

NBER WORKING PAPER SERIES

THE ILLUSION OF SUSTAINABILITY

Michael Kremer  
Edward Miguel

Working Paper 10324  
<http://www.nber.org/papers/w10324>

NATIONAL BUREAU OF ECONOMIC RESEARCH  
1050 Massachusetts Avenue  
Cambridge, MA 02138  
February 2004

We thank ICS Africa and the Kenya Ministry of Health for their cooperation in all stages of the project, and would especially like to acknowledge the contributions of Alicia Bannon, Elizabeth Beasley, Laban Benaya, Simon Brooker, Pascaline Dupas, Alfred Luoba, Sylvie Moulin, Robert Namunyu, Carol Nekesa, Peter Wafula Nasokho, Polycarp Waswa and the entire PSDP staff, without whom the project would not have been possible. Gratitude is extended to the teachers, school children, and households of Busia for participating in the study. Melissa Gonzalez-Brenes, Tina Green, Jean Lee, Emily Oster, and Avery Ouellette have provided excellent research assistance. We thank Andrew Foster, Caroline Hoxby, Guido Imbens, Botond Koszegi, Kaivan Munshi, Mark Rosenzweig, John Strauss, and Chris Udry for helpful conversations. We are grateful for financial support from the World Bank, NIH Fogarty International Center (R01 TW05612-02), and Berkeley Center for Health Research. All errors are our own. The views expressed herein are those of the authors and not necessarily those of the National Bureau of Economic Research.

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NBER Working Paper No. 10324  
February 2004, Revised August 2006  
JEL No. I1, I3, O2

### **ABSTRACT**

The history of foreign development assistance is one of movement away from addressing immediate needs and toward focusing on the underlying causes of poverty. A recent manifestation is the move towards "sustainability," which stresses community mobilization, education, and cost-recovery. This stands in contrast to the traditional economic analysis of development projects, with its focus on providing public goods and correcting externalities. We examine evidence from randomized evaluations on strategies for combating intestinal worms, which affect one in four people worldwide. Providing medicine to treat worms was extremely cost effective, although medicine must be provided twice per year indefinitely to keep children worm-free. An effort to promote sustainability by educating Kenyan schoolchildren on worm prevention was ineffective, and a "mobilization" intervention from psychology failed to boost deworming drug take-up. Take-up was highly sensitive to drug cost: a small increase in cost led to an 80 percent reduction in take-up (relative to free treatment). The results suggest that, in the context we examine, the pursuit of sustainability may be an illusion, and that in the short-run, at least, external subsidies will remain necessary.

Michael Kremer  
Harvard University  
Department of Economics  
Littauer Center 207  
Cambridge, MA 02138  
and NBER  
mkremer@fas.harvard.edu

Edward Miguel  
Department of Economics  
Evans Hall, #3880  
University of California, Berkeley  
Berkeley, CA 94720-3880  
and NBER  
emiguel@econ.berkeley.edu

## 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”<sup>1</sup>. In the 1950's and 1960's, it was widely argued that long-run economic performance depended on capital investment, and 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 1980's 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 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 1990's, 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.<sup>2</sup> 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 in most of their responsibilities. In a large water project in the Kenyan area we study, 43% 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% 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, then 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 et al. [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 et al., 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.<sup>3</sup>

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.<sup>4</sup>

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 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 stomach ache, 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 non-governmental 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 75 project schools include nearly all rural primary schools in the area, with over 30,000 enrolled pupils between the ages of six and 18, over 90% 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 sub-unit (zone) and by involvement in other non-governmental assistance programs, and were then listed alphabetically and every third school 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, while 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 be 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% 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.<sup>5</sup>

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<sup>6</sup> – 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 50 Group 1 and Group 2 schools were stratified by treatment group and geographic location and then 25 were randomly selected (using a computer random number generator) to pay user fees for medical treatment in 2001, while the other 25 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 25 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 the cost of drug purchase and delivery through this program (at US\$1.49) and 60% of the cost in the Tanzania program.<sup>7</sup>

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.<sup>8</sup> 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 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 five 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 64 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 non-random 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]).

