The Tide to Come

Elderly Health in Latin America and the Caribbean

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This article introduces a conjecture and reviews partial evidence about peculiarities in the aging of populations in Latin America and the Caribbean (LAC) that may impact future elderly health status. Using Survey on Health and Well-Being of Elders data (SABE; $n = 10,902$), the authors estimated effects of early childhood conditions on adult diabetes and heart disease. Using Waaler-type surfaces, the authors obtained expected mortality risks for SABE and also U.S. elderly (Health and Retirement System, $n = 12,527$). Expected mortality risks using Waaler-type surfaces among elderly in LAC reflected excesses supporting our conjecture. There was partial evidence of a relation between various indicators of early childhood nutritional status (knee height, waist-to-hip ratio) and diabetes and even stronger evidence of a relation between rheumatic fever and adult heart disease. There is some evidence, albeit weak, to suggest that the conjecture regarding elderly health status' connection to early conditions has some merit.

Keywords: *Childhood conditions; diabetes; heart disease; Latin America; health; health outcomes; nutritional status; mortality*

Birth cohorts who reach age 60 and older after 1990 in the Latin American and Caribbean (LAC) region are unique in that they are largely the product of medical interventions that increased childhood survival largely in the absence of significant improvements in stan-

dards of living. It is estimated that between 50% to 70% of the mortality decline that took place after 1945 was associated with medical interventions (Palloni & Wyrick, 1981; Preston, 1976). The remaining decline was probably associated with better standards of living, increased knowledge about exposure and resistance to illnesses, and assorted other factors. Furthermore, a large fraction of these gains were concentrated early in the life of individuals, between birth and age 5 or 10.

IMPLICATIONS OF PAST MORTALITY DECLINE

The pattern of mortality decline just described has an important and not well-known, or at least well-noted, implication. This is that the revolution that produced unprecedented gains in life expectancy half a century ago is a powerful driver of the current growth of the older population and will remain so for a time to come.

Just as the natural rate of increase expresses the proportionate change in the size of a population between two points in time, so age-specific rates of increase express the proportionate change of the size of the population in an age group between two points in time, t and $t + dt$. A number of inferences can be derived from this elementary fact (Horiuchi & Preston, 1988; Preston & Coale, 1982; Preston, Himes, & Eggers, 1989). The most important regularities can be summarized thus:

$$R(60+, t) = r_B(60, t) - I(60, t) - J(60, t) \qquad (1)$$

and

$$RF(60+, t) = r(60+, t) - r(t). \qquad (2)$$

Equation 1 is for the rate of increase of the population aged 60 or older, $R(60+, t)$. The rate is a function of $r_B(60, t)$, the rate of growth of births for the cohorts of births born between years $(t - x)$ and $(t + dt - x)$, $I(60, t)$, the sum of differences between the age specific mortality rates from age 0 to age x experienced by the two cohorts and, finally, $J(60, t)$, the sum of differences between mortality rates older than 60 for the cohorts aged 60 or older at time t and cohorts aged 60 or older at time $t + dt$. Equation 2 is for $RF(60+, t)$, the rate of increase in the fraction of the population aged 60 or older or $C(t)$. It is simply defined by subtracting $r(t)$, the rate of increase of the entire population.

The most important consequence of Equation 1 is that upward pressure on the absolute size of the population 60+ during the time interval $(t, t + dt)$ will occur because of any combination of three factors: cohorts reaching age 60 at time t experience improved mortality before reaching their 60th birthday (increases in the probability of surviving to age 60), do so thereafter (increases in life expectancy at age 60) or, alternatively, the size at birth of the cohort that reaches its 60th birthday between t and $t + dt$ is larger than the preceding one. $J(60, t)$ is entirely because of changes in mortality conditions at older ages, whereas $I(60, t)$ is determined by improvements in mortality in early childhood and, to a lesser extent, by improvements in mortality at adult ages. Finally, $r_B(60, t)$ is solely dependent on past fertility. Instead, Equation 2 suggests that the rate of increase in the proportion of the population older than 60 also depends on the total rate of increase of the population.

The key inference from these expressions runs counter to popular beliefs: It is that the demographic dynamics of the current and future elderly population is mostly a function of past developments in mortality and fertility and depends only partially and to a much lesser extent on mortality conditions at older ages and on current fertility. It is only the rate of growth of the fraction of elderly population that depends on current demographic parameters. The rate of increase of the elderly population, on the other hand, is entirely determined by past

mortality and fertility changes and only marginally by changes in survival at older ages.[1] More concretely, the trajectory of $R(60+, t)$ during the period 1990 to 2025, for example, will depend on three determinants:

1. Determinant 1, $r_B(60, t)$: changes in fertility during 1930 to 1965
2. Determinant 2, $I(60, t)$: changes in mortality before age 60 during 1930 to 1965
3. Determinant 3, $J(60, t)$: changes in mortality after age 60 during 1990 to 2025

With a handful of exceptions, all countries in the region experienced high fertility levels (Total Fertility Rate [TFR] above 5.0) on or before 1950 and large mortality declines beginning within the period 1930 to 1940 but particularly after 1950. Between 1950 and 1965 to 1970 and for reasons that are yet not altogether clear, some of these countries experienced moderate increases in fertility. Countries such as Argentina and Uruguay are oddities because they start out with relatively low levels of fertility (TFR's around 4). In Chile, Cuba, and Costa Rica, fertility begins to decline slowly between 1930 and 1940 but the onset of large and massive drops in fertility in these countries occurs only after 1950 and after 1975 in the remaining countries of the region.

