Analysis of Barmah Forest Virus Disease Activity in Queensland, Australia, 1993–2003: Identification of a Large, Isolated Outbreak of Disease

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ABSTRACT Barmah Forest virus (BFV) disease is the second most common mosquito-borne disease in Australia. Although the majority of notifications are received from Queensland, little is known about the distribution of the disease within the state, or the important mosquito vectors and nonhuman vertebrate hosts. We conducted a retrospective statistical analysis of the notifications received from Queensland residents from 1993 to 2003 to establish long-term local incidence rates and to identify disease outbreaks. In total, 4,544 notifications were received over the 10-yr period. Disease reporting peaked in autumn, although the peak transmission season encompassed both summer and autumn. Long-term standardized incidence rates for summer/autumn and winter/spring varied across the state, showing positive spatial autocorrelation in both 6-mo periods. Although 15 instances of increased disease activity were identified, only one major disease outbreak affecting eight contiguous local government areas was detected in summer/autumn 2002/2003. This outbreak contained 297 cases, 115 more than would be expected over this period. The factors important to this outbreak are unknown and require further investigation. Although the incidence rates for BFV disease are lower than Ross River virus disease, the most reported mosquito-borne disease in Australia, several factors indicate that this virus should be considered an important public health risk in Queensland. These include consistent endemic transmission, apparent underreporting of the disease, and the potential for outbreaks in major population centers.

KEY WORDS Barmah Forest, Ross River, virus, epidemic

IN AUSTRALIA, BARMAH FOREST VIRUS (BFV) disease is the second most common mosquito-borne disease, after Ross River virus (RRV) disease. The virus is a member of the *Alphavirus* genus and was first isolated from mosquitoes in Queensland and Victoria in the 1970s (Doherty et al. 1979, Marshall et al. 1982). It was first associated with human disease in 1988 (Boughton et al. 1988), and as such, it is the most recently recognized of the Australian mosquito-borne human pathogens. The first recognized outbreak of BFV disease occurred in Nhulunbuy in the Northern Territory in 1992 (Merianos et al. 1992), and subsequent outbreaks have been reported from several other areas, including southwestern Western Australia in 1993–1994 (Lindsay et al. 1995), the New South Wales (NSW) south coast in 1995 (Doggett et al. 1999), and Victoria in 2002 (Passmore et al. 2002). The NSW outbreak involved 135 confirmed cases and was attributed to unusually high numbers of *Ochlerotatus vigilax* (Skuse) as a result of above-average rainfall and a series of high tides late in 1994. This outbreak was limited to coastal areas and is consistent with the long-term distribution of BFV disease in NSW (Muscatello and McAnulty 2000, Doggett and Russell 2005).

In Queensland, routine serological screening for BFV infection commenced in 1991 (Hills and Sheridan 1997); however, there was serological evidence of extensive BFV activity throughout Queensland before this time. Serological screening of serum collected from residents in 1989 indicated that ≈0.23% of the population was infected per year (Phillips et al. 1990). This rate of exposure was ≈40% of that predicted for RRV. A review of BFV disease notifications between 1992 and 1995 indicated that clinical disease associated with BFV infection was widespread throughout Queensland, with the highest crude notification rates (44 cases per 100,000) in central and southwestern areas of the state (Hills and Sheridan 1997). However, the BFV notification rates were underestimated because health practitioners ordering BFV serology was relatively low, with only 36–48% of epidemic polyarthritis cases tested for antibodies to BFV. There was also geographic variation in testing patterns for BFV throughout Queensland with testing rates being gen-

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erally lower in northern Queensland compared with southern testing centers (Kelly-Hope et al. 2002).

Although direct comparisons of BFV disease notification rates are not justified for the above-mentioned reasons, we suggest that there are clear differences in epidemiology of BFV throughout Australia. Since the commencement of national reporting in 1995 (except for the Northern Territory, which commenced reporting in 1997), there has been an average of 830 cases each year (Australian Government Department of Health and Aging 2004), of which 43–69% have been reported from Queensland. Although BFV is considered to be endemic in Queensland, there has been considerable variation in the numbers of cases reported each year (between 310 and 870 cases each year; Australian Government Department of Health and Aging 2004).

