Early-Life Health and Adult Circumstance in Developing Countries

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Abstract

A growing literature documents the links between long-term outcomes and health in the fetal period, infancy, and early childhood. Much of this literature focuses on rich countries, but researchers are increasingly taking advantage of new sources of data and identification to study the long reach of childhood health in developing countries. Health in early life may be a more significant determinant of adult outcomes in these countries because health insults are more frequent, the capacity to remediate is more limited, and multiple shocks may interact. However, the underlying relationships may also be more difficult to measure, given significant mortality selection. We survey recent evidence on the adult correlates of early-life health and the long-term effects of shocks resulting from disease, famine, malnutrition, pollution, and war.
1. INTRODUCTION

A growing body of evidence documents that healthy children tend to become healthy and wealthy adults (Almond & Currie 2010, 2011; Currie 2011). For example, anthropometric markers such as birth weight and child height are related to future schooling, employment, earnings, family formation, and health. Findings from natural experiments suggest that these relationships in large part reflect a pathway running from childhood health to adult outcomes. To the extent that inequalities and shortfalls in adult outcomes are predicted by early-life health, these results may offer additional justification for policies aimed at improving child health.

Much of the existing literature focuses on rich countries, but researchers are increasingly taking advantage of new sources of data and identification to study the long reach of childhood health in developing countries. Far beyond simply extending the work on rich countries, this research on poorer countries has much policy relevance; health shocks in early life are widespread in the developing world. Strauss & Thomas (2008) and Bleakley (2010) review some of this work but do not focus on it. In this article, we review recent research on the long-term effects of health shocks before age 10 in developing countries. To distinguish ourselves from previous authors, we limit our attention to findings since 2004, many of which are not discussed in previous reviews.

We argue that the long-term effects of such shocks are likely to be larger in developing countries for several reasons. First, such shocks are simply more frequent in many developing countries than in the industrialized world, suggesting that the lingering effects of early-life health problems may well be more important in developing countries. Second, child health shocks are likely to interact. For example, a child who is malnourished may be less able to ward off, or to recover from, disease. Similarly, a mother who was malnourished may bear a child who is compromised in his or her ability to cope with health insults. Moreover, if there are nonlinearities in the production function for child health, then the same shock may have quite different effects depending on starting levels of health.

Third, the long-run consequences of early-life health shocks depend on the availability and effectiveness of mitigation strategies. To the extent that parents in rich countries are better able to compensate for shocks, the long-term effects of poor health in childhood may be greater in developing countries. For example, a relatively common congenital anomaly such as cleft palate is easily corrected in rich countries but may lead to a great deal of suffering in poorer settings. Conversely, if mitigation were universally ineffective across countries, then outcomes might be similar across countries, reflecting a purely biological response.

THE LASTING EFFECTS OF CHILDHOOD HEALTH IN THE CONTEXT OF LONG-RUN ECONOMIC DEVELOPMENT

Long-standing arguments that ill health impedes economic development hit a snag when evidence emerged that the global decline of infectious disease in the mid-twentieth century did not bring prosperity to the world’s unhealthiest countries (Acemoglu & Johnson 2007). Morbidity rates fell, but so too did mortality rates—especially among children—which increased population growth and thus undercut per-capita economic progress. This historical episode, commonly called the global epidemiologic transition, highlights the delicate interplay of health, population, and the macroeconomy. This issue is sure to become more important as countries tackle child mortality but fail to eliminate sickness and hunger among the legions of survivors. If positive effects occur at a lag, then existing research may underestimate the macroeconomic benefits of improving population health.
Not only may early-life health be a more important determinant of adult outcomes in developing countries, but its importance may also have increased as childhood mortality rates have declined. Here we confront the paradox of mortality selection: When children die, their long-term outcomes are not measured. Consequently, as infant and child mortality decrease, population average outcomes such as employment and earnings could actually fall, or at least rise more slowly than they would have otherwise, as more unhealthy children live to adulthood (see the sidebar, The Lasting Effects of Childhood Health in the Context of Long-Run Economic Development). The selection biases introduced by changes in the distribution of survivors cause concern in nearly all the papers we review, and we revisit them often in our discussion. In general, one cannot quantify the bias stemming from selective mortality; its size depends on a health condition’s (unmeasurable) relative morbidity and mortality burdens in the population of interest. However, under reasonable distributional assumptions, Bozzoli et al. (2009) show that the bias tends to be largest for health insults with large mortality burdens and in populations with high baseline mortality rates. As a result, we might expect to find selective mortality bias in studies of widespread mortality crises, as well as in studies of Africa.¹ This bias would tend to make the long-term effects of early-life health insults appear less pronounced.

Our review is divided in two broad parts. The first deals with associations between measures of early-life health and adult outcomes, whereas the second describes attempts to disentangle the causal path running from childhood to adulthood. The literature on associations of child health measures with adult outcomes goes back several decades, and in our discussion, we concentrate on the retrospective measure that the development literature commonly uses: height. We argue that height captures aspects of the early-life experience similar to those captured by measures more common in the literature on wealthy countries, such as birth weight. Yet height, unlike birth weight, does not require longitudinal data for linkage with other adult outcomes. Nonetheless, despite its broad informational content and ease of data collection, height does not allow researchers to identify which specific aspects of the childhood environment have long-lasting effects.

As a result, we devote the second section of our review to research that traces out the effects of specific changes in the child health environment. To structure the discussion, we separate this work into five thematic categories, each for a separate source of ill health: (chronic) malnutrition, famine, disease, pollution, and war. These health insults affect their childhood victims in diverse ways, and they cause differing degrees of mortality selection. Additionally, as we show below, the long-term effects of the same health condition often vary considerably by setting, gender, or even socioeconomic status.

Given that different afflictions may have different effects in the same population, and a single affliction may have varied effects across populations, much remains to be learned about the long-term effects of child health in poorer countries. Similarly, although the concepts of critical periods and catch-up growth have been in the literature for some time, we still know relatively little about which periods of childhood are critical for particular types of development or about how much recovery from health insults is possible. We conclude with some thoughts on fruitful directions for future research. Richer data are becoming available, and the subjects of a recent spate of randomized trials related to child health in developing countries

¹One could argue that selective mortality offers too many degrees of freedom to a researcher seeking an ex post explanation of a weak estimated effect. Although this point of view is correct for any single estimate, the statistical insights discussed by Bozzoli et al. (2009) provide structure for comparisons of multiple estimates across settings and health conditions.
(e.g., Baird et al. 2011) are starting to come of age. These developments are sure to give rise to many useful new insights.

2. HEIGHT AS A PROXY FOR THE EARLY-LIFE ENVIRONMENT

The size and shape of the human body provide a window into the cumulative shocks it has experienced over its lifetime. Researchers have thus used various measures of the body and its growth rate to gauge health. Of the various anthropometric measures studied, the most common are weight, typically a proxy for short-term nutritional status, and height, a marker of health and nutrition during the critical periods of growth in early life (especially from conception to age 3). Absent more direct measures of early-life health, investigations into the effects of early-life conditions have often relied instead on height and birth weight. A large literature explores the later-life correlates of birth weight in industrialized countries, but the literature on poorer countries has focused on height, given the paucity of good longitudinal data following individuals in those settings from birth into adulthood.

In the cases of both height and birth weight, bigger is better. On average, adults who stand taller or were born heavier fare better on a range of social, economic, and cognitive outcomes (see, e.g., Steckel 2008, Currie 2009). Interpretations of these patterns are fairly uniform for birth weight but more variable for height. Perhaps because of the proximity of birth weight to the intrauterine environment, few have questioned that its relationship with adult outcomes reflects the effects of early-life health, at least after appropriately controlling for family background characteristics.

In contrast, a surfeit of theories aims to explain the link between height and various measures of well-being and success. One class of theories, especially popular in the development economics literature, attributes the height premium to the greater strength and health of taller individuals, which increase their productivity (Haddad & Bouis 1991, Thomas & Strauss 1997). A closely related hypothesis stems from the observation that physical growth and cognitive development share inputs in early life. These common inputs induce a correlation between height and cognitive ability, so in addition to being stronger and healthier, taller individuals are smarter (Case & Paxson 2008). But several competing psychosocial theories offer explanations for the height premium that do not rely on the biology of skill formation. Some researchers posit that height affects individual achievement through self-esteem (Wilson 1968, Young & French 1996), social dominance (Klein et al. 1972, Hensley 1993), or discrimination (Loh 1993, Magnusson et al. 2006). These theories have attracted far more attention in rich countries than in poor ones, but even in rich countries, nationally representative data strongly support explanations rooted in the biology of skill formation (Case & Paxson 2008, Lundborg et al. 2009). Conversely, biological mechanisms and psychosocial mechanisms are not necessarily mutually exclusive.

2.1. A Statistical Framework

A simple statistical framework illustrates the theories that propose child development as the link between body size and achievement. Let $y$ be an adult outcome (e.g., the log wage), let $x$ be

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2The study of the economic, social, and psychological correlates of body size dates back at least to the work of eugenicists and biological anthropologists in the nineteenth and early twentieth centuries (Porter 1892, MacDonell 1902, Pearson 1906, Gowin 1915).
an anthropometric measure (e.g., birth weight or adult height), and let \( z \) be an (unobserved) index of the joint determinants \( y \) and \( x \). We think of \( z \) primarily as an index of the childhood environment. In reality, \( z \) may partly reflect genetics, although twin studies suggest that a substantial share of the relationship between anthropometric measures and adult achievement is attributable to nongenetic factors.\(^3\) Moreover, many adult outcomes are likely the product of epigenetic interactions between genes and the environment, suggesting that the quest to separate nature and nurture is misguided (see Jablonka & Lamb 2005 for an accessible discussion of genetics and epigenetics).

