Infectious bronchitis (IB) is among the most common and difficult of all poultry diseases to control. IB is highly contagious and results in significant economic losses in commercial broiler, layer and breeder chickens. The causative coronavirus, infectious bronchitis virus (IBV), frequently causes respiratory disease in young chickens and egg production losses in hens. In addition, some strains of the virus exhibit a renal (kidney) tropism and produce up to 30% mortality in affected flocks (Ziegler et al., 2002).

IBV targets the trachea and other respiratory tissues. Damage to defense mechanisms of the respiratory system which are responsible for trapping and clearing inhaled bacteria often predisposes chickens to secondary bacterial infections. Bacteria such as *E. coli* and mycoplasma may colonize the respiratory tract, produce toxic products causing airsacculitis, perihepatitis, and pericarditis. Chickens inoculated with IBV and *E. coli* were found to have more severe and persistent respiratory lesions than those inoculated with IBV alone (Nakamura et al. 1992). Other factors such as ammonia, temperature fluctuations, and social stress (pecking) contribute to the susceptibility of layer type chickens to bacterial infections (Phillip and Voss, 2001). In addition, immuno-suppressive infections with IBDV in combination with IBV reduced macrophage activity vs. *E. coli* (Naqi et al., 2001) and increased IBV persistence (Rosenberger and Gelb, 1978; Pejkovski et al., 1979)

IBV is perhaps best known for its existence as numerous antigenic types or serotypes. Although antigenic variation of the virus has been recognized for years, it is only since the mid-late 1990s that the scientific community has had the capability to truly appreciate the magnitude of the genetic diversity of the virus.

IBV is uniquely suited to undergo mutation during its replication cycle. Replication of the virus' RNA genome is error-prone resulting in mutations. The major target for mutation is the gene encoding the spike (S) envelope protein that the virus uses to attach to the host cell. Mutations in S result in antigenic changes and the emergence of variant strains as well as subtypes of recognized serotypes. The S protein gene is able to tolerate numerous mutations without compromising the virus' ability to replicate and cause disease.

New mutant IBV strains are subject to immunological selection so that only the most antigenically novel variants persist in poultry populations. A new variant that is not antigenically novel, e.g. one similar to a vaccine strain used on the farm, will not persist in the flock because the vaccine-induced immunity will eliminate it from the population. Conversely, newly mutated variants that are antigenically distinct from vaccine strains will, in essence, have a far greater potential to escape vaccine-induced immunity, persist in flocks, and have the potential to cause disease.
The primary source of highly novel IBV variants are commercial layer flocks. IBV variants arise most frequently in commercial layers raised on farms with many flocks of different ages also referred to as multiple age layer operations or multiple age farm complexes. These farms provide all the necessary conditions that favor the emergence of new mutant IBV variants. Layer flocks of different ages frequently numbering in excess of a million birds, are housed in close proximity on egg production farm complexes. Periodic introduction of new pullets, and the continual re-infection and recycling of IBV in the layers, results in a greater opportunity for infection and spread than occurs on farms using a single age, "all in-all out" management system. Novel IBV variants build up in layer complex houses over time since the premises are rarely, if ever, cleaned and disinfected. Importantly, vaccine induced immunological mechanisms provide a selective pressure for the most antigenically novel variants as new variants arise on a frequent basis. Variants that evolve in layer flocks pose a serious threat to nearby broiler flocks.

Another important and common source of pathogenic IBV is live attenuated vaccines that have undergone reversion to virulence under field conditions (Nix et al., 2001). This problem is often seen in the production of broiler chickens. Attenuated vaccines contain millions of highly related but not identical virus particles referred to as subpopulations. The subpopulations in the vaccines differ in their virulence (level of attenuation) and even slightly in their antigenic characteristics (subtype). Live IBV vaccines are considered to be unstable due to the presence of the different subpopulations and their ability to change (shift) depending on the host (embryonated egg vs. chicken) in which they are grown. For example, back-passage of embryonated egg-derived vaccine in the chicken results in shifting of the viral subpopulations in favor of a predominant virulent subpopulation over the attenuated subpopulation found in the original vaccine. Similarly, a slight shift or change in the antigenic characteristics of the vaccine may also occur through back-passage. Over time, and in the absence of farm clean out and disinfection, the back-passaged vaccine becomes established as a significant IBV field challenge causing disease losses. Indeed, laboratory studies have shown that vaccines may revert to partial or full virulence within only 3-6 back-passages in chickens (Hopkins and Yoder, 1983). Practices associated with enhancing vaccine back-passage should be discontinued. These include over-diluting (cutting) IBV attenuated vaccines, using sprayers that apply wide-ranging and large particle sizes, and using a vaccine strain(s) only on an inconsistent or seasonal (winter) basis.

