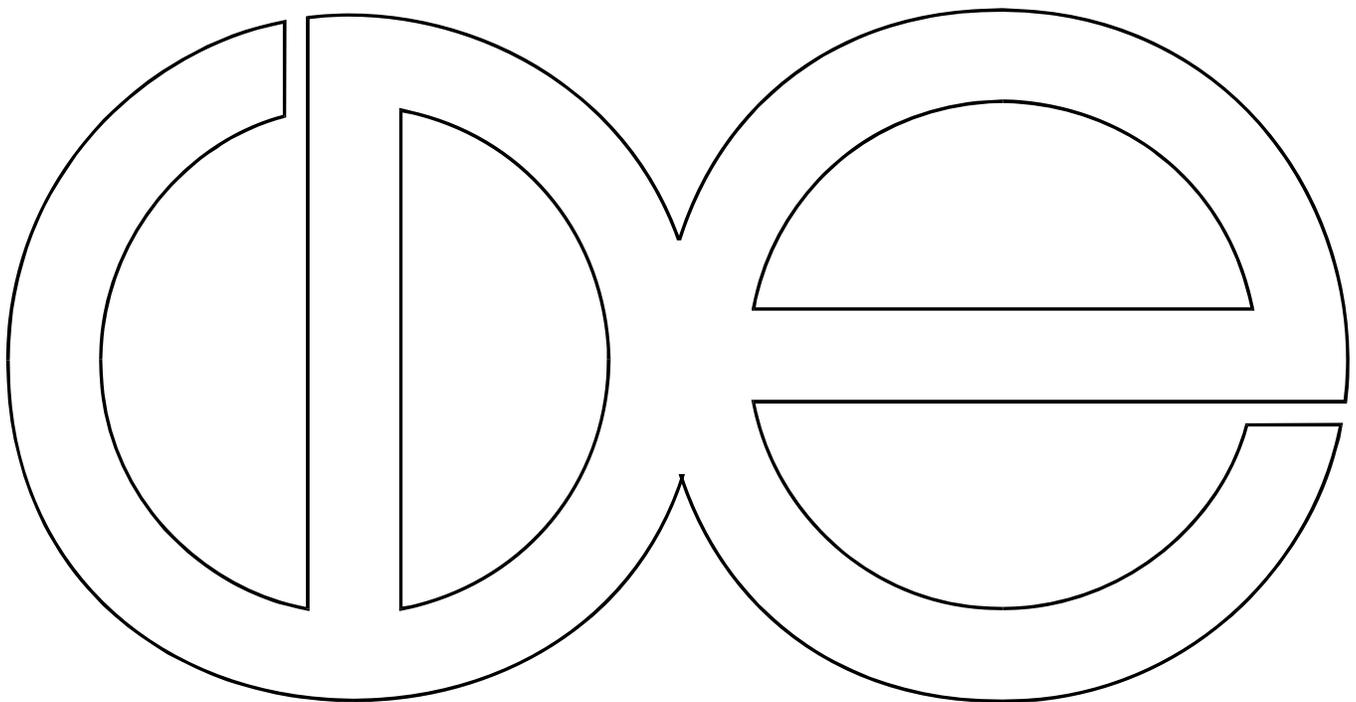# Center for Demography and Ecology

## University of Wisconsin-Madison

# Roadblocks for sustained improvements in
# life expectancy in Latin America and the Caribbean

**Alberto Palloni**
**Kenya Noronha**
**Mary McEniry**

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Authors:

**Alberto Palloni**

Affiliation: Northwestern University, Institute for Policy Research

Title: Board of Trustees Professor in Sociology, Northwestern University
Faculty Fellow, Institute for Policy Research

**Kenya Noronha**

Affiliation: Northwestern University, Institute for Policy Research and University of Wisconsin-Madison, Center for Demography and Ecology

Title: Postdoctoral Fellow

**Mary McEniry**

Affiliation: University of Wisconsin-Madison, Center for Demography and Ecology

Title: Associate Scientist

**Corresponding Author**: Alberto Palloni

Address: Institute for Policy Research

2040 Sheridan Rd. - Evanston, IL 60208-4100

e-mail: a-palloni@northwestern.edu

Telephone number: 847-491-3395     Fax number: 847-491-9916

# Roadblocks for sustained improvements in life expectancy in Latin America and the Caribbean [*]

**Abstract**: Future life expectancy in Latin America and the Caribbean could be compromised. Older people attaining age 60 after the years 1990-2000 are scarred by experiences that could translate into higher susceptibility to chronic conditions and higher mortality. Once dismissed as unthinkable, increases in mortality made their debut in modern societies, massively in Sub-Saharan Africa with HIV/AIDS and suddenly in the former Soviet Republics. We argue that the route to further increases in life expectancy in LAC is littered with obstacles. However, unlike Sub-Saharan Africa and the former Soviet Republics, changes in longevity in LAC may become driven neither by sudden overhaul of political regimes nor by the emergence of new diseases but, paradoxically, by conditions rooted in the unprecedented improvements in longevity that took place more than a half century ago. To support this view we assess empirical conditions characterizing two LAC countries, Mexico and Puerto Rico.

**I. INTRODUCTION**

By all accounts Western European countries experienced remarkably sustained decreases of mortality rates at adult ages during the post World War II. The estimated rate of decline of the force of mortality at older ages in the post 1950 period is approximately 1% per year (Kannisto 1994; Kannisto et al. 1994; National Research Council 2000). If maintained over a span of fifty years in mortality regimes with a life expectancy at age 60 between 15 and 20 years, as is the case in the bulk of countries in Latin American and the Caribbean (LAC), a 1% per year decline in mortality at all ages above 60 yields, on average, gains in longevity of the order of 0.10 years per year. Optimism regarding the potential for sustained increases in life expectancy is boosted by studies that reveal that since early in the century life expectancy has been increasing almost linearly worldwide, systematically attaining and surpassing ceilings optimistically forecasted in the past (Oeppen and Vaupel 2002). With notable exceptions, the sustained extension of life in most world regions has and 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 already taking place even at very old ages (Horiuchi and Wilmoth 1998; Wilmoth and Horiuchi 1999; Wilmoth, 1998).

The optimistic view is not without its detractors who more gloomily point out that lifestyle changes embraced by newer cohorts of elderly people in high or low income countries could oppose some resistance to further improvements in longevity (Olshansky et al. 2005; Preston 2005). But the optimistic scenario is remarkably consistent with recent evidence from adjusted data in Latin America (Palloni and Pinto 2004). This evidence shows that in the most advanced countries of LAC life expectancy at age 60 increased from about 18 years in 1950 to about 23 in 1995 in approximately *linear fashion*, yielding average yearly gains of 0.10 years per

year, fairly close to the rhythm of change experienced in Western Europe after 1950, albeit at lower levels of life expectancy at age 60. Such rates of increase imply that the rates of change of mortality risks are declining and must have attained values exceeding 1% per year at some point during the distant past and then dropped below 1% more recently.[1] This is *prima facie* evidence that while the early mortality decline in LAC is a feat of exceptional nature, it has weakened and encountered obstacles that constrain its continued reproduction.

