

mortality rates in their years of birth; the second regresses the heights of these adults on pre-adult mortality rates in their year of birth; and the third regresses the height-for-age  $z$ -scores of individual children from two rounds of India's Demographic and Health Survey on state-survey round level measures of neonatal and postneonatal mortality. Section 4.5 discusses the results, as well as the hypothesis that variation in neonatal mortality proxies for state level variation in maternal net nutrition. Section 4.6 concludes.

## **4.2 Context**

### **4.2.1 Early life determinants of height in developed countries**

In Europe, the relationships between height and the early life environment have been examined in the context of select groups, as well as at the population level. Schmidt et al. (1995) document a relationship between the adult heights of men who were conscripted in European armies and postneonatal mortality in the year of birth. Bozzoli et al. (2009) provide evidence for a similar relationship in the general populations of European countries and the United States for cohorts born between 1950 and 1980. They show that the relationship between adult height and postneonatal mortality is robust to a variety of controls, including neonatal mortality, log of GDP in the cohort's year and country of birth, and country and year fixed effects. Hatton (2011) finds a robust correlation between the heights of school children born between 1910 and 1950 in Britain and the infant mortality rates that prevailed when those children were between two and four years old.

All three of these papers posit that the mechanism linking height and early life mortality is the disease environment in early childhood. In Bozzoli et al. (2009), postneonatal deaths from pneumonia and diarrhea, diseases which are known to lead to stunting in childhood, correlate with adult heights, though death rates from pneumonia predict heights more strongly. None of the papers finds evidence of an effect of neonatal mortality on adult heights.

In these contexts, one would not expect adult height to be strongly influenced by factors determining neonatal mortality. Whereas in developing countries, temporal or regional differences in neonatal mortality might be due to variation in maternal nutritional deprivation during pregnancy, in developed countries, differences in the availability of life saving technology for premature infants would play a far more important role. Research on the relationship between maternal nutrition and infant outcomes in developed countries tends to focus on overweight and obese women, rather than on undernourished women.

Bozzoli et al. (2009) test for but do not find evidence of an effect of income on adult heights in Europe. The authors remind readers that their results do not rule out income or nutrition related constraints on adult heights for Western cohorts born pre-1950, nor do they rule out such a constraint on adult height in developing countries.

#### **4.2.2 Early life determinants of height in developing countries**

Silventoinen (2003) posits that the determinants of variation in height are different in developed and developing countries, and in particular, that compared to developed country settings, environmental variation (as opposed to genetic variation) in height is relatively large. This is because environmental insults to childhood growth are more severe in developing countries.<sup>3</sup>

Many factors determine heights in developing country and pre-industrial settings, and higher levels of development do not always lead to growth in physical stature (Komlos, 1998, 2003). However, at low levels of development, additional income may influence height through increasing calorie intake or the quality of the diet. Fogel (2004) discusses relationships between income, calorie availability and body size. In particular, Fogel proposes a “technophysio evolution” of body size corresponding with calorie availability, which in many contexts increases when income increases. The importance of shocks to income and

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<sup>3</sup>Indeed, Spears (2013) shows that the correlation between mothers’ heights and children’s heights are lower in India and sub-Saharan Africa than in the United States.

nutrition as determinants of early life height and health have also been demonstrated in developing country settings (Maccini and Yang, 2009; Bhalotra, 2010).

Empirical evidence suggests that the early life disease environment influences height in impoverished settings. Akachi and Canning (2010) find that with the exception of sub-Saharan Africa, where the authors hypothesize that improvements in infant mortality have not been importantly due to reductions in morbidity, changes in adult heights correlate with changes in infant mortality rates in developing regions. Using data from three European populations in the 1800s, Crimmins and Finch (2006) find relationships between adult height and pre-adult mortality<sup>4</sup> measured when a cohort was a year old. Additionally, Spears (2013) suggests that sanitation coverage, a measure of the fecal pathogens to which a child is exposed, is a strong predictor of children's heights in modern developing countries.

