Volume Title: Population and Economic Change in Developing Countries

Volume Author/Editor: Richard A. Easterlin, ed.

Volume Publisher: University of Chicago Press

Volume ISBN: 0-226-18027-1

Volume URL: http://www.nber.org/books/east80-1

Publication Date: 1980

Chapter Title: Causes and Consequences of Mortality Declines in Less Developed Countries during the Twentieth Century

Chapter Author: Samuel H. Preston

Chapter URL: http://www.nber.org/chapters/c9667

Chapter pages in book: (p. 289 - 360)

5 Causes and Consequences of Mortality Declines in Less Developed Countries during the Twentieth Century

Samuel H. Preston

Only a few countries of Africa, Asia, and Latin America can supply suitable data for estimating mortality levels in 1900. Many more can supply such data for 1940 or 1950. Without exception, the estimated levels of mortality prevailing in those years are higher than current levels. For those countries that can provide data at both earlier points, most improvement as indexed by life expectancy at birth has been achieved since 1940. It appears from fragmentary records that life expectancy at birth during 1935–39 was about 30 years in Africa and Asia and 40 years in Latin America. The respective levels in 1965–70 were on the order of 43, 50, and 60 (World Health Organization 1974*b*; United Nations, Population Division 1973).

The magnitude and the demographic character of this improvement have been documented in a number of excellent reviews, and for this reason they need not detain us here (United Nations 1963, 1973, 1974; Stolnitz 1974; Arriaga 1970; World Health Organization 1974*b*). These works suggest that the mortality improvements, when measured by the absolute decline in age-specific death rates, have tended to be largest at ages under 5 (especially infancy) and above 40. The proportionate declines, on the other hand, have been largest in the older childhood ages. Life expectancy gains for females have been larger than those for males. It is likely that gains have been more rapid in urban than in rural areas. In these matters, mortality experience in less developed countries

Samuel H. Preston is associated with the University of Washington.

The author is grateful to Avery Guest, James McCann, Peter Newman, and Masanori Hashimoto for comments and suggestions and to William Grady and Thomas Revis for research assistance. This research was aided by National Institutes of Health Center Grant 1POL HD 09397–01.

(LDCs) has roughly recapitulated that in more developed countries (MDCs). Life expectancy differences between MDCs and LDCs have narrowed, although the lagging pace of improvement in Africa has produced greater dispersion within LDCs themselves. The decadal rate of mortality decline in many LDCs surpasses that ever observed in populations of the now-developed world.

This paper has two purposes: to identify the factors responsible for these mortality improvements in LDCs and provide estimates of their relative importance; and to begin tracing the effect of these improvements on demographic and economic processes. Less developed countries are defined regionally to comprise Africa, Latin America, and Asia except Japan. Data on mainland China, North Korea, and what was formerly North Vietnam are not available, and for all practical purposes these countries are also excluded from the set under review. Conclusions reached about the importance of various factors in the mortality decline do not appear to conflict with the impressions of informed observers of these matters in China (Wegman, Lin, and Purcell 1973).

5.1 Causes of Declining Mortality

There is much more consensus on the fact of mortality decline in LDCs than on its causes. Considerable dispute remains about whether the decline has been principally a by-product of social and economic development as reflected in private standards of nutrition, housing, clothing, transportation, water supply, medical care, and so on or whether it was primarily produced by social policy measures with an unprecedented scope or efficacy. A third possibility is that technical changes reduced the relative costs of good health. This possibility is usually subsumed within the social policy position because it is clear to most observers that the major technical changes that have occurred—immunization against a host of infectious diseases, vector eradication, chemotherapy —had to be embodied in social programs in order to affect the mortality of the masses in LDCs. Demographers have almost unanimously favored the social policy–technical change interpretation of mortality decline (Davis 1956; Coale and Hoover 1958; United Nations, Population Division 1974; Stolnitz 1974). As evidence, they have principally cited the unprecedented rate of mortality reduction in many LDCs and certain dramatic examples of obviously effective government intervention, most notably in Sri Lanka and Mauritius. Many specialists in international health (Fredericksen 1961, 1966*a*,*b*; Marshall, Brown, and Goodrich 1971), medical historians focusing on primarily Western populations (McKeown and Record 1962; McKeown 1965), and some economists (Sharpston 1976) have opposed this interpretation, usually claiming

that social interventions have been largely ineffective or insufficiently widespread.

Kuznets (1975) and Coale and Hoover (1958) have argued that, in one sense, the distinction between economic development and public health interventions creates a false dichotomy. Development itself strengthens the nation-state, improves communications among nations and hence facilitates the transfer of medical technology, and routinizes scientific advance. While this position is unassailable, it leaves unanswered the question whether the mortality decline was a product of changes in private consumption or of public programs and technical change, regardless of whether the latter were in turn produced by economic development in its broadest sense. Even if public programs and technical changes were merely intervening variables in the relation between mortality and development, the importance of their role remains to be identified.

5.1.1 Effect of Private Income Levels on Mortality

That mortality rates are sensitive to private living standards, independent of the national level of economic development, scarcely needs documentation. Studies of mortality differentials among individuals by social or economic class in countries as disparate as India and the United States consistently reveal lower mortality rates among the upper classes (Kitagawa and Hauser 1973; Vaidyanathan 1972). The role of private living standards in creating the pattern of international mortality differentials is more difficult to assess. Richer countries not only have richer people but, in general, have larger and more effective social programs.

Some indication of the importance of private living standards for international mortality differences may be gained by examining the importance of income distribution as a factor in those differences. The international relation between national income per capita and life expectancy is decidedly nonlinear, with life expectancy showing strongly diminishing returns to increases in income (Preston 1975*a*; Vallin 1968). It is reasonable to expect that mortality also responds nonlinearly to individual income levels, in which case the distribution of income within a nation should influence its aggregate level of mortality. In particular, suppose that the relation between individual income and life expectancy is log linear:[1]

$$e^{o}_{0i} = a + b\ 1nY_i,$$

where e^{o}_{0i} = life expectancy at birth in income group i
Y_i = level of income received by group i
a, b = constants.

If the national level of life expectancy is simply the aggregate of these individual-level relations, with no contribution from the *national* level of income except insofar as it reflects individual incomes, then the life expectancy for the population, e^o_{0p}, will be equal to

$$e^o_{0p} = f[\sum_i (a + b\ 1nY_i)]$$

$$= a + b\ 1nY + b \times f \times \sum_i 1n\ (S_i/f),$$

where $Y =$ mean level of income in the population
$S_i =$ share of total national income earned by group i
$f =$ share of total population represented by group i, assumed to be constant among the groups.

Life expectancy will be a function of mean national income, Y, and of the distribution of income as represented by the term $\sum_i 1n(S_i/f)$. This term, which is related to the entropy measure of income distribution, ranges from 0 if income is perfectly evenly distributed to $(-\infty)$ if one group has no income. Strictly speaking, the weights that permit subgroup life expectancies to aggregate into population life expectancy are provided by births rather than by population size, but the two will be very highly correlated.

To examine the importance of private incomes for national life expectancy, the value of this income distribution measure was computed for fifty-two populations on which income shares were estimated in 5% population segments. The values and sources can be found in appendix table 5.A.2, along with values of e^o_0 and Y. The importance of private incomes can be inferred from the consistency of coefficients on Y and on the income distribution measure. If national life expectancy is simply a function of private income levels, the coefficients on $\sum_i 1n(S_i/.05)$ should be 0.05 of the coefficient on Y. If national income contributes independently of private income, the ratio should be less than 0.05. The equation as estimated by ordinary least squares on the fifty-two observations is

$$e^o_0 = 19.105 + \underset{(.859)}{6.984}\ 1n\bar{Y} + \underset{(.237)}{.375} \sum_i 1n(S_i/.05)$$

$$R^2 = .651$$

$$\bar{R}^2 = .644.$$

The coefficient of the income distribution term is in fact 0.0536 of that of national income, suggesting that relations between mortality and income at the national level are indeed dominated by relations between mortality and income at the individual level. This result should be treated with great caution because of inaccuracy and incomparability

in the measure of income distribution and because the log-linear functional form probably simplifies a more complex relationship. Furthermore, the standard error of the income distributional coefficient is large enough to prevent rejection of the hypothesis that the true coefficient is zero. Nevertheless, one direct implication of the result is that the mortality risks facing a family earning $10,000 per year or $100 per year are not strongly influenced by the prevailing level of average income in the nation in which they reside.

The suggestion that private incomes are very influential in determining national levels of life expectancy at a moment in time does not imply, of course, that changes in private incomes have been the dominant factor in mortality changes during this century. The actual changes in income may have been too small, in conjunction with the sensitivity of mortality to income, to account for the observed mortality changes. Before trying to establish the role played by changes in private living standards in LDC mortality declines, it is useful to make an assessment of the causes of death responsible for those declines.

5.1.2 Causes of Death Responsible for Mortality Declines

Interpretation of mortality declines in LDCs would depend on whether the cause of death responsible for the majority of declines were, for example, smallpox, diarrheal disease, or malaria, since it is clear that death rates from these causes are fundamentally responsive to different influences. Unfortunately, the causes of death responsible for mortality change in LDCs have never been documented on a broad scale. A large part of the reason is that most LDCs still cannot supply national-level data on cause patterns, and data for those that can undoubtedly reflect inaccurate diagnoses and incomplete coverage. Problems are magnified when attention is turned to the patterns of earlier years. Nevertheless, it is possible to piece together a picture that provides some useful clues about the order of magnitude of the causes responsible.

First, it is clear that, in high-mortality populations, infectious and parasitic diseases bear almost exclusive responsibility for shortening life below the modern Western standards of 69 years for a male and 75 for a female. Life tables by cause of death have been constructed for 165 populations at varying levels of mortality (Preston, Keyfitz, and Schoen 1972). When the aggregate of infectious and parasitic diseases were hypothetically eliminated from those life tables and life expectancy was recalculated, the common result was to produce a life expectancy between 65 and 70 for males and between 70 and 75 for females, regardless of a population's initial mortality level (Preston, Keyfitz, and Schoen 1973). In 1920, Chilean males would hypothetically have gone from a life expectancy of 28.47 to one of 65.68 and females from 29.85 to 69.76. In Taiwan, males would have enjoyed a life expectancy of

72.27 years instead of 26.68, and females 76.00 instead of 29.18 (Preston, Keyfitz, and Schoen 1972, pp. 150–51, 702–3).[2]

Despite the appeal of life table measures, they are an unnecessarily awkward vehicle for discussing causes of death because the causes are nonadditive in their effect on life table parameters. This problem is averted by the use of age-standardized death rates. Models have been constructed to represent the typical cause-of-death structure for populations at various levels of mortality as indexed by the age-standardized crude death rate from all causes combined (Preston and Nelson 1974). Of the 165 populations supplying data for these models, only 41 were from Africa, Asia, or Latin America, and of these 5 were for Japan and 3 for the Jewish population of Israel. Nevertheless, the results suggest that cause-of-death structures, controlling mortality level, vary less between MDCs and LDCs than they do among regional groups within MDCs. Lower cardiovascular mortality and higher mortality from diarrheal diseases and maternal causes in LDCs represent their only significant divergence from MDC patterns (Preston and Nelson 1974, p. 37). John Gordon, one of the leading epidemiologists whose work focuses on developing countries, states that "infectious disease in the tropics and in some other preindustrial areas is too often viewed elsewhere as a collection of odd processes peculiar to those regions. Such diseases as schistosomiasis, filariasis, paragonimiasis, and all the others do exist. The plight of children, however, [who account for the bulk of annual deaths in LDCs] is the result of the everyday infections of the intestinal and respiratory tracts and with the communicable diseases specific to early life everywhere" (Gordon 1969, p. 218). Even among adults, the exotic tropical diseases are typically much more important sources of morbidity than of mortality.

Thus, there is some justification for allowing LDC cause-of-death patterns to be represented by relationships calculated on the basis of a data set that includes both MDCs and LDCs. The typical cause structures pertaining to populations with age-standardized crude death rates of 0.035 and 0.020 are presented in table 5.1.[3] These are roughly the levels that probably best characterize the average mortality situation in LDCs in 1900 and 1970, since they correspond to life expectancies at birth of 27.5 and 50.[4]

What is perhaps surprising about the table is that the specific "name" infectious and parasitic diseases (the first two listed) account for only an estimated 26.1% of mortality change. More important than all of these diseases combined—tuberculosis, typhoid, typhus, cholera, measles, diphtheria, whooping cough, malaria—is the category of respiratory diseases, which comprises a wide assortment of respiratory difficulties that are concentrated largely in infancy and old age. To be sure, some of the deaths in this category are improperly assigned complications of

Table 5.1 **"Normal" Cause-of-Death Patterns at Standardized Crude Death Rates of 35/1,000 and 20/1,000 and Cause-Patterns of Change**

Cause of Death	Model Value (Mean, Male and Female) of Age-Standardized Crude Death Rate from Cause at Age-Standardized Crude Death Rate from all Causes Combined of		Percentage of Decline Attributable to Cause
	35/1,000	20/1,000	
Respiratory tuberculosis	3.85	1.42	9.5
Other infectious and parasitic diseases	4.00	1.51	16.6
Influenza/pneumonia/bronchitis	7.85	2.87	33.2
Diarrheal disease	2.32	1.34	6.5
Maternal causes	.27	.14	0.9
Certain diseases of infancy	1.37	.88	3.3
Violence	.89	.78	0.7
All other and unknown	15.45	11.06	29.3
Total			100.0

Source: Preston and Nelson (1974).

specific infectious diseases. After undergoing a careful review of the initial medical certification in 1962–64, death certificates assigned to influenza/pneumonia/bronchitis in ten cities in Latin America, San Francisco, and Bristol, England, suffered a net loss of 1.0% of the original deaths assigned at ages 15–74, while the total of specific infectious and parasitic diseases increased by 5% (Puffer and Griffith 1967, pp. 230, 235). In a similar study of deaths before age 5 in thirteen Latin American and two North American areas between 1968 and 1972, respiratory diseases forfeited 22% of their originally assigned deaths, while the specific infectious diseases gained 23% (Puffer and Serrano 1973, pp. 332, 342). Corresponding adjustment of figures in table 5.1 would equalize the contribution of the specific infectious diseases and of the respiratory diseases to mortality decline at about 28–30%. Nevertheless, the "name" infectious diseases remain relatively submerged compared with popular accounts of their role, a point also stressed by McDermott (1966). Part of the reason for overemphasis on the role of the "name" diseases in mortality decline is probably their preeminent importance in the relatively small-scale English decline from 1851 to 1901, as has been elegantly documented in a widely cited paper by McKeown and Record (1962).

Some indication of whether the patterns depicted in table 5.1 provide a suitable representation for LDCs can be gained by examining the few LDC records that are available. In so doing, it is useful to provide more

detail on specific members of the infectious and parasitic set. Mortality changes during long periods will be considered in order to avoid sampling periods during which specific public health interventions may have badly distorted the cause pattern of change.

Table 5.2 presents crude death rates from certain diseases of infectious origin for five populations of LDCs in the early twentieth century and for a more recent year. By and large, they support the previous estimate of the relative importance of respiratory tuberculosis and diarrheal diseases. Influenza/pneumonia/bronchitis is somewhat less important a source of declinc than depicted in table 5.1, but the reason is probably that by the latter date each of the four populations supplying information on this cause achieved a mortality level far superior to the 20/1,000 age-standardized rate assumed in table 5.1. This cause is generally a more important source of mortality decline in movements between high and intermediate levels than between intermediate and low ones (Preston and Nelson 1974, pp. 31–33). As a very shaky generalization based on these undoubtedly unrepresentative populations, it may not be too far off the mark to assign 2% of the twentieth-century mortality decline in LDCs as a whole to smallpox, 2% to whooping cough, and 1% each to typhoid, typhus, measles, cholera, and plague (the latter estimate accounting for the disease's heavy concentration in India), and 0.5% to diphtheria. The epidemic nature of typhoid, cholera, and plague add even more uncertainty to these figures.

But the major uncertainty relates to the role of malaria. If estimates for British Guiana and India are to be believed, malaria by itself has accounted for 18–35% of large-scale mortality declines, equaling or exceeding the contribution we have estimated for all infectious and parasitic diseases combined. It should be noted that both estimates are based upon assignment to malaria of an arbitrary portion of deaths originally ascribed to "fever." A less arbitrary approach was pursued by Newman (1965, 1970), who had access to regional mortality data before and after an eradication campaign in Sri Lanka, as well as to regional data on malarial endemicity. Newman estimates by indirect techniques that the malaria eradication campaign in 1946 reduced Sri Lanka's crude death rate (CDR) by 4.2/1,000 between 1936–45 and 1946–60 (1970, p. 157). The relative contribution of such a decline to the total mortality reduction depends, of course, on the size of the latter. Had it been experienced by one of the populations in table 5.2, where the average drop in CDR was 26/1,000, it would account for about 16% of the mortality reduction. As a component of the smaller decline during the shorter period considered by Newman in Sri Lanka, it represented 42%. Simple inspection of time-series data on crude death rates suggests that virtually complete malaria eradication reduced the crude death rate by

Table 5.2 **Crude Death Rates (per 1,000) by Cause in Certain LDCs in the Early Part of the Century and Recently**

Cause of Death	Chile, 1920[a]	Chile, 1971[b]	% of Decline	Taiwan, 1920[c]	Taiwan, 1966[d]	% of Decline
Typhoid	.665	.007	2.9	.045	0	.2
Typhus	.439	0	2.0	n.a.	—	—
Malaria	.026	0	0	2.123	0	7.7
Smallpox	.008	0	0	.065	0	.3
Measles	.697	.061	2.8	.358	.039	1.2
Whooping cough	.789	.006	3.5	.046	.004	.2
Diphtheria	.062	.006	.2	.013	.004	0
Influenza/pneumonia/ bronchitis	4.527	1.355	14.1	8.231	.613	27.8
Respiratory tuberculosis	2.404	.209	9.8	1.813	.333	5.4
Diarrhea, dysentery, cholera nostras	2.120	.397	7.7	1.691	.215	5.4
Cholera	n.a.	—	—	.452	0	1.6
Total	30.85	8.389		32.686	5.246	

[a]Chile, Oficina Central de Estadistica, *Annuario Estadistico*, vol. 1 (1920).

[b]World Health Organization, *World Health Statistics Annual*, vol. 1 (1972).

[c]Taiwan, Jinki Dotai Tokei, *Sotoku Kanbo Chosaka* (1920).

[d]United Nations, *Demographic Yearbook* (1967), table 24.

[e]Pani (1917, pp. 192–99).

[f]Mandle (1970, p. 303).

[g]United Nations, *Demographic Yearbook* (1973), table 33.

[h]Compiled from material in Davis (1951, pp. 33–53). The 8.7 malaria estimate assumes that one-third of fever deaths are due to malaria. All estimates are highly suspect because of very deficient coding. Data on cholera, plague, and smallpox are probably most accurate because they were most consistently "notifiable" causes of death at the provincial level. Coale and Hoover (1958, p. 67), using a completely different technique, estimate the pre–spraying program level of malaria death rates in India to be about 6/1,000, including deaths indirectly attributable to the disease.

[i]Crude death rate from United Nations, Population Division, *Selected World Demographic Indicators by Countries, 1950–2000* (1975), p. 125. Average, 1960 and 1965. Distribution of deaths by cause from United Nations, *Demographic Yearbook* (1967), table 24. Data are for medically certified deaths in Poona and Bombay corporations and deaths in public hospitals in Rajasthan. The World Health Organization provides some confirmation of the virtual eradication of malaria from India by noting a hundredfold decline in reported cases between 1952 and 1972. World Health Organization, *Fifth Report on the World Health Situation*, Official Records no. 225 (1975), p. 142.

