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Longevity in the Twenty-first Century: How Strong is the Tug of the Past?

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PREFACE

The Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat organized an Expert Group Meeting on "Priorities for Improved Survival: ICPD beyond 2014" at the United Nations Headquarters in New York on 21 and 22 October 2013. The meeting was convened to inform substantive preparations for the forty-seventh session of the Commission on Population and Development in April 2014. In light of the twentieth anniversary of the 1994 International Conference on Population and Development (ICPD), the Commission's theme for 2014 is an "Assessment of the status of implementation of the Programme of Action of the International Conference on Population and Development".

The meeting brought together experts from different scientific disciplines and regions of the world to address key questions about the progress in improving survival at different stages of life since the ICPD, as well as the challenges and opportunities for future mortality reduction. A selection of the papers prepared by experts participating in the meeting is being issued under the Expert Paper Series published on the website of the Population Division (www.unpopulation.org).

This paper focuses on the role that three sets of conditions may play in the course of future adult life expectancy in the Latin American and Caribbean region. First, it examines the implications of changing early life conditions that have shifted the composition of cohorts with respect to their frailty, thereby changing their susceptibility to mortality due to certain non-communicable diseases that emerge later in life. Next, it presents an analysis of 6 countries in the region showing that in countries with the highest smoking prevalence, increases in mortality attributable to cigarette smoking counteracted reductions in mortality from other causes, limiting the gains achieved in life expectancy at age 50. Finally, the paper assesses the possible impact of past trends in obesity prevalence and associated chronic conditions on life expectancy at age 50 using evidence from Mexico.

The Expert Paper series aims at providing access to government officials, the research community, non-governmental organizations, international organizations and the general public to overviews by experts on key demographic issues. The papers included in the series will mainly be those presented at Expert Group Meetings organized by the Population Division on the different areas of its competence, including fertility, mortality, migration, urbanization and population distribution, population estimates and projections, population and development, and population policy.

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A. INTRODUCTION

The past history of demographic regimes holds at least some clues about their future. Past trends of health and mortality and their determinants may offer, when properly used, an opportunity to produce informed health and mortality forecasts in the short to medium run. Inevitably, the unfolding of unforeseen events and shocks will leave imprints on health and mortality at various ages expressed as period effects. But equally deserving of attention are events and circumstances that shape the survival of entire birth cohorts whose fate may be tightly determined by conditions that mark their early upbringing, adolescent exposures to ecological and disease environments, and health behaviours. Different cohorts may have sharply different experiences with exposure to illnesses and with adoption of and adherence to behaviours whose effects may become visible only after considerable time lags. Patterns of smoking, diet, and physical activity, and the resulting trajectory of conditions such as obesity and chronic illnesses are an obvious illustration.

This paper focuses on the role that three sets of conditions may play in the course of future adult life expectancy in the Latin American and Caribbean (LAC) region. We argue that knowledge about these conditions will increase our ability to project future mortality levels and patterns, improve understanding of the mix of health risks that may become relevant and, finally, strengthen the stock of knowledge to inform general policies or particular interventions. These conditions are: (a) changing composition of cohorts by early conditions; (b) evolution of the smoking epidemic among adults; and (c) past trends of obesity prevalence and associated chronic conditions. These three phenomena share a useful feature: by definition, one knows or can measure with some precision the composition of cohorts by exposure to a risk and compute the magnitude of such risk. For example, we know that individuals belonging to birth cohorts born around 1950 who began smoking in their youth and who survived to be age 60 in 2010 are at high risk of contracting lung cancer, chronic obstructive pulmonary disease (COPD) or cardiovascular diseases (CVDs). Armed with knowledge about their smoking history and about the precise link between the behaviour and excess mortality, we can estimate smoking- attributable risk and associated excess mortality for those cohorts. Barring sudden advances in medical technology to screen, detect and treat the diseases, the estimated mortality excess should have a fairly narrow range of uncertainty. A similar argument, albeit one not blessed with the same qualifications about precision, applies to the other two phenomena, exposure to early childhood conditions and obesity.

The three conditions referred to above offer the researcher or policy maker advanced knowledge of the population composition by risk factor of interest (e.g., smoking, diet and obesity, early hepatitis B infection, rheumatic fever, etc.) and about the linkages between risk factors and mortality. It therefore becomes feasible to compute the drag exerted by a cohort's past exposures on its future mortality. Improved knowledge of these phenomena should not only increase accuracy of mortality projections but also strengthen the formulation of more responsive and better informed health policies.

The paper is organized as follows. Section B reviews the influence of changes in the composition of adult cohorts by early conditions. Section C examines the implications for mortality of cohorts implicated by the diffusion of smoking behaviour. Section D describes the potential impact on adult mortality of continued increases of the prevalence of obesity and associated chronic conditions, particularly cancers, type 2 diabetes and CVD. Section E concludes.

B. EXPOSURE TO EARLY CONDITIONS AND MORTALITY RISKS AT OLDER AGES

A large fraction of individuals born after 1930 in LAC (and in a majority of low- and middle-income countries elsewhere), survived beyond early childhood as a result of the spread of knowledge about infectious diseases, vector eradication campaigns and, most importantly, the diffusion of chemotherapy

and medical technologies that reduce the lethality of infectious diseases. Unlike their counterparts in Western Europe and Northern Europe at the turn of the century, the survival gains of members of these cohorts were not triggered by improvements in standards of living and nutritional status. While exposure to diseases was also reduced through intelligent use of germ theory, large public health programs, and expansions of basic infrastructure, the main engine behind regional increases in life expectancy between 1930 and 1970 was increased resistance and recovery induced by the introduction of novel chemotherapy, including antibiotics and sulphonamides (Arriaga and Davis 1969; Stolnitz 1965; Palloni and Wyrick 1981; Palloni and Pinto 2011; Preston 1976, 1980). Thus members of cohorts born after 1930-1940, who will attain their sixtieth birthday after the year 2000, are more likely than members of previous birth cohorts to have experienced early conditions known to be associated with higher risks of adult chronic conditions, such as COPD, heart diseases, type 2 diabetes, and other chronic conditions that dominate the landscape of adult mortality (Barker 1998; Gluckman and Hanson 2006; Langley-Evans 2004; Elo and Preston, 1992). Two factors determine whether or not these early experiences express themselves as higher incidence of chronic diseases: (a) the strength of the linkage between poor early conditions and adult chronic illnesses and mortality; and (b) the nature of the exposure to poor early conditions. Different mixtures of these factors generate variation in the impact that cohort compositional changes by early exposure can have on health status and mortality of older adults.

The downward shift in mortality experienced in LAC alters the composition of successive birth cohorts in a peculiar way: cohorts who benefit more from the mortality decline triggered by advances in medical technology will contain a larger fraction of individuals at higher risk of expressing the deleterious effects of poor early conditions. Thus, older age mortality among cohorts born more recently will reflect not just period effects but also the influence of compositional changes by early childhood exposures. The extent to which early conditions influence the old-age mortality risks of a cohort can be understood via the concept of frailty, which describes susceptibility to mortality risk whereby frailer individuals experience a higher probability of dying at any given age compared to less frail individuals.

