A REVIEW OF VIRAL INFECTIONS OF HORSES*

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Introduction

Viruses are classified on the possession of a core of either desoxyribose or ribonucleic acid, the symmetry and structure of the outer protein coat of the virus particles, biophysical and biochemical properties and serological identity. Viruses can only be grown in living cells and early investigations of equine viral infections were hampered by the need for the work to be done in horses. Developments in the technique of *in vitro* cultivation of monolayers of living cells (cell or tissue culture) in the last 20 years have greatly aided the isolation of viruses infecting horses.

At least 58 viruses are now known to infect horses (Scott 1971). Some 28 of these viruses have been found to cause clinical disease, and for the other 30 viruses serological evidence only of infection of horses is available. In this article, viruses infecting horses (Table 1) are reviewed with special reference to those viruses now known to infect horses in Australia.

Equine Virus Infections and Disease

Virus infections of horses will be considered in relation to body systems commonly affected. As similar clinical signs may be produced in response to infection by several different groups of viruses, identification of an aetiological agent may also require virological examination. Exotic viruses infecting horses are not discussed in this article, because they are to be considered in detail in publications by the Exotic Diseases Subcommittee of the Australian Commonwealth and States Veterinary Committee.

Respiratory System

The *upper respiratory disease complex* is commonly seen in foals and young horses, both overseas and in Australia. It is characterised by a transitory pyrexia and anorexia, slight swelling of the lymph nodes of the throat and serous nasal discharge, which becomes mucopurulent after a few days. These clinical signs may be produced by any of several groups of viruses (Studdert 1967), including equine herpesvirus type 1 (previously called equine influenza virus, then

equine rhinopneumonitis virus, Doll *et al* 1957), by two serotypes of equine rhinoviruses and also by parainfluenza 3 virus and equine influenza A virus, of which antigenically separate sub-types 1 and 2 are known to infect horses independently. A coronavirus, of similar morphology to avian infectious bronchitis virus, has also been isolated from a Thoroughbred with upper respiratory disease in Canada (Ditchfield 1969). The clinical differential diagnosis of equine upper respiratory disease has been outlined by both Blood (1967) and Lewis (1969), the latter also emphasising epidemiological differences for these viruses.

In Australia, extensive investigation of respiratory diseases seen in horses in Victoria and Queensland have shown that equine herpesvirus type 1 (rhinopneumonitis) is the most common aetiological agent (Duxbury and Oxe 1968; Bagust and Pascoe 1968, 1970). This virus is widespread in Australia, 55-85% of adult horses having detectable serum antibodies as a result of prior infection (Turner and Studdert 1970; Bagust *et al* 1972). Acute upper respiratory infection by equine herpesvirus type 1 is usually seen only in young horses in their first infection with this virus, and older horses with antibody show only mild clinical signs on re-infection, usually by respiratory droplet spread of infection from young horses.

Viruses other than equine herpesvirus type 1 have not been isolated from respiratory disease in horses in Australia, except for slowly cytopathic herpesviruses, which have been obtained from both clinically normal and diseased horses in Australia and overseas (Studdert *et al* 1970; Bagust 1971), but their relationship to equine disease is not known. Serological examinations made on Australian horses from 1958 to 1970 were negative for influenza A virus (M. F. Warburton 1971, personal communication), and it would appear that this virus is not a cause of equine respiratory disease in this country at present. Histopathological lesions suggestive of adenovirus infection have been reported in the lungs of Arab foals dying of pulmonary disease in New South Wales (Johnston and Hutchins 1967), and an equine adenovirus has been isolated in Queensland (Harden *et al* 1972). The disease significance of complement fixing anti-

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TABLE 1
Classification of Viruses Infecting Horses

Deoxyribonucleic Acid			Ribonucleic Acid		
Virus Grouping	Member Viruses	Present in Australia*	Virus Grouping	Member Viruses	Present in Australia Exotic Viruses
Adenovirus	Not yet definite	+	Arbovirus	Eastern, Western, Venezuelan encephalomyelitis Australian arboviruses	+ +
Herpesvirus	Herpesvirus type 1 (rhinopneumonitis)	+	Coronavirus	Not yet definite	NE
	Coital exanthema	+	Myxovirus	<i>Influenza A</i> <i>Parainfluenza 3</i>	- NE
	Slowly cytopathic (cytomegaloviruses)	+	Picornavirus	Rhinoviruses	NE
Papovavirus	Papillomatosis (Warts)	+	Reovirus	African horsesickness	+
Poxvirus	Horsepox	NE	Rhabdovirus	Rabies	+
				Vesicular stomatitis	+
			Unclassified	Equine arteritis	NE
Not included in above classification:			Equine cutaneous sarcoid		+
			Equine infectious anaemia		+

* Distribution. + Known. ± Suspected. - Probably absent. NE No evidence available.

bodies to the *Chlamydia* (psittacosis) group of organisms detected in the serums of Australian horses by Studdert (1969) is not clear.

