The current volume is evidence of the growing awareness within economics of the important connections between health and poverty in less developed countries. The aim of this chapter is to review recent evidence on one potential channel through which health may affect income: education.

Establishing evidence for a causal link of health on education may help to reconcile the seeming contradiction in existing empirical research that although the cross-country correlation between various measures of health status and income is extremely strong (Bloom and Sachs 1998), the microevidence on the impact of poor health on individual labor productivity (as measured by wages) conditional on education is often weak (Strauss and Thomas 1998). However, this does not mean that poor health status through life does not affect wages. Poor health could have a profound impact on lifetime earnings to the extent it affects educational attainment and skills acquisition, in light of the evidence linking education to wages in less developed countries (for recent evidence, see Duflo 2001). These links are likely to be particularly salient in sub-Saharan Africa, the poorest region and the one with the highest tropical disease burden.

Both child and parental health problems potentially affect schooling. Child health-education linkages are the first focus of this survey (section 1). To illustrate, children suffering from acute malaria, say, may be unable (or unwilling) to attend school, and even if they do attend, may have limited ability to focus on learning their lessons.

The second focus is the parental health channel, and in particular the impact of parent death—the most extreme parental health shock—on the schooling of their children (section 2). If parent mortality or morbidity reduces household income, it could affect the ability to invest in child education. This issue is of growing academic and
public policy interest given the rapid rise in the number of orphans during the HIV/AIDS epidemic, especially in Africa.¹

A brief overview of the main themes of this chapter is in order. A key point about existing work is that research progress in disentangling the causal relationships between health, education, and income has been slowed by econometric difficulties, primarily due to both endogeneity and omitted-variable biases (see Behrman 1995 and Strauss and Thomas 1998 for more comprehensive discussions). To illustrate, consider the case in which children with poor health and nutrition status also tend to have below-average school performance (in terms of school attendance, say) in a given sample.

One possible interpretation of this observed pattern is that poor health is the cause of higher absenteeism for these children. But a second plausible explanation is that these children come from households with unobservably lower socioeconomic status, and that this fact leads both to worse health and educational outcomes, or similarly, the children have parents with unobservably less interest in both their child’s health and education, leading to a correlation between child health and education that is not causal. In many existing studies, it is difficult—or impossible—to distinguish between these two explanations, especially when the data used in the analysis consists of a single cross-section of observations.

One goal of this chapter is to focus on recent studies that have made credible attempts to resolve these central identification concerns, through the use of either innovative research designs—including social experiments and randomized evaluations—or rich datasets, especially panel (longitudinal) microdata. There is a growing body of such work, and increasingly solid evidence that both child health status and parental death can have a large impact on education—in particular, on school attendance and enrollment—across a range of less developed country settings. We begin with a discussion of child health and schooling.

1 Child Health and Schooling

1.1 Evidence from Less Developed Countries

There is growing evidence that poor health reduces hours worked, but the evidence on the relationship between current health and wages—and by extension, productivity—conditional on education is largely inconclusive (Strauss and Thomas 1998). The existing research is often difficult to interpret since most studies examine the cross-sectional correlation between current health status and labor market outcomes, and as such are subject to well-known omitted-variable (confounding) and endogeneity (reverse causality) biases.

The parallel nonexperimental research on the impact of child health on education (as surveyed in Behrman 1996) reaches similarly ambiguous conclusions.² To illustrate in nonexperimental empirical research without panel data, Glewwe and Jacoby (1995) present cross-sectional evidence that delayed school enrollment in Ghana is related to child growth stunting. However, despite their creative efforts to address omitted-variable bias—using household-fixed effects (in which siblings are compared), and mother’s height as an instrumental variable—the identification strategy Glewwe and Jacoby employ is still subject to many of the same biases discussed above, and as such, their results are difficult to interpret. The use of longitudinal data can alleviate many of these estimation concerns by allowing researchers to control for unobserved time-invariant household characteristics—for instance, parental tastes regarding child health and education—although such studies are still prone to bias from unobserved time-varying characteristics.

Several studies use a longitudinal dataset from Cebu, Philippines and provide evidence that improved early childhood nutrition may translate into better educational and cognitive performance. For instance, Mendez and Adair (1999) examine the impact of stunting in the first two years of life on cognitive ability test scores and schooling enrollment at ages 8–11, and find strong evidence consistent with a large impact of improved nutrition on subsequent performance in Cebu. Glewwe, Jacoby, and King (2001) examine children from the same dataset and use a structural econometric framework that they argue partially addresses omitted-variable concerns using older sibling height at age 2 as an instrumental variable for the nutritional status of younger siblings when they reach primary school age. Glewwe et al. find that better nourished children enroll in school earlier and learn more per unit of time in school. However, once again the possibility of omitted variables (confounders) that change over time—for instance, a common shock that affects both child health and schooling—may lead to bias and somewhat complicates interpretation of the results.

Alderman et al. (2001) also utilize a panel dataset, this time for rural Pakistan, and once again find that early childhood health and nutrition have large consequences for school enrollment. Their structural modeling approach uses price shocks (from when the children were of
preschool age) as instruments, and they conclude that the effects of child health using this estimation approach are three times as large as one would find relying only on naïve observational estimates.

