**NTD Modelling Consortium Oxford Schistosomiasis (Schistox) Model**

Model framework

* **Stochastic individual based model** for transmission dynamics of schistosome infection (\*which can be used with specific parameter values).
* **Interventions:** mass drug administration (MDA), WASH, vaccination
* **Age-structured population**: Tracks each individual (by sex i.e. males/females) and potentially adding other markers, e.g. give each individual a unique identifier and then individual parameters looked up in a table of attributes.
* **Population demographics**: Ideally set an age-time-death rate and a population growth rate, such that the model works out the birth rate (per adult woman).
* **Single community model** assuming no migration (single environmental reservoir of infection)
* Accept a separate function for reporting (that can be written in terms of diagnostic characteristics, and group together into age groups(pre-SAC, SAC and adults whose ages can be set; SAC=school-aged children)
* Preferably a **R user interface** (background code can be run in R or more efficient language)
* Degree of parasite aggregation across hosts defined by the negative binomial probability distribution with a specified k value
* See references 1 - 6 for further details of schistosomiasis models.

Model options required

* \*Select **species** and its corresponding parameter values (e.g. *S. mansoni* and *S. haematobium*) which model is being simulated for. Parameters to include (example of further parameters shown in **Table 1**):
  + Age-profile of infection (age-related exposure to infection)
  + Transmission intensity R0
  + Drug efficacy
* Specify **mating function** for dioecious parasite and density-dependence in worm burden/egg production (density dependent fecundity – exponential): monogamous or non-monogamous mating.
* Specify **treatment programme** i.e. coverage levels for age groups, duration and frequency of treatment.
* Set level of **acquired immunity** (i.e. less likely to be infected as an adult if infected as a child). See references 5 - 6.
* Set individual level of **adherence** in order to simulate different adherence patterns, e.g. as an attribute. Examples: 1) when there is full adherence and coverage is 75% SAC this means we treat 75% SAC at random (i.e. not necessarily the same SAC) for each round of MDA; 2) when there is 10% non-adherence this means 10% of the population is not being treated at any round of MDA.
* Record **individual histories** (not all events, but most)

Model output and figures

* Model to save its state plus tables of events etc over N years. Include code to restart the model from any saved state.
* Prevalence of infection and intensity of infection over time for whole population and specific age groups (e.g. figures in references 7-8).
* Prevalence of heavy-intensity infections over time. Heavy-intensity infections definition set in species parameters (varies for *S. mansoni* and *S.* *haematobium*; e.g. figures in references 7-8).
* True prevalence and intensity.
* Calculate positive and negative predictive values (PPV and NPV) to check for elimination vs resurgence (e.g. reference 9).

Fitting model to data

* Fitting model parameters to prevalence and intensity of infection data for all/specific age-groups.
* Examples of published data available that could be used to parametrise model: Additional File 5 (figure 2A) in reference 10. Historical full age profiles of infection data for *S. mansoni* in reference 11 and for *S. haematobium* in reference 12.

Further model development

* Extend to multiple **communities** with migration: more than one environmental reservoir, giving different people different exposures to different sources. Potential to label different individuals (for different village membership etc).
* Add in **multiple infections/parasites** e.g. schistosomiasis and STH
* Add in **hybrid schistosomes** which have a higher fecundity (egg output)/ include zoonotic reservoir
* **Female genital schistosomiasis**
* **Diagnostic** sensitivity and specificity to be added (to give measured prevalence in addition to true prevalence). Diagnostics to include: Kato-Katz, CCA and CAA.

**References**

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[4] Chan MS, Guyatt HL, Bundy DA, et al. The development of an age structured model for schistosomiasis transmission dynamics and control and its validation for *Schistosoma mansoni*. *Epidemiol Infect.* 1995.

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[7] Toor J, Alsallaq R, Truscott JE, et al. Are We on Our Way to Achieving the 2020 Goals for Schistosomiasis Morbidity Control Using Current World Health Organization Guidelines? *CID*. 2018.

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[9] Toor J, Truscott JE, Werkman M, et al. Determining post-treatment surveillance criteria for predicting the elimination of *Schistosoma mansoni* transmission. *P & V.* 2019.

[10] Gurarie D, King CH, Yoon N & Li E. Refined stratified-worm-burden models that incorporate specific biological features of human and snail hosts provide better estimates of *Schistosoma* diagnosis, transmission, and control. *P & V.* 2016*.*

[11] Fulford AJ, Butterworth AE, Ouma JH & Sturrock RF. A statistical approach to schistosome population dynamics and estimation of the life-span of *Schistosoma mansoni* in man. *Parasitology*. 1995.

[12] S. haem Bradley and McCullough. Egg output stability and the epidemiology of *Schistosoma haematobium*. II. An analysis of the epidemiology of endemic *S. haematobium*. *Trans R Soc Trop Med Hyg.* 1973.

**Deterministic model codes previously shared by NTD Modelling Consortium:**

<https://www.ntdmodelling.org/diseases/schistosomiasis-mansoni>