Infectious bursal disease – a worldwide problem

Infectious bursal disease (IBD), first recognised in the early 1960s, remains a significant problem in the world's poultry industries.

The infection is encountered in all countries, with the exception of New Zealand, in various forms ranging from mild weight loss and low mortality in broilers associated with infection and immunosuppression, to severe losses following exposure to very virulent strains (vvIBDV) of the virus.

Since the virus is resistant to environmental exposure and persists in litter and housing, early exposure of flocks invariably occurs, resulting in the destruction of lymphoblasts in the bursa of Fabricius.

This reduces the production of B-lymphocytes, impacting the development of the immune system and production of antibodies.

Exposure of susceptible or incompletely vaccinated, immature flocks to IBDV may result in mortality, which can range from 2-25% depending on the proportion of immune chicks, the severity of virus and concurrent stress factors, including climatic extremes, malnutrition and exposure to other immunosuppressive viruses.

Vaccination of day old chicks for protection against IBD challenge, which is used extensively in Europe, Asia and Latin America.

Classification of viruses

IBD viruses are classified in the family Birnaviridae. Type-1 IBDVs are responsible for infectious bursal disease in chickens. Type-2 IBDVs, isolated from turkeys, are nonpathogenic.

Serotype-1 viruses can be differentiated by molecular biological techniques, including monoclonal antibody typing and gene sequencing.

From an epidemiologic perspective, classic Type-1 viruses are responsible for mild infection – the US Delaware variants A through E are extremely immunosuppressive, and the very virulent Type-1 strains which emerged in Europe in the late 1980s and early 1990s caused acute mortality exceeding 50% in susceptible and inadequately vaccinated flocks.

Over 40 isolates of IBDV have been isolated, characterised and described in the literature through 1995.

The relationship of isolates in specific regions of the poultry raising regions of the world can now be established using gene sequencing techniques.

Epidemiology of IBD

Mild Type-1 IBDV is widely distributed in poultry raising areas of the world. In the United States and Central America, the variant Delaware strains predominate.

The very virulent IBD strains which emerged in Europe during the late 1980s have extended throughout the continent, and are encountered in Asia and Africa.

Some vvIBDV strains with similarities to the European serotype have been isolated in the Caribbean and Brazil. North America is free of vvIBDV.

Infectious bursal disease virus is highly contagious and can be transmitted by both the direct and indirect routes.

Contact between susceptible and infected flocks, which occurs in multi-age broiler, breeder and pullet rearing operations, results in 'rolling infection' with exposure of chicks shortly after placement.

The virus can persist for weeks in poultry litter and is resistant to 0.5% formalin for six hours and in both phenolic and quaternary ammonium compounds under laboratory conditions for extended periods over a pH range of 2 through to 12.

The virus can survive in cooked chicken meat raised to an internal temperature of 74°C. Vehicles of transmission include contaminated clothing and footwear and equipment transferred among farms.

The virus has also been isolated from mosquitoes, Alphitobius beetles, rodents and wild birds which frequent the vicinity of chicken houses.

The virus is not transmitted vertically through the egg.

Because vaccines have been extensively applied at both the breeder and commercial flock levels for three

Continued on page 16

Healthy chicks from a well managed IBD programme.

Continued from page 15
decades, classic outbreaks of IBD are not generally encountered. The disease persists as low grade infection in replacement breeders, egg production pullets and in broilers. Clinical severity is a function of deficient vaccine protection and inappropriate biosecurity which contributes to early infection.

Clinical signs

Exposure to classic mild and variant strains of IBDV during the first 10 days generally does not result in any overt signs. Exposure of flocks with inadequate protective antibody at approximately 3-4 weeks of age results in flock morbidity ranging from 2-8%, depending on the proportion of susceptibles, the strain of the virus and concurrent disease challenge and environmental stress.

Affected birds are depressed and usually assume sternal recumbency for approximately 12-24 hours before dying.

Mortality is extremely variable.

In uncomplicated cases, acute infectious bursal disease in broilers is seldom responsible for more than 3% losses.

Flocks which have been immuno-compromised by early or mid-cycle IBDV generally show an escalation in mortality during the terminal third of the growing cycle or at the mid-point of the rearing period in replacement breeding stock and replacement egg strain pullets.

