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INTRODUCTION

Infectious bronchitis virus (IBV) is a coronavirus that causes an acute and highly contagious disease in chickens. The virus can cause substantial economic losses throughout the poultry industry worldwide. It can affect the upper respiratory tract and the reproductive tract, and some strains can cause nephritis (Cavanagh, 1997). IBV is the prototype species of the Coronavirus family, Gamma coronavirus genus, classified in the order Nidovirales, and it is the type species of the genus coronavirus of the domestic chicken (*Gallus gallus*) (Cavanagh and Naqi, 2003; Cook et al., 2012). Infectious bronchitis (IB) was first observed by
Schalk and Hawn (1931) in North Dakota in the United States in 2- to 3-week-old chickens. However, the nature of the infectious agent was not determined at that time and was assumed that IB was mainly a disease of young chickens. Hence, the disease was named “infectious bronchitis of young chicks.” Five years later, it was demonstrated that the causative agent of this disease is a virus, which was named IBV (Beach and Schalm, 1936). Since that initial discovery, many different serotypes, defined by neutralizing antibodies, and genetic types, based on the deduced amino acid sequence (from the nucleic acid sequence) of the spike gene, have been described around the world (El-Houadfi et al., 1986; Jackwood et al., 1997).

**EPIDEMIOLOGY OF INFECTIOUS BRONCHITIS VIRUS**

The disease was first reported in the early 1930s, and since then has been documented in all countries with an intensive poultry industry (Ignjatovic and Sapats, 2000). It has a wide geographical distribution and it was found in regions of Africa, Asia, Australia, Europe, and the United States (Khataby et al., 2016). There are several widely distributed classic and variant IBV genotypes (de Wit et al., 2011). Some IBV genotypes and serotypes are closely related to the vaccine strains while others are variants that are unique to their geographical regions (Bande et al., 2017). Generally, IBV serotypes show variations in approximately 20%—25% in their S1 glycoprotein sequences. However, the variation can sometimes be as high as 50%, which affects the cross-protection toward virus strains (Cavanagh et al., 1992). Recently, a S1-gene-based phylogenetic classification of IBV identified 6 different viral genotypes, 32 distinct lineages, and several unassigned recombinants with interlineage origin. Interestingly, the distribution and diversity of these IBV genotypes differ with geographical location (de Wit et al., 2011; Valastro et al., 2016). The global distributions of major IBV serotypes such as Mass-type, 4/91 (793B or CR88)-like, D274-like (D207, D212 or D1466, D3896), and D3128, QX-like, and Italy-02 are shown in Fig. 3.1.

**Infectious Bronchitis Virus Types in the United States**

In the United States, the first case of IB was reported in the early 1930s (Schalk and Hawn, 1931). Since then, many IBV strains have been identified. Mainly Connecticut, SE17, Delaware strains, and the most commonly isolated type of IBV is Arkansas (Ark) (Jackwood et al., 2005), and the presence of Ark-like isolates indicates that this virus continues to change (Nix et al., 2000).
The Delaware IBV variant, nominated DE072 (Gelb et al., 1997), was first reported in 1992 and found to be distributed across the Northeastern United States. Based on S1 sequence, this variant resembles the Dutch D1466 variant (Lee and Jackwood, 2001). It is not known how the D1466 variant entered the country. The variant was later found to be prevalent in Georgia. The DE072-specific vaccine was then used to control the infection with little or no success. However, the use of the DE072 vaccine probably leads to the emergence of Georgia 98 (GA98) and GA08 variants (Lee and Jackwood, 2001).

California-type viruses were first isolated in the 1990s and were designated California variant (Moore et al., 1998). In 1999 another related but unique virus designated CAL99 was reported (Mondal and Cardona, 2007; Schikora et al., 2003). Since those reports, several other unique California viruses including CA/557/03 and CA/1737/04 (Jackwood et al., 2007) have been reported, indicating that the California-type viruses continue to evolve.

