

# Deworming as HIV Prevention for Young Women: Evidence from Zimbabwe\*

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## Abstract

Nearly one-third of new HIV infections in Sub-Saharan Africa occur in young women, largely because their partners are from high-prevalence groups. Since marriage market matching is shaped by human capital, which is influenced by childhood health, can deworming girls lower their chances of contracting HIV as young women? To answer this question, I study Zimbabwe's school-based deworming program (2012-17), which substantially reduced rates of urogenital schistosomiasis. Using a difference-in-differences design, I find that 3 years after it began, young women's HIV prevalence fell 2.7 percentage points (p.p., 44 percent) more in high-schistosomiasis districts. Human capital's effects on marriage market matching appear to explain the results: young women's secondary school attendance rose 6.0 p.p. (9 percent), and they had less age-disparate and fewer sexual partners. These results show that a cheap treatment for a common childhood disease can also slow an expensive and deadly pandemic, substantially increasing deworming's estimated benefits.

*Keywords:* Childhood Health, Human Capital, Marriage Markets, HIV

*JEL Classification:* I15, J12, J24, O15

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## 1. Introduction

Along with its terrible human toll across Sub-Saharan Africa, untreated HIV infections have also imposed devastating economic costs on this region.<sup>1</sup> The advent of antiretroviral therapy (ART), which treats HIV, and its widespread distribution have thus been nothing short of a miracle. But while its costs are far less steep than those of untreated HIV, this miracle still does not come cheaply: in 2019, ART accounted for almost two-thirds of the more than \$3 billion spent on HIV-related commodities in 34 highly-affected countries by their governments and international organizations (Oum, Carbaugh and Kates, 2021). Because people living with HIV must take ART for their entire lives, these expenses will likely remain substantial for decades, even as donor funding to combat the HIV pandemic has plateaued and fallen since the late 2000s (Kates et al., 2020).

Therefore, averting new HIV infections—or even simply *delaying* them for several years—would yield significant savings for governments of high-HIV prevalence countries and international donors.<sup>2</sup> It would be especially true for Eastern and Southern African countries, where more than two-fifths of the world’s 1.7 million new HIV infections occurred in 2019 (UNAIDS, 2020). In the case of Zimbabwe, delivering ART costs around \$175 per patient per year (Benade et al., 2021), or 15 percent of GDP per capita; with 1.23 million HIV-positive adults (almost 13 percent) and 93 percent of them receiving treatment (UNAIDS, 2022), ART costs 1.2 percent of its GDP every year.

By far, the group comprising the largest share of new infections in Zimbabwe and its neighbors—nearly one-third—is women aged 15 to 24 (UNAIDS, 2020).<sup>3</sup> Their risk

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<sup>1</sup> Individuals suffering from AIDS were more often absent from their jobs or out of work entirely (Hab-yarimana, Mbakile and Pop-Eleches, 2010; Bor et al., 2012), pushing young sons into the labor force in their place (Thirumurthy, Graff Zivin and Goldstein, 2008). Also, savings and children’s schooling decreased as life expectancy declined, even in HIV-negative households (Baranov and Kohler, 2018). As these impacts reverberated through broader economies, rates of income growth fell across the region (Tompsett, 2020).

<sup>2</sup> With a 5-percent discount rate, the present value of lifelong ART for a 20-year old who lives to age 70 and contracts the virus 5 years from now is about one-fourth lower than if she contracts it today. The relative savings are greater if the drugs become cheaper over time, as has been the case for ART, or with a lag between infection and treatment initiation, though the amounts saved are smaller in absolute terms.

<sup>3</sup> Men 15-24 are 12 percent, and the remaining men and women under 50 are each around 30 percent.

is so disproportionately high due in large part to whom they match with in marriage and dating markets (henceforth marriage markets). An especially risky but common practice is engaging in age-disparate relationships, which can provide young women with economic and psychological benefits (Leclerc-Madlala, 2008). Nonetheless, as older men have exceptionally high rates of HIV, the result is a cycle of transmission from them to young women who, as they age, eventually spread the virus to their male peers (de Oliveira et al., 2017).<sup>4</sup> It is also often the case that young women have had multiple sexual partners in their lifetimes—simultaneously or sequentially—which further increases their chances of contracting HIV (Tanser et al., 2011).

Because these marriage market outcomes for young women can be shaped by their human capital (Becker, 1991), which is in turn shaped by their childhood health (Bleakley, 2010), can improving the health of girls lower their chances of contracting HIV as young women? If so, does it in fact work by changing the characteristics and number of partners they match with as well as their sexual behaviors in those relationships? And if the childhood health improvement is an exceptionally cheap one like parasitic worm (helminth) control—as in Miguel and Kremer (2004)—how much would it change the cost-benefit analysis of deworming?

I answer these questions by studying the effects of a nationwide deworming program in Zimbabwe (2012-17), where 8 percent of women aged 15 to 24 were HIV positive when it began. As I describe in Section 2, the predominant helminth in the country at that time was *Schistosoma haematobium*, which causes the urogenital form of schistosomiasis (also known as bilharzia), a neglected tropical disease estimated to affect over 100 million people in Sub-Saharan Africa and 230 million worldwide. *S. haematobium* infection can lead to genital ulcers and morbidity limiting learning and schooling (Colley et al., 2014).<sup>5</sup>

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<sup>4</sup> Notably, this cycle initially excludes young men, who must age into it. Sub-Saharan African countries have the highest rates of age-disparate relationships in the world, especially those in West Africa (Pew Research Center, 2019). However, it is likely that Islamic practices and social mores have prevented this region's HIV epidemic from becoming as devastating as the one in Southern Africa (Gray, 2004).

<sup>5</sup> Unlike soil-transmitted helminths such as hookworm, schistosomes are transmitted via freshwater. The form of schistosomiasis is intestinal, though it was far less common in Zimbabwe.

Therefore, urogenital schistosomiasis in adolescence could affect the HIV status of young women through 3 main channels: ulcers that facilitate the virus's entry into the bloodstream (a direct health effect), knowledge acquired in school about transmission risks (a direct schooling effect), and the human capital-marriage market pathway above.

After explaining these theoretical links, I then examine the empirical relationship between urogenital schistosomiasis and HIV prior to the deworming program. Combining rates of heavy schistosome infection—the main driver of morbidity—among students in 67 of Zimbabwe's districts with Demographic and Health Surveys (DHS) data from 2005 and 2010, I find strong pre-treatment correlations between heavy infection levels and HIV prevalence among the broader population. Importantly, the patterns in correlations by age group are consistent with the importance of age-disparate relationships in explaining the schistosomiasis-HIV connection: it existed among older men, young women, and older women but not among young men, who had not yet aged into the high-HIV transmission cycle mentioned above.

To test this relationship more rigorously, I exploit the quasi-experimental variation in urogenital schistosomiasis among school-age children generated by deworming. As I describe in Section 3, rates of *S. haematobium* infection fell significantly after the first annual round of drug administration and they remained low even prior to subsequent ones. Some districts thus experienced much greater reductions than others in students' rates of urogenital schistosomiasis. I use DHS data from the two pre-deworming waves and 2015 in a difference-in-differences setup to compare pre- and post-treatment trends in outcomes across high- and low-schistosomiasis areas.

I focus on those aged 15 to 20 in each DHS wave because they were of reproductive age when surveyed, and given steep attendance declines after age 17, those in the 2015 data were the most likely to have been in school when deworming began 3 years earlier—and thus were the cohorts most exposed to treatment. To test the robustness of any findings, I then restrict the sample to ages 15 to 18 to address concerns about migration

by older members of this group. I also limit the sample to rural areas because they had much higher rates of schistosomiasis, so any results should not be driven by urban respondents. As placebo tests to enhance the credibility of attributing the findings to the effects of deworming, I compare trends among adults aged 21 to 24 when surveyed because 18- to 21-year-olds in 2012 should have been (mostly) unexposed to treatment.