Finally, pre-natal conditions may also contribute to stunting in situations of scarcity. There is substantial evidence that maternal nutrition promotes birth weight in developing countries (Kusin et al., 1993), and that birth weight is an important predictor of height (Binkin et al., 1988; Adair, 2007). A longitudinal study from Brazil finds that low birth weight is correlated with low pre-pregnancy weight (Barros et al., 1992). Kramer (1987) reviews the literature on birth weight, and concludes that, in addition to low pre-pregnancy weight, low weight gain during pregnancy additionally correlates with low birth weights in developing countries. Hytten (1979) discusses this relationship in the context of food supplements in pregnancy, which increase birth weights where women are undernourished.

Adair (2007) establishes links between birth size and adult height in a sample from the Philippines. Binkin et al. (1988) show that two causes of low birth weight—*intrauterine growth retardation* and *prematurity*—are both associated with low adult heights, though the relationship is stronger for *intrauterine growth retarded* infants than for *premature*

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<sup>4</sup>The measures included  ${}_5q_0$ ,  ${}_5q_5$  and  ${}_5q_{10}$ . These are the probability of dying between the ages of 0 and 5, between 5 and 10, and 10 and 15 years old respectively.

infants.<sup>5</sup> Behrman and Rosenzweig (2004) show that differences in birth weight between identical twins predicts differences in height. Finally, Kusin et al. (1992) present experimental evidence from Indonesia that supplementation during pregnancy improves postnatal growth—that is, growth from age zero to age five. These findings are particularly important for India, where discrimination against women of child-bearing age, even in food consumption, is well documented (Jeffery et al., 1988; Das Gupta, 1995; Palriwala, 1993).

### 4.2.3 Causes of early life mortality in India

Cause of death data help identify those early life conditions that both kill children and stunt their growth, and those causes of death that are unlikely to also be, or to proxy for, major determinants of height. What are the leading causes of early life mortality in India?

Table 4.1 summarizes the results of the Million Deaths study, a national study of cause of death that was conducted in 2005 (Million Death Collaborators, 2010). Prematurity/low birth weight was the second leading cause of death at all ages and the leading cause of neonatal mortality, responsible for over a third of neonatal deaths. Neonatal infection was the second leading cause of neonatal death; according to Million Death Collaborators (2010), neonatal infection comprises neonatal pneumonia, septicemia and meningitis. Birth asphyxia and trauma are the third cause of neonatal death. Together these three causes account for about 80% of neonatal death. Table 4.1 also shows cause of death for children between 1 and 59 months of life. The leading causes of child mortality are pneumonia and diarrheal diseases. A number of other infectious diseases comprise a large fraction of the remaining child mortality.

Among these causes of early life mortality, which reflect conditions that would also affect height? As outlined in section 4.2.2, there is clear evidence that low birth weight is importantly caused by poor pre-natal nutrition, which is prevalent in India, and which

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<sup>5</sup>Villar and Belizán (1982) provide evidence that the relative contribution of intrauterine growth retardation to low birth weights is greater in developing countries than in developed ones.

also affects child and adult height. We would not expect birth asphyxia and trauma to cause stunting later in life. The extent to which the neonatal infections identified by the Million Deaths Study as important causes of death affect height is unclear. These infections are likely to be fast-acting, and are typically acquired before or during birth, or shortly thereafter (Duke, 2005).<sup>6</sup> While these early infections may have consequences for the heights of children who survive them, there is little evidence to suggest that they sap energy for growth in the way that postneonatal infections like respiratory and gastrointestinal infections do. In contrast, there is clear evidence that both respiratory and gastrointestinal infections impact height. Victora et al. (1990) and Bozzoli et al. (2009) find evidence that respiratory infections such as pneumonia stunt height, and Checkley et al. (2008) discuss the role of diarrheal disease in stunting.<sup>7</sup>

Because neonatal mortality is a particular focus on this paper, it would be useful to know whether the major causes of neonatal death in the 1970s and early 1980s were the same as in 2005, when the Million Deaths study was conducted. This is a difficult question to answer precisely due to lack of high quality cause of death data from this period. However, a cause of death study conducted by the Office of the Registrar General (ORG) in major Indian states in 1978 found that tetanus was the most common cause of neonatal mortality (Government of India, 1979). In contrast, the Million Deaths Study of 2010 found a death rate from neonatal tetanus of only 1.2 per 1000 live births. This suggests that the importance of neonatal tetanus has declined dramatically between the time in which the adults studied here were infants, and when the children studied here were infants.

