Newcastle disease control in poultry

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Newcastle disease (ND) is one of the most important diseases infecting poultry and other bird species and is a global threat to commercial poultry production throughout the world. ND is caused by specified viruses of the avian paramyxovirus type 1 (APM-1) serotype of the genus Avulavirus belonging to the subfamily Paramyxovirinae, family Paramyxoviridae.

The enveloped virus has a negative sense single stranded RNA genome of approximately 15 kilo-base pairs which codes for an RNA directed RNA polymerase, haemagglutinin-neuraminidase protein (HN), fusion protein (F) matrix protein (M), phosphoprotein (P) and nucleocapsid protein (NP).

The severity of the disease depends both upon the virus strain and the species of birds which are infected, as well as factors like the general health status of the birds.

Newcastle disease virus (NDV) isolates have been grouped into five pathotypes that relate to the disease signs produced in infected fully susceptible chickens:

- **Viscerotropic velogenic NDV**, which produces acute lethal infections in which haemorrhagic lesions are prominent in the gut.
- **Neurotropic velogenic NDV**, which produces high mortality preceded by respiratory and neurologic signs.
- **Mesogenic NDV**, which produces low mortality, acute respiratory disease and nervous signs in some birds.
- **Lentogenic NDV**, which produces mild or inapparent respiratory infections.

Cyanosis (purpling) of the comb in velogenic Newcastle disease.

- **Asymptomatic enteric NDV**, which are avirulent viruses that appear to replicate primarily in the gut.

These groups are not completely clear cut and some overlapping between the signs associated with the different groups has been reported.

Virulent ND is enzootic causing frequent epizootics throughout Africa, Asia, Central America, parts of South America and Australia. The occurrence of ND in commercial poultry is reportable to the World Organisation of Animal Health (OIE) and impacts international trade in poultry business. Worldwide control of ND could be approached only if all countries report outbreaks within their borders to international agencies.

The molecular basis for pathogenicity is dependent on the fusion protein cleavage site amino acid sequence and the ability of specific cellular proteases to cleave the fusion protein of different pathotypes.

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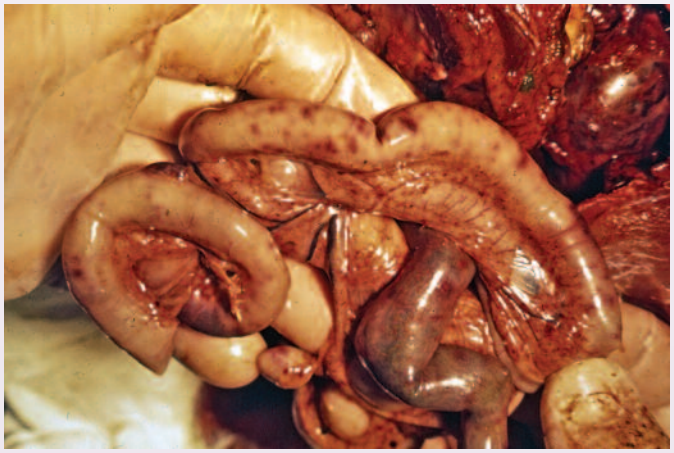
Left, intestinal and, right, caecal lesions in viscerotropic Newcastle disease.



Pictures courtesy of AAAP

Oedema in velogenic viscerotropic Newcastle disease.

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During replication, ND virus particles are produced with a precursor glycoprotein, F0, which has to be cleaved to F1 and F2 for the virus particles to be infectious. Viruses with monobasic amino acid motif at the F0 cleavage site are apathogenic because this substrate is susceptible only to extracellular trypsin like proteolytic enzymes excreted onto the surface of mucous membranes, therefore, the infection remains localised and asymptomatic. By contrast, viruses with dibasic amino acids in the fusion protein sequence allows for systemic spread of velogenic NDV.

Molecular biology technology has enabled a much greater understanding of the pathogenicity and antigenicity of NDV. Improved techniques for nucleotide sequencing and the availability of sequence data of more ND viruses could give meaningful results in phylogenetic analysis.

Genetic diversity

Considerable genetic diversity has been detected, but viruses sharing temporal, geographical, antigenic or epidemiological parameters tend to fall into specific lineages or clades and this has proven valuable in assessing both the global epidemiology and local spread of ND. Phylogenetic analysis revealed that two major separations occurred during the history of ND. An ancient division in the original reservoir (wild waterbird species) led to two basal sister classes, class I and II.

