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

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Abstract

I investigate whether a large-scale deworming intervention aimed at primary school pupils in western Kenya had long-term effects on young children in the region, exploiting positive externalities from the program to estimate the impact on younger children who did not receive treatment directly. I find large cognitive effects—equivalent 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 mass deworming was administered through schools, I also estimate effects among children who were likely to have older siblings in school to receive the treatment directly; in this subpopulation, effects are nearly twice as large. (*JEL:* I12, I20, I38, O1, O12, O15)

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1 Introduction

Shocks in early childhood can permanently damage 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 negative 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 (Paneth and Susser 1995, Almond and Mazumder 2005). More generally, important times during early childhood for cognitive development are referred to as "critical" and "sensitive" periods (Knudsen 2004). Yet because of the demanding longitudinal data required, very few studies have successfully documented these patterns; fewer still are able to establish causal relationships between external influences early in life and long-term outcomes.

However scant, the available evidence suggests that the phenomenon is real, and that the effects are dramatic. One panel study shows that reading test quartiles at age seven predict 20 percent differences in adult wages in Great Britain (Currie and Thomas 1999); another, that performance at age four on a delay-of-gratification task is a strong predictor of high school SAT score (Shoda, Mischel, and Peake 1990). Though these patterns survive the inclusion of important statistical controls, they may only illustrate simultaneous causation. Perhaps these results only appear because the same forces that determine performance in early childhood—parenting and genetics, for example—continue to determine outcomes in the decades that follow. One way to resolve this issue is to control for covariates throughout childhood; a paper using the Panel Study of Income Dynamics does this, showing that parental income in the first several years of a child's life matter much more for that child's eventual adult income than do shocks after the child's fifth birthday (Duncan, Ziol-Guest, and Kalil 2010). 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 (Alderman,

Hoddinott, and Kinsey 2006). In contrast, shocks that occur later in life do not appear to have significant impacts on long-run outcomes.

Though extreme shocks periodically affect a small fraction of the population, much of the world is afflicted by mild forms of disease and deprivation. Less is known about whether such conditions, more easily addressed by human intervention, can permanently change outcomes in this way. 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. 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 outside the reach of most government programs.

In this paper, I consider a non-lethal disease very 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 are almost never fatal, but they 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 (Bundy, *et al.* 2009). 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 reviewed by Albonico, *et al.* (2008) also document child health improvements in response to early childhood deworming. Despite promising short-term results, no study to date has shown whether early childhood deworming can have lasting benefits.

In this paper, I present the first evidence on the long-term effects of reducing helminth infection in early childhood by exploiting externalities from a randomized deworming intervention in Kenya. Though it was aimed only at school-age children, this kind of community-based deworming has large epidemiological spillovers both on other children (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 community to children from the same cohort in untreated communities.

I find large effects on cognitive performance equivalent to half a year of schooling, as well as improvements in physical stature, more than ten years after the original intervention. Effects are strongest among those who were likely to have an older sibling in school at the time of the original intervention, as one might expect from an epidemiological perspective. My results support the theories that sensitive periods in early childhood are essential for physical and cognitive development, and that inexpensive actions are available that can increase human capital formation for millions of children around the world.

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; Section 5 presents the results of my analysis in light of the existing literature; and Section 6 concludes.

2 Background

2.1 Biology

A handful of helminth species are responsible for infecting between one and two billion people (Hotez, *et al.* 2006): schistosomes (*Schistosoma mansoni*, *haematobium*, and variants); and soil-transmitted "geohelminths:" roundworm (*Ascaris lumbricoides*), whipworm (*Trichuris trichiura*), and hookworm (*Necator americanus* and *Ancylostoma duodenale*). Several of these species are endemic in western Kenya, and though these infections can be addressed inexpensively with existing drugs, they usually go untreated.[1] All of these parasites inhabit parts of the human digestive tract; female worms produce eggs that spread via human excrement.[2] 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

[1] Albendazole and mebendazole are anti-geohelminth medications, effective against hookworm, roundworm, and whipworm. Schistosomiasis is usually treated with a different medication, praziquantel.

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

ingesting a worm egg (often from contact with soil contaminated with feces). Other worms infect via dermal penetration; hookworms often penetrate through the bottom of the foot, while schistosomes enter the skin through lake or river water while part of a person's body is immersed (Bundy, Chan, Medley, Jamison, and Savioli 2001, Mott 2001).

2.2 Past intervention

In this setting, between 1998 and 2001, Miguel and Kremer (2004) randomly phased in deworming drugs to a group of 73 primary schools in western Kenya, in the "Primary School Deworming Project," PSDP. The program both reduced infections and increased school attendance. Only schoolchildren were dewormed, but the authors found large spillovers within the community: 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 reduced parasitic loads in adults who received no medication (Bundy, *et al.* 1990). Thus far, the effects of the intervention in Kenya have included gains of approximately 1cm in height, and 1kg in weight, as well as some preliminary evidence of increased rural to urban migration (Baird 2007). Cognitive and academic outcomes have yet to differ between treated and untreated groups.

