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Cognitive and Economic Development

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Abstract

A burgeoning literature has found that early childhood health conditions of individuals have large causal effects on their cognitive development, education and earnings. How much does early cognitive development contribute to the national economy? Although researchers have long studied the role of worker health for economic growth, they have not assessed the role of early cognitive development.

Cognitive ability is the foundation of human capital, affecting both educational attainment and economic growth. The risk factors for poor cognitive development are very high in many countries. Each risk factor also causes child mortality, making child survival a viable proxy for good cognitive development conditions. The cognitive development of current workers happened decades earlier when they were children, providing a predetermined correlate. Controlling for country characteristics, income levels and worker health among other variables, child survival from a generation ago is one of the strongest correlates of economic growth in both low and high income countries. Unusually clear

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causal microeconomic evidence together with a strong correlation with economic growth suggests that early cognitive development plays a significant role in economic development, in addition to being a determinant of life-long wellbeing.

1 Introduction

Cognitive ability is the characteristic that most distinguishes human behavior from that of other animals (Harari, 2015). It is the essential ability which enables the application of science and technology to production, the hallmark of modern economic growth and the source of high standards of living.

The causes of general economic development are inevitably assessed by comparing the experience of different countries. These causes are of vital interest, but the high standards of causal evidence in microeconomic research are largely unavailable. Potential causes such as effective institutions, exporting, convergence, education, or health have very little rigorous causal evidence. These hypotheses rely on careful assessment of correlations across countries.

Early cognitive development, which has received almost no attention as a cause of general economic development, has more credible causal evidence of substantial effects at an individual level than any of the more familiar potential causes of economic growth. This paper tries to assess the magnitude of these impacts on economic growth rather than evidence of causality.

The understanding of cognitive development has undergone a profound shift since the 1960s. Cognitive development before and after birth is much more sensitive to environmental influences than previously understood. Researchers have mapped a cascading chain of critical periods for the development of brain functions, where each stage depends on the progress of the previous stage. Catch-up becomes more difficult or impossible after a critical period ends (Hensch, 2004). Because brain plasticity declines sharply at about age 24 months, health researchers have identified the first 1000 days after conception as the brain's "window of opportunity" for good cognitive development (Cusick & Georgieff, 2013).

Undernutrition during pregnancy and early childhood, often in synergy with chronic illness, robs the developing brain of nutrients and causes cognitive deficits. Many factors have only recently been identified as affecting cognitive development (Walker et al., 2007). Specific micronutrients are necessary for good cognitive development, notably iron and iodine, which have very high rates of deficiency around the world. Certain diseases interfere with brain development, especially those like malaria and helminth infections which cause anemia. Exposure to violence is a major risk factor. Absence of nurture can cause severe cognitive deficits, and nurture mitigates the impact of many other risks. Certain toxics such as lead, air pollution (including from cookstoves and smoking) and alcohol during pregnancy diminish cognitive ability. Interactions between risk factors usually magnify the impact of individual risks.

Individuals with cognitive deficits at the level of intellectual disability (previously called mental retardation) clearly learn more slowly in school and are less productive at work, if they can find it. Recently, a large body of evidence shows that even modest levels of many risk factors, commonly occurring in both low and high income countries, worsen school performance and labor market outcomes for individuals (Almond et al., 2018). How big is the impact of fetal and early child cognitive development on economy-wide productivity and economic growth? It is intuitive that a workforce with diminished capabilities would make an economy less productive. But it is less clear whether cognitive deficiencies are common enough or severe enough to be important for the economy.

Cognitive ability includes the capacity to learn.[1] Hence it affects the efficiency of investment in education and the level of human capital in an economy. Cognitive ability impacts learning and adaptation on the job. I present a simple model in which cognitive development contributes to economic growth through its impact on the efficiency of learning in addition to its impact on

[1]The American Psychological Association defines cognition as "All forms of knowing and awareness, such as perceiving, conceiving, remembering, reasoning, judging, imagining, and problem solving" (APA, 2021). Tests for evaluating cognitive development deficiencies, such as Raven's Progressive Matrices (Raven, 2000), are tests of the ability to solve novel problems rather than tests of knowledge.

initial human capital. The model incorporates possible spillovers from the cognitive capacity of individuals onto the rest of the economy by increasing the rate of innovation and the adoption of new technology.

Data are not available at a national level for many specific risk factors for poor cognitive development. However, each significant risk is also a cause of child mortality. I therefore use child survival rates as a summary measure of the combined effects of many cognitive risk factors. Child survival has the advantage of incorporating interaction effects of multiple risk factors, for which there is abundant evidence. For instance, children are more likely to die or to have diminished cognitive capacity if they have both low birthweight (a measure of fetal malnutrition) and anemia than if they have just one or the other (Brabin et al., 2001).

Incorporating a measure of early childhood health conditions has implications for the literature on the effect of health on economic growth, which is an area of active debate. A variety of methods for estimating an aggregate production function have found that life expectancy has a large and robust correlation with economic growth (Bloom et al., 2019), while microeconomic estimates of the effect of adult health on wages usually find small effects (Currie & Madrian, 1999). Studies attempting to correct for possible endogeneity of adult health outcomes have found small (Weil, 2007) or even negative (Acemoglu & Johnson, 2007) correlations with economic growth, causing many economists to doubt the causal impact of health. Distinguishing between the effect of early childhood health on cognitive development and the direct effect of adult health on worker productivity provides a resolution of these differing estimates, because life expectancy is an amalgam of both child and adult survival.

The main findings are first, that in a panel economic growth regression when life expectancy is replaced by separate measures of child and and adult survival, only child survival is statistically significantly correlated with economic growth. Second, child survival from a generation earlier is strongly correlated with economic growth, as one would expect given that early child development affects worker productivity.

2 Determinants of Cognitive Development

Numerous discoveries in past half century have revealed that human brain development is much more sensitive to environmental stimuli than previously understood. For instance, in the 1960s David Hubel and Torsten Wiesel demonstrated that lack of visual stimulus from birth blocks creation of the visual processing circuitry in the brain (Nobel lecture in Wiesel, 1982). If sight is blocked long enough, as in the case of untreated glaucoma, the child will remain blind even after visual stimulus is restored despite having fully functioning eyes.

Neurobiologists refer to windows of rapid brain development as critical periods if absence of development is irreversible after the window closes, such as for visual processing, or as sensitive periods if retrieval is possible but more difficult after the period (Hensch & Bilimoria, 2012). The cumulative nature of brain development means that deficits in previous phases can compromise subsequent development. The rapid decline in neuronal plasticity around 24 months of age has prompted the medical community to identify the period from conception to age two as crucial for good child development (Cusick & Georgieff, 2013; Horton, 2008). I focus on cognitive ability because it is especially important for success in school and work, and central to technological progress. Non-cognitive sensory-motor and socio-emotional development also affect later life outcomes, but they share most risk factors with cognitive development, and data and research on their impact is scarce (Grantham-McGregor et al., 2007).

In tandem with advances in brain science, health researchers have found that environmental risk factors have much larger impacts on fetal and early childhood development than previously known. I discuss the impact of nutrition, disease, nurturing, violence, and toxins. The assessment of their role and relative importance in cognitive development is based on a wide-ranging and influential series of reviews in *Lancet* (especially Walker et al., 2007, and Black et al., 2013) unless otherwise noted.

Protein-calorie undernutrition[2] and disease in mothers are major causes of babies being born small for gestational age, a summary measure of faltering uterine growth. Small for gestational age births are estimated to be responsible for 20% of child stunting, a summary measure of child undernutrition (Black et al., 2013, p. 443). Stunting in mothers, which reflects undernutrition during their childhood, puts their own children at high risk for small for gestational age births, a mechanism for intergenerational transmission of poor health.

Child stunting is a well-established risk factor for poor cognitive development. For example, a comparison of studies across four developing countries found consistent estimates that a one standard deviation lower length for age at age 2 was associated with 0.17-0.19 lower standard deviations of cognition at ages 4-9 (Walker et al., 2011).

Researchers have identified many micronutrients that are important for early brain development including iron, iodine, zinc, folate, vitamins A and D, copper and calcium (Prado & Dewey, 2014). The most widespread sources of deficiency are iron and iodine (Walker et al., 2007, p. 147). Animal models and cross-sectional and longitudinal studies show significant effects of iron deficiency anemia on cognitive development. Severe iodine deficiency leads to cretinism with irreversible cognitive disability. Subclinical iodine deficiency is associated with lower IQ scores.

Childhood disease and undernutrition interact synergistically to inhibit child growth and development. Undernutrition compromises the immune system, making the risk and outcomes of disease worse. Undernutrition raises the death rate from diarrhea, pneumonia and measles in children (Black et al., 2013, p. 382). In turn, disease, particularly chronic gastrointestinal disease causing diarrhea, is a major cause of undernutrition because it prevents the absorption of nutrients from food in the gut.

Persistent disease taxes the large energy budget needed for early brain development. The brain's metabolic energy requirements peak at 66% of the body's total energy use at age five (Kuzawa et al., 2014). Some common

[2]Before the obesity epidemic, undernutrition was often called malnutrition, but now it is helpful to distinguish undernutrition from overnutrition.

childhood and maternal diseases interfere with cognitive development by other pathways. Helminth parasitic worms feed on human blood and tissues causing anemia, an important cause of poor cognitive development in addition to undernutrition. A fifth of the world's population (1.5 billion people) is infected with soil-transmitted helminths, and children and pregnant women are most vulnerable (WHO, 2021b). Other prevalent diseases causing anemia are tuberculosis (23% of the world or 1.7 billion people infected, CDC, 2020), schistosomiasis (240 million infections per year, WHO, 2021a), and malaria (230 million infections per year, UNICEF, 2021).

Malaria is endemic in most of sub-Saharan Africa and parts of South and Southeast Asia (Hay et al., 2009). Malaria kills close to a half million people a year, two thirds of them children under age five (UNICEF, 2021). Most severe malaria occurs in children under age five and pregnant women. Pregnant women have three times the risk of other adults because pregnancy suppresses their immune response (Schantz-Dunn & Nour, 2009). Cerebral malaria, the most acute form of malaria causing direct brain injury, is a leading cause of neurodisability in African children (Idro et al., 2010).

Measles leads to encephalitis in about one per thousand children, causing neurologic damage in up to a quarter of those cases (Gastanaduy et al., 2020). Measles infection was almost universal worldwide before a vaccine was developed in the 1960s, when it caused an estimated six million deaths per year. Due to vaccination campaigns, in 2019 cases had fallen to about 870,000 (Patel et al., 2020).

