Zoonotic Disease in Australia Caused by a Novel Member of the Paramyxoviridae

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Twenty-three horses and three humans in Queensland, Australia, were infected with a novel member of the Paramyxoviridae family of viruses in two geographically distinct outbreaks. Two of the humans died—one died of rapid-onset respiratory illness, and the other died of encephalitis. The third infected human developed an influenza-like illness and made a complete recovery. All infected humans had close contact with sick horses. Since the two outbreaks occurred at sites 1,000 km apart and no known contact between the two groups of humans and horses occurred, extensive testing of animals and birds common to the two areas was conducted. Fruit bats (Pteropus species) were found to carry a virus identical to that found in the infected humans and horses. Although there was no contact between the infected humans and the bats, some form of close contact between the horses and bats is the likely mode of infection.

A new member of the virus family Paramyxoviridae, coined the equine morbillivirus, which has recently been described in Australia [1–5, 5a], appears to truly qualify as a cause of an emerging infectious disease. It is an infection that has newly appeared in a population and thus fulfills one of the criteria for an emerging infectious disease as described by Morse [6]. The occurrence of this infectious disease demonstrates that the zoonotic pool appears to be by no means exhausted [6]. The disease mysteriously appeared in the state of Queensland, Australia (figure 1), in the latter half of 1994 and has been responsible for the deaths of two humans and 16 horses. We describe the clinical presentation of human infection with this virus and the subsequent seroepidemiological studies that were conducted. Although our aim is to concentrate on the clinical aspects of this infection, new developments in the taxonomy of the virus will be described briefly.

Case Reports

Patient 1. A previously healthy 49-year-old male horse trainer became ill in September 1994 [1–3]. A number of horses in the stables at which he worked had recently become ill, with loss of appetite, dyspnea, and a copious frothy nasal discharge. The horse trainer had had percutaneous exposure to nasal and oral secretions from the first sick horse via numerous abrasions on his hands and arms. In all, 18 horses at the stables became ill, and 14 died. Three horses were subsequently found to be seropositive for the virus but were asymptomatic.

After 4 days of nonspecific symptoms (myalgia, lethargy, sore throat, and headaches), the horse trainer developed nausea, vomiting, dyspnea, dry cough, and finally, respiratory compromise necessitating mechanical ventilation. Physical examination revealed that he was markedly hypoxemic, and there was diffuse alveolar shadowing on a chest radiograph (figure 2). Laboratory investigations revealed thrombocytopenia (platelet count, 46 × 10^9/L), mild hepatitis (aspartate aminotransferase level, 128 U/L [normal range, 2–40 U/L]; alanine aminotransferase level, 61 U/L [normal range, 2–40 U/L]), and elevated levels of lactate dehydrogenase (672 U/L; normal range, 100–200 U/L) and creatine phosphokinase (2,227 U/L; normal range, 10–60 U/L).

The initial differential diagnosis included legionellosis, viral pneumonitis, pneumococcal pneumonia, melioidosis, glanders, and paraquat poisoning. Empirical therapy with erythromycin and rifampin (for possible legionellosis), penicillin (for possible pneumococcal pneumonia), and ceftazidime (for possible glanders or melioidosis) was begun. Cultures of sputum, blood, and bronchial washings, obtained before initiation of antibiotic therapy, were negative for bacterial pathogens. Immunofluorescence assays of bronchial washings and serum were negative for Legionella species. Despite antibiotic therapy and the addition of methylprednisolone to the regimen, the patient’s respiratory and renal function deteriorated. Arterial thrombosis developed in his right leg. After 7 days of intensive therapy, and despite some improvement in respiratory function, he developed cardiac arrhythmias and died.

Autopsy revealed congested, hemorrhagic lungs. Microscopic examination of the lungs revealed foci of necrotizing alveolitis with giant cells, some syncytial formation, and viral...
pest, like measles and canine distemper viruses, is a member of the genus Morbillivirus, family Paramyxoviridae. Rinderpest virus antisera gave a very weak reaction by western blot but did not neutralize the virus [3]. However, on the basis of this weak reaction, primer pairs were designed to amplify regions from the matrix, fusion, or L proteins of morbilliviruses. With use of PCR, the matrix protein primer pair amplified a 400-bp product. This product was sequenced.

