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DEPARTMENT OF INTERNATIONAL ECONOMIC AND SOCIAL AFFAIRS

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PREFACE

The *Population Bulletin of the United Nations* presents brief articles relating to population which, by their nature, do not require separate publication. Material for the *Bulletin* is selected in the light of the interests and needs of Governments, international organizations, research institutions and individuals engaged in social and economic research, as well as the public interested in population.

The first seven issues of the *Population Bulletin* were prepared by the Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat between 1951 and 1963. In accordance with the endorsement and recommendation of the Population Commission at its eighteenth session, the *Bulletin* was reinstated as a United Nations publication, beginning with the publication of *Bulletin* No. 8 in 1977. As in the past, the *Bulletin* is prepared by the Population Division.

Most of the articles published in the *Bulletin* are prepared by the United Nations Secretariat in pursuance of the programme of work recommended by the Economic and Social Council and the Population Commission. Studies by the consultants and reports of meetings organized by the United Nations, or excerpts from such studies and reports, may also be included. In addition, contributions are solicited from the specialized agencies of the United Nations, the secretariats of the regional commissions and scholars.

Explanatory notes

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

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

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

A blank in a table indicates that the item is not applicable.

A minus sign (–) indicates a deficit or decrease, except as indicated.

A full stop (.) is used to indicate decimals.

A slash (/) indicates a crop year or financial year, e.g., 1970/71.

Use of a hyphen (-) between dates representing years, e.g., 1971-1973, signifies the full period involved, including the beginning and end years.

Reference to "tons" indicates metric tons, and to "dollars" ($) United States dollars, unless otherwise stated.

Annual rates of growth or change, unless otherwise stated, refer to annual compound rates.

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

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A COMPARISON OF THE WORLD POPULATION PLAN OF ACTION AND THE RECOMMENDATIONS FOR THE FURTHER IMPLEMENTATION OF THE WORLD POPULATION PLAN OF ACTION

*United Nations Secretariat**

SUMMARY

In the present paper, a substantive comparison is made of the World Population Plan of Action, adopted by the United Nations World Population Conference, held at Bucharest in 1974, and the recommendations for the further implementation of the Plan, adopted by the International Conference on Population, held at Mexico City in 1984. Among the changes discussed are the greater prominence given in the recommendations to socio-economic development, the environment and population, and the role and status of women and population. In addition, the 1984 recommendations deal with morbidity and mortality in a far more detailed and systematic manner and discuss the right of individuals and couples to decide freely and responsibly the number and spacing of their children with greater strength and urgency. Furthermore, they are more specific regarding migration and changing population structures.

INTRODUCTION

In 1974 the United Nations World Population Conference, held at Bucharest, adopted the World Population Plan of Action (WPPA).[1] Ten years later, the International Conference on Population, held at Mexico City, adopted recommendations for the further implementation of the WPPA (hereinafter referred to as the "Mexico City document").[2] The amount of progress made in the years between the Conferences is a matter of great concern, and understandably so. In the mind of many a reader, a comparison of the Plan and the recommendations—that is to say, of the WPPA and the Mexico City document—should provide a basis for assessing such progress.

However, a comparison of the two is made difficult by the fact that they are not of equivalent nature and stature. While the WPPA was meant to stand on its own, and therefore contained a detailed exposition of its background, principles and objectives, the Mexico City document was conceived as an instrument that would complement and further refine the WPPA, based on the explicit acceptance of the continued full validity of the principles and objectives of the WPPA. Thus, not every apparent omission in the Mexico City document can be interpreted as implying a change of attitude since Bucharest. Since there are no explicit disavowals, the comparison can only be based on what has been added, and the exercise therefore risks being unbalanced.

Given the limitations of the comparative approach, the present discussion focuses on the explicit changes, rather than on nuances and subtle shifts of emphasis. It is also beyond the scope of the discussion to explain the negotiation process by which the Conferences arrived at the formulations that were finally adopted, except where such information is essential to clarify the substantive content of a given formulation.

SOCIO-ECONOMIC DEVELOPMENT, THE ENVIRONMENT AND POPULATION

In what constitutes a major departure from the organization of the WPPA, the Mexico City document opens its recommendations for action with a section on socio-economic development, the environment and population. The new prominence clearly highlights the proposition that "social and economic development is a central factor in the solution of population and interrelated problems"—a principle already set forth in the Plan, but in a place of much less visibility (paras. 68-70). That area benefited from a decade of substantive debate in various forums. The objectives of the International Development Strategy for the Third United Nations Development Decade updated the corresponding reference in the WPPA to the International Development Strategy for the Second Development Decade and to the Programme of Action on the Establishment of a New International Economic Order. Concerning the issues of environment, the Mexico City document explicitly recommends that Governments should "implement appropriate policy measures to

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avoid the further destruction of the ecological equilibria and to take measures to restore them" (recommendation 4), whereas the WPPA makes a less focused reference to the imperative that "all countries . . . should adapt themselves to more rational utilization of natural resources, without excess, so that some are not deprived of what others waste" (para. 70).

Both documents make specific recommendations regarding the means to foster development, thereby offering interesting insights into the spirit of their times. The WPPA recommends that development plans emphasize the health and education sectors; the adoption of labour-intensive technologies is implicitly advocated; emphasis is placed on improved food production and distribution, international co-operation being specifically recommended "with the aim of ensuring the provision of fertilizers and energy and a timely supply of food-stuffs to all countries" (para. 69). The Mexico City document offers a quite different set of prescriptions: lower barriers to trade, increased international assistance that should also be improved in terms of quality and effectiveness, increased earnings from the export of commodities, increased volume of international lending at improved terms, encouragement of entrepreneurial initiatives, increased productive investment in appropriate industries and in rural and agricultural development (recommendation 3). On that issue, three industrialized countries, the Union of Soviet Socialist Republics, the Ukrainian Soviet Socialist Republic and the United States of America, while joining the consensus, made statements clarifying their views on specific points in the recommendation. (The two former countries stated that they could not accept the unbalanced wording of the recommendation, which implied an underestimation of the role that the State sector is playing in socio-economic development as reflected in relevant United Nations documents; the latter country stated that its endorsement did not change its known positions on commodity agreements or future lending resources for international financial institutions.) From a more technical point of view, the principal recommendation states that national development policies, plans and programmes as well as international development strategies should be formulated on the basis of an integrated approach that takes into account the interrelationships between population, resources, environment and development. The Mexico City conference, however, did not offer more specific guidance concerning the scope of the integrated approach and the substantive content of the interrelationships.

The role and status of women

The sharp focus and the heightened emphasis that were brought to women's issues in relation to population were among the most visible accomplishments of the Mexico City conference. In another major departure from the structure of the WPPA, the Mexico City document grouped a series of recommendations on the role and status of women in a separate section, giving that subject a far more prominent place near the beginning of the document. In addition, women's concerns were made systematically explicit in virtually every other substantive section of the document.

In the WPPA, the principle of equality of men and women was affirmed, discrimination was rejected, and the promotion of the status of women was placed among the objectives of the Plan. However, the recommendations on the status of women were combined in a single section of the document along with other recommendations on reproduction and the family, so that the importance of the role and status of women in other contexts, although recognized, was given far less visibility. The language of the Mexico City document is in that respect stronger and more specific than that of the WPPA; it conveys a greater sense of urgency, particularly in contrast to its more generally measured tone. Governments are, for instance, asked to "pursue more aggressively action programmes aimed at improving and protecting the legal rights and status of women" (recommendation 5). In another recommendation (6), the right of women to participate or not in the labour force is placed above considerations of demographic policy or cultural tradition. It is also stated that the biological role of women in the reproductive process should in no way be used as a reason for limiting women's right to work. In the Mexico City document there is perhaps a more clear indication that Governments should provide *both* due support to the important social role of women as mothers and opportunities for personal fulfilment in both familial and non-familial roles.

In the area of general legislation regarding the status of women, although the Mexico City document strongly urges Governments to sign and ratify or accede to the Convention on the Elimination of All Forms of Discrimination Against Women, the WPPA makes more specific demands: Governments should make a sustained effort not only to ensure that legislation regarding the status of women complies with the principles of the Declaration on the Elimination of Discrimination against Women and other instruments, but also "to reduce the gap between law and practice through effective implementation, and to inform women at all socio-economic levels of their legal rights and responsibilities" (para. 40(*d*)). On the other hand, the Mexico City document puts more emphasis than the WPPA on the sharing of family responsibilities by both partners, requesting Governments to promote and encourage through various means, including employment legislation and institutional support, "the active involvement of men in all areas of family responsibility, including family planning, child-rearing and housework" (recommendation 9). Another example: while the WPPA makes reference to "the establishment of an appropriate lower limit for age at marriage" (para. 32(*f*)), which, in the content of development goals, would tend to moderate fertility levels, the Mexico City document recommends that "Governments concerned should make efforts to raise the age of entry into marriage in countries in which

this age at marriage is still quite low (recommendation 8). In general, the pattern of change from the Bucharest Plan to the Mexico City recommendations in that area was to move from broad principle to specific measures.

Development of population policies

The section of the Mexico City document on population policies was originally an attempt at sharpening the focus of several passages of the WPPA concerning the formulation and implementation of such policies, by grouping some important general principles and recommendations in a separate section. The development of population policies is indeed an area in which a great deal of experience has been gained throughout the world during the past decade and it was generally felt that it would be appropriate for the recommendations of the Mexico City conference to reflect that experience. However, the change from the WPPA to the Mexico City document tended to be from a comparatively specific recommendation to a more general statement of principle, reiterating the pattern found in other areas.

In the WPPA, the subsection titled "Development and evaluation of population policies" has a technical focus: it calls for the evaluation of policies and programmes and recommends that organizational arrangements in national administrative structures reflect the integration of population and socio-economic planning. It is also located near the end of the Plan, in the section that also deals with statistics, research and programme management. In the Mexico City document, Governments are urged to adopt population and socio-economic policies that are mutually reinforcing. That proposal echoes several passages of the WPPA, including the stronger principle that population policies should not be considered substitutes for socio-economic development policies but rather should be integrated components of those policies. A principle that emerges strongly from the Mexico City document is the desirability of ensuring community participation: policies should be responsive to local values and needs and those persons directly affected should be involved in the decision process at all levels. The notion of promoting popular participation in the formulation and implementation of population policies is also to be found in the WPPA, but only as an objective of the Plan, and not as an operational means by which any population goals may be achieved. The Mexico City document also calls for special emphasis to be given to the linkages between population trends and various employment issues; apart from that mention of the topic, no further comment or recommendations are made. Finally, the Mexico City document recommends that population policies should be formulated with particular attention to the individual, the family and community levels, by implication calling attention to the importance of concerns felt at levels other than that of the national Government.

Population growth

The section of the Mexico City document dealing specifically with population growth contains only one recommendation, inviting Governments to consider pursuing relevant demographic policies, within the framework of socio-economic development, when population growth rates are perceived as an obstacle to the attainment of national goals. That portion of recommendation 13 is actually a paraphrase of paragraph 17 of the WPPA; it should be noted, however, that although the WPPA calls for policies "consistent with basic human rights and national goals and values", the Mexico document calls for such policies to respect "human rights, the religious beliefs, philosophical convictions, cultural values and fundamental rights of each individual and couple, to determine the size of its own family". That attempt to delineate the limits of population policies is not matched by any similarly detailed attempt to define the contents of such policies. In particular, no reference was made to the use of quantitative targets as an adjunct to population or socio-economic development policies. It is important to note that the original draft recommendation placed before the 1980 Conference had invited countries which considered their population growth rate to be detrimental to their national purposes to consider setting quantitative population growth targets. Therefore, the Conference rejected the reference to quantitative targets and also specified a range of factors to be respected in the pursuit of any demographic policies intended to influence population growth rates.

The WPPA, in paragraph 18, offered policy suggestions to countries that sought to achieve moderate or low population growth as well as to countries wishing to increase their rate of population growth. The former were encouraged to try to achieve their goals through a low level of birth and death rates, while the latter were invited to concentrate efforts on the reduction of mortality or, where appropriate, to increase levels of fertility and immigration. Such suggestions in the context of population growth are absent from the Mexico City document. At the same time, both the annual increments in world population and the rate of population growth, particularly in developing countries, are described in the Preamble as being "among the major challenges . . . in the area of population that are of primary concern to the international community" (para. 10); it is also observed that fertility rates remain "substantially higher or lower than those desired by Governments and peoples in some countries" (para. 10 (*g*)).

In the treatment of population growth issues, the Mexico City conference did not reopen the discussion on the idea that developed countries should adopt policies on population, consumption and investment based on the recognition that "per capita use of world resources is much higher in the developed than in the developing countries" (para. 19), as urged by the WPPA. Environmental concerns in relation to population are addressed in the Mexico City document at the

level of individual countries where there are imbalances between population growth, resources and environmental requirements, rather than at the global level, and, although population policies are mentioned, emphasis is on the promotion of "improved methods of identifying, extracting, renewing, utilizing and conserving natural resources" (recommendation 4). In the pragmatic tone of the Mexico City document, the objective is to redress such imbalances, whereas the WPPA urged the developed countries to bear in mind "the need for fundamental improvement in international equity" (para. 19) when adopting policies in population, consumption and investment.

Morbidity and mortality

The coverage of morbidity and mortality issues in the Mexico City document is far more detailed and systematic than in the WPPA. Recommendations are presented in three groups, dealing separately with goals and general guidance for health policies, with issues of infant, child and maternal morbidity and mortality and, finally, with issues of adult morbidity and mortality. In addition, specific targets of mortality reduction at year 2000 are set in terms of expectation of life and infant mortality rates for countries that are currently experiencing higher, intermediate and lower mortality levels. A quantitative goal is also set for the reduction of maternal mortality, in countries where the rate exceeds a specified level. In the WPPA, goals were set only in terms of expectation of life and infant mortality rates for the year 1985 and only for countries with the highest mortality levels.

The Mexico City document reflects a number of ideas and concerns that gained wide recognition after the International Conference on Primary Health Care[3] in 1978. Whereas the WPPA called for the integration of "health and nutrition programmes . . . within a comprehensive development strategy . . . supplemented by a wide range of mutually supporting social policy measures" (para. 25), the Mexico City document goes much further in its assertion that "the promotion and preservation of health should be the explicit concern of all levels and branches of government" and its strong urging that "governmental action in the area of mortality . . . should go beyond the health sector and involve all relevant sectors of national and community development"; more specifically, it is recommended that "all development programmes should be monitored and analysed . . . in order to assess and to improve their impact on health" (recommendation 16).

One of the characteristic aspects of the Mexico City recommendations in the area of morbidity and mortality is the emphasis placed on community participation. The community should be involved "in all possible ways in the planning, implementation and evaluation of health improvement programmes" (recommendation 15) and a wide array of organizations—governmental, intergovernmental, parliamentary and non-governmental—is requested to contribute to that goal. The education and mobilization of the community is referred to frequently in the recommendations on mortality and morbidity, while the WPPA does not mention the subject at all in that context.

Among the more specific recommendations on "post Alma-Ata" themes, Governments are urged to promote and support breast-feeding and to expand the use of certain techniques such as child growth monitoring, oral rehydration therapy, immunization and appropriate birth spacing (recommendations 19 and 20), in line with the "child-survival revolution" strategy advocated in recent years by the United Nations Children's Fund (UNICEF). At the same time, the International Code of Marketing of Breast-Milk Substitutes, adopted by the World Health Assembly, received measured support: "Governments which have accepted it should be urged to take the necessary steps to implement" the International Code.

In the WPPA as well as in the Mexico City document, the issue of abortion is addressed exclusively in terms of mortality and morbidity. In the WPPA, the reduction of illegal abortions is listed among the goals that call for particularly vigorous efforts, together with the reduction of involuntary sterility, subfecundity and defective births. The Mexico City document goes further than the WPPA in two ways: first, it urges Governments to help women avoid *all* abortions, both legal and illegal; secondly, it explicitly states that abortion in no case should be promoted as a method of family planning.

Among the specific actions that Governments are urged to undertake in order to reduce maternal morbidity and mortality, there is a call to support family planning as a health measure in maternal and child health programmes (recommendation 18 (*f*)). The directness with which that recommendation is stated is a clear measure of the consensus that has developed since the Bucharest conference on the acceptability of family planning.

In the area of adult mortality, the Mexico City document contains innovative recommendations urging Governments to initiate or strengthen programmes to reduce the consumption of tobacco, alcohol, drugs and other potentially dangerous products and also to take measures to eliminate the negative consequences for health that characterize many occupations.

Reproduction and the family

The section of the Mexico City document on reproduction is of critical importance—as was the corresponding section of the WPPA—if for no other reason than that family planning and related activities are the focal point of many—perhaps most—population policies and programmes throughout the world. In the view of most non-specialists, the degree of success of the Mexico City conference will be measured on the basis of what was achieved in that area.

The Mexico City document was indeed able to go considerably beyond the WPPA in the strength and urgency of the language that it adopted in favour of the right of couples and individuals to decide freely

and responsibly the number and spacing of their children. It reflects a growing acceptability of family planning programmes among countries that do not view their rates of population growth as being excessive as well as in countries where excessive rates of population growth are viewed as a barrier to development. Perhaps because of that broader and more stable consensus, the Mexico City document seems more focused and more operational than the WPPA in respect to the issues of reproduction and the family.

Although most of the ideas on reproduction and the family set forth in the WPPA are explicitly reaffirmed in the Mexico City document, there are a few notable exceptions. For instance, the WPPA made a point of recognizing the variety of national goals with regard to fertility and explicitly did not recommend any world family-size norm; the issue is not mentioned in the Mexico City document. The elimination of voluntary sterility and subfecundity, according to the WPPA, should be sought by family planning, medical and related social services; there is no explicit mention of such a goal in the Mexico City document, although in a different section there is a call for increased resources for research to address problems of infertility and subfecundity. Finally, the WPPA devoted considerable attention to the development goals that were thought to "have an effect on the socio-economic context of reproductive decisions that tends to moderate fertility levels" (para. 32) including, for instance, the promotion of social justice, the elimination of child labour and the establishment of social security and old-age benefits. None of those considerations were reexamined in the corresponding section of the Mexico City document.

On the other hand, the Mexico City document goes beyond the WPPA in its implicit recognition of adolescent sexuality. Governments are urged to ensure that adolescents of both sexes receive adequate family-life and sex education, and suitable family planning information and services should be made available to them. The systematic references to "changing individual and cultural values" or "the changing socio-cultural framework of each country", together with explicit mention of the specific difficulties faced by single parents, suggest that the Mexico City conference was very much aware of the transformations in socio-demographic structures and concomitant values that are taking place virtually throughout the world.

Unqualified support for family planning is requested in two similarly worded recommendations—one (recommendation 25) stating that "Governments should, as a matter of urgency, make universally available information, education and the means to assist couples and individuals to achieve their desired number of children" and the other (recommendation 30) urging Governments "to ensure that all couples and individuals have the basic right to decide freely and responsibly the number and spacing of their children and to have the information, education and means to do so". The references to urgency and universality and the specification of the rights of individuals as such were introduced at Mexico City; they were not found in the WPPA.

To illustrate the more pragmatic approach of the Mexico City document, it is useful to compare the recommendations concerning the strengthening of the family with the corresponding paragraphs in the WPPA. The Mexico City document urges Governments to promote the best conditions for family formation and family life (recommendation 26); and paragraph 46 (*c*) of the WPPA states, "In planning development, and particularly in planning the location of industry and business and the distribution of social services and amenities, Governments should take into account not only short-term economic returns or alternative patterns but also the social and environmental costs and benefits involved as well as equity and social justice in the distribution of the benefits of development among all groups and regions."

In the Mexico City document, recommendation 36, which reiterates existing international instruments that prohibit forcible transfer of population from occupied territories or the imposition of civilian populations by the occupying force, and which caused a highly publicized controversy at the Mexico City conference, was addressing a new issue. The delegation of the United States of America, while joining the consensus, strongly protested the inclusion of the recommendation. The related statement in the WPPA was a more general guideline to the effect that in formulating and implementing internal migration policies, measures which infringe the right of freedom of movement and residence within the borders of each State should be avoided, as enunciated in the Universal Declaration of Human Rights and other international instruments. On a related point concerning human rights and internal migration policies, the Mexico City document calls for the use of incentives rather than migration controls in the implementation of any policies intended to minimize undesired migration.

A notable innovation in the Mexico City document is the emphasis on the importance of various forms of migration (circular, seasonal, rural/rural and urban/rural) along with the more widespread concern for rural/urban migration and the support for family policies such as financial and/or other support to parents, the strengthening of child welfare services and child-care provisions, maternity and paternity leave, and assistance to acquire suitable housing. In contrast, the WPPA tends to speak on a more philosophical and less specific level, recommending, for example, that "the family be protected by appropriate legislation and policy without discrimination as to other members of society", and that "family ties be strengthened by giving recognition to the importance of love and mutual respect within the family unit".

Population distribution and internal migration

The Mexico City conference was expected by some analysts to introduce a shift in the approach to issues of internal migration, based on a less negative view of

the processes of urbanization than that presented by the WPPA. That expectation was not very fully borne out by the text adopted in Mexico City. As in the WPPA, the goals of urbanization policy remain the reduction of high rates of migration to capital cities and other large urban centres, the development of medium-sized towns and a reduction of rural/urban and regional inequalities. The recommendation of the Mexico City conference, urging Governments to base population distribution policies "on a comprehensive evaluation of costs and benefits to individuals, families, different socio-economic groups, communities, regions and the country as a whole", with the objective of achieving "broader societal goals, such as raising per capita incomes, increasing efficiency, making the distribution of income more equitable, protecting the environment and improving the quality of life" (recommendation 32), seems to echo concerns already expressed and which Governments were urged to take into account. Other new aspects of internal migration identified at the Mexico City conference were the rights of indigenous groups, the conditions of women migrants and rural dependents left unsupported in rural areas, and the difficulties faced in urban areas by migrant women of rural origin. The Mexico City document is innovative in its emphasis on domestic policy measures rather than measures that would depend upon action at a global level. The WPPA identified the adverse consequences of urbanization in many developing countries as "due in large part to the economic structures resulting from the dependent situation of those countries in the international economic system". And it noted that the correction of those shortcomings "requires as a matter of priority the establishment of equitable economic relations among peoples".

International migration

The Mexico City document is more structured than the WPPA in terms of recommendations on international migration. Its recommendations are organized in such a way that general guidelines, documented migrant workers, undocumented migrants and refugees are all dealt with in separate subsections.

Regarding the general guidelines for formulating international migration policies, the WPPA stresses international harmonization of policies, including measures to avoid brain-drain, and the protection of the human rights and the cultural identity of migrants. However, the Mexico City document places more emphasis on the necessity to promote the mutual adaptation of both immigrant groups and the population of the receiving country, on the need for the Governments of receiving countries to take into account the well-being of migrants and their families, on the demographic implications of migration, and on the rehabilitation of expelled and homeless people, which is viewed as a matter of high priority.

Recommendations in the WPPA concerning documented migrant workers affirm the importance of preventing discrimination in the labour market and in society. The Mexico City document goes beyond that, however, by recommending that the widest dissemination should be given, "*inter alia*, through the mass media, of information aimed at promoting public understanding of and preventing any activity prejudicial to the contribution of documented migrant workers to economic development and cultural interchange." The Mexico City document also contains stronger language concerning the promotion of family reunion; in particular, it states that demographic and other considerations should not prevent Governments from taking the relevant measures. One specific recommendation of the WPPA, inviting countries where immigration has proven to be of long-term nature to explore the possibilities of extending national civil rights to immigrants, is not restated in the Mexico City document.

In respect to undocumented migrants (described as "illegal migrants" in the WPPA), the Mexico City document simply reaffirms the recommendation of the WPPA, with an updating reference to the ILO Convention concerning Migrations in Abusive Conditions, 1975.

The Mexico City document gives considerably more attention than the WPPA to the problems of refugees. It considers not only the protection of the rights of refugees but also the urgency of finding durable solutions to the problems relating to refugees, refugee movements and the causes of those problems. It specifically recognizes the burden that refugee movements place on the countries of first asylum and calls for international support and assistance to be given to such countries.

Population structure

The WPPA discussed four types of structural issues, which it invited Governments to consider and take into account: age structure—its desirable balance and implications; sex structure—in the context of settlement and resettlement schemes and urban planning; socio-economic structures of the labour force, and particularly the shift from agriculture to non-agricultural industries in developing countries; and the dynamics of age structure and fertility—youthful population and high fertility in developing countries and aging population and low fertility in developed countries. The concern for the welfare of the elderly was the object of the most firmly worded recommendation: it states that "all countries should carry out . . . comprehensive, humanitarian and just programmes of social security for the elderly" (para. 66). The Mexico City conference, drawing on the results of the intergovernmental activities in the past decade that have focused on issues of demographic structure (International Year of the Child, International Youth Year and World Assembly on Aging), arrived at a more differentiated view of the contribution that each segment of the population could make to society, and the Mexico City document asserts that its recommendations "contain proposals to foster the growth and value of all age and

sex groups in the community" (para. 32). While calling for further efforts to analyse the issue of aging and urging Governments to secure the welfare and safety to older people–particularly older women–the Mexico City document emphasizes that "Governments should view the aging sector of the population not merely as a dependent group, but in terms of the active contribution that older persons have already made and can still make to the economic, social and cultural life of their families and the community" (recommendation 58).

Of no less importance is the recognition by the Mexico City conference of the shifts in family and household structures which have emerged with increasing strength during the past decade throughout the world. Governments are invited to consider those shifts in family and household structures and their implications for socio-economic planning. (Incidental references to such shifts in household structures are also to be found in recommendation 34 where the needs of single parents, in terms of housing, financial and other support are specifically mentioned.)

Data collection and analysis

The Mexico City document reaffirms the importance of data collection and analysis as an indispensable basis for the formulation, application and evaluation of population and development policies, a point already emphasized in the WPPA. Governments are thus requested to participate in the 1990 World Population and Housing Census Programme, to establish or strengthen civil registration systems, to make use of well-designed sample surveys and in general to ensure that statistics are available on a timely basis. The necessity of international co-operation and assistance to the developing countries for those activities is repeatedly called for, in both the data collection section and in a later section on the role of international co-operation. However, certain recommendations go more particularly beyond the WPPA, reflecting the fact that although considerable progress has been achieved in the field of data collection and analysis in the past decade, certain critical gaps were found to remain. For instance, Governments are urged to ensure that data are tabulated and published separately by sex, in order to more clearly reflect the situation of women; Governments are also encouraged to tabulate and publish data about minority groups; special efforts are called for in the area of migration, both internal and international, that being considered the least developed area of current demographic statistics. A suggestion is made that migration should be studied in the context of the family. Another recommendation urges Governments to ensure that confidentiality and the privacy of the individual are safeguarded in the collection, analysis and dissemination of statistical data.

Research

The WPPA called for an extremely broad research agenda on population problems, which were explicitly said to encompass issues of unemployment, starvation and poverty. That agenda, detailed in paragraph 78, covered topics ranging from population projections and social indicators to the review and analysis of national and international laws which bear directly or indirectly on population factors. In contrast with that approach, the Mexico City recommendations emphasize research in support of programmatic activity, particularly in the areas of fertility regulation and family planning. Biomedical research is given prominent and extensive attention, governmental and funding agencies being urged to allocate increased resources to it, in order to improve the safety and efficacy of existing family planning methods, to develop new methods, to develop better methods of recognizing the female fertility period and to address problems of infertility and subfecundity. Another recommendation calls for priority to be given to service and operational research. Social research into the determinants and consequences of fertility is given priority with a view to increasing the acceptance and improving the design of family planning service programmes. In more general terms, it is recommended that Governments and intergovernmental and non-governmental organizations endeavor to make population research relevant to policies and programmes, with the objective of making innovations in policy formulation, implementation and evaluation. The concluding recommendation maintains the emphasis on policy relevance but broadens the scope of research with a call for special emphasis to "be given to research on the integration of population processes with socio-economic development, considering not only applied but also theoretical and methodological topics" (recommendation 72).

Management, training, information, education and communication

Although several passages of the Mexico City document emphasize the importance of monitoring and evaluating policies and programmes, the section entitled "Management, training, information, education and communication" devotes comparatively little attention to questions of management as such. Rather, emphasis is placed on training and on information dissemination. Specific mention is made of technical co-operation among developing countries and the need to include women in all training activities, in order to ensure the increased participation of women in the design, management, implementation and evaluation of population programmes. As to the contents of training, emphasis is placed, *inter alia*, on family life, sex education, family planning and reproductive physiology. The Mexico City document also urges Governments and intergovernmental organizations to make more effective use of available population data and, for that purpose, to promote forums for assessing and if necessary, reorienting priorities in the population fields.

Role of national Governments

There is a sharp difference of tone between the WPPA and the Mexico City document in respect to the role of national Governments although the great majority of the recommendations made at both conferences are directed to Governments. In the WPPA, it is the principles, rather than the contents, of the role of national Governments that are discussed. Those principles essentially point up the sovereign and predominant role of national Governments in defining problems and needs in the field of population and in formulating and implementing the corresponding policies and plan of action. However, it is also emphasized that sovereignty should be exercised in the context of internationally accepted standards of human rights and bearing in mind that the effect of national action or inaction in that field may extend beyond national boundaries. The Mexico City document goes beyond the legal principle of national sovereignty to attach high priority to self-reliance in the management of population programmes and encourages technical co-operation among developing countries. Then, based on explicit recognition that "many factors, including the lack of definite commitment, inadequate resources, ineffective co-ordination and implementation and insufficient data, have limited the effectiveness of Governments" (para. 36), it recommends focus on specific means whereby those shortcomings can be corrected, at the level of the management of population programmes. Furthermore, the issue of community participation is once again raised, Governments being invited "to involve communities more actively in the planning and implementation of population programmes" (recommendation 77 (*d*)).

Role of international co-operation

The Mexico City document recognizes that international co-operation activities have achieved a number of notable successes in attaining the goals of the WPPA. However, it points out that, although resources have more than doubled in nominal terms, the increase has been insufficient to keep pace with growing needs. The document therefore emphasizes the important role which the international community should play in the further implementation of the WPPA, Governments being urged to increase the level of their assistance for population activities, without detriment to development assistance in other areas. The document also suggests that the existing criteria for setting co-operation activities should be reviewed, "bearing in mind considerations of regional equity and the proper balance between the various phenomena in the field of international co-operation" (recommendation 80).

In contrast with the general approach of the WPPA, which among other things urged that due attention be given to the Plan itself (and thus implicitly recognized that population was not yet fully established as an item on the international agenda), the Mexico City document offers a detailed list of activities to which the international community should give particular attention. They include research and action programmes; institutionalization of the integration of population planning in the development process; improvement of the status of women, biomedical and social science research; collection and analysis of needed data; review of programmes to ascertain the reasons for their success; implementation of monitoring and evaluation systems; promotion of exchanges between countries with common experiences and education and training in population matters. However, the recommendations for action on the role of international co-operation all proceed on the assumption that population is now fully established as an item on the international agenda and that there is no remaining need to urge the case.

The Mexico City document invites the Secretary-General to examine a recommendation that the United Nations Fund for Population Activities be further strengthened, so as to ensure the more effective delivery of population assistance, taking into account the growing needs in that field.

It also invites national non-governmental organizations to continue their pioneering work in opening up new paths for the further implementation of the WPPA. Donors are invited to increase their financial support to non-governmental organizations. Recognition is given to the role that policy makers, members of parliament, the scientific community, the mass media and others in influential positions can play in creating an awareness of population issues and to support appropriate ways of dealing with those issues; the UNFPA and other international organizations are invited to continue providing support for such actions.

Monitoring, review and appraisal

The WPPA provided fairly explicit guidelines concerning, on the one hand, the monitoring of population trends and policies and, on the other hand, the review and appraisal of progress made towards achieving the goals and recommendations of the Plan. In particular, a periodicity was established for the review of those activities by the appropriate bodies of the United Nations (biennially, beginning in 1977, for the monitoring, and quinquennially, beginning in 1979, for the review and appraisal). The Mexico City document recommends that those established activities continue to be undertaken by the Secretary-General, as specified in the Plan. In addition, the Mexico document recommends that the Secretary-General also undertake, through appropriate arrangements, a new activity: the monitoring of the multilateral population programmes of the United Nations system.

Notes

[1] See *Report of the United Nations World Population Conference, Bucharest, 19-30 August 1974* (United Nations publication, Sales No. E.75.XIII.3), chap. I.

[2] See *Report of the International Conference on Population, Mexico City, 6-14 August 1984* (United Nations publication, Sales No. E.84.XIII.8), chap. I, sect. B.

[3] *Primary Health Care. Report of the International Conference on Primary Health Care. Alma-Ata, USSR, 6-12 September 1978* (Geneva, World Health Organization, 1978).

RE-ESTIMATION OF STRUCTURAL PARAMETERS TO OBTAIN ESTIMATES OF MORTALITY IN DEVELOPING COUNTRIES*

*Alberto Palloni*** and Larry Heligman****

SUMMARY

Measures of mortality in developing countries have often been based on a range of techniques, generally known as "indirect techniques" which provide estimates of mortality for certain age groups of the population based on questions of survivorship of kin asked in demographic surveys or population censuses. The indirect techniques have been valuable in demographic analysis because of the lack of reliability of vital statistics in many developing countries and because of the possibility of correlating resulting mortality estimates with other social and economic responses included in surveys.

All of the techniques are based upon models of the structure of the demographic variables being studied. Specifically, the indirect procedures for measuring mortality from data on kin survivorship require models of the age patterns of both mortality and fertility. Traditionally, the most used models of mortality for the indirect estimation of mortality have been the model life tables proposed by Coale and Demeny. The Coale and Demeny life table system has been a very useful tool but its validity for demographic analysis in less developed countries is reduced by the fact that the empirical life tables that serve as its base include only a few countries which represent patterns of mortality found in developing countries.

With the publication of new model life tables for developing countries by the United Nations, it is now possible to provide more reliable formulations for indirect mortality measurements.

This paper provides improved regression equations for transforming survivorship of kin statistics into measures of infant, early childhood and adult mortality. After a short description of the mortality models upon which the new methods are based, the first section of this paper covers the treatment of estimation of mortality in infancy and early childhood. The second section is devoted to transformations of orphanhood data into conditional probabilities of survivorship for adults. Finally, the third section provides illustrative applications of the new equations.

INTRODUCTION

During the past 30 years the increasing integration of population variables into development plans has required reliable information on demographic change in developing countries. However, the statistical infrastructure has not always produced reliable data through the traditional means of civil registration systems. As a result, demographers working in developing countries have come increasingly to rely on what are generally called "indirect techniques", which permit the researcher to describe vital processes from a reduced set of observed indicators.

In the present paper we will be concerned exclusively with the indirect estimation of mortality and infancy, childhood and adulthood. By and large, the indirect techniques aim to estimate mortality by transforming collected data on survivorship of kin into pure mortality measures, taking into consideration certain distributional characteristics of the population. All of the techniques are based upon models of the structure of the demographic variables being studied. Specifically, the indirect procedures for measuring mortality from data on kin survivorship require models of the age patterns of both mortality and fertility.

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Traditionally, the most used models of mortality for the indirect estimation of mortality have been the model life tables proposed by Coale and Demeny.[1] The Coale and Demeny life-table system has been a very useful tool but its validity for demographic analysis in less developed countries is reduced by the fact that the empirical life tables that serve as its base include only a few countries which represent patterns of mortality found in developing countries. As more data of better quality have become available for less developed countries, it has become evident that the age patterns of mortality exhibited by their populations often differ from those recorded in the developed countries and consequently from those embodied in the models of Coale and Demeny. Thus, in theory at least, improved indirect estimates of mortality can be obtained from estimation procedures based on more representative age patterns of mortality. With the publication of new model life tables for developing countries by the United Nations in 1983,[2] it is now possible to provide more reliable formulations for indirect mortality measurements.

The present paper provides improved regression equations for transforming survivorship of kin statistics into measures of infant, early childhood and adult mortality. After a short description of the mortality models upon which the new methods are based, the first section of the paper covers the treatment of estimation of mortality in infancy and early childhood. The second section is devoted to transformations of orphanhood data into conditional probabilities of survivorship for adults. Finally, the third section provides illustrative applications of the new equations.

The United Nations mortality models

In 1983 the United Nations published a new set of model life tables based upon, and applicable to, mortality conditions and structures currently found in the third world.[2] The models were constructed based on the life tables of 72 less developed countries after the Second World War which had been selected after careful evaluation and study. All the life tables were based on recorded data by age and sex, either from demographic surveys or vital registration and censuses. When appropriate, adjustments were made for incompleteness of the data. The data covered a wide range of mortality levels and geo-cultural settings. The United Nations publication provides a description of the construction methodology that went into the new tables and of their use. The following brief description of the models is presented in the United Nations *Manual X*:[3]

> "The new United Nations model life tables are similar to the Coale and Demeny set in that distinct patterns of age-specific mortality schedules have been identified and are published in detail. In addition, the new models incorporate a greater degree of built-in flexibility, allowing the user to construct mortality patterns different from those actually published." [In that sense, they are more akin to the logit system proposed by Brass.[4]]

Four distinct patterns of mortality were identified on the basis of the data available. Because of the predominance of these patterns in certain geographic regions, they are identified in regional terms as the "Latin American," the "Chilean," the "South Asian" and the "Far Eastern" patterns. A fifth pattern, called the "general" pattern was constructed as the overall average of those listed above.

The Latin American model, when compared with the West family of the Coale and Demeny models, exhibits high mortality during the infant and childhood years (due mainly to excess diarrhoeal and parasitic diseases), and again during the young adult ages (largely due to accidents). It also exhibits relatively low levels at older ages, apparently because of comparative low death rates due to cardio-vascular diseases.

The Chilean pattern is characterized by extremely high infant mortality in relation to both the West family and its own child mortality. This excessive infant mortality appears to be due mainly to deaths from respiratory diseases and may also be related to early weaning.

The South Asian pattern is typified by extremely high mortality under age 15 and relatively high mortality at older ages (over age 55 approximately). Correspondingly, mortality during the prime adult ages is relatively low. Data about causes of death are scarce in this region; however, those gathered by the International Diarrhoeal Disease Research Centre in Matlab, Bangladesh, and by the Indian Model Registration Project reveal high rates of diarrhoeal and parasitic diseases at younger ages and high mortality from diarrhoeal and respiratory diseases at older ages.

The Far Eastern pattern exhibits very high death rates at the older ages compared with those of younger ages. There is some evidence that this distinctive pattern may be due to a past history of tuberculosis.

The general pattern, which can be considered an average of the four regional patterns described above, is very similar to the West family of the Coale-Demeny set. Although no distinctive patterns for the African region were developed, due to the extremely low quality of data from that region, the available evidence indicates that the South Asian pattern may be most appropriate to that region.