With the aim of providing a clearer understanding of the public health impact and the variation in the incidence of BFV disease, we characterized the spatial and temporal patterns of BFV disease notifications throughout Queensland between 1993 and 2003. To elucidate potential differences in disease patterns, notifications were mapped to local government areas (LGAs), and a Poisson distribution based model was used to examine the temporal variation in disease outbreaks within each area. Using this methodology, we were able to define unusually high levels of BFV disease incidence, relative to the long-term 10-yr pattern of disease. Because the majority of clinical cases are reported from Queensland, and there is a lack of knowledge on the ecology of BFV in Australia, this investigation of the pattern of human disease may help to define regional differences in the epidemiology of BFV.

Materials and Methods

Our study involved spatial-temporal analysis of historical notification data for BFV disease collected by Queensland Health. Data were analyzed at the LGA level, because it is at this level that mosquito control activities occur. Data for 10 yr (1993–2003) were examined by 3-mo periods (summer [December–February], autumn [March–May], winter [June–August], spring [September–November]) and by 6-mo periods (summer/autumn and winter/spring). Cases were classified by the date of onset of disease as provided by Queensland Health. Long-term average incidence rates (IRs) for each of 125 LGAs over the study period were determined. These long-term average incidence rates were used to identify periods of increased activity. Outbreak patterns were examined spatially and temporally.

Study Population. The study population consisted of all persons notified to the Queensland Health Notifiable Conditions System with a BFV infection confirmed by laboratory testing and an onset date between 1 June 1993 and 31 May 2003. A BFV disease notification was reported if serological testing by enzyme-linked immunosorbent assay indicated a fourfold change in BFV antibody titer between paired acute and convalescent sera or if IgM and IgG antibody levels against BFV were consistent with acute infection. Estimated resident populations for each LGA, in each year under study, were obtained from the Australian Bureau of Statistics and ranged from 210 to 917,216 persons. Population census data were used as denominators when calculating incidence rates.

Data Analysis. Statewide incidence rates were calculated for each year and season and by age and gender. Data were first cleaned to remove any entries with missing age and gender fields. The age groups used were 0–29, 30–59, and ≥60 yr. A procedure similar to that developed for RRV (Gatton et al. 2004) was used to determine long-term average incidence rates in each LGA for summer/autumn and winter/spring. This calculation involved the use of either the mean, median, or trimmed mean as an indicator of long-term incidence, depending on the population size and distribution of data.

A population size of 6,000 residents was used as the threshold; this was determined as the minimum population size required before LGAs with incidence rates at the state average (for winter/spring) would be expected to have a median of zero notifications during the study period. For LGAs with <6,000 residents, the crude incidence rate for the 10 yr of data was used. For LGAs with larger populations, the crude incidence rate was used if 1) the maximum number of notifications in the LGA for the period of interest was less than five, or 2) the crude incidence rate and trimmed incidence rate differed by <20% (i.e., crudeIR − trimmedIR/trimmedIR < 0.2). The trimmed incidence rate was calculated by omitting the years having the smallest and largest number of notifications from the calculation of the incidence rate. If neither of these conditions were met, then the median incidence rate was used. If the median was zero, the trimmed incidence rate was substituted (Gatton et al. 2004).

Direct standardization was used to calculate age/sex standardized rates for each LGA in each 6-mo period, by using the 10-yr state population (1993–2002) as the reference population. The standardized long-term incidence rates for each period (summer/autumn and winter/spring) were mapped separately using MapInfo Professional, version 7 (MapInfo Corporation, Troy, NY). The spatial autocorrelation between LGAs for the long-term IR was assessed for each period using the Moran's *I* statistic (Moran 1950). A Pearson correlation, calculated in SPSS, version 11.5 (SPSS, Inc., Chicago IL) was used to compare the summer/autumn and winter/spring incidence rates for each LGA.