Of course, the ideal economics study should allow researchers to credibly estimate the impact of child health on both education and later life outcomes such as income. However, the literature on links between child health, nutrition, and long-run life outcomes is even less conclusive, mainly because of the almost complete lack of panel datasets that track individuals from childhood into adulthood in less developed countries. Yet the childhood years are often considered the most important for determining life outcomes, and thus bear most directly on the feasibility of using public policy—including child nutrition and health programs—to break the cycle of poverty.

We are aware of only one existing study that both (1) was conducted with an experimental design, and (2) has a long-run panel dataset that allows the authors to estimate the impact of child health on nutrition and long-run life outcomes: the Institute for Nutrition in Central America and Panama (INCAP) nutritional project in Guatemala (Martorell, Habricht, and Rivera 1995). The project randomly divided four study villages into two groups, two villages where children (and expecting mothers) received a high-energy, high-protein drink (called “Atole”) and two villages where children received a low-energy, no-protein drink (“Fresco”). In follow-up studies conducted at least a decade after the end of the intervention, the research team found that treatment children in the Atole villages showed height and weight gains (Rivera et al. 1995), greater work capacity, especially among boys (Haas et al. 1995), and gains in certain cognitive measures that are likely to be linked to school performance (Pollitt et al. 1983).

This project is arguably the most convincing existing research to show that childhood nutrition and health affects subsequent life outcomes. However, despite its obvious strengths, this project also has several important methodological shortcomings. The most obvious weakness is the small sample size of only four villages and less than two thousand individuals. Moreover, several of the INCAP studies (including Haas et al. 1995, and Khan et al. 1995) apparently fail to account for the intracluster correlation of respondent outcomes within villages in their statistical analyses, probably leading them to overstate statistical significance. Another major concern is sample attrition. Attrition in the 1988 follow-up sample was 27 percent of the original sample, and there was sometimes limited success in obtaining survey consent even from the 73 percent of respondents who had not attrited (especially when blood draws were sought). Clearly, more research is needed to definitively document the impact of child health and nutrition gains on education and, ultimately, on adult living standards.

1.2 Worms and Schooling in Rural Kenya

In a recent paper, Miguel and Kremer (2004) focus on the educational impacts of treatment for intestinal helminth (worm) infection in Kenyan primary schools using a randomized evaluation methodology. The paper’s experimental design, large sample size, and extensive outcome measures make it a particularly useful setting to explore links between health and schooling.

Worm infections—including hookworm, roundworm, whipworm, and schistosomiasis—are among the most widespread diseases in less developed countries: recent studies estimate that 1.3 billion people worldwide are infected with roundworm, 1.3 billion with hookworm, 900 million with whipworm, and 200 million with schistosomiasis. Infection rates are particularly high in sub-Saharan Africa (Bundy et al. 1998; World Health Organization 1993). The geohelminths—hookworm, roundworm, and whipworm—are transmitted through poor sanitation and hygiene, and schistosomiasis is acquired by bathing in infected freshwater. School-aged children typically exhibit the greatest prevalence of infection and the highest infection intensity, as well as the highest disease burden (since morbidity is related to infection intensity), due to a combination of high exposure and immunological factors (Bundy 1988).

The adverse short-run health and nutritional impact of worm infections on children is reasonably well understood: intestinal helminth infections often lead to iron-deficiency anemia, protein-energy malnutrition, stunting (a measure of chronic undernutrition), wasting (a measure of acute undernutrition), listlessness, and abdominal pain (Pollitt 1990), and may also make individuals more prone to other infections by weakening the immune system. If untreated, the infections may have more serious consequences in some cases, especially for schistosomiasis (Bundy 1994).

Intestinal helminths are treated using low-cost single-dose oral therapies appropriate for delivery at infrequent intervals of six months to a year (Bundy and Guyatt 1996). The broad-spectrum anthelminthic albendazole is used for the geohelminths and praziquantel for
schistosomiasis. These drugs have only minor side effects (World Health Organization 1992). Medical treatment with albendazole and praziquantel is also inexpensive: a yearly treatment of albendazole costs less than 50 cents per person per year and praziquantel costs roughly one dollar for a primary school pupil of average weight (Partnership for Child Development 1999). School-based deworming programs that use the existing school infrastructure to deliver anthelmintics have been identified as especially cost-effective in high-prevalence areas, as mass treatment eliminates the need for costly individual screening.

The educational impacts of deworming are considered a key issue in assessing whether the poorest countries should accord priority to deworming, but existing research on these impacts is inconclusive (see Dickson et al. 2000 for a recent survey). Yet the existing randomized evaluations on worms and education suffer from several important methodological shortcomings that may partially explain their weak results. First, existing studies randomize the provision of deworming treatment within schools to treatment and placebo groups, and then examine the impact of deworming on cognitive outcomes. However, the difference in educational outcomes between the treatment and placebo groups understates the actual impact of deworming if placebo-group pupils also experience health gains due to local treatment externalities (due to breaking the disease transmission cycle). Second, although existing studies report the impact of deworming on tests of cognitive performance (such as tests of recall), they typically do not examine other outcomes of interest to policymakers, including health attendance, enrollment, academic tests, or ultimately, labor market outcomes. Finally, none of the existing studies adequately addresses sample attrition, an important issue to the extent that deworming improves school enrollment.