[j]Bunle (1954).

[k]Deaths under age 2 only.

[l]Malaria and undefined fevers.

[m]Pneumonia and bronchitis.

[n]Tuberculosis, all forms.

Table 5.2 (continued)

Cause of Death	Mexico City, 1904–12[e]	Mexico, 1922–25[j]	Mexico, 1972[b]	% of Difference, Mexico City–Mexico	% of Decline, Mexico
Typhoid	.068	.341	.065	0	1.7
Typhus	1.363	.040	.001	4.1	.2
Malaria	.076	1.471	.001	.2	9.0
Smallpox	.733	.826	0	2.2	5.0
Measles	.290	.427	.219	.2	1.3
Whooping cough	.284	.938	.080	.6	5.2
Diphtheria	.169	.062	.002	.5	.4
Influenza/pneumonia/ bronchitis	7.838	3.213	1.664	18.6	9.4
Respiratory tuberculosis	2.485	.653	.152	7.0	3.1
Diarrhea, dysentery, cholera nostras	9.785	1.861[k]	1.337	25.4	—
Cholera	n.a.	n.a.	—	—	—
Total	42.314	25.489	9.081		

Cause of Death	British Guiana, 1911–20[f]	Guyana, 1967[g]	% of Decline	India, 1898–1907[h]	India, 1963[i]	% of Difference
Typhoid	n.a.	—	—	n.a.	—	—
Typhus	n.a.	—	—	n.a.	—	—
Malaria	4.185[l]	0	18.1	6.0–8.7	0	24.3–35.2
Smallpox	n.a.	—	—	.27	.08	.8
Measles	n.a.	—	—	n.a.	—	—
Whooping cough	n.a.	—	—	n.a.	—	—
Diphtheria	n.a.	—	—	n.a.	—	—
Influenza/pneumonia/ bronchitis	3.680[m]	.055	15.7	n.a.	—	—
Respiratory tuberculosis	1.432[n]	.024	6.1	n.a.	—	—
Diarrhea, dysentery, cholera nostras	2.780	.026	11.9	1.96	.14	7.4
Cholera	n.a.	—	—	1.66	1.18	1.9
Plague	n.a.	—	—	1.82	0	7.3
Total	30.049	6.987		43.5	18.8	

about 3/1,000 in Guatemala (Meegama 1967, pp. 231–33), by 5–9 points in Mauritius (Titmuss and Abel-Smith 1968, pp. 49–50), and by 2–3/1,000 in Venezuela (Pampana 1954, p. 504).

The importance of malaria reduction as a source of declining mortality in a country obviously depends upon initial endemicity and the success of antimalarial campaigns. Southern Africa, southern Latin America, and northern Asia were never seriously afflicted with the disease; malaria in tropical Africa is highly endemic but, with few excep-

tions, it has not been successfully attacked (World Health Organization 1975*a*). A valuable compilation by Faust (1941, p. 12) suggests that recorded crude death rates from malaria in Mexico, Central America, and the West Indies during the 1930s, before any inroads had been made against the disease in rural areas, was 1.66/1,000. But he considers this "a figure probably far too low." The recorded CDR from malaria in cities of Burma in 1939 was 2.14 (Simmons et al. 1944, p. 11). The preprogram malarial CDR in Venezuela was 1.73/1,000 (Pampana 1954, p. 504). But recorded levels are often greatly in error. Newman calculates for Ceylon that each death assigned to malaria represented approximately four deaths that were directly or indirectly caused by it. But such an inflation factor is simply not tenable in Guatemala or Venezuela, where contemporaneous declines in crude death rates and malarial crude death rates suggest that a 2:1 ratio is the most that could have been sustained. Part of the inflation factor reflects malaria's role as "the great debilitator," but another part may be spurious. Spraying with residual insecticides reduces not only malaria but other vector-borne diseases such as yellow fever, typhus, and especially diarrheal disease. As Newman points out, an indirect approach that bases estimates on relations between regional changes in aggregate mortality and changes in spleen rates may well overascribe mortality decline to malaria reductions (but not to insecticide campaigns themselves). Finally, there is an often-quoted estimate, the basis of which is unknown, that worldwide malarial deaths have declined from 2.5 million per year to less than 1 million (*Lancet* 1970, p. 599), figures suggesting for LDCs a decline in crude death rates on the order of 1/1,000. The 2.5 million estimate evidently first appears in Pampana and Russell (1955) and presumably applies to 1955. Russell put the figure at at least 3 million for 1943 (1943, p. 601).

There is obviously much work to be done on this issue. In the present state of semi-ignorance, it seems judicious to adopt a range of CDR declines of 2–5/1,000, within which the twentieth-century LDC mortality decline attributable to antimalarial programs seems to have a better than even chance of falling. Sri Lanka, the best-documented case, falls within this range, and Sri Lanka was apparently intermediate in terms of initial endemicity, although the program there probably enjoyed unusual success and malaria reduction may not be solely responsible for the mortality decline produced by insecticide spraying. The range is also consistent with the apparently widespread malarial CDR declines of 1.5–2.0/1,000 and with inflation factors of 1.4–2.5.

These assorted scraps of information are pieced together in table 5.3, where diseases that seem in the main to be responsible for mortality declines in LDCs between 1900 and 1970 are classified into three groups according to their dominant mode of transmission. An estimate—obvi-

Table 5.3 **Diseases Responsible for LDC Mortality Declines and Methods That Have Been Used against Them**

Dominant Mode of Transmission	Diseases	Approximate Percentage of Mortality Decline in LDCs, 1900–1970, Accounted for by Disease	Principal Methods of Prevention Deployed[a]	Principal Methods of Treatment Deployed[a]
Airborne	Influenza/Pneumonia/Bronchitis	30		Antibiotics
	Respiratory tuberculosis	10	Immunization; identification and isolation	Chemotherapy
	Smallpox	2	Immunization	Chemotherapy
	Measles	1	Immunization	Antibiotics
	Diphtheria, Whooping cough	2	Immunization	Antibiotics
		45		
Water-, food-, and fecesborne	Diarrhea, enteritis, gastroenteritis	7	Purification and increased supply of water; sewage disposal; personal sanitation	Rehydration
	Typhoid	1	Purification and increased supply of water; sewage disposal; personal sanitation; partially effective vaccine	Rehydration, antibiotics
	Cholera	1	Purification and increased supply of water; sewage disposal; personal sanitation; partially effective vaccine; quarantine	Rehydration
		9		
Insectborne	Malaria	13–33	Insecticides, drainage, larvicides	Quinine drugs
	Typhus	1	Insecticides, partially effective vaccines	Antibiotics
	Plague	1	Insecticides, rat control, quarantine	
		15–35		

[a]Major sources: Paul (1964); Morley (1973); Hinman (1966).

ously highly tentative but nevertheless the first that appears to have been offered—of the relative importance of the various diseases in the decline is also supplied. The category totals are somewhat more robust than the figures for individual diseases because diagnostic confusions, disease interactions, and program externalities are more likely to occur within groups than between groups. Since progress was very slow between 1900 and 1935, the listing may serve as an adequate representation of declines between 1935 and 1970 as well. Finally, some indication of the public health and medical instruments that have been deployed against the various diseases is also provided. The modes of transmission that are listed are not mutually exclusive or exhaustive, but the classification is not seriously distortive.

5.1.3 Influences Operating on the Various Causes of Death

Mortality from every disease listed in table 5.3 would be expected to decline when personal living standards rise. Of the many linkages, probably the most important are those between nutritional status and influenza/pneumonia/bronchitis, diarrheal disease, and respiratory tuberculosis. The mechanisms of effect are not well known, but it appears that protein malnutrition impairs the production of circulating antibodies in response to bacterial and viral antigens and that undernutrition can produce atrophy of the organs responsible for the immune response (World Health Organization 1972, pp. 24–25). There is no question of the importance of poor nutrition as a factor underlying high mortality rates in LDCs. The PAHO study of child mortality in thirteen Latin American projects found that immaturity or malnutrition was an associated or underlying cause of 57% of the deaths before age 5 (World Health Organization 1974*a*, p. 279). Immaturity is in turn a frequent product of maternal malnourishment (Mata et al. 1972). The problem is apparently equally severe in Africa, although the data are much more fragmentary (Bailey 1975). Diet supplementation programs in Peru (Baertl et al. 1970) and Guatemala (Ascoli et al. 1967; Scrimshaw 1970) significantly reduced child mortality in test populations, but, oddly, without having substantial effects on indexes of child physical development. Despite the improvement, mortality and morbidity in the Guatemalan villages remained "shockingly high," which was attributed to irregular participation in the feeding program and to the continued heavy burden of infection to which the children were subject (Scrimshaw 1970, pp. 1689–90).

But nutritional status is not exclusively determined by diet, nor is diet determined only by the availability of calories or protein. There is now extensive evidence that infectious diseases themselves are an extremely important source of malnourishment, independent of the child's nutritional state at the time of attack (Mata et al. 1972; Scrim-

shaw 1970; World Health Organization 1972, p. 27). Infection increases metabolic demands and often reduces the absorption of nutrients and increases their excretion. Nutritional intake can also be reduced by nausea or through customs denying food to the sick. Infections among pregnant women can reduce birthweights and, among new mothers, milk secretion (Mata et al. 1972; Bailey 1975). Gordon (1969, p. 218) suggests that diet fails by far to explain all the prevalent malnourishment in LDCs. The frequency of inappropriate nutritional practices despite adequate food supplies does not require emphasis (Bailey 1975; Food and Agriculture Organization 1975). The importance of nutritional practices is indicated by the reported reversal of expected social class differences in infant mortality in Chile, a condition attributed to earlier weaning among children of upper-class women (Plank and Milanesi 1973). The point is simply that, even if nutritional status were the only influence on mortality from a disease, mortality declines from that cause do not necessarily imply that an improvement in food supplies has occurred. Nutritional practices and exogenous declines in the incidence of other infections must also be considered candidates for explanation. The "subsistence level" of food production is obviously fictitious if it is presumed to represent a fixed requirement that is independent of the state of prophylactic or nutritional arts.

Despite the undoubted influence of nutritional intake and other components of general living standards on mortality, it is clear from table 5.3 that many other influences have also been at work. Obvious as it may be, it is easy to forget that death from an infectious disease involves an encounter between a pathogenic organism and a vulnerable human host. The rate of death can be altered by changing the rate or terms of the encounter without any prior change in the host. It is not possible and is probably not necessary to document individually the preventive and curative measures that have been utilized for this purpose. With the exception of influenza/pneumonia/bronchitis, it seems likely that preventive measures have been more effective than curative ones.

That preventive measures have been widely deployed in each of the three categories can be demonstrated relatively easily. At the end of 1964, 1.935 billion persons lived in areas that were originally malarious. Of these, 41% were living in areas from which malaria had been eradicated; 16% were living in areas where incidence was very low and was being controlled by case detection and treatment; 24% were living in areas protected by extensive mosquito control measures; and 19% were living in areas without specific antimalarial measures, most of these in tropical Africa (World Health Organization 1975*a*). The cost of programs producing virtually complete eradication has been estimated at 10–30 cents per capita per year, with programs probably extending over a two- to three-year period and a continuing annual cost of 5 cents per

capita required for surveillance thereafter (Pampana and Russell 1955, table 1, provides the most complete cost compilation).

India has vaccinated 170 million persons against tuberculosis and in 1968 alone vaccinated 83 million against smallpox (World Health Organization 1975*b*, p. 142). It has finally succeeded in eliminating smallpox completely, as has every other country. Colombia dispensed 5.9 million vaccinations in 1972, one-third of the population size; Egypt vaccinated 15 million persons against cholera in the same year and 25 million against smallpox in 1970. All primary-school entrants in the Philippines are vaccinated against tuberculosis. Barbados has compulsory immunization against diphtheria, polio, smallpox, and tetanus upon school entrance (World Health Organization 1975). An expert committee assembled by WHO estimated that 80% of the 70 million children reaching age one in LDCs each year could be immunized against measles, polio, tuberculosis, pertussis, tetanus, diphtheria, and smallpox at a cost, exclusive of personnel, of $37.5–$60 million, or $0.67–$1.07 apiece (World Health Organization, 1975*c*, p. 2).

Conditions of water supply and sewage disposal have also been markedly improved, despite continued abysmal conditions in many areas. Many of the improvements were normal and integral parts of economic expansion and hence cannot be specifically interpreted as public health interventions of an unprecedented sort. WHO surveys of government officials in 1962 and 1970 indicated that the proportion of LDC urban populations served by house connections to public water supply increased from 0.33 to 0.50 during this eight-year period for the seventy-five countries that replied in both years (World Health Organization 1973, p. 726). No rural population comparisons could be made, but in 1970 only 12% of rural LDC populations had "reasonable access" to community water supply (public fountain or standpipe within 200 meters of a house). The figure was only 6% in India (World Health Organization 1973, pp. 727, 729). New urban house connections were estimated to cost an average of $35 per capita, and providing rural residents with easy access to safe water to cost $12 per capita. In 1970, 69% of LDC urban populations had sewage disposal facilities (27% were connected to public sewerage and 42% had private household systems). New connections to urban public sewerage cost an average of $29 per head. Only 8% of the rural population was judged to have adequate sewage disposal, although the average cost of providing such facilities was estimated at only $4 per capita (World Health Organization 1973, pp. 732–33, 738–43). No trends in sewage facilities could be established, but improvement is probably fairly rapid in urban areas and slow in rural areas. Clearly, the initial cost of such programs is considerably higher than that for programs of vector control and immunization. Water supply improvements were among the very first changes to modify mor-

tality patterns in European countries in the mid-nineteenth century. But they are lagging relative to other improvements in LDCs. This may explain why diarrheal disease remains relatively more prominent as a contributor to total mortality in LDCs than it was in European countries at the same general mortality level (Preston and Nelson 1974).

With the exception of water and sewerage improvements and smallpox vaccination, the techniques of preventive and curative health care that have been widely deployed in LDCs are twentieth-century products. Virtually all were facilitated by ultimate acceptance of the revolutionary germ theory of disease at the turn of the century. Even smallpox eradication has benefited from technical improvements such as freeze-dried vaccine and the forked needle (Foege et al. 1975). The next section attempts to identify the relative importance of these technical improvements, as typically embodied in government health programs enjoying some measure of external assistance or support, in LDC mortality improvements during the last three decades.

5.1.4 Structural Changes in Relations between Mortality and Other Development Indexes

That mortality reductions have not merely been residual by-products of socioeconomic development is best illustrated by showing that major structural changes have occurred in the relationship between mortality and other indexes of development. Important technical changes and exogenous increases in government health commitment or foreign health assistance should result in a shift in the average level of life expectancy that corresponds to a particular level of other development indicators. Preston (1975*a*) suggested that such a change had occurred in the relation between mortality and national income, and this section will attempt to supplement that observation by introducing new variables and a larger sample of countries. Data have been gathered on national levels of life expectancy, per capita income (in 1970 U.S. dollars), daily calorie consumption, and literacy for thirty-six nations in or about 1940, including seventeen LDCs and several others that today would be classified as LDCs if 1940 conditions had persisted. The LDC estimates of life expectancy are based largely upon indirect demographic techniques such as intercensal survival analysis rather than upon vital statistics. Data on these same variables have been generated for 120 nations in or around 1970. The data and sources are presented in appendix tables 5.A.1 and 5.A.2.

A preliminary indication that structural changes have occurred is presented in table 5.4. Countries are cross-classified by level of per capita national income (in 1970 U.S. dollars) and by daily calorie consumption per head. Complete information was available for only twenty-nine countries in 1940. Nevertheless, it is clear that, within every

Table 5.4 **Mean Life Expectancy at Birth of Countries in Various Ranges of National Income and Calorie Consumption, 1940 and 1970**

Daily Calories Per Capita	National Income per Capita in 1970 U.S. Dollars									
	<150		150–299		300–699		700+			
<2,100	42.7	(17)	51.5	(8)	53.3	(5)	69.5	(1)	47.5	(31)
	38.3	(5)	36.0	(1)		(0)		(0)	37.9	(6)
2,100–2,399	42.6	(16)	49.9	(14)	56.2	(7)	71.4	(2)	49.1	(39)
	40.0	(1)	43.9	(2)	46.1	(1)		(0)	43.4	(4)
2,400–2,899	45.4	(1)	57.9	(8)	61.3	(10)	68.0	(7)	61.4	(26)
		(0)	44.1	(2)	50.4	(4)	59.6	(2)	51.1	(8)
2,900+		(0)		(0)		(0)	71.6	(24)	71.6	(24)
		(0)		(0)	58.7	(2)	65.2	(9)	64.0	(11)
	42.7	(34)	52.4	(30)	57.8	(22)	70.8	(34)	55.9	(120)
	38.6	(6)	42.4	(5)	52.2	(7)	64.1	(11)	52.2	(29)

Source: Appendix tables 5.A.1 and 5.A.2.
Note: 1970 countries appear in the top rows, 1940 countries in the bottom rows. The number of countries is shown in parentheses.

one of the nine cells where both 1940 and 1970 populations appear, average life expectancy was higher at the later date. The average intracell gain is 8.7 years of life expectancy.

A somewhat more precise indication of the magnitude of structural changes can be obtained by regressing life expectancy on income, calories, and literacy separately for 1940 and 1970 observations. Because of nonlinearities expected on obvious inductive and deductive grounds, natural logarithms of calorie consumption and income are used as regressors. Daily calorie consumption is measured from 1,500, approximately the average level required to meet minimum daily metabolic demands. Literacy, a personal dichotomous variable, cannot act nonlinearly at the individual level, since it takes on only two values. Barring spillover effects whose existence in income relations was called into question in section A, the proportion literate should be linearly related to life expectancy at the aggregate level. No claim is made that the resulting equations are perfectly specified, but simply that the socioeconomic variables included are the only ones available in the 1930s. It seems unlikely that relations between terms omitted and terms present

would have changed in such as way as to influence the outcomes described below.

The equations as estimated by ordinary least squares are the following:[5]

$$1970: \quad e^o_0 = \underset{(7.4090)}{17.1464} + \underset{(.6524)}{4.2488} \times 1n\bar{Y} + \underset{(.0212)}{.2086} \times LIT + \underset{(1.3492)}{.3170} \times 1n\,CAL$$

$$N = 120, R^2 = .860$$

$$R^2 = .858$$

$$1940: \quad e^o_0 = \underset{(18.5102)}{-13.1035} + \underset{(2.3860)}{5.4352} \times 1n\bar{Y} + \underset{(.0626)}{.1654} \times LIT + \underset{(3.7176)}{2.9470} \times 1n\,CAL$$

$$N = 36, R^2 = .856$$

$$R^2 = .845,$$

where e^o_0 = life expectancy at birth, average male and female
$\bar{Y}$ = national income per capita, 1970 U.S. dollars
LIT = percentage literate of the adult population
CAL = excess of daily calorie consumption per capita over 1,500.