I present the results in Section 4. The main finding in Section 4.1 is that three years after deworming began, HIV rates among women ages 15 to 20 fell by 2.7 percentage points (p.p.) more in high-schistosomiasis districts, which was 44 percent of their pre-treatment mean. In contrast, there were no detectable effects on young men's HIV. This pattern is again consistent with the importance of age-disparate relationships in explaining the schistosomiasis-HIV link. The results for young women are also robust to excluding older ages and urban areas, and as expected, there was no effect detected for women or men in mostly untreated older cohorts, further suggesting that the effects of childhood health on young women explain the findings.

In Section 4.2, I begin to unpack the channels through which deworming reduces young women's HIV rates by examining its effects on the components of human capital. Using admittedly imperfect self-reported data on genital lesions, I find an absence of evidence for a direct health effect, as rates of ulcers and discharges do not appear to diverge following treatment.<sup>6</sup> However, the results for the education component of young women's human capital suggest that deworming clearly improves it: female secondary school attendance rates increased by 6.0 p.p. (9.0 percent) more in high-schistosomiasis districts after treatment. There is also little evidence of an effect on male attendance rates—consistent with the (lack of an) effect on schooling helping to explain the sex-specific HIV results.<sup>7</sup> In addition, the result is robust to restricting the sample to rural women ages 15 to 18, so the attendance result maps directly onto the HIV findings.

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<sup>6</sup> The concern with these data is that untrained respondents might fail to identify ulcers and discharges.

<sup>7</sup> A deworming effect on young women's secondary school attendance but on not young men's matches the findings that Baird et al. (2016) reported in their 10-year follow-up in Kenya.

I then turn to studying how deworming, most likely via its effects on schooling, may have affected the proximate causes of HIV infection for young women. The results in Section 4.3 provide some evidence of increased knowledge of HIV risk factors, as young women in high-schistosomiasis districts became 6.4 p.p. (8.8 percent) more likely to identify using condoms as a way to reduce HIV transmission. Nonetheless, deworming's effects on marriage market matching appear to be quantitatively more important. First, the share of young women in high-schistosomiasis districts in relationships with men at least 9 years older (the 75th percentile of partner age gaps) fell by over 16 p.p., which is exactly how much the share of them in relationships with 5- to 8-year age gaps (from the median to the 75th percentile) rose. There was also a 3.0-p.p. greater decline for these young women in having had 2 or more sexual partners in their lives, though this result is less precise. Second, there was a noisy 2.4-p.p. greater decline in condom use, which on the surface implies that increasing knowledge did not increase the behavior.<sup>8</sup>

Lastly, in Section 5 I conduct several cost-benefit analyses of deworming in a high-HIV prevalence country. In terms of health cost-effectiveness, I calculate that the discounted cost per disability-adjusted life year (DALY) averted fell by nearly half—from \$106 to \$57—after including the contributions of averted HIV infections among young women. I also examine the labor market returns to deworming from additional time in secondary school, which amounted to a net discounted value of \$108. In addition, given the need for high-HIV prevalence countries to bear more of the costs of combating their epidemics, I find that young women induced by deworming to avoid contracting the virus through 2015 must remain HIV negative through 2023 (an additional 8 years) for Zimbabwe's government to break even on urogenital schistosomiasis treatment as an HIV-prevention strategy. These calculations suggest that previous analyses of deworming have substantially understated its benefits, at least in Southern African countries.

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<sup>8</sup> One way to reconcile these results is by noting that if young women knew they were reducing their HIV risk by entering into less age-disparate relationships and having fewer sexual partners, the cost-benefit analysis of condom use might shift in favor of unprotected sex.

Taken together, these results contribute to several strands of the economics and public health literatures. First, I show that improving the health of girls is a cost-effective method of preventing later HIV infection when they will be at exceptionally high risk of contracting the virus as young women. This paper thus adds to the analyses of Africa’s devastating HIV epidemic applying insights from economics to one of the greatest public health crises of our time (Dupas, 2011; Robinson and Yeh, 2011; Oster, 2012; Björkman Nyqvist et al., 2018; Greenwood et al., 2019; Angelucci and Bennett, 2021).

More broadly, this paper furthers our understanding of the short- and long-run benefits of childhood health across countries and throughout history (e.g., Case, Fertig and Paxson, 2005; Maccini and Yang, 2009; Hoynes, Schanzenbach and Almond, 2016). It also shows that a health improvement later in childhood can still have important impacts for treated individuals as well as the fiscal health of governments. Therefore, it can potentially expand our view of the “better early than late” approach when it comes to such interventions (Almond and Currie, 2011; Currie and Almond, 2011; Gertler et al., 2014).

In particular, this paper adds a *quickly-realized* benefit in a novel domain to incorporate into justifications for childhood health interventions, especially deworming (Miguel and Kremer, 2004; Bleakley, 2007; Baird et al., 2016; Ozier, 2018; Hamory et al., 2021). Because discounting substantially reduces the present value of labor market returns a decade or more in the future, averting (at least 3 years of) ART can have an important impact on cost-benefit analyses, even if there is a lag between infection and treatment initiation.<sup>9</sup> The previously unexplored set of benefits in these results expands our view of the impacts of childhood health, raising the possibility that focusing exclusively on labor market returns understates the true value of interventions in this area.

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<sup>9</sup> There has been recent controversy over studies of deworming—see the discussion in Aiken et al. (2015), Davey et al. (2015), Hamory Hicks, Kremer and Miguel (2015), and Hargreaves et al. (2015)—and the relevant Cochrane Review is skeptical of the body of evidence on its health effects—see the discussion in Taylor-Robinson et al. (2019) and Croke et al. (2022). But even when taking the approach of GiveWell (2017), which continues to recommend donating to deworming charities because its extremely low costs are outweighed in expectation by uncertain but potentially substantial benefits, the HIV effects found in this paper help to increase the magnitude of the benefit side of the ledger, if not its certainty.



Finally, this paper highlights the role of childhood health in marriage markets, especially in a non-Western context (Chiappori, 2020). The importance of human capital for marriage and fertility has largely been established by studying education (Aaronson, Lange and Mazumder, 2014; Duflo, Dupas and Kremer, 2015; Chiappori, Costa Dias and Meghir, 2018), but health is also a major component of human capital and can affect decisions regarding the latter (Bleakley and Lange, 2009; Rocha and Soares, 2010). My results suggest that improving childhood health can improve inputs into and outcomes of marriage market matching, which has substantial consequences for women's welfare in the developing world (Ashraf et al., 2020; Corno, Hildebrandt and Voena, 2020).

## 2. Urogenital Schistosomiasis and HIV in Zimbabwe

In this section, I first describe how urogenital schistosomiasis was by far the most common helminth infection among Zimbabwean students, and likely the one causing the most morbidity. I then review the relevant information regarding this morbidity and hypothesize links between it and HIV. Lastly, I present the positive correlations between *S. haematobium* infection intensity and HIV prevalence in Zimbabwe prior to deworming, and I discuss what they suggest about the channels underlying this relationship.

### 2.1. Pre-Deworming Data on Helminth Infections

Midzi et al. (2014) reported results from the 2010-11 national helminthiasis survey conducted by the national government. Tests of over 13,000 students from nearly 300 schools across the country showed that rates of infection by either urogenital or intestinal schistosomes (above 22 percent) were over 4 times higher than rates for any soil-transmitted helminth (under 6 percent), with much greater rates of both helminthiases in rural areas. Of the two forms of schistosomiasis, urogenital (18 percent) was more than twice as common as intestinal (under 8 percent).

I focus specifically on the rate of heavy infection, which is the main determinant of



helminthiasis morbidity in general (Hotez et al., 2006) and especially so for urogenital schistosomiasis (Wiegand et al., 2021).<sup>10</sup> Because heavy infection rates for *S. haematobium* were nearly 20 times higher than for *S. mansoni* (5.5 percent versus 0.3 percent), it is clear that morbidity from urogenital schistosomiasis would have been the most affected by deworming. Therefore, I use these rates as the relevant measure of the pre-treatment helminth burden among school-age children. Figure A1 shows the variation in heavy *S. haematobium* infection in schools across districts (second-level administrative units) in 2010-11. The eastern half of the country had most of the urogenital schistosomiasis morbidity, but there was still significant variation within provinces.

