Correspondence

Spatial epidemiology of leptospirosis in Sri Lanka

To the Editor

Robertson and colleagues [1] analysed the spatial epidemiology of leptospirosis in Sri Lanka and showed a probable correlation between occurrence of leptospirosis and rainfall patterns in Sri Lanka. They also identified risk clusters for leptospirosis based on the spatial distribution of the reported cases. We appreciate this work which filled several knowledge gaps in the epidemiology of leptospirosis in Sri Lanka. However, we are concerned about some facts in the paper which need further explanation and corrections.

The disease rates in the paper were analysed using routinely reported data. Authors have correctly described that these were not confirmed cases. They further provide evidence to suggest that the reported cases would be valid for this analysis. The supportive evidence [2] provided for validity of reported cases was not actually for the reported cases. We carried out the particular study to validate the WHO proposed surveillance case definition. Although this case definition is recommended for case reporting, the clinical practice and reporting is completely different from this case definition. Since the laboratory diagnosis is not routinely available, reporting is entirely based on clinical judgement. We previously analysed this error in our study conducted during the 2008 epidemic, in Kandy, Kegalle and Matale, districts which showed that only 52.6% of the patients treated for leptospirosis actually had the disease. Furthermore, 46.2% of the patients who were treated for other conditions were retrospectively confirmed as leptospirosis cases [3]. To show the diversity of the clinical judgement we further analysed these cases by reporting hospitals. It showed that only 28.3% of cases from Matale were confirmed positive, compared to 58.3% and 55.0% in Kandy and Kegalle hospitals, respectively. According to the routinely reported data, Matale had the highest incidence of leptospirosis during the 2008 epidemic, which we proved as not the correct figure. It is clear that Matale had a higher number of cases during the 2008 outbreak. However, fewer than one third of the suspected and reported cases actually had the disease and it was significantly lower than in other areas. The reported data available in the surveillance system seems to be an overestimation of leptospirosis incidence in Matale and we suspect that this had affected the Robertson study in which the authors discussed Matale specifically. To further explain this diversity of clinical judgement, we looked at the cases reported from the three medical units in Kandy hospital. While one unit reported 54 clinically suspected cases during our study period of 4 months, the other two units each reported fewer than 10 suspected cases. This difference is highly unlikely, given that the same number of admission days are allocated to each unit. The most plausible explanation is that clinical suspicion varied widely according to treating physicians. While the 2008 outbreak of leptospirosis undoubtedly existed, exact case load and geographical distribution are questionable due to the lack of point-of-care diagnostic facilities and gross under-/overreporting of cases, based on the treating physician.

We have another major concern about the use of 2002 data for agriculture/paddy field distribution. In 2007, the government launched an island-wide programme (‘Api Vavamu, Rata Nagamu’), which made it mandatory to cultivate all abandoned paddy fields. This was seen especially in 2008, where a large number of people who were not traditional farmers got involved in paddy farming activities. This programme would have changed the paddy field distribution in 2002 considerably, and it may be a reason for not showing a significant association with the 2008 outbreak.

We would like to point out some other inaccuracies in the paper: (1) Sri Lanka does have a seasonal
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. Convention-
ally, 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 sur-
veillance data used in this analysis, highlights a gen-
eral 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, dis-
cussing the possibility of hantavirus or dengue pre-
senting 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 popula-
ations, 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 ex-
ploratory 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 prob-
able 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 un-
usual 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