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ABSTRACT

We investigate the childhood determinants of adult height in populations, focusing on the respective roles of income and of disease. We develop a model of selection and scarring, in which the early life burden of nutrition and disease is not only responsible for mortality in childhood but also leaves a residue of long-term health risks for survivors, risks that express themselves in adult height, as well as in late-life disease. Across a range of European countries and the United States, we find a strong inverse relationship between postneonatal (one month to one year) mortality, interpreted as a measure of the disease and nutritional burden in childhood, and the mean height of those children as adults. In pooled birth-cohort data over 30 years for the United States and eleven European countries, postneonatal mortality in the year of birth accounts for more than 60 percent of the combined cross-country and cross-cohort variation in adult heights. The estimated effects are smaller but remain significant once we allow for country and birth-cohort effects. In the poorest and highest mortality countries of the world, there is evidence that child mortality is positively associated with adult height. That selection should dominate scarring at high mortality levels, and scarring dominate selection at low mortality levels, is consistent with the model for reasonable values of its parameters.

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1. Introduction

This paper is concerned with the relationship between net nutrition in childhood and subsequent adult height, the latter interpreted as a measure of life-time health and which, in turn, predicts late-life morbidity and mortality. Net nutrition is gross nutrition from food, less nutritional losses to disease. Health policy can be directed at one or both of these, with income often seen as the instrument for gross nutrition, and public health and medicine, through such measures as sanitation and immunization (public health), or oral rehydration (medicine), as instruments against disease. Where to place the main focus is a key question for policymakers in poor countries today.

We look at the relationship between childhood disease, income, and adult height within a theoretical framework that emphasizes the countervailing forces of scarring and selection. Disease and deprivation in infancy generate health and height selective mortality, but they also "scar" the survivors whose health and height are lower than would otherwise have been the case. Our primary focus is on birth cohorts born between 1950 and 1980 in eleven European countries and the United States. In accord with the dominance of scarring over selection, we find a strong *negative* relationship between adult height and the disease burden in the first year of life, measured by postneonatal mortality, particularly postneonatal mortality from respiratory disease. Variations in postneonatal mortality account for more than sixty percent of the pooled cross-country and time-series variation in adult heights. Although the size of the effect is about half as big based on within-country estimation as it is when we combine cross and within country variation, it remains statistically significant and is so even in the presence of both year and country effects. Conditional on the disease burden, we find no role for income.

These results are broadly consistent with our model of selection and scarring. Even so, mortality rates in rich countries since 1950 are too low to allow exploration of our theoretical prediction that selection effects will be relatively stronger at high levels of child mortality. When we add data on heights from forty-three poor countries, we do indeed find such evidence, with some suggestion that, at very high levels of mortality, selection may dominate scarring, at which point further increases in mortality will result in *increased* adult heights in the surviving population. These results need to be interpreted with some caution given the much weaker data for the poor countries, as well as the large fraction of the cross-country variation in heights that is unexplained by mortality rates. Indeed, both income and disease play a role in high-mortality countries, and there are large unexplained differences between regions—with Africans taller, and South Asians shorter than would be expected—as well as remarkable variations across countries within continents, particularly within Africa and Latin America.

The paper is laid out as follows. Section 2 reviews the background literature and motivation for the analysis. Section 3 presents a model of childhood disease and its effects on adult height. It allows us to think about the long term effects of disease, and allows for long run damage and possible immunity effects, as well as the effects of selection on population health. Section 4 discusses our European and American data and presents preliminary graphical evidence. Section 5 presents our main regression results. Section 6 contains the comparative results from developing countries. Section 7 concludes.

2. Background

Height is of considerable interest to both economists and biologists. It is a direct and readily available measure of long-run, life-course health, and is an indicator of wellbeing in its own right. Economic historians have long used mean height as a supplementary measure of population living standards; height often, but not always, tracks measures of average wages or GDP per head, Floud, Wachter, and Gregory (1990), Steckel (1995). Skeletal remains have been used to provide estimates of living standards in the distant past, sometimes suggesting that the historical progress of living standards has been less unidirectional than is often thought, Steckel (2004). At the individual level, taller people earn more in both rich and poor countries, they are better educated, they are more likely to belong to non-manual occupations and to higher social classes, and they have lower mortality rates even controlling for other risk factors, Waaler (1984), Jousilhati, Tuomilehto, Vartiainen, Eriksson, and Puska (2000). Case and Paxson (2006) argue that height is a marker for cognitive development, and that the (on average) superior cognitive development of taller individuals may account for their superior outcomes in the labor market. Fogel (2004) has argued that for much of human history, the availability of calories constrained human growth, and it was the agricultural revolution, starting in Britain in the middle of the 18th Century, that set off a cumulative synergistic process in which both heights and output per head increased, enough to account for most of the increase in both economic living standards and in life expectancy over the last two hundred and fifty years.

The literature on human growth argues that an individual's stature is determined early in life, by age two or three; see Silventoinen (2003) for a review of the respective roles of

genetics, disease, and standards of living. The correlation of child's height with ultimate height, which is between 0.25 and 0.3 at birth, rises to between 0.7 and 0.8 at age two, and increases only slowly thereafter, Schmidt, Jorgensen, and Michaelsen (1995). Since nutrients are required for physical development, the simplest version of the story is that final height is a marker of the availability of nutrition in the first two years of life. But nutrition in this context means not just food intake, or *gross* nutrition, but the nutrition actually absorbed, or *net* nutrition. Net nutrition is determined not only by food availability, but by losses to diseases, most obviously diarrheal disease. Children who constantly suffer from diarrhea because they drink contaminated water are unlikely to be well-nourished, irrespective of the food available to them. It is therefore possible to tell a story of historical mortality decline that focuses as much on disease and public health as on the availability of food. In modern developing countries, a focus on childhood disease has quite different policy implications than a focus on economic growth, which is presumably the surest way to improve gross nutrition.

There is a large literature that links childhood disease to adult outcomes, including adult height as well as late-life disease and mortality. As early as Kermack, McKendrick, and McKinlay (1934), researchers have repeatedly found strong birth-cohort effects in lifetime mortality patterns, at least before recent years when smoking, antibiotics, and successful medical interventions have added important period effects. These results suggest that early life experiences have long-lasting effects on health, with the lifetime pattern of mortality set in the first few years of life. The long reach of net nutrition *in utero* and in early childhood, observable throughout life in childhood growth and adult height, is one possible mechanism that has been extensively studied following the

influential work of Barker, see Barker, Osmond, and Golding (1990) and Barker (1994). Others include infections that are acquired in childhood, and carried through life, such as respiratory tuberculosis, or, as has been argued by some, infections that predispose towards heart disease, see Elo and Preston (1992) and Costa (2000) for reviews.

Finch and Crimmins (2004) and Crimmins and Finch (2006) argue that a wide range of childhood infectious diseases lead to inflammatory responses, which are well adapted for short term survival, but which divert energy from growth and diminish adult stature, and which beyond that, increase the risk of cardiovascular disease in late life. If this hypothesis is correct, the links between late life mortality and adult stature, on the one hand, and childhood disease on the other, may go well beyond the obvious mechanism from net nutrition to height to mortality, and extend to a much wider range of childhood diseases. These are likely to include respiratory infections, such as pneumonia, that still exert a heavy toll among children in developing countries, where they kill nearly four million children each year, World Health Organization (2003). Such general effects of infectious disease on long-term health also strengthen the argument for collective action on traditional public health measures, as well as better delivery of antibiotics, and weaken the argument for relying on economic growth.

Crimmins and Finch, in addition to documenting the links between childhood infectious disease and old-age mortality in several European countries, use long-run nineteenth century data from France and Sweden to confirm a relationship between mean adult height (at age 20-21) for a birth cohort and its infant mortality rate, the latter interpreted as an indicator of the general disease environment in childhood. Their work complements that on modern data by Schmidt, Jorgensen, and Michaelsen (1995) who

document a relationship between the average height of 18-year old conscripts in eleven European countries and postneonatal (from 28 days to one year) mortality (PNM) in the year of their births. PNM is a better indicator of the disease environment than is infant mortality, because the latter includes neonatal mortality (mortality from birth to age of 28 days) changes in which reflect improvements in pre- and perinatal care rather than improvements of nutrition and prevalence of infections during infancy, Schmidt, Jorgensen, and Michaelsen, (1995). In Europe PNM has been declining in all countries, leveling out at different dates once a common low level has been achieved. Schmidt, Jorgensen, and Michaelsen document an inverse but matching pattern in conscript heights, with heights leveling off in the countries whose PNM reached low levels first, and with cross country height differences mirroring cross-country differences in PNM eighteen years before.

