

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

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Abstract

Young women comprise nearly one-third of new HIV infections in Sub-Saharan Africa, 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) that substantially reduced rates of urogenital schistosomiasis. Using a difference-in-differences design, I find that by 2015, young women's HIV prevalence fell 2.9 percentage points (p.p., 47 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 6.8 p.p. (10 percent), and their partners were closer in age and possibly fewer in number. 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.¹ 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.² 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).³ Their risk 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

¹ Individuals suffering from AIDS were more often absent from their jobs or out of work entirely (Habyarimana, 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).

² 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.

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

(de Oliveira et al., 2017).⁴ 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).⁵ 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

⁴ 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).

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

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 2.9 percentage points (p.p.) more in high-schistosomiasis districts, which was 47 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. There is a clear positive impact on the education component for young women: their secondary school attendance rates increased by 6.8 p.p. (10 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.⁶ In addition, the result is robust to restricting the sample to

⁶ A deworming effect on young women's secondary school attendance but on not young men's matches

rural women ages 15 to 18, so the attendance result maps directly onto the HIV findings.

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 9.0 p.p. (13 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 22.1 p.p., which is almost exactly how much the share of them in relationships with 5- to 8-year age gaps (from the median to the 75th percentile) rose. Second, there was also a 2.7-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. And third, there was no significant change in condom use despite the increase in knowledge of it as an HIV-safe practice.

Next, I perform a back-of-the-envelope quantification of the contributions of the reductions in partner age gaps and genital ulcers (which I cannot observe). Using a simple static model of young women's HIV prevalence as a function of these two inputs along with assumptions taken from the literatures on schistosomiasis and the virus, I estimate that two-thirds of the 2.9-p.p. decrease in young women's HIV is due to the marriage market channel and one-third is due to the ulcer effect. The implication is that the social consequences of schistosomiasis are an important part of its relationship to HIV, suggesting that the public health literature on this topic has missed this important dimension in its near-exclusive focus on its clinical effects.

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 \$55—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 \$122. 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.

the findings that Baird et al. (2016) reported in their 10-year follow-up in Kenya.

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 (Robinson and Yeh, 2011; Oster, 2012; Anderson, 2018; Björkman Nyqvist et al., 2018; Greenwood et al., 2019; Angelucci and Bennett, 2021). Specifically, it highlights the importance of age-disparate relationships and provides insights into what can be done to reduce this risk factor (Dupas, 2011; Denton-Schneider, 2022).

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 major impacts on those treated, and that disease control can improve both population and fiscal health in developing countries (Denton-Schneider and Montero, 2022). 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.⁷ 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.

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

⁷ 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.

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 helminthiasis 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).⁸ 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.

⁸ 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).

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.⁹

The cause of morbidity from schistosome infection is the immune response to eggs lodged in tissue.¹⁰ 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 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 only slightly below normal (Weiss, Ganz and Goodnough, 2019)—though whether it results from schistosomiasis alone or polyparasitic infections is unclear (Friedman, Kanzaria and McGarvey, 2005). Chronic urogenital schistosomiasis can also cause haematuria (blood in urine, which is 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

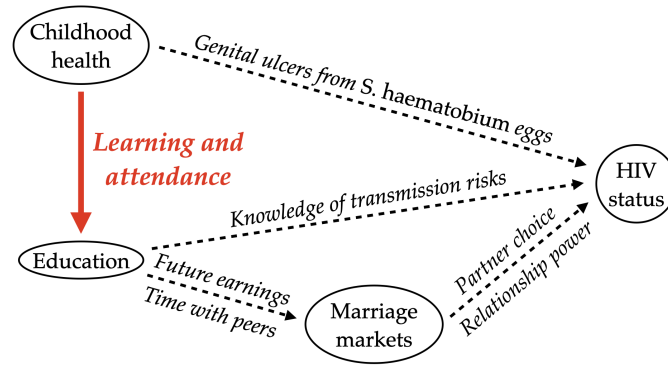
There are several important ways in which these symptoms could affect HIV status through their impacts on the components of childhood human capital. Figure 1a summarizes the existing hypotheses regarding human capital and HIV, highlights the novel one examined in this paper, and specifies how urogenital 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 facilitating the virus's entry into the bloodstream (Kjetland et al., 2006).¹¹

⁹ 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.

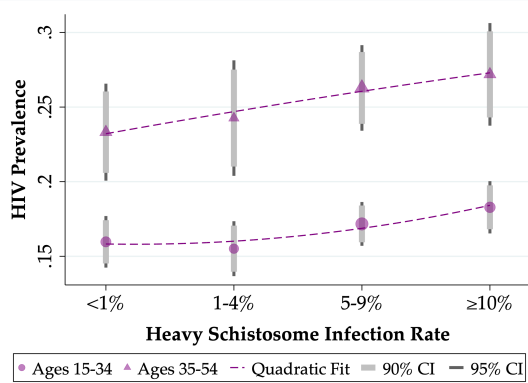
¹⁰ This aspect of schistosomiasis is notably different from other helminthiasis, in which morbidity is driven by worms consuming the host's blood and nutrition.

¹¹ 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).

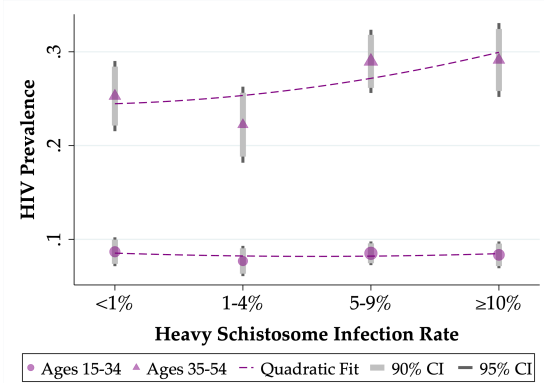
Figure 1: Hypothesized and Empirical Links between Schistosomiasis and HIV



(a) Proposed Channels



(b) Women: Schistosomiasis and HIV



(c) Men: Schistosomiasis and HIV

Notes: In the top panel, 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. In the bottom panels, plots show HIV prevalence within heavy schistosome infection ranges by sex in 2005 and 2010 for ages 15 to 34 (circles) and 35 to 54 (triangles). Ranges are taken from [Midzi et al. \(2014\)](#). Dashed lines denote fitted quadratic trends, bars under the shapes denote confidence intervals, and shape sizes reflect the number of respondents in each group.

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](#)).¹² 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 increase the share of peers in the pool of potential matches.

¹² [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

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

Given the multiple channels through which urogenital schistosomiasis could affect HIV prevalence, I study 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 [A1a](#) 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 [A1b](#) shows district-level prevalence in pre-treatment years, which was highest in the south and east.