Mist application of IBD vaccine to day old chicks, which is used primarily in Europe and Asia.

Mortality is usually attributed to E. coli septicemia, especially with concurrent infection with respiratory viruses, including Newcastle disease, low pathogenicity avian influenza and laryngotracheitis.

The immunosuppressive effect of IBDV is synergistic with other immunosuppressive agents including Marek's disease virus, chicken anaemia virus and dietary aflatoxin.

Diagnosis

Diagnosis is based on the history, including a record of previous outbreaks of IBD and obvious deficiencies in vaccination, especially in multi-age operations.

Postmortem changes are characteristic comprising enlargement of the bursa of Fabricius in the acute stage of infection, followed by atrophy of the organ in survivors.

It is noted that postmortem examination of both dead and sacrificed moribund birds during an outbreak at mid cycle will show a range in intensity of changes in the bursa encompassing acute inflammation to chronic atrophy.

The diagnosis can be confirmed by histological examination of bursal tissue from representative birds.

Retrospective serology, comparing acute and recovery stage sera, applying ELISA or serum virus neutralisation will show a significant elevation in antibody titre.

Under commercial conditions, it is necessary to differentiate between an antibody response following vaccination and the numerically higher titres following infection.

Bursal tissue can be submitted to a diagnostic laboratory for isolation and identification of IBDV. Rapid techniques including reverse transcriptase poly-

merase chain reaction can be used to identify RNA associated with IBDV and provide a diagnosis within a day of submission of specimens.

In contrast, traditional isolation techniques coupled with serum virus neutralisation require seven to 12 days to obtain a diagnosis.

Prevention of IBDV

Application of sound biosecurity is obviously appropriate for commercial replacement pullets, breeding stock and broilers placed according to an all-in/all-out cycle.

It is emphasised that commercial procedures used to decontaminate housing and equipment are generally inadequate to destroy IBDV.

Despite these limitations, management procedures should be selected to limit the possibility of introduction of IBDV until solid protective immunity has been stimulated by vaccination.

Vaccination programmes should be based on the risk and consequences of infection, and should take into account the strain(s) of IBDV to which flocks will be exposed.

All vaccination programmes should be monitored by periodic assay of antibody titre using ELISA technology. Protection of young flocks is based on a combination of adequate levels of maternal antibody transfer, followed by administration of sequential doses of live attenuated mild and then intermediate IBDV vaccines at an appropriate age.

Parent flocks can be immunised by administration of a mild vaccine at approximately 14 days of age to prime the immune system. This is followed by a less attenuated (intermediate) strain vaccine at approximately 35-40 days of age. Humoral immunity is further boosted by administration of an inactivated oil emulsion vaccine prior to onset of production.

Broiler flocks can be protected by administering mild strain IBDV vaccine by the in ovo route at transfer or at hatch in high risk areas.

When maternal antibody level is uniformly high and biosecurity procedures protect against early challenge, either mild or intermediate strain IBD vaccine is administered in nonchlorinated drinking water at approximately 7-10 days of age.

This is followed by an intermediate strain at 21-24 days of age. In areas with high risk of exposure to vvIBDV, more aggressive vaccination programmes are required.

It is appropriate to administer intermediate plus strains which will result in some bursal damage, but will provide adequate and durable immunity. Experience has shown that aggressive vaccination programmes against vvIBD result in displacement of field virus by the intermediate plus vaccine strains. Monitoring performance and reference to ELISA serology can guide decisions concerning selection of vaccine strains and age of administration.

It is necessary to modify vaccination programmes depending on season, changes in management practices or following the introduction of new strains of IBDV into the area of operation.

Effective control of IBD can only be accomplished with concurrent vaccination against Marek's disease and immunisation of parent flocks against CAV to prevent vertical transmission of this agent.

The deleterious effects of E. coli following IBDV immunosuppression can be ameliorated by eliminating mycoplasmosis, and control of respiratory viral pathogens by appropriate vaccination against infectious bronchitis, Newcastle disease and laryngotracheitis.

Maintaining the performance of flocks in the presence of IBDV requires a coordinated programme of biosecurity and vaccination against the primary pathogen and concurrent infections, in addition to exercising high standards of management and biosecurity. ■