## 2.2. Urogenital Schistosomiasis Morbidity

Colley et al. (2014) provided a thorough overview of human schistosomiasis that I briefly summarize here. Adult male and female worms live for 3 to 10 years in the veins near the bladder of an infected person, where they mate and release fertilized eggs. These eggs either become trapped in the tissue of the urogenital system or are discharged in urine into the environment, where those reaching freshwater will hatch and undergo a process that allows them to infect other humans.<sup>11</sup>

The cause of morbidity from schistosome infection is the immune response to eggs lodged in tissue.<sup>12</sup> In particular, clusters of white blood cells surround trapped eggs and generate inflammation within the urogenital system. Those infected for the first time at later ages than usual (most often adult travelers to endemic regions) can develop symptoms of acute schistosomiasis including fever and malaise lasting for 2 to 10 weeks.

Children born in endemic areas rarely experience this type of morbidity and instead

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<sup>10</sup> Heavy *S. haematobium* infection is defined as having visible haematuria (blood in urine) or at least 50 eggs per 10 milliliters of urine. A lower egg count constitutes a light infection (WHO, 2002).

<sup>11</sup> Specifically, the hatched eggs release swimming larvae that can infect certain snail species. These snails later release a different form of larvae that can penetrate the skin of humans coming into contact with the body of water. They then migrate to the target organs and mature into adult worms.

<sup>12</sup> This aspect of schistosomiasis is notably different from other helminthiases, in which morbidity is driven by worms consuming the host's blood and nutrition.

suffer from chronic schistosomiasis. One of its most commonly discussed symptoms is anemia of inflammation—generally a mild to moderate anemia in which there are too few red blood cells but their size and hemoglobin content are normal (Weiss, Ganz and Goodnough, 2019)—but whether it results from schistosomiasis alone or polyparasitic infections is not certain (Friedman, Kanzaria and McGarvey, 2005). Chronic urogenital schistosomiasis can also cause organ-specific symptoms like blood in urine (haematuria, often mistaken for menses) and painful urination. In females, *S. haematobium* eggs can become lodged in the genital tract and result in lesions (i.e., genital ulcers).

### 2.3. Theoretical Links to HIV

There are several important ways in which these symptoms could affect HIV status through their impacts on the components of childhood human capital. Figure 1 summarizes the existing hypotheses regarding human capital and HIV, highlights the novel one examined in this paper, and specifies how urogenital schistosomiasis would factor into these channels. First, the public health literature focuses primarily on the disease’s direct health effect: namely, ulcers caused by *S. haematobium* eggs lodged in the female genital tract that can facilitate the virus’s entry into the bloodstream (Kjetland et al., 2006).<sup>13</sup>

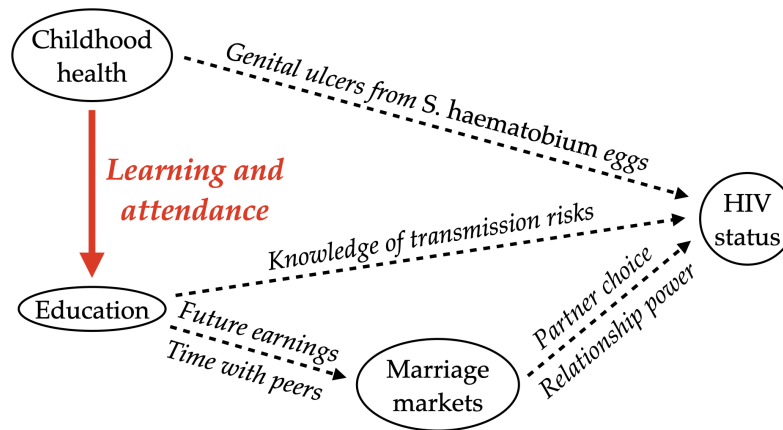
Separately, economists and public health scholars have linked the education component of human capital to HIV through knowledge of how the virus spreads and schooling’s impacts on sexual behaviors (Case and Paxson, 2013; Agüero and Bharadwaj, 2014; Behrman, 2015).<sup>14</sup> The idea behind the latter is that schooling heavily influences whom young women match with in marriage markets and their power in a relationship (Becker, 1991). Schooling may have such effects because of how it affects future labor market prospects (Peters and Siow, 2002), or simply because more time spent in school could

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<sup>13</sup> Urogenital schistosomiasis in men can also promote the spread of HIV: if they have both, their semen can contain more virus-hosting cells and viral RNA (Leutscher et al., 2005; Midzi et al., 2017).

<sup>14</sup> De Neve et al. (2015) showed that Botswana’s 1996 expansion of secondary schooling reduced the risk of HIV infection as adults but the census data used did not permit an investigation of the underlying channels. Also related is Baird et al. (2012), who found that cash transfers conditional on school attendance reduced the odds of HIV infection for females aged 13 to 22 by changing sexual behaviors.

**Figure 1:** Linking Childhood Human Capital to HIV via Urogenital Schistosomiasis



Notes: Black text and dashed arrows denote channels examined separately in previous research. Red bold text and solid arrow denote the novel linking of channels explored in this paper. See the text for references.

increase the share of peers in the pool of potential matches.

But in spite of the prominence of the link between childhood health and education in studies from both the developing and developed world (e.g., [Glewwe and Miguel, 2007](#); [Currie, 2009](#)), its role has not yet been explored in the context of HIV. Because better health in school-age years can increase learning and attendance (see [Bleakley, 2010](#), for a review), an increase in young women’s educational attainment due to deworming could thus improve their chances of matching with less-risky partners. These matches could also be more stable, leading these young women to have fewer total sexual partners.

## 2.4. Empirical Links to HIV

### 2.4.1. Data

Given the multiple channels through which urogenital schistosomiasis could affect HIV prevalence, I examine whether the intensities of these two diseases were in fact correlated in Zimbabwe prior to its deworming program. To do so, I compare the heavy schistosome infection data described above to HIV prevalence measured in the pre-deworming waves of the DHS (2005 and 2010; see Appendix Figure [A2a](#) for a map of survey clusters by year). In these surveys, random subsets of respondents were offered anonymized

HIV tests, and those that consented had their blood drawn. Figure A2b shows districts' HIV rates in pre-treatment years, which were highest in the south and east.

#### 2.4.2. Measuring Correlations

The visual similarity of these maps suggests a correlation between schistosomiasis morbidity and HIV prevalence. I examine this relationship formally by assigning DHS survey clusters their district's category of heavy schistosome infection and estimating

$$HIV_{i,c,t} = \alpha_{p(c)} + \gamma_t + \sum_{k=2}^4 \tau_k \times Category_{d(c)} + \mathbf{X}_i\beta + f(Lat_c, Lon_c) + \epsilon_{i,c,t}, \quad (1)$$