Figures [1b](#) and [1c](#) show correlations between schistosomiasis morbidity and HIV that are visually apparent in the maps. In these panels, I plot each sex’s pre-deworming HIV prevalence within the ranges of heavy schistosome infection reported by [Midzi et al. \(2014\)](#). Due to the importance of age-disparate relationships for the spread of the virus in Southern Africa, I split the data by age group (15 to 34 and 35 to 54) to determine if schistosomiasis intensity appears to interact with that transmission cycle.

For both older and younger women, HIV prevalence clearly increases as the range of heavy schistosome infection rates increases. But among men, the same is true only for the older age group, though with larger deviations from the trend. Taken together, these patterns are what would be expected if poor childhood health contributes to the 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 ([de Oliveira et al., 2017](#)). I examine these relationships more formally in Appendix [B](#) and find that these patterns remain apparent after making comparisons within provinces and survey years and controlling for individual-level and geographic characteristics.

reduced the odds of HIV infection for females aged 13 to 22 by changing sexual behaviors.

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 antihelminthic 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 led to post-treatment childhood health improvements that were substantially larger in some districts than others. I use this variation to identify its effects, as deworming began simultaneously in all districts. Specifically, I compare DHS respondents in each period in districts with high or the highest pre-program rates of heavy schistosome infection among schoolchildren (at least 5 percent) with those in districts that had low or moderate pre-program rates (below 5 percent), as heavy infection drives morbidity.¹³ This division places 43 districts into the high category and 28 into the low one.

¹³ 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](#)).

To compare them, I estimate the dynamic two-way fixed effects (TWFE) specification

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

where $y_{i,c,t}$ is the outcome for individual i in survey cluster c in year t , $\alpha_{d(c)}$ and γ_t are fixed effects for c 's district and the year, $\mathbf{1}[t = k]$ indicates whether an observation is from the given year k , $High_{d(c)}$ indicates whether a district had a heavy schistosome infection rate above 5 percent, \mathbf{X}_i are individual-level controls (age and age squared), \mathbf{Z}_c are cluster-level controls (altitude and a quadratic in latitude and longitude), and $\epsilon_{i,c,t}$ is the idiosyncratic error term.

The coefficients of interest are the τ_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 τ_{2005} implies that pre-program trends across these districts evolved in parallel, and a significant τ_{2015} indicates that the improvements in young adults' health as children resulting in outcomes diverging. To address concerns regarding TWFE estimation, I use the procedure developed by [de Chaisemartin and D'Haultfœuille \(2020\)](#) and report bootstrapped standard errors. For inference, I cluster observations by district, as treatment was assigned at that level.

Because 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).¹⁴ 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.¹⁵

¹⁴ 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.

¹⁵ Untreated groups still may have benefitted indirectly because nearby schoolchildren were no longer shedding *S. haematobium* eggs (as in [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 channels in [Figure 1a](#). Instead, effects could arise due to

3.3. Descriptive Statistics and Pre-Treatment Balance

Before testing for a post-deworming divergence in trends, in Appendix C I compare the pre-treatment characteristics of DHS respondents ages 15 to 20 and the clusters in which they live across high- and low-schistosomiasis districts. As expected from Figure 1, Appendix Table C1 shows that HIV 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.¹⁶ Of some concern are HIV test refusal rates for young men (12.8 versus 16.4 percent), which are not quite statistically different, but refusal rates for young women are effectively equal (10.8 versus 11.2 percent).

The broad balance between groups suggests that pre-deworming differences in young women's HIV were not driven by these observable factors. As a result, it is very much plausible that schistosomiasis morbidity explains their pre-treatment differences in HIV. It also suggests that deworming—not other characteristics or selection into blood testing—would drive any results, at least for young women (Kahn-Lang and Lang, 2020).

4. Results

I first examine the evolution of young adults' HIV prevalence across high- and low-schistosomiasis districts and present the results showing a greater decline for young women in Table 1. I then study the effects of deworming on treated cohorts' human capital and the proximate causes of HIV infection in Table 2. Figure 2 presents event-study plots for the central results. Lastly, I perform back-of-the-envelope calculations in Section 4.4 to benchmark the plausibility of the HIV result and estimate the contributions of the proposed channels.

4.1. HIV Prevalence

For women ages 15 to 20, Table 1 Column (1) and Figure 2a show that HIV differences across these areas evolved largely in parallel between 2005 and 2010, but diverged after

spillovers from dewormed cohorts' behaviors (e.g., as a result of changes in younger cohorts' marriage market matches) or having fewer genital ulcers as adults.

¹⁶ 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).

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

	Positive HIV Blood Test					
	Ages 15-20		Women 15-18		Ages 21-24	
	Women (1)	Men (2)	All (3)	Rural (4)	Women (5)	Men (6)
2005 × High	-0.001 (0.022)	0.037 (0.024)	0.007 (0.024)	-0.007 (0.020)	-0.045 (0.033)	0.007 (0.034)
2015 × High	-0.029 (0.017)	0.041 (0.024)	-0.036 (0.020)	-0.040 (0.022)	-0.009 (0.027)	0.005 (0.028)
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)	0.064	0.027	0.050	0.041	0.151	0.066

Notes: Estimates and bootstrapped standard errors (in parentheses) are obtained using the procedure developed by [de Chaisemartin and D'Haultfoeuille \(2020\)](#). Observations are clustered by district. High equals 1 if a district had high or the highest pre-deworming rates of heavy schistosome infection, and 0 otherwise. Regressions control for age, age squared, altitude, and a quadratic in latitude and longitude.

deworming began. There was an estimated 2.9-p.p. greater decline in HIV prevalence among young women in high-schistosomiasis districts, which is economically significant (47 percent of their pre-deworming rate) and mostly precise. The figures in Appendix D complement these results: prior to deworming, HIV increased faster with age in high-schistosomiasis districts, but this gap closed following treatment. These findings 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 estimate is of a large positive effect of deworming on their HIV status, but the estimate is notably both imprecise and similar in magnitude to the relative difference in 2005, 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. Appendix D also shows no clear shifts in young men's HIV from before to after treatment. It is thus difficult to conclude that deworming impacted young men's HIV prevalence in either direction.

4.1.1. Robustness and Credibility

I then address the concerns that the results for young women might arise due to migration at the upper end of the age range examined or urban areas with few schistosome infections. Column (3) shows that after restricting the sample ages 15 to 18, the estimated post-deworming decline in HIV prevalence in high-schistosomiasis districts was 3.6-p.p.

larger (72 percent of their pre-treatment rates), which is a substantial divergence from the largely parallel evolution of their pre-treatment outcomes. In Column (4), I further restrict the sample to respondents in rural areas and the estimated additional decline increases in magnitude (-0.4 p.p., or 91 percent of pre-deworming rates), though it is less precise. These results are not consistent with them being driven by the factors above.

