

Australian bat lyssavirus infection in three fruit bats from north Queensland

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Abstract

We report the case findings of Australian bat lyssavirus infection in two black flying foxes (*Pteropus alecto*) and one little red flying fox (*Pteropus scapulatus*) from north Queensland between January 1995 and August 1996. Although the *P. alecto* case in January 1995 is the first recognised case of Australian bat lyssavirus infection in Australia, this was a retrospective diagnosis made after identification of the index case at Ballina in May 1996. Eight persons had exposure to the three bats. Serum antibodies to classical rabies virus were measured in six of these persons; the only one seropositive was a veterinarian who had previously been vaccinated against rabies. Six persons received rabies vaccine following exposure. None of the in-contact humans developed signs of lyssavirus infection. For people exposed to Australian bat lyssavirus-positive bats who have not been scratched or bitten or had mucosal contamination by these bats, we suggest a post-exposure regime of five inoculations of the human diploid cell inactivated rabies vaccine. *Comm Dis Intell* 1997;21:117-120.

Introduction

Lyssavirus infection was first diagnosed in two black flying foxes (*Pteropus alecto*) from Ballina, New South Wales in 1996^{1,2}. Both bats exhibited neurological signs and had mild

to severe encephalitis². The lyssavirus isolated was found to be significantly different from known genotypes in the *Lyssavirus* genus².

In early November 1996, a bat carer from Rockhampton died

from a diffuse encephalitis. Australian bat lyssavirus was detected in her cerebrospinal fluid by polymerase chain reaction and her serum contained neutralising antibodies to classical rabies virus³. The woman had cared

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for fruit bats in the two to four weeks preceding her illness and had been scratched by them. She had also cared for an insectivorous bat six weeks prior to the onset of clinical signs and received a bite from the bat. The woman had also cared for a variety of native animals in the recent past.

We report three cases of fruit bats infected with Australian bat lyssavirus, and describe the public health actions associated with them.

Bat infections

Case 1 occurred in January 1995 and was an adult wild female black flying fox (*P. alecto*) found behaving aggressively in the back yard of a house in Townsville. Intracytoplasmic eosinophilic inclusions in neurones were detected in histological sections of brain. The bat also had histological and biochemical evidence of lead toxicosis.

Case 2 occurred in May 1996 and was an adult wild male black flying

fox (*P. alecto*) with hind limb paresis, found under a tree in Charters Towers. The brain appeared histologically normal, but changes in other organs indicated a bacterial septicaemia.

Case 3 was a little red flying fox (*Pteropus scapulatus*) found in August 1996, with hind limb paresis and clonic muscle spasm, in a suburban garden in Townsville. A non-suppurative meningoencephalitis was present on histology.

Table. Details of exposure of persons to Australian bat lyssavirus-positive bats from north Queensland and subsequent vaccination histories

Person	Date of exposure	Date of vaccination	Vaccination regime	Delay ¹	Comments
<i>Exposed to bat case 1</i>					
Vet 1	Jan 1995	previously vaccinated	booster Nov 1996	na	Exposed at necropsy; neutralising antibodies prior to booster >2 IU/mL
Bat carer 1	Jan 1995	Dec 1996	pre-exposure	23 months	Retrospective diagnosis 21 months; no penetrating wounds; opted for pre-exposure regime
Bat carer 2	Jan 1995	Dec 1996	pre-exposure	23 months	Retrospective diagnosis 21 months; no penetrating wounds; opted for pre-exposure regime
<i>Exposed to bat case 2</i>					
Bat carer 3	May 1996	Dec 1996	post-exposure	6 months	Retrospective diagnosis three months; no penetrating wounds; opted for post-exposure regime after scratch from another bat
Vet 2	Aug 1996	Oct 1996	post-exposure	2.5 months	Iatrogenic wound during necropsy of bat frozen for three months; lack of knowledge about pathogenicity delayed vaccination
Vet 3	Aug 1996	Oct 1996	pre-exposure	2 months	Exposed at necropsy, but no obvious high risk exposure factors; opted for pre-exposure regime
<i>Exposed to bat case 3</i>					
Bat carer 1	Aug 1996	see above	see above		No penetrating wounds
Bat carer 2	Aug 1996	see above	see above		No penetrating wounds
Vet 4	Aug 1996	previously vaccinated	na	na	Exposed at necropsy; annual antirabies titre protective
Vet 5	Aug 1996	nil	na	na	Exposure in clinical setting; no penetrating wound

