Social Biology

ISSN: 0037-766X (Print) (Online) Journal homepage: http://www.tandfonline.com/loi/hsbi19

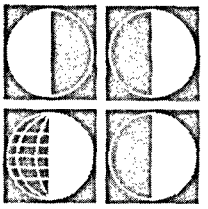
Mortality decline in Latin America: Changes in the structure of causes of deaths, 1950–1975

Alberto Palloni & Randy Wyrick

To cite this article: Alberto Palloni & Randy Wyrick (1981) Mortality decline in Latin America: Changes in the structure of causes of deaths, 1950–1975, Social Biology, 28:3-4, 187-216, DOI: 10.1080/19485565.1981.9988458

To link to this article: http://dx.doi.org/10.1080/19485565.1981.9988458

Mortality Decline in Latin America: Changes in the Structure of Causes of Deaths, 1950–1975



Alberto Palloni and Randy Wyrick

Center for Demography and Ecology, University of Wisconsin, Madison, Wisconsin; and Population Research Center, University of Texas, Austin, Texas

ABSTRACT: In this paper we study some of the characteristics of the process of mortality decline in Latin America during the years 1955 and 1973. This is done by analyzing the levels of overall mortality and cause-specific mortality and their changes in the period indicated, and by examining the relation between levels of mortality and their changes and a set of endogenous variables measuring socioeconomic conditions. This analysis is intended to measure in a somewhat crude manner the effects of diffusion of medical technology vis à vis the effects of changes in socioeconomic conditions.

It is well known that mortality decline in Latin America proceeded at a faster pace than in developed countries (Stolnitz, 1955, 1965; Arriaga and Davis, 1969; Arriaga, 1970). During the decade following the end of World War II, life expectancy increased at a staggering rate of about 0.9 years per year in countries not having experienced previous changes and of about 0.7 years per year in countries in which mortality decline had started somewhat earlier (Arriaga and Davis, 1970; United Nations, 1963, 1973; Arriaga, 1968). In the past five to ten years, however, there have been unmistakable signs that the process has slowed down considerably and, in some cases, stopped altogether (Gwatkin, 1980; Palloni, 1981). The factors explaining either the initial stages of the process or its apparent current status are only poorly understood due to the rather general character of most studies which, although providing essential understanding of the main features of the process, neglected to consider its details and complexities. In part, this can be explained by a well-entrenched reluctance to deal with data of doubtful quality and to speculate in the absence of any information whatsoever.

In this paper, we attempt to analyze the process of mortality decline in Latin America by examining the relative contribution of various causes of deaths to the resulting changes in levels of mortality. In addition, we try to explain both the variation in the overall levels and the changes in the structure of causes of deaths by reference to socioeconomic factors which directly or indirectly affect the health conditions of the population.

The first section of the paper presents a necessarily brief discussion of the sources and quality of the data utilized and the techniques that were applied to detect and correct errors. The second section deals with the general characteristics of mortality decline in Latin America during the period 1950–1973. Total and cause-specific standardized death rates are examined in an attempt to uncover the contribution of changes in the incidence of some diseases to the rate of decline in the initial stages of the process. The third and final section deals with the relationship between socioeco-

nomic factors and total as well as cause-specific levels of mortality.

MATERIALS AND METHODS

The sources of data and the procedures utilized to detect and correct errors have been described in detail elsewhere (Palloni, 1979). Here we will only summarize the basic steps taken to construct the measures utilized throughout the paper.

(*a*) Data on deaths by age groups and causes on various Latin American countries were collected from regular publications of the World Health Organization. Only those countries for which information was available at least at one point during the fifties and at least once during the sixties were included. A detailed list appears in Table 1.

(*b*) Adult deaths (above age 5) were adjusted for completeness using techniques by Brass (1975) and Preston (1979). Deaths in infancy and childhood were adjusted using estimates of completeness derived from extensions of a technique originally formulated by Brass (Palloni, 1980; Feeney, 1980).

(c) Causes[1] of deaths were grouped into categories that permitted the comparability of the sixth, seventh, and eighth revisions of the International Classification of Diseases. In addition, the categories selected permit the establishment of contrasts with other studies in the area (Preston, 1976). Although some procedures have been suggested (Palloni, 1981; Leaderman, 1955), we did not correct for the size of the category of ill-defined causes. Wherever it was possible, statistical controls were utilized. Some experimentation with procedures that permit distributing the deaths classified in the ill-defined category among those well defined, suggested that lack of correction would, in general, be a conservative strategy because the biases would work against the hypotheses to be tested in this paper. A detailed list of the groups of causes of death utilized in the paper appears in Table 2.

(*d*) Death rates specific for five-year age groups were computed. In order to minimize the effects of irregularities produced by age heaping and age misreporting, the curves of the logarithms of the death rates were smoothed by using simple polynomials in the appropriate age segments. The resulting death rates were then used to compute total and cause-specific death rates. To eliminate the influence of age composition, we calculated standardized death rates. The selected standard population was a female stable population with a natural rate of increase equal to 0.01, a life ex-

TABLE 1

COUNTRIES AND YEARS FOR WHICH CAUSE OF DEATH DATA WERE AVAILABLE STARTING IN 1955 AND UP TO 1973

Uruguay...	1955,	1965,	1973	...	...
Chile.....	1930,	1940,	1955,	1964,	1973*
Colombia..	1953,	1955,	1960,	1964,	1972
Venezuela .	1955,	1960,	1964,	1973	...
Mexico	1951,	1956,	1960,	1964,	1973
El Salvador	1952,	1955,	1960,	1964,	1973
Dom. Republic.	1951,	1955,	1960,	1965,	1973
Guatemala .	1952,	1956,	1961,	1964	...
Panama ...	1953,	1958,	1960,	1964	...
Costa Rica .	1953,	1960,	1964,	1973	...
Paraguay ..	1955,	1973	...	...	...

*With the exception of Chile 1930 and 1940, all other data were obtained from World Health Organization, Statistical Yearbook (various issues). Data for Chile 1930 and 1940 were obtained from Preston et al., 1972.

[1]These, as other procedures of "redistribution" of deaths assigned to ill-defined causes, are based on assumptions that can seldom, if ever, be verified.

TABLE 2

GROUPS OF CAUSES OF DEATHS*

Name	B List	A List
Respiratory Tuberculosis	B1	A6
Other Infectious and Parasitic Diseases	B2-B15, B17	A1 to A44 except A5
Malaria	B16	. . .†
Neoplasms	B18–19	A45 to A61
Cardiovascular Diseases	B22; B24–29	A80 to A85‡
Influenza-Pneumonia-Bronchitis	B30–32	A90 to A93
Diarrhea, Gastritis, Enteritis	B36	A5§
Chronic Diseases	B20,33,37,38	A64,A98,A102,A106
Maternal Mortality	B40	A112 to A118
Diseases of Infancy	B42–44	A131 to A135
Motor Vehicle Accidents	BE47	AE138
Other Accidents and Violence	BE48–50	AE139 to AE150
Senility, Ill-defined, and Unknown	B45	A136,A137
Other	Residual	Residual

*The equivalences shown here are consistent in the 6th, 7th and 8th revisions of the International Classification of Diseases.

†In WHO publications for 1973, malaria does not appear separately by age. However, by 1973 malaria produced very few deaths. The crude death rate was used for separate analysis.

‡It does not include A85 and A86 since they were unavailable for most countries before 1958.

§A5 in 1973 does not include some diseases included in B36 before 1970. However, they are quite insignificant and do not produce serious distortions. For the age group 0–1, most diseases attributed to diarrhea were classified in categories corresponding to diseases of infancy when following practices established before the 8th revision. After this revision was enacted, diarrheal diseases were classified as diarrhea (one of the titles included in the category A5) regardless of the age of the deceased. Thus, the death rate due to diarrhea for the years after 1965 may be slightly inflated by comparison to what it would have been in the absence of any changes.

pectancy at birth equal to 65.0 years, and an age pattern of mortality corresponding to model West (Coale and Demeny, 1966). Naturally, other selections of standards were possible. In particular, we would have preferred to use a slightly younger population, with a higher rate of natural increase, to give more weight to the levels of mortality at the youngest ages. However, we opted for the selected standard in an effort to maintain strict comparability with other studies (Preston, 1976)[2].

[2]In an effort to assess the robustness of our results to the choice of the standard, we repeated some of the basic regression analyses to be presented later, but recalculating the standardized death rates inputing a younger standard. The *numerical values* of the results are, of course, different (change of scale in one of the measures). However, the direction of the changes, if anything, indicates that the analysis presented in this paper is somewhat conservative, i.e., our conclusions receive stronger support with the selection of a younger standard. This is a natural outcome of the fact that a disproportionately high fraction of all deaths occur before age ten. Selecting a younger standard attaches higher weights to these deaths and increases somewhat the variability of the resulting standardized death rates (SDR).

RESULTS

DESCRIPTION OF MORTALITY DECLINE: GENERAL FEATURES 1950–1973

A descriptive analysis of mortality decline by causes is affected not only by the peculiarities of the data already discussed but also by the selection of the cases and the decision about the indicators to be used. In the first place, it is important to realize that the tempo and processes of mortality decline differ from one country to another. Thus the cross section *at one point in time* includes

countries at very disparate stages in the evolution of mortality conditions. As a consequence, the underlying structure of causes of deaths will be far from homogeneous. By the same token, the aggregate of countries taken at two points in time will reflect different sections of a process regardless of whether or not the latter is the same for all countries included. As a consequence, the *changes* in the structure of causes of deaths will also turn out to be heterogeneous. Thus, any analysis carried out with a cross-section will produce *average* structures, trends, or changes. The latter's profile must not be construed as representing any particular country's past experience or prospective evolution unless there is independent information at the country level to verify it.

In the second place, it is necessary to decide on the measures of mortality to be used. Although traditionally the life expectancy of the population has been the preferred choice, its analysis becomes unnecessarily cumbersome if one wants to relate variations in life expectancy to changes in some causes of deaths. A less appealing, but less awkward, measure is the standardized death rate which breaks down additively by age and causes. The difficulty that this selection engenders is, of course, the rather arbitrary decision about the most appropriate standard (see Footnote 2). Nevertheless, since the life expectancy of a population and the corresponding standardized death rate are closely related (the relation varying with the standard used) it will be possible when necessary to translate one measure into the other.

Table 3 displays the levels in the standardized death rates (SDR) by causes and the standardized proportionate distribution of total deaths by causes (figures in parentheses) at the beginning and end of the period considered. The average annual decrease in SDR, in the interval 1950–1973, is about 0.40 with a maximum of 0.49 in El Salvador and a minimum of 0.21 in Uruguay. These figures correspond to per-year increases in SDR of about 0.97, 1.19 and 0.51 respectively. By and large, the most significant *absolute* decreases occur in those countries with the highest initial levels of mortality. These are also countries in which only minor improvements in mortality conditions had taken place before the earliest date indicated in the table. Since the initial available room for changes may affect the magnitude of the subsequent declines, it is important to examine the *relative* annual rate of decline. Its values range from a minimum of 1.5 per cent per year in the Dominican Republic to a maximum of 2.3 per cent per year. No clear relationship emerges here between the magnitude of the relative changes and initial levels of mortality (see Table 4).

