Life expectancy, healthy life expectancy and the effects of early life conditions: the case of Latin America and the Caribbean *

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Abstract: Older people attaining age 60 after 1990 in Latin America and the Caribbean are survivors scarred by heavy exposure to infectious diseases early in life. According to influential theories these experiences could translate into higher susceptibility to adult chronic conditions and mortality. In this paper we test the conjecture that improvements in longevity initiated more than seventy years ago created conditions increasing the susceptibility to adult chronic diseases and mortality of cohorts that were beneficiaries of gains in survival. These effects could compromise future gains in life expectancy and healthy life expectancy. To evaluate the conjecture we use data for Mexico and Puerto Rico. We conclude that, even if theories supporting the conjecture are confirmed, it is unlikely that the passage of the affected cohorts through older ages will have significant negative effects on future life expectancy and healthy life expectancy.

I. INTRODUCTION

Western Europe experienced sustained decreases in adult mortality rates during the last fifty years. The rate of decline of rates at older ages after 1950 is estimated to be close to 1% per year (Kannisto, 1994, Kannisto et al., 1994, National Research Council, 2000). If maintained for fifty years with initial levels of life expectancy at age 60 between 15 and 20 years a 1% per year decline in mortality rates above 60 yields, on average, gains in longevity of the order of 0.10 years per year. Further optimism regarding the potential for sustained increases in life expectancy is boosted by evidence showing that since early in the century attained life expectancy systematically surpasses ceilings forecast in the past (Oeppen and Vaupel, 2002). With notable exceptions, the persistent worldwide extension of life will be fed from a single central source, namely, gains in survival accruing at ages older than 50 or 60. Indeed , recent research confirms that important gains in survival are taking place even at very old ages (Horiuchi and Wilmoth, 1998, Wilmoth and Horiuchi, 1999, Wilmoth, 1998).

This optimistic scenario is consistent with recent evidence from Latin America (Palloni and Pinto, 2004) that shows increases in life expectancies at age 60 from about 18 years in 1950 to about 23 in 1995 in approximately *linear fashion*. This rhythm of progress yields average yearly gains of 0.10 years per year, close to the rate of change in Western Europe.

The optimistic view, however, is not without its detractors who more gloomily point out that *lifestyle changes* embraced by newer cohorts of elderly people both in high and low income countries could oppose strong resistance to further improvements in longevity (Olshansky et al., 2005, Preston, 2005). An alternative conjecture tailored for countries in the Latin America and Caribbean region (LAC) rests on the idea that members of cohorts that will attain age 60 after the year 2000 experienced the full benefit of medical technologies deployed after 1940. The

widespread utilization of these technologies led to the fastest mortality decline ever recorded and operated by minimizing case-fatality rates and without always improving nutritional status, standards of living or even significantly reducing exposure to infectious diseases. If theories linking early nutritional status and health conditions to adult health and mortality prove to be correct, the past experience of these cohorts will exert a powerful brake on further reductions of morbidity and mortality.

Either of these alternative interpretations cast doubts on the validity of optimistic assessments of future improvements in longevity and healthy life expectancies. These two more pessimistic views are related to each other because one of the mechanisms that could explain rising prevalence of conditions such as obesity and diabetes is the interaction of changing life styles and early exposure to deleterious health conditions (Barker, 1998; Gluckman and Hanson, 2006)

In this paper we bring evidence to bear on the question of future changes in life expectancy and healthy life expectancy. We do so by examining the experience of two countries in the region, Mexico and Puerto Rico, for which we have very rich data sets[1]. We focus on the second pessimistic view and show that the changing composition of elderly by early health conditions cannot cause a slow-down in mortality changes and health improvements[2]. To support

[1] A word is in order to justify the choice of data sets for the analysis. After Brazil, Mexico is the country that will contribute the largest share of elderly population in the region. In addition, the demographic profile of Mexico is best viewed as typical among countries in LAC and represents well the nature of aging elsewhere in the region. Although Puerto Rico is not considered a 'country' on its own right, it is a society that shares a much larger stock of cultural heritage and demographic history with countries in the LAC region than it does with any other society. The fact that Puerto Rico has been a US territory for longer than a century no more destroys our ability to exploit the common legacy it has with other diplomatically recognized countries in LAC than the youth of independent nation-states in Africa undermines the justification of treating them as demographic entities different from France, England, Belgium or any other of the colonial powers that marauder in the region. The comparison between Mexico and Puerto Rico is not only legitimate and relevant but should shed a great deal of light on the nature the aging process in the entire LAC region.

[2] According to the intermediate United Nations (2002) population projection up to 2050, Mexican life expectancy at ages 60 and above will gain, on average, about 0.09 years per year within the period 2007-2027 and 0.06 years per year during the period 2027-2047. Thus, there is a presumption of a declining rate of improvements which, in any case, amounts to assume that mortality rates at ages above 60 will decline at an average rate somewhat below 1% per year. In the case of Puerto

this argument we estimate bounds for expected changes in life expectancy and healthy life expectancy at age 60 contributed by the changing composition of cohorts. We conclude that the resistance to improvements in life expectancy and healthy life expectancy embedded in the changing composition of cohorts by early health status is not enough by itself to halt or substantially alter future improvements.

The paper is organized as follows. In Section II we formulate the core of the argument. First, we review evidence supporting the existence of mechanisms linking early and late adult health, a phenomenon we refer to as the "early-late health connection". We then discuss the existence of different regimes of mortality decline, identify the conditions they seed for the expression of the early-late health connection and spell out their implications for longevity among older cohorts. In Section III we describe the data sets for Mexico and Puerto Rico and formulate estimation procedures. Section IV presents results and Section V concludes.

II. EARLY CHILDHOOD, ADULT HEALTH AND THE ROLE OF REGIMES OF MORTALITY DECLINE

Anticipating the future trajectory of mortality rates at older ages requires not only to assess the potential impact of new technology, behaviors and access to health services but also to understand the nature of life cycle experiences of the cohorts involved. In fact, under some conditions to be spelled out below, experiences in early life may partially determine mortality and morbidity in adult life. Is this the case in the LAC region? If so, how powerfully could they influence future trends in life expectancy and healthy life expectancy in the region?

Rico the UN projections imply that gains in life expectancy at age 60 will remain steady and close to 0.06 years per year through the end of the projection period implying reductions in the force of mortality above age 60 of the order of 0.4% per year}

A. Linkages between early childhood and adult health status

Rapidly accumulating evidence mostly from developed countries suggests there are a number of mechanisms through which early childhood conditions may affect the onset of adult chronic conditions and, in particular, adult diabetes (diabetes of type II) and heart disease. Some of these mechanisms are highly specific such as those associated with the *sequelae* of processes that start *in utero*, develop shortly before and/or around birth ("fetal origin hypothesis") or during other "critical periods" (Barker, 1998, Gluckman and Hanson, 2006). They include also a few, less specific pathways, such as those that operate through socioeconomic conditions experienced in early childhood, including among other things stressful environments, or thought to be associated with acute episodes of some childhood illnesses and their cumulative influence on the late onset of chronic diseases (Ben-Shlomo and Smith, 1991, Danese et al., 2007, Dowd, 2007, Elo, 1998, Elo and Preston, 1992, Hertzman, 1994, Kuh and Ben-Shlomo, 2004, Lundberg, 1991, Smith and Lynch, 2004). A somewhat different set of pathways involves the delayed effects of inflammatory processes triggered by recurrent exposure to and contraction of infections and parasitic diseases during early ages (Crimmins and Finch, 2006, Danesh et al., 2000, Fong, 2000, McDade et al., 2008, Finch and Crimmins, 2004). Empirically distinguishing between these various mechanisms or pathways is a thorny affair because, with some qualifications we examine later, they all lead to the same implication, namely, that the erosion of conditions that foster malnutrition and/or exposure to and contraction of infections and parasitic diseases will simultaneously reduce infant and early childhood mortality as well as subsequent adult mortality among members of the same birth cohort. As shown below, a partial escape from this identification problem can be secured through an understanding of the macro determinants of mortality changes. Thus, even in situations where the bulk of mortality changes may be

confined to early childhood, a single determinant may have repercussions spread across the entire lifespan of cohorts[3].