Nurturing, psychosocial stimulation and breastfeeding have powerful effects on child brain development (Black et al., 2013). This was starkly evident in the structural brain differences of Romanian children placed in orphanages with adequate nutrition but virtually no nurturing, and the children's partial recovery if randomly placed in nurturing households at a young-enough age (Sheridan et al., 2012). The role of nurturing is likely one reason that maternal depression and severe stress is strongly correlated with poor cognitive outcomes for children (Walker et al., 2007). Exposure to violence, whether in the household, the community, or in a war zone, has clear detrimental con-

sequences for later cognitive ability and mental health (Walker et al., 2007). Chronic physiological stress, characteristic of poverty, affects cognitive development via high blood pressure, dysregulated cortisol and inflammation among other routes (Evans & Kim, 2013).

Lack of nurture has a synergistic interaction with other deficiencies (Walker et al., 2007). Diseased and malnourished children are difficult and discouraging to nurture, and lack of nurturing makes other deficiencies more severe. Nurture can partially alleviate the impact of deficiencies like stunting (Walker et al., 2007) and even lack of iron (Lozoff et al., 2010).

Toxic substances typically have larger impacts on fetal and early childhood development than on later stages of life (Stein et al., 2002). Lead, a neurotoxin with irreversible effects on cognition, has no known safe level of exposure (Rees & Fuller, 2020). In addition to causing cognitive deficits, exposure in early childhood causes aggression. Lead exposure, even at moderate rates, is strongly correlated with violent crime in studies at the level of the individual, city, county, state and the nation. Reyes (2007) estimated that the phasing out of leaded gasoline in the 1970s accounted for half of the dramatic decline in violent crime in the United States starting in the 1990s. Feigenbaum and Muller (2016) found that the use of lead pipes in new city water systems in the late 19th century U.S. increased the homicide rate by 24% compared to cities not using lead pipes. Global lead production has doubled from 1994 to 2019, mainly for use in lead-acid vehicle batteries and electronics. Blood lead levels are extremely high in low-income countries and still significant in high-income countries (Rees & Fuller, 2020).

Outdoor air pollution in cities (Sunyer et al., 2015), and indoor air pollution from smoking and cooking with biomass (LaFave et al., 2021), compromise the cognitive development of children, as well as being a significant source of child mortality (Imelda, 2020).

It was discovered in the 1970s that alcohol consumption during pregnancy causes neurological birth defects and increases the risk of child death (KennethL. Jones et al., 1973). Since then, researchers have found that both prevalence and the strength of the impact of alcohol is greater than originally

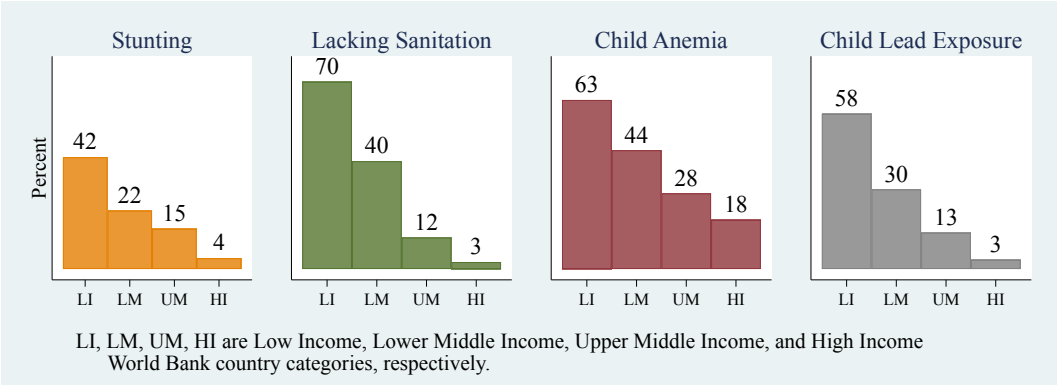

Figure 1: Risk factors for cognitive deficiencies (% of children or population)[3]

thought. For instance, a recent study found rates of fetal alcohol spectrum disorders of 1.5% to 5% of first graders in the United States (May et al., 2018). There is no known safe level of alcohol consumption during pregnancy (Williams et al., 2015).

Organophospate pesticides are a less-researched but potentially important risk for cognitive deficiencies, and their use worldwide is growing very rapidly. Sapbamrer and Hongsibsong (2019) report that 50 out of 57 studies reviewed found lower cognitive scores for children with organophospates in their urine or blood.

How prevalent are risk factors for cognitive deficiencies? We lack national data on many risk factors. Figure 1 shows four major risks for child cognitive development: child stunting (a measure of undernutrition), lack of sanitation, child anemia and child lead exposure. Forty-two percent of children under age five in low income countries are stunted. Two thirds of people in low income countries and one third of people in lower middle income countries lack access to sanitation, which greatly increases disease transmission.

The data for child anemia and lead exposure in Figure 1 are especially concerning. The rates are very high, and there is clear evidence that both cause

[3]Data for Figure 1: child stunting under age 5 in year 2000 (WHO, 2022), sanitation in year 2017 and child anemia under age 5 in year 2019 (World Bank, 2021), and child lead exposure under age 20 in year 2019 (Rees & Fuller, 2020).

irreversible cognitive damage. Child anemia is measured by the percentage of children under age five with blood hemoglobin below a threshold of 100 g/l. Most children in low income countries are anemic (63%). Even in high income countries, 18% of children under five are anemic (World Bank, 2021).

Child lead exposure at the level shown in Figure 1 (blood lead levels greater than 5 μg/dL), results in an average of 4 IQ point decrements (Lanphear et al., 2005) and systematically lower academic scores (Lanphear et al., 2000). Most children in low income countries exceed this blood lead level (Rees & Fuller, 2020).

Exposure to the four risk factors in Figure 1 is extremely high in low income countries and very worrisome in the others. Since the poor likely have a higher risk for each individual factor, they are most likely to be exposed to more than one risk, which usually magnifies the threat to their cognitive development. Growing up in poverty is an especially high risk for cognitive deficits.

Later life consequences

Over the past two decades, a large and steadily growing economics literature has evaluated the life consequences of fetal and early childhood circumstances for individuals. Currie, Almond and coauthors have regularly surveyed this research (Currie, 2009; Almond & Currie, 2011; Currie & Almond, 2011; Currie & Vogl, 2013; Almond et al., 2018). Almond et al. (2018, pp. 1360–2) conclude that most of the reviewed research papers have "compelling research designs", and a broad range of fetal and early childhood risk factors have life-long consequences of "large magnitude".

A seminal study by Currie and Hyson (1999) found that low birthweight British babies were 25% less likely to pass high school exams and, as 33-year-old men, were 8% less likely to be employed. Subsequent studies have found similarly consequential effects of low birthweight on schooling attainment, wages and adult health (for example Bharadwaj, Lundborg, et al., 2013; Bharadwaj, Løken, et al., 2013; Figlio et al., 2014).

In the context of a developing country, Glewwe et al. (2001) assesses grade

school outcomes in the Philippines according to child height for age, a measure of nutritional status. They find that a one standard deviation increase in height for age results in higher test scores in grade school equivalent to 1.1 extra years of school and double that gain for the most malnourished. This is in a context where the average mother has fewer than seven years of education.

The relatively short and well-defined early child development period has helped researchers apply convincing statistical identification strategies. The estimates are more precise than for many other questions and are often surprisingly large for modest health insults. Almond et al. (2018, Table 6) lists 16 recent studies showing substantial impacts of a set of eight risk factors on birthweight: nutrition, diseases, income, stress, violence, smoking, pollution and alcohol. A second group of 18 studies show that the set of eight risk factors also lowers schoolchildren's test scores. Additional risks for low test scores are lack of maternal leave, weather shocks and the education of the mother, a mechanism for the intergenerational transmission of disadvantage. A third group of 11 studies shows that the same set of eight risk factors, with the addition of child care practices and child medical coverage, reduce the adult wages of the children. Many estimated impacts for specific risks are substantially higher in poor than in better-off families, which suggests the compounding effect of multiple risk factors.

A striking aspect of many studies of early life insults is that modest exposures in high income countries nonetheless had significant education and labor market consequences. For instance, in Michigan exposure to Ramadan in the first trimester of pregnancy, during which most pregnant Muslim women fast during the day, resulted in lower birth weights, 19% higher rates of disability, and 6% lower adult wages (Almond & Mazumder, 2008). In New Jersey and Pennsylvania, pregnant women living within 2 kilometers of throughway toll plazas, where there is more air pollution from idling vehicles, were 11% more likely to have low birth weight babies than those living 2-10 kilometers away (Currie & Walker, 2011). Significant impacts in high income countries suggests that much higher exposures in many low income countries are likely to have more severe consequences.

3 A model of the impact of cognitive development on economic growth

If cognitive development has significant effects on individuals, how does that influence economic growth?

In this model, cognitive development impacts economic growth because cognitive skill is a fundamental determinant of human capital, which determines the productivity of labor in the economy. In real life, child cognitive deficits likely occur mainly due to constraints which prevent parents from providing better conditions, or lack of knowledge of its causes. In the model I do not assume that parents optimally choose to undernourish, infect, poison or neglect their children - determinants of cognitive skill are exogenous to parental decision-making. Scientists have only recently discovered some of the important risk factors for cognitive development, so it is unlikely that most parents have the medical knowledge or diagnostic tests to optimize their child's exposure. Public health programs, access to medical care and nutritious food, and shifts in societal child-raising norms cause most of the variation in individual parents' practices.

In the model, parents take into account the cognitive skill and future job prospects of their children when choosing their education. When children start school, their genetic endowment and early cognitive development have allowed them to accumulate a certain initial human capital, h_0. Good cognitive development improves a child's *ability* to learn (Glewwe et al., 2001) so that the rate of growth of human capital in school is also a function of cognitive skills. Human capital of a person, h, grows during schooling from the initial level h_0 according to

$$h = h_0 e^{\eta h_0 s}$$

where initial human capital, h_0, a parameter $\eta > 0$, and years of school, s, affect the growth rate. η can be interpreted as a measure of the quality of schooling because it alters the human capital produced by each year of schooling.

Output per worker in the economy, y, is a function of the level of technology A and human capital per worker, h, as in Soares (2005):

$$y = Ah^{\alpha}.$$

The parameter $0 < \alpha \leq 1$ represents constant or diminishing returns to human capital in production. For simplicity, the production function does not specify physical capital explicitly, assuming it will be optimally allocated.

Parents choose years of schooling to maximize the utility u of their child's stream of future consumption, $c(t)$, at each time t over their lifespan ending at age T

$$u = \int_0^T \frac{c(t)^{\sigma}}{\sigma} e^{-\rho t} dt$$

where ρ is the subjective discount rate and $\sigma < 1$ is a parameter making utility a concave function of consumption. The child's utility is constrained by a lifetime budget requiring the present value of consumption to equal the present value of income from work, which starts after the child finishes school at age s:

$$\int_0^T c(t)e^{-rt} dt = \int_s^T y(t)e^{-rt} dt$$

where r is the interest rate at which the child can borrow or lend.