The SDR by causes reveals some interesting but also some disappointing features. As for the latter, in both years and almost all countries (the exceptions being Chile, Uruguay, and Costa Rica), the highest share of the total SDR is associated with the residual category. At the beginning of the interval considered and among the well-defined diseases there is clear prevalence of infectious diseases (OI), respiratory diseases (influenza, pneumonia, and bronchitis) and diarrhea. At the end of the period, cardiovascular diseases and neoplasms invariably become the most prevalent diseases thus displacing the infectious diseases from this role. Respiratory diseases and diarrhea, however, remain

TABLE 3

STANDARDIZED DEATH RATES PER THOUSAND BY CAUSES FOR SELECTED LATIN AMERICAN COUNTRIES, 1950–1970

Country and Year	Total	RT	OI†	NEOP	CARD	IPB	DIA	CHR	MA	DI	ACC	AOU
Uruguay												
1955.....	12.22	0.32 (2.6)	0.33 (2.7)	2.34 (19.2)	3.78 (30.9)	0.63 (5.1)	0.30 (2.5)	0.52 (4.2)	0.04 (0.4)	0.70 (5.8)	0.70 (5.9)	2.50 (20.5)
1973.....	10.55	0.05 (0.5)	0.15 (1.4)	2.09 (19.9)	4.01 (38.1)	0.45 (4.3)	0.16 (1.5)	0.46 (4.3)	0.01 (0.1)	0.38 (3.5)	0.59 (5.6)	2.03 (19.2)
Chile												
1950.....	20.59	1.64 (8.0)	0.75 (1.4)	1.61 (7.8)	5.10 (24.8)	3.34 (16.3)	0.57 (2.7)	0.73 (3.5)	0.14 (0.6)	2.25 (10.9)	0.85 (4.0)	3.58 (17.4)
1973.....	12.61	0.29 (2.2)	0.22 (1.7)	1.93 (15.4)	3.02 (24.0)	1.37 (10.9)	0.29 (2.3)	0.83 (6.6)	0.04 (0.3)	0.48 (3.9)	1.28 (10.1)	2.85 (22.6)
Columbia												
1953.....	19.09	0.34 (2.0)	1.58 (8.3)	1.15 (6.0)	2.15 (11.3)	1.72 (9.0)	1.06 (5.6)	0.62 (3.3)	0.17 (0.9)	0.83 (4.3)	1.05 (5.4)	8.28 (43.5)
1972.....	12.20	0.22 (1.8)	0.48 (3.9)	1.29 (10.6)	3.54 (29.0)	1.10 (9.0)	0.61 (5.0)	0.47 (3.8)	0.07 (0.6)	0.28 (2.2)	0.97 (7.9)	3.14 (25.7)
Venezuela												
1955.....	15.46	0.55 (3.5)	0.64 (4.2)	1.32 (8.5)	2.40 (15.6)	0.56 (3.6)	0.69 (4.5)	0.68 (4.4)	0.07 (0.4)	0.49 (3.1)	0.71 (5.0)	7.35 (47.5)
1973.....	10.86	0.16 (1.5)	0.36 (3.3)	1.25 (11.5)	2.76 (25.4)	0.75 (6.9)	0.34 (3.1)	0.50 (4.6)	0.04 (0.3)	0.27 (2.4)	0.90 (8.3)	3.52 (32.4)
Mexico												
1951.....	22.53	0.59 (2.6)	2.80 (12.4)	0.71 (3.2)	2.00 (8.8)	3.34 (14.8)	3.24 (14.4)	1.08 (4.8)	0.14 (0.6)	0.83 (3.7)	0.64 (2.8)	7.16 (32.7)
1973.....	12.45	0.27 (2.1)	0.49 (3.9)	0.84 (6.8)	2.38 (19.1)	1.65 (13.2)	0.92 (7.4)	1.01 (8.1)	0.07 (0.6)	0.21 (1.7)	0.91 (8.2)	3.58 (28.8)
Salvador												
1952.....	21.51	0.48 (2.2)	2.35 (11.0)	0.53 (2.5)	1.01 (4.6)	1.16 (5.4)	3.35 (15.6)	0.35 (1.7)	0.13 (0.6)	0.49 (2.3)	1.13 (5.3)	10.50 (48.75)
1973.....	12.27	0.16 (1.3)	0.42 (3.4)	0.52 (4.2)	1.04 (8.5)	0.77 (6.3)	1.31 (10.7)	0.38 (3.1)	0.05 (0.4)	0.18 (1.5)	1.14 (9.2)	6.28 (51.2)
Domin. Repub.												
1951.....	20.90	1.38 (6.6)	3.59 (25.3)	1.02 (4.9)	1.85 (8.9)	1.38 (6.6)	1.53 (7.3)	0.47 (2.2)	0.15 (0.8)	0.70 (3.4)	0.53 (2.4)	8.28 (39.5)
1973.....	14.06	0.17 (1.2)	0.38 (2.7)	0.75 (5.3)	1.98 (14.1)	0.55 (3.9)	0.51 (3.6)	0.56 (4.0)	0.07 (0.5)	0.43 (3.0)	0.74 (5.2)	7.91 (56.3)
Guatemala												
1952.....	25.87	0.64 (2.4)	8.11 (31.4)	0.58 (2.3)	0.74 (2.8)	3.51 (13.6)	2.64 (10.2)	0.34 (1.3)	0.09 (0.4)	0.64 (2.5)	0.46 (1.7)	9.0 (3.50)
1964.....	18.66	0.41 (2.2)	3.08 (16.5)	0.64 (3.4)	1.18 (6.3)	2.88 (15.5)	2.24 (12.0)	0.33 (1.8)	0.11 (0.6)	0.96 (5.1)	0.62 (3.3)	6.2 (33.3)
Panama												
1953.....	16.05	0.64 (4.0)	1.06 (6.6)	1.30 (8.2)	3.16 (19.7)	1.34 (8.4)	0.49 (3.1)	0.57 (3.5)	0.15 (0.9)	0.34 (2.4)	0.78 (4.8)	6.18 (38.7)
1973.....	9.74	0.23 (2.3)	0.41 (4.2)	1.08 (11.1)	2.43 (25.1)	0.79 (8.1)	0.32 (3.3)	0.38 (3.8)	0.05 (0.4)	0.54 (5.5)	0.74 (7.7)	3.13 (32.1)
Costa Rica												
1953.....	16.09	0.37 (2.3)	1.38 (8.6)	2.03 (12.6)	2.71 (16.8)	1.81 (11.2)	1.19 (7.39)	0.57 (3.6)	0.09 (0.6)	0.71 (4.4)	0.54 (3.4)	4.69 (29.2)
1973.....	8.70	0.09 (1.0)	0.28 (3.2)	1.38 (16.0)	2.36 (27.2)	0.67 (7.7)	0.37 (4.3)	0.52 (6.0)	0.03 (0.3)	0.23 (2.7)	0.60 (7.0)	2.14 (24.5)

*In parentheses are the standardized causes of death distribution. All figures are based on corrected death rates. The correction proceeded by using the correction factors. RT = Respiratory Tuberculosis; OI = Other Infectious and Parasitic Diseases; NEOP = Neoplasms; CARD = Cardiovascular; IPB = Influenza, Pneumonia, Bronchitis; DIA = Diarrhea; CHR = Chronic Diseases; MA = Maternal Diseases; DI = Diseases of Infancy; ACC = Accidents; AOU = All other and unknown.

†OE *includes* malaria.

TABLE 4

ANNUAL PERCENTAGE DECREASE IN STANDARDIZED DEATH RATES BY SELECTED CAUSES*

Country	Total	RT	OI	IPB	DIA	MAT	DI	AOU+D
Uruguay	1.7	4.6	3.0	3.6	2.6	9.4	5.7	1.0
Chile	1.7	3.6	3.1	2.6	2.1	3.1	3.4	0.9
Colombia	1.9	1.9	3.7	1.9	2.2	3.1	0.4	3.3
Venezuela	1.7	4.3	2.4	. . .	4.8	2.4	2.5	2.8
Mexico	2.0	2.5	3.8	2.3	3.3	2.3	3.4	2.3
Salvador	2.0	3.2	3.9	1.6	2.1	2.9	3.0	1.9
Dom. Republic	1.5	4.0	4.1	2.7	3.0	2.4	1.8	0.2
Guatemala	2.3	3.0	5.2	1.5	1.3	†	†	2.6
Panama	2.0	3.2	3.1	2.1	1.7	3.3	†	2.5
Costa Rica	2.3	3.8	4.0	3.1	3.4	3.3	3.4	5.4
Average	1.91	3.41	3.58	2.38	2.65	3.22	2.95	2.29

*The abbreviations used correspond to (in order): Respiratory Tuberculosis; Infectious (includes Malaria); Influenza-Pneumonia-Bronchitis; Diarrhea; Maternal Diseases; Diseases of Infancy; Residual category plus other well-defined diseases.
†There was an increase of death rates in these categories.

even after twenty years as relatively outstanding contributors to the total SDR. Naturally, part of the increase of the incidence of cardiovascular diseases and neoplasms could be merely an artifact of improved diagnoses. However, it is more likely that they reflect both increased risks associated with environmental changes brought about by new economic and social conditions and the decreased prevalence of infectious conditions, respiratory diseases, and diarrhea among the population most susceptible to degenerative diseases.

Table 4 displays the annual per cent decrease by major groups of causes of deaths. By and large, the highest *rates of decrease* correspond to causes of deaths which had a relatively low contribution to the total SDR at the beginning of the period. Thus, respiratory tuberculosis and maternal diseases show rates of decrease that are larger than the rates for the more common diseases such as influenza, pneumonia, and bronchitis and diarrhea and only slightly lower than the rates corresponding to infectious diseases.

Perhaps not surprisingly, the standard death rates attributed to diarrhea have declined more slowly than most other cause-specific death rates. *In part*, this low rate of decrease may be due to changing classification practices. During the sixties and seventies, increasing evidence revealed that there was a close relationship between infectious diseases and some of the most common syndromes associated with diarrhea. In fact, it was shown that malnutrition, especially among infants and children may be at the root of both (Gordon et al., 1964; Scrimshaw et al., 1968; Puffer and Serrano, 1973). The resulting increased alertness to malnutrition and the stronger emphasis given to the classification of causes of deaths by other underlying and associated morbid conditions may have produced variations in the distribution of deaths by causes. Diarrhea-related deaths that during the fifties would have been assigned to one of the various categories of *infectious diseases* are, during the sixties and seventies, more likely to be assigned to the underlying conditions, namely malnutrition

and diarrhea, at the expense of the various categories of infectious diseases.

In addition, the changes implemented with the new classification system enacted during the sixties involve the classification of diarrhea of the newborn (0 to 4 weeks old) under the category corresponding to diarrhea, whereas in the previous classification system they were listed under categories included in diseases of infants. This change leads to an artifactual increase in the frequency of deaths attributed to diarrhea.

