The Decline of Childhood Mortality

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Introduction

One of the most dramatic and significant changes in the human condition over the last two centuries has been the sharp and sustained decline that has taken place in infant and child mortality. In the now-developed countries of Europe, North America and Oceania, the probability of dying by first birthday has declined from, in many cases, 200 per thousand live births to less than 10 per thousand live births in the span of 100 years. This transition has had, and continues to have, profound implications for the family and society. This chapter chronicles the trend, and examines a variety of explanations for it that have been proposed.

Before going any further, we must define what we mean by childhood and what measures of mortality we will use. In normal speech, childhood tends to cover the period from birth to adulthood, which is usually thought of as starting when a child becomes independent of its parents, typically around age 20. However, typical age patterns of mortality risks start high at birth, and then decline to a minimum level around age 10, before increasing again, at first gradually to around age 50, and then more and more rapidly. There is thus a natural break around age 10 where the decline with age of childhood reverses to become the increase with age of adulthood. In this chapter, we will be primarily concerned with the age span before this natural break, that is, before age 10, and especially with the shorter span before age five in which the majority of child deaths take place. The exception to this rule will be where data are only available for a somewhat longer span, for example up to age 20.

The most widely used measures for describing levels or changes of child mortality are probabilities of dying between exact ages, for example x and x+n, of childhood, typically derived from life tables[1] and denoted by $_nq_x$, and age-

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[1] A life table is a convenient way of representing mortality risks by age. An imaginary group of births are exposed to a given set of age-specific mortality risks, the number of survivors declining as age increases. A number of useful mortality indicators, such as probabilities of dying by or between exact ages and the expectation of life at birth, can be derived from the resulting survivorship

specific mortality rates, typically derived from deaths and population by age, and denoted by $_nm_x$. The infant mortality rate, a commonly quoted measure, is calculated directly from births and deaths before age one, and is approximately equal to the probability of dying by age one, $_1q_0$. Although widely quoted, the infant mortality rate is a less than ideal indicator of child mortality in general, since a substantial, but also variable, proportion of mortality in childhood takes place between the ages of one and five. The best overall indicator of child mortality is thus the probability of dying by age five, $_5q_0$, and where possible we will use this indicator for general descriptive purposes. However, other features of child mortality, such as its pattern by age and by cause of death, are of great importance to efforts to account for child mortality change, so we will also be concerned with age and cause of death patterns of child mortality.

Data Availability and Data Deficiency

Reasonably good information about child mortality only becomes available with the emergence of sophisticated civil registration systems for recording births, deaths and population. Such systems first developed in north-western Europe in the late eighteenth century[2], and have since spread to countries in many other parts of the world. All world regions with the major exception of sub-Saharan Africa are now represented by one or more countries with statistical systems adequate for monitoring child mortality levels and trends. However, these countries are unlikely to be statistically representative of their regions; for those countries lacking such systems, and for sub-Saharan Africa as a whole, non-conventional methods[3] are still required for measuring child mortality. Estimates of child mortality for periods prior to the emergence of civil registration systems also have to be derived from unconventional sources; for the

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curve.

[2] The systematic collection of other useful information such as cause of death starts somewhat later still, in the late 19th century.

[3] These methods use survey data for women concerning some or all of the children they have borne, and the survival of those children (see UN,1983).

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16th to the 19th centuries, these sources are generally family reconstructions, from parish registers, family histories, or for especially well-documented population sub-groups such as the English aristocracy. Prior to the 16th century, there are no sources of usable written evidence, though some estimates of mortality for historic and prehistoric periods can be derived from the analysis of ages at death based on the excavation of burial grounds. However, such data are both scanty and hard to interpret, so most hypotheses about child mortality prior to the 16th century have to be based either on general arguments about population dynamics or on extrapolations from recent conditions in some parts of the developing world.

We are on firm ground in tracing levels, age patterns and cause structures of child mortality from the latter part of the 19th century in Europe, covering the major decline. However, for the more recent child mortality decline in the developing world, we are rarely able to trace changes in cause patterns of mortality, only in a minority of cases able to follow changing age patterns of mortality, and in some cases not even able to track levels. Inevitably, the weaker the information base, the weaker the justification for explanations of observed changes.

Child Mortality in Prehistoric and Early Historic Times

The information now available about prehistoric child mortality is limited to skeletal remains at burial sites and recent observations of populations apparently living in largely prehistoric conditions. A number of studies have attempted to use skeletal remains, with estimated ages at death, to estimate the average age at death, and thus approximate expectation of life at birth (Acsadi and Nemeskeri 1970). Some of the studies have arrived at plausible estimates of overall mortality (for example, Indian Knoll skeletal remains indicate an average age at death of about 18.5 years (Johnston and Snow 1961)) but with implausible internal detail indicating age and sex patterns of mortality quite unlike any accurately recorded. Problems of selection of whom gets buried in a particular site and of differential skeletal decay rates by age make it virtually certain that satisfactory estimates of child mortality will never be derived from burial ground excavations (Brothwell 1975).

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Somewhat more promising are recent studies of hunter-gatherer populations with minimal exposure to modern life. The children born between 1919 and 1938 to interviewed women of the !Kung tribe in southern Africa (Howell 1979) experienced a $_1q_0$ of 164 per thousand and a $_4q_1$ of 268, for a reported $_5q_0$ of 388. This level of infant mortality is rather low relative to the estimate of child mortality in comparison with model life table patterns[4]; if the $_4q_1$ estimate is taken as correct, the $_1q_0$ would be closer to 300, and the $_5q_0$ would be about 490. Thus it is probably the case that among one very primitive modern population of hunters and gatherers, somewhere between 400 and 500 children born died before their fifth birthday. However, we cannot safely extrapolate from one modern primitive population to other modern primitive populations, nor to premodern primitive populations.

That said, we can draw some conclusions about child mortality in the distant past from theoretical constraints of population dynamics. Over the long haul of pre-recorded history, the human population survived, but grew very slowly, with an average annual growth rate of less than one per thousand (the process was in fact probably rather uneven, with periods of growth alternating with periods of contraction, but such unevenness does not affect the argument). Over the long haul, births and deaths have to have been in very close balance, and the net reproduction rate (the number of females surviving in the next generation to replace the mothers of this generation) must have averaged very slightly over 1.0. If we assume that on average the maximum number of female children a woman can have is about four (consistent with the highest fertility rates observed in the contemporary developing world), then in order for the population to survive, the female probability of surviving to the late 20s (roughly the average age of childbearing) must be at least 250 per thousand (that is, one in four). Such survival is consistent with a probability of dying by age five of about 600 per thousand, comfortably higher than the maximum level estimated for the !Kung. If, on the other side, we assume that the minimum number of female children that women will on average have, if they do not delay cohabitation and practice no

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[4] Model life tables (e.g. Coale and Demeny,1966) express empirically-observed regularities in age patterns of mortality at different mortality levels.