As mentioned earlier, these tables combine the advantages of the Coale and Demeny regional system and its detailed publication format with the type of flexibility inherent in the Brass logit system. Such characteristics were achieved by using principal component analysis to construct each model, after preliminary clustering of the data had been carried out. Clustering allowed the identification of the four distinctive patterns described above. Within each cluster equations of the form

$$\text{logit}\left[{}_nq_x\right] = U^c_{0x} + \sum_{i=1}^{k} a_i U^c_{ix}$$

were fitted, where ${}_nq_x$ is the observed probability of dying between ages x and $x+n$; U^c_{0x} is the overall average (in logit terms) for cluster c; U^c_{ix} represents the characteristic deviation of the observed from the average, and the coefficient a_i (loading factors) indicates the size of such deviations. Because the fitting procedure used identifies the U_{ix} values as the principal components of the observed ${}_nq_x$ vectors (with x ranging from 0 to ω), the U_{0x} vector can be interpreted as a measure of the average mortality model within each cluster, while U_{1x} may be interpreted as a measure of the typical deviations from that average as mortality levels change. Deviations from the overall average not due purely to changes in level are embodied by the second and third principal components, U_{2x} and U_{3x}, respectively. Hence, in constructing the one-parameter models printed in the tables, the equation presented above with $k = 1$ was used, but setting $k = 2$ or $k = 3$ and judiciously selecting other a_i values allow the user to derive mortality schedules whose pattern deviates from that of the printed tables. In that way, the flexibility of the models is enhanced. The indirect estimation equations presented in the present paper are based solely on the one-parameter models.

Estimation of mortality in infancy and early childhood from information of children surviving

General features of the procedures

If fertility and mortality remain unchanged, the proportion of children dead among those ever born to mothers exactly aged x is given by:

$$d(x) = \int_0^{x-\alpha} f(x-y)q(y)dy \Big/ \int_0^{x-\alpha} f(x-y)dy \quad (1)$$

where $f(x-y)$ and $q(y)$ denote, respectively, the risk of fertility at exactly age $x-y$ and the probability of dying before reaching age y. From the mean value theorem (1) can be simplified to

$$d(x) = q(\overset{*}{y}), \quad 0 < \overset{*}{y} < x-\alpha \quad (2)$$

where $\overset{*}{y}$ is an age that depends on the functions $f(\bullet)$ and $q(\bullet)$. That dependence indicates that the survivorship statistic $d(x)$ cannot be concerted into a pure indicator of mortality unless one knows the fertility pattern and the mortality risks to which the offspring have been exposed. However Brass[4] showed that in the Coale and Demeny life table system the probabilities of dying ($q(y)$) between birth and some age x (lower than 25) can be approximately expressed as a constant multiple of the same probabilities in some arbitrarily selected standard life table. As a result, the possibility existed for estimating the life table functions $q(y)$ from the survivorship statistics, $d(x)$ as long as the proportional age pattern of mortality in the chosen Coale and Demeny model was sufficiently similar to real ones. That characteristic of the Coale and Demeny system can be precisely formulated as follows:

$$q(\overset{*}{y}) = k\, q_s(\overset{*}{y}) \quad 0 < \overset{*}{y} < 25 \quad (3)$$

where $q_s(\overset{*}{y})$ is the probability of dying before age $\overset{*}{y}$ in the standard. From (3) it follows that if z is any other age under 25, then

$$\frac{q(\overset{*}{y})}{q(z)} = \frac{q_s(\overset{*}{y})}{q_s(z)} \quad (4)$$

Using that result in (2) yields

$$d(x) \quad \frac{q_s(\overset{*}{y})}{q_s(i_x)} q(i_x), \text{ or}$$

$$q(i_x) = d(x)\frac{q_s(i_x)}{q_s(\overset{*}{y})} \quad (5)$$

where i_x is an arbitrary integer age dependent on x and relatively near to $\overset{*}{y}$. Thus, knowledge of $\frac{q_s(i_x)}{q_s(\overset{*}{y})}$, what Brass called the "multiplier", would be sufficient to estimate $q(i_x)$ the unknown indicator of cumulative mortality. Brass showed that the appropriate multiplier could be predicted sufficiently accurately by knowing only rough characteristics of the childbearing pattern and suggested its calculation based on a few indicators of the fertility function. The resulting multiplier is then used to inflate (deflate) the observed survivorship statistic [$d(x)$] to bring it to the level of the (unknown) cumulated probability of dying. When women are grouped into five-year age groups, Brass's suggestion is formalized as follows

$$\hat{q}[i_x] = d_{x,x+5} \bullet \hat{M}_{x,x,+5} \quad (6)$$

where $\hat{}$ is a symbol to denote estimated quantities, $d_{x,x+5}$ is the observed proportion of children dead for women in age group $(x,x+5)$ and

$$\hat{M}_{x,x+5} = h(\{f_i\}) \quad (7)$$

where $h(f)$ is a function dependent only on $\{f_i\}$, a set of parameters specifying the fertility schedule.

The basic assumptions of the procedures described are the following:

(a) Mortality and fertility have remained constant during the past;

(b) There is no relation between age of mother, average parity, mortality of mothers and mortality of children;

(c) Age is well identified and no under (over) statement of children born or dead occurs (or if it occurs, the proportionate under (over) statement of children ever born does not vary across ages and is exactly equal to the proportionate under (over) statement of children dead);

(d) The model of mortality has been well identified.

Estimation of the multipliers based on age models

The calculation of the level of mortality ($q(i_x)$) through the transformation of the survivorship statistic

($d_{x,x+5}$) described in the above paragraphs was possible only because of the validity of equation (3) in the Coale/Demeny system. There is no *a priori* reason to believe that the new models of mortality calculated by the United Nations satisfy equation (3). In fact, it can be shown that, to a close approximation, the ratio of the probabilities of dying before age x in any two life tables pertaining to the same model pattern within the United Nations system is given by

$$\frac{q^1(x)}{q^2(x)} \doteq \frac{\int_0^x e^{U^c_{0,t}(1+a^1U^c_{1,t})}dt}{\int_0^x e^{U^c_{0,t}(1+a^2U^c_{1,t})}dt} \qquad (8)$$

where a^1 and a^2 are the loading factors measuring the actual deviation from the average and $U_{1,t}$ are the same quantities as defined earlier. It is not entirely obvious that (8) could be constant in x unless $U_{1,t}$ satisfies some regularity conditions. Yet, *empirically* the quantity in (8) turns out to be quite well approximated by a constant. That can be seen in table 1 where the ratios of the values $q(x)$ for three levels of mortality to the values of $q(x)$ in an intermediate life table ($e_0 = 54$) are displayed. The ratios shown are for female tables; the male counterparts exhibit similar constancy. The pattern of departures from equiproportionality is also illustrated in figure I, which displays the values of the ratios from table 1 for two extreme life tables ($e_0 = 45$ years and $e_0 = 65$ years) in the Far Eastern pattern of mortality (i.e., the pattern showing the worst departures from proportionality). The same figure also displays the corresponding values for identical levels of life tables from the West Model of the Coale/Demeny system. For both life-table systems the standard is a life table with life expectancy equal

TABLE 1. FACTORS OF PROPORTIONALITY IN THE UNITED NATIONS MODEL LIFE TABLES (FEMALES)

Age	Latin American model	Chilean model	South Asian model	Far Eastern model	General model
	Life expectancy at birth = 40 years				
1	1.655	1.711	1.650	1.848	1.716
2	1.752	1.780	1.745	1.964	1.817
3	1.793	1.811	1.785	2.016	1.861
4	1.815	1.828	1.806	2.045	1.884
5	1.828	1.839	1.817	2.064	1.898
6	1.831	1.843	1.820	2.071	1.903
7	1.835	1.848	1.822	2.079	1.908
8	1.838	1.852	1.825	2.086	1.913
9	1.841	1.856	1.827	2.093	1.917
10	1.845	1.860	1.830	2.099	1.921
11	1.845	1.862	1.830	2.102	1.922
12	1.846	1.864	1.831	2.105	1.924
13	1.846	1.866	1.831	2.107	1.925
14	1.847	1.868	1.831	2.110	1.926
15	1.847	1.870	1.832	2.113	1.927
16	1.848	1.872	1.832	2.116	1.928
17	1.848	1.875	1.832	2.118	1.929
18	1.848	1.878	1.833	2.121	1.930
19	1.848	1.880	1.833	2.124	1.931
20	1.848	1.883	1.883	2.126	1.931
21	1.849	1.886	1.834	2.130	1.932
22	1.848	1.888	1.834	2.130	1.932
23	1.847	1.890	1.834	2.130	1.932
24	1.847	1.892	1.834	2.131	1.932
Mean	1.829	1.854	1.816	2.085	1.906
Standard deviation	.043	.040	.041	.064	.049
Coefficient of variation	.024	.022	.022	.031	.026

TABLE 1 (*continued*)

Age	Latin American model	Chilean model	South Asian model	Far Eastern model	General model
	Life expectancy at birth = 62 years				
1	0.728	0.712	0.725	0.674	0.708
2	0.704	0.697	0.699	0.653	0.686
3	0.694	0.691	0.689	0.643	0.676
4	0.689	0.687	0.684	0.638	0.671
5	0.686	0.685	0.681	0.635	0.667
6	0.684	0.684	0.680	0.633	0.666
7	0.683	0.683	0.679	0.631	0.665
8	0.682	0.682	0.678	0.630	0.664
9	0.681	0.681	0.678	0.628	0.662
10	0.680	0.680	0.677	0.627	0.661
11	0.680	0.679	0.677	0.626	0.661
12	0.680	0.679	0.676	0.625	0.660
13	0.679	0.678	0.676	0.624	0.660
14	0.679	0.678	0.676	0.624	0.659
15	0.679	0.677	0.676	0.623	0.659
16	0.678	0.677	0.675	0.622	0.658
17	0.678	0.676	0.675	0.621	0.658
18	0.678	0.675	0.675	0.620	0.657
19	0.677	0.674	0.675	0.619	0.657
20	0.677	0.673	0.674	0.618	0.656
21	0.677	0.673	0.674	0.616	0.656
22	0.676	0.672	0.674	0.615	0.655
23	0.676	0.671	0.674	0.614	0.654
24	0.676	0.670	0.673	0.614	0.654
Mean	.683	.681	.680	.628	.664
Standard deviation	.011	.009	.011	.014	.012
Coefficient of variation	.016	.014	.016	.022	.108
	Life expectancy at birth = 70 years				
1	0.446	0.422	0.436	0.365	0.412
2	0.411	0.403	0.401	0.340	0.383
3	0.398	0.395	0.387	0.329	0.370
4	0.390	0.390	0.380	0.322	0.363
5	0.386	0.387	0.376	0.318	0.359
6	0.384	0.386	0.375	0.316	0.357
7	0.383	0.384	0.373	0.314	0.356
8	0.381	0.383	0.372	0.312	0.354
9	0.379	0.382	0.371	0.310	0.352
10	0.378	0.381	0.369	0.309	0.350
11	0.377	0.380	0.369	0.307	0.350
12	0.377	0.379	0.369	0.306	0.349
13	0.376	0.378	0.368	0.305	0.348
14	0.376	0.377	0.368	0.304	0.348
15	0.375	0.377	0.368	0.303	0.347
16	0.375	0.376	0.367	0.302	0.346
17	0.374	0.375	0.367	0.300	0.345
18	0.374	0.373	0.366	0.299	0.344
19	0.373	0.372	0.366	0.297	0.344
20	0.373	0.371	0.366	0.296	0.343
21	0.372	0.370	0.365	0.294	0.342
22	0.371	0.368	0.365	0.293	0.341
23	0.370	0.367	0.364	0.291	0.340
24	0.370	0.366	0.364	0.290	0.339
Mean	.382	.381	.374	.309	.353
Standard deviation	.017	.012	.016	.017	.016
Coefficient of variation	.043	.033	.042	.055	.046

NOTE: The standard used corresponds to a life tabel with $e_0 = 54$.

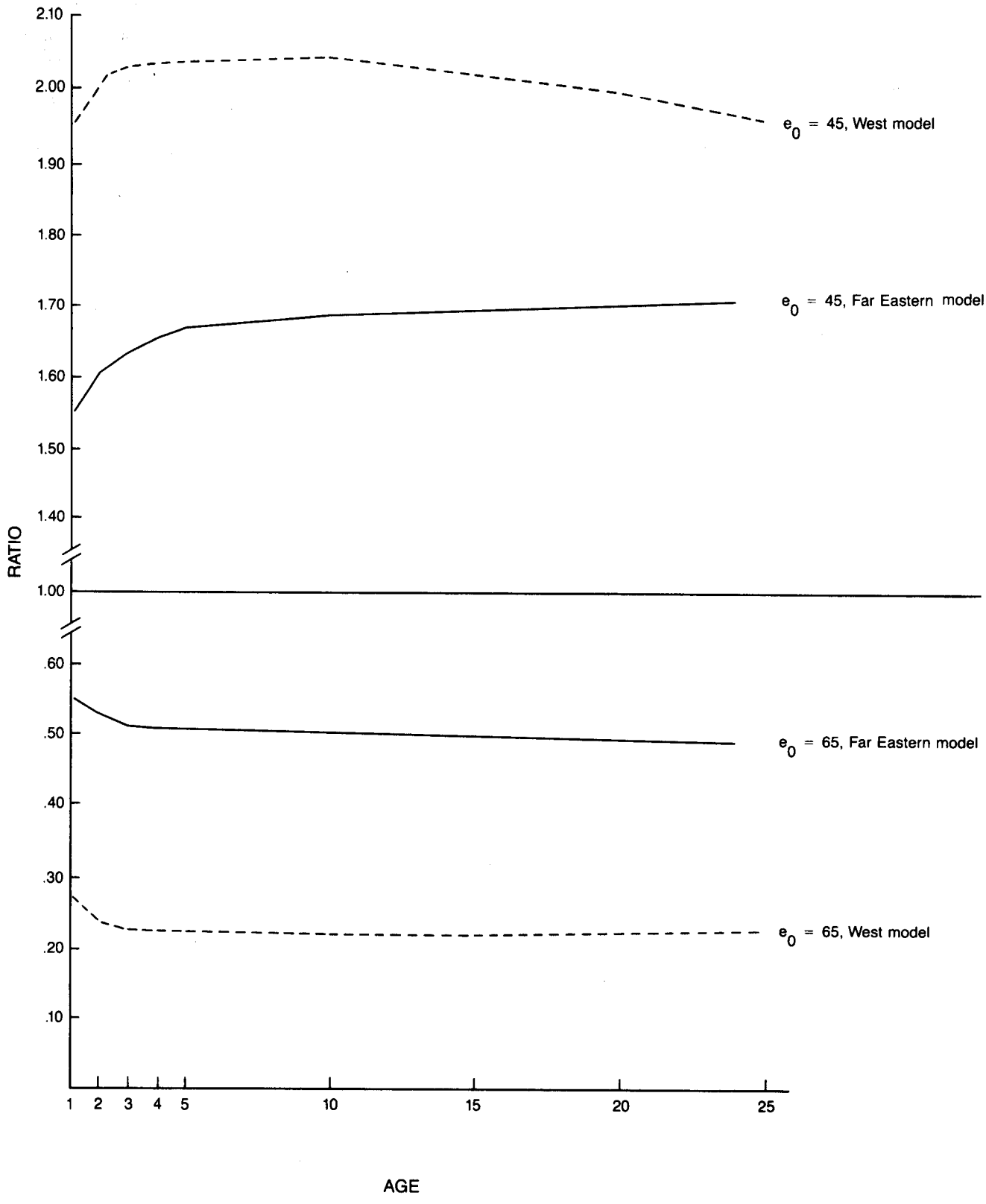

Figure 1. Ratios $q(x)/q_s(x)$ for the Far Eastern and West patterns of mortality. (The standard values, $q_s(x)$, are taken from the life tables with $e_0 = 54$ in each of the models.)

to 54. It is evident that departure from proportionality in the worst of the United Nations models is not too different from that of the West model.

With the validity of expression (3) for the United Nations system verified, it is mainly a technical exercise to estimate the parameters for regression equations to calculate the multipliers in equation (7). Two formulations of $h(\bullet)$ and its arguments were used (a) first, $h(\bullet)$ is specified as a linear function of PAR1 and PAR2, respectively, the ratio of the average parity of women 15-19 to the average parity of women 20-24 and the ratio of the latter to that of women aged 25-29. PAR1 and PAR2, however, are not very adequate indicators of the latter part of a childbearing schedule. Hence, one should expect that the accuracy of the corresponding values $M_{x,x+5}$ will decrease as the age of the mother increases. Hence a second specification was also devised: (b) $h(\bullet)$ is defined as a linear function of PAR1, PAR2 and the (population weighted) average age at childbearing. The new indicator permits better discrimination among fertility functions having different profiles of late childbearing.

The actual calculation of the multipliers was based on the formula

$$M_{x,x+5} = \frac{1}{q_s(x)} \left\{ \int_{z=x}^{z=x+5} \int_0^{z-\alpha} \tilde{C}(z) \, f(z-y) q_s(y) d_y d_z / \int_{z=x}^{z=x+5} \int_0^{z-\alpha} \tilde{C}(z) \, f(z-y) dy dx \right\} \qquad (9)$$

where $\tilde{C}(z)$ is the age structure of women reporting. The values of PAR1, PAR2 and average age at childbearing were determined as follows.

$$\text{PAR1} = \int_{15}^{20} \tilde{C}(x) \int_\alpha^x f(y) dy dx \; / \; \int_{20}^{25} \tilde{C}(x) \int_\alpha^x f(y) dy dz$$

$$\text{PAR2} = \int_{20}^{25} \tilde{C}(x) \int_\alpha^x f(y) dy dz \; / \; \int_{25}^{30} \tilde{C}(x) \int_\alpha^x f(y) dy dz$$

$$\text{AVE} = \int_\alpha^\beta \tilde{C}(x) f(x) \, x \, dx \; / \; \int_\alpha^\beta \tilde{C}(x) f(x) \qquad (10)$$

In (9) the standard of mortality in each model was set equal to the life tables with $e_0 = 54$. The sequence $\tilde{C}(z)$ was determined from a stable population with a survival schedule corresponding to $e_0 = 54$ and an intrinsic rate of growth equal to .03. In all, 96 schedules of fertility were used. They were selected among those constructed by Coale and Trussell[5] to represent the range of childbearing that is likely to be found among developing nations (see annexes). The multipliers in (9) were calculated for i_x values equal to 1, 2, 3, 5, 10, 15 and 20 when the age group of mothers are, respectively, 15-19, 20-24, . . . , 45-49.

Table 2a displays the estimated regression coefficients, coefficient of determination (R^2), the square root of the mean square error of the predicted values (RMSE), and their average value (mean), for the formulation $M_{x,x+5} = a + b\text{PAR1} + c\text{PAR2}$. The predictive power of the model declines sharply after age group 30-34; nevertheless the relative errors are not of an intolerably high magnitude. In the worst case, the average relative error in the multiplier is less than 2 per cent. Table 2b displays the regression coefficients for estimating the second specification, $Mx,x+5 = a + b\text{PAR1} + c\text{PAR2} + d\text{AVE}$, in which the average age at childbearing is added as a predictor. Quite clearly, the relative improvement in the fitting power is substantial for the first age group and last four age groups. The sizes of the relative errors in the multipliers are also considerably reduced.

ERRORS OF ESTIMATION DUE TO AGE MISREPORTING

One of the most common sources of error in carrying out the indirect estimation procedure is the misreporting of ages of women. Age misreporting can adopt two forms, each having quite different effects on the transformation which converts a survivorship statistic into a probability of surviving (or dying). On the one hand, age misreporting may take the form of systematic age over (under) statement, with the subsample of women whose age has been over (under) stated being a *selected* sample in terms of parity and/or mortality experience of their offspring. On the other hand, age misreporting may simply be the result of a tendency to prefer certain digits (0 or 5). While very little can be done to correct for the former type of error, the disturbing effects of age heaping can be avoided by appropriately selecting unconventional age groups.[6] The unconventional age groups ought to be formed in such a way as to include within them the ages ending in the preferred digit as well as those ages ending in "unattractive" digits. Since most age heaping takes place through systematic attraction towards digits 0 and 5, an optimal redefinition of women's age groups may be the following: 18-22, 23-27, 28-32, 33-37, 38-42, 43-47 etc. The estimates of the corresponding regression equations for the first four of these unconventional age groups and the pertinent measures of fit are exhibited in table 3. The specification of the model does not include the mean age at childbearing as a third indicator of the fertility pattern. The reason is simple: if there is pronounced age heaping, its effects also ought to distort the calculated mean value. By the same token, the values of PAR1 and PAR2 are modified to refer to the relationships between the average parities in the first three *unconventional* age groups.

ERRORS OF ESTIMATION DUE TO MORTALITY TRENDS

A much more consequential type of error results from violation of the assumption of constant mortality. If mortality during the past has been declining (and fertility remained unchanged), the transformation

TABLE 2A. ESTIMATES OF REGRESSION COEFFICIENTS FOR SPECIFICATION $M_{x,x+5} = a + b\,\text{PAR1} + c\,\text{PAR2}$ FOR CONVENTIONAL AGE GROUPS

Age group	a	b	c	R^2	RMSE	Mean	i_x
			Latin American model				
15-19	1.5865	−1.4553	.3581	.912	.0166	1.0559	1
20-24	1.7102	−.4692	−.1787	.985	.0051	1.0388	2
25-29	1.6764	.0730	−.3629	.993	.0026	.9885	3
30-34	1.6806	.3115	−.4230	.884	.0089	.9969	5
35-39	1.7028	.4075	−.4491	.761	.0132	1.0198	10
40-44	1.6791	.4088	−.4428	.732	.0139	.9998	15
45-49	1.6636	.3838	−.4322	.758	.0129	.9861	20
			Chilean model				
15-19	1.6906	−1.3677	.3316	.908	.0161	1.1592	1
20-24	1.6952	−.3163	−.1475	.984	.0038	1.0049	2
25-29	1.6410	.0662	−.2559	.990	.0020	1.0107	3
30-34	1.6275	.2115	−.2888	.879	.0063	1.0010	5
35-39	1.6379	.2756	−.3220	.782	.0092	1.0041	10
40-44	1.6397	.3162	−.3676	.769	.0109	.9872	15
45-49	1.6565	.3607	−.4242	.781	.0122	.9799	20
			South Asian model				
15-19	1.5876	−1.5053	.3762	.909	.0173	1.0587	1
20-24	1.7186	−.4589	−.1838	.984	.0054	1.0474	2
25-29	1.6807	.0854	−.3556	.991	.0032	1.0005	3
30-34	1.6761	.3038	−.3932	.870	.0088	1.0093	5
35-39	1.6789	.3680	−.3855	.732	.0119	1.0264	10
40-44	1.6392	.3294	−.3345	.692	.0112	1.0084	15
45-49	1.6072	.2541	−.2731	.726	.0087	.9981	20
			Far Eastern model				
15-19	1.5664	−1.1081	.2931	.901	.0131	1.0545	1
20-24	1.6573	−.3649	−.1314	.987	.0036	1.0278	2
25-29	1.6369	.0433	−.2929	.994	.0020	.9822	3
30-34	1.6492	.2480	−.3740	.903	.0076	.9819	5
35-39	1.6932	.3732	−.4646	.811	.0126	.9956	10
40-44	1.7115	.4767	−.5683	.785	.0163	.9730	15
45-49	1.7472	.5697	−.6646	.780	.0192	.9702	20
			General model				
15-19	1.5783	−1.2546	.3156	.910	.0144	1.0557	1
20-24	1.6822	−.4134	−.1537	.986	.0043	1.0332	2
25-29	1.6554	.0579	−.3265	.993	.0022	.9851	3
30-34	1.6631	.2805	−.3953	.892	.0082	.9898	5
35-39	1.6952	.3879	−.4445	.779	.0127	1.0117	10
40-44	1.6860	.4235	−.4724	.750	.0144	.9927	15
45-49	1.6853	.4319	−.4897	.762	.0145	.9838	20

NOTES: All numbers significant at less than .01 level.
RMSE is root of the mean square errors in $M_{x,x+5}$.
Mean is the average of $M_{x,x+5}$.
i_x is the corresponding age of child.

$$\hat{M}_{x,x+5} \bullet \hat{d}_{x,x+5}$$

will overestimate the current value of $q(i_x)$. The bias will increase as the children's length of exposure to past mortality risks increases. That causes the bias to be higher for older mothers and for populations with an earlier childbearing pattern. Table 4 shows the relative errors of estimation in $q(i_x)$ in cases in which the life expectancy has increased from 35 years to 60 years during a 30-year period. The magnitude of these errors is high enough to render the estimates unusable as indices of current mortality.

Although the estimates of $q(i_x)$ obtained from survivorship statistics under conditions of mortality decline are not good indicators of current mortality, they are acceptable representations of cumulated mortality risks during the past. In fact, when life expectancy increases linearly, it can be shown that the estimate of $q(i_x)$ is a good measure of the actual value of $q(i_x)$ in the life table that applied some years before.[7] The number of years before which the estimate refers to is independent of the *rate* at which mortality has been declining and is determined mainly by the fertility pattern and, to a lesser extent, by the pattern of mortality. Although exact values of t_x can be computed *directly* from the time distribution of children ever born or the age distribution of children surviving, it is easier to calculate them from estimated models. Two different models, of the form $t_{x,x+5} = a + b\text{PAR1} + c\text{PAR2}$, were estimated, depending on whether conventional or unconventional age groups are being handled. The estimated coefficients for the five mortality patterns in the United Nations system are displayed in tables 5a and 5b.

TABLE 2B. ESTIMATES OF REGRESSION COEFFICIENTS FOR SPECIFICATION $M_{x,x+5} = a + b\text{PAR1} + c\text{PAR2} + d\text{AVE}$ FOR CONVENTIONAL AGE GROUPS

Age group	a	b	c	d	R^2	RMSE	Mean	i_x
			Latin American model					
15-19	1.1892	−1.6937	.6464	.0106	.934	.0145	1.0559	1
20-24	1.8625	−.3778	−.2892	−.0041	.991	.0040	1.0388	2
25-29	1.5877	.0197*	−.2986	.0024	.996	.0018	.9885	3
30-34	1.2500	.0532	−.1106	.0115	.997	.0015	.9969	5
35-39	1.0605	.0222	.0170	.0171	.999	.0008	1.0198	10
40-44	1.0024	.0028**	.0048*	.0180	.999	.0003	.9998	15
45-49	1.0326	.0052**	.0256*	.0168	.999	.0005	.9861	20
			Chilean model					
15-19	1.3274	−1.5854	.5949	.0097	.928	.0143	1.1592	1
20-24	1.8129	−.2457	−.2329	−.0031	.991	.0029	1.0649	2
25-29	1.5632	.0196*	−.1996	.0021	.996	.0012	1.0107	3
30-34	1.3236	.0293	−.0684	.0081	.998	.0009	1.0020	5
35-39	1.1895	.0068	.0032	.0119	.999	.0003	1.0041	10
40-44	1.1098	−.0014**	.0166*	.0141	.999	.0004	.9872	15
45-49	1.0615	.0040**	.0073*	.0159	.999	.0008	.9299	20
			South Asian model					
15-19	1.1749	−1.7580	.6805	.0109	.931	.0151	1.0587	1
20-24	1.8716	−.3652	−.2966	−.0041	.990	.0044	1.0474	2
25-29	1.5899	.0299*	−.2887	.0024	.996	.0025	1.0005	3
30-34	1.2694	.0548	−.0934	.0108	.996	.0019	1.0093	5
35-39	1.1156	.0231	.0298	.0149	.998	.0013	1.0264	10
40-44	1.1077	.0040**	.0573	.0141	.999	.0010	1.0084	15
45-49	1.1952	.0018**	.0306	.0109	.999	.0006	.9981	20
			Far Eastern model					
15-19	1.2194	−1.3143	.5432	.0093	.903	.0110	1.0545	1
20-24	1.7671	−.2996	−.2105	−.0029	.992	.0028	1.0278	2
25-29	1.5668	.0017*	−.2424	.0019	.997	.0014	.9822	3
30-34	1.2833	.0307	−.1103	.0098	.998	.0012	.9819	5
35-39	1.0765	.0068	−.0202	.0165	.999	.0007	.9956	10
40-44	.9115	.0014**	.0083	.0213	.999	.0007	.9730	15
45-49	.8071	.0111	.0129	.0251	.999	.0009	.9702	20
			General model					
15-19	1.2210	−1.4686	.5746	.0095	.9335	.0124	1.0557	1
20-24	1.8115	−.3360	−.2475	−.0034	.992	.0033	1.0332	2
25-29	1.5768	.0109*	−.2695	.0021	.997	.0015	.9851	3
30-34	1.2682	.0439	−.1090	.0105	.997	.0013	.9898	5
35-39	1.0769	.0176	.0038	.0165	.999	.0007	1.0117	10
40-44	.9545	.0034	.0036	.0187	.999	.0002	.9927	15
45-49	.9760	.0071	.0246	.0189	.999	.0005	.9838	20

NOTES: *not significant at .01 level; **not significant at .05 level; all others significant at less than .01 level.

RMSE is root of the mean square errors in $M_{x,x+5}$.

Mean is the average of $M_{x,x+5}$.

i_x is the corresponding age of child.

Estimation of the multipliers based on duration models

Partly as a stratagem to avoid the effects of response errors in the ages of mothers and partly as a device to capture more exhaustively the variability attributable to fertility patterns, the models may be reformulated using as a grouping criteria the duration of marriage (union) rather than the age of the mothers.[8] The proportion of children dead among mothers of duration $d,d+5$ is given by

$$D_{d,d+5} = \frac{\int_d^{d+5}\int_0^{\infty} g(x)e^{-s(x+y)}p(x+y)\int_0^{y} r(x+t)q(y-t)\,dt\,dx\,dy}{\int_d^{d+5}\int_0^{\infty} g(x)e^{-s(x+y)}p(x+y)\int_0^{y} r(x+t)\,dt\,dx\,dy} \tag{11}$$

where $g(x)$ is the density of first marriages, $p(x+y)$ is the probability that a respondent will survive up to age $x+y$, $r(x+t)$ is the marital fertility rate at $x+t$, $q(y-t)$ is the probability that a child born $y-t$ years before will die before the survey or census, and s is a rate of growth in a stable age structure.

The calculation of the multipliers $M_{d,d+5}$ was carried out using the equation

$$M_{d,d+5} = \frac{q(i_d)}{D_{d,d+5}} \tag{12}$$

where i_d was set equal to 2, 3, 5, 10, 15, 20, 25 for duration groups 0-4, 5-9, 10-14, ..., 35-39, respectively.

The indicators of the fertility function PAR1, PAR2 were calculated as follows:

TABLE 3. ESTIMATES OF REGRESSION COEFFICIENTS FOR SPECIFICATION $M_{x,x+5} = a + b\text{PAR1} + c\text{PAR2}$ FOR UNCONVENTIONAL AGE GROUPS

Age group	a	b	c	R^2	RMSE	Mean	i_x
			Latin American model				
18-22.....	.9780	−.5984	.2086	.939	.0096	.8907	1
23-27.....	1.1114	−.2755	−.0931	.994	.0024	.9349	2
28-32.....	1.1534	.0111	−.3068	.998	.0012	.9292	3
33-37.....	1.2123	.1690	−.4333	.974	.0040	.9552	5
			Chilean model				
18-22.....	1.0815	−.5068	.1696	.944	.0078	1.0109	1
23-27.....	1.1233	−.1845	−.0812	.994	.0017	.9911	2
28-32.....	1.1296	.0151	−.2214	.997	.0010	.9704	3
33-37.....	1.1558	.1107	−.3045	.975	.0029	.9719	5
			South Asian model				
18-22.....	.9691	−.6014	.2093	.939	.0095	.8914	1
23-27.....	1.1197	−.2619	−.0983	.994	.0029	.9463	2
28-32.....	1.1580	.0252	−.3006	.997	.0026	.9461	3
32-37.....	1.2034	.1711	−.4005	.971	.0046	.9742	5
			Far Eastern model				
18-22.....	.9869	−.4672	.1649	.934	.0077	.9280	1
23-27.....	1.0815	−.2305	−.0700	.996	.0018	.9453	2
28-32.....	1.1240	−.0105	−.2543	.998	.0012	.9297	3
33-37.....	1.1869	.1212	−.3961	.979	.0038	.9379	5
			General model				
18-22.....	.9797	−.5303	.1857	.938	.0085	.9117	1
23-27.....	1.0992	−.2522	−.0811	.995	.0020	.9406	2
28-32.....	1.1378	.0018**	−.2800	.998	.0011	.9297	3
32-37.....	1.1964	.1488	−.4107	.977	.0038	.9483	5

NOTES: *not significant at .01 level; **not significant at .05 level; all others significant at less than .01 level.

RMSE is root of the mean square errors in $M_{x,x+5}$.

Mean is the average of $M_{x,x+5}$.

i_x is the corresponding age of child.

TABLE 4. PERCENTAGE ERROR IN THE ESTIMATES OF $q(i_x)$ UNDER CONDITIONS OF DECLINING MORTALITY[a]

Age	i_x	1	2	3	4	5
15-19.....	1	2.16	2.84	2.08	3.31	2.49
20-24.....	2	7.87	8.80	8.25	10.35	8.55
25-29.....	3	17.55	19.17	18.25	22.29	19.04
30-34.....	5	31.56	33.57	32.45	40.08	34.31
35-39.....	10	48.80	51.55	50.07	62.39	53.33
40-44.....	15	68.08	71.75	70.37	86.87	74.40
45-49.....	20	91.64	96.80	95.80	117.12	100.24

[a]In order to calculate the errors, we applied the multipliers derived from equations in table 2a to proportions of children dead. The latter were generated by the combination of 96 fertility functions and a cohort survival schedule dependent on a mortality decline equivalent to an annual rate of change in e_0 equal to .83 (progressing from a life expectancy of 35 to 60 in 30 years).

$$\text{PAR1} = \frac{\int_0^5 \int_0^\infty g(x)e^{-s(x+y)}p(x+y)\int_0^y r(x+t)dtdxdy}{\int_5^{10} \int_0^\infty g(x)e^{-s(x+y)}p(x+y)\int_0^y r(x+t)dtdxdy} \quad (13a)$$

$$\text{PAR2} = \frac{\int_5^{10} \int_0^\infty g(x)e^{-s(x+y)}p(x+y)\int_0^y r(x+t)dtdxdy}{\int_{10}^{15} \int_0^\infty g(x)e^{-s(x+y)}p(x+y)\int_0^y r(x+t)dtdxdy} \quad (13b)$$

For the computation of (12), (13a) and (13b), a standard schedule of mortality was used with a life expectancy of 54 years, and a rate of growth of .03. The functions $g(x)$ and $r(y)$ were calculated as suggested by Coale and Trussell.[5]

The first model to be estimated is one of the form

$$M_{d,d+5} = a + b\text{PAR1} + c\text{PAR2} \quad (14)$$

The corresponding regression coefficient and measures of goodness of fit are displayed in table 6. Unlike the age models, the fitting power of the duration models is quite high at all duration groups with only two indicators of the fertility function PAR1 and PAR2. As a result, models which include the mean duration of childbearing are not presented.

ERRORS OF ESTIMATION OWING TO MISREPORTING OF MARRIAGE DURATIONS

The primary advantage of using a duration rather than an age model depends on the higher precision

TABLE 5A. ESTIMATED COEFFICIENTS OF TIME REFERENCES FOR SPECIFICATION $t_{x,x+5} = a + b\text{PAR1} + c\text{PAR2}$ FOR CONVENTIONAL AGE GROUPS

Age group	a	b	c	R^2	RMSE	Mean	i_x
			Latin American model				
15-19	1.1703	.5129	−.3850	.4637[a]	.0179	.5290	1
20-24	1.6955	4.1320	−.1635	.999	.0079	1.7505	2
25-29	1.8296	2.9020	3.4707	.999	.0112	3.6886	3
30-34	2.1783	−2.5688	9.0883	.975	.1130	6.2683	5
35-39	2.8836	−10.3282	15.4301	.887	.3370	9.2451	10
40-44	4.4580	17.1809	10.4296	.797	.5641	12.4953	15
45-49	6.9351	−19.3871	23.4007	.789	.6685	16.2597	20
			Chilean model				
15-19	1.3092	1.9474	−.7982	.773[a]	.0297	.6748	1
20-24	1.6897	4.6176	−.0173	.999	.0094	1.9195	2
25-29	1.8368	2.6370	4.0305	.999	.0105	3.9607	3
30-34	2.2036	−3.3520	9.9233	.978	.1337	6.6333	5
35-39	2.9955	−11.4013	16.3441	.875	.3702	9.6889	10
40-44	4.7734	−17.8850	20.8883	.787	.5881	12.9500	15
45-49	7.4495	−19.0513	23.0529	.788	.6607	16.6323	20
			South Asian model				
15-19	1.1922	.7940	−.5425	.477[a]	.0240	.5203	1
20-24	1.7173	4.3117	−.1653	.998	.0097	1.7964	2
25-29	1.8631	2.8767	3.5848	.999	.0241	3.7595	3
30-34	2.1808	−2.7219	9.3705	.976	.1267	6.3561	5
35-39	2.7654	−10.8808	16.2255	.884	.3619	9.4041	10
40-44	4.1378	−18.6219	22.2390	.797	.6162	12.8468	15
45-49	6.4885	−22.2001	26.4911	.788	.7633	16.9556	20
			Far Eastern model				
15-19	1.2779	1.5714	−.6994	.707[a]	.0274	.6384	1
20-24	1.7471	4.2638	−.0752	.998	.0099	1.8729	2
25-29	1.9107	2.7285	3.5881	.999	.0119	3.8147	3
30-34	2.3172	−2.6259	9.0238	.973	.1185	6.3783	5
35-39	3.2087	−9.8891	14.7339	.885	.3260	9.2784	10
40-44	5.1141	−15.3263	12.2507	.800	.5097	12.2715	15
45-49	7.6383	−15.5739	19.7669	.804	.5557	15.5924	20
			General model				
15-19	1.2136	.9740	−.5247	.574[a]	.0216	.5775	1
20-24	1.7025	4.1569	−.1232	.999	.0086	1.7842	2
25-29	1.8360	2.8632	3.5220	1.00	.0105	3.7180	3
30-34	2.1882	−2.6521	9.1691	.974	.1162	6.3107	5
35-39	2.9682	−10.3053	15.3161	.885	.3371	9.2719	10
40-44	4.6526	−16.6920	19.8534	.796	.5500	12.4510	15
45-49	7.1425	−18.3021	22.4168	.793	.6371	16.0975	20

NOTES: All numbers significant at less than .01 level.
RMSE is root of the mean square errors in $t_{x,x+5}$.
Mean is the average of $t_{x,x+5}$.
i_x is the corresponding age of child.
[a] Low R^2 for first age groups is explained by the lack of variance of the calculated values of $t_{x,x+5}$.

with which simple indicators can capture the entire profile of the marital fertility schedule. Undoubtedly, additional benefits can be collected if reports of marital duration are of higher quality than those of age. That is likely to be true in societies in which entrance into sanctioned unions is an almost universal practice. However, patterns of union formation heavily dominated by consensual unions may produce more problems than those associated with identification of complete age or birthdate. In many of the societies in which the time spent in consensual unions may be as long as, or longer than, the time spent in sanctioned unions, it is not at all clear that a proper identification of the date of entrance into union is made by the respondent. The questions "How long ago did you first marry?" or "What was the date of your first marriage?" may elicit erroneous responses owing to the confusion between sanctioned union and consensual union. Moreover, the unstable character of consensual unions guarantees that the time elapsed since entrance into one of them is *not* a desirable proxy for exposure to risks of childbearing. Finally, there is little doubt that, under uncertainty about the proper dates, the respondent may select as a reference the date of another relevant event, such as the birth of the first child, and accordingly bias the information revealed about the length of the union. The latter distortion will be more consequential for mothers of lower rather than higher durations. The application of duration models under these circumstances may perform worse than age models, even if the latter are affected by age misreporting.

