EXPLORATORY SPATIAL ANALYSIS OF SOCIAL AND ENVIRONMENTAL FACTORS ASSOCIATED WITH THE INCIDENCE OF ROSS RIVER VIRUS IN BRISBANE, AUSTRALIA

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Abstract. We used geographic information systems and a spatial analysis approach to explore the pattern of Ross River virus (RRV) incidence in Brisbane, Australia. Climate, vegetation and socioeconomic data in 2001 were obtained from the Australian Bureau of Meteorology, the Brisbane City Council and the Australian Bureau of Statistics, respectively. Information on the RRV cases was obtained from the Queensland Department of Health. Spatial and multiple negative binomial regression models were used to identify the socioeconomic and environmental determinants of RRV transmission. The results show that RRV activity was primarily concentrated in the northeastern, northwestern, and southeastern regions in Brisbane. Multiple negative binomial regression models showed that the spatial pattern of RRV disease in Brisbane seemed to be determined by a combination of local ecologic, socioeconomic, and environmental factors.

INTRODUCTION

Ross River virus (RRV) infection is the most common vector-borne disease in Australia.1–3 It caused a large epidemic in 1979 and 1980 involving some Pacific Island nations.1 A recent study suggests that RRV is recurring in Fiji.4 This virus circulates enzootically in reservoir populations of marsupials in Australia. The virus is maintained in a primary mosquito-mammal cycle involving macropods (kangaroos and wallabies)5 and possibly other marsupials (e.g., possums),6 flying foxes, and native rodents.7 A human-mosquito cycle may occur in explosive outbreaks.8 Horses, dogs, and cats, which may act as amplifier hosts, also appear to develop joint and nervous system disease after infection with RRV.9 Mosquitoes are efficient vectors of the disease because of their susceptibility to the virus and the readiness with which they bite reservoir as well as human hosts.10–11

At least 20% of infected individuals develop an acute disease. The disease is characterized by headache, fever, rash, lethargy, and muscle and joint pain, with approximately 50% of patients having a rash.12,13 The arthritic symptoms may persist for months and can be severe and debilitating. There is no effective treatment for the disease; therefore, prevention remains the vital public health strategy. From 1992 to 2005, more than 58,749 laboratory confirmed cases of infection with RRV have been reported to the Department of Health and Aged Care in Australia.14 Infection with RRV appears to have increased in Queensland, Australia in the past decade, particularly in the Brisbane area.14,15 Possible causes for this increase include inadequate urban planning, increased tourism, ecosystem change, and increased awareness of this disease among health professionals and the general public.16–18 For example, in seasons with high temperatures and rainfall, the vegetation upon which kangaroos depend will flourish, and more non-immune reservoir hosts will be added to the temporally and spatially expanding population.18 However, spatio-temporal variations of RRV and key determinants of such variation need to be further examined.19–22

The purpose of this study was to identify the spatial distributions of reported RRV infections using geographic information system (GIS) and exploratory analysis and to identify major socio-ecologic determinants of RRV transmission in Brisbane, Australia.

MATERIALS AND METHODS

Study area. Brisbane is the capital of Queensland, Australia and is located in the southeastern corner of the state. Queensland is the state with the highest incidence of RRV disease in Australia over the last decade.14 Brisbane has a subtropical climate and rolling, undulating hills, which are conducive to an outdoor lifestyle. Within the administrative boundaries of Brisbane City Council, which also determined the study area of this investigation, the population size was 883,449 on July 1, 2001 (Figure 1).23 Brisbane was chosen as the study site because it has the highest number of cases of infection with RRV in the state, and RRV disease is a significant public health issue in this rapidly growing metropolitan city.

