CAN INCREASING LONGEVITY COME TO HALT?
ALTERNATIVE PATHS OF LONGEVITY IN LATIN AMERICA

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Introduction: forces that can derail future progress of longevity

Despite serious recent bumps, the march towards longevity in most regions of the world appears to be uncontested. The HIV/AIDS epidemic and the collapse of the Soviet Union are the only two blemishes that led to massive retrenchments of life expectancy in two large regions of the world. But, elsewhere, progress overwhelms occasional fluctuations and, in most cases, is so imposing that it is easy to ignore temporary and localized faltering of an otherwise impeccable record.

But underneath the veneer of invincibility of the march towards longer life, hides a more complicated reality. The course of future mortality in most populations depends on two sets of factors. The **first** has to do with prospective improvements in medical technologies that could reduce case fatality of important chronic diseases, including heart disease, stroke and some forms of cancer. By and large the reductions in adult mortality experienced in the last two decades are largely attributable to innovations that increase early detection and much improved treatments. An important exception is dementia that is slowly emerging as a new threat and should not be ignored as a possible obstacle to higher future levels of mortality. The science of its etiology has progressed at a painfully slow pace and barring spectacular developments it the near future it could become a barrier against further progress.

The **second set of factors** is related to changes in cohort composition of the elderly population. The distribution of deaths in modern mortality regimes is shifted toward older ages, with gradual increases in the modal age at death paired with a plummeting variance. Because of losses in the curvature of the survival function future gains in life expectancy will not only occur at older ages but will also require larger

proportionate decreases in age specific mortality rates. And therein lies the rub: since the history of exposures and experiences of a cohort constitutes the raw material with which their mortality in later life is sculpted, it is at least possible that the patterns of mortality rates accelerations, decelerations, or steadiness that we will observe in the future reflect the rhythm of inflow of cohorts with heterogeneous pasts. This potential malleability of older age mortality patterns is a product of two radically different forces.

a. The importance of past behaviors

The first is cohorts' exposure to risks that are the product of adoption of behaviors whose effects are visible only after considerable time lags. Patterns of smoking are the most obvious ones so much so that they explain about two thirds of the difference in life expectancy at age 50 between the US and other high income countries (National Research Council, 2010). These estimates are quite precise mostly because of the tight connection between lung cancer and COPD and smoking. Diet and physical activity and the resulting trajectory of obesity are another obvious illustration. Indeed, it is believed, though not conclusively proven, that set backs in the progress toward higher life expectancy in the US and other high income countries will emerge very soon as a direct result of rapidly increasing obesity prevalence at all ages. But unlike smoking, the potential impact of obesity is somewhat unclear because the relation between it and chronic ailments is not as tight as that between smoking and lung cancer and COPD.

b. The influence of past regimes of mortality decline

The second force that works through cohort composition is less obvious and applicable mostly to countries whose secular mortality decline is recent and is dominated by the revolution in medical technology that began after 1930. A large fraction of individuals born after 1930 in Latin America and other low to medium income countries were able to survive beyond early childhood not because of improvements in standards of living and nutritional status but because of the diffusion of chemotherapy and medical technologies that decreased fatality rates of the most lethal infectious diseases. While exposure to diseases was also reduced through large public health programs and expansion of infrastructure (albeit with a great deal of regional variability), the main mechanism behind the sharp increases in life expectancy between 1930 and 1970 was not a retrenchment of contraction rates but increased resistance and improved recovery (Palloni and Pinto, 2011; Preston, 1981).

The above suggests that members of cohorts who enter the age group 50 an over after the year 2000 will be scarred by early experiences that have the potential at least to induce increases in the risk of heart diseases and diabetes, precisely two of the chronic conditions that dominate the landscape of mortality at adult ages (Barker, 1998; Klugman and Hansen, 2006). Whether or not these early experiences manifest themselves in higher incidence of these chronic diseases will depend on the strength of the linkage between exposure to poor nutrition and experience with infectious diseases, on the one hand, and well-defined chronic conditions on the other (including COPD, heart disease, stroke, and diabetes). Estimates of this quantity vary widely and, as a consequence, the ultimate impact of this cohort related force on the future of life expectancy remains elusive. It should be noted what this cohort related force is not just a product of changes in the frailty or vulnerability composition of a cohort, a result of the decreasing severity of mortality risks experienced during infancy, early childhood and adolescence. Rather it is

an outcome of increased frailty due to early exposures that scar individual and express themselves only in later life, with lags that depend on latency periods or on interactions with subsequent exposures throughout the life course.