In this paper we examine the recent experience of two LAC countries, Mexico and Puerto Rico for which we have very rich data and argue that the changing composition of cohorts of elderly people by health conditions experienced early in life may explain part of the slow-down of the rate of decrease in the force of mortality at ages over 60. According to the intermediate United Nations (2006) 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 assuming that mortality rates at ages above 60 will decline at an average rate somewhat below 1% per year. In the case of Puerto 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. We argue that these projections may be excessively optimistic. We find some evidence suggesting that changing composition of cohorts by early health status offers non-trivial resistance to continued gains in life expectancy and healthy life expectancy at older ages. We also suggest that while the resistance embedded in the

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[1] This inference rests on the simplifying assumption that the rate of decline of mortality risks is age invariant over age 60. This may not reflect exactly the actual course of mortality decline, but it is probably close to the actual experience during the last twenty years or so.

changing composition of cohorts by early health status is not enough by itself to halt altogether all future improvements, it could be strong enough to decelerate somewhat the mortality decline at older ages. Ours is a 'soft' contrarian view that calls for caution in projections of life expectancy at ages older than 60 and tempers the more accepted creed of unfettered improvement at older ages.

The paper is organized as follows. In Section II we formulate the core of our argument. First, we invoke findings supporting the existence of mechanisms linking early and late adult health, a phenomenon we refer to as the early-adult 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, spell out the implications they have for gains in longevity among older cohorts, and formulate a contrarian argument. In Section III we describe the data sets for Mexico and Puerto Rico and discuss estimation procedures. Section IV presents results and Section V draws implications and concludes.

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

In this section we first summarize evidence in support of the existence of an early-late health connection and then argue that the precise empirical expression of such a connection is a function of the regime of mortality decline.

### A. Linkages between early childhood and adult health status

Rapidly accumulating knowledge in 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 very specific 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. We claim that a partial escape from this identification problem can be secured by understanding the macro forces that drive mortality changes. Even though the bulk of the impact of these macro forces may be confined to early childhood, they may, under some conditions, allow improvements (deterioration) of mortality to spread across the entire lifespan of cohorts rather than just acting simultaneously over multiple cohorts in a contracted period of time.[2]

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[2] There is dense literature contending that part of the secular improvements in mortality operated through changes that affected entire cohorts. This view is in contraposition with the idea that mortality improvements are dominated

Accepting the existence of early-adult health connections inevitably leads to a conjecture regarding the nature of mortality changes at older ages: to the extent that successive birth cohorts are exposed to changing regimes of morbidity and mortality early on in their life, it must be the case that their morbidity and mortality risks at older ages are, to some degree at least, dependent on the conditions set forth by their experiences early on in their life. If so, there is the potential for a strong within-cohort association between mortality and morbidity at early and at older ages. We argue below that the magnitude and direction of such an association is strongly dependent on the dominant regime of secular mortality decline. It is the nature of this regime that opens or closes the room for the expression of early-late health connections. Each regime has implications for the subsequent survival of cohorts' members who are either exposed, scarred, immune or escape altogether from conditions experienced during early childhood.

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

Proponents of theories that associate mortality changes at older ages with changes in cohorts' health experiences during early childhood have perhaps too quickly suggested that cohorts that benefit from mortality reductions attributable to nutritional improvements or to a reduced rate of exposure/contraction of infectious and parasitic diseases will, *ceteris paribus*, experience lower morbidity and mortality risks at older age (Crimmins and Finch 2006; Finch and Crimmins 2004). We argue that this may occur *only if some strict conditions pertaining to the nature of macro forces promoting improvement in early child mortality prevail*. Even if it were validated, and this is far from being the case, the hypothesis about the early-adult health connection itself does not always imply positively correlated changes at young and older ages within cohorts. The fact that a positive correlation has been observed in some of today's high

by sequences of period-changes, e.g. affecting all cohorts at the same time (Barbi and Vaupel 2005).

income countries is a result of peculiar historical features that could have turned out quite differently had the initial conditions precipitating mortality decline been different. To understand how the early-adult health linkage could be manifested under different regimes of mortality decline one must spell out in detail the nature of the regime and identify which of the alternative mechanisms producing the early-adult health connection, be it faulty intrauterine growth, inflammation processes, stress or poor early nutritional status, is more likely to be expressed. Proper inferences about the expected associations between mortality risks over the life cycle across cohorts require identification of *the macro forces that generate the secular mortality decline.*

### 1. Secular mortality decline and the expression of early-adult health connections

Leaving aside accidents and violence, changes in mortality risks at any age can be produced by exogenous forces acting through one or more of the following pathways: *exposure to risk of illness,* rate of *contraction of illness,* and their *lethality rate* (Johansson and Mosk 1987).[3] The root causes of secular mortality declines can be classified into four 'pure' types, each of which assigns primacy to one or a combination of the above mentioned pathways: (i) Causes dominated by rising standards of living and, particularly, by improved nutritional status due to better quantity and quality of nutrients. Increasing the supply of nutrients leads, *ceteris paribus,* to increased nutritional status. Because of the connection between resistance (immune function), recovery rates (intensity and duration of episodes of illness) and nutritional status (Scrimshaw 1997; Scrimshaw and SanGiovanni 1997; Scrimshaw et al. 1968),

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[3] Infant and child mortality improvements driven by fertility reductions should be classified as outcomes of forces that reshape resistance and recovery. However, to the extent that fertility reduction eventually leads to reduction on intrahousehold crowding, it can also promote reduction in exposure.

improvements in the quantity/quality of food supply virtually always translate into a reduction of disease contraction and lethality rates even if the size of exposure remains invariant.

(ii) Causes dominated by public health interventions and/or diffusion of knowledge that trigger *reduction of exposure*. Malaria eradication, improved water supplies and sewage, and dissemination of knowledge about basic hygiene (McKeown 1976; Preston and Haines 1991; Preston and van de Walle 1978; Szreter 1988) are all items belonging to this class of causes and they all act to decrease exposure to illnesses. Once exposure is reduced, individuals benefit by spill-over effects: a reduction of exposure loads generates surplus energy that promotes the physiology of growth and development. Thus, reduced exposure also translates into improved nutritional status even if the supply of nutrients remains invariant. As a consequence, part of the lagged effects of this type of mortality decline regime is *indistinguishable from the effects induced by improved nutrition.*

(iii) Causes dominated by the application of chemotherapy. By definition these act on lethality rates and on the duration and intensity of episodes of illness. The introduction of sulfa drugs and antibiotics is the classic example of a new regime of mortality decline shaped by the introduction of chemotherapy. But also in these cases there are substantial spill-over effects. To the extent that individuals are exposed to constant external conditions and constant rates of contraction but to shorter and/or less intense spells of illnesses, nutritional status could improve even if the supply of nutrients remains invariant. Perhaps the synergisms in this case are somewhat weaker than those identified before, but they surely exist. Thus, as before, a mortality decline driven by diffusion of chemotherapy will produce lagged effects that cannot be neatly separated from those that would be observed had the main force driving mortality decline been increased standards of living or improved nutrition.