The existence of an early-adult health connection implies that to the extent that successive birth cohorts are exposed to changing conditions early in life, their older age morbidity and mortality are, to some degree at least, dependent on conditions set forth by their early experiences. If so, there is potential for within-cohort associations between mortality and morbidity at early and at older ages. In such cases assessment of future changes in health status and mortality could be partially supported by examination of past levels and patterns of child mortality and health status. But, as we show below, the magnitude and direction of the association is largely dependent on the regime of mortality decline. It is the nature of this regime that widens or narrows the opportunity for the expression of early-late health connections. Each regime has distinctive implications for the subsequent survival of cohorts' members who are either scarred by, exposed or immune to, or escape altogether from conditions experienced during early childhood.

B. Regimes of mortality decline and the expression of early-adult health connections

The observation that in some cases there is a strong within-cohort correlation between mortality early in life and at older ages is not new (Hobcraft et al., 1982; Mason and Smith, 1983; McKermack et al., 1934). Its presence has been interpreted in two different ways. The first is a selection argument that sees attrition early in life as a filtering device resulting in the disproportionate survival at older ages of the least frail. In such case the within-cohort

[3] There is some literature contending that part of the secular improvements in mortality operated through changes that affected entire cohorts. This view is in opposition to the idea that mortality improvements are dominated by sequences of period-changes, e.g. affecting all cohorts at the same time (Barbi and Vaupel, 2005).

association is expected to be negative[4]. The second interpretation invokes the early-late health connection but, unlike the first one, it does not lead to an automatic expectation about the sign or magnitude of within-cohort correlation. This is because the precise outcome of the early-late health connection depends on whether or not the mortality regime is stationary. If not, the outcome will be a function of the dominant causes of mortality changes experienced by successive cohorts. Suppose that we express the within cohort relation between early and late mortality with a simple linear model between two arbitrary age groups:

$$\ln({}_{10}Q_{60}(t)) = \alpha(t) + \beta(t) \ln({}_{5}Q_{0}(t-60)) + \varepsilon(t)$$

where ${}_{10}Q_{60}(t)$ is conditional probability of dying between ages 60 and 70, ${}_{5}Q_{0}(t-60)$ is the probability of dying before age 5 in the life table 60 years before and α and β are coefficients. In a stationary mortality regime the coefficients are time invariant. When the mortality regime is changing, the coefficients could also change. Thus, for example, the relation estimated in the year 2000 will reflect the experience of cohorts born after 1930 whereas the one estimated in 2020 will reflect the experience of cohorts born after 1950. Thus the sign and magnitude of $\beta(t)$ is a function of the nature of *changes* in mortality experienced by cohorts born about 60 years before. The main relations that influence the magnitude and sign of $\beta(t)$ are displayed in the following diagram:

[4] The correlation could be negative as a result of reverse selection: those who are filtered early may be the least frail leaving a pool of survivors with increased frailty. This selection exists in the case of wars.

Early-Late Health mechanisms and Mortality regimes

	Type of early-late health connection(*)		
	Nutritional Status	Experience with particular diseases (rheumatic heart fever/ Helycobacterium pylori)	Experience with broad range of diseases (infections of digestive and respiratory tracts)
Mortality Regime			
Standards of living	[1] (++)	[2](?)	[3](+)
Public Health	[4](+)	[5](?)	[6](+)
Medical Innovations	[7](--)	[8](+)	[9](--)

(*) Numbers within squared brackets are cell numbers; symbols in rounded brackets represent positive relation (+), negative relation (--) and indeterminate sign(?)

Although highly stylized this diagram displays the main relations of interest. The columns represent the three main mechanisms producing the early-late health connection. The first operates through impaired fetal, prenatal and postnatal growth and is associated with nutritional status (Barker, 1998). The second depends on the onset and development of well-defined illnesses during early childhood that generate tissue damage or disable immune response but repercussions are seen only in adult life (Elo and Preston, 1992). The third is more diffuse as adult response is sensitive to early exposure to or contraction of an array of infectious illness

with the potential to generate a sustained, enduring inflammatory response (Crimmins and Finch, 2006)[5].

The rows in the diagram represent the dominant forces promoting mortality changes at early ages, say at time t-60. Again, the three forces identified in the diagram are not exhaustive as most historical mortality changes are driven by a combination of all three rather than by only one of them to the exclusion of the others.

Suppose that the mechanism generating the early-late health connection is nutritional status. When the regime of mortality changes affecting the early life of a cohort is driven by medical technologies only (cell 7), the expected sign of $\beta(t)$ should be negative as the implementation of technology will only reduce rates of contraction and increase rate of recovery (decreasing ${}_5Q_0(t\text{-}60)$, without inducing a major effect on nutritional status (not changing the risks of deleterious conditions to be expressed later in life).

Suppose next that the main mechanism responsible for the early-late connection is early experience with infectious diseases and that the main force behind mortality changes is a sweeping change in public health (cell 6). We expect $\beta(t)$ to be positive as the advantages accruing to cohorts in the form of reduced exposure to infectious diseases translate later into lower incidence of adult chronic illness that are sensitive to sustained inflammation.

Finally, suppose that the early-late health connection is due to the early experience of one well-defined disease. The sign and magnitude of $\beta(t)$ will be indeterminate and will depend on the actual change induced by a particular mortality regime. If medical technologies are dominant through increased rates of recovery and not through changes in rates of contraction, then $\beta(t)$ will

[5] There are of course other mechanisms that could be responsible for the early-late health connections. But the three identified in the diagram are the most studied in the literature.

be negative as enhanced survival of those who experience the disease will increase the pool of people among which the early late health connection can be expressed.

Cases in row 2 represent the experience of many developed societies. In most of these cases the initial push toward massive mortality decline is rooted in a public health revolution that went a long way toward minimization of exposure to infectious diseases. If the main mechanism accounting for the early-late connection worked through nutritional status (column 1) we would expect $\beta(t)$ to be positive and high. This is due to the synergistic relation between nutritional status and infections documented elsewhere (Scrimshaw and SanGiovanni, 1997): less exposure to infectious diseases implies improved nutritional status and this, in turn, lower likelihood of onset of adult illnesses related to poor early nutrition. Similarly, reduction in exposure to infectious diseases implies reduced load of inflammatory processes and higher likelihood of avoiding adult chronic conditions triggered by sustained inflammation.

C. The case of Latin America and the Caribbean

Historical conditions in the LAC region and, more widely, in most developing countries fit quite well in the third row in the above diagram. It is known that the mortality decline that took place in low income countries after 1940 or 1950 was largely driven by the dissemination of relatively cheap medical technologies (row 3) (Palloni, 1990, Arriaga and Davis, 1969). The second major force, e.g. improvements in public health, was also implicated but to a much lesser and with a much higher level of intercountry heterogeneity. With a few exceptions, improvements in standards of living played only a minor role in the initial phases of the decline.

Although there is substantial variability in the time of onset, most countries in the LAC region began an uninterrupted and sharp mortality decline around 1930 and, most definitely,

after 1950. To be sure, there are exceptions: Argentina, Uruguay, Costa Rica and Cuba are forerunners that resemble more the Western European style of mortality decline than their neighbors' experience. For the first 20 to 30 years, the largest fraction of the decline is associated with decreases in mortality before age five. After 1950 there is a sharp acceleration of the rate of mortality decline that coincides with a period during which chemotherapy makes its debut in the area and begins to be widely used. Empirical investigations show that the bulk of this decline is associated with the deployment of public health tactics and medical technology that diminished exposure to infectious and parasitic diseases and decreased their lethality (Palloni and Wyrick, 1981, Preston, 1976). The remaining mortality decline is associated with improvements in standards of living (income) and better levels of nutritional status. These estimates are coarse and somewhat fragile but shed some light on the processes that evolved in the region.

