	1 108	606	1.83	2 966	1 605	1.85
Greece	1994	1 115	501	2.23	2 779	1 548	1.79
Hong Kong	1994	1 140	557	2.05	2 977	1 635	1.82
Canada	1993	1 179	666	1.77	3 051	1 652	1.85
Israel	1993	1 181	730	1.62	3 024	2 033	1.49
Netherlands	1994	1 215	651	1.87	3 424	1 667	2.05
Albania	1993	1 234	494	2.50	3 232	1 520	2.13
China: urban	1994	1 238	851	1.45	3 898	2 577	1.51
England and Wales	1994	1 242	733	1.69	3 617	2 127	1.70
Norway	1993	1 250	641	1.95	3 471	1 710	2.03
Spain	1992	1 267	491	2.58	2 957	1 358	2.18
Italy	1992	1 280	560	2.29	3 118	1 513	2.06
New Zealand	1993	1 296	838	1.55	3 368	1 977	1.70
France	1993	1 354	523	2.59	2 844	1 211	2.35
Austria	1994	1 397	621	2.25	3 339	1 761	1.90
Ireland	1992	1 406	834	1.69	4 191	2 314	1.81
Finland	1994	1 411	550	2.56	3 532	1 682	2.10
Portugal	1994	1 420	610	2.33	3 443	1 731	1.99
Germany	1994	1 422	647	2.20	3 525	1 883	1.87
United States of America	1992	1 482	855	1.73	3 374	1 971	1.71
Singapore	1994	1 483	895	1.66	3 785	2 382	1.59
Denmark	1993	1 486	995	1.49	3 982	2 387	1.67
Northern Ireland	1994	1 502	844	1.78	3 798	2 216	1.71
Chile	1992	1 573	864	1.82	3 515	2 111	1.67
Scotland	1994	1 622	974	1.67	4 288	2 585	1.66
China: rural	1994	1 644	1 074	1.53	4 469	2 750	1.63
Korea, Republic of	1994	1 716	710	2.42	4 229	2 104	2.01
Argentina	1991	1 902	850	2.24	3 914	1 956	2.00
Czech Republic	1993	2 203	947	2.33	4 954	2 637	1.88
Bulgaria	1993	2 314	968	2.39	4 831	2 823	1.71
Poland	1994	2 381	931	2.56	4 881	2 503	1.95
Belarus	1993	3 029	1 136	2.67	5 147	2 743	1.88
Lithuania	1994	3 037	1 059	2.87	5 372	2 531	2.12
Hungary	1994	3 049	1 215	2.51	5 699	2 969	1.92
Kazakstan	1994	3 217	1 369	2.35	6 290	3 441	1.83
Ukraine	1992	3 252	1 392	2.34	6 161	3 529	1.75
Estonia	1994	3 536	1 285	2.75	5 817	2 891	2.01
Latvia	1994	3 979	1 422	2.80	6 326	3 004	2.11
Russian Federation	1994	4 177	1 494	2.80	6 741	3 281	2.05

Source: World Health Organization, *World Health Statistics Annual* (Geneva, WHO, 1995).

1970 onwards, and in Japan. The differences in trends between the sexes in Hungary are evident (figures 100 and 101).

### C. CAUSES OF MORTALITY

It is difficult to try to identify the causes of mortality for a whole population. In theory

and probably also in reality, some factors that protect against cardiovascular disease may promote certain cancers. Interactions among causal factors such as between saturated fat intake and smoking (Xie and others, 1991; Kesteloot and others, 1994) also play a role. The causes of mortality will only be identified if they offer an explanation for the differences in mortality between and within populations



TABLE 59. TOTAL CARDIOVASCULAR MORTALITY RATES PER 100,000, RANKED ACCORDING TO MALE MORTALITY  
IN THE AGE CLASS 55-64, 42 SELECTED COUNTRIES, 1992-1994

Country	Year	Men 55-64	Women 55-64	M/F 55-64	Men 65-74	Women 65-74	M/F 65-74
Japan	1994	255	107	2.38	654	371	1.76
Hong Kong	1994	280	135	2.08	830	563	1.47
France	1993	292	89	3.28	823	342	2.41
Switzerland	1994	303	104	2.90	1 054	450	2.34
Spain	1992	354	121	2.93	980	510	1.92
Italy	1992	389	135	2.88	1 105	552	2.00
Australia	1993	403	157	2.58	1 270	619	2.05
Portugal	1994	408	177	2.31	1 347	757	1.78
Canada	1993	417	158	2.64	1 194	560	2.13
Korea, Republic of	1994	430	258	1.66	1 412	908	1.55
Netherlands	1994	435	154	2.82	1 366	615	2.22
Chile	1992	443	230	1.92	1 179	728	1.62
Greece	1994	444	161	2.76	1 204	744	1.62
China: rural	1994	463	339	1.37	1 500	978	1.53
Sweden	1993	467	149	3.13	1 494	634	2.36
China: urban	1994	468	345	1.36	1 644	1 176	1.40
Israel	1993	472	226	2.09	1 354	888	1.52
Denmark	1993	505	231	2.19	1 642	802	2.05
Albania	1993	507	227	2.24	1 619	813	1.99
Norway	1993	511	155	3.29	1 644	676	2.43
Germany	1994	514	180	2.86	1 559	798	1.95
Austria	1994	536	174	3.08	1 592	819	1.94
England and Wales	1994	551	211	2.61	1 671	870	1.92
New Zealand	1993	566	239	2.37	1 559	847	1.84
United States of America	1992	586	260	2.25	1 426	753	1.89
Singapore	1994	603	346	1.74	1 531	1 098	1.39
Ireland	1992	636	252	2.53	2 012	965	2.09
Finland	1994	647	162	3.98	1 788	818	2.19
Scotland	1994	723	323	2.24	2 079	1 142	1.82
Northern Ireland	1994	730	296	2.47	1 893	1 025	1.85
Argentina	1991	823	323	2.55	1 841	915	2.01
Czech Republic	1993	1 012	377	2.68	2 694	1 464	1.84
Poland	1994	1 025	376	2.73	2 524	1 389	1.82
Bulgaria	1994	1 211	500	2.42	3 034	1 907	1.59
Hungary	1994	1 222	473	2.59	2 843	1 640	1.73
Lithuania	1994	1 293	447	2.89	2 849	1 541	1.85
Belarus	1993	1 436	608	2.36	2 868	1 804	1.59
Kazakstan	1994	1 445	685	2.11	3 391	2 140	1.58
Estonia	1994	1 638	587	2.79	3 386	1 882	1.80
Ukraine	1992	1 645	815	2.02	3 858	2 569	1.50
Russian Federation	1994	1 955	776	2.52	3 906	2 202	1.77
Latvia	1994	2 015	723	2.79	3 778	1 913	1.97

Source: World Health Organization, *World Health Statistics Annual* (Geneva, WHO, 1995).

and help explain ongoing trends. Many factors are perceived both by researchers and by the public as being related to health (Kesteloot, 1996). Both quality (morbidity) and duration (mortality) of life should be considered, but they are strongly related. (To live long, one should remain healthy until old age.) Causes of mortality are discussed with special attention to the importance of nutrition and physical exercise.

### 1. The level of medical care

Medicine has played an important role in reducing mortality and morbidity world-wide, especially through the introduction of hygienic measures and vaccinations. As a result, infectious disease has been almost eliminated in the industrialised world. New epidemics, however, always remain a possibility, as shown by the emergency of acquired immuno-

TABLE 60. MORTALITY RATES PER 100,000 PER YEAR, 42 SELECTED COUNTRIES, 1992-1994

		Mean	SD	Sex ratio <sup>+</sup>	SD	r <sup>#</sup>
All-cause mortality						
Age class 55-64	Men	1 805	865	2.14	0.42	0.901
	Women	832	285			
Age class 65-74	Men	4 012	1 130	1.88	0.20	0.935
	Women	2 152	617			
Total cardiovascular mortality						
Age class 55-64	Men	739	471	2.51	0.51	0.952
	Women	304	194			
Age class 65-74	Men	1 890	874	1.86	0.27	0.968
	Women	1 052	548			

Source: World Health Organization, *World Health Statistics Annual* (Geneva, WHO, 1995).

+ : Sex ratio, males/females, mean  $\pm$  SD.

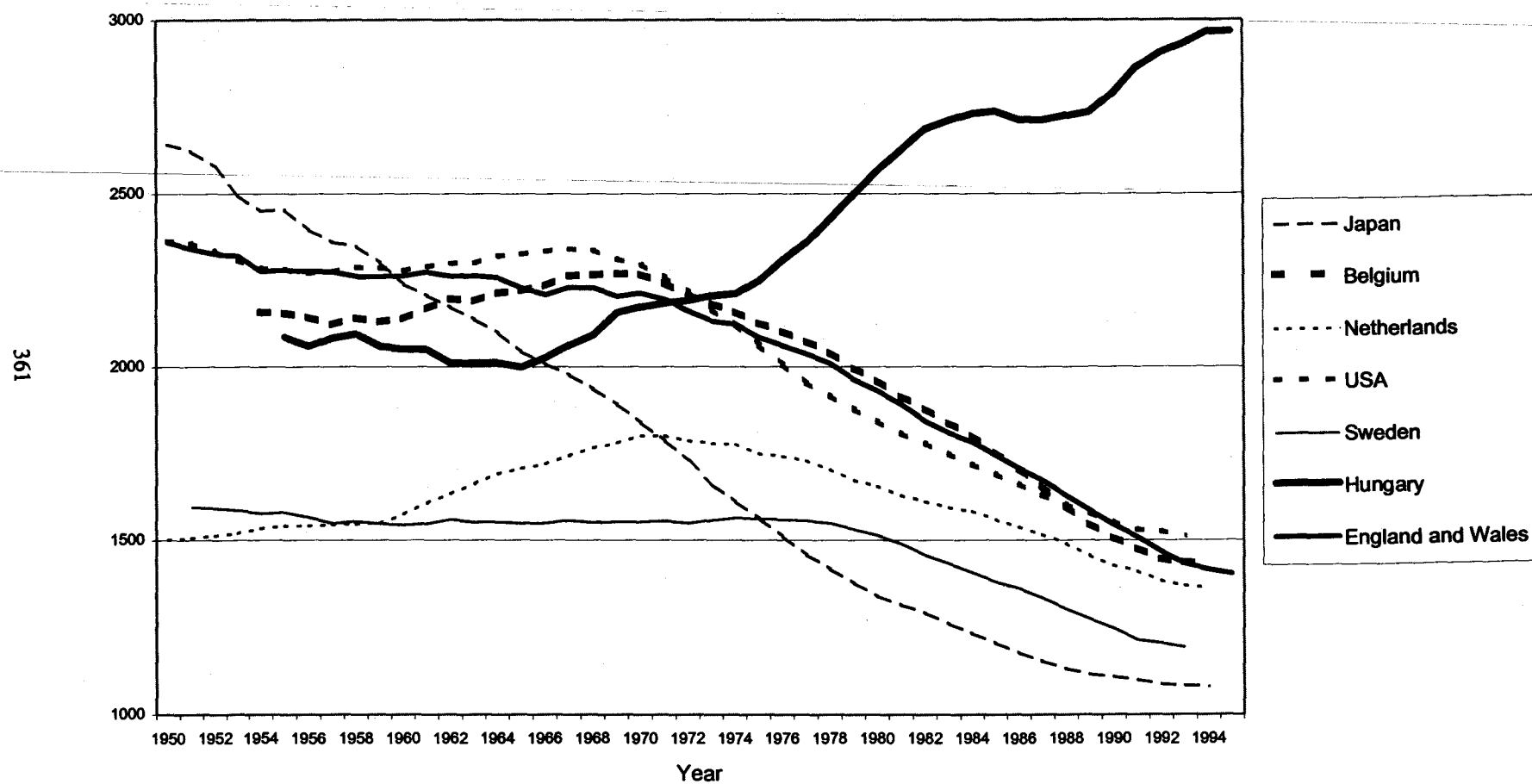
# : r : correlation coefficient between the sexes.

deficiency syndrome (AIDS). The level of medical care is perceived by the public as the most important determinant of mortality and, as a result, the population accepts large financial burdens in order to attain this goal. However, the level of medical care is more effective in reducing certain aspects of morbidity than mortality. Countries such as Albania and urban China have lower levels of medical knowledge, research and expenditure for health than the United States of America, but a lower mortality. The same is true, but to a lesser extent for Greece and Spain. The percentage of the gross domestic product (GDP) spent for health care, as published by the Organization for Economic Co-operation and Development (OECD) for a number of countries in 1994 does not correlate significantly with the mortality levels of the populations concerned (Organization for Economic Co-operation and Development, 1996). Most countries with low mortality levels such as Sweden, Japan, Spain and Greece belong to the lower third of the expenditure list, confirming the fact that low mortality is also associated with low morbidity. The higher mean age of these populations does not result in comparable higher expenditures for health care. The level of medical care and related health expenditures thus cannot explain the large differences in mortality between countries.

The OECD published data on total expenditure on health for 27 developed countries in 1996. The relationship between changes in all-cause mortality and changes in expenditure in purchasing power parity (PPP\$) and in changes in percentage of GDP spent on health, both expressed in percentage, were estimated for the period 1965-1990. In men, no significant correlation exists ( $p = 0.65$  and  $p = 0.56$  respectively), but in women the relationship with PPP\$ was significant ( $p = 0.015$ ) and with percentage change in GDP borderline ( $p = 0.07$ ). The reasons for this different behaviour between the sexes are not clear, but the mortality changes are more homogeneous in women compared to men. Moreover only countries with decreasing mortality rates were included in the analysis; no data were available for the Eastern European countries, where mortality rates were actually increasing during the period considered. Nor were low income countries with low mortality such as China and Albania included (Gjonça and others, 1997).

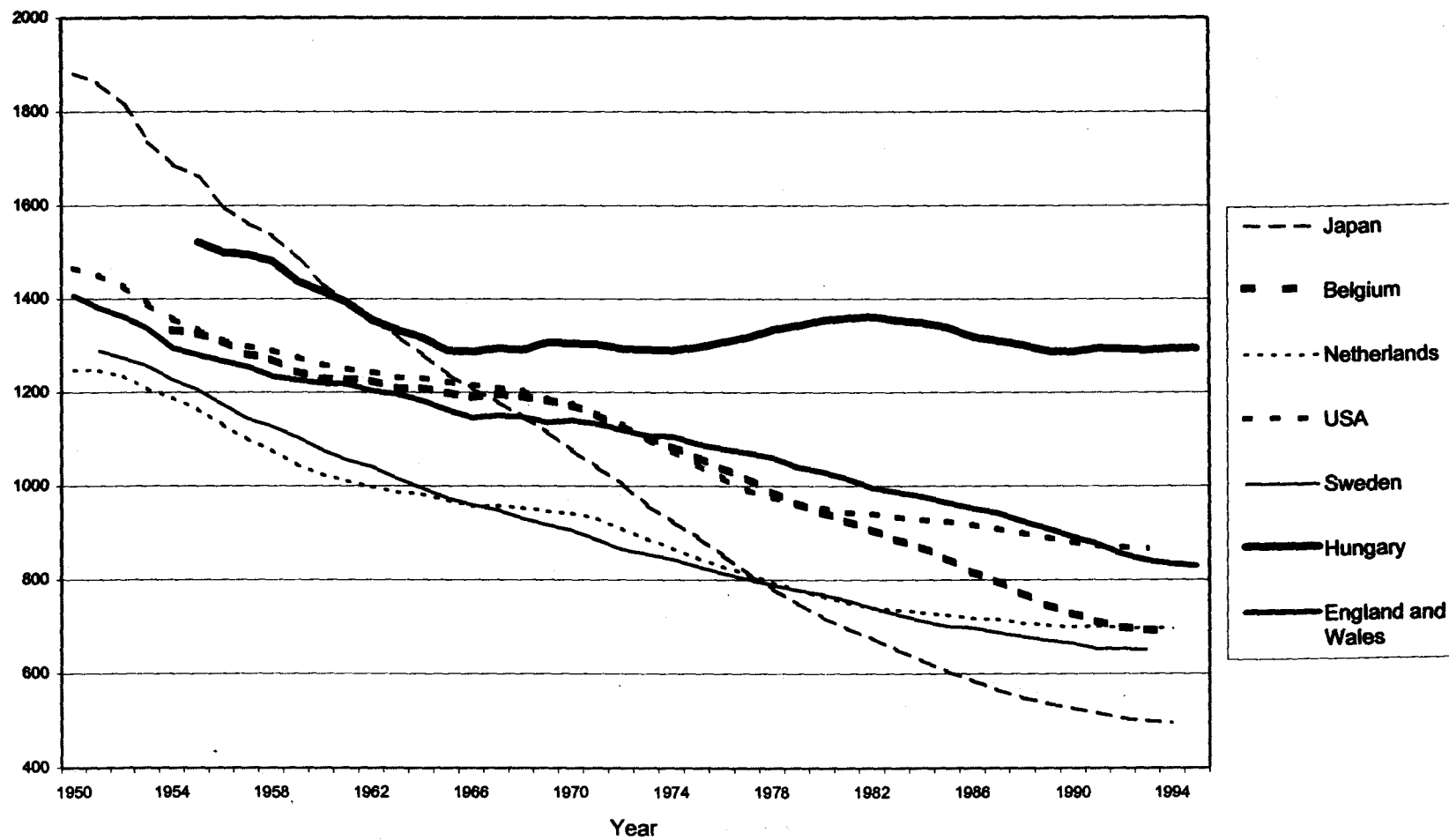
It has been claimed that above the age of 80 life expectancy is higher in the United States of America than in other countries with a high life expectancy at birth. This has been attributed to a better level of medical care for older people in the United States of America (Manton and Vaupel, 1995). However, the

**Figure 98. Male mortality rates, all causes, selected developed countries, 1950s to 1990s**



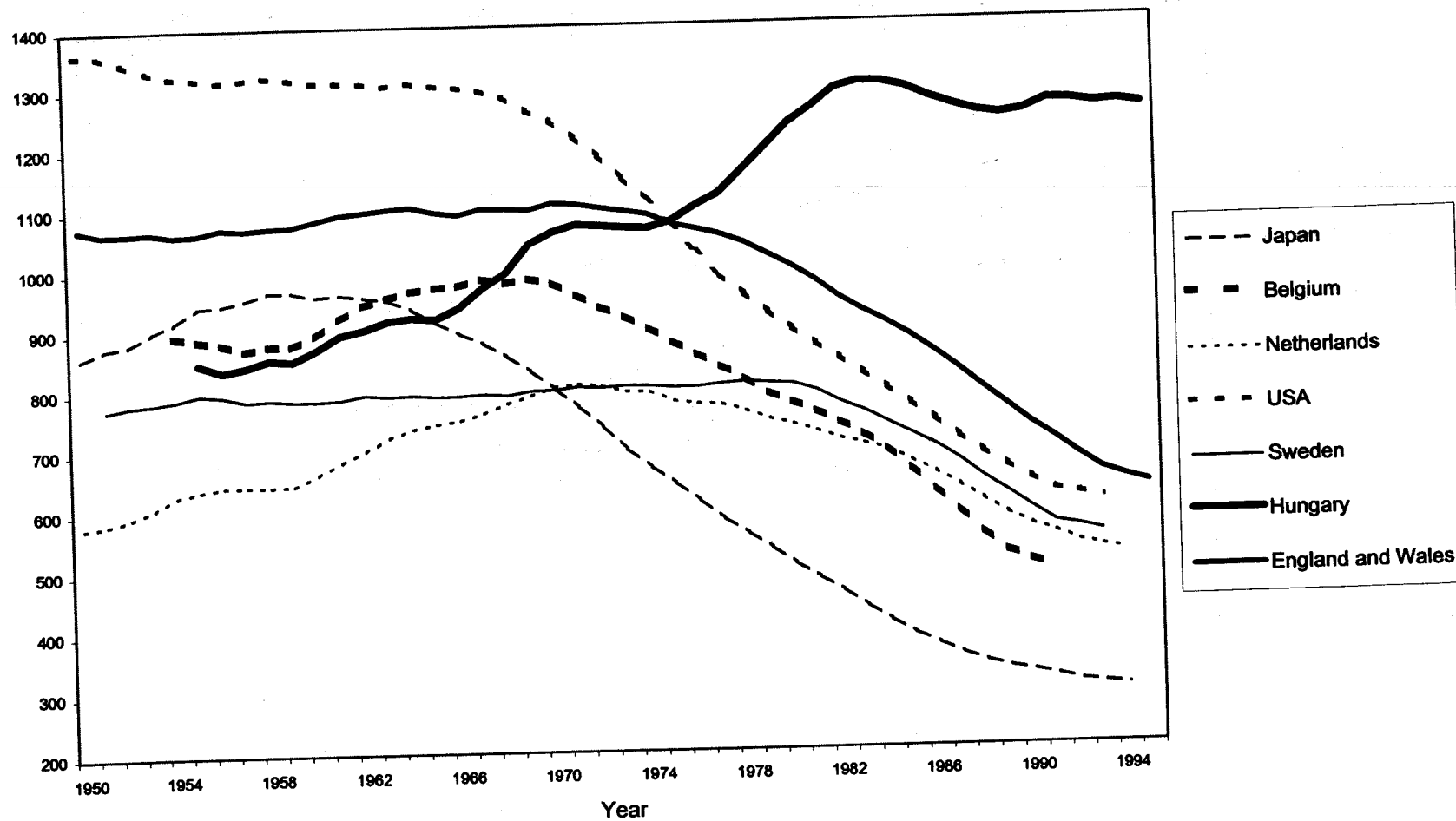
Source: World Health Organization, *World Health Statistics Annual, 1995* (Geneva, WHO, 1995).

**Figure 99. Female mortality rates, all causes, selected developed countries, 1950s to 1990s**



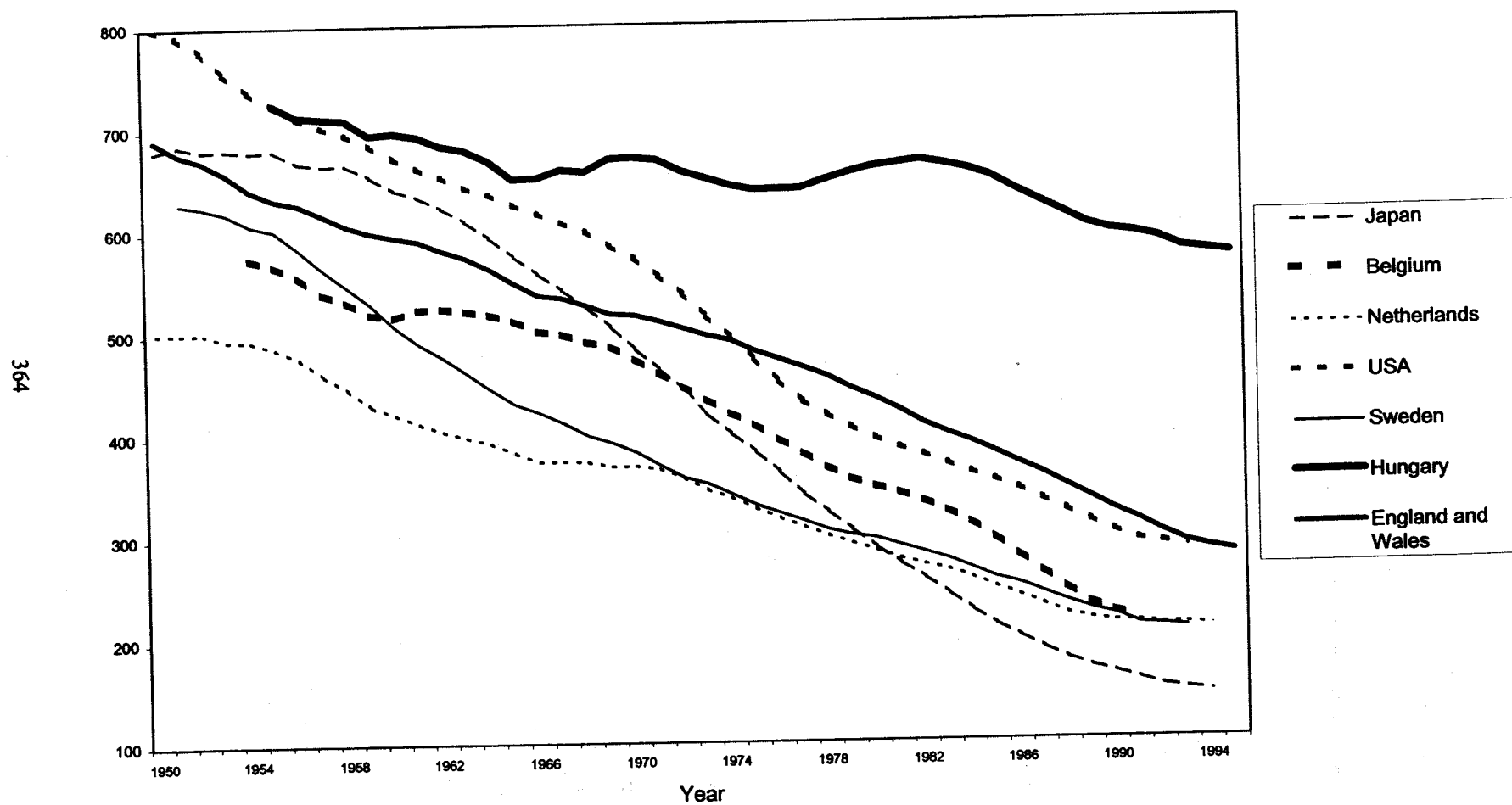
Source: World Health Organization, *World Health Statistics Annual, 1995* (Geneva, WHO, 1995).

**Figure 100. Male mortality rates, cardiovascular causes, selected developed countries, 1950s to 1990s**



Source: World Health Organization, *World Health Statistics Annual, 1995* (Geneva, WHO, 1995).

**Figure 101. Female mortality rates, cardiovascular causes, selected developing countries, 1950s to 1990s**



Source: World Health Organization, *World Health Statistics Annual, 1995* (Geneva, WHO, 1995).

authors admit that above the age of 80, blacks have higher life expectancies than whites. In the 29 states in the United States of America for which separate death rates for whites and blacks are available for the period 1989-1991, a crossover of mortality rates occurs. Blacks have lower mortality than whites above a median age of 85 (range 77-92) in men and 87 (range 79-89) in women (National Center for Health Statistics, 1998). Crossover ages were also obtained using extrapolation of the Gompertz equations obtained in blacks and whites in the age range 35-74 and 35-84. The crossover can be better explained as a selective survival of the fittest under unfavourable conditions and not by a better level of medical care. Blacks who survive until the crossover age are on average biologically fitter than whites at the same age.

## 2. Genetic factors

Genetic factors are given great importance both by the medical profession and the general public. They are crucial in several rare diseases and contribute to mortality and morbidity in some relatively common pathological conditions such as familial heterozygotic hypercholesterolemia. The generally determined levels of lipoprotein(a) and of apolipoprotein E are predictors of risk at the individual level (Rhoads and others, 1986; Sandholzer and others, 1992; Marshall and others, 1996). These effects are modulated by dietary factors. It has been shown, however, that migrant populations, in one or two generations, acquire the mortality and morbidity patterns of the host populations. More than one million people of Japanese origin live in Brazil but their life expectancy is about 10 years less than in Japan. The most important argument, however, against a dominant role of genetic factors is the rapid ongoing changes in mortality, in opposite directions, occurring during the last 30 years. The genetic pool of a population can only change to a limited extent over such a short period. Some changes in the genetic pool are possible if one postulates that those who previously died of e.g. tuberculosis, were genetically different from the rest of the population.

Genetic factors are important at the population level as can be shown by comparing peo-

ple with nearly identical life styles such as persons living in religious communities, prisons or psychiatric wards. They still have differences in serum lipid levels, blood pressure, degree of obesity, among others. But the differences are markedly less than in a free-living population. Genetic factors also explain most of the differences in mortality between the sexes. However, the major differences in the distribution of risk factors and in patterns of mortality differences between populations cannot be explained by genetic factors.

## 3. Pollution

Denmark, Scotland and Northern Ireland belong to the Western European countries with the highest mortality, but are among those with the lowest degree of pollution. Hungary, also with a very high mortality, is mostly an agrarian state. Hong Kong on the contrary is considered one of the highest polluted cities in the world, but has a low mortality. In a recent publication it was considered that only 3 per cent of all-cause mortality in Czechoslovakia, a country with a high mortality, could possibly be explained by pollution (Bobak and Feachem, 1995). At the population level the influence of pollution is presumably small, but remains to be exactly defined.

## 4. Stress

It is a popular notion that stress is an important cause of death. Stress remains difficult to define but it is generally accepted that it is high in Japan and very high in Hong Kong, regions with high life expectancies. In Belgium mortality was lower in 1942, in the midst of the Second World War, than in the 10 years preceding and following that year. Stress becomes important at the individual level once heart disease is established but is unimportant as a cause of death at the population level.

## 5. Obesity

World-wide obesity is increasing in industrialised countries but mortality is decreasing. Obesity, however, plays a role in the development of hypertension, heart disease and

diabetes among others. In general it is one of the causes of the so-called insulin resistance syndrome. However, a definite increase in mortality only occurs above a body mass index (BMI) ( $\text{kg/m}^2$ ) of 30 units. At the population level it plays only a subordinate role, but negatively influences the quality of life.

#### 6. Food additives

Food additives are frequently mentioned by the general population as a possible cause of cancer. Scientific evidence, however, is scanty and it is highly improbable that Sweden, Greece and Japan would use different additives than populations with a higher cancer or all-cause mortality.

#### 7. Socio-economic factors

Social class is an important determinant of mortality but its importance varies from country to country. It is especially evident in such countries as the United States of America and Great Britain. Universal health coverage by social security such as in Great Britain does not eliminate the differences. The lower socio-economic classes not only belong by definition to the lower education and income brackets but they also have different nutritional habits: they consume more meat, more salt, less fruit and vegetables, are more obese, smoke more, have different patterns of alcohol consumption (Joossens and others, 1980; Marmot and others, 1984; Marmot and others, 1991; Diez-Roux and others, 1995; Krebs-Smith and others, 1995). The question is how much of the observed differences in mortality can be attributed to these differences in life style. Moreover, socio-economic factors do not appear to be important between countries, e.g. the relatively low mortality in Albania and in urban China. In view of the great importance of social factors for the quality of life, the role of socio-economic factors remains to be more clearly defined.

#### 8. Smoking

Smoking is undoubtedly one of the major causes of death in Western countries, increasing cancer and ischemic heart disease

mortality (Peto and others, 1992). It explains part of the difference in mortality between men and women (Zhang and others, 1995; Valkonen and Van Poppel, 1997) and may partially explain the comparatively high female mortality in Scotland and Denmark where a high proportion of women smoke, but not in rural China where few women do so. The significant relationship between cigarette smoking and mortality at the population level is not equally important in all countries. Some countries with the highest smoking levels in the world, e.g., Japan, Cuba and Greece, have among the lowest mortalities from all causes and even from ischemic heart disease and lung cancer. Epidemiological evidence points to the importance of a synergistic effect of cigarette smoking and saturated fat intake on mortality (Kesteloot and others, 1994). Cigarette smoking is especially harmful in Western countries where saturated fat intake is high.

#### 9. Summary

Many factors act on mortality and important interactions between these factors exist. For this reason the most important factors acting on mortality have been treated separately, trying to define their relative importance in explaining the differences in mortality at the population level. The conclusion is that although each of the factors considered may be important, they cannot explain the differences in mortality between countries. However, they act as confounding factors that should be taken into account when the effects on mortality of nutrition and physical exercise are discussed.

#### D. NUTRITION AND MORTALITY

Tremendous differences in nutritional patterns exist world-wide. Culinary experts mention the Japanese, Korean and Chinese diet, the Indonesian and Indian diet, the French and Mediterranean diet, among others. Vegetarian, lacto-vegetarian, or macrobiotic diets are consumed by entire populations or parts of populations. Dietary habits are dictated in some cases by religious beliefs. Large differences also exist in the population's per-



ceptions of what is tasty. Western populations for example, but not Oriental populations such as the Chinese, like cheese. Raw fish is a delicacy for some populations, but is rejected by others.

The study of nutrition at the population level is very difficult. Representative population samples must be obtained and a standardised method used for the evaluation of consumption. Moreover, dietary habits change with time, and the diet-related diseases appearing now may be due to the dietary habits of 10 or 20 years ago. Taking this time lag into account is especially relevant for the study of the relationship between nutrition and cancer. Nutrition is in itself a very complex problem. Constituents of food include different types of fat, saturated, monounsaturated, polyunsaturated, omega-3 and omega-6 fatty acids, chains of fatty acids of different lengths, trans-fatty acids, single and complex carbohydrates, vegetal and animal protein, anti-oxidants, fibre, alcohol, and various cations among which sodium is the most important. Moreover, the nutritional intake of an individual is limited so that eating fish, for example, will result, in eating less meat. If fish intake is found to be beneficial, it is very difficult to establish whether or not it is the absence of meat (and meat fat) that makes the difference.

There is strong evidence linking saturated fat to atheromatous diseases, especially ischemic heart disease and stroke (Kesteloot, 1992). As mentioned, these diseases are leading causes of death in industrialised countries. The link between saturated fat and atheromatous diseases can be considered causal on the basis of existing clinical, experimental and epidemiological evidence. Its effect is modulated by genetic factors, and conditions such as smoking and obesity. The serum cholesterol level, a major risk factor for atheromatous diseases, can be considered a biomarker for the level of saturated fat intake of the population. Dietary sodium is a major cause of stroke, especially due to its effect on blood pressure. Epidemiological evidence points to saturated fat as a strong promoter of certain cancers, e.g. lung, rectum, prostate and breast cancer (Kesteloot and others, 1994) and to salt as a cause of stomach cancer (Joossens

and others, 1996). The intake of fat also influences coagulation and thrombosis responsible for acute coronary and cerebrovascular events, but this relationship remains largely unexplored at the population level. Moderate alcohol consumption, especially of wine, appears to protect against atheromatous diseases (Sasaki and Kesteloot, 1994). Wine producing countries such as those of the Mediterranean in general have low mortality. Salt intake and saturated fat correlate strongly with stroke mortality (Sasaki and others, 1995). Nutrition can be considered the major determinant of the differences in mortality between countries.

#### E. REGIONAL DIFFERENCES IN MORTALITY IN RELATION TO NUTRITION

Regional differences in mortality exist within countries, such as between East and West-Finland, the North and South of France, Scotland versus England and Wales, and the North and South of Belgium. The evidence points to differences in nutritional patterns as the most plausible explanation (Kesteloot, 1989; Den Hond and others, 1994; Den Hond and others, 1995). In Belgium regional differences in butter consumption are significantly related to the serum cholesterol levels and to regional mortality levels from all causes and from ischemic heart disease (Kesteloot and others, 1984). Important differences in mortality exist between the North and the South of Belgium. Mortality is lower in the North, and this has been related to a lower consumption of saturated fat. Significant dietary changes have been documented in the North of Belgium (Flanders), with a change in dietary ratio of polyunsaturated to saturated fat (P/S ratio) from 0.2 in 1960 (Joossens and others, 1966) to 0.6 in 1980 (Kesteloot, 1989). However, research on mortality differences within countries is hampered by a lack of reliable nutritional data.

#### F. NUTRITION AND AGING AT THE POPULATION LEVEL

Between the ages of 35-84, mortality from all causes can be expressed by the Gompertz equation, the log mortality rate as a function

of age, with a high degree of precision for both sexes and for all industrialised countries (Kesteloot and Sasaki, 1994). This is largely independent of the nature of the diseases from which the population is dying, which can differ markedly, as between Japan and Hungary, and of the homogeneity, as in Japan, or heterogeneity, as in United States of America. An example is given in figure 102 for men and in figure 103 for women from a country with a decreasing mortality (Finland) and with an increasing mortality (Hungary). Countries with low mortality have a low intercept and a high slope and countries with a high mortality a high intercept and low slope. The higher the slope of the rise of the mortality rate with age, the lower the mortality. This can be explained by the existence of a maximum life expectancy of the human race. This has been calculated by extrapolation at  $\pm 105$  years in men and  $\pm 109$  years in women (Kesteloot, 1996; Kesteloot and Sasaki, 1994). Some individuals live longer, but they are very exceptional. The slope of the Gompertz equation is inversely related to the speed of ageing of a given population. Above the age of 55 years the slope of the Gompertz equation remains relatively unchanged in men but increases in women. Starting from a fixed point an increase in slope means an increased mortality demonstrating the influence of the menopause on female mortality rate.

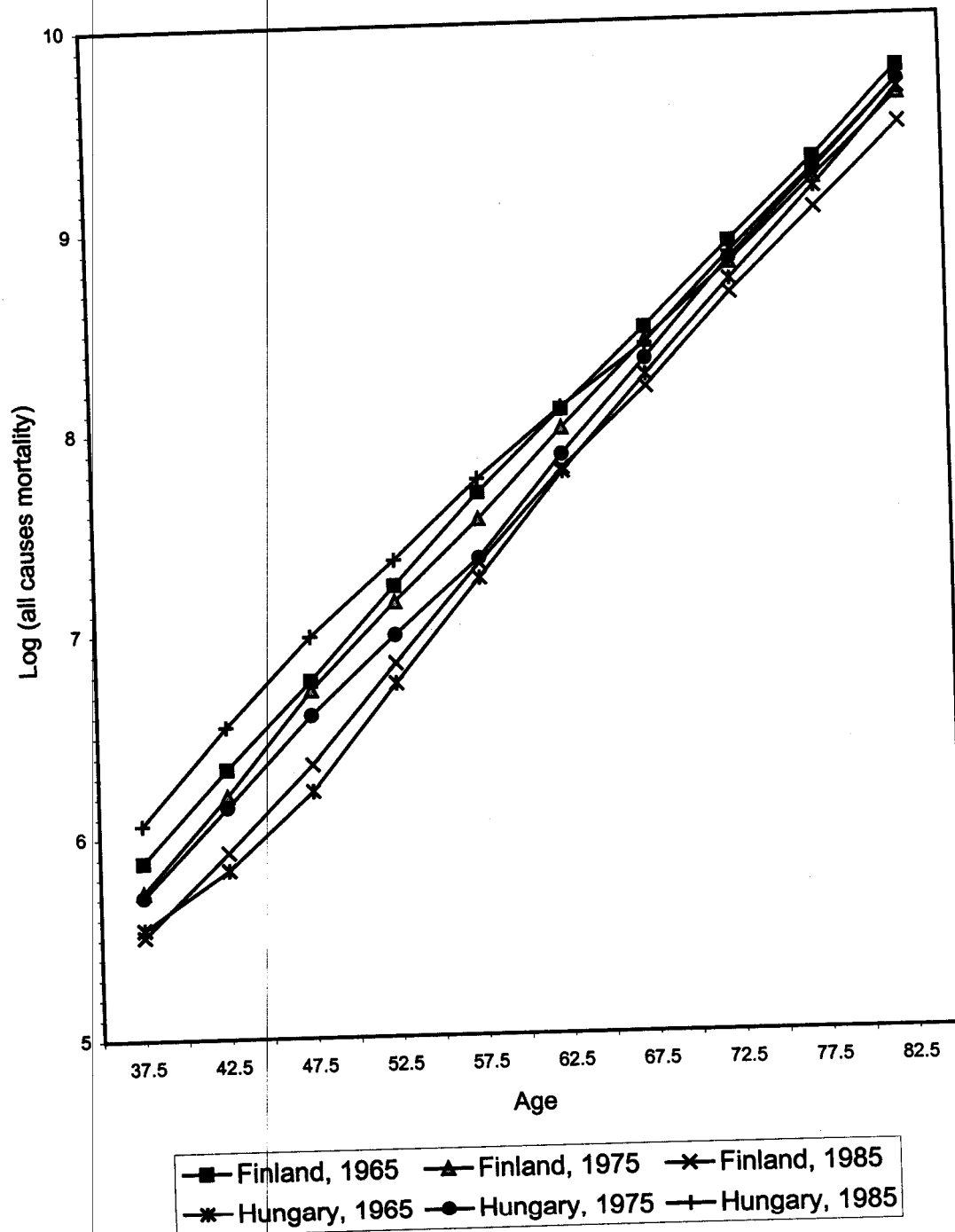
The importance of the Gompertz equation is that it allows defining the impact of mortality from all causes, in a given population at a given time, by two strongly correlated parameters, the intercept and the slope. This characteristic points to a common interception point of the slopes, the so-called point of iso-mortality, which can be shown generally to be close to the age of maximum mortality. When the slope is correlated with the intake of saturated fat and with the P/S ratio, it can be shown that saturated fat correlates positively with mortality and polyunsaturated fat negatively (Kesteloot and Sasaki, 1994). The level of saturated fat intake also correlates significantly with cancer mortality, especially in men (Kesteloot and others, 1994). The research, however, is hampered by a lack of reliable data on the nutritional habits of populations. The study of these nutritional habits should be given high priority.

#### G. PHYSICAL EXERCISE, ALL-CAUSE AND CARDIOVASCULAR MORTALITY

To try to quantify the level of physical exercise at the individual level is always difficult, and at the population level is nearly impossible. Manual workers belong predominantly to the lower social classes, have higher levels of physical activity than non-manual workers, but higher mortality. The dilemma has been clearly exemplified by the Finnish woodcutters in North-Karelia. They performed heavy physical work at low temperatures, had a high total caloric intake with a high intake of saturated fat and a high mortality due to myocardial infarction (Puska and Mustaniemi, 1975). A high caloric intake can lead to a high intake of fat in view of its caloric density. At the population level there is no evidence that the level of physical exercise is higher in Japan than in Hungary in order to explain the large differences in mortality between both countries. On the contrary, Hungarians have fewer cars and less automation, but a higher level of athletic performance than the Japanese. On an aggregate basis, the Hungarians historically obtained 16 times more Olympic medals than the Japanese. It should be mentioned also that at the population level both the level of physical exercise and of mortality is declining while at the same time in most Western populations obesity is increasing. This increase in body weight could be partially due to a decrease in cigarette consumption in the population.

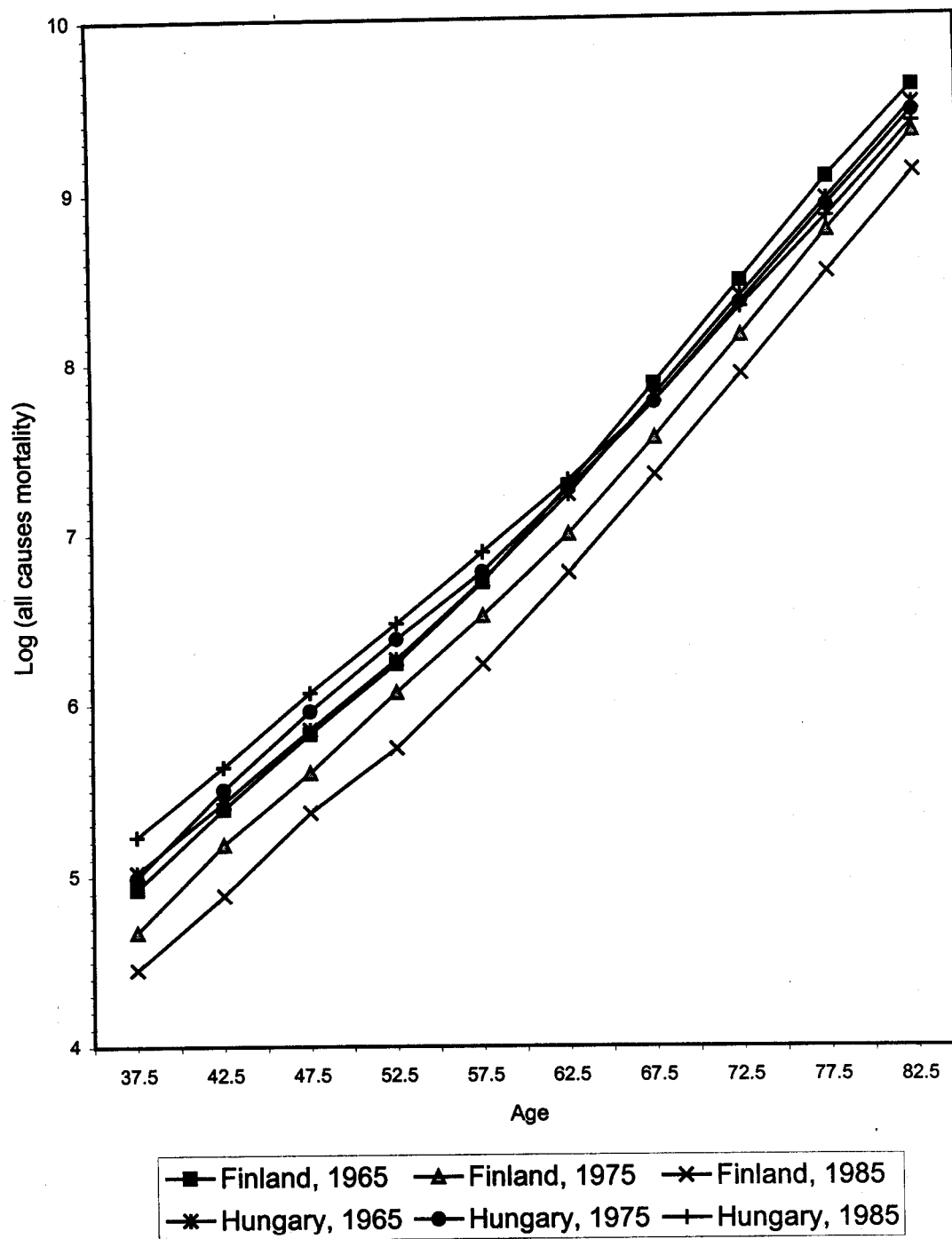
The role of physical activity, especially during leisure time, has been examined in several studies in relation to all-cause and cardiovascular mortality and morbidity within populations. Physical activity has been related to lower all-cause mortality (Paffenbarger and others, 1986; Bauman and Owen, 1991; Rosengren and Wilhelmsen, 1997), to lower cardiovascular mortality (Paffenbarger and others, 1993; Rodriguez and others, 1994; Sandvik and others, 1995; NIH Reports, 1996; Rosengren and Wilhelmsen, 1997) and morbidity (Rodriguez and others, 1994) and to the presence of healthier cardiovascular risk factors (Bauman and Owen, 1991; Twisk and others, 1996). Leisure time physical activity specifically also has been related to lower all-cause mortality (Paffenbarger and others,

Figure 102. Male mortality rates, all causes, Finland and Hungary, 1965, 1975 and 1985



Source: World Health Organization, *World Health Statistics Annual, 1995* (Geneva, WHO, 1995).

Figure 103. Female mortality rates, all causes, Finland and Hungary, 1965, 1975 and 1985



Source: World Health Organization, *World Health Statistics Annual, 1995* (Geneva, WHO, 1995).

1993; Haapanen and others, 1996; Kaplan and others, 1996), to lower cardiovascular mortality (Paffenbarger and others, 1993; Haapanen and others, 1996; Kaplan and others, 1996) and to healthier cardiovascular risk factors (Helmert and others, 1993; Mensink and others, 1996). A high degree of physical activity in children has been related to lower levels of lp(a), an independent cardiovascular risk factor that is to a large extent genetically determined (Taimela and others, 1994). Smokers in the top quartile of physical fitness have lower all-cause mortality than non-smokers in the lowest quartile (Sandvik and others, 1995). Current smokers in the upper tertile of physical fitness also have a lower risk of coronary heart disease than non-smokers in the lowest third (Haapanen and others, 1997). A less pronounced effect of exercise in smokers has also been found. Vigorous physical activity reduced mortality in smokers, but not to the level of non-smokers (Hedblad and others, 1997). However, it is difficult to study the independent relationship between leisure time activity and mortality and morbidity. Many of these physical activities are performed for their health promoting effects by people who are already health-conscious in other ways. They smoke less, are less obese, belong to a higher social class (Folsom and others, 1991; Helmert and others, 1993; Iribarren and others, 1997) and have a healthier diet. Although physical exercise also has a beneficial effect independently of most known cardiovascular risk factors, eliminating completely the effect of confounding factors is

nearly impossible. This is partially a result of the multiplicative effects of interactions between the various risk factors. It is also difficult to evaluate the effect of physical activity on the "quality of life". Group sports or other physical activities such as dancing and aerobics promote social contact, benefits that should also be taken into consideration. Among the negative aspects of physical activity are a propensity to injuries and an increase in sudden death in competitive athletes, which can only be partially eliminated by appropriate screening (Maron and others, 1996). A too vigorous exercise level may even increase the heart attack rate (Shaper and Wannamethee, 1991) and mortality (Paffenbarger and others, 1986).

#### H. CONCLUSION

This review of mortality factors suggests that nutrition is the most important determinant at the population level. Recommendations to the public concerning nutrition and other life style factors should be easily understandable and non-dogmatic with exception for the recommendations against smoking. Occasional deviations are not too important as far as the recommendations are applied in daily routine. Such a list of recommendations is given in table 61. The recommendations contain the secret of a long and healthy life. They also demonstrate the responsibility of each individual for his or her own life.

TABLE 61. DIETARY RECOMMENDATIONS TO THE POPULATION

- 
1. Avoid saturated fat (especially dairy products).
  2. Limit total fat intake (to about 1/4 of total caloric intake).
  3. Limit salt intake (to less than 5 g/day).
  4. Limit intake of dietary cholesterol (eggs).
  5. Eat more complex carbohydrates (bread, potatoes, rice, a.o.).
  6. Eat more fruits and vegetables (anti-oxidantia, fiber, potassium).
  7. Use moderate amounts of alcohol with a preference for red wine (upper limit: 2 conventional glasses a day).
  8. Eat more fish (including fatty fish).
- 

In addition: do not smoke, maintain an optimal body weight and perform regularly a fair amount of physical activity.

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The perceived causes are not ranked according to their potential impact on mortality.

