Exploiting Externalities to Estimate the Long-Term Effects of Early Childhood Deworming*

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Abstract

I investigate whether a school-based deworming intervention in Kenya had long-term effects on young children in the region. I exploit positive externalities from the program to estimate impacts on younger children who were not directly treated. Ten years after the intervention, I find large cognitive effects—comparable to between 0.5 and 0.8 years of schooling—for children who were less than one year old when their communities received mass deworming treatment. Because treatment was administered through schools, I also estimate effects among children whose older siblings received the treatment directly; in this subpopulation, effects are nearly twice as large. (*JEL:* I10, O12, O15)

1 Introduction

Shocks in early childhood can permanently transform an individual's potential lifetime health, earnings, and cognition. Several variations of this idea, as hypothesis or as stylized fact, are well-known. The lasting effects of nutrition shocks a child experiences in utero and in early childhood are collectively referred to as the "Barker Hypothesis;" specifically before birth, the "fetal origins" hypothesis (Almond and Currie 2011b). Collectively, the windows in a child's life during which inputs are most important for cognitive development are referred to as "critical" and "sensitive" periods (Knudsen 2004). While early nutritional deprivation can reduce adult height (Hoddinott and Kinsey 2001), early childhood is also a particularly important period for cognition (Cunha and Heckman 2008). Yet because of the demanding longitudinal data required, until recently, very few studies had successfully documented these patterns; fewer still were able to establish causal relationships between external influences early in life and long-term outcomes.

Panel studies have provided one empirical avenue for studying these effects: in Britain, cognitive skills at age seven predict around 20 percent of the variation in adult wages (Almond and Currie 2011a); in the US, parental income shocks in the first several years of a child's life matters much more for that child's eventual adult income than do shocks after the child's fifth birthday (Duncan, Ziol-Guest and Kalil 2010). In the US, variation in child height early in life predicts test scores later in life, even among children with the same mother (Case and Paxson 2010).[1]

[1]Smith (2009) follows a similar approach, showing that in the US, variation in childhood

A different strand in this literature approaches the problem by showing that specific exogenous environmental shocks have lasting repercussions when they occur in early childhood: in Indonesia, beneficial rains in the year of a girl's birth increase her adult height by more than half a centimeter, and raise her eventual educational attainment and wealth (Maccini and Yang 2009); in Zimbabwe, drought and civil war during the first two years of a child's life reduce his eventual height and educational attainment (Hoddinott and Kinsey 2001, Alderman, Hoddinott and Kinsey 2006a). In contrast, the same shocks occurring later in life do not have long-run effects on these outcomes.

Though unpredictable and extreme shocks periodically affect a small fraction of the population, less is known about whether policies—usually interventions aimed at addressing more mild conditions—can permanently alter human capital in this way. Still, a few exceptions stand out. Field, Robles and Torero (2009), for example, have shown that children who were in utero when their mothers received iodine supplementation eventually attain more years of schooling than their siblings who did not benefit from the iodine. Gertler, et al. (2014), provide a demonstration of the lasting socioeconomic benefits of early childhood interventions, by showing that an intensive early childhood stimulation intervention in Jamaica had large effects on eventual adult earnings. Similarly, in Guatemala, nutritional supplementation early in life led to earnings increases for men more than twenty years later (Hoddinott, Maluccio, Behrman, Flores and Martorell 2008). Improvements in early life health outcomes in the United States, brought about in part through racial

health predicts subsequent household income and wealth, even within families.

integration of hospitals, are thought to have narrowed the black-white test score gap substantially once those healthier children became teenagers (Chay, Guryan and Mazumder 2009). Early childhood, however, is a particularly difficult time in a child's life from the perspective of policy: neither in the womb nor yet in school, this "sensitive" period falls beyond the reach of many government programs.

In this paper, I examine the treatment of a disease that, while rarely fatal, is highly prevalent among children around the world: intestinal parasites. These helminths (worms) infect more than one billion people worldwide: predominantly young children in Asia and Sub-Saharan Africa (Hotez, *et al.* 2006). Helminth infections directly cause anemia and listlessness, and may result in chronic symptoms (Bleakley 2007). A variety of studies have shown gains in health, cognition, and school attendance among school-age children given deworming medication; current research suggests that deworming medication may be one of the most cost-effective possible ways to increase school attendance and improve adult outcomes (Miguel and Kremer 2004; Bleakley 2007; Bundy, *et al.* 2009). The existing long-term research on this topic follows individuals treated for deworming after entering primary school, rather than in early childnood (Baird, Hicks, Kremer and Miguel 2014).[2] Despite this growing body of evidence, a recent review has come to much more modest conclusions: in relation to an array of outcomes—school performance and cognition among them—the review finds either insufficient reliable information

[2]Bleakley's (2007) study follows impacts of deworming into adulthood, using a measure of treatment varying from 0 to 19 years of childhood; it provides clear evidence of lasting benefits of deworming, but not specific benefits in early childhood.

on whether deworming treatment has any beneficial effect, or finds reason to believe that there is no effect at all (Taylor-Robinson, Maayan, Soares-Weiser, Donegan and Garner 2012).

In the sections that follow, I present evidence on the long-term effects of reducing helminth infection in *early* childhood by exploiting externalities from a randomized deworming intervention in Kenya. I take a novel approach to the phased randomized intervention first studied by Miguel and Kremer (2004), in which deworming was randomly introduced into communities (once initiated, the program remained present): unlike Miguel and Kremer (2004) and its follow-up studies, I follow a different, younger cohort of respondents. Though mass deworming efforts are often aimed only at school-age children, this kind of community-based deworming has large epidemiological spillovers both on other schoolchildren (Miguel and Kremer 2004) and on others in the community (Bundy, *et al.* 1990). Taking advantage of these spillovers, I gathered new data in 2009 and 2010 in order to compare children who were in their first years of life at the time that treatment started in their communities to children from the same cohorts in as-yet untreated communities. This study–and that of Croke (2014)–together represent the first sets of evidence on long-term effects of deworming early in life.[3]

I find large effects on cognitive performance equivalent to half a year of schooling, robust to a variety of specifications, more than ten years after the original intervention. Effects are strongest among those who were likely to have

[3]While Croke follows a population in which many young children directly received deworming treatment (as I discuss in greater detail in Section 2.4), this study relies on spillovers from a (likely) higher-compliance intervention.

an older sibling in school at the time of the original intervention—and particularly so if that sibling was female—as one might expect from an epidemiological perspective. I do not find any statistically significant long-term effect on measures of stunting or height, suggesting that—consistent with the nature of the intervention and the nutritional status of the population—extreme caloric deprivation was neither a central issue for this population, nor (even if, for some, it is a central issue) was it the condition this intervention chiefly addressed. My results support the theories that sensitive periods in early childhood are essential for cognitive development, and provide evidence that inexpensive actions are available that could produce lasting improvements in the lives of millions. The main result, along with the identification strategy, is summarized in Figure 1, and is discussed in greater detail in the sections that follow.

In relation to the conventional wisdom–and literature gap–articulated by Taylor-Robinson et al. (2012), my findings and those of Croke (2014) corroborate one another in both filling the gap and defying the conventional wisdom: This study and Croke (2014) find large, lasting, positive effects of early childhood deworming on cognition.

The remainder of this paper is organized as follows: In Section 2, I discuss the nature of the disease and the original intervention in Kenya; in Section 3, I provide details on the new data collection undertaken in 2009 and 2010; Sections 4 and 5 present the identification, estimation, and results of my analysis in light of the existing literature; Section 6 discusses relative costs and benefits; and Section 7 concludes.

2 Background

2.1 Biology

A handful of helminth species are responsible for infecting at least one billion people (Hotez, *et al.* 2006): schistosomes, along with soil-transmitted "geohelminths:" roundworm, whipworm, and hookworm.[4] Several of these species are endemic in western Kenya, and though these infections can be addressed inexpensively with existing drugs, they usually go untreated.[5] All of these parasites inhabit parts of the human digestive tract; female worms produce eggs that spread via human excrement.[6] Subsequent infection of new hosts follows different routes depending on the parasite species. In the case of whipworm and roundworm, an individual is infected by ingesting a worm egg (often from contact with soil contaminated with feces). Other species infect simply via human contact with worms in a particular phase of their life cycles.[7]

Thus far, school-age children have been emphasized in studies of deworming because they are known to host the highest numbers of parasites (Bundy 1988). However, very recent studies also document child health improvements in response to early childhood deworming. Despite promising short-term results,

[4]Relevant species are *Schistosoma mansoni* and *haematobium*; *Ascaris lumbricoides*; *Trichuris trichiura*; and hookworms *Necator americanus* and *Ancylostoma duodenale*.

[5]Albendazole and mebendazole are anti-geohelminth medications. Schistosomiasis is usually treated with praziquantel. Diagnosis, which involves laboratory work, is much more expensive than the medications themselves; mass deworming with these modern drugs only began in the 1990s, but has recently become more common.