To focus on the basic mechanisms by which cognitive development can influence economic growth and returns to education, I make simplifying assumptions similar to Soares (2005). The subjective discount rate ρ and the interest rate r are set equal to zero to abstract from life-cycle considerations. These assumptions may also be reasonably realistic. It is not clear why parents would discount the utility of their child's consumption later in life more than earlier. If there is relatively little saving by the child over the course of their lifetime, consumption will be close to income, making interest rates not very relevant.

With these simplifying assumptions, lifetime consumption equals total in-

come during working years.

$$Tc = (T - s)y$$

Parents provide consumption for their children up through schooling age, and the child pays for their own children's consumption while working.

Parents choose schooling s to maximize their child's lifetime utility subject to the budget constraint:

$$\max_s u = T\frac{c^\sigma}{\sigma} \quad s.t. \quad Tc = (T - s)y$$

The chosen years of schooling is $s = T - \frac{1}{\alpha\eta h_0}$. The chosen level of schooling increases with the level of early cognitive development h_0 and lifespan T. Schooling also increases with higher educational quality, η.

Although parents take technology A as given, a country's level of technology A is affected by the level of the world technology frontier, A_w, and the country's average level of human capita, $\bar{h}$.

$$A = A_w\bar{h}^\beta = A_w h^\beta$$

$\beta > 0$ implies that countries with higher education levels use a higher level of technology. $\bar{h} = h$ since all individuals in the model are identical. Bils and Klenow (2000) argue that the level of human capital affects a country's technology level both because a more educated populace is more likely to know about and adopt frontier technology, and because a well-educated workforce is needed to implement high technology production. The common experience of being more productive when working in a more highly skilled team or organization suggests that human capital has productivity spillovers (Arcidiacono et al., 2017). Urban economists consider knowledge spillovers an important explanation for the growth and productivity of cities (Glaeser, 1999).

In this formulation, human capital has a positive spillover from the individual to society, by increasing the national level of technology. Since the individual worker has a vanishingly small effect on average national human

capital, parents ignore the spillover when when choosing education, but the positive externality does provide a social welfare motivation for governments to encourage education.

Applying the parents' choice of education to human capital growth, early cognitive development, h_0, affects economic growth via the following relationships:

$$h = h_0 e^{\eta T h_0 - 1/\alpha}$$

$$y = A_w h^{\alpha+\beta}$$

$$\ln y = \ln A_w \ + \ (\alpha+\beta)(\ln h_0 + \ \eta T h_0 - 1/\alpha).$$

Differentiating with respect to time, t, the growth rate of income per person, $\gamma \equiv \frac{\partial \ln y}{\partial t}$, is positively affected by the growth rate of world technology, γ_{A_w}, and an increase in the level of cognitive development, $\frac{\partial h_0}{\partial t}$,

$$\gamma = \gamma_{A_w} + (\alpha+\beta)(\eta T + \tfrac{1}{h_0})\tfrac{\partial h_0}{\partial t}.$$

Cognitive development affects economic growth because students arrive at school with more human capital (the $\frac{1}{h_0}$ term, where the effect is most potent at low levels of h_0). Cognitive development also enables students to learn faster in school (the ηT term). Cognitive development affects economic growth both due to higher individual worker productivity (the α term in $\alpha+\beta$) and due to greater societal use of frontier technology (the β term).

Increases in cognitive level ($\frac{\partial h_0}{\partial t}$) permanently raise the economic growth rate in the model. The increase on economic growth from higher h_0 due to arriving at school with better cognitive skills gradually peters out as the cognitive level rises (because $\frac{1}{h_0}$ gets small), but the impact due to learning faster in school does not, making the growth rate rise linearly.

In the model, higher cognitive development h_0 raises the chosen level of schooling and the rate of economic growth. What does it do to the societal return to investing in education? The social return to education, R_s, is defined as the percentage increase in income per person due to a one year increase in

average schooling:

$$R_s \equiv \frac{dy}{ds}\frac{1}{y} = (\alpha + \beta)\eta h_0.$$

Cognitive development increases the returns to education because children with better cognitive skills learn faster in school, raising the growth rate of human capital, ηh_0, per year of schooling. Higher average cognitive development, h_0, increases income levels both by raising individual worker productivity (the α term) and by raising the rate of technology diffusion (the β term).

Since the individual worker is not rewarded for his or her contribution to average human capital $\bar{h}$ which raises the level of technology A, the private rate of return to education, R_p, is lower than the social rate of return:

$$\text{if } \frac{dA}{dh} = 0 \text{ for the individual, } R_p = \frac{dy}{dh}\frac{dh}{ds}\frac{1}{y} = \alpha\eta h_0 < R_s.$$

Higher levels of cognitive development h_0 also raise returns to investment in school quality:

$$R_\eta \equiv \frac{dy}{d\eta}\frac{1}{y} = (\alpha + \beta)sh_0.$$

The period of intense cognitive development is short, about three years long, but has lifelong consequences for worker productivity. That makes spending on maternal and early childhood health spending more economically productive than spending on adult health, since the period of adult working years is much longer. In online Appendix A I show that an optimizing government should prioritize early childhood health spending in pursuit of higher living standards.

4 A proxy for cognitive development

It is difficult to create an index of cognitive development risk using measures of specific risk factors because national data are missing for many risk factors and because exposure to multiple risks magnifies the impact of individual risks on cognitive development.

A commonly used partial measure of fetal insults is birthweight, but this does not take into account the first two years after birth which is still critical for cognitive development. Moreover, average birthweight is not available for most countries, nor over time. There are direct measures of cognitive development such as Bayley Scales of Infant Development and various flavors of IQ tests, but they also lack national coverage over time.

The causes of poor cognitive development discussed above, whether maternal and child undernutrition and disease, micronutrient deficiency, lack of nurturing, or exposure to violence or toxins, are each significant causes of child mortality. The child survival rate is a measure of the impact of these *in utero* and childhood insults on survival.

The period of child survival, from birth to age five, does not exactly coincide with the most important period of cognitive development, from conception to age two. However most mortality in the first five years occurs in first one to two years.[4] Additionally, much of the mortality after age two reflects health conditions earlier, making child survival a reasonable proxy for the health conditions which can affect the cognitive development of the children who survive.

Not all risk factors for cognitive development are perfectly proportional to child mortality. For example the risk of death from malaria among the infected children is much higher than the risk of death from soil-transmitted helminth infections, but they are both major causes of anemia which is an important risk factor for cognitive development. Nonetheless, child survival is probably the best single measure of the whole range of risk factors for fetal and early child development, particularly because it reflects the additive and synergistic impacts of multiple risk factors which greatly heighten risk both to survival and for cognitive deficits.

An indication that child survival rates are a reasonable proxy for good cognitive development conditions is that they are highly correlated with several

[4]The world child mortality rate in 2015-2020 was 39.9 deaths per thousand by age 5 and 29.3 per thousand by age 1, which is 73% of deaths by age 5 (United Nations Population Division, 2019).

measures of cognitive capacity. The most direct measures of cognitive ability are IQ and related tests. The first widely used IQ test (Binet-Simon) was developed specifically to measure cognitive deficits in children (Boake, 2002).

National IQ data are imperfect. Lynn and Meisenberg (2010) collected IQ data from published studies in many countries, although the sample sizes, dates of the studies, and age of subjects all vary somewhat.[5] Wicherts et al. (2010) found that the IQ estimates were nonetheless highly correlated with national measures of academic achievement, except for some of the African data. The data used here are those of Lynn and Meisenberg (2010), but replace the Africa data with better quality estimates collected by Wicherts et al. (2010). Data are available for 78 countries and are calibrated to be comparable in 1990.[6]

Standardized knowledge tests for grade school children provide additional measures of cognitive skills across countries. The Trends In International Mathematics And Science Study (TIMSS) and Progress In International Reading Literacy Study (PIRLS) conducted comparable tests of nationally representative fourth graders in many countries over the past two decades. A panel of TIMSS data for 50 countries conducted every four years, and PIRLS data for 46 countries conducted every five years are combined with child survival, gross domestic product (GDP) per capita and education spending data from ten years earlier, all from the World Bank (2021).

Table 1 and Figure 2 show the correlation of child survival with average IQ across countries after controlling for average income levels. The child survival rates and levels of log GDP per capita are for 1980. Since most of the IQ studies tested children age 10-15, calibrated to 1990, 1980 is close to the period of rapid cognitive development for the tested children.

Despite prevalent measurement error in the IQ data, child survival rates account for more than half the variation in average IQ. Conditioned on child

[5]The debate about the cultural specificity of IQ tests is less relevant for these data because most of the studies collected, especially those in developing countries, were non-verbal pattern matching tests such as Raven's Progressive Matrices (Raven, 2000).

[6]Average IQ levels from studies in different years were adjusted for the observed rise in IQ levels over time, known as the Flynn effect (Flynn, 1984). The recent research on the importance of environmental factors for child cognitive development suggests that the Flynn effect could be due to improving health conditions over time.

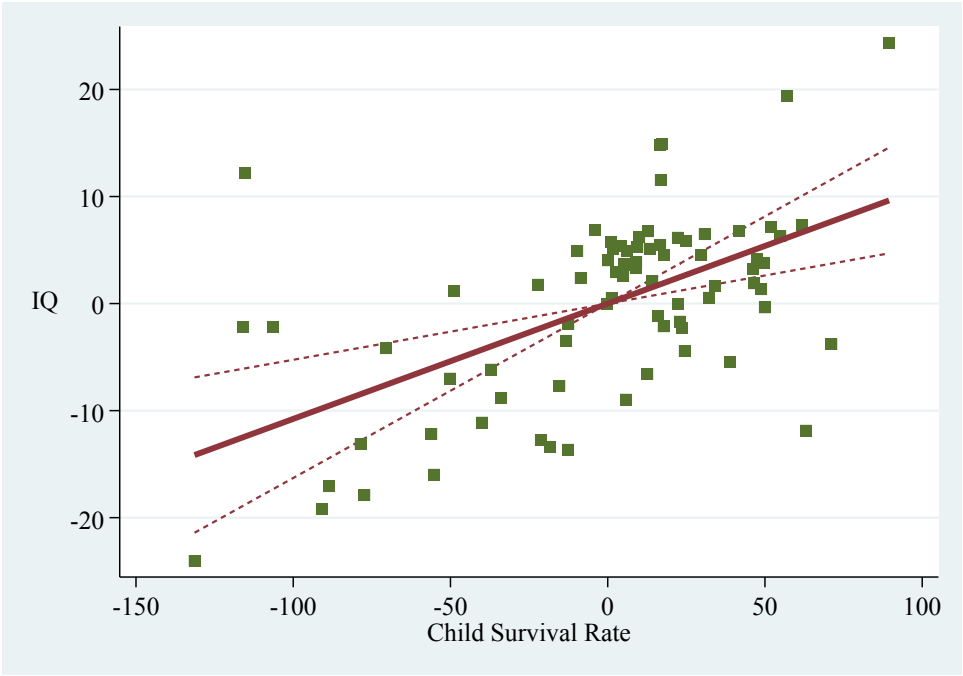

Figure 2: Child survival versus IQ controlling for income

survival, log GDP per capita is not positively correlated with IQ averages.

