Hahn and others\textsuperscript{1} have recently written a perspective piece, which was published in \textit{The American Journal of Tropical Medicine and Hygiene}, criticizing our study published in 2013.\textsuperscript{3} Here, we respond to their critique, commenting and clarifying some of the points raised. Our response is organized in the same order as the issues were raised.

Hahn and others\textsuperscript{1} provide literature that supports their view that intact forests can help eliminate local malaria transmission. They\textsuperscript{2} place special emphasis on a study that was based on a theoretical model parameterized to a different vector and applied to a completely different ecosystem (~1,000 km away from our study region) on a region that has not had any reported malaria cases for the past 30 years.\textsuperscript{5} Unfortunately, Hahn and others\textsuperscript{1} fail to acknowledge the large literature that support the opposite view regarding the role of forests, and most of those studies were conducted in the Brazilian Amazon.\textsuperscript{4–10}

Hahn and others\textsuperscript{1} claim that it is problematic to assume a constant population given that the Brazilian Amazon population increased from 2000 to 2010 by 23\%. First, this statement is misleading, because the length of our study corresponds to less than one-half of this time interval. Second, population data arise from the Brazilian Census, which was conducted in 2000, 2007, and 2010. To account for fluctuation in population size, one would have to interpolate between three data points for each county, and it is not clear if this method is a better solution than adopting the 2007 population count for the 2004–2008 study period. Nevertheless, we performed our analysis again (this time using only 2007 malaria data) and found that our original conclusions hold (results available on request).

Hahn and others\textsuperscript{1} then criticize the fact that we excluded rural health facilities and the two easternmost states in the Brazilian Amazon (Maranhao and Tocantins). First, we did not have data from Maranhao and Tocantins, and therefore, these data were not excluded. Second, as explicitly mentioned in ref. 2, we excluded the rural health facilities because we did not have their spatial coordinates, thus precluding the assessment of the effect of proximity to forests. Third, the remark that we only accounted for 4.8\% of the Brazilian Amazon region is misleading, because it ignores the fact that the human population in this region is highly clustered in the vicinities of established cities.\textsuperscript{11,12} Even if we had the geographical location of all health facilities, it is likely that the sum of their catchment area would still only account for a small proportion of the overall area. To dispel any questions regarding selection bias, we use all (urban and rural) available data from 2007, this time assuming that all health facilities are located in the vicinity of the established cities. We find that the same results still hold, regardless of adoption of a 20- (as in the original analysis) or 50-km buffer size (which encompasses the great majority of the population in each county; results available on request).

Hahn and others\textsuperscript{1} suggest that our analysis suffers from the classic ecological fallacy. Any analysis that aggregates data potentially suffers from this problem. However, aggregate data is often the only available data, particularly at the spatial scale of our analysis. Examples of studies that rely on aggregate data abound (including studies by the critique authors themselves\textsuperscript{13–15}), providing important insights regarding large-scale drivers and spatial patterns of disease risk. Furthermore, our findings do corroborate the results of several entomological and epidemiological site-specific studies in the Brazilian Amazon. Hahn and others\textsuperscript{1} then criticize the land use/land cover classification product that we used in our analysis. Interestingly, Hahn and others\textsuperscript{1} have also used the same remote sensing product to implicate deforestation in malaria risk.\textsuperscript{14} Finally, Hahn and others\textsuperscript{1} emphasize results from the works by Vittor and others\textsuperscript{16,17} on \textit{Plasmodium vivax}, while ignoring \textit{P. falciparum} results from the same study, despite \textit{P. falciparum} comprising approximately 40\% of all detected infections. The PhD thesis of Vittor,\textsuperscript{18} which is the basis of the claims by Hahn and others,\textsuperscript{1} indicates that \textit{P. falciparum} prevalence was negatively associated with deforested land, and these results directly conflict with their mosquitio and \textit{P. vivax} data.\textsuperscript{16,17} These \textit{Plasmodium} results were never published in a peer-reviewed journal because of the low numbers of detected infections (110 infections of a total of 2,938 individuals examined). However, Hahn and others\textsuperscript{1} do not hesitate to selectively report the results from \textit{P. vivax} to support their claim.

Hahn and others\textsuperscript{1} say that we ignore the fate of the cleared forest in our analysis. However, they do so in their earlier analysis, which pointed to deforestation as an important malaria incidence driver.\textsuperscript{14} Furthermore, they assert that...
(1) deforestation results mainly from timber production and mining in Para rather than pasture/cattle ranching and soybean and (2) protected areas (PAs) tend to be located in areas of high deforestation pressure. These assertions are incorrect and shocking for anybody that knows this region.19,20 Finally, Hahn and others1 criticize us for not distinguishing among different classes of PAs on malaria risk.

We combined all PAs because we were not interested in comparing the effect of different classes of PAs on malaria risk.

The role of biodiversity in decreasing disease risk has been and will probably continue to be the theme of a heated debate.22–26 However, to criticize the methods we employed while also making use of them in their most recent study published in 201427 is, at a minimum, awkward. To effectively move this debate forward, we have to focus on more constructive ideas and suggestions. To this end, one of the critique authors (i.e., Amy Vittor) and I have partnered to reanalyze the mosquito data in refs. 16 and 17 and review the evidence regarding the role of forests in malaria risk, hoping to gain a more coherent picture of what is known about this important relationship. I invite the other authors of the critique to be part of this new exciting work.

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REFERENCES