The Primary School Deworming Project (PSDP) in Busia, Kenya offers an opportunity to evaluate the impact of a school-based health program on education (as well as labor market, health, cognitive, and demographic outcomes in planned follow-up studies) within the context of a prospective study. The Kenyan nongovernmental organization (NGO) International Christelijk Steunfonds Africa (ICS) began carrying out the project in Kenya’s Busia district—a densely settled farming region in western Kenya adjacent to Lake Victoria—in late 1997. The 75 schools participating in the program consist of nearly all the rural primary schools in the Budalangi and Fynyula divisions in

the southern Busia district, and contained 32,565 pupils at the start of the study. Baseline parasitological surveys conducted by the Kenyan Ministry of Health indicate that these two divisions had high rates of helminth infection, at over 90 percent. Using modified World Health Organization (WHO) infection thresholds (described in Brooker et al. 2000), over one-third of children in the sample had a “moderate to heavy” infection with at least one helminth at baseline. Primary-school pupils in Kenya range roughly from 7–17 years old.

In January 1998, the 75 schools were randomly divided into three groups (groups 1, 2, 3) of 25 schools each: the schools were first stratified by administrative subunit (zone) and by their involvement in other NGO assistance programs, and were then listed alphabetically and every third school assigned to a given project group. Due to the NGO’s administrative and financial constraints, the schools were phased into the deworming program over the course of 1998–2001, and the order of phase-in was randomly determined, creating treatment and comparison groups (table 6.1). Group 1 schools began receiving free deworming treatment in 1998, group 2 schools in 1999, while group 3 schools began receiving treatment in 2001. To illustrate, the project design implies that in 1998, group 1 schools were treatment schools while group 2 and 3 schools were the comparison schools, and in 1999 and 2000, group 1 and 2 schools were the treatment schools and group 3 schools were comparison schools. Group 1, 2, and 3 schools were in fact similar along nearly all baseline characteristics, indicating that the randomization was successful at creating comparable groups of schools.

The program led to immediate health gains: treatment schools showed significant reductions in the prevalence of moderate-to-heavy helminth infections (25 percentage points); a significant reduction in the proportion of children reporting being sick (in answer to the

<table>
<thead>
<tr>
<th>Year</th>
<th>Group 1 (25 Schools)</th>
<th>Group 2 (25 Schools)</th>
<th>Group 3 (25 Schools)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1998</td>
<td>Treatment</td>
<td>Comparison</td>
<td>Comparison</td>
</tr>
<tr>
<td>1999</td>
<td>Treatment</td>
<td>Treatment</td>
<td>Comparison</td>
</tr>
<tr>
<td>2000</td>
<td>Treatment</td>
<td>Treatment</td>
<td>Comparison</td>
</tr>
<tr>
<td>2001</td>
<td>Treatment</td>
<td>Treatment</td>
<td>Treatment</td>
</tr>
</tbody>
</table>
question, "Were you sick during the past week?" from 45% to 41%); marginally significant gains in height-for-age Z-scores; and the proportion of children with severe anemia (Hb < 100 g/L) fell by half, from 4 percent to 2 percent.

In the paper's main finding, the intervention also reduced total school absenteeism by at least 7 percentage points—or one-quarter—in the first two years of the project (1998-1999), with particularly large school participation gains among the youngest primary school children. The school participation data were collected during unannounced school visits conducted by NGO enumerators, conducted approximately 4-5 times per year in each school. Figure 6.1 presents the time pattern of school participation in 1998 and 1999.

Younger children (grade 4 and below at baseline) had larger average school participation gains in the second year of the study (1999) than older children. These larger gains for the younger children are consistent with the fact that younger children typically have more intense worm infections than older children, and thus might be expected to show larger gains. There were similar school participation gains in 1998 among girls and boys, but girls showed considerably larger gains than boys in 1999. The school participation results are robust to the inclusion of individual and school controls, as expected when treatment is randomly assigned. In an instrumental variable specification—which implicitly assumes that all school participation effects work through the channel of reduced worm infection rates—we estimate that eliminating one moderate-to-heavy infection for one year boosts school participation by a fraction of 0.2 of a year.

In another important result, Miguel and Kremer (2004) find that deworming significantly reduces worm burdens and increases school participation among untreated children in treatment schools—including the older girls excluded from treatment—and among children in neighboring primary schools. Econometric identification of the cross-school treatment spillovers relies on the randomized design of the PSDP: conditional on the total local density of primary school pupils, there is random exogenous variation in the number of pupils assigned to deworming treatment through the program.

Identification of within-school externalities is not based on randomization, since pupils are selected into treatment, with 70-80 percent of eligible pupils receiving some treatment in a given year; however, the fact that new groups of schools are phased into deworming treatment in successive years allowed us to compare the 1998 outcomes of group 1 pupils who elected not to receive deworming treatment in 1998 to the outcomes of group 2 pupils who elected not to receive treatment in 1999 in order to estimate within-school externalities, partially addressing omitted-variable bias problems by comparing pupils who made the same treatment decision. The within-school externality in treatment schools (in the terms of the reduction of serious worm infections among untreated pupils) is over 70 percent as large as the overall effect of the program on treated pupils in the treatment schools—a large effect.