[6]Here, I discuss *Schistosoma mansoni* rather than *Schistosoma haematobium*, as urinary schistosomiasis is not endemic in western Kenya.

[7]Hookworms often penetrate the skin through the sole of the foot, while schistosomes enter the skin through lake or river water while part of a person's body is immersed (Bundy et al. 2001, Mott 2001).

no published study to date has shown whether early childhood deworming can have lasting benefits.

The current debate on deworming reflects limitations of the current evidence base. Few studies have been designed with long-run health and education outcomes in mind, or with the epidemiological spillovers of deworming treatment taken into account. As such, a recent review, focusing on randomized trials, found insufficient evidence on the question of whether deworming affects school-related outcomes. From a handful of studies, it interprets the current literature as finding no evidence for effects of deworming on cognition (Taylor-Robinson et al. 2012).

There remains substantial room for additional research in this area, as recent discussion has pointed out (Bundy, Walson and Watkins 2013). To take an example pertinent to this study, in order to reach the conclusion that there is no evidence for effects on cognition, the recent review chose study inclusion criteria that forced it to rely upon just two studies with short follow-up periods and low baseline rates of worm infection.[8]

2.2 Past intervention

Between 1998 and 2001, Miguel and Kremer (2004) randomly phased in deworming drugs to a group of 75 primary schools in western Kenya, in the "Primary School Deworming Project," PSDP: once PSDP deworming started in a community, it was continued thereafter. Children in this region suffered

[8]The criteria of the Taylor, et al. review focused on randomized or quasi-randomized trials. Bleakley's 2007 historical analysis of a natural experiment, finding long-term cognitive effects of deworming in the United States, was thus excluded from consideration.

from high rates of worm infection: at baseline, 92 percent of children had at least one type of worm infection, and many were infected with multiple species of worm; hookworm alone infected more than 70 percent of school-age children (Brooker, Miguel, Moulin, Luoba, Bundy and Kremer 2000, Miguel and Kremer 2004). The school-based deworming program therefore followed a mass-deworming protocol rather than testing before treatment. The program reduced infections, reduced anemia, and increased school attendance. Only schoolchildren were dewormed, but the authors found large spillovers within the community: in terms of school attendance, for example, children in dewormed areas who were not actually given medication still received nearly 60% of the benefits of direct deworming. This is consistent with evidence from Montserrat, where mass deworming of children aged 2-15 may have reduced parasitic loads in adults who received no medication (Bundy, *et al.* 1990). No significant test score gains were documented in the years immediately following the PSDP intervention, however. Thus far, the long-term effects of the intervention in Kenya on the recipients of medication have included, in early adulthood, clear increases in wage and hours worked (Baird et al. 2014). Improvements in labor market outcomes are attributed to improved health; test scores suggest increased human capital in the long run, but not through general intelligence measures.

2.3 Critical periods

Despite reducing anemia and improving school attendance, the original intervention appeared not to improve either measures of general intelligence in the

long run or academic test scores in the short run for the direct recipients of the deworming medication. Part of the reason for this may be that for some types of outcomes, the deworming intervention came too late for participants in the original study: they were already of school age. The crucial phases for some aspects of both physical and cognitive development are thought to be within the first two or three years of life (Grantham-McGregor, *et al.* 2007, Knudsen, *et al.* 2006); nutrition shocks and changes to environmental stimuli in this period matter much more than they do later in life.[9] Two recent studies use rainfall changes to measure this effect. Hoddinott and Kinsey (2001) find that children in Zimbabwe who are malnourished between the ages of one and two because of a drought remain permanently 1.5-2 cm shorter than their counterparts who were not exposed to the same conditions; older children exposed to the drought do not seem to suffer long-term harm. Maccini and Yang (2009) investigate long-term effects of good rainfall on children in Indonesia, and find that girls born in an area receiving 20 percent more annual rainfall than usual gain an additional 0.57cm in adult height, and complete an additional 0.22 grades of school, compared to children whose regions did not receive such beneficial rains.[10] Rainfall in other years had no significant long-term consequences.

Because the intervention for schoolchildren in Kenya had such large spillover

[9]Windows during which such outside influences have especially strong effects are referred to as "sensitive" periods (Knudsen 2004); when the consequences are permanent, these periods are referred to as "critical." But because "critical" and "sensitive" periods differ across faculties (Knudsen, *et al.* 2006); I remain agnostic on whether de-worming could intervene in a particular "critical" period, relying instead on evidence that analogous early childhood interventions had substantial effects on health and education.

[10]Rainfall shocks at age two have similar (though statistically insignificant) effects on both outcomes.

effects,[11] I hypothesize that children who were not yet old enough to attend school also garnered benefits. Because of their age at the time of the intervention, I further hypothesize that these younger cohorts may have been more sensitive to the intervention than the older children who actually received the drugs. Until recently, however, younger children were not thought to benefit substantially from deworming, because their parasitic load is typically much lower than it is in older children.

Several very recent studies demonstrate links between early childhood deworming and health, including four studies in East Africa all documenting short-term health gains. Alderman *et al.* (2006b), for example, found that de-worming brings about weight improvements in pre-school-age children in Uganda, in a district that borders the PSDP study area around Lake Victoria. Children in the Uganda study were between 1 and 7 years old, but the study did not disaggregate effects by age; however, the study by Stoltzfus *et al.* (2004) in Zanzibar did. They show that children who were treated when less than 30 months old gained the most. Within this young cohort, incidence of mild wasting[12] was cut nearly in half, from 36% in the control group to 18% in the treated group; older children did not improve nearly as much. The authors took note of this surprising aspect of their results: "The benefits thus occurred in the age group at highest risk for anemia and growth retardation, but in the age group with the lowest intensity of helminth infections."

The literature thus lays the groundwork for the present study. The simple

[11] Baird et al. (2014) summarize the spillover as being 78 percent as large as the direct effect of deworming.

[12] *Mild wasting:* having weight-for-height worse than one standard deviation below average, $WHZ < -1$

question I ask is whether children who were infants or not yet born at the time of school-based deworming in Kenya received spillover benefits from the original PSDP intervention, by experiencing early childhood in a low-worm-infection environment. They were at the right age for long-term impacts to be large; recent literature suggests that worm infections are important even in the first year outside the womb; and the PSDP study showed that children who were simply near the dewormed schools also benefited from spillovers.

2.4 Long-term studies of early deworming

This paper is not alone in its long-term study of early childhood deworming. The most recent contribution in this vein is that of Croke (2014), who conducts a long-term follow-up of the Alderman, et al. (2006) study by matching study areas to recently collected academic-subject-specific performance data in Uganda.[13] Croke demonstrates higher mathematics and English scores for children given deworming medication early in life; effect sizes are comparable to those here, though the mechanics of the intervention and the nature of the outcome variables differ somewhat. In that case, deworming medication was delivered as a component of "child health days" in Uganda; in the present case, deworming was delivered at school. Given the attendance rates involved, in any given round of deworming, this produced a lower compliance in Uganda than in the present study environment in Kenya. In Uganda, the target age of medication recipients was between 1 and 7 years old; in the present case, it was school-age children. This means that Croke estimates a long-run direct

[13]The dataset on academmic performance that Croke uses was collected independently by the Uwezo initiative.

effect, while the present case relies on spillovers. Finally, Croke's outcomes are centered on tests of academic skills rather than underlying cognitive abilities. Though these are overlapping areas, and measures of the two are inextricably linked, the conclusions we draw based on them may differ. I discuss this further in the results section.

3 Data collection, 2009-2010

In 2009 and 2010, a field team in Kenya collected height, weight, and migration data from more than 20,000 children at all of the deworming project schools in Samia and Bunyala districts of Kenya's Western Province.[14] For a subset of just over 2,400 children, the team also conducted detailed cognitive assessments. Children from the same age cohorts were included during both data collection years: in 2009, this meant including every child between the ages of 8 and 14; in 2010, it meant every child between the ages of 9 and 15.

These age cohorts were chosen both because they were still enrolled in primary school at the time of data collection, and because of how these cohorts align with the original intervention. The randomized design of the original deworming project at the community level permits its use for estimation in this study, as shown in Figure 1: In communities where deworming began in 1998, the children who were born in 1998 (and who I find in 2009 or 2010 as 11- or 12-year-olds) began experiencing the effects of community deworming in their year of birth; thus their age at the time of deworming, A_{id}, is less than

[14]Here, we follow the original Primary School Deworming Program school lists, and pursued those that were not flooded (causing a temporary program disruption) at the time of the intervention.

one year (see Panel A, middle column, top row). I consider them "treated" for the purposes of the present study. Children from the same birth cohort but in a community where deworming only began in 2001 had to wait until age three for school-based deworming to arrive; I consider them "untreated" for the purposes of the present study (see Panel A, middle column, bottom row). The figure outlines these definitions of treatment and comparison, by community and by treatment arm.[15] Because deworming started in different communities at different times, I can control for age at observation separately from age at the time of community treatment.