Even if the above estimates were on firmer ground it would be difficult to classify the LAC experience squarely in row 3. This is because there are synergisms between infectious diseases and nutritional status that cannot be overlooked. And while the deployment of new medical technology might indeed work through a reduction of lethality there can be spill over effects in the form of enhanced nutritional status due to a reduction in the intensity or duration of illnesses. Finally, vaccination campaigns were a late addition to the package of medical innovations deployed after 1950. These act entirely through reduction of exposure and therefore by diminishing the load of infectious diseases and enhancing nutritional status.

In summary, the historical data suggest that the experience of LAC belongs in row 3 of the diagram but consideration of synergisms raises a question about the relevance of forces related to improvements in nutritional status. Choosing conservatively we classify the LAC experience midway between rows 2 and row 3. If one assumed a broad interpretation of the mechanism that

causes the early-late health connection we would exclude column 2 from the diagram and this leads to predict a positive sign for $\beta(t)$ if synergisms are strong (cells 4 and 6) and negative if synergisms weak (cells 7 and 9).

D. What if synergisms are only modest?

Simple calculations show that at least 40% of the rate of increase of the population aged 60 above between the years 2000 and 2020 in the LAC region will be associated with the post-1940 mortality decline (Palloni et al., 2006, Palloni et al., 2007). This fact and the nature of the mortality decline described above, suggest that the rate of increase of the elderly in the region is partly the product of augmented survival among individuals who were exposed to and who experienced bouts of infectious and parasitic illnesses but who survived them in a new medical environment of bolstered recovery rates. In the post-1990 period an increasing fraction of elderly will belong to birth cohorts whose members survived infectious and parasitic diseases that prior to the mortality decline would have killed them. To the extent that a nontrivial part of this mortality decline results from the efficacy of chemotherapy, the fraction of adult individuals in a cohort who are likely to have experienced suboptimal nutrition and/or frequent episodes of infections and parasitic diseases during childhood, will increase rather than decrease once the mortality decline gets under way. It follows that the prevalence of adult chronic illnesses ought to increase over time[6]. And therein lies the rub: *under these conditions life expectancy and healthy life expectancies at older ages will increase more slowly or cease to increase altogether even if 'background' mortality (i.e. mortality unrelated to the target chronic conditions)*

[6] If the association between early exposure to infection and late inflammation is negative, as McDade et al. (2008) have found in the Philippines, then the inference should be that the new cohorts of elderly people should experience less, not more, risks of suffering from at least of cardiovascular diseases.

continues to decline. Thus, if no other forces operate the association between changes in early childhood and in old age mortality across cohorts will drift to zero and become negative ($\beta(t)<0$) and, as a consequence future levels of mortality among elderly will decline more slowly, cease to decline, or even increase.[7]

In what follows we analyze data that can falsify this conjecture. We use information for two countries in LAC, Mexico and Puerto Rico, for which we have empirical estimates of mortality rates among elderly adults, prevalence of selected chronic conditions, and of the effects associated with early childhood health status. Our goal is to *estimate bounds* for the effects of the above described influx of cohorts with worse early experiences. We show that even if one were to completely neglect the effect of synergisms and adopted rather extreme assumptions about the rate of increase in the proportion of individuals who have experienced early conditions, the bounds are very narrow and point toward very weak long term effects on longevity and healthy life expectancy.

III. DATA, MEASUREMENT AND ESTIMATION PROCEDURES

A. Data Sources

We use data from the Mexican Health and Aging Study (MHAS) and Puerto Rican Elderly: Health Conditions (PREHCO), two two-wave panels of nationally representative samples of elderly individuals. In both surveys, the interviews were conducted with elderly adults including those with cognitive limitations who required the presence of a proxy to provide information, and with their surviving spouses regardless of age. The data collected offers a

[7] Two conditions can preclude this fate: (a) decreases in lethality rates associated with the target adult chronic conditions are large enough to compensate for increases in their prevalence or (b) the decline in the rate of background mortality is large enough to more than offset increases in the prevalence of the target chronic conditions.

substantial amount of information within the limits permitted by face to face interviews. The questionnaire includes, among others, modules on demographic characteristics, health status, cognitive and functional performance, and anthropometric measurements.

PREHCO I project was designed to gather quality baseline data on issues related to the health of the non-institutionalized population age 60 and over resident of Puerto Rico as of June 1st, 2000.[8] The sample is a multistage stratified sample with over-sample of regions heavily populated by African descent and of individuals aged over 80. A total of 4,291 in-home face-to-face target interviews were conducted between May 2002 and May 2003. In addition 1,442 spouses were interviewed, 1,042 of them 60 or older. Only 4.7% refused to participate and the overall response rate was 93.9%. PREHCO II is a follow up of targets and spouses interviewed as part of PREHCO I. The survey took place between June of 2006 and November of 2007. A total of 3,891 interviews of targets and 1,260 spouses were carried out for an overall response rate of 90.6% for targets and 89.61% for spouses. Among targets and spouses over 60, there were 867 individuals who had died and 55 who were institutionalized in the inter-wave period, whose interviews were completed using a proxy. Four hundred and two targets and spouses over 60 were assigned a non-response code: 142 of them refused to be interviewed and most of the remaining 260 could not be located or moved to the mainland. [9]

MHAS is a nationally representative, prospective panel study of Mexicans aged 50 and over as in 2000 funded by the National Institute on Aging. Baseline interviews were completed in the summer of 2001 with about 15,000 respondents, including target and spouses. The individual non-response rate of 10.5% for a population based survey is very low. The second

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[8] The study, a joint venture between the Center for Demography and Ecology of the University of Wisconsin-Madison and the Graduate School of Public Health of the University of Puerto Rico, funded by the National Institute on Aging and supported by the Legislature of Puerto Rico, is the largest ever about the elderly population in Puerto Rico.

[9] For more information on PREHCO see the study website http://prehco.rcm.upr.edu/

wave of MHAS was successfully fielded in 2003. An exit-interview was sought in 2003 with a next-of-kin of deceased persons; about 540 next of kin interviews were obtained, a number consistent with expectations given the mortality levels in Mexico. Sample attrition was small (7% at the individual level) for a total of about 12,000 follow-ups with surviving individuals who were age 50 or older at the baseline. All data files for MHAS 2001 and the 2003 follow-up are now public use[10].

Table 1 displays a summary of the MHAS and PREHCO samples that will be used to estimate parameters. Both samples contain individuals who are observed at baseline and follow-up. In our analyses we used all elderly individuals who are above 60 years old (targets and spouses) including those who responded via proxies.

Table 1

B. Measurement of poor early childhood health conditions

We start with a disclaimer: the surveys we use are not ideal to retrieve indicators of the type of early conditions mechanisms invoked in the biological and epidemiological literature. Instead we are constrained to use indirect measures of early child and adolescent nutritional status and growth (knee height), self-reported experience with illnesses in childhood that have been linked to adult conditions (polio, tuberculosis, rheumatic heart fever), and finally, response to questions about health status before age 15 for Puerto Rico and before age 10 for Mexico. We experimented with different specifications of the indicator of poor early childhood health (PEC). We settled for a broad definition that considers different dimensions of conditions experienced early in life and also produces the most robust results. It attains a value of one if at least one of

[10] For details on the study and access to the data, see the study website at www.mhas.pop.upenn.edu.

the following applies: individual knee height is below the first quartile of the distribution (an indicator of early stunting); individual experienced any of the three diseases mentioned above; and respondents recalled having experienced poor health for long stretches of time during the childhood. According to this definition, about 38% of the Mexican elderly (MHAS 2000) experienced PEC whereas in Puerto Rico the figure is about 37% (PREHCO 2000).

C. Estimation Procedures

The plan of analysis is in four stages: first we estimate the effect of the indicator of poor early childhood conditions on the probability of having diabetes and/or heart disease. Second, we assess the effects of these conditions on the probability of dying in the interwave period. Third , we develop a simple procedure to project forward the prevalence of poor early conditions and use the projected values to assess future prevalence of diabetes and heart disease. Fourth and finally, we estimate life expectancy and healthy life expectancy consistent with projected values of poor early childhood conditions.