3. Childhood disease and adult height: a theoretical framework

Suppose that each child is born with some physiological characteristic h_i. This characteristic, which we might as well think of as (potential) *adult height*, is distributed in the population with distribution function $F(h)$. Newborn children with height less than a cutoff *z* cannot survive, so that the baseline infant mortality rate, which we can think of as mortality from factors (such as genetic abnormalities) that operate up to and including birth, is $F(z)$.

Into this baseline situation, we introduce an environmental disease or nutritional burden that varies from year to year. Write this as v_t, with larger values indicating heavier disease burden. We think of these as epidemics of childhood disease, smallpox

and whooping cough in the 17th and 18th centuries, measles and scarlet fever in the early to mid-20th century. They could also cover nutritional deficiencies, whether through famine-induced lack of food, or through infectious disease reducing nutritional intake.

The disease burden is assumed to reduce the endowed physiological characteristic, increasing the infant mortality rate. In the empirical work, postneonatal mortality is our best indicator of this burden, but for the theoretical development, we draw no distinction. Children die if the reduced characteristic is less than *z*, if

$$h_i - v_t \leq z \tag{1}$$

so that the mortality rate, taking account the disease burden, is now given by

$$m_t = F(z + v_t) \tag{2}$$

which varies from year to year. If we knew the distribution and the value of *z*, we could recover the disease burden from knowledge of the infant mortality rate using

$$v_t = F^{-1}(m_t) - z \tag{3}$$

In this sense, infant mortality is an indicator of the burden of disease, which justifies its frequent treatment as such in the literature. Note that *z* is measured in the same units as *h*, and in the empirical results below, will typically be presented as a "*z*-score," the number of standard deviations below the mean at which survival becomes impossible.

We need to add long-term effects or "scarring" to this story. Those who survive the epidemic are assumed to pay some permanent price in their long-term health. For example, this scarring might come from an infection acquired in childhood that is carried through the rest of life; respiratory tuberculosis, or *helicobacter pylorii*, would be examples from the time before there was effective chemical prophylaxis. Acute rheumatic fever in childhood might predispose to rheumatic heart disease in old age. This

scarring might affect adult mortality, or in the analysis here, adult height. We assume that some fraction θ of v_t is permanently deducted from their physiological parameter. Hence, for the survivors,

$$\tilde{h}_{it} = h_i - \theta v_t \tag{4}$$

We shall typically assume that θ is positive, because that appears to conform to the empirical evidence, but if the encounter with the disease confers some sort of lifetime immunity, as in the case of smallpox, for example, θ could be negative. The size of θ, which has the dimension of a pure number, measures the extent to which the forces that shift down the health endowment, and which lead to infant mortality, persist into final adult height. If the effects of early damage are permanent, θ will be unity; with some transitory effect or some recovery, it will be less.

Given the adult height of the survivors from equation (4), the average adult height of the survivors of the cohort born in *t* is given by

$$\bar{h}_t = \frac{\int_{z+v_t}^{\infty} h dF(h)}{1 - F(z + v_t)} - \theta v_t \tag{5}$$

The first term is *increasing* in the burden of disease v_t; childhood disease selects out the shorter people, leaving people who are on average taller, and taller by more the greater the severity of the disease. The second term, which is negative if θ is positive, is the scarring effect of the disease. If θ is negative, so that the childhood disease encounter confers immunity, the second term will reinforce the positive selection in the first term.

We also note that (5) is likely to be useful for consideration of the effects of childhood disease on other late-life events, such as the susceptibility to cardiovascular disease. Each such event would be characterized by selection and scarring effects but

with its own parameter θ. As a result, height will be (imperfectly) correlated with late-life health.

The effects of v_t on cohort height can be assessed by differentiating (5), which can be written as

$$\frac{\partial \bar{h}_t}{\partial v_t} = \left(\bar{h} - z - (1-\theta)v_t\right)\frac{f(z+v_t)}{1-F(z+v_t)} - \theta \tag{6}$$

Since height has to be larger than $z+v_t$ in order to survive, the shortest survivor has adult height $z+v_t-\theta v_t$, so that the first term on the right hand side of (6) is positive. This is the effect of childhood mortality selecting out the infants and children who have the lowest potential adult height. Depending on the values of the parameters, and the size of the burden v_t, the net effect can go in either direction, and the derivative in (6) can change sign over the range of v_t.

Given that the shock v_t is not observable, but that the mortality rate is, it is useful to combine (3) and (5) so as to link the average height of the survivors to the mortality rate of the birth cohort. This yields

$$\bar{h}_t = \frac{\int_{F^{-1}(m_t)}^{\infty} h dF(h)}{1-m_t} - \theta\left(F^{-1}(m_t) - z\right) \tag{7}$$

Equation (7) is more straightforward to handle if we assume that the (untruncated) original distribution of heights is normal with mean μ and variance σ^2. Substituting into (7) and performing the integration, we obtain

$$\frac{\bar{h}_t - \mu}{\sigma} = \frac{\phi[\Phi^{-1}(m_t)]}{1-m_t} - \theta\left[\Phi^{-1}(m_t) - \frac{z-\mu}{\sigma}\right] \tag{8}$$

where ϕ and Φ are the standard normal density and distribution functions, respectively. Equation (8) provides a relationship for the "z-score" of height (in relation to the standard that would prevail in the absence of mortality) in terms of the mortality rate, the survival cutoff (expressed in standard deviations from the mean), and the scarring parameter θ.

Figure 1 shows plots of average height from (8) against the mortality rate for a range of values of θ. We have assumed a value of *z* that is 2.5 standard deviations below the mean, so that the baseline mortality rate (when v_t is zero) is only 0.6 percent. Each graph therefore starts from a value of 6 for the mortality rate, with the resulting mild selection giving an average height of 1.8 percent of a standard deviation above the unselected mean. In these graphs, higher mortality is associated with higher height when scarring is unimportant (low θ) and with lower height when scarring is important (high θ). As the figure shows, for intermediate values of θ, the graph is non-monotonic in mortality, with scarring effects predominating at low mortality, and selection effects predominating at high mortality. The figure also shows that, at low mortality levels, scarring effects can make average heights extremely sensitive to changes in mortality patterns and even if there is no reversal, the scarring effects are gradually attenuated by selection as mortality rises. In Section 6 below, we shall examine the extent to which the worldwide distribution of heights matches Figure 1.

4. Data description

4.1. Height data

For ten European countries, Austria, Belgium, Denmark, Finland, Greece, Ireland, Italy, Portugal, Spain, and Sweden, height data were extracted from Garcia and Quintana-

Domeque (2006) who compute average heights using data from the European Community Household Panel (ECHP). The ECHP is a standardized annual longitudinal survey designed and coordinated by the European Commission's Statistical Office (EUROSTAT), which involves annual interviewing of individuals aged 16 and older from a representative panel of households (Peracchi, 2002).

Heights for the United States come from the National Health Interview Surveys (NHIS) for the years 1996, and 1998-2004. This NHIS is a series of cross-sections with no panel element. As in the European data, heights are self-reported. Height data for England are taken from the nationally representative Health Survey for England (HSE) for the years 1992-1999; unlike the other surveys, heights were measured by nurses, rather than self-reported. All calculations (except for Sweden, where the requisite data are unavailable, see Garcia and Quintana-Domeque, 2006) exclude those born outside of the country.