Failure to take these externalities (or spillovers) into account would lead one to substantially underestimate the cost effectiveness of deworming treatment: including the externality benefits of treatment, the cost per additional year of school participation is just $3.50, considerably less than the cost of any alternative method of increasing school participation of which we are aware (cf. Kremer 2003). Moreover, we find that internalizing these externalities would likely require not only fully subsidizing deworming, but actually paying people to receive treatment.
Yet we do not find any evidence that deworming increased either academic or cognitive test scores in Kenya. Deworming-treatment-effect point estimates are consistently near zero and statistically insignificant in the first three years of the program (1998–2000). This is consistent with other studies that concluded deworming has an immediate cognitive effect only for those with the heaviest worm burdens, or with other problems, such as severe undernutrition (Dickson et al. 2000).

However, this is not consistent with the Glewwe et al. (2001) study from the Philippines, which found that child health gains led to more learning per time spent in school. It is unclear exactly what the cause of this discrepancy is, although there are at least three possibilities. First, the deworming program in Kenya led to more crowded classrooms, and thus to potentially negative congestion effects for learning along the lines of Angrist and Lavy (1999); these may partially obscure the positive effects of deworming on learning in the treatment schools. Second, nonexperimental studies like Glewwe et al. and others may be suffering from omitted-variable bias (due to unobserved time-varying characteristics) that leads them to overstate the effect of health on learning. Third, the effects of health and nutrition gains on learning (as captured by tests) may differ between the Kenya and Philippines settings due to other factors, such as the quality of primary school instruction (for instance, the training of teachers or characteristics of the school curriculum), which could be complements to child health in the production of learning. Further empirical work is required to determine which of these explanations is correct.

Data from subsequent years of the Kenya project (2000–2002) have not been fully analyzed, but the preliminary results suggest that there were statistically significant educational attainment gains among girls in early treatment schools. To illustrate, girls initially in grades 5–7 in early treatment schools (groups 1 and 2) had attained nearly 0.2 more years of schooling through mid-2002 compared to girls in the late treatment (group 3) schools—a large impact—and this effect is statistically significant at over 90 percent confidence; by contrast, the gain for early treatment boys in the same grades is smaller and statistically insignificant.

Our results suggest that the impact of poor child health on educational attainment could account for part of the negative cross-country correlation between disease and income documented by Bloom and Sachs (1998), among others. The finding that deworming treatment externalities are large also suggests an important role for public policy, especially given that nearly half of Africa’s disease burden is due to infectious and parasitic diseases (World Health Organization 1999).

Yet the case for public subsidies would be considerably strengthened by further evidence on the long-run effects of deworming spillovers on adult labor market outcomes. The author and collaborators are currently collecting a new dataset, the Kenya Life Panel Survey (KLPS), in order to document the long-run impacts of the deworming program. The goal of that study is to resurvey a representative sample of 7,500 individuals from the baseline Kenya deworming (PSDP) sample many years after the start of the program as individuals enter adulthood, and assess the impact of the program on their labor market outcomes, fertility, marital choices, health (including mental health), cognitive skills, physical strength, and personal happiness, tracing out each step in the chain of causality—from child public health investments, to educational gains, to income and wellbeing later in life.

1.3 Anemia, Worms, and Preschool Participation in Delhi, India

A second project examines closely related issues in a different geographic setting (Bobonis, Miguel, and Sharma 2003). This study evaluates the impact of an NGO (Pratham, Delhi) preschool nutrition and health project in poor communities in eastern Delhi, India, which delivers a cheap package consisting of iron supplementation and deworming drugs to 2–6-year-old children through their existing preschool network. Approximately 68 percent of sample children were anemic (Hb < 11 g/dL) and 24 percent suffered from intestinal helminth (worm) infections at baseline in mid-2001. Anemia is among the world’s most widespread nutritional problems, especially for children (Hall et al. 2001).

The 200 preschools in the study were randomly divided into three groups, and the schools were gradually phased into the program as it expanded over the course of two years. In the first year of the program, the group 1 preschools received the assistance package of iron supplementation (delivered to the schools by NGO field workers, and given to the children by teachers), deworming drugs (400 mg albendazole), and vitamin A, while the group 2 and 3 preschools received only vitamin A and served as comparison schools. In the second school year (2002–2003), group 1 and 2 preschools received the full package, while
the group 3 schools served as the comparison group. This experimental design is thus very similar to the Kenya deworming project described above, and allows us to attribute differences between treatment and comparison schools to the health program rather than to omitted variables.

Existing results are described in an unpublished working paper. During the first year of the project, we find large gains in child weight—over 0.5 kg on average—in the treatment schools relative to comparison schools; estimated weight gains remain positive, although smaller and statistically insignificant, in the second year of the project. Most significantly for this chapter, average preschool participation rates increased sharply by 6.3 percentage points among assisted children over the two years, reducing preschool absenteeism by roughly one-fifth. (Unfortunately, we cannot decisively rule out that some of this effect is due to children attending school in the hopes of getting the iron pills, which were distributed daily at preschools in the weeks following health camps.)

