

Department of International Economic and Social Affairs

# Population Bulletin of the United Nations

No. 28 1989



**United Nations**

New York, 1990

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ST/ESA/SER.N/28

UNITED NATIONS PUBLICATION

Sales No. E.90.XIII.3

00900

ISBN 92-1-151189-5

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## PREFACE

The purpose of the *Population Bulletin of the United Nations*, as stipulated by the Population Commission, is to publish population studies carried out by the United Nations, its specialized agencies and other organizations with a view to promoting scientific understanding of population questions. The studies are expected to provide a global perspective of demographic issues and to weigh the direct and indirect implications of population policy. The *Bulletin* is intended to be useful to Governments, international organizations, research and training institutions and other bodies that deal with questions relating to population and development.

The *Bulletin* is prepared by the Population Division of the Department of International Economic and Social Affairs of the United Nations Secretariat and published semi-annually in three languages—English, French and Spanish. Copies are distributed widely to users in all member countries of the United Nations.

Although the primary source of the material appearing in the *Bulletin* is the research carried out by the United Nations Secretariat, officials of governmental and non-governmental organizations and individual scholars are occasionally invited to contribute articles.

## Explanatory Notes

Symbols of United Nations documents are composed of capital letters combined with figures. Mention of such a symbol indicates a reference to a United Nations document.

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

The term "billion" signifies a thousand million.

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

A hyphen between years (e.g., 1984-1985) indicates the full period involved, including the beginning and end years; a slash (e.g., 1984/85) indicates a financial year, school year or crop year.

A point (.) is used to indicate decimals.

The following symbols have been used in the tables:

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

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

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

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

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

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## FROM ONE DEMOGRAPHIC TRANSITION TO ANOTHER

*Léon Tabah* \*

### SUMMARY

High fertility and mortality rates, which characterized the demographic situation in most of the developing countries in the 1950s and early 1960s, have been declining. Demographers and other scholars label this declining trend "the demographic transition". It should not be viewed as a theory or a law, but simply as a process through which societies pass from a situation characterized by high fertility and mortality to a new one marked by low fertility and mortality. Although the term was coined to describe the evolution of fertility and mortality in Europe in modern times, it is evident that not all societies follow (or should follow) the same sequence. Today, an increasing number of Asian countries are well advanced in their transition from high to low fertility and mortality rates; in two or three decades some of them have traversed from one extreme of the spectrum to the other, a feat that took more than a century for some European countries. By contrast, the large majority of African countries are at the onset of their transition or have not yet entered it at all. Latin America is somewhere in between the two scenarios. And the industrialized countries are experiencing a post-transitional stage in which not even the replacement of their populations is guaranteed.

Many factors could be adduced to explain the differences between the Asian and the African transitions. The present article gives particular emphasis to the role of certain social, economic and cultural factors that are associated with the process of modernization. It is argued that while some elements tend to depress the supply of children (for example, postponement of entry into marriage accompanied by longer retention of women in the educational system), others tend to increase the supply, as is the case with the reduction of post-partum amenorrhoea associated with the shortening of the breast-feeding period.

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### THREE TRANSITION SCENARIOS

In the confused turmoil of everyday events, the fundamental problems, which are necessarily the long-term ones, are not about to disappear from international life. They will be with us for a long time yet, whether they concern population or the environment. In recent years, demographers have approached them from the standpoint of three main topics.

More than ever before, demographers are wondering about the future of the African continent and—no less than economists or ecologists—they recognize the tragic dimension of the situation there. Africa is the only region of the world where the demographic transition, far from taking its course as in the rest of the third world, has been delayed at best until the beginning of the next century. This presents the prospect of a 10-fold increase in the continent's population between 1950 and 2050, without any certainty of the process being completed by the middle of the next century.<sup>1</sup> Such potential for growth is unprecedented in the history of mankind. The demographic imbalance is all the more serious because it is occurring at a time when economic growth is making it very difficult to maintain current subsistence levels, which are already drastically inadequate. Dependence on outside sources of food is increasing steadily, although only 20 years ago reliable experts were talking about "granaries" capable on their own of feeding the entire continent. It was claimed that the "carrying capacity" of the Sudan could sustain from 5 to 10 times its current population. Ethiopia and, more generally, the tropical areas so well analysed by Pierre Gourou (1966, 1982) were seen as "lands of good hope".

By way of contrast, the second topic of interest to demographers, to which attention is sometimes quite forcefully drawn, somewhat as one might point out a good pupil, is the demographic transition in Asia. The situation would indeed be exceedingly alarming if Asia were to function like Africa, for Asia is home to nearly 60 per cent of the world's population and that proportion is likely to fall only slightly. China and the "four dragons"<sup>2</sup> are moving ahead with their transition at record speed, dragging almost the entire continent in their wake, some more quickly than others. There again the experience is entirely unprecedented. Under relentlessly strict control, China has undergone a veritable shock treatment. Its fertility rate fell by 54 per cent in the 10 years from 1971 to 1981. The only example anywhere approaching it is Japan—Asia again!—where the modestly titled "eugenic protection act" immediately opened the way for legal abortion, which has remained the prime method of limiting population growth. Japan has as many abortions as births and the highest rate of legal abortions in the world. The Republic of Korea is following China's example without having to resort to coercive methods. Other countries are just waiting to follow: Indonesia, the Philippines, Sri Lanka and Thailand.

The third and final topic of interest to demographers is the post-transition status of Europe. The first "baby boom" generation is approaching its mid-40s and the first "baby slump" generation is approaching its mid-20s. Europe as a whole is entering a post-transition period which is something of a blank slate, for it does not fit into any known framework.



It is unprecedented and, thus, an absolute mystery. Experts have had no experience of a phenomenon whose underlying causes are to be found in a society whose basic logic they do not understand, any more than their colleagues in the social sciences do (even though that is their job).

We are confronted with a transition that has failed to take place in Africa, one that has proceeded very rapidly (too rapidly?) in Asia and one that has become firmly entrenched in Europe. The situations which pose the greatest problems are, as always, at the extremes: before and after the transition.

Explanations for the two extreme paths are completely lacking. It must be acknowledged that any attempt to explain demography in terms of population processes would be illusory. Demography can explain only the demographic causes of phenomena—for example, changes in fertility attributable in part to changes in marriage patterns or to age structures; such explanations are purely internal and therefore limited. The real explanation of phenomena on the scale of the demographic transition must be looked for outside the discipline of demography and can be arrived at only through the concerted efforts of researchers in all the social sciences. So far, the problem has aroused little interest except among demographers. Yet it could be a fruitful area for collective endeavour.

We are talking about the long-term demographic transition so dear to Braudel (1969)—that is, with non-recurring and therefore not very predictable phenomena. (When will there be a demonstration of Kondratieff cycles in demography?) So far, demographers have not been very lucky in their predictions. While they have done quite well at predicting future population, sometimes because their mistakes have cancelled each other out, they have been less fortunate in predicting the major demographic events of the past 50 years. They failed to predict at least three major trends: first, the magnitude of the “baby boom”, which cannot be considered a mere deviation from a long downward spiral or a phenomenon of post-war recovery; secondly, the emergence, in the area of population policy, of political will combined with restrictive measures and the means of enforcing them; and, lastly, the vigorous population growth in the third world as a whole after decolonization, following a sharp drop during the last phase of colonization, at least in sub-Saharan Africa. Changes were inevitable, but the facts have always confounded the predictions—due not so much to error as to failure to predict anything that deviated from overall trends.

The three kinds of transition raise many questions. They cannot leave any continent untouched, for what happens in one will inevitably have consequences for the others. Discrepancies between phases of the transition will lead to population movements from one continent to another and will have significant consequences for economic relations.

#### THE SITUATION IN SUB-SAHARAN AFRICA

The first question which comes to mind is: Why is fertility in sub-Saharan Africa an exception in the third world, increasing there while

decreasing elsewhere? Why doesn't modernization, which translates into urbanization, industrialization, education and secularization, overturn the system of population regulation in Africa in the same way that it did in the past in the industrialized countries and is doing elsewhere in the third world? What is the reason for Africa's record population growth? Why is the trend in African fertility different from that of other third world countries at a similar level of development? We can even go further and ask: Why is traditional culture impeding the demographic transition in Africa, when in many Asian countries it is contributing to and accelerating that transition?

### *The population-regulating mechanism*

Africa had, for a long time, a population-regulating mechanism which functioned perfectly, permitting the replacement of generations with slight growth, interspersed with sudden drops brought about by a capricious, and increasingly hostile, natural environment and by epidemics. It would be wrong to think that, in their pre-transitional stage, traditional societies were unfamiliar with family planning. High fertility was, in effect, planned, in the sense that it was necessary for the survival of the group, because of a high mortality rate. Africa's so-called "natural" fertility has always been wanted and is linked to the preservation of certain values. We in the modern age are far too used to the idea of demographic planning as a means of reducing population. In fact, it may be a means of maintaining high fertility. The fertility rate should be considered in relation to the mortality rate—in particular, as a response to a high mortality rate. In traditional societies, the community itself very often puts pressure on families to maintain a level of fertility, in keeping with the norms. Etienne and Francine van de Walle (1988) note that, in Senegal, careless or irresponsible women who become pregnant too quickly are the targets of taunts, mockery and censure. In Senegal, the Wolof term *neffe*, meaning bad luck, is used to describe a woman who becomes pregnant before she has weaned her most recent child. Similar expressions exist among the Yoruba of Nigeria and in a number of other countries, including Mali, Togo and Zaire.

Traditional Africa's population-balancing mechanisms involve a subtle combination of "bridges" between demographic factors (age at the onset of fertility, birth-spacing and age at which reproductive capacity ends), psychological factors (demand for children), cultural factors (post-partum abstinence, the claim that "sexual relations can taint the mother's milk") and biological factors (post-partum sterility related to breast-feeding).

Of the three demographic factors which regulate a woman's reproductive life, it is the interval between births which offers African women a clear choice of possible action. When the interval is too short, it endangers the child's health. Hobcraft, McDonald and Rutstein (1984) have shown that children born after an interval of less than two years face a higher risk of mortality than other children.

Long periods of post-partum abstinence are justified by concern for the health of both mother and infant. The purpose is not so much to limit

births as to protect the lives of children already born and to preserve the mother's health so that she may conceive again. This is particularly important in the light of health and food conditions in sub-Saharan Africa.

The population-balancing mechanism works as follows: high mortality rates make couples reluctant to limit their cumulative fertility. On the contrary, they want to maintain it, for children can make a substantial contribution to the family economy. Children are a source of revenue and social prestige and an investment for old age. They are the poor man's "social security". Many calculations have been made—for instance, by Ryder (1974), McNicoll (1984) and Locoh (1984)—to show that the father of a family who aims to have at least two living sons when he reaches old age should produce eight children in the high mortality conditions observed in certain rural areas of black Africa. There is, therefore, a strong "demand for children"; moreover, because of the high mortality rate, women want to "replace" the children they have lost. The "supply of children" is reduced to half the physiological maximum by the poor health of mothers (more or less temporary sterility, related to the early age at which a woman first becomes pregnant, to sexually transmitted diseases and perhaps also to poor nutrition), the period of physiological sterility brought on by post-partum amenorrhoea and voluntary sterility as a result of post-partum abstinence. It was only recently that, thanks to the World Fertility Survey, it became possible to calculate the combined effect of the two post-partum sterilities on fertility in a number of African countries. According to Etienne and Francine van de Walle (1988), who cite various survey results, abstinence adds five months to the interval between births in Ghana and Lesotho, two months in Kenya and more among the Yoruba of Nigeria.

All things considered, the average interval between births is about three years in sub-Saharan Africa. In view of the high mortality rate, that may be considered the optimum interval for ensuring the long-term replacement of the population, the average number of births ranging from five in Gabon (where 30 per cent of the women are affected by physiological sterility) to more than six or seven elsewhere, while women's potential fertility is between 13 and 15 births.

The balancing mechanism appears to be perfectly regulated and seems to be perceived by individuals, especially by women, who are quite conscious of it even if they do not always have a good understanding of the direct relationship between breast-feeding and amenorrhoea (van de Walle and van de Walle, 1988).

#### *The detrimental effects of modernization*

Modernization has beneficial effects but, at the same time, has the paradoxical effect of upsetting the secular demographic balance. It erodes that balance by removing many obligations and prohibitions. This is indeed the sense in which Boudon (1977) refers to "perverse" effects, since the accumulation of individual decisions leads to undesirable results at the collective level.

The process of modernization has contradictory effects on fertility. It tends to increase the supply of children because it results in a better state of health for women, a decline in breast-feeding—or, more precisely, a reduction in its duration—and the abandonment of post-partum abstinence—or rather, here again, a reduction in its duration. But the process of modernization also tends to reduce the supply of children by increasing the age at marriage, which, as we shall see, is still very low in sub-Saharan Africa, by contrast with northern Africa or, more generally, the rest of the third world. The demand for children also tends to decline, particularly among the more educated, least economically disadvantaged classes, who live mainly in urban areas. Sooner or later, women want gradually to free themselves of the burden of repeated pregnancies and, therefore, modern contraception prevails, first with very low rates of use and then at higher levels.

These factors do not all operate simultaneously, however, and it is the lags that create problems: population growth speeds up as mortality declines. At first, the factors that tend to increase the supply of children are dominant, and fertility tends to increase. This period may last from 10 to 20 years and seems to be the one now prevailing in a number of African countries. Bongaarts and Frank (1988) note that in Kenya, where the fertility rate has increased to 8.3 births per woman, post-partum abstinence has declined to four months, and the combined effects of the reduction in the duration of abstinence and breast-feeding reduce the period of sterility to between 11 and 13 months. In Rwanda, where the fertility rate is even higher than in Kenya (8.6), post-partum abstinence has apparently almost disappeared.

If a period of decline is to begin and be maintained on a lasting basis in sub-Saharan Africa, the factors which tend to reduce both demand and supply must prevail over the factors which tend to increase supply. Meanwhile, contraceptive prevalence can reach high levels, as in Zimbabwe, without a significant decline in fertility (United Nations, 1989b). Indeed, despite a high rate of contraceptive use (38 per cent of married women of child-bearing age), Zimbabwe's fertility rate for the period 1985-1990 is estimated at 5.8 births per woman. Zimbabwe is the first breach in the wall of traditional society (and other countries will follow), but for the time being contraceptive prevalence in sub-Saharan Africa is still under 10 per cent on average and it will be one or two more decades before we see a reduction in the rate of population growth. And, as has been noted a number of times, what we are seeing now is an increase in the rate.

But to return to our basic question: Why is the effect of modernization in Africa contrary to its effect elsewhere in the third world, when there is no reason to believe that women's potential fertility varies from one population to another, when the economic contribution of children was a factor in the demand for children before transition and when breast-feeding and post-partum abstinence exist in all traditional societies? Why has the traditional system for the preservation of life proved more fragile in Africa than elsewhere in the third world? Why has the system broken

down? Putting the question in this way of course implies the assumption that a high rate of population growth gives rise to intractable problems, particularly at the economic level. Yet how can one *fail* to consider a growth of more than 3 per cent excessive in the current international economic environment where competition has never been so fierce?

### *Factors inhibiting access to the transitional stage*

#### *School attendance*

Caldwell (1979) and Caldwell and McDonald (1981) believe that the main factor inhibiting African societies from entering the transitional stage is children's school attendance. All the statistical evidence agrees that the rate of school attendance of African children is on the whole lower than that of Asian and Latin American children (with a few rare exceptions, such as Afghanistan, Bangladesh and perhaps also Pakistan), particularly for girls. Furthermore, the economic burden on parents always increases when school attendance becomes an increasingly widespread norm. Children contribute less to the household economy, they become "net consumers" instead of "net producers" and the "family as a means of production" is gradually replaced by the "labour market as a means of production" (Caldwell, 1979). These changes naturally give parents a growing incentive to limit the size of their families. Africa would undoubtedly not be experiencing its current problems if the educational system south of the Sahara were more advanced. Development capacity is considerably higher to the north of the Sahara, where school-attendance rates are high.

#### *Infant and child mortality*

A second cause of sub-Saharan Africa's exceptionally high fertility rate is the fact that infant and child mortality is higher there than elsewhere in the third world and creates a disincentive for limiting births. This disincentive remains even though such mortality has declined and despite an increasingly hostile environment marked by drought, famine and ecological disasters in certain areas. Of course, the improvement in mortality is not peculiar to Africa and is no more conspicuous there than elsewhere in the third world, but it is taking place in a different demographic context, in that fertility elsewhere has entered into a transition that is virtually non-existent in sub-Saharan Africa.

The preservation of life is the main driving force behind African reproductive behaviour. As we said earlier, after the death of one child, mothers want to make up with the birth of another; at least, they are not inclined to reduce their fertility. For them, children represent security in their married life and in their old age. Planned parenthood is for them anything but planned limitation. It is understandable that, taken collectively, a population does not shift in one day from planning in order to maintain or increase the number of births to planning for the very opposite. A period of adjustment to the reduction in infant mortality is neces-

sary, and that is the period which sub-Saharan Africa is now in the midst of.

### *Breast-feeding and post-partum abstinence*

A third factor explaining why modernization has the effect of increasing fertility in Africa is the disappearance of the long period of breast-feeding and—even more so—the period of post-partum abstinence. The Koran prescribes sexual abstinence for four months after childbirth. Caldwell and Caldwell (1977) say that the period is usually longer and can last 10 months or more among the Yoruba of Nigeria. For certain populations, abstinence ends with the weaning of the infant. For most populations, the period of abstinence and the duration of amenorrhoea are shorter than that of breast-feeding. The shortening of those periods restores to women their temporarily lost fecundity, and in the absence of incentives and adequate means to limit births, fertility is bound to increase.

### *Marriage patterns*

A fourth reason for the high fertility and rapid population growth in sub-Saharan Africa is to be found in African marriage patterns, which are fairly unusual and evolve very slowly (Gendreau and Gubry, 1988). Modernization, for the time being, has had little influence on the formation of families. African marriage is a historic institution, deriving more from custom than from law. The contract is almost always concluded by religious or customary ceremonies. It is more a contract between families than between individuals, and its purpose is procreation. Virtually all African women get married, permanent celibacy being practically unknown, in contrast with industrialized societies and even the Asian countries. African women marry at a very early age and are far younger than their husbands, a situation which encourages polygamy or makes it demographically possible, since the “supply” of women on the marriage market is far greater than the “demand” from men. Women usually marry without first becoming pregnant, although an increase in the number of unmarried pregnant adolescents has been observed as urbanization and school attendance have increased—one of the “perverse” aspects of modernization. According to Gyepi-Garbrah, Nichols and Kpedekpo (1985), increased school attendance among girls in sub-Saharan Africa raises the marriage age and simultaneously the proportion of pregnancies among unmarried adolescent girls.

Women thus embark very early on a “reproductive career” (Ilinigumugabo and Randriambanona, 1988), and they abandon it only when their fertility ends, although they remain in a marital partnership until the end of their lives. During their “career”, they may experience considerable marital mobility because, whether widowed, separated or divorced, they are rapidly “recycled”, with the result that few women are left on their own. (The opposite—many women left on their own—is fast becoming a major social problem in the industrialized countries.) Thus, African women change partners fairly often during their reproductive lives. Volun-

tary separation is frequent, with sterility often the pretext or cause. Sala-Diakanda (1988) has calculated that in Cameroon the proportion of infertile women married more than once was 23.7 per cent in 1978. The average length of first broken marriages is rather short: 6.4 years in Rwanda, 6 years in Kenya, 9.6 in Gabon etc. There is nearly always a second monogamous or polygamous marriage, and the period between two marriages is short, so that the impact in terms of lost fertility time is minimal.

Polygamy is more prevalent in sub-Saharan African than anywhere else in the world. It occurs more often there than in other parts of the third world, including the Muslim areas of Western Asia. To cite a few figures: in western Zaire, the proportion of women married or in a union who live in polygamous unions is 28.1 per cent for urban and rural areas combined and 33.6 per cent for rural areas alone; in Cameroon it is 43 per cent and in Ghana, 34.4 per cent (United Nations, forthcoming).

It is claimed that polygamy is a socially acceptable means of alleviating sub-fecundity, for want of a proper remedy for that condition. It is often justified on the grounds that it maximizes the reproduction of offspring. It clearly promotes post-partum abstinence while, conversely, the custom of lengthy periods of post-partum abstinence encourages polygamy. In all traditional societies, fertility—and beyond it, the family—is revered because the collective consciousness holds that children are all-important. Consequently, polygamy and its corollary, conjugal mobility, are permissible. Western societies have lost this notion of fertility as something sacred, even though they increasingly tolerate conjugal mobility, as demonstrated by the growing frequency of divorce and remarriage.

While it has long been argued that polygamy is bound to disappear in Africa and elsewhere in the relatively near future, the institution has proved to be entrenched in sub-Saharan Africa. Whereas it was formerly the prerogative of old men, particularly in rural areas, it is now increasingly seen in urban areas among young heads of family (Romaniuk, 1988). The systematic remarriage of women has much to do with the persistence of the practice, which demonstrates the hold which the forces of tradition continue to exert in Africa and how unlikely it is that rapid changes in behaviour will occur. Polygamy will disappear only gradually. Some authors (Pison, 1986) indeed hold that the marriage market would be thrown into chaos if the practice were abruptly ended.

There has also been speculation as to the effect which the disappearance of polygamy would have on fertility, since the proportion of sterile women is often higher in polygamous unions than in monogamous ones: husbands take a second wife when the first proves to be sterile, but do not divorce the first wife. In such a situation, the disappearance of polygamy might serve to depress fertility. It is also possible, however, that the fertility of non-sterile women living in polygamous unions may be lower than that of women of the same age living in monogamous unions, in which case the disappearance of polygamy would have a positive effect on fertility. In any event, a reduction in female sterility ought to lead to a decline in polygamy, since sterility could no longer be invoked by the hus-

band as a reason for taking another wife. This would be a test of the real motives underlying polygamy.

Of course, polygamy creates a disparity between reproduction rates for males and females. While there is little difference between the reproduction rates for men and women in monogamous populations, the case is somewhat different in polygamous populations. Pison (1988) calculated that among the Peul Bandé of Senegal the gross reproduction rate is 3.2 when based on mothers and daughters and 5.8 when based on fathers and sons, while cumulative lifetime fertility is 6.7 births for women and 11.2 births for men. Calculating intrinsic growth rates by sex would yield inherently contradictory results in sub-Saharan Africa. Actually, such calculations would be only theoretical in nature. It is a fact, however, that under polygamy, women have far fewer descendants than do men.

It is interesting to note that polygamy and conjugal mobility, which is both its cause and its consequence, do have an effect on genetic inheritance. In fact, the effect is twofold. First, polygamy increases the number of cousins and half-cousins. Pison has calculated that, among the Peul Bandé, the average number of first cousins having a common grandfather is 74, whereas 48 on average have a common grandmother. Given that certain genes are known to be sex-specific, the consequences of this cannot be overlooked. Such multiplication has the greatest impact on cousins and half-cousins on the paternal side. Secondly, the proliferation of cousins results in a significant increase in the degree of consanguinity among the population and thus a higher probability of genetic homogeneity than would be the case where polygamy does not exist.

### *Sterility*

A fifth reason for the increase in fertility which is accompanying modernization in Africa has to do with sterility resulting from sexually transmitted diseases. This is a relatively recent phenomenon in which "cultural" factors play an important role, as was noted first by Retel-Laurentin (1974) and more recently by Caldwell (1979) and Sala-Diakanda (1988). This sterility, which obviously is not peculiar to sub-Saharan Africa, but is more prevalent there, exists throughout a vast area covering many Central African ethnic groups, extending as far as the shores of Lake Victoria and Lake Chad. It lies at the root of many of the justifications given for polygamy and conjugal or non-conjugal mobility (the latter being referred to as "le deuxième bureau" in parts of Africa). Now, however, sterility is on the decline, largely as a result of efforts to combat venereal disease, and one can venture to say that the decline will lead to an increase in the number of births, unless it is offset by factors which have an opposite effect on fertility, such as raising the age at marriage and increasing the spread of voluntary infertility. However, neither of those phenomena is sufficiently widespread, except in Zimbabwe.

### THE SITUATION IN ASIA

Comparing the demographic transition in Africa with that in Asia is an exercise that, at first glance, yields clear-cut results if we limit our-



selves to demographic analysis of fertility and mortality. It would be an over-simplification to say that what we have in Africa is a world in which growing modernization contrasts with an economically undesirable rate of population growth and in Asia a world in which a number of countries are undergoing a relatively rapid transition and population is in harmony with economic growth.

A description of population trends in Asia is practically a description of trends in Africa in reverse. True, Asia does not present a homogeneous picture, and it would be difficult to formulate a composite. Differences are even more pronounced there than in sub-Saharan Africa, for while virtually all African countries are in the pre-transition phase, Asia offers every conceivable scenario, from pre-transition (Afghanistan, Pakistan and Western Asia) to post-transition (Hong Kong, the Republic of Korea, Singapore and Taiwan Province) through a number of intermediate situations, with some countries in the early stages of transition (Bangladesh, Islamic Republic of Iran, Lao People's Democratic Republic) and others in the middle (India, Indonesia, Malaysia, the Philippines, Viet Nam) or final (China, Thailand, Sri Lanka) stages. If we exclude the countries in which Muslim culture is dominant, we could say that all the Asian countries are at a comparatively advanced stage of the transition or have even emerged from it. And even the largest Muslim country, Indonesia, is in rapid transition, followed, although at a slower pace, by the Muslim population of Malaysia. The countries which have begun the transition in one way or another account for nearly 90 per cent of Asia's population, while the sub-Saharan African countries that have begun the transition account for only 10 per cent of that region's population.

The only Asian country whose situation with regard to the transition approaches that of the sub-Saharan countries is Pakistan. According to Cleland and Shah (1988), fertility there has increased slightly in recent years. The increase appears to be associated with a sharp decrease in the period of breast-feeding, by a process that is thus analogous to the one that accounts for the increase in African fertility. By contrast, fertility has begun to decline in Bangladesh, falling by approximately 10-20 per cent, according to the same authors.

What is striking in Asia is the speed at which the transition is taking place, a speed which often exceeds that of the industrialized countries during their transition. In the early 1960s, China's fertility level was comparable to the average for Africa. Today, China's fertility level ranks with that of the European countries only 15 years ago, as they emerged from their transition. One significant difference is that China completed the process so quickly that the age structure has not had time to catch up and holds a potential for growth that will not diminish for several decades to come.

The transition has been almost as rapid in the Republic of Korea, where the total fertility rate has fallen from more than 6 in 1960 to 2 in recent years, below the replacement level and even lower than that of China. This transition would not have been so rapid were it not for the transformations in Korean society that began immediately after the 1950-

1953 war that devastated the country. Per capita gross national product in constant 1960 US dollars had increased 6.5 times over by the end of the 1970s, and reached \$US 3,275 by 1985.<sup>3</sup> Anthropological studies on East Asia, where the Chinese culture is dominant, have shown that these changes took place while traditional values remained constant (Taeuber, 1959). The change in fertility began in the urban areas and subsequently spread throughout the country. In the absence of adequate information on contraception, abortion became widespread from the outset: one abortion for every two births (Kwon, 1988). This is an indication of how strongly people were motivated to have small families. The abortion rate was still one abortion for every two births in 1981.

The case of Thailand, whose economic success is seldom mentioned, although it is as impressive as that of the "four dragons", is equally remarkable. Its GNP has increased at an annual rate of 6 per cent since 1960 and per capita GNP is now \$US 1,942 a year. Yet, Thailand is a country that has undergone relatively little urbanization (20 per cent of the population may be classified as urban). The total fertility rate fell from 6.6 in 1950-1955 to 6.1 in 1965-1970, and plunged to 3.5 in 1980-1985. The rise in age at marriage (22.9 years for Buddhist women and 19.6 years for Muslim women) is one of the reasons for the drop in fertility, but the essential factor has been the Government's support for family-planning services, which are extremely well-managed and integrated in health services. The contraceptive prevalence rate in 1984 was one of the highest in the third world: 64.6 per cent (Robinson and Rachapaetayakom, 1988); by 1987 it was even higher: 67.5 per cent (United Nations, 1989b). (However, the proportion of women aged 15-29 years who have two children and have undergone sterilization is 20 per cent when the children are girls, 26 per cent when they are boys and 26 per cent when there is one boy and one girl, thus indicating an apparent preference for male births.) In general, though, one can surmise that Buddhist culture has been supportive of family planning. Religious sentiment permeates everyday life at all levels of Thai society. Few differences in fertility can be discerned among people of different educational levels, for religion governs behaviour regardless of social class.

Malaysia is another Asian country which has experienced rapid yet little-publicized economic growth. Per capita income soared from \$US 928 in 1965 to \$US 1,224 in 1975 and \$US 3,758 in 1985. At the same time, population trends have followed a pattern consistent with the transition theory. Age at marriage has increased: 20 per cent of women of Chinese or Indian background born in the period 1950-1954 were unmarried at age 30; the percentage was slightly lower among Malay women, whose culture is more rural and less affected by modernization (Leete and Tan, 1988). Fertility has declined to 3.5 births per woman (1980-1985), higher than the rates for China, Indonesia, the Republic of Korea, Sri Lanka and Thailand, even though Malaysia has a higher level of economic development than those countries. The decline in fertility in Malaysia seems to be slowing for two possibly related reasons: a change in the Government's attitude in 1982 from anti-natalist to pro-natalist and the emergence of a fundamental-

ist movement among the Malay population (Leete and Tan, 1988). It is interesting to note that the Malay population, which accounts for 57 per cent of the total population, had long been less fertile than the population of Chinese and Indian origin. Now, however, as a result of the more pronounced decline among the latter two groups, the Malay population has a higher fertility rate (4.7) than the Chinese (2.3) and Indian (3.0) populations. There is no doubt that the Malay population, with its Islamic culture, is resisting the rapid decline in fertility which the other two cultural communities are experiencing.

In the Philippines (1985 per capita GNP was \$US 1,409), the decline in fertility began relatively late—as recently as the early 1970s—but subsequently accelerated, only to slow down again in recent years, as in Malaysia. The total fertility rate, which was 7.3 in 1950-1955, a level comparable to that of many sub-Saharan African countries today, fell to 6.0 in 1965-1970 and is now around 4.3 (1985-1990), a rate comparable to that of India. The slowing down of the decline in fertility would appear to be due to a new phenomenon in Philippine society: an increase in pre-marital conception among the young generation (Cabigon, 1988), which appears to be yet another “perverse” effect of modernization.