The method described by Gatton et al. (2004) was used to classify the incidence of BFV in each LGA, in each period of each year, as an outbreak or not. This involved a comparison of the observed number of cases to the expected number of cases (based on the long-term age/sex rates), by using a Poisson distribution model. Six-month periods within each year were defined as an outbreak if there was less than a 1%

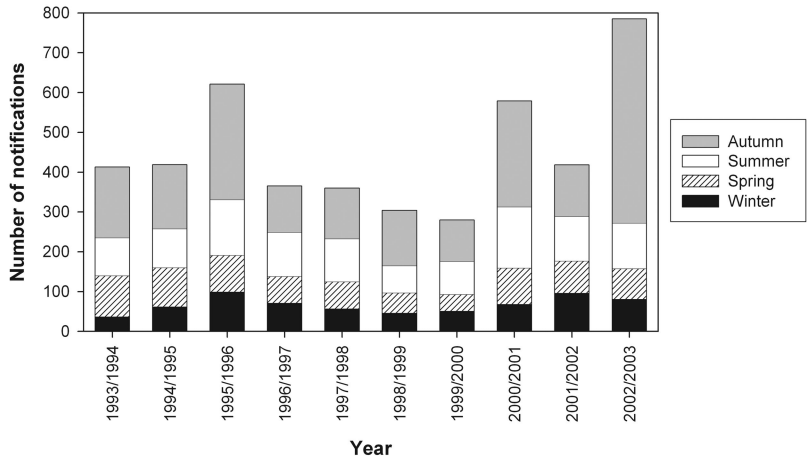

Fig. 1. Distribution of BFV notifications by year and season, in Queensland, between 1 June 1993 and 31 May 2003.

chance of obtaining the observed number of notifications based on the long-term incidence rate for the LGA. Those LGAs with a <5% chance of obtaining the observed number of notifications also were identified. If the expected number of notifications was less than one, LGAs were only declared to have had an outbreak if they had at least five notifications within the 6-mo period. This caveat ensured that LGAs with only two or three notifications were not declared as an outbreak because of mathematical criteria. Outbreaks were mapped and examined using joint count statistics with randomized sampling, as described by Lee and Wong (2000), to assess the spatial autocorrelation between LGAs having outbreaks in each 6-mo period. Outbreaks also were examined visually over time, to determine their frequency.

Results

The data set extracted for BFV notifications between 1 June 1993 and 31 May 2003 contained 4,565 cases. Fifteen of these cases were missing gender and six were missing age. These cases were discarded from the data set, leaving 4,544 notifications for the study.

Summary Notification Rates. The crude annual incidence rate for Queensland during the study period was 13.3 cases per 100,000 population. Seventy-one percent of notifications were received for people aged 30–59 yr, at a rate of 23.2 cases per 100,000 population. The incidence rate for males was slightly higher than that for females, at 13.8 and 12.7 cases per 100,000 population, respectively. Annual incidence rates for individual LGAs ranged from 0 to 87.0 cases per 100,000 population, with a median of 16.3 cases per 100,000 population. Five LGAs (Aurukun, Bulloo, Etheridge, Mornington, and Perry) reported no cases of BFV in the 10-yr period.

There were large variations in the number of notifications reported in each year in Queensland (Fig. 1), with annual crude rates ranging from 8.0 cases per 100,000 population in 1999/2000 to 21.2 cases per 100,000 population in 2002/2003. Crude incidence rates for each season were 12.6, 23.6, 7.8, and 9.0 cases per 100,000 population for summer, autumn, winter, and spring, respectively. Most variation in notifications for a year was due to differences in the number of autumn notifications (Fig. 1). Sixty-eight percent of notifications occurred in the summer/autumn period, with 24% of notifications recorded in summer and 45% recorded in autumn. In any one year, between 31 and 65% of notifications occurred in autumn.

Long-Term Incidence Rates. Initially, long-term average incidence rates and expected numbers of cases were calculated for each season, and these were then compared with the observed number of cases during each seasonal period. However, examination of the temporal distribution of cases indicated that the peak transmission season of BFV disease was split between summer and autumn. This finding, combined with the relatively small numbers of notifications in each LGA during the 3-mo periods caused us to pool the data into two 6-mo periods (summer/autumn and winter/spring) and to calculate corresponding long-term incidence rates. Based on the selection criteria described previously, the crude incidence rate was used to represent the long-term incidence rate for 68 LGAs in the summer/autumn period and 83 LGAs in the winter/spring period, whereas the median incidence rate was used for 49 LGAs in summer/autumn and 39 LGAs in the winter/spring period. The remaining LGAs had long-term incidence rates calculated using the trimmed incidence rate.