(iv) Causes dominated by blocking the rate of contraction or infectivity of illnesses. These refer to implementation of vaccination or inoculation, both interventions that boost the immune system so that even though exposure and contraction rates remain fixed, the consequences of episodes of illness are milder or avoided altogether. Once again, we should expect some synergism between (lack of) infection and nutritional status and, consequently we should expect lagged effects as a consequence of the improved nutritional status in the aftermath of widespread application of vaccination.

Thus, the last two set of causes produce outcomes that involve weak synergisms between nutritional status and infections whereas the first two induce strong synergisms. As a consequence, identification of the early-adult health connection that may be expressed under a regime of mortality decline is more difficult if the mortality regime is dominated by either of the first two set of causes than if it is a result of the last two.

Even a rough identification of the exact class of causes that produce sustained mortality declines in any particular case is a rare luxury in historical demography. And when it is possible to identify one or a group of forces, the interrelations described before complicate immensely the task of establishing precise attribution of causes. We know, for example, that the secular mortality decline in Western Europe began in earnest sometime during the middle of the nineteenth century. We also know that it is most likely to have been driven by causes (i) and (ii) since, given the state of medical knowledge and technology at the time, neither (iii) nor (iv) seem plausible (Evans 2008; Fogel 2004; McKeown 1976; Szreter 1988; Szreter 2002). But though we are almost certain of this, it is unlikely that we will ever know with precision the exact role played by improved nutritional status (enhancing resistance and recovery) and by the revolution in public health or knowledge dissemination (reducing exposure). Due to the synergism between

the most prevalent infectious diseases and nutritional status at the time, it is simply hopeless to cleanly separate the roles of increased resistance and reduced exposure. These forces do not operate separately from each other except in the mind of scientists who study them.

Even in a rosy scenario, one where the root causes of mortality decline could be exactly identified, we would still need additional evidence to make a claim about the expected early-adult health mechanism that is expressed and, therefore, to decide on the expected association between mortality risks at different stages in the life cycle of a cohort. Consider, for example, the case where mortality decline is fueled solely by improved nutritional status among young adults only. In this case there will also be amelioration of intrauterine growth over successive generations of mothers. Under these conditions the early-late health mechanism one would expect to see expressed could link intrauterine growth and adult ischemic heart disease or diabetes II. In this admittedly simplified case we will observe declining early adult mortality and, subsequently, declining early neonatal mortality through more favorable placental growth, gestational length and birthweight distributions. We would first observe a near zero correlation between changes in early adult mortality and older mortality followed by a closer association between changes in very early and older mortality for subsequent cohorts. And this association generated by the early-late health mechanism is a direct consequence first and foremost of increased nutrition among young adults that trickled down to infants.

Consider a second example. Suppose that nutritional improvements are confined to infants and children (by, say, more universal or longer breastfeeding). To the extent that the early-adult health connection involves post-neonatal nutritional status, these improvements in early survival will be translated into adult mortality changes when the target cohort ages. In this case we should observe a positive correlation between changes in early mortality and changes in

older mortality across cohorts. The age pattern of mortality changes through which the early-adult health linkage is allowed to be expressed in this example is quite different from the one that would be observed in the previous example.

Assume now that the driving mechanisms of the early-adult health linkage are inflammatory processes. Clinical, pathological and epidemiological evidence linking adult coronary artery disease (CAD) and inflammation caused by infection with *Chlamydia Pneumonia* is very strong (Fong 2000) . Indeed, it and the connection between CAD and periodontal disease (*Periodontitis*) are the *only* connections with some consequences for the within-cohort association between mortality rates at different ages that have a plausible biological mechanism and for which there is acceptable, albeit not water-tight, epidemiological, pathological and clinical evidence. Both infections are known to be more prevalent among adolescent and younger adults than among children (Fong 2000). Further, recent evidence from a developing country suggests that increased exposure to infectious diseases may actually be *protective* as its inflammatory response is reduced during adolescence and young adulthood among those with earlier higher exposure (McDade et al. 2008). It follows that if the causes of mortality decline alter the prevalence of these infectious diseases, one should not expect more than weak within-cohort association between *changes in early child mortality* and *changes in adult mortality.* We should expect either a positive correlation between *changes in adolescent and young adult mortality* and *changes at older age or, alternatively, a negative correlation between early exposure and adult mortality*. Thus, arguments about the role of inflammatory processes (Crimmins and Finch 2006; Finch and Crimmins 2004) have overstated the potential role of inflammation as root cause of subsequent changes in adult mortality.[4]

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[4] The proponents of this argument also overestimate the strength of the relation by focusing on the correlation between *absolute* levels of mortality rather than *relative change* of mortality at different points in the life span.

Three important conclusions follow. First, the dominant regime of mortality decline constrains the combination of early-late adult health mechanisms that could be expressed over the life time of successive cohorts. Second, because of spill-over effects and strong synergisms between infection and nutritional status, different mortality regimes can express virtually the same early-late health connection. It is thus not clear that one can find an easy avenue to attribute, for example, separate effects that induce adult mortality decline to reduction in inflammation, on the one hand, and increased nutritional status, on the other. Third, the simple within-cohort correlation between changes in mortality at early and late adult ages is not enough to identify the expression of any one of several early-late adult health connections. While we could hope to see palpable evidence of a relation in the form of correlations between mortality changes at several ages, it is somewhat hazardous to make inferences about the precise source(s) that produce such correlations even in the ideal case where we are able to identify the root causes triggering mortality decline.

## 2. An alternative conjecture for Latin America and the Caribbean

Contrast the aforementioned historical conditions with the experience of Latin American and the Caribbean (LAC) region and, more widely, in most developing countries. It is known that the mortality decline that took place in low income countries after 1940 or 1950 was heavily influenced by forces (iii) and (iv) (Palloni 1990; Arriaga and Davis 1969). Cause (ii) was implicated but in different degrees according to country and at different times. Cause (i) played a minor role, at least initially. Although there is substantial variability in the time of onset, most countries in the LAC region began an uninterrupted and sharp mortality decline sometime 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.

Before 1940 the largest fraction of the decline is associated with decreases in mortality before age five. After 1950 there is a clear acceleration of the rate of mortality decline that coincides with the period when chemotherapy makes its debut in the area and begins to be widely used. Empirical investigations show that the bulk of this decline before 1940's is associated with the deployment of public health tactics and medical technology that diminished exposure to infectious and parasitic disease 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.