There are, however, reasons to suspect that the low rate of decline of the death rates attributed to diarrhea may be the outcome not of procedural or definitional practices but of a lack of sufficient improvements in standards of living. As shown in many studies a substantial fraction of the syndromes commonly associated with diarrhea are linked with poor nutrition and inadequate levels of hygiene. It could happen that many of the advantages brought about by the application of novel medical technology may be quite insufficient to produce significant reductions in the death rates due to diarrhea if unaccompanied by transformations in quality of food intake, practices related to the disposal of refuse, access to sources of uncontaminated water, and cleaning habits. It will be seen that the magnitude of the changes in death rates due to diarrhea is not only relatively low but almost *totally* attributable to changes in socioeconomic variables and not to medical innovations.

THE STRUCTURE OF CAUSES OF DEATHS

To study the contribution of each group of causes of deaths to the overall change in the standardized death rates we must formulate models relating the two. It is well known that the standardized rates by causes are closely associated with the total SDR (United Nations, 1963; Preston, 1976). This relation occurs in part as an artifact of the additive character of the total SDR. However, it is also due to environmental influences which affect simultaneously and in the same direction more than one cause of death at a time. By and large, the latter mechanism is responsible for most of the observed relations.

Considering all observations without sex distinctions, we have a total of 49 cases (country-years). On the basis of these observations, we estimated the following equations:

$$d_i = \alpha + \beta d_T + \epsilon_i \qquad (1)$$

where d_i and d_T represent respectively the ith cause-specific standardized death rate and the total standardized death rate.

The coefficients of determination and regression coefficients are displayed in Table 5. The diseases best predicted by the total SDR are in descending order: influenza, pneumonia and bronchitis, maternal diseases, infectious diseases, diarrhea, and respiratory tuberculosis. The highest fraction of the total relation which is to be attributable to the artifacts of additivity corresponds to the case of influenza, pneumonia, and bronchitis, where about 0.15 of the total *correlation* coefficient is accounted for by the presence of the dependent variable as a component of the independent variable. Subtracting this quantity from the total correlation still leaves a genuine relationship to 0.66 (or a coefficient of determination close to 0.44). In all other

TABLE 5

RESULTS OF REGRESSION ANALYSIS FOR THE RELATION BETWEEN CAUSE-SPECIFIC AND TOTAL SDR

Name	Intercept	Reg. Coeff.	R^2
Respiratory Tuberculosis	−0.00073	0.07713	0.481
Infectious*	−0.002715	0.2511	0.590
Neoplasms	0.00166	−0.03013†	0.077
Cardiovascular	0.00269	−0.01681†	0.005
Inf/Pneum/Bronch	−0.00200	0.22280	0.652
Diarrhea	−0.00104	0.13010	0.575
Chronic	0.00046	0.00682†	0.024
Maternal	−0.00004	0.00903	0.638
Diseases of Infancy	−0.00017	0.05530	0.311
Accidents	0.00079	0.00333†	0.004
Other & Unknown	0.0011	0.2913	0.396

†Including Malaria.
*Not significant at less than 0.05. All others significant at less than 0.01.

cases the contribution of the artifactual effects is quite small.

Cardiovascular diseases and neoplasms not only do not appear to be strongly associated with total levels of mortality but also show negative signs in their regression coefficients[3].

Since the sum of the slope coefficients ought to be one (Espenshade, ms.), they can be interpreted as the proportionate variation in each cause of death associated with a unit variation in the total SDR. Thus, about 22 per cent of a reduction in SDR ought to be ascribed to influenza, pneumonia, and bronchitis, 38 per cent to infectious diseases and diarrhea, and about 8 per cent to respiratory tuberculosis. By and large, the main contributors to the decline in the standardized death rates are those diseases in the groups of causes of death that display a major influence in the predecline levels and simultaneously are most likely to have been affected by the massive application of breakthroughs in chemotherapy and preventive medicine.

Examination of these coefficients presents us with some difficulties, however. Firstly, it should be noticed that the slope estimates only capture the influence of a particular cause of death in its role as a primary (underlying) cause and do not reveal the magnitude of the real contribution via its dependence on other causes of deaths. Secondly, they only give an average estimate of the contribution of each cause of death and re-

[3]Introduction of controls for the effects due to the size of death rates attributed to ill-defined causes changes things dramatically. The regression coefficient of cardiovascular diseases becomes positive and reaches a magnitude of about 0.28. This finding implies that cardiovascular diseases contribute about 28 per cent of a unit decline in the total SDR. This should not be surprising since a fraction of heart diseases are infectious in nature, or, at least, the direct result of complications following the onset of an infectious condition. Similar controls introduced in the case of neoplasms changes only marginally the estimate already obtained. The results of this procedure to control for the magnitude of the ill-defined category should be taken cautiously since, in general, such a category contains residual but well-defined causes of deaths.

TABLE 6

PER CENT CONTRIBUTION TO DECLINE IN INFECTIOUS DISEASES, 1950–1973

Country	Total*	TBO†	Typhoid	Whooping Cough	Measles	Dysentery	Malaria	SDR‡ Malaria
Uruguay	10.8	12.6	11.7	15.0	2.1	0.3	...	...
Chile	6.6	41.0	16.0	17.2	0.0	2.6	...	...
Colombia	16.0	5.8	4.1	15.2	5.9	4.7	14.3	32.0
Venezuela	6.1	10.7	5.6	18.2	−14.1	4.4	1.1	1.2
Mexico	22.9	1.2	5.2	12.2	17.6	12.4	42.8	122.0
Salvador	20.9	0.3	1.2	3.3	36.0	2.2	35.0	100.5
Dom. Republic	46.9	0.3	7.9	1.2	− 0.8	4.1	48.8	170.1
Panama	10.3	1.6	0.2	6.5	4.8	4.8	34.2	29.5
Guatemala	69.8	0.5	0.5	25.2	13.9	3.6	41.8	328.8
Costa Rica	14.9	1.6	2.1	18.0	4.1	4.1	17.0	23.5

*Per cent contribution of infectious diseases to total variation in SDR.
†Tuberculosis other than respiratory.
‡Standardized death rate due to malaria circa 1950 (per 100,000).

veal nothing about the heterogeneity in the process. There is substantial variability in the observed contribution of infectious diseases.

Table 6 displays a breakdown of the actual contribution of detailed infectious diseases contained in the aggregated category. Notice that the contribution to the decline of the total death rate due to infectious diseases ranges from a minimum of 6.0 per cent to maximum of 66.0 per cent. Also, the countries in the table can be neatly divided into two groups: those highly malarious at the beginning of the period, and those in which malaria was virtually absent or contributed only slightly to the SDR. Among the first group we find Mexico, El Salvador, Dominican Republic Guatemala, Nicaragua, Honduras, and Venezuela[4]. The second group includes the rest of the countries. There is a clear association between the degree of incidence of malaria, the actual contribution of malaria to the total decline in infectious diseases, and the actual contribution of infectious diseases to the total decline in SDR.

Among the highly malarious countries, the actual contribution of malaria to the change in infectious diseases is well above 35 per cent whereas for the nonmalarious countries the contribution appears to be considerably lower.

The most significant relation, however, is the one between the incidence of malaria and the total contribution of infectious diseases. The highly malarious countries invariably display a higher actual contribution of infectious diseases to the total change. If this were only a result of the high levels of malaria and its overwhelming direct contribution to the total infectious diseases its relevance would be limited. However, the relation appears to transpire other, less trivial regularities. The eradication of malaria through the combined operation of effective chemotherapy and elimination of the carrier mosquito not only depresses the frequency of new malaria

[4]The grouping criteria was by necessity rather arbitrary. Countries with initial levels of death rates due to malaria in excess of 0.30 per thousand were considered malarious. All the others were considered nonmalarious.

cases and the rate of mortality due to malaria but it simultaneously affects the incidence of other diseases.

The synergism between malaria, infectious diseases, and diseases of the respiratory system is a well-verified though poorly understood phenomenon. The implication is that the elimination of malaria has to have a multiplicative effect by reducing the rate of incidence of other diseases which are easily contracted in areas where the population has been weakened by the constant presence of malaria. It follows that a reduction in the death rate due to malaria will spill over as a reduction in the death rates attributable to other, particularly infectious, diseases and will consequently magnify its total contribution to the reduction in the SDR. This "echo" effect of malaria eradication is difficult to prove directly with the kind of aggregated type of evidence we have available. It has been illustrated however, for the case of Ceylon (Meegama, 1967; Newman, 1965) and has been suggested in a variety of accounts about the nature of malaria eradication programs and their effects (Preston, 1980).

This argument suggests that the relation between the standardized death rates due to infectious diseases and the total standardized death rates is likely to be different in malarious and nonmalarious countries. In fact, if separate regression are run for the two groups of countries, the following results are obtained:

Malarious Countries

$$d_{\text{infectious}} = -0.004620 + 0.3782\, d_{\text{total}} \qquad (R^2 = 0.82)$$

Nonmalarious Countries

$$d_{\text{infectious}} = -0.000498 + 0.0745\, d_{\text{total}} \qquad (R^2 = 0.74)$$

Thus, not only does the overall fit increase relative to the original regression, but more importantly the regression coefficients differ sharply[5]. As expected, malarious countries experience reductions in infectious diseases that are much larger than in nonmalarious countries for equivalent levels of changes in the total standardized death rate.

CHANGES OVER TIME IN THE STRUCTURE OF CAUSES OF DEATHS

It is important to test whether or not the structure of causes of deaths has changed as a consequence of variations in the environment, living conditions, or medical technology. For example, we should expect that as a society becomes more modernized, and as its population experiences improved standards of living and an increased access to medical services and new technology, the levels of infectious diseases will become less responsive to total changes in levels of mortality, the latter being largely dominated by the prevalence of chronic and degenerative diseases. To test this hypothesis two different procedures were followed:

1. The relations between total and cause-specific standardized death rates were re-estimated including a dummy variable for time period. Clearly, the most adequate cutting point to study changes in the structure of causes of deaths is some year within a period during which the mortality transition changed pace. By and large, the most substantial gains in survivorship in Latin American countries took place before

[5]The number of observations in the first group was 29, whereas in the second group it was 20. The regression coefficients are significantly different than zero (at a level of less than 0.05).

1970. Thus, we would expect that the effects of changes in the structure of causes of deaths would be observable across a period stretching between 1960 and 1970.

For lack of a more suitable criterion, we selected 1965 as the year of the cutting point. The dummy variable assumed a value of 0 if the information corresponded to a year before 1965 and a value of 1 otherwise. The estimated relation was as follows:

$$d_i = \alpha_1 + \alpha_2 D + \beta_1 d_T + \beta_2 D d_T + \epsilon ,$$

where α_2 and β_2 represent the "excess" in the intercept and slope with respect to the relation prevailing in years before 1965.

Table 7 displays the results of the estimation. In general, the expectations are not confirmed. Only for the residual categories did we explain a significantly higher variance by the addition of the dummy variables. In all other cases, the proportion of additional explained variance and, as a consequence, the coefficients of the dummy variables were statistically insignificant. Lack of statistical significance does not imply, however, that no systematic changes have taken place. Indeed there is quite an important pattern of changes that needs to be illustrated.

