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## Commentary: Japanese Encephalitis Virus as another emerging infectious disease: is a lack of epidemiological tools the pig in the room?

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The recent (and ongoing) pandemic of COVID-19 is the worst emerging infectious disease (EID) we have seen for some decades—the worst since HIV in the 1980s. Despite a historical ‘honeymoon period’ when it was thought that medical science could overcome the infectious disease

threat to population health,<sup>1</sup> EIDs are here to stay. ‘New’ pathogens, like SARS-CoV-2 (the virus that causes COVID-19) and HIV, continue to emerge, generally accompanied by evidence of one or more of the characteristics that define EIDs: increasing case numbers, greater

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severity and wider geographic distribution.<sup>2</sup> Such new pathogens are virtually impossible to predict and correspondingly very difficult to control, despite some understanding of the conditions under which new pathogens evolve, and state-of-the-art public health infrastructure and advanced vaccine technology in many countries. With new pathogen epidemics, public health can be forgiven for struggling with control. Why then, one might ask, do we seem to have the same problem with well-known pathogens that intermittently (re-)emerge? For example, Japanese Encephalitis Virus (JEV) recently emerged for the first time in continental Australia, killing a small number of unfortunate people in four states<sup>3</sup>—yet this is a well-known arbovirus, with a well-understood ecology, intensively researched epidemiology and an existing vaccine. There are many answers to that ‘why then’ question, including local and global political, economic and social drivers that get in the way of implementing evidence-based infectious disease control. There is a role for epidemiology to contribute further to our understanding of intervention points that may be implementable despite such barriers and Walsh *et al.*<sup>4</sup> provide one such contribution in this issue.

As the reader will discover, Walsh *et al.*<sup>4</sup> use modelled JEV outbreaks based on case data and a range of (cleverly adjusted) environmental variables to analyse the landscape ecology of disease transmission. There are several strengths to the way this work deals with the complex and multidisciplinary drivers of disease ecology. An accurate assessment of exposure, accounting for potential confounders, is the bane of environmental epidemiology studies and the authors manage this challenge skilfully—despite the inevitable limitations of available, routinely collected data that are not necessarily intended for research purposes. For example, their adjusting for health system performance so that the ability to detect cases is not biasing the location and ecological correlates of outbreaks demonstrates a breadth and depth of integration that is difficult to achieve. Similarly, data on reservoir hosts (wading birds) and amplifying hosts (pigs) were weighted to account for potential reporting bias (however, data on domestic holdings of singleton chickens or pigs would arguably be limited). In a clever and cost-effective approach, they draw on existing environmental databases to allow the modelling to include hydrology, standing surface water, agricultural data and climate. Although habitat suitability modelling is well established in ecology,<sup>5</sup> it is not as often applied appropriately to the reservoirs and vectors of zoonotic pathogens in integrative epidemiological studies as it has been here. The modelling by Walsh *et al.*<sup>4</sup> is elegant in both ecological conception and biostatistical rigour.

Unfortunately, there is also a ‘but’ to add. The (accurately and intelligently derived) results show that wading birds and

mosquitoes occur in wetlands and that the risk of mosquito-borne disease outbreaks is highest there; we knew that. The study demonstrated that when humans impinge on such natural ecosystems with agriculture generally and animal husbandry particularly, the risk of zoonotic disease transmission increases; we knew that too. The findings also confirm that the presence of reservoir animals and amplification hosts is associated with outbreaks and that vulnerable communities are at greatest risk; we also knew that. We even knew that ‘ecotones play a role in a number of the most important EIDs’.<sup>6</sup> One might ask then whether the knowledge generated actually has ‘impact’—impact in the sense of what governments and philanthropists are now increasingly seeking to fund: work with demonstrable gains in one or more of the areas captured by a ‘triple or quadruple bottom line’ approach. Without taking anything away from the authors, do we really need more, bigger and better epidemiological studies? Or do we already know enough to refocus our efforts more on research translation, intervention studies, implementation science and advocacy? To better inform the policy makers and funders who are key to converting our epidemiological knowledge into a reduced disease burden on the ground, we need to provide tools and analyses that can predict the impact of our interventions—optimizing the use of limited resources. For JEV (and other EIDs) in complex ecological systems, there is an elephant—or pig—in the room: the ability to model the impact of interventions on ecosystem health and human health concurrently.

If, for example, our public health intervention consists of moving a piggery in an ecologically informed direction—away from an ecotone<sup>4</sup> or down a hill<sup>7</sup>—to reduce the risk of catching an environmentally mediated disease, then a policy maker might like to know what the trade-off would be between ecosystem disruption (with potential impacts on biodiversity and ecosystem services) and the morbidity and mortality of the human population (who are affected both directly by the disease risk and indirectly through ecosystem services). To answer such questions would require a cross-sectoral model that simultaneously estimates the impact of any proposed environmental interventions on both the environmental sector and health sector, but a recent comprehensive systematic search for any such infectious disease modelling tool by Stanhope *et al.*<sup>8</sup> turned up nothing. These authors provide guidance on how such a model may be developed, including identifying the range of data sources, spatiotemporal scales and system requirements that would need to be integrated into such a tool, and highlight the potential benefits of a hybrid-ensemble approach that integrates individual modelling techniques from other sources. The result would provide a means of prioritizing informed environmental interventions that

optimize the ‘net’ benefit—a godsend to planners and policy makers in both the health and the environment sectors.

Walsh *et al.*<sup>4</sup> provide an excellent example of how a rigorous and well-thought-out approach can identify drivers of infectious disease transmission and outbreaks in environments with even the most complex disease ecologies. Such studies have provided us with the understanding that should now allow us to take the next step and translate our understanding into action. Epidemiology can and should remain at the forefront of this challenge, with the development of new tools and analyses that can better support cross-sectoral decision-making and advocacy to sustainably improve public health.

### Conflict of interest

None declared.

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