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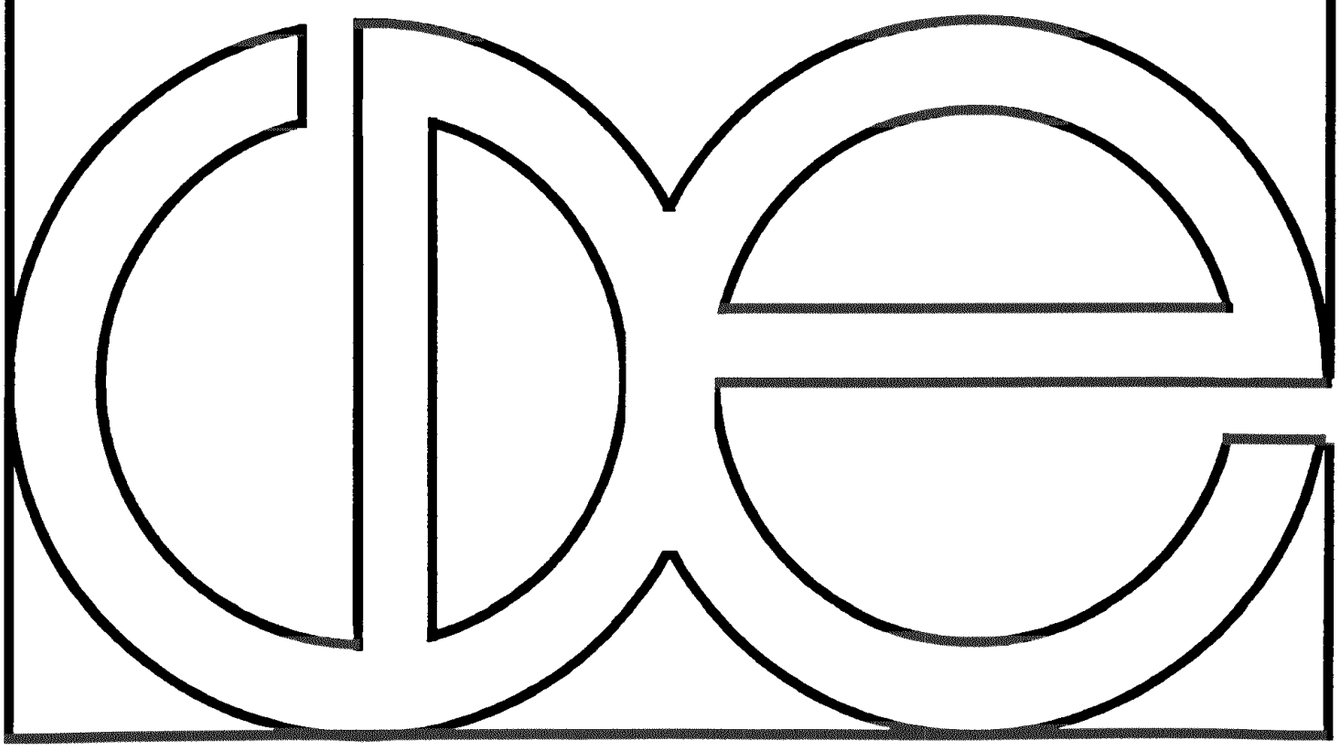
CDE Working Paper 92-22



The Effects of Economic Changes on Mortality by Age and Cause: Latin America, 1950-1990

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This is a revised version of a paper presented at the Seminar on the Demographic Consequences of Structural Adjustment in Latin America, Ouro Preto, Brazil, September 29-October 2, 1992. We thank Jorge Bravo, David Lam, Andrew Mason, Samuel Preston and George Tapinos for useful comments on the earlier draft. The research on which this paper is based was completed while Alberto Palloni was at the Center for Advanced Studies in the Behavioral Sciences, Stanford, California. We gratefully acknowledge support by a grant to the Center by the National Science Foundation (BNS-870084). In addition, research was carried out, in part, using facilities of the Center for Demography and Ecology at the University of Wisconsin-Madison, which receives core support for population research from the National Center for Child Health and Human Development (Grant P30-HD05876).

I. Economic swings and mortality changes.

A fairly recurrent idea of population theory is that the weakening of frequent and sharp fluctuations in the levels and age patterns of mortality is an important marker distinguishing pre- from post-industrialization mortality trends (Livi-Bacci 1992; Vallin 1991; Flinn 1974; Schofield and Reher 1991). The appearance and disappearance of epidemics, the outbreak and aftermath of wars, and the vagaries of weather left clear if not always durable imprints in the profiles of mortality before the middle of the XIXth century. With improvements in technology and standards of living, advances in sanitation and public health and, later during the XXth century, the application of vector eradication and chemotherapy, mortality levels began an apparently irreversible downward trend, while the frequency and duration of oscillations around secular trends were reduced to insignificance or, for all purposes, disappeared altogether.[1]

Although the idea that patterns of mortality of industrialized countries exclude even loose linkages between mortality changes and oscillations of levels of living has been forcefully challenged in the U.S. (Brenner 1983), it is nevertheless well established that the aggregate connection between the two is far from being tight and is undeniably much weaker that it was in the past.[2] The situation in some of today's Third World countries may well be different. Indeed, the context of mortality decline in developing nations differs in many respects from the one that characterized Western Europe and North America. The differences are sharp and leave sufficient room for the possibility that 'pre-industrial' short-run connections between mortality and in socioeconomic conditions are still operating.

One important feature of patterns of mortality decline in some parts of the developing world decline is the fact that, after experiencing unprecedented gains in

survival during the period immediately following World War II, changes proceed at a slower pace than expected (Gwatkin 1980); in some cases the downward trend in mortality has flattened prematurely (Palloni 1981), while in others there have been outright reversals (Muller and Accinelli 1978; Romero 1993; Carvalho and Wood 1988). While the evidence corroborating the details of this process is controversial and there is ample room for disagreement (Hill and Pebley 1989), there is less dissension about the fact that the process of mortality decline in developing countries could be on a fragile and vulnerable course, one that is considerable less insulated from exogenous changes in economic conditions.

A second empirical feature of the patterns of demographic changes in at least **some** developing countries is that they indeed have reproduced the connection between economic fluctuations and mortality that was characteristic of pre-industrial Europe. In a brief review of an admittedly short list of available studies R. D. Lee concluded that "the experience of European populations before the twentieth century is highly consistent with the experience of the poorer Third World countries up to the present" (Lee 1990, p.11). He then went on to add that the patterns of wealthier Third World countries are expected to conform more closely to those of population of the developed countries (e.g., with a lower influence of economic cycles on mortality). But the evidence that we study in this paper suggests that, at least in Latin America, this may well not be the case.

Finally, the protracted and severe economic recession that began late in the 1970s and that lasted in most Latin American countries until well into the 1980s (although until today it shows only weak signs of loosening its grip on large sections of the Third World) may have had social and economic consequences that set it apart from recessions that occurred in the past. Some have argued that its severity and duration

is such that even the most advanced among developing countries were on the verge of experiencing crushing setbacks in standards of living, in health and in mortality (Jolly and Cornia 1984; Cornia et al. 1987).

The evidence is ambiguous, the data insufficient and the methodologies applied are not always unassailable and leave ample room for questionable inferences about whether, when, and where the setbacks have occurred. The task of finding relations is made even more difficult for, as we discuss later, the nature of the mortality response in recessionary times is complex and need not always appear in tidy ways that make self-evident the connection between it and the deterioration of the economy. The same applies **mutatis mutandis** to mortality (or health) responses in periods of economic boom and expansion.

Does the available evidence support the idea of any response whatsoever, in periods of relative scarcity as well as in periods of relative abundance, in at least some areas of the developing world? The question that we attempt to answer in this paper concerns the association between economic fluctuations and mortality changes in Latin American countries during the period 1955-1990.[3] We are interested in elucidating the existence, magnitude, direction, and trends of linkages during a period that witnessed both sustained (but not continuous) economic prosperity - basically the years between 1949 and 1965 - as well as severe economic recession - the period after 1975. We are also interested in determining whether the association between economic oscillations and mortality varies across countries in ways that are consistent with the conjecture that adjudicates similarities between 'poor' Third World countries and pre-industrial Europe, on the one hand, and 'richer' Third World countries and contemporaneous developed countries, on the other (Lee 1990). Admittedly, Latin America is only part of the Third

World and may not be representative of trends elsewhere. Yet its experience is directly relevant to the hypotheses for if they fail to be verified there, they become considerably less relevant in explaining contemporary trends. If, however, the expected patterns turn out in countries of Asia or Africa, the hypotheses should be redefined and elaborated to account for contingencies that make them relevant in some social and economic contexts but irrelevant in others. A non-trivial advantage of our sample is that we are able to assemble a relatively long series of statistics on death by age and by cause, as well as socioeconomic indicators that enable us to test a broad variety of hypotheses.

It should be emphasized that the nature of the question that we address requires that we focus on short-term changes and associations, not on trends over the long run. The fact that post-1945 mortality trends in Latin America have, with one or two exceptions, followed a consistently downward slope while indices of socioeconomic development have, by and large, been on an upward trajectory, is not in itself immediately relevant for our purposes. Neither is the fact that about 50 percent of the secular decline in mortality may have been due to changes in socioeconomic conditions (Palloni and Wyrick 1981; Preston 1976). It is on the strength, direction and variability of associations between **departures** from long term trends that we focus, since they are the ultimate material to confirm or disprove the hypotheses of linkages between mortality and economic fluctuations. This does not imply that we dismiss the importance of (or even the relevance that short-term oscillations may have for) long-term relations between economic conditions and mortality. But in this paper we are able only to focus on the former rather than the latter.

Although our work is not completely original either in its substance or its methods,[4] we deal with well-known and less familiar problems using conventional as

well as experimental and untried solutions. Our results suggest that some of the conclusions that we and others have derived from the straightforward application of conventional time series methods may not be as solid as originally believed, that there are some persistent and intriguing regularities in all the countries that we examine, and that there is much more to be discovered with the data already available, provided that appropriate methodologies are judiciously applied.

The plan of the paper is as follows. In Section II we define alternative mechanisms linking economic fluctuations and mortality, and discuss a series of relevant hypotheses. We emphasize here that the nature of these mechanisms requires investigation of changes in mortality by age and cause, not just in total levels. In Section III we summarize the data and describe the methods applied to generate estimates of the magnitude, direction and variability of effects. In the main, the methods we employ follow closely those developed elsewhere for the analysis of short term fluctuations (Lee 1981; Galloway 1988; Bravo 1992; Hill and Palloni 1992). However, we introduce a few improvements that enable us to deal with causes of deaths, to generate seemingly stable and robust estimates of elasticities, to estimate changes over time in the magnitudes of the linkages and to control for the influence of fluctuations in the accuracy of reporting of causes of deaths. In Section IV we discuss our results and investigate their implications. In particular, we show that estimates of linkages between economic fluctuations and mortality changes by age and cause can be translated into estimates of changes in the overall level and pattern of mortality.

II. Mechanisms linking mortality changes and economic fluctuations.

A. **The effects on exposure, resistance and recovery.**

The relation between deterioration of economic conditions and mortality is mediated by changes in exposure and resistance to diseases and other mortality risks, as well as by the capacity of individuals to recover from ailments produced by illnesses or other health threatening conditions. A direct, non-mediated relation may be observable only in situations leading to outright starvation or to acute deficiencies in major nutrients (proteins, vitamins, and minerals) and their sequelae. The mechanisms through which exposure, resistance, and recovery are affected by economic conditions are numerous and vary by age and sex.

First, a drop in individual standards of living can translate directly into lowered nutritional intake and this in turn, if sustained long enough, can lead to deteriorated nutritional status. Deficiencies in nutritional status compromise immuno-competence and render the host more susceptible to some infectious diseases, while at the same time weakening its ability to ward off the effects of illness and erode its capacity for recovery. Nutritional status has been shown to strongly influence susceptibility and resistance to about ten infectious diseases including cholera, bacterial diarrhea, measles, respiratory tuberculosis, whooping cough and respiratory diseases. Other infectious diseases show a less clear, more variable relation to nutrition (Lunn 1991; Rotberg and Rabb 1983). Of particular importance are diseases associated with bacterial diarrhea and dysentery. In this case a synergistic relation exists whereby the infection weakens nutritional status even when nutritional intake is adequate, as the host's ability to absorb nutrients is impaired by the disease (Scrimshaw et al. 1968).[5] The population groups that are most vulnerable to nutritional status deficiencies are young children, the elderly, and women

of reproductive age. Infants who are fully breastfeeding are better protected by the cleanliness of breastmilk and by the immunities and nutrients transferred from the mother to the child. The benefits of lactation may last for as long as one year. At higher risk are children who are weaned early, and those who begin to be fully exposed to a host of pathogens and must rely on regular food intake to satisfy nutritional requirements. Women of reproductive status experience the burden of pregnancy and child feeding, both of which place heavy demands on their nutritional status. In societies where child spacing is typically short, the maternal health response to falls in levels of living standards will be exacerbated. However, since diseases such as pulmonary tuberculosis are more likely to strike women who experience pregnancy as a result of compromised cell-mediated immunity (Larsen 1983), one would expect that deteriorated social and economic conditions will have effects over and above those due to maternal depletion.

The observed connection between nutrition and survival, however, is likely to be exaggerated by the influence of other factors associated with sanitation, personal hygiene, poor housing, etc. In a recent review of the evidence, an author has remarked that while "we may agree that malnutrition does play its part in worsening the conditions of survival in the presence of other factors which favour the spreading of infections and act against their cessation (poor hygiene, poverty, ignorance)...the role of malnutrition is not uniform, being nil for some diseases, uncertain and variable for others, considerable and certain for still others" (Livi-Bacci 1991). This cautionary note is echoed in a review of the relations between nutrition and infection which suggests that "although nutrition plays a major part, the extent to which other socio-economic variables contribute to disease prevalence and mortality remains to be assessed" (Lunn

1991; see also Martorell and Ho 1984; McKeown 1988). Although spurious effects may be part of the observed relations, it is undoubtedly the case that the negative effects of nutrition on survival are **enhanced** by conditions of poverty. Thus, mortality responses will be more discernible among social groups living in precarious conditions. It is this expected (individual-level) relationship that leads to the conjecture according to which responses to economic crisis are probably attenuated in countries with higher standards of living.[6]

Second, a lowering of standards of living results in conditions and behaviors that increase exposure to diseases and that minimize the ability to recover from bouts of illness. First, individual or family reactions to the crisis could aggravate crowding, housing, and general hygiene conditions, thus providing a strong link between economic conditions and mortality. This is likely to occur, for example, when deterioration of economic conditions leads to rural-urban migration or to massive displacement of people between urban area. But these factors are only part of the story. In a recent review of the relations between nutrition and health, Livi-Bacci (1991) finds that observations from clinical studies, the historical experience of selected population groups, and the historical record of Western Europe all support the idea that the apparent response of mortality to reduced intake of food may be confounded by simultaneous outbreaks of epidemics (see also Braudel 1985). Since the evidence linking malnutrition and incidence of infectious diseases is not strong, the sharp increase in mortality that appears in response to economic crisis can only be explained successfully by social behavior that, in response to the crisis, augments the exposure to diseases. This is an observation which is pertinent in developing countries where the occurrence of significant increases in exposure (and lowered capacity to recover) is likely to require far more than transient

reductions in food intake. It probably necessitates serious deterioration or complete breakdown of infrastructure and considerable inaction, or outright paralysis of national and local governments. Consequently, mortality responses will be magnified if the downturn is sharp and sustained enough to inflict damage on sanitation and public health, the continuity of public work programs, food assistance and subsidies, and on the integrity of social welfare including preventive and curative health services. The partially protective shield conferred on citizens by centralized institutions is thus of fundamental importance as it may constitute a mechanism through which some of the effects of economic downturns are postponed or are simply never felt. This is not unique to developing countries. Indeed, there is evidence that in pre-industrial societies central governments were oftentimes successful in anticipating shortages and, accordingly, smoothed oscillations in food inventories through corrective interventions (Fogel 1989).

The most direct outcome of both individual and family accommodation to the crisis and deterioration of infrastructure is an increased exposure to infectious diseases. Living in crowded and cramped quarters as families double up to cushion the impact of poorer economic conditions, lack of access to uncontaminated water and food, and substandard disposal of refuse and sewage may all multiply exposure to transmittable diseases. First in line to be affected are diseases of the intestinal tract, respiratory ailments, and respiratory tuberculosis.[7] Furthermore, if governments' ability to support a network of health services delivery is undermined, families' access to vaccination, prevention, and proper medical attention will be compromised, thus simultaneously worsening exposure and threatening individuals' ability to recover from bouts of illness. Under these conditions, increases in respiratory tuberculosis among young adults and

older people, in respiratory ailments among the very young, and in the incidence of other infectious diseases (particularly bacterial diarrhea and dysentery) among young children and the elderly are to be expected. If the impaired ability to deliver health services involves prenatal care, infants are likely to be seriously affected as well.

A less direct effect is on exposure to conditions and behaviors that bolster the risk of non-infectious diseases. Consumption of alcohol and other drugs may increase,[8] adults may become more likely to engage in work conditions that entail higher risks to accident,[9] and added stress will augment susceptibility to chronic ailments, self-injury, homicide, and suicide. These conditions will affect most seriously young male adults and the elderly and should have virtually no effects among the very young, unless increased poverty and lack of access to resources forces families and women to cut down on safe child care practices.

To conclude this section we remark that the influence of economic downturns and upturns may operate anywhere in the chain from exposure to recovery. It is conceivable though unlikely that, under current conditions in developing countries, bad economic times affect both exposure and resistance but leave unscathed the capacity to provide treatment and aid recovery. This means that we should search for responses not in levels of mortality but in levels of morbidity. A muted response in mortality is ambiguous since it can mean one of two things: either economic crises simply do not affect health status in any significant way, or they affect it significantly but without undermining the capacity to bounce back from episodes of illness and thus without substantially altering mortality risks.[10]

B. **Timing and contingencies: who is affected by what and when?**

B.1. *Ages and causes of deaths.*

Almost all studies of the relation between socioeconomic fluctuations and mortality focus on total mortality (for an exception to this see the work by Bravo 1992). It should be clear from our previous discussion that the effects could vary across age groups and causes of deaths, and that the observed changes in total mortality may mask very different responses by age and causes of deaths. Thus, proper identification of effects calls for separate treatment of mortality by age and cause:

i) causes of death that are more closely connected to nutritional status (such as malnutrition and avitaminosis, bacterial diarrhea and dysentery, some infectious diseases such as measles, acute respiratory ailments and respiratory tuberculosis) are more sensitive to changes in socioeconomic conditions and among young children, young adults and the elderly. Infants should not be affected by this mechanism unless there is severe disruption of feeding patterns or unless the prevailing patterns of breastfeeding are not long and universal.

ii) accidents, suicides, homicides and some chronic ailments (such as cirrhosis, ulcers and ischemic heart disease) are likely to be the responses among adults.

iii) in the absence of changes in feeding patterns, infant mortality will be affected to the extent that pre and postnatal care deteriorate. Paradoxically, as a crisis may increase the incidence of stillbirths, a stronger selection of weaker newborns will operate and neonatal and possibly postneonatal mortality could be **favorably** affected by the crisis, although the effects will be short-lived.

