MURRAY VALLEY ENCEPHALITIS AND KUNJIN VIRUSES – THE VICTORIAN EXPERIENCE

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Introduction
Murray Valley encephalitis (MVE) and Kunjin (KUN) viruses are mosquito transmitted flaviviruses that infect humans and animals. MVE is endemic in much of Northern Australia, while occasional MVE outbreaks occur in south-eastern Australia. The Victorian Arbovirus Task Force (VATF), established by the Victorian Government in 1987, uses three monitoring methods to try to predict the reappearance of MVE virus in south-eastern Australia: serological testing of sentinel chicken flocks together with monitoring of vector mosquito species, rainfall (Forbes hypothesis) and barometric pressure (Nicholls hypothesis). Although one or more methods have suggested an increased risk of MVE or Kunjin outbreaks, notably in 2011, these did not eventuate. However the absence of clinical cases does not exclude silent circulation as only 1/800 infections generally result in symptomatic disease and the risk to human populations in Victoria remains unclear. To determine whether there had been transmission of MVE and KUN viruses to humans over the last 11 years, three serosurveys were carried out: the first was in 2002, VATF asked VIDRL to retrospectively test specimens collected as part of a Victorian Q Fever serosurvey, the second was in 2008 after 3 of 10 sentinel chicken flocks showed reactivity to MVE for the first time in 30 years and the third was in 2011 following seroconversion of chicken flocks in the contact of environmental conditions favourable to MVE spread.

Objectives:
To determine if there had been undetected subclinical infection of MVE and Kunjin viruses since the last major outbreak in 1974.

Methods:
Sera were tested using an in-house epitope blocking EIA for total antibody and an in-house immunofluorescence IgM assay to determine the seroprevalence for MVE and KUN viruses in the 3 serosurveys.
Sera were grouped according to postcode of residence. Seropositives were analysed by age and sex

Results:
In 2002, 529 specimens from 6 different regions around Victoria were tested. Twenty three of 529 (4.3%, 95%CI 2.8-6.4) were positive for MVE and 24/529 (4.5%, 95%CI 2.9-6.7) were positive for KUN total antibody (Ab). For MVE, prevalence in the Murray River region compared to other regions was 6.2% and 1.8% respectively (p=0.01). Similarly for KUN 6.5% and 1.8% respectively (p=0.01). Sex and age data were also studied. For MVE, males were more likely to be Ab positive than females (4.4% vs 0.8%, p=0.063), for KUN (4.4% vs 1.6%, p=0.185). Age was strongly associated with antibody positivity for MVE and to a lesser extent for KUN. As the last outbreak of MVE in Victoria was in 1974, positive results were analysed according to individuals <40 years of age vs > 40 years, MVE seropositivity was significantly different in the two age groups (p=0.049), but KUN seropositivity was not (p=0.38).
In 2008, 121 samples from the Kerang-Mildura district were tested. Only 1/121 (0.8%, 95%CI 0.02-4.6) was MVE Ab positive. Kunjin Ab testing was not performed.
In 2011, 1,115 specimens were tested. Twenty four of 1115 (2.1%, 95%CI 1.3 – 3.0) were positive for MVE total Ab, but all were negative for MVE IgM. Three positive MVE patients were less than 37 years of age, indicating infection since 1974. Thirty four of 116 (3.0%, 95%CI 2.0 – 4.0) were positive for KUN total Ab, 3 were positive for KUN IgM.

Conclusion:
Seroprevalence studies suggest minimal exposure to either MVE or Kunjin viruses in Victoria in recent decades, despite models predicting otherwise. People living near the Murray River and males were more likely to be positive. Age was associated more strongly with MVE seropositivity than with KUN.
The absence of predicted disease circulation raises important questions about how existing models are used to predict MVE virus outbreaks. In an outbreak situation a large proportion of the Victorian population remain at risk of infection.