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**Journal of the Royal Society of New Zealand**
Publication details, including instructions for authors and subscription information:
http://www.informaworld.com/smpp/title~content=t918982755

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Online publication date: 08 April 2010

**To cite this Article** Tompkins, DM , Paterson, R. , Massey, B. andGleeson, DM(2010) 'Whataroa virus four decades on: emerging, persisting, or fading out?', Journal of the Royal Society of New Zealand, 40: 1, 1 — 9
**To link to this Article: DOI:** 10.1080/03036751003641701
**URL:** http://dx.doi.org/10.1080/03036751003641701

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*Journal of the Royal Society of New Zealand*
Vol. 40, No. 1, March 2010, 1–9

Routledge
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# Whataroa virus four decades on: emerging, persisting, or fading out?

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(*Received 5 September 2008; final version received 18 January 2010*)

Whataroa virus, first detected in 1962 in bird populations around Whataroa township, is the only mosquito-borne virus circulating in New Zealand that has been isolated. Even though at least one other mosquito-borne pathogen (avian malaria) has greatly increased in prevalence in New Zealand in recent decades, no surveillance for Whataroa virus in its vertebrate hosts has been carried out for four decades. This is of concern since Whataroa virus may infect humans, possibly causing influenza-like symptoms. Here we reassess the status of Whataroa virus in the same bird species in the same area where it was detected previously. Molecular diagnostics identified Whataroa virus in three out of 95 non-native birds screened: two out of eight song thrushes *Turdus philomelos*, and one out of nine blackbirds *Turdus merula*. The detection of virus in birds, in contrast with recent screening of mosquitoes, highlights how wildlife surveillance for pathogens can be far more effective than vector surveillance. Results of this survey indicate the virus has not increased substantially in prevalence since last monitored, possibly because of little change in the local mosquito vector community. Finally, virus detection in the two *Turdus* species alone supports earlier claims that these non-native hosts act as reservoirs that maintain the virus, sourcing spillover infections in other native and non-native species. A similar role for the blackbird in avian malaria epidemiology in New Zealand has also recently been hypothesized.

**Keywords:** alphavirus; arbovirus; avian malaria; bird; emerging infectious disease; influenza; mosquito; Sindbis; Whataroa virus; vector

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## Introduction

Emerging Infectious Diseases (EIDs; disease-causing agents that rapidly increase in host range, geographic range or prevalence) are now a well-recognized threat to biodiversity, agriculture, and public health globally (Binder et al. 1999; Daszak et al. 2000; Morens et al. 2004), with the rate of disease emergence having risen significantly since the middle of the twentieth century (Jones et al. 2008). Analyses of risk factors indicate that emergence is being driven by multiple factors including socio-economic circumstances (Weiss & McMichael 2004; Jones et al. 2008), climate and land-use changes (Harvell et al. 2002; Patz et al. 2004, 2005), and 'pathogen pollution' (the anthropogenic global movement of pathogens; Williams et al. 2002; Cunningham et al. 2003). Given the significant burden that EIDs place on global economies and public health (Meslin et al. 2000; Morens et al. 2004), the monitoring of, mitigation of and early response to potential infectious disease threats are of the highest priority (Smolinski et al. 2003; Patz et al. 2004; Kuiken et al. 2005; King et al. 2006; Wolfe et al. 2007; Woolhouse 2008). These global concerns are reflected in New Zealand, with an increase in active

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ISSN 0303-6758 print/ISSN 1175-8899 online
© 2010 The Royal Society of New Zealand
DOI: 10.1080/03036751003641701
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surveillance for potential disease threats being advocated for the benefit of native wildlife, domestic stock and public health (Crump et al. 2001; Alley 2002; Tompkins & Poulin 2006; Cork et al. 2007; Derraik & Slaney 2007; French et al. 2007).

One of the most important taxonomic groups of pathogenic aetiological agents involved in disease emergence is viruses (Morse 1993). Recent analyses indicate that viruses (together with prions) have been involved in 25–44% of disease emergence events (Cleaveland et al. 2001; Taylor et al. 2001; Woolhouse & Gowtage-Sequeria 2005; Jones et al. 2008). In addition, a disproportionately large number of the new species of human pathogens reported in the last three decades have been viruses (Woolhouse & Gaunt 2007). Emergence of disease-causing viral agents is also generally one of the top concerns of wildlife, domestic animal, and public health agendas (Bricaire & Bossi 2006; Heeney 2006; Mackereth et al. 2007).

