El descenso de la mortalidad en América Latina: 1950-2005

The Decrease of Mortality in Latin America during the Twenty Century: A Reinterpretation[1]

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Resumen

El objetivo en este trabajo es construir una interpretación sobre las fuerzas que primero provocaron y después mantuvieron la caída veloz de la mortalidad en América Latina durante el periodo 1900-2000. En contraste con otros trabajos esta investigación se basa sobre datos que incluyen la mayor cantidad de países y años disponibles, hecho que provee una oportunidad única para formular conjeturas que son generalizables. Los estimadores se construyen a partir de estadísticas vitales y censos de población solamente y evitan el uso de métodos indirectos los cuales invocan supuestos más inflexibles sobre patrones de mortalidad por edad y por sexo. En marcado contraste con otros trabajos, los datos que son el fundamento para nuestros estimadores de esperanzas de vida están ajustados por cabalidad de los censos y registro de muertes así como también por los problemas producidos por mala declaración de edad en los censos y estadísticas vitales. Esto permite evaluar con mayor confianza las tendencias de mortalidad adulta, sobre todo en edades avanzadas.

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I. INTRODUCTION

This paper reviews mortality trends during the 20th century in selected countries of Latin America and the Caribbean (LAC). The paper focuses only on female and male mortality patterns over age 5 during the period 1900-2000. For some countries we reconstruct the trajectory from very high mortality prevailing at the beginning of the century to high levels of life expectancy attained by the year 2000. For others we are able to estimates trajectories starting in 1950. Somewhat surprisingly, current levels of life expectancies in some of these countries are lower than those experienced in high income countries.

The paper is organized as follows: in section II we briefly summarize the nature of the relevant information for LAC countries. In Section III we identify problems and gaps in extant estimates of life expectancies and trends. In this section we summarize the procedures we apply to estimate adjusted life tables for intercensal periods. In all cases we only use a combination of population censuses and vital statistics and ignore adult mortality estimates from indirect methods. The adjustments we implement are those designed to correct for completeness of census and death registration as well as for age misstatement in both sources. In Section IV we compare our estimates with alternative ones obtained using different methodologies, including those based on orpanhood, widowhood and sibling survival as well as with those generated by a number of agencies using a mixture of adjustment procedures whose origin and nature are not always transparent. Our decision to forego estimation using well-established indirect methods, such as those based on orphanhood and sibling survival, stems from a desire to avoid assumptions about underlying age patterns of mortality which, *per force*, must be invoked to generate a complete life table above age 5 if any of these methods are applied. We invoke a similar rationale for leaving out existing estimates of mortality for the period 1900 to 1950 for those countries with no existing vital statistics prior to 1950.

In Section V we summarize observed trends and attempt to indirectly identify the determinants responsible for the post-World War II gains in adult survival. In Section VI we estimate the contribution of major groups of causes of death to those survival gains; whereas in Section VII we estimate relations between life expectancy and macro determinants. We also assess the magnitude of gains accrued in the period 1950-2000 that

are associated with shifts independent of improvements in measured covariates. Section VIII examines patterns of mortality at older ages. Current theories of longevity and the assessment of the likely trajectory of future life expectancy have been informed by and based largely on what we know about mortality patterns in developed countries, those whose mortality transition began well before 1900. The contribution to this discourse from the experience of countries, whose mortality decline begins late, right before or immediately after World War II, is scarce. This is an important lacuna since the nature of mortality decline in these countries in general, and in LAC in particular, is sharply different from the mortality decline in more developed societies. The peculiarities of the mortality transition in LAC, including its highly compressed character and the evolution of a regime where modern chronic conditions coexist with infectious diseases, may have important implications for the potential trajectory of life expectancy. Section IX concludes.

II. A BRIEF HISTORY OF MORTALITY STATISTICS IN LAC

Theories of mortality decline have been formulated, tested and then reformulated mostly by exploring the characteristics of mortality changes in what are now high income countries.[4] The most fundamental of these contributions by McKeown (1976) is entirely based on the experience of England and Wales. So, it is the expansion of McKeown's conjecture formulated by Fogel (2004). Similarly, mortality trends in France, Italy and Spain (Vallin 2002; Caselli 2002; Schofield and Reher 2002), for example, have helped to refine the foundations of what could be considered a well-tested theory about the mechanisms that produce the transition from high to low mortality regimes. Nothing of the sort exists for low income countries in general and Latin America in particular. Perhaps the first incursion into this unknown territory can be traced back to the work by Arriaga (1968) and by Arriaga and Davis (1969). Arriaga not only produced the first set of historical life tables for the majority of Latin American countries but, in collaboration with Davis, formulated a theory, albeit simple, about the conditions that explained the evolution of mortality up until 1970. But the seminal work by Arriaga was never followed up, challenged, modified or updated with one exception. This was the work by

[4] These theories are distinct from classificatory frameworks such as those proposed by Omran (1982) in that they explicitly seek identification of the ultimate causes of changes in mortality and morbidity regimes.

(Stolnitz 1965) and especially by Preston who, in a series of classic papers, articulated a theory of mortality decline using statistics from a blend of low income and high income countries (Preston 1976; 1987; Preston and Nelson 1974). This research, however, is not distinctly applied to Latin America but is an accounting of mortality decline in general. In addition, and with counted exceptions, this body of work is based on unadjusted statistics for the low income countries included in the analyses.

During the period of 1970-2000 there was an explosion of new methods to estimate child and adult mortality using indirect techniques. Multiple estimates were produced for different countries largely for the period 1950-2000. But these efforts have three shortcomings. First, estimates were produced for the age group 0 to 5 or for adult mortality without generating entire life tables. Calculations of entire life tables were the result of stitching together the estimates for early childhood and adulthood, an operation that rested on assumptions about underlying age patterns of mortality. Second, the estimates were never assembled to generate a systematic account of the trajectory of mortality in the continent. Exceptionally, however, CELADE (Centro Latinoamericano de Demografia), the UN (United Nations) and PAHO (Pan American Health Organization) released adjusted estimates of mortality for almost all countries in Latin America for the period following 1950. These organizations employed a number of adjustments that rarely spelled out in detail the adjustment procedures. The exception was the work done in the late seventies by the United Nations which led to a redesign of models of mortality patterns (United Nations 1982). The data base used by the United Nations consisted of mortality statistics adjusted for completeness in the age segment 0-10 but rarely or never adjusted for adult mortality. Absent from this effort, however, was an attempt to explain the great intercountry heterogeneity. Third, with only one exception the statistics available were never analyzed under the scope of an explicit theory of mortality decline, such as the one put forward by Arriaga and Davis (1969) or Preston (1976). This exception (Palloni and Wyrick 1981) fell short and did not fill the gaping vacuum of knowledge since it focused on standardized death rates (total and by causes of deaths) and only some of these were known to be of sufficient accuracy while a cloud of uncertainty covered all others. One finding from this work that deserves attention, because it partially confirms Preston's conjectures, is that the mortality decline that took

place in LAC countries between 1950 and 1980 was associated mostly with the implementation of public health policies and the application of new medical technology and much less so with improvements in standards of living. Furthermore, and echoing the work that had been done in the case of Ceylon -- now Sri-Lanka -- (Fredericksen 1970; Gray 1974) Latin American countries whose mortality regime was heavily dominated by debilitating infectious diseases such as malaria, benefitted much more than expected from reduction of these diseases. Palloni and Wyrick proposed the idea that reduction or elimination of malaria in the period 1940-1960, following the massive application of dichloro-diphenyl-trichloroethane (DDT), led to improvements in immune function and nutritional status which, in turn, contributed to the decline of other infectious diseases even in the absence of additional interventions or improvements in the health care systems.

The last effort to produce a comprehensive review of adult mortality in Latin America is a volume edited by Timaeus and colleagues (1996). It consists of chapters devoted either to just one country, to an assessment of the quality of vital statistics, to a handful of diseases or, to an evaluation of estimates from indirect methods for a few countries with very poor vital statistics on adult mortality. The book lacks, and was not intended to produce, a consistent set of estimates for all countries covering a significantly long period of time nor does it venture into the riskier territory of theory construction and falsification.

In this paper we attempt to move the discussion forward and generate adjusted estimates of mortality above age 5. We also engage directly in a discussion of the nature of forces that led to the sharp mortality decline in the region.

III. ESTIMATION OF ADULT MORTALITY IN LAC DURING 1900-2000

A. The state of vital statistics in the region

Many countries of the Latin American and the Caribbean region have carried out censuses at regular intervals since 1900. And in most of them the establishment of a vital statistics system has made possible the collection of information on births, deaths and marriages since at least 1940 and from as early as 1900-1920 in only a handful of them. Andean and some Central American countries --suspected to have the highest levels of

mortality-- have yet to develop thorough vital statistics systems and only erratically carried out national censuses, though this has improved since 1970.

We concentrate exclusively on mortality above age 5. We do this for two reasons. The first is that this paper is mostly a review of and attempts to identify trends in adult mortality which are, as a rule, quite distinct from trends in childhood mortality. The second reason is that adjustment of estimates of mortality before age 5 must rely almost exclusively on sources other than vital statistics (that is, indirect methods), and requires special procedures to join them together with estimates of mortality above age 5 to arrive at a unique life table. Our aim was to rely exclusively on estimates derived from two sources, censuses and vital statistics, with no support from external assumptions regarding age patterns of mortality.

Armed with a series of censuses and intercensal vital statistics, it would be a routine matter to construct a series of life tables extending as far back as 1900-1920 for some countries or to 1950 for all of them were it not for the fact that the mortality rates thus calculated are affected by three types of errors: completeness of death registration completeness of census enumeration, and age misreporting.

i. Completeness of death registration

The first type of error and the one to which demographers have devoted most of their attention is due to the fact that vital statistics are incomplete. In some countries, such as the Dominican Republic for example, the completeness of death registration was not higher than 60 to 70 percent as recently as 1990. In others, such as Mexico, vital statistics improved steadily over time to the point that unadjusted estimates of life expectancy are deceiving and may give the impression of an excessively slow upward trend. Finally, in other countries, such as Argentina, vital statistics have been virtually complete for many years although their quality fluctuates from year to year and, more likely, from decade to decade.

ii. Completeness of census enumeration

The second difficulty is that completeness of censuses enumeration is highly variable and, in some cases, well below acceptable standards. Not all countries carry out post-enumeration surveys to calibrate censuses and pinpoint with some precision the amount of under(over) count. Furthermore, the naïve assumption that census coverage

inevitably improves over time, which is roughly satisfied in the case of vital events, is not always realistic as census coverage can fluctuate sharply, following the vagaries of political transformations, economic crises, and the dynamics of internal population displacements.

iii. Age misstatement

The two aforementioned problems can lead to estimates mortality rates that are either too high, too low or right on the mark. Thus, unless we have the means to estimate the completeness of death registration *relative* to census coverage we will not be able to construct accurate life tables, let alone an accurate time series of life expectancy. A number of methods developed in the late seventies by Brass (1975), Martin (1980), Hill (1987), Preston (1983), Bennet and Horiuchi (1981) and others had as a central objective the estimation of relative completeness of death enumeration. Most of these methods are flexible, do not rely on stifling assumptions about stability of the population or age patterns of mortality and are simple to apply. But although it can be shown that these procedures produce satisfactory results when all assumptions on which they rest are approximately satisfied, none of them addresses the third difficulty we face when attempting to estimate mortality in a context with deficient statistics. This difficulty is that there appears to be a large amount of age misstatement both in censuses and deaths. By this we mean not just age heaping (or concentration around preferred digits) but a systematic propensity to over(under)state the true age.

Surprisingly enough, rising awareness about the potentially harmful effects of age misstatement on mortality estimates attracted widespread attention in the U.S. with the controversy about the so called “mortality crossover”, according to which the survival curves of U.S. blacks tended to converge toward or even crossover that of whites (Eberstein et al. 2008). Some researchers attributed this to the role of unmeasured heterogeneity (Vaupel et al. 1979) whereas others argued that convergence and/or a crossover was a artifact of race differentials in age overstatement (Coale and Kisker 1990; Preston et al. 1996).The idea that age overstatement could lead to misleading results was extended by Preston and colleagues to other developed countries (Himes et al. 1980; Condran et al. 1991) as well as to a handful of countries in Latin America (Dechter and Preston 1991; Grushka and Preston 1995; Grushka 1966) and to Puerto Rico

(Rosenwaike and Preston 1984). In a nutshell, the main finding of this body of research is this: in most countries of the LAC region there is a pronounced and systematic tendency to overstate ages both in census and death statistics, but much more so in the latter than the former. This tendency is particularly strong at older ages (above age 45 or 50 and especially above age 60). The pattern is pervasive and affects all countries with available vital statistics (Dechter and Preston 1991; Grushka and Preston 1995; Grushka 1996). Furthermore, although there are empirical indications that age overstatement diminishes over time, the trend is by no means uniform because it is confounded with the contribution of errors associated with coverage in censuses and vital statistics. Our own investigation suggests that, after adjusting for completeness of death registration and errors of census coverage, the levels and patterns of age overstatement experience noticeable amelioration over time.

The most important problem posed by a pattern whereby age overstatement of deaths dominates age overstatement in the population is that mortality rates will be systematically biased downwards at older ages with the resulting overestimation of life expectancy for the older population.[5] Furthermore, the age pattern of mortality will appear to be one where the level of mortality over age 60 is lower than expected, given the level of mortality prevailing in the age span 5 to 60. As a consequence, the study of the relationship between early and late adult mortality and especially longevity and its progression over time can be severely compromised. The biases on estimates of life expectancy at age 5 are of lesser magnitude since the proportion of years lived over age 60 or so is, as a norm, not high relative to the proportion lived over age 5. But as mortality improves, the proportion of years lived over 60 as a proportion of years lived over 5 tends to increase more rapidly than the proportion of years lived between 5 and 60. As a result, the persistence of patterns of age overstatement of deaths will lead to biases

[5] Overstatement of ages in the population always leads to underestimates of the mortality rates provided that (a) the proportion overstating ages is invariant or increases with age and (b) the age distribution slopes downwards as age increases. The effect of age overstatement of ages at death is not as straightforward since it depends on three factors: (a) the age-dependency of the proportion of deaths whose age are overstated, (b) the magnitude of the downward slope of the age distribution at older ages and (c) the magnitude of the upward slope of the force of mortality at older ages. In most countries of the regions there are conditions that translate overstatement of ages at death into under (not over) estimation of mortality rates at older ages. If so, age overstatement of population and deaths will have offsetting effects and may actually lead to overstatement (not under) of mortality rates.

in estimates of life expectancy at age 5 that, *ceteris paribus*, will increase in magnitude as mortality improves. This, in turn, will lead to the misleading impression that the pace of gains is more rapid than it really is.