Table 1 and Figure 3 show that child survival rates are also highly correlated with average math, science and reading scores of fourth graders across countries. Conditioned on child survival, neither log GDP per capita nor educational spending as a percentage of GDP are positively correlated with test scores.

The high correlation of IQ and measures of grade school learning with child survival rates while controlling for living standards supports the notion that child survival is a good proxy for the cognitive development of young children.[7]

[7]G. Jones and Schneider (2006) finds that IQ itself has a robust correlation with a cross-section of national economic growth rates.

Table 1: Cognitive measures and child survival

	IQ	TIMSS Math	TIMSS Science	PIRLS Reading
Child Survival Rate	0.108	2.61	3.33	3.29
	[0.028]***	[0.65]***	[0.64]***	[0.47]***
log GDP per capita	-0.09	-3.8	-12.7	-19.7
	[1.47]	[14.3]	[13.5]	[12.6]
Education expenditure		-9.52	-7.95	-6.47
(% of GDP)		[6.19]	[5.75]	[3.51]
Constant	-10.60	-1,997	-2,621	-2,502
	[16.35]	[559]***	[551]***	[378]***
R^2	0.55	0.39	0.48	0.56
N	78	117	117	116

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

Robust standard errors [in brackets], which for TIMSS and PIRLS data are clustered by country.

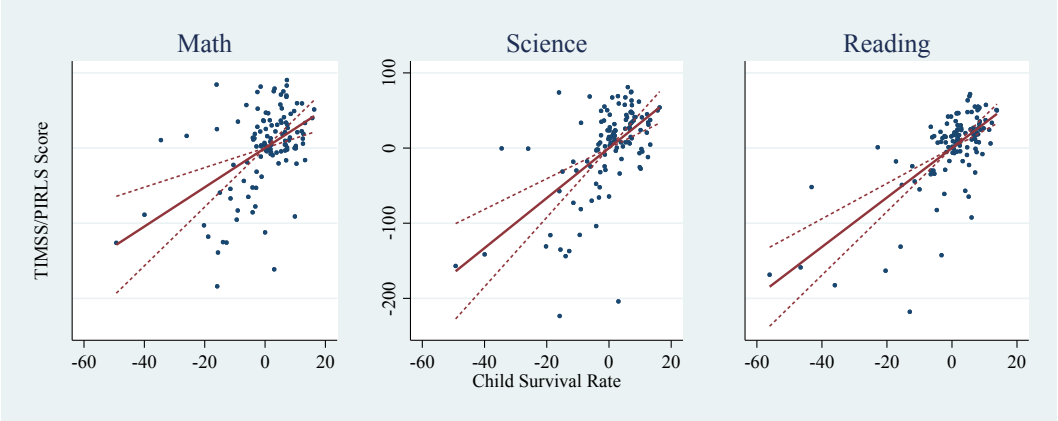

Figure 3: Child survival versus 4th grade TIMSS/PIRLS scores, controlling for income and education expenditure

5 Child survival and economic growth

Many aspects of economic development must be studied at an economy-wide scale to account for feedbacks and agglomeration processes. Microeconomic evidence about causes is inadequate to assess the magnitude of their impact on the economy. However, there is considerable skepticism about what can be learned from cross-country growth regressions. This is mainly due to three related problems. Empirical growth studies have frequently used a single cross-section of growth rates for each country regressed on initial values of variables thought to influence growth. This results in a very small sample size, with rarely more than a hundred countries reporting values for all the variables included. Second, many variables potentially affect economic growth, and most of them are correlated with each other. Constrained by a small sample, only a few variables can be included in a regression. It is difficult to be sure whether the choice of covariates is making the variable of interest statistically significant. Third, most variables affecting economic growth are likely endogenous to the process of development. In principle, this can be controlled by regressing subsequent economic growth on the initial values of explanatory variables, but correlated omitted variables can still bias the estimates. It is rare to find truly convincing instrumental variables to address bias from endogeneity or omitted variables since most variables measured at an economy-wide scale are causally related to economic growth.

The main variable of interest here, child survival, and the use of panel data makes it possible to address these statistical problems more directly than is usually the case. If the cognitive development of children determines the productivity of workers, economic growth should be affected not by contemporary measures of child health, but by child health conditions when current workers were young children. Measures of child health from decades ago are likely exogenous to the errors in current economic growth and are unlikely to erroneously capture correlations with other future variables, particularly after controlling for country effects, GDP per worker and other variables at the start of a period of economic growth.

Panel data on multiple periods of economic growth in each country increase the sample size substantially, making coefficient estimates more precise. Country fixed effects in the regressions greatly reduces the problem of omitted variables. Country-specific intercepts cover a multitude of sins, accounting for fixed or slowly changing country characteristics including geography, history, persistent aspects of culture, long-standing norms and the quality of institutions. Sala-I-Martin et al. (2004) found that only 18 of 67 potential correlates were robustly correlated with economic growth. Ten of the 18 variables were fixed characteristics of the countries (and most of the rest of the robust correlates are included in the regressions below). Panel data also makes it possible to include time period effects, which reduce the risk that the particular periods included in the sample affect the results.

I estimate the correlates of economic growth using the approach of Barro and Sala-i-Martin (1992). Define growth of income per worker y from time $t-1$ to time t as $\gamma \equiv \frac{1}{t}(\ln y_t - \ln y_{t-1})$. Taking a linear approximation of a neoclassical growth model near its equilibrium, economic growth is a combination of the source of sustained growth, the growth of technology, γ_A, and a transitory source of growth as the economy moves from its initial income level, y_{t-1} towards the equilibrium level of income for a given level of technology, y^*:

$$\gamma \approx \gamma_A + \theta \ln(y^*/y_{t-1}),$$

where θ is a constant parameter. Adding a country subscript i and a random error term u_{it}, the equation becomes

$$\gamma_{it} = \gamma_{Ai} + \theta \ln y_i^* - \theta \ln y_{i,t-1} + u_{it}.$$

Since technology growth, γ_{Ai}, and equilibrium income, y_i^*, cannot be observed directly, $\gamma_{Ai} + \theta \ln y_i^*$ is approximated by a linear combination of a vector of variables, $\mathbf{x}_{it}$, which are correlated with the growth of technology and equilibrium income. The estimating equation, with the addition of a country-specific

intercept μ_i and a time period indicator δ_t is

$$\gamma_{it} = -\theta \ln y_{i,t-1} + \mathbf{x}'_{it}\beta + \mu_i + \delta_t + u_{it}, \tag{1}$$

where θ, β, μ_i and δ_t are unknown parameters.

The combination of the country-specific intercept μ_i with the initial log income $\ln y_{i,t-1}$ as a regressor causes the initial income variable to be correlated with the error term. This correlation is seen most easily when taking deviations from the country-specific means to eliminate μ_i in Equation 1, as in Anderson and Hsiao (1981). To address the endogeneity of initial log income, I apply the Arellano and Bover (1995) and Blundell and Bond (1998) GMM estimator, which uses lagged values of exogenous or predetermined variables as instruments in a first differenced equation as well as using lagged differences as instruments in a level equation. The Arellano-Blover/Blundell-Bond estimator retains its statistical power when the initial log income variable has a high autocorrelation with future log income, which weakens the instruments in the Arellano and Bond (1991) estimator.[8]

I use an unbalanced panel of up to seven economic growth periods per country for each 5-year period from 1985-2019 for 105 countries. The exception

[8]The Arellano and Bond (1991) estimator, which is part of the Blundell and Bond (1998) system estimator used here, takes first differences of the estimation equation in order to to eliminate the fixed effect. This is typically presented in terms of an original equation in levels. Here I am estimating an equation where the dependent variable is a growth rate, which I think is easier to interpret. The coefficients on the regression with growth rates are identical to the coefficients on a levels equation except that the initial GDP coefficient is decreased by a constant, and the standard errors are identical for all coefficients. This is because the dependent variable is a function of the independent variables, and if the estimation equation is linear, adding a fixed fraction of one of the independent variables to a dependent variable just shifts the coefficient of the independent variable by that fraction. This can be seen in the simple case of OLS regressions of the first differences of a growth rate versus a level on a lagged dependent variable. The growth rate regression equation is $\Delta y_{it} = \theta y_{i,t-1} + \mu_i + \varepsilon_{it}$ and the level equation is $y_{it} = \beta y_{i,t-1} + \mu_i + \varepsilon_{it}$. The first differenced equations are $\Delta^2 y_{it} = \theta \Delta y_{i,t-1} + \Delta\varepsilon_{it}$ and $\Delta y_{it} = \beta \Delta y_{i,t-1} + \Delta\varepsilon_{it}$, respectively. Then $\hat{\theta} = \frac{\sum \Delta^2 y_{it} \Delta y_{i,t-1}}{\sum (\Delta y_{i,t-1})^2} = \frac{\sum \Delta y_{it} \Delta y_{i,t-1}}{\sum (\Delta y_{i,t-1})^2} - 1 = \hat{\beta} - 1$. The case of the Blundell-Bond estimator with additional independent variables can be shown in terms of the derivation in Footnote 9 below, but it is easier just to run an estimation in both levels and growth rates to see that the coefficients and standard errors are identical.

is the last episode from 2015 to 2019 which is four years long since the data end in 2019. Average income per worker is measured by the purchasing power parity real gross domestic product (GDP) per worker from the Penn World Tables (PWT), version 10.0 (Feenstra et al., 2015). The average annual growth rate within each period is the country-specific least squares linear time trend of the natural log of income per worker using the PWT variable GDP^{NA}, recommended for measuring growth rates. Using trend growth rates over a multiyear period reduces the influence of measurement error and idiosyncrasies of starting and ending years of the episodes. The log of initial GDP per worker uses the variable GDP^{O} which PWT recommends for income levels.

