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Avian influenza II

Low pathogenicity AI (LPAI)

LPAI outbreaks have been the cause of significant economic losses in poultry and this is especially the case when such outbreaks are accompanied by secondary viral, bacterial or mycoplasmal infections.

Zoonotic implications

With influenza viruses occasional interspecies transmission occurs but this very rarely involves the transmission of avian influenza viruses or their genes to man.

Less than a dozen incidents of limited natural infection in man with avian influenza virus are known and these have nearly always involved direct contact with sick birds (veterinarian, depopulation teams or living with village chickens). In one interesting case the owner of an infected fighting cockerel became infected after sucking mucus out of the infected bird's nostrils. Typically the infected human shows mild flu- like symptoms characterised by conjunctivitis and respiratory signs.

Aetiology

Avian influenza viruses are orthomyxoviruses. Their surfaces are covered by two different glycoprotein projections known as haemagglutinins and neuramidases. These can be of differing types and this is used to define serotypes when the haemagglutinins and neuramidases are respectively referred to as 'H' and 'N' and their different types designated by numbers, for example, H5N1.

Infection and replication

The avian influenza virus adsorbs through its haemagglutinin on to host cell receptors and this initiates a receptor mediated endocytosis which takes the virus into the cell. Viral replication then occurs. The resulting viral material then develops a close association with the avian cell's plasma membrane resulting in the budding (production) of the avian influenza viruses.

Viral properties

Avian influenza viruses are relatively unstable and factors such as heat, extremes of pH, hypertonicity and dryness can all inactivate avian influenza viruses. They are inactivated by organic solvents, detergents, β-propriolactone, aldehydes, phenolic and quaternary ammonium disinfectants, oxidising agents and weak acids.

Influenza viruses are protected by organic material such as faeces and nasal discharges and cool, damp conditions favour virus survival, while hot, dry conditions do not. Avian influenza viruses can survive in faeces for seven days at 20°C or 35 days at 4°C. Recent studies with H5N1 avian influenza

virus showed that it was capable of surviving for four days in chicken faeces stored at 25-32°C in the shade.

Effective disinfectants in the field with minimal organic matter present include 5.25% sodium hypochlorite, 2.0% sodium hydroxide, phenols, chlorine dioxide, strong oxidising agents and 4.0% sodium carbonate.

Cooking and pasteurisation are effective ways of inactivating avian influenza viruses.

Classification of avian influenza viruses

These belong to the influenza A group and are subtyped on the basis of their haemagglutinin (H) and neuraminidase (N) surface glycoproteins. There are 16 H subtypes and nine N subtypes and most combinations of these have been reported at one time or another.

A standardised approach is used for subtyping avian influenza viruses from man, poultry and pigs.

Immunity

The H antigen elicits antibodies that protect birds against death and clinical signs. Protection is H subtype specific and can last six months or more. Protection can be elicited by the N antigens and is again subtype specific but is not as good as the H related protection.

Viral reassortment

Full length genome sequencing of avian influenza viruses is now possible and as genetic reassortment between influenza viruses can occur this technology enables us to define the origins or 'parentage' of a specific virus isolate. Some influenza viruses have been shown to contain genetic material from two, or even all, avian, porcine and human avian influenza viruses.

Antigenic drift or shift

Influenza viruses can modify their antigenic make up so, over a period of time, change can occur. This is known as antigenic drift. In vaccinated poultry immune pressure can play a role in selecting antigenic variants. However, in areas of endemic LPAI, such as H9N2 in the Middle East, where vaccination is widespread, it is not clear whether such pressure comes from circulating field virus or vaccinal virus.

In antigenic shift two different influenza viruses infect a cell and each can acquire new H and N antigens from the other, resulting in the creation of new viruses.