#### 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 above, 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  $\gamma$ ; the effectiveness of the drug  $\phi$  (which incorporates the percentage reduction in worm load that results from taking the drug and the rapidity of re-infection)<sup>9</sup>; and an idiosyncratic individual specific taste for deworming  $\mu_i$  which 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  $\gamma$  may depend on individual characteristics  $X$ , and also 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  $C_\infty$ . The drug subsidies, health education, and verbal commitment interventions 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  $\phi$  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

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

where  $U$  is individual utility from deworming, conditional on the treatment choices of other individuals, and  $\omega_{it}$  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.

## 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  $\phi_{i0}$ , which may be greater or less than the actual effectiveness  $\phi$ . Priors could be less than  $\phi$  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 et al., 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  $\phi$  as a draw from a distribution believed to have mean  $\phi_0(X_i)$  and variance  $\sigma_\phi^2$ . While people can learn about the realization of  $\phi$  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  $\phi$  and variance  $\sigma_\epsilon^2$ .

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.<sup>10</sup> 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 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.

### 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  $\hat{\phi}_i = \phi$  and  $C = C_\infty$  for all individuals  $i$ .

Consider first the case in which  $\beta = 0$  (no pure imitation effects). Let  $\lambda$  denote the share of the population taking up treatment and let  $\lambda^*$  denote the steady state share such that if a proportion  $\lambda^*$  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:

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

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

While  $\lambda^*$  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  $\lambda^*$  is arbitrarily close to zero in the absence of imitation effects, if imitation effects are sufficiently strong 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 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.<sup>12</sup> 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.<sup>13</sup>

#### D. Take-up along the Transition Path

We next turn to modeling take-up along the transition path. By time  $\tau$ , 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  $[\sum_{k=2 \dots \tau} (k-1) \cdot \{p^2(1-p)^{k-2}\}]$ , and more generally the probability that a signal is transmitted from a  $j$ th order link is  $\sum_{k=j}^{\tau} \binom{k-1}{j-1} \cdot \{p^j(1-p)^{k-j}\}$  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  $j$ th 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  $j$ th order link by time  $\tau$  is thus:

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

An additional signal can impact take-up behavior, so that  $[(T_{it} \mid \text{Signal}) - (T_{it} \mid \text{No signal})]$  is nonzero, by changing beliefs about  $\phi$  (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:

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

where  $\phi_0(X_i)$  is the mean of her prior distribution,  $\phi_S$  is the sample average of signals received through the social network, and  $\sigma_N^2 \equiv \sigma_\varepsilon^2 / N_{it}^E$  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,  $\phi$ .

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 equation 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 above, 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 either 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 4.6, 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

##### 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 10 to 17 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.<sup>14</sup> 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 40 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 is 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 identification 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 50 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 km separating two schools, the average number of named links falls by 0.06 (standard error 0.005, statistically significant at 99% 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).<sup>15</sup>  $T_{ij}$  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.

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.  $Z_j$  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:

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

We include interaction terms between household characteristics and social links to estimate heterogeneous treatment effects, for example, as a function of respondent education, and also 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), or 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% level, but the coefficient is small (and, surprisingly, positive). Third-order links to early treatment schools are not significantly associated with any observable characteristics.

### 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 0.84



(standard error 0.11—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 0.015, 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 et al., 2001; Bandiera and Rasul, 2005].

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 0.09—but this is only suggestive.<sup>16</sup>

### 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% 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 6 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% confidence in some specifications (Appendix Table A.I., 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.<sup>17</sup>

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 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  $-0.041$ , 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 equation 5,  $\sigma_N^2$  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].<sup>18</sup>

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).<sup>19</sup>

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).<sup>20</sup> We are similarly unable to reject the hypotheses that social effects are the same for links to relatives versus non-relatives, 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 ( $\phi_{i0}$ ) 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. 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% 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 13 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% 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).<sup>21</sup>

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.

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 than 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% 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.

#### 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 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  $-3.1\%$  (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  $27\%$ ). 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  $90\%$  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 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.<sup>22</sup> 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 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  $(0.09)*(-0.3\%) = 0.03\%$ , rather than the -3.1% 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  $(0.5)*(-0.3\%) = -0.15\%$  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  $(\text{Total number of links to PSDP schools}) / (\text{Total number of Group 1, 2 pupils} / \text{Total number of Groups 1, 2, 3 pupils})$  is nearly identical at 1.91.