Two consequences of these trends for the aging process are worth noting. First, cohorts attaining their 60th birthday between 2000 and 2025 are inflated by the mild but ubiquitous surge of fertility of the years 1950 to 1970. Thus, the rate of increase of the age group 60 or older will increase in part because of these transient spikes in fertility levels (Determinant 1). Second and most importantly, cohorts that attain their 60th birthday between 2000 and 2025 are beneficiaries of unusually large improvements in survival, particularly during early childhood. Thus, for example, individuals born in 1960 experienced lower levels of early child mortality than those born in 1955. This will increase the relative size of the cohort attaining age 60 in 2020 relative to cohorts that reach age 60 in the year 2015 (Determinant 2).

Based on adjusted historical series of birth rates and life tables, we estimate the magnitude of the component of growth of the population aged 60 and older associated with past mortality decline. Figure 1 dis-
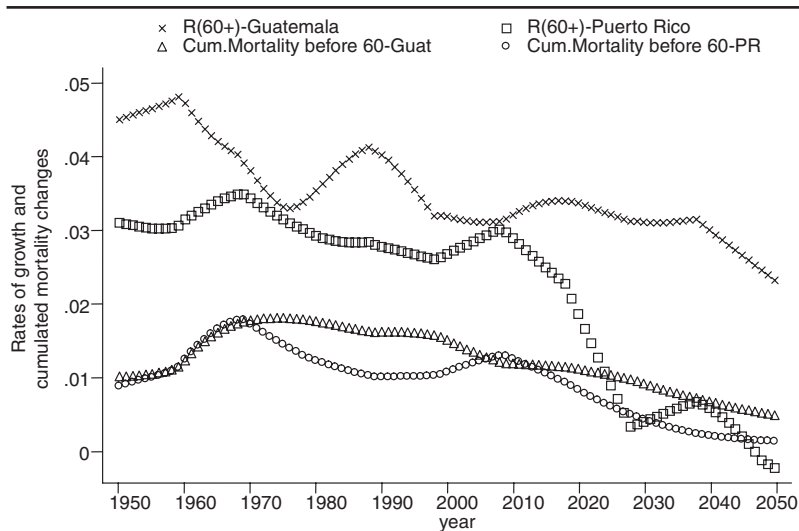
Figure 1. **R(60+) and culmulated mortality changes.**

plays the total rate of growth of the population older than 60 and the amount of that attributable to cumulated changes in mortality before age 60 for cohorts that will attain age 60 during the interval 1950 to 2050. We estimate these quantities for Puerto Rico and Guatemala, two countries that exemplify extremes in the demographic transition: The former experiences early mortality and fertility decline, whereas the latter experiences late declines in mortality and fertility. Similar results are obtained if we examine patterns in other countries (Palloni & Peláez, 2002).

Because the bulk of mortality decline, particularly during early childhood, occurs during the post–World War II years, the peak of rates of growth and of the contribution of cumulated mortality changes before age 60 are attained by cohorts born anywhere between 1940 and 1960 or those who begin to reach age 60 on or after 2000. The aftermath of the mortality decline will begin to be washed away only after 2010 in Puerto Rico and more than a decade later in Guatemala. Note that the contribution of mortality changes to the growth of the population older than 60 is substantial and exceeds 50% for several years after 2000. Other countries in the Latin American and

Caribbean region will experience population dynamics that fall within the range set by Puerto Rico and Guatemala.

The main idea conveyed by Figures 1 is this: A substantial fraction of future increases in $R(60+, t)$ and, therefore, of the aging reflected in changes in the proportion of the population older than 60 is attributable to mortality changes experienced during the period 1930 to 1990. As shown elsewhere (Palloni & Lu, 1995), about 70% of this change is because of changes in mortality associated with parasitic and infectious diseases in the first 10 years of life. This is a revealing statistic: It suggests that the relatively compressed schedule of aging in the region can, in part at least, be traced to the medical and public health revolution that triggered the mortality decline nearly half a century ago. This legacy of the past has implications for the health and disability status of the elderly after the year 2000.

THE STICKINESS OF EARLY HEALTH STATUS

So far, we established two facts. First, the aging process that countries in Latin American and the Caribbean region will undergo during the period 1990 to 2050 owes significantly to the contribution of the mortality decline experienced during the period following 1930. Second, the bulk of these mortality changes were because of the implementation and deployment of an assortment of medical innovations and public health interventions rather than, as was indeed the case among most developed countries (Fogel, 1994, 2003; McKeown, 1976), to increases in standards of living or to improvements in levels of nutrition.

As a rule, when mortality falls, the surviving members of cohorts experiencing changes are of higher average frailty (Alter & Riley, 1989; Vaupel, Manton, & Stallard, 1979). This is purely an artifact of the changing composition by frailty and will tend to happen regardless of the origin of the mortality decline. However, the lives saved by the mortality decline in the region were certainly not random relative to conditions affecting health status. Indeed, they are more likely to have been drawn from populations exposed to higher risks, those whose morbidity and mortality experiences were dominated by parasitic and infectious diseases and lack of adequate early nutrition. Whenever the root origin of mortality improvements triggers increases in survival

among those whose nutritional status and experiences with illness is worse than average, the frailty composition of the corresponding cohorts will become less favorable than under a regime of survival gains that induces evenly spread mortality reductions.

Under conditions described earlier, most childhood morbidity responsible for higher mortality before the interventions continued to affect children, albeit with reduced lethality. Their effects were shared by a growing proportion of survivors drawn from high mortality subpopulations. This has important implications if early childhood conditions exert an impact on adult health and mortality.