In order to account for missing values as well as cases lost to attrition we use Multiple Imputation (MI) procedures and generate five alternative complete data bases from which we obtain estimates for the parameters of the models. The estimates are then averaged over the five complete, multiple imputed data sets, and appropriate adjusted standard errors are computed (Rubin, 1987). We used the program ICE implemented by STATA to perform all estimations (Schaeffer, 1997; Royston, 2004). We then use these estimates to project forward elderly mortality and health conditions and assess changes in life expectancy and healthy life expectancy at age 60.

1. Empirical estimates of the strength of the early-adult health connection

We estimate logistic models for the probability of experiencing diabetes and heart disease. These are the two most common chronic conditions among elderly in Latin America and the ones most commonly associated with conditions in early childhood. All models include controls for age, gender, and educational level.

2. Excess mortality associated with heart disease, diabetes and childhood conditions

We can assess mortality risks over two and four years in Mexico and Puerto Rico respectively. We do this by estimating logistic models including controls for age, sex, and education, a dummy variable for conditions experienced early in life and two dummies for the two self reported chronic conditions (diabetes and heart disease). The latter are evaluated at the time of the first wave. The coefficients of PEC, diabetes and heart diseases capture excess mortality associated with those conditions. Throughout we assume that mortality at older ages follows a Gompertz curve[11]. Thus, when age is entered as a continuous (independent) variable, the estimate of the constant of the logistic models is, to a close approximation, a transformation of the Gompertz constant. The estimated effect of age is simply an approximation of the Gompertz slope over age 60. Thus, the model we estimate for mortality is tantamount to a model where mortality determinants affect the Gompertz baseline rate, in our case the mortality rate at age 60. If Z is a 0/1 covariate with an estimated effect equal to β, $\exp(\beta)$ is, approximately, the ratio of the mortality risk of individuals with Z=1 to the mortality risk among those with Z=0 and β can be interpreted as a relative risk.

3. Future prevalence of early health conditions, diabetes, heart diseases and mortality

[11] Although we could have also fitted a log-logistic or logistic function, the Gompertz model is more convenient tool since the parameters of the log-odds model have a more natural interpretation. Alternative parameterizations of older mortality are inconsequential for our final inferences

Using the results of the logistic models, we project five years ahead the prevalence of PEC, diabetes, heart diseases and, finally, mortality. First, we estimate the prevalence of PEC five years before the surveys. This serves two purposes: to identify an age pattern of short-term change in the age-specific prevalence of PEC and to determine its rate of change over a five year period. We then use the estimated rate of change in the prevalence of PEC and apply it to the period five years after the second survey to determine the expected composition by PEC of the population five years after the second survey. Second, we determine the prevalence rates of diabetes and heart disease consistent with the distribution of the older population by PEC five years after the surveys. Third, we calculate expected mortality levels consistent with the projected prevalence of PEC, diabetes and heart disease five years after the second surveys.

(i) Rate of change of prevalence of PEC. To estimate rates of change of prevalence of PEC we first calculate the proportions who would have self-reported PEC five years before the first wave of the surveys. This is done by back-projecting five years four subgroups among those who self reported PEC in the first wave: those with diabetes (but no heart disease), those with heart disease but no diabetes, those with both diabetes and heart disease and, finally, those with neither condition. We assume that the four subgroups who self-report PEC are exposed to mortality risks governed by a Gompertz mortality function that accounts for the presence of diabetes and heart disease. The corresponding equations for the back-projection are as follows:

$$P_x^{PEC}(t-5) = \sum (P_{x+5}^{PEC,j}(t) \times (SR_x^T / SR_{x^{PEC,j}})) \qquad (1)$$

where $P_x^{PEC}(t-5)$ *and* $P_{x+5}^{PEC,j}(t)$ are respectively the prevalence of poor early childhood conditions at ages x at time t-5 and in each of the four subgroups j defined by presence/absence

of diabetes and heart disease at age x+5 at time t. The values of $SR_x^T \; and \; SR_x^{PEC}$ are defined as follows:

$$SR_x^T = \exp(-\int_{x-5}^{x} (\mu^T(y)dy)$$

$$SR_x^{PEC,j} = \exp(-\int_{x-5}^{x} (\mu^{PEC,j}(y)dy) \qquad (2)$$

where $\mu^{PEC,j}(y)$ and $\mu^T(y)$ stand for the force of mortality among those with poor early conditions and belonging to subgroup j and the total population respectively. $SR_x^T \; and \; SR_x^{PEC,j}$ are the estimated probability of surviving five years, from age *(x-5-1, x-5)* to age *(x,x+1)*, for the total population and among those who self-report poor childhood health status and belong to one of the four subgroups j defined before respectively. Once we have values of $P_x^{PEC}(t-5) \quad and \quad P_x^{PEC,j}(t)$ we calculate estimates of the rates of increase (decrease) of prevalence of PEC and can project them five years ahead.[12]

(ii) Projection of diabetes and heart disease five years ahead. The next step consists of estimating rates of prevalence of diabetes and heart disease consistent with the prevalence rates of PEC projected five years ahead. This can be done using estimates from the logistic models for self-reported diabetes and heart diseases. To the extent that these models capture the effects of PEC on the probability of self-reporting those conditions, they can be readily used to produce expected prevalence rates given arbitrary values for prevalence of PEC. In all our

[12] For each age group this rate is equal to the difference between the rate of grow of the total population and the rate of growth of the population with poor early childhood conditions.

calculations we assume that the composition of the population by characteristics other than those we are working with, for example gender or education, remain constant.

(iii) Mortality, life expectancy and healthy life expectancy five years ahead. We can use projected prevalence of PEC to determine expected prevalence of diabetes and heart disease and then the expected levels of mortality associated with these two diseases. If we add background morality rates we can calculate projected life expectancy and healthy life expectancy over age 60. To avoid unnecessary proliferation of scenarios, we assume throughout that expected (background) improvements in mortality apply to subpopulations affected neither by heart disease nor diabetes but that these two subpopulations experience the mortality estimated from our models and they are subject neither to improvements nor deterioration. More concretely, we assume that in the five years following the last survey the background force of mortality declines at a rate of about 0.5% per year, implying gains of life expectancy of (roughly) 0.046 years per year or a total of about 0.23 years over five years[13].

4. Alternative scenarios: estimating bounds of the effects of PEC

We are now in a position to estimate bounds for the effects that prevalence of poor early childhood condition among successive cohorts of elderly may have on future life expectancy and healthy life expectancy. We will define an array of scenarios depending on the prevalence of PEC and on the magnitudes of effects of PEC on the probabilities of contracting either diabetes or heart disease. Throughout we assume that the *direct effects* of PEC on mortality are nil, as if PEC effects could only be mediated by heart disease and diabetes. Finally we assume that the magnitude of effects of these diseases on mortality is the same as estimated from the interwave period

The total effect of PEC on projected life expectancy is a product of two factors. The first is the sheer prevalence of PEC. The second is the effect that PEC has on mortality. If one ignores the direct effect of PEC on mortality, what remains is a product of two pathways: the effects of PEC on the probability of occurrence of diabetes and heart disease and the effect of the chronic conditions on mortality. To define plausible scenarios we vary (a) the future prevalence of PEC and (b) the magnitude of the effects of PEC on prevalence of diabetes and heart disease. The prevalence of PEC and the magnitude of its effects on these two chronic conditions translate the influence that PEC has on mortality and health status and determine the 'room' there is for EC to play a role in future life expectancy and healthy life expectancy.[14] The scenarios are designed to identify lower and upper bounds for these effects thus providing a basis for assessing the role PEC can play in the future trajectory of mortality and health status.