There was correlation in long-term incidence rates between the summer/autumn and the winter/spring periods within LGAs across the state ($r = 0.56$, $P < 0.01$). During both periods, low incidence rates were clustered in the southeastern corner of the state. High incidence rates were clustered in central Queensland, both inland and toward the coast (Fig. 2). The long-term incidence rates for the summer/autumn period showed positive spatial autocorrelation (Moran's $I =$

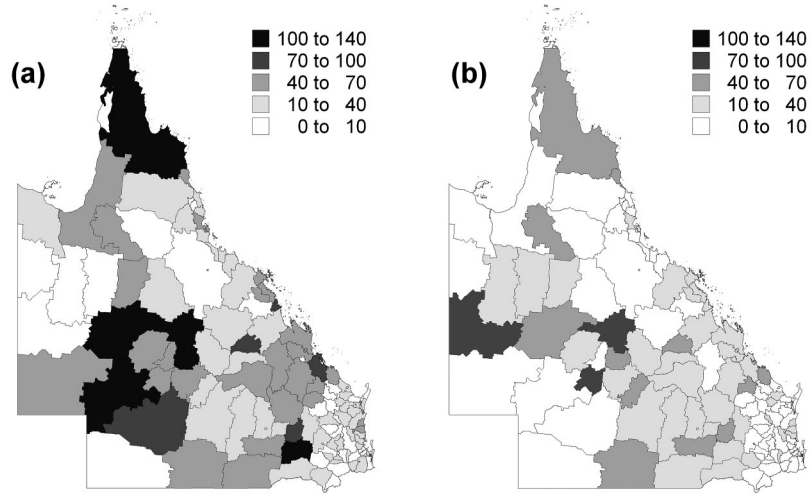

Fig. 2. Long-term (1993–2003) age-sex standardized incidence rates (per 100,000 population) for BFV disease notification in each Queensland LGA during (a) summer/autumn and (b) winter/spring.

0.4559, $P < 0.0001$), as did the incidence rates for the winter/spring period (Moran's $I = 0.2847$, $P < 0.0001$). This autocorrelation indicated that there was clustering of LGAs with similar incidence rates in each period.

Outbreaks. Baseline indicators were calculated for each LGA, in each 6-mo period, for each year (125 LGA × two 6-m periods × 10 yr = 2,500 indicators). When observed cases were compared with the indicators, only 15 cases were classified as outbreaks at the 1% cutoff. When a less stringent 5% cutoff was used, 24 outbreaks were identified. Only results for outbreaks meeting the 1% cutoff were reported hereafter. Nine of the 15 LGA outbreaks occurred in summer/autumn period 2002/2003. All but one of these outbreaks was located in the southeastern corner of the state (Table 1; Fig. 3). The outbreaks for this period showed significant positive autocorrelation ($z = -3.96$, $P < 0.0001$), indicating that LGAs experiencing outbreaks were clustered.

The remaining six outbreaks occurred sporadically over the other 9 yr. There did not seem to be any geographical or temporal relationship between the LGAs experiencing these outbreaks (Fig. 4). Three of the outbreaks occurred in the winter/spring period, and none of the outbreaks occurred concurrently (Table 1).

Of those LGAs that experienced outbreaks, only two (Toowoomba and Cooloola) experienced multiple outbreaks. Both Toowoomba outbreaks occurred in consecutive summer/autumn periods. The first Cooloola outbreak occurred during winter/spring, with the other outbreak occurring in summer/autumn 2.5 yr later.

