

pattern for rainfall, but being a tropical country, we do not have four seasons as described by the authors. (2) The authors referred to our study published in 2008 [4] as evidence for diversity of serovars during the 2008 outbreak. This report was for the 2002–2003 period, not for the 2008 outbreak. (3) The authors referred to the report on interim analysis of the 2008 outbreak and mentioned that nine serovars were isolated. There has been no published literature on serovar isolation from Sri Lanka recently. The citation in the paper was based on results of the microscopic agglutination test. (4) In the paper, the authors used MOH areas as the unit of analysis, and MOH was defined as ‘Ministry of Health’. This is incorrect – MOH areas are ‘Medical Officer of Health’ areas, which are divisional-level health administrative units in Sri Lanka.

We also were very interested as to why authors reported the ‘prevalence’ of leptospirosis. Conventionally, we express leptospirosis disease as incidence because it is an acute condition.

Declaration of Interest

None.

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The authors reply

We thank Dr Agampodi for his comments on our paper describing recent spatial-temporal patterns in suspected clinical leptospirosis in Sri Lanka, and hope to address some of the concerns raised in the letter. The first main criticism, being the validity of the surveillance data used in this analysis, highlights a general shortcoming of performing epidemiological analysis over large geographical areas in countries with inconsistent surveillance and reporting when timely diagnoses is neither sensitive nor specific. We acknowledged this issue throughout the paper, discussing the possibility of hantavirus or dengue presenting as leptospirosis, not to mention entitling our paper ‘suspected clinical leptospirosis’ as a way of further highlighting this uncertainty. This of course begs the question, whether it is worth doing analysis of risk for cases with uncertain diagnoses, perhaps due in part to variation in clinical practice. We would argue that this type of analysis is necessary for these data because of the uncertainty associated with such diagnoses. One of the key purposes of surveillance data is to monitor trends in the health status of populations, what labels we attach to these conditions matter less than the fact that the number of people with acute febrile illness was unusually high. So faced with this uncertainty, we looked for correlative risk factors. Geographical risk analysis of surveillance data at the scale done here is by its very nature exploratory and inductive.

In the analysis presented, we detected clusters of cases in space and time, correlated these clusters with risk factors, interpreted patterns in light of the probable mechanisms, and concluded with avenues for future research. To address the specific criticisms raised by Agampodi, the paper [1] which describes its aim to validate the leptospirosis case definition in Sri Lanka using the microscopic agglutination test (MAT), does not report variation in clinical practice as a limitation in that study. It is therefore not unusual that we would use its findings as supportive evidence for doing a geographical risk analysis based on surveillance data, despite our noted warnings about misdiagnosis and clinical uncertainties. We also

highlighted studies from Brazil [2] and India [3] which showed how leptospirosis is clinically indistinct and thus misdiagnosed as hantavirus. The purpose of our analysis was not definitively to confirm the number of cases, but rather to identify geographical risks associated with the outbreak.

The second major criticism relates to the currency of the survey mapping data representing paddy fields. Agampodi rightly points out that the amount of land cultivated for rice paddy during the period of study changed and this was not factored into our analysis. We recognize that the interaction between food prices, government policies promoting local food production (i.e. rice cultivation), and thus greater exposure risks in the population has been posited as a cause of the outbreak. The theory that rodent populations in abandoned fields were allowed to increase, and that the policy change in 2007 led to the increased exposures and cases is an intriguing hypothesis. We would only like to note on this point that we are in the process of looking at ways of mapping annual rice paddy areas based on classification of remotely sensed imagery and look forward to reporting results of this analysis. Finally, accuracy of the rice paddy basemap was checked qualitatively for areas where recent high-resolution land-use data was available and maps did seem to roughly coincide in those areas, although this was not a rigorous accuracy assessment.

Some final issues raised concern terminology of seasons (we correctly described variation in rainfall in terms of monsoon seasonality, *yala* and *maha*) and only described case distributions with reference to seasons common in the northern hemisphere. This description was intended to improve clarity by using more familiar descriptors for readers not versed in Sri Lankan monsoon seasons. Agampodi also rightly notes that we indicated that his serological results [4], were used as evidence of some of the serovars likely in circulation were from 2002–2003, and not 2008. This is true, and we acknowledge this discrepancy, although the point was mainly to highlight that the key serovars are largely unknown, and it is not unlikely that those in circulation in 2003 may also be 5 years later. The last substantive comment questions our

citation of serovars reported in the interim report [5], which are reported in Table 5, and described as serovars isolated from patients in Sri Lanka in 2008 based on analysis conducted at the Veterinary Research Institute.

We greatly appreciate some of the points raised in this letter and are very thankful for Dr Agampodi's expertise on this subject. His work has been instrumental to our own and we hope that some of the major issues raised have been clarified. Often the nature of large-area risk-factor analysis of surveillance data comes at the cost of specificity and sensitivity; however, with leptospirosis, we are confronted with clinical misdiagnosis, ineffective laboratory tests, and a multitude of potential reservoirs and transmission pathways. This complexity underscores the need for both large-area analysis of risks in the environment, individual-level studies, and laboratory studies, and thus integrative work between biologists, sociologists, geographers, epidemiologists, veterinarians, and physicians.

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