We consider the following scenarios:

Scenario I: the prevalence of PEC five years into the future is identical to the observed one (rate of growth of PEC prevalence is 0). Within this scenario we include 12 different cases each defined by a combination of values of the effects of PEC on diabetes and heart disease. The first case sets the values of effects equal to those estimated; in the second through the fourth cases we double each time the effect of PEC on diabetes while keeping constant its effect on heart disease. The fifth through the ninth case repeats the operation but this time doubling both effects each time. Finally, the ninth through the twelfth case are identical to cases two to four but with the role of the effects of diabetes and heart disease reversed.

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[13] This is about half the gains assumed by the Unites Nations' projections for the same period (United Nations, 2002). This difference, however, does not affect our calculations since we are not using the United Nations projections as benchmarks.

[14] The direct effect of PEC on mortality is small and ignoring it does not alter our conclusions. The effects of PEC on chronic conditions other than diabetes and heart disease has not been properly documented so we ignored them in our calculations.

Scenario II: the prevalence of PEC in five years is increased assuming a rate of growth of .02 per year which is estimated by fitting a linear trend to the observed and backward projected values of PEC prevalence (see above)

For the purpose of contrast we add a third scenario:

Scenario III: prevalence of PEC=0

Scenarios I and II set lower and upper bounds defined by the prevalence of PEC. The lower bound may arguably be viewed as too high since a future decrease in the prevalence of PEC is also possible. Thus the lower bound may contain an upward bias and is corrected by the values associated with scenario III. The cases included in scenarios I and II are arguably stacked in favor of the conjecture we are trying to falsify: note that in the most exaggerated of these the effects of PEC on diabetes and on heart disease are sixteen times as high as estimated. This certainly allows for ample room to compensate for biases in the estimated effects of PEC due to measurement errors.

The indicators we use to characterize the scenarios are the projected value of life expectancy and healthy life expectancy. To calculate the latter we ignore the role played by other diseases and estimate the number of healthy years lost associated with heart disease and diabetes only. We use using Sullivan's method (Sullivan, 1971) and adjust the number of person-years lived by a cohort by the prevalence of the selected chronic diseases.

IV. RESULTS

1. Effect of PEC on the probability of experiencing diabetes and heart disease

Table 2 displays estimated effects of early conditions on the probability of self-reporting heart disease and diabetes in MHAS and PREHCO. Individuals who self-report poor health

status early in life are more likely to self report diabetes and heart disease in both Puerto Rico and Mexico. The estimates are properly signed, of modest magnitude but statistically significant. These estimates imply that in Puerto Rico an individual who self reports poor early health status is, on average, about 24% more likely to self report diabetes (0.31 versus 0.25) and 11% more likely to self-report heart diseases (0.21 versus 0.19). In Mexico the estimates translate into excess probabilities of diabetes and heart disease among those who experienced PEC: 13% (0.18 versus 0.16) and 20% (0.06 versus 0.05) respectively[15].

Table2 about here

2. Effect of chronic diseases on mortality

Table 3 shows the results of the model estimated for the probability of dying among elderly population in Mexico and Puerto Rico. The effects of diabetes and heart diseases swamp everything else. They are large and quite similar in the two countries, particularly those associated with diabetes. The annualized relative risks of dying among those self-reporting diabetes are twice as high as among those not reporting it in Puerto Rico (0.11 versus 0.06) and Mexico (0.12 versus 0.06). The annualized relative risks among those reporting heart disease are about 1.4 higher in Puerto Rico (0.10 versus 0.07) and 1.83 as high in Mexico (0.11 versus 0.06) than among those not reporting the condition. Note that, as stated in footnote 12, the *direct effects* of PEC (e.g. those operating through illnesses other than diabetes and heart disease) are very small and statistically insignificant.

Table 3 about here

3. Projected prevalence of poor early childhood status

[15]The contrast in the prevalence of heart disease between Mexico and Puerto Rico stems from the fact that whereas MHAS probed for heart attacks, PREHCO probed for heart disease in general. Our results are replicated, however, if we use the less inclusive definition for Puerto Rico or if we modify the definition to include other related diseases, such as hypertension and diseases of the circulatory system.

Figure 1 displays the estimated rates of change in the proportion of individuals reporting PEC by single age for Mexico (left side Figure 1) and Puerto Rico (right side Figure 1). To be absolutely consistent with the conjecture, these rates should be positive and larger for the youngest cohorts, that is, those that experience the brunt of medical improvements. In both countries the estimated rates of changes are somewhat erratic and, though positive, they do not display the expected age pattern. In Mexico the estimated rates of changes are, on average, positive and close to 0.02 (per year) over five years and they tend to increase sharply at very old ages. It is possible that the increase at older ages reflects a higher ratio of noise to signal as the number of cases in the sample drops substantially at ages older than 85. An interesting feature is that the rates tend to be highest for the cohorts born soon after the end of the Mexican revolution in 1918-1920 (those aged between 76 and 82 in the year 2000).

In Puerto Rico the rates are mostly positive, they also hover around a mean of about 0.02 (per year) over five years, and there is again a marked upward trend with age. Since the onset of mortality decline in Puerto Rico was earlier than in Mexico and was strongly associated with eradication of infectious and parasitic diseases (malaria, dengue, yellow fever), the increasing trajectory of the rates of PEC with age should not be surprising.

Figure 1 about here

It is clear from Figure 1 that there is no basis to choose a rate of increase in the prevalence of PEC outside the range (0, .02) for both countries.

4. Projected prevalence of chronic diseases five years ahead.

Figure 2 displays the proportionate difference in prevalence of diabetes (first panel) and heart diseases (second panel) between a baseline scenario that assumes no increases in the current prevalence of PEC and one that assumes that the prevalence of PEC will increase in the

next five years at the average rate of change 0.02. The increases in diabetes and heart disease that follow an increase in the prevalence of PEC are quite modest and they never surpass 2% on average. The increases are larger for diabetes in Puerto Rico and for heart disease in Mexico. The muted effects are a result of relatively low prevalence of PEC (about 0.38 in Mexico and 0.37 in Puerto Rico) and of the subdued effects of PEC on the probability of experiencing diabetes and heart diseases.

Figure 2 about here

5. Projected life expectancy and healthy life expectancy five years ahead

Figures 3a and 3b display projected life expectancy and healthy life expectancy at age 60 for Mexico and Figures 4a and 4b for Puerto Rico. The three lines represent the three scenarios defined before. Each case within a scenario is represented as a point in the pertinent line. Cases 1 through 3 keep fixed the effect of PEC on heart diseases but successively double the effect of PEC on diabetes until this is 2^3 as large as the estimated one. Cases 4 through 8 do the same but simultaneously doubling both effects. Case 8 uses effects of PEC on diabetes and heart diseases that are 2^4 as large as estimated. Finally, cases 9 through 11 are like cases 1 through 3 with the roles of diabetes and heart diseases reversed.

Since the results for Mexico and Puerto Rico are so similar, we will consider them together. The line for scenario 3 is flat since it assumes that the prevalence of PEC is 0; hence it should not vary with changes in the effects of PEC on the diseases. The *observed* values of life expectancy and healthy life expectancy correspond to case 1 in the line for scenario 2 (rate of growth of prevalence is 0) and the estimates of effects are the observed ones). Note how small are the changes implied by alternative assumptions about prevalence of PEC: the differences between the three lines in case 1 are imperceptible. This leads to the first inference: given the

estimated effects of PEC on the prevalence of diabetes, changes in prevalence of PEC will be inconsequential. Life expectancy and healthy life expectancy will proceed according to forces that are not affected by changes in the composition of the elderly by experience of early conditions.

To obtain relatively large declines in life expectancy exceeding, say, 10 percent of current values we need to point to combinations of effects of PEC on diabetes and heart disease that are implausibly large. Indeed, losses of ten percent or more are associated with cases 5-8 which require effects to be at least 2^3 as large as those estimated. Healthy life expectancy is slightly more sensitive to changes in the effects, though not to changes in the prevalence of PEC, as can be verified by comparing the values across the three scenarios for case 1. Losses in healthy life expectancy that exceed 10 percent are attained even with 'mild' increases in the effects of PEC on the prevalence of diabetes and heart disease. But these 'mild' increases (cases 2 and 3, 5 thru 8, and 9 thru 11) require at least one doubling of one of the effects, a change that may be deemed rather exaggerated even considering the possibility of substantial attenuation biases due to measurement errors in PEC.