## REFERENCES

- Bauman, A., and N. Owen (1991). Habitual physical activity and cardiovascular risk factors. *Medical Journal of Australia*, vol. 154, pp. 22-28.
- Bobak, M., and R. G. A. Feachem (1995). Air pollution in Central and Eastern Europe: an estimate of the impact. *European Journal of Public Health*, vol. 5, pp. 82-86.
- Den Hond, E., M. De Schryver, A. Muylaert, E. Lesaffre and H. Kesteloot (1994). The Inter-regional Belgian Bank Employee Nutrition Study (IBBENS). *European Journal of Clinical Nutrition*, vol. 48, pp. 106-117.
- Den Hond, E. M., E. E. Lesaffre and H. Kesteloot (1995). Regional differences in consumption of 103 fat products in Belgium: a supermarket-chain sales approach. *Journal of the American College of Nutrition*, vol. 14, No. 6, pp. 621-627.
- Diez-Roux, A. V., J. Nieto, H. A. Tyroler, L. D. Crum and M. Szklo (1995). Social inequalities and atherosclerosis. The Atherosclerosis Risk in Communities Study. *American Journal of Epidemiology*, vol. 141, No. 10, pp. 960-972.
- Folsom, A. R., T. C. Cook, J. M. Sprafka, G. L. Burke, S. W. Norsted, D. R. Jacobs, Jr. (1991). Differences in leisure-time physical activity levels between blacks and whites in population-based samples: the Minnesota Heart Survey. *Journal of Behavioural Medicine*, vol. 14, pp. 1-9.
- Gjonca, A., C. Wilson and J. Falkingham (1997). Paradoxes on health transition in Europe's poorest country: Albania, 1950-90. *Population and Development Review*, vol. 23, pp. 585-609.
- Haapanen, N., S. Miilunpalo, I. Vuori, P. Oja and M. Pasanen (1996). Characteristics of leisure time physical activity associated with decreased risk of premature all-cause and cardiovascular disease mortality in middle-aged men. *American Journal of Epidemiology*, vol. 143, pp. 870-880.
- \_\_\_\_\_ (1997). Association of leisure time physical activity with the risk of coronary heart disease, hypertension and diabetes in middle-aged men and women. *International Journal of Epidemiology*, vol. 26, pp. 739-747.
- Hedblad, B., M. Ögren, S. O. Isacson, L. Janzon (1997). Reduced cardiovascular mortality risk in male smokers who are physically active. *Archives of Internal Medicine*, vol. 157, pp. 893-899.
- Helmert, U., B. Herman and S. Shea (1993). Moderate and vigorous leisure-time physical activity and cardiovascular disease risk factors in West Germany, 1984-1991. *International Journal of Epidemiology*, vol. 24, pp. 285-292.
- Iribarren, C., R. V. Luepker, P. G. McGovern, D. K. Arnett and H. Blackburn (1997). Twelve-year trends in cardiovascular disease risk factors in the Minnesota Heart Survey. *Archives of Internal Medicine*, vol. 157, pp. 873-881.
- Joossens, J. V., J. Claessens, J. Geboers and J. H. Claes (1980). Electrolytes and creatinine in multiple 24-hour urine collections (1970-1974). In *Epidemiology of Arterial Blood Pressure*, H. Kesteloot and J. V. Joossens, Eds. The Hague-Boston-London: Martinus Nijhoff Publishers, pp. 45-63.
- Joossens, J. V., M. J. Hill, P. Elliott, R. Stamler, J. Stamler, E. Lesaffre, A. Dyer, R. Nichols and H. Kesteloot (1996). Dietary salt, nitrate and stomach cancer mortality in 24 countries. *International Journal of Epidemiology*, vol. 25, pp. 494-504.
- Joossens, J. V., G. Verdonk and R. Pannier (1966). "Normal" serum cholesterol in Belgium as related to age and diet. A comparison with other countries. *Acta Cardiologica*, vol. 21, pp. 431-445.
- Kaplan, G. A., W. J. Strawbridge, R. D. Cohen and L. R. Hungerford (1996). Natural history of leisure-time physical activity and its correlates: associations with mortality from all causes and cardiovascular disease over 28 years. *American Journal of Epidemiology*, vol. 144, pp. 793-797.
- Kesteloot, H. (The B.I.R.N.H. Study) (1989). Nutrition and health: the conclusions of the B.I.R.N.H. Study. *Acta Cardiologica*, vol. 44, No. 2, pp. 183-194.
- Kesteloot, H. (1992). Nutrition and health. *European Heart Journal*, vol. 13, pp. 120-128.
- \_\_\_\_\_ (1996). Nutrition and ageing at the population level. *Verhandelingen Koninklijke Academie voor Geneeskunde van België*, vol. 58, pp. 117-139.
- \_\_\_\_\_, J. Bande, J. Pille, J. Geboers, E. Vertenten, J. De Hemptinne and O. Van Houte (1984). Serum lipid distribution and mortality in Belgium. *European Heart Journal*, vol. 5, pp. 778-783.
- Kesteloot, H. E. and S. Sasaki (1994). Nutrition and the ageing process: a population study. *American Journal of Geriatric Cardiology*, vol. 3, pp. 8-19.
- \_\_\_\_\_, G. Verbeke and J. V. Joossens (1994). Cancer mortality and age: relationship with dietary fat. *Nutrition and Cancer*, vol. 22, pp. 85-98.
- Krebs-Smith, S. M., D. A. Cook, A. F. Subar, L. Cleveland and J. Friday (1995). US adult's fruit and vegetables intakes 1989 to 1991. *American Journal of Public Health*, vol. 85, pp. 1623-1629.
- Manton, K. A., and J. W. Vaupel (1995). Survival after the age of 80 in the United States, Sweden, France, England and Japan. *New England Journal of Medicine*, vol. 333, pp. 1232-1235.
- Maron, B. J., J. Shirani, L. C. Poliac, R. Mathenge, W. C. Roberts and F. O. Mueller (1996). Sudden deaths in young competitive athletes. *Journal of the American Medical Association*, vol. 276, pp. 199-204.
- Marmot, M. G., G. Davey Smith, S. Stansfeld and others (1991). Health inequalities among British civil servants: The Whitehall II Study. *Lancet*, vol. 337, pp. 1387-1393.
- Marmot, M. G., M. J. Shipley and G. Rose (1984). Inequalities in death: specific explanations of a general pattern. *Lancet*, vol. 1, pp. 1003-1006.
- Marshall, J. A., M. I. Kambok, D. H. Besseren, S. Hoog, R. F. Kamman and R. E. Ferrell (1996). Association between dietary factors and serum lipids by apolipoprotein E polymorphism. *American Journal of Clinical Nutrition*, vol. 63, pp. 87-95.
- Mensink, G. B. M., M. Deketh, M. D. M. Mul, A. J. Schuit and H. Hoffmeister (1996). Physical activity and its association with cardiovascular risk factors and mortality. *Epidemiology*, vol. 7, pp. 391-397.
- National Center for Health Statistics (1998). US Decennial Life Tables 1989-91. Vol. II, State Life Tables.
- NIH Reports (1996). NIH develops consensus statement on the role of physical activity for cardiovascular health. *Special Medical Reports*, vol. 54, pp. 763-767.
- Organization for Economic Co-operation and Development (OECD) (1996). Health data, 1996. CREDES, electronic editors catalogue (CD-ROM). Paris: OECD.
- Paffenbarger, R. S., R. T. Hyde, A. L. Wing and C. C. Hsieh (1986). Physical Activity, all-cause mortality and longevity of college alumni. *New England Journal of Medicine*, vol. 314, pp. 605-613.
- Paffenbarger, R. S., Jr., R. T. Hyde, A. L. Wing, I. M. Lee, D. L. Jung and J. B. Kampert (1993). The association of changes in physical-activity level and other lifestyle characteristics with mortality among men. *New England Journal of Medicine*, vol. 328, pp. 538-545.
- Peto, R., A. D. Lopez, J. Boreham, M. Thun, C. Heath (1992). Mortality from tobacco in developed countries: indirect estimation from national vital statistics. *Lancet*, vol. 339, pp. 1268-1278.

- Puska, P., and H. Mustaniemi (1975). Incidence and presentation of myocardial infarction in North Karelia, Finland. *Acta Medica Scandinavica*, vol. 197, pp. 211-216.
- Rhoads, G. G., G. Dahlen, K. Berg, N. E. Morton and A. L. Dannenberg (1986). Lp(a) lipoprotein as a risk factor for myocardial infarction. *Journal of the American Medical Association*, vol. 256, pp. 2540-2544.
- Rodriguez, B. L., J. D. Curb, C. M. Burchfiel, R. D. Abbott, H. Petrovitch, K. Masaki and D. Chiu (1994). Physical activity and 23-year incidence of coronary heart disease morbidity and mortality among middle-aged men. The Honolulu Heart Program. *Circulation*, vol. 89, pp. 2540-2544.
- Rosengren, A., and L. Wilhelmsen (1997). Physical activity protects against coronary death and deaths from all causes in middle-aged men. Evidence from a 20-year follow-up of the Primary Prevention Study in Göteborg. *Annals of Epidemiology*, vol. 7, pp. 69-75.
- Sandholzer, C., N. Saha, J. D. Kark, A. Rees, W. Jaross, H. Dilpinger, F. Hoppichler, E. Boerwinkle and G. Utermann (1992). Apo(a) isoforms predict risk for coronary heart disease. A study of six populations. *Atherosclerosis and Thrombosis*, vol. 12, pp. 1214-1226.
- Sandvik, L., J. Erikssen, M. Ellestad, G. Erikssen, E. Thaulow, R. Mundal and K. Rodahl (1995). Heart rate increase and maximal heart rate during exercise as predictors of cardiovascular mortality: a 16-year follow-up study of 1960 healthy men. *Coronary Artery Disease*, vol. 6, pp. 667-678.
- Sasaki S., and H. Kesteloot (1994). Wine and non-wine alcohol: differential effect on all-cause and cause-specific mortality. *Nutrition and Metabolism Cardiovascular Diseases*, vol. 4, pp. 177-182.
- Sasaki, S., and X. H. Zhang and H. Kesteloot (1995). Dietary sodium, potassium, saturated fat, alcohol, and stroke mortality. *Stroke*, vol. 26, pp. 783-789.
- Shaper, A. G., and G. Wannamethee (1991). Physical activity and ischaemic heart disease in middle-aged British men. *British Heart Journal*, vol. 66, pp. 384-394.
- Taimela, S., J. S. A. Viikari, K. V. K. Porkka and G. H. Dahlen (1994). Lipoprotein (a) levels in children and young adults: the influence of physical activity. The Cardiovascular Risk in Young Finns Study. *Acta Paediatrica*, vol. 83, pp. 1258-1263.
- Twisk, J. W. R., H. C. G. Kemper, G. J. Mellenbergh, W. van Mechelen and G. B. Post (1996). Relation between the longitudinal development of lipoprotein levels and lifestyle parameters during adolescence and young adulthood. *Annals of Epidemiology*, vol. 6, pp. 246-256.
- Valkonen, T., and F. Van Poppel (1997). The contribution of smoking to sex differences in life expectancy. *European Journal of Public Health*, vol. 7, pp. 302-310.
- World Health Organization (1995). *World Health Statistics Annual, 1995*. Geneva: WHO.
- Xie, J., E. Lesaffre and H. Kesteloot (1991). The relationship between animal fat intake, cigarette smoking, and lung cancer. *Cancer Causes and Control*, vol. 2, pp. 79-83.
- Zhang, X. H., S. Sasaki and H. Kesteloot (1995). The sex ratio of mortality and its secular trends. *International Journal of Epidemiology*, vol. 24, pp. 720-729.

## XVII. ALCOHOL AND SMOKING AS RISK FACTORS

Alan Lopez\*

Numerous definitions, paradigms or models of health status and how it may be modified have been proposed and one or more of these are generally used to guide the health policy and planning process in countries, and at the international level. With scientific advances about the causes of disease and ill-health, and with increasing concern about the cost of health care provision, these paradigms have begun to focus more and more on the importance of primary exposures or risk factors which underlie the incidence of many diseases and injuries. While this exposure-outcome sequence is a factor in communicable disease occurrence, it is of particular importance in preventing chronic diseases and injuries, and in this regard, cigarette smoking and alcohol use are among the leading causes of death and disability.

This paper begins with a review of the types of studies that have been carried out to quantify the individual hazards of smoking and alcohol use for various diseases and injuries, and then considers estimated current levels of exposure to these two substances in different parts of the world. Next, population-level attributable risks (as opposed to individual risks) from smoking and alcohol use are summarised. The final section of the paper reviews the various policy responses to smoking and alcohol, and discusses the extent to which these have been adopted in various parts of the world, and how effective they might be.

### A. ASSESSING INDIVIDUAL HAZARDS FROM SMOKING AND ALCOHOL

#### 1. *Cigarette smoking*

Of all the risks to human health, perhaps none has been studied so extensively as tobacco use,

and particularly cigarette smoking.<sup>1</sup> Although the possible consequences of smoking for health had been suspected for several hundred years, reliable scientific assessment of tobacco hazards is a comparatively recent development. Following concern about rising lung cancer incidence in some populations, focussed epidemiological studies were carried out in the early 1950s to assess the risks arising from various exposures, including smoking. One of these was carried out in the United Kingdom by Richard Doll and Austin Bradford-Hill, who concluded that "smoking is a factor, and an important factor, in the production of carcinoma of the lung" (Doll and Hill, 1950). Their findings stimulated a massive research effort and a global response to tobacco control, which must rank as one of the major public health successes of the 20th century. More than 55,000 publications on tobacco hazards have since been prepared, and this literature is growing by about 2,000 publications every year (Ford, 1994).

#### *Methods for assessing health hazards of smoking*

Reliable scientific evidence on the hazards of smoking is based on observational studies comparing the incidence of disease in populations who smoke with incidence in populations of lifelong non-smokers. To begin with, these studies typically focussed on a single disease (e.g. lung cancer) which was of interest for some reason (such as rising incidence) and compared the characteristics of persons who had the disease (cases) with those who did not (controls). This type of epidemiological investigation is known as a *case-control* study and is a retrospective enquiry, usually carried out on a relatively small number of cases and controls (typically 100-200). Apart from the relatively small sample size, this type of study design suffers from a number of limitations including:

- Biases may result from the choice of controls. To minimise confounding by

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other (possibly unknown) factors, cases and controls should ideally be matched in all respects thought likely to influence risk of the disease under investigation, including age, sex, social class, marital status, medical history and so on. It is rarely possible to achieve such matching, although recently some innovative study designs have been proposed that might reduce the problems of bias (e.g. the "spouse-control: design pioneered by Richard Peto and colleagues in China - see Liu and others (1998)).

- Case-control methods only allow investigation of smoking risks for one disease at a time (cases) and a single study provides no evidence on how risks change over time with changing exposure.

This last point is particularly important. There is now considerable evidence to suggest that the hazards of tobacco use for major diseases increase with duration of exposure (e.g. USDHHS, 1989). Repeated case-control studies on identical populations using identical methods would be required, strictly speaking, if trends in risk were to be assessed, and then for a single disease only.

To overcome these difficulties, follow-up or longitudinal studies have been proposed and carried out in various countries. Following the pioneering case-control study of lung cancer published in 1950 by Doll and Hill, Doll began a large prospective evaluation of more than 40,000 British doctors, the follow-up of whom is still continuing today! This study, and others like it, uses a baseline assessment of smoking habits and other variables (with periodic subsampling to monitor changes in prevalence) to determine person-years of exposure according to various categories of exposure. Deaths of persons in the cohort are then linked to person-years for corresponding categories to calculate death rates. These rates in turn are compared to rates for specific diseases among lifelong non-smokers to determine the relative risk (RR) of death for smokers versus non-smokers. Since the study design is prospective, relative risks can be calculated for any disease of interest, and, by calculating RRs at different time periods, the evolution of the tobacco epidemic (in

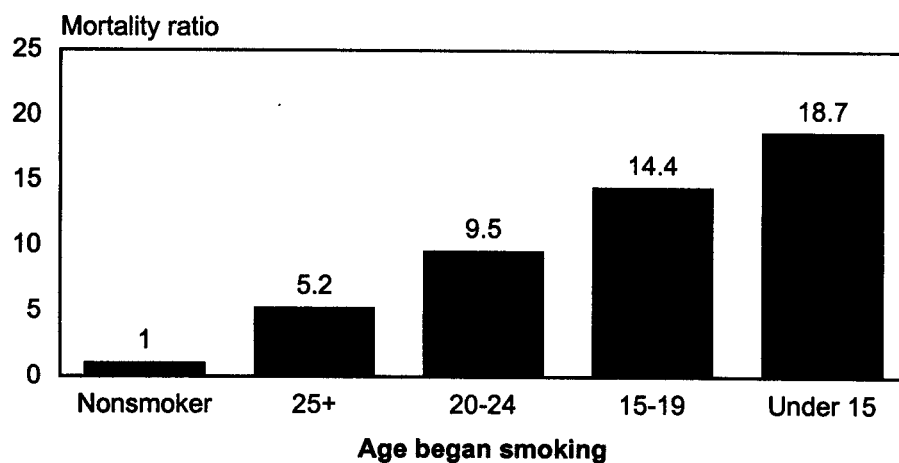
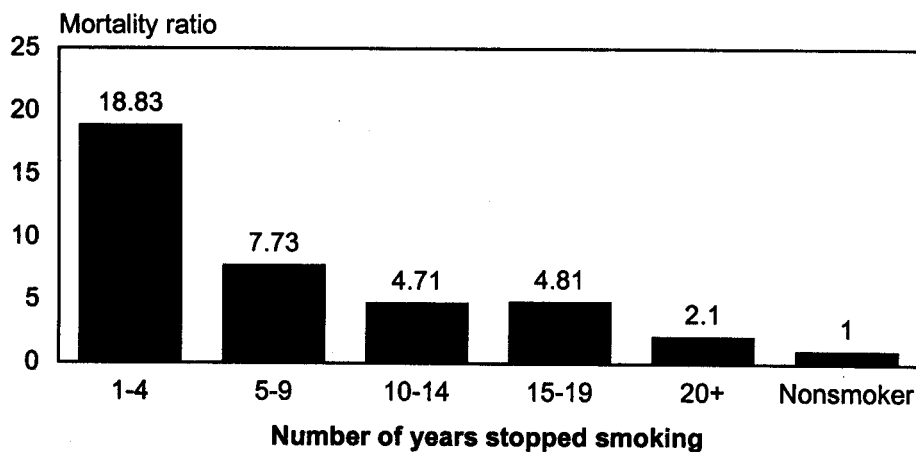
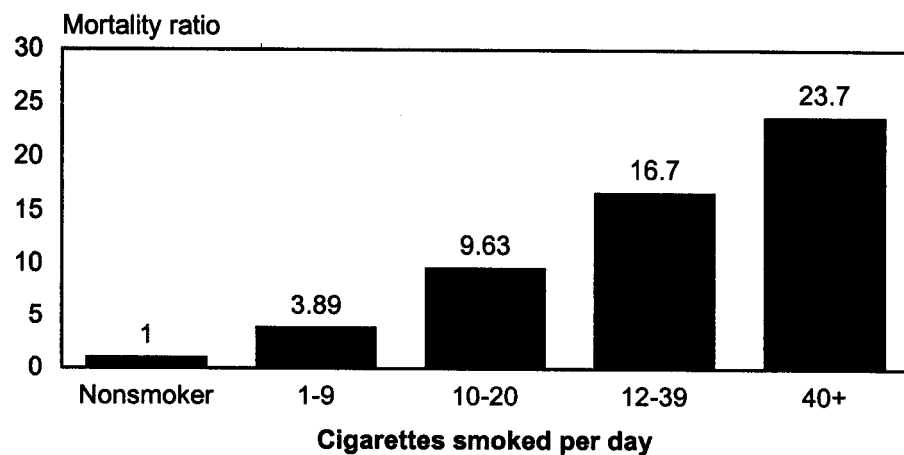
terms of mortality) can be monitored provided follow-up is sufficiently long.

Although methodologically perhaps more appealing than case-control studies, prospective studies are much more expensive, require that vital registration and cause of death coding systems (at least for the cohort but usually more generally) are in place, and that reliable follow-up of the cohort can be sustained for 10-20 years. Because of their design, the first results on tobacco hazards in the cohort usually only appear some 3-5 years after recruitment, whereas for case-control studies, the results are immediate.

Even though such studies may reveal large differences in risk (with death rates often 10-20 times higher in smokers than non-smokers), for some diseases it is difficult to decide how much of the excess mortality observed in smokers is actually caused by smoking, and how much is due to other factors which are more common among smokers and which increase the risk of disease. Only careful, intervention studies to produce disease would resolve this issue and they are not possible since to do so would be clearly unethical. Therefore, proof of causation in the strict sense is not possible although proof in the legal sense (i.e. beyond reasonable doubt) can be, and has been, established based on the accumulated evidence about the association between smoking and disease.

This evidence has focussed on the strength of the association, the dose-response relationships that have been observed (with increasing risks for greater amounts smoked), and on the observation that risk declines follow the cessation of smoking. To illustrate these latter two factors, data on lung cancer risk from a large prospective study carried out in the United States in the 1960s are shown in figure 104. Claims about tobacco hazards also need to be consistent with what is known about changing tobacco use patterns in populations (for example, between males and females) and, where possible, with laboratory studies or with corroborative biological evidence (for example, about changes in respiratory function following changes in smoking). Issues of causality are discussed more fully in Doll (1986).

**Figure 104. Mortality ratios for various exposure variables, USA Veterans study, 1954-1962**



Source: H. A. Kahn, "The Dorn study of smoking and mortality among U.S. veterans: Report on eight and one-half years of observation", in *Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases*, W. Haenszel, ed., NCI Monograph 19 (Washington, D.C., National Cancer Institute, 1966).

## *Review of evidence on health risks of smoking*

Of the numerous studies which have been carried out into the health effects of smoking, only a handful are large enough and cover a sufficiently long time period to yield information likely to be sufficiently reliable to document hazards. Most of these were cohort studies initiated in the 1950s, 1960s and 1970s, with follow-up mortality analyses pertaining to this period as well. An overview of such studies is given in table 62. All are limited to the study of mortality, and all give quantitatively similar results in terms of the relative risks of smoking for various diseases (and for all causes of death), despite the fact that the cohorts were recruited from countries as diverse as the United States of America, Sweden, Japan, Canada and the United Kingdom.

In the United States, United Kingdom and Canada, where men had been smoking in large numbers for decades before these studies were carried out, smoking was typically associated with a 70-80 per cent excess mortality from all causes. The relative risks varied substantially by disease, being largest for cancers of the lung and upper-aero digestive tract (mouth, pharynx, larynx and oesophagus), and lowest for vascular diseases. Typically, lung cancer death rates were 10-12 times higher in smokers than non-smokers, with the notable exception of Japanese men and Swedish women for whom the relative excess was 3-4 times that of non-smokers. Similarly, the all-cause mortality ratios (relative risks) were substantially lower in these two studies, reflecting the fact that tobacco use in the two populations had been much lower than for other cohorts shown in the table.

Two large cohort studies have produced more recent evidence on health hazards from smoking which have emphasised the increasing hazards of smoking with longer duration of use. These two studies are the 40-year follow-up of the 1951 British doctors cohort (see Doll and others, 1994) and the second American Cancer Society Cancer Prevention Study (CPS-II) cohort of over 1.2 million adults monitored since 1982.

The alarming size of the hazards now observable in populations that have been smoking for many decades is well illustrated by figure 105. Thus in the first 20 years of follow-up of the British doctors cohort (1951-1971), smokers had, on average, about a 1.5 to 2 fold higher death rate at each age, similar to the excess reported in other studies around that time (see table 62). With longer duration of smoking, death rates of smokers have increased substantially so that during the second period of follow-up (1971-1991), smokers in middle age had a three-fold higher death rate than non-smokers. A similar excess mortality ratio was found in the CPS-II cohort based on follow-up in the later half of the 1980s. These relative risks suggest that, on average, a smoker who begins smoking in young adult life and continues to do so has at least a 50 per cent chance of eventually being killed by tobacco, either in middle age or in old age.

The evidence from these two studies on the disease-specific risks associated with smoking is summarised in table 63 (British doctors) and table 64 (CPS-II). The results of the two studies are similar. Current smokers have about a 20-fold higher death rate from lung cancer than never smokers, among whom lung cancer death rates have remained low and constant. Smokers also incur a 10-20 fold excess mortality from chronic obstructive lung disease (primarily chronic bronchitis and emphysema), and a risk of death from major vascular diseases that is about twice that of non-smokers. There is epidemiological evidence to suggest that this is also the case in other populations, as well. For example, based on the two American Cancer Society studies with follow-up for 1950-1965 and 1982-1988 respectively, lung cancer death rates among lifelong never smokers were remarkably constant at 15.7 and 14.7 per 100,000 (age-standardised) for men, and 9.6 and 12.0 for women. Corresponding rates for current smokers for men were 187.1 and 341.3 and for women 26.1 and 154.6 (Thun and others, in press).

The excess mortality of smokers from vascular disease is particularly noteworthy. Since vascular disease death rates are typically much

TABLE 62. MORTALITY RATIOS OF CURRENT CIGARETTE-ONLY SMOKERS, BY CAUSE OF DEATH  
IN EIGHT PROSPECTIVE EPIDEMIOLOGIC STUDIES

Cause of death	British doctors <sup>1</sup>	Males in 25 States <sup>2</sup>		U.S. veterans <sup>1</sup>	Japanese study <sup>4</sup>	Canadian veterans <sup>5</sup>	Males in 9 States <sup>6</sup>	Swedish <sup>7</sup>		Califor- nia Occupa- tions <sup>8</sup>
		45-64	65-79					Males	Females	
<b>All cancers<sup>a</sup> (140-205)</b>		2.14	1.76	2.21	1.62		1.97			
Cancer of lung and bronchus (162-163)	14.0	7.84	11.59	12.14	3.64	14.2	10.73	7.0	4.5	15.9
Cancer of larynx (161)		6.09	8.99	9.96	13.59		13.10			
Cancer of buccal cavity (140-141)	13.0 <sup>b</sup>			4.09	7.04	3.9 <sup>b</sup>	2.80			
Cancer of pharynx (145-148)		9.90 <sup>c</sup>	2.93 <sup>c</sup>	12.54	2.81					1.0
Cancer of esophagus (150)	4.7	4.17	1.74	6.17	2.57	3.3	6.60			
Cancer of bladder and other (181)	2.1	2.20	2.96	2.15	0.98	1.3	2.40	1.8	1.6	0.7
Cancer of pancreas (157)	1.6	2.69	2.17	1.84	1.83	2.1		3.1	2.5	6.0
Cancer of kidney (180)		1.42	1.57	1.45	1.11	1.4	1.50			
Cancer of stomach (151)		1.42	1.26	1.60	1.51	1.9	2.30	0.9	2.3	
Cancer of intestines (152-153)				1.27	1.27	1.4	0.50			0.8
Cancer of rectum (154)	2.7	1.01 <sup>d</sup>	1.17 <sup>d</sup>	0.98	0.91	0.6	0.80			0.9
<b>All cardiovascular disease (330-334, 400-468)</b>		1.90	1.31	1.75			1.57			
CHD (420)	1.6	2.08	1.36	1.74	1.96	1.6	1.70	1.7	1.3	2.0
Cerebrovascular lesions (330-334)	1.3	1.38	1.06	1.52	1.14	0.9	1.30	1.0	1.1	1.8
Aortic aneurysm (nonsyphilitic) (451)	6.6	2.62	4.92	5.24		1.8		1.6		
Hypertension (440-447)		1.40	1.42	1.67	2.51	1.6	1.20	1.3	1.4	1.0
General Arteriosclerosis (450)	1.4			1.86		3.3	2.00	2.0	2.0	
<b>All respiratory disease (nonneoplastic)</b>							2.85			
Emphysema and/or bronchitis	24.7			10.08			2.30	1.6	2.2 <sup>f</sup>	4.3
Emphysema without bronchitis (527.1)		6.55	11.41	14.17		7.7				
Bronchitis (500-502)				4.49		11.3				
Respiratory tuberculosis (001-008)	5.0			2.12	1.27					
Asthma (241)				3.47						
Influenza and pneumonia (480-498)	1.4	1.86	1.72	1.87		1.4	2.60			2.4
<b>Certain other conditions</b>										
Stomach ulcer (540)	2.5 <sup>e</sup>	4.06	4.13	4.13	2.06 <sup>e</sup>					
Duodenal ulcer (541)		2.86	1.50	2.98		6.9	2.16			0.5
Cirrhosis (581)	3.0	2.06	1.97	3.38	1.35	2.3	1.93	2.4	0.8	4.0
Parkinsonism (350)	0.4			0.26						
<b>All causes</b>	1.64	1.88	1.43	1.84	1.22	1.52	1.70	1.4	1.2	1.78

Source: Studies cited are as follows: <sup>1</sup>R. Doll, and A. B. Hill, "Lung cancer and other causes of death in relation to smoking. A Second report on the mortality of British doctors", *British Medical Journal*, vol. 2, pp. 1071-1081 (1956); <sup>2</sup>E. C. Hammond, "Smoking in relation to the death rates of one million men and women, in *Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases. NCI Monograph 19*, W. Haenszel, ed. (Washington, D. C., United States Department of Health, Education and Welfare, pp. 127-204, 1966); <sup>3</sup>H. A. Kahn, "The Dorn study of smoking and mortality among U.S. veterans: Report on eight and one-half years of observation, in *Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases, NCI Monograph 19*, W. Haenszel, ed. (Washington D. C., National Cancer Institute, 1966); <sup>4</sup>I. Hirayama, *Smoking in Relation to the Death Rates of 265,118 Men and Women in Japan* (Tokyo, National Cancer Centre, 1967); <sup>5</sup>E. W. R. Best, G. H. Josie and C. B. Walker, "A Canadian

study of mortality in relation to smoking habits: A Preliminary report", *Canadian Journal of Public Health*, vol. 52, pp. 99-106 (1961); <sup>6</sup>E. C. Hammond, and D. Horn, "Smoking and death rates - Report on forty-four months of follow-up on 187,783 men. I. Total mortality", *Journal of the American Medical Association*, vol. 166, no. 10, pp. 1159-1172 (1958); <sup>7</sup>R. Cederlof, L. Friberg, Z. Hornbæk and U. Lorch, *The Relationship of Smoking and Some Social Covariables to Mortality and Cancer Morbidity. A Ten Year Follow-up in a Probability Sample of 55,000 Swedish Subjects Age 18-69, Part 1/2* (Stockholm, Karolinska Institute, 1975); <sup>8</sup>J. R. Dorn, G. Linden and L. Breslow, "Lung cancer mortality experience of men in certain occupations in California", *American Journal of Public Health*, vol. 50, no. 10, pp. 1475-1487 (1960).

<sup>9</sup>Numbers in parentheses represent International Classification of Diseases (ICD) codes.

<sup>10</sup>Includes cancers of larynx, buccal cavity, and pharynx.

<sup>11</sup>Includes cancers of buccal cavity and pharynx.

<sup>12</sup>Includes cancers of intestines and rectum.

<sup>13</sup>Includes stomach ulcer and duodenal ulcer.

<sup>14</sup>Includes emphysema, bronchitis, and asthma.

higher than those for cancer or other causes associated with smoking (see table 63), cardiovascular diseases (especially ischaemic heart disease and stroke) contribute more to smoking-attributable deaths at a population level than do other causes, including lung cancer for which the relative risk is much higher. Finally, it is worth noting that the all-age excess mortality ratio of about 2 from cardiovascular diseases masks a very significant age gradient in relative risks. This is clearly shown in figure 106 based on a large (46,000 persons) case-control study carried out in the United Kingdom. At younger ages (less than 50 years), smokers have a 5 to 6 times higher death rate than non-smokers, with the relative excess declining with age. What these data suggest is that if a smoker dies from vascular disease before about the age of 50 years, there is a 70-80 per cent chance that smoking caused it, and that this is the chief mechanism through which smoking causes a 3-fold excess mortality rate in middle age.

Cigarette smoking is typically only one of several causative factors that produce disease. This is especially true for ischaemic heart disease where smoking interacts synergistically with other factors such as hypercholesterolemia and hypertension to greatly increase risk of heart disease. Evidence suggests that the independent risk attributable to smoking is comparable to that of other major risk factors (USDHHS, 1989). This interaction with dietary parameters probably explains the currently lower proportions of ischaemic heart disease attributable to smoking in populations such as China, where low-fat diets have predominated (Liu and others, 1998).

## 2. Alcohol use

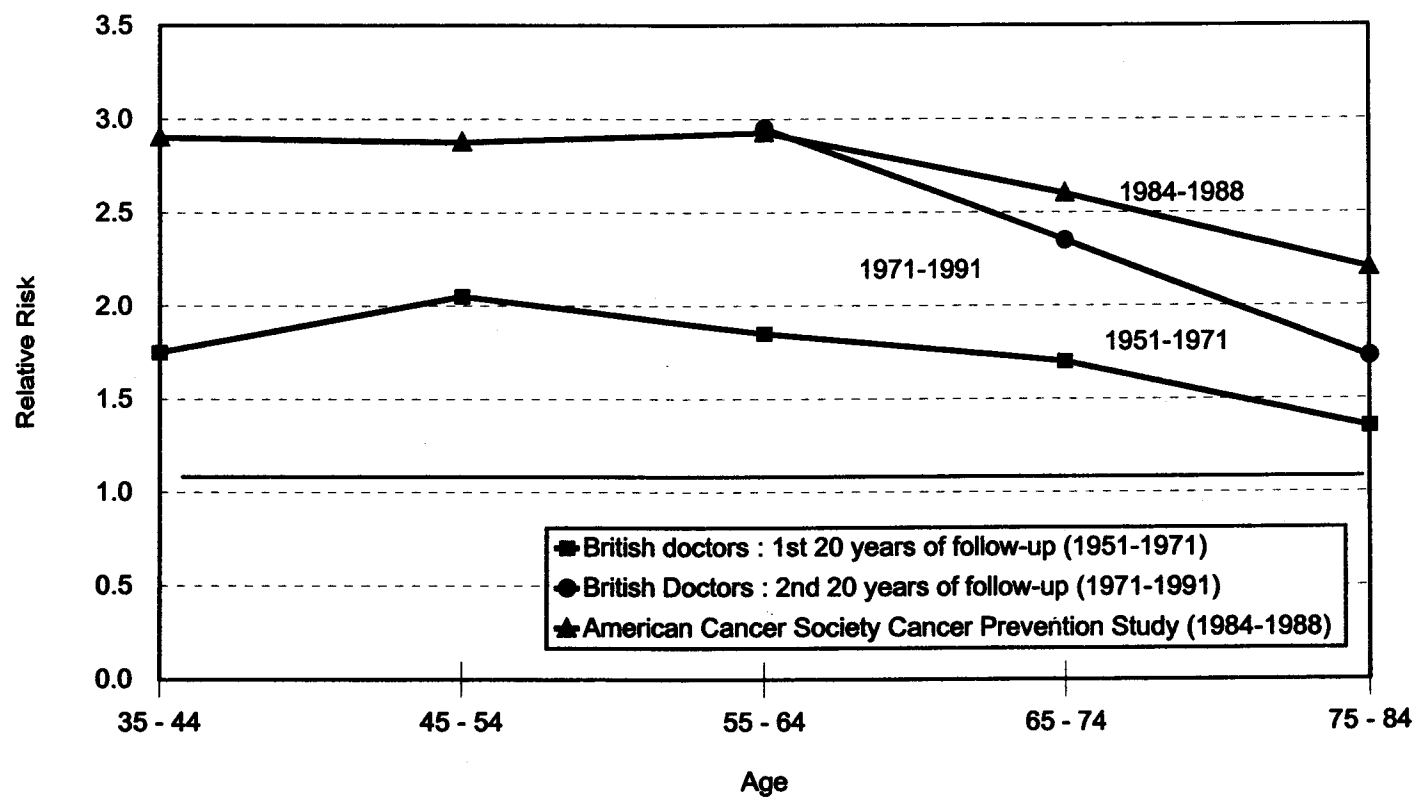
The epidemiology of alcohol use is considerably more complex than that for tobacco.

Unlike tobacco, for which only a handful of very minor conditions have some protective effects been suggested, alcohol use has been found to be both protective and causative of disease. In addition, while smoking is not a major cause of traumatic or violent deaths, alcohol is. It is therefore more meaningful to examine individual risks related to alcohol consumption according to these three public health domains.

Much of the evidence on alcohol and its association with disease emanates from the same types of epidemiological investigations (retrospective or prospective) as described earlier for tobacco. The relationship between alcohol use and traumatic events, on the other hand, has been largely determined on the basis of case series that yield data on the proportions of victims presenting with elevated blood alcohol concentrations.

The issue of causality in these observed associations is as important for alcohol as for smoking. Confounding, particularly with tobacco use, is a major concern when assessing alcohol hazards. Unless this issue is carefully and reliably dealt with, estimates of the attributable fraction of disease caused or prevented by alcohol use could be exaggerated. Drinkers probably systematically underestimate their consumption, which makes assessment of the dose-response relationship even less certain. Case series based on emergency room admissions are particularly subject to bias, and extrapolation to population-attributable fractions needs to be done very prudently. Notwithstanding the evident biases in such case series, for some major traumatic events where alcohol involvement is of principal concern, such as road traffic accidents, numerous studies have confirmed that the association meets the criteria for concluding that alcohol is indeed causa-

**Figure 105. Trend with age in relative risk of death for cigarette smokers compared with life-long smokers**



Source: Calculated from data given in R. Peto and others, *Mortality from Smoking in Developed Countries, 1950-2000* (Oxford, Oxford University Press, 1994).

TABLE 63. MORTALITY BY CAUSE IN FIRST AND SECOND 20 YEARS AMONG NON-SMOKERS AND CIGARETTE SMOKERS, BRITISH DOCTORS COHORT STUDY

CIGARETTE SMOKING, BRITISH DOCTORS CONSUMPTION					
Cause of death	Annual death rate per 100,000 men				
	Never smoked regularly		Current cigarette smokers		
	1951-1971	1971-1991	1951-1971	1971-1991	
All neoplastic causes		382	394	743	993
Lung cancer	17	17	264	314	
Other cancers substantially affected by smoking*	75	55	150	261	
Other neoplastic**	290	322	329	418	
All respiratory causes		163	121	384	466
Chronic obstructive lung disease	7	15	151	208	
Other respiratory disease	156	106	233	258	
All vascular causes		1 626	1 153	2 416	2 003
Cerebrovascular disease	401	278	516	501	
Cardiovascular disease	1 225	857	1 900	1 502	
Other diseases		258	198	370	388
Trauma and poisoning		94	74	119	156
All causes		2 523	1 954	4 077	4 026

Source: R. Doll, Richard Peto, Keith Wheatley, Richard Gray and Isabelle Sutherland, "Mortality in relation to smoking: 40 years observations on male British doctors", *British Medical Journal*, vol. 309, pp. 901-911 (1994).

\*Includes cancer of unknown site, plus remaining types recognised by the International Agency for Research on Cancer as being able to be caused by smoking.

\*\*For some of these sites (for example, stomach or liver) a small proportion of cases may be caused by smoking, and this category may include some misdiagnosed cancers of sites substantially affected by tobacco.

TABLE 64. SUMMARY OF ESTIMATED RELATIVE RISKS FOR CURRENT CIGARETTE SMOKERS, MAJOR DISEASE CATEGORIES CAUSALLY RELATED TO CIGARETTES, MALES AND FEMALES AGED 35 YEARS AND OLDER, CPS-I (1959-1965) AND CPS-II (1982-1986), UNITED STATES COHORT STUDY

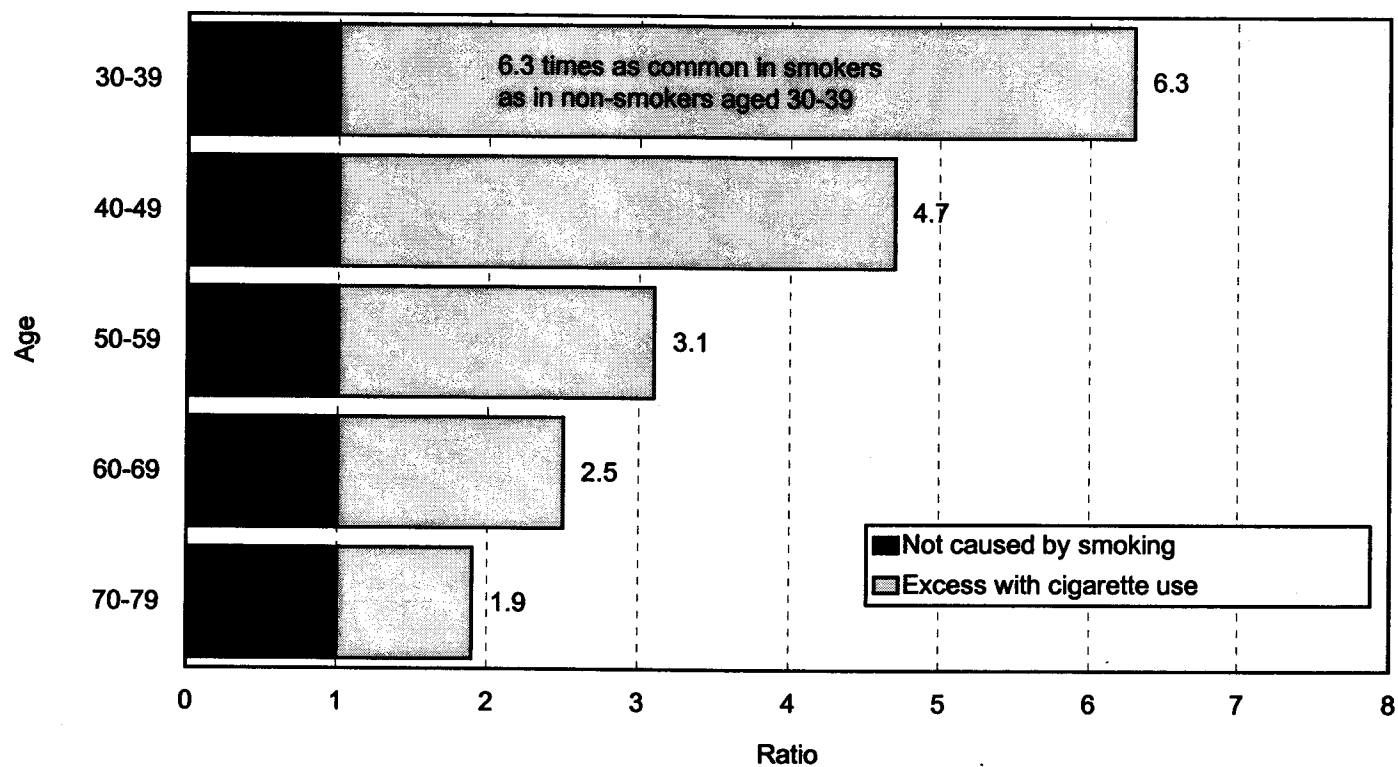
Underlying cause of death <sup>a</sup>	Males		Females	
	CPS-I	CPS-II	CPS-I	CPS-II
CHD, age > 35	1.83	1.94	1.40	1.78 <sup>b</sup>
CHD, age 35-64	2.25	2.81 <sup>b</sup>	1.81	3.00 <sup>b</sup>
Cerebrovascular lesions, age > 35	1.37	2.24 <sup>b</sup>	1.19	1.84 <sup>b</sup>
Cerebrovascular lesions, age 35-64	1.79	3.67 <sup>b</sup>	1.92	4.80 <sup>b</sup>
COPD	8.81	9.65	5.89	10.47
Cancer, lip, oral cavity and pharynx	6.33	27.48	1.96	5.59
Cancer, esophagus	3.62	7.60	1.94	10.25 <sup>b</sup>
Cancer, pancreas	2.34	2.14	1.39	2.33
Cancer, pararynx	10.00	10.48	3.81	17.78
Cancer, lung	11.35	22.36 <sup>b</sup>	2.69	11.94 <sup>b</sup>

Source: USDHHS, *Reducing the Health Consequences of Smoking: 25 years of Progress. A Report of the Surgeon General*. DHHS Publication No (CDC) 89-8411 (Washington, D.C., US Department of Health and Human Services, Centers for Disease Control, Office on Smoking and Health, 1989).

<sup>a</sup>See tables 65-68 for International Classification of Disease codes.

<sup>b</sup>95 per cent confidence intervals do not overlap between CPS-I and CPS-II.

**Figure 106. Ratio of heart attack rates: smokers versus non-smokers of the same age**



*Source:* Based on the ISIS study of over 10,000 UK heart attacks, S. Parish and others, "Cigarette smoking, tar yields, and non-fatal myocardial infarction: 14,000 cases and 32,000 controls in the United Kingdom, *British Medical Journal*, vol. 311, pp. 471-477 (1994).



tive (as summarised in English and others, 1995).

*Diseases for which risk is augmented by alcohol use*

There is a vast literature providing reviews of epidemiological studies on the hazards of alcohol consumption (see, for example, Anderson and others, 1993; Duffy, 1992; English and others, 1995; Verschuren, 1993). Even where confounding has been properly controlled for, the true nature of the dose-response curve is difficult to ascertain because of the lack of standardisation of what constitutes a "drink". This should be kept in mind in the discussion that follows, where dose-response curves from studies have been recalibrated according to a standard consumption measure, i.e. estimated grams/day of alcohol.

There are some conditions which, by definition, are entirely caused by alcohol consumption. These include alcoholic psychosis, alcohol dependence, alcoholic polyneuropathy, alcoholic cardiomyopathy and alcoholic gastritis. Typically, this cluster of conditions is not a major cause of death in most countries. Rather, the public health importance of alcohol as a cause of disease arises largely due to the increased risk of cirrhosis of the liver and several cancers.

*Cirrhosis of the liver.* Six studies that met criteria for evaluating the dose-response curve for cirrhosis of the liver are shown in figure 107 (Anderson, 1995). Five of the six studies show a clear dose-response relationship. Three are case-control studies and three are prospective studies covering populations of men in the USA, Japan and France. The general form of the risk curve suggested by these studies is an exponential increase in risk beyond a daily consumption of about 20 grams of alcohol. Each additional 20 grams of alcohol multiplies the relative risk of developing liver cirrhosis by about 2.2 (Duffy, 1992). Two of these case-control studies (Tuyns and Pequinot, 1984; Coates and others, 1986) also assessed risk curves in women and reported an even higher risk for women than men at any given consumption level.

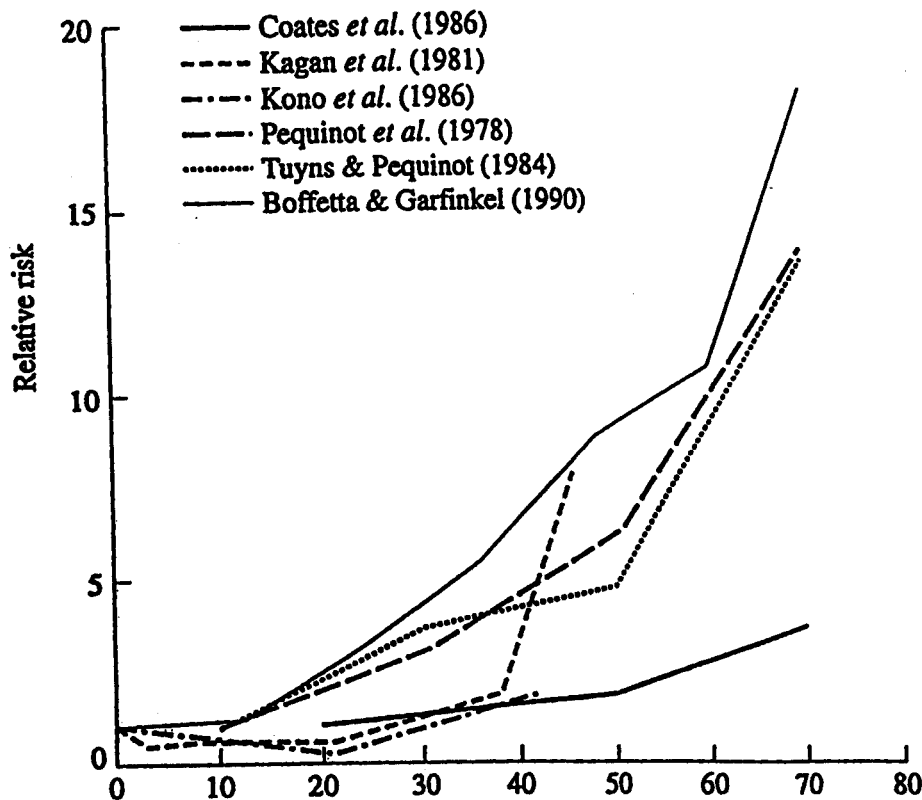
*Cancers.* One of the most comprehensive reviews of alcohol and cancer is that carried out by the International Agency for Research on Cancer. This review concluded that alcohol is a cause of cancers of the oral cavity, pharynx, larynx, oesophagus and liver, independent of cigarette smoking (IARC, 1988). Other reviews have come to much the same conclusion (see, for example, Duffy and Sharples, 1992; English and others, 1995; Doll and others, 1993; Anderson and others, 1993). These reviews have also concluded that alcohol is likely to be a cause of breast cancer, and may possibly increase the risk of cancers of the large bowel and stomach (Anderson, 1995).

The question of whether or not there is a consumption threshold below which the carcinogenic properties of alcohol are likely to be weak or non-existent is difficult to resolve in view of the very low relative risks observed once smoking is controlled for. The two large prospective studies mentioned earlier (British doctors, CPS-II) suggest that mortality risks for cancers causally related to alcohol only begin to rise markedly after about 3-4 drinks a day, with death rates of those having an average daily consumption beyond this level being 3-7 times higher than for non-drinkers (Doll and others, 1994; Thun and others, 1997).

There is now an increasing body of evidence to suggest that alcohol is a cause of female breast cancer. The 17 studies on the incidence of breast cancer in women reviewed by Anderson (1995), showed a significant positive association with alcohol consumption; five of the studies were based on large cohorts (see figure 108). With only one exception, all of the studies identified a significant dose-response relationship. This finding has been confirmed in the large CPS-II cohort analysis which found that breast cancer mortality was about 30 per cent higher in women reporting at least one drink a day compared with lifelong non-drinkers (Thun and others, 1997).

The relationship between alcohol consumption and cardiovascular disease is complex. On the one hand, high intake of alcohol has been found to increase the risk of haemorrhagic stroke (Klatsky and others, 1990; English and others,

Figure 107. Alcohol consumption and incidence of cirrhosis of the liver in men  
Data truncated at 70 g/day



Source: P. Anderson, "Alcohol and risk of physical harm", in *Alcohol and Public Policy: Evidence and Issues*, H. D. Holder and G. Edwards, eds. (Oxford, Oxford University Press, pp. 82-113, 1995).

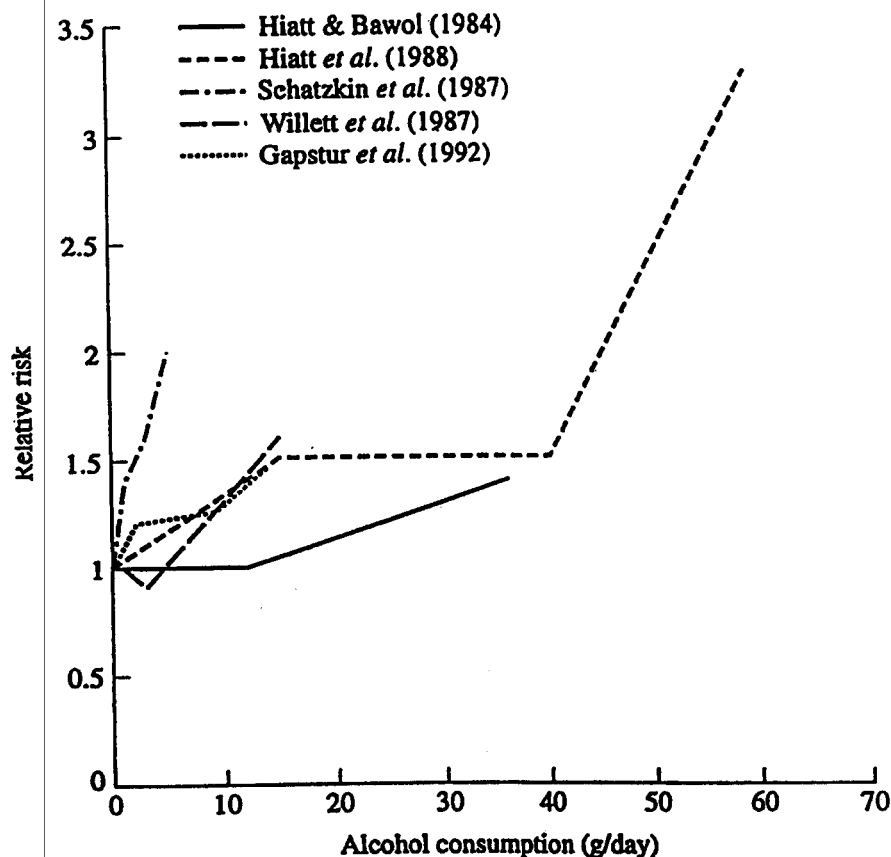
1995) cardiac arrhythmia, cardiomyopathy and sudden coronary death, none of which are major causes of vascular disease mortality in populations where the association with alcohol has been studied. There is also a linear dose-response relationship between alcohol consumption and blood pressure which underlies at least some of the effect on stroke (Anderson, 1995). It has been estimated that those drinking at hazardous levels incur a relative risk of stroke that is two to three times greater than low-level drinkers. Conversely, there is now a substantial body of evidence to suggest that alcohol con-

sumption reduces the risk of ischaemic heart disease (and quite possibly of ischaemic stroke as well), as discussed in the following section.