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. The post-deworming estimates in Columns (5) and (6) are small, suggesting that differences in HIV prevalence remained constant between 2010 and 2015. However, the pre-treatment estimate for women in Column (5) is large (-4.5 p.p.) and only somewhat imprecise, so the issue of suitable counterfactual trends also arises for them. In Appendix E, I conduct the same exercise for all 4-year age groups in the data without finding results similar to the one for women ages 15 to 18. As such, these placebo tests provide little evidence that deworming affected HIV prevalence among untreated cohorts.

4.2. *Childhood Human Capital*

4.2.1. *Health*

Next, I study the health components of human capital that may contribute to the link between schistosomiasis and young women's HIV (see Figure 1a). Unfortunately, as I explain in Appendix F1, the DHS data only contain proxies for urogenital lesions that almost certainly undercount them, so the null post-deworming estimates in Appendix Table F1 for these variables are not very informative or credible.¹⁷ I therefore use a different approach in Section 4.4 to assess the effect of genital ulcers due to schistosomiasis on young women's HIV rates.

The other relevant health outcome, anemia, is one that can be measured directly in the DHS data since all respondents ages 15 to 49 are offered a finger prick test. This condition is linked with schistosomiasis, albeit usually in the more mild form of anemia of inflammation (Friedman, Kanzaria and McGarvey, 2005), and it is an important input into both the returns from schooling as well as its opportunity cost (Bleakley, 2010). For women ages 15 to 20, the estimates in Table 2 Panel A Column (1) show no evidence of a substantive divergence in anemia trends following deworming. But in Panel A Column (2), anemia rates for men in the same age group decreased by 3.5 p.p. more (34 percent of their pre-treatment mean) after evolving in parallel, though the post-treatment estimate

¹⁷ Given that at least 5 percent of students in high-schistosomiasis districts had heavy infections, it is implausible that only about 2 percent of young women in these areas had urogenital lesions prior to treatment, but that share is what the DHS data report for these proxies.

Table 2: Effect of Deworming on Human Capital and Young Women’s HIV Risks

	(1)	(2)	(3)	(4)	(5)	(6)
<i>Panel A. Human Capital</i>						
	Anemic		Attending School			
	Women	Men	Women	Men	Rural Women	
	15-20	15-20	13-18	13-18	13-18	15-18
2005 × High	-0.013 (0.041)	0.007 (0.032)	-0.020 (0.037)	-0.004 (0.030)	-0.025 (0.041)	-0.022 (0.059)
2015 × High	0.022 (0.049)	-0.035 (0.035)	0.068 (0.038)	0.004 (0.037)	0.072 (0.044)	0.074 (0.063)
Observations	4,521	4,368	6,261	6,606	5,310	3,060
Districts	71	71	71	71	54	54
Pre-Deworming Mean (High=1)	0.290	0.102	0.669	0.718	0.679	0.514
<i>Panel B. HIV Risks, Women 15-20</i>						
	Know Reduces Risk		Partner Age Gap		≥2 Sex	Condom
	1 Partner	Condom	≥9 Years	5-8 Years	Partners	Last Sex
2005 × High	-0.031 (0.035)	0.004 (0.052)	-0.061 (0.098)	-0.002 (0.092)	-0.008 (0.021)	-0.012 (0.047)
2015 × High	0.011 (0.042)	0.090 (0.041)	-0.221 (0.083)	0.194 (0.113)	-0.027 (0.024)	-0.042 (0.044)
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)	0.814	0.717	0.265	0.400	0.077	0.091

Notes: Estimates and bootstrapped standard errors (in parentheses) are obtained using the procedure developed by [de Chaisemartin and D’Haultfoeuille \(2020\)](#). Observations are clustered by district. High equals 1 if a district had high or the highest pre-deworming rates of heavy schistosome infection, and 0 otherwise. Regressions control for age, age squared, altitude, and a quadratic in latitude and longitude.

is not precise. This evidence of at most an effect for young men thus suggests that anemia is not a major part of the observed schistosomiasis-HIV relationship for young women, likely because most of the anemia was mild.¹⁸

4.2.2. Education

I then turn to deworming’s effects on the education component of human capital, which I measure as whether a respondent is currently attending school. To capture the broadest range of those potentially in secondary school, I focus on ages 13 to 18. Panel A Column (3) and Figure 2b show that for women in high-schistosomiasis districts, school atten-

¹⁸ For this age group in high-schistosomiasis districts before deworming, 0.3 percent of women and 0.1 percent of men had severe anemia, 5.9 percent of women and 1.9 percent of men had moderate anemia, and 22.8 percent of women and 8.2 percent of men had mild anemia. The [Global Burden of Disease Collaborative Network \(2020\)](#) assigns disability weights to these anemia levels of 0.149, 0.052, and 0.004.

dance increased 6.8 p.p. (10 percent of pre-treatment rates) more after following mostly parallel trends before treatment. This estimate is also quite precise. In contrast, there is no treatment effect for young men in Panel A Column (4), which is consistent with the sex-specific effects of schistosomiasis control in Nigeria (Makamu, Azam and Kazianga, 2018) and the findings that Baird et al. (2016) reported regarding secondary school in their 10-year follow-up in Kenya.¹⁹ This pattern also matches the HIV results, suggesting that the increase (or lack thereof) in schooling induced by deworming contributes to the decrease (or lack thereof) in HIV rates.

4.2.3. Robustness and Credibility

As before, these attendance results might be driven by including both urban and rural observations as well as using the widest range of secondary school ages. To address the first concern, I limit the women's sample to rural observations in Panel A Column (5). The magnitude of the post-deworming estimate remains roughly constant (7.2 p.p., or 11 percent of pre-treatment rates), though it is slightly less precise. I further restrict the sample to rural women ages 15 to 18 in Column (6) to address the second concern, which yields an estimate that is again similar in magnitude (7.4 p.p., or 14 percent of pre-deworming rates) but far more imprecise. Nonetheless, these attendance results appear mostly robust to the concerns mentioned above and again reflect the patterns found in Nigeria (Makamu, Azam and Kazianga, 2018). In addition, placebo tests for ages 21 to 24 in Appendix F2 find no evidence of similar divergences in anemia or school attendance.