The compositional changes associated with early conditions are similar to but distinct from changes in composition by frailty invoked by a *conventional frailty* model (Vaupel et al. 1979; Vaupel and Yashin 1987). The conventional frailty model is based on the assumption that individuals are born with a randomly drawn frailty value, γ, $(\gamma >= 1)$, such that higher values increase individual lifetime mortality relative to a standard in the same proportion at all ages. The distribution of γ is defined at birth and changes over the life of a cohort as members endowed with higher values of γ die out first. The results from this model are two. First, when the mortality regime and frailty distribution at birth are invariant the average mortality rates at older ages will increase more slowly than the individual mortality rates. Second, if the population experiences mortality declines and the frailty distribution at birth remains invariant, any improvement in mortality will increase the average level of frailty at older ages, i.e., the cohort will be, on average, more frail at all ages than it would have been in the absence of mortality changes or more frail than its predecessors. These effects will underplay the observed rates of mortality decline at all ages, but especially late in life.

The phenomenon of interest here is a result of a different type of frailty, termed *Barker-frailty*[1], which refers to changes in the composition of cohorts by exposure to early conditions that produce excess mortality risk only at older ages and only through a handful of chronic illnesses. For simplicity, assume that individuals are characterized at birth by a random Barker-frailty value, $\varepsilon >= 1$, so that higher values signify increased susceptibility to express the early-late health connection. Elsewhere it is formally

[1]The term "Barker-frailty" (and "Barker-effect") is used as a shorthand and refers to all mechanisms classic Barker mechanism) producing early-late health connections.

demonstrated that a mortality regime driven both by conventional and Barker-frailty is characterized by two properties. First, when there are neither changes in the mortality regimes nor changes in the conventional and Barker-frailty distributions, older age mortality rates will operate subject to laws of conventional frailty but mortality at older ages will be higher than expected under the conventional frailty model. This is because individuals who are scarred by early conditions, $\varepsilon>1$, and survive to older ages experience higher excess mortality at those ages than they did at younger ages. Unlike the conventional frailty factor, γ, Barker-frailty, ε, increases mortality rates relative to a standard set of rates and does so disproportionally at older ages. The net result is that the upward concavity of the mortality curve will be sharper than expected under a conventional frailty model but still duller than the concavity of the individual mortality rates.

Second, if mortality declines at all ages and frailty distributions are invariant, mortality rates at older ages could decline more slowly, not at all, or even increase as a result of changing composition of cohorts by ε, with, for example, a progressively larger influx of individuals with higher values of ε that boost mortality risks at older ages. If there were no links between early conditions and adult health and mortality, changes in cohort composition by ε will have no effects at all on older age mortality and only the impact of changes in conventional frailty composition, γ, will be observed with a slower average mortality decline than the one actually affecting individual mortality. The tighter the link between exposure to early conditions and increased older age mortality is, the stronger the departure of older age mortality rates from the regime predicted by a conventional frailty model.

The operation of Barker-frailty requires two conditions. To simplify description assume that cohorts can be divided in two groups: one composed of individuals who would have died under the pre-transition mortality regime, A, and one with individuals who would have survived under that regime, B. The first condition is that mortality levels experienced before some target age, say 60 years, are higher in group A than in group B during the initial phases of mortality decline. The second condition is that differences in mortality at older ages (over 60) between A and B are at least in part the result of exposure to early conditions whose effects manifest mostly, if not solely, late in life. We refer to this mortality excess at older ages between groups A and B as the *Barker-effect*. In most empirical cases changes in survivorship experienced during the early stages of the secular mortality decline are proportionately larger in subpopulations more likely to experience poor early conditions or higher values of ε. As a consequence, the average value of ε at any age before older ages will increase over time as a result of survival gains.

1. A simple numerical example

A highly stylized numerical example provides a sense of magnitude and some insight on the relations described above. The main features of the exercise are as follows:

(a) Individuals in the population are characterized by a latent variable ε with a Gamma distribution $f(\varepsilon)$ with parameters 2 and 1 (mean and variance equal to 2). Whenever ε exceeds some threshold value (to be determined) the individual experiences poor early conditions and can potentially express excess mortality risks.

(b) The mortality decline is defined so that the mortality rate at age y and t years after the onset of mortality decline is $\mu(y,t) = g(t)^* \mu_s(y,t)$, where $\mu_s(y,t)$ is a standard mortality rate and $g(t)$ a (positive valued) function declining on t. Individuals who belong to group A experience mortality rates equal to $\mu_A(y,t) = g(t)^* \mu_s(y,t)^* \lambda_1$ at all ages below 60 and equal to $\mu_A(y,t) = g(t)^* \mu_s(y,t)^* \lambda_2$ at 60 and above, with $\lambda_2 > \lambda_1$. Members of group B experience $\mu(y,t) = g(t)^* \mu_s(y,t)$

(c) Values of λ_2 *and* λ_1 are set as small as 1.5 and as large as 5;

(d) 100 cohort mortality "regimes" are computed, mimicking the mortality decline in the LAC region from 1941 to 2000, and then from 2000 to 2040 following United Nations mortality projections.

Figures 1a through 1d illustrate selected results of the calculations for a scenario where the fraction of individuals experiencing early conditions is about 0.20 *(ε >=3)*, λ_1 *=2* and $\lambda_2 = 4$[2] . Figure 1a shows the initial distribution at age 60 of the early conditions factor, ε, among members of group A corresponding to three mortality regimes selected from the 100 regimes computed. The first represents an "old" regime before mortality decline with life expectancy at birth around 45 years; the second is an "intermediate" regime during the decline; and the third is a "new" regime well into the decline and with life expectancy at birth around 80 years. As mortality declines from the old to the new regime, the distribution of ε shifts towards higher values indicating greater average values of Barker-frailty among group members.

Figure 1b displays survival probabilities to age 60 at under the three mortality regimes. In the lower mortality regime, small increases in ε are associated with steep declines in the probability of surviving age 60, but that association becomes less steep as mortality declines to the intermediate and new mortality regimes.

Figure 1c displays the average values of the mortality rate λ_2 at age 60 through the regimes representing mortality decline to illustrate the effects of mortality decline on compositional changes across cohorts over the period of the decline. As mortality declines, the mean values of λ_2 increase substantially reflecting extra survival among individuals born with poor prospects.

Figure 1d displays the average mortality rates for group A and the standard mortality rates for group B at ages 65 years, 75 years and 85 years over the 100 mortality regimes observed. This figure shows the ultimate outcome of changes in composition induced by mortality decline in that the average mortality among group A at the three ages does not decline at the same rates as the baseline mortality rates among group B. Instead, the declines in average mortality among group A tend to be slower, with no decline or even increases observed during some periods.

The above exercise is limited to only one of many possible scenarios but the results are more general. Indeed, one can show that if early conditions are captured by a binary variable, a mortality decline resembling the one experienced in LAC countries will produce results consistent with the main conjecture as long as the fraction in group A exceeds 0.15 and the values of λ_1 and λ_2 are larger than 1.5

2. Application to extreme cases in LAC

Using rather simple models and available empirical data one can generate estimates of bounds of the ultimate impact of Barker-effects on adult mortality in the short run. Figures 2a and 2b display the values of life expectancies at age 60 under three different scenarios for the two countries that produce extreme results, Argentina and Guatemala[3]. At the beginning of the period under examination there can be only

[2] Other scenarios lead to similar conclusions. But, of course, more benign scenarios dilute the effects under scrutiny. For example, when the proportion of individuals who are exposed to early conditions is less than 5%, Barker effects are hardly visible and distinguishable, irrespective of values of λ_1 and λ_2, from those that would be expected if only conventional frailty operates.