Suppose that \( y \) and \( x \) have the following reduced-form production functions:

\[
y = \alpha_y + \beta_y z + \epsilon_y, \tag{1}
\]

\[
x = \alpha_x + \beta_x z + \epsilon_x. \tag{2}
\]

Both adult outcomes and body size increase linearly in \( z \). In the absence of a good measure of \( z \), however, one might run a regression of \( y \) on \( x \) to learn (imperfectly) about the parameters in Equations 1 and 2:

\[
y = a_{yx} + b_{yx} x + u. \tag{3}
\]

The slope coefficient in Equation 3 has the following probability limit:

\[
\text{plim } b_{yx} = \frac{\beta_x}{\beta_z} \times \frac{1}{1 + \frac{\sigma_x^2}{\beta_x^2 \sigma_z^2}}, \tag{4}
\]

where \( \sigma_x^2 = V[\epsilon_x] \) and \( \sigma_z^2 = V[z] \).

The expression in Equation 4 has several intuitive implications. The slope coefficient increases in the effect of the early-life environment on \( y \) and decreases in the effect of the early-life environment on \( x \). If the residual in Equation 2 has zero variance (as assumed, e.g., in Weil 2007), then the slope coefficient is equivalent to an instrumental variables estimator in which \( z \) is used as an instrument for \( x \) to estimate Equation 3. But as \( \sigma_x^2 \) increases relative to \( \beta_x^2 \sigma_z^2 \) (the component of the variance of \( x \) explained by \( z \)), \( x \) becomes a noisier signal of \( z \), which attenuates the slope coefficient. This result provides two reasons to expect \( b_{yx} \) to be higher in poor countries.\(^4\) First, early-life conditions are more variable in poor countries so that \( \sigma_x^2/(\beta_x^2 \sigma_z^2) \) is smaller holding \( \beta_x \) constant. Second, early-life conditions are worse on average in poor countries, which may increase \( \beta_x \) owing to decreasing marginal returns. If a decrease in \( \overline{x} \) raises \( \beta_y \) and \( \beta_x \) by the same proportion, the ratio \( \beta_y/\beta_x \) remains unchanged while \( \sigma_x^2/(\beta_x^2 \sigma_z^2) \) falls, leading to an increase in \( b_{yx} \). These two reasons for a higher \( b_{yx} \) in poor countries rely on cross-national differences in the ratio \( \sigma_x^2/(\beta_x^2 \sigma_z^2) \). But poor countries may also have higher \( b_{yx} \) because they may have higher \( \beta_y \). For instance, if poor countries had a higher return to skill, then taller workers would receive a higher wage premium, holding other parameters constant.

\(^3\)Siilentoine et al. (2000) and Sundet et al. (2005), both studying thousands of twin pairs, find that over half the relationship between height and cognition can be attributed to nongenetic rather than genetic factors. Notably, the studies use data from wealthy, egalitarian, Scandinavian countries, where the variability of childhood conditions is likely much smaller than in developing countries, making genes relatively more important.

2.2. The Association Between Height and Birth Weight

The framework posits that adult height and birth weight reflect similar underlying variables. Indeed, height and birth weight are highly correlated in settings rich and poor, both across and within families (for evidence on developing countries, see Haefliger et al. 2002 and Adair 2007; for evidence on industrialized countries, see Kuh & Wadsworth 1989, Sørensen et al. 1999, Pietiläinen et al. 2001, Behrman & Rosenzweig 2005, and Black et al. 2007). To give a sense of this empirical regularity, Table 1 examines the association between birth weight and young adult height in two cohorts, one born in 1958 in Great Britain and the other born in 1983–1984 in the Philippine metro area of Cebu (which encompasses both urban and rural communities). Toward the bottom of the table, we present the means and standard deviations of birth weight and height in the two samples. As might be expected given the nutritional disparities between the two settings, both measures of body size have significantly lower means in the Philippines than in Great Britain. Surprisingly, the variances of both variables are higher in Great Britain than in the Philippines. Based on this result alone, however, one cannot quantify differences in the variances of early-life conditions ($\sigma_z^2$) and the variances of the residuals ($\sigma_x^2$).

The forward and reverse regressions of the two variables shed some light on this issue. In the first two rows of Table 1, we present the slope coefficients from the regression of height on birth weight and the reverse, in both cases controlling for gender. All the slope coefficients are highly statistically significant, indicating a strong relationship between height and birth weight. A 1-kg increase in birth weight is associated with a 3–4-cm increase in height.5 Consistent with the prediction that $\sigma_x^2/(\beta_x^2 \sigma_z^2)$ is smaller in developing countries, both slope coefficients are higher in the Philippines than in Great Britain. However, the difference in coefficients approaches statistical significance only in the regressions of birth weight on height. For a summary statistic that more clearly describes the combined signal that height and birth weight have for the early-life environment, consider the product of the slope coefficients:

\[
\text{plim } b_{hw} b_{wh} = R_{bz}^2 R_{wz}^2,
\]

where $h$ refers to height and $w$ refers to birth weight. $R_{bz}^2$ is the fraction of the variance in height that is explained by early-life conditions $z$, and $R_{wz}^2$ is the same fraction for birth weight. In other words, these two quantities are the $R^2$’s from Equations 1 and 2. This product of these $R^2$’s, which varies between 0 and 1, summarizes the importance of joint input variation relative to residual variation in producing height and birth weight. A value of 1 would imply that $z$ explains all the variation in height and birth weight, whereas a value of 0 would imply that $z$ explains none of that variation. Unfortunately, the statistic does not have an intuitive interpretation for values between 0 and 1. However, if we take the square root of the product,

\[
\text{plim } \sqrt{b_{hw} b_{wh}} = \sqrt{R_{bz}^2 R_{wz}^2},
\]

then we obtain a statistic with the same scale as the $R^2$ from an ordinary least squares regression. In the third row of Table 1, we report the square root of the product of the slope coefficients for each of our samples. As expected, the relative importance of joint input variation is (marginally) significantly higher in the Philippines than in Great Britain. This result implies that the higher overall variances of height and birth weight in Great Britain derive from the residual variance,

5 After one controls for gender, the conditional correlation between the two variables is 0.26 in Great Britain and 0.30 in the Philippines.
rather than the variance of joint inputs. Perhaps contemporary Great Britain has greater genetic diversity than the Philippines does. Whatever the case, its greater variance in body size does not reflect a greater variance in childhood conditions.

Apart from the Cebu Longitudinal Study, precisely measured birth weight data are rare in large sample surveys from developing countries, as are longitudinal data following babies into adulthood. Consequently, the development literature—as well as the economic history literature, for similar reasons—has largely relied on height. An important exception to this generalization is a recent paper by Bharadwaj et al. (2010) that links Chilean birth data for all children born between 1992 and 2000 to data on educational attainment up to the eighth grade and to data on parental time use. Twin fixed effects models show a persistent positive impact of birth weight on educational attainment. Aside from this recent paper, however, most work on developing countries focuses on height.

### 2.3. Changes in Average Height

The literature on height is in part motivated by the remarkable increasing secular trend in heights over the past two centuries. The trend began in Europe, which economic historians attribute to rising living standards (Floud et al. 1990; Steckel 1995, 2008; Fogel 2004). Japan followed...
somewhat later (Shay 1994), as the Japanese economy entered a period of rapid growth in the late nineteenth century. Many countries still classified today as developing also saw rising average heights across cohorts born in the twentieth century (Strauss & Thomas 1998). These transformations in the average size of the human body occurred too quickly to be driven by evolution.

Indeed, researchers have now amassed considerable evidence that these secular increases in height have antecedents in early-life health. Many studies show that child and adult height are associated with parental socioeconomic status, nutrition, and access to clean water and sanitation during childhood (see, e.g., Thomas 1994, Adair & Guilkey 1997, Checkley et al. 2004). Additionally, a number of the quasi-experimental studies reviewed below suggest that these associations represent causal effects of childhood conditions.

Selective mortality presents an obstacle to detecting a relationship between early-childhood conditions and future outcomes. This issue arises in the quasi-experimental studies reviewed below, but it is also apparent in comparisons of average heights across countries and birth cohorts. Studying a country-birth cohort panel from the Demographic and Health Surveys, Deaton (2007) demonstrates that higher national income and lower child mortality in the year of birth are associated with increased height outside Africa. But within Africa, where mortality rates are high, declines in child mortality are associated with decreased height, and national income bears no relation to height after controlling for mortality. A likely explanation for these findings is that selective mortality shapes height distribution in Africa, where child mortality rates are high. In this sense, the findings offer one account of why Africans are much taller than one would expect in light of their incomes.

2.4. Height and Other Adult Outcomes

In settings with limited selective mortality, height correlates with skill; the inputs that promote healthy growth in childhood also promote the development of both physical and cognitive skills. Studies in a variety of countries, rich and poor, confirm this correlation. Table 2 provides an overview of four recent studies on developing countries. It shows that height is associated with adult cognitive test scores in China (Huang et al. 2012); in Mexico (Vogl 2012); and in urban areas in Barbados, Mexico, Cuba, Uruguay, Chile, and Brazil (Maurer 2010). For similar results in industrialized countries, readers are referred to Richards et al. (2002) (Great Britain), Tuvevo et al. (1999) and Magnusson et al. (2006) (Sweden), and Case & Paxson (2008) (United States) (see also Tanner 1979). Research assessing the relationship between adult height and physical skill is rarer, but Swedish data suggest a robust relationship between height and grip strength, muscular strength, maximum working capacity, and maximum power expended on a stationary bicycle (Lundborg et al. 2009). Height is also associated with noncognitive skills (Schick & Steckel 2010, Lundborg et al. 2009), but the importance of this correlation in developing countries remains unstudied.