The brief discussion presented above suggests that it does not seem advisable to generate "unconventional"

TABLE 5B. ESTIMATED COEFFICIENTS OF TIME REFERENCES FOR SPECIFICATION $t_{x,x+5} = a + b\text{PAR1} + c\text{PAR2}$ FOR UNCONVENTIONAL AGE GROUPS

Age group	a	b	c	R^2	RMSE	Mean	i_x
			Latin American model				
18-22	1.2284	2.1678	−1.0366	.793[a]	.0610	.789	1
23-27	1.8286	4.8413	−.6624	.992	.0328	2.718	2
28-32	1.4850	3.9584	3.5212	.998	.0242	5.148	3
33-37	.5814	−.4234	10.8981	.992	.0804	8.035	5
			Chilean model				
18-22	1.4559	2.8118	−1.2691	.830[a]	.0217	1.103	1
23-27	1.8747	5.0884	−.4812	.995	.0289	2.994	2
28-32	1.4213	3.7571	4.1666	.998	.0531	5.486	3
33-37	−.5760	−1.0379	11.9958	.991	.0814	8.489	5
			South Asian model				
18-22	1.2437	2.3440	−1.1305	.779[a]	.0287	.814	1
23-27	1.8602	4.9239	−.6418	.993	.0370	2.786	2
28-32	1.4934	3.9589	3.6727	.998	.0681	5.228	3
33-37	−.5389	−.5312	11.3917	.991	.1051	8.154	5
			Far Eastern model				
18-22	1.3942	2.4608	−1.1379	.806[a]	.1754	1.003	1
23-27	1.9036	4.7825	−.5379	.993	.0517	2.876	2
28-32	1.5659	3.7813	3.6471	.998	.0656	5.268	3
33-37	.8021	−.4850	10.7110	.992	.1554	8.123	5
			General model				
18-22	1.2951	2.2773	−1.0736	.800[a]	.0245	.881	1
23-27	1.8448	4.8051	−.6135	.992	.0355	2.757	2
28-32	1.4726	3.9214	3.6057	.998	.0658	5.185	3
33-37	.5972	−.5081	10.9786	.992	.0988	8.079	5

NOTES: All numbers significant at less than .01 level.
RMSE is root of the mean square errors in $t_{x,x+5}$.
Mean is the average of $t_{x,x+5}$.
i_x is the corresponding age of child.
[a] Low R^2 for first age groups is explained by the lack of variance of the calculated values of $t_{x,x+5}$.

duration based equations: what is relevant when duration is misreported is not the attractiveness of certain digits (or dates) but the individual recognition of traditional social institutions. Unlike the phenomenon of age heaping, the social bias in recognition of marriage and union is devoid of any regularity understood by demographers.

ERRORS OWING TO MORTALITY DECLINE

In so far as mortality has changed during the past, the estimation of current mortality levels will also be biased when duration models are applied to estimate the corresponding transformations. Time references, $t_{d,d+5}$, for each of the duration groups were directly calculated and then modeled as follows:

$$t_{d,d+5} = a + b\text{PAR1} + c\text{PAR2} \qquad (15)$$

The estimates of the regression coefficients for (15) and the corresponding measures of goodness of fit appear in table 7. Note that the goodness of fit—as measured by R^2—of the equation for the time reference corresponding to the *first* duration group is not nearly as high as the others. That is owing not to the lack of power of the marital fertility indicators but to an artifact of the values of $t_{0,5}$: their variability with childbearing patterns is so small that a prediction made on the basis of its mean value produces errors only slightly larger than those generated by the regression equations.

ESTIMATION OF ADULT MORTALITY

The case of constant mortality

The indirect estimation of adult mortality can follow two alternative paths. On the one hand, there are techniques which permit the adjustment of observed death rates for completeness of death registration and population enumeration. Those techniques result in the calculation of life tables starting at ages 5, 10 or 15. Their application is recommended when the pattern of observed death rates is not heavily affected by errors in the enumerated deaths and/or population, which vary systematically with age. On the other hand, there are techniques which depend on the transformation of survivorship statistics pertaining to adult ages, such as the proportion of the population whose mother (father) is still alive, or the proportion of the ever married population who have lost their first spouse. Those approaches are suitable when vital statistics are either lacking or are too unreliable to be of any use. In the discussion below we shall consider only the technique based on maternal orphanhood. The other techniques, based on paternal orphanhood and widowhood, have tended to produce inconsistent and unreliable results

TABLE 6. ESTIMATES OF REGRESSION COEFFICIENTS FOR SPECIFICATION $M_{d,d+5} = a + b\text{PAR1} + c\text{PAR2}$ FOR MARITAL DURATION GROUPS[a]

Duration group (in years)	a	b	c	R^2	RMSE	Mean	i_x
			Latin American model				
0-4	1.8181	−.5453	.1286	.9932	.00008	1.2047	2
5-9	1.7464	−.3530	−.1163	.9997	.00005	1.0281	3
10-14	1.8198	.0070	−.4608	.9995	.00006	1.0145	5
15-19	1.8941	.337	−.6712	.9942	.0003	1.0332	10
20-24	1.8930	.3187	−.6875	.9890	.0005	1.0147	15
25-29	1.8909	.2328	−.6543	.9742	.0007	1.0041	20
30-34	1.9128	.2505	−.6893	.9806	.0007	1.0073	25
			Chilean model				
0-4	1.7632	−.4131	.0952	.9938	.00006	1.1757	2
5-9	1.6890	−.2239	−.0950	.9997	.00004	1.0351	3
10-14	1.7208	.0062	−.3192	.9995	.00004	1.0141	5
15-19	1.7647	.2204	−.4584	.9950	.0002	1.0155	10
20-24	1.7978	.2222	−.5239	.9861	.0003	1.0016	15
25-29	1.8550	.2103	−.6008	.9829	.0005	.9982	20
30-34	1.9281	.2706	−.7246	.9849	.0005	1.0045	25
			South Asian model				
0-4	1.8265	−.5606	.1333	.9926	.00010	1.2111	2
5-9	1.7502	−.3447	−.1149	.9997	.0001	1.0367	3
10-14	1.8083	.0845	−.4369	.9994	.0002	1.0239	5
15-19	1.8496	.3121	−.5962	.9929	.0003	1.0359	10
20-24	1.8150	.2675	−.5503	.9799	.0006	1.0178	15
25-29	1.7677	.1567	−.4392	.9634	.0006	1.0090	20
30-34	1.7406	.1403	−.3974	.9762	.0005	1.0059	25
			Far Eastern model				
0-4	1.7419	−.4122	.0965	.9934	.00005	1.1556	2
5-9	1.6939	−.2840	−.0943	.9997	.00004	1.0176	3
10-14	1.7656	.0485	−.3892	.9997	.00005	1.0011	5
15-19	1.8675	.2775	−.6303	.9962	.0002	1.0147	10
20-24	1.9486	.3317	−.7957	.9888	.0003	.9961	15
25-29	1.9633	.3588	−.9649	.9834	.0006	.9982	20
30-34	2.1638	.4525	−1.1344	.9825	.0007	1.0110	25
			General model				
0-4	1.7941	−.4986	.1173	.9932	.00007	1.1902	2
5-9	1.7265	−.3248	−.1078	.9997	.00004	1.0250	3
10-14	1.7964	.0648	−.4301	.9996	.00005	1.0086	5
15-19	1.8809	.3099	−.6492	.9949	.00021	1.0269	10
20-24	1.9056	.3186	−.7115	.9838	.0005	1.0097	15
25-29	1.9352	.2661	−.7328	.9777	.0006	1.0039	20
30-34	1.9732	.2991	−.7947	.9811	.0007	1.0095	25

NOTES: All numbers significant at less than .01 level.
RMSE is root of the mean square errors in $M_{d,d+5}$.
Mean is the average of $M_{d,d+5}$.
i_x is the corresponding age of child.
[a]The ages of estimation were 2, 3, 5, 10, 15, 20 and 25 for duration groups 0-4, 5-9, . . . , 40-44, respectively.

and, as a consequence, are applied less frequently and with a great deal more scepticism.

The idea of using information on maternal orphanhood to estimate mortality was originally explored by Lotka and Henry, fully exploited by Brass, and its application considerably simplified by Hill and Trussell.[9] We present a very tight summary of the essentials of the technique and proceed to derive transformations based on the United Nations models of mortality.

The proportion of non-orphans at exact x age at any time y can be expressed as

$$\text{NOR}_y(x) = \frac{\int_\alpha^\beta n_{y-n}(a) f(a) \frac{p(a+x)}{p(a)}\, da}{\int_\alpha^\beta n_{y-x}(a) f(a)\, da} \qquad (16)$$

where $n_{y-x}(a)$ is the number of females aged a x years before y, $f(a)$ is the fertility rate at age a and $\frac{p(a+x)}{p(a)}$ is the conditional probability of surviving from age a to age $a+x$ for the female population. Expression (16) is valid only if fertility and mortality have remained constant over time. If the curve of fertility were collapsed towards its mean age (all mothers produce children only at the mean age of childbearing), it would follow that the proportion of non-orphans at age x equals the conditional probability of surviving x additional years since reaching the mean age of childbearing. Under more general conditions, however,

$$\text{NOR}(x) = \frac{\overset{*}{p}(\overset{*}{A}+x)}{\overset{*}{p}(\overset{*}{A})} \qquad (17)$$

TABLE 7. ESTIMATED COEFFICIENTS OF TIME REFERENCES FOR SPECIFICATION $t_{d,d+5} = a + b\text{PAR1} + c\text{PAR2}$ FOR MARITAL DURATION GROUPS

Duration group (in years)	a	b	c	R^2	RMSE	Mean	i_x
			Latin American model				
0-4	1.9413	.0750	−.0119	.8800	.0126	1.4042	2
5-9	2.1905	3.6285	−.2235	.9999	.0050	2.9038	3
10-14	1.2889	3.5121	4.6440	.9995	.0048	5.4921	5
15-19	−1.9784	−2.7698	15.1849	.9999	.0438	8.4969	10
20-24	−4.0367	−10.4675	26.5536	.9963	.1305	11.775	15
25-29	−3.4804	−13.2169	31.8843	.9863	.2189	15.1596	20
30-34	−2.5124	−12.8783	36.5354	.9842	.2503	19.6330	25
			Chilean model				
0-4	1.5502	.7134	−.1954	.9830	.0100	1.1788	2
5-9	1.9861	4.2179	−.0133	.9999	.0038	3.0943	3
10-14	.9268	3.1238	5.8022	.9996	.0035	5.8231	5
15-19	−2.4191	−3.9528	17.0030	.9998	.0331	8.9270	10
20-24	−4.2223	−11.6202	28.0375	.9957	.1119	12.2290	15
25-29	−2.9932	−13.4452	31.8196	.9849	.1985	15.5124	20
30-34	−1.5056	−12.5689	35.1967	.9839	.2359	19.7843	25
			South Asian model				
0-4	1.5593	.4561	−.1348	.9744	.0135	1.1339	2
5-9	2.0207	3.9311	−.1303	.9999	.0063	2.9123	3
10-14	1.1478	3.5490	4.9222	.9997	.0151	5.5528	5
15-19	−2.2434	−2.8448	15.8069	.9998	.0532	8.6248	10
20-24	−4.7090	−10.8582	28.1514	.9964	.1358	12.0667	15
25-29	−4.8974	−14.2269	35.3066	.9875	.2177	15.7851	20
30-34	−4.4591	−14.8160	41.7544	.9853	.2447	20.6694	25
			Far Eastern model				
0-4	1.8976	.2380	−.0816	.9197	.0085	1.4385	2
5-9	2.2198	3.6886	−.1035	.9999	.0037	3.0378	3
10-14	1.3151	3.2007	4.9208	.9996	.0062	5.6063	5
15-19	−1.6577	−3.0220	14.9400	.9998	.0341	8.5541	10
20-24	−2.8877	−10.0544	24.5141	.9956	.1124	11.6149	15
25-29	−1.1997	−11.6541	27.0305	.9842	.2023	14.5250	20
30-34	1.1153	−10.5753	30.1156	.9846	.2437	18.4920	25
			General model				
0-4	2.0219	−.0732	−.0119	.9157	.0109	1.4854	2
5-9	2.2402	3.6178	−.2111	.9998	.0043	2.9526	3
10-14	1.3013	3.4958	4.7043	.9995	.0038	5.5430	5
15-19	−1.8950	−2.8421	15.1546	.9998	.0408	8.5394	10
20-24	−3.6971	−10.3558	25.9914	.9961	.1232	11.7505	15
25-29	−2.8148	−12.7146	30.4876	.9858	.2122	15.0036	20
30-34	−1.8001	−12.2269	34.8030	.9845	.2465	19.3369	25

NOTES: All numbers significant at less than .01 level.
RMSE is root of the mean square errors in $t_{d,d+5}$.
Mean is the average of $t_{d,d+5}$.
i_x is the corresponding age of child.

where $\overset{*}{A}$ is an age, dependent on the survivorship schedule, the fertility curve and, to a lesser extent, on the age distribution of women x years before, $n(a,x)$.

Up to that point the similarities in the relations between the survivorship statistic and the underlying survivorship function in that case and in the case of childhood mortality are quite evident. However, the dependence of $\overset{*}{A}$ on the mortality level cannot be disposed of, as was done in the case of childhood mortality, because there is no equiproportionality in the values of the conditional probabilities of surviving pertaining to different levels of mortality within the same pattern. That is tantamount to saying that in order to transform NOR(x) into an indicator of a level of mortality, it is necessary to introduce some information about that level of mortality. Although that may seem an unsolvable problem, there is in fact a very simple solution to it: once the variability created by different patterns of fertility is held constant, the quantity NOR(x) is a good proxy for the underlying level of mortality. That suggests that a simple functional specification may permit the transformation of the survivorship statistics into an estimate of mortality level:

$$\frac{p(x+\text{AVE})}{p(x)} = a + b\text{NOR}(x) + c\text{AVE} \quad (18)$$

where AVE is, as before, the average age at childbearing.

In practical applications, however, it is desirable to work with populations in the usual five-year age groups and with a fixed conditional probability of surviving rather than one that systematically changes with

the fertility pattern. For convenience, it has been customary to estimate $\frac{p(25+x)}{p(25)}$, with x being 10, 15, . . . for respondents in the age groups 5-9, 10-14, . . . , respectively. The value of 25 replaces AVE in the left hand side of (18) because that is the age around which the mean age of childbearing patterns in developing countries varies. In summary, the procedure of estimation requires calculating $P(25+x)/p(25)$ from

$$\frac{p(x+25)}{p(25)} = a + b\text{NOR}_{x-5,x} + c\text{AVE} \qquad (19)$$

where $\text{NOR}_{x-5,x}$ is the proportion not orphaned in age group $(x-5,x)$. In order to calculate the parameters a, b and c in (19) we simulated populations by combining six levels of mortality corresponding to life expectancies at birth of 40, 45, 50, 55, 60 and 65 years with 96 fertility schedules (see annexes). The quantity $n(a,x)$ was, in all cases, equivalent to the age distribution in a stable population with an intrinsic rate of growth consistent with the particular combination of fertility and mortality functions and a gross reproduction rate of 3.00. The simulations were repeated for each of the five models of mortality in the United Nations system and in each set of simulations the parameters of expression (19) were estimated. The results with the associated measures goodness of fit are presented in table 8. In all cases the goodness of fit of the equations (R^2) are quite acceptable, but the relative errors of prediction increase as the age groups increase. Thus, for example, in the Latin American model of mortality, the relative error is about 0.10 per cent of the first age group but 8 per cent for the last age group. For that and other reasons related to age misreporting, it is recommended that the estimation of conditional probabilities should not go beyond age groups 40-44 or 45-49.

Unlike the procedures suggested by Hill and Trussell[9] and the United Nations *Manual X*,[3] the proposal here is that the transformation of $\text{NOR}_{x-5,x}$ into conditional probabilities of surviving be specific by mortality pattern. That is because there are non-trivial precision gains to be obtained whenever the model of mortality is identified properly. Table 9 displays the mean square errors that would be generated under all possible combinations of underlying and used model patterns for a selected set of age groups. The numbers in table 9 are obtained by averaging the errors obtained when the survivorship statistics generated by one model of mortality ("underlying" model) are transformed into mortality estimates using the equations corresponding to one of the other models of mortality ("used" model). The last column in each panel of the table contains the errors expected when the equations proposed by Hill and Trussell are used, and each of the five models of the United Nations is alternatively the true underlying one. Notice that under total uncertainty about the correct model pattern, the errors associated with the Hill and Trussell coefficients will always be larger than those produced by the best among those in the United Nations system (the General Pattern).

It was stated above, when expression (16) was introduced, that fertility must have remained constant over time for the maternal orphanhood technique to be applicable. Although theoretically that is true, and the assumption was made in estimating the parameters of expression (19), in practice a fertility decline can be accounted for by varying the value of AVE in expression (19). That is, rather than entering the current value of AVE in expression (19) for all age groups of respondents, a different value of AVE can be used for each age group of respondents, depending on the value of AVE in existence at the time of their birth.

The case of changing mortality

Violation of the assumption about constancy in mortality can affect to a significant degree the final estimates derived from information on mothers' survival status. In fact, the direction and order of magnitude of the errors are quite similar to the ones that resulted from violation of an analogous assumption for estimating mortality in infancy and childhood. Table 10 displays the average proportionate errors of the conditional probabilities of surviving that are obtained under different conditions regulating the mortality decline. The errors are higher for the higher age groups, naturally reflecting the fact that the mothers of the respondents have been subjected to higher-than-average risks. By the same token, more rapid changes in mortality bring about higher proportionate errors. The biases are by no means trivial. For the age groups that are most commonly used to estimate average levels of adult mortality (15-40), the proportionate errors fluctuate between 2.5 per cent and 32 per cent in the case of a mortality decline with an intermediate pace. When mortality has been declining more rapidly, the magnitude of the lower bound error increases to 5.1 per cent and that of the upper bound to 39 per cent.

The solution to the problem is again to "date" the estimated conditional probabilities of surviving. If the estimates obtained under the assumption of constant mortality do not describe current mortality conditions, they may still be good estimates of mortality risks applied to a recent period. Naturally, those time references should vary across age groups of respondents, following an increasing trend with age. Yet if they were invariant with the pace and type of decline and depended solely on known conditions, such as the fertility pattern or the (erroneously) estimated current mortality level, it would be possible to recover the actual mortality trend and, consequently, the current levels of mortality. As in the case of childhood mortality estimation, time references satisfying the two conditions do exist in the case of adult mortality estimation. Brass and Bamgbogye[10] have derived approximate expressions to calculate time references for estimates based on maternal and paternal orphanhood as well as those based on widowhood data. The conditions under which those expressions are valid are the following: (*a*)

TABLE 8. ESTIMATES OF PARAMETERS OF SPECIFICATION

$$\frac{p(25+x)}{p(25)} = a + b\mathrm{NOR}_{x-5,x} + c\mathrm{AVE}^{a}$$

Age group	x	a	b	c	R^2	RMSE	Mean
			Latin American model				
5-9	10	−.3910	1.3382	.0021	.998	.0010	.9411
10-14	15	−.2849	1.1991	.0034	.999	.0013	.9060
15-19	20	−.2580	1.1289	.0051	.999	.0017	.8669
20-24	25	−.2814	1.0917	.0074	.999	.0021	.8211
25-29	30	−.3429	1.0707	.0107	.999	.0027	.7655
30-34	35	−.4412	1.0639	.0148	.999	.0031	.6945
35-39	40	−.5561	1.0650	.0193	.999	.0033	.6049
40-44	45	−.6558	1.0670	.0229	.999	.0040	.4918
45-49	50	−.6832	1.0519	.0239	.995	.0065	.3621
50-54	55	−.5953	.9963	.0209	.986	.0086	.2325
55-59	60	−.3918	.8454	.0139	.969	.0084	.1211
			Chilean model				
5-9	10	−.4025	1.3342	.0027	.997	.0016	.9378
10-14	15	−.3033	1.1938	.0043	.998	.0020	.8995
15-19	20	−.2918	1.1300	.0063	.998	.0024	.8552
20-24	25	−.3261	1.0957	.0090	.998	.0028	.8028
25-29	30	−.3942	1.0766	.0125	.998	.0030	.7396
30-34	35	−.4910	1.0699	.0165	.999	.0031	.6602
35-39	40	−.5908	1.0682	.0204	.999	.0033	.5623
40-44	45	−.6556	1.0608	.0229	.998	.0049	.4440
45-49	50	−.6374	1.0313	.0224	.994	.0076	.3171
50-54	55	−.5204	.9613	.0184	.984	.0094	.1972
55-59	60	−.3293	.8164	.0118	.968	.0085	.1015
			South Asian model				
5-9	10	−.3232	1.2785	.0017	.995	.0011	.9656
10-14	15	−.2277	1.1451	.0032	.995	.0019	.9428
15-19	20	−.2277	1.0872	.0055	.994	.0028	.9136
20-24	25	−.2832	1.0591	.0088	.993	.0039	.8743
25-29	30	−.4000	1.0602	.0133	.994	.0048	.8176
30-34	35	−.5484	1.0716	.0187	.996	.0049	.7395
35-39	40	−.7049	1.0908	.0242	.998	.0038	.6326
40-44	45	−.8026	1.0983	.0276	.999	.0031	.5019
45-49	50	−.7856	1.0751	.0271	.995	.0061	.3552
50-54	55	−.6292	.9958	.0218	.984	.0089	.2156
55-59	60	−.3819	.8351	.0135	.963	.0085	.1085
			Far Eastern model				
5-9	10	−.3862	1.3105	.0029	.997	.0019	.9289
10-14	15	−.3059	1.1739	.0051	.998	.0027	.8858
15-19	20	−.3207	1.1184	.0079	.998	.0034	.8344
20-24	25	−.3849	1.0920	.0115	.998	.0038	.7714
25-29	30	−.4829	1.0841	.0157	.999	.0036	.6911
30-34	35	−.5765	1.0772	.0196	.999	.0033	.5952
35-39	40	−.6421	1.0665	.0222	.999	.0047	.4798
40-44	45	−.6373	1.0377	.0223	.996	.0076	.3546
45-49	50	−.5445	.9781	.0193	.989	.0100	.2334
50-54	55	−.3875	.8779	.0138	.976	.0101	.1320
55-59	60	−.2106	.7079	.0076	.958	.0074	.0600
			General model				
5-9	10	−.3804	1.3229	.0023	.998	.0012	.9395
10-14	15	−.2813	1.1837	.0038	.998	.0018	.9033
15-19	20	−.2715	1.1194	.0059	.998	.0023	.8614
20-24	25	−.3137	1.0875	.0089	.998	.0029	.8107
25-29	30	−.3960	1.0739	.0127	.999	.0033	.7468
30-34	35	−.5031	1.0701	.0170	.999	.0033	.6660
35-39	40	−.6120	1.0709	.0212	.999	.0034	.5648
40-44	45	−.6810	1.0653	.0238	.998	.0050	.4435
45-49	50	−.6592	1.0335	.0231	.993	.0078	.3111
50-54	55	−.5241	.9506	.0185	.992	.0096	.1864
55-59	60	−.3114	.7818	.0111	.962	.0082	.0900

NOTES: All numbers significant at less than .01 level.

[a]Mean is the mean value of $\frac{p(25+x)}{p(25)}$; RMSE is the square root of the mean square error.

TABLE 9. SQUARE ROOT OF MEAN SQUARE ERRORS FOR DIFFERENT COMBINATIONS OF UNDERLYING (TRUE) AND USED MORTALITY PATTERNS

True model	Latin American	Chilean	South Asian	Far Eastern	General	Hill and Trussell
			15-19 model used			
Latin American	.0017	.0032	.0060	.0078	.0034	.0048
Chilean	.0037	.0024	.0061	.0057	.0030	.0060
South Asian	.0051	.0042	.0028	.0056	.0033	.0053
Far Eastern	.0083	.0062	.0063	.0034	.0057	.0078
General	.0038	.0028	.0044	.0052	.0023	.0046
	.0083	.0062	.0063	.0078	.0057	.0078
			25-29 model used			
Latin American	.0027	.0045	.0080	.0105	.0050	.0042
Chilean	.0046	.0030	.0062	.0072	.0033	.0056
South Asian	.0088	.0066	.0048	.0066	.0059	.0092
Far Eastern	.0104	.0073	.0075	.0036	.0069	.0110
General	.0053	.0035	.0053	.0068	.0033	.0056
	.0088	.0073	.0080	.0105	.0069	.0110
			35-39 model used			
Latin American	.0033	.0045	.0106	.0096	.0053	.0047
Chilean	.0045	.0033	.0083	.0068	.0036	.0066
South Asian	.0101	.0078	.0038	.0062	.0067	.0124
Far Eastern	.0104	.0076	.0062	.0047	.0069	.0119
General	.0054	.0037	.0072	.0064	.0034	.0074
	.0104	.0078	.0106	.0096	.0069	.0124

NOTE: The last column corresponds to the error produced if the regression equations estimated by Hill and Trussell were used. The last line corresponds to the maximum error produced by the model used. The underlined entries correspond to the minimum of those maximums.

mortality conditions in the population can be approximated by a life table pertaining to a logit system, with declining mortality being represented by a level parameter (α) which varies linearly over time, and (b) the force of mortality to which mothers of children born T years ago are exposed can be represented as a linear function of time elapsed since some time reference $\overset{*}{T}$ ($T < \overset{*}{T} < 0$).

In the present paper we shall follow a slightly different route of estimation of time references. First of all, we shall not use life tables pertaining to a logit system but rather the life tables of the General Model of the United Nations. After numerous experimentations it was clear that the time references were virtually invariant across models and it was deemed unnecessary to re-estimate them for each model separately. Secondly, instead of presenting a closed-form expression for the time references, we suggest the use of regression equations dependent on fertility indicators and a proxy for mortality levels. Elsewhere it has been shown that, under suitable regularity conditions, a closed-form approximation does exist even if no logit system is used. The values calculated from that expression are very close to the ones obtained using the regression equations.[11] The basic assumption underlying our procedure is that life expectancy has been increasing linearly over the 30 or so years before the survey or census.

TABLE 10. ERRORS IN THE ESTIMATED CONDITIONAL PROBABILITIES OF SURVIVING THAT ARE PRODUCED WHEN MORTALITY IS DECLINING (GENERAL MORTALITY PATTERN OF THE UNITED NATIONS SYSTEM)

Age group of respondent	Fast relative[a] error(×100)	Intermediate relative error(×100)	Slow relative error(×100)
5-9	.89	.99	1.25
10-14	2.45	2.53	1.22
15-19	5.09	4.94	.63
20-24	9.16	8.44	.81
25-29	14.91	13.17	2.78
30-34	21.26	18.10	4.61
35-39	29.75	24.91	7.83
40-44	38.65	31.99	11.18
45-49	48.30	39.83	15.47

[a] This is the ratio of the values of the "actual error" to the true value of the current conditional probabilities.

The starting point to generate the regression equations is a set of simulated populations (see annexes) dependent on a set of 96 fertility schedules, each of them combined with different types of mortality declines. The latter are defined by imposing a range of rates of increase in life expectancy mirroring slow, intermediate and rapid gains in survivorship. Using the assumption of constancy in mortality (equations presented in table 8), we estimate conditional probabilities of surviving from the observed proportion of non-orphans for each of eight quinquennial age groups. Those are then "dated" by seeking *directly* the time at which they apply. The range of variation across types of mortality decline in the time references defined is small and susceptible to modelling with a few parameters.

There are three sources of systematic variation of the time references: one is the type of decline itself, the second is the pattern of fertility, and the third one is the combined effects of the pattern of fertility and the type of decline (interaction). By and large the highest fraction of the total variance of the reference times can be attributed to the fertility pattern. For a given age group of respondents, the older the fertility

schedule, the higher the time reference. That relation simply reflects the steeper gains in survivorship to mothers who bear children at ages in which mortality risks are higher. Accurate indicators of a fertility schedule are the population weighted mean age at childbearing and the ratios of average parity at ages 15-19 to 20-24 and 20-24 to 25-29. The effects of the type of decline are minor and can be accurately represented by using the proportion of non-orphans. Finally, since the *effects* of type of decline vary with fertility pattern, an interaction term was introduced to capture most of the unaccounted residual variance. The model to estimate the time reference is of the following form:

$$T_{x,x+5} = \beta_0 + \beta_1 \text{AVE} + \beta_2 \text{PAR1} + \beta_3 \text{PAR2} + \beta_4 \text{NOR}_{x,x+5} + \beta_5 \text{NOR}_{x,x+5} \bullet \text{AVE} + \epsilon \quad (20)$$

where AVE is the (population weighted) average age at childbearing, PAR1 is the ratio of the average parity of women 15-19 to the average parity of women 20-24, and $\text{NOR}_{x,x+5}$ is the proportion of non-orphans in the age group $x,x+5$. Table 11 displays the estimates of the regression coefficients and measures of goodness of fit, for the General Pattern of the United Nations models of mortality. The far right column displays the relative errors in the prediction of reference times associated with each equation. An important feature of the regression equations is that, although the coefficient of determination declines as the age group increases, the average and relative errors remain within tolerable limits.

To apply the equations contained in table 11, it is necessary first to determine whether or not the data themselves suggest the existence of mortality declines. That can be done by transforming each of the estimated conditional probabilities into a single index of mortality, such as life expectancy at birth. If the values of the latter slope downwards as the age group increases, then that can be taken as *prima facie* evidence of mortality decline. Caution should be exercised, however, since one can rarely discount the possibility that a downward slope in life expectancy is the product of error (adoption, selection processes, age misreporting) rather than an outcome of mortality trends. If the trend is solidly established, the researcher may use the equations producing the estimates of time references and proceed actually to reconstruct the past trend as well as to derive estimates of the current level of adult mortality. As always, it is important to search for independent validation of the estimates thus generated.

An illustrative application to the case of Peru (1960-1978)

Mortality in infancy and childhood

The requisite information for the application of the indirect techniques for measuring early age mortality in Peru is from several sources: the Encuesta Nacional de Fecundidad, taken in 1977-1978 as part of the World Fertility Survey (WFS); the 1975 National Survey (Encuesta Nacional); the 1972 Population Census; and the 1969-1970 KAP Survey. In addition, the birth histories collected as part of the WFS provide a basis for generating an independent series of estimates of the probabilities of dying before ages 1 and 5 against which those derived from the indirect methods can be contrasted. Data are presented and analysed separately for males and females. As a result, we do not make full use of the information contained in the 1972 census since its results have been published for both sexes together.

Tables 12a, 12b, 13a and 13b display the basic set of estimates derived, respectively, from the 1977-1978 and the 1969-1970 surveys. In each case, the estimates are dated by estimating time references, since in Peru there is strong evidence of substantial mortality change. Examination of the tables reveals a few important features. First, it is quite clear that, among the models used (the United Nations Latin American, Chilean, and general patterns and the Coale/Demeny West region), it is the Chilean model that yields results quite out of line with the rest. In fact, the conversion of $q(i_x)$ into $q(1)$ leads to values that are uniformly higher in the Chilean model. The results corresponding to the other three models fall within a narrow range having as a lower bound the estimates associated with

TABLE 11. REGRESSION COEFFICIENTS FOR THE ESTIMATION OF TIME REFERENCE FOR CONDITIONAL PROBABILITIES OF SURVIVING DERIVED FROM ORPHANHOOD DATA (GENERAL MODEL OF THE UNITED NATIONS)

Age group	β_0	β_1	β_2	β_3	β_4	β_5	R^2	*Mean reference*		
								RMSE[a]	*Time*	*CV*[b]
5-9	−339.7	13.53	−.254*	2.266	355.27	−13.99	.986	.0581	4.95	.0117
10-14	−167.0	6.84	.109*	2.691	181.69	−7.20	.987	.0574	7.39	.0078
15-19	−105.4	4.49	.289*	3.068	121.19	−4.81	.985	.0745	9.70	.0077
20-24	−72.2	3.28	.508*	3.417	90.95	−3.65	.981	.0935	11.95	.0078
25-29	−46.9	2.33	.746*	3.614	68.99	−2.76	.976	.1036	13.91	.0074
30-34	−18.3	1.26	.520*	2.427	44.33	−1.77	.967	.0666	14.97	.0044
35-39	14.3	.11	.658*	1.809	3.93	−.28	.834	.989	16.31	.0061
40-44	52.0	−1.22	.770*	.541	−60.05	1.98	.860	.3152	17.03	.0185
45-49	94.9	−2.71	.251*	−1.66	−167.51	5.69	.852	.7690	17.48	.0440

NOTE: *Coefficient not significant at the .01 level; all others significant at well below .01 level.

[a] Square root of mean square error.
[b] Ratio of RMSE to the mean value of reference time.

the Latin American model, and as an upper bound the estimates associated with the West model. The estimates associated with the general model fall in the middle of the range. Secondly, in all cases the estimated values of $q(1)$ slope upward as the age group of the mother increases. To be sure, there are some minor irregularities to that pattern in the results from both surveys. It can be seen, for example, that in tables 12a and 12b the estimates associated with the first age group are somewhat higher than the estimates associated with some of the age groups that follow. In tables 13a and 13b an analogous pattern occurs with the estimates corresponding to the second age group of mothers. Those irregularities can be accounted for by the association between infant mortality and age of mother. Thirdly, a comparison of the trends implied by the estimates obtained from the 1969-1970 and 1977-1978 data reveals some degree of consistency. This can be seen in table 14 which displays the estimated values of $q(1)$ at various years. The estimates were obtained using the grouped mean fitting technique on the observations in tables 12a, 12b, 13a and 13b (the observations corresponding to the first age group were always discarded). Despite some differences in the case of male children, the trends derived from the Latin American model are remarkably consistent. The agreement between the trend derived from the 1969-1970 data and that obtained from the 1977-1978 data—as measured by the square root of the mean square differences—is quite impressive: the best agreement corresponds to the Latin American model (.0058) and the worst to the West model (.0085). For males the agreement is less impressive but is again best for the Latin American model (.0173) and worst for the West model (.0318). Fourthly, not only does the Latin American model

TABLE 12A. ESTIMATES OF $q(i_x)$ AND $q(1)$ FOR FEMALES DERIVED FROM APPLICATION OF NEW MULTIPLIERS, PERU, 1977-1978

Age of mother	i_x	Proportion children dead	Estimates of $q(i_x)$ using models				Estimates of $q(1)$ in models				Estimated time reference[a,b] (general model)
			Latin American	Chilean	General	West	Latin American	Chilean	General	West	
15-19	1	.1297	.1327	.1464	.1331	.1380	.1327	.1464	.1331	.1380	1.04 (1976.5)
20-24	2	.1241	.1327	.1352	.1315	.1329	.0997	.1183	.1025	.1055	2.25 (1975.3)
25-29	3	.1448	.1504	.1515	.1492	.1482	.0998	.1242	.1037	.1075	3.69 (1973.8)
30-34	5	.1431	.1506	.1488	.1491	.1476	.0911	.1163	.0948	.0987	5.55 (1972.0)
35-39	10	.1858	.2003	.1944	.1987	.1949	.1054	.1391	.1098	.1170	7.74 (1969.8)
40-44	15	.2030	.2146	.2101	.2140	.2105	.1072	.1432	.1113	.1184	10.36 (1967.1)
45-49	20	.2480	.2586	.2567	.2598	.2550	.1179	.1590	.1213	.1291	13.66 (1963.8)

[a] The calendar periods to which the estimates apply are shown in parentheses.

[b] Owing to the virtual model invariance of the estimates, the time references are estimated using the regression equations of the general model.

TABLE 12B. ESTIMATES OF $q(i_x)$ AND $q(1)$ FOR MALES DERIVED FROM APPLICATION OF NEW MULTIPLIERS, PERU, 1977-1978[a]

Age of mother	i_x	Proportion children dead	Estimates of $q(i_x)$ using models				Estimates of $q(1)$ in models				Estimated time reference[b] (general model)
			Latin American	Chilean	General	West	Latin American	Chilean	General	West	
15-19	1	.1046	.1070	.1181	.1073	.1113	.1070	.1181	.1073	.1113	1.04 (1976.5)
20-24	2	.1194	.1277	.1301	.1265	.1279	.1036	.1178	.1055	.1059	2.25 (1975.3)
25-29	3	.1419	.1474	.1484	.1462	.1453	.1083	.1275	.1113	.1115	3.69 (1973.8)
30-34	5	.1674	.1762	.1741	.1744	.1727	.1166	.1406	.1202	.1216	5.55 (1972.0)
35-39	10	.1980	.2135	.2071	.2118	.2077	.1275	.1566	.1316	.1338	7.74 (1969.8)
40-44	15	.2229	.2356	.2307	.2349	.2311	.1336	.1662	.1377	.1413	10.36 (1967.1)
45-49	20	.2471	.2576	.2558	.2588	.2542	.1371	.1721	.1412	.1452	13.66 (1963.8)

[a] The values of PAR1 (.1444) and PAR2 (.4088) used in the regression equations predicting the multipliers were based on *total* births rather than on sex-specific births. Thus, the multipliers as well as the time reference estimates ought to be the same for male and female.

[b] The calendar periods to which the estimates apply are shown in parentheses.

TABLE 13A. ESTIMATES OF $q(i_x)$ AND $q(1)$ FOR FEMALES DERIVED FROM APPLICATION OF NEW MULTIPLIERS, PERU, 1969-1970

Age of mother	i_x	Proportion children dead	Estimates of $q(i_x)$ using models				Estimates of $q(1)$ in models				Estimated time reference[a] (general model)
			Latin American	Chilean	General	West	Latin American	Chilean	General	West	
15-19	1	.1563	.1606	.1771	.1610	.1675	.1606	.1771	.1610	.1675	1.06 (1968.4)
20-24	2	.1380	.1480	.1506	.1466	.1483	.1096	.1307	.1127	.1176	2.24 (1967.3)
25-29	3	.1641	.1707	.1718	.1693	.1682	.1107	.1388	.1152	.1202	3.66 (1965.8)
30-34	5	.1669	.1758	.1736	.1740	.1722	.1029	.1327	.1073	.1135	5.50 (1964.0)
35-39	10	.2175	.2346	.2276	.2327	.2284	.1191	.1584	.1240	.1350	7.71 (1961.8)
40-44	15	.2194	.2320	.2272	.2313	.2277	.1138	.1526	.1181	.1264	10.13 (1959.4)
45-49	20	.2642	.2755	.2736	.2769	.2719	.1237	.1672	.1272	.1399	(13.64) (1955.9)

[a] The calendar periods to which the estimates apply are shown in parentheses.