Coefficients of all three variables in both equations are properly signed. The explanatory power of the regression equations is virtually identical for the two years. Income and literacy terms are highly significant in both periods and retain approximately the same magnitude. This stability was unexpected because of the high degree of colinearity among regressors. The coefficients indicate that a 10 percentage point increase in literacy is associated at both points with a gain in life expectancy of approximately 2 years, and that a 10% gain in national income by itself increases life expectancy by approximately one-half year. Coefficients of the calorie term decrease over time but are insignificant in both periods. It is very unlikely that the availability of calories for daily consumption has no influence on mortality. The calorie variable is probably subject to greater measurement error than the other two, and the influence of calorie availability is probably being reflected through them. The constant term increases by about 30 years between 1940 and 1970, although by itself this change is not readily interpreted, since the zero-points on variables are well below the range of observed experience. The hazards of extrapolation are shown by the negative (though insignificant) intercept for 1940.[6]

The substantive significance of the structural shift, as reflected primarily in the intercept, is probably best illustrated in the following way. Each of the 120 countries in 1970, including 94 LDCs, can supply estimates for each of the three regressors. It is therefore possible to estimate what life expectancy would be for every country at its current developmental level if no structural change had occurred in the relation between mortality and socioeconomic development. This estimate is simply obtained by substituting values of the three regressors for 1970 into the 1940 regression equation. Differences between actual life expectancy and that predicted if 1940 relations had continued to prevail indicate the amount of change in life expectancy attributable to the structural shift. A weighted average of such differences will indicate the importance of the shift for LDCs as a whole.[7] Results of this exercise are presented in table 5.5.

Estimates presented in this table indicate that life expectancy for LDCs as a unit (exclusive of China, North Korea, and North Vietnam) would have been 8.66 years lower in 1970–75 if life expectancy had continued to be related to other development indexes as it was in 1940. Excluding South Vietnam, where special factors were obviously distorting life expectancy, the figure is 8.84. This is an estimate of the amount of increase in life expectancy that is attributable to factors exogenous to national levels of income, literacy, and calorie consumption.[8] What fraction of the total gain in life expectancy during the period this 8.84-year structural shift represents is difficult to assess. WHO estimates that life expectancy in LDCs was 32 years in 1935–39 (30 in Africa and Asia and 40 in Latin America) and 49.6 years in 1965–70 (World Health Organization 1974, p. 23). The earlier figure is based on very little information, but if we accept it, the implication is that the structural change accounts for about half (50.2%) of the total gain in life expectancy during these nearly equivalent 30-year periods. This estimate is lower than the 79.5% (9.7/12.2) figures estimated for MDCs and LDCs combined by Preston (1975, p. 238) not so much because the estimated Δe°_{0} attributed to structural shifts differ (the difference is in fact only 0.86 years) but because the estimated gains in life expectancy differ (12.2 years for the world as a whole between 1938 and 1963 by Preston versus 17.6 years for LDCs between 1935–39 and 1965–70 by WHO). Part of the discrepancy in the estimates probably results from differences in the universe covered. Because MDCs had achieved by 1940 levels of developmental indicators high enough that relatively little gain in life expectancy was to be expected from advances in living standards, it is likely that exogenous factors represented a larger fraction of the gains that occurred there than they did in LDCs. Another part may reflect differences in the periods covered, since the present estimate pertains to a somewhat later period. Suggestions that the pace of mor-

Table 5.5 **Life Expectancy in 1970–75 and Life Expectancy Predicted if 1940 Relations between Life Expectancy and Levels of Literacy, Income, and Calorie Consumption Had Continued to Prevail**

Africa	Predicted e^0_0*	Actual e^0_0	Difference	*Latin America*	Predicted e^0_0*	Actual e^0_0	Difference	*Asia*	Predicted e^0_0*	Actual e^0_0	Difference
Algeria	41.42	53.20	11.78	Argentina	61.84	68.20	6.36	Afghanistan	30.16	40.30	10.14
Angola	37.32	38.50	1.18	Bolivia	39.49	46.80	7.31	Bangladesh	31.49	35.80	4.31
Botswana	34.13	43.50	9.37	Brazil	50.85	61.40	10.55	Burma	41.23	50.00	8.77
Burundi	40.39	39.00	−1.39	Chile	56.62	62.60	5.98	Cyprus	56.21	71.40	15.19
Central				Colombia	51.36	60.90	9.54	India	40.11	49.50	9.39
African Rep.	35.16	41.00	5.84	Costa Rica	56.07	68.20	12.13	Indonesia	39.54	47.50	7.96
Chad	30.29	38.50	8.21	Dominican Rep.	48.38	57.80	9.42	Iran	43.23	51.00	7.77
Congo	42.47	43.50	1.03	Ecuador	47.14	59.60	12.46	Iraq	42.57	52.70	10.13
Dahomey	33.60	41.00	7.40	El Salvador	45.09	57.80	12.71	Israel	63.10	71.00	7.90
Egypt	41.95	52.40	10.45	Guatemala	43.86	52.90	9.04	Jordan	43.59	53.20	9.61
Ethiopia	30.39	38.00	7.61	Guyana	51.21	67.90	16.69	Khmer Rep.	47.19	45.40	−1.79
Gabon	41.65	41.00	−.65	Haiti	29.48	50.00	20.52	Korea, Rep. of	52.20	60.60	8.40
Gambia	33.47	40.00	6.53	Honduras	44.10	53.50	9.40	Laos	32.95	40.40	7.45
Ghana	43.10	43.50	.40	Jamaica	54.93	69.50	14.57	Lebanon	55.11	63.20	8.09
Guinea	30.43	41.00	10.57	Mexico	55.24	63.20	7.96	Malaysia	50.43	59.40	8.97
Ivory Coast	41.97	43.50	1.53	Nicaragua	48.51	52.90	4.39	Nepal	31.63	43.60	11.97
Kenya	37.37	50.00	12.63	Panama	55.47	66.50	11.03	Pakistan	37.05	49.80	12.75
Liberia	35.68	43.50	7.82	Paraguay	47.84	61.90	14.06	Philippines	47.95	58.40	10.45
Libyan Arab Rep.	52.23	52.90	.67	Peru	46.72	55.70	8.98	Saudi Arabia	42.56	45.30	2.74
Madagascar	39.55	43.50	3.95	Puerto Rico	59.60	72.10	12.50	Singapore	55.22	69.50	14.28
Malawi	33.05	41.00	7.95	Trinidad				Sri Lanka	46.86	67.80	20.94
Mali	28.99	38.00	9.01	and Tobago	54.17	69.50	15.23	Syria	44.14	54.00	9.86
Malta	58.07	70.80	12.73	Uruguay	59.54	69.80	10.26	Taiwan	54.32	69.40	15.08
Maritius	49.47	65.50	16.03	Venezuela	55.14	64.70	9.56	Thailand	48.09	58.00	9.91
Mauritania	33.50	38.50	5.00					Turkey	49.03	56.90	7.87

Table 5.5 (continued)

Africa	Predicted e^0_0*	Actual e^0_0	Difference	*Latin America*	Predicted e^0_0*	Actual e^0_0	Difference	*Asia*	Predicted e^0_0*	Actual e^0_0	Difference
Morocco	39.92	52.90	12.98	Mean difference, Latin America			= 10.90	Vietnam, Rep. of	49.08	40.50	−8.58
Mozambique	38.98	43.50	4.52					Yemen	31.53	44.80	13.27
Niger	30.20	38.50	8.30	1970 population-weighted mean difference, Latin America			= 9.54	Yemen, P.D.R.	31.79	44.80	13.01
Nigeria	37.52	41.00	3.48								
Rhodesia	38.39	51.50	13.11								
Rwanda	29.66	41.00	11.34								
Senegal	36.25	40.00	3.75								
Sierra Leone	35.25	43.50	8.25								
Somalia	28.37	41.00	12.63								
South Africa	50.59	51.50	.91								
Sudan	34.70	48.60	13.90								
Togo	33.93	41.00	7.07								
Tunisia	42.32	54.10	11.78								
Uganda	37.62	50.00	12.38								
Cameroon	45.40	41.00	−4.40								
Tanzania	32.00	44.50	12.50								
Upper Volta	28.92	38.00	9.08								
Zaire	37.16	43.50	6.34								
Zambia	45.02	44.50	−.52								
Mean difference, Africa			= 7.05	Mean, all LDCs			= 8.61	Mean difference, Asia			= 9.14
1970 population-weighted mean difference, Africa			= 7.22	1970 Population-weighted mean, all LDCs			= 8.66	1970 population-weighted mean difference, Africa			= 8.90

*Based on substitution of 1970 values of literacy, income, and calorie consumption into 1940 regression relating e^0_0 to these variables.

tality decline in LDCs has slowed in the past decade (Hansluwka 1975; World Bank 1975) imply that the shift in the mortality/development relation may have essentially ended by the early 1960s, while gains in living standards continue to exert an influence on mortality. In any case, the estimated amount of the structural shift is consistent between the two estimates at about 9 years of life expectancy at birth.

The structural shift has evidently been least pronounced for African countries and most pronounced for Latin America. Africa has unquestionably experienced the least penetration by modern public health measures of any region. The problem is not simply poverty but also a widely dispersed population that increases program costs (World Health Organization 1975*b*, p. 17). Several of the African countries have lower life expectancies in 1970–75 than could have been expected based on 1940 relations. The apparent advantage enjoyed by Latin American countries may be due to their special relations with the United States. The United States has been by far the largest bilateral donor in international health aid, and the bulk of aid appears to go to Latin American countries, either directly or through the Pan American Health Organization (World Bank 1975, pp. 68–69). It is worth noting that, of the ninety-four LDCs, Sri Lanka and Mauritius are two of the four whose estimated structural changes are largest. It is unfortunate that so much attention has focused on these unusual cases.

Attributing to all countries the relations prevailing in countries for which data are available is always risky. The preceding analysis of change can be complemented by one that focuses exclusively on the cases that can be documented. Each of the thirty-six countries providing data in 1940 can also supply data in 1970. According to the previous formulation, we should expect Δe^{o}_{0} to be linearly related to $\Delta 1nY$, $\Delta 1n$ *CAL*, and ΔLIT, with a relatively large positive intercept reflecting the structural shift. In the first specification of the model, we add three terms believed to reflect factors responsible for a portion of the structural shift. The first (*MAL*) is an estimate of the degree of malarial endemicity in 1940, which is a proxy for the effect of antimalarial programs on Δe^{o}_{0}. Each of the thirty-six countries with endemic malaria has had a major antimalarial campaign. It is hoped that the coefficient of this term will provide a clearer indication of the effect of antimalarial activities on gains in life expectancy than was previously available. The second (*AID*) is an estimate of the average annual per capita nonmilitary aid in United States dollars received from bilateral and multilateral donors between 1954 and 1972. The third (*WAT*) is an estimate of per capita aid received for water and sewerage projects between 1965 and 1970 (U.S. dollars). The latter two variables are assumed to be proxies for the amount of total per capita health aid received between 1940

and 1970. Their values are generally highest for the Latin American countries. Values of these variables are presented in appendix table 5.A.3.

The estimated equation with all six terms present is the following:[9]

$$\Delta e^{o}_{0} = \underset{(2.8468)}{6.5212} + \underset{(2.4111)}{3.4500} \times \Delta 1n\bar{Y} + \underset{(.0927)}{.0354} \times \Delta LIT$$

$$+ \underset{(4.9362)}{.5605} \times \Delta 1n\, CAL$$

$$+ \underset{(.9411)}{3.1328} \times MAL + \underset{(.2376)}{.1460} \times AID + \underset{(.3668)}{.1955} \times WAT$$

$$R^2 = .595$$

$$\bar{R}^2 = .506.$$

Each of the coefficients has the expected positive sign. The coefficient of income remains similar in absolute value to that estimated in the cross-sectional regressions, but that of literacy declines by a factor of five and calories remain an insubstantial factor. Receipt of external aid contributes positively but insignificantly to mortality improvement. The most interesting result refers to the constant term. For a country essentially free of malaria in 1940 ($MAL = 0$), it is estimated that life expectancy would have increased by 6.52 years in the absence of socioeconomic development and external aid during the three decades. For a country in which malaria was highly endemic ($MAL = 3$), the corresponding gain is 15.92 years, of which 9.40 is attributable to factors associated with malarial endemicity. The average life expectancy for the seventeen LDCs was 39.29 in 1940 and 59.42 in 1970, giving an average gain of 20.13 years. The average malaria endemicity score for these seventeen was 2.59. Of the total gain in e^{o}_{0}, 8.11 years (3.1328×2.59), or 40%, is attributable to factors associated with malarial endemicity, and the constant term of 6.5 years, or 23%, represents other exogenous factors. The sum of 72% is considerably higher than that implied by the previous procedure. The external aid terms contribute an additional 1.13 years, or 5.6%.[10]

Whether or not antimalarial programs themselves produced the gain of 8.11 years attributed to malarial endemicity remains in serious doubt. The malaria score is correlated with life expectancy in 1940 at —.873. It is thus acting as a proxy for the initial level of mortality from a host of potentially eliminable infectious and parasitic diseases. When the initial level of life expectancy is entered as an independent variable, the magnitude of the structural change remains roughly the same but the portion attributable to the malarial term declines to zero:

$$\Delta e^{o}_{0} = \underset{(8.5108)}{31.4722} + \underset{(2.0533)}{3.6048} \times \Delta 1nY + \underset{(.0790)}{.0430} \times \Delta LIT$$
$$- \underset{(4.2327)}{.9865} \times \Delta CAL$$
$$- \underset{(1.3059)}{.0211} \times MAL + \underset{(.2036)}{.0750} \times AID + \underset{(.3139)}{.2939} \times WAT$$
$$- \underset{(.1328)}{.4063} \times e^{o}_{0}(1940)$$
$$R^2 = .720$$
$$R^2 = .643.$$

Other coefficients are not affected in such a way as to substantially alter interpretations, but the coefficient of malarial endemicity becomes effectively zero. The amount of structural change for the seventeen LDCs, the gain that is not accounted for by changes in Y, LIT, or CAL, is 16.46 years of life, or 81.8% of the average gain during the period.[11] The estimated structural change is close to that estimated directly above, but malaria's role in it is now negligible. When malarial endemicity is operationalized as a series of dummy variables, none of the dummy coefficients is significant, and the relation between mortality change and endemicity is nonmonotonic. Other functional forms and variable operationalizations should be investigated; at the moment all we can conclude is that the longitudinal analysis provides no better fix on malaria's role than the largely inconclusive cause-of-death analysis.

Two estimates have been advanced of the fraction of LDC gains in life expectancy between 1940 and 1970 that are attributable to structural change. The first estimate of one-half was based on a regression-decomposition technique that assumed all nations had relations between mortality and development indexes in 1940 identical to those prevailing in nations that could supply data for that year. The second estimate of approximately 80% was based solely upon examination of trends in the latter group. There is an important technical reason to favor the former estimate, namely, that measurement error is likely to be a more important source of distortion in longitudinal than in cross-sectional data. Measurement error biases coefficients toward zero. If random measurement error were all that was reflected in our measured changes in income, literacy, and calorie consumption, then the entire change in life expectancy would be absorbed in the constant term and attributed to structural change, regardless of the actual importance of these factors. Development levels are undoubtedly better measured than development rates, giving greater stability to the analysis based on a comparison of cross sections. There are, however, indications that a fraction of the gain

larger than one-half would be attributed to structural shifts if analysis had focused more narrowly on the period between 1940 and 1960.

The estimate of one-half is roughly consistent with the preceding cause-of-death analysis. Influenza/pneumonia/bronchitis has accounted for perhaps a third of the mortality decline. No effective preventive measures have been deployed against these diseases, the effectiveness of immunization being minimal, and there are suggestions that antibiotics, sulfa drugs, and curative services are not widely enough available in LDCs to have substantially altered the disease picture (Sharpston 1976; Bryant 1969, pp. 314–23). Diarrheal diseases probably account for another 9% or so of the decline, and the principal method of control has been improvements in water supply and sewerage that, because of their expense, are closely associated with economic development.[12] It is likely that social and economic development—especially as reflected in water systems, nutrition, housing, and personal sanitary knowledge—have operated largely through these diseases. In the case of other diseases it appears that programs of a narrowly public health nature that have embodied inexpensive new techniques, especially vector control and immunization, have been the decisive forces in mortality reductions.

5.1.5 The Role of MDCs in LDC Gains

Many have argued that MDCs have played a decisive role in the mortality declines experienced by LDCs, although the case has not been well documented. Certain of the influences are clear enough. Sulfa drugs, antibiotics, and most vaccines and insecticides, including DDT, have been developed in laboratories within MDCs. MDCs contributed 5,764 technical assistance workers in health services to LDCs in 1968 (Organization for Economic Cooperation and Development, n.d., pp. 276–77). Governmental health agencies were often created under colonial auspices. The role of external financing has also been stressed, but the accounts have focused on the dramatic examples in relatively small countries where international campaigns have often been undertaken largely for their demonstration value.

Health assistance in the developing world began with the work of medical missionaries, who were established in the Philippines in 1577 and in China by 1835 (Maramag 1965; Bowers 1973).[13] The early efforts of colonial governments were designed primarily to protect the colonials from epidemic diseases (Beck 1970). Correspondingly, cooperative international health efforts principally attempted to protect Europe and North America from imported cholera, plague, and yellow fever (Howard-Jones 1974). An evolving social conscience in the interwar years led to greater concern with the health of the native population itself and to the establishment of local medical colleges (Beck 1970). The most effective international efforts of the period were undoubtedly

those of the Rockefeller Foundation, which led a successful campaign to eradicate yellow fever from Latin American cities in 1916–23, repelled the invasion of *Anopheles gambiae* into Brazil in 1938, financed medical schools around the world, and was "probably the largest single factor in improving the public health education of the world up to the creation of the WHO" (Goodman 1971, pp. 381, 266, 377–82). Its antimalarial activities began in 1915 and included demonstrations of the superior cost effectiveness of vector control compared with treatment and the feasibility of complete eradication. The antimalarial activities were considered by Russell, one of the world's leading malariologists, to be of fundamental importance in ultimate control of the disease. "In instance after instance the foundation provided the catalyst, or the inexpensive mainspring, or the seed money that resulted in control of the disease" (Russell 1968, p. 644).

The total amount of money appropriated by the Rockefeller Foundation from 1914 to 1954 for antimalarial activities, exclusive of salaries and overhead was only $5 million (Russell 1968, p. 644). This is a vivid illustration that contributions to mortality change are inaccurately reflected on financial ledgers. International aid for health purposes is a small part of total health expenditures in LDCs and probably always has been. But its cost effectiveness has certainly far surpassed the average for internally financed appropriations, which are too often focused on expensive curative services in urban areas.[14]

Only crude indications are available of the relative magnitudes of internal and external sources of health expenditures in LDCs. In 1970, government health expenditures were estimated for LDCs containing 1.89 billion people. Total government expenditure on health in these areas came to $7.67 billion, or about $4 per capita.[15] Private health expenditures in LDCs are probably slightly larger than public expenditures, judging from comparisons that can be made in seven countries.[16] Addition of private and public expenditure in countries not represented would bring the total annual expenditure perhaps to the range of $20–30 billion.

In contrast, the largest single source of international assistance for health, the World Health Organization, dispersed only $115 million in 1972, a figure that includes family planning activities and some dispersals to MDCs (World Bank 1975, pp. 68–69). The annual budget of the World Health Organization in 1970 was less than that of Massachusetts General Hospital! (Goodman 1971, p. 223). Of its regular budget, the United States contributed 31% and the USSR 13%, with no other country making a contribution larger than 7% (Goodman 1971, p. 220). The second largest source of international assistance in 1972, USAID, contributed $42 million. All together, the ten largest multilateral or bilateral sources of health aid contributed $300.7 million

in international assistance for health programs in 1972 (World Bank 1975, p. 68), probably between 1% and 2% of total health expenditures in LDCs. To this should be added a portion of the $79 million in loans and credits made by the World Bank for water supply and sewerage construction in that year (World Bank 1975, p. 48).