Possibly as a by-product of the correlation between height and various skills, height is associated with wage gains in many developing economies. In the rural Philippines, an extra centimeter of height is associated with 2% higher wages (Haddad & Bouis 1991); in Mexico, this semi-elasticity is 2.5 (Vogl 2012). Height and wages are also strongly correlated in Ghana and Brazil (Strauss & Thomas 1998, Schultz 2002), although the estimation strategies used for these settings

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7Abbott et al. (1998) find a similar result in a sample of Japanese-Americans in Hawaii, many of whom were born in Japan in the early twentieth century.
Table 2  Associations of height and other adult outcomes

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Shock</th>
<th>Methods</th>
<th>Data/size</th>
<th>Outcomes</th>
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<tr>
<td>Behrman et al.</td>
<td>Guatemala</td>
<td>Randomly assigned high-protein energy drink (<em>atole</em>) and placebo (<em>fresco</em>), two small and two large villages, 1969–1977; earthquake, 1976</td>
<td>OLS and IV with instrument of adult human capital and variables reflecting prices and policies, and individual and family endowments, to look at relation between cognitive and health human capital and labor market outcomes</td>
<td>INCAP longitudinal data on children born 1962–1977 from a nutritional supplementation trial, follow-up in 2002–2004 (962 obs.)</td>
<td>In OLS, an SS effect of adult height and fat-free body mass on wages (this disappears in the IV specifications), with similar results for annual hours and total income; only adult cognitive skills SS when both health and cognitive human capital measures are treated as endogenous; years of schooling and adult reading comprehension z scores SS in both OLS and IV.</td>
</tr>
<tr>
<td>Huang et al.</td>
<td>China</td>
<td>No shock; explores associations between height shrinkage and SES variables</td>
<td>Estimated preshrinkage height and shrinkage used as covariates in OLS regressions, using upper arm and lower leg length measurements, to estimate a preshrinkage height function and predict preshrinkage heights for an older population</td>
<td>China Health and Retirement Longitudinal Study; sample of 2,609 people ages 45–49 (“young” sample) and 5,868 people ages &gt;60 (“old” sample) in 2011</td>
<td>Negative correlation between shrinkage and SES variables such as schooling and household per-capita expenditure; measured height positively and significantly associated with all the cognition measures; height shrinkage strongly negatively correlated with these measures, suggesting the association between measured height and cognition partly occurs through height shrinkage.</td>
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<tr>
<td>Maurer (2010)</td>
<td>Barbados, Mexico, Cuba, Uruguay, Chile, and Brazil</td>
<td>No shock; examines relationship between cognitive function in later life and height, early-life conditions, and education</td>
<td>Sex-specific OLS/Tobit or 2SLS/IV-Tobit, depending on whether height is instrumented with knee height, and Tobit for potential ceiling effects (scores measure only cognitive function if below the maximum, otherwise are a lower bound)</td>
<td>Survey on Health, Wellbeing and Aging in Latin America and the Caribbean in 2000, representative urban samples of persons ages ≥60 (7,894 obs.)</td>
<td>SS positive association between height and later-life cognitive function, larger for women than men; significance lost for men when height is instrumented, suggesting that for men this association partly results from mid- and later-life circumstances; additional controls for childhood conditions do not affect the height coefficients, but including education does weaken the association</td>
</tr>
<tr>
<td>Vogl (2012)</td>
<td>Mexico</td>
<td>No shock; looks at the skill returns underlying the labor market height premium</td>
<td>OLS/logit of hourly earnings and occupation on height; addition of health and childhood covariates, education and cognitive test scores, to look at mechanisms</td>
<td>Mexican Family Life Survey, a nationally representative household survey with waves in 2002 and 2005, individuals ages 25–65</td>
<td>Wage gains of 2.1% (2.9%) for men (women) with a 1-cm increase in height; adding cognitive test scores leads to a 17% decrease in the earnings premium to taller workers, but about half the premium can be attributed to these workers being more educated or having higher-paying jobs (with greater intelligence and lower strength requirements)</td>
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</tbody>
</table>

Abbreviations: 2SLS, two-stage least squares; INCAP, Institute of Nutrition of Central America and Panama; IV, instrumental variables; OLS, ordinary least squares; SES, socioeconomic status; SS, statistically significant.
are not directly comparable with other studies. Consistent with the implications of the statistical framework earlier in this section, the relationship between height and earnings is positive but smaller in wealthy countries (Persico et al. 2004, Case & Paxson 2008, Lundborg et al. 2009). Cognitive and physical skills explain a large share of the earnings premium paid to taller workers in these richer settings.

Evidence on the role of skills in explaining the height premium in developing countries is scarcer. Many authors (e.g., Thomas & Strauss 1997, Schultz 2002) note that height is associated with educational attainment, although they generally reason that this result reflects correlated parental investments in health and education, rather than shared inputs in the production functions for height and skill. In more recent data from Mexico, Vogl (2012) finds mixed evidence on the role of cognitive skill in explaining the relationship between height and earnings. On the one hand, an adult cognitive test score explains only a small share of the height premium. On the other hand, taller workers sort into occupations with higher–cognitive skill requirements, consistent with a Roy model in which the return to cognitive (rather than physical) skill drives the height premium, and similar to the occupational sorting observed in industrialized countries (Case & Paxson 2008). In Mexico, the sorting of taller workers into higher-skill occupations almost entirely reflects their greater educational attainment. Collectively, these results suggest either that cognitive ability (and therefore height) increases the productivity of schooling or that parents who invest more in health and nutrition also tend to invest more in other forms of human capital.

Additional evidence on the relative importance of brains and brawn can be found in the literature relating short-term nutritional status to wages. One body of work uses local food prices as instruments for the body mass index and nutrient consumption, finding effects of these variables on wages for men but not women (Thomas & Strauss 1997). These estimated effects suggest that physical strength and robustness remain important in developing economy labor markets, especially for men. Note that instrumental variables methods are more appropriate for estimating the effect of body weight than for estimating the effect of height. Several studies, including Schultz (2002) and Behrman et al. (2009a), use early-life conditions as instruments in estimating the effect of height on wages. However, if height is primarily a proxy for early-life conditions, then we see more justification for studying the reduced-form effects of the instruments directly. Height becomes useful as an independent variable precisely when we cannot observe all relevant determinants of childhood development. For much the same reason, we cannot use adult height to distinguish the effects of in utero shocks from those of postnatal shocks. Nonetheless, given the ease of measurement, height provides a useful retrospective proxy for health and nutrition in the early years of life.

3. EVIDENCE REGARDING THE LONGER-TERM EFFECTS OF EARLY-LIFE SHOCKS

In the preceding section, we elaborate on the argument that early-life shocks might have larger effects in developing countries, provide a justification for viewing adult height as an outcome, and emphasize the potential importance of mortality selection. In this section, we review the recent literature regarding specific types of early-life shocks: malnutrition, famine, disease, pollution, and war.

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8The cognitive test in Vogl’s (2012) data has only 12 questions, making it a noisy measure of intelligence.
3.1. Nutrition

Table 3 provides an overview of some recent studies examining the longer-term impacts of poor nutrition in childhood. Here we focus on moderate and chronic malnutrition; we discuss famine in Section 3.2. Recent studies have focused on the roles of specific nutrients, and on the question of whether children recover from nutritional shocks during critical periods, such as while in utero or in the first year of life.

In one recent study, Maluccio et al. (2009) report on a long-running randomized INCAP (Institute of Nutrition of Central America and Panama) experiment that involved giving Guatemalan children ages 0–7 a high-protein energy drink (the treatment) or a placebo with a similar number of calories. The findings suggest that the treatment increased schooling attainment by 1.2 grades among females and cognitive ability by 0.24 standard deviations for both males and females. Hoddinott et al. (2008) study the same intervention and find that nutritional supplements in the first two years of life increased male wages by 46%. These large effects suggest that an early diet sufficient in calories but not in protein can have severe long-term consequences.

Quasi-experimental studies also provide evidence on the importance of specific nutrients. In one prominent example, Field et al. (2009) examine the effect of fetal exposure to iodine supplementation as part of a large but episodically implemented government program in Tanzania. Iodine deficiency in pregnant women has been linked to mental retardation in affected fetuses but has been virtually eliminated in developed countries. The authors find that children who benefited from the supplements in utero experienced large gains in educational attainment. The effects were larger for girls than for boys, which is consistent with scientific evidence that female fetuses are more vulnerable to damage from iodine deficiency owing to their greater cognitive sensitivity to maternal thyroid deprivation.

A number of recent studies examine the long-term effects of general nutritional deprivation at particular critical periods. Almond & Mazumder (2011) provide evidence on this front in their analysis of the effect of maternal fasting during Ramadan on children in utero. Their study relies on the variable timing of Ramadan so that the effects can be distinguished from seasonal variation. Using census data from Uganda and Iraq, they show that cohorts affected by maternal fasting are 22–23% more likely to be disabled in adulthood. In comparison, the estimated effect on disability rates among Arab-named adults in the United States is only slightly lower, at 19%.

Variation in adult outcomes by season of birth may also shed light on the effects of early-childhood health. In their examination of the age of heart disease onset in Puerto Rico, McEniry & Palloni (2010) focus on whether subjects were in utero during the “hungry season.” They find a monotonic relationship between length of exposure and the probability of heart disease. Among those exposed to poor conditions throughout pregnancy, 23% had heart disease, compared with 15–18% among individuals who were partially exposed and 11% among those who were not exposed. The authors note, however, that it is difficult to separate the effects of poor nutrition from the effects of exposure to disease or other seasonal factors in this type of research design. Additionally, the socioeconomic and demographic composition of mothers varies over the

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9In absolute terms, Hoddinott et al. (2008) find an effect of USD 0.67 per hour, with a 95% confidence interval of 0.16–1.17. The point estimate is 46% of the average wage, and the confidence interval runs from 11% to 80%.