Summary statistics on the study population are shown in Table 1. Roughly half the sample is female, the average age is between 11 and 12, and average height is roughly what would be expected for these ages, if a bit low. Roughly 28 percent of the sample had migrated since birth. In-migration to these communities in response to Kenya's 2008 post-election violence left school populations inflated with recent migrants from urban areas; for my results, I exclude those migrants from all regressions, since they were not present in these communities at the time of deworming in the late 1990s. Out-migration is much less of a concern, since these rural areas are moderately ethnically homogeneous, and did not experience notable conflict.

In Panels B and C of Table 1, I restrict attention to the sample of non-migrants. Panel B shows that the non-migrants are demographically much the

[15]For the borderline case of children whose age was approximately 1 when deworming arrived, I consider them neither clearly treated nor untreated, as I only measure age up to a precision of one year increments, and the literature is not definitive on how these cases should be handled, had measurement been precise. Empirically, this is absorbed through an additional dummy variable, as discussed further below.

same as the full sample, and goes on to tabulate several other characteristics: 21.6 percent of this population is stunted[16]; respondents had an average of 1.45 older siblings who attended the same primary school; 22.5 percent had at least three such siblings, while 37 percent had no older siblings who attended the same primary school. These measures are used to assess the likely intensity of the deworming spillover effects, as discussed further in Section 5. Panel C simply shows the distribution of indicators for age at the time of community deworming, explained in Figure 1.

In Panels D and E, I further restrict the sample to those for whom a cognitive survey was carried out. Because the cognitive survey takes roughly ten times as long as anthropometric measurement, the cognitive outcomes were gathered only for a random subsample of respondents. Panel E shows that the characteristics of the respondents sampled for cognitive surveys do not substantially differ from the characteristics of all respondents.

The cognitive module included two measures of "verbal fluency," in which children name as many items in a category as they can in one minute. The first category is foods; the second is animals. The Peabody Picture Vocabulary Test (PPVT-4, Form B) measures "receptive vocabulary," in which children point to one of four pictures that best matches a word that has been read aloud to them. There are eighteen levels of the test, each with twelve words; respondents proceed up through levels of increasing difficulty until they make nine mistakes in a single level, and are likely to be simply guessing. For reasoning, I use the 12-question Set B of J. C. Raven's Progressive Matrices, a

[16]Stunting: height-for-age Z-score less than -2

series of puzzles commonly used to measure nonverbal reasoning and general intelligence.[17] For short-term memory, I use "digit-spans" of increasing length, in which respondents attemp to repeat a string of numbers back to the interviewer, either forwards or backwards. I provide raw means and standard deviations in Table 1, but for all regressions, I consider standardized versions of these cognitive measures, each re-scaled to have mean zero and standard deviation one.

Though it is not tabulated, I also condense these six measures using their first principal component in some parts of the analysis. Interpretation of coefficients on cognitive tests is clarified in Appendix Tables A2 through A5. The first column of Table A2 shows the weights on each outcome that yield the first principal component used in the analysis. Weights are almost equal across the different cognitive outcomes.[18] Because almost equal weight is given to each measure, I also construct a simple normalized sum of cognitive measures as an outcome to confirm the robustness of the findings where relevant. Correlations among cognitive measures are shown in Table A3: all are positive. To orient the reader, the cross-sectional relationships between cognitive performance, age, and grade in school are shown in Appendix Tables A4 and A5. In the cross-section, coefficients on grade in school are typically one third larger than the coefficients on age, since pupils tend to repeat one grade out of every three. Conditional on grade in school, older children perform worse, since they have

[17] See discussion in Cattell (1971) and Raven (1989) of the matrices and what they measure.

[18] The lowest weight is for "Verbal Fluency: Foods," perhaps the noisiest measure because it was the first exercise in the cognitive module. Low R^2 for regressions with this outcome also speak to its relative noisiness.

typically chosen to repeat grades more frequently.

4 Estimation

I begin discussion of estimation with a simple equation. For each individual i, consider that the relationship between an outcome, Y_i, and an indicator, $Before_i^C$, for whether that individual's community participated in mass deworming before the individual was C years old, is given by:

$$Y_i = \beta_1^C \cdot Before_i^C + \epsilon_{1i} \tag{1}$$

. Because of the original randomized design, conditional on age and data collection year, this type of exposure was actually, itself, randomized. This leads to two slightly richer specifications. First, within a single birth cohort, and controlling for the data collection year with a dummy variable, $D_{Year_i=2010}$:

$$Y_i = \beta_2^C \cdot Before_i^C + \gamma_{2010} \cdot D_{Year_i=2010} + \epsilon_{2i} \tag{2}$$

. Second, aggregating across cohorts, and controlling for a set of interacted indicators for both year of data collection and respondent age:

$$Y_i = \beta_3^C \cdot Before_i^C + \sum_{A,Y} \gamma_{AY} D_{Age_i=A} \cdot D_{Year_i=Y} + \epsilon_{3i} \tag{3}$$

. Estimating Equation 3 will provide more statistically powerful tests than would Equation 2, simply because it uses more of the available data; however,

Equation 2 is a simple conceptual demonstration of the intended exercise. A challenge in estimating Equation 3 in the present environment is that children in this study report their age rounded to the year. From this type of data, I can roughly construct year of birth and thus, in relation to the randomized program rollout, the age at the time of community deworming. However, the equations above are specified in terms of whether deworming arrive in a child's community before the child turned C years old. For the cohort whose survey data suggest that their age was exactly C at the time of deworming, there is an equal probability that they really were or were not at least that exact age at the time deworming began. Direct estimation of β_3^C in equation 3 based on field data would thus be biased toward zero, because half of one cohort would be incorrectly categorized. This can be resolved, however, by including an indicator for reporting exactly a particular age at the time of deworming:

$$Y_i = \beta_4^C \cdot Before_i^C + \beta_4^{eC} \cdot D_{A_{id}=C} + \sum_{A,Y} \gamma_{AY} D_{Age_i=a} \cdot D_{Year_i=Y} + \epsilon_{4i} \quad (4)$$

. Finally, though it diminishes statistical power, an extension of this specification is to consider separately estimating the effect of community deworming arriving at each exact age, subject to the survey data concerns already decribed:

$$Y_i = \beta_5^C \cdot Before_i^C + \sum_{c=C}^{C_H} \beta_5^{ec} \cdot D_{A_{id}=c} + \sum_{A,Y} \gamma_{AY} D_{Age_i=a} \cdot D_{Year_i=Y} + \epsilon_{5i} \quad (5)$$

In case anything systematically differs for boys and girls in these communities and years, I can also absorb additional variation by separating the age and

data collection year indicators by gender. Thus, for example, equation 4 above becomes:

$$Y_i = \beta_6^C \cdot Before_i^C + \beta_6^{eC} \cdot D_{A_{id}=C} + \sum_{A,S,Y} \gamma_{ASY} D_{Age_i=a} \cdot D_{Sex_i=S} \cdot D_{Year_i=Y} + \epsilon_{6i} \quad (6)$$

. I do this for all the empirics that follow, though in practice, the results do not differ substantively if instead the gender interactions had been left out of the controls.

Because worms do not infect fetuses *in utero*, and are not as serious a health concern for mothers as they are for children outside the womb, the earliest sensible value to consider for C is $C = 1$. In what follows, I will consider a child "treated" with early deworming if her community started receiving deworming treatment before she was one year old.

In clarifying what this specification means, it is worth pointing out what it doesn't mean. There are certainly benefits to school-based mass deworming beyond age two, and all of the subjects in these datasets benefited in that way: by the time they were school-age, deworming was present in every community in this study. So to bring the analytical framework above to the variation in treatment timing in the study site in western Kenya is to arrive at a lower bound on deworming benefits: this approach estimates the differential benefit of particularly early deworming spillovers. Though it is the earliest sensible cutoff, $C = 1$ is not necessarily the "true" cutoff for a critical period in relation to deworming spillovers. The present data offer only limited variation to explore whether this cutoff appears sharp, or has more of a dose-response

structure under some threshold age. I discuss alternative specifications in relation to this question (and thus assumptions about the relevant cutoff, and what those assumptions would yield) in Section 5.2 and in Table A1.

An alternative specification, based on Bleakley (2007), is to consider exposure to early childhood deworming in years:

$$Y_i = \beta_7^E \cdot Exp_i^E + \sum_{A,S,Y} \gamma_{ASY} D_{Age_i=a} \cdot D_{Sex_i=S} \cdot D_{Year_i=Y} + \epsilon_{7i} \qquad (7)$$

. Above, exposure (Exp_i^E) is measured in years between birth and age E in which community deworming took place. I discuss this as a specification and robustness test in Section 5.2.

