

Ways to control avian meta pneumovirus

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The avian meta pneumovirus (aMPV) replicates in the respiratory tract and the reproductive tract. It initially causes respiratory disease in infected birds and may also cause drops in egg production in layers and breeders.

Infection in turkeys is commonly referred to as 'turkey rhinotracheitis' and in chickens aMPV infection is commonly associated with the condition known as 'swollen head syndrome' (SHS).

Turkey rhinotracheitis, a disease with up to 100% morbidity occurring in young poults, was first recognised in South Africa in 1978 by Buys and Du Preez. Its viral aetiology was determined subsequently.

In the mid-1980s a pneumovirus-like agent, called turkey rhinotracheitis virus (TRTV), was shown to be the causative agent of turkey rhinotracheitis in the UK and France.

Shortly after the discovery of TRTV in turkeys, the same virus was also found in chickens, where it is one of the agents associated with the so-called 'swollen head syndrome' and drops in production in egg

producing birds. A more appropriate name for the virus, therefore, seems to be avian rhinotracheitis virus (ARTV).

In scientific articles the term avian meta pneumovirus (aMPV) is preferred.

Avian meta pneumovirus principally causes disease in turkeys and chickens. Pheasants and guinea fowl are also susceptible and antibodies to the virus have also been demonstrated in wild birds and other domestic species.

Antigenic diversity

ELISA and cross-neutralisation studies with polyclonal sera and monoclonal antibodies showed that although aMPV strains isolated in different areas of Europe are antigenically related and belong to one serotype, at least two antigenic subtypes (A and B) can be distinguished.

In continental Europe mostly subgroup B strains have been found. In the UK subgroup A was the single type of aMPV found in the 1980s, but more recently subgroup B has become predominant in the UK as well.

For a long time aMPV was thought to be absent in the USA. Recently, however, aMPV was isolated in the USA. The virus is

antigenically quite different from the European A and B types of aMPV and is classified as type C.

Preliminary evidence seems to indicate that a fourth type of aMPV (called type D) exists in ducks in France.

Despite the differences found between subgroup A and B strains, cross-protection in both directions has been demonstrated.

Preinfection with subgroup A (vaccine or wild type virus) protects against clinical disease caused by aMPV of subgroup B and preinfection with subgroup B protects against challenge with subgroup A virus.

Intervet's subgroup A vaccine (Nobilis TRT/ Nobilis RTV 8544) and Intervet's subgroup B vaccine (Nobilis RTCV 1194) were shown to provide full protection against challenge with the US isolate.

Likewise, subgroup A and subgroup B vaccines seem to protect against the non-A and non-B strains of APV found in France.

Epidemiology

A serological survey has shown the presence of aMPV in most, if not all, European countries, Asia (including Japan), Africa, South- and Central America and, more recently, the USA. In many cases the virus has been isolated as well.

The disease is spread by direct and indirect contact. Egg transmission has not been detected.

Disease in chickens

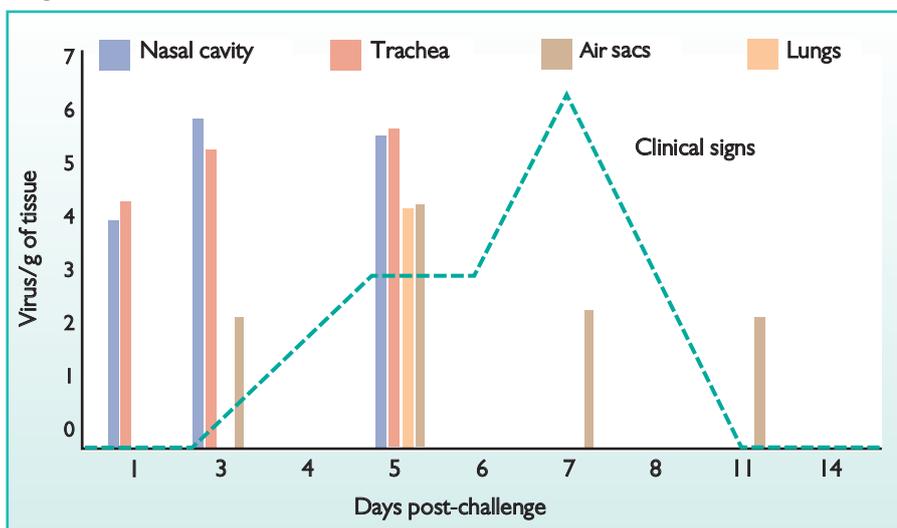
The virus appears to be capable of infecting chickens of all ages. However, clinical signs in broilers have been observed mostly between four and six weeks of age.