TABLE 13B. ESTIMATES OF $q(i_x)$ AND $q(1)$ FOR MALES DERIVED FROM APPLICATION OF NEW MULTIPLIERS, PERU, 1969-1970[a]

Age of mother	i_x	Proportion children dead	Estimates of $q(i_x)$ using models				Estimates of $q(1)$ in models				Estimated time reference[b] (general model)
			Latin American	Chilean	General	West	Latin American	Chilean	General	West	
15-19	1	.0690	.0709	.0782	.0711	.0739	.0709	.0782	.0711	.0739	1.06 (1968.4)
20-24	2	.1437	.1541	.1568	.1526	.1544	.1230	.1406	.1254	.1261	2.24 (1967.3)
25-29	3	.1578	.1641	.1652	.1628	.1618	.1191	.1407	.1225	.1232	3.66 (1965.8)
30-34	5	.1941	.2049	.2019	.2024	.2003	.1325	.1608	.1367	.1395	5.50 (1964.0)
35-39	10	.2139	.2307	.2238	.2289	.2246	.1363	.1678	.1407	.1450	7.71 (1961.8)
40-44	15	.2211	.2338	.2289	.2331	.2294	.1327	.1651	.1368	.1402	10.13 (1959.4)
45-49	20	.2671	.2785	.2766	.2799	.2749	.1467	.1845	.1511	.1552	13.64 (1955.9)

[a] The values of PAR1 (.1401) and PAR2 (.4041) used in the regression equations predicting the multipliers were based on *total* births rather than on sex-specific births. Thus, the multipliers as well as the time reference estimates ought to be the same for males and females.

[b] The calendar periods to which the estimates apply are shown in parentheses.

produce trends that are more consistent across surveys but it is also in agreement with estimates of mortality obtained from birth histories. In fact, a comparison of the estimates of infant mortality derived from the 1977-1978 birth histories (first column of table 14) with the trends from tables 12a and 12b, reveals a striking consistency when the Latin American model is used but much less so when the other models are used; the roots of the mean square deviations are, respectively, .0098, .0109 and .0176 for the Latin American, general and West models. In the case of males the values are .0122, .0183 and .0229 respectively. A similar ordering of the magnitudes of errors is preserved when the birth-history-based estimates are compared with the trends derived from the 1969-1970 indirect estimates of $q(1)$.

Application of the techniques based on unconventional age groups provided estimates nearly identical to the ones already presented. That implies that age heaping, if it exists, is not introducing large distortions. However, application of duration models yielded surprisingly different results. Table 15 displays the estimates of $q(i_x)$ from the duration and age models. It is quite apparent that, at least among the first three or four estimates, rather large discrepancies exist. The discrepancies do not seem to be a function of mortality models although their magnitudes might well be associated with them. Note also that the differences cannot be accounted for by discrepancies in the dating of the estimates. In fact, the time periods to which the first three or four estimates apply are very similar in the age and duration models. There are good reasons to suspect the quality of the duration rather than the age data. In fact, partial evidence appears to confirm that misstatement of the dates of first marriage (union), account for all or almost all the differences.[12] For that reason it seemed advisable to discard the duration-based results and instead utilize those derived from the

TABLE 14. SUMMARY OF TRENDS IN INFANT MORTALITY IN PERU, 1950-1977

Birth cohort	Birth history value of $q(1)$ (survey, 1977-1978)	Time period	1969-1970 survey				1977-1978 survey			
			Latin American	Chilean	General	West	Latin American	Chilean	General	West
					Females					
Before 1950	.2066	—	—	—	—	—	—	—	—	—
1950-1954	.1551	1952.5	.1298	.1842	.1342	.1500	.1383	.2053	.1434	.1605
1955-1959	.1226	1957.5	.1214	.1652	.1257	.1375	.1286	.1851	.1332	.1472
1960-1964	.1071	1962.5	.1131	.1462	.1172	.1251	.1188	.1649	.1231	.1338
1965-1969	.0988	1967.5	.1047	.1272	.1087	.1126	.1090	.1447	.1129	.1205
1970-1974	.1008	1972.5	.0964	.1082	.1001	.1002	.0992	.1245	.1028	.1071
After 1974	.0958	1976.5	.0987	.0930	.0933	.0902	.0914	.1083	.0947	.0964
Rate of decline (× 1,000)			1.67	3.80	1.72	2.49	1.96	4.04	2.03	2.67
Value in 1965.5			.1081	.1348	.1121	.1176	.1129	.1528	.1170	.1258
					Males					
Before 1950	.1837	—	—	—	—	—	—	—	—	—
1950-1954	.1719	1952.5	.1520	.2006	.1608	.1667	.1819	.2419	.1887	.1975
1955-1959	.1368	1957.5	.1417	.1733	.1425	.1463	.1648	.2152	.1707	.1776
1960-1964	.1418	1962.5	.1314	.1461	.1241	.1260	.1478	.1885	.1527	.1576
1965-1969	.1166	1967.5	.1212	.1188	.1057	.1057	.1307	.1618	.1347	.1377
1970-1974	.1131	1972.5	.1109	.0916	.0873	.0854	.1136	.1350	.1167	.1178
After 1974	.0913	1976.5	.1027	.0698	.0726	.0691	.0999	.1137	.1022	.1018
Rate of decline (× 1,000)			2.06	5.45	3.68	4.07	3.42	5.34	3.60	3.98
Value in 1965.5			.1253	.1297	.1131	.1138	.1375	.1724	.1419	.1457

NOTE: Values for each year in the column labelled "Time period" were obtained from an estimated straight line passing through the points formed by the values of reference times and their corresponding $q(1)$ values. The procedure of estimation used was grouped means, and the first observation was discarded. The estimated parameters of the straight line appear at the bottom of the table (rate of decline and value in 1965.5).

TABLE 15. COMPARISON OF ESTIMATES OF $q(i_x)$ OBTAINED FROM DURATION AND AGE MODELS, FEMALES, PERU, 1977-1978

i_x	Estimates based on age model			Estimates based on duration model[a]			Time references	
	Latin American	General	West	Latin American	General	West	Duration	Age
2	.1327	.1315	.1329	.0901	.0833	.0871	7.98	2.25
3	.1504	.1492	.1482	.1253	.1251	.1227	3.66	3.68
5	.1506	.1491	.1476	.1630	.1617	.1579	5.95	5.65
10	.2003	.1987	.1949	.1821	.1807	.1729	7.99	7.74
15	.2146	.2140	.2105	.2118	.2112	.2004	10.18	10.35
20	.2586	.2598	.2510	.2581	.2598	.2439	15.06	13.66

[a] Obtained using equations for multipliers based on duration models.

conventional age model. Furthermore, owing to the internal and external consistency of the corresponding estimates, we have selected the estimated trends derived from the WFS data which is consistent with the Latin American model. According to that, the level of infant mortality in Peru *circa* 1975 was around 91 per thousand births for females and 100 per thousand births for males. In addition, the pace of decline for male infant mortality was close to 3.42 deaths per thousand births per annum whereas for females it reached a value just below one half of the male rate (1.96 deaths per thousand births). It should be noted that the estimate of total infant mortality (96 per thousand births) that we arrive at is about 20 per cent lower than the estimate obtained from the prospective interviews of the Encuesta Nacional de Fecundidad.[13] According to the final report of that survey, the figure obtained therein is very likely to be subject to sampling errors of substantial magnitude given the low number of births and deaths during the period of observation.

Adult mortality

The sources for studying adult mortality are fewer in number, and perhaps lower in quality, than those available for the study of mortality in infancy and childhood. An official count of population for 1972 and registered deaths during the years 1969-1972 constitutes the first source. The 1961 and 1972 censuses in combination with an estimate of mortality in early childhood and an assumption about the model pattern of mortality constitutes the second source. Retrospective questions on orphanhood and widowhood status introduced as part of the Encuesta Nacional of 1975 and as part of the Mortality Module of the WFS are the third and fourth source, respectively. Finally, the

death counts obtained from the prospective part of the same survey constitute the last source of available information. Regrettably, the new information on orphanhood and widowhood status from the 1975 survey was not available to us. We had access only to the mortality tables constructed from such data, all of which incorporate a great number of transformations and smoothing and thus make comparison with other sources somewhat misleading. The information on widowhood status and paternal orphanhood collected as part of the mortality module of the WFS gave inconsistent results and was not used.

Finally, the death rates directly obtained in the prospective part of the Encuesta Nacional of 1975 did not reveal a very smooth age-specific profile, probably owing to the low number of cases involved. We shall use, however, a smoothed age pattern of mortality, if only to produce evidence for the level of mortality. The precise topography of the age pattern of mortality is very likely masked by a structure externally imposed into the raw data for purely cosmetic purposes.

We shall limit our analysis to female adult mortality. First, we will use the maternal orphanhood data by applying to it the transformations yielding female conditional survivorship from age 25 on. Secondly, those estimates will be transformed into an approximate trend of adult female mortality and then compared with the age pattern of mortality estimated from official death counts adjusted for underregistration. Finally, we will summarize briefly the consistency between adult mortality and mortality in infancy and childhood.

Table 16 displays the proportion of non-orphans by quinquennial age group, the corresponding conditional probabilities of surviving from age 25 to age $25+n$, and the life expectancy at birth consistent with such probabilities in four models of mortality (Latin American, Chilean, general and West). In addition, the last column displays the time references of each estimates. Those appear to be appropriate since there are strong indications of a mortality decline (note the presence of a downward slope in the sequence of life expectancy, regardless of model). In order to determine estimates for 1965 and 1970, we have fitted a linear trend through group means to the life expectancies for the first six age groups of respondents. The conditional probabilities of surviving corresponding to the life expectancies thus determined appear in the first eight columns of table 17. The ninth column of the table displays the conditional probabilities of surviving calculated from registered deaths adjusted for underregistration for the period 1969-1971. Note that there is a close correspondence between the values in that column and those in the first four columns: if anything, the latter seem to be slightly higher in most of the age range examined. By and large, the agreement is stronger for the general and West models, but the differences are so small that discrimination in the behaviour of the three models is virtually impossible. Note also that the values derived from vital statistics fall right in the middle of the range established between the estimates derived from orphanhood for the years 1970 and 1965. Thus, it may be that the reference period for the estimates should be one or two years more recent than indicated.

To summarize: the estimated trends of infant mortality with transformations within the Latin American model appear to be more consistent, internally and externally, than those estimated using other models. The results from the 1977-1978 World Fertility Survey indicate that female infant mortality around 1975 was close to 91 per 1,000 live births and that the level of male infant mortality was about 100 for the same period. Adult female mortality estimated from maternal orphanhood data is quite consistent with the independently derived survival curve (based on adjusted vital registration).

According to those results, can we say that the Latin American pattern fits well the experience of Peru? By and large, two of the other models considered here, the

TABLE 16. ESTIMATES OF PROBABILITIES OF SURVIVING FROM AGE 25 TO $25+n$ DERIVED FROM MATERNAL ORPHANHOOD DATA, PERU, 1977-1978

Age group	N	Proportion non-orphan	Conditional probabilities of surviving in models[a]				Life expectancies in models[b]				Time reference[c]
			Latin American	Chilean	General	West	Latin American	Chilean	General	West	
15-19	20	.9301	.9277	.9293	.9298	.9249	62.6	62.8	63.3	64.5	8.9 (1968.6)
20-24	25	.8885	.8878	.8896	.8907	.8853	61.0	61.6	62.1	62.0	11.0 (1966.5)
25-29	30	.8159	.8171	.8183	.8194	.8160	57.4	58.5	59.1	58.7	13.1 (1964.4)
30-34	35	.7450	.7484	.7489	.7501	.7494	56.7	58.2	58.9	58.1	14.3 (1963.2)
35-39	40	.6395	.6416	.6403	.6409	.6456	54.7	56.5	57.4	56.4	15.6 (1961.9)
40-44	45	.5165	.5098	.5060	.5073	.5174	53.2	55.4	56.2	55.1	16.1 (1961.4)

[a] Obtained using equations suggested by the National Academy of Sciences. See *Manual X: Indirect Techniques for Demographic Estimation* (United Nations publication, Sales No. E.83.XIII.2).

[b] Values are obtained by seeking through linear interpolation the life table consistent with the model-specific conditional probability of surviving. They are intended to be indices of the level of adult mortality and should not be taken as indicators of total levels of mortality.

[c] The calendar periods to which the estimates apply are shown in parentheses.

TABLE 17. COMPARISON OF FEMALE CONDITIONAL PROBABILITIES OF SURVIVING FROM AGE 25 TO 25 + n OBTAINED FROM DIFFERENT SOURCES

	Estimates for 1970[a]				*Estimates for 1965*[a]				*Estimates from adjusted vital registration 1969-1971*[b]
N	*Latin American*	*Chilean*	*General*	*West*	*Latin American*	*Chilean*	*General*	*West*	
10	.9759	.9761	.9753	.9720	.9626	.9655	.9649	.9607	.9646
15	.9589	.9594	.9585	.9542	.9380	.9425	.9421	.9370	.9413
20	.9424	.9376	.9351	.9325	.9096	.9143	.9147	.9094	.9153
25	.9111	.9085	.9084	.9043	.8751	.8784	.8796	.8756	.8864
30	.8756	.8696	.8684	.8654	.8313	.8321	.8325	.8307	.8525
35	.8253	.8147	.8119	.8122	.7718	.7687	.7678	.7712	.8080
40	.7537	.7378	.7323	.7358	.6903	.6834	.6788	.6881	.7442
45	.6508	.6284	.6225	.6288	.5793	.5672	.5637	.5770	.6591
50	.5172	.4933	.4821	.4845	.4426	.4302	.4219	.4335	.5508
$^{o}e_{0}$	64.81	64.43	64.70	65.97	59.10	59.98	60.56	60.54	

[a] The values in the first eight columns were obtained by calculating expected values of e_0 in the trends fitted to the data in table 1b. The fitting procedure, the grouped-means technique, was applied to the first three and last three values in each column of table 15.

[b] Estimates obtained from a life table which was constructed using registered deaths for the period 1969-1971 and the census of 1972 projected backwards to 1970. The registered deaths were inflated by a factor of 1.25 to correct for estimated underregistration. The procedure to calculate the level of underregistration is described in detail in S. H. Preston and K. Hill, "Estimating the completeness of death registration", *Population Studies*, vol. 34, No. 2 (1980).

general and West, did not produce results markedly different when they were separately applied to mortality in infancy and mortality past the age of 25. Yet neither of them, including the best fitting (Latin American), could reproduce the joint mortality levels of adults and children. In fact, whereas on the one hand, to reproduce the female experience of mortality around 1970 above age 25 one needs a life table consistent with a life expectancy of around 65, on the other hand, the estimated levels of female infant mortality are consistent with a life table having a life expectancy of about 52.0. The conclusion is then that the Peruvian female mortality can only be reproduced by splicing together two different levels within the Latin American model.

CONCLUSION

The introduction of new models of mortality is intended to provide a representation of age/sex patterns of mortality which are closer to those underlying the mortality experience of developing countries. Their validity depends exclusively on the scope of representation of the societies on which they are based and on the accuracy of the base life tables. Their utility depends on the gains in precision that are obtained when they are used to smooth observed patterns, project populations or to estimate entire life tables from incomplete information. In the present paper we have tried to implement procedures that permit the identification of mortality levels in infancy, early childhood and adulthood from appropriate survivorship statistics. Those procedures rely entirely on the new United Nations models of mortality and demonstrably have a better performance than those based on other models of mortality. That conclusion is naturally contingent on the intrinsic validity of the new mortality models: if they are better representations of actual mortality patterns in developing countries, then (and only then) will the parameters estimated in the present paper provide a basis for more accurate transformations of survivorship statistics. Whether or not the new United Nations models of mortality are in fact a better representation of patterns of mortality found in developing countries is a matter that will be confirmed only through the systematic comparisons of results originating in sources of information that do not depend on assumptions about the mortality pattern with those that are model dependent. As the exercise applied to Peru showed, such contrasts do not always lead to clear-cut conclusions. Nevertheless, they provide added support for the estimate (or set of estimates) obtained as outcome of the exercise. In general, it should be a routine practice to try out several "plausible" models—selected on the basis of geographical, ecological or historical considerations—and to compare the final estimates of global parameters (such as the life expectancy at age x) or partial indicators (such as infant mortality) with other independently obtained values, and select the one producing the most consistent results.

Throughout the present paper we have also emphasized the quite different issue of not just selecting between appropriate mortality models but also between appropriate techniques. Thus, for example, the choice between "duration" vs. "age" models may be substantially more important than is usually believed. That is because the net balance between the potential advantage derived from stronger fitting power of duration models and actual disadvantage owing to misrepresentation of marriage duration does not always unequivocally tilt in one direction or the other. By the same token, the choice between estimation models depending on the assumption of constancy in mortality should be tested by examining the estimates themselves and contrasting them with external (and independent) information.

This presentation of new algebraic transformations to convert survivorship statistics into mortality estimates may make the task of estimation more cumbersome simply because it adds to the available stock of choices. But if the models of mortality on which they are based are valid representations of actual mortality patterns, then it will also make the results of such tasks more reliable.

NOTES

[1] A. Coale and P. Demeny, *Regional Model Life Tables and Stable Populations* (Princeton, Princeton University Press, 1966).

[2] *Model Life Tables for Developing Countries* (United Nations publication, Sales No. E.81.XIII.7).

[3] *Manual X: Indirect Techniques for Demographic Estimation* (United Nations publication, Sales No. E.83.XIII.2).

[4] W. Brass and others. *The Demography of Tropical Africa* (Princeton, Princeton University Press, 1968).

[5] A. Coale and T. J. Trussell, "Model fertility schedules: variations in the age structure of childbearing in human populations," *Population Index*, vol. 40, No. 2 (1974).

[6] K. Hill, H. Zlotnik, and J. Durch, "Simple procedures for reducing the effects of age errors in indirect demographic estimation techniques", paper presented at the annual meeting of the Population Association of America, Denver, 1980.

[7] G. Feeney, "Estimating infant mortality trends from child survivorship data", *Population Studies*, vol. 34, No. 1 (1980).

[8] J. Sullivan, "Models for the estimation of the probability of dying between birth and exact ages of early childhood", *Population Studies*, vol. 25, No. 1 (1972).

[9] K. Hill and T. J. Trussell, "Further developments in indirect mortality estimation", *Population Studies*, vol. 31, No. 2 (1977).

[10] W. Brass and B. Bambogye, "The time location of reports of survivorship: estimates for maternal and paternal orphanhood and the ever-widowed", Working paper No. 81-1, Centre for Population Studies, London School of Hygiene and Tropical Medicine.

[11] A. Palloni, M. Massaglie and J. Marcotte, "Estimating adult mortality with maternal orphanhood data: analysis of sensitivity of the techniques", *Population Studies*, vol. 38, No. 2 (1984).

[12] A. Palloni, M. Tienda and V. Diaz, "Indirect estimates of mortality and fertility in Peru". Mimeograph. 1983.

[13] Instituto Nacional de Estadística, *Encuesta Nacional de Fecundidad* (Lima, 1977).

ANNEX I

Simulation of the data set used to estimate the regression coefficients for transforming the proportion of children dead into probabilities of dying

1. *Constant mortality: age models*

In each of the five models of mortality, a life table with a life expectancy (e_0) of 54 years was used to generate the proportion of children ever born who would have died at the time of a survey (or census). The probabilities of dying before exact ages x ($x=1,2,\ldots,35$) for both sexes combined were obtained by calculating the weighted averages of the male and female probabilities in the corresponding life tables:

$$q(x) = \frac{1.05\ q(x)^{\text{male}} + q(x)^{\text{female}}}{2.05}$$

That weighted average assumes that the sex ratio at birth is 1.05.

The schedule of probabilities of dying was then combined with a series of 96 schedules of fertility rates obtained from the set constructed by Coale and Trussell[a] to produce proportions of children dead by either age groups of mother or five-year marriage duration groups. The parameters identifying the fertility schedules were

$$a_0 = 13, 15$$

$$k = 17, 19, 21, 23$$

$$m = .0 \text{ through } 2.2 \text{ in intervals of } .2$$

In order actually to calculate the number of children dead, we applied two procedures:

(*a*) For a mother exactly age x at the time of the survey or census, the number of children dead among those born more than one previous to the survey (or census) was computed as follows:

$$d(x,t) = (1-{}_1L_t) \bullet (\bar{f}(x-t)) - \\ -1/12\ (p(t+1)-p(t))(f(x-t-1)-f(x-t))$$

${}_1L_t$ is the probability of surviving up to the middle of the age interval $t,t+1$; $\bar{f}(x-t)$ is the fertility rate centred in the age interval $(x-t-1)$; $x-t$, $p(t+1)$ and $p(t)$ are exact probabilities of surviving up to $t+1$ and t, respectively, and $f(x-t-1)$ and $f(x-t)$ are the fertility rates evaluated at exact ages $x-t-1$, and $x-t$.

(*b*) The number of children dead among those born between 0 and 1 years before the survey (or census) was calculated numerically by evaluating the integral

$$d(x,0) = \int_{t=0}^{t=2} (1-p(t))(f(x-t))dt$$

where $p(t)$ was assumed to follow a Weibull function—namely, $p(t) = \exp(-\lambda t^{\gamma})$ and λ and γ are two parameters estimated directly from the following two equations

$$p(1) = \exp(-\lambda t) \text{ (which yields } \hat{\lambda})$$

$${}_1L_0 = \int_0^1 \exp(-\lambda t^{\gamma})dt \text{ (which yields } \hat{\gamma})$$

The second equation ensures that the estimated Weibull parameters are consistent with the area under the survivorship curve below age 1.

Once the total number of dead children for mothers at exactly age x ($13 \leqslant x \leqslant 50$) was calculated, the number of children ever born that those same mothers produced during the past was calculated. That was done by evaluating the integrals

$$b(x) = \int_{t=0}^{x} f(x-t)dt$$

for every x from 13 to 50.

To calculate the proportion of children dead for 5-year age groups (conventional or unconventional), we took the ratio of the weighted number of children dead to the weighted number of children ever born to women in the corresponding range of ages. The weights were selected from the proportionate distribution of population in a female stable population with an intrinsic growth rate of .030 and the same mortality pattern and level applied to their children. Once the proportion of children dead was calculated it was possible to compute the values of the multipliers by age group of women.

2. *Constant mortality: duration models*

The procedures followed to calculate the multipliers by duration of marriage were analogous to those presented above. However, instead of using the function $f(x)$, we used the marital fertility schedule. As weights we selected the same female stable age structure in conjunction with the proportion of ever married women obtained from the Coale and Trussell models of fertility with the appropriate parameters a_0 and k.

3. *Declining mortality: age and duration models*

In order to simulate the proportion of children dead that would be obtained if mortality had been declining, the following steps were implemented:

(*a*) It was assumed that life expectancy had increased from 35 to 60 years in an interval of 30 years (at a rate of about .83 years per year). That is a rate very close to the one actually experienced in the period after the Second World War by countries in Asia and Latin America. It permitted generation of life tables for each of the years before a survey or census;

(*b*) The period life tables generated above were chained together to calculate cohort-specific probabilities of surviving up to the date of the survey (or census). The theoretical (continuous) expression for that probability for a child born x years before the survey (or census) is

$$p^c(x) = \exp\left[-\int_0^x \mu(v,x-v)dv\right]$$

where $\mu(v,x-v)$ is the force of mortality at age v in the life table corresponding to $x-v$ years before the survey (or census);

(*c*) The sequences of values $p^c(x)$—and the corresponding values of ${}_1L_x{}^c$—were then used in the expressions presented above for the case of constant mortality. The female stable age distribution used as weights was identical to the one applied in the case of constant mortality;

(*d*) Once the proportions of children dead by quinquennial age groups were calculated, the estimates of the probabilities of dying before ages 1,2, . . . , 20 (2,3, . . . , 25 if duration groups were used) were computed using *exact multipliers* (that is, those observed in the case of constant mortality rather than those predicted through regression equations). In each case a search was undertaken to determine the number of years before the survey (or census) to which the estimate applied. Those values, called "reference times", were then regressed on indices of the fertility curve to generate the regression parameters presented in the paper for the case of mortality decline.

NOTE

[a] A. Coale and T. J. Trussell, "Model fertility schedules: variations in the age structure of childbearing in human populations", *Population Index*, vol. 40, No. 2 (1974).

ANNEX II

Simulation of the data set used for the estimation of regression coefficients for transformation of proportion of maternal orphans

To generate the regression equations displayed in table 11, we used simulated populations with the following characteristics:

1. *Constant mortality*

Fertility. Fertility schedules were drawn from those created by Coale and Trussell.[a] The subset utilized was characterized by the following parameters:

$$a_0 = 13, 15$$

$$\text{SMAM} = 17, 19, 21, 33$$

$$m = .0, .20, \ldots, 2.2$$

In all, 96 fertility schedules were utilized.

Mortality. Survival functions were drawn from each of the five patterns in the United Nations model life tables. In each case, seven levels of mortality were selected corresponding to life expectancies of 40, 45, 50, 55, 60, 65 and 70.

Combination of mortality and fertility schedules. The formulae to evaluate the proportion of non-orphans in each age group required the calculation of the rate of growth (r). When mortality remains constant, that rate of growth corresponds to the intrinsic rate of growth of the stable population with a given fertility and mortality schedule and with a given gross reproduction rate (GRR). In all our calculations GRR was set equal to 3.0. When mortality is declining, the age structure of mothers, $n(a,t)$, at each time t was calculated directly by projecting forward, on a year-by-year-basis, a stable population characterized by a given fertility schedule and the mortality level applicable to 30 years before the hypothetical survey or census.

Evaluation of formulae. All formulae utilized in the paper involve at least one integration. Integration of functions was carried out numerically by breaking them down into integrations over one unit (year) intervals. They were evaluated, taking the functions at the midpoint of the one unit (year) interval and adjusting them upwards (downwards) according to the slopes of the functions involved within the interval. For example, the integral

$$\int_\alpha^\beta n(s,t)\, f(a)\, \frac{p(a+t)}{p(a)}da$$

was evaluated using the summation, over α and β, of the quantities

$$\overset{*}{n}(a,t)f(a)\frac{\overset{*}{p}(a+t)}{\overset{*}{p}(a)} - \frac{1}{12}(\text{slope 1})(\text{slope 2})$$

where $\overset{*}{n}(a,t)$ is the number of women in the age group a, $a+1$ at time (exact) t, $\overset{*}{f}(a)$ is the fertility rate in the age interval a, $a+1$, $\overset{*}{p}(a+t)/\overset{*}{p}(a)$ is the conditional probability of surviving from the middle of the age group a, $a+1$ to the age group $a+t$, $a+1+t$. Slope 1 refers to the slope of the functions $n(a,t) \cdot f(a)$ in the interval a, $a+1$. Similarly slope 2 refers to the slope of the function $\frac{p(a+t)}{p(a)}$ in the interval a, $a+1$. In cases in which evaluation of multiple integrals was required, the same procedure was followed; of course, some of the functions were integrals themselves.

Declining mortality. To simulate mortality decline, six different rates of increase in life expectancy over an interval of 30 years were generated:

Decline (type)	*Initial life expectancy*	*Terminal life expectancy*	*Rate (per year)*
1	35	45	.33
2	35	50	.50
3	35	55	.67
4	35	60	.83
5	35	65	1.00
6	35	70	2.17

Corresponding to each type of mortality decline there was a set of life tables applicable to each year before an hypothetical survey or census. Cohort schedules of survival for mothers were constructed by chaining together the conditional probabilities of surviving over the one-year intervals. A similar procedure was followed to obtain cohort schedules of survival for children. In the text the term "slow" decline refers to decline Type No. 2, the term "intermediate" decline refers to decline Type No. 4 and the term "fast" decline refers to decline Type No. 6.

NOTE

[a] A Coale and T. J. Trussell, "Model fertility schedules: variations in the age structure of childbearing in human populations", *Population Index*, vol. 40, No. 2 (1974).

MORTALITY AND DEVELOPMENT REVISITED*

*Samuel H. Preston***

SUMMARY

The period from the 1930s to the 1960s was one of unprecedented mortality declines in developing countries. In cross-sectional analyses of factors which may be responsible for that rapid improvement in survivorship, Preston (1975, 1980) demonstrated that only about 30 per cent of the improvement could be explained by measurable aspects of social and economic development such as income, literacy and nutrition. The remaining 70 per cent of improvement was explained by variables omitted in his analysis. Preston, as many others previously, postulated that those omitted variables were exogenous, non-development-related factors such as anti-malarial programmes, immunization and other vertical governmental and international programmes, and improvements in personal health practices.

A more sluggish pace of mortality decline apparently characterized the decade that began in the middle of the 1960s. The purpose of the present paper is to carry out an analysis similar to the earlier ones but concentrated on the later period. The analysis provides insights into the factors that accounted for the slow pace of mortality change and suggests directions for governmental and international efforts to increase survivorship. The study finds that, contrary to previous periods, the social and economic variables of income, literacy and nutrition were the dominating factors in explaining mortality decline during the 1965-1969 to 1975-1979 decade. That greater relative role does not result from faster improvements in social and economic conditions during the recent period or from an increased responsiveness of mortality to social and economic variables. Rather, the exogenous factors appear to have operated with sharply reduced intensity in the more recent period. Reduced international commitment to health in developing countries may be one explanation; surely Governments and international agencies continue to have many tools available for improving health. Results also suggest the major role that can be played by educational change in fostering mortality gains.

INTRODUCTION

The present paper is an attempt to update results reported in two earlier papers regarding the role of socio-economic factors in world-wide mortality declines since the 1930s. Preston (1975) demonstrated that the relationship between life expectancy at birth and per capita income (in constant dollars) had shifted between the 1930s and 1960s. A country at a particular level of national income per capita was estimated to have a level of life expectancy at birth that was, on average, 9.7 years higher in the 1960s than it would have been in the 1930s at the same level of income. That shift was clearly attributable to factors other than measured income gains, although it is possible that measured gains are biased indicators of true gains.

In an attempt to identify the contribution of advances in literacy and nutrition to the apparent shift, Preston (1980a) added those variables to income in regression equations estimated with data on 36 countries around 1940 and 120 countries around 1970. For the less developed countries (LDCs), the shift in the relationship between 1940 and 1970 was estimated to be 8.8 years after those variables were introduced along with income (Preston, 1980a:307). Thus, literacy and nutritional gains were responsible for relatively little of the shift. That new shift factor accounted for about half of the 18-year gain in life expectancy that was estimated to have occurred between those approximate dates (World Health Organization, 1974). However, the estimate of the structural shift did not include data on China, whereas China was included in data of the World Health Organization (WHO) on LDC life-

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expectancy gains. China, which represents 30 per cent of the total LDC population, had made remarkably rapid progress, having gained about 30 years in e_0 between those dates (Banister and Preston, 1981). LDCs excluding China had gained an average of only about 12 years in e_0 during the same period. So the 8.8 year shift represented around 70 per cent of the total gain for the group of LDCs excepting China. That is, about 70 per cent of the mortality decline among LDCs during the 1940-1970 period could not be accounted for by measurable gains in income, nutrition or literacy.

Those results seem sufficient to show that mortality trends are not dominated by a simple mechanistic input-output relation between mortality and other prominently observed features of the economy and society. While relations between mortality and those development indicators are very tight at both the household and the national levels, other factors are clearly playing a role, and it's the un-named factors which have been dominant in producing change during the period from the 1940s to 1970.

As Schultz (1979) quite correctly notes, exercises such as the above only help to rule out factors such as income, nutrition and literacy as the dominating force in mortality change but are not helpful in defining the factors that were actually operative. What are the factors? Preston (1980a) suggests that anti-malarial campaigns were a leading source of the shift, since they were cheap to implement (and often subsidized by international donors), widespread and evidently quite effective. But he notes that slight changes in statistical procedures have a large impact on the estimated size of malaria's contributions. Subsequently, he suggested, without convincing demonstration, that anti-malarial activities were the largest source of the gains, followed by immunization campaigns and improvements in personal health practices (Preston, 1980b).

Later work, though cross-sectional, would seem to underscore the importance of personal health practices. A microlevel study of child mortality in 15 developing countries identified mother's education and her ethnicity as the strongest predictors of child mortality (Mensch and others, 1984). The greater importance of mother's education than of father's education clearly points towards the importance of child-rearing practices in the household; ethnic variations in such practices are the most plausible explanation of the sizeable mortality differences among ethnic groups. At the same time, the unimportance of urban/rural residence in that study suggests that conventional health facilities, which are usually heavily biased towards urban areas, are not playing a major role in the developing country mortality picture. Such a conclusion is supported by Mosley (1983), who argues that governmental health care programmes have been largely ineffective because of administrative shortcomings and biases towards inappropriate western-style medical technology. He argues that educational gains are a more plausible explanation of recent mortality change in Kenya, although they cannot have been the dominant factors in advances over a broad range of countries.

It should be noted that knowledge of proper health care practices can spread from person to person, or from a "central source" such as the mass media, without any necessary involvement of the formal educational system. Such a diffusion is doubtless widespread, though difficult to quantify. In-depth interviews with women in the Sine-Saloum region of Senegal reveal their great interest in learning about proper child-care practices and a considerable degree of medical sophistication, probably much greater than was the case a generation earlier. One woman is quoted as saying, "The doctors on the radio say that mosquitoes give diseases; I have forgotten which ones but they tell us to use mosquito screens." (Garenne and van de Walle, 1984:8). Another, when asked "Do mosquitoes make people sick?", responded, "The radio says they give malaria". (Garenne and van de Walle, 1984:7).

Mortality declines since 1965

There have been many suggestions that the pace of mortality improvement in LDCs slowed from the middle of the 1960s to the middle of the 1970s (e.g., Hansluwka, 1975; Gwatkin, 1981). Such a decline would coincide with a period of lagging commitments of international aid for health purposes, especially for anti-malarial programs (Preston, 1980a). In a careful investigation of the question, the United Nations (1982) concluded that, for the 24 LDCs that could supply reasonably reliable mortality information in the 1950s, 1960s and 1970s, the average gain in life expectancy was .57 years per calendar year between the 1950s and 1960s and .49 years per calendar year between the 1960s and 1970s. Relaxing the restriction that a country have data at three times and admitting countries with only two observations, the study finds that the average pace of change declined from .61 years per year to .43 between the two decades. That relatively small decline in the rate of mortality advance was essentially confined to Latin American countries and some of the decline there was attributed to several countries' having reached high levels of life expectancy where rates of advance are normally slow.

The mildly pessimistic picture that emerged for the relatively few LDCs with adequate data requires emendation for developing countries as an aggregate. As noted earlier, China has had a remarkably rapid mortality decline, and much of it was concentrated in the period since the early 1960s. Recently released age distributions from the 1953 and 1964 censuses suggest that life expectancy at birth for the intercensal period was extremely low, probably around 44 years (Coale, 1984). The Great Leap Forward setbacks of 1959-1961 may have contributed to that low level. By 1981, life expectancy had risen to about 68 years (Coale, 1984), a gain of more than one year of life expectancy per calendar year over a period of more than two decades. Weighted averages of LDCs gains are highly

influenced by the rapid Chinese progress and therefore show faster progress than unweighted figures.

Unfortunately, we are not able to deal with China in the present paper because it lacks pertinent developmental indicators. We shall deal instead with the remaining 70 per cent of the developing world where, in the aggregate, there are indications of a slowdown in the pace of mortality change during the 1960s and early 1970s. Our goal is to estimate the amount of shift in the relation between mortality and other development indicators during the period from 1965-1969 to 1975-1979. The results will provide a reading on whether the shift in the relation has kept pace with the average shift of about three years in life expectancy per decade which was recorded during earlier, longer periods.

A wide variety of procedures was used to investigate the issue. In all cases, a mortality indicator was used as the dependent variable in a cross-national regression analysis that includes data from LDCs and from developed countries. The latter were included because there is overlap in all distributions (Southern and Eastern Europe having similarities to parts of South America and Western South Asia) and because they provide more stability to regression lines. In all cases, the set of independent variables included some transformation of the following: the percentage of adults who were literate; gross domestic product per capita in constant dollars; and the excess of per capita daily calories supplied above 1,500 (approximately the amount required to meet minimum metabolic demands). Data were drawn from standard United Nations, UNESCO and World Bank compendia. It should be stressed that the estimates, particularly for 1975-1979, are subject to large amounts of measurement error. For example, it is likely that fewer than half of the developing countries would have produced direct information on mortality conditions during 1975-1979 by 1982, when the United Nations data were published. When data were missing, the United Nations used various rules of thumb, empirical regularities and typical relations among groups of countries to fill the breach. We have no reason, however, to feel that the mortality (or other) estimates are systematically biased.

A number of alternatives were introduced into the estimation:

(*a*) Life expectancy at birth vs. infant mortality rates. The United Nations (1982b) recently completed a large project to estimate infant mortality rates for all countries since 1950. Therefore, it is possible for the first time to examine relations between an important age-specific mortality rate and various development indicators;

(*b*) Conventional measures of GDP per capita versus International Comparison Project (ICP) measures. The former measures are based upon international exchange rates, which are subject to many distortions. The latter measures are based on purchasing-power parities of various currencies, estimated by applying relations found in 16 intensively investigated countries to a much wider group of countries (Summers and others, 1980);

(*c*) Functional forms of equations. It is not obvious that any particular functional form of the regressions is to be preferred over all others, so several of the most attractive candidates were used. The forms includes log-linear and logistic equations estimated separately for 1965-1969 and 1975-1979. They also include equations estimated on the basis of pooled cross-sectional data for the two dates;

(*d*) Weighted and unweighted regressions. It is possible that smaller countries have greater variance in their mortality estimates, in which case weighted regressions would reduce heteroskedasticity.

Results using life expectancy

The unweighted regression equations for life expectancy in 1965-1969 and 1975-1979, using the same functional form used previously, are:

$$\underset{1965\text{-}1969}{e_0} = \underset{(6.651)}{5.506} + \underset{(0.486)}{2.684 ln Y} + \underset{(.017)}{.238 LIT} + \underset{(2.349)}{2.854 ln CAL} \quad \bar{R}^2 = .919, \; N = 106$$

$$\underset{1975\text{-}1979}{e_0} = \underset{(7.417)}{8.090} + \underset{(0.562)}{2.325 ln Y} + \underset{(.018)}{.230 LIT} + \underset{(1.452)}{3.012 ln CAL} \quad \bar{R}^2 = .899, \; N = 104$$

where Y = per capita GDP in 1975 U.S. dollars
LIT = percentage literate of adult population
CAL = excess of daily calorie supply per capita above 1,500.