Sri Lanka has been experiencing economic problems for several decades. Per capita GNP was \$US 1,268 in 1985. The country's fertility rate (2.7) is slightly higher than that of Thailand (2.6). The younger generation appears to have responded to the economic crisis by delaying both marriage and the birth of the first child (Thapa, Piccino and Tsui, 1988). In addition to economic difficulties, the expansion of education seems to have played an important role in the decline in fertility. More than the increase in age at marriage, the spontaneous adoption of family planning has played a decisive role in the sharp decline in marital fertility. It is interesting to note that in Sri Lanka there has been no change in the duration of amenorrhoea associated with breast-feeding, which remains relatively long (15.9 months) and has thus contributed to the decline in fertility. Thus in Sri Lanka, while the level of economic development is very low, the decline in fertility is attributable to a number of factors: a relatively high school-attendance rate, a higher age at marriage, well-organized family planning services and a relatively long breast-feeding period.

The case of Indonesia (per capita GNP in 1985 was \$US 1,135, fairly close to that of Sri Lanka, but is rising rapidly, owing to oil resources) is particularly interesting. It has the world's fifth largest population; among Islamic countries it has the largest population and the lowest fertility rate, owing to a strong governmental policy aimed at reducing the size of the population, redistributing the population geographically and promoting highly organized family planning services. The total fertility rate has declined from 5.1 in 1970-1975 to around 3.3 at present.

#### AFRICAN VULNERABILITY AND ASIAN DYNAMISM

We are now in a better position to try to answer the questions we

raised on the differences between population trends in African and Asian societies.

Let us first consider the demographic differences, remembering that purely demographic explanations are purely self-referential, bearing only on themselves, because all that they demonstrate are variables lying somewhere between basic causes and behaviours.

### *Demographic factors*

Fertility rates are stable or rising slightly in sub-Saharan Africa, whereas they are dropping everywhere else in the third world and particularly in Asia, except for certain countries with a Muslim tradition, notably those in Western Asia. In some Southern Asian countries with a Muslim tradition, however, we are seeing a decided drop in fertility.

In Asia as in Africa, the breast-feeding period is fairly long but is declining. There is no indication that, broadly speaking, breast-feeding differs much in duration from one continent to another. There are, however, considerable variations from one country to another within the same continent. One thing that does seem certain is that the prohibition on post-partum sexual relations appears to be more widely observed in Africa than in Asia, although it is starting to weaken in Africa as a result of modernization, leading to an earlier recovery of fecundity after birth.<sup>4</sup>

On the whole, people marry later and less frequently in Asia than in sub-Saharan Africa. The trend towards a higher age at marriage is more pronounced and began earlier in Asia. There, polygamy is virtually non-existent or minimal in the Muslim population, while it remains widespread in sub-Saharan Africa. General marriage conditions put women at greater "risk" of fertility during a longer period of their lives in sub-Saharan Africa than in Asia. Increased age at marriage has had a definite impact in terms of reducing fertility in Asia, but there has been little such impact in sub-Saharan Africa.

In Asia, the age of first birth has risen and the age at last birth has dropped, so that the period of "risk" has diminished considerably. McDonald (1988) estimates that the interval between the mean age at first birth and the mean age at last birth has dropped from 13-17 years to about 4 years in the Asian countries that have made the transition, as happened in the industrialized countries. On the other hand, the interval does not seem to have changed much in Africa.

Having a small number of children is considered a desirable goal in Asia and is culturally acceptable, while the reverse is true in sub-Saharan Africa (McDonald, 1988), where the community puts pressure on women to be extremely fertile. In Asia the community is neutral or even openly hostile to high fertility. The hostility is evident in China and will apparently soon be so in other Asian countries.

For a long time, the African countries were opposed to the idea that population growth could stand in the way of their development strategies, and that was the point of view which they and the Latin American coun-

tries defended and succeeded in imposing at the 1974 World Population Conference at Bucharest. Their opposition has gradually subsided, especially after their erstwhile Latin American allies joined the other camp. The result is that at the Arusha Population Conference in 1984 the African Governments took the position that a high rate of population growth could have an adverse effect on the attainment of national objectives (Economic Commission for Africa, 1984). Although there has been a definite change of attitude, Africa can still be said to fall far short of the overall position of the third world on the question of population and development; it still takes a mostly non-interventionist attitude to fertility (Chamie, 1988), as opposed to the strongly interventionist position long held by the Asian Governments.

The few African Governments that are in favour of adopting measures to curtail population growth are finding it difficult to achieve their objectives. That is especially true of Ghana, Kenya, Rwanda and Senegal, where, paradoxically, fertility indicators for some groups are not only among the highest in Africa but also on the rise. (Some writers use this as an argument to show that population policies will be ineffective in Africa as long as the conditions in which they are applied do not change.) One explanation for the paradox might be as follows: the countries that consider intervention in population matters necessary are also the ones most open to modernization, which, as we have seen, has the effect of temporarily increasing fertility. This appears to be the price that has to be paid in order to start the transition. European countries paid the same price at the turn of the century. In Europe, however, there was less of a rise in fertility in the pre-transition period, and it lasted for a shorter time. When Asian Governments decided population growth rates must be curbed, the population was generally fairly receptive to the idea, with the exception of Pakistan, where the situation in many ways resembles that in African countries.

In Asia generally, Governments have given substantial support to family-planning programmes. We should also mention the accessibility of services and—a key factor to which we shall return later—a cultural setting that is generally more receptive to population policies.

The laws on abortion and sterilization are very restrictive in Africa (Chamie, 1988). Legislation is decidedly more open to those practices in Asia, as can be seen from the statistics on abortion and sterilization in Japan, the Republic of Korea, Thailand and India, and of course China. Sterilization is fairly common in Asia and is even the birth-control method of choice in many countries of either Chinese or Indian culture. No African Government would venture into this area.

The demographic factors outlined above are not the only ones—far from it. We must add to them at least two other types of explanations, which are themselves interrelated: economic factors, and cultural factors. For how can we, in fact, separate the success of the population policies adopted by Asian Governments, especially those of Chinese culture, from their economic achievements in agriculture or industry or from the existence of value systems that create and encourage rational behaviour in

everyday life? One is tempted to say of the dominant ethical systems in Asia, especially the Chinese, what Max Weber (1930) said of the Protestant ethic in relation to the success of Anglo-Saxon capitalism: namely, that the religious value encouraged the emergence of, and reinforced, a "worldly asceticism".

### *Economic factors*

It must be acknowledged that the international economic environment is much more favourable to the Asian countries than to the African countries. The presence of Japan, which took 80 years to master modern technology—for which, let it be said, it paid dearly in social terms—has meant the inevitable diffusion of Japanese industry to a number of Pacific outposts, thereby favouring the emergence and development of Asia's new industrialized countries. Nothing similar exists in Africa, whose insolvency is such that investors are turning away from the continent.

The diversification, strengthening and global expansion of the Japanese boom is only in its early stages, but it has already shaken world commercial and financial structures, and has done so with little oil, few raw materials and at less social cost than in Latin America. It does not appear to be hampered by the population factor. On the contrary, moderate population growth is a powerful factor in sustaining development, as past experience has always demonstrated, in that it provides a steady influx of young workers who can adapt to modern technologies. Asia has shown that it is capable of borrowing the West's technological and intellectual tools at a staggering rate. Revel (1982) writes: "Given the extraordinary spirit of achievement of Asian civilizations, let us imagine for a moment what kind of a challenge the productivity and aggressive export policies of a capitalist China would represent for us: 10 Japans, 25 Republics of Korea, 55 Provinces of Taiwan (China), 200 Hong Kongs, 400 Singapores—we would be swamped. Yet, we shall have to face that challenge one day, if China ever emerges from the cold storage of history."

We can only agree with Gourou (1966) when he says that the development disparities between tropical sub-Saharan Africa and tropical Asia have little to do with the physical environment. Both regions are afflicted by diseases attributable to the heat and humidity, which are conducive to the proliferation and survival of germs, parasites and disease-transmitting insects and stand in the way of economic development. However, the medical, financial and administrative infrastructures used to tackle health problems differ in the two cases. Communicable and parasitic diseases are more rampant and more deadly in Africa primarily because children there, even when they are immunized, have less resistance because they are malnourished (Akoto and Hill, 1988) and are raised in large families where mothers frequently use traditional practices which can be dangerous even in the case of otherwise benign illnesses. (Akoto and Hill cite the example of the Komba, who eliminate water and milk from the diet of children with measles.) The strategy of primary health care, first introduced in Asia, has generally failed in Africa since 1980 (Van Lerberghe and Pangu, 1988).

The extent to which agricultural development has played a key role in all the issues facing the third world, including of course those having to do with health, can never be overstated. It has often been said that the key to successful development is to avoid sacrificing agriculture to industry or—more precisely, as Bairoch (1963) demonstrated—to give priority to agriculture over industrial development, as happened in Europe before its economic expansion. The Republic of Korea carried out its agrarian reform shortly after the 1950-1953 war. The Republic of Korea, Thailand, the Province of Taiwan (China), the Philippines and even Pakistan have achieved food self-sufficiency. India, too, has become self-sufficient in food, which does not of course mean that Indians are all well-nourished—only that they are less malnourished. Grain imports are declining in Malaysia and Sri Lanka. Thailand exports rice and grains. These examples stand in stark contrast to Africa, where nearly every country is experiencing growing food dependency. The underlying cause of Africa's agricultural deficiencies is not so much a hostile natural environment as technological backwardness. Nature offers the same potential in Africa as in tropical Asia, but people are unable to tap it because they do not have the necessary technology. It bears repeating that opportunities exist in abundance, and the frequently cited example of the Bamiléké of northern Cameroon, who have adopted European-style commercial food production to feed the population in their cities and are well organized in selling their products, shows that it is possible to make the transition from extensive to intensive farming in tropical Africa. There is little doubt that some civilizations are more receptive than others to new production techniques. Pierre Gourou (1982) said that the best example of disparities between similar regions was the development of river deltas. In Asia, they are usually highly developed, while in Africa and tropical Latin America, no river delta has ever been developed, for lack of expertise in hydraulic techniques and in mobilizing human resources.

There can be no economic future for Africa until African small farmers produce what the population consumes and Africa acquires an import capacity by regaining a place on the tropical commodities market in the current international division of trade, which is hardly any more favourable to Africa than it was in colonial times.

Even if it is true that, on the whole, Asia began its population transition under more favourable economic conditions than those prevailing in Africa today, economic factors alone do not explain Asia's success. The Republic of Korea underwent a lengthy period of Japanese colonization and emerged from the 1950-1953 war with a per capita income of some \$US 100 per year and a fertility comparable to the present average in Africa. In 1960, Thailand had a low per capita income and a fertility rate as high as that of the Republic of Korea. As a result of its economic crisis, the growth of Sri Lanka's GNP has been limited, yet its fertility rate, like that of Thailand, has fallen to a level close to that of Europe when it emerged from the transition.

The successful third world countries are those that have realized that they cannot content themselves with exporting their raw materials but must

adopt three interrelated policies: an effective agricultural policy; an industrialization policy geared from the outset towards exports; and a bold population policy. The three cannot be separated. They are all present in Malaysia, Taiwan Province (China) and the Republic of Korea and will soon be present in Indonesia, the Philippines and Thailand.

It should also be noted that, with the exception of China, most Asian countries have rejected state control of the economy and adopted the market economy system, which has always been associated with a liberal population policy.

### *Cultural conditions*

Many writers attach more weight to cultural factors than to economic factors in explaining the success of the Asian countries in making their demographic transition, or attribute Asia's economic success to a setting or a combination of circumstances in which cultural factors play a dominant role. Philosophies, beliefs, mentalities, religions, languages, family systems, land tenure systems and community institutions have helped to mobilize Asian populations and have moved them in a direction favourable to the development of natural resources. Tropical Asia offers considerable hope because of the existence of strong organizational capacities which play a central role in all areas, including family formation and growth for individuals as well as society. Technologies inherited from the past co-exist successfully with those acquired from the outside world. The generalized economic boom can be attributed to a mentality, methods and value systems that have enabled Asia to draw on the technological contributions of other civilizations without cutting itself off from its historical roots.

At a recent Bangkok seminar on the fertility transition in Asia, one idea that pervaded the debate was that although Asia's strength lay in the quality and variety of its cultures, the change in fertility had occurred without any major parallel change in traditions. In the Republic of Korea, population pressures were so great that fertility began to decline even before any economic development or industrialization took place, particularly at Seoul, where extreme poverty was rampant, the principal method of fertility reduction being abortion (Kwon, 1988). Raising the age at marriage played a key role, since the almost universal poverty forced young couples to postpone their marriage plans. Only later did social change begin to gather momentum, but it occurred so rapidly that traditions were left intact. Cultural changes certainly cannot be made so quickly. Koreans usually say that they are deeply attached to family values, which take precedence over individual interests.

Kwon (1988) reports on a study of the Japanese by Takao Sofue, which concludes that traditional values—group spirit, respect for rank, acceptance of goals handed down from above, obedience to rules and controls, submission to authority, respect for the family, deference and courtesy, acceptance and compromise, fatalism, bonds between mother and son, filial devotion, male supremacy, emphasis on education and knowledge—are still entirely valid today. All these rules of behaviour



would sum up the characteristics of Confucian thought rather well. It seems only natural that in such a context, the exhortations of authorities would be accepted and followed.

It is interesting to note that the cases of China, Indonesia, the Republic of Korea, Sri Lanka and Thailand show that Governments can achieve similar results under different political systems, without having to resort to coercive measures, but simply using incentives. We can speculate that China's fertility rate would have declined even if restrictive measures had not been taken. The cultural environment was ripe for such a change; the results simply would have been achieved more slowly.

In Africa, the impact of modernization has weakened some traditional customs—resulting in a reduction in the periods of post-partum sexual abstinence and breast-feeding—but has left others, such as early marriage, intact. It has not changed reproductive behaviour in terms of the adoption of family-planning measures. As a result, fertility was bound to remain as it was or even increase.

Modernization has had a greater impact in Asia, bringing about changes in both marriage patterns and reproductive behaviour. This is because the cultural setting, particularly among populations influenced by Chinese culture, lent itself to such changes.

There is one cardinal Confucian thought—what the Chinese call *he*—which, as it were, governs the responsibilities and duties between parents and children. Those responsibilities and duties have been profoundly changed by the modernization process. Ryder (1983) described the mechanism accurately in what he called the “generation gap”. When fertility is high, it is easy for children to fulfil their duties towards their parents because those duties are shared among a large number of children. It is more difficult for parents to fulfil their responsibilities towards their children because the family property has to be shared among a larger number, especially when child survival increases as a result of reduced mortality. The excess children will be tempted to find work away from the family, particularly in the cities, and will tend to marry later. Thus, when both mortality and fertility are very high, parents are afraid of growing old without a son and heir capable of carrying on the family business, as often happens with craftspeople and peasants; when mortality declines sufficiently and fertility remains the same, there is the opposite fear of having too many heirs, which results in fragmentation of the inheritance and dispersal of the family. The Chinese principle of *he* is perfectly compatible with this interpretation of the modernization process.

#### OVERVIEW AND CONCLUSION

Africa's population is now growing at a rate incompatible with its economic development, while, in Asia, countries are steadily making their demographic transition. Almost all of Africa is hesitating on starting its transition and even backing away from it, while almost all of Asia is striding through it.

It cannot be over-emphasized that the population/development link must be read from right to left and left to right, without giving priority to one direction over the other. The third world countries that develop inevitably make the demographic transition and, conversely, those that make the transition develop more easily and are better-placed in international economic competition. From now on, a development strategy that does not include a population policy will be inconceivable in the third world. Orderly development begins at home, with solutions to population problems—problems that only the people of a country can solve.

Describing the population trends in Africa as simply lagging temporarily behind those in other countries of the third world is simplistic and of little use, because it does nothing to explain the trends or advance solutions. It must be clearly understood that change is bound to be slow when cultural factors stand in the way of modernization.

Africa's future depends, first and foremost, on whether African population policies are flexible enough, as Locoh (1988a and 1988b) believes, to adapt fully to the modernization process. For now, the process is having a "perverse" influence, because it is incomplete: the period of breast-feeding is growing shorter, post-partum sexual abstinence is being abandoned and infant mortality is declining, but there has been no fundamental change in marriage patterns or reproductive behaviour. This experience once more confirms that socio-economic and cultural factors are the real driving forces of the transition, as Ansley Coale (1965 and 1971) demonstrated in the case of Europe. It also confirms that each transition depends on unique local socio-economic and cultural factors.

Although the increasing receptiveness of African Governments to population activities, particularly since the Second African Population Conference (Economic Commission for Africa, 1984), gives reason to believe that the slight increase in fertility in Africa is only a passing phenomenon, the gap between Africa and the rest of the world is widening. In the first quarter of the next century, average population densities in Africa will approach those of India today and, in some countries, the situation will be intolerable. Benin, Burundi, Comoros, Gambia, Ghana, Kenya, Lesotho, Madagascar, Malawi, Mauritius, Nigeria, Réunion, Rwanda, Sierra Leone, Swaziland, Togo and Uganda will all have densities higher than 100 inhabitants per square kilometre by 2025, and some countries will have far higher ones. Massive population movements and their attendant political problems can be expected. Expulsions such as that of thousands of Ghanaians from Nigeria in the early 1980s are bound to recur and to become more widespread.

In any case, it would be pointless to think that if one merely urges populations to change their marriage patterns and reproductive behaviour, they will do so. One must also give them the reasons—and the means—to do so, in the form of information and services. These means are still virtually inaccessible in Africa, however. To think that all one will have to do is publicize contraception and provide financial aid is misguided. Direct intervention, however, would be interpreted as a form of, or an outgrowth of, colonialism. One must suggest and encourage very discreetly. Informa-

tion and education must be directed at women, who will in turn instruct their children, and the effects will then be far-reaching.

Africa is in the throes of a profound and unprecedented crisis which it would be wrong to view as primarily economic and financial, because it affects the very foundations of African societies, as Africa's difficulties in achieving a new demographic balance demonstrate. Of course, Africa is made up of local situations, but the absolute food shortages, ecological difficulties (drought, grasshoppers and locusts in the Sahel, resurgence of malaria, re-emergence of the tsetse fly) and a sharp and steady drop in prices for raw materials and agricultural products mean that the entire continent is affected. The magnitude and seriousness of the situation call for a new approach to development. The conflict between modernization and tradition is compounded by demographic factors whose importance it would be irresponsible to deny. And now the continent is afflicted, apparently more than others, with acquired immunodeficiency syndrome. Some take the problem lightly, refusing to see it as a serious threat. Such an eminent demographer as Bongaarts (1988), however, predicts that the pandemic could affect 5 per cent of the population of sub-Saharan Africa by the end of the century, resulting in a considerable increase in mortality. In the most severely affected regions, the mortality rate could double. In such situations, health systems, the already shaky economy and the entire social fabric could be seriously threatened.

We can hope that Africa will be able to enjoy its proper share of wealth in tomorrow's world, in keeping with its size, its population and its natural resources. Countries with such potential are not necessarily doomed to underdevelopment. No one has ever been able to understand and predict how and why the political fortunes of countries rise and fall. What is certain is that the world is becoming a multipolar one in which Africa, with its great wealth, is not without assets.

The Europe of 1992 will have to turn to Africa as part of an ambitious Euro-African project and, in recognition of the interdependence between the two continents, help in the formation of development centres like the many being created in Asia and Latin America.

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#### NOTES

<sup>1</sup> Unless otherwise indicated, the population figures (size, growth, fertility and mortality) used throughout this article are taken from United Nations (1989a).

<sup>2</sup> Hong Kong, the Republic of Korea, Singapore and Taiwan Province (China).

<sup>3</sup> The gross national product (GNP) figures used in this article were calculated on the basis of purchasing power by the Centre d'études projectives et d'informations internationales (CEPII).

<sup>4</sup> It is interesting to note that there was no mention of this prohibition at the seminar on fertility transition in Asia (Bangkok, March 1988), organized by the International Union for the Scientific Study of Population, whereas the Third African Population Conference (Dakar, November 1988) dealt with the issue in several communications.

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## COMPARATIVE ANALYSIS OF COMPLETED PARITY DISTRIBUTIONS: A GLOBAL WFS PERSPECTIVE

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### SUMMARY

This paper studies completed parity distributions for all the industrialized and developing countries that participated in the World Fertility Survey (WFS). This is done by means of a table based on parity calculated for all ever-married women above age 40 by educational and residential sub-categories. The information is also used to compare mean family and mean sibship sizes and to study changes in the concentration of reproduction independent of the level of fertility. The cross-section of countries considered implies that, as fertility declines, the transition from high to low fertility is associated with an increase in the concentration—i.e., a smaller proportion of women having half the children. The big exception is China, where fertility declined steeply without an increase in concentration.

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### INTRODUCTION

Like any other distribution, the distribution of children ever born to a cohort of women may be characterized by its moments. While the first moment—the mean parity or mean family size—has been playing a central role in almost every kind of demographic fertility analysis, the variance or other distributional aspects of fertility have received little attention so far. One reason for this overwhelming dominance of the mean might also lie in the fact that it represents a link between the two major approaches of demographic analysis: cohort analysis and period analysis. The construction of a synthetic cohort, by adding up all age-specific fertility rates observed in a period, results in a mean number of children implied by period rates (the total fertility rate) that is isomorphic to the first moment of the completed parity distribution of a cohort. Hence, conventional period fertility analysis can give us the mean family size of a synthetic cohort but not the family size distribution itself. The estimation of period-completed parity distribution requires more sophisticated models based on

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parity-specific fertility rates (Feeney, 1983; Chiang, 1984; Lutz and Feichtinger, 1985).

Yet for many purposes the mean family size will suffice as an indicator of fertility. For a population projection that only attempts to forecast the sizes of age groups in a certain year in the future, all that matters is the number of persons in a cohort, which depends only on the average birth intensities and not on other aspects of the distribution of children over women. But already, when trying to discuss the plausibility of certain assumptions of average fertility, we have to go back to the level of individual behaviour, and there couples do not have average family sizes but a certain discrete number of children. At that level of analysis every change in the average level of fertility has to be understood as the sum of individual behaviour, a the result of many more or less conscious decision-making processes for a certain family size. There is no reason to assume that a change in the mean level would leave the shape of the distribution unchanged. On the contrary, the subsequent analysis will show that there are strong and characteristic changes in the shape of the distribution associated with observed trends in the average family size.

Historically, a lack of information on parity distributions was probably the other major reason for the disregard of distributional aspects in fertility analysis. Even the change from natural to controlled fertility, which is by definition a parity-specific phenomenon, had to be studied indirectly through the comparison of shapes of age-specific marital fertility schedules.<sup>1</sup> The success of this method is largely based on the strong positive correlation between age and parity. Systematic attempts to estimate the extent of fertility control directly from cohort parity distributions are much rarer.<sup>2</sup>

The World Fertility Survey provides very rich and reasonably reliable source material for parity-specific fertility analysis. And yet among the hundreds of WFS-based publications, very few studies have been made in this field<sup>3</sup> and none has tried to combine the results from high- and low-fertility countries. The analysis in this paper is based on WFS standard recode files for 41 developing countries<sup>4</sup> (compiled by WFS headquarters in London), which were combined with the WFS files for 14 European countries and the United States of America (centrally collected by the Economic Commission for Europe in Geneva).<sup>5</sup> This paper focuses on completed cohort distributions of ever-married women only. Since the highest age considered was 49 in most countries (only 45 in most European countries), the analysts had to consider all women above age 40 as having essentially completed their reproductive careers, in order to have sufficiently large samples. The restriction to ever-married women had become necessary for consistency, because in a large number of countries only ever-married women had been interviewed. The only two socio-economic background variables considered here are place of residence (urban/rural) and education (low/high), according to standard WFS conventions.

The descriptive tool used in this study to analyse the empirically observed completed parity distributions of cohorts and the underlying set



of parity progression ratios is the deterministic trunk model of the fertility table based on parity, introduced by Chiang and van den Berg (1982) and modified by Feichtinger and Lutz (1983).<sup>6</sup> That model, which was originally designed for period analysis to estimate the completed parity distribution implied by observed period parity-specific fertility rates and mean ages at birth, also turned out to be a powerful tool for the purely descriptive analysis of completed cohort fertility. This is especially true for a detailed analysis of the timing of parity progression when conditional mean ages at birth of given orders are considered.<sup>7</sup> But also in the context of this comparative study, concerned with the quantum aspect of reproduction only, the basic functions of the fertility table based on parity seem to be a useful descriptive tool.

The fertility table is essentially built up in analogy to an ordinary (mortality) life table where parity replaces age as the indexing variable. The parity progression ratios then correspond to the survival probabilities; their complements—the probabilities of death in the ordinary life table—give the probabilities of dropping out of the process of parity progression at a certain parity. Starting with a radix,  $l(0)$ , of 1,000 women entering the reproductive age, the  $l(i)$  column then gives the number of women who “survived” to parity  $i$ , or, in other words, who made it up to  $i$  or more children. Hence, as in a regular life table,  $l(i)$  is defined by

$$l(i) = l(i - 1) p(i - 1)$$

where  $p(i)$  is the parity progression ratio at parity  $i$ . The column of life table deaths,  $d(i)$ , then gives the number of women who drop out of the process of parity progression at that parity and hence remain at parity  $i$ . Again in analogy to the regular life table,  $d(i)$  is defined as

$$d(i) = l(i) (1 - p(i)) \quad \text{or} \quad d(i) = l(i) - l(i + 1).$$

Empirically this descriptive form of the fertility table pertaining to a cohort is entered through the  $d(i)$  column, which corresponds directly to the observed completed parity distribution (multiplied by the radix). Once the  $d(i)$  column is given, the  $l(i)$  and  $p(i)$  columns can be derived by simple algebraic transformations according to the definitions given above.

Table 1 gives an example of the cohort fertility table for Kenya. In addition to the  $p(i)$ ,  $l(i)$  and  $d(i)$  columns discussed above, it is also possible to calculate the mean number of children born beyond parity  $i$ ,  $F(i)$ , directly from the given data by

$$F(i) = \sum_{x=i+1}^m f(x)$$

where

$$f(i) = l(i)/l(0),$$

$m$  being the highest parity considered. The quantity  $f(i)$  gives the number of births of order  $i$  per woman. It is equivalent to what Ryder (1982) calls the total fertility rate for births of order  $i$ . Clearly the summation over all

TABLE 1. DESCRIPTIVE PARITY TABLE: KENYA,  
EVER-MARRIED WOMEN AGED 40-49

Parity	Parity progression ratio $p(i)$	Number of women, out of 1,000, reaching parity $i$ $l(i)$	Number of women, out of 1,000, remaining at parity $i$ $d(i)$	Total fertility rate for parity $(i)$ and above $F(i)$
0 .....	.9684	1 000	31	7.73
1 .....	.9751	968	24	6.77
2 .....	.9753	944	23	5.83
3 .....	.9657	920	31	4.91
4 .....	.9628	889	33	4.02
5 .....	.9341	856	56	3.16
6 .....	.8937	799	85	2.36
7 .....	.8347	714	118	1.65
8 .....	.7541	596	146	1.05
9 .....	.6555	449	155	.60
10 .....	.5510	294	132	.31
11 .....	.5185	162	78	.14
12 .....	.4375	84	47	.06
13 .....	.3878	36	22	.02
14 .....	.3158	14	9	.01
15+ .....		4	4	

Source: World Fertility Survey, standard recode tapes.

$f(i)$ s which is equal to  $F(0)$  is the mean number of children borne by the cohort (considering births of all parities).<sup>8</sup> Every mention of mean parity or mean family size of a cohort in the following sections will refer to this quantity  $F(0)$ .

#### COMPARATIVE DESCRIPTION OF COMPLETED PARITY DISTRIBUTIONS

Table 2 gives the observed distributions of children ever born to ever-married women aged 40-49 in all the 54 developed and developing WFS countries with standard recode files available. Since most WFS surveys took place around the middle of the 1970s and since at that point the observed cohorts of women were already past their prime child-bearing ages, the given parity distributions do not reflect very recent fertility patterns but rather those of (on average) from two to three decades ago. Table 2 presents the completed parity distributions that were used as input data to the analysis.

From table 2 itself differential shapes of the distribution do not become very clear at first sight. What can be seen is that in Cameroon the proportion of childless women is exceptionally high, with over 15 per cent; the proportion is lowest in the Republic of Korea, with 1.5 per cent. The mode of the completed parity distribution for high-fertility countries ranges mostly between six and nine children, whereas in all European

countries it lies at parity 2. Some countries, such as Benin and Jamaica, show bi-modal distributions. In Paraguay and Nigeria, the modes are exceptionally low (at parities 3 and 4, respectively) by the standards of high-fertility countries. Costa Rica has the highest proportion of women with 15 or more births (3.4 per cent), followed by Jordan (2.4 per cent) and Mexico (2.1 per cent).

The mean family size of the cohort considered ( $F(0)$ ) is also given in table 2. It is highest in Jordan (8.66), followed by Kenya (7.73) and the Syrian Arab Republic (7.66), and lowest in Czechoslovakia (2.34), leaving out the Netherlands (1.67), because the data for the Netherlands are not strictly compatible due to the selection of cohorts in the study.<sup>9</sup> In the more developed countries, average family sizes are highest in the United States of America (3.48) and Spain (3.10). These values seem rather high by current period total fertility standards, because they refer to ever-married women only and the cohorts covered are mostly those that had their prime child-bearing during the period of the post-war baby boom. From table 2 we also see clearly that the ranking according to mean family size is not identical to the rankings according to the mode or the proportion with 15 or more children or any other single parity category. To shed more light on the relationship between the shape of the distribution and the average level we have to consider further aspects.

Of the functions introduced in table 1, the parity progression ratios,  $p(i)$ , seem to show the most irregularity and sensitivity between populations and within populations. They represent the behavioural component in the fertility table. Because of this behavioural determination of the transitions to higher parity categories, the pattern of parity progression ratios is hard to describe by any standard function. The  $f(i)$  and  $l(i)$  functions must, by definition, decrease monotonically, and the integral under the  $d(i)$  function must equal  $l(0)$ ; but for the parity progression ratios there is no restriction other than that the values must lie between 0 and 1. Unlike its counterpart in mortality analysis—the force of mortality function—the  $p(i)$  function is very hard to describe by a parameterized model, because its shape is biologically determined only in the case of completely natural fertility.