The disease lasts for 2-3 weeks with a mortality rate of 1-5%. If complications occur (secondary infections) the mortality rate can be as high as 20-30%. Clinical signs seen are depression, decrease in food intake, nasal exudate, sneezing and coughing.

In a small proportion of cases this may progress into conjunctivitis, followed by facial oedema starting around the eye,

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Fig. 1. Avian meta pneumovirus can be isolated for only a short period of time after infection. Once flocks are showing clinical signs of infection the virus can often no longer be isolated.



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extending over the head and descending to the submandibular tissue, mostly accompanied with varying degrees of swelling of the infraorbital sinuses.

This 'swollen head syndrome' (SHS) develops long after the virus has disappeared and may be due to secondary infection effects. *E. coli* can be isolated rather consistently from many tissues of the affected birds.

Swollen head syndrome has never been reproduced experimentally, although combined infection of SPF chickens with aMPV and *E. coli* produced lesions corresponding to the initial stage of SHS.

It is generally accepted now that aMPV is

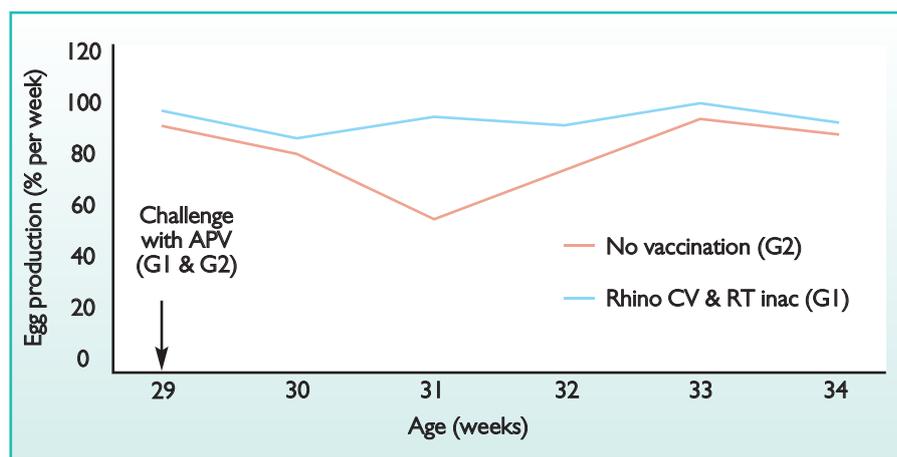


Fig. 2. A vaccination programme with live and inactivated APV vaccines protects against the negative effect on egg production caused by APV infections.

only one of the agents involved in SHS. In laying birds (broiler breeders as well as layers) aMPV primarily affects the birds at the peak of production or shortly before entering production.

The first signs of the disease are apathy, mild respiratory rales, sneezing, conjunctivitis and facial swelling. This may be followed by nervous signs like torticollis, opisthotonus and inability to move.

Morbidity ranges between 6 and 10%, mortality between 0 and 3%. The clinical signs are usually accompanied by a drop in egg production reaching 1-10% and lasting for 2-3 weeks.

Pathogenesis

Although dissemination studies in SPF chickens with both turkey-derived and chicken-derived aMPV were performed by several authors, the most extensive investigation of the pathogenesis of aMPV in chickens was recently published by Cook and co-workers.

In fact, this investigation was performed with the pathogenic subgroup B field isolate from which the Nobilis Rhino CV vaccine is derived.

