Mortality in Latin America: Emerging Patterns

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Not even the most pessimistic of forecasters two to three decades ago would have predicted an early slowdown in the mortality decline that characterized Latin American countries during the postwar years. On the contrary, the generalized mood was one of confidence that mortality conditions in the region were converging toward those in developed countries (Stolnitz, 1955, 1956; United Nations, 1973; Davis, 1956). Undoubtedly this optimism had a firm base in real processes. Latin America during the years immediately preceding and following the war experienced impressive successes in the conquest of diseases equaled in only a handful of Asian countries. Although the exact magnitude of the gains is unknown, on the average the countries of Latin America probably experienced annual increases in life expectancy close to nine-tenths of a year during the first postwar decade.

Some countries—such as Argentina, Chile, Uruguay, and possibly Costa Rica—achieved substantial gains in survivorship before new technology was deployed on a massive scale in the aftermath of World War II. These countries had been more heavily affected by European migration, were in a favorable position in the international market, and had, before World War II, already experienced some economic development. Other countries—such as the Dominican Republic, Guatemala, Nicaragua, Peru, and Bolivia—underwent little or no change in mortality before 1945, their life expectancy scarcely surpassing 40 years. By the close of the 1950s, only a handful of countries still had life expectancies below 50 years; a substantial portion of the preexisting intercountry differentials had been removed, the heterogeneity of economic development notwithstanding; and, finally, the average gap between mortality conditions on the continent and mortality conditions in Western Europe had been narrowed considerably (from about 20 years in 1935 to about 10 years in 1960). A disproportionate share of the gains in life expectancy everywhere took place very early

in life, through systematic reduction in infant and child mortality. As a consequence, the sharp inequalities between death risks in childhood and adulthood that had been observed in the predecline period were considerably smoothed.

It was widely anticipated that, shortly after the initial period of unprecedented speed in the change in life expectancy, the process would tend to follow a North American–Western European pattern, with survivorship gains accruing at a diminishing rate as life expectancies approached a biological ceiling value (Durand, 1980). The gap between the developing nations of Latin America and the developed nations in Western Europe and North America, which decreased so rapidly at the beginning, was expected to narrow at a slower rate as conditions of mortality in the two groups of nations converged. Implicit in these expectations was a set of assumptions about the process of mortality decline that was unilaterally dependent on the North American–Western European experience. These assumptions are as follows:

Irreversibility Once conditions of mortality show a marked and sustained improvement, the process is irreversible. An increase in life expectancy at birth from 30 to 40 years is taken as indicative of changes in life style, standard of living, or medical knowledge and technology that would engender even further increases. In North America and Western Europe such changes were the beginning of a process with no backward movements and sustained, although not necessarily constant, rates of increase in longevity (Stolnitz, 1955, 1956, 1965).

Continuity The *pace* of gains in life expectancy may be variable but it tends to slow markedly only as recognizable ceiling values are approached. These ceiling values reflect the limits on human longevity imposed by biological processes of growth and degeneration, as well as by the available knowledge and technology.

This assumption, too, is inextricably linked with the North American and Western European experience. The combination of changes in socioeconomic conditions and dramatic leaps in medical knowledge provided the grounds, first, for the conquest of infectious diseases and, then, for the increased control over diseases, malfunctions, and disorders of a noninfectious origin. The limits to gains in longevity are determined by the inability to control degenerative processes that accompany aging, the effects of environmental deterioration, and the persistence of various morbid conditions affecting the newborn. Although this continuity assumption frequently passes unnoticed, it has been the basis for quite common but erroneous beliefs: first, that the discovery of new methods of prevention and treatment of infectious diseases signals their virtual demise as dominant contributors to mortality levels, and second, that such discoveries will be always followed by even further improvements in mortality conditions derived from reductions in the incidence of noninfectious, nondegenerative diseases.

Similarity of mortality patterns The nature of most breakthroughs in the control of communicable diseases, in combination with the overwhelming influence of such diseases on the chances of survival of infants and children, produces gains in life expectancy that are disproportionately allocated at early ages. The consequence should be a change in the pattern of mortality, or in the relation of mortality risks at different ages, leading to a closer similarity with the European–North American pattern of mortality (United Nations, 1973). In contrast to the other two assumptions, which rest on an evaluation of total levels of mortality, this third assumption points toward expected dramatic—also continuous and irreversible—changes in infant and child mortality and in their relation to changes in adult mortality.

Lest I be misunderstood, it should be emphasized that these three assumptions have never been presented as verifiable propositions nor have they been used systematically as tools for description. They are at the core, however, of past mortality forecasts, predictions, and expectations that, when contrasted with recent evidence, have proved to be, to say the least, too optimistic. Taking the stated assumptions as a theoretical model would be simplistic, but ignoring their repeated use in a theoretical context could have undesirable consequences for our understanding of current and past trends. Some of the erroneous notions contained in them, much of the imagery they help to conjure, and the flaws in the forecasts and predictions they generate could be countered by a closer look at empirical evidence about current conditions. To anticipate some of the results to be presented here, this evidence reveals conditions of mortality decline in Latin America (and perhaps of other developing countries) that justify a less optimistic view of the future. The historical course thus far covered is essentially different from the Western European–North American model. Not only has the temporal array of causes of mortality transition been the reverse of that in North America and Western Europe but, also, the pattern of decline has shown a lack of internal balance, depending more heavily on the effects of diffusion of medical technology than on adequate access of the population to the benefits of higher standards of living. The currently sluggish pace of mortality improvements, the unexpected reversals, and the future prospects could probably be better understood if analysts were to cease to see the Latin American case as a variant of the Western European–North American experience. Instead, a new framework for analysis should be postulated, one that explicitly recognizes the peculiarities of the process being studied.[1]

Deriving an alternative framework: reversibility, discontinuity, and peculiar age patterns of mortality

Numerous researchers have attempted to illustrate the argument that most of the postwar gains in life expectancy experienced by developing countries are attributable to the beneficial influence of new medical technology. Revolution-

ary discoveries affected not only the stock of medical knowledge but, especially, its simple, low-cost application. The example of Sri Lanka's mortality decline and the protracted discussion about its nature and causes have left little doubt that exogenous factors (diffusion of vector eradication methods) had significant effects (Newman, 1965; Meegama, 1967; Fredericksen, 1961; Gray, 1974). In a different vein, Preston (1980) estimated that about 80 percent of the changes in life expectancy during 1930–70 in a sample of developed and developing areas could be attributed to changing technology. Using a different sample and a somewhat different model specification, the same author obtained a lower estimate (50 percent) for developing nations during 1940–70 (Preston, 1980). I have arrived at a similar figure using a sample of Latin American countries during 1950–70 (Palloni, 1979).

In a different context, McKeown and collaborators show convincingly that mortality decline in England during the nineteenth and part of the twentieth century is attributable to improved standards of living, including improvements in the quantity and quality of food intake and clothing and creation of water supply and refuse systems. McKeown claims that it is only after 1900, perhaps even after World War I, that breakthroughs in the application of new and already acquired medical knowledge had a decisive impact on life expectancy (McKeown, 1976; McKeown and Record, 1962). McKeown's findings are, in all likelihood, generalizable to other areas in Western Europe as well as to North America.