Now, suppose that conjectures a la Barker (1998), those connecting early life conditions and late adult health status, are at least partially valid. According to these conjectures, detrimental conditions—including nutritional status and experiences with illnesses and faltering growth, some of which take place in utero, whereas others occur around birth and during early childhood—increase the susceptibility to certain chronic diseases during adulthood and old age. Although evidence that early childhood conditions affect adult health is far from water-tight, it is clearly mounting fast and cannot be ignored. Empirical data as well as theoretical arguments (Elo & Preston, 1992; Schaffer, 2000) implicate a very broad array of mechanisms, from those involving latent effects (Barker, 1998) to those requiring circuitous pathways (Hertzman, 1994), critical periods (Barker, 1998; Cynader, 1994; Hertzman, 1994; Schaffer, 2000), or accumulation effects (Barker, 1998; Elo & Preston, 1992; Hertzman, 1994). The first mechanism is closely associated with the work of Barker and concentrates on the sequelae of processes that may start in utero or develop shortly before and around birth (fetal origin hypothesis). In general, these effects develop as a result of either fixed traits that individuals are born with or, most interestingly, of stresses and uneven development of physiological systems that follow periods of moderate and severe deprivation and that remain latent until late in life. Thus, unless one has markers of early deprivation, there is little that can be done to falsify the conjecture. Some of these markers have already been used (birth weight, placental weight, length of gestation, length of recently born, etc.). None of these markers is available to us in the data we will use in this article. Instead, we rely on indirect measures of early nutritional status, including height (adjusted for age),

knee height (a proxy for leg length), and the waist-to-hip ratio (WHR). These measures have already been used with some success by nutritionists based on the argument, not always well confirmed by empirical evidence, that they reflect early nutritional status among adults.

A second mechanism identified in the literature focuses on episodes of illnesses in early childhood and their influence on the late onset of some chronic diseases (Blackwell, Hayward, & Crimmins, 2001; Davey Smith & Lynch, 2004; Elo & Preston, 1992; Kuh & Ben-Shlomo, 2004; Wadsworth, 1986; Wadsworth & Kuh, 1997). The best-known example of this is the relation between rheumatic heart fever—a common infectious disease in developing countries at least prior to the massive mortality decline that took place after World War II—and the onset of heart disease. Because all the data we use here contain information on retrospectively recalled childhood diseases, we can at least attempt to assess the size of the effects. The strategy is by no means optimal because not only we must deal with faulty recall but there is a serious selection problem we cannot address because individuals with the most serious cases of rheumatic fever may not have survived to be in our sample.

Finally, some research focuses on broader mechanisms and attempts to find associations between socioeconomic conditions experienced in early childhood and adult health status (Davey Smith & Lynch, 2004; Hayward & Gorman, 2004; Hertzman, 1994; Kuh, Power, Blane, & Bartley, 2004; Lundberg, 1991; Rahkonen, Lahelma, & Huuhka, 1997; Wadsworth, 1986; Wadsworth & Kuh, 1997; Warner & Hayward, 2003). This type of work is a roundabout way to find some of the connections due to the two mechanisms mentioned before. Thus, finding an association between socioeconomic status (SES) early in life and health status among the elderly may simply reflect the relation between current or recent SES and health. For the most part, this work aims at finding net effects of early SES on adult or health—that is, those that remain after appropriately controlling for current or recent SES. The interpretation of the net effects conventionally invokes either the existence of Barker-effects or the influence of early illnesses. All the data sources we will use enable us to test for this as they contain retrospective evaluation of markers of early childhood poverty, deprivation, and SES.

If any of these mechanisms turns out to have more than modest effects, increases in frailty among elderly whose earlier experiences fits the description provided earlier are likely to be pronounced.[2] This means that the health status composition of elderly in LAC should be worse relative to what would have been had the growth of the more recent and forthcoming cohorts of elderly been associated, as was the case in more developed countries, with improving standards of living. Our understanding of the relations between early childhood exposures and adult health status is still too primitive to enable us to establish precise predictions regarding the nature of expected health impairments. But this conjecture can at least be used as a guiding torch to explore the evidence available to us.

HEALTH STATUS IN A NEW DISEASE ENVIRONMENT

There is an important albeit neglected set of conditions that may influence elderly health status in the region. It is neither a mystery nor a novelty that the regimes of morbidity and mortality experienced by elderly people in developing countries are unusual. First, as one would expect (Omran, 1982), there is an expansion of chronic conditions—such as heart and lung disease, cancers, diabetes, and arthritis—and elderly people continue to be assaulted by significant levels of parasitic and infectious diseases (Frenk, Frejka, Bobadilla, Stern, & Sepulveda, 1991). We simply do not know what the health effects of exposure to highly interactive environments such as these are. What should one expect, for example, under conditions where elderly people are simultaneously weakened by malaria and exposed to higher risks of congestive heart disease? Or, where increases in diabetes because of the adoption of a westernized diet (Albala, Kain, Burrows, & Diaz, 2000; Popkin, 1993) are combined with recurrent intestinal infections and high prevalence of respiratory tuberculosis? What are the implications of a mixed mode of exposure for comorbidities, disability, and impairments among the elderly? What are the implications for treatment? What effects does it have on demands for health care?

Admittedly, this mixed mode of exposure is more prevalent in countries with a late demographic transition (such as Guatemala, Honduras, Bolivia, Peru, Ecuador) than in the more advanced coun-

tries of the region. Although such a regime is certainly not absent in the latter, it is likely to be less influential.

Objectives of the Analysis

In this article, we have two aims. The first is to draw inferences about expected mortality levels among elderly adults in LAC given the anthropometric profiles of individuals. The second is to presents preliminary estimates of the effects of a number of early childhood conditions on adult health status. There are two conclusions that we draw from these analyses: (a) expected mortality of older adults in some countries of LAC are fairly high, and as expected from the tenor of the conjecture presented earlier, excess risks are associated with the type of demographic regime each country experienced in the past; and (b) there is some, albeit weak, evidence suggesting that some early conditions exert an important effect on adult health status.

Methods

DATA

To test the aforementioned hypotheses, we used the Survey on Health and Well-Being of Elders (SABE; SABE, 2003). SABE collected comprehensive data on representative samples totalling 10,902 elderly aged 60 and older from seven major cities (six of them capital cities) of the LAC region: Buenos Aires (Argentina), Bridgetown (Barbados), São Paulo (Brazil), Santiago (Chile), Havana (Cuba), Mexico City (Mexico), and Montevideo (Uruguay) in 2000. All seven surveys were strictly comparable although translated to three different languages (Spanish, Portuguese, and English). In this article, we did not use data from Argentina because no anthropometric data were collected in the sample of Buenos Aires. Table 1 summarizes the sample characteristics for the SABE data.