For the US and England, we compute average adult heights for each birth cohort from 1950 to 1980; for the rest of European countries average adult cohorts heights are taken from Garcia and Quintana-Domeque (2006). We exclude birth-cohorts born after 1980 in order to restrict ourselves to individuals who have fully attained their adult heights. In the ECHP data, the number of reported heights in the average varies from a low of 50 Danes in the birth-cohort of 1980 to a high of 1,358 Italians in the birth cohort of 1972; the effective sample sizes are substantially smaller because of the panel structure, with most individuals appearing three or four times, Garcia and Quintana-Domeque. For the English data, the average number of observations per cohort is 1,345, but the sample size for people born between 1975 and 1978 is small (218 per cohort, on average). In the US,

heights are averaged irrespective of race or ethnicity; the number of observations ranges from 4,667 for the 1961 cohort to 1,535 for the 1980 cohort. In all cases, we take men and women together; in order to avoid effects of the sex composition of the population we calculate average height as half the average height of men plus half the height average height of women. While it is not necessarily the case that the degree of sexual dimorphism is the same across countries, or is unaffected by environmental conditions in early life, this is not the topic of our analysis here, and we pool the data so as to obtain maximal precision on average heights by country and birth cohort.

Our height measures have both strengths and weaknesses compared with others in the literature. One strength is that the ECHP, NIHS, and HSE all provide nationally representative data, so that we are not limited to males or to male military conscripts. One weakness is that by identifying birth cohorts by age at (what is effectively) a single point of time, we are ignoring the effects of mortality on cohort height between the ages of 21 and 51, as well as any effects of health-selective emigration on the height of the resident cohort. We are also working with self-reported data, except for England. Comparisons of self-reported and directly measured heights in the United States, using the National Health Interview Survey (self-reported) and the National Health and Nutrition Examination Survey (directly measured), show that self-reported heights exaggerate measured height on average, and that the difference, which is greater for males than females up to age 70, increases with age, Thomas and Frankenberg (2002, Figure 1), Ezzati et al. (2006, Figure 2). However, these results also show little age-variation in the bias between 20 to 50, which may reasonably be interpolated to the European surveys;

even so, the directly measured English heights are likely to be somewhat understated relative to those from the other countries

4.2. GDP and mortality data

GDP data are taken from version 6.2 of the Penn World Table, Heston, Summers, and Aten (2006); we use the real (chained) value of GDP per capita in constant year-2000 purchasing power dollars. Although our height data are for England, we use GDP estimates for the United Kingdom: the inclusion of Scotland, Wales, and Northern Ireland in one set of estimates but not the other is unlikely to compromise our results. Postneonatal and neonatal mortality rates are taken from the 1953–1964 editions of the Annual Epidemiological and Vital Statistics (Part I: Vital Statistics and Causes of Deaths, WHO, Geneva) and from the 1965–1982 editions of the World Health Statistics Annual (Vol. I: Vital Statistics and Causes of Deaths, WHO, Geneva), supplemented where necessary by data from the electronic WHO mortality database (WHO, Department of Measurement and Health Information Systems Epidemiology and Burden of Disease, January 31, 2004). There is a substantial number of missing values, see Appendices A2 and A3 for a full description.

4.3 Main features of the data

Table 1 summarizes the height data. Although we shall use year-by-year observations in the analysis in the next section, there is substantial estimation uncertainty which induces year to year variation, so here we present five year averages (or six years for the birth cohorts 1950 to 1955.) Note again that we pool men and women; men are 10 to 11 centimeters taller than women on average. Males have higher postneonatal mortality (and

mortality generally) than do females, but we do not want to argue that this difference is in any way related to the height differences between men and women as adults.

The table shows that Denmark and Sweden are the two tallest countries, with Spain and Portugal the shortest. The inhabitants of richer countries are taller than the inhabitants of poorer countries, and Figure 2 shows the remarkably close correlation. Such findings are clearly consistent with (among other hypotheses) an important role for early life nutrition in adult height, and with greater availability of food in better-off countries. Table 1 also shows that, in the poorer, or more southerly, countries of Europe, height has been increasing across the birth cohorts from 1950 through to 1980, while in the richer and more northerly countries, height has either stagnated or increased for the earlier birth cohorts, and leveled off later. Figure 3 shows this pattern more clearly. The lower curve shows the average height by birth cohort for Greece, Italy, Portugal and Spain (each given equal weight), while the higher curve shows the unweighted average over the other eight countries. (The picture looks essentially the same if Ireland is moved to the lower group.)

Table 2 shows the five-year averaged data for postneonatal mortality (PNM) for those countries and periods where we have been able to locate the data. In 1950-55, PNM varied from 61 per 1,000 live births in Portugal to 5 per 1,000 live births in Sweden. It has fallen in all countries since then, to 11 in Portugal in 1976-80, and 2 in Sweden, a value that had also been attained by Finland, with Denmark not far behind. A rate of 2 deaths per thousand is presumably close to the minimum attainable. Already in 1950, the Swedes were close to this figure, while Portugal, Spain, Italy, and (presumably) Greece, still had a long way to go. Figure 4 shows the annual pattern of PNM averaged over the

same groups of countries as for heights in Figure 3, and shows its mirror image. Adults in the countries with high PNM were relatively short on average, and as their PNM rate fell, children born in the years of lower PNM were taller as adults. In the richer countries, with lower PNM to start and smaller improvements to make, heights were taller to start but did not grow over time. In the next section, we explore whether this timing matches on a country by country basis, and whether it survives competition with other explanations.

If Figure 2 is redrawn for neonatal mortality (NNM) in place of postneonatal mortality as shown in Figure 5, the two lines are much less far apart in the early years, and fall in parallel. There has been a great deal of progress in reducing NNM, for example through the use of neonatal intensive care units that help the survival of low birthweight babies, and this progress had shown no signs of slowing by 1980. PNM by contrast, is driven less by technological improvements, than by improving the disease environment and the ready availability of hospital care, something that had already been largely accomplished in the richer countries by the early 1950s.

5. Analysis of European and American heights

Table 3 presents a series of regressions in which height is the dependent variable. Column 1 shows that in the 316 pooled time-series cross-section observations for the 12 countries over 31 years of birth, variation in postneonatal mortality explains 62 percent of the variation in average heights. The parameter estimate is –0.16, so that a reduction in PNM by 20 per thousand, which is a modest improvement by historical standards, will increase average heights by 3.2 centimeters, which is more than most of the actual

increases shown in Table 1. The estimate is so high because we are forcing PNM to account for the time-series and cross-section simultaneously, and the inclusion of country fixed effects in column (2) cuts the estimated effect by a third to –0.10, which provides a closer match to the within-country time-series increases in height relative to the reductions in PNM. The inclusion of a time trend in column (3), with a secular increase in heights in all countries of a twentieth of a centimeter per year, or 1.5 centimeters over the thirty year period, reduces the estimated effect of PNM by a further third, to –0.06. This is likely too low, because some of the effect assigned to the time trend is presumably due to the secular reduction in PNM. Column (4) includes, in addition to the country fixed effects, a set of year fixed effects. This adds little to the explained variance over the time trend in column (3), and does not further change the size or significance of the estimated effect of PNM on heights.

Column (4) shows that the preliminary findings of Figures 3 and 4 carry through to a more thorough country-based analysis. By allowing for both year effects and country effects, the fact that PNM still exerts a significant negative effect on heights shows that the correlation is not simply a matter of common trends, one falling everywhere, and one rising everywhere. More than that, the cross-country patterns of the timing of the fall and stabilization of postneonatal mortality match (inversely) the cross-country patterns of the timing of the rise and stabilization of heights.

The right-hand panel of Table 3 looks at what happens when we introduce two other possible determinants of height, real income per head (measured by the log of real per capita GDP PPP), and neonatal mortality (NNM), both in the year of birth. Otherwise, we follow the same procedure as in the left-hand side of the table, sequentially introducing

country effects, a time (year of birth) trend, and a set of time effects. Column (8) shows that column (4) is essentially unaffected by the introduction of income and NNM. In the pooled time-series and cross-section, neither (the logarithm of) per capita GDP nor NNM are significantly different from zero, and neither the size nor the significance of PNM is much affected. For real GDP per capita, the insignificance persists in all specifications that contain country fixed effects, although in column (5), it plays a role in explaining cross-country variation in heights. Across columns (5) through (8), the size of the estimated coefficient is unstable from one specification to another. In columns (6) and (7), neonatal mortality attracts a significant coefficient, and it is possible that it, along with PNM, is linked to adult heights.

