

Infectious Bronchitis Virus: Classical and Variant Strains¹

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Summary

Infectious bronchitis (IB) is an acute and highly contagious respiratory disease of chickens. The disease is characterized by respiratory signs including gasping, coughing, sneezing, tracheal râles, and nasal discharge. In young chickens, severe respiratory distress may occur. In layers, respiratory distress, decrease in egg production, and loss of internal egg quality and egg shell quality are reported. Some strains of the virus cause severe kidney damage and may be associated with high mortality.

Occurrence

IB has been reported as a disease only in chickens. All ages of chickens are susceptible to infection, however, clinical disease severity varies. IB is considered to be worldwide in distribution. The incidence is not constant throughout the year, being reported more often during the cooler months.

History

The disease was first described in 1931 in a flock of young chickens in the USA. Since that time, the disease has been identified in broilers, layers and breeder chickens throughout the world. Vaccines to help reduce losses in chickens were first used in the 1950s.

Etiology

IB is caused by a coronavirus. It is an enveloped, single-stranded RNA virus. Three virus-specific proteins have been identified; the spike (S) glycoprotein, the membrane or matrix (M) glycoprotein, and the nucleocapsid (N) protein (Figure 1). The crucial spike glycoprotein is comprised of two glycopolypeptides (S1 and S2). These spikes or peplomers can be seen projecting through the envelope on electron micrographs, giving the virus its characteristic corona (Figure 2). Hemagglutination Inhibition (HI) and most SN antibodies are directed against this S1 portion. The unique amino acid sequences, epitopes, on this glycoprotein determine serotype. The virus is fairly labile (fragile), being easily destroyed by disinfectants, sunlight, heat and other environmental factors. IB virus has the ability to mutate or change its genetic makeup readily. As a result, numerous serotypes have been identified and have complicated efforts at control through vaccination. Three common serotypes in North America are the Massachusetts, Connecticut, and Arkansas 99 IB viruses. In Europe, various "Holland variants," usually designated using numbers (D-274, D-212) are recognized.

Several strains of IB virus have a strong affinity for the kidney (nephropathogenic strains). These strains may cause severe renal damage. This affinity for kidney tissue may have derived from mutation as a result of selection pressure

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following widespread use of the modified live IB vaccines. That is, after prolonged use of live IB vaccines, which provided protection against IB virus infection in respiratory tissues, new tissues where little protection was present were infected as a result of viral mutation. These viruses have become more prevalent in recent years.

Transmission

IB virus is spread by the respiratory route in droplets expelled during coughing or sneezing by infected chickens. Spread of the disease through a flock is very rapid. Transmission from farm to farm is related to movement of contaminated people, equipment, and vehicles. Following infection, chickens may remain carriers and shed the virus for several weeks. Egg transmission of the virus does not occur.

Clinical Signs - Chicks

Clinical signs include coughing, sneezing, râles, nasal discharge, and frothy exudate in the eyes. Affected chicks appear depressed and will tend to huddle near a heat source. In an affected flock, all birds will typically develop clinical signs within 36 to 48 hours. Clinical disease will normally last for 7 days. Mortality is usually very low, unless complicated by other factors such as *M. gallisepticum*, immunosuppression, poor air quality, etc.

Clinical Signs - Chickens

Clinical signs of coughing, sneezing and râles may be observed. A drop in egg production of 5 to 10% lasting for 10 to 14 days is commonly reported. However, if complicating factors are present, production drops may be as high as 50%. Eggs produced following infection may have thin shells, irregular shells, and thin, watery albumen. Loss of pigment in brown-shelled eggs is common. In severe complicated cases, chickens may develop airsacculitis. Chickens that experienced a severe vaccination reaction following chick vaccination or field infection during the first two weeks of life may have permanent damage in the oviduct, resulting in hens with poor production. In recent years, nephropathogenic strains have become more common in laying flocks. These strains may cause an elevated mortality during the infection or long after as a result of kidney damage that progresses to urolithiasis.

Lesions

Lesions associated with IB include a mild to moderate inflammation of the upper respiratory tract. If complicating factors are present, airsacculitis and increased mortality

may be noted, especially in younger chickens. Kidney damage may be significant following infection with nephropathogenic strains. Kidneys of affected chickens will be pale and swollen. Urate deposits may be observed in the kidney tissue and in the ureters, which may be occluded. Laying chickens may have yolk material in the body cavity and developing yolks in the ovary may be flaccid. Infection of very young chicks may result in the development of cystic oviducts.

Diagnosis

Serologic testing to determine if a response to IB virus has occurred in a suspect flock is performed by comparing 2 sets of serum samples, one collected at onset of clinical disease and the second at 3 ½ to 4 weeks later. Serologic procedures commonly used include enzyme labelled immunosorbent assay (ELISA), virus neutralization, and HI. Confirmation of IB requires isolation and identification of the virus. Typically this is done in specific pathogen-free chicken embryos at 9 to 11 days of incubation by the allantoic sac route of inoculation. Tissues collected for virus isolation attempts from diseased chickens include trachea, lungs, airsacs, kidney, and cecal tonsils. If samples are collected more than 1 week after infection, cecal tonsils and kidneys are the preferred sites for recovery of IB virus. Virus typing has traditionally been performed by neutralization using selected IB antisera. More recently, polymerase chain reaction (PCR) and restriction fragment length polymorphism (RFLP) have been used to differentiate IBV serotypes. Lesions in embryos are helpful in diagnosing IB. Affected embryos examined at 7 days after inoculation are stunted and have clubbed down and an excess of urates in the kidneys. The amnion and allantois membranes are thickened and closely invest the embryo. These embryos will not hatch. IB field virus may have to be serially passed in embryos to adapt the field virus to the embryos before typical lesions are recognized.