Because of synergisms, the fact that we can isolate with some precision the exogenous forces that launched the secular mortality decline in the region does not by itself entitle us to make inferences about the relative importance of nutritional improvements and inflammatory processes. Furthermore, in this historical case there is an added element to take into account: if the bulk of mortality decline is attributable to the deployment of chemotherapy, for example, it does not follow at all that cohorts that gained survival advantages will be comprised by individuals who, on average, either experienced less exposure or better nutritional status. Many countries in the region experienced sharp mortality declines due mostly, though not completely, to reductions in infant and child mortality. This mortality decline is largely associated with the deployment of medical technology rather than with improvements in living conditions or nutritional standards. While there might be secondary nutritional benefits to the medical improvements—in the form of weak synergism— the composition of cohorts that benefited from

these mortality improvements will be increasingly biased toward members who are more likely to express one of the early-late health mechanisms that depend on poor early nutrition.

Consider now the following empirical regularity: 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 just described, 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 the elderly will belong to birth cohorts whose members survived infectious and parasitic diseases that prior to the onset of 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 if the early-adult health conjecture is valid, then the prevalence of adult chronic illnesses ought to increase over time.[5] And therein lies the rub: *under these conditions life expectancy (as well as healthy life expectancy) at older ages may increase more slowly or cease to increase altogether even if 'background' mortality (i.e. mortality unrelated to the target chronic conditions) continues to decline.*

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[5] 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 cardiovascular diseases.

Two conjectures follow immediately: (a) the association between changes in early childhood and in old age mortality across cohorts could drift to zero or become negative *if no other forces are operating* and (b) future levels of mortality among elderly could decline more slowly, cease to decline or even increase.

Two conditions can preclude the latter fate: (a) decreases in the 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. If neither (a) nor (b) occur, older people in cohorts that experience this type of mortality decline could be exposed to higher mortality levels or higher morbidity than older people in preceding cohorts.

In what follows we explore data that can be brought to bear on the second conjecture. We use information for two countries in LAC, Mexico and Puerto Rico, for which we have robust 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 not to pose a strong version of a contrarian argument according to which there are unambiguous signs that progress in longevity will run out of steam in the short run. Rather, we espouse a weak version of the argument, one that insists on a case-by-case probing of empirical conditions that could constrain and oppose resistance to uninterrupted gains in longevity.

## 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), both 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

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.[6] The sample is a multistage stratified sample with over-sample of regions heavily

populated by individuals of African descent and 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.[7]

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[6] 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.

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

MHAS is a nationally representative, prospective panel study of Mexicans aged 50 and over in 2000 funded by the National Institute on Aging. Baseline interviews were completed in the summer of 2001 with about 15,000 respondents, including targets and spouses. The individual non-response rate of 10.5% for a population-based survey is very low. The second 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 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 available for public use.[8]

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 about here

**B. Measurement of poor early childhood health conditions**

We start with an important disclaimer: the surveys we use are not exactly suitable to retrieve measures or 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

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

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 very broad definition that considers the largest number of conditions experienced early in life. It attains a value of one if at least one of 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

In what follows we discuss the models used to estimate the probability of experiencing two major chronic diseases (heart disease and diabetes) and for the probability of dying. Both diabetes and heart diseases are self-reported conditions and are thus subject to misreporting. 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 databases 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 (Schafer 1997). 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. We also control for obesity[9] as this is a risk factor for both diabetes and heart disease. To the extent that we do so, we are bound to retrieve only net effects of PEC, not its gross effects, e.g. including the effects operating through obesity. Thus, our results should underestimate the total (gross) effects of PEC and will underplay the tug towards lower rate of gains in life expectancy.

### 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). These self-reported conditions are evaluated at the time of the first wave. The coefficients of PEC, diabetes and heart disease capture excess mortality associated with those conditions. Throughout we assume that mortality at older ages follows a Gompertz curve.[10] 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

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[9] Obesity is determined by calculating BMI (weight/height squared). In the case of PREHCo both weight and height were obtained via anthropometric measurement. In MHAS weight and height are self-reported. An individual is classified as obese if BMI exceeds the 60th percentile of the BMI country-sex specific BMI distribution

[10] Although one could have used a log-logistic or logistic function to fit the force of mortality at ages above 60, the Gompertz model is a more convenient tool since the parameters of the log-odds model have a more natural interpretation. We did not experiment with alternative parameterizations of older mortality (such as the logistic or log-logistic). But it is unlikely that they would have more than trivial effects on final inferences.

case the mortality rate at age 60. So, for example, if Z is a 0/1 covariate with an estimated effect equal to β, exp(β) 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. Projecting prevalence of early health conditions, diabetes, heart disease and mortality*

Using the results of the logistic models, we project five years ahead the prevalence of PEC, diabetes, heart disease and, finally, mortality[11]. We proceed in three stages. First, we estimate the prevalence of PEC five years before the surveys took place. 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

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[11] We choose five years as the projection horizon since it is over a period of only five years that we can calculate rates of change of PEC prevalence. Five years is reasonably short to be both informative and to minimize uncertainties.

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:

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

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:

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

$$\qquad (2)$$

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

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

$P_x^{PEC}(t-5)$ *and* $P_x^{PEC,j}(t)$ 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 disease. 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 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.

We assume two scenarios: in scenario I cohorts five years ahead will experience the same rates of prevalence of poor early health as observed in the first surveys. In scenario II cohorts experience an increase determined by the age-weighted average of the age-specific rate of change in the prevalence of PEC retrieved from the back-projection. Each scenario is associated with prevalence rates of diabetes and heart disease that are consistent with the assumed age-specific rates of prevalence of PEC.

*(iii) Mortality, life expectancy and healthy life expectancy five years ahead.* When each of the two scenarios specified above is combined with assumptions about background mortality and mortality of those who experience diabetes and heart disease, it becomes feasible to calculate projected life expectancy and healthy life expectancy over age 60. To simplify calculations and avoid unnecessary proliferation of scenarios, we will assume that expected (background) improvements in mortality applies to the subpopulations affected by neither heart

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[12] For each age group this rate is equal to the difference between the rate of growth of the total population and the

disease nor diabetes and that the latter two subpopulations experience the mortality estimated from our models. This assumption stacks the deck against the contrarian argument for it removes the possibility of deterioration in mortality among those affected by the two target chronic conditions, a possibility that should not be discarded lightly in view of the fact that these conditions are increasingly penetrating the populations in the lowest levels of socioeconomic conditions. We assume that in the five years following the last surveys the background force of mortality declines at a rate of about 0.5% per year, implying gains in life expectancy of roughly 0.046 years per year or a total of about 0.23 years over five years.[13]

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. In turn, the latter is a product of three pathways: the effects of PEC on the probability of occurrence of a chronic condition, the effect of the chronic conditions on mortality, and the **direct effect** of PEC on mortality (that is, a pathway involving diseases other than heart disease and diabetes). The prevalence of PEC and the magnitude of its effects translate the influence that PEC has on life expectancy and determine the 'room' there is for EC to play a role in future mortality. To shed light on the maximum room that EC has to operate we also estimate a range of values for life expectancy using three counterfactual cases: when all the population experiences PEC (Scenario 3 in Table 5), when nobody experiences them (Scenario 4 in Table 5), when the effects of PEC on chronic diseases (heart disease and diabetes) is three times as high as the estimated ones (Scenario 5 in Table 5). In all cases the benchmark for comparison is the scenario with the estimated effects of current prevalence of PEC, diabetes and heart disease.