We now turn to the causes of PNM. It is not possible to do this in a comprehensive way, but at the price of reducing the number of data points to 297, we can split PNM into four components, mortality from pneumonia, mortality from intestinal disease, mortality from congenital anomalies, and mortality from other causes; Appendix A1 provides the definitions of each category. We also note that, if only because of uncertainty over diagnosis, information on cause of death is always likely to be less precise than the mortality data themselves. The large differences between high PNM and low PNM countries, for example Portugal and Italy on the one hand, and Sweden and the US on the other, lie in mortality from intestinal disease, followed by mortality from pneumonia. The Swedish postneonatal mortality rate from intestinal disease has been zero (or at least less than 0.5 per 1,000) since 1972. There are also substantial differences in the “other” category, but the differences in mortality rates from congenital abnormalities are small. Over time and within countries, all categories of PNM have fallen, with mortality from

congenital abnormalities falling the least, and mortality from pneumonia and intestinal disease falling the most rapidly, and in about equal measure from about 8 and 6 per thousand for the birth cohort of 1950 to 0.4 and 0.2 respectively for the birth cohort of 1980.

Table 4 repeats the sequence of regressions in Table 3, but with postneonatal mortality split into its four components. The most important result in the table is that PNM from pneumonia in infancy is a consistently significant predictor of adult height across all of the specifications, with the size of the estimated effect varying according to the specification in very much the same way as did the estimated effect of overall PNM in Table 3. In the regressions without country effects, PNM from intestinal disease is also significant—it is the component that varies most across countries—but its effect is less than a quarter of that of PNM from pneumonia, and it fades into insignificance once country dummies are included. Mortality from congenital diseases has a significant effect on adult height in the regressions with country dummies and no time effects or only a time trend. The penultimate row of the table shows that the estimated effects of PNM from different causes are statistically different, at least in the more parsimoniously parametrized models. Otherwise, the results in Table 4 are similar to those in Table 3, albeit with reduced statistical significance reflecting the smaller sample size and the fact that all of the components of PNM share downward trends, so that these regressions are pushing our data close to the limit of the information that they contain.

The finding that it is childhood respiratory disease that predicts adult height more strongly than childhood intestinal disease is somewhat surprising given the direct and obvious effects of intestinal disease on net nutrition. Even so, that pneumonia should

stunt growth is predicted by the work of Crimmins and Finch discussed in the background section, and our finding is supportive of their ideas. It also suggests a further refocusing of attention towards the effects of childhood disease on adult health and away from food and its retention.

6. Selection versus scarring

The European and American results can be directly interpreted in terms of the model of selection and scarring in Section 3. In particular, if we maintain the assumption that the underlying distribution of potential heights is normal, we can use equation (8). We assume that, in the absence of infant mortality, adult heights would have a mean of 176 centimeters, and a standard deviation of 6.0, which were the actual figures for Denmark for the cohorts born between 1976 and 1980. Given this, and given that the first term on the right-hand side is parameter free and so can be subtracted from the normalized mean height on the left-hand side, we can estimate the parameters θ and *z* by estimating (8) by linear regression. We therefore use data on $\bar{h}_t$ and m_t to estimate the equation

$$\frac{\bar{h}_t - \mu}{\sigma} - \frac{\phi[\Phi^{-1}(m_t)]}{1-m_t} = \theta\left[\frac{z-\mu}{\sigma}\right] - \theta\Phi^{-1}(m_t) \qquad (9)$$

so that the parameter θ is recovered as the coefficient on $\Phi^{-1}(m_t)$ and the *z*-score $(z-\mu)/\sigma$ or *z* itself from the estimated constant term given the estimate of θ.

The scarring parameter θ is estimated to be 1.18, and the disease-free cutoff for mortality is 3.12 standard deviations below the untruncated mean. A value of θ near one implies that the disease- or famine-based shift in potential heights in childhood persists unmodified into adulthood. Although some later magnification of the childhood effect

seems theoretically possible, we expect θ to be less than the full persistence value. If we follow the general scheme of Table 3, and add in country fixed effects, the estimate of the scarring parameter falls to 0.77, and the cutoff is 3.16 standard deviations below the mean. With a time-trend, we get 0.64 and 3.42, and these values change little if we allow year effects. As was the case for the regressions in Table 3, the time trend (or year effects) almost certainly absorbs some of the effect of mortality decline, causing the scarring effect to be underestimated. The fits of these equations are only slightly inferior to those obtained by adding a quadratic in PNM to the original regressions. When the logarithm of real income is added, it is insignificantly different from zero in the absence of country fixed effects, though it becomes significant in their presence, essentially because within-country year to year fluctuations in income are correlated with within-country variations in cohort heights.

Given the skeletal nature of the scarring and selection model, we view these results as supportive of its predictions. However, there is a limited range of mortality variation in Europe and North America after 1950, and the curvature of the relationship between adult height and postneonatal mortality rests heavily on the experience of the highest mortality countries, particularly Portugal. To investigate the model further, we turn to a different source of data with a much wider range of mortality experience.

The new information on heights is from women who were measured in the international system of Demographic and Health Surveys (DHS). These surveys, whose main subject is reproductive and child health, have measured the heights of women aged 15 through 49 in more than 40 countries since the late 1990s. The countries that we use are listed in Appendix A3; there are 27 in Africa, 8 in Latin America and the Caribbean,

3 (India, Bangladesh, and Nepal) in South Asia, and 5 in Central Asia (which we group with the Egyptian and Moroccan surveys from Africa to form a Middle East and North African region.) As is to be expected, other data availability for these countries is not as good as in Europe and the United States. Although we have annual data on income per capita in PPP terms from the Penn World Table which can be matched to the adult height of each birth cohort in most of the surveys, we do not have any consistent international data on postneonatal mortality and, indeed, for many of these countries, such data do not exist. Instead, we turn to the *infant mortality* rates provided by the United Nations population division which are available for five year intervals after 1950. (Childhood mortality, up to age 5, is often more accurately measured than infant mortality rates because of uncertainty about birth dates, but is not available from the UN, and World Bank data begin only in 1960.) We also note that, compared with the rich countries of Europe and North America, the poor countries have much greater mortality between infancy and adulthood, when heights are measured, something that is not taken into account in the model.

The switch from postneonatal to infant mortality, and the move from rich to poor countries changes the way in which we need to think about the effects of income and disease on mortality rates, as well as on adult heights. For the rich countries after 1950, postneonatal mortality was largely a reflection of the disease environment, not of gross nutritional adequacy in terms of food intake, which was unlikely to have been a limiting factor. Infant mortality includes both neonatal and postneonatal mortality, and in poor countries in Africa and South Asia, it is almost certainly affected by the availability of food, as well as by disease, as well as by interactions between them, Scrimshaw, Taylor,

and Gordon (1968). Food inadequacy makes children more likely to succumb to at least some infections, and the growth inhibition from the infection will be made up later only if there is adequate nourishment. Hence, and unlike the case in the rich countries, infant mortality rates capture the effects *both* of disease and of food inadequacy (or low income), while food inadequacy itself is likely to affect adult height even conditional on infant or childhood mortality.

Figure 6 shows a plot of women's heights against the infant mortality rate (pooling the DHS with the European and North American data); there are 288 observations across countries and five year periods. Each point is the mean of women's height in a five-year birth cohort versus the infant mortality rate for the same period; we use data only on women aged 20 or older because women in poor countries sometimes attain adult height at later ages than in rich countries. (Indeed, age 20 is too young for South Asia, but an older cutoff would eliminate many otherwise valid data points.) Imposed on the scatterplot, we show two lines. The broken line is a non-parametric regression fit, which shows height declining with infant mortality in the year of birth throughout most of the range, but with an upturn beginning at levels of infant mortality above 150 per thousand. The solid line shows the fitted values of the selection and scarring model, again using the Danish standard, and with fitted estimates for θ and z of 1.38 and 3.59 standard deviations below the mean, respectively. This value of θ is too high to give non-monotonicity, even at high values of infant mortality, but it otherwise provides a reasonable fit to the general pattern of the data.