B.2. *Time lags and direction of effects.*

The timing and direction of the expected effects are more difficult to pin down

with precision. First, effects operating through nutrition should lag by at least one year, except under wretched initial conditions. Shorter lags should be expected for infants if the patterns of breastfeeding are severely disrupted. Second, effects associated with increases in neo and postnatal mortality are likely to occur within one or at most two years of the initial changes. Increases in respiratory ailments are unlikely to take place before a year and increases in death due to respiratory tuberculosis will not be observed before two years.[11] Finally, effects associated with accidents, suicides and homicides, and chronic ailments are more likely to operate in full force after there has been some time for the consequences of the crisis to be felt at the individual level.

The effects of downward economic swings will not always be in the direction of increasing mortality. We have already noted that the changing composition of births may indeed induce lower, not higher, infant mortality as a higher proportion of those whose prenatal health status is substandard are stillbirths. A similar argument applies to young and adult mortality: after the initial response the composition by frailty of the surviving population will lead to lower than normal average mortality risks. Thus, the increase in mortality above normal levels soon after an economic downturn should be followed by a decrease below normal levels (Caldwell and Caldwell 1987; Palloni 1988). This is exactly the pattern observed in the majority of the empirical studies that have been carried out so far (Lee 1990): the lag-pattern of effects is wave-like, displaying a sinusoidal profile that alternates negative with positive responses whose magnitude gradually converges toward zero as the lag increases. But the nature of the pattern of effects by lag is partly dependent on the composition by frailty of the initial population. Groups who are at the lower end of the frailty scale will definitely show it, whereas those who are better off may experience multiple (but decreasing) echoes of the mortality

increase as people who are weakened during the initial stages of the response are never able to fully recover. We expect that the wave-like pattern will have different profiles by age and by cause of death. Selection effects should be stronger among the very young and the very old than among younger adults. The implication is that the echoes will be more salient for young children and the elderly. Similarly, selection effects should be stronger for wasting diseases such as respiratory tuberculosis than they are for other infectious diseases. Finally, selection may operate in unexpected directions and produce reinforcing rather than offsetting responses, as when an increase in death through violence among the most fit segment of the population increases the average frailty of the subpopulation that is less exposed to violence.

A potentially powerful selection effect could operate indirectly through effects on fertility. It has been shown that both marital fertility and nuptiality respond quite readily to economic fluctuations (Galloway 1988; Lee 1981; Reher 1989; Hill and Palloni 1992; Palloni, Pinto and Hill 1993). In particular, the number of births drops significantly in response to the initial impact of a recession. Since the composition of births by risk factors such as parity (proportions of first births), mother's age (proportion to younger mothers), and birth interval (proportion born after exceedingly short intervals) is likely to change favorably, infant and even early child mortality will tend to decrease, thus partially offsetting the increases due to other forces directly affecting mortality. As the fertility response wanes and the original composition of births is restored, infant mortality will increase at higher lags.

A general conclusion to be drawn from the discussion in this and the preceding subsection is the following: as conjectured before, one important consequence of the differentials in the timing, direction and magnitude of responses by causes of deaths and

age-groups is that the observed effects of economic fluctuations on total mortality could mask important variability in its components. This holds generally except in cases when there is only one single dominant mechanism producing the mortality response. Indeed, the absence of an observed mortality response may be the result of a perverse pattern of age-cause specific responses by lags. As we show later, our data do reveal the presence of important potentially offsetting effects, though we found no example of the extreme case of fully offsetting effects.

B.3. *Social and behavioral contingencies.*

Heterogeneity in the nature and duration of the crises, the social and political conditions that prevail in a country during the crisis, and the mixture of social groups and their bargaining power are important sources of variation in the magnitude and even direction of the mortality response (Hill and Palloni 1992).

Not all economic downturns (upturns) have the same effects even though they may be reflected in similar empirical fluctuations of economic indicators. Recessions that have their origins in international crises and that lead to draconian reorganization of internal consumption patterns, massive loss of purchase power, and substantial cuts in government spending will hit the urban working class and lower white collar occupational groups more severely than those whose income depends on rural wage labor, subsistence agriculture, or on activities associated with large export sectors. Economic downturns that are more localized, less influenced by international recessions, and associated with sagging demand for selected exports, for example, will have a less serious and immediate impact. As has been shown elsewhere, the recession currently affecting Latin American countries belongs more to the first class and is one of many others of the same type that have hit the continent with some recurrence since the times

of independence (Marichal 1989; Altimir 1984; Frieden 1991).

A related though distinct factor that affects the magnitude of the response is the duration of the downturn (or upturn). Protracted crises are more likely to exhaust reserves and inventories or to outlast the shielding effect of public interventions. Longer exposure times are also more likely to trigger effects that require thresholds to effectively lead to heightened mortality (for example, malnutrition). If a long series including the occurrence of multiple crises were available to us, one could test for the effects of crises of different durations. As we show later, however, our data set does not allow detailed tests of this nature and instead we are forced to use an indirect albeit fairly crude assessment of duration effects: since the analysis covers only a very restricted period of time, we estimate the existence of differential effects over time by modelling the effects of pre-1975 recessions in an attempt to assess the uniqueness of the most recent one.

Given the nature and duration of the crisis, different social groups and economic sectors will be unequally affected by it. Some groups and sectors will be more insulated from its main effects, others will be able to adapt and accommodate using survival strategies of different caliber and, finally, others will resist erosion of standards of living by mobilizing and successfully applying political pressure on decision makers. Convincing evidence of social differentials in the impact of the most recent crisis in Latin America has been obtained from a series of surveys in selected urban areas and analyzed at length elsewhere (Altimir 1984). As Frieden (1991) has forcefully argued, the impact on different social groups and economic sectors is largely a function of their actual position in the national economy and vis-a-vis the state, of their interrelations with elites and of the degree and type of class conflict. The long time series by social groups that are required to assess the nature of these differentials are generally unavailable although

glimpses of it and suggestive evidence illustrating their presence can be obtained from case studies (Frieden 1991) or from cross-sectional surveys (Altimir 1984). The issue is important, for just as the observed responses of total death rates conceal the detail of age and cause specific processes, so do population-based responses mask group-specific dynamics.[12]

In the absence of information on social groups we test hypotheses at more aggregate levels regarding two possibly important contingencies. First, when crises are of roughly comparable magnitudes the response should be more muted in countries where the mortality decline starts earlier, has been more sustained, and is better entrenched, than in others where mortality decline is more recent and more dependent on newer institutions and programs. This is because the former are more likely to have a more adequate infrastructure for the delivery of services so that health education, sanitation, and public health can efficiently reach a majority of the population. It should be noted that, although the correlation is not perfect, countries of the former type are those that are higher up in a continuum of development. Thus, if empirically verified, the expected differential effects will probably coincide with differentials in the levels of development as well.

Second, the effect on infant mortality was postulated to be mediated by practices of infant feeding. Among groups that rely less on breastfeeding, an economic downturn may have an immediate impact on child morbidity and mortality, whereas among those where there is universal and prolonged breastfeeding (more than one year) the effects will be attenuated. An analogous though admittedly less persuasive contrast can be drawn at the more aggregate levels of countries, namely, in countries where traditional breastfeeding patterns prevail mortality levels will be less affected.[13]

III. Data and Methods.

A. **Description of the data.**

A.1. *Assessing economic fluctuations.*

In pre-industrial societies annual fluctuations in the price of grain were the most dominant determinants of variations in the real wage and could be taken, with some important qualifications, as good indicators of fluctuations in families' standards of living (Galloway 1988; Lee 1981; Weir 1984; Livi-Bacci 1991; Meuvret 1946; Corradi 1973; Appleby 1979). In contemporaneous Third World countries with fairly diversified and open economies the prices of one or a combination of commodities and the real wages (derived from it and from nominal wages) are unlikely to be a good gauge of individual standards of living. Diversified production and individual consumption renders futile the attempt to single out a combination of price of staples as indicator of household budgetary pressure. Real wages are not always available and, furthermore, they represent the experience of a variable subset of the population. In addition, fluctuations of real wages are heavily influenced by the strength and fortunes of segments of the working class and their political organizations and may trace the movements of the economy only poorly. Elsewhere we have argued that a good indicator of standards of living is the level of personal consumption (Hill and Palloni 1992). Due to the nature of the current crisis which is associated with high levels of international indebtedness, this could be a compelling choice. Yet, as shown in Figure 1, the variability of this indicator is less than that of other candidates such as GDP (and GNP) and about the same as that of the ratio of debt servicing to exports.[14] As GNP and GDP do include non-personal consumption elements such as investments and exclude borrowing to sustain individual levels of consumption, they may in theory be more weakly related

than personal consumption to the mechanisms that trigger a mortality response. But, on the other hand, GNP and GDP include elements intimately related to the maintenance and functioning of infrastructure, sanitation, and public health that are excluded from personal consumption but are central to our argument. In some of our analysis at least we use real GDP per capita and private consumption separately and show that despite some differences, the inferences are quite similar.[15]

Conventional economic indicators may not reflect well the timing and nature of hardships generated by recessions in Latin America. Indeed, either as a response to the protracted current crisis or as a broader and more entrenched adaptation to long-term economic stagnation, many Latin American countries have experienced the growth of an informal sector that absorbs a significant fraction of the labor force, occupies massive amounts of resources and capital, and satisfies a demand for products and services that the formal sector of the economy does not address at all (see DeSoto 1990). For the most part, the indicators of economic activities from national accounts do not reflect transactions and production in the informal sector although, in principle at least, prices and wages should reflect the value of all goods and services. Worse yet, if the activities in the informal economy intensify precisely during pronounced downturns, the indicators we have chosen will lead us astray. This problem is analogous to one faced by historical investigations that focus on price trends of staples when a substantial fraction of food resources is not purchased by means of market transactions but originates in subsistence activities, bartered, or received in exchange for services and labour.[16]

A.2. *The data on causes of deaths.*

Although data of both mortality and morbidity would have been desirable, the available information on the latter is spotty and unreliable. As indices of mortality we use detrended values, namely, the ratio of the observed to the predicted *number* of deaths in the total population and, in age groups 1-4, 5-14, 15-64 and 65+, classified by causes of deaths (see Appendix 1). As argued elsewhere (Lee 1981) the analysis of short term fluctuations can omit reference to denominators of rates when these change only gradually over the period of time considered. Detrending also minimizes the effects of gradual changes affecting completeness of death registration.[17] The case of infant mortality is different since the number of infant deaths is a function of the number of births, which itself is responsive to economic oscillations. To reduce the impact of this second order effect we use the ratio of infant deaths in a calendar year to the weighted number of births during that year and the immediately preceding one.

An important problem is that the time series by causes of deaths reveals discontinuities that are the result of changes in the classification of causes, idiosyncracies of practices and routines applied by national statistical offices or, more importantly, empirical variation in the probability that deaths of a given cause will be classified as ill-defined. The groups of causes of deaths that we define in Appendix 1 are a compromise between what is of substantive interest (see B1 and B2 in Section II) and what enables us to minimize distortions.[18] In the section on methods we suggest a procedure that should minimize the effects of distortions due to changes in the propensity to classify deaths in ill-defined category.

Our final series of mortality and socioeconomic indicators is for 9 countries (see Appendix 2) and, for the most part, covers the period 1955-1990.

B. **Methods.**

The most general form for the model we propose is the following:

$$y_{nk}(t) = \alpha_{nk} + \Sigma_j \beta_{nk,j}\, x(t-j) + \Sigma_r \Sigma_j \lambda_{nk,jr}\, z_r(t-j) + \varepsilon_{nk}(t) \qquad (1)$$

where $y_{nk}(t)$ and $x(t)$ are, respectively, the detrended values of the indices of mortality for cause k and age group n and of economic levels evaluated at time t, $z_r(t-j)$ is the lagged value of the rth control variable evaluated at time t-j, β's and λ's are regression coefficients, and $\varepsilon_{nk}(t)$ is an error term following an autoregressive process to be specified later. Under these conditions, the β's can be interpreted as elasticities, e.g., they are proportionate changes in the mortality indicator attributable to a proportionate change in the socioeconomic indicator. Adding the β's across all lags yields the total (net) effect generated by a proportionate change in the economic indicator (s).

The model in (1) is not well-defined, however. Several factors need to be discussed in order to completely specify it.

B.1. *Detrending.*

Although detrending procedures have followed fairly well established conventions involving the calculation of centered moving averages of variable length (Lee 1981; Galloway 1988; Hill and Palloni 1992), they are quite costly in terms of degrees of freedom, particularly for short series like those available to us, which include at most 35 observations. An 11-year moving average is considered desirable and is perhaps ideally suited for relatively short series (not exceeding, say, 200 hundred years). But it would eliminate 10 observations from the outset and if the maximum lag is fixed at 5, we would be left with only 26 observations. Reducing the length of the moving average

is possible but at the cost of making the detrending procedure more vulnerable to the variability we are trying to remove. An alternative procedure is to fit a trend to the data and then use the predicted values as denominators for detrending (Bravo 1992). However, this requires a functional form for the trends and the choice of functional form is not always empirically obvious or unequivocally dictated by theoretical considerations.

A solution that combines the best of the previous two procedures without being hindered by their respective drawbacks is to use local least squares, a technique that provides a robust fit of the data without imposing a *global* functional form. Indeed, the resulting trend is retrieved by locally fitting robust straight lines to portions of the data (Cleveland 1979). This is an optimal strategy: it utilizes all points without deleting information and, simultaneously, does not force a *single* parametric form to the data.[19]

B.2. *Controls.*

The controls refer to variables that could have an effect on the nature of the response of mortality. In our country-by-country applications $z_r(t)$ refers to either a dummy for time-location that retrieves changing effects over time, or to the detrended value of deaths to ill-defined causes to neutralize possible distortions due to misclassification of deaths by causes.

i) Retrieving changing effects.

To model changes in responses over time we assume that $\beta_{nk,j}$ is time dependent and that its value can be broken down into two components, one that is time-invariant and one that depends on time, that is, $\beta_{nk,j}(t) = \delta_{nk,j} + \phi_{nk,j}{}^{*}w(t)$, where w(t) is a dummy variable which attains the value 1 if $t < t_o$ and 0 otherwise. Substituting this in expression (1) yields

$$y_{nk}(t) = \alpha_{nk} + \Sigma_j \delta_{nk,j} x(t-j) + \Sigma_j \phi_{nk,j} x(t-j)*w(t) + \varepsilon_{nk}(t) \quad (2)$$

depending on x(t-j) and z(t-j)= x(t-j)*w(t). In this paper we set t_o to be equal to 1975 in the belief that the recession that began after 1975 elicits responses that are different from those that occurred before. This is a simplified representation of another stochastic process - the one generating the effects or 'parameters' being estimated. It should enable us to test the hypothesis that more or less 'normal' cyclical recessions (those occurring before 1975) are conducive to qualitatively different responses than deeper recessions (after 1975).[20]

ii) Neutralizing the effects of misclassification of deaths.

Spurious fluctuations of causes of deaths due to changes in the category of 'ill-defined causes' are likely to occur. In theory at least, the proportionate change in the number of deaths due to ill-defined causes must be totally accounted for by a proportionate change in the frequency of all other causes of deaths. If periods of hardship have a non-trivial effect on the accuracy of recording of cause of deaths, we would expect that the ill-defined category be sensitive to economic downturns and that the estimated response of deaths due to well-defined causes be underestimated. To partially correct for this we include a control for the (detrended) number of deaths assigned to the category 'ill-defined' at time t. This is not an optimal solution but should, except under warped conditions, produce unbiased estimates of responses. A justification of this is in Appendix 3.

B.3. *The autoregressive process and the lag structure.*

The nature of the autoregressive process governing the error terms needs to be defined. Two types of processes have been estimated in the literature: a first and a

second order autoregressive process. However, we have found no compelling argumentation anywhere that would justify choosing one over the other. For the most part the selection is made to preserve comparability with what others have done rather than to represent processes inherent in the data being analyzed. In this paper we chose a first order autoregressive model for purely heuristic reasons. Second order autoregressive processes are more appropriate with data that represent very fine units of time (months or quarters), a situation where in-built dependency is more likely to retain some inertia. Also, our series are not long enough to withstand the data demands required to identify second-order autoregressive processes. Indeed, in most cases where we experimented with the second order autoregressive process, we were unable to consistently find convergence in the iterative process.

The nature of the lag structure that we estimate is also dictated by a combination of heuristic and theoretical considerations. Although some of the hypothetical responses might take longer, we set the maximum lag to 4 completed (5 exact) years. This is the same lag structure used in previous work in Western Europe (Lee 1981; Galloway 1988) and involves one extra lag relative to our previous work (Hill and Palloni 1992). All past research indicates that lags beyond the fifth are of no importance whatever.

B.4. *Handling multicollinearity.*

A problem that models with distributed lags encounter frequently is the high level of collinearity between the variables representing the various lags. This is probably more serious in contemporary developing countries than in pre-industrial societies, for the series of economic indicators that we use are quite stable despite sharp fluctuations. The result of this inherent stability is that the detrended values of a series will incorporate a fair degree of cross-lag collinearity. This will result in inefficient and

unstable estimates of effects and may lead to the appearance of a pattern of effects with alternating signs as one moves from one lag to the next.

There are many solutions to this problem but none of them is entirely satisfactory (Judge et al. 1985). In this paper we first estimate models with all pertinent lags and then suggest a simplified technique that enables us to reduce the parameter space, increase parsimony, and reduce instability while simultaneously preserving the central features of the lag distribution of effects. In particular, we will assume that responses across lags follow a 'triangular' pattern with effects reaching a maximum at lag two and decaying to 0 at lag 0 and 4.[21]

IV. Analysis.

A. **Results from basic models.**

A.1. *General Patterns.*

The estimated elasticities, adjusted R-Square and flags for statistical significance are displayed in Table 1 (Tables 1a through 1f). Each of these tables applies to one age group. We have included information for all age groups and for causes of deaths with regular patterns that were more immediately relevant for the testing of hypotheses.[22] Table 2 is a compact representation of the patterns uncovered in the more massive Table 1. This table shows the frequency of significant coefficients by lag and groups of causes of deaths. Table 3 is a simpler distillation of Table 2, and Table 4 is a stricter synthesis of the results in Table 1 describing the **net** value of the responses across lags.[23] Table 2 and its summary in Table 3 reveal several interesting features.

First, responses that are statistically significant are somewhat infrequent, even with the rather liberal criteria used in the table.[24] Thus, for example, in panel A of

Table 3 we see that for lag 2 the proportion of estimated responses that were negative ranged between .29 and .50, and those that were significantly positive ranged from .03 to .11. Other lags reveal responses that are in closer in agreement with our expectations. The age groups where most of the negative and statistically significant responses occur are 0, 1-4, and 65+ (in that order) followed closely by 15-64. Panel B of the same table highlights the patterns of responses by causes of deaths and age regardless of lag. This panel shows that infectious diseases, diseases of the respiratory system, and ill-defined causes respond more frequently in the expected direction. The panel also helps to signal the role of two sets of infectious diseases whose responses stand out above all others: respiratory tuberculosis and diarrheas.

Second, there is some evidence in Table 4 to support the idea that responses to economic changes do exist and that, by and large, they are in the expected direction and follow the anticipated age pattern. Note that with few exceptions, in all countries and all pertinent age groups the net effects on infectious and respiratory diseases and on diarrheas and respiratory tuberculosis are negative and quite substantial. But neither the net effects nor the sum of negative effects reach statistical significance very often. By and large, the sum of negative effects becomes statistically significant only for all infectious diseases, respiratory diseases, and respiratory tuberculosis and diarrhea.