In addition to initial log GDP per worker, the independent variables include measures of institutional quality, trade openness and education levels. Institutional quality is measured by the Political Risk Index from the International Country Risk Guide (PRS Group, 2012), an annual evaluation of the quality and stability of governments and policy. Trade openness is the sum of imports and exports of goods and non-financial services as a percentage of GDP, from the World Development Indicators (World Bank, 2021). This variable is intended to measure the integration of the economy in world markets. Education levels are the average years of education of people aged fifteen and older from Barro and Lee (2013), a measure of the human capital of workers.

The influence of health on economic growth has most commonly been explored using life expectancy as a proxy for general health conditions. As a point of comparison with child survival, I first estimate the correlation of life expectancy (United Nations Population Division, 2019) with economic growth, controlling for a commonly included set of other correlates (column 1 of Table 2).

As is usually found, national economic growth is significantly negatively correlated with initial income levels and positively correlated with a large share of imports and exports in output, and higher life expectancy. Although education was often not strongly correlated with economic growth in the earlier cross-sectional growth literature, which usually covered the period 1960 to 1980 or 1990 (Pritchett, 2001), its coefficient is statistically significant in this

Table 2: Correlates of growth in GDP per worker

	(1) Life expectancy	(2) Current CSR	(3) Lagged CSR	(4) Low vs. High Income
Initial log GDP per worker	-4.015	-3.448	-3.642	-3.566
	[0.825]***	[0.434]***	[0.662]***	[0.798]***
Institutional quality (0-100)	0.045	0.031	0.032	0.028
	[0.024]	[0.018]	[0.019]	[0.019]
Trade (% of GDP)	0.006	0.006	0.005	0.005
	[0.002]**	[0.002]*	[0.002]**	[0.002]**
Schooling (years)	0.576	0.346	0.215	0.222
	[0.192]**	[0.139]*	[0.137]	[0.150]
Life expectancy (years)	0.164			
	[0.057]**			
Adult survival (per 1000)		0.002	0.008	0.007
		[0.003]	[0.003]*	[0.003]*
Child survival (per 1000)		0.038		
		[0.012]**		
Child survival, previous generation			0.022	
			[0.006]***	
Child survival, gen[-1], low income				0.022
				[0.006]***
Child survival, gen[-1], high income				0.022
				[0.006]***
Constant	22.968	-7.317	8.248	8.409
	[5.392]***	[9.587]	[4.287]	[5.728]
Year effects	yes	yes	yes	yes
N	743	743	743	743
Countries	117	117	117	117
$R^2(Corr(\hat{y}, y)^2)$	0.45	0.46	0.46	0.46
ABond z test for AR(1)	-2.63**	-2.63**	-2.62**	-2.62**
ABond z test for AR(2)	1.01	0.98	1.04	1.04
Hansen overid χ^2 test	84.17	109.55	82.71	82.15
% Growth from 1 s.d. Δ CSR		1.89	1.76	
% Growth from 1 s.d. Δ ASR		0.25	0.86	
% Growth from 1 s.d. Δ CSR L.I.				1.63
% Growth from 1 s.d. Δ CSR H.I.				0.94

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

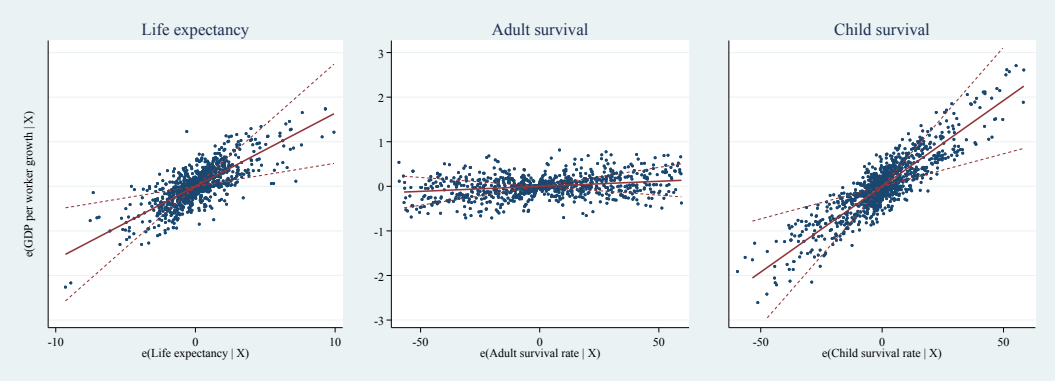

Figure 4: Survival versus economic growth, controlling for other variables

regression. The correlation of the Political Risk Index of institutional quality with economic growth is small and statistically insignificant. Since institutions are likely to change slowly, the lack of significance for institutional quality is probably partly due to the inclusion of country-specific intercepts which absorb the unchanging country characteristics. Due to the fixed effects, all the coefficients represent the partial correlation of changes of the independent variables with changes of economic growth rates. The lack of significance of the Political Risk Index does suggest that improvement in institutions was not a significant contributor to economic growth.

The Arellano and Bond (1991) test for autocorrelation of the error term finds first-order but not second order autocorrelation. For this reason, the instruments for initial income levels were reduced from second order and further lags to third order and further lags (Roodman, 2009). To avoid bias from potential endogeneity of life expectancy, it is also instrumented with its own lagged values and lagged values of the predetermined variables institutional quality, trade, schooling and the year indicators. The Hansen (1982) test for overidentification does not reject the validity of the instrument sets.

Life expectancy is a synthetic measure calculated from a cross-section of contemporary age-specific survival rates. The estimation in column 2 of Table 2 replaces life expectancy with the adult survival for the working ages of 15 to 60 and child survival for ages 0 to 5 (United Nations Population Division,

2019). Adult survival and child survival are instrumented to void possible endogeneity. The adult survival coefficient is very small and statistically insignificant. The child survival coefficient is large and highly statistically significant. The partial correlations of life expectancy with economic growth in column 1, and adult survival and child survival with economic growth in column 2, controlling for the other correlates, are shown as added variable plots in Figure 4.[9,10]

Essentially all of the correlation of life expectancy with economic growth is due to child survival, not adult survival. The coefficients on the other variables in the first two regressions in Table 2 are similar to each other. The hypothesis that life expectancy is correlated with growth because it measures the health of workers looks suspect. It is also clear, though, that contemporaneous child health cannot be a direct cause of economic growth because young children do not participate in the economy. If child survival is an indicator of favorable conditions for child cognitive development which in turn has a causal impact on economic growth, the relevant childhood conditions of current workers are health conditions from decades ago when the workers were children.

Since United Nations child mortality estimates extend back only to 1950, I use the average child survival rates from 15 to 35 years before as an indicator of the child cognitive development conditions for current workers. This leaves out the child conditions of workers older that 40 years, but younger workers

[9]Added-variable plots display partial correlations by graphing the residuals of an independent and the dependent variable each regressed on all other independent variables (Gallup, 2020). In Figure 4, these are Blundell and Bond (1998) GMM residuals. The estimated coefficients can be written, with some manipulation, as $\hat{\beta}_{GMM} = (\mathbf{X}^{*\prime}\mathbf{X}^{*})^{-1}\mathbf{X}^{*\prime}\mathbf{y}^{*}$ where $\mathbf{X}^{*} = \hat{\Omega}^{1/2}\mathbf{Z}(\mathbf{Z}'\hat{\Omega}\mathbf{Z})^{-1}\mathbf{Z}'\mathbf{X}$, $\mathbf{y}^{*} = \hat{\Omega}^{1/2}\mathbf{Z}(\mathbf{Z}'\hat{\Omega}\mathbf{Z})^{-1}\mathbf{Z}'\mathbf{y}$, $\hat{\Omega}$ is the estimated variance-covariance matrix of the error terms, and $\mathbf{Z}$ are lagged values of $\mathbf{X}$ variables used as instruments for $\mathbf{X}$. Using the Frisch-Waugh-Lovell theorem (Lovell, 1963), the estimated coefficient for a particular $\mathbf{X}$ variable, say $\mathbf{x}_1$, is $\hat{\beta}_1 = (\mathbf{e}'_{\mathbf{x}_1^*}\mathbf{e}_{\mathbf{x}_1^*})^{-1}\mathbf{e}'_{\mathbf{x}_1^*}\mathbf{e}_{\mathbf{y}^*}$, where $\mathbf{e}_{\mathbf{x}_1^*}$ is the residual from the regression of $\mathbf{x}_1^*$ on $\mathbf{X}_2^*$, $\mathbf{e}_{\mathbf{y}^*}$ is the residual from the regression of $\mathbf{y}^*$ on $\mathbf{X}_2^*$ and $\mathbf{X}^* = [\mathbf{x}_1^*\mathbf{X}_2^*]$. The added-variable plot is a scatterplot of $\mathbf{e}_{\mathbf{x}_1^*}$ versus $\mathbf{e}_{\mathbf{y}^*}$ and a line graph of the OLS regression fit of these values, which has slope $\hat{\beta}_1$. The estimate $\hat{\beta}_1$ is statistically different from zero if the dashed 95% confidence interval excludes $\mathbf{e}_{\mathbf{y}^*} = 0$ for $\mathbf{e}_{\mathbf{x}^*} \neq 0$.

[10]In Figure 4, the displayed range of the adult survival rate residuals has been limited to the range of child survival rate residuals to represent the relative slopes of the two rates.

may be more apt to introduce new technology and use their cognitive skills more intensively than older workers.[11] In column 3 of Table 2 the child survival rates from 15 to 35 years earlier are labeled "previous generation". As in the previous regression, adult survival is instrumented to avoid potential endogeneity. Child survival for 15 and more years ago is not, because it is almost certainly predetermined. However the estimates are almost unaffected by instrumenting lagged child survival, or not instrumenting for either adult or child survival. The estimates are also not affected substantially by using World Bank GDP data instead of Penn World Tables GDP data. These variations are not shown, but available upon request.

The central empirical finding of this paper is that a proxy for child cognitive development conditions from decades earlier has a large, statistically significant correlation with economic growth, controlling for other likely determinants. A one standard deviation higher level of child survival predicts a 1.76% higher rate of economic growth per year. It is difficult to think of a non-causal explanation of this large correlation. What omitted variable would be both correlated with child survival from decades ago and also cause economic growth in the present, that would not accounted for either by the country fixed effect, the level of initial income or the quality of institutions?

Figure 5 shows the country observations of the partial correlation between child survival and economic growth in the first growth episode 1985-1990, with other time observations displayed in gray and a trendline of the full panel estimation. The points are the values of child survival 1950 to 1970 and economic growth in 1985-1990, after accounting for their correlation with the other explanatory variables. Low and middle income countries which closely adhere to the estimated correlation with low child survival and low growth include Peru, Jordan, Bolivia, Gabon and Bangladesh. Lower income countries with higher than expected child survival and growth include Cameroon, Egypt, Morrocco and Pakistan. Among high income countries, those with weaker child survival and growth include Australia, New Zealand and the United States,

[11] Aghion et al. (2010, Table 4) finds that life expectancy at 40 is uncorrelated with economic growth, once life expectancy at birth is accounted for.