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rate of growth of the population with poor early childhood conditions.
[13] This is about half the gains assumed by the Unites Nations' projections for the same period (United Nations 2006). This difference, however, does not affect our calculations since we are not using the United Nations projections as benchmarks.

Just as we evaluate the effects of PEC on projected life expectancy so can we assess its impact on healthy life expectancy. Ignoring the role played by other diseases we estimate healthy life expectancy considering the number of healthy years lost associated with heart disease and diabetes. Healthy life expectancy is estimated using Sullivan's method (Sullivan 1971) which consists of adjusting the number of person-years lived by a cohort by the prevalence of the selected chronic diseases.

## IV. RESULTS

### A. 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.The table reveals that 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 28% more likely to self report diabetes (predicted probabilities equal to 0.32 and 0.25 respectively) and 11% more likely to self-report heart disease (predicted probabilities equal to 0.20 and 0.18 respectively). In Mexico the estimates translate into excess probabilities of diabetes and heart disease among those who experienced PEC: 19% (predicted probabilities equal to 0.19 and 0.16) and 25% (predicted probabilities equal to 0.05 versus 0.04) respectively.[14]

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[14] Note that 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,

Table 2 about here

**B. 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 results reveal the existence of a single and salient regularity: the effects of diabetes and heart disease swamp everything else. These effects are large and are quite similar in the two countries, particularly those associated with diabetes. The relative conditional probabilities of dying among those self-reporting diabetes are twice as high as among those not reporting it in Puerto Rico (predicted conditional probabilities of 0.08 and 0.04 respectively) and Mexico (predicted conditional probabilities of 0.07 and 0.03 respectively). The relative conditional probabilities of dying those reporting heart disease are about 1.4 times higher in Puerto Rico as those not self-reporting it (predicted conditional probabilities of 0.07 and 0.05 respectively) and 1.75 times as high in Mexico (predicted conditional probabilities of dying of 0.07 versus 0.04) than among those not reporting the condition. The *direct effects* of PEC (e.g., those operating through illnesses other than diabetes and heart disease) are statistically insignificant and the variable was removed from the final models predicting mortality. Because the direct effect of PEC on mortality is statistically insignificant in both countries, we only explore the effects of PEC on mortality that work through the increased prevalence and excess mortality associated with diabetes and heart disease.

Table 3 about here

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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.

**C. Projected prevalence of poor early childhood status**

The foregoing results establish a foundation on which we can construct a test of the contrarian conjecture, namely, that mortality and health prospects of future cohorts of elderly population in Latin American countries could be compromised. Below we proceed to show that while the evidence in Tables 2 and 3 is not robust enough to make a strong case for the modified conjecture, it is sufficient to spouse a weak form of it.

*1. The rate of change in the prevalence of poor early health status*

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 our conjecture, these rates should be positive and larger for the youngest cohorts, those that experience the brunt of medical improvements, and positive but smaller in size for older cohorts. Thus, the rates of change in the proportion experiencing poor childhood health should be well described by a monotonically declining curve. 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 rates tend to increase sharply at very old ages and their age-weighted average is positive and close to 0.02. 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 the case of Puerto Rico the rates are mostly positive, and their age weighted average also hovers around a mean of about 0.02 and there is a marked upward trend with age. Since the onset of mortality decline in Puerto Rico was earlier than in Mexico and more of it was

associated with eradication of infectious and parasitic diseases (including malaria, smallpox, dengue and 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 we have little statistical basis to choose something other that a constant rate of increase of the population who experienced PEC in the course of 10 years. We estimate the rate of change as a weighted average of the age-specific rates of change for each cohort and it is equal to 0.02 for Mexico and Puerto Rico.

2. *Projected prevalence of chronic diseases five years ahead.*

Figure 2 displays the proportionate difference in prevalence of diabetes (first panel) and heart disease (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 estimated from the previous five years. The increases in diabetes and heart disease that accompany an increase in the prevalence of PEC are quite modest and they never surpass 0.2% on average. The increases are larger for diabetes in Puerto Rico and for heart disease in Mexico. The small magnitude of these effects are a result of relatively low prevalence of PEC (about 0.38 in Mexico and 0.37 in Puerto Rico). They also reflect the fact that the effects of PEC on both diabetes and heart disease are substantial but not huge.


Figure 2 about here

*3. Projected life expectancy and healthy life expectancy five years ahead*

Table 4 displays projected life expectancies (first panel) and healthy life expectancies (second panel) five years after the last wave of each survey for five different scenarios. The first scenario (column 2 for Puerto Rico and 7 for Mexico) displays values for the case when we assume the current prevalence of PEC in conjunction with the change in the background mortality defined before. The second scenario (column 3 for Puerto Rico and 8 for Mexico) displays values for the case when the PEC prevalence is increased by the average rate of change in PEC estimated for the past five years (0.02). The third scenario (column 4 for Puerto Rico and 9 for Mexico) displays values associated with a scenario where the prevalence of PEC is set to 1. The fourth scenario (column 5 for Puerto Rico and 10 for Mexico) corresponds to a case where nobody experiences PEC. The last scenario (column 6 for Puerto Rico and 11 for Mexico) is one where the PEC prevalence is as observed in the surveys but the effects of PEC on the probability of experiencing diabetes and heart disease is multiplied by a factor of 3.


Table 4 about here


Consider first the extreme cases (when PEC is set to 1 and 0, 3rd and 4th scenarios respectively). Given the estimated effects of PEC, these two cases determine the maximum range of effects on mortality: if everybody experienced PEC, life expectancy at age 60 would decline by about 1.6% in Puerto Rico and by 0.5% in Mexico; if nobody experienced PEC, life expectancy at age 60 would increase by about 0.92% in Puerto Rico and by 0.56% in Mexico. Expected changes in healthy life expectancy express a much larger range: losses and gains could

be on the order of 9 and 4 percent respectively in Puerto Rico and of 4 and 2 percent respectively in Mexico. Second, consider the case when the rate of increase of PEC prevalence is set to 0.02 (second scenario). In this case one would observe virtually the same life expectancy and healthy life expectancy that apply when PEC does not increase. Finally, notice that expected life expectancies would decrease by larger amounts (2 and 1 percent in Puerto Rico and Mexico respectively) if the estimated effects of PEC on the prevalence of chronic conditions were three times as high as it is. The impact on healthy life expectancy is five and four times as large in Puerto Rico and Mexico respectively.

