EQUINE MORBILLIVIRUS INFECTION: EPIDEMIOLOGICAL ASPECTS OF THE 1994 QUEENSLAND OUTBREAKS

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ABSTRACT

Two separate outbreaks of disease due to infection with equine morbillivirus (EMV) have occurred in Queensland. Although first reported as occurring in Brisbane, the first outbreak actually occurred near Mackay in northern coastal Queensland in August 1994 and involved the death of 2 horses and a male horse breeder. The second outbreak occurred in Brisbane in September 1994 where 14 horses and their trainer died and a further 7 horses and a stablehand were infected but survived. The 7 infected horses were later destroyed. Despite considerable effort, no link has yet been established between the 2 outbreaks.

Extensive follow-up investigations of in-contact horses as well as clinical and serological surveillance in horses and other animals following both outbreaks has revealed no further evidence of infection in Queensland. Although other sources cannot be ruled out at this stage, it is thought that EMV infection in horses is the result of spillover from an unknown wildlife reservoir. Wildlife surveillance for EMV has not yet revealed a specific reservoir of infection but investigations are continuing. Finding a reservoir will be a difficult task.

The evidence to date suggests that EMV is not endemic in the Queensland horse population, and that spillover of EMV from a wildlife reservoir is rare. In addition, in both outbreaks transmission appears to have occurred only from the index case, suggesting that EMV is not transmitted easily among horses.

INTRODUCTION

The first descriptions of disease due to infection with equine morbillivirus (EMV) in man (Selvey and Sheridan 1994; Selvey *et al.* 1995) and in horses (Murray *et al.* 1995) arose from an outbreak which occurred in a Brisbane racehorse stable complex in September 1994. Subsequently, investigation of an acute encephalitis in a farmer, in October 1995, revealed that a smaller outbreak of EMV had occurred near Mackay in north Queensland during August 1994 (Allworth *et al.* 1995). These 2 incidents are the only known outbreaks of EMV infection to have occurred anywhere in the world.

This paper describes the 2 outbreaks with an emphasis on epidemiological aspects. The outbreaks are described in the chronological order of events rather than in the order of the investigations.

THE MACKAY OUTBREAK

Although this episode was first investigated in October 1995, the outbreak occurred in August 1994 on a property near Mackay in north coastal Queensland. The property, owned and managed by a farmer and his wife, a veterinary surgeon, was used for growing sugar cane and breeding Thoroughbred horses.

On 1st August 1994, a heavily pregnant, 10-year-old brood mare (Horse A) died suddenly in a paddock. Reported clinical signs were severe respiratory distress, ataxia and swelling of the head, particularly the area of the infraorbital fossa and the cheeks. A second horse, a 2-year-old stallion (Horse B) from an adjoining paddock was reported to have licked the carcase. This horse died on 12th August within 24 h of becoming ill with signs of aimless pacing, muscle trembling and a haemorrhagic nasal discharge. Post mortem examinations were carried out on both horses and specimens were sent to a private veterinary laboratory. Histopathological findings were inconclusive.

The farmer assisted with the treatment and subsequent autopsies of both horses. He became ill in August/September 1994 with a mild meningo-encephalitis which improved with antibiotic therapy. In mid-September 1995, he developed a severe encephalitis-like syndrome and died on 21st October 1995. He had IgG antibody to EMV and his cerebrospinal fluid was positive to EMV on polymerase chain reaction (Allworth *et al.* 1995).

The farmer's death led to a retrospective investigation of the horse deaths. Limited preserved

material from both horses was still available in October 1995 and was tested for presence of EMV using PCR and immunostaining. The conclusion was that, in Horse B, EMV was the cause of death and that Horse A was infected but the cause of death could not be confirmed because there was insufficient material for a histopathological diagnosis. From all the available evidence it is reasonable to assume that Horse A was a case of EMV and that Horse B was infected from Horse A.

Horse A had been in the same paddock in the company of a number of other horses for 2 months prior to death. Approximately 10 days prior to death she received tetanus toxoid, had her feet trimmed and was wormed.

Extensive serological surveillance was undertaken and the initial sampling completed by 23rd November 1995 with negative findings. This surveillance included bleeding all other domestic animals on the affected and surrounding properties as well as horses which had spent any time on the property from July 1994. In addition, a serological survey of Queensland horses kept in paddocks was undertaken with negative findings in 2,024 animals tested (Ward *et al.* 1996). Sampling of wildlife and some avian species was also undertaken in an attempt to identify a possible reservoir host, again with negative findings (Rogers *et al.* 1996). The overall serological surveillance for both the Mackay and Brisbane outbreaks is summarised in Table 1.

TABLE 1: Summary of total serological surveillance for equine morbillivirus with negative findings by serum neutralisation test from September 1994 to April 1995

Animal species	Number sampled
Horse - Mackay*	2,869
Horse - Brisbane†	1,964
Donkey	1
Cattle, goat, pig	287
Chicken, duck, goose, turkey	33
Other birds	2
Cat	564
Dog	23
Cane toad, bandicoot, flying fox, kangaroo, mouse, snake, wallaby, other wildlife	166
Total	**5,909**

*Horses sampled in 1995 following detection of the Mackay outbreak.
†Horses sampled in 1994 following the Brisbane outbreak.