2.3 Critical periods

Part of the reason may be that for some children (or some outcomes), this intervention came too late: the first two or three years of life are thought to represent crucial phases for both physical and cognitive development (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.[3] 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

[3]Windows during which such outside influences have especially strong effects are referred to as "sensitive" periods (Knudsen 2004); when the consequences are not only large, but also permanent, these periods are referred to as "critical." But because "critical" and "sensitive" periods differ across cognitive faculties (Grantham-McGregor, *et al.* 2007, 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.

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.[4] Rainfall in other years had no significant long-term consequences.

Because the intervention for schoolchildren in Kenya had such large spillover effects, 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 de-worming and health, summarized by Albonico, *et al.* (2008). Four studies in East Africa all found short-term health gains; among these, Alderman *et al.* (2006) 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[5] 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."

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

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

3 Data collection, 2009-1010

In 2009 and 2010, a field team in Kenya collected height, weight, migration and cognitive data from more than 20,000 children at the 73 deworming project schools in Samia and Bunyala districts of Kenya's Western Province. During both data collection years, children from the same age cohorts were included. 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 Table 1: for example, in communities where deworming began in 1998, the pupils I find in 2009 at age 11 began experiencing the effects of community deworming at age zero. Pupils I find in 2009 at age 11 in other communities, where deworming began in 1999, for example, only began experiencing the effects of community deworming at age one. 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 2. 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 2, I restrict attention to the sample of non-migrants. Panel B shows that the non-migrants are demographically much the same as the full sample, and goes on to tabulate several other characteristics: 21.6 percent of this population is stunted[6];

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

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 indicators for age at the time of community deworming, explained in Table 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 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 the levels until they make nine mistakes in a single level. For reasoning, I use the 12-question Set B of J. C. Raven's Progressive Matrices, a series of puzzles commonly used to measure of general intelligence.[7] For short-term memory, I use forward and backward digit-spans of increasing length. I provide raw means and standard deviations in Table 2, 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 A1 through A4. The first column of Table A1 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.[8] Correlations among cognitive measures are shown in Table A2: all are positive. The relationships between

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

[8] 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.

cognitive performance, age, and grade in school are shown in Appendix Tables A3 and A4. 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 typically chosen to repeat grades more frequently.

4 Estimation

Because of the original randomized design, I begin estimation with a simple specification. For each individual i, I estimate 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. I also include fixed effects for every combination of age, sex, and data collection year:

$$Y_i = \beta_1^C \cdot Before_i^C + \sum_{A,S,Y} \gamma_{ASY} D_{Age_i=A} \cdot D_{Sex_i=S} \cdot D_{Year_i=Y} + \epsilon_i \quad (1)$$

However, because respondents were generally only able to report their age to the nearest year, even if equation 1 is correctly specified, estimation of β^C may be biased down. A more flexible specification also includes one or more $Exact_i^c$ terms, indicators for whether deworming took place when individual i was exactly c years old:

$$Y_i = \beta_2^C \cdot Before_i^C + \sum_{c=C}^{C_H} \beta_2^{ec} \cdot Exact_i^c + \sum_{A,S,Y} \gamma_{ASY} D_{Age_i=a} \cdot D_{Sex_i=S} \cdot D_{Year_i=Y} + \epsilon_i \quad (2)$$

As the number of terms in the $\{C \ ... \ C_H\}$ summation increases, however, statistical power for estimating β_2^C diminishes.

A less flexible, but possibly appropriate specification includes only one $Exact_i^C$ term, but restricts the coefficient on it to be a scaled version of the coefficient on $Before_i^C$:

$$Y_i = \beta_3^C \cdot \left(Before_i^C + \alpha \cdot Exact_i^C\right) + \sum_{A,S,Y} \gamma_{ASY} D_{Age_i=a} \cdot D_{Sex_i=S} \cdot D_{Year_i=Y} + \epsilon_i \quad (3)$$

5 Results

Results are shown in Tables 3 through 6. In Table 3, I report the estimated β_2^C coefficient from equation 2 with only one extra term, $C = C_H = 1$ year, 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 $\hat{\beta_2^C}$ 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 on Raven's 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. These effects are equivalent to between 0.5 and 0.8 additional grades in school: the relationships between cognitive measures and current grade in school are shown in Appendix Tables A3 and A4, and are only slightly larger than the coefficients being estimated here. Of all the cognitive measures included in this module, performance on Raven's Matrices is the most closely related to innate intelligence; its responsiveness to the intervention suggests that even mild disease burdens early in childhood can alter cognitive development.

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.

One of the key issues in the child development literature is the decreasing plasticity of physiological and neural development with age. The age at which spillover effects of community deworming are most valuable to a child has not been documented before, and may shed light on child development more generally. In Table 4, I show a variety of specifications based on variations of Equation 2. In the first seven columns, I vary the value of C, the age

before which deworming took place, from negative two to positive four.[9] 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_2^3 or β_2^4 (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 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 1. The specification in column 10 yields the coefficients shown earlier in Table 3. The evidence here is that 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; deworming after age 1 cannot be statistically distinguished from deworming much later.

