AVIAN INFLUENZA IN OSTRICHES (*struthio camelus*): A REVIEW

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**Introduction**

Ostrich is increasingly becoming an important commercial farming bird throughout the world. The resent boom in ostrich farming has been influenced, not only by the market of the historical product, the feathers, but also by the high quality hide (leather), healthy low cholesterol-red meat, eggs and egg-shells, and fat. Probably the use of ostrich eyes and tendons might be useful in human surgery. The ostrich is also an excellent zoo bird because of its size, plumage and response to visitors.

Apart from all these features that hint good future for the ostrich industry, consumption and export of ostrich products has in the past, been affected by outbreaks of avian influenza in ostrich farms in Italy (2000) and in South Africa (2004). In Saudi Arabia in 2007, thousands of ostriches were destroyed after an outbreak of AI in ostrich and poultry farms and some outlets selling ostrich products were closed-down. The occurrence of avian influenza in poultry in Asia in which a number of human cases were reported has further scared some consumers off ostrich products especially the meat.

**The disease**

Avian influenza (AI) is a highly contagious viral infection of birds. AI is caused by various strains of influenza virus. Many of the strains that circulate in the wild birds are either non-pathogenic or mildly pathogenic for poultry. However, a virulent strain may emerge either by genetic mutation or by reassortment of less virulent strains. Once introduced in a flock of domestic poultry, AI is highly contagious, the virus is shed in faeces, nasal and ocular discharges. The virus is then spread by the movement of birds, equipments, vehicles and attendants (FAO, 2005).

**Host-range**

The AI viruses are widespread in free-living and migratory birds which serve as reservoirs for infection to poultry. A wide range of bird species including the ostriches, are susceptible to AI infection. The domestic fowl, ducks, geese, turkeys, guinea fowl, quail and pheasants are susceptible. Many species of wild birds particularly water birds and seabirds are also susceptible, but infections in these birds are generally subclinical.
The Aetiology

The influenza virus belongs to a family known as Orthomyxoviridae. The influenza viruses that constitute this family are classified into types A, B or C basing on the antigens on their nucleoprotein and matrix protein. Avian influenza viruses belong to type A. The influenza viruses are further categorized into subtypes according to the antigens of the haemagglutinin (H) and neuraminidase (N) projections on their surfaces. There are 14 haemagglutinin subtypes and 9 neuraminidase subtypes of influenza-A viruses. The AI viruses have representatives in all of these subtypes, but the highly pathogenic AI viruses belong to H5 and H7 subtypes. However, not all H5 and H7 subtype viruses are virulent to poultry. The pathogenicity is correlated to the ability of trypsin to cleave the haemagglutinin molecule into two subunits. The highly pathogenic strains of H5 and H7 viruses have several amino acid residues at the cleavage site. Trypsin sensitivity and amino acid sequencing can be used diagnostically to determine whether or not an isolate is potentially pathogenic.

A particular isolate may produce severe disease in one species but not in other species. For this reason it is impossible to generalize on the host range for the highly pathogenic avian influenza (HPAI). The HPAI is likely to vary with the isolate. For instance, the classic fowl plaque virus is H7N7, but in the major epidemics reported in Eastern USA in poultry in 1983 and 84-87 the isolate was a H5N2 subtype. In one of the most serious epidemics in recent times in Hong Kong (1997-98, 2003), H5N1 was isolated in poultry and was also associated with human infections and deaths. H5N1 subtype was also isolated in poultry in South Korea in 2003 (FAO, 2005). From 2003 to 2007 serious outbreaks of H5N1 AI subtype occurred worldwide, from Indonesia, Vietnam and China across Middle East, including Saudi Arabia and Kuwait through Turkey to Europe. The most affected areas being Asian where millions of poultry were culled-off. According to WHO and FAO human deaths amounted to 208, the most hit country being Indonesia.

History of Outbreaks in Ostriches and Other Ratites

In South Africa H7N1 subtype (1991-92) and H5N9 subtype (1994) were isolated during the outbreaks of the disease in ostriches (Allwright et al., 1993; Anon, 1994). H5N2 subtype was isolated in ostriches during an outbreak of avian influenza in South Africa in 2004 and thousands of ostriches were culled-off to contain the diseases (CIDRAP, 2004).

