Three Papers On Deworming

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WORMS: IDENTIFYING IMPACTS ON EDUCATION AND HEALTH IN THE PRESENCE OF TREATMENT EXTERNALITIES

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Intestinal helminths—including hookworm, roundworm, whipworm, and schistosomiasis—infect more than one-quarter of the world's population. Studies in which medical treatment is randomized at the individual level potentially doubly underestimate the benefits of treatment, missing externality benefits to the comparison group from reduced disease transmission, and therefore also underestimating benefits for the treatment group. We evaluate a Kenyan project in which school-based mass treatment with deworming drugs was randomly phased into schools, rather than to individuals, allowing estimation of overall program effects. The program reduced school absenteeism in treatment schools by one-quarter, and was far cheaper than alternative ways of boosting school participation. Deworming substantially improved health and school participation among untreated children in both treatment schools and neighboring schools, and these externalities are large enough to justify fully subsidizing treatment. Yet we do not find evidence that deworming improved academic test scores.

KEYWORDS: Health, education, Africa, externalities, randomized evaluation, worms.

1. INTRODUCTION

HOOKWORM, ROUNDWORM, WHIPWORM, and schistosomiasis infect one in four people worldwide. They are particularly prevalent among school-age children in developing countries. We examine the impact of a program in which seventy-five rural Kenyan primary schools were phased into deworming treatment in a randomized order. We find that the program reduced school absenteeism by at least one-quarter, with particularly large participation gains among the youngest children, making deworming a highly effective way to boost school participation among young children. We then identify cross-school externalities—the impact of deworming for pupils in schools located near treatment schools—using exogenous variation in the local density of treatment school pupils generated by the school-level randomization, and find that deworming reduces worm burdens and increases school participation among

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children in neighboring primary schools. There is also some evidence of within-school treatment externalities, although given that randomization took place across schools, rather than across pupils within schools, we cannot use experimental identification to decompose the overall effect on treatment schools into a direct effect and a within-school externality effect, and must rely on necessarily more tentative nonexperimental methods.

Including the externality benefits, the cost per additional year of school participation is only $3.50, making deworming considerably more cost-effective than alternative methods of increasing school participation, such as school subsidies (see Kremer (2003)). Moreover, internalizing these externalities would likely require not only fully subsidizing deworming, but actually paying people to receive treatment.

We do not find any evidence that deworming increased academic test scores. However, the school participation gains we estimate are not large enough to generate statistically significant test score gains given the observed cross-sectional relationship between school attendance and test scores.

There is a large literature documenting positive correlations between health and economic outcomes. Our results suggest a causal link running from health to education.[2] The finding that treatment externalities are large also suggests a potentially important role for subsidies for treatment, especially given that nearly half of Africa's disease burden is due to infectious and parasitic disease (WHO (1999)).

Our approach can be distinguished from that in several recent studies in which treatment is typically randomized at the individual level and its educational impact is estimated by comparing cognitive ability among those treatment and comparison pupils who attend a later testing session. Dickson et al. (2000) review these studies and conclude that they do not provide convincing evidence for educational benefits of deworming. However, these studies fail to account for potential externalities for the comparison group from reduced disease transmission. Moreover, if externalities benefit the comparison group, outcome differences between the treatment and comparison groups will understate the benefits of treatment on the treated. This identification problem is closely related to the well-known issue of contamination of experimental job programs in active labor markets, where programs have externality effects on program nonparticipants (typically by worsening their outcomes, as discussed in Heckman, LaLonde, and Smith (1999)).

[2]Refer to Strauss and Thomas (1998) for a survey of the literature on health and income. While nonexperimental studies have found that poor early childhood nutrition is associated with delayed primary school enrollment and reduced academic achievement in Ghana (Glewwe and Jacoby (1995)) and the Philippines (Glewwe, Jacoby, and King (2001)), and several prospective studies suggest iron supplementation improves academic outcomes of anemic children (Nokes, van den Bosch, and Bundy (1998)), Behrman's (1996) review argues that given the limited experimental evidence and the difficulty of inferring causality from correlations in nonexperimental data, aside from anemia, the existing literature on child health and education is inconclusive.

We use two approaches to deal with the problem of identification in the presence of local externalities. First, because randomization took place at the level of schools, we are able to estimate the overall effect of deworming on a school even if there are treatment externalities among pupils within the school. Second, we identify cross-school externalities—the impact of deworming for pupils in schools located near treatment schools—using exogenous variation in the local density of treatment school pupils generated by the school-level randomization. As discussed above, we find large deworming treatment externalities both on health and education, and our analysis suggests that failure to account for these externalities would lead to substantially underestimating the impacts of deworming.

The paper is organized as follows. Section 2 reviews the existing literature on helminths and education. Section 3 describes the project we evaluate in rural Kenya and presents the baseline educational and medical characteristics. Section 4 describes the estimation strategy. Sections 5, 6, and 7 discuss the program's effect on health, school participation, and test scores, respectively. Section 8 examines the cost-effectiveness of deworming relative to other ways of improving health and school participation and argues the estimated externalities justify fully subsidizing deworming. The final section summarizes and discusses implications of the results.

2. INTESTINAL HELMINTH (WORM) INFECTIONS

Hookworm and roundworm each infect approximately 1.3 billion people around the world, while whipworm affects 900 million and 200 million are infected with schistosomiasis (Bundy (1994)).While most have light infections, which may be asymptomatic, a minority have heavy infections, which can lead to iron-deficiency anemia, protein-energy malnutrition, abdominal pain, and listlessness. [3] Schistosomiasis can also have more severe consequences, for instance, causing enlargement of the liver and spleen.

Low-cost single-dose oral therapies can kill the worms, reducing hookworm, roundworm, and schistosomiasis infections by 99 percent, although single-dose treatments are only moderately effective against severe whipworm infections (Butterworth et al. (1991), Nokes et al. (1992), Bennett and Guyatt (2000)). Reinfection is rapid, however, with worm burden often returning to eighty percent or more of its original level within a year (Anderson and May (1991)), and hence geohelminth drugs must be taken every six months and schistosomiasis drugs must be taken annually. The World Health Organization has endorsed mass school-based deworming programs in areas with high helminth infections, since this eliminates the need for costly individual parasitological screening (Warren et al. (1993), WHO (1987)), bringing cost down to as little

[3]Refer to Adams et al. (1994), Corbett et al. (1992), Hotez and Pritchard (1995), and Pollitt (1990).

as 49 cents per person per year in Africa (PCD (1999)). Known drug side effects are minor, and include stomach ache, diarrhea, dizziness, and vomiting in some cases (WHO (1992)). However, due to concern about the possibility that the drugs could cause birth defects (WHO (1992), Cowden and Hotez (2000)), standard practice in mass deworming programs has been to not treat girls of reproductive age (Bundy and Guyatt (1996)).[4]

Medical treatment could potentially interfere with disease transmission, creating positive externalities. School-aged children likely account for the bulk of helminth transmission (Butterworth et al. (1991)). Muchiri, Ouma, and King (1996) find that school children account for 85 to 90 percent of all heavy schistosomiasis infections in nine eastern Kenyan villages. Moreover, conditional on infection levels, children are most likely to spread worm infections because they are less likely to use latrines and more generally have poor hygiene practices (Ouma (1987), Butterworth et al. (1991)).[5]

Treatment externalities for schistosomiasis are likely to take place across larger areas than is typical for geohelminth externalities due to the differing modes of disease transmission. Geohelminth eggs are deposited in the local environment when children defecate in the "bush" surrounding their home or school, while the schistosomiasis parasite is spread through contact with infected fresh water. Children in the area are often infected with schistosomiasis by bathing or fishing in Lake Victoria, and children who live some distance from each other may bathe or fish at the same points on the lake. Moreover, the water-borne schistosome may be carried considerable distances by stream and lake currents, and the snails that serve as its intermediate hosts are themselves mobile.

In the absence of frequent reinfection, individual worm burdens are likely to fall rapidly given the relatively short typical life spans of intestinal worms: twelve months for roundworm and whipworm, two years for hookworm, and three years for schistosomiasis (Bundy and Cooper (1989), Anderson and May (1991)), so that if the age of worms within a human host is uniformly distributed, worm burden may halve in six to eighteen months depending on the worm. There is existing only limited empirical evidence on deworming treatment externalities, but that which exists suggests that school-based deworming may create substantial externalities.[6] However, these studies rely on pre-post

[4]With a lengthening track record of safe use, this practice is now changing.

[5]Animal-human transmission is not a serious concern in this area for hookworm, whipworm, and schistosomiasis (Cambridge University Schistosomiasis Research Group (2000), Corwin (2000)), and is unlikely to be a major concern for roundworm. A roundworm species that predominantly infects pigs (*Ascaris suum*) may also sometimes infect humans, but is unlikely to be a major problem in this area since fewer than 15 percent of households keep pigs at home.

[6]Adult worm burden fell by nearly fifty percent after fifteen months on the island of Montserrat in communities where children were mass treated for worms (Bundy et al. (1990)). We examine four other related studies—two of which do not explicitly discuss externalities, but whose published results allow us to compute them—and find reductions of up to fifty percent in infec-

comparisons in the same villages to estimate externalities for untreated individuals. This leaves them without a plausible comparison group, which is particularly problematic since infection rates vary widely seasonally and from year to year due to rainfall variation and other factors (Kloos et al. (1997)). The randomized phase-in across schools of the deworming intervention that we examine allows us to capture the overall effect of deworming even in the presence of externalities across individuals within schools. School-level randomization also naturally generates local variation in the density of treatment that we use to estimate spillovers across schools. Our sample of 75 schools is also much larger than existing studies, which were typically conducted in five or fewer villages.

The educational impact of deworming is considered a key issue in assessing whether the poorest countries should accord priority to deworming (Dickson et al. (2000)). It has been hypothesized that intense worm infections reduce educational achievement (Bundy (1994), Del Rosso, Miller, and Marek (1996), Drake et al. (1999), Stoltzfus et al. (1997)), either by inducing anemia, which is known to affect educational outcomes (Nokes, van den Bosch, and Bundy (1998)), or through other channels, including protein-energy malnutrition. However, in an influential Cochrane review published in the *British Medical Journal*, Dickson et al. (2000) claim that "the evidence of benefit for mass [deworming] treatment of children related to positive effects on [physical] growth and cognitive performance is not convincing. In light of these data, we would be unwilling to recommend that countries or regions invest in programmes that routinely treat children with anthelmintic drugs."

Yet the existing randomized evaluations on worms and education on which Dickson et al. (2000) base their conclusions suffer from several shortcomings. First, existing studies randomize the provision of deworming treatment *within* schools to treatment and placebo groups, and then examine the impact of deworming on cognitive outcomes. Their within-school randomization designs prevent existing studies from credibly estimating externality benefits. Moreover, the difference in educational outcomes between the treatment and placebo groups understates the actual impact of deworming on the treatment group if placebo group pupils also experience health gains due to local treatment externalities. In fact, re-examination of these recent randomized studies suggests that untreated placebo pupils often experienced substantial worm load reductions, as would be consistent with the hypothesis of within-school externalities.[7]

tion intensity among untreated individuals in communities where school children received mass deworming (Butterworth et al. (1991), Holland et al. (1996), Muchiri, Ouma, and King (1996), Thein-Hlaing, Than-Saw, and Myat-Lay-Kyin (1991)).

[7]In Simeon, Grantham-McGregor, Callender, and Wong (1995), all pupils started with heavy whipworm infections (over 1200 eggs per gram, epg). Thirty-two weeks into the study, heavy infections fell 95 percent in the treatment group and 43 percent among the placebo group, and treatment and placebo pupils showed an identical gain of 0.3 in body mass index (low body mass index is associated with acute nutritional deficiencies). Simeon, Grantham-McGregor, and Wong

A second shortcoming of existing randomized studies is that although they report the impact of deworming on tests of cognitive performance (such as tests of recall), they typically do not examine other outcomes of interest to policymakers, including school attendance, enrollment, academic test scores, or grade promotion. Only two studies examine effects on attendance and both should be interpreted with caution since the data were drawn from attendance registers, which are notoriously inaccurate in many developing countries. Treating growth-stunted Jamaican children with heavy whipworm infections increased school attendance by 9.9 percentage points, reducing absenteeism by one-third (Simeon, Grantham-McGregor, Callender, and Wong (1995)).Thirty-five percent of pupils were missing attendance data. Watkins, Cruz, and Pollitt (1996a, 1996b) find no effect of treatment of roundworm and whipworm on primary school attendance. However, periods of extended school absence are dropped, leading to high rates of recorded attendance (90 percent). If treated pupils were healthier and had fewer inactive periods, this creates attrition bias and will thus understate the true impact of deworming on school attendance. However, nonexperimental studies suggest that worms do affect school participation. [8]

To the extent that deworming increases school participation, as we suggest, other existing studies may also suffer serious attrition bias. For example, Nokes et al. (1992) report test score data for 89 percent of students in their treatment group but only 59 percent in their comparison group.[9]

(1995), which was conducted among a subsample of the study population in Simeon, Grantham-McGregor, Callender, and Wong (1995), find that median whipworm load fell from 2523 epg for the treatment pupils pre-treatment, to 0 epg after 32 weeks, while among placebo pupils median whipworm load fell from 2946 to 1724 epg, a drop of roughly one-third among placebo pupils. In Nokes et al. (1992), average hookworm infection intensity fell by fifty percent among the placebo pupils (although there was no change in roundworm or whipworm infection for placebo pupils). Since the samples in these studies were selected based on high worm load, the fall in worm load among placebo pupils could potentially be due to mean reversion as well as to externalities. However, Watkins, Cruz, and Pollitt (1996) did not select their sample based on worm load, and find that mean roundworm epg fell roughly 25 percent among placebo pupils after twenty-four weeks of treatment with albendazole.

[8]Geissler et al. (2000) interviewed school children from a nearby region of western Kenya, and argue that worms may caused school absence in five percent of all interviews (and account for nearly half of all absences). Bleakley (2002) finds that areas in the U.S. South with higher hookworm infection levels prior to the 1910–1920 Rockefeller Sanitary Commission deworming campaign experienced greater increases in school attendance after the intervention, and estimates that each case of hookworm reduced the number of children attending school by 0.23 (which is similar to our estimates presented below). Although it is difficult to fully rule out omitted variable bias using a nonexperimental approach, an important strength of Bleakley (2002) is that the Rockefeller campaign was introduced throughout a large geographic area, and thus the estimates are not subject to the biases faced by medical studies that randomize treatment at the individual level. (Brinkley (1994) argues that the Rockefeller campaign also dramatically increased agricultural productivity.)

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3. THE PRIMARY SCHOOL DEWORMING PROJECT IN BUSIA, KENYA

We evaluate the Primary School Deworming Project (PSDP), which was carried out by a Dutch nonprofit organization, Internationaal Christelijk Steunfonds Africa (ICS), in cooperation with the Busia District Ministry of Health office. The project took place in southern Busia, a poor and densely-settled farming region in western Kenya, in an area with the highest helminth infection rates in Busia district. The 75 project schools consist of nearly all rural primary schools in this area, and had a total enrolment of over 30,000 pupils between ages six to eighteen.

In January 1998, the seventy-five PSDP schools were randomly divided into three groups of twenty-five schools each: the schools were first stratified by administrative subunit (zone) and by their involvement in other nongovernmental assistance programs, and were then listed alphabetically and every third school was assigned to a given project group.[10] Due to ICS's administrative and financial constraints, the health intervention was phased in over several years. Group 1 schools received free deworming treatment in both 1998 and 1999, Group 2 schools in 1999, while Group 3 schools began receiving treatment in 2001. Thus in 1998, Group 1 schools were treatment schools, while Group 2 and Group 3 schools were comparison schools, and in 1999, Group 1 and Group 2 schools were treatment schools and Group 3 schools were comparison schools.

3.1. *Baseline Characteristics*

ICS field staff administered pupil and school questionnaires in early 1998 and again in early 1999. Prior to treatment, the groups were similar on most demographic, nutritional, and socioeconomic characteristics, but despite randomized assignment—which produces groups with similar characteristics in expectation—Group 1 pupils appear to be worse off than Group 2 and 3 pupils along some dimensions, potentially creating a bias against finding significant program effects (Table I). There are no statistically significant differences across Group 1, 2, and 3 schools in enrolment, distance to Lake Victoria, school sanitation facilities, pupils' weight-for-age,[11] asset ownership, self-reported malaria, or the local density of other primary school pupils located within three kilometers or three to six kilometers. Helminth infection rates in the surrounding geographic zone are also nearly identical across the three groups. School attendance rates did not differ significantly in early 1998 before the first round of medical treatment, although this baseline attendance

[10]Twenty-seven of the seventy-five project schools were also involved in other NGO projects, which consisted of financial assistance for textbook purchase and classroom construction, and teacher performance incentives. Appendix Table AI presents a detailed project timeline.

[11]Unfortunately, due to problems with field data collection, we do not have usable baseline height data.

TABLE I
1998 AVERAGE PUPIL AND SCHOOL CHARACTERISTICS, PRE-TREATMENT[a]

	Group 1 (25 schools)	Group 2 (25 schools)	Group 3 (25 schools)	Group 1 – Group 3	Group 2 – Group 3
Panel A: Pre-school to Grade 8					
Male	0.53	0.51	0.52	0.01	−0.01
				(0.02)	(0.02)
Proportion girls <13 years, and all boys	0.89	0.89	0.88	0.00	0.01
				(0.01)	(0.01)
Grade progression (= Grade − (Age − 6))	−2.1	−1.9	−2.1	−0.0	0.1
				(0.1)	(0.1)
Year of birth	1986.2	1986.5	1985.8	0.4**	0.8***
				(0.2)	(0.2)
Panel B: Grades 3 to 8					
Attendance recorded in school registers (during the four weeks prior to the pupil survey)	0.973	0.963	0.969	0.003	−0.006
				(0.004)	(0.004)
Access to latrine at home	0.82	0.81	0.82	0.00	−0.01
				(0.03)	(0.03)
Have livestock (cows, goats, pigs, sheep) at home	0.66	0.67	0.66	−0.00	0.01
				(0.03)	(0.03)
Weight-for-age Z-score (low scores denote undernutrition)	−1.39	−1.40	−1.44	0.05	0.04
				(0.05)	(0.05)
Blood in stool (self-reported)	0.26	0.22	0.19	0.07**	0.03
				(0.03)	(0.03)
Sick often (self-reported)	0.10	0.10	0.08	0.02**	0.02**
				(0.01)	(0.01)
Malaria/fever in past week (self-reported)	0.37	0.38	0.40	−0.03	−0.02
				(0.03)	(0.03)
Clean (observed by field workers)	0.60	0.66	0.67	−0.07**	−0.01
				(0.03)	(0.03)
Panel C: School characteristics					
District exam score 1996, grades 5–8[b]	−0.10	0.09	0.01	−0.11	0.08
				(0.12)	(0.12)
Distance to Lake Victoria	10.0	9.9	9.5	0.6	0.5
				(1.9)	(1.9)
Pupil population	392.7	403.8	375.9	16.8	27.9
				(57.6)	(57.6)
School latrines per pupil	0.007	0.006	0.007	0.001	−0.000
				(0.001)	(0.001)
Proportion moderate-heavy infections in zone	0.37	0.37	0.36	0.01	0.01
				(0.03)	(0.03)
Group 1 pupils within 3 km[c]	461.1	408.3	344.5	116.6	63.8
				(120.3)	(120.3)
Group 1 pupils within 3–6 km	844.5	652.0	869.7	−25.1	−217.6
				(140.9)	(140.9)

TABLE I
(CONTINUED)

	Group 1 (25 schools)	Group 2 (25 schools)	Group 3 (25 schools)	Group 1 – Group 3	Group 2 – Group 3
Total primary school pupils within 3 km	1229.1	1364.3	1151.9	77.2 (205.5)	212.4 (205.5)
Total primary school pupils within 3–6 km	2370.7	2324.2	2401.7	−31.1 (209.5)	−77.6 (209.5)

[a]School averages weighted by pupil population. Standard errors in parentheses. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence. Data from the 1998 ICS Pupil Namelist, 1998 Pupil Questionnaire and 1998 School Questionnaire.

[b]1996 District exam scores have been normalized to be in units of individual level standard deviations, and so are comparable in units to the 1998 and 1999 ICS test scores (under the assumption that the decomposition of test score variance within and between schools was the same in 1996, 1998, and 1999).

[c]This includes girls less than 13 years old, and all boys (those eligible for deworming in treatment schools).

information comes from school registers, which are not considered reliable in Kenya.

To the extent that there were significant differences between treatment and comparison schools, treatment schools were initially somewhat worse off. Group 1 pupils had significantly more self-reported blood in stool (a symptom of schistosomiasis infection), reported being sick more often than Group 3 pupils, and were not as clean as Group 2 and Group 3 pupils (as observed by NGO field workers). They also had substantially lower average scores on 1996 Kenyan primary school examinations than Group 2 and 3 schools, although the difference is not significant at traditional confidence levels.

In January and February 1998, prior to treatment, a random sample of ninety grade three to eight pupils (fifteen per grade) in each of the 25 Group 1 schools were selected to participate in a parasitological survey conducted by the Kenya Ministry of Health, Division of Vector Borne Diseases.[12] Ninety-two percent of surveyed pupils had at least one helminth infection and thirty-seven percent had at least one moderate-to-heavy helminth infection (Table II),[13] although these figures understate actual infection prevalence to the extent that the most heavily infected children were more likely to be absent from school on the day of the survey. Worm infection rates are relatively high in this region by international standards, but many other African settings have similar infection

[12]Following the previous literature, infection intensity is proxied for worm eggs per gram (epg) in stool (Medley and Anderson (1985)). Each child in the parasitological sample was given a plastic container and asked to provide a stool sample; samples were examined in duplicate within twenty-four hours using the Kato-Katz method. Group 2 and Group 3 schools were not included in the 1998 parasitological survey since it was not considered ethical to collect detailed health information from pupils who were not scheduled to receive medical treatment in that year.

[13]Following Brooker et al. (2000b), thresholds for moderate infection are 250 epg for *Schistosomiasis. mansoni* and 5,000 epg for Roundworm, the WHO standards, and 750 epg for Hookworm and 400 epg for Whipworm, both somewhat lower than the WHO standard.

TABLE II
JANUARY 1998 HELMINTH INFECTIONS, PRE-TREATMENT, GROUP 1 SCHOOLS[a]

	Prevalence of infection	Prevalence of moderate-heavy infection	Average infection intensity, in eggs per gram (s.e.)
Hookworm	0.77	0.15	426 (1055)
Roundworm	0.42	0.16	2337 (5156)
Schistosomiasis, all schools	0.22	0.07	91 (413)
Schistosomiasis, schools <5 km from Lake Victoria	0.80	0.39	487 (879)
Whipworm	0.55	0.10	161 (470)
At least one infection	0.92	0.37	–
Born since 1985	0.92	0.40	–
Born before 1985	0.91	0.34	–
Female	0.91	0.34	–
Male	0.93	0.38	–
At least two infections	0.31	0.10	–
At least three infections	0.28	0.01	–

[a]These are averages of individual-level data, as presented in Brooker et al. (2000b); correcting for the oversampling of the (numerically smaller) upper grades does not substantially change the results. Standard errors in parentheses. Sample size: 1894 pupils. Fifteen pupils per standard in grades 3 to 8 for Group 1 schools were randomly sampled. The bottom two rows of the column "Prevalence of moderate-heavy infection" should be interpreted as the proportion with at least two or at least three moderate-to-heavy helminth infections, respectively.

The data were collected in January to March 1998 by the Kenya Ministry of Health, Division of Vector Borne Diseases (DVBD). The moderate infection thresholds for the various intestinal helminths are: 250 epg for *S. mansoni*, and 5,000 epg for Roundworm, both the WHO standard, and 750 epg for Hookworm and 400 epg for Whipworm, both somewhat lower than the WHO standard. Refer to Brooker et al. (2000b) for a discussion of this parasitological survey and the infection cut-offs. All cases of schistosomiasis are *S. mansoni*.

profiles (Brooker et al. (2000a)). Moderate-to-heavy worm infections are more likely among younger pupils and among boys. Pupils who attend schools near Lake Victoria also have substantially higher rates of schistosomiasis. Latrine ownership is negatively correlated with moderate-to-heavy infection (results not shown).

3.2. *The Intervention*

Following World Health Organization recommendations (WHO (1992)), schools with geohelminth prevalence over 50 percent were mass treated with albendazole every six months, and schools with schistosomiasis prevalence over 30 percent were mass treated with praziquantel annually.[14] All treatment

[14]The medical protocol was designed in collaboration with the Partnership for Child Development, and was approved by the Ethics Committee of the Kenya Ministry of Health and Busia

schools met the geohelminth cut-off in both 1998 and 1999. Six of twenty-five treatment schools met the schistosomiasis cut-off in 1998 and sixteen of fifty treatment schools met the cut-off in 1999.[15] Medical treatment was delivered to the schools by Kenya Ministry of Health public health nurses and ICS public health officers. Following standard practice (Bundy and Guyatt (1996)), the medical protocol did not call for treating girls thirteen years of age and older due to concerns about the potential teratogenicity of the drugs (WHO (1992)).[16]

In addition, treatment schools received worm prevention education through regular public health lectures, wall charts, and the training of teachers in each treatment school on worm prevention. Health education stressed the importance of hand washing to avoid ingesting roundworm and whipworm larvae, wearing shoes to avoid hookworm infection, and not swimming in infected fresh water to avoid schistosomiasis.

ICS obtained community consent in all treatment schools in 1998. A series of community and parent meetings were held in treatment schools, at which the project was described and parents who did not want their child to participate in the project were asked to inform the school headmaster. Under the recommendation of the Kenya Ministry of Health, beginning in January 1999 ICS required signed parental consent for all children to receive medical treatment; consent typically took the form of parents signing their name in a notebook kept at school by the headmaster. This is not a trivial requirement for many households: travelling to school to sign the book may be time-consuming, and some parents may be reluctant to meet the headmaster when behind on school fees, a common problem in these schools.

District Medical Officer of Health. The 30 percent threshold for mass praziquantel treatment is less than the WHO standard of 50 percent, although in practice few schools had schistosomiasis prevalence between 30 to 50 percent. Pupils in the parasitological subsample who were found to be infected with schistosomiasis, but attended schools that did not qualify for mass treatment with praziquantel, were individually treated. However, there were few such pupils: the proportion of moderate-to-heavy schistosomiasis among the thirty-four schools that fell below the 30 percent threshold in 1999 was just 0.02.

[15]In 1998, pupils received 600 mg albendazole doses during each round of treatment, following the protocol of an earlier Government of Kenya Ministry of Health deworming project in Kwale District; in 1999, pupils were treated with 400 mg albendazole (WHO (1992)). Praziquantel was provided at approximately 40 mg/kg (WHO (1992)) in both 1998 and 1999. The NGO used generic drugs in 1998, and SmithKline Beecham's Zentel (albendazole) and Bayer's Biltricide (praziquantel) in 1999.

[16]Pregnancy test reagent strips are not practical during mass treatment (Bundy and Guyatt (1996)). Personal interviews (i.e., asking girls when they had their most recent menstrual period) may not be effective in determining pregnancy in this setting because pregnant girls might fear that the information would not be held in confidence; pregnant girls are often expelled from Kenyan primary schools (although this is not official government policy).

3.3. *Assigned and Actual Deworming Treatment*

Seventy-eight percent of those pupils assigned to receive treatment (i.e., girls under thirteen years old and all boys in the treatment schools) received at least some medical treatment through the program in 1998 (Table III).[17] Since approximately 80 percent of the students enrolled prior to the start of the pro-

TABLE III
PROPORTION OF PUPILS RECEIVING DEWORMING TREATMENT IN PSDP[a]

	Group 1		Group 2		Group 3	
	Girls <13 years, and all boys	Girls ≥ 13 years	Girls <13 years, and all boys	Girls ≥ 13 years	Girls <13 years, and all boys	Girls ≥ 13 years
	Treatment		*Comparison*		*Comparison*	
Any medical treatment in 1998 (For grades 1–8 in early 1998)	0.78	0.19	0	0	0	0
Round 1 (March–April 1998), Albendazole	0.69	0.11	0	0	0	0
Round 1 (March–April 1998), Praziquantel[b]	0.64	0.34	0	0	0	0
Round 2 (Oct.–Nov. 1998), Albendazole	0.56	0.07	0	0	0	0
	Treatment		*Treatment*		*Comparison*	
Any medical treatment in 1999 (For grades 1–7 in early 1998)	0.59	0.07	0.55	0.10	0.01	0
Round 1 (March–June 1999), Albendazole	0.44	0.06	0.35	0.06	0.01	0
Round 1 (March–June 1999), Praziquantel[b]	0.47	0.06	0.38	0.06	0.01	0
Round 2 (Oct.–Nov. 1999), Albendazole	0.53	0.06	0.51	0.08	0.01	0
Any medical treatment in 1999 (For grades 1–7 in early 1998), among pupils enrolled in 1999	0.73	0.10	0.71	0.13	0.02	0
Round 1 (March–June 1999), Albendazole	0.55	0.08	0.46	0.08	0.01	0
Round 1 (March–June 1999), Praziquantel[b]	0.53	0.07	0.45	0.07	0.01	0
Round 2 (Oct.–Nov. 1999), Albendazole	0.65	0.09	0.66	0.11	0.01	0

[a]Data for grades 1–8. Since month of birth information is missing for most pupils, precise assignment of treatment eligibility status for girls born during the "threshold" year is often impossible; all girls who turn 13 during a given year are counted as 12 year olds (eligible for deworming treatment) throughout for consistency.

[b]Praziquantel figures in Table III refer only to children in schools meeting the schistosomiasis treament threshold (30 percent prevalence) in that year.

[17]In what follows, "treatment" schools refer to all twenty-five Group 1 schools in 1998, and all fifty Group 1 and Group 2 schools in 1999.

gram were present in school on a typical day in 1998, absence from school on the day of drug administration was a major cause of drug noncompliance. Nineteen percent of girls thirteen years of age or older also received medical treatment in 1998. This was partly because of confusion in the field about pupil age, and partly because in the early stages of the program several of the Kenya Ministry of Health nurses administered drugs to some older girls, judging the benefits of treatment to outweigh the risks. This was particularly common in schools near the lake where schistosomiasis was more of a problem.

A somewhat lower proportion of pupils in school took the medicine in 1999. Among girls younger than thirteen and boys who were enrolled in school for at least part of the 1999 school year, the overall treatment rate was approximately 72 percent (73 percent in Group 1 and 71 percent in Group 2 schools), suggesting that the process of selection into treatment was fairly similar in the two years despite the change in consent rules. Of course, measured relative to the baseline population of students enrolled in early 1998, a smaller percentage of students were still in school in 1999 and hence, treatment rates in this baseline sample were considerably lower in 1999 than in 1998: among girls under thirteen years of age and all boys in treatment schools from the baseline sample, approximately 57 percent received medical treatment at some point in 1999, while only nine percent of the girls thirteen years of age and older received treatment.[18]

Only five percent of comparison school pupils received medical treatment for worms independently of the program during the previous year, according to the 1999 pupil questionnaire.[19] An anthropological study examining worm treatment practices in a neighboring district in Kenya (Geissler et al. (2000)), finds that children self-treat the symptoms of helminth infections with local herbs, but found no case in which a child or parent purchased deworming

[18]The difference between the 72 percent and 57 percent figures is due to Group 2 pupils who dropped out of school (or who could not be matched in the data cross years, despite the efforts of the NGO field staff) between years 1 and 2 of the project. Below, we compare infection outcomes for pupils who participated in the 1999 parasitological survey, all of whom were enrolled in school in 1999. Thus the parasitological survey sample consists of pupils enrolled in school in both 1998 and 1999 for both the treatment and comparison schools. To the extent that the deworming program itself affected enrolment outcomes—1999 school enrolment is approximately four percentage points higher in the treatment schools than the comparison schools—the pupils enrolled in the treatment versus comparison schools in 1999 will have different characteristics. However, since drop-out rates were lower in the treatment schools, this is likely to lead to a bias toward zero in the within-school health externality estimates, in which case our estimates serve as lower bounds on true within-school effects.

[19]A survey to assess the availability of deworming drugs in this area, conducted during May to July 1999, found no local shops surveyed carried either WHO-recommended broad-spectrum treatments for geohelminths (albendazole and mebendazole) or schistosomiasis (praziquantel) in stock on the day of the survey, though a minority carried cheaper but less effective drugs (levamisole hydrochloride and piperazine). Some clinics and pharmacies carried broad-spectrum drugs, but these were priced far out of range for most of the population.

TABLE IV
PROPORTION OF PUPIL TRANSFERS ACROSS SCHOOLS

School in early 1998 (pre-treatment)	1998 transfer to a Group 1 school	1998 transfer to a Group 2 school	1998 transfer to a Group 3 school	1999 transfer to a Group 1 school	1999 transfer to a Group 2 school	1999 transfer to a Group 3 school
Group 1	0.005	0.007	0.007	0.032	0.026	0.027
Group 2	0.006	0.007	0.008	0.026	0.033	0.027
Group 3	0.010	0.010	0.006	0.022	0.036	0.022
Total transfers	0.021	0.024	0.021	0.080	0.095	0.076

drugs. To the extent that children in Busia also self-treat helminth symptoms with herbs, in this study we measure the net benefit of deworming drugs above and beyond the impact of herbs and of any individually purchased medicines.

Although pupils assigned to comparison schools could also potentially have transferred to treatment schools to receive deworming medical treatment through the program, there is no evidence of large asymmetric flows of pupils into treatment schools, which could bias the results (Table IV). Among sample pupils, approximately two percent transferred into a different school in 1998, with nearly equal proportions transferring into Groups 1, 2, and 3 schools, and approximately eight percent of pupils had transferred into a different school by the end of 1999, again with similar proportions transferring to all three groups (the transfer rates from early 1998 through the end of 1999 are substantially higher than rates through the end of 1998 because most transfers occur between school years). As we discuss in Section 4, we also use a standard intention-to-treat (ITT) estimation strategy, in which pupils are assigned the treatment status of the school in which they were initially enrolled in early 1998 even if they later switched schools, to address potential transfer bias.

3.4. *Health Outcome Differences Between Group 1 and Group 2 Schools*

Before proceeding to formal estimation in Section 4, we present simple differences in health outcomes between treatment and comparison schools, although as we discuss below, these differences understate overall treatment effects if there are deworming treatment externalities across schools. The Kenyan Ministry of Health conducted a parasitological survey of grade three to eight pupils in Group 1 and Group 2 schools in January and February 1999, one year after the first round of treatment but before Group 2 schools had been treated. Overall, 27 percent of pupils in Group 1 (1998 treatment) schools had a moderate-to-heavy helminth infection in early 1999 compared to 52 percent in Group 2 (1998 comparison) schools, and this difference is significantly different than zero at 99 percent confidence (Table V). The prevalences of moderate-to-heavy hookworm, roundworm, schistosomiasis, and whipworm infections were all lower in Group 1 (1998 treatment) schools than in Group 2

TABLE V
JANUARY TO MARCH 1999, HEALTH AND HEALTH BEHAVIOR DIFFERENCES BETWEEN GROUP 1 (1998 TREATMENT) AND GROUP 2 (1998 COMPARISON) SCHOOLS[a]

	Group 1	Group 2	Group 1 – Group 2
Panel A: Helminth Infection Rates			
Any moderate-heavy infection, January–March 1998	0.38	–	–
Any moderate-heavy infection, 1999	0.27	0.52	−0.25***
			(0.06)
Hookworm moderate-heavy infection, 1999	0.06	0.22	−0.16***
			(0.03)
Roundworm moderate-heavy infection, 1999	0.09	0.24	−0.15***
			(0.04)
Schistosomiasis moderate-heavy infection, 1999	0.08	0.18	−0.10*
			(0.06)
Whipworm moderate-heavy infection, 1999	0.13	0.17	−0.04
			(0.05)
Panel B: Other Nutritional and Health Outcomes			
Sick in past week (self-reported), 1999	0.41	0.45	−0.04**
			(0.02)
Sick often (self-reported), 1999	0.12	0.15	−0.03**
			(0.01)
Height-for-age Z-score, 1999	−1.13	−1.22	0.09*
(low scores denote undernutrition)			(0.05)
Weight-for-age Z-score, 1999	−1.25	−1.25	−0.00
(low scores denote undernutrition)			(0.04)
Hemoglobin concentration (g/L), 1999	124.8	123.2	1.6
			(1.4)
Proportion anemic (Hb < 100g/L), 1999	0.02	0.04	−0.02**
			(0.01)
Panel C: Worm Prevention Behaviors			
Clean (observed by field worker), 1999	0.59	0.60	−0.01
			(0.02)
Wears shoes (observed by field worker), 1999	0.24	0.26	−0.02
			(0.03)
Days contact with fresh water in past week	2.4	2.2	0.2
(self-reported), 1999			(0.3)

[a]These are averages of individual-level data for grade 3–8 pupils; disturbance terms are clustered within schools. Robust standard errors in parentheses. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence.

Obs. for parasitological results: 2328 (862 Group 1,1467 Group 2); Obs. for hemoglobin results: 778 (292 Group 1, 486 Group 2); Obs. for 1999 Pupil Questionnaire health outcomes: 9,102 (3562 Group 1, 5540 Group 2 and Group 3).

Following Brooker et al. (2000b), moderate-to-heavy infection thresholds for the various intestinal helminths are: 250 epg for *S. mansoni*, and 5,000 epg for Roundworm, both the WHO standard, and 750 epg for Hookworm and 400 epg for Whipworm, both somewhat lower than the WHO standard. Kenya Ministry of Health officials collected the parasitological data from January to March 1998 in Group 1 schools, and from January to March 1999 in Group 1 and Group 2 schools. A random subset of the original 1998 Group 1 parasitological sample was resurveyed in 1999. Hb data were collected by Kenya Ministry of Health officials and ICS field officers using the portable Hemocue machine. The self-reported health outcomes were collected for all three groups of schools as part of Pupil Questionnaire administration.

(1998 comparison) schools. The program was somewhat less effective against whipworm, perhaps as a result of the lower efficacy of single-dose albendazole treatments for whipworm infections, as discussed above.[20]

Note that it is likely that substantial reinfection had occurred during the three to twelve months between 1998 deworming treatment and the 1999 parasitological surveys, so differences in worm burden between treatment and comparison schools were likely to have been even greater shortly after treatment. In addition, to the extent that pupils prone to worm infections are more likely to be present in school on the day of the parasitological survey in the Group 1 schools than the Group 2 schools due to deworming health gains, these average differences between Group 1 and Group 2 schools are likely to further understate true deworming treatment effects.

Group 1 pupils also reported better health outcomes after the first year of deworming treatment: four percent fewer Group 1 pupils reported being sick in the past week, and three percent fewer pupils reported being sick often (these differences are significantly different than zero at 95 percent confidence). Group 1 pupils also had significantly better height-for-age—a measure of nutritional status—by early 1999, though weight-for-age was no greater on average.[21]

Although Group 1 pupils had higher hemoglobin concentrations than Group 2 pupils in early 1999, the difference is not statistically different than zero. Recall that anemia is the most frequently hypothesized link between worm infections and cognitive performance (Stoltzfus et al. (1997)). Severe anemia is relatively rare in Busia: fewer then 4 percent of pupils in Group 2 schools (comparison schools in 1998) fell below the Kenya Ministry of Health anemia threshold of 100 g/L in early 1999 before deworming treatment. This is low relative to many other areas in Africa, of which many have substantial helminth problems: a recent survey of studies of anemia among school children in less developed countries (Hall and Partnership for Child Development (2000)) indicates that there is considerably less anemia in Busia than in samples from Ghana, Malawi, Mali, Mozambique, and Tanzania.[22]

[20]The rise in overall moderate-to-heavy helminth infections between 1998 and 1999 (refer to Table II) is likely to be due to the extraordinary flooding in 1998 associated with the El Niño weather system, which increased exposure to infected fresh water (note the especially large increases in moderate-to-heavy schistosomiasis infections), created moist conditions favorable for geohelminth larvae, and led to the overflow of latrines, incidentally also creating a major outbreak of fecal-borne cholera.

[21]Although it is somewhat surprising to find height-for-age gains but not weight-for-age gains, since the latter are typically associated with short-run nutritional improvements, it is worth noting that Thein-Hlaing, Thane-Toe, Than-Saw, Myat-Lay-Kyin, and Myint-Lwin's (1991) study in Myanmar finds large height gains among treated children within six months of treatment for roundworm while weight gains were only observed after twenty-four months, and Cooper et al. (1990) present a similar finding for whipworm, so the result is not unprecedented.

[22]One possible explanation for low levels of anemia in this area is geophagy (soil eating): Geissler et al. (1998) report that 73 percent of a random sample of children aged 10–18 in a

Health education had a minimal impact on behavior, so to the extent the program improved health, it almost certainly did so through the effect of anthelmintics rather than through health education. There are no significant differences across treatment and comparison school pupils in early 1999 in three worm prevention behaviors: observed pupil cleanliness,[23] the proportion of pupils wearing shoes, or self-reported exposure to fresh water (Table V).

4. ESTIMATION STRATEGY

4.1. *Econometric Specifications*

Randomization of deworming treatment across schools allows estimation of the overall effect of the program by comparing treatment and comparison schools, even in the presence of within-school externalities.[24] However, externalities may take place not only within, but also across schools, especially since most people in this area live on their farms rather than being concentrated in villages, and neighbors (and even siblings) often attend different schools since there is typically more than one primary school within walking distance. Miguel and Gugerty (2002) find that nearly one-quarter of all households in this area have a child enrolled in a primary school which is not the nearest one to their home. We estimate cross-school externalities by taking advantage of variation in the local density of treatment schools induced by randomization. Although randomization across schools makes it possible to experimentally identify both the overall program effect and cross-school externalities, we must rely on non-experimental methods to decompose the effect on treated schools into a direct effect and within-school externality effect.

We first estimate program impacts in treatment schools, as well as cross-school treatment externalities:[25]

$$(1)\qquad Y_{ijt} = a + \beta_1 \cdot T_{1it} + \beta_2 \cdot T_{2it} + X'_{ijt}\delta + \sum_d (\gamma_d \cdot N^T_{dit}) + \sum_d (\phi_d \cdot N_{dit}) + u_i + e_{ijt}.$$

neighboring region of Western Kenya reported eating soil daily. Given the average amount of soil children were observed eating daily, and the measured mean iron content of soil in this area, Geissler et al. conclude that soil provides an average of 4.7 mg iron per day—over one-third of the recommended daily iron intake for children. Unfortunately, geophagy could also increase exposure to geohelminth larvae, promoting reinfection.

[23]This also holds controlling for initial 1998 levels of cleanliness, or using a difference-in-differences specification.

[24]Manski (2000) suggests using experimental methods to identify peer effects. Other recent papers that use group-level randomization of treatment to estimate peer effects include Duflo and Saez (2002) and Miguel and Kremer (2002). Katz, Kling, and Liebman (2001), Kremer and Levy (2001), and Sacerdote (2001) use random variation in peer group composition to estimate peer effects.

[25]For simplicity, we present the linear form, but we use probit estimation below for discrete dependent variables.

Y_{ijt} is the individual health or education outcome, where i refers to the school, j to the student, and $t \in \{1, 2\}$ to the year of the program; T_{1it} and T_{2it} are indicator variables for school assignment to the first and second year of deworming treatment, respectively; and X_{ijt} are school and pupil characteristics. N_{dit} is the total number of pupils in primary schools at distance d from school i in year t, and N^T_{dit} is the number of these pupils in schools randomly assigned to deworming treatment. For example, in Sections 5 and 6, $d = 03$ denotes schools that are located within three kilometers of school i, and $d = 36$ denotes schools that are located between three to six kilometers away.[26] Individual disturbance terms are assumed to be independent across schools, but are allowed to be correlated for observations within the same school, where the school effect is captured in the u_i term.

Since local population density may affect disease transmission, and since children who live or attend school near treatment schools could have lower environmental exposure to helminths, which would lead to less reinfection and lower worm burdens, worm burden may depend on both the total number of primary school pupils (N_{dit}) and the number of those pupils in schools randomly assigned to deworming treatment (N^T_{dit}) within a certain distance from school i in year t of the program.[27] Given the total number of children attending primary school within a certain distance from the school, the number of these attending schools assigned to treatment is exogenous and random. Since any independent effect of local school density is captured in the N_{dit} terms, the γ_d coefficients measure the deworming treatment externalities across schools. In this framework $\beta_1 + \sum_d(\gamma_d \overline{N}^T_{dit})$ is the average effect of the first year of deworming treatment on overall infection prevalence in treatment schools, where $\overline{N}^T_{dit}$ is the average number of treatment school pupils located at distance d from the school, and $\beta_2 + \sum_d(\gamma_d \overline{N}^T_{dit})$ is the analogous effect for the second year of deworming. β_1 and β_2 capture both direct effects of deworming treatment on the treated, as well as any externalities on untreated pupils within the treatment schools.[28]

[26]Under spatial externality models in which a reduction in worm prevalence at one school affects neighboring schools and this in turn affects their neighbors, some externalities would spill over beyond six kilometers. To the extent that there are externalities beyond six kilometers from the treatment schools, equation (1) yields a lower bound on treatment effects, but we think any such spillovers are likely to be relatively minor in this setting.