One of the main causes of virus emergence is changes in the distribution and abundance of vectors of infection (Hui 2006; Purse et al. 2006), because a large proportion of emerging diseases are vector-borne (Edman 2005; Jones et al. 2008). In particular, arboviral diseases vectored by mosquitoes are among the most important of the emerging infectious disease problems facing the world (Gubler 2001; Mackenzie et al. 2004), with emergence generally comprising the spread (and often re-emergence) of existing viral agents (Gubler 2002). This is in contrast to host-shifts from wildlife, being the most common mechanism of emergence for both directly transmitted viral (Woolhouse & Gaunt 2007), and other major human infectious diseases (Wolfe et al. 2007). Such spread and re-emergence is due to the mosquito vectors of these disease-causing agents typically being highly sensitive to changes in environmental conditions (Liehne 1988; Rogers & Randolph 2000; Kovats et al. 2001) and land use (Patz et al. 2004; Hui 2006), and highly prone to anthropogenic dissemination to new geographic areas (Lounibos 2002; Tatem et al. 2006).

In spite of a regular influx of viremic travellers, particularly from Australia (Kelly-Hope et al. 2002; Derraik & Calisher 2004), only a single mosquito-borne virus circulating in New Zealand has been isolated (Holder et al. 1999; Crump et al. 2001). The Sindbis-like alphavirus Whataroa virus, also detected in Australia (Saleh et al. 2004), but in New Zealand known only from the area around the town of Whataroa on the west coast of the South Island, was first detected in bird populations there in 1962 (Ross et al. 1964; Maguire et al. 1967). Virus isolation, serological surveys and experimental infection trials have shown that the virus circulates enzootically in New Zealand in a transmission cycle in which both native and exotic bird species are infected (Miles et al. 1971; Miles 1973), and two endemic mosquito species (*Culex pervigilans* and *Culiseta tonnoiri*) are vectors (Ross et al. 1964; Maguire et al. 1967; Miles et al. 1973). The virus has also been linked to reports of rural summer epidemics of influenza-like disease in humans (Miles 1973), although the serological evidence for human infection, while strongly suggestive, is not conclusive (Hogg et al. 1963; Ross et al. 1964; Derraik & Maguire 2005).

In spite of arboviral diseases being recognized as an increasing threat to New Zealand for over a decade (Maguire 1994; Weinstein et al. 1995, 1997; Derraik & Slaney 2007), and calls for improved surveillance for mosquitoes with disease-vector potential (Hearnden et al. 1999; Derraik & Slaney 2007), no surveillance for Whataroa virus in its vertebrate hosts in New Zealand has been carried out for four decades. Motivated by a recent survey showing that the prevalence of another mosquito-borne pathogen, avian malaria, has increased in wild bird populations in New Zealand over roughly the same time period (Tompkins & Gleeson 2006), here we reassess the status of Whataroa virus in the same bird species in the same area where the virus was detected previously. The aim was to gain a preliminary assessment of whether

this mosquito-borne pathogen is emerging, persisting, or can no longer be detected. An indication of increased prevalence would motivate further research into its current hosts, spatio-temporal distribution and causes of emergence, and studies to better characterize disease impacts (if any) on both humans and native birds. In contrast, failure to detect the virus would support results of a recent survey using molecular diagnostics that failed to detect Whataroa virus in 4221 mosquitoes collected from the Whataroa area (Snell et al. 2007); these studies together suggesting that the virus may have failed to persist (suffered 'fade-out') over time.

## Methods

### *Field sampling*

A 5-year time series of Whataroa virus sero-prevalence data in native and non-native birds is available for the area around Whataroa township in South Westland (43°16′S, 170 °22′E), from 1964 to 1969 (Miles et al. 1971; Miles 1973). Here, we collected blood samples from non-native birds in the same area during December 2006 and February 2007 (summer), with spring to early summer being identified as the main period of virus spread (Miles et al. 1971; Miles 1973).

Blood samples were collected by brachial puncture from birds either shot or caught with mist nets and released. Released birds were banded with numbered metal bands to prevent re-sampling. Up to 0.1 ml of blood was collected from each individual, and stored in 0.3 ml EDTA in individual vials at 4°C until tested for the presence of Whataroa virus in June 2007.