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form of birth control other than breastfeeding or post-partum abstinence, is 2.5, then the female probability of surviving to the late 20s must be no higher than 400 per thousand, to avoid overly rapid growth. Such a value implies a probability of dying by age five of roughly 440, below the maximum level but above the original estimate for the !Kung. Thus the requirements of population dynamics indicate that, over the long haul of prehistory, the probability of dying by age five for females was probably no lower than 440 per thousand live births, and was probably no higher than 600.

The cause of death mix in childhood for prehistoric man would have been very different from that of a couple of centuries ago. The major infectious diseases with short incubation periods and conferring on survivors long and high immunity (measles, smallpox, diphtheria, pertussis, etc.) would have been rarities because the population groups would have been too small to support them (infective agents, whether bacterial or viral, require a supply of infectible hosts for their own survival). Diseases such as tuberculosis and malaria which can be maintained for long periods in a host and create only moderate immunity would have existed (vector permitting, in the case of malaria), as would diseases with animal reservoirs such as yellow fever and trypanosomiasis, and probably intestinal infections (Lancaster 1989). Starvation would have been a much more important cause of death in a hunter-gatherer population with no means for storing food surpluses against bad times, and violent or accidental deaths may be assumed to have been more important also than among settled populations. Infanticide is sometimes put forward as an important cause of infant death in primitive populations, partly to explain high mortality in the absence of the major infectious diseases. The !Kung and other primitive groups (Daly and Wilson 1984) are reported to have practiced infanticide routinely in cases of birth defects or other situations in which the child would be unlikely to survive, but not as a method of population control. There is some evidence that female infanticide was practiced in China into this century. However, it seems unlikely that infanticide can have been very common among hunter-gatherer populations. It will not normally maximise genetic representation in subsequent generations, and is observed in primates only in species with dominant male breeding groups, and only then at times of change over from one dominant male to another (Hausfater and Hrdy 1984).

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The transformation from hunting and gathering to settled agriculture in river valleys with higher population density, and from there to the development of the large cities of the classical world, probably had little effect on average mortality in childhood. However, it will have affected the cause structure of mortality, with a shift away from starvation (as surpluses can be stored and later transported to even out lean years) and violent deaths to infectious diseases supported by increased transmission resulting from greater crowding and larger susceptible populations. Firm evidence on the level of child mortality during this period is lacking; some attempts have been made to estimate mortality from stone inscriptions from the Roman empire (MacDonell 1913), but such analyses fall foul of the same problems of selectivity that affect the analysis of skeletal remains (Hopkins,1966). Although some analyses have produced plausible-looking estimates of overall mortality, expressed for example in terms of expectation of life at birth, the age-specific detail is such that the evidence either has to be dismissed or it has to be accepted that the age pattern of mortality then existing was completely different from any human mortality patterns that have been accurately observed. All we can do is conclude from the population dynamic argument that child mortality was on average high throughout early history. It is likely that, with the development of settled agriculture and epidemic disease, cycles of population growth and decline may have occurred; thus the expansion of the Roman empire probably occurred during a period of population expansion driven by a slight mortality decline arising from much-improved transportation systems and food distribution, a decline that will have been reversed during the empire's collapse. The development of cities, with high levels of crowding and thus increased rates of disease transmission, will also have given rise to the development of child mortality differentials favoring rural areas; city populations will have relied upon continued migration from rural areas for their very existence (Wrigley 1972), in sharp contrast to the situation of developing countries today.

Child Mortality in Europe from 1500 to 1800

Beginning around 1500, we start to have documentary evidence about child mortality levels in Europe, at least for some population sub-groups. The earliest observations are from family reconstructions for the aristocracy or high

bourgeoisie, by no means representative of the population at large, but as we move forward into the 17th and 18th centuries, the number and variety of populations that can be reconstructed from religious records increases substantially. Table 1 summarizes the child mortality estimates obtained by a number of these studies.

The British peerage (Hollingsworth 1964) provide the most complete and detailed sequence. The probability of dying by age five in the mid 16th century is around 250 per thousand live births, rising steadily to around 350 by the mid 17th century, before starting a steady decline to below 200 by the mid 18th century and around 100 by the mid 19th century. The rise from the 16th to the 17th centuries might be taken as evidence of data errors (omission of child deaths) in the earlier period, were it not for the facts that both a less-detailed series for the English royal families (a group Henry (1965) argues would be less likely to suffer from omission) and adult mortality for the peerage itself show very similar general trends (though at a slightly higher level in the case of the royal families). Thus child mortality probably did rise for this very small population sub-group from the 16th to the 17th century, perhaps reflecting the adoption of a more urban life style (which might also help to explain why the royal families had slightly higher child mortality than the peerage). Female children have in general somewhat lower child mortality than male, though for most of the 17th and 18th centuries the differences are small and inconsistent. With respect to the extent to which the peerage may be taken as representative of a broader population, it is instructive to compare the figures for the period after 1775 with those obtained from virtually complete registration for Sweden; around 1800, the British peerage have child mortality levels only half those of Sweden. Clearly, privilege not only had its obligations but also its benefits in terms of lower child mortality.

The Geneva Bourgeoisie (Henry 1956) show high child mortality, with a probability of dying by age five close to 400, until the end of the 17th century; thereafter, child mortality drops rapidly, following a trend very similar to that of the British peerage. Geneva, being a city, would probably have experienced high overall child mortality, and being of bourgeois origins provided little protection, it seems, until the start of the 18th century.

The third series in Table 1 shows child mortality in English parishes (Wrigley and Schofield 1983) which were either rural or consisted of moderate-size market towns. Among this population, which includes no major towns, child mortality apparently rose somewhat during the 17th and early 18th centuries, before declining slightly in the late 18th century. The level of mortality is surprisingly low, however, starting at levels well below the British peerage, and only clearly exceeding the peerage's level in the late 18th century. There are also very marked differentials in child mortality among the 13 parishes studied; in the most rural parishes, the probability of dying by age five was only about 200 per thousand live births, whereas in the market towns, the comparable figure exceeded 400 for some time periods. Overall, the series no doubt underestimates child mortality in England, since it excludes major towns, but the authors believe it probably captures trends. No breakdown by sex is available for this series, but the reported age pattern of child mortality shown is very similar to that of a Coale-Demeny 'North' model life table.