5 Results

Results in this study are clear enough that they can be measured without aggregating cohorts at all, following Equation 2. For birth cohorts in which deworming arrived before age one for children in some communities, but afterwards for children in other communities, this estimation can be accomplished by simply considering indicators for the original Miguel and Kremer study arms, and regressing outcomes on these indicators within-cohort (including a data collection year indicator) to quantify the effects. I begin by documenting effects on a measure of general non-verbal reasoning: correct answers to a series of visual puzzles from Raven's Progressive Matrices. Of all the cognitive measures included in this study, performance on Raven's Matrices is the most closely related to innate intelligence.

This is demonstrated in Figure 1. In Panel A, I explain the alignment of deworming timing to birth cohorts, to clarify which cohorts permit the relevant within-cohort comparisons. In Panel B1, I carry out those comparisons and show them using shaded bars; in Panel B2, I aggregate the effects to form a single coefficient estimate.[19] The effect of deworming spillovers early in life on nonverbal reasoning ten years later, measured in this very simple framework, aggregates to just under 0.3 standard deviations across all cohorts; this is larger than the impact of many education interventions.

The within-cohort estimation strategy makes transparent that because of the original Miguel and Kremer randomized phase-in design, within-cohort estimation of effects in the present study treat the last arm of their study to start deworming ("Group 3") as the comparison group. If children in that group of communities were systematically different from those in the others, the results I show might simply be spurious. However, the estimation strategy lends itself to immediate falsification tests in relation to this hypothesis: checking whether there are systematic differences between the original study arms in cohorts where the absence of differential early deworming suggests that there should not be. As shown in Panels B1 and B2, differences are uniformly larger and more positive in the true measurement of effects than in the falsification tests. Comparing true tests of this paper's hypothesis with their falsification analogs, two out of three low-power within-cohort two-arm true tests are statistically significant in the estimation of Equation (2); zero of seven analogous falsification tests are significant. These falsification tests

[19] A full set of all pairwise treatment-arm-within-cohort comparisons is provided in Appendix Figure A1.

serve two purposes: they provide additional certainty that the original randomization yielded balanced study arms; and whether or not it did, reinforces that the present study has an additional source of identification beyond the original one. Here, treatment is defined by the interaction of the Miguel and Kremer study arms with birth cohorts.

While this figure provides a direct window into the patterns in the data, results from estimating Equation 6 in a standard regression framework are shown in Tables 2 and 3. In Table 2, I report the estimated the β_6^1 coefficient, so that community deworming before (but not including) age 1 is compared with community deworming after (but not including) age 1. Each row in the table reports $\hat{\beta}_6^1$ from a separate regression for a different outcome variable, Y_i. The effects are striking: community deworming before a child's first birthday brings about a 0.2-standard-deviation improvement in performance in non-verbal reasoning (Raven's Progressive Matrices), a decade after the intervention. Estimated effects on vocabulary measures are similar in magnitude, but not always as significant; effects on memory are not statistically distinguishable from zero. A summary measure, the first principal component of all six cognitive measurements, also shows a roughly 0.2-standard-deviation effect.

One way to benchmark these effects is to compare them to the cross-sectional association between grade in school and cognitive measures; these relationships are shown in Appendix Tables A4 and A5. An additional grade in school is associated with an increase of roughly 0.4 standard deviations in the overall (first principal component) measure, and an increase of roughly

0.25 standard deviations in general reasoning (Raven's Matrices). Considering the impact of early childhood deworming spillovers on the first principal component and reasonsing measures, I take the ratio of coefficients in Table 2 to those in Appendix Tables A4 and A5. Thus, the effects of early deworming spillovers that I document are comparable to between 0.5 and 0.8 additional grades in school. That Raven's Matrices are so responsive to the intervention suggests that even mild disease burdens early in childhood can alter cognitive development. One of the key issues in the child development literature is the decreasing plasticity of physiological and neural development with age. The early age at which spillover effects of community deworming can impact child cognition has not been documented before, and may shed light on child development more generally.

While these cognitive effects are robust to a number of specifications, the effect of community deworming spillovers on height, height-for-age, and stunting all appear statistically indistinguishable from zero. These estimates may be thought of as lower bounds, because even respondents in the excluded (comparison) group lived in communities that received treatment starting when they were aged two and older, and thus still may have experienced some beneficial effects.[20]

The absence of effects on stature is less surprising than it might appear at first glance, for at least two reasons. First, a well-known effect of reducing worm infections is the concomitant reduction of anemia. This, for example,

[20]Extreme stunting, defined as height-for-age Z score below -3, occurs with a frequency of roughly 4 percent in the sample. As with other measures of stature, there is also no discernible effect on extreme stunting.

was demonstrated in Miguel and Kremer (2004), Table V. However, while hemoglobin levels may affect cognition, they do not directly affect stature. Second, only 22 percent of the population in this study experiences stunting; other populations are not so lucky. For example, among children over 30 months old in a deworming study in Zanzibar, 41 percent were stunted - a rate almost twice as high as in the present study (Stoltzfus, et al., 2004).[21] Extreme malnutrition thus does not appear to be the main problem this population faces, and as such, eliminating worm infections is less likely to have dramatic impacts on stature.

5.1 Heterogeneity and mechanisms

To help untangle the mechanisms behind this large effect on cognition, I consider different subpopulations in Table 3. I begin in the first column by repeating the specification shown in earlier tables, for reference. No matter what the mechanism, one might expect the spillovers to be larger within a household where older siblings receive treatment at school than in a household without such older siblings. Respondents were generally not certain of the ages of their older siblings, but as a simple rule, I consider those with at least three older

[21] At first glance, the Demographic and Health Surveys from Kenya appear to disagree with the stunting rate I measure: Table 11.1 of the 2008-2009 DHS report shows that 35.3 percent of Kenyan children are stunted; 34.2 percent in Western Province are stunted (Kenya National Bureau of Statistics 2010). However, the underlying data reveal that the rate of stunting in the two DHS clusters nearest to the site of the present study is actually 21.2 percent (author's calculations): almost exactly the same level I measure in this original data collection. Though the precision of averages based on only a few DHS clusters is low, one can marginally reject (at the 10 percent level) that the rate of stunting in these two clusters is equal to the national or provincial means of 34 or 35 percent; intra-cluster correlation in stunting rates is significantly different from zero, even after accounting for province fixed effects. Thus, calculations based on DHS data do partially corroborate the stunting rates I observe.

siblings attending the same primary school to have had a sibling in school at the time of the deworming campaign. When the sample is restricted to this group, shown in column 2, the effect size nearly doubles.

This raises the question of whether there are any spillovers for children who did not have siblings in the primary school that participated in deworming. If so, an epidemiological mechanism is supported; if not, a behavioral or financial within-household mechanism might be more plausible. Again, because of the imprecision of responses, I consider only respondents who did not have any older siblings attending the relevant primary school as the subsample best suited to answer this question; estimates are shown in column 3. The effect is similar in magnitude to that of the full sample, and while for Raven's Matrices it is statistically significant, it is not for the first principal component of all cognitive measures. With this, evidence leans in favor of an epidemiological mechanism: fewer worms in the community mean fewer infections in early childhood for these respondents.[22]

To further explore the sibling sample in column 2, I divide that group into those who had more female than male older siblings at the same primary school in column 4, and vice-versa in column 5. Sample size is quite small at this point, and standard errors widen, but it appears that the benefit is largest for those with older sisters at the primary school rather than older brothers. This may reflect the relative frequencies with which girls and boys are tasked

[22]One can also test for effects at varying distances from Lake Victoria; results suggest effects both within 5km of the lake and beyond 5km from the lake, making geohelminths likely to be involved in the mechanism rather than exclusively schistosomes. This intuitively aligns with the more localized spillovers one would expect from soil-transmitted worms, as discussed in Miguel and Kremer (2004).

with caring for younger siblings: care for infants and toddlers by older female children is common in this study area, where the predominant ethnic group is Luhya. Weisner, et al. (1977) and subsequent authors have discussed how this care pattern is common across many cultures, and is salient in Kenya in particular. Weisner and co-authors call out the Luhya as a culture in which this pattern is especially strong: a high fraction of interactions among children are caretaking interactions with infants, and older female siblings were more than twice as likely to act as caretakers for infants as were their male counterparts (ibid., p. 175).

This pattern suggests that those who are in frequent physical contact with infants could be a key channel through which worms, or their absence through treatment, can affect infants and toddlers. Thus, this pattern provides further evidence in favor of an epidemiological mechanism. An alternative story could be that of a household budget constraint, in which healthier, dewormed older siblings would loosen budget constraints through reduced direct and indirect health costs, thereby freeing total resources to be devoted to the younger child. But in that story, health costs that determine the budget constraint would arise from both male and female older siblings. The pattern in the data seem to provide evidence against this story, as dewormed older male siblings appear not to have an impact on the younger ones in this sample.