#### *Diseases for which risk is reduced by alcohol consumption*

Epidemiological studies have repeatedly found a reduced risk of ischaemic (or coronary) heart disease in drinkers compared with non-drinkers. Although the evidence is not entirely consistent, it is sufficient to meet the criteria for causality. In his review of 14 studies on men,

**Figure 108. Alcohol consumption and incidence of female breast cancer**  
Data from five prospective studies



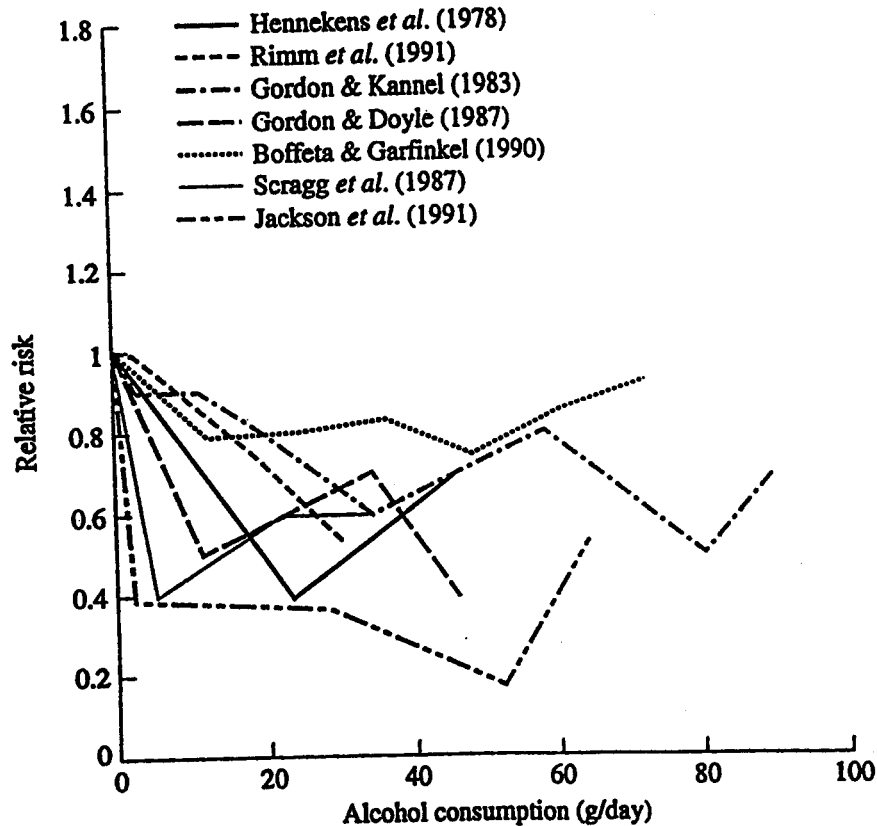
Source: P. Anderson, and others, "The risk of alcohol", *Addiction*, vol. 88, pp. 1493-1508 (1993).

Anderson (1995) reported that seven found a significant negative association between consumption and mortality, three a non-significant negative association, one no relationship, and three a non-significant positive association. The nature of the association for the seven studies where statistical significance was established is shown in figure 109. English and others (1995), in an even more extensive review, found evidence of a protective effect of low alcohol consumption in 32 cohort and case-control

studies, while three reported an increased risk and another three found no effect.

The cardio-protective effect of alcohol consumption on mortality has been confirmed by the British doctors study and by the CPS-II analysis. Doctors drinking about 18 British units per week (i.e. about 20 grams of alcohol per day) had death rates about 30-40 per cent lower than non-drinkers: at levels above this, death rates increased slightly but never reached

Figure 109. Alcohol consumption and incidence of coronary heart disease in men  
Data from seven studies with significant negative association



Source: P. Anderson, "Alcohol and risk of physical harm", in *Alcohol and Public Policy: Evidence and Issues*, H. D. Holder and G. Edwards, eds. (Oxford, Oxford University Press, pp. 82-113, 1995).

the level of non-drinkers. A similar finding was found in the CPS-II cohort, for both men and women (Thun and others, 1997).

Overall, epidemiological studies suggest that alcohol consumption leads to a reduction in risk of ischaemic heart disease of about one-third (English and others, 1995). There appears to be little relationship to amount consumed, except perhaps at extremely high levels of consumption which are not commonly reported in epidemiological studies, where risk of sudden coronary death is greatly increased. Moreover, this

finding is supported by the results of international ecological studies and is consistent with plausible biological mechanisms which show that alcohol elevates high density lipo-protein (HDL) cholesterol levels, and inhibits blood coagulation through reduced plasma fibrinogen concentrations and lower platelet activity.

The reduction in coronary heart disease mortality at low to moderate levels of consumption appears to persist even when those with pre-existing disease are removed from the analysis (Boffeta and Garfinkel, 1990; Klatsky and

others, 1989; Thun and others, 1997) and is largely unaffected by whether or not former drinkers are included with non-drinkers to compute relative risks (Doll and others, 1994). The protective effect appears to be greatest when consumption is spread throughout the week rather than concentrated on one or two days (Rimm and others, 1991) and appears to be present for all beverage types (Renaud and others, 1993; Doll and others, 1994).

#### *Alcohol and injuries*

The role of alcohol as a cause of injury is due to the psychological effects of intoxication, however mild, which include reduced co-ordination and balance, increased reaction time, and impaired attention and judgement, all of which act to increase the risk of accidents. The extent and importance of alcohol involvement in causing accidents will vary according to the degree of intoxication, the context in which alcohol is consumed and risk is incurred, and the type of injury. In general, the involvement of alcohol in injuries is less well documented for non-vehicular events, no doubt due to the fact that these injuries are generally less dramatic and of less public interest, and that legislative measures are less applicable (Romelsjö, 1995).

The assessment of the role of alcohol in producing injuries is particularly problematic. The preferred measurement is obviously a measure of alcohol involvement at the time of the event. Measures of blood-alcohol concentration (BAC) in most cases yield lower values at testing than actual levels at or before the injury takes place. There are biases that arise from case refusal or from drivers or victims who consumed alcohol after the event. Equally important is the study design. For example, in a case-control study, fatal accident victims may be excluded from cases (as might those with minor injuries), among whom alcohol involvement may be quite different to other cases (Romelsjö, 1995).

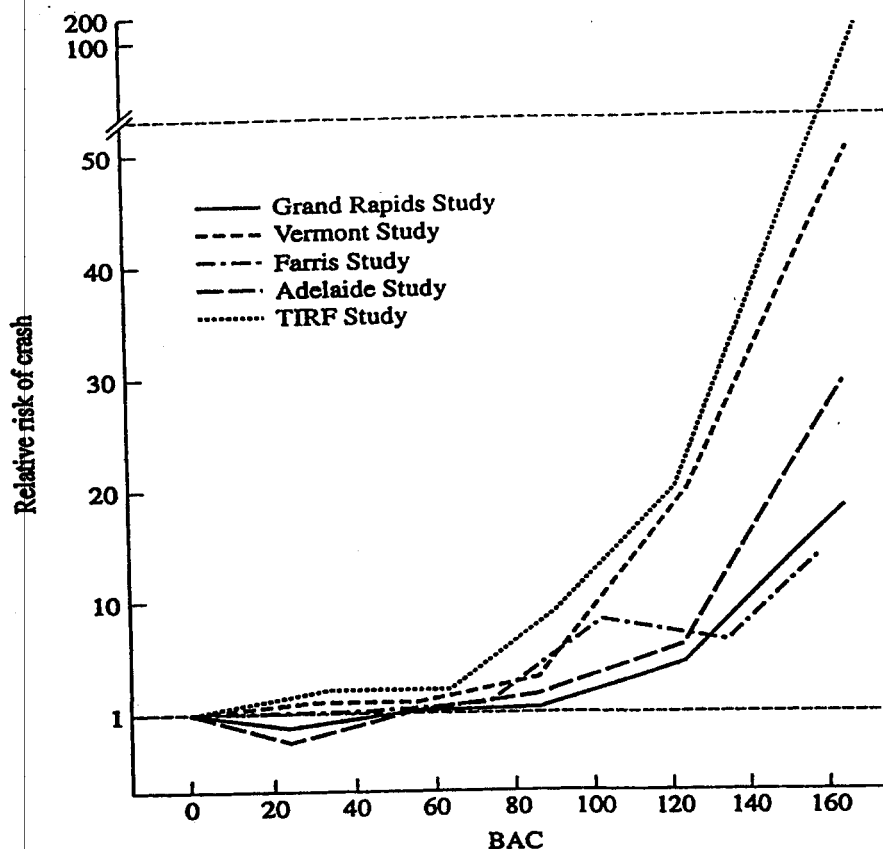
Despite these caveats, it is clear that alcohol is a major cause of accidents, in particular traffic crashes involving young men, and that

the risk of accident increases with higher blood alcohol concentration. In the case of motor vehicle accidents, laboratory tests have demonstrated that impairment is already evident at BACs of 0.02-0.03 mg per hundred, and that by 0.05 mg per hundred, the legal limit in many countries, the risk of accident is already about twice the normal risk (Voas, 1993). By 0.08 mg per hundred, this risk is increased by about four-fold over that of a driver with zero BAC, rising to eight-fold at 0.1 mg per hundred, and 27 times at 0.15 mg per hundred (Romelsjö, 1995) (see figure 110). Intoxicated pedestrians are about 3 to 4 times more likely to be hit by a vehicle than those who are not intoxicated (Irwin and others, 1983), and studies of other vehicular accidents have suggest that alcohol is involved in, if not the main cause of between 20-40 per cent of these events.

Alcohol is a leading contributor to falls, with roughly 20-60 per cent of victims being intoxicated at the time of the fall in populations where this has been studied (Hingson and Howland, 1993), and in drowning, burns and hypothermia, with various studies reporting alcohol involvement ranging from 30-50 per cent (Howland and Hingson, 1988; Penttilä and others, 1979). Alcohol consumption has been repeatedly identified as a cause of occupational injuries with estimates of 3 to 13 per cent of fatal cases being intoxicated at the time of the injury (Stallones and Kraus, 1993; Lewis and Cooper, 1989). Something like 20-36 per cent of suicide victims in various studies had a history of alcohol abuse or had been drinking at the time of the event (Roizen, 1982).

Perhaps one of the best epidemiological studies to investigate the role of alcohol in risk of traumatic death is the 13-year follow-up cohort study of 49,464 Swedish military recruits reported by Andreasson and others, 1988. The results of this study are summarised in figure 111. There is a clear, monotonic increase in risk of violent death with increasing levels of alcohol consumption reported at baseline (conscription). It is quite probable that a similar linear risk curve applies for other populations of young adults as well, particularly men.

Figure 110. Relative likelihood of a motor vehicle collision as a function of blood alcohol concentration (BAC)



Source: A. Romelsjö, "Alcohol consumption and unintentional injury, suicide, violence, work performance and inter-generational effects, in *Alcohol and Public Policy: Evidence and Issues*, H. H. Holder and G. Edwards, eds. (Oxford, Oxford University Press, pp. 114-142, 1995).

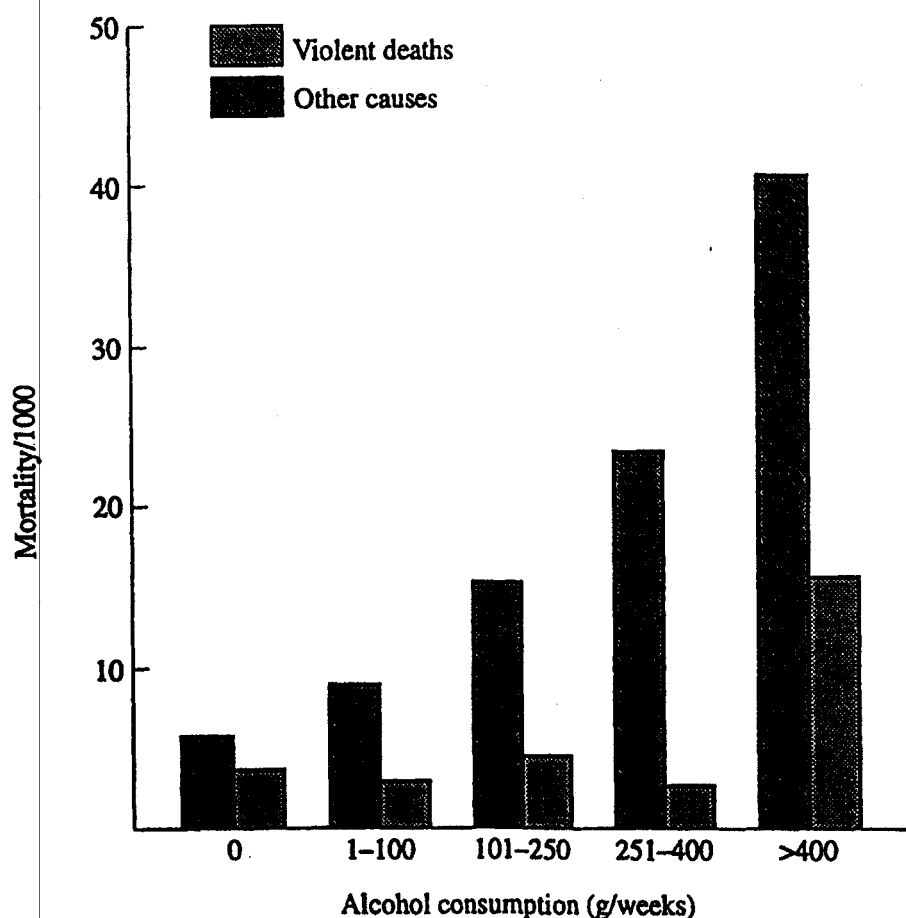
## B. PATTERNS AND TRENDS IN SMOKING AND ALCOHOL USE

### 1. Cigarette smoking

To assess the extent of cigarette smoking in various populations, it is important to know both the proportion of adults (by sex) who smoke (i.e. smoking prevalence), and the amount of cigarettes consumed, on average, by adults in a given year (cigarette consumption). International comparisons and assessments of

prevalence of consumption are greatly hampered by variations in basic definitions of what is a smoker and particularly, what is a regular smoker. Since most prevalence data are collected from sample surveys, variations and biases in study design and methods will also influence results and hence comparability across countries and regions. Many countries have recently carried out prevalence surveys and these have been reviewed and used to estimate global and regional smoking prevalence in the early 1990s (World Health Organization, 1997a).

Figure 111. Association between alcohol consumption at conscription, and mortality (violent and other deaths) during follow-up



Source: S. Andreasson and others, "Alcohol and mortality among young men. Longitudinal study of Swedish conscripts", *British Medical Journal*, vol. 296, pp. 1021-1025 (1988, fig. 1).

Consumption is generally estimated indirectly from production and trade data on imports and exports. Consumption can be, and often is measured in surveys of smokers but this method typically leads to an underestimation of consumption of the order of 30-50 per cent (Collishaw, 1987). Population-level consumption estimates are also affected by the extent of non-manufactured cigarette use, and of smuggling, which in some countries (Canada and Belgium for example) has been estimated to account for

10-30 per cent of all cigarettes consumed (World Health Organization, 1997a).

#### *Smoking prevalence in the early 1990s*

Despite the caveats mentioned above, a sufficiently large number of countries covering about 85 per cent of the world's adult population have carried out prevalence surveys with roughly comparable methods and definitions to

permit international assessments and comparisons. The estimates are shown in table 65 and have been used to prepare the regional and global estimates of prevalence for the early 1990s shown in table 66. Based on these data, the World Health Organization has estimated that there are about 1.1 billion daily smokers in the world, or about one-third of the world's population aged 15 years and over. Eight hundred million of these smokers are males, 700 million of whom live in developing regions. Two-thirds (200 million) of regular smokers in developed countries are men.

Globally, almost half (47 per cent) of men smoke, but considerably fewer women do so (12 per cent). There are, however, marked regional variations as might be expected. Thus in the established market economies, 37 per cent of men are regular smokers, compared with about 60 per cent of men in the former socialist economies of Europe and China. Smoking prevalence is high in other developing regions as well, estimated at around 40 per cent in India, Latin America and North Africa/Western Asia, and even higher (54 per cent) in other parts of Asia.

Typically, women smoke less than men, even in the rich countries, where female smoking prevalence is about half to two-thirds the level for men. The proportion of women smoking in developing countries is still low (on average, about 7 per cent) but could rise dramatically, particularly among younger women, unless effective tobacco control policies are implemented.

The significance of smoking among young women for their future mortality should not be underestimated. Overall population prevalence estimates such as those reported in table 64 can mask very significant age variations in smoking prevalence, particularly for women. Spanish females are an excellent example. According to the table, about 25 per cent of Spanish women smoke. However, virtually all of these are young women, as figure 112 indicates. At ages 18-44, about 40 per cent of young Spanish women now smoke, but few older Spanish women do so. As a result, current lung cancer rates among Spanish women are comparable to those of lifelong non-smokers (see figure 113).

However, if these young women continue to smoke in large numbers into middle age, smoking-attributable mortality among Spanish women (and among others where similar patterns are evident) will rise dramatically.

#### *Trends in cigarette consumption*

Because of the longer time series of available data on cigarette consumption, trends in the regions of the world can be estimated over the last two decades or so. On the other hand, the commodity and production data sources do not readily permit male-female consumption patterns and trends to be estimated. These are, however, likely to closely reflect patterns and trends in prevalence data.

Using these various data sources, and comparing them with prevalence estimates to ensure internal consistency, national estimates of cigarette consumption for 1990-1992, and trends over the previous two decades, have been prepared. These are shown in table 67, along with national rankings of consumption in 1970-1972, 1980-1982, and 1990-1992. The success of tobacco control programmes in some developed countries is clear from the change in ranking and levels of consumption. Canada, for example, had the highest per capita consumption in the world in 1970-1972 (3910 cigarettes per adult per year), but had declined to 13th rank in 1990-1992 (following a 35 per cent decline in consumption). An equally dramatic reduction in smoking has occurred in the United Kingdom. Consumption per adult has fallen by 32 per cent since the early 1970s, resulting in large fall in their comparative smoking position (5th to 21st). In other regions of the world, smoking has increased substantially over the last two decades or so. This is particularly evident in parts of Eastern Europe, particularly Poland and Hungary, as well as in Greece, Japan and the Republic of Korea.

As has been done for prevalence, these national estimates can be aggregated to yield regional and global estimates of cigarette consumption (see table 68). Over the decade 1980-1982 to 1990-1992, cigarette consumption declined by 1.5 per cent per year in industrialised countries. It remained relatively constant in Eastern Europe, and it rose by an annual



TABLE 65. ESTIMATED SMOKING PREVALENCE AMONG MEN AND WOMEN 15 AND OVER, BY COUNTRY,  
LATEST AVAILABLE YEAR (RANKED IN ORDER OF MALE SMOKING PREVALENCE)

Rank	Country (year of survey)	Men	Women	Rank	Country (year of survey)	Men	Women
1	Republic of Korea (1989)	68.2	6.7	42	India (1980s)	40.0	3.0
2	Latvia (1993)	67.0	12.0	42	Iraq (1990)	40.0	5.0
2	Russian Federation (1993)	67.0	30.0	42	Malta (1992)	40.0	18.0
4	Dominican Republic (1990)	66.3	13.6	42	Mongolia (1990)	40.0	7.0
5	Tonga (1991)	65.0	14.0	42	Uzbekistan (1989)	40.0	1.0
6	Turkey (1988)	63.0	24.0	50	Brazil (1989)	39.9	25.4
7	China (1984)*	61.0	7.0	51	Egypt (1986)	39.8	1.0
8	Bangladesh (1990)	60.0	15.0	52	Morocco (1990)	39.6	9.1
9	Fiji (1988)	59.3	30.6	53	Lesotho (1989)	38.3	1.0
10	Japan (1994)	59.0	14.8	53	Mexico (1990)	38.3	14.4
11	Sri Lanka (1988)	54.8	0.8	55	El Salvador (1988)	38.0	12.0
12	Algeria (1980)	53.0	10.0	55	Italy (1994)	38.0	26.0
12	Indonesia (1986)	53.0	4.0	55	Portugal (1994)	38.0	15.0
12	Samoa (1994)	53.0	18.6	58	Chile (1990)	37.9	25.1
15	Saudi Arabia (1990)	52.7	N/A	59	Guatemala (1989)	37.8	17.7
16	Estonia (1994)	52.0	24.0	60	Denmark (1993)	37.0	37.0
16	Kuwait (1991)	52.0	12.0	61	Germany (1992)	36.8	21.5
16	Lithuania (1992)	52.0	10.0	62	Norway (1994)	36.4	35.5
16	South Africa (1995)	52.0	17.0	63	Honduras (1988)	36.0	11.0
20	Poland (1993)	51.0	29.0	63	Netherlands (1994)	36.0	29.0
21	Seychelles (1989)	50.9	10.3	63	Switzerland (1992)	36.0	26.0
22	Bolivia (1992)	50.0	21.4	66	Colombia (1992)	35.1	19.1
23	Albania (1990)	49.8	7.9	67	Costa Rica (1988)	35.0	20.0
24	Cuba (1990)	49.3	24.5	67	Slovenia (1994)	35.0	23.0
25	Bulgaria (1989)	49.0	17.0	69	Swaziland (1989)	33.0	8.0
25	Thailand (1995)	49.0	4.0	70	Luxembourg (1993)	32.0	26.0
27	Spain (1993)	48.0	25.0	71	Singapore (1995)	31.9	2.7
28	Mauritius (1992)	47.2	3.7	72	Belgium (1993)	31.0	19.0
29	Greece (1994)	46.0	28.0	72	Canada (1991)	31.0	29.0
29	Papua New Guinea (1990)	46.0	28.0	72	Iceland (1994)	31.0	28.0
31	Israel (1989)	45.0	30.0	75	Australia (1993)	29.0	21.0
32	Cook Islands (1988)	44.0	26.0	75	Ireland (1993)	29.0	28.0
33	Czech Republic (1994)	43.0	31.0	75	United Kingdom (1992)	29.0	28.0
33	Jamaica (1990)	43.0	13.0	78	United States of America (1991)	28.1	23.5
33	Philippines (1987)	43.0	8.0	79	Pakistan (1980)	27.4	4.4
33	Slovakia (1992)	43.0	26.0	80	Finland (1994)	27.0	19.0
37	Cyprus (1990)	42.5	7.2	81	Turkmenistan (1992)	26.6	0.5
38	Austria (1992)	42.0	27.0	82	Nigeria (1990)	24.4	6.7
39	Malaysia (1986)	41.0	4.0	83	Paraguay (1990)	24.1	5.5
39	Peru (1989)	41.0	13.0	84	Bahrain (1991)	24.0	6.0
41	Uruguay (1990)	40.9	26.6	84	New Zealand (1992)	24.0	22.0
42	Argentina (1992)	40.0	23.0	86	Sweden (1994)	22.0	24.0
42	France (1993)	40.0	27.0	87	Bahamas (1989)	19.3	3.8
42	Hungary (1989)	40.0	27.0	88	Benin (1990)	8.9	6.6

Source: World Health Organization, *Tobacco or Health: a Global Status Report* (Geneva, WHO, 1997a).

\*More recent data collected in 1991 on the smoking habits of men and women in the context of a prospective study of the health effects of smoking in China suggest that there has been little change in smoking prevalence since 1984.

TABLE 66. DAILY SMOKING PREVALENCE, MEN AND WOMEN AGED 15 AND OVER, SELECTED REGIONS, EARLY 1990S

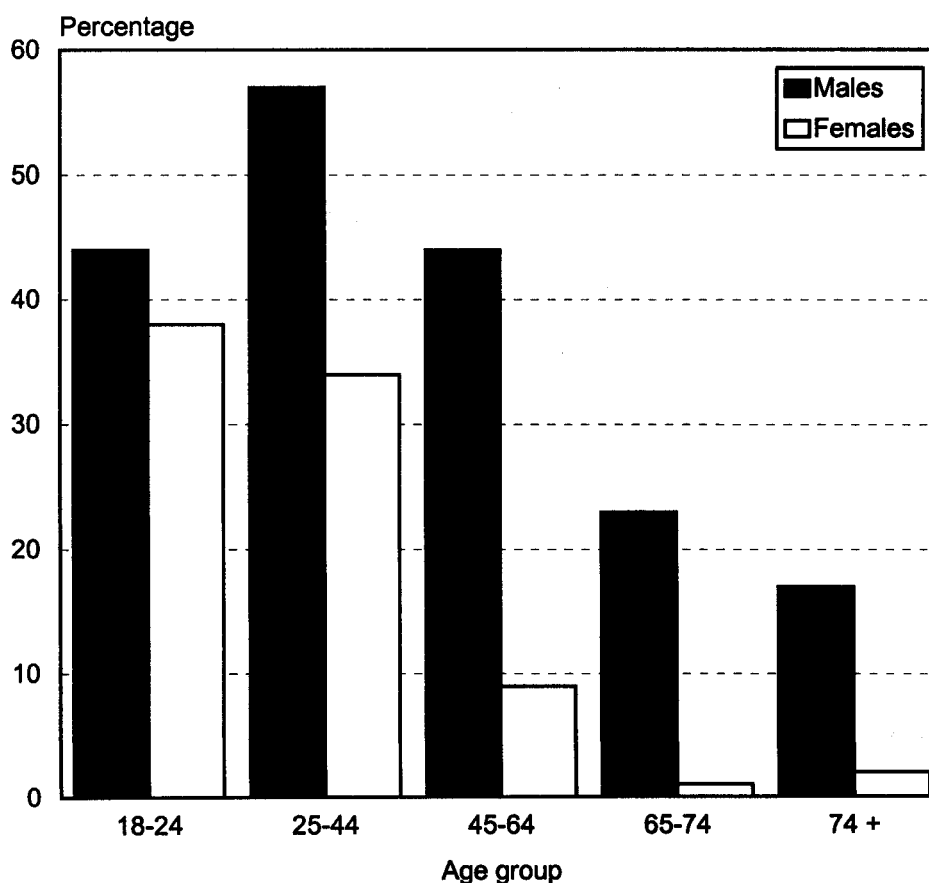
	Men	Women
World Health Organization regions		
African region*	29	4
American region	35	22
Eastern Mediterranean region	35	4
European region	46	26
Western Pacific region	60	8
More developed countries	42	24
Established market economies	37	23
Formerly socialist economies of Europe	60	28
Less developed countries	48	7
China (1984)	61	7
India (1980s)	40	3
Other Asia and islands	54	7
Middle Eastern crescent**	41	8
Sub-Saharan Africa*	25	3
Latin America and the Caribbean	40	21
World	47	12

Source: World Health Organization, *Tobacco or Health: a Global Status Report* (Geneva, WHO, 1997a).

\*Smoking prevalence estimates for Africa, are based on very limited information and should be used with caution

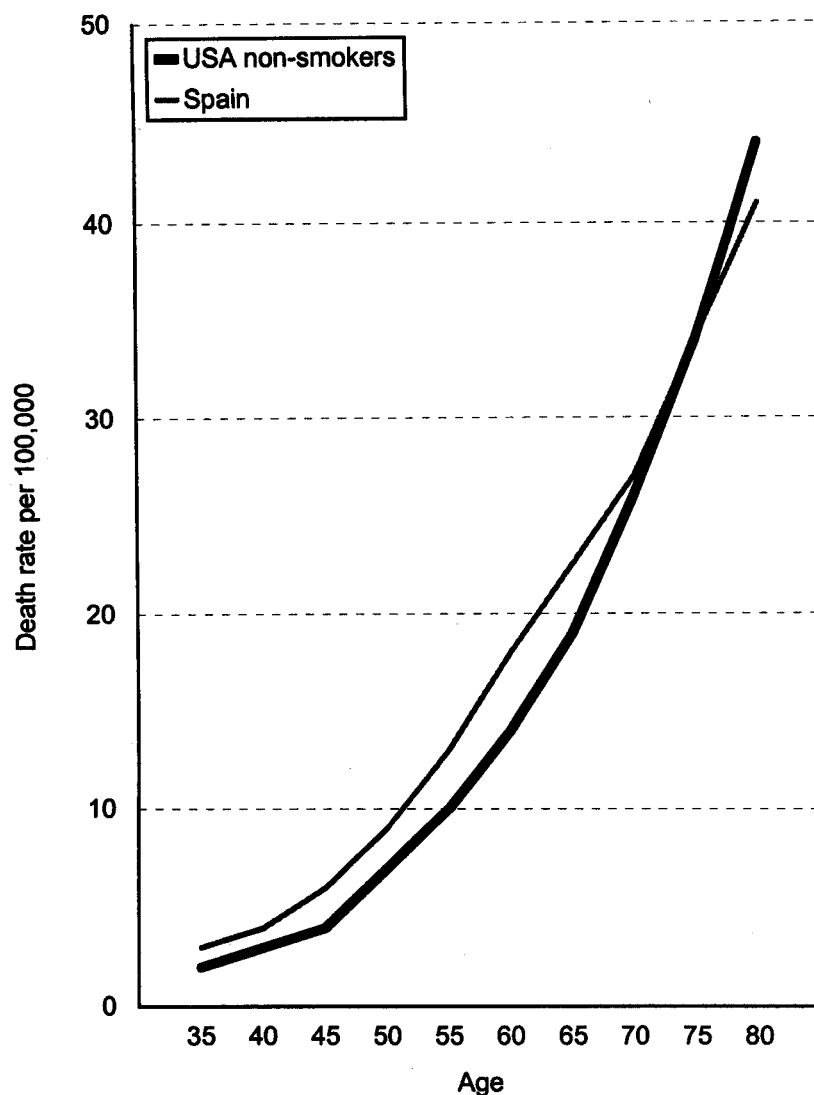
\*\*Includes countries of Northern Africa, Western Asia and the Central Asian Republics of the former Soviet Union.

Figure 112. Daily smoking prevalence, Spain, 1993



Source: World Health Organization, *Tobacco or Health: a Global Status Report* (Geneva, WHO, 1997a).

**Figure 113. Age-specific lung cancer death rates for women: Spain (1993) and USA non-smokers (1984-1988)**



Source: World Health Organization Mortality Database.

average of 1.4 per cent in developing countries. As a result, global consumption has remained relatively constant at around 1,650 cigarettes per adult per year. There is clearly considerable heterogeneity in the trends among developing countries, some of which are particularly alarming. In China, for example, per adult consumption has almost tripled since the early 1970s, rising to almost 2,000 cigarettes per adult per year. Almost all smokers are men,

suggesting that their per capita consumption is closer to 4,000 cigarettes per year. One-third of the 5.2 cigarettes smoked in the world each year are consumed in China alone.

The reasons for the increasing public health concern about rising tobacco consumption levels in developing countries are well illustrated by figure 114 which shows the trend in the ratio of per adult consumption in developed

TABLE 67. ESTIMATED PER CAPITA CONSUMPTION OF CIGARETTES PER ADULT 15 YEARS OF AGE AND OVER, SELECTED COUNTRIES,  
1970-1972 TO 1990-1992, RANKED ACCORDING TO CONSUMPTION IN 1990-1992

Per capita consumption						Per capita consumption					
1970-1972	Rank	1980-1982	Rank	1990-1992	Rank	1970-1972	Rank	1980-1982	Rank	1990-1992	Rank
Poland	3 010	11	3 400	6	3 620	1	Egypt	730	73	1 180	61
Greece	2 640	16	3 440	4	3 590	2	Indonesia	500	83	950	72
Hungary	2 940	13	3 320	7	3 260	3	Chile	1 310	49	1 380	53
Japan	2 950	12	3 430	5	3 240	4	Guyana	1 220	53	1 280	57
Korea, Republic of	2 370	20	2 750	15	3 010	5	Paraguay	1 190	55	1 030	68
Switzerland	3 700	2	3 060	10	2 910	6	Thailand	810	69	1 080	66
Iceland	2 940	14	3 230	9	2 860	7	Senegal	430	88	760	79
Netherlands	3 150	6	3 290	8	2 820	8	El Salvador	1 260	50	1 030	67
Yugoslavia	2 330	21	3 030	12	2 800	9	Dominican Republic	910	66	1 010	69
Australia	3 410	4	3 440	3	2 710	10	Bangladesh	510	81	680	82
United States	3 700	3	3 560	2	2 670	11	Mexico	1 600	39	1 370	54
Spain	2 190	22	2 440	21	2 670	12	Korea, Democratic				
Canada	3 910	1	3 800	1	2 540	13	People's Republic	1 050	60	1,210	60
New Zealand	3 060	9	2 890	13	2 510	14	Panama	1 150	58	950	71
Ireland	3 050	10	3 030	11	2 420	15	Iran	900	67	1 160	62
Germany	2 430	18	2 420	22	2 360	16	Morocco	680	75	1 120	63
Belgium	3 090	7	2 880	14	2 310	17	Congo	880	68	890	73
Israel	2 060	23	2 400	23	2 290	18	Ecuador	650	77	830	74
Cuba	2 690	15	2 630	17	2 280	19	Jamaica	1 400	43	990	70
Bulgaria	1 770	35	1 880	36	2 240	20	Honduras	1 090	59	1 080	65
United Kingdom	3 250	5	2 740	16	2 210	21	Sierra Leone	460	86	810	76
Austria	2 390	19	2 620	18	2 210	22	Yemen	470	85	570	88
Saudi Arabia	1 220	52	1 940	35	2 130	23	Vietnam	N/A	111	790	77
France	1 860	31	2 080	29	2 120	24	Angola	740	71	740	80
Turkey	1 950	29	2 250	25	2 100	25	Cameroon	270	98	590	87
Luxembourg	3 090	8	2 580	19	2 080	26	Cote d'Ivoire	800	70	810	75
Portugal	1 440	40	1 800	41	2 010	27	Benin	640	78	770	78
Syria	950	63	1 730	45	2 000	28	Pakistan	630	79	720	81
Italy	1 800	34	2 310	24	1 920	29	Laos	510	82	600	86
Venezuela	2 060	24	2 210	26	1 920	30	Haiti	170	104	630	85
Denmark	2 050	25	2 020	31	1 910	31	Nepal	170	105	290	103
China, People's Republic of	730	72	1 290	56	1 900	32	Kenya	420	89	560	89
Surinam	1 160	56	1 870	37	1 870	33	Togo	560	80	480	92
Norway	2 030	26	1 950	33	1 830	34	Madagascar	270	99	470	93
Mauritius	1 310	48	1 940	34	1 830	35	Mozambique	370	95	460	94
Trinidad and Tobago	1 440	41	1 960	32	1 780	36	Zimbabwe	700	74	660	83
Philippines	2 010	27	2 190	27	1 760	37	Bolivia	400	92	560	90
Colombia	1 880	30	1 790	42	1 750	38	Sri Lanka	460	87	520	91
Tunisia	1 380	44	1 590	49	1 750	39	Zambia	500	84	430	96

TABLE 6 (continued)

Per capita consumption						Per capita consumption							
	1970-1972	Rank	1980-1982	Rank	1990-1992	Rank		1970-1972	Rank	1980-1982	Rank	1990-1992	Rank
Finland	2 000	28	1 800	40	1 740	40	Liberia	390	93	420	97	420	96
South Africa, Republic of	1 340	46	1 600	48	1 720	41	Tanzania, United						
Uruguay	1 630	38	1 720	46	1 700	42	Republic of	380	94	370	99	370	97
Jordan	1 020	61	1 840	39	1 680	43	Nigeria	290	97	350	100	370	98
Malaysia	1 400	42	2 050	30	1 630	44	Peru	410	90	390	98	350	99
Singapore	2 510	17	2 550	20	1 610	45	Guatemala	660	76	640	84	340	100
Argentina	1 810	33	1 770	43	1 610	46	Malawi	200	102	330	101	330	101
Algeria	950	64	1 580	50	1 600	47	Uganda	300	96	300	102	300	102
Fiji	1 150	57	1 650	47	1 590	48	Zaire	220	101	240	105	270	103
Romania	1 740	36	2 130	28	1 550	49	Ghana	410	91	440	95	250	104
Sweden	1 700	37	1 840	38	1 550	50	Niger	110	108	100	110	170	105
Brazil	1 330	47	1 750	44	1 500	51	Sudan	170	106	150	108	150	106
Nicaragua	1 380	45	1 440	52	1 460	52	Myanmar	90	109	140	109	150	107
India	1 010	62	1 310	55	1 370	53	Solomon Islands	250	100	250	104	140	108
Costa Rica	1 850	32	1 520	51	1 340	54	Afghanistan	150	107	160	107	140	109
Iraq	1 250	51	1 090	64	1 280	55	Ethiopia	60	110	70	111	90	110
Cambodia	940	65	1 260	58	1 220	56	Cape Verde	200	103	220	106	N/A	111
Albania	1 220	54	1 230	59	1 220	57							

Source: World Health Organization, *Tobacco or Health: a Global Status Report* (Geneva, World Health Organization, 1997a).

TABLE 68. GLOBAL AND REGIONAL ESTIMATES AND TRENDS IN CONSUMPTION OF CIGARETTES  
PER ADULT 15 YEARS AND OVER, 1970-1972 TO 1990-1992

	<i>Cigarettes per adult 15 years and over</i>			<i>Annual percentage change</i>		
	1970-1972	1980-1982	1990-1992	1970-1972 to 1980-1982	1980-1982 to 1990-1992	1970-1972 to 1990-1992
World Health Organization regions						
African region	460	570	590	2.1	0.3	1.2
American region	2 580	2 510	1 900	-0.3	-2.8	-1.5
Eastern Mediterranean region	700	940	930	2.9	-0.1	1.4
European region	2 360	2 500	2 340	0.6	-0.7	-0.0
South-East Asia region	850	1 140	1 230	2.9	0.8	1.8
Western Pacific region	1 100	1 610	2 010	3.8	2.2	3.0
More developed countries	2 860	2 980	2 590	0.4	-1.4	-0.5
Established market economies	2 910	3 000	2 570	0.3	-1.5	-0.6
Formerly socialist economies of Europe	2 450	2 830	2 770	1.4	-0.2	0.6
Less developed countries	860	1,220	1 410	3.5	1.4	2.5
China	730	1 290	1 900	5.7	3.9	4.8
India	1 010	1 310	1 370	2.6	0.4	1.5
Other Asia and islands	780	1 130	1 190	3.7	0.5	2.1
Middle Eastern crescent	950	1 240	1 200	2.7	-0.3	1.2
Sub-Saharan Africa	410	490	500	1.8	0.2	1.0
Latin America and the Caribbean	1 430	1 540	1 310	0.7	-1.6	-0.4
World	1 410	1 650	1 660	1.6	0.1	0.8

Source: World Health Organization, *Tobacco or Health: a Global Status Report* (Geneva, WHO, 1997a).

to less developed countries. By the early 1990s, per capita consumption in rich countries was only a little over 1.8 times that in poorer countries (compared with a more than three fold excess two decades earlier). If this trend continues, cigarette consumption per adult in the developing world will exceed that in developed regions within the next ten years.

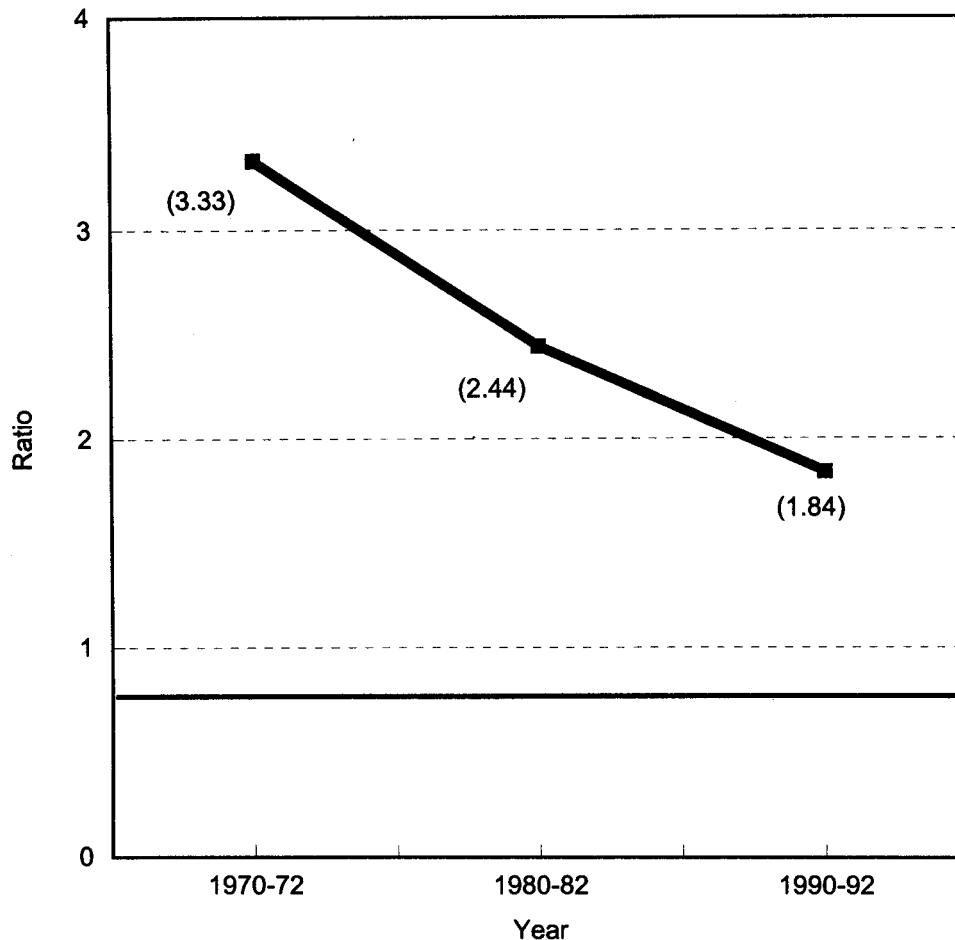
## 2. Alcohol use

Data sources and methods for data collection on alcohol are similar to what has been described for tobacco. Per adult consumption figures are typically estimated indirectly from production and trade data and also from industry publications such as *World Drink Trends* (Dutch Distillers Association, 1992). Estimates of the prevalence of drinking per se are of limited relevance for public health since, as the previous section has demonstrated, much of the

public health harm from alcohol is dependent on the typical amount drunk per occasion, the timing and context of drinking (e.g. the home or public places) and the extent of "binge" drinking. Survey data do not always include these variables and where they do, the data are not always comparable among surveys due to different definitions and survey methods. It is therefore much more difficult to provide national, let alone regional estimates of alcohol use, which are relevant for assessing public health hazards (and benefits). Rather, information from some countries in different regions of the world is presented below which may (or may not) be indicative of broader patterns and levels of population exposure to alcohol.

Trends in alcohol consumption per capita (not per adult) as estimated by the drinks industry (the Brewers and Licensed Retailers Association, 1997) are shown in table 69. Compared

Figure 114. Relative change in cigarette consumption: developed versus developing countries



Source: World Health Organization, *Tobacco or Health: a Global Status Report* (Geneva, WHO, 1997a).

with developing countries, recorded alcohol consumption is substantially higher in developed countries although the data do suggest that there has been a generalised decrease in consumption in many of the leading wine producing countries (France, Italy, Spain), as well as in Australia, Finland and North America in recent years. Overall, per capita consumption in the 15 European Union countries has declined by almost 20 per cent since 1980 (World Health Organization, 1997b).

There are several limitations of these data that need to be borne in mind when considering their public health implications. These figures yield

no information on how alcohol consumption is distributed among various population groups (e.g. by age and sex), or by drinking occasions. Moreover, they are likely to be considerable underestimates of consumption in many developing countries and in some parts of Europe (particularly Eastern Europe) due to extensive local production of alcohol which is not recorded in official statistics. For example, in India, illicit production is estimated at 50 per cent of recorded consumption (S. Saxena, personal communication). Similarly in Russia, it has been estimated that unreported alcohol consumption since the mid-1980s accounts for 50-60 per cent of total estimated alcohol con-

TABLE 69. ALCOHOL CONSUMPTION PER HEAD IN SELECTED COUNTRIES, 1970-1995

Country	(Litres per head of 100 per cent alcohol)								
	1970	1975	1980	1985	1990	1992	1993	1994	1995
European Union									
Austria	10.3	11.0	11.0	11.3	11.9	12.0	11.8	10.9	10.8
Belgium and Luxembourg	9.0	10.6	10.9	10.6	11.1	10.9	10.5	10.3	10.1
Denmark	6.8	8.8	9.3	10.3	9.8	10.0	9.9	10.0	10.1
Finland	4.3	5.9	6.1	6.3	7.8	7.1	6.8	6.6	6.4
France	17.2	17.0	15.6	13.8	12.6	12.1	12.3	11.8	11.9
Germany	12.0	13.1	13.3	12.5	11.7	12.3	11.8	11.5	11.2
Greece	..	..	..	..	7.5	7.4	7.5	7.4	7.2
Ireland, Republic of	5.9	7.8	7.4	6.6	7.3	7.5	7.3	7.1	7.2
Italy	16.0	14.9	13.9	12.5	9.5	8.4	8.0	8.2	8.0
Netherlands	5.5	8.6	8.6	8.3	8.1	8.2	7.8	7.9	7.9
Portugal	9.8	13.1	10.8	12.9	11.7	10.7	11.4	10.5	10.6
Spain	12.0	13.9	13.5	11.8	10.8	10.2	9.7	9.7	9.6
Sweden	5.8	6.3	5.6	5.3	5.8	6.0	6.0	6.1	5.7
United Kingdom	5.3	6.8	7.3	7.2	7.6	7.1	7.1	7.2	7.2
The rest of Europe									
Bulgaria, Republic of	6.7	8.2	8.7	8.8	9.4	7.1	6.0	6.3	5.6
Croatia, Republic of						9.5	9.9	11.3	11.5
Yugoslavia, former	7.9	8.1	7.8	7.7	7.3				
Slovenia, Republic of						8.6	8.1	7.8	7.7
Czech Republic						12.7	12.3	12.5	11.7
	10.7	11.6	11.9	11.5	11.3				
Slovak Republic						7.1	7.3	8.4	10.1
Hungary	9.9	11.0	12.9	12.6	12.1	12.2	10.7	10.7	11.0
Norway	3.6	4.3	4.6	4.1	4.1	3.8	3.8	3.8	3.9
Poland	5.1	6.9	8.4	6.7	6.7	6.1	6.0	6.2	6.2
Romania	5.8	7.3	7.6	7.4	8.5	8.7	8.6	8.7	9.0
Russian Federation						5.6	5.5	5.3	5.2
USSR, former	6.5	6.6	8.5	7.1	4.8				
Ukraine						4.7	4.7	4.1	3.8
Switzerland	10.8	10.7	11.1	11.5	11.4	10.5	10.2	9.8	9.8
Africa									
Nigeria	..	..	..	..	..	2.1	..	..	..
South Africa	3.0	3.6	3.8	4.2	5.1	4.9	4.6	4.8	4.8
Asia									
China	..	..	..	..	..	..	3.2	3.3	3.5
Japan (includes sake)	4.8	5.4	5.6	6.1	6.5	6.7	6.8	6.8	6.8
Korea, Republic of (beer only)	..	..	..	..	1.2	1.4	1.4	1.5	1.5
Philippines	..	..	4.0	3.7	..	..	..	..	..
Australasia									
Australia	7.8	9.2	9.4	9.2	8.4	7.8	7.8	7.9	7.7
New Zealand	6.3	7.8	8.2	8.0	7.9	7.5	7.4	7.4	7.2
North America									
Canada	6.4	8.3	8.7	8.0	7.3	6.5	6.2	6.1	6.1
United States of America	7.0	7.7	8.1	7.8	7.4	6.8	6.6	6.6	6.6
Central and South America									
Argentina	..	..	11.5	8.8	7.6	7.8	7.7	7.4	7.7
Brazil (beer and wine)	0.7	1.0	1.3	1.4	2.5	2.0	2.0	2.2	2.8
Chile (beer and wine)	5.6	5.4	6.4	5.5	4.5	4.5	3.6	3.2	3.3
Colombia (beer only)	1.4	1.4	1.8	2.3	2.5	2.1	2.4	2.4	2.1
Cuba (beer only)	0.8	1.1	1.2	1.3	1.5	1.5	0.9	0.7	0.6
Mexico	1.9	2.0	2.5	2.5	2.7	2.9	2.9	2.9	2.8
Peru (beer and wine)	1.0	1.5	1.6	1.6	1.4	2.0	1.6	1.5	1.7
Venezuela (beer and wine)	2.6	2.5	3.8	3.1	3.4	3.8	3.8	3.6	3.8

Source: Brewers and Licensed Retailers Association, *Statistical Handbook. A Compilation of Drinks Industry Statistics* (London: Brewing Publications Limited, 1997).

NOTE: These figures include beer, wine and spirits and exclude cider.



sumption in the country (Shkolnikov and Nemstov, 1997). Similar findings have been reported for other Eastern European countries (World Health Organization, 1997b).

The World Health Organization is currently compiling national alcohol profiles similar to what has been done for tobacco (see World Health Organization, 1997a). Preliminary findings from this study confirm that alcohol consumption is rising rapidly in many developing regions. For example, Brazil, China, India, Mexico, Nigeria, South Africa and Uganda all report that beer consumption has risen sharply since the 1970s. In Asian populations, almost all alcohol is consumed by men; in Africa and Latin America, prevalence among women is also significant, but tends to be much lower than for men.

Women also generally consume alcohol in less hazardous ways than men do. For example, in South Africa, several studies have reported that approximately 30 per cent of African men drink at levels considered to be hazardous for health, and are five times more likely to drink heavily on the weekend than on weekdays (C. Parry, personal communication). Evidence from other African countries confirms that heavy and hazardous drinking is common among males. For example, a 1993 survey in Nigeria found that nearly three-quarters of men in the sample who consumed alcohol reported drinking at least three bottles of beer in a typical drinking session.

This pattern of hazardous consumption is also found in many developed countries where typically 10-20 per cent of the men (and somewhat lower proportions of women) drink at hazardous levels (World Health Organization, 1997b). There has, however, been some progress towards reducing hazardous drinking in many countries. For example, in France, the proportion of drinkers consuming more than 30 grams of alcohol daily (3 standard drinks) declined from 44 per cent in 1980-81 to 36 per cent in 1991-1992 among men, and from 9.5 per cent to 5.1 per cent among women. Whilst not all, and perhaps not even the majority of alcohol-related harm occurs among heavy drinkers, significant reductions in hazardous consumption

must remain a public health priority, along with significant reductions in overall consumption.

### C. QUANTIFYING POPULATION-ATTRIBUTABLE MORTALITY

#### 1. *Cigarette smoking*

Estimates of the population-attributable risk (PAR) for a given risk factor or exposure in a defined population are generally calculated using the classical attributable risk formula which relates the prevalence of exposure (P) to the relative risk (RR) of death from a specific cause for those exposed compared to those not exposed (see Miettinen, 1974). While this may be theoretically correct, the practical application of this approach will always be difficult since observed population prevalence (P) for any population is not an adequate representation of population exposure corresponding to an observed set of relative risks. For example, in the case of smoking, current smoking prevalence does not include other critical aspects of exposure to tobacco smoke such as duration, amount, degree of inhalation, etc., all of which affect relative risks for disease. Looked at from the point of view of relative risks, even if one has reliable estimates of smoking prevalence for a population, to what set of relative risks should this "exposure" variable be applied?

Peto, Lopez and colleagues (see Peto and others, 1994) have proposed an indirect method to estimate smoking-attributable mortality which circumvents these theoretical difficulties with the attributable-risk approach. Their method is based on the assumption that the excess (over and above non-smoker rates) lung cancer rate observed in a population is the best indicator of cumulative population exposure to smoking hazards. A "Smoking-Impact Ratio" (SIR) is calculated as the relative excess of lung cancer in the observed population compared with the observed excess of smokers over non-smokers, calculated from the CPS-II follow-up for 1984-1988. This SIR is then used to scale the relative risks from CPS-II in order to apply them to other populations. To allow for confounding, and in an attempt not to exaggerate the hazards

of smoking, these "scaled" relative risks were then halved and applied to national cause of death data for those populations (all developed countries) where the assumptions of the method might be reasonably applicable.