These fragments of data provide a basis for some conclusions. First, even well-to-do families suffered high child mortality levels not very different from the !Kung (and presumably not very different from the population at large) as recently as the 17th century. Second, mortality decline for these privileged populations started early, apparently by 1700. Third, there were very large urban-rural differentials in child mortality, the risks in urban areas apparently being double those in rural areas. Fourth, overall child mortality was declining in Sweden from the late 18th century onwards. In the context of the earlier discussion of the limits placed on child mortality by the requirement of long-run negligible population growth, it is worth noting that the relatively low mortality in rural England was associated with late and less-than-universal marriage, tending to hold fertility down, and that the sharp decline in child mortality among the Geneva bourgeoisie in the 18th century was associated with a rapid decline in marital fertility. Thus these relatively low levels of child mortality are not inconsistent with the requirements of population dynamics discusssed earlier.

Sustained Decline in Child Mortality in the Developed World, 1800 - 1990

Civil registration data on child mortality become increasingly available for European countries in the first half of the 19th century. The country with the longest sequence of infant mortality rates unbroken by war or other disturbance is Sweden; Figure 1 shows the decline in infant mortality in Sweden from 1751 to 1988 by single calendar years. Infant mortality appears to have fluctuated around an approximately constant level of about 200 per thousand live births until about 1810. It then declined fairly steadily to a level of about 100 around 1900, fell somewhat more rapidly to around 20 in 1950, and then continued to decline slowly towards its current level of about five deaths per thousand live births. A dramatic feature of Figure 1 is the way in which the variability of infant mortality has declined as the level itself has declined; even in the early 19th century, before the level had changed much, the fluctuations are much reduced. Series for other European populations show similar patterns, though with different initial levels, and mostly later starting points for sustained decline. For example, infant mortality in Germany was as high as 300 in the 1870s, did not fall below 200 until the first decade of the present century, was still over 100 in the 1920s, but fell to 10 in the 1980s; thus the starting point was higher and the decline both later and more rapid than in Sweden.

As was stressed in the introduction, infant mortality is a major part of childhood mortality, but not the only part. It is interesting also to examine how the age pattern of child mortality changed as the level declined. Figure 2 shows the relationship between the probability of dying between birth and age one, $_1q_0$, and the probability of dying between the ages of one and five, $_4q_1$, by sex, for three western European populations (France, the Netherlands and Sweden) from the mid 19th century to the mid 20th century, thus covering the period of rapid child mortality decline. The general pattern, very clearly shown by France and the Netherlands, but only roughly followed by Sweden, was for mortality between ages one and five to decline somewhat before any major decline in mortality in infancy begins. In the second phase of decline, both infant and post-infant mortality decline proportionately, creating parallel straight lines in Figure 2, with infant mortality declining faster than post-infant mortality. The change in pattern from declining post-infant and stable infant to declining infant and post-infant mortality occurs in the late 19th century, around 1875 in Sweden, 1885 in the Netherlands, and 1895 in France; the association of this point with

date appears to be stronger than its association with overall level. The pattern and timing of decline are practically identical for males and females, though the lines for females are slightly to the right (ie, heavier post-infant mortality relative to infant) and below (ie, lower overall mortality in childhood) those for males.

Trends in mortality by cause are difficult to interpret because of changes over time in the classification system, errors of classification, and specificity of classification. The impact of such problems can be reduced, but not eliminated, by considering fairly broad groups of causes, namely infectious and parasitic diseases (broadly the immunizeable diseases of the 20th century, and here including tuberculosis), respiratory diseases (mainly pneumonia and bronchitis), diarrhea, certain diseases of infancy (largely prematurity and developmental diseases) and other or unknown causes. Diagnostic ambiguities between such groups are likely to be small compared to such ambiguities surrounding classifications within the groups. England and Wales provide the longest sequence of recording of cause of death, from 1848 onwards, thus covering the entire period of the 19th and 20th century child mortality decline. Figure 3(a) shows the trends in male mortality rates in infancy by cause group from the 1860s to 1960, expressed as an index taking the average of the rates for each cause in 1861 and 1871 as unity. Figure 3(b) shows comparable trends for the age group 1 to 4. Male trends are shown for simplicity; female trends are almost identical. It should be remembered that the points are figures for single calendar years, and are thus affected by short-term fluctuations.

In the base period, the cause group making the largest contribution to infant mortality is the other and not known category (31%), followed by certain diseases of infancy (primarily prematurity and infantile convulsions) (28%). The three more specific groups, infectious and parasitic diseases, respiratory diseases, and diarrhea and enteritis all contribute similar amounts (14, 15 and 12 percent respectively). Overall mortality in infancy declines briefly to 1881, all cause groups contributing to the decline, with diarrhea showing the greatest fall, and respiratory diseases the smallest. Overall mortality then rebounds to 1891 and 1901; all cause groups contribute to the rebound in 1891, though respiratory deaths increase most, but in 1901, all causes of death drop, with the

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sole exception of diarrhea, which increases enough to keep overall mortality more or less constant. Mortality from diarrhea is even higher in 1911, but other causes fall sharply enough for overall infant mortality to drop substantially. After 1911, all cause groups and infant mortality as a whole decline over each period, with one exception, respiratory mortality, which increases slightly from 1921 to 1931. Overall mortality clearly falls fastest from 1911 to 1921, and then falls rapidly again from 1940 to 1951, falling by nearly 50 percent in each period.

