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## The ecology of climate change and infectious diseases: reply

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Paul Epstein (2010) objects to my hypothesis that climate change should result in shifts (as opposed to net increases) in the distribution of infectious diseases (Lafferty 2009*a, b*). My first exposure to the link between climate change and infectious disease came from a Scientific American article by Epstein (2000) showing maps of malaria transmission risk increasing dramatically in New England and Europe. I used these maps to frighten my North American students into caring about malaria. In 2005, I attended a Cary Conference where David Rogers and Sarah Randolph challenged Epstein's view that climate change would unilaterally increase disease. They advocated two-tailed hypotheses (disease can go up or down), followed by sophisticated analyses that control for temporal and spatial confounds and apportion variance in disease to climate and myriad other factors. Those talks convinced me ecologists possessed the skills needed to address this important issue and that a review aimed at ecologists might help get them into the game.

Epstein gives two arguments for *why* disease should respond positively (but not negatively) to climate change. The first considers that the future might have warmer winters and disproportionate warming at higher latitudes. As a result, the globe would not necessarily be hotter than at present, just less cold. The tropics would stay the same and the temperate latitudes would become

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more tropical. I agree with Epstein that this scenario could lead to net expansions of climate suitability for some tropical diseases, though it would remain to be seen whether this expansion in suitability could trump other factors, such as economics. I'm less convinced by Epstein's second argument that diseases will generally increase if climate disruption leads to more extremes in temperature and precipitation. He notes that an increase in climate variance could change climate patterns from unimodal bell-shaped to bimodal, destabilizing ecological relationships and increasing infectious disease. However, one can make equally plausible arguments that environmental disruption can impair infectious disease transmission. Epstein bolsters his argument by selectively focusing on the warm mode of the bimodal pattern (tropical storms and hurricanes) that can favor mosquitoes, while ignoring the other mode of hard frosts and droughts. Then, he gives examples of how some infectious diseases will increase with rain and others will increase with drought. He's right; diseases will increase whether it gets drier or wetter, but just some diseases in some places. Other diseases, in other places, will likely decline. To assume otherwise is to suggest that infectious diseases are so poorly adapted to current conditions that any change will favor them. This is opposite to the argument made by conservation biologists for how climate change can impact biodiversity. For Epstein to be right, either conservation biologists are misled that climate disruption will impact biodiversity or parasitic and free-living organisms play by opposite rules of adaptation.

While most review papers (including several by Epstein) emphasize potential increases in disease with climate change, many of the recent empirical papers on the subject convincingly argue the two-tailed hypothesis of disease shifts with climate changes. An important recent paper suggests how climate change in Africa will shift the suitability for malaria, resulting in net decreases in disease (Peterson 2009). However, like Epstein, some empirical studies embrace an increase in disease by simply ignoring areas where disease might decrease. For instance, Meerburg and Kijlstra (2009) conclude that the common human protozoal parasite, *Toxoplasma gondii*, will increase in incidence as climate changes. They note that *T. gondii* is more prevalent in tropical countries, then use a climate change scenario that predicts which parts of northwestern Europe will become warmer and wetter, assuming a direct correspondence between these

climate measures and *T. gondii* distribution. They conclude that a human vaccine against *T. gondii* is essential. Beyond the lack of any formal biological model or statistical-based linkage between climate variables and *T. gondii* incidence, the paper focuses exclusively on the potential for *T. gondii* to increase. Lacking from their key figure are the parts of Europe (Spain, Portugal, and France) where one would expect decreases in suitability for *T. gondii* (cysts perform poorly if it is too hot or too dry). The sole mention of these areas is that climate change is likely to affect *T. gondii* in southern Europe, with no mention of the direction of change. Obviously, a decline in toxoplasmosis throughout larger areas of Europe undercuts the idea of an increased need to develop a human vaccine for toxoplasmosis. Emphasizing examples of disease increases while downplaying examples of decreases, generates more concern than enlightenment.

Meta-analyses could test questions such as: How much is climate change likely to shift the suitability for infectious disease, and how does this vary among diseases? What will the net result be in terms of land mass or individuals exposed? What other factors explain variation in these don't distributions? What fraction of studies test for negative as well as positive responses, implement stationarity, or avoid extrapolation beyond the input data? We might also review the reviews to see if their emphasis is representative of the empirical data.

In closing, I thank Paul Epstein for his early recognition of the link between climate change and infectious disease, genuine concern for global human health issues, and contribution to this discussion on climate change and infectious disease.

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