[27]Since cross-school externalities depend on the number of pupils eligible for treatment rather than the total number of pupils, we use the number of girls less than 13 years old and all boys (the pupils eligible for deworming in the treatment schools) as the school population (N_{dit} and N^T_{dit}) for all schools in the remainder of the paper. Measurement error in GPS locations—due to U.S. government downgrading of GPS accuracy until May 2000—leads to attenuation bias, making it more difficult to find treatment externalities.

[28]Unfortunately, we do not have data on the location of pupils' homes, and hence cannot examine if pupils living near treatment schools actually obtain greater externality benefits.

The assigned deworming treatment group is not significantly associated with the density of other local treatment school pupils within three kilometers or within three to six kilometers (Table I); in other words, approximately as many treated pupils are located near Group 1 schools as near Group 2 or 3 schools. The 1998 and 1999 deworming compliance rates are also not significantly associated with the local density of treatment school pupils conditional on the total local density (Appendix Table AII).

Cross-school deworming externalities are likely to increase with the proportion of the local population that receives deworming treatment. Although the school-level randomization induced a range of variation in local treatment densities in our sample, with only 49 schools we cannot estimate how marginal externalities vary with local treatment levels.[29] Yet since large-scale deworming programs in most poor countries would likely use community consent for treatment, rather than individual parental consent—as in the first year of the program we examine—we estimate the likely extent of treatment externalities under conditions of interest to public health policymakers.

Including school and pupil variables X_{ijt} controls for those pre-treatment differences across schools that were present despite randomization, increasing statistical precision. These controls include the average school score on the 1996 Kenya government district exams for grades 5 to 8;[30] the prevalence of moderate-to-heavy helminth infections in the pupil's grade and geographic zone (the pre-treatment average); indicators for school involvement in other nongovernmental organization assistance projects; time controls (indicator variables for each six-month period capture the downward trend in school participation due to dropouts); and grade cohort indicator variables.

4.2. *Estimating Within-School Externalities*

Because randomization was conducted at the level of schools, rather than individuals within schools, it is possible to both estimate the overall treatment effect on treated schools and to conduct a cost-benefit analysis using equation (1). However, it is not possible to experimentally decompose the effect for treatment schools into a direct effect on treated pupils and an externality effect on untreated pupils within treatment schools. It is not valid to use assignment to a treatment school as an instrumental variable for actual medical treatment

[29]Quadratic terms of local treatment densities are not significantly related to the rate of any moderate-to-heavy helminth infection (results not shown), and thus we opt to focus on the linear specification, as in equation (1).

[30]Average school scores from 1996—two years before the first year of the project—were employed since the district exam was not offered in 1997 due to a national teacher strike. Average school exam scores are used because individual exam results are incomplete for 1996. However, the 1996 scores are corrected to be in units of individual level standard deviations, and are thus comparable to the 1998 and 1999 test scores under the assumption that the decomposition of test score variance within and between schools was the same in 1996, 1998, and 1999.

in the presence of such externalities (Angrist, Imbens, and Rubin (1996)) since the exclusion restriction fails to hold: assignment to a treatment school affects pupil health through externalities, rather than only through the likelihood of receiving medical treatment.

In thinking about nonexperimental approaches to such a decomposition, it is worth bearing in mind that there is no evidence that sicker pupils were more likely to obtain deworming treatment; in fact if anything, the evidence seems more consistent with the hypothesis that pupils with higher worm load were somewhat less likely to obtain treatment, either because they were less likely to be in school on the day of treatment or because their households were less willing and able to invest in health. As Panels A and B in Table VI indicate, among girls under 13 and all boys, the children who would remain untreated were slightly more likely to be moderately to heavily infected prior to the intervention than those who ultimately obtained treatment, both for Group 1 schools (in 1998) and Group 2 schools (in 1999). Among girls at least 13 years of age, there is little difference in 1998 infection rates (prior to treatment) between Group 1 pupils who later obtained treatment and those who did not, while the Group 2 pupils who later obtained treatment were substantially less likely to have been moderately to heavily infected in early 1999 than their counterparts who later went untreated.

As suggested above, a major cause of missing treatment is school absenteeism: a 2001 parent survey indicates that most noncompliance from absenteeism is due to pupil illness, and we show in Section 6 that pupils with worms miss school more often. Poorer pupils may also have lower compliance if parents who have not paid school fees are reluctant to visit the headmaster to provide consent.

We assume in what follows that children obtain treatment if the net gain from treatment is more than a cut-off cost. Formally, $D_{1ij} = 1(S(X_{ijt}, e_{ijt}) + \varepsilon_{ijt} > C_t)$, where D_{1ij} takes on a value of one if individual j in school i received treatment in the first year that her school was eligible for treatment (1998 for Group 1, 1999 for Group 2), and zero otherwise; here, $1(\cdot)$ is the indicator function, C_t is the total cost to the household of obtaining treatment in year t (which varies between the two years due to the changing consent requirements), and ε_{ijt} is an unobserved random variable that could depend on the distance of the pupil's home from school, or whether the pupil was sick on the treatment day, for example.

Given that there was no randomization of treatment within schools, Group 1 pupils who did not receive treatment in 1998 are compared to Group 2 pupils who did not receive treatment in 1999, the year that Group 2 schools were incorporated into treatment, to at least partially deal with potential bias due to selection into medical treatment. For the health outcomes, we compare these two groups as of January to February 1999, when Group 1 schools had already been treated (in 1998) but Group 2 schools had not, while for school participation we compare Groups 1 and 2 during the first year of treatment.

TABLE VI

DEWORMING HEALTH EXTERNALITIES WITHIN SCHOOLS, JANUARY TO MARCH 1999[a]

	Group 1, Treated in 1998	Group 1, Untreated in 1998	Group 2, Treated in 1999	Group 2, Untreated in 1999	(Group 1, Treated 1998) – (Group 2, Treated 1999)	(Group 1, Untreated 1998) – (Group 2, Untreated 1999)
Panel A: Selection into Treatment						
Any moderate-heavy infection, 1998	0.39	0.44	–	–	–	–
Proportion of 1998 parasitological sample tracked to 1999 sample[b]	0.36	0.36	–	–	–	–
Access to latrine at home, 1998	0.84	0.80	0.81	0.86	0.03	−0.06
					(0.04)	(0.05)
Grade progression (= Grade − (Age − 6)), 1998	−2.0	−1.8	−1.8	−1.8	−0.2**	−0.0
					(0.1)	(0.2)
Weight-for-age (Z-score), 1998 (low scores denote undernutrition)	−1.58	−1.52	−1.57	−1.46	−0.01	−0.06
					(0.06)	(0.11)
Malaria/fever in past week (self-reported), 1998	0.37	0.41	0.40	0.39	−0.03	−0.01
					(0.04)	(0.06)
Clean (observed by field worker), 1998	0.53	0.59	0.60	0.66	−0.07	−0.07
					(0.05)	(0.10)
Panel B: Health Outcomes						
Girls <13 years, and all boys						
Any moderate-heavy infection, 1999	0.24	0.34	0.51	0.55	−0.27***	−0.21**
					(0.06)	(0.10)
Hookworm moderate-heavy infection, 1999	0.04	0.11	0.22	0.20	−0.19***	−0.09*
					(0.03)	(0.05)
Roundworm moderate-heavy infection, 1999	0.08	0.12	0.22	0.30	−0.14***	−0.18**
					(0.04)	(0.07)
Schistosomiasis moderate-heavy infection, 1999	0.09	0.08	0.20	0.13	−0.11*	−0.05
					(0.06)	(0.06)
Whipworm moderate-heavy infection, 1999	0.12	0.16	0.16	0.20	−0.04	−0.05
					(0.16)	(0.09)
Girls ≥13 years						
Any moderate-heavy infection, 1998	0.31	0.28	–	–	–	–
Any moderate-heavy infection, 1999	0.27	0.43	0.32	0.54	−0.05	−0.10
					(0.17)	(0.09)
Panel C: School Participation						
School participation rate, May 1998 to March 1999[c]	0.872	0.764	0.808	0.684	0.064**	0.080**
					(0.032)	(0.039)

[a]These are averages of individual-level data for grade 3–8 pupils in the parasitological survey subsample; disturbance terms are clustered within schools. Robust standard errors in parentheses. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence. The data are described in the footnote to Table V. Obs. for the 1999 parasitological survey: 670 Group 1 treated 1998, 77 Group 1 untreated 1998, 873 Group 2 treated 1999, 352 Group 2 untreated 1999.

[b]We attempted to track a random sample of half of the original 1998 parasitological sample. Because some pupils were absent, had dropped out, or had graduated, we were only able to resurvey 72 percent of this subsample.

[c]School averages weighted by pupil population. The participation rate is computed among pupils enrolled in the school at the start of 1998. Pupils present in school during an unannounced NGO visit are considered participants. Pupils had 3.8 participation observations per year on average. Participation rates are for grades 1 to 7; grade 8 pupils are excluded since many graduated after the 1998 school year, in which case their 1999 treatment status is irrelevant. Pre-school pupils are excluded since they typically have missing compliance data. All 1998 pupil characteristics in Panel A are for grades 3 to 7, since younger pupils were not administered the Pupil Questionnaire.

As we discussed above, the parental consent rules changed between 1998 and 1999, leading to a reduction in the fraction of pupils receiving treatment within treatment schools. Thus, restricting the sample to Group 1 and Group 2 schools (and holding the X_{ijt} terms constant for the moment, for clarity):

$$
\begin{aligned}
(2)\qquad & E(Y_{ij1}|T_{1i1}=1, X_{ij1}, D_{1ij}=0) - E(Y_{ij1}|T_{1i1}=0, X_{ij1}, D_{1ij}=0) \\
&= \beta_1 + \sum_d \gamma_d \cdot \big[E(N^T_{di1}|T_{1i1}=1, D_{1ij}=0) \\
&\qquad\qquad - E(N^T_{di1}|T_{1i1}=0, D_{1ij}=0)\big] \\
&\quad + \sum_d \gamma_d \cdot [E(N_{di1}|T_{1i1}=1, D_{1ij}=0) - E(N_{di1}|T_{1i1}=0, D_{1ij}=0)] \\
&\quad + [E(e_{ij1}|T_{1i1}=1, X_{ij1}, D_{1ij}=0) - E(e_{ij1}|T_{1i1}=0, X_{ij1}, D_{1ij}=0)],
\end{aligned}
$$

where T_{1i1} is the treatment assignment of the *school* in 1998 ($t = 1$), and this takes on a value of one for Group 1 and zero for Group 2 schools. The first term on the right-hand side of the equation (β_1) is the within-school externality effect. The second and third terms are effects due to differing local densities of primary schools between treatment and comparison schools; these are approximately zero (as we show in Table I) and in any case we are able to control for these densities in the estimation. The key final term, which can be rewritten as

$$
\begin{aligned}
& E(e_{ij1}|T_{1i1} = 1, X_{ij1}, C_1 - S(X_{ij1}, e_{ij1}) > \varepsilon_{ij1}) \\
&\quad - E(e_{ij1}|T_{1i1} = 0, X_{ij1}, C_2 - S(X_{ij2}, e_{ij2}) > \varepsilon_{ij2}),
\end{aligned}
$$

captures any unobserved differences between untreated pupils in the Group 1 and Group 2 schools. If $C_1 = C_2$, then by randomization this term equals zero and (2) can be used to estimate β_1. However, it is likely that $C_2 > C_1$ due to imposition of the signed parental consent requirement in 1999. In our sample, infected people are no more likely to be treated—and in fact seem somewhat *less* likely to be treated—and this is robust to conditioning on the full set of X_{ijt} variables described above (results not shown).[31] If S is in fact nondecreasing in e_{ijt}(which can be thought of as unobserved characteristics associated with good health outcomes in this specification), then $C_2 > C_1$ implies that the final term will be zero or negative, so the left hand side of the equation will if anything underestimate the within-school externality, β_1.[32] In other words, due to changes in the process of selection into treatment, some Group 2 pupils who would have been treated had they been in Group 1 were in fact not treated in 1999, and this implies that average unobservables e_{ijt} will be at least as great among the untreated in Group 2 as among the untreated in Group 1 (and also

[31] Pooling 1998 data for Group 1 pupils and 1999 data for Group 2 pupils, the estimated marginal effect of a moderate-to-heavy infection on drug take-up is −0.008, and this effect is not significantly different than zero.

[32] This claim also relies on the assumption that individual e_{ijt} terms are autocorrelated across the two years.

that average e_{ijt} will also be at least as great among the treated Group 2 as among the treated Group 1).

The change in overall infection rates between the first two years of the program (captured in X_{ijt} in the above model) may also have affected individual deworming treatment decisions. Infection rates changed across years both due to sizeable cross-school treatment externalities associated with the program, which acted to reduce infection levels, as well as to natural intertemporal variation (e.g., the 1998 flooding) which led to higher rates of moderate-to-heavy infection. This second effect appears to have dominated, leading to higher overall infection rates in 1999 relative to 1998 (Tables II and V), and complicating efforts to sign the direction of the bias in the within-school externality estimates. However, the fact that fewer people obtained treatment in year 2 than year 1 suggests that overall, given the changed consent requirements, the process of selection into treatment became more stringent, so that it is plausible that e_{ijt} is at least as great among the Group 2 pupils who were untreated in their first year of eligibility as among Group 1 pupils who were untreated in their first year of eligibility.

Turning to the data suggests that Group 1 pupils untreated in 1998 and Group 2 pupils untreated in 1999 are in fact similar, and that any bias is likely to be small. First, as noted earlier, moderate-to-heavily infected pupils are no more likely to seek treatment than their less infected fellow pupils. Second, there are no statistically significant differences between the Group 1 pupils untreated in 1998 and the Group 2 pupils untreated in 1999 in five baseline characteristics likely to be associated with child health—latrine ownership, grade progression, weight-for-age, self-reported health status, and cleanliness—and point estimates suggest that the Group 1 untreated pupils are actually somewhat less healthy, less clean, and less likely to have access to a latrine than their counterparts in Group 2 (Table VI, Panel A).[33] These results are consistent with the hypothesis that e_{ijt} in part reflects differences among households in ability and willingness to take action to improve their children's health, and that those pupils with high values of e_{ijt} were somewhat more likely to obtain treatment.[34,35]

A further piece of evidence comes from comparing the initial moderate-heavy infection rates (in early 1998) of Group 1 pupils treated in 1998 *and*

[33]The analogous comparison with the larger sample used in the school participation estimation (in Table IX) also suggests that Group 1 pupils untreated in 1998 and the Group 2 pupils untreated in 1999 are similar along these characteristics (results not shown).

[34]In other words, as the cost of treatment increased between years 1 and 2, the individuals who still opted to receive treatment in year 2—those with higherε_{ijt}, conditional on observables—had higher values of e_{ijt} than the individuals who were not treated in year 2 but would have been treated given the year 1 cost. Thus e_{ijt} and ε_{ijt} must be positively correlated among these individuals at the margin of receiving treatment.

[35]We have also calculated Manski bounds on within-school externalities in the presence of selection into treatment, but these are largely uninformative given the change in take-up between 1998 and 1999 (results not shown).

treated in 1999, to those treated in 1998 but *not* treated in 1999; this is not a perfect comparison, since Group 1 pupils were in their second year of treatment in 1999, while Group 2 pupils were experiencing their first year of treatment in 1999, but it still provides useful information on how changing the costs of treatment affects take-up. We find that the initial 1998 infection rates of the Group 1 pupils treated in 1999 and those untreated in 1999 differ by less than one percentage point (results not shown), providing further evidence that the change in consent rules between 1998 and 1999 did not substantially change the health status of those who chose to receive treatment through the program.

If the expectation of e_{ij1} is the same for the Group 1 pupils who missed their first year of treatment in 1998, and the Group 2 pupils who missed treatment in 1999, then we can estimate both within-school and cross-school treatment externalities in 1998 using equation (3):

$$Y_{ijt} = a + \beta_1 \cdot T_{1it} + b_1 \cdot D_{1ij} + b_2 \cdot (T_{1it} * D_{1ij}) + X'_{ijt}\delta + \sum_d (\gamma_d \cdot N^T_{dit}) + \sum_d (\phi_d \cdot N_{dit}) + u_i + e_{ijt}. \tag{3}$$

Here, β_1 is the within-school externality effect on the untreated, and $(\beta_1 + b_2)$ is the sum of the within-school externality effect plus the additional direct effect of treatment on the treated. If the final term in equation (2) is negative, as we suggest above, this specification underestimates within-school externalities and overstates the impact on the treated within treatment schools; of course, the estimation of overall program effects based on equation (1) is independent of the decomposition into effects on the treated and untreated within treatment schools. The total externality effect for the untreated in treatment schools is the sum of the within-school externality term and the cross-school externality in equation (3). In certain specifications we interact the local pupil density terms with the treatment school indicator to estimate potentially differential cross-school externalities in treatment and comparison schools.

4.3. *Initial Evidence on Within-School Deworming Externalities*

Before presenting results using this unified estimation framework in Sections 5, 6, and 7, we preview the within-school externality results by comparing the January–March 1999 infection levels of the Group 1 pupils who did not receive treatment in 1998 and the Group 2 pupils who did not receive treatment in 1999 (the year that Group 2 schools were incorporated into the treatment group). Among girls under thirteen years of age and all boys—those children who were supposed to receive medical treatment through the project—rates of moderate-to-heavy infections were 21 percentage points lower among Group 1 pupils who did not receive medical treatment in 1998 (34 percent) than among Group 2 pupils who did not receive treatment in 1999 (55 percent), and this difference is significant at 95 percent confidence (Table VI). These differences are negative and statistically significant for hookworm and roundworm, and negative but insignificant for schistosomiasis and whipworm; since the overall difference in whipworm infection between Group 1 and 2 schools was minimal, and there is evidence that single-dose albendazole treatments are sometimes ineffective against whipworm, it is not surprising that evidence of within-school externalities is weaker for whipworm. By way of contrast, Group 1 pupils who were treated in 1998 had a 24 percent chance of moderate-to-heavy infection in January to February 1999, while Group 2 pupils who would obtain treatment later in 1999 had a 51 percent chance of infection, for a difference of 27 percentage points. Thus at the time infection status was measured in early 1999, the difference in the prevalence of moderate-to-heavy infections among the untreated was approximately three-quarters the difference in prevalence for the treated (21 versus 27 percentage points).

The relatively large ratio of externality benefits to benefits for the treated is plausible given the timing of 1998 treatment and the 1999 parasitological survey. Following treatment of part of a population at steady-state worm infection intensity, the treated group will be reinfected over time and their worm load will asymptote to its original level. As discussed in Section 2, other studies have found that prevalence of hookworm, roundworm, and schistosomiasis falls by over 99 percent immediately after treatment, but that reinfection occurs rapidly. On the other hand, worm load among the untreated will gradually fall after the treatment group is dewormed, since the rate of infection transmission declines. Eventually, however, worm load among the untreated will rise again, asymptoting to its original steady-state level as the treated population becomes reinfected. The ratio of worm load among the treated to that among the untreated then approaches one over time. Since we collect data on worm infections some time after treatment—the January–March 1999 parasitological survey was carried out nearly one year after the first round of medical treatment and three to five months since the second round of treatment—and worm loads among the treated are substantial by this point, it seems reasonable to think that reinfection subsequent to the date of treatment accounts for much of observed worm load, and that the average difference in prevalence between treatment and comparison schools over the course of the year was likely to have been considerably greater than the difference observed in early 1999.

Two additional sources of evidence are consistent with positive within-school deworming treatment externalities. First, although girls aged 13 years and older were largely excluded from deworming treatment, moderate-to-heavy infection rates among older girls in Group 1 schools were ten percentage points lower than among similar girls in Group 2 schools, though this difference is not significantly different than zero (Table VI, Panel B).[36]

[36]It is not surprising that the magnitude of within-school externalities is somewhat smaller for older girls than for the population as a whole since these girls have lower rates of moderate to heavy infection (Table II), and are also twice as likely to wear shoes (results not shown), limiting reinfection. As a robustness check, we also estimate equation (3) using an instrumental variables

Second, a parasitological survey of 557 children entering preschool who had not yet had any opportunity to receive medical treatment through the program found that in early 2001, before Group 3 schools had begun receiving deworming treatment, children entering preschool in Group 1 and 2 schools had 7.1 percentage points fewer moderate-to-heavy hookworm infections than those entering Group 3 schools, an effect that is significantly different than zero at 90 percent confidence (results not shown). Given that only 18.8 percent of the Group 3 preschool children suffered from moderate-to-heavy hookworm infections, this constitutes a forty percent reduction in the proportion of such infections. The effects for the other worms were not statistically significant, which is not surprising for whipworm, since the direct treatment effects were small, or for schistosomiasis—for which externalities likely are less localized, and may not be as relevant for young children who are likely to stay near home, rather than going fishing in Lake Victoria—but is somewhat unexpected for roundworm (note, however, that Nokes et al. (1992) also find externalities for hookworm but not other geohelminths).

5. DEWORMING TREATMENT EFFECTS ON HEALTH AND NUTRITION

Formal estimation confirms that children in deworming treatment schools experienced a range of health benefits, and provides evidence that these benefits spilled over both to nontreated pupils in the treatment schools and to pupils in neighboring schools. Consistent with the differing modes of disease transmission, geohelminth externalities were primarily within schools, while schistosomiasis externalities were primarily across schools.

Estimation of equation (1) indicates that the proportion of pupils with moderate to heavy infection is 25 percentage points lower in Group 1 schools than Group 2 schools in early 1999 and this effect is statistically significant at 99 percent confidence (Table VII, regression 1). We next estimate equation (3), which decomposes the effect of the program on treated schools into an effect on treated pupils and a within-school externality effect. The within-school externality effect, given by the coefficient estimate on the Group 1 indicator variable, is a 12 percentage point reduction in the proportion of moderate-to-heavy infections, while the additional direct effect of deworming treatment is approximately 14 percentage points, and both of these coefficient estimates are significantly different than zero (Table VII, regression 2). Children who attend primary schools located near Group 1 schools had lower rates of moderate-to-heavy helminth infection in early 1999: controlling for the total number of

approach, instrumenting for actual deworming treatment with an indicator variable taking on a value of one for girls under 13 years of age and for all boys interacted with the school treatment assignment indicator. This yields a negative, but statistically insignificant, effect of treatment of schoolmates on infection among older girls (Appendix Table AIV). We cannot reject the hypothesis that the IV estimates of the within-school externality are the same as the probit estimates presented below.

TABLE VII
DEWORMING HEALTH EXTERNALITIES WITHIN AND ACROSS SCHOOLS, JANUARY TO MARCH 1999[a]

	Any moderate-heavy helminth infection, 1999			Moderate-heavy schistosomiasis infection, 1999			Moderate-heavy geohelminth infection, 1999		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Indicator for Group 1 (1998 Treatment) School	−0.25***	−0.12*	−0.09	−0.03	−0.02	−0.07	−0.20***	−0.11**	−0.03
	(0.05)	(0.07)	(0.11)	(0.03)	(0.04)	(0.06)	(0.04)	(0.05)	(0.09)
Group 1 pupils within 3 km (per 1000 pupils)	−0.26***	−0.26***	−0.11	−0.12***	−0.12***	−0.11**	−0.12*	−0.12*	−0.01
	(0.09)	(0.09)	(0.13)	(0.04)	(0.04)	(0.05)	(0.06)	(0.07)	(0.07)
Group 1 pupils within 3–6 km (per 1000 pupils)	−0.14**	−0.13**	−0.07	−0.18***	−0.18***	−0.27***	0.04	0.04	0.16
	(0.06)	(0.06)	(0.14)	(0.03)	(0.03)	(0.06)	(0.06)	(0.06)	(0.10)
Total pupils within 3 km (per 1000 pupils)	0.11***	0.11***	0.10**	0.11***	0.11***	0.13***	0.03	0.04	0.02
	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)	(0.03)	(0.03)	(0.03)
Total pupils within 3–6 km (per 1000 pupils)	0.13**	0.13**	0.12*	0.12***	0.12***	0.16***	0.04	0.04	0.01
	(0.06)	(0.06)	(0.07)	(0.03)	(0.03)	(0.03)	(0.04)	(0.04)	(0.04)
Received first year of deworming treatment, when offered (1998 for Group 1, 1999 for Group 2)		−0.06*			0.03**			−0.04**	
		(0.03)			(0.02)			(0.02)	
(Group 1 Indicator) ∗ Received treatment, when offered		−0.14*			−0.02			−0.10***	
		(0.07)			(0.04)			(0.04)	
(Group 1 Indicator) ∗ Group 1 pupils within 3 km (per 1000 pupils)			−0.25*			−0.04			−0.18**
			(0.14)			(0.07)			(0.08)
(Group 1 Indicator) ∗ Group 1 pupils within 3–6 km (per 1000 pupils)			−0.09			0.11			−0.15
			(0.13)			(0.07)			(0.10)
Grade indicators, school assistance controls, district exam score control	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Number of observations	2328	2328	2328	2328	2328	2328	2328	2328	2328
Mean of dependent variable	0.41	0.41	0.41	0.16	0.16	0.16	0.32	0.32	0.32

[a]Grade 3–8 pupils. Probit estimation, robust standard errors in parentheses. Disturbance terms are clustered within schools. Observations are weighted by total school population. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence. The 1999 parasitological survey data are for Group 1 and Group 2 schools. The pupil population data is from the 1998 School Questionnaire. The geohelminths are hookworm, roundworm, and whipworm. We use the number of girls less than 13 years old and all boys (the pupils eligible for deworming in the treatment schools) as the school population for all schools.

(age and sex eligible) children attending any primary school within three kilometers, the presence of each additional thousand (age and sex eligible) pupils attending Group 1 schools located within three kilometers of a school is associated with 26 percentage points fewer moderate-to-heavy infections, and this coefficient estimate is significantly different than zero at 99 percent confidence. Each additional thousand pupils attending a Group 1 school located between three to six kilometers away is associated with 14 percentage points fewer moderate-to-heavy infections, which is smaller than the effect of pupils within three kilometers, as expected, and is significantly different than zero at 95 percent confidence (Table VII, regression 1).[37] Due to the relatively small size of the study area, we are unable to precisely estimate the impact of additional treatment school pupils farther than six kilometers away from a school, and thus cannot rule out the possibility that there were externalities at distances beyond six kilometers and possibly for the study area as a whole, in which case the estimates presented in Table VII (and discussed below) would be lower bounds on actual externality benefits.[38,39]

[37]We experimented with alternative measures of infection status. One such measure normalizes the egg count for each type of infection by dividing each egg count by the moderate-heavy infection threshold for that helminth, and then summing up the normalized egg counts across all four infections (hookworm, roundworm, schistosomiasis, and whipworm) to arrive at an overall infection "score." The results using this measure are similar to those using the moderate-to-heavy infection indicator, although the estimated reduction in worm prevalence due to within-school externalities becomes statistically insignificant (results available upon request).

[38]The use of the intention-to-treat estimation method could potentially create spurious findings of cross-school deworming externalities, since students initially in comparison schools who transfer into treatment schools in time to receive treatment are still classified as comparison pupils. However, we do not think this is a serious problem in practice since our results are nearly identical when we classify students not by their original school, but by the school they actually attended at the time of the parasitological survey (results available upon request). The relevant transfer rate between March 1998 and November 1998 is simply too small to account for the externalities we detect: only 1.6 percent of students in Groups 2 and 3 transferred into Group 1 schools during 1998, and only 1.4 percent of students in Group 1 transferred to Groups 2 or 3 (Table IV). Given that some of the Group 2 and 3 children presumably transferred too late in the school year to benefit from treatment, and that some early transfers did not receive treatment, fewer than 1 percent of comparison pupils were treated (Table III3).

[39]These results are largely robust to including the proportion of Group 1 pupils in the surrounding area as the explanatory variable, rather than the total number of Group 1 pupils in the surrounding area (see regressions 3 and 7 in Appendix Table AIII). The use of spatially correlated disturbance terms does not lead to substantial changes in standard errors and confidence levels (see regressions 2 and 6 in Appendix Table AIII). The school participation results in Table IX are also robust to the use of spatially correlated disturbance terms (results not shown). We examined the extent of spatial correlation across schools using Conley (1999) and Chen and Conley's (2000) semi-parametric framework, and as expected, find a positive and declining relationship between the correlation in infection rates and distance between schools, although the spatial correlation is relatively small once we condition on school-level characteristics. The cross-school externality results are also robust to controlling for initial 1998 infection levels among the sample of Group 1 pupils with both 1998 and 1999 parasitological data (see regressions 4 and 8

We estimate that moderate-to-heavy helminth infections among children in this area were 23 percentage points (standard error 7 percentage points) lower on average in early 1999 as a result of health spillovers across schools—over forty percent of overall moderate-to-heavy infection rates in Group 2 schools. To see this, note that the average spillover gain is the average number of Group 1 pupils located within three kilometers divided by 1000 ($\overline{N}^T_{03}$) times the average effect of an additional 1000 Group 1 pupils located within three kilometers on infection rates (γ_{03}), plus the analogous spillover effect due to schools located between three to six kilometers away from the school (refer to equation (1)). Based on the externality estimates in Table VII, regression 1, this implies the estimated average cross-school externality reduction in moderate-to-heavy helminth infections is $[\gamma_{03} * \overline{N}^T_{03,1} + \gamma_{36} * \overline{N}^T_{36,1}] = [0.26 * 454 + 0.14 * 802]/1000 = 0.23$.

Note that deworming drugs kill worms already in the body, but the drugs do not remain in the body and do not provide immunity against future re-infection, so it is plausible that the benefit from having fewer sources of re-infection is reasonably orthogonal to current infection status. However, own treatment and local treatment intensity need not simply have an additive effect on moderate-to-heavy infections: the interaction effect will be negative if cross-school externalities alone do not typically reduce infection levels below the moderate-to-heavy infection threshold for comparison school pupils as of the date of the parasitological survey, but the interaction of own treatment and externalities often does reduce infection below the threshold for treatment school pupils.[40] We find that the average cross-school externality reduction in moderate-to-heavy infections for comparison school (Group 2) pupils is 9 percentage points, while the effect for treatment school (Group 1) pupils is considerably larger, at nearly 29 percentage points (Table VII, regression 3). As discussed below, this difference is primarily due to geohelminths externalities, since externalities for the more serious schistosomiasis infections are similar for treatment and comparison schools.

The existence of cross-school health externalities implies that the difference in average outcomes between treatment and comparison schools—a "naïve" treatment effect estimator—understates the actual effects of mass deworming treatment on the treated. If externalities disappear completely after six kilometers, the true reduction in moderate-to-heavy infection rates among pupils in Group 1 schools is the sum of the average cross-school externality for comparison school pupils (9 percentage points) and the effect of being in a treatment school in early 1999 presented in Table VII, regression 1 (25 percentage

in Appendix Table AIII). We can only control for initial 1998 infection levels in the subsample of Group 1 schools, since this data was not collected for the other schools.

[40]More generally, the distribution of individual worm infection relative to the threshold level is also important for gauging the likely interaction effect between own treatment and the local treatment intensity.

points), for a total of 35 percentage points (the standard error is 9 percentage points, taking into account the covariance structure across coefficient estimates from Table VII, regression 3). The cross-school externality is thus over one-quarter as large as the total effect on the treated. The estimated number of moderate-to-heavy helminth infections eliminated through the program is thus $(0.35) * (9{,}817$ pupils in Group 1 schools$) + (0.09) * (19{,}493$ Pupils in Group 2 and 3 schools$) = 5190$ infections.

This is nearly one infection eliminated per treated child in Group 1 schools. Even this figure underestimates the actual total treatment effect of the program by excluding any benefits to schools more than six kilometers from treatment schools, and benefits for school-age children not enrolled in school, other community members not of school age—such as the pre-primary children discussed above—and people who live in villages bordering the study area, whom we did not survey.

As discussed in Section 2, externalities are likely to operate over larger distances for schistosomiasis than for geohelminths. In fact, the cross-school externality effects are mainly driven by reductions in moderate-to-heavy schistosomiasis infections (Table VII, regression 4), while cross-school geohelminth externalities are negative and marginally significant within three kilometers but not significantly different than zero from three to six kilometers (regression 7). The within-school effect is driven by geohelminth infections (coefficient estimate -0.10, standard error 0.04, regression 8), while the within-school schistosomiasis externalities are negative but insignificant (regression 5).

Finally, the coefficient estimates on interaction terms between treatment group and local treatment intensity are not statistically significantly different than zero for moderate-to-heavy schistosomiasis infections (Table VII, regression 6), but the interaction between treatment group and local treatment intensity from zero to three kilometers is negative and significant for moderate-to-heavy geohelminth infections (regression 9). In other words, pupils in comparison and treatment schools benefit similarly from proximity to treatment schools in terms of reduced schistosomiasis infection, but treatment school pupils experience larger cross-school geohelminth externalities than comparison pupils.[41]

6. DEWORMING TREATMENT EFFECTS ON SCHOOL PARTICIPATION

This section argues that deworming increased school participation in treatment schools by at least seven percentage points, a one-quarter reduction in to-

[41]For schistosomiasis, one explanation for this results is that cross-school externalities are sufficiently large to reduce infection levels below the moderate-to-heavy threshold for many pupils in both treated and comparison schools, and as a result coefficient estimates on the interaction terms are not significant.

tal school absenteeism.[42] Deworming may have improved school participation by allowing previously weak and listless children to attend school regularly or by improving children's ability to concentrate, which may have made attending school increasingly worthwhile relative to other activities, such as agricultural labor, staying at home, or fishing.

As with the health impacts, deworming creates externalities in school participation both within and across schools; after accounting for externalities we estimate that overall school participation in this area likely increased by at least 0.14 years of schooling per pupil actually treated through the program. This effect is larger than would be expected from nonexperimental estimates of the correlation between worm burden and school participation, as we discuss below.

Our sample consists of all pupils enrolled in school or listed in the school register during the first term in 1998.[43] Since many pupils attend school erratically, and the distinction between an absent pupil and a dropout is often not clear from school records, it is difficult to distinguish between dropping out and long-term absenteeism; moreover, measuring pupil attendance conditional on not dropping out is unattractive since dropping out is endogenous. We therefore focus on a comprehensive measure of school participation: a pupil is considered a participant if she or he is present in school on a given day, and a nonparticipant if she or he is not in school on that day. Since school attendance records are often poorly kept, school participation was measured during unannounced school visits by NGO field workers. Schools received an average of 3.8 school participation check visits per year in 1998 and 1999. Note that since the days of medical treatment were pre-announced, and the school

[42]School participation in the area is irregular, and the large effect we estimate is consistent with the hypothesis that many children are at the margin of whether or not to attend school given the cost of school fees and uniforms, low school quality, and perceived declining returns to education (Mensch and Lloyd (1997)). Further evidence that many children are at the margin of whether to attend school is provided by a program in the same region that paid for required school uniforms, increasing school participation by 15 percent (Kremer, Moulin, and Namunyu (2002)).

[43]Since many pupils who were recorded as dropouts in early 1998 re-enrolled in school at some point during the 1998 or 1999 school years, we include them in the sample. However, many initial dropouts were not assigned a grade by the NGO field staff, complicating the analysis of participation rates by grade. Such pupils are assigned their own grade indicator variable in Table IX. Some pupils have missing year of birth information due to absence from school on days of questionnaire or exam administration, and certain assumptions need to be made regarding the treatment assignment status of girls with missing age information (since older girls were supposed to be excluded from treatment). Girls in treatment schools in preschool and grades 1, 2, and 3 are assumed to be eligible for treatment, while those in grades 7 and 8 are assumed not to be, since all but a small fraction of girls in these grades meet the respective age eligibility criterion. We do not know if girls with missing ages in grades 4, 5, and 6 were younger than 13 and hence were supposed to receive treatment, and therefore we drop them from the sample, eliminating 99 girls from the sample of approximately 30,000 children. An additional 119 pupils are dropped from the sample due to both missing age and sex information.

participation figures do not include attendance on these days, effects on attendance are not due to children coming to school in the hope of receiving medicine.

6.1. *School Participation Differences across Treatment and Comparison Schools*

Before proceeding to formal estimation using equations (1) and (3), we first present differences in school participation across the project groups and through time. Since these do not take cross-school externalities into account, they potentially underestimate overall treatment effects. Among girls younger than thirteen years old and all boys, the difference in school participation for the five post-treatment participation observations in the first year after medical treatment is 9.3 percentage points, and this is significantly different than zero at 99 percent confidence (Table VIII). The difference is larger among boys and young girls than among the older girls (5.7 percentage points), which is consistent with the fact that a far smaller proportion of older girls actually received medical treatment (Table III).

The differences in 1999 school participation for boys and younger girls are also large and significantly different than zero at 90 percent confidence for both Group 1 (1998 and 1999 treatment schools) and Group 2 (1999 treatment schools), at 5.0 and 5.5 percentage points, respectively. Average school participation rates fall during the second year of the study as children from the original sample—and especially those in the older grades—left school through graduation or dropping-out.

One possible explanation for the smaller impact of the program on school participation in 1999 is the lower proportion of pupils taking deworming drugs compared to 1998 (Table III), which should reduce both treatment effects on the treated and externality effects. The larger participation differences between treatment and comparison schools in 1998 may also have been due to the widespread El Niño flooding in this region in early 1998, which substantially increased worm loads between early 1998 and early 1999 (to see this, compare Tables II and V). Finally, the difference may be due in part to chance: we cannot reject the hypothesis that gaps between treatment and comparison schools in 1998 and 1999 are the same.

The time pattern of school participation differences is consistent with a causal effect of deworming on school participation. Figure 1 presents school participation rates from May 1998 to November 1999 for girls under thirteen and for all boys. Diamonds represent the differences in average school participation between Group 1 and Group 3 schools, and squares represent the difference between Group 2 and Group 3 schools. School participation rates for Group 1 schools are consistently higher than rates in Group 3 schools in both 1998 and 1999, and the gap stands at nearly ten percentage points by November 1999. Group 2 schools have lower school participation than Group 3 schools in 1998 when both groups were comparison schools, but begin to show

TABLE VIII
SCHOOL PARTICIPATION, SCHOOL-LEVEL DATA[a]

	Group 1 (25 schools)	Group 2 (25 schools)	Group 3 (25 schools)		
<u>*Panel A:*</u>					
First year post-treatment (May 1998 to March 1999)	*1st Year Treatment*	*Comparison*	*Comparison*	*Group 1 − (Groups 2 & 3)*	*Group 2 − Group 3*
Girls <13 years, and all boys	0.841	0.731	0.767	0.093***	−0.037
				(0.031)	(0.036)
Girls ≥13 years	0.864	0.803	0.811	0.057**	−0.008
				(0.029)	(0.034)
Preschool, Grade 1, Grade 2 in early 1998	0.795	0.688	0.703	0.100***	−0.018
				(0.037)	(0.043)
Grade 3, Grade 4, Grade 5 in early 1998	0.880	0.789	0.831	0.070***	−0.043
				(0.024)	(0.029)
Grade 6, Grade 7, Grade 8 in early 1998	0.934	0.858	0.892	0.059***	−0.034
				(0.021)	(0.026)
Recorded as "dropped out" in early 1998	0.064	0.050	0.030	0.022	0.020
				(0.018)	(0.017)
Females[b]	0.855	0.771	0.789	0.076***	−0.018
				(0.027)	(0.032)
Males	0.844	0.736	0.780	0.088***	−0.044
				(0.031)	(0.037)
<u>*Panel B:*</u>					
Second year post-treatment (March to November 1999)	*2nd Year Treatment*	*1st Year Treatment*	*Comparison*	*Group 1 − Group 3*	*Group 2 − Group 3*
Girls <13 years, and all boys	0.713	0.717	0.663	0.050*	0.055*
				(0.028)	(0.028)
Girls ≥14 years[c]	0.627	0.649	0.588	0.039	0.061*
				(0.035)	(0.035)
Preschool, Grade 1, Grade 2 in early 1998	0.692	0.726	0.641	0.051	0.085**
				(0.034)	(0.034)
Grade 3, Grade 4, Grade 5 in early 1998	0.750	0.774	0.725	0.025	0.049**
				(0.023)	(0.023)
Grade 6, Grade 7, Grade 8 in early 1998	0.770	0.777	0.751	0.020	0.026
				(0.027)	(0.028)
Recorded as "dropped out" in early 1998	0.176	0.129	0.056	0.120*	0.073
				(0.063)	(0.053)
Females[b]	0.716	0.746	0.648	0.067**	0.098***
				(0.027)	(0.027)
Males	0.698	0.695	0.655	0.043	0.041
				(0.028)	(0.029)

[a]The results are school averages weighted by pupil population. Standard errors in parentheses. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence. The participation rate is computed among all pupils enrolled in the school at the start of 1998. Pupils who are present in school on the day of an unannounced NGO visit are considered participants. Pupils had 3.8 participation observations per year on average. The figures for the "Preschool–Grade 2"; "Grade 3–5"; "Grade 6–8"; and "Dropout" rows are for girls <13 years, and all boys.

[b]396 pupils in the sample are missing information on gender. For this reason, the average of the female and male participation rates does not equal the overall average.

[c]Examining girls ≥14 years old eliminates the cohort of girls in Group 1 schools (12 year olds in 1998) who were supposed to receive deworming treatment in 1998.

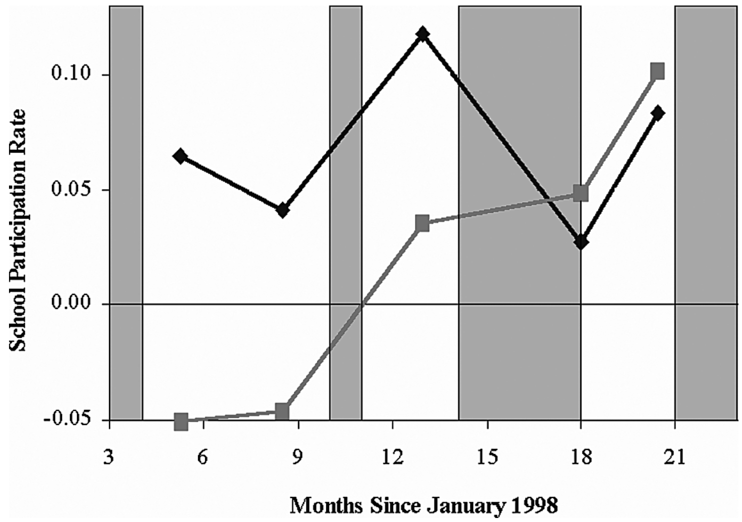

FIGURE 1.—School participation rate May 1998 to November 1999 for girls under 13 years old and for all boys (difference between Group 1 and Group 3 (diamonds), and difference between Group 2 and Group 3 (squares)).[a]

[a]The shaded regions are periods in which medical treatment was being provided (in March–April and November 1998 to Group 1 schools, and March–June and October–November 1999 to Group 1 and Group 2 schools).

participation gains in early 1999. Participation in Group 2 schools is substantially greater than in Group 3 schools by mid-1999 when the first round of 1999 treatment was concluded. These gains resulted primarily from a greater proportion of pupils with participation above 80 percent, although there were also substantially fewer dropouts (results not shown).

The school participation gains are particularly large among the youngest pupils: in 1998 the average difference in participation between treatment and comparison groups for preschool through grade 2 was 10.0 percentage points (significantly different than zero at 99 percent confidence), while for pupils in grades 6 to 8 it was 5.9 percentage points, and in 1999 the comparable gains for Group 2 pupils were 8.5 percentage points and 2.6 percentage points, respectively. The larger impact of treatment in lower grades may partially result from higher rates of moderate-to-heavy infection among younger pupils (Table II). It is also possible that school participation is more elastic with respect to health for younger pupils; many Kenyan children drop out before reaching the upper primary grades, so older children who remain in school may be the most academically serious and determined to attend school despite illness.