The most volatile cause group is diarrhea and enteritis, which falls to 1881, rises steeply to 1901 and 1911, before falling sharply to low levels by 1931. The earliest group to show sustained decline is the infectious and parasitic diseases, which decline rapidly from 1881 to 1921, by which time their mortality rate is only one quarter what it had been in the two base years; the decline continues steadily to 1961, by which time the mortality rate is less than one percent of its base value. The other and not known group follows infectious and parasitic diseases quite closely until 1931, suggesting that many of these non-specific deaths actually belonged in the infectious and parasitic group; from 1931 onwards, the residual group's mortality declines more slowly, as true other causes, including accidents and violence, become the predominant component of the group. The certain diseases of infancy group tracks overall mortality closely until 1901, after which it declines somewhat more slowly than overall mortality, reflecting the continued mortality from prematurity. Respiratory mortality stays above overall mortality until 1911, including a sharp spike in 1891, and then stays well above overall mortality during the 1920s and 1930s, with only a small decline, before dropping back to an average decline by the 1950s. In summary, mortality in infancy fell by 90 percent from the base period to 1960; the cause group certain diseases of infancy dropped least, to about a quarter of the base value; the two groups infectious and parasitic diseases and diarrhea and enteritis dropped most, to less than one percent of their base rates; respiratory mortality and the residual other group each fell by about the average amount, to about 10 percent of the initial level. Mortality from respiratory infections and diarrhea prevented an earlier decline in infant mortality that would have resulted from sustained falls in mortality from infectious and parasitic diseases, continued high levels of respiratory mortality prevented a faster

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decline in mortality in the 1920s and 1930s, and relatively slow declines in mortality from certain diseases of infancy braked the decline in overall infant mortality in the post-war period.

The patterns of mortality decline in the age range 1 to 4 show some interesting similarities and some interesting differences. The overall decline is virtually uninterrupted from the base point, 1861 and 1871, to less than three percent of its initial value by 1960, except for a slight upward shift in 1891. The initial structure of mortality by cause is rather different; infectious and parasitic diseases were responsible for nearly half the baseline deaths, so the decline in such deaths has a much larger effect on overall mortality. The other and not known category accounted for 31 percent of deaths at the baseline, the largest specific components being infantile convulsions and accidents and violence. Respiratory deaths accounted for 17 percent, about the same as in infancy, but diarrhea deaths accounted for only seven percent. The two largest groups, infectious and parasitic and other and not known, decline at much the same rate as mortality overall, not surprisingly since they represent nearly 70 percent of baseline mortality; the former group is consistently slightly below overall mortality, whereas the latter group is below until 1931, but then above from 1941 onwards, reflecting the intransigent mortality in the residual group. Respiratory mortality rises sharply to 1901, and then declines rather more slowly than overall mortality to 1931, after which it declines much more sharply through 1941 and 1951. Diarrheal mortality declined very sharply to 1881, to less than 40 percent of its base value, than rose sharply to 1911, above its base value, and then fell extremely sharply to 1931, after which it stays at low levels.

The infectious and parasitic disease group is of special interest, because it is in this group that sustained decline started, and it is a group that consists of a number of well-differentiated conditions. Although the data (Logan 1950) are not shown here, mortality from each component cause, despite their diversity, had declined to less than 10 percent of its pre-decline value by the 1950s, both in infancy and between ages one and five. That said, different diseases did decline at different times and at different speeds. Scarlet fever, the second most important infectious disease killer of children in the mid 19th century, and smallpox and typhus, probably already much reduced as killers by the

time observation begins in mid century, had fallen to below five percent of their mid-century mortality by the beginning of the 20th century. A second group, consisting of the most important cause of mortality in the group, non-respiratory tuberculosis, and including measles, diphtheria and pertussis, decline more slowly to the second world war, to some 10 percent of their pre-decline levels, and then declined very sharply in the 1950s.

Factors Accounting For Developed Country Child Mortality Decline

The two extreme explanations of child mortality decline are on the one hand that the improvements are due to medical science and on the other that they are due to general improvements in economic and social conditions; somewhere in between is the public health explanation, that mortality fell at least in large part as a result of improvements in environmental factors such as water supply and excreta disposal. It is useful in a discussion of this issue to distinguish three ways in which mortality can be reduced: reduced exposure, increased host resistance, or improved recuperation (therapy). Exposure can be reduced through social progress (e.g., better housing) and public health measures (improved water supply, sanitation, vector control). Host resistance can increase through improved nutrition or lower disease burdens, or through immunization. Therapeutic measures arise from advances in medical science. Exposure factors are influenced largely by general development factors and by public health measures, guided by medical science to some extent. Resistance factors can be divided into the general, the result of economic and social advance, and the specific, the result of medical science.

The first view, giving preeminence to medical advances, was the conventional wisdom until the 1950s, but has, since then, been largely discredited. As has been seen in the previous section, child mortality fell from a number of diseases (e.g., tuberculosis, pneumonia) in the early part of the 20th century for which there was no effective therapy or immunization. This is not to say that medical science had no impact. Mortality from smallpox in Sweden fell to virtually nothing in the first two decades of the 19th century, following the introduction of vaccination (Hofsten and Lundstrom 1976); mortality from diphtheria fell sharply in the early 20th century with the introduction of serotherapy in 1895,

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and fell again in the 1940s with the introduction of vaccination; mortality from respiratory diseases remained relatively high until the 1930s, when a sharp fall coincided with the introduction first of sulfonamides and a few years later of antibiotics. Thus medical advances probably contributed to the pace of mortality decline in the early 19th century, and again in the early 20th century, but major declines took place for which medical science cannot take responsibility.

The second view, giving preeminence to social and economic development with particular emphasis on improved nutrition, has been argued forcefully (McKeown 1976) since the 1950s. The development view encompasses reductions in exposure to pathogens through improved water supply, sanitation and housing conditions, increases in resistance via improved nutritional status, and improved recovery chances also through improved nutrition. However, there is little direct empirical evidence for the key role of nutrition; the indirect argument, which follows Sherlock Holmes' dictum, that if all other alternatives are clearly disproved, whatever remains, however implausible, must be correct, is less than satisfying. The account also fails to explain a number of the features of the mortality decline, such as the virtual disappearance of scarlet fever over a period of only thirty years, or the marked rural advantage in child mortality in Victorian England despite lower development and probably poorer nutritional levels, or the only modest child mortality advantage of the English royal family and peerage, population sub-groups that enjoyed high standards of living and ample nutrition.

There can be little doubt that public health measures contributed substantially to the pace of child mortality decline through reduced exposure. Studies of urban France show close agreement between child mortality decline and the provision of clean and copious water (Preston and van de Walle 1978). The sharp increase in diarrheal mortality in England and Wales in the last decade of the 19th and first decade of the 20th centuries coincided with a string of hot summers that reduced water availability and cleanliness (Woods et al. 1988); after 1911, diarrheal mortality dropped faster than any other element of child mortality as chlorination of water became widespread. However, public health measures would have a major impact only on mortality from diarrhea (through improved water and sanitation) and from tuberculosis (both bovine, through

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control of the milk supply, and respiratory, through control of transmission mechanisms such as spitting), accounting for about 20 percent of mid 19th century deaths. The contribution of public health measures should be split between economic development, which provided the resources, and medical knowledge, which provided guidance on targets and priorities.