It seems very likely, then, that total external health aid received by LDCs is less than 3% of their total health expenditures. The figure may have been somewhat higher earlier in the postwar period. The cumulative United States contribution to antimalarial activities through national research and international assistance has been estimated to be about one-half billion dollars (Russell 1968), but the annual contribution has declined drastically (Weller 1974; World Health Organization 1975*a*). But even the cumulative total is a paltry figure compared with annual expenditures in LDCs themselves. MDC contributions to mortality declines in LDCs have not been primarily financial; according to the estimates of the preceding section, the financial contributions are associated with an increase in e^0_0 of about one year in the seventeen LDCs between 1940 and 1970. Instead, they seem to have consisted of the development of low-cost health measures exploitable on a massive scale, demonstration of their effectiveness in relatively small areas, training and provision of personnel, and occasionally the initiation of large-scale programs whose major cost was often absorbed by the recipient country.[17] When action appeared to be remarkably cost effective and timely, such as a campaign to eradicate smallpox from West and Central Africa, the entire burden of effort was occasionally absorbed by an MDC (Foege et al. 1975).

5.2 Consequences of Mortality Reductions

In this section we can do no more than begin to sketch in the major influences of these mortality declines on populations of the less developed world, since these declines affect virtually every aspect of individual and collective life in a manner that undoubtedly varies with a host of initial conditions present in the population. It is probably wise to begin with the most concrete and least variable effects, the demographic ones.

Other things remaining the same, mortality declines increase the rate of population growth. The initial effect obviously is to increase the crude rate of natural increase by the absolute amount of the decline in the crude death rate. To a close approximation, the long-run effect of a permanent decline is to increase the rate of natural increase by the average (unweighted) decline in age-specific death rates between age zero and the mean age of childbearing (Preston 1974). This effect is almost fully realized within two generations. In neither case is the growth response strongly conditioned by the prevailing level of fertility. Not a

shred of doubt remains that the vast majority of the acceleration in world population growth during the twentieth century is attributable to mortality decline rather than to a rise in fertility.

It is important to recognize that changes in rates of population growth typically have very different effects on demographic, economic, and social processes depending on their source. Coale and Hoover (1958) in their classic study were careful to point out that they were studying the economic implications of variation in fertility, but the study has often been misinterpreted as suggesting the deleterious effects of rapid population growth per se. Application of a modified Coale-Hoover model by Barlow (1967, 1968) demonstrated much more ambiguous economic effects when the source of growth acceleration was mortality decline. Loose discussions of relations between population and economic growth are usually aimed implicitly at the fertility component, even though it is mortality variation that has been the root of trends in population growth.

The fundamental reason why effects differ is a difference in the ages of persons affected. Changes in fertility initially affect only the number of zero-year-olds, and permanent changes permanently affect the age distribution of the population. Mortality changes typically affect all ages, and age distributional changes are relatively minor (Coale 1956; Stolnitz 1956). Such as they are, the short-run age distributional changes induced by mortality decline typically increase the proportion of the population at ages below 5 or 10 and above 40 and decrease the proportion at other ages. The pertinent index is the age-specific death rate, μ_x. When this declines by more than the population-weighted average, the proportion of the population in the immediately succeeding ages will rise. Since mortality declines have tended to be largest in absolute (but not proportional) terms at the extremes of life, the dependency burden initially rises. The long-run effect of a permanent decline in mortality is typically to increase the proportion of the population at ages below 20 and above 75. The pertinent age-specific index here is the cumulative change (unweighted) in age-specific death rates since age zero relative to an appropriately defined average (Preston 1974). The long-run effect on the dependency burden is also positive. With the gross reproduction rate fixed at 2.5, a rise in female life expectancy from 30 to 50 to 70 years in stable populations characterized by "West" mortality patterns increases the ratio of those outside of labor force age (15–64) to those within from 0.635 to 0.764 to 0.847 (Coale and Demeny 1966, pp. 82, 98, 114). All of this increase is sustained in the ages below 15. These changes are not trivial, but they are rather small relative to those induced by movements of fertility within its observed range.

It follows from this discussion and the formal analysis that supports it that if all ages experience an identical decline in death rates (usually

termed a "neutral" decline), the age composition of the population will be unaffected in both the short run and the long run. The probability of survival from age x *to* age $x+n$ is equal to $\exp\{-\int_x^{x+n} \mu(t)dt\}$, where $\mu(t)$ is the death rate at exact age t. A decline in mortality by amount k at all ages will raise all n-year survival probabilities by the factor, $\exp\{kn\}$. Since the population at each age grows by the same factor (including infants via the greater survivorship of prospective parents), the proportionate age distribution is unaffected. Barring behavioral changes, a decline that is neutrally distributed among population subgroups, however defined (e.g., occupational or educational groups), will not affect population composition. The point is worth emphasis: it is *differential* mortality change that affects population composition. A change that is equally shared affects only size and growth. To the degree that typical mortality changes have been differentially distributed, the first-order changes induced in population composition have been economically unfavorable. Not only have the very young and the very old profited disproportionately, but so have women and unskilled or semiskilled workers.[18] Unlike programs of human resource development, which usually aim directly at an upgrading of population composition in ways that relate to production, programs of mortality decline have typically increased population size and reduced, at least initially, the desirability of its configuration.

Although mortality and fertility variation have very different effects on population composition, the mechanism by which they influence population size is the same in the long run: changes in the annual number of births. The principal long-run effect of mortality decline on population size arises not from the greater survivorship of persons who would have been born in any event, but from the larger number of births that are produced. To see this clearly, suppose that a neutral mortality change occurs to an initially stable population such that all ages experience a permanent reduction in death rates of .02. If age-specific fertility rates remain unchanged, the rate of population growth will increase by .02 and the rate of increase in the annual number of births will also rise by .02. Consider the number of 20-year-olds in the population 60 years after the mortality decline. The original number born into this cohort will be larger as a result of the mortality decline by $[e^{.02(40)}-1] = 123\%$, whereas their improved survivorship after birth will have increased their numbers by $[e^{.02(20)}-1] = 49\%$, a growth factor less than half as large. More than half the members of the cohort would not have been born had mortality not declined. This fraction continues to grow over time, but the improved survivorship factor does not. Stated more vividly, any LDC child "saved" from death today adds only one to the population size for a time. But the progeny of that child will ultimately

be infinite in numbers if current rates of mortality and fertility are maintained by all generations. The prevailing practice in health economics of ignoring the offspring of the population "saved" seriously misrepresents the effect of health programs on populations (see reviews in Weisbrod 1975 and Klarman 1967).

5.2.1 Economic and Behavioral Responses

In discussing aggregate economic and behavioral responses to mortality decline, it is useful to recognize that all of the responses must make themselves felt through one of four indexes. This follows directly from a formal identity:

$$CDR = CBR + CRNM - R_p + R_{pc},$$

where CDR, CBR, $CRNM$ = crude rates of death, birth, and net migration

R_p, R_{pc} = proportionate rate of growth of total production and of production per capita.

When the crude death rate declines, one of the terms on the right side must change to keep the identity in balance. The first three terms on the right side—birthrates, migration rates, and the economic growth rate—primarily reflect behavioral adjustments to mortality change. The fourth —the growth rate of output per capita—is basically a default option, inevitably activated if none of the other three terms change. If none of the four terms on the right can change, or change for very long, the decline in CDR cannot be sustained. This is the basic Malthusian model, in which the "passion between the sexes" placed a floor on the crude birthrate, a subsistence level of production bounded R_{pc} from below, migration was defined as impossible, and slow technical change and rapidly diminishing returns to labor constrained R_p from above.

As an identity, any term in it could be isolated on one side of the equation and the others forced to "respond" to its changes. The justification for isolating the CDR is provided in the first part of the paper: a substantial fraction of changes in CDR have been induced by factors independent of any term on the right side, and it is reasonable to view them as being forced to respond to it. To the extent that declines in CDR have been produced by increases in rates of growth of production per head, the equation as presented is misleading.[19]

In most of the remainder of this section we will consider the various ways populations appear to have responded to mortality declines, taking each of the possibilities in turn. The review attempts to be positive and historical rather than normative.

Declines in Crude Birthrates

There are a multitude of ways that changes in mortality can induce changes in fertility. Three of the effects are quasi-biological. Declines in

mortality change the age structure in such a way as to reduce the proportion of the population in the childbearing years and to reduce crude birthrates if age-specific fertility rates remain constant. Using the earlier example, gains in female life expectancy from 30 to 50 to 70 years, with age-specific fertility held constant at a level that produces a gross reproduction rate of 2.5, changes the crude birthrate from 38.78/1,000 to 37.12 to 35.99. The decline in the crude death rate over this range is from 33.34 to 5.78, so that the decline in the birthrate compensates for 10.1% of the decline in crude death rates through this age-structural route (Coale and Demeny 1966).

A second biological mechanism operates through breast-feeding. Breast-feeding inhibits ovulation, particularly in poorly nourished populations. Survival of the previous birth, by extending lactation, tends to delay the arrival of the next birth. Estimates of the average amount of net delay range as high as 12–13 months in Senegal, Bangladesh, and certain preindustrial European populations, although an estimate of about 7 months is probably more representative of poor agrarian populations (see the review in Preston 1975*b*). Since average interbirth intervals in such populations average about 30–35 months, the compensating variation in fertility to a change of infant mortality rates can be as high as 35% but is more likely to be in the area of 20%. That is, if all a woman's children die in infancy, her average interbirth intervals will be shorter by 20% and she will have approximately 20% more births over her reproductive life than if all had survived. In urban Latin American settings, where breast-feeding is usually short if it occurs at all, the compensating variation from this source is negligible (Rutstein and Medica 1975).

A third quasi-biological influence operates in the opposite direction but is probably fairly weak. Mortality declines make it more likely that marriages will survive through the end of the partners' reproductive periods. In areas such as India where sanctions against widow remarriage are strong, reductions in the incidence of widowhood probably exert an upward pressure on fertility. Arriaga (1967) has argued that this mechanism is responsible for substantial postwar increases in child-woman ratios in certain Latin American populations. However, his argument neglects the age-structural changes that are directly produced by mortality declines. The maximum effect of mortality changes on a woman's completed fertility can be estimated by assuming that no remarriage is possible and that childbearing occurs at a constant rate throughout her reproductive life up to the death of her husband or for 25 years, whichever comes first. Then her completed fertility is simply proportional to the expected number of years lived by the husband in the first 25 years of marriage. Assuming that males are age 20 at marriage, this expectation goes from 20.97 years in a population with a male life expectancy at birth of 30.08 to 24.49 when life expectancy is 68.56

(Coale and Demeny 1966, pp. 7, 23). Fertility would increase by 16.8%, or by perhaps 6 points, while the death rate would decline by perhaps 25 points. Complete prohibition of widow remarriage could thus boost the growth acceleration induced by mortality decline by some 25%, making it roughly equivalent to but opposite in sign from the lactational effect. However, it does not appear that taboos on widow remarriage are sufficiently widespread outside of India to have anywhere near this effect.

In addition to the three quasi-biological links, there are many possible behavioral ones. Since these are the subject of another conference paper (chap. 3), they will not be reviewed here (see also O'Hara 1975). Suffice it to say that the magnitude of one relationship has now been investigated rather carefully in a variety of populations in Asia and Latin America, as well as in preindustrial Europe. It has repeatedly been shown that, among women of a particular parity, those who had experienced one additional child death subsequently bore, on average, far fewer than one additional birth. Furthermore, some of the additional childbearing that did occur could be traced indirectly to the biological link identified above. The studies have attempted to control other characteristics of women believed to influence fertility (see studies by Chowdhury, Khan, and Chen; Heer and Wu; Rutstein and Medica; and Knodel in Committee for . . . Demography 1975). The largest "replacement" effect was 0.28 identified in Taiwan by Heer and Wu. It may be that the dead children were replaced in advance, but the simple demography of death makes this an inefficient reproductive strategy. The large majority of children who die before adulthood do so in the first two or three years of life, and their death can be observed and reacted to by parents during their own reproductive period.

The apparent failure of parents in many LDCs to behave as though they were pursuing a single reproductive target framed in terms of surviving children should not be surprising. It is clear that many of the social norms and sanctions that regulate reproductive behavior in LDCs refer to age at entry into union, frequency of intercourse, postnatal abstinence, number of partners, lactation, remarriage, and so on, rather than specifically to the number of children born or surviving (Polgar 1972). It is not necessary to reject the view that reproducers are goal-directed, but only to recognize that social norms and expectations have established other goals than "the" number of children. These norms and expectations are not responsive to an individual's experience with child death, although they may be responsive to aggregate mortality rates. Such conditioning is in fact the basis of functionalist theories of fertility. Davis (1955) has repeatedly argued that high-mortality populations must adopt a set of institutions and customs producing high fertility or else face extinction. The major adaptive institution he points to is the

extended family, which encourages early entry into union by arranging marriages and stimulates fertility by removing many of the child-rearing costs from the parents. The expected positive effect of family extension on fertility has not been observed in most empirical studies at the household level (see the review in Burch and Gendell 1970). More to the point, it is not clear how the group-selection processes that are supposed to have created such institutions in the first place would operate to change them when mortality conditions relax.

There is one aggregate-level linkage between death rates and birthrates that deserves mention because of its apparent importance in preindustrial Europe. In a spatially limited system where land is the basis of wealth and accession to land the prerequisite for marriage, the rate of marriage and hence childbearing will depend upon the rate at which land becomes available, hence on mortality. Exogenous declines in mortality will, more or less automatically, reduce fertility by slowing the turnover of land and delaying marriage. Such a system was apparently an important feature of demographic-economic relations in Western Europe from 1600 to 1800 and may account for the generally late age at marriage in these populations (Habakkuk 1971; Wrigley 1969; see Eversley 1957 for a vivid numerical description of the expected responses to an epidemic). More important, economic historians have suggested that this mechanism, the "European marriage pattern," was a fundamental basis of the industrial revolution since it facilitated capital accumulation by severing the link between the level of mortality and the rate of growth of income per capita (Wrigley 1969; Habakkuk 1971). How important this mechanism is in contemporary less developed countries is unclear. Increased rates of population growth have been accompanied by declining proportions married throughout much of Asia since 1960 or so (Smith 1976). However, many other modernizing influences have also been at work. In Asia, at least, the prevalence of an extended family system in rural areas reduces the dependence of marriage on individual acquisition of land and presumably attenuates the link between population pressure and individuals' marital behavior. In urban areas, however, a "suitable" job may come to play an analogous, though no doubt less decisive, role.

That the sum of responses of fertility to mortality declines in LDCs has been quantitatively weak is shown clearly in the history of population growth rates. Durand (1967, p. 7) puts the annual rate of population growth for LDCs at 0.3% for 1850–1900 and 2.1% for 1950–65. He further suggests that, with few exceptions, birthrates have not declined. The United Nations Population Division "estimates and conjectures" that CDRs in LDCs were 38/1,000 in 1850–1900 and 17/1,000 in 1960–70, whereas CBRs were 40/1,000 and 41/1,000 in the two periods (United Nations 1971, p. 7). These figures suggest no compen-

sating variation in fertility whatever, although mortality declines may have reduced the increase in fertility that would otherwise have occurred. It is true that several of the postulated relations should operate with a lag, and widespread (but small) declines in fertility since 1970 in LDCs might be partially attributed to prior mortality decline. But the evidence is that changes in birthrates have not been a major mechanism of adjustment to date.

Declines in Crude Rates of Net Migration

An increase in out-migration for the world as a whole is clearly impossible, and it has been scarcely more of an option for LDCs as a bloc. Perceived cultural, economic, and political difficulties attendant upon migration from LDCs to MDCs have resulted in MDC immigration quotas that are fixed at a point where they represent a tiny fraction of annual natural increase in LDCs. International migration among LDCs faces similar obstacles (see the discussion in Myrdal 1968, pp. 1459–62). In this matter the present LDC situation is again markedly at variance with that of European populations in the eighteenth and nineteenth centuries, when a substantial amount of population increase was drained off via overseas or transcontinental migration. Friedlander (1969) stresses the importance of this "safety valve" for delaying fertility reductions in England and Sweden. My calculations indicate that 15–25% of persons born in Sweden in the middle decades of the nineteenth century died outside its borders.

When subnational territories are considered, the migration response probably becomes more consequential, because the export of population growth from one area to another faces fewer legal, cultural, and institutional impediments. In Indonesia, the Philippines, and Ceylon, government programs have attempted to redistribute population from dense, rapidly growing areas to sparsely populated ones, although relative to natural increase the movements have been small (Myrdal 1968, pp. 2139–49). How important mortality declines in rural areas have been for rural-urban migration in LDCs has simply not been identified, to my knowledge. The region with apparently the sharpest mortality decline since 1900, Latin America, has also had the most rapid rural to urban migration (United Nations 1971). But both regional peculiarities may have been caused by its more rapid economic growth. Unlike the European situation, net rural-urban migration is not required to maintain constant proportions in the two sectors. Rural birthrates are higher, but so in general are rural death rates. Rates of natural increase are not widely disparate (Davis 1973). It has often been suggested that, in Asia at least, a large fraction of rural natural increase (which accounts for the large bulk of the annual volume) simply cannot be exported to urban areas because the cities cannot create enough new jobs. Even if these

predictions prove pessimistic, it remains that, for national aggregates, changes in net migration rates have not been and will not be an important response to mortality decline.

Increases in Growth Rates of Total Production

The effect of population growth on economic growth is obviously a topic whose scope and complexity are too vast to be adequately reviewed here. While the effects of changes in death rates on output are difficult to partial out (see Denison 1962 for one attempt), economic history and economic theory are quite consistent with the view that R_p has been the principal respondent to changes in CDR.

A decline in CDR generally increases the growth rate of the labor force in roughly equal measure. As noted above, the labor force growth rate is typically incremented in the short run by a slightly smaller amount than the population growth rate. But even after accounting for this tendency, if we accept standard estimates of the elasticity of production with respect to changes in the quantity of labor input on the order of 0.5 to 0.8, more than half of the "response" can be accounted for without any change in other inputs or in technology.

There are reasons to believe that other inputs have typically changed in a reinforcing fashion. In some instances (South America, Southern Africa, and much of Asia) the growing population has had access to unutilized land not markedly inferior to that already under production (Myrdal 1968). According to D. Gale Johnson (1974, p. 89), between 1935–39 and 1960 approximately 75% of the increased grain output in developing countries resulted from expansion of the planted area. In some cases the mortality reduction campaigns themselves have liberated large areas of previously inhospitable territory. Taylor and Hall (1967) cite examples of such effects from antimalarial programs in Nepal, Sri Lanka, Sardinia, and Mexico (see also Sorkin 1975). Schultz (1964, pp. 63–70) shows that the response is also present when mortality changes in the opposite direction. An estimated 8% fall in the Indian labor force resulting from the 1918–19 influenza epidemic was accompanied by a reduction of 3.8% in acreage sown.

Expansion of land use obviously cannot continue indefinitely, and to the (apparently minor) degree that fixed factors are important in production, the particular effect of accelerated population growth on per capita production will be negative. For most purposes it is more important to know the effects of mortality decline on the capital supply. With one possibly important exception, the effects should not be radically different from those of increased fertility. The age distributional effects are similar in nature, though muted in the case of mortality change. When mortality declines, the proportion of the population in a stage of dissaving increases—permanently. Households face an increased depen-

dency burden and governments a greater press of immediate consumption demands. Business optimism regarding future demand for their products rises with the growth rate of potential consumers. Business profits and internally financed investments may increase as the labor supply curve shifts outward, unless the shift also reduces consumers' purchasing power. These effects have been reviewed elsewhere in conjunction with fertility effects, and it is not profitable to reconsider them here.