10Another recent analysis of a natural experiment involving nutrient supplementation can be found in the work of Linnemayr & Alderman (2011), who evaluate a nutrition enhancement program in Senegal that was phased in at the village level. The program had multiple components, including vitamin A and iron supplements for pregnant women. Although the results vary somewhat with the estimation method, they find consistent evidence that maternal participation during pregnancy was associated with improvements in child weight for age. We do not report this paper in Table 3 because the weight measurements are from childhood as well.
### Table 3  Effects of childhood nutrition on adult outcomes

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<tr>
<td>Almond &amp; Mazumder (2011)</td>
<td>United States, Iraq, Uganda</td>
<td>Days of Ramadan exposure in utero</td>
<td>OLS with percent of pregnancy overlapping with Ramadan on future disability status, home ownership, years of schooling, employment, and earnings</td>
<td>Michigan natality files 1989–2006, Uganda 2002 census (80,000 obs., ages 20–80), Iraq 1997 census (250,000 people born 1958–1977 who were ages 20–39 in 1997)</td>
<td>In Uganda (Iraq), Muslims in utero during Ramadan 22% (23%) more likely to be disabled and 2.6% (1.4%) less likely to own a home (and, in Iraq, less likely to be employed); no effect on schooling</td>
</tr>
<tr>
<td>Field et al. (2009)</td>
<td>Tanzania</td>
<td>Iodized oil capsule distribution program, 1986–1995</td>
<td>District-level FE regression to examine impact of prenatal iodine supplementation on schooling, exploiting variation in program activity (gaps and delays)</td>
<td>2000 Tanzanian Household Budget Survey, children ages 10–13 (1,395 obs.)</td>
<td>Large and SS increase in progression through school with supplement distribution; children treated in utero attain 0.35–0.56 years of additional schooling relative to siblings and older and younger peers; girls consistently benefit more than boys</td>
</tr>
<tr>
<td>Hoddinott et al. (2008)</td>
<td>Guatemala</td>
<td>Randomly assigned high-protein energy drink (<em>atole</em>) and placebo (<em>fresco</em>), two small villages and two large villages, 1969–1977; earthquake, 1976</td>
<td>OLS for relationship between exposure to <em>atole</em> or <em>fresco</em> at specific ages between 0 and 7 years and economic variables</td>
<td>Data from 60% of the children (ages 0–7) in the original nutrition intervention program, follow-up 2002–2004 (1,424 people, ages 25–42)</td>
<td>Exposure to <em>atole</em> before age 3 associated with higher hourly wages for men; wages rose by USD 0.67 per hour (46% increase) for those exposed to <em>atole</em> from ages 0–2; hours worked were less and annual incomes were greater but not SS</td>
</tr>
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Table 3 (Continued)

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<th>Methods</th>
<th>Data/size</th>
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<tbody>
<tr>
<td>Linnemayr &amp; Alderman (2011)</td>
<td>Senegal</td>
<td>Nutrition enhancement program: vitamin A and deworming for children 6–59 months, iron for pregnant women, bednets, breastfeeding promotion, cooking workshops, 2004–2006</td>
<td>OLS, 2SLS (instrument actual receipt with planned treatment status), and a combination of DD and propensity score matching to estimate effect of package of services on weight-for-age z scores</td>
<td>Data from the program's two survey rounds (2004 and 2006), children under age 5 (10,127 obs.)</td>
<td>Insignificant impact of planned treatment status on z scores and of instrumented intervention; both show positive, SS impact on weight-for-age z scores for children who benefited from the program in utero; TOT estimates show z scores improved by 0.1, with a larger impact of 0.27 for the propensity score matching estimate</td>
</tr>
<tr>
<td>Maccini &amp; Yang (2009)</td>
<td>Indonesia</td>
<td>Rainfall during year of birth, 1953–1974</td>
<td>IV regression with instrument of rainfall measured at the second to fifth closest rainfall stations for impact of early-life rainfall on health, schooling, and SES</td>
<td>Global Historical Climatology Network and Indonesian Family Life Survey 2000 (8,883 obs.)</td>
<td>Positive effect of higher early-life rainfall for girls; for adult women, 20% higher rainfall in birth year leads to 0.57-cm increase in height, 3.8-PP decrease in self-reporting of poor health, 0.22 more years of schooling, and a 0.12-SD increase on an asset index</td>
</tr>
<tr>
<td>Maluccio et al. (2009)</td>
<td>Guatemala</td>
<td>Randomly assigned high-protein energy drink (atole) and placebo (fresco), two small villages and two large villages, 1969–1977; earthquake, 1976</td>
<td>DD to study impact on educational outcomes of nutrition by contrasting individuals exposed at different periods in atole versus fresco villages; 2SLS with instrument of supplement intake with exposure to intervention</td>
<td>INCAP longitudinal data, children ages 0–7 at any point during intervention, follow-up rounds in 1988–1989 and 2002–2004 (1,471 obs.)</td>
<td>Impact of exposure at ages 0–3 to the nutritional intervention 25 years after it ended: increased schooling for women by 1.2 grades (0.33 SD); for men and women increased reading comprehension by 0.28 SD (with stronger effect for women) and cognitive ability by 0.24 SD; 2SLS point estimates approximately twice the OLS</td>
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<tbody>
<tr>
<td>McEniry &amp; Palloni (2010)</td>
<td>Puerto Rico</td>
<td>Rural population’s exposure to poor nutrition/disease during pregnancy from seasonality in sugar cane harvests, late 1920s to early 1940s</td>
<td>Kaplan-Meier hazard estimates, age of heart disease onset by exposure level during late gestation; Cox and log logistic regression models to estimate effect of exposure on heart disease timing</td>
<td>Puerto Rican Elderly: Health Conditions survey of rural population ages ≥60 and their spouses, 2003 and 2006–2007 (1,438 obs.)</td>
<td>Highest prevalence of heart disease (23%) for those fully exposed (born in fourth quarter) to poor nutrition and infectious diseases during late gestation; those with partial exposure had prevalence of 15–18%, and unexposed individuals (second-quarter birth) had 11%; risk of developing the disease if fully exposed was twice as high as for the unexposed at later ages; no impact on timing of onset or for urban population</td>
</tr>
<tr>
<td>Pathania (2009)</td>
<td>India</td>
<td>Effects of drought shocks at birth on height of rural women in India, 1950–1999</td>
<td>OLS of effect of various drought measures on height, with various FE and linear time trends, depending on specification</td>
<td>Historical rainfall data and height of ever-married women ages 15–49 from NFHS-2, 1998–2000 (60,641 obs.)</td>
<td>Drought at birth associated with 0.3-cm drop in height of upper-caste women but 0.4-cm gain in height of the Scheduled Tribes, a low-caste group; effects mostly driven in utero by drought exposure; does not find a caste gradient in height outcomes for more recent birth cohorts</td>
</tr>
</tbody>
</table>

Abbreviations: 2SLS, two-stage least squares; DD, difference-in-differences; FE, fixed effects; INCAP, Institute of Nutrition of Central America and Panama; IV, instrumental variables; NFHS, National Family Health Survey; OLS, ordinary least squares; PP, percentage point; SD, standard deviation; SES, socioeconomic status; SS, statistically significant; TOT, treatment on the treated.
year, which presents additional challenges to interpreting this work (Lam & Miron 1994, Buckles & Hungerman 2013).

Research on rainfall shocks provides more convincing evidence on the effects of experiencing hunger in early life. In one such study, Maccini & Yang (2009) link adults in the Indonesia Family Life Survey to data on rainfall in their birth districts during their birth years. They hypothesize that higher rainfall is linked to better nutrition in the first year through its effects on harvests. Indeed, higher early-life rainfall affects height and a range of other adult outcomes for women but not men. For women, a 10% increase in local rainfall in the year of birth leads to a 0.3-cm increase in height, a 0.1-year increase in education attainment, and a 0.06–standard deviation increase in an index of household durable goods ownership. The results for men are statistically insignificant and less consistent, although the (imprecise) estimates of the effect of rainfall on height remain positive. One could interpret the weaker results for men as reflecting mortality selection, as boys are more vulnerable than girls to dying in childhood.11

Further evidence on the importance of mortality selection can be found in Indian women, among whom early-life rainfall increases average adult height for upper castes but lowers average adult height for lower castes (Pathania 2009). The child mortality rates of the lower castes may be more environmentally sensitive, increasing the likelihood of selective mortality. This reasoning implies that higher early-life rainfall does not diminish the health of children from lower castes but rather keeps unhealthy ones alive. These patterns have diminished in more recent cohorts, which have experienced lower rates of child mortality. According to new research by Shah & Millett-Steinberg (2012), exposure to drought in utero reduces test scores among Indian school-age children regardless of parental socioeconomic status.12 Similar results emerge in northeast Brazil, where Rocha & Soares (2011) find that exposure to drought in utero reduces infant health and subsequent primary school performance.

3.2. Famine

Several attributes distinguish famines from the localized rainfall shocks that Maccini & Yang (2009) and Pathania (2009) study. First, in general equilibrium, famines affect both food prices and farmers’ incomes, whereas localized rainfall shortfalls affect only incomes. Second, perhaps in part because of price adjustments, famines are typically more severe than localized shocks, with massive effects on mortality. As a result, concerns about selective mortality are likely to be more relevant in studies of famine. Third, unlike localized weather shocks, famines arguably have as much to do with politics as with food output (Drèze & Sen 1989). Thus, research strategies designed to measure the effects of localized rainfall shocks may fail to generate plausible estimates of the effects of famine.