Figure 5: Economic growth 1985-90 versus child survival
(other years in gray)

and stronger child survival and growth include France, Japan, Panama and Italy.

When lagged child survival is included, adult survival becomes statistically significant. A one standard deviation higher level of adult survival predicts a 0.86% higher growth rate, also substantial. However, improving adult survival for ages 15 to 60 requires improving health over a span of 45 years whereas improving child survival requires improving health for just five years.

The coefficient on schooling in Table 2, column 3 is smaller than in the previous estimations and loses statistical significance. This is due to the high correlation of schooling with lagged child survival. Table 3 shows that schooling is the variable most correlated with lagged child survival (0.87), even higher than child survival's correlation with life expectancy (0.85) and adult survival (0.67). This shift of estimated coefficients is likely causal. Child cognitive development is a crucial input into school attainment. Microeconomic research showing that child development is a predictor both of school success and adult health suggests that the impact of child survival conditions could be an under-

Table 3: Summary statistics and correlations with child survival

Variables	Mean	Standard deviation	Correlation w/CSR_{-1}
Child survival, 15-35 yrs before (per 1000)	908.47	80.85	1.00
Adult survival (per 1000)	823.54	111.77	0.67
Life expectancy (years)	70.32	9.29	0.85
GDP per worker growth (%)	1.46	2.95	0.04
log of GDP per worker (ln($))	10.17	1.05	0.80
Institutional quality (0-100)	65.66	14.71	0.67
Trade (% of GDP)	79.80	55.93	0.33
Schooling (years)	7.79	2.86	0.87
N	743		

estimate. The estimated coefficient for lagged child survival increases by 30% when the schooling variable is excluded from the estimation (not shown), although other inputs like spending on schools likely affect education outcomes.

The last estimation in Table 2 calculates separate estimates for child survival in low and high income countries. The low income countries are those classified as either "Low Income" and "Lower Middle Income" at the start of each five year period by the World Bank (World Bank, 2022). The high income countries are those classified as either "Upper Middle Income" or "High Income".

Surprisingly, lagged child survival rates have virtually the same correlation with economic growth whether in high or low income countries, and the same significance levels.

High income countries have less room for improvement in lagged child survival (mean of 957/1000), so the predicted growth in GDP per worker from a one standard deviation improvement (43/1000) is 0.94%. Low income countries start with lower mean child survival rate (847/1000) with a larger standard deviation (75/1000). The predicted growth of a one standard deviation improvement is higher at 1.63%. On the other hand, high income

countries have had lower economic growth in the sample (1.16%) than low income countries (1.83%), so the predicted growth increments are more similar as a fraction of average growth in the two groups: 0.94%/1.16% = 81% for high income countries versus 1.63%/1.83% = 89% for low income countries.

Additional growth estimation results shown in Tables 4 and 5 explore the robustness of the correlation of past child survival to alternative estimation methods and alternative measures of institutional quality. Complex estimation methods like Blundell and Bond (1998) are likely to be fragile when misspecified. Durlauf et al. (2005) point out that moving from OLS to fixed effects to GMM estimators represents a trade-off where more and more information is discarded in pursuit of less potential endogeneity bias. The OLS estimation uses cross-country variation to identify the parameters. Fixed-effects estimation discards this variation, identifying parameters only from within-country variation over time. Since a lagged dependent variable (initial income in our case) is correlated with the error in a fixed effects specification, the GMM estimators like Arellano-Bond and Blundell-Bond instrument the lagged dependent variable with lagged values, reducing potential bias but increasing the influence of measurement error.

For all five estimation methods in Table 4, the child survival rates from a previous generation are highly statistically significant, which is not consistently true of trade openness, schooling and adult survival rates. The child survival coefficients are also large, though they vary in size. Bond et al. (2001) explain that a lagged dependent variable coefficient in a panel data model will be downward biased when estimated by OLS and upward biased when estimated by fixed effects, while GMM estimates should be consistent. This pattern of relative size of the coefficients on initial income and child survival holds in Table 4, where the OLS estimates are the smallest, the fixed effects estimates accounting for autocorrelation are the largest and the two GMM estimates (Arellano-Bond and Blundell-Bond) are in between. This pattern suggests the size of the GMM estimates are plausible relative to the estimates from the other simpler, but likely biased, estimation methods.

Skeptics of a causal impact of health on economic growth often point to

Table 4: Various estimation specifications

	OLS	Fixed Effects	Fixed Effects AR(1)	Arellano Bond	Blundell Bond
Initial log GDP per worker	-1.811	-5.135	-4.956	-3.642	-3.642
	[0.190]***	[0.720]***	[0.496]***	[0.662]***	[0.662]***
Institutional quality (0-100)	0.014	-0.000	-0.037	0.032	0.032
	[0.011]	[0.022]	[0.017]*	[0.019]	[0.019]
Trade (% of GDP)	0.004	-0.001	-0.003	0.005	0.005
	[0.002]	[0.007]	[0.006]	[0.002]**	[0.002]**
Schooling (years)	0.268	-0.097	0.202	0.215	0.215
	[0.076]***	[0.218]	[0.178]	[0.137]	[0.137]
Adult survival (per 1000)	0.004	0.007	0.005	0.008	0.008
	[0.001]**	[0.003]*	[0.003]	[0.003]*	[0.003]*
Child survival, previous generation	0.006	0.020	0.028	0.022	0.022
	[0.003]*	[0.006]**	[0.006]***	[0.006]***	[0.006]***
Constant	7.396	29.099	23.820	8.248	8.248
	[1.918]***	[8.889]**	[5.066]***	[4.287]	[4.287]
Year effects	yes	yes	no	yes	yes
N	743	743	626	743	743
Countries	117	117	117	117	117
$R^2(Corr(\hat{y}, y)^2)$	0.14	0.48	0.45	0.46	0.46
% Growth from 1 s.d. Δ CSR	0.47	1.61	2.26	1.76	1.76
% Growth from 1 s.d. Δ Trade	0.20	-0.03	-0.16	0.30	0.30

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

Table 5: Institutional quality variables

Institutional index	Institutional coefficient	Child survival coefficient
Political Risk Index	0.029	0.019**
Government Stability	0.015	0.018**
Socioeconomic Conditions	0.001	0.020**
Investment Profile	0.003	0.020**
Internal Conflict	0.026*	0.019**
External Conflict	0.003	0.021**
Corruption	0.017	0.020**
Military in Politics	0.011	0.021**
Religious Tensions	-0.013	0.021**
Law and Order	0.027*	0.018**
Ethnic Tensions	-0.001	0.021**
Democratic Accountability	-0.002	0.021**
Bureaucracy Quality	0.020*	0.022**

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

effective institutions as the cause of both good health and successful economic growth. For instance Acemoglu et al. (2001) argue that rather than malaria influencing contemporary economic growth, it inhibited European settlement in colonies centuries ago, causing malarial countries to have less effective institutions, which currently limits economic growth. The estimates presented above do include one index of institutional quality, but it can be difficult to measure institutions (or even to define them). The Political Risk Index used in the previous regressions is a weighted average of 12 subindices of different institutional outcomes. Table 5 shows the estimated coefficients on each of these institutional subindices from regressions otherwise specified as in column 3 of Table 2 (Lagged CSR), substituting a different institutional variable each time, as well as the corresponding coefficient on the lagged child survival rate.

The child survival coefficients fall within a narrow range around the preferred estimate of 0.022, even when some of the institutional indices turn out to

be statistically significant. For this set of institutional quality indices, there is little sign that the child survival estimates are suffering from omitted variable bias.

In addition to the strong correlation of economic growth with a proxy for cognitive risks, Appendix B argues that the role of cognitive development helps explain the mismatch between microeconomic and macroeconomic estimates of the impact of both worker health and education on productivity.

If child cognitive development plays a causal role in economic growth, it would be useful to know the relative importance of different risk factors of child development. Appendix C shows that variables for anemia, clean water and sanitation access, vaccinations and undernutrition account for 97% of the improvements in child survival, with anemia and access to water and sanitation each accounting for a third of the improvements.

6 Conclusion

Even modest levels of risk factors significantly affect the cognitive development of children (Walker et al., 2007) and have surprisingly large impacts on later economic, education, and health outcomes of individuals (Almond et al., 2018). Researchers find substantial impacts in both high and low income countries.

The high levels of risk factors around the world raise the possibility that cognitive development conditions could have large impacts on economic growth. In addition to direct consequences for individuals, the level of cognitive development may have spillovers, in education from teacher to student and peer to peer, and in the workplace by creating the conditions for technology adoption and innovation.

Human capital is central to economists' conception of economic production. Cognitive development establishes a human capital base which develops further in school. Cognitive development also improves the *rate* of learning. For these reasons, better cognitive development can raise the long-run rate of economic growth.

The debate about the role of health in economic growth in the empirical

literature has focused on the correlation of life expectancy with growth. When life expectancy is decomposed into child survival and adult survival, the strong correlation of life expectancy with economic growth is almost entirely due to child survival rates, not adult survival.

The child survival rate is a plausible proxy for the good cognitive development of children because each risk factor for cognitive deficiency is also a risk factor for mortality. Child survival rates from a generation or more ago reflect the conditions for child development of current workers. Child survival rates a generation ago have a large correlation with national economic growth in panel estimations controlling for country fixed effects and other likely correlates. The correlation of child survival with economic growth is robust to a variety of estimation methods and correlates.

Child survival is a stronger correlate of economic growth than trade openness or improved institutional quality. The estimated impact of child survival is just as large in high-income as in low-income countries.

Many economists are skeptical of the cross-country economic growth literature which was focused on data from about one hundred countries. The main concerns are the small sample size, possible endogeneity of explanatory variables, and omitted variables, particularly those reflecting the history, culture, institutions and geography in each country.

This study is less subject to these concerns than the earlier cross-country growth studies. A 35-year panel of growth rates provides a respectable sample size of more than seven hundred. Individual country fixed effects account for the impacts of history, culture and geography which do not change during the study period, and the study includes numerous measures of institutional quality. The main variable of interest, early childhood health conditions from a generation earlier, is predetermined and plausibly non-endogenous relative to current economic growth.

What omitted variable would be both correlated with childhood survival decades in the past and cause economic growth in the present which is not accounted for by country effects, the current level of income and other included variables? Correlations are imperfect measures of causality, but large corre-

lations need a plausible alternative hypothesis to be ignored. The impact of early cognitive development on individual economic outcomes is better supported by rigorous microeconomic evidence than almost any other commonly-hypothesized cause of general economic growth.