Keeping the great potential for irregularities in the parity progression ratios in mind, it is surprising to see how regular they turn out to be in developing countries: the cohorts of ever-married women with completed parity show almost monotonically declining parity progression ratios from a maximum at parity 0 to a minimum at the highest parity. There are a few exceptions. In some countries (e.g., in Cameroon) the parity progression ratios at parity 0 are smaller than those at parity 1, and in a relatively large number of countries the ratios level off or even increase at high parities. This may be partly due to irregularities because of small numbers in those categories, but a comparison with industrialized countries, where the phenomenon is much stronger, suggests that it may be a real effect due to heterogeneity in the population: there is one small group of women with extremely high fertility which, beyond a certain parity, dominates the picture.

TABLE 2. OBSERVED DISTRIBUTION OF CHILDREN EVER BORN  
TO EVER-MARRIED WOMEN AGED 40-49

Country	Total fertility rate F(0)						Completed
		0	1	2	3	4	5
<b>Africa</b>							
Benin .....	6.14	32	25	62	70	105	89
Cameroon .....	4.91	154	96	71	76	62	71
Côte d'Ivoire .....	6.78	40	51	42	43	62	76
Egypt .....	6.64	38	32	45	48	72	104
Ghana .....	6.38	23	19	51	66	94	99
Kenya .....	7.73	31	24	23	31	33	56
Lesotho .....	5.22	52	71	94	99	103	101
Mauritania .....	6.01	47	48	70	83	89	102
Morocco .....	7.08	69	37	39	40	47	57
Nigeria .....	5.41	73	53	73	94	114	107
Senegal .....	6.92	35	40	43	46	71	68
Sudan .....	6.00	75	50	67	66	69	76
Tunisia .....	6.78	36	21	29	48	71	106
<b>Americas</b>							
Colombia .....	6.90	28	38	55	86	84	91
Costa Rica .....	6.92	28	35	71	105	89	72
Dominican Republic ..	6.39	49	60	58	78	109	80
Ecuador .....	6.98	18	26	58	74	94	97
Guyana .....	6.46	54	64	46	73	65	84
Haiti .....	5.80	34	61	61	87	101	110
Jamaica .....	5.51	73	86	91	73	106	82
Mexico .....	7.03	37	37	55	61	76	82
Panama .....	5.74	33	41	91	106	102	134
Paraguay .....	6.27	29	74	122	208	95	107
Peru .....	6.76	22	35	51	82	86	92
Trinidad and Tobago ..	5.50	58	66	78	104	110	97
<b>United States of</b>							
America .....	3.48	57	72	228	233	152	115
Venezuela .....	6.21	20	43	102	84	99	104
<b>Asia and Pacific</b>							
Bangladesh .....	6.95	25	27	40	55	49	87
Fiji .....	6.31	51	39	55	69	69	100
Indonesia .....	5.25	66	84	83	91	94	98
Jordan .....	8.66	23	9	21	27	43	42
Malaysia .....	6.13	29	50	68	88	95	103
Nepal .....	5.65	39	36	76	73	127	123
Pakistan .....	6.91	36	28	35	50	57	87
Philippines .....	6.86	24	31	43	66	81	87
Republic of Korea .....	5.42	15	38	48	77	147	187
Sri Lanka .....	5.73	36	52	76	89	98	104
Syrian Arab Republic ..	7.66	36	13	23	41	44	72
Thailand .....	6.36	25	40	56	69	99	101
Turkey .....	6.08	27	17	76	102	117	129
Yemen .....	6.65	21	23	38	71	95	109
<b>Europe</b>							
Belgium .....	2.56	74	212	291	195	106	52
Czechoslovakia .....	2.31	39	165	451	212	85	30
Denmark .....	2.53	61	116	370	242	147	32

parity distribution (per 1,000 women)

6	7	8	9	10	11	12	13	14	15
120	143	141	106	51	38	9	3		
89	101	92	78	43	30	17	6	4	1
96	107	135	146	82	71	22	10	6	2
118	131	118	103	91	38	31	11	7	5
134	138	135	110	77	32	5	9	0	
85	118	146	155	132	78	47	22	9	4
118	114	97	73	40	15	12	5		
100	125	76	106	54	42	36	10	4	
95	97	124	119	108	63	57	20	11	9
108	101	88	74	54	27	20	14		
85	117	139	128	95	88	35	6		
96	118	130	87	89	44	21	5		
126	133	123	133	72	47	30	9	7	0
83	94	93	77	80	70	39	29	26	18
88	76	80	72	68	54	58	42	20	34
102	58	84	73	64	75	58	37	8	
96	96	97	96	72	57	50	32	14	18
101	98	103	93	77	47	47	22	12	7
114	108	110	97	62	34	11	3		
74	101	97	60	60	39	34	12	10	4
91	97	94	107	75	61	46	37	16	21
105	95	83	55	59	43	31	10	4	
86	56	71	44	35	32	26	8		
104	107	98	97	71	60	47	19	10	11
117	90	85	50	48	36	29	20	3	
54	36	21	15	12	2	2	1		
93	99	67	78	81	61	34	17	11	
112	153	128	127	87	51	30	10	7	4
111	124	114	94	77	45	21	8	7	8
114	105	97	64	44	32	17	3	1	
74	90	87	141	136	111	73	61	32	24
103	109	101	84	70	47	28	8	5	5
119	131	107	87	42	21	10	3		
97	149	141	117	95	47	32	13	5	3
111	107	112	119	90	55	34	20	6	6
167	149	95	39	20	9	3	1		
140	117	90	78	50	35	14	8	3	1
108	104	139	114	106	83	54	26	16	12
116	118	106	100	76	36	27	11	5	5
100	112	81	82	56	40	25	12	10	5
109	128	128	100	66	59	28	16	2	
38	18	4	5	3	1	3			
13	2	4							
19	8	4							

TABLE 2. (continued)

Country	Total fertility rate F(0)	Completed					
		0	1	2	3	4	5
<i>Europe (continued)</i>							
Finland .....	2.67	48	145	336	244	118	60
France .....	2.69	60	158	308	228	122	54
Italy .....	2.38	58	170	387	228	94	36
Netherlands <sup>a</sup> .....	1.67	171	289	303	184	39	13
Norway .....	2.75	48	100	299	281	175	77
Poland .....	2.78	31	140	357	223	125	58
Portugal.....	2.01	58	182	312	161	102	56
Spain.....	3.10	40	80	295	254	166	76
United Kingdom .....	2.58	60	139	354	217	136	49
Yugoslavia .....	3.05	43	121	351	183	110	56

Source: World Fertility Survey, standard recode tapes.

NOTE: Because of rounding, the sum of the parity distribution may not in all cases equal 1,000.

Figure I shows a sample of different shapes of the parity progression functions in five countries with different fertility levels. Jordan has the highest cohort fertility among all WFS countries. There, parity progression ratios stay above 0.96 until parity 5. We can see some increase from parity 0 to 1 in Cameroon. In other words, in Cameroon, the probability of a birth is higher for women who already had one birth than for women who are still childless, thus proving their fecundity. This observation can be made mainly in eastern and central Africa and is probably due to the high incidence of infecundity resulting from venereal disease and malnutrition. Between parities 4 and 9 the parity progression ratios in Cameroon decrease at an accelerating speed. After parity 9, the pattern is more irregular but generally declining.

The pattern for the Republic of Korea is quite different: a slow and almost linear decline between parities 0 and 3, followed by a steeper but also linear decline for parities 4-8 and 9-12, excepting a slight increase between parities 8 and 9. Typical patterns of parity progression ratios in low-fertility industrialized countries are shown by Portugal and Czechoslovakia. The pattern is characterized by a steep decline in parity progression ratios until parity 2, after which the curve levels off or even increases. An increase in parity progression ratios at higher parities can be observed in many low-fertility countries and is due to selectivity of a few high-fertility women.

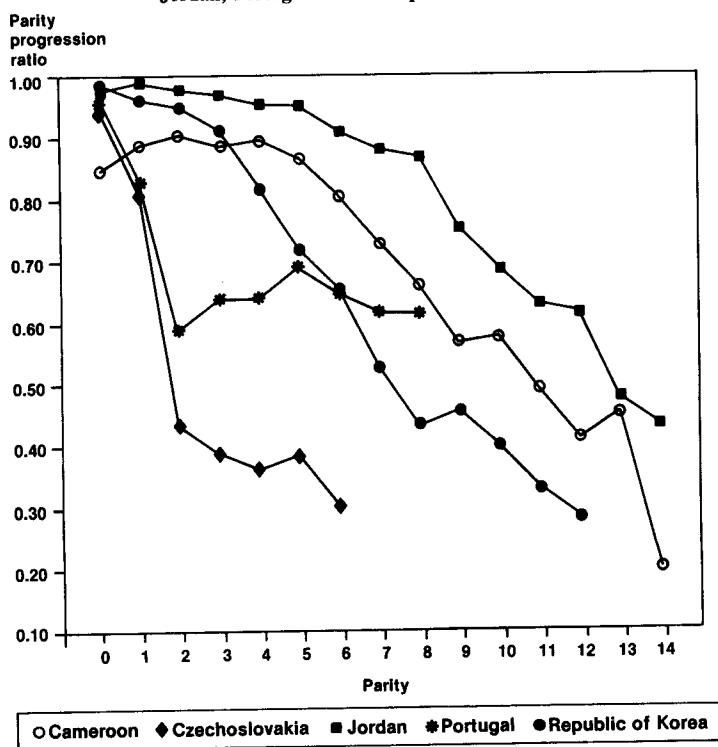
What is the reason for this dramatic shift in the pattern of parity progression ratios from high-fertility countries to low-fertility countries? Theoretically, a decline in fertility can happen in many different ways, ranging from a proportional decline at each parity of the typical developing country curve to a step-wise function, with high progression ratios up to a certain threshold parity and low ratios thereafter. The observed pattern of change becomes plausible when we think in terms of the paradigm

parity distribution (per 1,000 women)

	6	7	8	9	10	11	12	13	14	15
22	13	5	5	3	1					
36	23	3	3	5						
10	7	4	5							
20										
33	14	7	8	1	1	1	1			
44	31	19	9	8	7	3	2			
42	22	10	6	3	3	1				
28	13	4								
52	40	21	22							

<sup>a</sup>In this context the data for the Netherlands are not strictly comparable to those for other countries, because they are restricted to the marriage cohorts 1963-1973.

Figure I. Parity progression ratios in Cameroon, Czechoslovakia, Jordan, Portugal and the Republic of Korea



Source: World Fertility Survey, standard recode tapes.

of natural versus controlled fertility. In a natural-fertility population (which we may assume for Kenya), women do not deliberately control their fertility in dependence on the number of children already born. Under such a fertility régime the pattern of parity progression ratios depends only on the change in fecundability and an increased prevalence of sterility with age and parity. Those biological factors result in a monotonous decline which tend to be steeper after a certain threshold. In a controlled-fertility situation, however, couples tend to follow their fertility intentions, and parity progression ratios will be relatively high up to the mode of the desired family size distribution and lower thereafter.

A *prima facie* study suggested that in a great number of countries a level of 0.8 in the parity progression ratio might be considered a threshold, because the pace of decline increases after that level is achieved. Table 3 ranks total and urban female populations aged 40-49 according to the parity at which their parity progression ratios fall below 0.8. We can see that the total female population is distributed into two groups: the

TABLE 3. COUNTRIES LISTED ACCORDING TO THE PARITY AT WHICH EVER-MARRIED WOMEN AGED 40-49 REACH PARITY PROGRESSION RATIOS BELOW 0.8

<i>Parity</i>	
<i>Total ever-married women</i>	
1 .....	Belgium, Netherlands
2 .....	Czechoslovakia, Denmark, Finland, France, Italy, Norway, Poland, Portugal, Spain, United Kingdom, United States, Yugoslavia
5 .....	Panama, Republic of Korea
6 .....	Ghana, Haiti, Indonesia, Lesotho, Nepal, Nigeria, Sri Lanka, Trinidad and Tobago
7 .....	Bangladesh, Benin, Cameroon, Egypt, Fiji, Jamaica, Malaysia, Mauritania, Pakistan, Peru, Sudan, Thailand, Tunisia, Turkey, Venezuela, Yemen
8 .....	Colombia, Côte d'Ivoire, Dominican Republic, Ecuador, Guyana, Kenya, Mexico, Morocco, Paraguay, Philippines, Senegal, Syrian Arab Republic
9 .....	Costa Rica, Jordan
<i>Urban ever-married women</i>	
1 .....	Belgium, Czechoslovakia, Netherlands, Portugal
2 .....	Denmark, Finland, France, Italy, Norway, Poland, Spain, Yugoslavia
3 .....	Paraguay
4 .....	Dominican Republic, Haiti, Jamaica, Republic of Korea, Thailand, Turkey
5 .....	Fiji, Panama
6 .....	Benin, Cameroon, Costa Rica, Egypt, Ghana, Malaysia, Sri Lanka
7 .....	Côte d'Ivoire, Ecuador, Guyana, Indonesia, Mauritania, Mexico, Morocco, Pakistan, Peru, Philippines, Senegal, Sudan, Trinidad and Tobago, Tunisia
8 .....	Colombia, Syrian Arab Republic

Source: World Fertility Survey, standard recode tapes.



low-fertility countries are heavily concentrated at parity 2, whereas the high-fertility countries range between 5 and 9, with a heavy concentration at parities 7 and 8. For urban women the distribution is more even over all parities. This is mostly due to the high socio-economic differentials in Latin American countries, where the ratio for urban women falls below 0.8 already at parities 3, 4 and 5.

A comparison between the list in table 3 and the distribution of average completed family sizes,  $F(0)$ , reveals that a later decline in parity progression ratios does not necessarily mean a higher mean number of children. The reason is that average completed parity also depends on the shape of the curve of parity progression ratios before and after our chosen value of 0.8. In Costa Rica, for instance, the parity progression ratio remains higher than 0.8 until parity 9, but many other countries with a lower threshold have higher fertility levels. Generally, however, for developing countries the empirical correspondence between the ranking in table 3 and average completed fertility is quite good, because the shapes of the progression ratio curves are similar. For the developed countries the ranking according to the critical point of 0.8 is less informative. The reason for this is the fast decline at low parities to levels below 0.8 and the high variance at higher parities.

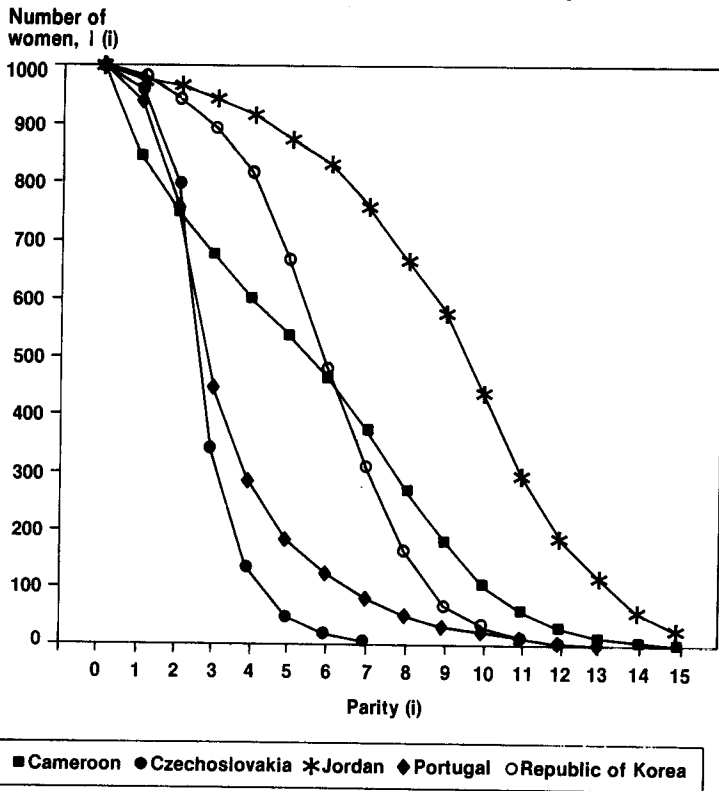
The  $l(i)$  column in table 1 gives the number of women out of a cohort of 1,000 who are still in the process of parity progression at parity  $i$ . The curve of  $l(i)$  declines by definition from 1,000 to 0 for every country. Differentials in the fertility level can be seen from the extent to which the curve is convex or concave.

Figure II plots the  $l(i)$  function for five countries with different levels of fertility. The curve for Jordan lies to the far right of the other curves and is clearly convex. It somehow resembles the familiar pattern of a convex curve of age-specific marital fertility rates in natural fertility countries. This is not surprising, because age and parity are strongly correlated in a population without family limitation. To the far left lie the convex curves for Portugal and Czechoslovakia, showing typical patterns of modern low-fertility countries. The other curves lie between those two extremes. Cameroon exhibits an almost straight decline with parity whereas the Republic of Korea follows the more general S-curve pattern that is typical of the majority of developing countries.

Tables 4 and 5 give global overviews on inter- and intra-country differentials in the  $l(x)$  function.<sup>10</sup> Table 4 shows what proportion of the initial 1,000 women had a third birth during their lifetimes. On the national level the values range from 95 per cent in Jordan to about 35 per cent in Czechoslovakia.<sup>11</sup> The range is even wider for residential or educational sub-populations.

Among urban European women it was only in Norway and Spain that more than 50 per cent of the cohort had a third child. In rural areas, however, two thirds of the European countries show proportions of over 50 per cent. Similar differentials appear with respect to education, although among better-educated women in Belgium, the Netherlands and Spain, a higher proportion had third children as compared to less-educated women.

Figure II. Number of women,  $l(i)$  out of 1,000, reaching parity  $i$  in Cameroon, Czechoslovakia, Jordan, Portugal and the Republic of Korea



Source: World Fertility Survey, standard recode tapes.

The relative extent of educational and residential differentials in Europe is quite irregular. Usually the residential differentials were greater. In four countries, most prominently in Yugoslavia, educational differentials were stronger.

Among the developing countries socio-economic differentials are highest in Latin America. In several African countries and in Bangladesh and Indonesia (both Islamic), educated women had higher probabilities of having a third child. With respect to place of residence, all countries (except for the Sudan) show higher proportions of rural women with third children than urban women. Among the 40 developing countries studied here, there are only five countries—namely, Cameroon, Indonesia, Jamaica, Lesotho, and Trinidad and Tobago—where less than 80 per cent of the women had third children. In Cameroon the percentage is only 68 per cent, which comes rather close to the highest European values. The reason lies in the relatively high proportion of childless and low-parity women in Cameroon.

TABLE 4. NUMBER OF WOMEN, OUT OF 1,000, WHO HAD REACHED AT LEAST PARITY 3, ACCORDING TO PLACE OF RESIDENCE AND LEVEL OF EDUCATION

	Total	Rural	Urban	Absolute difference	Relative difference (percentage)	Education		Absolute difference	Relative difference (percentage)
						Low	High		
Africa									
Benin	880	889	848	41	95	..	..	..	..
Cameroon	676	693	588	105	84	..	..	..	..
Côte d'Ivoire	866	869	853	16	98	..	..	..	95
Egypt	884	891	875	16	98	899	860	39	96
Ghana	906	922	859	63	93	910	958	-49	105
Kenya	922	..	..	..	..	758	794	-36	104
Lesotho	782	..	..	..	..	830	837	-7	100
Mauritania	834	853	803	50	94	..	..	..	..
Morocco	854	881	806	75	91	..	..	..	..
Nigeria	801	..	..	..	..	..	..	..	..
Senegal	882	895	847	48	94	..	..	..	..
Sudan	807	792	845	-54	106	..	..	..	..
Tunisia	913	923	903	19	97	..	..	..	..
Americas									
Colombia	878	901	863	38	95	900	833	68	92
Costa Rica	865	928	802	127	86	918	814	105	88
Dominican Republic	832	906	768	138	84	847	804	43	94
Ecuador	897	941	845	96	89	951	836	115	87
Guyana	835	877	755	122	86	904	820	84	90
Haiti	843	877	746	131	85	..	..	..	..
Jamaica	748	784	682	101	87	..	..	..	..
Mexico	870	925	831	95	89	903	852	51	94
Panama	834	908	782	125	86	899	797	102	88
Paraguay	826	859	782	77	91	870	773	97	88
Peru	891	919	873	46	94	931	858	73	92
Trinidad and Tobago	796	811	786	25	96	886	776	110	87
United States of America	644	..	..	..	..	667	634	33	95
Venezuela	834	..	..	..	..	885	784	102	88

TABLE 4 (continued)

	Total	Rural	Urban	Absolute difference	Relative difference (percentage)	Education		Absolute difference	Relative difference (percentage)
						Low	High		
Asia and Pacific									
Bangladesh.....	907	868	826	..	..	904	913	-9	101
Fiji.....	854	767	755	42	95	879	837	42	95
Indonesia.....	765	767	755	12	98	752	814	-59	107
Jordan.....	947	858	836	..	..	952	930	21	97
Malaysia.....	852	858	836	22	97	869	822	47	94
Nepal.....	847	909	871	38	95	..	..	..	..
Pakistan.....	900	911	877	34	96	927	889	38	95
Philippines.....	901	930	869	61	93	913	885	28	96
Republic of Korea.....	898	845	780	65	92	863	821	42	95
Sri Lanka.....	835	934	921	12	98	932	894	38	95
Syrian Arab Republic.....	927	888	813	75	91	879	874	5	99
Thailand.....	878	941	802	139	85	937	779	159	83
Turkey.....	879	941	802	139	85	937	779	159	83
Yemen.....	916	..	..	..	..	..	..	..	..
Europe									
Belgium.....	423	434	421	13	97	416	425	-9	102
Czechoslovakia.....	348	471	297	174	63	487	292	195	59
Denmark.....	453	656	391	265	59	497	366	131	73
Finland.....	471	573	387	186	67	537	389	148	72
France.....	474	564	435	129	77	500	428	72	85
Italy.....	385	403	362	41	89	418	287	131	68
Netherlands.....	237	429	164	265	38	231	240	-9	103
Norway.....	556	594	505	89	85	656	519	137	79
Poland.....	472	678	338	340	49	594	276	318	46
Portugal.....	447	519	295	224	57	549	315	234	57
Spain.....	585	626	578	48	92	583	614	-31	105
United Kingdom of Great Britain and Northern Ireland.....	448	..	..	..	..	462	444	18	96
Yugoslavia.....	484	579	399	180	68	515	148	367	28

Source: World Fertility Survey, standard recode tapes.

TABLE 5. NUMBER OF WOMEN, OUT OF 1,000, WHO HAD REACHED AT LEAST PARITY 7, ACCORDING TO PLACE OF RESIDENCE AND LEVEL OF EDUCATION

	Total	Rural	Urban	Absolute difference	Relative difference (percentage)	Education		Absolute difference	Relative difference (percentage)
						Low	High		
Africa									
Benin .....	494	506	453	53	89	..	..	..	..
Cameroon .....	375	401	238	163	59	..	..	..	..
Côte d'Ivoire .....	586	593	556	36	93	..	..	76	86
Egypt .....	539	604	459	145	75	568	492	80	84
Ghana .....	510	524	469	55	89	523	443	-44	106
Kenya .....	715	..	..	..	..	704	748	-47	114
Lesotho .....	358	..	..	..	102	328	374	-61	114
Mauritania .....	457	453	462	-9	81	422	484	..	..
Morocco .....	613	649	527	122	81	..	..	..	..
Nigeria .....	378	..	..	..	..	..	..	..	..
Senegal .....	610	625	575	50	91	..	..	..	..
Sudan .....	497	500	487	13	97	..	..	..	..
Tunisia .....	559	586	529	57	90	..	..	..	..
Americas									
Colombia .....	531	599	489	110	81	585	419	166	71
Costa Rica .....	508	682	335	347	49	660	361	299	54
Dominican Republic .....	460	626	316	311	50	538	306	233	56
Ecuador .....	535	633	411	222	64	658	388	270	58
Guyana .....	510	577	377	200	65	601	489	111	81
Haiti .....	428	471	289	182	61	..	..	..	..
Jamaica .....	410	497	252	245	50	..	..	..	..
Mexico .....	558	684	217	217	68	650	507	143	77
Panama .....	383	556	261	295	46	570	281	289	49
Paraguay .....	460	619	258	360	41	615	274	341	44
Peru .....	524	651	437	214	67	680	393	287	57
Trinidad and Tobago .....	364	434	319	115	73	479	340	139	71
Venezuela .....	451	..	..	..	..	596	312	284	52

TABLE 5 (continued)

	Total	Rural	Urban	Absolute difference	Relative difference (percentage)	Education		Absolute difference	Relative difference (percentage)
						Low	High		
Asia and Pacific									
Bangladesh	602	..	..	..	..	593	652	-58	109
Fiji	502	560	391	169	69	549	468	81	85
Indonesia	366	361	389	-27	107	358	398	-40	111
Jordan	760	..	..	..	..	826	569	256	68
Malaysia	460	496	379	117	76	490	405	85	82
Nepal	404	..	..	..	..	..	..	..	..
Pakistan	607	610	590	21	96	..	..	..	..
Philippines	553	611	427	183	69	694	497	196	71
Republic of Korea	318	450	193	257	42	437	224	213	51
Sri Lanka	400	423	296	126	70	495	356	139	71
Syrian Arab Republic	660	692	629	63	90	700	439	261	62
Thailand	489	527	284	243	53	510	473	37	92
Turkey	427	554	260	295	46	556	202	354	36
Yemen	530	..	..	..	..	..	..	..	..

Source: World Fertility Survey, standard recode tapes.

Table 5 gives comparable figures for the proportions of women that had a seventh child. Since the proportions are extremely low in low-fertility countries, those countries were not included in the table. Within the developing countries, national levels range from a high of 76 per cent in Jordan to lows of 32 per cent in the Republic of Korea, and 36-37 per cent in Cameroon, Indonesia, Lesotho and Nigeria.

Differentials in the proportions of women with a seventh birth with respect to place of residence and women's education are very substantial in most countries and greater than the differentials with respect to third births. In all cases except for Bangladesh, Indonesia, Kenya, Lesotho and Mauritania, differentials go in the expected directions, with higher fertility in rural areas and for women without schooling. In the five countries that are the exceptions, the differentials are lower. Without going into a more detailed country-specific analysis, one might assume that in those countries the reproductive behaviour of educated and urban women was still traditional, and higher socio-economic status resulted in higher fecundability, probably combined with less breast-feeding. Another reason for the unexpected fertility differentials might also be differential quality of reporting, with educated and urban women giving more complete birth histories. After all, these figures refer to women who had been through their prime child-bearing ages about 20 years before the survey. If a similar bias were also assumed for all other countries, it would mean even higher socio-economic differentials than those observed in the survey.

Comparing continents, we observe that, generally, socio-economic differentials in respect to the frequency of a seventh child are highest in Latin America. Absolute and relative differences are highest in Costa Rica, the Dominican Republic, Panama and Paraguay for both place of residence and mother's education. The rural figures tend to be twice the urban in many cases. The relative importance of the residential and educational differentials varies from Central America and the Caribbean, where urban/rural differentials tend to be higher, to South America, where women's education seems to be more important. The Asian countries tend to take an intermediate position between South America and Africa. Exceptions are the Republic of Korea, Thailand and Turkey, which stand out with very high differentials. Concerning the relative importance of the differentials, the pattern is very irregular in Asia. In the Syrian Arab Republic, for instance, the difference in respect to schooling is four times the residential difference, whereas in Thailand the urban/rural differential is many times higher than the educational one. In Africa differentials tend to be moderate.

#### MEAN FAMILY SIZES AND CONCENTRATION OF THE DISTRIBUTION

It has been mentioned above that, for many purposes, the mean number of children, even without information on the full distribution, provides sufficient information. But when talking about the impact of mean family sizes, we must be aware of the fact that there are two different means, both of which have significance: the mean from the mothers' per-

spective (mean parity) and the mean from the children's perspective (mean sibship size). While the first is the usual arithmetic mean of the distribution, the second is called the contra-harmonic mean. Intuitively, the difference becomes clear when we think of asking a population of children for their family size: there will be no child who can say that its mother had 0 children, whereas childlessness is possible in the population of women. A family with, for example, eight children will have eight times as many children in the children's population, who will say that they have a sibship size of eight, as a family with just one child, who will state it has sibship size one—although for the calculation of mean parities, both families get equal weight. From this we can already see that the mean sibship size is always greater than the mean parity. Only in the hypothetical case in which all women had exactly the same number of children would the two means be identical.

Formally the relationship between the two means can be described in the following manner (Preston, 1976): let  $f(x)$  be the proportion of women with completed parity  $x$ . Then the mean parity is

$$\bar{x} = \sum_1^m f(x)x$$

where  $m$  is the maximum parity considered. The mean sibship size is then

$$\bar{c} = \frac{\sum_1^m \frac{f(x)x}{\sum_1^m f(x)x}}{\sum_1^m f(x)x} = \frac{\sum_1^m f(x)x^2}{\bar{x}}$$

where the weight in the summation represents the proportion of children from families of size  $x$ .

It can be shown (Preston, 1976) that the difference between women's mean family size ( $\bar{x}$ ) and children's mean family size ( $\bar{c}$ ) is a function of the second moment of the distribution:

$$\bar{c} = \frac{\sigma_x^2}{\bar{x}} + \bar{x}$$

where  $\sigma_x^2$  is the variance of the distribution of family sizes among women.<sup>12</sup>

This difference between the mean family sizes for women and children has several demographic and non-demographic consequences, which will be considered in the discussion session below. Here, the difference between the two means is taken as one possible indicator of the unevenness in the completed parity distribution.

