# Supporting Information

## Ndeffo Mbah et al. 10.1073/pnas.1221396110

### SI Text

#### Web Appendix

**Model Structure.** We developed a mathematical model of the interplay between HIV and female genital schistosomiasis (FGS). We used data from a cross-sectional study in rural Zimbabwe (1, 2) to determine the posterior distributions of model parameters through a Bayesian analysis. Based on these parameter estimates, we quantified the cost-effectiveness of a community-based intervention that integrates annual praziquantel administration with provision of clean water, sanitation, and health education (WSH) for reducing the transmission of *Schistosoma hematobium* and HIV in the Zimbabwean rural district of Mount Darwin, where the cross-sectional study was conducted (1, 2).

**Model Formulation.** We first developed a model for *S. hematobium* dynamics. We considered an age-structured model where the population is subdivided into children younger than 15 y of age and adults aged 15 y and older. These age groups differ in their risk for schistosomiasis acquisition and resultant morbidity. The population dynamics are modeled using a compartmental aging model:

$$\frac{dX_c}{dt} = \sigma_b X_a - (\mu_c + \tau)X_c \qquad \text{[S1.1]}$$

$$\frac{dX_a}{dt} = \tau X_c - \mu_a X_a, \qquad \text{[S1.2]}$$

where $X_c$ and $X_a$ are, respectively, the total child and adult populations. The per capita birth rate is $\sigma_b$, the transition (aging) rate is $\tau$, and the child and adult mortality rates are $\mu_c$and $\mu_a$, respectively. Mortality from *S. hematobium* is negligible (3). We modeled schistosomiasis dynamics using the following snail-human model:

$$\frac{dX_c^F}{dt} = \sigma_b N_a/2 - (\mu_c + \tau + A_c S_I)X_c^F + \gamma Y_c^F \qquad \text{[S2.1]}$$

$$\frac{dX_c^M}{dt} = \sigma_b N_a/2 - (\mu_c + \tau + A_c S_I)X_c^M + \gamma Y_c^M \qquad \text{[S2.2]}$$

$$\frac{dY_c^F}{dt} = A_c S_I X_c^F - (\gamma + \tau + \mu_c)Y_c^F \qquad \text{[S2.3]}$$

$$\frac{dY_c^M}{dt} = A_c S_I X_c^M - (\gamma + \tau + \mu_c)Y_c^M \qquad \text{[S2.4]}$$

$$\frac{dS_I}{dt} = \left(B_c\left(Y_c^F + Y_c^M\right) + B_a\left(Y_a^F + Y_a^M\right)\right)(1 - S_I) - \nu S_I \qquad \text{[S2.5]}$$

$$\frac{dX_a^F}{dt} = \tau X_c^F - (\mu_a + A_a S_I)X_a^F + \gamma Y_a^F \qquad \text{[S2.6]}$$

$$\frac{dX_a^M}{dt} = \tau X_c^M - (\mu_a + A_a S_I)X_a^M + \gamma Y_a^M \qquad \text{[S2.7]}$$

$$\frac{dY_a^F}{dt} = \tau Y_c^F - (\mu_a + \gamma)Y_a^F + A_a S_I X_a^F \qquad \text{[S2.8]}$$

$$\frac{dY_a^M}{dt} = \tau Y_c^M - (\mu_a + \gamma)Y_a^M + A_a S_I X_a^M, \qquad \text{[S2.9]}$$

where $S_I$ is density of infected snails, $\gamma^{-1}$ is the duration of schistosomiasis infection, $A_c$ is the snail-to-child transmission rate, $B_c$ is the child-to-snail transmission rate, $A_a$ is the snail-to-adult transmission rate, and $B_a$ is the adult-to-snail transmission rate. Because *S. hematobium* is endemic in Zimbabwe, we parameterized the schistosomiasis transmission rate by running the model to equilibrium and using the least-squares approach to fit the equilibrium prevalence of *S. hematobium* predicted by the model to epidemiological studies from the Zimbabwean rural district of Mount Darwin (58% for school-aged children, 39% for adults, and 10% for snail) (4–6). To test the identifiability of our model, we numerically searched for a global optimum to the least-square objective function. To do so, we solved the least-square minimization problem 100 times, using random initial estimates of the model parameters, selecting the most optimal solution. Although these searches are not exhaustive, and thus do not constitute formal proof of identifiability, they strongly suggest that our model is identifiable.