[3] A more thorough presentation of results appears elsewhere (Palloni and Souza, 2013)

small differences between alternative scenarios since the fraction of the total projected population 'saved' by the mortality decline is still very small. When the influx of cohorts born after 1950 into age groups over 60 begins to grow, the different scenarios start diverging and yield different life expectancies. A mild scenario (with weak early-adult health linkages, called the "low-relative risks" scenario in figure 2) applied to Argentina produces differences of the order of 0.1 to 0.4 years of life expectancy at age 60. A harsher mortality regime among those exposed to deleterious conditions (strong early-late linkage, called the "high-relative risks" scenario in figure 2) leads to larger differences, about 0.66 years of life expectancy. These values are small since Argentina's mortality decline began very early in the twentieth century, twenty to thirty years before significant medical innovations were introduced. As a consequence, the population saved after 1950 and exposed to Barker effects is small and exposure to excess adult mortality is minimized.

In contrast to Argentina, Guatemala is a country more typical of those that started the mortality decline after 1950 and the potential losses in life expectancy at age 60 are larger. A low relative risks set of parameters produces differences of about 0.80 years. But when those exposed to adverse early conditions are subjected to a high relative risks mortality regime, the differences are as large as 1.5 years or close to 8 per cent of the total life expectancy at age 60.

Overall, these are not massive differences but neither are they trivial. Consider the following: during the period 2010-2050 Guatemala is projected to add 2.3 years of life expectancy at age 60; thus, the potential losses calculated above are about half of the total projected gains. In Argentina the potential losses are, on average, one sixth of the expected gains. By the same token during the period 1980-2000 life expectancy at age 60 increased by about 5 years. Figures 2a and 2b reveal that as little as 8 per cent and as much as 20 per cent of this gain can be forfeited in a span of 50 years solely as a function of changes in cohort composition by exposure to early conditions. And yet, figures 2a and 2b correspond to an optimistic scenario, one where an entire section of the population that includes individuals who would have survived in the absence of a mortality decline are spared exposure to early conditions and any excess mortality associated with them. This is a rather extreme assumption and surely leads to under-estimates of the effects we seek to identify. Thus the theoretical 'losses' displayed in figures 2a and 2b must be considered as conservative, at least within the framework proposed here.

C. SMOKING 'EPIDEMICS' AND EXPECTED EXCESS FUTURE MORTALITY

According to a standard typology (Lopez et al., 1994; Ezzati and Lopez, 2004) countries in LAC span a broad range of experiences in the smoking epidemic, from those in the mature stages (Argentina, Chile, Cuba, and Uruguay), to those with more recent onset (Mexico and Brazil). Argentina, Cuba, Chile and Uruguay have higher rates of smoking than males in the United States, whereas Brazil and Mexico have lower rates. Cuba stands out as an outlier with the highest prevalence of smoking, which is reflected in the country's highest excess adult mortality associated with smoking.

As expected given patterns observed in other countries and regions, female smoking prevalence in LAC lags behind male smoking prevalence, but despite this lag female smoking has already grown close to 20 per cent in some populations (Argentina and Chile) and will become increasingly influential on female mortality risks in the near future. The age-specific smoking prevalence rates in LAC are highly heterogeneous and reflect characteristics that are typical of different stages of the epidemic. They also reveal surprising anomalies that are relevant for the future adult mortality: an exceptionally high prevalence among the population younger than age 25 in Chile, signifying a recrudescence of the smoking epidemic; and unexpectedly low levels of youth smoking in Brazil, an indication of successful anti-smoking campaigns. [4]

[4] See Palloni et al., 2013

What are the mortality implications of past trends in smoking behaviours? We can estimate with high precision the connection between aggregate smoking prevalence and smoking-related attributable mortality and the estimates can be converted into number of years of life lost due to smoking over the past 20 to 30 years. Using a number of techniques described elsewhere (Palloni et al., 2013a) researchers estimate counterfactual mortality trends that would have been observed if smoking-attributable mortality were set to zero. The main results are in figure 3, which displays trends in observed and counterfactual life expectancy at age 50 for the period 1950-2010 in six LAC countries. In all countries included in the figure, differences between counterfactual and observed mortality are larger for males than they are for females, consistent with the fact that in most countries female smoking has not yet left a powerful imprint on mortality. Note, however, that the disparities between the two female regimes are increasing over time.

Among males the differences between observed and counterfactual trends in the life expectancy at age 50 are large: between four and six years in Argentina, Cuba and Uruguay (about 20 per cent of their current life expectancy at age 50), somewhat less than four years in Chile (about 15 per cent of its current life expectancy at age 50), and less than two years in Brazil and Mexico (about 8 per cent of their life expectancy at age 50). During the most recent period (2000-2005), absolute and relative differences between observed and counterfactual life expectancies have been steady or increasing slightly in Argentina and Uruguay but have systematically increased in Cuba, Brazil and Mexico. The case of Cuba is a glaring example of the damaging power of smoking behaviour: the foregone gains in adult male life expectancy due to smoking (about 20 per cent to 25 per cent of the current value of life expectancy at age 50) are equivalent to the *observed total gains in survival the country experienced in the last 20 years*. In other words, it took twenty years of unprecedented progress against infectious (and some chronic conditions) to offset losses that smoking was silently inflicting during the same period of time. The bulk of the differences between counterfactual and observed life expectancies everywhere is associated with diseases of the heart and circulatory system as well as cancers other than lung. The remainder of the difference is associated with lung cancer.

The smoking epidemic in LAC is well established, but countries are navigating through its various stages with different timings and at different pace. As a consequence, the health and mortality effects are highly diverse. But the threat of large impact on mortality trends is there all the same, particularly in countries where rapid and pervasive smoking uptake began more recently. Estimates suggest that during the decades 1980-2000 the impact of smoking is equivalent to losses in male life expectancy at age 50 of 2 to 6 years. To date these losses are only virtual, not observed. They represent how much higher life expectancy at age 50 could have been in the absence of smoking. And although making forecasts is always risky, there are two regularities about which one should be certain. First, current male and female smoking prevalence between ages 20 and 50 in countries with high prevalence of smoking will inevitably translate into virtual losses of the same or higher magnitude than those calculated above. No sophisticated forecasting machinery is needed to predict this since smoking behaviour has a substantial momentum that takes years to disappear. Second, given patterns of smoking uptake in countries cruising through the early stages of the smoking epidemic, one should expect the geographic spread and replication of foregone gains in longevity already experienced by countries that started much earlier.