Also, the age pattern of effects is consistent with expectations. However, although the largest effects take place in the age groups 1-4 and 65+ as we suspected they would, there are also somewhat unexpected strong effects at age 0.

Are the patterns of breastfeeding associated at all with responses at age 0? It should be noted that the effects at age 0 are lowest (or slightly positive) in Guatemala, Ecuador, and Panama. As it turns out, the median length of breastfeeding in Ecuador

and Guatemala hovers around 13.0 months whereas in Mexico, Panama, and Costa Rica it is 6.7, 3.7, 1.8 respectively. Although no national estimates are available for Chile and Uruguay, it is known that they are at the lowest end of the spectrum. Thus, excluding the case of Panama, the conjecture formulated at the outset about the effects of patterns of breastfeeding on the overall responsiveness of infant mortality appears to be borne out by this association. However, as this is only aggregate evidence, its relevance for the testing of the hypothesis should not be overplayed.

Is the conjecture that there is a relation between the magnitude of the responses and the countries' levels of mortality supported by the data? Figure 2 shows plots of levels of life expectancy during 1980-1985 and the net response for selected causes of deaths. Although in all cases the direction of the association appears to be as expected, none of the graphs exhibit a sufficiently tight association to support robust inferences. Excluding the most deviant cases from each plot leads to somewhat tighter and stronger relations in the cases of all deaths, deaths due to infectious diseases, deaths due to ill-defined causes and, more importantly, deaths due to respiratory tuberculosis.

Is the lack of an overwhelming presence of statistically significant effects decisive for our conclusion? Although the fact that only nearly half of all responses are statistically significant is indeed limiting, the **patterns** we have retrieved and those that we discuss in the following section are simply too regular to be dismissed. It is important to note that during the period under study there is essentially only one important crisis that dominates the historical landscape (Hill and Palloni 1992). As Marichal has pointedly remarked, "[D]uring the three decades that followed World War II, there were numerous economic crises in different Latin American nations, but there was no major financial upheaval that shook the entire region..." (Marichal 1989, p. 231).

Recessions may have punctuated the history of countries everywhere during this period but none of them in isolation was a large enough departure from the secular trend to trigger mortality responses of significant relevance and durability. It is only after the mid-1970s that the specter of genuine economic crisis looms large. Supporting this statement, Figure 3 displays for selected countries a plot of detrended GDP and detrended total deaths. The plots show the graphic profile taken by a succession of minor crises followed by a major one. Note that, with the exception of the early part of the 1950s and 1960s for Venezuela, there is a clear negative association between the course followed by the GDP series and the series of total deaths. Note also that in all cases, the downturn of GDP is considerably more pronounced after 1970.

All cautionary appeals notwithstanding, the fact that we are able to uncover systematic, recurrent patterns of effects that agree broadly with our hypotheses is probably more important than their statistical significance within the confines of a single experiment. We now examine more closely the nature of such patterns.

A.2. *The lag-age-cause pattern of effects.*

We begin by studying the box-plot in Figure 4 that shows in a synthetic graphic form the same data contained in Table 4. There are three features that deserve to be mentioned. First, **regardless** of the age group, the net response of infectious and respiratory diseases is always predominantly negative, as expected. Furthermore, for the last three age groups, the negative response of respiratory TB and diarrhea is uniformly negative. Second, the variability of responses is higher at the lower end of the age spectrum and for deaths due to chronic illnesses, violence, and ill-defined. The effects on respiratory TB and diarrhea exhibit least variability. These two features strongly imply a third one, namely, that the observed total net response conceals a considerable

amount of age-cause specific heterogeneity in responses.

An important feature of our results is better displayed in Figure 5 and its various panels. In this figure we plot the median response by lag and by age groups. In the first panel the median total response (all deaths for the entire population) reveals a topography which is similar to the median from various estimates for developing and developed countries calculated by Lee (1990). To facilitate comparisons, we show those patterns in the same panel.[25] In Latin America, the response at lag 0 is followed by one of lower magnitude at lag 1, an inverted response at lag 2, and then more muted reactions bearing negative signs and converging to 0 for lags 3 and 4. In the pattern calculated by Lee, the absolute magnitude of the responses is larger, the reversal occurs earlier (lag 1) and, when they subside, the effects change from positive to 0 (instead of from negative to 0 as in Latin America).

The similarity in the profiles of the patterns, particularly in Latin America and pre-industrial Europe, is quite remarkable in view of the fact that the indicators used by us (real GDP) and by Lee (prices) are quite different. Furthermore, it could well be that the differences that remain are due to the relation conjectured before, namely, that societies at the extremes of the mortality spectrum should show different response patterns. To provide a partial test of this we have plotted the median of the total responses in Uruguay, Costa Rica, and Chile - the three countries with the highest levels of life expectancy - and contrasted them with those of Guatemala, Ecuador, and Mexico - the countries with the lowest levels of life expectancy. The plot appears in the second panel of Figure 5. If the conjecture were correct we should see that responses in the first group of countries cluster close to the European pattern. The comparison is facilitated by the fact that the pattern in low mortality countries is somewhat flatter (as is the one

found in Western Europe) than the one for high mortality countries. But despite similarities between the low mortality Latin American countries and Western Europe, they remain distinct whereas the patterns of response of low and high mortality are not easy to tell apart.

The third and fourth panels of Figure 5 display the median patterns of responses by age, and partially confirm the heterogeneity that was hypothesized before: strong responses are found at ages 1-4 and 65+, and less in the age group 15-64. There is always a wave-like pattern by lag with a maximum response reached at lag 2, reversals at lag 3 and then gradual reductions toward 0. The figures confirm that sharp responses (with similar lag-patterns) also occur at ages 0 and 5-14 and, furthermore, suggest that the strongest reaction takes place between 0 and 5 or between 0 and 15 rather than at the older ages.

The advantages of disaggregating responses by age and causes are made evident in the two panels of Figure 6. The graphs display the median response for selected causes of death for each of the age groups considered. The patterns by lags are not the same across causes (holding constant age). Thus, for example, respiratory tuberculosis displays steeper responses than infectious diseases and peaks at lags 2 and 3 rather than at lag 0 as infectious diseases tend to do. By the same token, the patterns by lags are not the same across ages (holding constant cause of death). For example, the elasticities for diarrheas are stronger for ages 0 and 1-4 than they are for all other age groups.

A.3. *The responses of respiratory TB and diarrheas.*

Newberne and Williams (1970) remark that there are four ways in which malnutrition influences infection: "(1) by effects on the host which facilitate initial invasion of the infectious agent; (2) through effects on the agent once it is established on

the tissues; (3) through effects on secondary infection; or (4) by retarding convalescence from infection." The groups of causes that we labeled 'diarrhea' include some of the illnesses that are known to be very sensitive to nutritional status, partly because of suppression of immunocompetence (factors 2 and 3 above) and partly because of synergism that aggravates nutritional deficiencies present at the outset (factors 2 and 4 above). Furthermore, for the most part, the illnesses in this group have proven connections even with **mild states** of malnutrition (McKeown 1988). Although the link forged by nutritional status is strong enough to dominate the relation between diarrhea and economic downturns, there is an additional source of association: deterioration of hygiene and services (water, sewage, living conditions) can quickly escalate to increase exposure to agents of disease. Acting in conjunction, increased exposure due to breakdowns in services and hygiene and increases in susceptibility with lowered resistance are more than sufficient to explain the response that was estimated from the data.

That the progress of respiratory tuberculosis is associated with nutritional status has been vigorously argued before (McKeown 1988). It is known that although people may harbor the bacterium, they are not necessarily struck by the disease. Whether or not they are depends on their ability to fend off the advances of *Mycobacterium tuberculosis*. And this, in turn, is largely a function of a host of factors including the prevalence of other infectious diseases (Szreter 1988), stress, malnutrition, and vitamin deficiencies (Wingfield 1924; Davies 1985). Suggestive epidemiological evidence comes to us from historical records in the Northern and Western European countries, where the death rate due to the diseases began a secular decline at around 1870 or 1880 and was broken only by wild spasmodic leaps during World War I and then again during World

War II. A sharp decline in food consumption and erosion of a balanced diet has been associated with both interruptions of the secular decline (Puranen 1991; Keers 1978).

Admittedly the epidemiological evidence is only circumstantial since the effects of other contributor factors are usually not controlled for. But, added to the clinical evidence, it suggests that there is a strong case to bolster the argument that the connection between respiratory tuberculosis and economic downturns is likely to operate through the nutritional status of populations. This evidence helps to shed light on our findings which single out respiratory tuberculosis as a disease with one of the strongest responses to economic indicators.[26]

B. **The robustness of the findings.**

To test the robustness of our estimates to violations in the assumptions and to changes in model specification we proceed to apply a battery of tests. We will report briefly only the most important of these.

B.1. *Alternative measures of economic performance.*

Table 5 of the main text summarizes (only) the net responses by cause estimated from models that include alternative independent variables drawn from national accounts: private consumption (model II) and the magnitude of the external debt relative to the value of exports (model III). We contrast these with the results from the model with GDP (model I) for four 'test' countries which span the spectrum of mortality levels.[27] The full table of responses by lags (not shown) indicates that there is a handful of cross-model differences in statistical significance of effects. Table 5 reveals some changes in the magnitude and, in a few cases, reversals of signs of coefficients. For example, whereas the total response to GDP in Chile is positive (.29), the response to Private Consumption (PC) and debt service as proportion of exports is negative (-.09

and -.15 respectively). The main culprit for the reversal appears to be deaths from respiratory illnesses, which shows a strong negative response to changes in PC but positive (and high) net responses to changes in GDP. The contrast occurs in Ecuador and, to a lesser extent, in Uruguay. For the most part, however, the conclusions that we drew before regarding the sensitivity of infectious diseases, respiratory ailments, respiratory tuberculosis, and diarrhea stand unaffected when PC is used as an indicator of economic well-being. Instead, the models estimated with the index for the load of external debt fit very poorly and yield the lowest estimates for the responses. This is in keeping with what was anticipated earlier in view of the observed lack of variability and intrinsic information content of the measure.

B.2. *The lag structure.*

A more 'favorable' parameter space can be conceived in light of our previous findings. In particular, one could represent the effects with a lag-pattern that follows an n-degree polynomial or a sinusoidal function. Allowing for different coefficients for different causes of death and age groups should enable us to replicate the variability detected throughout the analysis. This representation has the advantage of simplicity and provides an escape from the multicollinearity problem that affects the detrended series. In an initial exploration we estimate a lag-pattern with a triangular structure that imposes a maximum effect at lag 2 and then symmetric declines from the maximum to zero (see footnote 21). Since the location of the maximum is fixed **a priori**, this representation requires only two parameters identifying respectively the magnitude of the maximum effect and the slope of the decline of the effect. For diseases that increase with deterioration in economic conditions our hypotheses suggest that the 'maximum' effect should be highly negative and that the slope should be positive. For diseases that

decline with deterioration in economic conditions the situation is reversed: the maximum should be positive and the slope should be negative.

Table 6 displays the results for the same four 'test' countries we used in Table 5. In the table, b_o refers to the estimated maximum and R to the estimated slope. To facilitate comparisons with the previous models we also display the **net** effects implied by b_o and R over a duration of 5 years. Note first that only in Ecuador do the values of b_o associated with infectious diseases, respiratory ailments, respiratory TB, and diarrhea take on the 'wrong' sign (positive). This is not surprising since Ecuador also shows anomalous results from the more conventional models. In Chile and Costa Rica the implied net elasticities are 'biased' toward zero when compared to those estimated with a free lag structure (see Table 5), whereas those for Uruguay attain much higher (absolute) values.

A few conclusions can be drawn from this handful of experiments. On the one hand, our lag-free model is certainly preferable for it is less restrictive. On the other hand, the problems created by multicollinearity are cleared in the triangular model by imposing more structure. But since none of the coefficients b_o or R in Table 6 are statistically significant, our inferences are hardly strengthened. It is likely, however, that a triangular lag-structure is excessively restrictive and should attenuate the estimated net responses in most cases.

B.3. *Control for changes in the ill-defined category.*

A third test was performed to assess the possible effects that misclassification of deaths into the category of ill-defined could have on our estimates. As explained above and in Appendix 3, the test consists of controlling for the current detrended value of the number of deaths in the ill-defined category. The derivation in Appendix 3 suggests that

controlling for the non-lagged detrended value of the deaths due to ill-defined causes is an approximation to the correct (but unfeasible) procedure to eliminate the effects of oscillations in misclassifications. We should expect the estimated effects for well-defined diseases to increase (in absolute value) and the estimated effect associated with the control variable to be negative. Table 7 displays the results for the same four 'test' countries used before. Model I is the model discussed before and does not include a control. Model II includes a control for deaths due to ill-defined causes. The results obtained are somewhat disappointing, since in two of the four countries (Ecuador and Uruguay) the estimated regression coefficient for the control has a sign opposite to the expected one for all relevant causes (infectious, respiratory, respiratory TB and diarrhea). In Chile and Costa Rica the estimated coefficient is properly signed but rarely significant. In most cases, the introduction of the control has little or no impact on the net responses.[28]

B. 4. *Changes over time.*

Mortality responses must have been correspondingly weaker during past crises if they were less durable than more recent crises. Testing for this hypothesis is somewhat treacherous for a couple of reasons. First, one should effectively control for the presence (absence) of mechanisms that attenuate the impact of economic downturns, particularly those affecting the most vulnerable groups. If social interventions become more efficient over time in their quest to shield these groups and if we are unable to adequately measure such changes, the estimates of the **differences** in responses attributable to the seriousness of the crises will contain a downward bias and may even have the wrong sign. Second, if, as is likely to happen, the completeness of death registration becomes less affected by economic crises, the estimated response will appear to be larger for the

more recent periods. Only if we ignore these two difficulties--which introduce opposite biases--will we be able to model any period effects.[29]

Table 7 (model III) shows the estimates corresponding to a model that controls for the interaction between response and time period. The parameter shown in the table measures the difference in response - assumed constant across all lags - between the years before and after 1975. A positive sign is indicative of **smaller** effects in the past. The results are mixed since they are strongly dependent on country and only a few estimates are statistically significant or properly signed. In Chile and Costa Rica the estimates are positive, but only those associated with infections, diarrheas and respiratory diseases are statistically significant in Chile whereas none of them is in Costa Rica. In Ecuador and Uruguay the coefficients for infections and respiratory diseases are negative whereas those for diarrhea and respiratory tuberculosis are positive but insignificant. The grouping of countries that emerges from these results is at least consistent with the view held by some authors (Altimir 1984) that the crisis hit harder in Chile and Costa Rica than in some other countries. In summary, it is only for some causes of deaths that the effects are uniformly stronger during the most recent period. Indeed, the estimates for diarrhea and respiratory tuberculosis are positive in all four countries, but only in one (Chile) are some of these effects statistically significant. If these results are reliable at all, they would partially confirm the conjecture that longer and more intense crises, such as those that took place after 1975, do indeed lead to stronger responses at least for causes of deaths that are highly sensitive to changes in standards of living. Naturally the results we obtain could be affected by violations of the underlying assumption about a unique time marker separating types of crises which holds for all countries. Finally, it could well be that, instead, or in addition to, the shifts in the effects of crises over time different countries adopt different profiles or patterns.

V. Refinements and Extensions.

A. **Distributed lags on causes of deaths: additivity properties.**

The estimation procedure we have used is not as efficient as it could be. An optimal strategy is to estimate simultaneously all elasticities in a parameter space that satisfies simple restrictions. In particular, if model (1) is correct for all causes of deaths that we care to recognize, the weighted sum over all k of the $\beta_{nk,j}$'s should equal the coefficient for the jth lag for all causes combined, $\beta_{nT,j}$. Analogous constraints across ages (and causes) are easily derived from simple additivity rules. Ideally, the resulting constraints should be imposed **a priori** and the parameters estimated using constrained generalized least squares estimators. However, if one does not impose the constraints it is possible to derive a 'measure of fit' of the models. Indeed, the magnitude of the difference between $\theta = \Sigma_k (w_{nk} * \beta_{nk,j})$, where w_{nk} is the proportional distribution of causes of deaths in age n, and $\beta_{nT,j}$, the j-lag response from all causes of deaths combined, is a measure of the degree of fit of the model.

In the interest of brevity we examine only one out of many possible tests of consistency. In particular we calculate the net elasticity by adding the (weighted) total cause-specific elasticities (expected total elasticity) and compare it with the elasticity for total deaths estimated directly (observed total elasticity).[30] Table 8 displays the values of the corresponding test statistics when GDP is used as the measure of economic well-being. In all countries (including those not in the table) the values of the expected and estimated responses are remarkably close to each other, thus providing alternative confirmation about the goodness of fit of the models.

B. **The effects on the patterns of mortality.**

The estimated elasticities of mortality by age and causes of deaths provide a first

approximation for an assessment of the magnitude of effects of economic oscillations on patterns of mortality. A more refined but also more heuristic appraisal consists of transforming the estimated elasticities into changes in life expectancy at birth (or at any other ages). To accomplish this we utilize Pollard's (1988) expression that translates changes in age-cause-specific forces of mortality into changes in life expectancy:

$$\eta_0 = \Sigma_k \int_0^\infty (\mu_k(x) - \mu'_k(x))\, \omega(x)\, dx \qquad (3)$$

where η_0 is the total change in life expectancy, $\mu_k(x)$ and $\mu'_k(x)$ are the forces of mortality at age x due to cause k before and after an economic recession (expansion), and $\omega(x)$ is the average given by:

$$\omega(x) = .5 * (p(x)e'_x + p'(x)e_x) \qquad (4)$$

where p(x) and p'(x) are the probabilities of surviving up to age x in the life tables prior to and after the oscillation, and e_x and e'_x are the corresponding life expectancies at age x.

To implement equation (3) we calculate the function $\mu'_k(x)$, taking into account responses at all lags as $\mu_k(x) * (1 + \Sigma_j \beta_{kn,j})$, and then obtain the corresponding probabilities of survival and life expectancies. Note that, like their counterparts in absolute numbers, all the calculations involved in (3) can be partitioned by lags, ages, and causes. These operations are tantamount to the calculation of the partial derivatives of life expectancy with respect to changes in economic indicators. Table 9 shows the result of implementing equation (3) to a 'median' country (with a median age-cause structure of mortality rates) subjected to a ten percent drop in GDP and to age-cause-specific

elasticities equal to the median of our sample. We only examine the responses and contributions of causes of death that altogether represent about 60 percent of the total deaths. Of the total change in life expectancy triggered by the downturn (.70), only a fraction (.23) can be accounted for in a model that takes into account the causes of death we chose to examine. The difference is due to the lack of fit of the model by cause and also to the contribution of causes that we do not consider in the table. Not surprisingly, the most important contribution corresponds to changes at age 0: about 87 percent of the total (explained) change can be attributed to worsening conditions at this age. This is simply due to the fact that the figures in Table 8 reflect both the responsiveness to the economic changes and the more than proportionate influence exerted by infant deaths in the determination of life expectancy. Infectious diseases account for about 51 percent of the (explained) changes, whereas respiratory diseases contribute slightly less (39 percent). The total contribution of respiratory tuberculosis is small both among infectious diseases (1 percent) and as a fraction of the total changes (.7 percent). Diarrheas, on the other hand, are the most influential of infectious diseases and operate mostly at age 0.