These categories emerged only in the secondary host (chicken) and only in the recent past (after the 1960s).

Class II contains genogroups that showed region specific occurrence and temporal distribution with also apparent links to well defined epizootics.

These groups include all the available virulent strains known to be responsible for early ND outbreaks. Genotype I comprise viruses that reside either in feral waterbirds or occasionally in chickens.

Interestingly, the virulent descendants of the Australian avirulent endemic strains belong to this specific genotype. One such avirulent virus which is similar to those that had endemically infected chicken flocks in Australia acquired virulence.

Lentogenic strains were found in chickens in North America in the 1940s and correspond to genotype II. These strains have been used as live vaccines since then (La Sota, B-1) and a more recent strain (VGGA) has been placed in the same group.

Regardless of whether control is applied the objective is either to prevent susceptible birds from becoming infected or to reduce the number of susceptible birds. Protection against ND is achieved using live and/or inactivated vaccine strains. Vaccination protects susceptible birds against disease by producing an antibody response either locally, systemically or both.

The mucosal application of attenuated live virus induces both systemic and local immunity, whereas parental immunisation with inactivated vaccine generally induces systemic immunity with little local protection.

Mucosal antiviral immunity is believed to depend on locally produced antibodies that are released across the epithelium onto the mucosal surface.

IgA is mainly produced locally and transported through the epithelial cells by a secretory component with virus neutralising activity which limits, but does not prevent viral multiplication after mucosal challenge.

B1, LaSota and VG/GA strains induced NDV specific IgA production at the upper respiratory tract. However, in the intestinal tract the VG/GA strain induced higher IgA levels as well. After vaccination, the VG/GA strain has been detected by reverse transcriptase polymerase chain reaction (RT-PCR) in the respiratory and intestinal tract, with a preferential tropism for the latter.

Different policies

Some countries have adopted eradication policies with compulsory slaughter of infected birds, their contacts and products. Other countries allow only the administration of specific live vaccines (produced from lentogenic strains) and consider some vaccines to be excessively virulent as well.

Conversely, some countries have continuing presence of circulating highly virulent virus, which is not seen as overt disease because of vaccination.

ND virus strains used in commercial live virus vaccines fall into three groups: asymptomatic such as VG/GA, V4; lentogenic vaccines such as Hitchen-B1, La Sota, F; and mesogenic vaccines such as Roakin, Mukteswar and Komarov. These strains have been subjected to selection and cloning to fulfil different criteria in their production and application.

The mesogenic vaccine viruses all have two pairs of basic amino acids at the F0 cleavage site and ICPI values of around 1.4. Most live virus vaccines are grown in the allantoic cavity of embryonated fowl eggs but some, notably some mesogenic strains have been adapted to a variety of tissue culture systems. Administration can be either by individual or massive vaccination methods.

Potent inactivated vaccines

Inactivated vaccines are prepared from allantoic fluid that has had its infectivity inactivated and incorporated into an emulsion with mineral oil, thus these vaccines are more expensive than live vaccines.

Both virulent and lentogenic strains have been used as inactivated vaccines, but from the aspect of safety control, the use of the latter appear more suitable because the manipulation of large quantities of virulent virus is involved, as well as the dangers of inadequate inactivation and possible subsequent contamination.

A high yield of virus to produce a potent vaccine is important, as no virus multiplication takes place. Exceptionally high titers can be obtained by the Ulster 2C strain, however, some commercial inactivated vaccines are produced when Hitchner B1, La Sota or F strains are used as seeds.

Their use ensures individual handling and injecting birds intramuscularly or subcutaneously, thus each bird receives its individual standard dose.

ND immunisation programmes involving the use of live and inactivated oil emulsion vaccines usually result in serum higher antibody titers that should persist longer throughout the life of the bird. This would result in a shorter period of excretion of the challenge virus and, consequently, less contamination of the poultry house. Birds will require more challenge virus load to become detectably infected.

Several groups have reported immunisation with the HN gene expressed in recombinant fowl poxvirus, vaccinia virus, pigeon poxvirus or turkey herpesvirus.

Nevertheless, some of the issues of this new vector vaccine technology for use in commercial poultry relate to efficacy in the face of maternal immunity.

Individual applications of two or more doses to protect against clinical signs, plus the fact that recombinant products have been shown to provide excellent systemic immunity, are advantageous.

However, their ability to provide local protection, including partial protection of the trachea is less than that seen with conventional live NDV vaccines and needs to be considered. ■