New Zealand Veterinary Journal
Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/tnzv20

Evidence for absence of equine arteritis virus in the horse population of New Zealand

A MJ McFadden [a] , P V Pearce [b] , D Orr [a] , K Nicoll [c] , T G Rawdon [d] , H Pharo [c] & M Stone [c]

[a] Ministry for Primary Industries , Wallaceville , PO Box 40742, Upper Hutt , 5018 , New Zealand

[b] Equine Health Association , Marton 4710, Manawatu, New Zealand

[c] Standards Branch , Ministry for Primary Industries , PO Box 2526, Wellington , New Zealand

[d] Animal Health Laboratory, Wallaceville , Ministry for Primary Industries , PO Box 40742, Upper Hutt , 5018 , New Zealand

Accepted author version posted online: 21 Dec 2012.Published online: 24 Apr 2013.

To cite this article: A MJ McFadden , P V Pearce , D Orr , K Nicoll , T G Rawdon , H Pharo & M Stone (2013) Evidence for absence of equine arteritis virus in the horse population of New Zealand, New Zealand Veterinary Journal, 61:5, 300-304, DOI: 10.1080/00480169.2012.755664

To link to this article: http://dx.doi.org/10.1080/00480169.2012.755664

Short Communication

Evidence for absence of equine arteritis virus in the horse population of New Zealand

AMJ McFadden*§, PV Pearce†, D Orr*, K Nicoll‡, TG Rawdon#, H Pharo‡ and M Stone‡

Abstract

AIM: To summarise investigation and laboratory data collected between 2001 and 2011 to provide evidence that equine arteritis virus is not present in the horse population of New Zealand.

METHODS: Analysis was carried out on results from laboratory tests carried out at the Ministry for Primary Industries Animal Health Laboratory (AHL) for equine arteritis virus from horses tested prior to being imported or exported, testing of stallions as part of the New Zealand equine viral arteritis (EVA) control scheme and testing as part of transboundary animal disease (TAD) investigations for exclusion of EVA. Horse breeds were categorised as Thoroughbred, Standardbred or other.

RESULTS: A total of 7,157 EVA serological test records (from import and export testing, EVA control scheme testing and TAD investigations) were available for analysis between 2005 and 2011. For the three breed categories a seroprevalence of ≤1.6% at the 95% confidence level was determined for each category. Between 2001 and 2011, as part of the EVA control scheme, the EVA status of 465 stallions was determined to be negative. During 2005–2011 EVA was excluded from 84 TAD investigations.

CONCLUSIONS: There was no evidence of equine arteritis virus being present in the general horse population outside of carrier stallions managed under the EVA control scheme.

CLINICAL RELEVANCE: Equine arteritis virus is absent from the general horse population of New Zealand.

KEY WORDS: *: Equine arteritis virus, equine viral arteritis, EVA, equid, horse, freedom, absence*

Introduction

Equine arteritis virus is an RNA virus from the Arteriviridae family. It was first isolated from an outbreak of respiratory disease and abortion of horses in the United States of America in 1953 (Doll *et al.* 1957). The virus has a worldwide distribution in the horse population with only Japan and Iceland considered to be free (Holyoak *et al.* 2008). The seroprevalence has been found to vary both within and between countries. Within an affected country, the main factors determining variation in prevalence relate to breed and horse sector. The influence of breed on prevalence may relate more to cultural and management factors than to genetics (Holyoak *et al.* 2008).

Despite worldwide distribution of the virus, outbreaks involving overt clinical disease are reported only sporadically, with most infections being asymptomatic (Timoney and McCollum 2004). The severity of clinical disease varies with the virus strain and environmental stressors (Holyoak *et al.* 2008). Where the virus has high pathogenicity there can be a direct financial cost through the effect of disease on individual animals, and the costs of controlling an outbreak. EVA is one of the listed diseases of horses by the OIE (World Organisation for Animal Health; Anonymous 2012), so the most important impact is its effect on trade, which is generally unrelated to the severity of disease and the pathogenicity of the strain of virus present.