Table 4 summarizes a growing literature aimed at detecting the long-term health effects of famine in developing countries. As clearly shown in the table, most of this literature has focused on the effect of exposure in utero, with relatively little information available about the effect of exposure in early childhood. This focus is inspired by the literature on the Barker hypothesis (Barker 2001), which suggests that events during pregnancy can have long-term effects by

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11As a test for mortality selection, Maccini & Yang (2009) estimate the effect of early-life rainfall on cohort size, finding no effect. However, they run this test either for women only or for both men and women. (The paper is unclear on this issue.) Theory might predict an effect on male cohort size only.

12Interestingly, Shah & Millett-Steinberg (2012) also find that current exposure to drought improves test scores in school-aged children, which they interpret as a substitution effect due to drought-related reductions in parents’ and children’s opportunity costs of time.
### Table 4  Effects of childhood famine exposure on adult outcomes

<table>
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<tr>
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<tr>
<td>Almond et al. (2010)</td>
<td>China</td>
<td>1959–1961 famine</td>
<td>Aggregate weighted death rate in year/month proxy for famine intensity for impact in utero on outcomes; mainland-born (exposed to famine) and Hong Kong–born (control) mothers also compared</td>
<td>1% sample of the 2000 Chinese census and Hong Kong natality micro data 1984–2004</td>
<td>Women (men) in most exposed famine cohorts 7.5% (9%) more likely to be illiterate, 3% (5.9%) more likely not to work, and 13% (12%) more likely to be disabled; men 6.5% less likely to be married and 8.2% more likely to have never married</td>
</tr>
<tr>
<td>Dercon &amp; Porter (2010)</td>
<td>Ethiopia</td>
<td>1984 famine</td>
<td>OLS with household FE for effects of exposure to famine in utero and in early childhood on height and primary school completion</td>
<td>479 young adults (ages 17–25) from sixth round of Ethiopian Rural Household Survey 2004, with data from previous rounds</td>
<td>Those exposed to famine in utero or within the first 36 months of life 3.9 cm shorter; children exposed at ages 2–3 years 2% less likely to finish primary school; no effects on schooling for cohort exposed in utero</td>
</tr>
<tr>
<td>Gørgens et al. (2012)</td>
<td>China</td>
<td>1959–1961 famine</td>
<td>OLS/family FE in an econometric model of relationship between height of parents and children; children of famine and control cohorts used to sort out stunting from selection (famine survivors with greater average potential height would have children who were taller than nonfamine cohorts)</td>
<td>First four waves of the China Health and Nutrition Survey, 2,113 families in rural sample and 1,080 families in urban sample</td>
<td>In rural population, large and SS stunting for those under age 5 during famine; for mothers (fathers), estimated effects are 1.49–2.22 cm (1.47–1.80 cm); estimates of selection (the famine coefficient in the OLS regression minus the estimated stunting) for mothers (fathers) are 1.92–2.64 cm (0.85–1.18 cm); results for older famine cohort (born 1948–1956) and the urban sample are weaker but also show stunting and positive height selection</td>
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<tr>
<td>Meng &amp; Qian (2009)</td>
<td>China</td>
<td>1959–1961 famine</td>
<td>2SLS with instrument of exposure with the interaction of nonfamine grain production and birth year in quantile analysis using cross-sectional and cohort variation in famine exposure, measured as the average county-level cohort size of survivors born during the famine, effect of exposure on the 90th percentile of outcome distributions estimated</td>
<td>1990 population census, 1989 China Health and Nutritional Survey, 1997 China Agricultural Census, and the FAO’s GAEZ data on crop suitability; sample is county-birth year cells for cohorts born 1943–1966</td>
<td>Larger adverse impact at higher percentiles with famine exposure; in comparison of individuals exposed and not exposed to famine, those in 90th percentile are relatively worse off than those in 10th percentile; for early-childhood cohort, exposure on average decreased height by 1.6% (2.7 cm), weight by 5% (3.03 kg), weight for height by 1.2% (0.004 kg cm⁻¹), and labor supply by 13.9% (12.7 hours per week)</td>
</tr>
<tr>
<td>St. Clair et al. (2005)</td>
<td>China</td>
<td>1959–1961 famine</td>
<td>Adjusted relative risk ratios across cohorts to find effect of famine in utero and in early childhood on schizophrenia</td>
<td>All psychiatric case records (1971–2001) from Wuhu region of Anhui, 191–779 cases per year for people born 1956–1965</td>
<td>Twofold increase in likelihood of developing adult schizophrenia with prenatal and infant exposure to famine; findings replicate the results from Dutch data</td>
</tr>
<tr>
<td>Umana-Aponte (2011)</td>
<td>Uganda</td>
<td>1980 famine</td>
<td>OLS/probit of effect of famine in utero and in early childhood and famine intensity on years of schooling and literacy, with different combinations of FE across specifications</td>
<td>1991 and 2002 Uganda Population and Housing Censuses from IPUMS-I (702,233 and 302,833 obs., respectively) and Ugandan 2006 DHS (10,743 obs.)</td>
<td>0.364 fewer years of schooling and decreases in the likeliness of completing primary school (4.2%) and being literate (3.1%) in cohort exposed to famine in utero; with family FE, children exposed to famine in utero 7–10% less likely to ever attend school</td>
</tr>
</tbody>
</table>

Abbreviations: 2SLS, two-stage least squares; BMI, body mass index; DD, difference-in-differences; DHS, Demographic and Health Survey; FAO, Food and Agriculture Organization, FE, fixed effects; GAEZ, global agro-ecological zones; GLS, generalized least squares; IPUMS, Integrated Public Use Microdata Series; OLS, ordinary least squares, RE, random effects; SS, statistically significant.
“programming” the development of the fetus (see Almond & Currie 2011 for a review of this fetal effects literature).

Most studies of the in utero effects of famine identify cohorts of affected children and follow them over time. One potential difficulty is that children who were already born at the time of the famine are also subject to nutritional deprivation, although at a later stage. This partial exposure of prefamine cohorts suggests that the in utero cohort must be compared with cohorts in utero after the famine only. However, individuals in utero after the famine were conceived during or after the famine, which raises concerns about fertility responses to the famine, which may change the composition of parents having children. Postfamine cohorts may thus have different background characteristics from the in utero cohort.

The cohort-based approaches provide some evidence of the effects of famine on long-term outcomes. Meng & Qian (2009) and Umana-Aponte (2011) evaluate the effect of famine exposure in utero on years of schooling in China (1959–1961) and Uganda (1980), respectively. Relative to pre- and postfamine cohorts, cohorts exposed to famine in utero attained approximately 0.58 fewer years of schooling in China and 0.364 fewer years of schooling in Uganda. In results from Ethiopia, Dercon & Porter (2010) estimate that a 1984 famine reduced the probability of completing primary school by 2% for the cohort exposed at ages 2–3, but had no effect on education for the cohort exposed in utero. They do, however, find effects on height in both cohorts.

The Chinese famine has received particular attention, and several papers provide evidence complementary to Meng & Qian’s. As with the studies described in the previous paragraph, analyses of the Chinese famine use both pre- and postfamine cohorts as control groups. Chen & Zhou (2007) examine the effects of the Chinese famine on height, finding a decrease of 3 cm for those born or conceived during the famine. Gørgens et al. (2012) report a similar stunting effect of the famine on the rural Chinese population for whom the famine was most severe. In further analyses of socioeconomic outcomes, Almond et al. (2010) estimate that women (men) exposed to the Chinese famine in utero were 3% (5.9%) less likely to work and 13% (12%) more likely to be disabled and that men were 6.5% less likely to be married. Finally, building on earlier research examining the effects of the Dutch “hunger winter” of 1944 on a wide range of outcomes (see, e.g., Lumey et al. 2011), two papers investigate the effects of the 1959–1961 Chinese famine on other health outcomes. Fung (2009) demonstrates that in utero exposure to famine predicts obesity for women, but not for men, whereas St. Clair et al. (2005) find that prenatal and infant exposure to famine doubles the likelihood of developing schizophrenia.

Because famine often involves substantial mortality, it can be difficult to interpret the magnitudes of the effects. Indeed, the data suggest that mortality selection may severely dampen the estimated effects of early-life shocks on the average height of adult survivors. For example, data on Chinese adults show little sign of a decline in average heights among cohorts exposed to the famine in early life unless one accounts for selection (Gørgens et al. 2012). In Meng & Qian’s study (2009), stunting effects are most noticeable at the upper quantiles of the height distribution, as would be expected if mortality culled individuals from the bottom tail of the height distribution.

### 3.3. Disease

Historical studies in developed countries suggest that the introduction of effective treatments for childhood disease had profound effects on future outcomes and population health. For example, Bhalotra & Venkataramani (2011) examine the introduction of sulfa drugs in the United States in the 1930s, which were highly efficacious for pneumonia. Pneumonia was a leading killer of children in the United States, much as it is in the developing world today. They compare areas with initially high death rates due to pneumonia with areas with initially lower rates, arguing that sulfa
drugs ought to have had a greater impact in the former than in the latter. They find that sulfa drug availability in utero and in early childhood increased years of schooling by 0.09 years, income by 2.3%, and employment by 1.2% for men. The results for women are small and insignificant, which they interpret as evidence that the gains to men arose primarily because better health led to increased returns in the labor market. Using a somewhat similar research design, Bleakley (2007) compares school enrollment, attendance, literacy, and years of schooling of individuals born in areas with high and low hookworm endemicity, before and after the eradication of hookworm in the United States. He finds that a one-standard deviation reduction in the hookworm rate increased school enrollment by 0.25 standard deviations and increased literacy and full-time attendance, but it had no effect on years of schooling.