A pair of primers (5′-CATGTAGATGCCGGAGTCAT and 5′-TTGTGTTCGGGTCCTCTGGC) was designed to be specific for this amplified product. These primers gave a specific product of ~200 bp when the equine viral RNA and the virus isolated from the kidney of the human case were used. Sequence analysis revealed that PCR products from the viruses isolated from the horses and the patient were identical. This sequence had ~50% similarity with the matrix proteins of morbilliviruses. The sequence was quite different from that of the original Rinderpest virus used in the initial immunologic testing. Phylogenetic analyses of the matrix protein sequences indicated that the newly discovered virus was distantly related to other known Paramyxoviridae.

Figure 1. Map of Australia and Papua New Guinea, showing the locations of the two outbreaks in Queensland (Brisbane and Mackay). Bats infected with the novel virus have been detected in Brisbane and Mackay as well as Madang, Darwin (Northern Territory), and Melbourne (Victoria).

inclusions (figure 3). Serology for a comprehensive range of respiratory viruses (including hantaviruses, arboviruses, and HIV) and for *Leptospira* species, *Coxiella burnetti*, and *Burkholderia pseudomallei* was negative [1].

Lung, liver, kidney, and spleen samples obtained at autopsy were inoculated onto Vero, LLC-MK2 (rhesus monkey kidney cells), and MRC5 (human fetal lung cells) cell lines at the Centre for Public Health Sciences in Brisbane, Queensland, Australia. After 12 days, the MRC5 and LLC-MK2 cell lines inoculated with kidney material exhibited prominent syncytia. Virus had already been isolated from the tissues of the affected horses at the Commonwealth Scientific and Industrial Research Organization (CSIRO) Australian Animal Health Laboratory, Geelong, Victoria, Australia [2, 7]. Molecular, electron microscopic, and serological analyses of the human isolate showed it to be identical to the equine isolates. Electron microscopic examination of the cultured virus revealed a pleomorphic, enveloped virus, ranging in size from 38 nm to 600 nm. The envelope had a double-fringed appearance because of coverage with 10-nm and 18-nm surface projections. Free-lying herringbone-shaped nucleocapsids, 18 nm wide with a periodicity of 5 nm, were seen [8].

As these observations were consistent with the appearance of a member of the family Paramyxoviridae, the virus was tested against antisera to these viruses. With use of immunofluorescence and protein immunoblot analyses, all antisera except that to Rinderpest virus gave negative reactions. Rinder-
and anxiety, he has remained well after >2 years of follow-up. Examination of blood obtained in May 1996 showed that he continued to have significant levels of neutralizing antibodies in his serum (titer, 1:160) but that there was no evidence of viral genetic material by PCR.

**Patient 3.** A 36-year-old male developed aseptic meningitis in August 1994 [4, 5]. He was a sugar cane farmer who lived on a horse stud in Mackay, Queensland, Australia. This city is ~1,000 km (600 miles) north of Brisbane (figure 1). The man had cared for and assisted in the autopsies of two horses that had become ill several weeks before he developed meningitis. Like patient 1, he had several abrasions on his limbs related to the physical nature of his occupation. Stored, formalin-fixed tissue specimens obtained from the horses were retrospectively found to be infected with the novel member of the Paramyxoviridae [9]. Neither the horses nor this patient had recently been in Brisbane. There was no known link between patients 1 and 2 and this patient, nor had his horses been in contact with the horses described in the first case.

The initial illness in August 1994 consisted of 12 days of sore throat, malaise, headache, anorexia, and vomiting. Physical examination revealed a drowsy but rousable man with a stiff neck. A CT of the brain was normal. Two courses of oral antibiotics had been given during the initial course of his illness. Examination of CSF, obtained by lumbar puncture during oral antibiotic therapy, revealed a WBC count of 560 \(10^6/\text{L} (82\% \text{polymorphonuclear cells}). The CSF protein level was elevated (1.82 g/L), but the glucose level was normal. Bacterial and viral cultures of CSF were negative. A diagnosis of partially treated bacterial meningitis was made, and the patient was given intravenous ceftriaxone and benzylpenicillin for 10 days. He made a full recovery.