The method is crude and somewhat arbitrary, but validation against national studies using the classical attributable-risk approach suggests that it does not yield wildly implausible results (Valkonen and van Poppel, 1997). The advantage of the method is that it yields annual estimates of deaths from smoking by age, sex and cause which have been calculated for all developed countries for the period 1955-1995 and published elsewhere (Peto and others, 1994).

A summary of the levels and trends of smoking-attributable mortality estimated from the application of the Peto-Lopez method to mortality data for developed countries is given in tables 70 and 71. Between 1950 and 2000, smoking is estimated to be the cause of about 62 million deaths in the developed countries (Europe, including the former USSR, Australia, Japan, North America, New Zealand), most of these being men (52 million). Current (1995) annual mortality from smoking in the developed countries is estimated to be about 1.9 million (1.44 million men, 0.48 million women), 1.2 million of which occur in the established market economies and the remainder in the former socialist economies of Europe.

Estimates of current annual mortality from smoking in less developed regions are much more difficult to prepare given the lack of representative, direct evidence from epidemiological

studies on tobacco hazards. Given the large background risks of lung cancer among non-smokers in many developing regions, and the lack of reliable cause of death data, the Peto-Lopez method is not likely to be widely applicable in these countries without appropriate adjustment. The single exception is China, where recent analyses of large retrospective and prospective evidence suggests that smoking is already killing about 800,000 people each year, mostly men (Liu and others, 1998).

Using a modification of the Peto-Lopez method, Murray and Lopez (1997a) have prepared estimates of smoking-attributable mortality for other developing region as well. Firstly lung cancer mortality rates were estimated for each region, by age and sex, using methods reported elsewhere (Murray and Lopez, 1997a). Next, the non-smoker lung cancer rates in each region were estimated on the basis of local epidemiological evidence, and the SIR in the Peto-Lopez method adjusted (lowered) accordingly. These revised "scalars" were then applied to estimated regional cause of death patterns in 1995 in the manner adopted by Peto and others (1994) (see table 72).

To the extent that this method is reasonable for developing countries, smoking is estimated to cause about 1.6 million deaths each year in developing countries, half of them in China alone. Most deaths in developing countries (1.4 million) occur among men, which is at least consistent with the sex differences in exposure described earlier. Annual mortality from smoking in developing countries is also much lower than in more developed regions,

TABLE 70. ESTIMATED DEATHS CAUSED BY SMOKING IN DEVELOPED COUNTRIES, 1950 TO 2000

Mid-decade year	Men			Women		
	No. of deaths in mid-decade year	Per cent of all deaths	Per cent of deaths at ages 35-69	Number	Per cent of all deaths	Per cent of deaths at ages 35-69
1955	447 000	10	20	26 000	<1	2
1965	793 000	17	28	70 000	2	4
1975	1 119 000	21	31	165 000	3	7
1985	1 369 000	24	35	317 000	6	11
1995	1 442 000	25	36	476 000	9	13
Total of all deaths caused by smoking (1950-2000)	52 million	20	30	10.5 million	4	7

Source: R. Peto, A. D. Lopez, J. Boreham, M. Thun and C. Heath, *Mortality from Smoking in Developed Countries, 1950-2000* (Oxford, Oxford University Press, 1994).

TABLE 71. ESTIMATED PERCENTAGE OF DEATHS CAUSED BY SMOKING, BY SEX, AGE AND MAJOR CAUSE OF DEATH GROUPINGS, ALL DEVELOPED COUNTRIES, 1995

Sex	Age	All causes	All cancer	Lung cancer	Upper aero-digestive cancer*	Other cancer	Chronic obstructive pulmonary disease	Other respiratory diseases	Vascular diseases	Other causes
Men	35-69	36	50	94	70	18	82	29	35	35
	70 and above	21	36	91	59	13	73	11	12	12
	All ages	25	43	92	66	15	75	14	21	18
Women	35-69	13	13	71	34	2	55	16	12	15
	70 and above	8	13	74	38	2	54	7	5	6
	All ages	9	13	72	36	2	53	7	6	7
Both sexes	35-69	28	35	89	65	10	73	25	28	27
	70 and above	13	25	86	52	7	65	9	8	8
	All ages	17	30	87	60	8	66	10	13	12

Source: R. Peto, A. D. Lopez, J. Boreham, M. Thun and C. Heath, *Mortality from Smoking in Developed Countries, 1950-2000* (Oxford, Oxford University Press, 1994).

\*Cancers of the mouth, oesophagus, pharynx and larynx.

TABLE 72. ESTIMATED NUMBER OF DEATHS FROM TOBACCO BY REGION, 1995

	Men	Women	Total
Developed countries	1 440 000	475 000	1 915 000
Established market economies	840 000	375 000	1 215 000
Formerly socialist economies of Europe	600 000	100 000	700 000
Developing countries	1 385 000	195 000	1 580 000
China	690 000	120 000	810 000
Other Asia and North Africa	500 000	40 000	540 000
Sub-Saharan Africa	90 000	5 000	95 000
Latin America and the Caribbean	105 000	30 000	135 000
World	2 825 000	670 000	3 495 000

Sources: For developed countries: R. Peto, A. D. Lopez, J. Boreham, M. Thun and C. Heath, *Mortality from Smoking in Developed Countries, 1950-2000* (Oxford, Oxford University Press, 1994); for developing countries: C. J. L. Murray, and A. D. Lopez, "Quantifying the burden of disease and injury attributable to ten major risk factors," in *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries and Risk Factors in 1990 and Projected to 2020*, C. J. L. Murray and A. D. Lopez, eds. (Cambridge, MA, Harvard University Press, 1996).

again a finding that is consistent with the observation that cigarette consumption, until recently has been relatively low in much of the developing world.

The pattern is changing rapidly, however. It is perhaps even more relevant for public health to ask what will be the mortality in the future if current smoking trends persist, than to estimate current i.e. 1995 smoking deaths (about 3.5 million) from past smoking levels. Based on projections of causes of death to 2020, and projections of lung cancer, Murray and Lopez (1997b) have projected that smoking will cause

about 8.4 million deaths annually by 2020; 6.0 million of them will, on present trends, occur in the developing world. Smoking by the year 2020 is projected to be by far the leading cause of death in the world. A similar annual death toll has been forecast by Peto and Lopez (1991) using much cruder methods, with a prediction of 10 million deaths a year from smoking around the late 2020s/early 2030s.<sup>2</sup>

## 2. Alcohol consumption

Estimating national and international mortality from alcohol use is even more difficulty

given the generally less conclusive, yet more complex epidemiological evidence, the paucity of data on drinking patterns and their health effects, and the lack of reliable information on population-level consumption against which to validate estimates of mortality. Furthermore, the lack of a convenient "index" condition such as lung cancer for smoking makes the application of a Peto-Lopez indirect approach very difficult.

Some attempts at national level have been made to estimate alcohol-attributable mortality (see, for example USDHHS, 1993; English and others, 1995) but these have typically focussed only on deaths caused by alcohol, and have not made adequate allowance for any cardio-protective effect. A very crude attempt at estimating the components of alcohol's contribution to mortality at global and regional level has been made by Murray and Lopez (1996) using qualitative scaling of disease and injury-specific attributable fractions estimated for industrialised countries to allow for probable regional variations in alcohol consumption and drinking patterns.

Based on the evidence about the role of alcohol in disease and injury outlined in section 1, mortality estimates were produced for (a) diseases caused by alcohol, (b) ischaemic heart disease (protective), and (c) injuries attributable to alcohol. The methods were as follows: for injuries, based on the meta-analysis of epidemiological studies carried out by English and others, (1995), the attributable fractions proposed for Australia from this study were assumed to apply to the established market economies. These are shown in table 72 (age-specific fractions were in fact calculated but are not shown in the table) and indicate, for example, that 37 per cent of male road traffic deaths (and 18 per cent of female deaths) are attributable to alcohol. For other regions, these attributable fractions were qualitatively scaled on the basis of evidence and opinion from each region about consumption levels and typical drinking patterns. These scalars for males varied from 0.2 for the Middle Eastern Crescent (MEC) (North Africa/Western Asia Region) to 1.5 for the former socialist economies of Europe (FSE) and from 0.1 to 0.9 for women (the extremes being the same regions).

Deaths from diseases attributable to alcohol have been estimated separately using a two-stage procedure. For the established market economies (EME), the attributable fractions estimated from the English and others meta-analysis were used. For other regions, these attributable fractions were first scaled to reflect regional differences in estimated per adult alcohol consumption based on the reviews of regional consumption by Smart (1991) and Simpura (1995). Next, the estimated fraction of cirrhosis of the liver mortality attributable to hepatitis B was estimated for each region. Finally, one minus this fraction was then used to adjust the scaled attributable fractions to estimate the regional patterns of alcohol-attributable deaths (see Murray and Lopez, 1996 for further details).

The extent of the protective effect for ischaemic heart disease suggested by the literature was based on the evidence from the two recent large-scale epidemiological studies in the United Kingdom (Doll and others, 1994) and the United States of America (Thun and others, 1997). The pooled estimate of IHD deaths "saved" by alcohol consumption in the EME, based on these studies and estimates of abstinence levels in men and women, was 0.18 for men and 0.16 for women. These protective fractions were then scaled according to regional information on abstinence and consumption, by sex, to estimate applicable fractions for IHD.

Table 73 provides a summary of the global and regional patterns of alcohol mortality estimated by Murray and Lopez (1996) based on these procedures. The net annual global mortality from alcohol use is estimated at around 775,000 deaths, 640,000 of which are estimated to occur in developing regions. Of these 775,000 net deaths caused by alcohol, a little over 710,000 are estimated to occur in males. This net mortality is in fact the result of an estimated 1.25 million deaths caused by alcohol (625,000 from injuries, 620,000 from diseases) and an estimated 470,000 deaths "saved" from ischaemic heart disease.

There are, as might be expected, marked regional variations in the pattern of alcohol-related mortality. The estimated impact of alcohol on injuries is particularly high in Sub-

TABLE 73. ESTIMATED ALCOHOL-ATTRIBUTABLE FRACTIONS FOR SELECTED INJURIES,  
ALL AGES, ESTABLISHED MARKET ECONOMIES, EARLY 1990s

<i>Injury</i>	<i>Males</i>	<i>Females</i>
Road traffic accidents	0.37	0.18
Falls	0.34	0.34
Fires	0.44	0.44
Drowning	0.34	0.34
Suicide	0.12	0.08
Homicide	0.47	0.47

Source: D. R. English, C. D. J. Holman, E. Milne and others, *The Quantification of Drug-Caused Morbidity and Mortality in Australia, 1995 edition* (Canberra, Commonwealth Department of Human Services and Health, 1995).

Saharan Africa (120,000 male deaths, 15,000 female deaths) per year, and in Latin America (75,000 deaths among males, 10,000 among females). Almost 100,000 alcohol-attributable injury deaths occur in the EME each year, 70 per cent of them among males. Overall, for the EME region, alcohol is estimated to "save" about as many lives from ischaemic heart disease as it claims from all other diseases combined (160,000 - 170,000 per year) (table 74). However, the age-distribution of these deaths caused by alcohol-induced disease is much younger than those protected from IHD (see figure 115). This, together with the 100,000 or so injury deaths attributable to alcohol, the majority of which occur among young and middle-aged adults, make alcohol use a major concern for public health in all regions where it is consumed in significant quantities.

#### D. POLICY RESPONSES FOR TOBACCO AND ALCOHOL

##### 1. Tobacco use

The World Health Organization has repeatedly called for comprehensive tobacco control programmes and policies containing the following nine elements:

- measures to ensure that non-smokers receive effective protection from involuntary exposure to tobacco smoke, in enclosed public places, restaurants, transport, and places of work and entertainment;

- measures to promote abstinence from the use of tobacco so as to protect children and young people from becoming addicted;
- measures to ensure that a good example is set in all health-related premises and by all health personnel;
- measures leading to the progressive elimination of those socio-economic, behavioural, and other incentives that maintain and promote the use of tobacco;
- prominent health warnings, which might include the statement that tobacco is addictive, on cigarette packets, and containers of all types of tobacco products;
- the establishment of programmes of education and public information on tobacco and health issues, including smoking cessation programmes, with active involvement of the health professions and the media;
- monitoring of trends in smoking and other forms of tobacco use, tobacco-related diseases, and effectiveness of national smoking-control action;
- the promotion of viable economic alternatives to tobacco production, trade and taxation;
- the establishment of a national focal point to stimulate, support, and co-ordinate all the above activities.

Many elements of an effective and comprehensive tobacco control policy will eventually involve some form of legislative action, whether in the form of adopting or amending laws,

TABLE 74. ESTIMATED DEATHS CAUSED (OR PREVENTED) BY ALCOHOL USE, BY REGION, 1990  
(in thousands)

Region	Males			Females		
	Injuries	Diseases	"Protected"	Injuries	Diseases	"Protected"
Established market economies	70	115	-99	28	45	-76
Formerly Socialist economies of Europe	84	51	-62	15	19	-54
India	65	66	-37	16	24	-21
China	60	39	-5	10	17	-6
Other Asia and Islands	34	69	-20	6	21	-13
Sub-Saharan Africa	119	46	-13	16	17	-14
Latin America and Caribbean	85	62	-23	10	18	-15
Middle Eastern Crescent	7	5	-5	1	3	-6
World	524	454	-265	102	165	-206

Source: C. J. L. Murray, and A. D. Lopez, "Quantifying the burden of disease and injury attributable to ten major risk factors", in *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries and Risk Factors in 1990 and Projected to 2020*, C. J. L. Murray and A. D. Lopez, eds. (Cambridge, MA, Harvard University Press, 1996).

regulations or government decrees. These include:

- protection for children from becoming addicted to tobacco;
- effective protection from involuntary exposure to tobacco smoke;
- prominent health warnings on tobacco product packaging;
- the use of financial measures, such as higher tobacco taxes, to discourage tobacco consumption.

#### *Protecting children from becoming addicted to tobacco*

In the early 1990s, about 25 countries had laws that prohibited the sale of cigarettes to minors, with the age of prohibition varying from 16 to 21 years of age. Related measures often include bans or restrictions on cigarette sales from vending machines, prohibitions on sales of tobacco products and smoking in schools, prohibiting the offering of free samples of tobacco products and prohibiting the sales of single cigarettes. Unfortunately, even when such laws exist, relatively few countries have been suc-

cessful in ensuring that laws banning the sale of cigarettes to minors are respected.

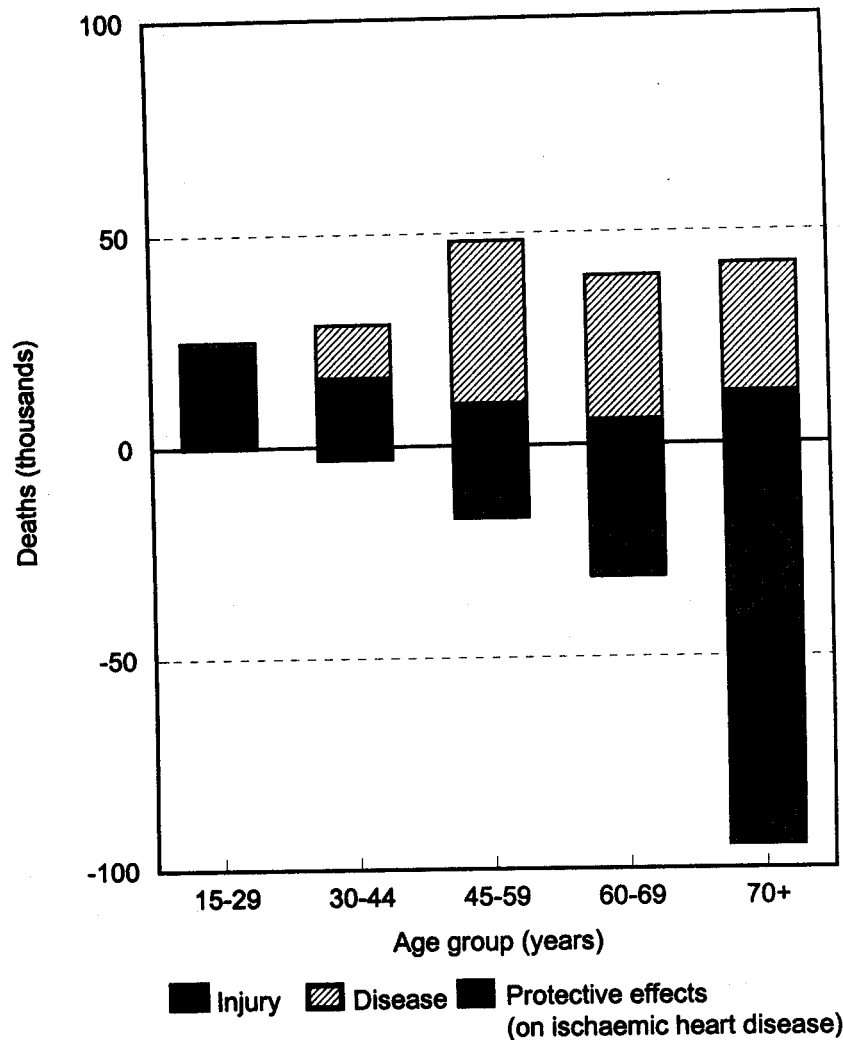
#### *Protection from involuntary exposure*

Many jurisdictions have laws that ban or restrict smoking in public places, workplaces, and transit vehicles. Generally, these laws are most successful if they were implemented once a large number of enterprises had already taken action to ban or restrict smoking. The laws serve to guarantee that some level of protection is extended to 100 per cent of public places, workplaces and transit vehicles.

Successful policies or legislation to restrict or prohibit smoking in workplaces, public places and transit vehicles generally have the following characteristics:

- flexibility for adaptation to local situations;
- labour-management consultations;
- employee involvement in decision-making via questionnaires and other means;

**Figure 115. Male deaths attributable to, and averted by, alcohol use, Established Market Economies (EME), 1990**



Source: C. J. L. Murray and A. D. Lopez, "Quantifying the burden of disease and injury attributable to ten major risk factors", in *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries and Risk Factors in 1990 and Projected to 2020*, C. J. L. Murray and A. D. Lopez, eds. (Cambridge, MA, Harvard University Press, pp. 295-324, 1996).

- education and information sessions;
- work-site smoking cessation programmes;
- suitable phase-in-periods for implementation of policy and enforcement strategies.

#### *Health promotion and education*

Various elements of effective health promotion and health education programmes include

celebration of no-tobacco days, media advocacy, the use of paid media advertising, school-based health promotion programmes, community-based health promotion programmes and sponsorship of cultural, sporting and community events. Countries with successful comprehensive tobacco control programmes incorporate most or all of these strategies in their tobacco control policies and programmes. Many countries have successfully offset the costs of operating such programmes with a portion of the

revenue collected from tobacco taxes. Programmes have proven to be most effective when they are part of a comprehensive tobacco control policy, as has been recommended by the World Health Assembly.

### *Smoking cessation*

Smoking cessation programmes also achieve best results when they are part of a broad range of comprehensive tobacco control policies and programmes. However, even in a favourable and supportive environment, dependence on nicotine, an addictive drug, remains a significant barrier to successful smoking cessation. Where this dependence has been measured, about 75-80 per cent of smokers want to quit and about one-third have made at least three serious attempts to do so. The reality is that less than half of all smokers succeed in stopping permanently before the age of 60.

Smoking cessation strategies ideally would train all health professionals, including doctors, nurses, pharmacists and dentists in techniques of providing smoking cessation counselling and advice. It would also be desirable to make available a broad range of smoking cessation strategies, including group counselling, physician advice and, where appropriate, nicotine replacement therapy.

Telephone counselling services (quit lines) to assist people who wish to quit smoking can be a valuable component of an overall smoking cessation strategy. In Australia and South Africa, quit-line telephone numbers are included along with required health information printed on every package of cigarettes.

### *Health warnings*

In the early 1990s, about 80 countries required health warnings to appear on packages of tobacco products. However, in most countries the warnings are small, inconspicuous and provide little information about the many serious health consequences of tobacco use. Evaluation studies have found such warnings to be ineffective.

By the mid-1990s, however, a number of countries had adopted much more stringent

warning systems, involving direct statements of health hazards, multiple messages, as well as large and prominent message display. Such warnings are now required in a number of countries including Australia, Canada, Iceland, Norway, Singapore, South Africa and Thailand. Greatly enhanced effectiveness has been found with legislated health warning systems that have the following characteristics:

- multiple warnings, with each appearing in approximately equal proportion on packages of tobacco products;
- warnings on all kinds of tobacco products, with the text appropriate to each product;
- strong, uncompromising messages in the text of each warning such as "Smoking harms your family", "Smoking causes cancer", "Cigarettes are addictive", "Smoking causes heart disease" and "Tobacco smoke causes fatal lung disease in non-smokers".
- display in black-on-white or white-on-black format, occupying 20 per cent of more of the largest surfaces of packages of tobacco products.

### *Advertising bans*

A number of countries have successfully passed laws to ban all or nearly all forms of tobacco advertising as part of comprehensive tobacco control measures. Frequently, however, further legislative action has been necessary to tighten the restrictions on advertising, as bans on tobacco advertising are frequently circumvented by the use of indirect advertising and other means.

As of 1990, 27 countries had total or near-total bans on advertising. Since then, however, the number has declined to 18. While Australia and Kuwait recently implemented bans on tobacco advertising, tobacco advertising bans that had been in place became inoperative in Canada and the newly independent states of Central and Eastern Europe. However, Canada and many Central and East European countries are considering draft legislation to re-establish bans on tobacco advertising.

Experience with partial bans on tobacco advertising has shown them to be less effective



than first envisioned. For example, tobacco advertisements were banned from radio and television in the United States in the early 1970s, but later analysis showed that total tobacco advertising volume and expenditure continued to increase. Advertising placements were simply transferred to other media.

### *Price policies*

A number of studies from several countries have shown that for every 10 per cent increase in the price of tobacco products, consumption can be expected to decline by two to eight per cent (Townsend, 1993). Price has been found to have an even stronger effect on reducing tobacco consumption among two key target groups - adolescents and people with lower socio-economic status (Townsend, 1993). While the size of the effect may vary, it has been clearly demonstrated that raising the price of tobacco products will reduce tobacco consumption.

### *Effectiveness of comprehensive tobacco control policies*

Perhaps the most comprehensive review of the effectiveness of tobacco control policies is the study by Laugesen and Meads (1991) which used multiple regression techniques to evaluate the effect of advertising restrictions, price and income on tobacco consumption in 22 OECD countries from 1960 to 1986. Above threshold levels, both price relative to income and advertising restrictions were found to be significant in decreasing tobacco consumption. Moreover, the most comprehensive tobacco control programmes that included both high prices for tobacco products, and comprehensive bans on tobacco advertising, together with stringent health warning labelling requirements for cigarette packages, had the strongest effect on reducing tobacco consumption. The authors estimate that a ban on tobacco advertising, including strong and varied health warnings on packages, together with a 36 per cent increase in real price, would have reduced tobacco consumption by 13.5 per cent in 1986, had any of the OECD countries under study taken such measures in that year.

Several countries now have comprehensive tobacco control policies of the sort recommended by the World Health Assembly. The positive experience of these countries with these policies (the United Kingdom and Finland are perhaps the most notable examples in terms of lung cancer reductions), and the clear evidence of their effectiveness should serve to encourage a wider adoption of comprehensive national tobacco control policies.

## *2. Alcohol use*

Against a background of increasing awareness and evidence of the significant public health impacts of alcohol, many countries and communities are starting to rethink and strengthen their responses to these problems.

Experience from around the world has shown that alcohol use and related problems cannot be significantly reduced by any single and limited measure. It is well documented that a single strategy undertaken in isolation is unlikely to produce real outcomes in terms of health risk and that, to a large extent, a range of interdependent strategies and a mix of initiatives are required if lasting change is to be achieved. Responding effectively to the problem of alcohol-related health and social harms therefore requires a comprehensive and co-ordinated approach across a range of organizations and agencies.

A comprehensive approach is likely to include measures to regulate the purchase and consumption, pricing, marketing, wholesale and retail sale, import and export, and production of alcohol, and the production of the raw materials for making alcohol. The policy options for exercising these regulations vary from country to country and can be broadly categorised into the following:

- price and taxation controls
- restrictions on availability of alcohol
- purchasing restrictions - sales to minors, intoxicated persons, etc.
- increasing the probability of detection and punishment for alcohol related offences - e.g. drinking driving deterrence measures

- regulation of advertising, marketing and promotion of alcoholic beverages
- health warnings and contents labelling
- controls on production
- education of the community - mass media campaigns, community based programmes, family based programmes
- education of health and welfare workers to detect and treat alcohol problems.

Although there is general agreement among experts and policy-makers over the strategies to be followed to address the negative consequences of alcohol use, many controversies and doubts still exist concerning the right balance to be struck between different strategies and the best ways and means to achieve improvements in public health (Lehto, 1995). To be effective, such control measures should be developed within a public health policy framework which seeks to create a balance between the various strategies, while at the same time developing a level of public support for the policy measures to be applied. A number of these specific policy measures are discussed below.

A large number of studies of the effects of price changes on alcohol consumption have now been carried out in a wide range of countries. Some of these studies have looked into the direct effects of price changes on levels of alcohol-related harm. The main conclusion of the studies has been that alcoholic beverages are like any other commodity in respect of price. All other factors being constant, an increase in the price relative to other commodities generally reduces per capita consumption of alcohol. In addition, it is clear that even heavy drinkers and alcohol dependent drinkers are influenced by price changes, at least as much if not more than light drinkers. Such price changes are generally manipulated through specific taxation measures. To take an example, the government in Gambia has introduced a tax structure to discourage the importation and consumption of alcohol. The duty on production, import and export of alcoholic beverages is increased annually. The high taxes and duties are expected to indirectly raise the price of both locally produced and imported alcohol and to limit consumption by curtailing buying power (Kortteinen, 1989).

Restrictions on availability are being increasingly used in response to research which shows that such restrictions on access can restrain consumption and harm. Typical restrictions include controls over the density of outlets, controls over opening hours and days of sale, and minimum drinking ages.

Despite evidence that controls over availability are effective measures for reducing alcohol-related public health problems, the general trend in many countries seems to be towards an extension of opening hours. Countries where such controls are still prominent include Botswana, where bottle stores are only able to conduct business between the hours of 10 am and 7 pm, and are closed on Sunday, and New Zealand, where bottle stores are also closed on Sundays.

Currently about fifty-five countries around the world have introduced laws that prohibit the sale and supply of alcohol to young people. The age of prohibition varies from 16 to 21 years. Despite this widespread introduction of age restrictions, it appears that enforcement of this policy measure is weak in many countries.

In addition to these restrictions on the sale and supply of alcohol to young people, a number of countries have devoted significant attention to enforcing restrictions over the supply of alcohol to intoxicated persons. Measures introduced typically focus on training the servers of alcohol to ensure that they restrict the sale and supply of alcohol to a person who has reached the point of intoxication. Such approaches are well developed in Canada, the United States, and the Netherlands.

Nearly all countries have introduced some form of product restrictions, usually through the development of licensing systems. In many countries the producer, importer, or wholesaler of alcohol needs a government licence. A licensing system can be used for many purposes. For example, when combined with limits on the amounts that can be produced, it can be used to restrict the volume of various beverages for consumption.

Another common policy measure has been to introduce restrictions over activities allowed under the influence of alcohol. Such activities include driving a motor vehicle, piloting a plane or boat. The most widely known are the restrictions on driving a motor vehicle while under the influence of alcohol. These have been progressively created and tightened over the last few decades, and BAC limits for driving are now in place in well over 50 countries, with limits ranging from 0mg per 100 ml of blood (e.g., Ukraine, Czech Republic, Azerbaijan) to 100 mg per 100 ml of blood (e.g., India). The vast majority of countries, however, have set limits at either 50 or 80mg per 100ml of blood, levels at which the risk of accident is still roughly two and four fold higher, respectively, over drivers with a zero BAC. Again, there appear to be wide variations in the degree to which these limits are enforced. Australia has introduced a zero level for learners and drivers under 25 years of age, and random breath testing of drivers by the police is widespread.

Alcohol is one of the most heavily advertised and promoted commodities around the world. A major review of bans on alcohol advertising revealed significant effects, with countries having advertising bans showing lower levels of consumption and lower motor vehicle fatality rates (Edwards and others, 1994). The importance of measures to restrict and control the way alcohol is marketed and promoted has received growing consideration over the last decade. Most industrialised countries and a number of developing countries have introduced at least a few restrictions in this area. Such restrictions are usually aimed either at the controlling the content and targeting of particular advertisements or promotions, or at effecting partial or total bans in the various media (print, radio, television, outdoor advertising). Controls are typically introduced through specific legislation and regulations, or through voluntary agreements or codes developed with various industry groups. Examples of countries that have developed voluntary codes for restricting advertising include Austria, Belgium, Czech Republic, Georgia and New Zealand. More formal legislative mechanisms to regulate alcohol advertising have been introduced in a number of countries including Ukraine, Venezuela, and Brazil (with the exception of beer).

Of the softer policy approaches, mass education on the harmful effects of drinking has also been widely supported in a number of countries. However, despite their widespread appeal, evaluations have shown that such campaigns by themselves are likely to have a limited effect on drinker's knowledge and attitudes, and almost no impact on their behaviour. Where these campaigns are likely to be more effective is when they are introduced in conjunction with other measures, for example police enforcement of drink-driving laws. Such campaigns can also play a valuable role in increasing public support for other policy measures aimed at reducing the public health consequences of alcohol.

As with many regulatory measures, their effectiveness largely depends on the rigour with which they are enforced. Naturally this varies from country to country, and is also significantly influenced by the level of resources (both human and financial) which can be dedicated to their enforcement. Restrictions such as those on the sales to minors and to intoxicated persons are only effective if their enforcement is effective and if they have broad popular support. Drink-driving laws can only be effective as a deterrent if there is a reasonable chance that a driver who drinks will be apprehended by the police and charged with an offence.

Getting the policy balance and public support to introduce the required measure to address the public health consequences of alcohol is critical to success. A comprehensive alcohol policy cannot be based only on a scientific evaluation of the effectiveness of different policies. It also needs political wisdom, political commitment and political skills to broaden support for long-term public health alcohol policy (Lehto, 1995).

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#### NOTES

<sup>1</sup>This paper will focus only on the health risks of cigarette smoking. Many studies have documented that other forms of tobacco use, especially chewing, pipes and cigars are associated with an increased risk of cancers of the upper aerodigestive tract (e.g., lip, tongue, mouth, pharynx, larynx and oesophagus) but, in general, these risks are less extreme than for smoking (see Doll and Crofton, 1996 for a more extensive review).

<sup>2</sup>Essentially these projections are based on assumptions about the estimated number of young people currently alive at ages 0-19 years who will, on present patterns, become regular smokers (about 800 million) and the proportion of them, again on current evidence, who will eventually be killed by tobacco (about 300 million), combined with estimates of the number of current smokers (1.1 billion) who might be expected to die in different decades during the first half of the 21st century.

#### REFERENCES

- Anderson, P. (1995). Alcohol and risk of physical harm. In *Alcohol and Public Policy: Evidence and Issues*, H.D. Holder and G. Edwards, eds. Oxford: Oxford University Press, pp. 82-113.
- \_\_\_\_\_, A. Cremona, A. Paton, C. Turner and P. Wallace (1993). The risk of alcohol. *Addiction*, vol. 88, pp. 1493-1508.
- Andreasson, S., A. Romelsjö, and P. Allebeck (1988). Alcohol and mortality among young men. Longitudinal study of Swedish conscripts. *British Medical Journal*, vol. 296, pp. 1021-1025.
- Best, E. W. R., G. H. Josie and C. B. Walker (1961). A Canadian study of mortality in relation to smoking habits: A Preliminary report. *Canadian Journal of Public Health*, vol. 52, pp. 99-106.
- Bofetta, P., and L. Garfinkel (1990). Alcohol drinking and mortality among men enrolled in an American Cancer Society prospective study. *Epidemiology*, vol. 1, pp. 342-348.
- Brewers and Licenced Retailers Association (1997). *Statistical Handbook. A Compilation of Drinks Industry Statistics*. London: Brewing Publications Limited.
- Cederlof, R., L. Friberg, Z. Hornbec and U. Lörich (1975). The Relationship of Smoking and Some Social Covariables to Mortality and Cancer Morbidity. A Ten Year Follow-up in a Probability Sample of 55,000 Swedish Subjects Age 18-69, Part 1/2. Stockholm: Karolinska Institute.
- Coates, R.A., and others (1986). Risk of fatty infiltration or cirrhosis of the liver in relation to ethanol consumption: a case-control study. *Clinical and Investigative Medicine*, vol. 9, pp. 26-32.
- Collishaw, N. C. (1987). Cigarette consumption in Canada, 1981-1986. In *Smoking and Health, 1987. Proceedings of the 6th World Conference on Smoking and Health, Tokyo, 9-12 November 1986*. M. Aoki, S. Hisamishi and S. Tomimaga, eds. Amsterdam Elsevier Science Publications, pp. 701-703.
- Doll, R. (1986). Tobacco: an overview of health effects. In *Tobacco: A Major International Health Hazard*. IARC Scientific Publications No 74, D. Zaridze and R. Peto, eds. Lyon, International Agency for Research on Cancer, pp. 11-22.
- \_\_\_\_\_, and J. Crofton, eds. (1996). *Tobacco and Health*. British Medical Bulletin, vol. 52, No. 1 (special issue). London: Royal Society of Medicine Press Limited.
- Doll, R., D. Forman, C. La Vecchia and F. Woutersen (1993). Alcoholic beverages and cancers of the digestive tract. In *Health Issues in Relation to Alcohol Consumption*, P.M. Verschuren ed. Brussels: ISLI Press, pp. 125-166.
- Doll, R., and A. B. Hill (1950). Smoking and carcinoma of the lung-Preliminary report. *British Medical Journal*, vol. ii, pp. 739-748.
- \_\_\_\_\_, (1956). Lung cancer and other causes of death in relation to smoking. A Second report on the mortality of British doctors. *British Medical Journal*, vol. 2, pp. 1071-1081.
- Doll, R., Richard Peto, Keith Wheatley, Richard Gray and Isabelle Sutherland (1994). Mortality in relation to smoking: 40 years observations on male British doctors. *British Medical Journal*, vol. 309, pp. 901-911.
- Dorn, J. R., G. Linden and L. Breslow (1960). Lung cancer mortality experience of men in certain occupations in California. *American Journal of Public Health*, vol. 50, no. 10, pp. 1475-1487.
- Duffy, J. C., ed. (1992). *Alcohol and Illness: the Epidemiological Viewpoint*. Edinburgh: Edinburgh University Press.
- Duffy, S. W., and L. D. Sharples (1992). Alcohol and cancer risk. In *Alcohol and Illness*, J.C. Duffy, ed.. Edinburgh: Edinburgh University Press, pp. 1-18.
- Dutch Distillers Association (1992). *World Drink Trends*. Amsterdam: DDA.
- Edwards, G., P. Anderson, T. F. Babor and others (1994). *Alcohol Policy and the Public Good*. Oxford: Oxford University Press.
- English D. R., C. D. J. Holman, E. Milne and others (1995). *The Quantification of Drug-Caused Morbidity and Mortality in Australia, 1995 edition*. Canberra: Commonwealth Department of Human Services and Health.
- Ford, B. J. (1994). *Smokescreen: A guide to the personal risks and global effects of the cigarette habit*. Perth: Halcyon Press.
- Hammond, E. C. (1966). Smoking in relation to the death rates of one million men and women. In W. Haenszel (ed). *Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases. NCI Monograph 19*. Washington, D.C.: United States Department of Health, Education and Welfare, pp. 127-204.
- \_\_\_\_\_, and D. Horn (1958). Smoking and death rates - Report on forty-four months of follow-up on 187,783 men. I. Total mortality. *Journal of the American Medical Association*. vol. 166, no. 10, pp. 1159-1172.
- Hingson, R., and J. Howland (1993). Alcohol and non-traffic unintentional death and injuries. *Addiction*, vol. 88, pp. 877-884.
- Hirayama, I. (1967). Smoking in Relation to the Death Rates of 265,118 Men and Women in Japan. Tokyo: National Cancer Centre.
- Howland, J., and R. Hingson (1988). Alcohol as a risk factor for drownings. A review of the literature (1950-1985). *Accidents Analysis and Prevention*, vol. 20, pp. 19-25.
- International Agency for Research on Cancer (1988). *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Alcohol Drinking*, vol. 44. Lyon: IARC.
- Irwin, S.T., C. C. Patterson and W. H. Rutherford (1983). Association between alcohol consumption and adult pedestrians who sustain injuries in road traffic accidents. *British Medical Journal*, vol. 286, p. 522.
- Kahn, H. A. (1966). The Dorn study of smoking and mortality among U.S. veterans: Report on eight and one-half years of observation. In *Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases*, W. Haenszel, ed. NCI Monograph 19. Washington D.C.: National Cancer Institute.
- Klatsky, A. L., M. A. Armstrong and G. D. Friedman (1989). Alcohol and cardiovascular deaths. *Circulation*, vol. 80, pp. 611-614.
- \_\_\_\_\_, (1990). Risk of cardiovascular mortality in alcohol drinkers, ex-drinkers and non-drinkers. *American Journal of Cardiology*, vol. 66, pp. 1237-1242.
- Kortteinen, T. (1989). *State monopolies and alcohol prevention*. Social Research Institute of Alcohol Studies, Helsinki.
- Laugesen, M., and C. Meads (1991). Tobacco advertising restrictions, price, income and tobacco consumption in OECD countries, 1960-1986. *British Journal of Addiction*, vol. 86, pp. 1343-1354.

- Lehto, J. (1995). *Approaches to Alcohol control policy*. WHO Regional Publications, European Series, No. 60, Finland.
- Lewis, R. J., and S. P. Cooper (1989). Alcohol, other drugs and fatal work-related injuries. *Journal of Occupational Medicine*, vol. 31, pp. 23-27.
- Liu, Bo-Qui, Richard Peto, Zheng-Ming Chen and others (1998). Tobacco hazards in China: proportional mortality study of one million deaths. *British Medical Journal* (in press).
- Miettinen, O. S. (1974). Proportion of disease caused or prevented by a given exposure, trait or intervention. *American Journal of Epidemiology*, vol. 99, pp. 325-332.
- Murray, C. J. L., and A. D. Lopez (1996). Quantifying the burden of disease and injury attributable to ten major risk factors. In *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries and Risk Factors in 1990 and Projected to 2020*, C. J. L. Murray and A. D. Lopez, eds. Cambridge, MA: Harvard University Press, pp. 295-324.
- \_\_\_\_\_. (1997a). Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. *Lancet*, vol. 349, pp. 1436-1442.
- \_\_\_\_\_. (1997b). Alternative projections of mortality and disability by cause, 1990-2020. Global Burden of Disease Study. *Lancet*, vol. 349, pp. 1498-1504.
- Parish S., R. Collins, R. Peto and others (1994). Cigarette smoking, tar yields, and non-fatal myocardial infarction: 14,000 cases and 32,000 controls in the United Kingdom. *British Medical Journal*, vol. 311, pp. 471-477.
- Penttilä, A., S. Pöppönen and J. Pikkariainen (1979). Drunken driving with motorboats in Finland *Accident Analysis and Prevention*, vol. 11, pp. 237-239.
- Peto, R., and A. D. Lopez (1991). Worldwide mortality from current smoking patterns. In *Tobacco and Health: The Global War. Proceedings of the Seventh World Conference on Tobacco and Health*, B. Durston and K. Jamrozik, eds. Perth, Australia: Health Department of Western Australia, pp. 66-68.
- \_\_\_\_\_, J. Boreham, M. Thun and C. Heath (1994). *Mortality from Smoking in Developed Countries, 1950-2000*. Oxford: Oxford University Press.
- Renaud, S., M. H. Criqui, G. Farchi and J. Veenstra (1993). Alcohol drinking and coronary heart disease. In *Health Issues in Relation to Alcohol Consumption*, P.M. Verschuren ed. Brussels: ISLI Press, pp. 81-123.
- Rimm, E. B., and others (1991). Prospective study of alcohol consumption and risk of coronary disease in men. *Lancet*, vol. 338, pp. 464-468.
- Roizen, J. (1982). Estimating alcohol involvement in serious events. In National Institute on Alcohol Abuse and Alcoholism, *Alcohol Consumption and Related Problems*. Alcohol and Health Monograph No 1. Washington DC: NIAAA, pp 179-219.
- Romelsjö, A. (1995). Alcohol consumption and unintentional injury, suicide, violence, work performance and inter-generational effects. In *Alcohol and Public Policy: Evidence and Issues*, H. H. Holder and G. Edwards, eds. Oxford: Oxford University Press, pp 114-142.
- Shkolnikov, V. M., and A. Nemtsov (1997). The anti-alcohol campaign and variations in Russian mortality. In *Premature Death in the New Independent States*, J. L. Bobadilla, C. A. Costello and Faith Mitchell, eds. Washington DC: National Academy Press, pp 239-261.
- Simpura, Jussi (1995). Trends in alcohol consumption and drinking patterns: lessons from world-wide development. In *Alcohol and Public Policy: Evidence and Issues*, H. H. Holder and G. Edwards, eds. Oxford: Oxford University Press, pp 9-37.
- Smart, R. G. (1991). World trends in alcohol consumption. *World Health Forum*, vol. 12, pp. 99-103.
- Stallones, L., and J. Kraus (1993). The occurrence and epidemiologic features of alcohol-related occupational injuries. *Addiction*, vol 88, pp 945-952.
- Thun, M. S., R. Peto, A. D. Lopez (1997). Alcohol consumption and mortality in middle-aged and elderly US adults. *New England Journal of Medicine*, vol. 337, pp. 1705-14.
- Townsend, J. (1993). Policies to halve smoking deaths. *Addiction*, vol. 88, pp. 43-52.
- Tuyns A. J., and G. Pequinot (1984). Greater risk of ascitic cirrhosis in females in relation to alcohol consumption. *International Journal of Epidemiology*, vol. 14, pp. 53-57.
- United States Department of Health and Human Services (USDHHS) (1989). *Reducing the Health Consequences of Smoking: 25 years of Progress. A Report of the Surgeon General*. DHHS Publication No (CDC) 89-8411. Washington, D.C.: US Department of Health and Human Services, Centres for Disease Control, Office on Smoking and Health.
- \_\_\_\_\_. (1993). *Alcohol and Health: Eighth Special Report to the US Congress*. Washington D.C.: United States Department of Health and Human Services, National Institute on Alcohol Abuse and Alcoholism.
- Valkonen, T., and F. van Poppel (1997). The contribution of smoking to sex differences in life expectancy. *European Journal of Public Health*, vol. 7, pp. 302-310.
- Verschuren, P. M., ed. (1993). *Health Issues Related to Alcohol Consumption*. Brussels: International Life Sciences Press.
- Voas, R. (1993). Cross-national comparisons of crash data. *Addiction*, vol. 88, pp. 959-967.
- World Health Organization (1997a). *Tobacco or Health: a Global Status Report*. Geneva: WHO.
- \_\_\_\_\_. (1997b). *Smoking, Drinking and Drug Taking in the European Region*. Copenhagen: WHO Regional Office for Europe.

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## XVIII. ACCIDENTS AND OTHER INJURIES

*Dinesh Sethi\* and Anthony Zwi\**

### A. BACKGROUND

Intense media interest and public attention accompany the occasional massive crashes or massacres that take dozens of lives in a single event: far less attention is devoted to the hundreds of thousands more lives that are lost in the relentless and steady toll of injuries occurring daily. The public, and to a lesser extent the public health community, are either unaware or apparently unprovoked by the disproportionately large burden that injuries place on young people, making them a leading cause of premature loss of productive life, of high medical care costs and of significant degrees of disability.

Historically, injuries have been neglected as a public health problem for a variety of reasons. Communities, governments and donors have not perceived them to be amenable to public health interventions; the health and economic burden has been little documented; and there has been limited appreciation that injuries can be prevented through the organised efforts of society. The public's perception of "accidents", the most common term used to describe injurious events, highlights the perception of helplessness, chance, or the "acts of God" that are typically associated with injuries. Vested interests have opposed safety promotion where this has led to increased costs, especially where the associated benefits have been inadequately documented. There has been exceedingly little documentation, knowledge, and analysis of "best practice" in promoting policy action to facilitate the multifaceted and multisectoral interventions necessary for developing an appropriate response to injuries.

Recent analyses of the demographic and epidemiologic transitions, and influential studies such as the World Bank/WHO Global Burden

of Disease, have highlighted the substantial national and global burdens of injuries, and have drawn attention to the remarkably low-key responses to these problems (Murray and Lopez, 1996a, 1996b). Now, on the cusp of the millennium, rethinking global health policies and examining the scope for effective and more equitable health-promoting investments, injuries are becoming recognised as urgently in need of attention. This contribution seeks to review some of the background data that have contributed to appreciating the burden of injuries, and explores the nature and form of desirable policy action.

The epidemiologic, demographic, and socio-political transitions underway in many countries are likely to increase the frequency of injury events as a result of intensifying urbanisation, industrialisation, motorisation and the acquisition and diffusion of potentially harmful technologies. Infectious diseases are still an extremely important cause of death, and represent a set of concerns that needs continued attention if the poor are not to suffer further neglect (Gwatkin and Heuveline, 1997). However, they are being replaced by non-communicable disease and injuries are often the leading causes of premature mortality and morbidity in these countries (Omran, 1971).

Little attention, despite increasing concerns by national policy-makers and public health personnel, has been devoted to injuries in low and middle-income countries (LMICs), and in countries undergoing rapid economic or political transitions (Black, 1990). That which has been undertaken has been patchy, focused around concerned practitioners with limited development of systems of response and has tended to be greatly under-resourced. Only a handful of LMICs and countries in transition have systematically begun to develop mechanisms for the prevention of injuries and the management of their consequences.

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Where responses to injuries are underway, it is apparent that identifying the content of appropriate interventions is a small part of what is needed. Attention also needs to be devoted to the context in which injuries occur, to the processes through which a wide range of different stakeholders can be engaged in developing appropriate interventions, and to identifying the resources and systems necessary to assure success. There is extensive community-based experience for tackling various aspects of injuries: violence against women is increasingly on the global public health agenda (Heise and others, 1994) and is also addressed in a variety of ways across the globe at community level. Community responses to promote safety and security are scattered across many cities and towns world-wide and many non-governmental organisations have sought to address the problems of traffic, occupational and child safety. There are increasing calls to base not only health care, but health policy, on "evidence". In relation to injury control these challenges are immense, given the limitations of current knowledge and documented experience.

This paper touches upon the global scale of the burden injury, variations by region, and considers the availability of interventions. The term "injuries" represents a wide range of different types of problems, each with different sets of risk factors, risk situations and risk groups: we consider traffic injuries as our main example, but also illustrate our discussion with reference to other injuries for illustrative purposes. We do not seek to present a fully comprehensive discussion. Rather we highlight a range of problems and begin to explore elements of an appropriate policy response and how this may be supported by international organisations. We emphasise the importance of policy decisions being sensitive to local contexts. We also stress the need to develop interventions through consultative processes, to develop a shared sense of what needs to be done, and to base actions on evidence of effectiveness, efficiency and equity.

## B. MAGNITUDE OF THE INJURY PROBLEM

Injuries represent a significant public health burden, whether measured by mortality, mor-

bidity, disability, health services utilisation or a combination of these. Both intentional (homicide, suicide, war) and unintentional injuries (road traffic injuries, drowning, falls, burns and scalds, poisoning) contribute substantially to this burden. While helpful in raising their profile, presenting injuries as a single broad group does not necessarily assist us in determining how best to respond to them.

In the following section we briefly review aspects of this burden and draw attention to its magnitude and distribution between regions.

### 1. *Mortality*

There were an estimated 5 million deaths from injuries in 1990, accounting for 10 per cent of global mortality, 12.5 per cent of all male deaths and 7.4 per cent of those in females (Murray and Lopez, 1997a). The ratio of male to female deaths from injuries was 2:1. In ages 15-44, injuries caused 15.1 per cent of the deaths among women (suicide 7 per cent, war 4.4 per cent, road traffic accidents 3.7 per cent). Males are generally much more affected by injuries than females; two exceptions in some countries are suicide and attempted suicide, as well as various forms of intimate partner abuse.

### *Vital registration*

There were an estimated 50 million deaths world-wide in 1990; of these 39 million were in developing countries. It is estimated that only 30-35 per cent of all deaths are captured by vital registration (Murray, 1994). Policy makers, however, need reasonably accurate estimates in order to target resources to the most important causes of death and morbidity, and to maximise health gain for a given investment. Of the eight main regions considered by the Global Burden of Disease (GBD) Study, only the Established Market Economies and Former Socialist Economies have complete vital registration (Murray and Lopez, 1996a, table 75). In China surveillance of a panel of 10 million persons from rural and urban areas provides extremely useful data. India does not have a reliable vital registration system and urban areas have more complete death certification than rural areas, where verbal autopsies form the basis of a survey of causes of death. In sub-Saharan

Africa (SSA) registration is more complete in certain countries, but even in wealthier countries such as South Africa data collection is far worse in certain areas, notably the historically neglected and disempowered rural and bantustans (table 75). Other countries have partial registration.

TABLE 75. THE COMPLETENESS OF VITAL REGISTRATION BY REGION IN 1990

Region	Under 5 years	5 years and over	Total
Established Market Economies (EME)	99	99	99
Former Soviet Economies (FSE)	99	99	99
India	sample registration and surveys		
China	sample registration		
Other Asia and Islands (OAI)	2	14	10
Sub-Saharan Africa (SST)	<1	2	1
Latin America and the Caribbean (LAC)	28	47	43
Middle Eastern crescent	12	27	22

Source: Murray, C. J. L. and A. D. Lopez (1996). *Global Health Statistics: A Compendium of Incidence, Prevalence, and Mortality Estimates for over 200 Conditions. Vol. II.* Cambridge: Harvard University Press.

Best estimates of mortality and incidence have been modelled using available data, population samples and the results of epidemiological surveys. The full methodology for the GBD Study has been discussed elsewhere (Murray and Lopez 1996b) and despite shortcomings still provide the most comprehensive data allowing for inter-region comparisons between males and females at different ages from different causes of morbidity and mortality. Data from country-level analyses of burden of disease are beginning to emerge (see, for example, Lozano and others, 1995; Bobadilla and Cowley, 1995) and once again highlight the under-researched and neglected response to injuries.