Brisbane consists of 162 statistical local areas (SLAs). We obtained a variety of data from different sources collected in 2001 when the latest census was undertaken. These included a computerized data set of the notified monthly cases of infection with RRV in Brisbane from the Queensland Department of Health, as well as data on a variety of population characteristics including overseas visitors (usually resident in Australia for less than one year), indigenous population, labor workers (as defined by the Australian Standard Classification of Occupations),24 educational level and family income by SLA from the Australian Bureau of Statistics (ABS) (Canberra, Australia), an extensive environmental GIS database with vegetation from the Brisbane City Council, and monthly Southern Oscillation Index (SOI) data from Australian Bureau of Meteorology. The SOI data varied temporarily but not spatially at the SLA level.

Data preparation. MapInfo Professional software was used to display the spatial and temporal distributions of RRV cases.25 The digital base map data sets used for constructing the GIS were obtained primarily from the ABS. These data were manipulated to facilitate the accurate identification of
the spatial locations of SLA and their linkages with the other data layers. The locations of case of infection with RRV by SLA were geo-coded to the digital base maps of localities using MapInfo and Microsoft Access software.

Spatial distribution of RRV. The spatial analysis was performed at the SLA level. Standardized incidence rates (SIRs) were computed using the direct method of standardization, with the total population in Brisbane in 2001 as the standard population because the different age structure in different SLAs might affect the likelihood of RRV infection. Smoother transitions were applied using a continuously varying color spread to reduce constrains caused by political boundaries (i.e., SLAs). The spatial distribution of RRV was analyzed using MapInfo professional software package.

Spearman correlation analyses were conducted to assess the bivariate associations between the monthly SIRs of RRV and potential determinants of RRV disease. To examine the impacts of social and environmental factors on RRV disease, where data were over-dispersed relative to the Poisson distribution, a generalized linear model was adopted with a negative binomial link. The potential determinants of RRV incidence comprised human population density, the proportion of indigenous population, the proportion of overseas visitors, average family incomes, the proportion of labor workers, the proportion of population with lower educational attainment (only attended primary school), SOI at a lag of three months, vegetation proportion and seasonality (coded as spring: September-October-November; summer: December-January-February; autumn: March-April-May; winter: June-July-August). These variables were selected on the basis of our professional knowledge and the availability of data. The SOI at a lag of three months was chosen because it appeared to have the strongest correlation with RRV incidence ($r_s = 0.27$). To control for autocorrelation, a first-order autoregressive term was fitted in the model. Analyses were performed using the Statistical Analysis System software with the GENMOD procedure.

Spatial autocorrelation is defined as an auto-correlated association of a certain variable with its spatial location. We used semivariogram analysis to investigate the spatial structure and spatial autocorrelation of RRV transmission. The semivariogram estimates how data are related with distance and direction, and presents the variability between pairs of observations that are separated by the same distance. If there is spatial autocorrelation in the data, values are typically low and the semivariance increases with separation distance. Semivariogram values were calculated on the basis of model residuals. The semivariogram analyses were performed using S+ SpatialStats.

RESULTS

Spatial pattern of RRV incidence. Figure 1 shows the distribution of raw cases of RRV infection. Figure 2 indicates a
substantial variation in incidence rates of RRV by SLA in Brisbane. The SIRs were \(\geq 163/100,000\) for 6 SLAs (4%); \(\geq 82 \text{ to } < 163/100,000\) for 13 SLAs (8%); \(\geq 38 \text{ to } < 82/100,000\) for 43 SLAs (26%); \(\geq 10 \text{ to } < 38/100,000\) for 55 SLAs (34%); and \(\geq 0 \text{ to } < 10/100,000\) for 45 SLAs (28%) in 2001. The SIR was highest in Rochedale (354.3/100,000), and the average SIR was 39.2/100,000.