As was the case for Europe and the US, the fit is improved and the estimate of θ reduced (to 0.66) if we include dummies for five regions of the world (Europe/US,

Middle East and North Africa, South Asia, Latin America and Caribbean, and sub-Saharan Africa). The regional dummies are important, and given the infant mortality levels in their year of birth, women in Latin America and in South Asia are shorter and women in sub-Saharan Africa taller than would be expected. This is the adult version of the child malnutrition–child mortality puzzle previously noted by Klasen (2006). Within the model, the regional effects are interpretable as regional differences in potential height, possibly through genetic differences, although it is perhaps more likely that the adaptation of height to the tightening or loosening of environmental constraints may take many generations. There are limits to the size of children that small women can bear, and it has been argued that the century and a half of secular increase in heights in Europe, which was both large and too rapid to be genetically based, was simply the time taken to adjust to the better environment after 1850, Cole (2000).

If we add income to these height regressions, there are different effects depending on which other factors are included, and as expected, there are indications of interactions between income and infant mortality rates in some specifications. The addition of income does little or nothing to reduce the importance of the regional dummies nor to explain the differences in adult height between Africa and Asia. Nor does income reduce the marked heteroscedasticity in Figure 6, with the scatter of adult heights around the regression much larger in the countries with high mortality. Again, much of this comes from Africa, where there is enormous diversity of average heights across countries, presumably reflecting local nutritional, environmental, and disease conditions (or even genetic differences) that are not captured either by income per capita or by infant mortality rates.

7. Discussion and conclusions

We have used data on 31 birth-cohorts from eleven European countries and from the United States to investigate the early childhood determinants of adult height. We find that infant mortality, particularly postneonatal mortality, predicts the adult height of the birth cohort that survives it. Given that postneonatal mortality is a sensitive indicator of the disease environment in the first year of life, these results support accounts in which some form of "scarring" in infancy negatively affects lifetime health, as marked by adult height. In these countries, the scarring effect of childhood disease dominates any possible height-based selective mortality in childhood that would induce a positive relationship between disease in early life and adult health.

In our pooled cross-section and time-series data from Europe and the US, variations in postneonatal mortality can explain more than sixty percent of the variation in adult heights, and the fall in postneonatal mortality can account for almost all of the increase in adult heights between those born in 1950 and those born in 1980. More importantly, postneonatal mortality displays a distinct historical pattern, falling to its minimum attainable level, and then flattening out. This pattern is common across countries, but its timing differs from country to country. The international variation in the timing of the pattern of postneonatal mortality closely matches the country by country timing in the rise and flattening out of average heights. Fluctuations in national income play no such role, even though national income and average height are closely correlated in the cross-section.

We also find that the component of postneonatal mortality that most closely predicts adult heights is mortality from pneumonia, not mortality from congenital anomalies,

mortality from intestinal disease, or mortality from other causes. Our finding is consistent with the literature that argues that childhood diseases elicit inflammatory responses that make heavy demands on nutrition, and that compromise ultimate growth. The lack of any response to per capita GDP, albeit at best a weak measure of nutrition, suggests a relatively weak role for gross nutrition.

The situation is different once we examine poor and rich countries simultaneously. Among the poorest, highest mortality countries, we find evidence for distinct effects on adult height of both disease and of food availability, as represented by income; these results suggest that early childhood development is constrained both by food and by disease in poor countries while, in now rich countries since 1950, the food constraint has not been important. More notably, albeit with evidence that is best viewed as suggestive, we find that selection may be stronger than scarring at high levels of mortality and low levels of income, which would explain at least part of the African height paradox, that Africans are relatively tall in spite of extremely unfavorable income and disease environments. There is much stronger evidence that the effect of infant mortality on adult height is weaker at higher mortality rates, which we attribute to the fact that selection is stronger at high mortality.

Our results are also relevant for understanding the link between adult height and late-life chronic disease, and how that correlation might change historically and geographically. There is evidence that height is protective against cardiovascular disease in late-life, which is consistent with an interpretation in which both adult height and late life disease are caused by scarring in childhood. It is also possible that childhood disease, the quantity v_t in the model of Section 3, has a different effect on adult height from its effect on late

life disease, although there will still be an apparently protective effect of height on disease reflecting the role of childhood disease on both. If so, and if it is true that, in the highest mortality environments, selection outweigh scarring on adult heights, the negative correlation between adult height and late-life chronic disease may be eliminated or even reversed. In rich, low infant mortality countries, taller people have a survival advantage, even conditional on obesity and other risk factors, but this advantage may not exist in poor, high mortality countries now, and may not have existed historically in the now rich countries. This is because the survivors of extreme negative environments, although selected for both height and health, are still heavily scarred by their childhood environment, and it may be the scarring more than the selection, that predicts late-life chronic disease, even though both affect height in the same way. Unfortunately, we do not have data on mortality from cardiovascular disease from sub-Saharan Africa on which this prediction might be tested. Even so, recent work by Costa (2002, 2004) and by Costa, Helmchen, and Wilson (2007) has shown or inferred very high levels of late-life morbidity among veterans of the 19th century Union Army in the US, including morbidity associated with arteriosclerosis and ischemic heart disease, at least some of which the authors attribute to early life infections. Interestingly, and consistently with our account here, Costa, Helmchen and Wilson find that adult height at enlistment did not predict arteriosclerosis in late life among the survivors. In the historical record, as in the poorest countries now, the prevalence of late-life chronic disease is masked by the importance of infectious diseases. But it is quite possible that the age-specific prevalence of chronic disease was higher in the past than now, and is currently high in some of the currently poorest countries of the world.

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Table 1: Average heights around 2000 for birth cohorts
(men and women together, centimeters)

Year of birth	1950-55	1956-60	1961-65	1966-70	1971-75	1976-80
Austria	171	172	173	172	173	173
Belgium	170	171	171	173	173	174
Denmark	174	173	174	175	175	176
England	169	170	170	170	170	170
Finland	171	172	173	172	172	172
Greece	169	170	171	171	172	172
Ireland	169	169	170	170	171	171
Italy	167	168	169	169	170	172
Portugal	164	165	165	165	167	168
Spain	166	166	167	169	170	171
Sweden	172	173	174	174	174	174
U.S.	171	172	172	172	172	172

Source: Authors' calculations from the National Health Interview Survey for the US, the Health Survey of England, and Garcia and Quintana-Domeque (2006) for the other countries.

Table 2: Average postneonatal mortality, mortality per 1,000 live births

Year of birth	1950-55	1956-60	1961-65	1966-70	1971-75	1976-80
Austria	23	16	8	7	6	5
Belgium	18	13	9	7	6	4
Denmark	10	6	5	4	3	3
England	9	6	6	6	6	5
Finland	14	9	4	3	2	2
Greece	..	27	18	12	7	5
Ireland	18	11	9	9	..	5
Italy	31	23	16	11	6	4
Portugal	61	57	48	35	21	11
Spain	47	..	..	..	6	5
Sweden	5	4	3	2	2	2
U.S.	8	7	7	6	5	4

Notes: .. indicates not available for any year in the span; otherwise averages are taken over all available data.
Source: World Health Organization.

Table 3: Regressions of height on postneonatal mortality and other variables

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
PNM	–0.161	–0.100	–0.057	–0.057	–0.121	–0.048	–0.052	–0.044
	(0.007)	(0.007)	(0.009)	(0.009)	(0.012)	(0.013)	(0.014)	(0.017)
NNM	--	--	--	--	–0.029	–0.080	–0.069	–0.047
					(0.026)	(0.028)	(0.030)	(0.035)
Ln(GDP)	--	--	--	--	0.993	0.542	0.149	0.833
					(0.299)	(0.517)	(0.678)	(0.809)
Year of Birth	--	--	0.054	--	--	--	0.018	--
			(0.008)				(0.018)	
Country dummies?	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Year dummies?	No	No	No	Yes	No	No	No	Yes
R^2	0.623	0.898	0.914	0.923	0.640	0.916	0.916	0.924
Sample size	316	316	316	316	316	316	316	316

Notes: Heteroskedasticity robust standard errors are reported in parentheses.