Control

Prevention of IB is best achieved through an effective biosecurity program. As a second line of defense, chickens in IB problem areas should be vaccinated with modified live vaccines to provide protection. The multiplicity of serotypes identified in the field presents a challenge in designing an effective vaccination program. To be successful in protecting chickens against challenge, it is essential to identify the prevalent serotypes in the region and to determine the cross-protective potential of available vaccines. In North America, the common serotypes used in most vaccination programs are the Massachusetts,

Connecticut, and Arkansas serotypes. These serotypes are available in both modified live vaccines and inactivated water-in-oil emulsions. Regionally important serotypes (e.g. California strains) may be included in inactivated vaccines. In Europe, various “Holland variants” usually designated by number (e.g. D-274, D-1466) are recognized. Polyvalent vaccines, which contain multiple strains, are also available. Control of other respiratory diseases (e.g. Newcastle, MG) and strongly immunosuppressive diseases (e.g. IBD, MD) must not be forgotten.

Vaccine Selection

IB vaccination programs in broilers involve the use of modified live vaccines. Vaccination of layers has historically involved administering a series of live vaccines and progressively increasing the aggressiveness of the route of vaccination (i.e. start with water administration and progress to fine particle spray) and strain of vaccine (highly attenuated to less attenuated). In breeders, a similar program is often followed, however, prior to onset of production, an inactivated vaccine is also administered to stimulate antibody production. Inactivated vaccines stimulate higher levels of circulating antibodies than live vaccines and would be of value in a breeder program where maternal antibody protection is needed. However, modified live vaccines provide better stimulation of cell mediation (T cell system) and elicit a superior local antibody (IgA) response as a result of local mucosal infection and thus would be of more value in protecting commercial layers.

With dozens of IBV strains having been identified around the world, choosing appropriate strains for vaccination may seem a daunting task. The immune response produced to one strain, however, often shows a significant degree of cross protection to heterologous challenge. Cross protection has been demonstrated especially for the live type vaccines. If the prevalent strains for a region have been identified, it is often possible to design a program using commercially available vaccine strains (Table 1).

Although no reasonable combination of IB vaccine strains provides full protection against all heterologous challenges, there are combinations which are broad in coverage. Once the prevalent serotypes in an area have been identified, use of modified live vaccines containing carefully chosen strains can be used to immunize broilers, layers and breeders. Additionally, polyvalent inactivated vaccines can be administered at point-of-lay to breeders. It has been demonstrated that “classical” strains of IBV can act at least as partial primers for subsequent administration of an inactivated IB vaccine containing variant and standard

strains. Inactivated IB vaccines do not stimulate local and cell-mediated immunity as effectively as modified live virus IB vaccines, however, they can provide a degree of immunity against variant strains without the risk of introducing new strains of IB into a poultry operation. Imprudent overuse of live IB vaccines results in the vaccines being the problem rather than part of the solution.

While deciding which strains to use in an IB vaccination program, the basics must not be ignored. Good vaccination practices are especially important when administering live IB vaccines. It is a relatively fragile virus and can easily be inactivated if proper vaccination procedures (e.g. protection from sunlight, removal of sanitizers from water used for mixing/administration, use of skim milk stabilizer, etc.) are not followed.

Table 1. Percent cross-protection afforded SPF white leghorn chickens vaccinated by eyedrop at 2 to 3 weeks of age with live infectious bronchitis virus (IBV) vaccines and challenged 4 weeks later with homologous and heterologous reference strains and variant field isolates. (adapted from Gelb et al, Variant Serotypes of Infectious Bronchitis Virus Isolated from Commercial and Broiler Chickens. *Avian Diseases* 35:82-87, 1991.)

Challenge Virus	VACCINE				
	Mass (Holland)	Mass (L-1) + Conn	Mass (Holland) + Ark	Mass (L-1) + Ark	Mass (Connaught) + Ark
Mass 41	84	93	87	86	100
Ark DPI	47	27	87	100	93
Conn	57	100	100	87	100
JMK	80	86	73	93	93
Holte	70	33	79	40	93
Florida	77	80	78	60	80
mean % =	69	70	84	78	93
Layer variants					
46C	27	33	47	73	40
16VT	47	20	73	60	67
33VT	60	13	80	87	60
3330	47	13	87	53	53
mean % =	45	20	71	68	55
Broiler variant					
06	93	100	80	87	80
Figures indicate percent protection — the percentage of chickens not yielding virus from tracheal swabbings collected 5 days after challenge-virus inoculation. "mean % =" represents percent protectio of an IBV vaccine against reference strain.					