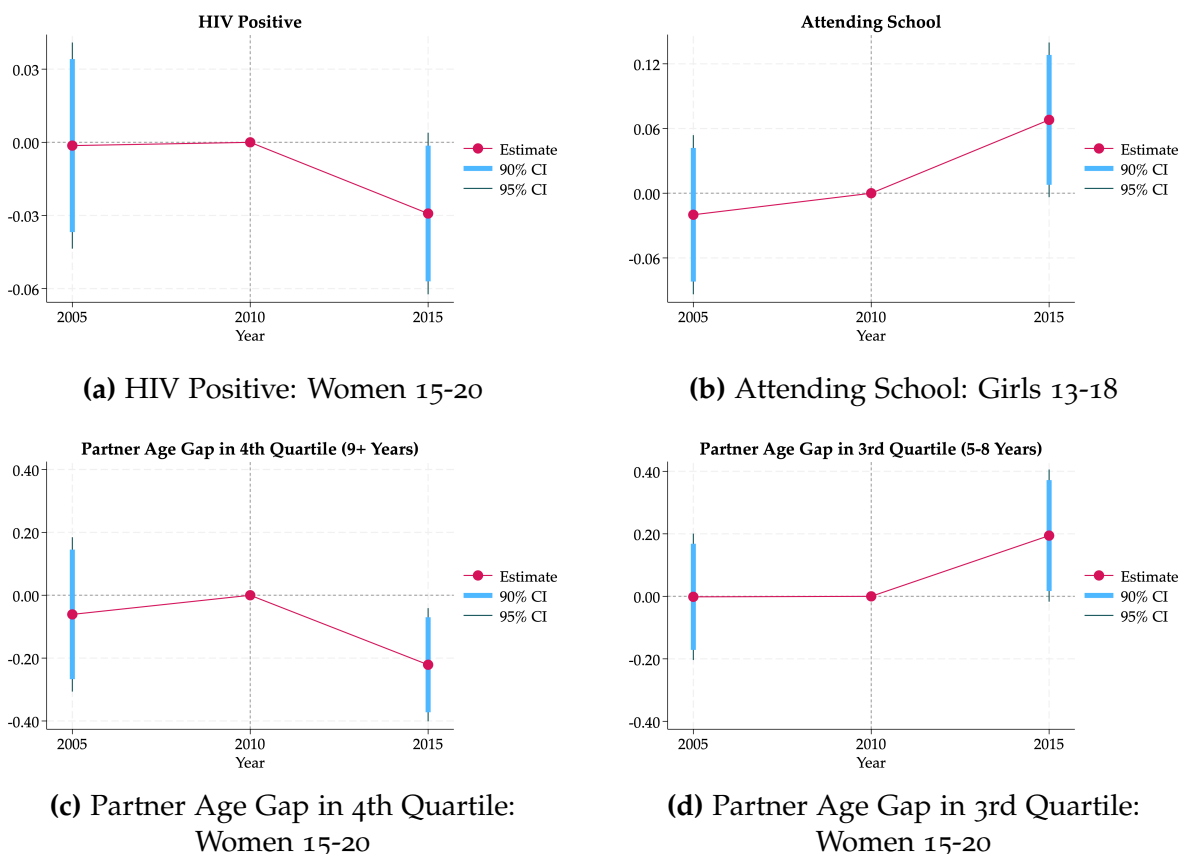
4.3. Young Women's HIV Risks

I now study whether deworming—most likely via the increase in young women's human capital—affected proximate causes of HIV infection. As summarized in Figure 1a, the effect 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 Table 2 Panel B, and in Appendix G I restrict the sample to women ages 15 to 18 (pooling urban and rural areas as well as examining the latter separately).²⁰

¹⁹ 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.

²⁰ I examine both the pooled and rural samples because statistical power is a concern with just one-third to one-half as many respondents in these groups as in Table 2 Panel B.

Figure 2: Event-Study Plots



Notes: The panels show event-study plots corresponding to the estimates in Table 1 Column 1 (top left), Table 2 Panel A Column 3 (top right), and Panel B Columns 3 (bottom left) and 4 (bottom right).

4.3.1. Knowledge of HIV Transmission

To test for a knowledge effect, I use whether young women correctly identify having only one sexual partner and using a condom as reducing the risk of contracting the virus.²¹ Panel B Column (1) shows no evidence of increased awareness of the former’s protective effects, but there is a substantially larger and precisely estimated increase in knowledge of the latter’s in high-schistosomiasis districts after deworming (9.0 p.p., or 13 percent of their pre-treatment mean). This effect is plausibly linked to increased school attendance, though whether it leads to more condom use is an empirical question I study below.

²¹ 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.

4.3.2. *Marriage Market Matching*

I also examine whether deworming changed the type and quantity of men whom young women matched with in marriage markets. For the former, I create indicator variables for whether a woman's relationship had an age gap in the highest quartile (the man is 9 or more years older than she is) and second-highest quartile (5 to 8 years older) given the importance of partner age disparities in Southern Africa's HIV epidemic (Leclerc-Madlala, 2008; de Oliveira et al., 2017). The estimates in Panel B Columns (3) and (4) along with the plots in Figures 2c and 2d show that young women in high-schistosomiasis districts experienced a 22.1-p.p. greater decline in relationships with 9-plus-year age gaps and a 19.4 p.p. greater increase in those with 5-to-8-year age gaps (respectively 83 and 49 percent of pre-treatment means). Both estimates are precise, and they suggest that deworming shifted young women to partners with nearly half of the HIV prevalence.²² As for sexual partners, Panel B Column (5) shows that young women's rates of having had 2 or more in their lifetimes declined 2.7 p.p more in high-schistosomiasis districts after treatment (35 percent of pre-deworming rates), though the estimate is imprecise. Nonetheless, this result is suggestive of deworming reducing another important HIV-risky behavior in marriage markets.

4.3.3. *Condom Use*

Lastly, I investigate whether respondents reported using a condom in their last sexual intercourse. In Panel B Column (6), there is a large negative estimate (-4.2 p.p., or 46 percent of pre-treatment rates) but it is imprecise. At first glance, this larger decrease is a surprising given the increase in condom knowledge shown in Column (2). However, it is possible to reconcile these findings in the following manner: if 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.

4.3.4. *Robustness, Credibility, and Spillovers*

In Appendix G, I first test the robustness of these results by restricting the sample to women ages 15 to 18 as well as further limiting it to rural areas. For the most part, the estimates are similar but precision decreases, as sample sizes fall by one-third to one-half for the pooled results and by one-half to two-thirds for the rural-only results.

²² For 20-year-old women in high-schistosomiasis districts prior to treatment, moving from men who were between 9 and 12 years older to between 5 and 8 years older would have reduced their chances of having HIV-positive partners from 20.7 percent to 11.1 percent. The analogous decrease for 18-year-old women would have been from 14.9 percent to 8.2 percent.

One notable exception is that the estimated post-deworming decline in having had 2 or more sexual partners gets slightly larger and much more precise in both samples. I then address concerns that treatment could have changed these women’s rate of entering partnerships or age of sexual debut by showing that there is no evidence for differential post-deworming changes in being single (i.e., never in a partnership) or having had sex.