The one possibly important difference is that members of a lower-mortality population can look forward to longer lives in which to reap the benefits of personal investment. There is no such effect when fertility is the source of growth acceleration. The present value of investments with a long gestation period, such as extended schooling, retirement equities, or children, necessarily rises when mortality rates fall. Mushkin (1964), Schultz (1975), and others have suggested that such effects may represent an important economic benefit of reduced mortality.

It seems indisputable that such effects operate, and are probably reinforced by the increased proportion of the population who are in the investment stage. Nevertheless, it appears that the effects are relatively small and can be easily overwhelmed by minor variations in discount rates. To illustrate, define the present value of an investment in the standard manner prior to a change in mortality:

$$P.V. = \int_0^\infty p(a) \left[\frac{B(a) - C(a)}{e^{ra}} \right] da,$$

where $p(a)$ = the probability that the investor will survive a years from the time of investment according to the mortality schedule in effect prior to the mortality decline

$B(a), C(a)$ = benefits and costs of the investment realized or incurred in year a

r = subjective rate of discount, continuously applied.

Now superimpose a neutral mortality decline of 0.01 per year, equivalent to a reduction in the CDR of 10/1,000, that is, a large decline. This is the average reduction in age-specific death rates between ages 15 and 50 when male life expectancy at birth increases from 30.1 to 51.8 years (Coale and Demeny 1966, pp. 7, 16). The new present value is

$$P.V.' = \int_0^\infty p(a)\, e^{.01a} \left[\frac{B(a) - C(a)}{e^{ra}} \right] da$$

$$= \int_0^\infty p(a) \left[\frac{B(a) - C(a)}{e^{(r-.01)a}} \right] da.$$

That is, such a major change in mortality conditions has the same effect on present value as reducing the discount rate by only 0.01 and retain-

ing the initial mortality conditions. In view of the wide variation that seems to prevail in discount rates, as partially reflected in the common analytic practice of applying several that differ by 0.05 or even more, it is readily seen that mortality prospects are a relatively minor influence on present value.

As a more concrete illustration, we will compute the internal rate of return on investment in schooling and show how it is affected by empirically observed variation in mortality. We have chosen Mexico for the illustration because Carnoy (1967) provides all of the necessary information except life tables.[20] We will compute the internal rate of return for a 15-year-old male who has completed grade 8 from his subsequent completion of grades 9, 10, and 11. Three mortality schedules are used: the male life table of Mexico, 1921, having a life expectancy at birth of 33.66 years (Arriaga 1968); the male life table of Mexico, 1966, having a life expectancy at birth of 59.49 years (Keyfitz and Flieger 1971); and immortality. Results are shown in table 5.6.

It is clear from this table that the increment to the private or social rate of return that results from replacing an e^{o}_{0} of 33.7 by immortality is only 1.5 points. This is approximately the difference it makes to use continuous rather than once-a-year compoundings. The reason for the weak effect is simply that mortality rates in young and middle adulthood are not high enough even in very high mortality populations to substantially alter expected payoffs. The large variability in rates experienced in childhood do not figure in the calculation, and the large variability at older ages is heavily discounted.

The effect of mortality variation would be somewhat stronger if individuals were predominantly risk-aversive and made decisions on the basis of the entire distribution of expected outcomes rather than simply on the basis of the mean. Higher mortality adds greater variability to the distribution of expected outcomes as well as reducing the mean. It increases the chance that zero or negative returns will accrue to investment, and this increase is faster than is the reduction in mean. Nevertheless, for the bulk of investors—those in early and middle adulthood

Table 5.6 **Internal Rate of Return from Completing Grades 9, 10, and 11 in Mexico, 1963, under Varying Assumptions about Mortality**

Assumption	Private Rate of Return	Social Rate of Return
Mexican life table of 1921	12.8	9.9
Mexican life table of 1966	13.9	10.9
Immortality	14.3	11.4

Sources: Carnoy (1967); Keyfitz and Flieger (1971); Arriaga (1968).

—it does not appear that even very radical mortality change could exert much influence on perceived investment profitability.

On balance, it does not appear that the effects of population growth acceleration on capital formation when mortality is the source should be markedly different *in nature* from the effects when fertility is the source. The Coale-Hoover model postulates a less-than-proportionate increase in capital supply when the population grows faster via higher fertility, and this feature is retained when the effects of mortality change are simulated (Barlow 1967, 1968). Largely for this reason, Barlow concludes that the antimalarial campaign in Ceylon ultimately had negative effects on per capita income growth, even after the improved health of the labor force is accounted for. Confidence in this conclusion depends on one's confidence in the savings-investment assumptions of the Coale-Hoover model.

It is also possible that mortality reductions foster economies of scale in production and an intensification of individual work effort. Again, the subject is very complex, and in most respects the analysis of mortality variation need not differ from that of fertility variation. A postulated difference is that mortality control programs have an important demonstration effect (Mushkin 1964; Fein 1964; Malenbaum 1970; and others). That is, they demonstrate that individuals can control their own destiny through the rational application of science and technology. They attest to the power of man in contrast to that of the supernatural and hence spark work effort and a stronger motivation toward self-fulfillment. Malenbaum (1970) argues that this is the basis of observed associations between mortality rates and labor productivity, but there are surely other mechanisms that offer more plausible interpretations. The contention has not been put to a rigorous test. It seems inconsistent with observations that poor illiterate farmers in LDCs have always responded quickly to new and profitable opportunities and technologies (Johnson 1974). A sense of personal control over the environment is one of the strongest components of "modern" attitudes (Sack 1974, p. 90). But the importance of mortality declines in the development of this attitude and the influence of the attitude on output remain to be demonstrated, important as the issue may be.

Changes in Growth Rates of Production per Capita

With constant technology and no changes in nonlabor factors of production, it is reasonable to expect that a CDR decline of 10/1,000 would raise the growth rate of total production only by 0.6–0.7% or so. If no birthrate or migration adjustments were forthcoming, the rate of growth of per capita income would decline by 0.3–0.4%. This is not a large amount relative to prevailing growth rates, and it is not surprising that

the mortality declines have left no unmistakable imprint on per capita national economic growth rates.

Nevertheless, there are circumstances where the mortality decline has apparently had a decisive negative effect on economic well-being. Perhaps the most vivid account is offered by William Allan (1965, especially chap. 21). Allan outlines a cycle of land degeneration in East Africa that was, he argues, initiated by a mortality reduction. Population pressure at the kin-group level led to land subdivision and fragmentation, since landowners were expected to share their holdings with needy kin. Subdivision led in turn to a shortening of the fallow period and to soil depletion and erosion. Declining yields led to accelerated shortening of the fallow and to ultimate soil exhaustion. "Perhaps the greatest 'sin' of the suzerain powers was the saving of life, the lives of millions of men who under the old conditions would have died in early childhood, or in later life, of famine, disease, and violence" (Allan 1965, p. 338). Ultimately the response was out-migration, but not before a stage of economic misery was encountered. The initial stages of intensified land use are those described by Boserup (1965), but the outcome is very different. Instead of self-sustaining growth supported by a newly developed work ethic, the result was simply impoverishment. Obviously, some soils can support extreme intensification and multiple cropping and others cannot. Population pressure by itself is clearly not sufficient for sustained technological change, nor does it appear to be necessary once the possibilities of trade are opened up.

It is clear that general statements cannot be made about how populations have reacted or will react to exogenous mortality declines. The reaction will depend on a wide variety of initial conditions. In agrarian populations it appears that the most important conditioning factors are type of soil, land tenancy and kinship-marriage systems, density, possibilities for out-migration, savings and investment relationships, and the saliency of surviving-children goals. A great deal of work in recent economic and demographic history remains to be done before the quantitative details of the outline sketched in these sections can be confidently filled in.

Appendix

Table 5.A.1 Estimates of National Indexes about 1940

Country	Life Expectancy at Birth	Year of Estimate	Source	Daily Calories per Capita Available at Retail Level	Year of Estimate	Source: (6) Unless Noted
Australia	66.09	(40)	(2)	3,128	(40)	
Belgium	59.08	(38–40)	(3)	2,885	(40)	
Canada	64.62	(40–42)	(1)	3,109	(40)	
Czechoslovakia	56.79	(37)	(1)	2,761	(40)	
Chile	38.10	(40)	(4)	2,481	(40)	
Colombia	36.04	(38)	(4)	1,860	(38)	(7)
Denmark	66.31	(40)	(2)	3,249	(40)	
Egypt	38.60	(36–38)	(1)	2,199	(40)	
Finland	57.39[a]	(40)	(1)	2,950	(40)	
Greece	54.37	(40)	(1)	2,523	(40)	
Guatemala	30.40	(40)	(4)	—	—	
Honduras	37.50	(40)	(4)	2,079	(40)	
Hungary	56.57	(41)	(1)	2,815	(40)	
India	32.27	(41)	(5)	2,021	(40)	
Ireland	60.02	(40)	(1)	3,184	(40)	
Japan	49.12	(39–41)	(3)	2,268	(40)	
Korea	48.90	(38)	(1)	1,904	(40)	
Luxembourg	60.07	(38–42)	(3)	2,820	(34–38)	(8)
Mexico	38.80	(40)	(4)	1,909	(40)	
Netherlands	65.43	(40)	(2)	2,958	(40)	
New Zealand	67.00	(36)	(2)	3,281	(40)	
Nicaragua	34.50	(40)	(4)	—	—	
Panama	42.40	(40)	(4)	—	—	
Peru	36.50	(40)	(4)	2,090	(40)	
Philippines	46.26	(38)	(1)	2,021	(40)	
Portugal	51.06	(40)	(2)	2,461	(40)	
Puerto Rico	46.09	(39–41)	(1)	2,219	(40)	
Spain	50.18	(40)	(1)	2,788	(40)	
Sweden	66.64	(40)	(1)	3,052	(40)	
Switzerland	64.88	(40)	(2)	3,049	(40)	
Taiwan	47.80	(41)	(1)	2,153	(40)	
Thailand	40.02	(37–38)	(1)	2,173	(40)	
Turkey	33.91	(35–45)	(15)	2,619	(40)	
United Kingdom	61.64	(40)	(2)	3,005	(40)	
United States	63.74	(39–41)	(1)	3,249	(40)	
Venezuela	39.91	(41)	(1)	—	—	

National Income per Capita in 1970 U.S. Dollars	Year of Estimate	Source: (9) Unless Noted	Percentage Illiterate	Age Range	Year of Estimate	Source: (10) Unless Noted	Population in Thousands (1940)(13)
1,128	(38–40)		2.5	—	(40)	(14)	7,039
715	(38–39)		4.5	—	(40)	(14)	8,301
1,041	(38–40)		3.16	10+	(40)		11,682
438	(38)		3.6	—	(37)	(14)	14,429
371	(38–40)		28.2	10+	(40)		5,063
190	(38–40)		44.10	10+	(38)		8,702
971	(38–40)		1.5	—	(40)	(14)	3,832
167	(39)		84.9	10+	(37)		16,008
419	(39)		8.8	—	(40)	(14)	3,698
187	(38)		34.4	—	(40)	(14)	7,319
78	(40)		65.4	7+	(40)		2,201
109	(41–42)		66.35	10+	(45)		1,146
318	(38–40)		6.0	10+	(41)		9,344
67	(38–39)		86.5	10+	(41)	(10,11)[b]	316,004
665	(40)		1.5	—	(40)	(14)	2,993
260	(39)		—	—	—		71,400
—	—		68.6	10+	(30)		21,817
795	(39)		4.4	—	(40)	(14)	292
138	(40)		51.5	10+	(40)		19,815
889	(38)		1.5	—	(40)	(14)	8,879
1,055	(38–40)		1.5	—	(40)	(14)	1,573
105	(40)		63.0	7+	(40)	(12)	825
374	(40)		35.25	10+	(40)		620
89	(40)	(16)	56.35	10+	(40)		7,033
113	(38)		37.75	10+	(48)		15,814
—	—		48.7	10+	(40)		7,696
413	(38–40)		31.5	10+	(40)		1,880
361	(40)	(16)	23.2	10+	(40)		25,757
1,091	(40)		0.1	10+	(30)		6,356
1,246	(40)		1.5	—	(40)	(14)	4,234
—	—		78.7	5+	(40)		6,163
128	(39)	(11)	46.25	10+	(47)		14,755
212	(39)		79.1	10+	(35)		17,620
1,334	(38–40)		1.5	—	(40)	(14)	41,862
1,549	(40)		4.2	10+	(40)	(13)	132,594
291	(40)		56.50	10+	(41)		3,803

Table 5.A.1 (continued)

Sources:
1. United Nations, *Statistical Yearbook* (1967).
2. Preston, Keyfitz, and Schoen (1972).
3. Keyfitz and Fleiger (1968).
4. Arriaga (1968).
5. Estimated by Davis (1951, pp. 62–63).
6. Estimates prepared by the Food and Agriculture Organization of the United Nations, cited in "Food, Income, and Mortality," *Population Index* 13, no. 2 (April 1947): 96–103.
7. United Nations, *Statistical Yearbook* (1951).
8. United Nations, Food and Agriculture Organization, *Production Yearbook* (1958).
9. Estimates prepared by the Technical Group, U.S. Bureau of the Budget; cited in "Food, Income, and Mortality," *Population Index* 13, no. 2 (April 1947): 96–103. All figures have been converted to 1970 U.S. dollars by application of the consumer price index from U.S. Bureau of the Census, *Statistical Abstract of the United States*, various issues.
10. United Nations, *Statistical Yearbook* (1949–50).
11. United Nations, *Statistical Yearbook* (1955).
12. United Nations Educational, Scientific and Cultural Organization, *Basic facts and figures: Illiteracy, libraries, museums, books, newspapers, newsprint, film and radios* (Paris, 1952).
13. United Nations, *Demographic Yearbook* (1960).
14. Banks (1971).
15. Calculated by author from estimates of e_5^0 presented in Shorter (1968). The Coale-Demeny "South" model mortality pattern was assumed to apply.
16. United Nations, *National Income Statistics of Various Countries, 1938–1948* (Lake Success, N.Y., 1950).

[a]Average 1936–40 and 1941–50.
[b]Interpolated from data for 1931 and 1951.

Table 5.A.2 **National Indexes about 1970**

	Life Expectancy at Birth 1970–75 (1)	Percentage Illiterate of the Adult Population (2)	Year of Estimate (2)	1970 National Income per Capita (1970 U.S. $) (3)	1970 Daily Calories Available for Consumption, per Capita (4)	1970 Population (in 1,000s) (1)	Index of Income Inequality (5)	Year of Estimate	Coverage
Africa									
Algeria	53.2	52.5	1971	295	1,710	14,330	—		
Angola	38.5	87.5	1973*	280	1,910	5,670	—		
Botswana	43.5	87.0	1971	132	2,040	617	—		
Burundi	39.0	35.0	1974*	68	2,330	3,350	—		
Central African Rep.	41.0	82.0	1975*	122	2,170	1,612	—		
Chad	38.5	90.0	1975*	70	2,060	3,640	—		
Congo	43.5	65.0	1970*	281	2,160	1,191	—		
Dahomey	41.0	80.0	1975*	81	2,250	2,686	—		
Egypt	52.4	62.0	1975*	202	2,360	33,329	−6.5660	1965	Nat. Household
Ethiopia	38.0	93.0	1975*	72	2,150	24,855	—		
Gabon	41.0	88.0	1974*	468	2,210	500	−17.2442	1960	Nat. Population
Gambia	40.0	90.0	1971*	99	2,370	463	—		
Ghana	43.5	56.5	1971	236	2,200	8,628	—		
Guinea	41.0	92.5	1971*	79	2,040	3,291	—		
Ivory Coast	43.5	80.0	1973*	325	2,490	4,310	−9.8088	1970	Nat. Income Recip.
Kenya	50.0	75.0	1975*	130	2,350	11,247	−13.1644	1969	Nat. Income Recip.
Liberia	43.5	88.0	1970	181	2,040	1,523	—		
Libyan Arab Rep.	52.9	68.0	1974*	1,450	2,540	1,938	—		
Madagascar	43.5	60.0	1975*	123	2,350	6,932	−9.8964	1960	Nat. Population
Malawi	41.0	75.0	1976*	68	2,150	4,360	−7.0502	1969	Nat. Household
Mali	38.0	90.0	1972	50	2,170	5,047	—		

Table 5.A.2 (continued)

	Life Expectancy at Birth 1970–75 (1)	Percentage Illiterate of the Adult Population (2)	Year of Estimate (2)	1970 National Income per Capita (1970 U.S. $) (3)	1970 Daily Calories Available for Consumption, per Capita (4)	1970 Population (in 1,000s) (1)	Index of Income Inequality (5)	Year of Estimate	Coverage
Mauritius	65.5	20.0	1974*	223	2,370	824	—		
Mauritania	38.5	95.0	1972*	147	2,060	1,162	—		
Morocco	52.9	78.6	1971	225	2,400	15,126	—		
Mozambique	43.5	80.0	1972	228	2,190	8,234	—		
Niger	38.5	94.0	1973*	70	2,180	4,016	—		
Nigeria	41.0	74.0	1973	135	2,290	55,073	—		
Rhodesia	51.5	95.0	1972	257	2,550	5,308	−14.8362	1968	Nat. Income Recip.
Rwanda	41.0	90.0	1973*	57	2,160	3,679	—		
Senegal	40.0	95.0	1971*	201	2,300	3,925	−12.1394	1960	Nat. Population
Sierra Leone	43.5	90.0	1974*	150	2,240	2,644	—		
Somalia	41.0	95.0	1974*	85	1,770	2,789	—		
South Africa	51.5	56.0	1974*	680	2,730	21,500	−14.3543	1965	Nat. Population
Sudan	48.6	80.0	1973	109	2,130	15,695			
Togo	41.0	90.0	1976*	125	2,160	1,960			
Tunisia	54.1	60.0	1972	257	2,060	5,137	−9.7467	1961	Nat. Population
Uganda	50.0	70.0	1976*	127	2,230	9,806			
United Rep. of the Cameroon	41.0	35.0	1976*	183	2,230	5,836	—		
United Rep. of Tanzania	44.5	71.0	1967	94	1,700	13,273	−8.1535	1967	Nat. Household
Upper Volta	38.0	90.0	1972	62	1,940	5,384	—		
Zaire	43.5	65.0	1971	118	2,040	21,638	—		
Zambia	44.5	52.7	1969	345	2,040	4,295	—		

Table 5.A.2 (continued)

	Life Expectancy at Birth 1970–75 (1)	Percentage Illiterate of the Adult Population (2)	Year of Estimate (2)	1970 National Income per Capita (1970 U.S. $) (3)	1970 Daily Calories Available for Consumption, per Capita (4)	1970 Population (in 1,000s) (1)	Index of Income Inequality (5)	Year of Estimate	Coverage
Asia									
Afghanistan	40.3	92.5	1973	83	1,950	16,978	—		
Bangladesh	35.8	90.0	1973	111	1,860	67,692	−3.5534	1967	Nat. Household
Burma	50.0	30.0	1974*	73	2,230	27,748	—		
Cyprus	71.4	18.0	1973	688	2,460	633	−3.3164	1966	Urban Household
India	49.5	40.0	1971	93	2,060	543,132	−7.6103	1968	Nat. Household
Indonesia	47.5	40.0	1971	98	1,920	119,467	—		
Iran	51.0	65.5	1974	352	2,080	28,359	−8.2776	1968	Urban Household
Iraq	52.7	70.0	1974*	311	2,250	9,356	—		
Israel	71.0	12.8	1974*	1,654	2,970	2,958	−3.1183	1970	Urban Household
Japan	73.3	2.0	1975*	1.636	2,310	104,331	−3.3217	1963	Nat. Household
Jordan	53.2	62.5	1972*	260	2,470	2,280	—		
Khmer Rep.	45.4	15.0	1973	123	2,410	7,060	—		
Korea, Rep. of	60.6	8.5	1970	252	2,420	30,721	−2.2963	1971	Nat. Household
Laos	40.4	75.0	1970*	71	2,080	2,962	—		
Lebanon	63.2	14.0	1975*	521	2,380	2,469	—		
Malaysia	59.4	24.0	1970	295	2,400	10,466	−10.0406	1970	Nat. Household
Nepal	43.6	86.0	1971	80	2,050	11,232	—		
Pakistan	49.8	83.0	1973	164	2,280	60,449	—		
Philippines	58.4	16.5	1970	225	1,920	37,604	−8.6939	1971	Nat. Household
Saudi Arabia	45.3	75.0	1973*	495	1,920	7,740	—		
Singapore	69.5	24.5	1974	918	2,080	2,075	—		