A number of other theories have been put forward to explain why child mortality declined so fast and at the time that it did so. It has recently been argued (Woods et al. 1988/9) that it was fertility decline that triggered the decline in infant mortality in England and Wales. Support for this explanation lies mostly in observed differentials in child mortality by birth order, but fertility changes cannot explain why mortality after infancy declined three decades before infant mortality, but in apparently rather similar ways in terms of cause of death mix, nor why rural England had infant mortality that was so much lower than in urban England. In fact, the whole notion that one single factor was predominantly responsible should be abandoned, in favor of a view that economic development, social development (through education and spread of ideas), medical knowledge, and, at least in the 20th century, medical practice, contributed to the decline in child mortality. The suddenness of the decline in infant mortality in England and Wales is a historical accident, as comparison with the case of Sweden, with a much smoother downward slope, suggests; infant mortality would have declined several decades earlier in England but for an upsurge first in respiratory deaths, and then in diarrheal deaths, the latter fairly clearly linked to a short term climatic fluctuation. The real issue for England is not why infant mortality declined when it did, but why it had not declined earlier.

Infant and Child Mortality in the Developing World

Relatively few less-developed countries (LDC's)have civil registration systems that are complete enough to provide accurate measures of child mortality,

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even for the recent past[5]. The number of countries with good records prior to 1950 is very small indeed. One of the few countries with an unbroken record of reasonably accurate infant mortality rates since 1900 is Chile; Figure 4 shows the decline in infant mortality in Chile since 1901. Early in the century, infant mortality was around 300 per 1,000 live births, with variations of close to 100 points between the highest and the lowest years. Around 1920, the variations become much less pronounced, and a pattern of sustained decline starts. However, the decline is interrupted twice, once in the 1930s, when infant mortality increases briefly, and again between 1955 and 1965, when the decline apparently stalls. There are thus both similarities and dissimilarities with the case of Sweden in Figure 1. In both cases, the decline begins with a narrowing of annual fluctuations, after which a fairly steady decline takes place. However, in the case of Chile, the starting point is higher, around 300 per thousand against around 200 per thousand in Sweden, the decline starts about 120 years later, around 1920 as opposed to 1800, proceeds faster, falling below 20 per thousand in a span of 75 years against 150 years, and shows more pronounced fluctuations around the trend (Sweden shows weak stalls in the 1890s and 1920s, but no major reversals such as that seen in the 1930s for Chile).

In the more distant past, the now less-developed countries experienced child mortality levels consistent with fairly high fertility and very slow population growth, but rather few observations of such pre-transition populations exist. Data for rural China in the 1920s indicate a probability of dying by age five of around 380 per thousand live births (Barclay et al. 1978); the infant mortality rate for Chile prior to 1920 is consistent with a $_5q_0$ of about 400; the $_5q_0$ observed for a rural village in the Gambia in the 1950s was close to 430 (McGregor et al. 1961); the value for the !Kung in the first half of the century was probably around 400. Since then, all regions of the developing world have experienced rapid declines in child mortality. Figure 6 shows the $_5q_0$'s estimated for each region of the developing world from the early 1950s to the early 1980s

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[5] A recent United Nations exercise to describe levels and trends in child mortality worldwide since 1950 could use civil registration data alone for only 17, and civil registration in combination with other data for a further 16, developing countries, out of a total of 124.

by a recent United Nations (1988) study. All regions have seen substantial declines. Africa, South Asia and Latin America have all experienced declines of at least 100 points over the three decades, the $_5q_0$ roughly halving over the period. East Asia, dominated by China, showed an even more dramatic decline in $_5q_0$, falling by 80 percent in three decades.

The trends in Figure 5 can be criticized on the grounds that, for many countries, neither levels nor trends of recent child mortality are known with any accuracy. However, an examination of trends in just those countries for which reasonably well substantiated estimates exist supports the broad trends in Figure 5 (Hill and Pebley 1989). The pace of child mortality decline in terms of percent change in $_5q_0$ seems to have been roughly constant over the period from the early 1960s to the early 1980s. Africa has tended to under-perform, but other regions show very similar rates of change. Age patterns of child mortality, and changes in such patterns, are rather similar to those found in Europe a century earlier; countries of the developing world have seen child mortality change in ways that would fit quite easily into Figure 2.

Even less is known about trends and structures of cause of death in the developing world. Cause of death data from Chile for the early part of the century suggest a rather lower burden of infectious and parasitic diseases and higher levels of respiratory ailments than for England and Wales, but the quality of the data is suspect (Preston et al. 1972). Early data from a tropical population, Taiwan, also show relatively high levels of respiratory mortality, but surprisingly low mortality from diarrhea, relative to European experience (Preston et al.,1972). Though the data are fragmentary, there can be little doubt that malaria is, or has been, an important cause of child mortality in much of the tropics, but particularly in Africa.

There is some controversy for developing countries as well as developed over the reasons for the sharp decline in child mortality in the last few decades, particularly focussing on the relative roles of development versus medical knowledge and practice. There are some important differences between developing country mortality declines and earlier declines in developed countries, such as the existence of lower child mortality in urban areas of LDC's, and the existence

of strong socio-economic differentials, particularly pronounced with female education. At the same time, there are very strong similarities, such as the apparent resistance to reversal in downward trends, and the age patterns of improvement. Perhaps the key difference for causal interpretation is in the link to economic development; whereas in Europe, child mortality fell during a period of unprecedented rise in per capita income, in LDC's, child mortality has fallen sharply in some countries, such as Sri Lanka and China, that have experienced very little increase in per capita income. Thus in the developing country case, economic growth per se can be ruled out as a major determinant of decline. The strong link of child mortality to mother's education suggests a very important role for diffusion of health-related knowledge and behavioral change. However, there can be little doubt that medical interventions, often consisting early in the decline of public health style vector control programs (such as in Sri Lanka) and consisting more recently of mass immunization programs have contributed substantially to the pace of decline. As a practical example, the village in Gambia reported above as having high child mortality in the 1950s continued to have high mortality until the mid-1970s. At that time, an intensive program of medical intervention and surveillance was introduced, and the probability of dying by age five dropped from about 250 per thousand to about 50 per thousand in the period 1974/75 to 1982/83 (Lamb et al.,1984). The authors attribute the pace of the decline largely to "regular, controlled surveillance of women [and children] by a physician and a qualified midwife...." Clearly, such intensive services are not generalizable, but they show what medical services can potentially achieve without much alteration in underlying living conditions.