Tables 3a and 3b

Tables 4a and 4b

V. DISCUSSION

The objective of this paper is to assess the influence that changes in composition of cohorts by type of early life experiences may have on future pathways of life expectancy and healthy life expectancy in Latin American Countries (LAC). Declines in mortality verified in the last 40 years in most LAC were largely associated with improvements in medical interventions

and less so with decreases improvements in living standards. Paradoxically, the conditions that led to lower child mortality rates in the past could threaten gains in future life expectancy and healthy life expectancies at old ages. Could it be the case that future changes in the composition of elderly cohorts by early health status will influence the trajectories of life expectancy and health life expectancy at older ages? And how large would these changes have to be to have a significant impact? Could these changes trump improvements heralded by better medical care, better access and even better prevention or will they add to the apparent burden imposed by adoption of deleterious behaviors?

The argument made at the outset is that the combination of the nature of mortality decline in the LAC region and the most likely mechanisms promoting an early-late health connection leads to the expectation of a negative within-cohort association between health and mortality during childhood and adulthood. If so, it is possible that future gains in life expectancy and healthy life expectancy could be compromised. The data available to us are not enough to generate compelling projections but they do permit the calculation of bounds for the effects of (a) changes in the composition of the elderly by early life experiences triggered by the nature of mortality decline during the post 1950 period, and (b) changes in the effects that early experiences might have on the prevalence of adult chronic illness, a result of mechanisms linking early and late health.

Estimates of lower and upper bounds for the effects of changing composition of elderly by early life experiences suggest two inferences. The first is that if effects of PEC on diabetes and heart disease, the two chronic conditions thought to be most sensitive to early life experiences, are as in Mexico and Puerto Rico, not even a large change in the composition of the elderly population could derail increases in longevity. This is simply a reflection of weak effects

of PEC on the likelihood of contracting diabetes or heart disease. Since these estimates may be substantially attenuated due to measurement error, one could argue that the previous inference is vitiated. To circumvent this we assess bounds associated with variation in the estimated effects. The resulting analysis lead to our second inference: changes in the composition by early experience could have more than trivial consequences on longevity and healthy life expectancy only under implausibly large effects of PEC on prevalence of diabetes and heart disease.

Admittedly the calculation of bounds does not allow us to produce accurate forecasts. But it is enough to infer that even if the early-late health connection is relevant and even though it could translate into negative within-cohort health and mortality correlations, the magnitude of effects is too small to make a significant dent on the future trajectory of life expectancy and healthy life expectancy in the LAC region.

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Table1. Description Variables in MHAS and PREHCO

Variables	MHAS				PREHCO			
	Mean	Missing (%)			Mean	Missing (%)		
		Total	No Proxy	With Proxy		Total	No Proxy	With Proxy
Death	0.06	0.1	0.1	0	0.16	0	0	0
Age	69	0	0	0	72	0	0	0
Sex (female)	0.53	0	0	0	0.60	0	0	0
Poor Early Conditions	0.38	13.4	6.0	100	0.37	20.0	8.3	100
Short Knee height	0.3	3.1	3.1	3.3	0.3	9.4	4.2	44.9
Polio [a]	0.003	9.4	1.7	100	0.004	13.0	0.3	100
Rheumatic Fever [a]	0.014	9.5	1.8	100	0.023	13.6	1.0	100
Tuberculosis [a]	0.007	9.4	1.7	100	0.008	13.0	0.2	100
Poor general Health [a]	0.11	9.9	2.3	100	0.072	15.8	3.4	100
Heart Diseases [b]	0.044	3.8	3.6	5.7	0.19	0.7	0.5	2.2
Diabetes	0.17	3.7	3.6	4.8	0.28	0.2	0.2	0.3
0 yrs school.	0.33	1.1	1.0	1.4	-	2.1	1.4	6.7
1-5 yrs school.	0.38	1.1	1.0	1.4	0.31	2.1	1.4	6.7
6 yrs school.	0.15	1.1	1.0	1.4	0.08	2.1	1.4	6.7
7+ yrs school.	0.14	1.1	1.0	1.4	0.55	2.1	1.4	6.7
Proxy interview	0.08				0.12			
Total Observations (sample)	*7,604*				*5,286*			

Source: From author calculations of MHAS (2001-2003) and PREHCO (2000-2006) data.

[a] In both MHAS and PREHCO, individuals who needed proxy to answer the questionnaire were not asked to answer the questions about the self-reported conditions experienced early in life.

[b] In MHAS, the only variable available to define heart diseases is if individuals suffered from heart attack. For PREHCO, this variable includes more general heart conditions (e.g. angina, coronary disease, and others cardiac problems).

Table 2. Estimates of Logistic Models Predicting Self-Reported Diabetes and Heart Disease in MHAS and PREHCO

Independent Variables	Mexico				Puerto Rico			
	Diabetes		Heart Diseases [(a)]		Diabetes		Heart Diseases [(a)]	
65-69 yrs old	0.12		0.31	†	0.04		0.27	*
70-74 yrs old	0.04		0.52	**	0.12		0.39	***
75-79 yrs old	-0.15		0.68	***	-0.06		0.49	***
80+ yrs old	-0.18		0.82	***	-0.31	***	0.50	***
Ref. cat.: 60-64								
Female	0.32	***	-0.37	**	0.04		-0.04	
PEC	0.16	*	0.29	*	0.30	***	0.14	
1-6 yrs school.	0.09		0.05		-0.05		-0.07	
6 yrs school.	0.17	†	0.40	*	-0.27	†	0.03	
7+ yrs school.	0.05		0.22		-0.18	†	-0.37	***
Ref. Cat.: 0 yrs school.								
Constant	-1.87	***	-3.51	***	-0.89	***	-1.57	***
Number Obs.	*7,604*		*7,604*		*5,286*		*5,286*	

Source: Estimated from MHAS (2001) and PREHCO (2000) data.

Notes:

(1) Estimates were obtained using the entire data sets after performing multiple imputation to include missing data (Schafer 1997).

[a] In MHAS, the only variable available to define heart diseases is if individuals suffered from heart attack. For PREHCO, this variable includes more general heart conditions (e.g. angina, coronary disease, and others cardiac problems).

† $p<.10$; * $p<.05$; ** $p<.01$; *** $p<.001$

Table 3. Estimates of logistic models predicting mortality in MHAS and PREHCO

Independent Variables	Mexico		Puerto Rico	
Age *minus* 60	0.09	***	0.10	***
Female	-0.24	*	-0.50	***
Poor early health conditions	-0.12		0.13	
Heart diseases [(a)]	0.63	***	0.39	***
Diabetes	0.70	***	0.57	***
1-6 yrs school.	-0.17		0.23	†
6 yrs school.	-0.19		0.15	
7+ yrs. school	-0.09		-0.12	
(ref. cat.: 0 yr)				
Constant	-3.63	***	-3.18	***
Number Obs.	*7,604*		*5,286*	

Source: From author calculations of MHAS (2001-2003) and PREHCO (2000-2006) data.

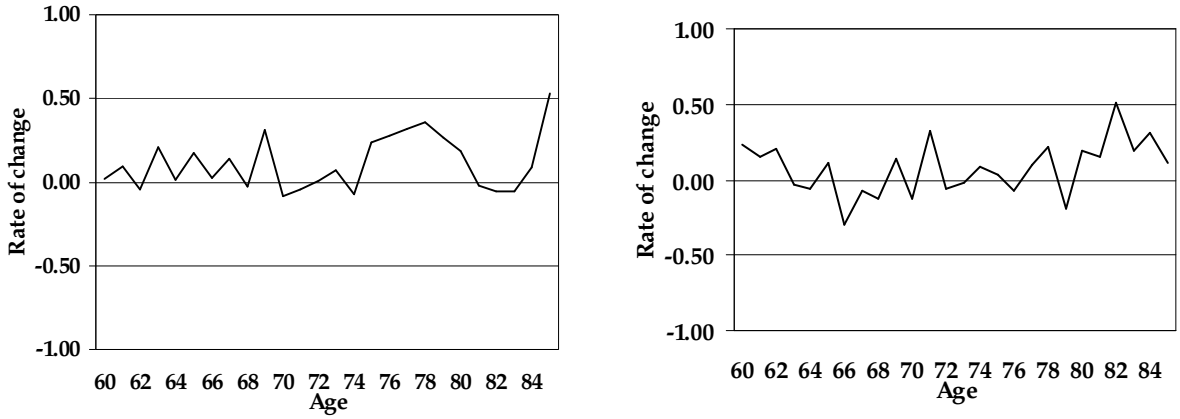
Notes:

(1) Estimates were obtained using the entire data sets after performing multiple imputation to include missing data (Schafer 1997).