Standard errors of coefficients are shown in parentheses; *ln* indicates that a natural log transformation was used.

The equations appear remarkable for their similarity. Each coefficient is in the expected positive direction and each is significant at the .05 level in both years. There is very little change in the size of the coefficients between the two dates.[1] The intercept rises, but cannot itself be directly interpreted because the zero point on *ln Y* and *ln*CAL is far beyond the range of observation. If we take a prototypical poor developing country with a Y of 300, LIT of .5, and CAL of 500, the predicted e_0 in 1965-1969 is 50.45 years and in 1975-1979 it is 51.56 years. The "shift" for such a country is 1.11 years during the 10-year period, only about a third of that which would have been expected on the basis of the pace of change in the earlier period. (The shift was 8.8 years for the 30-year 1940-1970 period.) Note that since coefficients on variables change, the "shift" is not constant across countries but depends upon their conditions. That is, it will not be represented by the intercept alone but by the entire set of regression coefficients.

Similar results are found when alternate specifications and data are used. Substituting ICP data

on gross domestic product in 1970 dollars (designated Y^*) into the equations gives

$$1965\text{-}1969\ e_0 = \underset{(6.473)}{-4.466} + \underset{(0.718)}{4.002 ln Y^*} + \underset{(.019)}{.219\text{LIT}} + \underset{(1.184)}{3.141 ln\text{CAL}}\ \bar{R}^2 = .920,\ N = 106$$

$$1975\text{-}1979\ e_0 = \underset{(6.358)}{0.264} + \underset{(0.762)}{4.113 ln Y^*} + \underset{(.020)}{.199\text{LIT}} + \underset{(1.310)}{2.621 ln\text{CAL}}\ \bar{R}^2 = .911,\ N = 104$$

Repeating the above exercise for our prototypical developing country with the ICP-based regressions gives an expected e_0 of 48.89 in 1965-1969 and 49.96 in 1975-1979. The "shift" is 1.07 years, close to the 1.11 value estimated above. For a country with a per capita income of $800, a literacy percentage of 75 and daily caloric availability of 2,400, the shift is (60.51 − 60.15) = 0.36 years, suggesting that the shift has been even smaller for the better-off among LDCs.

It is interesting to note that the coefficient of Y^* is about 50 per cent higher than the coefficient of Y for the same period, and that more variance is explained in regressions using Y^*. Similar results are maintained throughout the paper. An obvious explanation of that result is that the ICP estimates of Y^* contain less measurement error than the exchange rate-based estimates of Y, since measurement error in an independent variable biases coefficients downwards. One concrete way in which that occurs in that Y^* raises income estimates for lower-income countries relative to higher-income countries, thus helping to linearize the relationships between e_0 and the log of income. The results help to confirm widespread suspicions that Y^* is generally a superior series, but they are hardly definitive.

A number of equations based upon pooled data from the two periods were estimated. Results are presented in table 1. In equations without terms involving interactions between time period and other covariates, the coefficient of the shift variable is .84 years when Y is used and .73 years when Y^* is used (equations 1 and 4). Both coefficients are insignificant at a 5 per cent level, preventing the rejecting of the hypothesis that no across-the-board shift had occurred between the dates. However, though insignificant, the coefficients remain the best estimate of the amount of across-the-board shift that occurred. (Note that the estimate includes developed countries.)

When an interaction term (the product of the shift dummy and Y or Y^*) is added to the equations, the coefficients of the intercept shift term increase in size and become significant (equations 2, 5, 6 and 7), even though the interaction term itself is significant only in equation 7.[2] Evidently, the failure of the shift dummy to be significant in equations 1 and 4 resulted from the fact that the shift was quite small for the high income developed countries. In order to illustrate the amount of shift that occurred as a function of income level, we substitute arbitrary income values into the equations.

Predicted amount of gain in e_0 between 1965-1969 and 1975-1979 that is exogenous to income, literacy and caloric availability for countries at 1975-1979 per capita GDP levels of

	$150	$400	$650	$900
Unweighted, exchange-rate-based income (eq. 2)	1.38	1.29	1.20	1.11
Unweighted, ICP-based income (eq. 5)	1.23	1.12	1.10	.90
Weighted, exchange-rate-based income (eq. 6)	1.54	1.51	1.47	1.43
Weighted, ICP-based income (eq. 7)	1.46	1.36	1.26	1.16

Source: Compiled from table 1

For the range of incomes between $150 and $900, the shift is usually estimated to be in the range of 1.0-1.5 years of life expectancy at birth during the decade. The shift is larger than that estimated without the interaction term, presumably because the shift was very small for the developed countries. The pace of the shift is still only one third to one half of that observed during the earlier period. The shift is estimated to be somewhat greater when weighted regressions are used, suggesting that larger countries may have benefited more than smaller ones from the exogenous factors.

More complex functional forms were also used to estimate the shift factor. A logistic function fitted by unweighted maximum likelihood procedures is

$$e_0 = \frac{46.96 + .2567\text{LIT} + 1.07\ T}{1 + \exp\{-.9226 = .00146\ Y - .00048\ \text{CAL}\}}$$

All coefficients are significant at the .05 level. The shift factor can be estimated by substituting values of Y and CAL into the equation. For Y = $500 and CAL = 500 (i.e., 2,000 calories per day), the denominator is 1.07, so that moving from $T = 0$ to $T = 1$ (i.e., advancing the observation from 1965-1969 to 1975-1979) would increase e_0 by 1.07/1.07 = 1 year, *ceteris paribus*. The logistic form thus retains the basic result observed above.

Results using the infant mortality rate

Very similar results are achieved when life expectancy at birth is replaced by the infant mortality rate. Coefficients for all regressions for 1965-1969 and 1975-1979 have the correct sign, although there is somewhat less stability in coefficients than when e_0 is used. Coefficients of Y^* continue to exceed in absolute value the coefficients of Y. The following are ICP-based regression equations for the two periods.

$$1965\text{-}1969\ \text{IMR} = \underset{(39.70)}{329.58} - \underset{(4.403)}{10.135 ln Y^*} - \underset{(7.260)}{13.413 ln\text{CAL}} - \underset{(0.114)}{1.326\text{LIT}}\ \bar{R}^2 = .877 = 106$$

$$1975\text{-}1979\ \text{IMR} = \underset{(36.75)}{282.83} - \underset{(4.406)}{13.675 ln Y^*} - \underset{(7.573)}{4.178 ln\text{CAL}} - \underset{(0.013)}{1.268\text{LIT}}\ \bar{R}^2 = .873 = 106$$

TABLE 1. COEFFICIENTS OF LIFE EXPECTANCY REGRESSIONS FOR POOLED CROSS-SECTIONAL DATA, 1965-1969 AND 1975-1979

	Constant	Coefficient of shift dummy 1 if observation for 1975-1979, 0 otherwise	Coefficient of ln Y or ln Y*	Coefficient of LIT	Coefficient of ln CAL	Coefficient of interaction term shift dummy times Y or Y*	Coefficient of interaction term, shift dummy times LIT	R^{-2}	N
Unweighted regressions									
				Using exchange rate GDP					
1	6.855[a]	0.835[a]	2.486	.236	2.863	—	—	.914	212
	(4.890)	(.492)	(0.363)	(.012)	(0.927)				
2	4.309[a]	1.436	2.866	.232	2.913	−.00036[a]	—	.914	212
	(5.028)	(0.577)	(0.410)	(.012)	(0.921)	(.00018)			
3	4.336[a]	1.573[a]	2.850	.233	2.913	−.00034[a]	−.0029[a]	.913	212
	(5.043)	(1.027)	(0.422)	(.015)	(0.923)	(.00022)	(.0180)		
				Using ICP estimates of GDP					
4	−2.328[a]	0.734[a]	3.910	.213	2.966	—	—	.918	215
	(4.433)	(0.475)	(0.506)	(.013)	(0.859)				
5	−4.994[a]	1.302	4.220	.211	3.079	−.00045[a]	—	.918	215
	(4.853)	(.637)	(.556)	(.013)	(0.862)	(.00033)			
Weighted regressions									
				Using exchange rate GDP					
6	14.563	1.567	3.223	.194	1.378[a]	−.00015[a]	—	.936	211
	(4.116)	(0.482)	(0.430)	(.013)	(.896)	(.00013)			
				Using ICP estimates of GDP					
7	1.625[a]	1.522	5.880	.161	.863[a]	−.00040	—	.952	211
	(3.120)	(0.446)	(0.490)	(.012)	(.691)	(.00019)			

[a] Insignificant at .05 level.

All coefficients are significant at 5 per cent except those pertaining to the calorie variable.

The variance explained by the IMR regressions is somewhat less than that for life expectancy regressions, probably reflecting the fact that socio-economic variables cannot capture significant determinants of infant mortality such as breast-feeding and weaning practices nor variations in the definition of what constitutes a live birth and an early infant death.[3] The importance of breast-feeding may also explain the instability and lack of significance of calorie availability as an influence on IMR, although that is highly speculative.

For a country with a Y^* of 300, LIT of .5, and CAL of 500, the predicted IMR in 1965-1969 is 122.1/1000 and in 1975-1979 it is 115.5/1000. For a country with $Y^* = 800$, LIT = 75, and CAL = 900, the predicted IMR is 71.1/1000 in 1965-1969 and 67.9/1000 in 1975-1979. Again, the shift appears to be larger for the poorer country. Table 2 makes that tendency more explicit by pooling data from the two periods and adding to the equation an intercept shift term and an interactive term involving period and income. The

TABLE 2. COEFFICIENTS OF INFANT MORTALITY RATE REGRESSION FOR POOLED CROSS-SECTIONAL DATA, 1965-1969 AND 1975-1979

	Constant	Coefficient of shift dummy	Coefficient of ln Y or ln Y*	Coefficient of LIT	Coefficient of ln CAL	Coefficient of interaction term shift dummy times ln Y or ln Y*	R^{-2}	N
Unweighted regressions								
				Using exchange rate GDP				
	283.70	−17.15[a]	−9.770	−1.349	−6.993[a]	1.937[a]	.880	211
	(29.88)	(14.59)	(2.414)	(0.0715)	(5.366)	(2.193)		
				Using ICP estimates of GDP				
	316.05	−15.82[a]	−12.415	−1.303	−9.384[a]	1.759[a]	.878	211
	(29.49)	(19.73)	(3.540)	(0.080)	(5.212)	(2.935)		
Weighted regressions								
				Using exchange rate GDP				
	227.65	−18.41	−11.432	−1.321	2.811[a]	1.850[a]	.939	211
	(19.84)	(8.19)	(1.973)	(0.062)	(4.140)	(1.236)		
				Using ICP estimates of GDP				
	275.45	−22.13[a]	−15.095	−1.280	−0.650[a]	2.477	.939	211
	(17.24)	(11.74)	(2.671)	(0.066)	(3.745)	(1.732)		

[a] Insignificant at .05 level.

intercept shift term is insignificant in three of four regressions and the interactive term is always insignificant. Nevertheless, all terms have the expected sign and imply that a shift has occurred that is larger for poorer countries:

Predicted amount of drop in IMR (per 1,000) between 1965-1969 and 1975-1979 that is exogenous to income, literacy and calorie availability for countries at a 1975-1979 per capita GDP level of

	$150	$400	$650	$900
Unweighted, using exchange-rate GDP	7.4	5.5	4.6	4.0
Unweighted, using ICP GDP	7.0	5.3	4.4	3.9
Weighted, using exchange-rate GDP	9.1	7.3	6.4	5.8
Weighted, using ICP GDP	9.7	7.3	6.1	5.3

Source: Compiled from table 2.

The predicted declines of 4=9/1000 in IMR resulting from the shift in the relationship are highly consistent with the predicted gains in e_0 of 1 to 1.5 years that were described during 1965-1979, a gain of one year in e_0 is typically associated with the reduction of 4.8/1000 in IMR.[4]

Discussion

We have estimated that life expectancy at birth increased by 1 to 1.5 years between 1965-1969 and 1975-1979 as a result of factors other than income, literacy and calorie availability. To narrow that range to a single average figure, we calculate the mean of variables in 1965-1969 and 1975-1979. For the 84 LDCs with data on all variables for both dates, the (weighted) means are:

	1965-1969	1975-1979
Life expectancy at birth	48.6	52.5
ICP per capita GDP (Y^*)	$477.7	$642.2
Percentage literate of adults (LIT)	40.7	48.2
Daily calories per capita (1,500 + CAL)	2,132	2,226

Life expectancy at birth increased, on average, by 3.9 years. The amount of the change attributable to the shift in the relation between e_0 and other developmental indicators can be estimated by taking the mean of the 1965-1969 and 1975-1979 values of the independent variables and substituting those means into the regression equations for each period separately. Those equations, using Y^*, are presented above. The predicted value of life expectancy in 1965-1969 is 51.14 years and in 1975-1979, 52.23 years. Thus, the shift in the relation accounts for 1.09 years out of the average increase of 3.9 years. The balance of 2.81 years of gain is thus attributable to improvements in income, literacy and caloric consumption.

An alternative way of calculating the shift is to use the regression estimated for both periods combined, including a shift term and a period-income interaction (equation 5 in table 1). At the mean income for 1965-1969 and 1975-1979 of $560, the shift is 1.05 years, very similar to the above result. However, using weighted regression (equation 7 in table 1), the shift factor is 1.30 years. The result again indicates that larger countries may have benefited disproportionately from whatever factors are operating to induce the shift.

Thus, there is good agreement among the alternative methods that the shift factor accounted for about 1.1-1.3 years of gain in e_0 between 1965-1969 and 1975-1979. That is a substantial reduction from the average pace of shift of about three years per decade that was recorded for earlier periods. The picture may be altered when more data for 1975-1979 and later years are added, but there is no reason to feel that such data are more likely to increase the estimated pace of the shift than to decrease it.

If the previously observed shift of years in e_0 per calendar year had continued during the period from 1965-1969 to 1975-1979, then an additional annual gain of .17 to .19 years in e_0 would have been registered, bringing the average yearly gain from the shift factor to 0.3 years of e_0. That would raise the average annual total gain in e_0 to .39 + (.17 or .19) = .56 or .58. The gain in e_0 during the period 1950-1955 to 1965-1969 occurred at an average rate of .58 years per calendar year (United Nations, 1982c). So the reduced rate of shift in the relation between life expectancy and other development indicators seems to be exclusively responsible for the apparent deceleration in the rate of mortality advance. The endogenous factors seem to have been operating with much the same force as in earlier decades.

What factors are responsible for the slowdown in the pace of mortality decline from "exogenous" factors? Since those factors themselves have not been identified with any precision, it is impossible to demarcate clearly their varying roles. Of the two major factors that have been suggested—governmental programmes and the diffusion of good health knowledge and practices—the former seems more capable of abruptly changing in intensity. As noted above, there is some evidence that governmental programmes have lagged. Part of the reason is that some of the easier gains, especially from anti-malarial programmes in Asia and Latin America, were already registered before 1965. Malaria in Africa is a far less tractable problem, and the rate of progress has been very slow (Molineaux, 1983). Finally, the considerable decline in real terms of international aid for health from the largest donor, United States Agency for International Development (USAID), may be mentioned as a contributing factor to the slowdown.

Although some of the easiest gains have already been scored, there is no question that a battery of programmatic approaches, demonstrably successful in carefully controlled settings, are still available for implementation.[5] In considering ways to advance health, however, Governments should not neglect the importance of socio-economic factors. One of the striking conclusions of the present paper, confirming earlier results at both the micro and macro levels, is the extremely powerful role of literacy in determining

a population's level of mortality. The coefficient of literacy in life expectancy regressions is robust .2, suggesting that moving from 0 to 100 per cent literacy would increase e_0 by about 20 years, *ceteris paribus*; alternatively, it would reduce the infant mortality rate by about 130/1000. Since universal literacy can be achieved for about 1-4 per cent of national income per capita (Lewis, 1968), it is clear that programmes to advance education—especially, judging from micro studies, for education for women—are potentially one of the most cost-efficient ways to improve mortality in developing countries, although the effects obviously operate with a lag of, on average, about 20 years. How does that effect compare to that of income gains *per se*? The use of ICP-based estimates of GDP per capita has increased the income coefficient relative to earlier estimates, and the new coefficients of 4-5 suggest that a 1 per cent increase in GDP per capita would raise e_0 by about .04-.05 years. That still represents a relatively small effect. If that hypothetical 1 per cent gain in income were used to increase literacy, the ultimate increase in e_0 is estimated to be (25 to 100) $\times$ .2 = 5-20 years, at least 100 times greater than the effect of income gain by itself.[6] Since that literacy gain operates with a lag, discounting would reduce its relative size, but the result would still be an increase that is more than an order of magnitude greater for literacy increase than for income growth.

Notes

[1] Except for the intercept, the equation is also very similar to one estimated for 1940 in Preston (1980a:306):

$$e_0 = -13.103 + 5.435 lnY + .165 LIT + \underset{(18.510)}{} \underset{(2.386)}{} \underset{(.063)}{}$$
$$+2.949 lnCAL \qquad \bar{R}^2 = .845$$
$$(3.718) \qquad N = 36$$

In that equation Y is measured in 1970 (rather than 1975) dollars, which affects the intercept of the equation.

[2] Interaction terms between the shift dummy and the log of Y or Y^* were also used but resulted in slightly lower R^2s and lower significance levels for the interaction terms than interactions involving untransformed versions of Y or Y^*.

[3] As evidence of the activity of these special factors, Coale and Demeny (1966) find IMR to have generally the lower correlation with other age-specific death rates in an international collection of life tables.

[4] The relation is derived from "West" female model life tables (Coale and Demeny, 1966: 15-16).

[5] The literature here is vast. For reviews, see Walsh and Warren (1979) and Gwatkin and others (1980). On immunization specifically, see Foege (1983); on anti-malarial programmes, see Molineaux (1983).

[6] That calculation clearly deals with first-order effects only and neglects the effect of literacy changes and the investment required to achieve them on income and consumption streams.

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A RE-EVALUATION OF LEVELS AND TRENDS OF MORTALITY IN EAST AFRICA

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SUMMARY

The present study deals with the estimation of trends of infant, child and adult mortality in Kenya, Uganda and the United Republic of Tanzania. The data used by the author are census data. He drew heavily on new techniques, including methods for estimating mortality from age distribution when fertility and/or mortality are not stable, methods for using orphanhood data from two censuses, a method for adjusting child survival data for differences in infant mortality by birth order, and an approach to estimating simultaneously the trends and the age patterns of child mortality.

The estimated trends show that all three countries experienced substantial declines over the period for which data are available. While Kenya has continued to have a substantially lower infant mortality rate than the United Republic of Tanzania, the gap between Uganda and Kenya seems to have virtually disappeared between 1950 and 1965.

A surprising result of the analysis is the extent to which 1948 and 1957-1962 censuses provide useful data. Although there are cases in which early census data are clearly useless, on the whole in the countries reviewed they seem to provide more useful data than was expected.

INTRODUCTION

Recent improvements in indirect methods for estimating mortality and data from the recent round of censuses provide new opportunities for estimating levels and trends of mortality in East Africa. The new techniques include methods for estimating mortality from age distributions when fertility and/or mortality have been changing, techniques for using orphanhood data from two censuses, a method for adjusting child survival data for differences in infant mortality by birth order, and an approach for estimating simultaneously the trends and the age patterns of child mortality. The analysis of the previous round of East African censuses was completed before the development of techniques for estimating the time period to which each estimate from the child survival and orphanhood techniques apply.

The data from the recent round of censuses have just become available from the Kenya census of 1979 and the United Republic of Tanzania census of 1978. No data are available from the 1979 census of Uganda.

The paper reviews the trends in infant and child mortality in each of the three countries first, and then discusses adult mortality.

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INFANT AND CHILD MORTALITY IN KENYA

The earliest national estimate of mortality in Kenya is the infant mortality rate estimated from the 1948 census. The census asked each woman how many children she had borne and how many of them had died during the first year of life. Women aged 16-45 reported an infant mortality rate of 184 per 1,000 which applies to the years surrounding 1940. Although early writers felt that might be a high estimate (Martin, 1953), it is quite consistent with the estimates derived below.

Later censuses provide much more useful information from which to estimate mortality. The censuses of 1962, 1969 and 1979 all provide data on child survival by age of mother. Our analysis uses two new approaches. The first method adjusts the child survival estimate for the fact that infant mortality varies with birth order and age of mother. For example, the births reported by women aged 25-29 include an unusually high proportion of second- and third-order births which have lower mortality risks than first-order births and births of order 5 and higher. Therefore the estimate of mortality based on the reports of women aged 25-29 may understate the true level of mortality. A method of adjusting for that was developed for Bangladesh (Ewbank, 1982) and has been extended for use in countries for which estimates of infant mortality by birth order are not available (Ewbank, 1983).

The second method applied to the Kenya data is an attempt to estimate simultaneously the trends in mortality and the age pattern of child mortality. The estimated level of mortality can be very sensitive to the choice of an age pattern in two ways. The first is that the multiples used to estimate the proportion dying by a given age, $q(x)$, from the reported proportion deceased among the children born to women in a given age group depend on the assumed age pattern of mortality.

The second effect of the assumed age pattern occurs when the estimates of survival to various ages are translated into estimates of infant mortality (or any other common index of mortality) so that they can be compared for consistency and trends. For example, an estimated $q(5)$ of 200 becomes an estimated IMR of 119 using Coale and Demeny's North model and 134 using their West model. That effect increases in importance as x, the age of the $q(x)$ estimate, increases. The second effect is far more important than the first when we are concerned with a discussion of time trends in mortality.

In order to derive estimates of the age pattern of mortality, we have used a logit model of mortality to select the age pattern which leads to the greatest consistency between estimates of mortality for the same time period derived from data from different censuses. A preliminary estimate of the age pattern parameter in Brass's logit system, β, was derived for each of the 41 districts of Kenya using a regression of the form:

$$Y(x) = \alpha + \beta\, Y_s(x) = \gamma t \qquad (1)$$

where $Y(x)$ is the logit of the estimate of $q(x)$ from the child-survival technique adjusted for birth-order differences in infant mortality, $Y(x)$ is the logit of the standard life table (Coale and Demeny's North level 12 with a sex ratio at birth of 1.03) and t is the year to which the estimated $q(x)$ applies. The estimates of t and the child-survival estimates are both based on the estimation equations for the North model presented by Hill, Zlotnik and Trussell (1983). (For the whole country that equation, with the West level 12 as the standard, produces virtually identical estimates of infant mortality.)

Equation (1) is misleading in its simplicity for two reasons. First, the parameters in (1) cannot be estimated accurately from the data from a single census or survey because the high correlation between the $Y_s(x)$ and the t estimates leads to multicollinearity and instability in the parameter estimates. However, when two or three censuses are available the correlation between the $Y_s(x)$ and the t drops enough that reliable estimates of β and γ are possible.

The second problem with equation (1) is that it can be misleading if the trends in mortality have not been linear over the whole time period or if some of the $q(x)$ estimates are unreliable. Four tests have been made for each of the 41 districts of Kenya to insure that the estimate of β is reasonable. First the estimated value of $q(2)$ from the 1969 census applies to approximately the same time period as the $q(15)$ estimate from the 1979 census. Therefore those two points provide an estimate of β which is not affected by mortality trends. The same is true for the 1969 estimate of $q(3)$ and the 1979 estimate of $q(20)$. Those two pairs of $q(x)$ values give two estimates of β which should be similar to the regression estimates from equation (1). A second test involves plotting the estimates of the infant mortality rate for various periods using each of the $q(x)$ estimates and the value of β estimated from equation (1). In several districts the plots showed that mortality had not been decreasing steadily over the period covered by the child survival estimates. In those cases it was generally possible to re-estimate equation (1) using only those $q(x)$ estimates for the time periods when mortality appears to have been declining at a regular pace or remaining constant.

A third test of the reasonableness of the estimates is a comparison of the estimated β values from child survival with β estimated from a linking of child survival and orphanhood estimates. Although the age patterns of child and adult mortality need not be consistent with the same β given a particular standard, there were several cases in which that test reinforced the β estimate from equation (1). For example, the estimates of β for child mortality in Meru district from equation (1) was 1.19, significantly higher than the values for most districts (generally below 0.95). That estimate gains credence when we note that the β for females derived by linking child and adult survival also leads to an unusually high β of 1.10. The fourth test of the estimates of β was a comparison of the estimates for contiguous districts inhabited by similar groups.

The first step in examining the child survival data for each district was a regression estimate of β from equation (1). Each of the regressions gave a weight of 10 to the $Y(x)$ from the 1979 census because the 1962 and 1969 censuses collected child survival data from only 10 per cent of the women in any district. Those estimates of β were then compared with estimates from nearby similar districts and with estimates of β from adult survival. Finally the trend in infant mortality implied by the β estimate and the $q(x)$ values was plotted.

Since the district boundaries changed following the 1962 census, it was not possible to include the 1962 data in the regressions for many of the districts. In some cases it was possible to test the reliability of the estimates by grouping districts. For example, we estimated β and the trends in IMR for Kisumu and Siaya districts from the censuses and compared them with the data for Central Nyanza district in 1962. The use of the 1962 census data allows for additional tests, for example, estimating β from the $q(2)$ estimate from 1962 and the $l(10)$ estimate from 1969 which apply to the same year.

In a few cases it was not possible to estimate β because of obvious underreporting of deceased children in the 1962 and 1969 censuses. Those districts tend to be in sparsely populated parts of the country where it is safe to assume that there have not been

significant trends in mortality, and therefore we can assume that γ is equal to 0.0. Problems also arise for the two urban districts, Nairobi and Mombasa. Because of continuous in-migration from areas with elevated mortality, the child survival reports reflect the levels and trends in both the city and the areas which send migrants. For Nairobi and Mombasa, the estimate of β was selected by examination of the estimates for surrounding areas.

Table 1 presents estimates of the infant mortality rate (IMR) for 1939, 1949, 1959, 1969 and 1979 for each of the seven provinces and for the country as a whole. The national estimates show that mortality has declined quite steadily from about 184 in 1941 to 85 in 1979, reaching less than half of its 1940 level by about 1975. That amounts to an average annual decline of about 2.0 per cent per year. The estimates for 1939 are very similar to the estimates for 1949 derived from the 1962, 1969 and 1979 censuses, which suggests that the mortality decline may have begun during the middle of the 1940s. If we date the start of the national decline in 1949, then the annual rate of decline from 1949 to 1979 is 2.5 per cent. The estimates for Rift Valley are probably underestimates, especially for 1949. An estimate for 1949 of 160 would raise the national estimate for 1949 by about 7 points.

TABLE 1. INFANT MORTALITY RATES FOR PROVINCES OF KENYA, 1941-1979

			Infant mortality rate		
Province[a]	1939[b]	1949	1959	1969	1979
Central	130	128	101	66	45
Coast	—	169	149	133	123
Eastern	—	156	119	91	71
Northeast	—	144	128	117	105
Nyanza	228	224	164	146	116
Rift Valley	—	119	102	87	74
Western	—	205	152	122	105
Nairobi	—	80	70	71	61
TOTAL	184	160	125	105	84

[a] Provincial boundaries as they were set after 1963.
[b] Unpublished data from the 1948 Census.

The estimates show substantial differences among the rates and the rates of decline for the provinces. Nyanza province along Lake Victoria and Western province along the border with Uganda have had declines from over 200 to almost 100. Central province to the north of Nairobi has apparently reached an infant mortality rate of 45, which is probably one of the lowest for a non-urban area in sub-saharan Africa. The most disappointing trend is for Coast region which shows very little decline between 1949 and 1979, dropping from 161 to 123.

INFANT AND CHILD MORTALITY IN THE UNITED REPUBLIC OF TANZANIA

The child mortality data from the United Republic of Tanzania were analysed by using newly developed techniques that are slightly different from those used to analyse the Kenya data. The approach depends on a return to the original integration equations that are the basis of the Brass techniques rather than depending on the various sets of multipliers that have been used to simplify those methods. Returning to the original equations has several advantages. The first is that it is possible to adjust for differences in infant mortality by maternal age by incorporating in the integrations child survival rates that are specific for maternal age. In order to do that, we have used a standard schedule of differences in infant mortality by maternal age based on data from prospective studies in several countries. Although that standard schedule is probably not perfect for East Africa, it is probably much better than the assumption that there are no differences by maternal age.

A second advantage is that we can derive mortality estimates based on non-standard age groups and for women aged 50-54 and 55-59. The method therefore increases the usefulness of the data from the 1948 and 1959 censuses which include only two age groups of women: 16-45 and 46 and over. The availability of estimates based on the reports of women 50-54 and 55-59 increases the number of cohorts for which two consecutive censuses provide estimates and therefore assists in the estimation of the parameters in equation (1). Although the estimates based on the reports of older women often seem to involve underreporting of deceased children, in many cases they provide valuable information on mortality trends.

The return to the original integration equations also makes it possible to estimate the cohort to which each estimate applies. Therefore the estimation of the age pattern and the trend in mortality assume a cohort trend rather than the time trend that was used to analyse the Kenya data.

In the logit analysis of the Tanzanian data using equation (1) (with the T for time replaced by C for cohort), the standard life table for each region was selected from the Coale and Demeny North model tables to match the level of $q(5)$ in the period immediately preceding the 1967 census. That differs from the analysis of the Kenya data, where level 12 was used for all districts. The procedure should produce slightly better estimates since the simple logit transformation does not reflect fully the changes in the age pattern of mortality that typically accompany changes in the level of mortality. Tests with the data from Kenya suggest that the use of those different approaches would not alter the estimates for Kenya substantially.

The 1948 and 1957 censuses of the United Republic of Tanzania (then known as Tanganyika) inquired about children ever-borne and children surviving in a sample of areas. Women were also asked whether deceased children had died before or after their first birthday. The data used here are those for two age groups, women 16-45 and 46 and over, since exact age was reported by a minority of women. The proportion deceased reported by women aged 16-46 provides an estimate of $q(9)$ for the period of about 10 years

before the census data. For women 16 and over, the proportion deceased is approximately equal to $q(13)$ for the period of about 14 years before the survey.

Full data on child survival are available from the 1967 and 1978 censuses and the 1973 National Demographic Survey. However, the 1973 data appear to understate child survival.

Table 2 presents estimates of the infant mortality rate for each region of the United Republic of Tanzania for 1955, 1965 and 1975. Each of the regions is defined according to the boundaries at the time of the 1978 census. The estimates for each region are based on the use of equation (1) or similar estimation equations which incorporate estimates of changes in the rate of decline in mortality. For example, the data for many regions suggest quite clearly that the decline in infant and child mortality began in the late 1940s or early 1950s. For those regions, the birth year of the cohort was replaced in equation (1) by the year in which the mortality decline began if the cohort was born before that date.

The estimation equation was used to compare the estimates from the 1948, 1967 and 1978 censuses for each of the regions as they were defined in 1978. The 1959 census was not included because estimates are available only for larger geographical areas. District data from the 1948 and 1967 censuses were used to construct estimates for the 1978 regions. In a few cases the 1948 census data were more consistent with the 1978 data than were the 1967 data. The use of the 1948 data were therefore quite useful in choosing between the 1967 and 1978 data when they were not consistent.

The estimates of infant mortality shown in table 2 show a great deal of variation in both the levels and the trends. The most significant regional difference is the concentration of low mortality areas in the northeastern part of the country and the concentration of high mortality along the southern border and in the southwest. The lowest mortality rates are those in Coast, Tanga and Zanzibar regions along the northeastern coast and Kilimanjaro, Arusha and Mara regions along the northern border with Kenya. All of them have infant mortality rates below 105. The highest infant mortality rates (between 150 and 170) are found in Iringa, Lindi, Mbeya and Rukwa regions.

TABLE 2. ESTIMATES OF INFANT MORTALITY FOR THE UNITED REPUBLIC OF TANZANIA, 1955-1975

Region	Infant mortality rate			Percentage change, 1955-1975
	1955	1965	1975	
Arusha	106	103	103	2.8
Coast	123	116	103	15.9
Dar es Salaam	130	115	100	23.1
Dodoma	137	122	109	20.0
Iringa	200	174	151	24.7
Kigoma	199	162	130	34.8
Kilimanjaro	106	86	69	35.6
Lindi	159	159	159	.0
Mara	153	127	104	32.0
Mbeya	169	169	169	.0
Morogoro	163	150	137	16.0
Mtwara	136	164	137	−.9
Mwanza	159	138	120	24.5
Rukwa	172	164	156	9.1
Ruvuma	194	171	149	23.0
Shinyanga	131	125	137	−4.0
Singida	181	147	118	34.5
Tabora	141	139	135	4.5
Tanga	108	104	100	7.3
West Lake	193	133	113	41.7
Zanzibar	143	117	117	18.2
TOTAL	148	134	122	17.7

The national estimates show a decline from 146 in 1955 to 120 in 1975 with a constant rate of decline of about 1 per cent per year. Those estimates differ substantially from earlier estimates. For example, the United Nations estimated that the IMR declined from 173 in 1950-1955 to 119 in 1970-1975, which leads to an annual rate of decline of 1.9 per cent. The estimates given here agree substantially with the United Nations estimates for the period around 1967, but are quite different for earlier and later periods. The reason for those differences is that, until the publication of the 1978 census data and without access to the unpublished data from the 1948 census, it was virtually impossible to estimate the rate of decline in the IMR. The present analysis shows that the rate of decline in infant mortality has been substantially slower than previous estimates suggest.

The estimated rate of decline varies substantially among the regions with declines of less than 5 per cent over the period 1955-1975 in Arusha, Lindi, Mbeya, Mtwara, Shinyanga and Tabora. On the other hand, Kigoma, Kilimanjaro, Mara, Singida and West Lake had declines of more than 30 per cent.[1]

INFANT AND CHILD MORTALITY IN UGANDA

The 1948 census sample of Uganda provides reports from women aged 16-45 on the proportion of their children who died before their first birthday (Martin, 1953:195 and unpublished detailed data by district) and data on the proportion of children deceased from women aged 18-22, 23-27, 33-37 and 43-47. Similar data are available for all four provinces from the 1959 census. The 1959 census also provides data for each province for the standard five-year age groups for women who reported their exact age; however, the census analytical volume states that only in Buganda province was a real effort made to get exact ages for all women.

In general the data from the three censuses are very consistent for each of the four provinces. The estimates show that in 1955 Buganda, Eastern and Western provinces all had infant mortality rates of about 140 while Northern province had a rate of about 165. By 1965 Buganda's rate had dropped to about 91 while Eastern and Western provinces had IMRs of about 115 and 108. The rate in Northern province had declined to about 133. Altogether, Uganda's infant mortality rate declined from about 146 in 1955 to 112 in 1965, a rate of decline of 2.7 per cent per year. Those estimates are

slightly lower than previous estimates made by the United Nations Populations Division.

Techniques for estimating adult mortality

There are four basic types of information about mortality over age 20 which are available for East Africa. The first is data on deaths by age reported in the vital registration system, a census or a single round survey. (The vital registration systems in East Africa are so incomplete that the data are not useful for estimating mortality levels or trends. Data on reported deaths in the previous 12 months from censuses and surveys are frequently affected by serious reporting errors, such as extensive overcounting, as in the 1967 Census of the United Republic of Tanzania, or severe undercounting, as in the 1973 National Demographic Survey of that country.) The second source is reports of the survival of parents by persons of various ages. Those orphanhood data are available for all three countries from various censuses and surveys. The third source of information is data on whether women of various ages have ever been widowed. Those data do not seem to provide useful estimates for East Africa, probably because of the complications introduced by remarriage. Finally, mortality can be estimated from age distributions for two different dates.

For East Africa the most useful types of data on adult mortality are the parental survival data and the comparison of age distributions. Those two types of data are the basis for a number of different estimation techniques, each of which has been used to estimate life expectancy at age 20, e_{20}. Some of the techniques are well known (for example, the census survival technique and the basic orphanhood techniques), while others are very new and unpublished (for example, Preston's intercensal orphanhood methods). Therefore, before proceeding to a discussion of the data, we will describe each of the techniques that we have applied. The descriptions include only a brief discussion of the data requirements, the basic logic, and the advantages and disadvantages of each technique.

Census survival

In a population which is not affected by migration, the population over age 10 at one census is the survivors of the population 10 years earlier. Therefore we can estimate mortality by using model life tables and selecting the level of mortality which correctly projects the population aged 10 and over from a census 10 years previous. However, that is complicated by age-misreporting and age-selective errors of enumeration. Therefore, in practice the population over age x is projected from the population over age $x-10$ from the previous census for several values of x. Then for each age x, a level of mortality is selected and the final choice is the median of those estimated levels.

Preston/Bennett method

The Preston/Bennett method is one of several which uses the idea of age-specific growth rates to study population dynamics. If a population's birth and death rates have been constant, there has been no migration and the population size has been constant, then the age distribution is given by the ${}_nL_x$ column in the life table. In such a population, a single age distribution will provide an estimate of the life table. If fertility and/or mortality have been changing, we can use two age distributions to remove the effects of past changes by calculating age-specific growth rates, the intercensal growth rate of each age group (Preston and Bennett, 1983). Using Preston and Bennet's equation for e_x we calculate e_{20} as:

$$e_{20} = \int_{20}^{\infty} \frac{N(a)}{N(20)} \exp\left[\int_{20}^{a} r(x)dx \right] da$$

where $N(a)$ is the population at age a at the mid-point between the censuses, and the $r(x)$ are the age-specific growth rates.

Preston's integrated method

Preston (1983a) has shown that

$$p(y)\frac{\exp(-\int_0^a r(x)dx)}{c(a)} = \frac{1}{b} + \frac{K}{b}\frac{{}_yq_s(a)}{{}_yp_s(a)}$$

where $c(a)$ is the proportion of the population at age a, ${}_yq_s(a)$ is the proportion dying between ages y and a in a standard mortality schedule, ${}_yp_s(a)$ is $1-{}_yq_s(a)$, and $p(y)$ is the estimate of the proportion surviving to age y in the population being studied. In addition b is the population crude birth rate and K is e^{β} where β relates the population life table to the standard using the logit system. Given age distributions from two censuses, $1/b$ and K/b can be estimated using regression analysis. K then provides an estimate of the level of mortality.