Finally, since a number of shocks and interventions in developing countries have been shown to have gender-specific impacts, I split the sample according to the sex of the respondent in columns 6 and 7. The coefficients are not appreciably (or statistically) different for boys and girls, though they are

slightly higher for girls. This suggests that there is no substitution towards or away from any other nutrition or stimulation input that would be gender-specific, as is sometimes seen for interventions at later ages (Pitt, Rosenzweig and Hassan 2012).

5.2 Variations on the empirical specification

In Table A1, I show a variety of specifications based on variations of Equation 5. In the first seven columns, I vary the value of C, the age before which deworming took place, from negative two to positive four.[23] For the indicators of subsequent deworming, I set C_H to four, and as C increases, the number of terms in the summation of later deworming indicators decreases.

Several regularities appear across the first seven columns of the table. First, the coefficients on deworming before age C cannot be statistically distinguished from one another for the first five columns (*before age -2* through *before age 2*), but after that, the coefficients lose significance and fall in magnitude. Either β_4^3 or β_4^4 (columns 6 and 7) can be statistically rejected as being equal to any of the coefficients from earlier columns. Second, the latest exact age indicator to be statistically significant is always age zero (in columns 1 through 3); conversely, in the first four columns of the table, the earliest coefficient to be statistically insignificant because of its lower magnitude is always that for deworming at exactly age one.

By including as many later deworming indicators as I do in columns 1 through 7, however, I sacrifice statistical power by reducing the size of the

[23]Deworming "before age 0" means deworming before birth; "before age -1" means more than one year before birth; and so on.

omitted group. Because of the two patterns described above, I repeat the specification from column 4 ($C = 1$ year) in columns 8 through 11, but decreasing C_H across the columns, until the specification in column 11 is simply that of Equation 3. The specification in column 10 yields the coefficients shown earlier in Table 2.

An alternative reading of Table A1 is that rather than narrowly favoring the main specification, it suggests one where children dewormed in their first year of life receive some fixed fraction of the treatment effect (perhaps half). This pattern would be consistent with either some misreporting of age, or with a gentle tapering of the most sensitive period for this effect; the data here unfortunately do not allow these possibilities to be empirically distinguished from one another.

Yet a different approach is that of Bleakley (2007), who considers years of deworming exposure. In his case, he interacts the program exposure with baseline intensity of hookworm infection, and considers up to the first 19 years of life for exposure. In this case, baseline rates may be noisily measured, so a simple measure of exposure is used in Equation 7. This is the number of years of community deworming that took place between the participant's birth and age E, for which I consider $E = 1$, $E = 2$, and $E = 3$. If deworming spillovers after year E of life have important effects, the equation estimates only a lower bound. A fall in coefficient magnitude moving from $E = 1$ to $E = 3$ would be consistent with the first year mattering more than subsequent years. Indeed, in Appendix Table A6, this is exactly what I find.

In summary, then, the evidence here corroborates the results in the earlier

sections: community deworming prior to age 1 brings about a 0.2-standard-deviation improvement in performance on Raven's Matrices later in life; deworming at age 1 may have some positive effect, but smaller, though this could simply be due to noisy measurement of child age; deworming after age 1 cannot be statistically distinguished from deworming much later.

5.3 Discussion of results

Others have also found effects of deworming on cognition, though typically only in the short term. An observational study by Jukes, *et al.* (2002) investigated the relationship between cognitive function and helminth infections among Tanzanian schoolchildren, and found that after controlling for potential confounds, heavy schistosome infection was associated with lower performance on tests of short-term memory, reaction time, and information processing. A double-blind medical trial by Nokes, *et al.* (1992) found that the administration of albendazole led to immediate gains in memory skills in a population of Jamaican schoolchildren infected with whipworm and roundworm, and an experimental de-worming study with Tanzanian schoolchildren in the same region as the 2002 observational study also found cognitive gains in response to de-worming (Grigorenko, *et al.* 2006). Bleakley (2007) provides historical anecdotes that corroborate these patterns.

That I find effects mainly on reasoning–and to some extent, vocabulary–rather than memory speaks to the differences between slowed cognitive development and the more immediate cognitive impairments brought about by concurrent disease. Memory improves with age, but seems to depend less on

health in early child development. Reasoning, however, shows a long-term response to improved health in early childhood. That stature is not affected suggests that worms do not cause severe nutritional deprivation in early childhood; the low intensity of worm infections at this age is in accord with this evidence.

Though the evidence here is consistent with an effect that was largest for those under one year of age at the time of community deworming, the empirical variations explored in section 5.2 do not completely rule out a more gentle decline in effect with age, nor do they rule out any benefit received by all study participants for having any deworming in childhood. Croke (2014), for example, finds long-term benefits on mathematics and English scores for children who were roughly between 1 and 7 years old at the time of direct deworming. This could either be an effect that is above and beyond the one that I measure here, or both of our studies could be estimating closely related effects, with noisily-measured age complicating both of our analyses. For Croke, the intensity of treatment tapers off for both the youngest children and the oldest children in the study, so it is difficult in that setting to separate a relatively more sensitive period for intervention from the intensity of treatment that children receive. Bleakley (2007) analyzes a natural experiment, and finds long-term benefits of deworming in the United States that include improvements in school attendance and literacy, though he considers any treatment in the first two decades of individuals' lives. This study adds clear evidence that whatever the benefit of direct deworming, the benefit of a less worm-infected community is felt most strongly by the youngest cohorts.

5.4 Threats to identification

5.4.1 Demographics

Changes in the composition of cohorts in this study that are due to variation in deworming treatment by community could potentially confound the analysis. Changes of this sort could arise if deworming changed mortality rates, leaving disproportionately healthy children as survivors.[24] One could also imagine that if adults adjusted their fertility patterns in response to school-based mass deworming—in either direction—such adjustment might change the interpretation of estimated effects. Bleakley and Lange (2009), for example, document decreases in fertility in response to the Rockefeller Sanitary Commission deworming work in the US South. A simple approach to mortality and fertility is to test whether respondents exposed to community deworming from birth have more or fewer siblings than those who were exposed only later. An analogous approach is to test whether the actual quantity of age-eligible respondents in each community systematically varies as a function of deworming exposure. Tests of these hypotheses are shown in Table A7. I find no evidence of either pattern using either approach.

5.4.2 Attrition

One could imagine differential health or academic performance inducing different attendance rates among those whose communities initially experienced deworming at different times. Such a pattern could bias coefficient estimates

[24]Note, however, that a mortality mechanism does not have empirical support from studies that have looked at it directly; see Awasthi, Peto, Read, Richards, Pande, Bundy and DEVTA Team (2013).

if different treatment groups attrit differentially from the sample via absenteeism. However, comparing the number of students on the schools' enrollment rosters to the number of students interviewed by enumerators reveals no empirical evidence of differential attendance rates by treatment group, so I do not view this as a serious threat to the analytical design.

6 Cost-effectiveness

The policy implications of a program's impact depend on its cost-effectiveness. As has been well-established, the costs of deworming a child directly are quite low, at around $0.59 per pupil per year (as discussed by Baird, et al., 2014). This paper examines only spillover impacts, so the effects documented here may be thought of as additional benefits beyond those documented by Baird, et al., without any additional costs.

To quantify the benefits here, we must extrapolate (as Baird, et al., do) to earnings impacts of early changes in cognition. For this, I rely on a pair of studies: one that documents an early childhood intervention changing cognitive measures, in a developing country, and a followup that shows changes in wages in adulthood. Grantham-McGregor, et al. (1997) found that an early childhood stimulation intervention in Jamaica increased performance on a variety of measures several years later, when the study participants were 7 to 8 years old. In a follow-up, Gertler, et al., find that this intervention eventually increased wages by 25 percent.

In Table 4, I compare coefficients on four cognitive measures in the Grantham-

McGregor, et al. (1997) study to the coefficients in the present study. Taking their ratio, and multiplying by the 25 percent wage increase found to result at age 22 by Gertler, et al. (2014), I arrive at potential extrapolated percentage wage increases listed in the last column. The figures range from 12.5 percent to 83.3 percent: even the smallest of these is quite substantial.

In order to place this in dollar terms, I must also set this in relation to typical earnings. For this, I adopt the approach of Baird, et al.; they estimate net present values of benefits of deworming from a public finance perspective. This task includes calculation of the net present value (NPV) of typical lifetime earnings for an individual in western Kenya, for which they arrive at a figure of $1509.96.[25]

Though many of their effects appear to operate through years of schooling and health, here the effects appear to operate through cognition. As such, the simplest calculation does not involve any government-borne costs of additional schooling. The question is, by what fraction will lifetime earnings rise, and how much additional revenue will the government eventually collect as a consequence? Multiplying the NPV of earnings by the most conservative percentage from Table 4, this is an additional NPV of just over $180 in earnings. Scaling that down by the 16.5 percent tax rate to calculate government revenues, I find an additional NPV of government revenue of just over $30 per child benefiting from spillovers.