#### 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).<sup>23</sup> 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% of respondents claimed that helminth infections are “very bad” for child health, even though take-up is much lower than 92%. 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 to 10). Respondents could name only 1.8 of ten common symptoms on average.<sup>24</sup> 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.

#### 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 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.<sup>25</sup> 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 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 30 opportunities to adopt (15 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%) 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% (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% 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 part of the 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 non-experimental 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 et al. [1995] find utilization fell by 52% in 1989. Our analysis uses random assignment to estimate the effect of cost sharing.<sup>26</sup> 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. 75% of households in the free treatment schools received deworming drugs in 2001 (Table I, Panel C), while the rate was only 19% 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 62 percentage points (Table VII, regression 1), with the effect similar across households with various socioeconomic characteristics (regression 2).<sup>27</sup> 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% 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 first-hand. 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<sup>28</sup>, 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, 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

re-infection. 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 et al. [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 2001 Pupil Questionnaire administration, 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% 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 was 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.

## VIII. Conclusion

A program which 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 suggest 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

Appendix Table A.I  
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)} <sup>2</sup>	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)	1678	1678	1678	1678
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 than 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 km, 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.

Michael Kremer  
Harvard University  
The Brookings Institution  
The Center for Global Development  
NBER

Edward Miguel  
University of California, Berkeley  
NBER

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#### Notes

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1. Refer to the Oxfam website for the details ([http://www.oxfam.org.uk/about\\_us/history/history2.htm](http://www.oxfam.org.uk/about_us/history/history2.htm)).
  2. Sustainability has other meanings, including an environmental meaning, but we focus on financial sustainability.
  3. Lengeler [1999] reaches similar conclusions.
  4. Refer to Adams et al. [1994], Corbett et al. [1992], Hotez and Pritchard [1995], and Pollitt [1990].
  5. The project followed the standard practice at the time in mass deworming programs of not treating girls of reproductive age – typically aged 13 years 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 et al. [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.
  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 are ethnically Luhya (a Bantu-speaking group) though Luos are four percent of our sample. However, traditional Luo views are closely related to views found among other African groups [Green et al., 1994; Green, 1997].



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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.

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.

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 takeup rate  $\lambda^*$  is a sufficient statistic for the entire history since the take-up rate is the same in every period.

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.

14. Survey refusal rates were low, as is typical for this region. 13% 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.

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

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 than 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.

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).

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.

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19. The results are also robust to a specification without socioeconomic controls (Appendix Table A.I, 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 3 km 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 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% 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).
21. Refer to the working paper version [Miguel and Kremer, 2003] for further discussion of child social effects.
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.
23. A fourth option, “effective, but the worms come back” was rarely chosen by respondents.
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.
25. We focus on the following range of parameter values for the model:  $\underline{b}=0$ ,  $\bar{b}=2$ ,  $\sigma_0^2=1$ ,  $\sigma_\epsilon^2=1$  to 10,  $C=0.1$  to 2,  $\phi=0.75$ ,  $\tau=6$ , annual discount rate  $\delta=0.9$  to 1, and  $p=0.05$  to 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 50 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  $\sigma_0^2$ , choosing various values for  $\sigma_\epsilon^2$  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.
26. Gertler and Molyneux [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 4 above.
28. This also holds controlling for initial 1998 cleanliness or using differences-in-differences (regressions not shown).