Conclusions

Over the last three or four centuries, child mortality has fallen dramatically. In developed countries, this transition from high to low mortality is essentially complete; whereas prior to the decline, a child had only about a 60 percent chance of surviving to age five, today a newborn has better than a 99 percent chance of reaching age five. The decline occurred through reduced exposure to pathogens, largely as a result initially of public health measures, and later as a result of living in a healthier population, through improved nutrition and reduced crowding, and through specific medical preventive and

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therapeutic interventions. As the decline has progressed, the medical effects have become increasingly important. In developing countries, the transition is largely incomplete, though it has started everywhere. In a few countries in Africa, the risk of dying by age five is still over 300 per thousand; in parts of South Asia, it is still over 200 per thousand; in parts of Latin America, it is still over 150 per thousand. Practically everywhere, barring civil wars or natural disasters, it is declining at a fairly steady pace, and in some countries (Chile, Costa Rica) has reached levels of which Europe was proud only three decades ago. This decline is much more strongly associated with medical knowledge and medical practice than was the earlier decline in Europe. Indeed, the development and implementation of cheap and effective preventive measures has probably been the decisive factor in child mortality decline in LDC's over the last two decades.

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TABLE 1: Child Mortality (Probability of Dying by Age Five) in Europe, 1550 to 1849: Selected Historical Studies

| Period	British Peerage[a]		Geneva Bourgeoisie[b]		13 English Parishes[c]	Sweden (Registration)[d]	
	M	F	M	F	M+F	M	F
1550-74	265	221					
			423	406	N/A	N/A	N/A
1575-99	256	240					
1600-24	268	247					
			388	312	237	N/A	N/A
1625-49	314	382					
1650-74	338	323					
			393	308	254	N/A	N/A
1675-99	291	296					
1700-24	276	279					
			283	225	281	N/A	N/A
1725-49	273	278					
1750-74	192	187				N/A	N/A
			195	177	252		
1775-99	147	154				332	308
1800-24	142	123				313	284
			122	151	N/A		
1825-49	109	105				258	229

Sources:
[a] Hollingsworth (1965)
[b] Henry (1956); reported probabilities of dying by age 20 have been converted to probabilities of dying by age 5 using the Coale-Demeny 'North' Model Life Tables.
[c] Wrigley and Schofield (1983)
[d] Keyfitz and Flieger (1968)

FIGURE 1: Infant Mortality Rate, Sweden, 1751 to 1988, with Smoothed Median Trend.
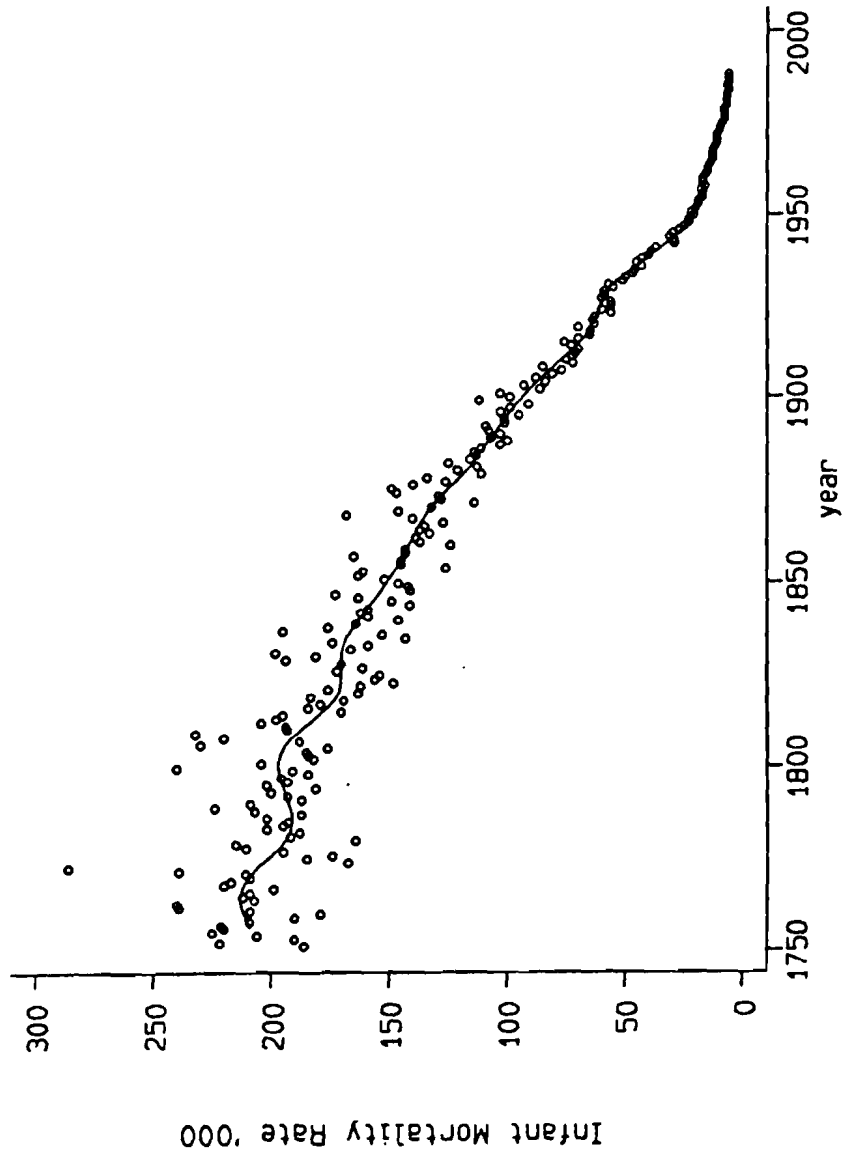
Source: Mitchell, B.R. (1975) and
United Nations Demographic Yearbooks

FIGURE 2: The Relations Between Probablilities of Dying in
Infancy and Between Ages 1 and 5 in the 19th and
20th Centuries by Sex: The Netherlands, France
and Sweden.