For comparison purposes and to establishing a benchmark, we used 12,527 targets aged 60 and older from the 2000 University of Michigan Health and Retirement Study (Health and Retirement Study [HRS], HRS Core public use dataset, 2000). The HRS surveys more

Table 1
Basic Sample Information

Condition/ Variable	Overall (n = 10,902)	Argentina (n = 1,043)	Barbados (n = 1,808)	Brazil (n = 2,143)	Chile (n = 1,306)	Cuba (n = 1,905)	Mexico (n = 1,247)	Uruguay (n = 1,450)
Response rate	0.85	0.60	0.85	0.85	0.84	0.95	0.85	0.66
Age	72 (8)	71 (7)	72 (8)	73 (8)	72 (8)	72 (9)	70 (8)	71 (7)
60-64	23%	23%	19%	20%	22%	25%	31%	22%
65-69	23%	24%	23%	18%	25%	21%	25%	25%
70-74	19%	24%	21%	16%	19%	18%	18%	23%
75-79	17%	15%	17%	22%	16%	13%	13%	17%
80-84	11%	8%	11%	14%	10%	11%	8%	9%
85+	8%	5%	9%	10%	8%	11%	6%	5%
Gender								
Females	62%	63%	60%	59%	66%	63%	59%	63%
Education								
Primary	71%	71%	77%	85%	68%	57%	74%	65%
Secondary	20%	23%	18%	5%	24%	37%	11%	21%
Higher	9%	6%	5%	10%	9%	7%	15%	14%
Race								
White	55%	—	5%	71%	43%	63%	—	90%
Black	34%	—	93%	16%	1%	36%	—	4%
Mestizo	6%	—	1%	8%	30%	—	—	6%
Other	4%	—	1%	5%	26%	1%	—	1%
Marital Status								
Never married	7%	6%	18%	5%	7%	3%	4%	4%
Married/union	46%	43%	45%	52%	44%	37%	54%	49%
Separation/divorce	13%	10%	14%	7%	13%	24%	10%	11%
Widowed	34%	42%	24%	35%	36%	35%	32%	37%

Source. SABE data, respondents ages 60 and above. Palloni, A., & Guend, H. (2005). Stature prediction equations for elderly Hispanics by gender and ethnic background. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 60*, 804-810. Copyright The Gerontological Society of America. Reproduced by permission of the publisher.

Note. Numbers in parentheses are standard deviations where appropriate. Numbers rounded to nearest whole number. For race: the category Black includes Blacks and Mulattos; the category Other includes indigenous, Asian and all other; information on race is not available for Mexico or Argentina.

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than 22,000 Americans older than 50 every 2 years. The study paints an emerging portrait of an aging America's physical and mental health, insurance coverage, financial status, family support systems, labor-market status, and retirement planning. The sample we used in this article included 12,527 target respondents (no spouses) aged 60 and older.

MEASURES

We defined binary variables for diabetes and heart disease using responses to the following question: "Has a medical doctor ever told you that you had _____?" Obesity was defined using body mass index (BMI) $(kg/m^2) \geq 30$. We used height and knee height as markers of net malnutrition (Chumlea et al., 1998; Palloni & Guend, 2005) and defined short height and knee height using the gender and country specific lowest quintile of height (knee height).[3] We also employed retrospective questions to define childhood socioeconomic conditions (SES; "In general, would you say that the economic conditions in your home in which you grew up were good, fair or poor?"), childhood health during the period when the respondents were aged 5 to 15 years ("Would you say that your health was excellent, very good, good, fair, or poor?"), and experience of illnesses ("When you were a child, did you experience_____?").

METHODS I: WAALER SURFACES

Because SABE does not provide information on actual mortality, we cannot estimate directly the levels of mortality risks. There is, however, an indirect way of doing so using available anthropometric measures. Indeed, we can estimate the relationships between height, weight, and mortality risk using so-called Waaler surfaces.[4] These surfaces emanate from the work of Waaler (1984) and others (Costa, 2002; Fogel, 2003; Kim, 1993), and we are now examining their applicability in LAC countries (Palloni & McEniry, 2005). Assuming that Norwegian mortality risks associated with height-weight combinations apply to LAC and assuming that Norwegians in general were, most likely, exposed to better living conditions than individuals in our sample of LAC elderly, Waaler surfaces identify lower bounds for the

risks associated with height-weight combinations (i.e., less risk than might actually be the case). It is likely that Norwegians are in better health than U.S. elderly. In this case, Waaler surfaces also identify a lower bound of mortality risks for U.S. elderly (lower risk than might actually be the case).

For any health status indicator, such as obesity or diabetes, that leads to a partition of the sample into classes or groups (obese and non-obese, with and without diabetes, with and without heart diseases), we can approximately identify relative mortality risks for each class or group by calculating their mean weight and height and locating the corresponding point in a Waaler surface. To do so, we proceeded as follows: First, using Waaler's original data (Waaler, 1984), we estimated Waaler-type surfaces for (the log of) relative mortality risks as quadratic functions of height (cm) and weight (kg) plus interaction terms.[5] We then identified optimal lines—the locus of points yielding a weight that minimizes the mortality risk for a given height—and finally, we drew severe obesity lines defined as the locus of points where the height-weight combination yields a body mass index of 35.

Second, we used SABE and HRS data to obtain average height-weight for obese and non-obese 60 to 74 year old respondents and plotted these points on the surfaces. Under the assumptions stated before, the location of these points in the surfaces identifies a minimum mortality risk among obese (and non-obese) individuals in LAC and HRS respondents.

