Getah virus is an arbovirus that was first isolated from mosquitoes in Malaysia in 1955. Outbreaks of infection by Getah virus have been recorded in horses in Japan and India. Infection can occur in a wide range of vertebrates, and the virus is probably maintained in a mosquito-pig-mosquito cycle. Clinical disease in horses is generally mild, characterised by pyrexia, oedema of the limbs, urticaria and a stiff gait. An inactivated whole-virus vaccine is available in Japan.

Introduction
Getah virus is an arbovirus that was first isolated from mosquitoes (Culex gelidus) in Malaysia in 1955 (Berge 1975). However, it was not until 1978 that the virus was shown to be responsible for a mild disease among racehorses in Japan (Kamada et al. 1980; Sentsui and Kono 1980a; Timoney 2004). Subsequent outbreaks of Getah virus infection have been documented in racehorses in Japan in the 1970s and 1980s (Sentsui and Kono 1985) and among breeding horses in India in 1990 (Brown and Timoney 1998). Infection with the virus also occurs in other domesticated animals, including pigs where it has been associated with illness and death in young piglets aged up to 18 days. Serological evidence of infection has been found in a large number of vertebrate species (mammals, birds and reptiles) in which the infection appears to be subclinical (Doherty et al. 1966). There is no evidence that the virus can infect man (Fukunaga et al. 2000).

Aetiology
Getah virus is a positive-stranded RNA virus belonging to the genus Alphavirus of the family Togaviridae (Calisher et al. 1980; Timoney 2004). It is classified in the Semliki Forest complex along with Semliki Forest, Bebaru, Ross River, Chikungunya, O’nyong-nyong, Una and Mayaro viruses (van Regenmortel et al. 2000). Several subtypes of Getah virus are recognised, including Sagiyama, Ross River and Bebaru viruses (Calisher and Walton 1996).

Getah virus possesses haemagglutinating and complement fixing antigens, which have been used to develop diagnostic tests for the virus. These tests in addition to the neutralisation test have been used to investigate antigenic relationships between different strains and subtypes of virus (Fukunaga et al. 2000).

Epidemiology
Getah virus is transmitted by mosquitoes, and is widely distributed in southeast Australasia and surrounding countries, including Australia, Borneo, Cambodia, China, Indonesia, Japan, Korea, Malaysia, Mongolia, the Philippines, Russia, Thailand, Sarawak, Siberia, Sri Lanka and Vietnam (Calisher and Walton 1996; Fukunaga et al. 2000; Timoney 2004). Natural infection in wildlife is believed to be subclinical (Fukunaga et al. 2000). In tropical areas of Asia, pigs appear to be important in maintaining a reservoir of infection via a mosquito-pig-mosquito cycle (Chanas et al. 1977). Pigs, and possibly other vertebrates, may play an important role as amplifying hosts for the virus (Fukunaga et al. 2000).

Serological surveys of horses in Japan have shown that the virus is widespread in this country, with seropositive rates up to 93% (Imagawa et al. 1981; Sentsui and Kono 1980b; Brown and Timoney 1998; Sugiura and Shimada 1999). The highest rates of seropositivity occurred in older horses in the cooler regions of northern Japan. Seroprevalence was reported as 17% in India and 25% in Hong Kong (Kamada et al. 1991; Shortridge et al. 1994). Many
cases of Getah virus infection in horses are subclinical (Fukunaga et al. 2000).

The principal vectors for the transmission of Getah virus to horses are mosquitoes (species of *Culex* or *Aedes*) (Calisher and Walton 1996; Fukunaga et al. 2000), although horse to horse transmission could also occur (Sentsui and Kono 1980a). High levels of virus can be found in nasal secretions of horses experimentally infected by Getah virus by the nasal route (Kamada et al. 1991), thereby permitting horse to horse spread in animals in close contact. Nonvector spread of the virus was believed to have contributed to the outbreak of Getah virus in India in 1990 (Brown and Timoney 1998).