C. **The effects of periodic crises on excess mortality.**

A more precise assessment of the consequences of periodic economic downswings is to calculate the average relative excess mortality induced by any number of them during a fixed period of time. The calculations are fairly straightforward: we first estimate the total death rate during the chosen time interval, assume a frequency of crises and their location in time and, for each of them, estimate the excess number of deaths by applying the net total elasticities estimated before (by age and cause of deaths). As an illustration, we use the following initial conditions: i) a 'median' Latin

American country that experiences two downturns of GDP equivalent to 10 percent over a period of 28 years; ii) the crises are centered within the first and last five years of the period. We estimate that such a regime of crises combined with the patterns of median net responses would increase the total death rate by about 3.5 percent over the 'crises-free' death rate of about 8.9 per thousand. A breakdown of contributions by causes of death again shows that the bulk of the response is attributable to infectious diseases and respiratory ailments and that, of these, an excess of deaths due to diarrhea explains most of the changes.

VI. Summary and Conclusions.

Far from being an arcane feature of pre-industrial societies, mortality responses are still noticeable in demographic regimes of developing countries. In Latin America the responses follow a profile by age and cause that is consistent with theoretical expectations. Thus, infectious and respiratory diseases and, in particular, respiratory tuberculosis and diarrhea are the causes of deaths that are most responsive to economic downturns. Infants and young children as well as youngsters up age 15 appear to be the most affected.

Less clear than the existence of the responses themselves are the regularities that underlie them. We found some aggregate evidence suggesting that the reactions of the levels of mortality at age 0 are inversely related to the patterns of breastfeeding. This is in agreement with the idea that vulnerability of the very young is maximized in societies where the norm of universal and long breastfeeding has crumbled. Furthermore, although the patterns of response are roughly consistent with those found in other developing societies, there are only weak grounds for suspecting that the

magnitude of the mortality upturns have any association at all with levels of development or average levels of mortality. Nor is it evident that there is any systematic relation to the character or nature of the crisis.

Although the empirical evidence for the Latin American countries that we examine does indeed weaken the idea asserting that short-term reactions to economic changes are dissociated from mortality changes, it simply does not reveal very strong associations. This is not just an issue of statistical significance but one of scale of responses. In the last part of the paper we showed that a moderately large drop of GDP would lead to a comparably small response of mortality both in terms of intrinsic changes in life expectancies (Table 8) and in terms of excess deaths. These two characteristics permeating our data - patterned responsiveness and muted scale of the response - are not inconsistent and indeed are a marker of pre-industrial patterns as well, when mortality crises supposedly played a more dominant role than they do today. This being said, we emphasize that excess mortality is just the tip of the iceberg, and that wretched conditions of large segments of the population are perfectly compatible with mortality increases of only small magnitudes. The actual deterioration of conditions will probably remain concealed until we refocus our attention to other dimensions of health status.

Lastly, it should be emphasized that the paper utilizes definitions and a methodology that could be refined in several respects. First, it is clear that one could focus on other causes of death and explore the possibilities of re-grouping causes of death in ways that are more consistent with theories linking economic changes and morbidity and mortality. Second, we should enlarge our sample not only to increase the power of some tests but also to do justice to the variability of patterns in the continent.

Third, one could explore more thoroughly the alternative economic indicators as gauges of actual changes in standards of living of some or all social classes. A final but equally important issue is designing a procedure of estimation that does not rely on the assumption systematically invoked in our distributed lag models that the magnitude of responses to a booming economy ought to be opposite in sign but equal in magnitude to the responses in recessionary times. This seems to be a fairly restrictive and limiting assumption that if relaxed could lead to different results. Until some or all of these issues are addressed it will be difficult to derive more than tentative conclusions.

APPENDIX 1
The groups of causes of deaths we use and the corresponding items in the various ICD lists are as follows:

Title of group	ICD 6th and 7th	ICD 8th	ICD 9th
1. Respiratory TB	A001	A006	B020-B021
2. Typhoid (+)	A012-A013	A002-A003	B011
3. Measles	A032	A025	B042
4. Whooping cough	A022	A016	B034
5. Hepatitis (+)	A034	A028	B046
6. Diarrheas	A016; A104; A132	A004; A005	B014-B016
7. Ulcers/cirrhosis	A099-A100; A105	A098; A102	B341; B347
8. Pneumonia, bronchitis and influenza (+)	A088-A093; A087	A090-A093; A089	B320-B323; B310-B312
9. Heart disease	A070; A079-A086	A080-A088	B25-B30
10. Diseases of infancy	A127-A135	A126-A135	B44-B45
11. Avitaminosis (+)	A064	A065	B19
12. All infections	A001-A043; A104; A132	A001-A044	B01-B07
13. Suicide, homicide and accidents	AE138-AE150	AE136-AE137	B47-B56
14. Ill-defined	A136-A137	A136-A137	B46

The groups of causes used in the paper and the groups listed in the table are associated in the following way:

Paper	Table (no. on the list)
Infectious	12
Respiratory	8
Chronic	7
Heart	9
Violence	13
Ill-defined	14
Respiratory TB	1
Diarrhea(s)	6

Remarks:

1. For some countries and some years it was necessary to establish a correspondence between the observed causes of death listed according to the B list and the target A list. The problematic linkages affect a handful of causes of deaths and should have no effects on the analysis presented in the paper.
2. Note that group 12 (infectious) includes A104 and A132 in the ICD 6th and 7th. These are special diseases of infancy and diarrheas of the newborn which were in later versions assigned to well-defined infectious diseases. The problems of correspondence should be minor and, in any case, only affect age 0. Non-inclusion of A104 and A132 leads to irreconciliable trends in infant mortality due to infectious diseases before or after the adoption of the 8th revision.
3. For causes marked with '+' there are discontinuities for some countries before 1955 that we were not able to resolve. In these cases our series start after 1955. In the case of avitaminosis we were not able to resolve discontinuities that are apparent at several points in time and for several countries. For this reason we decided not to include this cause of death separately in our analysis.

APPENDIX 2	
Countries and years included	
Uruguay	1955-1987
Chile	1954-1989
Costa Rica	1956-1984
Guatemala	1958-1985
Mexico	1956-1986
Panama	1955-1989
Trinidad	1951-1984
Venezuela	1955-1989
Ecuador	1961-1988

APPENDIX 3

I. An informal justification.

An informal justification for the introduction of the control is fairly simple to develop. If economic downswings lead to increases in the number of deaths due to cause k that are allocated to the ill-defined category, the estimated response of cause k will be underestimated. Controlling for the detrended values of deaths due to ill-defined enables us to estimate the magnitude of this association and thus purges out the downward bias in the estimated response due to the well defined cause. If our model is correct the **observed** (absolute) deviation from a trend in the number of deaths due to cause k, δy_k, is given by:

$$\delta y_k(t) = \Sigma_j \beta_j \delta x(t-j) - \rho k \, \delta y_{ill}(t)$$

where $\delta x(t-j)$ and $\delta y_{ill}(t)$ refer, respectively, to the observed absolute deviations from trends of the economic indicator and the number of deaths due to ill-defined causes at lags j and 0, β is the unstandardized response to changes in income, and ρ_k is the fraction of ill-defined deaths that should have been allocated to cause k. This equality provides sufficient rationale to include the detrended number of deaths due to ill-defined causes as a control in the equation for the observed detrended number of deaths due to cause k: the estimated coefficients for income will be corrected and should be larger (in absolute value) than the uncorrected ones. The estimate of ρ_k should be less than one in absolute value but must bear a negative coefficient.

II. A more rigorous derivation.

We suggest now a more rigorous procedure to correct for the biases introduced by misclassification of causes of deaths. However, the procedure involves a complicated model that we are not able to estimate due to the small number of observations available to us. For simplicity, assume that we only have non-lagged effects so that β_k is the true elasticity of cause of death k to changes in income. If a fraction η_k of deaths due k end up as ill-defined, the observed elasticity will be $\beta_k*\eta_k$. One can show that the correct specification for the deaths due to ill-defined causes is one where the independent variables are all terms constructed as the products of: a) the ratio of the detrended number of deaths due to cause k to the detrended number of deaths due to ill-defined and b) the detrended value of income, e.g., $x(t)*y_k(t)/y_{ill}(t)$. The elasticity with respect to these interaction terms are the products $\beta_k*(1-\eta_k)$. Consequently if one estimates the equation for cause of death k we obtain the (biased) coefficients $\beta_k*\eta_k$. If we then estimate the equation just specified for the ill-defined we will obtain the estimates $\beta_k*(1-\eta_k)$. These two sets of estimates are sufficient to obtain the correct values of β_k. We can relax the assumption on non-lagged effects and the same algebraic equivalences will follow on a lag-specific basis.

Note that if there are, say, 10 causes of deaths we need to estimate an equation for deaths in the ill-defined category that contains 10 *l number of variables, where l is the total number of lags to be considered. Since in our case we set l=5, we would need 50 independent variables, far larger than the actual number of observations available.

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1. Although apparently harmless, the idea that the disappearance of crises-mortality can be taken as a marker of modern mortality ought to be carefully qualified since it is by no means clear that their contribution to the high levels of mortality prevailing during pre-industrial times was inordinately high (see Fogel, 1989).

2. We point out at the outset that the evidence regarding the association between economic changes and mortality in pre-industrial societies is murky. Although there is a general pattern reflecting the existence of short-term associations (Galloway, 1988; Hammel, 1985; Richards, 1984; Lee, 1981), the strength of such association is less than impressive. Some have argued that the weak association of short run fluctuations masks stronger connections that are confounded by the nature of the indicators (Fogel, 1989).

3. For most countries we have information after 1955 but before 1987 or 1988. For a few countries information stretches back to 1951 and reaches up to 1990.

4. Bravo (1992) and Bravo and Vargas (1990) have paved the way for the analysis of mortality by causes in Latin America. Hill and Palloni (1992) suggested procedures and analyzed a partial data base on total deaths. Galloway and Lee (1985) and Reher (1989) have pursued similar ideas on the analysis of demographic fluctuations in China and colonial Mexico, respectively.

5. There is also evidence that respiratory tuberculosis and enteric diseases may be interrelated in complex synergy (see Szreter 1988).

6. A different but complementary factor that leads to stronger responses among impoverished groups has been advanced by Fogel to interpret findings from preindustrial societies (Fogel 1989, 1991). In economies with highly unequal distribution of assets, particularly land, the propertyless groups tend to have less of a cushion during hard times and their mortality response may be consequently augmented. Substantial group differentials in responses to the crisis of 1980's for urban areas in Latin America have been documented by Altimir (1984).

7. Puranen has argued that death rates of pulmonary tuberculosis may be more responsive to exposure than they are to nutritional status. His conclusion, however, is derived from evidence for small populations (Puranen 1991). Cronjè, on the other hand, has studied evidence suggesting that exposure was probably more important in Victorian England (Cronjè, 1984). The evidence that pulmonary tuberculosis is linked to nutritional status is strong (see also Barnes, 1992). What is unclear, however, is the magnitude of the deterioration in nutritional status that is needed to induce an increase in active cases, and the time lags involved.

8. We note that the incidence and case fatality of respiratory tuberculosis may be causally related to alcohol consumption (Puranen 1991).

9. Evidence for the most recent recession in the United States suggests that the rate of accidents associated with work tends to decrease rather than to increase.

10. It should be noted that for the most part the direct effects of malnutrition, for example, are nowhere very prevalent even though chronic malnutrition could be well entrenched in many places. Under these conditions, economic crises could well lead to changes in the prevalence of certain diseases but it does not follow that case fatality rates will increase proportionately (McKeown 1988).

11. Increases in the death rates due to respiratory tuberculosis can be due to a combination of increased incidence and increased mortality among those already infected. Increased exposure (due to overcrowding, for example) can have increased incidence but not before one or two years. And a reduction in nutritional status is unlikely to have effects that will be felt before a year or two. The expected reaction of respiratory tuberculosis may come early, however, if the occurrence of other infectious diseases weakens the resistance of those already infected with tuberculosis.

12. The idea of differential impact has been explicitly formalized in the study of responses to economic crisis in pre-industrial times (Fogel 1989)

13. These relations should hold only when confounding factors are controlled.

14. Private consumption and debt service are affected by potentially damaging measurement errors. The calculus of private consumption in national accounts is oftentimes of a residual nature and final estimates are obtained after total GDP and gross investments have been accounted for. The total value of debt servicing can reflect changes in a host of external prices that bear no clear relation to a country's standards of living. In theory at least, the ratio of long term debts service to the value of exports should be well suited to the task to the extent that crises originate in excessive external borrowing. However, it may be poorly correlated with the actual effects of internal economic policies.

15. Ideally we would have liked to use both indicators but our series are too short to accommodate more than one independent trend. Real GDP was calculated using national figures on GDP per capita per annum, deflated and expressed in constant USA dollars of 1970.

16. The existence of an informal economy changes the institutional context within which mortality responses may occur. Let us assume that the true relation is as follows:

$$D=kI^{\alpha}$$

where D is the death rate, k is a constant (that can be standardized to be equal to 1), I is the economic indicator and α is the elasticity of mortality relative to economic conditions (as measured by I). The existence of informal economies can be posited to affect the value of α. If so, the equation above is misspecified and, depending on the exact role played by the informal economy and its degree of responsiveness to recessions in the formal economy, it will yield a biased estimate of the true mortality response.

17. This statement is invalid, of course, if the recession itself affects the levels of completeness of death registration. It is quite possible that registration of vital events becomes more inaccurate during recessionary periods. If this is so, we will underestimate

the magnitude of the response to the crisis.

18. Other groupings of causes of deaths were tried. In particular, we estimated effects on deaths due to several infectious diseases, including measles and typhoid. In most cases, however, individual infectious diseases yield unstable results.

19. To implement local least squares it is necessary to define the range of observations that are relevant for fitting any particular point (the so-called 'band-width,' or bw for short). Since the aim is to produce a trend that is not affected by sharp deviations from it, we choose to use bw values between .90 and 1.00. This means that almost **all** observations are used to fit a particular point, but that the weight given to points that are far removed from the fitted one will be proportionately less than the weight given to points that are close to it. Although we estimated models employing bw values ranging from .20 to 1.00, the results that we present here, unless otherwise specified, correspond to bw values equal to .90. It is important to emphasize that although the numerical values of the estimates change as we alter bw, the *main inferences* that we draw are very similar.

20. Although other specifications are possible, they are excessively demanding of the data. For example, if recessions of longer durations are likely to elicit stronger responses, we could have introduced a measure of duration of the crisis as a dummy variable and then estimate the 'added' elasticity that accompanies recessions of longer duration.

21. This parameterization starts from the assumption that the effect at lag 2 is a maximum (minimum) and that the effects before and after lag 2 decline (increase) linearly:

$$\beta_j = \beta + \text{abs}\ (j-2) * \sigma \quad \text{if } 0 < j < 5$$

where σ is the rate of decline from the maximum value of the response. We expect that $\sigma > 0$ if $\beta < 0$ and that $\sigma < 0$ if $\beta > 0$. We have not yet experimented with obvious modifications to this model, including consideration of polynomials or trigonometric functions. It should be noted that imposing this structure (or any alternative one) entails a risk of biasing the estimated effects. Whether the magnitude of this bias is larger than the losses of precision due to multicollinearity is an issue that deserves to be investigated.

22. Although we estimated effects for the group of causes associated with heart diseases, we have excluded presentation of the corresponding results to avoid congestion. Without exception, the patterns of responses for this group were erratic and did not conform to what we expected. It is important to note that Diarrhea and Respiratory Tuberculosis are listed separately but are also included in the group of infectious diseases (see Appendix 1).

23. The **net** value of a given response is the sum of the estimated elasticities across all lags. There are three statistical tests that we perform. The first is an F-test to falsify the null hypothesis that the true model is one with no lagged terms (that is, the best fitting

model is one with only a constant term). The second corresponds to the null hypothesis that the sum of effects (the sum of the coefficients) is zero. The third is a test of the null hypothesis that the sum of the negative coefficients is zero. The first test simply reveals whether or not oscillations around a trend are explained at all by oscillations of the economic indicator. The second test provides evidence of whether or not the net effect--after initial impact and subsequent echoes are taken into account--amounts to something. The third test is designed to draw inferences about the magnitude of the responses that are in the expected direction, regardless of the magnitude of echoes that ultimately offset them. To avoid excessive cluttering we only present the results of the tests for all deaths (total) by cause.

24. Throughout the paper and unless otherwise noted, we use two-tailed tests and infer significance at levels of .05 or less.

25. Since the data used by Lee were on prices and mortality, a positive response was equivalent to a negative response in our data. Thus, to make comparisons easier we have reversed the signs of Lee's graph.

26. Other infectious diseases strongly linked to nutritional status, such as measles, do not show the same patterns as respiratory TB. Indeed in results not shown we identified only erratic patterns for deaths due to measles. It is likely that vaccination campaigns as well as seasonality conceal some of the fluctuations due to deterioration of nutritional status. Of course, it may also be that some of the deaths attributable to other diseases (particularly diarrhea) have measles as an underlying cause.

27. Although we obtained results for all countries, we display only those corresponding to four of them to avoid presentation of an excessive number of tables. All the conclusions we reach examining the selected set are confirmed by those not shown.

28. The same applies for the remaining countries in our sample (Guatemala, Mexico, Panama, Venezuela and Trinidad)

29. There are two strategies to model period effects. The first is to assume that the lagged effects within one period are proportional to the lagged effects within another period and that a single proportionality factor suffices to capture variability. According to this reasoning the proper model should include a single interaction term corresponding to the sum of the products of the lagged exogenous variables and a dummy for period. The second strategy imposes the less restrictive assumption that the ratios of lagged effects across two periods varies with the lag. Thus, the corresponding model includes one interaction term per lag. Each of these equals the product of the lagged exogenous variable and the dummy for period. This model uses considerable more degrees of freedom and, as it turns out, does not fit the data any better than the model corresponding to the first strategy. To simplify presentation we display the results of the first model only. The results of the more cumbersome model corresponding to the second strategy do not reveal different patterns.

30. The alternative tests are fairly obvious. A first test statistic is the difference between age-specific estimated elasticities and the one that obtains by adding across causes the age-cause-specific estimated elasticities. The second test statistic focuses on the

difference between cause-specific estimated elasticities and the one that obtains by adding the age-cause-specific elasticities across ages. These test statistics are easily derived by imposing additivity to the age-by-cause matrix of elasticities. Indeed, by adding the values of $\beta_{nT,j}$ across ages (n) we will obtain estimates of the total mortality response (regardless of age); if model (1) is correct, these estimates should equal the total response estimated directly. Under these conditions, the response distribution of lags-by-cause can be extended to include age as well:

$$\lambda_{nk,j} = \beta_{nk,j} \; / \; \Sigma_n \, \beta_{nT,-j} \; . \; \hat{w}_n \qquad (A)$$

where $\hat{w}^{\wedge}_n$ is the age-distribution of deaths.

The joint distribution in (A) can be readily converted into a marginal distribution to assess the magnitude of the responses across causes only (regardless of age) or across age only (regardless of cause). These quantities may also be used to test some of the hypotheses put forward before regarding the nature of the effects on some age segments.

Another refinement to investigate the nature of the mortality response is the joint distribution of lags-by-cause:

$$\lambda'_{nk,j} = \beta_{nk,j} \; / \; \beta_{nT,j} \qquad (B)$$

or the proportion of the total response that is attributable to cause k in age group n at lag j. These statistics are implicitly used in the next section to check that our expectations regarding the role of different causes is empirically confirmed.