Table 5.A.2 (continued)

	Life Expectancy at Birth 1970–75 (1)	Percentage Illiterate of the Adult Population (2)	Year of Estimate (2)	1970 National Income per Capita (1970 U.S. $) (3)	1970 Daily Calories Available for Consumption, per Capita (4)	1970 Population (in 1,000s) (1)	Index of Income Inequality (5)	Year of Estimate	Coverage
Asia (continued)									
Sri Lanka	67.8	22.0	1971	160	2,240	12,514	−4.4313	1970	Nat. Household
Syria	54.0	60.0	1970	258	2,530	6,247	—		
Taiwan	69.4(7)	5.0(6)	1965	295(7)	2,662(7)	14,334	−3.6354	1964	Nat. Household
Thailand	58.0	18.0	1970	167	2,330	35,745	−8.3579	1962	Nat. Household
Turkey	56.9	44.0	1970	348	2,770	35,232	−11.6389	1968	Nat. Household
Vietnam, Rep. of	40.5	23.0	1971	232	2,340	17,952	−3.8293	1964	Rural Household
Yemen	44.8	82.5	1975*	77	1,970	5,767	—		
Yemen, P.D.R.	44.8	90.0	1970*	96	2,020	1,436	—		
Latin America									
Argentina	68.2	8.0	1973*	1,065	3,150	23,748	−6.2544	1961	Nat. Household
Bolivia	46.8	58.5	1973	191	1,840	4,780	—		
Brazil	61.4	33.0	1970	376	2,600	95,204	−17.7304	1970	Nat. Household
Chile	62.6	12.0	1970	618	2,460	9,369	−8.4001	1968	Nat. Household
Colombia	60.9	21.5	1973	358	2,250	22,075	−10.6656	1970	Nat. Economic Active Population
Costa Rica	68.2	10.0	1973	522	2,470	1,737	−6.4897	1971	Nat. Household
Dominican Rep.	57.8	32.0	1970	334	2,060	4,343	—		
Ecuador	59.6	30.0	1970	255	2,040	6,031	−17.3614	1970	Nat. Economic Active Population
El Salvador	57.8	40.0	1971	283	1,890	3,516	−8.1625	1969	Nat. Population
Guatemala	52.9	62.0	1974*	343	2,120	5,298	−2.8074	1966	Rural Household

Table 5.A.2 (continued)

	Life Expectancy at Birth 1970–75 (1)	Percentage Illiterate of the Adult Population (2)	Year of Estimate (2)	1970 National Income per Capita (1970 U.S. $) (3)	1970 Daily Calories Available for Consumption, per Capita (4)	1970 Population (in 1,000s) (1)	Index of Income Inequality (5)	Year of Estimate	Coverage
Latin America (continued)									
Guyana	67.9	14.0	1974*	319	2,080	709	—		
Haiti	50.0	90.0	1974*	100	1,720	4,325	—		
Honduras	53.5	53.0	1974*	259	2,180	2,553	−14.9221	1968	Nat. Household
Jamaica	69.5	18.0	1970	600	2,300	1,882	—		
Mexico	63.2	24.0	1970	655	2,560	50,313	—		
Nicaragua	52.9	47.0	1971	423	2,380	1,970	—		
Panama	66.5	21.5	1973	646	2,520	1,458	−11.2564	1969	Nat. Economic Active Population
Paraguay	61.9	38.0	1973	230	2,800	2,301	—		
Peru	55.7	44.3	1970	293	2,310	13,248	−14.7827	1971	Nat. Economic Active Population
Trinidad and Tobago	69.5	30.4	1970	732	2,360	955	—		
Uruguay	69.8	9.0	1975*	799	2,860	2,955	−6.8128	1967	Nat. Household
Venezuela	64.7	35.2	1971	954	2,460	10,559	−10.5676	1962	Nat. Household
North America									
Canada	72.4	1.0	1975*	3,369	3,190	21,406	−4.0518	1965	Nat. Household
United States	71.3	1.0	1969	4,289	3,270	204,879	−6.6384	1966	Nat. Household
Puerto Rico	72.1	27.9	1970	1,744	2,450	2,743	—		

Table 5.A.2 (continued)

	Life Expectancy at Birth 1970–75 (1)	Percentage Illiterate of the Adult Population (2)	Year of Estimate (2)	1970 National Income per Capita (1970 U.S. $) (3)	1970 Daily Calories Available for Consumption, per Capita (4)	1970 Population (in 1,000s) (1)	Index of Income Inequality (5)	Year of Estimate	Coverage
Europe									
Austria	71.2	1.0	1974*	1,730	3,340	7,447			
Belgium	72.9	2.0	1975*	2,421	3,390	9,638			
Bulgaria	71.8	5.0	1975*	2,726	3,300	8,490	−1.5225	1962	Nat. Workers
Czechoslovakia	69.3	0.0	1974*	3,013	3,190	14,339	−1.1290	1964	Nat. Workers
Denmark	73.9	1.0	1974*	2,898	3,230	4,929	−4.9775	1966	Nat. Income Recip.
Finland	70.4	0.0	1975*	1,998	3,020	4,606	−9.8462	1962	Nat. Income Recip.
France	72.6	3.0	1975*	2,550	3,210	50,670	−11.4365	1962	Nat. Household
Germany, W.	70.6	1.0	1970*	2,752	3,230	60,700	−7.5125	1964	Nat. Income Recip.
Greece	71.8	15.6	1971	1,051	2,900	8,793	—		
Hungary	69.5	2.0	1975*	2,244	3,180	10,338	−2.1548	1969	Nat. Population
Ireland	71.8	1.0	1974*	1,244	3,420	2,954	—		
Italy	72.0	7.0	1975*	1,591	3,170	53,565	—		
Luxembourg	70.8	2.0	1975*	2,613	3,390	339	—		
Malta	70.8	12.0	1974*	721	2,680	326	—		
Netherlands	73.8	2.0	1973*	2,232	3,290	13,032	—		
Norway	74.5	0.0	1974*	2,458	2,920	3,877	—		
Poland	70.1	2.2	1970	5,766	3,270	32,473	−2.2046	1964	Nat. Workers
Portugal	68.0	45.0	1970*	684	2,890	8,628	—		
Spain	72.1	19.9	1970	884	2,620	33,779	−5.1072	1965	Nat. Household
Sweden	73.3	0.1	1975*	3,724	2,800	8,043	−6.1269	1963	Nat. Income Recip.
Switzerland	72.4	0.0	1973*	2,963	3,250	6,267	—		
United Kingdom	72.3	10.0	1975*	1,990	3,140	55.480	−4.0915	1968	Nat. Household

Table 5.A.2 (continued)

	Life Expectancy at Birth 1970–75 (1)	Percentage Illiterate of the Adult Population (2)	Year of Estimate (2)	1970 National Income per Capita (1970 U.S. $) (3)	1970 Daily Calories Available for Consumption, per Capita (4)	1970 Population (in 1,000s) (1)	Index of Income Inequality (5)	Year of Estimate	Coverage
Oceania									
Australia	72.4	1.5	1975*	2,633	3,050	12,552	−3.6895	1968	Nat. Household
New Zealand	72.0	2.0	1975*	2,008	3,330	2,820	—		

Sources: (1) United Nations, *Selected World Demographic Indicators by Countries 1950–2000* (Population Division, Department of Economic and Social Affairs of the United Nations, 1975).
(2) Unstarred: United Nations Educational, Scientific, and Cultural Organization, *Statistical Yearbook, 1973* (Paris, 1974), table 1.4; starred: United States, State Department, *Background Notes*, individual country volumes, various years 1970–76.
(3) United Nations, *Statistical Yearbook, 1974*, tables 181, 188.
(4) United Nations, Food and Agriculture Organization, *The State of Food and Agriculture 1974: World Review* (Rome, 1975).
(5) Jain, Shail, "Size Distribution of Income" (International Bank for Reconstruction and Development, Bank Staff Working Paper no. 190, November, 1974).
(6) Kenneth Clark et al., *Area Handbook for the Republic of China* (Washington, D.C.: Department of the Army, 1969), p. viii.
(7) Taiwan, Council for International Economic Cooperation and Development, *Taiwan Statistical Data Book* (1972). All figures refer to 1970.

Table 5.A.3 **National Indexes Used in Analysis of Mortality Change, 1940–70**

	Malaria Endemicity in 1943[a] (1)	Annual Average International Economic Aid Received 1954–72[b] [c] (2)	External Assistance Received for Community Water Supply and Sewage Disposal Projects 1966–70[b] (3)
Australia	1	—0	0
Belgium	0	—0	0
Canada	0	—0	0
Chile	1	13.29	.14
Colombia	3	5.13	2.30
Czechoslovakia	0	—0	0
Denmark	0	—0	0
Egypt	2	7.61	.18
Finland	0	—0	0
Greece	2	19.55(4)	0
Guatemala	3	3.35[d]	3.45
Honduras	3	4.05[d]	.90
Hungary	0	—0	0
India	3	1.71	0
Ireland	0	—0	0
Japan	1	—0	0
Korea (South)	1	10.15	.52
Luxembourg	0	—0	0
Mexico	3	1.50	.34
Netherlands	0	—0	0
New Zealand	0	—0	0
Nicaragua	3	5.78[d]	3.40
Panama	3	10.79	19.48
Peru	2	4.13	3.55
Philippines	3	2.02	.58
Portugal	1	—0	0
Puerto Rico	2	—0	0
Spain	1	9.22(4)	0
Sweden	0	—0	0
Switzerland	0	—0	0
Taiwan	3	7.09	0
Thailand	3	1.63	.09
Turkey	3	—0	0
United Kingdom	0	—0	0
United States	0	—0	0
Venezuela	3	2.82	3.71

Sources: (1) Shattuk (1951, p. 4; 1943 map prepared by U.S. Army Medical Intelligence Branch); Faust (1941); Boyd (1949).
(2) United Nations, *Statistical Yearbook* (1958–74).
(3) World Health Organization, *World Health Statistics Report* (1973), vol. 26, no. 11.
(4) Organization for Economic Cooperation and Development, *Development Cooperation: Efforts and Policies of the Members of the Development Assistance Committee, 1973 Review* (Paris, 1973).

Notes

1. The zero-order correlation between e^0_0 and the natural log of national income per capita for the 120 countries in appendix table 5.A.2 is 0.859; the correlation between e^0_0 and national income itself is only 0.693.

2. Causes eliminated include influenza/pneumonia/bronchitis, diarrheal disease, and maternal mortality, and also a proportion of "other and unknown causes" equal by age to the proportion of known causes assigned to infectious diseases at that age.

3. The age distribution used for direct standardization is that of a female "West" stable population with $e^0_0 = 65$ and $r = .01$ (Coale and Demeny 1966).

4. WHO in conjunction with the United Nations Population Division has estimated that life expectancy for LDCs as a whole was 49.6 in 1965–70 (World Health Organization 1974*b*, p. 23). Life expectancy in 1900 is the author's guess based upon life tables calculated by Arriaga for Latin America and on life tables for India, Taiwan, and Japan around the turn of the century. Corresponding ASCDRs were computed by the author based on relationships between the two mortality measures in the set of 165 populations employed by Preston and Nelson (1974).

5. Pairwise deletion was employed for missing data in the 1940 regressions. That is, results are based upon correlation matrices computed exclusively on the basis of data that were available. N is taken as 28, the number of cases for which observations were complete. One observation was missing on literacy, three on national income, and four on calorie consumption. For no country was more than one piece of information missing. The data on LDCs were considered too valuable to sacrifice all information because one piece was missing. Standard errors are shown in parentheses.

6. Several alternative specifications of these equations were employed, but results were not appreciably altered. These included polynomial representations of income and calories and measurement of CAL from different base points. The R^2's for equations including first- and second-degree terms for Y and CAL were lower than those for the specification presented in the text despite the addition of two variables. The use of other base points for CAL left R^2 unaffected to four decimals. Population-weighted regressions were rejected on the grounds that they gave too much weight to India, where measurement of variables was believed to be unusually poor, especially in 1940.

7. There are of course many ways to attribute differences to changes in values of variables and to changes in coefficients, none of them clearly preferable. In this instance we are constrained by the unavailability of data for most LDCs in 1940. Substitution of 1970 values of the regressors into the 1970 equation will not, of

---

[a]0 = virtually none; 1 = low; 2 = moderate; 3 = high.

[b]Current U.S. dollars per capita.

[c]Includes net official flow of external resources to individual countries from developed market economies and multilateral agencies and bilateral commitments of capital by centrally planned economies. "Military expenditures and contributions are excluded as far as possible."

[d]Information not available for 1966 through 1968.

[e]Information for 1970–72 from source 4.

course, usually yield the correct 1970 life expectancy for a country. But the predictions must be very nearly correct in the aggregate, since the regression plane must pass through the mean of the variables and since 94 of the 120 observations are LDCs.

8. This figure is close to the 9.7 estimate derived by Preston (1975*a*, p. 238), who considered only income and used cruder, regional income distributions of 1963 to evaluate changes between 1938 and 1963.

9. Once again, pairwise deletion is employed for cases of missing data.

10. The mean values of *AID* and *WAT* for the 17 LDCs were 4.77 and 2.21, respectively.

11. $16.46 = 31.47 - .0211(2.59) - .4063(39.39) + .0750(4.77) + .2939(2.21)$. Of the 16.46 year gain, 1.01 years is attributed to external aid.

12. A large majority of countries in the WHO survey listed lack of financing as the principal barrier to expanded water supply and sewerage systems (World Health Organization 1973).

13. For an account of the impact of the Catholic diocese of Oklahoma on mortality in a Maya village, see Early 1970. The experience was probably repeated hundreds of times.

14. For ample documentation, see World Bank (1975) and Bryant (1969).

15. Compiled from United Nations, *Statistical Yearbook*, 1974, table 197. Figures refer to thirty-nine LDCs.

16. WHO has compiled estimates of private consumption expenditures on health care in certain LDCs (World Health Organization 1970). The percentage of total private consumption that is spent on health can be compared with the proportion of GNP represented by government health expenditure, a procedure that reduces incomparabilities resulting from differences in the years of estimate. The percentages are the following (private consumption appearing first): Sierre Leone, 2.9 and 0.9; Jordan, 0.6 and 2.8; Thailand, 3.6 and 1.2; the Philippines, 1.7 and 0.5; Malaysia, 2.4 and 2.5; Panama, 4.0 and 2.2; Jamaica, 1.1 and 2.7. Government health expenditures are from World Bank (1975, annex 3).

17. WHO, for example, does not absorb any materials costs but views its role exclusively as providing technical, advisory, and educational assistance (Goodman 1971, pp. 203–4). Dispute over the provisions of assistance for material led to the "resignation" from WHO of Soviet-bloc countries between 1949 and 1955.

18. The class distribution of gains in mortality is not well documented. There was a sharp contraction of age-standardized mortality ratios for various classes in England during the twentieth century (Antonovsky 1967, p. 63). Even constant ratios would entail reductions in absolute differences, which is the pertinent index when population composition is considered.

19. This is a less serious problem than it might at first appear. The coefficients of $\ln Y$ presented earlier range from 3.6 to 7.0 (the latter observed when no other term except income distribution is present in the cross-sectional analysis). A 1-point increase in the rate of per capita economic growth would thus be associated with at most a gain in life expectancy of 0.07 years. A gain in e^o_0 of 2 years is roughly associated with a drop in CDR of 0.0015. Thus, a 0.01 gain in the rate of economic growth would be expected to reduce the CDR by at most 0.07 $(.0015/2) = 0.000053$, or by 0.53% of the change in R_{pc}.

20. Earnings differentials were obtained from a 1963 survey of 4,000 urban wage earners. Adjusted earnings differentials presented in Carnoy's (1967) table 3 are employed. Retirement was assumed to occur at age 65. The continuously compounded rate of return was calculated using the formula presented in the text, with

P.V. set equal to zero. For calculation of social costs, the average annual public expenditure per student by grade is added to direct personal outlays and income forgone.

Comment J. D. Durand

I will attempt to fit some of Preston's findings with regard to the determinants of mortality, and related results in Ronald Lee's paper (chap. 9), into a sketch of salient features of the evolution of determinants of the overall levels and trends of mortality during recent centuries and decades.

Results of recent work in this field at the University of Pennsylvania suggest that the typical form of the trend of expectation of life in countries making the transition from premodern to modern regimes of mortality may be represented by an essentially logistic curve, which can be divided into fairly distinct phases as follows:

0—pretransitional phase, in which expectation of life fluctuates around a nearly constant long-term level;

1—initial phase of transition, in which expectation of life increases irregularly at a relatively slow long-term average rate;

2—"takeoff" phase, in which expectation of life rises at a steady, rapid rate;

3—final phase, in which expectation of life rises slowly and appears to be approaching a ceiling at a high level.

I will attempt to draw a tentative general sketch of principal causal factors that have contributed to the increases in expectation of life (e) during each of these phases of the transition, in terms of the following simplified formula: $e = f(y, k, s, n, \ldots)$ where y stands for income per head, k for knowledge of the causes of disease and death and methods of prevention and treatment (including what the layman knows as well as what the physician knows), s for social action in the broad field of health protection, and n for natural factors.

This is not a comprehensive formulation of the determinants of mortality. Preston shows that the distribution of income and nutrition are influential; among other factors that may have played significant roles in the gains of life expectancy during modern times are the advance of popular education and the decline of fertility; no doubt urbanization has influenced the trends in various countries, and other factors could be

J. D. Durand is professor of economics and sociology and a research associate of the Population Studies Center, University of Pennsylvania.

mentioned. However, probably the greater part of the increases in expectation of life in most parts of the world during the last two centuries can be attributed to the growth of y, the advances of k and s, and some favorable changes in n. I do not presume that the effects of these factors have been simply additive. It is not to belittle the value of Preston's regression models to postulate, for example, that the effects of given changes in each and all of the factors have varied with the levels of e, as is shown vividly by the smallness of recent gains of life expectancy in countries where the highest levels have been achieved.