Will smoking behaviour eventually lead from virtual to real losses in adult life expectancy? Or will the damage only derail future gains, as it has done up to now? Or, finally, will it become totally irrelevant? Uncertainty about these issues is rooted in two areas. The first is the march of the smoking patterns themselves: will countries now at the early stages contain it with timely and successful public health interventions? Will countries in more advanced stages converge rapidly to conditions under which smoking is confined to small pockets of population? The second, and the one that generates most of our

uncertainty about the future, is the effect of smoking on mortality: Can we optimistically count on a steady stream of future medical innovations for treatment and detection that may alter the age pattern of fatality rates of non-smoking-related and smoking-related diseases? In LAC there is some, albeit scarce, room for offsetting effects originating in further reduction of infectious diseases. Only attenuation of mortality risks due to chronic diseases can oppose effective resistance to the heavy tug of smoking histories. What we do not know is whether this offsetting force will emerge at all and, if it does, whether it will be sufficiently large and arrive soon enough to blunt the effects of the physiological damage already inflicted by past smoking.

D. OBESITY, CHRONIC ILLNESSES AND EXCESS FUTURE MORTALITY

It is suspected, but not yet well established, that lifestyle changes embraced by recent cohorts of adults in both high- and low-income countries could oppose strong resistance to further improvements in longevity (Olshansky et al. 2005; Flegal et al., 2013). While the smoking epidemic offers an example of behavioural changes that are already shaping mortality trends at older ages, the implications of what has been termed the "obesity epidemic" appear to be a bit more remote. Increasing prevalence of obesity is an outcome, among other things, of recent behavioural adaptations that affect children and adults alike. These may have large effects on health status, mortality, and health care expenditures (Aschner 2002; Kain, Vio and Albala 2003; Barcelo et al., 2003) In this section we estimate expected losses in adult life expectancies associated with current levels and past trends of obesity prevalence. The estimates pertain to only one LAC country, Mexico, for which we have available highly accurate estimates both of the levels of obesity and of the linkage between obesity, chronic diseases, and excess mortality.

According to a recent report issued by the United Nations Food and Agriculture Organization (FAO, 2013), Mexico's prevalence of obesity among adults (33 per cent) surpassed that in the US (31 per cent) and became the country with the highest rate of obesity in LAC[5]. Even if one allows for statistical inaccuracies and definitional ambiguities, Mexico unambiguously ranks in the first quintile of the obesity distribution in LAC and North American countries. The country attained this top ranking riding on a trend of very rapid growth: ten years ago the prevalence of obesity was estimated to be about 70 per cent of what it is now, implying a doubling time for obesity prevalence of only 19 years. Recent research suggests that among adults this state of affairs is likely to get worse before it gets better since the rates of early obesity are still climbing at all ages and early obesity is a precursor of adult obesity. Indeed, forecasts for a number of Latin American countries currently doing better than Mexico predict surges of adult obesity prevalence to levels as low as 50 per cent and as high as 80 per cent (Webber, Kilpi et al. 2012). The factors that explain the rapid ascent of Mexico to the top of the weight distribution are still a matter of controversy. Some are associated with the role of poverty and sudden shifts in diet (Razak, Corsi et al. 2013); others involve sharply reduced levels of physical activity (Webber et al. 2012, Razak et al. 2013), and still others invoke the existence of interactions between these two, environmental exposures and genetic traits (Popkin 2004). What is not in doubt is the load of chronic disease that accompanies the increase of obesity prevalence, namely, elevated risks of pre-diabetes and type 2 diabetes (Allison and Saunders 2000), dyslipidemia (Terry et al. 1989), coronary heart disease (Eckel and Krauss 1998), sleep apnea (Mitchell et al. 2011), cognitive dysfunction (Gunstad, Paul et al. 2007, Boeka and Lokken 2008, Fergenbaum et al. 2009), cancer (Reeves et al. 2007, Renehanet al. 2008), and liver and kidney disease (Matteoni et al. 1999).

[5] We use the following, conventional categories: underweight are those with BMI<18.5; normal are those with 18.5<=BMI<25; overweight are those with 25<= BMI<30 and obese are those with BMI>=30And Definition of categories of obesity

What follows is an account of the estimation of excess mortality due to obesity among adults in Mexico during the period 2000-2012. The main data set on which estimation is based is the Mexican Health and Aging Survey (MHAS), a three-wave panel, modelled after the U.S. health and retirement Study (HRS). MHAS has followed a representative sample of Mexican adults aged 50 and older and their spouses since 2001. The estimation described below uses all the information from the first wave (2001), second wave (2003) and third wave (2012). The baseline sample consisted of 15,402 interviews carried out during 2001. Respondents were selected following the National Employment Study sampling scheme, conducted by the Instituto Nacional de Estadistica, Geografia e Informatica (INEGI), which allowed coverage of both urban and rural residents in all 32 Mexican states. States with high emigration rates to the United States were oversampled. Interviews were conducted in-person by INEGI professional interviewers who were trained by MHAS personnel and INEGI supervisors to secure appropriate follow-up. Field supervisors also administered various anthropometric measures, including height, weight, knee height, and hip and waist circumference, to a 20 per cent random subsample. Two follow-up interviews, with surviving respondents were conducted during 2003 and 2012. New spouses and partners were also included in the subsequent waves. The second wave consists of 14,386 respondents and a new sample of 220 new spouses. The third wave is made up of 12,569 respondents and a new sample of 5,896 new subjects and spouses. Next-of-kin proxy respondents reported 546 deceased respondents from the 2001 baseline and 2,742 deceased from the 2003 wave. For more details on MHAS see http://www.mhasweb.org/

The model representing the associations between obesity, chronic conditions and mortality is shown in figure 4. We estimate direct (and indirect) effects through a combination of models. First, to estimate α, the association between obesity status and morbid conditions, we use conventional logistic models for type 2 diabetes, cancer, and CVD incidence over a ten year interwave period. Second, modified Gompertz models are employed to estimate values of γ, the association between obesity status and mortality, and β, the association between morbid conditions and mortality (with and without unmeasured heterogeneity). These estimates are then combined to compute estimates of the excess mortality attributable to obesity only, net of smoking behaviour.

1. Obesity and chronic conditions

The first two columns of table 1 display estimates of logistic models for the incidence of diabetes (waves 1 to 2; 1 to 3; and 2 to 3) and of any of three chronic illnesses (type 2 diabetes, cancer and heart diseases) using as predictors the indicators of obesity from self-reported measures. The probability of contracting type 2 diabetes in the interwave period between 2001 and 2012 is between 44 per cent and 56 per cent higher for those who are obese at baseline relative to those with normal body mass index (BMI) and between 36 per cent and 42 per cent higher for those who are overweight. Slightly higher increases of incidence of any chronic diseases are associated with overweight and obesity (column 2). These relations are preserved in the models that use objective measures of overweight and obesity (columns 3 and 4). These estimates point to large effects that translate into considerable health care costs and losses to labour productivity (Barcelo et al. 2003, Abegunde et al. 2007). But these costs surely pale relative to the loss of life implied by very large excess mortality associated with type 2 diabetes and with other chronic conditions triggered by obesity. [6]

[6] The effects of obesity on diabetes incidence estimated here are effects over a ten-year period of exposure and are akin to effects on the *cumulated* conditional incidence function , not on the yearly risk.