METHODS II: LOGIT MODELS OF SELF-REPORTED DIABETES AND HEART DISEASE

Our main conjecture suggests that the population surviving in the age group 60 to 74 reaped most of the benefits of improved childhood survival resulting from the post–1930 to 1940 mortality decline period. Accordingly, it is among them that we might be able to better observe the impact of early childhood conditions on late adult health status. Thus, for our statistical analyses, we limited our sample to individuals who, at the time of each survey, were aged 60 to 74.

We used diabetes and heart disease to examine the connection between early childhood and adult health status. We chose diabetes

because assessments discussed elsewhere (Palloni & McEniry, 2004) identify diabetes as one of the key chronic conditions whose prevalence is salient among elderly adults in the SABE cities. We also know that diabetes is one of the diseases that appears to be responsive to early childhood conditions (Aboderin et al., 2002; Barker, 1998). We used heart disease because it is a primary target among researchers searching for a connection between early and adult health status (Aboderin et al., 2002; Barker, 1998) and because there is at least one illness (rheumatic heart fever) that has been frequently connected to the adult onset of heart diseases (Elo & Preston, 1992).

Is there any evidence in countries of the region that current diabetes (or heart disease) is related to early childhood conditions and development? A simple way to identify the direction and magnitude of effects is to estimate for each city the relation between indicators of early health status and the probability of self-reporting diabetes (or heart disease). This is a blunt tool for a number of reasons. First, focusing on current diabetes (or heart disease) status constrains the universe of study to those who were able to survive with the disease. It is likely that those in worst health had a lower chance of surviving and of being interviewed. Second, although self-reported diabetes is generally quite accurate (Goldman, I-Fen, Weinstein, & Yu-Hsung, 2002; Palloni, Soldo, & Wong, 2003), even mild measurement errors can lead to powerful attenuation of estimated effects. The same applies to heart disease. Third, indicators of early child conditions—anthropometry and retrospective questions—are retrieved in a population-based study, carried out via person-to-person interviews, not in a clinical setting. As a consequence, the anthropometry may be subject to random errors attenuate estimates of the association between variables.

For the analyses that follow we used standard logit models for self-reported prevalence of the disease (diabetes or heart disease). We controlled for current conditions as we attempt to identify net effects of early childhood conditions. We are aware that self-reports on chronic conditions underestimate true prevalence of conditions but we also know that this would lead to downward biases in our estimates. A worse affliction of discrete models for prevalence is the existence of differential attrition of individuals with and without the condition as this leads to serious downward biases in the estimates of effects of

covariates (Palloni, Soldo, Wong, & McEniry, 2004). Thus, we know that our estimates of net effects will be lower bounds and can be used as conservative tests of conjecture and corollaries.

Results

HEALTH PROFILES: NAVIGATING THROUGH WAALER SURFACES

Figures 2 and 3 display curves of the log of the relative mortality risks for various combinations of height and weight among Norwegian males and females, respectively. The figures also display the points corresponding to the average height-weight combination among obese and non-obese males in our data sets. The highest mortality risk is found in the lower right hand corner of the graphs. These are cases of short height and high weight. Mortality risk increases as height decreases, and as weight increases for a particular height, there is a set of optimal points where mortality risk is minimized (optimal line). A particular weight-height combination always yields higher mortality risks for males than for females (compare iso-mortality risk curves across graphs), a feature inherent in the Norwegian data and likely to apply to other societies.

Several patterns deserve attention. First, non-obese elderly (represented with points bearing the symbol n) can be located in an area of lower mortality risks than obese elderly (represented with points bearing the symbol y). Indeed, the latter are always to the right and almost always below the level of the former (lower height). This is evidence of a clear mortality divide between obese and non-obese males as the former are always located in loci closer to the curve representing higher mortality risks, whereas the latter cluster very close to the optimal line. This is a feature characterizing all data sets under examination and is also present among females (Figure 3), albeit in a more attenuated fashion. The fact that the observed mortality divide between obese and non-obese is in the expected direction gives us some confidence for using the Waaler surfaces to produce stronger inferences.

An interesting feature is that the ordering of countries in the graph is tightly associated with their historical mortality regimes: those that
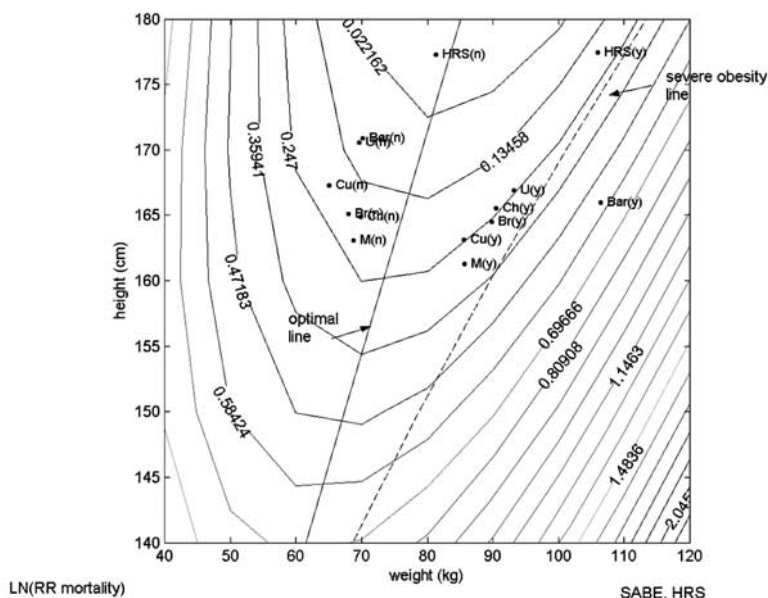
Figure 2.   **Waaler mortality surface with obese and non-obese males (aged 60 to 74).**

experience later mortality declines and were more influenced by the development of post–World War II medical technology display higher expected mortality risks among both obese and non-obese but more so among the latter than the former. Thus, for example, Uruguay always experiences a lower expected profile of mortality risks, followed by Barbados and Cuba, three countries that were forerunners in the process of mortality decline in the region. By the same token, in all cases the expected mortality risks are higher than those associated with U.S. respondents from HRS.