The findings summarized above provide some useful suggestions to interpret the course of mortality decline in Latin America. First, there appears to be some degree of independence between the effects of socioeconomic development and the effects of medical innovation. However, whereas the former can contribute to mortality decline in the absence of the latter, as in the English case, the effects of medical innovations are conditioned by socioeconomic development. Not only does the stock of knowledge to produce efficient medical interventions depend on the achievement of some degree of industrialization—a relationship that is becoming less and less relevant in that backward economic systems can to various degrees utilize new technology produced elsewhere—but, more importantly, a low level of socioeconomic development sets boundaries on the possibility of absorbing efficiently a certain type of technology. Thus, for example, spraying DDT to eradicate the malaria-producing mosquito requires a minimum of political organization (regional administrative machinery, some degree of community acceptance of local authorities' directives), minimum development of infrastructure in the form of a communications network, and, of course, insertion of the country in an international market through which the spraying materials can be bought or simply received as part of international aid programs. Other technologies, however, are viable only after certain threshold levels of economic and social development have been reached. A drastic reduction in the deleterious effects of diarrhea, for example, is inconceivable without some transformation of hygienic conditions, development of institutions to provide treatment for dehydration and other secondary effects, and, most important, adequate levels of nutrition and diffusion

of information among the population to stimulate the introduction of healthy habits and practices.[2]

The findings cited above also suggest that the order of events in the English (and by extension in the Western European and North American) mortality decline is totally reversed in the case of Latin America (and other developing countries). With a few exceptions (Arriaga and Davis, 1969), the levels of mortality in Latin American countries dropped sharply only after exposure to medical innovations had taken place, yet prior to major gains in socioeconomic development. By contrast, in Western Europe and North America substantial gains in life expectancy were made before any significant breakthrough in medicine could reach the majority of the population. Consequently, after Latin America experienced an initial drop in mortality, the potential for a slowdown of the process of mortality decline remained if socioeconomic development failed to occur at a sufficient pace. A rather perverse consequence of isolated technological innovations unaccompanied by harmonic socioeconomic development is a possible worsening of morbidity and mortality levels related to some causes. Thus, for example, in some African and Asian countries, innovations in irrigation systems stimulate the propagation of schistosomiasis, a debilitating disease that increases the morbidity and mortality of the population and seriously undermines the productivity of labor. Also the sudden, largely unregulated migration flows and the establishment of new transportation networks have facilitated the transmission of diseases (sleeping sickness, for instance) the incidence of which had formerly been purely local (Hughes and Hunter, 1970).

From these considerations and as a tool for orienting the analysis that follows, two working hypotheses can be derived:

1 The gains in survivorship at relatively low levels of life expectancy, below 40 or 45 years, in societies at low levels of socioeconomic development that are exposed to medical innovations should proceed at a much more rapid pace than in Western Europe–North America at equivalent levels of life expectancy. This is because the influence of medical innovations is more sudden and spreads over much shorter intervals of time than the contribution of improvements in standards of living. By contrast, gains in survivorship that developing societies experience at higher levels of life expectancy, 55 to 60 years, should proceed at a slower pace than the gains in Western Europe–North America at equivalent levels of life expectancy, if insufficient economic and social progress takes place. This is because the combined influence of rising standards of living and rapid diffusion of new medical technology that Western Europe and North America underwent during the 1930s and 1940s—the period within which such levels of life expectancy were reached—produced more significant gains than are likely to accrue in situations where there is slow economic growth and little room for the application of more viable medical interventions.

2 Past breakthroughs in the application of medical technology had an impact on mortality levels by: improving the survival chances after the onset of a disease (chemotherapy, for example); reducing the rate of contagion (vaccination, quarantine, for example); and minimizing the opportunities for direct

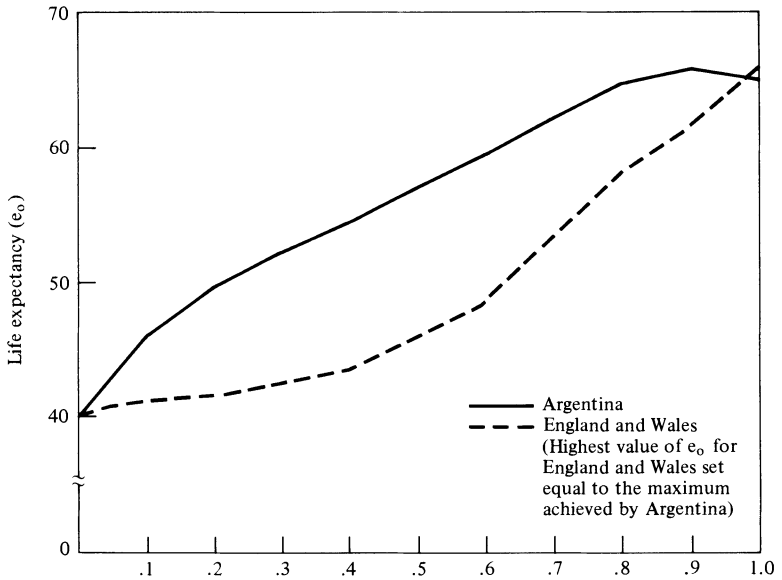
contact between the host and the agent of disease (spraying to eliminate vector-carrying agents, for example). In the absence of economic and social transformation, the generalized application of this technology could alleviate substantially the often fatal consequences of infectious diseases but could only rarely reduce their effects to insignificance. It is well known, for example, that under conditions characterized by lack of nutrition, poor clothing, precarious living quarters, and insanitary practices and habits, preventive and curative measures operate at a low level of efficiency and their effects may even be completely overridden (Scrimshaw et al., 1968). Furthermore, the influence of standard of living is particularly important among infants and children because of the precariousness of their resistance capacity. Thus, the joint occurrence of a low standard of living and contact with new medical technology could have ambiguous consequences for the youngest members of the population who have not yet fully developed natural immunities, and are dependent on others to satisfy basic needs. On the one hand, upon introduction of medical technology, the rate of decline of their mortality risks ought to be faster than for the adult population. But, on the other, the improvements may not be sustained long enough to suppress the rather sharp inequalities of death risk in infancy, childhood, and adulthood that characterize developing countries before the onset of mortality decline. As a consequence, in countries whose mortality transition has been largely dependent on medical innovations, the mortality levels among infants and children should be excessive relative to adult mortality. The excess could be quantified by comparison with the levels prevailing in countries that underwent a mortality transition heavily dependent on the joint contributions of medical innovations and socioeconomic progress. Furthermore, the stronger the dependency of mortality changes on the diffusion of medical technology, the higher should be the excess infant and child mortality.[3]

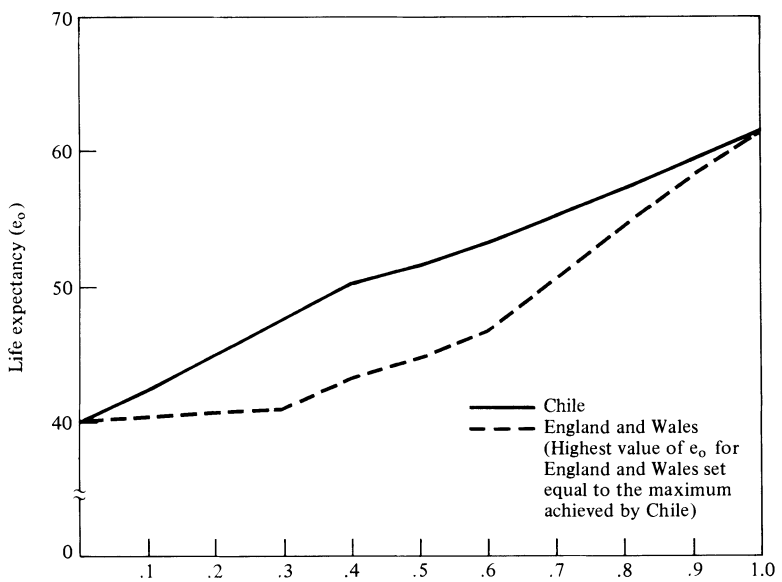
In what follows I will try to provide some illustrative evidence supporting these two working hypotheses.

The pace of overall mortality: reversibility and discontinuity

A graph of the time trend of life expectancy in Western European countries such as France, England and Wales, and Sweden displays an S-shaped curve resembling the topographical contours of a logistic process but with a rather flat curvature. By contrast, the graph of the time trend of life expectancy in countries such as Argentina, Chile, Colombia, Nicaragua, and Mexico, although still showing the logistic shape, displays a sharper curvature, with a steeper rise at the beginning and a somewhat less smooth flattening at the end. Superimposing the curves for the European and Latin American countries with an appropriate scale modification of the time axis[4] results in a gap between the curves that grows at the beginning and shrinks considerably at the end. This gap is a measure of the differential paces in gains in longevity in the two groups of countries (see Figure 1). If this comparison proved to be representative for

FIGURE 1 Comparison of the evolution of life expectancy in Argentina and Chile with that in England and Wales





SOURCES: Values of life expectancy for Argentina up to 1960 were taken from Somoza (1971). The estimate for 1970 was taken from Muller and Accinelli (1978). Values of life expectancy for Chile up to 1960 were taken from Arriaga (1968); the estimate for 1970 was taken from Pujol (1976). The values of life expectancy for England and Wales were taken from Keyfitz and Flieger (1968).

other Latin American countries, it would be revealing of a premature slowdown in the process of mortality decline there.