B. Methods for the adjustment of observed death rates

In a companion paper (Palloni and Pinto 2004) we describe in detail the strategy to adjust observed intercensal death rates. What follows is a sketchy summary of the procedure.

We first estimate relative completeness of any two consecutive censuses using a procedures suggested by Brass (1975). As first intuited by Hill (2002) this estimate is remarkably robust to departures from stability and age misstatement. We then use this estimate to obtain corrected age specific intercensal rates of population of growth. These are then used as inputs for the method first proposed by Bennet and Horiuchi (1981) to estimate relative completeness of death registration under conditions of non-stability.[6] The final step consists of using estimates obtained from simulations of age misstatement to retrieve the level of net overstatement of both population and deaths once these are adjusted for completeness. The level of net overstatement is relative to a standard pattern of age over(under)statement, referred to as the "Costa Rican standard" for it was derived from the only empirical research that establishes a relation between 'true' age and age declared in a census for a representative sample of a national population (Rosero and Brenes 2003). We assume that this observed pattern underlies both the age pattern of age misstatement of population censuses and the age pattern of misstatement of age at deaths in all countries of the region (Palloni and Pinto 2004).[7] Table 1 shows a list of countries and census years as well as intercensal deaths that were used in the estimation exercise.

Table 1 about here

Table 2 displays the adjusted values of life expectancy at ages 5 and 60 for males and females for all intercensal periods in the countries included in the data base.

Table 2 about here

[6] In a companion paper (Palloni and Pinto, 2004) we justify the choice of Bennet-Horiuchi methods as the one producing minimum errors over a variety of simulated conditions

[7] Note that we are not requiring that the level of age misstatement be the same as the Costa Rican but only the age pattern.

IV. Evaluation of estimates

To evaluate the quality of our estimates we follow two strategies. The first contrasts our estimates of life expectancies at age 5 and 60 with alternative ones. The second examines the implied age patterns of the adjusted adult mortality estimates and searches for anomalies to reveal residual errors.

A. Global assessment: alternative estimates[8]

For the period 1950-2000, there are alternative estimates of life expectancy at age 5 and 60. For the period prior to 1950, there is a handful of estimates available to us. In the post-1950 period the estimates of life expectancy at age 5 and 60 that we use in this paper are always slightly higher (by not more than 5 percent) that those from CELADE. We suspect that this behavior is due to the fact that CELADE estimates may be constructed adjusting separately, rather than jointly as we do here, for completeness of death registration and censuses. To test this, we performed a few experiments which suggest that CELADE's estimates may take into account only completeness of death registration, sometimes ignoring entirely census completeness (Jaspers and Orellana 1996). By and large, though, both sets of estimates are quite consistent with regard to trends.

For the pre-1960 period we were able to contrast our estimates with those calculated by Arriaga (1968). Of a total of 62 possible comparisons 16 are for the period before 1950 and involve Brazil, Chile, Colombia, Costa Rica and Mexico. In all these cases Arriaga's estimates are lower than ours by 0.5 to 4 years. Of the remaining contrasts, 24 pertain to the period 1950-1959 and in the bulk of these cases Arriaga's estimates are either very close or slightly lower (not more than 5 percent) than ours. For the period 1960-1969 (22 contrasts), Arriaga's estimates are slightly higher than ours. This result for the most recent period is consistent with expectations: the backbone of Arriaga's method, the assumption of stability, is weaker after the year 1950 for some countries and after 1960 for all of them. A quasi-stable population induced by mortality decline will lead the observer who assumes stability to underestimate the force of

[8] To save space we only provide a brief evaluation based on tables and figures that are available on request from the authors.

mortality. Since our adjusted life expectancies do not depend on stability, they are not affected by mortality decline and we should expect them to be, *ceteris paribus*, lower than those derived from a procedure that assumes stability. It is more difficult to explain why Arriaga's estimates are lower than ours for the period when these populations were approximately stable, that is before 1950. One possible explanation is that since our estimates depend on mortality statistics whereas those of Arriaga's depend only on population enumerations, the error associated with age exaggeration of deaths imparts a larger upward bias to our estimates of life expectancy than to those from Arriaga's. But if this is so for Brazil 1945-1950, Chile, 1920-1945, Colombia 1940-1950, Costa Rica 1930-1945, why is not also the case for Mexico1930-1950? Or, for that matter, for any of the other countries with more deficient vital registration system.

An important factor to consider is that whereas we adjust intercensal rates of increase for relative under(over) enumeration in population censuses, Arriaga does not. It is well known that errors in the observed rate of natural increase lead to sizeable biases in stable-based estimates (Coale and Demeny 1967), though the direction of these biases will depend on the nature of the under(over) count and should not always lead to overstated estimates of mortality.

Despite these caveats, we should not make too much of the observed discrepancies for two reasons. First, only in a few cases do they exceed 5 percent. The worst discrepancies are for Paraguay, a wretched case for which the stable population assumption is completely unrealistic since the age distribution is severely distorted by wars that devastated the male population. Second, for periods when we cannot produce estimates due to lack of vital statistics our estimates line up remarkably well with those produced by Arriaga for the earlier period. The consistency is so tight that simple linear backward extrapolations of our estimates yield values that are indistinguishable from those obtained by Arriaga, *precisely for the period when the assumption of stability was more reasonable.* In summary, the contrasts with Arriaga's estimates suggest that there are some irregularities in a few of our estimates perhaps attributable to age misstatement but also that there is an overall consistency in the estimation of trends.

<u>B. Local assessments: age patterns</u>

An important innovation of our estimates is that they include adjustments for age overstatement, a flaw known to be pervasive in Latin American's vital statistics and censuses. To check the outcome of these adjustments we calculate "expected values" for life expectancy at age 60 implied by our estimates of life expectancy at age 5 using all four Coale-Demeny models mortality patterns. We know that age overstatement is more likely to occur at ages over 45 or 50 (Dechter and Preston 1991) and that this should impart an upward bias to life expectancies above those ages, but particularly at ages over 60. If age overstatement swamps our estimates the expected life expectancy at age 60 from Coale-Demeny life tables with equivalent life expectancy at age 5 should be always lower than our estimates *regardless* of model pattern. This is not the case. Indeed, as shown in Figures 1a-1d, the values implied by the North model lead to a quasi normal dispersion of deviations centered at 0, as it should be if there is no systematic deviation from the pattern. Instead, the deviations associated with the remaining models are almost always centered at values well over 0. Why should the North model lead to a 'better' fit of adult patterns of mortality? One explanation is related to the nature of the morbidity and mortality regimes that underlie the Coale-Demeny North model life table. This model is based on mortality in Northern European countries, most of which experienced high endemicity of respiratory tuberculosis (TB) before the beginning of the 20th century (Coale and Demeny 1966). Thus, life expectancy at age 5 should be lower relative to life expectancy at older ages in the North model when compared to other model mortality patterns due to the presence of selection, because high prevalence of active TB inflates mortality levels for younger adults relative to that of older adults. Is this consistent with the experience of the LAC region? Although TB was endemic in some of these countries and levels of mortality due to TB around 1950-1960 remained quite high, we find only a weak relationship between the magnitude of deviations associated with all four sets of deviations and lagged mortality rates due to TB in all age groups.[9]

[9] We used mortality rates due to TB for age groups 0-19, 20-39, 40-59 and 60+ with lags of 5 and 10 years. Rates were averaged over five and then over ten years prior to the time for which we had the measure of model pattern deviation. We used pooled estimators of effects and in no case did we obtain a positive and significant effect of mortality rates due to TB and magnitude of the deviations. On the assumption that the observed deviation could be due to age overstatement and mortality rates due to TB we also estimated a fixed effect model (that assumes that effects of age over statement are invariant over time). But the expected patterns did not materialize.

A second feature of interest is that the absolute magnitude of deviations between observed and expected life expectancy at age 60 increases gradually over time. This is inconsistent with the explanation invoking prevalence of respiratory TB as this would lead one to expect that deviations should *decrease over time*, apace with the dissolution of a mortality regime with high endemicity of respiratory TB. Overall, we conclude that one cannot impute the observed agreement with the North model and the pattern of deviations from the other models to the influence of respiratory TB.

Figures 1a-1d about here

It is indeed possible that our adjusted mortality rates may still be contaminated by age overstatement that *mimic the effects that past levels of respiratory TB would impart on the age pattern of mortality*. That is, the similarity between LAC adult mortality patterns and those in the North model could be an artifact of age overstatement. But this interpretation is also unsupported by the data. Evidence for severe age overstatement decreases sharply and steadily over time, as shown by the systematic convergence to unity of the age specific ratio of expected to observed deaths. As an illustration, Table 3 displays these ratios for two countries. Ratios for other country-intercensal periods show similar patterns. A decrease in the value of the indicator in Table 3 signals certain decrease in the severity of age overstatement. But this improvement overtime is incompatible with the foregoing interpretation that attributes overtime increases of deviations from model patterns to age overstatement. We cannot have it both ways: either the deviations from model patterns increase overtime due to increased severity of age overstatement, in which case the indicator in Table 3 should have a behavior opposite to the observed one, or the indicator in Table 3 reflects a true decrease in age overstatement in which case the increased deviations from model patterns must be unrelated to age overstatement and instead may reveal a peculiarity of old age mortality in the region.

Table 3 about here

An alternative test is to contrast the age pattern of adjusted old age mortality in countries of the region with those obtained for developed countries. To do this we estimate models of the following form:

$$\ln (Mx /(1-Mx)) = \alpha + \beta \varphi(x)$$

where $\varphi(x)$ is the logit transform of a standard set of mortality rates estimated by Himes and colleagues (Himes et al. 1994). Systematic overstatement of ages at death must lead to two outcomes. The first is a downward bias (away from unity) in the estimate of β. The second is an exaggeration of the absolute value of α.[10] Estimates of β systematically below 1 and relatively high negative values of α, are consistent with the conjecture about age overstatement. Table 4 displays the estimates of both parameters for the populations included in our analyses. A glance at the table reveals an important feature: estimates of β are systematically below one but much less so in the past than in recent years. This is consistent with the conjecture of gradual improvements in declaration of ages at death. The behavior of the parameter α is, as it should be, a bit more erratic as it reflects both the push of changes in levels of mortality and the impact of age overstatement, both of which will lead to increments in its absolute magnitude. With some exceptions, the estimated (absolute) value of α increase regularly over time. An important feature is that countries reputed to have the best quality vital statistics (Argentina, Chile, Costa Rica, Cuba, Panama and Uruguay) all yield estimates of β that are close to 1, particularly in the most recent periods.

While this test shows that the behavior of our estimates of adult life expectancies is consistent with an interpretation invoking distortions due to overstatement of ages at death, it does not prove the case, much in the same way as the test based on the Coale-Demeny mortality patterns could not: true peculiarities of old age mortality in the region could produce exactly the observed pattern.

In summary, evaluation of the quality of age adjustments of mortality rates is not unequivocal but instead sends painfully mixed signals. First, our estimates of life expectancy at age 5 line up fairly well with alternative estimates and there are no strong signals that the adjustments leave in systematic biases. Second, the estimated age patterns of mortality reveal evidence of peculiarities and one of the alternative explanations, but not the only one, is age at death overstatement. The evidence from model patterns is

[10] If α is held constant, subestimation of Mx produced by age overstatement should lead to *overestimates* of β. However, when both parameters are free to vary, the best linear fit is always achieved at the expense of a more negative value of α and an estimate of β that is smaller than one.

contradictory. On the one hand, findings from comparisons with Coale-Demeny mortality models are inconsistent with a view that singles out age overstatement as an important factor. On the other hand, evidence from comparisons with a modern standard of mortality at old ages behaves as we would expect if the adjusted estimates continue to contain effects of systematic age overstatement.

Table 4 about here

V. MORTALITY TRENDS

Gains in life expectancy at age 5 during a period of time, say (t, t+n), are a function of the initial level of mortality associated with causes of death that contribute the most to mortality at time t and that, simultaneously, are potentially more vulnerable to the effects of new infrastructure (water and sewage), diffusion of knowledge, adoption of new behaviors and public health interventions, improvements in standards of living and nutrition, and medical technology. Infrastructure refers to large undertakings designed to supply clean water and safe disposal of sewage. They require large investments and almost always depend on interventions by a central state. Diffusion of knowledge refers to new ideas that influence beneficial behavior, such as personal hygiene, that minimize exposure to disease. As plentiful empirical evidence in Western Europe (McKeown, 1976) and North America (Preston and Haines, 1991) shows, the role of these factors is influential particularly at the beginning of the secular mortality change. Adoption of deleterious behaviors, such as diet, sedentary life styles and smoking, become important brakes of mortality decline but only after the initial phase of the transition toward lower mortality. Due to lack of information we can only speculate on their influence on illnesses such as diabetes, heart disease and neoplasms. Improvements of standards of living and nutrition refer to average levels of per family income and supply of sources of calories (as opposed to nutritional status)[11] respectively. Finally, medical technology refers to resources generated by improved medical knowledge that are deployed to reduce exposure to disease (vector eradication campaigns), increase resistance (antibiotics and vaccines) and improve recovery (new medications and treatments).

[11] See Preston (1976) and Fogel and Costa (1997).

Increases in life expectancy after 1950 were in all likelihood fueled by the widespread diffusion of medical technology, even though other determinants continued to play a role in most countries. The bulk of mortality improvements over age 5 associated with the diffusion of medical technology throughout the region must have taken place after 1950 since prior to that period, direct medical interventions on a massive scale were rare in most countries. The widespread diffusion of antibiotics, sulfas, and vaccines that made a significant dent on the prevalence of infectious diseases was not feasible before 1950 for two reasons. First, because generalized application of these innovations in more developed countries took well over thirty years and was not firmly established until the period around World War II. Second, because their implementation requires institutional strength and complexity which, with some exceptions, was not in place in LAC until much later, during the post-World War II period.

Increases in life expectancy that took place prior to 1950 are more reasonably attributed to one of two determinants. The first is deployment of public health measures, including large-scale infrastructure (water purification systems, piped water, sewage processing and disposal), foreign-funded campaigns to eradicate vector borne diseases (mainly malaria, dengue, yellow fever), and sanitation techniques to prevent exposure in households and among individuals. The second determinant is increases in standards of living and improvements in nutrition.

The two determinants identified above reinforce each other and produce synergisms that prevent a clear-cut attribution of effects to each one of them separately. For example, eradication of malaria through DDT spraying reduces exposure and has spill-over effects by reinforcing individual immune function, thus promoting increased resistance to other infectious diseases. Similarly, reduction of incidence of diarrhea and other wasting intestinal conditions, a result of water purification, sewage treatment and shifts in individual behavior, boost nutritional status, even in the absence of direct increases in nutrient intake. In turn, improved nutritional status strengthens individuals' resistance and recovery.