Untreated pupils in Group 1 (1998 treatment) had higher school participation than their counterparts in Group 2 schools who were later untreated during 1999, consistent with deworming externalities on school participation. Among girls under thirteen years old and all boys, May 1998 to March

1999 school participation was 8.0 percentage points greater among untreated Group 1 pupils, which is significantly different than zero at 95 percent confidence (Table VI, Panel C). Group 1 pupils who were treated in 1998 had 6.4 percentage points higher May 1998 to March 1999 school participation than Group 2 pupils who were treated in 1999.[44]

The large participation gains among older girls—who were not supposed to be treated through the program—in 1998 and 1999 also suggest that school participation externality benefits were substantial (Table VIII). Although the 1998 gains among older girls could have been driven in part by nontrivial rates of medical treatment, there were also large participation gains among older girls in Group 2 schools in 1999 despite the fact that only ten percent of them received medical treatment (Table III). An alternative, nonhealth explanation for the participation gains among older girls is that the improved school participation of younger siblings allowed them to attend school more regularly, as we discuss below.

6.2. *Estimating Overall School Participation Impacts*

School participation externality estimates across schools using individual-level data are presented in Table IX. The dependent variable is average individual school participation in either the first year (May 1998 to March 1999) or the second year (April 1999 to November 1999) of the project. Regressions 1 and 2 present "naïve" treatment effects that ignore the possibility of externalities. The average school participation gain for treatment schools relative to comparison schools across both years of the project is 5.1 percentage points, and this is significantly different than zero at 99 percent confidence (regression 1). Point estimates are 6.2 percentage points for the first year of treatment and 4.0 percentage points for the second year, with significance levels of 99 percent and 90 percent, respectively (regression 2), although confidence intervals are wide enough that we cannot reject the hypothesis that the effect is the same in both years. The magnitude of the effects remains nearly unchanged when pupils initially recorded as dropouts in early 1998 are excluded from the sample (results not shown).

The ratio of externalities to direct effects is likely to be smaller for measured school participation than for measured worm load, since the ratio of externalities to direct effects is very low immediately after treatment but then asymptotes to one. As we discussed in Section 4, worm load is measured between three months to a year after deworming treatment, while school participation

[44]It may seem odd that the point estimate of the absolute increase in school participation is greater for the untreated, but it is worth noting that the proportional decline in school nonparticipation was one-third for the treated while the decline among the untreated was one-fourth, and that we cannot reject the hypothesis that the difference for treated pupils is somewhat larger than for untreated pupils.

TABLE IX
SCHOOL PARTICIPATION, DIRECT EFFECTS AND EXTERNALITIES[a]
DEPENDENT VARIABLE: AVERAGE INDIVIDUAL SCHOOL PARTICIPATION, BY YEAR

	OLS (1)	OLS (2)	OLS (3)	OLS (4) May 98– March 99	OLS (5) May 98– March 99	OLS (6) May 98– March 99	IV-2SLS (7) May 98– March 99
Moderate-heavy infection, early 1999						−0.028***	−0.203*
						(0.010)	(0.094)
Treatment school (T)	0.051***						
	(0.022)						
First year as treatment school (T1)		0.062***	0.060***	0.062*	0.056***		
		(0.015)	(0.015)	(0.022)	(0.020)		
Second year as treatment school (T2)		0.040*	0.034*				
		(0.021)	(0.021)				
Treatment school pupils within 3 km (per 1000 pupils)			0.044**		0.023		
			(0.022)		(0.036)		
Treatment school pupils within 3–6 km (per 1000 pupils)			−0.014		−0.041		
			(0.015)		(0.027)		
Total pupils within 3 km (per 1000 pupils)			−0.033**		−0.035*	0.018	0.021
			(0.013)		(0.019)	(0.021)	(0.019)
Total pupils within 3–6 km (per 1000 pupils)			−0.010		0.022	−0.010	−0.021
			(0.012)		(0.027)	(0.012)	(0.015)
Indicator received first year of deworming treatment, when offered (1998 for Group 1, 1999 for Group 2)					0.100***		
					(0.014)		
(First year as treatment school Indicator) ∗ (Received treatment, when offered)					−0.012		
					(0.020)		
1996 district exam score, school average	0.063***	0.071***	0.063***	0.058	0.091**	0.021	0.003
	(0.021)	(0.020)	(0.020)	(0.032)	(0.038)	(0.026)	(0.023)

is measured continuously beginning immediately following treatment, including the period when the ratio of externalities to direct effects is likely to be low.[45]

[45]The cross-school externalities for school participation may also be weaker than worm infection externalities because only schistosomiasis has robust health externalities across schools, and moderate to heavy schistosomiasis infection is rarer than geohelminth infection (only seven percent of Group 1 pupils had moderate to heavy schistosomiasis infections prior to treatment, while

TABLE IX
(CONTINUED)

	OLS (1)	OLS (2)	OLS (3)	OLS (4) May 98– March 99	OLS (5) May 98– March 99	OLS (6) May 98– March 99	IV-2SLS (7) May 98– March 99
Grade indicators, school assistance controls, and time controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R^2	0.23	0.23	0.24	0.33	0.36	0.28	–
Root MSE	0.273	0.272	0.272	0.223	0.219	0.150	0.073
Number of observations	56487	56487	56487	18264	18264	2327	49 (schools)
Mean of dependent variable	0.747	0.747	0.747	0.784	0.784	0.884	0.884

[a]The dependent variable is average individual school participation in each year of the program (Year 1 is May 1998 to March 1999, and Year 2 is May 1999 to November 1999); disturbance terms are clustered within schools. Robust standard errors in parentheses. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence. Additional explanatory variables include an indicator variable for girls <13 years and all boys, and the rate of moderate-heavy infections in geographic zone, by grade (zonal infection rates among grade 3 and 4 pupils are used for pupils in grades 4 and below and for pupils initially recorded as drop-outs as there is no parasitological data for pupils below grade 3; zonal infection rates among grade 5 and 6 pupils are used for pupils in grades 5 and 6, and similarly for grades 7 and 8). Participation is computed among all pupils enrolled at the start of the 1998 school year. Pupils present during an unannounced NGO school visit are considered participants. Pupils had approximately 3.8 attendance observations per year. Regressions 6 and 7 include pupils with parasitological information from early 1999, restricting the sample to a random subset of Group 1 and Group 2 pupils. The number of treatment school pupils from May 1998 to March 1999 is the number of Group 1 pupils, and the number of treatment school pupils after March 1999 is the number of Group 1 and Group 2 pupils.

The instrumental variables in regression 7 are the Group 1 (treatment) indicator variable, treatment school pupils within 3 km, treatment school pupils within 3–6 km, and the remaining explanatory variables. We use the number of girls less than 13 years old and all boys (the pupils eligible for deworming in the treatment schools) as the school population for all schools.

We estimate equation (1) in regression 3 and find that each additional thousand (potentially age and sex eligible) pupils attending treatment schools within three kilometers leads to an increase of 4.4 percentage points in average school participation (significant at 95 percent confidence). The effect of treatment pupils located between three to six kilometers is negative, but not significantly different than zero. Given the number of Group 1 pupils and Group 2 pupils within three kilometers, and between three to six kilometers, of the average primary school, the results of regression 3 imply that school

over thirty percent had some moderate to heavy geohelminth infection (Table II)). The coefficient estimates on the interactions between treatment indicators and distance to lake Victoria—which is highly correlated with the prevalence of schistosomiasis in this area (Table II)—are not significantly different than zero, indicating that school participation treatment effects among those infected with both schistosomiasis and geohelminths are not considerably larger than the effects for children with geohelminth infections alone, and supporting the view that school participation effects work mainly through geohelminths.

participation was approximately 2.0 percentage points (standard error 1.3 percentage points) higher on average throughout this area in 1998 and 1999 due to deworming externalities, which is marginally statistically significant.[46] Regression 3 also implies that the total effect of deworming on school participation in treatment schools was 7.5 percentage points (standard error 2.7 percentage points) over 1998 and 1999.

To estimate the overall school participation gain due to the program, recall that the program increased school participation by about 2.0 percentage points on average among pupils in comparison schools, while children in treatment schools had about 7.5 percentage points higher participation. For every two treated children in a treatment school, there was almost exactly one untreated child on average in 1998 and 1999, and for each child in a treatment school there was one comparison school child for 1998 and 1999 (since one-third of schools were treated in 1998 and two-thirds in 1999). Hence treating one child led to an estimated lower bound increase in school participation of $(1*0.075)+(0.5*0.075)+(1.5*0.020)=0.14$ school years (standard error 0.05).

To estimate within school externalities using equation (3) we can only use data from the first year of treatment, and so for comparison purposes, regression 4 presents the basic specification for the first year of data, and estimates a 6.2 percentage point school participation gain. Within-school participation externality benefits were positive and statistically significant at 99 percent confidence (5.6 percentage points) for untreated pupils in the treatment schools in the first year of the program (regression 5), and there is no significant difference in school participation rates between treated and untreated pupils in these schools (which is consistent with the externality results from Table VI, Panel C, reported above). In this restricted 1998 sample, the estimated cross-school externality effects are statistically insignificant.[47]

6.3. *Comparing Experimental and Nonexperimental Estimates*

Pupils who were moderately or heavily infected in early 1999 had 2.8 percentage points lower school participation over the period May 1998 to March

[46] Unlike infection rates, coefficient estimates on the interactions between school treatment indicators and local treatment school pupil densities are not significantly different than zero for school participation (results not shown), so we do not consider differential externality benefits for the three project treatment groups in the calculation of overall program impacts. There are at least two reasons why the cross-school externality relationships differ. First, if school participation varies continuously with infection levels, the threshold effects found for moderate to heavy infections might not apply. Second, school attendance is measured continuously over the study period, while infection levels are measured only once, up to one year after initial treatment.

[47] We obtain qualitatively similar results using the instrumental variables approach discussed in Section 5, which compares outcomes for older girls (who were largely excluded from deworming treatment) across the treatment and comparison schools to estimate the within-school externality. The IV results for within school externalities for school participation are insignificant, but we also cannot reject the hypothesis that the IV estimates are the same as the OLS results in Table IX (refer to Appendix Table AIV, regression 4).

1999 (Regression 6, Table IX). This nonexperimental estimate is restricted to the subsample of 2327 pupils in grades three to eight for whom there is 1999 parasitological data, and we thus lack information on the preschool, grade 1, and grade 2 pupils that exhibit the largest experimental treatment effect estimates. In contrast, an instrumental variable specification—which imposes the condition that all school participation gains work through changes in measured worm infection status—suggests that each moderate to heavy worm infection leads to 20.3 percentage points lower school participation on average (regression 7). The instrumental variables in regression 7 are the Group 1 (treatment) indicator variable, treatment school pupils within 3 km, and treatment school pupils within 3–6 km.

There are at least three reasons why the IV estimates of the impact of moderate-heavy infection on school participation are substantially larger than OLS estimates. First, since we measure infection up to a year after treatment, when many pupils will already have been reinfected with worms, the difference in infection levels between treated and untreated pupils was likely much greater on average over the interval from deworming treatment to the parasitological exam than it was at the time of the parasitological exam (given the documented efficacy of the drugs and high reinfection rates). As we discussed in Section 4, the parasitological exam data almost certainly understates the total number of moderate to heavy infections eliminated as a result of the program immediately after treatment. If 99 percent of pupils with moderate-to-heavy infections were in fact initially cleared of infection, the implied school participation gain for each pupil cleared of moderate to heavy infection (presented in regression 7) would be cut approximately in half.

Second, the exclusion restriction—that the program only affects pupils' school attendance by changing their health—may not hold, due to complementarities in school participation. For example, if the pre-schoolers, first-graders, and second-graders for whom we estimate the largest school participation effects stay home sick with worms in the comparison schools, their older sisters may also stay home to take care of them, and this may partly explain the relatively large treatment effects we find for older girls.[48] More generally, there may be complementarity in school attendance if children are more inclined to go to school if their classmates are also in school, so school participation gains in treatment schools may partially reflect increased school participation among children who were not infected with worms. Such effects would influence the impact of a large-scale deworming program on school participation and are captured in a prospective evaluation (like ours) in which treatment is randomized at the school level, but they would not be picked up in an individual-level regression of school participation on worm levels, or in a prospective study in which treatment is randomized at the individual level.

[48]Since we do not have data on family relationships, we cannot directly test this hypothesis in this setting.

A final reason why instrumental variable estimates of the deworming effect are larger than suggested by our nonexperimental estimates is attenuation bias due to error in measuring the severity of disease.[49]

7. DEWORMING TREATMENT EFFECTS ON TEST SCORES

Deworming could improve test scores both by increasing time spent in school and by improving learning while pupils are in school, but could also potentially reduce test scores through congestion or negative peer effects. We describe these various positive and negative mechanisms in Section 7.1, and then present the test score results in 7.2.

7.1. *Mechanisms Linking Deworming and Test Score Performance*

Deworming could potentially increase test scores by increasing the total amount of time spent in school, but this effect is likely to be weak given the observed impact of deworming on school participation and the cross-sectional relationship between school participation and test performance. In 1998 and 1999, ICS administered English, Mathematics, and Science-Agriculture exams to pupils in grades 3 to 8. Restricting attention to these grades reduces the sample size in Table X relative to Table IX. Exams were modelled on those given by the district office of the Ministry of Education, and prepared using the same procedure. The average score across all subjects is employed as the principal test score outcome measure for each set of tests, although the basic results are

[49]Measurement error in binary variables leads to bias toward zero in the OLS specification, provided errors are not too extreme (Aigner (1973), Kane et al. (1999)); the technical condition is that Pr(Type I Error) + Pr(Type II Error) < 1, which is reasonable in our case. Unfortunately, measurement error in binary variables can also lead to bias away from zero in IV estimates, which would lead us to somewhat overstate the effect of worm infection on attendance in Table IX, regression 7; the effect of a moderate-heavy worm infection on school participation is thus likely to lie between the OLS and IV coefficient estimates. Measurement error could take several forms: pure measurement error performing egg counts in the lab; time variation in worm burden, so that those who were moderately to heavily infected in early 1999 were not necessarily the same ones who were most heavily infected over the course of the school year; coarseness in our binary measure of worm burden; heterogeneity in the impact of different worm species on school participation; and interactions among worms that are not captured by our measure, so that some individuals who are classified as having multiple light worm infections in fact suffer substantial morbidity. Moreover, epidemiologists have argued that there is an imperfect relationship between worm egg counts—the standard measure of infection intensity—and actual worm infection burden (Medley and Anderson (1985)), further exacerbating error. Heterogeneous treatment effects may also interact with sample attrition to further exacerbate estimation biases because those pupils for whom high measured worm burdens are not associated with absenteeism are more likely to be in school on the day of the parasitological exam and hence to make it into our sample. Note, however, that this measurement error and resulting bias does not affect our main experimental estimates of program impacts presented above, but does help account for the difference between the experimental and nonexperimental estimates.

TABLE X
ACADEMIC EXAMINATIONS, INDIVIDUAL-LEVEL DATA[a]

	Dependent variable: ICS Exam Score (normalized by standard)		
	(1)	(2)	(3) Among those who filled in the 1998 pupil survey
Average school participation (during the year of the exam)	0.63***		
	(0.07)		
First year as treatment school (T1)		−0.032	−0.030
		(0.046)	(0.049)
Second year as treatment school (T2)		0.001	0.009
		(0.073)	(0.081)
1996 District exam score, school average	0.74***	0.71***	0.75***
	(0.07)	(0.07)	(0.07)
Grade indicators, school assistance controls, and local pupil density controls	Yes	Yes	Yes
R^2	0.14	0.13	0.15
Root MSE	0.919	0.923	0.916
Number of observations	24958	24958	19072
Mean of dependent variable	0.020	0.020	0.039

[a]Each data point is the individual-level exam result in a given year of the program (either 1998 or 1999); disturbance terms are clustered within schools. Linear regression, robust standard errors in parentheses. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence. Regression 3 includes only pupils who completed the 1998 Pupil Questionnaire. Additional explanatory variables include an indicator variable for girls <13 years and all boys, and the rate of moderate-to-heavy infections in geographic zone, by grade (zonal infection rates among grade 3 and 4 pupils are used for pupils in grades 4 and below and for pupils initially recorded as dropouts as there is no parasitological data for pupils below grade 3; zonal infection rates among grade 5 and 6 pupils are used for pupils in grades 5 and 6, and similarly for grades 7 and 8). The local pupil density terms include treatment school pupils within 3 km (per 1000 pupils), total pupils within 3 km (per 1000 pupils), treatment school pupils within 3–6 km (per 1000 pupils), and total pupils within 3–6 km (per 1000 pupils). We use the number of girls less than 13 years old and all boys (the pupils eligible for deworming in the treatment schools) as the school population for all schools.

The ICS tests for 1998 and 1999 were similar in content, but differed in two important respects. First, the 1998 exam featured multiple-choice questions while the 1999 test featured short answers. Second, while each grade in 1998 was administered a different exam, in 1999 the same exam—featuring questions across a range of difficulty levels—was administered to all pupils in grades 3 to 8. Government district exams in English, Math, Science-Agriculture, Kiswahili, Geography-History, Home Science, and Arts-Crafts were also administered in both years. Treatment effect estimates are similar for both sets of exams (results not shown).

unchanged if subjects are examined separately (regressions not shown). For both 1998 and 1999, test scores were normalized to be mean zero and standard deviation one among comparison pupils initially enrolled in the same grade in early 1998.

A one percentage point increase in measured school participation is associated with a 0.63 standard deviation increase in test scores (Table X, regression 1). The coefficient estimate suffers from attenuation bias due to sampling error since the school participation measure for each individual is the average of only 3.8 participation observations per year, but it is straightforward to correct since the participation rate and the number of participation observations are known for each pupil.[50] The corrected coefficient estimate is 2.17, implying that a ten percentage point gain in attendance is associated with a 0.217 standard deviations higher score on the ICS exam. If deworming leads to test score gains solely through improvements in attendance, and average school participation in treatment schools exceeds that in comparison schools by approximately 5.1 percentage points as a result of deworming over 1998 and 1999 (Table IX), then the estimated "effect" of deworming on test scores in the absence of omitted variable bias would be $(0.051) * (2.17)$, or approximately 0.11 standard deviations.

However, the coefficient estimate on average school participation in this regression is likely to overstate the true impact of increased participation on test scores for two reasons. First, it reflects not only the causal impact of higher participation on test scores, but also unobserved pupil characteristics correlated with both test scores and school participation. Second, in a related point, the coefficient estimate on school participation is likely to reflect the impact of better attendance over the course of a child's entire school career, whereas this study only examines attendance gains over one or two years; 5.1 percentage points higher school participation for two years translates into fewer than twenty additional days of schooling, and this might plausibly have a limited effect on academic performance. For example, if omitted variable bias accounted for half of the observed correlation between test scores and school attendance, and if the remainder of the correlation reflects the effects of the past five years of schooling on academic performance, then one would expect that increasing attendance by 5.1 percentage points for two years would increase test scores by less than 0.02 standard deviations, a very small effect.

The second channel through which deworming could increase scores is by improving the efficiency of learning per unit of time spent in school. However, since severe anemia is rare in this area and there were only small differences in anemia between treatment and comparison schools (Table V), the most frequently hypothesized link between worm infections and cognitive performance may not have been operative during the study. Some evidence that the program did not increase the efficiency of learning is provided by a battery of cognitive exams—including picture search, Raven matrix, verbal fluency, digit span, Spanish learning, and a "dynamic" test using syllogisms—which were conducted in all three groups of schools during 2000. Deworming treatment effects are not significantly different than zero for any component of the cognitive exam (results available upon request).

[50]The true coefficient estimate on average annual attendance β is related to the coefficient estimate b by the standard attenuation bias formula: $\beta = b(\sigma_T^2/(\sigma_T^2 - \sigma_S^2))$, where the sampling variance of average annual participation is σ_S^2, and the total variance in average annual school participation is σ_T^2. We take into account that the number of participation observations differs across individuals in calculating the attenuation bias correction.

On the other hand, deworming could potentially have reduced test scores in treatment schools through congestion and peer effects. Classrooms were more crowded in treatment schools as previously ill children attended school more regularly, and the presence of these additional pupils in the classroom may have imposed negative learning externalities on other pupils.[51]

7.2. *Test Score Results*

The estimated differences in test scores between pupils in treatment and comparison schools are −0.032 standard deviations for the first year post-treatment and 0.001 standard deviations for the second year, neither of which is significantly different than zero (Table X, regression 2). The average cross-school deworming externality effect is statistically insignificant at −0.049 (standard error 0.052), and within-school externality effect estimates are also statistically insignificant (results not shown).

The results could potentially have been affected by differential attrition across treatment and comparison schools, if the additional treatment school pupils who participated in the exam after deworming were below-average performers. The fact that 85 percent of Group 1 pupils took the 1998 ICS exams, compared to 83 percent of Group 2 and Group 3 pupils, suggests that this is a possibility, although the attrition bias is likely to be small.[52] To address this issue, we restrict the sample to pupils who were administered the 1998 pupil questionnaire, eliminating over twenty percent of the sample and much of the potential exam participation bias since nearly identical proportions of these pupils took the ICS exam in treatment and comparison schools. Treatment effect estimates using this restricted sample are similar to those using the complete sample and remain insignificantly different than zero at traditional confidence levels (Table X, regression 3), suggesting that at least among this subsample, deworming did not substantially raise test scores.

It remains possible that benefits may have accrued disproportionately among the 15 percent of pupils who missed the ICS exam, especially if they suffered from the most intense helminth infections. However, we do not find a strong association between worm burden and the likelihood of missing the exam within the sample of students in the parasitological sample (results not shown). [53]

[51] Assuming that the relationship between class size and academic outcomes for Israeli schools in Angrist and Lavy (1999) holds in Kenya, deworming participation gains of the magnitude we found would lead to a drop of 0.02–0.05 standard deviations in average exam scores (calculations available from the authors upon request).

[52] Lee's (2002) bounds on the deworming treatment effect are near zero and statistically insignificant, both for this test and for the cognitive exams, given the relatively small difference in attrition between treatment and comparison schools (results available upon request).

[53] A subset of pupils who did not take the 1998 ICS exam (including dropouts) were followed up in 20 deworming schools and encouraged to sit for the exam, allowing us to impute test scores for dropouts. In total, 214 pupils were administered the follow-up exam in these schools. Among

A higher grade promotion rate would also have resulted if deworming increased learning among weak students who did not take ICS exams. Although promotion rates in treatment schools between 1998 and 1999 are in fact two percentage points higher than in comparison schools, this difference is not significantly different than zero (results not shown).

Given the observed cross-sectional relationship between participation and test scores, the absence of a strong time-in-school effect on test scores may not be surprising. However, the data do not support the hypothesis of a strong effect on the efficiency of learning per unit of time in school for the subsample who took the test. It is worth mentioning that several other primary school interventions in this region of Kenya—including textbook provision (Glewwe, Kremer, and Moulin (1999)) and school grant provision—have also had limited success in improving academic test scores. Note that there is an analogous result in the literature on health and labor productivity in less developed countries, namely, that although poor health typically reduces hours of labor supply, the existing empirical evidence on the impact of poor health on wage rates—a proxy for individual productivity—is largely inconclusive (Strauss and Thomas (1998)).

8. COST EFFECTIVENESS AND WELFARE ANALYSIS

We explore the controversy over whether mass school-based deworming treatment should be a public policy priority for the poorest countries using four different approaches. Under the *health cost effectiveness approach*, health projects are considered cost-effective up to some threshold cost per Disability-Adjusted Life Year (DALY) saved, perhaps $25 to $100 per DALY in the poorest countries. We also consider the *educational cost effectiveness* of promoting school participation through deworming rather than through alternative educational interventions. The *human capital investment approach* estimates the rate of return to deworming in future earnings. The *externality approach* attempts to identify the subsidy that would lead individuals to fully internalize treatment externalities.

The health externalities and school participation effects examined in this paper turn out to play an important role under a variety of approaches. For example, as discussed below, we find that under the health cost effectiveness approach, treatment of schistosomiasis is extremely cost effective, but that a naïve

grade 3–8 pupils with missing ICS exams, similar proportions were administered the follow-up exam in Group 1 (treatment) schools—34 percent—and Group 2 and 3 (comparison) schools—32 percent—suggesting that attrition bias is unlikely to be large. Missing 1998 ICS test score data was imputed in two steps. First, the normalized test scores of the follow-up pupils were regressed on a set of variables for grade, geographic zone, and school assistance group (assistance from other NGO projects) separately for Groups 1, 2, and 3 schools. Second, missing test score values for other pupils with missing tests are imputed as predicted values of this regression, again separately for Group 1, 2, and 3 schools. Treatment effect estimates remain insignificantly different than zero using this augmented sample (results not shown).

estimate ignoring externalities would severely underestimate its cost effectiveness. Treatment of geohelminths would not meet standard cost-effectiveness criteria in the poorest countries based on its health impact alone, but is extremely cost effective relative to other ways of increasing school participation that have also been examined using prospective evaluations in this part of Kenya. While estimates of the long-run labor market impact of deworming are of course speculative, our best estimate is that deworming is an excellent human capital investment given its impact on school participation, and that the externalities from deworming justify fully subsidizing treatment.

8.1. *Health Cost Effectiveness*

Annual government expenditure on health in Kenya was approximately five U.S. dollars per capita from 1990 to 1997 (World Bank (1999)), so mass deworming is only one of many health interventions competing for scarce public resources. For example, the vaccination rate against measles and DPT (diptheria, pertussis, and tetanus) among Kenyan infants of less than one year of age was just 32 percent in 1997 (World Bank (1999)), and these vaccinations are thought to be highly cost effective, at only 12 to 17 U.S. dollars per disability-adjusted life year (DALY) saved.

We use deworming program cost estimates from the Partnership for Child Development (PCD (1999)), which reports costs of 0.49 US dollars per pupil per year in a large-scale government intervention in Tanzania. These costs are probably more relevant for potential large scale programs than the PSDP costs, since the PSDP was not able to fully realize economies of scale in drug purchase and delivery, and since it is difficult to disentangle evaluation and delivery costs in the PSDP. [54]

According to the World Health Organization, schistosomiasis infections are associated with much greater disease burden per infected individual than geohelminths, on average.[55] Approximately 18 percent of those infected with helminths globally are thought to suffer morbidity as a result of their infection, and in our cost-effectiveness calculations we assume that the entire disease

[54]Excluding the costs most clearly linked to the evaluation yields a cost per pupil treated through the PSDP in 1999 of 1.46 US dollars, with nearly half of this cost in drug purchases. However, the PSDP used trained nurses, held meetings to explain consent procedures, individually recorded the names of all pupils taking medicine, and was headquartered in Busia town, several hours drive away from many project schools. These costs might have been unnecessary in a large-scale program that did not include an evaluation component.

[55]Given data on the burden of disease in WHO (2000), and the number of people infected worldwide, the implied average DALY burden per person infected is 0.0097 for schistosomiasis, 0.0013 for hookworm, 0.0005 for whipworm, and 0.0004 for roundworm.

burden is concentrated among individuals with moderate-to-heavy infections (Bundy et al. (2001)).[56]

In calculating the overall reduction in disease burden due to the program, we consider overall treatment effects (corrected for cross-school externalities) on the treated in treatment schools, externality effects (corrected for cross-school externalities) on the untreated in treatment schools, and externalities for untreated pupils in comparison schools, using results from specifications similar to regression 3 in Table VII, but including the within-school externality terms from Table VII, regression 2 (estimated separately for each type of worm infection). Given the randomized design, we assume that the Group 3 schools (which lack 1999 parasitological data) experienced the same externality benefits as Group 2 schools through early 1999, when neither group had received deworming treatment.

Summing these three components of the treatment effect, the total number of DALY's averted as a result of the program is 649, which translates into a cost of approximately $5 per DALY averted, using the costs of the PCD program in Tanzania. This estimate still ignores the health spillover benefits for other untreated children and adults in the treatment area, thus underestimating cost-effectiveness. Even if the PCD costs were underestimated by a factor of two, deworming would still be among the most cost-effective health interventions for less developed countries.

The externality benefits of treatment (both within and across schools) account for 76 percent of the DALY reduction. A naïve treatment effect estimate that failed to take externalities into account would underestimate program treatment effects, not only because externalities would be missed, but also because gains among the treatment group would be underestimated. Consequently, the naïve estimate would overestimate the cost per DALY averted by a factor of four, leading to the mistaken conclusion that deworming does not meet the strictest cost-effectiveness standards.

The health gains are overwhelmingly attributable to reductions in the prevalence of moderate-to-heavy schistosomiasis: 99 percent of the total DALY reduction is due to averted schistosomiasis. We can separately calculate the cost per DALY averted for the geohelminths; geohelminth infections lead to less morbidity according to the WHO, but are also much cheaper to treat than schistosomiasis. Assuming that drug delivery costs remain the same, but considering only albendazole drug costs in this exercise, the cost per geohelminth DALY averted would be $280, which implies that mass geohelminth treatment in areas without schistosomiasis would not meet strict cost effectiveness cri-

[56]Note that this implies that the burden of disease per infected individual in our sample is greater than the world average, which is appropriate, since levels of moderate-heavy infection are relatively high in this setting.

teria in the poorest countries based solely on health impacts.[57] As discussed below, however, it is likely to be justified on other grounds.

8.2. *Educational Cost Effectiveness*

Deworming was by far the most cost-effective method of improving school participation among a series of educational interventions implemented by ICS in this region of Kenya that were subject to randomized evaluations. ICS has implemented and evaluated textbook provision, grants to school committees, training for teachers, and incentives for teachers based on student test scores and dropout rates. Given that the deworming program increased school participation by approximately 0.14 years per treated child (see Section 6), a large scale program with the Tanzania PCD cost of 0.49 US dollars per child would cost approximately $0.49/0.14, or $3.50 US dollars per additional year of school participation, including both effects on the treated and externality benefits. Aside from deworming, the program which was most successful in increasing school participation was the ICS Child Sponsorship Program (CSP). This program had a number of components, but the key component was substantially reducing the cost of school attendance by paying for the uniforms that Kenyan children are required to wear to school. Even under optimistic assumptions, reducing the cost of schooling in this way costs approximately $99 per additional year of participation induced (refer to Kremer, Moulin, and Namunyu (2002)).[58,59]

8.3. *Deworming as Human Capital Investment*

Given that the PSDP increased school participation but not test scores, and that the empirical literature on effects of schooling examines years of schooling

[57]The cost per DALY for geohelmnith treatment would be lower if albendazole were delivered as part of an ongoing school-based project in areas where schistosomiasis is being treated, although schools would still have to be visited at least once more per year for an additional round of albendazole treatment.

[58]The assumptions about the cost of attracting children to school by reducing the cost of school are optimistic because we assume that CSP's impact on school participation was due entirely to reducing the cost of school. The program also provided textbooks and new classrooms; another evaluation in the same area found that provision of textbooks did not affect school participation. School participation improved immediately through CSP, while classrooms were only provided several years into the CSP program. In any case, if textbook or classroom costs are included in CSP, deworming appears even more cost effective.

[59]Even under the extreme assumptions that uniforms are a pure transfer to parents so the social cost of the CSP is simply the deadweight loss associated with raising tax revenue, and that households obtained no consumption benefits from the deworming program, the social cost of deworming per year of extra school participation is likely to be far lower than that of purchasing school uniforms.

completed rather than days of school participation, any calculation about its effects on human capital accumulation must necessarily be speculative. Nonetheless, a rough calculation suggests that the labor market benefits of deworming may far outweigh their costs. Knight and Sabot (1990) estimate returns to education in Kenya controlling for a wide range of variables including cognitive tests. They decompose the returns to education into a return to cognitive performance (on tests of literacy, numeracy, and reasoning) and a direct return to years of schooling and find that years of schooling alone accounts for approximately forty percent of the 17 percent rate of return to education.[60] If one interprets this as a human capital effect rather than a signalling effect, the return to an additional year of primary school would be approximately 7 percent.

Including externalities, the program increased school participation by 0.14 school years per pupil treated, as discussed in Section 6. Output per worker in Kenya is $570 (World Bank (1999)). To calculate the effect on the net present value of discounted wages, we assume that sixty percent of output per worker in Kenya is wages, and that wage gains from higher school participation are earned over forty years in the workforce and discounted at five percent per year. We assume no wage growth over time. Against this long-run wage increase, we set the opportunity cost of schooling, as children may work rather than attend school. However, children who are heavily infected with worms are unlikely to be particularly productive as workers and may not work at all. We assume that the average primary school child who misses school due to worms is half as productive as the average adult; this is likely to represent an upper bound on productivity of school-aged children in general, let alone sick children.[61] Under these assumptions, deworming increases the net present value of wages by over $30 per treated child at a cost of only $0.49.

Even if increased school participation led to negative congestion externalities by increasing class size, the benefits are large enough to pay for the additional teachers needed to offset the class size increases. To see this, note that the program increased school participation by 0.14 school years per pupil treated, and that with one teacher per thirty pupils, this would require an additional 0.0047 teachers. We estimate teacher compensation at $1942 per year (see Kremer, Moulin, and Namunyu (2002)), so this amounts to $9.06 per treated pupil. So a program that provided deworming and additional teachers

[60]Knight and Sabot (1990) performed this decomposition for returns to secondary education, but it serves as a useful approximation in the absence of a similar decomposition for primary education.

[61]Udry (1996) finds that children's agricultural labor productivity is much less than one-half that of adult agricultural labor productivity in another rural African setting (Burkina Faso). If one assumes that the children who miss school as a result of worms were only one-fifth as productive as adults, then the benefit-cost ratio for the program is still over ten even if the rate of return to an additional school year is only 1.5 percent (calculations not shown).

would generate at least \$30 in future wage benefits at a cost of approximately $\$9.06 + \$0.49 = \$9.55$.[62]

8.4. *Externalities and Optimal Deworming Subsidies*

The externality benefits of deworming in terms of future wages (as calculated in Section 8.3) alone appear to be far larger than the costs of deworming, suggesting a rationale for subsidies even under an orthodox externalities analysis. The total net externality gain (within and across schools) per child treated is then \$15.90 per child treated, over thirty times as large as the \$0.49 cost of deworming. This figure is likely to once again understate the true externality benefits, since it excludes the potentially substantial benefits experienced by school-age and younger children not enrolled in school, by adults in these communities, and individuals in areas bordering the study area. Even if increased school participation led to negative congestion externalities by increasing class size, the positive externalities (\$15.90) are more than fifty percent larger than the cost of additional teachers needed to offset class size increases plus drug costs (\$9.55), suggesting that a large government deworming subsidy is optimal.[63]

To summarize, treatment of schistosomiasis appears to be an extremely cost-effective health intervention under standard health cost effectiveness criteria for less developed countries, although this is less true for the treatment of geohelminths alone. Even in areas with geohelminths but little schistosomiasis, however, deworming is a cost-effective way to boost school participation relative to other educational interventions evaluated in the same area, such as directly reducing the cost of schooling through the provision of school uniforms. It also appears likely that deworming can be justified as a human capital investment. Finally, the externality benefits from deworming in the program we examine are likely sufficient to justify fully subsidizing treatment. Since externalities across schools are substantial, public subsidies should be determined at levels higher than local school committees, such as the district or provincial level.

Note that while we can conclude that there were substantial externalities from the deworming treatment provided through the PSDP, it is difficult to draw firm conclusions about optimal deworming subsidies in the absence of a fully-fledged behavioral and epidemiological model, since the marginal positive externalities from treatment depend on how many others are also being

[62] In future work, we hope to track the children in this study as they enter the labor market in order to estimate how child health gains from deworming affect adult income and other socioeconomic outcomes.

[63] Even under the assumption of a ten percent discount rate, and maintaining the conservative assumption that children are half as productive as adults, the externality benefit–cost ratio is approximately one.

treated. While positive externalities from PSDP were large, it is difficult to gauge how large treatment externalities would be at alternative coverage levels. In theory, depending on epidemiological parameters, some incomplete level of coverage could potentially be sufficient to eliminate the disease from the population, in which case there would be no point in raising subsidies above an amount that would generate this level of coverage. However, Miguel and Kremer (2002) find that use of deworming drugs is very low even at modest positive prices, so it seems likely that the externality benefits of deworming would be sufficient to warrant a zero price. Caution is needed in extrapolating these results to areas with different worm prevalence, since while the direct benefits of deworming may be proportional to worm burden, the externality benefits are likely to vary nonlinearly with worm burden. Clearly, additional research is needed to determine optimal deworming subsidies in this and other settings.

9. CONCLUSION

A school-based deworming program in Kenya led to a 7.5 percentage point average gain in primary school participation in treatment schools, reducing overall school absenteeism by at least one-quarter. Treatment created positive health and school participation externalities for untreated students. A rough calculation suggests that these spillovers alone are sufficient to justify not only fully subsidizing deworming treatment, but perhaps even paying people to receive treatment.

Our results have methodological implications for the literature on the educational effects of deworming, and for the design of randomized evaluations more generally. Existing estimates, from medical studies that randomize treatment within a school, doubly underestimate the effects of deworming programs. First, they entirely miss the external effects of deworming, and second, they underestimate the direct effects to the extent that the comparison group benefits from externalities, biasing existing treatment effect estimates toward zero. This problem can be addressed by randomizing at the level of larger units, such as schools rather than at the individual level. To the extent that spillovers take place within groups, group-level randomization allows identification of overall program impact on the group. Moreover, by the law of large numbers, group-level randomization creates more variation in local treatment densities than individual-level randomization, and this random variation can be used to estimate cross-group externalities. While group-level randomization can be used in other settings with externalities localized, either geographically or along some other dimension, such as the analysis of school vouchers or information transmission and technology diffusion, it cannot be used to estimate more global spillovers, such as those arising through general equilibrium price effects.

When local treatment externalities are expected, field experiments can be purposefully designed to estimate externalities by randomizing treatment at various levels.[64] A prospective research design for identifying externalities both within and across schools in rural Kenya would randomize treatment across pupils within schools, across schools within "clusters" of schools, and then among these clusters. Treatment rates chould be varied across clusters to estimate externalities at various treatment levels. However, this multi-level design may not be practical in all contexts: for example, in our context it was not possible to randomize treatment within schools. Randomization at the level of clusters of schools also dramatically increases the sample size needed for adequate statistical power, raising project cost. The large improvement in school participation following deworming estimated in this study points to the important role that tropical diseases such as intestinal worms may play in reducing educational attainment in sub-Saharan Africa and provides microeconomic support for claims that Africa's high tropical disease burden is a causal factor contributing to its low income.[65] Our results also suggest that microeconomic and macroeconomic studies that estimate the impact of health on income conditional on educational attainment are likely to systematically underestimate its impact, since some of the overall health effect works through the education channel. To the extent that the treatment of other tropical infectious diseases also generates spillover benefits similar to deworming, the externality findings of the current study provide an additional rationale for a substantial public role in subsidizing medical treatment for infectious diseases in less developed countries. Miguel and Kremer (2002) examine the design of programs to promote deworming, why a large minority of children did not take the free deworming drugs, and the role of drug cost, social learning, and other behavioral factors in influencing take-up of deworming drugs.

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[64] See Duflo and Saez (2002).

[65] Of course, worms' impact on wages through education can only explain a small fraction of the enormous income gap between African and industrialized countries.

APPENDIX

APPENDIX TABLE AI

PRIMARY SCHOOL DEWORMING PROJECT (PSDP) TIMELINE, 1997–1999

Dates	Activity
1997	
October	Pilot Kenya Ministry of Health, Division of Vector Borne Disease (DVBD) parasitological survey. Pilot Pupil Questionnaire
1998	
January–March	Parent-teacher meetings in Group 1 schools
	Pupil Questionnaire administration in grades 3 to 8, and School Questionnaire administration in all schools
	DVBD parasitological survey for grades 3 to 8 in Group 1 schools
January–May	Heavy precipitation and widespread flooding associated with the El Niño weather system
March–April	First round of 1998 medical treatment (with albendazole, praziquantel) in Group 1 schools
October–November	ICS (NGO) examinations administered in grades 3 to 8 in all schools
November	Second round of 1998 medical treatment (with albendazole) in Group 1 schools
1999	
January–March	Parent-teacher meetings in Group 1 and Group 2 schools
	Pupil Questionnaire administration in grades 3 to 8, and School Questionnaire administration in all schools
	DVBD parasitological and hemoglobin surveys for grades 3 to 8 in Group 1 and Group 2 schools
March–June	First round of 1999 medical treatment (with albendazole, praziquantel) in Group 1 and Group 2 schools
May–July	Deworming drug availability survey of local shops, clinics, and pharmacies
October	ICS (NGO) examinations administered in grades 3 to 8 in all schools
October–November	Second round of 1999 medical treatment (with albendazole) in Group 1 and Group 2 schools

APPENDIX TABLE AII

LOCAL DENSITIES OF OTHER PRIMARY SCHOOLS AND DEWORMING COMPLIANCE RATES[a]

	Dependent variable:	
	1998 Compliance rate (any medical treatment) OLS (1)	1999 Compliance rate (any medical treatment) OLS (2)
Treatment school pupils within 3 km	−0.04	−0.08
(per 1000 pupils)	(0.06)	(0.09)
Treatment school pupils within 3–6 km	0.04	−0.01
(per 1000 pupils)	(0.07)	(0.05)
Total pupils within 3 km	0.05	0.05
(per 1000 pupils)	(0.05)	(0.08)
Total pupils within 3–6 km	−0.06	−0.02
(per 1000 pupils)	(0.06)	(0.05)
Grade indicators, school assistance controls, district exam score control	Yes	Yes
R^2	0.60	0.57
Root MSE	0.082	0.131
Number of observations	25	49
Mean of dependent variable	0.66	0.42

[a] Robust standard errors in parentheses. Observations are weighted by total school population. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence. The 1998 compliance data is for Group 1 schools, and the 1999 compliance data is for Group 1 and Group 2 schools. The pupil population data is from the 1998 School Questionnaire. We use the number of girls less than 13 years old and all boys (the pupils eligible for deworming in the treatment schools) as the school population for all schools. The number of treatment school pupils in 1998 is the number of Group 1 pupils, and the number of treatment school pupils in March 1999 is the number of Group 1 and Group 2 pupils.

APPENDIX TABLE AIII
DEWORMING HEALTH EXTERNALITIES—ROBUSTNESS CHECKS[a]

	Any moderate-heavy helminth infection, 1999				Moderate-heavy schistomiasis infection, 1999			
	Probit	OLS, spatial s.e.	Probit	Probit (Group 1 only)	Probit	OLS, spatial s.e.	Probit	Probit (Group 1 only)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Indicator for Group 1 (1998 Treatment) School	−0.25***	−0.24***	−0.28***		−0.03	−0.08*	−0.04	
	(0.05)	(0.05)	(0.05)		(0.03)	(0.04)	(0.04)	
Group 1 pupils within 3 km	−0.26***	−0.17**		−0.30***	−0.12***	−0.13***		−0.06***
(per 1000 pupils)	(0.09)	(0.07)		(0.07)	(0.04)	(0.04)		(0.02)
Group 1 pupils within 3–6 km	−0.14**	−0.18***		−0.07	−0.18***	−0.20**		−0.05***
(per 1000 pupils)	(0.06)	(0.04)		(0.06)	(0.03)	(0.07)		(0.01)
Total pupils within 3 km	0.11***	0.09	0.07	0.04	0.11***	0.14***	0.10***	0.03***
(per 1000 pupils)	(0.04)	(0.06)	(0.05)	(0.04)	(0.02)	(0.03)	(0.03)	(0.01)
Total pupils within 3–6 km	0.13**	0.16***	0.08	0.03	0.12***	0.13**	0.08**	0.04
(per 1000 pupils)	(0.06)	(0.04)	(0.05)	(0.06)	(0.03)	(0.05)	(0.03)	(0.01)
(Group 1 pupils within 3 km)/			−0.29***				−0.13**	
(Total pupils within 3 km)			(0.11)				(0.07)	
(Group 1 pupils within 3–6 km)/			−0.12				−0.41***	
(Total pupils within 3–6 km)			(0.22)				(0.11)	
Any moderate-heavy helminth infection, 1998				0.25***				
				(0.03)				
Moderate-heavy schistosomiasis infection, 1998								0.22***
								(0.10)
Grade indicators, school assistance controls, district exam score control	Yes	No	Yes	Yes	Yes	No	Yes	Yes
R^2	–	0.57	–	–	–	0.48	–	–
Root MSE	–	0.177	–	–	–	0.168	–	–
Number of observations	2326 (pupils)	49 (schools)	2326 (pupils)	602 (pupils)	2326 (pupils)	49 (schools)	2326 (pupils)	603 (pupils)
Mean of dependent variable	0.41	0.41	0.41	0.25	0.16	0.16	0.16	0.08

[a] Grade 3–8 pupils. Robust standard errors in parentheses. Disturbance terms are clustered within schools for regressions 1, 3, 4, 5 and 7. Disturbance terms are allowed to be correlated across spaces using the method in Conley (1999) in regressions 2 and 6. Observations are weighted by total school population. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence. The 1999 parasitological survey data are for Group 1 and Group 2 schools. The pupil population data is from the 1998 School Questionnaire. We use the number of girls less than 13 years old and all boys (the pupils eligible for deworming in the treatment schools) as the school population for all schools.