Table 4: Regressions of height on postneonatal mortality by cause and other variables

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
PNM								
Pneumonia	–0.531	–0.228	–0.126	–0.137	–0.408	–0.099	–0.101	–0.105
	(0.053)	(0.041)	(0.042)	(0.046)	(0.066)	(0.046)	(0.048)	(0.053)
Intestinal	–0.082	0.004	–0.014	–0.008	–0.099	–0.021	–0.021	–0.014
	(0.037)	(0.028)	(0.026)	(0.027)	(0.038)	(0.027)	(0.027)	(0.027)
Congenital	–0.110	–0.406	–0.165	–0.168	0.097	–0.248	–0.242	–0.207
	(0.161)	(0.176)	(0.090)	(0.098)	(0.150)	(0.099)	(0.099)	(0.106)
Other	–0.034	–0.125	–0.083	–0.083	0.014	–0.082	–0.084	–0.063
	(0.046)	(0.034)	(0.032)	(0.034)	(0.049)	(0.040)	(0.041)	(0.043)
NNM	--	--	--	--	–0.028	–0.089	–0.085	–0.063
					(0.030)	(0.027)	(0.031)	(0.036)
Ln(GDP)	--	--	--	--	0.832	0.006	–0.102	0.585
					(0.347)	(0.574)	(0.752)	(0.916)
Year of Birth	--	--	0.042	--	--	--	0.005	--
			(0.009)				(0.020)	
Country dummies?	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Year dummies?	No	No	No	Yes	No	No	No	Yes
R^2	0.651	0.907	0.913	0.922	0.658	0.915	0.915	0.923
F-test	16.78	12.26	2.95	3.03	11.67	2.48	2.46	1.87
Sample size	297	297	297	297	297	297	297	297

Notes: Heteroskedasticity robust standard errors are reported in parentheses. The *F*-tests in the last panel are tests of the hypothesis that the coefficients on the four categories of PNM are identical; those in columns 1, 2, and 5 are significant at better than one percent, those in columns three and four at better than five percent, and those in the last three columns are insignificant.

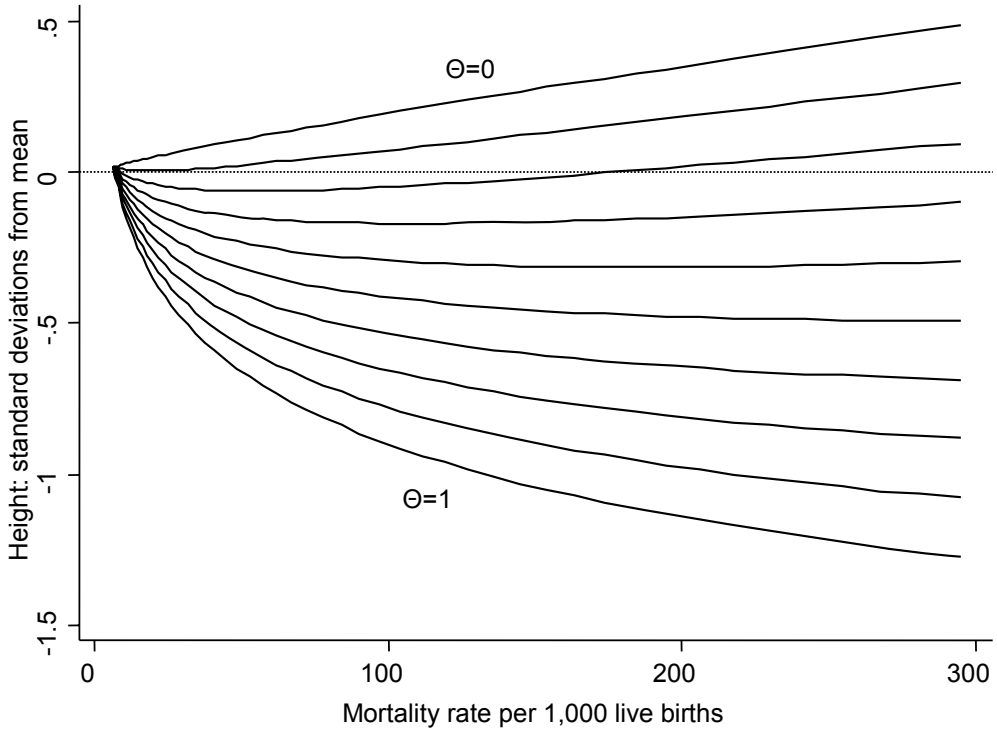

Figure 1: Theoretical deviation of height in standard deviations from mean of parent distribution in relation to mortality rate

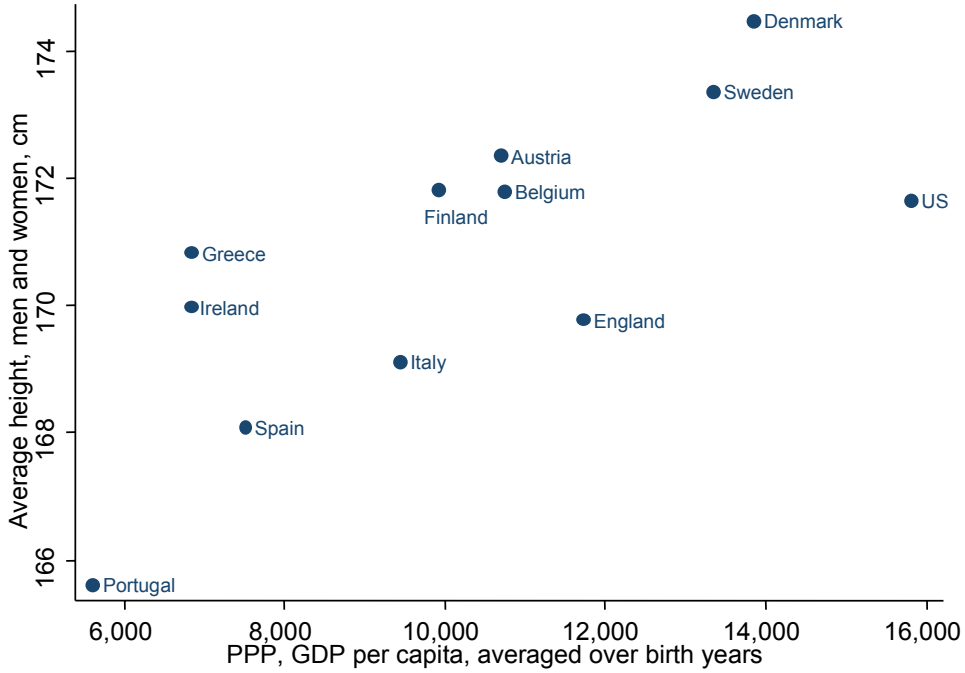

Figure 2: Adult height and average real GDP per capita, averaged over matching dates of birth