In what follows we summarize the trajectory of gains in life expectancy at age 5 during the period 1950-2000 and, whenever possible, attempt to identify the determinants that played the most important role. To facilitate description we divide our observations

as follows: we classify as "forerunners" (Argentina, Chile, Costa Rica, Cuba, Panama and Uruguay) countries that by 1950 had already attained levels of life expectancy at age 5 of 58.7 years among females and 56.6 years among males. These values correspond to life expectancies at age 5 attained by Norway around 1900, that is, after mortality had been declining for fifty or more years in *the absence of advances of medical technology.* Thus, the level attained around 1900 in Norway could only be associated with public health or better standards of living or a combination of the two. The remaining countries are classified as 'laggards'.

Figure 2 displays the values of life expectancies at age 5 (E5) for females by group of countries. Two lines are drawn at values of E5 for Norway during 1900 and 2005.[12] The estimated difference in E5 between the two groups of countries around 1950 was close to four years but by the year 2000 it was reduced to no more than 2 years. The reduction in the difference between the two groups is explained by the fact that after 1950 the laggard group experiences the beneficial effects of medical technology, public health and better standards of living simultaneously whereas the forerunners had already succeeded in controlling an important fraction of infectious diseases before 1950. Thus, the diffusion effects of medical technology among the forerunners should have been more muted and contributed somewhat less to gains in life expectancy.[13]

Figure 2 about here

Gains in life expectancy at age 5 during a period of time, say (t, t+n), are a function of the initial level of mortality associated with causes of death that contribute the most to mortality at time t and that, simultaneously, are potentially more vulnerable to the effects of new infrastructure (water and sewage), diffusion of knowledge and public health interventions, improvements in standards of living and nutrition, and medical technology. We use McKeown (1976) classification of diseases (water borne, vector

[12] It will not go unnoticed that the trends in both groups of countries are almost linear, with a slight but noticeable decreased slope in the last ten years among forerunners. The same applies to trends of life expectancy at age 60 (see Section VIII). These regularities combined with the fact that the rate of decline in infant and child mortality has decreased in the last ten years, imply that the trajectory of life expectancy cannot be linear but rather must follow a quadratic form. This goes against inferences made by Oppen and Vaupel (2002) from a pooled sample of life expectancies using a mixture of countries with wildly heterogeneous mortality regimes.

[13] The graph for males is omitted since it leads to the same conclusions.

borne and airborne) to identify the relation between their initial levels and subsequent gains in life expectancy at age 5. We predict the relative rate of change in life expectancy at age 5 during a period, say (t, t+k) as a function of lagged values of the log of the mortality rates for each of the groups of causes mentioned above. To attenuate the influence of fluctuations we construct the average rate over the first five years prior to the beginning of the period (between t-4 and t) and then the average value over the second past five years (between t-5 and t-9). We will refer to these as the first and second lag respectively. We should expect that relative changes in life expectancy at age 5 during the period (t, t+k) should be responsive to changes in the determinants identified above. Since we do not have information on these changes, we use as a proxy the levels of mortality caused by illnesses that could be improved (more or less) by the unmeasured interventions. Thus, for example, in a country with high prevalence of malaria an intervention designed to eradicate the vector will result in a sizeable change in life expectancy. If the intervention is inefficient or inexistent, it should have no effect on changes in life expectancy, regardless of how high the mortality level due to malaria may be. If instead mortality rates due to malaria are low, the interventions should have no effects regardless of how effective it might be. The expected relations are as follows:

Disease Group	Eradication	Intervention Infrastructure (other public health)	Medical	Behavioral
Vector Borne	+	-	-	-
Water Borne	-	+	+	+
Airborne	-	-	+	+

where '+' signifies that the intervention has strong impact, whereas '-' signifies that the intervention has weak impact. As an illustration, assume that vector borne diseases weigh heavily on life expectancy at age 5 at time t and that effective interventions are in place also at time t. We would then expect that the estimated effects of mortality rates due to vector borne diseases (with pertinent lags) on the relative change in life expectancy at age 5 during the period (t, t+k) will be high and significant. Instead if a group of causes exerts

little influence on life expectancy at age 5 and/or there are no efficient interventions in place, the relations will be weak.

Table 5 displays estimated regression coefficients using the first lag for the mortality rates and the absolute change in life expectancy between two successive intercensal periods.[14] The results are presented separately for each of the two groups of countries (laggards and forerunners). Vector borne and water borne diseases play the most important role for countries in the laggard group. Indeed, the effects of vector borne mortality rates on the absolute change in life expectancies over two intercensal periods are substantial and statistically significant. Thus, for example, a one percent change in the magnitude of the initial level of mortality due to vector borne diseases brings about a gain in life expectancy of about 0.023 per year. A similar change in water borne diseases entails gains that are twice as large, 0.044 per year. Among forerunners, the heavy lifting is done by airborne diseases as neither vector borne nor water borne diseases play an important role. A one percent increase in the initial levels of mortality due to airborne diseases potentiates life expectancy gains of the order of 0.044 per year.

These results are in agreement with expectations. First, with one or two exceptions (Cuba and Panama) vector borne diseases were more prevalent among laggards and played no significant role in sculpting the mortality patterns among forerunners. Second, although eradication campaigns in Cuba and Panama began early in the century, most other countries experienced the benefits of vector eradication later in the century, mainly after World War II. Third, water borne diseases were highly prevalent throughout LAC but the large infrastructure required to contain them was in place before 1950 only among forerunners, not among laggards. This suggests that after 1950 they should have played a major role among the latter not the former. The data bear this out. Finally, airborne diseases (streptococcus pneumonia, respiratory TB, measles) are highly responsive to vaccination campaigns which were probably more efficiently implemented among forerunners and increasingly so during the post World War II period. This is reflected in the stronger influence of airborne diseases on the absolute changes in life expectancies.

[14] Estimates in the table are from pooled sample. Fixed effects models yield somewhat different values for the estimates but lead to the same conclusions and are not presented here. The addition of lag 2 (ten years) does not improve model fit and was ignored.

Table 5 about here

VI. THE CONTRIBUTION OF CAUSES OF DEATH

In this section we summarize the contribution of groups of causes of deaths to gains in life expectancy between 1950 and 2000.

A. Trends in mortality by causes of death

Trends by causes of deaths in Latin American countries reflect advances in medical technology, improvement in health care systems, and changes in lifestyles and living conditions of their populations. As suggested by the 'epidemiological transition' framework (Omran, 1982) we find a sudden shift in the profile of deaths by causes and age groups from one dominated by communicable diseases to one swamped by chronic and degenerative diseases. However, in contrast to Omran's framework we will also observe that in some countries chronic disease coexist with still prevalent infectious diseases.

Figures 3a through 3f display time trends of leading groups of causes of death, namely, Neoplasms, Circulatory Diseases, Diabetes, Infectious Diseases, Accidents and Violence, and Ill-Defined causes in 18 Latin American countries. On average, these groups of causes account for nearly 70 percent of all deaths in the period under examination, from 1950 to 2005.

Figure 3a reveals mostly upward trends for neoplasms. There are some irregularities in the middle of the period but, by and large, the rates move up particularly during the last ten years of the period examined. This increase in mortality due to neoplasms is universal: it applies to both genders and to forerunners as much as laggards though there is important heterogeneity in the rates of acceleration.

Figure 3b shows evidence of large heterogeneity in trends of circulatory diseases and there are only faintly discernible patterns. Among forerunners mortality rates due to circulatory diseases tend to drop or to stay steady after the middle of the period (circa 1960-1970) whereas the majority of laggards experience increases from the beginning or from the middle of the period under examination. The drop among forerunners mirrors the experience of more developed countries where death rates due to circulatory diseases

have come down substantially. The experience of laggards appears to follow the trajectory of countries that started the epidemiological transition more recently.

Figure 3c is perhaps the most telling. It shows an almost universal and sometimes sharp increase in mortality rates due to diabetes. Cuba, Costa Rica and Uruguay are the only countries where the impact of diabetes remains steady, behaves somewhat erratically around fixed levels, or declines slightly. The sharp increase elsewhere is a hallmark of these countries where the obesity epidemic has progressed swiftly, independently of the past history of mortality decline. An important similarity is that death rates due to diabetes do not show the stark gender contrast observed in mortality due to cancer of circulatory diseases.

Figure 3d shows an unsurprising and universal precipitous decline in infectious diseases. In some cases there are small short-run increases, perhaps reflecting the impact of economic crises (Palloni and Noronha 2010). This is particularly the case in Argentina and Brazil, two of the countries that experienced massive economic contractions and sharp increases of poverty after the middle of the nineties.

As illustrated in Figure 3e, and as happens in most high income countries, there is a marked excess of male death rates due to Accidents, Suicides and Violence. There are no clearly identifiable time trends, only spikes of significance in countries that experience war and protracted political upheavals (El Salvador, Guatemala, and Colombia).

Finally, mortality rates due to ill-defined causes (including those classified as senility) displayed in Figure 3f drop significantly in all countries as a result of increases in the proportion of properly certified deaths. The magnitude of the rates and the slope of the downward trends are much flatter in countries that have a long tradition of virtually complete death coverage, most of which pertain to the group of forerunners.[15]

Figures 3a through 3f about here

B. Contributions of causes of deaths to increases in life expectancies

[15] If the category 'ill-defined' had a distribution of cause of deaths proportional to the observed one, none of the inferences drawn before would change. There is no evidence to suggest that the observed distribution is unlike that of 'ill-defined causes' and even less reason to assume that deaths categorized as ill-defined are attributable to causes that are difficult to diagnose. It is more likely that the ill defined causes are composed disproportionately of deaths associated with mortality among the poorest segments of the population, namely, infectious diseases.

Figures 4a and 4b display the contribution of number of years gained (lost) of life expectancy at age 5 during the period 1950-2000 associated with each group of causes. Figure 4a is for forerunners whereas Figure 4b corresponds to laggards. These calculations were carried out using a decomposition method suggested by Pollard (1988) and Arriaga (1984).

With the exception of diabetes, which leads to losses of life expectancies of the order of 0.5 for both males and females, changes in all other groups of causes of deaths tend to increase life expectancy at age 5 among forerunners. The single most important contributor is the group of causes associated with circulatory diseases that contributes with average gains between 1 and 2 years among males and between 1.8 and 3 among females. Lower contributions are associated with neoplasms and infectious diseases.

The contrast with countries in the laggard group could not be starker. Among these the single most important contributor to life expectancy gains are reductions in infectious diseases which led to gains of between 0.5 and 5 years of life. The only commonality between forerunners and laggards is found in the perverse role of diabetes since also among laggards this disease leads to losses in life expectancy of about 0.5 years.

Figure 4a about here

Figure 4b about here

Finally, Table 6 displays estimates of the contributions made by mortality reductions in broad age groups and by groups of causes within the two groups of countries. These figures show that among forerunners the reduction in mortality during the period is mostly attributable to adult ages (over 20) whereas among laggards the largest contributions are associated with ages between 5 and 20 and with infectious diseases. The figures for diabetes reveal that despite the generalized upward trend, it is mostly among individuals in the oldest age group that the disease has a significant impact

Table 6 about here

VII. THE DETERMINATS OF MORTALITY TRENDS: 1950-2000

What were the forces behind the rapid change in mortality during the period 1950-2000? In a previous section we conjectured that forerunners, well into the transition at the beginning of the period under study, experience early improvements that were mostly rooted in increases of standards of living and nutritional status as well as in the creation of massive infrastructure for water and sewage that reduced exposure to water borne diseases particularly. These changes took place before 1950, perhaps beginning immediately before and after World War I, and were associated with incipient industrialization, large flows of foreign capital, and an active export sector and export based social class that secured access not just to solid reserves in foreign currency but also constituted a portal for the dissemination of new ideas about minimization of exposure. Furthermore, in two of these countries (Cuba and Panama), early eradication campaigns played an important direct and indirect role. Forerunners benefitted during the post World War II period from the diffusion of modern medical technology but the spill over effects that these might have had otherwise should be lower since standards of living had already inched upwards and exposure to waterborne diseases had been considerable reduced.

This story line does not fit well laggard countries. Mortality reductions in this group were most likely originated in vector eradication financed by foreign countries which were heavily invested in export sectors and, especially, in the importation of medical technology. The role of better standards of living and improved infrastructure is probably secondary.

To test these conjectures we follow closely the ideas put forward by Preston (1976, 1980) and by Palloni and Wyrick (1981). We first estimate the relation between life expectancy at age 5 and indicators of standards of living (GDP [Gross Domestic Product]), proxies for infrastructure (proportion of households with potable water and electricity; proportion of population living in urban areas) and, finally, the proportion literate among the adult population. The relation is estimated for two periods, the years before 1970 and those after 1970. Unlike previous research, we use three different models to assess the relation between the variables of interest.

The first is a logistic model for country i and year t of the form:

$$E5_{it} = \alpha_t / (1+\exp(\boldsymbol{\beta}\mathbf{Z}_{it})) \quad (1)$$

α_t is a free parameter reflecting the maximum value of E5, $\boldsymbol{\beta}$ is a vector of parameters including a constant and $\mathbf{Z}_{it}$ a vector of covariates for country i and year t, including a column of ones. No error term is specified.

The second specification is a double log model for the pooled sample in each period. It has the following form

$$\ln E5_{it} = \boldsymbol{\varphi}\mathbf{Z}_{it}^{*} + \varepsilon_{it} \quad (2)$$

where $\boldsymbol{\varphi}$ is a vector of elasticities of E5 relative to a vector of covariates $\mathbf{Z}_{it}^{*}$ (in log form) and ε_{it} is a normally distributed error term independent of the covariate vector

The third model is the fixed effects version of model (2) and takes the following form:

$$\ln E5_{it} = \lambda_i + \boldsymbol{\theta}\mathbf{Z}_{it}^{*} + \delta_{it} \quad (3)$$

where λ_i is a country-specific fixed effect and δ_{it} are independent, normally distributed variates (with mean 0 and variance 1). All three models are estimated separately for the pooled years 1950-1969 and 1970+. Since we only use data for intercensal intervals (rather than year-to-year observations) each country is at most represented twice in each pooled period.