Central Nervous System

At least 11 viruses, mainly arthropod-borne, infect the equine central nervous system (Scott 1971) and include the classic encephalomyelitides produced by Western, Eastern, Venezuelan and Japanese B encephalitis viruses. Equine herpesvirus type 1 has also been associated with lumbar paralysis in stallions (Saxegaard 1966). In Australia, virus infection of the equine nervous system has not yet been reported, although Murray Valley encephalitis virus has been suspected to infect horses (Hungerford 1962), and antibodies to this arbovirus have been detected in the serums of horses in South Australia, Western Australia and Queensland (Spradbrow 1966).

Genital System

Equine coital exanthema, long recognised in Europe and the USA (Lieux 1963), is manifested by the formation of papules, pustules and ulcers on the genitalia of horses. In several countries, including Australia, this disease is considered to be caused by a herpesvirus (Pascoe *et al* 1968, 1969), which may be aided by trauma of the genital mucosa at coitus and the presence of pyogenic bacteria on the mucosal surfaces (Pascoe *et al* 1972). Turner *et al* (1970) have reported that an Australian strain of equine herpes-

virus type 1 can also produce a vulvo-vaginitis after experimental intra-vestibular infection.

Equine herpesvirus type 1 is known to be a cause of both sporadic and widespread *equine abortion* in many countries, but coital exanthema herpesvirus is not abortigenic (Bryans 1968). Foetuses are aborted by equine herpesvirus type 1 from 5 - 9 months of gestation, and gross and histopathological lesions on autopsy are characteristic (Doll 1963). Although infection with this virus is widespread in Australia, the virus has not yet been isolated from aborted foals in this country. Turner *et al* (1970) have shown that a strain of equine herpesvirus type 1 isolated from respiratory disease of horses in Australia was abortigenic when inoculated directly into the gravid uterus. A picornavirus has been isolated from an aborted equine foetus in Germany (Bohm 1964), but the significance of this isolation is still not known. The role of natural predators in hindering disease investigations of aborted foetuses in Australia (Bain 1969), as well as the heat-lability of viruses, should not be overlooked.

Skin and Appendages

Virus diseases of the skin of horses in Australia have not been investigated in detail, but there is ample clinical evidence for the occurrence of *equine cutaneous papillomatosis* (warts), caused by a host-specific papovavirus and appearing approximately 2 - 3 months after infection. *Papular dermatitis*, which may be viral in aetiology, has been reported to occur annually in

yearlings in training in the Sydney area as firm multiple papular swellings, which form scabs and recede after 2-3 weeks (Hutchins 1960). *Greasy heel* conditions and the transplantable cutaneous fibrosarcoma of *equine sarcoid* may also be of viral aetiology. *Horsepox virus*, which produces nodules, vesicles, then pustules and scabs on the limbs, lips and buccal mucosa, has been reported in Europe (Blood and Henderson 1968), but not in Australia.

Alimentary System

With the exception of a *chronic diarrhoea*, which may be of viral aetiology (Rooney 1969), virus infections directly involving the equine alimentary system are not common. However, a secondary effect of many virus infections is to cause functional disorders of this system.

Generalised Infections

Equine infectious anaemia is a virus infection characterised by an acute initial attack, then a long chronic illness, and was diagnosed as present in Australia by Brooks *et al* (1959) and Winter (1960). Invasion by this virus causes damage to the vascular system, particularly the intima of small blood vessels, and excessive destruction of erythrocytes. Inflammatory changes are produced in the parenchymatous organs, especially in the liver, and mares may also abort. Infected horses should be destroyed, because present therapy is ineffective and transfer to uninfected horses may occur by contaminated discharges or by a common syringe needle.