2. Estimates of magnitudes of change can be obtained by measuring the difference between the expected levels of cause-specific death rates as would be obtained in the relations prevailing before and after 1965. Thus, for example, a country's standardized death rates are associated with a certain level of cause-specific death rate in the relation applying to years before 1965. If the relation has shifted during the following years, the same value of the standardized death rate will be associated with a different level of the cause-specific death rate. The difference between the two is a rough measure of the magnitude of the shift of that level of mortality.

TABLE 7

RESULTS OF REGRESSION ANALYSIS FOR THE RELATION BETWEEN CAUSE-SPECIFIC, TOTAL SDR AND DUMMY VARIABLE FOR TIME PERIOD

Name	Intercept	Dummy Intercept	Regres. Coeff.	Dummy Reg/Coeff.	R^2
Respiratory Tuberculosis	−0.00084	0.00071*	−0.08338	0.0533 *	0.501
Infectious†	−0.00350	0.00012	0.28730	−0.06070	0.601
Neoplasm	0.001703	0.00057*	−0.03030	−0.05185*	0.109
Cardiovascular	0.00220	0.00198*	0.00881*	−0.1381*	0.037
Inf/Pneum/Bronch	−0.00274	0.00124*	0.25620	−0.05990*	0.672
Diarrhea	−0.00124	−0.00016*	0.13846	0.0251 *	0.583
Chronic	0.00040	0.00017*	0.00950*	−0.01118*	0.031
Maternal	−0.00001 *	−0.00004*	0.00779	0.00200*	0.655
Diseases of Infancy	0.00019 *	−0.00063*	0.03894	0.03093*	0.351
Accidents	0.00063	−0.00002*	0.01000*	0.01225*	0.063
Other & Unknown	0.00252 *	−0.00438*	0.22370	0.26500*	0.439

*Not significant at p less than 0.05. All the others significant at less than 0.01.
†Including Malaria.

TABLE 8

DIFFERENCE BETWEEN PREDICTED CAUSE-SPECIFIC DEATH RATE IN 1950–1965 AND EXPECTED, USING EQUATIONS FOR 1965–1975 AND TOTAL STANDARDIZED DEATH RATE IN 1950–1965

Country	Level of SDR in 1950–55	TBC	OI	IPB	DIA
Uruguay	0.0122	0.00006	0.00062	−0.00051	−0.00015
Chile	0.02059	0.00039	0.00113	−0.00001	−0.00036
Colombia	0.01909	0.00031	0.00104	−0.00010	−0.00032
Venezuela	0.01546	0.00011	0.00082	−0.00031	−0.00023
Mexico	0.02253	0.00049	0.00125	0.00011	−0.00041
El Salvador	0.02151	0.00044	0.00119	0.00005	−0.00038
Domin. Repub.	0.02090	0.00040	0.00115	0.00001	−0.00036
Guatemala	0.02587	0.00067	0.00145	0.00031	−0.00049
Panama	0.01605	0.00015	0.00085	−0.00028	−0.00024
Costa Rica	0.01609	0.00015	0.00086	−0.00028	−0.00024

Table 8 displays the differences for four major groups of causes of deaths for all countries included in the sample. Notice that the structure of causes of deaths has shifted, producing advantages (positive differences in the table) in terms of levels of respiratory tuberculosis and infectious diseases but producing disadvantages (negative differences in the table) in the levels of influenza-pneumonia-bronchitis and diarrhea. In fact, for all countries, the levels of death rates due to the first two groups of causes of death would have been less had the *level* of the total death rate for the beginning of the period prevailed in conjunction with the *relation* estimated for the years after 1965. The highly malarious countries again appear as a distinctive cluster evoking higher than average gains.

However, the differences are negative for half of the countries in the case of influenza-pneumonia-bronchitis and for *all countries* in the case of diarrhea. This implies that the levels of death rates due to these diseases would have been higher if the *relation* estimated for the years after 1965 had prevailed together with the *levels* of mortality experienced at the beginning of the period. It could be argued that this result should be expected since, after all, changes in mortality have to be reflected somehow in changes in the composition of causes of deaths.

The significant issue here, however, is that the highest and most systematic disadvantages that appear as a result of mortality changes seem to be associated with the complex of diseases contained in the category of diarrhea. These diseases, as emphasized before, are less sensitive to medical innovations that take place unaccompanied by socioeconomic transformation. If *both* socioeconomic condition and medical innovations had had a significant impact on the cause of death structure, the expected shift would have been in the direction of assigning a higher weight to causes of deaths which are known to be resilient to variation in those factors (such as some morbid processes affecting the newborn) and a lower weight to other causes of deaths (such as diarrhea and influenza-pneumonia-bronchitis). This is not evident from the results elaborated here. Quite the contrary, the changes in the structure of causes of

death appears to reveal little of the impact of substantial modifications in socioeconomic conditions.

PECULIARITIES OF THE LATIN AMERICAN STRUCTURE OF CAUSES OF DEATHS

It would be erroneous to assume that the structure of causes of deaths found in Latin America is in any sense a typical one for countries that are or have been at similar levels of mortality. Variations in environmental and socioeconomic factors may have powerful influences in the determination of levels of incidence of various causes of deaths and their relations. In fact, the heterogeneity in age patterns of mortality observed across geographical regions is largely a product of forces that shape different patterns of causes of deaths. Since Latin American countries appear to have a peculiar age pattern of mortality (Palloni, 1981) it would be useful to be able to link it to an unusual or, at any rate, deviant cause of death structure. For lack of a better alternative we have selected as a standard for comparison the typical relations between levels of mortality and incidence of causes of deaths embedded in a sample of Western European, Asian, and Latin American countries (Preston, 1976).

In Table 9 we have calculated the values of death rates due to diarrhea, infectious diseases, and influenza-pneumonia-bronchitis that would be expected if the standard relation prevailed. These are contrasted with the observed ones in Latin America at the beginning and end of the period considered. By and large, it is again the malarious countries which show the most deviant behavior. At the beginning of the period they invariably appear to have excessive mortality due to infectious diseases and diarrhea. At the end of the period, the excesses from infectious diseases have disappeared but have remained at a somewhat lower level in the case of diarrhea. All other countries, with the exception of Colombia and Costa Rica, show values within the expected range.

Although the high incidence of malaria may be a powerful determinant of mortality excesses, it is difficult to insist on its importance at the end of the period when the disease had all but disappeared. In particular, it is likely that the excess of diarrhea is more closely associated with levels of nutrition and hygiene affecting predominantly the health of infants and children. As has been shown elsewhere, the impact of diarrhea is mostly concentrated among children 0 to 5 years of age (Palloni, 1981).

The values in Table 9 should be taken as lower bounds for the estimates of excesses in mortality or as a conservative appraisal of the deviant character of the structure of causes of deaths in Latin America. There are two reasons for this. First, no attempt was made to distribute the deaths in the ill-defined category. If this had been done, the cause-specific death rates due to the three major groups of diseases would have increased proportionately more than others. Second, the standard selected for comparison is a bad choice. In fact, the standard relation was estimated including cases that do possess potentially deviant structures of causes of death. The effect is to impart to the overall relation a more "deviant" character than would have been the case had those countries been excluded from the analysis.

In summary, the following seem to be the major features of mortality decline in the two decades following 1950:

(*a*) Mortality has declined very rap-

TABLE 9

COMPARISON OF EXPECTED DEATH RATES AND OBSERVED DEATH RATES FOR THREE GROUPS OF CAUSES OF DEATHS*

COUNTRY	BEGINNING OF PERIOD†						END OF PERIOD‡					
	OI		Diarrhea		IPB		OI		Diarrhea		IPB	
	Range	Obs.	Range	Obs.	Range	Obs.	Range	Obs.	Range	Obs.	Range	Obs.
Uruguay	0.28–0.61	0.33	0.28–0.57	0.30	0.86–1.37	0.63	0.04–0.37	0.15	0.11–0.40	0.26	0.39–0.97	0.45
Chile	1.50–1.71	0.75	1.16–1.44	0.57	3.23–3.41	3.34	0.34–0.66	0.22	0.32–0.61	0.29	0.97–1.47	1.34
Colombia	1.28–1.57	1.58	1.00–1.29	1.06	2.80–3.05	1.72	0.28–0.61	0.48	0.28–0.57	0.61	0.85–1.37	1.10
Venezuela.....	0.75–1.06	0.64	0.62–0.91	0.69	1.78–2.16	0.56	0.08–0.42	0.36	0.14–0.43	0.34	0.47–1.04	0.75
Mexico.....	1.78–2.05	2.80	1.37–1.65	3.24	3.78–3.88	3.34	0.32–0.64	0.49	0.31–060	0.92	0.92–1.43	1.65
Salvador	1.64–1.91	2.35	1.26–1.54	3.35	3.49–3.64	1.16	0.29–0.66	0.42	0.28–0.58	1.31	0.87–1.39	0.77
Domin. Repub. .	1.55–1.82	3.59	1.19–1.48	1.53	3.32–3.49	1.38	0.55–0.87	0.38	0.48–0.76	0.51	1.38–1.82	0.55
Guatemala	2.27–2.52	8.11	1.72–1.99	2.64	4.70–4.72	2.64	1.22–1.51	3.08	0.96–1.24	2.24	2.68–2.94	2.88
Panama	0.84–1.14	1.06	0.69–0.97	0.49	1.94–2.31	1.34	0.08–0.26	0.41	0.02–0.31	0.32	0.16–0.77	0.79
Costa Rica.....	0.85–1.15	1.38	0.69–0.97	1.19	1.96–2.32	1.81	0.23–0.12	0.28	0.09–0.21	0.37	0.14–0.52	0.67

*The range has as a maximum the expected cause-specific standardized death rate in the female equation. The minimum always coincides with the expected cause-specific standardized death rates in the male equations (see Preston, 1976, p. 18).

†Around 1955.

‡Around 1973.

idly producing reductions in SDR equivalent, on the average, to gains of about 0.7 years of life expectancy per year. *Absolute* decline in SDR is higher in magnitude for countries with the highest initial levels of mortality. The relative decline in SDR, however, has had very little variance and appears to be slightly higher among countries with the lowest levels of mortality.

(*b*) The major contributors to mortality decline have been infectious diseases, influenza-pneumonia-bronchitis, and diarrhea. The contribution of infectious diseases to the total decline overshadows other contributions among countries with initially high levels of malaria.

(*c*) Although we failed to find statistically significant shifts in the structure of causes of death, it appears that the changes have resulted in a structure of cause of deaths with a disproportionately high contribution of diseases associated with diarrhea and, to a lesser extent, with influenza-pneumonia-bronchitis.

(*d*) The structure of causes of death in Latin America is not homogeneous. One group of countries deviates consistently from an arbitrarily selected standard whereas all the others are contained within an expected range. The outlying cause of death structure appears to be characterized by high death rates due to infectious diseases and diarrhea. Although high incidence of malaria prevailing at the beginning of the period under study is associated with the deviations, important socioeconomic factors may be at play to foster persistent mortality excesses attributable to the complex of diseases contained under the categories of diarrhea and infectious conditions.

