THE PROBLEM

We argue that, despite steady past progress in longevity, the short and medium term prospects for life expectancy in Latin America and the Caribbean (LAC) could be compromised. Older people who began attaining age 60 after the year 2000 carry the scars of experiences that can potentially translate into higher susceptibility to chronic conditions and higher levels of mortality. Once dismissed as unthinkable, declines in life expectancy already made their debut in modern societies: massively in Sub Sahara Africa with the entrenchment of the HIV/AIDS epidemic and, more sparsely, but equally unexpectedly, in countries traumatized by the collapse of the Soviet regime. Contrary to other researchers (Oeppen and Vaupel. 2002; Wilmoth, 1988; Crimmins and Finch, 2006; Finch and Crimmins, 2004) we suggest that the route to further increases in life expectancy may be shutting down in LAC. Unlike Sub-Saharan Africa and the former Soviet Republics, however, longevity in the region will be driven neither by sudden shifts 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.

THEORETICAL JUSTIFICATION

a. Linkages between early childhood and adult health status

Rapidly accumulating knowledge in developed countries suggests there are several mechanisms through which early childhood conditions may affect the onset of two of the most prevalent conditions in LAC, namely, diabetes II (diabetes mellitus) and heart disease. Some of these mechanisms are highly specific such as those associated with *sequelae* of processes that may start *in utero*, develop shortly before and/or around birth ("fetal origin hypothesis") or during other "critical periods" (Barker,1994). They include also a few, less specific mechanisms such as those that operate through socioeconomic conditions experienced in early childhood, or some believed to be associated with acute episodes of very specific childhood illnesses and their cumulative influence on the late onset of some chronic diseases (Kuh and Shlomo, 1997; Smith,2002). A somewhat different set of mechanisms involves the delayed effects of inflammatory processes triggered by recurrent contraction of infections and parasitic diseases during early ages (Finch and Crimmins, 2004; Crimmins and Finch, 2006). Empirically distinguishing between these mechanisms is a thorny affair because not least because they all lead to the same implication, namely, the removal of conditions that induce malnutrition and or exposure/contraction of infections and parasitic diseases will simultaneously reduce infant and/or early childhood mortality and mortality at older ages among members of the same cohort. Thus, mechanisms linking early childhood and adult life provide an opportunity for the macro forces driving mortality changes to express themselves by shaping cohorts' fortunes throughout their entire lifespan. Acting as a conveyer belt, these forces tend to spread mortality decline within cohorts over long stretches of time rather than altering mortality rates simultaneously for multiple cohorts in a short interval (Barbi and Vaupel, 2005; Kannisto, 1994).

b. Expected outcomes of the 'early child health-adult health connections

Proponents of theories that associate mortality changes at older ages with changes in cohorts' health experiences during early childhood have drawn one and only one corollary, namely, that cohorts that benefit from mortality reductions attributable to nutritional improvements and/or to reduced rate of exposure/contraction of infectious and parasitic diseases will, *ceteris paribus*,

experience lower mortality risks at old age[1]. We argue that this may be so but **only when strict conditions pertaining to the nature of forces that explain improvement in early child mortality prevail.** Even if it were thoroughly validated, and this is far from being the case, the conjecture about the 'early health-adult health' connection itself does not necessarily imply solely positively correlated changes at young and older ages within cohorts. The fact that this may have been so in high income countries is no more than a historical accident that could have turned out quite differently had the initial conditions that precipitated mortality decline been different. To fully spell out the empirical implications of secular mortality declines in the presence of early 'childhood health-adult health' linkage one cannot stop the investigation after having identified mechanisms that produce the linkage, whether it is intrauterine growth, inflammation processes, or nutritional status during the first year of life. Full understanding of the expected relations between mortality risks over the life cycle across cohorts can only be decided after accounting for *the nature of forces that generate the secular mortality decline.*

c. Causes of secular mortality decline and a paradox

We examine four classes of causes of mortality declines, the conditions they are likely to influence, and the terrain they prepare for the expression of the 'early health-adult health' connection (section not shown in this extended abstract). We then review empirical evidence showing that the historical conditions prevailing in LAC should lead to the expression of mechanisms that generate *a near zero or negative correlation between changes in early childhood mortality and older age mortality within cohorts* (section not shown in this extended abstract). In view of this we conjecture that, barring drastic changes of conditions, mortality decline at older ages may grind to a halt and perhaps even reverse thus effectively bringing an end to five decades of continued increases in life expectancy.

c.1. The history of LAC: It is known and relatively uncontroversial that the mortality decline that took place in LAC or, for that matter, in other low income countries, after 1940 or 1950 was heavily influenced by public health interventions and a revolution in immune-chemotherapy. Improvements in standards of living and associated increases in nutrition may have been implicated as well but only marginally so. Two important remarks follow. First, just because we can isolate with some precision the forces that triggered the secular mortality decline, it does not mean we can draw inferences about the relative importance of nutrition or inflammatory processes as factors responsible for observed correlations between mortality rates at different ages. This is because of dense synergisms between infections and nutrition. Second, in this particular historical case there is an added element we must consider: if the bulk of mortality decline is attributable to a revolution in immune-chemo-therapy it does not follow at all that cohorts blessed by improvements as children will be comprised by individuals who, on average, either experienced less inflammatory process or belong to better nourished groups. Whether or not this conventionally expected outcome obtains depends on the details of this particular instance of mortality decline. Indeed, we argue that conditions in the region are such as to lead to a somewhat unexpected corollary of the 'early health-adult health' connection: because of the nature of forces responsible for the decline the composition of cohorts that benefitted from

[1] The literature on the topic is notoriously sloppy about the precise 'formal' translation of the corollary. While most authors suggest that there should be a correlation between **levels** of early child and older mortality across cohorts, we show in the paper that this is incorrect. What one should expect , instead, is a correlation between **lagged changes** of mortality rates at different ages across cohorts.