I also examine these outcomes for women ages 21 to 24, which are placebo tests for the knowledge outcomes and condom use but tests for spillovers in the marriage market outcomes, as the actions of dewormed young women can change the pool of partners available to others. As expected, there are no substantive divergences in knowledge or condom use following treatment. But there is some evidence of spillovers in marriage markets: women in this age group in high-schistosomiasis districts experienced an 8.9-p.p. larger increase in relationships with the highest quartile of age gaps and a 10.4-p.p. larger decrease in relationships in the second-highest quartile (respectively 41 and 29 percent of pre-deworming means), though the former estimate is imprecise. However, the apparent increase in their age gaps did not affect their HIV rates (see Section 4.1), likely because this change led to only a small increase in their partners’ HIV prevalence.²³

4.4. Assessing Plausibility and Quantifying Channels’ Contributions

I then conduct a back-of-the-envelope exercise to benchmark whether the size of the HIV effect for young women is plausible and decompose it into contributions from the reductions in partner age gaps (the only precise estimates for the HIV risk factors in Table 2 Panel B) and urogenital lesions (which I could not measure accurately in the data).²⁴ My starting point is a simple static model of young women’s HIV prevalence

$$\text{HIV} = \sum_{r \in \{\leq 4, 5-8, \geq 9\}} \mathbb{P}[\text{age gap} \in r] \times \mathbb{P}[\text{partner HIV} | r] \times \sum_{u=0}^1 \mathbb{P}[\text{ulcer}=u] \times \mathbb{P}[\text{contract HIV} | u], \quad (2)$$

where $\mathbb{P}[\text{age gap} \in r]$ is the probability that partner age gaps are in the range r (highest quartile, second-highest quartile, or below-median), $\mathbb{P}[\text{partner HIV} | r]$ is the probability their male partners have HIV given the age gap, $\mathbb{P}[\text{ulcer}=u]$ is the probability that women

²³ For 24-year-old women in high-schistosomiasis districts prior to treatment, moving from men who were between 9 and 12 years older to between 5 and 8 years older only would have reduced their chances of having HIV-positive partners from 23.5 percent to 20.7 percent.

²⁴ I thus assume that education has no direct effect on HIV, as the increased awareness of condoms reducing HIV risk did not appear to generate significant changes in their use. Instead, my assumption is that it affects HIV only through its impacts on marriage markets.

have genital ulcers, and $\mathbb{P}[\text{contract HIV}|u]$ is the probability of women contracting HIV given their genital ulcer status.

In Appendix H, I detail the assumptions I make and the values I take from the literature on HIV transmission to calculate this number. Doing so yields an estimate of pre-deworming HIV prevalence among women ages 15 to 20 of 8.1 percent. Notably, it is fairly close to the measured rate of 6.4 percent reported in Table 1, which is somewhat surprising given how simple this model is.

I estimate the expected size of deworming's effect on HIV by taking the derivative of equation (2) with respect to heavy schistosome infection rates,

$$\begin{aligned} \frac{d\text{HIV}}{d\text{Schisto}} &= \sum_{r \in \{\leq 4, 5-8, \geq 9\}} \frac{\partial \mathbb{P}[\text{age gap} \in r]}{\partial \text{Schisto}} \times \mathbb{P}[\text{partner HIV}] \times \sum_{u=0}^1 \mathbb{P}[\text{ulcer}=u] \times \mathbb{P}[\text{contract HIV}|u] \\ &+ \sum_{r \in \{\leq 4, 5-8, \geq 9\}} \mathbb{P}[\text{age gap} \in r] \times \mathbb{P}[\text{partner HIV}] \times \sum_{u=0}^1 \frac{\partial \mathbb{P}[\text{ulcer}=u]}{\partial \text{Schisto}} \times \mathbb{P}[\text{contract HIV}|u], \end{aligned} \tag{3}$$

using the estimates in Table 2 Panel B for the first partial derivative term, and making the same assumptions detailed in Appendix H. Plugging these values into equation (3) and taking the sum of the products yields an estimated decrease in young women's HIV rates of 2.6 p.p., which is exceptionally close to the 2.9-p.p. decrease in Table 1.

Importantly, I can decompose this estimate into the effects of decreasing young women's partner age gaps and decreasing the probability they have genital ulcers: the former accounts for 1.7 p.p. of the decline in HIV (65 percent) and the latter accounts for 0.9 p.p. (35 percent). Taken with the appropriate caveats, this back-of-the-envelope calculation suggests that the social (i.e., marriage market) effects of schistosomiasis are a substantial contributor to its relationship with HIV. This implication is important because it highlights the need to focus on more than the clinical consequences of the disease, which has effectively been the entire focus of the public health literature.

5. Cost-Benefit Analyses

Lastly, 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.²⁵ 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.²⁶ I also assume that the additional 2.9-p.p. reduction in 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 2.9-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 \$55. 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 6.8-p.p. greater increase in secondary school attendance 3 years later, net of their opportunity

²⁵ 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.

²⁶ 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.

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.²⁷ 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 above \$122.

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 expenditures. Far larger than the direct costs of the program (calculated above) were the costs of the 6.8-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 \$30.34. Therefore, the total discounted costs equaled \$31.40 per student treated.

The value of the reduction in ART outlays depends on how long the 2.9 percent 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.²⁸ Because the results in Section 4 show that marriage market matches for young women changed substantially, it is very much 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

²⁷ This assumption differs substantially from the authors' given their focus on primary school.

²⁸ Alternatively, initiating ART immediately after becoming HIV positive reduces it to just over 3 additional years (through 2018), or 6 years total.

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.

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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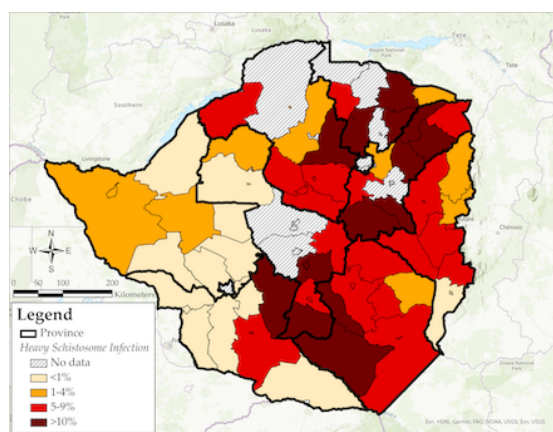
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Appendix A. Geographic Distribution of Schistosomiasis and HIV

A1. Heavy Schistosome Infection

Figure A1: Heavy Schistosome Infection [6]

