Table S1. A Summary of Mathematical Models for Helminth Infections of Humans

Helminth infection and causal agent	Role of model	Type of model and framework	Questions explored	References
Soil-transmitted helminthiases, in particular ascariasis and hookworm infection	Population biology & transmission dynamics	Deterministic; infection intensity	Role of density-dependent fecundity	[1,2]
		Stochastic	Mode of transmission of infective stages, trickle or clumped	[3]
	Exploration of intervention scenarios	Deterministic, age-structured, intensity and morbidity	Compare impact of MDA and age-targeted chemotherapy on morbidity	[4,5] (EpiWorm)
		Stochastic, individual-based model	Assess the relative impact of chemotherapy and vaccination against human hookworm infection	[6]
Intestinal and urinary schistosomiasis due to *Schistosoma mansoni* and *S. haematobium*	Population biology & transmission dynamics	Deterministic; prevalence of infection in humans and snails	Use of models for evaluating possible control strategies; limitations and uncertainties	[7]
		Deterministic; infection intensity in humans; infection prevalence in snails	Heterogeneities; snail dynamics; miracidia & cercaria dynamics; acquired immunity	[8,9]
		Stochastic	Investigation of the role of immunity in observed patterns in endemic communities	[10,11]
	Exploration of intervention scenarios	Deterministic; infection intensity in humans	Chemotherapy; vaccination; snail control; larval stage control; improved water & sanitation; health education	[12–15]
			Dynamical aspects of the interactions between treatment, immunity and morbidity and consequences for control programmes	[16–18]
	Population biology & exploration of control scenarios	Deterministic; age-structured; infection intensity and prevalence in humans; prevalence of heavy infections	Phenomenological representation of acquired immunity; MDA vs. age-targeted chemotherapy; impact on morbidity	[16,19–21] (EpiSchisto)
		Deterministic; infection intensity in humans and prevalence in snails	Infected snails suffer excess mortality and no reproduction	[22]
	Fitting models to data; estimation of transmission parameters	Deterministic; infection intensity in humans	Changes in the force of infection after annual treatment in Uganda	[23]
	Inclusion of human-worm- and infection-related aspects	Stochastic; lacking transmission-related mechanisms	Short-term effects of schistosome surveys & treatments	[24] (SCHISTOSIM)
	Exploring the evolutionary outcomes of control; drug resistance in schistosomiasis	Differential equations, time delays, mating structure	Exploration of the role of mating structure in the maintenance of genetic diversity	[25–27]

Table S1. Continued

Helminth infection and causal agent	Role of model	Type of model and framework	Questions explored	References
Zoonotic schistosomiasis, *S. japonicum*	Population biology & exploration of control scenarios	Deterministic; prevalence of infection in humans and bovines	MDA vs. targeted treatment of high prevalence groups of humans and bovines	[28,29]
		Infection intensity in humans; infection prevalence in snails; connected villages	Relative roles of environmental and social connectivity for parasite spread, persistence and control	[30]
	Population biology & exploration of control scenarios. Estimation of transmission parameters	Infection intensity in humans (subdivided by occupational and residence groups); prevalence in snails	Occupation-targeted chemotherapy; snail control; environmental aspects of contamination and exposure	[31–34]
		Prevalence of infection in humans, rodents and snails	Chemotherapy; snail control / molluscicides; elimination from Bohol Island, Philippines	[35]
	Fitting model to data. Estimation of transmission parameters	Humans stratified by sex, age, and intensity class Infection prevalence for a range of hosts and villages	Relative importance of transmission from different mammals to snails and from snails to mammals	[36,37]
	Transmission dynamics and its interaction with environmental variables	Coupling a hydrological model with a delay-differential equation schistosomiasis transmission model	Influence of hydrological factors on the prevalence, intensity and timing of infection; influence of hydrologic variability greater on the cercarial stage of the parasite than the miracidial stage	[38]
Onchocerciasis	Population biology & transmission dynamics at endemic equilibrium	Deterministic; infection intensity in humans and simuliid vectors	Role of density-dependent parasite establishment in humans and vectors; propensity of vectors to feed on humans; density-dependent host choice; threshold biting rates in Africa and Latin America	[39–42]
	Exploration of intervention scenarios	Deterministic; infection intensity in humans and simuliid vectors	Impact of nodulectomy alone vs. nodulectomy plus mass ivermectin administration	[43] (also a review of onchocerciasis models)
	Fitting model to data; estimation of (exposure) parameters	Deterministic; host age- and sex-structured	Differential age- and sex-specific exposure to vectors according to human ecology	[44]
	Investigation of ivermectin resistance	All of the above plus parasite genetic-structure regarding ivermectin susceptibility	Sampling protocols to maximize detection of ivermectin resistance	[45]