2. Obesity, chronic conditions and excess mortality

The fully parametric Gompertz model to estimate mortality as a function of obesity and chronic conditions has the following form:

$$\mu (x_o + t) = \lambda \exp((x_o - 50) + t)\gamma)$$

where x_o is age at first wave, t is the duration since first wave, λ is the Gompertz level parameter and γ is Gompertz's slope. The Gompertz function is only a good approximation to mortality at ages over 50 and we rescale age to be the difference between age at first wave and 50. It is clear from the above equation that the regression coefficient associated with the rescaled age variable (x_o -50) in the proportional hazard formulation must be constrained to equal the slope, φ. Finally, note that λ represents the mortality rate at age 50. Throughout, the parameter λ is defined as a function of covariates, including controls for age, gender, and a dummy for education as well as those of interest to us namely, self-reported conditions, particularly diabetes, cancer and heart diseases. The most important estimates of effects of obesity and selected chronic conditions appear in table 2.

3. The combined impact of obesity on chronic diseases and mortality

We now combine estimated excess mortality among diabetics with the estimated risk of becoming diabetic given obesity at baseline and compute the overall effects of obesity on mortality. The excess mortality attributable to obesity (overweight and obese) that is mediated by diabetes is equivalent to p(d|o)(e-1)), where p(d|o) is the conditional probability of diabetes among the overweight and obese and e is the excess mortality among diabetics. With a p(d|o) ~0.20 , as in our sample for those aged 50, the excess mortality due to diabetes only is of the order of 1.17. A similar calculation but including three diseases (diabetes, cancer and heart diseases) yields an excess mortality among overweight and obese individuals of about 1.20 (0.23*0.86). If overweight and obesity prevalence among those older than 50 is of the order of 69 per cent (as in our sample), the overall excess mortality attributable to effects operating through diabetes only is 1.12 whereas the excess operating through all three chronic diseases is 1.15. If one insists on accounting for potentially favourable direct effects of obesity, the mortality excess calculated above should be reduced by approximately 0.19*0.42 = 0.08 where 0.19 is the reduction in mortality among overweight (the "apparent" direct effect) and 0.42 is the fraction of the population over 60 who is overweight in our sample.

If the prevalence of adult overweight and obesity grows to 75 per cent, as could happen in the next few years, the expected excess mortality associated with diabetes and all three chronic conditions will be 1.21 and 1.25 respectively. This is a sizeable mortality excess, equivalent to losses of about 3 to 4 years of life expectancy at age 50 (about 13 per cent), about one year more than the losses due to cigarette smoking. These figures should prompt a reconsideration of the idea that obesity has ambiguous or even beneficial effect on survival at older ages. As smoking has done in the past and will continue to do in the future, obesity has had and will continue to have a strong impact on health status, health care, health costs, and will gradually erode or exert strong resistance to future improvements in longevity.

To sum up, the effects of obesity that operate through diabetes only will raise mortality by close to 17 per cent among overweight and obese individuals and, at current obesity prevalence, by about 12 per cent in the total population. These mortality excesses represent losses of life expectancy at age 50 of about two to three years, or 9 per cent to 12 per cent of current values of life expectancy at age 50 in Mexico. Thus, the magnitude of the mortality impact of obesity estimated for Mexico is inching closer to

and exceeding the mortality impact of cigarette smoking elsewhere in LAC, at least among countries navigating the middle stages of the smoking epidemic[7].

E.CONCLUSION

An overview of potential future changes in adult health and mortality should include consideration of shifts in cohorts' composition by traits known to be associated with health and mortality risks. This is in in contrast to a perspective that relies on the premise that all important changes in mortality are due to period events and that the influence of cohort-specific determinants is overwhelmed by period factors and need not be considered in any depth. While future mortality trends depend in part on the unfolding of events that we may anticipate but know little about—such as sudden climate change, emergence of new diseases and re-emergence of old ones, a burst of innovation in medical technology—a balanced perspective should include an account of changes that we can anticipate with improved accuracy because they depend strongly on events we have already been observed and on linkages between exposure to risks and mortality that are well established.

This perspective may have benefits in two areas. First, it can provide materials to improve mortality forecasts. The extent to which it does so will depend on the accuracy of estimates of cohort composition by relevant traits and of excess mortality associated with those traits. Second, it can inform better the design of health policies that will be more suitable for and responsive to a changing landscape of health status.

It is argued in this paper that there are at least three known forthcoming shifts in cohort composition that may become influential in low- and middle-income countries. The first consists of changes by the intensity and type of exposure to risks affecting infancy and early childhood. The second involves past trends of smoking uptake and desistance among adolescents and young adults. The third is associated with trends in early and late adult obesity and its implications for the emergence and persistence of chronic illnesses and their sequelae. Although these are neither the only nor the most important cohort changes that could be experienced, they share a commonality: all three involve the presence of traits in several cohorts, the changing composition of the cohorts by the presence of the traits, and relatively well-known linkages between the traits and excess health or mortality risks.

The estimates discussed here suggest that all three cohort shifts are consequential as they could account for virtual losses of life expectancy at age 50 in the range of three to seven years of life. For a number of reasons examined in the paper, these figures may be lower bounds and could contain substantial downward biases. Furthermore, the estimates are related to mortality risks only and ignore implications for changes in health status composition as well as computations of health care costs. By the same token, estimates were derived from a small and admittedly not representative subset of low- and middle-income countries where conditions may be superior to those experienced in other peer countries.

The document avoids altogether the intriguing but knotty matter of interaction between the three conditions. We simply do not know if and how the combination of exposures to early conditions, smoking uptake, and obesity may combine to produce health outcomes and mortality risks. Ignoring the issue could lead to substantial underestimation of health and mortality risks.

[7] For reasons discussed elsewhere it is almost certain that the estimates of excess mortality due to obesity are lower bounds and contain a sizeable downward bias

While the discussion above points towards potential damage that could be larger than what the estimates suggest, there is an important caveat that may void the warning. All three traits ultimately lead to excess risks associated with a handful of conditions, including cancer to selected sites, COPD, type 2 diabetes and cardiovascular diseases. Although these are not the only critical chronic conditions that dominate the modern adult health landscape they are certainly important. However, their impact on mortality may be attenuated considerably with a burst of innovative medical advances and the sudden development and efficient dissemination of technology for the detection, treatment and management of these conditions. Technological innovation and dissemination is plausible but should not be factored in as a realized fact in mortality projections or policy designs. Instead, the expected impacts of changes in composition by early conditions, smoking and obesity should be included since we know within reasonable certainty what the expected effects will be.