It was determined that serum samples (from horses that had recovered from the infection), which were able to neutralize the newly described virus, also neutralized virus isolated from the first patient [2, 3]. Furthermore, immunoelectronmicroscopy revealed that the virus from the patient reacted with serum from recovered horses and the serum samples from the patient [8]. Homogenates of spleen and lung from the dead horses were used to determine if other horses could be infected [2]. Two healthy horses, at a different geographic location, were given intravenous or nebulized tissue homogenates. After a little over 1 week, they developed high fevers and respiratory illnesses. The virus was isolated from lungs, liver, kidneys, and lymph nodes of these experimental horses.

**Patient 2.** A previously well, 40-year-old male stablehand became ill at the same time as the outbreak mentioned above [1–3]. He developed myalgia, sore throat, headaches, lethargy, and vertigo. He had no cough, chest pain, or dyspnea. The results of physical examination, chest radiography, a biochemical examination, and hematologic examination were normal. He did not require hospitalization and made a complete recovery. Neutralizing antibodies were detected in his serum 10 and 20 days after the onset of his illness. Apart from some fatigue respiratory illness but not in stored serum obtained in May 1994. It was determined that serum samples (from horses that had recovered from the infection), which were able to neutralize the newly described virus, also neutralized virus isolated from the first patient [2, 3]. Furthermore, immunoelectronmicroscopy revealed that the virus from the patient reacted with serum from recovered horses and the serum samples from the patient [8]. Homogenates of spleen and lung from the dead horses were used to determine if other horses could be infected [2]. Two healthy horses, at a different geographic location, were given intravenous or nebulized tissue homogenates. After a little over 1 week, they developed high fevers and respiratory illnesses. The virus was isolated from lungs, liver, kidneys, and lymph nodes of these experimental horses.

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The patient had an exacerbation of long-standing low back pain after his meningitic illness, and in January 1995 he underwent an uncomplicated left lumbosacral laminectomy and nerve root decompression, with relief of his symptoms. After this procedure, he had a mild nonproductive cough that persisted for 3 months, but he was otherwise asymptomatic and returned to work. He became ill again in September 1995, when he reported a 2-week history of irritable mood and recurrent back pain. He then had three focal motor seizures involving his right arm, followed by his first generalized tonic-clonic seizure. Findings on a CT of the brain were reportedly normal, and CSF obtained by lumbar puncture contained 440 \(10^6\text{WBCs/\text{L}}, 9\% \text{of which were polymorphonuclear cells}.

The CSF glucose level was normal, but the protein level was again elevated at 0.88 g/L. Bacterial and viral cultures were negative. Serology for all viruses known to cause neurological illnesses in Australia, including Japanese encephalitis, Australian encephalitis, Kunjin, Alfuy, Kokobera, Stratford, and Edge Hill viruses, was negative.

Fevers, frequent focal motor seizures and generalized tonic-clonic seizures, and altered consciousness followed. There were no respiratory symptoms or signs. Penicillin, ceftazidime, tri-
methoprim-sulfamethoxazole, iv acyclovir (10 mg/kg every 8 hours), and dexamethasone were given, without improvement in the patient’s condition. An MRI study of the brain initially revealed subtle multifocal cortical changes, with marked deterioration and progression of his illness (figure 4). He died 25 days after admission to the hospital.

CSF obtained during his initial illness in August 1994 was not available. Serum collected in August 1994 was positive for neutralizing antibodies to the novel member of the Paramyxoviridae described in the first case report. Although the titer of antibodies was low (1:4), it was quite specific, since screening of several hundred serum samples from individuals who had had close contact with the infected horses or the patients provided completely negative results (titer, 1:2). A nested PCR assay of this patient’s serum sample was also positive. Epidemiologically and microbiologically, therefore, it seems that he acquired the infection from the two sick horses, which were also positive. Evidently the virus had remained latent until his second illness in September 1995. Serum samples obtained at this time showed a marked rise in titer compared with those obtained during the 1994 illness. Indirect immunofluorescence was also positive. An ELISA, developed at the CSIRO Australian Animal Health Laboratory by Dr. P. Selleck, was also positive. A PCR assay of CSF from September 1995 revealed a product of identical molecular weight to that of the product from the clinical specimens obtained from patient 1. Sequencing of this product revealed it to be identical to the product obtained from patient 1 and to that from the horses affected in that outbreak [5].