A. The nature of the variables

While the analysis carried out initially by Preston (1976) placed heavy emphasis on the role of income, the later versions of his model were more fully specified and included a number of covariates among them literacy (Preston 1987). Similarly, the analysis by Palloni and Wyrick (1981) relies on a model that places more emphasis on a specification with variables that reflect different dimensions of the process. The formulation that we follow here has a simple rationale. A measure of income (real GDP) is a proxy for general standards of living though it probably leaves much to be desired on that front as much as it does regarding nutritional status. GDP is not the best measure of material wealth, general standards of living or nutritional status. But it is the only we

have at our disposal. The most important interventions to reduce exposure to infectious diseases are vector eradication campaigns and large infrastructure to supply clean water and safe disposal of sewage. We use proportion of households served by piped water and with electricity to proxy for the magnitude and reach of infrastructure and complement these with a measure of urbanization. Instead of using the mortality level associated with vector borne illnesses at the outset of the period as a proxy for the potential for vector eradication campaigns, we leave it unspecified, as part of the error term in the equations. We attempt *ex-post* to account for its role.

The variable literacy deserves special consideration. There is a fair amount of research indicating that education is the best predictor of fertility and mortality both at the individual and aggregate levels. What is less known and still the object of controversy is exactly what the nature of the relation is. At a very aggregate level, the connection between literacy and mortality is probably minimally related to individual levels of ability to obtain, process, and react to information. It is more plausible that literacy level, as in the case of the analysis of fertility, is a good proxy of institutional complexity, social integration, and the existence of flows from large institutions to individuals. This much was intuited by Caldwell (1976) in his classic treatment of the relation between massive schooling and fertility decline. Something similar could be at work with mortality. As we show later, literacy is as close to a perfect predictor for life expectancy at age 5 as we can possible aspire to.[16]

B. The nature of the models

The logistic model is defective. Though originally suggested by Preston for the very broad cross section of countries he studied, it is under-identified in our sample since the observations we have do not allow estimation of the inflection point of the logistic curve. The result is that the parameter estimates reflect only the upward bending part of the logistic curve. Although the fit of this model is very good the interpretation of parameters is not the as clear cut as could be had we been able (perhaps with more observations) to identify the point of inflection of the logistic curve.

[16] The effect of education we are posing here is broader than and distinct from the effect usually identified in the literature that is reflected in the association between individual level of education and mortality. What we have in mind is that the aggregate level of education (in this case literacy) represent not just the average individual effects but an added, and more important, influence of the strength of social institutions.

The pooled log model replicates closely the reformulated Preston model (Preston 1987) and the one suggested by Palloni and Wyrick after a search in the Box-Cox space of transforms. Both the logistic and the pooled log model are vulnerable to an important threat: the estimates will be inconsistent if the error is correlated with some of the covariates. We know for sure that we are omitting a variable reflecting eradication campaigns and another proxying for magnitudes of the flows of medical technology (in the form of vaccination or effective use of antibiotics). These factors are likely to be related to one of the variables included in the model. The solution is to estimate a fixed effects model where the effect of the country-specific unmeasured factor is removed.

C. Results

Estimation of effects is carried out in a pool of observations corresponding to the period before 1970 and in another one containing observations for the period after 1970. The rationale behind this partition is that during the earlier period wealth should have played the most important role whereas after it (unmeasured) interventions and medical innovations should play a more important role.

After estimating alternative specifications we settled on one that ignored the variables for electricity, water and urbanization since they contribute trivially to the explained variance in all three models. We settled on a specification that included GDP and literacy. The astonishing part of our results is that literacy is not only properly signed everywhere but also predicts almost perfectly the dependent variables, regardless of the role played by GDP. Furthermore, the estimated elasticity of life expectancy relative to literacy varies across models within a very small range (0.15 - 0.31) and the effects are highly significant regardless of model specification. Table 7 displays the main results. Several features of the estimates are worth noting. First, the logistic model fits extremely well (R^2=.99) in both periods. The effects of literacy are properly signed and highly significant. In contrast the direction of the effect of GDP is opposite to the expected though statistically insignificant. Second, the pooled log model also fits very well in both periods though is clearly less powerful than the logistic if one judges by the R-square. Again, the effects of literacy are powerful and bear the proper sign whereas the effects of GDP are trivial, incorrectly signed and statistically insignificant.

The fact that the estimated effects of GDP bear the wrong sign and are statistically insignificant is suspicious. Admittedly it could be explained partly by invoking measurement error and partly by arguing that mean income without a measure of inequality reveals very little. An alternative explanation is that there are unmeasured factors affecting life expectancy at age 5 that are themselves related to GDP. Figure 5 suggests potential representation of the (unobserved) relations. Now suppose that flows (and their effects) of medical technology as well as vector eradication campaigns are positively related to life expectancy ($\gamma>0$) but that the relation between GDP and the unmeasured traits is negative ($\beta<0$). The total (estimated) effect of GDP is $\alpha+ \beta*\gamma$ and $\alpha>0$. It follows that our estimated will be biased downwards and may have a negative sign. Why would β be negative? There are at least two powerful reasons to suspect that this will be so. First, import of medical technology may have occurred selectively and flows of vaccines, antibiotics and the like may have disproportionally targeted low income countries. The same applies to interventions involving vector eradication campaigns. Second, both interventions should have been more effective in low income countries since it is there were the pool of potentially troublesome communicable diseases is more densely populated.

The estimates from the fixed effects model are consistent with our interpretation. First, note that the estimated effects of literacy are again powerful and statistically significant in both periods and, for the latest period, larger than in the other two models. However, the effects of GDP are properly signed and, in the earliest but not in the latest period, strong and statistically significant. This suggests that at least during the earliest period country wealth played a role that complemented that of institutional factors proxied by literacy.

Figure 5 about here .

Table 7 about here

D. Decomposition of effects

The next step is to decompose the gains in life expectancy at age 5 into two components. The first is associated with changes in literacy and GDP across the two periods. The second is associated with changes in the relation between the variables and

must be attributed to the role played by unmeasured factors among which we include vector eradication campaigns, diffusion of medical technology and unmeasured effects of infrastructure. To do this we need to settle on a model. To increase comparability with results obtained by Palloni and Wyrick (1984) and by Preston (1988) we settled on the logistic model. None of the inferences we draw from the decomposition exercise are different if we had instead used any of the other two models.

The first application of the previous estimates is to decompose the gains in life expectancy at age 5 between the two periods for each country. Figure 6 displays the estimated proportion of the total change that is attributable to changes in the variables and changes in the parameters. Among laggards, more than 60 percent of the changes in life expectancy at age 5 are attributable to changes in the values of parameters. Instead among forerunners and with the exception of Paraguay, the bulk of gains are associated with changes in literacy. There is a clear shift in the relation which favors laggard countries. This shift should be attributed to unmeasured conditions that have either a strong (among laggards) or a weak (forerunners) influence. Two factors can account for such a shift. The first is interventions to eradicate vector borne diseases. Rapid elimination of malaria, for example, is likely to have a multiplying effect since it not only leads to gains associated with deaths due to malaria but it also affects the average resistance of the population to other infectious diseases by boosting the immune function. Secondary gains will be obtained as increased resistance to other infectious diseases improves nutritional status.

The second factor is the diffusion of medical technology, particularly antibiotics and vaccinations. These surely had important effects in all countries but their impact must have been large in those before 1950 had made only minor inroads in survival gains. If these two factors are indeed responsible for the shift we should see an association between the fraction of gains in life expectancy at age 5 and proxies for the potential gains associated with the aforementioned factors. Table 8 displays estimates of the regression coefficients of the log of the proportion of gains in life expectancy at age 5 associated with changes in parameters and variables and the log of deaths rates due to water, vector and airborne diseases as of the beginning of the period. As expected, both vector and water borne diseases are positively related to the log of the fraction of gains

attributable to shifts in the relations but only the coefficient of waterborne diseases is marginally significant. The opposite is true for airborne diseases: they are positively related to the fraction of gains attributable to changes in the variables and negatively related to the fraction of gains attributable to changes in parameters. Although these estimates analyses are based on few cases, they suggest that the conjectures we put forward at the outset are not contradicted by the observed relations.

In summary, gains in life expectancy at age 5 during the period 1950-2000 are tracked tightly by increases in literacy, not by changes in measures of wealth or indicators of infrastructure. This relation is intriguing but not unknown to demographers studying fertility as there too the association is very tight. Rather than attributing to literacy effects related to mechanisms involving access to and use of information only, it is preferably to deploy a broader interpretation suggesting that this indicator is a proxy for social integration and, most important, for the ability of central governments to allocate resources to the population at large. These flows may imply a number of things, including learning and information, access to health care and to resources such as clean water supply, adequate housing and, more generally, to environments that reduce exposure to infectious diseases. Yet, literacy cannot proxy for all of the factors that explain changes overtime. Indeed, our analysis suggests that the relation shifted in the period 1950-2000 and that countries experienced gains in life expectancy even if levels of literacy remained unchanged. All laggards benefited greatly from this shift. Among forerunners the relative gains associated with the shift are of marginal importance. We interpret this as an indication that the shift was produced by changes induced by two factors that are omitted in our models: vector eradication campaigns and the diffusion of medical innovations, particularly those that control exposure and increase resistance to waterborne diseases. Indeed, there is a tangible though not strong relation between the magnitude of gains associated with the shift of the relation and the magnitude of death rates due to illnesses that could be attenuated but these two factors.

VIII. MORTALITY AT OLD AGES

In this last section we make a brief incursion into an important territory, that of progression of longevity. For this purpose we focus on mortality over age 60 and the best indicator we have available is life expectancy at age 60. Figure 7 displays the values of

female life expectancy at age 60 for laggards and forerunners. In this case we only draw one horizontal line at the top of the graph at around 24.8, the life expectancy at age 60 among females in Norway circa 2005. At the outset the differences between the two groups of countries are of the order of two years and, like the case of life expectancy at age 5, there is some convergence though a gap still remains at the end of the period. Importantly, and unlike the case for life expectancy at age 5, life expectancy at age 60 among forerunners grows linearly and there is a hint of a slow-down of improvements by the end of the period of reference.

Figure 7 about here

The trajectory of mortality at old ages is nothing short of astonishing. Gains per year are of the order 0.13 in both groups of countries. If one assumes that the force of mortality at older ages has been decreasing at a constant and age-invariant rate during the period, the observed yearly gains in life expectancy at age 60 imply an average reduction per year of about 0.045. This is four times as high as the rate of reduction in the yearly force of mortality experienced by developed countries after 1960 (Kannisto et al. 1994) though, admittedly these are progressing from higher levels of life expectancy at age 60. But even conceding that point, the march toward longevity occurs at a very accelerated pace.

To what at extent are these gains an artifact of age overstatement?

As pointed out before, age overstatement is particularly serious at older age and, despite our best efforts, some residual errors may remain. But these should affect the level of life expectancy at age 60, not the trends. Or, put in another way, if there is residual age overstatement and if we also observe a marked tendency towards better age declaration overtime, the observed trend must underestimate the speed of change in the force of mortality. Thus, it is very likely that the rate of gains in survival over age 60 that we estimate here is a lower bound.

An entirely different matter is whether the rhythm of gains observed in the past can be sustained for too long. We argue elsewhere that there are ominous signs pointing to the possibility of rapid deceleration of gains as a result of the nature of mortality decline and the consequent composition by frailty of cohorts who will become 60 and

over during the next thirty years (Palloni and Pinto 2004; Palloni and Noronha 2010). But this is not all. In fact, Figure 7 does show signs of a slow down among forerunners at least. Indeed, among forerunners the rates of increase per year in life expectancy at age 60 has dropped from 0.14 in the period before 1980 to 0.09 after that period. Laggards, on the other hand, experienced lower rates of gains in before 1980 (0.12) but increased it after 1980. Thus, among countries that have attained higher levels of life expectancy at age 60 there are clear signs of deceleration.

Table 8 about here

IX. CONCLUSIONS

Although the inferences contained in this paper are not inconsistent with a body of research done in the past, they do introduce more precision and they allow a more nuanced interpretation of the mechanisms that were at play in the rapid decrease of mortality in the LAC region. First, we work with a body of data that avoids strong assumptions about model mortality patterns. Mortality rates have been adjusted for completeness of death registration, census enumeration and age misstatement. If any errors remain they are likely to be of trifle magnitude and cannot constitute a stumbling block against which the inferences could run into.

Second, the evidence we marshal here indicates that adult mortality (over age 5) was reduced at unprecedented speed beginning before 1940 in selected countries and after 1950 for most of them. The levels attained as of the end of the Twentieth century are close to those in developed countries for a handful of countries. For others a gap remains. This gap, however, is closing rapidly. Of particular importance is the rapid decrease in mortality over age 60 and the gains in longevity that they imply. The fact that Costa Rica and Cuba experience levels of life expectancy at age 60 similar to or even above those in some developed countries may be startling but cannot be attributed to faulty data. The progress during the period 1950-2000 has been fast but appears to be slowing down and may run into important obstacles in the years to come.

Third, for the most part mortality trends by causes have progressed according to expectations though there are singularities that stand out. Thus, infectious diseases have

plummeted and circulatory diseases have ceased to be as dominant as they were early in the period. The most important peculiarity is the rapid increase in diabetes.

Fourth, the analysis of determinants confirms the broad outlines but not the detail of Preston's original conjectures. They also replicate the findings previously uncovered by Palloni and Wyrick in LAC made with a much less refined data base. Wealth, as measured by GDP, plays an important role early on the process but a much more modest one in the latter part of the period under study. Instead, literacy is always a potent driver of changes, perhaps reflecting the effects of institutional changes unrelated to country wealth that are required to alter the exposure and resistance to diseases.

Fifth, decomposition of gains over the period 1950-2000 shows that countries who are forerunners in the mortality decline took advantage of structural changes (as reflected in GDP and literacy) as these explain the bulk of gains in life expectancy at age 5. Instead, countries whose mortality decline starts late in the period showcase the important role of selected interventions and of the diffusion of medical technology, neither of which we are able to measure directly. More than sixty percent of these countries' gains are associated with these two unmeasured factors and the rest with improvements in institutional contexts and standards of living. This estimate is much smaller among forerunners. Our analysis thus illustrates the pay offs of examining shifts in the relation between mortality and its determinants separately by groups of countries with very heterogeneous experiences.

What remains undone is a more thorough analysis of gender differentials which, at least under some conditions, may provide clues about underlying determinants. Similarly, the extension of the analysis to the period 1900-1950 for a larger number of countries is sorely needed to complete the description of mortality trajectories. This will also helps us to identify and assess the role of standards of living, nutritional status, and of early interventions embedded in the creation of large infrastructure as these must have undoubtedly contributed to diminish exposure to infectious diseases well before medical technologies and massive eradication campaigns sealed once and for all the transition to very low mortality levels.