2002/2003 Outbreak in Southeastern Queensland. The dynamics of the outbreak detected in summer/autumn 2002/2003 differed among LGAs in southeastern Queensland. The LGAs of Beaudesert, Cooloola, and Redcliffe were the first to reach their expected number of notifications (early April), whereas the remaining LGAs passed their expected number by the end of May (Fig. 5). Four LGAs in the region were not classified as experiencing an outbreak during this period. Only one additional LGA would have been clas-

Table 1. Summary of the cluster of Barmah Forest virus disease outbreaks occurring in 2002/2003 and the six outbreaks occurring before 2002/2003, in Queensland

LGA	Yr	Six-mo period	Expected no. of cases	Observed no. of cases
Banana	1993/1994	Winter/spring	4	14
Maryborough	1996/1997	Winter/spring	2	8
Toowoomba	1997/1998	Summer/autumn	4	10
Toowoomba	1998/1999	Summer/autumn	4	11
Mount Isa	1999/2000	Summer/autumn	2	6
Cooloola	2000/2001	Winter/spring	2	7
Beaudesert	2002/2003	Summer/autumn	6	14
Brisbane	2002/2003	Summer/autumn	57	79
Cooloola	2002/2003	Summer/autumn	7	17
Gladstone	2002/2003	Summer/autumn	6	16
Logan	2002/2003	Summer/autumn	7	18
Maroochy	2002/2003	Summer/autumn	56	80
Noosa	2002/2003	Summer/autumn	23	51
Pine Rivers	2002/2003	Summer/autumn	17	28
Redcliffe	2002/2003	Summer/autumn	3	10

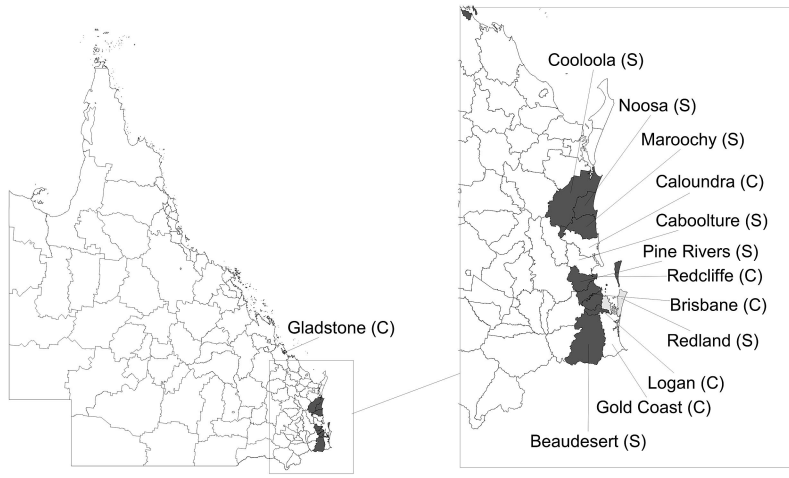

Fig. 3. Geographical distribution of BFV disease outbreaks (dark gray shading) occurring during summer/autumn 2002/2003 in Queensland. An LGA located in the southeast, which also experienced an outbreak by using the 5% cutoff criteria, is highlighted (light gray shading).

sified as experiencing an outbreak by using the 5% cutoff criteria (Redland), whereas the other three showed only slightly elevated notification levels compared with that expected. During this summer/autumn period, 297 cases were notified from the cluster of eight LGAs that experienced an outbreak.

