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Letter

Response to Charlier *et al.*: Climate–Disease Feedbacks Mediated by Livestock Methane Emissions Are Plausible

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Uncovering links between climate change and infectious disease is a pressing global health challenge. Our article called attention to potential positive feedbacks between climate change and animal infectious diseases via methane emissions from hosts [1]. Studies on the effects of climate on infectious diseases far outnumber studies on the reciprocal question. Therefore, much work is needed to assess whether such feedbacks are plausible and how to mitigate them. Given the scarcity of data on how diseases impact climate, we used three case studies to illustrate how diverse parasites affect methane emissions: helminths in sheep, bacterial mastitis in dairy cows, and rinderpest virus in wildlife. For helminths and sheep, we used a worked example to underscore the point. This exercise aimed to stimulate multidisciplinary discussion between scientists and policy-makers whose input is required to address climate–disease feedbacks. Charlier *et al.* [2] argue that more empirical data, interdisciplinary collaboration, and modeling will help identify and address the problem. We emphatically agree. However, the assertion that our worked example is unrealistic because of its simplifying assumptions and specific context, too casually dismisses the rationale underlying our hypothesis: that reciprocal, positive, feedbacks between climate and disease may operate across multiple livestock–parasite systems. Here, we re-emphasize the merits of this idea and outline specific steps to evaluate the hypothesis.

Which Infectious Diseases Are Most Sensitive to Climate Change?

Understanding climate–disease feedbacks requires the bidirectional study of climate and disease. In our example, we made no *a priori* assumptions about the magnitude or form of the relationship between climate and infection rates. Rather, we explored how methane emissions could change over a range of variation in helminth prevalence (from 0 to 100%). However, a growing number of studies are developing models that can critically evaluate how infection rates depend on climate [3–5]. For example, projections based on the thermal mismatch hypothesis – the idea that disease risk will be greatest for hosts from cooler and warmer climates when temperatures are abnormally warm or cool, respectively – suggest that multiple parasites (bacteria, fungi, helminths, and viruses) should experience a net increase in prevalence at higher latitudes and a net decrease at lower latitudes under changing climatic conditions [3]. Models like these can provide a mechanistic framework for linking changes in climate to variation in infection rates, thereby identifying geographic regions and parasite or host attributes that may contribute disproportionately to positive climate–disease feedbacks. Once critical host–parasite pairs are identified, integrating epidemiological models with metabolic theory [6,7] could enable further insight by accounting for the temperature dependencies of infectious disease impacts, enteric fermentation or other processes that produce methane, and their possible interactions.

How Will Changes in Infectious Diseases Affect Methane?

Few studies have investigated how infectious diseases affect methane emissions. In a rare experimental study [8], growth rates and methane emissions of lambs with and without subclinical helminth infection were quantified. We used these data to explore how individual-level effects of this magnitude might translate into population-level emissions if none (reflecting how current livestock emissions are estimated) or all of the global livestock population were infected with helminths. Our estimate illustrates how large an effect these parasites could exert on global methane emissions, serving to contextualize the potential problem. However, understanding the true magnitude of the effect of any parasite on methane requires a rigorous approach that accounts for variation in many interrelated variables (Figure 1).

For example, Charlier *et al.* [2] suggest that considering severity of infection (i.e., intensity) is key for helminth parasites. We agree; however, no studies have examined how variation in helminth intensity affects methane emissions. Indeed, infection intensity/severity is not only important for helminths. Viral and bacterial infection severity can also influence methane production. For example, for dairy cows during first lactation, estimated enteric methane emissions increased with increasing severity of mastitis infection [9]. Thus, multiple sources of variation, such as intensity of infection, stage, and co-infection should be considered in studies that quantify effects of parasites on methane; likewise, host and habitat attributes must also be considered (Figure 1). Finally, although some effects of infection on livestock methane emissions are attributable to production inefficiencies (e.g., slower weight gain), others are not [8]. Therefore, studies on the mechanistic basis of parasite effects on methane yield are also essential.

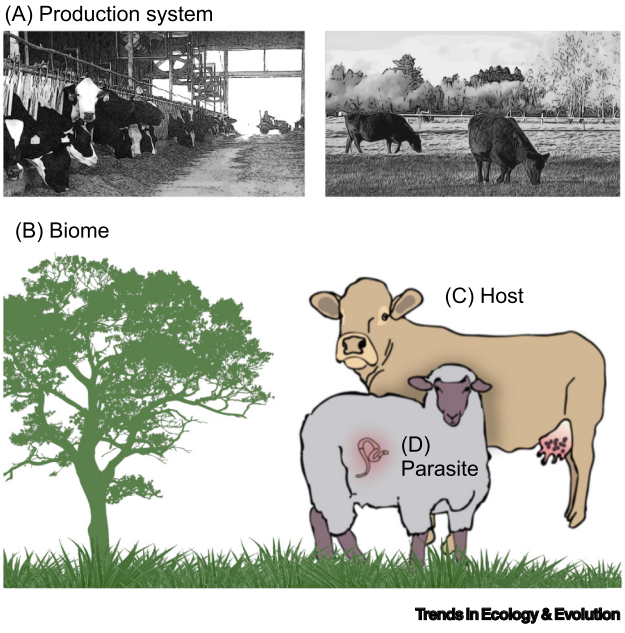

Figure 1. Multiple Interrelated Variables Influence How Infectious Diseases Impact Methane Emissions in Livestock. Habitat-level attributes, including the (A) production system (e.g., dairy farming or pastoralism) and (B) biome (e.g., tropical savanna or temperate woodland) will affect animal management, production targets, and the influence of disease intervention strategies; (C) host attributes, including life stage, will determine relative contributions to methane emissions and the infectious diseases of most concern; and (D) parasite attributes, ranging from the type, timing, and intensity/severity of infection to the occurrence of co-infections, will shape both indirect (i.e., production-mediated) and direct effects of disease on methane.

Images in (A) were modified from 20110419-RD-LSC-0599 and 20170426-KS-Reclamation-0108 by USDAgov, which are marked under CC PDM 1.0.

Moving Forward

We suggest that climate–disease–methane interactions are among the many pathways by which future changes in climate and infectious diseases may be exacerbated. Crucially, this pathway may be general, operating across diverse parasites, agricultural systems, and spatial scales. This hypothesis requires testing. To make rigorous predictions, multidisciplinary work must determine which interacting factors need to be explicitly represented in models and which can remain implicit. We do not claim to have performed such an exercise. On the contrary, our purpose is to introduce the hypothesis, launch a discussion on its merits, and call for its testing.

Declaration of Interests

No interests are declared.

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