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Table 1. Countries and data sets used in the estimation exercise

Countries	Census	Deaths	Countries	Census	Deaths
Argentina	1947	1947-1960	Guatemala	1950	1950-1964
	1960	1960-1970		1964	1964-1973
	1970	1970-1980		1973	1973-1981
	1980	1980-1991		1981	1981-1994
	1991	1991-2001		1994	1994-2002
	2001			2002	
Brazil	1980	1980-1991	Honduras	1950	1950-1961
	1991	1991-2000		1961	1961-1974
	2000			1974	1974-1988
				1988	
Chile	1920	1920-1930	Mexico	1921	1921-1930
	1930	1930-1940		1930	1930-1940
	1940	1940-1952		1940	1940-1950
	1952	1952-1960		1950	1950-1960
	1960	1960-1970		1960	1960-1970
	1970	1970-1982		1970	1970-1980
	1982	1982-1992		1980	1980-1990
	1992	1992-2002		1990	1990-2000
	2002			2000	
Colombia	1938	1938-1951	Nicaragua	1950	1950-1963
	1951	1951-1964		1963	1963-1971
	1964	1964-1973		1971	1971-1995
	1973	1973-1985		1995	
	1985	1985-1993			
	1993	1993-2001			
	2001				
Costa Rica	1927	1927-1950	Panama	1950	1950-1960
	1950	1950-1963		1960	1960-1970
	1963	1963-1973		1970	1970-1980
	1973	1973-1984		1980	1980-1990
	1984	1984-2000		1990	1990-2000
	2000			2000	
Cuba	1953	1953-1970	Paraguay	1950	1950-1962
	1970	1970-1981		1962	1962-1972
	1981	1981-2002		1972	1972-1982
	2002			1982	1982-1992
				1992	1992-2002
				2002	
Dominican	1950	1950-1960	Peru	1961	1961-1972

Republic	1960	1960-1970		1972	1972-1981
	1970	1970-1981		1981	1981-1993
	1981	1981-1993		1993	1993-2005
	1993	1993-2002		2005	
	2002				
Ecuador	1950	1950-1962	Uruguay	1963	1963-1975
	1962	1962-1974		1975	1975-1985
	1974	1974-1982		1985	1985-1996
	1982	1982-1990		1996	1996-2004
	1990	1990-2001		2004	
	2001				
El Salvador	1950	1950-1961	Venezuela	1950	1950-1961
	1961	1961-1971		1961	1961-1971
	1971	1971-1992		1971	1971-1981
	1992			1981	1981-1990
				1990	1990-2001
				2001	

Note: In all cases we used reported five year distributions, which were converted into single year distributions by applying Sprague multipliers
Source: United Nations Demographic Yearbooks

Table 2. Life Expectancies at age 5 and 60, Latin America countries 1925- 2000

Countries	Years	Males		Females	
		E5	E60	E5	E60
			Forerunners		
Argentina	1953	60.9	14.4	65.8	17.7
	1965	62.4	15.0	69.1	19.3
	1975	63.7	15.8	70.2	19.9
	1985	64.8	16.2	71.7	20.7
	1996	66.3	17.2	73.0	21.6
Chile	1925	46.8	12.5	48.0	13.2
	1935	49.5	12.5	51.0	13.9
	1946	52.1	13.1	55.4	15.0
	1956	58.7	14.8	62.7	16.8
	1965	59.8	15.4	65.1	17.8
	1976	61.8	15.7	68.1	19.1
	1987	65.9	17.5	72.1	20.8
	1997	68.8	19.1	74.4	22.4
Costa Rica	1938	52.8	12.8	54.8	13.9
	1956	64.1	17.0	66.5	18.4
	1968	66.6	18.2	70.0	19.9
	1978	68.5	19.3	72.9	21.5
	1992	70.9	20.8	75.5	23.4
Cuba	1961	63.8	16.4	66.1	17.9
	1975	67.9	18.3	71.2	20.7
	1991	68.8	19.4	72.1	21.3
Panama	1955	60.6	15.7	61.8	17.3
	1965	64.1	16.9	65.8	18.4
	1975	66.1	18.1	68.9	19.8
	1985	67.9	19.1	72.3	21.5
	1995	69.0	20.2	74.0	22.9
			Laggards		
Brazil	1985	61.5	15.8	68.1	18.5
	1995	63.0	17.0	70.2	20.3
Colombia	1944	52.9	13.9	55.8	15.1
	1957	59.2	15.8	61.5	16.7
	1968	61.0	15.2	64.7	17.5
	1979	62.9	16.7	68.2	19.0
	1989	63.8	18.4	70.2	19.9

	1999	64.7	19.3	72.1	21.2
Dominican Republic	1955	55.9	14.9	58.7	16.9
	1965	59.7	15.3	63.1	17.7
	1975	62.5	16.0	66.0	18.4
	1987	64.5	17.4	69.5	19.9
	1997	66.2	19.2	72.1	22.0
Ecuador	1956	56.1	15.8	57.2	16.5
	1968	60.9	16.7	62.7	17.7
	1978	63.7	18.2	67.3	19.7
	1986	65.5	18.7	69.4	20.2
	1995	67.2	21.0	71.4	22.1
El Salvador	1955	52.9	14.5	56.2	15.5
	1966	59.0	16.1	62.6	17.2
	1981	55.6	16.2	66.0	18.6
Guatemala	1957	51.7	14.0	52.8	14.4
	1968	55.8	15.3	58.0	15.9
	1977	57.6	16.2	61.3	17.0
	1987	59.5	16.6	64.7	18.1
	1998	62.0	18.8	68.1	19.9
Honduras	1955	51.4	15.3	54.0	16.1
	1967	56.9	15.4	58.4	16.4
	1981	61.4	17.1	65.0	17.9
Mexico	1925	43.8	12.9	46.7	12.9
	1935	45.4	12.3	47.0	12.2
	1945	50.8	13.8	53.9	14.4
	1955	57.1	15.9	60.1	16.4
	1965	60.5	16.9	63.8	17.7
	1975	62.9	17.9	67.5	19.3
	1985	64.1	18.4	70.5	20.6
	1995	67.6	20.0	72.2	21.4
Nicaragua	1956	51.5	13.8	54.2	14.2
	1967	56.2	15.0	59.4	15.6
	1983	60.4	16.9	65.8	18.1
Paraguay	1956	62.4	16.3	66.1	19.0
	1967	63.1	16.4	67.1	19.3
	1977	64.2	16.6	68.2	19.4
	1987	66.2	17.5	69.4	19.6
	1997	67.0	18.5	70.9	20.6
Peru	1966	57.7	15.2	59.9	16.9
	1976	61.9	16.6	65.4	18.8
	1987	64.6	17.9	68.1	19.8

	1999	66.0	18.4	70.5	20.7
Uruguay	1969	63.6	15.4	69.9	19.3
	1980	64.8	16.1	71.1	20.1
	1990	65.6	16.3	72.5	21.0
	2000	66.5	17.2	73.9	22.3
Venezuela	1955	58.5	14.5	60.4	15.8
	1966	61.4	15.3	65.0	17.2
	1976	63.2	16.5	68.2	18.8
	1985	65.2	17.6	71.0	20.5
	1995	66.0	18.7	71.7	21.1

Source: Own estimates using data from U.N. Demographic Yearbooks

Table 3. Ratio of Enumerated to Expected Population in Chile and Uruguay

Age groups	1930-1940	1940-1952	1960-1970	1982-1992	1992-2002	1963-1975	1975-1985	1985-1996	1996-2004
			Chile				**Uruguay**		
Males									
40 +	1.749	1.639	1.398	1.437	1.412	1.379	1.336	1.310	1.264
45 +	1.104	1.006	0.994	1.049	1.025	0.993	1.009	1.017	0.993
50 +	1.188	1.100	1.027	1.067	1.047	0.999	1.029	1.033	1.008
55 +	1.311	1.141	1.078	1.099	1.070	1.026	1.052	1.052	1.014
60 +	1.468	1.268	1.073	1.105	1.070	1.052	1.077	1.075	1.027
65 +	3.505	2.320	1.200	1.160	1.108	1.145	1.151	1.160	1.037
70 +	(--)	(--)	1.488	1.319	1.175	1.295	1.205	1.313	1.084
Females									
40 +	1.632	1.538	1.336	1.359	1.332	1.315	1.295	1.241	1.210
45 +	1.096	1.030	0.986	1.037	1.010	1.000	1.026	1.013	0.992
50 +	1.212	1.123	1.023	1.058	1.032	1.020	1.051	1.026	1.004
55 +	1.344	1.213	1.062	1.081	1.049	1.046	1.086	1.040	1.006
60 +	1.577	1.394	1.065	1.110	1.054	1.090	1.115	1.069	1.018
65 +	7.325	4.450	1.254	1.199	1.108	1.230	1.227	1.147	1.020
70 +	(--)	(--)	1.639	1.438	1.201	1.545	1.394	1.288	1.069

Note: In parenthesis are negative values
Source: Own estimates using data from UN Demographic Yearbooks

Table 4. Estimated coeffcient of the regression between the logit of old age mortality rates in LAC countries and and European mortality standard, 1925-2000

Countries	Years	Males		Females	
		α	β	α	β
		Furerunners			
Argentina	1953	-0.061	0.874	-0.132	0.860
	1965	-0.156	0.880	-0.169	0.911
	1975	-0.287	0.870	-0.219	0.921
	1985	-0.199	0.911	-0.233	0.947
	1996	-0.311	0.922	-0.442	0.924
Chile	1925	-0.240	0.712	-0.136	0.684
	1935	-0.264	0.713	-0.128	0.711
	1946	-0.380	0.713	-0.235	0.729
	1956	-0.518	0.746	-0.302	0.776
	1965	-0.689	0.709	-0.388	0.782
	1976	-0.521	0.781	-0.361	0.840
	1987	-0.413	0.889	-0.285	0.930
	1997	-0.436	0.955	-0.409	0.955
Costa Rica	1938	-0.320	0.727	0.008	0.756
	1956	-0.815	0.844	-0.662	0.817
	1968	-0.412	0.927	-0.311	0.894
	1978	-0.593	0.915	-0.401	0.925
	1992	-0.641	0.954	-0.492	0.961
Cuba	1961	-0.193	0.930	-0.158	0.865
	1975	-0.295	0.986	-0.442	0.893
	1991	-0.440	0.961	-0.365	0.918
Panama	1955	-0.422	0.823	-0.483	0.744
	1965	-0.266	0.920	-0.343	0.826
	1975	-0.425	0.918	-0.392	0.861
	1985	-0.454	0.948	-0.385	0.922
	1995	-0.570	0.951	-0.478	0.936
		Laggards			
Brazil	1985	-0.280	0.852	-0.063	0.904
	1995	-0.557	0.817	-0.433	0.862
Colombia	1944	-0.485	0.717	-0.408	0.692
	1957	-0.550	0.779	-0.561	0.752

	1968	-0.379	0.835	-0.142	0.794
	1979	-0.400	0.869	-0.553	0.825
	1989	-0.576	0.872	-0.317	0.885
	1999	-0.654	0.884	-0.398	0.912
Dominican	1955	-0.690	0.695	-0.609	0.688
Republic	1965	-0.467	0.799	-0.611	0.726
	1975	-0.432	0.846	-0.584	0.764
	1987	-0.546	0.866	-0.500	0.850
	1997	-0.849	0.816	-0.790	0.817
Ecuador	1956	-0.670	0.760	-0.415	0.730
	1968	-0.460	0.841	-0.332	0.803
	1978	-0.773	0.801	-0.574	0.811
	1986	-0.725	0.840	-0.511	0.850
	1995	-1.050	0.775	-0.817	0.795
El Salvador	1955	-0.551	0.713	-0.523	0.667
	1966	-0.646	0.755	-0.512	0.729
	1981	-0.814	0.685	-0.520	0.773
Guatemala	1957	-0.584	0.687	-0.229	0.682
	1968	-0.635	0.722	-0.333	0.741
	1977	-0.708	0.731	-0.313	0.782
	1987	-0.705	0.749	-0.346	0.806
	1998	-1.016	0.718	-0.589	0.796
Honduras	1955	-1.031	0.599	-0.898	0.582
	1967	-0.419	0.801	-0.219	0.753
	1981	-0.582	0.830	-0.321	0.824
Mexico	1925	-0.681	0.590	-0.145	0.661
	1935	-0.418	0.655	0.194	0.728
	1945	-0.526	0.691	-0.149	0.731
	1955	-0.748	0.705	-0.352	0.748
	1965	-0.785	0.735	-0.432	0.773
	1975	-1.016	0.709	-0.653	0.774
	1985	-0.850	0.769	-0.563	0.835
	1995	-0.950	0.799	-0.586	0.855
Nicaragua	1956	-0.547	0.694	-0.532	0.635
	1967	-0.843	0.663	-0.905	0.592
	1983	-0.775	0.745	-0.578	0.749
Paraguay	1956	-0.199	0.911	-0.576	0.783
	1967	-0.021	0.967	-0.152	0.890
	1977	-0.227	0.925	-0.253	0.881
	1987	-0.292	0.942	-0.292	0.891
	1997	-0.504	0.906	-0.509	0.858

Peru	1966	-0.657	0.722	-0.510	0.723
	1976	-0.584	0.803	-0.550	0.783
	1987	-0.549	0.861	-0.512	0.826
	1999	-0.533	0.894	-0.534	0.857
Uruguay	1969	-0.123	0.910	-0.104	0.935
	1980	-0.258	0.891	-0.228	0.928
	1990	-0.128	0.949	-0.269	0.954
	2000	-0.330	0.922	-0.591	0.905
Venezuela	1955	-0.351	0.789	-0.316	0.738
	1966	-0.427	0.808	-0.388	0.775
	1976	-0.574	0.804	-0.454	0.809
	1985	-0.616	0.833	-0.525	0.848
	1995	-0.707	0.842	-0.533	0.863

Note: α is the constant and β the slope of the logistic model

Table 5. Estimated coefficients of the regression between absolute change
In life expectancy and lagged causes of deaths, LAC countries, 1950-2005

Causes of Deaths	All Countries	Laggards Countries	Forerunners Countries
Vectorborne	0.155 *	0.023 *	0.007
	(0.006)	(0.009)	(0.008)
Waterborne	0.022	0.045 *	0.004
	(0.013)	(0.017)	(0.020)
Airborne	0.020	-0.004	0.044
	(0.022)	(0.027)	(0.038)
Intercept	0.167	0.221	0.096
	(0.071)	(0.090)	(0.120)
Adj R-squared	0.541	0.590	0.474
N	37	24	13

Note: The independent variables are the natural logarithm of the causes of death

* $p < 0.05$

Table 6. Contributions of Changes in Life Expectancy at Age 5, LAC countries, 1950-2005