Table 6 gives the mean parities and mean sibship sizes for all WFS countries. We see that a ranking according to mean sibship size turns out to be quite different from one according to mean parity, although Jordan has the highest values for both means. Kenya, which has the second



TABLE 6. MEAN FAMILY SIZES AND CONCENTRATION INDICES  
FOR ALL WFS COUNTRIES

	Mean parity (1)	Mean sibship size (2)	Absolute difference (3) = (2) - 1	Relative difference (4) = (1)/(2)	0.5 fractile (5)	Index of dissimilarity (6)	Gini coefficient (7)
<b>Africa</b>							
Benin .....	6.10	7.41	1.31	0.82	0.335	0.187	0.257
Cameroon .....	4.86	7.53	2.67	0.65	0.256	0.317	0.414
Côte d'Ivoire .....	6.72	8.32	1.60	0.81	0.332	0.196	0.266
Egypt .....	6.59	8.12	1.53	0.81	0.330	0.190	0.264
Ghana .....	6.33	7.54	1.21	0.84	0.342	0.175	0.240
Kenya .....	7.69	8.96	1.27	0.86	0.362	0.155	0.218
Lesotho .....	5.19	6.91	1.73	0.75	0.297	0.239	0.323
Mauritania .....	5.96	7.78	1.82	0.77	0.303	0.224	0.308
Morocco .....	7.03	8.92	1.89	0.79	0.324	0.206	0.285
Nigeria .....	5.46	7.34	1.89	0.74	0.291	0.244	0.335
Senegal .....	6.90	8.37	1.47	0.82	0.339	0.187	0.257
Sudan .....	5.95	7.83	1.88	0.76	0.309	0.232	0.313
Tunisia .....	6.72	8.06	1.35	0.83	0.342	0.175	0.242
<b>Americas</b>							
Colombia .....	6.84	8.86	2.02	0.77	0.303	0.222	0.301
Costa Rica .....	6.86	9.14	2.27	0.75	0.290	0.239	0.321
Dominican							
Republic .....	6.35	8.54	2.19	0.74	0.289	0.247	0.330
Ecuador .....	6.94	8.71	1.77	0.80	0.314	0.206	0.283
Guyana .....	6.42	8.48	2.06	0.76	0.301	0.232	0.317
Haiti .....	5.76	7.32	1.57	0.79	0.312	0.215	0.291
Jamaica .....	4.65	7.27	2.61	0.64	0.249	0.284	0.356
Mexico .....	6.98	8.94	1.97	0.78	0.311	0.215	0.296
Panama .....	5.69	7.48	1.78	0.76	0.295	0.227	0.310
Paraguay .....	4.85	6.82	1.97	0.71	0.265	0.259	0.348
Peru .....	6.70	8.44	1.73	0.79	0.314	0.206	0.282
Trinidad and							
Tobago .....	5.45	7.51	2.06	0.73	0.281	0.250	0.341
United States							
of America .....	3.47	4.77	1.31	0.73	0.283	0.236	0.329
Venezuela .....	6.17	8.04	1.87	0.77	0.297	0.229	0.308
<b>Asia and Pacific</b>							
Bangladesh .....	6.89	8.24	1.35	0.84	0.344	0.170	0.240
Fiji .....	6.26	8.01	1.75	0.78	0.316	0.211	0.291
Indonesia .....	5.21	7.15	1.94	0.73	0.287	0.253	0.342
Jordan .....	8.61	9.95	1.34	0.87	0.362	0.154	0.214
Malaysia .....	6.09	7.83	1.74	0.78	0.308	0.217	0.298
Nepal .....	5.61	7.02	1.41	0.80	0.321	0.204	0.279
Pakistan .....	6.85	8.27	1.42	0.83	0.342	0.176	0.247
Philippines .....	6.80	8.34	1.54	0.82	0.328	0.191	0.262
Republic of Korea .....	5.39	6.34	0.95	0.85	0.348	0.164	0.229
Sri Lanka .....	5.68	7.33	1.65	0.78	0.308	0.216	0.297
Syrian Arab							
Republic .....	7.59	9.05	1.46	0.84	0.345	0.170	0.237
Thailand .....	6.30	7.88	1.58	0.80	0.320	0.200	0.275
Turkey .....	6.03	7.70	1.67	0.78	0.306	0.210	0.288
Yemen .....	6.61	7.97	1.36	0.83	0.335	0.183	0.251

TABLE 6 (continued)

	Mean parity (1)	Mean sibship size (2)	Absolute difference (3) = (2) - 1	Relative difference (4) = (1)/(2)	0.5 fractile (5)	Index of dissimilarity (6)	Gini coefficient (7)
Europe							
Belgium.....	2.57	3.85	1.28	0.67	0.261	0.267	0.368
Czechoslovakia.....	2.32	2.94	0.62	0.79	0.316	0.194	0.267
Denmark.....	2.53	3.26	0.73	0.78	0.315	0.209	0.285
Finland.....	2.67	3.61	0.94	0.74	0.298	0.223	0.309
France.....	2.69	3.75	1.06	0.72	0.283	0.239	0.331
Italy.....	2.37	3.18	0.81	0.75	0.304	0.217	0.300
Netherlands.....	1.67	2.46	0.79	0.68	0.267	0.287	0.379
Norway.....	2.75	3.38	0.63	0.81	0.328	0.193	0.264
Poland.....	2.78	3.78	1.00	0.74	0.293	0.221	0.306
Portugal.....	2.91	4.60	1.69	0.63	0.239	0.278	0.384
Spain.....	3.09	4.08	0.99	0.76	0.301	0.207	0.295
United Kingdom of Great Britain and Northern Ireland.....	2.58	3.42	0.84	0.75	0.300	0.224	0.306
Yugoslavia.....	3.05	4.36	1.31	0.70	0.263	0.249	0.343

Source: World Fertility Survey, standard recode tapes.

highest mean parity, is, under the children's perspective, surpassed by Costa Rica and the Syrian Arab Republic, both of which show mean sibship sizes of more than nine children. The reason for the changes in the rank order lies in differential shapes of the parity distributions, resulting in different relative variations of the distribution.

Columns (3) and (4) of table 6 give the absolute and relative differences between the two means. Seven countries—namely, Cameroon, Colombia, Costa Rica, Dominican Republic, Guyana, Jamaica and Trinidad and Tobago—have a difference of more than two children between mean parity and mean sibship. Six of those countries are in Latin America, indicating that the unevenness in the parity distribution tends to be greatest there. In Europe the absolute differences are on the order of between 0.62 (Czechoslovakia) and 1.69 children (Portugal).

Relative differences (column 4),<sup>13</sup> however, are a better measure of unevenness, because they abstract from the level of fertility and are therefore better suited to a comparison between high- and low-fertility countries. The relative difference between mean parity and mean sibship size turns out to be greatest in two countries as different as Portugal (63 per cent) and Cameroon (65 per cent). The parity progression ratios in figure I tell us that in Portugal, this is due to unusually high progression ratios at higher parities, compared to the typical pattern of a low-fertility country; in Cameroon we find the opposite form of deviation from the typical high-fertility pattern—namely, unusually low progression ratios at low parities. Both deviations from the average high- and low-fertility patterns result in increased relative variance or, in other words, a stronger heterogeneity of the countries' female populations. Next to these two extreme

cases come several Latin American and European countries, where the mean parities tend to be between 65 per cent and 75 per cent of the mean sibship sizes. The lowest relative difference is found in some African and Asian countries (Jordan, 87; Kenya, 86; Republic of Korea, 85; and Ghana, 84 per cent).

Another way to look at the unevenness in the fertility distribution is to view it in terms of concentration. Concentration analysis generally studies the degree to which a certain proportion of producers dominates the market—that is, makes a large proportion of the products.<sup>14</sup> In the case of fertility analysis, women may be seen as potential producers, whereas the children are considered to be the products. Hence, we study what proportion of women produces what proportion of children. The best way to describe this for the complete parity distribution is the Lorenz curve, which on the *x*-axis has the cumulated proportions of women at each parity and on the *y*-axis the cumulated proportions of children borne by those women (see fig. III). Women are ranked from most productive (highest parity category) to least productive (childless). Table 7, which is derived from the information given in table 2, provides an illustration of the method used to construct the curve.

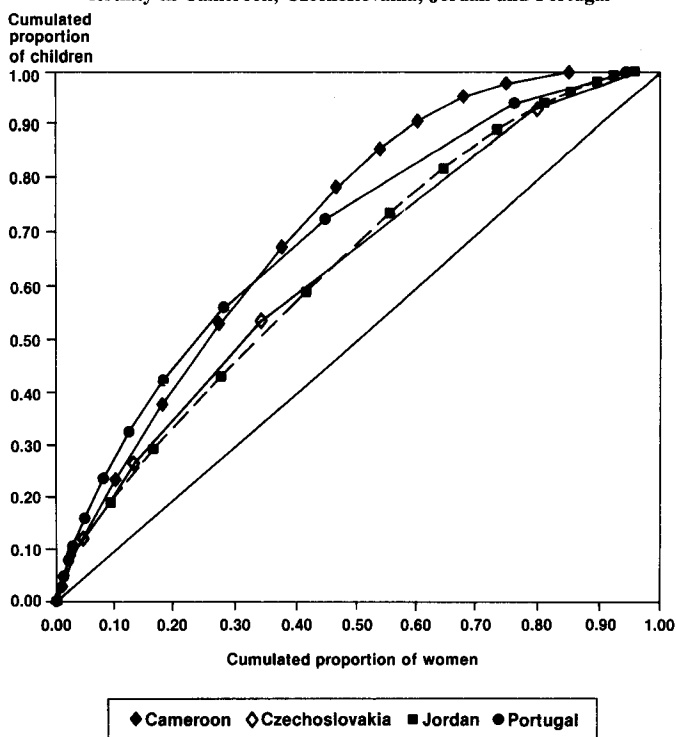
Figure III gives Lorenz curves for four selected WFS countries. The diagonal stands for the case of a completely even distribution, with all women having the same number of children. The farther the Lorenz curve moves away from the diagonal, the greater is the concentration of the distribution. It is interesting to see that Jordan, the country with the highest level of fertility, and Czechoslovakia, with one of the lowest levels, show an equally low degree of concentration. The Lorenz curves of Cameroon and Portugal clearly lie further away from the diagonal, indicating a significantly higher degree of concentration in the distribution of completed family sizes. A cross-over of the curves indicates that the major sources of concentration lie at different ends of the parity distribution. (The case of Portugal and Cameroon has been discussed above.)

TABLE 7. LORENZ CURVE CONSTRUCTION FOR  
CZECHOSLOVAKIA, EVER-MARRIED WOMEN AGED 40-45

Parity <i>i</i>	Proportion of women at parity <i>i</i>	Proportion of children born to women of parity <i>i</i>	Cumulated proportion of women at parity <i>i</i> and above	Cumulated proportion of children born to women of parity <i>i</i> and above
0 .....	.039	0	1.000	1.000
1 .....	.165	.071	.961	1.000
2 .....	.451	.389	.797	.930
3 .....	.212	.274	.346	.541
4 .....	.085	.147	.134	.267
5 .....	.030	.065	.048	.120
6 .....	.013	.034	.019	.055
7+ .....	.006	.021	.006	.021

Source: World Fertility Survey, standard recode tapes.

Figure III. Lorenz curves for measuring concentration of fertility in Cameroon, Czechoslovakia, Jordan and Portugal



Source: World Fertility Survey, standard recode tapes.

The Lorenz curves give a complete picture of the distribution and contain all the information necessary to describe differential concentration patterns in detail. For many analytical purposes, however, it is desirable to have a single indicator of concentration rather than the full Lorenz curve. Many such indices exist in the literature. The most popular ones for the study of relative concentration are the Gini coefficient and the index of dissimilarity. Both can be directly derived from the Lorenz curve: the Gini coefficient gives the area between the Lorenz curve and the diagonal as a fraction of the full triangle under the diagonal, whereas the index of dissimilarity gives the maximum vertical distance from the Lorenz curve to the diagonal.<sup>15</sup> Other possible indices that have a more intuitive interpretation are the fractiles. They indicate what proportion of women have 10 per cent, 25 per cent, 50 per cent etc. of all children. A disadvantage of fractiles that are close to one end of the Lorenz curve lies in the fact that they are rather insensitive to changes at the other end of the curve. For that reason the 50 per cent fractile is preferable to the others as a summary indicator of the whole curve. Graphically the 50 per cent fractile is the X-

value at which a horizontal line at the level of  $Y = 0.5$  crosses the Lorenz curve (see fig. III). It can be easily interpreted as the proportion of women who have half the children.<sup>16</sup>

To facilitate a comparison between the different indices of concentration, table 6 lists the 0.5 fractile, the index of dissimilarity and the Gini coefficient. For the latter two, a higher value of the index means higher concentration; for the 0.5 fractile, a lower percentage of women who have half the children indicates higher concentration. Since all three indices attempt to summarize the information given by the Lorenz curve by different means, it is not surprising that they show essentially the same pattern. Calculating correlation coefficients between the various indicators over all WFS countries results in coefficients of above 0.96.<sup>17</sup> It is also not surprising that the correlation coefficient between any of the concentration indices and the measure of relative differences between mean parity and mean sibship (column 4 in table 6) is much higher (above 0.96) than in relation to the absolute differences (around 0.45), because unlike the concentration indices and the relative difference, the absolute difference depends on the level of fertility.

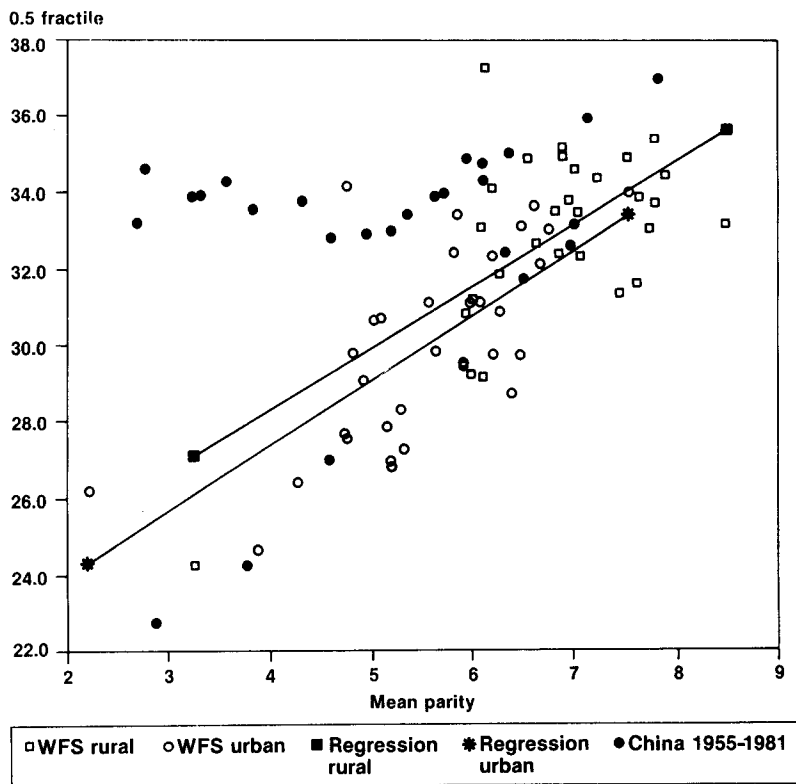
It is, however, surprising to see that the level of fertility (mean parity) shows a rather clear negative association to concentration: the lower the level of fertility, the higher the concentration. This results in a correlation coefficient of 0.59 between mean parity and the 0.5 fractile. If only the 41 WFS developing countries are considered, the correlation even increases, to 0.981. A closer analysis of this pattern reveals that, over the course of demographic transition, a declining level of average fertility is associated with increasing relative variation in the distribution and, hence, increasing concentration. This strong association has also been shown for marital fertility declines in Germany and Austria between 1895 and 1939, by the United States of America in occupational groups (Lutz and Vaupel, 1987) and for time series of parity distributions (Vaupel and Goodwin, 1987; King and Lutz, 1988). In addition to this evidence, most high-fertility countries for which parity distributions are available by age groups beyond reproductive age confirm the strong negative association between the level of fertility and concentration over the course of demographic transition (Lutz, 1987).

For the European countries and the United States of America, that general pattern no longer holds. At their stage of development, lower average fertility does not necessarily increase concentration, which is generally lower at a given level of fertility than could be expected from the developing countries' association between the two variables. There seem to be two reasons for this: first, in Europe, where sizeable proportions of women remain unmarried, the fact that the WFS samples include only married women makes the pattern more homogeneous; secondly, most of the cohorts studied in the European surveys had taken part in the post-war baby boom, which, above all, had resulted in an amazingly homogeneous pattern of child-bearing (Lutz, 1987; Vaupel and Goodwin, 1987). Recent trends observed from other statistical sources (Lutz and Vaupel, 1987) seem to indicate that recent fertility declines in Europe are again associ-

ated with increasing concentration, mostly because of increasing proportions of childless women.

Figure IV plots the association between the mean parity and the 0.5 fractile for rural and urban subsamples of WFS developing countries. Although they represent cross-sectional data, one might interpret them in terms of a trend when we assume that the countries are, with some variation, at different stages of a universal movement from high to low fertility. The pattern appearing for the WFS cross-section corresponds exactly to the time-series evidence from other sources stated above. Even the regression lines for the subsets of urban and rural populations are almost exactly parallel. Generally, Asian countries tend to lie above the regression lines—that is, reveal less concentration at a given level of fertility, whereas Latin American countries tend to lie below the line. The Republic of Korea is the most extreme case of the Asian pattern (outlier above the regression line), but even there the line from the rural to the urban sub-populations runs parallel to the general trend.

Figure IV. Relationship between mean parity and the concentration of fertility (0.5 fractile) for a cross-section of developing countries and China, 1955-1981



Source: World Fertility Survey, standard recode tapes.

An interpretation for this seemingly universal pattern of association between the level of fertility and its concentration over the course of demographic transition is that women tend to adopt the new patterns of reproductive behaviour at different speeds. In a natural-fertility situation, differential fecundability is the major source of variation in the completed parity distribution. When, however, parts of the population start to practise family limitation while others stay with traditional patterns, an additional source of variation in the fertility distribution is introduced which results in higher concentration. This is not a statistical artefact; if reduction in fertility were proportional, it would not affect concentration. It can be shown that this association between the level and the concentration of fertility is not a statistical artefact<sup>18</sup> but reflects real changes in the degree of heterogeneity in reproduction. There is, however, one very prominent exception to the general pattern which proves that strong fertility declines do not automatically lead to higher concentration.

#### *China: the great exception*

Feeney and Yu (1987) recently presented estimates of period parity progression ratios for China for the period 1955-1981, based on the National One-per-thousand Fertility Survey. These parity progression ratios can be easily converted into completed parity distributions implied by observed period behaviour and serve as input data to an analysis of distributional aspects of Chinese fertility trends.

Table 8 gives the mean parity, mean sibship size and the 0.5 fractile as a measure of concentration for all years from 1955 to 1981. Since these measures are based on period data, they are also subject to short-term period fluctuations as well as longer-term trends. The period mean parity given is comparable to the total fertility rate calculated from age-specific observations: both give the mean number of children of a synthetic cohort, based on period observations. Here, the mean family sizes calculated from completed parity distributions are not exact in considering births of orders eight and above.<sup>19</sup> Further, the time series of total fertility rates and mean parities under a parity-specific approach cannot be expected to be identical, because one approach considers the age distribution of the population while the other is based on the parity distribution. But since age and parity are highly correlated, the empirical findings will not be very different (Lutz and Feichtinger, 1985).

The series of period fertility levels in China show two strong declines since 1955: a precipitous decline from a mean parity of above 7 in 1957 to as low as 2.88 in 1961, followed by a fast and full recovery in the following two years, and a somewhat slower but lasting decline since the early 1960s. While the first may be considered a short-term fertility fluctuation due to crisis and famine, the second represents the great and extraordinarily rapid Chinese fertility transition. The decline was especially impressive in the urban areas of China, where the fertility level in 1981 was lower than that in many European countries. Aside from this well-known and often-referred-to trend in the level of Chinese fertility, table 7 also

TABLE 8. MEAN FAMILY SIZES AND CONCENTRATION OF FERTILITY IN CHINA, 1955-1981

Year	Total			Rural			Urban		
	Mean parity	Mean sibship size	0.5 fractile	Mean parity	Mean sibship size	0.5 fractile	Mean parity	Mean sibship size	0.5 fractile
1955	6.99	8.14	0.33	7.08	8.19	0.33	6.36	7.70	0.32
1956	6.53	7.92	0.32	6.56	7.94	0.32	6.36	7.74	0.32
1957	7.02	8.08	0.33	7.04	8.11	0.33	6.79	7.89	0.33
1958	6.35	7.65	0.33	6.39	7.71	0.32	6.09	7.29	0.34
1959	4.57	6.54	0.27	4.55	6.62	0.27	4.60	6.10	0.30
1960	3.77	5.94	0.24	3.62	5.94	0.23	4.31	5.80	0.29
1961	2.88	4.86	0.23	2.86	4.88	0.22	3.05	4.74	0.25
1962	6.12	7.19	0.34	6.30	7.34	0.35	5.16	6.24	0.33
1963	7.83	8.27	0.37	7.95	8.34	0.37	6.89	7.77	0.35
1964	7.13	7.88	0.36	7.38	8.02	0.36	5.28	6.32	0.33
1965	6.37	7.33	0.35	6.92	7.72	0.36	3.91	4.77	0.33
1966	6.11	7.07	0.35	6.80	7.57	0.36	3.29	4.05	0.33
1967	5.20	6.30	0.35	5.83	6.84	0.34	3.20	3.83	0.34
1968	5.95	6.86	0.35	6.45	7.24	0.36	4.00	4.77	0.34
1969	5.64	6.62	0.34	6.06	6.94	0.35	3.79	4.59	0.34
1970	5.72	6.71	0.34	6.15	7.04	0.36	3.61	4.33	0.34
1971	5.36	6.39	0.34	5.79	6.72	0.35	3.23	3.90	0.34
1972	4.93	5.97	0.33	5.33	6.29	0.34	3.09	3.73	0.33
1973	4.57	5.55	0.33	4.66	5.80	0.32	2.93	3.45	0.35
1974	4.28	5.10	0.34	4.65	5.43	0.35	2.61	3.03	0.36
1975	3.83	4.59	0.34	4.15	4.88	0.35	2.41	2.84	0.35
1976	3.55	4.23	0.34	3.85	4.49	0.35	2.23	2.63	0.34
1977	3.29	3.92	0.34	3.53	4.14	0.35	2.17	2.55	0.35
1978	3.21	3.79	0.34	3.43	3.99	0.35	2.12	2.42	0.38
1979	3.22	3.81	0.34	3.43	4.00	0.35	2.00	2.31	0.38
1980	2.72	3.23	0.35	2.93	3.41	0.35	1.62	1.89	0.37
1981	2.66	3.25	0.33	2.92	3.47	0.34	1.62	1.89	0.37

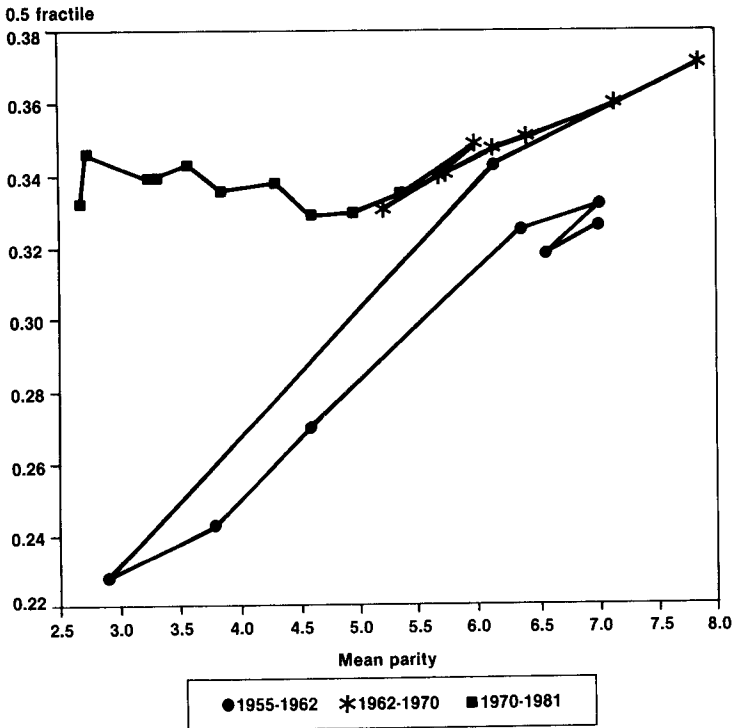
Source: Feeney and Yu, "Period parity progression measures of fertility in China", *Population Studies*, vol. 41, No. 1 (March), pp. 77-102.



reveals a much less known feature: the fertility transition was not associated with an increase in concentration but seems to have taken place in an extremely homogeneous manner.

Figure V illustrates the association between the level of fertility and its concentration in China between 1955 and 1981. Until 1958 the pattern was not different from that in the high-fertility WFS countries described above. The extreme short-term decline in period fertility during the years of famine and crisis (1958-1961) brought about a very significant increase in concentration, followed by a return to the pre-transitional régime in both the level and concentration of fertility. During those years of crisis and recovery, the association between the level and concentration followed almost exactly the regression line shown in figure IV for the cross-section for developing WFS countries. The recent fertility decline since 1963, however, was of a very different nature: after some initial increase in concentration, since 1970 the 0.5 fractile remains at an almost constant level of low concentration. This deviation from all other known cases of fertility

Figure V. Relationship between mean parity and the concentration of fertility (0.5 fractile) for China, 1955-1981



Source: Feeney and Yu, "Period parity progression measures of fertility in China", *Population Studies*, vol. 41, No. 1 (March 1987), pp. 77-102.

transition is even more extreme in the urban areas of China, where, at very low levels of fertility (mean parity: below two children), degrees of evenness were achieved that to our knowledge have not been experienced by any other sizeable population.

A consequence of the stable level of concentration (or even decrease in urban areas) is that the mean sibship—that is, the mean family size from the child's perspective—declined even more strongly than the mean from the women's perspective: from 8.27 in 1963 to 3.25 in 1981. Contrarily, during the extraordinary fertility decline of 1959-1961 the mean from the children's perspective declined less than that from the women's perspective because of simultaneously increasing concentration. By 1981 the mean family size from the children's perspective had declined to the very low value of 1.89 in the cities of China. That is probably the lowest value of mean sibship size of any sizeable population in the whole world, including the very low-fertility cities of the Federal Republic of Germany or northern Italy. The reason is that even in a modern industrialized city where the total fertility rate might be lower than in Chinese cities, the mean family size from the children's perspective is greater because of higher concentration: this is mainly a consequence of the high proportions of women expected to remain childless (generally more than 30 per cent) in European cities. In sharp contrast to this, the parity-specific fertility pattern of urban China in 1981 implies that only 2.1 per cent<sup>20</sup> of all women remain childless.

#### DISCUSSION

The number of children in a family is of fundamental importance not only to the parents and to the children but also to society. Past and present fertility patterns have far-reaching consequences on matters ranging from the size of future generations to the mode of individual socialization in childhood and are thus of sociological, economic, political, psychological and even cultural concern. Scientific literature on the consequences of fertility levels is abundant. There is no doubt that the level of fertility is important, but the nature of the effects is still quite controversial. In the paragraphs below, a much less recognized aspect of the consequences of fertility patterns will be discussed—namely, the effects of the distributional aspects of fertility that are independent from the level of fertility. Hence, we want to discuss whether, at a given level of fertility, higher or lower relative variance or concentration of the distribution makes any difference. A few selected aspects of this will be briefly mentioned below without any in-depth discussion.

First, in the strictly demographic field it can be shown that some degree of orientation<sup>21</sup> of daughter's family size on the mother's family size, together with some concentration (non-zero variance) in the parity distribution, will result in higher fertility levels in the next generation. To make this point clearer, we may think of the extreme theoretical case: every daughter having the same number of children as her mother. If the parity distribution were completely even—that is, if every woman had the

same number of children—then the level of fertility would not change from one generation to the next. If, however, the distribution had some variance and consequently the mean sibship size of the daughters were higher than the mean parity of the mothers, then the level of fertility would increase from mother's generation to daughter's generation by exactly the difference between the two means. This model has been generalized by Lutz and Pullum (1988) for the more realistic cases of only weak, positive orientation instead of perfect replication of the mothers' family sizes. The fact that the empirically observed level of fertility does not increase in most countries, despite observed weak orientations of daughters' family sizes on their mothers' family sizes and observed variance in the distribution, can then be due only to a declining trend in fertility levels induced by the numerous other fertility determinants. In other words, without this small positive effect of the relative variance in parity distributions, the decline would have been even stronger.

Another more obvious consequence of the distributional aspect of fertility lies in the degree of kin availability for the elderly. In an aging population, one of the major social concerns is the care for the increasing number of elderly people. And the care provided by family members—mostly daughters—plays a crucial role in most societies. This is where the distribution of children becomes very important. Obviously, the number of elderly without any living children is—even at a given level of fertility—much greater if the proportion of childless women and therefore the concentration of the distribution is greater. On the other hand, if the fertility pattern is very homogeneous and every woman has, for example, two children, then the problem of elderly without living children or siblings would be almost non-existent. In some Asian countries where the elderly are largely dependent on family support, this aspect is of utmost importance.

Family size distributions have other important consequences in the areas of housing demand and consumer goods. Aside from general trends in the level of fertility, the structure of housing demand is affected by whether there are many childless families and at the same time a number of very large families or whether all families are about the same size. Parity distributions also affect the demand for durable consumer goods designed for children (e.g., toys, children's furniture, etc.). The demand for such goods will be greater in the case of homogeneity, where every family will buy the goods, than in the case of some large families which can use the same goods for all of their children. The structure of consumer goods demanded is more significantly influenced by the effect of children on the per capita income of the family and the resulting inequality in economic standing.

Many other consequences of the distributional aspect of fertility patterns can be identified—even individual socialization in the family, where it seems to make a difference whether one is a single child or has many brothers and sisters. The major point of this discussion, however, is not to compile a complete list of the consequences of fertility distributions but rather to indicate that distribution does indeed have an impact on many matters besides the average level of fertility.

Much more research on parity distributions is needed. In particular, the function and the modeling of parity distributions in terms of certain parameters merit further exploration. Pullum, Tedrow and Herting (forthcoming) have suggested a method based on the Brass relational logit model that focuses on the parity attainment proportion, a function which is equivalent to the  $l(i)$  function in our life-table notation above. For completed parity distributions of the United States cohorts born 1873-1933, they find the existence of a formal continuity among the successive distributions. However, for the 54 WFS distributions studied here, logit transformations of the  $l(i)$  functions for many countries did not come sufficiently close to linearity and hence resulted in a very heterogeneous picture. This indicates that for a global analysis of parity distributions from very different societies, models should have a higher degree of differentiation, possibly with different standard functions, such as in the case of regional model life tables.