Autopsy revealed mild interstitial pneumonitis, with endothelial cell multinucleation in the liver, spleen, and lungs [5]. Examination of the brain and meninges revealed lymphocytic leptomenigitis. Foci of necrosis were seen in the neocortex, basal ganglia, brainstem, and cerebellum. Occasional multinucleated endothelial cells were seen in the brain. Immunohistochemistry of brain tissue with use of rabbit antiserum to the novel member of the Paramyxoviridae was positive. Electron microscopy also showed aggregates of nucleocapsids within the neurons, and these nucleocapsids reacted with serum from the recovered horses, as visualized by immunoelectronmicroscopy [8].

**Seroepidemiologic Studies**

In view of the high attack rate among the horses described in the first case report and the patient’s rapidly fatal illness, there was considerable concern about spread to the rest of the human population. This concern escalated with the death of patient 3. The results of serological testing of humans who have had contact with horses has been presented by McCormack et al. [10]. Fifteen individuals who had had contact with the infected horses at postmortems were tested serologically and were negative. Six others who had had close contact with the sick horses in Brisbane or Mackay and 113 individuals who had had lesser contact with the horses were also seronegative.

Concern was also expressed that the virus could be transmitted to health care workers in the course of their duties. One hundred fifty-nine health care workers who had had varying levels of contact with the patients in cases 1 and 3 were seronegative [10].

Since it was speculated that the new virus might have a natural host in a native animal species, particularly one that might be capable of migrating between the two outbreak sites or be present at both sites, widespread testing of animal populations was performed. More than 2,000 horses were tested and were negative. More than 5,000 serum samples from 46 other species were tested and were also negative [11, 12]. However, subsequent investigations revealed that 20 of 224 serum samples from four species of Australian bats (belonging to the suborder Megachiroptera) were positive for neutralizing antibodies to the newly described member of the Paramyxoviridae [13, 13a]. In September 1996, a paramyxovirus was isolated from a grey-headed flying fox (*Pteropus poliocephalus*), which was closely related, if not identical, to the previously described virus [13]. As a result, concern was expressed that humans
handling bats might be at risk of acquiring the virus [14]. At least 70% of bat handlers have reported being bitten by bats, and 88% have reported being scratched, raising concern about parenteral exposure to bat secretions. One hundred twenty-eight bat handlers were tested for antibodies to the virus, and all were negative [14]. As many of these people had cared for sick bats, and many had cared for bats for a considerable time, it would seem that transmission of the virus between bats and humans must be extremely rare, if it occurs at all.

The results of serologic studies of humans and animals are summarized in Table 1.

**Taxonomy of the Virus**

Although the virus was originally termed equine morbillivirus, it now appears that it is not a morbillivirus at all. Antisera to known morbilliviruses do not neutralize the virus or react with infected cells in immunofluorescence tests [3]. The surface projections of known morbilliviruses are \( \sim 15 \) nm in length, but the new virus possesses surface projections of two distinct lengths, 15 nm and 18 nm (Figure 5) [8]. Although the genome of the new virus (Figure 6) has an overall structure identical to that of the morbilliviruses, sequence homology between the new virus and the known morbilliviruses is limited [5a, 15]. However, sequence analyses have shown that the virus is clearly a member of the Paramyxovirinae subfamily. Furthermore, morphologically, it is enveloped, pleomorphic, and covered with surface projections and contains herringbone-shaped nucleocapsids, just like other Paramyxoviridae [3, 8]. Characterization of its proteins by polyacrylamide gel electrophoresis show that its structural proteins resemble those of other Paramyxoviridae [5a].