APPENDIX TABLE AIV

IV ESTIMATES OF HEALTH AND SCHOOL PARTICIPATION EXTERNALITIES[a]

	Any moderate-heavy helminth infection, January–March 99		Average individual school participation, May 98–March 99	
	Probit (1)	IV-2SLS (2)	OLS (3)	IV-2SLS (4)
Indicator for Group 1 (1998 Treatment) School	−0.12*	−0.04	0.056***	0.024
	(0.07)	(0.10)	(0.020)	(0.028)
Group 1 pupils within 3 km (per 1000 pupils)	−0.26***	−0.22***	0.023	0.020
	(0.09)	(0.07)	(0.036)	(0.035)
Group 1 pupils within 3–6 km (per 1000 pupils)	−0.13**	−0.11**	−0.041	−0.041
	(0.06)	(0.05)	(0.027)	(0.026)
Total pupils within 3 km (per 1000 pupils)	0.11***	0.11***	−0.035*	−0.034*
	(0.04)	(0.04)	(0.019)	(0.019)
Total pupils within 3–6 km (per 1000 pupils)	0.13**	0.11**	0.022	0.021
	(0.06)	(0.05)	(0.027)	(0.027)
Indicator received first year of deworming treatment, when offered (1998 for Group 1, 1999 for Group 2)	−0.06*	−0.06	0.100***	0.013
	(0.03)	(0.05)	(0.014)	(0.030)
(First year as treatment school Indicator) ∗ (Received treatment, when offered)	−0.14*	−0.21*	−0.012	0.059
	(0.07)	(0.12)	(0.020)	(0.046)
Grade indicators, school assistance controls, district exam score control	Yes	Yes	Yes	Yes
Time controls	No	No	Yes	Yes
R^2	–	–	0.36	–
Root MSE	–	0.446	0.219	0.221
Number of observations	2326	2326	18264	18264
Mean of dependent variable	0.41	0.41	0.784	0.784

[a]Disturbance terms are clustered within schools. Robust standard errors in parentheses. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence. The two instrumental variables are an indicator for girls under age 13 and all boys (ELG), and (ELG) ∗ (Group 1 indicator). The coefficient on the Group 1 school indicator variable serves as an estimate of the within-school externality effect in 1998. This IV approach could overestimate the treatment effect if the treatment effect is heterogeneous, with sicker pupils benefiting most from treatment, and if among the girls over 13, the sickest girls are most likely to be treated in treatment schools. However, among the subsample of older girls, the compliance rate was not significantly related to infection status in 1998 (Table VI), and in 1999 under ten percent of older girls were treated (Table III). We find similar effects even when we exclude the schools near the lake where older girls were likely to be treated (results not shown). Note that the IV estimates of within-school participation externalities should be interpreted as local average treatment effects for the older girls. Since school participation treatment effects are largest for younger pupils, it is not surprising that the IV externality estimates among the older girls are smaller than the OLS estimates, which are for the entire population. We use the number of girls less than 13 years old and all boys (the pupils eligible for deworming in the treatment schools) as the school population for all schools.

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THE ILLUSION OF SUSTAINABILITY*

MICHAEL KREMER AND EDWARD MIGUEL

We use a randomized evaluation of a Kenyan deworming program to estimate peer effects in technology adoption and to shed light on foreign aid donors' movement towards sustainable community provision of public goods. Deworming is a public good since much of its social benefit comes through reduced disease transmission. People were less likely to take deworming if their direct first-order or indirect second-order social contacts were exposed to deworming. Efforts to replace subsidies with sustainable worm control measures were ineffective: a drug cost-recovery program reduced take-up 80 percent; health education did not affect behavior, and a mobilization intervention failed. At least in this context, it appears unrealistic for a one-time intervention to generate sustainable voluntary local public goods provision.

I. INTRODUCTION

The history of overseas development assistance can be viewed as a series of attempts to identify and address ever more fundamental causes of global poverty. Oxfam, for example, founded in 1942 as the Oxford Committee for Famine Relief, later shifted to "support for self-help schemes whereby communities improved their own water supplies, farming practices, and health provision".[1] In the 1950s and 1960s, it was widely argued that long-run economic performance depended on capital investment and that raising savings through a "big push" [Rosenstein–Rodin 1943] would launch countries into self-sustaining growth or "take-off" [Rostow 1960]. Accordingly, the World Bank largely funded infrastructure like dams and roads. By the 1980s international financial institution policymakers decided that capital accumulation and technological progress depended not so much on investment and careful engineering but rather on a better

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1. Refer to the Oxfam website for the details (http://www.oxfam.org.uk/about_us/history/history2.htm).

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economic policy environment [Williamson 1990; World Bank 1993a]. Development assistance was extended conditionally to encourage countries to adopt economic policies associated with this "Washington Consensus" view, characterized by reduced tariffs, appropriate foreign exchange rates and low inflation. By the 1990s, this approach also became seen as inadequate by many. According to a new consensus, these policies would have only limited impact in the absence of more fundamental institutional reforms [World Bank 1998].

Part of this new consensus in overseas development assistance involved reforms to national level institutions, but given widespread central government failures in delivering public goods, another strand emphasized encouraging local communities to sustainably provide their own public goods. Whereas orthodox public finance analysis suggests that governments or donors should indefinitely fund activities that generate positive externalities, advocates of sustainability emphasize the importance of local project "ownership" and promote public goods projects that only require start-up funding and can then continue without external support. These efforts typically rely on voluntary activities by community members rather than on the granting of coercive fundraising powers to local governments.

The idea that development projects should aim at financial sustainability through voluntary local action has had tremendous influence in development thinking, in areas from microfinance to the environment.[2] In public health and water supply, sustainability advocates concentrate on cost recovery from beneficiaries, community mobilization, and health education rather than simply building wells or subsidizing medical treatments that generate externalities. The idea of replacing dependency on aid with a one-time investment that leads to long-run sustainability is certainly ideologically attractive.

Yet anecdotal evidence suggests that financial sustainability has often been an illusion, and sometimes a costly one. Morduch [1999] argues that the pursuit of sustainability by microfinance organizations has led them to move away from serving the poor. Meuwissen [2002] argues that a health cost-recovery program in Niger led to unexpectedly large drops in health care utilization and that the local health committees set up by the program failed

2. Sustainability has other meanings, including an environmental meaning, but we focus on financial sustainability.

in most of their responsibilities. In a large water project in the Kenyan area we study, 43 percent of borehole wells were useless ten years after the shift from external donor support for water-well maintenance to the training of local maintenance committees [Miguel and Gugerty 2005].

While it is certainly true that in some cases communities have developed institutions that lead individuals to contribute to local public goods [Ostrom 1990], it is less clear that external interventions, such as training sessions or the formation of user committees by donors, reliably lead to sustainable voluntary provision of local public goods. It is difficult for outsiders to understand how other societies' institutions and politics function, let alone how to influence them in a way that creates the correct incentives and does not generate unforeseen negative consequences.

In this paper we seek to shed light on these issues using evidence from a randomized evaluation of a deworming program in Kenya. Intestinal worms infect one in four people worldwide. They can be fought in several different ways. One approach emphasizes periodic medical treatment with low-cost drugs. Public provision of deworming medicine can likely be justified on standard public finance grounds since an estimated three quarters of the social benefit of treatment comes through reducing disease transmission [Miguel and Kremer 2004]. However, some argue that too much emphasis has been placed on just handing out deworming drugs. Since people soon become reinfected, deworming drug treatment must be continued twice per year indefinitely. In a *Lancet* article entitled "Sustainable Schistosomiasis Control—The Way Forward," Utzinger et al. [2003] argue that, rather than focusing narrowly on drugs, a broader approach with greater emphasis on health education would be more sustainable. Other potential ways to make anti-worm programs sustainable include requiring cost-sharing payments from those taking the drugs, promoting the diffusion of worm prevention information and behaviors through social networks, and encouraging local ownership of deworming programs.

In this study we find that, first, the introduction of a small fee for deworming drugs ("cost-sharing") led to an 80 percent reduction in treatment rates, consistent with the hypothesis that people have low private valuation for deworming. Take-up dropped sharply when going from a zero price to a positive price but was not sensitive to the exact (positive) price level, suggesting that it may be particularly counter-productive to charge small positive prices for the treatment of infectious diseases. Second, an intensive school health education intervention had no impact on worm prevention behaviors. Third, a verbal commitment "mobilization" intervention—in which people were asked in advance whether they planned to take deworming drugs, exploiting a finding from social psychology that individuals strive for consistency in their statements and actions—had no impact on adoption.

We also examine peer effects in adoption since, if imitation effects in technology adoption are sufficiently strong, a sufficiently large temporary investment to introduce deworming drugs could move society from a low-adoption to a high-adoption equilibrium. A number of recent papers, including Foster and Rosenzweig [1995], Conley and Udry [2000], Burke, Fournier, and Prasad [2003], and Munshi [2004] find evidence for peer effects in technology adoption using nonexperimental data. Like Duflo and Saez [2003], we exploit experimental variation in exposure to a new technology to address the well-known econometric challenges in estimating peer effects [Manski, 1993]. We develop a theoretical framework that allows for peer effects from pure imitation, social learning about how to use technologies optimally, social learning about the benefits of new technologies, and epidemiological externalities. This model suggests that as long as a small fraction of the population receives subsidies sufficient to induce their adoption, further subsidies will affect steady-state take-up only in the presence of imitation effects. We collect data on the network structure of links between school communities and use this to empirically estimate the impact on adoption decisions not only of individuals' direct social links, but also of higher-order social links. Rather than imposing a preexisting definition of social links, based, for example, on geography [Foster and Rosenzweig 1995; Burke, Fournier, and Prasad 2003], we allow survey respondents to specify their social links themselves and estimate the impact of learning through different types of links. We then simulate the impact of alternative ways of seeding the new technology given the observed network structure of links across schools in our sample.

We find that additional social links to early treatment schools reduce the probability that children take deworming drugs and increase the probability that parents say that deworming drugs are "not effective." This negative take-up result holds both for direct social links and for indirect second order connections. We find evidence that Granovetter's [1973] "weak ties" are important, with individuals learning both from "close" and "distant" contacts, as measured by the frequency with which they communicate. There is also some evidence for learning through child networks in addition to the parent networks that form the core of the analysis. In contrast, analysis of our data using nonexperimental methods would imply that individuals are more likely to take the drugs if they have greater social contact with others who have recently been exposed to deworming, suggesting substantial omitted variable bias in the nonexperimental estimates.

The lower take-up among those with more knowledge may be due to the high proportion of deworming benefits flowing not to the treated child or her family, but to others in the local community through externalities. People may only have realized how much of the benefits were external as they gained experience with the program. Negative social effects on take-up are especially large empirically for families with more schooling, a group who start out with particularly favorable beliefs about the technology but then rapidly revise their beliefs downwards as they acquire more information.

Our results are consistent with peer effects due to learning from others about the benefits of the technology and suggest that, at least in this context, peer effects due to imitation or due to learning about how to use the technology are small. In this context, a policymaker uncertain about the benefits of a new technology might want to subsidize a small number of people to adopt in hopes of spurring a shift to a new equilibrium, but temporary subsidies beyond this level would not affect steady-state adoption.

Overall, the empirical results on cost-sharing, health education, and social learning are all consistent with the hypothesis that people put limited private value on deworming. Miguel and Kremer [2004], however, suggest the social value is large. Together these results suggest that large ongoing external subsidies may be necessary to sustain high take-up. These results may generalize to other infectious and parasitic diseases characterized by large positive treatment externalities. More generally, it is probably an illusion to think that a one-time infusion of external assistance will lead to the indefinitely sustainable voluntary provision of most local public goods. There may simply be no alternative to ongoing subsidies financed by tax revenue raised either from local or national governments, or international donors.[3]

The remainder of the paper is structured as follows. Section II provides information on worm infections and the project we study. In Section III we present a simple theoretical framework for understanding the determinants of deworming take-up. Section IV describes the empirical take-up impacts of direct and higher-order social links. Sections V, VI, and VII describe the cost-sharing, health education, and verbal commitment results, respectively. The final section discusses broader implications for public finance and development assistance in less developed countries. Readers interested primarily in social learning may wish to focus on Sections III and IV while those interested in development policy issues could focus mainly on Sections V through VIII.

II. The Primary School Deworming Project

Over 1.3 billion people worldwide are infected with hookworm, 1.3 billion with roundworm, 900 million with whipworm, and 200 million with schistosomiasis [Bundy 1994]. Most have light infections, which are often asymptomatic, but more severe worm infections can lead to iron-deficiency anemia, protein energy malnutrition, stunting, wasting, listlessness, and abdominal pain. Heavy schistosomiasis infections can have even more severe consequences.[4]

Helminths do not reproduce within the human host, so high worm burdens are the result of frequent reinfection. The geohelminths (hookworm, roundworm, and whipworm) are transmitted through ingestion of, or contact with, infected fecal matter. This can occur, for example, if children defecate in the fields near their home or school, areas where they also play. Schistosomiasis is acquired through contact with infected freshwater. For example, in our Kenyan study area people often walk to nearby Lake Victoria to bathe and fish. Medical treatment for helminth infections creates externality benefits by reducing worm deposition in the community and thus limiting reinfection among other community members [Anderson and May 1991]. The geohelminths

3. Lengeler [1999] reaches similar conclusions.
4. Refer to Adams et al. [1994], Corbett et al. [1992], Hotez and Pritchard [1995], and Pollitt [1990].

and schistosomiasis can be treated using the low-cost single-dose oral therapies of albendazole and praziquantel, respectively. The drugs sometimes cause unpleasant and salient, but medically minor, side effects including stomachache, diarrhea, fever, and, occasionally, vomiting [WHO 1992], but these effects rarely last more than one day. Side effects are more severe for heavier schistosomiasis infections but can be mitigated by not consuming the drugs on an empty stomach. Private benefits of deworming may not always be particularly salient to individuals since they typically occur gradually as individual nutritional status improves in the months following treatment.

Miguel and Kremer [2004] found that deworming treatment can generate large externality benefits by interfering with disease transmission. Providing treatment to Kenyan school children led to large reductions in worm infections and increased school participation among both treated and untreated children in the treatment schools and among children in neighboring schools. Three quarters of the social benefit of treatment was in the form of externalities. Since deworming costs only $3.50 per extra year of school participation generated, it is likely one of the most cost-effective ways to boost participation.

Both this paper and Miguel and Kremer [2004] study the Primary School Deworming Project (PSDP), a school health program carried out by a Dutch nongovernmental organization (NGO), ICS Africa, in cooperation with the Kenyan Ministry of Health. The project took place in Busia district, a poor and densely-settled farming region in western Kenya, and the seventy-five project schools include nearly all rural primary schools in the area with over 30,000 enrolled pupils between the ages of six and eighteen, over 90 percent of whom suffer from intestinal worm infections. In January 1998, the schools were randomly divided into three groups (Group 1, Group 2, and Group 3) of twenty-five schools each: the schools were first divided by administrative subunit (zone) and by involvement in other nongovernmental assistance programs and were then listed alphabetically. Every third school was assigned to a given project group.

The intervention included both health education on worm prevention behaviors and the provision of deworming medicine. Due to administrative and financial constraints, the program was phased in over several years. Group 1 schools received assistance in 1998, 1999, 2000, and 2001, and Group 2 schools in 1999, 2000, and 2001 while Group 3 began receiving assistance in 2001. This design implies that in 1998 Group 1 schools were treatment schools while Group 2 and Group 3 schools were the comparison schools; and in 1999 and 2000, Group 1 and Group 2 schools were the treatment schools and Group 3 schools were comparison schools. At each school, the project started out with a community meeting of parents and teachers organized by the NGO, which included a discussion of worm infections, the nature of medical deworming treatment, and worm prevention measures. All primary school communities in the baseline sample agreed to participate in the project. Starting in 1999, the Ministry of Health required signed individual parental consent whereas in 1998 only community consent had been required, with individuals having the ability to opt out of the program if they wished. This change in 1999 may have reduced take-up in some cases if parents were reluctant to visit the school headmaster, particularly if they were late on other school fee payments.

Health education efforts focused on preventing worms through hand washing, wearing shoes, and avoiding infected fresh water. This included classroom lectures and culturally appropriate Swahili language health education materials. This health education effort was considerably more intensive than is typical in Kenyan primary schools, and, thus, the program may have been more likely than existing government programs to impact child behavior. Two teachers in each school attended a full-day training session on worm prevention lessons as well as on the details of the deworming program and were instructed to impart these lessons during school hours. These classroom lessons were supplemented through lectures by an experienced NGO field team (the team leader was a trained public health technician), which visited each treatment school several times per year.

At all schools where helminth prevalence was sufficiently high, the project provided periodic treatment with deworming drugs to be taken at the school. The World Health Organization has endorsed mass school-based deworming in areas with prevalence over 50 percent since mass treatment eliminates the need for costly individual screening [Warren et al. 1993; WHO 1987], and the drugs are cheap when purchased in bulk.[5]

5. The project followed the standard practice at the time in mass deworming programs of not treating girls of reproductive age—typically aged thirteen years

Our best estimate is that teacher training, teacher lessons at school, the lectures delivered by the NGO field team, and the classroom wall charts and other educational materials, taken together, cost at least US$0.44 per pupil per year in the assisted schools[6]—which is comparable to the total cost of deworming drug purchase and delivery in a nearby Tanzanian program, at US$0.49 [PCD 1999]. In our case, it is difficult to break out the costs of health education, data collection, and drug delivery since the same field team was responsible for all activities, so cost estimates should be seen as approximate.

The NGO we worked with has a policy of using community cost-recovery in its projects to promote sustainability and confer project ownership on beneficiaries. In the case of deworming, the NGO temporarily waived this policy initially and then planned to phase it in gradually. The fifty Group 1 and Group 2 schools were stratified by treatment group and geographic location and then twenty-five were randomly selected (using a computer random number generator) to pay user fees for medical treatment in 2001, while the other twenty-five continued to receive free medical treatment that year; all Group 3 schools received free treatment in 2001. The deworming fee was set on a per-family basis like most Kenyan primary school fees at the time. This introduced within-school variation in the per-child cost of deworming since households have different numbers of primary school children, variation that we also use to estimate the effect of price on drug take-up. Of the twenty-five Group 1 and Group 2 schools participating in cost-sharing, two-thirds received albendazole at a cost of 30 Kenyan shillings per family (US$0.40 in 2001), and one-third received both albendazole and praziquantel at a cost of 100 shillings (approximately US$1.30). Whether praziquantel was given depended on the local prevalence of schistosomiasis. Since parents have 2.7 children in school on average, the average cost of deworming per child in cost-sharing schools was slightly more than US$0.30—still a heavily subsidized price, about one-fifth

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and older in practice—due to concern about the possibility that albendazole could cause birth defects [WHO 1992; Cowden and Hotez 2000]. The WHO recently called for this policy to be changed based on an accumulating record of safe usage by pregnant women (see Savioli, Crompton, and Niera [2003]).

6. This figure is based on an estimate that each health education teacher taught two full hours on worm prevention behaviors in each grade per school year (given an annual teacher salary and benefits of approximately US$2,000) and that the NGO team also provided two hours of health education per school per year.

the cost of drug purchase and delivery through this program (at US$1.49) and 60 percent of the cost in the Tanzania program.[7]

The study area seems fertile ground for encouraging voluntary community provision of local public goods like deworming control. Kenya has a long history of community self-help programs, and indeed the national motto of "*Harambee*" refers to such programs. The project we examine was conducted at primary schools, one of the most widespread and firmly established institutions in rural Kenya. All primary schools have a committee composed of parents and community representatives, and historically these committees have been entrusted with raising funds locally for most non-salary costs of running the school, including everything from chalk to classroom construction.

Cultural understandings of health, and particularly worms, in our study area also merit a brief discussion; this account draws heavily on the work of Geissler [1998a, 1998b, 2000], who studies deworming take-up in the Kenyan district that borders our study area. Medical anthropologists have long pointed out that people can simultaneously hold traditional and biomedical views of health in a manner similar to religious syncretism, and Geissler argues that this is the case for views about worms in western Kenya. In the traditional view, worms are an integral part of the human body and necessary for digestion, and many infection symptoms are attributed to malevolent occult forces ("witchcraft") or breaking taboos [Government of Kenya 1986]. Educated people are more likely to engage in the biomedical discourse and thus more likely to treat illnesses medically rather than using traditional remedies. Geissler finds that most people do not place much value on deworming treatment because worms are not seen as a pressing health problem, especially compared to malaria and HIV/AIDS.[8] As a result, there was almost no deworming outside the school health program he studies, and most children relied on local herbal remedies to alleviate the abdominal discomfort caused by worms.

Local knowledge regarding private benefits of receiving treatment under a mass deworming program was likely very poor

7. Kenyan per capita income was US$340 [World Bank 1999], and incomes may be even lower in Busia.

8. Geissler studies an ethnically Luo population (Luos speak a Nilotic language). The majority of our sample is ethnically Luhya (a Bantu-speaking group), though Luos are 4 percent of our sample. However, traditional Luo views are closely related to views found among other African groups [Green, Jurg, Djedje 1994; Green 1997].

in our study area. The project we study was the first mass deworming treatment program in the district, to our knowledge. Albendazole and praziquantel were only approved for human use in the mid-1980s and by 1998 were still rarely used in the area. Prior to the program, fewer than 5 percent of people reported taking deworming drugs [Miguel and Kremer 2004]. While many medicines, such as aspirin and anti-malarials, are cheaply available in nearly all local shops, deworming was only available in a few shops and at high mark-ups, presumably due to a thin market. In fact, none of sixty-four local shops surveyed in 1999 had either albendazole (or its close substitute, mebendazole) or praziquantel in stock, though a minority carried less effective deworming drugs (levamisole hydrochloride and piperazine). Albendazole and praziquantel were available in some local health clinics. Inference about likely mass treatment impacts based on observed individual impacts was complicated for local residents by nonrandom selection into treatment, as well as the possibility of spillover effects.

III. A Framework for Understanding the Adoption of a New Health Technology

We model the spread of information and the evolution of take-up of a new technology in a social network. The model provides a framework for the empirical estimation of adoption peer effects and helps clarify the conditions under which a one-time subsidy can change the long-run level of adoption and thus achieve "sustainability."

We develop a simple framework in which people adopt deworming if expected private benefits exceed the expected cost. They are heterogeneous both in their taste for deworming and in their priors about the effectiveness of the drugs. People are linked in a social network and receive signals about adoption, drug effectiveness, and how to use the drugs. The model nests four types of peer effects proposed in the existing literature. Others' adoption can (i) influence own adoption through the disease environment, (ii) directly enter the utility function through a pure imitation effect, (iii) provide information about how to effectively use the technology (as in Jovanovic and Nyarko [1996] or Foster and Rosenzweig [1995]), or (iv) provide information on the benefits of the technology (as in Banerjee [1992] or Ellison and Fudenberg [1993]).

III.A. Assumptions

We assume that an individual i decides to adopt a new technology (or health practice) if the expected private benefits are greater than the costs, conditional on her prior beliefs and the information received from social contacts. As noted earlier, the cost of deworming adoption is privately incurred, immediate and salient, while much of the benefit is in the form of externalities and even the private benefits are delayed, so private benefits may not exceed costs, particularly for people with high discount rates.

Suppose that the total private benefit to taking the deworming drug depends on the individual's infection level γ, the effectiveness of the drug ϕ (which incorporates the percentage reduction in worm load that results from taking the drug and the rapidity of reinfection)[9], and an idiosyncratic individual specific taste for deworming μ_i that is assumed to have a continuous distribution with no mass points and a sufficiently large support such that some individuals always take up the drug. (Note that policymakers can always guarantee that some take up the drug by heavily subsidizing a small fraction of consumers.) Individual infection γ may depend on individual characteristics X and on others' treatment history. Because worms are transmitted through environmental contamination rather than from person to person, infection levels are likely to depend on average population treatment rather than an individual's social links.

Financial, time, or utility costs of treatment are denoted by $C > 0$. Below we allow for the possibility that people may learn from their own experience and from others about how to reduce the cost of using the technology (for example, how to control side effects by taking food with the medicine), but, as in Jovanovic and Nyarko [1996] and Foster and Rosenzweig [1995], we assume this learning is bounded so that C approaches some positive C_∞. The drug subsidies, health education, and verbal commitment inter-

9. The effect of other people's treatment choices on the magnitude of private treatment benefits is unclear *a priori*. As a benchmark, if helminth reinfection rates are independent of own current worm load and if the health burden of infection is linear in own worm load, the private health benefits of treatment are independent of others' choices. If, instead, the health costs of infection are convex in worm load, deworming benefits will be greater in an environment that is expected to have high exposure to worms in the future. Thus, the net private benefits of treatment will be lower if others are treated. The opposite holds with concavity. Miguel and Kremer [2004] estimate average deworming treatment spillovers and find that they are roughly linear in local treatment rates, but due to data limitations have little power to detect nonlinear higher order terms. Here we assume the benchmark linear case.

ventions discussed in Sections V, VI, and VII can be regarded as changing the adoption cost.

Finally, a desire to imitate one's social contacts may influence the decision to take up the technology. The parameter $\beta > 0$ captures the importance of this effect.

Let $\hat{\phi}_{it}$ denote the individual's beliefs in period t about drug effectiveness ϕ conditional on prior beliefs and any signals received, and let $T_{it} \in \{0, 1\}$ be an indicator variable for drug take-up in period t. Then the individual's expected private benefit from adoption can be expressed as

$$E[U(T_{it} = 1) - U(T_{it} = 0)] = \hat{\phi}_{it} h(\gamma_{it}) \mu_i - C + \beta \omega_{it} \tag{1}$$

where U is individual utility from deworming, conditional on the treatment choices of other individuals, and ω_{it} is the share of social contacts who took up the drug in the previous period.

We assume that individuals decide whether to adopt deworming at time t based on the current costs and benefits of adoption and do not consider the additional motive of adopting in order to learn more about the impact of the technology or how to use it in the future. This is partly to keep the model tractable but is also a reasonable assumption in our context. Discount rates were likely high given the temporary nature of the program and the limited foresight of schoolchildren. Moreover, deworming was introduced at the level of whole schools, so most people offered the chance to take it would have many opportunities to learn about impacts from classmates, limiting the marginal value of their own experience.

III.B. Information Structure

At the moment the new technology is introduced, individual i has a prior belief about the effectiveness of taking deworming medicine as part of a mass campaign, denoted ϕ_{i0}, which may be greater or less than the actual effectiveness ϕ. Priors could be less than ϕ due to traditional beliefs about worms in the study region [Geissler 1998a, 1998b]. However, people could also have had overly optimistic estimates about private benefits. The enthusiasm of NGO field officers promoting deworming at schools may have reflected the drugs' social rather than private benefits. Although the scripts made clear that the medicine kills worms in the body but does not prevent reinfection, people may not have realized how quickly they would be reinfected. Moreover, if people estimated their expected private benefits by comparing individuals in treatment versus comparison schools, they would incorrectly assign some of the school-wide treatment externality to private benefits, again making prior beliefs about private deworming benefits overly optimistic.

Priors about deworming effectiveness could also vary systematically with individual characteristics, such as education. This is a departure from the standard assumption of common priors but is plausible for Kenya. In the context of rural Kenya, formal schooling is considered an important predictor of favorable views about new health technologies [Akwara 1996; Kohler, Behrman, and Watkins 2001]. This could reflect either the causal impact of education or simply the fact that people who are more open to "modern" or "Western" ideas and technologies obtain more education. We formalize this variation in prior beliefs by modeling the common effectiveness parameter ϕ as a draw from a distribution believed to have mean $\phi_0(X_i)$ and variance σ_0^2. While people can learn about the realization of ϕ through signals from their social links, beliefs about its distribution need not have converged to a common prior before the program intervention since mass deworming had not taken place in the area before.

All individuals who take the drug obtain a signal about effectiveness. These signals are noisy due to individual time-specific shocks to health status (e.g., malaria, typhoid, cholera) that are hard to distinguish from drug effects. Let these signals have mean ϕ and variance σ_ε^2.

We assume information diffuses through an infinite social network with a simple structure in which the network, viewed from the perspective of any node, is a proper tree. This implies that a single path connects any two nodes.[10] Each individual has m direct social links, people with whom they may exchange information, where m is a positive integer. Each of those links, in turn, also has m direct links. In the special case where $m = 2$, this is equivalent to people being arrayed along an infinite line, each with direct links to two immediate neighbors.

Time is discrete. At the beginning of each period, individuals can send messages to their direct links with information both from their own signals received and from others' signals. Signals

10. As observed by Watts and Strogatz [1998], the addition of even a few links to a sparsely connected network greatly reduces the average path length between any two nodes, so, in general, information will propagate more quickly in more densely connected networks than in the simple tree we consider.

are transmitted to each link with probability p each period. Later in the same period, people receive these messages from their social contacts. These lags in information diffusion are consistent with the data from Kenya, as discussed below.

III.C. Steady-State Adoption

We first solve the steady state of this model, before turning to the transition path.

Note that in our model as long as some fraction of people always adopt, information will eventually diffuse completely. This implies that in steady state $\hat{\phi}_{it} = \phi$ and $C = C_\infty$ for all individuals i. Consider first the case in which $\beta = 0$ (no pure imitation effects). Let λ denote the share of the population taking up treatment, and let λ^* denote the steady-state share such that if a proportion λ^* of the population took the drug in the past, the same proportion will find it optimal to take the drug. An individual will adopt in the steady state if:

$$\phi h(\gamma(\lambda^*, X_i))\mu_i - C_\infty > 0 \tag{2}$$

and forgo treatment if not. It is straightforward to show that there exists a unique equilibrium cutoff value $\lambda^* = \int\int 1\{\phi h(\gamma(\lambda^*, X))\mu - C_\infty > 0\} \cdot P(X,\mu)dXd\mu$, where $P(X,\mu)$ denotes the probability of those values occurring in the population.[11]

While λ^* is unique if $\beta = 0$, there can be multiple steady states under sufficiently strong pure imitation effects, in which others' take-up decisions directly enter the utility function in a manner complementary with own take-up. Even if parameters are such that λ^* is arbitrarily close to zero in the absence of imitation effects, if imitation effects are sufficiently strong so that $\beta > C_\infty - \phi \min_i\{h(\gamma(1,X_i))\mu_i\}$, there will be another steady state in which everyone uses the technology since then: $\phi h(\gamma(1, X_i))\mu_i - C_\infty + \beta > 0$ for all i. A sufficiently large temporary subsidy can in this case lead to a switch from the partial use equilibrium to the full-use equilibrium, leading to sustainable increases in take-up.

Peer effects in technology adoption are sometimes cited as a rationale for why temporary subsidies may have long-run effects. The model suggests that subsidizing a small number of people

11. Note that infection status will, in general, be a function of the entire treatment history of the network. In the steady state, however, the equilibrium take-up rate λ^* is a sufficient statistic for the entire history since the take-up rate is the same in every period.

will be sufficient to ensure that those people will learn both the returns to the technology and how to best use the technology. In the absence of pure imitation effects, this will be enough to assure widespread long-run adoption of technologies with positive private returns.[12] There is no need to subsidize a large number of people to achieve steady-state diffusion. While this result is specific to this particular model, we conjecture that similar results will apply under other Bayesian learning models. If policymakers are uncertain about the benefits of a particular technology, then providing heavy subsidies to a few people seems much more prudent than widely subsidizing what may turn out to be an unattractive technology.[13]

III.D. Take-Up Along the Transition Path

We next turn to modeling take-up along the transition path. By time τ, the probability that a signal is transmitted from a first-order link to the receiver is $[1-(1-p)^{\tau}]$, the probability that signal is transmitted from a second-order link to the receiver is $[\Sigma_{k=2...\tau}(k-1)\cdot\{p^2(1-p)^{k-2}\}]$, and more generally the probability that a signal is transmitted from a jth order link is $\Sigma_{k=j}^{\tau}\binom{k-1}{j-1}\cdot\{p^j(1-p)^{k-j}\}$ for $j\leq\tau$, and 0 for $j>\tau$.

Holding fixed the take-up behavior of intermediate nodes, the direct impact of an additional signal acquired by a jth order link on take-up is then the probability that the signal is transmitted, multiplied by an indicator for whether the receiver changes her take-up decision in response to the new signal. Let i index an individual node as above. Take-up occurs ($T_{it}=1$) if and only if $E[U(T_{it}=1)-U(T_{it}=0)]>0$, and the direct impact of an additional signal from a jth order link by time τ is thus:

12. We conjecture that even in the presence of peer effects, if social connections are in a tree network structure as modeled here, then subsidizing a small group of tightly socially linked people may be sufficient to ensure adoption and further diffusion of the technology unless private returns are low enough and peer effects strong enough that people will not adopt unless a majority of contacts adopt. This is because subsidizing a small group of interconnected people will be sufficient to ensure adoption within this group, and once learning takes place within the group, adoption can then spread outwards to others.

13. Of course, additional subsidies may be justified if there is learning by doing in production. Here we examine the extent to which social learning by consumers generates a case for subsidies.

$$(3)\quad \left[\sum_{k=j}^{\tau}\begin{pmatrix} k-1 \\ j-1 \end{pmatrix}\cdot\{p^{j}(1-p)^{k-j}\}\right]\cdot[1\{\hat{\phi}_{it}h(\gamma_{it})\mu_i - C + \beta\omega_{it} > 0|\text{Signal}\} - 1\{\hat{\phi}_{it}h(\gamma_{it})\mu_i - C + \beta\omega_{it} > 0|\text{No signal}\}].$$

An additional signal can impact take-up behavior so that $[(T_{it}|\text{Signal}) - (T_{it}|\text{No signal})]$ is nonzero, by changing beliefs about ϕ (or, similarly, by reducing the cost of take-up C, as discussed below). If a Bayesian individual has N_{it}^{E} total signals from early treatment school links, both direct (first-order) and indirect (higher-order), she then weights her prior beliefs and signals received from social links such that the posterior belief on expected effectiveness becomes:

$$(4)\qquad \hat{\phi}_{it} = \left[\left\{\frac{\sigma_N^2}{\sigma_N^2+\sigma_0^2}\right\}\cdot\phi_0(X_i) + \left(1-\left\{\frac{\sigma_N^2}{\sigma_N^2+\sigma_0^2}\right\}\right)\cdot\phi_S\right]$$

where $\phi_0(X_i)$ is the mean of her prior distribution, ϕ_S is the sample average of signals received through the social network, and $\sigma_N^2 \equiv \sigma_\varepsilon^2/E_{it}^{E}$ denotes the variance of the sample average. As individuals accumulate more signals through their social network, the variance of the sample average goes to zero, and the value of both the sample average and posterior beliefs approach the true expected effectiveness, ϕ.

When the prior belief is greater than the true expected effectiveness ($\phi_0(X_i) > \phi$), individuals with more early treatment social links tend to have falling posterior beliefs about expected effectiveness, and thus the likelihood of adoption declines in the number of early treatment links. From (4), the decline in the expected benefit of treatment with respect to early links will be convex, as the posterior asymptotically approaches the true expected effectiveness. Similarly, when the prior is less than the true expected effectiveness, the posterior asymptotically approaches the true benefit from below. When $\phi_0(X_i) > \phi$ for all education levels (X_i) and the prior is increasing in X_i, then individuals with more education generally have higher adoption, but additional early links will lead to sharper drops in their adoption.

Similarly, the framework allows for the possibility that people may learn from signals they receive as well as from their own experience about how to use the technology so $C(\bullet)$ is a decreasing function of the total number of signals ever received about the technology, N_{it}^{E}, with $C'(\bullet) < 0$, $C''(\bullet) > 0$, $C(0) > 0$, and $C(\infty) = C_{\infty}$.

Although epidemiological effects are likely to depend on the broader population rather than immediate social contacts, because worm infections result from contamination of water or soil rather than direct person-to-person transmission, it is worth considering the possibility that children whose families have close social interactions with households in early treatment schools may experience somewhat lower helminth infection rates and, thus, reductions in infection intensity. We model this by allowing the infection level to be a function of the share of direct social contacts treated.

The impact of early treatment links on the expected private benefits to adoption is thus

$$(5)\quad \frac{\partial E[U(T_{it}=1)-U(T_{it}=0)]}{\partial N_{it}^{E}} = \left[\frac{-\sigma_N^2\sigma_0^2}{(\sigma_N^2+\sigma_0^2)^2 N_{it}^{E}}\right]\cdot(\phi_0(X_i)-\phi_S)$$

$$\cdot h(\gamma(\omega_{it},X_i))\cdot\mu_i - \frac{\partial C(N_{it}^{E})}{\partial N_{it}^{E}} + \hat{\phi}_{it}\frac{\partial h}{\partial\gamma}\cdot\frac{\partial\gamma(\omega_{it},X_i)}{\partial\omega_{it}}\cdot\frac{\partial\omega_{it}}{\partial N_{it}^{E}}\mu_i + \beta\frac{\partial\omega_{it}}{\partial N_{it}^{E}}.$$

The first right-hand side term is the social effect from *information on drug effectiveness* and can be positive or negative depending on the difference between priors and true private adoption benefits. The second term captures the social effect from *learning how to use the drugs* described earlier and is always positive. The third term is the *infection social effect*, which should be negative because having more early treatment links could lead to a lower individual infection level (due to epidemiological externalities), which, in turn, reduces treatment benefits. The positive *imitation effect* is captured in the fourth term.

We conclude that, to the extent that we observe negative overall social effects empirically, this is evidence that the combined effect of the information and infection externalities is larger than the learning-by-doing effect plus the pure imitation effect. Furthermore, since infection externalities appear small empirically, as we show below, we interpret negative estimated social effects as strong evidence that social effects work through the transmission of information about drug effectiveness. We find no evidence for learning-by-doing or imitation here although we cannot rule out small effects of these types.

These formulae describe the impact of an additional signal, holding fixed the behavior of intermediate nodes in the social network. In the long run, with repeated opportunities for adoption, there will be additional effects mediated by the effect of a link's information on the take-up behavior of intermediate nodes and, thus, on the subsequent number of signals that intermediate nodes possess and can send to the receiver, as well as any effects on the information and take-up of intermediate nodes mediated by imitation effects. These indirect effects would accumulate over time, but since in our experiment people could only adopt every six months and they were only able to adopt the drugs through the program for zero, two, or three years (depending on their treatment group), we focus above on the case in which the direct effects of signals dominate the indirect effects. In Section IV.F, though, we report results from a simulation of the transition path allowing for these indirect effects.

IV. Empirical Results on Networks, Social Learning, and Technology Adoption

IV.A. Data, Measurement, and Estimation

We test whether households with more social links to schools randomly chosen for early treatment were more likely to take deworming drugs, conditional on their total number of links to all project schools.

The PSDP Parent Questionnaire was collected in 2001 during household visits among a representative subsample of parents with children currently enrolled in Group 2 and Group 3 schools. A representative subsample of children (typically ten to seventeen years old) present in school on the survey day were administered a pupil questionnaire.

Parent questionnaire respondents were asked for information on their closest social links: the five friends they speak with most frequently, the five relatives they speak with most frequently, additional social contacts whose children attend local primary schools, and individuals with whom they speak specifically about child health issues. These individuals are collectively referred to as the respondent's direct "social links." The survey also collected information on the deworming treatment status of social links' children and the effects of treatment on their health, how frequently the respondent speaks with each social link, which primary schools links' children attend, the global positioning system (GPS) location of the respondent's home, and the respondent's knowledge of worm infections and attitudes toward deworming. The parent questionnaire was administered in two rounds in 2001 with households randomly allocated between the rounds. The Round 2 survey collected more detailed information on the impact of deworming on links' children. Two different samples are used in the analysis. Sample 1 contains the 1,678 parents surveyed in either Round 1 or 2 with complete child treatment and parent social network data.[14] Sample 2 contains the 886 parents surveyed in Round 2.

On average, parent respondents have 10.2 direct (first-order) social links with children in primary school, of whom 4.4 attend the respondent's child's own school, 2.8 attend other project schools (Groups 1, 2, or 3), and 1.9 attend nearby "early treatment schools" (Groups 1 and 2—Table I, Panel A). There is considerable variation in the number of direct early treatment links: the standard deviation is 2.0, and approximately one-third of respondents have no social links to Group 1 or 2 schools, one-third have one or two links, and one-third have three or more links.

Approximately forty parents were surveyed in each Group 2 and Group 3 school to construct second-order link measures. For each school we compute the average number of links that parents have to early treatment (Group 1, Group 2) schools and to late treatment (Group 3) schools, once again excluding links to their own school. We do not have information on the social links' own social contacts at the *individual* level and so rely on average school social network contacts in the higher-order analysis. In all main specifications, we exclude all self-referential links, in other words, all direct and higher-order links back to the respondent's own school.

The school average of second-order social connections is likely to be a noisy proxy for the true individual level second-order measures, first, due to idiosyncratic variation in the number of social contacts to particular schools and, second, due to the fact that the social network data are based on surveys with samples of Group 2 and 3 parents alone, rather than with all parents in all local schools. This measurement error should not be systematically correlated with the randomized deworming group assignment of social contacts' schools, preserving the identifica-

14. Survey refusal rates were low, as is typical for this region. Thirteen percent of households were dropped due to either missing network information, treatment information, household characteristics, or difficulty matching across the 2001 surveys and earlier PSDP datasets.

TABLE I
SUMMARY STATISTICS

	Mean	Std dev.	Obs.
Panel A: Parent social links (Round 1 and Round 2 data)			
Total direct (first-order) links	10.2	3.4	1,678
With children in own school	4.4	2.8	1,678
With children not in Group 1, 2, or 3 schools	3.0	2.4	1,678
With children in Group 1, 2, 3 schools—not own school	2.8	2.4	1,678
With children in Group 1, 2 schools—not own school ("early treatment")	1.9	2.0	1,678
With children in Group 1 schools—not own school	0.9	1.4	1,678
Proportion with children in early treatment schools	0.66	0.37	1,358
With children in early treatment schools with whom respondent speaks at least twice per week ("close links")	1.2	1.6	1,678
With children in early treatment schools with whom respondent speaks less than twice per week ("distant links")	0.7	1.1	1,678
Second-order exposure to Group 1, 2, or 3 schools (not own school), through parent links	4.5	4.1	1,678
Second-order exposure to early treatment schools (Groups 1 and 2, not own school) through parent links	2.9	2.9	1,678
Third-order exposure to Group 1, 2, or 3 schools (not own school) through parent links	3.9	5.3	1,678
Third-order exposure to early treatment schools (Groups 1 and 2, not own school) through parent links	2.8	4.1	1,678
Panel B: Parent social links (Round 2 data)			
With children in own school who received deworming	1.5	2.2	886
With children in early treatment schools who received deworming	0.31	0.89	886
With children in early treatment schools who received deworming and had "good effects" (according to respondent)	0.21	0.76	886
With children in early treatment schools who received deworming and had "side effects" (according to respondent)	0.02	0.18	886
With children in early treatment schools who received deworming, respondent does not know effects	0.10	0.43	886
With children in early treatment schools, respondent does not know whether they received deworming	1.34	1.77	886
With children in early treatment schools who did not receive deworming	0.05	0.31	886
Panel C: Deworming treatment take-up			
Took deworming drugs in 2001 (Group 2 and 3)	0.61	0.49	1,678
Proportion deworming drug take-up in 2001, respondent's own school	0.61	0.28	1,678
Took deworming drugs in 2001, free treatment schools	0.75	0.43	1,255
Took deworming drugs in 2001, cost-sharing schools	0.18	0.38	423
Provided parental consent for deworming drugs in 2001, free treatment schools	0.67	0.41	1,678
Panel D: Cost-sharing interventions			
Cost-sharing school indicator	0.25	0.43	1,678
Cost-sharing school indicator, albendazole only treatment	0.17	0.38	1,678
Cost-sharing school indicator, albendazole and praziquantel treatment	0.08	0.27	1,678
Effective price of deworming per child (Kenyan shillings)	6.3	15.7	1,678

Notes: From 2001 parent questionnaire and NGO administrative records. The "proportion in early treatment schools" variables exclude respondents with no links to program schools (other than their own), hence, the reduced sample since the denominator is zero in that case.

tion strategy. However, it is likely to generate some attenuation bias towards zero in the estimated impact of second- (and higher-) order social contacts on deworming take-up.

In order to keep the theoretical framework tractable, above we considered a network of individuals with uncorrelated signals arranged in a proper tree such that two individuals are linked by a single pathway and there are no redundant links. In practice, however, signals on the impact of deworming are likely to be correlated among individuals within the same school (due to the geographic proximity of particular local schools), and there will be cases in which School A is linked directly to School B both directly through first-order links, and indirectly through second-order links to School C, which, in turn, has direct links to School B. In such cases, the second-order links will still convey some new information since the correlation among signals within a school is not perfect, but they are likely to convey less additional information than second-order links to a school where an individual has no direct first-order links. We focus below on specifications that exclude all such redundant higher-order links to a school, but results are similar when redundant links are included (results not shown).