Pathogenic IBV vaccine-derived field isolates are now thought to be the primary cause of many of the Arkansas vaccine problems in broilers on Delmarva in the middle 1990s (Nix et al., 2001). The back-passage of vaccine should be considered as a source of infection in situations where the incidence of isolation of a given serotype of IBV is high in regions where a vaccine serotype has been used with little or no apparent reduction in disease incidence.

**Diagnosis of IB in Broilers**

Control of IB in commercial broilers can be a significant challenge. The first step in controlling a suspect IB problem is confirming that the problem is in fact IB. Diagnosis is essential. Affected broiler flocks generally present with a clinical history such as respiratory disease. However, respiratory disease may be due to other viral and bacterial causes. Laboratory tests are needed to confirm an IB diagnosis. ELISA serology is inexpensive and specifically
identifies IBV antibodies produced in response to infection. ELISA serology can provide a basis for analysis of samples to determine if IBV antibody levels are rising in response to infection. Antibody responses to vaccination generally produce lower ELISA titers than is observed following a virulent field challenge.

Hemagglutination-inhibition (HI) tests may be performed on sera from commercial broilers to identify a causative serotype responsible for IBV infection. However, HI antibodies produced in response to IBV vaccinations and field exposures can become cross-reactive and non-specific and thus may not be indicative of the strains responsible for recent disease episodes (Gelb and Killian, 1987). Caution must be taken when interpreting HI results.

Virus isolation and identification is critical for diagnostic purposes. Virus isolation may be accomplished through sentinel studies using IBV-vaccinated or susceptible chickens (Gelb et al., 1989) or directly from the diseased broilers. We prefer samples of trachea or lung rather than cecal tonsil specimens because the latter often are persistently infected with IBV vaccine strains as well as other viruses and complicate isolation. Identification of IBV is much faster and easier today with the application of reverse transcription polymerase chain reaction (RT-PCR) assays for the virus (Kwon et al., 1993; Keeler et al., 1998; Kingham et al., 2000). Samples of isolated IBV may be shipped to laboratories for RT-PCR and results may be obtained as rapidly as several weeks. For testing in the USA, IBV specimens originating from other countries must be inactivated prior to shipment and accompanied by a government-issued import permit.

Control of IB through Vaccination

As mentioned above, IB can be very difficult to control. There are only a very limited number of vaccines available given the tremendous number of recognized strains of the virus. However, effective control strategies can be developed to reduce the impact of the disease.

Broiler Breeders

Broiler breeders in the USA are raised on single-aged farms, so the strategies used to control IB are quite different than in commercial layers. For example, drinking water vaccination of broiler breeder pullets is more common than spray. Inactivated vaccines are not consistently given to pullets. When used however, inactivated vaccines are given at about 18 weeks of age, 6-8 weeks later than in commercial layers. Vaccination of broiler breeders in the USA is not practiced, again primarily because flocks are raised in the single age management and good biosecurity is used to reduce the introduction of variant IBVs.

Broilers

Broiler chickens are routinely vaccinated in the USA. Only live vaccines are used, however, because inactivated vaccines are expensive and are not particularly effective for broilers. Live vaccines are commonly combined with Newcastle disease virus (NDV) vaccines such as B1 or LaSota, and are given via coarse spray in the hatchery and the field between the ages of 14 and 18 days. Live vaccines are available for four serotypes of IBV; Mass, Conn, Ark and Delaware 072. The vaccine serotypes are generally given as bivalent (e.g. Mass + Conn or Mass + Ark) combinations to broilers. Application of the vaccines should be done in a way to minimize back-passage and reversion to virulence as discussed above. Selection of appropriate vaccine serotypes is essential to achieve the best protection against endemic field strains of IBV. Some
vaccine combinations (e.g. Mass + Ark) give better cross-protection against variants than others (e.g. Mass + Conn).

Of course, vaccination programs are only effective when combined with other efforts such as farm biosecurity, ongoing clean-out and disinfection programs, and control of immunosuppression. More information on IB and IBV is available (Cavanagh and Naqi, 1997; Gelb and Jackwood, 1998).

References


Nix, W. A., D. S. Troeber, B. F. Kingham, C. L. Keeler, Jr. and J. Gelb, Jr. Emergence