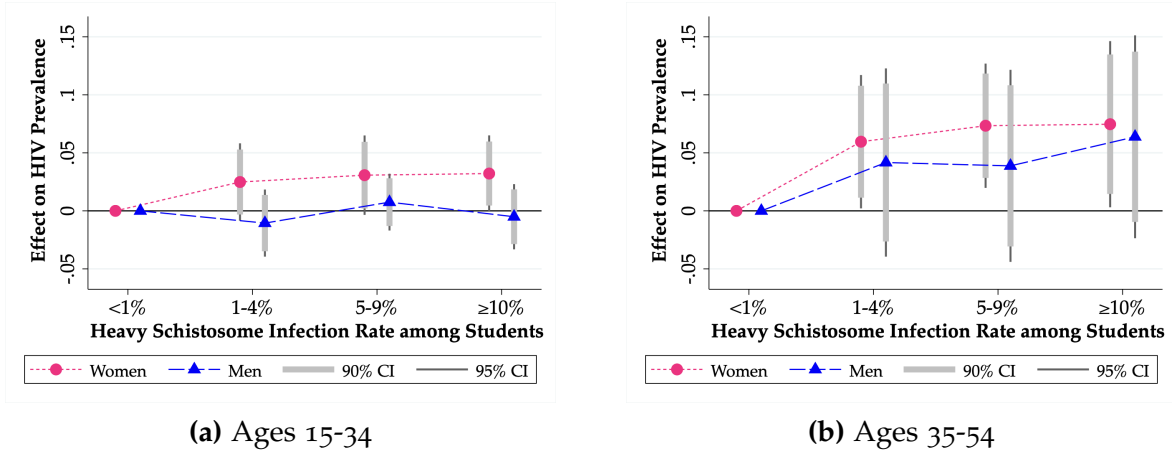
where  $HIV_{i,c,t}$  indicates whether individual  $i$  in survey cluster  $c$  in year  $t$  is HIV positive,  $\alpha_{p(c)}$  and  $\gamma_t$  are fixed effects for  $c$ 's province and the year,  $Category_{d(c)}$  is  $c$ 's district's category of heavy schistosome infection (corresponding to those in Figure A1, with 4 being the highest),  $X_i$  are individual-level controls (age and age squared),  $f(Lat_c, Lon_c)$  is a quadratic polynomial in  $c$ 's latitude and longitude coordinates, and  $\epsilon_{i,c,t}$  is the idiosyncratic error term. Due to the importance of age-disparate relationships for the spread of HIV in Southern Africa (see the discussion above), I split the data by sex and age group to determine if schistosomiasis intensity appears to interact with that transmission cycle.

The coefficients of interest are the  $\tau_k$ , which measure the effect of a district being in the given category of heavy schistosome infection relative to being in the lowest one (<1%). These estimates come from comparing individuals across districts within provinces and years after adjusting for the individual- and cluster-level controls. For inference, I cluster standard errors by the 67 districts represented in the 2005 and 2010 DHS data.

#### 2.4.3. Results

Figures 2a and 2b show the respective ecological relationships between schistosomiasis and HIV for these groups before the deworming program began. HIV prevalence increased with schistosomiasis morbidity for both younger and older women, but among

**Figure 2: Pre-Deworming Correlations between Schistosomiasis and HIV**



*Notes:* Plots show the regression-adjusted relationships between districts' categories of heavy schistosome infection for students and HIV prevalence for each age group and sex in 2005 and 2010, with coefficients estimated relative to the lowest category (<1%). Categories are taken from [Midzi et al. \(2014\)](#). Regressions control for year and province fixed effects, age, age squared, and a quadratic polynomial in latitude and longitude. Standard errors are clustered by the 67 districts in the sample. Regressions use 7,625 observations for females and 5,656 for males in (a) and 2,673 for females and 2,134 for males in (b).

men, there was only such a relationship for the older group. These patterns are what would be expected if poor childhood health contributed to the [de Oliveira et al. \(2017\)](#) HIV transmission cycle driven by age-disparate relationships, in which older men pass high infection rates to younger women who, as they age, pass them to male peers.

### 3. Empirical Strategy

While the above links between urogenital schistosomiasis and HIV are clear, they do not imply a causal relationship or shed light on the underlying channels. For more rigorous evidence in this vein, I exploit the quasi-experimental variation in heavy *S. haematobium* infection generated by deworming. Below, I describe the program, explain the difference-in-differences strategy I use to measure its impacts, and test for pre-treatment balance.

#### 3.1. Success of Zimbabwe's Deworming Program

In September 2012, Zimbabwe conducted its first round of mass administration of the antihelmintic drug praziquantel in schools across the country. Five additional rounds

followed in October 2013, January 2015, November 2015, November 2016, and November 2017. To measure the program’s impact, Mduluza et al. (2020) selected a cohort of children across 35 sentinel schools with a range of pre-treatment *S. haematobium* prevalence rates to follow over time. The authors tested their urine for eggs and haematuria immediately prior to each round as well as 6 weeks later.

They found that the deworming program had rapid and sustained successes. After just one round of drug administration, the prevalence of *S. haematobium* infection in the cohort of tracked students fell from about one-third to around 1 percent. Some were reinfected between rounds: at the start of the second and third, rates had risen back to around 5 percent. But each time, drug administration significantly lowered infection rates again, and they never exceeded 2 percent at any subsequent point in the study.

### 3.2. Difference-in-Differences Strategy

The variation in pre-treatment heavy *S. haematobium* infection rates combined with the nationwide success of Zimbabwe’s deworming program caused some districts to have substantially larger improvements in childhood health than others. I use this variation to identify its effects because deworming began at the same time in all districts. Specifically, I compare in each period those with high or the highest pre-program rates of heavy schistosome infection among schoolchildren (at least 5 percent) to those with low or moderate pre-program rates (below 5 percent), as heavy infection drives morbidity.<sup>15</sup> Forty-three districts were thus in the high category and 28 were in the low one.

To make these comparisons, I estimate the dynamic two-way fixed effects (TWFE) specification

$$y_{i,c,t} = \alpha_{d(c)} + \gamma_t + \sum_{k \in \{2005, 2015\}} \tau_k \times (\mathbf{1}[t = k] \times High_{d(c)}) + \mathbf{X}_i \beta + f(Lat_c, Lon_c) + \epsilon_{i,c,t}, \quad (2)$$

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<sup>15</sup> Zimbabwe’s treatment guidelines were for (near-)universal drug administration among students where there was at least 5-percent heavy schistosome infection rates. Below that threshold, drug administration was to be much more targeted (Midzi et al., 2014).

which is similar to equation (1) except in the following respects:  $\alpha_{d(c)}$  is a fixed effect for the district  $d$  in which a cluster lies,  $High_{d(c)}$  indicates whether a district had high or the highest pre-program schistosomiasis morbidity (i.e., heavy infection rate above 5 percent), and  $1[t = k]$  indicates whether an observation is from the given year  $k$ .

The coefficients of interest are the  $\tau_k$  in 2005 and 2015, which measure the difference in an outcome between high- and low-schistosomiasis districts in the given year relative to the size of that difference in 2010 (the omitted year). An insignificant estimate in both the statistical and economic senses for  $\tau_{2005}$  implies that pre-program trends across these districts evolved in parallel, and a significant  $\tau_{2015}$  indicates that the improvements in young adults' health as children resulting in outcomes diverging. For inference, I cluster standard errors by district because treatment was assigned at that level.

As school-based helminth control began in 2012 and secondary school attendance drops sharply after age 17 in Zimbabwe, the reproductive-age cohorts that were (mostly) exposed to the program for any length of time before the post-treatment DHS survey were between ages 15 and 20 in 2015 (i.e., ages 12 to 17 in 2012).<sup>16</sup> Therefore, I expect that the clearest effects should arise among young adults in this age range. But because older cohorts might have migrated from their districts of childhood residence, I also examine the robustness of these results by limiting the oldest age to 18. As another plausibility check, given that rural areas had higher schistosomiasis prevalence, I present results for this reduced age range excluding urban areas as well. To increase the credibility of any findings, I also examine outcomes for young adults ages 21 to 24 (i.e., ages 18 to 21 in 2012) as a placebo test since they were close in age but almost entirely untreated, and thus should not have directly benefitted from the deworming program.<sup>17</sup>

<sup>16</sup> Prior to the program, just under half of girls and just under three-fifths of boys aged 17 attended school. Rates for 18-year olds are half of those attendance figures.

<sup>17</sup> Untreated groups still may have benefitted indirectly because nearby schoolchildren were no longer shedding *S. haematobium* eggs (e.g., Miguel and Kremer, 2004). But given these adults' ages when the program began, improved childhood health should not explain any benefit accruing to them, so there should not be evidence consistent with the education-related channels in Figure 1. Instead, any effect on HIV would likely arise from having fewer genital ulcers as adults (e.g., Kjetland et al., 2006).



To improve the precision of the results, I also estimate the static TWFE specification

$$y_{i,c,t} = \alpha_{d(c)} + \gamma_t + \tau \times (Post_t \times High_{d(c)}) + \mathbf{X}_i\beta + f(Lat_c, Lon_c) + \epsilon_{i,c,t}, \quad (3)$$

where  $Post_t$  indicates whether an observation is from 2015. The difference between equation (2) and this one is that the coefficient of interest  $\tau$  is now estimated relative to the pooled pre-treatment periods, which can help to reduce noise in the data. However, it comes at the cost of reducing the flexibility of the estimates, though with only 3 periods (2 prior to deworming and 1 afterward), in practice the reduction should only be minor. I view the estimates from these specifications as complementary because they each address a potential weakness in the other.