## 2. Regional variations

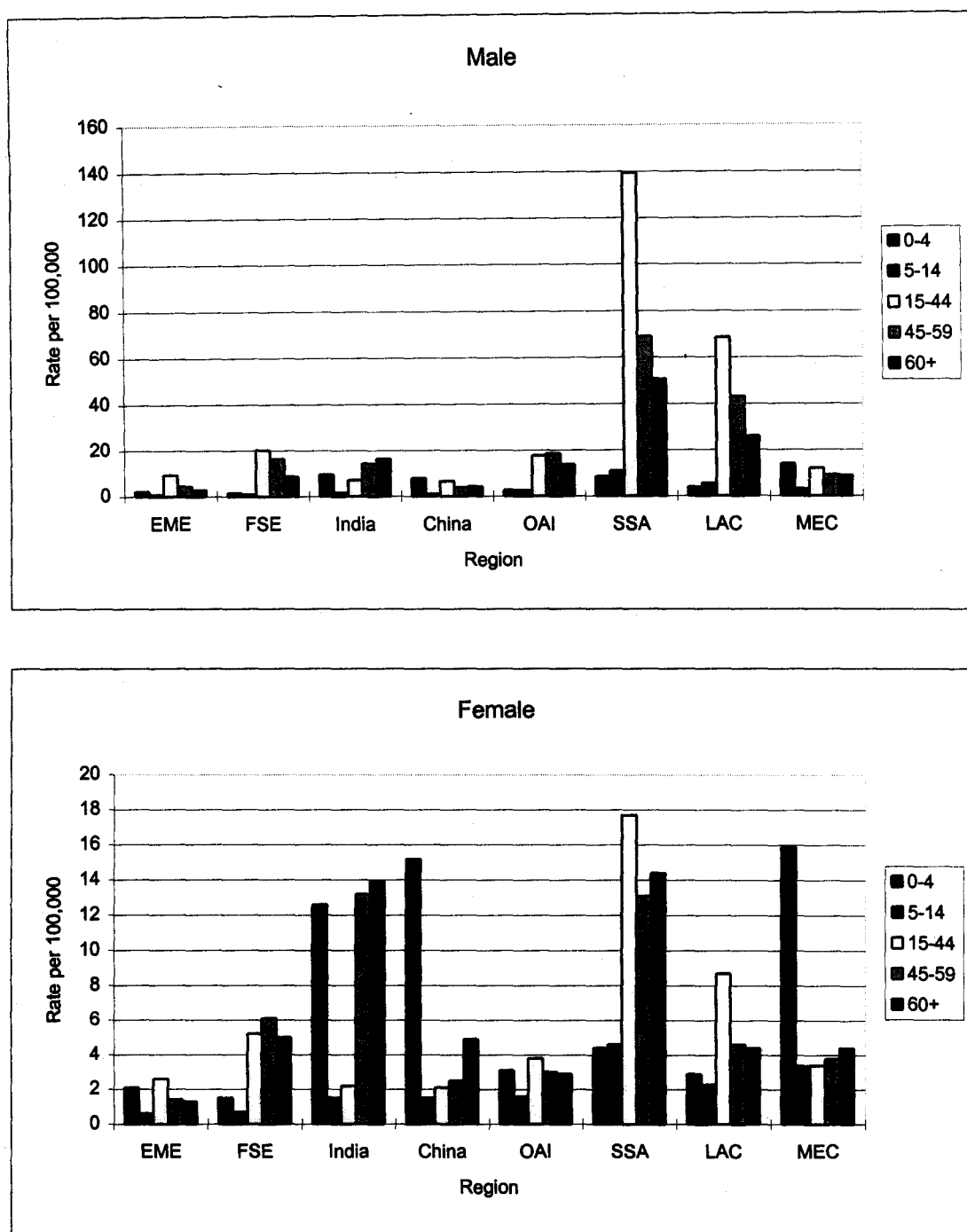
Marked regional variations are present in the eight regions used for analysing the Global Burden of Disease: Established Market Economies (EME), Former Socialist Economies (FSE), India, China, Other Asia and Islands (OAI), Sub-Saharan Africa (SSA), Latin America and the Caribbean (LAC), and the Middle Eastern Crescent (MEC). In the EME homicides cause 6 per cent of all deaths compared to 12-13 per cent in LAC and SSA. In these latter two regions homicides cause one in six of all male deaths. Fifty-six per cent of all female suicides were in China where there are high rates in the rural areas. Forty percent of the male homicides were in SSA and 20 per cent were in LAC. Figure 116 shows the mortality rates and highlights regional differences and differences in the patterns of deaths from violence. Men aged 15-44 years, especially in SSA are at risk of violent death; girls under the age of five years have much poorer life chances in India, China and the MEC.

Identifying particularly high mortality in a region, age and sex group, should help initiate a research agenda. Tackling all the elements of an appropriate research response (see box 1) calls for more systematic study, from both a qualitative and quantitative perspective, employing both social science and epidemiologic approaches. Anthropological, ethnographic, sociological, psychological and social policy analyses will contribute greatly to understanding the unique features of injury mortality in a particular area: the explanation for high suicide rates in Chinese women, burns in Indian women, homicide in African men, firearm-related deaths in the United States, high suicide levels among elderly men in a rural South African area (Tollman, Kahn and Garenne, in press).

A comparative study using government statistics for the population of the United States of America and a segment of China's population covering 10 million people, showed a greater mortality from drowning, poisoning, falls, and suicide in China whereas road traffic accidents (RTAs), fires, and homicides were more frequent in the United States (Li and Baker, 1991).



Figure 116. Mortality rates from violence by age, gender and region in 1990



Source: C. J. L. Murray and Alan D. Lopez, *Global health statistics: a compendium of incidence, prevalence, and mortality estimates for over 200 conditions. Volume II* (Cambridge, Harvard University Press, 1996a).

**Box 1. Generic list of priority injury questions**

	<b>Health burden</b>	<b>Economic costs</b>	<b>Policy</b>
<b>Descriptive</b>	<b>A</b> Who is affected? By what types of injuries? How large is the problem? When and how do these injuries occur? What are the trends in injury occurrence? Do injuries occur more frequently in particular age, gender of social class groups?	<b>B</b> What are the direct and indirect costs of injuries? What are the costs of resultant disabilities/lost production and loss of life? Who incurs these costs? What do people pay to avoid these injuries? To treat them?	<b>C</b> Who are the stakeholders with a concern for injuries? What are their interests, commitments and priorities? What is the context within which interventions need to be considered?
<b>Analytic</b>	<b>D</b> Why do injuries occur? What are the risk factors and risk situations that predispose to injury occurrences? How effective and efficacious are available interventions? What is the link between poverty (relative and absolute) and the occurrence of injuries?	<b>E</b> What is the cost-effectiveness of available interventions? What priority should be given to primary, secondary and tertiary interventions? What are the opportunity costs of introducing interventions? What is the link between the occurrence of injuries and disability and subsequent declines in household livelihoods and sustainability?	<b>F</b> What resources should be allocated to injury control? How should it rank in relation to other health conditions? What is the role of the state and the private sector?
<b>Interventions</b>	<b>G</b> Towards evidence-based policies: which policies are known to be effective? In what sorts of contexts are they applicable? How feasible are these policies in the context at hand? How can their implementation be increased? What incentives can be offered to the range of stakeholders to increase their support for desirable interventions? What are the equity implications of the proposed policies? How sustainable are they?		

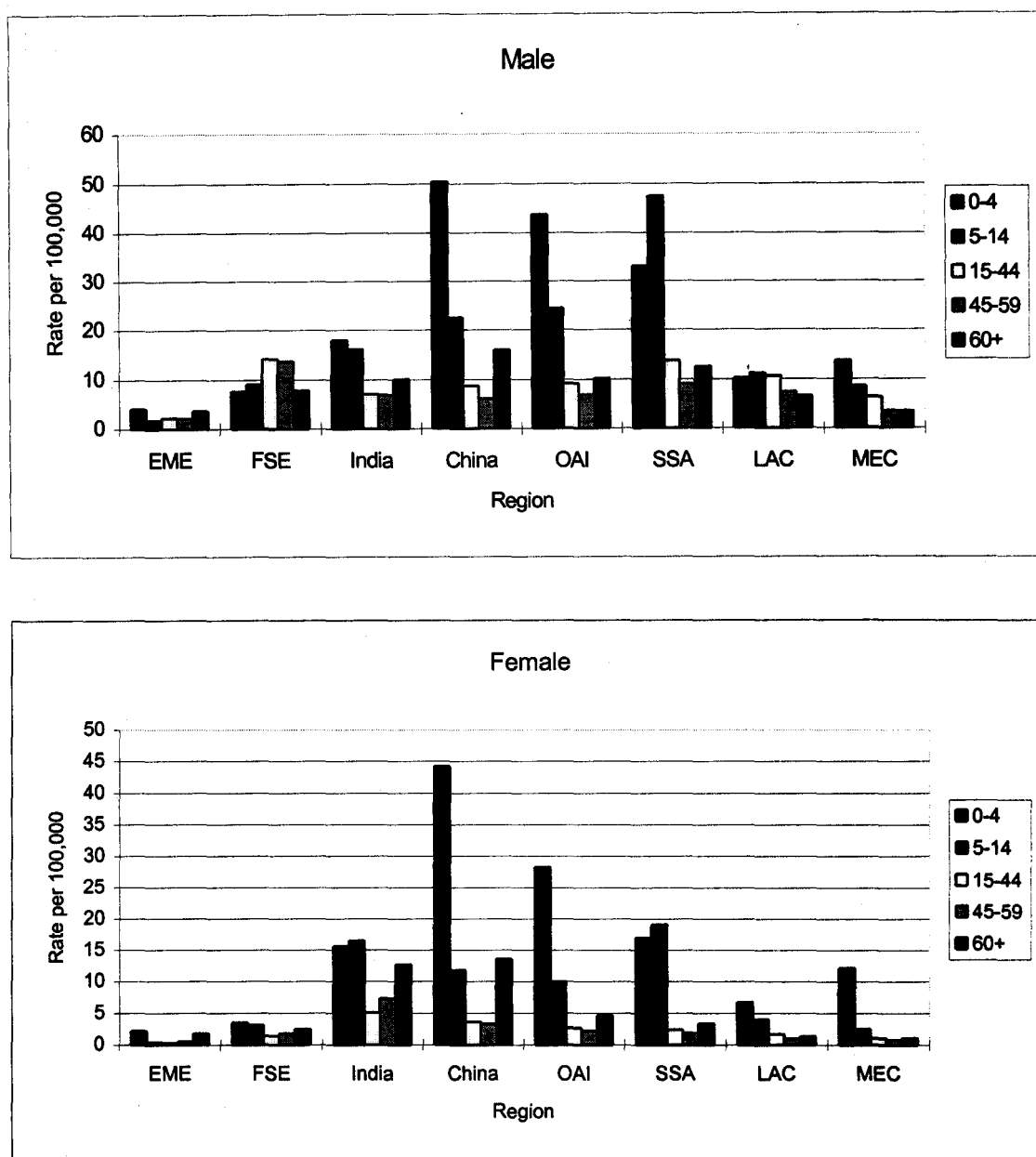
  

A	Know a great deal in some countries; situation-specific analyses required.
B	Know something; gaps in all settings, some contexts more than others
C	Little known outside of industrialised country contexts
D	Little known outside of limited number of industrialised country contexts; especially important to appreciate in countries undergoing rapid epidemiologic, demographic, political and economic transitions
E	Almost no information from low or middle income, or transitional countries.
F	Almost no knowledge of current practice or best practice
G	Key objective if injury control is to be promoted

While the limitations of cross-national comparisons are numerous, they serve to highlight the very different issues that require local attention. Drowning, for example, is a major cause of injury deaths (figure 117) in many countries, especially in China, India and SSA. However, it is only in the wealthier countries of the North, where the focus has been on leisure-related drownings, that any attention at all has been devoted to this problem (Ellis and Trent, 1995).

In Zimbabwe drowning was, in 1988, the fourth most important cause of injury deaths (Zwi and others, 1993) and has emerged *en passant* in community-based studies of mortality in communities elsewhere such as in Ghana. There is limited knowledge of the circumstances of these deaths, community perceptions of them, first aid responses, nor of what interventions might be effective and at what cost. An agenda for action (or at least exploration) lies before us; one of

Figure 117. Mortality rates from drownings by age, gender and region in 1990



Source: C. J. L. Murray and Alan D. Lopez, *Global health statistics: a compendium of incidence, prevalence, and mortality estimates for over 200 conditions. Volume II* (Cambridge, Harvard University Press, 1996a).

many in which even our most basic knowledge (cells A and B, box 1) is patchy and incomplete.

Table 76 shows the top 30 leading causes of death world-wide. Amongst these RTA, self-inflicted injuries, violence, drowning and war injuries rank among the top 25. Many of these, particularly RTAs, are predicted to increase in the coming decades. For one thing, they are

strongly related to increases in gross national product (GNP) per capita. In general, it isn't until GNP per capita reaches US\$ 5000 that RTA control efforts are effectively introduced (Soderlund and Zwi, 1995).

Injuries are a leading cause of death in young people and are responsible for about half the deaths in the 10-24 year age group, (Berger and

TABLE 76. THIRTY LEADING CAUSES OF DEATH WORLDWIDE IN 1990

Rank	Cause of deaths	Number of deaths (x1000)	Rank	Cause of deaths	Number of deaths (x1000)
1	Ischaemic heart disease	6 260	16	Diabetes mellitus	571
2	Cerebrovascular disease	4 381	17	Violence	562
3	Lower respiratory infections	4 299	18	Tetanus	542
4	Diarrhoeal diseases	2 946	19	Nephritis and nephrosis	536
5	Perinatal disorders	2 443	20	Drowning	502
6	Chronic obstructive airways disease	2 211	21	War injuries	502
7	Tuberculosis (not HIV)	1 960	22	Liver cancer	501
8	Measles	1 058	23	Inflammatory heart disease	495
			24	Colon and rectum cancers	472
10	Trachea, bronchus and lung cancers	945	25	Protein-energy malnutrition	372
11	Malaria	856	26	Oesophagus cancer	358
			27	Pertussis	347
13	Cirrhosis of the liver	779	28	Rheumatic heart disease	340
14	Stomach cancer	752	29	Breast disease	322
15	Congenital abnormalities	589	30	HIV	312

Source: C. J. L. Murray and A. D. Lopez, "Mortality by cause for the eight regions of the world: Global Burden of Disease Study", *Lancet*, Vol. 349, pp. 1269-1276 (1997a).

Mohan, 1996). For example, in Papua New Guinea, injuries were the leading cause of death in the 15-44 age group (Graitcer, 1992). Blum (1991) has suggested that the morbidity and mortality trends for young people in LMICs are paralleling those of industrialised countries. Intense urban migration, unemployment, disruption of traditional social structures, availability of weapons, access to alcohol, and increasing inequalities all contribute to this emerging picture. Other reports also highlight this problem (Bungdiwala and Anzola-Perez, 1990).

### 3. Morbidity and Disability Adjusted Life Years (DALYs)

Mortality data only measure the extreme end of the spectrum of the burden of disease. They do not take into account the morbidity as evidenced by people who attend emergency rooms, are admitted for treatment of their injuries or who remain with a residual disability. In 1990, 90 per cent of the world-wide burden of disease

occurred in LMICs where only 10 per cent of the health expenditure occurred (Murray and Lopez, 1997b). Injuries as a whole caused 10 per cent of the mortality world-wide but 15 per cent of the DALYs lost (table 77).

### Regional variations in DALYs lost

DALYs lost as a result of all injuries ranged from 12 per cent in EME to 19 per cent in the FSE. In SSA injuries were responsible for more DALYs lost than any other single health condition. Unintentional injuries caused more than 10 per cent of the DALYs lost in all areas except for the MEC, EME and SSA (figure 118). Intentional injuries caused the highest disability in SSA, LAC, FSE, China, and MEC. Seven causes of injuries, RTAs, falls, war injuries, self-inflicted injuries, violence, drownings and burns were among the top 30 conditions contributing to the global burden of disease. It is estimated that injuries account for 10 to 30 per cent of hospital admissions and about 78 million disabilities per year (Berger and Mohan, 1996).

TABLE 77. PERCENTAGE DISTRIBUTION OF DALYs BY CAUSE AND REGION, 1990

Region	EME	FSE	India	China	OAI	SSA	LAC	MEC	World
Group 1: Infectious disease, perinatal and maternal disorders									
Total	7.1	8.8	56.4	24.2	44.7	65.9	35.3	47.7	43.9
Group 2: Non-communicable disease									
Total	81.0	72.6	29.0	58.2	40.9	18.8	48.2	39.3	40.9
Group 3: All injuries									
Total	11.9	18.7	14.6	17.6	14.4	15.4	16.4	13	15.1
Unintentional injuries	8.7	12.9	13.0	12.9	12.1	9.3	11.9	6.8	11.0
Intentional injuries	3.2	5.8	1.5	4.7	2.3	6.0	4.5	6.2	4.1

Source: C. J. L. Murray and A. D. Lopez, "Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study," *Lancet*, vol. 349, pp. 1436-1442 (1997).

In Indonesia and Egypt injuries are the leading cause of hospitalisation (Graitcer, 1992). Of interest, the rural population in China have a pattern of disease similar to LAC whereas urban populations have a pattern similar to higher income countries (Lawson, 1994).

Men have a lower life expectancy at birth than women, and also have a higher percentage of their life-span lived with disability than women. Much of this disability is attributable to alcohol and to other exposures that lead to injuries. Alcohol was a major cause of death and disability due to injuries especially in the LAC and SSA areas (Murray and Lopez, 1997b). Russian mortality rates from violence, injuries and alcohol poisoning have increased sharply since market liberalisation after the end of the Cold War and have coincided with increases in alcohol consumption (Leon and others, 1997). Alcohol may also have played some part in the massive increases in RTA-deaths occurring in the FSE around the end of the Cold War. Rapid political and economic transition may pose risks to conditions whose control requires substantial degrees of co-ordinated multisectoral efforts. Unintentional injuries are the most common cause of death in Russian adults under the age of 45 years. The relationship between alcohol and deaths from unintentional injuries and suicides has been described by many studies (Andreasson and others, 1988). Alcohol may also play some part in the sharp increase in crime, homicides and

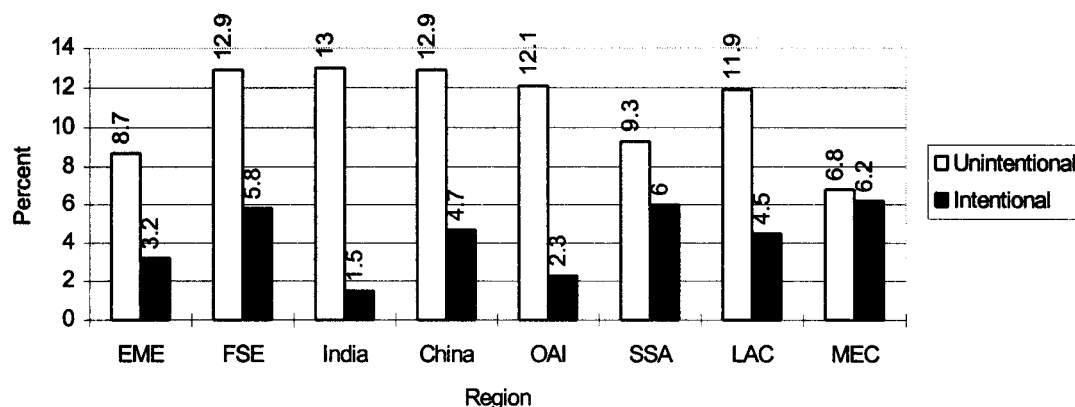
unintentional injuries since the collapse of the Soviet Union.

With particular reference to persons aged 15-44, injuries in the developing regions led to 55 million DALYs lost among males in 1990, a third of all DALYs lost from all causes in this age group. Figures 119-126 show the percentage of DALYs lost by males and females in this age range from the main groups and injuries for the various regions. Group I causes are the infectious disease, perinatal, maternal and nutritional conditions and Group II are the non-communicable diseases. For males the largest proportion of DALYs lost from all injuries was in SSA (46 per cent), followed by the FSE (40 per cent), MEC (37 per cent) and the smallest proportion was in EME (28 per cent) and India (25 per cent). In SSA violence (12 per cent) and wars (11 per cent) were the largest causes.

Violence was a leading cause of DALYs lost in LAC (11 per cent) followed by RTAs (9 per cent). In the FSE and EME, RTAs were the leading cause (11 per cent each) followed by self-inflicted injuries (6 per cent and 5 per cent respectively). Wars were the leading cause (14 per cent) in the MEC followed by RTAs (7 per cent).

In contrast the proportions lost due to injuries are smaller among females, partly because they are less at risk but also because in LMICs they experience a larger burden of disease from maternal conditions and other Group I condi-

Figure 118. Percentage of DALYs lost due to unintentional and intentional injuries by region in 1990



Source: C. J. L. Murray and Alan D. Lopez, *Global burden of disease. A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Volume I* (Cambridge, Harvard University Press, 1996b).

tions. Regional variations exist and DALYs lost due to injuries are highest in China (22 per cent), MEC (16 per cent) and India (15 per cent) and lowest in LAC (11 per cent), OAI (11 per cent) and EME (10 per cent). Of concern are the high proportion of DALYs lost due to self-inflicted injuries in China (11 per cent) and the large burden attributable to burns in India (5 per cent). In SSA, MEC and FSE, wars had a high toll (8 per cent, 9 per cent and 3 per cent respectively). RTAs are the leading cause in EME (5 per cent) and in FSE (3 per cent).

#### *Projections of disability*

The Global Burden of Disease Study estimated that deaths from injuries would increase from 5.1 million in 1990 to 8.4 million in the year 2020 (Murray and Lopez, 1997c). RTAs (in 1990 ranking ninth) and self-inflicted injuries (twelfth) would rank among the top 10 causes of death in 2020 (sixth and tenth, respectively) DALYs from both unintentional and intentional injuries would also increase from 15.2 per cent of the global burden to 20.1 per cent. Injuries are prominent amongst the top 15 causes of DALYs lost as projected for 2020: RTAs (rank third), war injuries (eight), violence (twelfth), and self-inflicted injuries (fourteenth). The largest increase in DALYs from injuries is expected in SSA.

The demographic transition partly explains these changes. The population of those at particular risk such as children and adolescents under 15 will increase by 25 per cent by 2020 but the biggest change will be seen in adults aged 45-69 (140 per cent). Although caution is advised when these projections are used because of their large confidence limits, their usefulness to public health planners cannot be underestimated.

Certain forms of injuries such as traumatic brain injuries, spinal injuries, major fractures and amputations may not only decrease life expectancy but will also pose an immense burden in the form of permanent disability, substantial handicap and social and economic burden on survivors, their families, and the health care services. Serious burns will pose similar concerns, with high mortality an additional risk. Improvements to tertiary prevention efforts will be especially relevant in relation to these problems.

#### *Hospital based studies*

Hospital based studies do not inform us of the population-burden of injuries. However, they have some value in estimating the main health care costs associated with injury treatment and rehabilitation. In the West Indies 20 per cent of

surgical admissions were due to injuries (Crandon and others, 1994). In Ghana burns and RTAs are major causes of mortality and disability from injuries in patients appearing in hospital (Mock and others, 1995). A two week prospective study of national trauma referral centres was undertaken in Trinidad and Tobago. It showed that those under 20 constituted 42 per cent of attendees. Of these, 63 per cent were male and 17 per cent were under the age of 4 years. Among the leading causes, falls accounted for 44 per cent, intentional injuries for 14 per cent, and RTAs for 7 per cent. As mortality in this age group is relatively low, hospital utilisation proved a more comprehensive source of information (Kirsch and others, 1996). In the same country, a case note review at a teaching hospital demonstrated that the commonest causes of admission for severe injury were blunt injury and penetrating injury highlighting the burden of violence on health services. Mortality improved after the introduction of advanced trauma life support training for physicians (Ali and others, 1993).

One way of improving data collection may be through the use of capture-recapture techniques as was done for head injuries and spinal injuries

in Taiwan (Chiu and others, 1993). This technique will be most appropriate where a number of different sources of care or contact with seriously injured patients is present, thus enabling estimation of the magnitude of people affected.

### C. SPECIFIC INJURY TYPES

#### 1. Road traffic "accidents"

##### *High income countries*

Table 78 shows that in EME countries RTA fatalities have steadily declined to half their 1970 levels. These are expressed as the mean risk of inhabitants being involved in a fatal accident on the basis of the home population (killed per 100000 inhabitants) and the mean risk resulting from road usage and mobility on the basis of vehicle kilometres (killed per 1 billion vehicle km). Even within the European Union there are inter-country variations. In 1992 this ranged from the mean risk rates of 7.5 per 100,000 inhabitants in Great Britain to 33 per 100,000 in Portugal. In contrast there was a marked increase in the FSE such as Hungary

TABLE 78. MEAN NUMBER OF FATALITIES PER 100,000 POPULATION AND DEATH RATES PER 1 BILLION VEHICLE KILOMETRES FOR 1970 AND 1993 FOR SELECTED EME AND FSE COUNTRIES

Country	Killed per 100,000 inhabitants	Killed per 100,000 inhabitants	Killed per 1 billion km	Killed per 1 billion km
Year	1970	1993	1970	1993
Austria	34.5	16.2	109.2	20.9
France	32.5	16.6	90.4	20.3
Belgium	31.8	16.5	-	-
West Germany	31.4	10.6	82	14.1
Switzerland	26.6	10.5	55.8	12.3
Netherlands	24.5 (for 1972)	8.2	54.4	12
Denmark	-	-	50.5	14.2
Ireland	20.5	12.6	-	-
Sweden	16.3	7.3	-	-
Hungary	15.8	16.3	-	-
East Germany	14.4	19.3	55.8 (for 1980)	37.5
United States of America	-	-	29.6	10.9
Great Britain	13.9	6.7	37.4	9.3

Source: E. Bruhning, "Injuries and deaths on the roads—an international perspective", in *Health on the Crossroads: transport policy and urban health*, T. Fletcher and A. J. McMichael, eds. (Chichester, John Wiley and Sons, pp. 109-124, 1997).

and East Germany; this was likely to have been due to increased car ownership and mobility in these countries following the politico-economic reforms and the unification of Germany. They also suggest that rapid political transition may adversely affect the systems necessary to maintain traffic controls and promote traffic safety, although they rapidly recover after greater political stability is established. Data from Southern Europe is less comprehensive although recent figures for Portugal and Spain suggest that road fatality is among the highest in Europe (Bruhning, 1997). Figure 121 shows mortality rates from RTAs in the world's main regions in 1990. RTAs are the leading cause of DALYs lost from injuries in males aged 15-44 in the EME and FCE regions (figures 119 and 120).

Injury priorities vary by nation. Great Britain has the highest percentage of traffic-related fatalities occurring in pedestrians; West Germany has the highest percentage of passenger car fatalities. Among the latter, the 18-24 year old age group is particularly at risk, especially men, and this is true for all countries. There is emerging evidence that the risk of crashes and collisions declines with the experience of the driver and if driving commenced at an older age (Bly, 1996). The type of vehicle and type of roads also influence fatalities. Many of these RTAs also involve intoxication with alcohol an important cause of road traffic fatalities in both industrialised and low income settings (Odero, and others, 1997). Pedestrians, especially the old and children, and bicyclists are particularly vulnerable on the road and suffer higher fatalities.

The decline in RTA fatalities in EME countries is due to the development of a comprehensive policy response comprising legislation (speed control, alcohol and driving, seatbelt use, child restraints in vehicles, helmet use in motorcycle riders), improved road design, traffic calming measures (especially in residential areas), health education, better car design and improved emergency medical systems. The greatest successes appear to result from successful multi-sectoral efforts: countries such as New Zealand have developed effective responses through the establishment of compre-

hensive road safety plans and strategies (Officials Committee on Road Safety, 1991). Future research initiatives need to address the problem areas, such as the high proportion of deaths occurring in younger males.

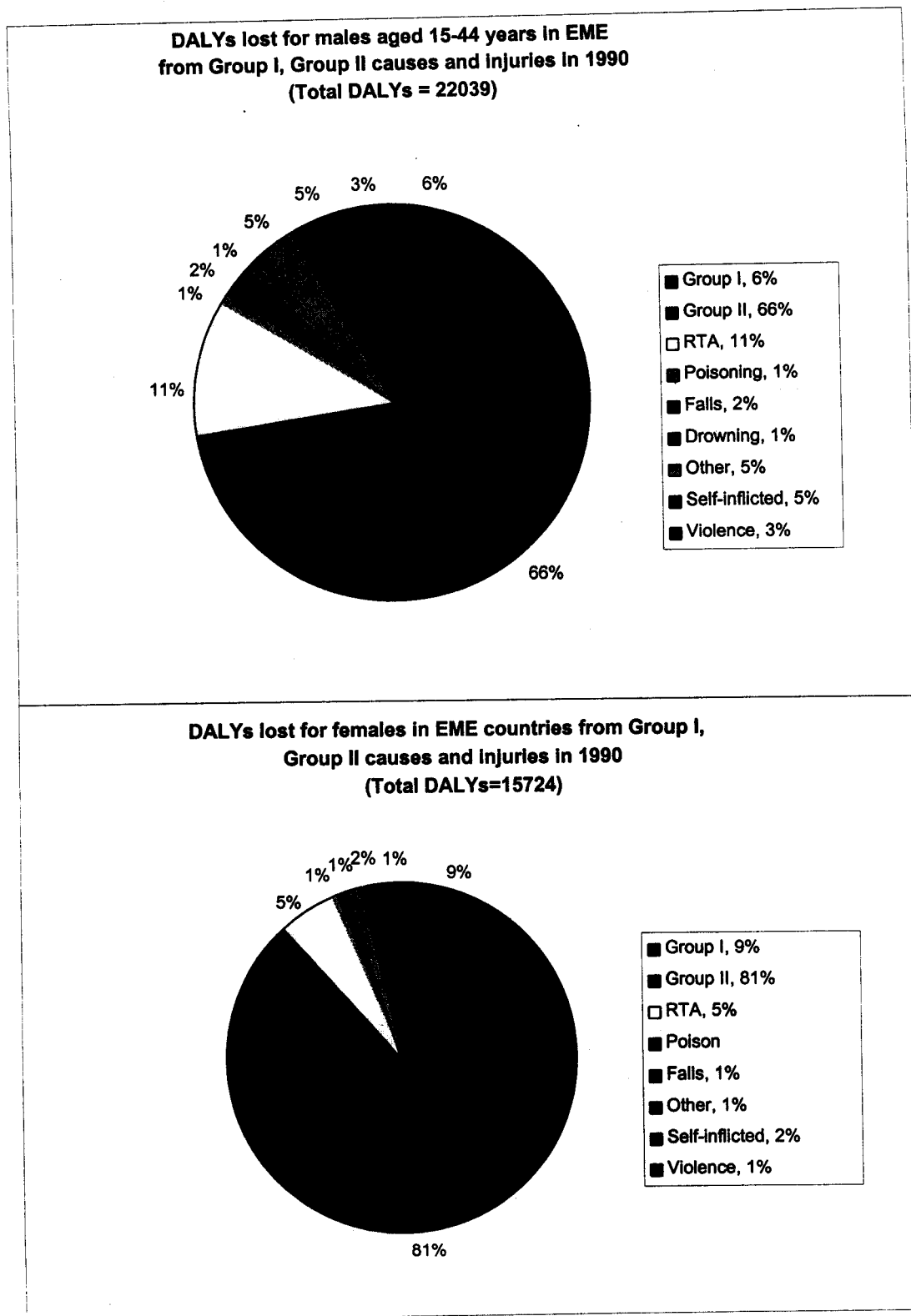
#### *Low and Middle Income Countries (LMICs)*

African and Asian countries with relatively low vehicle densities are experiencing higher RTA fatality rates in proportion to population size than in Europe or the United States (Odero and others, 1997; see also figure 121). It has been estimated that 70 per cent of the 600,000 fatalities per annum and 15 million injuries occur in LMICs (World Bank, 1990). A more recent study estimated that there were 856,000 road deaths annually and that 76 per cent occurred in LMICs (World Bank, 1993). RTAs are a leading cause of death in adolescents and young adults (Smith and Barss, 1991), posing an immense economic burden given this loss during their most productive years. Eighty percent of the fatalities were male. Between 1968 and 1985, RTA fatalities increased by 150 per cent (fatalities per 10,000 registered vehicles) in Asian countries and by 300 per cent in African countries. This is in contrast to European countries where there was a decline of 25 per cent over the same period (Jacobs, 1996). RTAs impose a considerable strain on the already scarce financial resources of these countries, and were estimated to cost one to two percent of a country's gross national product. (Jacobs and Fouracre, 1977; Zwi and others, 1996). In terms of 1996 figures this would amount to \$25 billion (Jacobs 1996). The proportion of DALYs lost from RTAs in males aged 15-44 years are particularly high in LAC (9 per cent), MEC, India and OAI (7 per cent each) (figures 122, 124, 126 and 127).

Road fatalities in developing countries are characterised by the predominance of accidents involving pedestrians, motorcycles, bicycles, buses, trucks and other public service vehicles (Downing, 1991). Bicycle and motorcycle fatalities constitute the highest proportion of RTA fatalities in India (Mohan and Bawa, 1985). The high levels of pedestrian mortality demand attention to protecting both vehicle occupants and other road users.



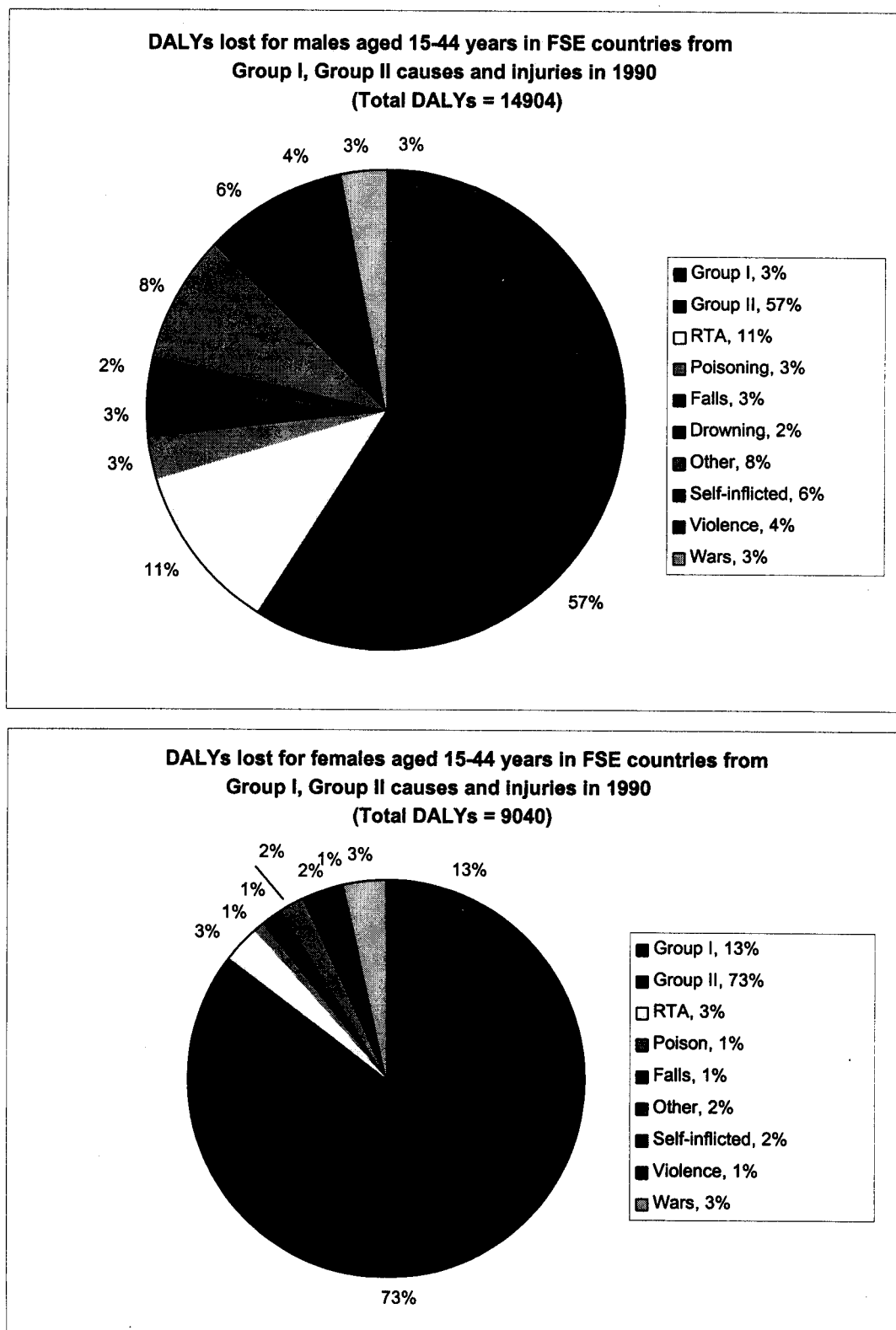
**Figure 119. Percentage of DALYs lost in EME by gender for ages 15-44 in 1990**



Source: C. J. L. Murray and Alan D. Lopez, *Global burden of disease. A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Volume I* (Cambridge, Harvard University Press, 1996b).

Total DALYs in thousands. Injuries constituting less than 1 per cent not shown.

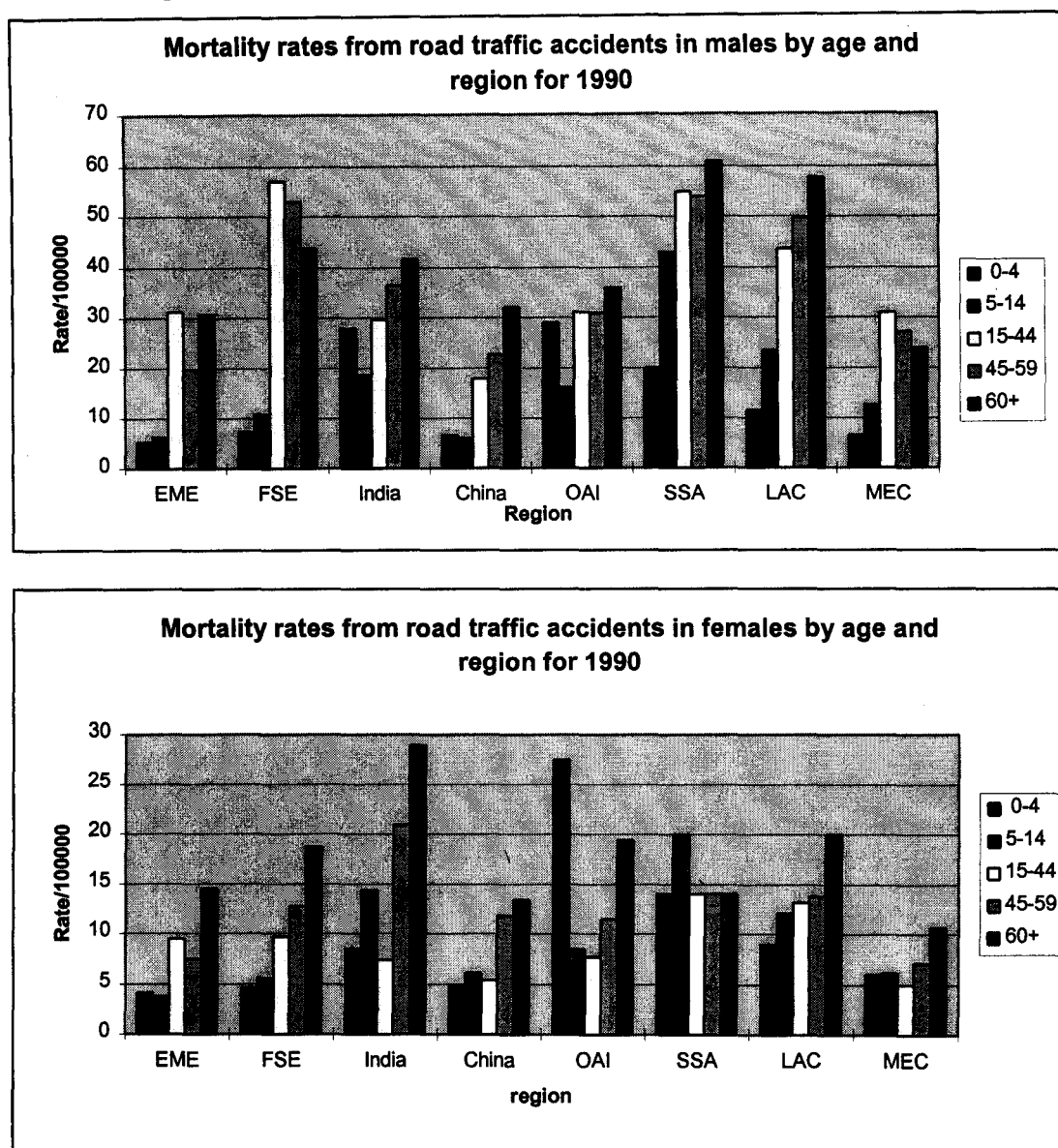
Figure 120. Percentage of DALYs lost in FSE by gender for ages 15-44 in 1990



Source: C. J. L. Murray and Alan D. Lopez, *Global burden of disease. A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Volume I* (Cambridge, Harvard University Press, 1996b).

Total DALYs in thousands. Injuries constituting less than 1 per cent not shown.

Figure 121. Mortality rates for road traffic injuries by sex, age and region, 1990



Source: C. J. L. Murray and Alan D. Lopez, *Global health statistics: a compendium of incidence, prevalence, and mortality estimates for over 200 conditions. Volume II* (Cambridge, Harvard University Press, 1996a).

Contributory factors to accidents are commonly assigned to road user error (95 per cent of accidents in Great Britain versus 64-94 per cent in LMICs), vehicle defects (8 per cent versus 2-17 per cent), and adverse road conditions (28 per cent versus 1-20 per cent). Traffic violations such as speeding have been mentioned in some studies to be as high as 60 per cent in Côte d'Ivoire and 10 per cent in Jordan. It has been suggested that poor road conditions in LMICs have a greater role than these figures suggest. Driver discipline, training and alcohol intoxication are also likely to be contributory.

For example, in a study in Zimbabwe 56 per cent of driver and 72 per cent of pedestrian fatalities had some level of alcohol in their blood compared to equivalent figures of 30 per cent for Great Britain (Sandwith, 1979). Routine alcohol testing in RTAs varies between countries (Odero and others, 1997); recent studies have suggested that the use of simple technologies such as alcohol breathalysers, will provide a reliable means with which to estimate the linkage between alcohol and driving (Odero and Zwi, in press). These technologies can also be more readily used to promote

enforcement of appropriate drink-driving legislation.

Factors contributing to the unsafe road environment, in addition to those raised above, include the large ratio of pedestrians to vehicles (Berger and Mohan, 1996), the mixture of pedestrians, non-motorised and motorised transport on the roads, inadequate and overloaded public transport networks, inadequate illumination and signposting, poor maintenance and development of roads, and the presence of roadside hazards such as trees, ditches and steep edges. Improvements to vehicle design, occupant protection and vehicle maintenance have also contributed to reducing traffic fatality levels.

In Zimbabwe between 1985 and 1994, there was a 55 per cent increase in traffic related crashes, resulting in an increase of injuries by 59 per cent and of fatalities by 42 per cent (Zwi, 1996). Fatality rates for various countries are shown in table 79. Another factor contributing to the high fatality rates following traffic crashes and collisions is the lack of high quality emergency medical services and pre-hospital care, to collect, resuscitate and treat the injured. Improving tertiary prevention through the development of effective emergency medical systems, trained pre-hospital care personnel, and the establishment of good quality care within accident and emergency services and

trauma units, will contribute to improving outcomes in terms of reduced mortality and disability. The use of injury rating scales, such as the Abbreviated Injury Scale, would enable the comparison of outcomes in order to improve the quality of health care (Baker and others, 1992).

In order to reduce road fatalities, a necessary starting point must be an appropriate information gathering and surveillance system (Forjuoh and Gyebi-Ofosu, 1993). Simple computerised systems developed by the Transport Research Laboratory, for example, would greatly assist the standardisation and completeness of such data collection (Transport and Road Research Committee, 1991).

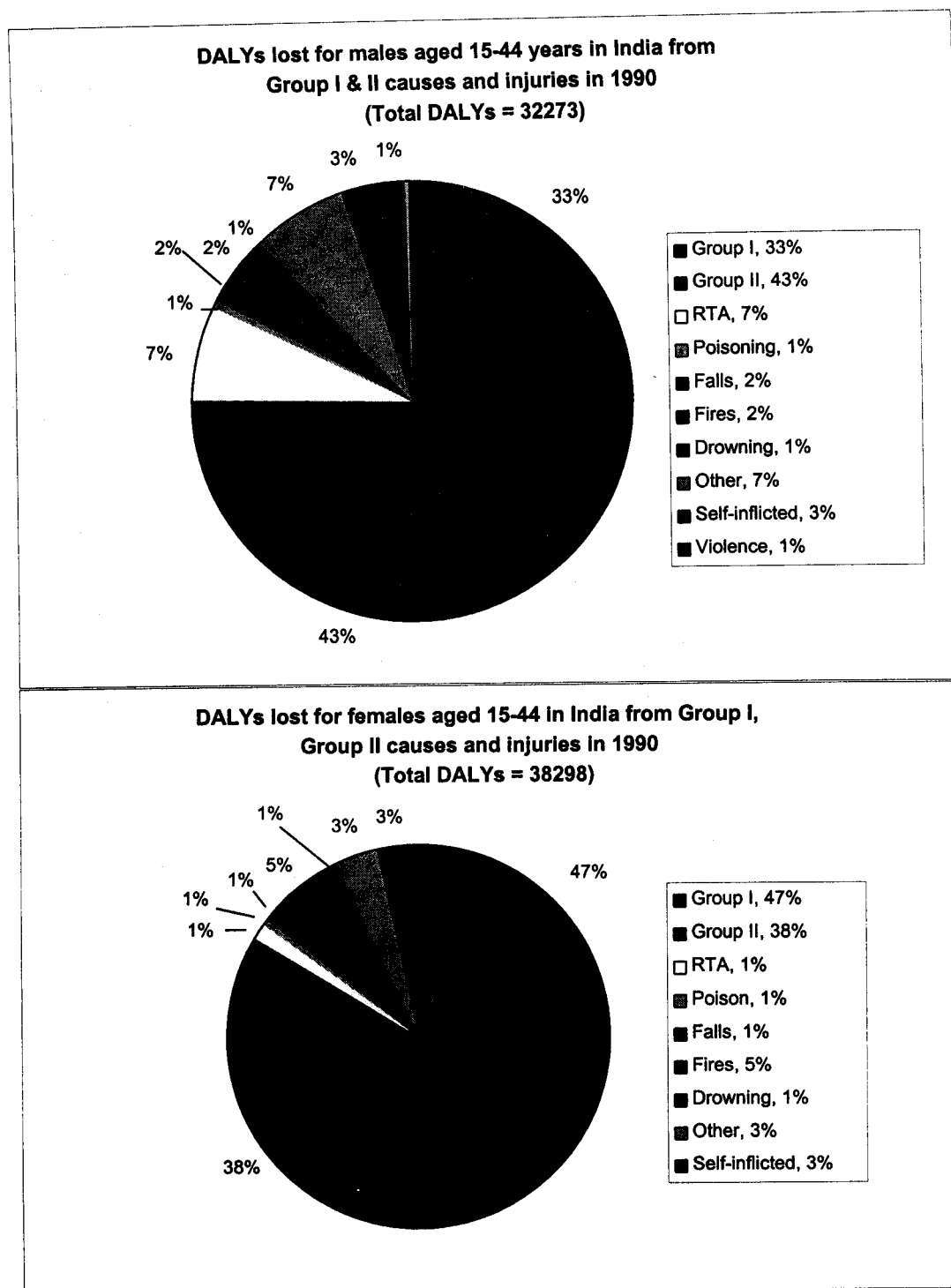
The establishment of an effective national road safety body provides an effective avenue for formulating and implementing policy with an appropriate balance between efforts to improve road design, vehicle safety, and driver and traffic police training. Attempts to promote such activity in Zimbabwe, for example, have been complicated but have nevertheless begun to involve the range of actors with a stake in addressing this problem. In view of the high level of pedestrian fatalities, a priority in LMICs must be to separate pedestrians from vehicles on the roads; simple modifications to road design have been suggested by the Transport Research Laboratory to achieve this (Transport and Road

TABLE 79. FATALITY RATES IN VARIOUS LMICS

<i>Author and year</i>	<i>Country</i>	<i>Rate per 10,000 vehicles</i>	<i>Rate per 100,000 population</i>
NRSC 1992	Kenya	68	11.4
Zwi 1993	Zimbabwe	28.8	11.1
Jayasuria 1991	Papua New Guinea	67.4	9.9
Mohan 1985	India	12.4	11.6
Krishnan 1992	Malaysia	8.4	2.2
Jadaan 1989	Jordan	23.5	-
Olivares-Urbina 1968	Mexico	40	-
Bangdiwala 1987	Haiti	302	31.7

Source: W. Otero, P. Garner and A. B. Zwi, "Road traffic injuries in developing countries: a comprehensive review of epidemiological studies", *Tropical Medicine and International Health*, vol. 2, pp. 445-460 (1977).

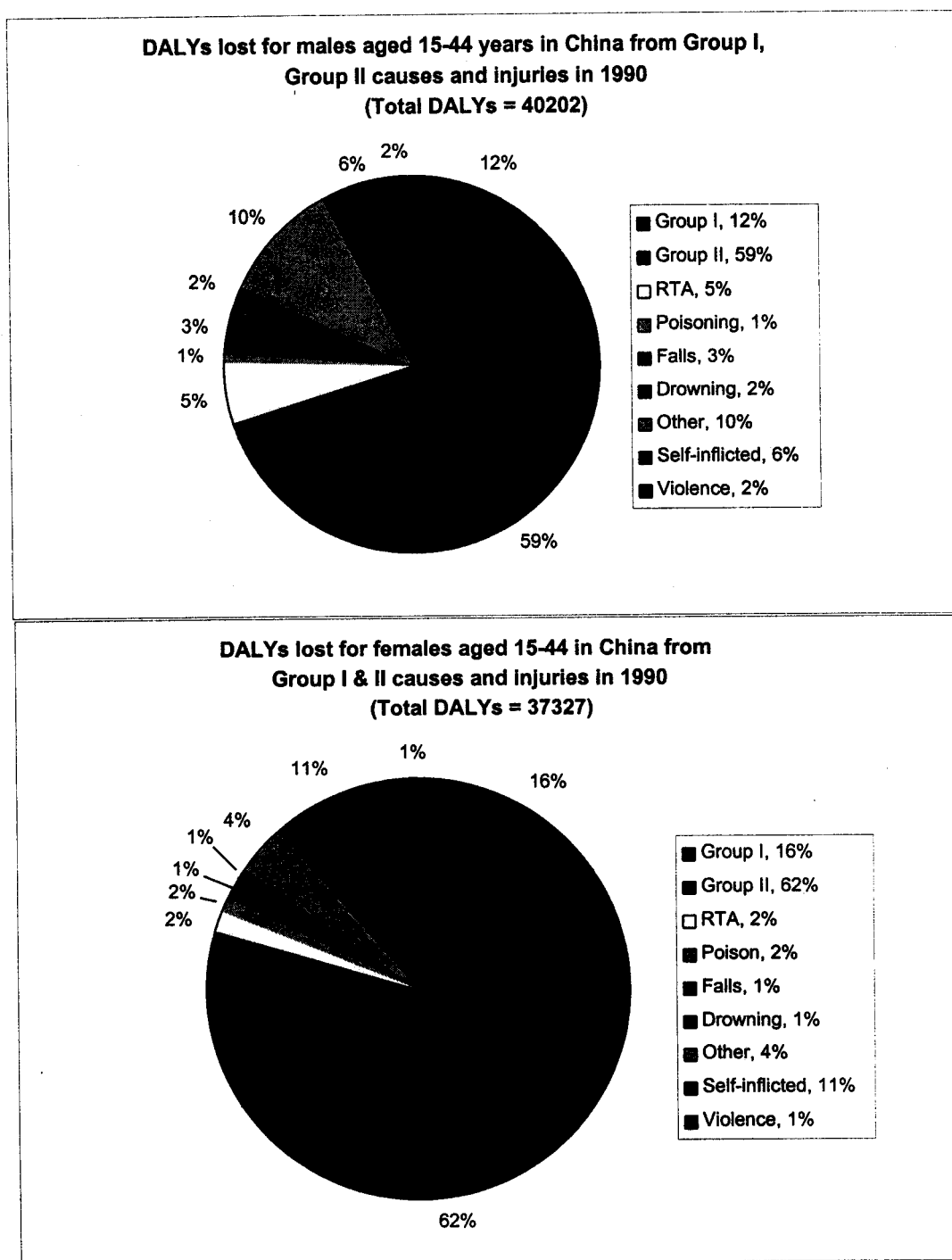
**Figure 122. Percentage of DALYs lost in India by gender for ages 15-44 in 1990**



Source: C. J. L. Murray and Alan D. Lopez, *Global burden of disease. A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Volume I* (Cambridge, Harvard University Press, 1996b).

Total DALYs in thousands. Injuries constituting less than 1 per cent not shown.