Transmission of the virus occurs venereally from infected carrier stallions or through respiratory infection from recently infected equids. Excretion of the virus in the semen of infected stallions can be life-long, but its excretion in respiratory secretions is relatively short lived, generally less than 3 weeks (McCollum *et al.* 1971). The chronically infected stallion is the maintenance host of infection, with the virus found in the secondary sex glands; particularly the ampulla of the vas deferens (Timoney and McCollum 2004). Maintenance of infection is testosterone dependant, with the carrier state not being maintained in the gelding. There can be a temporary effect on fertility from an acute infection but there is no effect on the fertility of persistently infected stallions (Holyoak *et al.* 2008).

* Ministry for Primary Industries, Wallaceville, PO Box 40742, Upper Hutt 5018, New Zealand
† Equine Health Association, Marton 4710, Manawatu, New Zealand
‡ Standards Branch, Ministry for Primary Industries, PO Box 2526, Wellington, New Zealand
# Animal Health Laboratory, Wallaceville, Ministry for Primary Industries, PO Box 40742, Upper Hutt 5018, New Zealand
§ Author for correspondence. andrew.mcfadden@mpi.govt.nz

http://dx.doi.org/10.1080/00480169.2012.755664

AHL	Animal Health Laboratory
EVA	Equine viral arteritis
OIE	World Organisation for Animal Health
TAD	Transboundary animal disease
VNT	Virus neutralisation test

Equine arteritis virus was first determined to be present in horses in New Zealand in 1988. The release of the virus was considered to have occurred from horses imported from North America (McKenzie 1988; Ricketts 1998). Positive serological tests for equine arteritis virus had been reported in 1981, 7 years prior to confirmation of the virus (Jolly *et al.* 1986); however it is likely that these results were false positives caused by non-specificity of the virus neutralisation test (VNT) used at the time (McKenzie 1989; Ricketts 1998). A serological survey carried out in 1989 showed that the virus had been circulating widely in the Standardbred sector, with 54 (95% CI 45–63)% of Standardbreds being serologically positive. In Thoroughbreds, only three (95% CI 0.4–5.5)% were seropositive for the virus using the VNT (McKenzie 1989).

In 1989, soon after the detection of equine arteritis virus in New Zealand, the disease was made notifiable (Anonymous 2010) and an EVA control scheme implemented. The ultimate aim of the scheme was eradication of the virus from the horse population in New Zealand (McKenzie 1988, 1989). The main components of the scheme were serological testing of breeding stallions, with additional viral culture of semen where the stallion was seropositive. The scheme involved a number of controls on the use of carrier stallions and included the quarantine of inseminated mares (McKenzie 1990; Ricketts 1998; O'Flaherty and Reid 2005).

An estimate of the seroprevalence in New Zealand was updated in 1990 from the results of additional stallions tested as part of the EVA control scheme (McKenzie 1990). At this time three (95% CI 1–5)% of Thoroughbred and 37 (95% CI 31–43)% of Standardbred stallions were seropositive to equine arteritis virus using the VNT. Low VNT titres were obtained from the Thoroughbred stallions tested compared with very high titres from the Standardbreds. All seropositive Thoroughbred stallions were semen tested using viral culture and none were found to be carriers of the virus. There were no seropositive stallions detected from testing 121 horses of other breeds (95% CI 0–4). During the period 1997–1998 a Standardbred stallion previously confirmed as free of EVA, and who had stood at the same stud as a carrier stallion, was found to be semen test-positive. It was determined that the semen from the carrier stallion had been inadvertently used to service mares outside the required quarantine regime. A trace back of contacts identified only this one additional carrier stallion (Horner 2004). Consequentially the scheme was modified by incorporating controls for the use of semen from shedder stallions and vaccination against equine arteritis virus of stallions standing alongside carrier stallions (O'Flaherty 2002).