### *Molecular analysis*

Samples were analysed for the presence of alphavirus particles by the application of a highly sensitive reverse transcription polymerase chain reaction (RT-PCR) adapted from Pfeffer et al. (1997), in which cDNA is synthesized and amplified from the gene encoding non-structural protein 1 (nsP1) of any alphavirus present in a sample. Since the diagnostic used is genus-specific for alphaviruses, the identification of Whataroa virus required sequencing of cDNA from all positive samples, and comparison with existing nsP1 data on the NCBI GenBank nucleotide database.

We used the M78 strain of Whataroa virus (isolated from *Culex pervigilans* mosquitoes collected from Whataroa in 1962 and stored as a vero cell supernatant at −80°C; Ross et al. 1964; Maguire et al. 1967) as the positive control for the molecular diagnostic. Positive controls took two forms, with the supernatant being added to samples of blackbird blood collected from the North Island of New Zealand and stored in EDTA, both before and after the plasma separation stage (see below). The negative control for the molecular analysis was the PCR mastermix by itself at the amplification phase (see below).

#### *RNA extraction*

For each sample, the blood–EDTA mix was initially pulse-centrifuged to settle all cell clumps to the bottom of the sample tube. Next, 500 μl of sample containing no clots or clumps were mixed with 500 μl of Histoplaque-1077 (Sigma Chemical) in a new microcentrifuge tube. The mixture was centrifuged at 400*g* for 30 min at room temperature before 100 μl of supernatant containing plasma were removed and viral RNA extracted and isolated using the PureLink™ Viral RNA kit (Invitrogen), following the manufacturer's instructions.

#### *Reverse transcription*

For RNA denaturation, 4 μl of each sample potentially containing viral RNA (the cDNA template) obtained from the previous step was mixed with 5.5 μl of nuclease-free water, 4 μl of 5 × first strand buffer, 2 μl of RNAsinPlus and 0.5 μl of random Hexamer primers, with the resulting mixture incubated at 70°C for 10 min, centrifuged, and left at room temperature for

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15 min. Then, 4 μl of a transcription mix (consisting of 2 μl DTT, 0.5 μl RNAsinPlus, 1 μl 10 mM dNTPs and 0.5 μl Superscript II-RT) was mixed into the denaturation mix, followed by incubation at 42°C for a further 60 min. The resulting sample potentially containing cDNA was stored at −20°C.

*Polymerase chain reaction*

A 434 bp region ('the amplicon') of the non-structural protein encoding gene (nsP1) was targeted for PCR amplification using the degenerate primers M2W and cM3W (Pfeffer et al. 1997). PCR amplifications were carried out in a GeneAmp PCR System 9700 thermocycler (Applied Biosystems). PCR reactions were carried out in a total volume of 25 μl, which contained 10 × PCR buffer and 2 mM of $MgCl_2$, 2 μM of dNTPs, 0.2 mM of each primer, 1.0 units of Faststart Taq (Roche), and 2 μl of cDNA. PCR profile conditions consisted of an initial denaturation for 4 min at 95°C, followed by 45 denaturation cycles of 20 s at 94°C, 30 s at 50°C, and extension for 1 min at 72°C, followed by a final extension for 7 min at 72°C. Following PCR, 10 μl of resulting products were analysed using 2% agarose gel in Tris Acetate EDTA and visualized under UV light following ethidium bromide staining, with the appearance of a band corresponding to the expected 434 bp amplicon indicating a positive sample.

*Sequencing*

The 434 bp amplicon obtained from the positive samples was purified using a Purelink™ PCR Purification Kit (Invitrogen), following the manufacturer's instructions. Sequences were analysed on an Applied Biosystems 3100-Avant genetic analyser using DNA Sequencing Analysis Software Version 5.1 (Applied Biosystems). Chromatograms were aligned using the computer program Sequencher 4.6 (Gene Codes); alignments were edited manually and subsequently identified using the BLAST search algorithm on the NCBI GenBank nucleotide database.

## Results

Blood samples for Whataroa virus screening were collected from 95 non-native birds (Table 1). Molecular diagnostics were successfully carried out on all samples, with both positive controls successfully producing the target amplicon, and negative controls being non-responsive.