A simpler and potentially more conservative approach is to consider the

[25] One could, instead, extrapolate lifetime earnings using Kenya's GNI per capita as reported by the 2014 World Development Indicators. At $860 in current (non-PPP) dollars (World Bank 2014), the relevant steps would yield a larger total figure. I use the Baird, et al., approach to remain conservative.

effects in terms of years of schooling. As discussed above, the cognitive effects appear comparable to half a year of school. If the returns to education are, conservatively, six percentage points per year (Duflo 2001, Card 2001), and are due exclusively to the cognitive human capital that is accrued through schooling, then an appropriate calculation is the same as the one above, but with three percent rather than 12.5 percent of the NPV of lifetime earnings. The result is then a more modest $7.50 gain for public coffers.

While this figure should be accurate for each child receiving spillovers, the last piece of this calculation is to consider how many such children there are for each child receiving deworming medication. This depends on the structure of the deworming program, but a simple approach is to consider a population in which all cohorts are of equal size. In this setting, an ongoing deworming program deworms approximately eight cohorts of primary school children each year, and this spillover benefit is received by the birth cohort that year. This scales down the benefit by a factor of eight, per year per pupil dewormed. For comparison to the cost-benefit calculus of the 1998-2001 Primary School Deworming Program in Kenya, this figure should be scaled back up by the 2.41 years of deworming, on average, that each dewormed pupil received. This still produces conservative figures of between $2.25 and $9 of additional benefit per pupil dewormed in the original deworming program, using either the Grantham-McGregor and Gertler papers to extrapolate or simply using the returns to schooling. These benefits increase the already substantial public finance benefits of roughly $13 per pupil dewormed (shown by Baird, et al.) by 17 or 70 percent, depending on the method of extrapolation - all benefits

reaped from a roughly $1 per person subsidy for deworming medication.

7 Conclusion

In this study, I measure the effect of deworming spillovers during early childhood. I find improvements in cognitive performance equivalent to between 0.5 and 0.8 years of schooling. Effects are nearly twice as large for children with an older sibling likely to have received deworming medication directly. This bolsters theories of sensitive periods for cognitive development, and provides evidence that an inexpensive intervention can benefit children immensely at this time. In light of the patterns of heterogeneity seen in the data, the most plausible explanation appears to be an epidemiological spillover, transmitted via older siblings and neighbors to infants in dewormed communities.

In relation to deworming specifically, this evidence lends further support to expanding initiatives worldwide that treat deworming en masse. Taken together with the recent work of Baird, et al., (2014) and Croke (2014), this study helps paint a complete picture of long-run benefits of deworming in developing countries. This expanding body of evidence has already led to real policy initiatives. In Kenya, for example, national deworming was undertaken in 2009, and began recurring annually in 2012. Infection levels in Kenya have dropped substantially since the original Miguel and Kremer study began in 1998 (Mwandawiro, et al., 2013). However, high-intensity infections remain prevalent around Sub-Saharan Africa and the world.

More broadly, the evidence that early childhood health shocks have ramifi-

cations for subsequent human capital in a variety of forms builds on, and gives empirical substance to the models of Grossman (1972), Cunha and Heckman (2008), and their successors. The present study, demonstrating the presence of early childhood deworming spillovers, in essence asks whether this inexpensive health intervention can act as an input to early cognitive skills. As in these and other models of human capital formation, the present findings do not rule out the value of interventions later in life. In fact, a successful early childhood intervention such as this one might well be complementary to the more frequently-studied interventions that become relevant later in the life cycle. As elements of policy, however, the cost-effectiveness of deworming in highly worm-infected settings, and potentially that of other early-life health and nutritional interventions, will be difficult to match.

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Figure 1: Identification strategy, and key comparisons in raw data

Panel A defines "treatment" for this study. Each column represents a birth cohort; each row, a group of communities where deworming began in a specific year. Shading corresponds to treatment status, defined in terms of child age when deworming began, A_{id}: dark gray indicates younger than one ("treated"); white indicates older than one ("untreated"); and light gray indicates one year old.

Panels B1 and B2 show the within-cohort differences between "treated" and "untreated" groups in terms of scores on Raven's Matrices questions, standardized within the sample.

Panel B1 shows comparisons for cohorts aligned with those in Panel A. Dark gray bars indicate the treatment effect, the within-cohort difference between "treated" and "untreated" groups, as defined above. White bars indicate "placebo tests," in which two groups in a given cohort have the same treatment status as defined in Panel A. An "X" symbol appears wherever the difference would involve a group categorized as neither "treated" nor "untreated." A full set of pairwise comparisons is shown in the appendix; all comparisons are combined in a regression framework and are discussed further in the text.

Panel B2 shows the result of aggregating these pairwise comparisons in a simple regression: the dark bars yield to a coefficient of 0.294 (p=0.002), while aggregating the light bars yield a coefficient of 0.015 (p=0.82), as shown.

In Panels B1 and B2, the only control is a data collection year dummy (interacted with cohort dummies in B2), and the sample is restricted to non-migrants. 95 percent confidence intervals are shown, clustered by school-cohort.