Figure 6.2 presents the time pattern of program impacts, comparing group 1 to group 3 schools (diamonds) and group 2 to group 3 schools (squares). Just as in Kenya, school participation rates increased substantially in the months after schools were phased into the program: group 1 began receiving treatment in December 2001, and group 2 in November 2002.

Given the low cost of the intervention—less than $2 (USD) per child per year, on average—these results suggest that the package of iron supplementation and deworming is a highly cost-effective means of improving child school participation in a poor urban setting where anemia and worm infections are widespread. The results of Bobonis, Miguel, and Sharma (2003) thus largely confirm the earlier Kenya findings that child health gains translate into higher school participation. The demonstration that a nearly identical relationship holds in another geographic setting (urban India), with a younger age group and a different health intervention, provides additional confidence that there exists a robust relationship between child health, nutrition, and school participation in poor countries.

However, note that the claim that improved health and nutrition boosts learning (as captured in academic test scores) is, at present, based solely on the results of nonexperimental studies, as in Glewwe et al. (2001). Due to the young age of the children in the urban India sample, it was not possible to estimate the effect of the program on academic performance. And unfortunately, unlike the rural Kenya study site, high rates of mobility in the dense urban communities of Delhi has made successful tracking of the study sample for follow-up tests logistically impossible given the project's financial resources.4

Randomized evaluations like these in Kenya and India provide particularly transparent and credible evidence to policymakers on program impacts, and have the potential to exert considerable influence on actual policy choices, as argued recently by Kremer (2003) and others. For instance, given the results presented in Bobonis et al. (2003), the Indian NGO that conducted this project is currently planning to expand the preschool health model examined in Delhi to several other Indian cities. The school health program evaluated in Miguel and Kremer (2004) has recently been named a Kenya Ministry of Education "pilot site" for a future national program, and the NGO that conducted the Kenya project has included school health components in its projects elsewhere in Africa, including a districtwide school health project in Mato, Tanzania.
2 Parent Death and Schooling

2.1 Evidence from Less Developed Countries

Understanding the impact of parent death on children has taken on a new urgency with the emergence of the global HIV/AIDS pandemic, especially for Africa, the poorest region and the region most affected by the disease. According to UNAIDS (2002), by 2001 12 percent of all children under the age of 15 years in sub-Saharan Africa had lost at least one parent, and the rate is forecast to continue rising rapidly. For instance, in the rural Kenyan setting in which the deworming study took place, and among the children who had been enrolled in primary school in 1998, by 2002 nearly 20 percent were orphans (either maternal or paternal), and a large proportion of them are likely to be AIDS orphans, given the estimated local adult HIV prevalence of 20–30 percent.

Several studies have examined the issue of parent death and child schooling in recent years, using a variety of empirical methods and data sources, and they yield divergent results. In what follows, in addition to summarizing the existing results, we focus on the methodological differences across studies that could be driving these different estimates. Then, in subsection 2.2, we present unpublished results from Evans and Miguel (2003), a study from rural Kenya that uses longitudinal data in an attempt to address some of the methodological weaknesses of existing work.

In particular, note that many existing studies consist of cross-sectional estimates of the observed differences between orphans and nonorphans at a single point in time, controlling for a limited set of currently observable child characteristics. The results of such studies may be misleading due to both omitted variables and endogeneity; in the absence of longitudinal data, it is impossible to know whether orphans and nonorphans were truly comparable before the parent death, and most significantly, current child and household characteristics used as regression controls may have themselves been affected by the death. Moreover, since parent death is relatively rare in most populations, few studies have sufficient statistical precision to reliably estimate moderate impacts.

The first two studies we focus on find that parent death does substantially reduce child school enrollment. Case, Paxson, and Ableidinger (2002) employed demographic and health surveys (DHSs) collected across 10 sub-Saharan African countries between 1992 and 2000 to estimate the impact of parent death on school enrollment. The large number of survey rounds (19 in all), combined with the relatively high incidence of parent death in their African sample, allowed them to precisely estimate its impact. Their main finding is that orphans are significantly less likely to be enrolled in school than nonorphans, and the result holds up even when including household-fixed effects: orphans are less likely to be enrolled in school than the nonorphans in their household. Part of this estimated effect is accounted for by the fact that orphans are more likely to stay with distant relatives than nonorphans, and they point to provocative theories suggesting that more distant relatives have fewer genetic "incentives" to care for these children. Case, Paxson, and Ableidinger (2002) find no significant difference in parent death effects between boys and girls.

Despite its many strengths, particularly its impressive data effort, the study has a number of limitations that complicate the interpretation of its findings. Foremost, because the data set consists of repeated cross-sections rather than a true panel, the study is unable to address obvious omitted-variable bias problems. Namely, it is unclear whether the estimates are capturing the impact of parent death, or rather the impact of being from the type of family where parents tend to die, perhaps because of particular health behaviors, or other unobserved characteristics (e.g., high discount factors). Although the DHS surveys do contain some household asset controls, this data is collected contemporaneously with the measurement of child orphan status and for that reason is potentially endogenous: households fostering orphans may choose to sell off some assets thus becoming poorer. It would be economically preferable to measure socioeconomic characteristics prior to the parent death. Finally, the DHS data gives no indication of how long a child has been an orphan.