Table I  
Summary statistics

	Mean	Std dev.	Obs.
<u>Panel A: Parent Social Links (Round 1 and Round 2 Data)</u>			
Total direct (first-order) links	10.2	3.4	1678
With children in own school	4.4	2.8	1678
With children not in Group 1, 2, or 3 schools	3.0	2.4	1678
With children in Group 1, 2, 3 schools – not own school	2.8	2.4	1678
With children in Group 1, 2 schools – not own school (“early treatment”)	1.9	2.0	1678
With children in Group 1 schools – not own school	0.9	1.4	1678
Proportion with children in early treatment schools	0.66	0.37	1358
With children in early treatment schools, with whom respondent speaks at least twice per week (“Close Links”)	1.2	1.6	1678
With children in early treatment schools, with whom respondent speaks less than twice per week (“Distant Links”)	0.7	1.1	1678
Second-order exposure to Group 1, 2 or 3 schools (not own school), through parent links	4.5	4.1	1678
Second-order exposure to early treatment schools (Groups 1 and 2, not own school), through parent links	2.9	2.9	1678
Third-order exposure to Group 1, 2 or 3 schools (not own school), through parent links	3.9	5.3	1678
Third-order exposure to early treatment schools (Groups 1 and 2, not own school), through parent links	2.8	4.1	1678
<u>Panel B: Parent Social Links (Round 2 Data)</u>			
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
<u>Panel C: Deworming Treatment Take-up</u>			
Took deworming drugs in 2001 (Group 2 and 3)	0.61	0.49	1678
Proportion deworming drug take-up in 2001, respondent’s own school	0.61	0.28	1678
Took deworming drugs in 2001, free treatment schools	0.75	0.43	1251
Took deworming drugs in 2001, cost-sharing schools	0.19	0.39	427
Provided parental consent for deworming drugs in 2001	0.76	0.43	1678
<u>Panel D: Cost-Sharing Interventions</u>			
Cost-sharing school indicator	0.25	0.43	1678
Cost-sharing school indicator, albendazole only treatment	0.17	0.38	1678
Cost-sharing school indicator, albendazole and praziquantel treatment	0.08	0.27	1678
Effective price of deworming per child (Kenyan shillings)	6.3	15.7	1678

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.

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 (1)	Probit (2)	OLS (3)	Probit (4)	OLS (5)	Probit (6)
Explanatory variables:						
# Parent links with children in early treatment schools (Group 1, 2, not own school)	0.017 (0.085)	-0.004 (0.012)	-0.039 (0.067)	0.029** (0.014)	-0.178 (0.128)	-0.003 (0.018)
# Parent links with children in Group 1, 2, or 3 schools, not own school	0.086 (0.096)	0.007 (0.013)	-0.047 (0.072)	-0.016 (0.017)	0.295*** (0.101)	-0.030 (0.025)
Second-order exposure to early treatment schools (Groups 1 and 2, not redundant with first order links), parent links	-0.122 (0.083)	-0.004 (0.010)	-0.060 (0.060)	0.021* (0.011)	-0.168* (0.086)	0.044* (0.023)
Second-order exposure to Group 1, 2 or 3 schools (not redundant with first order links), parent links	0.058 (0.072)	-0.000 (0.007)	0.044 (0.048)	-0.012* (0.009)	0.031 (0.096)	-0.023 (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 (0.104)	-0.008 (0.015)	0.008 (0.055)	0.006 (0.017)	-0.103 (0.097)	-0.168 (0.021)
Third-order exposure to Group 1, 2 or 3 schools (not redundant with first and second order links), parent links	0.101 (0.077)	0.006 (0.011)	0.015 (0.051)	-0.002 (0.012)	0.044 (0.084)	0.024 (0.018)
Cost-sharing school indicator	0.16 (0.289)	0.003 (0.042)	0.073 (0.232)	0.011 (0.059)	1.361 (0.852)	0.03 (0.096)
Group 2 school indicator	-0.578* (0.290)	-0.033 (0.04)	0.091 (0.202)	0.021 (0.048)	-0.090 (0.308)	-0.209*** (0.069)
Other social link controls, socio-economic controls (excluding dependent variable)	Yes	Yes	Yes	Yes	Yes	Yes
Number of observations (parents)	1678	1678	1678	1678	1678	745
Mean (s.d.) of dependent variable	4.6 (3.9)	0.58 (0.49)	5.5 (2.3)	0.61 (0.49)	1.7 (2.0)	0.27 (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 99 (\*\*\*), 95 (\*\*), 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 # Parent links, total.

Table III  
Non-experimental social effect estimates (Groups 2 and 3)

Explanatory variables:	Dependent variable: Child took deworming drugs in 2001		
	(1)	(2)	(3)
Proportion deworming drug take-up in 2001, respondent's own school (not including respondent)	0.84*** (0.11)		
# Parent links with children in respondent's own school whose children received deworming		0.015 (0.011)	
# Parent links with children in early treatment schools whose children received deworming and had "good effects"			-0.005 (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			-0.004 (0.049)
# Parent links with children in early treatment schools whose children did not receive deworming			0.006 (0.056)
# Parent links with children in early treatment schools, respondent does not know whether they received deworming			-0.010 (0.019)
Total social link controls, socio-economic controls	Yes	Yes	Yes
Number of observations (parents)	1678	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 than 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 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 km, 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, we can reject that the coefficient estimates on (# Links with children in early treatment schools whose children received deworming and had good effects) and on (# Links with children in early treatment schools whose children received deworming and had side effects) are equal at 90% confidence (p-value=0.092).