DETERMINANTS OF MORTALITY DECLINE: EXOGENOUS OR ENDOGENOUS FACTORS?

The experience of Western European countries has showed us the important role that economic factors as reflected in standards of living have played in the decline of mortality (McKeown, 1976). Although undoubtedly the invention of new medical techniques (chemotherapy, immunization, surgery) did have a strong impact on mortality changes, a significant part of the latter seems to have preceded the advances in germ theory, probably the single most influential contribution to the control of mortality conditions.

The situation in developing countries in general and Latin America in particular is indeed different. First of all, substantial mortality declines were observed in most Latin American countries in a short period following the end of World War II. Thus, although in the last few years the process seems to have slowed down, the bulk of the decline took far less time than in Western European countries (at least when the latter are considered at comparable levels of mortality). Second, in only a few of those countries (particularly Chile, Uruguay, and Argentina) was there substantial economic development before the onset of mortality decline. Although it is not possible to deny that some improvements have occurred, the point is that equivalent changes in life expectancy appear to have required, or been associated with, less gains to economic development than was the case in Western Europe and North America. Thus, there would seem to be more grounds than in the case of Western Europe to impute an important fraction of the mortality decline to exogenous factors, particularly medical technology

imported at low cost from developed countries.

Some objections can be raised against this line of reasoning, however. In fact, although the direct cost of immunization programs and the direct and indirect costs of making some type of chemotherapy accessible to the population, may not take a substantial fraction of national budgets, their application, maintenance, and surveillance for sustained periods of time require a relatively well-organized institutionalized machinery which is not always present in these countries. As a consequence, they will be relatively high priced "commodities" from a political and administrative point of view. This suggests that to try to extricate the effects of medical technology from those attributable to endogenous factors associated with economic development is to distort the formulation of the problem from the start.

There are some reasons to believe, however, that the deployment of *some type* of medical technology may be carried out quite independent of socioeconomic (and political) development. It is very likely that the reach of such technology is limited and its effects may wither away soon after application has started. This leads to the question of the magnitude of the initial changes that cannot be accounted for by advances in standards of living and should be presumably imputed to the volatile effects of such type of medical technology (Newman, 1970; Meegama, 1967; Gray, 1974; Fredericksen, 1961).

In the next section we try to provide rough estimates of the contribution to Latin American mortality decline during the years 1955 and 1973 that could be imputed to noneconomic factors. We will show that these "exogenous factors" turn out to be less significant than has been previously thought and appear to have had little influence in changing the incidence of causes of deaths which are to be held responsible for the currently high death rates in some Latin American countries.

THE RELATION BETWEEN SOCIOECONOMIC FACTORS AND TOTAL STANDARDIZED DEATH RATES

For the purpose of studying the role that socioeconomic factors have played in the process of mortality decline, we had to include more observations into our data base. In all, 18 Latin American countries with known age-specific death rates for 1955 and 1973 and with accessible information on socioeconomic variables were selected.[6]

Although life expectancy is an attractive measure of mortality conditions, we preferred to use the SDR. This was done in part to maintain continuity with the results of the previous section and in part because the hypotheses to be tested require the introduction of decomposition, a task which is not easily achieved if life expectancy is used.

The first and most evident relation that should be examined is the one between the SDR and a single measure of overall economic conditions. Traditionally, the Gross National Product per capita (GNP) has been the preferred choice (although there are grounds to select others such as Energy Consumption per capita). We should expect the relation between SDR and GNP to be nonlinear since the returns in the form of mortality improvements to amelioration of economic conditions ought to be

[6]In all cases corrections for underregistration were applied. The procedures utilized were those indicated in the first section of this paper.

higher at the lower levels of the GNP scale. To incorporate the effects of diminishing returns to improvements in socioeconomic conditions, researchers have experimented with a variety of functional specifications. For example, Preston (1976) and Dyson et al. (1978) have suggested a logistic curve to describe the relation between life expectancy and income. Later Preston (1980) utilized a simpler formulation with life expectancy being a linear function of the logarithm of income, a measure of education (proportion of adult population who is literate), and a measure of services (proportion of population with access to piped water). Recently, Chao (1979) attempted to fit a more complicated model whereby life expectancy was expressed as a logistic function of several covariates other than income.

In studies of mortality in infancy and early childhood, other suggestions have been proposed. Thus, for example, Rodgers (1979) experimented with a hyperbolic specification in which the infant mortality rate was a function of the reciprocal of GNP whereas Flegg (1981) and Palloni (1981) have utilized double logarithmic functional forms, i.e., the logarithm of infant mortality rate is expressed as a linear function of the logarithm of several covariates.

Column 1 of Table 10 shows a subset of commonly used functional forms. In this table the dependent variable is the standardized death rate. For simplicity of presentation, only one independent variable (income) has been considered. Column 2 of Table 10 displays the (approximate) magnitude of the change in the standardized death rate due to a small change in the independent variable. The third column displays the *elas-*

TABLE 10

ALTERNATIVE MODEL SPECIFICATIONS OF THE RELATION BETWEEN STANDARDIZED DEATH RATE AND A SINGLE COVARIATE MEASURING ECONOMIC DEVELOPMENT

(1)	(2)	(3)
Model 1		
$y = \alpha + \beta \ln x$	$\Delta y = \beta \frac{\Delta x}{x}$ $(\beta < 0)$	$\frac{\beta}{y}$ or $\frac{\beta}{\alpha + \beta \ln x}$
Model 2		
$\ln y = \alpha + \beta x$	$\Delta y = \beta \Delta x y$ $(\beta < 0)$	$\frac{\beta}{x}$
Model 3		
$\ln y = \alpha + \beta \ln x$	$\Delta y = \beta \frac{\Delta x}{x} y$ $(\beta < 0)$	β
Model 4		
$y = \alpha + \beta x^{-1}$	$\Delta y = -\beta \frac{\Delta x}{x} \frac{1}{x}$ $(\beta > 0)$	$-\beta \frac{1}{xy}$ or $\frac{-\beta}{\alpha x + \beta}$
Model 5		
$y = \eta - \frac{\alpha}{1 + \gamma e(4)^{-\beta \ln x}}$	$\Delta y = -\beta\gamma \frac{\Delta x}{x} (\eta - y)\left(1 - \frac{\eta - y}{\alpha}\right)$ $(\beta > 0)$	$\frac{-\beta\gamma}{y} (\eta - y)\left(1 - \frac{\eta - y}{\alpha}\right)$

ticities of the standardized death rate to the independent variable (i.e. the ratio of the relative change in the dependent variable to the relative change in the independent variable).

In all cases, with the exception of Model 1, a given proportionate increase in x generates a reduction in SDR the magnitude of which diminishes for high values of x (low values of SDR). Similarly, with the exception of Models 1 and 3, the elasticities of SDR to x diminish as x increases (or as SDR decreases).

If one wishes to maintain the restriction that the *proportionate* returns to *proportionate* improvements in socioeconomic conditions ought to remain constant or diminish as the levels of socioeconomic development improve, then Model 1 cannot be tolerated among acceptable functional forms. This is a highly desirable restriction. In fact, in societies with low levels of development and high levels of mortality, minor changes in wealth can be substantially stretched to produce significant reductions in mortality. This is due to the fact that at low levels of income economic improvements generally result in higher health expenditures and a considerable upgrading in the general levels of nutrition and hygiene. By and large, this is sufficient to bring under control the deleterious effects of widespread infectious diseases. At lower levels of mortality and higher levels of development, comparable proportional gains in survivorship require relative heavier health expenditures and larger investments in high priced medical technology which permit the treatment of chronic and degenerative diseases. Even then, the outcomes of such investments produces only marginal gains in survivorship. Thus, the most appropriate formulation of the relation between SDR and GNP ought to be one in which there is explicit recognition of constant or diminishing elasticities. This is of extreme importance even in the analysis of a sample of countries like ours, one where the levels of SDR fall within a range in which the effects of diminishing returns have not yet been felt to any significant extent. However, according to recent evidence discussed elsewhere (Palloni, 1981; Gwatkin, 1980), developing countries may be undergoing a mortality transition characterized by a rather premature slowdown of the rate of increase in life expectancy or, equivalently, by a faster decay of the survivorship benefits derived from improvements in the levels of socioeconomic development.

This discussion justifies the selection of functional expressions with constant or decreased income elasticity of mortality and hence supports the exclusion of Model 1 in Table 10. Selection between all the other functional forms, however, is much more problematic and will be addressed below by elaborating a criterion based on the statistical theory of power transformations.

Our purpose at this stage of the analysis can be stated as follows: Provided that we use one among the acceptable functional specifications, is it possible to estimate the contribution of socioeconomic changes separately from the contribution of exogenous, nonsocioeconomic factors? If so, and the functional specifications are comparable, do the estimates confirm or disprove the conclusions reached by other research?

One of the first serious attempts to separate out the contribution to mortality change of endogenous and exogenous mortality changes was carried out by Preston (1976). He used a logistic

functional specification for the life expectancy and a single measure of socioeconomic development (GNP). In order to replicate the analysis, we have estimated the parameters of Model 5 fitted to the standardized death rates for the years 1955 and 1973 with GNP as the only covariate of interest. The estimates of the parameters for the two logistic curves are displayed at the bottom of Table 11[7]. For 1955 and 1973 the proportions of explained variance (R^2) are 0.68 and 0.48 respectively. Although low, they and the coefficients β are significant at less than 0.05. The reduction of the explained variance in the last period could be taken as an indication of the weakening link between economic and mortality conditions. The change or shift in the relation prevailing in 1955 is not only restricted to the fact that mortality appears to be less responsive to changes in GNP in the sample of 1973 than in the sample of 1950. In fact, the levels of death rates, at equivalent levels of GNP, would be lower had the relationship estimated for 1973 prevailed throughout the period.

An admittedly crude way of estimating these changes is to average the differences between the predicted death rates in 1950 and 1970 obtained using the corresponding values of GNP in the relation estimated for the other period. Thus, for example, with the levels of GNP prevailing in Argentina in 1955, the predicted (standardized) death rate is 14.03. In the relationship estimated for 1973, the same value of GNP would have produced a (standardized) death rate equal to 11.53 or a difference of 2.50. The converse combination yields a difference of 2.58. On the average, the magnitude of the change in the death rate that cannot be attributed to socioeconomic factors (as measured by GNP) is about 2.54.