**Bivariate analyses.** Table 1 shows the linear associations between the SIRs of RRV and the socioeconomic and environmental determinants in Brisbane. It also summarizes the bivariate linear relationships between all the independent variables. Both human population density and SOI at a lag of three months were all significantly associated with RRV disease. Figure 3 shows the age-standardized distribution of RRV disease by SLA in Brisbane. It appears that there were a few hot spots that were located in the northeastern, northwestern, and southeastern regions of Brisbane. The northeastern suburbs are characterized by wetlands and extensively covered with mangrove communities and paperbark swamps; the western suburbs are associated with Gold Creek and dense woodlands with the Nerang-Beenleigh alliance, and the southeast suburbs are typically densely vegetated areas with broad-leaved white mahogany, bloodwood, and smooth-barked apple woodland around Tingalpa Creek.

**Association between RRV incidence and socioeconomic factors.** After consideration of multicollinearity, family income was subsequently excluded from models because of its high correlation with the proportion of residents with lower education \((r_s = -0.79)\), the proportion of the indigenous population \((r_s = -0.67)\) and the proportion of labor workers \((r_s = -0.75)\). The negative binomial regression models showed that RRV disease incidence in Brisbane was significantly associated with several variables (Table 2). These were SOI at a lag of three months \((RR = 1.12, 95\% \text{ CI } 1.06–1.17)\), the proportion of people with lower levels of education \((RR = 1.02, 95\% \text{ CI } 1.01–1.03)\), the proportion of labor workers \((RR = 0.97, 95\% \text{ CI } 0.95–1.00)\) and vegetation density \((RR = 1.02, 95\% \text{ CI } 1.00–1.04)\) (Table 2). This suggests that on average in Brisbane, there were 25 more cases a year for an increase of 10 units of SOI, 23 more cases for an increase of 10% of the people with lower levels of education, and 23 more cases for an increase of 1% of the area covered by vegetation. However, there was a decrease of 21 cases a year for an increase of 10% of the labor workers in Brisbane. The semivariogram analyses indicated little spatial structure of residuals in the distribution of RRV disease after accounting for the above variables (the data are available on request). Overall, the model fitted the data well because the residuals were randomly distributed.

**DISCUSSION**

The results of this study indicate that there was remarkable variation in the spatial distribution of RRV incidence in Bris-
bene. This incidence was statistically significantly associated with SOI at a lag of three months, the proportion of people with lower levels of education, the proportion of labor workers, and vegetation density. However, the strength of these associations was generally weak and varied with each independent variable. The spatial pattern of RRV disease in Brisbane appeared to be determined by a combination of local ecologic, socioeconomic, and environmental factors. Although RRs for most of these socioecologic factors were not large, the public health implications may be significant when these findings are applied to the whole population.

In this study, SOI had a consistent and independent impact on the RRV disease. The SOI is defined as the normalized difference in atmospheric pressure between Darwin (Australia) and Tahiti (French Polynesia). The SOI accounts for up to 40% of variation in temperature and rainfall in the Pacific. The lagged effect of SOI on the incidence of RRV infection is very important. Such delays appear to be consistent with the development of mosquitoes, the external period of incubation of RRV within mosquitoes, incubation period of the virus in the host, and the possible delay in disease occurrence and reporting. The lagged effect of mosquito abundance on the incidence of RRV infection may have significant public health implications because it will provide an early warning for environmental health practitioners to mobilize the resources to control and prevent this disease.

There is emerging evidence that education might be a risk factor for vector-borne disease. People with lower levels of education often have poor health knowledge about disease transmission and personal protection from a mosquito biting. This may also relate to poor quality of housing because people with lower levels of education usually have lower incomes ($r_s = 0.79$ for average income and the proportion of people with low education). Poorly constructed dwellings and poor health behavior have an increased risk for mosquito-
than RRV disease. Our study shows that the proportion of labor workers was negatively associated with RRV disease. The possible explanation is that labor workers may have previously been infected by RRV, and therefore, may have high immunity. Previous studies show that prevalence rates for antibodies to RRV differ among geographic areas, and it was estimated that approximately 45% of people of aboriginal ancestry and laborers were potentially immunized against RRV infection.