Figure 1a. Proportional distribution at age 60 of early condition factor in group A for three mortality regimes representing declining mortality over time

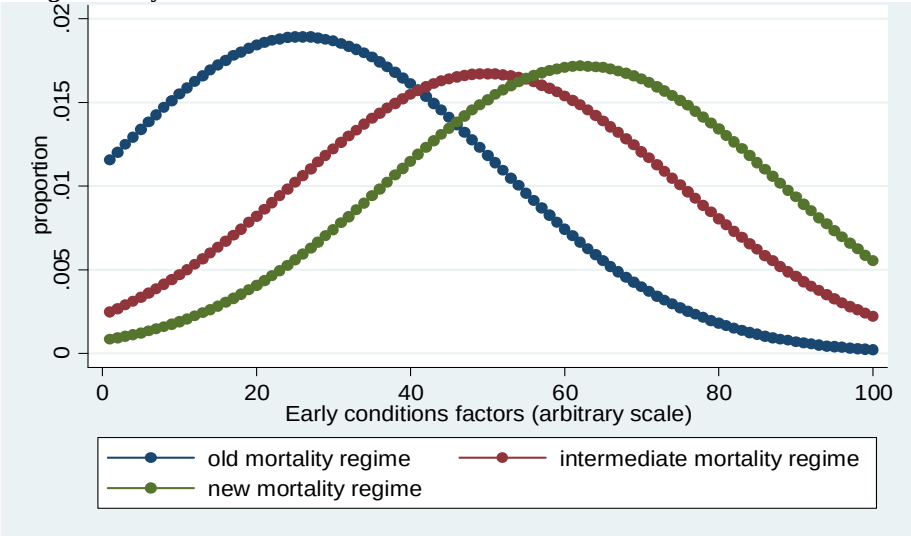

Figure 1b. Probability of surviving to age 60 by early condition factor for three mortality regimes representing declining mortality over time

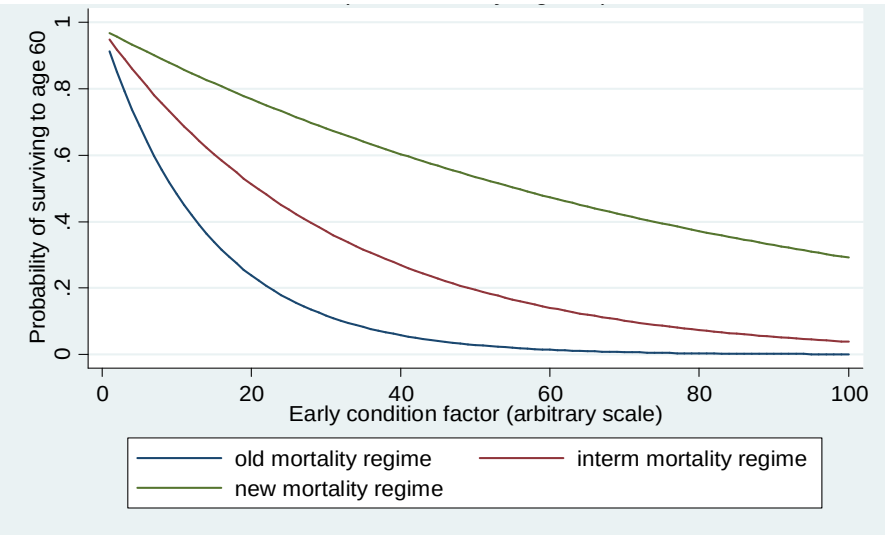

Figure 1c. Average mortality rate at age 60 by mortality regimes representing declining mortality over time

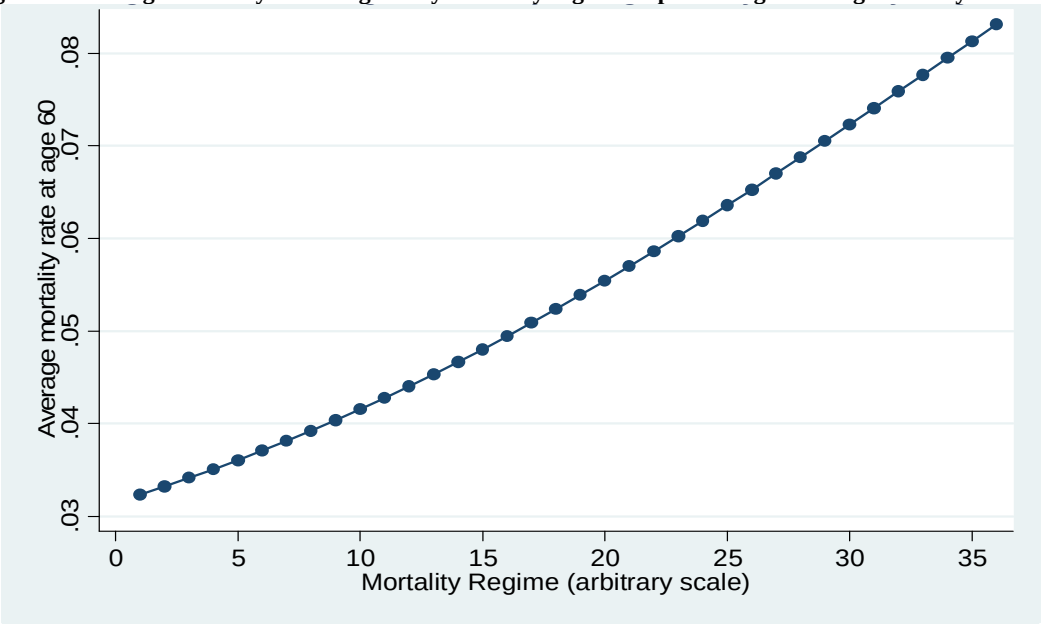

Figure 1d. Difference between average mortality in group A and baseline mortality in group B in the context of declining mortality over time

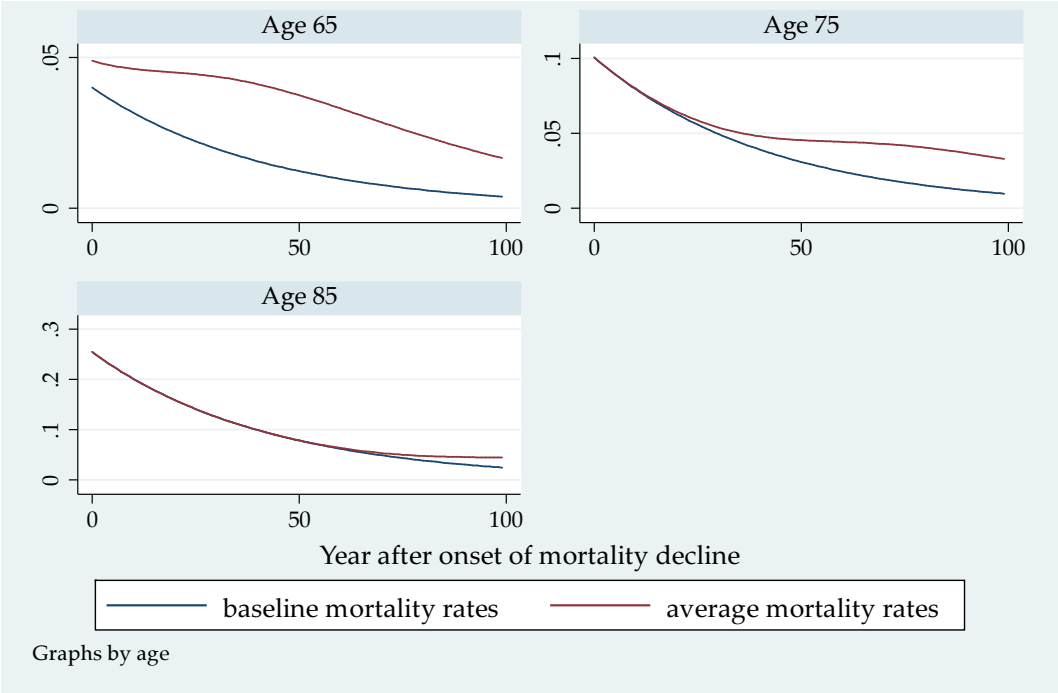

Figure 2a. Life expectancy at age 60 (in years) projected under three scenarios describing the strength of the association between early life conditions and mortality risks at older ages, Argentina, 2010-2045