Table 5 summarizes several recent papers examining the effect of early-life disease exposure on future outcomes in developing countries. The papers deal with both endemic and epidemic diseases, taking advantage of both increases and decreases in incidence. In particular, several studies exploit variation in the incidence of endemic disease—such as helminth infection and malaria—that was induced by large-scale eradication campaigns.

One set of papers (Nelson 2010 on Brazil and Lin & Liu 2011 on Taiwan) investigates the long-term effects of in utero exposure to influenza using a cohort comparison methodology similar to that in Almond (2006). These papers examine the impact of exposure to the 1919 influenza epidemic by focusing on the affected cohort’s break from trend relative to cohorts born just before and after the outbreak. Following Almond (2006), both Nelson (2010) and Lin & Liu (2011) also incorporate geographic variation in mortality as an indicator for the intensity of prenatal influenza exposure. The 1919 Brazilian cohort that was exposed to the pandemic in utero finished 0.2 fewer years of schooling, had 20% lower wages, and was 8.6% less likely to be employed. In Taiwan, each 1 percentage point increase in the birth-year maternal mortality rate (a proxy for influenza incidence) led to a reduction of 0.343 years of schooling. Those affected were also 3.6%, 2.1%, 1.6%, and 0.06% less likely to complete elementary school, junior high school, high school, and college, respectively, relative to cohorts born just before or just after 1919.

It is somewhat difficult to compare effect sizes when baseline levels of outcomes such as education are so different across countries. Almond (2006) reports that the US cohort affected by the 1919 epidemic had 0.125 fewer years of schooling, earned 5% lower wages, and was 20% more likely to be disabled. This comparison suggests that flu exposure may have had a larger effect on future wages in Brazil than in the United States. Kelly (2009) finds small effects of the 1957 Asian flu pandemic on British children’s test scores at ages 7 and 11. Just as we have speculated that the effects of early health shocks might be smaller in developed countries than in developing ones, it is possible that the effects of the 1957 flu pandemic were smaller than those of the 1919 flu pandemic because of improvements in medical care or in the underlying health of those affected. Note that if the extent of mortality selection had fallen between 1919 and 1957, then we would expect the estimated effect of the later epidemic to be larger than that of the earlier epidemic.

Several studies estimate the effect of specific diseases by examining eradication campaigns in a difference-in-differences framework, much as in Bhalotra & Venkataramani (2011) and Bleakley (2007) described above. This research strategy compares cross-cohort changes in areas with high initial disease prevalence with cross-cohort changes in areas that have lower initial disease prevalence because the former are more likely to be affected by the eradication campaigns.

Three papers apply this method in asking whether early-life malaria exposure affects socio-economic outcomes in adulthood. Using data from the US South and Latin America, Bleakley (2010) estimates that 100% eradication of malarial infections increased subsequent adult income by 47% in the United States, 45% in Brazil, 45% in Colombia, and 41% in Mexico. Bleakley also finds positive effects on literacy but mixed results for years of schooling. Cutler et al. (2010),
### Table 5  Effects of childhood disease exposure on adult outcomes

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<tr>
<td>Baird et al. (2011)</td>
<td>Kenya</td>
<td>Primary schooling deworming project, 1998–2009</td>
<td>Calibrated version of the Grossman model, in which investments in health increase future endowments of healthy time</td>
<td>Data from Miguel &amp; Kremer (2004), follow-up with participants a decade later</td>
<td>Increases in schooling (0.3 years) and test scores (0.112 SD) for deworming, but no effect on grade completion; those treated also report better health; in full sample, hours worked increased by 12%; for those working for wages, hours worked increased by 20%</td>
</tr>
<tr>
<td>Bleakley (2010)</td>
<td>United States, Brazil, Colombia, and Mexico</td>
<td>DDT spraying in the 1950s (1920s for the United States)</td>
<td>OLS and 2SLS with temperature and altitude as instruments to compare labor market outcomes of children born well before and just after eradication campaigns in regions with different pretreatment malaria rates</td>
<td>For United States, Brazil, and Mexico (Colombia), IPUMS censuses 1960–2000 (1973–1993), males ages 25–55, born 1905–1975 (1918–1968), various sources for malaria data</td>
<td>For the United States, 0.47 and 0.60 (occupational income score and Duncan index); for Brazil, 0.59 and 0.45 (log total income and log earned income); for Colombia, 0.45 (industrial income score); and for Mexico, 0.41 (log earned income) for effect on adult income per probability of childhood infection (reduced-form difference divided by pre-eradication malaria infection rate)</td>
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<tr>
<td>Cutler et al.</td>
<td>India</td>
<td>DDT spraying campaigns, 1950s and 1960s</td>
<td>District and cohort FE to compare log household monthly per-capita expenditure for those born before and after the eradication campaign in areas with different pretreatment malaria prevalence</td>
<td>National Sample Survey 1987, men (111,218 obs.) and women (107,551 obs.) ages 20-60, government information on 1948 malaria endemicity</td>
<td>No robust evidence of an effect of malaria eradication on human capital attainment; assuming malaria levels were reduced to zero after campaign, 40-PP reduction in the spleen rate (as in the most malarious states) associated with a 2% increase in per-capita household expenditure for treated men; no impact on women</td>
</tr>
<tr>
<td>Lin &amp; Liu (2011)</td>
<td>Taiwan</td>
<td>1918 influenza pandemic</td>
<td>Maternal mortality rate as a proxy for the degree of exposure with outcomes regressed on maternal mortality rate, infant mortality rate, region dummies, and linear trend to find impact of influenza in utero on later-life outcomes</td>
<td>Dynamic Census of the Taiwanese Population, 1980 Population and Housing Census, 1989 Survey of Health and Living Status of the Elderly, 1927 Health Statistics for School Students</td>
<td>1-PP increase in maternal mortality rate associated with a decrease in the probability of being literate (2.9%); finishing elementary (3.6%), junior high (2.1%), and senior high school (1.6%), and college (0.6%); decrease in cohort size (1%); increase in probability of having kidney disease (29%), vertigo (45%), tinnitus (23%), circulatory disease (27%), thyroid problems (53%), and respiratory disease (23%); and decrease in average student weight (0.5 kg) and height (0.5 cm)</td>
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<tr>
<td>Lucas (2010)</td>
<td>Paraguay and Sri Lanka</td>
<td>Malaria eradication campaigns with DDT interior residual spraying, initiated in 1945 in Sri Lanka, 1967 in Paraguay</td>
<td>Region and cohort FE used to compare education outcomes of women born before/after eradication campaigns in regions with different pre-eradication malaria intensity; also uses percentage of years less than 18 before eradication</td>
<td>1987 DHS, ever-married women ages 18–49 (5,822 obs.) for Sri Lanka; 1990 DHS, ever-married women ages 18–49, born 1958 or later (2,931 obs.) for Paraguay</td>
<td>Large and SS increase in schooling from malaria eradication, 2–3 years for worst-off regions; 10-PP decrease in the incidence rate in Sri Lanka (Paraguay) leads to SS increase in schooling by 0.114 (0.118) years, primary schooling by 0.051 (0.056) years, high literacy by 0.924 (2.083) PP, and minimal literacy by 0.846 (0.631) PP</td>
</tr>
<tr>
<td>Miguel &amp; Kremer (2004)</td>
<td>Kenya</td>
<td>Primary schooling deworming project, 1998–1999</td>
<td>Estimation of cross-school treatment externalities by including number of pupils in primary schools within 3 km from each school each year and number treated to look at effect of program on health and education outcomes; also tries to back out within-school externalities</td>
<td>INCAP pupil/school questionnaires 1998 and 1999, parasitological survey by Kenyan Ministry of Health, 75 schools with total enrollment of 30,000 children, ages 6–18</td>
<td>7-PP increase in school participation in treatment schools; proportion of pupils with moderate/heavy infection 23 PP lower in treated schools, strongly SS; this is decomposed into a 12-PP reduction from within-school externalities and a 14-PP reduction from direct effects of deworming; cross-school externalities: each additional 1,000 attending a treated school within 3 (3–6) km associated with 26 (14) PP fewer infections</td>
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<td>Nelson (2010)</td>
<td>Brazil</td>
<td>1918 influenza pandemic</td>
<td>Education and labor market outcomes regression on an indicator for being born in 1919 (or quarter of birth), yearofbirth, and yearofbirth², with logit for binary and OLS for continuous variables</td>
<td>Pesquisa Mensal de Emprego 1986–1998, people born 1912–1922 in the six largest metro areas (379,930 obs., 41,315 with wage data)</td>
<td>Those born in 1919 13% less likely to graduate from college, 5% less likely to be employed, 8.6% less likely to have formal employment, and with 0.046 less years of schooling than those born in other years from 1912 to 1922; for each outcome, the coefficient on first- or second-quarter birth dummy (or both) is SS and in the direction that would support the fetal origins hypothesis</td>
</tr>
<tr>
<td>Ozier (2011)</td>
<td>Kenya</td>
<td>Primary schooling deworming project, 1998–2010</td>
<td>Various cognitive outcomes regression on indicators for age at deworming to look for long-term effects of deworming; estimates impact on younger children who did not receive treatment directly</td>
<td>Height, weight, migration, and cognitive data on &gt;20,000 children (ages 8–14 in 2009 or 9–15 in 2010) at the 73 deworming project schools</td>
<td>Large cognitive effects of deworming on children who were less than age 1 when their communities received mass deworming treatment, equivalent to 0.5–0.8 years of schooling; effects also estimated among children likely to have older siblings in school to receive the treatment directly; in this subpopulation, effects are nearly twice as large</td>
</tr>
</tbody>
</table>

Abbreviation: 2SLS, two-stage least squares; DHS, Demographic and Health Survey; FE, fixed effects; INCAP, Institute of Nutrition of Central America and Panama; IPUMS, Integrated Public Use Microdata Series; OLS, ordinary least squares; PP, percentage points; SD, standard deviation; SS, statistically significant.
studying India’s malaria eradication campaign of the 1950s, also detect no effect on educational attainment. However, consistent with Bleakley (2010), they find that malaria eradication increased household consumption, perhaps implying that the eradication of malaria increases incomes by allowing people to work more, or at higher-productivity jobs. These results highlight that reductions in disease can work through multiple channels and can have effects on income through improved health or labor force participation, even if they have no effects on schooling or cognition. Indeed, in some circumstances, improvements in health may have positive effects on earnings even if they decrease educational attainment. Even so, not all studies of malaria eradication fail to find effects on educational attainment. In her study of eradication in Paraguay and Sri Lanka, Lucas (2010) estimates that a 10–percentage point reduction in malaria incidence increases female schooling by 0.1 years and female literacy by \( \sim 1–2 \) percentage points.