Most of what we know about contributors to economic growth come from country comparisons. "Natural experiment" strategies which identify impacts from sudden changes in child health are attractive, but are only applicable to specific risk factors. Studies of early life exposure to helminthic worms (Bleakley, 2007), malaria (Malpede, 2022; Kuecken et al., 2021; Venkataramani, 2012; Bleakley, 2010; Cutler et al., 2010), famine (Almond et al., 2010) and influenza pandemic (Almond & Mazumder, 2005) show significant impacts on individual economic outcomes at a regional or national level, but they are unable to measure the impact of all risk factors nor spillovers of good cognitive development on the productivity of others.

The role of cognitive development helps resolve the mismatch between microeconomic and macroeconomic estimates of the impact of health and education on economic outcomes. Not accounting for cognitive development inflates the estimated impact of worker health on economic growth and deflates the estimated impact of education on economic growth.

Anemia accounts for one third of differences in child survival according to a simple decomposition of risk factors. Anemia together with safe water and sanitation accounts for 72% of the differences. Improving maternal and child health has long been a public health goal, and there is a large and mostly optimistic literature about the potential of interventions and policies (see Britto et al., 2017; for a survey of research on the impact of interventions and Richter et al., 2017, for a survey of early childhood development policy). In most of the world, though, child health is a lower priority than adult health.

A broader view of economic development which values equity and human capabilities in addition to living standards places a greater priority on good cognitive development. Poor cognitive development is perhaps the most fundamental unequal opportunity. Unlucky children find their ability to learn and work compromised before they start school. Good cognitive development

provides a fair chance in life for everyone. A large impact of child health on economic growth means that improving child health now will generate the resources to provide a better life for the future generations.

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For Online Publication

Appendix A Economic growth with government provision of health care

The model in the main paper showed that cognitive development affects schooling and economic growth, but it didn't specify the source of cognitive development. This section models the impact of government health spending on the economy through its effect on cognitive development.

Most health spending around the world is by governments (on average 60% in 2017 according to World Bank, 2021). Much of the private spending is only possible due to a government-created network of health facilities, medical training and insurance mandates.

Prenatal care for pregnant women, which is consequential for fetal cognitive development, depends in the first instance on its availability. Undernutrition of young children, a contributor to poor cognitive development, is mostly a reflection of lack of choice. Parents are likely unable to assess whether their children are deficient in crucial micronutrients like iron, iodine and folate without access to blood tests. A strong indication that parents are not making informed decisions in this area is that rates of iron deficiency are very high although adequate iron can be obtained at very low cost. Options include eating iron-rich greens or beans, cooking with iron pots or placing a piece of rusty metal in a water tank (Werner et al., 1992, p. 117). Iodine and folate can also be obtained at low cost from certain natural or fortified foods. On the other hand, governments can provide iron, iodine and folate for the whole population by fortifying foods like salt, rice, or flour at extremely low cost (Ramírez-Luzuriaga et al., 2018; Dietrich et al., 2005). Exposure to important risk factors like contagious disease, clean water, sanitation, pollution, communal violence or war largely depend on government action or inaction.

Define f as the proportion of time a person is healthy. f is the proportion of healthy time that can be devoted to early cognitive development, h_0,

human capital development in school, h, or working time, producing income y. Assume that $f(g)$ is an increasing, concave function of government health spending g, where $f(g) > 0 \quad \forall \quad g > 0$ and $f'(g) \equiv \frac{\partial f(g_0)}{\partial g_0} > 0$. The level of annual health spending on young children during the fetal and early childhood period is g_0, and health spending on children in school and adults is g. Since the period of cognitive development after which many deficiencies are irreversible starts at conception and ends at about age two, take the period during which initial human capital h_0 develops to be about 3 years, so that cognitive potential at conception is h_{-3}, normalized to equal 1.

If $f = 1$, a person realizes a full year of early development, learning in school, or productive work. When $f < 1$, they are only healthy enough for productive activities for the fraction f of a year. Initial human capital h_0, completed human capital h and income y are then

$$\begin{aligned} h_0 &= f(g_0)h_{-3} = f(g_0) \\ h &= h_0 e^{\eta h_0 f(g) s} \\ y &= A[f(g)h]^{\alpha} \end{aligned}$$

Technology is a function of the average human capital of workers while they are healthy,

$$A = A_w[f(g)h]^{\beta}.$$

Substituting,

$$y = A_w\Big[f(g)f(g_0)e^{\eta f(g_0)f(g)s}\Big]^{\alpha+\beta}.$$

Keeping people healthy during working years (the first $f(g)$) and during gestation and infancy (the first $f(g_0)$) each have the same impact on income (ignoring for the moment the impact of g_0 and g on learning in $e^{\eta f(g_0)f(g)s}$). The symmetry of $f(g)$ and $f(g_0)$ means that keeping people healthy during working years or during infancy are equally economically beneficial. However, they have very different lengths and very different costs. Infancy is short, about three years, and working years is longer, say 48 years to make the ratios simple. If the level of healthcare expenditure per person g were the same for

adults and infants,[12] the total cost to the government of providing healthcare to workers is $48g$, twelves times the lifetime spending on infant healthcare ($3g$).

This is the main intuition of the model. Keeping infants healthy from inception to age two is equally economically productive as keeping adults healthy throughout their working years. Keeping an infant healthy is vastly cheaper because infancy is a much shorter time period, so the government should be spending more on the health of an infant than an adult. Accounting for the impact of infant and student health spending on the rate of learning provides an additional impetus to prioritize infant health, since infancy is typically shorter than years in school.[13]

An optimizing government would invest substantially *more* per person for maternal and early childhood health than for working adults because it is much more economically productive per dollar spent. Exactly how much more should be spent per year on infants depends on the concavity of the health function $f(\cdot)$.

To show this formally, the government will maximize the lifetime utility of the representative person by choosing health spending for each age group, g_0 for infants and g for school children and workers, conditional on the parents' choice of education s.

Parents choose schooling s to maximize their child's lifetime utility subject to the budget constraint:

$$\max_s u = T\frac{c^\sigma}{\sigma} \quad s.t. \;\; Tc = (1-\tau)(T-s)y$$

where c is consumption, $(1-\tau)(T-s)y$ is post-tax lifetime income and $0 <$

[12] In the U.S. the annual cost of healthcare for working adults is, if anything, higher than the cost for children. The cost for infants 0-3 years old 79% of the cost of adults aged 19-64 and the cost of school-age children aged 4-18 only 35% of adults (Table 3 of Herrera & Nelson, 2012)

[13] By the same logic, governments should spend more on school children than adults for economic reasons, since school years are fewer than work years, but for simplicity, in this model the government spends the same amount g on both students and workers each year.

$\tau < 1$ is the tax rate. The parents' choice of schooling is

$$s = T - \tfrac{1}{\alpha\eta h_0}.$$

Note that parents choose more schooling when the government spends more on the health of infants since $h_0 = f(g_0)$, making people more productive their whole lives. The parents' choice of schooling is unaffected by health spending for school age children and workers, g:

$$\frac{\partial s}{\partial g_0} = (T-s)\frac{f'(g_0)}{f(g_0)} > 0; \qquad \frac{\partial s}{\partial g} = 0.$$

The government maximizes the utility of the representative person subject to the individual's post-tax lifetime budget constraint and the government's budget constraint. Health spending, $3g_0 + Tg$, must equal tax revenues $\tau(T - s)y$ from a tax rate τ:

$$\begin{aligned} \max_{g_0,g} u &= T\frac{c^\sigma}{\sigma} \\ s.t.\ \ Tc &= (1-\tau)(T-s)y \\ \tau(T-s)y &= 3g_0 + Tg \end{aligned}$$

The maximization provides a relationship between health spending g and g_0:

$$\frac{f'(g)}{f(g)} = \frac{T}{3}B\frac{f'(g_0)}{f(g_0)}$$

where

$$B \equiv 1 + \frac{\frac{f(g)}{\alpha} - \frac{1}{\alpha+\beta}}{1 + \eta h_0 s f(g)}.$$

B reflects the complicated tradeoffs from better cognitive development improving the rate of learning, causing children to stay in school longer, which raises their productivity and income, but also reduces their years working. The magnitude of B depends on the value of its parameters, but for plausible

real world values, $B \approx 1$.[14]

$f(\cdot)$ is an increasing function, so $f'(g) > 0$. $\frac{f'(g)}{f(g)} \approx \frac{T}{3}\frac{f'(g_0)}{f(g_0)}$ means that optimal government spending on maternal and infant health, g_0, will be substantially higher than spending on student and worker health, g:

$$g_0 > g$$

If the health spending function $f(g)$ is linear, school (including preschool) and working years after age 2 is $T = 60$ years, and gestation and infancy is 3 years, then

$$\frac{f'(g)}{f(g)} = \frac{1}{g} \text{ and } g_0 = 20g$$

so the government should optimally spend 20 dollars on early child health for every dollar spent on adult and school age health.

In reality, $f(g)$ is almost certainly concave due to diminishing returns to health spending, so the differences in optimal health spending across age groups would be less dramatic. The effects of health on economic productivity would still mean that substantially more should be spent on maternal and early childhood health per person than on working adults.

[14] $B \approx 1$ if $\frac{f(g)}{\alpha} \approx \frac{1}{\alpha+\beta}$ or if $\frac{f(g)}{\alpha} - \frac{1}{\alpha+\beta}$ is small relative to $1 + \eta h_0 s f(g)$. $\frac{f(g)}{\alpha} = \frac{1}{\alpha+\beta}$ if $f(g)\frac{\alpha+\beta}{\alpha} = 1$. Since we are denominating g as annual per person health expenditures, $1 - f(g)$ is the average annual work lost due to poor health. Empirical estimates for these losses are low in percentage terms, at less than 1% for Great Britain (less than 2 work days lost per 252 working days per year, HSE, 2022), 1.1% in Japan (Chimed-Ochir et al., 2019, p. 682), and 4.5% in a high-disease area of rural Nigeria (8.2 days lost during a 6 month growing season Unekwu, 2014, p. 8489). So it is likely that $f(g) > 0.95$. If β, the impact of average human capital on the level of technology adoption is at least 5% of α, the labor share of income going to workers in a competitive economy, then $B \geq 1$.

If alternatively $\beta = 0$ and $f(g)$ is still 0.95, then $\frac{f(g)}{\alpha} - \frac{1}{\alpha+\beta}$ is still likely to be small relative to $1+\eta h_0 s f(g)$. As in the previous section, the private return to education is still $R_p = \alpha\eta h_0$, so $1 + \eta h_0 s f(g) = 1 + \frac{R_p s f(g)}{\alpha}$. Most estimates for private returns to education R_p are at least 7% with higher rates in lower income countries (Card, 1999; Psacharopoulos, 1994). Average years of schooling across countries is 9 years in 2015 (Barro & Lee, 2013) plus 3 years of preschool would make $s = 12$. Karabarbounis and Neiman (2014) estimate α, the labor share, at 0.52 or higher. With these parameters, $B = 1+\frac{f(g)-1}{\alpha+R_p s f(g)} = 1+\frac{0.95-1}{0.52+0.07\times12\times0.95} = .96$, which is still reasonably close to 1 (relative to $\frac{T}{3}$).