TABLE 1a: EFFECTS BY LAG FOR SELECTED CAUSES OF DEATHS AND COUNTRIES: TOTAL POPULATION [a, b]

COUNTRY	LAG	ALL	EFFECTS ON: INFECTIOUS	CHRONIC	RESPIRATORY	VIOLENCE	ILL-DEFINED	RESPIRATORY TB	DIARRHEA
CHILE (n = 31)	0	.24	.08	.62	1.27*	.39	-1.17*	- .12	.02
	1	- .25+	- .22	- .00	-1.67*	.19	- .30	- .40	- .00
	2	.23	- .36	.10	1.02	.26	- .20	- .01	- .32
	3	- .02	- .02	- .02	- .13	.12	- .37	- .82*	- .09
	4	.08	- .35	.44	.61	.16	- .55	- .27	- .36
Adj. R^2		- .02	- .11	- .13	.19	.11	.23	.17	- .17
COSTA RICA (n = 29)	0	- .10	.58*	.22	- .05	.74*	- .11	.50	-1.23
	1	- .21	-2.05*	.22	- .69	.67	.20	- .85	-2.23+
	2	- .04	.23	- .47	- .29	-1.16*	1.93	.45	- .21
	3	- .46	- .59	- .93	- .58	.58	-1.36	-1.29	- .17
	4	.46	1.49*	.97	.86	- .20	.36	1.14	1.90
Adj. R^2		.21	.75	- .06	.06	.54	- .01	- .04	.62
ECUADOR (n = 23)	0	- .16	- .42	.09	- .29	- .01	- .30+	.23	- .03
	1	.03	.40	- .38	.16	- .22	- .04	.12	.16
	2	- .30	- .30	.48	- .46	.48+	- .46+	- .07	.11
	3	.22	- .19	- .08	- .14	.08	.30	- .38	- .82
	4	- .03	- .21	.07	- .10	.07	.05	.28	.03
Adj. R^2		.06	- .08	- .07	.11	.04	.49	.01	- .19

COUNTRY	LAG	ALL	EFFECTS ON: INFECTIOUS	CHRONIC	RESPIRATORY	VIOLENCE	ILL-DEFINED	RESPIRATORY TB	DIARRHEA
URUGUAY (n = 28)	0	- .02	.52	- .22	.83	- .15	.46*	.54	1.23*
	1	.04	- .71	- .45	- .75	.51*	- .02	- .33	-1.16
	2	- .45*	- .69	- .19	-1.67*	.33	- .54	- .66	- .66
	3	.49*	.23	1.00*	1.92	.05	.58*	.04	.83
	4	- .38	- .24	-1.07*	-1.48*	- .49*	.04	- .32	- 1.00
Adj. R^2		.28	.24	.27	.14	.27	.10	- .03	.07
GUATEMALA (n = 23)	0	- .38	-1.59*	1.08	- .73	[c]	- .93	- .77	-1.64*
	1	- .69	.43	-1.55	- .74	--	.13	-2.20	1.79
	2	1.26	1.19	.52	.46	--	1.38	2.19	- .01
	3	- .33	-1.08	- .21	.83	--	-2.07*	- .97	-1.22
	4	- .56	- .34	- .28	-1.48*	--	.47	.03	- .07
Adj. R^2		- .02	.82	- .23	.08	--	.02	.10	- .06
MEXICO (n = 25)	1	- .07	-1.66	1.72*	-1.12	1.60	-2.24*	-1.16*	-1.57
	2	- .09	.19	- .48	- .26	- .04	.05	- .15	.02
	3	.23	.07	.70	.62	.06	.39	.18	.23
	4	- .02	1.84	-1.34*	.68	-1.59	.70	.34	1.13
Adj. R^2		- .14	.51	- .30	.19	.19	.16	.51	.40
PANAMA (n = 30)	0	.11	- .20	.59	.24	.23	.19	.16	- .59
	1	- .09	- .59	- .15	- .01	- .18	.15	- .09	- .29
	2	.22	- .67	.42	.20	.53	- .35	.40	-1.51
	3	.32	.65	.78	.20	.27	.31	- .05	1.51
	4	.13	.94	-1.20	.73	-1.19*	.74	- .41	1.23
Adj. R^2		- .11	- .09	- .05	- .16	.03	- .15	- .06	.09

COUNTRY	LAG	ALL	EFFECTS ON: INFECTIOUS	CHRONIC	RESPIRATORY	VIOLENCE	ILL-DEFINED	RESPIRATORY TB	DIARRHEA
VENEZUELA (n = 30)	0	.09	.10	.04	.39	.01	2.72*	- .20	.29
	1	- .11	- .96+	.18	- .61	.75*	-2.40+	- .33	-1.52+
	2	.29	.46	.84*	.47	.14	.02	- .09	.78
	3	- .03	- .36	- .24	-1.49*	.22	1.06	- .13	- .25
	4	- .19	- .65	- .01	- .01	.05	-1.38+	- .21	- .88
Adj. R^2		- .06	.13	.18	.06	.49	.15	.07	.19
TRINIDAD (n = 29)	0	- .17	-1.36	.31	- .68	- .06	-1.22	-2.70	-1.23
	1	.10	-1.46	.03	.20	.22	-1.77+	-3.75	-2.33
	2	.10	1.61	- .77	.32	.39	3.11*	3.31	2.10
	3	- .06	- .48	- .53	-1.06	- .55	- .23	- .86	-1.26
	4	- .08	-1.21	- .23	- .07	- .17	.18	.78	-1.56
Adj. R^2		- .16	.08	.10	.03	- .11	.40	.23	.02

FOOTNOTES TO TABLES 1a THROUGH 1f

a The symbol "*" indicates statistical significance at $p < .05$ ($^*t^9 > 2.00$).
The symbol "+" indicates statistical significance at $.05 < p < 10$ ($1.7 < {}^+t^9 < 2.0$).

b In some countries the number of cases for age group 0 is greater than for the other age groups. This is because for age group 0 we found more updated information that enabled us to extend the series by one or two years.

c Deaths by violence in Guatemala exhibit a very irregular pattern that yields implausible large (or small) responses.

c:\pal\table_1a

TABLE 1b: EFFECTS BY LAG FOR SELECTED CAUSES OF DEATHS AND COUNTRIES: AGE GROUP 0 [a,b]

COUNTRY	LAG	ALL	EFFECTS ON: INFECTIOUS	RESPIRATORY	VIOLENCE	ILL-DEFINED	DIARRHEA	DISEASES OF INFANCY
CHILE (n = 31)	0	.21	- .06	.76	.03	-1.37	- .22	.00
	1	- .31	- .35	- .67	.65	3.68+	- .11	- .56
	2	- .05	- .81	.04	.41	-1.81	- .63	.14
	3	.10	- .27	- .32	- .95	-4.61*	- .73	.20
	4	- .19	- .40	.19	.51	.82	- .04	- .21
Adj. R^2		- .07	- .08	- .05	- .01	.31	- .11	- .14
COSTA RICA (n = 29)	0	- .63+	-1.68*	-1.24+	-1.90	- .92	-2.31*	- .23
	1	- .78+	- .11	- .08	2.94	.29	.20	.00
	2	.07	- .18	.05	- .96	1.44	- .25	- .17
	3	- .72+	-2.21*	-1.93*	-3.17	-3.07*	-2.16*	- .76
	4	- .11	-1.19+	- .16	- .97	-1.40	- .76	.65
Adj. R^2		.69	.67	.41	.47	.28	.60	- .05
ECUADOR (n = 23)	0	- .10	- .11	- .18	1.46	.05	.26	.22
	1	.32	.64	.34	3.43+	.32	.49	.29
	2	- .08	.24	- .17	-6.88*	- .83	.33	- .97+
	3	- .13	- .82	- .18	3.31	.43	-1.10	.99+
	4	.07	- .31	- .47	- .62	- .13	- .11	1.10*
Adj. R^2		- .22	.05	.06	.21	- .16	- .07	.39

COUNTRY	LAG	ALL	EFFECTS ON: INFECTIOUS	RESPIRATORY	VIOLENCE	ILL-DEFINED	DIARRHEA	DISEASES OF INFANCY
URUGUAY (n = 28)	0	.40	.85	1.68+	.83	2.21*	1.20	.10
	1	- .64	-1.80+	-1.73*	.01	- .78	- 1.82+	.46
	2	- .00	.07	- .09	1.85	- .72	- 1.23	- .43
	3	- .22	- .95	- .89	-6.17+	.18	.40	.11
	4	.10	.37	- .57	4.36	- .23	- .17	- .19
Adj. R^2		- .03	.15	.28	- .10	.31	- .05	- .17
GUATEMALA (n = 24)	0	.06	- .06	-2.11+	c	-2.54	.55	3.96
	1	- .62	- .60	1.75	--	2.39	- .60	-4.99
	2	.68	-1.46	- .97	--	.75	1.35	2.93
	3	.12	-1.11	.30	--	-2.65	-1.53	1.97
	4	- .41	.19	-1.14	--	- .84	.84	.32
Adj. R^2		- .23	- .23	- .01	--	.28	- .22	- .04
MEXICO (n = 25)	0	- .88	-1.32+	-1.72+	-1.39	-2.20	-1.03	.08
	1	- .59	- .46	-1.02	- .56	-5.02	- .36	.50
	2	- .28	- .66	- .27	- .52	1.58	- .85	- .42
	3	- .17	- .09	- .27	-1.10	- .49	.17	.31
	4	.80	1.00	1.37	2.39	1.35	.75	.12
Adj. R^2		.32	.39	.35	- .06	.05	.28	- .23

COUNTRY	LAG	ALL	EFFECTS ON: INFECTIOUS	RESPIRATORY	VIOLENCE	ILL-DEFINED	DIARRHEA	DISEASES OF INFANCY
PANAMA (n = 30)	0	- .48	-1.35	.51	.01	- .09	-1.57+	- .22
	1	.01	- .05	- .06	- .06	.38	.16	- .04
	2	- .55	-2.22+	- .04	-2.13	-1.20	-3.33*	.15
	3	.38	1.42	.19	2.66	.44	2.43	- .45
	4	1.05	2.35	2.34	1.80	1.24	2.76+	.72
Adj. R^2		.29	.21	- .01	.04	- .10	- .36	- .13
VENEZUELA (n = 30)	0	.17	.40	.87	.54	2.03+	.24	.17
	1	- .43	-1.13	-1.60	-1.37	-2.31+	-1.45+	- .62+
	2	.65	.84	.47	- .22	.94	1.21	.63+
	3	- .25	- .46	-2.02+	- .87	.69	- .07	- .05
	4	- .46	- .81	.11	- .46	-2.60*	-1.14+	- .67*
Adj. R^2		- .09	- .02	.12	.08	.23	.03	.23
TRINIDAD (n = 29)	0	- .21	-2.01	-1.40+	-1.22	1.12	-2.01	.97
	1	- .54	-2.21	-1.98+	1.42	- .62	-2.25	- .75
	2	.43	2.03	.73	2.54	4.99+	2.03	.46
	3	.48	-1.29	1.61	-3.18	-4.10+	-1.33	1.00
	4	- .66	-2.42	- .72	4.03*	2.66	-2.39	.75
Adj. R^2		- .10	.15	.37	.22	.02	.11	.08

FOOTNOTES TO TABLES 1a THROUGH 1f

a The symbol "*" indicates statistical significance at $p < .05$ ($^9t^9 > 2.00$).
The symbol "+" indicates statistical significance at $.05 < p < 10$ ($1.7 < {}^9t^9 < 2.0$).

b In some countries the number of cases for age group 0 is greater than for the other age groups. This is because for age group 0 we found more updated information that enabled us to extend the series by one or two years.

c Deaths by violence in Guatemala exhibit a very irregular pattern that yields implausible large (or small) responses.

TABLE 1c: EFFECTS BY LAG FOR SELECTED CAUSES OF DEATHS AND COUNTRIES: AGE GROUP 1-4[a,b]

			EFFECTS ON:				
COUNTRY	LAG	ALL	INFECTIOUS	RESPIRATORY	VIOLENCE	ILL-DEFINED	DIARRHEA
CHILE (n = 31)	0	.49	.56	.65	.24	- .64	- .24
	1	- .66*	- .37	-1.23*	.08	-1.05	- .20
	2	.27	- .69	.95*	.14	.11	- .54
	3	.35	.68	- .21	- .10	- .23	.06
	4	- .08	- .42	.08	.17	- .38	- .66
Adj R^2		.18	- .09	.34	- .13	- .10	- .15
COSTA RICA (n = 29)	0	- .78	-1.23+	-2.41*	2.52*	- .33	-1.09
	1	-1.81+	-3.15*	- .52	-3.19*	- .73	-2.47+
	2	1.70+	2.51*	1.90	.03	- .30	.57
	3	-1.48	-2.56*	-1.32	1.52	- .17	-2.04
	4	1.43	3.06*	1.22	-1.00	1.22	3.60*
Adj R^2		.45	.71	.44	.19	.20	.71
ECUADOR (n = 23)	0	- .61	- .72	- .51	- .11	- .66+	- .27
	1	.02	- .07	- .08	.17	- .63	.02
	2	- .26	- .09	- .03	- .10	.59	- .02
	3	.71	.58	.11	.02	.69	- .57
	4	- .58	- .62	- .38	- .01	- .90+	.09
Adj R^2		.24	- .10	- .01	- .26	.15	- .22

COUNTRY	LAG	ALL	EFFECTS ON: INFECTIOUS	RESPIRATORY	VIOLENCE	ILL-DEFINED	DIARRHEA
URUGUAY (n = 28)	0	1.04	3.00+	2.10+	.32	1.87+	3.28*
	1	.19	.11	-2.05	.62	1.47	-3.69*
	2	-1.78+	-3.38	- .19	-1.41	-3.86+	.75
	3	.23	1.20	- .10	1.32	1.43	1.27
	4	.39	- .25	- .10	- .71	.10	-1.84
Adj R^2		.24	.09	.03	- .03	.18	.15
GUATEMALA (n = 23)	0	- .48	- .95	- .18	c	.48	-1.78
	1	-1.33	- .63	-2.20+	--	-1.45	2.38
	2	1.57	1.33	1.68	--	1.63	-1.09
	3	.09	.03	1.13	--	-1.57	.16
	4	-1.15	-1.30	-1.75*	--	.36	-1.01
Adj R^2		.06	- .05	.31	--	- .12	- .08
MEXICO (n = 25)	0	- .99	-1.37	- .63	.03	- .94	-1.00
	1	- .24	- .42	-1.09	- .85*	-2.00	- .79
	2	- .00	.42	.27	.24	.18	.56
	3	.18	- .12	.35	- .37	- .00	- .08
	4	.62	1 .15	.79	.11	.06	1.18
Adj R^2		.03	- .02	- .07	.13	.04	.05
PANAMA (n = 30)	0	.45	.04	.16	- .24	.08	- .25
	1	- .34	- .63	- .16	- .83	.74	.12
	2	1.38+	1.44	1.41+	.41	.75	.30
	3	- .59	- .78	-1.16	.19	- .40	.48
	4	- .31	.15	- .44	-1.10	.58	.45
R^2		- .04	- .14	- .05	.05	- .07	- .15

			EFFECTS ON:				
COUNTRY	LAG	ALL	INFECTIOUS	RESPIRATORY	VIOLENCE	ILL-DEFINED	DIARRHEA
VENEZUELA (n = 30)	0	- .21	- .99	- .41	- .21	1.40	- .55
	1	- .61	- .40	- .80	.08	-1.14	- .40
	2	.40	.36	.96	- .19	1.00	.42
	3	- .11	- .85	-1.23+	.40	.50	- .84
	4	- .77	- .78	- .36	- .37	-2.58*	- .32
Adj R^2		- .09	- .12	.01	- .13	.16	.10
TRINIDAD (n = 29)	0	.33	-1.87	2.05+	1.19	2.16	-1.34
	1	.29	- .35	- .97	1.93	-1.81	-1.40
	2	.43	1.22	1.57	-2.33+	9.10+	1.72
	3	- .81	-2.77	- .92	.73	-2.42	-3.33+
	4	.84	- .59	.98	1.33	-5.71	- .10
Adj R^2		- .08	.04	.02	.11	.13	.03

FOOTNOTES TO TABLES 1a THROUGH 1f

a The symbol "*" indicates statistical significance at $p < .05$ ($^*t^* > 2.00$).
The symbol "+" indicates statistical significance at $.05 < p < 10$ ($1.7 < {}^+t^* < 2.0$).

b In some countries the number of cases for age group 0 is greater than for the other age groups. This is because for age group 0 we found more updated information that enabled us to extend
the series by one or two years.

c Deaths by violence in Guatemala exhibit a very irregular pattern that yields implausible large (or small) responses.