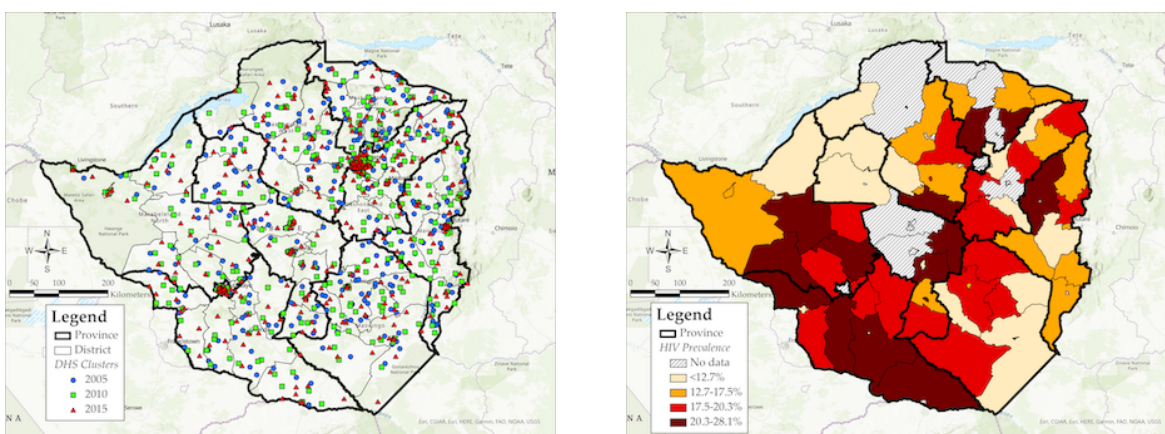


Heavy *S. haematobium* Infection, Students

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 A1: DHS Clusters and HIV Prevalence [9]



(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 squared), 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 between 14 and 774 respondents in each district (median: 209). Levels of prevalence are grouped into quartiles.

Appendix B. Pre-Deworming Relationships between Schistosomiasis and HIV

Table B1: Pre-Deworming Relationships between Schistosomiasis and HIV [9]

	Women		Men	
	15-34 (1)	35-54 (2)	15-34 (3)	35-54 (4)
<i>Heavy Schistosome Infection Range</i>				
1-4%	0.020 (0.017)	0.056 (0.030)	-0.015 (0.016)	0.036 (0.044)
5-9%	0.033 (0.017)	0.074 (0.028)	0.009 (0.013)	0.041 (0.042)
≥10%	0.029 (0.016)	0.073 (0.036)	-0.008 (0.015)	0.061 (0.045)
Observations	7,625	2,673	5,656	2,134
Districts	67	67	67	67
Pre-Deworming Mean (Range: <1%)	0.160	0.233	0.087	0.253

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.

I formally examine the pre-deworming relationship between schistosomiasis and HIV by assigning DHS survey clusters their district’s category of heavy schistosome infection and estimating

$$HIV_{i,c,t} = \alpha_{p(c)} + \gamma_t + \sum_{k=2}^4 \tau_k \times Category_{d(c)} + \mathbf{X}_i\beta + \mathbf{Z}_c\delta + \epsilon_{i,c,t}, \quad (\text{B1})$$

where $HIV_{i,c,t}$ indicates whether individual i in survey cluster c in year t is HIV positive, $\alpha_{p(c)}$ and γ_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), \mathbf{X}_i are individual-level controls (age and age squared), \mathbf{Z}_c are cluster-level controls (altitude and a quadratic in latitude and longitude), and $\epsilon_{i,c,t}$ is the idiosyncratic error term.

The coefficients of interest are the τ_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.

The same patterns visible in Figure 1 emerge in Table B1: for young and older women, HIV prevalence mostly increases as heavy schistosome infection rates increase, while this relationship exists only for older men.

Appendix C. Comparing High- and Low-Schistosomiasis Districts prior

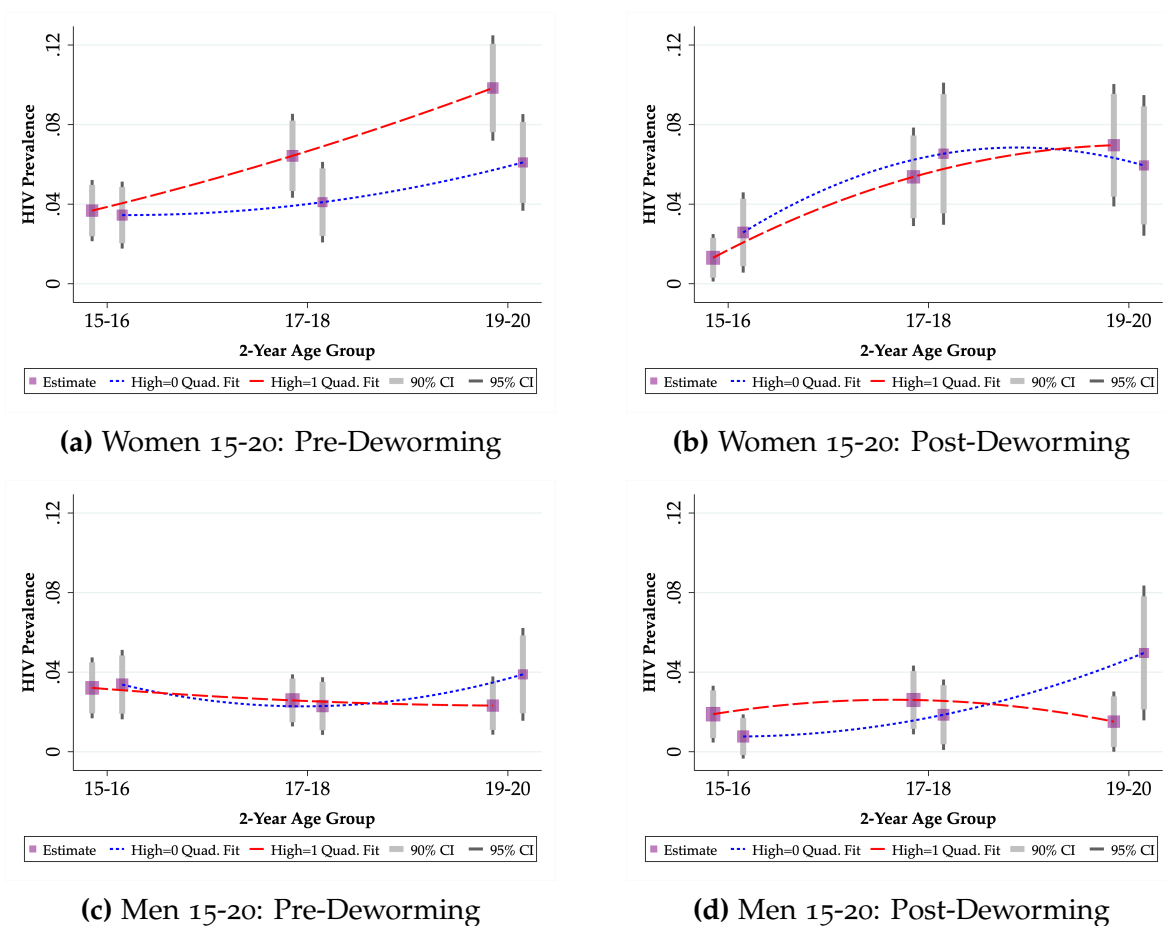
Table C1: Descriptive Statistics and Balance Tests [12]

	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
Districts			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)
Altitude	1,049.7 (289.3)	1,015.3 (282.9)	-34.433 (76.501)	1,027.8 (277.8)	1,024.6 (273.5)	-3.254 (72.421)
Observations			3,583			3,658
Districts			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
Districts			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)
Refused Hemoglobin Test	0.069	0.075	0.007 (0.010)	0.123	0.097	-0.025 (0.021)
Observations			3,371			3,401
Districts			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.