Causes	Forerunners					Laggards				
	Total	5 - 19	20 - 39	40 - 59	60 +	Total	5 - 19	20 - 39	40 - 59	60 +
	Males									
Neoplasms	0.514	0.024	0.073	0.237	0.180	-0.039	-0.015	0.007	0.048	-0.080
Circulatory	1.698	0.048	0.119	0.516	1.015	0.362	0.023	0.071	0.138	0.130
Respiratory	0.368	0.098	0.094	0.131	0.045	0.605	0.162	0.134	0.172	0.136
Digestive	0.409	0.046	0.097	0.143	0.124	0.954	0.231	0.177	0.242	0.303
Diabetes	0.008	0.003	0.010	0.009	-0.014	-0.169	0.000	-0.007	-0.066	-0.096
Infections	0.910	0.147	0.228	0.316	0.219	1.776	0.531	0.413	0.471	0.360
Accidents	0.729	0.162	0.349	0.173	0.045	-0.563	-0.093	-0.318	-0.120	-0.032
Ill-defined	1.142	0.161	0.196	0.313	0.472	3.176	0.443	0.525	0.820	1.388
	Females									
Neoplasms	0.750	0.024	0.071	0.305	0.350	0.039	-0.015	0.035	0.075	-0.056
Circulatory	2.251	0.057	0.190	0.506	1.499	0.523	0.026	0.135	0.140	0.222
Respiratory	0.483	0.119	0.112	0.112	0.140	0.730	0.187	0.187	0.160	0.197
Digestive	0.469	0.045	0.105	0.164	0.155	1.184	0.243	0.250	0.306	0.385
Diabetes	0.067	0.006	0.004	0.017	0.040	-0.278	-0.002	-0.008	-0.080	-0.188
Infections	0.840	0.165	0.296	0.221	0.158	1.859	0.575	0.500	0.400	0.384
Accidents	0.257	0.086	0.096	0.033	0.042	-0.064	-0.012	-0.050	-0.008	0.007
Ill-defined	1.411	0.167	0.320	0.292	0.633	3.958	0.502	0.759	1.004	1.693

Note: Figures represent unweighted average of age-cause-specific mortality rates

Table7. Estimated coefficients of the regression between life expectancy at age 5 and socioeconomic determinants, LAC countries, 1950- 2005

Type of specification and period	Intercept	Gross Domestic Product (GDP)		Literacy		Adj R-squared	N
Logistic before 1970	138.221	0.007		-0.365	*	0.999	31
	(5.229)	(0.007)		(0.031)			
Logistic after 1970	147.348	0.008	**	-0.424	*	0.999	45
	(3.249)	(0.004)		(0.032)			
Linear before 1970	4.236	-0.003		0.206	*	0.813	31
	(0.042)	(0.004)		(0.020)			
Linear after 1970	4.302	-0.004	**	0.233	*	0.795	45
	(0.023)	(0.002)		(0.019)			
Fixed effects before 1970	3.561	0.072	**	0.155	**	0.328	31
	(0.275)	(0.028)		(0.070)			
Fixed effects after 1970	4.130	0.015		0.313	*	0.625	45
	(0.123)	(0.011)		(0.057)			

Note: The dependent variable is the logarithm of Life Expectancy at age 5 in Linear and Fixed Effect Models. The independent variables are the natural logarithm of the GDP and Literacy

* $p < 0.00$, ** $p < 0.05$

Table 8. Estimated coefficients of the regression between the proportion proportion of gains in life expectancy at age 5 and cause of death, LAC countries, 1950-2005

Causes of Deaths	Gains associated to changes in parameters		Gains associated to changes in variables	
Vectorborne	0.045		-0.026	
	(0.036)		(0.055)	
Waterborne	0.232	*	-0.346	*
	(0.123)		(0.191)	
Airborne	-0.189	*	0.256	
	(0.101)		(0.156)	
Intercept	-0.658	*	-0.857	
	(0.371)		(0.573)	
Adj R-squared	0.446		0.275	
N	16		16	

Note: Dependent and independent variables are expressed as natural logarithm. Rates of mortality are considered for the total population.

* $p < 0.10$

Figure 1. Differences between Observed and Expected Life Expectancies at Age 5, for West, North, East and South models, LAC countries, 1925-2000

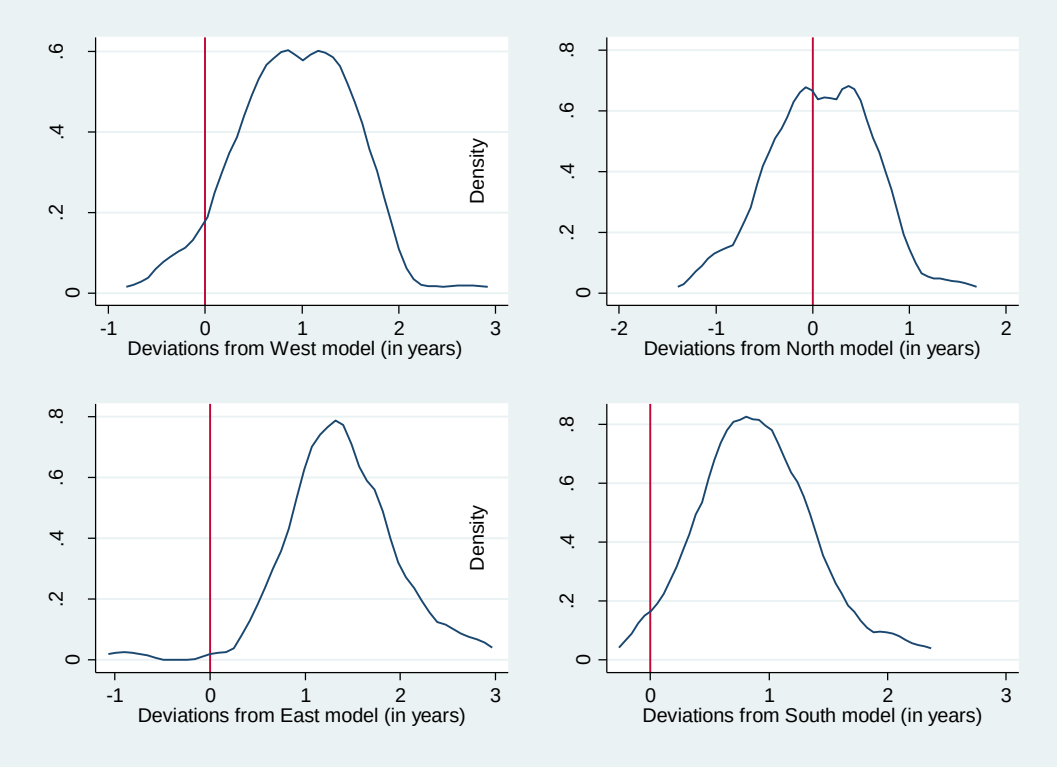


Figure 2. Female Life Expectancies at Age 5 by Group of LAC countries, 1925-2000

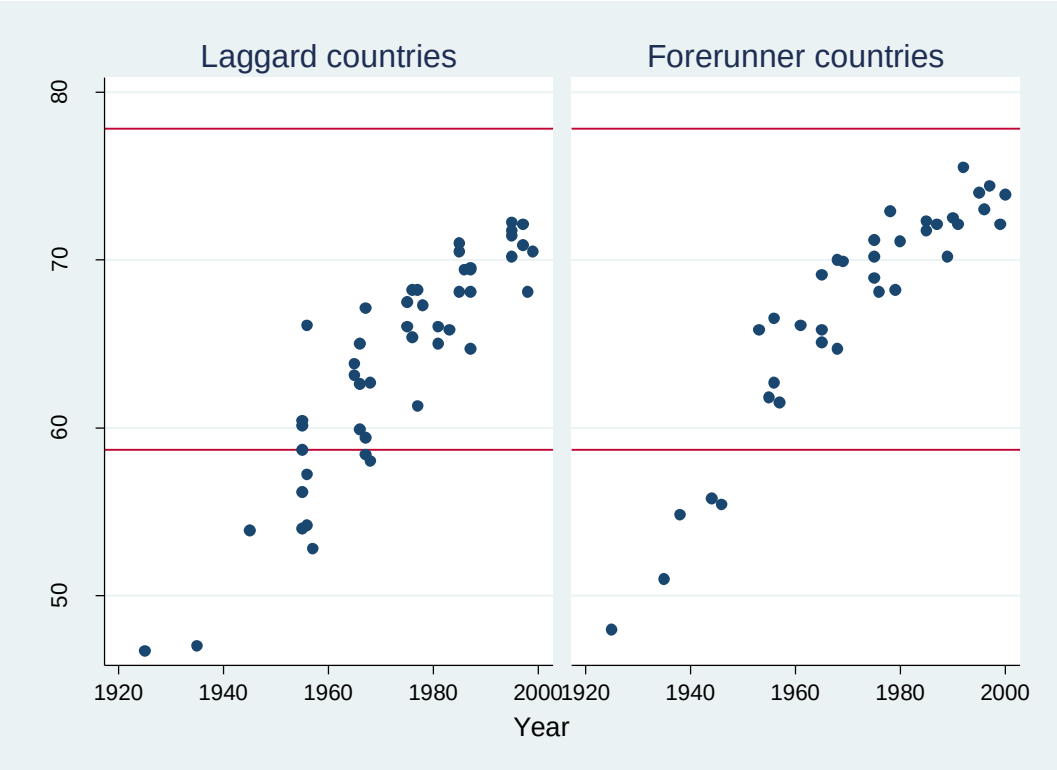
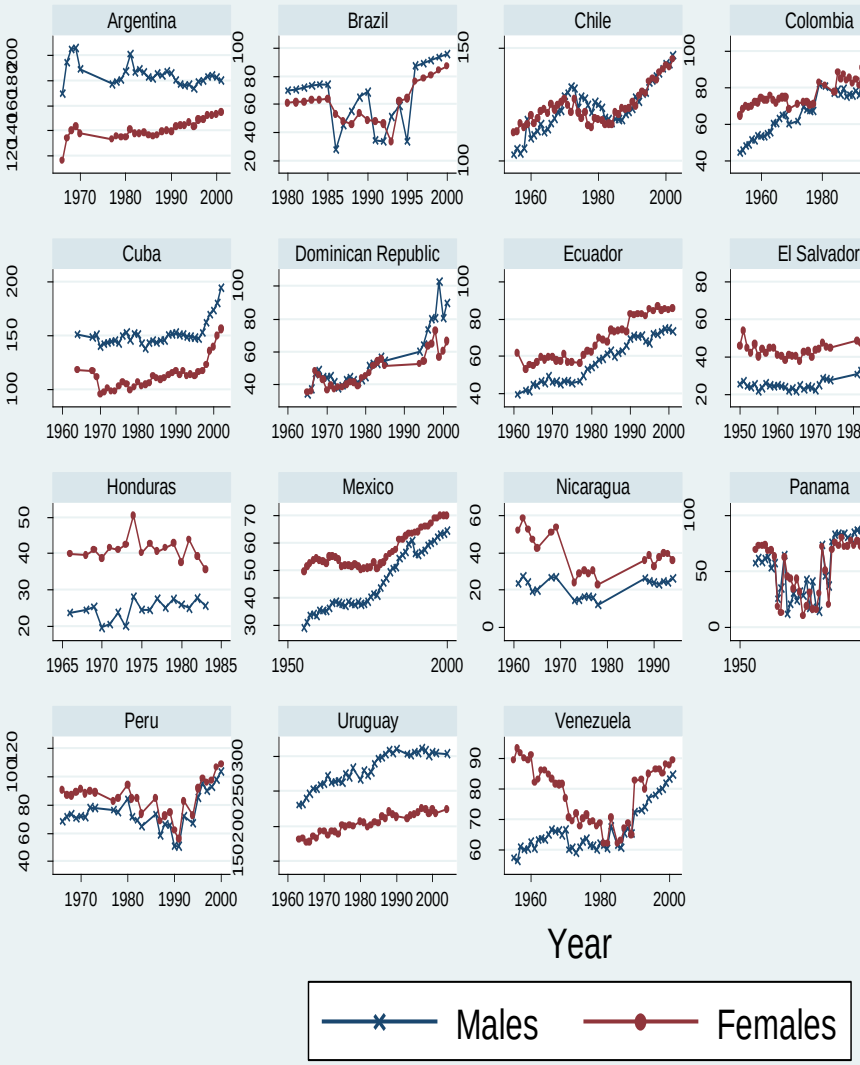
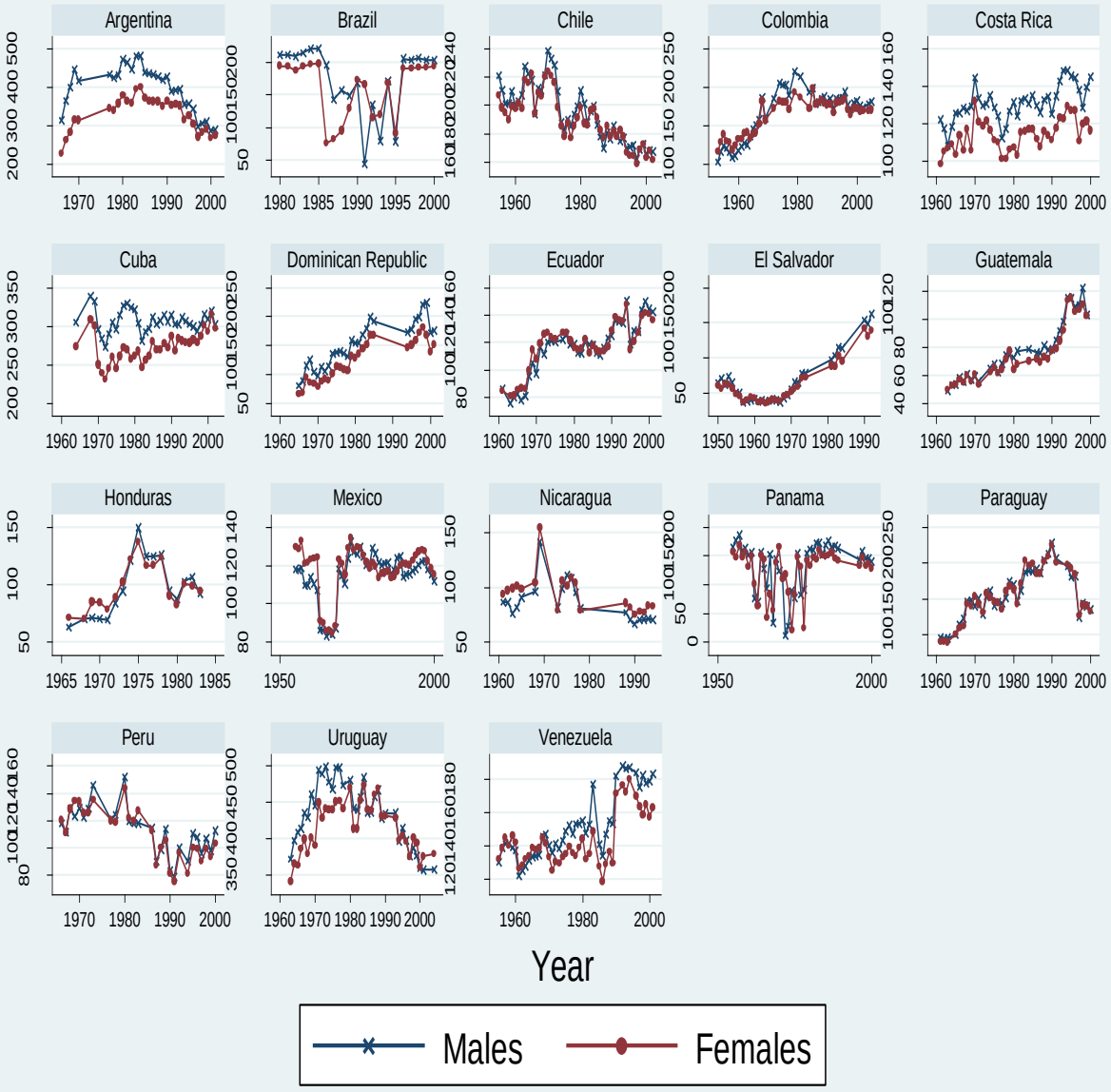


