

The challenge of avian pneumovirus

by Marcos Bentué, DVM, poultry technical services, Laboratorios Hipra SA, Girona, Spain.

The replication of APV (avian pneumovirus) in the presence of secondary agents and environmental distress lead to the development of respiratory clinical signs in the birds. As secondary agents, *Escherichia coli*, *Pasteurella*, *Ornithobacterium rhinotracheale* (ORT), *Mycoplasma gallisepticum* and others are frequently isolated from field cases of broiler flocks showing SHS (Swollen Head Syndrome). The participation of respiratory viruses has also been proved.

Understanding the virus

The APV virus is a pneumovirus member of the Paramyxoviridae family, but without neuraminidase and haemagglutinin activity. This might explain the lower mortalities that the APV virus primarily produces in comparison to other members of the family.

The genome of this virus has some particularities that can clarify some of its behaviours. A single strand RNA genome containing nine fragments codifies for distinct structural and functional proteins. The most important polypeptides are G glycoprotein, responsible for the attachment of

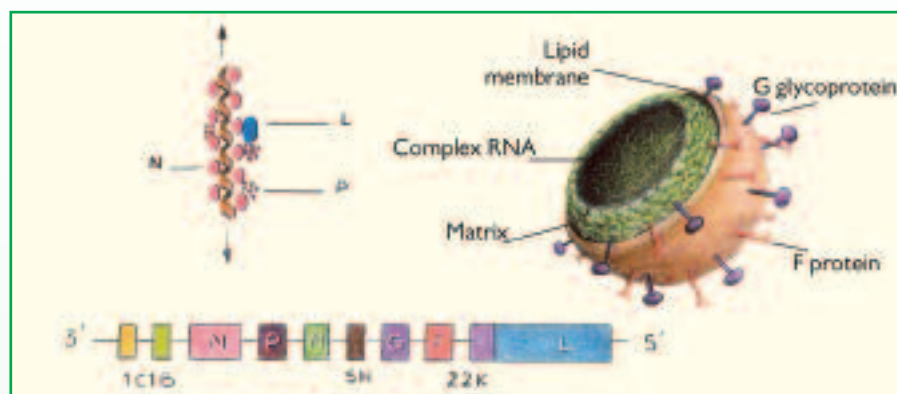


Fig. 1. The genome of avian pneumovirus.

the virus to the target cell, and the F protein, which permits the fusion and penetration of the virus within the cell, as well as the fusion of infected cells to the adjacent cells. This fact increases the transmission of the virus among neighbouring cells.

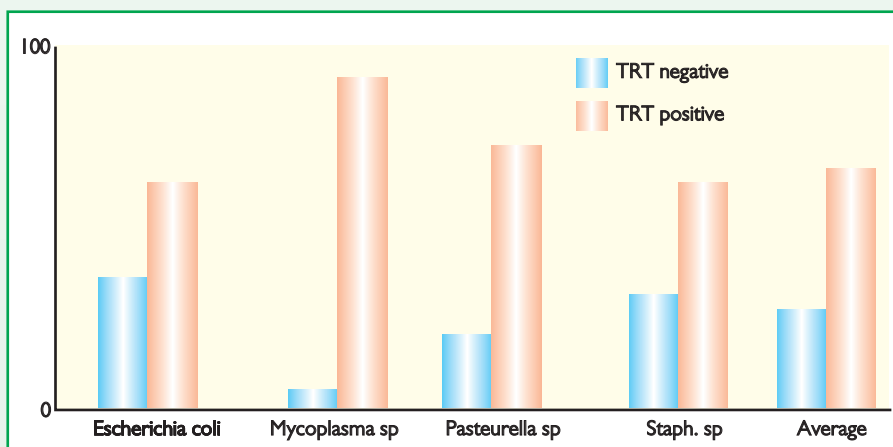
The M protein, stands for the matrix of the virus, the N protein, is related to the nucleoprotein and both L and P proteins build the viral transcriptase of the virus. N, L and P proteins are closely attached to the RNA of the virus and conform the viral nucleocap-

side. Immunologically, the most important proteins seem to be G and F, which concentrate the most research efforts (see Fig. 1).

There are three antigenic types that have been currently identified – A, B and C. Predominance of A or B depends on the country and C (Colorado strain) is until now exclusively confined to the United States.

Recently, the involvement of diverse avian species opens new debates on the epidemiology of APV infection.

Fig. 2. From 250 bacterial isolates identified from common respiratory outbreaks an average of 70.37% of the birds were positive to APV virus. Indirect ELISA was employed for the test (CIVTEST AVI APV, Indirect ELISA). These data indicate that APV virus may act as an enhancer of the action of bacteria, facilitating the entrance and the action of bacterial pathogens. In particular, *Mycoplasma sp.* showed the clearest difference, since the 95.24% of the birds from which mycoplasma was isolated as responsible for a clinical outbreak were positive to APV virus.

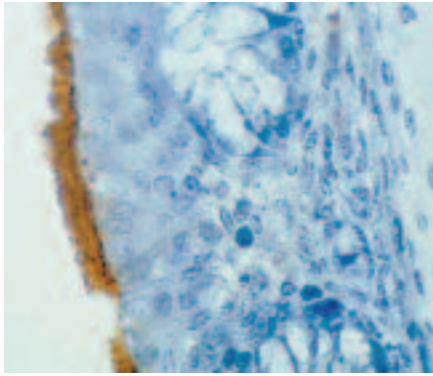


After some studies, it has been proved that some populations of pheasants, guinea fowl, geese, sparrows, pigeons and other wild birds have been positive to the presence of viral antigen by PCR or have developed specific antibodies against APV.