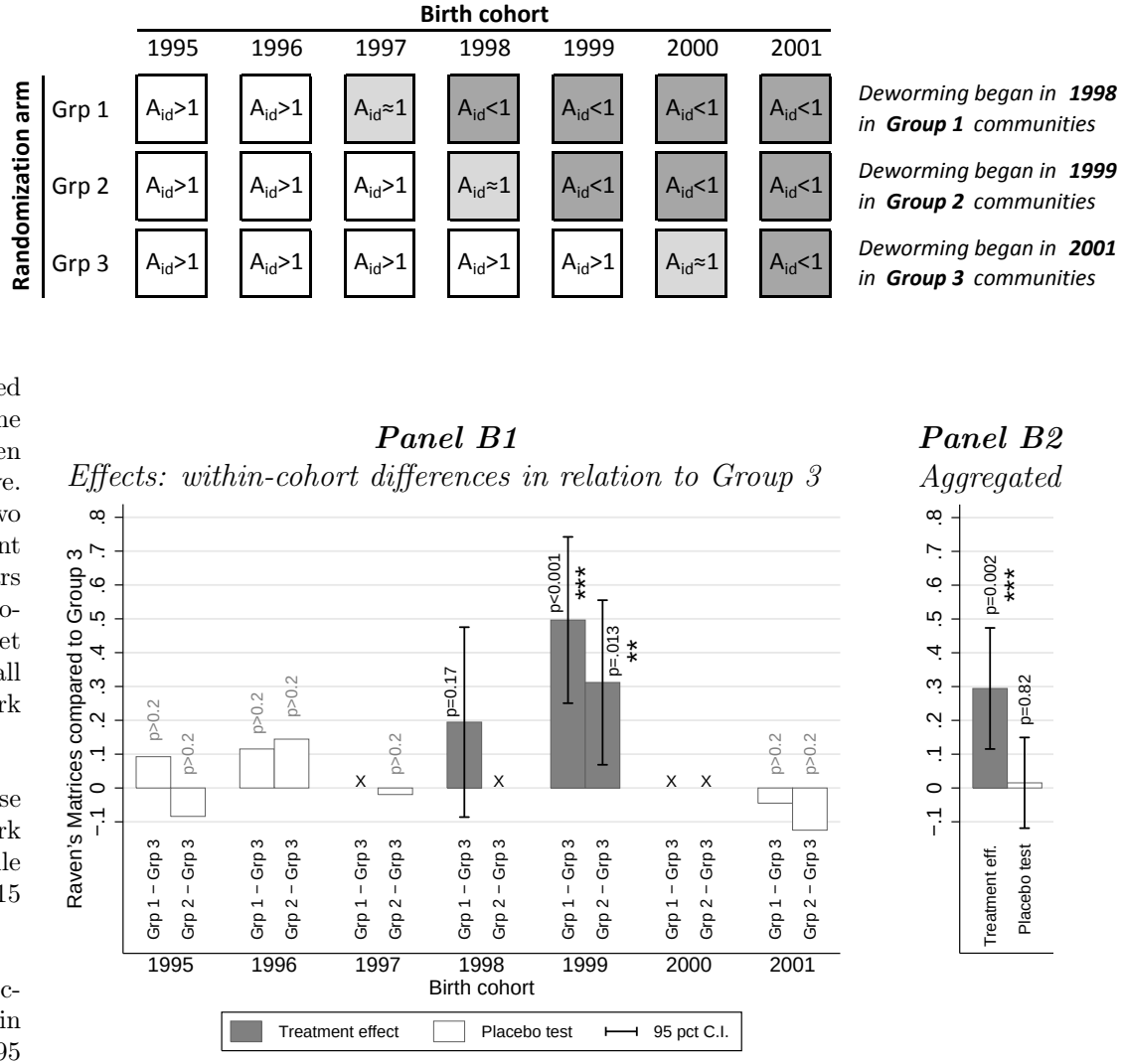


Table 1: Summary Statistics

Panel A: Characteristics, unconditional			
Characteristic	Mean	Standard Dev.	N
Age	11.486	(1.951)	21870
Female	0.488	(0.500)	21844
Height (cm)	141.545	(12.656)	21429
Ever migrated	0.284	(0.451)	21870
Panel B: Characteristics, conditional on non-migration and complete data			
Age	11.397	(1.954)	15633
Female	0.473	(0.499)	15633
Height (cm)	140.970	(12.712)	15322
Stunting (WHO 2007 HAZ< -2)	0.216	(0.411)	15435
Older siblings at same school	1.452	(1.594)	15633
At least 3 such siblings	0.225	(0.417)	15633
No such siblings	0.370	(0.483)	15633
Panel C: Deworming cohort, conditional on non-migration and complete data			
Deworming before age -1	0.162	(0.368)	15633
Deworming starting at age -1	0.115	(0.319)	15633
Deworming starting at age 0	0.128	(0.334)	15633
Deworming starting at age 1	0.146	(0.353)	15633
Deworming starting at age 2	0.152	(0.359)	15633
Deworming starting after age 2	0.298	(0.457)	15633
Panel D: Cognitive data, conditional on non-migration and complete data			
Verbal Fluency: Foods	9.265	(2.957)	2474
Verbal Fluency: Animals	8.874	(3.230)	2474
Vocabulary: highest PPVT level	6.078	(3.350)	2471
Reasoning: Raven's Matrices	3.640	(1.948)	2473
Memory: Digit Span Forwards	3.358	(1.744)	2455
Memory: Digit Span Backwards	0.954	(1.239)	2418
Panel E: Characteristics, conditional on non-migration and ***cognitive*** *data*			
Age	11.555	(1.926)	2584
Female	0.467	(0.499)	2584
Height (cm)	141.856	(12.903)	2561
Stunting (WHO 2007 HAZ< -2)	0.213	(0.410)	2408
Older siblings at same school	1.424	(1.614)	2584
At least 3 such siblings	0.219	(0.413)	2584
No such siblings	0.384	(0.486)	2584

Table 2: Main effects: community deworming before age one

Outcome	**Effect**
Raven's Matrices	0.220***
	(0.078)
PPVT Level	0.154
	(0.096)
Verbal fluency	0.190**
	(0.090)
Memory: digit span forwards	0.134
	(0.095)
Memory: digit span backwards	0.021
	(0.087)
All cognitive: First principal component	0.209**
	(0.097)
All cognitive: Normalized sum	0.209**
	(0.097)
Height (cm)	0.177
	(0.300)
Height-for-age z-score	0.025
	(0.044)
Stunting (HAZ<-2)	0.007
	(0.016)

In the table above, the excluded group comprises the cohorts whose communities were dewormed during their second year of life or later. Each coefficient comes from a separate regression of the indicated outcome on indicators for the age at deworming. Standard errors are clustered at the school-cohort level; gender×age×data collection year fixed effects are included. All cognitive outcomes are standardized (variance=1). Only non-migrants are included in this analysis.

Table 3: Effects of community deworming before age one: different subpopulations

Subpopulation: / Outcome:	[1] Full sample	[2] With older siblings[a]	[3] Without older siblings[a]	[4] Female siblings[b]	[5] Male siblings[b]	[6] Female[c]	[7] Male[c]
Raven's Matrices	0.220***	0.423**	0.250**	0.842***	0.074	0.224**	0.214*
	(0.078)	(0.164)	(0.119)	(0.267)	(0.199)	(0.113)	(0.124)
All cognitive: First PC	0.209**	0.396**	0.173	0.771***	0.247	0.241**	0.175
	(0.097)	(0.159)	(0.132)	(0.254)	(0.237)	(0.120)	(0.134)
All cognitive: Normalized sum	0.209**	0.383**	0.177	0.752***	0.259	0.238**	0.179
	(0.097)	(0.159)	(0.133)	(0.256)	(0.235)	(0.121)	(0.134)
Observations	2412	541	910	240	228	1129	1283

In the table above, the excluded group comprises the cohorts whose communities were dewormed during their second year of life or later. Each coefficient comes from a separate regression of the indicated outcome on indicators for the age at deworming. Standard errors are clustered at the school-cohort level; gender×age×data collection year fixed effects are included. All cognitive outcomes are standardized (variance=1). Only non-migrants are included in this analysis. Column [1] repeats the specification shown in Table 2, for reference. (a) In column [2], the sample is restricted to respondents who have at least three older siblings who attended the same primary school; in column [3], it is restricted to those for whom no older siblings attended the same primary school. (b) In column [4], the restriction is similar to that in column [2], but with the added restriction that more female than male older siblings attended the same primary school; in column [5], it is reversed: more male than female older siblings attended the same primary school. (c) In columns [6] and [7], the original sample is simply split according to the gender of the respondent.

Table 4: Extrapolating benefits of early cognition interventions

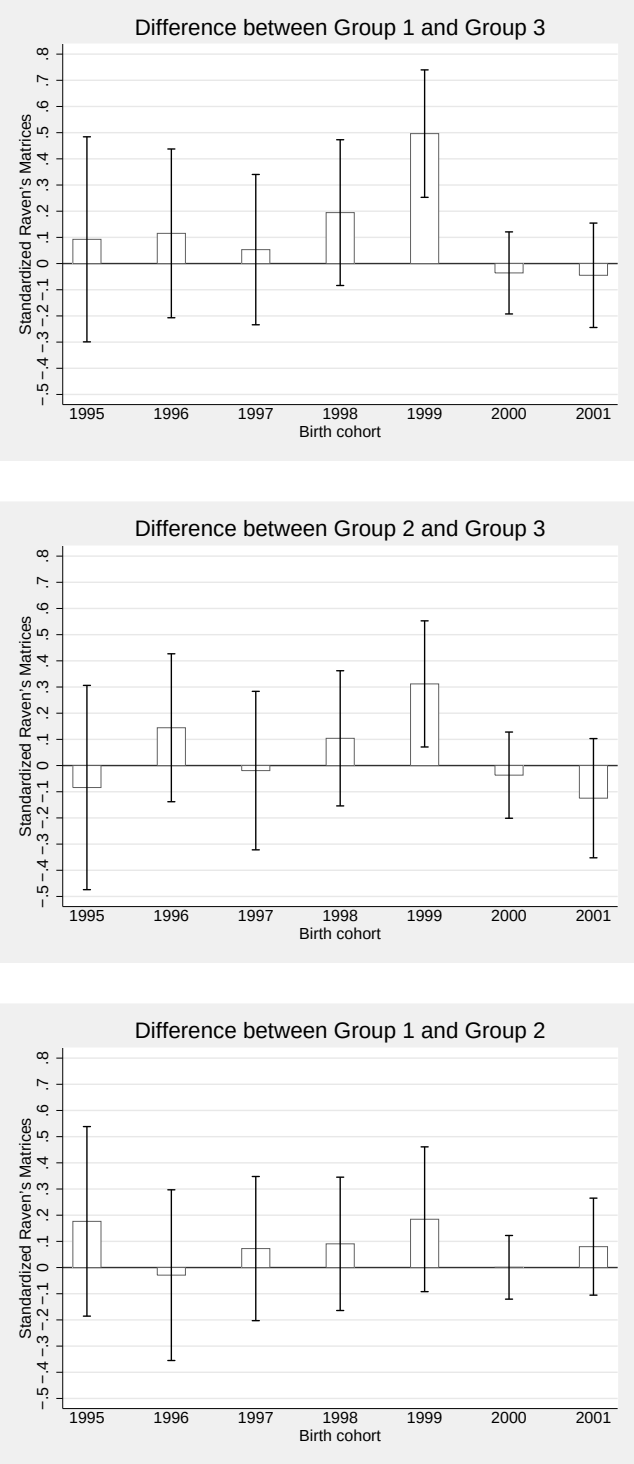
Cognitive measure	**Coefficients**		**Ratio**	**Wage change**
	1997 study (point est.)	**This study** (point est.)		(extrapolated pct.)
Raven's Matrices	0.86 questions	0.43 questions	0.497	12.5
Forward digit span	0.20 digits	0.12 digits	0.59	14.8
PPVT vocabulary	3.0 words	3.4 words	1.13	28.3
Verbal Fluency	0.3 answers	1.0 answers	3.33	83.3

The 1997 study is Grantham-McGregor, Walker, Chang and Powell (1997). Note that the measure of verbal fluency used by Grantham-McGregor, et al., included three categories of answers, each for one minute, while the present study only included two. As such, an alternative calculation would scale the coefficient in the present study by 1.5 for better comparability, yielding a coefficient ratio of 5, and a predicted wage increase of 125 percent.