Parents have 2.9 second-order social links to early treatment schools (standard deviation 2.9) and 4.5 second-order links to all program schools (excluding the respondent's own school, Table I, Panel A). There remains considerable variation in these second-order link measures across individuals, and similar patterns hold for third-order social links.

We have also examined the structure of social connections among the fifty Group 2 and Group 3 schools with complete social network data. In our data there is not a marked sense in which some schools are net "senders" and others net "receivers" of information. The social network is remarkably symmetric: the correlation coefficient of the average number of social links to School A named by individuals in School B, and the average number of links to School B named by individuals in School A, is high at 0.82. The pattern of connections between schools is most strongly influenced by physical distance: for every additional 10 kilometers separating two schools, the average number of named links falls by 0.06 (standard error 0.005, statistically significant at 99 percent confidence). Perhaps surprisingly, schools with the same dominant ethnic group do not have significantly more social connections, nor do schools with similar test score results. An indicator for the location of one of the schools in a market center is not statistically significantly associated with more social connections at traditional confidence levels (regressions not shown). Thus, there does not appear to be huge scope for take-up gains here by exploiting knowledge of the social network to optimally "seed" deworming interventions, and we expand on this point below in the simulations (Section IV.F).

The social effect analysis with parent network data is conducted at the household level using probit estimation, and the outcome measure takes on a value of one if any child in the household was treated with deworming drugs in 2001, and zero otherwise (although results are similar if the analysis is conducted using the child as the unit of observation, results not shown).[15] T_{ij} is the main dependent variable, the 2001 treatment indicator, where i is a household in school j. The idiosyncratic deworming benefit term, e_{ij}, captures unobserved variation in parent beliefs about deworming benefits, tastes for deworming, or the costs of obtaining treatment (for instance, whether the pupil was sick on the treatment day, which increases the cost of walking to school). The individual treatment decision becomes $T_{ij} = 1(N_{ij}^{E'}a + X_{ij}'b + e_{ij} > 0)$, where N_{ij}^{E} is a vector of social links to early treatment schools, defined in 2001 as the Group 1 and 2 schools (not including the respondent's own school). This vector may include both direct (first-order) social links as well as higher-order exposure to early treatment schools.

Among the explanatory variables, X_{ij}, we include total links to all program schools other than the respondent's own school (both for direct and higher-order links), as well as the number of links to non-program schools, and these are represented by the vector N_{ij}. Given the randomized design of the original deworming program, the number of social links to early treatment schools is randomly assigned conditional on total links to other program schools. The interpretation of the coefficient on the total number of links is complicated by the possibility that more sociable individuals (i.e., those able to name more social links) differ from less sociable people in certain unobserved dimensions. However, given the design, this does not affect the estimated impact of early treatment links since the number of early treatment links is orthogonal to the error term conditional on total named links.

15. Treatment within a family is highly correlated, as expected, so we use the household as the unit of analysis.

The cost-sharing indicator variable, $COST_j$, takes on a value of one for schools participating in the cost-sharing project, where the financial cost of treatment was higher. Z_{ij} is a vector of additional household socioeconomic characteristics (parents' education and asset ownership), demographic characteristics (respondent fertility), and other controls (respondent membership in community groups, and a Group 2 indicator) that may affect real or perceived deworming benefits and costs. Idiosyncratic disturbance terms are allowed to be correlated within each school as a result of common influences, such as headmaster efforts in promoting the program. Equation 6 presents the main probit specification:

$$\text{(6)} \qquad \Pr(T_{ij} = 1) = \Phi\{N_{ij}^{E'}a + N_{ij}'b_1 + b_2 COST_j + Z_{ij}'b_3 + e_{ij}\}.$$

We include interaction terms between household characteristics and social links to estimate heterogeneous treatment effects, for example, as a function of respondent education and estimate effects of different types of social connections (e.g., links to relatives versus friends).

To validate the identification strategy, we first confirm that the randomization succeeded in creating program groups balanced along observable dimensions: the number of direct (first-order) social links and second- and third-order exposure to early treatment schools, as well as the Group 2 indicator variable and the cost-sharing indicator, are not significantly associated with most observable household characteristics (Table II), including parent years of education, community group membership (e.g., women's or farming groups), the total number of children in the household, or with household ethnic group or religious affiliation variables (ethnic and religious results not shown). The numbers of first-order and second-order early links are, however, positively and significantly associated with iron roof ownership in one specification (Table II, regression 4), and we thus include these controls in most specifications below to control for any independent effects they may have on take-up. The measure of second-order links to early treatment schools is significantly associated with moderate to heavy infection in 2001 at the 10 percent level, but the coefficient is small (and, surprisingly, positive). Third-order links to early treatment schools are not significantly associated with any observable characteristics.

IV.B. Nonexperimental Social Effect Estimates

We first present nonexperimental social effect estimates. In a specification similar to many existing studies, we examine the take-up rate of children in a predefined local social unit—here the primary school—as the key explanatory variable. We find that the local school treatment rate (excluding the respondent) is strongly positively correlated with take-up, as expected, with coefficient estimate 0.852 (standard error 0.107—Table III, regression 1). Take-up among children who are members of the respondent's own ethnic group in their school is somewhat more influential than take-up in other ethnic groups (regression not shown), a finding similar to Munshi and Myaux [2002], although in our case we argue that this pattern is likely due to omitted variable bias rather than to actual social learning as they claim in their context. Similarly, there is a positive, though not statistically significant, relationship (estimate 0.016, standard error 0.011, regression 2) between the number of treated first-order links named in the survey (among those attending the respondent's school) and take-up, in a specification similar to several other recent studies [Kohler, Behrman, and Watkins 2001; Bandiera and Rasul 2006].

Social links' experiences with deworming may also affect individuals' choices. In particular, we test whether take-up is higher when first-order links had "good" experiences with the technology, as in Conley and Udry [2000]. Having more links whose children had "good effects" is not associated with higher take-up, but those who had more links with "side effects" are somewhat less likely to be treated (Table III, regression 3)—the *p*-value on the hypothesis that the two estimates are equal is .09—but this is only suggestive.[16]

16. The experiences and choices of people in social links' communities may theoretically affect respondent take-up [Munshi 2004]. For each early treatment school, we computed the average difference in 1999 school participation between treated and untreated pupils and use this to classify schools into "large treated minus untreated difference" schools (those above the median difference) versus small difference schools. The treated minus untreated difference captures the average observed private benefit to deworming in that school. However, the effect of links to early treatment schools in large difference schools is not significantly different from the effect in small difference schools. Similarly, links to early treatment schools with low take-up do have a somewhat more negative effect on respondent treatment rates than links to schools with high take-up, but the difference is not significant (not shown). However, omitted variable bias concerns and limited statistical power mean these results should be interpreted cautiously.

TABLE II
VALIDATING THE RANDOMIZATIONS (GROUP 2 AND GROUP 3 HOUSEHOLDS)

	Dependent variable:					
	Respondent years of education	Community group member	Total number of children	Iron roof at home	Distance home to school (km)	Moderate-heavy infection, 2001
	OLS	Probit	OLS	Probit	OLS	Probit
	(1)	(2)	(3)	(4)	(5)	(6)
Explanatory variables:						
# parent links with children in early treatment schools (Group 1, 2, not own school)	0.018	–0.004	–0.039	0.029*	–0.178	–0.003
	(0.085)	(0.012)	(0.067)	(0.014)	(0.128)	(0.018)
# parent links with children in Group 1, 2, or 3 schools, not own school	0.086	0.007	–0.047	–0.016	0.294**	–0.030
	(0.096)	(0.013)	(0.072)	(0.017)	(0.101)	(0.025)
Second-order exposure to early treatment schools (Groups 1 and 2, not redundant with first-order links), parent links	–0.122	–0.004	–0.060	0.021***	–0.168***	0.044***
	(0.083)	(0.010)	(0.060)	(0.012)	(0.086)	(0.023)
Second-order exposure to Group 1, 2 or 3 schools (not redundant with first-order links), parent links	0.058	–0.000	0.044	–0.012	0.031	–0.023
	(0.072)	(0.007)	(0.048)	(0.009)	(0.096)	(0.018)
Third-order exposure to early treatment schools (Groups 1 and 2, not redundant with first- and second-order links), parent links	–0.057	–0.008	0.008	0.006	–0.103	–0.168
	(0.104)	(0.014)	(0.055)	(0.017)	(0.097)	(0.021)

TABLE II
(CONTINUED)

	Dependent variable:					
	Respondent years of education	Community group member	Total number of children	Iron roof at home	Distance home to school (km)	Moderate-heavy infection, 2001
	OLS	Probit	OLS	Probit	OLS	Probit
	(1)	(2)	(3)	(4)	(5)	(6))
Third-order exposure to Group 1, 2 or 3 schools (not redundant with first- and second-order links), parent links	0.101	0.006	0.015	–0.003	0.044	0.024
	(0.078)	(0.011)	(0.051)	(0.013)	(0.084)	(0.018)
Cost-sharing school indicator	0.164	–0.003	0.074	0.013	1.353	0.032
	(0.289)	(0.042)	(0.231)	(0.058)	(0.849)	(0.098)
Group 2 school indicator	–0.581*	–0.030	0.090	0.020	–0.089	–0.210**
	(0.289)	(0.041)	(0.203)	(0.048)	(0.309)	(0.069)
Other social link controls, socio-economic controls (excluding dependent variable)	Yes	Yes	Yes	Yes	Yes	Yes
Number of observations (parents)	1,678	1,678	1,678	1,678	1,678	745
Mean (s.d.) of dependent variable	4.6	0.58	5.5	0.61	1.7	0.31
	(3.9)	(0.49)	(2.3)	(0.49)	(1.9)	(0.45)

Notes: Data from 2001 parent survey, 2001 parasitological survey, and 2001 administrative records. Robust standard errors in parentheses. Disturbance terms are clustered within schools. Significantly different than zero at 95 (*), 99 (**), and 90 (***) percent confidence. The socioeconomic controls include respondent years of education, community group member, total number of children, iron roof at home, and distance from home to school (but when any of these is the dependent variable, it is not included as an explanatory variable). The other social link controls include # parent links with children not in Group 1, 2, or 3 schools, and number of parent links, total.

TABLE III
NONEXPERIMENTAL SOCIAL EFFECT ESTIMATES (GROUPS 2 AND 3)

	Dependent variable: Child took deworming drugs in 2001		
	(1)	(2)	(3)
Explanatory variables:			
Proportion deworming drug take-up in 2001, respondent's own school (not including respondent)	0.852*** (0.107)		
# parent links with children in respondent's own school whose children received deworming		0.016 (0.011)	
# parent links with children in early treatment schools whose children received deworming and had "good effects"			0.004 (0.025)
# parent links with children in early treatment schools whose children received deworming and had "side effects"			−0.152* (0.080)
# parent links with children in early treatment schools whose children received deworming and respondent does not know effects			0.003 (0.049)
# parent links with children in early treatment schools whose children did not receive deworming			−0.006 (0.055)
# parent links with children in early treatment schools, respondent does not know whether they received deworming			−0.010
Total social link controls, socio-economic controls	Yes	Yes	Yes
Number of observations (parents)	1,678	886	886
Mean of dependent variable	0.61	0.56	0.56

Notes: Data from 2001 parent survey and 2001 administrative records. Marginal probit coefficient estimates are presented. Robust standard errors in parentheses. Disturbance terms are clustered within schools. Significantly different from zero at 99 (***), 95 (**), and 90 (*) percent confidence. Social links controls include total number of parent links, number of parent links to Group 1, 2, 3 schools (not own school), and number of links, parent to nonprogram schools. Other controls include respondent years of education, community group member indicator variable, total number of children, iron roof at home indicator variable, and distance from home to school in kilometers, as well as the Group 2 indicator and cost-sharing school indicator. Regression 1 presents results from Round 1 and Round 2 of the 2001 Parent Survey, and regressions 2 and 3 present results from Round 2 alone, since only Round 2 has detailed information regarding deworming treatment impacts on social links. In regression 3, the difference between the coefficient estimates on number of links with children in early treatment schools whose children received deworming and had "good effects" and on number of links with children in early treatment schools whose children received deworming and had "side effects" is marginally significant (p-value = 0.09).

IV.C. Experimental Social Effect Estimates

Experimental social effect estimates are markedly different from the nonexperimental estimates above, suggesting that omitted variable bias in the nonexperimental estimates is large and positive. We begin by considering direct first-order social effects to be comparable with existing work before moving onto higher-order social effect estimates.

Each additional direct parent social link to an early treatment school is associated with 3.1 percentage points lower likelihood that the respondent's children received deworming drugs in 2001, and this effect is significantly different from zero at over 95 percent confidence. (Table IV, regression 1 presents marginal probit estimates evaluated at mean values.) This suggests that the respondent's small, self-defined social network has a major impact on treatment choices: having two additional early treatment links (roughly a one standard deviation increase) reduces take-up by six percentage points.

This result cannot simply be due to imitation or to social effects related to learning about how to use the new technology since the overall effect is negative. This implies that learning about the benefits of the technology plus the infection externality, taken together, are negative and larger in magnitude than the sum of the effect of imitation and the effect due to learning to use the technology. A quadratic term in parent social links to early treatment schools is also statistically significantly different from zero at 95 percent confidence in some specifications (Appendix Table X, regression 1). However, this quadratic term is not significant for interactions with household characteristics, nor is the quadratic second-order early treatment exposure term statistically significant (regressions not shown), so we principally focus on the linear measure for simplicity in what follows.[17]

None of the demographic or socioeconomic controls is significantly associated with 2001 take-up except for distance from home to school, which is negatively related to take-up and large: take-up drops nearly two percentage points for each additional kilometer from home to school (using GPS measures). Distance apparently makes it costlier for parents to walk to school to provide written consent for deworming and for children to attend school, a first piece of empirical evidence that take-up is sensitive

17. Given the correlation of information among individuals in the same school, it is theoretically possible that the first signal in a particular school would be more influential than subsequent signals. We estimated these effects in our data but, due to limited statistical power, cannot reject the hypothesis that the first, second, and third links to a particular early treatment school all have the same impact on take-up (regressions not shown).

TABLE IV
EXPERIMENTAL SOCIAL EFFECT ESTIMATES

	Dependent variable: Child took deworming drugs in 2001				
	(1)	(2)	(3)	(4)	(5)
Explanatory variables:					
# parent links with children in early treatment schools (Groups 1 and 2, not own school)	−0.031**	−0.040**			−0.002
	(0.014)	(0.017)			(0.018)
# parent links with children in early treatment schools		0.017			
* Group 2 school indicator		(0.029)			
Proportion direct (first-order) parent links with children in early treatment schools			−0.098**		
			(0.045)		
# parent links with children in early treatment schools, with whom respondent speaks at least twice/week				−0.030**	
				(0.016)	
# parent links with children in early treatment schools, with whom respondent speaks less than twice/week				−0.033	
				(0.033)	
# parent links with children in Group 1, 2, or 3 schools, not own school, with whom respondent speaks at least twice/week				0.008	
				(0.012)	
# parent links with children in Group 1, 2, or 3 schools, not own school, with whom respondent speaks less than twice/week				0.026	
				(0.027)	
# parent links with children in early treatment schools					−0.0062*
* Respondent years of education					(0.0032)
# parent links with children in Group 1, 2, or 3 schools, not own school	0.013	0.012	−0.006		−0.014
	(0.011)	(0.017)	(0.009)		(0.014)
# parent links with children not in Group 1, 2, or 3 schools	−0.007	−0.008	−0.005	−0.007	−0.008
	(0.007)	(0.009)	(0.007)	(0.007)	(0.011)
# parent links, total	0.019***	0.029***	0.021***	0.018***	0.013
	(0.005)	(0.007)	(0.007)	(0.005)	(0.008)

TABLE IV
(CONTINUED)

	Dependent variable: Child took deworming drugs in 2001				
	(1)	(2)	(3)	(4)	(5)
Respondent years of education	0.003	0.003	0.002	0.002	−0.016
	(0.003)	(0.003)	(0.004)	(0.003)	(0.012)
Community group member	0.027	0.031	0.037	0.029	0.023
	(0.026)	(0.026)	(0.029)	(0.026)	(0.026)
Total number of children	0.005	0.006	0.004	0.005	0.006
	(0.006)	(0.006)	(0.007)	(0.006)	(0.006)
Iron roof at home	0.011	0.008	0.011	0.011	0.009
	(0.026)	(0.027)	(0.032)	(0.026)	(0.027)
Distance home to school (km)	−0.018**	−0.018**	−0.015	−0.018*	−0.018**
	(0.009)	(0.009)	(0.010)	(0.009)	(0.009)
Group 2 school indicator	0.015	0.201**	0.007	0.015	0.015
	(0.045)	(0.086)	(0.046)	(0.046)	(0.045)
Cost-sharing school indicator	−0.580***	−0.577***	−0.578***	−0.581***	−0.582***
	(0.054)	(0.054)	(0.058)	(0.054)	(0.054)
Number of observations (parents)	1,678	1,678	1,358	1,678	1,678
Mean of dependent variable	0.61	0.61	0.61	0.61	0.61

Notes: Data from 2001 Parent Survey and 2001 administrative records. Marginal probit coefficient estimates are presented. Robust standard errors in parentheses. Disturbance terms are clustered within schools. Significantly different from zero at 99 (***), 95 (**), and 90 (*) percent confidence. Regression 2 also includes interaction terms (number of parent social links with children in Group 1, 2, or 3 schools, not own school)*(Group 2), (number of parent social links with children not in Group 1, 2, or 3 schools)*(Group 2), and (number of parent social links, total)*(Group 2). Regression 3 excludes parents for which (number of parent social links with children in Group 1, 2, or 3 schools, not own school) = 0, since the proportion of links is undefined, leading to the reduction in sample size. Regression 5 also includes interaction terms (number of parent social links with children in Group 1, 2, or 3 schools, not own school)*(Respondent years of education) and (number of parent social links with children not in Group 1, 2, or 3 schools)*(Respondent years of education), not shown.

to treatment costs. Parent years of education (typically maternal education in our sample) is positively but not statistically significantly associated with higher take-up (point estimate 0.003, standard error 0.003, Table IV, regression 1).

Social effects are more negative for Group 3 schools (point estimate −0.040, Table IV, regression 2) than for Group 2 (−0.023, the sum of the direct effect of early treatment links and its interaction with the Group 2 indicator), although the difference is not statistically significant. This pattern of coefficient estimates is reasonable: Group 2 parents had by 2001 already observed the impact of deworming treatment in their own household and community and should therefore be less influenced than Group 3 parents by early links (i.e., in (5), σ_N^2 is smaller for Group 2 parents than Group 3 parents). Nonetheless, the persistent influence of early links on Group 2 households after two years of the program is noteworthy. One possible non-Bayesian explanation is that initial pieces of information carry disproportionate weight in subsequent decision making [Rabin and Schrag 1999].[18]

The results are robust to including the proportion of links with children in early treatment schools rather than the number of such links (Table IV, regression 3), and to controlling for the total number of parent social links nonparametrically using a set of indicator variables (results not shown). An interaction between the cost-sharing indicator and the number of early treatment links is imprecisely estimated, but is near zero and not statistically significant (estimate −0.013, standard error 0.039—regression not shown).[19]

18. A finding that casts some doubt on the "first impressions matter" explanation, however, is the fact that links to Group 1 schools (phased in during 1998) have nearly identical impacts as links to Group 2 schools (phased in during 1999, estimates not shown). Note that the persistent effects of early treatment links on take-up might be reconciled with Bayesian learning, though, if individuals believed there was an important school-year specific random component to treatment effects, leading them to place extra weight on outcomes in schools other than their own.

19. The results are also robust to a specification without socioeconomic controls (Table X, regression 2) and to the inclusion of additional ethnic and religious controls and indicators for whether the respondent is a member of the dominant local ethnic and religious group (regression 3); none of the six ethnic group indicator variables is significantly related to take-up. The results are similar when the local density of early treatment school pupils (located within three kilometers of the respondent's school) and the density of all local primary school pupils are included as controls (regression 4). However, the point estimate on early links falls by more than a third and loses statistical significance, possibly because the local density of early treatment schools picks up part of the effect of

Several pieces of evidence suggest that learning takes place not only among individuals with strong social ties but also among those with relatively weak ties, along the lines of Granovetter [1973]. When the framework is extended to include different types of parent social links—"close" friends, defined as those with whom the respondent speaks at least twice a week, versus relatively "distant" friends—each additional close link to an early treatment school is associated with 0.030 lower probability of deworming treatment in 2001 and the estimated effect of distant links is similar, although not statistically significant due to reduced precision (Table IV, regression 4, estimate −0.033, standard error 0.033).[20] We are similarly unable to reject the hypotheses that social effects are the same for links to relatives versus nonrelatives, or for members of the respondent's own ethnic group versus other groups, conditional on being named a social link (results not shown).

Social effects are more strongly negative for respondents with more education (Table IV, regression 5). Other studies—most notably Foster and Rosenzweig [1995]—find that educated individuals learn most rapidly about new technologies and adopt first. Note that the overall impact of an additional year of schooling on deworming take-up remains positive though not statistically significant when all the education interaction terms, including the terms interacting education with total links, are considered (interaction term coefficient estimates not shown in regression 5).

Additional social links could have a larger impact on more educated individuals in the theoretical framework presented above if they had overly optimistic prior beliefs (ϕ_{i0}) about the drugs rather than any greater receptiveness to new information. Although we cannot decisively distinguish these two explanations empirically, the relation between respondents' education and their stated belief that deworming drugs are "very effective" does provide further evidence supporting the overoptimism model.

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interactions with other individuals not named in the social links roster. An F-test indicates that the early treatment social links and local density of early treatment pupils terms are jointly significant at 99 percent confidence.

20. Using another definition of link strength yields similar results. While most links were provided in response to questions about the individuals with whom the respondent speaks most frequently, others were provided in response to prompts about contacts in particular local schools. There is not a statistically significant difference in the effects of "unprompted" and "prompted" links (in fact, prompted links are somewhat more influential—not shown).

Among Group 3 parents interviewed in Round 1, before deworming treatment was phased into their schools, individuals who had completed primary school were 17 percentage points more likely to believe deworming drugs are "very effective" than parents who had not completed primary school. However, several months after deworming had been introduced into their schools, this falls by about half to a 9 percentage point gap between more educated and less educated Group 3 parents interviewed in Round 2 (recall that parents were randomly allocated between survey rounds), and there is a similar gap among Group 2 parents in 2001, at 10 percentage points, two years after these schools had begun receiving treatment. Presenting the result in levels rather than differences, among Group 3 parents who completed primary school the perceived effectiveness of deworming also fell dramatically from 59 to 45 percent from Round 1 to Round 2 but fell only slightly among the less educated. To summarize, through exposure to deworming over time, views toward the drugs partially converged across parents with different educational levels, and the drugs were increasingly viewed as ineffective among Group 3 parents. As the medical effectiveness of the drugs is well-documented, we conjecture that their disillusionment with the drugs is due to reinfection.

We also estimate social effects as a function of child social contacts in early treatment schools using the 2001 pupil questionnaire data. Average social connections across schools (for the Group 2 and Group 3 schools) are very similar for parents and children with a correlation coefficient of 0.92, and this complicates the task of distinguishing between parent and child impacts. Among those children aged thirteen years and older, the estimated effect of direct child social links is negative, similar to the parent first-order early treatment estimate and statistically significant at over 95 percent confidence in a specification analogous to those in Table IV (point estimate −0.028, standard error 0.012). However, the point estimate is much smaller for younger children (−0.006, standard error 0.014—regressions not shown). Multiple interpretations of this pattern are possible, including the possibility that adolescents are more influenced by peer information or pressure than younger children, as claimed by Steinberg and Cauffman [1996], or perhaps that younger children are less able to process health information from their social contacts, or that the interaction of information from parents and adolescents is particularly influential.

Unfortunately, we only have limited statistical power to disentangle parent and child impacts or to investigate possible interaction effects due to the high correlation of parent and child social networks and because matched information on both parent and child social networks exists for only a limited subset of children, reducing the sample size in the child network regressions by over half. When parent and child first order social links to early treatment schools are both included as explanatory variables, both coefficient estimates remain negative but are no longer statistically significant due to the large increase in standard errors (regression not shown).[21]

We next consider higher-order exposure to early treatment schools through parent social networks. After reproducing the main direct first-order social link result (Table V, regression 1), we examine the impact of second-order exposure to early treatment schools, where second-order links are constructed using school average connections, and find that second-exposure to early treatment schools is also associated with significantly lower deworming drug take-up in 2001 (estimate −0.035, standard error 0.013, regression 2), conditional on total second-order exposure to all program schools. When both first-order and second-order social networks terms are included, the estimated second-order effect is −0.047, nearly identical to the average first-order effect of −0.044, and both effects are statistically significant at high levels of confidence (regression 3). While the theoretical framework predicts that coefficients should decline monotonically for higher-order links along the transition path to steady state (since information from more distant social links is less likely to have reached the individual), we cannot reject the hypothesis that the coefficient estimates on the first-order and second-order links are equal or that first-order effects are somewhat more negative, so we do not emphasize this difference. An increase of one standard deviation in second-order early treatment school exposure is associated with a very large 19 percentage point reduction in deworming take-up. Mirroring the first-order results, more total second-order exposure to all schools (not just early treatment schools) is associated with higher take-up, which we interpret as reflecting a positive correlation between overall individual “sociability” and positive priors toward deworming in our sample.

21. Refer to the working paper version [Miguel and Kremer 2003] for further discussion of child social effects.

TABLE V
FIRST-ORDER AND HIGHER-ORDER SOCIAL EFFECT ESTIMATES (GROUPS 2 AND 3)

	Dependent variable: Child took deworming drugs in 2001				
	(1)	(2)	(3)	(4)	(5)
Explanatory variables:					
# parent links with children in early treatment schools (Groups 1 and 2, not own school)	−0.031**	—	−0.044***	—	−0.037***
	(0.014)		(0.015)		(0.015)
# parent links with children in Group 1, 2, or 3 schools, not own school	0.013	—	0.021	—	0.021
	(0.011)		(0.015)		(0.015)
Proportion direct (first-order) parent links with children in early treatment schools	—	—	—	−0.140***	—
				(0.048)	
Second-order exposure to early treatment schools (Groups 1 and 2, not own school), parent links	—	−0.035***	−0.047***	—	−0.049***
		(0.013)	(0.013)		(0.013)
Second-order exposure to Group 1, 2, or 3 schools (not own school), parent links	—	0.021**	0.032***	—	0.032***
		(0.010)	(0.012)		(0.012)
Proportion second-order parent links with children in early treatment schools	—	—	—	−0.231***	—
				(0.087)	
Third-order exposure to early treatment schools (Groups 1 and 2, not own school), parent links	—	—	—	—	−0.015
					(0.012)
Third-order exposure to Group 1, 2, or 3 schools (not own school), parent links	—	—	—	—	0.008
					(0.010)
Total social link controls, socio-economic controls	Yes	Yes	Yes	Yes	Yes
Number of observations (parents)	1,678	1,678	1,678	1,173	1,678
Mean of dependent variable	0.61	0.61	0.61	0.61	0.61

Notes: Data from 2001 parent survey and 2001 administrative records. Marginal probit coefficient estimates are presented. Robust standard errors in parentheses. Disturbance terms are clustered within schools. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence. Social links controls and other controls are included in all specifications. Social links controls include total number of parent links, number of parent links to Group 1, 2, 3 schools (not own school), and number of parent links to nonprogram schools. Other controls include respondent years of education, community group member indicator variable, total number of children, iron roof at home indicator variable, and distance from home to school in kilometers, as well as the Group 2 indicator and cost-sharing school indicator.

The negative second-order effects we estimate suggest that higher-order links can affect behavior not only by influencing the take-up *behavior* of first-order links, but also through changing the *information* of first-order links. To see this, note that theoretically one could imagine negative imitation effects, if people like to be different from their neighbors. However, a model in which higher-order links affect behavior only through changing the behavior of intermediate links (such as a pure imitation model) would imply that the impact of second-order link adoption should be equal to the square of the first-order link effect of −0.044, or 0.002. Given the results below ruling out large infection externalities at the level of individual social contacts, the large negative coefficient on second-order adoption we estimate thus provides additional evidence that diffusion works via information transmission through social networks.

This negative social learning result holds and is highly statistically significant for both first-order and second-order links when the proportion of early treatment exposure is used (Table V, regression 4) rather than the number of links. The interaction between second-order early treatment school exposure and respondent education remains negative, as was the case for first-order links, but the point estimate is not statistically significant (regression not shown). The second-order exposure results also hold if the first-order exposure is constructed using average *school* social network connections in a manner analogous to the construction of the higher-order links (coefficient estimate is −0.077, standard error 0.036, significant at 95 percent confidence—regression not shown).

Extending the analysis, we find that third-order exposure to early treatment schools—constructed analogously to the second-order links, using school averages for higher-order connections—is not statistically significantly associated with deworming take-up, although the point estimate is again negative (Table V, regression 5). Within the theoretical framework we outline in Section III, a possible explanation for the weaker estimated third-order effect is that insufficient time had passed for some third-order social contacts' information to reach respondents, perhaps because social contacts only discuss deworming infrequently, as suggested by our survey data.

IV.D. Further Econometric Identification Issues

The estimated negative peer effect in technology adoption implies that social learning about the benefits of deworming and the infection externality taken together are negative and far larger in magnitude than any possible social learning about how to use the new technology plus imitation effects. Here we argue that infection effects cannot empirically explain even a small fraction of the overall direct first-order social effect of –3.1 percent (Table IV, regression 1), since any plausible estimate of the effect of early treatment school social contacts on infection status, times the effect of infection on take-up, is much smaller. Thus social learning about deworming benefits appears to be the key channel driving our results.

First, having additional direct social links to early treatment schools is associated with lower rates of moderate-heavy helminth infection, as expected (Table II, regression 6), but the effect is small and not statistically significant (coefficient estimate −0.3 percentage points, relative to a mean moderate-heavy infection rate of 27 percent). An additional second-order social link to early treatment schools is even associated with a somewhat higher rate of infection, though the estimate is only statistically significant at 90 percent confidence. Note that this relatively weak relationship between early treatment school social links and child infection is not inconsistent with the strong infection externality findings in Miguel and Kremer [2004]. Worm infections are not transmitted directly from person to person but rather through contaminated soil and water, and a child's named social links constitute only a small fraction of all people who defecate near the child's home, school, and church, or who bathe at the same points on Lake Victoria.

In terms of the second step—from infection status to take-up—prior infection status is not significantly associated with drug treatment for either Group 1 in 1998 or Group 2 in 1999 [Miguel and Kremer 2004], or for Groups 2 and 3 in 2001 (results not shown), and the point estimates suggest that moderate-heavy worm infection is weakly *negatively* related to treatment rates.[22] Of course, the cross-sectional correlation between infection and treatment cannot be interpreted as causal due to omitted variables: children from unobservably low socioeconomic status households may have both high infection rates and low take-up, for example. However, the treated and untreated children look

22. The 2001 worm infection results are for a subsample of only 745 children who were randomly sampled for stool collection and were present in school on the day of the parasitological survey. Due to the relatively small sample size, we do not focus on the parasitological data in the main empirical analysis.

remarkably similar along many observable baseline socioeconomic and health characteristics [Miguel and Kremer 2004], and the relationship is similar using school-level average infection rates rather than individual data (not shown), weakening the case for strong selection into deworming treatment.

Further evidence that more infected people are not much more likely to take up the drugs is provided by the 1999 cross-school infection externality estimates, identified using exogenous program variation in the local density of early treatment schools. Although we find large average reductions in moderate-heavy worm infection rates as a result of cross-school externalities (an average reduction in infection of 0.23 [Miguel and Kremer 2004]), proximity to early treatment schools leads to an average reduction in drug take-up of only 0.02, which has the expected sign but is near zero (regression not shown). Using this estimate, having a moderate-heavy infection is associated with a 0.02/0.23 = 0.09 reduction in the likelihood of treatment, and this implies a drop in take-up due to infection first-order social effects of only (0.09)*(−0.3 percent) = 0.03 percent, rather than the −3.1 percent overall reduction we estimate. Even if eliminating a moderate-heavy infection reduced the likelihood of drug take-up by a massive 0.5 on average (rather than the 0.09 we estimate), health externalities would account only for a (0.5)*(−0.3 percent) = −0.15 percent reduction in take-up.

Pupil transfers among local primary schools are another potential concern, but any resulting bias would likely work against our findings. For example, parents with more health-conscious social contacts, whose children may have been more likely to transfer into early treatment schools to receive deworming, may themselves also be more health-conscious and eager to have their own children receive treatment. This would bias the estimated social effect upward in which case our negative social effect estimate would be a bound on the true negative effect. In any case, the rate of pupil transfers between treatment and comparison schools was low and nearly symmetric in both directions [Miguel and Kremer 2004], suggesting that any transfer bias is likely to be small.

A related identification issue concerns whether social networks measured in 2001—three years after the program started—were themselves affected by the program. Any extent to which health-conscious individuals became more socially linked to individuals with children in early treatment schools would again lead to an upward bias, working against the negative effects we estimate. However, respondents were statistically no more likely to name early treatment links than links to other schools: the average number of links to early treatment schools is 1.92, while (total number of links to PSDP schools) × (total number of Group 1, 2 pupils/total number of Groups 1, 2, 3 pupils) is nearly identical at 1.91.

IV.E. Parent Attitudes and Knowledge

Respondents with more direct (first-order) early treatment links are significantly more likely to claim that deworming drugs are "not effective" (respondents could choose between "not effective," "somewhat effective," and "very effective," Table VI, row 1).[23] This is consistent with the hypothesis that some people initially thought deworming would provide large and persistent private benefits but learned otherwise from their early treatment school contacts. We do not find a significant impact of additional early links on beliefs that deworming drugs are "very effective" although the point estimate is negative (row 2), nor that the drugs have "side effects" (row 3). This last result is evidence against the possibility that drug side effect rumors were the key driver of lower take-up among those with more early treatment links.

Second-order early treatment exposure does not have a statistically significant effect on parents' belief that deworming drugs are "not effective" (regressions not shown). The discrepancy between first-order and second-order effects on deworming attitudes may be due to the deterioration of information quality with higher-order social connections: speculatively, individuals may learn from their higher-order social contacts that deworming is basically "not good" even though the precise reason why is lost to them.

Although direct first-order early treatment links do affect the belief that deworming drugs are "not effective," they do not affect beliefs that "worms and schistosomiasis are very bad for child health" (Table VI, row 4). However, some parents may report what they think the survey enumerator wants to hear regarding worms' health consequences: 92 percent of respondents claimed that helminth infections are "very bad" for child health, even

23. A fourth option, "effective, but the worms come back" was rarely chosen by respondents.

though take-up is much lower than 92 percent. The number of direct early treatment links has no effect on parents' self-reported knowledge of the ICS (NGO) deworming program, the effects of worms and schistosomiasis (rows 5 and 6), or the deworming treatment status of their own child (not shown). It also did not affect their objective knowledge of common worm infection symptoms (rows 7–10). Respondents could name only 1.8 of ten common symptoms on average.[24] This suggests health education messages failed to spread.

Nonexperimental methods would have suggested different results. The actual number of treated social links and the number of social links with whom the respondent speaks directly about deworming are both positively and significantly related to most deworming attitudes and knowledge outcomes (Table VI). The observed positive correlation in outcomes within social networks in the study area appears to be due to omitted variables rather than actual peer effects. Those with unobservably more interest in child health plausibly discuss worms more frequently with social links, who are themselves more likely to have their own children receive treatment.

IV.F. Simulating Take-Up along the Transition Path

The framework in Section III suggests that subsidies to take-up will not affect steady-state adoption under social learning either about how to use new technologies or learning about their benefits, as long as at least a subset of the population uses the technology. However, subsidies could potentially have effects along the transition path to the steady state. We therefore use the empirical school-to-school social connections matrix to simulate the take-up gains along the transition path from a one-time drug subsidy for parameter values that match the estimated first-order social effects. We consider a hypothetical technology where true private benefits exceed most people's expectations as it is of more general interest to study technologies where social learning could potentially contribute to take-up.

The simulation is based on the theoretical framework in Section III with several functional form assumptions made for

24. The ten symptoms (row 7) include fatigue, anemia, weight loss, stunted growth, stomach ache, bloated stomach, blood in stool, worms in stool, diarrhea, and fever. Parents were asked: "Could you name the symptoms of worm and schistosomiasis infections?" and their unprompted responses were recorded by the enumerator.

TABLE VI
EFFECTS ON DEWORMING ATTITUDES AND KNOWLEDGE

	Estimate on # parent links with children in early treatment schools	Estimate on # parent links with children in early treatment schools whose children received deworming	Estimate on # parent links with children in early treatment schools with whom respondent spoke about deworming	Mean dep. var.
	Experimental	Nonexperimental	Nonexperimental	
Dependent variable:				
Panel A: attitudes				
Parent thinks deworming drugs "not effective"	0.017**	0.009	0.009**	0.12
	(0.007)	(0.009)	(0.004)	
Parent thinks deworming drugs "very effective"	−0.007	0.042**	0.040***	0.43
	(0.010)	(0.013)	(0.007)	
Parent thinks deworming drugs have "side effects"	0.000	0.004	0.003*	0.04
	(0.003)	(0.003)	(0.002)	
Parent thinks worms and schisto. "very bad" for child health	−0.001	0.001	−0.006*	0.92
	(0.006)	(0.008)	(0.003)	
Panel B: knowledge				
Parent "knows about ICS deworming program"	0.004	0.054***	0.055***	0.70
	(0.011)	(0.014)	(0.011)	
Parent "knows about the effects of worms and schistosomiasis"	−0.001	0.055***	0.039***	0.68
	(0.013)	(0.014)	(0.009)	
Number of infection symptoms parents able to name (0–10)	−0.029	0.078***	0.076***	1.8
	(0.025)	(0.029)	(0.015)	

TABLE VI
(CONTINUED)

	Estimate on # parent links with children in early treatment schools	Estimate on # parent links with children in early treatment schools whose children received deworming	Estimate on # parent links with children in early treatment schools with whom respondent spoke about deworming	Mean dep. var.
	Experimental	Nonexperimental	Nonexperimental	
Parent able to name "fatigue" as symptom of infection	−0.004	0.032***	0.021***	0.20
	(0.010)	(0.008)	(0.006)	
Parent able to name "anemia" as symptom of infection	0.005	−0.001	0.010**	0.22
	(0.009)	(0.011)	(0.005)	
Parent able to name "weight loss" as symptom of infection	0.002	0.002	−0.001	0.06
	(0.006)	(0.004)	(0.004)	

Notes: Data from 2001 parent survey and 2001 administrative records. Marginal probit coefficient estimates are presented for all binary variables, and each entry is the result of a separate regression. Robust standard errors in parentheses. Disturbance terms are clustered within schools. Significantly different from zero at 99 (***), 95 (**), and 90 (*) percent confidence. Social links controls and other controls are included in all specifications. Social link controls include total number of parent links, number of parent links to Group 1, 2, 3 schools (not own school), and number of parent links to non-program schools. Other controls include respondent years of education, community group member indicator variable, total number of children, iron roof at home indicator variable, and distance from home to school in kilometers, as well as the Group 2 indicator and cost-sharing school indicator. The number of observations (parents) across regressions ranges from 1656 to 1678 depending on the extent of missing data for the dependent variable.

The ten possible infection symptoms (row 7) include fatigue, anemia, weight loss, stunted growth, stomach ache, bloated stomach, blood in stool, worms in stool, diarrhea, and fever. Parents were asked: "Could you name the symptoms of worm and schistosomiasis infections?" Their responses were recorded by the enumerator.

tractability. We assume that the health benefits of the technology times idiosyncratic utility from using the technology (the $\gamma(X_i) \cdot \mu_i$ term) is uniformly distributed on the interval $(\underline{b}, \bar{b})$; assume that everyone in a given school starts out with the same prior belief on benefits but that priors differ across schools (and thus focus on the diffusion of information across schools rather than on heterogeneity within schools); and assume that all the social effects we observe are due to learning about the benefits of the technology.

One time period in the simulation roughly corresponds to one month. Information may diffuse between schools in each period, but individuals only get an opportunity to adopt the technology once every six months (as in the program we study). Our results are qualitatively robust to either shorter ($\tau = 1$) or longer ($\tau = 12$) lags between adoption opportunities. For tractability we assume that information diffuses instantly within schools.

We consider parameter values for which the simulated first-order social effects fall within two standard deviations of the first-order social effect estimated empirically, though again we consider diffusion of a hypothetical technology for which actual returns exceed prior beliefs, so social learning speeds adoption.[25] While we do not explicitly match parameter values to the empirically estimated second-order social effect, the simulated second-order effect is, on average, close to the estimated second-order effects. As in our data, the simulated second-order effect is of a similar magnitude to the simulated first-order effect—the difference between the simulated second-order and first-order social effects is, on average, 0.006 (relative to an average simulated first-order effect of 0.02, a slightly smaller magnitude than the effect estimated in Section IV.C above).

For a wide range of parameter values, we find that beliefs about the technology and take-up rates converge quickly (within five adoption opportunities) to very close to the correct long-run

25. We focus on the following range of parameter values for the model: $\underline{b} = 0$, $\bar{b} = 2$, $\sigma_0^2 = 1$, $\sigma_\varepsilon^2 = 1$–10, $C = 0.1$–2, $\phi = 0.75$, $\tau = 6$, annual discount rate $\delta = 0.9$–1, and $p = 0.05$–0.2. In the simulation, we assume that all students within a school receive separate signals and exchange information. However, to compensate for making this extreme assumption, we also assume there are only a maximum of fifty possible signals that can be received per school with full take-up; with more signals per school, convergence is even faster. Given $\underline{b}$ and $\bar{b}$, varying C between 0.1 and 2 covers all of the relevant cases. Similarly, fixing σ_0^2, choosing various values for σ_ε^2 covers all of the interesting cases, since only their relative magnitudes influence weight placed on signals versus prior beliefs. The simulation code and complete results are available from the authors upon request.

value. Even in a case where signals have high variance (e.g, $\sigma_\varepsilon^2 = 9$), by the third adoption opportunity the variance of posterior beliefs is, on average, less than 0.01.

Optimal "seeding" of a particular school with a one-time drug subsidy (in period one) makes little difference to total discounted technology take-up. After thirty opportunities to adopt (fifteen years of a program like the one we study), the difference in total discounted take-up between seeding the single "best" school—the school that generates the highest total discounted take-up when seeded—versus the average of seeding a randomly chosen school in the sample is negligible (less than 0.01 percent) for our range of plausible parameter values. This finding of small gains to "optimal seeding" is consistent with the largely symmetric observed social network structure across schools (Section IV.A). Given that it may be costly to identify the optimal school to subsidize and that those funds could alternatively be spent on subsidizing drugs for additional schools (or subsidizing them for a longer period), efforts to target temporary drug subsidies to influential "opinion leader" schools appear misguided in our context.

Finally, even the take-up gains from one-time subsidies to additional schools are quite small on average. Since information diffuses rapidly, these gains are primarily comprised of the direct effect of the subsidy on take-up in the initial round; the impact of information spillovers is negligible. The indirect effects on take-up (through the generation of additional information) are small in magnitude and exhibit diminishing returns to additional subsidies. Total discounted take-up increases by only 0.027 percent (as a percentage of take-up in the absence of the subsidy) on average above and beyond the direct effect of the subsidy when a single school is subsidized at random. Going from subsidizing five to ten schools yields an additional marginal gain of only 0.016 percent per school.

Thus, at least in this particular context, there is little reason to think temporary subsidies will lead to a sustainable increase in technology adoption. More generally, even if a hypothetical social planner knew the returns to a particular technology were better than people expected, subsidizing even a small fraction of the population for a relatively brief period would have been sufficient to assure long-run diffusion. In the absence of strong imitation effects, the fact that dynamic gains to subsidizing additional schools are small suggest that a "big push" is unnecessary for a technology that spreads naturally—and, of course, is futile in the long run for a technology where social effects are negative. To be effective in boosting adoption, ongoing subsidies appear necessary in that case.

V. The Impact of Subsidies on Drug Take-Up

In the remainder of this paper, we examine the effects of three other approaches to making deworming sustainable: cost-sharing through user fees (Section V), health education lessons (Section VI), and a mobilization intervention (Section VII).