Admittedly, Waaler surfaces are a blunt tool to disprove the mortality-corollary introduced before. However, they allow us to state that elderly Latin Americans in general are characterized by an anthropometric profile that places them at a disadvantage relative to elderly in countries that are either wealthier (United States) or in societies where the growth of the elderly population is produced by an array of forces implying later mortality decline and driven by medical technology rather than by better standards of living.
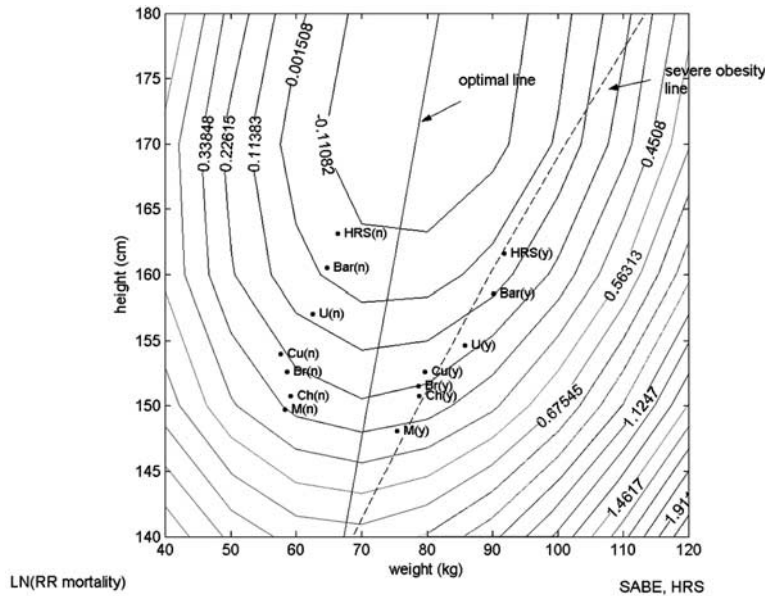
Figure 3.   **Waaler mortality surface with obese and non-obese females (aged 60 to 74).**

EARLY CHILDHOOD CONDITIONS AND
INDICATORS OF ADULT HEALTH STATUS

Tables 2 and 3 display the estimated effects of logistic models for diabetes and rheumatic fever, including indicators of early nutritional status, early socioeconomic and health status, and experience with rheumatic fever. The indicator for knee height is a dummy variable attaining the value of 1 if the individual's knee height is less than the first quintile of the corresponding country-gender distribution. The indicator for height is a dummy variable attaining the value of 1 if the individual's stature is less than the first quintile of the corresponding country-gender distribution. Finally, the indicator for WHR is a dummy variable attaining the value of 1 if the individual's WHR is larger than the first quintile of the corresponding country-gender distribution. In all cases considered, these indicators are entered one at a time while controlling for other relevant variables. All models control for age (two dummy variables for age groups *60 to 64*, *65 to 69*, with

Table 2
Effects of Markers of Nutritional Status and Self-Reported Heart Disease

			Model		
	OR	p *Value*	*Log L*	n	*Pseudo* R^2
Barbados					
Knee height (KH)	.95	.803	−548	1,065	.02
Height	1.19	.399	−549	1,066	.03
Waist-to-hip ratio (WHR)	2.47	.000	−537	1,059	.04
Brazil					
KH	.92	.698	−434	947	.02
Height	.62	.090	−435	949	.02
WHR	2.40	.000	−424	949	.05
Chile					
KH	1.67	.040	−315	796	.01
Height	1.15	.614	−317	799	.004
WHR	1.40	.223	−316	798	.01
Cuba					
KH	.88	.576	−442	1,107	.03
Height	.90	.684	−444	1,108	.03
WHR	1.60	.027	−442	1,105	.03
Mexico					
KH	1.51	.037	−393	770	.03
Height	1.11	.662	−395	770	.03
WHR	1.50	.036	−391	767	.04
Uruguay					
KH	1.02	.937	−329	877	.03
Height	1.02	.943	−348	914	.03
WHR	1.72	.043	−329	873	.03

Source. SABE and PREHCO, 60 to 74 years old.
Note. Log L stands for the log likelihood of the logistic models, and *n* for the number of cases in each sample. All models include controls for gender, age, education, and a dummy variable for obesity. We also include controls for indicators of poor childhood health and poor childhood socioeconomic conditions.

the residual category corresponding to the age group *75+*), gender (one dummy equal to 1 for females), current education (three dummy variables—one for *no schooling*, one for *primary*, and one for *secondary* with *higher education* being the residual category), early childhood conditions (one dummy for *poor*), and early health status (one dummy for *poor*). In addition, the logistic models for self-reported diabetes control for an indicator of obesity (one dummy equal to 1 if BMI greater than or equal to 30). A control for obesity is required because the tenor of the conjecture is directed at the net effects of early

Table 3
Associations Between Markers of Nutritional Status and Self-Reported Heart Disease

	OR	p *Value*	*Rheumatic Fever*		*Model*		
			OR	p *Value*	*Log L*	n	*Pseudo* R^2
Barbados							
Knee height (KH)	1.04	.901			−326	1,052	.02
Height	1.09	.775			−326	1,054	.02
Waist–to–hip ratio (WHR)	1.01	.983			−325	1,047	.02
Brazil							
KH	1.38	.128	1.61	.381	−431	942	.03
Height	1.14	.585	1.62	.371	−432	944	.03
WHR	1.39	.062	1.58	.400	−431	943	.03
Chile							
KH	1.34	.140	3.71	.014	−485	785	.01
Height	.70	.118	3.46	.020	−488	788	.01
WHR	.65	.070	3.40	.022	−486	787	.02
Cuba							
KH	.74	.108	2.61	.150	−574	1,098	.02
Height	.68	.085	2.52	.165	−574	1,099	.02
WHR	1.03	.863	2.44	.177	−574	1,096	.02
Mexico							
KH	1.68	.070	3.63	.040	−230	762	.03
Height	1.20	.590	3.36	.053	−232	762	.03
WHR	.93	.802	3.29	.056	−232	759	.03
Uruguay							
KH	.79	.308	4.17	.001	−427	873	.03
Height	.85	.508	4.41	.001	−448	909	.03
WHR	1.08	.754	4.02	.002	−428	868	.03