### 3.3. Descriptive Statistics and Pre-Treatment Balance

Before testing for a post-deworming divergence in trends, I make pre-treatment comparisons among individuals aged 15 to 20 across high- and low-schistosomiasis districts in Appendix B1. As expected from Figure 2, Appendix Table B1 shows that prevalence was significantly greater in high-schistosomiasis districts for young women (6.4 versus 4.5 percent) but not young men (2.7 versus 3.1 percent). High-morbidity districts are also much farther east (as discussed in Section 2.1) and respondents living there were somewhat more likely to be in wealthier households, attending school, and in rural areas, but these three differences are not statistically significant.<sup>18</sup> Of some concern are young men's HIV test refusal rates (12.8 versus 16.4 percent), which are not quite statistically different but raise the specter of selection into testing.

Nonetheless, the broad balance between groups suggest that young women's HIV was most clearly associated with schistosomiasis morbidity. As a result, it plausibly explains their pre-treatment HIV differences and further suggests that deworming—not other characteristics or selection—would drive any results (Kahn-Lang and Lang, 2020).

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<sup>18</sup> The higher rural share is a more substantive difference (16.9 versus 21.9 percent urban for young women, 12.8 versus 16.4 percent urban for young men), but it is to be expected given greater schistosomiasis prevalence in rural areas (Midzi et al., 2014).

## 4. Results

### 4.1. HIV Prevalence

I first examine the evolution of HIV prevalence among young adults across high- and low-schistosomiasis districts and present the results in Table 1. For women ages 15 to 20, Panel A Column (1) shows that HIV differences across these areas evolved largely in parallel between 2005 and 2010, but diverged after deworming began. There was an estimated 3.4-p.p. greater decline in HIV prevalence among young women in high-schistosomiasis districts, which is both precisely estimated and economically significant (55 percent of their pre-deworming rate). In Panel B, the static estimate of a 2.7-p.p. greater decline is less precise and slightly smaller (44 percent of the pre-treatment mean), as it incorporates the estimated 1.4-p.p. relative difference in 2005 into the comparison. Nonetheless, the results are quite similar and suggest that childhood health mattered substantially for young women's HIV.

In contrast, there is little evidence in Column (2) that deworming had a similar effect among men ages 15 to 20. Indeed, the post-treatment dynamic estimate is of a positive effect of deworming on their HIV status (1.6 p.p., or 53 percent of their pre-treatment mean). But the estimate is notably both imprecise and smaller than the relative difference in 2005 (2.7 p.p.), raising the possibility that trends in young men's HIV in low-schistosomiasis districts are not a suitable counterfactual for those in high-morbidity ones. In addition, when pooling the pre-treatment periods for the static specification, the estimate is effectively null (0.2 p.p.). It is thus difficult to conclude that deworming impacted HIV prevalence in either direction for young men.

#### 4.1.1. Robustness and Credibility

As discussed in Section 3.2, maximizing power by including all respondents ages 15 to 20 comes at the cost of the results potentially being driven by those at the upper end

**Table 1:** Effect of Deworming on Young Adults' HIV Prevalence

	Exposed to Deworming				Placebo	
	Ages 15-20		Women 15-18		Ages 21-24	
	Women (1)	Men (2)	All (3)	Rural (4)	Women (5)	Men (6)
<i>Panel A. Dynamic Estimates</i>						
2005 × High	-0.014 (0.018)	0.027 (0.016)	-0.019 (0.018)	-0.013 (0.019)	-0.010 (0.028)	0.027 (0.041)
2015 × High	-0.034 (0.016)	0.016 (0.018)	-0.041 (0.020)	-0.040 (0.021)	-0.009 (0.029)	-0.007 (0.030)
<i>Panel B. Static Estimates</i>						
Post × High	-0.027 (0.016)	0.002 (0.012)	-0.031 (0.019)	-0.034 (0.017)	-0.004 (0.029)	-0.020 (0.024)
Observations	4,309	4,126	3,011	2,499	2,435	1,559
Districts	71	71	71	54	71	70
Pre-Deworming Mean (High=1)	0.062	0.030	0.050	0.044	0.147	0.057

of this range (who may have migrated) or those in urban areas (where schistosomiasis was far less common). I address these concerns in Columns (3) and (4). The former shows that after restricting the sample to those ages 15 to 18, the estimated decline in HIV prevalence in high-schistosomiasis districts after deworming was 4.1-p.p. larger in the dynamic specification and 3.1-p.p. larger in the static specification (82 and 62 percent of their pre-treatment rates). However, the static estimate is somewhat imprecise.

I then further restrict the sample to respondents in rural areas. In Column (4), the additional estimated decline in 2015 decreases slightly to 4.0 p.p. while the static estimate increases to 3.4 p.p. (91 and 77 percent of the pre-deworming mean) because there was less of a relative difference in 2005. Both of these effects are also precisely estimated. Taken together, these findings suggest that the HIV results for young women are robust to concerns that they might be driven by migration or urban areas.

Lastly, I study trends in HIV prevalence among women and men ages 21 to 24, who were slightly too old in 2012 to have been (mostly) exposed to deworming in secondary school. Consistent with the expectation of a placebo test, Column (5) shows that there

was no divergence in these women's HIV trends across high- and low-schistosomiasis districts. Importantly, both the dynamic estimate (-0.9 p.p) and static estimate (-0.4 p.p.) are quite small relative to the much higher pre-treatment mean (14.7 p.p.). In Column (6), the 2015 estimate for men (-0.7 p.p.) is also small, though as in Column (2), there is an equally large but noisier difference in pre-treatment trends that makes drawing inferences difficult and distorts the static estimate (-2.0 p.p.). Nonetheless, the results of this placebo test help to strengthen the case that the HIV effect for young women exposed to deworming in secondary school is indeed due to this treatment.

#### ***4.2. Components of Childhood Human Capital***

I then turn to studying the channels that may explain the link between childhood human capital and young women's HIV prevalence, which are summarized in Figure 1.

##### ***4.2.1. Health: Urogenital Lesions***

The hypothesis in the public health literature is that the schistosomiasis-HIV relationship should arise through genital ulcers: these lesions offer the virus a direct pathway into a woman's bloodstream, and the semen of HIV-positive men with urogenital schistosomiasis may transmit the virus more easily (Leutscher et al., 2005; Kjetland et al., 2006; Midzi et al., 2017). The DHS attempts to measure genital lesions by asking respondents whether they have had a genital ulcer or a genital discharge in the last year, which may be imperfect measurements because providing an accurate answer requires identifying each correctly. With this caveat, I use these answers to study the contribution of urogenital lesions caused by schistosomiasis to young women's HIV.

Table 2 Columns (1) and (2) show the results for genital ulcers and discharges. For the former, the 2015 estimate in Panel A is of a 1.0-p.p. greater decrease for women ages 15 to 20 in high-schistosomiasis districts, which is 56 percent of pre-treatment rates. However, it has very wide confidence intervals and is slightly smaller than the 2005

**Table 2:** Effect of Deworming on Components of Childhood Human Capital

	Health: Urogenital		Education: Attending School			
	Women 15-20		Ages 13-18		Rural Women	
	Ulcer (1)	Discharge (2)	Women (3)	Men (4)	13-18 (5)	15-18 (6)
<i>Panel A. Dynamic Estimates</i>						
2005 $\times$ High	-0.012 (0.012)	-0.003 (0.019)	-0.005 (0.037)	-0.037 (0.034)	-0.002 (0.041)	-0.030 (0.062)
2015 $\times$ High	-0.010 (0.013)	-0.006 (0.010)	0.057 (0.036)	-0.030 (0.033)	0.069 (0.035)	0.072 (0.046)
<i>Panel B. Static Estimates</i>						
Post $\times$ High	-0.004 (0.011)	-0.004 (0.009)	0.060 (0.035)	-0.010 (0.031)	0.070 (0.038)	0.087 (0.048)
Observations	4,854	4,850	6,261	6,606	5,310	3,060
Districts	71	71	71	71	54	54
Pre-Deworming Mean (High=1)	0.018	0.024	0.666	0.711	0.674	0.513

estimate (-1.2 p.p.), yielding a static estimate that is much smaller in size (-0.4 p.p.) and equally imprecise. The results for genital discharges in Column (2) present much the same picture: the dynamic (-0.6 p.p.) and static (-0.4 p.p.) estimates are both imprecise and much smaller relative to pre-treatment rates of 2.1 percent.