Cost-sharing through user fees has been advocated as necessary for the sustainability of public health services in many less developed countries [World Bank 1993b]. Revenues from these fees could be used to improve the quality of health services (i.e., through expanded drug availability) or to fund other government expenditures. User fees could theoretically promote more efficient use of scarce public resources if those in greatest need of health services are willing to pay the most for them.

Several nonexperimental studies from Africa have found large drops in health care utilization after the introduction of user fees (e.g., McPake [1993], Meuwissen [2002]), including in Kenya, where Mwabu, Mwanzia, and Liambila [1995] find utilization fell by 52 percent in 1989. Our analysis uses random assignment to estimate the effect of cost sharing.[26] The theoretical framework in Section III suggests that increasing the monetary cost of deworming should lead to lower drug take-up, but the actual elasticity of demand needs to be estimated. Seventy-five percent of households in the free treatment schools received deworming drugs in 2001 (Table I, Panel C), while the rate was only 19 percent in cost-sharing schools (the survey data used in these regressions is described in Section IV). A regression analysis suggests the small fee-reduced treatment by 58 percentage points (Table VII, regression 1), with the effect similar across households with various socioeconomic characteristics (regression 2).[27]

26. Gertler and Molyneaux [1996] find that utilization of medical care is highly sensitive to price in an experimental study in Indonesia, but since the unit of randomization in their analysis is the district, and their intervention affected only eleven districts, statistical power is relatively low. In a large-scale experimental study, Manning et al. [1987] find in contrast that the price elasticity of demand for medical services in the United States is a modest −0.2.

27. Results are unchanged if Group 1 households are included in the analysis (results not shown). They are excluded here since they lack the social networks data that we use as explanatory variables here and in Section IV above.

TABLE VII
THE IMPACT OF COST-SHARING

	Dependent variable: Child took deworming drugs in 2001		
	(1)	(2)	(3)
Explanatory variables:			
Cost-sharing school indicator	−0.580***	−0.459***	−0.572***
	(0.054)	(0.122)	(0.080)
Cost-sharing *Respondent years of education		0.002	
		(0.007)	
Cost-sharing *Community group member		0.021	
		(0.072)	
Cost-sharing *Total number of children		−0.021	
		(0.016)	
Cost-sharing *Iron roof at home		−0.047	
		(0.064)	
Effective price of deworming per child(= cost/# household children in that school)			−0.001
			(0.002)
1/(# household children in that school)			−0.348***
			(0.066)
Social links, other controls	Yes	Yes	Yes
Number of observations (parents)	1,678	1,678	1,678
Mean of dependent variable	0.61	0.61	0.61

Notes: Data from 2001 parent survey and 2001 administrative records. Marginal probit coefficient estimates are presented. Robust standard errors in parentheses. Disturbance terms are clustered within schools. Significantly different from zero at 99 (***), 95 (**), and 90 (*) percent confidence. Social links controls include total number of links, number of links to Group 1, 2, 3 schools (not own school), and number of links to non-program schools (as in Table IV above). Other controls include respondent years of education, community group member indicator variable, total number of children in the household, iron roof at home indicator variable, and distance from home to school in kilometers, as well as the Group 2 indicator (as in Table IV above).

This negative effect of monetary cost is consistent with our finding (in Table IV) of large negative effects of household distance to the school, which proxies for the time costs as parents need to walk to school to provide written consent.

The drop in take-up in cost-sharing schools cannot be attributed to the hypothesis that user fees help ensure that scarce health resources are directed to those who need them most. In fact, sicker pupils were no more likely to pay for deworming drugs: the coefficient estimate on the interaction between 2001 helminth infection status and the cost-sharing indicator is not statistically significant (not shown).

Variation in the deworming price per child was generated by the fact that cost-sharing came in the form of a per family fee, so that parents with more children in the primary school in 2001 effectively faced a lower price per child. Cost-sharing reduced treatment rates regardless of the per-child price that the household was required to pay (Table VII, regression 3). Ariely and Shampan'er [2004] similarly find sharp decreases in demand for goods with a small positive price relative to goods with a zero price in lab experiments. This regression specification also includes the inverse of the number of household children in primary school and the total number of household children of all ages as additional explanatory variables to control for the direct effects of household demographic structure on deworming drug demand and, thus, to isolate the price effect. However, we cannot explicitly control for the interaction between family size and price changes, given the school-level randomization design.

The cost-sharing results suggest that introducing a small positive user fee is a particularly unattractive policy in this context, since it dramatically reduces take-up while raising little revenue and typically requires considerable administrative cost. Yet, this is precisely the approach that many less developed countries, including Kenya, have adopted in the health sector [World Bank 1994; McPake 1993]. The net public cost per pupil treated in our program under a full subsidy was US$1.478. Assuming a US$15 per school fixed cost of visiting a school (which we base on actual field costs), and a US$0.03 cost per pupil of collecting funds, the net public cost per student treated under cost sharing was US$1.374. Pupils contributed about US$0.30 additionally in cost-sharing schools. For a fixed public budget B, the difference between the total number of students treated under cost-sharing versus under a full subsidy in this case will be $(B/1.374) - (B/1.478) = B^*0.0512$. The extra revenue collected from the private sector under cost-sharing will be US$0.30*$(B/1.374) = B^*0.2183$. The cost per additional student treated under cost-sharing is thus $(B^*0.2183)/(B^*0.0512) =$ US$4.26. One can understand why a program administrator with a fixed public budget might institute cost-sharing, but since the cost per additional student treated under a full subsidy would be only US$1.478, the deadweight cost of taxation would have to be enormous to make it rational for governments to seek to finance deworming out of user fees rather than through taxation.

It is worth bearing in mind the sequencing of the current project in interpreting the cost-sharing results. Prior to the program, fewer than 5 percent of people reported taking deworming drugs [Miguel and Kremer 2004]. The schools received free treatment for two or three years, after which half the Group 1 and 2 schools were assigned to cost-sharing, following NGO policy. One rationale behind this sequencing was that people may be more likely to spend money on a new product if they can first try it and witness its benefits firsthand. However, some could argue that it is essential to introduce cost-sharing from the outset, because after becoming accustomed to free treatment, people will develop a sense of entitlement and will refuse to pay when positive prices are later introduced. Although we are unable to directly test either hypothesis here, given the study design, it is worth noting that there was no significant difference in the impact of cost-sharing on take-up across Group 1 and Group 2 schools, despite their differing lengths of exposure to free treatment (three versus two years, respectively—regression not shown), exposure that could theoretically have provided a stronger sense of entitlement among Group 1 households.

The huge drop in take-up with cost-sharing and the extremely low level of private deworming purchases both suggest that most households in the study area place little value on deworming drugs. Even if deworming is socially beneficial, perceived private gains were smaller than private costs for most households under the cost-sharing regime. The social learning results indicate that additional information about deworming through social contacts only reinforces this view, further depressing adoption.

VI. The Impact of Health Education

There were no significant differences across treatment and comparison school pupils in early 1999 (one year into the program) on the three worm prevention behaviors that the program emphasized: pupil cleanliness (of the hands and uniform) observed by enumerators[28], the proportion of pupils observed wearing shoes, or self-reported exposure to fresh water (Table VIII, Panel A). The results do not vary significantly by pupil age,

28. This also holds controlling for initial 1998 cleanliness or using differences-in-differences (regressions not shown).

TABLE VIII
PSDP HEALTH BEHAVIOR IMPACTS (1999)

	Group 1	Group 2	Group 1– Group 2 (s.e.)
Panel A: Health behaviors, all pupils (Grades 3–8)			
Clean (observed by field worker), 1999	0.59	0.60	−0.01 (0.02)
Wears shoes (observed by field worker), 1999	0.24	0.26	−0.02 (0.03)
Days contact with fresh water in past week (self-reported), 1999	2.4	2.2	0.2 (0.3)
Panel B: Health behaviors, girls ≥13 years old			
Clean (observed by field worker), 1999	0.75	0.77	−0.02 (0.02)
Wears shoes (observed by field worker), 1999	0.39	0.42	−0.03 (0.06)
Days contact with fresh water in past week (self-reported), 1999	2.3	2.2	0.0 (0.3)

	Overall cross-school externality effect for Group 2
Panel C: Health behaviors, all pupils (Grades 3-8)	
Clean (observed by field worker), 1999	0.09 (0.21)
Wears shoes (observed by field worker), 1999	−0.01 (0.08)
Days contact with fresh water in past week (self-reported), 1999	0.96 (0.67)

Notes: These results use the data from Miguel and Kremer (2004). These are averages of individual-level data for grade 3–8 pupils; disturbance terms are clustered within schools. Robust standard errors in parentheses. Significantly different than zero at 99 (***), 95 (**), and 90 (*) percent confidence.

The effects in Panel C are the result of a regression in which the dependent variable is the change in the health behavior between 1998 and 1999 (school average). The local density of Group 1 pupils within three kilometers (per 1000 pupils), Group 1 pupils within three to six kilometers (per 1000 pupils), total pupils within three kilometers (per 1000 pupils), and total pupils within three to six kilometers (per 1000 pupils) are the key explanatory variables. Grade indicators, school assistance controls (for other NGO programs), and the average school district mock exam score are additional explanatory variables (as in Miguel and Kremer [2004]).

gender, or grade (results not shown). As we found with cost-sharing for deworming drugs, individuals appear unwilling to take a costly private action—here, buying shoes for their children or adopting new hygiene practices—that help to combat worms in their local community.

One alternative explanation is that treatment school children neglected to adopt worm prevention practices precisely because they were also taking deworming drugs and, thus, (falsely) felt protected from reinfection. This does not seem to explain the lack of health education impacts, however, since there was no evidence of behavioral change even among older girls who did not receive the medical treatment (due to concerns about potential embryotoxicity (Table VIII, Panel B)). The lack of basic knowledge about worm infections in this area makes remote the possibility that older girls in treatment schools neglected to adopt better worm prevention practices because they realized that they were benefiting from spillovers.

Moreover, there is no evidence that other children benefiting from treatment spillovers changed their prevention behavior: children attending comparison (Group 2) primary schools located near deworming treatment schools in early 1999 showed large reductions in worm infection levels [Miguel and Kremer 2004], but they did not receive health education, and there was no significant change in their worm prevention behaviors either (Table VIII, Panel C), although one limitation of this analysis is that these cross-school effects are very imprecisely estimated.

Although we cannot directly measure the depreciation of knowledge, other researchers find that depreciation of health education knowledge and practices is often rapid even in settings where direct short-run program impacts were positive (Aziz et al. [1990], Haggerty et al. [1994], Hoque et al. [1996]).

VII. The Impact of Commitment

Advocates of the sustainability approach in development argue that projects should only be implemented if there is local "ownership," often conveyed by beneficiaries making an affirmative commitment to the project. In the project we study, for instance, treatment took place only after the community collectively decided to participate during a village meeting.

The notion of ownership also relates to the claim in social psychology that asking individuals whether they plan to take an action will make it more likely that they go through with it.

A number of studies suggest that individuals can be motivated to take socially beneficial, but individually costly, actions by being asked whether they intend to perform them. Most people answer that they do, and many then feel motivated to follow through with their commitment. For example, Cioffi and Garner [1998] find large impacts of such commitments on blood donation on a U. S. university campus. (Greenwald et al. [1987] find such effects for voting behavior among university students in the United States, but in recent work Smith, Gerber, and Orlich [2003] fail to reproduce this finding using a much larger and more representative sample of U. S. voters.)

In an application of this technique, a random subsample of pupils in PSDP schools were asked whether they would take deworming drugs in the upcoming treatment round, in an attempt to boost drug take-up without providing additional external subsidies. During pupil questionnaire administration in 2001, a random subsample of pupils were asked whether they were planning to come to school on the treatment day and whether the PSDP workers should bring pills for them on that day: 98 percent of children answered "yes" to both questions. All pupils interviewed—including both those offered the opportunity for verbal commitment and those not offered this opportunity—were provided the same information on the effects of deworming and the upcoming date of medical treatment. (All respondents were, of course, also informed that participation in data collection and treatment were voluntary.)

The verbal commitment intervention failed, reducing drug take-up by one percentage point in 2001, although this effect is not statistically significant (Table IX, regression 1). This result is robust to controls for pupil age and gender (regression 2), and the impact of the intervention did not vary significantly with child characteristics (regression 3). The effect is somewhat more negative for pupils in cost-sharing schools and those with moderate–heavy worm infections, although in neither case are the estimates on these interactions significantly different than zero (results not shown).

These results underscore the need for further research clarifying when and where marketing techniques based on prior commitments have an impact.

TABLE IX
THE IMPACT OF A VERBAL COMMITMENT

	Dependent variable: child took deworming drugs in 2001		
	(1)	(2)	(3)
Verbal commitment intervention indicator	−0.014	−0.013	0.023
	(0.021)	(0.021)	(0.145)
Pupil age		−0.004	−0.003
		(0.006)	(0.006)
Pupil female		−0.048**	−0.050
		(0.024)	(0.035)
Verbal commitment intervention indicator *Age			−0.003
			(0.010)
Verbal commitment intervention indicator *Female			0.005
			(0.055)
Social links, other controls	Yes	Yes	Yes
Number of observations (pupils)	3,164	3,164	3,164
Mean of dependent variable	0.54	0.54	0.54

Notes: Data from 2001 parent and pupil surveys and administrative records. Marginal probit coefficient estimates are presented, robust standard errors in parentheses. Disturbance terms are clustered within schools. Significantly different from zero at 99 (***), 95 (**), and 90 (*) percent confidence. Social links controls are described in Miguel and Kremer (2003). Other controls include respondent years of education, community group member indicator variable, total number of children, iron roof at home indicator variable, and distance from home to school in kilometers, as well as the Group 2 and cost-sharing school indicators. Summary statistics from the 2001 pupil questionnaire (Mean [s.d.]): Pupil age (12.9 [2.3]), pupil female indicator (0.23 [0.42]) (older girls were dropped from the sample because they were not eligible for deworming, due to the potential embryotoxicity of the drugs).

VIII. CONCLUSION

A program that provided free deworming drugs for primary school students led to high drug take-up, large reductions in moderate–heavy worm infections, and increased school participation, all at low cost. Most of the deworming program benefit was in the form of externalities due to reduced disease transmission [Miguel and Kremer 2004]. Yet mass deworming treatment programs like the one we study are rare, and one in four people worldwide still suffer from these easily treated infections.

One reason for this failure is that rather than allocating funding on the basis of a standard public finance analysis, development agencies often prefer to fund "sustainable" interventions that do not require continued external funding. We examine several "sustainable" approaches to worm control in this paper, including cost-recovery from beneficiaries, health education, and individual mobilization and find all were ineffective at combating worms relative to the provision of free deworming drugs. The fact that drug take-up fell as more individuals were exposed to deworming through their social network is consistent with the idea that private valuation is low and casts doubt on the notion that a temporary intervention could lead to a sustainable long-run increase in deworming take-up through a process of social learning in this context. The analysis suggests people learned about the private benefits of deworming but provides no evidence for large pure imitation effects. Our model suggests that, in the absence of such effects, expending temporary subsidies beyond a small number of people will not affect long-run take-up.

Taken together, these findings suggest that continued subsidies may be needed to control diseases characterized by large positive treatment externalities, like worms. In Africa, where half the disease burden is associated with infectious and parasitic diseases [WHO 1999], this means extensive and indefinite health care subsidies may be needed to adequately address public health problems.

A broader lesson of this paper is that it may be difficult for external interventions to promote sustainable voluntary local public good provision. If local public goods are to be provided, they will likely have to be paid for by tax revenue collected either by local governments, national governments, or by external donors. Standard theories of fiscal federalism suggest local governments might be best suited to this task, but in Kenya as in many other developing countries, there are no locally elected bodies with taxation powers or control over revenue, perhaps because this could threaten central government primacy by creating rival power centers. National governments in Africa have not historically supplied deworming and have a poor record on local public goods provision. Donors have sometimes provided local public goods, but typically not on a long-term basis. Rather they often structure projects so as to be able to claim they are sustainable.

Donors may simply choose not to provide local public goods under these circumstances, or they may choose to provide them on an ongoing long-run basis, but there is little economic rationale for pursuing the illusion of sustainability. Even if donors wish to fund investment activities rather than consumption, there is little reason why they should seek projects that are sustainable on a project-by-project basis rather than taking a broader view of what constitutes a good investment. For instance, a public health project providing subsidized deworming may not be financially sustainable by itself in the short-run—in the sense that communities will not voluntarily provide it—but it will help children obtain more education, and this can contribute to long-run development for society as a whole. If donors are concerned that projects such as roads or wells will go awry without regular maintenance, they could endow funds earmarked for this purpose rather than counting on potentially illusory voluntary local contributions for maintenance.

Why then do aid agencies place so much emphasis on financial sustainability? We believe that rather than reflecting an economic social welfare calculation on behalf of optimizing donors, this reflects the politics of aid and principal-agent problems between aid agencies and their ultimate funders in wealthy countries, who are generally ill-informed about conditions in countries receiving aid. Aid agencies competing for limited donor funds have incentives to make bold claims about what their programs can achieve. In the short-run, these claims may be useful fundraising tools if the ultimate funders find it impossible to distinguish between, say, genuine claims regarding the temporary health benefits of providing free deworming medicine (as in the project we study) versus overstated claims about the permanent benefits of a one-time worm prevention health education intervention. Individual claims about spectacular project "bang for the buck" typically remain unchallenged since aid agencies are not directly accountable to their programs' beneficiaries through either political mechanisms (e.g., democratic elections) or through the market mechanism, and rigorous development program evaluations remain rare.

In the longer-term, of course, pursuing sustainability leads to failed projects, disillusionment among donors, and the search for the next development panacea. Rather than pursue the illusion of sustainability, development organizations and developing country governments would be better off rigorously evaluating their projects, ultimately identifying a limited number with high social returns, and funding these interventions on an ongoing basis.

Appendix

TABLE X

Robustness of Social Effect Results—Parent Networks

	Dependent variable: Child took deworming drugs in 2001			
	Probit (1)	Probit (2)	Probit (3)	Probit (4)
# parent links with children in early treatment schools (Group 1, 2, not own school)	−0.071*** (0.023)	−0.027* (0.014)	−0.029** (0.014)	−0.016 (0.014)
{# parent links with children in early treatment schools (Group 1, 2, not own school)}^{2}	0.0064** (0.0029)			
# pupils in early treatment schools <3 km from home (per 1000 pupils)				−0.20*** (0.07)
# Pupils in all schools <3 km from home (per 1000 pupils)				0.14** (0.07)
Parent social links controls	Yes	Yes	Yes	Yes
Other household controls	Yes	No	Yes	Yes
Ethnic, religious controls	No	No	Yes	No
Number of observations (parents)	1,678	1,678	1,678	1,678
Mean of dependent variable	0.61	0.61	0.61	0.61

Notes: Data from 2001 parent survey and 1999 and 2001 administrative records. Probit estimation, robust standard errors in parentheses. Disturbance terms are clustered within schools. Significantly different from zero at 99 (***), 95 (**), and 90 (*) percent confidence. Parent social links controls include total number of parent links, number of parent links to Group 1, 2, 3 schools (not own school), and number of parent links to non-program schools. Other household controls include respondent years of education, community group member indicator variable, total number of children, iron roof at home indicator variable, and distance from home to school in kilometers, as well as the Group 2 indicator and cost-sharing school indicator.

Ethnic controls include indicators for Luhya-Samia, Luhya-Nyala, Luo, Luhya-Khayo, Luhya-Marachi, and Teso groups and an indicator for being a member of the largest ethnic group in the school (which is near zero and statistically insignificant). Religion controls include indicators for Catholic, Anglican, Pentecostal, Apostolic, Legio Mario, Roho, and Muslim faiths and an indicator for being a member of the largest religious group in the school (which is negative and marginally statistically significant). In regression 2, no household controls are included as explanatory variables other than the standard social link controls from Table IV.

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Worms at Work: Long-run Impacts of Child Health Gains[*]

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We examine the impact of a child-health program on adult living standards by following participants in a deworming program in Kenya that began in 1998. The effective tracking rate was 83% over a decade. Treatment individuals received two to three more years of deworming than the comparison group. Self-reported health, years enrolled in school, and test scores improve significantly, hours worked increase by 12%, and work days lost to illness fall by a third in the treatment group. Treatment individuals report eating an average of 0.1 additional meals per day. Point estimates suggest substantial externalities among those living within 6 km of treatment schools, although significance levels vary due to large standard errors. Within the subsample working for wages, earnings are 21 to 29% higher for the treatment group. Most of the earnings gains are explained by sectoral shifts, including a doubling of manufacturing employment. Small business performance also improves among the self-employed. The results indicate that school based deworming is a very attractive public investment.

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1. Introduction

Many child public health measures – from immunization to water treatment, deworming and insecticide treated nets – have far from universal take-up in low-income countries and are not routinely provided for free by governments. There has been a lively debate between those who argue that governments should provide these goods for free, or even subsidize them, and those who argue that individuals should decide on their own whether to purchase these goods (Kremer and Miguel, 2007; Kremer and Holla, 2009; Ashraf, Berry, and Shapiro, 2010; Dupas 2011). A growing literature suggests that many people who will utilize these measures when they are free will not use them when they must pay. However, to understand whether public investments are worthwhile, it is also important to know the impact of these investments, both on the people who use the technologies and on others who may be affected by externalities from reduced transmission of infectious disease. After all, one view might be that low willingness to pay for these goods implies that people in poor countries have other priorities and that subsidies are not justified.

Advocates of public health spending in low-income countries often argue that, even setting aside the immediate utility benefits of improved health, such programs have high rates of return as investments because of their impact on adult living standards. Yet assessing the long-run causal impacts of public health measures has been problematic given the relative lack of both panel data sets tracking children into adulthood, and convincing causal identification from experimental variation.

We provide evidence from a prospective study on the impact of deworming of children in rural Kenyan primary schools on outcomes nearly a decade later, when most respondents were 19 to 26 years old. This analysis is based on a new longitudinal data set with an effective tracking rate of 83% among a representative subset of individuals enrolled in these schools. The combination of exogenous variation in child health investments with a long-term panel (longitudinal) dataset featuring high tracking rates, together with our ability to estimate spillover benefits of deworming treatment, sets this study apart from most of the existing literature.

Intestinal worm infections – including hookworm, whipworm, roundworm and schistosomiasis – are among the world's most widespread diseases, with roughly one in four people infected (Bundy 1994, de Silva *et al*. 2003). School age children have the highest infection prevalence of any group, and baseline infection rates in our Kenya study area are over 90%. Although light worm infections are often asymptomatic, more intense infections can lead to lethargy, anemia and growth stunting. Fortunately, worm infections can be treated infrequently (once to twice per year) with cheap and safe drugs. There is a growing body of evidence that school-based deworming in African settings can generate immediate improvements in child appetite, growth and physical fitness (Stephenson *et al.* 1993), and large reductions in anemia (Guyatt *et al.* 2001, Stoltzfus *et al.* 1997).

Treating worm infections also appears to strengthen children's immunological response to other infections, potentially producing broader health benefits in regions with high tropical disease burdens. For instance, a recent double-blind placebo controlled randomized trial among Nigerian preschool children finds that children who received deworming treatment for 14 months showed reduced infection prevalence with *Plasmodium*, the malaria parasite (Kirwan *et al.* 2010), and other authors have hypothesized that deworming might even provide some protection against HIV infection (e.g., see Fincham *et al.* 2003, Hotez and Ferris 2006, Watson and John-Stewart 2007). Chronic parasitic infections in childhood are known to generate inflammatory (immune defense) responses and elevated cortisol levels that lead substantial energy to be diverted from growth, and there is mounting evidence that this can produce adverse health consequences throughout the life course, including atherosclerosis, impaired intestinal transport of nutrients, organ damage, and cardiovascular disease (Crimmins and Finch 2005).

Due to the experimental design, deworming treatment group individuals in our sample received two to three more years of deworming than the control group. Previous work in this sample shows that deworming treatment led to large medium-run gains in school attendance and health outcomes, and, due to worms' infectious nature, that sizeable externality benefits accrued to the untreated within treatment communities and to those living near treatment schools (Miguel and Kremer 2004), as well as to the younger siblings of the treated (Ozier 2010).

In this paper, we first present a simple model (building on Bleakley 2010) to illustrate the conditions under which child health gains might affect educational investments and later income. We next find empirically that self-reported health improved, years enrolled in school increased by approximately 0.3 years, and some test scores rose in the treatment group. Although we cannot decompose how much of our labor market impacts are working through health versus education without imposing strong assumptions, these patterns suggest that both channels are playing a role.

We next generate unbiased estimates of the average impact of deworming on long-run outcomes by comparing the program treatment and control groups during 2007 to 2009. Treatment individuals report eating 0.1 more meals per day, consistent with higher living standards. Hours worked increase by 12% and work days lost to illness fall by a third. Point estimates suggest substantial externalities among those living within 6 km of treatment schools.

Among the subsample with wage employment, we find that earnings are 21 to 29% higher in the deworming treatment group. These labor market gains are accompanied by marked shifts in employment sector for the treatment group, with more than a doubling of well-paid manufacturing jobs (especially among males) and declines in both casual labor and domestic services employment. Changes in the subsector of employment account for nearly all of the earnings gains in deworming treatment group in a Oaxaca-style decomposition. This pattern indicates that health investments not only boost productivity and work capacity in existing activities, but, by leading individuals to shift into more lucrative economic activities (like manufacturing employment), may also contribute to the structural transformation of the economy a whole. Understanding how to promote this transition has long been a central theme within development economics (see Lewis 1954, among many others), and our results provide a piece of suggestive evidence that health investments may speed this transition.

Measuring labor productivity is more challenging for the majority of our subjects who were either self-employed or working in subsistence agriculture, rather than working for wages, although even in these groups there is some evidence of positive impacts. The estimated impacts on the small business performance of the self-employed, namely measures of profits and employees hired, are also positive and relatively large.

alone justify fully subsidizing school-based deworming.

Our findings contribute to several strands of existing work. The most closely related studies are by Bleakley (2007a, 2007b, 2010), who examines the impact of a large-scale deworming campaign in the U.S. South during the early 20th century on schooling and adult earnings, by comparing heavily infected versus lightly infected regions over time in a difference-in-difference design. He finds that deworming raised adult income by roughly 17%, and, extrapolating these findings to the even higher worm infection rates found in tropical Africa, estimates that deworming in Africa could lead to income gains of 24%, similar to our estimated earnings gains. Taken together, these findings lend credence to the view that treating intestinal worm infections can substantially increase labor productivity.[1] As Bleakley (2010) notes, the fact that deworming reduces morbidity but has negligible effects on mortality means it is particularly likely to boost per capita living standards.

Beyond deworming, our findings contribute to the growing literature on the long-run economic impacts of early life health and nutrition shocks. The well-known INCAP experiment in Guatemala described in Hodinott *et al.* (2008), Maluccio *et al.* (2009), and Behrman *et al.* (2009) provided nutritional supplementation to two villages while two others served as a control, and finds gains in

[1] There has been a lively debate in public health and nutrition about the cost-effectiveness of deworming (see Taylor-Robinson *et al.* 2007). Early work by Schapiro (1919) using a first-difference research design found wage gains of 15-27% on Costa Rican plantations after workers received deworming. Weisbrod et al (1973) document relatively weak cross-sectional correlations between worm infections and labor productivity, test scores, and fertility in St. Lucia. Bundy *et al*. (2009) argue that many existing studies understate deworming’s benefits since they fail to consider externalities (thus understating true treatment gains) by using designs that randomize within schools; focus almost exclusively on biomedical criteria and ignore cognitive, education and income gains that are key components of overall benefits; and do not deal adequately with attrition. The current paper attempts to address these three concerns. Beyond Miguel and Kremer (2004) and the current paper, Alderman *et al.* (2006b) and Alderman (2007) also use a cluster randomized controlled design and find large positive child weight gains in Uganda.

male wages of one third, improved cognitive skills among both men and women, and positive intergenerational effects on the nutrition of beneficiaries' children. Beyond the small sample size of four villages, a limitation of the INCAP studies is their relatively high attrition rate over the approximately 35 years of follow-up surveys, at roughly 40%.[2] While many studies argue that early childhood health gains *in utero* or before age three have the largest impacts (World Bank 2006, Hodinott *et al.* 2008, Almond and Currie 2010 are but a few examples), our findings show that even health investments made in school aged children can have important effects.

The rest of the paper is organized as follows. Section 2 presents a simple model of health, educational investments and income. Section 3 contains background on the school deworming project and the follow-up survey. Section 4 lays out the estimation strategy and describes the impacts of deworming on health, education, and labor market outcomes. The final section concludes, discussing external validity and implications for research and policy.

2. Understanding the impact of health gains on educational investments and lifetime income

We present the comparative statics of a simple textbook model of health, educational investment and income to illustrate the channels through which deworming may affect labor market outcomes. While many existing studies focus on educational attainment as the most likely channel linking child health

[2] A series of other influential studies have shown large long-run economic impacts of *in utero* or child health and nutrition shocks resulting from natural experiments, including the worldwide influenza epidemic of 1918 (Almond 2006), war-induced famine in Zimbabwe (Alderman *et al.*, 2006a), and economic shocks driven by rainfall variation in Indonesia (Maccini and Yang, 2009). Other studies that attempt to address the issue of long-run impacts of child health are those that deal with low birthweight (Sorenson *et al.*, 1997; Conley and Bennett, 2000); iodine deficiency *in utero* (Xue-Yi *et al.*, 1994; Pharoah and Connolly, 1991; Field *et al.*, 2007) and in early childhood (Fernald and Grantham-McGregor, 1998); whether children were breastfed (Reynolds, 2001); early childhoold malaria prophylazis, and early childhood under nutrition (Alderman *et al.*, 2003; Mendez and Adair, 1999; Glewee *et al.*, 2001), among many others. Though these studies are generally non-experimental (Jukes *et al.*, 2006 is an exception), taken together they provide considerable evidence that adult cognitive performance may be affected by nutrition in the womb and early childhood. Related work on the long-run benefits of child health and nutrition investments in the U.S. include Currie and Thomas (1995), Currie, Garces and Thomas (2002), and Case and Paxson (2010). Other noteworthy micro-empirical contributions on nutrition, health and productivity include Schultz (2005), Alderman (2007), Thomas *et al.* (2008), and Pitt, Rosenzweig and Hassan (2011), and recent contributions in macroeconomics on health and economic growth include Acemoglu and Johnson (2007), Ashraf, Lester and Weil (2009), and Aghion, Howitt and Murtin (2010).

gains to higher adult earnings, Bleakley (2010) rightly points out that standard models do not necessarily imply that education is the key mechanism. Here we present a simple model related to Bleakley's to illustrate this and other points.

We consider a model in which individuals choose how much education (denoted e below) to obtain to maximize discounted lifetime earnings, y, and examine how these schooling investments change as a function of child health (denoted h). The discounted future income benefits to schooling are b(e,h), and the costs (including both direct tuition costs and the opportunity cost of time spent in school rather than working) are c(e,h). Both the benefits and costs are increasing in education and health (b_e, b_h, c_e and c_h are all positive), but the marginal benefit of schooling declines with more education ($b_{ee} < 0$) while costs are convex ($c_{ee} > 0$). Both benefits and costs increase mechanically with health status if "non-sick" time increases, thus expanding the effective time budget. An individual's optimal educational investment level e^* is determined by the first order condition $y_e(e^*,h) = 0$, and equates marginal benefits to marginal costs, $b_e(e^*,h) = c_e(e^*,h)$.

The first relevant question for our analysis is how optimal educational investment levels change as child health improves. It is straightforward to show that:

(eqn. 1) $$\frac{de^*}{dh} = -\frac{b_{eh} - c_{eh}}{b_{ee} - c_{ee}}$$

By the usual assumptions above, the denominator is negative, but the numerator is more difficult to sign. Both derivatives are likely to be positive, in other words, improved child health boosts the marginal benefit of both school learning ($b_{eh} > 0$) and the opportunity cost of time (as labor productivity improves, $c_{eh} > 0$), but *a priori* there is no obvious sign on the difference. To the extent that the additional marginal benefits and costs are similar, there will be little change in schooling attainment, and it is even possible for schooling to fall after a positive health shock if the gains in current labor productivity outweigh the future gains from schooling. To the extent that the foregone earnings accruing to better health rise with age – i.e., good health is more relevant to the labor market success of an 18 year old than an 8 year old, whose current labor productivity is probably near zero regardless of his health status – we would expect optimal educational investments to respond most positively to improved health at younger ages.

We next derive the change in discounted lifetime income with respect to improved child health. There are two main channels, the direct labor benefits of better health (the first right-hand side term in eqn. 2) and effects through education (the second term):

(eqn. 2) $$\frac{dy^*}{dh} = \left.\frac{\partial y}{\partial h}\right|_{e*} + \left.\frac{\partial y}{\partial e}\right|_{e*} \times \frac{de^*}{dh}$$

In an application of the envelope theorem, the change in lifetime income with respect to educational investment at optimal investment is zero, implying that the second term is zero. To the extent that individuals are making optimal educational investment choices, then, schooling gains will not be able to account for later income gains, and we certainly cannot use an exogenous change in health as an instrumental variable to identify the returns to schooling. Rather, it is the direct effects of health on adult productivity (for instance, if healthier people are stronger or have more stamina), and on other dimensions of human capital (for instance, more learning per unit of time spent in school, as captured by the test score, say, rather than school attainment alone), that drives any later income gains.

However, there are some conditions under which increased educational investment generated by child health gains might be a key channel, for instance, when educational investment choices are not initially optimal in the sense described above. While there are many reasons why $e \neq e^*$ is possible, a leading explanation is that child disease morbidity constrains educational investment below the optimal level. This is plausible in a setting like ours with high levels of baseline intestinal worm infection levels. Imagine a case in which children are simply too sick to attend school once every s days, and thus school attendance is 1/s lower than children would choose in the absence of poor health. If a health intervention like deworming reduced sickness-induced school absenteeism from 1/s to 1/s′, where s′ > s, it would allow children to get closer to their ideal educational investment level, yielding first-order welfare gains.[3] Miguel and Kremer (2004) found large school attendance gains among deworming treatment pupils, especially among younger children.

In assessing the welfare impacts of increased adult earnings, a further application of the envelope theorem would imply that these are best captured in wage (productivity) gains rather than in increased hours worked. However, this only holds if individuals with poor health are already at or near their optimal labor supply. To the extent that they are not, and better health improves the capacity to work longer hours, then the total gain in earnings (rather than just gains generated by higher wages per hour worked) is a more appropriate welfare metric; we return to this issue below in our discussion of the returns to deworming investment.[4] The seminal model of health capital developed in Grossman (1972) argues that the fundamental difference between health capital and other forms of human capital, such as those created through education, is precisely the fact that better health status increases "the total amount of time [one] can spend producing money earnings and commodities" (p. 224). It is worth noting that the increases in adult hours worked and reduction in work days lost due to sickness among deworming treatment individuals that we report below are consistent with the view that healthier adults have greater work capacity and are thus better able to attain their ideal labor supply, leading to first-order welfare gains.

[3] Bleakley (2010) makes a similar observation about child school attendance gains. In the framework laid out above, this attendance effect is consistent with either the health investment allowing children to avoid some sickness-induced absenteeism, or with deworming shifting the marginal benefits of education more than the marginal costs ($b_{eh} > c_{eh}$). An alternative explanation for suboptimal educational investment could be agency problems or imperfect altruism within the household that leads parents to place too little weight on future child labor market gains from education. Note that in such a setting, improving child health (and labor productivity) today might instead boost current school drop-out rates.

[4] The relevant expression is $\frac{du^*}{dh} = \left.\frac{\partial u}{\partial h}\right|_{L^*} + \left.\frac{\partial u}{\partial L}\right|_{L^*} \times \frac{dL^*}{dh}$,

where L denotes hours worked and u is individual utility, in the context of a model where individuals face a labor-leisure trade-off. If individuals are initially working the optimal number of hours (L^*) then the second right-hand side term equals zero, implying that increased hours worked should not be considered in assessing the welfare gains from better health, but this does not hold if poor health constrains labor supply below L^*.

3. Background on the Primary School Deworming Program and Kenya Life Panel Survey

This section describes the study site, the deworming experiment, and follow-up survey, including our respondent tracking approach. We then present sample summary statistics.

3.1 The Primary School Deworming Program (PSDP)

In 1998, the non-governmental organization ICS launched the Primary School Deworming Program (PSDP) to provide deworming medication to individuals enrolled in 75 primary schools in Busia District, a densely-settled farming region of rural western Kenya adjacent to Lake Victoria. The schools participating in the program consisted of 75 of the 89 primary schools in Budalangi and Funyula divisions in southern Busia (with 14 town schools, all-girls schools, geographically remote schools, and program pilot schools excluded), and contained 32,565 pupils at baseline. Parasitological surveys conducted by the Kenyan Ministry of Health indicated that these divisions had high baseline helminth infection rates at over 90%. Using modified WHO infection thresholds (described in Brooker *et al.* 2000a), over one third of children in the sample had "moderate to heavy" infections with at least one helminth at the time of the baseline survey, a high but not atypical rate in African settings (Brooker *et al.* 2000b, Pullan et al. 2011). The 1998 Kenya Demographic and Health Survey indicates that 85% of 8 to 18 year olds in western Kenya were enrolled in school, indicating that our school-based sample is broadly representative of western Kenyan children as a whole.

Busia is close to the Kenyan national mean along a variety of economic and social measures. The 2005 Kenya Integrated Household Budget Survey shows that 96% of children aged 6 to 17 in Busia had "ever attended" school compared to 93% nationally, the gross enrollment rate was 119 compared to 117 nationally, while 75% of Busia adults were literate versus 80% nationally. However, Busia is poorer than average: 62% of Busia households fall below the poverty line compared to 41% nationally. Given that Kenyan per capita income is somewhat above the sub-

Saharan African average (if South Africa is excluded), the fact that Busia is slightly poorer than the Kenyan average probably makes the district more representative of rural Africa as a whole.

The 75 schools involved in this program were experimentally divided into three groups (Groups 1, 2, and 3) of 25 schools each: the schools were first stratified by administrative sub-unit (zone), listed alphabetically by zone, and were then listed in order of enrollment within each zone, and every third school was assigned to a given program group; Supplementary Appendix A contains a detailed description of the experimental design. The groups are well-balanced along baseline demographic and educational characteristics, both in terms of mean differences and distributions, where we assess the latter with the Kolmogorov-Smirnov test of the equality of distributions (Table 1).[5] The same balance is also evident among the subsample of respondents currently working for wages (see Supplementary Appendix Table A1).

Due to the NGO's administrative and financial constraints, the schools were phased into the deworming program over the course of 1998-2001 one group at a time. This prospective and staggered phase-in is central to this paper's econometric identification strategy. Group 1 schools began receiving free deworming treatment in 1998, Group 2 schools in 1999, while Group 3 schools began receiving treatment in 2001; see Figure 1. The project design implies that in 1998, Group 1 schools were treatment schools while Group 2 and 3 schools were the comparison schools, and in 1999 and 2000, Group 1 and 2 schools were the treatment schools and Group 3 schools were comparison schools, and so on. The NGO typically requires cost sharing, and in 2001, a randomly chosen half of the Group 1 and Group 2 schools took part in a cost-sharing program in which parents had to pay a small positive price to purchase the drugs, while the other half of Group 1 and 2 schools received free treatment (as did all Group 3 schools). Kremer and Miguel (2007) show that cost-sharing led to a sharp drop in deworming treatment, by 60 percentage points, introducing further

[5] Miguel and Kremer (2004) present a fuller set of baseline covariates for the treatment and control groups.

exogenous variation in deworming treatment that we can exploit. In 2002 and 2003, all sample schools received free treatment.

Children in Group 1 and 2 schools thus were assigned to receive 2.41 more years of deworming than Group 3 children on average (Table 1), and these early beneficiaries are what we call the deworming treatment group below. We focus on a single treatment indicator rather than separating out effects for Group 1 versus Group 2 schools since this simplifies the analysis, and because we find few statistically significant differences between Group 1 and 2, as discussed below. The fact that the Group 3 schools eventually did receive deworming treatment will tend to dampen any estimated treatment effects relative to the case where the control group was never phased-in to treatment. In other words, a program that consistently dewormed some children throughout childhood while others never received treatment might have even larger impacts. However, persistent differences between the treatment and control groups are plausible both because several cohorts "aged out" of primary school (i.e., graduated or dropped out) before treatment was phased-in to Group 3, and to the extent that more treatment simply yields greater benefits.

Deworming drugs for geohelminths (albendazole) were offered twice per year and for schistosomiasis (praziquantel) once per year in treatment schools.[6] We focus on intention-to-treat (ITT) estimates, as opposed to actual individual deworming treatments, in the analysis below. This is natural as compliance rates are high. To illustrate, 81.2% of grades 2-7 pupils scheduled to receive deworming treatment in 1998 actually received at least some treatment. Absence from school on the day of drug administration was the leading reported cause of non-compliance. The ITT approach is also attractive since previous research showed that untreated individuals within treatment

[6] Following World Health Organization recommendations (WHO 1992), schools with geohelmith prevalence over 50% were mass treated with albendazole every six months, and schools with schistosomiasis prevalence over 30% mass treated with praziquantel annually. All treatment schools met the geohelminth cut-off while roughly a quarter met the schistosomiasis cut-off. Medical treatment was delivered to the schools by Kenya Ministry of Health public health nurses and ICS public health officers. Following standard practices at the time, the medical protocol did not call for treating girls thirteen years of age and older due to concerns about the potential teratogenicity of the drugs.

communities experienced significant health and education gains (Miguel and Kremer 2004), complicating estimation of treatment effects on the treated. Miguel and Kremer (2004) show that deworming treatment improved self-reported health and reduced school absenteeism by one quarter during 1998-1999. Large externality benefits of treatment also accrued to individuals attending other schools within 6 kilometers of program treatment schools. There were no statistically significant academic test score or cognitive test score gains during 1998-2000.

3.2 Kenya Life Panel Survey (KLPS)

The first follow-up survey round of the PSDP sample, known as the Kenyan Life Panel Survey Round 1 (KLPS-1), was launched in 2003. Between 2003 and 2005, the KLPS-1 tracked a representative sample of approximately 7,500 individuals who had been enrolled in primary school grades 2-7 in the 75 PSDP schools at baseline in 1998. The second round of the Kenyan Life Panel Survey (KLPS-2) was collected during 2007-2009, and tracked this same sample of individuals. The KLPS-2 includes detailed questions on the employment and wage history of respondents (with questions based on Kenyan national surveys), as well as education, health, and other life outcomes.

A notable feature of the KLPS is its respondent tracking methodology. In addition to interviewing individuals still living in Busia District, survey enumerators traveled throughout Kenya and Uganda to interview those who had moved out of local areas; one respondent was even surveyed in London (in KLPS-1). Searching for individuals in rural East Africa is an onerous task, and migration of target respondents is particularly problematic in the absence of information such as forwarding addresses or home phone numbers, although the recent spread of mobile phones has been helpful. The difficulty in tracking respondents is especially salient for the KLPS, which follows young adults in their late teens and early twenties, when many are extremely mobile due to marriage, schooling, and job opportunities. Thus, it is essential to carefully examine survey attrition. If key explanatory variables, and most importantly deworming treatment assignment, were strongly related to attrition, then resulting estimates might suffer from bias.

The 7,500 individuals sampled for KLPS-2 were randomly divided in half, to be tracked in two separate waves. KLPS-2 Wave 1 tracking launched in Fall 2007 and ended in November 2008. During the first part of Wave 1, all sampled individuals were tracked.[7] In August 2008, a random subsample containing approximately one-quarter of the remaining unfound target respondents was drawn. Those sampled were tracked "intensively" (in terms of enumerator time and travel expenses) for the remaining months, while those not sampled were no longer actively tracked. We re-weight those chosen for the "intensive" sample by their added importance to maintain the representativeness of the sample. The same two phase tracking approach was employed in Wave 2 (launched in late 2008). As a result, all figures reported here are "effective" tracking rates (ETR), calculated as a fraction of those found, or not found but searched for during intensive tracking, with weights adjusted properly. The effective tracking rate (ETR) is a function of the regular phase tracking rate (RTR) and intensive phase tracking rate (ITR) as follows:

(eqn. 3) $$ETR = RTR + (1 - RTR)*ITR$$

This is closely related to the tracking approach employed in the Moving to Opportunity project (Kling *et al.* 2007, Orr *et al.* 2003).