Because timing is such an important parameter in child development, I take one other approach to measuring it, shown in Table 5. I consider three specifications: the simple model shown in Equation 1, the inclusion of one additional dummy as shown in Equation 2 (with C_H=C), and the scaled single dummy as shown in Equation 3. I estimate each of these specifications for values of C from -2 to +4 in order to see which value of C yields the best fit (R^2). For the latter two specifications, deworming before age 1 (C=1) fits the data best; for the first specification, deworming before age 2 (C=2) does. To see how variable

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

this measure of fit is, I then draw 10,000 bootstrap samples from the data (the same sample size, but with observations drawn with replacement from the original dataset) and repeat the procedure for each bootstrap sample, tabulating the fraction of replications for which each value of C has the best fit. Results confirm the fit found in the original dataset. Reading across the columns, the same value of C is chosen in the bootstrap procedure as in the original data 80.2, 84, and 91.2 percent of the time, respectively.[10] Clearly community deworming before age one yields a large long-term cognitive benefit, but whether or not the same benefits would be conveyed between ages one and two is not completely answered by the data in this study.

To help untangle the mechanisms behind this effect, I consider different subpopulations in Table 6. 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 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.

[10] Percentages this high are rarely observed when the data generating procedure is pure noise (one-sided $p<0.05$).

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 with caring for younger siblings, still supporting an epidemiological story, but it seems to provide evidence against a household budget constraint story, in which healthier older brothers might play a key role.

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 different for boys and girls, though they are slightly higher for girls.

5.1 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).

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 a severe nutritional deprivation in early childhood;

the low intensity of worm infections at this age is in accord with this reasoning.

6 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.

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Table 1: Age at community deworming by region and age at data collection

Age in 2009:	8	9	10	11	12	13	14
1998 Deworming region	-3	-2	-1	0	1	2	3
1999 Deworming region	-2	-1	0	1	2	3	4
2001 Deworming region	0	1	2	3	4	5	6

Age in 2010:	9	10	11	12	13	14	15
1998 Deworming region	-3	-2	-1	0	1	2	3
1999 Deworming region	-2	-1	0	1	2	3	4
2001 Deworming region	0	1	2	3	4	5	6

The table above illustrates the design used in this analysis. The deworming phase-in was randomized in so that some communities started receiving mass treatment in 1998, others in 1999, and still others in 2001. As such, for example, a twelve-year-old child living in a community where deworming began in 1998 who participated in the 2009 round of data collection for this paper would have been one year old at the time mass deworming began in his community. The variation in age at community deworming illustrated above is the basis for the design presented in this paper.

Table 2: 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 3: Main effects: community deworming before age one

Outcome	**Effect**
Raven's Matrices	0.220***
	(0.078)
PPVT Level	0.161*
	(0.096)
Verbal Fluency: animals	0.182**
	(0.088)
Verbal Fluency: foods	0.160*
	(0.089)
Memory: digit span forwards	0.135
	(0.095)
Memory: digit span backwards	0.023
	(0.086)
All cognitive: First principal component	0.215**
	(0.097)
Height (cm)	0.204
	(0.297)
Height-for-age z-score	0.029
	(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 4: 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 5: Locating the critical period: bootstrapping different models

	Model 1 (From eqn. 1) *Simple cutoff*	Model 2 (From eqn. 2) *Simple cutoff with separate effect for subsequent year*	Model 3 (From eqn. 3) *Half-effect in subsequent year*
Best fit value of C:	2	1	1
Bootstrap results:			
Cutoff year (C):	Fraction of replications with best fit:		
Before age -2	0.00%	0.00%	0.00%
Before age -1	0.04%	1.01%	0.00%
Before age 0	0.18%	0.53%	0.68%
Before age 1	16.53%	84.00%	91.20%
Before age 2	80.20%	12.30%	6.50%
Before age 3	0.54%	1.04%	0.75%
Before age 4	2.51%	1.12%	0.87%

The table above is generated using 10,000 bootstrap replications. For each replication, and for each column, seven regressions are carried out for the bootstrapped sample. The outcome is always performance on Raven's Matrices, but for each column, each of the seven age cutoffs is separately estimated according to the model specified by the column. The best fitting model of the seven (maximizing R^2) is tabulated; each column thus sums to one.

Table 6: 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**	0.249**	0.842***	0.074	0.224**	0.214*
	(0.078)	(0.164)	(0.118)	(0.267)	(0.199)	(0.113)	(0.124)
All cognitive: First principal component	0.215**	0.396**	0.188	0.771***	0.247	0.241**	0.187
	(0.097)	(0.159)	(0.132)	(0.254)	(0.237)	(0.120)	(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 3, 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.

A Appendix

Table A1: 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 A2: 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 A3: 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 A4: 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