Table IV  
Experimental social effect estimates (Groups 2 and 3)

Explanatory variables:	Dependent variable: Child took deworming drugs in 2001				
	(1)	(2)	(3)	(4)	(5)
# Parent links with children in early treatment schools (Groups 1 and 2, not own school)	-0.031** (0.014)	-0.041** (0.017)			-0.002 (0.018)
# Parent links with children in early treatment schools * Group 2 school indicator		0.018 (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				0.025 (0.028)	
# Parent links with children in early treatment schools * Respondent years of education					-0.0062* (0.0032)
# Parent links with children in Group 1, 2, or 3 schools, not own school	0.013 (0.011)	0.013 (0.017)	-0.006 (0.009)		-0.014 (0.014)
# Parent links with children not in Group 1, 2, or 3 schools	-0.008 (0.007)	-0.008 (0.009)	-0.005 (0.007)	-0.008 (0.007)	-0.008 (0.011)
# Parent links, total	0.019*** (0.005)	0.029*** (0.007)	0.021*** (0.007)	0.018*** (0.005)	0.013 (0.008)
Respondent years of education	0.003 (0.003)	0.003 (0.003)	0.002 (0.004)	0.002 (0.003)	-0.016 (0.012)
Community group member	0.029 (0.025)	0.033 (0.026)	0.038 (0.029)	0.031 (0.025)	0.025 (0.025)
Total number of children	0.005 (0.006)	0.006 (0.006)	0.005 (0.007)	0.005 (0.006)	0.006 (0.006)
Iron roof at home	0.010 (0.027)	0.007 (0.027)	0.010 (0.032)	0.010 (0.027)	0.008 (0.027)
Distance home to school (km)	-0.018** (0.009)	-0.018** (0.009)	-0.014 (0.010)	-0.017* (0.009)	-0.018** (0.009)
Group 2 school indicator	0.01 (0.05)	0.20** (0.09)	0.01 (0.05)	0.01 (0.05)	0.01 (0.05)
Cost-sharing school indicator	-0.62*** (0.08)	-0.62*** (0.08)	-0.62*** (0.09)	-0.63*** (0.08)	-0.63*** (0.08)
Number of observations (parents)	1678	1678	1358	1678	1678
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. Regression 2 also includes interaction terms (# Parent social links with children in Group 1, 2, or 3 schools, not own school)\*(Group 2), (# Parent social links with children not in Group 1, 2, or 3 schools)\*(Group 2), and (# Parent social links, total)\*(Group 2). Regression 3 excludes parents for which (# 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 (# Parent social links with children in Group 1, 2, or 3 schools, not own school)\*(Respondent years of education) and (# Parent social links with children not in Group 1, 2, or 3 schools)\*(Respondent years of education), not shown.



Table V  
First-order and higher-order social effect estimates (Groups 2 and 3)

Explanatory variables:	Dependent variable: Child took deworming drugs in 2001				
	(1)	(2)	(3)	(4)	(5)
# Parent links with children in early treatment schools (Groups 1 and 2, not own school)	-0.031** (0.014)		-0.044*** (0.015)		-0.037** (0.015)
# Parent links with children in Group 1, 2, or 3 schools, not own school	0.013 (0.011)		0.020 (0.015)		0.02 (0.015)
Proportion direct (first-order) parent links with children in early treatment schools				-0.14*** (0.05)	
Second-order exposure to early treatment schools (Groups 1 and 2, not own school), parent links		-0.035*** (0.013)	-0.047*** (0.013)		-0.048*** (0.014)
Second-order exposure to Group 1, 2 or 3 schools (not own school), parent links		0.025** (0.012)	0.032** (0.012)		0.033*** (0.012)
Proportion second-order parent links with children in early treatment schools				-0.23*** (0.09)	
Third-order exposure to early treatment schools (Groups 1 and 2, not own school), parent links					-0.014 (0.012)
Third-order exposure to Group 1, 2 or 3 schools (not own school), parent links					0.008 (0.01)
Total social link controls, socio-economic controls	Yes	Yes	Yes	Yes	Yes
Number of observations (parents)	1678	1678	1678	1173	1678
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 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 km, as well as the Group 2 indicator and Cost-sharing school indicator.