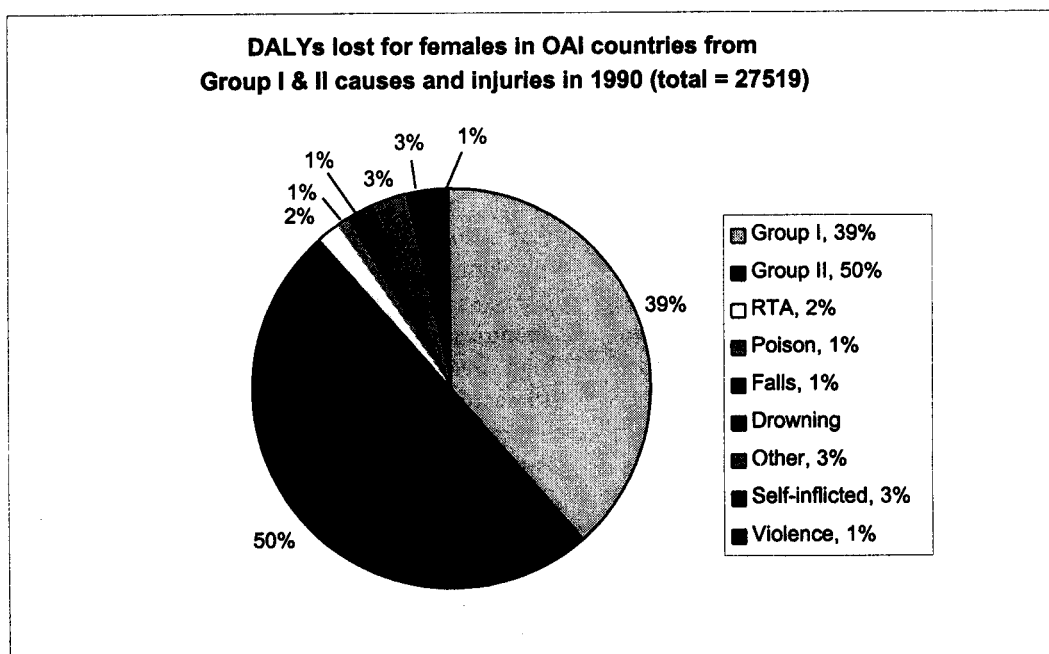
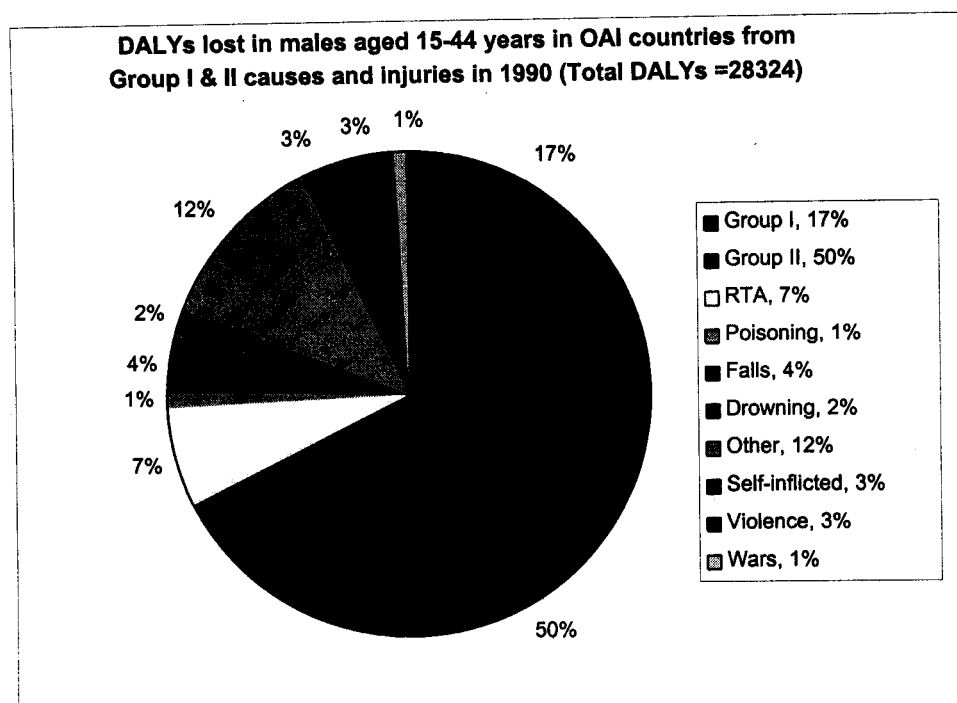
**Figure 123. Percentage of DALYs lost in China by gender for ages 15-44 in 1990**



Source: C. J. L. Murray and Alan D. Lopez, *Global burden of disease. A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Volume I* (Cambridge, Harvard University Press, 1996b).

Total DALYs in thousands. Injuries constituting less than 1 per cent not shown.

Figure 124. Percentage of DALYs lost in OAI by gender for ages 15-44 in 1990



Source: C. J. L. Murray and Alan D. Lopez, *Global burden of disease. A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Volume I* (Cambridge, Harvard University Press, 1996b).

Total DALYs in thousands. Injuries constituting less than 1 per cent not shown.

Research Committee, 1991). A safety audit has also been suggested as a routine part of donor investments in infrastructure development, in particular the construction of roads. The increase in traffic crashes and collisions may increase in the presence of particular forms of macroeconomic development; amongst these are the rapid development of infrastructure in periods following intense civil conflicts: the authors have an impression (as yet unstudied) that countries such as Uganda and Cambodia which receive large amounts of donor assistance in the aftermath of wars may compromise some of that investment through increased levels of traffic crashes and collisions.

Many of the innovations in road and car safety developed in industrialised countries need to be assessed for their relevance to LMICs. However it has been suggested that there are some technologies that are transferable (Forjuoh and Li, 1996). The equity implications of proposed policies also need consideration: although interventions in wealthier countries have focused upon vehicle occupants, in lower income countries these would only affect the rich and investments in improving the protection of pedestrians, and the quality of public transport vehicles would be desirable. The education of the general public and of vehicle drivers is often assumed to be an important intervention: the magnitude of the benefits and the costs deserve rigorous evaluation, however.

Finally, it should be noted that differences in definitions and variations in the completeness of reporting make international comparisons difficult. Greater reliance has therefore been placed on fatality statistics. Even with these there is a need for standardisation with regard to denominators. For example, the Transport Research Laboratory for LMICs have expressed fatalities per 10,000 vehicles registered although data per 10,000 or 100,000 population would be more informative from a public health perspective. Few data are available, especially from poor countries, to carefully assess the vehicle-kilometres travelled, another important base upon which to compare crash, collision and RTA-fatality rates between countries. Non-fatal motorist injuries are more completely collected than non-motorised road injuries such as those associated with bicycles or road-side events

such as falling. It is thought that whereas there is almost complete reporting of fatal injuries, this diminishes to about 70 per cent for slight injuries in Great Britain but to about 45 per cent in Germany. Police records do not routinely link with hospital data thus limiting the completeness of information on the burden associated with RTAs. Numerous biases result from the presence of incentives (or more usually, disincentives) to report, such as the penalties for driving under the influence of alcohol.

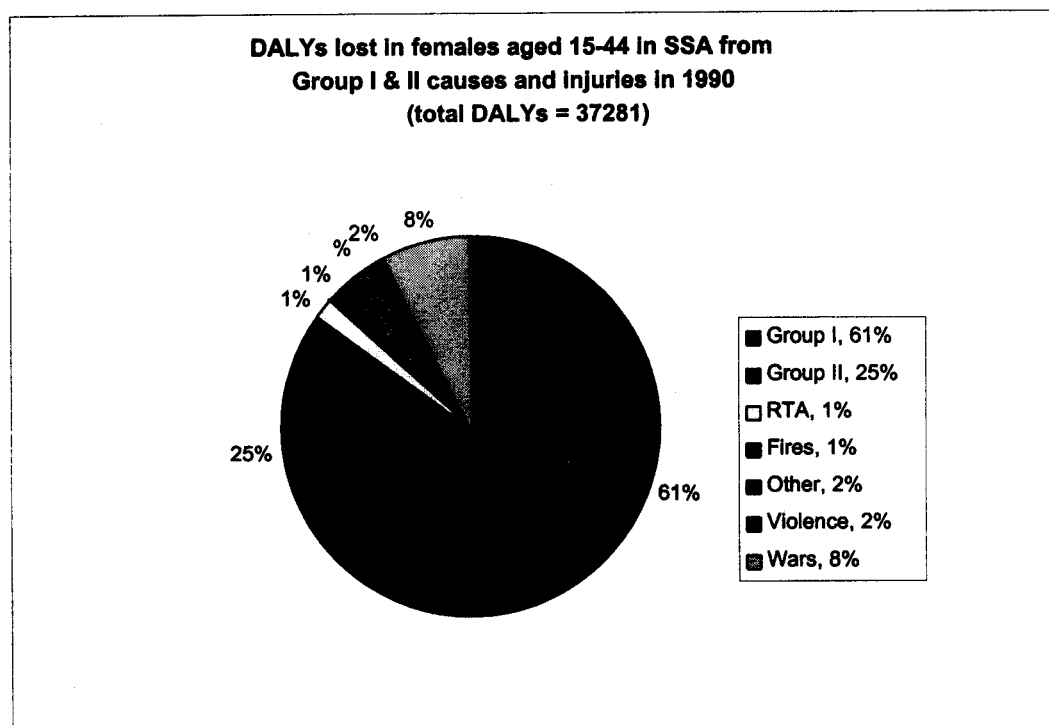
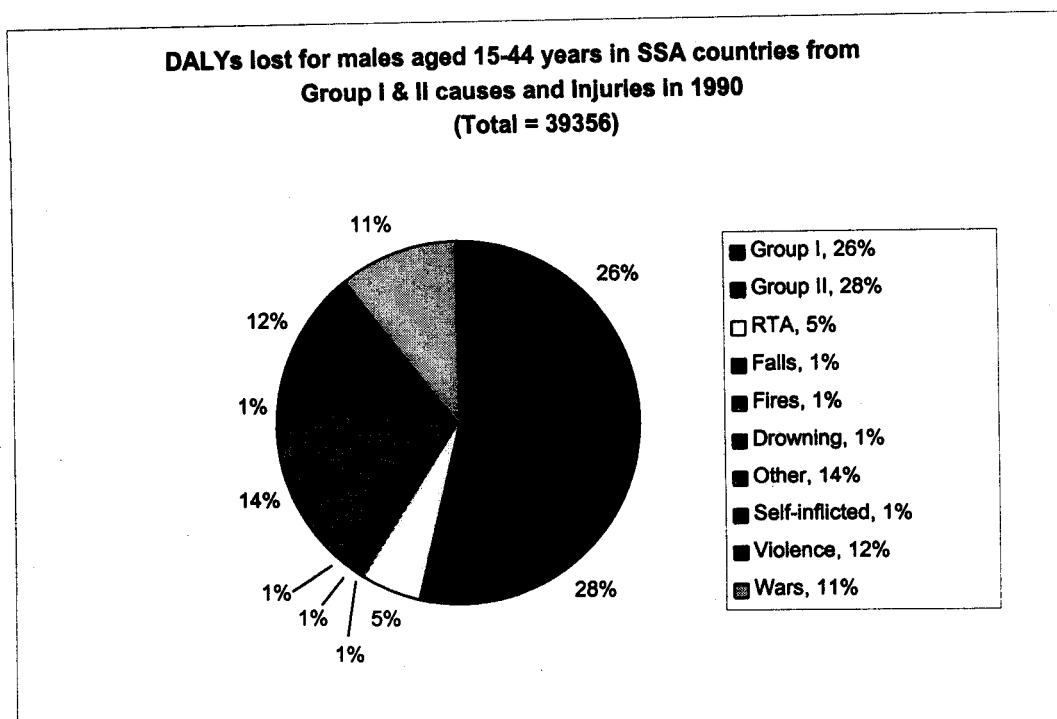
## *2. Interpersonal violence*

Mortality rates from violence are shown in figure 116. The particularly high rates for males between the age of 15 and 44 years in SSA and LAC are striking. Violence contributes to 12 per cent and 11 per cent of DALYs lost in SSA and MEC in this age group (Murray and Lopez, 1996a, also see figures 125 and 126). These are supported by other studies and in Mexico homicide has been shown to be the largest cause of DALYs lost (Lozano and others, 1995). The roles of alcohol and of substance misuse as risk factors in some settings have been highlighted. Rates vary within countries, and some of the higher rates are in urban communities, with poverty, disruption of social networks and marginalisation as key problems (Sathiyasekaran, 1996; Wallace 1997). It has been argued that relative rather than absolute deprivation is especially important in relation to violence occurrence. Within the EME, striking differences are present between countries. The United States has particularly high firearm-associated mortality. Among 15-19 year olds in the United States, 27 per cent of injury deaths resulted from firearms; in Great Britain the proportion was closer to 1 per cent. The majority of these are due to homicides with rates of 20.7 per 100,000 in the United States and 0.7 per 100,000 in Great Britain (Baker, 1997). This higher rate has been linked to the availability of firearms, inner city deprivation and misuse of substances. However the problem is particularly under-researched in SSA, and requires urgent attention.

Violence against women is a significant health problem affecting all societies and warranting far more attention in its own right. Prevalence studies highlight the scale of the



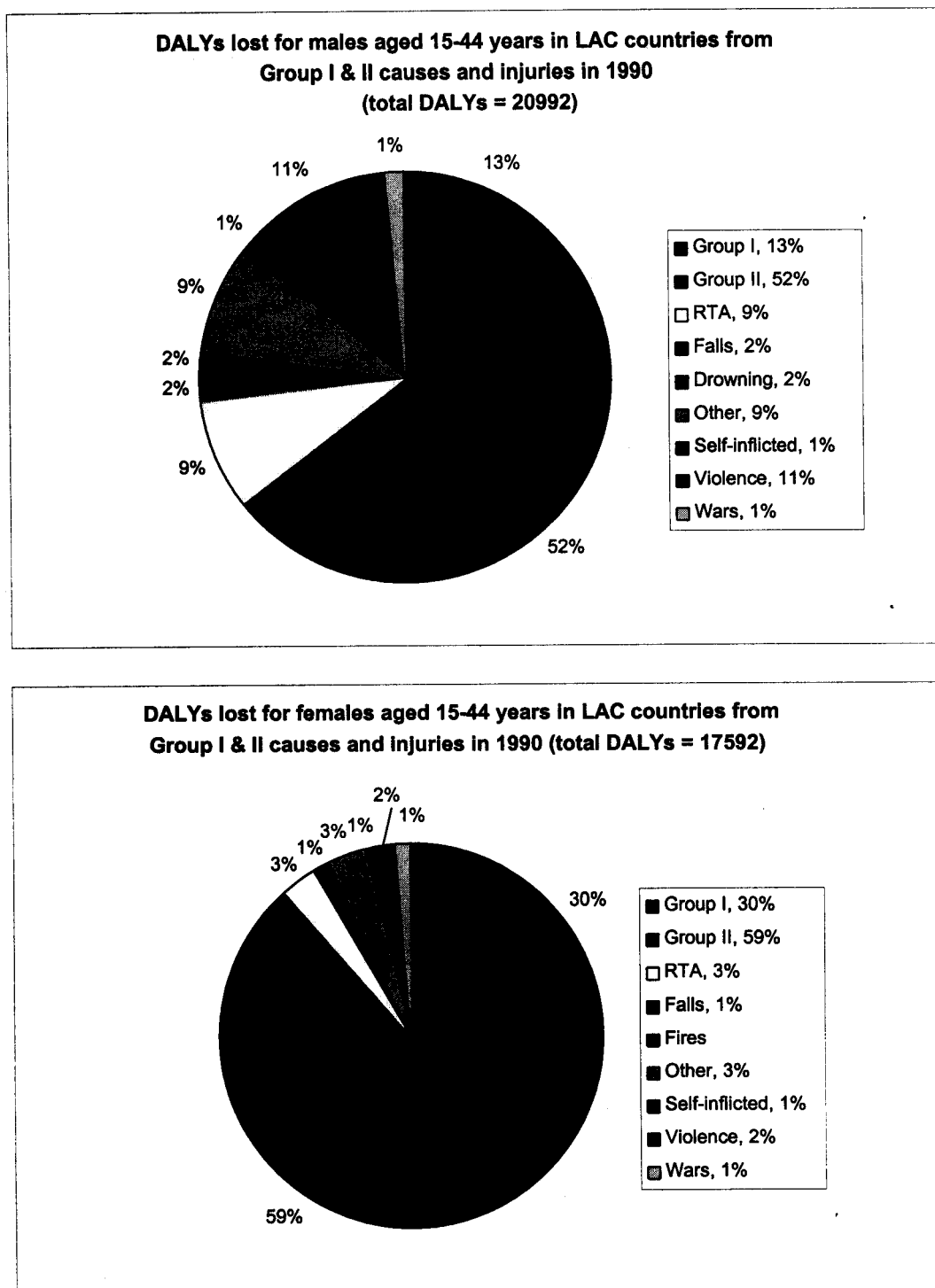
**Figure 125. Percentage of DALYs lost in SSA by gender for ages 15-44 in 1990**



Source: C. J. L. Murray and Alan D. Lopez, *Global burden of disease. A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Volume I* (Cambridge, Harvard University Press, 1996b).

Total DALYs in thousands. Injuries constituting less than 1 per cent not shown.

**Figure 126. Percentage of DALYs lost in LAC by gender for ages 15-44 in 1990**



Source: C. J. L. Murray and Alan D. Lopez, *Global burden of disease. A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Volume I* (Cambridge, Harvard University Press, 1996b).

Total DALYs in thousands. Injuries constituting less than 1 per cent not shown.

problem although differing methodologies and weak study designs impede regional and national comparisons. Lifetime rates of exposure to various forms of violence range from 22-35 per cent in the United States, 30 per cent in Barbados, 42 per cent in Kenya to 60 per cent in Ecuador (Heise 1994). The violence may result in, but is not always associated with injuries although psychological and sexual abuse also occurs. In most cases the perpetrator is the intimate partner. This is another under-researched area. Prevention requires an understanding of the context in which the abuse occurs and of the potential for developing appropriate interventions through a process involving a range of stakeholders (Heise and others, 1994; Zwi 1996).

A key issue is determining how best to address social problems, such as violence against women, by involving the health sector alongside other sectors, but without redefining the problem as a health problem alone. It is primarily through mobilising a comprehensive response to focus upon social and cultural factors, economic conditions, gender relations, and the legislative framework, that violence against women will be able to be effectively tackled.

### 3. Wars

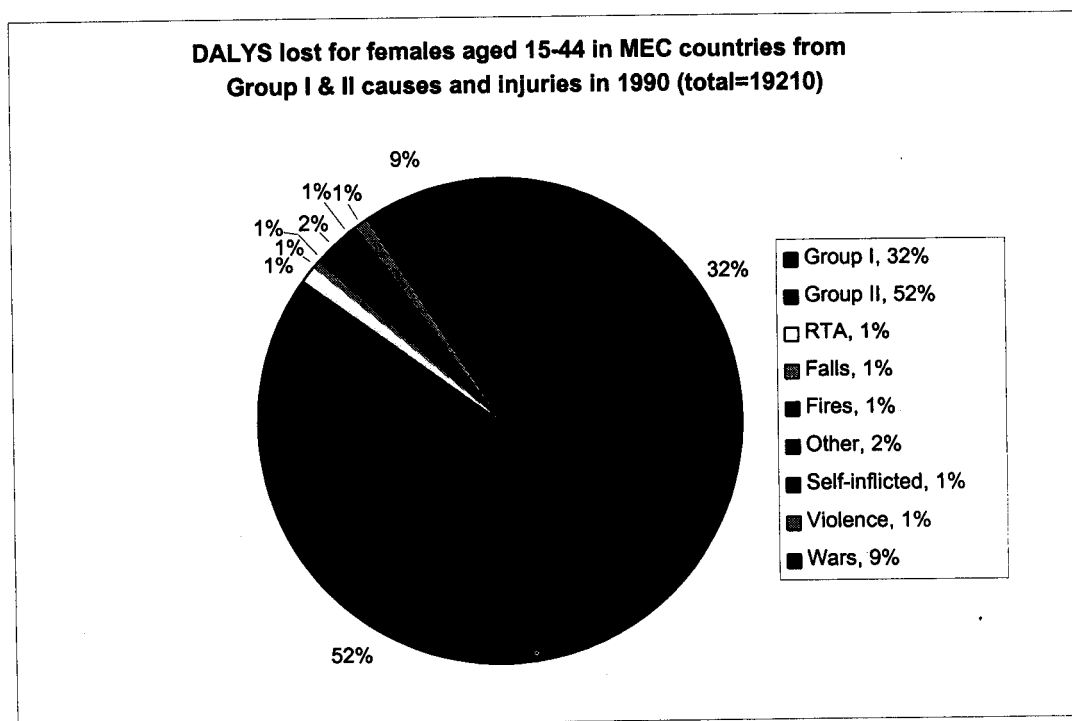
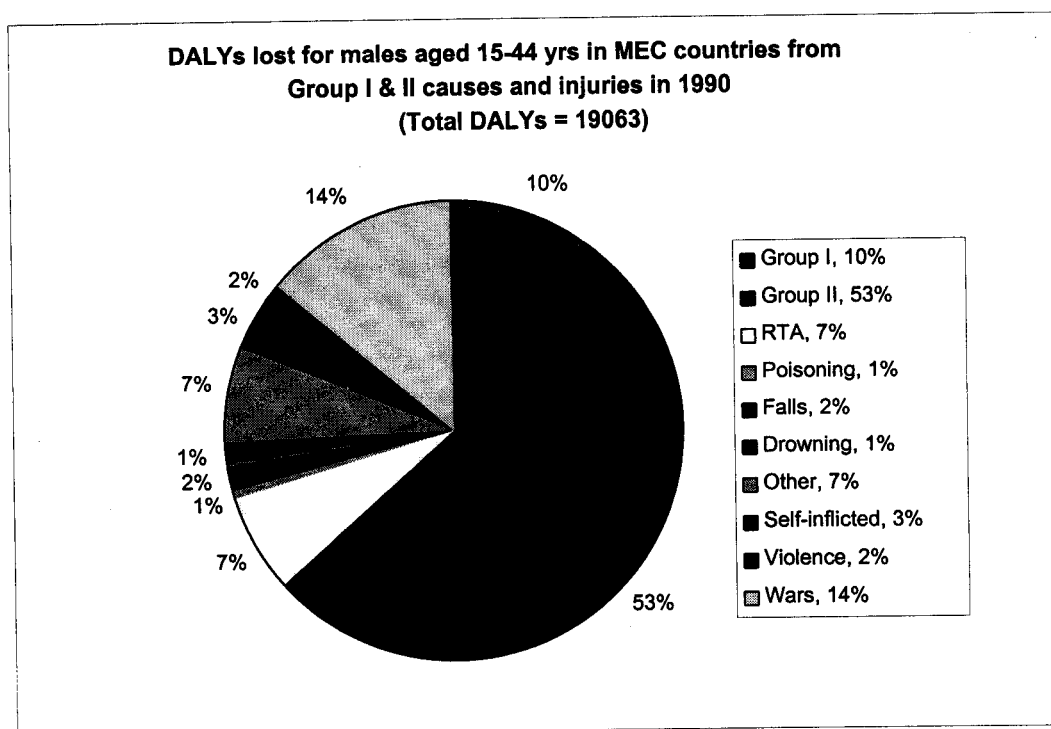
Wars contribute substantially to injury mortality and morbidity. Since 1980 there have been about 130 armed conflicts world-wide, with a marked change in their distribution since the end of the Cold War in 1991. More conflicts are now reported from the North, in particular from former members of the Soviet Union and from other countries in Central and Eastern Europe. Conflicts have increasingly targeted civilian populations, resulting in high casualty rates, widespread human rights abuses, forced migration and in some countries the total collapse of governance (Toole and Waldman, 1997, Davis 1996). The number of refugees increased from 5 million in 1980 to more than 20 million by late 1994. At least an equal number are estimated to be internally displaced (US Committee for Refugees, 1995). Severe public health consequences have been documented due to complex emergencies following armed conflict, especially in developing countries. In Africa crude mortality rates have

increased by 80 times the base rate where many of the deaths have been due to nutritional deficiencies and communicable disease. Nevertheless, fatal and non-fatal injuries due to war trauma and landmines have been common, especially among internally displaced persons and those who have been trapped in areas of conflict (Cutting and Agha, 1992; Toole and Waldman, 1990 and 1993). There are few publications that examine injuries in refugee or internally displaced populations (Pillai, 1994). Figure 128 shows that the greatest mortality from wars is in SSA, followed by the MEC and FSE. The age group with the highest burden of disease is the 15-44, as shown in figures 120, 125 and 127.

Before the Second World War in Europe and North America, a higher proportion of deaths in war was due to communicable disease than to injury. In modern warfare the development of high technology explosive and projectile weapons has resulted in a reversal of this ratio (Garfield and Neugut, 1991). War-related trauma has been the most common cause of death associated with conflict in the Former Soviet sphere. In Sarajevo between April 1992 and March 1993, 57 per cent of all deaths were due to war-related trauma; a period during which the crude mortality rate in the city increased fourfold (Center for Disease Control and Prevention, 1993). Sexual abuse is common in many unstable situations and is used as a weapon of war in order to undermine local population cohesion and disempower women. Sexual abuse has, with few exceptions such as the rape of 20,000 or more women in the former Yugoslavia, been relatively neglected, despite the significant public health burden associated with psychological distress, sexually transmitted diseases and HIV infection, and unwanted pregnancies. Survivors need protection from violence as well as access to treatment and rehabilitation where appropriate.

Hospitals and health services in affected areas are overwhelmed by the needs of the war wounded and tertiary prevention and rehabilitation have been hampered by resource constraints. Despite the recent award of the Nobel Peace Prize to the international campaign to ban them, landmines still represent a massive prob-

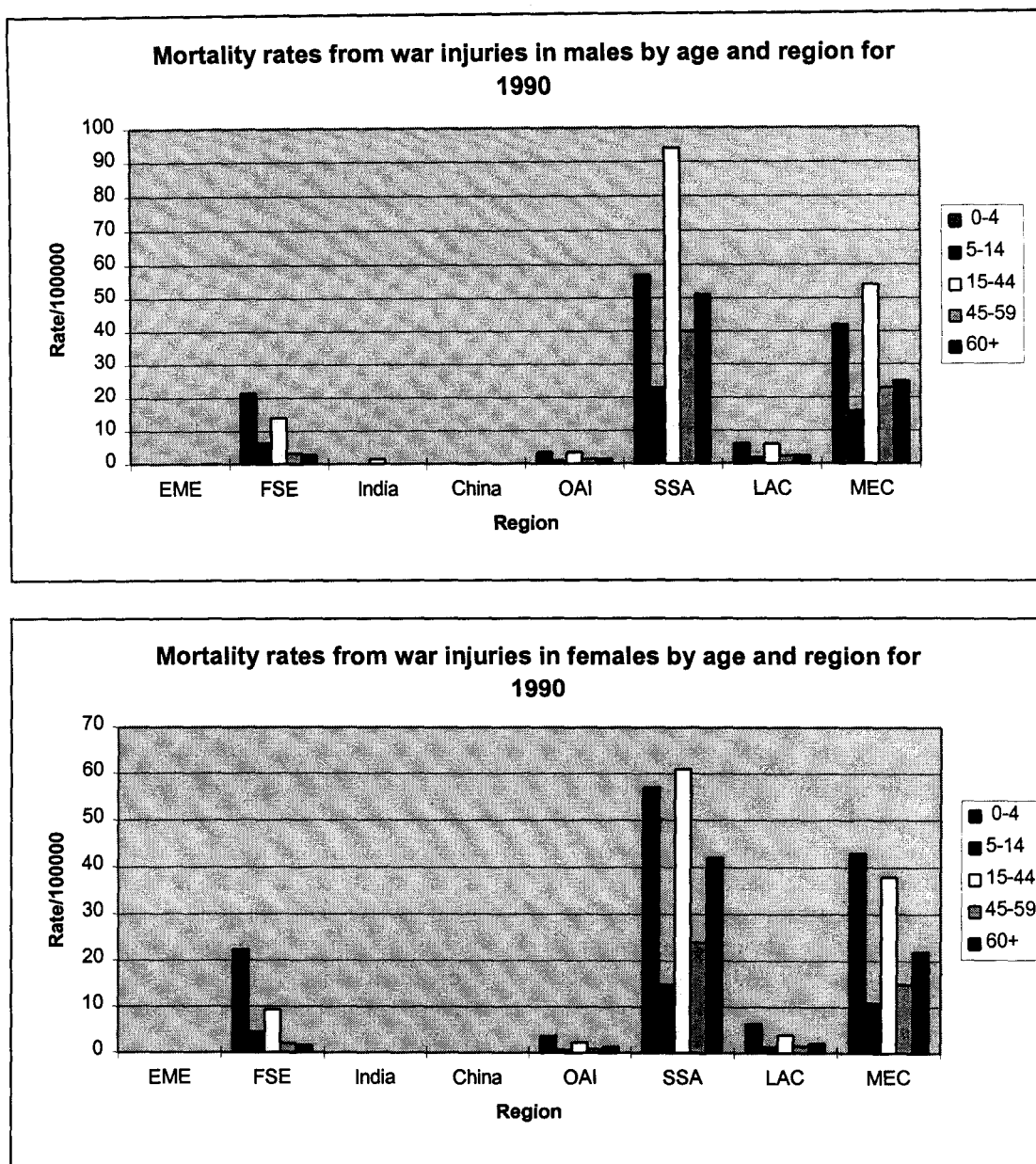
Figure 127. Percentage of DALYs lost in MEC by gender for ages 15-44 in 1990



Source: C. J. L. Murray and Alan D. Lopez, *Global burden of disease. A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Volume I* (Cambridge, Harvard University Press, 1996b).

Total DALYs in thousands. Injuries constituting less than 1 per cent not shown.

Figure 128. Mortality from war injuries by sex, age and region, 1990



Source: C. J. L. Murray and Alan D. Lopez, *Global health statistics: a compendium of incidence, prevalence, and mortality estimates for over 200 conditions. Volume II* (Cambridge, Harvard University Press, 1996a).

lem causing deaths, injuries and disabilities. Increasing attention is required to determine the effectiveness of interventions such as mine awareness campaigns and to maximize the benefits of mine clearance activities (see box 1).

The problems of collecting accurate data are most pronounced in unstable situations where there is population displacement and uncertainty

regarding the size of the affected population. Data problems in estimating mortality include: a) lack of standard reporting procedures; b) failure of families to report all deaths; c) inaccurate estimates of affected populations for calculating mortality rates; and d) poorly representative population surveys. In general mortality rates have tended to be underestimated because of under counting of deaths and by exaggerated population estimates (Toole and Waldman,

1997, Center for Disease Control and Prevention, 1992). Aside from real difficulties in collecting data in conflict situations, a wide range of biases and statistical manipulations also occur given the politicised nature of monitoring and disclosing war-related deaths and disabilities (Zwi, 1996b).

#### D. COSTS OF INJURIES

The burden of disease has been described in terms of DALYs, premature mortality and hospitalisation. These result in high costs to society due to the human costs, loss of productivity, damage of property, health service costs, and costs to the family and community. There are few studies that comprehensively quantify the economic costs of injuries.

In the United States, the National Safety Council (1993) described a morbidity rate of 61 per 100,000, with 150,000 fatalities in all ages, being the leading cause of death to those aged 1-44. The economic cost to the nation was estimated at US\$ 400 billion. Malaysia has experienced increasing affluence and rapid industrialisation. This has resulted in a surge of road and occupational injuries, 75 per cent of these occurring in the under 45 years of age. The costs have been estimated at US\$ 725m (Arokiasamy, 1994). In Thailand, the death rate in 1987 from injuries was 33 per 100,000 and 30 per cent hospital beds occupation was due to injuries. The economic cost to the nation was estimated at US\$ 1.5 billion (World Health Organization, 1987; Barss and others, 1998). Various approaches can be used to assess the cost-of-disease and although such analyses have substantial limitations they do indicate the burden on society associated with these problems. What remains unclear is the proportion of that burden that could be avoided or constrained by appropriate interventions. The use of measures such as costs per DALY saved can be used to ensure that interventions are effective investments in reducing the burden of disease.

A disproportionately small amount is spent on injury research relative to the economic costs incurred. For example the total lifetime costs of all injuries that occurred in 1985 in the United States were estimated at \$158 billion, six times

the losses for cardiovascular disease. Yet the federal research expenditure for injuries was only \$160 million, whereas that for cardiovascular research was \$930 million (Baker and others, 1992). There is an even more dramatic neglect in terms of external assistance provided internationally; for leprosy this was US\$ 50 per DALY, \$4 for HIV and other sexually transmitted disease, but only \$0.01 for injuries (Michaud and Murray, 1994).

A significant challenge is to find ways of mobilising funds to address the problem of injuries. Involving the private sector is key and means should be identified to involve the national and transnational vehicle, insurance and oil industries in contributing something back to the societies in which they make their profits. A range of stakeholders are identifiable in relation to particular types of injuries and their full involvement in promoting a safer environment and improving care and rehabilitation should be actively solicited.

Few studies of the cost-effectiveness of injury interventions have been undertaken (see cell E, box 1), and even fewer in LMICs, making it difficult for policy makers and donors to determine where best to invest scarce health-related resources. It is likely however, that certain interventions which are relatively simple will return a high yield e.g. black spot investigations to identify and correct sites at which traffic crashes and collisions are especially frequent.

#### E. INTERVENTIONS AND POLICY

Injuries are of profound importance given the predicted increases in the burden of disease that they will cause, as well as the young age at which most injury deaths and disabilities occur. Injuries account for one in seven healthy life years lost world-wide; by 2020 they will account for one in five, with low and middle-income countries bearing the brunt of this increase. World-wide, intentional injuries (suicide, homicide and war) account for almost the same number of disability-adjusted life years (DALYs) lost as either sexually transmitted diseases plus HIV infection combined, or tuberculosis. Unintentional injuries cause as many

DALYs lost as diarrhoea, and more than the DALYs lost due to cardiovascular disease, malignant neoplasms, or vaccine-preventable childhood infections. In developing regions in 1990, injuries to males aged 15-44 years led to 55 million DALYs lost, over one third of all DALYs lost from all causes in this sex and age-group. There are upward trends in injuries in many regions, especially those undergoing major economic, political and social transitions.

Poverty and injuries are linked in a variety of ways. Those in absolute and relative poverty are at increased risk through greater exposure to risk situations. Those who suffer injuries and disability require medical and rehabilitation care that they may not be able to afford, leading to the disposal of essential assets. Those disabled or killed represent considerable lost income generation for the affected families and households. Equity concerns should be increasingly incorporated by policy-makers as they determine how best to promote appropriate policy based on carefully assessed empirical data.

Interventions of proven effectiveness can be adapted to low and middle income countries (Forjuoh, 1996; Zwi, 1996). *Primary prevention* requires pre-event action: such as installing safety devices on dangerous machines and tools, improving the road environment and conditions of vehicles, placing child-proof caps on pesticide, medicine and kerosene containers to avoid poisoning, using fire-resistant fabrics and raising or enclosing cooking areas to prevent burns and scalds to children, controlling the availability of firearms and banning the use of landmines, promoting driver, pedestrian and worker education (Haddon, 1963).

*Secondary prevention* aims to reduce the impact of an event that may result in injury: promoting the use of child seats and restraints, airbags, seatbelts, side-impact bars and reinforced metal roofs in vehicles, promoting motorcycle and bicycle helmet use. *Tertiary prevention* highlights the post-event care of injuries, through improving pre-hospital care with effectively trained ambulance staff and systems, good quality accident and emergency and surgical care, as well as high standards in

the quality and coverage of rehabilitation services.

Upstream interventions need to identify and respond to those factors which heighten injury risk or propel people into risk situations, such as poverty and inequity, overcrowding, environmental decline, gender inequalities and substance abuse. Lessons can be learned from examining the features of a number of injury problems.

Preventing violence against women requires an understanding of the context in which different forms of violence occur. Upstream population-wide approaches to reducing gender discrimination and violence imply changes to how boys and girls are socialised, how the media portrays relationships, how conflicts are resolved within the household, and what messages society conveys regarding the social acceptability of physical, sexual and psychological abuse (Heise and others, 1994). The focus should be not only on the affected individual but also on the entire population in an attempt to shift behaviours and practices (Rose, 1992). These approaches are complex and long-term, and need to be combined with other concurrent interventions. Increasing awareness and facilitating the documentation and quantification of violence against women is crucial to provoking a social policy and health sector response. Health workers can assist in early identification of those at risk, providing sensitive care for those affected, referral to appropriate support agencies and ongoing advocacy. Enhancing the health sector response requires the mobilisation of resources, training, and ongoing audit. Each of the three responses will have to be fought for in these times of increasingly constrained resources.

Responses to other types of injuries, such as those occurring on the roads or at work, have elements of the same approaches. Developing appropriate policies and ensuring their implementation remains complex and require the interweaving of educational, engineering, environmental, fiscal, legislative and enforcement interventions.

A wide range of common-sense and effective road safety interventions exist. They include

improved road design (e.g. do not build a large road with a school and shops on one side and residential areas on the other), enforcement of drinking and driving laws and regulations, detection of and response to clusters of fatal road crashes (black spots), reducing and monitoring vehicle speeds, introduction of traffic calming measures in built-up environments and educating road users to utilise seat-belts, child seats, motorcycle and bicycle helmets. Particular attention to improving safe use of motorcycles, buses, trucks, and public transport vehicles is especially important in low and middle-income countries. A co-ordinated road-safety plan that is context-specific, formulated through a consultative process involving key stakeholders, and has access to expertise in a range of fields, is a valuable vehicle for developing an appropriate and co-ordinated multisectoral response.

Occupational injuries remain a major problem in LMICs and may increase in situations where informal sector employment is increasing and state systems of regulation and enforcement of standards decline. Injury rates are especially high in certain industries such as mining, construction, agriculture and transport industries (Baker and others, 1992); responding to these problems requires negotiation between the key stakeholders, notably labour, government, private sector and other interested parties. Poor working conditions, along with unsafe design and maintenance of local and imported machines, long working hours, lack of emergency care services, low literacy levels, weak trades unions, and the increasingly competitive global market conspire to place workers at high risk of injury in many countries.

Interpersonal violence has clear links with absolute and relative deprivation, alcohol consumption, availability of firearms and other weapons, and with dominant societal values and modes of conflict resolution. An assessment of the evidence regarding the effectiveness, costs, equity and acceptability of violence-related interventions is required. In the few countries where interventions have been widely employed, such as the United States, the evidence for the effectiveness, for example of educational initiatives in schools, remains limited. Analyses of how and why certain communities, municipi-

palities (e.g. Cali, Colombia), or nations have developed their response to violence would be of great value.

Landmines are a significant element of war-related injuries that demand attention. Aside from supporting upstream activity to ensure a world-wide ban on their production and distribution, efforts to increase the effectiveness of mines awareness and demining activities are necessary. Policy lessons from the successful International Landmines Campaign should be rapidly learned. They include the value of quantitative and qualitative data, the involvement of prestigious personalities, and the mechanisms of developing international advocacy activities. They can be of value to the campaigns focussed on injuries in general and on other global problems. Consideration should be given to the way investments in prior support for emergency assistance to landmine victims can best be generalised and sustained in extending emergency surgery and rehabilitation facilities.

Childhood injuries, including burns, poisoning, fractures and drowning are all key issues which have received relatively little public attention in LMICs. Tertiary prevention (i.e. improving care after an injury event) is a key task of the health sector if costs and outcomes are to be improved. Information is required about community level knowledge and perceptions, awareness of first aid measures, skills and quality of care provided by traditional and other private providers, and of the systems of care provided by the public sector. Evidence-based population-wide preventive interventions also deserve attention. Child-centred campaigns may provide a ready means of drawing together a wide range of stakeholders in support of a developing agenda, given the potential to obtain support for making childhood safer (Reich, 1995). The lack of an active civil society is considered to also be one of the reasons for poor response to childhood injuries in the Eastern and central Europe (ECOHST, 1998).

A number of wealthy countries have, in the past decade, devoted considerable resources to tackling the problem of injuries and to ensuring that safety and security are increased for the majority of their citizens. The most notable



success, at least in attracting attention to the issue and in devoting resources to it, has been the United States. A number of European nations and those in the Antipodes have also begun to address these social and health problems. An example is the Health of the Nation policy in England (1992), which has targeted "accidents" as one of the five health gain areas and which has promoted multisectoral collaboration (1992). Experience in LMICs is extremely limited, although a number of countries, including those in the Caribbean, Malaysia and Zimbabwe, have highlighted attention to injuries as part of their forthcoming health agendas (Forjuoh and others, 1997). Certain issues such as violence against women are being actively tackled while others have been much neglected.

In Zimbabwe attention to the problem began with the Ministry of Health and Child Welfare (MOHCW) identifying injury control as a priority, given that it was the leading cause of death in young adults and posed a significant load on out-patient, in-patient and rehabilitation services. The MOHCW, with some support from overseas donors and academic institutions, sought to systematically identify the burden and respond to injuries. A detailed situation analysis brought together all secondary sources of information (Zwi and others, 1993). Routine data showed marked increases in recent years from traffic-related injuries and fatalities, violence, and rape. The burden of injuries and associated costs to the health services were considerable. Data from government, voluntary and private sector actors engaged in injury-related activities were assembled to understand ongoing activities and priorities and to assist in developing policies and strategies likely to win widespread support. A lead person within the MOHCW was mandated to co-ordinate activities. That provided the basis for developing and supporting an Intersectoral Injury Control Committee and running workshops to identify and support data-gathering, research, and intervention initiatives. A key intervention is the potential to establish a national road safety plan. Similar strategies in response to violence against women, work injuries and child welfare are feasible and should be explored. Recognition must be given to supporting local initiatives, within local government, the health services, other sectors, and most importantly,

communities. Key elements to be included are improving the coverage and quality of care, through the application and careful evaluation of interventions to assess their effectiveness, efficiency, cost and equity implications (Sethi and others, 1995).

Regional and multisectoral exploration of solutions is desirable. Funding support to establish an exchange of information and expertise in sub-Saharan African, Latin American, or Asian low income countries, and transitional Eastern European countries would be a helpful investment. Identifying funding to support collaboration between countries is a priority that multilateral organisations, bilateral donors, and international non-governmental organisations should actively support. Mobilisation of government and non-government, as well as of public and private sector activity is necessary in order to build on local energy and commitment, build alliances and consolidate existing interventions.

Injuries demand a response now. There is much experience, however, patchy, of what can be done to counter the injury epidemic. Investments at all levels of knowledge generation around injuries are required.

#### REFERENCES

- Ali, J., R. Adam, A. K. Butler and others (1993). Trauma outcome improves following the advanced trauma life support program in a developing country. *Journal of Trauma*, vol. 34, pp. 890-899.
- Arokiasamy, J. T., and P. Krishnan (1994) Some epidemiological aspects and economic costs of injuries in Malaysia. *Asia-Pacific Journal of Public Health*, vol. 7, pp. 16-20.
- Andreasson, S., P. Allebeck, A. Romesljo (1988). Alcohol and mortality among young men: longitudinal study of Swedish conscripts. *British Medical Journal*, vol. 296, pp. 1021-1025.
- Baker, S. P., B. O'Neill, M. J. Ginsburg and G. Li (1992). *The Injury Fact Book*. Oxford: Oxford University Press.
- Baker, S. P. (1997). International comparisons: useful or odious? *Injury Prevention*, vol. 3, p. 3.
- Bangdiwala, S. I., and E. Anzola-Perez (1987). Traffic accidents as a serious health problem in selected developing countries of the Americas. *Bulletin of the Pan American Health Organization*, vol. 21, pp. 38-47.
- \_\_\_\_\_. (1990). The incidence of injuries in young people: II. Log-linear multivariable models for risk factors in a collaborative study in Brazil, Chile, Cuba and Venezuela. *International Journal of Epidemiology*, vol. 19, pp. 125-132.
- Barss, P., G. Smith, S. Baker and D. Mohan (1998). *Injury prevention: An international perspective*. *Epidemiology*,

- surveillance and policy. New York and Oxford: Oxford University Press.
- Berger, L. R., and D. Mohan (1996). *Injury control: a global view*. Oxford: Oxford University Press.
- Black, R. E. (1990). Prevention in developing countries. *Journal of General Internal Medicine*, vol. 5, pp. S132-135.
- Blum, R. W. (1991). Global trends in adolescent health. *Journal of the American Medical Association*, vol. 265, pp. 2711-2719.
- Bly, P. (1996). Road safety in the developing world. In *Health on the Crossroads: transport policy and urban health*, T. Fletcher and A. J. McMichael, eds. Chichester: John Wiley and Sons, pp. 125-140.
- Bobadilla, J. L., and P. Cowley (1995). Designing and implementing packages of essential health services. *Journal of International Development*, vol. 7, No. 3, pp. 543-554.
- Bruhning, E. (1997). Injuries and deaths on the roads--an international perspective. In *Health on the Crossroads: transport policy and urban health*, T. Fletcher and A. J. McMichael, eds. Chichester: John Wiley and Sons, pp. 109-124.
- Center for Disease Control and Prevention (1992). Famine affected, refugee, and displaced populations: recommendations for public health issues. *Morbidity and Mortality Weekly Report*, vol. 41, RR-13.
- \_\_\_\_\_ (1993). Status of Public Health - Bosnia and Herzegovina, August-September 1993. *Morbidity and Mortality Weekly Report*, vol. 42, pp. 973 and 979-982.
- Chiu, W. T., S. R. Dearwater, D. J. McCarty, T. J. Songer and R. E. LaPorte (1993). Establishment of accurate incidence rates for head and spinal cord injuries in developing and developed countries: a capture-recapture approach. *Journal of Trauma*, vol. 35, pp. 206-211.
- Crandon, I., R. Carpenter and A. McDonald (1994). Admissions for trauma at the University Hospital of the West Indies. A prospective study. *West Indian Medical Journal*, vol. 43, pp. 117-120.
- Cutting, P. A., and R. Agha (1992). Surgery in a Palestinian refugee camp. *Injury*, vol. 23, pp. 405-409.
- Davis, A. P. (1996). Targeting the vulnerable in emergency situations: who is vulnerable? *Lancet*, vol. 348, pp. 868-871.
- Downing, A. J. (1991). Pedestrian safety in developing countries. In *The Vulnerable Road User: Proceedings of the International Conference on Traffic Safety*. New Delhi: McMillan India Ltd.
- European Centre on Health of Societies in Transition (ECOHST) (1998). Childhood injuries. *A priority area for the transition countries of Central and Eastern Europe and the Newly Independent States*. ECOHOST, London School of Hygiene and Tropical Medicine.
- Ellis, A. A., and R. B. Trent (1995). Hospitalisations for near drowning in California. *American Journal of Public Health*, vol. 85, pp. 1115-1118.
- Forjuoh, S. N., and E. Gyebi-Ofosu (1993). Injury surveillance: should it be a concern to developing countries? *Journal of Public Health Policy*, vol. 14, pp. 355-359.
- Forjuoh, S. N. (1996). Injury control in developing nations: what can we learn from the industrialised countries. *Injury Prevention*, vol. 2, pp. 90-92.
- \_\_\_\_\_, and G. Li (1996). A review of successful transport and home injury interventions to guide developing countries. *Social Science and Medicine*, vol. 43, pp. 1551-1560.
- Forjuoh, S. N., A. B. Zwi and C. N. Mock (1998). Injury control in Africa: getting governments to do more. *Tropical Medicine and International Health*, vol. 3, no. 5, pp. 349-356.
- Garfield, R. M., and A. I. Neugut (1991). Epidemiologic analysis of warfare. *Journal of the American Medical Association*, vol. 266, pp. 688-692.
- Graitcer, P. L. (1992). Injury surveillance in developing countries. *Morbidity and Mortality Weekly Report* (Center for Disease Control and Prevention), vol. 1, pp. 15-20.
- Gwatkin, D. R., and P. Heuveline (1997). Improving the health of the world's poor. Communicable diseases among young people remain central. *British Medical Journal*, vol. 315, p. 497.
- Haddon, W., Jr. (1963). A note concerning accident theory and research with specific reference to motor-vehicle accidents. *Annals of the New York Academy of Sciences*, vol. 107, pp. 635-646.
- Health of the Nation (1992). London: Her Majesty's Stationary Office.
- Heise, L., A. Raikes, C. Watts and A. B. Zwi (1994). Domestic violence in developing countries: a neglected public health issue. *Social Science and Medicine*, vol. 39, pp. 1165-1179.
- Jacobs, G. D., and P. R. Fouracre (1977). Further research on accident rates in developing countries. *TRRL Supplementary Report 270*. Crowthorne: Transport and Research Laboratory.
- Jacobs, G. D. (1996). Road safety in the developing world. In *Health on the Crossroads: transport policy and urban health*, T. Fletcher and A. J. McMichael, eds. Chichester: John Wiley and Sons, pp. 141-157.
- Jadaan, K. S., and A. Bener (1993). The epidemiology of road accidents in Jordan. *Journal of Traffic Medicine*, vol. 21, pp. 399-401.
- Kirsch, T. D., R. W. Beaudreau, Y. A. Holder and G. S. Smith (1996). Paediatric injuries presenting to an emergency department in a developing country. *Pediatric Emergency Care*, vol. 12, pp. 411-415.
- Krishnan R. (1992). Safe travel - a worthwhile destination. *World Health Forum*, vol. 13, pp. 163-164.
- Lawson, J. S., and V. Lin (1994). Health status differentials in the People's Republic of China. *American Journal of Public Health*, vol. 84, pp. 737-741.
- Leon, D. A., L. Chenet, V. M. Shkolnikov and others (1997). Huge variations in Russian mortality rates 1984-94: artefact, alcohol, or what? *Lancet*, vol. 350, pp. 383-388.
- Li, G. H., and S. P. Baker (1991). A comparison of injury death rates in China and the United States, 1986. *American Journal of Public Health*, vol. 81, pp. 605-609.
- Lozano, R., C. J. L. Murray, J. Frenk and J. L. Bobadilla (1995). Burden of disease assessment and health sector reform: results of a study in Mexico. *Journal of International Development*, vol. 7, pp. 555-563.
- Mock, C. N., D. Denno and E. S. Adzotor (1993). Paediatric trauma in the rural developing world: low cost measures to improve outcome. *Injury*, vol. 24, pp. 291-296.
- Michaud, C., and C. J. Murray (1994). External assistance to the health sector in developing countries: a detailed analysis, 1972-90. *Bulletin of the World Health Organization*, vol. 72, pp. 639-651.
- Mohan, D., and P. S. Bawa (1985). An analysis of road traffic fatalities in Delhi, India. *Accident Analysis and Prevention*, vol. 17, pp. 3-45.
- Murray, C. J. L. (1994). Quantifying the burden of disease: the technical basis for disability-adjusted life years. *Bulletin of the World Health Organization*, vol. 72, pp. 429-445.
- \_\_\_\_\_, and A. D. Lopez (1996a). *Global health statistics: a compendium of incidence, prevalence, and mortality estimates for over 200 conditions. Volume II*. Cambridge: Harvard University Press.
- \_\_\_\_\_, (1996b). *Global burden of disease. A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Volume I*. Cambridge: Harvard University Press.

