Equine Influenza Virus

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KEYWORDS

• Influenza A virus • Virus evolution • Epidemiology • Diagnosis • Control

KEY POINTS

- Despite extensive use of vaccines, equine influenza virus continues to be one of the most important equine viral respiratory pathogens.
- Control of equine influenza virus is hampered by continued genetic evolution of the virus (antigenic drift).
- Antigenic drift negatively affects the degree of immunoprotection evoked by inactivated vaccines.
- Isolation and genetic characterization of currently circulating equine influenza viruses remains a priority, and is essential for vaccine strain selection.

INTRODUCTION

Influenza is a well-known and ancient disease. In fact, a disease resembling influenza was described by Hippocrates in 412 BC. Yet hardly a month goes by without a new headline about influenza in the media. Although millions of dollars have been spent on research, influenza virus continues to challenge our understanding of its ecology and our ability to control its spread. Two key reasons why influenza virus has remained one of the most important causes of viral respiratory disease are its potential for establishing genetic and antigenic diversity and its ability to occasionally transmit between different host species.[1]

For decades the horse has been viewed as an isolated or "dead-end" host for influenza A viruses, with equine influenza virus being considered as relatively stable genetically. Although equine influenza viruses are genetically more stable than their human-lineage counterparts, they are by no means in evolutionary stasis. Moreover, recent transmission of equine-lineage influenza viruses to dogs also challenges the horse's status as a dead-end host. This article reviews recent developments in the epidemiology and evolution of equine influenza virus. In addition, the clinical presentation of equine influenza infection, diagnostic techniques, and vaccine recommendations are briefly summarized.

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ETIOLOGY

Equine influenza virus is a member of the influenza A viruses, which belong to the Orthomyxoviridae family. The Orthomyxoviridae family comprises 5 genera (Fig. 1), all containing enveloped viruses with segmented, single-stranded, negative-sense RNA genomes. In contrast to the rather narrow host ranges of influenza B and C viruses, influenza A viruses can infect a wide variety of species (see Fig. 1).

Influenza A viruses possess a host-cell–derived lipid envelope, which contains 3 envelope glycoproteins: the hemagglutinin (HA), neuraminidase (NA), and M2 ion channel protein (Fig. 2). Both the HA and NA are major surface antigens of the virus, and antibodies to these proteins are associated with resistance to infection.

- Based on antigenic properties of the HA and the NA, influenza A viruses are divided into subtypes.
- Cross-protection between subtypes (heterotypic immunity) is weak.
- Eighteen HA subtypes (H1–H18) and 11 NA subtypes (N1–N11) have been found.
- Equine influenza has been caused by viruses of H7N7 (A/equine/1) and H3N8 (A/equine/2) subtypes.

In addition to the envelope glycoproteins, the influenza A virus genome encodes for an additional 5 structural (M1 matrix protein, nucleoprotein [NP], and polymerase complex [PA, PB1, PB2]) and 3 "nonstructural" proteins (NS1, NS2 [also referred to as the NEP "nuclear export protein"], and PB1-F2) (see Fig. 2).

During infection antibodies are also produced to the internal proteins, but these antibodies are not protective. Similarly, the cytotoxic T-cell (CTL) response, primarily

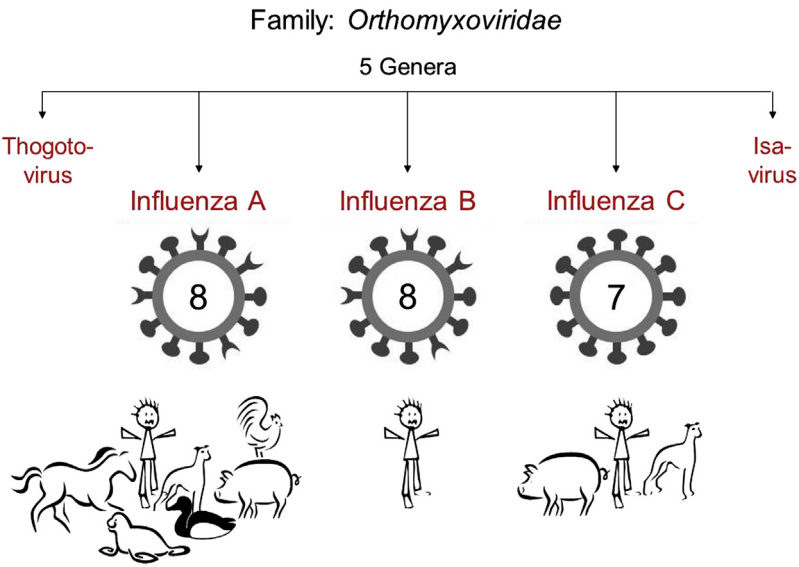

Fig. 1. The 5 Orthomyxoviridae genera. In contrast to the wide host range of influenza A viruses, influenza B and C viruses have a more narrow host range. Whereas influenza A and B contain 8 separate segments of single-stranded RNA, influenza C viruses possess only 7.