TABLE 11

ESTIMATES OF STRUCTURAL SHIFT IN RELATION SDR AND GNP

Country	Average Difference Due to Shift in the Relation
Argentina	2.54
Bolivia	5.27
Brasil	4.41
Colombia	4.72
Costa Rica	3.73
Chile	2.55
Ecuador	5.20
Salvador	4.85
Honduras	5.03
Mexico	3.64
Nicaragua	4.39
Peru	3.88
Uruguay	2.30
Venezuela	2.55
Paraguay	5.14
Guatemala	4.29
Panama	3.41
Domin. Repub.	4.63
Average	4.03

Parameters of the Logistic Functions	*1955*	*1973*
η	22.00	19.50
α	9.70	12.00
ρ	13.12	2.62
β	0.00387	0.00168
R^2	0.68	0.48

[7]It should be noted that the estimation procedure we applied does not require preassigning the value of the asymptotes before an estimate of the other parameters can be obtained. In fact, the numerical procedure used, a modification of the so-called Marguardt's compromise, is sufficiently powerful to estimate all parameters simultaneously. Preston, however, began by preassigning an arbitrary value to the asymptotic value of the life expectancy and only after that did he calculate estimates for the remaining parameters. In this sense, our results are not obtained from a totally equivalent replica of Preston's procedure. Of course, the other difference between this model and that estimated by Preston is the specification of the dependent variable. Since the relation between one and the other (life expectancy and SDR) is linear and very close, the effects of this difference should not be significant.

Table 11 displays the average values of the differences for all countries included in the sample. The unweighted average of these differences is 4.03. Between 1955 and 1973, Latin American countries experienced reductions in SDR of about 5.59. Our results suggest that nearly 72 per cent of this total gain was due to exogenous or noneconomic factors. This estimate is quite close to the one obtained in a much larger sample of developed and developing countries over the period 1930–1970 (Preston, 1975).

THE RELATION BETWEEN SOCIOECONOMIC FACTORS AND CAUSE-SPECIFIC STANDARDIZED DEATH RATES

Three modifications to the procedure applied require consideration. First, the previously estimated relation could be misspecified because not all relevant socioeconomic variables have been included in the equation. If this were the case, the contribution of exogenous factors could be upwardly biased. Among other indices of socioeconomic development, we have deliberately left out two of considerable importance: education and service availability.

On the one hand, indicators of education have been found to be among the most powerful predictors of mortality. This not only applies to aggregate level associations but to relations holding at the individual level as well (Preston, 1976; Caldwell, 1979; Schultz, 1979; Palloni, 1981). Aggregate levels of education appear to be not only a good proxy for the extant levels of available social resources (other than economic assets) that can be brought to bear to check mortality risks but also for the level of inequality in the distribution of both economic assets and social resources. It is not surprising then that indices of literacy are as a rule very highly correlated with indices of income inequality.

On the other hand, perhaps the single most important service whose availability may control mortality levels is access to piped water. One should remember the extraordinary importance that the access to decontaminated water has had historically in the changes in mortality conditions (Flinn, 1981; Winslow, 1980). It is of particular relevance in countries for which the principal causes of deaths are water- and food-borne diseases—diarrhea, dysentery, and other gastrointestinal infections. To measure education we have used the proportion of the adult population which is *illiterate*. To measure access to water services we have used the proportion of the total population with direct access to piped water (within the actual home or outside of it, in a common facility). Both variables will be used in their natural logarithmic form to ensure that, within acceptable functional formulations, the magnitude of changes in SDR due to a change in the independent variable depends on *the relative magnitude of the latter.* [8]

The second modification that needs to be introduced requires a better assessment of the most accurate and efficient functional form expressing the relation between mortality and the covariates. How can it be possible to select unobtrusively between the models presented in Table 10? Notice that the

[8]Illiteracy and water availability are, at the individual level, dichotomous variables. Therefore, their effects can only be linear. However, at the aggregate level both indicators are defined over a (bounded) continuum and hence, as occurred with GNP, their effect on mortality should diminish as their levels increase (decrease).

logistic formulation is statistically inefficient since within the range of observation of the independent and dependent variables its contours must be very similar to others included in Table 10. Yet its estimation requires the estimation of four parameters rather than two. Furthermore, a relatively high premium is attached to the correct estimation of the asymptotic value of the dependent variable under conditions in which the observations at hand contain little information on it. The disadvantage of using some of the other functions included in Table 10 is that inevitably we will lose a degree of freedom trying to select among them. We consider this a minor price to be paid for additional robustness.

We propose to consider positive valued functions within the following family of power transformations:

$$y^{(\lambda)} = \begin{cases} (y^{\lambda} - 1 \div \lambda & \text{if } \lambda \neq 0 \\ ln\, y & \text{if } \lambda = 0 \end{cases}$$

where β is a column vector of parameter, X is a matrix of the natural logarithm of the covariates, Y is a column vector of standardized death rates, yi, and ϵ is a vector of normally distributed errors, ϵi, with mean 0 and variance σ. This family of power transformations was suggested by Box and Cox (Box and Cox, 1964) to deal with cases of strictly positive dependent random variables associated with one or several fixed covariates. λ is an additional parameter that needs to be simultaneously estimated with the vector β. The procedure for estimation requires numerical evaluation of the logarithm of the corresponding maximum likelihood functions. Table 12 shows a set of models than can be generated when λ assumes three values within the range $(-1, 1)$. Notice that Models 2 and 3 have their counterparts in Table 10 whereas Model 1 produces effects analogous to those of Models 2 and 4 in Table 10. By and large, λ is found in the range $[-2.0, 2.0]$. Selection of a particular value of λ is carried out maximizing the corresponding log-likelihood function.

The third and last modification to be introduced to the procedure described before is quite simple. In fact, if we are to decompose the contribution of exogenous and endogenous factors to the

TABLE 12

MODELS RELATING SDR TO ONE COVARIATE FOR VARIOUS VALUES OF λ IN THE BOX-COX FAMILY OF POWER TRANSFORMATIONS

Models	(1) Value of λ	(2) Changes in y	(3) Elasticities
Model 1			
$\lambda = -1$	$y^{-1} = \alpha + \beta\, ln\, x$	$\Delta y = \beta y^2 \frac{\Delta x}{x}$	βy
Model 2			
$\lambda = 0$	$ln\, y = \alpha + \beta\, ln\, x$	$\Delta y = \beta \frac{\Delta x}{x} y$	β
Model 3			
$\lambda = 1$	$y = \alpha + \beta\, ln\, x$	$\Delta y = \beta \frac{\Delta x}{x}$	$\frac{\beta}{y}$

changes in *total* mortality, it would be theoretically more advantageous and logically more appealing to estimate a decomposition for each major cause of death. After all, endogenous and exogenous factors can have an impact on the total levels of mortality only by way of modifying the intensity of the major causes of deaths. There is no guarantee, and one should not expect it, that the cause-specific effects will behave in the same way as the total effects. In order to carry out the cause-specific decomposition, we estimated the best-fitting power transformation for total and each of four cause-specific standardized mortality rates: respiratory tuberculosis, other infectious diseases, influenza-pneumonia-bronchitis, and diarrhea. The results appear in Table 13[9]. For the first time period, the optimum representation turns out to be a logarithmic form ($\lambda = 0$), whereby standardized death rates are modeled as in a production function with the independent variables as inputs. Figure 1 (lower panel) shows the contours of the log-likelihood function for SDR in 1950–1955 for different values of λ. Similar graphs are obtained for each of the four major causes of deaths. Their maximum is always attained at $\lambda = 0$. Notice that the explanatory power of each equation is quite high and that all coefficients, with the exception of β_3 for respiratory tuberculosis and influenza-pneumonia-bronchitis, are properly signed. It is for these last two causes of deaths that the second most powerful explanatory variables, illiteracy, fails to be statistically significant. The first most powerful explanatory variable, GNP, only fails to be statistically significant in the case of respiratory tuberculosis. By and large, the effects of income and education are strongest for infectious diseases and diarrhea: a 1 per cent increase in GNP (or decrease in illiteracy) is associated with directly or indirectly generated decreases in the corresponding cause-specific death rate of more than half of a per cent.

The results for 1973 are quite different, however. In the first place it is no longer true that a single power transformation is sufficient for all causes of death considered. Thus, whereas the optimum model for respiratory tuberculosis, influenza-pneumonia-bronchitis, and diarrhea preserves the logarithmic form ($\lambda = 0$), this is no longer true in the case of total SDR and death rates due to infectious diseases. In fact, for the last two death rates the optimum model requires $\lambda = -1$[10]. Figure 1 (upper panel) shows the contours of the log-likelihood function for total SDR (the maximum is attained at $\lambda = -1$). This heterogeneity in the response of death rates to socioeconomic conditions had already been anticipated and should be adequately preserved rather than blurred by imposing a single functional representation for all causes of death.

The second feature revealed in Table

[9]For countries with no information on cause-specific death rates at the beginning of the period we proceeded to estimate them on the basis of the relation between cause-specific death rates and total death rates in the total sample. The regressions were estimated in logarithmic form. This increased substantially their predictive power and eliminated the possibility of obtaining inadmissible values for the cause-specific death rates.

[10] The reader should recall that if $\lambda = -1$, the functional specification becomes

$$-1 \div y = (-1 + \alpha) + \beta_1 ln\ \text{GNP} + \beta_2 ln\ \text{Illit} + \beta_3 ln\ \text{Wa}.$$

Thus, a positive coefficient implies that positive increments in the covariates force positive increments in the dependent variable.

TABLE 13

ESTIMATED REGRESSION EQUATIONS OF THE FORM
$d_i^{(\lambda)} = \alpha + \beta_1 ln\text{GNP} + \beta_2 ln\ \text{Illit} + \beta_3 ln\text{Wa}$,
LATIN AMERICAN COUNTRIES, 1950–1973*

Years	λ	α	β_1	β_2	β_3	R^2	Adjusted R^2	F Ratio
Years 1950–1955								
Cause:								
SDR	0	2.71	−0.102 (0.044)	0.219 (0.036)	−0.001 (0.012)	0.901	0.883	51.40
Respiratory Tuberculosis ..	0	−1.16	−0.146 (0.245)	0.378 (0.203)	0.034 (0.066)	0.470	0.377	5.03
Infect. Dis. ...	0	1.85	−0.651 (0.225)	0.737 (0.225)	−0.037 (0.060)	0.852	0.826	32.68
Influ-Pneu-Bronchitis	0	4.41	−0.655 (0.304)	0.024 (0.252)	0.001 (0.082)	0.404	0.298	3.84
Diarrhea	0	1.96	−0.597 (0.204)	0.570 (0.169)	−0.078 (0.055)	0.825	0.794	26.71
Years After 1973								
Cause:								
SDR	−1.0	0.928	0.002 (0.009)	0.011 (0.004)	−0.014 (0.008)	0.672	0.614	11.61
Respiratory Tuberculosis ..	0	−4.625	0.614 (0.763)	1.062 (0.364)	−1.156 (0.732)	0.605	0.536	8.71
Infect. Dis. ...	−1.0	−0.503	0.053 (0.411)	0.543 (0.196)	−0.669 (0.394)	0.677	0.623	12.02
Influ-Pneu-Bronchitis	0	−3.361	0.345 (0.437)	0.769 (0.208)	−0.399 (0.419)	0.639	0.576	10.05
Diarrhea	0	0.666	−0.317 (0.307)	0.470 (0.146)	−0.159 (0.294)	0.739	0.693	16.04

*In parentheses appear the standard errors of the regression coefficients.