A Appendix

FOR ONLINE PUBLICATION

Figure A1: Full set of comparisons between treatment arms



Note: Bars represent 95-percent confidence intervals.

Table A1: Locating the critical period: different simple specifications

	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]	[9]	[10]	[11]
Deworming before age -2	0.284*	.	.	.	.	.	.	.	.	.	.
	(0.148)										
Deworming before age -1	.	0.245*	.	.	.	.	.	.	.	.	.
		(0.134)									
Deworming before age 0	.	.	0.291**	.	.	.	.	.	.	.	.
			(0.132)								
Deworming before age 1	.	.	.	0.28**	.	.	.	0.273***	0.22**	0.22***	0.137**
				(0.127)				(0.105)	(0.092)	(0.078)	(0.066)
Deworming before age 2	.	.	.	.	0.229*	.	.	.	.	.	.
					(0.121)						
Deworming before age 3	.	.	.	.	.	0.11	.	.	.	.	.
						(0.112)					
Deworming before age 4	.	.	.	.	.	.	0.081	.	.	.	.
							(0.098)				
Deworming age -2	0.235*	.	.	.	.	.	.	.	.	.	.
	(0.135)										
Deworming age -1	0.316**	0.319**	.	.	.	.	.	.	.	.	.
	(0.136)	(0.136)									
Deworming age 0	0.27**	0.267**	0.273**	.	.	.	.	.	.	.	.
	(0.132)	(0.132)	(0.131)								
Deworming age 1	0.193	0.194	0.196	0.194	.	.	.	0.187*	0.139	0.139*	.
	(0.125)	(0.125)	(0.125)	(0.125)				(0.098)	(0.086)	(0.077)	
Deworming age 2	0.051	0.051	0.05	0.049	0.046	.	.	0.042	-0.0005	.	.
	(0.118)	(0.118)	(0.118)	(0.118)	(0.118)			(0.097)	(0.088)		
Deworming age 3	0.09	0.09	0.091	0.092	0.09	0.058	.	0.085	.	.	.
	(0.104)	(0.104)	(0.104)	(0.104)	(0.104)	(0.103)		(0.089)			
Deworming age 4	0.012	0.012	0.013	0.012	0.021	-0.008	-0.017	.	.	.	.
	(0.13)	(0.13)	(0.13)	(0.13)	(0.13)	(0.129)	(0.127)				
Observations	2472	2472	2472	2472	2472	2472	2472	2472	2472	2472	2472
R^2	0.135	0.135	0.135	0.135	0.134	0.133	0.132	0.135	0.135	0.135	0.133

In the table above, each column represents a separate regression with standardized performance on Raven's Matrices as the outcome variable. In columns [1]-[7], the omitted category is respondents for whom community deworming took place when they were five years old or older. Because this is a relatively small group, columns [8]-[11] show the same estimation as in column [4], but with different omitted categories: community deworming after ages four and older; three and older; two and older; and one and older, respectively. Gender×age×data collection year fixed effects are included in all specifications, all samples are restricted to non-migrants, and standard errors are clustered at the school-cohort level.

Table A2: Cognitive measures: Principal Components

Principal component:	(1)	(2)	(3)	(4)	(5)	(6)
Verbal Fluency: Foods	0.3612	-0.6743	0.0027	0.2230	0.5550	-0.2390
Verbal Fluency: Animals	0.4443	-0.4238	-0.0030	-0.0594	-0.5293	0.5825
Digit Span Forwards	0.3814	0.2288	0.6677	-0.5286	0.2687	0.0693
Digit Span Backwards	0.3875	0.3937	0.2948	0.7742	-0.0915	-0.0117
Vocabulary: PPVT	0.4762	0.0878	-0.2600	-0.2420	-0.4023	-0.6910
Raven's Matrices	0.3870	0.3882	-0.6322	-0.0965	0.4115	0.3481
Explained variance:	0.4665	0.6214	0.7464	0.8482	0.9344	1.0000

Table A3: Cognitive measure correlations

	Fluency: Foods	Fluency: Animals	Digit Span Forwards	Digit Span Backwards	Raven's Matrices	Vocab: PPVT
Foods	1.0000					
Animals	0.5007	1.0000				
Digit Span Forwards	0.2400	0.3389	1.0000			
Digit Span Backwards	0.2323	0.3183	0.3778	1.0000		
Raven's Matrices	0.2218	0.3014	0.2742	0.3477	1.0000	
PPVT	0.3490	0.5204	0.3989	0.3899	0.5083	1.0000

Table A4: Cognitive performance (first principal component, normalized) as a function of observables

	All			Boys			Girls		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Grade	0.451***	0.382***	.	0.459***	0.407***	.	0.449***	0.355***	.
	(0.011)	(0.007)		(0.015)	(0.009)		(0.016)	(0.01)	
Age	-0.089***	.	0.261***	-0.069***	.	0.292***	-0.118***	.	0.226***
	(0.011)		(0.009)	(0.015)		(0.012)	(0.015)		(0.012)
Constant	-0.872***	-1.607***	-3.021***	-1.071***	-1.652***	-3.369***	-0.606***	-1.554***	-2.624***
	(0.095)	(0.032)	(0.103)	(0.134)	(0.043)	(0.146)	(0.133)	(0.048)	(0.145)
Observations	2583	2583	2585	1372	1372	1373	1203	1203	1204
R^2	0.555	0.543	0.254	0.582	0.576	0.287	0.532	0.51	0.218

Table A5: Cognitive performance (normalized) as a function of observables

	Outcome											
	Vocabulary: PPVT		Verbal fluency: Foods		Verbal fluency: Animals		Memory: Digit Span Forwards		Memory: Digit Span Backwards		Reasoning: Raven's Matrices	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Grade	0.372***	.	0.196***	.	0.279***	.	0.219***	.	0.222***	.	0.247***	.
	(0.007)		(0.009)		(0.008)		(0.009)		(0.009)		(0.009)	
Age	.	0.261***	.	0.143***	.	0.212***	.	0.118***	.	0.139***	.	0.17***
		(0.009)		(0.01)		(0.009)		(0.01)		(0.01)		(0.009)
Constant	-1.565***	-3.012***	-0.81***	-1.642***	-1.169***	-2.444***	-0.918***	-1.363***	-0.936***	-1.608***	-1.034***	-1.960***
	(0.032)	(0.101)	(0.042)	(0.112)	(0.039)	(0.107)	(0.042)	(0.115)	(0.043)	(0.115)	(0.041)	(0.111)
Observations	2661	2665	2664	2667	2664	2667	2633	2635	2591	2593	2663	2667
R^2	0.519	0.255	0.145	0.078	0.292	0.168	0.179	0.052	0.184	0.072	0.227	0.107

Table A6: Linear effects of years of deworming before different ages

Outcome:	Before age 1 [1]	Before age 2 [2]	Before age 3 [3]
Raven's Matrices	0.137**	0.110***	0.076**
	(0.066)	(0.039)	(0.031)
All cognitive: First PC	0.164**	0.105**	0.069*
	(0.077)	(0.048)	(0.037)
All cognitive: Normalized sum	0.164**	0.105**	0.069*
	(0.078)	(0.048)	(0.036)
Observations	2412	2412	2412

The table above presents an alternative specification. Here, each coefficient comes from a separate regression of the indicated outcome on years of community deworming between a child's birth and a particular age (Equation 7). Thus in column 1, the independent variable takes the value 0 or 1; in column 2, it is either 0, 1, or 2; and in column 3, it ranges from 0 to 3. As usual, standard errors are clustered at the school-cohort level; gender×age×data collection year fixed effects are included; all cognitive outcomes are standardized (variance=1); only non-migrants are included.

Table A7: Testing for fertility or mortality responses to deworming

	Sibling N [1]	Respondent N [2]
Deworming before 1 (main specification)	0.056	0.062
	(0.055)	(0.799)
Deworming before birth (alternative for fertility)	0.061	0.862
	(0.049)	(0.832)
Observations	15630	1740

The table above presents tests of a fertility response to community deworming. In the first column, the outcome variable is the number of younger siblings reported by the respondent. In the second column, observations have been aggregated at the level of the { data collection year × birth year × gender × migration indicator × school }. Thus, in the second column, the outcome is simply the count of observations in these bins. The first row presents the same specification as elsewhere in the paper, showing an indicator for deworming arriving in the community in the respondent's year of birth or earlier; the second row presents an alternative specification, using an indicator for whether deworming arrived in the community before the respondent was born. In any of the four cells, a significant coefficient could indicate a change in fertility in response to mass school-based deworming starting in that community. In the first column, standard errors are clustered at the school-cohort level. In the second column, because observations are already aggregated, standard errors are simply heteroskedasticity-robust.