Table S1. Continued

Helminth infection and causal agent	Role of model	Type of model and framework	Questions explored	References
Onchocerciasis	Transmission dynamics; exploration of intervention scenarios; feasibility of parasite elimination	Stochastic for parasites and humans; deterministic for vectors	Duration of vector control; combination of vector control and mass ivermectin distribution for control and elimination	[46–49] (ONCHOSIM) [50] (SIMON, West Africa; SIMON-A, Americas)
	Population biology at endemic equilibrium	Stochastic	Adult worm aggregation in nodules; density-dependent parasite establishment and fecundity; threshold biting rates in Africa	[51–54]
	Exploration of intervention scenarios	Deterministic; exposure heterogeneity in the human population	Exploration of mass vs. targeted ivermectin distribution	[55]
	Investigation of transmission breakpoints	Full transmission model with positive and negative density-dependent processes	Factors determining the magnitude of transmission breakpoints (vector density, vector competence, propensity to feed on humans) and interactions with treatment	[56,57]
	Investigation of ivermectin effects and efficacy on adult worms and microfilariae	Deterministic; adult female worm population structured in fecund and non-fecund worms	Dynamics of microfilarial reappearance in skin and anti-fecundity effects after single dose treatment	[58]
	Investigation of sub-optimal responses to ivermectin	Individual-based model	Variability in rates of microfilarial reappearance in the skin between populations ivermectin naïve and multiply treated	[59]
	Investigation of anthelmintic resistance	Deterministic; over-dispersed parasite distribution; genetic structure	Resistance allele frequencies in different life-stages following treatment	[60] (also for STHs)
Lymphatic filariasis	Population biology & transmission dynamics; infection and morbidity control; endpoints; monitoring & evaluation	Deterministic; host age-structured, infection intensity in humans and vectors	Morbidity reduction; treatment with drugs exerting different effects on adult worms and microfilariae; vector control; programme endpoints	[61–65] (EpiFil)
	Investigation of transmission breakpoints	Deterministic and stochastic frameworks	Factors determining local variation in elimination thresholds, dependent on vector composition, vector density and initial endemicity	[66,67]
	Population biology & transmission dynamics; exploration of control scenarios	Stochastic	Role of acquired immunity; development of morbidity; effects of control measures (drug treatment and/or mosquito control)	[68–70] (LYMFASIM)
	Investigation of benzimidazole and ivermectin resistance	Deterministic; host age- and parasite genetically-structured, based on EPIFIL	Role of parasite inbreeding; linked resistance; interpretation of molecular genetics surveys	[71–73]

Table S1. Continued

Helminth infection and causal agent	Role of model	Type of model and framework	Questions explored	References
Taeniasis, cysticercosis, and hydatic disease	Exploration of intervention scenarios for *Taenia solium*	Deterministic and stochastic versions of a modified Reed-Frost model	(i) Use of latrines, meat inspection, cooking habits; (ii) rapid detection and treatment of human carriers; pig vaccination; (iii) treatment of humans or pigs	[74]
	Evaluation of control programmes against *Taenia solium* cysticercosis	Stochastic simulation model	Effect of treatment of humans and pigs (alone or in combination) linked to economic evaluation	[75]
	Population dynamics of *Echinococcus granulosus* and exploration of control interventions	Mathematical models of the transmission dynamics between dogs and livestock for Australia, New Zealand, Middle East and Asia	(i) Intense anthelmintic treatment of the dog population; (ii) introduction of newly developed sheep vaccine	[76]
	Population dynamics in echinococcosis and cysticercosis	Integro-differential equations	Population dynamics of *Taenia hydatigena* and *T. ovis*; role of cross immunity and impact of control	[77,78]

The need to develop mathematical frameworks for food-borne trematodiasis transmission under a range of conditions, enabling identification of opportunities for implementation of control measures, has been expressed [79]. However, no mathematical models have yet been developed for the transmission and control of *Clonorchis sinensis*; *Opisthorchis viverrini*, *Opisthorchis felineus* or *Paragonimus* spp. Consideration of models for fasciolasis is outside the remit of the Disease Reference Group for Helminth Infections.

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