Key:
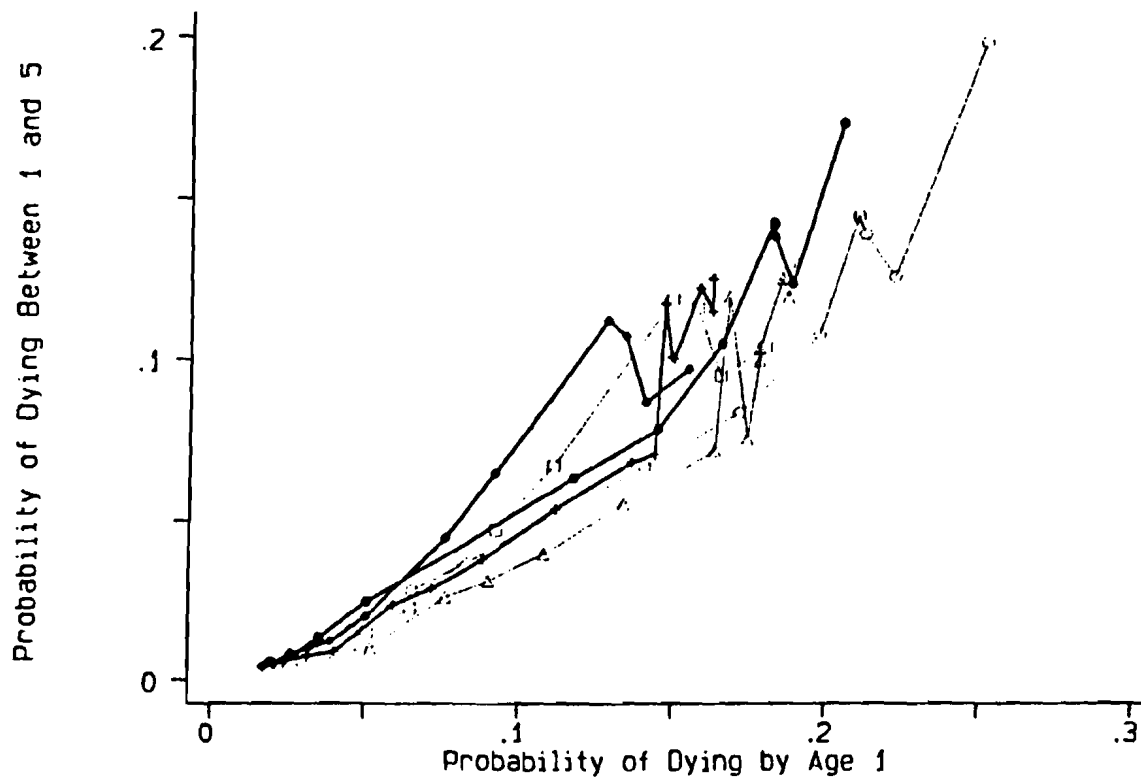
o Netherlands Male   • Netherlands Female
ʌ France Male   ✦ France Female
▫ Sweden Male   ◆ Sweden Female

FIGURE 3:   Indices (1861-71 = 1) of Mortality Rates in Infancy and
            Childhood by Major Cause of Death Group; Males, England
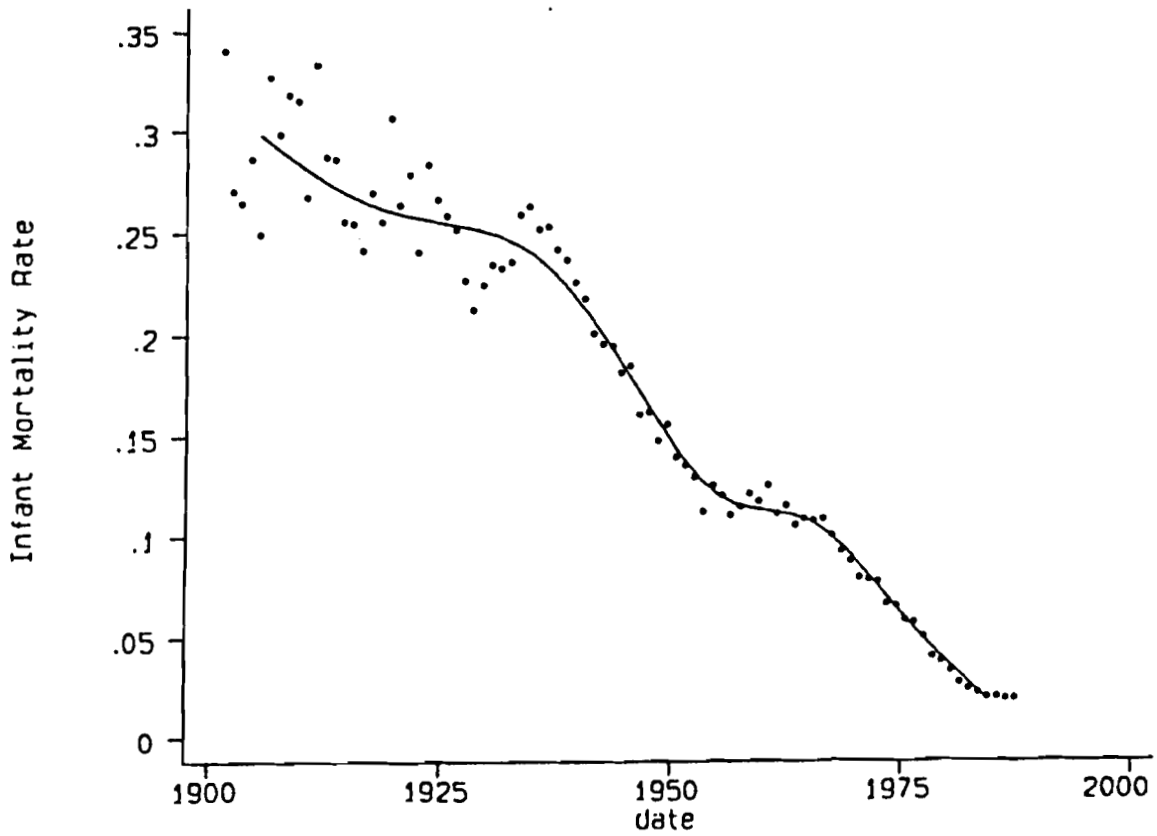   and Wales.