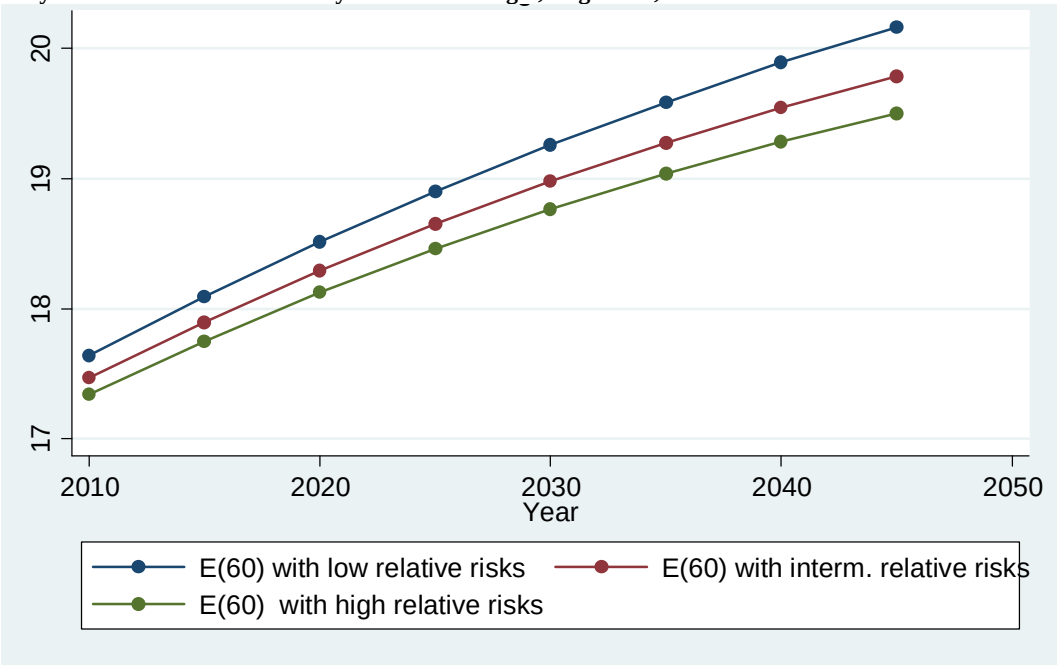

Figure 2b. Life expectancy at age 60 (in years) projected under three scenarios describing the strength of the association between early life conditions and mortality risks at older ages, Guatemala, 2010-2045

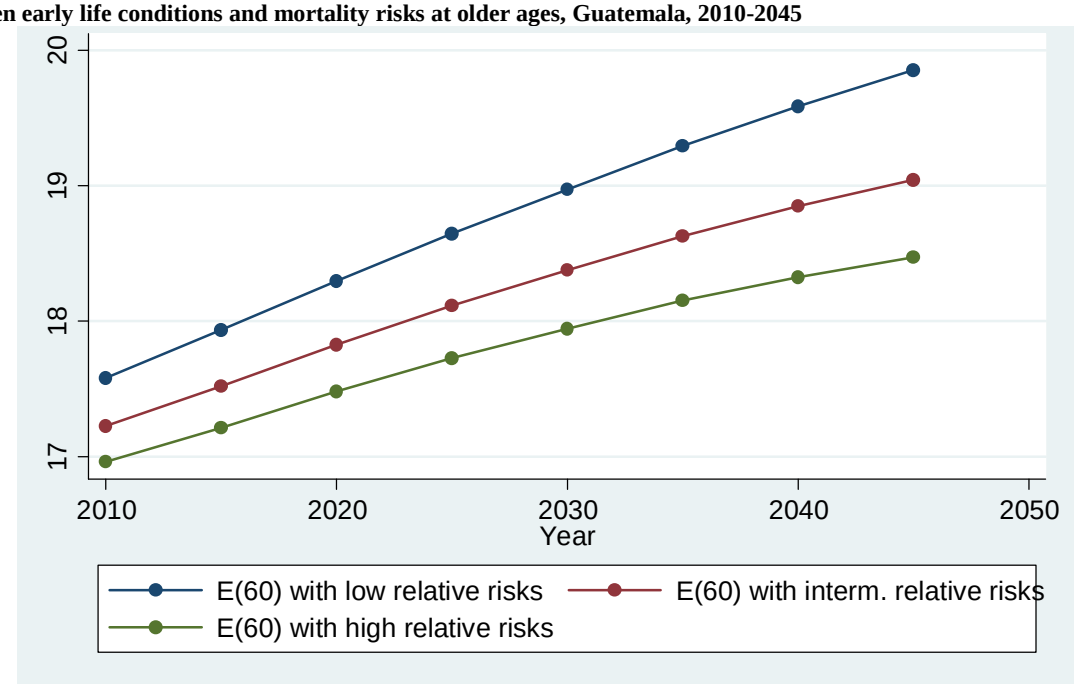

Figure 3. Life expectancy at age 50 (in years) observed and estimated as a counterfactual in the absence of smoking, males and females in six countries in Latin America and the Caribbean, 1980-2010