Equine viral arteritis has not been detected in Australian horses, but the disease is important in that clinical signs of acute infection (fever, depression, ocular and nasal discharges, oedema of the eyelids, limbs and abdomen, coughing and difficulty in breathing, colic, enteritis, jaundice, abortion) could be confused with infection by several viruses previously discussed. In the USA, equine viral arteritis is also seen as abortion in mares following a mild non-specific febrile illness (Rooney 1968). The pathognomonic lesion of equine arteritis virus infection is degeneration and necrosis of the media of small arterioles throughout the body. Outbreaks of this virus infection have been reported to occur only sporadically, virus being spread in the discharges of infected animals. The virus has been isolated only in the USA and Switzerland to date.

Diagnosis

Virological diagnosis is made on the isolation of viruses from infected animals and on serological evidence of infection, and these two

methods are best used in conjunction where possible. Freshly-collected swabs or washings for virus isolation should be stored in a transport medium (Burrows 1968), which will protect the virus from heat-inactivation, and transported to the laboratory as quickly as possible on wet ice. Viruses are grown in the laboratory in susceptible cell cultures, and isolates are identified by pathological effects on cell monolayers, by specific tests, and by serological relationships with antisera to known groups of viruses. Direct visualisation of virus particles by the electron microscope (Spradbrow and Francis 1969) and fluorescent labelling of antibodies to specific viruses can allow the more rapid identification of viruses.

Serological demonstration of the absence of serum neutralising or complement fixing antibody to a viral agent in a serum sample collected from a horse at the time of acute clinical disease, and detection of antibody in another serum collected 2-3 weeks later strongly indicates a relationship between the virus infection and disease. Serological studies of the age incidence and spread (the epizootiology) of a virus may also be of value in interpreting the extent of disease caused by a virus in a horse population.

Treatment and Control

Viral infections of horses may be complicated by bacterial diseases if an inadequate period is allowed for convalescence, or if horses are stressed and fatigued when infected. Specific antiviral chemotherapeutic agents are not yet available for use in equine virus infections, but antibiotic therapy may aid healing of the lesions by controlling secondary bacterial infection, such as therapy suggested by Pascoe (1969) for equine herpesvirus type 1 infection of horses occurring under Australian conditions.

Apart from the classical control measures of segregation of affected animals and disinfection (Benyon and Miller 1963), prophylactic vaccination where merited is the main practical means of control. Satisfactory vaccines are not available for several equine viruses (Scott 1971), but efficient vaccines containing live attenuated viruses are in use overseas for viruses spread by contagion, such as equine herpesvirus type 1 and equine influenza A viruses. A vaccine of cell culture origin for equine arteritis virus has been favourably recommended (Anon 1969). Inactivated vaccines for Eastern and Western equine encephalomyelitis are in use in the USA (Anon 1969), and recent Japanese work has indicated the possibility of immunising horses against equine infectious anaemia using a live attenuated virus (Kono *et al* 1970).

Vaccines for virus infections of horses are not currently used in Australia, there being little justification for their use on present evidence of disease losses to the horse breeding industry. The future accidental introduction and establishment of equine influenza A virus in the Australian horse population would probably result in an explosive spread of infection (Lewis 1969), and necessitate the use of prophylactic vaccination in this country. In the event of an introduction of a pathogenic equine virus at present exotic to Australia, slaughter of affected horses and "ring" vaccination of horses with inactivated vaccines would probably be necessary control measures.

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BOOK REVIEW

THE JOURNAL OF THE ROYAL ARMY VETERINARY CORPS

Vale — with Volume 42, Spring 1971, the *Journal of the Royal Army Veterinary Corps* comes to an end, victim of rising costs. Its passing is much regretted, but the RAVC is still active and is capable of adding to the sum of veterinary knowledge, and in the final issue of the *Journal* the Colonel Commandant exhorts members of the Corps to continue to publish the results of their observations and experiments — in other journals. The Director regrets the death of the *Journal* but says, "Death, we must remember with Bacon, although very sad, opens the gate to great fame and extinguishes envy".

The final issue has papers on Sensitivity of lymphocyte chromosomes irradiated *in vitro* and *in vivo*, Observations on gastric activity in adult Alsatian dogs,

Transport of mules by air from Cyprus to Hong Kong, Management Colic (a case), Small animal "Immobilon" and "Revivon". A specially interesting article entitled *Hearts and Minds in the Sand* gives an interesting commentary on the Dhofar province of the Sultanate of Oman and the activities of the Veterinary Officer — including his embarrassment on being presented with a woman for wife in return for treating some sick camels, and further when "four women were led forward who 'wished to carry the Tabib al Haiwan's (vet's.) chicks in their tummy'".

There is news from many units in Great Britain and overseas, poems on boar hunting, some reminiscences and a final editorial. Vale!

Hugh McL. Gordon