Recently there have been some noteworthy efforts aimed at showing that the process of mortality decline in Latin America may have hit serious obstacles. Muller and Accinelli (1978) have drawn attention to the loss in years of life expectancy in Argentina as a whole and in some of the most urbanized areas of the country, such as the city of Buenos Aires, in particular. The deterioration of health conditions in urban settings could be closely related to increasing social inequalities and/or real losses in the purchasing power of large portions of the population. Carvalho and Wood report similar findings for Brazil and the city of Sao Paulo; their findings, however, remain partial and require additional confirmation (Carvalho and Wood, 1978). Gwatkin (1980) has shown that the pace of change of mortality levels in developing countries has slowed down significantly during the 1960s and early 1970s.

Further evidence to support the hypothesis of a reversible and discontinuous process of mortality decline can be obtained by comparing the actual evolution of mortality in Latin America with the expected evolution had these countries followed the footsteps of Western Europe. For this purpose I have assembled a set of pairs of estimates of life expectancy for two points during the period 1960–75. The need for comparability makes the sample leaner than desired. In particular, there are no comparisons for the period before 1960.[5] According to our hypothesis, the rates of gains in life expectancy in Western Europe–North America and in Latin American countries at equivalent levels of life expectancy should display contrasting patterns, reflecting different processes of attainment of equivalent levels of life expectancy.

To carry out the comparison, the following steps were taken: (1) A logistic function was fitted to the values of life expectancy, $e_0(t)$, for three European countries: Sweden, France, and England and Wales. (2) Once the parameters of the logistic curve were estimated, the proportionate change in life expectancy that followed the attainment of any value of life expectancy could be calculated. This allowed the comparison of the actual value of proportionate changes in a Latin American country with the value that would be expected over an interval of time of equivalent length and at the same initial level of $e_0(t)$ if the logistic curve estimated for the European countries described the Latin American process. Since the latter turned out to be very close to each other, the average of the three fitted logistic curves was taken. The resulting expected proportionate gains per year were then subtracted from the observed ones. These appear in column 5 of Table 1. A lower absolute value for the difference implies a closer resemblance of the pattern of mortality decline followed by the Latin American and the Western European countries. Similarly, the more negative the difference, the slower the pace of the Latin American country's mortality decline as compared with the Western European decline. About half of the countries in the sample showed lower proportionate gains than expected. Among them are Argentina (with a net loss of life expectancy between 1960 and 1970) and Uruguay (in an almost stationary condition for more than ten

TABLE 1 Observed gains in life expectancy in Latin America and the difference between observed and expected proportionate gains

Country	Year	Observed $_{e_0}$	Year	Observed $_{e_0}$	Observed minus expected proportionate gains
Argentina (Ar)	1960	66.5	1970	65.7	−.72
Barbados (Ba)	1960	65.0	1970	68.4	−.09
Bolivia (Bo)	—	—	1975	98.7	—
Chile (Ch)	1960	57.1	1970	61.5	.02
Colombia (Co)	1964	58.8	1973	59.6	−.57
Costa Rica (CR)	1963	62.3	1973	69.3	.45
Cuba (Cu)	1960	64.0	1970	69.9	.28
Dominican Republic (DR)	1965	54.3	1975	58.9	.06
Ecuador (Ec)	1962	54.2	1974	59.7	.05
El Salvador (ES)	1961	54.5	1971	57.4	−.26
Guatemala (Gu)	1964	48.3	1971	52.8	.44
Honduras (Ho)	1961	42.3	1974	52.5	.86
Jamaica (Ja)	1960	64.6	1970	68.4	−.04
Mexico (Me)	1960	58.6	1970	60.9	−.34
Nicaragua (Ni)[a]	1963	53.2	1971	54.9	−.43
Panama (Pa)	1960	61.9	1970	64.9	−.20
Paraguay (Par)	—	—	1971	63.5	—
Peru (Pe)	1962	51.0	1972	55.1	−.04
Trinidad and Tobago (Tr)	1958	62.4	1973	69.4	.09
Uruguay (Ur)	1963	68.5	1975	69.0	−.51
Venezuela (Ve)	1961	62.9	1972	64.7	−.41

[a]The life expectancy for 1971 refers to the female population only. Hence the estimate of the deficit in gains is a conservative one.

SOURCES: Estimates of life expectancy for Latin American countries are based on adjusted life tables. The adjustment procedures and sources of death rates are described in detail in a longer version of this paper (Palloni, 1981). Estimates of life expectancy for England and Wales, France, and Sweden were taken from the historical summary presented by Keyfitz and Flieger (1968). The following periods were considered: England and Wales, 1861–1971; France, 1851–1972; Sweden, 1778–1973. The most recent estimates were taken from the published information in the *Demographic Yearbook* (United Nations, 1974). The procedure to fit the logistic curve was a modification of the Newton-Gauss method (see Palloni, 1981).

years), two of the most highly developed countries in Latin America, as well as Nicaragua, a country that ranks among the most backward.

In Table 2 the sample of countries has been cross-classified by level of life expectancy at the beginning of the period considered and by the magnitude of the difference between the observed and expected gains. A sluggish pace of mortality decline appears to be characteristic of those countries that initially had reached relatively high values of life expectancy (above 55 years). Countries that started at lower levels of life expectancy, however, appear as likely to experience lower as higher values in the difference between observed and expected gains. Thus, Honduras and Guatemala show a higher than expected pace of gain, whereas Nicaragua and Peru show poorer performances than expected.

These results conform to the expectation of an unequal distribution of differences between observed and expected gains, reflecting different stages of mortality decline in various countries. Some countries, having not yet felt the

TABLE 2 Cross-classification of Latin American countries by level of life expectancy around 1960 and subsequent proportionate gains (frequencies of countries appear in cells)

Level of life expectancy (around 1960)	Difference between observed and expected proportionate gains			
	−.50 to −.25	−.25 to .0	.0 to .25	.25 to .50
60+	3	3	2	1
55–60	1	1	1	
50–55		2	2	
<50		1	1	1

SOURCE: Table 1.

full consequences of exposure to new medical technology, should show positive values in the differences at relatively low levels of life expectancy. Some, having exhausted the effects of medical interventions within a certain level of socioeconomic development, should show negative values in the differences at relatively high levels of life expectancy. Finally, countries that have progressed relatively rapidly in social and economic transformation should show positive or no differences at relatively high levels of expectancy. The complete individualization of these trends and their association with measures of economic and social development would be necessary to confirm the hypothesis.

It is important to emphasize that the illustrative evidence presented above is weak in at least one sense: the comparison performed over the logistic curve only took into account two consecutive values of life expectancies (for periods around 1960 and 1970). A more thorough evaluation of the actual changes in the pace of mortality improvements would require at least one additional observation. Thus, although it appears that there is a slowdown in the rate of increase of life expectancy, new data for the 1970s may well show a return to the more rapid pace experienced during the 1950s. If so, the phenomenon detected during the 1960s could turn out to be a temporary lapse in an otherwise regular process of change. Obviously, however, the future recurrence of past trends would in no way reduce the need to explain the irregularity during the 1960s.

Patterns of mortality in infancy and childhood

The hypothesis is that the levels of infant and child mortality across Latin American countries are higher than would be expected given overall mortality levels. Technically, this implies that the pattern of Latin American mortality is characterized by a U-shaped curve with a much higher left arm than would be predicted given its height at other ages. If this turns out to be the case, it should not be taken only as a peculiar statistical feature but must be linked systematically to existing levels of living, geographic and climatic conditions, and viability of medical technology.

Quantification of the magnitude of mortality excesses

The first problem to be resolved before anything can be said about levels and patterns of infant and child mortality is one of measurement. The quality of vital statistics in Latin America is quite deficient, and only through systematic use of indirect techniques have we arrived at measures that, at this point, are in all likelihood the most accurate available (Palloni, 1981). The second problem is to select a standard pattern of mortality against which to evaluate the Latin American pattern. Ready to hand is the mortality pattern that reflects the Western European–North American mortality experience at various levels of life expectancy, embedded in the life tables constructed by Coale and Demeny and grouped together in the Model West. As an indicator of adult mortality I have selected e_{15}, the life expectancy at age 15, because it is relatively free of the influence of mortality risks in early and late childhood. For each Latin American country with available data, I calculated the values of ${}_1q_0$ and ${}_4q_1$—the probabilities of dying before age 1 and in the interval 1–5—that would be expected if the North American–Western European pattern prevailed. (These will be referred to as "adult-equivalent" levels of early mortality.) Table 3 compares observed and expected values. With only one exception (Trinidad and Tobago), the observed values of ${}_1q_0$ are higher than the expected ones. Similarly, with three exceptions (Argentina, Chile, and Trinidad and Tobago), the observed values of ${}_4q_1$ exceed the expected ones.