Upon experimental oculonasal infection of young (SPF) chickens, the virus replicates to high titre in the respiratory tract, including the infra-orbital sinuses, for approximately five days. Then replication ceases very quickly. Infectious virus was never isolated from non-respiratory tract tissues of these birds. Upon sequential immunohistological examination aMPV can be found in the epithelium of upper respiratory tract tissue for up to five days post-inoculation. No aMPV and/or histopathological damage was found in non-respiratory tissues, including the oviduct.

Khehra and Jones have specifically investigated the pathogenicity of a turkey-derived and a chicken-derived aMPV strain for the chicken oviduct. Although oviduct epithelium proved susceptible to aMPV infection in vitro, in vivo studies failed to show APV

replication in the hen oviduct. This is in accordance with the findings from dissemination studies by Cook and co-workers.

However, upon intravenous application of a chicken-derived aMPV strain to laying hens, Cook and co-workers repetitively found a drop in egg production and an increase of the percentage of low quality eggs (eggs with soft and thin shells).

Immunohistological investigation detected aMPV antigen in the oviduct epithelium up to nine days post-infection. Although virus application was by a very unnatural route here, the results of the studies prove the potential of the virus to affect the laying performance as seen in the field.

In another study Khehra and Jones confirmed that infection with aMPV is self-limiting and not persistent.

major factor in respiratory disease in chickens and the positive effect vaccination against aMPV will have.

Nobilis Rhino CV vaccine is a live vaccine against APV infection in chickens.

The vaccine contains an attenuated strain of APV. The parental strain, designated TRT 11/94, was isolated in the UK in 1994 from commercial breeding chickens experiencing some mortality, poor egg production and signs of swollen head syndrome and was shown to be a subgroup B strain of APV.

The vaccine can be applied oculonasally and by spray to chicks from day-old.

The vaccine protects against the development of clinical signs of the disease caused by wild type APV infection of broiler chick-

ens and, in combination with subsequent vaccination with inactivated APV vaccine, against the negative effects of wild type APV infection on the laying performance of laying birds.

One vaccination with the product results in protective immunity during the whole rearing period of a broiler bird and, after subsequent vaccination with inactivated APV vaccine, for the whole normal laying period of laying birds.

A big practical advantage is that the Nobilis Rhino CV has shown compatibility when applied with other respiratory vaccines. This is essential to obtain optimal protection in a situation where multiple respiratory vaccinations are present in rearing poultry. ■

Diagnosis

Clinical diagnosis should be confirmed by virus isolation and identification on tracheal organ culture or by passage in embryonated eggs inoculated via the yolk sac. aMPV is difficult to isolate, due to the fact that the virus can not be reisolated at the moment clinical signs appear (Fig. 1).

Detection of the virus in clinical specimens by specific immuno-staining of turbinate or sinus sections or by modern molecular-biological techniques like RT-PCR or detection of aMPV infection by serological means is more likely to be successful.

Prevention and control

Basically, respiratory disease in chickens is a multifactorial disease. Infection with avian pneumovirus is one of these factors, predisposing the chickens for serious secondary bacterial infections such as caused by *E. coli* or *Ornithobacterium rhinotracheale*.

Obviously, preventive vaccination against these secondary infections and/or treating them by antibiotics will reduce the severity of the disease.

A more direct and effective approach, however, is eliminating one of the primary disease causing factors by vaccination against aMPV. Infection with aMPV protects against the effects of subsequent infection with aMPV.

The relevance of the aMPV vaccination approach in disease reduction in chickens was shown earlier with Intervet's turkey-derived RTV 8544 vaccine.

Field trials performed with the vaccine in broilers showed a highly significant ($p < 0.001$) reduction of mortality in broilers after live vaccination, compared to previous rounds without vaccination.

In parallel flocks of broiler-breeders the non-vaccinated flock showed respiratory and SHS signs, but in the vaccinated flock, that became infected with aMPV at the same time, signs were absent. These studies show both the importance of aMPV as a