Complementing Bleakley’s (2007) work on the historical United States, several studies examine the effect of deworming on medium- and long-term outcomes in developing countries. In a well-known paper, Miguel & Kremer (2004) analyze the experimental introduction of deworming treatments in Kenyan schools. The deworming intervention increased school attendance by 7 percentage points but had no effect on test scores. In a follow-up study, however, Baird et al. (2011) find that self-reported health, years of schooling, and test scores increased for the treated group. Hours worked rose 12% in the full sample and 20% among those who were working for wages. Moreover, Ozier (2011) shows that deworming school-aged children had positive externalities on very young children in the same community, fitting for a communicable disease such as helminth infection. Children less than one year old during the treatment experienced IQ gains on the order of 0.2 standard deviations, which translated into an additional 0.5–0.8 years of schooling.

3.4. Pollution

The literature on determinants of child health in developing countries has traditionally focused on disease and nutrition.\(^{13}\) However, children are more vulnerable than adults to the effects of many types of pollutants, both because they are still developing and because their small size means that they may end up with relatively high doses per unit body mass. For example, children are more susceptible to lead poisoning because the blood-brain barrier that protects adults is not fully formed. These considerations suggest that the alarmingly high levels of contamination of air, water, and soil in many developing countries could have extremely harmful long-term consequences for child development.

In many cases, the pollution levels in developing countries are outside the range that has been studied in the developed world and may be outside the range of pollution levels that existed historically. For example, in Chay & Greenstone’s (2003a,b) pathbreaking work examining reductions in total suspended particles (TSPs) in the 1970s and early 1980s in the United States, low TSP concentrations were 44 \( \mu \text{g m}^{-3} \) and high TSP concentrations were 92 \( \mu \text{g m}^{-3} \). Wang et al. (1997) study pollution in residential areas of Beijing from 1988 to 1991 and consider TSP levels ranging from 150 to 700 \( \mu \text{g m}^{-3} \). Similarly, roughly 70% of the river water in China is unsafe for human consumption, although many people in rural areas rely on these sources for drinking water (World Bank 2006). Brainerd & Menon (2011) find that a 10% increase in chemical runoff from fertilizers in the water increases infant mortality by 11.26% and decreases height for age by 0.14 standard deviations.

\(^{13}\)In many settings, these killers remain the greatest threats. For example, Field et al. (2011) show that when households in Bangladesh abandoned tube wells that were contaminated with arsenic, mortality from diarrheal disease doubled.
There is now considerable evidence from developed countries that air pollution has harmful effects on birth weight and infant mortality (see Currie 2011 for a recent summary of this literature), and a growing literature shows that this is also true in the developed world. Foster et al. (2009) study air pollution in Mexico using satellite measures of aerosol optical depth. Variation in pollution comes from a voluntary certification program, and there is regional variation in the supply of auditors available to implement the program. The authors find that a 3.6% improvement in aerosol optical depth resulted in a 16% decline in infant mortality due to respiratory illness but no decline from deaths due to external causes (a control cause). Jayachandran (2009) estimates ordinary least squares models in which an average aerosol index (TOMS) captures the variation in pollution levels that resulted from the 1997 Indonesian wildfires. Her results indicate that areas affected by the smoke suffered a 1.2% reduction in birth cohort size, with the largest effects among the poor, which highlights the importance of selection once again. Despite these concerns about selection bias, Wang et al. (1997) find that each 100-μg m⁻³ increase in SO₂ (TSP) exposure in the third trimester reduces birth weight by 7.3 g (6.9 g) and increases the probability of low birth weight by 11% (10%).

The question of precisely which pollutants are most harmful is important for policy and is difficult to address, given that many pollutants come from the same sources and are highly correlated. Moreover, mechanisms are not well understood. For example, particulates do not cross the placenta (as carbon monoxide does) but may cause an inflammatory response in the mother. Additionally, although a good deal of information has accumulated about the “criteria air pollutants” that are routinely tracked in the United States because of the Clean Air Act, much less information is available about thousands of other toxic chemicals in use.

Little research in any country has tracked the long-term impacts of pollution in any setting, rich or poor. There are a few notable exceptions. Nilsson (2009) finds that in Sweden, reductions in ambient lead concentrations have positive long-term effects on children’s eventual educational and labor market outcomes.¹⁴ Ferrie et al. (2011) show that higher exposure in childhood to waterborne lead reduced test scores for male World War II enlistees. Almond et al. (2009) study the effects of radiation exposure in utero on educational outcomes for children affected by the Chernobyl nuclear disaster in Sweden. In the Chernobyl study, children exposed to radiation at 8–25 weeks gestation in the most affected municipalities were 4% less likely to qualify for high school.

A final caveat is that even in the presence of a clearly defined pollution problem, such as smoke from primitive cooking stoves, the most obvious solutions are not always the most effective. Hanna et al. (2012) evaluate a large-scale randomized trial in which clean cooking stoves were introduced to Indian villages. The stoves apparently had little effect on health mainly because households did not use them. Clearly, much work remains to be done on this topic.

3.5. War

Similar to malnutrition, disease, and pollution, war and exposure to violence threaten health in the short term. Several studies examine their effects on birth and early-childhood outcomes. For example, Mansour & Rees (2012) study the 2000–2005 al-Aqsa Intifada in Palestine using maternal fixed effects models, finding that each additional conflict fatality in the first trimester increases low birth weight by 0.003 percentage points. Similarly, maternal fixed effect estimates from Colombia

¹⁴The lead levels Nilsson studies were already less than the US “thresholds for concern” at the beginning of his sample period, suggesting that even declines from already low levels had positive effects.
indicate that (indirect) exposure to land mine explosions during the first trimester reduces birth weight by 2.8 g (Camacho 2008). Other studies use difference-in-differences methods at the regional level instead of the maternal level. Drawing on this approach, Bundervoet et al. (2009) and Akresh et al. (2007, 2012) estimate the effect of early-childhood violence exposure on height. For example, Bundervoet et al. (2009) exploit variation in civil war timing across cohorts and provinces in Burundi. Among children ages 6–60 months, height-for-age $z$ scores decreased by 0.047 for each month of exposure to civil war.

A growing literature, reviewed in Table 6, suggests that exposure to war in early childhood may also have significant long-term scarring effects. Aguero & Deolalikar (2012) estimate the effect of childhood exposure to the Rwandan genocide on women’s height-for-age $z$ scores. Using difference-in-differences methods for exposed cohorts with Zimbabwean women as the control group, they find that Rwandan women exposed to the conflict have 0.2–standard deviations lower height-for-age $z$ scores. Taking Akresh & Verwimp (2007) and Aguero & Deolalikar (2012) together, it appears that the Rwandan Civil War greatly affected the height of girls in Rwanda and that even exposure late in adolescence affected adult female height.

Several papers evaluate the effects of conflict on years of schooling. Alderman et al. (2006) combine maternal fixed effects and instrumental variables estimation to show that exposure to civil war and drought shocks in early childhood in Zimbabwe reduced children’s height-for-age $z$ scores, which subsequently had negative effects on years of schooling and adult height. Akresh & de Walque (2008) estimate a difference-in-differences model and find that children exposed to the Rwandan genocide earned 0.4 fewer years of schooling. In Peru, León (2010) estimates that for each additional year of civil war exposure in early childhood, children acquired 0.07 fewer years of schooling. Therefore, a child exposed for the first three years of life attains, on average, 0.21 fewer years of schooling. Some of this effect on education may work through the destruction of school buildings.

Because the exposure occurred in childhood, rather than in utero, the authors posit that the channel through which the violent event affects height is economic or nutritional. Some authors also emphasize the possible effects of extreme stress or of being physically unable to attend school. These types of explanations may also apply to other papers about the effects of exposure to disaster, such as that by Sotomayor (2013), who compares Puerto Rican cohorts in utero during the hurricane years of 1929 and 1933 with surrounding cohorts, finding the in utero cohort more likely to have been diagnosed with high blood pressure, high cholesterol, or diabetes as adults.

