

Supporting Information S1 File: The Stratified Worm Burden (SWB) model and its calibration.

A. Human - snail transmission system

The stratified worm burden (SWB) approach to modeling *Schistosoma* transmission stratifies the at-risk human host population by their worm burdens, $H = \sum_{m \geq 0} h_m(t)$, each stratum carrying m worms/person (more generally between $m\Delta w \leq V < (m+1)\Delta w$, with specified worm step $\Delta w > 1$). The transition among strata is described by matrix $M(\lambda, \gamma, \mu)$, and age/group specific source term $\vec{S} = \{S_m(t)\}$ (see [1-3])

$$\frac{d\vec{h}}{dt} = M(\lambda, \gamma, \mu) \cdot \vec{h} + \vec{S} \quad (1)$$

The parameters of matrix M include snail-to-human force of infection (FOI), λ , the human population turnover rate, μ , and worm mortality, γ . The infectivity of the SWB within the human population is determined by the mean mated worm count (MMC), ϕ_m , in each stratum, and their weighted mean across strata,

$$\Phi = \sum_{m > 0} h_m \phi_m \quad (2)$$

(see, e.g. [2, 4-8]). Alternatively, one can simplify and assume a specific worm distribution such as a negative binomial distribution with mean w and aggregation constant, k . (e.g., the NB(w, k) in a MacDonald-type mean worm burden (MWB) model [9]), and get a closed functional form of $\Phi(w, k)$. In either case, MWB or SWB, human infectivity is the product of worm fecundity, ρ , and MMC,

$$E = \rho \Phi \quad (3)$$

In contrast to the MWB NB assumption for worm/person distribution, in our SWB approach, no a priori assumption is made about the worm distribution in the human host community. Egg release by mated females and individual hosts still depends on age-specific worm fecundity, ρ , and MMC ϕ_m (for the h_m - stratum). In our approach, a host in m -th stratum is assumed to release random daily egg amounts following a negative binomial (NB)-distribution with mean

$E_m = \rho \phi_m$, and aggregation parameter $k_m = k \phi_m$ [3, 10, 11]. So any test pool of an SWB community with population strata $\{h_m\}$ is viewed as random draw of the NB-mixture distribution with weights h_m .

At the endemic (equilibrium) state, strata variables $\{h_m(\lambda)\}$ are determined by age-specific FOI λ . Hence mean mated count $\Phi(\lambda)$ and human infectivity $E(\lambda)$ become functions of λ .

Figure 2 illustrates typical equilibrium distributions for 3 sample villages.

The **snail** population-transmission model employed in our analysis was developed in earlier papers [3, 10, 12]. Here we use a simplified (S-I) version made of susceptible prevalence variables $x + y = 1$, assuming a stationary snail environment,

$$\frac{dy}{dt} = \Lambda(1 - y) - \nu y \quad (4)$$

Snail-to-Human FOI, λ - the human per capita rate of worm accumulation, depends on i) the intermediate larval stage (cercaria) density, C , in local water bodies, ii) the rate of human exposure to affected water bodies (contact rate), ω , and iii) human susceptibility to infection, α (probability of worm establishment/ water contact),

$$\lambda = \alpha \omega C = A y \quad (5)$$

-a linear function of infected snail prevalence.

Human-to-Snail FOI, Λ , depends on parasite miracidial density, M , snail density, N , snail susceptibility (probability of successful invasion), β , and the maximal rate of snail invasion by miracidia, Λ_0 . In the present model, we propose the following nonlinear (saturated) function for Λ ,

$$\Lambda = \Lambda_0 \left(1 - e^{-\beta M/N}\right) \quad (6)$$

Fraction $\left(1 - e^{-\beta M/N}\right)$ is derived from hypothesized Poisson distribution of the “miracidia per snail” variable with mean value M/N . It represents the probability of one or more successful

invasions. Miracidial density, $M = b H E$, depends on human host population size H , and human infectivity E , so

$$\Lambda(E, N) = \Lambda_0 \left(1 - e^{-b E H / N}\right) \quad (7)$$

Maximal invasion rate is a function of snail density N , and other biological/environmental inputs (see, e.g. [13]). However, in the current analysis we treat it as a single (uncertain) parameter to be estimated from other transmission inputs.

The nonlinear function $\Lambda(E, N)$ (7) can be approximated by the linear form, $\Lambda \approx B E$, provided exponent $b E H / N \ll 1$, that is, the combined “human contagion” is small relative to snail density. Such a linear assumption for snail FOI was commonly used in past transmission modeling (e.g. [1, 4, 14, 15]). The resulting transmission coefficient B , is then proportional to human population size, H , and their water contact rate ω .