Low pathogenic avian influenza (LPAI) subtypes H9N2 (1995), H8N8 (1998) and H10N1 (2001) were isolated in ostriches in South Africa. In the Netherlands in 1994, H5N9 subtype was isolated in emus and cassowary, these birds were healthy, but when tested on SPF chickens, severe mortality occurred (Marvel et al., 1996).

In Zimbabwe in 1995, H5N2 subtype was isolated in ostriches; none of the isolates H7N1, H8N9 and H5N2 in South Africa and Zimbabwe was pathogenic to poultry (Marvel et al., 1996).

Apathogenic H5N2 subtype virus was isolated in ostriches under quarantine in Denmark (Jorgensen et al., 1998). H7N1 and H5N2 subtypes virus were isolated in emus and rheas
in Texas, USA (Panigrahy et al., 1995). During an outbreak of avian influenza in ostriches in Italy, H_7N_1 subtype virus was isolated (Capua et al., 2000).

In Saudi Arabia, 14 separate outbreaks of H_3N_1 AI occurred between 2003 and 2007. Affected birds included falcons, peacocks, turkeys, parrots, chicken and ostriches. In one outbreak about 13,500 ostriches and about 4 million poultry were culled-off to contain the disease (SPA, 2007).

**The Role of Wild-bird Population in Epidemiology of AI in ostriches**

When the H_6N_8 AI subtype was isolated in ostriches in South Africa (1998), the same subtype was isolated in Egyptian goose (*Alopochen aegyptiacus*) at the same locality. H_5N_2 AI subtype was isolated in Egyptian goose (*Alopochen aegyptiacus*), two weeks prior to the outbreak of AI disease in ostriches in 2004 (Sinclair et al., 2006). The most recent outbreaks suggest that wild and migratory birds may have played a role in transmitting the disease between countries and regions. However, in the majority of cases the movement of domestic poultry and/or products have been largely implicated in the spread of the disease in the South East Asia (ADU, 2007; Khawaja et al., 2005). Influenza viruses have been shown to affect all types of domestic or captive birds around the world, but the frequency with which the primary infections occur in any type of bird depends on the degree of contact with feral birds. Secondary spread is usually associated with human involvement, probably by transferring infective faeces from infected to susceptible birds (Khawaja et al., 2005).

**Disease Presentation in Ostriches**

Clinical signs, severity and lesions that have been reported in ostriches are very variable and have been shown to be influenced by a number of factors. The factors that influence the disease presentation include age, concurrent infection especially with *E. coli*, *Pseudomonas aeruginosa*, *Aspergillus fumigatus* and *Staphylococcus aureus*.

Clinical signs include depression, respiratory distress, ocular discharges and green discoloration of urine. Other signs are ruffled feathers, and complications arising from secondary infections. Younger birds are more severely affected than mature ones. Mortality rate is also variable, in young ostriches it reaches up 60-80% and acute mortality may be first indication of presence of disease in a flock (Shane and Tully, 1996; Huchzermeyer, 1998).

Pathological findings in ostriches that have died of AI infection include enlarged, mottled and friable liver, coagulative necrosis of the liver surrounded by marked heterophil-infiltration often in proximity of blood vessels which show variable degrees of vasculitis; congested proximal intestines filled with mucoid contents, severe congestion and necrosis of the tips of the villi; necrosis of the spleen and the pancreas (Huchzermeyer, 1998). Nephrosis and fibrinous airsacculitis have been reported in advanced cases (Shane and Tully, 1996). A number of cases of AI in ostriches have been reported with considerable variations in clinical signs and pathological findings.
Allwright et al. (1993) reported influenza-A virus of the H7N1 subtype isolated from dead young ostriches. The flock showed variable clinical signs including green discoloration of urine, weakness, respiratory distress and variable mortality according to age, concurrent infections and other stress factors. Using haemagglutination-Inhibition test (HIT), anamnestic response was recorded in ostriches that recovered. Pathogenicity test revealed that the virus was of low virulence to domestic fowls.

Jorgensen et al. (1998) reported isolation of influenza-A virus, subtype H5N2 from a flock of ostriches under quarantine in Denmark. More than 28% of the ostriches died within the first three weeks, majority of the dead were young under 10kg bodyweight. Intravenous pathogenicity index (IVPI) of each isolate for chicken was 0.00. Other findings were isolation of avian paramyxovirus type-1 (APV-1) Pathological findings were impaction, enteritis and stasis and multifocal necrotic hepatitis.