What makes the difference?

APV virus might not be as lethal as Newcastle disease virus or influenza virus, but still has a significant economic and

Continued on page 8



APV virus (in orange) replicating in the nasal turbinates epithelium showed by the streptavidine biotin immunoperoxidase technique (IP).

Continued from page 7

pathological importance in modern poultry production. Its replication in the upper respiratory tract cells, mainly in the nasal turbinates, the trachea, the conjunctive membranes and the sinuses damages the ciliated cells and disturbs the efficacy of this mechanical defensive barrier, permitting secondary agents to replicate.

The most frequent clinical signs are nasal distillation, watery eyes, sneezes and unspecific respiratory symptoms, among which the most recognisable might be the so-called swollen head.

Additionally, it has also been proved that replication in pulmonary tissue may produce even pneumonia.

The explosive dissemination of the virus

can raise morbidity to 100%. Mortality can reach 30% depending on the occurrence of secondary agents.

Above all, the most relevant particularity is its special ability to paralyse cilia activity (see photo), handicapping the important cleaning function of these cell organs. This simple fact increases dramatically susceptibility to common pathogens (see Fig. 2) and environmental factors.

Harmful consequences

As mentioned previously, inflammation of the trachea, nasal turbinates, ocular conjunctiva and sinuses cause breathing difficulties, secretions, sneezes, swollen sinuses, and other non-specific respiratory signs.

Particularly, the infection in laying birds and broiler breeders causes severe drops in production as high as 30% and pales brown eggs due to the replication of the virus in the oviduct.

Not only this, but nervous signs can also appear in breeders up to 1%. Indirectly, irritating factors like dust, ammonia, unbalanced humidity or bad ventilation, can cause an easier colonisation of respiratory tract by bacteria.

The severity of economic consequences is directly related to virus pressure in the field and the incidence of secondary agents.

Diagnostic tools

Differential diagnosis from other respiratory processes should be performed. Virus isolation becomes challenging since the virus has often been cleared up from the flock when clinical signs are evident. It is advisable to sample birds from the houses which are at the early stages of infection and do not show any clinical signs yet. Classical isolation techniques with chicken embryos generally fail, since the virus grows better in turkey and chicken primary organ cultures, ideally in tracheal organ cultures (TOC).

Cilia paralysis is observed 4-8 days after inoculation of suspicious samples. Serology, with serum neutralisation, immunofluorescence and ELISA offers good confirmation options. Molecular biology techniques, like PCR are continuously offering new advantageous approaches to diagnosis.

Vaccines: proven protection

As for any syndrome, control strategy should be based on multiple factors—not only primary, but also secondary agents and environmental factors should be seriously considered. There is no effective treatment for APV infection. Treatment with antibiotics should be set for the control of secondary bacteria and optimum ventilation conditions should be provided to the birds.

Vaccination against APV virus is the most effective control method and should be

implemented in problematic SHS farms. Use of both live attenuated and inactivated vaccines is proven to be the most suitable vaccination strategy to protect broiler breeders and layers.

They are most susceptible to SHS by the peak of production but cases have been reported at any age, so vaccination is highly effective to avoid the unwanted clinical signs and production drops.

Live vaccines should be administered paying attention to vaccination technique, since 100% of the birds should be vaccinated to obtain the best results.

An accurate vaccination method will permit the vaccine to replicate fast in the respiratory tract and homogeneously throughout the flock, promoting a good cell-mediated immunity and blocking efficaciously the entrance of the virus.

This local action is very important to achieve quick and effective protection against challenge.

Best results are obtained by ocular-nasal route or by spray vaccination and more difficulties are encountered by oral route since poorer uniformities are achieved and local immunity seems not be so efficaciously built up.

Inactivated vaccines stimulate B-lymphocytes to produce specific Ig G against APV virus antigens. There exist live vaccines from either chicken or turkey origin.

Humoral immunity also seems to play a relevant role to obtain long term protection and adequate neutralisation of the virus.

When a live vaccine precedes an inactivated vaccine, immune response can reach superior levels than with inactivated vaccines alone.

Some precautions should be taken on establishing the vaccination programme, since no other vaccine with respiratory tropism should be administered at least one week before and after vaccination with live APV virus.

Interferon secretion and viral competition could inhibit the replication of the vaccine virus, impairing correct immunisation of birds.

Broiler vaccination is practiced in problematic areas with satisfactory results but needless to say that firstly we should keep under control lethal and immunosuppressive viruses, which may jeopardise the efficacy of the APV vaccine.

The most common broiler vaccination programme is one single live vaccine, but in some areas broilers are vaccinated twice depending on the field pressure.

Conclusions

The importance of this disease lays on the fact that the virus has a special tropism for ciliated respiratory cells.

Together with APV virus, bacteria and other damaging elements can more easily penetrate within the respiratory tract of the birds, causing sub-optimal productions,

common respiratory processes, and diverse unspecific respiratory symptoms.

In conjunction with strict biosecurity measures, good management and a thorough control of hygiene, a sound vaccination programme combining live and inactivated vaccine should be applied. In particular, a good vaccination technique should be employed to ensure the success of immunisation, since local immunity plays an essential role in protection mechanisms.

The better knowledge of APV virus has permitted us to achieve efficacious control strategies, but further investigations on diagnosis, pathogenicity, immunity and other important areas must still be carried out to fully palliate the harmful consequences of this interesting disease. ■