Human infectivity, E , in equation (7) depends on host population makeup and age –specific exposure/ contamination patterns of different population age groups (children (C) and adults (A)). A single SWB has $E = \rho \Phi(\lambda)$; for mixed population groups C and A, infectivities $\{E_C, E_A\}$ are weighted in proportion to population fractions (H_i), and relative (adult vs. child) exposure factors, $\omega = \omega_A / \omega_C$ [3, 10]. The net result is combined human infectivity

$$E(\omega) = \rho_C \left(H_C \Phi(\lambda_C) + \frac{\rho_A}{\rho_C} \omega H_A \Phi(\lambda_A) \right). \quad (8)$$

-function of calibrated human parameters $\{(\lambda_i, \rho_i)\}$ drawn from the posterior ensemble of best fit parameters based on our calibration (explained below).

B. Coupled human-snail system and model calibration

Human and snail equations (1) - (4) are coupled via two transmission coefficients A (snail-to-human) and B (human-to-snail). The A -coefficient is included in the human FOI expression $\lambda = A y$ (infected snail prevalence), and coefficient B is included in the exponent of the nonlinear snail FOI, Λ . The details of model calibration and estimated parameter values are explained in previously published papers [3, 10]. It proceeds in two steps: (i) *human calibration* for (λ_i, k_i, ρ_i) which are snail-to-human FOI, aggregation, and worm fecundity, estimated from

test data (stool eggs per gram (epg, *S. mansoni*) or eggs per 10 mL by urine filtration (*S. haematobium*)); (ii) *transmission calibration*, for other coefficients (see Table A1).

Model inputs include

- (i) **Human parameters:** age-specific triplets (λ_i, k_i, ρ_i) , $i = \{C, A\}$ which enter human infectivity function, $E_i = \rho_i \Phi(\lambda_i)$. Table A2 gives the results of human calibration (parameters statistics) for 3 sample communities, and **Figure 1** shows their marginal distributions
- (ii) **Snail inputs:** infected prevalence y^*
- (iii) **Environmental/behavioral inputs:** b – child transmission coefficient, ω - relative (adult/child) exposure contamination

Specifically, for A, coefficients are estimated from calibrated human FOI λ_i and (known or hypothesized) infected snail density y

$$A_i = \frac{\lambda_i}{y}; i = \{C, A\} \quad (9)$$

For linear form of snail FOI (used in model M1), transmission coefficient B is estimated from the equilibrium snail equation (4) as

$$B(\omega) = \frac{\nu y^*}{(1 - y^*) E(\omega)} \quad (10)$$

with combined infectivity function **Error! Reference source not found..**

For the nonlinear form of snail FOI (used in model M2) calibration proceeds differently: instead of estimating the pairs (A_i, B) , we estimate (A_i, Λ_0) ,

$$\Lambda_0(b, \omega) = \frac{\nu y^*}{(1 - y^*)(1 - e^{-b E(\omega)})} \quad (11)$$

We sample model uncertainties to provide estimates of variance, and generate a 95% uncertainty interval (UI) in the transmission projection for control interventions.

Table A1: Summary of model parameters

	Known, fixed	Calibrated	
Human	Demographic inputs	Posterior distributions of $\{\lambda^i, \rho^i, k^i\}$, for age-groups $i = \{C, A\}$, estimated from community test data ([16, 17])	
			C A
	Population fractions		0.49 0.51
	Host turnover/year		0.06 0.025
	Worm mortality/year	0.2 0.25	
Snail	Mortality: $\nu = 4$ /year, Infected prevalence (baseline endemic value) ^a : $y^* = .3$ (range .15–.45)	Estimated Max invasion (FOI) rate: Λ_0	
Environment/ behavior	Basic (child) transmission rate: $.5 < b < 5$ Relative exposure/contamination rates ("adult/child): $.5 < \omega < 1.5$	Transmission coefficients $A_i = \lambda^i / Z$	

^a These ranges are broadly consistent with published data [16, 18].