Gertler, Levine, and Ames (2004) overcome some of the identification problems in Case, Paxson, and Ableidinger 2002 using panel data from Indonesia, and estimate similar parent death impacts. They use data from 600,000 households in Indonesia’s National Socioeconomic Survey (SUSENAS) during the mid-1990s. This survey contains information on the occurrence of a first parent death within the twelve months previous to the survey. Thus they have individual-level panel data based on a recall of recent parent deaths, an improvement over several other studies. They are limited, however, by their inability to estimate the impacts of parent death over periods of time longer than one year.
Gertler, Levine, and Ames (2004) find striking effects in Indonesia; a parent death during the past twelve months leads to a doubling of the probability of a child dropping out of school that year. Recognizing the challenge that omitted-variable bias may pose, the authors use matching methods to create a comparison group of children from the same geographic area. One drawback to this technique is that current characteristics are again used to perform the matching, rather than characteristics previous to parent death, which would be ideal. Another limitation is the Indonesian setting, in which parent death is relatively rare, which makes it problematic to generalize findings to African settings, where parent death rates are much higher. Like Case, Paxson, and Apleyding (2002), they find no robust differences in the impact of parent death by orphan or parent gender.

A number of earlier studies, however, do not find any evidence of large parent death impacts. Ainsworth, Beegle, and Koda (2002) analyze a true panel dataset of 1,215 children in northwestern Tanzania—an important methodological improvement over most other studies in this literature—and find much weaker impacts of parent death on schooling than either Case, Paxson, and Apleyding (2002) or Gertler, Levine, and Ames (2004) did. In particular, child school enrollment is unaffected by parent death for nonpoor households, whereas for poor households they find that enrollment is merely delayed for younger children and basically unaffected for older children. Note that although Ainsworth, Beegle, and Koda (2002) control for baseline household characteristics, they do not use fixed effects to capture unobserved differences between households that experience parent death and those that do not.

Several other studies echo Ainsworth, Beegle, and Koda (2002) in finding little or no difference between orphans and nonorphans in terms of school enrollment (see Kamali et al. 1996; Ryder et al. 1994; Lloyd and Blanc 1996), although many of these studies rely on cross-sectional datasets for their analysis, and thus may be less persuasive than the panel studies. Ainsworth and Filmer (2002) employ data from 28 countries across the developing world and show diverse impacts of parent death on school enrollment, providing a possible explanation for the heterogeneity of findings across different settings in the existing studies. A number of reports have claimed that there are gender differences in parent death impacts, with girls suffering more than boys in terms of schooling (World Bank 2002; UNAIDS 2002).

The absence of consistent estimated impacts of parent death on children in sub-Saharan Africa has sometimes been attributed to the strength of extended family (or community) networks that care for orphans (Foster and Williamson 2000; Gregson et al. 2002). One explanation for differences across settings, then, is the possibility that these insurance networks weaken or break down when local orphan rates surpass a certain critical level—although the large estimated effects in Indonesia shown in Gertler, Levine, and Ames (2004) and the small effects in Tanzania (Ainsworth, Beegle, and Koda 2002) do not seem to fit this interpretation given the much higher rate of orphanhood in Tanzania.

An alternative interpretation for the weak orphan effects in Africa is the possibility that HIV/AIDS victims are somewhat better-off than nonvictims in certain settings, at least at the start of the epidemic (perhaps since some occupations whose practitioners are vulnerable to infection—including truckers, soldiers, teachers, and prostitutes—tend to be relatively affluent). To the extent that this household socioeconomic variation is at least partially unobserved by the econometrician, this will lead to an upward bias in the estimated impact of being an orphan on life outcomes in studies relying on cross-sectional analysis. Hence it may partially obscure the true negative impacts of parent death.

2.2 Parent Death and Schooling in Rural Kenya

Evans and Miguel (2003) use the Busia, Kenya deworming study (PSDF) data set, and restrict attention to 13,748 individuals whose parents were all alive at baseline in January 1998 in grades 1 to 8. For fully 7,790 of these individuals (those in grades 3 to 8 present in school on the day of survey administration) we also have baseline 1998 survey information on socioeconomic status and other characteristics. This is a large longitudinal data set, spanning nearly five years, from January 1998 to October 2002, in an African area with high HIV/AIDS prevalence. In this sense, Evans and Miguel 2003 is methodologically closest to Ainsworth, Beegle, and Koda 2002.