A summary of testing carried out as part of the EVA control scheme in 2002 showed that during the period between 1989 and 2002, despite the breakdown in 1997–1998, the programme had been effective, with a declining seroprevalence in the horse population as well as a reduction in the number of known carriers. The number of carrier stallions declined from a maximum of 20 in 1991–1992 to three in 2002 (Horner 2004). In June 2012 the last carrier stallion was subject to euthanasia at the age of 20 years. No stallion known to be a carrier of equine arteritis virus remains in New Zealand. Clinical signs of disease have not been observed in horses in New Zealand since EVA was first diagnosed in 1988 (Evans 1991; Reid 2009).

The aim of this paper is to provide a summary of investigation and laboratory data collected between 2001 and 2011 to provide evidence that EVA is not present in the general horse population of New Zealand.

Methods

In New Zealand three different surveillance streams have been used to detect equine arteritis virus if it were present. These surveillance streams were: testing for EVA in horses being imported or exported, the New Zealand EVA control scheme, and transboundary animal disease (TAD) investigations of suspect cases of EVA. Analysis was carried out on the total serological data accumulated from all three of these surveillance streams between 2005 and 2011. The status of all animals tested serologically with the VNT for equine arteritis virus outside known carrier animals was determined to be negative, with any titres investigated and excluded as being caused by exposure to the virus.

Additional analysis was carried out on data from the EVA control scheme (2001–2011), and from TAD investigations (2005–2011). Serological data from the EVA scheme and TAD investigations represent a subset of all serological testing carried out for EVA.

Total serological testing 2005 to 2011

Data were obtained from results of serological testing of horses for EVA at the Animal Health Laboratory (AHL) to fulfil the requirements of export and import health standards, testing of stallions as part of the New Zealand EVA control scheme and testing as part of TAD investigations for exclusion of EVA. Due to the way that data had been recorded, it was not always possible to differentiate between tests carried out for export and import purposes. Some records represented multiple tests on the same horse, as the import health standard for some countries requires that a horse be tested twice. Duplicate testing may also arise out of the requirements of the EVA control scheme or during testing of stallions in export semen collection centres, with some stallions tested a number of times during their period of standing at stud. The laboratory methods used to carry out the VNT have been previously described by Horner (2004). Data collected from results of laboratory tests carried out at the AHL were analysed for the period from January 2005 to November 2011. Each horse tested was grouped into one of three breed categories: Thoroughbred, Standardbred, or other.

EVA control scheme 2001 to 2011

Serological data from stallions tested by VNT with or without supplementary semen culture from the EVA control scheme were collated between April 2001 and the end of 2011. This date range was selected on the basis that it represented the time period that had elapsed since the last known carrier stallion in New Zealand had been identified. As part of the scheme any stallion found to have positive serology was semen tested. The laboratory methods used to carry out virus culture have been previously described by Horner (2004).

Transboundary animal disease investigations 2005 to 2011

Data from investigations of suspect TAD for the period from January 2005 to November 2011 were collated by reviewing the internal AHL summary report for each investigation carried out. The AHL was notified of any suspicions of EVA based on clinical signs through a toll-free hotline. Reports were received from many different sources including horse owners, veterinarians and regional laboratory pathologists. A decision tree was used to determine the need to investigate further (McFadden and Stone 2006).

For all of these notifications the veterinarian or horse owner/trainer concerned was interviewed to confirm the clinical presentation, determine if other horses were affected, and identify any risk factors such as the movement history of the horse that could provide a pathway for exposure of the horse to equine arteritis virus. As part of the investigation planning, a specific differential list was developed to ensure all TAD of concern were investigated. For the more routine notifications, paired serology for EVA was all that was required for exclusion.

