Correspondence

CHK reports a grant from the Schistosomiasis Consortium for Operational Research and Evaluation. APG declares no competing interests.

*Charles H King, Alison P Galvani
chk@cwru.edu

Center for Global Health and Diseases, Case Western Reserve University School of Medicine, Cleveland, OH 44106, USA (CHK); and Center for Infectious Disease Modeling and Analysis (CIDMA), Yale School of Public Health, New Haven, CT, USA (APG)


Authors’ reply

Charles King and Alison Galvani cite three specific concerns in their commentary regarding the Global Burden of Disease (GBD) Study1 2016 and we respond briefly to them.

King and Galvani state that environmental factors were not considered within the revised Bayesian meta-regression of the GBD Study 2016. However, we did use environmental factors in our model. The schistosomiasis prevalence model in the GBD Study 2016 underwent a substantial revision that included a systematic review and literature data extraction and required developing models of both environmental suitability for schistosomiasis transmission (used as a proxy for identifying populations at risk [PAR]) and prevalence within those at-risk populations. To estimate PAR, we used a boosted regression tree method to generate environmental suitability maps for schistosomiasis that used georeferenced data and geospatial covariates (including vegetation, precipitation, and population). Geotagged epidemiological information was paired with several hypothesised environmental drivers of schistosomiasis presence to define the environment that best characterises the reported occurrences of the parasite. We produced species-specific maps and determined the optimal thresholds for transmission using the geopositioned dataset. Species-specific maps were combined into one all-species schistosomiasis map to show areas that are environmentally suitable for transmission of schistosomiasis. We overlaid this map with population data at a scale of 25 km² to generate a national estimate of the total population living within areas that are suitable for schistosomiasis transmission. We used DisMod-MR 2.1, the Bayesian meta-regression tool developed for GBD 2016 estimations, to estimate prevalence within this PAR, such that environmental factors and localised data were both used to inform our prevalence estimates.

King and Galvani state that “many of the case count data remain inferred”. Although the prevalence model infers prevalence information from countries in similar regions, this prevalence model is representative of the PAR and only uses data in those at risk. These prevalence estimates were then scaled to represent prevalence at the GBD location level through the PAR estimates extracted from the environmental suitability maps, which used the association between environmental factors and more than 12 000 geolocated records.

King and Galvani also state that “severe and yet common clinical outcomes of schistosomiasis were not integrated into the disability weights” and “serious clinical manifestations of intestinal schistosomiasis were not incorporated”. We will address this in three parts. First, we thank the commentator for the suggestion to consider adding genital lesions as an outcome of schistosomiasis. We plan to review the associated literature on this outcome in a subsequent iteration of the GBD Study. We also will review the new evidence on the cognitive deficits associated with schistosomiasis, as presented in their referenced article. Until now, our assessment has been that the evidence for a causal link between schistosomiasis infection and loss of cognition was not strong enough to warrant inclusion in the GBD studies. Second, anaemia is a sequela of schistosomiasis that has been included since GBD 2010. Finally, the “minimal disability weights” assigned to schistosomiasis infection are derived from large-scale population surveys. Until new evidence emerges that challenges the values of these disability weights, we will continue to use the current values.

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Ellen M Goldberg, David Pigott, Shreya Shirude, Jeffrey Stanaway, Simon I Hay, *Theo Vos
tvos@uw.edu

Institute for Health Metrics and Evaluation, Seattle, WA 98121, USA