Finally, the above analysis shows that the extent of concentration of fertility or the "division of labour" for society's reproduction is far from being a universal constant.<sup>22</sup> Our study indicated, however, that over the course of demographic transition, there seems to be a regular pattern of increasing concentration with declining fertility levels. The exceptional case of China once more proves that this is not a demographic axiom but is highly dependent on the structure of society and its heterogeneity, not only in socio-economic terms but also with respect to its value system. These questions seem to merit much further analysis, especially on the level of subnational populations differentiated by ethnic, regional, religious or socio-economic criteria. Reproductive heterogeneity between such groups with inheritable characteristics is a major determinant of the future population composition.

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#### NOTES

<sup>1</sup> This indirect measurement approach was introduced by Coale (1971) and further developed by Coale and Trussell (1974 and 1978).

<sup>2</sup> Recently P. David and others (1988) suggested a model of cohort parity analysis (CPA) that measures the extent and timing of the adoption of fertility control within marriage. Earlier, Glass and Grebenik (1954) and Matras (1965) used model parity distributions to compare different fertility régimes.

<sup>3</sup> Some of the WFS-based publications that explicitly consider parity distributions are Hobcraft and McDonald (1984), Hodgson and Gibbs (1980) and Lutz (1985).

<sup>4</sup> For some reason Portugal was treated as a developing country by WFS, although its fertility level was lower than that of Spain, Yugoslavia or the United States of America.

<sup>5</sup> The author is grateful to the WFS and ECE authorities for giving him the opportunity to use the data sets in London and Geneva.

<sup>6</sup> A sensitivity analysis of the model with respect to age-distributional effects was conducted by Lutz and Feichtinger (1988).

<sup>7</sup> Within the concept and notation of this model, data on mean ages at birth of certain orders broken down by completed parity can be used to calculate correct birth intervals (and avoid the mistake of comparing the crude mean ages that refer to different groups of women) and several measures of the family life cycle, such as the mean duration from birth of order  $i$  to completion of family size. For a more detailed description, see Lutz and Feichtinger (1985).

<sup>8</sup>To account for the fact that some women in the  $m+$  category have more than  $m$  children, the mean of this category was assumed to be  $m+1$  in our calculation.

<sup>9</sup>In this context the data for the Netherlands are not strictly comparable to those for the other countries because they are restricted to the marriage cohorts 1963-1973.

<sup>10</sup>Educational and residential differentials were given only if a subgroup included more than 120 women and if it comprised between 20 and 80 per cent of the total. The dichotomies rural/urban and low-high education correspond to the definitions generally used by WFS. In developing countries, the category of low education generally refers to women without any formal education and the other category to women with at least some education.

<sup>11</sup>The Netherlands has only 24 per cent with a third child, but the sample is not strictly representative.

<sup>12</sup>Vaupel and Goodwin (1985) modified Preston's algebra slightly by presenting the mean ratio:

$$P = \frac{\bar{x}}{c} = \frac{1}{I + 1}$$

where  $I$  is the square of the coefficient of variation of the distribution of women by number of children. That is,

$$I = \frac{\sigma_x^2}{\bar{x}^2}$$

$I$  was introduced by Crow (1958) as a summary measure of unevenness of a distribution.

<sup>13</sup>Identical with the mean ratios introduced in note 12.

<sup>14</sup>It is helpful to distinguish between absolute concentration, which looks at the share produced by a certain absolute number of producers (e.g., the top 10), and relative concentration, which refers to a certain proportion of all producers (e.g., the top 10 per cent). In this study of fertility concentration, we are interested in relative concentration only.

<sup>15</sup>If  $X_i$  and  $Y_i$  are respective cumulative percentages and  $n$  is the number of units, then the Gini coefficient  $G$  is defined by

$$G = \left[ \sum_{i=1}^n X_i Y_{i+1} \right] - \left[ \sum_{i=1}^n X_{i+1} Y_i \right]$$

The index of dissimilarity may be defined as the sum of the positive differences between the two percentage distributions  $x_i$  and  $y_i$ :

$$DI = \frac{1}{2} \sum_{i=1}^n [x_i - y_i]$$

<sup>16</sup>For this reason Vaupel and Goodwin (1985) refer to the 0.5 fractile as the "have-half".

<sup>17</sup>The Gini coefficient and the index of dissimilarity seem to be very closely related to a correlation coefficient of 0.993.

<sup>18</sup>If the reduction in fertility were proportional in all groups of women, this would not affect concentration.

<sup>19</sup>Since the parity progression ratios given by Feeney and Yu (1987) ended at parity 8, one must make adjustments for higher-order births. In this paper it is assumed that women with eight or more births have, on the average, nine births.

<sup>20</sup>The percentage is low even under the assumption that all of the 0.7 per cent of the women who remain unmarried according to the period rates also remain childless. Of all married women the expected proportion childless is only 1.4 per cent.

<sup>21</sup>We do not use the notion of correlation here, because the correlation coefficient could also be high if the daughter's fertility were generally lower by a constant (e.g., one child) than the mother's fertility. Hence, orientation could be defined as analogous to correlation but without the insensitivity to shifts up and down the scale. For a detailed discussion of this, see Lutz and Pullum (1988).

<sup>22</sup>Vaupel and Goodwin (1987) mentioned selected pieces of evidence that might imply the existence of a universal constant of about 25 per cent of the women having half the children. But, as they also point out, these seem to be points on a complicated pattern and not instances of a universal demographic constant.

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## **PREDICTING THE “VAGARIES” OF MORTALITY: THE CAUSES OF DEATH IN BAMAKO FROM 1974 TO 1985**

*N. Bonneuil and P. Fargues \**

### SUMMARY

The overall downward trend in African mortality is interrupted by recurring epidemics. This article investigates the nature of those temporary fluctuations, using cause-of-death data drawn from deaths registered in Bamako over an 11-year period. The use of time-series analysis produces findings that can be used in three ways: first, to provide a short-term forecasting tool for public health officials; secondly, to clarify the relationship between the cause of death and exogenous mortality factors, particularly climatic and economic; thirdly, to assist in the analysis of synergies between disease and malnutrition, and intra-household transmission as an aggravating factor contributing to the seriousness of a disease.

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African mortality is finally yielding its secrets. We now know that it is declining everywhere, in some places very sharply (Hill, 1987) and in cities more than in the countryside (Antoine and Diouf, 1988). We also know that the decline has not freed mortality from the seasons, which set the rhythm of death in both the cities and the countryside, nor are the trends immune to set-backs (Fargues and Nassour, 1987). Rather, the decline is a trend marred by recurring epidemics. We shall attempt in this paper to expand on its description: Does an apparent unevenness in the decline reflect regular, non-seasonal trends—for example, cycles of several years varying in length and scope according to the cause of death? Or is the irregularity due to accidents in climatic series which would explain the seasonal mortality fluctuations? The irregular and the unexpected are the factors that have so far defied description.

A primary objective is to provide those in charge of public health with a short-term forecasting tool. The methods of statistical analysis of time series which we propose to apply to registered deaths by cause make it possible to simulate the future behaviour of a variable on the basis of past observation.

A second objective is to clarify the relation between the cause of death and exogenous mortality factors, climatic and economic. Demogra-

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phers have traditionally examined seasonal mortality variations based upon monthly averages established over several years of observation (see fig. I). We reintroduce here the contingent character of the time series. When deaths tend to occur at the same time during the year, which is in turn characterized by a particular climatic condition, they are considered seasonal deaths. The correspondence that may be observed between average monthly series of climatic indicators, on the one hand, and death by specific cause, on the other, highlights the periods of risk for each illness. However, this correspondence, observed as a long-term average, does not indicate whether the degree of risk varies with the height of the temperature, maximum precipitation or the duration of the dry season etc. We know, for example, that an outbreak of measles or meningitis may be expected in the month of March. It is not known whether vigilance should be increased if the month of March is hotter than usual. It is also known that malnutrition usually kills with the rains—that is to say, at the time of change. Are there more deaths when the rain comes after a year of deficit rainfall?

Figure I. Seasonal variations in mortality at Bamako, 1974-1985

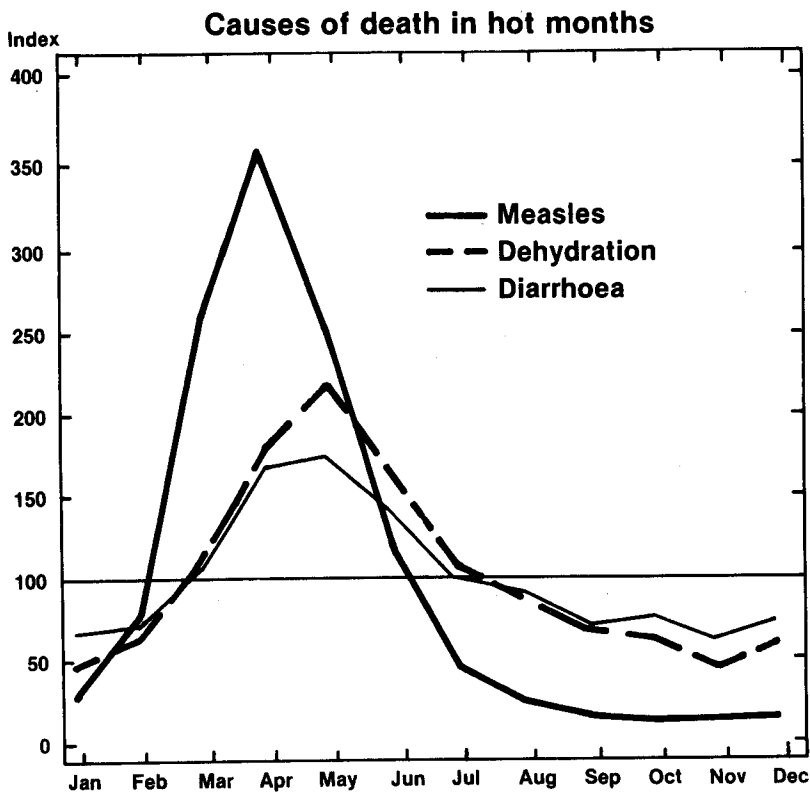
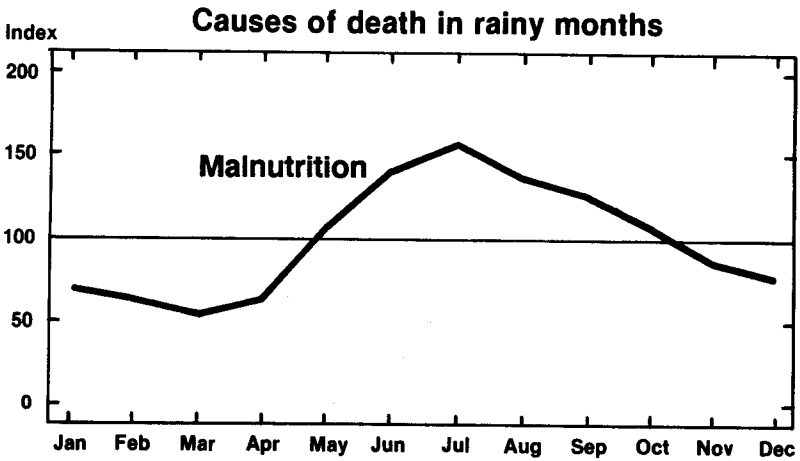
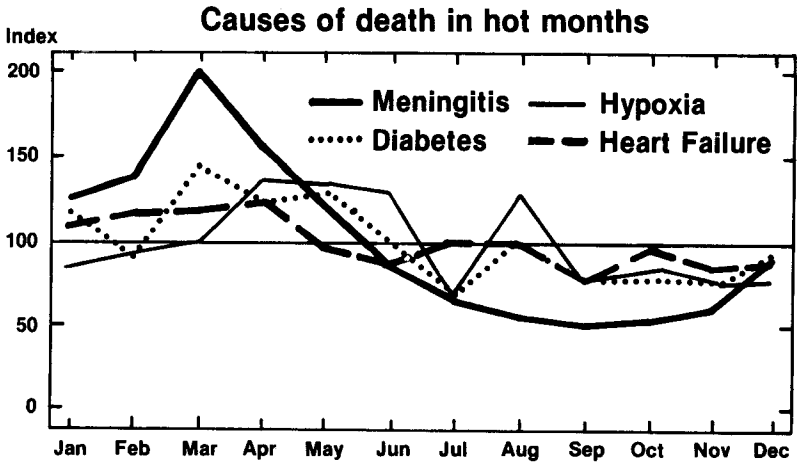


Figure I (continued)



Source: Christian Gourieroux and Alain Montfort, *Cours de séries temporelles* (Paris, Economica, 1983).

A third objective is to contribute to some basic debates on the causes of mortality and the systems they form among themselves. By analysing a series of deaths caused by measles, malnutrition and infectious diarrhoea, we shall attempt to establish elements that will either confirm or weaken some hypotheses regarding the synergy between malnutrition and infection (Solimano and Vine, 1982; Mosley and Chen, 1984), the link between measles and diarrhoeal diseases or the transmission of infectious diseases in the home from older to younger siblings as an aggravating factor (Aaby, 1987).

After presenting data gathered in Bamako and recalling the principal findings relevant to this study, we shall propose a general method aimed at answering the kinds of questions asked above. Finally, we shall review the principal results and discuss their significance.

#### DATA AND PREVIOUS FINDINGS

The data analysed are those of the death registers kept by the Bureau of Hygiene, which issues burial permits for the cemeteries of the city of Bamako: 49,829 deaths and 5,427 still births registered from 1 January 1974 to 31 December 1985.<sup>1</sup>

These records represent an urban population, that of the district of Bamako (city and suburbs), give or take some "mortality migration". We refer here to the move that may precede or follow death, when a rural person dies in a city where he has gone to be treated or, conversely, when a city dweller is buried in a village.

While these registers are exceptionally well kept for Africa, they are far from complete. The rate of coverage varies according to sex and age. For the period 1974-1985, the average rates of coverage are indicated in table 1.

In the records, six characteristics of death are noted: date of death; sex; age; place of death (hospital, home, public thoroughfare); place of birth; cause of death.

TABLE 1. RATE OF COVERAGE IN DEATH REGISTERS  
IN BAMAKO (AVERAGE, 1974-1985)

Age <sup>a</sup>	Male (percentage)	Female (percentage)
0-4 years .....	60.8	53.4
5 years and over .....	82.1	72.1

Source: Philippe Fargues and Ouaidou Nassour, *Douze ans de mortalité urbaine au Sahel: âges, saisons et causes de décès à Bamako de 1974 à 1985* (Paris, Presses universitaires de France, 1988).

<sup>a</sup> Estimate of coverage rate is based on a method which allows for a constant rate within these two age groups.

*Variables used: date and cause of death*

The data used in this article consist of distributions of deaths by date and cause.

The date variable (month and year of death) consists of 144 categories numbered from 1 (January 1974) to 144 (December 1985). Since the delay between death and burial is always brief, we consider the dates to be accurate.

The cause of death shown on the registers is always very briefly stated; sometimes it is erroneous. It is brief in that only one cause is reported: it may be the initial cause, or the immediate cause, or at other times one of the factors leading to death, but never all three. Deaths having the same origin may thus appear under different categories. Moreover, the cause recorded in the registers may be erroneous because it is not always the result of examination of the deceased. The most favourable conditions exist when death occurs in a "health facility" (60.1 per cent of all deaths), where the cause recorded may be considered accurate, although perhaps incomplete. On the other hand, when death occurs at home, probable cause is established after the fact by a kind of "verbal autopsy". The diagnosis is accurate if the disease causing death is identified properly (Fontaine and Garenne, 1988), which is not always the case.

In the case of malaria, for example, we shall see that multivariate analysis offers methods that eliminate mis-reporting. Whether it is handled properly or not, the verbal autopsy questionnaire yields few details on the disease: for example, meningitis is reported without specification of its bacterial or non-bacterial origin. For this reason a small number of causes appear with high frequency. The only causes of death used in this article are those showing a frequency of over 250 cases (table 2). We have analysed them as recorded on the registration certificates, even if they are imprecise and do not conform to the international disease classification (IDC). Such is the case with icterus, for instance, which is a symptom—not a cause of death.

Unlike conventional demographic analyses based on rates and therefore requiring adjustment for deaths (the numerators) when vital statistics are incomplete, the methods we use here employ absolute numbers of events directly (shown in solid lines on figs. II-XI). This avoids the common source of error in the adjustment techniques used. At the same time, these methods imply an additional hypothesis. For each cause considered separately, use of the distribution of numbers of deaths by month requires that we assume that the rate of errors of omission does not vary significantly over time. We have indicated elsewhere (Fargues and Nassour, 1988) that fluctuations of these rates from year to year are small during the period under study.

Some causes of death have a seasonal profile that varies with age. At times, we shall work with a distribution of deaths by date, cause and age, using age-group categories that are generally less affected by error in age reporting: 0 years, 1-4 years, 5 years and over.

TABLE 2. DISTRIBUTION OF DEATHS ACCORDING TO SPECIFIC CAUSE (MOST FREQUENT CAUSES), BAMAKO, 1974-1985

Cause (as recorded in the registers)	ICD code <sup>a</sup>	Number of cases (total)	Percentage	Contribution to decline in mortality from 1974 to 1985 <sup>b</sup> (percentage)
Measles.....	55	5 910	11.9	4.3
Malaria (or pernicious attacks).....	84	4 652	9.3	10.0
Diarrhoea (or infectious diseases).....	9	3 290	6.6	3.4
Prematurity.....	765	2 920	5.9	17.0
Cardiac insufficiency....	428-429	1 606	3.2	1.9
Malnutrition.....	269	1 597	3.2	6.4
Lung disease.....	518	1 492	3.0	0.9
Cirrhosis.....	571-572	1 347	2.7	2.8
Pneumonia.....	485-486	1 206	2.4	4.7
Tuberculosis.....	10-18	1 199	2.4	0.8
Meningitis.....	320-322	1 190	2.4	1.3
Sudden death.....	798	900	1.8	c
Dehydration.....	276	883	1.8	2.1
Icterus.....	782	742	1.5	1.9
Cranial traumatism.....	854	688	1.4	c
Abdominal pain.....	789	676	1.4	1.7
Anaemia.....	285	675	1.4	-0.2
High blood pressure....	401	661	1.3	c
Tetanus.....	37	520	1.0	3.7
Kwashiorkor.....	260	391	0.8	1.5
Renal insufficiency.....	586	362	0.7	c
Hypoxia.....	768	309	0.6	2.3
Asthma.....	493	292	0.6	c
Other causes.....		16 321	32.8	
All recorded deaths.....		49 829	100.0	100.0

Source: Philippe Fargues and Ouaidou Nassour, *Douze ans de mortalité urbaine au Sahel: âges, saisons et causes de décès à Bamako de 1974 à 1985* (Paris, Presses universitaires de France, 1988).

<sup>a</sup> International disease classification.

<sup>b</sup> Contribution of cause of death to the decline in mortality is defined by the variation between two periods, 1974-1979 and 1980-1985, of the number of years of life expectancy that are lost because of c as compared to the overall variation of life expectancy: it is part of the decline in the general mortality rate defined by the evolution of mortality caused by c.

<sup>c</sup> Between -0.05 and +0.05 per cent.

#### BACKGROUND: DECLINING MORTALITY, WITH MARKED SEASONAL CHARACTERISTICS

The death registers of Bamako provide a rich source of material which may be used for the analysis of a broad range of topics. We refer briefly here to two conclusions from previous work on the data which led to the present study. They concern, on the one hand, recent mortality trends and, on the other, the seasonal profiles of the causes of death, both of which are discussed at length in two publications (Fargues and Nassour, 1987 and 1988).

From 1974 to 1985, mortality declined sharply in Bamako. However, the drop had certain characteristics. For example, it varied by age—the principal beneficiaries being children under five years of age, and particularly the newborn (table 3).

It also varied by cause. The part played by a given cause in the mortality decline does not generally correspond to the size of its contribution to total mortality. Thus, prematurity, which is the fourth cause of death and which has apparently declined due to extended prenatal care, accounts for the largest part of the gains made (17.0 per cent). After that come malaria and malnutrition, while the childhood infectious diseases such as meningitis, diarrhoea and measles remain far behind and were particularly resistant during the period under observation.

The drop was also irregular. The last year observed shows a sharp break in the trend. Due to a very strong measles epidemic in 1985, mortality between the ages of 0 and 5 almost doubled, as compared with 1984 ( $5q_0$  went from 90 to 165 per thousand, and  $e_0$  from 59.1 to 54.3 years).

The period under observation is long enough to reveal the seasonal cycles of mortality by specific cause. Monthly averages calculated for the period 1974-1985 show two peaks of excess mortality, the highest in March-April and the second in August-September. This average seasonality results from three specific profiles which are combined in variable proportions according to age (see fig. 1):

TABLE 3. INDICATORS DRAWN FROM GENERAL MORTALITY TABLES BY SEX IN BAMAKO, 1974-1979 AND 1980-1985

Indicator	Sex	1974-1979	1980-1985
Life expectancy at birth (years) .....	Male	45.7	55.1
	Female	50.3	57.6
Under-five mortality ( $5q_0$ ) per thousand live births .....	Male	222.8	130.8
	Female	218.3	136.8
Neonatal mortality ( $q_{nn}$ ) per thousand children aged one month .....	Male	55.1	24.0
	Female	37.3	18.4
Post-natal mortality ( $q_{pn}$ ) per thousand children aged one month .....	Male	65.4	44.4
	Female	68.3	49.5
Infant mortality ( $1q_0$ ) per thousand live births.....	Male	116.9	67.3
	Female	103.1	67.0
Child mortality ( $4q_1$ ) per thousand children aged one year .....	Male	120.0	68.0
	Female	103.1	74.8

Source: Philippe Fargues and Ouaidou Nassour, *Douze ans de mortalité urbaine au Sahel: âges, saisons et causes de décès à Bamako de 1974 à 1985* (Paris, Presses universitaires de France, 1988).

(a) Causes of death in the hot months: these are the important infectious diseases which affect mostly infants and children (1 month to 5 years of age), particularly measles, infectious diarrhoea and meningitis;

(b) Causes of death in the rainy months: these are active either at the time of rainfall (malnutrition, kwashiorkor, mostly from 1 to 5 years of age) or a few weeks later (malaria, at all ages);

(c) Causes of death without marked seasonality: these prevail in the period of premature birth (prematurity) and in adults (liver diseases, pulmonary tuberculosis, heart diseases).

## METHODS

The Gaussian process and the techniques of Box and Jenkins make it possible to model the record of observations of a specific cause of death. This is regarded as a stochastic process for which we propose a mathematical representation in order to render an optimum account of the natural phenomenon and to quantify as faithfully as possible the empirical data without, however, attempting to explain them.

The purpose is not to study a model established *a priori* on a standard decomposition of time series into longer-term trends and seasonality but to select an appropriate formulation based upon the finite number of observations in the series themselves (144 is a high enough number to validate the laws of large numbers) and to estimate the parameters. The expression obtained will be validated in order to forecast the subsequent behaviour of the process ( $X_t$ ), on the basis of the given data ( $X_1, \dots, X_{144}$ ). The observation of the past and short-term forecasting provide a means to examine the phenomenon.

If the analyst has additional information available in the form of one or more other time series, it is possible to examine the intertemporal correlations between all of the series taken together, without establishing an *a priori* structural model and without setting maximum lags beforehand. In that way the scattered time series can be crystallized into a structure of causal relationships (in an explicit statistical sense), which in turn makes possible an improvement in the predictability of every significant time variable. (In this type of multivariate analysis, all series are first deseasonalized.)

We refer the reader to annex I and to Gourieroux and Montfort (1983), Maurel (1987), Rabemananjara (1986) and Tassi (1986).

## RESULTS: A READING GRID OF TIME PROCESSES

The major causes of death in Bamako were screened through univariate and multivariate ARIMA models. Each cause could be juxtaposed with all the others and with *a priori* exogenous factors (rainfall, price index), tested for exogeneity according to Sims's (1972) method, so as to yield a system. Three types of time-series models were thus isolated. Non-exclusive, they represent three successive levels of analysis:

- (a) Causes of death correlated to their own history;
- (b) Systems of causes of death correlated to each other;
- (c) Causes of death correlated to economic and ecological exogenous factors.

*Causes of death correlated to their own history*

The frequency of deaths due to a cause at a given time may be correlated solely to its past values. The series appears to evolve in an intrinsic manner independent of any explanatory variable.

One would expect this to be true, for example, in the case of deaths caused by a disease which confers long-term immunity upon those who do not die of it. After an epidemic, the more extended the outbreak, the more people will be immunized and also the more people will have died. The scope of an epidemic will be inversely proportional to those preceding it. Moreover, the "memory" of past values may vary in accordance with the size of the age interval at risk for the particular disease. An epidemic has immunizing properties only for those persons who belong in that interval: its effect is "forgotten" as new generations come into it.

Inversely, one can expect to see an amplifying effect from one epidemic to the next in the case of diseases in which the relapses are more serious than the primary infections.

We shall examine in detail a disease that seems to belong in this category—measles—and will mention later the other causes of death that show the same characteristics.

The ARIMA result validated by us for measles shows its perfect seasonal nature with a 12-month cycle:

$$(1 + 0.705 B^{12})(1 + 0.814 B^{24})(1 - 0.382 B^{36})(1 - 0.599 B)measl_t = \frac{\epsilon_t}{1 - B^{12}}$$

(0.090)                      (0.126)                      (0.138)                      (0.076)

which may be developed as follows, in order to facilitate its interpretation:

$$(1 + 0.705B^{12} + 0.814B^{24} + 0.192B^{36} - 0.269B^{48} - 0.311B^{60} + \dots) \times \frac{\epsilon_t}{1 - B^{12}}$$

In order to understand fully this combination of effects, which is negative at 12, 14 and 16 months and positive at 48 and 60 months, the four characteristics of measles should be recalled:

- (a) It is a seasonal disease appearing in March and disappearing in June;
- (b) It is infectious;
- (c) It is an immunizing disease;
- (d) In Bamako, the disease is most lethal in the 6-to-36-months age group: the rate of mortality (average for both sexes, 1974-1985) amounts



to 10.3 per thousand at 0 years of age, 15.7 for 1-year-olds, 11.0 for 2-year-olds, 5.9 for 3-year-olds and 2.3 for 4-year-old children (Fargues and Nassour, 1988).

Let us start with a major outbreak occurring in a given month,  $m$ . All children over six months of age (measles rarely affects younger children) catch the disease. Those who do not die of it are immune for life. The next year, when the measles season comes around again (at  $m + 12$ ), those children under six months of age at  $m$ —that is to say, the group of children under 18 months of age at  $m + 12$ —are practically the only ones susceptible to it. But at that age the child does not move freely and only comes in contact with its elders in the family, all of whom belong to the immunized generations. The measles spreads with difficulty, and the epidemic is limited. By the same token, immunity against the disease is not widespread.

The next year during the measles months ( $m + 24$ ) the group of unimmunized children consists of those under 30 months of age. Here, the mechanism described above still prevails, although less effectively, and limits the epidemic.

When the measles season comes again for the fourth time (at  $m + 48$ ), the poorly immunized group extends to all children under 60 months of age. It therefore covers the entire high-risk age group (6-48 months). The children have by then become sufficiently mobile to be contaminated outside the home. The time is ripe for another great outbreak.

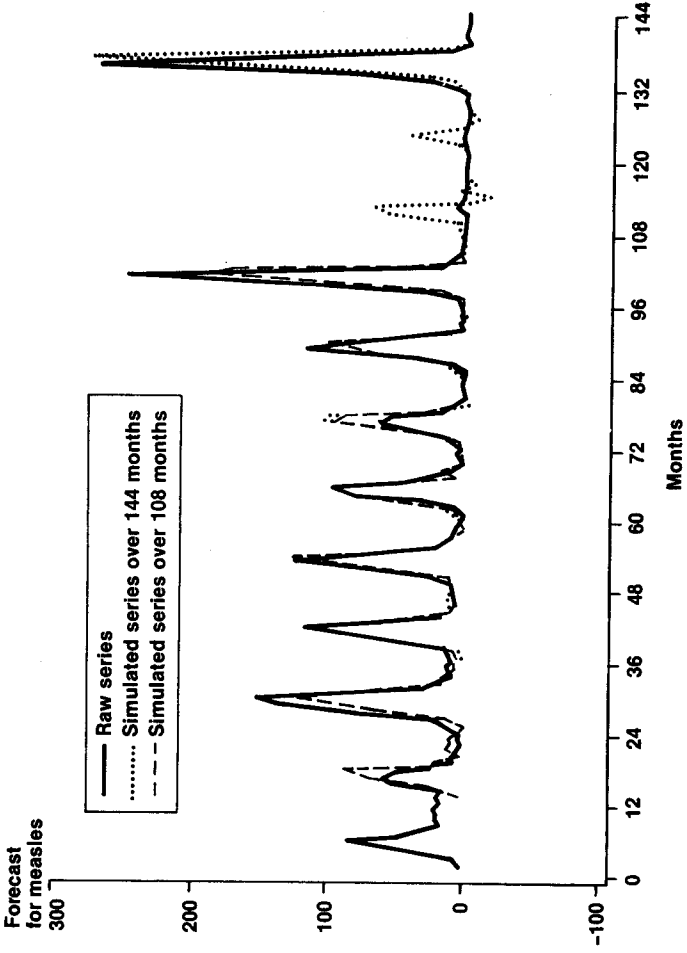
In the raw series shown in the graph of death from measles, two distinct periods stand out. From January 1974 ( $t = 1$ ) to December 1982 ( $t = 108$ ), the outbreak occurs yearly. From January 1983 to December 1985, it becomes triennial. After the 1982 epidemic, it was decided to launch an immunization campaign which broke the "natural" cycle of the disease. While controlling the epidemic for two years in a row, the campaign nevertheless blocked the transmission of measles and its resulting natural immunization. The immune population was thus gradually restricted to those who had been vaccinated. The extent of the last epidemic (1985) was due to the exceptional number of people who were entering the measles season without immunization. If we take into account only the first 108 months, the ARIMA results confirm the perfectly seasonal aspect of measles (cycle of 12 months:  $(1 - B^{12})$ ), with attenuated effects at 12 and 24 months. Immunization thus enhances the explosiveness of the next outbreak:

$$(1 + \underset{(0.156)}{0.511 B^{12}})(1 + \underset{(0.201)}{0.391 B^{24}})(1 - \underset{(0.080)}{0.648 B})measl_t = \frac{\epsilon_t}{1 - B^{12}}$$

which is very close to the previous equation:

$$(1 + \underset{(0.090)}{0.705 B^{12}})(1 + \underset{(0.126)}{0.814 B^{24}})(1 - \underset{(0.138)}{0.382 B^{36}})(1 - \underset{(0.076)}{0.599 B})measl_t = \\ = \frac{\epsilon_t}{1 - B^{12}}$$

Figure II. Deaths from measles



(The raw data curve and the two model curves are shown in fig. II.)