Figure 3a. Total Neoplasm Mortality Rates (per 100,000 persons) LAC countries, 1950-2000



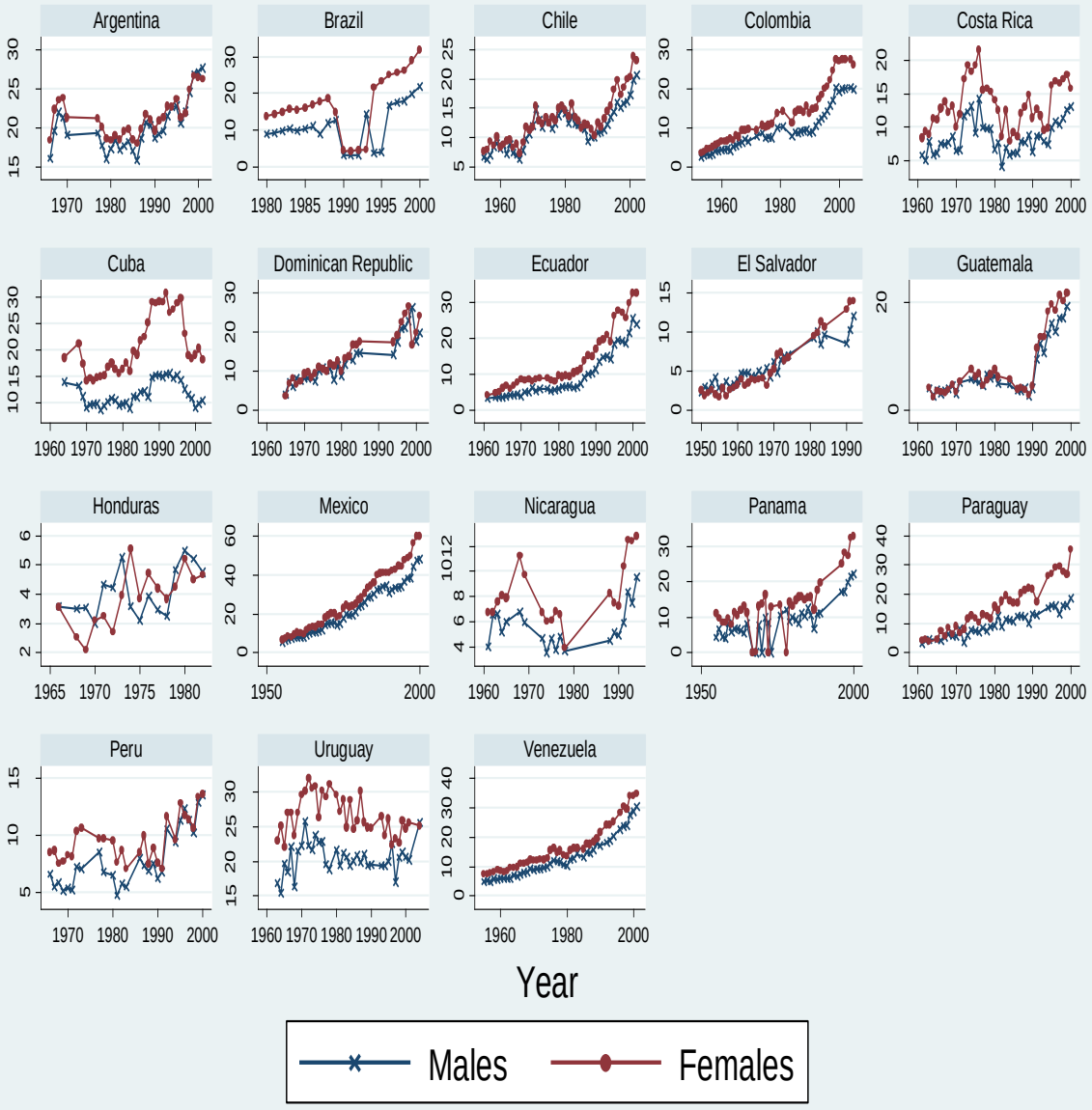
Source: WHO Database

Figure 3b. Total Circulatory Mortality Rates (per 100,000 persons) LAC countries, 1950-2000



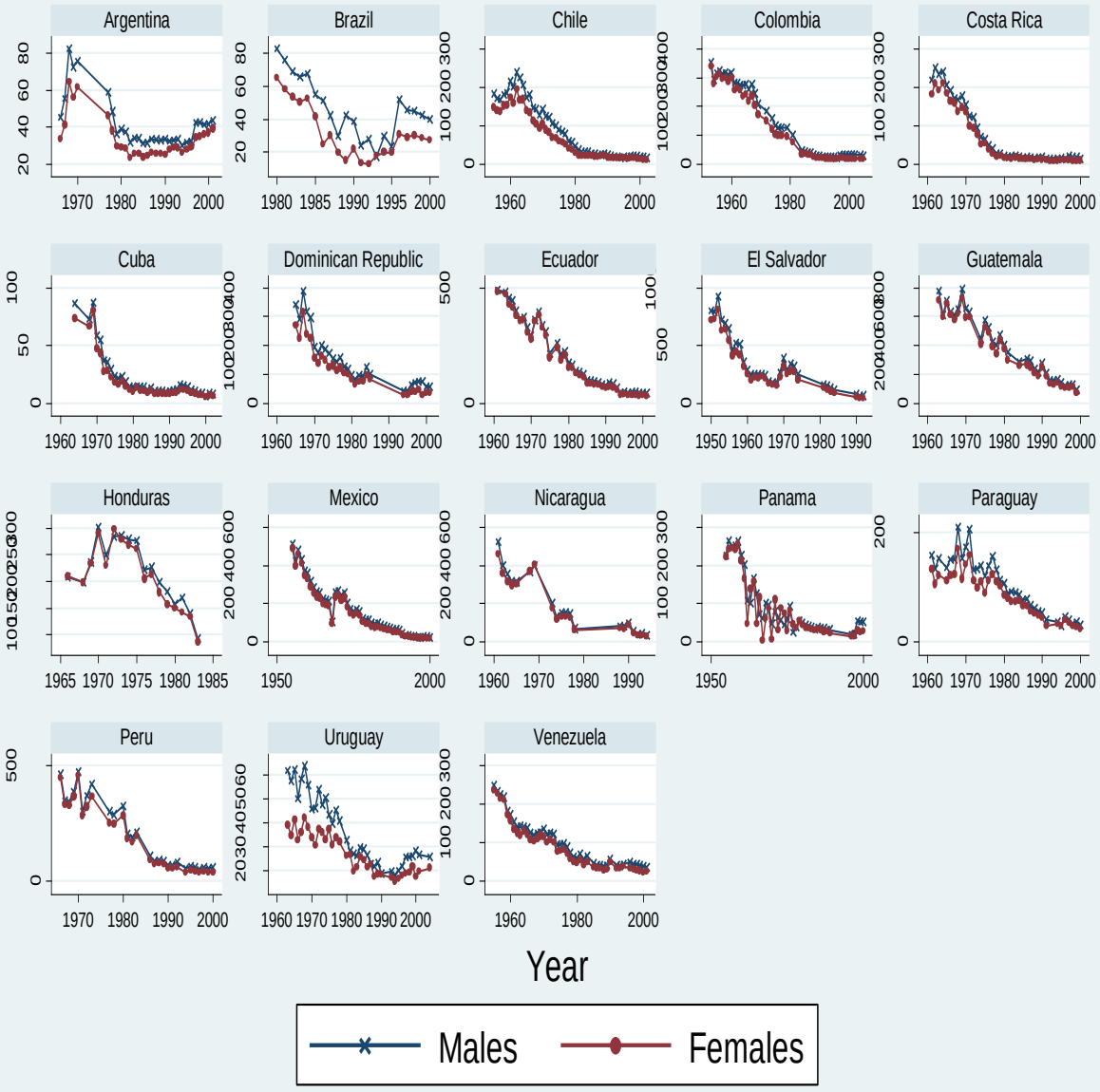
Source: WHO Database

Figure 3c. Total Diabetes Mortality Rates (per 100,000 persons) LAC countries, 1950-2000



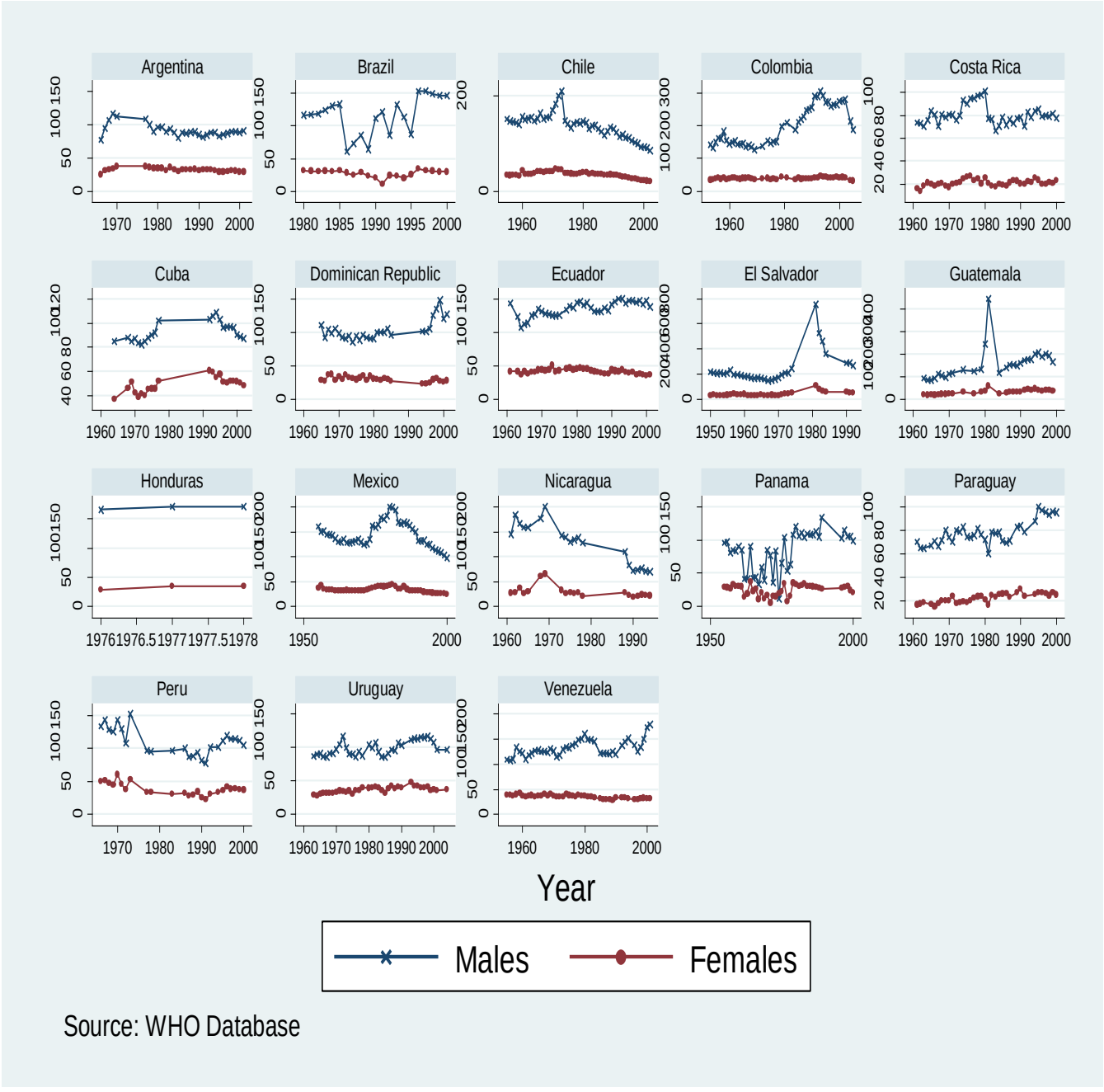
Source: WHO Database

Figure 3d. Total Infectious Mortality Rates (per 100,000 persons) LAC countries, 1950-2000



