Xavier Rodó

Institut Català de Ciències del Clima |
Barcelona, Spain
xavier.rodo@IC3.cat

Resolving the interplay between climate forcing, transmission, host immunity and intervention measures in dynamic approaches to infectious diseases

The full quantification of the mechanisms accounting for the coupling of climate to the dynamics of infectious diseases is poorly served by the scarcity of long-term time-series. At present there are less than twenty long-term data sets for infectious diseases from which we can construct predictive models for regional disease outbreaks. Shorter spatio-temporal records exist where the high spatial resolution can provide alternative valuable information on disease dynamics (e.g., Chikungunya outbreaks of Reunión island, and the recent cholera epidemic in Haiti). One way to quantify the influence of climate variability on rates of transmission relates to the reproductive number of an infectious disease and consists of examining changes in infection with host age and then predicting how climate-driven changes in transmission affect this relationship. Related quantities that consider only part of the transmission cycle by focusing on the vector, such as vectorial capacity and the critical density threshold of vector populations, have also been used to produce risk maps as a function of climate change. However, the population dynamics of cholera, malaria and other key water-borne, and vector-transmitted pathogens have the potential to resonate with both annual and longer-term climate cycles to produce more complex patterns of long-term epidemic behaviour. It has been recognized that the propensity of disease dynamics to oscillate with the waxing and waning of herd immunity in the host population, can place significant constraints on the ability to detect potential climate signals in time series records for important tropical diseases. Innovations in quantitative approaches have been developed to retrospectively disentangle the effects of endogenous disease dynamics, for example, as the result of 'herd' immunity, vs. exogenous drivers such as climate variability. Lessons learnt may not transfer in a simple way to the case of vector-borne infections, because of the more complex transmission cycle as well as the more complex nature of immunity acquisition and loss that is intimately related to the antigenic diversity of the parasite. I will illustrate how decoupling occurs between incidence and climate forcing in a model of malaria transmission that incorporates parasite diversity in the form of superinfection. or alternately how the coupling can facilitate strain dominance in historic cholera pandemics. The interaction of control efforts with climate variability should be of particular relevance when intervention measures are implemented in response to previous increases in incidence, for example for residual insecticide spraying in desert malaria in NW India. A clear need manifests to develop a new class of semi-mechanistic models with limited parameters to minimize confounding effects of over-parameterization. Current approaches tend either to include a myriad of parameters that cannot realistically be specified in ordinary differential equations systems, or conversely, use over-simplistic statistical methodologies that are at most extrapolated, and provide little expectation of success in the future for different environmental ranges.