[a] In MHAS, the only variable available to define heart diseases is if individuals suffered from heart attack. For PREHCO, this variable includes more general heart conditions (e.g. angina, coronary disease, and others cardiac problems).

† $p<.10$; * $p<.05$; ** $p<.01$; *** $p<.001$

Figure 1. Five Years Age-Specific Rates of Change in the Proportion of Poor Early Health Conditions Among Elderly in Mexico and Puerto Rico



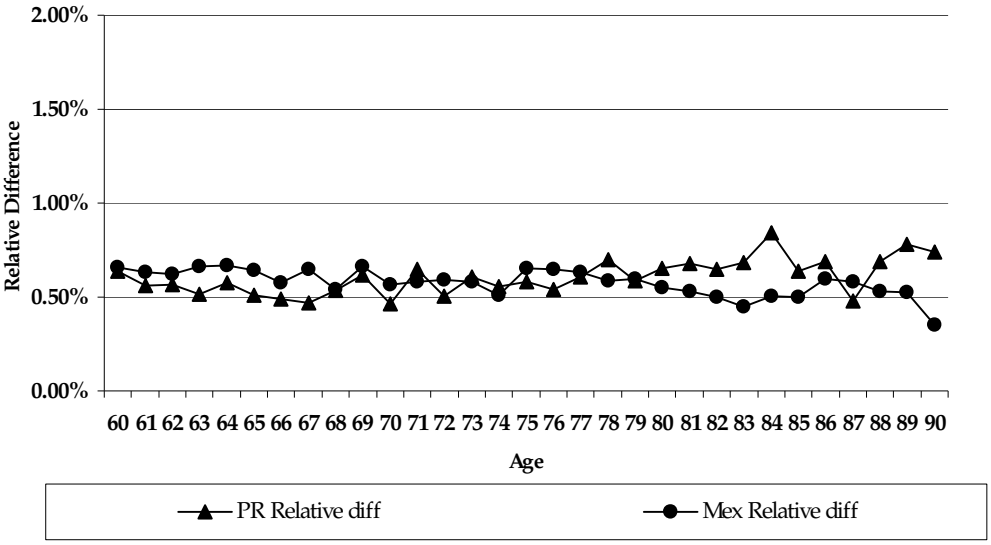
a. Mexico **b. Puerto Rico**

Source: MHAS (2001-2003); PREHCO (2000-2006).

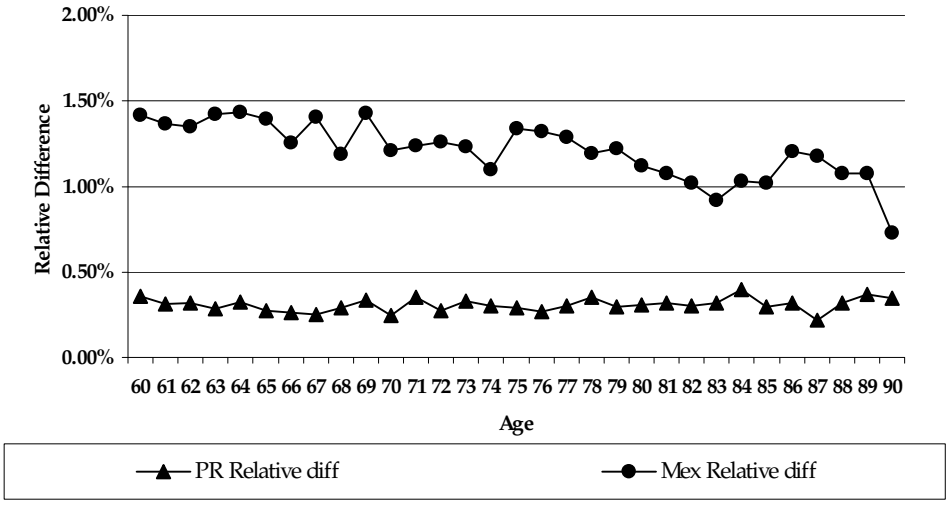
Note:

The weighted average rate of change in the probability of experiencing poor early conditions over five years in Mexico and Puerto Rico is 0.12 and 0.08 respectively.

Figure 2. Relative Difference Between Counter-Factual and Observed Prevalence of Diabetes and Heart Diseases Among Elderly in Mexico and Puerto Rico



i. Diabetes



ii. Heart Diseases

Source: MHAS (2001-2003); PREHCO (2000-2006)

Note: *Counter-factual* prevalence of chronic diseases is defined by assuming increases in the prevalence of poor early conditions at the weighted average 5-years rate of change in the probability of experiencing poor early conditions. In Mexico and Puerto Rico, the 5-years rate of change is 0.12 and 0.08 respectively.