Taking these results at face value, it does not appear that the direct health effect of urogenital schistosomiasis contributes to the HIV result for young women, but this assessment is based on the absence of evidence in very imperfect data. Indeed, considering that at least 5 percent of students in high-schistosomiasis districts were heavily infected, pre-treatment rates of genital ulcers and discharges of around 2 percent among women aged 15 to 20 seem too low. It suggests that the issue of respondents correctly identifying these symptoms may affect the results.

#### 4.2.2. Education: Attending School

Next, I examine deworming's effects on the education component of human capital, which I measure as whether a respondent is currently attending school. I focus on

those aged 13 to 18 because deworming may have led to more time in school for those who would have dropped out. Consistent with this hypothesis, the dynamic estimate in Column (3) is of a 5.7-p.p. greater increase in female school attendance in high-schistosomiasis districts. The effect size is 8.6 percent of pre-treatment rates, though it is slightly imprecise, and the 2005 estimate of -0.5 p.p. implies little deviation from parallel trends prior to treatment. In addition, the static estimate is of effectively equal size (6.0 p.p., or 9.0 percent) and it is more precisely estimated. These results suggest that deworming had meaningful impacts on young women's secondary school attendance.

However, there is once again a stark contrast with the effect on young men. The dynamic estimate in Column (4) is of a slightly negative effect of deworming on attendance for those ages 13 to 18 (3.0 p.p., or 4.3 percent of pre-treatment rates), which is not statistically significant. It is also smaller than the dynamic estimate for 2005 (-3.7 p.p.), and the static estimate is effectively null (-1.0 p.p.). There is thus little evidence of an effect of deworming on males' secondary school attendance rates. This combination of results mirrors what [Baird et al. \(2016\)](#) found, which was that deworming led to increased secondary school attendance for females but not males.<sup>19</sup>

These attendance results are also consistent with the hypothesized role of childhood health in the HIV results. In particular, as there was an effect on young women's prevalence but not young men's, observing these same patterns in attendance suggests that the increase (or lack thereof) in schooling induced by deworming contributes to the decrease (or lack thereof) in HIV rates. Additionally, it casts some doubt on the notion that floor effects may be responsible for young men's null HIV result.

#### 4.2.3. *Robustness*

As before, it is possible that these attendance results are driven by including both urban and rural observations as well as using the widest range of secondary school ages. To

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<sup>19</sup> It could be that deworming increases young women's returns from remaining in school more than those from leaving to enter the labor force, whereas for men it has equal effects (e.g., [Bleakley, 2010](#)).

address the first concern, I limit the sample to rural observations in Column (5). Doing so increases the magnitudes of the dynamic (6.9 p.p.) and static (7.0 p.p.) estimates, which are slightly larger relative to pre-treatment attendance rates of 67.4 percent. It also increases their precision, and once again the pre-treatment relative difference was quite small (-0.2 p.p.). Estimating larger effects for rural women is reassuring: because schistosomiasis was more prevalent in these areas, the greater improvements in childhood health induced by deworming should lead to greater increases in school attendance.

I further restrict the sample in Column (5) to rural women ages 15 to 18 so the attendance and HIV results map onto each other more directly. The 2015 estimate is of a 7.2-p.p. greater increase in attendance in high-schistosomiasis areas (14.0 percent of pre-treatment rates), though it is somewhat imprecise. The static estimate is even larger (8.7 p.p.) due to the negative dynamic estimate for 2005 (-3.0 p.p.), and it is more precise. As a result, the female attendance results appear robust to the concerns mentioned above.

### **4.3. *Young Women's HIV Risks***

Lastly, I turn to whether deworming—most likely via the increase in young women's human capital—affected proximate causes of HIV infection. As summarized in Figure 1, it could occur via a direct channel (knowledge of transmission risks) or an indirect one (shaping marriage market matching). I focus on women ages 15 to 20 in the main text.

#### *4.3.1. Direct Effect: Knowledge of HIV Transmission*

I use respondents' answers to questions about HIV-safe practices to test for evidence of a knowledge effect. Specifically, I focus on whether they correctly identify having only one sexual partner and using a condom as reducing the risk of contracting the virus.<sup>20</sup> For the former, Table 3 Panel A Column (1) shows that there was a small positive estimate for 2015 (0.2 p.p.), but it is effectively null relative to the high pre-treatment rate of

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<sup>20</sup> These practices were 2 pillars of the ABC approach to HIV prevention (Abstain, Be faithful, and use Condoms). A question about abstinence reducing the risk of contracting the virus was asked only in 2005.



**Table 3:** Effects of Deworming on Young Women's HIV Risks

	Direct: Knowledge		Indirect: Marriage Market			
	Reduces Risk		Partner Age Gap		Partners	Last Sex
	1 Partner (1)	Condom (2)	$\geq 9$ Years (3)	5-8 Years (4)	$\geq 2$ in Life (5)	Condom (6)
<i>Panel A. Dynamic Estimates</i>						
2005 $\times$ High	-0.043 (0.034)	0.014 (0.062)	-0.063 (0.082)	0.011 (0.067)	0.007 (0.030)	-0.001 (0.042)
2015 $\times$ High	0.002 (0.031)	0.071 (0.039)	-0.195 (0.087)	0.171 (0.102)	-0.026 (0.021)	-0.025 (0.039)
<i>Panel B. Static Estimates</i>						
Post $\times$ High	0.024 (0.027)	0.064 (0.041)	-0.164 (0.068)	0.166 (0.080)	-0.030 (0.020)	-0.024 (0.035)
Observations	4,679	4,677	1,308	1,308	4,861	1,778
Districts	71	71	70	70	71	70
Pre-Deworming Mean (High=1)	0.817	0.724	0.267	0.407	0.075	0.097

Notes: Women ages 15 to 20.

correct answers (81.7 percent). The static estimate in Panel B is much larger (2.4 p.p.), but its magnitude is due to the non-trivial negative estimate for 2005 (-4.3 p.p.). It is thus difficult to say that deworming led to more awareness of monogamy's protective effects.

On the other hand, the evidence for greater knowledge of condom use as an HIV-safe practice is much stronger. In Panel A Column (2), the 2015 estimate is of a 7.1-p.p. larger increase in answering this question correctly in high-schistosomiasis districts, which is 9.8 percent of their pre-treatment rate. The 2005 estimate is also much smaller (1.4 p.p.), suggesting there were only small pre-treatment differences in trends, but it still makes the static estimate in Panel B smaller (6.4 p.p.) and less precise. Nonetheless, it appears that deworming girls increased their likelihood as young women of knowing that condoms protect against HIV, which is plausibly linked to their greater school attendance.

#### 4.3.2. Indirect Effect: Marriage Market Behaviors

Next, I study whether deworming affected young women's HIV risk by changing whom they matched with in marriage markets as well as their condom use in those rela-

tionships. The first dimension of matching outcomes that I examine is the age gap between partners, which has played a major role in Southern Africa's HIV epidemic (Leclerc-Madlala, 2008; de Oliveira et al., 2017). Measuring it as the man's age minus the woman's, I create an indicator variable for whether she is in a relationship with an age gap at or above the 75th percentile (9 years), and one for whether it below that value but at or above the median (5 years).