Brass-Hill orphanhood method

The questions on parental survival can be turned into estimates of survival from an age B near the mean age of parents at the birth of a child to age $B+x$ where x is the mean age of respondents in age group. That transformation was done using multipliers calculated by Hill (Brass, 1975:68, 81-83). Those estimates of l_{x+B}/l_B and an estimate of l_{20} can be used to estimate the slope of the logit line relating the real-life table to a standard-life table. The procedure is similar to that described by Hill, Zlotnik and Trussell (1983). The estimated l_{20} was selected to apply to the same time period as the l_{x+B}/l_x estimated from the procedure developed by Brass and Bamgboye (1981). Therefore the estimates of β are unaffected by time trends in mortality. For all of the applications described below, the survival from B to early ages (e.g., x values of 22.5, 27.5 based on the reports of people aged 20-24 and 25-29) leads to unrealistically low values of β. The survival to higher ages (e.g., $x+B$ values of 70 and 75) leads to much more reasonable values. Although some variation is noted below, in

general, the maternal orphanhood data lead to values of β around 0.8, while the values for males are higher–about 1.05 for the United Republic of Tanzania and 1.15 for Kenya. One disadvantage of that method is that the most reliable estimates apply to the period of about 12-15 years preceding the census date.

NAS intercensal orphanhood

The methods manual prepared by Hill, Zlotnik and Trussell (1983) presents a method for using data on orphanhood from two censuses to estimate mortality for the intercensal period. The method is based on the development of a set of proportions orphaned by age for a synthetic cohort by chaining together the ratios of the proportions with a surviving parent at the first census who still have a surviving parent at the second census. The main advantage of that orphanhood technique is that the resulting estimates apply to a period near the date of the most recent census (five years in the case of decennial censuses).

Preston's intercensal orphanhood method

Preston has recently developed an intercensal orphanhood method which is based on the age-specific growth rates of the total population and the population of persons with a surviving parent. If the population in a given group who have a surviving mother is increasing faster then the general population of that age group, then mortality among adults must be declining. We can use that fact to remove the effect of the trends in adult mortality on the proportion with a surviving mother (or father) on estimates of adult mortality for the intercensal period.

Preston's integrated intercensal orphanhood method

Preston has shown that the population of people with a surviving mother (or father) can be studied using age-specific growth rates in a manner similar to the study of the whole population. The population with a surviving mother is affected by two mortality rates, the mortality of the population and the mortality of the mothers. If we are willing to assume a model life table, we can sort out those two components of mortality. The method is improved if we consider only the population above some early age, y–say, age 5 or 10. By ignoring the earliest age groups, we reduce the problem of persons with an adopted parent reporting a surviving parent. That approach requires an estimate of the proportion surviving to age y which we can derive from the Brass child survival analysis. That method therefore requires orphanhood data from two censuses and Brass child-survival data from the second census.

In the application of all of the orphanhood methods, we have used only the responses of females about the survival of parents. The reason for that is that the age reporting of women is probably less affected by age misreporting in the age groups of most interest, ages 30-49. For those methods which require a model life table, we have used Coale and Demeny's North model 12 transformed by a logit model with a estimated from the basic orphanhood methods.

ADULT MORTALITY ESTIMATES FOR KENYA

Table 3 presents a series of estimates of e_{20} for males and females for the intercensal period, roughly 1974. It also summarizes the data used to derive each estimate. The estimates for sexes combined is simply the average of the estimates for the two sexes, since the estimates of e_{20} for the sexes differ by approximately 3 per cent, which simply compensates for the sex ratio at birth.

The estimates fall into two distinct groups. The first includes the Preston/Bennett estimate and the Preston integrated-method estimates. Those procedures give estimates of e_{20} for the total population of about 42 years. The other group includes the census-survival

TABLE 3. ESTIMATES OF ADULT MORTALITY, e_{20}, BY VARIOUS METHODS, KENYA, 1974

	Data used for technique						
	Age distribution	*Orphanhood*		*Child survival*	e_{20}		
	1969-1979	*1969*	*1979*		*Males*	*Females*	*Total*
Preston integrated method							
No child survival	X				43.0	41.6	42.3
$p(5)$	X			X	43.0	41.3	42.1
$p(20)$	X			X	44.0	38.8	41.4
Preston/Bennett	X				42.6	42.0	42.3
Reverse survival	X				44.7	46.7	45.7
Integrated orphanhood extrapolated, using child survival trend		X	X	X	42.5	48.7	45.6
Preston intercensal orphanhood	X	X	X	X	46.4	46.9	46.6
NDS integrated intercensal orphanhood		X	X		43.3	49.9	46.6
Preston integrated intercensal orphanhood							
$p(5)$		X	X	X	47.0	48.6	47.8

estimate and all of the methods which make use of the orphanhood data. Those estimates cluster around an e_{20} of 47. The latter group seems to be more reliable for several reasons. First, the pattern of age-specific growth rates used to calculate the Preston/Bennett and the Preston integrated estimates do not seem reasonable. In particular, the growth rate for the 10-14, 15-19 and 20-24 year age groups are unreasonably high, ranging from 3.86 per cent for males 10-14 to 4.88 per cent for females aged 15-19. That pattern may be due to a combination of differential underreporting and age-misreporting in the two censuses. However, the same pattern appears in the data for the United Republic of Tanzania. The census survival method which uses the same data (the age distributions from the 1969 and 1979 censuses) seems to be less affected by that problem. The most likely reason is that the final estimate from the census-survival method is the median of several estimated values, some of which are biased downward by that problem while others are biased upward by age exaggeration at the older ages.

Estimates of the trend in adult mortality can be derived using the orphanhood data from the 1969 and 1979 censuses. The 1969 census provides estimates of e_{20} for males and females for the period around 1955 while the 1979 census provides estimates for 10 years later. The intercensal methods provide estimates which apply to about 1974. Table 4 summarizes the estimated trends. Life expectancy at age 20 for sexes combined increased from about 43.0 years in 1955 to 45.3 years in 1965 and 47.0 in 1974. That amounts to an annual increase of 0.23 years per year between 1955 and 1965 and 0.19 years per year between 1965 and 1974. The estimated sex difference ranges from 1.7 years to 3.1 years, with females always having the higher e_{20}.

TABLE 4. ESTIMATES OF e_{20} FOR KENYA, VARIOUS DATES

Year	*Males*	*Females*	*Total*
1955	39.3	43.9	41.6
1965	41.1	46.8	44.0
1974	44.7	48.6	46.7

ADULT MORTALITY IN THE UNITED REPUBLIC OF TANZANIA

The most reliable estimates of adult mortality for the United Republic of Tanzania are based on the orphanhood data from the 1973 National Demographic Survey and the 1978 census. The former provides data on both survival of father and survival of mother, while the latter provides data only on maternal survival. Those data lead to an estimated e_{20} for 1955 of 41 and for 1975 of about 43.

A comparison of the age distribution from the 1969 census with the preliminary distribution for the 1978 census gives estimates of e_{20} for females in 1974 of about 41-44 and an unreasonably high estimate for males about 56.

ADULT MORTALITY IN UGANDA

By linking the orphanhood data from the 1969 census with our estimates of child survival, we estimate that in 1955 e_{20} was 40.0 for females, 40.1 for males and 40.0 for sexes combined. That is 1.6 years lower than the similar estimate for Kenya and 1.1 years below the estimate for the United Republic of Tanzania.

No estimate of the trend in adult mortality will be possible until the 1979 census has been tabulated.

Table 5 summarizes the estimates of e_{20} for the two sexes for 1955 for the three countries. The values of e_{20} are very similar for the United Republic of Tanzania and Kenya (both about 41 years), with a slightly lower estimate for Uganda (about 40).

TABLE 5. ESTIMATES OF LIFE EXPECTANCY AT AGE 20, e_{20}, AND THE AGE PATTERN OF ADULT MORTALITY FOR KENYA, UNITED REPUBLIC OF TANZANIA AND UGANDA, 1955

	Males	*Females*	*Total*
Kenya	39.3	43.9	41.6
United Republic of Tanzania			41.1
Uganda	40.1	40.0	40.0

Table 6 presents estimates of expectation of life at birth, e_0 for the three countries in 1955-1975. Although it has not been possible to estimate mortality for Uganda for 1975, it is not likely that the level of e_0 has increased since 1965. It appears, therefore, that the situation of relatively similar levels of mortality in 1955 in the three countries has been replaced by substantial mortality differentials among the three. While all three countries had life expectancies of about 42 to 54 years in 1955, the range in 1975 was about 46 to 56.

TABLE 6. ESTIMATES OF LIFE EXPECTANCY AT BIRTH,[a] KENYA, UNITED REPUBLIC OF TANZANIA AND UGANDA, 1955, 1965 AND 1975

	1955	*1965*	*1975*
Kenya	45.0	51.0	55.5
United Republic of Tanzania	42	44	45.7
Uganda	43.1	49.5	—

[a] e_0.

The substantial differences among the three countries are echoed in the large differentials within each country. In Kenya, the mortality rates are still quite high along the Coast and around Lake Victoria, and the coastal areas have not experienced substantial reductions in mortality during the period. In the United Republic of Tanzania, there are very large differences between the mortality rates in the north-eastern quarter of the country and the southern half, and those differences have increased over time because of the lack of improvement in the south.

SUMMARY AND CONCLUSIONS

The study of mortality in sub-saharan Africa is still an inexact science, and the study of mortality trends in

the area will always be hampered by the quantity and quality of the historical data. However, constant improvement in methods of analysis and an increasing amount of information make it possible to sharpen our picture of both past and recent trends. In evaluating indirect and incomplete data, the goal is always to abstract a picture of reality which is as consistent as possible with the data and with our preconceived notions of the nature of demographic processes. In our analysis we have relaxed the usual assumptions about the age pattern of mortality and thereby increased the weight attached to the data. It appears from the analysis that slight changes in the assumed pattern of mortality often lead to a series of estimates which are more consistent with the data.

The age patterns of mortality suggested by the data are similar to the frequently used North model tables, as indicated by the fact that the values of β are generally quite close to 1.0.

Although our results are similar to those that would have been reached using the North model, that result was not obvious from the start and was not a necessary outcome. For example, a preliminary examination of the Kenya data using the West model 12 as the standard led to IMR estimates for 1950 and 1970 which differed by about 0.5 per 1,000 from the estimates based on the North model, while the estimates of β differed by about 0.22. However, if we impose the West model of mortality without allowing for a different β, the resulting IMR sometimes differs by as much as 30 points from the estimates derived from the North model. Therefore, the use of equation (1) makes it possible to choose between the various Coale and Demeny models and also allows for deviations from the selected standard.

The estimated trends show that all three countries experienced substantial declines over the period for which data are available. While Kenya has continued to have a substantially lower IMR than the United Republic of Tanzania, the gap between Uganda and Kenya seems to have virtually disappeared between 1950 and 1965. Although it is tempting to assume that the trends in Uganda between 1950 and 1965 have continued, that is an unwarranted assumption. The political, social and economic problems in Uganda during the 1970s make it unlikely that the IMR has kept pace with the declines in the other two countries.

A surprising outcome of our analysis was finding the extent to which the 1948 and 1957-1962 censuses provide useful data. The 1948 censuses provide data for two large age groups only and have long been thought to be generally useless. However, the estimates for many regions and provinces from those censuses appear to be consistent with a pattern of constant, high mortality up to the late 1940s or early 1950s, when a mortality decline began. Although there are cases in which the early census data are clearly useless, on the whole they seem to be more reasonable than was previously assumed.

Note

[1] Much of the difference in level and trend between Kilimanjaro and Arusha regions is explained by the fact that, although Kilimanjaro and Arusha districts are very similar, 71 per cent of the population of Arusha region is in Masai and Mbulu districts, which are very different in almost all respects.

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THE ESTIMATION OF LIFETIME EMIGRATION FROM DATA ON THE RESIDENCE OF CHILDREN: THE CASE OF COLOMBIA

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SUMMARY

The present paper describes a method that uses information on the place of residence of surviving children as reported by their mothers to estimate the level of lifetime emigration. Estimates derived include the total number of emigrants and their distribution by age and sex. The method described is applied to the case of Colombia, where the 1978 National Household Survey gathered the necessary information. Additional information on specific country of residence allows the estimation of the number of Colombian emigrants present in Venezuela.

INTRODUCTION

The study of international migration from a demographic perspective is generally beset by difficulties regarding the availability and reliability of data. The possibility of using indirect methods to measure certain aspects of international migration has arisen only recently, and much remains to be done to validate the results and expand their use. The present paper presents a description of one of the most promising approaches available and discusses in some detail the results obtained when it was applied to data gathered in Colombia. It is hoped that analyses of a similar type may further promote the use of the indirect approach to the estimation of international migration.

DATA NEEDED

Because the essential characteristic of the estimation method presented is that it makes use of special types of information, it is important to devote some attention to its data requirements. Somoza (1977) was the first to propose that data on the residence of surviving children be used to estimate global emigration levels from a given country. Given that during the 1970 round of censuses, several countries gathered information on the number of surviving children of each woman of reproductive age (15-49) with the purpose of estimating infant and child mortality, it seemed sensible to suggest that the questions used to gather that information be extended so as to provide information on the number of surviving children living outside the country (by asking, for example: "How many children have you ever had who live in this country?" and "How many children have you ever had who live in another country?") and that those questions be posed to all women aged 15 and over. In addition, because the estimation method used requires that some adjustment be made for the incidence of orphanhood, a question on maternal orphanhood should also be posed. It must be noted, however, that information on orphanhood is also useful for the estimation of adult mortality levels and, therefore, its investigation in a census or survey inquiry is recommended in its own right. Lastly, given that migration is usually age- and sex-selective, information on the sex of emigrant children is essential. Therefore, the questions proposed above should be posed for daughters and sons separately.

All those requirements imply that, in order to estimate the level of lifetime emigration,[1] at least four items of information on each woman aged 15 and over must be collected, plus at least one more from every member of the population. Although such data requirements are moderate, particularly when the multiple uses of the data gathered are considered (i.e., the data are also useful in the estimation of child mortality by sex and of adult female mortality), their collection would certainly add to the complexity of a census questionnaire and to its processing costs. Therefore, although ideally it would be desirable to obtain the information described above for the whole population, in practice the questions proposed usually will be posed on a sampling basis. The use of a sample is recommended not only because of cost considerations but also because sample surveys are more likely to be carried out by well-trained interviewers and be subject to stricter supervision than censuses, so that they often yield data of better quality. Furthermore, sample surveys provide the best means to investigate more detailed characteristics of the process considered, such as "place of destination" in the case of emigration, an aspect that is likely to be of particular interest for the sending country. The main drawback of the sample

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survey as a tool to measure emigration is that, because emigration levels in most of the sending countries are relatively low, large sample sizes are necessary to measure the phenomenon with an acceptable degree of accuracy. In that respect, however, the methods of estimation proposed here are advantageous because, by aiming at the estimation of lifetime emigration levels rather than at that of current emigration levels (which are generally much smaller), they require somewhat smaller sample sizes.

Zaba (1983) provides some guidance regarding the minimum sample size required to obtain information on approximately 1,000 emigrants from a population whose expectation of life at birth is approximately 60 years and whose average annual rate of growth is about 2 per cent. In such a population, if the average mean number of children ever born to women aged 15 and over is denoted by C, H is the average household size, G is the proportion of the population that is native born, and E is an estimate of the annual emigration rate, then the minimum sample size required to obtain information on 1,000 emigrants is given by

$$\frac{1{,}000}{5.0(C)(H)(G)(E)} \qquad (1)$$

Thus, if C and H were 3.5 and 4, respectively; G were equal to 1.0 and E to 0.002, the minimum sample size required to obtain information on some 1,000 emigrants would be of approximately 7,150 households, a size that is well within the ranges usually covered by sample surveys.

The estimation procedure applied to the case of Colombia

Colombia has carried out two surveys in which the residence of children has been recorded. Unfortunately, the first one (1978 National Household Survey) did not gather residence data separately for each sex. For that reason, the analysis presented here will be carried out only on the data gathered by the second survey, the 1980 National Household Survey, which, being more complete, allows greater flexibility in the estimation process.

Table 1 presents the number of children who were living abroad at the time of the 1980 survey, classified by sex and by five-year age group of mother. Those data will be used to estimate the total level of lifetime emigration and the age distribution of emigrants.

The total number of emigrants from a given country can be classified into several categories according to the survivorship and residence status of their mothers. Following Somoza (1981b), the following categories are defined:

Mother's residence	*Mother's survivorship*	
	Alive	*Dead*
In country	(1)	(2)
Abroad	(3)	(4)

Category (1), constituted of those emigrants whose mothers are alive and present in their country of birth, is obtained directly from the data gathered. Category (2), which includes all emigrants whose mothers were non-emigrant but who have died, can be estimated by using information on the incidence of maternal orphanhood. Category (3), made up of emigrants whose mothers are alive and are also emigrants, may be derived from estimates of categories (1) and (2) by taking into account the fertility of emigrant women; and lastly, category (4), constituted by emigrants with mother emigrant and deceased, can also be estimated by using information on the incidence of maternal orphanhood.

Table 2 shows the proportions of respondents with mother alive classified by five-year age group of respondent. Those data were gathered by the 1980

TABLE 1. BASIC DATA ON RESIDENCE OF CHILDREN: COLOMBIA, 1980

Age group of mother	*Number of women*	*Children surviving (thousands)*		*Emigrant children (thousands)*	
		Males	*Females*	*Males*	*Females*
15-19	1 422.3	107.3	93.3	–	–
20-24	1 125.3	497.3	482.6	–	–
25-29	789.2	808.3	791.9	–	0.5
30-34	598.5	944.2	919.6	0.6	1.2
35-39	553.9	1 162.5	1 125.5	6.0	6.0
40-44	477.9	1 177.4	1 199.0	9.5	12.7
45-49	480.6	1 342.3	1 326.0	29.3	37.6
50-54	371.1	1 027.3	976.1	28.5	19.6
55-59	276.2	761.4	716.9	27.4	33.3
60-64	217.3	489.3	546.6	20.2	23.3
65-69	453.7[a]	378.4	350.8	19.0	13.9
70-74	–	239.8	244.3	10.9	8.2
75+	–	364.3	376.0	17.2	14.0
TOTAL	6 765.8	9 299.8	9 148.6	168.4	170.4

Source: M. Vargas, "Un avance en la estimación de la emigración internacional con base en la información sobre residencia de hijos sobrevivientes", master's thesis in demography, Latin American Demographic Centre, p. 12.

[a] Female population aged 65 and over.

National Household Survey, and are necessary for the estimation of categories (2) and (4), as defined above. Note, however, that the basic data on children's residence presented in table 1 are classified by age of mother, while those on maternal orphanhood are classified by age of respondent. Therefore, in order to use the proportions listed in table 2 to adjust the data in table 1, it is necessary to estimate the age distribution of reported emigrants.

Estimating the age distribution of surviving children

Clearly, because the age of the mother of each reported emigrant is known, the overall age distribution of emigrants may be, at least partly, inferred from the relationship existing between mothers' and childrens' ages in the population under study. In particular, if fertility and mortality were known, the number of children aged x whose mothers were aged y at the time of interview ($C(x,y)$) would be

$$C(x,y) = W(y)f(y-x)l(x) \qquad (2)$$

where $W(y)$ is the number of women aged y at the time of interview, $f(y-x)$ is the fertility rate prevalent among those women x years before and $l(x)$ is the probability of surviving to age x of children born x years before the survey. From equation (2) the derivation of the age distribution of children by age of mother is straightforward, according to the following equation:

$$Z(x,y) = C(x,y)/\int_{y-15}^{y-49} C(u,y)du \qquad (3)$$

In the case of Colombia, a set of consistent fertility and mortality estimates for the 1950-1978 period is available (Zlotnik, 1982), and by extrapolating their values to the beginning of the century and to 1980,[2] it was possible to use equations (2) and (3) to estimate the age distribution of surviving children by age group of mother (see table 3).

When the use of equations (2) and (3) was first proposed (Somoza, 1977), it was suggested that, in countries lacking adequate information on fertility and mortality, the equations could be applied by using constant levels of fertility and mortality throughout. In general, it was expected that the levels used might be inferred from indirect estimates of fertility, child and adult mortality derived from data gathered at the time of the survey or census being considered.

More recently, it has been suggested that, in order to simplify the estimation procedure, models be used to generate a set of age distributions of surviving children by age of mother from which the one most appropriate for the population under study may be selected and applied. Hill (1981a) has proposed two such distributions (see table 4). Selection between them is carried out on the basis of the observed ratio ACS(20-24)/ACS(25-29), where ACS stands for "average children surviving" and the indices in parentheses indicate the age group of women to which the average refers. (Note that averages are calculated on the basis of all women in a given age group, and not only among those who are mothers). Hill's models have been applied to the 1980 Colombian data by Vargas (1982), but the results obtained will not be presented here, since it is judged that models based on essentially stable conditions are not appropriate for a population which has experienced sizeable changes in fertility and mortality during the recent past.

Estimating the age distribution of emigrant children

Once the age distribution of surviving children is available, it is used to infer that of the emigrant children reported by their mothers. Several proposals have been made regarding methods to make this inference. Somoza (1977) first suggested that if independent information on the age structure of the resident population with mother alive and also resident were available, the number of persons in each age group could be subtracted from the corresponding one obtained from all surviving children, and the distribution of the differences thus obtained could be used to distribute the total number of reported emigrant children. That procedure may be adequate in the context of census information that covers the whole population. However, when the basic data have been gathered by a sample survey, sampling errors are likely to have serious distorting effects on the differences being calculated, thus rendering the age distribution of the latter useless. Because of such problems, Somoza (1979) later suggested that the age distribution estimated for all surviving children be used without modification to distribute the reported emigrants. Adoption of such a procedure would mean that the age selectivity of migration operates only through the age linkage between mother and children. However, since in practice that age selectivity is not likely to depend on mother's age, such an assumption is difficult to justify.

TABLE 2. PROPORTION OF RESPONDENTS WITH SURVIVING MOTHER: COLOMBIA, 1980

Respondent's age group	*Proportion with mother alive*
0-4	0.9915
5-9	0.9758
10-14	0.9620
15-19	0.9428
20-24	0.9115
25-29	0.8413
30-34	0.7391
35-39	0.6883
40-44	0.5766
45-49	0.4246
50-54	0.3029
55-59	0.2035
60-64	0.0873
65-69	0.0623
70-74	0.0137

Source: "Un avance en la estimación de la emigración internacional con base en la información sobre residencia de hijos sobrevivientes", master's thesis in demography, Latin American Demographic Centre, p. 12.

TABLE 3. ESTIMATED DISTRIBUTION OF SURVIVING CHILDREN BY OWN AGE, AGE OF MOTHER AND SEX: COLOMBIA, 1980

Children's age group	Age group of mother											
	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75+
							Female						
0-4	1.000	0.852	0.518	0.301	0.180	0.095	0.028	0.002					
5-9		0.148	0.407	0.370	0.270	0.181	0.107	0.034	0.003				
10-14			0.075	0.291	0.324	0.262	0.195	0.126	0.043	0.004			
15-19				0.038	0.205	0.285	0.263	0.215	0.152	0.065	0.009		
20-24					0.020	0.162	0.252	0.248	0.210	0.154	0.068	0.009	
25-29						0.016	0.142	0.232	0.236	0.204	0.154	0.069	0.003
30-34							0.014	0.131	0.221	0.230	0.205	0.158	0.024
35-39								0.013	0.124	0.214	0.229	0.208	0.068
40-44									0.012	0.118	0.210	0.228	0.119
45-49										0.011	0.114	0.206	0.165
50-54											0.011	0.111	0.194
55-59												0.011	0.181
60-64													0.128
65+													0.119
							Male						
0-4	1.000	0.852	0.520	0.304	0.183	0.097	0.028	0.002					
5-9		0.148	0.406	0.371	0.272	0.183	0.109	0.035	0.003				
10-14			0.074	0.288	0.323	0.262	0.196	0.127	0.044	0.004			
15-19				0.037	0.203	0.283	0.263	0.216	0.154	0.066	0.009		
20-24					0.020	0.160	0.250	0.247	0.211	0.156	0.069	0.009	
25-29						0.016	0.140	0.231	0.236	0.206	0.156	0.071	0.003
30-34							0.014	0.129	0.220	0.230	0.207	0.160	0.025
35-39								0.013	0.122	0.212	0.229	0.209	0.071
40-44									0.012	0.116	0.208	0.228	0.122
45-49										0.011	0.112	0.204	0.168
50-54											0.011	0.109	0.196
55-59												0.010	0.181
60-64													0.125
65+													0.109

TABLE 4. MODEL DISTRIBUTIONS OF CHILDREN BY OWN AGE AND AGE OF MOTHER

Children's age group	Age group of mother												
	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75+
	ACS(20-24)/ACS(25-29) > 0.475												
0-4	0.989	0.778	0.461	0.285	0.182	0.104	0.032	0.003					
5-9	0.011	0.218	0.416	0.324	0.228	0.160	0.100	0.032	0.003				
10-14		0.003	0.122	0.301	0.268	0.207	0.157	0.101	0.033	0.003			
15-19			0.002	0.089	0.250	0.242	0.201	0.158	0.102	0.034	0.003		
20-24				0.001	0.071	0.222	0.233	0.200	0.158	0.104	0.034	0.003	
25-29					0.001	0.064	0.214	0.232	0.200	0.189	0.105	0.035	0.002
30-34						0.001	0.062	0.212	0.231	0.200	0.160	0.107	0.014
35-39							0.001	0.061	0.211	0.230	0.201	0.162	0.080
40-44								0.001	0.061	0.209	0.231	0.203	0.138
45-49									0.001	0.060	0.207	0.230	0.186
50-54										0.001	0.058	0.204	0.217
55-59											0.001	0.055	0.208
60+												0.001	0.145
Proportion of children under 15	1.000	1.000	0.998	0.910	0.678	0.471	0.289	0.136	0.036	0.003	—	—	—
Mean age under 15	1.52	3.25	5.62	7.57	8.20	8.72	9.90	11.52	12.85	13.77	—	—	—
	ACS(20-24)/ACS(25-29) ≤ 0.475												
0-4	1.000	0.932	0.591	0.345	0.213	0.120	0.037	0.003					
5-9		0.068	0.380	0.383	0.267	0.184	0.114	0.036	0.003				
10-14			0.029	0.253	0.304	0.238	0.179	0.116	0.038	0.003			
15-19				0.019	0.201	0.269	0.230	0.180	0.117	0.038	0.003		
20-24					0.015	0.176	0.258	0.228	0.180	0.118	0.039	0.004	
25-29						0.013	0.169	0.257	0.226	0.180	0.119	0.040	0.002
30-34							0.013	0.167	0.256	0.228	0.182	0.121	0.027
35-39								0.013	0.166	0.256	0.229	0.183	0.089
40-44									0.012	0.165	0.255	0.230	0.155
45-49										0.012	0.162	0.254	0.209
50-54											0.011	0.158	0.240
55-59												0.010	0.187
60													0.091
Proportion of children under 15	1.000	1.000	1.000	0.981	0.784	0.541	0.330	0.155	0.041	0.003	—	—	—
Mean age under 15	0.80	2.25	4.44	6.92	8.14	8.72	9.91	11.52	12.05	13.77	—	—	—

Source: K. Hill, "Notas sobre la estimación de la distribución por edades de los hijos emigrantes sobrevivientes", *Notas de Población*, vol. IX, No. 27 (1981), pp. 45-66.

To avoid that problem, Hill (1981b) proposed that a model of the incidence of emigration by age be used in conjunction with the age distribution of surviving children to derive the age distribution of emigrant children. The model suggested refers only to the probability of emigrating between ages 15 and 60, and its use without other adjustments would imply that emigration can only take place within that age span. Letting $e(a)$ denote the probability of emigrating at age a among those who will eventually emigrate, the model proposed defines it as

$$e(a) = (20)(45)^{-5}(a-15)(60-a)^3 \text{ for } 15 < a < 60 \quad (4)$$

According to the model, the probability of emigrating by age z among those who will eventually emigrate is

$$E(z) = \int_{15}^{z} e(a)da = \left[\frac{15-4z}{45}\right]\left[\frac{60-z}{45}\right]^4 + 1.0 \quad (5)$$

and therefore, the probability that those in a given age group—say, a to $a+5$—would have emigrated already would be

$$EM(a) = \frac{1}{5}\int_{a}^{a+5} E(z)dz \quad (6)$$

The values of *EM* for age groups ranging from 15-19 to 60+ have been calculated by Hill and are presented in table 5. They can be interpreted as the proportions in each age group who have already emigrated among all those who will eventually emigrate (at or before age 60), assuming that the shape of the emigration schedule remains constant. Hill suggests that those proportions be used to weight the age distribution of surviving children, $Z(a,y)$, so as to obtain the age distribution of emigrants. Thus, denoting by $ZE(a,y)$ the proportion of emigrants aged a to $a+5$ whose mother was aged y at the time of interview, its values are obtained as follows:

$$ZE(a,y) = Z(a,y)EM(a)/\sum_{a} Z(a,y)EM(a) \quad (7)$$

where $Z(a,y)$ is the estimated proportion of surviving children aged a to $a+5$ among those whose mothers were aged y at the time of the survey (obtained by cumulating $Z(x,y)$, as defined by equation (3)).[3]

Although that procedure is certainly more satisfactory than the one proposed by Somoza, several questions regarding its acceptability need to be addressed. First, how well does the model reflect actual emigration schedules? The answer to that question can only be partial, since lack of adequate data on emigration precludes valid comparisons in most cases. However, Vargas (1982) has compared Hill's model with the age patterns of immigrants to Argentina[4] and with the model migration patterns that Morales (1974) derived from entry statistics gathered from different countries. Such comparisons show that Hill's model is adequate,

TABLE 5. PROBABILITIES OF EMIGRATING BY A GIVEN AGE GROUP AMONG THOSE WHO WILL EMIGRATE BY AGE 60, ACCORDING TO THE MODEL EMIGRATION SCHEDULE PROPOSED BY HILL

Age group	*Proportion of emigrants*
15-19	0.0347
20-24	0.1986
25-29	0.4250
30-34	0.6415
35-39	0.8099
40-44	0.9190
45-49	0.9753
50-54	0.9960
55-59	0.9999
60+	1.0000

although it would seem to overestimate slightly the incidence of emigration between ages 25 and 40, and to underestimate that over age 45. Yet, its simplicity of use far outweighs the possible distorting effects that may arise because of those discrepancies with respect to observed data, which in any case do not necessarily reflect the Colombian experience.

The second question that arises regards the validity of assuming that the age pattern of emigration has remained constant. Strictly speaking, the validity of that assumption should be tested specifically for Colombian emigrants before using the emigration model for the purpose of estimation. However, the data available on Colombian immigrants in the main destination countries (Venezuela and the United States of America) do not permit adequate testing of that hypothesis, mainly because data regarding the entry of Colombians into Venezuela are almost totally lacking and those available fail to reflect illegal or undocumented migration. Yet, even in the absence of adequate data, it is possible to cast doubts on the assumption made, if only because during the present century important changes have taken place in economic development, transportation, communication and immigration laws and policies which are unlikely to have yielded a stable migration phenomenon. However, in the absence of better information, the simplest hypothesis will be adopted.

A third question concerns the applicability of the emigration model to stocks of emigrants. That is, even if the model reflects adequately the age pattern of persons leaving in a given year or during a given period, it might not reflect equally well the age structure of those who remain abroad, since the latter will depend not only on the age pattern of emigration but also on that of the migrants who return. Evidently, more information regarding the migration process is needed to assess the relationship between those two age structures, and given the lack of information on return migration there is no possibility of making such an assessment.

Lastly, although Hill's emigration model may be adequate for those migrating after age 15, those migrating prior to that age cannot be ignored. The estimation of migrants under 15 is rather complex because several groups of migrants need to be con-

sidered separately. The largest, constituted by children who migrate with their mothers, will be estimated as part of categories (3) and (4). Yet it is also necessary to consider the few cases of persons under 15 who migrate without their mothers (they will be said to migrate "independently") and who are left out of the migration model proposed by Hill.

By assuming that independent migration of persons under 15 occurs at a fixed rate that does not change with age, for each age of mother one can estimate the expected proportion of emigrant children under 15 by multiplying the fixed rate of emigration, em_{15-}, by the average age of children under 15 for a given age of mother. Thus, if $MA_{15-}(y)$ is the mean age of children under 15 whose mothers were aged y at the time of interview, the proportion emigrating prior to age 15, $pe_{15-}(y)$ is

$$pe_{15-}(y) = em_{15-}MA_{15-}(y) \qquad (8)$$

Therefore, if $Z(0\text{-}14,y)$ is used to denote the proportion of children under 15 among those reported by women aged y, and $C(y)$ is the total number of children surviving reported by those women, an estimate of the number who emigrated prior to age 15 and who were still under 15 at the time of the survey is given by

$$CE_{15-}(0\text{-}14,y) = pe_{15-}(y)Z(0\text{-}14,y)C(y) \qquad (9)$$

and an estimate of those who emigrated before being 15 years of age but who are 15 or older at the time of the survey is

$$CE_{15-}(15+,y) = (15.0)em_{15-}(1.0-Z\ 0\text{-}14,y))C(y) \qquad (10)$$

The estimated $CE_{15-}(0\text{-}14,y)$ and $CE_{15-}(15+,y)$ can then be distributed by age according to the estimated distribution of all surviving children (i.e., $Z(a,y)$ for 0_{-a} 15 in the case of $CE_{15-}(0\text{-}14,y)$ and $Z(a,y)$ for 15_{-a} in the case of $CE_{15-}(15+,y)$), and the remainder emigrant children,

$$CE_{15+}(y) = CE(y) - CE_{15-}(0-14,y) - - CE_{15-}(15+,y) \qquad (11)$$

can be distributed according to the proportions estimated in (7).

Thus, if em_{15-} is known or can be estimated, the distribution of emigrant children can be derived by making allowance for independent migration below age 15. Hill suggests that em_{15-} be estimated on the basis of the data referring to the surviving children of women under 30 whose ages are generally less than 15. Thus, if $C(30-)$ and $CE(30-)$ are used to denote the total number of surviving children and the total number of emigrant children, respectively, reported by women under 30,

$$em_{15-} = CE(30-)/(C(30-)MA_{15-}(30-)) \qquad (12)$$

where $MA_{15-}(30-)$ is the mean age of the surviving children considered.

In the case of Colombia, as table 1 indicates, there were no emigrant sons reported by women under 30, and only one emigrant daughter was reported by women in that age group (the number listed in table 1 has been expanded). That outcome implies not only that the incidence of independent emigration below age 15 is very low but also that it is probably not reflected accurately by a survey of the size of the 1980 NHS. Consequently, it is probably not worthwhile to apply the complex estimation procedure outlined above, since the low value of em_{15-} will introduce very minor differences. However, for the sake of illustration, the procedure was applied, letting em_{15-} equal 0.00009 for both sexes (i.e., the estimated value for females was imputed to males). Table 6 shows the results obtained.

The estimation of orphaned emigrants

Once the age distribution of emigrants whose mothers are alive and living in their country of origin is obtained, it is a simple matter to adjust the figures for the incidence of maternal orphanhood, provided data on that topic are available. In the case of Colombia, the proportions of respondents with mother alive are presented in table 2. Then, denoting by $EM_1(x)$ the preliminary estimate of the number of emigrants aged x obtained above and by $NO(x)$ the proportion of per-

TABLE 6. ESTIMATION OF THE NUMBER OF CHILDREN EMIGRATING BEFORE AGE 15 AND OF THEIR AGE DISTRIBUTION: COLOMBIA, 1980

Age group of mother	Female emigrant children			Male emigrant children			Age distribution of emigrants		
	Under 15		Number over 15	Under 15		Number over 15	Age group	Female	Males
	Mean age	Number		Mean age	Number				
15-19	1.19	0.01	0.00	1.19	0.01	0.00	0-4	0.8	0.8
20-24	2.75	0.12	0.00	2.75	0.13	0.00	5-9	1.0	1.0
25-29	5.07	0.37	0.00	5.05	0.37	0.00	10-14	1.2	1.2
30-34	7.39	0.60	0.02	7.37	0.61	0.02	15-19	1.2	1.2
35-39	8.52	0.68	0.20	8.49	0.70	0.20	20-24	1.0	1.1
40-44	9.22	0.55	0.47	9.19	0.54	0.46	25-29	0.8	0.8
45-49	10.32	0.41	0.84	10.29	0.42	0.84	30-34	0.5	0.5
50-54	11.74	0.17	0.88	11.72	0.18	0.92	35-39	0.3	0.3
55-59	12.97	0.04	0.81	12.96	0.04	0.86	40-44	0.1	0.1
60-64	13.85	0.00	0.69	13.84	0.00	0.62	45-49	0.0	0.0

sons aged x with mother alive, an adjusted estimate of the number of emigrants aged x is obtained as

$$EM_2(x) = EM_1(x)/NO(x) \qquad (13)$$

The results for the case of Colombia are shown in table 7.

The estimation of those emigrating with their mothers

The number of emigrants in category (3)—that is, those whose mothers are alive and are themselves emigrants—remains to be estimated. The estimation of that category is perhaps the one affected by the greatest degree of arbitrariness, since there is generally no evidence allowing the determination of the place of birth of the children of emigrant women and, strictly speaking, only those who were born in the country of origin should be considered.

At this point, it seems worthwhile to make a small digression regarding the definition of "migrant" and of "foreign population". While, from a purely demographic perspective, a migrant is a person who has changed place of residence, usually for some minimal length of time, from the legal perspective a person may be considered "immigrant" or "foreign" even if his/her place of residence has never changed. That is the case, for example, in several Western European countries where the fact of being born in the country does not entitle one to citizenship. Therefore, although from the purely demographic perspective children of emigrants who were born abroad should not be considered emigrants, from the economic, social, political, administrative and even statistical perspectives, it is often important to know the total number of offspring that emigrants have had, since that group constitutes a subpopulation which may require special treatment and which, statistically, is often not distinguished from true emigrants in the demographic sense (as in the case of countries that publish data on the "foreign" population rather than on those who are foreign-born). Hence, although the estimation in the case of Colombia will proceed by assuming that only true emigrants, in the demographic sense, are to be counted, in certain cases it may be more advantageous to count even the foreign-born children of emigrants, a strategy that would simplify considerably the estimation procedure used and that may yield estimates of greater immediate relevance.