Ainsworth et al.’s use of longitudinal data and rich set of baseline educational, household, and health information allow them to avoid some of the methodological shortcomings in existing research. In Evans and Miguel 2003, the identification strategy is simple: we compare those children whose parents died during the period 1998–2002...
to those whose parent did not die, and make the case that that these two groups of individuals—the "became orphan" and "never orphan" groups—are remarkably similar along a range of observable characteristics at baseline. Note that of 11 baseline observables—including measures of child nutrition and health, household socioeconomic status, and school participation—there is a statistically significant difference across the two groups at 90 percent confidence in just one case (observed child cleanliness). Baseline 1998 school participation is nearly identical in the two groups. We also have information on 1997 school participation for a subset of 27 sample schools (that were participating in a previous NGO assistance project), and find that the "became orphan" and "never orphan" groups have nearly identical participation levels in that year, further evidence that the two groups were not experiencing differential levels of time trends with respect to school participation in the years before parent death.

These findings cannot completely eliminate concerns about the suitability of our comparison group—and in the absence of a clean natural experiment, it may be impossible to—but we feel that we are able to lay most potential concerns about the comparability of the two groups. If "became orphan" and "never orphan" households indeed differed sharply on unobservable dimensions—for instance, parents’ commitment to their children—it is likely that these differences would also be reflected along some observable dimension given the rich set of characteristics we employ; however, we do not find any such pattern of observed differences.

The information on child orphan status was collected in late 2002 during a follow-up survey of the 75 deworming program schools. For those pupils who were no longer attending the school, or were not present on the day of the survey, other students in the school were asked about their orphan status and year of parent death. In all, orphan status was unknown, or deemed insufficiently reliable by enumerators, for 25 percent of the original 1998 sample—predominantly those initially in the upper grades, who had been out of school longer than younger pupils (and thus were not as well known in the school); these missing cases are dropped from the analysis. The individuals without reliable orphan data tend to have worse school participation outcomes than other pupils, so note that to the extent that orphan status data are more likely to be missing for orphans (who are fostered in distant geographic districts, say) than for nonorphans, our estimates may underestimate true parent death effects due to attrition bias.

<table>
<thead>
<tr>
<th>Year</th>
<th>Became Orphans</th>
<th>Never Orphans</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1997 School participation rate</td>
<td>0.83</td>
<td>0.81</td>
<td>0.02</td>
</tr>
<tr>
<td>1998 School participation rate</td>
<td>0.92</td>
<td>0.92</td>
<td>0.00</td>
</tr>
<tr>
<td>Child weight-for-age (Z-score)</td>
<td>-1.40</td>
<td>-1.45</td>
<td>0.05</td>
</tr>
<tr>
<td>Child had malaria in past month</td>
<td>0.40</td>
<td>0.39</td>
<td>0.01</td>
</tr>
<tr>
<td>Child wears shoes to school</td>
<td>0.13</td>
<td>0.14</td>
<td>0.01</td>
</tr>
<tr>
<td>Child wears school uniform to school</td>
<td>0.85</td>
<td>0.87</td>
<td>0.02</td>
</tr>
<tr>
<td>Child appears &quot;clean&quot;</td>
<td>0.58</td>
<td>0.62</td>
<td>0.04</td>
</tr>
<tr>
<td>Household has latrine</td>
<td>0.81</td>
<td>0.82</td>
<td>0.02</td>
</tr>
<tr>
<td>Household owns cattle</td>
<td>0.49</td>
<td>0.49</td>
<td>0.00</td>
</tr>
<tr>
<td>Household owns goats</td>
<td>0.38</td>
<td>0.41</td>
<td>0.03</td>
</tr>
<tr>
<td>Household owns chickens</td>
<td>0.93</td>
<td>0.93</td>
<td>0.00</td>
</tr>
</tbody>
</table>

Notes: ** = Significant at 95 percent confidence, * = Significant at 90 percent confidence.

Moreover, to the extent that information on the exact year of parent death is captured with some error using this indirect survey method, this should lead us to further underestimate parent death impacts under plausible assumptions on measurement error (Aigner 1973). Finally, a third reason to think our results are likely to underestimate the true effects on children is the possibility that the health of parents who die begins to decline in the year (or years) before their death; in this case, the difference between child school enrollment immediately before and after the parent death understates the total effect of parent illness and death taken together.

Now to the findings themselves. Parent death has a large negative impact on the child school participation rate, defined as the total proportion of unannounced school attendance verifications that the child attended: on average, school participation falls by 0.039 (standard error 0.013) in a specification including extensive individual baseline heath and socioeconomic controls, and 0.028 (standard error 0.014) when individual fixed effects are included. All specifications include birth year cohort-gender indicator variables and polynomial time trends to capture different enrollment patterns by different demographic groups. Effects are robust to the use of an alternative measure of school participation, an indicator variable for enrollment in school
yielding estimated magnitudes similar to Gertler et al.’s (2004) findings in Indonesia, more than a doubling of the drop-out rate. The impacts of maternal and paternal death are nearly identical, and there are no significantly different impacts by child age or gender; the lack of difference in orphan impacts by child gender appears to be one of the most robust results of the recent literature (Case, Paxson, and Apley 2002; Gertler, Levine, and Ames 2004).

Figure 6.3 presents the time patterns of the main effects, and indicates that although school participation rates were similar in the years before parent death, they drop sharply in the year of the death and then continue to fall for up to three years afterward. In other words, there is no evidence of orphan recovery after parent death as households adapt to their new circumstances.

