Veterinary Microbiology 167 (2013) 205–214

Contents lists available at SciVerse ScienceDirect

Veterinary Microbiology

journal homepage: www.elsevier.com/locate/vetmic

Review

Equine influenza—A global perspective

A. Cullinane [a,*], J.R. Newton [b]

[a] Virology Unit, The Irish Equine Centre, Johnstown, Naas, Co. Kildare, Ireland
[b] Animal Health Trust, Centre for Preventive Medicine, Lanwades Park, Kentford, UK

ARTICLE INFO

Article history:
Received 5 February 2013
Received in revised form 25 March 2013
Accepted 28 March 2013

Keywords:
Equine influenza
Epidemiology
Laboratory diagnosis
Management
Vaccination

ABSTRACT

To date, equine influenza outbreaks have been reported all over the world with the exception of a small number of island nations including New Zealand and Iceland. Influenza is endemic in Europe and North America and is considered to be of potentially major economic significance to the equine industry worldwide. The importation of subclinically infected vaccinated horses, and inadequate quarantine procedures have resulted in several major outbreaks in susceptible populations for example, in Australia (2007) when more than 76,000 horses on over 10,000 properties were reported as infected. This review summarises the current understanding of, and recent research on, equine influenza, including epidemiology, pathogenesis, clinical characteristics, laboratory diagnosis, management and prevention. Recent advances in diagnostic techniques are discussed as are the merits of different vaccination regimes.

Contents

1. Introduction

Equine influenza viruses are minus-strand RNA viruses of the *Orthomyxoviridae* family. Containing eight RNA segments (the genes which encode 11 distinct proteins are described as segments 1–8), they are categorised as type A influenza viruses based on the antigenicity of the nucleocapsid (NP) and matrix (M) proteins. They are further classified on the basis of the composition of the surface glycoproteins haemagglutinin (HA) and neuraminidase (NA). These two proteins make up approximately 45% of the total mass of the virus particle and are major targets for the development of vaccines and antiviral drugs. The primary natural reservoir of influenza A viruses are aquatic birds and equine influenza viruses are considered to be of avian origin. Like avian influenza viruses, equine influenza viruses have an affinity for sialic acid α2-3 galactose containing receptors on cell surfaces.

* Corresponding author. Tel.: +353 45 866266; fax: +353 45 866273.
E-mail address: acullinane@equine-centre.ie (A. Cullinane).

0378-1135/$ – see front matter © 2013 Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.vetmic.2013.03.029

Many different combinations of 16 of the 17 known HA subtypes and 9 known NA subtypes occur in birds and although avian H5N1 has been associated with respiratory disease in donkeys in Egypt (Abdel-Moneim et al., 2010) only two stable subtypes have so far been reported in horses, H7N7 and H3N8. The H7N7 viruses co-circulated with H3N8 viruses in horses for many years, but the former viruses have not been isolated for over two decades and are considered to be extinct (Webster, 1993). Since 1979, all outbreaks for which there are virus isolates for study, have been attributed to H3N8 viruses. Influenza viruses are codified according to their type, host species, subtype and place and year of isolation for example, A/equine 2/Sydney 2007 (H3N8).

Although equine influenza viruses are more stable than human influenza viruses they also undergo antigenic drift, i.e. point mutations occur in the HA and NA genes that result in amino acid sequence changes in the protein. These mutations can cause significant changes in the antigenicity of the virus. As a result of antigenic drift the H3N8 subtype evolved in the late 1980s into two distinct lineages designated the "American-like" lineage and the "European-like" lineage based on the initial geographical distribution of viruses (Daly et al., 1996). Three American sub-lineages subsequently emerged (Lai et al., 2001) of which the Florida sub-lineage is now predominant. As illustrated in Fig. 1 this sub-lineage has itself diverged into two antigenically different clades (known as clade 1 and clade 2). Clade 1 viruses predominate on the American continent but have caused large outbreaks of disease in Africa, Asia and Australia (King and Macdonald, 2004; Yamanaka et al., 2008a; Watson et al., 2011). They have also spread to Europe (Bryant et al., 2009; Bryant et al., 2011; Gildea et al., 2012). Similarly, Clade 2 viruses predominate in Europe but have caused large outbreaks in Asia (Qi et al., 2010; Virmani et al., 2010; Yondon et al., 2013)