We integrated our calibrated *S. hematobium* model with HIV transmission to generate a coinfection model of HIV-FGS dynamics within the adult population. Women enter the coinfection model either infected with FGS or uninfected. We assume that women who reach adulthood without FGS may acquire it at a rate $\lambda_g$, which remains constant from the age of 15–49 y. Because FGS is a persistent disease in areas endemic for *S. hematobium* (7, 8), we assume that women do not recover naturally from FGS (9). Using a mass-action framework, we define the force of infection of HIV from men to women (respectively women to men), $\lambda_{m,f}$ (resp. $\lambda_{f,m}$), as $\beta_{m,f}(Y_{M,h})/N_M$ (resp. $\beta_{f,m}(Y_{F,h} + Y_{F,gh})/N_F$), where $\beta_{m,f}$ (resp. $\beta_{f,m}$) is the transmission rate from men (resp. women) to women (resp. men) and $Y_{M,h}$ (resp. $Y_{F,h}$) is the number of men (resp. women) infected only with HIV, $Y_{F,gh}$ is the number of women infected with both HIV and FGS, and $N_M$ (resp. $N_F$) is the total number of adult men (resp. women). Given that an HIV-infected man is more likely, per contact, to infect a susceptible female partner than an infected woman is to infect a susceptible male partner (10), we set $\beta_{m,f}$ to be equal to $\theta\beta_{f,m}$, where $\theta$ is the elevation in increase of HIV transmission rate from men. To capture the observed leveling off of HIV prevalence in Zimbabwe, we assume that HIV transmission decreases with HIV/AIDS mortality rate: $\beta_{f,m} = \beta_0 \exp(-\alpha(D/N)^n)$, where the transmission parameter takes the value $\beta_0$ at the start of the epidemic, $D$ is the number of annual HIV/AIDS related deaths, and $N$ is the total population size (11, 12). The rate at which HIV transmission declines as the number of HIV/AIDS-related deaths increases is $\alpha$, and $n$ is the factor by which HIV/AIDS-related deaths reduce the HIV transmission rate. Death occurs from every compartment at baseline mortality rate $\mu_a$, with an additional HIV-associated rate $\mu_h$. The model can be expressed with the following system of differential equations:

$$\frac{dX_F}{dt} = (1-P_G)\theta(N)/2 - \lambda_{m,f}XF - \mu_a X_F - \left(\lambda_g P_{F,S}\right)X_F \quad \textbf{[S3.1]}$$

$$\frac{dX_M}{dt} = \theta(N)/2 - \lambda_{f,m}X_M - \mu_a X_M \quad \textbf{[S3.2]}$$

$$\frac{dY_{F,g}}{dt} = P_G\theta(N)/2 - c_G\lambda_{m,f}Y_{F,g} - \mu_a Y_{F,g} + \left(\lambda_g P_{F,S}\right)X_F \quad \textbf{[S3.3]}$$

$$\frac{dY_{F,h}}{dt} = \lambda_{m,f}X_F - (\mu_a+\mu_H)Y_{F,h} - \left(\lambda_g P_{F,S}\right)Y_{F,h} \quad \textbf{[S3.4]}$$

$$\frac{dY_{M,h}}{dt} = \lambda_{f,m}X_M - (\mu_a+\mu_H)Y_{M,h} \quad \textbf{[S3.5]}$$

$$\frac{dY_{F,gh}}{dt} = c_G\lambda_{m,f}Y_{F,g} - (\mu_a+\mu_H)Y_{F,gh} + \left(\lambda_g P_{F,S}\right)Y_{F,h}, \quad \textbf{[S3.6]}$$