However, the size of the genome of the virus is significantly larger (>18 kb) than that of other members of the Paramyxovirinae (ranging from 15 kb to 16.5 kb) [16]. This larger genome is due to the significantly larger intergenic sequences seen in the new virus. In contrast, the size of the genes of the new virus is very similar to that of the genes of other viruses in the family, with the exception of the P gene, which is larger. In addition, the order of genes is identical (Figure 6) [15]. On the other hand, the sequence of the genome is substantially different from that of the genomes of other members of the Paramyxovirinae [15].

On the basis of these findings, it has been suggested that this virus should be included as a new genus within the family Paramyxoviridae, subfamily Paramyxovirinae [5a]. It appears to occupy a taxonomic place between the morbillivirus and paramyxovirus genera. Since its genome is larger than those of all other members of the Paramyxoviridae, one suggestion has been to call the genus megamyxovirus and to term the virus itself, Hendra virus, after the suburb of Brisbane in which the first outbreak occurred [6, 15].

**Discussion**

No other human infections with the virus have been diagnosed since September 1995. It appears that all human and equine cases became medically evident at around the same time (Table 2). The two clusters of infection occurred \( \sim 1,000 \) km apart, and there was no contact between the horses and humans associated with the Brisbane outbreak and those associated with the Mackay outbreak. However, it was always considered that there was a potential wildlife reservoir that could be widely distributed. The isolation of a closely related virus in fruit bats raises the likelihood that this mobile species of nonterrestrial mammal was the link between the two outbreaks. However, it is not known how the virus crossed from bats into horses. Horses may have grazed into bat-excrement-contaminated areas under fruit trees or have been scratched or contaminated by an infected bat. The occurrence of outbreaks from August to October, which coincides with the birthing season for fruit bats, together with the observation that the virus can be isolated from uterine fluids [13a], suggest that horses may have been infected from contact with parturient bats. A novel lyssavirus has recently been described in these bats [16]. Therefore, another intriguing possibility is that a neurologically damaged bat may have transmitted infection to the horses.

Transmission of the virus from horses to humans appears most likely to be through extensive contact with infectious respiratory secretions. Both patients who died had breaches in skin integrity that may have been portals of entry. The lack of transmission to those conducting autopsies on the horses described in the first case report or to the health care workers involved in both cases argues against frank droplet transmission. Standard precautions would appear to be sufficient to prevent transmission within health care facilities.

It is of interest that previously unrecognized Paramyxoviridae that have affected marine mammals—phocine distemper virus, porpoise morbillivirus, and dolphin morbillivirus—have recently been described [17, 18]. No human infections have been recorded. These viruses have, however, shown an ability to appear in unexpected species, suggesting that their host range

### Table 1. Results of serological studies used to test for a new member of the Paramyxoviridae in humans and animals.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Serum neutralizing antibody</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. (%) positive</td>
</tr>
<tr>
<td>Humans in close contact</td>
<td></td>
</tr>
<tr>
<td>with sick or dead horses</td>
<td>3 (2.2)</td>
</tr>
<tr>
<td>Humans in contact with</td>
<td>0</td>
</tr>
<tr>
<td>other human cases</td>
<td>0</td>
</tr>
<tr>
<td>Humans in contact with bats</td>
<td></td>
</tr>
<tr>
<td>with horses</td>
<td>23 (0.9)</td>
</tr>
<tr>
<td><em>Pteropus</em> bats</td>
<td>39 (12.5)</td>
</tr>
<tr>
<td>Other animals in Australia</td>
<td>0</td>
</tr>
</tbody>
</table>

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can often be wider than that observed in the first instance [18]. For example, canine distemper virus has been transmitted from jackals and wild dogs to lions, hyenas, leopards, and foxes in Tanzania in recent years [19].