Ecological studies have revealed that avian influenza viruses persist in lake water and can overwinter in the ice of northern nesting lakes. Experimental studies indicated equine influenza viruses can survive in tap water for 14 days at 4 °C, for 2 days at 24 °C and 37 °C and in canal water for 18 days at 22 °C and 14 days at 37 °C (Yadav et al., 1993). However, equine influenza viruses are very labile and show very little resistance to ultraviolet light, detergents and disinfectants customarily used for viral infections (Yadav et al., 1993).

2. Epidemiology

To date, equine influenza outbreaks have been reported all over the world with the exception of a small number of island nations including New Zealand and Iceland. Influenza is endemic in Europe and North America and is considered to be of potentially major economic significance to the equine industry worldwide. The importation of subclinically infected vaccinated horses, and inadequate quarantine procedures have resulted in several major outbreaks in susceptible populations for example, South Africa (1986 and 2003), India (1987), Hong Kong (1992) and Australia (2007). The virus is highly contagious and is primarily spread by the respiratory route through direct contact between infectious and susceptible horses in close proximity. Personnel and fomites also contribute to virus spread. In the absence of release of horses from the quarantine station at Eastern Creek in Sydney, New South Wales, Australia in 2007, it was concluded that the virus escaped on the person, clothing or equipment of a groom, veterinarian, farrier or someone else who had contact with the infected horses and left the station without implementing adequate biosecurity measures[1]. In South Africa in 1986 and 2003 contaminated vehicles were implicated in the spread of the virus (Guthrie et al., 1999; King and Macdonald, 2004).

In unvaccinated, susceptible horses, the short incubation period (see below) and persistent coughing which releases large amounts of virus into the environment contribute to the rapid spread of the infection. In a susceptible group of horses, morbidity can be as high as 100%. Horses stabled under intensive conditions are at risk from a build-up of infective virus in the common airspace. The number of clinical cases in this type of environment has been reported to peak within 8 days of the index case (Powell et al., 1995). Dispersal of horses after horse shows, sales, race meetings and other events where equine influenza virus has been circulating may lead to widespread dissemination of virus to the wider equine population. This occurred in the UK in 1979 and in Ireland in 1989 when outbreaks appeared to originate at the Olympia and Royal Dublin Horse Show respectively (Burrows et al., 1981; van Maanen and Cullinane, 2002). In the first outbreak of equine influenza in Australia in 2007, the initial spread of the virus in the general horse population was linked to a "one-day event" at Maitland, New South Wales in mid-August. By December, when the last case was reported, more than 76,000 horses on over 10,000 properties were reported as infected (Cowled et al., 2009).

Mortality is very rarely associated with equine influenza but a small number of fatalities have been reported in young foals that have not acquired maternal antibodies (Miller, 1965a,b; Patterson-Kane et al., 2008), and in affected donkeys and horses that are not adequately rested (Powell et al., 1995). In northeastern China in 1989, a mortality rate of up to 20% in some herds was associated with a large outbreak of equine influenza. This outbreak was caused by an H3N8 virus (A/equine 2/Jilin 89) that genetic analysis indicated was more closely related to avian influenza viruses than to other equine H3N8 influenza viruses (Webster and Guo, 1991; Guo et al., 1992). Fortunately, this avian virus did not appear to persist in the horse population after 1990 or to spread beyond China to other countries. When it emerged in horses it was no longer infectious to ducks, which reduced the possibility of spread by aquatic birds through migration.

Although equine influenza virus spread is frequently explosive in naïve populations the majority of outbreaks in endemic populations are contained with limited spread

[1] http://www.equineinfluenzainquiry.gov.au.