In terms of the magnitude of effects, note that the estimated effect of a parent death in reducing school participation (0.03–0.04), although not trivial, is far smaller than the effect of eliminating one moderate-to-heavy worm infection for a year (approximately 0.2, see section 2)—although it remains likely that our parent death estimates are lower bounds on true effects, as discussed above. For another comparison, the effect of parent death is similar to the estimated impact of several measures of poverty. For example, households without latrines or toilets at their home compound (about 20% of the sample) have school participation rates during 1998–2002 that are about 0.03 less than households with latrines, conditional on the other household controls. Thus, the findings are broadly consistent with much of the existing literature. We find that the impact of parent death is indeed negative in terms of school participation, but that the average effect is relatively small, even in an area of Kenya with high HIV/AIDS prevalence—a finding that echoes work by Ainsworth, Beegle, and Koda (2002).

Also note that, again like Ainsworth, Beegle, and Koda (2002), we find that parent death impacts differ across socioeconomic groups, with children from the poorest households experiencing the greatest reductions in school participation: children from households without latrines or toilets experience a drop in school participation after parent death of 0.096 (significant at 99% confidence), whereas the effect for children from households with latrines is −0.012 (though not statistically significant). Similarly the likelihood that a child drops out of school (through 2002) after a parent death is 0.061 for children from households without cattle (about half of the sample does not have cattle), but near zero for households with cattle. This finding suggests that household wealth buffers children from the shock of parent death, or perhaps that households with greater wealth can call on relatives with more resources to foster the orphans, or at least assist in the payment of school expenses.

In contrast, community level characteristics do not lead to differential effects of parent death. Most significantly, there is no evidence that orphans fare any worse in primary school communities with higher rates of orphanhood. Nor is there significantly lower average school participation in communities with high rates of orphanhood, suggesting that nonorphans are not suffering from being in communities with more orphans. The large variation in orphan rates across communities in the Kenyan study area in Evans and Miguel 2003 makes it particularly well suited to explore this issue—there is considerable variation in local orphan rates within our study area, from 10 percent in the northern Funyula division (a subset of our study area) up to nearly 40 percent in southern Budalangi. These findings argue against the claim that social networks in rural western Kenya are largely breaking down under the strain of HIV/AIDS deaths, and that the growing number of orphans cannot be effectively taken care of by surviving relatives and
other community members. Of course, further research is needed to understand how general these initial findings are beyond rural western Kenya.

3 Conclusions

Taken together, we conclude that there is increasingly strong evidence that both child health shocks and parent death have substantial effects on school participation rates, and thus presumably human capital accumulation, across a range of African, Asian, and Latin American settings. The evidence on how child health affects school learning (as measured by test scores) is, however, less conclusive than the now considerable evidence on school attendance and enrollment. Existing work also suggests that child health and nutritional status have a much larger impact on education than parent health, even in the extreme case of parent death.

Further long-term longitudinal studies are needed in less developed countries to definitively make the claim that health shocks affect income later in life, and to thus make the link with economic growth and development. The recent Indonesia Family Life Survey (IFLS) is the type of high-quality household panel that is needed for this endeavor, and presumably in future survey rounds it will allow investigators to approach these issues. Another ongoing data collection effort that may shed light on these issues is the Kenya Life Panel Survey (KLPS), which is discussed in section 1 above.

How large could the long-run effects of poor health on economic growth and development really be? Unfortunately, the answer to this question remains elusive, despite recent research progress in economics (much of which is surveyed in this volume). But there is suggestive evidence from at least one once-developing country—the United States—that the long-run effects of public deworming investments could be very large indeed. Recent economic history research finds that the Rockefeller Sanitary Commission’s deworming campaigns in the U.S. South in the 1910s had major impacts on educational attainment and income (Bleakley 2002) and on agricultural productivity (Brinkley 1994). In fact, Bleakley estimates that each case of hookworm reduced school attendance by 0.2—which is nearly identical to the Kenya estimates presented in Miguel and Kremer 2004. This historical evidence provides hope that current public health investments in chil-

dren in less developed countries might hold the seeds of their future prosperity.

Acknowledgments

I thank Gustavo Bobonis for his insightful comments. All errors remain my own. I also thank the Economics and Health Research Center at Universitat Pompeu Fabra for their unrestricted financial support via the Merck Foundation Company.

Notes

1. Another possible channel is through teacher health. To the extent that ill teachers miss class or teach poorly, this could also negatively affect child learning. Unfortunately, there has been limited empirical work—to my knowledge—on this issue and so it is not a focus of this survey.

2. One notable exception to this generalization is the literature on the impact of iron supplementation. Several studies on iron supplementation employing randomized evaluation methodologies find subsequent test score gains (see Nokes, van den Bosch, and Bundy 1998 for a survey).

3. In 2001, parents in half of the group 1 and group 2 schools were also required to pay a small amount of money for the deworming drugs in an NGO cost-recovery program.

4. In fact, by the end of the second year of the study, sample attrition rates were already high, owing mainly to the closure of numerous preschools between the first and second academic year (although fortunately for the econometric analysis, attrition rates are nearly identical between the treatment and comparison preschools).

References


