



Mosquito-borne diseases in New Zealand: has there ever been an indigenously acquired infection?

Imported cases of a number of mosquito-borne diseases are regularly reported in New Zealand. The list of imported diseases includes malaria, yellow fever, dengue fever, Ross River virus, Barmah Forest virus, and Japanese encephalitis. There is, however, controversy regarding the previous occurrence or not of an indigenously acquired mosquito-borne infection in man in New Zealand. Claims seem to be made more frequently regarding human infection caused by Whataroa virus (*Togaviridae: Alphavirus*).¹

Whataroa virus has been isolated from the endemic mosquitoes *Culiseta tonnoiri* (Edwards) and *Culex pervigilans* Bergroth in South Westland, and detailed descriptions of the ecology of this virus were published in the 1960s and 1970s,²⁻⁵ in which the evidence suggested a bird-mosquito cycle.³ Native and exotic bird species have been infected by Whataroa virus, but there is no evidence of illness or death amongst the birds studied indicating that the infection is clinically unapparent.³

Although Maguire et al suggested at the time that there was indication that Whataroa virus infected man,² Hogg et al seems to be the only publication ever to provide evidence of human infection with an arbovirus in New Zealand.⁶ However, the evidence provided cannot be considered conclusive. Haemagglutination inhibition (HI) antibodies are only significant if a considerable rise (8–16 fold or greater) in titre from the acute to the convalescent phase of an illness a few weeks later can be shown.

This was not the case in Westland, and the titers identified by Hogg et al were relatively low.⁶ In addition, the antibodies reacted with Group B antigens, but 10 years of subsequent work only led to the isolation of Whataroa virus in the area, which is Group A.

The low levels of antibody detected by HI methods in Westland are not sufficient to make the claim that any specific virus has been confirmed as being present in New Zealand, especially since HI tests are not specific for any one agent, but are broadly group-specific. The conclusive evidence would be the isolation of the agent, accompanied, if possible, by positive antibody results. Therefore, one cannot say with confidence that human infection with any arbovirus has ever occurred in Westland.

The only reliable evidence of a mosquito-borne infection acquired in New Zealand seems to be the case of a man infected with malaria in 1927.⁷ The diagnosis was established beyond doubt, and since the patient had never been to a malarious country, had not left New Zealand for 13 years, and had not been ill before, the only possible conclusion was that infection occurred in the country, probably in Auckland.⁷

There are no records of a population of anopheline mosquitoes in New Zealand, and the known established species are very unlikely to be competent vectors of malaria (even though no studies have been done to test this assumption). The described infection appears to have been an incident of 'seaport malaria'. Anophelines could have arrived in this country from Australia or Panama by ocean transport,⁷ and since

numerous people regularly returned to New Zealand after contracting malaria abroad, transmission by a hitch-hiking mosquito was possible.

Nonetheless, we are yet to see a confirmed indigenously acquired infection in humans in New Zealand, in which a pathogen was transmitted by a female mosquito from a local vector population. Over many years, the Virus Research Unit at the University of Otago (Dunedin) carried out serological studies on some thousands of New Zealand human sera searching for evidence of local arbovirus infection, but the only positive results were obtained from people who had travelled overseas.

New Zealand can, therefore, still be considered a 'virgin soil' when it comes to mosquito-borne diseases.

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