THE BRISBANE OUTBREAK

In September 1994 a focal outbreak of an acute respiratory syndrome resulted in the death of 14 horses and their trainer (Selvey and Sheridan 1994). Presenting signs were suggestive of African Horse Sickness, a disease exotic to Australia, or a toxicosis. Subsequent investigations revealed the cause to be a hitherto undescribed morbillivirus now known as equine morbillivirus (Murray *et al.* 1995).

The outbreak occurred in a stable complex in the suburb of Hendra in Brisbane. The putative index case was a pregnant mare which was ill on 7th September when moved from a paddock in the suburb of Cannon Hill to the Hendra stables where 23 other horses were located.

A further 16 horses became ill between the 16th and 23rd September and 12 of these died from several hours to 3 days after the onset of illness. Another horse was reported to have died on approximately 2nd September in the Cannon Hill paddock but it could not be established whether or not it was a case of the same syndrome. Three other horses seroconverted but did not show demonstrable clinical signs. Two of the recovered horses developed mild neurological signs consisting of myoclonic twitches similar to those seen in some dogs following infection with canine distemper virus. The signs were transient in one case but persisted until euthanasia in the other (Rogers *et al.* 1996). The trainer and one of the other people in close contact with the putative index case also became ill and, despite intensive medical care, the trainer died. The incubation period in the natural cases in horses was mostly 8–11 days with a maximum of 16 days.

The pattern of the outbreak suggested that all other cases were a result of transmission from the putative index case. The most likely means of natural transmission was via frothy nasal discharges as a consequence of close contact or mechanical transfer. Aerosol transmission seemed unlikely as sneezing and coughing were not a feature of the syndrome. Transmission did not appear to occur during the incubatory phase of the disease. All of the available evidence suggested that, under the conditions of the outbreak, EMV was not highly contagious (Baldock *et al.* 1995).

Quarantine and strict movement restrictions were used to contain the outbreak. Surveillance showed that the outbreak was contained successfully and was an isolated focal event. The surveillance strategy had 3 components: targeted clinical and serological surveillance through tracing of risky contacts; passive clinical surveillance based on reports from private practitioners and the

general public; and a planned serological survey to ascertain whether or not infection with EMV was more widespread (Baldock *et al.* 1995).

Similarities between the Mackay and Brisbane outbreaks

Despite considerable effort, no link has yet been established between the Mackay and Brisbane incidents. However, the fact that they occurred so close together in time, in addition to other similarities, suggests there may be some, as yet undiscovered, link. The points of similarity between the 2 incidents are worth summarising:

- Both outbreaks occurred in a 2-month period in late winter to early spring 1994;
- The index case was an older, heavily pregnant mare at pasture;
- The mare had been in the paddock for a duration well in excess of the incubation period observed in other horses;
- Other horses which were in the paddock with the index case remained well and did not seroconvert;
- Transmission occurred from the index case but there was apparently no secondary transmission in horses;
- Infection appears to have been transmitted from a horse to a person.

Susceptibility of other species

The 2 outbreaks have shown that both horses and people are susceptible. Because of the risk to man, EMV is regarded as a biohazard level 4 agent requiring maximum biosecurity. The only veterinary laboratory in Australia with a capacity to work with such agents is the Australian Animal Health Laboratory (AAHL) at Geelong (L. Gleeson personal communication).

Limited experimental studies at AAHL have shown that cats and some guinea pigs succumb to the disease following inoculation with large doses of the virus. Affected animals showed respiratory signs in the terminal stages with pulmonary lesions similar to those occurring in horses (Westbury *et al.* 1995).

Mice, rats, rabbits, chickens and dogs did not show clinical signs, there were no gross or microscopic lesions evident 21 days after challenge, virus could not be isolated and mice and chickens did not seroconvert. However, equivocal neutralising antibody titres were detected in some rats and one of 2 inoculated dogs whereas the 2 inoculated rabbits had significant titres of 1:2560 and 1:320 (Westbury *et al.* 1995).

Discussion

The characteristic and severe nature of EMV infection, as well as the lack of evidence for exposure in the Queensland horse population, indicates that horses are not a maintenance host for EMV. If the virus persists in a wildlife reservoir then spillover is rare and the subsequent outbreak in susceptible species appears to be short and self limiting.

EMV is not highly infectious and requires direct contact for natural transmission to occur, possibly involving nasal discharges which occur in the terminal stages of the disease. The microscopic pathology of affected lungs indicates that such discharges are likely to contain large quantities of virus. In the Mackay incident, the route of infection for Horse B was most likely either oral or nasal because it was reported to have licked the dead mare through a fence. The evidence for a specific route of infection in the Brisbane outbreak is not as strong but a naso-oral route is probable. In both outbreaks, evidence indicated that transmission only occurred from the index case with no secondary transmission. In addition, fully susceptible horses which were in contact with cases did not become infected. There is no evidence for transmission from horses during the incubatory phase of the disease.

As at April 1996 the source of the virus remained unknown. Finding the source of EMV is likely to prove difficult and is the subject of ongoing research. The other identified research priorities are the development of diagnostic tests which do not require use of live virus, pathogenesis and environmental persistence, and therapy and prevention, particularly in man.

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