While we cannot guess the impact of future medical technology it is possible to estimate bounds for the effects of the other set of factors, namely, composition of cohorts by past behaviors and by past exposures. In this paper we focus on the latter only and compute bounds for the impact on future life expectancy and healthy life expectancy of changing composition of cohorts by early exposures. We do this for selected countries in the Latin American region that together span the entire array of mortality decline that took place after 1930 in the region: Argentina, Brazil, Costa Rica, Chile, Mexico. We show that the influence of regimes of past mortality decline could have significant and durable influence on future older age mortality if the impact of early child conditions is as large as estimated from very recent data sets.

Methods and data

There are two quantities we need to estimate. The first is the fraction of the population 50 and over that is more likely to be at risk of expressing conditions experienced early in life as adult chronic disease. The second is the excess morbidity and mortality risks among those who are at risk, namely, those who are more likely to have survived regimes the combined poor nutritional status, high exposure to infectious diseases but lower fatality rates. Although slightly more difficult to obtain, these two quantities lead to the computation of an attributable risk which can be translated into lost years of life expectancy and healthy life expectancy.

a. The population at risk

The procedure to estimate the population at risk requires two steps. First, we project forward the population aged 50 and over starting in 1950 and ending in 2050. We do this using observed (1950-2010) and UN-projected life tables (2010-2050). We then repeat the projection but keeping mortality at the same levels it had in 1950. The difference in the size and rate of growth of the population 50+ during the period 2010-2050 can then be partitioned into a fraction contributed by improvements in mortality at ages 50+ and a fraction contributed by the improvements in mortality at ages between 0 and 50. In turn the latter can be decomposed into a part due to improvements between ages 0 and 10 and another one due to improvements at ages 10 and above. Finally, all these quantities can be partitioned into components associated with different groups of causes of death. In all cases we can also estimate the magnitude of the changes due to changes between 1950 and 1980 and between 1980 and 2050. Let θ_{xi} be the counterfactual population in the age group (x, x+1) that would not have survived to that age after 2010 had the mortality decline associated with group of cause of death i between ages 0 and 10 and experienced during the period 1950-80 not taken place at all. The quantity $\Phi_x = \Sigma_i \theta_{xi}$ is simply the total counterfactual population, irrespective of cause of death. In what follows we will work with Φ_x but the algebra applies equally well to any of its components.

The second step is to estimate the size of the population that is more likely to express the effects of early conditions. We do this by first assessing the role of medical

technologies in the mortality decline between 1950 and 1980 (Palloni and Pinto, 2010). We estimate alternative values of the fraction of mortality decline during 1950-1980 that is attributable to the diffusion of antibiotics and, less so, to innovations of public health. Let λ_k be the k^{th} alternative value of this parameter. It follows, that the upper bound of the size of the population aged (x,x+1) that could express the deleterious consequences of early conditions is $\lambda_k * \Phi_x$. This is a true and consistent upper bound only if the fraction of the counterfactual population associated with improvements in nutrition and standard of livings (and not with medical technology) who can express early conditions at adult ages is near zero.

b. Excess risks

The second quantity that needs to be estimated is the excess mortality and morbidity risks among those who experience poor early conditions. We estimate these potential excesses using three two wave surveys of elderly people (Mexico, Costa Rica and Puerto Rico) from which we can retrieve markers of early conditions (including socioeconomic conditions, early nutritional status and exposure and contraction of infectious diseases). Because these are two-wave surveys we can observe mortality and disease incidence. In all cases individuals who are classified as at risk of expressing early conditions experience higher mortality, higher incidence of heart diseases and diabetes as well as higher incidence of disability. Let σ_{xh} and σ_{xd} be the excess risk mortality risks associated with heart disease and diabetes respectively experienced in the age group (x,x+1) and let φ_x be the total excess mortality in the age group (x,x+1) associated with σ_{xh} and σ_{xd} . It follows that the total mortality risk in the age group (x,x+1) and year t can be approximated by:

$$\mu(x,t) = \mu_o(x,t)*\{(N(x,t)- \Phi_x)/N(x,t)\}+\{(1- \lambda_k)* \Phi_x/N(x,t)\}+ \{(\mu_o(x,t)* \varphi_x * \lambda_k* \Phi_x/N(x,t)\} \quad (1)$$

where N(x,t) is the total projected population in the age group (x,x+1) at time t. This expression applied to all ages above 50 leads to an estimate of the life expectancy at age 50 and time t which can then be compared with the projected life expectancy using life tables that do not account for the potential influence of early conditions. Analogous calculations enable us to calculate counterfactual estimates of healthy life expectancy.

Importance of the results

The estimates described before constitute bounds for the impact of changes in cohort composition by early exposure on future life expectancy in countries of the Latin American region. These can then be joined with estimates of the bounds of effects of increasing proportion of smokers and obese individuals to yield upper and lower bounds of one (out of two) set of factors that may slow-down or stop altogether future increases in life expectancy in the region. Whether or not these effects are manifested at all will depend on the speed of improvements in medical technology, a factor whose influence in the past has been substantial but whose future contribution is difficult to anticipate.