In addition to reported clinical suspicions, investigations were carried out to determine the cause of any VNT titres to equine arteritis virus detected through pre-export testing or testing as part of New Zealand's EVA control scheme. Investigation involved a variety of methods to exclude exposure to equine arteritis virus, including retesting the seropositive horse and testing in-contact horses to determine if there was evidence of exposure to circulating virus. For stallions, semen testing by virus isolation and/or PCR was undertaken (McFadden and Stone 2006).

Data were categorised on the basis of the sector making the report, clinical signs present, routine haematology findings, single or multiple horses affected, the age of the horse affected and the range of definitive diagnoses reached, for those investigations where these data were available.

Statistical analysis

All VNT serological data relating to EVA testing were imported into Microsoft Access 2003 and stratified by test reason (import/export testing, EVA control scheme or TAD investigation) and by breed category. Given the negative status for EVA of all horses tested, 95% CI around non-detection were determined for each category based on the number of horses sampled/tested. CI were calculated using the epiR package in R v2.15 (R Development Core Team, 2011; R Foundation for Statistical Computing, Vienna, Austria).

Results

Total serological testing

A total of 7,157 EVA serological test records were available for analysis for 2005–2011. Of these records 6,598 were from import/export tests, 283 were from stallions tested as part of the EVA scheme and 276 were from TAD investigations. Twenty-nine breeds of horses or horse-types were represented in the data. The horse-types included equestrian, sport horse, warm blood and polo pony. The median number of horses within these breed groups was seven (min 1, max 5,369). After categorisation into three breed categories, a seroprevalence of ≤1.6% was determined for each category (Table 1).

Table 1. Number of serum samples collected from horses between 2005 and 2011 and tested for Equine viral arteritis using virus neutralisation tests, by breed category, with 95% CI for zero prevalence. Samples were from horses tested as part of import and export requirements, the New Zealand equine viral arteritis control scheme and from transboundary animal disease investigations.

Breed category	Number tested	95% CI
Thoroughbred	5,369	0–0.1%
Standardbred	344	0–1.6%
Other	826	0–0.7%
No breed data	618	0–0.9%
Total	7,157	0–0.1%

EVA control scheme

Between 2001 and 2011 the EVA status of 465 stallions was found to be negative as part of the EVA control scheme. Negative status was determined either through serological testing (n=389) or from negative virus culture of semen where stallions were serologically positive as a result of vaccination (n=93). Of the 93 stallions that had semen cultured, 43 (46%) had been semen tested in multiple years. Where breed was identified, 83/89 (93%) semen samples were from Standardbred stallions, indicating a high rate of vaccination in this breed and the need to use virus culture as a method of exclusion.

After categorisation of the 465 stallions into three breed categories, a seroprevalence of ≤9% was determined for stallions of the three categories (Table 2). In the 'other' category there were 27 breeds of stallion, with 65/181 (36%) being Quarter Horse, and 45/181 (25%) Appaloosa.

Transboundary animal disease investigations

During 2005–2011 there were 84 equine TAD investigations carried out to exclude EVA. Whilst some of the investigations were initiated because of positive serology, the majority were initiated because of suspicious clinical signs or haematological findings in the affected horse/s. For investigations initiated on these grounds, 48 notifications were received from regional veterinary laboratories and 17 from private veterinarians.

The array of clinical signs apparent in animals where an investigation was undertaken was reviewed. In these cases, 38/69 (55%) had clinical oedema and 31/69 (45%) anaemia alone, while 14/69 (20%) had both these signs. Only 9/49 (18%) showed abnormal respiratory signs, 6/71 (8%) had a history of recent abortion, 19/64 (30%) were recorded as being pyrexic, and 37/68 (54%) had inflammatory changes evident on a leucogram. The majority (70/77; 91%) of investigations concerned a single affected horse at a property, and the remainder were on properties with more than one affected animal. The change in the denominator presented in these figures reflects missing data on the presence of clinical presentation in affected horses from some investigations.