TABLE 3 Comparison of observed and expected values of ${}_1q_0$ and ${}_4q_1$ in Latin America

Country	Year	e_{15}	${}_1q_0$ (× 1000)		${}_4q_1$ (× 1000)	
			Observed	Expected	Observed	Expected
Argentina	1970	56.1	59.7	39.9	9.5	12.0
Barbados	1970	57.3	43.9	31.9	6.3	7.4
Bolivia	1975	49.8	163.0	91.9	81.3	43.5
Chile	1970	53.4	77.1	59.9	14.5	27.6
Colombia	1973	52.4	77.2	68.1	41.9	28.5
Costa Rica	1973	59.3	50.9	23.1	12.0	4.5
Cuba	1970	59.0	37.9	24.0	12.6	4.8
Dominican Republic	1975	54.6	100.9	50.7	46.6	18.2
Ecuador	1974	56.6	101.7	36.5	59.8	10.1
El Salvador	1971	55.3	105.4	45.6	75.6	15.3
Guatemala	1971	50.4	85.4	86.1	88.4	39.7
Honduras	1974	50.5	127.2	85.6	61.1	39.0
Jamaica	1970	57.3	34.9	31.9	NA	—
Mexico	1970	53.6	81.8	58.4	38.4	22.7
Nicaragua	1971	52.5	113.9	67.3	65.8	28.0
Panama	1970	56.5	57.0	37.2	32.8	10.5
Paraguay	1971	55.9	66.1	41.3	30.8	12.8
Peru	1972	54.5	140.5	51.5	82.9	18.7
Trinidad and Tobago	1973	54.3	31.4	53.0	7.9	19.5
Uruguay	1975	58.1	46.9	26.7	5.8	4.5
Venezuela	1972	57.2	51.1	32.5	22.1	7.8

SOURCES: Observed values of ${}_1q_0$ and ${}_4q_1$ are adjusted for underregistration. See Palloni (1981). The expected values for ${}_1q_0$ and ${}_4q_1$ were obtained using the average predicted value from a linear and logarithmic function fitted to the pairs e_{15}, ${}_1q_0$ and e_{15}, ${}_4q_1$ in Model West of the Coale/Demeny system.

Figure 2 displays a set of points formed by plotting the observed against the predicted values of ${}_1q_0$. (Ignore the arrows for a moment.) Points below the 45-degree line from the origin correspond to countries with observed values of infant mortality lower than predicted. There is a conspicuous clustering of countries in two groups. The first group, contained below a second line also of 45 degrees but with a displaced origin, has both lower observed infant mortality and lower excess mortality than the group clustered on or above this line. In fact, within this first group we find, on the average, an excess of about 15 deaths per thousand live births, whereas in the second group the excess is, on the average, about 50 deaths per thousand live births. In addition, countries in the second cluster show a slight tendency to experience more than proportional increases in ${}_1q_0$ for a unit increase in the adult-equivalent level of infant mortality (${}_1q_0$ predicted), whereas the points in the first cluster can be well described by a straight line with a slope approximately equal to one.

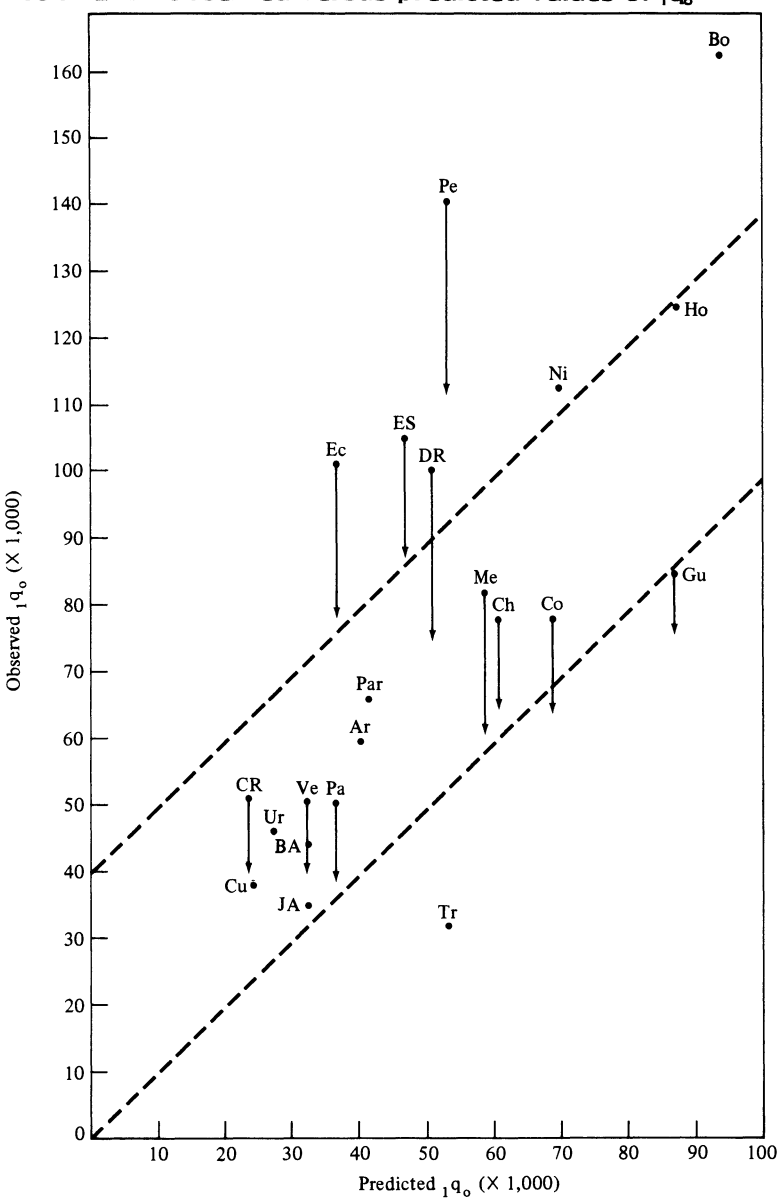
The corresponding set of points (not shown here but derivable from Table 3) formed by plotting the observed versus the predicted values of ${}_4q_1$ displays a much more irregular pattern. However, a clustering around the line with a slope of one and intercept of zero occurs for countries such as Cuba, Costa Rica, Uruguay, Barbados, Argentina, Chile, and Trinidad and Tobago, the last four showing lower than expected values of ${}_4q_1$. Higher than expected values appear characteristic of the rest of the countries. With the exception of Guatemala, the highest departures from the adult-equivalent level of ${}_4q_1$ correspond to those countries with equally high departures from the adult-equivalent level of ${}_1q_0$.[6]

Although the results obtained in Table 3 may be construed as supporting evidence for the working hypothesis on the age pattern of mortality, they could also be produced by the operation of factors completely unrelated to the actual quantum and tempo of mortality decline. First, consider measurement error. Any discrepancies between the accuracy of measurement of infant and early child mortality, on the one hand, and of adult mortality on the other could yield spurious excesses. Of potential relevance are the errors associated with the measurement of mortality at older ages. Either the existence of extensive overreporting of the ages at death or the prevalence of higher underregistration in the older than in the middle-age segments would produce too high values of life expectancy at age 15 and thus would impart an upward bias to the estimated excesses. In order to check the sensitivity of the measurement of excess mortality to changes in the indicator of adult mortality, I recalculated the predicted values of ${}_1q_0$ and ${}_4q_1$, utilizing as a measure of adult mortality the partial life expectancy between ages 15 and 60. This quantity is defined as:

$$\tilde{e}_{15} = (T_{15} - T_{60}) / l_{15}$$

where T_{15} and T_{60} represent the stationary population above ages 15 and 60 respectively and l_{15} is the number of survivors at exact age 15. When the recalculated excesses of mortality are plotted against the original ones, the

FIGURE 2 Observed versus predicted values of $_1q_o$



SOURCE: See Table 3.

correspondence although not perfect is quite close. The clustering of countries becomes more marked in the case of ${}_1q_0$, although it is attenuated in the case of ${}_4q_1$. All of this indicates that using the measure of excess mortality based on e_{15} provides a relatively conservative picture of the excess in the interval 0–1, particularly for countries with high levels of infant mortality (the exceptions being Peru and Nicaragua), and a slightly exaggerated picture of the excess in the interval 1–5, particularly for countries with high levels of early childhood mortality (the exception being Honduras). In the remainder of the paper, the original excesses are used since they provide a more appealing measure of inequalities of death risks in infancy, early childhood, and adulthood than those based on the partial life expectancies at age 15 ($\tilde{e}_{15}$).