Source. SABE & PREHCO, 60 to 74 years old.
Note. All models control for gender, age, education and also include controls for indicators of poor childhood socioeconomic conditions and poor childhood health. Very few cases of self-reported rheumatic fever in Barbados resulted in not including this variable in the mode.

childhood conditions, not the gross effects. Because part of the latter operate via increased predisposition to develop obesity, it is important to control for this variable. In the models for heart disease, on the other hand, we also introduce self-reported early childhood experience of rheumatic fever (one dummy variable).

To avoid cluttering, the tables display only estimates and *p* values for variables under scrutiny, namely, the indicators of early nutrition (knee height, height, and WHR) and rheumatic heart fever. Thus, the effects of knee height, for example, are effects net of those associated

with poor early childhood health and poor socioeconomic conditions among individuals of the same age, gender, and educational level.

The results for anthropometry are mixed. Unlike findings obtained by other authors for the United States (Costa, 2002; Fogel, 1994; Kim, 1993), we find no evidence at all supporting the idea that height per se has any relation to the probability of diabetes once the effects of current nutritional status (as reflected in BMI) are controlled for.

Knee height is not only a good predictor of current height in populations whose skeletal mass is compressed by age-related processes (Chumlea et al., 1998; Palloni & Guend, 2005), but it, as well as leg-length, is a marker of early malnutrition. But in only two of the six countries (Chile and Mexico) do we find credible evidence that being in the lowest quintile of knee height is related to current diabetes status. The odds ratios are fairly high, 1.51 in Mexico and 1.67 in Chile.

Finally, WHR ratio is a powerful predictor of current diabetes status in five of the six countries. This finding is interesting but admits two very different interpretations. On one hand, evidence for poor populations suggests that WHR is affected by early malnutrition (Martorell, Stein, & Schroeder, 2001; Schroeder, Martorell, & Flores, 1999). If so, the estimated effects shown in the table could reflect the impact of early malnutrition on the propensity to develop diabetes as an adult. On the other, WHR is a measure of central adiposity and, in addition, could also reflect hormonal and metabolic disorders produced, for example, by sustained stress (Adler, Epel, Castellazzo, & Ickovics, 2000; Ostrove, Adler, Kuppermann, & Washington, 2001). If so, the estimated effects shown in the table are only indicative of the relation between stress in the recent past, metabolic imbalances, and diabetes. This mechanism does in no way implicate early malnutrition.

Together with the finding of a strong association between WHR and self-reported diabetes, the estimated strong effect of experience with rheumatic fever in Table 3 is worthy of note: In three of the five countries with relevant information, the estimated effect is large, statistically significant, and in the right direction. This confirms other findings regarding the relation between rheumatic heart fever and the adult onset of heart disease (Elo, 1998; Elo & Preston, 1992). However, two caveats are necessary. Firstly, although the relations are significant, the proportion of individuals who experience rheumatic

Table 4
Effects of Early Child Health and Early SES on Diabetes

	Child SES		Child Health		Model		
	OR	p *Value*	OR	p *Value*	*Log L*	n	*Pseudo* R^2
Barbados							
Model A	1.28	.111	.94	.919	−549	1,066	.03
Model B	1.28	.115	.94	.927	−548	1,065	.02
Model C	1.27	.126	.97	.968	−537	1,059	.04
Brazil							
Model A	1.39	.077	1.52	.180	−435	949	.02
Model B	1.42	.060	1.47	.214	−434	947	.02
Model C	1.34	.116	1.51	.190	−424	949	.05
Chile							
Model A	.80	.386	1.09	.838	−318	799	.00
Model B	.78	.335	1.17	.705	−315	796	.01
Model C	.80	.377	1.12	.778	−316	798	.01
Cuba							
Model A	.83	.327	1.32	.468	−444	1,108	.03
Model B	.84	.353	1.33	.464	−442	1,107	.03
Model C	.81	.273	1.33	.462	−442	1,105	.03
Mexico							
Model A	1.19	.351	1.61	.209	−395	770	.03
Model B	1.21	.325	1.59	.221	−393	770	.03
Model C	1.21	.325	1.65	.187	−391	767	.04
Uruguay							
Model A	.56	.029	1.30	.609	−348	914	.03
Model B	.53	.020	1.38	.528	−329	877	.03
Model C	.57	.037	1.36	.549	−329	873	.03

Source. SABE, 60 to 74 years old only.
Note. All models controlled for gender, age, education, and obesity. Model A also controls for height, Model B controls for knee height, and Model C for waist-to-hip ratio.

fever is fairly low and thus not consequential as a factor responsible for heart disease at adult ages. Secondly, our findings admit an alternative interpretation. Suppose that individuals who experience and self-report a heart condition as adults are also more sensitized and more prone to admit or reveal early heart conditions. If this were the case, the strong association we observe in the data is all a product of self-reporting bias.