where $N\,(=N_F+N_M)$ denotes the number of adults. We assumed that individuals enter the HIV-FGS model at sexual debut (age of 15 y) at a rate $\theta(N)$, which is given by $\sigma_b N(t-\bar{t})$. The birth rate is $\sigma_b$, and $\bar{t}$ is set to 15 y and gives the delay between birth and reaching adulthood, because we only include adults in the HIV dynamics. The exacerbation of HIV transmission due to FGS is $c_G$. $P_G$ and $P_{F,S}$ denote FGS prevalence among girls aged 15 y and *S. hematobium* prevalence among adult women, respectively. $P_G = G_S\kappa$, where $G_S$ denotes the prevalence of *S. hematobium* among girls aged 15 y and $\kappa$ is the proportion of *S. hematobium*-infected girls who have FGS. *S. hematobium* prevalences were derived from the *S. hematobium* dynamic model (Eq. **S2.1**–**S2.9**). The compartmental diagram in Fig. S1 illustrates the flow of individuals as they face the possibility of acquiring each infection.

**Model Fitting.** In a cross-sectional study, Kjetland et al. (1) identified the prevalences of HIV and FGS, as well as the odds ratio of having HIV with or without FGS (Table 2) among women of the Zimbabwean rural district of Mount Darwin. We developed a likelihood function for our Bayesian analysis by assuming normal distributions for HIV and FGS prevalence and a lognormal distribution for the odds ratio. We used a Bayesian Markov chain Monte Carlo (MCMC) approach to estimate the value of $c_G$, the coefficient by which FGS exacerbates HIV transmission. For all other parameters of the HIV-FGS model, prior distributions were determined from epidemiological studies (Tables S1 and S2). To implement the Bayesian MCMC, we developed a MATLAB (MathWorks) code based on the Metropolis–Hastings algorithm (13).

For each iteration of parameters, we ran the models for 30 y from the initial conditions, simulating the introduction of HIV in ∼1970 until 1999, when cross-sectional studies used to parameterize our model were completed (1, 14). We ran 10 separately initialized MCMC simulations for 200,000 iterations, with each using the Metropolis–Hastings method. Convergence was assessed using the Brooks–Gelman–Rubin diagnostic criterion (15).

**Community-Based Intervention.** We considered a combined community-based intervention for schistosomiasis control, including annual praziquantel administration to school-aged children (aged 5–14 y), provision of clean water through improved water supply (e.g., piped supply), provision of sanitation through the construction of ventilated improved pit-type latrines, and a health education campaign, as well as annual praziquantel administration to school-aged children. We assumed that adherence to mass administration of praziquantel was 80% (range: 75–90%) for school-aged children (16). We also assumed that mass administration of praziquantel has a cure rate of 75% (range: 57–93%) for *S. hematobium* (16). For adult women without FGS who have received praziquantel during childhood, we assumed they have a reduced risk of 50% (range: 40–70%) (7) for acquiring FGS relative to those who did not receive treatment during childhood.

We incorporated the effect of WSH on *S. hematobium* transmission in our model by assuming that it reduces *S. hematobium* transmission rates $(A_c, A_a, B_c, B_a)$ by $1-\pi$, where the efficacy of WSH, $\pi$, is varied from 10–70%.

**Costs.** To calculate the cost of praziquantel treatment, we assumed that treatment was delivered using the dose of praziquantel recommended by World Health Organization (2.5 tablets of 600 mg per child per year) (17). As base values, the cost of a 600-mg tablet of praziquantel was US $0.08 (16) and the total delivery cost per child treated, including delivery, training, social mobilization, capital equipment, and administrative costs, was US $0.21 (16). Medical costs for HIV treatment and care included provider-initiated testing (both diagnostic and monitoring), treatment and prophylaxis for opportunistic infections, antiretroviral therapy, and palliative care, totaling US $3,469 in 2004 US dollars at a 5% discount rate for the duration of an HIV infection in sub-Saharan Africa (18). We recalculated the costs using a 3% discount rate to be US $3,695. The antiretroviral therapy coverage in Zimbabwe was assumed to be 34% (19). The Zimbabwean government expenditure on health, other than HIV-related spending, was estimated to be US $26 per capita annually (20, 21). To compute the non-HIV/AIDS health expenditure per HIV case averted, we assumed that the average age of HIV/AIDS acquisition in Zimbabwe is 25 y (22), and that the life expectancy at the age of 25 y is 40 y (23). All costs were discounted at 3% and given in 2004 US dollars (24).