Despite the importance of neonatal tetanus in determining the mortality rates for the adult cohorts I study, it is unlikely to have been an important cause of their stunting. In the

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<sup>6</sup>Indeed, in a 1996-2003 trial of home based neonatal sepsis management in Maharashtra, India, researchers found that over four fifths of deaths from neonatal sepsis occurred in the first week of life (Bang et al., 2005).

<sup>7</sup>More recently, research has focused on environmental enteropathy (Korpe and Petri, 2012; Humphrey, 2009; Spears, 2013), which although not a main cause of mortality, may be another important cause of stunting.

1970s and 80s, fatality from neonatal tetanus would have been extremely high. UNICEF (2011) reports that fatality rates from untreated neonatal tetanus can be as high as 100%, and Sokal et al. (1988) report on a study from Côte d’Ivoire estimates a fatality rate from neonatal tetanus of 90%. If nearly all children who suffered from tetanus died of it, it is unlikely to have been a major cause of stunting.

The 1978 ORG study suggests that low birth weight/prematurity was the second most important cause of neonatal death during the period in which the adults studied in this paper were infants (Government of India, 1979).<sup>8</sup> This cause of death likely reflects conditions of poor pre-natal nutrition that would have also played an important role in determining heights. The correlation between adult height and neonatal mortality that will be discussed in section 4.4 may reflect statewise variation in pre-natal nutrition when these adults were *in utero*, a hypothesis which will be discussed further in section 4.5.

## 4.3 Data & modeling approach

### 4.3.1 Data sources & descriptive statistics

#### Adult analyses

**Mortality indicators.** Mortality indicators from the 1970s and 1980s used for the analysis of adult heights come from the Sample Registration System (SRS), a vital statistics system run by the Office of the Registrar General at the Indian Ministry of Home Affairs. Trained SRS enumerators register vital events in sample localities in order to estimate demographic rates at the state and national levels. The SRS data cover the 17 major Indian states;<sup>9</sup> data

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<sup>8</sup>Only “prematurity” was listed as a cause of death in this study, birth weight was not specifically mentioned. However, it is unlikely that this survey accounted for pregnancy length, or that women even knew the precise length of their pregnancies. Thus, any low birth weight infant who did not die from another obvious cause might have been said to have died from “prematurity.”

<sup>9</sup>The SRS reports NNM, PNM, IMR, and other mortality rates for each year between 1970 and 1983 in the following states: Andhra Pradesh, Assam, Gujarat, Harayana, Himachal Pradesh, Karnataka, Kerala, Madhya Pradesh, Maharastra, Orissa, Rajasthan, Tamil Nadu and Uttar Pradesh. There is no data for union territories in the SRS.

are available beginning in 1970. Data from the small northeastern states are missing from the SRS.<sup>10</sup> Bihar, Jammu & Kashmir and Punjab also have missing data: data from Bihar are missing from 1970-1980; data from Jammu & Kashmir are missing from 1970-1971; and data from Punjab are missing from 1970. Government of India (1968) and Bhat et al. (1984) provide detailed information about the SRS.

**Height.** Data on adult height are taken from two nationally representative surveys, the National Family Health Survey 2 (NFHS 2), which collected data on the heights of adult women in 1998-1999, and the National Family Health Survey 3 (NFHS 3), which collected data on the heights of adult men and women in 2004-2005. The analysis of adult heights includes only individuals aged 22 and older at the time that their heights were measured. The age of 22 was chosen because Deaton (2008) finds that men and women in India do not reach their adult heights until their early 20s. Since the SRS began measuring mortality in 1970, I have included in the sample women measured in the NFHS 2 and born between 1970 and 1977, as well as men and women measured in NFHS 3 and born between 1970 and 1983.

When matching the heights of individuals measured in NFHS 3 to the SRS data, it is necessary to account for the fact that some state boundaries changed between 1970 and 2005. NFHS 3 height from both Uttarkhand and Uttar Pradesh are matched with SRS data for Uttar Pradesh. Similarly, Bihar and Jharkhand, and for Chattisgarh and Madhya Pradesh.