mortality improvements will be biased toward members who are more likely to express the mechanism linking early conditions and adult chronic illnesses. An increasing fraction of elderly will belong to birth cohorts whose members survived (but were not spared) infectious and parasitic diseases that prior to the decline would have killed them. To the extent that a nontrivial part of the decline is a result of the efficacy of immune-chemo-therapy, the fraction of individuals in a cohort likely to have experienced suboptimal nutrition or frequent episodes of infections and parasitic diseases during childhood will increase steadily for years after the mortality decline gets under way[2]. If the conjecture relating early conditions and adult chronic diseases holds then, other factors being equal, the prevalence of those diseases ought to increase over time as well. And here is the rub: barring sharp changes in medical technology, life expectancy at older ages may begin to grow at a significantly lower rate or cease to increase altogether even if 'background' mortality [3] continues to decline. As a consequence, the correlation between mortality changes in early childhood and adult ages for cohorts involved in the process may converge to zero and even become negative rather than being strongly positive as expected from conventional renditions of the early health-adult health connection.

This pessimistic outlook will fail to materialize if and only (a) lethality rates associated with the target chronic conditions decrease enough to compensate for the increases in their prevalence or (b) the rate of background mortality decline is large enough and more than offsets increases in their prevalence. If neither (a) nor (b) occurs, older people in the newer cohorts will experience higher mortality than older people in the preceding ones.

c.2. Are the associations large enough to matter? 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 1940 and most definitely after 1950. For the first twenty to thirty years, 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: this is the period when chemo-immune-therapy makes its debut in the area and begins to be massively use (some would say misused). Empirical investigations show that more than fifty percent of this decline (Preston, 1976; Palloni and Wyrick, 1981) is associated with the deployment of public health tactics and medical technology that diminished exposure to infectious and parasitic disease and decreased their lethality. The remaining fraction is associated with improvements in standards of living (income) and, surely, with elevated nutritional status. These estimates are coarse but shed light on the processes that evolved in the region.

Furthermore, consider the fact that at least forty percent of the total increase in the rate of increase of the population aged 60 above after the year 2000 in the LAC region is associated with the post-1940 mortality decline (Palloni et al, 2005). This regularity and the nature of the mortality decline just described, suggest that the rate of increase of the elderly in the region can be directly linked to augmented survival among individuals who were exposed to and who experienced bouts of infectious and parasitic illnesses but who survived them in a new environment of bolstered recovery rates. The expected negative or zero relation between changes in early childhood and old age mortality over cohorts follows immediately.

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[2] A caveat is necessary. Even if chemotherapy had been the only factor behind massive mortality reduction, one would expect some improvements in nutritional status as secondary outcome of shorter or less intense episodes of illnesses.

[3] By 'background' mortality conditions we understand those which are unrelated to the target chronic conditions, e.g. those implicated in the early health-adult health connection, and could be the result of period effects.

d. Empirical conditions for reversals of increasing longevity trends

By all accounts European countries have experienced remarkably sustained decreases of mortality rates at adult ages. The estimated rate of decline is approximately 1 percent per year. If maintained over a span of fifty years in mortality regimes with a life expectancy at age 60 between 15 and 20 years, as in Puerto Rico and Mexico, a 1 percent per year decline in mortality at all ages above 60 yields, on average, gains in longevity of the order of .10 years per year. Recent evidence from adjusted data (Palloni and Pinto, 2004) indicates that life expectancy at age 60 for the most advanced LAC countries increased from about 18 years in 1950 to about 23 in 1995 in approximately *linear fashion* thus yielding (average) yearly gains of just about .10 years per year. Note that linear increases in life expectancy imply that the force of mortality decreases but at a *decreasing rate*. Clearly, the rates of change of mortality risks must have attained values exceeding 1 percent per year at some point during the past[4]; this reinforces the idea that mortality decline in LAC is a feat of exceptional nature. But, is it justifiable to assume that this LAC exceptionalism will rule in the near future as is assumed, for example, in the United Nations or CELADE population projections for the region?

Our aim is not to pose a strong version of a contrarian argument according to which future gains in life expectancy will unambiguously converge to zero or even become negative. We rather spouse a weak version, namely, that prevailing empirical conditions constrain and oppose stiff resistance to uninterrupted progress in longevity in the LAC.

EMPIRICALLY TESTING THE ALTERNATIVE CONJECTURE

To prop the weak version of the contrarian view we draw empirical evidence from eight surveys of elderly people in LAC and show that the following regularities hold:

d.1. The effects of early childhood health status on adult health, particularly on the prevalence of diabetes and heart disease, are strong and as expected if the 'early health-adult health' mechanisms operate;

d.2. Using estimated mortality from two panels (Mexico and Puerto Rico) we project backwards the population reported to have poor early childhood status and estimate cohort trends that show constant or mild increases in the proportion of older people who experienced poor health status as children;

d.3.We project forward the force of mortality for people older than 60 assuming that cohort trends uncovered in (d.2) prevail, that the incidence of diabetes and heart disease conditional on early health status is as estimated in the eight surveys and, finally, that the excess risk of mortality associated with diabetes and heart disease are as estimated in the two panel studies.

d.4. We show that the resulting force of mortality projected forward follows a trajectory implying either decreases of life expectancy at age 60, trivial increases and, at any rate, induces marked disruption in the trend of steadily increasing life expectancy experienced in LAC for more than half a century.

[4] These and other arguments made in the paper rest on the simplifying assumption that the rate of decline of mortality risk is age invariant (over age 60). This may not reflect well the process, at least at its beginning, but it is probably close to the actual experience during the last twenty years or so.