Panel A Column (3) shows that high-schistosomiasis districts experienced a 19.5-p.p. greater decline in the share of young women with at least 9-year partner age gaps after deworming began, which is very large relative to the pre-treatment rate (26.7 percent) and precisely estimated. Because the 2005 coefficient is non-trivial (-6.3 p.p.), the static estimate in Panel B is smaller (-16.4 p.p.) but still economically significant and precisely estimated. Interestingly, the 2015 and static estimates for 5- to 8-year age gaps in Column (4) are nearly identical in size and precision to those in Column (3) but have the opposite sign (17.1 p.p. and 16.6 p.p.). These results imply that deworming induced young women with the largest partner age gaps to shift into relationships with smaller ones, consistent with the idea that human capital contributes to the prevalence of this important HIV risk factor in Southern Africa.

The second dimension of marriage market matching that I study is whether a respondent has had 2 or more sexual partners in her life. In Panel A Column (5), the 2015 estimate is of a 2.6-p.p. greater decline in this HIV risk factor in high-schistosomiasis districts following deworming. This effect is large relative to pre-treatment rates of 7.5 percent, though it is imprecisely estimated. The static estimate in Panel B is larger (-3.0 p.p.) due to the slightly positive 2005 effect and is more precise, but it is still statistically insignificant at conventional levels. Nonetheless, these results are suggestive of deworming reducing another important HIV-risky behavior in marriage markets.

Lastly, I investigate whether respondents reported using condoms in their most recent sexual intercourse. At first glance, there is a surprising contrast between the knowledge

results in Column (2) and actual condom use in Column (6): the dynamic and static estimates are of equally large negative effects on young women's condom use in high-schistosomiasis districts (-2.5 p.p. and -2.4 p.p.). While they are not precisely estimated, they are still meaningfully large relative to pre-treatment rates of 9.7 percent. However, it is possible to reconcile these findings in the following manner: if these young women did know more about HIV transmission and consciously chose safer and fewer partners, then the costs of condom use may have outweighed its reduced HIV-prevention benefits.

#### 4.3.3. *Robustness*

In Appendix B2, I test the robustness of the results on young women's HIV risks. Appendix Table B2 shows that for the most part, restricting the sample to rural women ages 15 to 18 has only minor impacts on the marriage market estimates. One change of note is that after the sample size in Columns (3) and (4) falls by nearly two-thirds to less than 500, the partner age gap effects are much noisier (especially for age gaps of 9 or more years), though the substantive conclusion remains the same. Conversely, the results in Columns (5) and (6)—respondents having had 2 or more sexual partners in their lives and having used a condom in their most recent sexual intercourse—become larger and more precise than in Table 3.

Another concern regarding the marriage market outcomes is that deworming may have also affected the rates at which young women enter partnerships. While the DHS data do not contain information on how many unions a respondent has been in, I can observe whether they are still single (i.e., never entered a union). Table B3 shows that for rural women ages 15 to 18, trends in their marriage rates did not materially change across high- and low-schistosomiasis districts after deworming. Thus, the marriage market results above are unlikely to have been driven by an effect of deworming on rates of entering marriage or cohabitation.

## 5. Cost-Benefit Analyses

Finally, I perform several simple cost-benefit analyses of deworming in a high-HIV prevalence country. I mirror two of the approaches in Miguel and Kremer (2004): health cost effectiveness (using disability-adjusted life years, or DALYs), and the expected labor market returns from additional schooling. Given the Zimbabwean government's need to bear a greater share of the costs of combating the epidemic (Kates et al., 2020), I also calculate how long the young women whom deworming induced to avoid contracting the virus must remain HIV negative for it to breakeven on its investment.

In these calculations, I assume that only those with heavy schistosome infections suffered morbidity (Wiegand et al., 2021), and that treatment led to a 7-p.p. greater decline in heavy infection rates in high-schistosomiasis districts.<sup>21</sup> I compute the costs and benefits from the perspective of 2012 (when the deworming program began), though I convert all values to 2022 US dollars. I also use a 5-percent discount rate.

### 5.1. Health Cost Effectiveness

Ndeffo Mbah et al. (2013) estimated that the recommended dose of praziquantel cost the equivalent of \$0.10 per student per year, and that administering the program cost the equivalent of \$0.27 per student per year. Thus, from the perspective of 2012, the discounted value of 3 years of deworming was \$1.06 per student treated. To calculate the rate of DALYs averted directly from reducing schistosomiasis and indirectly from reducing HIV infection, I follow the authors and use disability weights of 0.05 for the former and 0.167 for the latter.<sup>22</sup> I also assume that the additional 2.7-p.p. reduction in

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<sup>21</sup> When assigning districts the midpoint of their heavy schistosome infection category and top-coding the highest group at 10 percent, the difference in the pre-deworming average heavy infection rate across high- and low-schistosomiasis districts was just under 7 percent.

<sup>22</sup> The Global Burden of Disease Collaborative Network (2020) estimates disability weights due to moderate anemia from schistosomiasis of 0.052, and those from several other symptoms (e.g., dysuria) to be 0.011. King, Dickman and Tisch (2005) called for chronic schistosomiasis's values to be between 0.02 and 0.15. The HIV weight I use from Ndeffo Mbah et al. (2013) is for infected individuals receiving ART.

HIV lasted only until 2015, and that 9 years (the lag between infection and AIDS) pass before they initiate ART.

Only considering direct effects of the reduction in heavy schistosome infections yields a discounted value of 0.01 DALYs averted per student treated over the 3-year period, which means the cost per DALY was just under \$106. Including indirect effects on HIV—a 2.7-p.p. additional decline, which averted a discounted value of 0.0087 DALYs per student treated—the cost per DALY falls by nearly half to less than \$57. It is thus clear that failing to account for deworming’s effects on HIV substantially understates its health cost effectiveness.

## *5.2. Returns to Schooling*

The private benefits of deworming also include young women’s returns from the 0.6-p.p. greater increase in secondary school attendance 3 years later, net of their opportunity costs. In line with the approach of Miguel and Kremer (2004), I make the following assumptions: the rate of return to secondary schooling is 19 percent (Bigsten et al., 2000), of which 40 percent is accounted for by years of schooling; wages are 60 percent of 2012 GDP per worker of \$2,707 (World Bank, 2022); individuals spend 40 years in the labor force after secondary school; and wages do not grow over time. In addition, I assume that a young woman leaving secondary school due to heavy schistosome infection is as productive as the average worker.<sup>23</sup> With these parameters, the discounted value of the increased earnings from an additional 0.18 years of secondary school minus the opportunity cost was thus equivalent to just below \$108.

## *5.3. Government Finances*

However, policymakers may be more immediately concerned with how long it takes for deworming as an HIV-prevention strategy to break even in terms of reducing public

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<sup>23</sup> This assumption differs substantially from the authors’ given their focus on primary school.

expenditures. Far larger than the direct costs of the program (calculated above) were the costs of the 6.0-p.p. additional increase in female secondary school attendance after 3 years. Assuming 1 teacher for 30 students (Miguel and Kremer, 2004), an additional 0.002 needed to be hired per student treated. With a teacher's annual salary in 2012 converting to \$4,681 (Mavhunga, 2012), the discounted value of hiring that many immediately after deworming and paying them for 3 years was equivalent to \$26.77. Therefore, the total discounted costs equaled \$27.83 per student treated.

The value of the reduction in ART outlays depends on how long the 2.7 p.p. of young women in high-schistosomiasis districts induced by deworming to avoid the virus for 3 years can continue to do so. With the costs of delivering ART at \$175 per patient per year (Benade et al., 2021) and assuming a 9-year lag between infection and ART initiation, the breakeven point is reached if they stay HIV negative for another 8 years (through 2023), or 11 years total.<sup>24</sup> Because the results in Section 4 show that marriage market matches for young women changed substantially, it is very possible that deworming reduced their long-run HIV risk to this extent.

## 6. Conclusion

These results show the importance of childhood health in reducing the spread of HIV in Sub-Saharan Africa, especially for the young women who are at great risk of contracting the virus. The effect appears at least in part to operate through the effect of human capital on marriage market matching, as it may shift these young women toward having less age-disparate and fewer partners. How exactly it occurs is unclear: it could be that they simply spend more time around similarly-aged men in the classroom, or it may result from improved labor market opportunities reducing their economic reliance on older men, or both.