Table A2: Calibrated posterior parameters for 3 Kenyan communities (H – heavy, M – moderate, L –light)

		λ_C	λ_A	k_C	k_A	ρ_C	ρ_A
<i>H</i>	Min	1.8	0.51	0.018	0.014	9.8	4.6
	Max	5.9	1.7	0.083	0.045	35.	12.
	Median	2.8	0.85	0.044	0.026	20.	8.3
	Mean	3.1	0.91	0.046	0.027	21.	8.4
	SD/Mean	0.31	0.31	0.36	0.3	0.3	0.23
<i>M</i>	Min	0.54	0.18	0.049	0.007	40.	2.9
	Max	0.69	0.95	0.077	0.048	55.	14.
	Median	0.6	0.33	0.063	0.023	47.	8.2
	Mean	0.61	0.37	0.063	0.024	47.	8.2
	SD/Mean	0.059	0.42	0.11	0.42	0.079	0.33
<i>L</i>	Min	0.37	0.035	0.045	0.016	28.	3.3
	Max	0.51	0.2	0.084	0.12	44.	14.
	Median	0.43	0.067	0.064	0.059	36.	8.
	Mean	0.43	0.077	0.064	0.062	36.	8.2
	SD/Mean	0.078	0.44	0.14	0.43	0.11	0.32

Captions

Figure 1: Marginal distributions of calibrated posterior distribution for 3 sample communities from coastal Kenya [20]. Demographic makeup consists of children (0-20) adult (20+). For each group, EPG test results were fitted to its (λ, ρ, k) - parameters

Figure 2: Typical endemic (baseline) SWB distributions in 3 sample communities based of calibrated posterior values $\{\lambda_C, \lambda_A\}$

Figures

Figure 1.