We turn now to the relation between self-reported retrospective indicators of early childhood socioeconomic and health status and current diabetes and heart disease status (Tables 4 and 5). Models A,

Table 5
Effects of Early Child Health and Early SES on Heart Disease

	Child SES		Child Health		Model		
	OR	p *Value*	OR	p *Value*	*Log L*	n	*Pseudo* R^2
Barbados							
Model A	.66	.079	1.60	.542	−325	1,069	.02
Model B	.69	.112	1.57	.563	−325	1,067	.02
Model C	.68	.108	1.73	.480	−324	1,062	.02
Brazil							
Model A	1.43	.052	.47	.074	−437	957	.03
Model B	1.42	.060	.47	.069	−436	955	.03
Model C	1.40	.070	.48	.082	−436	956	.03
Chile							
Model A	1.03	.855	1.66	.078	−497	802	.01
Model B	1.01	.967	1.69	.071	−495	798	.01
Model C	1.04	.815	1.56	.123	−495	801	.01
Cuba							
Model A	.98	.886	.96	.904	−577	1,106	.02
Model B	.97	.848	.96	.908	−577	1,105	.02
Model C	.96	.816	.98	.958	−578	1,103	.02
Mexico							
Model A	1.16	.594	4.38	.000	−234	771	.04
Model B	1.17	.563	4.29	.000	−233	771	.04
Model C	1.17	.578	4.41	.000	−234	768	.04
Uruguay							
Model A	1.25	.249	1.30	.53	−456	913	.02
Model B	1.32	.169	1.36	.453	−435	876	.02
Model C	1.29	.201	1.12	.784	−436	871	.02

Source. SABE, 60 to 74 years old only.
Note. All models control for gender, age, education, and obesity. Model A also controls for height, Model B controls for knee height, and Model C for waist-to-hip ratio.

B, and C differ with regard to which indicator of early malnutrition is being controlled for, but all three of them include controls for age, gender, and current education. The focus is on the effects of two dummy variables: *poor health status during early childhood* and *poor socioeconomic conditions during early childhood*, both constructed from respondents' self-reports.

We see that the estimated effects are insignificant in all cases except for the case of Uruguay, where the effect is in the opposite expected direction, and marginally significant in Brazil but in the expected direction. However, these findings do not appear to be robust enough

to make a convincing case for the existence of relations. Table 5 tells the same story for heart disease: With the exception of Brazil, where poor early health status appears to have some influence on heart disease, there are no associations worthy of notice.

It is important to note that these tables report the net effects of early socioeconomic and health status—that is, the effects that remain while we control for indicators of early malnutrition (alternatively knee height, height, and WHR). Therefore, the interpretation should proceed accordingly—namely, that there is no evidence of a negative relation not mediated by early malnutrition. Results not shown indicate that with a handful of marginal exceptions, the gross affects of early childhood health and early SES are not relevant either. This finding leaves us with the impression that aside from some direct relations between indicators of malnutrition and adult illnesses, the story that can be told is highly vulnerable.

Discussion

In this article, we examined a conjecture regarding health status of elderly people in LAC that involves relations between early childhood conditions and adult health status. A full examination of the conjecture requires data on several cohorts of individuals. However, with the cross-sectional data from SABE, we are able to obtain insights and at least some empirical leverage. First, using anthropometric information collected in the SABE study, we find that expected mortality risks among elderly in LAC reflect excesses that are in line either with past and current levels of well-being or with the type of mortality decline we conjecture to have produced peculiar cohorts of individuals who will become 60 after the year 2000. Second, we also find that there is some evidence, albeit weak, to suggest that our conjecture regarding elderly health status' connection to early conditions has some merit. Indeed, there is partial evidence of a relation between various indicators of early childhood nutritional status and diabetes, one of the more salient chronic diseases in the region and even stronger evidence of a relation between rheumatic fever and adult heart disease. As argued at the outset of the article, this is uniquely relevant in the region where

demographic aging has proceeded via a singular path and where the cohorts of individuals who will become 60 and older during the next two decades or so were the beneficiaries of a unique mortality decline that took place starting at the end of the second quarter of the century.

But, overall, the evidence is fragile and does not constitute a strong foundation for the conjecture as we only get ambiguous signals from the data. This lack of success in our search for connections between early childhood conditions and adult health status may be a result of lack of appropriate information as much as of an inappropriate interpretation. Two problems in particular deserve special attention. First, we confined our attention to examination of self-reported measures of two chronic conditions that may be affected by important measurement errors leading to attenuation of associations. Second, although we include only those cohorts to which the conjecture really applies—namely, those born after 1925 to 1930—we cannot solve an important selection problem—namely, that those who did not survive to the year of the survey may represent a large stock among the population to whom the conjecture applies.

Despite these difficulties, the relations we find between knee height and WHR and diabetes (after controlling for the effects of obesity) are worth investigating. We interpret the relation with WHR as a partial reflection of early malnutrition and propensity to develop adult diabetes, although the findings admit an alternative interpretation. Similarly, the robust finding of a relation between rheumatic heart fever and adult heart disease confirms other researchers' findings (Elo, 1998; Elo & Preston, 1992) and needs to be studied in more depth.

The high prevalence of diabetes and obesity in LAC is worrisome. It is known that the health cost implications of diabetes are staggering, even if the disease presents itself with a normal, expected profile of associated comorbidities. Regardless of whether the origin of the large prevalence is largely a matter of adoption of a Westernized life style or, as we argued here, it also involves early exposure, a continued increase on diabetes prevalence will pose severe constraints in the health system. In the next 50 years, perhaps the most important health policy initiatives regarding the elderly will have to be related to prevention of obesity and diabetes.

NOTES

1. This statement is only accurate in societies that during the past 60 or so years have experienced drastic mortality changes at younger ages. It cannot hold for societies whose survival curve can only be modified by improving mortality at old ages.

2. The argument holds, of course, if we assume that the effects of mortality selection are only mild and if the effects of changes in behavioral profiles and medical technology (exogenous or not) are only weak.

3. Because of discontinuities in the data, the identification of the first quintile was not exact but simply a close approximation.

4 Throughout, we used measured (not self-reported) height and weight.

5. The relative mortality risk is defined as the ratio of the mortality rate for a particular weight-height combination to the average mortality rate for the population.

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