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<sup>24</sup> Alternatively, initiating ART immediately after becoming HIV positive reduces it to 3 additional years (through 2018), or 6 years total.

Additionally, these results provide evidence of a novel benefit from controlling neglected tropical diseases, and helminthiases in particular. They show that along with its other important effects, a cheap intervention against a common childhood infection in a high-HIV prevalence country can also help combat one of the modern world's deadliest diseases. The fact that it can do so in an exceptionally cost-effective manner is also significant: donor funding to combat the global HIV pandemic has continued to decline, so the governments of some of the poorest countries in the world must bear a greater share of these costs moving forward.



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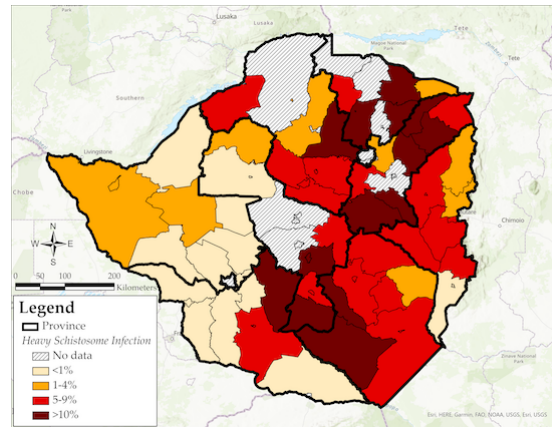
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## Appendix A. Additional Figures

### A1. Geographic Distribution of Heavy Schistosome Infection

**Figure A1: Heavy Schistosome Infection [7]**

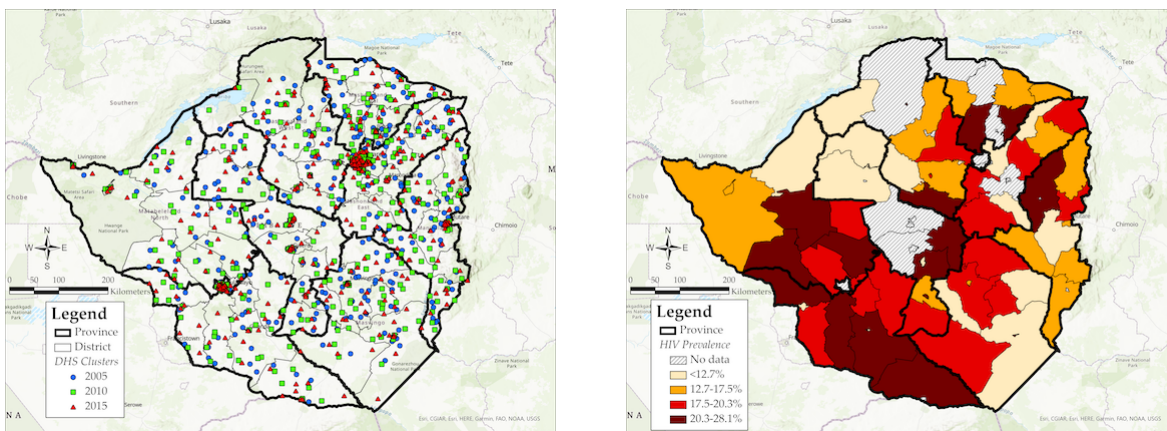


Heavy *S. haematobium* Infection, Students

*Notes:* Map shows the prevalence of heavy schistosome infection among students at the district level, with darker colors representing higher values. Ranges of heavy schistosome infection rates among students are taken from [Midzi et al. \(2014\)](#) and correspond to low (15 districts), moderate (13), high (27), and highest morbidity (16).

### A2. DHS Data: Clusters and HIV Prevalence

**Figure A2: DHS Clusters and HIV Prevalence [10]**



(a) Georeferenced DHS Survey Clusters

(b) HIV Prevalence, Ages 15-49

*Notes:* The left panel shows the locations of survey clusters in the 2005 (blue circles), 2010 (green squares), and 2015 (red triangles) waves of the DHS. The right panel shows HIV prevalence calculated from the 2005 and 2010 DHS surveys using blood test results from 14 and 774 respondents in each district (median: 209). Levels of prevalence are grouped into quartiles.

## Appendix B. Additional Tables

### B1. Comparing High- and Low-Schistosomiasis Districts prior to Deworming

**Table B1:** Descriptive Statistics and Balance Tests [15]

	Women 15-20			Men 15-20		
	Low (1)	High (2)	Difference (3)	Low (4)	High (5)	Difference (6)
<i>Panel A. Main Outcome</i>						
HIV Positive	0.045	0.064	0.019 (0.010)	0.031	0.027	-0.004 (0.008)
Observations			2,780			2,575
Clusters			67			66
<i>Panel B. Predetermined</i>						
Age	17.404 (1.718)	17.383 (1.704)	-0.020 (0.067)	17.325 (1.615)	17.375 (1.640)	0.049 (0.074)
Latitude	-18.861 (1.284)	-19.033 (1.479)	-0.172 (0.378)	-18.931 (1.313)	-18.971 (1.448)	-0.040 (0.385)
Longitude	29.765 (1.980)	31.158 (0.977)	1.393 (0.472)	29.821 (1.989)	31.184 (0.972)	1.363 (0.483)
Observations			3,583			3,658
Clusters			67			67
<i>Panel C. Socioeconomic</i>						
Asset Index	2.718 (1.411)	2.864 (1.271)	0.146 (0.272)	2.589 (1.322)	2.825 (1.165)	0.236 (0.239)
Attending School	0.361	0.386	0.025 (0.023)	0.464	0.512	0.048 (0.042)
Urban	0.219	0.169	-0.050 (0.101)	0.157	0.125	-0.032 (0.087)
Observations			3,583			3,658
Clusters			67			67
<i>Panel D. Selection</i>						
Refused HIV Test	0.112	0.108	-0.004 (0.021)	0.164	0.128	-0.036 (0.029)
Observations			3,371			3,401
Clusters			67			67

Notes: Columns (1), (2), (4), and (5) contain mean values for the respective groups with standard deviations below in parentheses for continuous variables. Columns (3) and (6) contain differences between means with standard errors clustered by district below in parentheses.

## B2. Robustness: Young Women's HIV Risks

**Table B2:** Effects of Deworming on HIV Risks for Rural Women Ages 15 to 18 [24]

	Direct: Knowledge		Indirect: Marriage Market			
	Reduces Risk		Partner Age Gap		Partners	Last Sex
	1 Partner (1)	Condom (2)	≥9 Years (3)	5-8 Years (4)	≥2 in Life (5)	Condom (6)
<i>Panel A. Dynamic Estimates</i>						
2005 × High	-0.027 (0.057)	0.089 (0.083)	-0.098 (0.088)	0.105 (0.108)	0.001 (0.026)	-0.068 (0.070)
2015 × High	0.033 (0.048)	0.107 (0.051)	-0.124 (0.088)	0.210 (0.133)	-0.038 (0.019)	-0.127 (0.053)
<i>Panel B. Static Estimates</i>						
Post × High	0.046 (0.038)	0.061 (0.053)	-0.077 (0.079)	0.160 (0.119)	-0.038 (0.020)	-0.095 (0.056)
Observations	2,616	2,616	494	494	2,751	728
Districts	54	54	54	54	54	54
Pre-Deworming Mean (High=1)	0.797	0.701	0.215	0.465	0.047	0.105

**Table B3:** Effects of Deworming on Marriage Rates [24]

	Never in Union	
	Women Ages 15-20 (1)	Rural Women 15-18 (2)
<i>Panel A. Dynamic Estimates</i>		
2005 × High	-0.037 (0.035)	-0.040 (0.046)
2015 × High	-0.046 (0.043)	-0.020 (0.039)
<i>Panel B. Static Estimates</i>		
Post × High	-0.027 (0.038)	0.000 (0.034)
Observations	5,367	3,060
Districts	71	54
Pre-Deworming Mean (High=1)	0.666	0.772