Table VI  
Effects on deworming attitudes and knowledge

Dependent variable:	Estimate on # Parent links with children in early treatment schools [Experimental]	Estimate on # Parent links with children in early treatment schools whose children received deworming [Non-experimental]	Estimate on # Parent links with children in early treatment schools with whom respondent spoke about deworming [Non-experimental]	Mean dep. var.
<u>Panel A: Attitudes</u>				
1) Parent thinks deworming drugs “not effective”	0.017** (0.007)	0.013 (0.008)	0.009** (0.004)	0.12
2) Parent thinks deworming drugs “very effective”	-0.007 (0.010)	0.026** (0.013)	0.040*** (0.007)	0.43
3) Parent thinks deworming drugs have “side effects”	0.000 (0.003)	-0.001 (0.003)	0.003* (0.002)	0.04
4) Parent thinks worms and schisto. “very bad” for child health	-0.001 (0.006)	-0.004 (0.006)	-0.006* (0.003)	0.92
<u>Panel B: Knowledge</u>				
5) Parent “knows about ICS deworming program”	0.004 (0.011)	0.050*** (0.014)	0.055*** (0.011)	0.70
6) Parent “knows about the effects of worms and schistosomiasis”	-0.001 (0.013)	0.045*** (0.013)	0.039*** (0.009)	0.68
7) Number of infection symptoms parents able to name (0-10)	-0.006 (0.005)	0.018** (0.008)	0.010** (0.005)	1.8
8) Parent able to name “fatigue” as symptom of infection	-0.004 (0.010)	0.028*** (0.009)	0.021*** (0.006)	0.20
9) Parent able to name “anemia” as symptom of infection	0.005 (0.009)	-0.003 (0.011)	0.010** (0.005)	0.22
10) Parent able to name “weight loss” as symptom of infection	0.002 (0.006)	0.004 (0.005)	-0.001 (0.004)	0.06

Notes: Data from 2001 Parent Survey, and 2001 administrative records. Marginal probit coefficient estimates are presented, and each entry is the result of a separate regression. 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 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 km, 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?”, and their responses were recorded by the enumerator.

Table VII  
The impact of cost-sharing

Explanatory variables:	Dependent variable: Child took deworming drugs in 2001		
	(1)	(2)	(3)
Cost-sharing school indicator	-0.62*** (0.08)	-0.49*** (0.15)	-0.62*** (0.12)
Cost-sharing * Respondent years of education		0.003 (0.007)	
Cost-sharing * Community group member		0.027 (0.067)	
Cost-sharing * Total number of children		-0.015 (0.016)	
Cost-sharing * Iron roof at home		-0.04 (0.06)	
Effective price of deworming per child (= Cost / # household children in that school)			-0.001 (0.002)
1 / (# household children in that school)			-0.35*** (0.07)
Social links, other controls	Yes	Yes	Yes
Number of observations (parents)	1678	1678	1678
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 than 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 km, as well as the Group 2 indicator (as in Table IV above).

Table VIII  
PSDP health behavior impacts (1999)

	Group 1	Group 2	Group 1 – Group 2 (s.e.)
<u>Panel A: Health Behaviors, all pupils (Grades 3-8)</u>			
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)
<u>Panel B: Health behaviors, girls ≥ 13 years old</u>			
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)
<u>Overall cross-school externality effect for Group 2</u>			
<u>Panel C: Health behaviors, all pupils (Grades 3-8)</u>			
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	0.98 (0.68)		

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), and the local density of Group 1 pupils within 3 km (per 1000 pupils), Group 1 pupils within 3-6 km (per 1000 pupils), Total pupils within 3 km (per 1000 pupils) and Total pupils within 3-6 km (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).

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 (0.021)	-0.013 (0.021)	0.023 (0.145)
Pupil age		-0.004 (0.006)	-0.003 (0.006)
Pupil female		-0.048** (0.024)	-0.050 (0.035)
Verbal commitment intervention indicator * Age			-0.003 (0.010)
Verbal commitment intervention indicator * Female			0.005 (0.006)
Social links, other controls	Yes	Yes	Yes
Number of observations (pupils)	3164	3164	3164
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 than 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 km, 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).