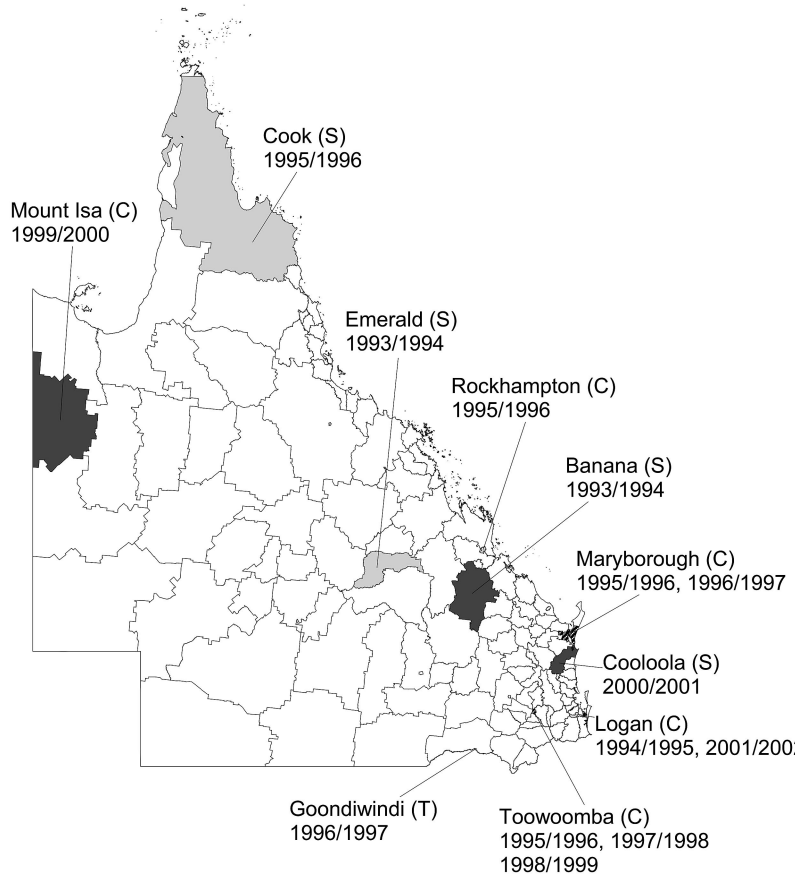

Fig. 4. Geographical distribution of BFV disease outbreaks that occurred before 2002/2003.