A second set of factors that could affect the measure of excess mortality and its distribution by country is related to the differential distribution of births by parity. It is well known that infant mortality increases among high-parity births. The curve of mortality by parity is almost J-shaped, with a slight upturn at the lowest parity (Puffer and Serrano, 1973).[7] Given the same adult mortality levels and even the same parity-specific risks of mortality, a society with a distribution of births by parity with a higher mean will tend to show higher levels of infant mortality. In order to remove the effects of differential parity distribution, an indirect standardization procedure was tried on countries with the requisite data. The arrows drawn from the points plotted in Figure 2 measure the magnitude of the resulting reductions in ${}_1q_0$.[8] It is obvious that changes in reproductive behavior have substantial effects, even if no changes in the level of the parity-specific risks take place. However, it is also evident that the grouping of countries according to the excess infant mortality has barely been affected. Admittedly, those countries with preexisting low values in the differences between observed and predicted fall very close to the line representing no excess in infant mortality. Yet the countries of the second group, even if showing reductions, maintain their character as a separate cluster. Thus, there are important additional factors accounting for the level of excesses and the persistent clustering of countries.[9]

Excess mortality and the structure of causes of deaths

According to the second working hypothesis, infant and child mortality reflects the disproportionate disadvantages to which young children are exposed under conditions of slow socioeconomic development. This would suggest a direct relation between such excesses and the levels of mortality attributed to causes of death whose incidence is most vulnerable to changing socioeconomic conditions. Ideally, the overall level of mortality—given a known age pattern—ought to be sufficient to predict the incidence of each cause of death in a particular age segment. This will not occur, however, if the risks of death due to some causes are, within the lower age segment, disproportionately increased due to lack of nutrition, higher incidence of contact with disease agents, inade-

quate preventive care, and so on. However, the resulting excess in mortality should be closely related to the level of mortality attributed to those causes.

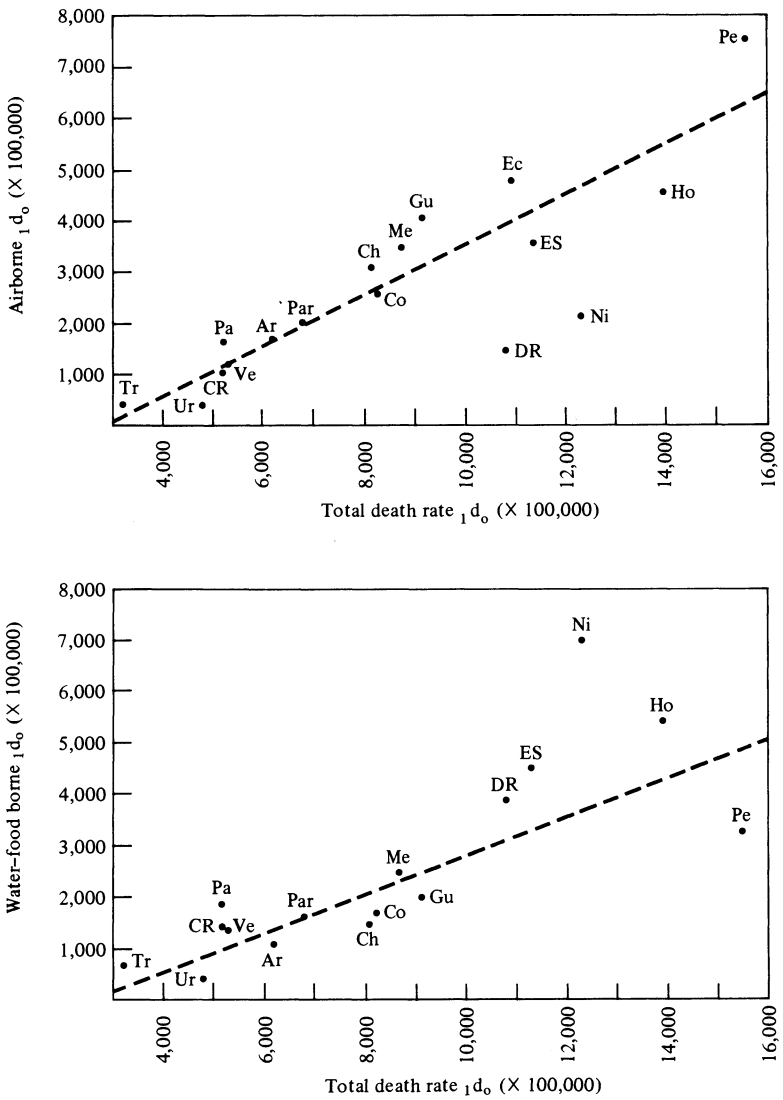
Another corollary directly derived from the above hypothesis is that the pattern of causes of death in countries showing the highest excesses should be significantly different from the pattern of causes of death in those countries experiencing stronger similarities between the observed levels of ${}_1q_0$ and ${}_4q_1$ and the adult-equivalent values of ${}_1q_0$ and ${}_4q_1$. In particular, the former should experience a disproportionate incidence of deaths attributable to factors reflecting insufficient improvements in standards of living.

I will deal with these two consequences of the hypothesis in reverse order. Notice that their verification depends on being able to estimate the structure of causes of death. Since Latin American data on causes of death are somewhat distorted by the excessively high proportion of deaths attributed to a residual ("other and unknown") category, I undertook a correction. Adjustment factors were applied to bring the cause-specific mortality rates, ${}_1d_0^i$ and ${}_4d_0^i$, in line with the total mortality rates d_0 and d.[10] Finally, the causes were grouped into aggregates distinguished by the mode of transmission or production of the disease: water and food borne, airborne, and vector borne. Other causes utilized were accident, avitaminosis, disease of infancy, and "all others." Within the first group, diarrhea is the most important cause of death, accounting for about 85 percent of the total water-food borne. Within the second group, the complex influenza-pneumonia-bronchitis (IPB) is dominant, accounting for almost 80 percent of the total airborne diseases. The incidence of diarrhea and IPB is, according to all available evidence, the most vulnerable to changing standards of living. Consequently the corresponding cause-specific death rates will be taken as the focus of analysis (Puffer and Serrano, 1973; Scrimshaw et al., 1968; Gordon et al., 1964; Greene, 1977; Winslow, 1944).

Outlying cause of death structures Typically, the levels of mortality in a particular age segment should be closely associated with the levels of mortality attributable to the various causes of death operating in that age segment. The relation could adopt a variety of forms, but more often than not a simple linear function captures its main features fairly well. In Figures 3 and 4 the death rates corresponding to airborne and water-food-borne diseases are plotted against the total death rates in the age intervals 0–1 and 1–5. In order to minimize the "pulling" effects of outliers from an underlying regular relation, the straight lines were fitted using a resistant technique developed by Tukey (Mosteller and Tukey, 1977).[11] A glance at Figures 3 and 4 immediately reveals that the countries forming the clusters discovered in Figure 2 have distinctive behaviors.[12] The Dominican Republic, El Salvador, Honduras, and Nicaragua show excessively high levels of mortality due to water-food-borne diseases and relatively low levels of mortality due to airborne diseases, while Peru, Ecuador, and Guatemala show the opposite pattern. The former group of countries is located at low altitudes and in tropical areas, while large portions of the population of the second group of countries are located at high altitudes and semitropical areas. Whereas the former areas are more conducive to the