1. 'Delay' is the time from exposure to Australian bat lyssavirus-positive bat until vaccination.
na = not applicable

For all cases, samples of brain stored at -70°C were submitted to the CSIRO Australian Animal Health Laboratory (AAHL). Brain impression smears stained strongly for lyssavirus on the immunofluorescent antibody test (IFAT), and Australian bat lyssavirus was isolated.

Human contacts

At least eight people had close contact with the three fruit bats (Table). These included three bat carers and five veterinarians, four of whom performed post-mortem examinations on the bats. Time from contact with a particular bat to knowledge of the infection status of the bat ranged from two weeks to 21 months. None of the carers had sustained obvious penetrating wounds or scratches. One veterinarian (Vet 2) had cut a finger with a scalpel blade during the necropsy.

Neutralising antibodies to classical rabies virus were measured at AAHL for six of the eight persons after exposure. One veterinarian (Vet 4) had been vaccinated against rabies virus in 1989 and since then had had annual confirmation of a protective antirabies antibody titre. His antibody status was not measured on this occasion. Five of the six persons tested were negative, while one veterinarian (Vet 1) was strongly positive with a level >2.0 IU/mL. He had received a full course of three doses of human diploid cell rabies vaccine in 1985, with three additional boosters, the last in 1987, and the antibody response was considered to be due to vaccination. The veterinarian (Vet 2) who had been cut during the post-mortem had received no rabies vaccinations and had a negative titre against rabies virus ten weeks after the event.

Vet 2 received a post-exposure vaccination regime of five inoculations of human diploid cell inactivated rabies vaccine commencing two and a half months after the injury, while Vet 3 who assisted in the necropsy received a three-inoculation regime. Bat carer 3 received a standard five-inoculation post-exposure regime six months after caring for Case 2 after she was scratched by another bat with neurological signs. The latter bat was Australian bat lyssavirus-negative, but the regime was started before

infection status was known. The other two carers received a three-dose regime 23 months after interacting with Case 1. These carers were also exposed to Case 3. A veterinarian who had clinical contact with Case 3 and had no antibodies to rabies virus has not received any rabies inoculations to date, six months after exposure. None of the human contacts of these bats has shown clinical signs of encephalomyelitis.

Discussion

Bat infections

Neurological signs were present in all three fruit bats from north Queensland infected with Australian bat lyssavirus. Fruit bats will exhibit aggressive behaviour in specific types of social interactions, but the aggression shown by Case 1 was excessive. Cases 2 and 3 had hind limb paresis and showed no aggression. The signs of lyssavirus disease in these cases are similar to those seen in classical rabies cases⁴. Paralysis was seen in two of our cases and paralytic rabies is a more common presentation than furious rabies in most species⁴.

Case 1 predates the Australian bat lyssavirus infections in the black flying foxes from Ballina, the earliest of which was in March 1995². Currently Case 1 is therefore the first known Australian bat lyssavirus infection in Australia.

Neither of the black flying foxes (*P. alecto*) from north Queensland (Cases 1 and 2) had an encephalitis, although brain smears from both reacted strongly to the IFAT for lyssavirus antigen. Case 1 had histological and biochemical signs of lead toxicosis, while Case 2 had a terminal septicaemia in addition to the Australian bat lyssavirus infection. Both of these cases illustrate that other diagnoses with the potential to cause neurological signs do not exclude the possibility of infection with Australian bat lyssavirus.

The occurrence of Australian bat lyssavirus infection in Case 3, the little red flying fox (*P. scapulatus*), is the first report of Australian bat lyssavirus in this species. The little red flying fox (Case 3) had an encephalitis similar to that of the black flying foxes (*P. alecto*) from Ballina, and it is thus the third report of a fruit bat in Australia with

encephalitis caused by Australian bat lyssavirus.