TABLE 1d: EFFECTS BY LAG FOR SELECTED CAUSES OF AGE GROUP 5-14[a, b]

			EFFECTS ON:				
COUNTRY	LAG	ALL	INFECTIOUS	RESPIRATORY	VIOLENCE	ILL-DEFINED	DIARRHEA
CHILE (n = 31)	0	.36	.75	1.16+	.62+	- .69	1.19
	1	- .18	- .17	-1.53*	.60	- .62	- .47
	2	- .13	- .76	.95	- .40	-1.03	.75
	3	.11	- .16	.87	- .10	.41	-1.56
	4	- .04	- .44	.11	.36	- .69	.16
Adj R^2		- .08	- .01	.05	.11	- .04	- .09
COSTA RICA (n = 29)	0	.27	- .59	-1.54	1.40*	- .10	-1.70
	1	- .86	-4.41*	1.26	.47	- .48	-2.21
	2	.53	2.09	- .37	-1.66+	.61	-1.56
	3	- .54	- .38	-2.28	.64	- .55	- .58
	4	.35	1.11	2.35	- .13	.52	3.19
Adj R^2		- .10	.57	- .03	.39	- .25	.55
ECUADOR (n = 23)	0	- .37	- .73	- .28	.08	- .60+	- .18
	1	.08	.32	- .04	- .20	- .50	- .33
	2	- .51	- .83	- .58	.37	.06	- .43
	3	.32	.44	- .47	- .22	.91	- .38
	4	- .15	- .20	.38	- .01	- .76+	- .24
Adj R^2		.14	.02	.02	- .19	.28	- .10

COUNTR	COUNTRY	LAG	ALL	EFFECTS ON: INFECTIOUS	RESPIRATORY	VIOLENCE	ILL-DEFINED	DIARRHEA
VENEZU (n = 30)	URUGUAY (n = 28)	0	.58*	1.81	2.65	.19	- .33	9.10*
		1	- .65+	.23	-3.86	- .60	2.67+	-5.03
		2	.12	-1.57	3.07	.89	-3.37+	-2.47
		3	- .81+	-1.21	-1.40	-1.57*	.96	3.72
		4	.46	1.47	-1.43	.67	.57	-2.39
Adj R^2	Adj R^2		.34	.09	- .02	.19	.03	.08
TRINIDA	GUATEMALA (n = 23)	0	- .52	-2.71+	-1.21+	c	- .69	-4.14+
(n = 29)		1	-2.92	.30	-2.57*	--	-1.35	4.12
		2	2.77	.85	1.96	--	1.91	-1.64
		3	- .72	.18	1.10	--	-2.20+	- .47
		4	- .92	-1.68	-1.99*	--	.70	-1.45
Adj R^2	Adj R^2		.06	.11	.62	--	.19	- .02
FOOTNO								
a The syn	MEXICO (n = 25)	0	- .40	-1.50	- .35	.09	- .46	- .78
The sym								
b In some								
the serie		1	- .22	- .86	-1.07	- .48	-1.57	-1.69
c Deaths		2	.41	.72	.11	.48	.19	1.58
		3	- .20	- .30	.63	- .87*	.43	- .79
		4	.30	1.13	.51	.40	- .53	2.34
	Adj R^2		- .14	.01	- .10	- .07	- .01	.09
	PANAMA (n = 30)	0	- .49	- .40	-1.81+	- .75	- .58	- .46
		1	.38	- .11	.90	.54	.87	.15
		2	.83	1.68	2.81+	- .19	.98	.72
		3	- .93	-1.44	-2.91	.33	- .89	- .14
		4	- .09	.14	.53	- .31	.83	.32
	Adj R^2		- .11	- .16	- .03	- .14	- .12	- .19

TABLE 1e: EFFECTS BY LAG FOR SELECTED CAUSES OF DEATHS AND COUNTRIES: AGE GROUP 1-4[a,b]

			EFFECTS ON:						
COUNTRY	LAG	ALL	INFECTIOUS	CHRONIC	RESPIRATORY	VIOLENCE	ILL-DEFINED	RESPIRATORY TB	DIARRHEA
CHILE (n = 31)	0	.35+	- .03	.80	1.73*	.43	-1.34*	.06	.20
	1	- .09	- .30	- .03	-2.72*	.16	- .47	- .37	- .07
	2	.14	- .04	.01	1.31	.30	- .17	.08	- .41
	3	- .04	- .63*	.30	- .29	.25	- .17	- .91*	.74
	4	.23	- .21	.39	1.21	.14	- .62	- .16	.02
Adj R^2		- .03	.05	- .11	.22	.11	.11	.08	.03
COSTA RICA (n = 29)	0	.30+	- .37	.31	.76	.80	.09	1.03	-3.20
	1	- .14	-1.21	1.10	- .20	.64	1.09	- 1.14	-3.20
	2	- .19	.32	-.19	-1.32	-1.07	1.77	- .18	2.11
	3	- .08	- .41	- .54	.11	.41	-1.19	-1.07	-2.71
	4	.10	.25	.49	.49	- .08	- .33	1.41	2.47
Adj R^2		.02	.08	- .19	- .16	.58	- .03	.04	.73
ECUADOR (n = 23)	0	.11	.12	.13	- .08	.02	- .12	.28+	.33
	1	- .19+	.02	- .33	.26	- .24	- .02	- .12	.32
	2	- .02	- .11	.43	- .94+	.43+	- .58*	.02	- .29
	3	.11	- .06	- .38	- .61	.15	.31	- 1.06	- .15
	4	- .04	.10	.06	.43	- .16	.34*	.14	.26
Adj R^2		- .06	- .23	- .16	.31	.11	.57	- .10	.09

			EFFECTS ON:						
COUNTRY	LAG	ALL	INFECTIOUS	CHRONIC	RESPIRATORY	VIOLENCE	ILL-DEFINED	RESPIRATORY TB	DIARRHEA
URUGUAY (n = 28)	0	.02	1.21*	- .25	.52	- .26	.41	1.56	3.58*
	1	.14	- .93	- .04	- .37	.58	.18	-1.24	-1.61
	2	- .29*	- .99	- .21	-2.00	.39	- .16	-1.54	- .96
	3	.40*	1.02	1.04*	3.67*	- .01	.05	.90	3.51+
	4	- .42*	-1.44*	-1.44*	-2.41+	- .43	.13	-2.80*	-2.21+
Adj R^2		.20	.25	.22	.01	.14	.06	.29	.17
GUATEMALA (n = 23)	0	- .70	-2.67+	1.42	-1.50+	c	-1.47	- .70	-3.36+
	1	.18	1.54	-2.15	- .27	--	.39	-2.34	3.50
	2	1.41	1.11	1.54	.22	--	2.12	2.59	.18
	3	-1.00	-1.98	-1.00	.68	--	-3.48	-1.63	-2.53
	4	- .01	.19	.57	-1.14	--	1.45	.35	- .65
Adj R^2		- .20	- .04	- .18	.23	--	.08	.11	- .10
MEXICO (n = 25)	0	.10	- .29	.14	-1.05	.64	- .20	- .79*	- .57
	1	.13	-1.15*	.65+	- .39	.30	- .93	- .79+	-1.44
	2	- .07	.51	- .22	-1.20	.40	- .21	- .10	.60
	3	.18	- .44	.63+	.95	- .10	- .03	- .21	- .49
	4	- .06	.95	- .23	.22	- .10	.12	.40	1.97
Adj R^2		- .06	.13	.25	.11	.07	- .20	.66	.10
PANAMA (n = 30)	0	- .05	- .05	.04	- .12	- .01	.50	- .02	- .83
	1	- .26+	.05	- .22	.98	- .84*	.41	.47	- .90
	2	.03	- .28	- .78	- .24	.85+	- .34	-1.48+	- .01
	3	.31	.51	2.66*	.98	.16	.89	1.29	1.92
	4	- .22	.87	-1.58+	.67	-1.23*	.51	.52	.54
Adj R^2		.00	- .09	- .01	- .11	.26	- .13	.17	- .07

			EFFECTS ON:						
COUNTRY	LAG	ALL	INFECTIOUS	CHRONIC	RESPIRATORY	VIOLENCE	ILL-DEFINED	RESPIRATORY TB	DIARRHEA
VENEZUELA (n = 30)	0	.07	- .68*	.06	.19	.02	2.81*	- .30	- .92
	1	.15	.42	.10	.07	.47+	-1.82	.17	.34
	2	.14	.07	.77*	.42	.23	- .24	- .29	- .34
	3	.01	- .27	- .34	-1.42+	.11	1.45	- .41	- .57
	4	.04	.29	.00	- .20	- .22	-1.10	.09	.82
Adj R^2		.15	.06	.13	- .01	.25	.16	.05	.01
TRINIDAD (n = 29)	0	- .14	- .82	- .32	-1.10	- .24	- .70	-2.19	.92
	1	.02	-1.08	- .01	1.85	.13	-2.44	-4.33+	-4.00+
	2	.17	1.42	- .78	-1.26	.74	3.80+	2.32	4.88+
	3	- .12	.13	- .06	- .59	- .71	- .20	- .48	-1.65
	4	.08	.47	-1.14	- .12	- .18	.98	2.86	- .55
Adj R^2		.16	.25	.19	- .03	- .09	.14	.24	- .00

FOOTNOTES TO TABLES 1a THROUGH 1f

a The symbol "*" indicates statistical significance at $p < .05$ ($^*t^9 > 2.00$).
The symbol "+" indicates statistical significance at $.05 < p < 10$ ($1.7 < {}^*t^9 < 2.0$).

b In some countries the number of cases for age group 0 is greater than for the other age groups. This is because for age group 0 we found more updated information that enabled us to extend the series by one or two years.

c Deaths by violence in Guatemala exhibit a very irregular pattern that yields implausible large (or small) responses.

TABLE 1f: EFFECTS BY LAG FOR SELECTED CAUSES AGE GROUP 1-4[a,b]

			EFFECTS ON:						
COUNTRY	LAG	ALL	INFECTIOUS	CHRONIC	RESPIRATORY	VIOLENCE	ILL-DEFINED	RESPIRATORY TB	DIARRHEA
CHILE (n = 31)	0	.29*	.09	.49	1.61*	.55*	-.72*	-.17	-.02
	1	-.48*	-.39*	-.17	-2.62*	-.24	-1.06*	-.42	-.49
	2	.40*	-.42*	.34	1.88*	.31	.24	-.09	-.56
	3	-.14	.25	-.24	-.77	-.06	.06	-.51+	-.11
	4	.16	-.07	.31	1.31	.16	-.61+	-.26	-.18
Adj R^2		.07	.28	-.08	.19	.04	.25	.03	.05
COSTA RICA (n = 29)	0	-.11	-2.77*	.07	.68	.11	-.96	-.03	-6.41*
	1	-.17	-.26	.76	-1.36	.98	1.67	-.90	-.19
	2	-.09	1.54	-1.11	-.38	-.85	1.35	2.43	.14
	3	.18	-1.13	-1.50	.54	1.63	-1.54	-3.36+	.85
	4	-.10	.63	1.89*	-.34	-1.15	.64	1.69	.12
Adj R^2		-.03	.56	-.02	-.06	.10	.14	-.02	.76
ECUADOR (n = 23)	0	.05	.33	.07	-.07	.29	-.15	.31	.44
	1	-.12	.07	-.14	.28	-.16	.02	.54	.12
	2	-.24	.18	.09	-.96+	.03	-.61	.07	-.06
	3	-.23	-.91*	-.14	-.52	.09	-.05	-1.01*	-1.56*
	4	.26	.47	-.30	.62	-.04	.60*	.45	.85
Adj R^2		.62	.01	-.09	.23	-.07	.59	.24	.08

			EFFECTS ON:						
COUNTRY	LAG	ALL	INFECTIOUS	CHRONIC	RESPIRATORY	VIOLENCE	ILL-DEFINED	RESPIRATORY TB	DIARRHEA
URUGUAY (n = 28)	0	- .09	- .65+	- .17	.97	.40	.34	- .02	1.89*
	1	.10	.49	-1.09+	- .83	1.00	- .19	- .04	-1.93+
	2	- .54*	- .27	- .04	-1.75	.17	- .54	.22	1.19
	3	.64*	.51	1.07*	2.08+	.40	.83	-1.14	.28
	4	- .40*	.58	- .77	-1.17+	- .51	.03	.29	- .72
Adj R^2		.18	.15	.33	.13	.55	- .01	- .12	.04
GUATEMALA (n = 23)	0	- .72	- .76	.26	- .62	--	-1.80+	.27	- .82
	1	- .96	- .69	- .74	-1.33	--	.13	-2.41	- .51
	2	1.54+	1.89	.26	- .00	[c]	2.10+	1.49	1.84
	3	-1.62+	-1.02	-1.61	1.22	--	-2.47+	1.14	-2.15
	4	.32	- .84	1.18	-1.36+	--	.38	-1.64	- .44
Adj R^2		.27	- .17	- .07	.23	--	.20	- .11	- .18
MEXICO (n = 25)	0	- .35+	- .49	- .21	-1.49+	.13	- .52	- .96*	- .54
	1	.34	- .64	.06	.50	- .10	- .65	- .52	- .86
	2	- .39	.33	- .06	-1.58	.31	- .26	- .34	.49
	3	.27	- .23	.24	1.08	- .23	.20	.23	- .44
	4	- .03	.38	- .10	.95	- .02	.55	- .24	1.13
Adj R^2		.00	- .05	- .14	.32	- .23	- .14	.35	.07
PANAMA (n = 30)	0	.18	.14	.34	.12	.25	.20	.26	- .23
	1	.12	.37	- .67	.45	.12	.41	- .09	.52
	2	.10	- .14	1.63+	.20	- .22	.42	- .06	.43
	3	.46	1.67+	-1.80+	.96	- .57	.30	1.87+	1.59
	4	.08	.66	1.01	- .00	1.21+	.52	- .54	- .48
Adj R^2		- .09	- .04	- .06	- .15	.08	- .11	- .05	- .14

			EFFECTS ON:						
COUNTRY	LAG	ALL	INFECTIOUS	CHRONIC	RESPIRATORY	VIOLENCE	ILL-DEFINED	RESPIRATORY TB	DIARRHEA
VENEZUELA (n = 30)	0	.02	-1.24	.07	.51	.11	2.56*	- .63	-2.04*
	1	- .18	.61	- .43	- .44	- .48	-1.91+	- .37	1.90*
	2	.20	.56	.77	.28	.55	.32	.82	.09
	3	- .02	- .37	- .06	-1.26+	- .10	1.34	- .07	-2.30*
	4	- .11	.33	- .50	- .15	- .50+	-1.24	- .42	1.92+
Adj R^2		- .13	.20	- .08	.03	.00	.20	- .07	.41
TRINIDAD (n = 29)	0	- .30	- .06	- .29	- .48	1.32+	-1.21+	-1.28	- .49
	1	.07	- .09	.51	- .10	-2.43*	- .77	1.67	-1.38
	2	.10	- .59	- .75	.76	2.02+	2.25*	1.52	.28
	3	- .12	2.05	-2.04	-1.92	-1.31	.05	- .29	1.40
	4	- .17	-1.77	1.08	- .06	.50	- .44	- .55	-2.15
Adj R^2		- .14	- .09	.15	- .04	.08	.27	.10	- .12

FOOTNOTES TO TABLES 1a THROUGH 1f

a The symbol "*" indicates statistical significance at $p < .05$ ($^*t^* > 2.00$).
The symbol "+" indicates statistical significance at $.05 < p < 10$ ($1.7 < {}^*t^* < 2.0$).

b In some countries the number of cases for age group 0 is greater than for the other age groups. This is because for age group 0 we found more updated information that enabled us to extend the series by one or two years.

c Deaths by violence in Guatemala exhibit a very irregular pattern that yields implausible large (or small) responses.

TABLE 2: FREQUENCIES OF RESPONSES ACCORDING TO THEIR DIRECTION AND SIGNIFICANCE, BY LAG, AGE GROUP, AND CAUSE OF DEATH*

AGE GROUP	CAUSE OF DEATH	LAG 0			LAG 1			LAG 2			LAG 3			LAG 4		
		SIG	NOT SIG	NOT SIG	SIG	NOT SIG	NOT SIG	SIG	NOT SIG	NOT SIG	SIG	NOT SIG	NOT SIG	SIG	NOT SIG	NOT SIG
ALL																
	All	0	6	0	1	5	0	1	3	0	0	5	1	1	5	0
	Infections	1	5	0	1	6	0	0	4	0	0	6	0	0	6	1
	Chronic	0	2	0	0	5	1	0	4	1	0	6	1	2	3	0
	Respiratory	0	5	1	1	6	0	1	3	1	1	4	1	2	4	0
	Violence	0	3	1	0	3	3	1	1	1	0	1	0	2	4	0
	Ill-defined	3	3	2	3	3	0	1	3	1	1	3	1	0	2	0
	Respiratory TB	0	5	0	1	7	0	0	6	0	1	5	1	0	4	0
	Diarrheas	1	5	1	2	5	0	0	5	0	0	6	0	0	5	1
0																
	All	1	4	0	1	6	0	0	4	0	1	4	0	0	5	1
	Infections	2	5	0	1	7	0	1	3	0	1	7	0	1	4	0
	Respiratory	3	2	2	1	6	0	1	5	0	2	4	0	0	5	1
	Violence	0	3	0	0	4	1	1	4	0	2	5	0	0	3	1
	Ill-defined	1	4	2	1	3	1	0	4	1	2	3	0	2	3	0
	Diseases of infancy	0	3	0	1	4	0	0	5	1	0	4	0	1	2	0
	Diarrheas	1	3	0	1	5	0	0	3	1	1	4	0	1	5	1
1-4																
	All	0	6	1	1	4	0	0	4	1	0	4	0	0	6	0
	Infections	0	6	1	1	7	0	1	3	0	1	4	0	0	6	1
	Respiratory	1	4	3	2	7	0	0	2	2	0	6	0	1	4	0
	Violence	0	3	1	2	2	0	0	4	0	0	3	0	0	5	0
	Ill-defined	1	3	0	1	5	0	1	1	1	0	6	0	2	2	0
	Diarrheas	0	8	1	2	4	0	0	3	0	1	4	0	0	5	1

AGE GROUP	CAUSE OF DEATH	LAG 0			LAG 1			LAG 2			LAG 3			LAG 4		
		-		+	-		+	-		+	-		+	-		+
		SIG	NOT SIG	NOT SIG	SIG	NOT SIG	NOT SIG	SIG	NOT SIG	NOT SIG	SIG	NOT SIG	NOT SIG	SIG	NOT SIG	NOT SIG
5-14																
	All	0	5	1	1	6	0	0	3	0	1	5	0	0	7	0
	Infections	1	6	0	2	4	0	0	4	1	0	7	0	0	5	0
	Respiratory	1	4	1	2	3	0	0	2	0	0	6	0	1	2	0
	Violence	1	1	1	0	5	0	1	4	0	2	4	1	0	4	0
	Ill-defined	2	6	1	0	6	1	1	1	0	1	2	1	3	3	0
	Diarrheas	1	3	1	0	7	0	0	5	0	1	7	0	0	4	0
15-64																
	All	0	3	2	2	2	0	1	3	0	0	5	1	1	4	0
	Infections	2	5	0	1	4	0	0	4	0	1	5	0	1	1	0
	Chronic	0	2	0	0	7	1	1	5	0	0	5	3	2	2	0
	Respiratory	1	4	1	0	7	0	1	4	1	1	3	1	1	3	1
	Violence	0	3	1	1	2	0	1	0	2	0	3	0	1	7	0
	Ill-defined	1	4	1	1	6	0	2	3	0	1	4	0	1	2	1
	Respiratory TB	1	4	1	2	6	0	2	4	0	2	4	1	1	2	0
	Diarrheas	2	5	1	1	4	0	0	5	1	1	5	1	0	3	0
65+																
	All	1	3	4	1	4	0	1	4	1	2	4	0	1	4	1
	Infections	3	3	0	1	4	0	1	3	0	1	4	1	0	4	0
	Chronic	0	3	0	1	5	0	0	4	1	0	7	0	1	3	1
	Respiratory	1	3	1	1	5	0	1	4	1	1	3	1	1	6	1
	Violence	0	1	3	1	5	0	0	3	1	0	5	0	1	4	1
	Ill-defined	3	3	1	2	3	0	2	2	0	1	4	1	1	6	1
	Respiratory TB	1	5	0	0	8	0	0	4	6	3	3	1	0	6	0
	Diarrheas	2	5	1	1	5	1	0	3	0	2	3	0	0	5	1

*SIG: The estimated coefficient is significant at p-level of .10 or less ($|t| > 1.7$)
NOT SIG: The estimated coefficient is not significant at p-level of .10 or less

TABLE 3: SUMMARY OF RESPONSES ACCORDING TO THEIR DIRECTION AND SIGNIFICANCE, BY LAG, AGE GROUP, AND CAUSE OF DEATH

A: Proportion of negative effects (negative significant effects) by lag and age

AGE	LAG 0	1	2	3	4
All	.52 (.04)	.68 (.13)	.46 (.06)	.54 (.04)	.43 (.10)
0	.44 (.11)	.57 (.08)	.43 (.04)	.50 (.13)	.44 (.07)
1-4	.44 (.03)	.53 (.13)	.26 (.03)	.40 (.03)	.43 (.04)
5-14	.43 (.08)	.50 (.07)	.29 (.03)	.50 (.07)	.40 (.06)
15-64	.51 (.10)	.64 (.11)	.50 (.11)	.56 (.08)	.44 (.11)
65+	.51 (.15)	.65 (.11)	.44 (.07)	.60 (.14)	.60 (.07)

B: Proportion of negative effects (negative significant effects) by cause and age

AGE	CAUSE All	Infectious	Chronic	Respiratory	Violence	Ill-defined	Respiratory TB	Diarrhea
All	.60 (.07)	.64 (.04)	.49 (.07)	.60 (.11)	.33 (.07)	.49 (.18)	.53 (.04)	.64 (.07)
0	.58 (.07)	.71 (.38)	.44 (.04)[a]	.64 (.16)	.49 (.07)	.51 (.13)	-[b]	.53 (.09)
1-4	.56 (.02)	.64 (.07)	NA	.60 (.09)	.36 (.02)	.49 (.11)	-[b]	.60 (.07)
5-14	.62 (.04)	.64 (.07)	NA	.47 (.09)	.49 (.09)	.56 (.16)	-[b]	.62 (.04)
15-64	.47 (.09)	.51 (.11)	.53 (.07)	.56 (.09)	.40 (.07)	.56 (.13)	.62 (.18)	.58 (.09)
65+	.47 (.13)	.53 (.13)	.53 (.19)	.58 (.11)	.44 (.04)	.60 (.20)	.67 (.09)	.58 (.11)

[a] Diseases of infancy

[b] The frequency of deaths due to respiratory TB in the age groups 0, 1-4 and 5-14 was too low and did not yield useful estimates.

