TITLE: Maternal height, childhood nutritional status and adult mortality

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AIMS

A large and growing number of studies show that child and adolescent height is an effective predictor of mortality and morbidity during adolescence and adulthood. There are two distinct and quite separate strands in this literature. First, there is research mostly in low income countries that highlights the predictive power of early childhood height. This is mostly due to the high fidelity of height as an indicator of genetic traits , environmental factors, and their interactions acting over extended periods of time from in-utero exposures to adulthood. Height at early ages is sensitive to nutritional intake, energy demands associated with infectious and parasitic diseases, stresses originating in precarious household conditions, limited parental care, and sibling competition, and to interactions between these and genotypic traits. An important, albeit not well-established, fact is the role played by parental height and its importance as a marker of parent-children influences via phenotypic and epigenetic changes. It is possible that there is an intergenerational linkage due to, among other things, in-utero and very early life (0 to 1 years of age) deprivation (Delgado et al 1982, Martorell 1995, Scrimshaw 1997, Stein et al 2010).

Second, historical studies have repeatedly shown that levels of overall mortality experienced by entire populations during a short period of time are tightly associated with average height of adolescents some twenty to twenty five years before (Fogel 1994, Fogel 2004, Ruel et al 1995). With few exceptions these studies rest on demographic and physiological information collected from historical records in Western Europe and North America.

This paper seeks to join these two research tracks. We assess the strength of the relation between parental and offspring heights, likelihood of early stunting, and infant and child mortalityin a number of low income countries in Latin America. We then employ these relations jointly with exceptional information of adult female and male heights for the period 1820-1930 and estimate prevalence of childhood stunting and infant and child mortality for that period. We verify the quality of the estimates obtained using independent sources of information about child and adult mortality.

BACKGROUND

Early childhood nutritional deprivations is strongly associated with prevailing levels of stunting and wasting, infant and early childhood mortality (Martorell 1981, Ozaltin et al 2010, Black et al 2013). While infant and child precarious nutritional status is largely a result of poor nutritional intake both before and after birth, it is amplified by synergistic relations with exposure and contraction of infectious and parasitic diseases. Repeated bouts of dysentery and infectious diarrhea, for example, are more likely in the presence of nutritionally driven impaired immune function. But, one of the outcomes of these ailments is the destruction of the stomach lining and microbiota and consequent

impaired ability to absorb nutrients thus aggravating initial nutritional status deficiency.

Adolescent and early adulthood height are effective predictors of adult morbidity and mortality risks in adulthood. The relationship between adult height and chronic diseases such as cardiovascular morbidity, diabetes type 2, and congestive obstructive pulmonary diseases (COPD), as well as late life disabilities, is well-documented (Fogel 2004, Silventoinen et al 1999). By and large these relations are thought to be the result of a close association between in utero and early life conditions, growth, development and exposures during adolescence, and adult diseases and disabilities (Barker et al 1989, Barker et al 1993, Gluckman and Hanson 2004)

Finally, there is robust evidence documenting a strong relation between maternal (paternal) and offspring heights. This relation could be due to exposure to shared conditions such as poverty and generalized adversity (Wadsworth & Kuh 1989, Wadsworth 1997, Webb et al 2008, Tucker-Seeley et al 2011) but could also involve maternal nutritional and health status (during pregnancy,delivery, and post-partum), maternal child care, including breastfeending onset and duration (Lechtig 1975, Delgado et al 1981, Victora et al 2008) and, finally, genetic and epigenetic pathways. This relations open up a startling possibility, namely, that maternal height reflects conditions whose effects are felt at various stages in the life course of children and involve child nutritional status and risk of stunting, early childhood mortality, adolescent height and, ultimately, adult mortality and morbidity risks Intriguingly, the inverse relationship of maternal height and child stunting, morbidity and mortality levels persists even after adjusting for multiple indicators of mother-child shared conditions and child specific factors such as maternal age at birth and parity (Kuh and Wadsworth 1989, Martorell 1981, Ozaltin et al 2010). A recent analysis using DHS data conducted between 1991 and 2008 in fifty-four countries shows that children under 5 years of age who were born to the shortest mothers have a 40% increased risk of mortality (after controls). These effects are so strong that they are virtually equivalent to effects of maternal education and poverty (Ozaltin *et al*. 2010).

The foregoing empirical findings imply but do not prove the long reach of maternal phenotypic and genotypic characteristics (Dewey and Begum, 2011). If child stunting risks are shaped by maternal traits encapsulated in maternal height, it follows that these traits will impose constraints on offspring adolescent and adult health and mortality conditions. This is because stunting carries physiological damages that irreversibly affect health and development over the life-course. High glucose concentrations, blood pressure and harmful lipid profiles in adulthood are some of the disorders associated with growth impairment during early life.

The implications of the relations described above are manifold. One of these is that if maternal height reflects conditions that are different from those shared by mother and child (social class, poverty etc…) then there is a distinct possibility that the intergenerational transmission of health and mortality risks works through multiple pathways (Black et al 2008, Stein et al 2010). Another implication, the one that is at the core of our paper, is that if one has information of adult height (for female and/or males)

at a particular time, it should be possible to estimate (a) prevalence of child stunting at time t, (b) early childhood mortality at time t+5, and (c) life expectancy for the time interval between t and t+60 approximately.

MODELS, DATA AND EMPIRICAL ESTIMATION

The paper is in three sections. In the first we estimate models for the relation between maternal height and child height, stunting risks, and infant and child mortality. We use fixed and random effects models and data from the Demographic and Health Surveys from Latin American countries, including , Bolivia, Brazil, Colombia, Dominican Republic, Guatemala, Nicaragua and Peru. DHS data allows the examination of the relationship between maternal height with childhood outcomes while controlling for potentially confounding factors, and are well suited nationally representative samples with 'measured' rather than 'reported' body size measures. An important part of this estimation exercise is to establish the mediating role of breastfeeding onset and duration.

In the second section we use the results obtained above jointly with exogenous estimates of the relation between child and adult height, on one hand, and adult height and adult mortality, on the other, to establish ranges for adult mortality rates for the DHS birth cohorts. These are then compared with conventional mortality projections for the year 2020.

In the third section of the paper we propose to utilize information on female height for the period 1850-1930 for three of the DHS countries (Mexico, Colombia and Guatemala) to estimate (a) extant levels of childhood stunting and (b) prevailing levels of child mortality. We then use these to estimate overall life expectancy for the period 1850-1930. These estimates,based on information of parental height, are then contrasted with computations that rely on information completely independent of assessment of height (generalized stable populations based on census enumerations).

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