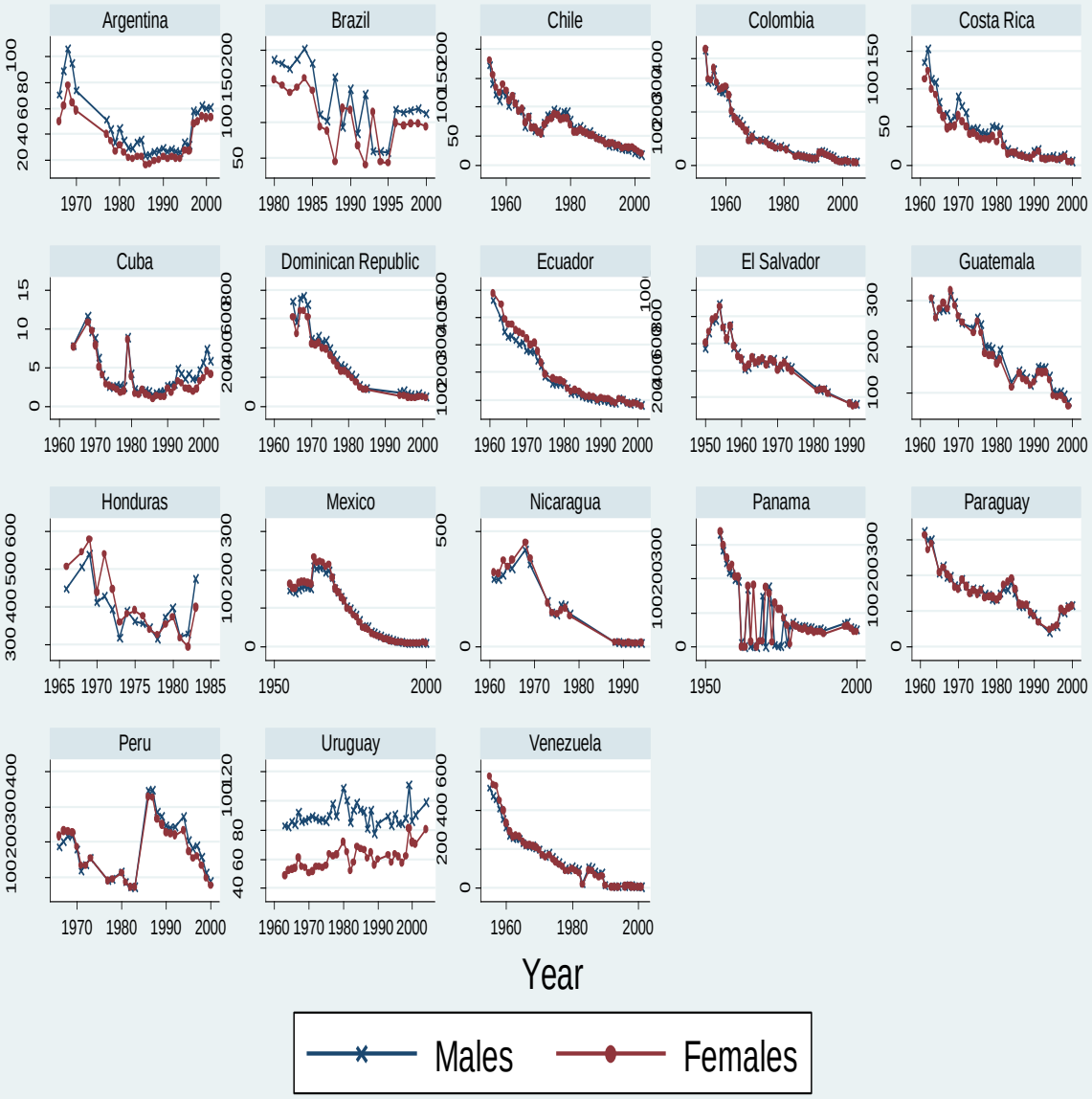
Source: WHO Database

Figure 3e.Total Accidents, Suicides and Violence Mortality Rates (per 100,000 persons) LAC countries, 1950-2000



Source: WHO Database

Figure 3f. Total Ill-defined and Senility Mortality Rates (per 100,000 persons) LAC countries, 1950-2000



Source: WHO Database

Figure 4a. Contributions of Changes in Life Expectancies at Age 5, Forerunner Countries, 1950-2000

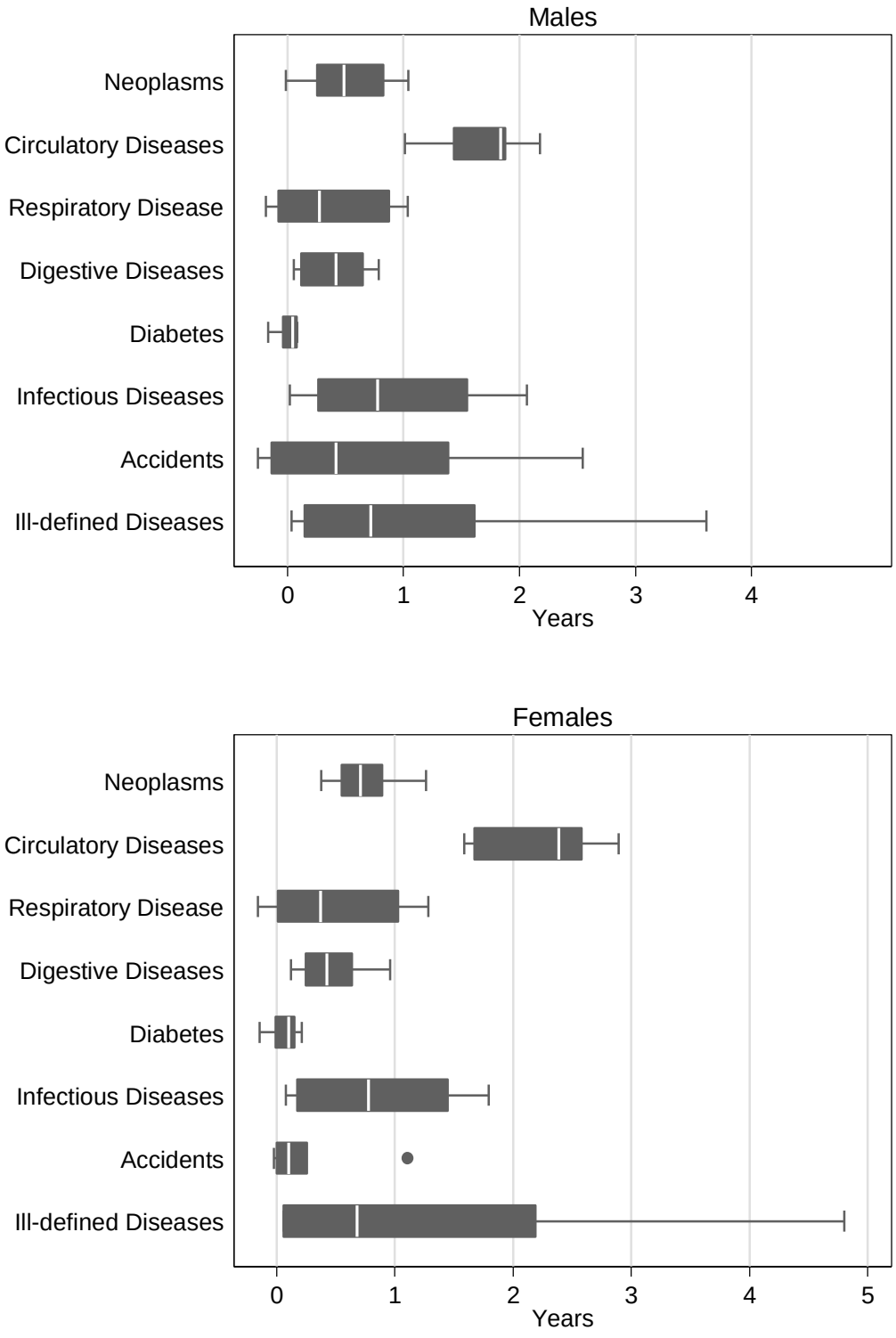


Figure 4b. Contributions of Changes in Life Expectancies at Age 5, Laggard Countries, 1950-2000

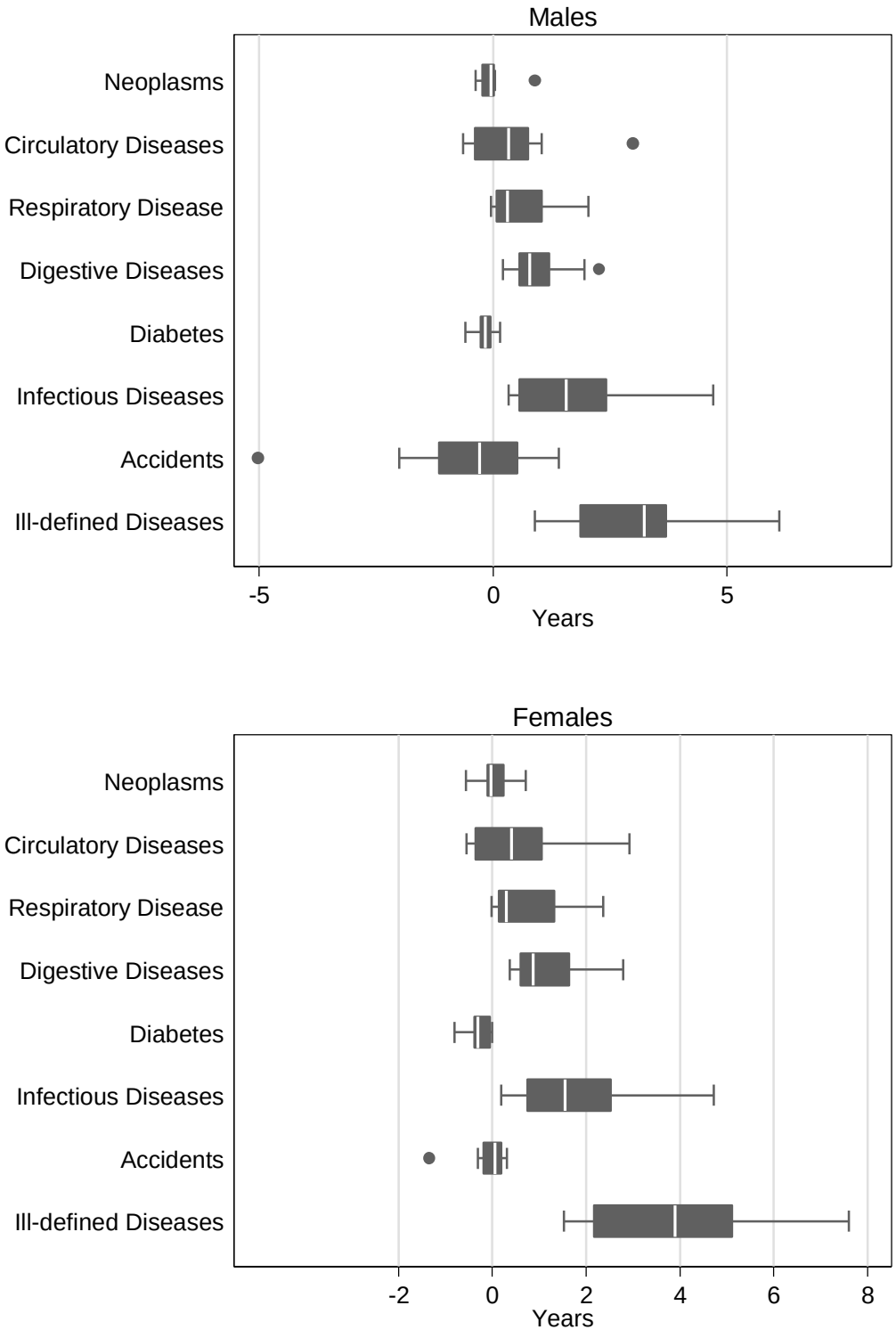


Figure 5: Relations between a measure of wealth (GDP), life expectancy at age 5 and unmeasured conditions

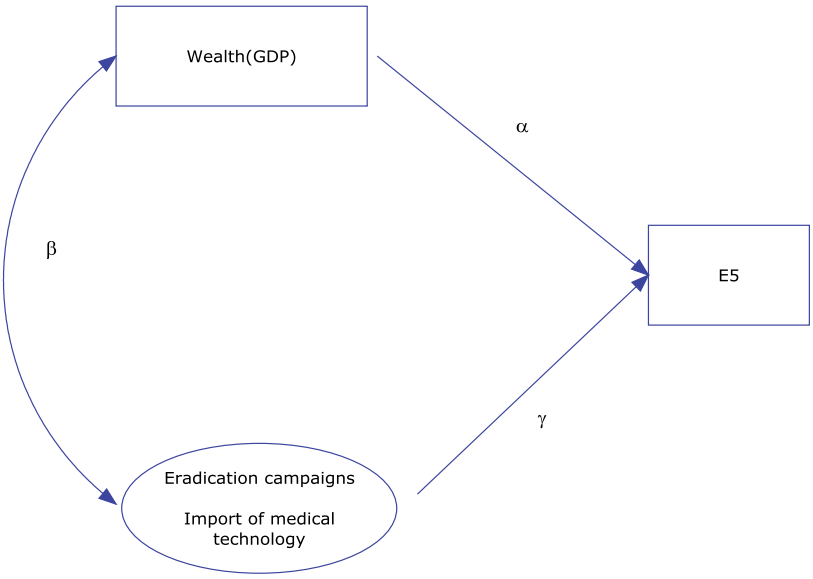


Figure 6. Estimated Percentage of the Total Change in Life Expectancies at Age 5 attributable to changes in Variables and Parameters, LAC Countries, 1925-2000

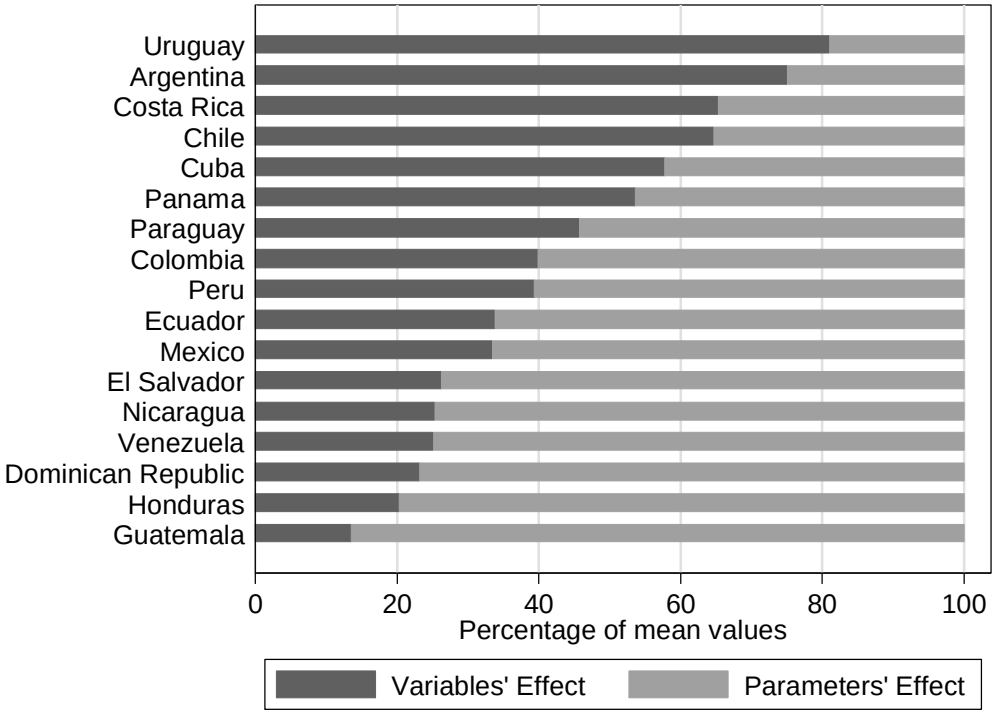


Figure 7. Female Life Expectancies at Age 60 by Group of LAC countries, 1925-2000