In table 4, we have gathered the results obtained through this type of statistical processing for other causes of death.

Meningitis (fig. III), despite its very different epidemiology, is well described by a comparable equation, as are those deaths caused by dehydration (fig. IV), whose link with measles we will examine below. Both causes of death are characterized by their strong annual seasonality to which is added a biannual cycle. The death cause reported as abdominal pain shows strong annual seasonality. Causes of death less characterized by their seasonal aspect may also be processed by the ARIMA techniques, but they yield little information. This is the case with diseases such as tuberculosis, which cause deaths without seasonal characteristics even if they have been contracted during a favourable season. This is due to random delays of several years, on average, between infection and death.

In other cases, such as head injury, the correlations are difficult to interpret.

TABLE 4. ARIMA MODEL OF CERTAIN SPECIFIC CAUSES OF DEATH

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meningitis:	
$(1+0.659 B^{12})(1+0.483 B^{24})(1-0.209 B)menin_t = \frac{\epsilon_t}{1-B^{12}}$	
(0.071)	(0.089) (0.086)
dehydration:	
$(1+0.608 B^{12})(1+0.281 B^{24})(1-0.337 B-0.236 B^2)dehy_t = \frac{\epsilon_t}{1-B^{12}}$	
(0.088)	(0.108) (0.087) (0.087)
abdominal pain:	
$(1-0.2235 B^3-0.252 B^4)(1+0.279 B^{12})abdo_t = -0.781 + \frac{\epsilon_t}{1-B^{12}}$	
(0.084)	(0.084) (0.086) (0.299)
tuberculosis:	
$(1-0.195 B^{24})tuber_t = 5.85 + \epsilon_t$	(0.288)
hypertension:	
$(1-0.298 B^2)hyper_t = 3.041 + \epsilon_t$	(0.181)
asthma:	
$(1-0.208 B^1)asthm_t = 1.452 + \epsilon_t$	(0.114)
lung disease:	
$(1-0.185 B^1-0.290 B^2)pneumo_t = 7.160 + \epsilon_t$	(0.546)
(0.081)	(0.081)
septicaemia:	
$(1-0.243 B^1)septi_t = 1.096 + \epsilon_t$	(0.109)
cranial traumatism:	
$(1-0.21 B^5-0.218 B^7)cran_t = 3.42 + \epsilon_t$	(0.262)
(0.081)	(0.082)
heart disease:	
$(1-0.252 B^1)cardi_t = 7.84 + \epsilon_t$	(0.318)
(0.081)	

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Figure III. Deaths from meningitis

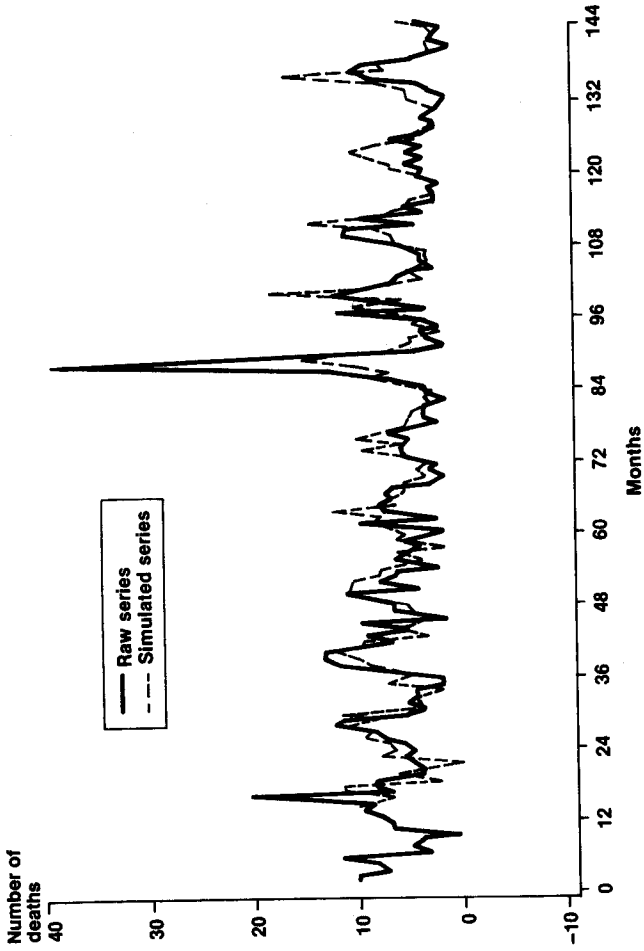
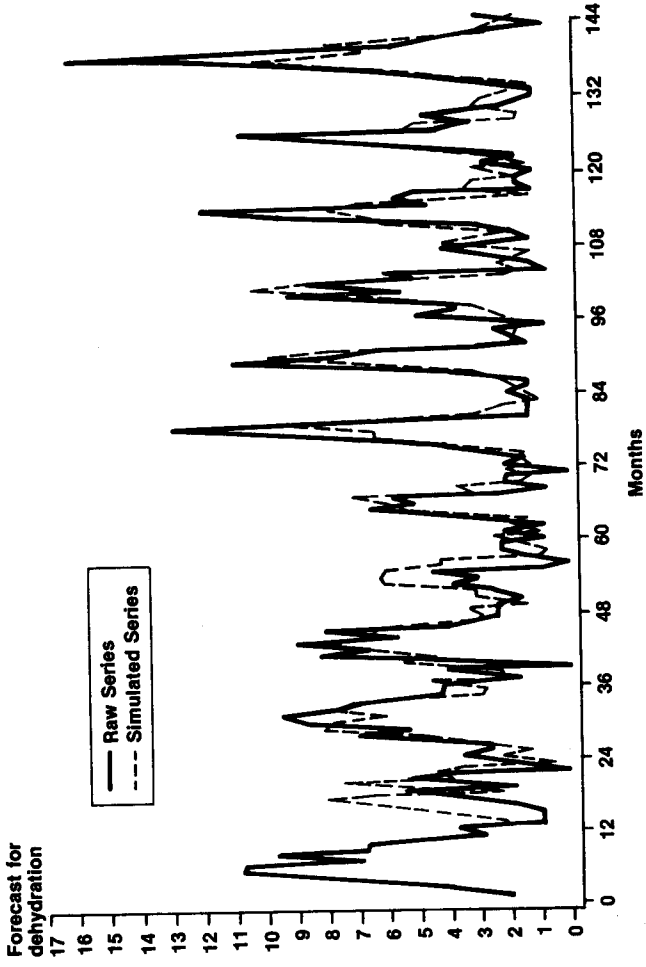


Figure IV. Deaths from dehydration



### *Systems of interrelated causes of death*

The causes of death may be interrelated when a disease,  $D_1$ , appears as a frequent complication of disease  $D_2$  or the number of deaths due to cause  $D_1$  follows the course of disease  $D_2$ . The response of  $D_1$  to  $D_2$  may occur with or without delay, depending on the diseases involved. It is also possible that the longer the reaction is delayed for  $D_1$ , the more it extends over time. It is quite likely that time series allow the detection of this type of interdependence only when the response from  $D_1$  to  $D_2$  is rapid. Liver complications due to malaria, for example, occur generally at too slow a rate to be translated into coincidences among time series.

We may also be dealing with two causes of death, with the "principal" cause (for example, measles) bringing about the "immediate" cause of death (diarrhoea), which is frequently associated with the same disease.

Causes of death of different kinds may interfere with one another, either by affecting the same population group (malnutrition/infection synergy) or by affecting two different sub-populations (mothers and newly born children, in death in childbirth and hypoxia). The last two cases lead us back to the complex problems raised by death by multiple causes (Nam, 1988).

A fictitious interdependence links two causes of death when the same illness is reported indiscriminately under various categories (icterus and hepatitis, for example).

Whereas "classic" descriptive models of demographic analysis postulate the probable independence of causes of death in order to isolate the effects inherent in each cause, we are attempting to restore the interdependence, free of any seasonal effects occurring simultaneously.

Let us now attempt to test a system well known to epidemiologists, the measles complex, in which the following causes are associated:

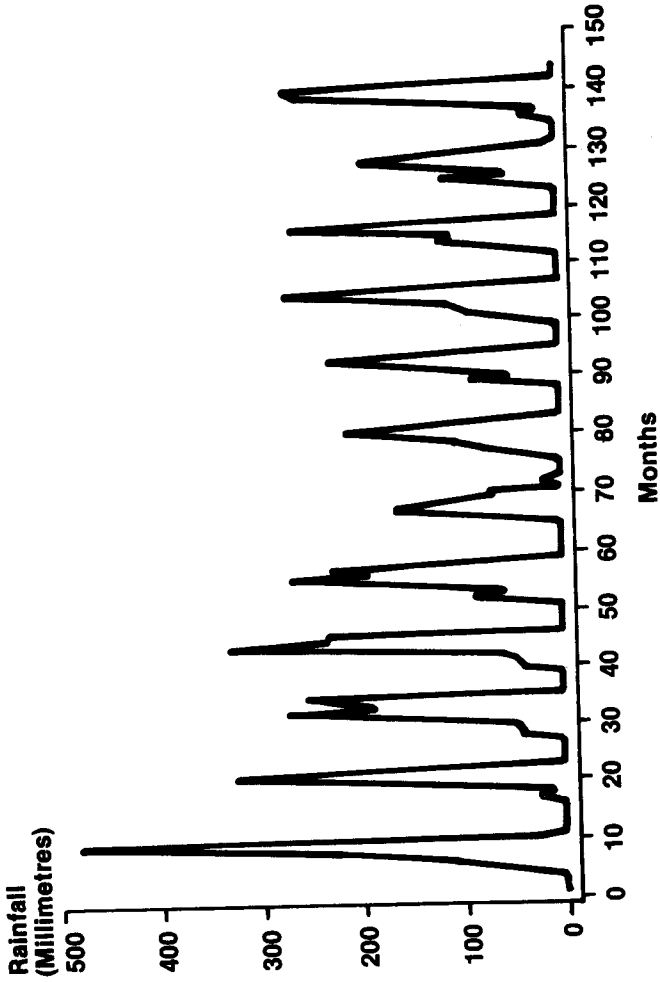
(a) Malnutrition, thought to act synergistically with infectious diseases (Mosley and Chen, 1984), in the particularly virulent form it takes in the measles age group: kwashiorkor;

(b) Dehydration and diarrhoea (intestinal infections), frequently linked to measles as a principal cause;

(c) Rainfall (see fig. V), an exogenous climatic factor likely to influence kwashiorkor in particular.

In order to test the malnutrition/infection synergy (measles or diarrhoea), we would have to observe the presence or absence in the same individual of malnutrition and death by infectious disease—that is to say, we would have to know the nutritional condition of the deceased. Like death records everywhere, those of Bamako do not mention nutritional conditions. This makes it necessary to fall back on an indirect approach. The presence or extent of malnutrition in the population is reflected by deaths of which the reported cause is malnutrition. If the record of death by malnutrition is implicated (in the statistical sense) with that of death by infection, we then have an indication that malnutrition is indeed a factor that promotes or aggravates infection.

Figure V. Rainfall recorded at Bamako



While investigating the effects of malnutrition on infections, no order of temporal priority is postulated *a priori* between the two causes of death. They may occur simultaneously or at different times: either with the expected priority of cause on effect, with death by infection presumed to be a delayed reaction to malnutrition; or, inversely, with premature effect, where malnutrition acting alone would lead to death more slowly than the infection it promotes. Among the sub-population suffering from malnutrition, only those who did not have an infection would die of malnutrition.

Here is the system we arrived at (adjusted series of seasonal variations):

$$\left\{ \begin{array}{l} \text{measl}_t = 0.611 \text{measl}_{t-1} + 0.669 \text{diarrh}_t + \epsilon_{1,t} \\ \quad \quad \quad (0.062) \quad \quad \quad (0.153) \\ \text{diarrh}_t = 5.814 + 0.463 \text{diarrh}_{t-1} + 0.707 \text{dehy}_t + \epsilon_{2,t} \\ \quad \quad \quad (1.614) \quad (0.073) \quad \quad \quad (0.258) \\ \text{kwash}_t = 0.483 \text{kwash}_{t-1} + 0.033 \text{diarrh}_t + 0.008 \text{rain}_t + \epsilon_{3,t} \\ \quad \quad \quad (0.072) \quad \quad \quad (0.016) \quad \quad \quad (0.003) \\ \text{dehy}_t = 1.724 + 0.404 \text{dehy}_{t-1} + 0.047 \text{diarrh}_t + \epsilon_{4,t} \\ \quad \quad \quad (0.451) \quad (0.076) \quad \quad \quad (0.021) \end{array} \right.$$

First of all, a positive effect at time  $t - 1$  is noted, whatever the cause. This shows the epidemic nature of the disease itself (measles) or of the diseases with which that cause may be associated (diarrhoea, dehydration, kwashiorkor), each epidemic extending over several consecutive months. One then observes the presence of diarrhoea in 'all equations: measles, kwashiorkor and dehydration are thus all able to assume the form of a diarrhoeal disease. And finally, some symmetry is observed between diarrhoea and dehydration: the contribution of deaths by dehydration to deaths by diarrhoea (70.7 per cent of deaths by dehydration) is of the same absolute order of magnitude as the contribution (4.7 per cent) of deaths by diarrhoea to deaths by dehydration. Furthermore, a constant attests to the presence of the disease throughout the year, independent of seasonal factors.

On the other hand, there is no evidence of malnutrition in deaths due to measles, diarrhoea or dehydration. Thus, no statistical evidence exists of the effects which malnutrition would have in promoting or aggravating the infection.

Using the same techniques, let us examine the relations between a death occurring during childbirth (maternal mortality) and death of the newborn due to low birth weight or hypoxia (still birth).

#### *Maternal mortality and hypoxia*

Corrected for seasonal variations, these two causes of death are linked by an econometric equation:

$$\left\{ \begin{array}{l} \text{Maternal mortality} = 1.293 + 0.147 \text{hypox}_t + \epsilon_t \\ \quad \quad \quad (0.204) \quad (0.080) \\ \epsilon_t = 0.246 \epsilon_{t-1} + \eta_t \\ \quad \quad \quad (0.081) \end{array} \right.$$

Since both variables introduced are of the same degree—namely, the number of deaths—the coefficient 0.147 may be interpreted to mean that



14.7 per cent of the deaths by hypoxia in the newborn are accompanied by the mother's death in childbirth.

*Prematurity, maternal mortality and still birth*

In the equations below, *B* stands for births, *stil* for still birth, *prema* for death due to prematurity, *mater* for the death of women aged 15-45 years of age from all causes (maternal mortality being confused with other causes of death). We are trying, *a priori*, to link time variables by a system of correlations, after adjustment for seasonal variations. The final econometric model of the simultaneous equations obtained is:

$$\left. \begin{aligned}
 \text{mater}_t &= 7.993 + 0.175 \text{mater}_{t-1} + 0.225 \text{mater}_{t-2} + \\
 &\quad (1.434) \quad (0.078) \quad (0.078) \\
 &\quad + 248.99 \text{prema}_{t-2}/B_{t-2} + \epsilon_1 \\
 &\quad (50.798) \\
 \nabla \text{stil}_t/B_t &= 0.556 \nabla \text{stil}_{t-1}/B_{t-1} + 0.292 \nabla \text{stil}_{t-2}/B_{t-2} + \\
 &\quad (0.076) \quad (0.074) \\
 &\quad + 0.258 \nabla \text{prema}_{t-2}/B \\
 &\quad (0.082) \\
 \nabla \text{prema}_t/B_t &= 0.631 \nabla \text{prema}_{t-2}/B_{t-2} + 0.178 \nabla \text{stil}_{t-1}/B_{t-1} + \epsilon_{3,t} \\
 &\quad (0.033) \quad (0.063)
 \end{aligned} \right\}$$

in which the  $(\epsilon_{i,t})$  are not correlated. According to their respective correlation charts, the variables *stil<sub>t</sub>/B<sub>t</sub>* and *prema<sub>t</sub>/B<sub>t</sub>* display a marked drift, which we have removed by differentiation. Each equation brings into play an auto-regressive form of the order of 1 and 2, which indicates marked inertia from one month to the next.

Prematurity and still birth interact. Still births in a given month are proportionate to the number of deaths due to prematurity occurring two months before. This may mean that a single factor may cause either a still birth at the normal time or premature birth (two months before term) followed by death. The fact that death in the newborn due to prematurity has an effect on maternal mortality with a delay of two months simply reflects the simultaneity of maternal mortality and still birth.

A SPECIAL SYSTEM: MEASLES IN DIFFERENT AGE AND SEX CATEGORIES

Time-series analysis enables us to test time correlations between deaths from the same disease in different sex and age groups.

Recent work has highlighted the anthropological dimension of the epidemiology of measles (Aaby, Bush and Hoff, 1986; Aaby, 1987). The degree of lethality of the disease appears to be commensurate with the duration of contagious contact. On the average, the disease develops into a more severe form on the average in children who contracted it at home than in the individuals who transmitted it after catching it elsewhere. Consequently, sex and age become possible factors of differentiation. Within the same family, children who do not leave the home will catch the disease from those who are in contact with the streets. Regardless of sex, children under one year of age have no freedom of movement. As a result, we can

expect that they will contract measles later in the season and in a more lethal form than their elders. Girls of the same age go out of the home less often than their brothers and thus are likely to catch measles later and in a more lethal form than boys.

In order to test these hypotheses, it would be necessary to know the relationships between persons whose deaths are recorded in the registers. Unfortunately, that information does not appear. As was the case for the malnutrition/infection synergy, this lack of information requires us to employ an indirect approach: we will surmise that sub-population  $P_1$  transmits the measles to sub-population  $P_2$  if the death rate due to measles in  $P_2$  occurs later (to allow for the incubation period) than in  $P_1$ . Moreover, we will suspect aggravation upon transmission if variations in the incidence of measles increase from  $P_1$  to  $P_2$ .

We have separated each sex into three age groups: 0 years of age; 1-4 years; and 5 years and over—or six series in all, which we may combine as we wish, with care to avoid multicollinearity.

Statistical comparisons show that the only significant relation appears to be between series of deaths among boys over 1 year old and boys 0-1 year old and girls of all ages (see fig. VI). All other tests among other combinations in these series do not permit the rejection of the hypothesis of a null regression coefficient. We therefore note:

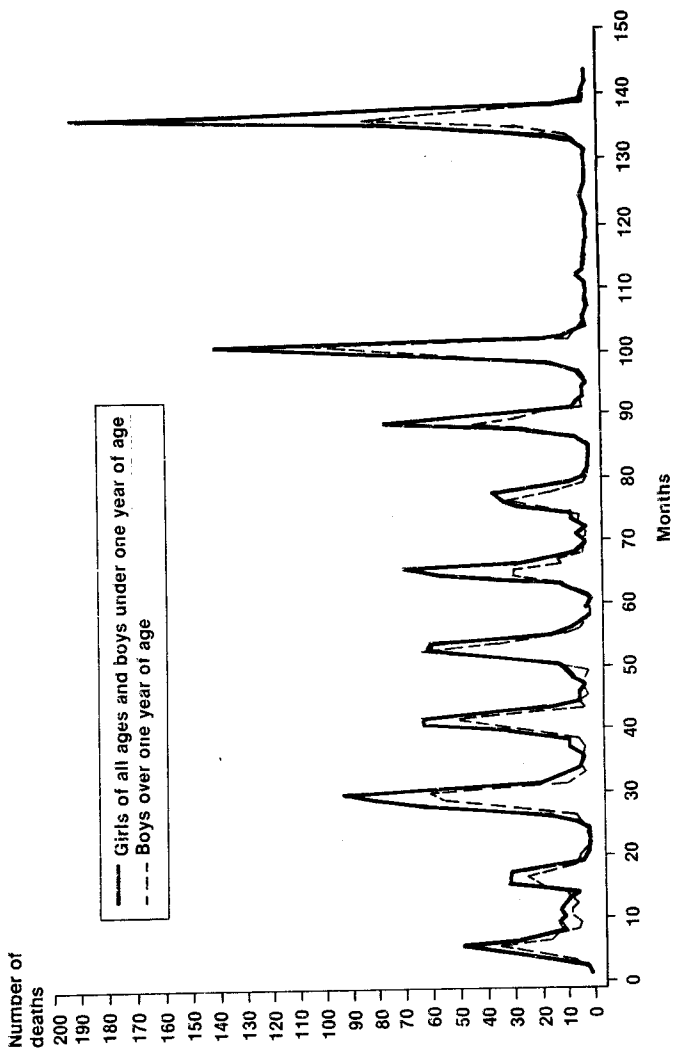
$$\begin{aligned}
 & (measlfem + measlmasc_t^{(0-1yr)}) - (1.527 + 0.178 B^1) measlmasc_t^{(>1yr)} = \\
 & \qquad \qquad \qquad (0.075) \qquad (0.074) \\
 & \qquad \epsilon_t \\
 & = \frac{\epsilon_t}{(1 - B^{12})(1 - 0.306 B^1)(1 + 0.881 B^{12})(1 + 0.615 B^{24})} \\
 & \qquad \qquad \qquad (0.087) \qquad (0.057) \qquad (0.104)
 \end{aligned}$$

In terms of causality, we have shown significant precedence in deaths due to measles affecting boys over 1 year old as compared to girls of any age and boys under 1 year of age. We also know that the male death rate due to measles (7.9 per thousand at 1-4 years) is inferior to the female rate (9.4 per thousand) (Fargues and Nassour, 1988). These results seem to confirm aggravation of the disease upon transmission from boy to girl. We may go even further: girls and boys under 1 year of age are proportionately less vulnerable to measles to the extent that their elder siblings were more vulnerable to it 12-24 months before. During a given season, boys under 1 year of age and girls of the same family are less at risk for measles to the extent that their elder siblings who have more contact with the outside world are immunized by having contracted measles during a preceding year.

#### *Causes of death correlated to exogenous factors—ecological or economic*

Infectious diseases in which the pathogenic agent appears under certain non-permanent climatic conditions (humidity, air temperature) may be said to belong to the exogenous group. A similar link exists when some causes of death linked to nutrition react to fluctuating market prices.

Figure VI. Deaths from measles in boys over 1 year of age and of girls of all ages and boys under 1 year of age



We have at our disposal the time series of prices of the "African" food market in Bamako.<sup>2</sup> It is a rather irregular series, with a marked rising non-linear trend. Prices show a rather clear average seasonal profile, with a peak at the beginning of the rainy season (July) (see fig. VII), when the new crops have not yet become available. This peak coincides with that of deaths due to malnutrition; consequently, it would be tempting to see a direct effect of food prices on the nutritional state of the population. However, when we go from a comparison of average seasonal profiles to analysing the history, the relation loses significance. From one year to the next, the peak of deaths due to malnutrition always recurs in July while that of the prices moves around its July average (see fig. VIII).

None of our attempts to link either the price, its logarithm or its Box and Cox transformation, or again the spread of prices in relation to a non-linear trend, to the causes of death at our disposal has enabled us to reject a null hypothesis. The data would thus indicate an absence of causality between deaths related to malnutrition and the price of food. The prices reflect a quantitative relationship between food supply and demand, while malnutrition reflects a qualitative worsening of nutrition. Regardless of demand, such a deterioration occurs regularly during seasons when agriculture provides only a small variety of products.

Only rainfall, which does not show a regular series, seems to influence these causes of death. Deaths caused by malnutrition are markedly seasonal (see fig. IX) and are dependent upon the level of rainfall:

$$\begin{aligned} (1 - 0.1895 B^1 - 0.238 B^2)(1 + 0.369 B^{12})(malnu_t - 0.017rain_t) = \\ \begin{matrix} (0.205) & (0.0024) & (0.084) & (0.006) \end{matrix} \\ = \frac{\epsilon_t}{1 - B^{12}} \end{aligned}$$

Thus, the instantaneous effect of rainfall on malnutrition can be identified: it is during the rainy season that children die of malnutrition; it is the overlapping season in the countryside which provides food for the Bamako market. We do not see any effects brought about by drought or abundant rainfall in the more distant past.

Kwashiorkor, a particular form of malnutrition at weaning, follows a very similar model (see fig. X) and shows the same pattern of dependence with regard to rainfall:

$$\begin{aligned} (1 - 0.237 B^1)(1 + 0.441 B^{12})(kwash_t - 0.013rain_t) = \frac{\epsilon_t}{1 - B^{12}} \\ \begin{matrix} (0.089) & (0.081) & (0.0037) \end{matrix} \end{aligned}$$

Diarrhoea (fig. XI) is linked to rainfall, as we have already observed, in the measles/diarrhoea/malnutrition system:

$$\begin{aligned} (1 - 0.310 B)(1 + 0.524 B^{12})(1 + 0.298 B^{24})(diarrh_t - 0.929 B^3rain_t) = \\ \begin{matrix} (0.085) & (0.090) & (0.102) & (0.354) \end{matrix} \\ = \frac{\epsilon_t}{1 - B^{12}} \end{aligned}$$

Figure VII. Comparative seasonality of malnutrition and price changes in terms of non-linear trend