Pretransitional Regimes

Lee's study of preindustrial England provides a most valuable example of conditions and factors of mortality in a pretransitional state. In this case, we may disregard factors k and s, assuming that they were constant in effect, at least up to the eighteenth century. From the thirteenth to the eighteenth century, there were important variations of mortality in England, both long-term and short-term, and Lee finds that these were due mainly to noneconomic factors; that is, presumably natural factors in the main. The identification of these natural factors remains an unsolved puzzle. Lee says, "they may have been climatic, or the by-product of independent epidemiological changes, or the result of voyages of exploration."[1] As regards climate, Le Roy Ladurie's work is rather discouraging to hopes of finding in its variations a satisfactory explanation for the long swings in mortality, but the question of its influence on the hazards of disease as well as on agriculture has by no means yet been disposed of. There were also important long- and short-term ups and downs of y, represented in Lee's analysis by indicators of wages and prices, and he finds that their influence on mortality was not negligible, although it was less potent than the influence of n. One of the most interesting features is the low ceiling over e. Apparently even the wealth of kings and dukes would not purchase more than about 25 to 35 years of life expectancy, depending on the conditions of n. The ceiling is much higher today, but it is still firm. Unlimited growth of national income per head seems unlikely under present conditions to bring expectation of life for the two sexes much above 75 years.

England's economic situation during the centuries shortly before the industrial revolution was relatively favorable compared with that of most other countries, as Lee points out. Both e and y were probably considerably lower in most of the rest of the world, and it is likely that the influence of changes of y over time may have been stronger elsewhere than it was in England. Lee suggests this with reference to Goubert's observations on mortality in Beauvais during the seventeenth and eighteenth centuries. However, I would hypothesize that n was a major

factor in both temporal variations and international differences in mortality under the pretransitional regimes throughout the world.

Preston (1975) gives a chart of the changing relation between e and y in international cross sections about 1900, 1930, and 1960. If data were available to draw such a chart with reference to conditions around 1750 and earlier dates, I presume that the correlation between e and y would be seen to have been weaker, the curve representing the relation between the two variables would exhibit a less steep positive slope, and it would shift erratically up and down from one date to another under the influence of changing natural factors. A major feature of the transition to modern regimes of mortality has been progressive neutralization of the influences of n as a result of the growth of y and advances of k and s.

First Phase of the Transition

Although it is not easy to define the date of beginning of the mortality transition in any country, the secular trend of slowly rising expectation of life identified with the first phase of the transition was clearly general in Western Europe during the nineteenth century, and indications of accelerating rates of population growth suggest that it was also widespread in Eastern Europe, North Africa, and Latin America. The trend of mortality in the United States before the closing decades of the nineteenth century remains an unresolved question.

In the countries in the vanguard of industrial development during the nineteenth century, the growth of income per head was undoubtedly a major factor contributing to the decline of mortality in this first phase, but the effect of increasing y was reinforced by advances in k and s. Under the heading of k, in addition to the important innovation of smallpox vaccination, I surmise that increasing understanding of the importance of hygiene and proper feeding of children, linked with the advance of popular education in the industrializing countries, played an influential part. Under s were such social actions as protection of water supplies, urban sewerage, swamp drainage, quarantine practices, restriction of child labor, and regulation of conditions of women's employment. Such health-protective social actions were not confined to the wealthiest countries; Sanchez-Albornoz (1974) traces their development in Latin American cities during the nineteenth century.

With regard to the historical antecedents of Preston's (1975) chart of changing relations between e and y during the twentieth century, I postulate, although I cannot provide statistical proof, that the developments related to the first phase of the mortality transition during the nineteenth century had the following effects: making the correlation between e and y stronger than it had been under the pretransitional

regimes; gradually shifting upward the curve of e values corresponding to given levels of y; and making the slope of the curve steeper—that is, widening the differences in e between richer and poorer nations.

Second Phase of the Transition

The decided quickening of the rate of gain in life expectancy that marks the beginning of the second phase of the transition took place in the 1890s or about a decade earlier or later in the more developed countries of Europe and America. It seems clear that this turn of the trend was primarily a result of the first revolution in death-control technology produced by the validation and wide acceptance of the germ theory of disease. The effect of this was not limited to the new techniques of immunization and therapy for particular diseases that began to be invented late in the nineteenth century. Meanwhile, increasing income and health-protective social actions continued to contribute to gains in e, and it seems a reasonable hypothesis that the tightening control of fertility may also have contributed to the quickening reduction in child mortality.

The less developed countries in Latin America, Asia, and Africa were slow to get much benefit from the advances in k at this stage. They were handicapped in applying the new knowledge by low income, low levels of popular education, small resources at the disposal of the governments, and perhaps colonial administrations' lack of interest in taking very costly actions to protect the health of the indigenous people. So the beginnings of the second phase of the transition were delayed in most of these countries until after World War I, and in many until the 1940s or 1950s. As a result, the differences in life expectancy between more and less developed countries widened during the early decades of the present century, and the slope of the curve of e in relation to y grew steeper as it shifted upward more rapidly in the higher than in the lower brackets of per capita income.

It might be tempting to infer that countries had to reach some threshold of income and development in other respects to be eligible for rapid progress in the reduction of mortality under the conditions of this period. But some observations imply that if this were true, the level of the threshold was not high enough to explain fully why so many less developed countries were so long retarded in entering the second phase of the mortality transition:

1. The case of the eastern and southern European countries: Although they were considerably less developed than the northwestern European countries, they were quick to join their richer neighbors in the sharp acceleration of gains in e around the turn of the century or shortly afterward. In spite of handicaps in income, education, and other

aspects of development, the countries in eastern and southern Europe generally managed to keep pace with those of northwestern Europe in rates of gain in life expectancy until about the 1940s, when they began to overtake the lead of the latter.

2. The case of Cuba: In a new study of the trend of mortality in Cuba since the late nineteenth century, Diaz Briquets (1977) shows that a spectacular reduction of mortality was achieved there during the few years of United States military occupation following the Spanish-American War, by a campaign of sanitary reforms and mosquito control instigated and aided by the army of occupation. He estimates that the crude annual death rate in the city of Havana dropped from a prewar average of 32 per 1,000 in 1891–95 to 20 in 1903–7, and a large decrease was achieved in the rest of the country also, in the face of general poverty and illiteracy.

3. The case of Taiwan under Japanese rule during early years of this century is another precocious example of the effective transfer of k and s from a more developed to a less developed country (Barclay 1954). This is even more remarkable than the Cuban case, because when the Japanese arrived Taiwan was a good deal less developed than Cuba, and the Japanese themselves had not yet reached a very high level of either e or y.

4. A decided upturn of the trend of e took place during the 1920s in a number of less developed countries (Cuba, Japan, Taiwan, and others). Diaz Briquets (1977) observes that this seems to have been especially characteristic of countries where export industries were dominant, and he suggests that an economic boom in such countries in the 1920s owing to expanding demand and rising prices for their exports might account for their having entered the second phase of the mortality transition earlier than other less developed countries did. The interest of their more developed trading partners in making these countries healthy places to do business with and in may also have been a factor.

The relevance of the trend of income to the life expectancy gains in less developed countries is illustrated in reverse by the example of Cuba in the 1930s and early 1940s, when the misfortunes of the international market for sugar cast Cuba into economic doldrums. Diaz finds that the decline of mortality in Cuba was checked and probably temporarily reversed during this period, and that worsening nutrition and diminishing public and private expenditures on health services were important factors.

A second revolution in the technology of disease control began about 1935 and progressed rapidly during the 1940s and 1950s, with major advances of k especially in the fields of immunization, chemotherapy, and chemical control of disease vectors. This time, the less developed

countries were the principal beneficiaries. Although measures of trends in e since 1940 are lacking for many of these countries, especially among those at the lowest levels of development, it is apparent that substantial gains since that time have been practically universal in the less developed regions of the world. Preston's findings suggest that 50% to 80% of the gains between 1940 and 1970 in less developed countries may be attributable to k and s factors, but advances on these fronts have not gone so far as to make economic factors irrelevant. Gains in e since the 1940s have been less spectacular in the least developed countries, particularly in Africa, than in those that were somewhat more developed, and Preston finds a positive association between rates of increase in e and y among less developed countries.

A tendency toward slackening rates of gain in e in less developed countries is apparent in the 1960s and 1970s. While this might be due partly to slowing economic growth, Preston links it with a diminishing rate of "structural change," that is, slowdown of the advance of k and s. He observes that only a few innovations of major importance to health technology have been made during the last decade. The implication is that upward shifting of e in relation to y may be drawing to an end and that e gains in less developed countries henceforth may depend mainly on their ability to move up on the scales of y and related social developments. This has been suggested in a number of recent studies. However, there may still be a good deal of scope for raising e in less developed countries where it remains relatively low, through the pursuit of s actions to take fuller advantage of existing k at their present levels of y. To cite once again the example of Cuba: the series of Cuban life tables compiled by Diaz Briquets (1977) shows expectation of life at birth for the two sexes increasing from 58.8 years in 1953 to 72.1 in 1971 (70.6 for males, 73.9 for females). The 1971 figure compares favorably with that of the United States, especially for males. Diaz presumes that most of the gain in Cuba since 1953 has taken place since the establishment of the socialist government, and he attributes the high rate of gain since that time mainly to more equal distribution of income and government policies aimed at equalizing access to health services for all categories of the population.

Although the less developed countries were the main beneficiaries of the new advances in k since the 1930s, the more developed countries also benefited to an important extent. Examination of the trends in a number of more developed countries shows that the 1940s were a bumper decade for gains in life expectancy. Preston's analysis indicates that a major share of the gains in more developed as well as less developed countries since 1930 has been due to the upward shifting of the curve of e in relation to y and other indicators of development, which may be attributed to the advances of k and s.

Third Phase of the Transition

In more developed countries, the rates of gain in e have slowed conspicuously since the 1950s, and in many of them hardly any gains have been registered during the past ten years. The same tendency is noticeable in recent statistics from some less developed countries that have attained levels of e comparable to those of more developed countries. This feature is commonly interpreted as meaning that the expectation of life in countries where it is now highest is approaching a ceiling that cannot be surpassed by increasing income or by other means unless a new revolution in medical technology is achieved—a revolution that would make possible important gains in control over the so-called degenerative diseases. If this interpretation is correct and if the less developed countries where e is still well below such a ceiling manage to continue progress in reducing their mortality rates through economic development and fuller application of the present medical knowledge, the time may come when levels of mortality will be nearly equalized among countries around the world. The advances of k and s would then have neutralized, to a large extent, the influence of y as well as that of n.

An interesting aspect of the recent trends in countries where e is high is that they seem to be leveling off at considerably different values of e. If they are coming up against a ceiling, the level of the ceiling seems not to be the same in all countries. In the United States, for example, e seems to be stagnating some four or five years below the level achieved in the Scandinavian countries. On the surface, these differences do not seem very consistently related to per capita income. I mentioned earlier that Cuba's estimated expectation of life in 1971 compared favorably with that of the United States. The position of Puerto Rico and Hong Kong is similar, although they are far below the United States in per capita income. Perhaps a part of the explanation for such anomalies might be found in factors associated with advanced economic development that are unfavorable to health and longevity. I think it would be interesting to make a systematic study of factors related to the different levels of e, and of mortality rates for sex and age groups, causes of death, and so forth, at which the trends seem recently to have been stalling in many countries. The distribution of income, governmental action in fields of health care, environmental pollution, diets, and behavioral patterns relevant to health are among the factors that might usefully be examined.

Note

1. McNeill (1976) argues that major factors in the long-range trends of mortality and population growth in Europe between the fourteenth and eighteenth centuries were new diseases resulting from increased contacts with the Orient during the Mongol conquests and a subsequent gradual adaptation to these diseases, as well as variations of climate.

Comment

Victor R. Fuchs

The paper by Preston has two principal purposes: to explain the increase in life expectancy in the less developed countries over the past several decades and to consider the effect of this increase on population size, output, and output per capita. The bulk of the paper is concerned with the first question, and I shall limit my comment to that. Furthermore, I shall examine only one aspect of Preston's multifaceted discussion—the attempt to partition the gain in life expectancy into the portion attributable to increased per capita income and the portion due to a structural shift in the relationship between life expectancy and per capita income.

Using the data Preston provides, I have run regressions of life expectancy (*LE*) on the natural logarithm of per capita income (*LnY*) for four separate groups of countries: LDCs in 1940 (*L40*), MDCs in 1940 (*M40*), LDCs in 1970 (*L70*), and MDCs in 1970 (*M70*).[1] The results are presented in table C5.1. I have also plotted the predicted (from the regressions) relationship between life expectancy and per capita income for each group in figure C5.1. The curves are plotted over the range of per capita income actually observed for each group.

Table C5.1 **Results of Regressing Life Expectancy on Logarithm of per Capita Income across Less Developed and More Developed Countries in 1940 and 1970**

$LE = a + b \ln Y$	L40	M40	L70	M70
b	4.99	6.65	7.82	.13
σ_b	(1.95)	(1.21)	(1.10)	(.85)
a	13.3	17.1	12.5	70.8
σ_a	(10.0)	(8.0)	(6.7)	(6.6)
R^2	.27	.68	.77	−.08
N	16	15	16	15

Inspection of figure C5.1 suggests that the structural relation between life expectancy and per capita income in the LDCs in 1940 was different from that in the MDCs in the same year. Given the level of income, life expectancy seems to have been appreciably higher in the MDCs. It is therefore inappropriate to pool the two groups of countries, as Preston does, without allowing for differences in structure. It should also be

Victor R. Fuchs is professor of economics at Stanford University and is a research associate of the National Bureau of Economic Research.

The author acknowledges the financial support of the Robert Wood Johnson Foundation, the research assistance of Sean Becketti, and the helpful comments of Robert J. Willis.

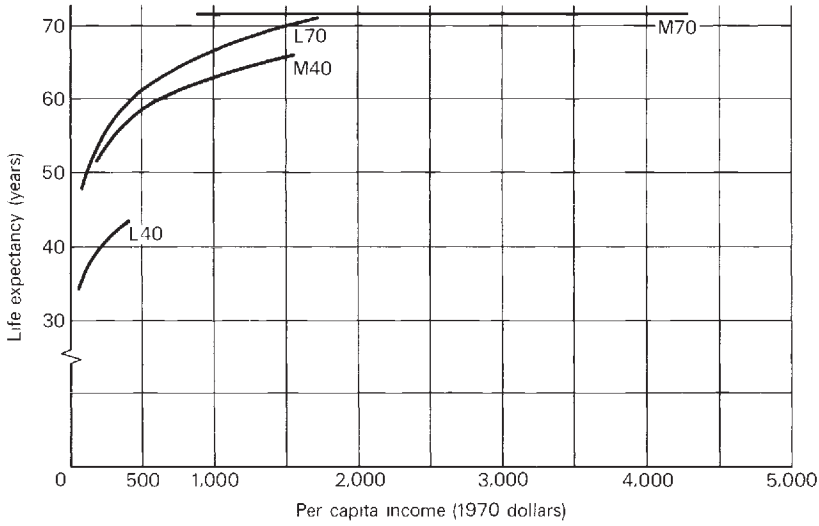

Fig. C5.1 The relationship between life expectancy and per capita income across less developed and more developed countries in 1940 and 1970.

noted that in the MDCs in 1970, the relationship between life expectancy and per capita income has disappeared, a phenomenon I have called attention to before (Fuchs 1965, 1974). There can be no question, therefore, of attempting to assess shifts in the function between the MDCs in 1940 and 1970 or between the LDCs and MDCs in 1970.

I have run regressions that pool LDCs and MDCs in 1940 and LDCs in 1940 and 1970 with dummy variables inserted to allow for differences in intercepts and slopes. These results are reported in table C5.2.

The first regressions (part A) constrain the slopes of the pooled groups to be equal but allow the intercepts to vary. We see that the shift coefficients are large and highly significant in both cases. This means that, compared with the LDCs in 1940, both the MDCs in 1940 and the LDCs in 1970 had substantially higher life expectancy for any given level of per capita income. The second set of regressions (part B) constrains the intercepts to be equal and allows the slopes to vary within each pair of groups. We now find that the slopes do differ significantly. The third set of regressions (part C) allows both the intercepts and the slopes to vary, and with this specification none of the interactions are statistically significant.

If one does not demand statistical significance, it is possible to answer Preston's question fairly unambiguously along the following lines. The mean life expectancy of the LDCs rose from 38.8 years in 1940 to 59.6

Table C5.2 Pooled Regression Results with Interactions

	L40 + M40	L40 + L70
A. $LE = a + b\ ln\ Y + c$ Intercept		
b	5.83	6.83
σ_b	(1.14)	(1.04)
c	−13.4	−14.6
σ_c	(2.10)	(1.70)
a	22.4	18.4
σ_a	(7.54)	(6.34)
$\bar{R}^2$	.91	.89
N	31	32
B. $LE = a + b\ ln\ Y + d$ Slope		
b	7.00	7.77
σ_b	(1.00)	(.95)
d	−2.29	−2.69
σ_d	(.35)	(.30)
a	14.7	12.8
σ_a	(6.50)	(5.74)
$\bar{R}^2$	.91	.90
N	31	32
C. $LE = a + b\ ln\ Y + c$ Intercept + d Slope		
b	6.65	7.82
σ_b	(1.63)	(1.27)
c	−3.79	.78
σ_c	(13.60)	(11.80)
d	−1.66	−2.83
σ_d	(2.31)	(2.15)
a	17.1	12.5
σ_a	(10.7)	(7.73)
$\bar{R}^2$	.91	.90
N	31	32

years in 1970, a rate of increase of 1.4% per annum (see table C5.3).[2] Over that same period the mean per capita income (in 1970 dollars) rose from $194 to $560. We can estimate what the change in life expectancy would have been as a result of income change alone by moving along either the 1940 predicted relation or the 1970 predicted relation. The former tells us that life expectancy would have changed from 39.6 years to 44.8 years, an increase of 0.4% per annum. If we calculate the change along the 1970 curve, we get a predicted increase of 0.5% per annum, from 53.7 years to 62.0 years.

Alternatively, we can look at the implied rates of change attributable to structural shift by comparing predicted life expectancies at the same per capita income in the two years. At $194 the implied change is 1.0% per annum; at $560 it is 1.1% per annum. Thus, either approach indicates that about one-third of the observed change in life expectancy in

Table C5.3 **Life Expectancy in Less Developed Countries in 1940 and 1970: Actual and Predicted Changes**

	Mean Life Expectancy (Years)
Actual 1940 (A40)	38.78
Actual 1970 (A70)	59.59
Predicted 1940 (P40:40) (from L40 regression)	39.56
Predicted 1970 (P70:40) (from L40 regression)	44.85
Predicted 1940 (P40:70) (from L70 regression)	53.68
Predicted 1970 (P70:70) (from L70 regression)	61.97
	Rates of Change (Percentage per Annum)
A40 to A70	1.4
P40:40 to P70:40	0.4
P40:70 to P70:70	0.5
P40:40 to P40:70	1.0
P70:40 to P70:70	1.1

the LDCs between 1940 and 1970 can be attributed to the growth of per capita income, and about two-thirds to a shift in the life expectancy–income relationship. Preston presents one estimate of 50% due to structural change and another of 80% due to that source. The results presented here are quite consistent with those estimates.

Notes

1. The LDCs are all in Asia, Africa, and Latin America, and all had life expectancies below 50 years in 1940. The MDCs are all in Europe, North America, and Australia, and all had life expectancies above 50 years in 1940.

2. The change is expressed in percentage per annum in order to minimize the problem of interaction between shifts in the function and movements along the function.

Comment

Richard W. Parks

Preston presents an interesting regression test of the relative importance of private and national income in determining life expectancies. In light of the Kuznets and Fishlow discussions of interaction between the income distribution and the age distribution of the population, it may be useful to point out a possible bias in the Preston regression in section 5.1.1.

The income distribution as commonly measured does not correct for the age distribution of the population. Thus, in a hypothetical world with *no* differences among individuals in their lifetime income streams, we will observe considerable income inequality as conventionally measured if individuals follow the usual life-cycle pattern of earning and saving followed by retirement. For the determination of the effect of income on life expectancy, it appears that a permanent income rather than a measured income concept makes more sense, but given the data available to him, Preston relies on the distribution of measured income.