- \_\_\_\_\_ (1997a). Mortality by cause for the eight regions of the world: global burden of disease study. *Lancet*, vol. 349, pp. 1269-1276.
- \_\_\_\_\_ (1997b). Global mortality, disability, and the contribution of risk factors: global burden of disease study. *Lancet*, vol. 349, pp. 1436-1442.
- \_\_\_\_\_ (1997c). Alternative projections of mortality and disability by cause 1990-2020: global burden of disease study. *Lancet*, vol. 349, pp. 1498-1504.
- National Road Safety Council of Kenya (1992). *Accident statistics, 1983-1990*. Nairobi: Ministry of Public Works, Government of Kenya.
- Odero, W., P. Garner and A. B. Zwi (1997). Road traffic injuries in developing countries: a comprehensive review of epidemiological studies. *Tropical Medicine and International Health*, vol. 2, pp. 445-460.
- Odero, W. and A. B. Zwi (1999). Comparing the utility of hand held breathalysers with blood alcohol estimation in a casualty department in Kenya. *Accident Analysis and Prevention* (in press).
- Officials' Committee on Road Safety (1991). *National Road Safety Plan*, Wellington, New Zealand: Ministry of Transport.
- Olivares-Urbina, C. (1968). Increase of traffic accidents in the Distrito Federales, Mexicano. *Salud Publica (Mexico)*, vol. 10, pp. 233-236.
- Omran, A. (1971). The epidemiologic transition: a theory of the epidemiology of population change. *Millbank Memorial Fund Quarterly*, vol. 46, pp. 509-538.
- Pillai, S. K. (1994). Curative and preventive services in Batticaloa district. Victims of war in Sri Lanka. Conference proceedings, Colombo, pp. 99-103.
- Reich, M. (1995). The politics of agenda setting in international health: Child health versus adult health in developing countries. *Journal of International Development*, vol. 7, No. 3, pp. 489-502.
- Rose G. (1992). *The strategy of preventive medicine*. Oxford: Oxford University Press.
- Sandwith, A. (1979). *Traffic Safety in Zimbabwe*. Harare: Zimbabwe Road Safety Board.
- Sathiyasekaran, B. W. (1996). Population-based cohort study of injuries. *Injury*, vol. 27, pp. 695-698.
- Sethi, D., C. Makoni, L. Levy, S. Murugusampillay, A. B. Zwi, L. Gilson, J. Fox-Rushby (1995). *Effectiveness of accident and emergency services in relation to burns and head injuries in two hospitals in Zimbabwe*. London School of Hygiene and Tropical Medicine and Ministry of Health and Child Welfare, Zimbabwe.
- Smith, G. S., and P. G. Barss (1991). Unintentional injuries in developing countries: the epidemiology of a neglected problem. *Epidemiology Reviews*, vol. 13, pp. 228-266.
- Soderlund, N., and A. B. Zwi (1995). Traffic-related mortality in industrialised and less developed countries. *Bulletin of the World Health Organization*, vol. 73, No. 2, pp. 175-182.
- Toole, M. J., and R. J. Waldman (1990). Prevention of excess mortality in refugee and displaced populations in developing countries. *Journal of the American Medical Association*, vol. 263, pp. 3296-3302.
- \_\_\_\_\_ (1993). Refugees and displaced persons. War, hunger, and public health. *Journal of the American Medical Association*, vol. 270, pp. 600-605.
- \_\_\_\_\_ (1997). The public health aspects of complex emergencies and refugee situations. *Annual Review of Public Health*, vol. 18, pp. 283-312.
- Transport and Road Research Committee (1991). *Towards safer roads in developing countries. A guide for planners and engineers*. Crowthorne: Transport Research Laboratory.
- United States Commission on Refugees (1995). *World Refugee Survey*. Washington, DC: United States Government Printing Office.
- Wallace, R., and Wallace D. (1997). Community marginalisation and the diffusion of disease and disorder in the United States. *British Medical Journal*, vol. 314, pp. 1341-1345.
- World Bank (1990). Road safety: a lethal problem in the Third World. *The Urban Edge*, vol. 14, special edition.
- \_\_\_\_\_ (1993). *World Development Report: Investing in Health*. Oxford: Oxford University Press.
- World Health Organization (1993). Handle Life With Care. Prevent violence and negligence. *World Health Day* (7 April).
- \_\_\_\_\_ (1987). *Report of the Asian seminar on road safety*. Geneva: World Health Organization, IPR/APR 218G.
- Zwi, A. B., S. Murugusampillay, B. Msika and others (1993). *Injury surveillance in Zimbabwe: a situation analysis*. London School of Hygiene and Tropical Medicine, and Ministry of Health and Child Welfare, Zimbabwe.
- Zwi, A. B., S. F. Forjuoh, S. Murugusampillay, W. Odero, C. Watts (1996). Injuries in developing countries: policy response needed now. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, vol. 90, pp. 593-595.
- Zwi, A. B. (1996a). Injury control in developing countries: context more than content is crucial. *Injury Prevention*, vol. 2, pp. 91-92.
- \_\_\_\_\_ (1996b). Numbering the dead: Counting the casualties of war. In *Defining violence*, H. Bradby, ed. Aldershot, Avebury.

## XIX. HIV-INFECTION AND AIDS-RELATED DEATHS IN AFRICA

*Jacob Adetunji\**

The human immunodeficiency virus (HIV) infection and acquired immune deficiency syndrome (AIDS) have become major public health problems in many African countries. Current estimates suggest that about two out of every three cases of HIV infections are in sub-Saharan Africa. This high prevalence of HIV has led to drastic mortality increases and threatens to wipe out the gains that have been made in life expectancy at birth in many of these countries. It is unfortunate that appropriate data necessary for studying the dynamics of the HIV and AIDS epidemic are often inadequate. Most of the existing data are from small-scale, facility-based or geographically limited studies. Typically, the results are not generalisable to the national level and there is a lack of standardisation regarding the clinical definition of AIDS. As a result, much of what we know about the HIV/AIDS epidemic in Africa is based on models. The objective of this paper is to review the prevalence of HIV infection in Africa and AIDS-related mortality and to assess the quality of the data available from the region. The paper also discusses the consequences of the high prevalence of HIV infections and AIDS, expected future trends of the epidemic in Africa and the policy options available.

The paper is divided into four sections. The first section presents information available on the prevalence of HIV/AIDS in Africa and variations in the pattern of the epidemic. There is also an assessment of data needs and a discussion of the quality of the data that have gone into the major models used to study HIV/AIDS in Africa. The second section reviews the consequences of the epidemic while the third section examines future trends. The last section discusses some of the policy options that could be adopted to mitigate the consequences of the HIV/AIDS epidemic in Africa and influence its projected future course.

### A. PREVALENCE OF HIV IN AFRICA

A review of the available information on the prevalence rates of HIV infection indicates that sub-Saharan Africa has the highest rates in the world. By the beginning of 1997, about 22.6 million people world-wide either had contracted HIV infection or had AIDS, about 14 million (63 per cent) of whom were in sub-Saharan Africa (World Health Organization, 1997). By the end of 1997, the Joint United Nations Program on HIV/AIDS (UNAIDS) had adjusted the global HIV/AIDS estimates upwards to 30.6 million. Their estimates put about 21.8 million of the HIV/AIDS in sub-Saharan Africa (United Nations Program on HIV/AIDS, 1997). Figure 129 presents the yearly estimate for HIV infection among adults in sub-Saharan Africa. It shows that as of 1981, the year the AIDS virus was first identified in five gay men in Los Angeles, United States of America, about 111,000 (67 per cent) of the estimated world total of 165,000 infections occurred in Africa. The annual number of infections is estimated to have passed 1 million in 1988, and from 1992 to 1995, about 1.5 million HIV infections were estimated to occur annually in sub-Saharan Africa. Yearly estimates of HIV infections in sub-Saharan Africa relative to the world are shown in figure 130. Figure 131 presents the cumulative totals.

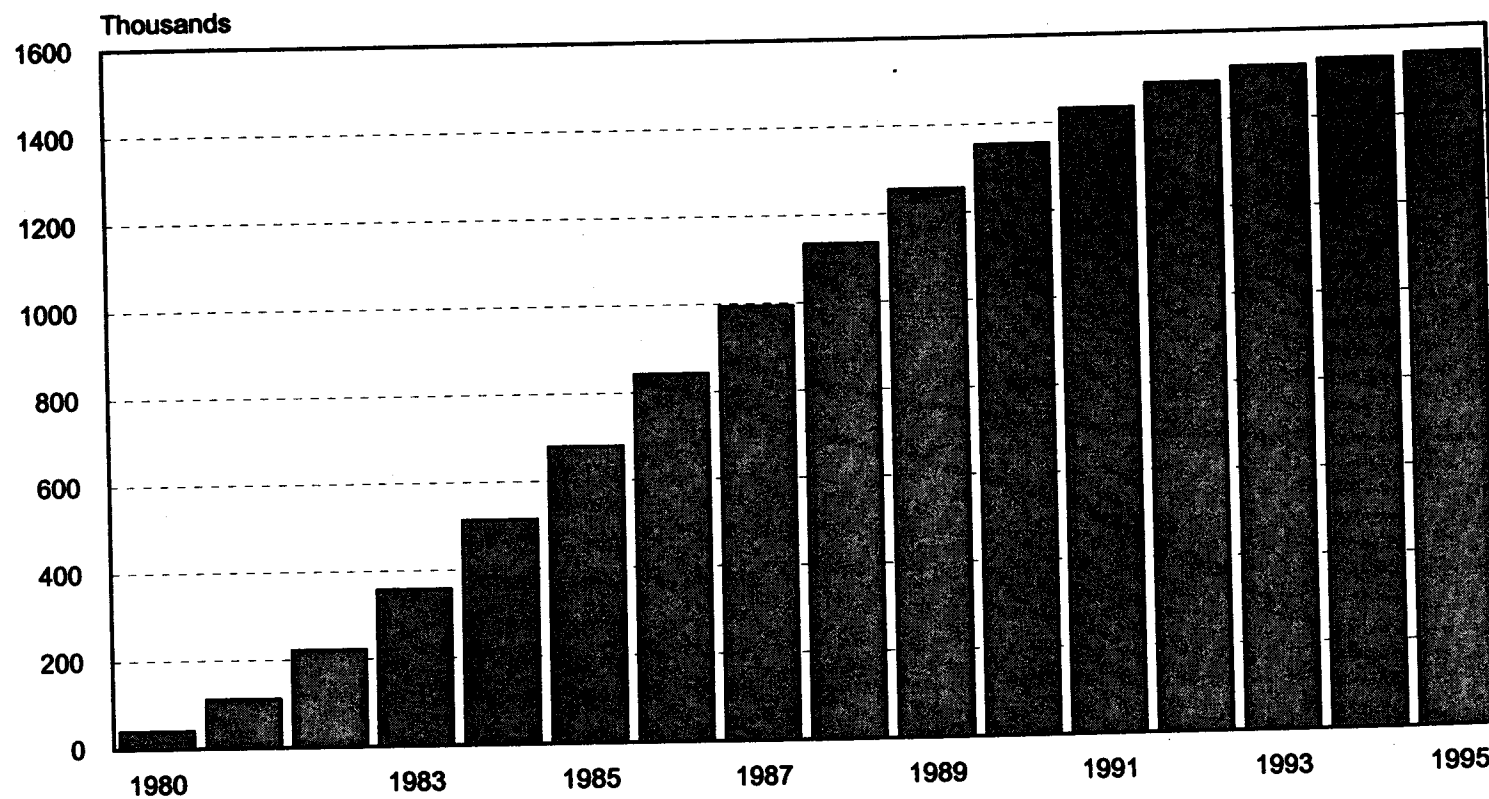
The figures show that while the annual number of infections in sub-Saharan Africa relative to the world total seems to have reached a plateau in the 1990s, the cumulative total of estimated HIV infections is still rising in Africa. For example, the annual number of HIV infections estimated to be occurring in the region shows an inverted U-shaped curve relative to the world total (see figure 132). The proportion of the total HIV infections occurring annually in Africa is estimated to have increased from 67 per cent in 1981 to a peak of 79 per cent in 1988 and then declined to 43 per cent in 1995.

In terms of deaths from HIV and AIDS-related causes, the World Health Organization

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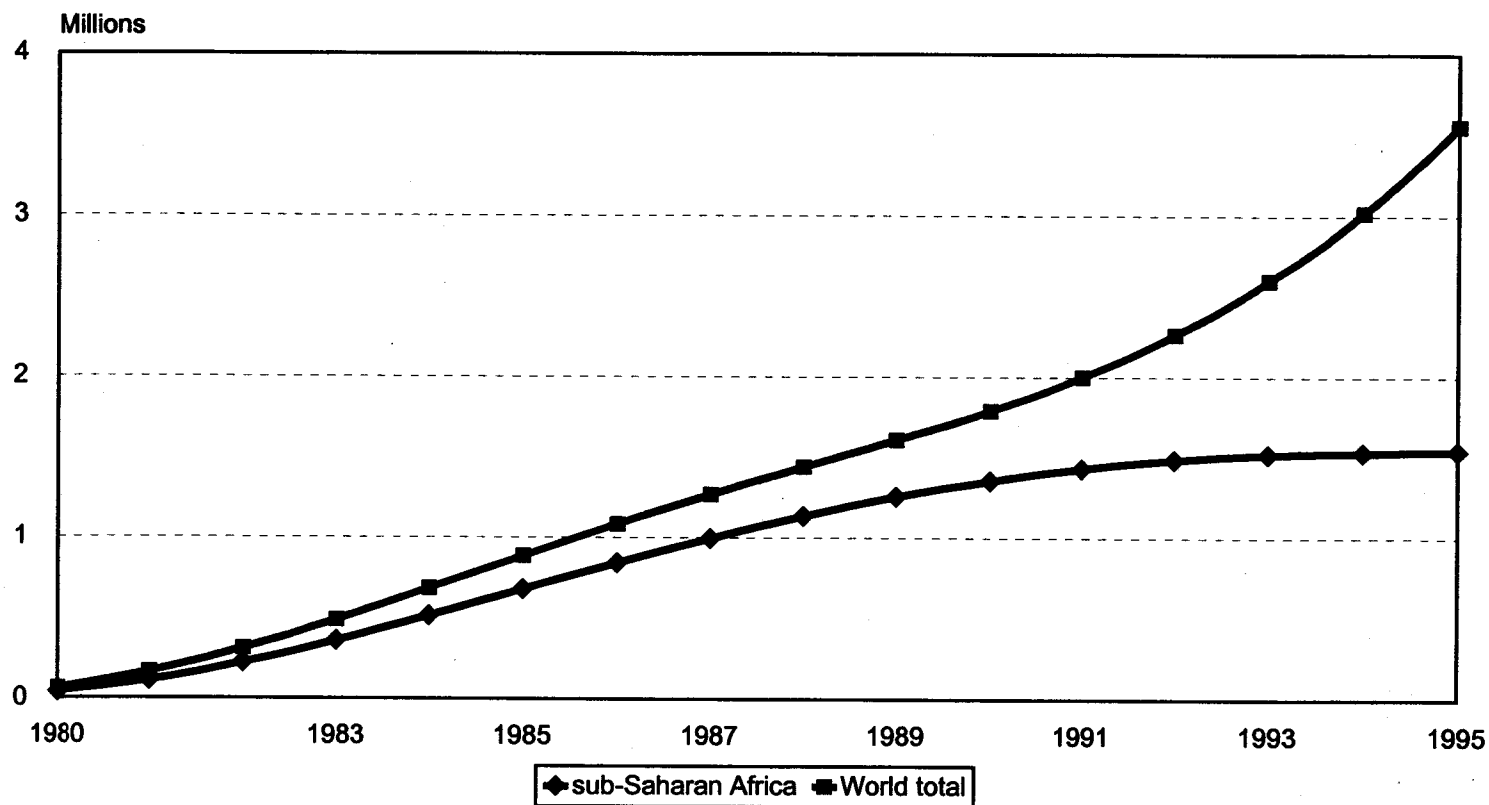
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Figure 129. Estimated yearly number of HIV infections in sub-Saharan Africa (1980-1995)



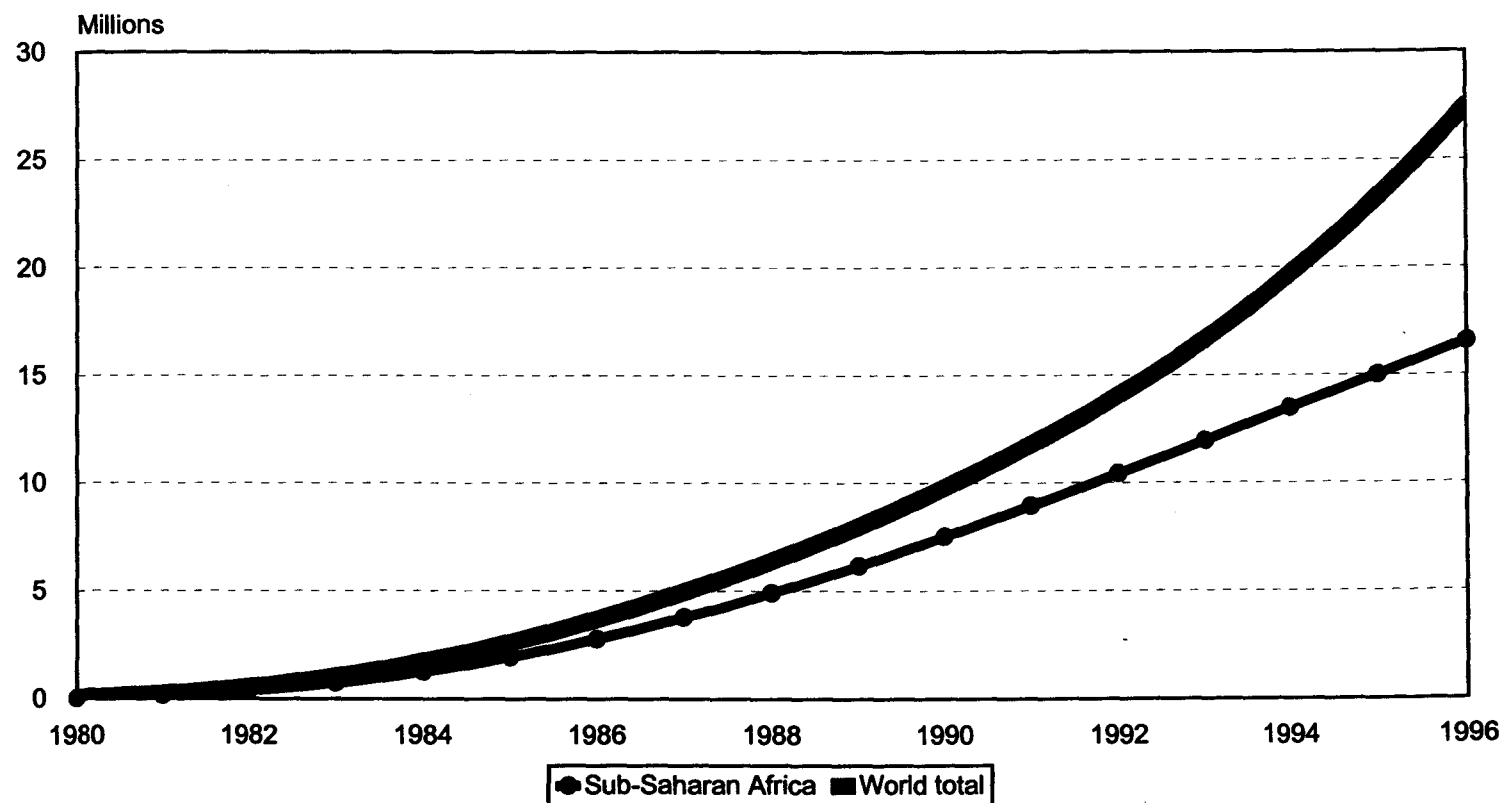
Source: Adapted from Jonathan Mann and D. Tarantola, eds., *AIDS in the World II: Global Dimensions, Social Roots and Responses* (New York, Oxford University Press, 1996, p. 12).

Figure 130. Estimated number of HIV infections occurring yearly in sub-Saharan Africa relative to the world total



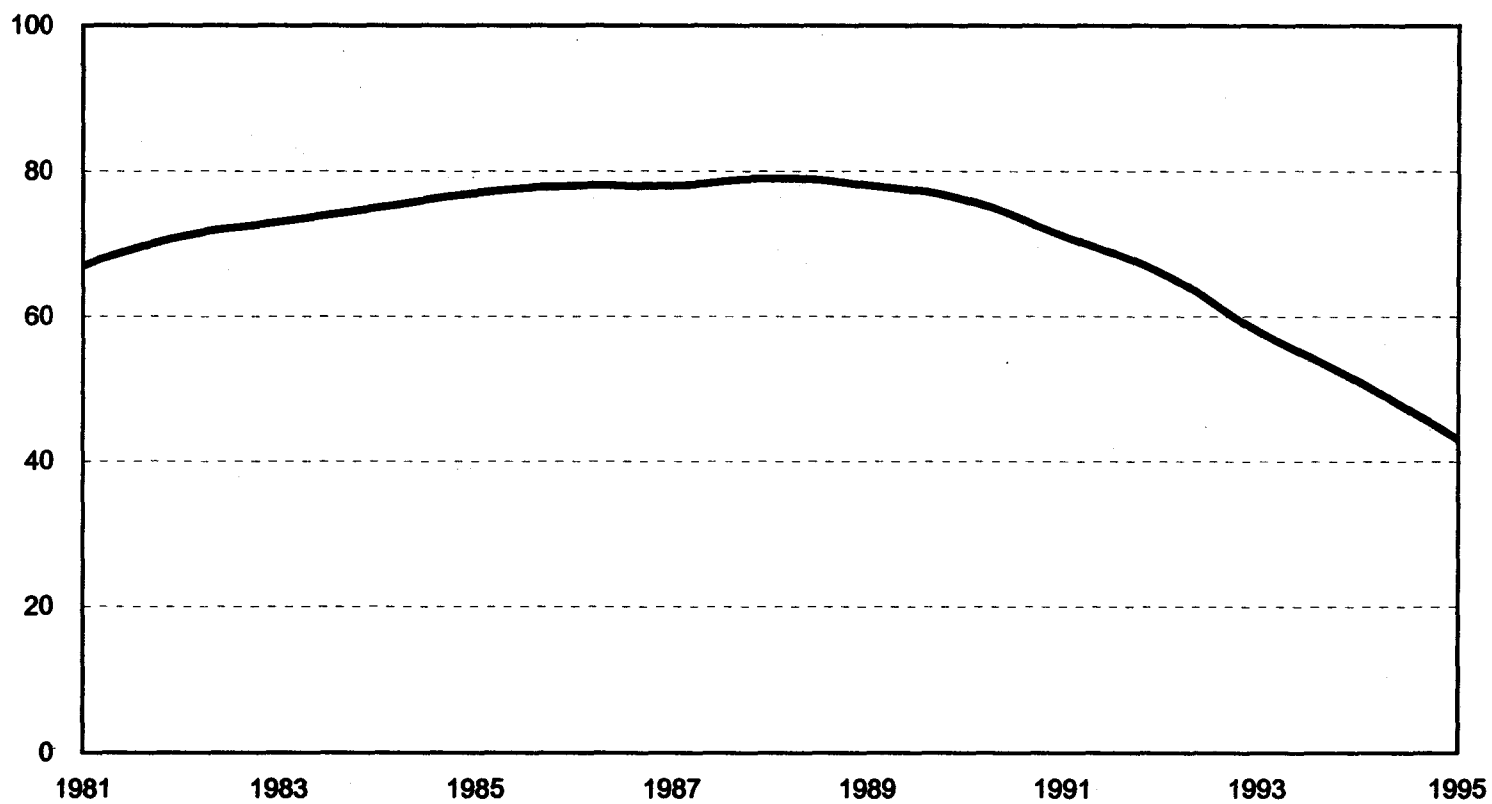
Source: Adapted from Jonathan Mann and D. Tarantola, eds., *AIDS in the World II: Global Dimensions, Social Roots and Responses* (New York, Oxford University Press, 1996, p. 12).

Figure 131. Cumulative adult HIV infections: sub-Saharan Africa relative to world total (1980-1996)



Source: Adapted from Jonathan Mann and D. Tarantola, eds., *AIDS in the World II: Global Dimensions, Social Roots and Responses* (New York, Oxford University Press, 1996, p. 12).

**Figure 132. Percentage of estimated global HIV infection occurring annually in sub-Saharan Africa**



*Source:* Adapted from Jonathan Mann and D. Tarantola, eds., *AIDS in the World II: Global Dimensions, Social Roots and Responses* (New York, Oxford University Press, 1996, p. 12).



(WHO) estimates that about 6.4 million people have died of AIDS since the epidemic began in 1981 and that 74 per cent of these deaths occurred in sub-Saharan Africa (World Health Organization, 1997). Harvard-based HIV/AIDS experts at the Center for Health and Human Rights estimate that AIDS-related cumulated deaths total 9.2 million world-wide as of January 1996, and that 82 per cent of these (7.6 million) occurred in sub-Saharan Africa (Mann and Tarantola, 1996). These estimates are substantially different from the number of AIDS cases reported to WHO (see figure 133). Most reported AIDS cases are from the United States, but it is believed that the figures from developing countries are highly underreported due to factors such as nonreporting, delayed reporting, and inadequate diagnosis. Therefore, these figures are adjusted upward on the basis of other available sources of information.

In discussing the prevalence of HIV infections and AIDS-related deaths in Africa, there are at least three aspects of the epidemic that should be kept in mind: (1) the heterogeneity of the HIV/AIDS epidemic in Africa, (2) the absence of quality data for cross-national comparisons, and (3) the role of models and their assumptions in understanding the course of the epidemic. Each of these factors is examined below.

### *1. Heterogeneity of HIV infection in Africa*

Africa is not a homogenous continent. Its people and cultures are as diverse as its geography. Similarly, the manifestations of the HIV/AIDS epidemic in Africa vary widely. First, there are regional (mainly North-South) differences, and the predominant type of the human immunodeficiency virus is not the same everywhere. Table 1 shows the estimated prevalence of HIV infection among adults by country. Most of the countries in North Africa have less than one adult per thousand infected with HIV. Similarly, the island countries of Mauritius and Comoros have very low prevalence rates. In West Africa, most of the countries have intermediate levels of infection (less than 30 adults in a thousand). The countries with the highest prevalence rates (with some exceptions) are geographically clustered in what has been called the "AIDS belt of sub-Saharan Africa". They are a chain of countries stretching from middle Africa through East Africa to

southern Africa (Caldwell and Caldwell, 1996) and include the Central African Republic, southern Sudan, Uganda, Kenya, Rwanda, Burundi, Tanzania, Zambia, Malawi, Zimbabwe and Botswana.

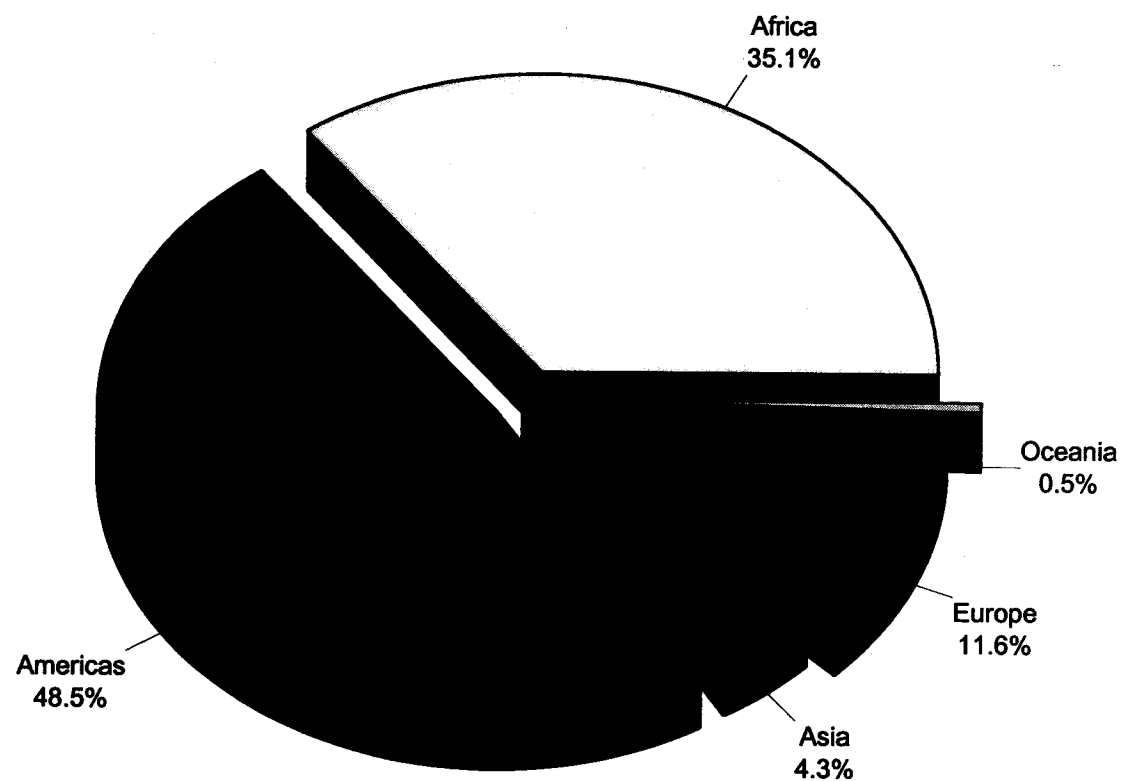
Factors that explain the spatial clustering of countries with high levels of HIV infection are not fully understood. Several hypotheses have been advanced to explain the phenomenon; these include the African origin of AIDS hypothesis; male circumcision hypothesis; chancroid sores hypothesis; abrasive heterosexual intercourse hypothesis; scarification instruments hypothesis; high prevalence of STDs hypothesis; and prolonged post-partum abstinence and delayed marriage hypothesis. Most of these hypotheses have been discarded for lack of supporting evidence. Only the hypotheses relating to the practice of circumcision and the prevalence of chancroid and STDs have some tenuous support from available data (Caldwell and Caldwell, 1996). Whatever the explanation for the clustering, estimates in table 80 shows that the six African countries with the highest HIV prevalence are all located in the AIDS belt. In fact, only three of the 15 countries with estimated HIV prevalence of at least 50 per 1,000 (table 80) are located outside the AIDS belt.

An important aspect of the distribution of HIV prevalence rates in sub-Saharan Africa is the presence, almost exclusively in West Africa, of another variant of HIV known as HIV-2 (Way and Stanecki, 1994; de Cock and Brun-Vezinet, 1996). The risk factors of both types are very similar and both result in AIDS. The major difference is that the incubation period for HIV-2 is longer than that for HIV-1 and vertical transmission of HIV-2 (i.e., from mother to child) is about 21 times lower than HIV-1. Even when vertical transmission occurs, infants infected with HIV-2 have a much higher survival rate. Because HIV-2 is not as risky or as widespread as HIV-1, more attention is usually given to HIV-1 and that will be the case in this paper.

### *2. Scarcity of quality data*

Lack of reliable data has been a major problem facing researchers seeking to understand the dynamics of HIV/AIDS in Africa. The near absence of necessary data has affected not only

**Figure 133. Cumulated AIDS cases reported to WHO, by region (June 1997)**



*Source:* World Health Organization, *Weekly Epidemiological Record*, vol. 72, No. 27, pp. 197-204 (Geneva, WHO, 1997b).

TABLE 80. HIV PREVALENCE RATE AMONG ADULTS (AGE 15-59 YEARS), AFRICA, END OF 1994

Rank	Country	Prevalence rate, per 1,000
1	Botswana	164.5
2	Zimbabwe	158.8
3	Zambia	156.6
4	Uganda	133.1
5	Malawi	122.8
6	Kenya	76.8
7	Togo	75.8
8	Rwanda	65.0
9	Congo	64.6
10	Ivory Coast	60.6
11	Burkina Faso	59.4
12	Tanzania	58.3
13	Namibia	57.5
14	Mozambique	51.4
15	Central African Republic	51.4
16	Swaziland	34.3
17	Zaire	33.6
18	South Africa	28.7
19	Eritrea	28.4
20	Guinea Bissau	27.6
21	Lesotho	27.2
22	Cameroon	27.0
23	Sierra Leone	26.7
24	Djibouti	26.7
25	Burundi	24.5
26	Chad	23.9
27	Ethiopia	22.4
28	Ghana	20.3
29	Nigeria	19.4
30	Gabon	19.3
31	The Gambia	18.9
32	Senegal	12.2
33	Liberia	11.9
34	Mali	11.4
35	Benin	10.7
36	Equatorial Guinea	10.2
37	Niger	9.5
38	Angola	9.3
39	Sudan	8.9
40	Mauritania	6.1
41	Guinea	5.4
42	Somalia	2.3
43	Comoros	0.8
44	Mauritius	0.7
45	Algeria	0.7
46	Libya	0.5
47	Tunisia	0.4
48	Réunion	0.4
49	Madagascar	0.4
50	Morocco	0.3
51	Egypt	0.2

Source: World Health Organization, *The Global Situation of AIDS*, 30 June 1996 (Geneva, WHO, 1996).

researchers, but also policy makers and funding agencies. Even the data that are available are often not of satisfactory quality. This is the conclusion of the US Bureau of Census, an organisation that has taken a special interest in monitoring available data on HIV prevalence in Africa using both published and unpublished sources. As of January 1997, the International Program Center of the US Bureau of the Census had collected in its HIV/AIDS Surveillance Data Base a total of 30,302 individual data records drawn from 3,782 publications and presentations (International Program Center, 1997). The following is a description of problems that have been identified in the existing data.

#### *Nonrepresentativeness*

Nonrepresentativeness is a major problem with African studies on HIV and AIDS. Many studies of HIV prevalence in Africa are based on specially selected categories of respondents, making it difficult to generalize to the larger population. National seroprevalence surveys are rare. Table 2 presents a list of studies regarded by the US Bureau of Census as being the best documented among those with reasonable sample sizes (International Program Center, 1997). The table presents the sample size, the risk group and sub-population studied, the year of the study, and the method of serology used. A few things are noticeable from the table. First, most of the samples are small; the largest samples are from The Gambia (29,670) and South Africa (18,630). Second, national estimates are rare; of the studies listed in table 81, only the samples from South Africa and Swaziland are national samples, and even these are pregnant women only. Third, subjects in the studies are usually not representative of the general population; the overwhelming majority of the studies focus on pregnant women, patients attending STD clinics, and prostitutes.

It is often argued that women attending antenatal clinics could represent the general population in terms of their seroprevalence levels (Cohen and Trussell, 1996, pp. 70). However, seroprevalence among pregnant women may differ from the general population: (1) on average, pregnant women are more sexually active than other women and so may have increased risk of infection; (2) pregnant women are on average younger than a sample of all women

and, given what we know about the age distribution of people infected with HIV, women of childbearing age are more concentrated in the age groups with the highest risk of HIV infection; (3) pregnant women are not likely to be using condoms for some months before conception or before they are seen at clinics; and (4) most pregnant women are currently married women so their patterns of infection are not representative of those of unmarried women (Mann and Tarantola, 1996, p. 487). It is possible for HIV prevalence rates among those attending an antenatal clinic to be lower than in the general population of adults, if women with HIV infection have lower pregnancy rates than women who are HIV-negative.

#### *Geographic bias*

Not only are the sample sizes small and often unrepresentative, an overwhelming majority of the existing seroprevalence studies in sub-Saharan Africa (see table 81) are based on samples taken from populations convenient for medical research teams drawing blood for testing. For this reason, many of the studies are facility-based. Such samples are self-selected, mostly urban-based, and not representative of the general population. Use of convenience samples creates the likelihood of geographic bias, in which results represent people who are more accessible than those in less accessible areas of a country. Given the higher rates of HIV infection in urban centres and high-traffic areas, the seroprevalence observed from these samples may be overestimated if the results are generalised to the whole country. The fact that most of the studies available are highly concentrated in the AIDS belt in sub-Saharan Africa creates the possibility of geographic bias in relation to the whole of Africa.

#### *Testing bias*

Testing bias relates especially to serologic tests that were done at the initial stage of the epidemic. It has been reported that testing techniques, especially early forms of the enzyme-based immunosorbent assay (ELISA) test (of which there are many), resulted in a number of false positives. Positive results of these early forms of ELISA must be confirmed by a second method, usually the Western Blot. Not all studies report confirmatory testing, although this has improved over time (International

TABLE 81. DETAILED LISTING OF ESTIMATES OF HIV-1 AND HIV-2 SEROPREVALENCE BY RESIDENCE AND RISK FACTORS, AFRICA, EARLY 1990S

Region/Country	Risk area	Geographic area	Year	Sub-population	Sex	Data type	Prevalence rate	Sample size	Test type
Algeria	UH	Not specified	1991	Prostitutes	F	HIV-1	0	na	UNK
Angola	UL	Luanda	1995	Pregnant women	F	HIV-1	1	695	ELISA, WB
	UL2	Luanda	1995	Pregnant women	F	HIV-2	0	695	ELISA, WB
	OL	Namibe province	1995	Pregnant women	F	HIV-1	0.5	420	ELISA, WB
	OL2	Namibe province	1995	Pregnant women	F	HIV-2	0	420	ELISA, WB
Benin	UL	Porto Novo	1993	Pregnant women	F	HIV-1	1.4	499	ELISA, WB
	UH	Cotonou/towns	1993-1994	Prostitutes	F	HIV-1	50.8*	366	UNK
	UH2	Cotonou/towns	1993-1994	Prostitutes	F	HIV-2	12.9*	366	UNK
	OL	Zou & mono/rural	1993	General population	B	HIV-1	4.9*	820	ELISA, WB
	OL	Zou & mono/rural	1993	General population	B	HIV-2	3.8*	820	ELISA, WB
Botswana	UL	Six sites	1995	Pregnant women	F	HIV	32.4	2 624	ELISA*2
	UH	Six sites	1995	STD patients	M	HIV	41.6	891	ELISA*2
	OL	Six sites	1994	Pregnant women	F	HIV	16	508	ELISA*2
Burkina Faso	UL	Bobo Dioulasso	1995	Pregnant women	F	HIV	12	824	ELISA, LIA
	UH	Ouaga & Bobo	1994	Prostitutes	F	HIV-1,2	60.4	424	ELISA, Rapid
Burundi	UL	Bujumbura	1992	Pregnant women	F	HIV	20	1 287	ELISA
	OL	Rural areas	1992	Pregnant women	F	HIV	1.8	1 231	ELISA
Cameroon	UL	Douala	1994	Pregnant women	F	HIV-1	5.7 °	599	UNK
	UL2	Yaounde	1994	Pregnant women	F	HIV-2	0	301	ELISA*3, Rapid
	UH	Douala	1992	Prostitutes	F	HIV-1,2	45.3	236	Rapid, WB
	OL	Manyemen (rural)	1991-1992	Pregnant women	F	HIV-1	2.9	382	ELISA, WB
	OL2	Manyemen (rural)	1991-1992	Pregnant women	F	HIV-2	0	382	ELISA, WB
	OH	Banka (semi rural)	1994	STD clinic pts	B	HIV-1	9	100	ELISA*3, Rapid
	OH2	Banka (semi rural)	1994	STD clinic pts	B	HIV-2	0	100	ELISA*3, Rapid
Central African Republic	UL	Bangui	1993	Pregnant women	F	HIV	16	218	UNK
	UH	Bangui	1993	STD clinic pts	B	HIV	31	283	UNK
	OL	Batangafu	1993	Pregnant women	F	HIV	6.5	200	UNK
	OL2	Berberati (rural)	1992	Adults		HIV-2	0	332	IFA, WB
Chad	UL	Sarh	1992	Pregnant women	F	HIV-1,2	4.1	435	Rapid, WB
Congo	UL	Brazzaville	1994	Pregnant women	F	HIV	7.1	600	UNK
	UH	Brazzaville	1990	STD patients	B	HIV	17.6	400	ELISA
	OL	Owando	1992	Pregnant women	F	HIV	2.6	300	UNK
Cote d'Ivoire	UL	Abidjan	1995-1996	Pregnant women	F	HIV-1	12.5*	2 559	UNK
	UL2	Abidjan	1995-1996	Pregnant women	F	HIV-2	2.0*	2 559	UNK
	UH	Abidjan	1992-1994	Prostitutes	F	HIV-1	77*	1 209	ELISA, LIA, WB
	UH2	Abidjan	1992-1994	Prostitutes	F	HIV-2	32.7*	1 209	ELISA, LIA, WB
Djibouti	UH	Djibouti	1991	Street prostitutes	F	HIV	43	300	ELISA, WB

TABLE 81 (continued)

Region/Country	Risk area	Geographic area	Year	Sub-population	Sex	Data type	Prevalence rate	Sample size	Test type
	UH2	Djibouti	1990	Street prostitutes	F	HIV-2	0	115	ELISA, WB
Egypt	UH	Alexandria	1992-1994	Blood donors	B	HIV-1,2	0	283	ELISA*2, WB
	UH	Cairo	1994	High risk people	B	HIV	5.3	150	ELISA
	OL	S. Sinai/rural	1994	Adults	B	HIV	0	467	ELISA
Eritrea	UL	Asmara	1991	Blood donors	B	HIV-1	1.6	516	ELISA*2, WB
Ethiopia	UL	Addis Ababa	1994	Adults	F	HIV-1	6.7	1 804	ELISA
	UH	Addis Ababa	1990	Prostitutes	F	HIV	54.2	1 225	WB
	OL	Jimma (rural)	1993-1994	Mothers	F	HIV	8.6	747	UNK
	OH	Nazareth	1991	Prostitutes	F	HIV	65.6	215	ELISA, WB
Gabon	UL	Libreville	1994	Pregnant women	F	HIV-1	1.7	1 684	ELISA, LIA/ WB
	UL2	Franceville	1993	Adults	B	HIV-2	0.8 *	674	ELISA, WB
The Gambia	UL	8 sites	1993-1995	Pregnant women	F	HIV-1	0.6 *	29 670	UNK
	UL2	8 sites	1993-1995	Pregnant women	F	HIV-2	1.2 *	29 670	UNK
	UH	3 towns	1993	Prostitutes	F	HIV-1	13.6 *	213	UNK
	UH2	3 towns	1993	Prostitutes	F	HIV-2	26.7 *	213	UNK
Ghana	UL	Kumasi	1995	Pregnant women	F	HIV-1	3.2 *	500	ELISA, Rapid, LIA
	UL2	Kumasi	1995	Pregnant women	F	HIV-2	0.4 *	500	ELISA, Rapid, LIA
	UH	Kumasi	1994	STD clinic pts	B	HIV-1	5.2 *	502	ELISA, Rapid, LIA
	UH2	Kumasi	1994	STD clinic pts	B	HIV-2	1.2 *	502	ELISA, Rapid, LIA
	OL	Nalerigu (semi rural)	1995	Pregnant women	F	HIV-1	1.0 *	500	ELISA, Rapid, LIA
	OL2	Nalerigu (semi rural)	1995	Pregnant women	F	HIV-2	0.2 *	500	ELISA, Rapid, LIA
Guinea	UL	Conakry	1991	Blood donors	B	HIV-1	0.7 *	2 003	ELISA, WB
	UL2	Conakry	1991	Blood donors	B	HIV-2	0.4 *	2 003	ELISA, WB
	UH	Conakry	1994	Prostitutes	F	HIV	36.6	112	WB
	OL	Samoe	1992	rural population	B	HIV-1	0.3	928	ELISA, Rapid, WB
	OL2	Samoe	1992	rural population	B	HIV-2	0.1	928	ELISA, Rapid, WB
Guinea	UL	Bissau	1995	Pregnant women	F	HIV-1	2.6 *	1 496	ELISA, WB
Bissau	UL2	Bissau	1995	Pregnant women	F	HIV-2	5.0 *	1 496	ELISA, WB
	OL	Rural area	1991	Adults	B	HIV-1	0.5 *	2 770	ELISA, LIA, WB
	OL2	Rural area	1991	Adults	B	HIV-2	8.3 *	2 770	ELISA, LIA, WB
Kenya	UL	Nairobi	1996	Pregnant women	F	HIV-1	18.1	210	INK
	UH	Nairobi	1992	Prostitutes	F	HIV	85.5	330	ELISA*2
	OL	Karurumo	1995	Pregnant women	F	HIV	10.3	322	UNK

TABLE 81 (continued)

Region/Country	Risk area	Geographic area	Year	Sub-population	Sex	Data type	Prevalence rate	Sample size	Test type
Lesotho	UL	Maseru	1993	Pregnant women	F	HIV	6.1	459	ELISA*3
	UH	Maseru	1993	STD pts.	B	HIV	11.1	377	ELISA*3
	OL	Maluti District	1993	Pregnant women	F	HIV	4.2	402	Rapid, ELISA
	OH	Maluti District	1993	STD pts.	B	HIV	21.3	221	Rapid, ELISA
Madagascar	UL	3 cities	1995	Pregnant women	F	HIV	0.1	1 587	ELISA, WB
	UH	3 cities	1995	STD pts.	B	HIV	0.3	1 575	ELISA, WB
	OL	Toliary prov.	1995	Pregnant women	F	HIV	0	500	UNK
	OH	Toliary prov.	1995	STD pts.	B	HIV	0.4	500	UNK
Malawi	UL	Blantyre	1995	Pregnant women	F	HIV	32.8	400	ELISA
	UH	Blantyre	1995	STD pts.	B	HIV	70.4	250	ELISA
	OL	Kasina rural	1995	Pregnant women	F	HIV	11.8	119	ELISA
Mali	UL	Bamako	1994	Pregnant women	F	HIV-1,2	4.4	205	ELISA, Rapid
	UH	Bamako	1995	Prostitutes	F	HIV-1,2	55.5	146	ELISA, Rapid
	OL	7 regions	1992	General pop.	F	HIV-1,2	3.4	2 990	LIA, WB
	OH	5 regions	1992	Prostitutes	F	HIV-1,2	52.8	178	LIA, WB
Mauritania	UL	Nouakchott	1993-1994	Pregnant women	F	HIV-1	0.5 *	1 106	ELISA*2, Rapid
	UL2	Nouakchott	1993-1994	Pregnant women	F	HIV-2	0.2 *	1 106	ELISA*2, Rapid
	UH	Nouakchott	1993-1994	STD pts.	F	HIV	0.9	430	ELISA*2, Rapid
Mauritius	UH	Not specified	1988-1991	STD clinic pts	B	HIV	0.8	1 470	ELISA, WB
Morocco	UL	Rabat	1993	Pregnant women	F	HIV-1	0.2	671	ELISA, WB
	UL2	Casablanca	1991	Pregnant women	F	HIV-2	0	300	ELISA, WB
	UH	3 cities	1996	STD pts.	M	HIV	1.4	223	ELISA, WB
Mozambique	UL	Maputo	1994	Pregnant women	F	HIV	2.7	800	ELISA, Rapid
	UH	Maputo	1995	STD pts	B	HIV	7.6 <sup>b</sup>	na	ELISA, Rapid
	OL	Zambezia	1992-1993	Displ preg. women	F	HIV-1	1.5 *	1 728	ELISA, WB
	OL2	Zambezia	1992-1993	Displ preg. women	F	HIV-2	0.5 *	1 728	ELISA, WB
Namibia	UL	6 urban sites	1996	Pregnant women	F	HIV	17.6	1 290	ELISA*2
	OL	4 rural sites	1996	Pregnant women	F	HIV	10.3	565	ELISA*2
Niger	UL	Niamey	1993	Pregnant women	F	HIV-1	1.3 *	400	ELISA, Rapid
	UL2	Niamey	1993	Pregnant women	F	HIV-2	0	400	ELISA, Rapid
	UH	Niamey	1993	Prostitutes	F	HIV-1	12.6 *	253	ELISA, LIA
	UH2	Niamey	1993	Prostitutes	F	HIV-2	6.3 *	253	ELISA, LIA
	OL	Tahoua reg (rural)	1992	Pregnant women	F	HIV	1.4	650	ELISA, WB
Nigeria	UL	Lagos	1993-1994	Pregnant women	F	HIV	6.7 <sup>c</sup>	1 894	ELISA*2 Rapid
	UH	Lagos	1993-1994	Prostitutes	F	HIV	29.1 <sup>c</sup>	292	ELISA*2 Rapid
Rwanda	UL	Kigali	1995	Pregnant women	F	HIV	25.4	500	Rapid
	UH	Kigali	1990-1992	STD pts	B	HIV	73.2	395	ELISA, LIA

TABLE 81 (continued)

Region/Country	Risk area	Geographic area	Year	Sub-population	Sex	Data type	Prevalence rate	Sample size	Test type
Senegal	UL	Dakar	1995	F.P. clinic attd	F	HIV-1	1.7 <sup>a</sup>	230	UNK
	UL2	Dakar	1995	F.P. clinic attd	F	HIV-2	0.4 <sup>a</sup>	230	UNK
	UH	Dakar region	1994	Prostitutes	F	HIV-1	10.1 <sup>a</sup>	347	ELISA, WB
	UH2	Dakar region	1994	Prostitutes	F	HIV-2	8.0 <sup>a</sup>	347	ELISA, WB
	OL	Ziguinchor reg	1994	Pregnant women	F	HIV-1	0.6 <sup>a</sup>	671	ELISA, WB
	OL2	Ziguinchor reg	1994	Pregnant women	F	HIV-2	1.0 <sup>a</sup>	671	ELISA, WB
	OH	Ziguinchor reg	1994	Prostitutes	F	HIV-1	9.2 <sup>a</sup>	141	ELISA, WB
	OH2	Ziguinchor reg	1994	Prostitutes	F	HIV-2	27.6 <sup>a</sup>	141	ELISA, WB
Sierra Leone	UH	Freetown	1995	Prostitutes	F	HIV	26.7	150	UNK
South Africa	UL	Natal/Kwazulu	1995	Pregnant women	F	HIV	18.2 <sup>b</sup>	na	UNK
	UH	Johannesburg	1994	STD pts.	B	HIV	20.1 <sup>c</sup>	4 395	UNK
	OL	National	1994	Pregnant women	F	HIV	6.4	18 630	ELISA*2, Rapid
Sudan	UL	Juba	1995	Pregnant women	F	HIV	3.0 <sup>b</sup>	na	UNK
Swaziland	UL	National	1993	Pregnant women	F	HIV	21.9 <sup>b</sup>	na	UNK
	UH	National	1992	Pregnant women	F	HIV	11.1	1 489	UNK
Tanzania	UL	Dar es Salaam	1995-1996	Pregnant women	F	HIV	13.7	6 525	ELISA, WB
	UH	Dar es Salaam	1993	High risk women	F	HIV	49.5	925	ELISA
	OL	Mbeya reg	1994	Pregnant women	F	HIV	15.0 <sup>b</sup>	na	UNK
	OH	Mbeya reg	1992	STD pts.	B	HIV	7.3	na	ELISA,*2, WB
Togo	UH	5 regions	1993	Military recruits	M	HIV	3.1	1 357	ELISA, WB
	OL	Dapaong	1993	Pregnant women	F	HIV-1	3	497	ELISA, WB
	OL2	Dapaong	1993	Pregnant women	F	HIV-2	0	497	ELISA, WB
	OH	Dapaong/rural	1993	STD pts.	M	HIV-1,2	7.3 <sup>b</sup>	na	ELISA, WB
Tunisia	UL	Tunis	1991	Pregnant women	F	HIV-1	0	1 030	ELISA, WB
Uganda	UL	Kampala	1995	Pregnant women	F	HIV	18.5 <sup>b</sup>	na	ELISA*2
	UH	Kampala	1995	STD pts.	F	HIV	38.5 <sup>b</sup>	na	ELISA*2
	OL	Karabole rural	1994	Pregnant women	F	HIV	6.5 <sup>b</sup>	na	UNK
Zaire	UL	Kinshasa	1992	Pregnant women	F	HIV-1	5.0 <sup>c</sup>	1 298	Rapid
	UH	Kinshasa	1995	Prostitutes	F	HIV	30.3	601	UNK
	OL	Musoshi	1991	Pregnant women	F	HIV-1	2.9	1 143	ELISA, WB
	OH	Haut-Zaire reg	1991	Prostitutes	F	HIV-1	25.4	126	ELISA, IFA/WB
Zambia	UL	Urban areas	1994	Pregnant women	F	HIV	27.9	5 320	ELISA
	UH	Lusaka	1992-1993	STD clinic pts	F	HIV	58	329	ELISA
	OL	rural areas	1994	Pregnant women	F	HIV	12.7	6 219	UNK
	OH	Mukinge	1990	STD pts.	B	HIV-1	36.0 <sup>b</sup>	na	ELISA
Zimbabwe	UL	Harare	1995	Pregnant women	F	HIV	32	2 783	ELISA*2
	UH	Harare	1994-1995	Prostitutes	F	HIV	86	147	ELISA
	OL	Manicaland	1993	Pregnant women	F	HIV	16.0 <sup>b</sup>	na	UNK
	OH	Murewa dist	1991-1993	STD clinic pts	M	HIV-1	46	215	ELISA, WB
	OH2	Murewa dist	1991-1993	STD clinic pts	M	HIV-2	0	215	ELISA, WB



TABLE 81 (continued)

Source: International Program Center, US Bureau of the Census, "Recent HIV Seroprevalence Levels by Country: January, 1997", *Research Note*, No. 23 (Washington D. C., 1997).