Appendix D. Young Adult HIV Prevalence before and after Deworming

Figure D1: Young Adults' Pre- and Post-Deworming HIV Prevalence [12]



Notes:

I show the main result visually by plotting pre- and post-deworming HIV prevalence for young women and men by 2-year age groups, the use of which reduces noise in the data. Prior to deworming, HIV prevalence among young women in high-schistosomiasis districts increased more quickly with age (top left panel); following treatment, the gap had closed (top right panel). No such change occurred for men, as there were no substantive differences by age in HIV prevalence before deworming (bottom left panel) or afterward (bottom right panel), although the latter is more noisy.

Appendix E. Placebo Tests: HIV Prevalence in Untreated Age Groups

E1. Placebo Test: HIV Prevalence by Age Group

Table E1: HIV Prevalence by 4-Year Age Group [13]

	2005 \times High (1)	2015 \times High (2)	Obs. (3)	Districts (4)	Pre-Deworming Mean (High=1) (5)
<i>Panel A. Women</i>					
21-24	-0.045 (0.027)	-0.009 (0.037)	2,435	71	0.151
25-28	0.038 (0.038)	-0.052 (0.049)	2,742	71	0.250
29-32	0.004 (0.058)	-0.004 (0.061)	1,944	71	0.290
33-36	0.012 (0.048)	-0.032 (0.066)	2,089	71	0.337
37-40	0.035 (0.065)	-0.153 (0.116)	1,281	71	0.323
41-44	0.021 (0.085)	0.005 (0.102)	1,067	71	0.243
45-48	0.021 (0.115)	-0.050 (0.092)	947	70	0.195
<i>Panel B. Men</i>					
21-24	0.007 (0.034)	0.005 (0.035)	1,559	70	0.066
25-28	0.007 (0.057)	0.015 (0.049)	1,742	71	0.112
29-32	0.078 (0.057)	0.066 (0.051)	1,268	71	0.182
33-36	-0.086 (0.086)	-0.048 (0.052)	1,438	71	0.264
37-40	0.020 (0.060)	-0.090 (0.077)	949	70	0.331
41-44	0.110 (0.099)	-0.034 (0.112)	720	71	0.295
45-48	0.110 (0.096)	0.028 (0.143)	679	71	0.350

Notes: Columns (1) and (2) contain the estimates and bootstrap standard errors (in parentheses) for each age group using the procedure developed by [de Chaisemartin and D'Haultfoeuille \(2020\)](#). Observations are clustered by district. High equals 1 if a district had high or the highest pre-deworming rates of heavy schistosome infection, and 0 otherwise. Regressions control for age, age squared, altitude, and a quadratic in latitude and longitude.

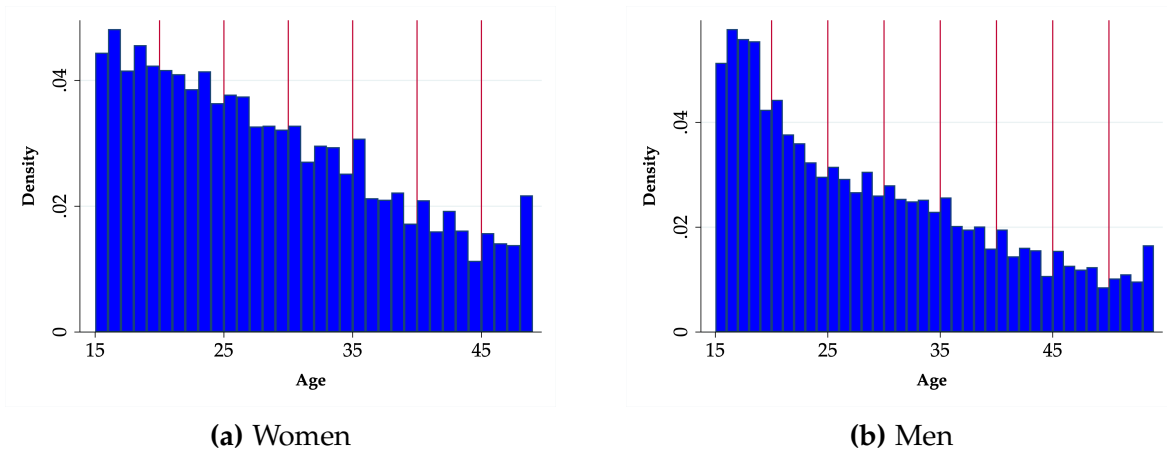
In every case, no age group has a statistically significant estimate for the post-deworming period (2015), and almost all of those with economically significant coefficients for 2015 are matched by 2005 estimate of a similar magnitude. The main exception is for women and men ages 37 to 40—both groups have a small pre-period estimate

and a large negative post-deworming coefficient—and men ages 45 to 48 have a smaller pattern in this vein.

However, as the histograms in Figure E1 show, there is significant 5-year age heaping starting at 30 for both women and men. Because all three of these groups contain a problematic age, there are substantial concerns regarding the data's reliability in this range.

E2. Histograms: Age in the DHS HIV Data

Figure E1: Histograms of Age by Sex



Notes:

Appendix F. Childhood Human Capital

F1. Urogenital Lesions in the DHS Data

Table F1: Effect of Deworming on Measures of Urogenital Lesions [14]

	Genital Ulcer (1)	Genital Discharge (2)
2005 × High	-0.013 (0.008)	-0.011 (0.015)
2015 × High	-0.007 (0.012)	-0.007 (0.011)
Observations	4,854	4,850
Districts	71	71
Pre-Deworming Mean (High=1)	0.018	0.024

Notes: Estimates and bootstrap standard errors (in parentheses) for each age group using the procedure developed by [de Chaisemartin and D’Haultfoeuille \(2020\)](#). Observations are clustered by district. High equals 1 if a district had high or the highest pre-deworming rates of heavy schistosome infection, and 0 otherwise. Regressions control for age, age squared, altitude, and a quadratic in latitude and longitude.

The hypothesis in the public health literature is that the schistosomiasis-HIV relationship should arise through genital ulcers. In particular, 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](#)).

In the DHS data, respondents self-report whether they had any genital ulcers or discharges in the last year, which are likely imperfect measures because providing accurate answers requires identifying them correctly. I attempt to use these data to examine whether there was decline in urogenital lesions coinciding with the drop in young women’s HIV prevalence. But the small and noisy estimates in Table F1 Columns (1) and (2) along with the extremely low reported rate of ulcers (1.8 percent) and discharges (2.4 percent) among young women in high-schistosomiasis districts prior to deworming suggests there is severe undercounting. As a result, it is not clear from these data what truly happened to urogenital lesions following deworming.