Preston's distribution measure, which I shall call $D = \Sigma\, 1n(S_i/.05)$, takes values ranging from $-\infty$ to 0 on a scale representing increasing equality. Thus we can represent the partial relationship between life expectancy e^o_o, and D as shown in figure C5.2.

We expect a positive association. Suppose we start at point A with given (unequal) distribution and low life expectancy. If incomes were to become more equal (in a life-cycle sense), we would expect to find a new point at B showing greater equality and higher life expectancy. However, even with the pattern of life-cycle income corresponding with point B, there is likely to be an effect on the measured income distribu-

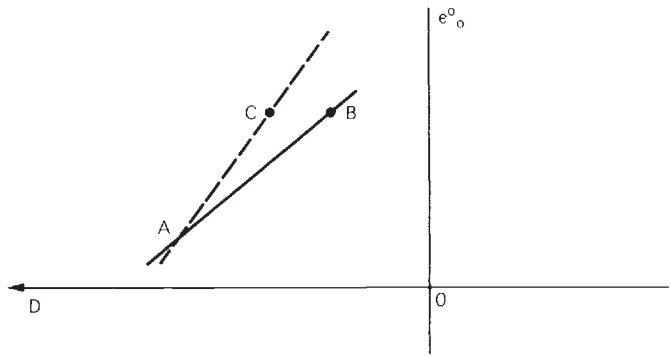

Fig. C5.2

Richard W. Parks is associated with the Department of Economics, University of Washington.

tion arising from the altered life expectancy. An increase in the share of population in nonproductive years, for example, older age groups, will have the effect of increasing the observed inequality. The observation based on measured income will be at point *C*, giving an upward bias to the slope coefficient. Since the crucial test of the relative importance of national and private incomes in the determination of life expectancy depends on the size of the slope coefficient, the upward bias would tend to suggest the absence of an effect for national income even when it was in fact important.

References

Allan, William. 1965. *The African husbandman.* London: Oliver and Boyd.

Antonovsky, Aaron. 1967. Social class, life expectancy, and overall mortality. *Milbank Memorial Fund Quarterly* 45: 31–73.

Arriaga, Eduardo. 1967. The effect of a decline in mortality on the gross reproduction rate. *Milbank Memorial Fund Quarterly* 45: 333–52.

———. 1968. *New life tables for Latin American populations.* Berkeley: Institute of International Studies, University of California.

———. 1970. *Mortality decline and its demographic effects in Latin America.* Berkeley: Institute of International Studies, University of California.

Ascoli, Werner; Guzman, Miguel A.; Scrimshaw, Nevin S.; and Gordon, John E. 1967. Nutrition and infection field study in Guatemalan villages, 1959–1964. IV. Deaths of infants and preschool children. *Archives of Environmental Health* 15 (October): 439–49.

Baertl, Juan M.; Morales, Enrique; Verastegui, Gustavo; and Graham, George. 1970. Diet supplementation for entire communities: Growth and mortality of children. *American Journal of Clinical Nutrition* 23(6): 707–15.

Bailey, K. V. 1975. Malnutrition in the African region. *Chronicle of the World Health Organization* 29: 354–64.

Banks, Arthur. 1971. *Cross-polity time-series data.* Cambridge: MIT Press.

Barclay, George W. 1954. *Colonial development and population in Taiwan.* Princeton: Princeton University Press.

Barlow, Robin. 1967. The economic effects of malaria eradication. *American Economic Review* 57(2): 130–48.

———. 1968. *The economic effects of malaria eradication.* Economic Research Series no. 15. Ann Arbor: University of Michigan School of Public Health.

Beck, Ann. 1970. *A history of the British medical administration of East Africa, 1900–1950.* Cambridge: Harvard University Press.

Boserup, Ester. 1965. *The conditions of agricultural growth: The economics of agrarian change under population pressure.* Chicago: Aldine.

Bowers, John. 1973. The history of public health in China to 1937. In *Public Health in the People's Republic of China*, ed. Myron E. Wegman, Tsung-Yi Lin, and Elizabeth F. Purcell, pp. 26–45. New York: Josiah Macy, Jr., Foundation.

Boyd, M. F. 1949. *Malariology.* Vols. 1 and 2. Philadelphia: W. B. Saunders.

Bryant, John. 1969. *Health and the developing world.* Ithaca: Cornell University Press.

Bunle, Henri. 1954. *Le mouvement naturel de la population dans le monde de 1906 à 1936.* Paris: Institut d'études demographiques.

Burch, Thomas K., and Gendell, Murray. 1970. Extended family structure and fertility: Some methodological and conceptual issues. *Journal of Marriage and the Family* 32(2): 227–36.

Carnoy, Martin. 1967. Rates of return to schooling in Latin America. *Journal of Human Resources* 2(3): 359–74.

Coale, Ansley J. 1956. The effects of changes in mortality and fertility on age composition. *Milbank Memorial Fund Quarterly* 34: 79–114.

Coale, Ansley J., and Demeny, Paul. 1966. *Regional model life tables and stable populations.* Princeton: Princeton University Press.

Coale, Ansley J., and Hoover, Edgar M. 1958. *Population growth and economic development in low-income countries.* Princeton: Princeton University Press.

Committee for International Coordination of National Research in Demography. 1975. *Seminar on infant mortality in relation to level of fertility.* Paris.

Davis, Kingsley. 1951. *The population of India and Pakistan.* Princeton: Princeton University Press.

———. 1955. Institutional patterns favoring high fertility in underdeveloped areas. *Eugenics Quarterly* 2: 33–39.

———. 1956. The amazing decline of mortality in underdeveloped areas. *American Economic Review* 46 (May): 305–18.

———. 1973. Cities and mortality. *Proceedings*, International Population Conference, Liège, Belgium, 3: 259–81.

Denison. Edward F. 1962. *The sources of economic growth in the United States and the alternatives before us.* New York: Committee for Economic Development.

Diaz Briquets, Sergio. 1977. Mortality in Cuba: Trends and determinants, 1880 to 1971. Ph.D. diss., University of Pennsylvania.

Durand, John R. 1967. A long-range view of world population growth. *Annals of the American Academy of Arts and Sciences* 369: 1–8.

Early, John. 1970. The structure and change of mortality in a Maya community. *Milbank Memorial Fund Quarterly* 48: 179–201.

Eversley, D. E. C. 1957. A survey of population in an area of Worcestershire from 1660 to 1850 on the basis of parish registers. *Population Studies* 10: 253–79.

Faust, Ernest Carroll. 1941. The distribution of malaria in North America, Mexico, Central America, and the West Indies. In *A symposium on human malaria*, ed. F. R. Moulton, pp. 8–18. Washington, D.C.: American Association for the Advancement of Science.

Fein, Rashi. 1964. Health programs and economic development. In *The economics of health and medical care. Proceedings of the conference on the economics of health and medical care, May 10–12, 1962*, ed. H. E. Klarman. Ann Arbor: Bureau of Public Health Economics and Department of Economics, University of Michigan.

Foege, William H.; Millar, J. D.; and Henderson, D. A. 1975. Smallpox eradication in West and Central Africa. *Bulletin of the World Health Organization* 52: 209–22.

Food and Agriculture Organization of the United Nations. 1975. *Population, food supply, and agricultural development*. Rome: FAO.

Fredericksen, Harald. 1961. Determinants and consequences of mortality trends in Ceylon. *Public Health Reports* 76 (August): 659–63.

———. 1966*a*. Determinants and consequences of mortality and fertility trends. *Public Health Reports* 81 (August): 715–27.

———. 1966*b*. Dynamic equilibrium of economic and demographic transition. *Economic Development and Cultural Change* 14 (April): 316–22.

Friedlander, Dov. 1969. Demographic responses and population change. *Demography* 6(4): 359–82.

Fuchs, V. R. 1965. Some economic aspects of mortality in the United States. National Bureau of Economic Research Study Paper.

———. 1974. Some economic aspects of mortality in developed countries. In *The economics of health and medical care*, ed. Mark Perlman, pp. 174–93. Proceedings of a conference held by the International Economic Association at Tokyo. London: Macmillan.

Goodman, Neville M. 1971. *International health organizations and their work*. Edinburgh: Churchill Livingstone.

Gordon, John. 1969. Social implications of nutrition and disease. *Archives of Environmental Health* 18(2): 216–34.

Habakkuk, H. J. 1971. *Population growth and economic development*. New York: Humanities Press.

Hansluwka, Harald. 1975. Health, population, and socio-economic development. In *Population growth and economic development in the Third World*, ed. Léon Tabah, 1: 191–250. Dolhain, Belgium: Ordina.

Hinman, E. Harold. 1966. *World eradication of infectious diseases.* Springfield, Ill.: Charles C. Thomas.

Howard-Jones, N. 1974. The scientific background of the International Sanitary Conferences, 1851–1938. *Chronicle of the World Health Organization* 28(10): 455–70.

Johnson, D. Gale. 1974. Population, food, and economic adjustment. *American Statistician* 28(3): 89–93.

Keyfitz, Nathan, and Fleiger, Wilhelm. 1968. *World population: An analysis of vital data.* Chicago: University of Chicago Press.

———. 1971. *Population: Facts and methods of demography.* San Francisco: W. H. Freeman and Company.

Kitagawa, E. M., and Hauser, P. M. 1973. *Differential mortality in the United States: A study in socioeconomic epidemiology.* Cambridge: Harvard University Press.

Klarman, Herbert E. 1967. Present status of cost-benefit analysis in the health field. *American Journal of Public Health* 57(11): 1948–53.

Kuznets, Simon. 1975. Population trends and modern economic growth: Notes towards an historical perspective. In *The population debate: Dimensions and perspectives*, 1: 425–33. Papers of the World Population Conference, Bucharest: New York: United Nations.

Lancet. 1970. Strategy of malaria eradication: A turning point? *Lancet* editorial. 1970 (21 March): 598–600.

McDermott, W. 1966. Modern medicine and the demographic-disease pattern of overly traditional societies: A technological misfit. *Journal of Medical Education* 41(9): 138–62.

McKeown, Thomas. 1965. Medicine and world population. In *Public health and population change*, ed. Mindel C. Sheps and Jeanne Clare Ridley. Pittsburgh: University of Pittsburgh Press.

McKeown, Thomas, and Record, R. G. 1962. Reasons for the decline of mortality in England and Wales during the 19th century. *Population Studies* 16: 94–122.

McNeill, William H. 1976. *Plagues and peoples.* Garden City, N.Y.: Anchor Press/Doubleday.

Malenbaum, Wilfred. 1970. Health and productivity in poor areas. In *Empirical studies in health economics*, ed. Herbert E. Klarman, pp. 31–57. Baltimore: Johns Hopkins University Press.

Mandle, Jay R. 1970. The decline in mortality in British Guiana, 1911–1960. *Demography* 7, no. 3 (August): 303.

Maramag, Ileana. 1965. The cost of public health programs in the Philippines. *Journal of the American Medical Women's Association* 20 (September): 848–57.

Marshall, Carter L.; Brown, Roy E.; and Goodrich, Charles H. 1971. Improved nutrition vs. public health services as major determinants of world population growth. *Clinical Pediatrics* 10 (July): 363–68.

Mata, Leonardo, et al. 1972. Influence of recurrent infections on nutrition and growth of children in Guatemala. *American Journal of Clinical Nutrition* 25 (November): 1267–75.

Meegama, S. A. 1967. Malaria eradication and its effect on mortality levels. *Population Studies* 26(3): 207–38.

Morley, David. 1973. *Paediatric priorities in the developing world.* London: Butterworth's.

Mushkin, Selma J. 1962. Health as an investment. *Journal of Political Economy* 70 (part 2, supplement): 129–57.

———. 1964. Health programming in developing nations. *International Development Review* 6(1): 7–12.

Myrdal, Gunnar. 1968. *Asian drama: An inquiry into the poverty of nations.* Vols. 1–3. New York: Random House.

Newman, P. 1965. *Malaria eradication and population growth: With special reference to Ceylon and British Guiana.* Research Series no. 10. Ann Arbor, Michigan: Bureau of Public Health Economics, School of Public Health, University of Michigan.

———. 1970. Malaria control and population growth. *Journal of Development Studies* 6: 133–58.

O'Hara, Donald J. 1975. Microeconomic aspects of the demographic transition. *Journal of Political Economy* 83(6): 1203–15.

Organization for Economic Cooperation and Development. N.d. *Resources for the developing world: The flow of financial resources to less developed countries, 1962–1968.* Paris: OECD.

Pampana, E. J. 1954. Effect of malaria control on birth and death rates. *Proceedings of the World Population Conference, 1954*, Rome, pp. 497–508. New York: United Nations.

Pampana, E. J., and Russell, Paul F. 1955. Malaria: A world problem. *Chronicle of the World Health Organization* 9(2–3): 33–100.

Pani, Alberto. 1917. *Hygiene in Mexico.* New York: G. P. Putnam's Sons.

Paul, Hugh H. 1964. *The control of diseases.* 2d ed. Baltimore: Williams and Wilkins.

Plank, S. J., and Milanesi, M. L. 1973. Infant feeding and infant mortality in rural Chile. *Bulletin of the World Health Organization* 48: 203–10.

Polgar, Steven. 1972. Population history and population policies from an anthropological perspective. *Current Anthropology* 13(2): 203–11.

Preston, S. H. 1974. Effect of mortality change on stable population parameters. *Demography* 11(1): 119–30.

———. 1975*a*. The changing relation between mortality and level of economic development. *Population Studies* 29(2): 231–48.

———. 1975*b*. Introduction. In *Seminar on infant mortality in relation to the level of fertility*, pp. 10–22. Paris: Committee for International Coordination of National Research in Demography.

Preston, S. H.; Keyfitz, N.; and Schoen, R. 1972. *Causes of death: Life tables for national populations*. New York: Seminar Press.

———. 1973. Cause-of-death life tables: Application of a new technique to worldwide data. *Transactions of the Society of Actuaries* 25 (December): 83–109.

Preston, S. H., and Nelson, V. E. 1974. Structure and change in causes of death: An international summary. *Population Studies* 28(1): 19–51.

Puffer, R. R., and Griffith, G. W. 1967. *Patterns of urban mortality*. Scientific Publication no. 151. Washington, D.C.: Pan American Health Organization.

Puffer, R. R., and Serrano, C. V. 1973. *Patterns of mortality in childhood*. Scientific Publication no. 262. Washington, D.C.: Pan American Health Organization.

Russell, Paul F. 1943. Malaria and its influence on world health. *Bulletin of the New York Academy of Medicine, September*, pp. 599–630.

———. 1968. The United States and malaria: Debits and credits. *Bulletin of the New York Academy of Medicine* 44(6): 623–53.

Rutstein, Shea, and Medica, Vilma. 1975. The effect of infant and child mortality in Latin America. In *Seminar on infant mortality in relation to the level of fertility*, pp. 225–46. Paris: Committee for International Coordination of National Research in Demography.

Sack, Richard. 1974. The impact of education on individual modernity in Tunisia. In *Education and individual modernity in developing countries*, ed. Alex Inkeles and Donald B. Holsinger, pp. 89–116. Leiden: E. J. Brill.

Sanchez-Albornoz, Nicholas. 1974. *The population of Latin America: A history*. Berkeley: University of California Press.

Schultz, Theodore. 1964. *Transforming traditional agriculture*. New Haven: Yale University Press.

Schultz, T. Paul. 1976. Interrelationships between mortality and fertility. In *Population and development: The search for selective interventions*, ed. Ronald G. Ridker, pp. 239–89. Baltimore: Johns Hopkins University Press.

Scrimshaw, Nevin S. 1970. Synergism of malnutrition and infection. *Journal of the American Medical Association* 212(10): 1685–92.

Sharpston, Michael J. 1976. Health and the human environment. *Finance and Development* 13(1): 24–28.

Shattuk, George. 1951. *Diseases of the tropics.* New York: Appleton-Century-Crofts.

Shorter, Frederick. 1968. Information on fertility, mortality, and population growth in Turkey. *Population Index* 34(1): 5.

Simmons, James Stevens; Whayne, Tom F.; Anderson, Gaylord West; Horack, Harold MacLachian; and collaborators. 1944. *Global epidemiology: A geography of diseases and sanitation.* Vol. 1. Philadelphia: J. B. Lippincott.

Smith, Peter C. 1976. Asian nuptiality in transition. Paper presented at the Seventh Summer Seminar in Population, East-West Population Institute, Honolulu.

Sorkin, Alan L. 1975. *Health economics: An introduction.* Lexington, Mass.: D. C. Heath, Lexington Books.

Stolnitz, George J. 1956. Mortality declines and age distribution. *Milbank Memorial Fund Quarterly* 34: 178–215.

———. 1974. International mortality trends: Some main facts and implications. Background Paper for United Nations World Population Conference E/CONF.60/CBP/17, Bucharest.

Taylor, Carl E., and Hall, Marie-Francoise. 1967. Health population and economic development. *Science* 157 (August): 651–57.

Titmuss, Richard M., and Abel-Smith, Brian. 1968. *Social policies and population growth in Mauritius.* London: Frank Cass.

United Nations, Population Branch. 1963. Population bulletin of the United Nations no. 6 (with special reference to the situation and recent trends of mortality in the world). New York.

United Nations, Department of Economic and Social Affairs. 1971. *The world population situation in 1970.* Population Study no. 49. New York.

———. 1973. *The determinants and consequences of population trends.* Vol. 1. New York: United Nations.

United Nations, Population Division. 1974. Recent population trends and future prospects. World Population Conference Paper E/CONF. 60/3.

Vaidyanathan, K. E. 1972. Some indices of differential mortality in India. In *Studies on mortality in India*, ed. K. E. Vaidyanathan. Gandhigram: Institute of Rural Health and Family Planning.

Vallin, J. 1968. La mortalité dans les pays du Tiers Monde: Evolution et perspectives. *Population* 23: 845–68.

Wegman, Myron E.; Lin, Tsung-yi; and Purcell, Elizabeth F., eds. 1973. *Public health in the People's Republic of China.* New York: Josiah Macy, Jr., Foundation.

Weisbrod, Burton A. 1975. Research in health economics: A survey. *International Journal of Health Services* 5(4): 643–61.

Weller, T. H. 1974. World health in a changing world. *Journal of Tropical Medicine* 77(4), suppl. 54: 54–61.

World Bank. 1975. *Health: Sector policy paper.* Washington, D. C.

World Health Organization. 1970. Special subject: Health expenditure. In *World health statistics annual, 1966–1967* 3: 236–45.

———. 1972. *Human development and public health.* Technical Report Series no. 485. Geneva: WHO.

———. 1973. Community water supply and sewage disposal in developing countries. *World Health Statistics Report* 26(11): 720–83.

———. 1974*a*. Childhood mortality in the Americas. *Chronicle of the World Health Organization* 28: 276–82.

———. 1974*b*. Health trends and prospects, 1950–2000. *World Health Statistics Report* 27(10): 672–706.

———. 1975*a*. The malaria situation in 1974. *Chronicle of the World Health Organization* 29: 474–81.

———. 1975*b*. *Fifth report on the world health situation.* Official Records of the World Health Organization, no. 225. Geneva: WHO.

———. 1975*c*. *First WHO seminar on expansion of the use of immunization in developing countries.* Offset Publication no. 16. Geneva: WHO.

———. 1976. Smallpox eradication in 1975. *Chronicle of the World Health Organization* 30: 152–57.

Wrigley, E. A. 1969. *Population and history.* New York: McGraw-Hill.