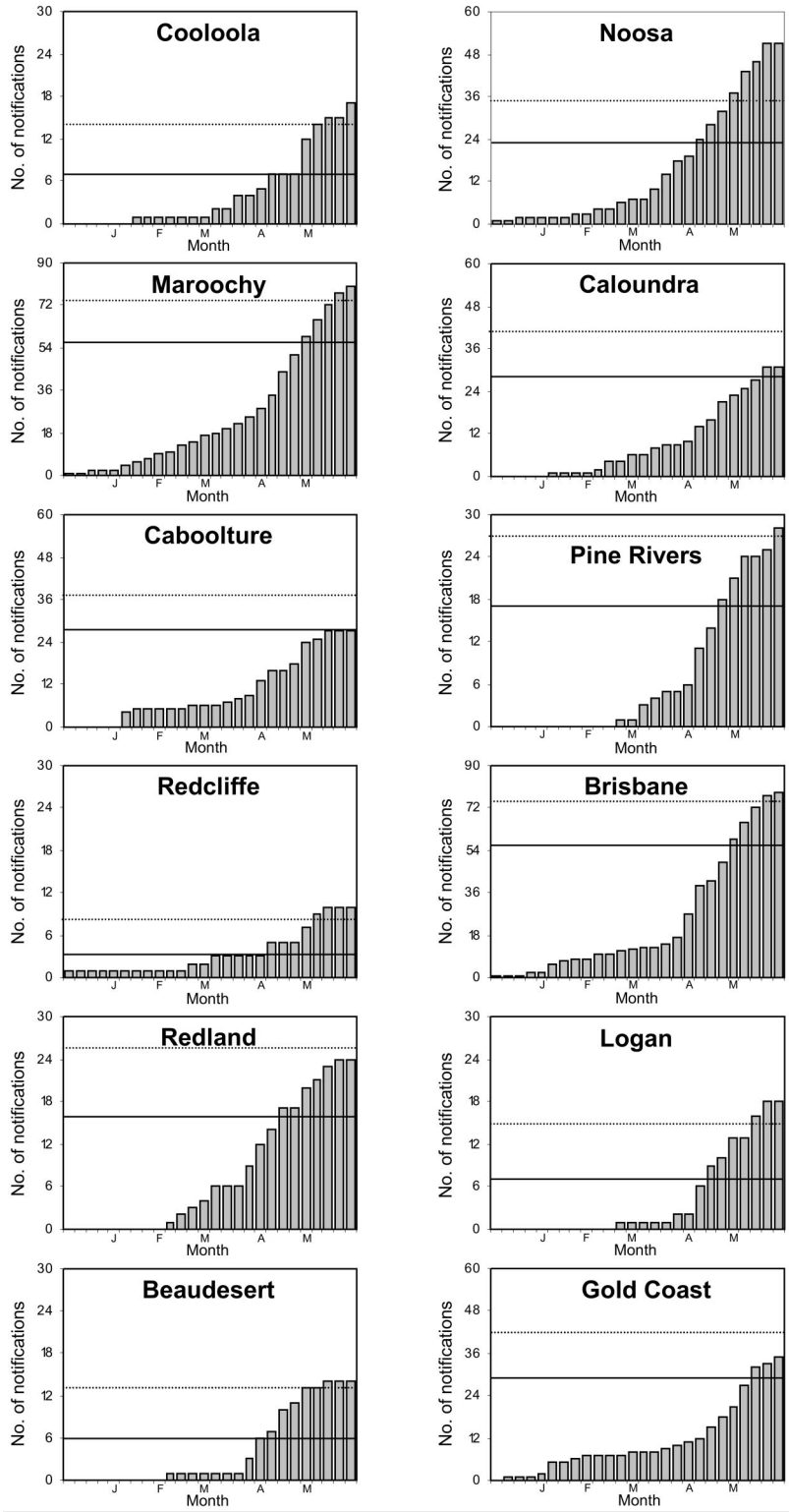

Fig. 5. Cumulative BFV disease notifications each week during the 2002/2003 outbreak, from 12 LGAs located in southeastern Queensland. The expected number of notifications for the 6-mo period for each LGA is represented by a solid line (—), and the 1% cutoff for an outbreak is represented by a dotted line (····). The graphs for the LGAs in southeastern Queensland are presented in order from north to south as indicated in Fig. 3.

Discussion

In contrast to the different temporal and spatial patterns of RRV disease notifications throughout Queensland (Gatton et al. 2004), we found that BFV, in general, was responsible for sporadic, low numbers of disease notifications throughout the state. The exception to this was the relatively large outbreak of BFV disease in the southeastern region of the state between March and May 2003. Based on our conservative cutoff for a type I error of 1%, we classified nine LGAs as having higher than expected numbers of BFV disease cases during summer/autumn 2002/2003. The geographic extent of the affected communities ranged from Cooloola in the north to Beaudesert in the south and encompassed an area of ≈250 by 75 km. Based on published descriptions of outbreaks of BFV disease, the 2002/2003 outbreak of 297 serologically confirmed BFV disease cases represented the largest outbreak of BFV disease in Queensland since routine laboratory testing commenced in 1991. Although BFV disease incidence is generally regarded as low compared with RRV disease, the BFV disease attack rates in some LGAs in southeastern Queensland during 2002/2003 were up to 137.1 cases per 100,000 population. These rates were among the highest reported in any Australian outbreaks.

Doggett and Russell (2005) reported that BFV disease also has emerged as an important public health problem in the mid-north coast area in NSW. The Northern Rivers and Mid North Coast area health districts, which are comprised mainly of coastal populations spanning a distance of 500 km, reported 242–365 BFV disease notifications each year between 2000/2001 and 2003/2004. The annual incidence rates during this 4-yr period (21.8–84.8 cases per 100,000 population) were generally higher than those recorded in the previous 6 yr (14.4–37.4 cases per 100,000 population) but comparable with rates in LGAs in southeastern Queensland during the 2002/2003 epidemic. The relatively high numbers of BFV disease cases from these coastal areas in NSW, and also in coastal areas in southeastern Queensland during 2002/2003, are of concern, because the human population growth in this region ranks among the highest of any area in Australia (Australian Bureau of Statistics 2004). At a minimum, health authorities should make residents aware of possible risks associated with exposure to mosquitoes and also advise medical practitioners to request both RRV and BFV serological testing for suspected polyarthritis cases.

Although coastal areas in southeastern Queensland and northern NSW both experienced increased BFV activity during 2002/2003, there are clear differences in the general epidemiology of BFV between the two states. In Queensland, BFV disease notifications were distributed throughout the state, and there was no clear clustering of LGAs with high incidence rates in either inland or coastal areas (Fig. 2). In contrast, in NSW there was an obvious difference in the epidemiology of BFV between coastal and inland areas. Coastal areas accounted for 91% of all BFV disease cases between 1994 and 2004, and the combined incidence rate in the coastal population was 460% higher than that in the inland population (Doggett and Russell 2005). The reasons for these differences are unknown, mainly because there is little information on the important vectors and vertebrate hosts of BFV.