These estimates of expected losses (gains) in life expectancy induced by changes in PEC prevalence may seem small and trivial. However, they should be assessed in a context where life expectancy at age 60 is projected to improve by about 0.05 per year or, given current values, no more than 0.26% per year or 1.3% over five years. The most conservative of scenarios, assuming a rate of increase in PEC of about 0.02 over five years, results in declines in life expectancy of 0.02% in Puerto Rico and 0.01% in Mexico. These losses amount to about 2% of the assumed gains over a period of about five years. But if the rate of increase were larger and/or the effects of PEC were underestimated in our model, the resistance opposed to projected gains in longevity could be much larger.

## V. DISCUSSION

The objective of this paper is to shed light on the 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 in the exposure to infectious and parasitic diseases and improvements

in living standards. Paradoxically, the conditions that led to lower child mortality rates in the past can now undermine sustained gains in future life expectancy at old ages. Could it be the case that the changing composition of cohorts by early health status is in any way related to the slowing down of the rate of decrease in the force or mortality at ages over 60? And, if so, how strong is the resistance that such changes impose on future mortality decreases? Is it strong enough to bring the mortality decline to a complete halt or to reverse it altogether?

We argue that standard mortality projections, such as those of the UN covering the interval 2007-2027, are perhaps too optimistic. The evidence we gather relates to three features: (a) steady state or deterioration of the composition of cohorts by early childhood health status, (b) steady state or deteriorating prevalence of key chronic conditions (heart disease and diabetes); and, (c) large excess mortality associated with these two chronic conditions. The empirical evidence we marshal is not water-tight and the inferences we draw must remain unproven conjectures. This is a weakness of our analysis but not any more than in exercises where it is *ex ante* assumed that life expectancy at older ages can increase uninterruptedly over the short to medium run.

The paper delivers four messages. The first is purely theoretical and refers to the need to specify initial conditions before anticipating the possible within-cohort association between mortality changes at various ages. These initial conditions must be derived from close examination of the nature of mortality decline that fuels changes in mortality rates over the life cycle of a cohort. The second message is that, if verified, the early-adult health connection can be the foundation for a corollary that reverses the relation some researchers have focused on, namely, that given a particular set of initial conditions associated with mortality decline, the within-cohort association between changes in mortality (health status) at early and older ages

may weaken or even become negative. The third message is that, given the nature of mortality decline in LAC, initial conditions there appear to be more propitious to fuel health and mortality trajectories that could weaken the props for continued longevity gains. The fourth message indicates that poor health in early childhood has relatively little room to operate and that its influence on future life expectancy and future healthy life expectancy while not a dominant force is not trivial. Our results suggest that the indirect association between PEC and mortality (via chronic conditions) is crucial: on one hand early conditions have a significant effect on the probability of experiencing diabetes and heart disease and, on the other hand, diabetes and heart disease are important determinants of mortality. We found that individuals who experienced PEC are 28% and 19% more likely to self-report diabetes than those who do not in Puerto Rico and Mexico respectively. The excess risk of suffering heart disease is around 11% in Puerto Rico and 25% in Mexico.

The main inference from these findings is as follows: if poor self-reported health status in early childhood is indeed a proxy for the experience of repeated infections and/or recurrent illnesses, our results are consistent with the inflammation hypothesis reformulated before. But, with the exception of phenomena involving rheumatic heart fever and adult heart disease, they are also consistent with the early nutrition hypothesis. Overall, our findings show small potential losses in life expectancy and healthy life expectancy at older ages when we project PEC five years ahead. Of course, the estimated relative losses in life expectancy will increase if we widen the window of projection. The proportionate losses in life expectancy implied by a conservative scenario, where the proportion of individuals who experienced PEC remains invariant at current levels, relative to a scenario where no individuals experience PEC are on the order of 0.002 per year over a period of five years in Puerto Rico and 0.0010 in Mexico (see column 4 for Puerto

Rico and 9 for Mexico in Table 4). If maintained over 40 years, the potential losses in life

expectancy at age 60 would be on the order of 0.074 in Puerto Rico and 0.040 in Mexico. These

numbers are admittedly small but to put them in perspective they should be compared with

alternative projections. As shown before, the United Nations projections assume that over a

period of 40 years the gains per year in life expectancy at 60 in Mexico will be on the order of

0.06 per year. This figure represents total gains of about 2.4 years in the period considered or an

increase of roughly 13.3% over current values. If our conservative scenario plays out about 2%

of those gains (0.040/2.4) will be unrealized in Mexico whereas in Puerto Rico the gains

foregone will be of the order of 2.6% (0.074/2.8). None of these estimates point toward large

effects but they do suggest the existence of second order effects of some importance. The fact

that they are relatively small is simply an outcome of two factors: the first is that the direct

effects of early conditions on mortality and chronic conditions are not large relative to other

effects. The second is that the prevalence of early childhood conditions is relatively low.

The counterfactual analyses (in Table 4 compare columns 4, 5 and 6 for Puerto Rico and

9, 10 and 11 for Mexico) allows us to elucidate the maximum range of change that early

conditions can induce on life expectancy and healthy life expectancy According to our results,

the main reason for the relatively weak traction of PEC as a determinant of life expectancy has

more to do with the fact that the indirect effects of poor early conditions are somewhat small, not

with the magnitude of prevalence. Extreme changes in the prevalence of poor early health

conditions produce relative gains or losses in life expectancy at age 60 that are quite small. The

highest variation is observed by increasing the effects of early health conditions on mortality and

on target chronic diseases. The losses or foregone gains stretched over a period of 40 years are

tantamount to a whopping 3.2 years of life expectancy at age 60 in Puerto Rico and to 1.6 years

in Mexico. Foregone gains in healthy life expectancy are much larger.

There are good reasons to suspect that our estimates of effects of poor early childhood conditions are on the low side and, therefore, that the figures obtained assuming higher effects are not to be dismissed too lightly. One of the reasons for attenuated effects is errors in self reports of conditions. Even if there were no self reporting errors, the estimates of the effects of early conditions on chronic diseases and mortality may be underestimated because the indicator of early conditions we use here is an exceedingly blunt instrument to assess the types of conditions commonly associated with it in the medical and epidemiological literature. As a consequence, despite the fact that self-reported conditions and the anthropometric measures considered in the construction of the indicator are fairly reliable, we are probably no more than scratching the surface of the effects of individuals' early experiences. Another factor that plays against the contrarian view is that we have only considered effects of poor early health conditions that operate directly, not through obesity. If the latter are relevant, the estimated losses we obtain straight from our model are most definitely on the low side.