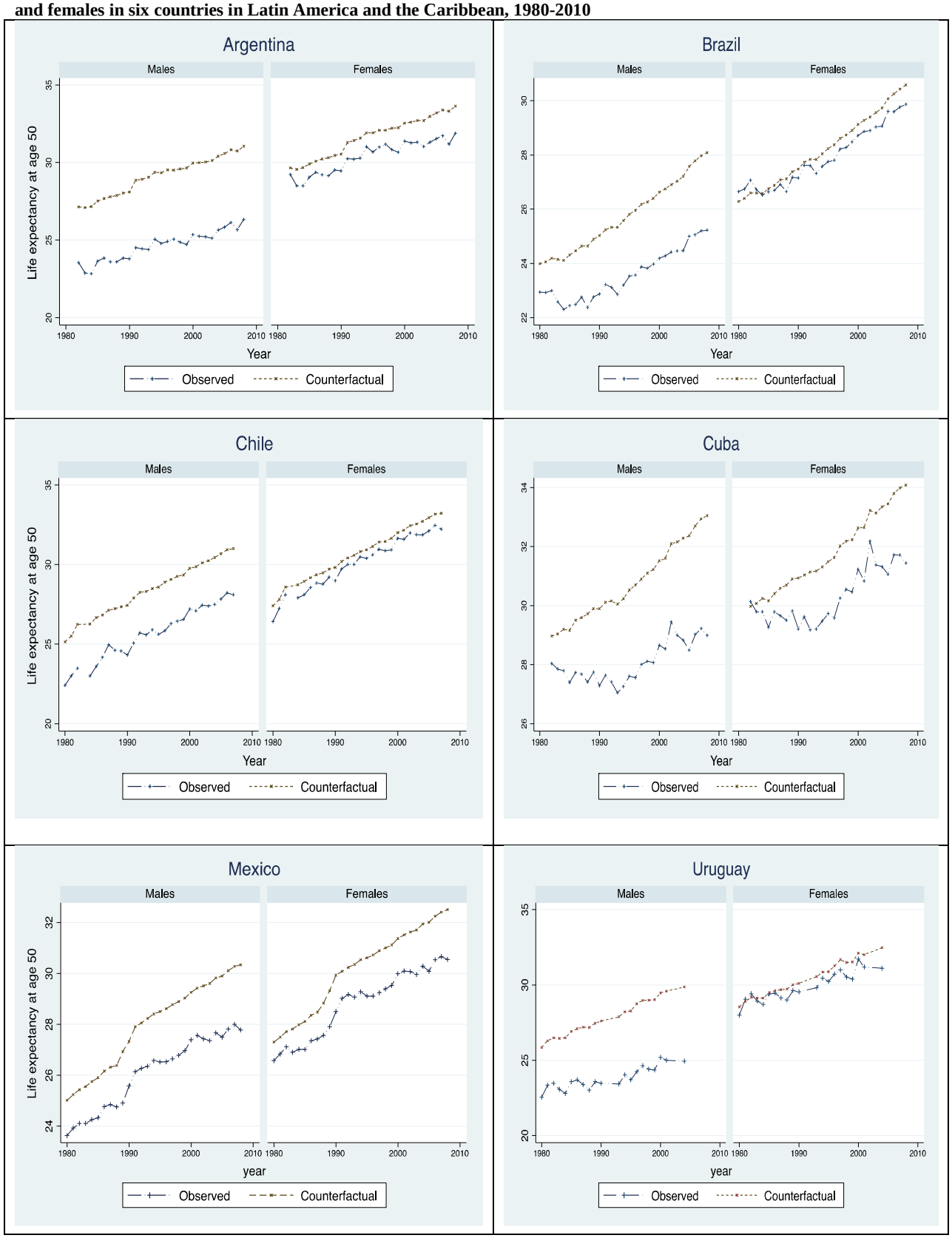

Figure 4. Model of relationships between obesity, morbidity and mortality

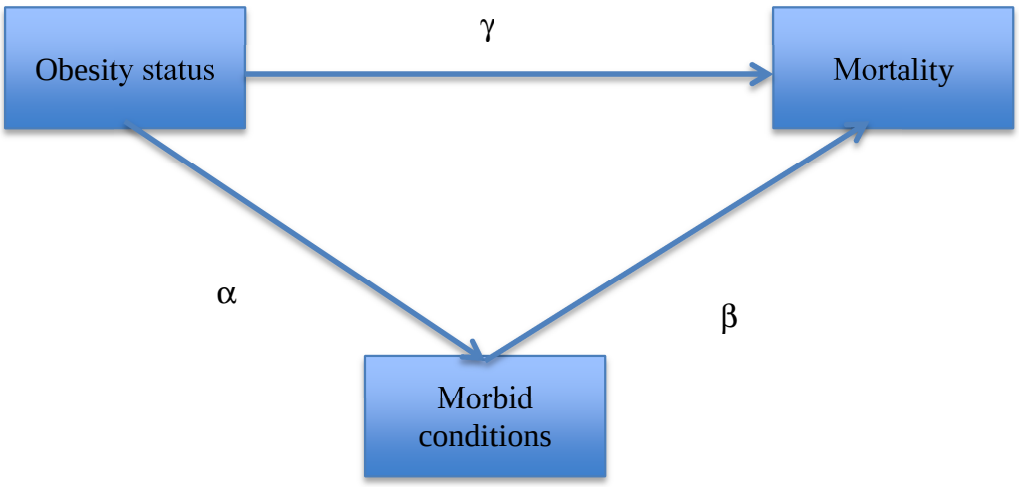

TABLE 1. LOGIT MODELS FOR INCIDENCE OF DIABETES AND ANY CHRONIC ILLNESS: 2001-2012 [1]

	(1)	(2)	(3)	(4)
	Diabetes	Any chronic illness	Diabetes	Any chronic illness
	Coefficient (standard error)	Coefficient (standard error)	Coefficient (standard error)	Coefficient (standard error)
Log(age)	0.16*** (0.03)	0.21*** (0.03)	0.13 (0.07)	0.17** (0.06)
Gender	-0.28*** (0.06)	-0.23*** (0.05)	-0.31* (0.13)	-0.29* (0.12)
Education	-0.29*** (0.06)	-0.16** (0.06)	-0.23 (0.16)	-0.03 (0.15)
Underweight (self-reported)	-0.50* (0.24)	-0.31 (0.20)		
Overweight (self-reported)	0.19** (0.07)	0.11 (0.06)		
Obese (self-reported)	0.35*** (0.07)	0.34*** (0.07)		
Underweight (objective)			0.38 (0.48)	0.10 (0.48)
Overweight (objective)			0.22 (0.16)	0.23 (0.15)
Obese (objective)			0.33 (0.18)	0.24 (0.17)
constant	-1.81*** (0.09)	-1.68*** (0.09)	-1.84*** (0.22)	-1.69*** (0.21)
N	8936	8926	1645	1642
AIC	8314	9332	1517	1708
BIC	8364	9381	1555	1746
Log Likelihood	-4150	-4659	-751	-847

(1) Case completed sample; Standard errors in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

TABLE 2. GOMPERTZ HAZARD MODELS AND CHRONIC ILLNESSES [1]

	Model (1)	Model (2)	Model (3)	Model (4)	Model (5)	Model (6)
	Coefficient (standard error)	Coefficient (standard error)	Coefficient (standard error)	Coefficient (standard error)	Coefficient (standard error)	Coefficient (standard error)
Age	0.07***	0.07***	0.08***	0.08***	0.07 ***	0.08***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Gender	0.44***	0.44***	0.44***	0.45***	0.30***	0.44***
	(0.05)	(0.05)	(0.07)	(0.07)	(0.04)	(0.07)
Education	-0.11	-0.10	-0.17*	-0.16	-0.20***	-0.16*
	(0.06)	(0.06)	(0.08)	(0.08)	(0.06)	(0.08)
Underweight	0.42	0.45			0.42	
	(0.13)	(0.13)			(0.13)	
Overweight	-0.23**	-0.22**			-0.23	
	(0.058)	(0.058)			(0.058)	
Obese	-0.133	-0.129			-0.13	
	(0.07)	(0.07)			(0.07)	
Diabetes	0.62***	0.62***	0.55***	0.56***		
	(0.06)	(0.06)	(0.08)	(0.08)		
Cancer	0.79***		0.65**			
	(0.14)		(0.21)			
Heart	0.18		0.24			
	(0.11)		(0.14)			
At least one chronic illness					0.62***	0.55***
					(0.05)	(0.07)
constant	-5.75***	-5.75***	-6.01***	-6.00***	-5.75***	-6.00***
	(0.09)	(0.09)	(0.10)	(0.10)	(0.09)	(0.10)
Gamma	0.07***	0.07***	0.08***	0.08***	0.07***	0.08***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
AIC	9233	9275	5348	5368	9229	5350
BIC	9327	9349	5411	5413	92	5394
N	10908	10914	7852	7858	10910	7853
Log Likelihood	-4607	-4629	-2667	-2679	-4613	-2670

(1) Case completed sample; Standard errors in parentheses ; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

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