The median age of horses investigated was 4 years (mean 6.9 years, min 4 months, max 35 years). Of the cases investigated 48/76 (63%) were male (geldings, colts and stallions). Of the 56 horses where breed was recorded, 35 were Thoroughbreds, 11 Standardbreds, four warm bloods, two Arabians, two Clydesdales, one Appaloosa and one Shetland pony.

Table 2. Number of stallions tested for Equine viral arteritis as part of the New Zealand equine viral arteritis control scheme between 2001 and 2011, by breed category, with 95% CI for zero prevalence.

Breed category	Number tested	95% CI
Thoroughbred	57	0–9%
Standardbred	117	0–5%
Other	181	0–3%
No breed data	110	0–4%
Total	465	0–1%

Equine viral arteritis was excluded from all investigations undertaken. A definitive diagnosis was reached in only 19 cases.

Where positive serological results for equine arteritis virus initiated the investigation it was determined that these titres were either due to cross reactions or due to vaccination prior to the horse being imported into New Zealand. A total of 276 sera were tested for equine arteritis virus as part of these TAD investigations, with none being positive. Case examples of investigations conducted to exclude infectious causes of seropositive samples have been previously described (Stone 2005; McFadden and Stone 2006; Bingham 2006, 2007, 2011, 2012).

Discussion

This paper has provided evidence for the absence of circulating equine arteritis virus in the horse population of New Zealand. Other than known carrier Standardbred stallions and mares quarantined under the rules of the EVA control scheme, no new infections have been detected for over a decade. For this period on-going import health controls, surveillance and the EVA control scheme measures have prevented further incursions from occurring and provided a means of detecting evidence of the virus if it were present. Analysis was carried out on the three surveillance streams that have been used over this period. The New Zealand EVA control scheme has focused on detecting and isolating carrier stallions responsible for venereal transmission. General serology data supported by TAD investigations has demonstrated absence of any transmission in the general horse population.

Analysis of seven years of serological test data established that exposure of horses to EVA, if present, was less than 2% within each of three breed categories. Limited on-farm biosecurity provides the potential for transmission between infected mares and other horse classes (agistment, finishing, training) either through direct or indirect pathways (Rogers and Cogger 2010; Rosanowski *et al.* 2012a,b). Hence a pathway remains for the transmission of equine arteritis virus to susceptible equids outside a stud property if an unidentified carrier stallion were present. If that were to happen, epidemics in the general horse population could occur through respiratory transmission of the virus.

The confidence provided by negative serological data that equine arteritis virus is not present in New Zealand, and the external validity of the findings from this analysis, is dependent on one key assumption; that there has been sufficient mixing of horses within each breed category to allow spread of the virus by the respiratory route from recently infected horses. That being the case, the deficiencies of interpreting results from non-random testing of horses will be negated. Mixing of horses from different breed categories is known to occur, with horse properties often having horses for multiple purposes such as recreation, racing, breeding and competition (Rosanowski *et al.* 2012c). However, for some types of specialty breeds mixing may not be a valid assumption. In addition, the amount of mixing by breed or breed type may be variable, as horses that do not have a purpose associated with income or competition have fewer movements and less consequential mixing than those that do (Rosanowski 2013). Hence, transmission of infectious respiratory disease may occur at a lower rate for some breeds than for Thoroughbreds and Standardbreds.

Whilst it is possible that a carrier could exist in a minor breed of horse with little transmission outside its immediate breeding contacts, there has never been any evidence of a carrier animal existing in any breed of horse in New Zealand other than the Standardbred. Given the level of testing of stallions in the EVA control scheme and in the horse export industry, it is considered likely that if equine arteritis virus had been present in a minor breed, it would have eventually spilled over into one of the horse sectors that are intensively tested for EVA.

Where sera tested positive for antibodies to equine arteritis virus, an investigation was carried out to exclude the possibility of exposure of the horse to the virus. These positive sera generally had low titres, and in all instances an investigation involving retesting of serum at an OIE reference laboratory, or testing of in-contacts or semen testing of stallions, failed to detect any evidence that there was circulating virus. All investigations that were initiated on the basis of clinical signs consistent with EVA ruled out equine arteritis virus as a cause. As there was a wide array of clinical signs initiating investigations it is considered that the sensitivity of surveillance was high.