NOTES: UL = Urban low risk group, UH = urban high risk; OL = outside city low risk; OH = outside city high risk group; 2 = data refer to HIV2; STD = sexually transmitted diseases; M = male; F = female; B = both sexes.

\*Rates represent co-infection with HIV-1 and HIV-2 and should not be combined.

<sup>b</sup>Data are the best available but could be biased because of small sample.

<sup>c</sup>Data combined.

na = not available.

Program Center, 1997, p. 2). Another problem is that HIV-1 and HIV-2 may overlap, especially in countries where both are present and tested for. Testing will result in an underestimate if only HIV-1 infections are reported, and double counting may occur if HIV infection is reported as the sum of both HIV-1 and HIV-2 without adjustment for overlapping infection.

#### *Clinical case definition of AIDS*

A major source of bias that affects data from studies on AIDS in Africa is the use of different definitions of AIDS in different settings. The procedure for AIDS identification is not uniform. Prior to 1986, the diagnostic procedure accepted by WHO for surveillance purposes was the case definition for AIDS by the Centers for Disease Control and Prevention (CDC). However, the CDC definition was based on establishing an etiological diagnosis, and was difficult to use in countries with low diagnostic resources. So, WHO devised a provisional clinical case definition of AIDS without recourse to serologic testing. This Bangui definition for AIDS (named after the meeting that led to its formulation) became the standard for obtaining cases that were reported to WHO (see table 82). However, the WHO definition has been found to be inexact since organ systems can have similar responses to different pathogens and pathologies (Gilks, 1991). For example, persistent diarrhoea and weight loss may be associated with opportunistic infection as well as with ordinary enteric parasite or bacteria. As a result, many people who have no HIV infection can be classified as AIDS cases, as has been reported on occasions (Swai and others, 1989, p. 976; Chintu and others, 1993, p. 650). Take the case of paediatric AIDS; WHO developed a clinical paediatric AIDS definition in 1985 with a revision in 1989. The algorithm provided in the WHO clinical case definition of paediatric AIDS has been found inadequate for the diagnosis of the disease in Zambia (Chintu and others, 1993). It was found that the WHO criteria have a sensitivity of 64 per cent, speci-

ficity of 64 per cent, and positive predictive value of 37 per cent, while the corresponding figures for the Zambia criteria were 79 per cent, 91 per cent, and 87 per cent. Similar results have been found in other countries. Gilks (1991) found in Kenya that the WHO case definition of AIDS for adults had low positive predictive value (30 per cent). In Abidjan, the sensitivity was found to be about 35 per cent (De Cock and others, 1991).

One very illuminating study on how large the classification error could be in an African setting was conducted in Kigali (Leroy and others, 1995). The study enrolled a cohort of HIV-positive and HIV-negative women and followed them for a period of four years, observing how the diseases used for clinical definition of AIDS occurred in both groups of women. The results showed that both major and minor signs occurred in both groups, although the proportion was significantly higher in women who were HIV positive. The relative ratios of the incidence of these signs in the two groups were usually about 3:1 or less. For example, the incidence of weight loss of 10 percent or more was 12.6 per cent among HIV-positive and 5.9 per cent among HIV-negative women; generalised lymphadenopathy was 28.8 per cent among HIV-positive and 16.3 per cent among HIV-negative women; and chronic diarrhoea was 5.4 per cent among HIV-positive but 1.7 per cent among HIV-negative women. Despite this high probability of error in the WHO AIDS definition, cases have been documented of African doctors using it for clinical diagnosis rather than as an epidemiological surveillance tool. The initial objective of WHO was to use the case definition for surveillance purposes, but it is clear that this objective has been extended.

The World Health Organization has taken steps to address some of the criticisms. In 1994, WHO issued an expanded and adjusted algorithm for the clinical definition of AIDS among adolescents and adults; it is still being evaluated in developing countries around the

TABLE 82. TWO FORMS OF WHO CASE DEFINITIONS FOR AIDS SURVEILLANCE IN ADULTS AND ADOLESCENTS

### WHO Case Definition for AIDS Surveillance I (1993)

For the purposes of AIDS surveillance and adult or adolescent (>12 years of age) is considered to have AIDS if at least 2 major signs are present in combination with at least 1 of the minor signs listed below, and if those conditions are not known to be due to a condition unrelated to HIV infection.

#### Major signs

- weight loss of 10% or more of body weight
- chronic diarrhoea for more than 1 month
- prolonged fever for more than 1 month (intermittent or constant)

#### Minor signs

- persistent cough for more than 1 month (including TB)
- generalised pruritic dermatitis
- history of herpes zoster
- oropharyngeal candidiasis
- chronic progressive or disseminated herpes simplex infection
- generalised lymphadenopathy

The presence of either generalised Kaposi sarcoma or cryptococcal meningitis is sufficient for the diagnosis of AIDS for surveillance purposes.

### Expanded WHO Case Definition for AIDS Surveillance II (1993)

For the purposes of AIDS surveillance and adult or adolescent (>12 years of age) is considered to have AIDS if a test for HIV antibody gives a positive result and 1 or more of the following conditions are present:

- greater than or equal to 10% body weight loss or cachexia, with diarrhoea or fever, or both, intermittent or constant, for at least 1 month, not known to be due to a condition unrelated to HIV infection
- cryptococcal meningitis
- pulmonary or extra-pulmonary tuberculosis
- Kaposi sarcoma
- neurological impairment that is sufficient to prevent independent daily activities, not known to be due to a condition unrelated to HIV infection (for example, trauma or cerebrovascular accident)
- candidiasis of the oesophagus (which may be presumptuously diagnosed based on the presence of oral candidiasis accompanied by dysphagia)
- clinically diagnosed life threatening or recurrent episodes of pneumonia, with or without etiological confirmation
- invasive cervical cancer

Source: Adapted from World Health Organization, *Weekly Epidemiological Record*, vol. 37, no. 4, pp. 274 (Geneva, WHO, 1994).

world. Table 82 includes both these case definitions. Given the lack of laboratory facilities in most rural clinics and hospitals in Africa, and the expensive procedures necessary to identify AIDS cases that are available in developed countries, various built-in errors are possible in the reporting of AIDS cases. In many cases results have been published with these errors in place. Currently, there are multiple case definitions in existence that have varying degrees of accuracy. These include the CDC 1987, PAHO, European 1993, and CDC 1993 expanded case definitions. These

are in addition to the standard HIV serological testing.

The conclusion from this assessment is that pertinent and good quality data necessary for a thorough understanding of the HIV epidemic and for meaningful cross-national comparisons are inadequate in Africa. Standardisation of the existing data is also urgently needed. Until this situation is rectified, the use of models will continue to shape beliefs about the HIV/AIDS epidemic and the policies implemented to deal with it.

### 3. The role of models

The major role that mathematical and epidemiological models have played in our understanding of HIV/AIDS is, in part, a response to the absence of pertinent data. Lacking reliable national-level data on HIV prevalence and AIDS in most African countries, and given the pressing need to understand and monitor the trends and dynamics of the epidemic, the use of models was the only viable course. Examples of models that have been used to understand HIV/AIDS in Africa are the Epi Model developed by Chin and Lwanga (1991) for the World Health Organization Global Programme on AIDS, Bongaarts' (1988, 1995) model; Bulatao and Bos' (1992) model developed for the World Bank for understanding the demographic impact of AIDS; and the model of the Inter-agency Working Group (or iwgAIDS model) published by the US Bureau of the Census (Way and Stanecki, 1991; 1993). There are numerous other models available, each with its own unique assumptions. Unfortunately, many users of estimates from models forget that models are not reality, they only approximate reality (given valid assumptions, a good theory, and reliable input data). A model is only as good as its assumptions, but models are an ingenious way of getting insight into complex problems, such as the HIV/AIDS epidemic in Africa.

Problems arise when the necessary input data for a model are not available and can only be guessed at or estimated. A recent review of the type of behavioural data available in both developed and developing regions concludes that the data needs of those interested in modelling the spread of HIV have not been adequately met in most studies (Catania and others, 1996). If the estimated input data are themselves wrong, the model based on them will also produce biased results. As an illustration of how difficult it may be to get necessary input data, we examine what the requirements of two models: the iwgAIDS model and the World Bank (Bulatao and Bos) model.

#### *The iwgAIDS Model*

The model was developed by a team of researchers under the sponsorship of the US Department of State and requires many data

inputs that are not readily available. These include:

- estimates of the proportion "promiscuous" or having casual sexual contacts: the estimate imputed into the model is that 25 per cent of married men and 75 per cent of single men in urban areas are promiscuous in sub-Saharan Africa. The proportions are about 20 per cent for married women and 60 per cent for single women in rural areas.
- number of sexual contacts per partner: estimated to be 4 per month for single men and 1 per month for married extra-marital sexual contact.
- STD transmission rate per sexual contact (assumed to be 40 per cent), and the average duration of infection (assumed to be 2 months for urban male, 2.5 months for urban female, 2.5 months for rural males, and 3.5 for rural women).
- use of condoms, estimated to be 0.4 per cent

The model also requires information on transmission history using estimated population by infection status and risk group; rate of standard infectivity per sexual contact (estimated to be 0.003 for male to female and 0.0015 for female to male); relative HIV infection risk associated with STD infection (estimated to be +50 per cent for people with STD); rate of progression from HIV infection to AIDS (estimated to be 7.8 years for adults and 1.9 years for children); rate of progression from AIDS to death (estimated as 12 months for adults and children); and rate of perinatal transmission between mothers and infants (estimated to be 39 per cent). The pattern of seroprevalence by age for Africa was based on the Uganda National Serosurvey; the level was adjusted to match another estimate from WHO of the prevalence of HIV in sub-Saharan Africa. Because it is difficult to get reliable information for these estimates in the region, the possibility exists of multiple biases resulting from the estimated input data and the various assumptions of the model.

#### *Bulatao and Bos Model*

The model developed by Bulatao and Bos (1992) for the World Bank also requires input

data for the following parameters, most of which had to be estimated:

- male to female transmission rate (estimated to be 0.03, medium variant)
- female to male transmission rate (estimated to be 0.01, medium variant)
- sex acts involving condoms (estimated to be 10 per cent, medium variant)
- annual single men promiscuity with non-prostitutes (estimated to be 8 contacts, medium variant)
- annual single men promiscuity with prostitutes (estimated to be 24 contacts, medium variant)
- female prostitutes (estimated to be 143 contacts for medium variant)
- other promiscuous single female (estimated to be 12.6 contacts for medium variant)
- promiscuity of men with nonprostitutes (estimated to be 4.3 contacts for medium variant)
- promiscuity of men with prostitutes (estimated to be 2.1 contacts for medium variant)
- promiscuity of other promiscuous women (estimated to be 2.8 contacts for medium variant)
- per cent of adults seropositive 1985 (estimated to be 3.4 medium variant).

The model also requires some assumptions about the distribution of the population with regard to the proportion of sexually active people by marital status, the proportion of single women that are prostitutes, and the proportion of married men that engage in sexual promiscuity with prostitutes and with nonprostitutes. These data are difficult to obtain.

In any case, models have been useful for understanding the impact of various levels of HIV seroprevalence on mortality, fertility, and population size, and for understanding the possible effects of intervention programs. Models can serve as useful tools to assess the workability of various scenarios. Nevertheless they must be used continuously, with a clear understanding of their limitations.

## B. CONSEQUENCES OF HIV/AIDS IN AFRICA

In the early phases of HIV/AIDS research, there may have been many people who thought that the Malthusian prediction had caught up

with sub-Saharan Africa, and that HIV/AIDS would eventually do for the region what years of family planning program efforts could not—reduce population growth. Many observers anticipated that by the end of the decade, African populations would have fallen to levels below those reported in the late-1980s. This gloomy picture has not been realised, however. Demographic projections indicate that the impact of the epidemic on population growth may be much lower than was expected. At the same time, while there may be hope that Africa will be spared the “predicted” 10 years ago (Anarfi, 1994), the epidemic will have grave consequences, especially in countries with high HIV prevalence. The consequences are demographic, social, and economic.

### 1. Demographic consequences

The demographic consequences of AIDS in sub-Saharan Africa are of particular interest to demographers and people working in the area of reproductive health and family planning. This topic has been addressed by various organisations and many conferences have been called to discuss it. Because HIV/AIDS is primarily a health problem, much of its impact on population dynamics is expected to operate through mortality.

#### *Mortality impact*

There is as yet no cure for AIDS. All cases of HIV are likely to progress to AIDS and eventually to death from AIDS-related causes. In some African populations, AIDS has become the leading cause of adult deaths. The mortality impact of HIV/AIDS can be direct or indirect. The direct mortality impact results from HIV infection and related opportunistic infections that finally kill a seropositive individual. The indirect effect of HIV/AIDS on mortality involves deaths that are not directly a result of HIV infection but occur as associated consequences of HIV/AIDS. This section first addresses the direct mortality effect of HIV/AIDS and then discusses its indirect mortality impact.

The lack of a cure for AIDS suggests that the region with the highest levels of HIV prevalence—currently sub-Saharan Africa—would suffer the greatest mortality impact. Even without developing symptoms of AIDS, the risk of mortality among those who are HIV positive

is very high. Various studies have shown this to be the case in sub-Saharan Africa (Todd and others, 1997; Morgan and others, 1997; Mulder and others, 1994; Abouya and others, 1992). For example, a Ugandan study indicates that persons age 13-44 years who were infected with HIV-1 were 60 times more likely to die during a two-year observation period than those who were seronegative. Similarly, the rate of mortality among those aged 13-24 who were HIV positive was 87 times the rate among those who were seronegative (Mulder and others, 1994, p. 1022). In a five-year follow up, the age-standardised mortality rate was 157 per 1,000 person years for those who were seropositive, compared with 14 per 1,000 among those who were seronegative. In a rural area of Tanzania, it was observed that mortality among those who were seropositive was 94, compared with 6 among those who were seronegative after two years of observation (Todd and others, 1997). An obvious conclusion from these studies is that HIV infection increases mortality rates, by up to 50 per cent in some cases. In Kigali, Rwanda, Leroy and others (1995) found after a four-year follow up of a cohort of HIV-negative and HIV-positive women in a maternity ward that the mortality rate was 5 per 1000 women years among HIV-negative women and 44 among HIV-positive women. In the West African city of Abidjan, available data were analysed by Garenne and others (1996) and it was observed that trends in death rates for adults age 25-44 from 1973 to 1992 showed a major increase coinciding with the beginning of the HIV/AIDS epidemic in 1985 and continued to increase until the data was censored in 1992.

HIV/AIDS also has an indirect impact on mortality. For example, HIV-induced chronic morbidity and death of adult caretakers exposes young children to increased risk of death, even if they are not HIV positive. A three-year study in Kinshasa Municipal Hospital followed 335 newborn children of 327 seropositive women and the fathers of these children. There was also a control group of 341 newborn children of 337 seronegative women and the fathers of these children. It found that families in which the mother was HIV-1 seropositive experienced five- to tenfold higher maternal and early childhood mortality rates than families in which the mother was HIV-1 seronegative (Ryder and others, 1994). There are other avenues by which HIV/AIDS can increase mortality. They

include the vertical transmission of HIV from mother to child, resulting in the death of the child.

Unfortunately, the number of studies that focus on such topics is still small. Additionally, those that are available are small-scale and concentrated in the so-called AIDS belt. Therefore, it is difficult to generalize to the whole of Africa without the use of models. The iwgaIDS model projects into the future some of the patterns that may be induced by AIDS mortality in the region. The results with regards to mortality indicate that death rates in urban areas where AIDS is present would be more than double the level expected if AIDS were not present. The gains in mortality reduction that have been made over the last decade would be wiped out and the downward trend in under-five mortality would be reversed. The model indicates that by the year 2015, urban life expectancy would be reduced by 18 years from what it would have been without AIDS, and less than half of the population would survive up to age 45 in urban areas of sub-Saharan Africa.

#### *Impact on fertility and population growth rate*

HIV/AIDS may affect fertility through a number of avenues. One way is reduction in the number of men and women available in the prime childbearing ages. While it may be argued that many infected adults may be using condoms either because they are not willing to pass the virus on vertically (to their child) or horizontally (to their husband/partner), the fact that most infected people may not know they are infected makes such use of a contraceptive method a less likely avenue for a fertility reduction impact of HIV. However, those who already are HIV positive disease may be too ill to have frequent sexual intercourse or achieve a pregnancy. A recent study in a rural area of Uganda followed a cohort of women for six years and observed that the fertility of women with HIV infection was 20 per cent lower than that of women who were not HIV infected (Carpenter and others, 1997). The reduction in fertility was associated with STD coinfection. Another population-based cohort study in the same country was conducted by Gray and others (1998) between 1989 and 1992. It found that HIV infection reduced the odds of becoming pregnant by 55 per cent and that the odds ratio

of losing a pregnancy was 50 per cent higher in HIV positive women than those without an HIV infection. The study attributed the fertility-reduction effect of HIV-1 infection in the population to reduced rates of conception and increased prevalence of pregnancy loss among infected women.

Regarding population growth rates, model estimates have indicated that the effect of HIV and AIDS on population growth rates in sub-Saharan Africa will be, at best, moderate (Way and Stanek, 1991; 1993). The reduction in the growth rate is estimated to be less than one percentage point. This may not be devastating for a population growing at an annual rate of 3 per cent; however, it would be a threat if the population were growing at less than 1 per cent annually.

## *2. Economic consequences*

Economically, the effects of the HIV/AIDS epidemic will be felt at the national level, by businesses, and by individuals in their households. There is no consensus in the literature on how severe the macro level effects will be (Chevallier and Floury, 1996). Estimates from models and studies have found no significant change in the dependency ratio - persons under 15 years and 65 years and over to those aged 15-64 - because of AIDS (Bulatao and Bos, 1992; Nakiyingi and others, 1997). The same conclusion was reached by the model projection results for selected Africa countries prepared by the United Nations (United Nations, 1995). At the same time, various national-level models in Africa suggest that the morbidity and mortality impact of HIV/AIDS will reduce the growth rates of African economies (Whiteside, 1996). They suggest that HIV/AIDS affects economic growth through its impact on (1) human capital, and (2) on savings. In terms of human capital loss, the choice of people that HIV/AIDS affects in sub-Saharan Africa is disturbing. Available evidence suggests that HIV/AIDS kills a high proportion of people who are in the age group that is most economically productive, best able to give support to both older and younger family members, and most likely to contribute to the political and social lives of their societies. For example, the greatest mortality impact of HIV/AIDS is among those age 15 years and above not only in Africa but in other parts of the world (Mukiza-

Gapere and Ntozi, 1995; Garenne and others, 1996; Singh and others, 1996). In Tanzania, about 86 per cent of those who developed AIDS between 1987 and 1993 were age 20 and 49 (Ministry of Health, 1994, p. 16), and 91 per cent of those who died of AIDS in Muhimbili Medical Centre in Dar es Salaam were between 21 and 50 years of age (Mbagi and others, 1990). A study from Ghana found that AIDS sufferers were apparently more educated than their relatives, and that 77 per cent were age 20 to 29 years (Anarfi, 1995, p. 256). Another study in Malawi found a positive correlation between husband's educational level and pregnant wife's serologic status: the odds of a woman being HIV-1 positive if her husband had 8 or more years of schooling was 2.2 times the seroprevalence levels among women whose husbands had less than 8 years of schooling (Dallabetta and others, 1993).

The epidemic has also affected public and private businesses through reduction in productivity among workers, increased absenteeism, added costs of hiring and training replacement workers, time lost through attendance at burials, and many other indirect costs. Deaths of workers increase the cost of labour per unit of capital because the investment in those workers is lost. Given that AIDS is relatively more prevalent among educated persons, the loss of these persons to society can have a significant impact. Indirectly, care of sick people further reduces the productivity of workers who are healthy: they miss work because of the high demand on their time by sick relations and time spent attending funerals (Cohen and Trussell, 1996, p. 231).

There are few empirical studies directly focussed on the economic consequences of HIV/AIDS. The few that have been conducted often focus on encouraging the private sector to participate in funding AIDS prevention activities. One such study in Kenya (Forsythe and Roberts, 1995) found that the average cost of HIV/AIDS per annum to businesses was \$45 per employee in 1992 and was projected to increase to \$122 in the year 2005. The hardest hit businesses are those that are labour-intensive such as wood processing and sugar production. A typical company in Kenya was estimated to be losing about \$72,000 in 1992, projected to increase to about \$193,000 in 2005. The cost includes decreased revenues resulting

from absenteeism, funeral attendance, labour turnover, and labour lost during training. Another study in Zambia has assessed the impact of mortality among employees, but has not attached a monetary figure to the costs (Bagaley and others, 1994).

Other sectors that are affected by the AIDS epidemic include agriculture and food production, mining and extraction, the health sector, and the educational system. For example, the African economy is primarily agrarian; thus, the death of a substantial proportion of men in the most productive ages could result in the loss of vital farm labour, leading to a reduction in food availability, and its dire consequences.

### 3. *Social consequences*

There are many social consequences deriving from the HIV/AIDS epidemic in sub-Saharan Africa. For example, it threatens the survival of traditions such as widow inheritance and the levirate system. In cases where a surviving adult male is supposed to inherit the widow of a deceased brother, if HIV/AIDS is suspected in the death of the deceased husband, asking a younger brother to procreate through the widow is like asking him to receive a death sentence. This may lead to anger, bitterness, and intra-family fighting. In Uganda, it is reported that fear of AIDS has affected marriage patterns: many young people now fear getting married since they are not sure of the serostatus of the person they might want to marry. Others are fatalistic about it (Anarfi, 1994). AIDS deaths particularly affect young adults, many of whom have young children that are left behind. These children are now referred to as AIDS orphans. AIDS orphans also place additional responsibility on the extended family (Ryder and others, 1994). The increased number of AIDS orphans and widows place many social relationships under stress. A study in Uganda showed that being orphaned is associated with limited access to money, which usually limits access to education (Muller and Abbas, 1990).

### C. FUTURE TRENDS OF THE HIV/AIDS EPIDEMIC IN AFRICA

No one knows with any certainty what will happen in the future regarding the HIV/AIDS epidemic. The nature of the virus and its con-

trol continues to challenge researchers. There are at least ten sub-types of HIV-1, and the long period of incubation of HIV infection makes it difficult to identify and control. While it is possible that within a few years, human ingenuity and technological advances will devise ways to eliminate the virus and cure AIDS, it is unlikely for various reasons that such cure will become immediately available to infected people in poor countries. Therefore, the horrors of the AIDS epidemic may continue for many more years. That human behaviour underlies much of the transmission of HIV infection may be a reason for hope, given that human beings are adaptable and could avoid the types of behaviour that put their lives at risk. How human beings will react in the face of HIV may depend on many factors, and this makes it difficult to forecast the future of the infection.

From a theoretical standpoint, Mann and Tarantola (1996) expect that HIV/AIDS may have more than one peak, and the World Health Organization (1997, p. 20) talks about the possibility of an unexpected explosion of AIDS cases resulting from a natural disaster, war, or population displacement. The theoretical surmise of Mann and Tarantola divides people in a population into two broad groups: those that have high and those that have low vulnerability. In the early stages of the epidemic, people with high vulnerability are the first to be infected; the rate of infection is high and the increase is rapid. As the number of people in the high vulnerability group decreases, however, the epidemic reaches the first peak and may gradually decline. At the same time, the number of people in the low vulnerability group who are getting infected may increase, thereby pushing the epidemic to another peak and so on. Currently, there is little hard data to support such projections and surmises.

Results of various models (Bongaarts, 1995; United Nations, 1995; Way and Stanecki, 1993, 1994; Bulatao and Bos, 1992) suggest that HIV infection would increase sevenfold over a period of 25 years, and by 2015 there would be about 70 million cases in sub-Saharan Africa. The United Nations has examined the impact of AIDS on mortality in 15 sub-Saharan African countries where the adult HIV seroprevalence rate in 1992 was above 1 per cent (Benin, Burkina Faso, Burundi, Central African Republic, Congo, Côte d'Ivoire, Kenya, Malawi,

Mozambique, Rwanda, Uganda, United Republic of Tanzania, Zaire, Zambia, and Zimbabwe). The results of the projections with and without AIDS indicate that as of 1990-1995, AIDS has caused an estimated 1.7 million deaths in the 15 countries, and by the year 2000-2005, it will have caused an additional 4.3 million deaths (United Nations, 1995, p. 42). The past, present and projected numbers of deaths from AIDS in 15 countries of sub-Saharan Africa are presented in figure 134.

The results of the United Nations' projections are similar to those by Bongaarts (1995) and the US Bureau of the Census (Way and Stanecki, 1994). According to Bongaarts's projections, the number of AIDS deaths as a proportion of deaths from all causes will increase from 7.6 per cent in 1995 to 12.9 per cent in the year 2005. Bongaarts estimates that between 1995 and the year 2000, AIDS will cause about 620,000 deaths annually in sub-Saharan Africa. In light of the available evidence, many of these deaths will be adult deaths.

#### D. POLICY OPTIONS

Researchers have shown that responses to the HIV/AIDS epidemic have grown with the level of knowledge of the disease. The history of HIV/AIDS has been organised into three periods in the literature (see Mann and Tarantola, 1996): the discovery period (1981-1984), the early response period (1985-1988), and the current period (1989-present). Each period has distinguishable response categories. At the initial stage, the programmatic focus was on providing information about risk behaviours to stimulate individual behavioural change: reduce the number of sexual partners, or use condoms. At the second stage, the focus shifted to risk reduction, which comprised information and education, health and social services, and non-discrimination against HIV-infected people and those with AIDS. The focus was still on the individual and his/her behaviour. In the present stage, the focus is on societal dimensions of risk—widening the scope of relevant actors and of interventions to bring in social support networks to reduce HIV transmission, provide care and support, reduce vulnerability, and alleviate the impact of the disease. These are the goals identified by the new UN-wide agency UNAIDS (Piot, 1996). In looking ahead, the

options that should be pursued now are those that take a broader look at social systems, are multi-sectoral, and involve social institutions rather than isolating the HIV/AIDS victim for intervention. At least two types of policies are necessary: the first is aimed at reducing HIV infection through both heterosexual transmission and vertical (mother-child) transmission; the second is aimed at reducing AIDS mortality.

##### 1. *Reduction in HIV transmission*

###### *Promotion of condom use*

In the area of heterosexual transmission, a few options are available: one is to encourage the use of condoms; another is to treat STDs. Both of these will reduce the level of heterosexual transmission of HIV. There have been many AIDS-awareness campaigns in Africa aimed at increasing knowledge of AIDS and what can be done by individuals to reduce the risk of contracting the disease. Although many of these campaigns have been criticised as not being well planned (d'Cruz-Grote, 1996), it seems that many were effective in increasing the general knowledge of AIDS in the population. The next step in the process is to turn knowledge into behavioural response. There are many indications that people in sub-Saharan Africa are beginning to respond behaviourally to the epidemic. An example is the recent increase in condom sales and condom use; reports indicated that in Malawi, condom sales have increased from less than 15,000 per month in 1994 to more than 500,000 per month through social marketing programs. In Tanzania, condom sales have jumped from less than 10,000 per month in 1991, to 1,000,000 per month, currently; in Rwanda, sales have jumped from 35,000 per month in 1993 to over 200,000 per month, currently (PSI, 1997).

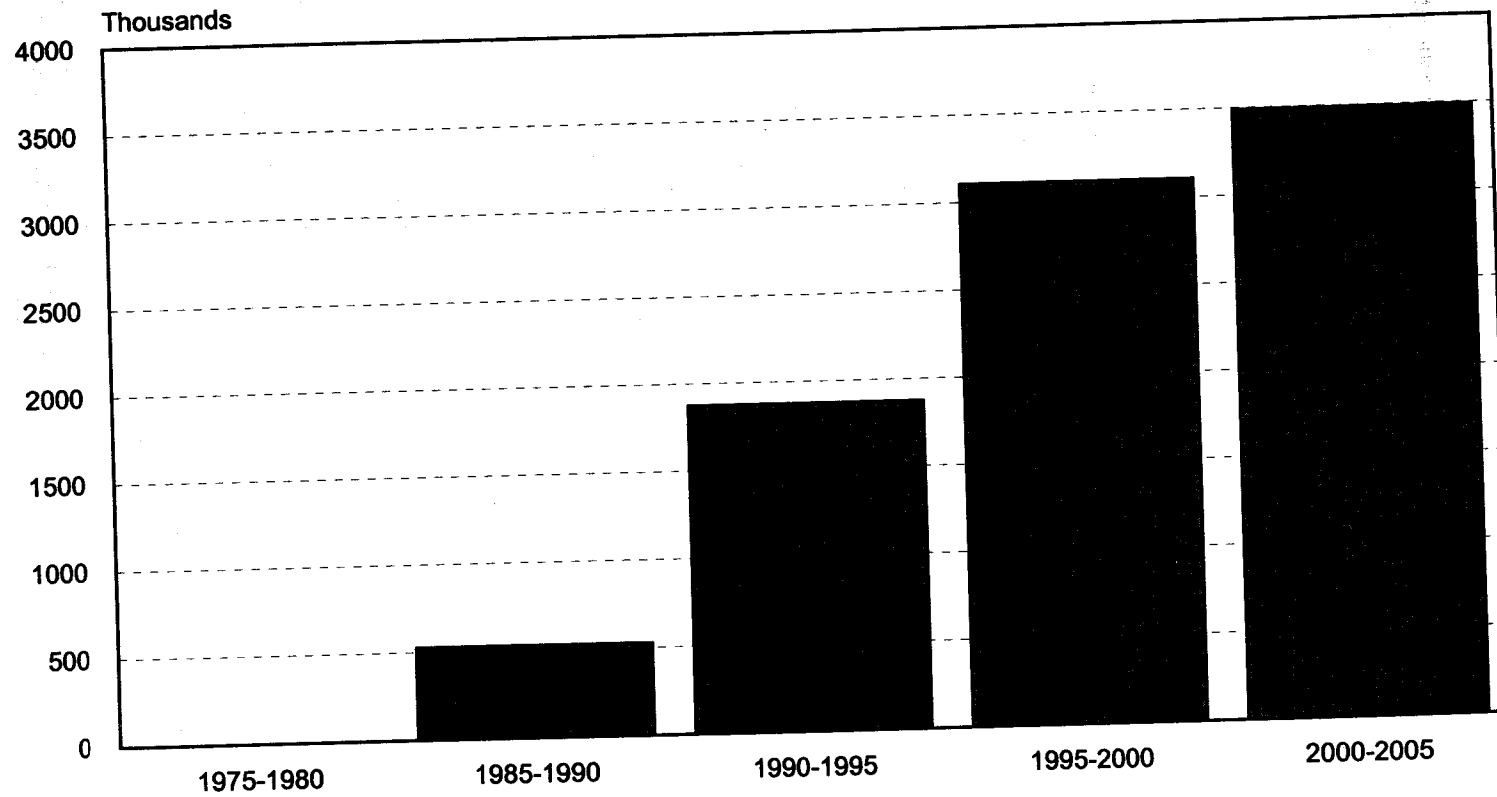
###### *Treatment of sexually transmitted diseases (STD)*

Treating STDs is another possible route to reducing heterosexual transmission of HIV. Model estimates suggest that this is a reasonable policy to pursue. For example, results of the simulation in the iwG AIDS model showed that a 20 per cent reduction in STDs would result in reduction of seroprevalence levels below current levels (Way and Stanecki, 1991; 1993).



**Figure 134. Projected number of deaths from AIDS in sub-Saharan Africa (1980-2005)**

A fifteen-country aggregate



Source: United Nations, *World Population Prospects: The 1994 Revision* (New York, Sales No. E.95.XIII.16, 1994, p. 26).

Evidence from Uganda, a country that in the early 1990s had one of the highest levels of HIV infection, suggests that treating STDs can result in a reduction in the prevalence of HIV. A Tanzanian study in the Mwanza region showed a 40 per cent reduction in new HIV cases though a comprehensive, public health STD-prevention program (World Health Organization, 1997, p. 21). Of course, increased use of condoms to prevent AIDS will have a synergistic effect on STD reduction. Similarly, co-infections of HIV, especially tuberculosis, need to be treated.

#### *Encourage reduction in casual sex*

Another intervention that can be promoted by deliberate policy is encouraging individuals to reduce the number of sexual partners they have. Many studies conducted in Africa indicate that women and men often have more than one sexual partner. In some cases, this is a survival strategy in the face of poor living conditions. This is particularly true of women who have few marketable skills and are looking for an extra source of income. There are also documented cases of schoolgirls and out-of-school adolescents in underprivileged and poor environments who trade sex for money. These individuals are in need of help, and that help is mainly in the form of means to afford the basic necessities of life. The most effective preventive measure in this case is one that effectively creates a supportive social and economic environment, makes information available, and at the same time provides the skills and confidence needed to take action. Many people in Africa who have contracted HIV are in a situation where they cannot help themselves because they lack socio-economic support. Commercial sex workers may risk not using a condom in the hope of earning more money; this is especially true when there is competition for customers. A young girl who is poor and wants to trade sex for income is not empowered to negotiate for condom use. Thus, availability and use of female controlled vaginal microbicide might become a substitute for a fundamental restructuring of the balance of power (Cohen and Trussell, 1996).

#### *Avoid procrastination*

Populations in which HIV prevalence is low have an advantage: they can act quickly to

prevent the spread of HIV/AIDS when cases are identified. Prevention in this case is vital because of the deadly nature of the disease. Failure to act quickly to curtail the spread of the disease may result in a high price to pay in future. African governments need to take control of their future in the search for solutions to the HIV/AIDS epidemic. Important targets for behaviour modification interventions are children who have not yet reach puberty, adolescents, and young adults who are not infected. These are the future generation and efforts should be made to spare them of infections that would make their lives short and miserable. Uganda is a country that has acted quickly to wage a war against the onslaught of HIV and AIDS and seems to be succeeding.

#### *Equip people with knowledge and skills*

Promotion of low-risk behaviour and development of risk-reduction skills (through life-skills education) are important ways to reduce the transmission of HIV/AIDS. Messages need to be specific, carefully targeted, culturally appropriate and locally relevant. Low-risk behaviours that can be promoted include partner reduction, postponement of the initiation of sexual activity, mutually faithful monogamy, and condom use (Cohen and Trussell, 1996). Continuously looking outward for support in the battle against HIV/AIDS is not realistic. Countries need to develop their own plans for effectively dealing with the epidemic. Greater action on the part of governments in developing countries is particularly important because of recent changes in policies at the global level, particularly reduction of donor assistance. Withdrawal has been blamed on donor fatigue arising from the realisation that the epidemic is not likely to affect the West and that the money that was expended by donors in sub-Saharan Africa has hardly influenced the course of the epidemic. Moreover, it is unreasonable to think that donors will continue to finance the care of patients in countries where the basic infrastructure for such care is lacking. The withdrawal of donor assistance may even help to gain a better picture of the actual situation in sub-Saharan Africa by removing the pressure to play up the numbers in order to attract attention and donor aid. However, withdrawal could hurt some countries in which donor aid is the only hope of relief for impoverished people and governments

that can ill afford the cost of care for AIDS patients.

Given the huge success that Africa has witnessed in increasing immunisation rates among children in the past decade, it would have been equally helpful if a vaccine against HIV had been developed. However, this solution still appears to be a long way off, first because of lack of adequate understanding of the role the immune system plays in protecting against HIV (there are as yet no patients who have recovered from HIV), and second because most of the people pressing for AIDS research are people who are already infected with the virus and favour research to identify drugs that will cure AIDS. Most of the uninfected population have not felt sufficiently threatened or at risk to push for research to produce a vaccine against the virus. Moreover, from the point of view of business interests, therapeutics are more profitable than vaccines. Individuals buy drugs, while governments purchase vaccines. The drugs that have already been developed are far too expensive for the majority of AIDS sufferers in sub-Saharan Africa. For example, using a therapeutic cocktail of protease inhibitors combined with other antiviral drugs to combat HIV may be effective in the early years of the infection, but it is a treatment that costs about \$20,000 per annum (Elmer-Derwit, 1996, pp. 54-55) and is too expensive for most of the estimated 21 million HIV carriers in sub-Saharan Africa.

## *2. Reduction in AIDS mortality*

The AIDS epidemic hit the countries of sub-Saharan Africa at a particularly bad time, when for many their economies were in the worst shape since independence. It was a time of structural adjustment when government subsidies on basic services such as health care were being drastically cut as a way to reduce government spending. Therefore, much of the burden of providing care for persons with HIV/AIDS has been borne by families; and many of these cannot afford the cost of health care. The lack of care for persons with HIV may contribute to the short interval between AIDS diagnosis and death.

One way to reduce AIDS mortality is to procure effective treatment of the opportunistic infections, the major one being tuberculosis

(TB). Evidence has shown that when opportunistic infections such as TB are treated, people who are HIV-positive live longer. Acceptance by others could also give people hope for living, while rejection leads to a desire to die, or denial of infection status and refusal to seek treatment. The new focus of UNAIDS on the societal approach is commendable and needs to be supported. In particular, help is needed for orphans and the elderly, both of whom suffer and die because of the indirect effects of AIDS.

## E. SUMMARY AND CONCLUSIONS

This paper has presented data on the prevalence and trends of HIV and AIDS in Africa. The consequences of the AIDS epidemic have been discussed and policy options that could be recommended for Africa in the face of possible future trends of the epidemic have been presented. Since much of what is known about the prevalence of HIV/AIDS and its impact at national and regional levels is based on models, considerable attention was paid to the quality of data and the assumptions that are fed into the models. This paper found that the database used to create HIV/AIDS models is still very limited, and there is need for more studies on both the prevalence and incidence of HIV/AIDS. The evidence reviewed in this paper suggests the need for improvement in the standardisation of information published by the World Health Organization, especially with regard to case definition of AIDS.

Available evidence suggests that HIV/AIDS will remain an important public health concern in many sub-Saharan Africa in the near future. Its mortality impact will reverse the gains that have been made in reducing under-five mortality in these high prevalence countries. However, its projected mortality and fertility impact, although high in many sub-Saharan African countries, is not sufficient to achieve zero-population growth rate in the region. Consequently, the population of sub-Saharan Africa will continue to grow in the near future. Because of the age groups where these mortality and morbidity effects are concentrated, HIV/AIDS is projected to result in a major loss of human capital. The policy options available to countries with high HIV prevalence rates include promoting low-risk behaviours, such as partner reduction and use of condoms, reducing

vertical transmission of the infection to children, treating and preventing sexually transmitted diseases, treatment of important co-infections such as TB, and providing some assistance to families with AIDS patients or AIDS orphans. These actions must be taken without delay. Efforts are also needed to improve the quality of data used to monitor and evaluate interventions in the region.

#### REFERENCES

- Abouya, Y.L., A. Beaumel, S. Lucas, A. Dago-Akribi, G. Coulibaly, M. N'Dhatz, J. B. Konan, A. Yapi, and Kevin de Cock (1992). Pneumocystis carinii pneumonia: an uncommon cause of death in Africa patients with Acquired Immunodeficiency Syndrome. *American Review of Respiratory Disease* (New York), vol. 145, pp. 617-620.
- Anarfi, John K. (1994). HIV/AIDS in Sub-Saharan Africa: Its Demographic and Socio-economic Implications. *African Population Paper*, No. 3 (December). Nairobi: African Population and Environment Centre.
- \_\_\_\_\_. (1995). The condition and care of AIDS victims in Ghana: AIDS sufferers and their relations. *Health Transition Review* (Canberra), vol. 5, *Supplement*.
- Baggaley, Rachel, Peter Godfrey-Fraussett, Roland Msiska, Diane Chilangwa, Eusabio Chitu, John Porter, and Michael Kelly (1994). Impact of HIV infections on Zambian businesses. *British Medical Journal*, vol. 309, pp. 1549-1550.
- Bongaarts, John (1995). Global Trends in AIDS Mortality. *Research Division Working Papers*, No. 80. New York: The Population Council.
- \_\_\_\_\_. (1988). Modelling the Spread of HIV and the Demographic Impact of AIDS in Africa. *Center for Policy Studies Working Papers*, No. 140 (October). New York: The Population Council.
- Bulatao, Rodolfo, and Eduard Bos (1992). Projecting the Demographic Impact of AIDS. *World Bank Policy Research Working Paper*, WPS 941 (August). Washington, D. C.: The World Bank.
- Caldwell, John, and Pat Caldwell (1996). The African AIDS epidemic. *Scientific American* (New York), March, pp. 62-68.
- Carpenter, Lucy, Jessica Nakiyingi, Anthony Ruberantwari, Samuel Malamba, Anatoli Kamali, James Whitworth, (1997). Estimates of the impact of HIV-1 infection on fertility in a rural Ugandan population cohort. In *The Socio-Demographic Impact of AIDS in Africa*. A collection of papers of a conference in Durban, February 3-6 Liege, International Union for the Scientific Study of Population.
- Catania, Joseph A., Judith T. Moskowitz, Monica Ruiz and John Cleland (1996). A review of national AIDS-related behavioural surveys. *AIDS* (Philadelphia, PA), vol. 10, *Supplement A*, pp. S183-S190.
- Chevallier, Eric, and Delphine Floury (1996). The socio-economic impact of AIDS in sub-Saharan Africa. *AIDS* (Philadelphia, PA), vol. 10, *Supplement A*, pp. S205-S211.
- Chin, J., and S. Lwanga (1991). Estimation and projection of adult AIDS cases: a simple epidemiological model. *Bulletin of the World Health Organization*, vol. 69, No. 4, pp. 399-406.
- Chintu, C., A. Malek, A. Nyumbu, C. Luo, J. Masona, H. L. DuPont, and A. Zumla (1993). Case definitions for paediatric AIDS cases: the Zambian experience. *International Journal of STD and AIDS*, vol. 4, pp. 83-85.
- Cohen, Barney, and James Trussell, eds. (1996). *Preventing and Mitigating AIDS in Sub-Saharan Africa. Panel on Data and Research Priorities for Arresting AIDS in Sub-Saharan Africa*. Washington, D. C.: National Academy Press.
- Dallabetta, Gina, Paolo G. Miotti, John D. Chipangwi, Alfred J. Saah, George Liomba, Nancy Odaka, Francis Sungani, and Ronald Hoover (1993). High socio-economic status is a risk factor for human immunodeficiency virus type 1 (HIV-1) infection but not for sexually transmitted diseases in women in Malawi: implications for HIV-1 control. *Journal of Infectious Diseases* (Chicago, IL), vol. 167, pp. 36-42.
- De Cock, Kevin, and Francoise Brun-Vezinet (1996). HIV-2 infection: current knowledge and uncertainties. In *AIDS in the World II: Global Dimensions, Social Roots and Responses*, Jonathan Mann and Daniel Tarantola, eds. New York: Oxford University Press, chapter 12, pp. 171-176.
- De Cock, Kevin, Bernard Barrere, M-F. Lafontaine, L. Diaby, E. Gnaore, D. Pantobe, and K. Odehouri (1991). Mortality trends in Abidjan, Cote d'Ivoire, 1983-1988. *AIDS* (Philadelphia PA), vol. 5, pp. 393-398.
- D'Cruz-Grote, Doris (1996). Prevention of HIV infection in developing countries. *Lancet*, vol. 348, pp. 1071-1074.
- Elmer-Derwitt, Philip (1996). Turning the tide. *Time* (New York), Special Double Issue, vol. 148, No. 29 (December 30), pp. 54-55.
- Forsythe, Steven, and Matthew Roberts (1995). The impact of HIV/AIDS on Kenya's commercial sector. *AIDSCaptions* (Family Health International, Research Triangle Park, NC), vol. 2, No. 1 (February), pp. 23-25.
- Garenne, Michel, Maria Madison, Daniel Tarantola, Benjamin Zanou, Joseph Aka, and Raymond Dogore (1996). Mortality impact of AIDS in Abidjan, 1986-1992. *AIDS* (Philadelphia, PA), vol. 19, pp. 1279-1286.
- Gilks, Charles F. (1991). What use is a clinical case definition for AIDS in Africa? *British Medical Journal*, vol. 303, pp. 1189-1190.
- Gray, Ronald H., Maria J. Wawer, David Serwadda, Nelson Sewakambo, Chuanjun Li, Frederick Wabwire-Mangen, Lynn Paxton, Noah Kiwanuka, Godfrey Kigozi, Joseph Konde-Lule, Thomas Quinn and Charlotte Gaydos (1998). Population-based study of fertility in women with HIV-1 infection in Uganda. *Lancet*, vol. 351, pp. 98-103.
- International Program Center, US Bureau of the Census (1997). Recent HIV Seroprevalence Levels by Country: January 1997. *Research Note*, No. 23. Washington, D.C.: International Programs Center, US Bureau of the Census.
- Leroy, Valentine, Philippe Msellati, Philippe Lepage, Jean Batungwanayo, Deo-Gratias Hitimana, Henri Taelman, Jos Bogaerts, Francoise Boineau, Philippe Van de Perre, Arlette Simonon, and Francois Dabis (1995). Four years of natural history of HIV-1 infections in African women: a prospective cohort study in Kigali (Rwanda), 1988-1993. *Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology* (New York), vol. 9, pp. 415-421.
- Mann, Jonathan, and Daniel Tarantola, eds. (1996). *AIDS in the World II: Global Dimensions, Social Roots and Responses*. New York: Oxford University Press.
- Mbaga, J.M., Pallangyo K.J., Bakari M. And Aris E.A. (1990). Survival time of patients with acquired immune-deficiency syndrome: experience with 274 patients in Dar es Salaam. *East African Medical Journal* (Nairobi), vol. 67, No. 2, pp. 95-99.
- Ministry of Health, Tanzania (1994). *Health Statistics Abstract 1994*. Dar es Salaam, Tanzania: Ministry of Health.
- Morgan, Dilys, Samuel Malamba, Gillian H. Maude, Martin Okongo, Hans-Ulrich Wagner, Daan Mulver, James Whitworth (1997). An HIV-1 natural history cohort and survival times in rural Uganda. *AIDS* (Philadelphia, PA), vol. 11, No. 5, pp. 633-640.

- Mukiza-Gapere, J and Ntozi, John P.M. (1995). Impact of AIDS on the family and mortality in Uganda. *Health Transition Review* (Canberra), vol. 5, Supplement.
- Muller, Olaf, and N. Abbas (1990). The impact of AIDS mortality on children's education in Kampala (Uganda). *AIDS Care* (Abingdon, Oxfordshire), vol. 2, No. 1, pp. 77-80.
- Mulder, Daan W., Andrew J Nunn, Anatoli Kamali, Jessica Nakiyingi, Hans-Ulrich Wagner, Jane Kengera-Kayondo, (1994). Two-year HIV-1-associated mortality in a Ugandan rural population. *Lancet*, vol. 343, pp. 1021-1023.
- Nakiyingi, Jessica, Samuel Malamba, Anatoli Kamali, Lucy Carpenter, Andrew Nunn, and James Whitworth (1997). Household composition and the HIV-1 epidemic in a rural Ugandan Population. In *The Socio-Demographic Impact of AIDS in Africa*. Collection of papers of a conference in Durban, February 3-6. Liege, International Union for the Scientific Study of Population.
- Pinching, Anthony J. (1996). AIDS research: solidarity? Rivalry? Fraternity? In *AIDS in the World II: Global Dimensions, Social Roots and Responses*. Jonathan Mann and Daniel Tarantola, eds. New York: Oxford University Press, chapter 18, pp. 205-211.
- Piot, Peter (1996). Why UNAIDS? In *AIDS in the World II: Global Dimensions, Social Roots and Responses*. Jonathan Mann and Daniel Tarantola, eds. New York: Oxford University Press, Box 34-1, pp. 370-371.
- Population Services International (PSI) (1997). *Revitalising Social Marketing Programs* (Washington DC). Profile, September.
- Ryder, Robert W., Malanda Nsuami, Wato Nsa, Munkolenkole Kamenga, Nsanga Badi, Mulenda Utshudi, and William Heyward (1994). Mortality in HIV-1 seropositive women, their spouses and their newlyborn children during 36 months of follow-up in Kinshasa, Zaire. *AIDS* (Philadelphia PA), vol. 8, pp. 667-672.
- Stanecki, Karen, and Peter Way (1996). The dynamic of HIV/AIDS pandemic. In *AIDS in the World II: Global Dimensions, Social Roots and Responses*. Jonathan Mann and Daniel Tarantola, eds. New York: Oxford University Press, chapter 2, pp. 41-56.
- Stein, Zena, and Mervyn Susser (1997). Editorial: AIDS - an update on the global dynamics. *American Journal of Public Health*, vol. 87, No. 6, pp. 901-904.
- Swai, A. B., PJ Lyimo, F. Rutayuga and D. McLarty (1989). Diabetes mellitus misdiagnosed as AIDS. *Lancet*, vol. 2, No. 8669, p. 976.
- Todd, James, Rebecca Balirra, Heiner Grosskurth, Philippe Mayaud, Frank Mosha, Gina ka-Gina, Arnoud Klokke, Reverianus Gabone, Awena Gavyole, David Mabey, Richard Hayes (1997). HIV-associated adult mortality in a rural Tanzanian population. *AIDS* (Philadelphia, PA), vol. 11, No. 6, pp. 801-807.
- Torrey, Barbara B., and Peter O. Way (1990). Seroprevalence of HIV in Africa: Winter 1990. *CIR Staff Paper*, No. 55 (May). Washington, D.C.: US Bureau of the Census.
- United Nations Program on HIV/AIDS (UNAIDS) (1997). *Report on the Global HIV/AIDS Epidemic*. UNAIDS/WHO Working Group on Global HIV/AIDS and STD Surveillance. Geneva: UNAIDS/WHO. Document retrieved from the Internet (<http://www.unaids.org/highband/document/epidemio/report97.html>).
- United Nations (1995). *World Population Prospects: The 1994 Revision*. New York: United Nations. Sales No. E.95.XIII.16.
- Way, Peter and Karen Stanecki (1991). The Demographic Impact of an AIDS Epidemic on an African Country: Application of the iwgAIDS Model. *Bureau of the Census, Center for International Research Staff*. Working Paper No. 58. Washington, D.C.: Center for International Research, US Bureau of the Census.
- \_\_\_\_\_ (1993). How bad will it be? Modelling the AIDS epidemic in Eastern Africa. *Population and Environment* (New York), vol. 14, No. 3, pp. 265-278.
- \_\_\_\_\_ (1994). *The Impact of HIV/AIDS on World Population*. Washington, D.C.: US Bureau of the Census.
- Whiteside, Alan (1996). Economic impact in selected countries and the sectoral impact, in *AIDS in the World II: Global Dimensions, Social Roots and Responses*. Jonathan Mann and Daniel Tarantola, eds. New York: Oxford University Press, chapter 7, pp. 110-116.
- World Health Organization (WHO) (1986). *Weekly Epidemiological Record* (Geneva), vol. 61, pp. 361-368.
- \_\_\_\_\_ (1994). *Weekly Epidemiological Record* (Geneva), vol. 37, No.4, pp. 273-280.
- \_\_\_\_\_ (1997). *Weekly Epidemiological Record* (Geneva), vol. 72, No. 4, pp. 17-24.
- \_\_\_\_\_ (1997b). *Weekly Epidemiological Record* (Geneva), vol. 72, No. 27, pp. 197-204.

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