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Appendix B The Paradox of Health, Education and Economic Growth

The strength of the correlation of life expectancy with economic growth and weakness of the correlation of education with economic growth is puzzling given the opposite pattern in microeconomic studes, summarized in Table 6. On the one hand, for individual workers, education has consistently large correlations with earnings, while health usually has small correlations with earnings. On the other hand, at a national scale, education is inconsistently correlated with economic growth, while health measures are strongly correlated with economic growth. The impact of cognitive development of children can resolve this paradox because risk factors for cognitive development are components of many measures of health as well as drivers of educational attainment.

Table 6: Correlations with income/growth

Variable	Macroeconomic	Microeconomic
Health	large	small
Education	small	large

Card (1999) surveys estimates of the causal impact of education in high income countries on earnings at the individual level. His conclusion implies a rate of return of an additional year of education in the range of 9%, making it an excellent investment.[15] Psacharopoulos (1994) finds that the rate of return to education for individuals in low and middle income countries is usually higher than in high income countries.

At a national level, Pritchett (2001) called attention to the small and weak correlation between education levels and economic growth, when controlling

[15]Assessing the myriad potential sources of bias in estimating the impact of education, Card (1999, p. 1802) concludes that OLS estimates are likely overestimated by about 10%. OLS estimates of the rate of return to education are often about 10% (as in Table 1 of Card's paper), implying an unbiased estimate of about 9%.

for other factors. The frequent lack of statistical significance for the education variable in Table 2 and Table 4 is typical of economic growth regressions.

Health follows the opposite pattern. Estimating the causal impact of health on individual earnings is even more daunting than for education due to problems of simultaneity and measurement of health. In high income countries, the main evidence for any impact of health on employment is that older workers with identifiable health limitations tend to work less, but not necessarily for lower wages (Currie & Madrian, 1999). In developing countries, the main finding is that earnings are correlated with height (Thomas & Strauss, 1997; Schultz, 2002). Height, though, is a consequence of fetal and child health, not adult illness, and may principally affect earnings via cognitive skills. Pitt and Rosenzweig (1986) showed that farm profits in Indonesia were unaffected by farmer illness when the farmers were able to hire replacement labor.

At the national level, life expectancy, interpreted as a proxy for health conditions, is one of the strongest correlates of economic growth. Of thirteen studies reviewed in Bloom et al. (2003, Table 1), twelve found a significant correlation between life expectancy and economic growth, ranging from 2-7% higher economic growth per additional year of life. More recent studies by Lorentzen et al. (2008), Aghion et al. (2010), Cervellati and Sunde (2011), and Bloom et al. (2019) find similar results. Unlike most other variables used in economic growth regressions, life expectancy is robustly correlated when a wide range of other covariates are included in regressions (Sala-I-Martin et al., 2004; Rockey & Temple, 2016; Bruns & Ioannidis, 2020).

The impact of cognitive development on worker productivity can help resolve the micro-macro paradox for both education and health. The quality of education in a country is not well measured by the average number of years studied, as shown by the wide range of skills at a specific school grade in Figure 3. Hanushek and Kimko (2000) show that the absence of a measure of education quality can explain the typically modest correlation of average years of education with economic growth, despite the high microeconomic rate of return within countries. Child survival or life expectancy as indicators of cognitive development are also proxies for educational quality. Cognitive

development likely raises the quality of education since children learn more during each year of school and teachers are more capable. This is shown by the high correlation of child survival with test scores in Figure 3. Including child survival or life expectancy in an economic growth regression lowers the coefficient on years of schooling, with which they are correlated. When the child survival rate is dropped from the regression in column 3 of Table 2, for example, the schooling years coefficient doubles and becomes significant at the 0.1% level (not shown). Child survival or life expectancy in a growth regression are partly measures how much has been learned in school and partly a measure of abilities not learned in the classroom.

Accounting for cognitive development can also reconcile the mismatch between micro and macro estimates of the impact of health. Life expectancy is a composite of both child survival and adult survival, making its high correlation with economic growth an overestimate of the impact of adult health alone if the cognitive skills of workers play an important productive role. When child and adult survival are disaggregated as in Table 2 above, the coefficient on adult survival is more consistent with the modest but still significant estimates of Weil (2007), which are calibrated from microeconomic estimates.[16] Using adult survival in place of life expectancy, without child survival, is also problematic because adult survival is correlated with omitted child survival. Distinguishing adult survival from child survival provides more credible estimates of the direct impact of worker health on economic growth, which are more consistent with the small microeconomic estimates.

Cognitive development has implications for the influential finding of Acemoglu and Johnson (2007) that changes in life expectancy since 1940 are *negatively* correlated with economic growth across countries. If child health conditions affecting cognitive development are important, health improvements before 1940, as well as after 1940, should affect economic growth after 1940. Indeed, Bloom et al. (2014) finds that including the level of life expectancy in

[16]Microeconomic estimates of the impact of individual health on earnings by construction ignore any spillovers of health between workers, such as productivity spillovers or risk of contagion, so are underestimates of the value to the economy of worker health.

1940 reverses the negative correlation of change of life expectancy with growth. Acemoglu and Johnson focus on the impact of big medical breakthroughs after 1940. However, their data show that life expectancy increased more between 1900 and 1940 than the increase they study from 1940 to 1980 (Acemoglu & Johnson, 2007, Table 1, Column 1). The main health breakthrough of the earlier period was the provision of sanitation and clean water (Cutler et al., 2006), which was implemented to some degree even in developing countries (Hungerford & Smiley, 2016; Kooy & Bakker, 2015). The influence of conditions as of 1940 on the results in Acemoglu and Johnson raises the possibility that the study period happened to be a time of catch up for late health adopters, who nonetheless had lower economic growth because post-1940 improvements in child health had not yet raised the cognitive skills of workers.

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Appendix C Proximate determinants of child survival

If child cognitive development plays a substantive role in economic growth, it would be useful to know the relative importance of different risk factors for child development. Since data covering many countries for most causes of mortality are only available since year 2000, their coverage is too sparse to include directly in the economic growth regressions. However, we can look at the correlations of different causes of mortality with overall child survival.

The risk factor data which are both available for most countries and likely to be important proximate determinants of child survival include child anemia, safe water and sanitation, undernutrition and child immunizations. All data are from the World Bank (2021).

Anemia reflects not only the lack of iron in the diet, but also the incidence of malaria, helminth infections (24% of the world population, WHO, 2021b), tuberculosis (23% of the world, CDC, 2020) and schistosomiasis (3% of the world, WHO, 2021a). Unsafe water and poor sanitation transmit gastrointestinal disease causing diarrhea, a major source of undernutrition. Poor sanitation also transmits helminths and schistosomes, which cause undernutrition and anemia.

Table 7 shows regressions of national child survival rates on risk factors using annual data from 2001 to 2019. Country fixed effects are included to account for geographical and other time-invariant omitted factors. For example the burden of disease tends to be higher in tropical areas due to a more conducive climate for pathogens (State of the Tropics, 2019).

All the risk factors except tuberculosis and measles immunizations are statistically significantly correlated with child survival at the 5% level or lower. The R^2 is very high at 0.97. A test for serial correlation in the errors (Drukker, 2003) accepts the absence of an AR(1) process with a p value of 0.24.

To assess the relative importance of the risk factors, column 2 shows the Shapley decomposition which attributes the contribution of each risk factor to the R^2 (Shorrocks, 2013). Anemia alone accounts for a third of the variation in

Table 7: Correlates of child survival

	Fixed Effects	Shapley % of R^2	Including GDP	Shapley % of R^2
Anemia (% of children < 5)	-1.672	33.3	-1.549	33.4
	[0.069]***		[0.074]***	
Safe water (% of households)	1.039	25.7	0.951	25.6
	[0.055]***		[0.064]***	
Sanitation (% of households)	0.102	13.8	-0.022	
	[0.044]*		[0.049]	
Immunization, DPT (% of children < 2)	0.305	9.5	0.284	9.5
	[0.037]***		[0.040]***	
Undernutrition (% of pop.)	-0.306	9.1	-0.361	9.4
	[0.047]***		[0.051]***	
Immunization, Measles (% of children < 2)	0.047	5.9	0.045	6.2
	[0.036]		[0.038]	
Immunization, TB (% of children < 2)	-0.019	2.7	-0.011	3.0
	[0.030]		[0.032]	
log GDP per capita, lagged 10 yrs			5.317	12.9
			[1.041]***	
Constant	905.177		785.952	
	[6.094]***		[24.807]***	
N	2,371		2,193	
Countries	131		124	
$R^2(Corr(\hat{y}, y)^2)$	0.97		0.96	

+ $p < 0.1$; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

child survival not explained by the country fixed effects. Anemia together with safe water and sanitation accounts for 72% of the variation. Undernutrition has a smaller share (8%), but it is closely related to access to sanitation and safe water. The three immunizations together account for 20% of the variation, which is notable given their modest cost.

It could be that the levels of risk factors just reflect income levels, which are really responsible for good child survival. The regression in column 3 includes the natural log of GDP per capita, lagged ten years to avoid possible reverse causality and because it is likely that higher income levels require some time to impact health conditions. The results show that income level is unlikely to drive the relation between the risk factors and child survival. The only substantial change to the estimated coefficients due to including income is for sanitation, which becomes statistically insignificant. The other risk factors are not highly correlated with income levels with the highest correlation coefficient for safe water of 0.36. The Shapley decomposition in column 4 is also largely unchanged, where sanitation is excluded due to the illogical negative but insignificant correlation. The estimated contribution of GDP per capita is only 12% of the explained variation in child survival, indicating that it plays a small additional role in child health.

The country fixed effects reveal lower rates of child survival in the tropics which is not explained by the included risk factors. The child survival rate in tropical countries is 36/1000 lower than in non-tropical countries. After accounting for the risk factors, including GDP per capita, the average difference in tropical versus nontropical country effects is still 31/1000 (statistically different from zero at a 0.1% level), or 86% of the raw difference.

The decomposition of child survival into risk factors on a world scale is not a subtle exercise, but it may still be a useful indication of the relative importance of different risk factors for child cognitive development. The correlations indicate that changes in anemia, water, vaccinations and sanitation, in order of importance, account for more than 90% of the improvements in child survival.

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