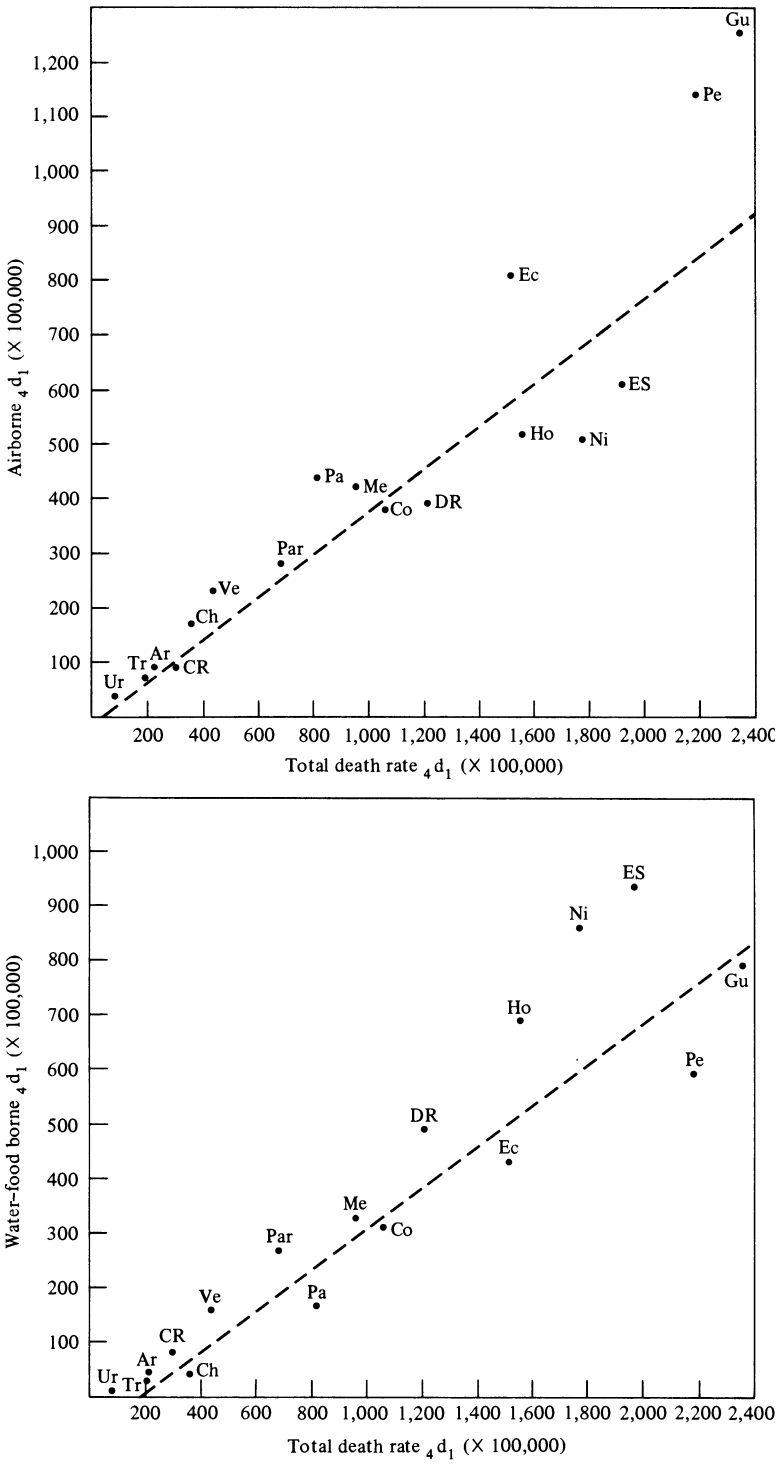
preservation and transmission of enteritis, bacillary dysentery, bacterial diarrhea, typhoid, and other diseases of the digestive system, the latter are characterized by climatic and geophysical conditions that favor the transmission of infections of the upper respiratory tract. However, one should be cautious in drawing conclusions from this evidence, since it is difficult to rule out the

FIGURE 3 Relation between the total death rate and the death rates for airborne and water-food-borne diseases in the age group 0–1



SOURCE: Palloni (1981).

FIGURE 4 Relation between the total death rate and the death rate for airborne and water-food-borne diseases in the age group 1–5



SOURCE: Palloni (1981).

possibility that different practices of death registration by cause could contribute at least partially to the formation of the distinctive clustering.

Contribution of some causes of death to excess infant and child mortality Although the evidence presented above is suggestive of the validity of the hypothesis and, in addition, helps to explain the conspicuous clustering discovered in Table 3 and Figure 2, it does not produce estimates of the contribution to the observed excess of each cause of death. These can be derived fairly easily from the relation between excess mortality in one age segment and levels of mortality due to certain causes of death. For example, the partial regression coefficient of excess mortality on the central death rate (number of deaths divided by number of person-years lived) for water-food-borne diseases, keeping constant the adult-equivalent mortality level, should indicate the amount of increase in the excess given an increase in the central death rate for water-food-borne diseases. Ideally it should be close to one. However, lack of independency among the various causes of death could increase the value above one; and indeed, the results of ordinary least squares regressions for age groups 0–1 and 1–5, using as independent variables the central death rates for W/F/A (water-food and airborne diseases taken together) and the adult-equivalent mortality level, are indicative of spillover effects, since the regression coefficient of W/F/A borne disease is larger than one. Furthermore, these effects seem to be more significant in the oldest age segment, as shown by a correspondingly larger regression coefficient of W/F/A borne diseases.[13] Thus an improvement in health conditions (not affecting the levels of adult mortality) leading to a reduction in diarrheal diseases equivalent to one death per person at risk is associated with a decrease in the level of excess mortality of about 1.6 deaths per person at risk in the age group 1–5 and of about 1.08 in the age group 0–1.

In summary, the brief examination of some outlying structures of causes of death and the estimation of the influence of predominant diseases reveal that in all likelihood the country heterogeneity in the levels of excess infant and child mortality is due to the disproportionate weight of the complex of water-food-airborne diseases in some countries. In turn, this suggests the existence of widespread deleterious effects that can only emerge under conditions of unsatisfactory levels of socioeconomic development and the generalized inability of the population to obtain adequate medical care facilities and services.

To recapitulate: the two implications of the hypothesis have been shown to be consistent with the available evidence. Not only do Latin American countries show an excess of infant and child mortality, but, more importantly, the levels of such excess appear to be related to a disproportionate contribution of the complex of water-food-airborne diseases. This complex is less responsive to the introduction of isolated medical interventions and depends heavily on standards of living, sustained public health interventions, and levels of information and health care practices of the population.

The role of socioeconomic conditions

In previous sections I have tried to draw attention to the importance of socioeconomic development in the current phase of mortality decline in Latin America. This was done indirectly by studying the patterns of gains in life expectancy and by examining the peculiarities in the relation between mortality at young and older ages. Evidently, the sharp differentials in socioeconomic conditions are partly responsible for the discrepancies in the patterns of mortality in Latin America. But how much can be accounted for by variability in socioeconomic conditions? Which are the most important factors? What changes ought to occur for the excess infant and childhood mortality to diminish or disappear?

This section analyzes the responsiveness of mortality risks in the age intervals 0–1, 1–5, 0–5 to a set of indicators of socioeconomic conditions. In each age segment considered, the death rate was partitioned into cause-specific components each of which was assumed to be related to socioeconomic conditions through a production function with the cause-specific death rates as outputs. Through the derivation of appropriate weights, it was possible to estimate for each country the proportionate change in the age-specific death rate that could be attributable to changes in the inputs, or socioeconomic conditions (Palloni, 1981).

Following the lead of other researchers (Chao, 1979; Preston, 1980; Caldwell, 1980; Behm, 1979; Rao, 1970; Puffer and Serrano, 1973), I have selected as best indicators of socioeconomic development a measure of income per capita, the proportion of the adult population that is illiterate, and, finally, a measure of income inequality. Other indicators were considered, but their actual performance did not justify their inclusion.

Table 4 displays income and education elasticity of each age-specific death rate. These elasticities measure the percent change in the age-specific death rate produced by a 1 percent change in the measure of education or income. The most remarkable feature of this table is the disproportionate influence of the measure of education on mortality. Its importance is notably higher in the age group 1–5 than in the age group 0–1, in which income and education have comparable influence. The strength of the responsiveness of mortality in the age interval 1–5 to the measure of education simultaneously reflects the very high influence that the latter has on variation in death rates attributed to diarrhea and influenza-pneumonia-bronchitis (about 90 percent of the variance is explained by education alone) and the inordinately large contribution of these causes of death to the overall death rate in the age group. A second feature of Table 4 also deserves consideration: it is among the countries that were shown to have high levels of excess mortality at 0–1 and 1–5 that the education elasticity of mortality reaches the highest values. As before, this is a result of the predominance of the water-food-airborne diseases in those countries. Countries with low or no excess appear to be affected much less by changes in education and slightly more by changes in income. Finally, the first

two columns of the table reveal quite conclusively an additional, though related, regularity: the responsiveness of death rates in the age group 0–5 to education is invariably higher precisely among the countries with the most deviant pattern of causes of death. Thus, the Dominican Republic, Ecuador, El Salvador, Honduras, Guatemala, Nicaragua, and Peru would experience the highest reductions in mortality if the degree of illiteracy were to decrease (or if the factors for which illiteracy serves as a proxy were to change in a favorable direction).