The clinical illnesses of the patients described above appear quite different, although the findings at autopsy suggest that both patients with fatal cases had disseminated infection. There are several possible explanations for the third patient’s biphasic presentation. One is that the initial illness was akin to a sero-converting illness that was followed by a latent stage. Alternatively, his illness could have had a pathogenesis similar to that of subacute sclerosing panencephalitis (SSPE). Similarities between the two illnesses include their causes (a paramyxovirus) and progressions (initial infection, followed later by neurological decline and death). We were unable to isolate virus from the brain of the third patient, which raises an additional question that this virus was in a defective, cell-associated form (as with SSPE). However, the time course and microscopic appearances of specimens in case 3 are quite different from those of SSPE. Reinfection during the time between the two neurological illnesses seems unlikely, as the patient had no exposure to sick horses during this time.

<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
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<tbody>
<tr>
<td>August 1994</td>
<td>Two horses in Mackay, Queensland, Australia, become ill and die</td>
</tr>
<tr>
<td>August 1994</td>
<td>Patient 3 develops aseptic meningitis</td>
</tr>
<tr>
<td>September 1994</td>
<td>Eighteen horses in Brisbane, Queensland, Australia, become ill, and 14 die</td>
</tr>
<tr>
<td>September 1994</td>
<td>Patient 2 develops an influenza-like illness</td>
</tr>
<tr>
<td>September 1994</td>
<td>Patient 1 develops a fatal respiratory illness</td>
</tr>
<tr>
<td>September 1995</td>
<td>Patient 3 develops fatal encephalitis</td>
</tr>
<tr>
<td>September 1996</td>
<td>Virus is isolated in bats</td>
</tr>
</tbody>
</table>

Table 1. Time course of infections with a newly described member of the Paramyxoviridae.

Figure 5. Electron micrograph of the novel member of the Paramyxoviridae. The nucleocapsid is apparent (arrowhead), as are the outer spikes (large arrow) and inner spikes (small arrow) (bar, 100 nm). Electron micrograph courtesy of A. Hyatt.

Figure 6. Genome structure of the novel virus. The boxes indicate protein coding regions, the numbers in parentheses after the protein name represent protein size in amino acid residues, while the sizes of noncoding intergenic regions are indicated by nucleotides (nt) with a down-pointing open arrow. The vertical bar in between each gene represents the position of intercistronic sequences. Data from [15].
The only antiviral treatment that has been used against the virus has been acyclovir (patient 3). This therapy appeared not to have any impact on the outcome of the infection. Ribavirin has been used with some success against another member of the Paramyxoviridae (respiratory syncytial virus) [20], and its use intravenously should probably be attempted in future cases of the infection. Vitamin A has been used to decrease the severity of measles [21] and could also be considered. Measles vaccination is highly unlikely to be protective against the new virus, as both patients 1 and 3 had IgG antibodies to measles virus at the time of the onset of illness.

We do not know if subsequent cases of this viral infection will appear, but as with most emerging infectious diseases, the initial cases reported present many difficult questions for clinicians and researchers. These cases have taught us that the prompt isolation of a causative organism is a key not just to solving an individual diagnostic dilemma but also to understanding why a disease may emerge. Sufficient expertise and financial and technical resources must be available to make new diagnoses. In addition, the disparate presentations in cases 1 and 3 remind us that the diagnostic net needs to be cast widely in cases of severe life-threatening illness. New diseases may present in a manner that has not been previously encountered.

Addendum

Since our report was submitted, a new virus of the family Paramyxoviridae, distinct from the virus we described, has been isolated from pigs in New South Wales, Australia. Two patients with intense occupational exposure to pigs had an influenza-like illness with rash and serological evaluation demonstrated high convalescent-phase antibody titers to this new virus but not to the virus that affected horses and humans in Queensland, Australia [22, 23].

Acknowledgments

Numerous organizations and individuals have contributed to the virological research and the care of patients infected with this new member of the Paramyxoviridae. These have included Linda Selvey and John Sheridan (Communicable Diseases Branch, Queensland Health); Alex Hyatt, Paul Selleck, and Lawrie Gleeson (the CSIRO Australian Animal Health Laboratory); Rachel Wells (Centers for Disease Control and Prevention); Tony Allworth and John O’Sullivan (Royal Brisbane Hospital); Lester Hiley (Centre for Public Health Sciences, Brisbane); and Russell Rogers, Peter Young, Kim Halpin, and Hume Field (Queensland Department of Primary Industries).

References