Since we do not possess the proverbial crystal ball, we cannot guess what will happen in the medium run. But we can state with some certainty that changes in composition of the cohorts by early childhood health experiences that could be expected given the nature of mortality decline in Mexico and other countries of the LAC region will act as a brake and oppose some resistance to further increases in longevity.

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**Table 1.    Descriptive Analysis of Variables in MHAS and PREHCO**

| Variables | MHAS Mean | MHAS Missing (%) Total | MHAS Missing (%) No Proxy | MHAS Missing (%) With Proxy | PREHCO Mean | PREHCO Missing (%) Total | PREHCO Missing (%) No Proxy | PREHCO Missing (%) 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 Disease [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 | 0.06 | 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 |
| Normal weight [c] | 0.2 | 17.6 | 17.5 | 19.1 | 0.2 | 10.9 | 5.3 | 48.7 |
| Obese [d] | 0.2 | 17.6 | 17.5 | 19.1 | 0.2 | 10.9 | 5.3 | 48.7 |
| Low weight [e] | 0.4 | 17.6 | 17.5 | 19.1 | 0.4 | 10.9 | 5.3 | 48.7 |
| High weight [f] | 0.2 | 17.6 | 17.5 | 19.1 | 0.2 | 10.9 | 5.3 | 48.7 |
| 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 a 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 disease is if individuals suffered from heart attack. For PREHCO, this variable includes more general heart conditions (e.g. angina, coronary disease, and other cardiac problems).

[c] Normal weight: BMI between the 40th and 60th percentile of the distribution

[d] Obese: BMI between the 60th and 80th percentile of the distribution

[e] Low weight: BMI lower than the 40th percentile of the distribution

[f] High weight: BMI greater than the 80th percentile of the distribution

# Table 2. Results of Logistic Models Predicting Self-Reported Diabetes, Heart Disease and High Weight among Elderly in Mexico and Puerto Rico

| Independent Variables | Mexico | | | Puerto Rico | | |
|---|---|---|---|---|---|---|
| | High BMI | Diabetes | Heart Disease [a] | High BMI | Diabetes | Heart Disease [a] |
| 65-69 yrs old | -0.23 * | 0.15 † | 0.34 * | -0.29 * | 0.08 | 0.28 * |
| 70-74 yrs old | -0.13 | 0.08 | 0.54 ** | -0.15 | 0.17 † | 0.42 *** |
| 75-79 yrs old | -0.32 ** | -0.10 | 0.69 *** | -0.18 | 0.03 | 0.53 *** |
| 80+ yrs old | -0.60 *** | -0.13 | 0.91 *** | -0.55 ** | -0.15 | 0.57 *** |
| Ref. cat.: 60-64 | | | | | | |
| Female | 0.37 *** | 0.32 *** | -0.39 ** | 0.68 *** | -0.01 | -0.09 |
| PEC | 0.15 * | 0.16 * | 0.30 ** | -0.19 * | 0.33 *** | 0.15 † |
| 1-5 yrs school | 0.06 | 0.09 | 0.05 | 0.08 | -0.04 | -0.18 |
| 6 yrs school | -0.20 † | 0.18 † | 0.40 * | -0.19 | -0.23 | -0.08 |
| 7+ yrs school | -0.33 ** | 0.06 | 0.27 | -0.06 | -0.18 | -0.48 ** |
| Ref. Cat.: 0 yrs school | | | | | | |
| Obese [b] | | 0.13 | 0.13 | | 0.11 | 0.17 |
| Low Weight [c] | | -0.11 | -0.04 | | -0.48 *** | -0.08 |
| High Weight [d] | | 0.16 † | 0.38 * | | 0.15 | 0.32 ** |
| Ref. cat: Normal weight [e] | | | | | | |
| Constant | -0.73 *** | -1.91 *** | -3.64 *** | -0.85 *** | -0.83 *** | -1.55 *** |
| *Number Obs.* | 4,572[c] | 7,604 | 7,604 | 3,166[c] | 5,286 | 5,286 |

*Source*: From author calculations of MHAS (2001) and PREHCO (2000) data.

*Notes*:

*(1)* Estimates were obtained using the entire data set after performing multiple imputations to include missing data (Schafer 1997).

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

[b] Obese: BMI between the 60th and 80th percentile of the distribution

[c] Low weight: BMI lower than the 40th percentile of the distribution

[d] High weight: BMI greater than the 80th percentile of the distribution

[e] Normal weight is BMI contained between the 40th and 60th percentile of BMI distribution

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

**Table 3.** **Results of Logistic Models Predicting Mortality among Elderly in Mexico and Puerto Rico** (2$^{nd}$ specification for mortality)

| Independent Variables | Mexico | | Puerto Rico | |
|---|---|---|---|---|
| Age (*minus* 60) | 0.09 | *** | 0.09 | *** |
| Female | -0.25 | * | -0.47 | *** |
| Poor early health conditions(PEC) | -0.11 | | 0.12 | |
| Heart disease [a] | 0.62 | ** | 0.43 | *** |
| Diabetes | 0.73 | *** | 0.67 | *** |
| 1-6 yrs school | -0.14 | | 0.16 | |
| 6 yrs school | -0.17 | | 0.07 | |
| 7+ yrs. school | -0.07 | | -0.18 | |
| (ref. cat.: 0 yr) | | | | |
| Obese [b] | 0.35 | | 0.09 | |
| Low weight [c] | 0.55 | ** | 0.78 | *** |
| High weight [d] | 0.27 | | 0.08 | |
| (ref. cat.: normal weight) | | | | |
| Constant | -4.00 | *** | -3.47 | *** |
| *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 set after performing multiple imputations to include missing data (Schafer 1997).

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

[b] Obese: BMI between the 60$^{th}$ and 80$^{th}$ percentile of the distribution