a) Infancy   b) Between Ages 1 and 5
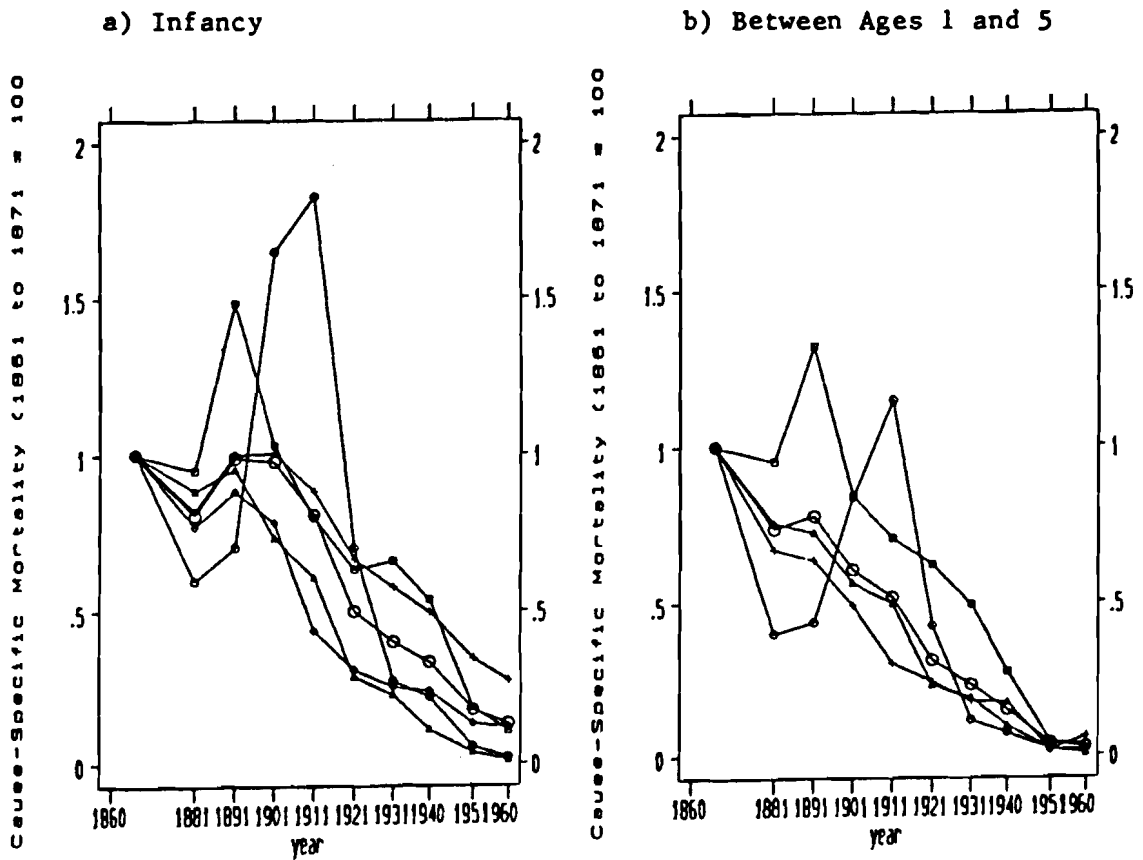
Key:
  o All Causes
  ▲ Infectious and Parasitic
  ▫ Respiratory
  ○ Diarrhea and Enteritis
  · Certain Diseases of Infancy ((a) only) ; All Others ((b) only)
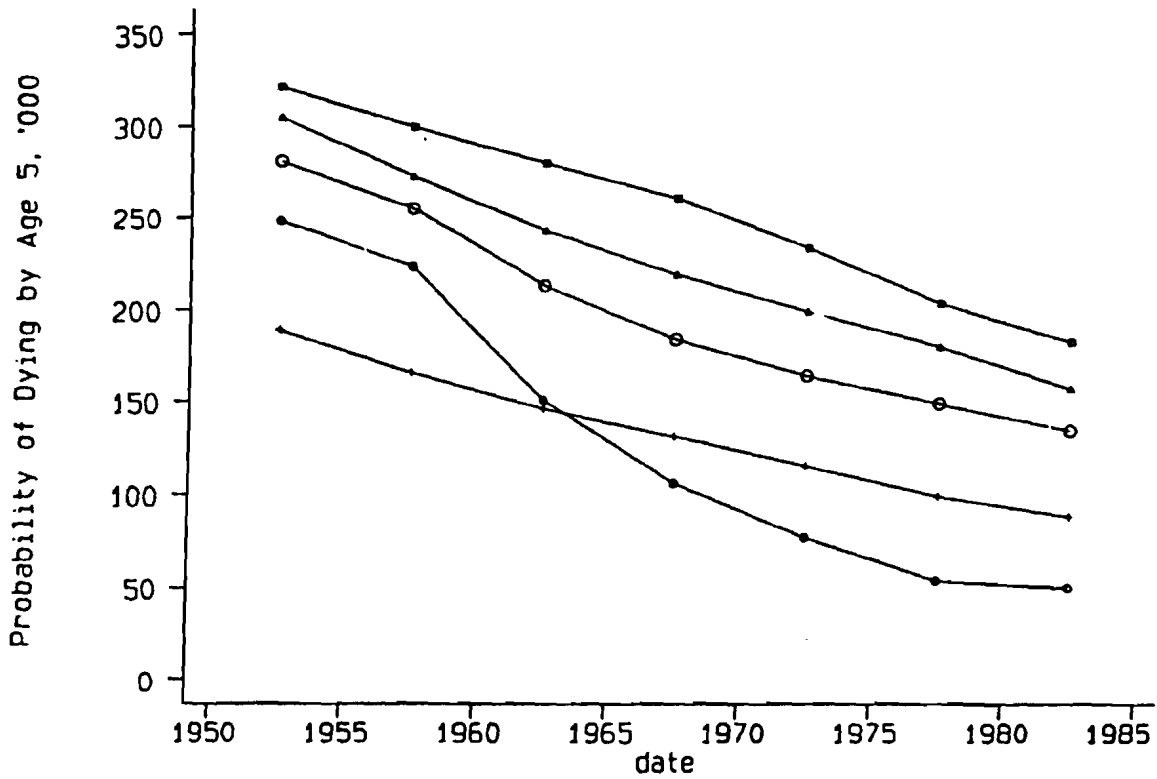 • All Others ((a) only)

Source: Preston et al. (1972)

FIGURE 4: Infant Mortality Rate, Chile, 1901 to 1987,
with Smoothed Median Trend

Source: Mitchell, B.R. (1983) and
United Nations Demographic
Yearbooks

FIGURE 5: Trends in the Probability of Dying by Age 5 in the Developing World; by Region, 1950-1985.

Key:
o   All LDC's
.   Latin America
o   East Asia
▴   South Asia
▫   Africa

Source: United Nations (1988)