Table 2, Panel A provides a summary of tracking rates in KLPS-2. Over 86% of respondents were located by the field team, with 82.5% surveyed while 3% were either deceased, refused to participate, or were found but were unable to be surveyed. These are very high tracking rates for any age group over a decade, and especially for a highly mobile group of adolescents and young adults, and they are on par with some of the best-known panel survey efforts in less developed countries,

[7] After 12 months of tracking, 64% of the Wave 1 sample (2,404 pupils) had been successfully surveyed, refused, or had died. Among the remaining 1,341 respondents, for budgetary reasons a representative one quarter were "intensively" tracked. As expected, individuals found during the intensive phase were more likely to be living outside of Busia, are somewhat older, and are also less likely to work in agriculture, see supplementary Appendix Table A2. Baird, Hamory and Miguel (2008) has a more detailed discussion of the KLPS tracking approach.

such as the Indonesia Family Life Survey (Thomas *et al*. 2001, 2010), and several recent African panel surveys.[8] Reassuringly, tracking rates are nearly identical in the treatment and control groups.

We also have information on where surveyed respondents were living (Table 2, Panel B); the locations of residence (for at least four consecutive months at any point during 1998-2009) are presented in the map in Appendix Figure A1. There is considerable migration out of Busia District, at nearly 30%, which once again is balanced between the treatment and control groups. Since the approximately 14% of individuals we did not find, and thus did not obtain residential information for, are plausibly even more likely to have moved out of the region, these figures almost certainly understate true out-migration rates. Nearly 8% of individuals had moved to neighboring districts, including just across the border into the Ugandan districts of Busia and Bugiri, while 22% of those with location information were living further afield, with most in Kenya's major cities of Nairobi, Mombasa or Kisumu. While there are some significant differences in the migration rates to Nairobi versus Mombasa across the treatment and control groups, they are relatively minor in magnitude.

We focus on the KLPS-2 data, rather than KLPS-1, in this paper since it was collected at a more relevant time point for us to assess adult life outcomes: the majority of sample respondents are adults by 2007-09 (with median age at 22 years as opposed to 18 in KLPS-1), have completed their schooling, many have married, and a growing share are engaging in wage employment or self-employment, as shown graphically in Appendix Figure A2. While the most common economic occupation is farming, as expected in rural Kenya, 16% worked for wages in the last month and 24% at some point since 2007, while 11% were currently self-employed outside of farming (Table 2, Panel C). The rates of wage work and self-employment are nearly identical across the deworming treatment and control groups, as discussed further below. This pattern simplifies the interpretation of some impacts estimated below, although they are somewhat surprising given the deworming impacts

[8] Other successful recent longitudinal data collection efforts among African youth are described in Beegle *et al.* (2010) and Lam et al (2008). Pitt, Rosenzweig and Hassan (2011) document high tracking rates in Bangladesh.

we estimate on other labor market dimensions, including the shifts across employment sectors among wage earners. The issue of selection into the wage earning subsample is discussed further below.

4. Deworming impacts on health, education and labor market outcomes

This section lays out the estimation strategy and describes deworming impacts on health, education and labor outcomes.

4.1 Estimation strategy

The econometric approach relies on the PSDP's prospective experimental design, namely, the fact that the program exogenously provided individuals in treatment (Group 1 and 2) schools two to three additional years of deworming treatment. We also adopt the approach in Miguel and Kremer (2004) and estimate the cross-school externality effects of deworming. Exposure to spillovers is captured by the number of pupils attending deworming treatment schools within 6 kilometers; conditional on the total number of primary school pupils within 6 kilometers, the number of treatment pupils is also determined by the experimental design, generating credible estimates of local spillover impacts.

In the analysis below, the dependent variable is a labor market outcome (such as wage earnings), $Y_{ij,2007\text{-}09}$, for individual i from school j, as measured in the 2007-09 KLPS-2 survey:

(eqn. 4) $$Y_{ij,2007\text{-}09} = a + bT_j + X_{ij,0}'c + d_1N_j^T + d_2N_j + e_{ij,2007\text{-}09}$$

The labor market outcome is a function of the assigned deworming program treatment status of the individual's primary school (T_j), and thus this is an intention to treat (ITT) estimator; a vector $X_{ij,0}$ of baseline individual and school controls; the number of treatment school pupils (N_j^T) and the total number of primary school pupils within 6 km of the school (N_j); and a disturbance term $e_{ij,2007\text{-}09}$, which is clustered at the school level.[9] The $X_{ij,0}$ controls include school geographic and demographic

[9] Miguel and Kremer (2004) separately estimate effects of the number of pupils between 0-3 km and 3-6 km. Since the analysis in the current paper does not generally find significant differences in externality impacts across these

characteristics used in the "list randomization", the student gender and grade characteristics used for stratification in drawing the KLPS sample, the pre-program average school test score to capture school academic quality, the 2001 cost-sharing school indicator, as well as controls for the month and wave of the interview.

The main coefficients of interest are b, which captures gains accruing to deworming treatment schools, and d_1, which captures any spillover effects of treatment for nearby schools. Bruhn and McKenzie (2009) argue for including variables used in the randomization procedure as controls in the analysis, which we do, although as shown below, the coefficient estimates on the treatment indicator are robust to whether or not the baseline individual and school characteristics are included as regression controls, as expected given the baseline balance across the treatment and control groups. Results are also robust to accounting for the cross-school spillovers. In fact, accounting for externalities tends to increase the b coefficient estimate; in other words, a failure to account for the program treatment "contamination" generated by spillovers dampens the "naïve" difference between treatment and control groups (and also potentially leads the researcher to miss a second dimension of program gains, the spillovers themselves). Certain specifications explore heterogeneity by interacting individual demographic characteristics with the deworming treatment indicator.

We also use an instrumental variables approach to generate a more structural estimate of the impact of eliminating intestinal worm infections per se. On the representative subsample of respondents administered parasitological stool sample exams during 1999, 2001 and 2002, we first estimate the first stage relationship by regressing an indicator for individual moderate-heavy worm infection on the deworming treatment school and externality variables (and other standard controls)

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two ranges, we focus on 0-6 km for simplicity. The externality results are unchanged if we focus on the proportion of local pupils who were in treatment schools as the key spillover measure (i.e., N_j^T / N_j, results not shown). Several additional econometric issues related to estimating externalities are discussed in Miguel and Kremer (2004).

in a specification similar to equation 4 above.[10] We present these first stage results in Table 3 below. This generates the predicted number of years with moderate-heavy worm infections between 1998-2001 at the individual-level, which serves as the endogenous variable in the IV specifications. We then use a two-sample IV approach with bootstrapped standard errors (Angrist and Pischke 2008) to generate the estimated impact of eliminating a moderate-heavy worm infection for one year.

The IV specification imposes the condition that the impact of different interventions that affect worm loads (e.g., free treatment, cross-school spillovers, and cost-sharing) is proportional to the reduction in moderate-heavy infection. This is restrictive if some gains are instead the result of reduced worm loads that are insufficient to meet the moderate-heavy threshold. The exclusion restriction may also not hold due to complementarities in schooling outcomes—if children are more inclined to go to school if their classmates are also in school, for instance. The IV estimates appear likely to overstate the effects of eliminating a worm infection for another reason. As Miguel and Kremer (2004) discuss, since worm infections were measured up to a year after treatment, when many pupils will already have been reinfected with worms, the difference in infection levels between treated and untreated pupils was likely much greater on average over the interval from deworming treatment to the parasitological exam than it was at the time of the parasitological exam (given the documented short-term efficacy of the drugs and rapid rate of reinfection). Thus the first stage probably understates the total number of moderate-heavy infections eliminated immediately after treatment, perhaps leading us to overstate labor market impacts per infection eliminated. While these factors suggest that one should be cautious about interpreting these results as a consequence of eliminating a moderate-heavy infection alone, the IV estimates may in fact represent the most accurate estimates of the impact of a general deworming program.

[10] Since the parasitological exams were collected very early in each calendar year, we follow Miguel and Kremer (2004) in assuming that the worm infection measures are relevant for understanding the previous year, i.e., that the early 1999 parasitological survey captures infection levels in 1998. For ethical reasons, parasitological surveys were only collected for groups that were to be treated in that year, so Group 1 schools have parasitological data for 1998-2002, Group 2 schools for 1999-2002, and Group 3 schools for 2001-2002.

4.2 Impacts on health and nutrition

We first document that deworming led to large reductions in moderate to heavy worm infections (defined as in Miguel and Kremer 2004) during the course of the original deworming intervention, using the parasitological stool sample data from 1999 and 2001 (Table 3, Panel A). As in the earlier study, there are large direct impacts of being assigned to a treatment school (-0.245, s.e., 0.030) as well as externality benefits for those living within 6 kilometers of treatment schools (-0.075, s.e., 0.026).[11] There is weak evidence of improved hemoglobin status (1.03, s.e. 0.81). In a 1999 survey conducted among a representative subsample of pupils, there is also a significant reduction in self-reported "falling sick often", by 3.7 percentage points (s.e. 1.5). The growing evidence that deworming improves immunological resistance to other infections, such as malaria (i.e., Kirwan *et al.* 2010), also implies that deworming might generate broader health benefits. We are able to assess the claim about malaria with the 1999 survey data, and find that self-reported malaria in the last week fell in the treatment group by 1.9 percentage points (s.e. 1.7), with an externality effect that is similar in magnitude. Although not statistically significant, this is a large reduction of nearly 10% given the self-reported malaria rate of 21.8 percentage points in the control group, providing weak suggestive evidence that deworming might have led to broader childhood health benefits in the treatment group.

Adult health also improved as a result of deworming: respondent self-reported health (on a normalized 0 to 1 scale) rose by 0.041 (s.e. 0.018, significant at 95% confidence, Table 3, panel B). Many studies have found that self-reported health reliably predicts actual morbidity and mortality even when other known health risk factors are accounted for (Idler and Benyamini 1997, Haddock *et al.* 2006, Brook *et al.* 1984). Note that it is somewhat difficult to interpret this impact causally since it may partially reflect health gains driven by the higher adult earnings detailed below, in addition to

[11] The time pattern of moderate-heavy worm infections across deworming treatment groups 1, 2 and 3 are presented graphically in Appendix Figure A3.

the direct health benefits of earlier deworming. Yet the fact that there were similar positive and statistically significant impacts on self-reported health in earlier periods, namely, in the 1999 survey before most were working, suggests that at least part of the effect is directly due to deworming.

In terms of other health outcomes, there is no evidence that deworming improved self-reported happiness or wellbeing or reduced major health shocks. Deworming did not lead to higher body mass index, nor are there detectable height gains, even when we restrict attention to younger individuals (those in grades 2-4 in 1998, regression not shown). Total health expenditures by the respondent in the last month are significantly higher in the treatment group (91.1 Shillings, s.e. 30.0). One possible interpretation is that people in the treatment group saw positive effects of biomedical treatment through the program, and that this experience led them to be more willing to invest in such treatments in the future. However, it is also possible that this reflects higher overall income levels or different health needs.

4.3 Impacts on education

We examine school enrollment and attendance using two different data sources in Table 4. We first report school participation, namely, being found present in school by survey enumerators on the day of an unannounced school attendance check. This is our most objective measure of actual time spent at school, and was a main outcome measure in Miguel and Kremer (2004), but two important limitations are that it was only collected during 1998-2001, and only at primary schools in the study area; the falling sample size between 1998 to 2001 (shown in appendix Table A3) is mainly driven by students graduating from primary school. Total school participation gains are 0.129 of a year of schooling (s.e. 0.064, significant at 95% confidence, Table 4, Panel A).

Another outcome variable is school enrollment as reported by the respondent in the KLPS-2 survey, which equals one if the individual was enrolled for at least part of a given year. These show consistently positive effects from 1999 to 2007 both on the deworming treatment indicator and the externalities term, and the total increase in school enrollment in treatment relative to control schools over the period is 0.279 years (s.e. 0.147, significant at 90% confidence). The treatment effect estimates are largest during 1999-2003 before tailing off during 2004-07 (Appendix Table A3), as predicted in the educational investment framework laid out above since the opportunity cost of time rises relative to the later benefits of schooling as individuals age. Given that the school enrollment data misses out on attendance impacts, which are sizeable, a plausible lower bound on the total increase in time spent in school induced by the deworming intervention is the 0.129 gain in school participation from 1998-2001 plus the school enrollment gains from 2002-2007 (multiplied by average attendance conditional on enrollment), which works out to nearly 0.3 years of schooling.

Despite the sizeable gains in years of school enrollment, there are no significant impacts on either total grades of schooling completed (0.153, s.e. 0.143) or attending at least some secondary school (0.032, s.e. 0.035), although both estimates are positive. A likely explanation is that the increased time in school is accompanied by increased grade repetition (0.060, s.e. 0.017, significant at 99% confidence). To summarize, deworming treatment individuals attended school more and were enrolled for more years on average, but do not attain significantly more grades in part because repetition rates rise substantially. Despite the absence of significant attainment effects, the increase in time spent in school may still yield some labor market returns through improved social or other non-cognitive skills (Heckman, Stixrud, and Urzua 2006).

Test score performance is another natural way to assess deworming impacts on human capital and skills. While the impact of deworming on primary school academic test score performance in 1999 is positive but not statistically significant (Table 4, Panel B), there is some evidence that the passing rate did improve on the key primary school graduation exam, the Kenya Certificate of Primary Education (point estimate 0.046, s.e. 0.031), and that English vocabulary knowledge (collected in 2007-09) is higher in the treatment group (impact of 0.076 standard deviations in a normalized distribution, s.e., 0.055). The mean effect size of the 1999 test score, the indicator for passing the primary school leaving exam, and the English vocabulary score in 2007-09 taken together yields a normalized point estimate of 0.112 that is significant at 90% confidence (s.e. 0.067), providing suggestive evidence of moderate human capital gains in the treatment group. As expected, there is no effect on the Raven's Matrices cognitive exam, which is designed to capture general intelligence rather than acquired skills. While many would argue that nutritional gains in the first few years of life could in fact generate improved cognitive functioning as captured in a Raven's exam – as Ozier (2010) indeed does find among younger siblings of these deworming beneficiaries – it was seemingly already "too late" for such gains among the primary school age children in our study.

It is difficult to disentangle the precise contributions of the education versus health gains we document in driving deworming's impact on labor market earnings, as the causal impacts on earnings of schooling attainment, other measures of skill (like our test of English vocabulary), self-reported health and our other measures are themselves not well-understood, and interactions among these channels are also possible. We are able to show in the cross-section, however, that the education and health factors we focus on are correlated with higher earnings among the control group. For instance, a Mincerian regression indicates that the return to a year of schooling is between 6 to 12 log points (and highly significant, not shown), and both academic test scores and self-reported health are also associated with higher earnings. At a minimum, these associations establish as plausible the claim that the health and education channels that we focus on might contribute to higher earnings.

4.4 Deworming Impacts on Living Standards and Labor Market Outcomes

Household consumption is commonly used to assess living standards in rural areas of less developed countries, where most households engage in subsistence agriculture rather than wage work. Our first measure, the number of meals consumed by the respondent yesterday, is narrower than total consumption but has the advantage that we collected it for the entire sample. Deworming treatment individuals consume 0.096 more meals (s.e. 0.028, significant at 99% confidence, Table 5, Panel A) than the control group, and the externality impact is also large and positive (0.080, s.e. 0.023, 99% confidence). This suggests that deworming led to higher living standards in the full sample.[12]

Turning to labor market outcomes, hours worked increase substantially in the deworming treatment group. Considering the full sample first, hours worked (in any occupation) increased by 1.76 hours (s.e. 0.97, Table 5, Panel B) on a control group mean of 15.3 hours, a 12% increase in the full sample that is significant at 90% confidence. The increase in hours worked is even more pronounced among the 66.2% of the sample that worked at all in the last week, at 2.40 hours (s.e. 1.16), on a base of 23.0 hours in the control group. Note that equal proportions of treatment and control group individuals worked at all in the last week, with a small and not significant difference of just 1.0 percentage points between the groups. Hours worked for wages or in-kind in particular increases substantially in the deworming treatment group by 5.2 hours (significant at 90% confidence), an increase of 12% on a base of 42.2 hours worked on average in the control group. There is also a large, positive and significant coefficient estimate on the term capturing local deworming treatment externalities, at 6.6 (s.e. 2.9). Some of these gains appears to be the direct result of improved health boosting individual work capacity among wage earners: days lost to poor health in the last month falls by a third, or 0.499 of a day (s.e. 0.235) in the treatment group. There are even larger increases in hours worked in self-employment in the last week, at 8.9 hours (s.e. 3.0) and again a large and statistically significant externality effect (8.0, s.e. 3.0). Impacts on hours worked in agriculture are small and not statistically significant.

[12] A consumption expenditure module was also collected as a pilot for roughly 5% of the KLPS-2 sample during 2007-09, for a total of 254 complete surveys. Such surveys are time-consuming and project budget constraints prevented us from collected a larger number of surveys. The data indicate that per capita average consumption levels in the control group are reasonable for rural Kenya, at US$580 (in exchange rate terms), and that food constitutes roughly 64% of total consumption. The estimated treatment effect for total consumption is near zero and not statistically significant (-$14, s.e. $66), though the confidence interval is large and includes substantial gains.

The distributions of hours worked (in all occupations), as represented in kernel densities, for the treatment and control groups are presented in Figure 2, panel A. There are few striking differences between these two distributions, both of which have considerable mass near zero. In the wage-earning subsample (panel B), though, a noticeably larger share of treatment individuals were working approximately full-time (roughly 40 hours per week) with fewer working part-time.

The distribution of wage earnings is also shifted sharply to the right in deworming treatment schools (Figure 3), another piece of evidence that deworming affected labor market outcomes.[13] In the regression analysis, we find that deworming treatment leads to higher earnings in: log transformations of earnings (Table 6, columns 1-4) and linear specifications (columns 5-8); with and without regression controls; and when cross-school externalities are accounted for. In the specification without the local externality controls (column 2), the estimated impact is 18.7 log points (s.e. 7.6, significant at 95% confidence), or roughly 21 percent. In our preferred specification with the full set of regression controls (column 3), the impact is 25.3 log points (standard error 9.3, 99% confidence), or approximately 29 percent, a large effect. The earnings gains are slightly smaller for Group 2 schools, as expected since they received one less year of deworming treatment, but the difference between Groups 1 and 2 (that together comprise the treatment group) is not significant (column 4), and there are similarly no statistically significant differences between Group 1 and 2 for a range of other labor market outcomes, including hours worked (not shown).

While the coefficient estimate on the local density of treatment pupils (in thousands) is not significant at traditional confidence levels (19.9 log points, s.e. 16.8, in column 3), it reassuringly has the same sign as the main deworming treatment effect, and a substantial magnitude: an increase of one standard deviation in the local density of treatment school pupils (917 pupils), which Miguel and Kremer (2004) found led to large drops in worm infection rates, would boost labor earnings by

[13] Here and below we present real earnings measures that account for the higher prices found in the urban areas of Nairobi and Mombasa. We collected our own comparable price surveys in both rural western Kenya and in urban Nairobi during the administration of the KLPS-2 surveys, and base the urban price deflator on these data.

roughly (917/1000)*(19.9 log points) = 18.2 log points, or 20 percent. We also include an indicator for inclusion in the randomly chosen group of 2001 cost-sharing schools in all specifications; recall that cost-sharing was associated with much lower deworming take-up in 2001. Consistent with this drop, the point estimate on the cost-sharing indicator in the regression shown in Table 6, column 3 is negative and marginally significant at -15.9 log points (s.e., 8.8). This provides further evidence that deworming treatment is associated with higher earnings.

The earnings result is almost unchanged to trimming the top 1% of earners, so the result is not driven by outliers (Table 7, Panel A). The earnings result is also robust to including a full set of gender-age fixed effects (estimate 0.270, s.e. 0.093, significant at 99%), to including fixed effects for each of the "triplets" of Group 1, Group 2 and Group 3 schools from the list randomization, and considering cross-school cost-sharing externalities (not shown).

The next set of results in Table 7 summarizes a wider set of labor market outcomes among wage earners, using our preferred specification with the full set of regression controls (equivalent to equation 4 and as in columns 3 and 6 in Table 6). Log wages (computed as earnings per hour worked) rise 16.5 log points in the deworming treatment group, and the effect is marginally significant (t-stat=1.4). Trimming the top 1% of wages leads to similar results (not shown). Positive wage earnings impacts are similar in the larger group of individuals, 24% of the sample, who have worked for wages at any point since 2007, where we use their most recent monthly earnings if they are not currently working for wages. The mean impact on log earnings is 0.211 (s.e. 0.072), and there is once again suggestive evidence of positive externality effects (Table 7, Panel B).

We find no significant evidence that deworming earnings gains differ by gender (Appendix Table A4, column 1), individual age at baseline (column 2) or the local level of serious worm infections at baseline (column 3). The relatively weak worm infection interaction effect may be due to use of the zonal-level infection rate, rather than individual-level data (which was not collected at baseline for the control group for ethical reasons); using zonal averages is likely to introduce measurement error and attenuation bias. There is marginally significant evidence that the gains in hours worked are larger among females (column 7), but it is notable that the gain in work hours is not larger among individuals who were initially younger at baseline (in grades 2-4, column 8). The gains in hours worked are no higher in areas with higher worm infection rates at baseline (column 9).

4.5 Selection into Wage Earning

The degree of selection into the wage earner subsample is a key issue in assessing the validity of the earnings results. For example, estimates could be biased downward if deworming led some individuals with relatively low labor productivity to enter the wage earner sample. While there is no single ideal solution, we present several pieces of evidence – including demonstrating that (i) there is no differential selection into wage earning subsamples, (ii) the observable characteristics of wage earners in the treatment and control groups are similar, (iii) there are significant impacts on certain labor market outcomes in the full sample, (iv) results are robust to a Heckman selection correction model, (v) and to restricting analysis to a subsample where labor market participation is substantially higher than average – all of which indicate that selection bias is unlikely to be driving these results.

Confirming the result in Table 2, we again find no evidence that deworming treatment individuals are more likely to be working for wages or in-kind in the last month (Table 7, Panel A, estimate -0.015, s.e. 0.018). There is similarly no differential selection into the subsample who have worked for wages at any point since 2007 by treatment group (Panel B, estimate 0.000, s.e. 0.021). While it remains possible that deworming led different types of individuals to enter wage earning while leaving the overall proportion unchanged, the lack of deworming impacts on the proportion of individuals working in both self-employed and agriculture as well makes this appear less likely.

We further confirm that there is no differential selection into the wage earner sample by gender (Appendix Table A4, column 4) or age (column 5). There is some evidence of greater selection into the wage earner subsample among deworming treatment individuals in zones with high worm infection rates at baseline (column 6), but the coefficient is only marginally significant and quite small. A one standard deviation increase in the baseline local moderate-heavy infection rate is 0.2, so an increase of this magnitude leads to a (0.2) x (0.028) = 0.0056 increase in the likelihood that individuals are wage earners, a small percent increase on the base of 0.166 in the control group. Baseline characteristics, including academic performance measures, are also indistinguishable across the treatment and control groups in the wage earner subsample (Appendix Table A1).

We focus on earnings in the full sample in Table 7, Panel C (before turning to more detailed analysis of the self-employed and agriculture subsamples below). While there is no effect on mean total labor earnings (setting non-wage earnings to zero for those without a job), total labor earnings are significantly higher in the treatment group at the 95th percentile in a quantile regression, and the same is true for other percentiles above the 90th (not shown).

The Heckman (1979) approach explicitly models the process of selection into wage earning. We use a marital status indicator and marital status interacted with gender as variables that predict selection into earning but are excluded from the earnings regression; marital status is strongly positively (negatively) correlated with any wage earning among males (females), results not shown. Keeping in mind the standard caveats to selection correction models, this approach yields an almost unchanged estimated impact of deworming on log wage earnings of 0.285 (s.e., 0.108, Table 7, Panel C), and similar impacts on the larger subsample that had earnings since 2007 (not shown).

An additional approach that partially addresses selection concerns restricts the analysis to males in our sample, who have a much higher rate of participation in wage employment since 2007, at 32%, than females (15%), and thus for whom selection bias is potentially less severe. The estimated treatment effect in this subsample among those currently working for wages is 0.217 (s.e. 0.117), and among those working since 2007 is 0.196 (s.e. 0.101), with both effects statistically significant at 90% confidence.

4.6 Impacts on employment sector

The increased earnings in the deworming treatment group can largely be accounted for by pronounced shifts in the sector of employment, out of relatively low-skilled and low wage sectors into better paid sectors. We present the share of control group individuals working in each of the major employment sectors in the first column of Table 8, where the sectors presented taken together account for over 90 percent of the entire wage earning subsample. The largest sectors are services, accounting for 41.7% of the wage earner subsample, with domestic work and food services being the largest subsectors; agriculture and fishing (21.0%); retail (at 15.3%); trade contractors (9.2%); casual labor or construction labor (2.9%); manufacturing (2.9% overall and 5.7% among males); and wholesale trade (2.7%). We then present the deworming treatment effect and the estimated externality impacts in the next two columns, respectively, and in the final two columns present average earnings and hours worked in this sector in the control group.

The most striking impacts are a large increase in manufacturing work for deworming treatment individuals, with a point estimate of 0.072 (s.e. 0.024, Table 8), signifying a tripling of manufacturing employment overall. The gains among males are particularly pronounced at 0.090 (s.e. 0.030). The two most common types of manufacturing jobs in our sample are in food processing and textiles, with establishments ranging in size from small local corn flour mills up to large blanket factories. On the flip side, casual labor employment falls significantly (-0.038, s.e. 0.018), as does domestic service work for females (-0.174, s.e. 0.110), although this latter effect is only marginally significant. Local deworming spillover effects have a consistent sign in all of these cases, and are significant for domestic employment among females (-0.435, s.e. 0.180). Not surprisingly given these shifts, a somewhat larger proportion of treatment group wage earners live in urban areas.

Manufacturing jobs tend to be quite highly paid, with average real monthly earnings of 5,311 Shillings (roughly US$68), compared to casual labor (2,246 Shillings) and domestic services (3,047 Shillings). Manufacturing jobs are also characterized by somewhat longer work weeks than average at 53 hours per week. A decomposition along the lines of Oaxaca (1973) indicates that over 90% of the increase in labor earnings for the treatment group, and nearly a third of the increase in hours worked, can be explained by the sectoral shifts documented in Table 8. While there are standard errors around these estimates and thus the exact figures should be taken with a grain of salt, they indicate that the bulk of the earnings gains can be accounted for by sectoral shifts.

4.7 Impacts on self-employment and agricultural outcomes

As with wage earning, there is no evidence of differential selection into self-employment or own agricultural work among deworming treatment individuals (Table 9, Panels A and B), simplifying the interpretation of the estimated impacts in these subsamples. Unfortunately, reliable measures of productivity are much harder to generate among the self-employed and those working on their own farms relative to wage work, making it more difficult to assess whether deworming had positive living standards impacts on these individuals. For instance, it is unclear how the self-employed are pricing their time (and the time of the family members and friends who assist them) when reporting their profits. Similarly, measuring the on-farm productivity of an individual worker in the context of a farm where multiple household members (and sometimes hired labor) are all contributing to different facets of the production process is notoriously difficult, and our survey instrument did not even attempt to disentangle individuals' separate contributions. As a result, we focus on a set of standard but imperfect proxies in this subsection.

Business outcomes improved considerably among the self-employed. The estimated deworming treatment effect on the profits of the self-employed (as directly reported in the survey) is positive (343 Shillings, s.e. 306, Panel A), although this 19% gain is not significant at traditional confidence levels, and there are similarly positive but not significant impacts on reported profits in the last year, on a profit measure based directly on revenues and expenses reported in the survey, as well as on the total number of employees hired (0.446, s.e. 0.361). The mean effect size of the three profit measures and the total employees hired taken together is positive, relatively large and statistically significant at 95% confidence at 0.175 (s.e., 0.089), where the magnitude is interpretable as 0.175 standard deviations of the normalized control group distribution, a sizeable effect.

Among those who work primarily on their own farm, there is no indication that deworming led to higher crop sales in the past year or adoption of “improved” agricultural practices including fertilizer, hybrid seeds or irrigation (Table 9, Panel B). The failure to find increased crop sales may, in part, be due to the fact that households are consuming more of the grain they produced, as suggested by the increase in meals eaten, a finding that also holds in the subset of agricultural households (not shown). While these results should be read with a grain of salt as we cannot easily measure individual on-farm productivity, there are no clear impacts on agricultural outcomes.

4.8 Instrumental Variable Estimates

We next go beyond intention to treat estimates and generate instrumental variable estimates of the impact of years of moderate-heavy worm infections on later outcomes. The first stage results are presented in Table 3 (panel A), and show that assignment to a treatment school, as well as geographic proximity to other treatment schools, both lead to significantly lower individual worm infection levels. The two-sample IV results are broadly similar to the ITT estimates in terms of statistical significance levels, although magnitudes and interpretation differ (Appendix Table A5). The estimates indicate that experiencing one fewer year with a moderate-heavy worm infection during childhood increases hours worked by 3.14 hours in the last week (s.e. 1.24) and earnings in the most recent month worked by 26.6 log points (s.e. 10.8). As mentioned above, in our view these estimates are likely to overstate the true impacts of eliminating a moderate-heavy worm infection for one year since the worm infection measures are taken with a considerable lag after treatment and thus understate the true number of infection eliminated due to rapid reinfection.

5. Conclusion

We exploit an unusually useful setting for estimating the impact of child health gains on adult earnings and other life outcomes. The Kenya Primary School Deworming Program was experimentally phased-in across 75 rural schools between 1998 and 2001 in a region with high rates of intestinal worm infections, one of the world's most widespread diseases. As a result, the treatment group exogenously received an average of two to three more years of deworming treatment than the control group. A representative subset of the sample was followed up for roughly a decade through 2007-09 in the Kenya Life Panel Survey, with high survey tracking rates, and the labor market outcomes of the treatment and control groups are compared to assess impacts.

There were large increases in average hours worked (by 12%), and a reduction in work days lost to sickness as a result of deworming. Among those working for wages, average adult earnings rise by approximately 21 to 29%, and these gains are accompanied by sharp shifts in employment towards high-paying manufacturing sector jobs (especially for males) and away from casual labor and domestic services employment (for females). The finding that shifts into different employment sectors account for the bulk of the earnings gains suggests that characteristics of the broader labor market – for instance, sufficient demand for manufacturing workers – may be critical for translating better health into higher living standards. These findings complement Bleakley's work on historical deworming programs in the U.S. South in the early 20th century, and the correspondence between the two sets of results – using distinct research designs and data – increases confidence in the external validity of both findings.

The main implication of this paper is that childhood health investments like school-based deworming can substantially boost adult earnings. It goes without saying that deworming alone, and its associated increase in earnings, cannot make more than a small dent in the large gap in living standards between poor African countries like Kenya and the world's rich countries. Yet that obvious point does not make deworming any less attractive as a public policy option given its extraordinarily high social rates of return, and the fact that boosting income by one quarter would have major welfare impacts for households living near subsistence.

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Table 1: Baseline (1998) summary statistics and PSDP randomization checks

	All mean (s.d.)	Treatment mean (s.d.)	Control mean (s.d.)	Treatment – Control (s.e.)	Kolmogorov-Smirnov p-value
Age (1998)	11.9	11.9	12.0	-0.04	0.258
	(2.6)	(2.6)	(2.6)	(0.11)	
Grade (1998)	4.23	4.22	4.25	-0.03	0.450
	(1.68)	(1.70)	(1.66)	(0.05)	
Female	0.470	0.469	0.473	-0.004	--
				(0.019)	
Assignment to the deworming treatment group	0.678	1	0	--	--
Years of assigned deworming treatment, 1998-2003	3.31	4.09	1.68	2.41***	--
	(1.82)	(1.52)	(1.23)	(0.08)	
Primary school located in Budalangi division	0.370	0.364	0.381	-0.017	--
				(0.137)	
Population of primary school	476	494	436	58	0.307
	(214)	(237)	(146)	(54)	
School average test score (1996)	0.029	0.024	0.038	-0.013	0.310
	(0.427)	(0.436)	(0.406)	(0.109)	
Total treatment (Group 1, 2) primary school students within 6 km	3,180	3,085	3,381	-296	0.206
	(917)	(845)	(1,022)	(260)	
Total primary school students within 6 km	4,709	4,698	4,732	-34	0.119
	(1,337)	(1,220)	(1,555)	(389)	

Notes: The data are from the PSDP, and includes all individuals surveyed in the KLPS2. There are 5,084 observations for all variables, except for Age (1998) where there are 5,072 observations due to missing survey data. All observations are weighted to maintain initial population proportions. All variables are 1998 values unless otherwise noted. Years of assigned deworming treatment is calculated using the treatment group of the respondent's school and their grade, but is not adjusted for the treatment ineligibility of females over age 13 or assignment to cost-sharing in 2001. Those individuals who "age out" of primary school are no longer considered assigned to deworming treatment. The average school test score is from the 1996 Busia District mock exam, and has been converted to units of normalized individual standard deviations. The "Treatment – Control" differences are derived from a linear regression of the outcome on a constant and the treatment indicator, but results are similar if we include further controls (for survey wave, 1998 administrative zone of residence, cost sharing school indicator, and baseline 1998 population of the individual's primary school). Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence. The Kolmogorov-Smirnov p-values are only presented for the non-binary variables, where it is informative.

Table 2: Attrition and residential location patterns, KLPS2 (2007-09)

	All mean (s.d.)	Treatment mean (s.d.)	Control mean (s.d.)	Treatment – Control (s.e.)
Panel A: Sample attrition, KLPS2 I-module				
Found[a]	0.862	0.860	0.867	-0.007
				(0.017)
Surveyed	0.825	0.824	0.827	-0.003
				(0.018)
Not surveyed, dead	0.017	0.018	0.014	0.004
				(0.004)
Not surveyed, refused	0.015	0.014	0.017	-0.003
				(0.005)
Panel B: Residential location information				
Have residential location information (2007-09)	0.824	0.823	0.826	-0.003
				(0.018)
Among those with residential location information:				
Residence in Busia district	0.705	0.708	0.700	0.007
				(0.022)
Residence in districts neighboring Busia district[b]	0.078	0.082	0.069	0.013
				(0.011)
Residence outside of Busia and neighboring districts	0.217	0.210	0.230	-0.020
				(0.020)
In Nairobi	0.102	0.093	0.120	-0.027*
				(0.014)
In Mombasa	0.037	0.043	0.024	0.019**
				(0.008)
In Kisumu	0.018	0.018	0.017	0.002
				(0.006)
Residence outside of Kenya	0.052	0.056	0.043	0.012
				(0.010)
Panel C: Employment patterns				
Worked for wages or in-kind in last month[c]	0.158	0.154	0.166	-0.013
				(0.016)
Self-employed in the last month[d]	0.107	0.110	0.100	0.010
				(0.013)
Worked in agriculture in the last week[e]	0.519	0.513	0.531	-0.018
				(0.023)

Notes: The sample used in Panel A and for the variable "have residential location information" includes all individuals surveyed, found deceased, refused participation, found but unable to survey, and not found but sought in intensive tracking during KLPS2, a total of 5,569 individuals (3,686 treatment and 1,883 control). The remainder of Panels B and C include all individuals surveyed in the KLPS2. All observations are weighted to maintain initial population proportions. The "Treatment – Control" differences are derived from a linear regression of the outcome on a treatment indicator, but results are similar if we include further controls (for survey wave, 1998 administrative zone of residence, cost sharing school indicator, and baseline 1998 population of the individual's primary school). Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence. The Kolmogorov-Smirnov p-values are not presented since all variables in this table are binary variables.

[a] The proportion "Found" is the combination of pupils surveyed, found deceased, refused and found but unable to survey. [b] Districts neighboring Busia include Siaya, Busia (Uganda), and other districts in Kenya's Western Province. [c] Employment includes only those who earned a positive salary or payment in kind. [d] Self-employment includes only those who earned positive profits, and excludes household farming activities. [e] Agriculture includes both farming and pastoralist activities.

Table 3: Impacts on health, wellbeing and nutritional outcomes

Dependent variable	Comparison group variable mean (s.d.)	Coefficient estimate (s.e.) on deworming treatment indicator	Coefficient estimate (s.e.) on deworming Treatment school pupils within 6 km (in '000s), demeaned
Panel A: Health outcomes during 1999-2001			
Moderate-heavy worm infection (1999, 2001 parasitological surveys)	0.321	-0.245***	-0.075***
	(0.467)	(0.030)	(0.026)
Hemoglobin (Hb) level (1999, 2001 parasitological survey samples)	126.1	1.03	0.91
	(14.7)	(0.81)	(0.96)
Falls sick often (self-reported), 1999	0.154	-0.037**	0.001
	(0.361)	(0.015)	(0.014)
Malaria in the last week (self-reported), 1999	0.218	-0.019	-0.018
	(0.413)	(0.017)	(0.018)
Panel B: Health, wellbeing and nutritional outcomes, KLPS-2 (2007-09)			
Self-reported health "very good"	0.673	0.041**	0.028
	(0.469)	(0.018)	(0.022)
Self-reported currently "very happy"	0.673	0.020	0.028
	(0.469)	(0.018)	(0.023)
Index of wellbeing (0 to 1)	0.831	0.018	-0.013
	(0.290)	(0.012)	(0.012)
Body mass index (BMI = Weight in kg / (height in m)^2)	27.2	0.024	0.064
	(1.3)	(0.044)	(0.053)
Height (cm)	167.3	-0.12	-0.39
	(8.0)	(0.26)	(0.33)
Respondent health expenditures (medicine, in/out-patient) in past month (KSh)	119.2	91.1***	40.7
	(389.9)	(30.0)	(55.9)

Notes: The sample size in Panel A is 2,720 for worm infection, 1,765 for Hb, and 3,861 for health self-reports. Representative subsets of pupils in all schools were surveyed for these 1999 and 2001 pupil surveys. The sample in Panel B includes all individuals surveyed in KLPS-2. Each row is from a separate OLS regression analogous to equation 4. All observations are weighted to maintain initial population proportions. Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence. All regressions include controls for baseline 1998 primary school population, geographic zone of the school, survey wave and month of interview, a female indicator variable, baseline 1998 school grade fixed effects, the average school test score on the 1996 Busia District mock exams, total primary school pupils within 6 km, and the cost-sharing school indicator. Self-perceived health "very good" takes on a value of one if the answer to the question "Would you describe your general health as somewhat good, very good, or not good?" is "very good", and zero otherwise. Self-reported currently "very happy" takes on a value of one if the answer to the question "Taking everything together, would you say you are somewhat happy, very happy or not happy?" is "very happy", and is zero otherwise. The underlying index of well being takes on a value of 0 to 4 where 4 implies answering no to all of the following four questions: "In the past week, have you felt tense, nervous or worried?" "In the past week have you generally not enjoyed your daily activities?" "In the past week have you felt more unhappy than usual?" "In the past week have you found it difficult to make decisions?" Indicator of no major health problem since 1998 takes on a value of one if they answer no to the following question "Have you experienced any major health problems that seriously affected your life or work, since 1998?".

Table 4: Impacts on schooling and test score outcomes

Dependent variable	Comparison group variable mean (s.d.)	Coefficient estimate (s.e.) on deworming treatment indicator	Coefficient estimate (s.e.) on deworming Treatment school pupils within 6 km (in '000s), demeaned
Panel A: School participation, enrollment and attainment			
Total primary school participation, 1998-2001	2.51	0.129***	0.056
	(1.12)	(0.064)	(0.048)
Total years enrolled in school, 1998-2007	6.69	0.279*	0.138
	(2.97)	(0.147)	(0.149)
Grades of schooling attained	8.72	0.153	0.070
	(2.21)	(0.143)	(0.146)
Indicator for repetition of at least one grade (1998-2007)	0.672	0.060***	0.010
	(0.470)	(0.017)	(0.023)
Attended some secondary school	0.421	0.032	0.000
	(0.494)	(0.035)	(0.039)
Panel B: Test scores			
Mean effect size (1999 test, passed primary school exam, 2007-09 English test)	0.000	0.112	0.068
	(1.000)	(0.067)*	(0.058)
Academic test score (normalized across all subjects), 1999	0.026	0.059	0.158
	(1.000)	(0.090)	(0.101)
Passed primary school leaving exam during 1998-2007	0.509	0.046	0.032
	(0.500)	(0.031)	(0.030)
English vocabulary test score (normalized), 2007-09	0.000	0.076	0.067
	(1.000)	(0.055)	(0.053)
Raven's Matrices cognitive test score (normalized), 2007-09	0.000	-0.011	0.055
	(1.000)	(0.048)	(0.042)

Notes: Each row is from a separate OLS regression analogous to equation 4 using the full KLPS-2 sample. All observations are weighted to maintain initial population proportions. Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence. All regressions include controls for baseline 1998 primary school population, geographic zone of the school, survey wave and month of interview, a female indicator variable, baseline 1998 school grade fixed effects, the average school test score on the 1996 Busia District mock exams, total primary school pupils within 6 km, and the cost-sharing school indicator.

Table 5: Deworming impacts on living standards and labor supply

Dependent variable	Control group variable mean (s.d.)	Coefficient estimate (s.e.) on deworming Treatment indicator	Coefficient estimate (s.e.) on deworming Treatment pupils within 6 km (in '000s), demeaned	Obs.
Panel A: Living standards				
Number of meals eaten yesterday	2.16	0.096***	0.080***	5,083
	(0.64)	(0.028)	(0.023)	
Panel B: Labor supply				
Hours worked (for wages, self-employed, agriculture) in last week	15.2	1.76*	1.54	5,084
	(21.9)	(0.97)	(1.16)	
Hours worked (for wages, self-employed, agriculture) in last week, among those with hours worked > 0	23.0	2.40**	2.75**	3,514
	(23.4)	(1.16)	(1.36)	
Hours worked (for wages) in the last week, among those with hours worked > 0	42.2	5.19*	6.60**	693
	(24.7)	(2.74)	(2.93)	
Days of work missed due to poor health (among those working for wages), past month (negative binomial)	1.46	-0.499**	-0.337	718
	(2.99)	(0.235)	(0.305)	
Hours worked (as self-employed) in last week, among those with hours worked > 0	33.9	8.9***	8.0***	583
	(25.7)	(3.0)	(3.0)	
Hours worked (in agriculture) in last week, among those with hours worked > 0	9.5	0.48	-0.75	2,829
	(9.1)	(0.53)	(0.48)	
Indicator for hours worked > 0 (for wages, self-employed, agriculture) in last week	0.662	0.010	-0.007	5,084
	(0.473)	(0.022)	(0.025)	

Notes: Each row is from a separate OLS regression analogous to equation 4, except the negative binominal. The household consumption expenditure per capita results trim the top 2% of households. All observations are weighted to maintain initial population proportions. Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence. All regressions include controls for baseline 1998 primary school population, geographic zone of the school, survey wave and month of interview**,** a female indicator variable, baseline 1998 school grade fixed effects, the average school test score on the 1996 Busia District mock exams, total primary school pupils within 6 km, and the cost-sharing school indicator.

Table 6: Deworming impacts on labor earnings (2007-2009)

	Dependent variable:							
	Ln(Total labor earnings, past month)				Total labor earnings, past month (in Kenya Shillings)			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Deworming Treatment indicator	0.193**	0.187**	0.253***	0.277***	611**	627**	780*	906*
	(0.077)	(0.076)	(0.093)	(0.104)	(285)	(306)	(417)	(504)
Deworming Treatment pupils within 6 km (in '000s), demeaned			0.199	0.194			451	424
			(0.168)	(0.170)			(740)	(740)
Total pupils within 6 km (in '000s), demeaned			-0.098	-0.094			-201	-179
			(0.127)	(0.129)			(575)	(571)
Group 2 school indicator				-0.060				-327
				(0.099)				(449)
Cost sharing school (in 2001) indicator	-0.104	-0.139	-0.159*	-0.154*	-390	-540	-584	-557
	(0.085)	(0.094)	(0.088)	(0.090)	(370)	(425)	(410)	(409)
Additional controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
R^2	0.064	0.176	0.182	0.183	0.060	0.125	0.126	0.127
Observations	710	710	710	710	710	710	710	710
Mean (s.d.) in the control group	7.86	7.86	7.86	7.86	3,739	3,739	3,739	3,739
	(0.88)	(0.88)	(0.88)	(0.88)	(3,744)	(3,744)	(3,744)	(3,744)

Notes: The sample used here includes all individuals surveyed in the KLPS2 who report positive labor earnings at the time of survey. Labor earnings include cash and in-kind, and are deflated to reflect price differences between rural and urban areas. All observations are weighted to maintain initial population proportions. All regressions include controls for baseline 1998 primary school population, geographic zone of the school, and survey wave and month of interview. Additional controls include a female indicator variable, baseline 1998 school grade fixed effects, and the average school test score on the 1996 Busia District mock exams. Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence.