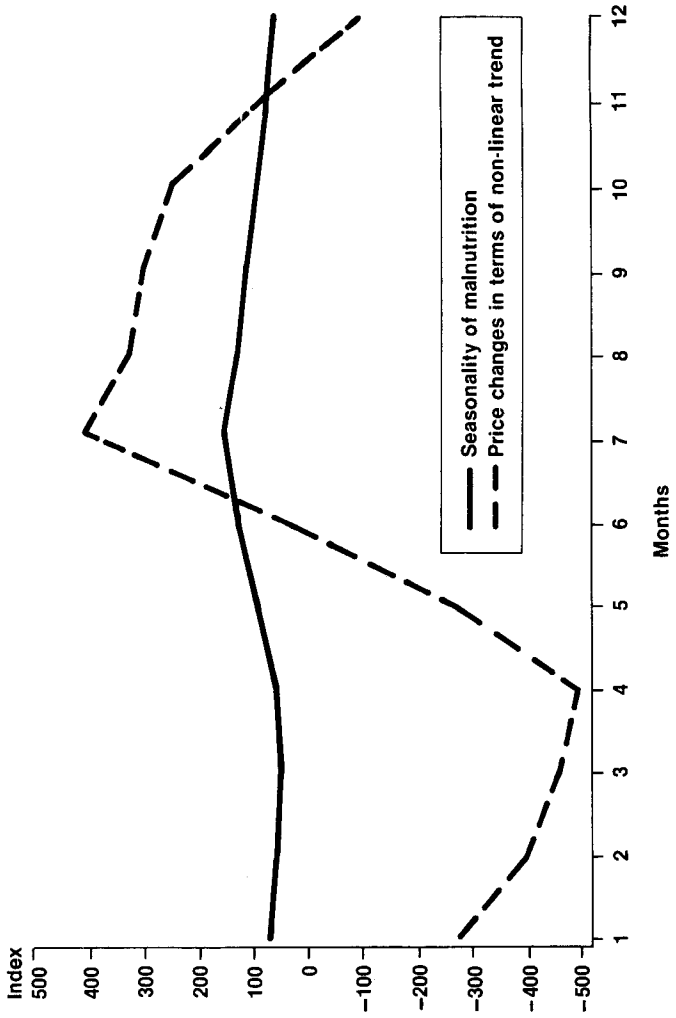


Figure VIII. Price of a market basket on the Bamako food market

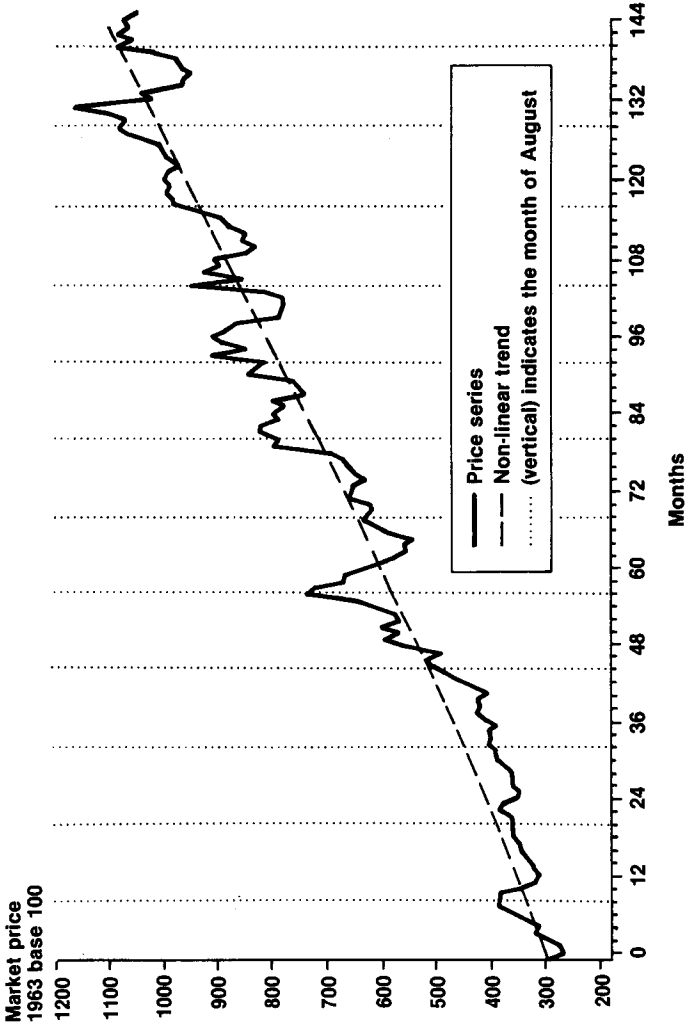


Figure IX. Deaths from malnutrition

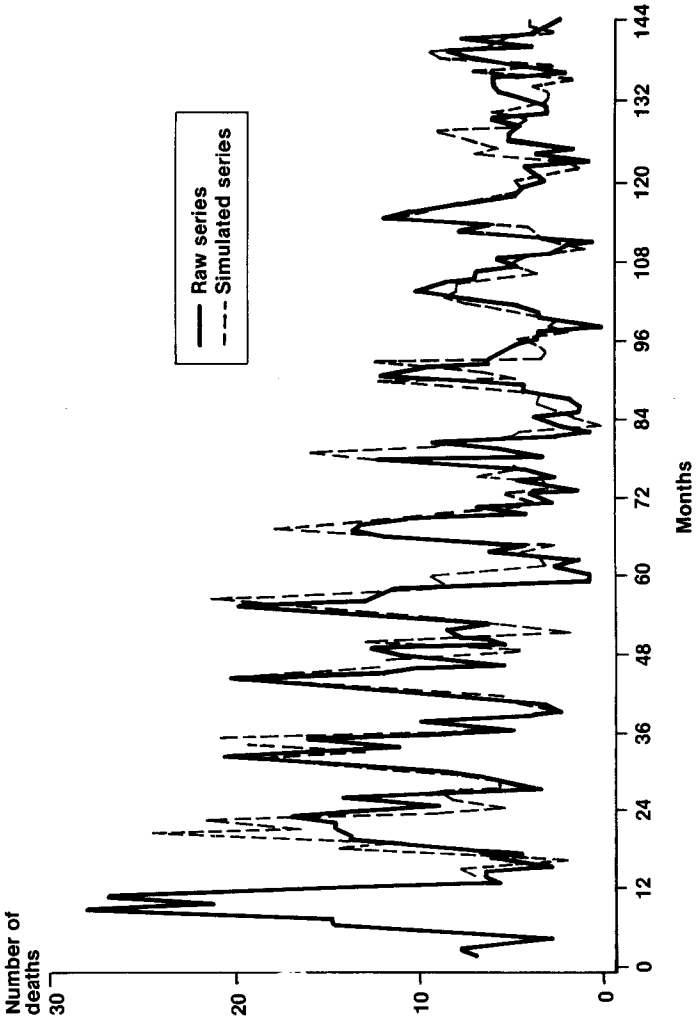


Figure X. Deaths from kwashiorkor

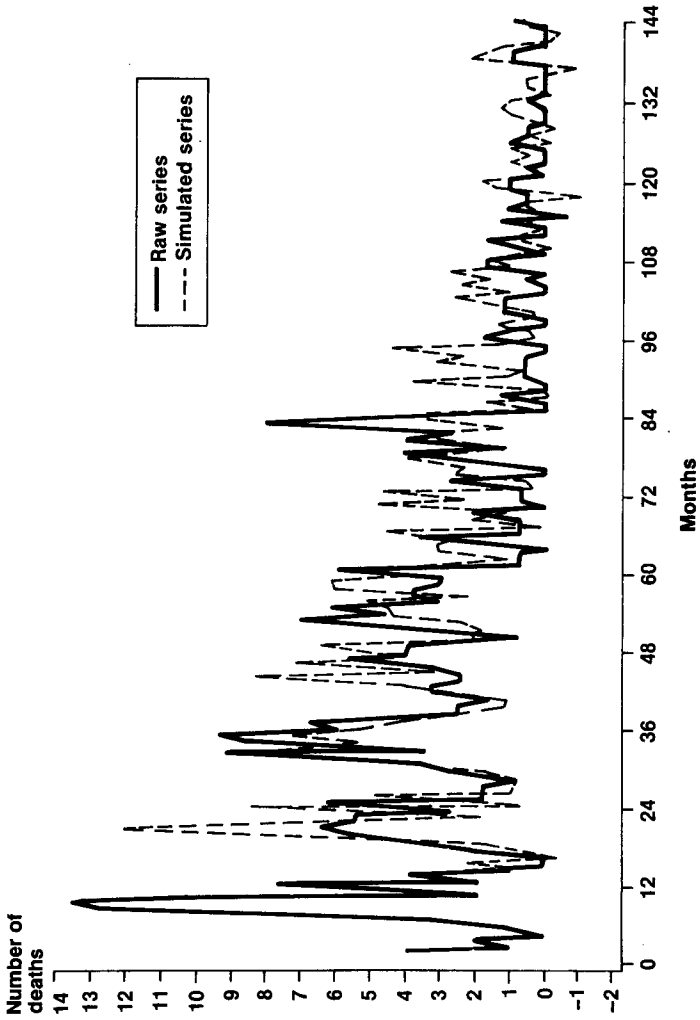
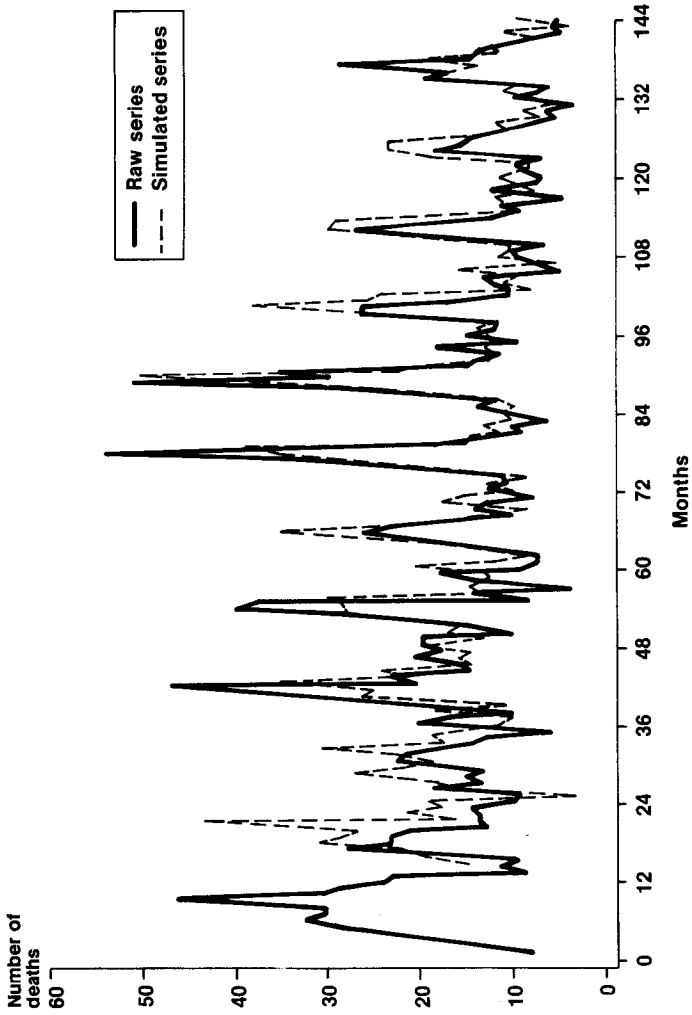




Figure XI. Deaths from diarrhoea



Deaths caused by renal deficiency tend to decrease during years of heavy rainfall: the abundance and quality of water could explain this.

$$ren'ins_t = 3.67 - 0.007B^1(1 - B^{12})rain + \frac{\epsilon_t}{(1 - 0.342B^4)} \quad (0.001)$$

Icterus

$$icter_t = 1.725 + (1 + 0.918B^{12} + 0.884B^{24} + \dots)(1 - B^{12})rain_t + \frac{\epsilon_t}{(1 - 0.277B^3)}$$

Rainfall would seem to promote icterus as a cause of death, with marked seasonal characteristics.

### *Malaria*

Malaria attacks and the fear they engender are a common feature of West African life. However, it is often difficult to distinguish genuine malaria from fevers of other origin, especially without careful medical diagnosis. Thus, deaths not caused by malaria are frequently classified under that name, and the reverse is also true. How can we detect real malaria, given such conditions?

Although methods derived from linear models are poorly adapted to time-series analysis, we must nevertheless call on them to deal with explanatory variables that we know to have an influence on malaria. We shall therefore abandon models of the Box and Jenkins type at this point and turn to econometric models. Our choice of model and explanatory variables and our handling of econometric tools are explained in detail in the annexes below. We note with a tilde ( $\sim$ ) the observed variables. Thus, deaths attributed to malaria are covered by two terms: deaths due to "genuine" malaria, or "*realmala*," and a group of other causes of death, erroneously attributed to malaria, which we will call "false" malaria, or "*falsemala*". Thus we note:

$$mala = realmala_1 + falsemala$$

With regard to genuine malaria, it is broken down into real malaria so reported ("*realmala*<sub>1</sub>") and real malaria reported under another term: "*realmala*<sub>2</sub>". Thus:

$$realmala = realmala_1 + realmala_2$$

Let us formulate a first hypothesis: only one other infectious disease may be confused with malaria. Since malaria is itself classified as an infectious disease, we can assume:

$$infecdis = realmala + falsemala + others$$

If we assume, as a second hypothesis, that "false malaria" constitutes—give or take a disturbance—a constant proportion of all infectious diseases other than real malaria, we can assume:

$$falsemala = k(infecdis - realmala) + \epsilon$$

with  $0 \leq k \leq 1$ .

As a result of these two hypotheses, we now start with the following system:

$$\begin{cases} mala &= realmala_1 + falsemala \\ infecdis &= realmala + falsemala + others \\ falsemala &= k(infecdis - realmala) + \epsilon \\ realmala &= realmala_1 + realmala_2 \end{cases}$$

In order to extract the variable *realmala*, unobserved, we eliminate *falsemala*:

$$\begin{cases} mala &= realmala_1 + k(infecdis - realmala) + \epsilon \\ (1 - k) infecdis &= (1 - k) realmala + others + \epsilon \end{cases}$$

As a third hypothesis, let us assume that real malaria, reported as such, remains a constant proportion, over time, of total real malaria, plus or minus an error factor:

$$realmala_1 = \gamma realmala + \eta$$

with  $0 \leq \gamma \leq 1$ .

Let us now assume that rain exerts an influence (Mashaal, 1986), without knowing the lags involved. For this reason, we assume an *a priori* model:

$$realmala = (\alpha_0 + \alpha_1 B + \dots + \alpha_h B^h) rain + \frac{\nu}{1 - B^{12}}$$

The econometric treatment is continued in annex II below.

Thus, for the malaria variable (cause of death so reported), we obtained:

$$\begin{aligned} mala &= 11.749 + 0.0134rain_t + 0.0178rain_{t-1} + 0.0161rain_{t-2} + \\ &+ 0.0110rain_{t-3} + 0.0054rain_{t-4} + 0.00222411rain_{t-5} + \\ &+ 0.00416049rain_{t-6} + 0.0140879rain_{t-7} + \\ &+ 0.0608infecdis_t + \epsilon_t \end{aligned}$$

with

$$\epsilon_t = -0.412\epsilon_{t-1} - 0.202\epsilon_{t-2} + \nu_t$$

where  $\nu_t$  is a "white noise" factor. Autocorrelations of a higher order are not significant.

We have estimated  $(\gamma - k)\alpha_i$ . But since  $k$  is weak before 1, and it may be assumed that some proportion of malaria greater than this  $k$  is real malaria, we have  $(\widehat{\gamma - k}) > 0$ . Assuming that the estimators of  $\gamma - k$  and  $\alpha_i$  are identifiable, the estimator  $(\gamma - k)\alpha_i$  may be decomposed into  $(\gamma - k)\hat{\alpha}_i$ , we obtain an estimate of  $\alpha_i$  give or take some positive factor, and thus the order of significance of rainfall's temporal effects upon real malaria.

Our conclusions are as follows. Death due to malaria is related positively to the volume of rainfall in the current month and in the seven preceding months. Rainfall reaches maximum influence with a lag of one (0.0178) or two (0.0161) months. At present we do not know whether such lags in fact correspond to a combination between the reproductive cycle of the mosquito, the incubation time of the disease (from 9 to 14 days for *M. falciparum* (Aaby, 1987)) and the time between the onset of the disease and death. A high constant (11.749) points to the endemic nature of malaria in Bamako.

Age-specific functions show a more pronounced peak during the dry season for age group 0-1 than for all others. That age group therefore seems to conceal more false malaria than the others. This leads us to investigate malaria according to age.

Using the same econometric process for each age group in order to extract real malaria cases, we obtain:

*Children under 1 year of age:*

$$\begin{aligned} mala_0 = & 2.218 + 0.0038 rain_t + 0.0053 rain_{t-1} + 0.0052 rain_{t-2} + \\ & + 0.0041 rain_{t-3} + 0.0027 rain_{t-4} + 0.0015 rain_{t-5} + \\ & + 0.0013 rain_{t-6} + 0.0026 rain_{t-7} + 0.0060 rain_{t-8} + \\ & + 0.0123 rain_{t-9} + 0.0174 infecdis + \epsilon_t \end{aligned}$$

with

$$\epsilon_t = -0.336\epsilon_{t-1} + \nu_t$$

and  $\nu_t$  is "white noise".

*Children in age groups 1-4 years:*

$$\begin{aligned} mala_{1-4} = & 0.0477 + 0.0067 rain + 0.0106 rain_{t-1} + 0.0121 rain_{t-2} + \\ & + 0.0017 rain_{t-3} + 0.0101 rain_{t-4} + 0.0078 rain_{t-5} + \\ & + 0.0054 rain_{t-6} + 0.0033 rain_{t-7} + 0.0021 rain_{t-8} + \\ & + 0.0024 rain_{t-9} + 0.0047 rain_{t-10} + 0.0096 rain_{t-11} + \\ & + 0.0431 infecdis + \epsilon_t \end{aligned}$$

with

$$\epsilon_t = -0.351\epsilon_{t-1} + \nu_t$$

and  $\nu_t$  is "white noise".

*Persons 5 or more years of age:*

$$\begin{aligned} mala_5 = & 3.5037 + 0.0017 rain + 0.0027 rain_{t-1} + 0.0031 rain_{t-2} + \\ & + 0.0030 rain_{t-3} + 0.0026 rain_{t-4} + 0.0020 rain_{t-5} + \\ & + 0.0013 rain_{t-6} + 0.00068 rain_{t-7} + 0.00023 rain_{t-8} + \\ & + 0.00011 rain_{t-9} + 0.00042 rain_{t-10} + 0.00132 rain_{t-11} + \\ & + 0.0029 rain_{t-12} + 0.0102 infecdis_t + \epsilon_t \end{aligned}$$

with

$$\epsilon_t = -0.251\epsilon_{t-1} + \nu_t$$

and  $\nu_t$  is "white noise".

Beyond the first birthday, the effect of rainfall two or three months earlier seems to predominate in mortality due to malaria, for any given month. Let us point out, however, two significant differences:

(a) A much weaker constant term for those 1-4 years than for those 5 years and over would indicate that chronic malaria sufferers are still rare among young children in Bamako;

(b) Higher coefficients attached to rainfall in the 1-4 age group confirm those results and show that in the early years malaria is mostly caused by recent rainfall.

In children under 1 year of age, two results seem at first surprising: a markedly higher constant, and the predominant effect of rainfall which occurred nine months before. The younger the infant, the rarer is death due to malaria, since the infant had no time to contract the disease. On the other hand, the seasonality could well be that of the effects of the mother's malaria on infant mortality: weakened at birth by a mother who is a chronic sufferer of malaria (high constant: 2.218) or who had an attack in the first months of pregnancy (0.01234 in  $t - 9$ ), the child may be more exposed to early death due to a fever erroneously reported as malaria. Thus, for children under 1 year of age, more deaths would be attributed to false malaria than after that age, but a proportion would be directly attributable to the real malaria of the mother.

#### CONCLUSIONS

Analysis has made it possible to formulate time series for the principal causes of death, either by reference to the past alone (ARIMA models), or by invoking exogenous factors for causes of death linked to climatic factors (temperature, rainfall) or economic factors (food prices), or by setting up a system among several series which may be "explained" (in Granger's sense) among themselves with possible lags.

This mathematical presentation enables us to make short-term predictions for some causes of death. The determinants of mortality are made up of factors with high or low inertia. The former—for example, the level of education, sanitary facilities, the nutritional habits prevailing in a community etc.—establish the average level of mortality, while the second determine its fluctuations. Modelling of fluctuations and past trends enables us here to approximate satisfactorily the graphic time-pattern of such diseases as measles and meningitis. The introduction of exogenous explanatory factors further improves short-term predictions.

Analysis of chronological data may throw new light on general questions regarding mortality. Thus, by observing that in Bamako girls caught measles later and in more lethal form than boys, we discovered a factor that emphasized the role played by transmission in the worsening of the

disease. Since we did not manage to find evidence of a direct presence of malnutrition in measles, even within the system connecting it to diarrhoea and dehydration, we would be inclined to agree with those who attribute the heavy mortality rate due to measles in Africa to contagious conditions rather than the underlying malnutrition.

Finally, in the particular case of malaria, we have suggested an econometric approach that eliminates erroneous reports of cause of death and make it possible to restore an explanatory, intertemporal relation between fluctuations in its time series and rainfall. Thus we have provided a means to detect real malaria and a tool for its short-term prevention.

## ANNEX I

### Statistical methods of time series: review

#### MODELLING OF A SINGLE VARIABLE TIME SERIES

The first operation consists in correcting, where necessary, the few extreme points which may appear in the raw series. That series, in turn, is converted to a stationary series if it is not already one. For this, the operator eliminates a first-degree tendential polynomial in  $t$ . In a more general way  $dX_t = (1 - B)^d X_t$  eliminates a degree  $t$  tendential polynomial. This transformation  $(1 - B^s)$  eliminates a period  $s$  seasonality. Thus, for monthly series such as those with which we shall work,  $(1 - B^{12})$  eliminates annual seasonality. If this last is not extracted by  $(1 - B^s)$ ,  $(1 - B^s)^s$ ,  $S > 1$  may apply. Finally, if an exponential trend is found, the  $X_t$  logarithm may be used, or its Box and Cox transformation  $\frac{X_t^\gamma}{\gamma}$ ,  $\gamma = 0$ .

Once the deviations, seasonal components or exponential trends are eliminated, the memory of the process may be described as a numerable amount of uncorrelated white noises—that is to say, a dynamic linear system  $\Psi$  subject to random shocks  $\epsilon_t$ :

$$X_t = \mu + \Psi(B)\epsilon_t$$

with

$$\mu = E(X_t) \text{ and } \Psi(B) = 1 + \Psi_1 B + \Psi_2 B^2 + \dots$$

In practice, most stationary processes may be approached through an auto-regressive moving average (ARMA) model—Wold decomposition—as follows:

$$\Phi(B)X_t = \Theta(B)\epsilon_t$$

with

$$\Phi(B) = 1 - \phi_1 B - \dots - \phi_p B^p \text{ and } \Theta(B) = 1 - \theta_1 B - \dots - \theta_q B^q$$

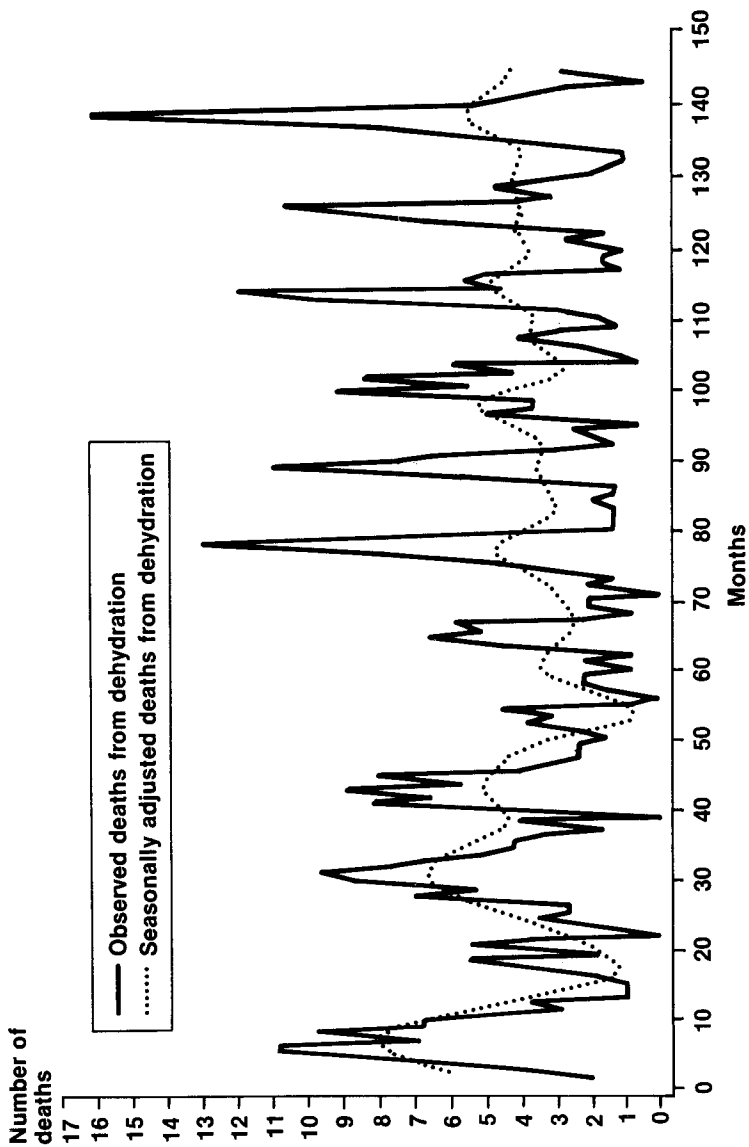
The parameters  $p$  and  $q$  are identified with the assistance of empirical autocorrelation and partial autocorrelation coefficients. Coefficients  $\phi_i$ ,  $\theta_i$  and variant  $\sigma_\epsilon^2$  of  $\epsilon_t$  are estimated by a maximum likelihood procedure under the hypothesis of a Gaussian  $\epsilon_t$  white noise. This last hypothesis is tested (a "Portmanteau" test) in order to validate or invalidate the proposed specification of the model.

We are almost always able to express the ARMA model as an auto-regressive (AR) alone.

#### *Deaths due to dehydration*

A survey of the series of deaths caused by dehydration, adjusted to the size of the present population during month  $t$ , (or  $(dehy_t)_{t \in \{1, 144\}}$ ) and of its graph (fig. XII) points to the existence of marked seasonality, with the highest levels occurring in the month of May. A more refined analysis made with CENSUS-X11, an American program, reveals a changing

Figure XII. Series of deaths from dehydration and deseasonalized series



seasonal profile from one year to the next. The general trend seems to be rising slightly after an uneven drop during the first 60 months.

The graphic presentation of the raw series shows that it cannot be thought to have been engendered by a stationary process. By applying sequentially the operators  $(1 - B)$ ,  $(1 - B^{12})$ ,  $(1 - B)(1 - B^{12})$ ,  $(1 - B^{12})^2$ , a transformed series is obtained whose graphic presentation would prompt us not to discard the hypothesis of stationarity for  $(1 - B^{12})X_t$  and  $(1 - B)(1 - B^{12})X_t$ . The inverted correlation function  $(1 - B)(1 - B^{12})X_t'$  behaves like that of a non-stationary process, which would indicate excess differentiation. We will henceforth use  $(1 - B^{12})X_t$  as a stationary series. ( $B$  is the lag function:  $BX_t = X_{t-1}, \dots, B^m X_t = X_{t-m}$ .)

We now identify parameters  $\theta_i$  and  $\phi_i$  on the basis of the partial relationship which leaves two significant values. We are thus led to propose and estimate the model

$$(1 - \phi_1 B - \phi_2 B^2) dehy_t = \frac{\epsilon_t}{1 - B^{12}}$$

At this exploratory level, the diagram of autocorrelations of residuals suggests that we are not dealing with a white noise. There remains a strong correlation to 12 and 24 lags, and the Portmanteau test leads us to reject this hypothesis.

Two models of the residuals are possible: an AR or an MA with lags of 12 and 14. It is estimated as follows:

$$(1 - \phi_1 B - \phi_2 B^2) dehy_t = (1 - \theta_{12} B^{12})(1 - \theta_{24} B^{24}) \frac{\epsilon_t}{1 - B^{12}}$$

or

$$(1 - \phi_1 B - \phi_2 B^2)(1 - \phi_{12} B^{12})(1 - \phi_{24} B^{24}) dehy_t = \frac{\epsilon_t}{1 - B^{12}}$$

resulting in an AR:

$$\underset{(0.085)}{(1 - 0.336 B} - \underset{(0.085)}{0.254 B^2)} dehy_t = \underset{(0.071)}{(1 - 0.665 B^{12})} \frac{\epsilon_t}{1 - B^{12}}$$

or an ARMA:

$$\underset{(0.088)}{(1 - 0.337 B} - \underset{(0.109)}{0.236 B^2})(1 + \underset{(0.087)}{0.608 B^{12}})(1 + \underset{(0.087)}{0.281 B^{24}}) dehy_t = \frac{\epsilon_t}{1 - B^{12}}$$

In both cases, the Portmanteau test validates the hypothesis that the white noise of residuals is less than 5 per cent.

As can immediately be seen, these expressions are almost equivalent: by dividing the two sides of the ARMA equation by  $1 - 0.665 B^{12}$ , one obtains the next AR model, which is very close to that which was obtained directly:

$$(1 - 0.336 B - 0.254 B^2)(1 + 0.665 B^{12})(1 + 0.442 B^{24} + \dots) dehy_t = \frac{\epsilon_t}{1 - B^{12}}$$

Since it is always possible to return to an AR model, which is in fact an AR  $\infty$  truncated at a finite distance, we shall express our findings in this way only, as it makes interpretation easier.

#### *Influence of one time-series on another*

##### *Vector auto-regression presentation (VAR)*

As long as the observer has at his disposal no information other than the history of the cause of death which he is investigating, auto-regressive integrated moving average (ARIMA) models enable him to quantify the memory of the process. But should he have additional information in the form of one or several time-series, he may seek the intertemporal correlations of all the series taken together without assuming an *a priori* structural model and



without establishing the maximum lag in advance. For that purpose, multidimensional autoregressive models give a measure of causal relations between those series, in the sense defined by Granger (Gourieroux and Monfort, 1983). We shall express the bi-dimensional stationary process  $(Y_t, Z_t)$  as:

$$\Phi(B)(Y_t, Z_t)' = (\epsilon_t, \nu_t)'$$

where  $\phi(B)$  is the matrix:

$$\Phi(B) = \begin{bmatrix} \Phi_Y(B) & \Phi_{YZ}(B) \\ \Phi_{ZY}(B) & \Phi_Z(B) \end{bmatrix} = \sum_{i=0}^p \Phi_i B^i$$

where  $\Phi_i(B)$  are  $2 \times 2$  square matrices. The process  $(\epsilon_t, \nu_t)'$  is white noise of the covariance matrix  $\Omega$ .

Student's and Fisher's tests make it possible to test the various hypotheses (Gourieroux and Monfort, 1983), as follows:

- (a) "Z does not cause Y" corresponds to the null hypothesis  $\Phi_{YZ}(B)$ ;
- (b) "Z and Y are independent" corresponds to the null hypothesis  $\Phi_{ZY}(B)$ ;
- (c) "Z and Y do not cause instantaneously" corresponds to  $cov(\epsilon_t, \nu_t) = 0$ .

The usefulness of this presentation is to identify (according to a certain criterion) the lags that should be taken into account, to reveal the  $\epsilon_t$  as pure innovations in an ARMA representation, which then indicates the underlying structure.

Once established, this framework enables us to reconstruct a more classic econometric model of simultaneous equations in which it is only necessary to estimate correctly the coefficients assigned to the different variables and their lags.

#### The transfer function

In the particular case in which Y does not cause Z, one can write a regression equation  $Y_t$  on Z, termed a "transfer function" model.

$$Y_t = \sum_{j=0}^{\infty} \alpha_j Z_{t-j} + \omega_t$$

The whole series  $\sum_{j=0}^{\infty} \alpha_j B^j$  may be approached by a rational fraction  $\omega(B)/\sigma(B)$  in which  $\omega$  and  $\sigma$  are polynomials; similarly, the stationary process  $(\omega_t)_t$  is approached by an ARMA process  $\omega_t = (\alpha(B)/\beta(B))\epsilon_t$  in which  $\alpha$  and  $\beta$  are polynomials and  $\epsilon$  white noise (Gourieroux and Montfort, 1983), so that

$$deh y_t = \frac{\omega(B)}{\sigma(B)} rain_t + \frac{\alpha(B)}{\beta(B)} \epsilon_t$$

Let us return to the example of dehydration. Knowing that a child who caught measles (principal cause) may die of dehydration (immediate cause), the field observer probes the time links that may exist between the two causes.

Having assembled their respective histories over a continuous period of 144 months and having corrected the seasonal variations, we have selected, according to Akaike's criterion (Akaike, 1976; see also Maurel, 1987, and Rabemananjara, 1986, the following multidimensional auto-regressive mode:

$$\begin{cases} measl = 2.156 deh y_t - 1 + 0.590 measl_{t-1} + \epsilon_{1,t} \\ \quad (1.110) \quad \quad \quad (0.067) \\ deh y_t = 0.650 deh y_t - 1 + 0.010 measl_{t-2} + \epsilon_{2,t} \\ \quad (0.110) \quad \quad \quad (0.003) \end{cases}$$

in which the variance-covariance error term matrix is equal to

$$\Omega = \begin{bmatrix} 2.81 & 4.15 \\ 4.15 & 701.73 \end{bmatrix}$$

We reject the hypothesis that  $measl_t$  and  $dehy_t$  are "not caused instantaneously", and we will test their instantaneous relation directly through the linear econometric model of simultaneous equations which enables us to free ourselves of the average mobile part of VAR.

The VAR may be reduced to its matrix form:

$$\begin{pmatrix} 1 - 0.590 B & -2.156 B \\ -0.010 B^2 & 1 - 0.650 B \end{pmatrix} \begin{pmatrix} dehy_t \\ measl_t \end{pmatrix} = \begin{pmatrix} \nu_t \\ \epsilon_t \end{pmatrix}$$

Along this same line, let us seek the various factors comprised in a Granger-causality system with the series of deaths caused by dehydration and with that of deaths caused by measles.

We may be dealing with other causes of death, but also with such phenomena as rainfall and the price index. Causality should go in a one-way direction from the level of rainfall to dehydration. In the single variable model, the "transfer function" never yields a significant coefficient of the polynomial in  $B$  of rainfall: the level of precipitation, on which the available volume of water would depend, does not affect dehydration in the least in Bamako.