NA = not applicable

TABLE 4: SUM OF EFFECTS (NET RESPONSE) OF LAGS 0 TO 4 BY CC CAUSES OF DEATHS, AND AGE GROUPS[a,b,c,d]

COUNTRY	AGE GROUP	CAUSE OF DEATH							
		ALL	INFECTIOUS	CHRONIC[a]	RESPIRATORY	VIOLENCE[b]	ILL-DEFINED	RESPIRATORY TB[c]	DIARRHEA
CHILE	ALL	.29	- .87	1.13	1.12*//	1.11	-2.58	-1.62*//	- .75
	0	- .24	-1.89	- .43	- .01	.66	-3.30	--	-1.72
	1-4	.37	- .24	--	.24	.53	-2.19	--	-1.57
	5-14	.16	- .78	--	1.56	1.08	-2.61	--	- .69
	15-64	.59	-1.22	1.46	-1.18	1.27	-2.78	-1.29	-1.00
	65+	.23	- .41	.74	1.41	.72	-2.08	-1.45	-1.32
COSTA RICA	ALL	- .35	-1.53*//	.02	- .75	.63	.30	- .07	-1.93//
	0	-2.17	-5.37	- .51	-3.36	-4.04	-3.65	--	-5.28
	1-4	- .95	-1.36	--	-1.13	- .12	- .31	--	-1.40
	5-14	- .25	-2.16	--	- .58	.72	.00	--	-2.85
	15-64	- .02	-1.42	- .04	- .45	.70	1.44	.04	-4.52
	65+	- .29	-1.99	.11	- .85	.72	1.16	- .16	-5.49
ECUADOR	ALL	- .24	- .72	.19	- .82	.40	- .38	.19	- .55
	0	.08	- .36	1.64	- .66	.70	- .16	--	- .13
	1-4	- .72	- .91	--	- .73	.03	- .92	--	- .75
	5-14	- .63	- .99	--	-1.00	.04	- .88	--	-1.20
	15-64	- .04	.07	- .09	- .93	.19	- .06	.17	- .43
	65+	- .27	.14	- .41	- .64	.20	- .19	.36	- .21
URUGUAY	ALL	- .32*//	- .88//	- .93	-1.13*	.25	.52	- .73	- .74
	0	- .36	-1.46	.05	-1.57	.88	.68	--	- .51
	1-4	.08	.67	--	- .34	.14	1.01	--	- .22
	5-14	- .31	.75	--	- .95	- .42	.52	--	2.93
	15-64	- .15	-1.11	- .90	- .58	.24	.61	-3.10	2.33
	65+	- .29	.66	-1.00	- .81	.67	.48	- .70	3.07

COUNTRY	AGE GROUP	A
TRINIDAD	ALL	-
	0	-
	1-4	
	5-14	-
	15-64	
	65+	-

[a]For age group 0 "chronic" diseas

[b]In the case of Guatemala estimat

[c]Deaths due to respiratory TB in (

[d]For a definition of the three stati

*Sum of effects significantly differe

*Sum of negative effects significant

#F-statistics for inclusion of all lags

COUNTRY	AGE GROUP	CAUSE OF DEATH							
		ALL	INFECTIOUS	CHRONIC[a]	RESPIRATORY	VIOLENCE[b]	ILL-DEFINED	RESPIRATORY TB[c]	DIARRHEA
GUATEMALA	ALL	- .70	-1.37	.13	-1.65	--	-1.01	-1.72	-1.15
	0	- .15	- .11	4.20	-2.17	--	-2.89	--	.62
	1-4	-1.28	-1.33	--	-1.32	--	- .55	--	-1.34
	5-14	-2.31	-3.06	--	-2.69	--	-1.63	--	-3.09
	15-64	- .12	-1.81	.34	-2.01	--	-3.89	-2.86	-2.28
	65+	-1.45	-1.41	- .64	-2.09	--	-1.65	-1.71	-2.08
MEXICO	ALL	- .20	-1.03#	.68	-1.08	.52	-1.64	-1.42#	- .99#
	0	-1.11	-1.52	.60	-1.91	-1.18	-4.76	--	-1.32
	1-4	- .42	- .32	--	- .32	- .84	-2.69	--	- .14
	5-14	- .11	- .81	--	- .16	- .38	-1.94	--	.66
	15-64	.27	- .42	.96	-1.47	1.13	-1.25	-1.50	.07
	65+	- .15	- .64	- .07	-1.56	.10	- .69	-1.83	- .23
PANAMA	ALL	.68	.13	.43	1.36	- .33	1.04	.25*	.35
	0	.41	.26	1.16	2.09	2.29	.78	--	.44
	1-4	.58	.22	--	- .19	-1.58	1.75	--	1.10
	5-14	- .30	- .12	--	- .49	- .38	1.20	--	.58
	15-64	- .20	1.10	.13	2.27	-1.07	1.98	.69	.72
	65+	.94	1.38	.51	1.73	.79	1.85	.54	1.83
VENEZUELA	ALL	.05	-1.40*#	.81	-1.25	1.17	.02	- .96	-1.58*#
	0	- .32	-1.17	.54	-2.17	-2.38	-2.59	--	-1.21
	1-4	-1.30	-2.65	--	-1.85	- .29	-1.72	--	-1.69
	5-14	- .11	-2.19	--	- .30	.59	-1.22	--	-3.71
	15-64	.38	- .17	.59	- .94	.60	-1.10	- .75	- .67
	65+	- .09	- .11	- .15	-1.05	- .42	1.08	- .67	- .44

TABLE 5: COMPARISONS OF SELECTED PARAMETER ESTIMATES OBTAINED WITH ALTERNATIVE INDEPENDENT VARIABLES

Country	Model		Causes of death							
			All	Infectious	Chronic	Respiratory	Violence	Ill-defined	Respiratory TB	Diarrhea
Chile	I	Net response	.29	- .87	1.13	1.12	1.11	-2.58	-1.62	- .75
		(Adj R^2)	- .02	- .11	- .13	.19	.11	.23	.17	- .17
	II	Net response	- .09	- .58	-1.32	- .98	.49	-1.02	-1.68	- .62
		(Adj R^2)	- .08	- .08	- .11	.11	.39	- .07	.14	- .13
	III	Net response	- .15	- .34	.07	- .69	- .03	- .12	- .08	- .58
		(Adj R^2)	.06	- .08	- .15	.27	- .02	- .16	- .12	- .06
Costa Rica	I	Net response	- .35	- 1.53	.02	- .75	.63	.30	- .07	-1.93
		(Adj R^2)	.21	.75	- .06	.06	.54	- .01	- .04	.62
	II	Net response	- .33	- 1.69	.23	.00	.38	2.08	- .18	-3.93
		(Adj R^2)	.36	.30	- .10	- .06	.40	.11	.05	.37
	III	Net response	.18	.74	.14	.45	- .18	- .22	- .22	.73
		(Adj R^2)	.19	.37	.08	.14	.27	- .02	.08	.37
Ecuador	I	Net response	- .24	.72	.19	- .82	.40	- .38	.19	- .55
		(Adj R^2)	.06	- .08	- .07	.11	.04	.49	.01	- .19
	II	Net response	.90	1.52	.75	.85	.54	.57	- .72	1.45
		(Adj R^2)	.35	.09	.05	.03	.04	.19	- .09	- .01
	III	Net response	.05	.07	.06	.06	- .08	.18	- .06	.04
		(Adj R^2)	- .12	- .20	- .02	- .22	.38	.16	.20	- .25

Country	Model		Causes of death							
			All	Infectious	Chronic	Respiratory	Violence	Ill-defined	Respiratory TB	Diarrhea
Uruguay	I	Net response	- .32	- .88	- .93	-1.13	.25	.52	- .20	- .73
		(Adj R^2)	.28	.24	.27	.14	.27	.10	- .03	.07
	II	Net response	- .17	- .68	.53	-2.71	.61	- .06	-1.79	- .83
		(Adj R^2)	.04	.00	- .09	- .06	.47	.04	.07	.04
	III	Net response	.02	.13	- .02	.14	- .01	- .01	- .03	.16
		(Adj R^2)	.14	.29	- .12	.07	.20	- .02	.01	- .02

I Results of model with GDP
II Results of model with private consumption
III Results with model with debt as proportion of exports

TABLE 6: ESTIMATED NET EFFECTS AND ESTIMATED PARAMETERS FOR A MODEL WITH A TRIANGULAR STRUCTURE FOR THE LAG PATTERN OF EFFECTS

Country	Parameter	Cause							
		All	Infectious	Chronic	Respiratory	Violence	Ill-defined	Respiratory TB	Diarrhea
Chile	bo	.033	- .092	- .054	.042	- .040	- .092	- .300	- .040
	R	- .002	.038	.015	- .001	.012	- .023	.039	.035
	Adj R^2	- .07	- .03	- .07	.08	- .07	- .05	.040	- .06
	Estimated net response	.15	- .23	- .18	.20	- .13	- .60	-1.27	.01
Costa Rica	bo	- .081	- .179	.013	- .116	- .195	.020	- .071	- .421
	R	.023	.008	- .032	.040	.077	.143	- .013	-.039
	Adj R^2	- .066	- .08	- .08	- .10	.04	.09	- .10	- .018
	Estimated net response	- .27	- .85	- .13	- .34	- .051	.96	- .28	-2.34
Ecuador	bo	.118	.149	.078	.107	.189	.123	.000	.020
	R	- .031	- .039	- .019	- .017	- .046	- .034	- .028	- .022
	Adj R^2	- .05	- .10	- .02	- .103	.050	- .05	.059	- .10
	Estimated net response	.40	.72	.38	.43	.67	.41	- .01	- .13
Uruguay	bo	- .053	- .575	- .017	- .618	.165	- .119	- .602	- .988
	R	- .006	- .049	- .042	.014	.004	.017	.033	- .072
	Adj R^2	- .05	.29	- .01	- .044	- .05	- .08	- .004	.26
	Estimated net response	- .30	-3.17	- .34	-3.01	.25	- .49	- 2.81	-5.37

TABLE 7: ESTIMATED NET EFFECTS IN MODELS CONTROLLING FOR ILL-DEFINED AND TIME PERIOD

Country Model		Cause of death							
		All	Infectious	Chronic	Respiratory	Violence	Ill-defined	Respiratory TB	Diarrhea
Chile									
I	Net response	.29	- .87	1.13	1.12	1.11	-2.58	-1.62	- .75
	(Adj R^2)	- .02	- .11	- .13	.19	.11	.23	.17	- .17
II	Net response	NA	- .66	1.02	.94	.83	NA	-1.47	.26
	(Adj R^2)	NA	- .09	- .17	.15	.10	NA	.13	.10
	Coefficient	NA	.24	- .04	- .07	- .10	NA	.06	.52+
III	Net response	.27	- .66	1.02	1.04	1.20	- 2.56	-1.51	- .32
	(Adj R^2)	.13	.02	- .13	.25	.14	.21	.16	.02
	Coefficient	.008*	.035*	.013	.022*	- .007	- .006	.027	.060*
Costa Rica									
I	Net response	- .35	-1.53	.02	- .75	.63	.30	- .07	-1.93
	(Adj R^2)	.21	.75	- .06	.06	.54	- .01	- .04	.62
II	Net response	NA	-1.46	.10	-1.12	.65	NA	- .07	-1.55
	(Adj R^2)	NA	.74	- .04	.50	.52	NA	- .10	.72
	Coefficient	NA	- .05	- .13	.47*	- .03	NA	- .03	- .46*
III	Net response	- .54	-1.39	.05	- .81	.67	.78	.00	1.73
	(Adj R^2)	.24	.75	- .12	.07	.51	- .05	- .10	.61
	Coefficient	.0055	.0064	.0019	.0043	.0014	- .016	.0039	.0092

Country Model		Cause of death							
		All	Infectious	Chronic	Respiratory	Violence	Ill-defined	Respiratory TB	Diarrhea
Ecuador									
I	Net response	- .24	.72	.19	- .82	.40	- .38	.19	- .55
	(Adj R^2)	.06	- .08	- .07	.11	.04	.49	.01	- .19
II	Net response	.07	- .04	- .08	- .35	.01	NA	.12	- .05
	(Adj R^2)	.45	.18	.05	.40	.26	NA	- .03	- .11
	Coefficient	.67+	1.51+	- .59*	1.13*	- .59*	NA	.15	1.28+
III	Net response	- .27	- .79	.24	- .88	.41	- .45	.20	- .65
	(Adj R^2)	.08	- .08	- .04	.15	.11	.46	- .04	- .08
	Coefficient	- .0041	- .0126	.0079	- .012	- .0063	.0026	.0032	- .036
Uruguay									
I	Net response	- .32	- .88	- .93	-1.13	.25	.52	- .20	- .73
	(Adj R^2)	.28	.24	.27	.14	.27	.10	- .03	.07
II	Net response	.43	-1.05	- .90	-1.00	.28	NA	- .93	-1.28
	(Adj R^2)	.53	.22	.24	.10	.23	NA	- .07	.15
	Coefficient	.25*	.30	- .09	- .16	- .05	NA	.32	1.04+
III	Net response	- .40	-1.02	- .99	-1.45	.16	.56	- .53	- .62
	(Adj R^2)	.42	.25	.24	.18	.26	.06	- .05	.03
	Coefficient	- .0037*	- .0064	- .0024	.0161	- .0052	- .0012	.0085	.0056

I Results of model with GDP
II Results of model with GDP and control for ill-defined
III Results of model with GDP and control for time dummy
* In all cases the 'coefficient' refers to the estimated regression coefficient of the control.
*Significant at less than .05
+Significant at less than .10

TABLE 8: COMPARISON OF OBSERVED AND EXPECTED NET RESPONSE FOR ALL DEATHS FOR SELECTED COUNTRIES[a], [b]

	Model	Actual Response	Expected Response
Chile	I	.29	.17
	II	- .09	-.34
	III	- .15	-.22
Costa Rica	I	- .35	-.54
	II	- .33	-.32
	III	.18	.19
Ecuador	I	- .24	-.35
	II	.90	1.02
	III	.05	.08
Uruguay	I	- .32	-.34
	II	- .17	-.31
	III	.02	.02

[a] See text for definition of "observed" and "expected" net response
[b] See table for 5 definition of models I, II and III

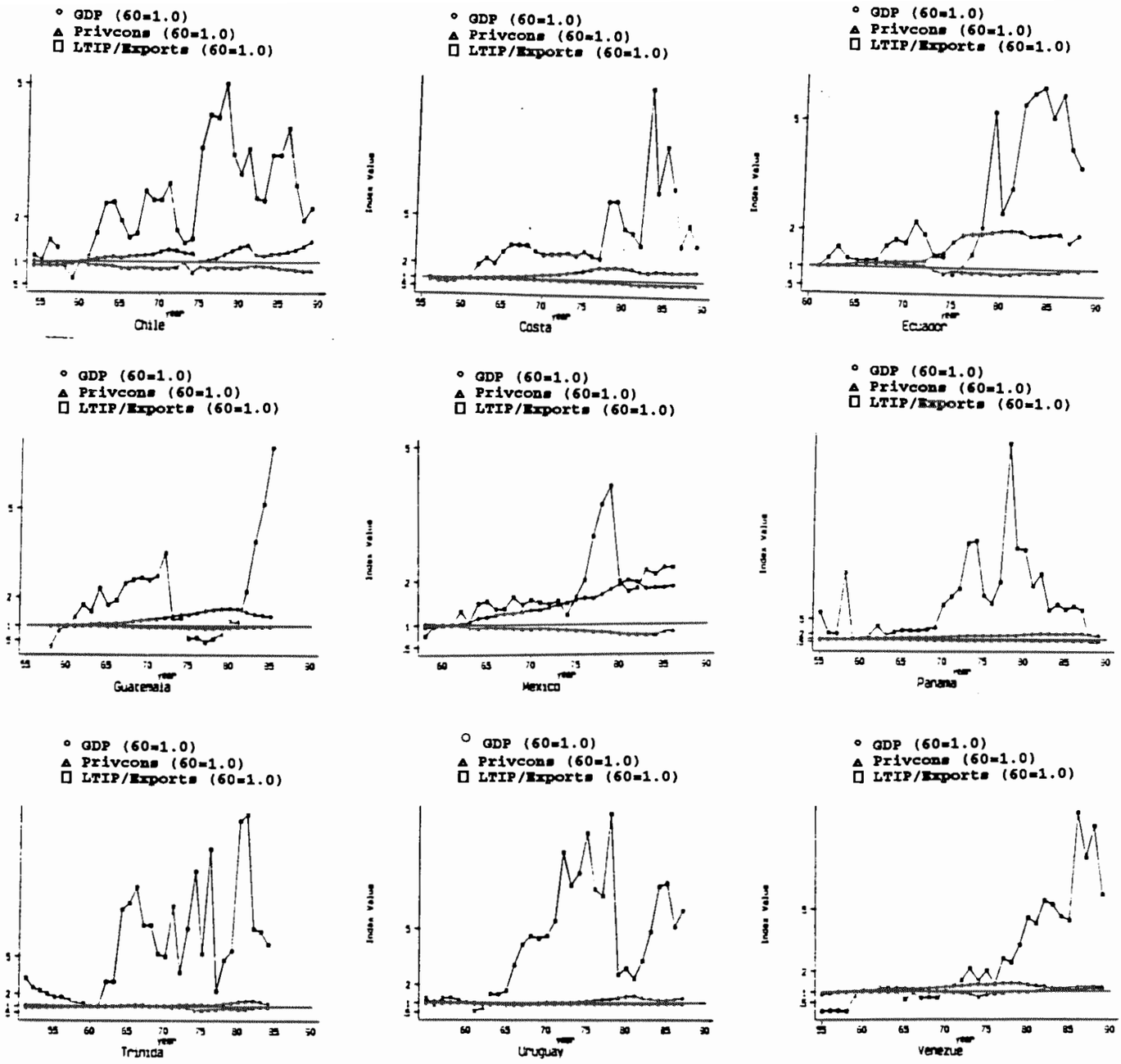

FIGURE 1: Index Value of Three Economic Indicators for Selected Latin American Countries

Footnote: GDP is the value (in 1970 U. S. dollars) of the real gross domestic product per capita. Privcons is the fraction of GDP represented by private consumption. LTIP/Exports is the ratio of long term interest payments to the value of exports