We aggregated the cost of providing WSH over the duration of the intervention period, assuming that the cost of water supply and sanitation included preparation, installation, maintenance, and operational costs. We assumed that preparation and installation costs, which are generally the bulk of water supply and sanitation expenditure, occurred during the first year of the intervention, whereas the maintenance and operational costs mainly occurred during the subsequent years of intervention. The aggregated cost of provision of clean water and sanitation was computed over a 20-y period, which is the average lifespan of latrines and standpipes (25).

**Effectiveness.** We quantified the effectiveness of the community-based intervention on schistosomiasis and HIV in terms of disability-adjusted life years (DALYs), which is a common measure of health burden resulting from mortality and disability (26). The average period of HIV infection ($\mu_H^{-1}$) was estimated by fitting the model to epidemiological data, as described above. We assumed that any infection period includes $\mu_H^{-1}-1$ y of limited morbidity with disability weighting of 0.135 (range: 0.123–0.136) and a year of severe disease with disability weighting of 0.505, both of which are based on empirical estimates (27). We assumed that HIV-infected individuals who received antiretroviral therapy have a disability weighting of 0.167 (range: 0.145–0.469) (27, 28). The antiretroviral therapy coverage in sub-Saharan Africa was assumed to be 37% (range: 34–40%), which is also consistent with empirical estimates (19). The average age at HIV/AIDS acquisition was assumed to be 25 y (22), and life expectancy at the age of 25 y is estimated to be 40 y (23). DALYs for HIV/AIDS and *S. hematobium* were computed with a discount rate of 3% annually but without age weighting. The disability weight associated with *S. hematobium* infection was assumed to be 0.05 (range: 0.005–0.15), which is in line with empirical estimates (16, 27). The annual number of DALYs averted was calculated by multiplying the annual number of infections averted for HIV and *S. hematobium* by the total DALYs averted per case due to disability and premature death (29).

**Cost-Effectiveness Analysis.** We evaluated the cost-effectiveness of the combined community-based intervention for reducing the transmission of *S. hematobium* and HIV in the Zimbabwean rural district of Mount Darwin, in which the status quo is limited provision of clean water and sanitation (6). We assumed that the population of the Mount Darwin district was 150,000 in 2000 (30). We conducted a cost-effectiveness analysis from the perspective of health payers, such as the national government or international donors, which are the major providers of mass treatment of schistosomiasis and HIV antiretroviral therapy in sub-Saharan Africa (31–33).


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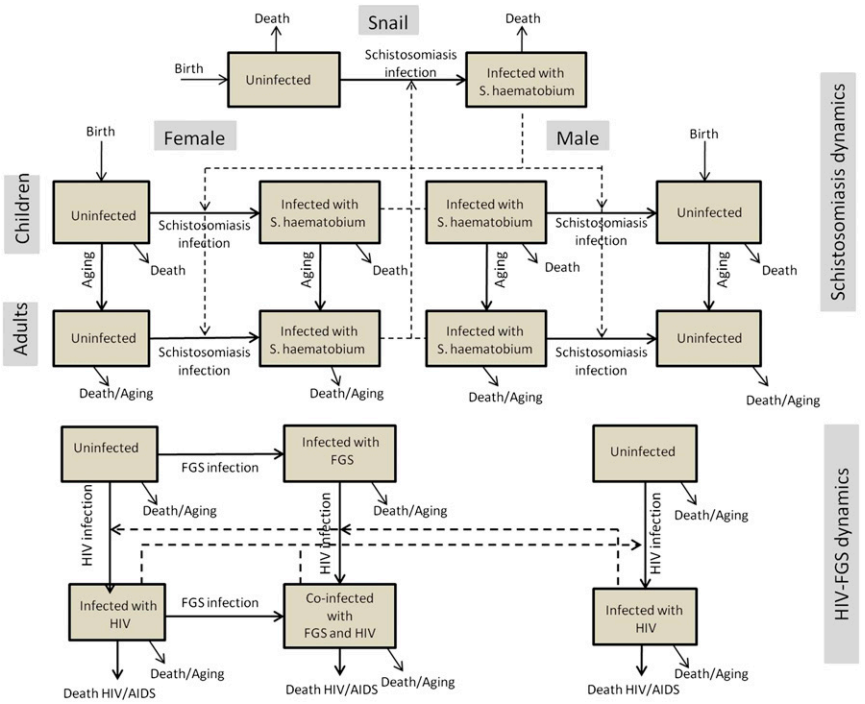