Analysis of serological data was restricted to that collected over a 7-year period. Given the high confidence of absence of EVA provided by this analysis, we considered that inclusion of additional data would exceed the objectives of this survey. It is doubtful that there would have been sufficient data from a substantially longer period of analysis to stratify data by individual breed, rather than the three breed categories used, due to the large number of breeds of horse.

There is likely to be bias in the general serology data examined, as most testing was carried out for the purposes of import and export testing. There was an obvious breed bias in the data with the majority of horses from the Thoroughbred sector, where carrier stallions have not been detected. The data are also likely to be biased by factors specific to horses being traded. Given the cost of international horse travel, higher value horses are more likely to be traded between countries. This bias was also observed in the TAD investigation data with Thoroughbreds being the predominant breed investigated for suspicious clinical signs. On the other hand, stallions tested as part of the EVA control scheme or at export semen collection centres were more likely to be Standardbreds. There are likely to be other biases affecting the investigation data relating to socioeconomic factors and the type of activity of the horse influencing the probability of a veterinary callout and subsequent submission of blood to a laboratory. Hence horses with low-intensity health management are less likely to be included as part of general surveillance.

Venereal transmission is likely to be more important than respiratory transmission of equine arteritis virus, as the number of carrier stallions is a key determinant of the prevalence of EVA within a breed (Holyoak *et al.* 2008). No new carriers were detected from 465 stallions tested as part of the EVA control scheme over the 11-year period of this study. Approximately one-third of those stallions tested were breeds other than Thoroughbred and Standardbreds, demonstrating that these breeds in New Zealand have remained consistently free of equine arteritis virus.

Analysis of data collected between 2001 and 2011 has provided evidence that through its import health controls and EVA control scheme New Zealand has been successful in preventing the circulation of equine arteritis virus in the horse population.

With the death of the last carrier stallion managed under the EVA control scheme, New Zealand can confidently declare absence of equine arteritis virus from its horse population.

Acknowledgements

The authors would like to thank past and present laboratory technical staff at the Animal Health Laboratory (AHL) for carrying out the rigorous testing, Gary Horner for test development, and Susan Cork, Wlodek Stanislawek, Kok-mun Tham, Jianning Wang, Richard Clough, David Pulford and Grant Munro for test supervision; members of the Animals and Marine Surveillance and Investigation Team and the large number of Ministry for Primary Industry staff, New Zealand Equine Health Association committee members and private veterinarians who have been committed to gaining EVA freedom over the last three decades; and Matthew McFadden for critically reviewing the manuscript. All funding for the project was provided as a part of investigation of transboundary animal disease carried out by the AHL.

References

*Anonymous. *Biosecurity (Notifiable Organisms)* Order 2010. http://www.legislation.govt.nz/regulation/public/2010/0265/latest/whole.html?search=ts_regulation_biosecurity_resel&p=1 (accessed 13 July 2012). New Zealand Government, Wellington, NZ, 2010

*Anonymous. *OIE Listed Diseases*. http://www.oie.int/animal-health-in-the-world/oie-listed-diseases-2012/ (accessed 20 June 2012). OIE, Paris, France, 2012

*Bingham P. Quarterly report of investigations of suspected exotic diseases. *Surveillance* 33 (1), 17–23, 2006

*Bingham P. Quarterly report of investigations of suspected exotic diseases. *Surveillance* 34 (3), 27–31, 2007

*Bingham P. Quarterly report of investigations of suspected exotic diseases. *Surveillance* 38 (1), 33–44, 2011

*Bingham P. Quarterly report of investigations of suspected exotic diseases. *Surveillance* 39 (1), 23–6, 2012