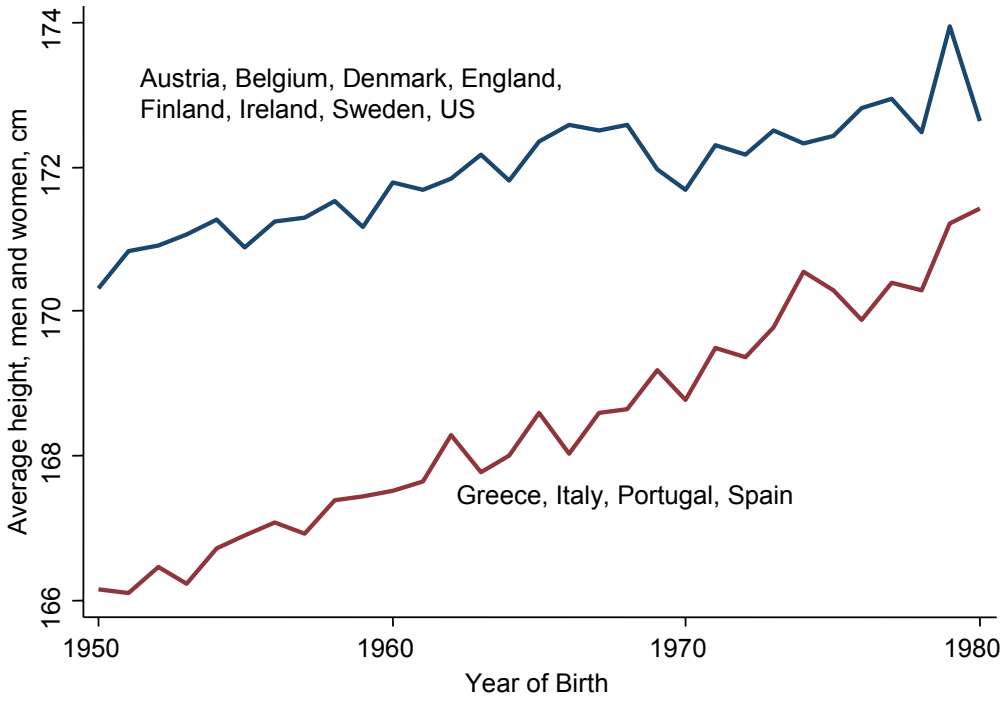

Figure 3: Average height for two groups of countries

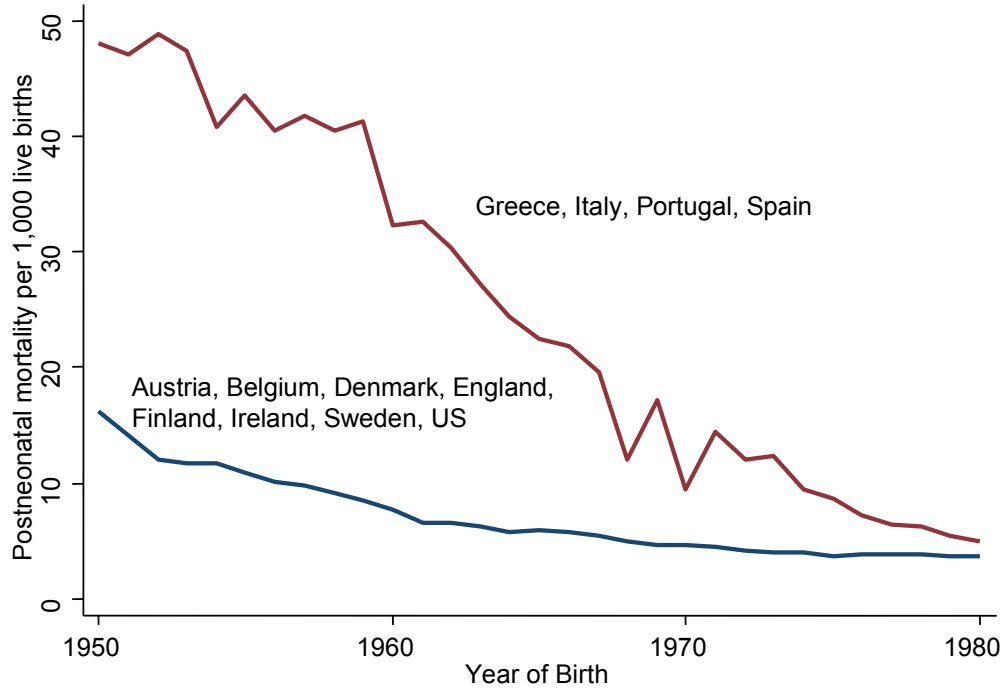

Figure 4: Postneonatal mortality for two groups of countries

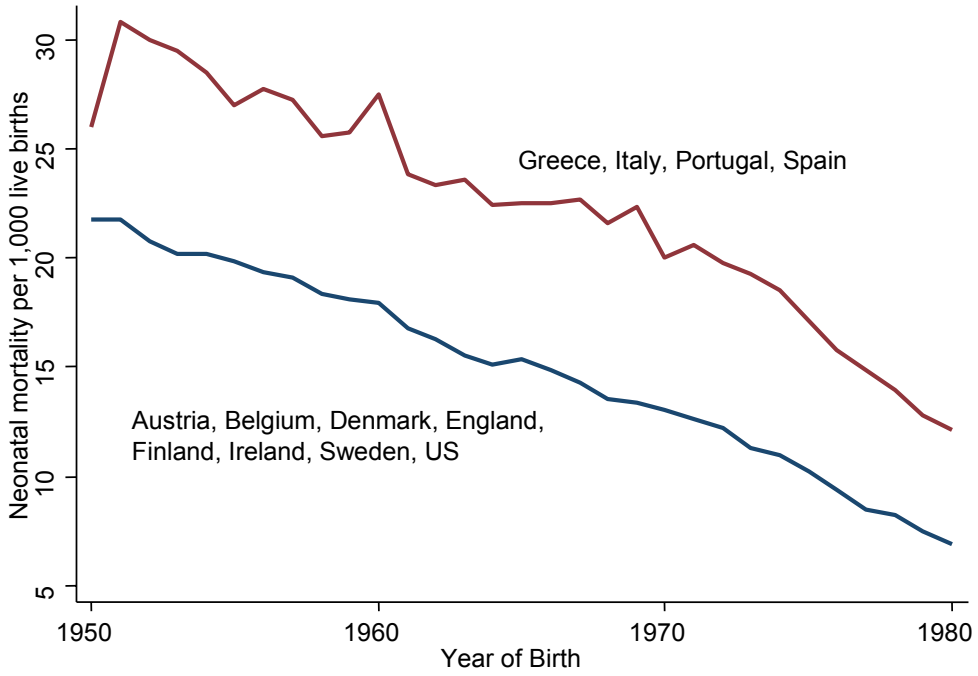

Figure 5: Neonatal mortality for two groups of countries

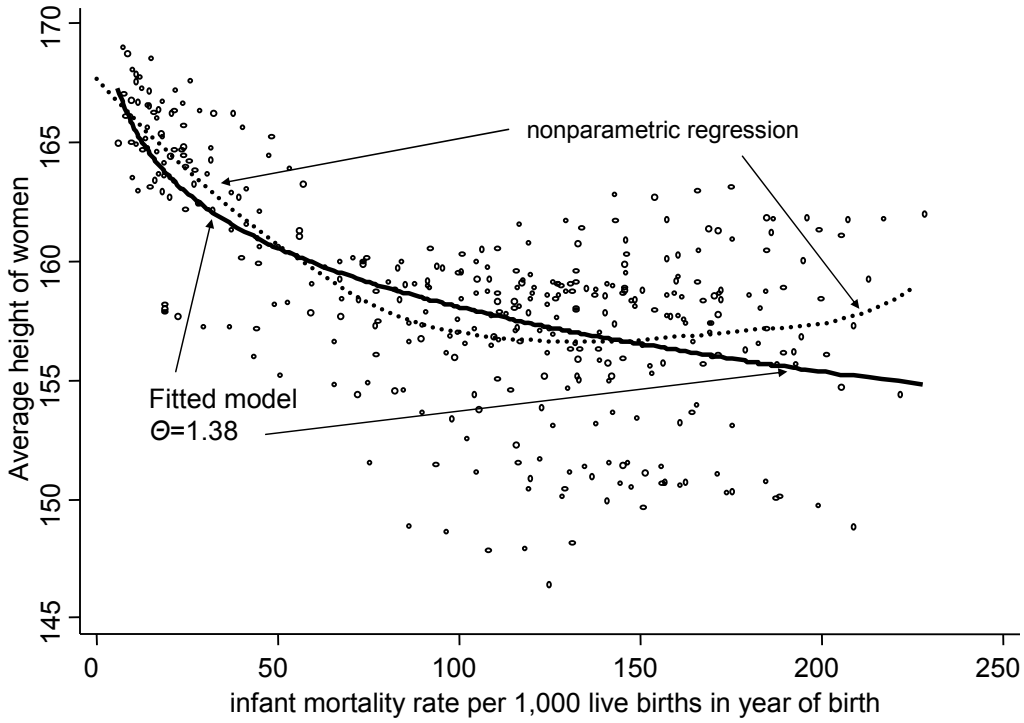

Figure 6: Average women's heights and infant mortality rates around the world

APPENDIX

A1. CLASSIFICATIONS OF PNM CAUSES

PNM from Pneumonia (Cause 1):
ICD5: Bronchopneumonia (107), Lobar pneumonia (108), Pneumonia (unspecified) (109)
ICD7: Bronchopneumonia (491), Lobar pneumonia (490), Primary atypical pneumonia (492), Pneumonia, Other and unspecified (493)
ICD7A: Bronchopneumonia (A090), Lobar Pneumonia (A089), Primary atypical, Other and unspecified pneumonia (A091)
ICD8A: Viral Pneumonia (A091), Other Pneumonia (A092)
ICD9B: Pneumonia (B321)