**Fig. S1.** Model structure. The flow between epidemiological classes for the transmission dynamics of FGS and HIV is shown.

**Table S1. Parameter definitions and prior distributions: *S. hematobium* dynamic model**

| Parameter | Definition | Value | Source |
|---|---|---|---|
| $\sigma_b$ | Per capita birth rate | 0.034 $y^{-1}$ | 1 |
| $\mu_c$ | Child mortality rate | 0.02 $y^{-1}$ | 1 |
| $\mu_a$ | Adult mortality rate | 0.02 $y^{-1}$ | 2 |
| $\nu$ | Snail mortality rate | 52/9 $y^{-1}$ | 2 |
| $\tau$ | Aging rate from youth to adulthood | 1/10 $y^{-1}$ | — |
| $A_c$ | Snail-to-child transmission rate | 3.95 | Estimated* |
| $B_c$ | Child-to-snail transmission rate | 0.70 | Estimated* |
| $A_a$ | Snail-to-adult transmission rate | 1.21 | Estimated* |
| $B_a$ | Adult-to-snail transmission rate | 0.25 | Estimated* |

*Parameters were estimated using least squares to fit the *S. hematobium* dynamic model to Zimbabwean prevalence data.

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**Table S2. Parameter definitions and prior distributions: HIV-FGS dynamic model**

| Parameter | Definition | Values (distribution) | Ref. |
|---|---|---|---|
| $c_G$ | Enhance HIV transmission due to FGS | Min = 0, Max = 10 (uniform) | — |
| $\mu_H^{-1}$ | Duration of HIV/AIDS infection | Mean = 8.5, SD = 0.5 (Weibull) | 1 |
| $\beta_0$ | Intrinsic HIV transmission rate | Mean = 0.3, SD = 0.2 (lognormal) | 1, 2 |
| $\theta$ | Relative increase HIV transmission from men | Min = 1, Max = 5 (uniform) | 1 |
| $\alpha$ | Decline rate of HIV transmission | Min = 1, Max = 50 (uniform) | — |
| $n$ | Scale of influence of deaths on HIV transmission | Min = 1, Max = 50 (uniform) | — |
| $\kappa$ | Probability of FGS given childhood infection | Min = 0.33, Max = 0.75 (uniform) | 3, 5, 6 |
| $\lambda_g$ | Probability of FGS given adulthood infection | Mean = 0.01, SD = 0.005 (Beta) | |

Max, maximum; Min, minimum.

*Parameters were estimated using least squares to fit the *S. hematobium* dynamic model to Zimbabwean prevalence data.


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**Table S3. Cross-sectional study data: Confidence interval and distribution approximation**

| Statistic | Values | Proposal distribution |
|---|---|---|
| Prevalence of HIV | 28% (CI: 24–32%) | Mean = 0.28, SE = 0.021 (normal) |
| Prevalence of FGS | 46% (CI: 42–50%) | Mean = 0.46, SE = 0.021 (normal) |
| Odds ratio | 2.1 (CI: 1.2–3.5) | Mean = 2.1, SE = 0.352 (lognormal) |

Data from Kjetland et al. (1, 2). CI, confidence interval.


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