13 is that the explanatory power of the equation for all causes of deaths (including the total SDR) had diminished considerably by 1973. Furthermore, the coefficients for the variable GNP show signs opposite to the expected, although they are always insignificantly different from zero. Only in the case of diarrhea does an increase in GNP appear to be associated with lower standardized death rates.

Illiteracy and access to water are now the most important variables (in all cases statistically significant and properly signed). However, even though illiteracy preserves its predictive power, the sensitivity of mortality to changes in illiteracy has been greatly diminished. This is clearly seen in Figure 2 which displays plots of total SDR against the logarithm of illiteracy for both periods according to the optimum functional

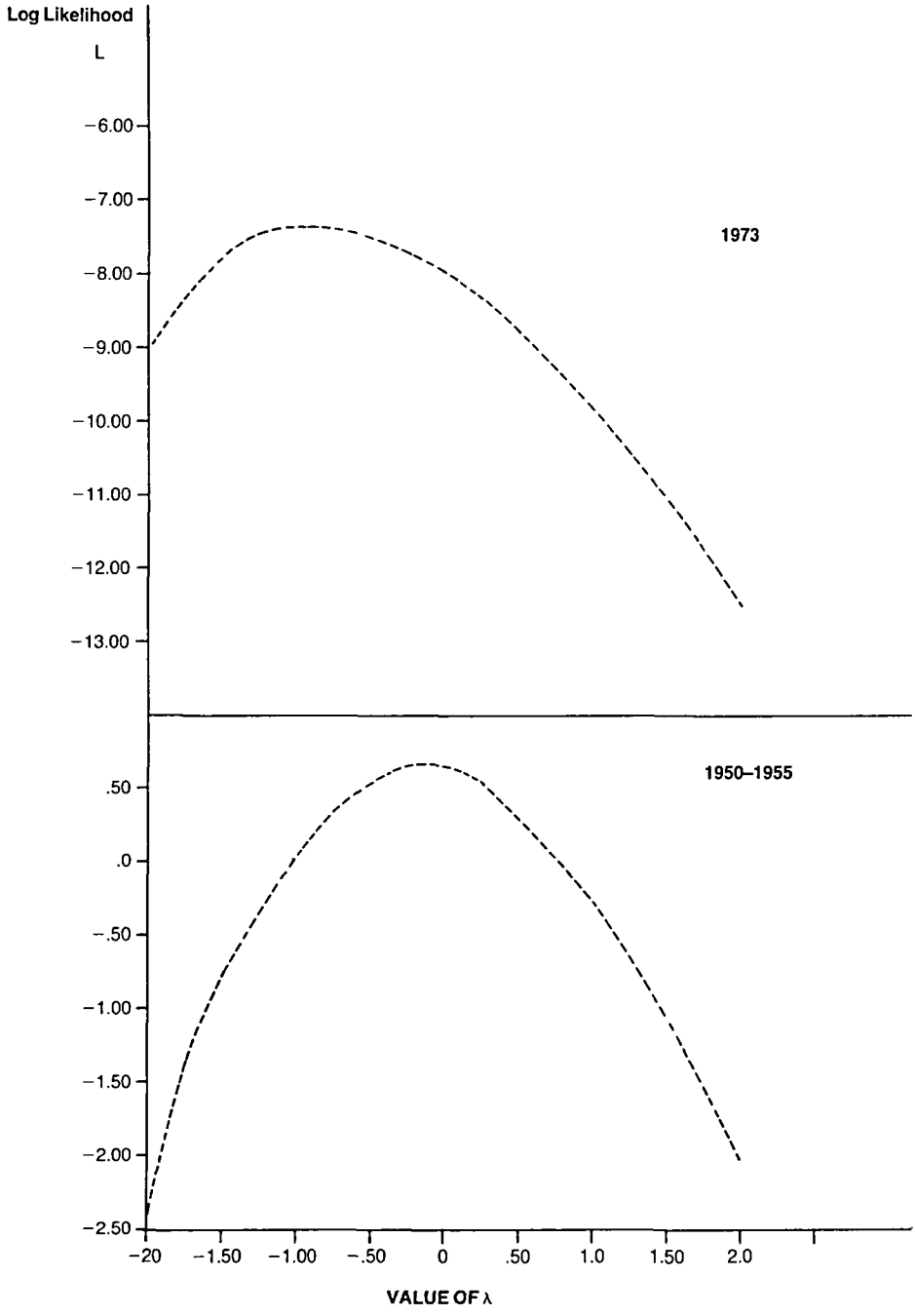


FIG. 1.—Values of log-likelihood function for Box-Cox power transformations.

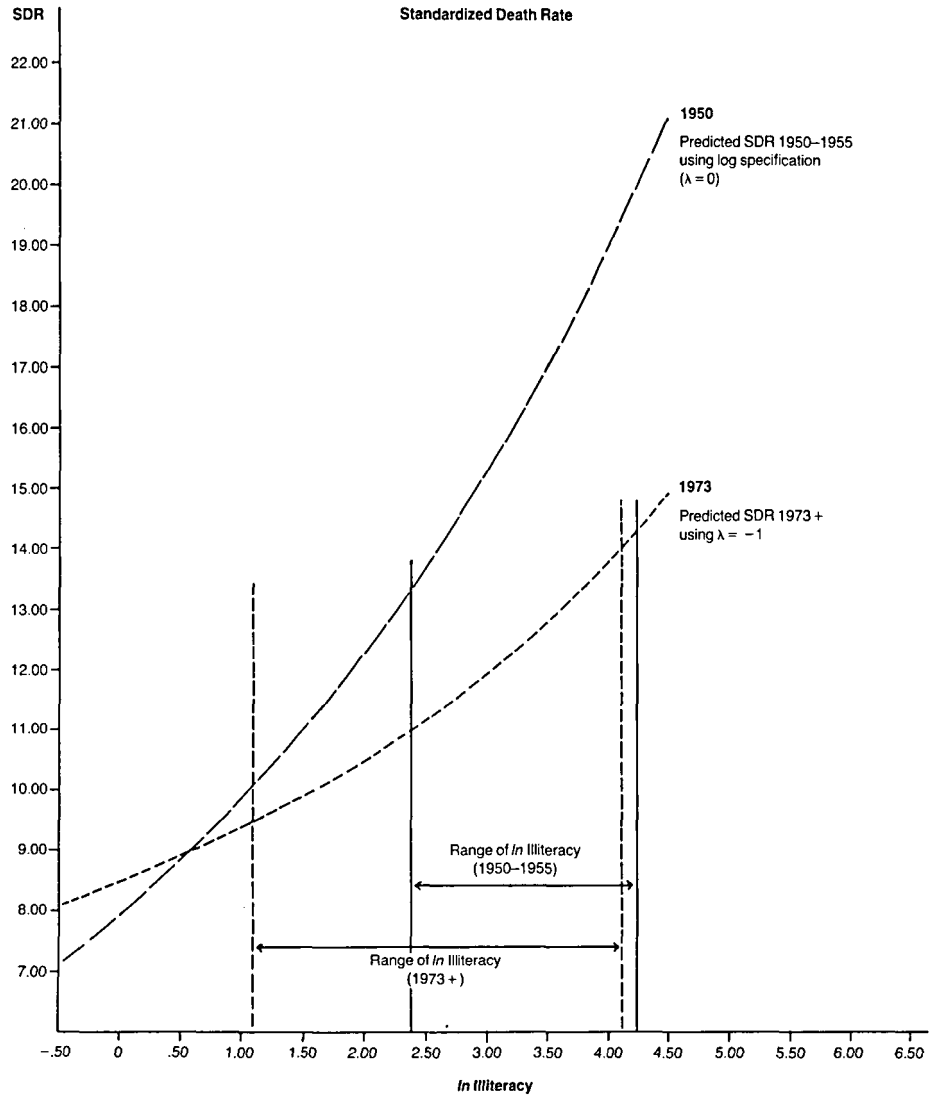


FIG. 2.—Comparison of the curves for SDR in 1950–55 and 1973. (Illiteracy is the only covariate changed.)

specification (the other two variables were set equal to their average values). Notice that, within the range of observed illiteracy, the curve for 1950–1955 rises much more steeply in 1950–1955 than in 1973 and achieves rapidly diminishing returns at much lower levels of illiteracy.

In summary, it is apparent that expenditures in education may produce higher returns than equivalent increases in GNP. However, unlike previous years, the proportionate returns are dependent on the attained levels of mortality (see Table 12, first model) and reach lower magnitudes. Thus, for example, a

1 per cent decrease in illiteracy during 1950–1955 was associated with a total decrease in SDR of about 0.22 of per cent. According to the estimated parameters for 1973, the same decrease in illiteracy produces a total decrease of about 0.14 of 1 per cent (0.011×12.46, where 12.46 is the average SDR for the second period).

The reversal of sign and uniform insignificance of the effects for the variable GNP could be due to several factors. First, the levels of GNP could, in fact, be poorer indicators of the actual level of socioeconomic conditions affecting mortality levels. Inequalities in the distribution of wealth not captured by the other two variables, social and geographical integration, as well as the efficiency of governmental machineries may be much less responsive than before to changes in the aggregate levels of wealth. Second, the generalized sensitivity of levels of mortality to changes of GNP may be negligible due to the fact that at lower levels of mortality equivalent proportionate gains in survivorship are harder to attain. This is the finding that most other research has uncovered for other countries.

There is a third factor that could be responsible for this observed phenomenon, and yet it is of artifactual nature. In fact, high multicollinearity in the estimated models may increase the unreliability of the coefficients. However, the correlation coefficients between GNP and illiteracy are -0.638 and -0.635 in 1950–1955 and 1973 respectively. These are higher than desirable, but by no means do they create an unacceptable level of multicollinearity. In fact, the size of this coefficient is moderate and routinely encountered in research of this type. There is thus no clear rationale for deleting the variable GNP from the model specification.

Following the same procedure described before we partitioned the changes in death rates into two components: one attributable to changes in the standards of living (as measured by the observed variation in the independent variables), and the other attributable to changes in exogenous factors (as measured by observed shifts in the estimated relations). Table 14 displays the results of the decomposition separately for causes of deaths and for two groups of countries, distinguished according to malaria endemicity at the beginning of the period. The overall contribution of exogenous factors, equivalent to 45.0 per cent of the total changes is now much lower than estimated before using the logistic formulation with only one covariate. It is highest for infectious diseases (56.0 per cent) and much lower for tuberculosis, IPB, and diarrhea. This finding is consistent with the hypotheses presented before which suggested that: (*a*) mis-specification of the model leads to overestimation of the contribution of exogenous factors, and (*b*) the contribution of exogenous factors should stand at low levels for causes of deaths that are not easily manipulated by the deployment of low-cost medical technology.