**Table 1** RT- PCR screening for Whataroa virus in blood samples collected during December 2006 and February 2007 from non-native birds around Whataroa township, contrasted with the average prevalence of Whataroa virus neutralising antibody in the same species during the 1964–1969 serological survey of Miles et al. (1971). Note that the measures reported by the two diagnostic techniques are not directly comparable (see Discussion).

| | RT-DNA diagnostic 2005–2006 | | | Serology 1964–1969 |
|---|---|---|---|---|
| Species | Sample size | Number positive | Prevalence (95% CI) | Prevalence (95% CI) |
| Blackbird *Turdus merula* | 9 | 1 | 0.11 (0.00–0.48) | 0.11 (0.09–0.14) |
| Song thrush *Turdus philomelos* | 8 | 2 | 0.25 (0.03–0.65) | 0.19 (0.16–0.22) |
| Chaffinch *Fringilla coelebs* | 27 | 0 | 0.00 (0.00–0.11) | 0.21 (0.17–0.26) |
| Red poll *Carduelis flammea* | 4 | 0 | 0.00 (0.00–0.53) | 0.10 (0.06–0.15) |
| House sparrow *Passer domesticus* | 20 | 0 | 0.00 (0.00–0.14) | 0.07 (0.00–0.34) |
| Hedge sparrow *Prunella modularis* | 7 | 0 | 0.00 (0.00–0.35) | 0.19 (0.14–0.24) |
| Starling *Sturnus vulgaris* | 20 | 0 | 0.00 (0.00–0.14) | 0.17 (0.05–0.37) |

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The target amplicon was also amplified from three out of the 95 samples collected (two out of eight from song thrushes *Turdus philomelos*, and one out of nine from blackbirds *Turdus merula*), indicating alphavirus presence.

Sequencing of the 434 bp amplicon from the three positive bird samples and the two positive controls, and comparison with the NCBI GenBank, identified all as identical to the originally isolated M78 strain of Whataroa virus (Ross et al. 1964; Maguire et al. 1967), and distinct from the closest related other known alphavirus strains (Australian strains of Whataroa virus; Saleh et al. 2004). The same 325 bp region of the 434 bp amplicon from all three sequences obtained from the positive bird samples was identical to that of the M78 strain of Whataroa virus present in GenBank (Accession no. U94606.1). Although we did not carry out virus isolation here we are confident that the virus detected by RT-PCR is indeed Whataroa virus; the highly improbable alternative being that it is a previously undetected virus infecting birds in the Whataroa area that aligns closer to the M78 strain of Whataroa virus than even other strains of Whataroa virus.

## Discussion

By detecting Whataroa virus in blackbirds and song thrushes around Whataroa township in this study we have demonstrated that the virus has not faded out in the four decades since neutralizing antibody to the virus was last detected in wild birds in the area (Miles et al. 1971; Miles 1973). This demonstrates that the failure of a previous study to detect the virus in mosquitoes known to vector the virus, collected from the same area and tested with a similar RT-PCR methodology to that employed here (Snell et al. 2007), was probably due to insufficient mosquitoes being screened (a likelihood that the authors of the study raise). These data also suggest that failure to detect the virus in smaller numbers of mosquitoes collected from other regions of New Zealand in the same study may not be informative of virus status in those regions.

Here we detected Whataroa virus by screening tens of birds, as opposed to the thousands of mosquitoes screened in Snell et al. (2007), and indeed the thousands shown to be required for virus detection in Australia (Doggett et al. 2000). This highlights how the surveillance of wildlife hosts for infectious agents is far more efficient in terms of number of samples required than surveillance for agents in vectors. This will particularly be the case for new incursions or agents persisting at low levels. Wildlife surveillance may also be the more cost-effective option in temperate-climate countries such as New Zealand, given that (1) larger vector sample sizes may be required to detect pathogens compared with more tropical climates, due to environmental effects on vector competence, and (2) in many locations and seasons in temperate climates it is too time-consuming or even unfeasible to collect large numbers of adult vectors.