### Clinical signs

Clinical infection caused by Getah virus has been observed almost exclusively in the horse. Outbreaks of the disease in horses have been sporadic, and have not been associated with any mortality (Fukunaga et al. 2000). The morbidity rate in one outbreak of infection in racehorses was 38% (Kamada et al. 1980; Sentsui and Kono 1980a), with slow and irregular spread of infection.

The clinical signs associated with Getah virus infection in horses vary depending on the strain of virus and viral dose (Timoney 2004). Some infections only produce a febrile response with associated inappetence and depression (Sentsui and Kono 1980a). The febrile stage lasts 1–4 days. Other signs seen in infected horses include lower limb oedema (Fig 1), swelling of the submandibular lymph nodes (Fig 2), urticaria (especially on the neck, shoulders and hind quarters) (Fig 3) and a stiff gait (Kamada et al. 1980; Sentsui and Kono 1980a; Fukunaga et al. 1981a; Brown and Timoney 1998). Mild colic, icterus and scrotal oedema have also been reported (Timoney 2004). Limb oedema and urticaria usually appear several days after the onset of pyrexia. Mild anaemia and a transient lymphopenia may be present (Fukunaga et al. 2000). Serum alkaline phosphatase can be elevated during the acute phase of the infection, whereas serum lactic dehydrogenase and glutamine pyruvic transaminase rise during convalescence (Calisher and Walton 1996). Most affected horses make a full clinical recovery within one week, although a small number may require up to 2 weeks to recover (Timoney 2004). Abortion is not a feature of the disease, and foals born to mares that...
have had the disease during gestation are normal
(Brown and Timoney 1998). In experimental
infections, the clinical signs are similar, but infected
horses also commonly develop a serous nasal
discharge (Kamada et al. 1991).

Diagnosis
In view of its clinical similarity to a range of other
infectious and noninfectious equine diseases, a
provisional clinical diagnosis must be confirmed
through laboratory examination. Included in a
differential diagnosis of Getah virus infection are
equine viral arteritis, equine rhinopneumonitis,
equine encephalosis, equine influenza, equine
infectious anaemia, African horse sickness fever,
purpura haemorrhagica and hoary alyssum toxicosis.
Virus isolation or demonstration of viral nucleic acid
by reverse-transcription polymerase chain reaction
may be performed on nasal swabs, unclotted blood or
saliva of acutely infected animals (Kamada et al.
1980; Sentsui et al. 1980; Fukunaga et al. 1981a). The
virus can be cultivated in a range of equine and
nonaqueine cell culture systems, especially Vero and
RK-13 cell lines, as well as in suckling mice
inoculated by the intracerebral route. Highest rates
of virus isolation have been achieved from plasma
(Fukunaga et al. 1981b). Detection of Getah virus is
optimal where specimens are collected as early as
possible after the onset of fever.

Serum antibodies to Getah virus can be detected
by haemagglutination inhibition, complement
fixation, enzyme-linked immunosorbent assay
(ELISA) and neutralisation tests (Kamada et al.
1980; Sentsui and Kono 1980; Sentsui and Kono
1985; Brown and Timoney 1998). If possible, paired
samples taken during the acute and convalescent
stages of the disease should be assayed. The
neutralisation test is considered the most specific
test at differentiating Getah virus infection from
other antigenically related alphaviruses (Chanas
et al. 1977).

Control
Control of Getah virus in endemic areas relies on
control of the mosquito vector (Fukunaga et al. 2000;
Timoney 2004). This can be achieved by eliminating
or reducing mosquito breeding sites, and use of
larvicides and adulticides. The risk of exposure of
horses to virus-infected mosquitoes can be reduced by
housing them during dusk and at night.

An inactivated whole-virus vaccine is available in
Japan. Horses receive an annual booster dose of the
vaccine in May or June, prior to the onset of the
mosquito season. No epidemics of Getah virus
infection have been recorded in horses in Japan since
the current vaccination programme was implemented
in 1979 (Timoney 2004).

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