Oc. vigilax, is a confirmed vector of BFV; based on repeated isolation of virus from wild caught adult mosquitoes from coastal areas in Western Australia, NSW, and Queensland (Lindsay et al. 1995, Doggett et al. 1999, Ryan et al. 2000), high vector competence (Ryan and Kay 1999, Boyd and Kay 1999), and its tendency to feed on a broad range of mammalian hosts, including humans (Lee et al. 1984). Although *Oc. vigilax* was probably involved in BFV transmission during the 2002/2003 outbreak in coastal areas in southeastern Queensland, the mosquito vectors involved in transmission in inland areas are unknown.

Barmah Forest virus was originally isolated from the freshwater mosquito *Culex annulirostris* (Skuse), collected from the Murray Valley region of southeastern Australia (Marshall et al. 1982). Although a NSW mosquito population was shown to be moderately susceptible to experimental infection (Wells et al. 1992), laboratory vector competence experiments indicated that field-collected *Cx. annulirostris* from southeastern Queensland were almost totally refractory to BFV infection (Ryan and Kay 2000). Therefore, the role of *Cx. annulisrostris* in BFV transmission in Queensland is unclear. Interestingly, *Ochlerotatus procax* (Skuse), a floodwater species, and *Ochlerotatus notoscriptus* (Skuse), which uses a range of natural and artificial water containers and is widely distributed throughout Australia, have been shown to be competent laboratory vectors of BFV (Doggett and Russell 1997, Watson and Kay 1999). Either one or both of these species could be involved in BFV transmission throughout Queensland; however, field investigations are required to define locally important BFV vectors. Until this occurs, it will be difficult to focus mosquito control activities to prevent human infection.

To compound the problem, little is known about important nonhuman vertebrate hosts. Serological surveys have indicated that marsupials are commonly infected (Vale et al. 1991, Boyd 2004, Johansen et al. 2005), and neutralizing antibodies to BFV also have been found in livestock (horses, cows, and goats) (Vale et al. 1991, Boyd 2004) and in companion animals, including dogs and cats (Boyd 2004). Infection with BFV also has been found in birds (Karabatsos 1985). Although experimental infection of brushtail possums, cats, and dogs with BFV was successful, the resulting viremias were considered too low to infect mosquitoes (Boyd et al. 2001, Boyd and Kay 2002). More data are required before mammals (either placental or marsupial) and/or birds can be ascribed as hosts for the virus (Russell 1998). This lack of information prohibits a full understanding of the ecology of BFV and subsequent risk factors for human disease.

In terms of detecting human disease, Queensland's existing laboratory-based serological testing procedure for mosquito-borne viral diseases, combined with

an information system capable of providing timely information to health departments and local governments, may be of use in terms of rapid identification of BFV and RRV epidemics. Public health and vector control workers would need to have access to a system that provides alerts when substantial numbers of excess cases are expected, and such alerts should be sensitive, specific, and timely (Teklehaimanot et al. 2004). The underlying premise for such a system is that early detection of an epidemic will result in the initiation of specific interventions, and these interventions will result in a reduction in the number of new cases. The World Health Organization has advocated the use of alerts when monthly numbers of malaria cases exceed the 75th percentile determined from 5 yr of retrospective case data (Nájera et al. 1998). Historical data sets for BFV and RRV diseases should be examined to determine whether alerts of higher than expected numbers of cases may be useful in terms early detection. In terms of practical interventions to prevent human infection with BFV, any warning of a potential outbreak will most likely result in broad public health warnings and instructions on the use of personal protective measures, given that currently there is little information on which vectors and vertebrate hosts are important in terms of transmission. The capacity of local authorities in Queensland to instigate such interventions has not been previously considered, but it is instrumental in seeking to curtail future disease outbreaks. This situation cannot be improved until more applied research on the biology and ecology of BFV is undertaken.

Our study has documented the incidence and distribution of BFV disease in Queensland, Australia. Although the disease is endemic, we identified one major outbreak in the southeastern part of the state that affected several contiguous LGAs. The factors important in triggering this outbreak are unknown and require further investigation. Although the incidence rates for BFV disease are lower than RRV disease, the apparent underreporting of the disease and the potential for outbreaks in major population centers suggests that BFV should be considered an important mosquito-borne disease in Queensland.

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