F2. Robustness: Placebo Tests

Table F3: Placebo Test: Health and Schooling for Ages 21 to 24 [16]

	Anemic		Attending School	
	Women	Men	Women	Men
	21-24 (1)	21-24 (2)	21-24 (3)	21-24 (4)
2005 × High	0.049 (0.052)	-0.002 (0.026)	-0.008 (0.019)	0.001 (0.024)
2015 × High	-0.025 (0.068)	-0.003 (0.039)	0.011 (0.020)	-0.008 (0.034)
Observations	2,565	1,703	3,009	2,288
Districts	71	71	71	71
Pre-Deworming Mean (High=1)	0.314	0.033	0.025	0.068

Notes: Estimates and bootstrapped standard errors (in parentheses) are obtained using the procedure developed by [de Chaisemartin and D’Haultfœuille \(2020\)](#). Observations are clustered by district. High equals 1 if a district had high or the highest pre-deworming rates of heavy schistosome infection, and 0 otherwise. Regressions control for age, age squared, altitude, and a quadratic in latitude and longitude.

Appendix G. Young Women's HIV Risks

G1. Robustness: HIV Risks for Women Ages 15 to 18

Table G1: Effects of Deworming on HIV Risks for Women Ages 15 to 18 [18]

	Know 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. All</i>						
2005 × High	-0.067 (0.048)	-0.028 (0.068)	-0.098 (0.134)	0.134 (0.123)	-0.009 (0.017)	-0.088 (0.069)
2015 × High	-0.005 (0.046)	0.054 (0.049)	-0.202 (0.114)	0.323 (0.138)	-0.029 (0.019)	-0.077 (0.078)
Observations	3,221	3,221	547	547	3,361	823
Districts	71	71	64	64	71	67
Pre-Deworming Mean (High=1)	0.807	0.707	0.215	0.470	0.046	0.124
<i>Panel B. Rural</i>						
2005 × High	-0.057 (0.057)	0.030 (0.066)	-0.129 (0.097)	0.148 (0.123)	-0.005 (0.017)	-0.081 (0.071)
2015 × High	0.007 (0.058)	0.055 (0.062)	-0.174 (0.130)	0.298 (0.155)	-0.036 (0.026)	-0.051 (0.070)
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

Notes: Estimates and bootstrap standard errors (in parentheses) for each age group using the procedure developed by [de Chaisemartin and D'Haultfœuille \(2020\)](#). Observations are clustered by district. High equals 1 if a district had high or the highest pre-deworming rates of heavy schistosome infection, and 0 otherwise. Regressions control for age, age squared, altitude, and a quadratic in latitude and longitude.

G2. Robustness: Partnerships and Sexual Debuts for Young Women

Table G2: Effects of Deworming on Young Women’s Partnerships and Sex [18]

	Never in Partnership		Never Had Sex	
	Ages 15-20 (1)	Rural 15-18 (2)	Ages 15-20 (3)	Rural 15-18 (4)
2005 × High	-0.006 (0.038)	-0.040 (0.043)	-0.035 (0.034)	-0.087 (0.037)
2015 × High	-0.014 (0.045)	-0.055 (0.053)	-0.012 (0.042)	-0.058 (0.054)
Observations	4,863	2,752	4,863	2,752
Districts	71	54	71	54
Pre-Deworming Mean (High=1)	0.649	0.749	0.594	0.713

Notes: Estimates and bootstrap standard errors (in parentheses) for each age group using the procedure developed by [de Chaisemartin and D’Haultfœuille \(2020\)](#). Observations are clustered by district. High equals 1 if a district had high or the highest pre-deworming rates of heavy schistosome infection, and 0 otherwise. Regressions control for age, age squared, altitude, and a quadratic in latitude and longitude.

G3. Placebo and Spillover Tests

Table G4: Placebo and Spillover Test: HIV Risks for Women Ages 21 to 24 [18]

	Know 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)
2005 × High	-0.062 (0.044)	-0.024 (0.039)	0.058 (0.062)	-0.011 (0.063)	-0.057 (0.044)	0.019 (0.035)
2015 × High	-0.030 (0.050)	-0.065 (0.052)	0.089 (0.071)	-0.104 (0.058)	-0.065 (0.049)	-0.013 (0.045)
Observations	2,640	2,638	1,901	1,901	2,690	2,225
Districts	71	71	71	71	71	71
Pre-Deworming Mean (High=1)	0.893	0.821	0.216	0.357	0.259	0.072

Notes: Estimates and bootstrap standard errors (in parentheses) for each age group using the procedure developed by [de Chaisemartin and D’Haultfœuille \(2020\)](#). Observations are clustered by district. High equals 1 if a district had high or the highest pre-deworming rates of heavy schistosome infection, and 0 otherwise. Regressions control for age, age squared, altitude, and a quadratic in latitude and longitude.

Appendix H. Assessing Plausibility and Quantifying Channels' Contributions

To determine how closely the model in equation (2) can match pre-deworming HIV prevalence for women ages 15 to 20, I take the first probability from the pre-treatment means in Table 2 Panel B and the second from calculations like those in footnote 22 for 20-year-old women.¹ As up to 75 percent of those with urogenital schistosomiasis have *S. haematobium* eggs in their genitals (Kjetland et al., 2005) and most of the high-schistosomiasis districts had overall schistosome infection rates of between 10 and 50 percent (Midzi et al., 2014), I assume that 20 percent of these women had genital ulcers.

Calculating the probabilities of contracting HIV conditional on ulcer status requires several inputs. The meta-analysis by Boily et al. (2009) reports that the per-act probability of male-to-female HIV transmission without commercial sex exposure in low-income countries is 0.3 percent, and genital ulcers increase transmission risk 5.3 times. Based on Gray et al. (2001), I assume that couples have sex twice per week (i.e., 104 times per year) and the average woman in this age group has been sexually active for 3 years. As a result, the probability of a woman with genital ulcers having contracted HIV from an infected partner over this period was $1 - (1 - 0.003 \cdot 5.3)^{104 \cdot 3} = 99.3$ percent, and for a woman without these lesions, it was 60.8 percent.

Plugging these numbers into equation (2) and adding the products together yields 8.1 percent HIV prevalence among women ages 15 to 25: 3.8 percent among those with age gaps in the highest quartile, 3.0 percent among those with age gaps in the second-highest quintile, and 1.3 percent among those with below-median age gaps. Recall that pre-deworming HIV prevalence in this age group in high-schistosomiasis districts was 6.4 percent (see Table 1).

¹ Prior to treatment in high-schistosomiasis districts, 20-year-old women in relationships with 0-to-4-year age gaps would have had a 5.6-percent chance that their partners were HIV positive.