Doll ER, Bryans JT, McCollum WH, Crowe M. Isolation of a filterable agent causing arteritis of horses and abortion by mares: its differentiation from the equine abortion (influenza) virus. *Cornell Veterinary* 47, 3–40, 1957

*Evans MHD. Observations on the clinical effects of equine viral arteritis. *Surveillance* 18 (4), 40, 1991

Holyoak GR, Balasuriya UBR, Broaddus CC, Timoney PJ. Equine viral arteritis: current status and prevention. *Theriogenology* 70, 403–14, 2008

Horner GW. Equine viral arteritis control scheme: a brief review with emphasis on laboratory aspects of the scheme in New Zealand. *New Zealand Veterinary Journal* 52, 82–4, 2004

Jolly PD, Fu ZF, Robinson AJ. Viruses associated with respiratory disease of horses in New Zealand: an update. *New Zealand Veterinary Journal* 34, 46–50, 1986

McCollum WH, Prickett ME, Bryans JT. Temporal distribution of *equine arteritis virus* in respiratory mucosa, tissues and body fluids of horses infected by inhalation. *Research in Veterinary Science* 12, 459–64, 1971

*McFadden AMJ, Stone MAB. Surveillance and incursion response planning for equine viral arteritis, equine infectious anaemia and equine influenza in New Zealand. *Proceedings of the Annual Seminar of the New Zealand Equine Veterinary Association, New Zealand Veterinary Association*. Pp 67–77, 2006

*McKenzie JS. Equine viral arteritis and trade in horses from the USA. *Surveillance* 15 (5), 6–7, 1988

*McKenzie JS. Survey of stallions for equine viral arteritis. *Surveillance* 16 (4), 17–18, 1989

*McKenzie JS. Compulsory control programme for *equine viral arteritis*. *Surveillance* 17 (1), 19–20, 1990

*O'Flaherty J. Equine viral arteritis control scheme. *Surveillance* 29 (2), 23–4, 2002

*O'Flaherty J, Reid W. Equine viral arteritis control scheme. *Surveillance* 3 (2), 26, 2005

*Reid W. Equine viral arteritis control scheme. *Surveillance* 36 (2), 20, 2009

*Ricketts W. Equine viral arteritis. *Surveillance* 25 (3), 12–3, 1999

Rogers CW, Cogger N. A cross-sectional survey of biosecurity practices on Thoroughbred stud farms in New Zealand. *New Zealand Veterinary Journal* 58, 64–8, 2010

Rosanowski SM, Rogers CW, Cogger N, Benschop J, Stevenson MA. The implementation of biosecurity practices and visitor protocols on non-commercial horse properties in New Zealand. *Preventive Veterinary Medicine* 107, 85–94, 2012a

Rosanowski SM, Cogger N, Rogers CW. An investigation of the movement patterns and biosecurity practices on Thoroughbred and Standardbred stud farms in New Zealand. Preventive Veterinary Medicine, doi: 10.1016/j.prevetmed.2012.08.003, 2012b

Rosanowski SM, Cogger N, Rogers CW, Benschop J, Stevenson MA. A description of the demographic characteristics of the New Zealand non-commercial horse population with data collected using a generalised random-tessellation stratified sampling design. *Preventive Veterinary Medicine*, 107, 242–52, 2012c

Rosanowski SM, Cogger N, Rogers CW, Bolwell CF, Benschop J, Stevenson MA. Analysis of horse movements from non-commercial horse properties in New Zealand. *New Zealand Veterinary Journal*, doi: 10.1080/00480169.2012.750571, 2013

Stone M. Quarterly report of investigations of suspected exotic diseases. *Surveillance* 32 (3), 16–9, 2005

Timoney PJ, McCollum WH. Equine viral arteritis. In: Coetzer J, Tustin R (eds). *Infectious Diseases of Livestock*. Pp 924–32. Oxford University Press, Capetown, South Africa, 2004

Submitted 27 July 2012

Accepted for publication 19 October 2012

First published online 21 December 2012

*Non-peer-reviewed