War has long been regarded as one of the “four horsemen of the apocalypse,” suggesting that people are well aware of its unique capacity for destruction. This new literature does not help us to distinguish the mechanisms underlying its long-term effects on children, although it does confirm the long reach of such disastrous events. At the same time, we should note that wars vary in duration and intensity, and the effect of a sustained civil war probably differs from that of an acute stressful period during gestation.15

4. DISCUSSION AND CONCLUSIONS

A growing literature shows that events in early life have long-term consequences for adult health, cognition, and labor market success, both in developed and in developing countries. Still, many basic questions remain unanswered. We still know relatively little about mechanisms. And it is

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15In fact, issues of duration and intensity affect the interpretation of many studies discussed in the earlier sections. For example, the effects of short-term variations in malaria incidence may differ from the effects of a sustained eradication of malaria.
### Table 6 Effects of childhood war exposure on adult outcomes

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Shock</th>
<th>Methods</th>
<th>Data/size</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Akresh &amp; de Walque (2008)</td>
<td>Rwanda</td>
<td>Rwandan genocide, 1994</td>
<td>DD to look at whether children ages 6–15 in 2000 (exposed to the genocide) have lower educational attainment than similar children in 1992, relative to older cohorts (16–35) with completed schooling</td>
<td>DHS for Rwanda 1992 and 2000, 27,114 and 18,528 individuals, respectively, ages 6–35</td>
<td>0.421 fewer years of schooling for children exposed to the genocide, effect stronger for males and those from nonpoor households (who previously had an advantage)</td>
</tr>
<tr>
<td>Aguero &amp; Deolalikar (2012)</td>
<td>Rwanda</td>
<td>Rwandan genocide, 1994</td>
<td>DD across cohorts and country of birth to compare heights in treated group (Rwandan women under 21 in 1994) to various controls (mainly older Rwandan women and women in Zimbabwe)</td>
<td>DHS of Rwanda 2001 and 2005, all women ages 6–40 in 1994, and Zimbabwe 1999 and 2005–2006, Kenya, Tanzania, Mozambique, and Zambia (27,910 obs.)</td>
<td>Decrease in height-for-age z scores of 0.16–0.2 SD and an increase in probability of being stunted by 6.9–7.3 PP for genocide exposure; shock effect decreases with age, but SS impacts found even for those ages 13–18 during the genocide</td>
</tr>
<tr>
<td>Akresh et al. (2011)</td>
<td>Nigeria</td>
<td>Nigerian Civil War of 1967–1970</td>
<td>DD across war exposure and ethnicities to look at impact of war (months of war exposure interacted with exposed ethnicity) on height; controls for war exposure and ethnicity-specific linear trends, and ethnicity, birth year, state, and survey-year FE</td>
<td>Nigerian DHS 2003 and 2008, 13,407 women born 1954–1974</td>
<td>SS effect of war on height for each age group; war-exposed girls ages 0–3 during the conflict had average exposure of 17.5 months and 0.7-cm decrease in adult height relative to unexposed girls in same cohort; similar effects for girls ages 4–12 during the war; effects larger for girls ages 13–16, who suffered a height deficit of 4.53 cm (two-thirds the sample SD) from a mean exposure of 20.6 months</td>
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<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Shock</th>
<th>Methods</th>
<th>Data/size</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alderman et al.</td>
<td>Zimbabwe</td>
<td>Civil war (log of number of days a child was alive before August 18, 1980), two-year drought (1982–1984)</td>
<td>IV-maternal FE regression using civil war and drought shocks as an IV for early-childhood nutritional status across siblings to look at impact of child height on grade attainment and age starting school</td>
<td>Longitudinal surveys in three resettlement areas, 400 households in 1983–1984, 1987, and annually 1992–2001, 680 children ages 6 months to 6 years at baseline, with 19 years of follow-up</td>
<td>SS decreases in child height-for-age z scores by $-0.035$ to $-0.049$ (–0.576 to –0.729) for civil war exposure (drought); in maternal FE-IV regressions, larger preschool height associated with greater attained height in adolescence, number of grades attained, and (more weakly) starting school younger</td>
</tr>
<tr>
<td>Blattman &amp; Annan (2010)</td>
<td>Uganda</td>
<td>Indiscriminate abduction of children (as young as age 5, mostly 10–15) to be soldiers for the Lord’s Resistance Army (LRA), late 1990s</td>
<td>OLS, weighted least squares, and treatment effect bounding for selective attrition to look at impact of abduction on education, labor market, and health outcomes</td>
<td>Survey of War Affected Youth 2005–2006, 741 boys born 1975–1991 in Northern Uganda, 462 were once with the LRA</td>
<td>Substantial loss of education (0.78 years, 11% decrease) and literacy (17% less likely to be literate) with abduction; no impact on probability of employment, but abductees half as likely to be in skilled work; 22–36% lower wages; psychological distress in those that experienced the most violence (abducted or not)</td>
</tr>
<tr>
<td>León (2010)</td>
<td>Peru</td>
<td>Peruvian civil conflict, 1980s and 1990s</td>
<td>District and birth-year FE and a province-specific cubic time trend to look at effect of exposure to violence (in years) in each period of life (ages 2–3, 4–6, 7–12, 13–17) on educational attainment</td>
<td>1993 and 2007 censuses, and data on human rights violations from the Peruvian Truth and Reconciliation Commission</td>
<td>0.12–0.19 fewer years of education for average person exposed to violence; for each additional year of civil war exposure in early childhood, children acquired 0.07 fewer years of schooling</td>
</tr>
</tbody>
</table>

Abbreviation: DD, difference-in-differences; DHS, Demographic and Health Survey; IV, instrumental variables; FE, fixed effects; OLS, ordinary least squares; PP, percentage points; SD, standard deviation; SS, statistically significant.
difficult to say much about the relative magnitudes of the effects of different types of shocks, or about possible interactions between them, given data limitations and differences in methodology across studies.

Moreover, although many suggest that critical periods—times in the life of the child when negative health insults are most harmful—exist, it is generally unclear when these periods are and to what extent they differ across insults. Much of the recent literature has focused on shocks sustained in utero. This focus is partly because of the belief that gestation is a critical period, but also because it is a well-defined period with a distinct beginning and end—unlike, for example, early childhood. The sharp definition facilitates the measurement of exposure.

Similarly, little evidence is available on intergenerational effects, although these effects represent one of the most intriguing explanations for how the consequences of health shocks can persist over long periods. Along these lines, Bhalotra & Rawlings (2011) link mothers in 38 developing countries to infant health and height-for-age $z$ scores in early childhood. They show that a one-standard deviation decrease in a mother’s height is associated with a 7.4% increase in the probability that her child has a low birth weight and a 9.3% higher neonatal mortality rate. The effects were largest for mothers at the bottom of the income distribution. In a companion paper, Bhalotra & Rawlings (2013) argue that improvements in the health and educational environment were associated with reductions in these intergenerational correlations. Although these associations are compelling, they are not obviously causal. Even so, a follow-up study of the INCAP nutritional intervention discussed above provides additional evidence on intergenerational links. Behrman et al. (2009b) show that children born to mothers who received a protein supplement in childhood had babies who were 116 g heavier and 0.26 $z$ scores taller on average. Furthermore, Fung & Ha (2010) find evidence that the Great Chinese Famine reduced the heights and weights of survivors’ children.

That events in early childhood have long-term consequences says little about the extent to which the actions of parents, schools, or other institutions can ameliorate these consequences. The extent to which parents reinforce or compensate for early-life shocks is an active area of research: Rosenzweig & Zhang (2009) find evidence of reinforcement in China, whereas Liu et al. (2009) and Bharadwaj et al. (2010) find evidence of compensation in the Philippines and Chile, respectively. Parents’ decision to compensate or reinforce may depend not only on beliefs and values, but also on the practical supports available to them.

Finally, to our own surprise and chagrin, we find that little research on the impact of HIV/AIDS on children falls under the scope of our review. Perhaps this omission results from the fact that the epidemic so obviously appears to be a catastrophe with long-term impacts for many children’s lives. It is estimated that more than a quarter-million children under age 15 die of AIDS-related causes each year, while more than 3 million are currently living with HIV/AIDS, 90% of them in sub-Saharan Africa (WHO 2011). Beyond the direct health impacts, HIV/AIDS may also have indirect effects through the devastation of families. As of 2003, an estimated 15 million children had lost at least one parent to AIDS, 80% of them in sub-Saharan Africa (UNAIDS et al. 2004), where research suggests that parental death reduces school enrollment (Case et al. 2004, Case & Ardington 2006, Evans & Miguel 2007). Many others have sick caretakers as a result of the virus.

Case & Paxson (2011) make the further important point that the societal response to disease can impact long-term child outcomes through its effects on health infrastructure. Using data from 14 sub-Saharan African countries, they find that in countries heavily impacted by HIV/AIDS, skilled birth attendance, antenatal care, and immunization rates declined between 1988 and 2005. Countries with a minimal burden from HIV experienced no such declines. This deterioration, which presumably occurred because of a diversion of health resources to fight the AIDS epidemic, may have lasting effects on children, even if neither they nor their parents are HIV positive.
In summary, research in the past decade has shown that health shocks in early life have long-term effects on adults in both the developed and developing worlds. But the literature still provides little understanding of heterogeneity in these effects. Future research should focus on identifying pathways and mechanisms, measuring the relative magnitudes of the effects of different health shocks, examining interactions between shocks, and revisiting the question of critical periods. Beyond these issues, policy makers have much to learn from new findings on the efficacy of interventions designed to help children reach their full potentials.

SUMMARY POINTS

1. Height is a proxy for the early-life environment.
2. There is a strong and consistent relationship among birth weight, height, and adult outcomes such as education, earnings, and health status.
3. Estimated effects of early-life health shocks may be obscured by mortality selection.
4. There is substantial evidence that childhood exposure to famine or less severe nutritional shocks, disease, pollution, or war and violence has a significant long-term effect on adult outcomes.

FUTURE ISSUES

1. What are the mechanisms connecting health shocks in childhood with adult outcomes?
2. What are the relative magnitudes of the effects of different types of shocks, and how do they interact?
3. Are there critical periods, and when do they occur?
4. Are there intergenerational effects of negative health shocks in early life?

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