[c] Low weight: BMI lower than the 40$^{th}$ percentile of the distribution

[d] High weight: BMI greater than the 80$^{th}$ percentile of the distribution

[†] *p<.10*; [*] *p<.05*; [**] *p<.01*; [***] *p<.001*

**Table 4.**    **Observed and Counter-Factual Life Expectancy and Healthy Life**

**Expectancy at age 60, 70 and 80 in Puerto Rico and Mexico**

| Age | Puerto Rico | | | | | Mexico | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Scenarios | | | | | Scenarios | | | | |
| | 1st: Observed | 2nd: Rate=.02 | 3rd: PEC=1 | 4th: PEC=0 | 5th: Higher Effect | 1st: Observed | 2nd: Rate=.02 | 3rd: PEC=1 | 4th: PEC=0 | 5th: Higher Effect |
| | Life Expectancy | | | | | | | | | |
| 60 | 19.3 | 19.3 | 19.0 | 19.5 | 18.9 | 19.2 | 19.2 | 19.1 | 19.3 | 19.0 |
| 70 | 12.2 | 12.2 | 11.9 | 12.3 | 11.8 | 12.4 | 12.4 | 12.3 | 12.5 | 12.2 |
| 80 | 6.9 | 6.9 | 6.7 | 7.0 | 6.6 | 7.4 | 7.4 | 7.3 | 7.5 | 7.3 |
| | Healthy Life Expectancy | | | | | | | | | |
| 60 | 11.4 | 11.4 | 10.3 | 11.9 | 10.2 | 15.3 | 15.3 | 14.8 | 15.6 | 14.5 |
| 70 | 7.0 | 7.0 | 6.3 | 7.3 | 6.1 | 9.9 | 9.9 | 9.5 | 10.1 | 9.4 |
| 80 | 4.1 | 4.1 | 3.7 | 4.3 | 3.6 | 6.0 | 6.0 | 5.7 | 6.1 | 5.7 |
| | Relative Difference: Life expectancy (%) | | | | | | | | | |
| | - | -0.02% | -1.70% | 0.92% | -2.15% | - | -0.01% | -0.93% | 0.50% | -1.34% |
| | - | -0.03% | -2.19% | 1.28% | -2.96% | - | -0.01% | -1.26% | 0.62% | -1.68% |
| | - | -0.03% | -2.78% | 1.78% | -4.06% | - | -0.01% | -1.83% | 0.73% | -1.96% |
| | Relative Difference: Healthy Life expectancy (%) | | | | | | | | | |
| | - | -0.09% | -9.19% | 4.72% | -10.50% | - | -0.04% | -3.36% | 1.92% | -4.96% |
| | - | -0.11% | -9.79% | 5.46% | -11.82% | - | -0.04% | -3.87% | 1.99% | -5.17% |
| | - | -0.12% | -9.65% | 6.04% | -12.80% | - | -0.04% | -4.69% | 1.94% | -5.01% |

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

**Figure 1.     Age-Specific Rates of Change in the Proportion of Poor Early Health**

**Conditions among Elderly in Mexico and Puerto Rico**
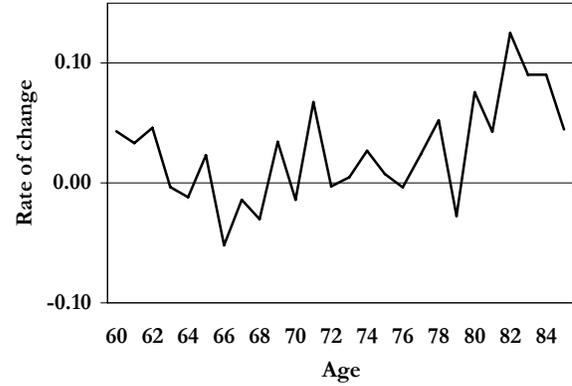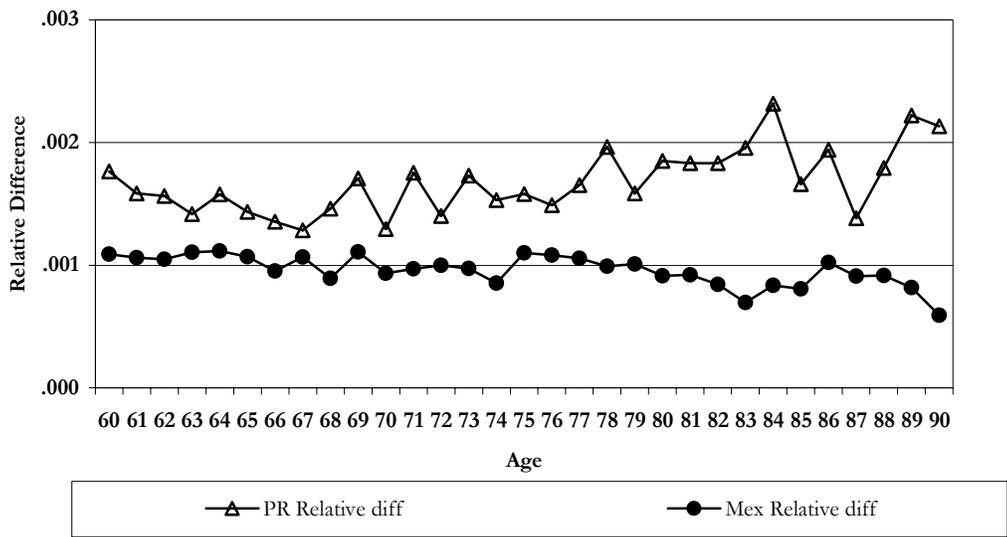


|         a. Mexico         |         b. Puerto Rico         |

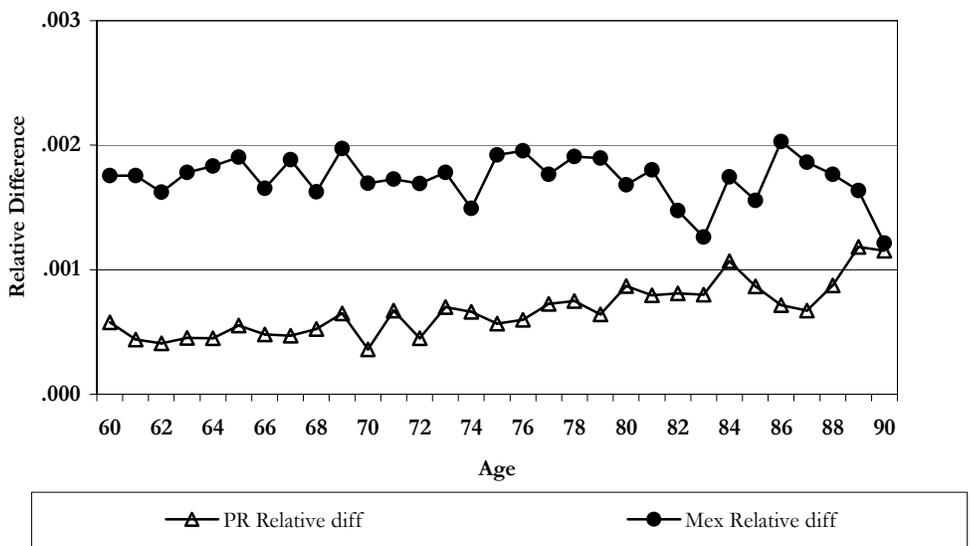*Source*: MHAS (2001-2003); PREHCO (2000-2006).

*Note*:

The weighted average rate of change in the probability of experiencing poor early conditions in Mexico and Puerto Rico is

0.02.

**Figure 2.       Relative Difference between Counter-Factual and Observed Prevalence of**

**Diabetes and Heart Disease among Elderly in Mexico and Puerto Rico**



i. Diabetes



ii. Heart Disease

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

*Note*: *Counter-factual* prevalence of chronic disease is defined by assuming increases in the prevalence of poor

early conditions at the weighted average rate of change in the probability of experiencing poor early conditions. In

Mexico and Puerto Rico, the rate is 0.02.