Table 14 also indicates that the contribution of exogenous factors to the changes in total death rate is higher for malarious countries (56.0 per cent against 41.0 per cent). Their contribution to infectious diseases is 20 per cent higher in the case of malarious countries than in the case of nonmalarious countries (61.0 per cent against 51.0 per cent). In the case of diarrhea, the contribution of exogenous factors is 77 per cent higher for malarious than for non-

TABLE 14

DECOMPOSITION OF CHANGES IN DEATH RATES DURING THE PERIOD 1950–1973*

SUBSAMPLE	Total	CAUSE OF DEATH			
		Tuberculosis	IPB	Infectious	Diarrhea
Total changes†					
Total	5.03	0.33	0.70	1.06	0.56
Malarious	6.06	0.34	0.45	1.47	0.62
Nonmalarious	4.51	0.33	0.83	0.86	0.53
Changes to exogenous factors†‡					
Total	2.26	0.05	0.23	0.59	0.19
Malarious	3.39	0.02	0.06	0.90	0.29
Nonmalarious	1.84	0.07	0.32	0.44	0.14
Proportion of total changes due to exogenous factors					
Total	0.45	0.15	0.33	0.56	0.34
Malarious	0.56	0.06	0.13	0.61	0.46
Nonmalarious	0.41	0.21	0.39	0.51	0.26

*There were 7 nonmalarious and 14 malarious countries. Slight departures from strict additivity are due to rounding errors.
†In units of standardized death rates per 1,000.
‡The differences between the entries in this panel and those in the first correspond to contribution of changes in standards of living.

malarious countries (46 per cent against 26 per cent). The opposite relation is found for tuberculosis and respiratory diseases. This figure supports the argument made before suggesting that malaria eradication ought to produce major spillover effects within the category of infectious diseases and diarrhea rather than among other causes of death. Notice that the four selected groups of causes of deaths account, on the average, for not more than one-half of the total change attributable to exogenous factors and that about one-fourth of it is associated with infectious diseases alone.

The exercise undertaken here is merely illustrative and can hardly provide a thorough validation of the hypotheses formulated before. The reason is that an accurate allocation of contribution to changes in death rates to endogenous and exogenous factors cannot be made without having direct measures of *both* of them. The imputation of "unexplained" shifts to the operation of medical technology is an easy way out because it assumes both that the specification of the model is correct and that the errors of measurement are not only negligible but unrelated to the magnitude of such contributions. Furthermore, it masks an important issue, namely the extent to which one can properly separate both effects even if requisite indicators and measures were available. The independence of public health interventions, through which innovations are diffused, from private behavior (household endowments), community level organization and administration, is not a matter to be settled a priori but to be investigated. Although the execution of some programs like those formulated to achieve malaria eradication are certainly more independent from endogenous factors than other interventions (like the elimination of diarrhea), the distinctions are not always clear-cut. The obligated conclu-

sion is that it is likely that the estimated fraction of total change attributed to exogenous factors would be biased even if the model contained all the relevant *socioeconomic variables*. The only way of avoiding such biases is by introducing a systematic treatment of types of technology and their potential relationship to socioeconomic factors.

SUMMARY AND CONCLUSIONS

In this paper, we have tried to illuminate some of the characteristics of the process of mortality decline in Latin America during the years 1955 and 1973. Concentration of our attention to this period has evidently confused somewhat the issues due to the nonsimultaneous character of the mortality decline in each country and, very likely due to the inherent heterogeneity that accompanies nonsimultaneous processes. Also we are limited by the quality of the data. In particular, the deficient classification of deaths by causes tends to distort not only absolute levels of mortality and the proportional distribution by causes but, more important, it helps to conceal genuine trends and thus makes difficult the task of uncovering the factors responsible for mortality changes.

With these limitations, the analysis presented here tends to support the idea that the major contributors to the rapid process of mortality decline were, in this order: infectious diseases, influenza-pneumonia-bronchitis, and diarrhea. Respiratory tuberculosis and other diseases of early infancy are responsible for about 12 per cent of the total decline. Lately there has been an apparent increase in deaths due to cardiovascular diseases and neoplasms. However, we indicated that cardiovascular diseases (probably of infectious origin) may have contributed positively to mortality decline, perhaps as much as 28 per cent of the total decline (net of the effects of changes in the category of "ill defined" deaths). One of the most reassuring findings was the association between the decline in malaria and the concomitant decline in other infectious diseases. This points to the confirmation of hypotheses which attribute substantial weight to medical innovations not so much because of their capacity to directly check a wide variety of diseases but because of the synergism among the diseases themselves.

The source of the changes in mortality are found to correspond in almost equal measure to rising standards of living and to the contribution of exogenous factors: about 45 per cent of the changes between 1955 and 1973 are due to rising standards of living. Although this holds true as an average there is some variation among countries and across causes of deaths. The most remarkable, although anticipated, feature of the decomposition we carried out is that exogenous factors seem to have left a more decisive imprint among countries in which malaria was endemic and within categories of diseases (such as infectious) which are more likely to be controlled without imposing the necessity of substantial transformations in standards of living.

The regularities that we have examined in this paper are the outcome of processes of rather diverse nature. In fact, not only did the mortality transition vary across countries but also the processes themselves were quite discontinuous. Thus, according to all available evidence, the course of mortality decline before 1965 appears to be quite dif-

ferent than its evolution during the second half of the sixties and the seventies. We have neglected the examination of these discontinuities and preferred instead to describe and analyze the "average" of somewhat different processes. It should be emphasized that although our decision of not recognizing completely the contours of time-regions heterogeneity may have served the purpose of maintaining simplicity in the analysis, it may have simultaneously introduced some distortions in the description. A full account of determinant factors, the resolution of some of the puzzles outlined here, and the removal of the uncertainties in the prediction of the future of mortality in Latin America are all issues that will not be resolved unless a more thorough identification of the diversity of a deceivingly homogenous process is completed.

ACKNOWLEDGMENTS

An earlier version of this paper was presented at the Population Association of America meetings in Philadelphia, 1979.

REFERENCES

ARRIAGA, E. E. 1968. New life tables for Latin American populations in the nineteenth and twentieth century. Population Monograph Series, No. 3, University of California, Berkeley.

———. 1970. Mortality decline and its demographic effects in Latin America. Population Monograph Series, No. 6, University of California, Berkeley.

———. 1979. Infant and child mortality in selected Latin American countries. U.S. Bureau of the Census. Government Printing Office, Washington, D.C.

ARRIAGA, E. E., and K. DAVIS. 1969. The pattern of mortality change in Latin America. Demography **6**:223–242.

BOX, G. E. P., AND D. R. COX. 1964. An analysis of transformation. J. Roy. Statist. Soc., Series B **26**:211–252.

BRASS, W. 1975. Methods for estimating fertility and mortality from limited and defective data. Laboratories for Population Statistics, University of North Carolina, Chapel Hill.

CALDWELL, J. C. 1979. Education as a factor in mortality decline: An examination of Nigerian data. Pop. Stud. **33**:395–413.

CHAO, D. 1979. Income, human capital, and life expectancy. Paper presented at the Population Association of America meetings, Philadelphia, Pennsylvania.

COALE, A., and P. DEMENY. 1966. Regional model life tables and stable, populations. Princeton Univ. Press, Princeton, N.J.

DYSON, T. P., C. L. G. BELL, and R. H. CASSEN. 1978. Fertility, mortality and income—changes over the long run: Some simulation experiments. *In* G. Hawthorn (ed.), Population and development. Frank Cass, London.

ESPENSHADE, T. A generalized matrix proof of an adding-up theorem. Ms., 1972. University of California, Berkeley.

FEENEY, G. 1980. Estimation of mortality trends from child survivorship data. Pop. Stud. **34**(1):109–128.

FLEGG, A. 1982. Inequality of income and medical care as determinants of infant mortality in underdeveloped countries. Population Studies, in press.

FLINN, M. W. 1981. The European demographic system, 1500–1820. The Johns Hopkins Univ. Press, Baltimore.

FREDERICKSEN, H. 1961. Determinants and consequences of mortality trends in Ceylon. Public Health Reports **76**:659–663.

GORDON, J. E., M. BEHAR, and N. S. SCRIMSHAW. 1964. Acute diarrheal disease in less developed countries: Three methods for prevention and control. Bull. World Health. Organ. **31**:21–28.

GRAY, R. H. 1974. The decline in mortality in Ceylon and the demographic effects of malaria control. Pop. Stud. **28**(2):205–229.

GWATKIN, D. 1980. Indications of change in developing country mortality trends: The end of an era? Pop. Develop. Rev. **6**:615–644.

LEADERMAN, S. 1955. La repartition das deces de causes indeterminees. Revue de l'Institute International de Statistique **23**:47–57.

MCKEOWN, T. 1976. The modern rise of population. Academic Press, London.

MEEGAMA, S. A. 1967. Malaria eradication and its effects on mortality levels. Pop. Stud. **21**(3):207–227.

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

———. 1970. Malaria control and population growth. J. Develop. Stud. 133–158.

PALLONI, A. 1979. Mortality decline in Latin America. Paper presented at the Population Association of America meetings, Philadelphia, Pennsylvania.

———. 1980. Estimating infant and childhood mortality under conditions of changing mortality. Pop. Stud. **34**(1):129–142.

———. 1981. Current conditions of mortality in Latin America with emphasis on infancy and early childhood. Paper presented at the Population Association of America meetings, April, 1981, Washington, D.C.

PRESTON, S. 1976. Mortality patterns in national populations. Academic Press, New York.

———. 1978. Estimating the completeness of death registration. Population Division, United Nations, New York.

———. 1980. Causes and consequences of mortality declines in less developed countries during the 20th century. *In* R. Easterlin (ed.), Population and economic change in developing countries. University of Chicago Press, Chicago.

PRESTON, S., N. KEYFITZ, and R. SCHOEN. 1972. Causes of death: Life tables for national populations. Seminar Press, New York.

PUFFER, R., and C. SERRANO. 1973. Patterns of mortality in childhood. Scientific Publication No. 252. Pan American Health Organization, Washington, D.C.

RODGERS, G. B. 1979. Income and inequality as determinants of mortality: An international cross-section analysis. Pop. Stud. **33**:343–351.

SCHULTZ, T. 1979. Interpretation of relations among mortality, economics of the household and the health environment. WHO/UN Proceedings of Meeting on Socioeconomic Determinants and Consequences of Mortality, Mexico City. D.F.

SCRIMSHAW, N., C. TAYLOR, and J. GORDON. 1968. Interactions of nutrition and infection. Monograph Series No. 57, World Health Organization, Geneva.

STOLNITZ, G. 1955. A century of international mortality trends: I. Pop. Stud. **9**:20–55.

———. 1965. Recent mortality trends in Latin America, Asia and Africa. Pop. Stud. **18**:117–138.

UNITED NATIONS. 1963. Population Bulletin of the United Nations, No. 6. United Nations, New York.

———. 1973. Determinants and consequences of population growth p. 107–158. United Nations, New York.

WINSLOW, E. A. 1980. The conquest of epidemic diseases. Univ. of Wisconsin Press, Madison.

WORLD HEALTH ORGANIZATION. Epidemiological Statistical Annual. Various issues.

———. 1948. Manual of the International Classification of Diseases, Injuries and Causes of Deaths. Geneva.

———. 1965. International Classification of Diseases. Geneva.