**Control variables.** For the analysis using adult heights, controls for state level income from the 1970s and 1980s come from the Economic & Political Weekly (EPW) Research Foundation, which produced an annual series of net domestic product by state, at 1970

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<sup>10</sup>These states are: Sikkim, which had previously been a British protectorate and was founded in 1975; Tripura, Meghalaya and Manipur, all of which were founded in 1972; Arunchal Pradesh, which was formed as a union territory out of Assam in 1972, and became state in 1987. An early report on the SRS from the Office of the Registrar General suggests that data from the region that became the union territory of Arunchal Pradesh were not included in the Assam estimates from 1970-1972. Together, these omitted states made up less than 1% of the population of India in the 2011 census.

prices, for the period from 1970-1983. The EPW Research Foundation (2009) explains that net state domestic product is “the totality of commodities and services produced during a given period of time within the geographical boundary of the state in monetary terms counted without duplication” (175). State net domestic product per capita does not include, for example, remittance income from migrants working outside the state. The EPW series has complete information on Indian states during the time period of interest.

The analyses using data on the heights of adults additionally control for whether the state is in the north, as well as for survey round and year of birth.

**Summary statistics.** Figure 4.1 presents summary statistics that reveal large variation at the state level in early life mortality rates during the period from 1970 to 1983, but figure 4.2 shows that there was much less variation in mortality rates over time during this period. Figure 4.1 shows the average neonatal and postneonatal mortality rates in each state from the period of 1970-1983, and figure 4.2 shows the national time trends for neonatal and postneonatal mortality. National neonatal mortality changed very little, from 75 deaths per thousand to about 70. Decline in postneonatal mortality, from about 65 to about 38, was responsible for much of the national decline in infant mortality.<sup>11</sup>

## Child analyses

**Mortality indicators.** In the child level analysis, I compute neonatal and postneonatal mortality from the National Family Health Surveys (NFHS) at the state-survey round levels. I use children born in the three years before the survey in order to compute these measures. A child, whether dead or alive at the time of the survey, is included in the computation of neonatal mortality only if at least a month has passed since his birth. Likewise, a child is used in the computation of postneonatal mortality only if at least a

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<sup>11</sup>Deaton (2006) points out that child mortality (a large fraction of which is infant mortality) was much higher in India than in neighboring China during this period, and that the decline in child mortality was much slower.



year has passed since his birth. I scale the fraction of children who died by 1000 to allow for a simple interpretation of the coefficients.

**Height.** Height-for-age  $z$ -scores of children under three years of age are the dependent variable.  $z$ -scores provide a measure of the heights of children relative to a healthy population. These scores were computed based on the WHO 2006 growth reference standards (De Onis, 2006).<sup>12</sup> For children in NFHS 3, scores are provided in the published data; for children in the NFHS 2, scores were computed based on child sex, age and height in centimeters using WHO Anthro software (WHO, 2011).

**Control variables.** All specifications include a vector of controls for the age-in-months of the child. Controls for economic well-being include a vector of dummy variables for ownership of household assets, including whether or not the child's household owns a radio, a TV, a fridge, a bicycle, a motorcycle, a car, whether the household has electricity, and the household's drinking water source. A control for mother's height, a measure of the child's genetic potential, is also included.

**Summary statistics.** Figure 4.3 presents summary statistics of the state level neonatal and postneonatal mortality rates used in the child level analysis. The Indian states shown in figure 4.3 are ordered by their neonatal mortality rates from the NFHS 2, which range from approximately 13 in Kerala, to over 50 in Madhya Pradesh. When using the NFHS 3 data for this figure, and for all the following analyses, I merge data for the states that split between 1998 and 2005 into the state that existed in 1998. Therefore, Jharkhand and Bihar are represented by Bihar, Chattisgarh and Madhya Pradesh are represented by Madhya Pradesh, and Uttarkhand and Uttar Pradesh are represented by Uttar Pradesh.

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<sup>12</sup>For more on using the WHO 2006 charts in the Indian context, see Tarozzi (2008).

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