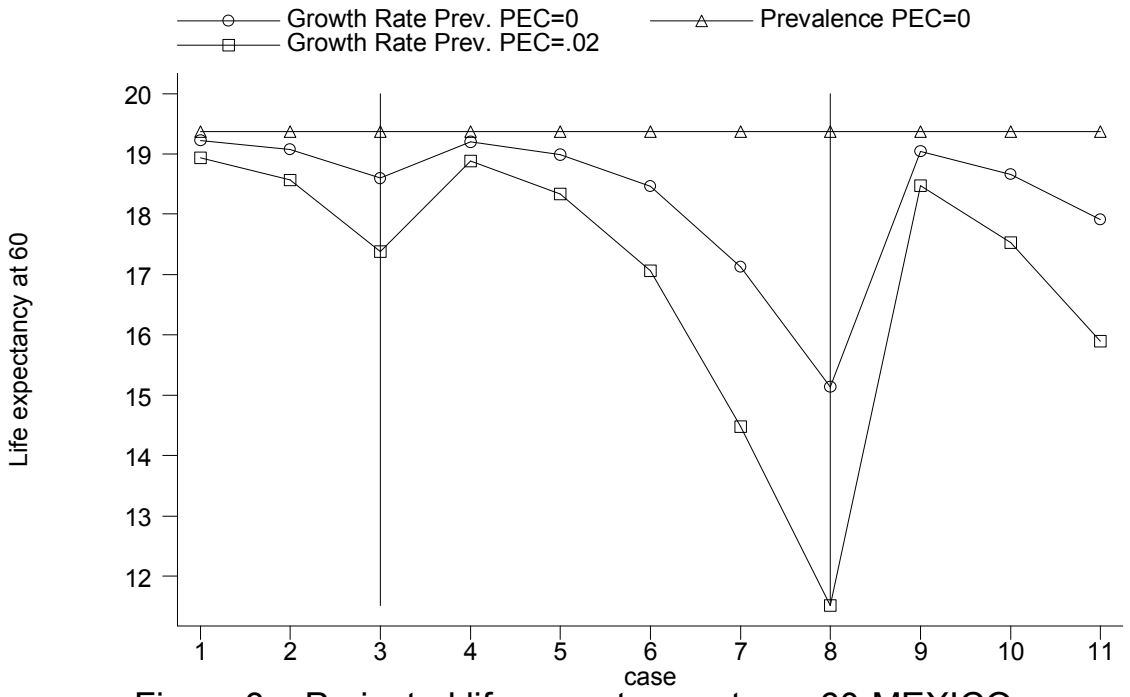


Figure 3a: Projected life expectancy at age 60-MEXICO

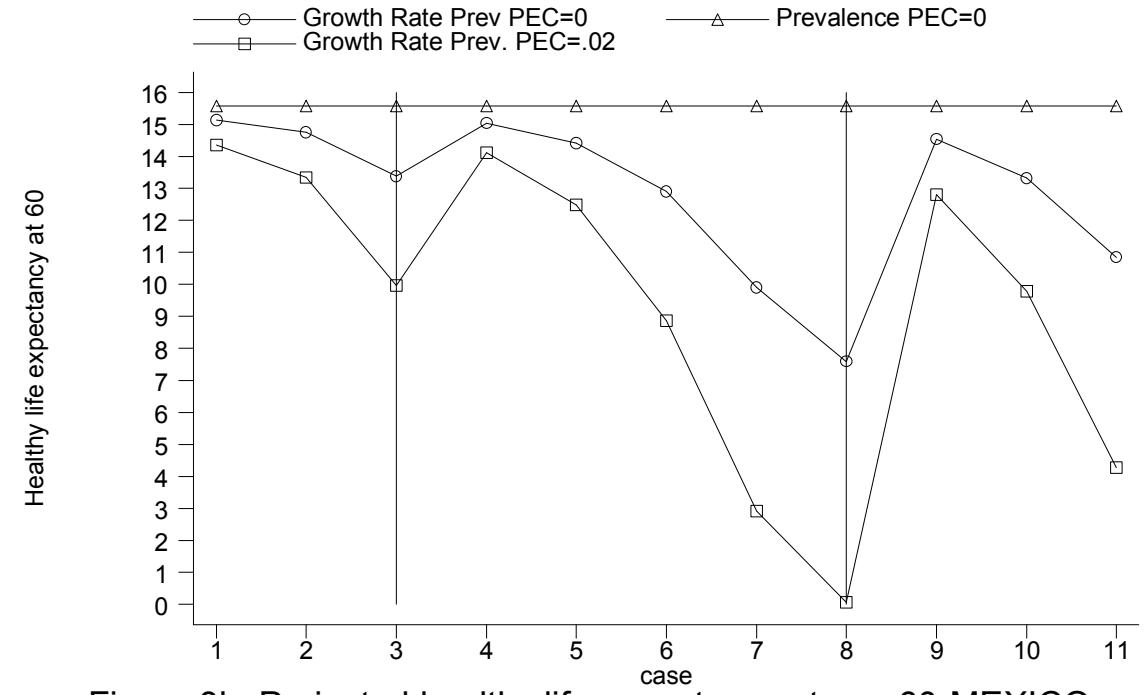


Figure 3b: Projected healthy life expectancy at age 60-MEXICO

*

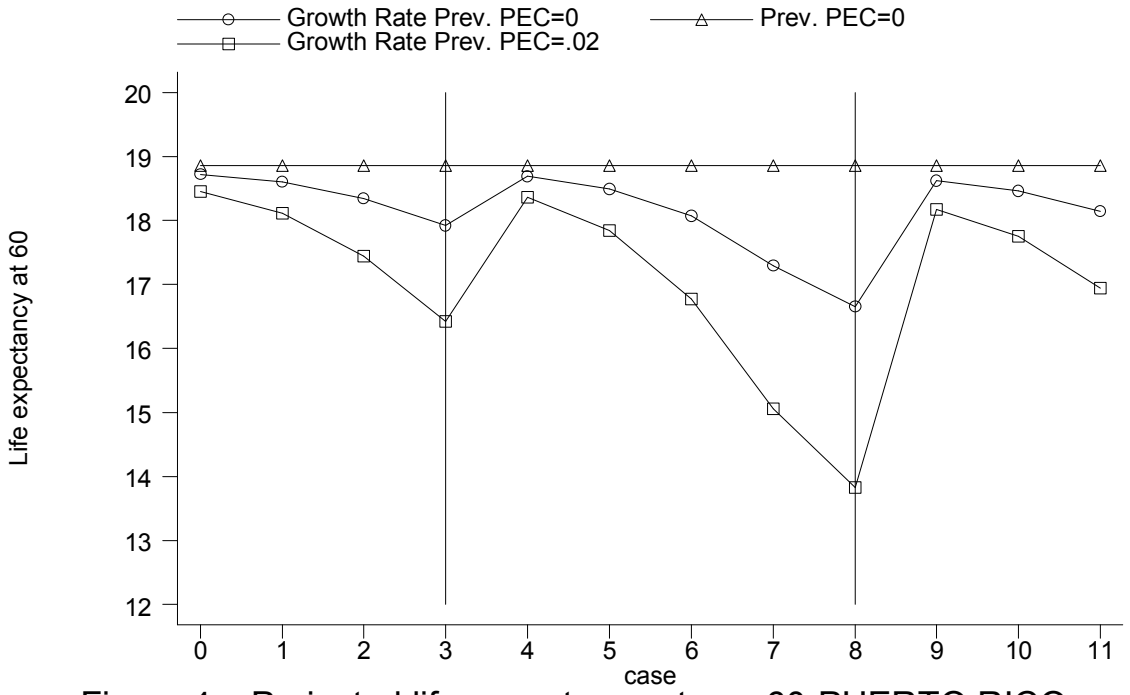


Figure 4a: Projected life expectancy at age 60-PUERTO RICO

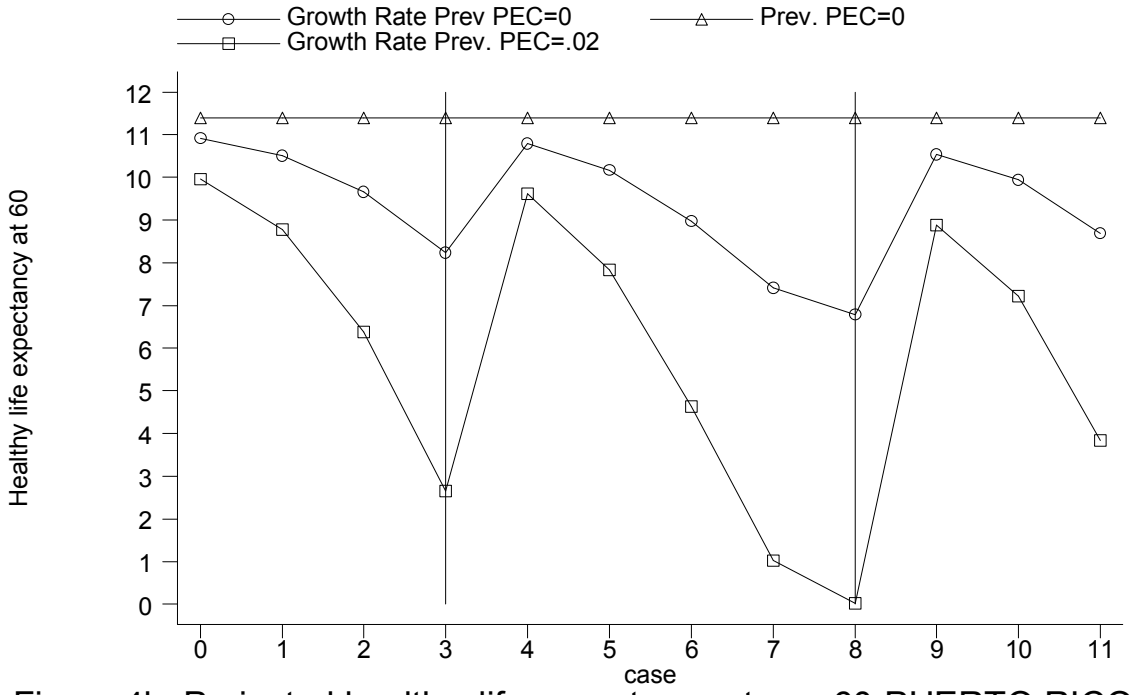


Figure 4b: Projected healthy life expectancy at age 60-PUERTO RICO