PNM from Intestinal Disease (Cause 2):
ICD5: Enteritis and diarrhea with/without mention of ulceration of the intestines (except duodenum) (119), Enteritis and diarrhea without mention of ulceration, or ulceration of the intestines (except duodenum) (120)
ICD7: Gastritis and duodenitis (543), Gastro-enteritis and colitis, except ulcerative, age 4 weeks and over (571), Chronic enteritis and ulcerative colitis (572)
ICD7A: Gastritis and duodenitis (A101), Gastro-enteritis and colitis, except diarrhea of the newborn (A104)
ICD8A: Enteritis and other diarrhoeal diseases (A005)
ICD9B: Intestinal infectious diseases (B01) (Cholera, Typhoid fever, Shigellosis, Food poisoning, Amoebiasis, Intestinal infections due to other specified organism, Ill-defined intestinal infections, other)

PNM from Congenital Anomalies (Cause 3):
ICD5: Congenital hydrocephalus (157a), Spina bifida and meningocele (157b), Congenital malformation of heart (157c), Monstrosities (157d), Congenital pyloric stenosis (157e), Cleft palate and harelip (157f), Imperforate anus (157g), Cystic disease of kidney (157h). Congenital malformations of the central nervous system (157ia), Congenital malformations of the circulatory system (157ib), Congenital malformations of the digestive system (157ic), Congenital malformations of the genitor-urinary system (157id), Other stated malformations (157ie), Congenital malformations (unspecified) (157j)
ICD7: Monstrosity (750), Spina bifida and meningocele (751), Congenital hydrocephalus (752), Other congenital malformations of the nervous system and sense organs (753), Congenital malformations of the circulatory system (754), Cleft palate and harelip (755), Congenital malformations of the digestive system (756), Congenital malformations of the genitor-urinary system (757), Congenital malformations of bone and joint (758), Other and unspecified congenital malformations, not elsewhere classified (759)
ICD7A: Spina bifida and meningocele (A127), Congenital malformations of circulatory system (A128), All other congenital malformations (A129)
ICD8A: Spina bifida (A126), Congenital anomalies of heart (A127), Other congenital anomalies of circulatory system (A128), Cleft palate and cleft lip (A129), All other congenital anomalies (A130)
ICD9B: Congenital anomalies (B44), (Spina bifida and hydrocephalus, Other deformities of central nervous system, Congenital anomalies of heart and circulatory system, Cleft palate and cleft lip, Other deformities of digestive system, Undescended testicle, Congenital dislocation of hip, Other congenital anomalies of musculoskeletal system, Other).

SWEDEN 1950
Causes of death according to the Swedish list of 1931: PNM from Pneumonia: Pneumonia and bronchopneumonia (3520-3530) PNM from Intestinal Disease: Diarrhea and enteritis (1150, 4020) PNM from Congenital Anomalies: Congenital malformations (0001)

A2. TABLE ON MISSING CAUSES

	1950	1951	1952	1953	1954	1955	1956	1957	1958	1959
Austria	A	A	A	B	B	B	C	C	C	C
Belgium	..	..	..	..	B	B	C	C	C	C
Denmark	A	B	B	B	B	B	C	C	C	C
England and Wales	B	B	B	B	B	B	C	C	C	C
Finland	B	B	B	B	B	B	C	C	C	C
Greece	..	..	..	..	..	..	..	..	..	..
Ireland	B	B	B	B	B	B	C	C	C	C
Italy	A	B	B	B	B	B	C	C	C	C
Portugal	A'	A'	A	B	B	B	C	C	C	C
Spain	A	..	..	..	..	..	..	..	..	..
Sweden	S	B	B	B	B	B	C	C	C	C
U.S.	B	B	B	B	B	B	C	C	C	C

	1960	1961	1962	1963	1964	1965	1966	1967	1968	1969
Austria	C	..	..	..	..	C	C	C	C	D
Belgium	C	C	C	C	C	C	C	C	D	D
Denmark	C	C	C	C	C	C	C	C	C	D
England and Wales	C	C	C	C	C	C	C	C	D	D
Finland	C	C	C	C	C	C	C	C	C	D
Greece	C	C	C	C	C	C	C	C	D	D
Ireland	C	C	C	C	C	C	C	C	..	..
Italy	C	C	C	C	C	C	C	C	D	D
Portugal	C	C	C	C	C	C	C	C	..	C
Spain	..	..	..	..	..	..	..	..	..	..
Sweden	C	C	C	C	C	C	C	C	C	D
U.S.	C	C	C	C	C	C	C	C	D	D

	1970	1971	1972	1973	1974	1975	1976	1977	1978	1979	1980
Austria	D	D	D	D	D	D	D	D	D	D'	E
Belgium	D	D	D	D	D	D	D	D'	..	..	..
Denmark	D	D	D	D	D	D	D	D	..	D'	D'
England and Wales	D	D	D	D	D	D	D	D	D'	E	E
Finland	D	D	D	D	D	D	D'	D'	D'	..	..
Greece	D	D	D	D	D	D	D	D	D	E	..
Ireland	..	..	..	..	..	..	D'	D'	D'	..	..
Italy	D	D	D	D	D	D	D'	D'	D'	..	..
Portugal	..	D	D	D	D	D	D'	D'	D'	D'	..
Spain	..	..	..	..	..	..	D	D'	D'	..	..
Sweden	D	D	D	D	D	D	D	D	D	D'	D'
U.S.	D	D	D	D	D	D	D	D	D'	..	..

Notes:		
	A	List of 1938
	A'	List of 1929
	S	Swedish List of 1931
	B	List of 1948
	C	List of 1955
	D	List of 1965
	D'	ICD 8A
	E	ICD 9

A3. NOTES ON MISSING CAUSES

1950 Austria: Causes of death are only available at the Infant Mortality Rate level. **Spain:** "Congenital Malformations" (157) and "Diseases peculiar to the first year of life (158-161)" are reported together. Hence, this observation is discarded. **Portugal:** "Congenital Malformations" (157) and "Diseases peculiar to the first year of life (158-161)" are reported together. Hence, this observation is discarded.

1951 Austria: Causes of death are only available at the Infant Mortality Rate level. **Portugal:** "Congenital Malformations" (157) and "Diseases peculiar to the first year of life (158-161)" are reported together. Hence, this observation is discarded.

1952 Finland: Cause 2 is only available at the Infant Mortality Rate level. **Ireland:** Cause 2 is only available at the Infant Mortality Rate level.

1955 Austria: Cause 2 is missing. **England and Wales:** Cause 2 is only available at the Infant Mortality Rate level.

1956 Austria and Ireland: Cause 2 is missing.

1961 and 1964 Ireland: Cause 2 is only available at the Infant Mortality Rate level.

1965 Austria and Ireland: Cause 2 is only available at the Infant Mortality Rate level.

1966 Ireland: Cause 2 is only available at the Infant Mortality Rate level.

1967 England and Wales: Cause 2 is only available at the Infant Mortality Rate level.

1972, 1973, 1975 Portugal: Cause 3 is only available at the Infant Mortality Rate level.

A4. COUNTRY AND YEAR LISTING OF DHS SURVEYS

Sub-Saharan Africa: Burkina Faso, 2003, Benin 2001, Central African Republic 1994, Cote d'Ivoire 1998, Cameroon 2004, Chad 2004, Comoros 1996, Ethiopia 2000, Gabon 2000, Ghana 2003, Guinea 1999, Kenya 2003, Lesotho 2004, Madagascar 1997, Mali 2001, Malawi 2004, Mozambique 2003, Nigeria 2003, Niger 1998, Rwanda 2000, Togo 1998, Tanzania 1998, Uganda 1995 and 2000, Zambia 2001, Zimbabwe 1999

Middle-East and North Africa: Armenia 2000, Egypt 1995 and 2000, Kazakhstan 1999, Kyrgyz Republic 1997, Morocco 2003, Turkey 1998, Uzbekistan 1996.

Latin America and Caribbean: Bolivia 1993, 1998 and 2003, Brazil 1996, Colombia 1995, 2000 and 2004, Dominican Republic 1996, Guatemala 1998, Haiti 1994 and 2000, Nicaragua 2001, Peru 1996 and 2000.

South Asia: Bangladesh 2004, India 1999, Nepal 2001.