Sick and dead bats that are presented to veterinarians in Townsville are now routinely necropsied, and specimens are sent to AAHL for testing for lyssavirus infection. Bat carer coordinators from Townsville report that in the past two years they have seen three other fruit bats which have exhibited abnormal aggressive behaviour prior to death. Two of these flying foxes chased and attacked other flying foxes. One of these aggressive flying foxes was seen at the same release cage as Case 1 and may have been bitten by Case 1 five weeks previously. Necropsies were not performed on these aggressive bats.

Human contacts

Two of the people who had contact with the three Australian bat lyssavirus infected bats received a post-exposure vaccination regime for rabies using the five-inoculation regime. Three of the remaining six chose to receive the three-inoculation pre-exposure regime, due to the lack of any penetrating wounds. This variation in management of humans exposed to lyssavirus-positive bats may reflect the lack of information available prior to the November 1996 guidelines of the Lyssavirus Expert Group. Two veterinarians had already been fully immunised against rabies, and both had protective levels of antibody.

The delays between potential exposure to Australian bat lyssavirus and post-exposure vaccination were due to several factors. Lyssavirus infection in two fruit bats was retrospectively diagnosed on archived specimens, leading to a delay in discovering the infection status of the bats. Lack of knowledge by individuals about the potential of Australian bat lyssavirus to cause disease in humans or animals other than bats resulted in poor motivation to seek vaccination. Results from pathogenicity studies on the Ballina isolate at the Centers for Disease Control and Prevention were not available until early November 1996, and the death of a human occurred after the cases described here. Prior to the meeting of the Lyssavirus Expert Group in November 1996⁵, protocols concerning actions to be taken after exposure to Australian bat lyssavirus were not defined.

The Lyssavirus Expert Group noted that inapparent exposure to lyssavirus could occur⁶. The current guidelines do not offer any definite advice for people who have been exposed to a lyssavirus-positive bat, but who are not aware of receiving any penetrating wound or contamination of mucous membranes with secretions⁵. We suggest that such persons should receive the standard five-inoculation

post-exposure regime using killed human diploid cell rabies vaccine.

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Salmonella in Victoria, 1997: the story so far

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Abstract

The Infectious Diseases Unit of the Department of Human Services, Victoria, reported an increased incidence of *Salmonella* infections in early 1997. To 21 April 1997, 944 notifications had been received, passing the previous year's total of 915. Five outbreaks of five separate serovars have been investigated and traced to their sources. The outbreaks, their sources and the control measures undertaken are described. Further clusters of other *Salmonella* serovars are being investigated. *Comm Dis Intell* 1997;21:120-122.

Introduction

The number of notified cases of *Salmonella* infections in Victoria has varied between 712 and 1,062 in the years 1991 to 1996. Notification rates per 100,000 population have been 23.7, 21.7 and 20.4 for 1994, 1995 and 1996 respectively. Notification rates to the National *Salmonella* Surveillance Scheme for the same three years were 21.7, 19.7 and 18.1 per 100,000 population. The Australian average notification rate in 1996 was 31.0 per 100,000 population.

To 21 April 1997, 944 notifications of *Salmonella* had been received in Victoria, passing the previous year's total of 915. A number of clusters were investigated.

Notable outbreaks which have been traced to a source in the past have included 47 cases of *Salmonella* Typhimurium 135 in 1991 associated with Italian-style ice cream (using uncooked eggs), and 54 cases of *Salmonella* Mbandaka in 1996 associated with peanut butter.

In late 1996, there was an outbreak of 36 cases of *Salmonella* Typhimurium RDNC A015 traced to a cafe in an outer suburban shopping centre. The implicated food in this outbreak was mayonnaise, which was made on the premises using raw eggs.

Methods

Investigations of *Salmonella* clusters begin with a weekly review of all notifications, including *Salmonella*, compared with historical data. Once it is identified that there is a cluster of

cases of the same serovar, an outbreak investigation is commenced. A standard questionnaire is administered by telephone to all notified cases in the cluster by staff of the Infectious Diseases Unit of the Department of Human Services. The questionnaire asks about the person's food history in the three days prior to becoming ill, and about foods consumed as part of their routine diet. Premises nominated by cases where foods have been purchased are also recorded. The data are constantly reviewed for possible links, and food sampling either from cases' homes or from nominated premises is undertaken as appropriate.

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