Table 7: Deworming impacts on labor earnings and wages

Dependent variable	Control group variable mean (s.d.)	Coefficient estimate (s.e.) on deworming Treatment indicator	Coefficient estimate (s.e.) on deworming Treatment pupils within 6 km (in '000s), demeaned	Obs.
Panel A: Wage earner subsample				
Ln(Total labor earnings, past month)	7.86	0.253***	0.199	710
	(0.88)	(0.093)	(0.168)	
Ln(Total labor earnings, past month) – top 1% trimmed	7.83	0.269***	0.237	698
	(0.85)	(0.092)	(0.161)	
Ln(Total labor earnings, past month) – with all gender-age fixed effects	7.86	0.270***	0.197	710
	(0.88)	(0.093)	(0.159)	
Ln(Wage = Total labor earnings / hours, past month)	2.82	0.165	0.012	625
	(0.96)	(0.117)	(0.160)	
Indicator for worked for wages (or in-kind) in last month	0.166	-0.015	-0.002	5,081
	(0.372)	(0.018)	(0.020)	
Panel B: Wage earner since 2007 subsample				
Ln(Total labor earnings, most recent month worked)	7.88	0.211***	0.170	1,175
	(0.91)	(0.072)	(0.116)	
Indicator for worked for wages (or in-kind) since 2007	0.244	0.000	0.040	5,081
	(0.430)	(0.021)	(0.024)	
Panel C: Full sample				
Ln(Total labor earnings, past month) – Heckman selection correction	7.86	0.285***	0.148	5,082
	(0.88)	(0.108)	(0.170)	
Total labor earnings, past month, earnings=0 for non- earners	619	27	-17	5,084
	(2,060)	(81)	(97)	
Total labor earnings, past month – 95th percentile (quantile regression), earnings=0 for non-earners	619	290**	123	5,084
	(2,060)	(117)	(140)	

Notes: Each row is from a separate OLS regression analogous to equation 4, except the quantile regression in Panel C. Ln(Wage) adjusts for the different reporting periods for earnings (month) and hours (week), and is missing for those with zero earnings. All observations are weighted to maintain initial population proportions. Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence. All regressions include controls for baseline 1998 primary school population, geographic zone of the school, survey wave and month of interview**,** a female indicator variable, baseline 1998 school grade fixed effects, the average school test score on the 1996 Busia District mock exams, total primary school pupils within 6 km, and the cost-sharing school indicator.

Table 8: Deworming impacts on employment sector and occupation

Employment sector:	Control group proportion	Coefficient estimate (s.e.) on deworming treatment indicator	Coefficient estimate (s.e.) on deworming treatment pupils within 6 km (in '000s), demeaned	Mean (s.d.) earnings in sector, past month (Kenya Shillings), control	Mean (s.d.) hours per week worked in sector, control group
Agriculture and fishing	0.210	-0.038	-0.152*	2,872	35
		(0.059)	(0.080)	(1,804)	(25)
Retail	0.153	-0.018	0.025	2,049	39
		(0.038)	(0.043)	(1,713)	(29)
Trade contractors	0.092	-0.005	0.060	3,172	27
		(0.028)	(0.004)	(2,170)	(22)
Manufacturing	0.029	0.072***	0.041	5,311	53
		(0.024)	(0.031)	(3,373)	(24)
Manufacturing – males only	0.057	0.090***	0.031	6,277	49
		(0.033)	(0.033)	(3,469)	(20)
Wholesale trade	0.027	0.023	0.022	4,727	44
		(0.029)	(0.035)	(3,963)	(14)
Services (all)	0.417	0.032	0.037	4,694	47
		(0.054)	(0.075)	(5,013)	(24)
Domestic	0.115	-0.012	-0.026	3,047	61
		(0.032)	(0.038)	(1,754)	(18)
Domestic – females only	0.335	-0.174	-0.435***	2,795	65
		(0.110)	(0.180)	(888)	(17)
Restaurants, cafes, etc.	0.060	-0.029	0.024	4,194	53
		(0.023)	(0.034)	(3,567)	(21)
Casual/Construction laborer	0.029	-0.038**	-0.020	2,246	51
		(0.018)	(0.017)	(1,576)	(31)
Other	0.030	-0.028*	-0.013	4,600	47
		(0.015)	(0.014)	(1,740)	(13)

Notes: The sample used here includes all individuals surveyed in the KLPS2 who report working for pay (with earnings greater than zero) at the time of the survey. Each row is from a separate OLS regression analogous to equation 4. All observations are weighted to maintain initial population proportions. Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence. All regressions include controls for baseline 1998 primary school population, geographic zone of the school, survey wave and month of interview**,** a female indicator variable, baseline 1998 school grade fixed effects, the average school test score on the 1996 Busia District mock exams, total primary school pupils within 6 km, and the cost-sharing school indicator.

Table 9: Deworming impacts on other economic outcomes

Dependent variable	Control group variable mean (s.d.)	Coefficient estimate (s.e.) on deworming Treatment indicator	Coefficient estimate (s.e.) on deworming Treatment pupils within 6 km (in '000s), demeaned	Obs.
Panel A: Self-employed profits, hours and employees				
Mean effect size (three profits measures, and total employees hired)	0.000	0.175**	0.014	555
	(1.000)	(0.089)	(0.097)	
Total self-employed profits (self-reported) past month (among those >0)	1,766	343	-151	585
	(2,619)	(306)	(320)	
Total self-employed profits (constructed) past month (among those >0)	1,535	1,211	2,088	595
	(6,524)	(1,091)	(1,886)	
Total self-employed profits (self-reported) past year (among those >0)	12,193	1,952	-1,753	566
	(17,346)	(2,286)	(2,590)	
Total employees hired (excluding self), among the self-employed	0.188	0.446	0.044	633
	(0.624)	(0.361)	(0.492)	
Indicator for self-employed earnings in last month	0.100	0.015	0.004	5,083
	(0.300)	(0.012)	(0.011)	
Panel B: Agricultural work, sales, hours and practices				
Total value (KSh) of crop sales past year (if farm household)	576	-81	-460**	3,758
	(2458)	(148)	(206)	
Uses "improved" agricultural practice (if farming household)	0.310	0.032	0.005	3,766
	(0.462)	(0.026)	(0.024)	
Indicator for respondent did agricultural work in last week	0.531	-0.010	0.005	5,080
	(0.499)	(0.025)	(0.031)	

Notes: Each row is from a separate OLS regression analogous to equation 4. "Agricultural work" includes both farming and pastoral activities. The average of "typical monthly" and last week recall is used for household consumption. Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence. All regressions include controls for baseline 1998 primary school population, geographic zone of the school, survey wave and month of interview**,** a female indicator variable, baseline 1998 school grade fixed effects, the average school test score on the 1996 Busia District mock exams, total primary school pupils within 6 km, and the cost-sharing school indicator.

Figure 1: Project Timeline of the Primary School Deworming Program (PSDP) and the Kenya Life Panel Survey (KLPS)

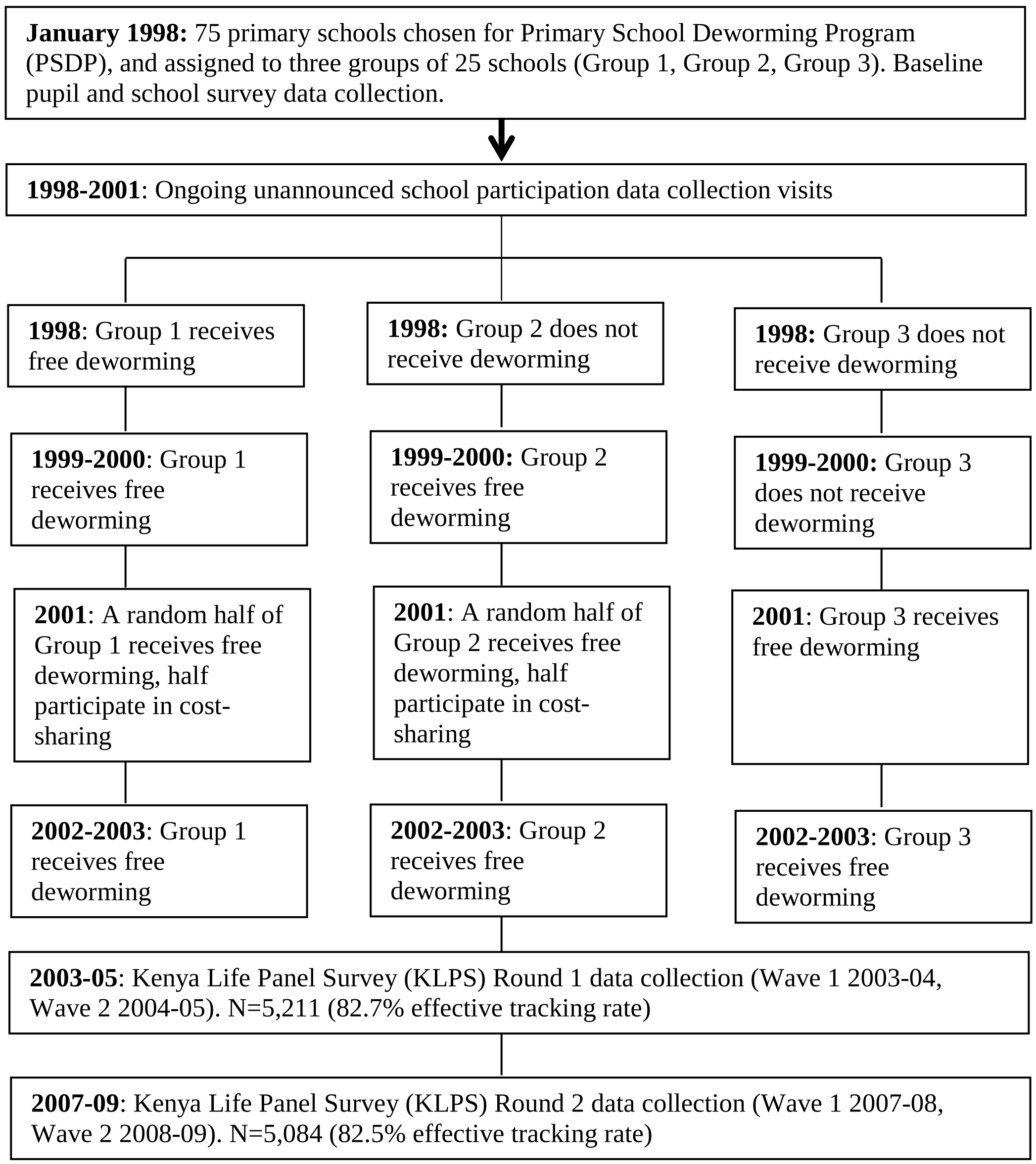
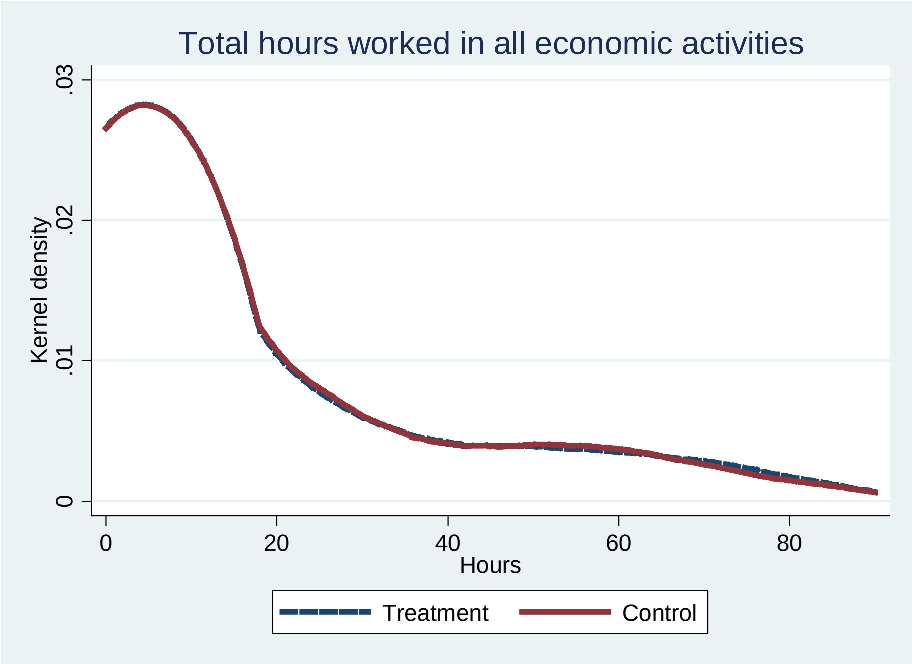
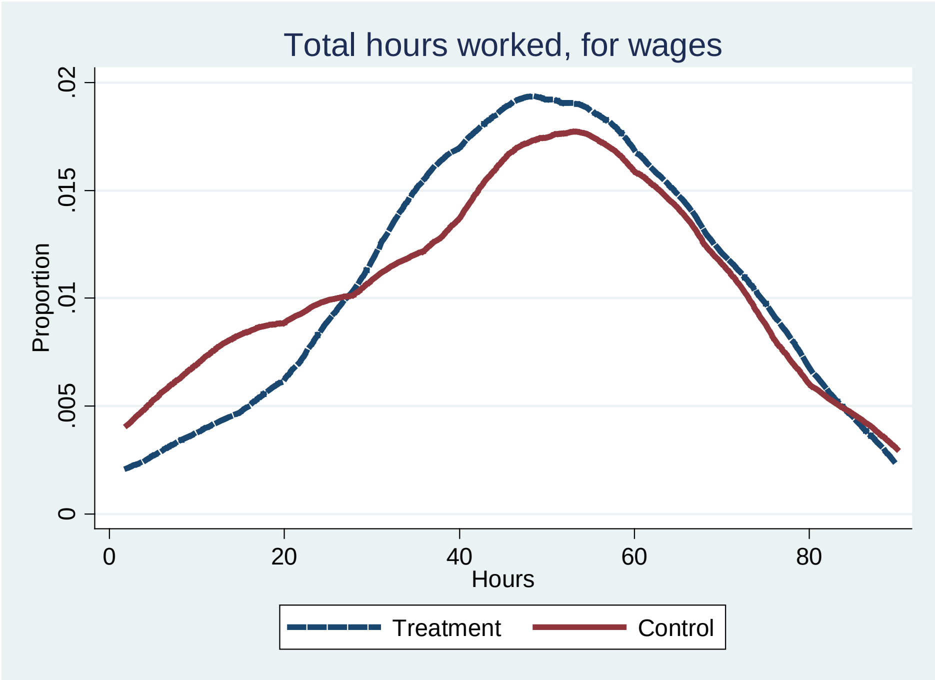


Figure 2:

Panel A: The distribution of hours worked in the last week (among those working for wages, self-employment or in agriculture),deworming treatment versus control

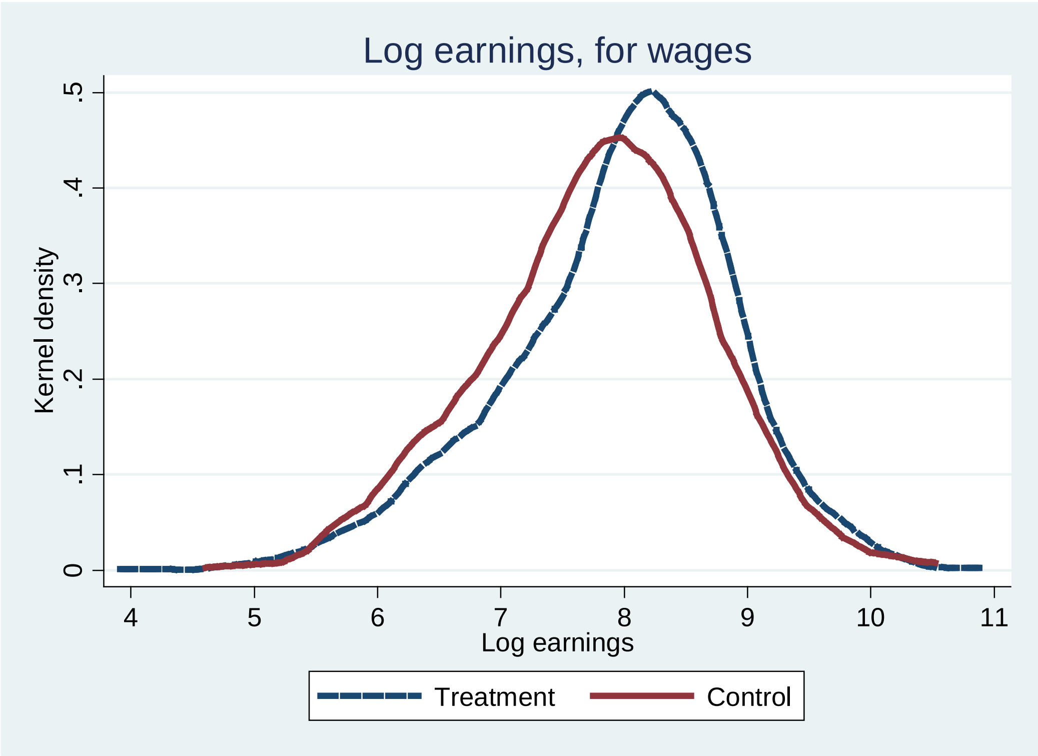


Panel B: The distribution of hours worked in the last week (among those working for wages), deworming treatment versus control



Notes: The sample used here includes all individuals who were surveyed in KLPS-2 and reported working for wages or in-kind in the last month. All observations are weighted to maintain initial population proportions.

Figure 3: The distribution of log labor earnings in the last month, deworming treatment versus control (among those with positive labor earnings)



Notes: The sample used here includes all individuals who were surveyed in KLPS-2 and reported working for wages or in-kind in the last month. All observations are weighted to maintain initial population proportions.

Supplementary Appendix A: Research Design Appendix (not intended for publication)

A.1 Selection of Primary Schools for the PSDP Sample:

There were a total of 92 primary schools in the study area of Budalangi and Funyula divisions, across eight geographic zones, in January 1998. Seventy-five of these 92 schools were selected to participate in PSDP. The 17 excluded schools include: town schools that were quite different from other local schools in terms of student socioeconomic background; single-sex schools; a few schools located on islands in Lake Victoria (posing severe transportation difficulties); and those few schools that had in the past already received deworming and other health treatments under an earlier small-scale ICS (NGO) program.

In particular, four primary schools in Funyula Town were excluded due to large perceived income differences between their student populations and those in other local schools. In particular, Moody Awori Primary School, Namboboto Boys Primary School, and Namboboto Girls School charged schools fees well in excess of neighboring primary schools, and thus attracted the local "elite". Nangina Girls Primary School is a private boarding school, and charged even higher fees, and was similarly excluded.

Four other primary schools in Budalangi division were excluded from the sample due to geographic isolation, which introduced logistic difficulties and would have complicated deworming treatment and data collection. Three of these schools – Maduwa, Buluwani and Bubamba Primary Schools – are located on islands in Lake Victoria. The fourth, Osieko Primary School, is separated from the rest of Budalangi by a marshy area.

Two additional schools were excluded. Rugunga Primary School in Budalangi division served as the pilot school for the PSDP in late 1997, receiving deworming treatment before other local schools, and thus it was excluded from the evaluation. Finally, Mukonjo Primary School was excluded since it was a newly opened school in 1998 with few pupils in the upper standards (grades), and thus was not comparable to the other sample schools.

Seven schools had participated in the ICS Child Sponsorship Program/School Health Program (CSP/SHP). In 1998, it was felt that identification of treatment effects in these schools could be complicated by the past and ongoing activities in those schools, including health treatment (and deworming in particular), and hence they were excluded from the sample. The NGO's earlier criteria in selecting these particular seven schools (in 1994-1995) is not clear.

A.2 Prospective Experimental Procedure:

Miguel and Kremer (2004) contains a partial description of the prospective experimental "list randomization" procedure, and we expand on it here. Schools were first stratified by geographical area (division, then zone)[16], and the zones were listed alphabetically (within each division), and then within each zone they were listed in increasing order of student enrolment in the school. Table 1 shows there is no significant difference between average school populations in the treatment and control groups.

While the original plan had been to stratify by participation in other NGO programs, the actual randomization was not carried out this way. Schools participating in the intensive CSP/SHP program were dropped from the sample (as detailed above), while 27 primary schools with less intensive NGO programs were retained in the sample. These 27 schools were receiving assistance in the form of either free classroom textbooks, grants for school committees, or teacher training and bonuses. It is worth emphasizing that the randomized evaluations of these various interventions did

[16] There are two divisions (Budalangi and Funyula) containing a total of eight zones (Agenga/Nanguba, Bunyala Central, Bunyala North, Bunyala South, Bwiri, Funyula, Namboboto, Nambuku).

not find statistically significant average project impacts on a wide range of educational outcomes.[17] The schools that benefited from these previous programs were found in all eight geographic zones; the distribution of the 27 schools across the eight zones is: Agenga/Nanguba (5 schools), Bunyala Central (1), Bunyala North (4), Bunyala South (2), Bwiri (4), Funyula (5), Namboboto (1), Nambuku (5). The results in the current paper are robust to including controls for inclusion in these other NGO programs (results not shown).

The schools were "stacked" as follows. Schools were divided by geographic division, then zone (alphabetically), and then listed according to school enrolment (as of February 1997, for grades 3 through 8) in ascending order. If there were, say, four schools in a zone, they would be listed according to school enrolment in ascending order, then they would be assigned consecutively to Group 1; Group 2; Group 3; Group 1. Then moving onto the next zone, the first school in that stratum was assigned to Group 2, the next school to Group 3, and so on. Thus the group assignment "starting value" within each stratum was largely arbitrary, except for the alphabetically first zone (in the first division), which assigned the school with the lowest enrolment in its geographic zone to Group 1. Finally, there were three primary schools (Runyu, Nangina Mixed, and Kabwodo) nearly excluded from the original stacking of 72 schools that were added back into the sample for the original randomization, to bring the sample up to 75. These schools were originally excluded for similar reasons as listed above – e.g., Runyu is rather geographically isolated, and Nangina Mixed is a relatively high quality school located near Funyula Town. However, in the interests of boosting sample size, these three schools were included in the list randomization alphabetically as the "bottom" three schools in the list.

Deaton (2010) raises concerns about the list randomization approach, in the case where the first school listed in the first randomization "triplet" is different than other schools (in our case, it has lower than average school enrolment); the same concerns would apply to several other well-known recent field experiments in development economics, most notably Chattopadhyay and Duflo's 2004 paper "Women as policymakers: Evidence from a randomized policy experiment in India" in *Econometrica*. However, this is not a major threat to our empirical approach. Following Bruhn and McKenzie (2009) we include all variables used in the randomization procedure (such as baseline school enrolment) as explanatory variables in our regression specifications, thus controlling for any direct effect of school size, and partially controlling for unmeasured characteristics correlated with school size. Table 3 shows that the estimate on the deworming treatment indicator is unchanged whether or not additional explanatory variables are included, suggesting that any bias is likely to be very small. The difference in average school enrollment between the treatment and control groups is small and not statistically significant (Table 1). Moreover, even if the first school in the first randomization triplet were an outlier along some unobserved dimension (which seems unlikely), given our sample size of 75 schools and 25 randomization triplets, and the fact that school size is not systematically related to treatment group assignment for the other 24 randomization triplets (as discussed above), approximately 96% of any hypothesized bias would be eliminated. Taken together, the prospective experimental design we exploit in the current paper is likely to yield reliable causal inference.

[17] See Glewwe, Paul, Michael Kremer, and Sylvie Moulin. (2009). "Many Children Left Behind? Textbooks and Test Scores in Kenya", *American Economic Journal: Applied Economics*, 1(1): 112-135.

Supplementary Appendix Table A1: Baseline (1998) summary statistics and PSDP randomization checks, wage earner subsample

	All mean (s.d.)	Treatment mean (s.d.)	Control mean (s.d.)	Treatment – Control (s.e.)	Kolmogorov-Smirnov p-value
Age (1998)	13.2	13.2	13.0	0.204	0.202
	(1.8)	(1.9)	(1.7)	(0.391)	
Grade (1998)	4.87	4.86	4.91	-0.054	0.445
	(1.61)	(1.63)	(1.57)	(0.141)	
Female	0.233	0.209	0.280	-0.071	--
	(0.423)	(0.407)	(0.450)	(0.045)	
Primary school located in Budalangi division	0.412	0.430	0.378	0.052	--
	(0.493)	(0.496)	(0.486)	(0.144)	
Population of primary school	477	504	425	78	0.342
	(218)	(246)	(136)	(56)	
School average test score (1996)	-0.010	-0.027	0.024	-0.050	0.273
	(0.408)	(0.415)	(0.391)	(0.106)	
Total treatment (Group 1, 2) primary school students within 6 km	3206	3115	3383	-267	0.172
	(908)	(802)	(1064)	(283)	
Total primary school students within 6 km	4731	4731	4730	1.72	0.342
	(1332)	(1173)	(1598)	(420)	

Notes: The data are from the PSDP, and includes all individuals surveyed in the KLPS2 who had worked for wages in the past month at the time of the interview. All observations are weighted to maintain initial population proportions. All variables are 1998 values unless otherwise noted. The average school test score is from the 1996 Busia District mock exam, and has been converted to units of normalized individual standard deviations. The "Treatment – Control" differences are derived from a linear regression of the outcome on a constant and the treatment indicator, but results are similar if we include further controls (for survey wave, 1998 administrative zone of residence, cost sharing school indicator, and baseline 1998 population of the individual's primary school). Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence. The Kolmogorov-Smirnov p-values are only presented for the non-binary variables, where it is informative.

Supplementary Appendix Table A2: Baseline (1998) summary statistics and attrition checks

	Full KLPS Sample	Found: Regular Tracking	Found: Intensive Tracking	Not Found	Found (Regular and Intensive) – Not Found
Age (1998)	12.4	12.4	12.5	12.7	-0.37***
	(2.2)	(2.2)	(2.2)	(2.1)	(0.09)
Grade (1998)	4.26	4.24	4.24	4.32	-0.105
	(1.69)	(1.68)	(1.70)	(1.70)	(0.063)
Female	0.486	0.461	0.495	0.535	-0.072***
	(0.500)	(0.499)	(0.501)	(0.499)	(0.016)
Assignment to the deworming treatment group	0.675	0.681	0.665	0.664	0.006
	(0.468)	(0.466)	(0.473)	(0.472)	(0.020)
Group 1 school	0.357	0.355	0.354	0.362	-0.015
	(0.479)	(0.479)	(0.479)	(0.481)	(0.025)
Group 2 school	0.318	0.326	0.311	0.302	0.021
	(0.466)	(0.469)	(0.463)	(0.459)	(0.021)
Years of assigned deworming treatment during 1998-2003	3.29	3.32	3.25	3.22	0.069
	(1.83)	(1.82)	(1.83)	(1.85)	(0.090)
Primary school located in Budalangi division	0.380	0.361	0.389	0.420	-0.067***
	(0.486)	(0.480)	(0.488)	(0.494)	(0.023)
Population of primary school	484	480	465	496	-20**
	(221)	(223)	(178)	(222)	(8)
School average test score (1996)	0.043	0.035	0.023	0.066	-0.026
	(0.439)	(0.434)	(0.416)	(0.453)	(0.021)
Total treatment (Group 1 and 2) primary school students within 6 km	3171	3182	3174	3149	30
	(910)	(915)	(918)	(900)	(36)
Total primary school students within 6 km	4678	4713	4691	4602	93
	(1340)	(1342)	(1335)	(1334)	(62)
Number of observations [a]	7530	4891	421	2218	7530

Notes: The regression results (Found – Not Found) in column 5 reweights appropriately for intensive tracking. [a] The number of observations is correct except for the Age (1998) variable, which has somewhat more missing data.

Supplementary Appendix Table A3: Impacts on school enrollment and participation

Panel A: Dep. var.: School enrollment indicator	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	Total
Deworming Treatment indicator	N/A	0.021*	0.036**	0.047**	0.046**	0.046*	0.028	0.035	0.017	0.003	0.279*
		(0.011)	(0.016)	(0.019)	(0.021)	(0.022)	(0.026)	(0.027)	(0.027)	(0.027)	(0.147)
Deworming Treatment pupils within 6 km	N/A	0.011	0.014	0.024	0.026	0.015	0.008	0.016	0.034	-0.011	0.138
(in ‘000s), demeaned		(0.013)	(0.015)	(0.017)	(0.018)	(0.025)	(0.027)	(0.027)	(0.029)	(0.031)	(0.149)
Mean in the control group		0.924	0.834	0.757	0.696	0.653	0.584	0.474	0.426	0.342	6.690
Observations		5,037	5,037	5,037	5,037	5,037	5,037	5,037	5,037	5,037	5,037
Panel B: Dep. var.: Primary school participation											
Deworming Treatment indicator	0.074***	0.068***	0.013	0.057**	N/A	N/A	N/A	N/A	N/A	N/A	0.129**
	(0.023)	(0.023)	(0.020)	(0.024)							(0.064)
Deworming Treatment pupils within 6 km	0.019	-0.008	-0.019	0.009							0.044
(in ‘000s), demeaned	(0.024)	(0.018)	(0.020)	(0.017)							(0.049)
Mean in the control group	0.839	0.709	0.686	0.586							2.513
Observations	4,900	4,821	4,342	3,831							5,037

Notes: The sample used in Panel A includes all individuals who were surveyed in KLPS2. The sample used in Panel B includes a subset of these individuals who additionally have school participation data from at least one of the years between 1998 and 2001. All regressions include controls for baseline 1998 primary school population, geographic zone of the school, cost-sharing school in 2001 indicator, a gender indicator and pupil grade. The treatment indicator in 1998 is the Group 1 indicator. There is no estimated result for 1998 in Panel A since all individuals were enrolled in school in 1998 (as this was a study inclusion criterion). All observations are weighted to maintain initial population proportions. Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence.

Supplementary Appendix Table A4: Deworming impacts on labor market outcomes among subgroups

	Dependent variable:								
	Ln(Total labor earnings, past month)			Indicator for worked for wages or in-kind in last month			Hours worked (for wages, self-employed, agriculture) last week		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Deworming Treatment indicator	0.219**	0.297*	0.255***	0.002	-0.018	-0.016	3.40**	1.53	1.79*
	(0.103)	(0.152)	(0.092)	(0.024)	(0.019)	(0.018)	(1.40)	(1.10)	(0.96)
Female	-0.473***			-0.128***			-3.43**		
	(0.140)			(0.022)			(1.68)		
Female * Treatment	0.121			-0.035			-3.41*		
	(0.195)			(0.027)			(1.98)		
Grades 5-7 in 1998		0.497***			0.105***			7.46***	
		(0.164)			(0.023)			(1.71)	
Grades 5-7 * Treatment		-0.069			0.004			0.43	
		(0.186)			(0.028)			(2.02)	
Moderate-heavy worm infection rate at the zonal level (1998), demeaned			-0.048			-0.035*			-0.70
			(0.084)			(0.018)			(0.85)
Moderate-heavy infection rate * Treatment			0.071			0.028*			0.81
			(0.078)			(0.015)			(0.77)
Additional controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R^2	0.183	0.170	0.183	0.093	0.085	0.094	0.065	0.057	0.064
Observations	710	710	710	5081	5081	5081	5084	5084	5084
Mean (s.d.) in the control group	7.86	7.86	7.86	0.166	0.166	0.166	15.2	15.2	15.2
	(0.88)	(0.88)	(0.88)	(0.372)	(0.372)	(0.372)	(21.9)	(21.9)	(21.9)

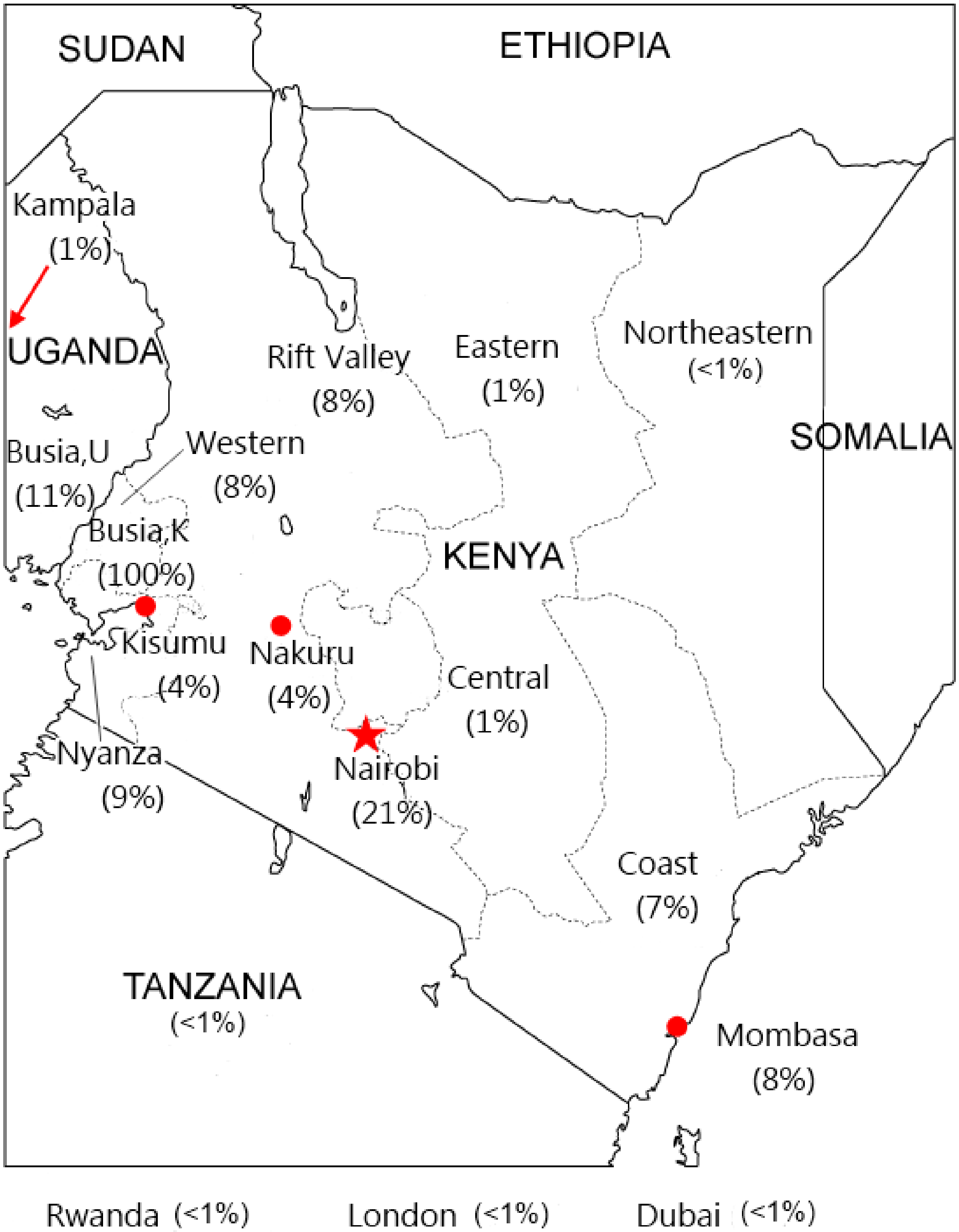
Notes: The sample used in columns (1)-(3) includes all individuals surveyed in the KLPS2 who report positive labor earnings at the time of survey and include data for the relevant dependent variable. The sample used in columns (4)-(6) includes all surveyed individuals with non-missing information on wage employment. Labor earnings include cash and in-kind. All observations are weighted to maintain initial population proportions. Additional controls include a gender indicator, baseline grade fixed effects, geographic zone fixed effects, the mean pre-program school test score, baseline school population, cost-sharing school in 2001 indicator, survey wave indicator, and month of interview fixed effects, as well as both the total number of deworming treatment school pupils and the total number of primary school pupils within 6 km (in '000s), demeaned (coefficient estimates not shown). Standard errors are clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence.

Supplementary Appendix Table A5: The impact of eliminating moderate-heavy worm infections on economic outcomes, two-sample instrumental variable estimates

Dependent variable	Control group variable mean (s.d.)	IV-2SLS coefficient estimate (s.e.) on predicted years of moderate-heavy worm infection	Obs.
Self-reported health "very good", 2007-2009	0.673	-0.093***	5070
	(0.469)	(0.030)	
Total years enrolled in school, 1998-2007	6.69	-0.229	5037
	(2.97)	(0.203)	
Number of meals eaten yesterday	2.16	-0.099*	5083
	(0.64)	(0.047)	
Hours worked (for wages, self-employed, agriculture) in last week	15.2	-3.14**	5084
	(21.9)	(1.24)	
Hours worked (for wages, self-employed, agriculture) in last week, among those with hours worked > 0	23.0	-3.23*	3514
	(23.4)	(1.59)	
Hours worked (for wages or in-kind) in the last week	42.2	-7.96**	693
	(24.7)	(3.65)	
Ln(Total labor earnings, past month)	7.86	-0.380**	710
	(0.88)	(0.133)	
Ln(Total labor earnings, most recent month worked)	7.88	-0.266**	1175
	(0.91)	(0.108)	
Ln(Wage = Total labor earnings / hours, past month)	2.82	-0.175	625
	(0.96)	(0.154)	

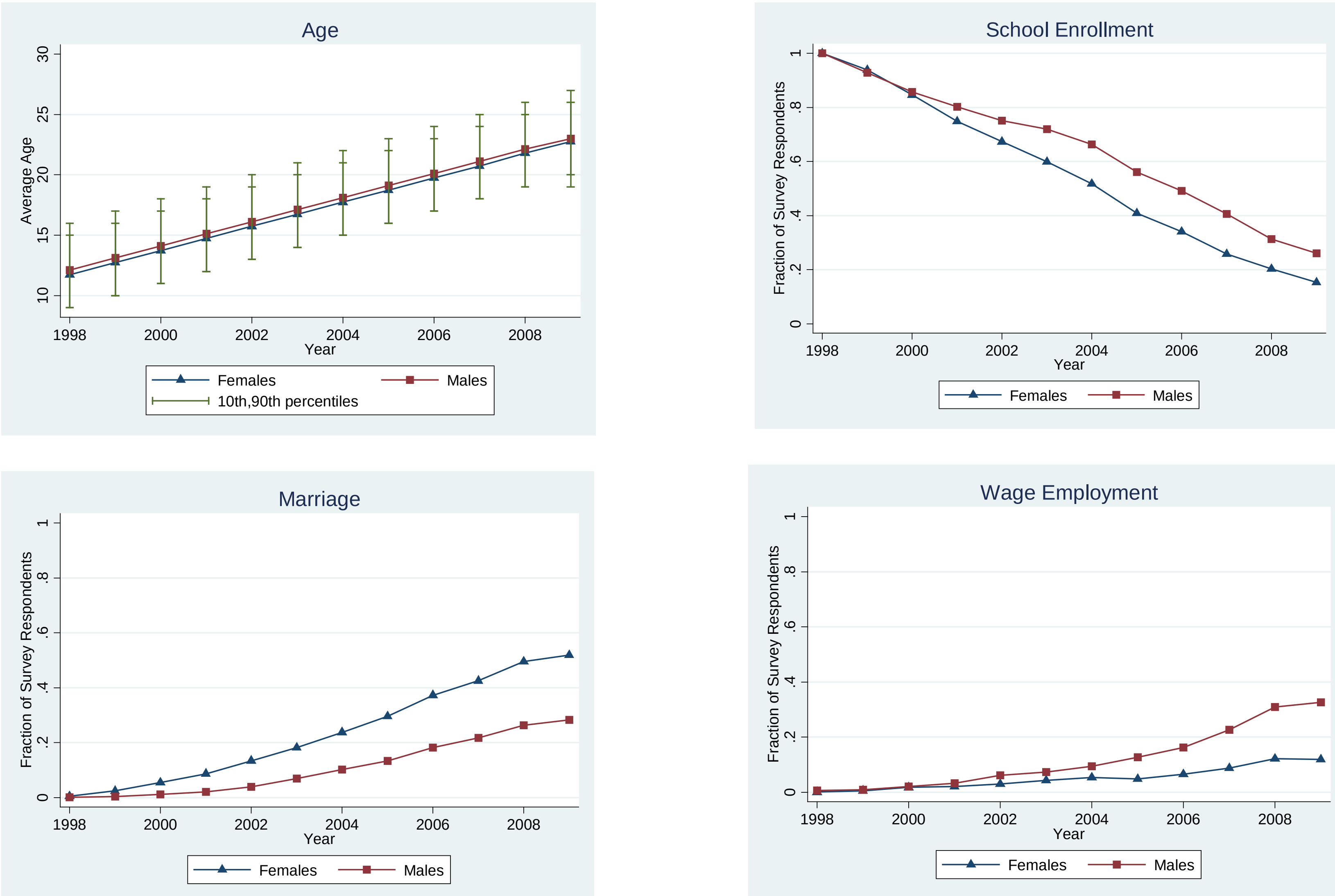
Notes: Two-sample instrumental variable estimates. Standard errors are bootstrapped and clustered by school. Significant at 90% (*), 95% (**), 99% (***) confidence. All regressions include controls for baseline 1998 primary school population, geographic zone of the school, survey wave and month of interview, a female indicator variable, baseline 1998 school grade fixed effects, the average school test score on the 1996 Busia District mock exams, total primary school pupils within 6 km, and the cost-sharing school indicator. The instrumental variables in the first-stage are the deworming treatment indicator, the number of deworming Treatment pupils within 6 km (in '000s) demeaned, and the cost-sharing indicator variable.

Supplementary Appendix Figure A1: Migration residential location map

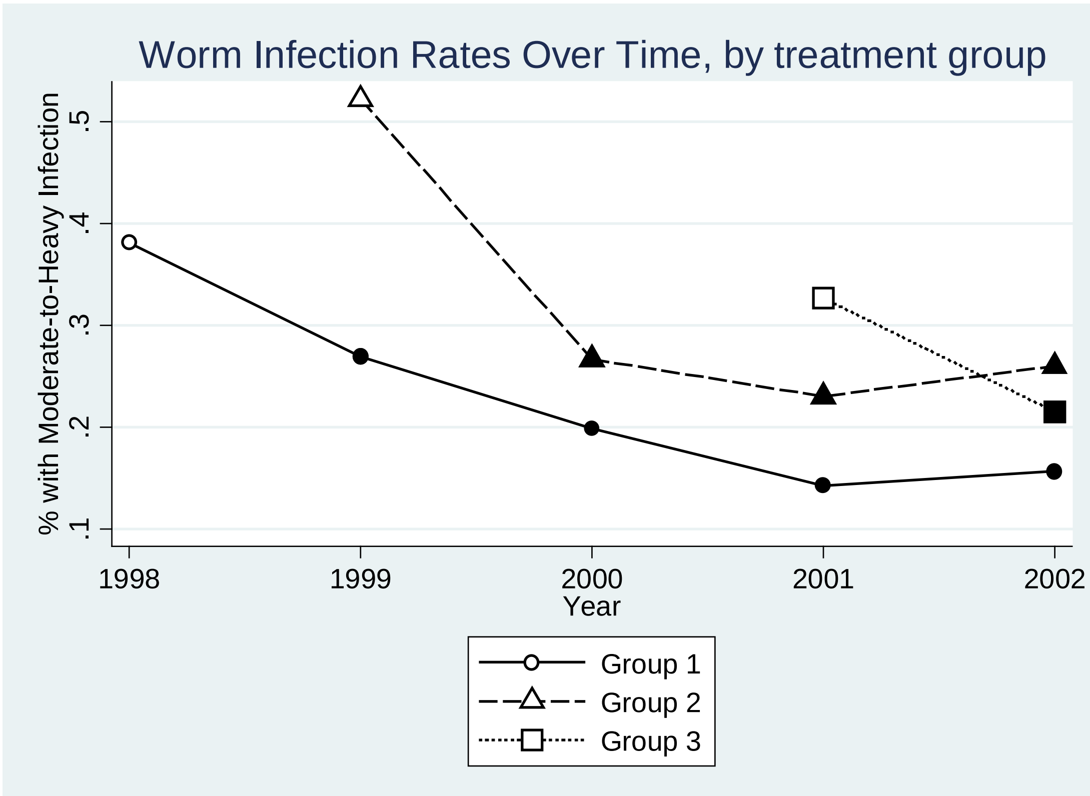


Notes: Percentages sum to greater than one, since they capture residential location (for at least four consecutive months) at any point during 1998-2009.

Supplementary Appendix Figure A2: Age, School Enrollment, Marriage and Employment Patterns over 1998-2009



Supplementary Appendix Figure A3: Moderate-heavy worm infection rates over time by PSDP treatment group



Notes: Hollow symbols (circles, triangles, squares) denote pre-deworming observations (for the group), and filled symbols denote post-deworming. Group 1 and Group 2 schools are jointly considered “treatment” in most of the analysis. Note that half of the Group 1 and Group 2 schools took part in deworming cost-sharing in 2001, likely accounting for some of the slight rise in infection rates observed in those groups between 2001 and 2002.