TABLE 7. ESTIMATION OF THE TOTAL NUMBER OF EMIGRANTS AND THEIR AGE DISTRIBUTION: COLOMBIA, 1980

Age group	Proportion non-orphan	Females					Males				
		Reported emigrants		Emigrant children		Total emigrants	Reported emigrants		Emigrant children		Total emigrants
		Observed	Adjusted[a]	Estimated	Adjusted[a]		Observed	Adjusted[a]	Observed	Adjusted[a]	
						(thousands)					
0-4	0.992	0.8	0.8	14.4	14.5	15.3	0.8	0.8	14.9	15.1	15.9
5-9	0.976	1.0	1.0	27.5	28.2	29.2	1.0	1.0	28.4	29.1	30.1
10-14	0.962	1.2	1.2	29.2	30.4	31.6	1.2	1.2	29.8	30.9	32.2
15-19	0.943	11.1	11.8	24.8	26.3	38.1	9.5	10.1	25.1	26.6	36.7
20-24	0.912	33.3	36.5	18.2	20.0	56.5	29.1	31.9	18.3	20.0	51.9
25-29	0.841	40.0	47.5	14.2	16.9	64.4	37.9	45.0	14.2	16.9	61.9
30-34	0.739	30.5	41.3	12.1	16.4	57.7	31.4	42.4	12.1	16.3	58.8
35-39	0.688	22.2	32.3	10.5	15.2	47.5	22.2	32.3	10.3	15.0	47.3
40-44	0.577	12.8	22.2	8.1	14.0	36.2	14.4	25.0	7.9	13.7	38.7
45-49	0.425	7.0	16.4	5.1	11.9	28.3	8.9	20.9	4.9	11.6	32.4
50-54	0.303	4.1	13.6	2.2	7.4	21.0	9.2	17.2	2.1	7.1	24.2
55-59	0.204	2.7	13.5	0.5	2.6	16.1	3.4	16.6	0.5	2.5	19.0
60-64	0.140[b]	1.9	13.3	0.0	0.3	13.6	2.2	16.0	0.0	0.3	16.3
65-69	0.090[b]	1.1	12.2	0.0	0.0	12.2	1.3	14.1	0.0	0.0	14.1
70+	0.060[b]	0.6	10.5	0.0	0.0	10.5	0.7	11.4	0.0	0.0	11.4
TOTAL		170.3	274.0	166.8	204.1	478.1	169.2	286.0	168.5	205.1	491.1
						(percentage)					
0-4		0.5	0.3	8.6	7.1	3.2	0.5	0.3	8.9	7.3	3.2
5-9		0.6	0.4	16.5	13.8	6.1	0.6	0.4	16.8	14.2	6.1
10-14		0.7	0.5	17.5	14.9	6.6	0.7	0.4	17.7	15.1	6.6
15-19		6.5	4.3	14.9	12.9	8.0	5.6	3.5	14.9	13.0	7.5
20-24		19.6	13.3	10.9	9.8	11.8	17.2	11.2	10.8	9.8	10.6
25-29		23.5	17.4	8.5	8.3	13.5	22.4	15.8	8.4	8.2	12.6
30-34		17.9	15.1	7.3	8.1	12.1	18.5	14.8	7.2	8.0	12.0
35-39		13.1	11.8	6.3	7.4	9.9	13.1	11.3	6.1	7.3	9.6
40-44		7.5	8.1	4.8	6.9	7.6	8.5	8 8	4.7	6.7	7.9
45-49		4.1	6.0	3.0	5.9	5.9	5.2	7.3	2.9	5.6	6.6
50-54		2.4	5.0	1.3	3.6	4.4	3.1	6.0	1.3	3.5	4.9
55-59		1.6	4.9	0.3	1.3	3.4	2.0	5.8	0.3	1.2	3.9
60-64		1.1	4.9	0.0	0.2	2.9	1.3	5.6	0.0	0.2	3.3
65-69		0.6	4.4	0.0	0.0	2.5	0.7	4.9	0.0	0.0	2.9
70+		0.4	3.8	0.0	0.0	2.2	0.4	4.0	0.0	0.0	2.3
TOTAL		100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

[a] Adjusted for the incidence of maternal orphanhood.
[b] Adjusted, since use of the reported proportions led to clearly inconsistent results.

In estimating the native-born children of emigrants who are themselves emigrants, at least two separate questions must be addressed. First, what is the fertility of emigrant women? Ideally, that fertility should be estimated from information gathered in the immigration country. When such information is lacking, however, educated guesses are the most likely alternative. Unfortunately, there are few guidelines for making guesses. Even the direction of the differential between the fertility levels of the non-migrant population and the emigrant population is debatable. For example, while Vargas (1982) has shown that immigrant women in Argentina tend to have lower fertility than their counterparts in the sending countries, the distribution of emigrant women by region of origin in the case of Colombia suggests that they may experience fertility levels above the national average (because among emigrant women, a proportion higher than would be expected on the basis of regional importance originates in regions with above-average fertility). In spite of those conjectures, in estimating emigration from Colombia it was decided to adopt the assumption that the fertility of emigrant women is equal to 80 per cent of that corresponding to the total non-migrant female population.

Secondly, a scheme must be devised to distribute the offspring of emigrant women according to place of birth and, thus, according to emigration status. Vargas (1982) was the first to suggest that a distribution of emigrant women according to the period of emigration be used to effect that distribution. The scheme is based on the assumption that emigration proceeds in an orderly fashion: women emigrate taking all their surviving children with them, and those children remain with them in the country of immigration. Following the same basic idea, but using Hill's emigration schedule to estimate the proportion of emigrant women who had already emigrated by each age, it is possible to estimate the proportion of surviving, native-born children who migrated with them. Thus, among $WE(y)$ emigrant women aged y at the time of the survey, a proportion $E(y-x)/E(y)$ had already emigrated by age $y-x$. Therefore, only the surviving children of the remainder,

$$CEM(x,y) = (1.0 - E(y-x)/E(y))\times \\ \times WE(y)f(y-x)1(x) \qquad (14)$$

are considered to be emigrants. The results obtained for the case of Colombia are shown in table 7.

The estimation of orphaned children of emigrants

To conclude the estimation procedure, allowance must be made for the incidence of orphanhood among the offspring of emigrant mothers. Once more, the proportions of respondents with mother alive shown in table 2 are used to adjust the estimates of emigrant children in category (3), in a manner similar to that embodied by equation (13). The results obtained are shown in table 7.

COMMENTS ON THE RESULTS OBTAINED

As table 7 shows, the different adjustments described above contribute to an inflation in the reported number of emigrants to a considerable extent, so that the final estimates of lifetime emigration levels are more than three times the reported ones. Among the adjustments made, the one for orphanhood contributes to an inflation in the reported number by about 65 per cent, while that for the number of emigrant children with mother abroad (whether alive or dead) contributes to an inflation in the basic reports by about 120 per cent. Expressed in a slightly different manner, among the 969,000 estimated total number of emigrants, only 340,000 were reported directly by their resident mothers, 220,000 would have been reported had their mothers been alive and nearly 409,000 are "second-generation emigrants"—that is, persons whose mothers are or were also emigrants. As already stated, at least the last figure is subject to a great degree of uncertainty and can only be interpreted with caution, especially since little is known about the propensity of whole family groups to migrate in the specific context of Colombian emigration.

The results presented in table 7 also show not only that the adjustment for the fertility of emigrant women may be controversial but that the one for the incidence of orphanhood may also lead to suspect results, at least for certain age groups. In fact, had the reported proportions with mother alive among respondents aged 60 and over been used to adjust the preliminary age distribution of reported emigrants, clearly unacceptable results would have been obtained. Such outcome may be due, at least in part, to the fact that the mortality estimates used to generate the age distributions of surviving children may be inadequate. However, errors in the reporting of mother's survival or in age reporting that would bias the reported proportions with mother alive cannot be entirely ruled out. Moreover, the existence of errors at older ages stemming from the adjustment for the incidence of maternal orphanhood have strong, undesirable effects on the adjustment for the offspring of emigrants, since, given the characteristics of the emigration schedule being used and of the scheme used to differentiate native-born offspring from those born abroad, older emigrant women are more likely to contribute greater proportions of their offspring to the missing category (3). That is, a positive bias in the number of second-generation emigrants may be due to the compounded effects of over-correction for the incidence of maternal orphanhood.

Unfortunately, lack of data regarding the number or characteristics of Colombian emigrants in the main receiving country, Venezuela, precludes the possibility of carrying out a deeper assessment of the results obtained. One can note, however, that the age distribution of the total number of emigrants under 60 seems acceptable, although the proportion over 60 is probably too high; and that even the estimate of total lifetime emigration shown in table 7 (which may overstate

its true level) falls far short of claims made about the level of illegal immigration of Colombians to Venezuela (which often put the figure at a minimum of 2 million). In addition, data on legal migration to Venezuela (obtained from the 1970 census results) and the results of the 1980 drive to regularize illegal immigrants in the country suggest that by that year there were at least 443,000 Colombians present in Venezuela. Given that, among the emigrants reported in the 1980 National Household Survey of Colombia, about 68 per cent were in Venezuela, the estimates of table 7 imply that at most 659,000 Colombians were in the neighbouring country by mid-1980. Hence, the number remaining there illegally would be at most 216,000, and that figure probably overstates the true situation.

Thus, although the emigration estimates derived from indirect data fall far short of the standards of reliability usually associated with other types of indirect estimates, they can nevertheless be very useful tools in assessing the plausibility of claims that, in spite of having no scientific or objective foundation, are all too often the only "information" available on international migration.

Notes

[1] The term "lifetime emigration level" is used to denote the total number of persons born in a given country who, at the time of interview, live outside of that country, irrespective of the time at which they left. Thus, it is a measure of the cumulated emigration experience of a country, similar in meaning to the total foreign-born population enumerated in the country of immigration at a given point in time.

[2] In terms of mortality, it was assumed that up to 1910 the population had been subject to mortality level 5 of the West family of models proposed by Coale and Demeny (1966), and that by 1930 mortality had reached level 7 of the same family. From 1930 to 1950, where the estimates proposed by Zlotnik (1982) start, the logit system was used for interpolation purposes (linear interpolation on α and β was used). In terms of fertility, its level and pattern were assumed to remain constant up to 1964, at which time the declining estimates proposed by Zlotnik were adopted. For the years 1979 and 1980, it was assumed that the 1978 estimates prevailed.

[3] Equation (7) can also be written in continuous form, using $E(a)$ instead of $EM(a)$ and $Z(x,y)$ instead of $Z(a,y)$. In the application shown, a discrete version of equation (7) based on single-year age groups was used.

[4] Tabulations from a 2 per cent sample of the 1970 census of Argentina include one on the number of immigrants classified by age and period of arrival. Such data permit the estimation of age patterns of immigration for a given period. An average of the patterns estimated for three different periods was compared with Hill's model.

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WHAT DO WE KNOW ABOUT CAUSES OF SEX DIFFERENCES IN MORTALITY? A REVIEW OF THE LITERATURE*

*Ingrid Waldron***

SUMMARY

The evidence reviewed in the present paper indicates that sex differences in mortality are influenced by a wide variety of environmental and genetic factors. The relative importance of particular factors varies greatly, depending on socioeconomic and cultural conditions.

In contemporary industrial societies the single most important cause of higher mortality for males has been greater cigarette smoking by males. Other sex differences in behaviour have also contributed to males' higher mortality, including men's heavier alcohol consumption and more frequent employment in hazardous occupations. The behaviour that contributes to males' higher mortality has been socially more accepted or expected of males. It appears that cultural influences on sex differences in behaviour have been one important environmental factor contributing to sex differences in mortality in industrial societies. Genetic factors that may play a role include a possible protective effect of endogenous female sex hormones that may reduce women's risk of ischaemic heart disease.

In many non-industrial societies those factors have played a less important role and other factors have had a major influence on sex differences in mortality. For example, under conditions of inadequate health care and nutrition, women's inherent vulnerability to maternal mortality can make a substantial contribution to excess female mortality at reproductive ages. In addition, discrimination against females can result in less adequate nutrition and health care for females, with consequent increased vulnerability to infectious diseases and higher mortality for girls and young women than for boys and young men.

Historical trends and cross-cultural variation in sex differences in mortality reflect the differential effects on male and female mortality of variation in technology, economic conditions and cultural influences on behaviour. The widespread introduction of cigarettes, together with social pressures against cigarette smoking by women, was a major cause of the increasing male mortality disadvantage in industrial countries in the twentieth century. In addition, improvements in health care and the general standard of living have contributed to decreases in mortality for several causes of death with a female excess, and that was another cause of the growing female mortality advantage. Sex roles have had variable effects on sex differences in mortality owing to variations in social norms concerning appropriate behaviour for males and females, variation in exposure to occupational hazards, and varying effects of male and female economic roles on the extent of discrimination against females.

Additional topics discussed include the relative importance of sex differences in incidence of disease vs. sex differences in prognosis or survival rates in determining sex differences in mortality. Data for industrial countries indicate that for most types of cancer and ischaemic heart disease, sex differences in prognosis are relatively unimportant and sex differences in incidence of disease are the primary determinants of sex differences in mortality. The findings that for most types of cancer and ischaemic heart disease there is little or no sex difference in prognosis and little or no sex difference in seeking medical care suggest that differential use of curative medicine is not an important cause of sex differences in mortality in those cases.

*The present paper is part of a study on levels, trends and causes of sex differentials in mortality currently being carried out by the Population Division of the Department of International Economic and Social Affairs, United Nations Secretariat. The financial support of the World Health Organization in the preparation of the paper is gratefully acknowledged.

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INTRODUCTION

The present paper reviews current evidence concerning the causes of sex differences in mortality. One useful approach to the topic has been to identify major causes of death that contribute to sex differences in total mortality and then to identify factors that contribute to sex differences for those causes of death. Results of that approach are summarized in the first section of the present review. The second section summarizes evidence concerning the causes of historical and cross-cultural variation in sex differences in mortality. The third section discusses several general issues and hypotheses concerning the causes of sex differences in mortality, together with relevant evidence.

The evidence to be presented shows that a wide variety of factors influence sex differences in mortality. Genetic factors of relevance include effects of inherent sex differences in reproductive anatomy and physiology, effects of sex hormones, and effects of X-chromosome linked genes. Environmental factors of importance include cultural influences on sex differences in risky behaviour, such as cigarette smoking, and cultural influences that determine sex differences in access to resources, such as nutrition and health care. Additional environmental factors of importance include technological and economic changes that have decreased or increased mortality risks to which either males or females are particularly vulnerable. Those varied factors have interacting effects on sex differences in mortality and the importance of any given factor varies considerably in different situations.

In a review of the present type, it is obviously necessary to be selective rather than inclusive. Several topics will be treated only very briefly, including descriptions of the patterns of sex differences in mortality. (Illustrative data are given in the figure and in the table.) Much additional information is available in previous reviews and major data analyses (Lopez and Ruzicka, 1983; Nathanson, 1984; Preston, 1976; Retherford, 1975; Verbrugge, 1982; Waldron, 1976, 1982a, 1983a, 1983b; Wingard, 1984).[1]

CAUSES OF SEX DIFFERENCES FOR MAJOR CAUSES OF DEATH

The present section reviews factors that influence sex differences in mortality for infectious and parasitic diseases, accidents and other violence, ischaemic heart disease, and malignant neoplasms. The contribution of each of those categories to sex differences in total mortality varies depending on age and historical and

TABLE. AGE-STANDARDIZED DEATH RATES AND SEX DIFFERENCES IN AGE-STANDARDIZED DEATH RATES BY SEVERAL MAJOR CAUSES OF DEATH, SELECTED DEVELOPED AND DEVELOPING COUNTRIES
(per 100,000 population)

Country, year and sex	*Total mortality*	*Infectious and parasitic diseases*	*Maternal mortality*	*Accidents, poisonings and violence*	*Ischaemic heart disease*	*Malignant neoplasms*
United States, 1930						
Male death rates.....	1 401	180		155	. . .	107[a]
Female death rates.....	1 180	145	23	60	. . .	134[a]
Sex differences.....	221	35	—23	95	. . .	—27[a]
United States, 1978						
Male death rates.....	816	8	—	98	260	165
Female death rates.....	472	6	—	35	129	109
Sex differences.....	345	3	—	63	131	56
Japan, 1978						
Male death rates.....	627	15		62	41	144
Female death rates.....	400	7	1	25	24	86
Sex differences.....	227	8	—1	37	18	58
Chile, 1978						
Male death rates.....	1 009	61		135	97	141
Female death rates.....	645	38	4	33	55	114
Sex differences.....	364	23	—4	102	42	28
Guatemala, 1976						
Male death rates.....	1 543	371		285	11	45
Female death rates.....	1 409	349	14	234	9	57
Sex differences.....	134	22	—14	51	2	—13

Sources: Adapted from Ingrid Waldron, "Sex differences in human mortality: the role of genetic factors", *Social Science and Medicine*, vol. 17, No. 6 (1983); based on data from World Health Organization, *World Health Statistics Annual: Vital Statistics and Causes of Death*, for 1977, 1980 and 1981 (Geneva); Samuel H. Preston, Nathan Keyfitz and Robert Schoen, *Causes of Death: Life Tables for National Populations* (Seminar Press, New York, 1972). The standard population used for age-standardization of rates is from M. Segi, K. Aoki and M. Kurihara, *Cancer Mortality and Morbidity Statistics*, GANN Monograph on Cancer Research No. 26 (Tokyo, Japan Scientific Press, 1981), p. 122, table A, last column.

NOTE: In a few cases rounding of the death rates has resulted in slight discrepancies between the sex differences between the rounded male and female rates.

[a] Includes benign neoplasms and those of unspecified nature.

cultural conditions, but each is of major importance in some cases.

Infectious and parasitic diseases

Males tend to have higher death rates than females for infectious and parasitic diseases, particularly for ages above 30 (see the figure; see also Holzer and Mijakowska, 1983; Preston, 1976; United Nations 1982). For the age range 1-30, reversals of that sex difference, with higher infectious and parasitic disease death rates for females, have been fairly common. Possible causes of those general patterns of sex differences in infectious disease mortality are reviewed below. However, it should be mentioned that the patterns of sex differences do vary somewhat for different types of infectious and parasitic disease and the impact of the factors to be discussed also varies for different diseases.

Males' generally greater susceptibility to infectious disease mortality may be due in part to genetic factors (Waldron, 1983a). Females have higher levels than males of one of the major classes of immunoglobulins, IgM. It appears that the X chromosome carries one or more gene(s) that influence IgM production, and the pair of X chromosomes in females' cells results in higher IgM levels than the single X chromosome in males' cells. There are, however, some problems with that hypothesis. No explanation has been offered for the observation that females have higher IgM levels only between about 5 and 65 years of age. Also, the age pattern for sex differences in IgM levels differs from the age pattern of sex differences in infectious disease mortality, and that raises some doubts concerning the importance of the contribution of sex differences in IgM levels. The X chromosome carries a variety of additional genes which influence the functioning of the immune system. Thus it may be that X-linked deficiencies in a variety of immune characteristics contribute to males' greater susceptibility to infectious disease mortality.

The effects of sex hormones on the immune system may also influence sex differences in infectious disease mortality (Waldron, 1983a). The effects of sex hormones on immune responses vary depending on specific experimental conditions, and it is at present unclear to what extent or under what conditions the effects of sex hormones may favour greater resistance for females or for males.

An environmental factor that appears to influence sex differences in infectious disease mortality in some cases is differential access to nutrition and health care. For example, in parts of South Asia and Algeria and according to some historical data from Europe, girls have had higher death rates for infectious diseases than boys, and that appears to have been due in large part to less adequate nutrition and health care for girls than for boys (Chen and others, 1981; D'Souza and Chen, 1980; Johansson, 1980; Miller, 1981; Tabutin, 1978; Vallin, 1983). Preston's analysis of international variation in sex differences in mortality provides evidence that discrimination against females, in combination with low nutritional levels, reduces or reverses the male's excess for infectious disease mortality (Preston, 1976).

Another environmental factor that may influence sex differences in mortality due to infectious diseases is differential exposure. In some cases, women may have greater exposure to infection—for example, due to their role as care-takers for sick family members, while in other cases men may have greater exposure—for example, due to greater contact with parasites in irrigation water.

In summary, sex differences in infectious disease mortality are influenced by cultural factors, such as differential access of males and females to food and medical care, and probably also by genetic factors, such as X-linked differences in levels of one major type of immunoglobulin.

Fatal accidents and other violent deaths

Death rates for accidents and other violent causes have been higher for males than for females in almost all available historical and international data, although the magnitude of the male excess has varied considerably (see the figure and the table; Lopez and Ruzicka, 1983; Preston, 1976). Analyses of data for the United States of America and other Western countries have shown that a variety of sex differences in behaviour contributes to men's higher mortality due to accidents and other violence.

In the United States, men drive more than women and men have more fatal accidents per mile driven, and both factors appear to contribute to men's higher death rates for motor vehicle accidents (Veevers, 1982; Waldron, 1976, 1982a). Men's higher rates of fatal accidents per mile driven suggest that they drive less safely, and that has been confirmed by observational studies showing that men tend to drive faster and less cautiously than women and men more often violate traffic regulations.

For accidents other than motor vehicle accidents, a major cause of men's higher rates (at least in the United States) is accidents on the job (Waldron, 1976, 1980, 1982a). Men have more work accidents than women because more men are employed and because the jobs men hold are more physically hazardous on the average. United States data also indicate that males have much higher rates than females for accidental drownings and fatal gun accidents.

Men's greater use of guns appears to contribute to their higher suicide rates (Waldron, 1976). In suicidal behaviour, men more often use guns and women more often use drugs. Since guns are more lethal than drugs, that contributes to men's higher suicide rates and women's higher rates of non-fatal suicide attempts. However, sex differences in suicide methods are not the only factor that contributes to sex differences in suicide rates, since men have higher suicide rates for each method and, also, men have higher suicide rates even in a population where males and females used

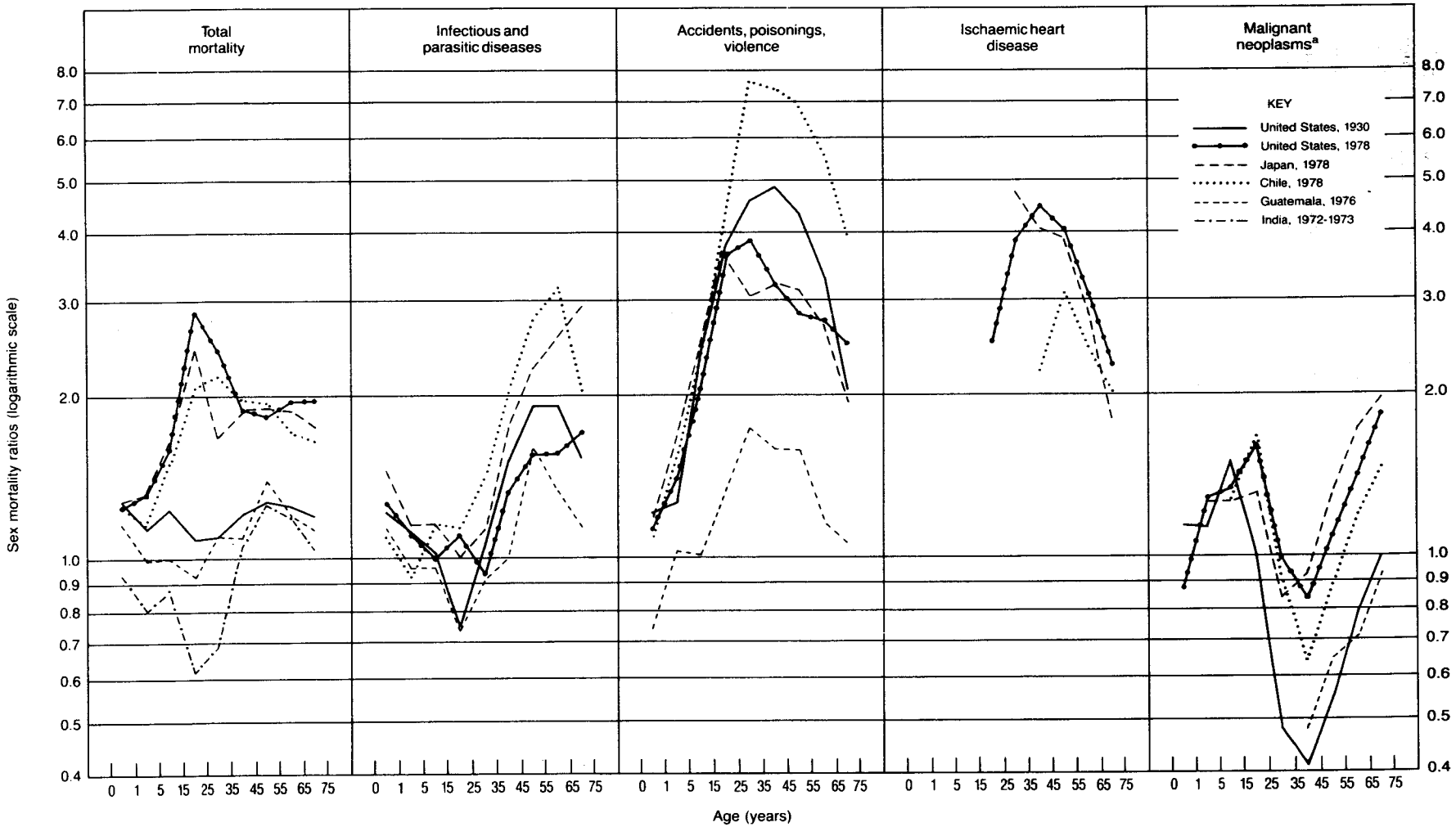

Figure. Sex mortality ratios by age for total mortality and several major causes of death, selected developed and developing countries

Sources: Graphs adapted from Ingrid Waldron, "Sex differences in human mortality: the role of genetic factors", *Social Science and Medicine*, vol. 17, No. 6 (1983); based on data from World Health Organization, *World Health Statistics Annual: Vital Statistics and Causes of Death*, for 1977, 1980 and 1981 (Geneva); National Academy of Sciences, National Research Council, Committee on Population and Demography, Panel on India, *Vital Rates in India, 1961-1981*, Report No. 24 (Washington, D.C., National Academy Press, 1984); Samuel H. Preston, Nathan Keyfitz and Robert Schoen, *Causes of Death: Life Tables for National Populations* (Seminar Press, New York, 1972).

NOTE: Sex mortality ratios are the ratios of male-to-female death rates. Age groups with less than 100 deaths for a particular cause in a particular country have been excluded. For India, reliable death rates by cause of death were not available, so data are given only for total mortality.

[a] Data for the United States for 1930 include benign neoplasms and those of unspecified nature.

similar suicide methods (Alexander and others, 1982; Waldron, 1976). One factor that may be important is that women may more often use a suicide attempt as a desperate, last-ditch plea for help, while men, who feel more pressure to be strong, may be less willing to plead for help and more likely to carry a suicidal act through to a fatal conclusion (Waldron, 1976).

Heavier alcohol consumption among men is another cause of their higher rates of fatal accidents and other violent deaths (Ferrence, 1980; Hetzel, 1983; Waldron, 1976, 1982). In Western countries, male drivers are considerably more likely than female drivers to have high blood-alcohol levels, and drunken drivers are responsible for roughly one quarter to one half of fatal auto accidents, including those in which the driver him or herself is killed. (Men's heavier consumption of alcohol is also the major cause of men's higher rates of cirrhosis of the liver, at least in Western countries.)

Much of the behaviour that contributes to men's higher rates of fatal accidents and other violent deaths has been more socially accepted or expected of males than of females. Use of guns, drinking alcohol, taking risks physically and working at sometimes hazardous jobs have all been more expected of males than of females in Western societies as well as in many non-Western societies (Waldron, 1976, 1982a). Even in infancy sex differences in relevant types of behavior are expected and encouraged. For example, experiments have shown that babies presented as boys were encouraged to be more physically active than the same babies presented as girls (Honig, 1983). Differential socialization of boys and girls is widespread cross-culturally, and those cultural influences appear to be an important reason for the sex differences in behaviour that contribute to males' higher rates of fatal accidents and other violent deaths (Waldron, 1976, 1983a).

Another hypothesis is that male sex hormones may predispose males to higher levels of physical activity and physical aggressiveness (Hines, 1982; Schiavi and others, 1984; Waldron, 1983a). Although some of the available evidence supports that hypothesis, inconsistent results and methodological problems prevent any definite conclusions. A related hypothesis has been that the Y chromosome contributes to increased aggression. That hypothesis has been tested in studies that have compared males who have the normal XY genotype with males who have either XYY or XXY genotypes. Findings from those studies do not support the hypothesis that an extra Y chromosome specifically contributes to greater aggressiveness, but rather suggest that defects resulting from an extra chromosome may contribute to deviance.

The observation that mortality due to accidents and other violent causes is higher for males in almost all available data, together with the observation that related sex differences in behaviour are very widespread cross-culturally, has suggested that there may be a genetic basis for those sex differences in behaviour. However, it is possible that the contribution of genetic factors is a rather indirect one—namely, that inherent sex differences in reproductive function have influenced the cultural evolution of sex roles, including sex differences in risk-taking behaviour (Waldron, 1983a). Specifically, it appears that because only women can bear and nurse children, women have been assigned the primary responsibility for the care of babies and young children in all or almost all cultures. Probably as a consequence, dangerous tasks and other tasks that are incompatible with child care have been assigned primarily to men. Related to those widespread differences in adult sex roles, there are differences in the socialization of boys and girls which are widespread cross-culturally and which prepare boys to participate in the more hazardous activities typically associated with the male role. Thus, inherent sex differences in reproductive function appear to have influenced the cultural evolution of sex roles, and the cultural environment in turn fosters behaviours in males which contribute to their higher rates of fatal accidents and other violent deaths.

Ischaemic heart disease

Death rates for ischaemic heart disease (previously called coronary heart disease) have consistently been higher for men than for women in almost all available international and historical mortality data (see the figure and table; Johansson and others, 1983; Lopez 1983). Because ischaemic heart disease is such a major cause of death in Western countries, the substantial male excess for ischaemic heart disease is, on the average, responsible for about one third of the sex difference in adult mortality. However, the magnitude of the sex difference for ischaemic heart disease has varied considerably in different regions, historical periods and ethnic groups.

One major reason why death rates for ischaemic heart disease are higher for men than for women is that more men than women smoke cigarettes and, among smokers, men generally have more hazardous smoking habits than women (United States Department of Health and Human Services, 1980; Waldron, 1983a). One major effect of cigarette smoking is to increase atherosclerosis of the coronary arteries. Thus, it appears that men's higher rates of cigarette smoking contribute to the greater atherosclerosis of their coronary arteries, which is in turn a major cause of men's higher ischaemic heart disease mortality (Johansson and others, 1983; United States Department of Health and Human Services, 1980; Waldron, 1983b). The importance of the sex differences in cigarette-smoking habits is indicated by the observation that sex differences in ischaemic heart disease mortality are considerably lower among non-smokers than among the general population (Waldron, 1976, 1982a). Data for the United States suggest that, depending on the age range, approximately one quarter to three fifths of the sex difference in ischaemic heart disease mortality may be related to cigarette smoking.

Sex differences in cigarette smoking habits are very

widespread cross-culturally. Surveys have shown that more men than women smoke cigarettes in Western and Eastern Europe, the United States, Australia, New Zealand, Jerusalem, Latin America, the Far East, South Asia, and the Pacific Islands (Anon., 1982; Benjamin, 1982; Finau and others, 1982; Joly, 1975; LeMeitour-Kaplan, 1977; Miller and Ashcroft, 1971; Pool, 1983; United States Department of Health and Human Services, 1980; Waldron, 1983a). A greater prevalence of cigarette smoking among men has been observed in both urban and rural areas. A few exceptions to the pattern have been observed, for teenagers in the United States and Western Europe in recent years and among the Maoris in New Zealand. Nevertheless, it is clear that a greater prevalence of cigarette smoking among men has been widespread cross-culturally and historically. In addition, among smokers, men smoke more cigarettes per day than women on the average and more men than women inhale deeply. Those sex differences in smoking habits are also widespread cross-culturally, having been observed in the United States, Western Europe, Latin America and Tonga. Thus, a considerable body of evidence indicates that in many different parts of the world men are more likely than women to smoke cigarettes and that among cigarette smokers, men have smoking habits that are more hazardous to health than the smoking habits of women.

One important reason for the sex differences in cigarette-smoking habits has been greater social pressure against cigarette smoking by women (Waldron, 1983a). Social pressures against cigarette smoking by women have been reported for areas as diverse as the United States during the early twentieth century and Sri Lanka and several Pacific islands in recent decades. Social pressures against cigarette smoking by women have varied in intensity, and that appears to be a major reason for the considerable variation in the magnitude of sex differences in cigarette smoking in different regions and different historical periods. Another factor that has been hypothesized to contribute to the lower prevalence of smoking among women is that females may more often feel sick as a result of smoking their first cigarette and that may deter them from smoking.

Although sex differences in cigarette smoking are a major cause of men's higher rates of ischaemic heart disease mortality, other factors must also contribute. Even among non-smokers men have substantially higher rates of ischaemic heart disease mortality than women (Doll and others, 1980; Shurtleff, 1974; Waldron, 1976). An additional behavioural factor that may contribute to sex differences in risk of ischaemic heart disease is the Type-A or coronary-prone behaviour pattern (Waldron, 1976, 1978, 1982a; Wingard, 1984), a hard-driving style of life characterized by aggressiveness, competitiveness, hostility, impatience, a chronic sense of time urgency, and a strong drive to achieve. For general population samples in the United States it has been found that Type-A behaviour is more common among men than among women. Since Type A behaviour is associated with a substantially increased risk of developing and dying of ischaemic heart disease, the greater prevalence of Type-A behaviour among men may contribute to men's higher rates of ischaemic heart disease mortality.[2]

Another factor that may contribute to sex differences in ischaemic heart disease risk may be a protective effect of the female sex hormones secreted by women's ovaries. Such an effect is suggested by the finding that the risk of ischaemic heart disease may be increased for women who have had both ovaries removed and for women who have had an early natural menopause (Johansson and others, 1983; Waldron, 1983a). That conclusion must be considered tentative because some studies have found no difference in risk of ischaemic heart disease between post-menopausal women and pre-menopausal women of the same age, and one study even suggests a lower risk for post-menopausal women (Rosenberg and others, 1983).

Several observations suggest the need for caution in interpreting any association between early menopause and increased risk of ischaemic heart disease (Johansson and others, 1983; Waldron, 1983a). First, women who smoke cigarettes are more likely to have an early natural menopause, and it appears that greater cigarette smoking may account for part—but not all—of the increased risk of myocardial infarction for women with early natural menopause. Secondly, many of the women who had their ovaries removed also had their uterus removed, and removal of the uterus alone appears to be associated with an increased risk of ischaemic heart disease (although the increase in risk appears to be less than when the ovaries are removed). Since removal of the uterus does not appear to interfere with hormone secretion by the ovaries, those findings suggest that some women have characteristics that predispose them to a higher risk for both hysterectomy and ischaemic heart disease or that effects of hysterectomy not directly related to ovarian function contribute to increased risk of ischaemic heart disease. One hypothesis for which there is suggestive evidence is that anxiety, depression and neuroticism increase the probability that a woman will have a hysterectomy (Gath and others, 1982; Waldron, 1976) and also increase the risk of death due to ischaemic heart disease (Jenkins, 1982). Another hypothesis is that menstruating women are protected by regular loss of blood which prevents iron accumulation and in both post-menopausal women and men, iron accumulation may damage the heart (Sullivan, 1983). However, it should be noted that the type of heart damage due to iron accumulation is very different from either the heart damage due to ischaemic heart disease or the atherosclerosis of the coronary arteries that is a major underlying cause of ischaemic heart disease (Waldron, 1981).

In summary, current evidence suggests that endogenous female sex hormones may reduce women's risk of ischaemic heart disease, but inconsistencies and complications in the evidence prevent any firm conclusion. In contrast to the apparent protective effects of

endogenous female sex hormones in women, current evidence indicates that endogenous estrogens in men may actually be associated with an increased risk of ischaemic heart disease. A number of studies have found that estrogen levels are elevated in men who have had a myocardial infarction and probably also in men with other manifestations of ischaemic heart disease (Phillips and others, 1983; Waldron, 1983a).

Studies of the effects of exogenous female sex hormones provide additional evidence that, under some conditions, female sex hormones may increase the risk of ischaemic heart disease (Johansson and others, 1983; Waldron, 1983a). Studies of estrogen therapy in men have found that high doses of estrogen generally result in increased risk of ischaemic heart disease. Use of oral contraceptives also increases the risk of myocardial infarction. Studies of postmenopausal estrogen therapy generally suggest little or no effect on ischaemic heart disease risk, perhaps reflecting the relatively low doses of estrogen typically used in that type of therapy.

In summary, endogenous female sex hormones may reduce the risk of ischaemic heart disease for women, but endogenous estrogens in men and exogenous female sex hormones for either sex do not appear to reduce risk and may even increase risk. Those observations suggest that there may be something about the specific types, quantities or patterns of hormones secreted by women's ovaries that contributes to the protective effect of endogenous female sex hormones for women. To understand why the effects of female sex hormones may be protective or harmful, depending on specific conditions, it should be kept in mind that those hormones have multiple counteracting effects on the cardiovascular system. For example, estrogens may increase the risk of ischaemic heart disease by enhancing blood coagulation processes, but they may also decrease risk by reducing LDL cholesterol and increasing HDL cholesterol (the latter being a protective factor) (Johansson and others, 1983; Waldron, 1983a). The balance between the harmful and beneficial effects of female sex hormones apparently varies, depending on specific conditions.

Less information is available concerning the effects of male sex hormones on the risk of ischaemic heart disease (Phillips and others, 1983; Waldron, 1983a). No consistent relationship has been found between levels of male sex hormones and various manifestations of ischaemic heart disease. It has been hypothesized that testosterone increases males' risk of cardiovascular disease by stimulating increased susceptibility to platelet aggregation, thrombus formation and/or vascular contractility (Ramey and Ramwell, 1984). However, this hypothesis is based on data for rats and other non-primate mammals and it is doubtful whether these findings should be extrapolated to humans since there are substantial differences between species. For example, susceptibility to platelet aggregation has been found to be greater for males than for females for rats, but not for humans. Other available data also provide no persuasive evidence that male sex hormones contribute to men's higher risk of ischaemic heart disease.

Several other hypotheses have proposed additional physiological or anatomical bases for the sex differences in ischaemic heart disease mortality, but none are supported by persuasive evidence at present.

In conclusion, sex differences in cigarette-smoking habits are a major cause of sex differences in ischaemic heart disease mortality. The observed sex differences in ischaemic heart disease mortality among non-smokers indicate that additional factors must pay a role. Additional factors of importance may include sex differences in the Type-A behaviour pattern and possible protective effects of women's endogenous sex hormones.

Malignant neoplasms

Mortality due to malignant neoplasms or cancer is higher for males than for females over most of the age span in many countries (see the figure and table; United Nations, 1982; Waldron, 1983a). However, reversals with higher female than male cancer mortality have been fairly common, particularly for the age span of approximately 25-50 years old, in contemporary data for Latin American countries and in historical data for the more developed countries. It should be noted that malignant neoplasms constitute a variety of diseases with disparate causes, and thus both the pattern and causes of sex differences vary for different types of malignant neoplasms.

A major cause of men's higher cancer mortality has been the sex differences in cigarette smoking habits described in the previous section. Two studies in the United States have found that for adults who had never smoked regularly there was little or no sex difference in cancer mortality (Enstrom and Godley, 1980; Hammond and Seidman, 1980). In national mortality statistics for the United States, if cancers known to be related to cigarette smoking are excluded, sex differences in cancer mortality are very small (Waldron, 1982a). Lung cancer alone is responsible for approximately three quarters of men's excess cancer mortality in developed countries, and sex differences in cigarette-smoking habits are the major cause of men's higher lung cancer mortality (Lopez, 1983; Waldron, 1976). (Sex differences in cigarette-smoking habits are also a major cause of men's higher mortality for other diseases of the respiratory system, such as emphysema and bronchitis (Preston, 1970; Waldron, 1976).)