The relevance of education in the determination of levels of mortality has been widely discussed in the literature (Behm, 1979; Rao, 1970; Chao, 1979; Preston, 1980; Caldwell, 1980). It has been assumed that, at least at the individual level, the degree of education of the mother greatly influences the knowledge at her disposition to take care of the health of infants and children. Also it is believed that higher education promotes awareness and use of available health care services. Recently Caldwell has argued that educated mothers exercise more authority within the extended family and are more likely to seek "modern" health care for infants and children than to follow traditional norms and practices (Caldwell, 1980). Although all these factors may be operative, it is difficult to believe that they are the only ones reflected in a measure of aggregate education. The extent of illiteracy in a society reflects not only the limitations of individuals but, more importantly, the capacity of a system to organize and mobilize to fulfill societal necessities. From this point of view the proportion illiterate in a population is less an indication of the fraction of mothers with inadequate knowledge to treat and feed a sick child or to challenge the

TABLE 4 Estimated elasticities in 0–1, 1–5, and 0–5 age groups for income and education

	0–5		0–1		1–5	
Country	Income per capita	Adult illiteracy	Income per capita	Adult illiteracy	Income per capita	Adult illiteracy
Argentina	−.20	.18	−.21	.08	−.11	.90
Chile	−.16	.24	−.17	.16	−.07	.85
Colombia	−.16	.46	−.19	.16	−.11	1.08
Costa Rica	−.17	.28	−.18	.15	−.10	.99
Dominican Republic	−.14	.42	−.16	.21	−.08	1.24
Ecuador	−.11	.55	−.15	.26	−.04	1.17
El Salvador	−.12	.68	−.16	.31	−.05	1.32
Guatemala	−.10	.74	−.15	.25	−.03	1.30
Honduras	−.12	.62	−.15	.31	−.06	1.26
Mexico	−.11	.53	−.13	.28	−.05	1.19
Nicaragua	−.14	.65	−.17	.38	−.05	1.35
Panama	−.14	.45	−.18	.11	−.07	1.09
Paraguay	−.20	.41	−.24	.10	−.08	1.19
Peru	−.13	.53	−.17	.25	−.05	1.19
Trinidad and Tobago	−.15	.23	−.16	.11	−.12	.87
Uruguay	−.23	.01	−.24	−.08	−.17	.70
Venezuela	−.14	.43	−.16	.18	−.07	1.13

NOTE: Regressions of cause-age-specific death rates on socioeconomic indicators. See Palloni (1981) for a more thorough description of the model specification.

authority of elders than a reflection of the degree of social and political maturity of the system above and beyond the amounts of wealth at its disposal and the degree of equality of its distribution.

To illustrate this interpretation of the indicator, I have borrowed some evidence recently gathered in Latin America by H. Behm and collaborators (Behm, 1979). They estimated the probabilities of dying before age 2 by education of the mother. These results are reproduced in Table 5. Two features of this table are worth mentioning. First, the sequence of q(2) slopes downward as education increases. This is to be expected under the "individual effects" interpretation of education. Second, the magnitude of q(2) for equivalent educational groups and the within-country variation differ substantially across countries. An extreme case appears to be Cuba, a country in which there are virtually no mortality differentials by educational group. This appears to be a result of that country's effort at organizing and mobilizing to provide services to the population regardless of the existing individual demand for them (Diaz-Briquets, 1978).

Many intriguing features of Table 5 call into question the suggestion that individual education is the most powerful predictor of mortality differentials. The last column of the table contains the average difference in q(2) resulting from comparisons of adjacent educational categories. Whereas the improvements in mortality are clearly related to mother's education, the amount of improvement differs widely by country. Thus, if on average the passage to a higher educational group implies for a Chilean woman a reduction of only

TABLE 5 Probabilities of dying during the first two years of life ($_2q_0$), by education of mother

Country	Total	Years of education of mother					Average slope[e]
		0	1–3	4–6	7–9	10+	
Cuba[a]	41	46	45	34	29		5.67
Paraguay	75	104	80	61	45	27	19.25
Costa Rica	81	125	98	70	51	33	23.00
Colombia[b]	88	126	95	63	42	32	23.50
Chile	91	131	108	92	66	46	21.25
Dominican Republic	123	172	130	106	81	54	29.50
Ecuador	127	176	134	101	61	46	35.00
Honduras	140	171	129	99	60	35	34.00
El Salvador	145	158	142	111	58	30	32.00
Guatemala	149	169	135	85	58	44	31.25
Nicaragua	149	168	142	115	73	48	30.00
Peru[c]	169	207	136	102	77	70	34.30
Bolivia	202	245	209	176	110[d]		45.00
Argentina	58	96	75	59	39	26	16.80

[a]Provisional figures based on the *Encuesta Nacional de Ingresos y Egresos de la Poblacion, 1974*. Years of education of mother are 0, 1–5, 6, and 7.
[b]Years of education of mother are 0, 1–3, 4–5, 6–8, and 9+.
[c]Years of education of mother are 0–2, 3–4, 5, 6–9, and 10+.
[d]Corresponds to 7+.
[e]Calculated by averaging the differences in $_2q_0$ between successive educational categories.
SOURCES: Behm and Primante (1978).

about 21.25 deaths per thousand births, the same passage implies for a Bolivian woman a reduction that is twice as large. Furthermore, a Chilean woman in the middle education category can expect just under one half of the number of child deaths that a Bolivian woman in the same education category can expect. This variability must be explained by factors other than individual education. Although a differential distribution of other relevant individual variables such as income and occupation or even the lack of cross-country equivalence of educational categories in terms of educational content might be responsible for these contrasts, they are more likely to be related to contextual characteristics. If one pursues the idea that the degree of illiteracy in a society is more a reflection of the degree of advancement of the system than of individuals, it is possible to interpret some of the striking features of Table 5. In particular, contextual and community characteristics for which illiteracy serves as a proxy ought to explain not only the cross-country differentials in mortality at the same educational level of the mother but also the cross-country heterogeneity in the degree of inequality of the death risks across educational level of the mother.

Simple regression and analysis of covariance applied to the data in Table 5 reveal that about 86 percent of the variability in the risks of dying before age 2 can be accounted for by the mother's level of education and the aggregate measure of education (proportion illiterate). In addition, the analysis indicates that a 1 percent change in the measure of aggregate education implies a reduction of 1.2 deaths per thousand children born to mothers in the average education category but over 5.6 deaths per thousand in the lowest education category. Finally, the inequality in the mortality differentials by mother's education would be reduced by about 4.5 deaths per thousand births on average if the proportion illiterate decreased by 1 percent (or if the factors for which illiteracy serves as a proxy were changed in a favorable direction and by a magnitude equivalent to a 1 percent change in illiteracy). In the rather unlikely event that all mothers reached the highest educational category without there being changes in the social context, the differentials in mortality would persist. Thus, in the Dominican Republic the value of q(2) would be approximately 81 deaths per thousand births, whereas in Argentina it would be 26 deaths per thousand births. The difference between the two countries would be reduced by about 2.4 deaths per thousand births if the proportion of illiterate women in the Dominican Republic decreased by 1 percent (or if equiproportional changes in the factors for which illiteracy serves as a proxy took place).

In summary, the effects of mother's education on child mortality are contingent on social setting. A disadvantageous setting is more severely felt at the lowest levels of education, but it operates at all educational levels. Conditions leading to reductions in illiteracy will not only result in favorable changes in the proportional distribution of mothers by educational group but will also generate a reduction in within-country heterogeneity and cross-country inequalities. Theoretically, the education elasticities displayed in Table 4 capture the combination of these effects. To what extent do these effects eliminate

outlying cause of death structures? To what extent do they suppress the clustering of countries with high excesses in infant and child mortality? Simple calculations show that changes of 10 to 20 percent in the rate of illiteracy (or equiproportional changes in the factors for which illiteracy serves as a proxy) can reduce the observed excess mortality by as much as 60 percent, virtually eliminate outlying cause of death structures, and all but destroy the clustering of countries observed in Figure 2. The importance of these potential changes is enhanced by the fact that, in the countries with highest potential for change (e.g., those with the highest excess mortality), between 30 and 50 percent of the adult populations are illiterate.