An outbreak of a highly pathogenic avian influenza infection in ostriches farmed in Italy was reported by Capua et al. (2000). The virus isolated was of the subtype H7N1. More than 40% of young birds died. Clinical signs included sudden deaths, incoordination, complete loss of appetite, severe depression 48hours prior to death and brilliant green urine which often contained urates. Faeces were hemorrhagic. There were no clinical signs or mortality among the breeders.

**Diagnosis of AI in Ostriches**

Diagnosis of AI includes the clinical signs of sudden death and green discoloration of urine; differential diagnosis should be considered. Sample collection and laboratory procedures confirm the diagnosis.

Bacteriological tracheal and cloacal swabs are collected aseptically. The material collected on the swabs should be mixed into 3ml aliquots of transport medium in sterile bottles and swabs discarded. The transport medium may be sterile brain-heart infusion containing 5000ug of streptomycin per ml, or equal parts of glycerol and phosphate-buffered saline with the same antibiotics added.

Specific-pathogen-free chicken eggs are inoculated on the 10th day of incubation via chorioallantoic route. Viral isolates causing embryo death and haemagglutination of avian erythrocytes are subtyped using a panel of antisera. Serum of live birds can be screened for influenza antibodies using agar gel diffusion procedure (FAO, 2005; Shane and Tully, 1996).

**Differential Diagnosis of AI infection in Ostriches**

Differential diagnosis of AI infection includes Newcastle disease, infectious bronchitis, acute poisoning and acute Pasteurellosis.

**Vaccination of Ostriches against AI**

An inactivated emulsified AI vaccine was prepared and tested widely in the field in South Africa. It is reported to have protected against morbidity and mortality, but it did not however, prevent the shedding of virus (Huchzermeyer, 1998) Inactivated vaccines
may also promote selection of more pathogenic variants (Shane and Tully, 1996). All avian influenza viruses have the ability and tendency to mutate and recombine (Huchzermeyer, 1998). Due to the existence of a large number of virus subtypes together with the known variation of stains within a subtype, vaccination cannot be used as a routine tool for the disease control. And also there is no cross protection among the 15 H-subtypes (FAO, 2005). Management of mild cases includes supportive therapy comprising fluids and antibiotics against secondary bacterial infections. However, vaccination and treatment are not advised due to the fact that the world market for the ostrich products restricts import from affected areas. Instead total culling of affected flock is currently practised.

**Preventive Measures**

The most important preventive measures against introduction of AI in ostrich production establishments are elimination or at least reduction of contact between ostriches and the wild birds that are attracted to their feed; and strict biosecurity which involves control of human movements, use of protective clothing and shoes for the staff and no-visitors policy. Other measures include quarantine and testing of all new birds to be introduced to the farm, control of movement and disinfection of equipments and vehicles and strict exclusion of poultry and pet birds (Shane and Tully, 1996; Huchzermeyer, 1998; FAO, 2005). Control of other viral and bacterial diseases is important as they may confuse and complicate the AI presentation.

**Zoonosis risk of avian influenza**

In the most serious epidemics in recent times in Asia (1997-98, 2003-2007), AI virus subtype H5N1 was isolated in poultry and was also associated with human infections and deaths (FAO, 2005; SPA, 2007). According to FAO and WHO, from 2003 to 2007, 205 human deaths have been reported worldwide due to infection by AI virus of the subtype H5N1. Most of the deaths occurred in Asia mainly in Indonesia, Vietnam, Hong Kong (SPA, 2007).

All avian influenza viruses have the ability and tendency to mutate and recombine; therefore, there are possibilities that outbreaks such as those reported in humans around the world could erupt again from any outbreak of AI in poultry.

**Recommendations**

- Based on the significance of wild birds as carrier of AIVs it is recommended to study, keep records and follow the migratory patterns of the wild birds.
- It is also recommended to seromonitor wild birds on regular basis so that the status of AIVs in specific localities can be evaluated and so help in developing timely emergency measures.

**References**
http://www.uct.ac.za/depts/stats/adu/avianflu.htm