Occupational exposures also contribute to men's higher cancer mortality, including men's higher lung-cancer mortality (Waldron, 1980, 1982a; Walker and others 1983). For example, in the United States many more men than women have been exposed to asbestos occupationally, and it has been estimated that those occupational exposures to asbestos may contribute to one tenth or more of men's lung-cancer deaths. Men are exposed more than women to a variety of additional occupational carcinogens that have been linked

to lung cancer, bladder cancer and a number of other types of cancer.

The importance of cigarette smoking and occupational exposures as causes of men's higher cancer rates has been confirmed by an analysis of the causes of cancer for males and females in one region of England and in Greater Bombay, India (Higginson and Muir, 1979). The analysis also indicates that sex differences in personal habits, such as alcohol consumption or betel chewing, contribute to men's higher cancer rates. The contribution of men's heavier alcohol consumption to their higher cancer rates has been confirmed in data for other developed countries as well (Garros and Bouvier, 1978; Hetzel, 1983).

In addition to those behavioural factors, sex differences in reproductive anatomy and the effects of sex hormones influence the sex differences in cancer mortality (Waldron, 1976, 1983a). Malignant neoplasms of the genital organs are obviously sex-specific, and death rates for malignant neoplasms of those organs have generally been higher for women than for men. In addition, women have a much higher risk of breast cancer than men, and that also reflects inherent sex differences in anatomy and physiology. The female excess for breast and genital cancers is primarily responsible for the female excess for total cancer mortality generally observed in the 25-50-year-old range (data from World Health Organization, 1980, 1981). Additional effects of hormones and other inherent sex differences may contribute to sex differences in cancer mortality, but they appear to have less impact than the factors already discussed.

In summary, sex differences in cancer mortality are influenced by multiple behavioural, anatomical and physiological differences between the sexes. Sex differences in cigarette smoking, as well as in occupational exposures and alcohol consumption, are important factors contributing to higher cancer mortality for men. Sex differences in reproductive anatomy and effects of sex hormones contribute to higher cancer mortality for women, particularly for breast cancer. It appears that in many cases the balance of those multiple effects results in higher cancer mortality for males, but in other cases the balance of effects results in higher cancer mortality for females.

Causes of Variation in Sex Differences in Mortality

Although males have higher mortality than females in all contemporary data for more developed countries, females have had higher mortality than males in many less developed countries, particularly in childhood and young adulthood (figure; United Nations, 1982, 1983; Tabutin, 1978). In addition, there has been great variation historically and cross-culturally in the magnitude of sex differences in mortality. Evidence concerning the causes of variation in sex differences in mortality is reviewed in the present section. The patterns of variation and the major causes of variation differ by age, so the evidence is presented by major age groups.

Foetal and infant mortality

During the first month after conception, foetal mortality appears to be very high (Miller and others, 1980). However, virtually nothing is known about the sex ratio of those very early foetal deaths because the conceptus is rarely recovered for sex determination. For the second through the sixth months of foetal life, there have been numerous studies of sex differences in mortality, but results have been contradictory and inconclusive, owing to substantial methodological problems (Waldron, 1983a). One recent study that appears to have used the best available methodology indicates that foetal mortality is higher for males than for females at that stage of foetal development (Hassold and others, 1983).

Studies of late foetal mortality—i.e., foetal mortality from the seventh month on—have encountered fewer methodological problems and have yielded quite consistent results (Waldron, 1983a). For the period 1930-1960, data for Europe and the United States show a higher risk of late foetal mortality for males than for females. The male excess for late foetal mortality has been decreasing, and recent data for several European countries, the United States and New Zealand show an approximately equal risk of late foetal mortality for males and females. The trends in sex differences in late foetal mortality appear to reflect interactions between genetic factors and changing environmental conditions. It appears that, because of inherent sex differences, males have a higher risk of late foetal mortality due to difficult labour, birth injuries, and diseases and accidents of the mother, while females have a higher risk of late foetal mortality due to congenital malformations of the central nervous system. As obstetric practice and maternal health have improved, the importance of late foetal mortality due to difficult labour, birth injuries and diseases and accidents of the mother has declined, and correspondingly the male excess for late foetal mortality has declined and disappeared in several advanced industrial societies.

Infant mortality—that is, mortality during the first year after birth—is higher for males than for females in most available data for different countries and different historical periods (see the figure; United Nations, 1983; Preston, 1976; Vallin, 1983; Waldron, 1982a). The male excess for infant mortality is particularly consistent for the first half year after birth, which suggests that inherent sex differences in vulnerability play a role during that period. The nature of the postulated sex differences in vulnerability is poorly understood at present. One contributing factor may be that the lungs of males appear to mature more slowly during foetal development due to the effects of testosterone (Nielsen and others, 1982; Waldron, 1983a). The greater immaturity of male newborns' lungs may contribute to their greater vulnerability to respiratory distress, an important cause of mortality for young infants.

In some cases infant mortality has been higher for females than for males, particularly during the second

half of the first year (D'Souza and Chen, 1980; Vallin, 1983; Waldron, 1982a). Higher infant mortality for females appears to be due in large part to discrimination against females, resulting in less adequate care for female babies than for male babies. That type of discrimination against females has been observed also for children and young adults and is discussed in the section below.

Mortality of children and young adults

Sex differences in mortality have been very variable for children and young adults (referring here to an age of approximately 1-40) (see the figure; D'Souza and Chen, 1980; Enterline, 1961; Kennedy, 1973; Lopez and Ruzicka, 1983; Tabutin, 1978). On the one hand, in many non-industrial societies and some early industrial societies, females have had higher mortality than males in some or all of that age range. On the other hand, in most industrial societies males have had higher mortality than females throughout the age range, and in many cases the ratio of male to female death rates reaches a maximum at ages 15-24. Historical evidence for quite a number of countries indicates that there has been a shift from excess female mortality to excess male mortality for children and young adults. In addition, in most developed countries there has been a substantial increase in excess male mortality for teenagers and young adults during the twentieth century. Limited data indicate a reverse trend in an earlier period in Europe. Specifically, data for several European countries indicate that in the nineteenth century or early twentieth century, excess female mortality emerged in that age range following a period of excess male mortality (Johansson, 1984; Kennedy, 1973; Tabutin, 1978). Evidence concerning the causes of those variable patterns of sex differences in mortality for children and young adults is summarized below.

One factor that contributes to excess female mortality at young adult ages is maternal mortality, which can make a substantial contribution to the mortality of young women under conditions of inadequate health care and nutrition. Indeed, in parts of South Asia and Algeria it appears that maternal mortality has accounted for about 30-100 per cent of female excess mortality at ages 15-44 (Nadarajah, 1983; Vallin, 1983; Waldron, 1982a).

In addition, in some circumstances girls and young women have had higher rates of infectious disease mortality than boys and young men, and that has been a major cause of excess female mortality at those ages (Preston, 1976; Tabutin, 1978; Waldron, 1983a). At reproductive ages, one cause of women's vulnerability to infectious diseases may have been physiological strains due to repeated childbearing and lactation. However, for girls at pre-reproductive ages, other factors must have been responsible for the excess female mortality for infectious diseases. As discussed in an earlier section, a major factor contributing to higher female rates of infectious diseases appears to have been less adequate nutrition and health care for females. Other factors that may have contributed to excess female mortality in pre-industrial Europe include exposure to infectious diseases while caring for sick family members, exposure to accidental burns and scalds while working in the kitchen, and the debilitating effects of the very heavy workload of farm women (Bideau, 1981; Johansson, 1980; Kennedy, 1973).

Several lines of evidence suggest that female excess mortality may be greater in those circumstances in which females make less contribution economically. For example, in regions of India where women's work makes a smaller contribution to agricultural production and family income, there appears to be more discrimination against girls in terms of nutrition and health care and a greater mortality disadvantage for girls (Miller, 1981; Rosenzweig and Schultz, 1982).

Variation in the economic roles of men and women may also explain some of the trends in sex differences in mortality in Western countries (Hammel and others, 1983; Johansson, 1980, 1984; Kennedy, 1973). For example, in several European countries in the nineteenth century there was a shift from excess male mortality to excess female mortality in the childhood to young adult age ranges. Those trends may have been due to increased discrimination against females, perhaps reflecting a decrease in the value of farm women's production relative to farm men's production as farms became more integrated into the market economy. In the nineteenth century in several European countries and in Massachusetts, the female mortality disadvantage was observed primarily in rural areas and was smaller or reversed in urban areas; that pattern may have been related to greater economic opportunities for girls and women in urban areas.

It should be noted that the type of effect of sex differences in economic roles on sex differences in mortality may be restricted to situations with relatively limited material resources. Preston's (1976) analysis of international data for 1960-1964 for a sample that included predominantly economically developed countries showed that neither sex differences in participation in the non-agricultural labour force nor sex differences in education were related to sex differences in total mortality. Rather, it was the combination of discrimination against women, together with low nutritional levels, that was associated with a relative mortality disadvantage for females. It appears that as economic development proceeds and more economic resources become available, sex differences in status may have less impact on sex differences in mortality. In addition, it appears that in many cases, as economic development proceeds, the status of women may improve. Both those factors may have contributed to the historical shifts from a female excess to a male excess for several major causes of death (see the figure; Nadarajah, 1983; Pool, 1983; Preston, 1976).

Another important factor that has contributed to the trends towards an increasing male mortality disadvantage has been the decline in importance of several

causes of death which had higher mortality for females and the increasing relative importance of several causes of death with a male excess (see the table; Enterline, 1961; Nadarajah, 1983). Declining mortality for causes of death with excess female mortality, such as maternal mortality and tuberculosis, was due to improvements in health care, nutrition and general standard of living. For example, historical evidence for the United States indicates that in the 1930s and 1940s maternal mortality decreased substantially due to improvements in medical care, including the introduction of new medical technology such as blood banks and sulfa drugs and more careful regulation of medical practice to prevent the spread of infection by physicians and to reduce unnecessary and risky obstetric surgery (Antler and Fox, 1976). In contemporary industrial countries maternal mortality is low and has very little impact on sex differences in mortality (see the table; Preston 1976).

As deaths due to maternal mortality and infectious diseases have decreased in importance, deaths due to motor vehicle accidents and other violence have become increasingly important components of total mortality at those childhood and young adult ages. Because mortality due to accidents and other violence is generally much higher for males than for females, the increasing importance of motor vehicle accidents and other violence has contributed to the increasing male disadvantage for total mortality (Enterline, 1961; Tabutin, 1978; Vallin, 1983; Waldron, 1982a). Indeed, for teenagers and younger adults in contemporary industrial countries, fatal motor vehicle accidents and other violence are the principal cause of excess male mortality (Lopez, 1983).

In summary, sex differences in mortality for children and young adults have been highly variable, ranging from a female excess in many pre-industrial or early industrial countries to a large male excess in many contemporary industrial countries. The major causes of excess female mortality have been high levels of maternal mortality and higher infectious disease death rates for females than for males. Those reflect the combined effect of inadequate health care and nutrition and discrimination against females in access to limited material resources. Excess female mortality due to discrimination against females has been observed primarily in cultures where women's work makes a smaller contribution economically and where material resources are inadequate. During the twentieth century, improved medical care and general standard of living have contributed to decreases in maternal mortality and several other causes of death with a female excess. As a result the relative importance of accidents and other violence has increased, and that is the major cause of the substantial male excess mortality observed for teenagers and younger adults in contemporary industrial societies.

Mortality of older adults

For older adults (older than about age 40), mortality has been higher for males than for females in most cases (see the figure; United Nations, 1982, 1983). In a few cases higher mortality for females has been observed, and it has been suggested that excess mortality for females at those ages may be related to long-term deleterious effects of high fertility, particularly where diet, health care and other conditions for women are poor (Morgan, 1983; Preston, 1976).

The most striking trend for older adults has been the increase in the excess of male over female mortality during the twentieth century in the more developed countries (see the figure; Enterline, 1961; Lopez, 1983; Preston, 1970; Retherford, 1975). That increase has been due in large part to a growing male excess for cardiovascular diseases and cancer, reflecting primarily increases in men's rates of ischaemic heart disease and lung cancer. One major cause of those trends has been an increase in cigarette smoking, a habit which was adopted considerably earlier by men than by women. Cigarette smoking results in substantially greater health risks than earlier forms of tobacco use, such as pipe or cigar smoking or chewing tobacco. It appears that one reason for the increase in cigarette smoking was innovation in the techniques of manufacture and marketing of cigarettes (United States Department of Health and Human Services, 1980). Reasons why cigarette smoking was initially more socially acceptable for men than for women remain speculative.

In more recent decades, as social acceptance of women's smoking has increased, sex differences in cigarette smoking habits have decreased in many Western countries. Correspondingly, sex differentials in lung cancer mortality have begun to decrease in some countries, following the smoking trends with a latency of roughly three decades (Lopez, 1983).

Despite the major importance of cigarette smoking as a cause of trends in sex differences in mortality for older adults, it is clear that other factors also play a role. For example, the increasing mortality disadvantage for older men in France has been linked to increasingly heavy alcohol consumption by men, as well as cigarette smoking (Garros and Bouvier, 1978; Vallin, 1983).

The importance of factors other than cigarette smoking is also illustrated by the discrepancies between recent trends in sex differences in cigarette smoking and sex differences in ischaemic heart disease mortality in several Western countries. Sex differences in cigarette smoking have decreased, but in England and Wales sex differences in ischaemic heart disease mortality have increased and in Canada, the United States and Australia there has been relatively little change in sex differences in ischaemic heart disease mortality as death rates for ischaemic heart disease have decreased in parallel for men and women in recent years (Hetzel, 1983; Kleinman and others, 1979; Nicholls and others, 1981; Waldron, 1982a). The causes of those recent trends in ischaemic heart disease mortality are in dispute.

In some cases the recent trends in ischaemic heart disease and lung cancer mortality have contributed to

the ending or reversal of earlier long-term trends towards an increasing male disadvantage for total mortality. Specifically, for 45-64-year-olds in the United States and Australia, the long-term trend towards increasing sex mortality ratios has recently been replaced by a decrease or stabilization of sex mortality ratios (Lopez, 1983; Waldron, 1982a; Wingard, 1984).

Total mortality

Analyses of mortality over the whole age span confirm many of the conclusions for the specific age ranges discussed above, as well as indicating several additional conclusions concerning causes of variation in sex differences in mortality. As mentioned above, Preston (1976) has shown that the male mortality disadvantage tends to be smaller in countries where there is evidence of discrimination against women in combination with low levels of nutrition. Preston's analysis also showed that the male mortality disadvantage tends to be larger in more urban and less agricultural countries. Comparisons of sex differentials in mortality in different regions within a country have also shown a greater disadvantage for males or a smaller disadvantage for females in more urban areas (Johansson, 1980, 1984; Kennedy, 1973; POLIWA, 1977), although some data show reversals of that pattern (Holzer and Mijakowska, 1983). Where males have had a relative disadvantage in urban regions, that may have been due to higher levels of cigarette smoking and less exercise, particularly for males, and less discrimination against females, perhaps related to relatively greater economic opportunities for females in urban areas.

The possibility that the mortality disadvantage for males may be smaller under conditions of social stability and greater under conditions of social disruption is suggested by an analysis of regional variation in sex differences in mortality in contemporary Belgium (POLIWA, 1977). Specifically, the excess mortality for males was smaller in regions which had a larger proportion of people who have lived in the same area since birth and in regions with high "integration"—i.e., low rates of divorce, illegitimacy, suicide and cirrhosis and low proportions of foreigners in the population. Those findings parallel findings from individual-level data from several more developed countries indicating that the mortality disadvantage for males is smaller among the married than among the unmarried (Retherford, 1975; Stroebe and Stroebe, 1983; Wingard, 1984). It appears that men may be more vulnerable than women to the disruption of social relationships, including marriage. That may reflect in part women's greater ability to maintain various alternative sources of social support, such as a confidant other than one's spouse. Also, there may be sex differences in responses to stress (including social disruption), and those differences may expose men to greater mortality risks (Waldron, 1983b). For example, in Western countries it appears that in response to stress women are more likely to use psychotropic drugs, whereas men are more likely to drink alcohol and, as discussed above, men's heavy alcohol consumption exposes them to substantial mortality risks.

In summary, historical trends and cross-cultural variation in sex differences in mortality have been influenced by a wide variety of factors. Varying levels of medical care and standard of living have influenced the relative importance of particular causes of death which have contributed to either female or male excess mortality. In some circumstances discrimination against females has resulted in less adequate nutrition and health care and consequently higher mortality for females. The widespread introduction of cigarettes in the early twentieth century, together with changing social mores concerning women's smoking, have strongly influenced sex differences for several major causes of death. It appears that in the twentieth century the dominant trends in the causal factors discussed have favoured females and those trends appear to have been primarily responsible for the increasing mortality advantage for females observed in many countries. However, a number of the trends discussed have favoured males, and they have resulted in a decrease in the male mortality disadvantage or the emergence of a male mortality advantage for various age groups in certain time periods.

General issues and hypotheses

Sex differences in illness incidence and prognosis

In analysing the causes of sex differences in mortality, one important question is the extent to which those differences are due to sex differences in incidence of disease (i.e., the probability of developing a disease) or sex differences in prognosis (i.e., the probability of surviving once a person has developed a disease). Considerable data are available concerning that question for various types of cancer and ischaemic heart disease in the more developed countries.

For most types of cancer, sex differences in incidence are the primary determinant of sex differences in mortality (Waldron, 1983b). In general, sex differences in prognosis are smaller and less consistent than sex differences in incidence, and sex differences in prognosis appear to make little or no contribution to sex differences in mortality for most types of cancer. Factors such as sex differences in cigarette smoking, alcohol consumption and occupational exposures make substantial contributions to sex differences in cancer incidence, but those factors appear to make much smaller contributions to sex differences in prognosis. One major reason is that prognosis is assessed only for individuals who have already developed a disease and sex differences in behavioural risk factors tend to be much smaller for those who have developed a disease than for the general population.

It has been hypothesized that women may be inclined to notice symptoms earlier and to seek medical care earlier than men, and a shorter lagtime in obtaining medical care might contribute to a better

prognosis for women. However, available evidence indicates that men and women do not differ in the lag-time between first noticing symptoms and seeking medical care for various types of cancer (Marshall and others, 1982; Waldron, 1983b). Furthermore, in most cases there do not appear to be sex differences in the stage of cancer at first diagnosis. The apparent absence of sex differences in seeking medical care for cancer is congruent with the absence of consistent or substantial sex differences in prognosis for most types of cancer.

Similar results have been found for those forms of ischaemic heart disease than can be diagnosed on the basis of relatively objective criteria (Johansson and others, 1984; Martin and others, 1983; Waldron, 1983b). The incidence of sudden coronary death and myocardial infarction is substantially higher for men than for women, and men have more atherosclerosis of the coronary arteries. In contrast, sex differences in prognosis are generally small and inconsistent in different studies of patients with myocardial infarction or substantial atherosclerosis of the coronary arteries. The lagtime between onset of symptoms of an acute episode of ischaemic heart disease and obtaining medical care is at least as long for women as for men. Also, the proportion of myocardial infarctions that have not been recognized and have not been treated medically is as high for women as for men. Thus, it appears that women are not more likely than men to seek prompt medical care, and that is compatible with the absence of any consistent female advantage in prognosis for those forms of ischaemic heart disease.

A contrasting picture emerges for angina pectoris, a form of ischaemic heart disease which is diagnosed primarily on the basis of patient reports of a characteristic type of pain (Waldron, 1983b). For angina pectoris without prior myocardial infarction, sex differences in incidence are smaller and do not always show a male excess. Prognosis appears to be somewhat worse for men than for women. One possible interpretation of that pattern is that relatively mild cases of angina are more likely to be diagnosed for women than for men because women are more sensitive to symptoms or more willing to report the symptoms that provide the basis for the diagnosis of angina. Some of the available evidence supports that hypothesis, although other evidence suggests that there may be sex differences in the underlying patho-physiological processes that may contribute to the observed patterns of sex differences.

In conclusion, for most types of cancer and ischaemic heart disease, sex differences in incidence are the primary determinants of sex differences in mortality. Sex differences in prognosis are smaller and make little or no contribution to the sex differences in mortality. A major reason why sex differences in prognosis are generally smaller than sex differences in incidence is that sex differences in risk factors are generally smaller for those who have already developed a disease (for whom prognosis is assessed) than for the general population (for whom incidence is assessed). Also, current research has found little or no sex difference in seeking prompt medical care for those diseases, so it appears that there is little or no contribution of differential use of medical care to sex differences in prognosis. Those conclusions are of importance because they apply to causes of death that make very major contributions to sex differences in mortality in the more developed countries. Nevertheless, it is important to recognize the limitations of those generalizations, since exceptions are observed even for certain types of cancer and ischaemic heart disease, as well as for other causes of death not discussed here–for example, motor vehicle accidents and suicide (Fife and others, 1984; Waldron, 1976) and probably for infectious diseases in some less developed countries (Chen and others, 1981; D'Souza and Chen, 1980).

Risk-taking and preventive behaviour

It has been proposed that in Western countries males are more likely than females to take risks that may injure their health, and females are more likely than males to engage in preventive behaviours designed to preserve and improve their health (Nathanson, 1977; Waldron, 1976). The evidence summarized below indicates that in Western countries males do engage in more risk-taking behaviours of certain types, particularly behaviours that involve physical daring or illegal behaviour. However, for other types of risky behaviour and for preventive behaviour sex differences are variable.

In the United States males engage in more physically risky recreational activities and that is one factor contributing to their higher accident rates (Waldron, 1983b). Men also tend to drive faster and less cautiously than women and men more often drive while under the influence of alcohol, and such risky behaviour contributes to men's higher rates of fatal motor vehicle accidents. Evidence of greater risk-taking by men is also provided by the pattern of sex differences in the use of psychoactive substances (Nathanson, 1977; Waldron, 1976, 1983b). More men than women use illegal psychoactive substances and drink alcohol heavily, both of which carry substantial health risks. In contrast, women are more likely than men to use medically prescribed psychotropic drugs.

Cigarette smoking is another type of hazardous substance use which has been more common among men than among women in many different parts of the world, and among smokers men have more risky smoking habits than women. However, recent trends in cigarette smoking suggest that sex differences in the propensity to take risks are not the primary determinant of sex differences in cigarette smoking. Sex differences in cigarette smoking have decreased during the recent period in Western countries at the same time as the dangers of cigarette smoking have become more evident and more widely publicized (LeMeitour-Kaplan, 1977; United States Department of Health and Human Services, 1980; Waldron, 1983b). It appears that the major determinant of sex differences

in cigarette smoking has been the changing patterns of social acceptability of cigarette smoking for women, and those patterns appear to have varied independently of knowledge of the health risks involved.

With respect to preventive behaviour, sex differences are quite variable depending on the type of behaviour and the culture considered. In the United States and Canada women make more use of some types of preventive medical care (Kohn and White, 1976; Nathanson, 1977; Verbrugge, 1982). However, in some European countries more men than women had been immunized within the previous twelve months or had had a physical examination for reasons other than illness, injury or pregnancy. As discussed in the section above, women are no more likely than men to seek early medical care for symptoms of various types of cancer or acute ischaemic heart disease. However, in Western countries women do make more frequent visits to physicians than men, and that may result in more effective control of some life-threatening conditions such as hypertension (Apostolides and others, 1980; Verbrugge, 1982; Waldron, 1982a, 1983b).

For other types of preventive behaviour, sex differences are also inconsistent (Waldron, 1983b). For example, no consistent sex differences have been found in the use of seat belts. Data for the United States indicate that women more often than men take vitamins, but men get as much (or more) vigorous exercise as women. Thus, the available evidence does not support a general pattern of a greater propensity for males to take risks and for females to engage in preventive behaviour. Rather there are varied sex differences in risk-taking and preventive behaviour, and those reflect multiple cultural influences on sex differences in specific types of behaviour. In addition, there may be an inherent tendency for males to be more active physically which may contribute to the observed patterns.

Sex roles

There appear to be a number of important links between traditional sex roles and sex differences in mortality. Most of them have been discussed above, but it is useful to summarize briefly the most important points.

One important link between traditional sex roles and sex differences in mortality is the effects of employment on health. More men than women are employed and men's jobs tend to be more hazardous than women's jobs (data for the United States in Waldron, 1976, 1980, 1982a). Men's greater exposure to occupational carcinogens contributes to their higher rates of lung cancer and bladder cancer. Accidents on the job are an important cause of men's higher accident rates. It appears likely that sex differences in exposure to accidents on the job were particularly important in the early twentieth century when jobs were probably more hazardous and accidents and other violence were responsible for about half of the sex difference in life expectancy in the United States (Retherford, 1975).

Recently there has been considerable interest in the question of whether the increased employment of women in Western countries will lead to increased mortality for women. Current evidence for the United States suggests that, on the average, women's jobs are less hazardous than men's jobs and consequently employment has had less harmful effects on women's health than on men's health. Longitudinal data for a United States sample indicate that, on the average, employment had neither harmful nor beneficial effects on the general health of married, middle-aged women (Waldron and others, 1982).

Those observations indicate that the effects of employment have varied, depending on the types of jobs and the extent of hazardous exposure. It appears that the health effects of traditional female roles have also varied, although only limited evidence is available on that question.

In addition to the direct effects of sex differences in economic roles on sex differences in health, several more indirect effects are suggested by evidence presented in previous sections. For example, it appears that, if women's work makes a relatively smaller contribution economically, that may be associated with discrimination against females and, if material resources are inadequate, such discrimination can result in higher mortality for females. Other evidence suggests that differences in traditional male and female economic roles have contributed to the cultural evolution of differences in the socialization of males and females, and those sex differences in socialization contribute to the sex differences in behaviour that are primarily responsible for men's higher death rates for accidents and other violence (Waldron, 1983a). In conclusion, current evidence suggests a considerable variety of effects of sex roles on sex differences in mortality.

Genetic and environmental influences on sex differences in mortality

Evidence presented in previous sections indicates a number of genetic factors that influence sex differences in mortality. Inherent sex differences in reproductive anatomy and physiology are responsible for women's higher rates of maternal mortality and breast cancer. Effects of endogenous sex hormones may reduce women's risk of ischaemic heart disease. Effects of X chromosome-linked genes may contribute to greater resistance to infectious diseases for females. In addition, males are more vulnerable to X-linked recessive disorders, although that appears to make only a minor contribution to male excess mortality (Waldron, 1976, 1983a).

Evidence presented in previous sections also indicates several environmental factors that influence sex differences in mortality. For example, cultural influences on sex differences in cigarette smoking and alcohol consumption make a major contribution to excess male mortality in more developed countries. Culturally influenced behaviour has also resulted in less adequate nutrition and health care for females in

some cases that in turn has contributed to excess female mortality.

Culturally influenced behavioural effects can reinforce or counteract inherent sex differences in risk. For example, in many countries sex differences in cigarette smoking have reinforced the apparent protective effects of endogenous female sex hormones with respect to ischaemic heart disease. In contrast, in some regions greater access of males to food and health care appears to have counteracted any inherent disadvantage males may have with respect to resistance to infectious diseases.

Another type of interaction between environmental and genetic factors is that environmental factors influence the levels of mortality for causes of death with an inherent sex bias. One example, discussed above, is that improved health care has contributed to a decline in maternal mortality and that has been one factor responsible for the growing mortality advantage for females relative to males at young adult ages. Improved health care has also contributed to a decline in stillbirths due to difficult labour and birth injuries and, as discussed above, that has contributed to a decreasing male disadvantage for late foetal mortality.

Those examples illustrate the important point that the impact of a given genetic factor on sex differences in mortality varies considerably depending on environmental conditions. Thus it can be seen that any estimate of the relative contributions of genetic and environmental factors to sex differences in mortality will apply only for the specific conditions studied and cannot be extrapolated to other conditions.

Numerous additional methodological difficulties have been encountered in attempts to estimate the relative contributions of genetic and environmental factors to sex differences in mortality (Waldron, 1983a). For example, Madigan (1957) attempted to estimate the contribution of genetic factors by studying a group for whom he argued there was little sex difference in environment or social roles—namely, Roman Catholic brothers and sisters engaged in educational work. However, a variety of evidence indicates that there may have been important environmental differences—for example, a more widespread prohibition of smoking for the sisters. Consequently the study does not provide an adequate basis for estimating the relative contributions of genetic and environmental factors to sex differences in mortality (Waldron, 1983a).

Another line of argument has been that the "universality" of male excess mortality among humans, non-human primates and other animals indicates that male excess mortality is due primarily to a fundamental biological difference between the sexes (Mitchell, 1979). Several lines of evidence argue against that hypothesis. A male mortality disadvantage is by no means universal, since excess female mortality has frequently been observed for humans and for many non-human animals (Lopez and Ruzicka, 1983). Also, the history of human sex differences in mortality suggests a lack of continuity between any male mortality disadvantage observed for non-human primates and the male mortality disadvantage for contemporary humans. Data for Europe indicate that from the Paleolithic through the Middle Ages, women generally had higher mortality than men and only in more recent times has it become common for men to have higher mortality than women (Waldron, 1983a). Thus, it appears unlikely that there is any direct link between any general biological disadvantage for males that may be observed in other animals and the contemporary male mortality disadvantage observed for humans. Indeed, the rather rapid historical variations in sex differences in mortality indicate the importance of environmental influences on sex differences in mortality.

In conclusion, there is clear evidence that a variety of genetic and environmental factors influence sex differences in mortality, and that there are important interactions between those genetic and environmental factors. Due to methodological problems, it has not been possible to make quantitative estimates of the relative importance of genetic and environmental factors in determining sex differences in mortality.

Estimates of the contribution of specific factors to sex differences in adult mortality

One approach to estimating the contribution of a specific factor to sex differences in mortality has been to identify a group of men and women who do not differ with respect to that factor and to evaluate sex differences in mortality in that group. For example, two studies have used mortality data for men and women who have never smoked regularly as a basis for estimating the contribution of smoking to sex differences in adult mortality in recent decades in the United States. Retherford (1975) estimated that, for life expectancy from ages 37 to 87 in 1962, 47 per cent of the sex difference was due to smoking (or factors associated with smoking such as heavier alcohol consumption). In contrast, Miller and Gerstein (1983) attributed sex differences in longevity almost entirely to sex differences in cigarette smoking. They based their conclusion on their finding that there was no significant sex difference in life expectancy for adults who had never smoked cigarettes regularly, if deaths due to violence were excluded. (Their data were for adults aged 30 and older in Erie County, Pennsylvania, in 1972-1974.) There are several reasons to believe that Miller and Gerstein's conclusion exaggerates the role of cigarette smoking. At least seven other studies have found that, even among non-smokers, men have higher mortality than women (Doll and others, 1980; Enstrom, 1984; Enstrom and Godley, 1980; Higgins, 1984; Shurtleff, 1974; Waldron, 1976; Wingard and others, 1983). It appears possible that, due to methodological problems, the Miller and Gerstein study may have underestimated the sex difference in mortality for non-smokers and consequently may have overestimated the contribution of cigarette smoking to sex differences in mortality (Enstrom, 1984; Feinleib and

Luoto, 1984). One methodological factor of importance is Miller and Gerstein's exclusion of violent deaths. National mortality statistics show that violent death may be responsible for as much as half of the sex difference in mortality at the younger end of the age range included in their study (calculated from data in United States Department of Health, Education and Welfare, 1977).

A related group of studies has evaluated mortality for Seventh-day Adventists (who for the most part do not smoke or drink) and for alcoholics (who had similar heavy drinking and smoking habits for both sexes) (Phillips and others, 1980; Schmidt and Popham, 1980). In both the cited studies, female mortality was as high as male mortality for the 35-50-year-old age range, but substantial male excess mortality was observed for older adults. That suggests that cigarette smoking, alcohol consumption and other life-style factors may make a large contribution to sex differences in mortality for middle-aged adults, but those factors may be proportionately less important for older adults. Similarly, studies of non-smokers in general population samples suggest that cigarette smoking makes a more important contribution to sex differences in mortality for middle-aged adults than for older adults (Enstrom and Godley, 1980; Waldron, 1976).

Recently, Wingard developed an interesting new approach for estimating the contribution of various factors to sex differences in adult mortality. Sample data are used to evaluate sex differences in various characteristics, such as cigarette smoking, and the effects of each of the characteristics on mortality. The sex difference in mortality is computed with and without statistical adjustment for the characteristics and, if the sex difference in mortality is lower after adjustment, that is interpreted as evidence that those characteristics account for part of the sex difference in mortality. The results have differed for the two contemporary California samples studied, probably due in part to differences in the variables available in the two studies. For one sample, adjustment for sex differences in characteristics produced only minor changes in the estimated sex difference in mortality (Wingard, 1982). It appears that the effects of adjusting for cigarette smoking and high alcohol consumption, which were more common among men, were balanced in part by the effects of adjusting for physical inactivity, which was more common among women. For the second sample, adjustment for individual characteristics reduced the sex mortality ratio by about half, apparently because the effects of adjustment for greater smoking and higher blood pressures among men outweighed the effects of adjustment for higher serum cholesterol among women (Wingard and others, 1983). Differences in the findings of the two studies may reflect not only differences in the variables available for analysis but also differences in the characteristics of the two samples studied. Clearly, the possibility of generalizing findings from studies of that type will be limited by the representativeness of the samples studied. For example, for the first sample studied, sex differences in cigarette smoking and sex differences in mortality were smaller than they are in national data for the United States (Waldron, 1982b). That suggests that the contribution of sex differences in cigarette smoking to sex differences in mortality may be underrepresented in that particular sample.

In conclusion, due to methodological difficulties and limited available data, accurate quantitative estimates of the contribution of specific factors to sex differences in mortality are in general not available. Nevertheless it is clear that sex differences in cigarette smoking make a major contribution to sex differences in adult mortality in the contemporary United States. The relative importance of other factors that influence sex differences in mortality is less clear.

Conclusion

The evidence presented demonstrates that a considerable variety of environmental and genetic factors influence sex differences in mortality, and the relative importance of particular factors varies greatly in different situations. For example, cultural influences on behaviour appear to make a major contribution to sex differences in mortality in most circumstances, but the specific cultural influences of importance vary in different countries and different historical periods. In industrial societies, the most important cultural influences have been social pressures that have encouraged men more than women to smoke cigarettes, to drink heavily, to work in hazardous occupations and to engage in certain other risky behaviour. In some preindustrial societies, it appears that cultural practices that result in less adequate nutrition and health care for females have been a major factor contributing to higher mortality for girls and young women than for boys and young men.

The relative importance of specific genetic factors also varies depending on the situation. Under conditions of inadequate health care and nutrition, women's inherent vulnerability to maternal mortality can make a substantial contribution to excess female mortality at reproductive ages. In industrial societies maternal mortality is relatively unimportant, and the most important genetic contribution may be a protective effect of women's endogenous sex hormones, which may reduce women's risk of ischaemic heart disease.

Historical trends and cross-cultural variations in sex differences in mortality have been linked to a variety of environmental causes. For example, the increasing male mortality disadvantage in industrial countries in the twentieth century was due in large part to the widespread introduction of cigarettes, which, in combination with greater social acceptance of men's smoking, contributed to increasing ischaemic heart disease and lung cancer mortality for males. In addition, improvements in the quality of medical care and general standard of living contributed to decreases in mortality for several causes of death with a female excess and increases in the relative importance of other causes of death with a male excess. Other causes of

historical trends and cross-cultural variation in sex differences in mortality include variation in the extent and impact of discrimination against females and variation in social norms concerning acceptable or desirable male and female behaviour (for example, varying social acceptance of women's smoking).

In light of the diversity and complexity demonstrated by current evidence, it is necessary to reject or to qualify several generalizations that have been proposed previously. For example, earlier claims of a universal male mortality excess are countered by the evidence that excess female mortality has been common for children and young adults in less developed countries. Similarly, although historical trends in sex differentials in mortality often show an increasing male mortality disadvantage, in some cases trends towards a decreasing male mortality disadvantage or the emergence of a female mortality disadvantage have been observed. Thus, analyses of the causes of sex differences in mortality and the causes of trends in sex differences in mortality must take those more complex patterns into account.

Several hypotheses concerning the causes of sex differences in mortality in Western countries should also be modified on the basis of current evidence. For example, it has been hypothesized that there is a general tendency for men to take more health risks and for women to engage in more preventive behaviour. That generalization may be valid for risk-taking that involves physical daring or illegal behaviour, but for other types of risk-taking and for preventive behaviour, patterns of sex differences are inconsistent. Another hypothesis has been that in Western countries women use medical care more than men and that may contribute to women's greater longevity. However, for most types of cancer and ischaemic heart disease, women delay as long as men in seeking medical care, and there is little or no sex difference in prognosis. That suggests that for those major causes of sex differences in mortality, sex differences in use of curative medicine are small and make a negligible contribution to males' mortality disadvantage. Thus, there is a need to clarify the specific ways in which females' greater use of medical care may contribute to their greater longevity.

In conclusion, recent research has provided much interesting information concerning the causes of sex differences in mortality. However, many questions remain unanswered, and much remains to be discovered concerning the multiple, interacting causes of sex difference in mortality. Useful directions for further research have been proposed in previous reviews (Lopez and Ruzicka, 1983; Nathanson, 1984; Waldron, 1983a, 1983b; Wingard, 1984).

Notes

[1] Due to limitations of space and because a number of those topics have been reviewed recently by the present author or others, reference is frequently made to the previous reviews where extensive references to the original sources may be found.

[2] Nathanson has argued against that hypothesis partly because in two studies of employed men and women, no sex difference in Type-A behaviour was found (Nathanson, 1984). However, current evidence indicates that housewives are less Type-A than employed women and, on the average, women are less Type-A than men (Waldron, 1978; Wingard, 1984). Nathanson further argues that there is no evidence that historical trends and cross-cultural variation in sex differences in ischaemic heart disease are paralleled by similar variation in Type-A behaviour. However, in at least one case she cites, the available evidence does suggest a parallelism between variation in Type-A behaviour and ischaemic heart disease risk, since the low ischaemic heart disease risk of Japanese men may be related to low levels of Type-A behaviour (Cohen and others, 1979). Furthermore, since Type-A behaviour is proposed as only one of several factors contributing to the sex difference in ischaemic heart disease mortality, it would be expected that in many cases, variation in sex differences in ischaemic heart disease risk would be due, not to variation in Type-A behaviour, but rather to variation in other factors, particularly cigarette smoking. An additional point raised by Nathanson is that we lack adequate data on whether sex differences in ischaemic heart-disease risk decrease when controls for Type-A behaviour are introduced, and that is a real weakness in the evidence for that hypothesis.

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