Source: Palloni and Hill data bank

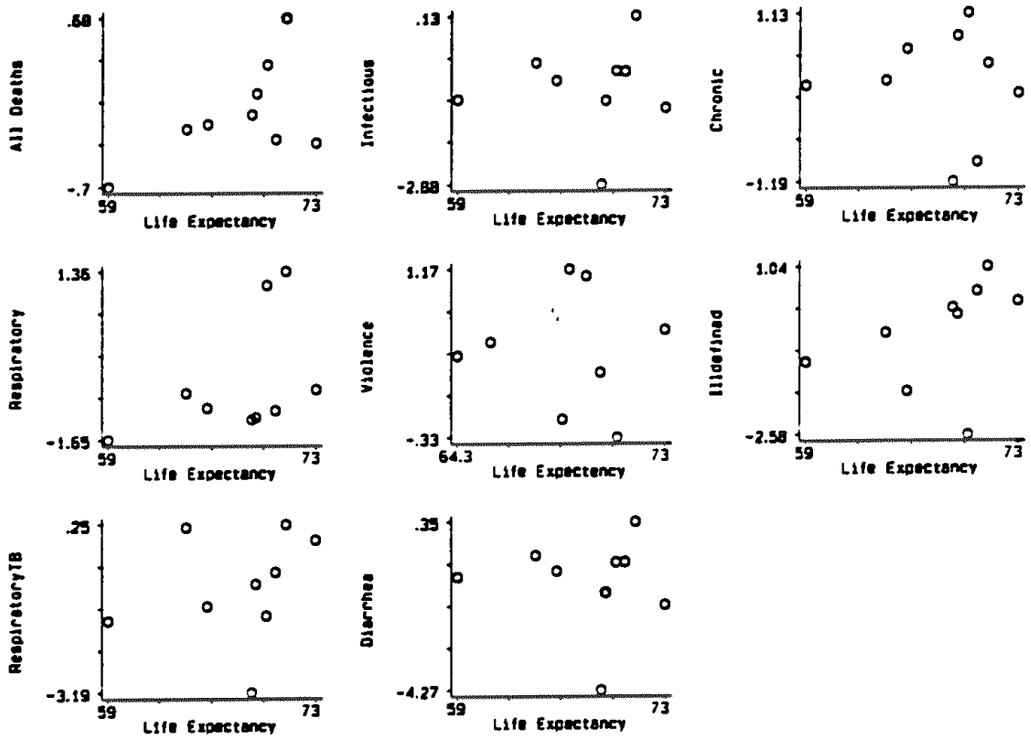

FIGURE 2: Relations Between Life Expectancy circa 1980-85 and Net Elasticities by Causes of Death

Source: Estimates of life expectancy from Palloni (1991); estimates for elasticities from Table 1

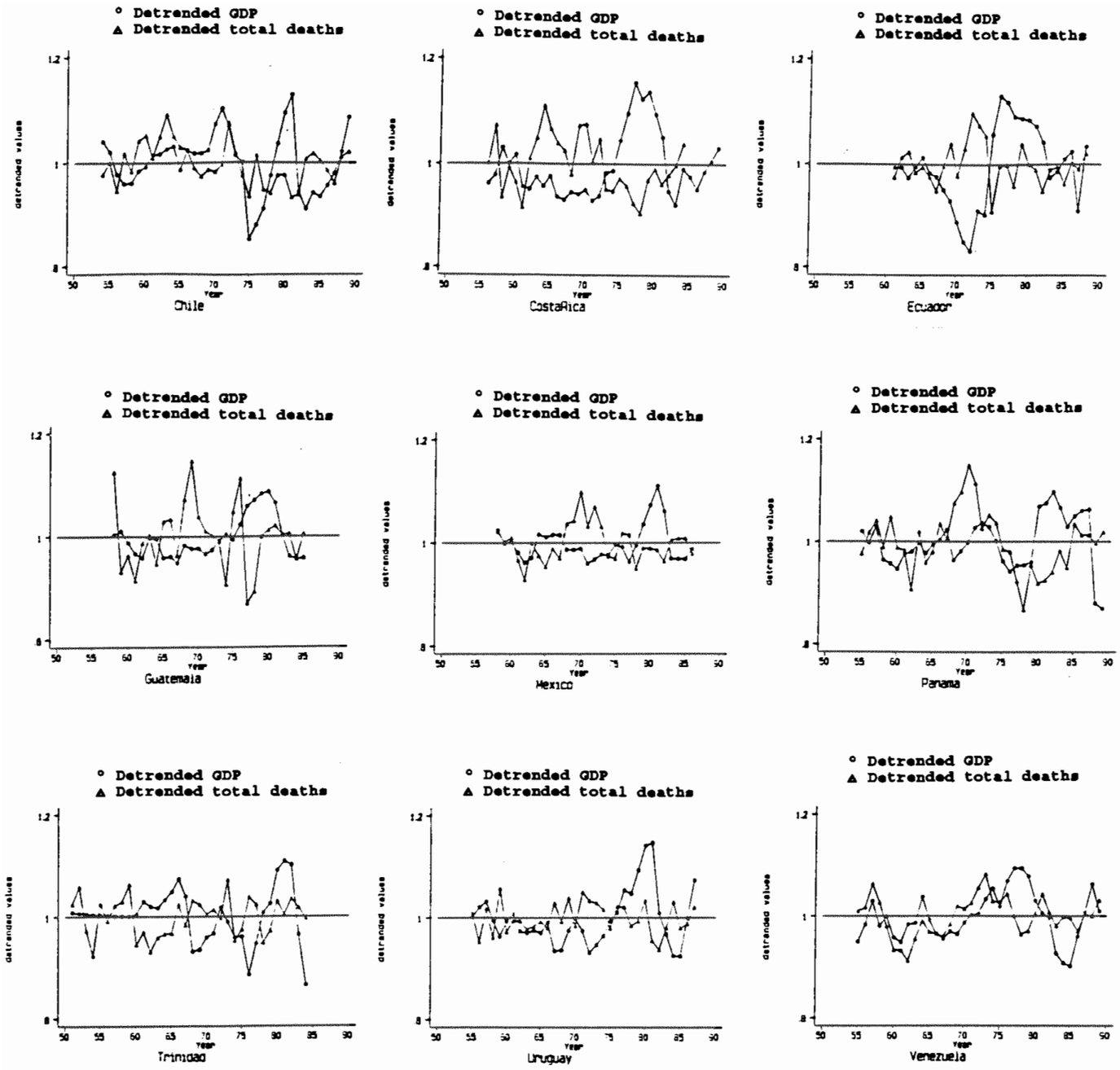

FIGURE 3: Detrended GDP and Detrended Total Deaths for Selected Countries

Source: Palloni and Hill data bank

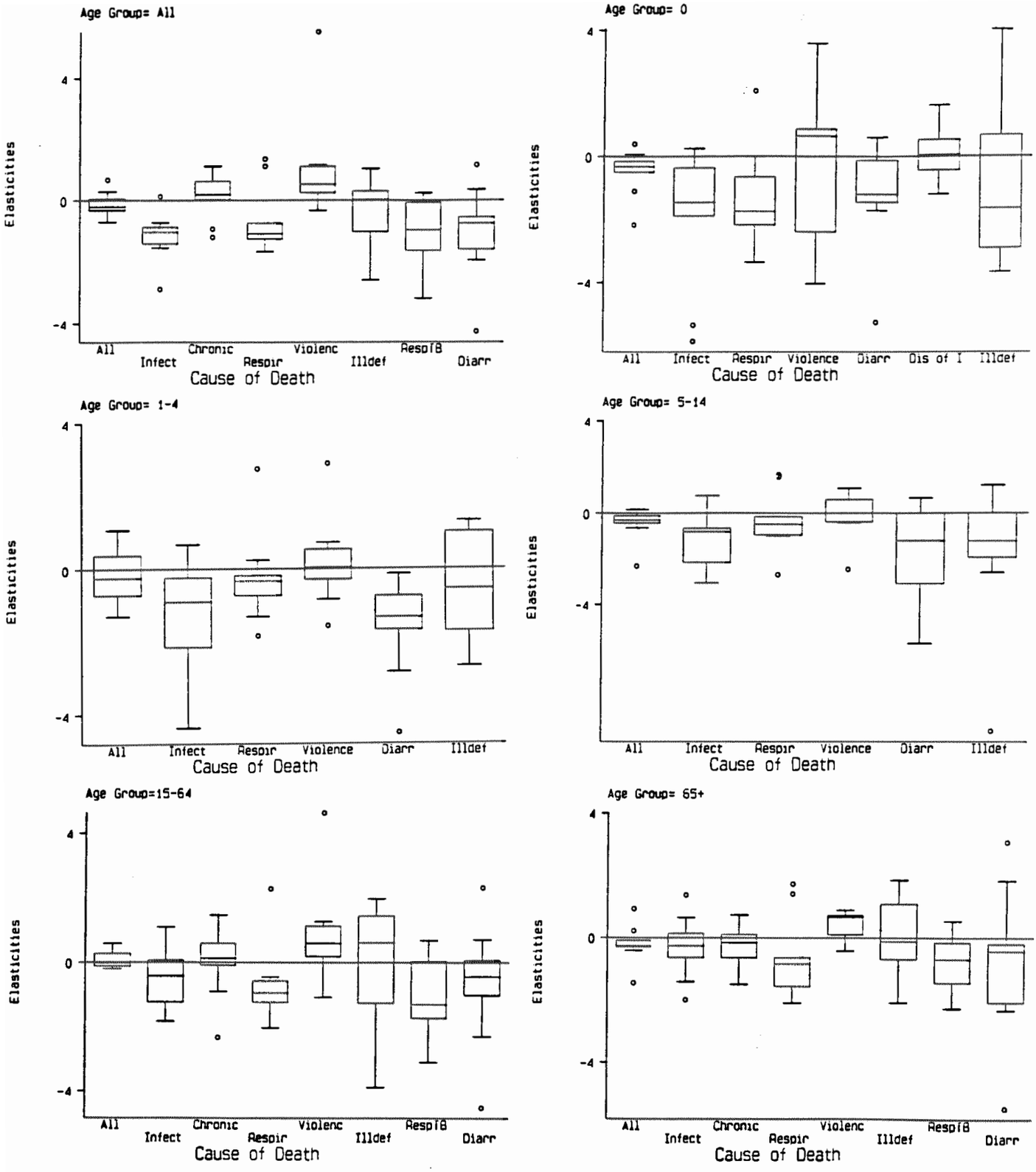

FIGURE 4: Box Plots of Net Elasticities by Causes of Death and Age Group

Source: Table 2 in the text

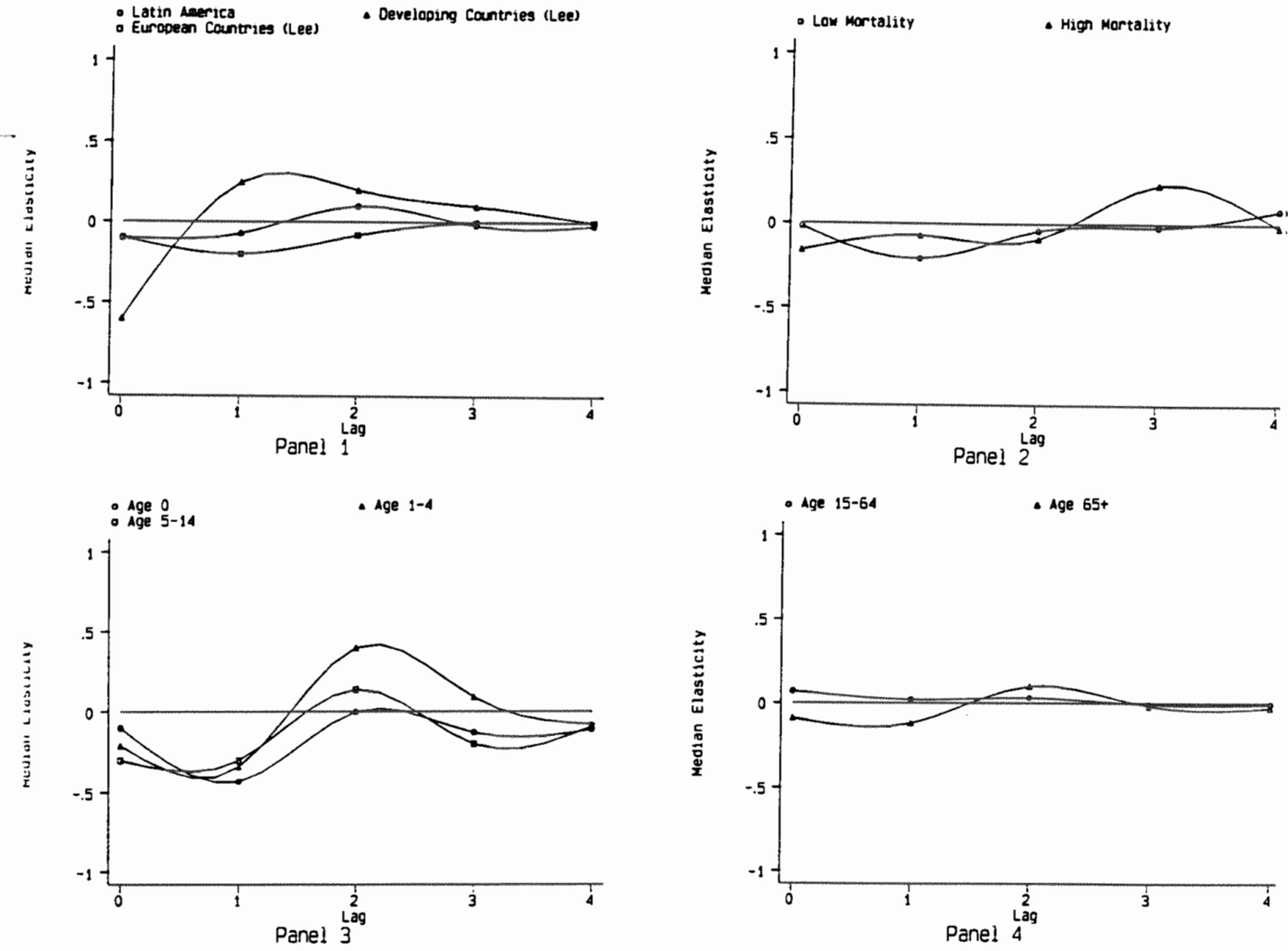

FIGURE 5: Estimated Elasticities by Lag in Different Social Contexts and for Different Age Groups

Footnote: In panel 1 the curves for "developing countries" and "Europe" were taken from Lee (1991) with signs reversed

Source: Table 1

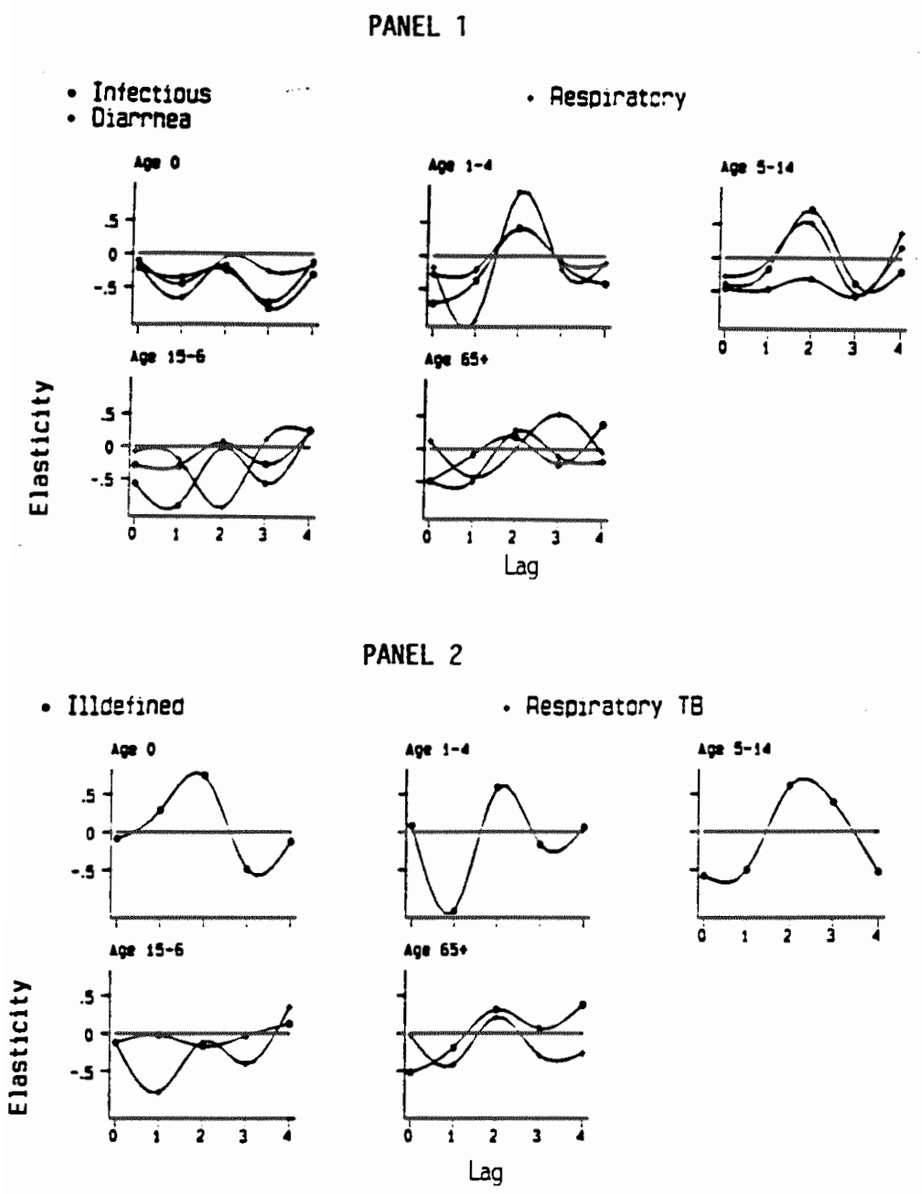

FIGURE 6: Lag-pattern of Elasticity by Age Group and Cause of Death

Source: Table 1

TABLE 8: COMPARISON OF OBSERVED AND EXPECTED NET RESPONSE FOR ALL DEATHS FOR SELECTED COUNTRIES[a], [b]

	Model	Actual Response	Expected Response
Chile	I	7.9 .29	.17
	II	- .09	-.34
	III	- .15	-.22
Costa Rica	I	- .35	-.54
	II	- .33	-.32
	III	.18	.19
Ecuador	I	- .24	-.35
	II	.90	1.02
	III	.05	.08
Uruguay	I	- .32	-.34
	II	- .17	-.31
	III	.02	.02

[a] See text for definition of "observed" and "expected" net response
[b] See table for 5 definition of models I, II and III

TABLE 9: DECOMPOSITION OF CHANGES IN LIFE EXPECTANCY INDUCED BY A TEN PERCENT DROP IN GDP[a]

	Contributions due to:					
Age groups	Infectious	Respiratory	Ill-defined	Respiratory TB	Diarrhea	Age specific [b]
0	.1230	.0760	- .005	--	.0850	.1990
1-4	.0070	.0010	.0009	--	.0040	.0089
5-14	.0070	.0033	.0008	--	.0008	.0111
15-64	.0007	.0011	-.0007	.0008	.0003	.0011
65+	.0013	.0072	-.0008	.0007	.0012	.0077
Cause specific[c]	.1390	.0886	-.0048	.0015	.1004	.2278

Life expectancy before downturn = 73.17

Life expectancy after downturn - 72.47

Estimated difference in life expectancies = .70

Calculated from table = .22

[a] The figures on this table were calculated using Mexico 1980 as a baseline, the median estimated age-cause-specific elasticities and the equation defined in the text

[b] Row sums (excluding respiratory TB and diarrheas which are included among infectious diseases)

[c] Column sums

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