Some studies show the likelihood of being infected with mosquito-borne diseases is related to proximity to vegetation. Our study found that vegetation density was associated with RRV disease in Brisbane, which is consistent with a previous report that there is a significant relationship between RRV infection rate and vegetation type (e.g., wetland, riparian, and bushland). Adult mosquitoes may rest in the shade of moderate densities of vegetation especially where these are close to oviposition sites (e.g., water bodies for *Culex annulirostris* and damp earth for *Aedes vigilax* in salt marshes and mangroves) so that gravid females can access the soil surface and ensure that eggs are protected from desiccation immediately after oviposition. In this study, we only used natural or semi-natural vegetation as an index of vegetation density. Other vegetation types (such as gardens and non-nature parkland) are also relevant as potential mosquito habitats. However, the spatial distribution of RRV mostly coincides with known major mosquito breeding sites: the northeast suburbs are characterized by wetlands, the western suburbs are associated with a creek system and dense woodlands, and the southeast suburbs contain typically densely vegetated areas, including smooth-barked apple woodlands around Tingalpa Creek.

This study has three major strengths. First, to our knowledge, this is the first epidemiologic study examining the spatial variation of RRV disease and a range of associated variables at SLA levels. Second, detailed information on socioeconomic factors by SLA was incorporated in the statistical models, and it may be used as the baseline data for further ecologic research. Finally, research outcomes from this study may have important implications for public health decision-making in the control and prevention of RRV infection.

The limitations of this study must also be acknowledged. First, although some studies indicate that the geographic distribution of RRV cases reflects fairly accurately the locations in which infections actually occur, differences may exist between these two places, particularly in holiday seasons. Nevertheless, a recent survey suggests that the locations where RRV cases were notified matched well with those where infections occurred in Brisbane. Second, marsupials (such as kangaroos and wallabies) are thought to be major vertebrate hosts. A number of domestic and wild animals, including horses, rabbits, and fruit bats, have also been implicated on the basis of serologic evidence. Humans are thought to be vertebrate hosts to a limited extent in epidemic periods. However, we do not have detailed data on hosts. Finally, we found a few hot spots of RRV transmission in Brisbane by using a smooth interpolation approach. However, because some west suburbs areas had lower population densities, some of these areas appeared to have high incidences even though the numbers of cases in these areas were low.

There has been a global resurgence of arboviral diseases. Ecologic changes and human behavior may be important in the spread of these diseases. Because the geographic expansion of RRV transmission is likely to continue, with a concomitant increase in disease incidence, it is essential that public health resources are directed into these areas. Epidemiologists, public health physicians, microbiologists, ecologists, and environmental health practitioners need to work more closely in the development of comprehensive strategies for the control and prevention of this widespread disease.

TABLE 2

<table>
<thead>
<tr>
<th>Variables (measurements units)</th>
<th>RR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human population density (per km²)</td>
<td>1.02</td>
<td>0.99–1.05</td>
</tr>
<tr>
<td>Indigenous proportion (%)</td>
<td>1.00</td>
<td>0.97–1.02</td>
</tr>
<tr>
<td>Overseas visitors proportion (%)</td>
<td>1.01</td>
<td>1.00–1.01</td>
</tr>
<tr>
<td>Lower education proportion (%)</td>
<td>1.02</td>
<td>1.01–1.03</td>
</tr>
<tr>
<td>Labor proportion (%)</td>
<td>0.97</td>
<td>0.95–1.00</td>
</tr>
<tr>
<td>SOI at lag 3 months</td>
<td>1.12</td>
<td>1.06–1.17</td>
</tr>
<tr>
<td>Vegetation density (%)</td>
<td>1.02</td>
<td>1.00–1.04</td>
</tr>
</tbody>
</table>

* RR = relative risk; CI = confidence interval; SOI = Southern Oscillation Index.
† Adjusted for all independent variables and seasonality.

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