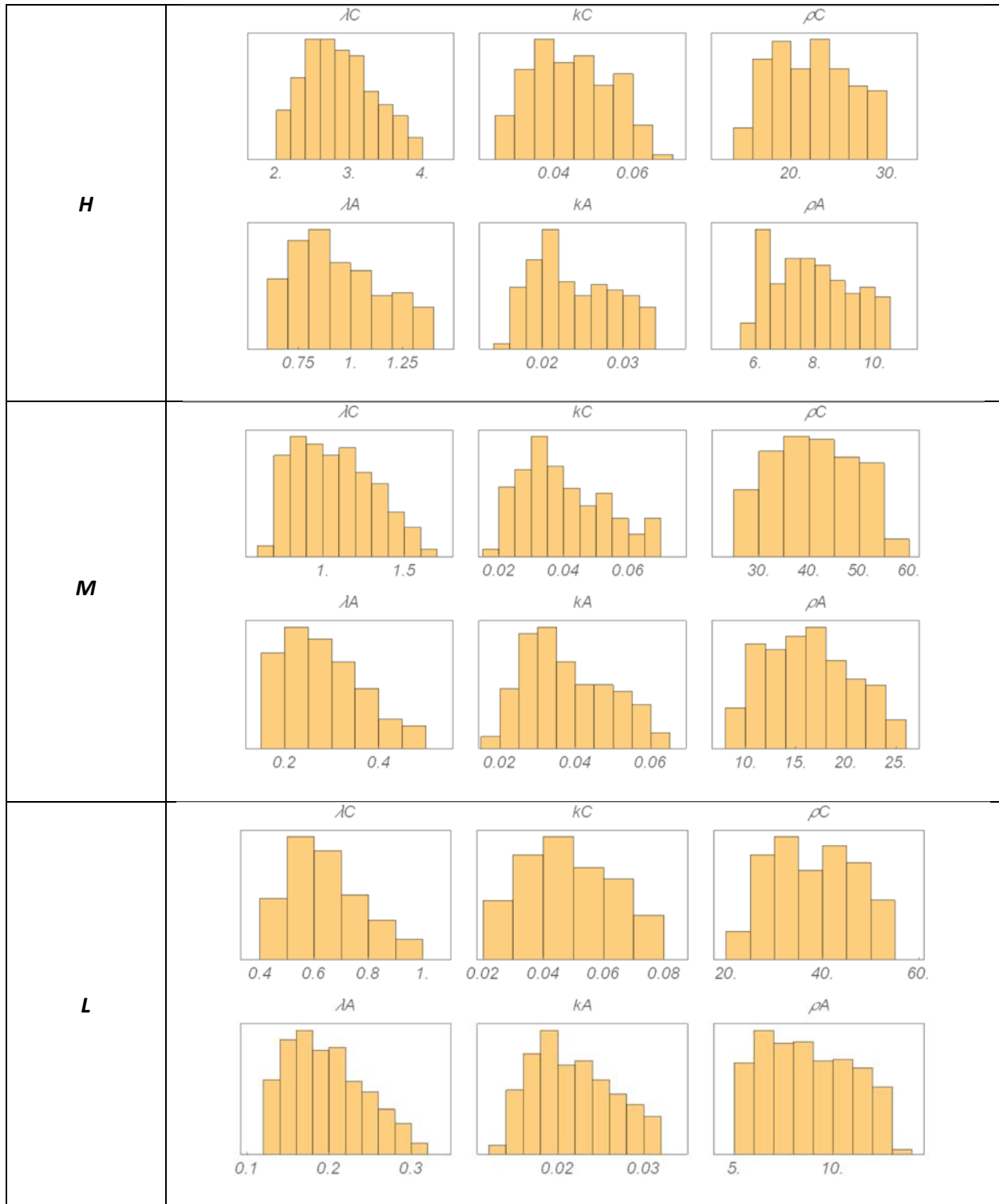
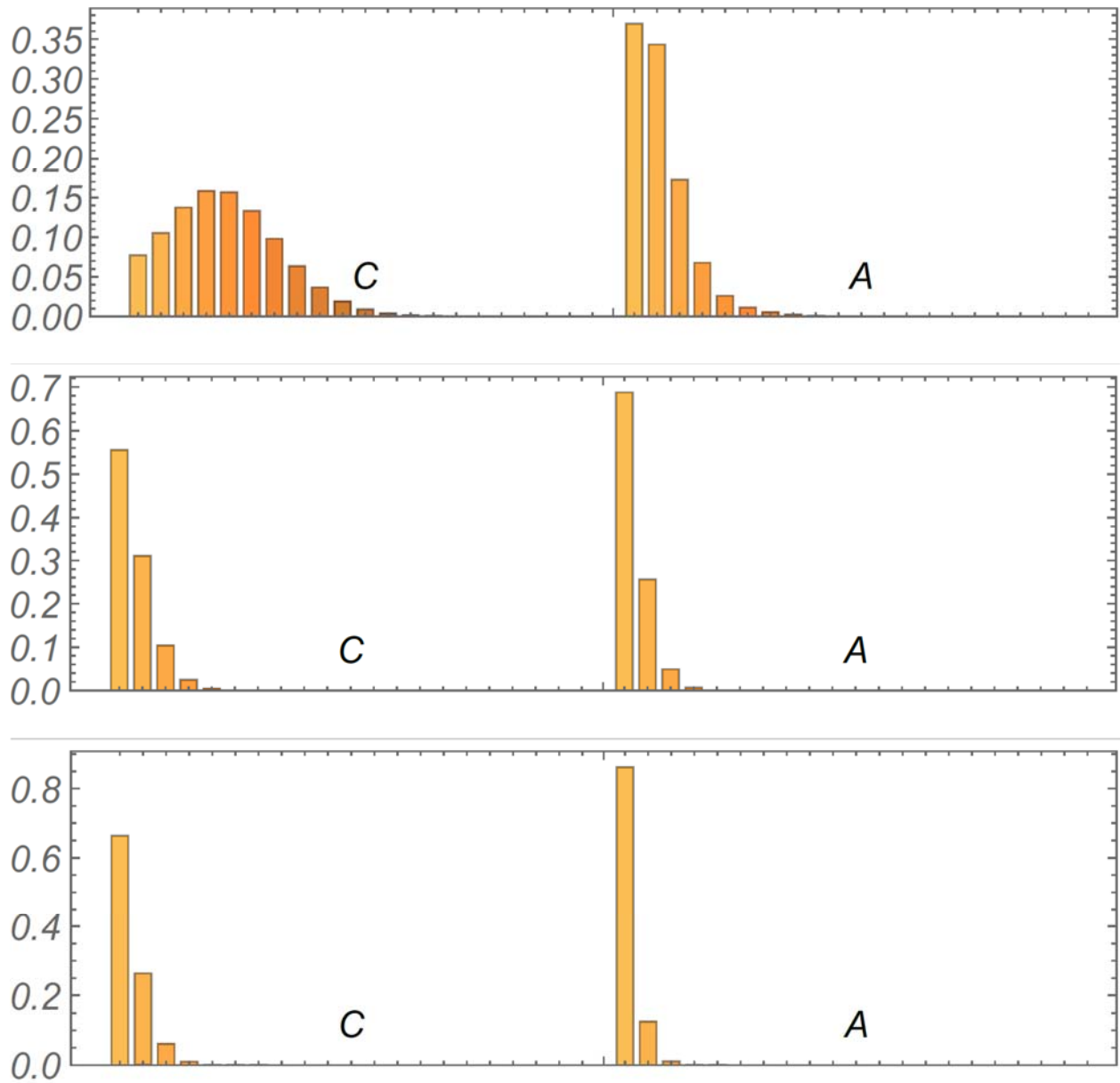


Figure 2.



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