Although we have demonstrated that Whataroa virus has not faded out, its status in New Zealand as emerging or persisting is hard to interpret from our results. Sensitivity of detection is probably not an issue, since the RT-PCR used here and the serology techniques used for the 1964–1969 survey are both highly sensitive (Miles et al. 1971; Pfeffer et al. 1997). However, the two techniques are measuring fundamentally different biological parameters. The serological test used for the previous survey detects Whataroa virus neutralizing antibody in samples, whereas the RT-PCR only detects live virus. For alphaviruses infecting birds, neutralizing antibodies can persist for years post-infection (Main et al. 1988; Lundstrom & Niklasson 1996), while active viraemia generally only lasts for a few days (Lundstrom et al. 1993). However, it is interesting to note that the prevalence of infection by live virus in blackbirds and song thrushes detected here was of equal magnitude to the seroprevalence recorded in the 1960s (Table 1). Even though our field survey was conducted

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during the main period of virus spread, for such agreement to occur either (1) the prevalence of infection by live virus has increased substantially since the 1960s (i.e., the virus has 'emerged') or (2) the duration of viraemia in blackbirds and song thrushes is unusually long.

If virus emergence has indeed occurred, one would expect similar agreement between the two measures for the other species surveyed here. This is clearly not so, with no live virus being detected in 78 individuals from species in which the average prevalence of Whataroa virus neutralizing antibody ranged from 0.07 to 0.21 in the 1964–1969 surveys. Hence the case for emergence does not hold. Rather, our observations raise some interesting questions about infection dynamics in the two *Turdus* species, as opposed to other non-native birds. If Whataroa virus infections persist longer in these species than is the norm, it would make them potential reservoirs from which 'spillover' infection to other bird species occurs. Indeed, this hypothesis was originally proposed by Miles et al. (1971), who concluded from the 1964–1969 survey that the virus was being maintained by *Turdus* spp., since the proportion of these birds having virus neutralizing antibody was relatively constant from season to season. Interestingly, blackbirds are also hypothesized to be a reservoir of avian malaria in New Zealand (Tompkins & Gleeson 2006; Sturrock & Tompkins 2008).

Given that mosquito-borne diseases have emerged globally (Edman 2005; Jones et al. 2008), regionally in Australia and the Pacific (Mackenzie et al. 2004; Russel & Kay 2004), and locally in New Zealand (Tompkins & Gleeson 2006) over the past few decades, the question remains as to why Whataroa virus appears not to have likewise emerged in New Zealand. Although, based on the present study, we cannot comment on past versus present geographical distribution of the virus, the data reported here indicate that a substantial increase in prevalence in the Whataroa area has not occurred. This is in contrast to avian malaria, where prevalence of infection has increased greatly over the past few decades, from only a few local patches of very low prevalence to prevalence levels of 50% or greater in some bird species in several parts of the country (Tompkins & Gleeson 2006).

With mosquito-borne disease emergence generally linked to changes in mosquito community composition and abundance (Gubler 2002), the most parsimonious explanation for the apparent lack of Whataroa virus emergence is that neither of these vector characteristics has changed substantially in the Whataroa area of New Zealand over the past four decades. This explanation agrees with the observation that two invasive exotic mosquito species in New Zealand for which there is evidence of competence as alphavirus vectors (*Aedes notoscriptus* and *Culex quinquefasciatus*) have not yet been recorded in the Whataroa area (Holder et al. 1999); of particular interest is *C. quinquefasciatus*, already hypothesized to be a factor behind the emergence of avian malaria in New Zealand (Tompkins & Gleeson 2006). If these mosquito species do spread to this region, a programme of virus surveillance in wild birds is recommended. Such surveillance should also extend to the wild brushtail possum (*Trichosurus vulpecula*) population, in which 17 of 76 animals sampled by Miles et al. (1971, 1973) had serum neutralizing antibodies indicative of Whataroa virus infection, to further investigate the role of this species in the virus transmission cycle.

## Acknowledgements

We thank Vernon Ward and Andrew Mercer of the Department of Microbiology and Immunology, University of Otago, for providing the M78 strain of Whataroa virus and for advice on the RT-PCR. Thanks also to Pete Lei for field assistance, David Slaney for discussion, Phil Cowan and two anonymous referees for comments on a previous version of the manuscript, and Christine Bezar and Anna Meyer for editorial assistance. This work was funded by the Ministry of Research, Science and Technology (New Zealand) Capability Fund, and

was carried out under Landcare Research Animal Ethics Approval 06/10/04.

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