Conclusion

Regardless of how rapid the changes in Latin American mortality have been in the past, they remain painfully insufficient to close the gap with the more developed countries. Undoubtedly, if a slowdown in mortality decline were to become a generalized phenomenon, the closing of the gap hoped for a decade or so ago would become a chimera. Unusual, or at any rate unexpected, patterns of infant and child mortality are quite widespread. They can be traced in large part to excessively high mortality attributable to a complex of diseases associated with diarrhea and infections of the respiratory tract. This occurs despite the fact that the majority of Latin American countries have already been widely exposed to medical technology directed at controlling infectious diseases.

Dramatic reduction in mortality need not be a thing of the past. The influence of socioeconomic factors on mortality at various ages is paramount in any prognosis for further reductions. Social and economic factors related to the rate of adult literacy appear to be by far the most relevant; they are surely among those to be manipulated if any further improvements are to be realized.

During the next few years, new breakthroughs in medical technology may make possible new changes. However, to place hopes solely in this possibility would be unfortunate and unnecessary when the slowdown in mortality decline and the excessive infant and child mortality can be countered by enhancing the capacities of all social groups for utilizing the breakthroughs already made.

Notes

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1 Here as elsewhere in the paper I have used the European–North American experience as a counterpart for comparison. This choice reflects pragmatic considerations of data availability and the desire to maintain continuity

with past practices. It is not based on the belief that mortality changes in all developed countries lacked significant variability. Among countries of Western Europe and North America, there were variations in timing and pace, although the general profile of mortality changes was similar. Second, developed countries of Southern and Eastern Europe experienced rather different mortality transitions, with accelerations and slowdowns that resembled those observed in Latin America but were less marked.

2 Notice that the technologies available to control the incidence of diarrhea include such seemingly trivial things as feeding and caring practices followed by a population. It is well known, for instance, that discontinuing the intake of food when a child is ill aggravates his condition. Changes in such practices, however, may be harder to bring about than temporary control of the reproductive cycle of the malaria-carrying mosquito.

3 An additional factor certainly affects the shape of the pattern of mortality in the expected manner. In most developing countries, contact with modernized society has favored the rapid spread of bottle feeding to replace breastfeeding. Shortening of the breastfeeding interval may have catastrophic consequences not only because the products being marketed are less nutritious than human milk but also because: (a) natural immunities contained in human milk are not replaced; (b) the preparations are supplied to the infant under less than ideal conditions, e.g., contaminated water supply; and (c) the preparations are rarely if ever complemented with an adequate intake of solids.

4 The scale of the time axis is transformed from calendar year into fractions of time elapsed since the beginning of the process of mortality decline. The date of initiation of mortality decline is set at the calendar year at which life expectancy reached a value of 40. The end point of the interval is set at the year at which the highest value of life expectancy for the developing country occurs.

5 The estimates of life expectancy used in this paper were obtained from corrected official death registration data. An effort was made to ensure comparability across time of the estimated values to avoid the biases introduced by changing (presumably improving) quality of vital statistics or caused by differential robustness of methods utilized to correct the observed figures. For a thorough review of sources of data and correction procedures see a longer version of this paper (Palloni, 1981).

6 The case of Guatemala appears as an exception throughout this analysis. Although errors in the estimates can be responsible for this behavior, it is more likely that certain social practices and customs are producing lower excesses in the interval 0–1 and higher excesses in the interval 1–5. In particular, the population of Guatemala seems to have maintained a pattern of breastfeeding of longer duration than the population of other Latin American countries. This can result in the observed dissimilarity of the patterns of infant and early child mortality. However, given the lack of adequate information to substantiate the point, this explanation must remain as a simple suggestion. See United Nations (1979).

7 The factors explaining this regularity are several. First, women of higher parity are likely to present more precarious conditions for pregnancy and delivery, with predictable effects on the child (low birth weight, birth complications). Second, and more important, mothers of higher parity are less likely to breastfeed for long periods of time, either because of age-related deterioration or because of complications associated with and accumulated from previous pregnancies. Finally, a child born within an already too numerous group will tend to compete more for attention and be exposed to more hazards than a first or second child. Any of these factors may generate conditions having a negative impact on the health status of the child.

8 To remove the effects of differential parity distribution of births from Figure 2 is hardly an easy task. However, by relying on certain simplifying assumptions, one can arrive at approximate estimates of the excess infant mortality that would exist if all countries showed a "favorable" parity distribution. This is a typical problem of standardization for

$$ {}_1q_o = \sum_{i=1}^{n} b_i \, {}_1q_o^i $$

where b_i is the proportion of all births of parity i and ${}_1q_i$ is the i-th parity-specific infant mortality rate. I assumed that the parity pattern of ${}_1q_o^i$ was the same for all countries and only their level was allowed to vary. In particular,

the Chilean pattern of ${}_1q_0^i$ was adopted as a standard. It was also assumed that the most favorable parity distribution that one of these countries could experience was that observed in the United States around 1970. This was then used in conjunction with the estimated sequence ${}_1q_0^i$ to produce values of ${}_1q_0$.

9 Unlike the case of ${}_1q_0$ it is not possible to remove the variation in ${}_4q_1$ which is due to the heterogeneity of birth distribution by parity, since the required data for carrying out the computations are lacking. But its effects ought to be of even less import than in the case of ${}_1q_0$.

10 For details on the correction procedures, see Palloni (1981). For lack of a more suitable alternative, it was assumed that the overall level of completeness of death registration was invariant across various causes of deaths.

11 The estimated parameters of the regression equations (biweight least squares) are:

Age group and cause	Intercept	Slope
0–1		
Airborne	−1061.1	.39
Water-food borne	−1359.0	.50
1–5		
Airborne	− 28.3	.40
Water-food borne	− 59.7	.37

12 In order to summarize the pattern of deviations, a table was constructed classifying the countries sharing high deviations from the fitted lines to observations in the age groups 0–1 and 1–5:

	Cause of death and age group	
Countries	Water-food borne	Airborne
	0–1	
Group A	+ deviations 2.2	− deviations 2.9
Group B	− deviations 1.0	+ deviations 1.7
	1–5	
Group A	+ deviations 3.4	− deviations .96
Group B	− deviations 1.3	+ deviations 3.2

+ = positive − = negative

Group A includes Dominican Republic, El Salvador, Honduras, and Nicaragua;
Group B includes Ecuador, Peru, and Guatemala.
The numbers in the cells correspond to the value of the ratios of the median residual (in absolute value) in each subgroup of countries to the median of the residuals (in absolute value) of all countries.
SOURCES: Cause-specific death rates were calculated utilizing information contained in magnetic tapes created by the World Health Organization (WHO, 1973). The following is the set of ICD codes corresponding to the 8th Revision of the A list that are included in the groupings utilized for the analysis carried out in this paper: Airborne: A15, A16, A17, A24, A25, A89–A96; Waterborne: A1–A5, A7–A10, A28; Vector Borne: A30, A31; Diseases of Infancy: A131–A135; Accidents: A138–A150; Avitaminosis: A75; Ill-defined and unknown: A136, A137; Diarrhea: A5; Influenza, Pneumonia, Bronchitis: A90–A93.

13 Water-food-airborne diseases were pooled together in order to avoid the problems that could be caused by the diversity in the patterns of causes of death that were detected in Figures 2, 3, and 4. Results of OLS regressions to estimate equations of the form: Excesses$=\alpha+\beta\ d_x^i + \gamma\hat{d}_x$ are as follows:

	Age group	
	0–1	1–5
R^2	.99	.94
$\hat{\alpha}$	307.4	132.3
$\hat{\beta}$	1.08*	1.16*
$\hat{\gamma}$	−1.04*	−1.04*

*significant at $p < .01$

where

d_x^i = central death rate associated with combination of water-food and airborne diseases ,

$\hat{d}_x$ = expected value of central death rate given e_{15} ,

and the values of excesses operating as dependent variables were obtained by converting the values of ${}_nq_x$ (observed) and ${}_n\hat{q}_x$ (expected) presented in Table 3 into central death rates and subtracting the latter from the former.

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