We might just as well have introduced rainfall as an exogenous factor in the measles/dehydration system which we set out at length above. If we assume the absence of "causality" of the causes of death towards rainfall, we arrive at the following system:

$$\begin{aligned} measl_t &= 2.281 dehy_{t-1} + 0.593 measl_{t-1} + \epsilon_{1,t} \\ &\quad (1.120) \qquad\qquad (0.067) \\ dehy_t &= 0.645 dehy_{t-1} + 0.010 measl_{t-2} + \epsilon_{2,t} \\ &\quad (0.111) \qquad\qquad (0.003) \\ rain_t &= 0.178 rain_{t-1} + \epsilon_{3,t} \\ &\quad (0.08) \end{aligned}$$

with the variance covariance matrix

$$\Omega = \begin{pmatrix} 702.14 & 4.19 & -42.09 \\ 4.19 & 2.857 & 0.578 \\ -42.09 & 0.578 & 1647.76 \end{pmatrix}$$

When testing the correlations of residuals we are able to accept, at the 5 per cent level, the "non-causality" hypothesis for the relationship between rainfall and measles. Moreover, there is no evidence of a 12-month rainfall lag variable: heavy rainfall in a given year may contribute to the volume of the available drinking water reserves, but it does not modify the mortality due to measles and dehydration during the next dry season. (It should be noted that this does not imply that the arrival of the rainy season does not coincide with a drop in measles.)

If we form the same system, but without imposing the exogenousness of rainfall in relation to the other variables, we are able to test whether variable causes of death enter significantly into the equation on rain. That is the case, and this demonstrates the possible presence of other delayed variables omitted in the analysis. By testing, from within the VAR system, the exogenousness of a variable which we know cannot be anything but exogenous, we in fact validate or invalidate the completeness of the model specified at the outset (see Sims, 1972).

## ANNEX II

### Econometric treatment of equations relating to malaria

Our purpose is to find the lag  $h$  and significant coefficients  $\alpha_i$ , given our hypotheses. For this purpose, we use the regression equation:

$$mala = \dots (\gamma - k)(\alpha_0 + \alpha_1 B + \dots + \alpha_h B^h) \bar{rain} + k \inf \bar{cdis} + \epsilon'$$

with

$$\epsilon' = (\gamma - k) \frac{\nu}{1 - B^{12}} + \epsilon + \eta$$

We shall attempt to estimate  $\gamma$ ,  $\alpha_1$ ,  $k$ ,  $h$ .

The difficulty lies in the marked multicollinearity of the explanatory variables. As a first step, we shall use the Frisch-Waugh theorem, with  $M$  as projector on the  $Vect(infecdis)$  orthogonal.

$$Mmala = (\gamma - k)M(\alpha_0 + \alpha_1 B + \dots + \alpha_h B^h)rain + Me$$

or

$$Mmala = (\gamma - k)\alpha_0 M rain + \alpha_1 M B rain + \dots + \alpha_h M B^h rain + \epsilon$$

Since there is strong multicollinearity approximated, and because of the regularity of the explanatory variable  $rain$ , following the Almon method, we take for the distribution of the  $(\gamma - k)\alpha_i$  a polynomial in  $i$  of low degree, assuming  $-1$  as root,

$$= P(i) = a + a_0 i + a_1 i^2 + a_2 i^3,$$

and we transform the explanatory variables so that:

$$Mmala = a + a_0 \sum_0^h (1+i) M B^i rain + a_1 \sum_0^h (1+i) i M B^i rain + a_2 \sum_0^h (1+i) i^2 M B^i rain + u_t$$

in which  $P$  is of third degree here and  $h$  is the maximum lag. The value of  $h$  is tested to a given degree of  $P$  (see annex I). (For this purpose,  $H_0$  is tested:  $h = h$  against  $H_1 : h = h + 1$  in the supermodel:

$$\begin{aligned} Mmala = & (b + b_0 \sum_0^{h+1} (1+i) M B^i rain + b_1 \sum_0^{h+1} (1+i) i M B^i rain + \\ & + b_2 \sum_0^{h+1} (1+i) i^2 M B^i rain + u_t) + (1 - \gamma)(a + a_0 \sum_0^h (1+i) M B^i rain + \\ & + a_1 \sum_0^h (1+i) i M B^i rain + a_2 \sum_0^h (1+i) i^2 M B^i rain + u_t^i) \end{aligned}$$

the  $b_i$ 's are estimated by MCOs in the second model (1) with  $(h = h + 1)$ , from which we get  $b_i$ ; then the  $a_i$ 's are estimated and the nullity of  $\lambda$  is tested in

$$\begin{aligned} Mmala = & (a + a_0 \sum_0^h (1+i) M B^i rain + a_1 \sum_0^h (1+i) i M B^i rain + a_2 \sum_0^h (1+i) i^2 M B^i rain) + \\ & + \lambda (b + b_0 \sum_0^{h+1} (1+i) M B^i rain + b_1 \sum_0^{h+1} (1+i) i M B^i rain + \\ & + b_2 \sum_0^{h+1} (1+i) i^2 M B^i rain) + v_t \end{aligned}$$

If  $\lambda$  is significant,  $H_0$  is rejected against  $H_1$ .)

Once the smallest significant  $h$  is obtained, and thus the maximum lag of the effects of rainfall on malaria, we draw the significant values of  $a_i$  from which we deduce the estimates  $((\gamma - k)\alpha_i) = P(i)$ . We then estimate  $k$ , on the basis of model (1), as coefficient of  $infecdis_t$ . Finally, for the malaria (cause of death so reported) variable we get:

$$\begin{aligned} mala = & 11.749 + 0.0134 rain + 0.0178 rain_{t-1} + 0.0161 rain_{t-2} + 0.0110 rain_{t-3} + \\ & + 0.0054 rain_{t-4} + 0.00222411 rain_{t-5} + 0.00416049 rain_{t-6} + \\ & + 0.0140879 rain_{t-7} + 0.0608 infecdis_t + \epsilon_t \end{aligned}$$

with

$$\epsilon_t = -0.412\epsilon_{t-1} - 0.202\epsilon_{t-2} + v_t$$

where  $v_t$  is white noise. Auto-correlations of a higher order do not prove significant.

We have estimated the  $(\gamma - k)\alpha_i$ . However, since  $k$  is weak before 1 and since we may assume that a proportion of malaria greater than this  $k$  is real malaria, we get  $(\gamma \hat{>} k) > 0$ . If we suppose that the  $\gamma - k$  and  $\alpha_i$  estimators are identifiable, estimator  $(\gamma \hat{>} k)\alpha_i$  may be broken down into  $(\gamma \hat{>} k)\hat{\alpha}_i$ . In this case we arrive at an estimation of the  $\alpha_i$ , give or take one positive factor, and deduce the hierarchy of time effects of rainfall on real malaria.

## NOTES

<sup>1</sup> Undertaken within the framework of a project jointly sponsored by the Institut du Sahel (Bamako) and the Institut national d'études démographiques (INED), Paris, whose results were published in Fargues and Nassour (1988).

<sup>2</sup> Bulletin mensuel de statistique, Direction nationale de la statistique et de l'informatique, Bamako.

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## **CORRELATES OF FERTILITY IN SELECTED DEVELOPING COUNTRIES**

*United Nations Secretariat \**

### SUMMARY

The impact of differentials in key socio-economic variables on fertility levels in 32 developing countries is assessed through multiple regression analysis of aggregate-level data on 27 developing countries for three recent quinquennia, grouped into four categories according to region (Latin America vs. Asia/Oceania/Africa) and stage of fertility transition (recent vs. relatively prolonged fertility decline). The results demonstrate the substantial impact of differences in child survival and educational attainment on the intercountry variance of fertility (the total explained variance in the total fertility rate ranges from 46 to 84 per cent), while economic indicators (per capita gross national product and per cent labour force in agriculture) have slight net impact.

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The relationship between fertility levels and various measures of economic and social development has long been a preoccupation of researchers. At one time, cross-sectional comparisons between countries at different levels of development and analysis of the historic experience of individual countries suggested that there was a strong association between economic development and fertility. More recently, policy-oriented research has indicated that improved education and health, buttressed by vigorous family-planning programmes, offer the quickest route to lower fertility rates; purely economic indicators add little to the variance in fertility rates explained in multiple regression analysis. Moreover, recent reviews of the literature on socio-economic determinants of fertility levels and trends among developing countries (Cutright, 1983; Bulatao, 1984; Jain, 1985; Safilios-Rothschild, 1985; Bongaarts, 1986; Cleland and Wilson, 1987; Stolnitz, ed., n.d.) suggest an emerging consensus that social—rather than economic—factors may well be the key factors behind fertility declines.

For the multiple regression analysis presented here, the sample consists of developing countries with populations of 300,000 or more in 1985 and available fertility estimates for three consecutive five-year periods

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\* Population Division of the Department of International Economic and Social Affairs.

(1971-1975, 1976-1980 and 1981-1985). Since the results of such an investigation may be heavily affected by the quality of fertility estimates, a special effort has been made to work only on countries with reliable data (United Nations, forthcoming). The dependent variable is the total fertility rate (TFR), averaged over a five-year period (see table 1). The sample comprises 32 countries: 4 from Africa, 13 from Asia and Oceania and 15 from Latin America.

The selection of proper independent variables is always a crucial matter for multiple regression analysis. The larger the number of variables, the greater the risk of collinearity, the more restricted the sample and the larger the potential measurement bias. At the same time, in the

TABLE 1. TOTAL FERTILITY RATES, 1966-1986

Country/area	1966-1970	1971-1975	1976-1980	1981-1986
Brazil .....	5.60	5.00	4.35	3.79
Chile .....	3.67	3.14	2.50	2.46
China.....	5.91	4.54	2.76	2.27
Colombia .....	6.26	4.70	4.23	3.38
Costa Rica.....	5.91	4.34	3.79	3.61
Cuba.....	4.12	3.31	2.07	1.77
Cyprus.....	3.01	2.28	2.33	2.44
Dominican Republic ..	7.09	5.71	4.72	3.80
Ecuador .....	6.87	6.23	5.36	4.34
El Salvador.....		5.89	5.59	4.44
Fiji.....	4.25	3.57	3.40	3.30
Guatemala .....	6.13	6.02	6.07	6.48
Hong Kong.....	3.37	3.24	2.29	1.71
Indonesia.....	5.57	5.20	4.66	3.77
Israel.....	3.77	3.77	3.35	3.13
Jamaica.....	5.93	5.00	3.64	3.35
Korea, Republic of ...	4.73	4.28	2.86	2.19
Malaysia .....	5.10	4.65	4.02	3.83
Mauritius .....	4.49	3.32	3.04	2.27
Mexico .....	6.97	6.18	5.06	4.03
Morocco .....		6.86	5.91	4.80
Panama .....	5.09	4.65	3.84	3.27
Peru .....	6.80	6.31	5.57	4.46
Philippines.....	6.13	5.56	5.08	4.90
Réunion .....	6.41	4.12	3.21	2.87
Singapore .....	3.52	2.69	1.89	1.65
Sri Lanka .....	4.73	4.01	3.83	3.18
Thailand.....	6.20	4.75	3.86	2.36
Trinidad and Tobago.	3.69	3.38	3.14	3.14
Tunisia.....	6.43	5.81	5.46	5.09
Turkey.....		5.46	4.31	4.05
Venezuela.....	5.72	4.97	4.50	3.88

Source: United Nations, *World Population Monitoring, 1989* (to be issued as a United Nations publication).

case of fertility analysis, some of the most useful variables, such as measures of intergenerational wealth flows or preponderant family and kinship structures, are unavailable for international comparison, while large differences in the definitions of urban settlements preclude the use of potentially very important urbanization indices. For the analysis presented here, five independent variables were selected (see table 2). They fall into three broad categories: health, education and economy. They are:

(a) An indicator of child survival: under-5 mortality (U5M);

(b) Two indicators of education—the gross enrolment ratio for females at the secondary level (FSER) and the gross enrolment ratio for both sexes at the primary and secondary levels of schooling combined (PSER);

(c) Two macro-economic indices—gross national product (GNP) per capita in constant 1980 United States dollars and percentage of labour force in agriculture (LFA). It has to be noted that the recently developed estimates of mortality of children under age 5 (United Nations, 1988b) is more appropriate as a potential determinant of child-bearing behaviour than the conventional infant mortality rate (United Nations, 1987). On the other hand, the FSER index, being a flow measure, is not an ideal approximation of the level of completed education; unfortunately, data on more appropriate educational characteristics, such as the proportion of female population of child-bearing age with completed secondary education, are not available for enough developing countries.

The relationship between child mortality and fertility is expected to be positive. Both indicators of education should be negatively related to fertility. First, not only are higher levels of parental education (as approximated by FSER) likely to increase the desire for a non-traditional life-style and improve the ability to use contraception, but, in addition, higher levels of the wife's education would tend to increase the opportunity cost of the wife's withdrawal from the labour force. Secondly, high current enrolment in formal school education raises the cost of child-rearing and simultaneously depresses the children's economic value. GNP, being the most commonly used single indicator of the overall development level of a country, is quite conventionally expected to be negatively related to TFR. Lastly, LFA is anticipated to be positively related to fertility, for in agricultural settings, children contribute most towards family welfare, and women's economic roles are more compatible with child-rearing. Since these variables include all of the most frequently cited structural determinants of fertility, with the major exception of accessibility of contraception (or "strength of family-planning effort"), the results of the multiple regression analysis may be interpreted as quantitative measures of demand for fertility control, while the impact of family-planning programmes is encompassed in the "residuals" or unexplained variance of TFR.

Every country in the sample has estimates of the TFR for three points in time and also of all of the independent variables. It should be noted that some of the independent variables have their impact on fertility after some interval, rather than simultaneously; obviously, women acquire their education before the age of most intensive child-bearing. Moreover, recent

TABLE 2. CORRELATES OF FERTILITY LEVELS

Country	Under-5 mortality (per thousand)			Gross enrolment ratio at the secondary level for females (percentage)		
	1960- 1965	1965- 1970	1970- 1975	1960	1965	1970
	Brazil.....	152	139	125	10	17
Chile.....	136	112	79	24	36	42
China.....	162	113	83	14	19	28
Colombia.....	135	119	102	11	16	22
Costa Rica.....	112	88	64	21	26	29
Cuba.....	77	61	45	14	24	23
Cyprus.....	34	34	33	39	37	50
Dominican Republic.....	184	158	132	14	25	20
Ecuador.....	175	156	136	10	16	31 <sup>b</sup>
El Salvador.....	190	161	137	10	14	21
Fiji.....	90	73	58	10	12	29
Hong Kong.....	47	32	21	20	30	37
Indonesia.....	225	201	173	4	8	8
Israel.....	36	30	27	48	51	34
Korea, Republic of.....	99	76	61	14	25	34
Malaysia.....	91	72	62	13	21	29
Mauritius.....	89	93	77	17	22	33 <sup>b</sup>
Mexico.....	127	113	100	8	13	17
Morocco.....	250	220	190	2	12	8
Panama.....	97	82	68	32	37	43
Peru.....	220	200	167	13	23	31
Philippines.....	128	114	101	25	40	49
Singapore.....	42	31	23	26	45	45
Sri Lanka.....	101	87	79	27	35	51
Thailand.....	136	118	91	5	8	16
Trinidad and Tobago.....	53	50	37	24	34	38
Tunisia.....	245	210	180	7	10	13
Turkey.....	239	206	184	8	9	16 <sup>c</sup>
Venezuela.....	103	84	67	21	28	39

Sources: For under-5 mortality: United Nations, *Mortality of Children under Age 5: World Estimates and Projections* (United Nations publication, Sales No. E.88.XIII.IV). For female secondary enrolment ratios and primary and secondary enrolment ratios for both sexes: UNESCO, *UNESCO Statistical Yearbook, 1975 and 1987* (Paris). For gross national product: data bank of the Population Division, Department of International Economic and

studies have demonstrated that the impact of child survival on fertility is much greater if channelled through lasting "transition" and "insurance" effects rather than through short-term "physiological" and "replacement" effects (United Nations, 1987; Lloyd and Ivanov, 1988). A plausible and straightforward lag of 10 years was adopted for U5M and for FSER for



Gross enrolment ratio at the primary and secondary levels, both sexes (percentage)			Gross national product per capita (1980 \$US)			Labour force in agriculture (percentage)		
1970	1975	1980	1970	1975	1980	1970	1975	1980
55	72	81	1 138	1 599	1 930	45	38	31
87	93	92	2 103	1 848	2 428	23	20	16
85	88	80	147 <sup>a</sup>	188 <sup>a</sup>	247 <sup>a</sup>	78	76	74
61	77	82	913	1 089	1 292	39	37	34
76	79	79	1 592	1 842	2 019	43	37	31
74	88	94	867 <sup>a</sup>	1 268 <sup>a</sup>	1 520 <sup>a</sup>	30	27	24
68	68	68	2 542	2 136	3 517	38	32	26
65	74	81	773	1 029	1 155	55	50	46
69	73	85	773	1 218	1 373	51	45	39
55	63	63	713	796	733	56	49	43
80	90	94	1 358	1 682	2 017	51	49	46
79	78	81	2 706	3 408	5 508	4	3	2
43	55	72	273	363	460	66	62	57
77	87	87	4 200	5 040	5 298	10	8	6
76	81	93	892	1 222	1 583	50	45	39
62	67	70	861	1 262	1 724	54	48	42
66	70	73	801	1 147	1 160	34	31	28
68	76	87	1 873	2 185	2 612	44	40	37
32	37	50	618	637	763	58	52	46
75	87	85	1 324	1 494	1 763	42	37	32
78	85	91	996	1 010	974	47	43	40
90	87	95	510	611	732	55	53	52
77	78	81	2 180	3 222	4 524	3	3	2
73	61	77	209	245	278	55	54	53
57	62	66	439	547	706	80	75	71
81	83	85	6 351	4 787	5 499	19	15	10
64	60	64	771	1 139	1 365	42	38	35
65	66	72	948	1 254	1 312	71	65	58
72	77	76	5 965	4 467	3 921	26	21	16

Social Affairs (DIESA). For labour force in agriculture: United Nations, *World Demographic Estimates and Projections, 1950-2025* (ST/ESA/SER.R/79).

<sup>a</sup> Net material product per capita, instead of GNP.

<sup>b</sup> 1971.

<sup>c</sup> 1982.

this study. The other independent variables need not be lagged, for they relate to the societal context of fertility decision-making. Because countries of Latin America differ considerably from the rest of the developing world with respect to the most salient features of the general process of socio-economic development (Akademia, 1976) and to the strength and/or sign

of association of social factors with fertility levels (Kirk, 1971; Anker, 1978; Salas and Valentei, 1986), the general sample is subdivided into two sub-samples—one comprising the countries from Asia, Africa and Oceania, and the other the Latin American countries.

Table 3 shows the strength of relationships between pairs of variables. A few conclusions may be drawn from it. First, all zero-order correlation coefficients confirm *a priori* expectations for the directions of relationships. Secondly, the indicator of child survival has the strongest relationship with fertility for Asia/Oceania/Africa and is closely associated with fertility in Latin America. Thirdly, in the general sample and in the African/Asian sub-sample, female education is consistently more strongly correlated with both fertility and child survival than the other social or economic indicators. Fourthly, patterns of association in the Latin American sub-sample substantially differ from the other two data sets, with agricultural employment being more closely related to all demographic and social variables and with a much stronger (negative) impact of current school enrolment (PSER) on fertility. Fifthly, GNP plays a very modest role in predicting not only the level of fertility but the values of its closest correlates (such as child survival and education variables) as well.

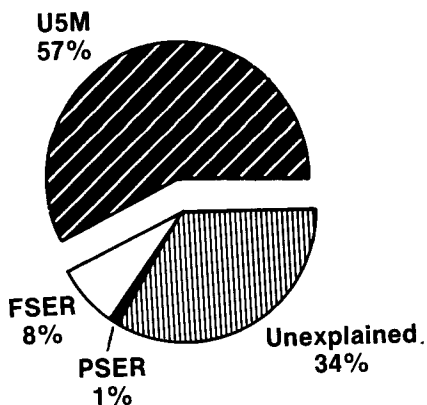
Figures I-IV illustrate the major results from multiple regression analysis for two regional sub-samples separately. Moreover, since patterns of causation are likely to change as the process of the fertility transition develops, multiple regressions were run separately for two categories of countries (in each region): countries with a fairly long history of fertility decline are contrasted with countries with a more recent onset of decline. The TFR at 5.5 in 1966-1970 is selected as the threshold separating the

TABLE 3. ZERO-ORDER CORRELATIONS BETWEEN PAIRS OF VARIABLES, BY GEOGRAPHICAL AREA

Variable	TFR	U5M	FSER	PSER	GNP
All countries					
U5M .....	0.78				
FSER .....	-0.64	-0.71			
PSER .....	-0.52	-0.55	0.61		
GNP .....	-0.43	-0.58	0.52	0.36	
LFA .....	0.51		-0.57	-0.39	-0.78
Africa, Asia and Oceania					
U5M .....	0.82				
FSER .....	-0.58	-0.75			
PSER .....	-0.47	-0.61	0.57		
GNP .....	-0.55	-0.63	0.60	0.41	
LFA .....	0.53	0.61	-0.57	-0.30	-0.83
Latin America					
U5M .....	0.72				
FSER .....	-0.77	-0.59			
PSER .....	-0.70	-0.52	0.71		
GNP .....	-0.25	-0.49	0.34	0.27	
LFA .....	0.78	0.76	-0.69	-0.71	-0.66

Figure 1. Correlates of fertility, eight countries of Asia and northern Africa where TFR was 5.5 or higher in 1966-1970

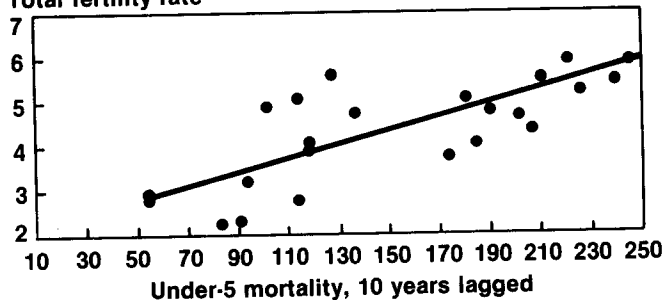
Percentage of TFR variance explained by



Significant at .05 or .001 level  
N-24 DF-20 F-13.1

Relationship between 10 years lagged child mortality and fertility

Total fertility rate



• Observed TFR — Regression line

China, Indonesia, Morocco, Philippines,  
Réunion, Thailand, Tunisia, Turkey  
N-24 R<sup>2</sup>-0.57 F-33.9 p-.001

Figure II. Correlates of fertility, nine countries of Asia, Africa and Oceania where TFR was lower than 5.5 in 1966-1970

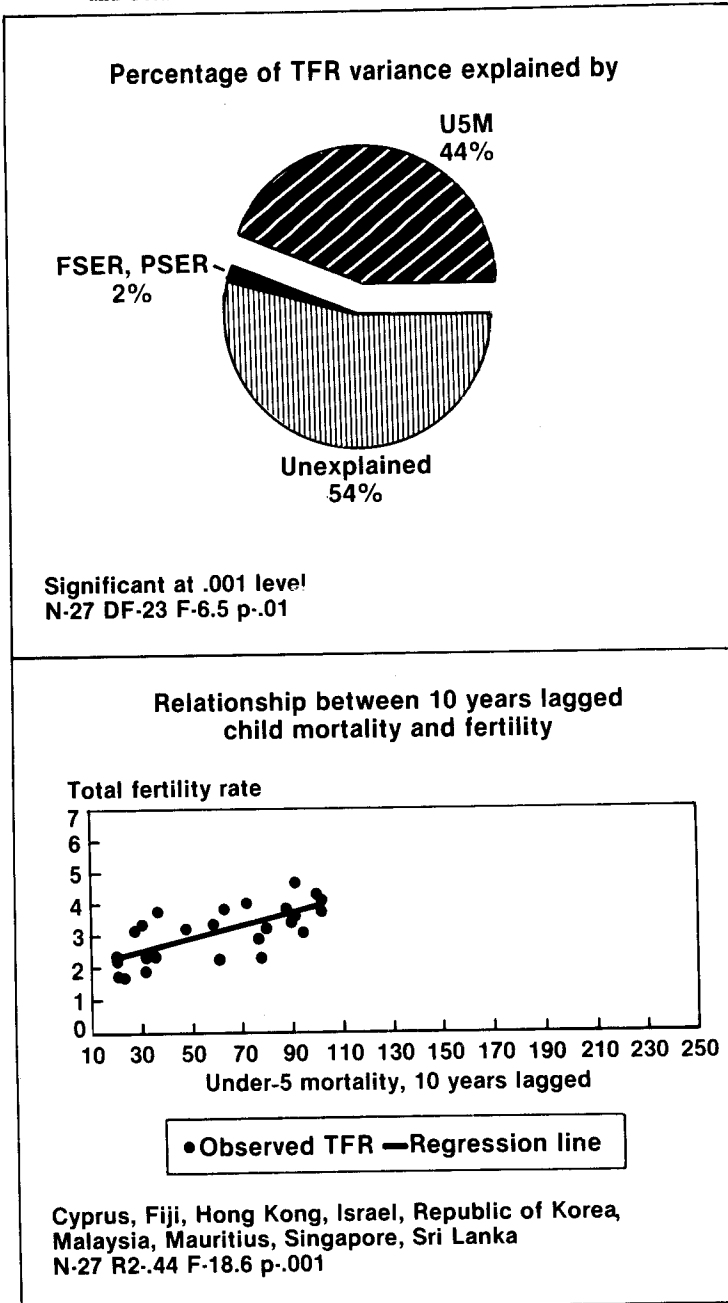
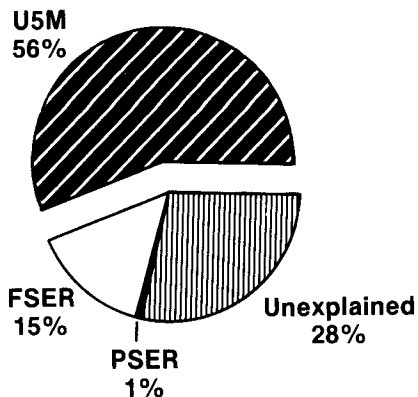


Figure III. Correlates of fertility, 11 countries of Latin America where TFR was 5.5 or higher in 1966-1970

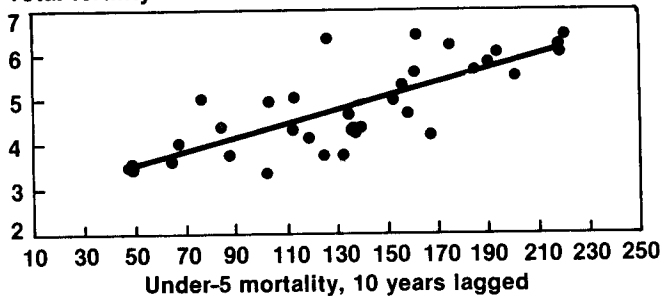
Percentage of TFR variance explained by



Significant at .001 level  
N-33 DF-29 F-24.4 p-.001

Relationship between 10 years lagged child mortality and fertility

Total fertility rate

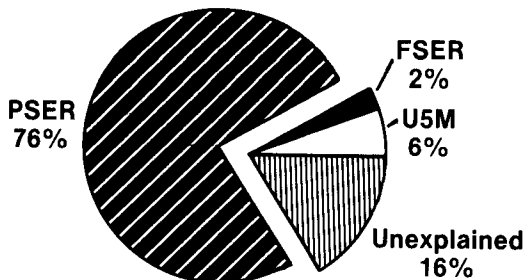


• Observed TFR — Regression line

Brazil, Colombia, Costa Rica, Dominican Republic, Ecuador, Peru, El Salvador, Guatemala, Jamaica, Mexico, Venezuela

Figure IV: Correlates of fertility, four countries of Latin America where TFR was lower than 5.5 in 1966-1970

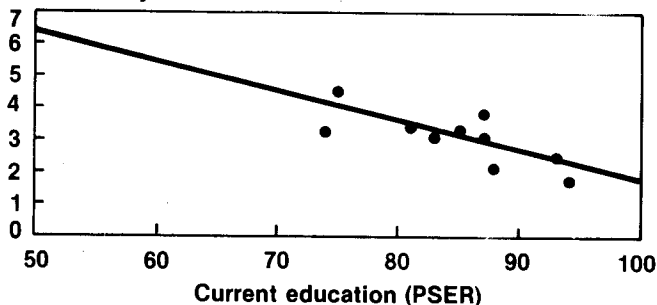
Percentage of TFR variance explained by



Significant at .001 level  
N-12 DF-8 F-13.6 p-.01

Relationship between current education and fertility

Total fertility rate



• Observed TFR — Regression line

Chile, Panama, Cuba, Trinidad and Tobago  
N-12 R<sup>2</sup>-.76 F-37.0 p-.001

first category from the second. Examination of fertility determinants within four groups of countries separately should reduce the potential distortion of estimates of causal effects that may exist when all the countries form a common pool of observations. Also, replication of results between the groups should heighten the credibility of the empirical results. It must be noted that the reported results do not include the impact of economic indicators on fertility. This is not only because GNP and LFA did not have significant coefficients when included in the additive ordinary least square regression models and consistently added only a few percentage points to the explained variance of national fertility levels but also because in most trials their inclusion decreased the F-statistics below acceptable levels of significance.

The pie charts in figures I-IV show the increments in multiple coefficients of determination due to the inclusion of variables in the hierarchical decomposition of the TFR variance. The plots illustrate the dispersion of TFR values along the (zero-order) regression line for the most important predictor. Explained variance for all four groups of countries is high, ranging from 46 per cent in advanced transition countries of Asia, Africa and Oceania (fig. II) to 84 per cent in advanced transition Latin American countries (fig. IV). Considering the relatively small number of degrees of freedom in each sub-sample, the results are reassuring in the sense that the most significant coefficients got the expected signs (except FSER in the sub-sample of Asian and northern African countries with "recent" fertility declines (see fig. I).

The most important conclusion from this regression analysis is the major contribution of improvements in child survival to fertility decline. In fact, in all but one group of countries (four fairly advanced Latin American countries (see fig. IV), differences in U5M explain approximately half of the overall variance in TFR about 10 years later, the slopes of regression lines are similar and the deviations of observed TFRs from those lines are relatively small (see figs. I, II and III). The highest unexplained variance of TFRs in advanced transition countries of Asia, Africa and Oceania (fig. II) may imply an especially important independent role of strong family-planning programmes in many of those countries (e.g., Hong Kong, Republic of Korea, Malaysia, Singapore, Sri Lanka). Finally, the quite particular pattern of causation of fertility decline in the four Latin American countries (fig. IV) highlights the role of education in settings characterized by relatively low child mortality and fairly modernized social structures.

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