In 2007 and 2008, two new IBV variants were detected Georgia and South Carolina broilers with respiratory disease, wherein Mass and Ark vaccines do not protect. The viruses were distinct from each other and designated GA07 and GA08. The molecular analysis of the glycoprotein S1 has shown that the two variants have a unique sequence of the gene S1 (de Wit et al., 2011; Jackwood, 2012). Also, the sequence analysis showed the GA07 virus to be similar to CA1737/04 and the GA08 virus to be somewhat similar to CA/557/03, suggesting possible origins (Jackwood, 2012).
Infectious Bronchitis Virus Types in South America

In South America, Mass was first isolated in Brazil (Hipolito, 1957). Later in 1986, the variant Ark emerged, causing devastations to Brazilian poultry (De Wit et al., 2011). Subsequently, 12 new Brazilian isolates were identified based on S1-gene-specific reverse transcription polymerase chain reaction (RT-PCR) and restriction fragment length polymorphism (RFLP). Interestingly, the IBVPR07 isolate, belonging to the Mass serotype, was found to have high tropism for the gonads and trachea (Montassier et al., 2008). In Mexico, several different genotypes have now been isolated which include Conn, Mass, and Ark type (Jackwood, 2012). Similarly, in 2001, new variants were identified. Of these, Max/1765/99 variant was isolated from 64% of chickens showing respiratory problems in Mexico, three new isolates were found to be similar with BL-56 earlier reported in 1996, whereas two other indigenous isolates were antigenically similar to Conn genotypes (Gelb et al., 2001). In recent years, IBVQ1 type, originally isolated in China, has been detected in Chile, Peru, Argentina, and Colombia (Sesti et al., 2014).

Infectious Bronchitis Virus Types in Europe

Until the late 1970s, it was believed that only IBVs of the Mass serotype were important causes of disease in Europe (Jones, 2010; de Wit et al., 2011). In 1980s the Doorn Institute of The Netherlands isolated four serotypes designated as D207 (also known as D274), D212 (also known as D1466), D3896, and D3128, from Mass isolate-vaccinated flocks (Davelaar et al., 1984). Many IBV variants were isolated, from other European countries including IBV PL-84084 in France (Picault et al., 1987), B1648 in Belgium (Meulemans et al., 1987), 624/I, Fa 6881/97, AZ 27/98, AZ 20/97, and BS 216/01 in Italy (Capua et al., 1994; Zanella et al., 2003), and Spanish strains of 97/314, 98/313, 00/337, and 00/338 (Dolz et al., 2006).

In the United Kingdom, 793/B (also known as 4/91 and/or CR88) was identified as the predominant serotype (Cavanagh et al., 1999). It was shown to have a nucleotide sequence in the hypervariable regions of the S1 spike gene quite distinct from Mass and Dutch variant viruses (Cavanagh et al., 2005).

In Italy, an early variant designated Italy/624I/94 was reported (Capua et al., 1994) as well as a more recent type designated Italy-02 (Italy/Italy-02/497/02). Italy-02 was found in almost all European countries but in 2004, it was reported to be declining in prevalence in all countries except Spain (Worthington et al., 2008). Because of its pathogenicity, the most significant IBV type to become widespread in Europe in recent years is the QX IBV type and so-called QX-like types (Monne et al., 2008).
Infectious Bronchitis Virus Types in Africa

In many African countries, the Mass IBV serotypes cause sporadic IB outbreaks in the commercial poultry industry. A number of local variants are reported in Africa in addition to the widely known vaccine serotypes such as Mass and 4/91 strains (de Wit et al., 2011).

In North Africa, an unusual variant with tropism to gastrointestinal system, known as IBV G strain, was isolated in Morocco in the early 1980s (El-Houadfi et al., 1986). However, recent studies identified several other local nonvaccine types, including the QX-like strains and Italy-02, originally localized in China and Europe, respectively (Bande et al., 2017).

In Tunisia, three isolates were identified by molecular test and virus neutralization (VN) test and designated TN20/00, TN200/01, and TN335/01. These isolates were found closely related to European strains including D274 and 793B (Bourogaˆa et al., 2009).

In Eastern Libya, recent studies showed the presence of 12 IBV strains that are phylogenetically classified into two distinctive clusters which were detected by RT-PCR in broiler flocks (Awad et al., 2014).

In Egypt, the variants of the IBV have been identified since the 1950s, with the isolation and identification by tests of sero-neutralization of a variant closely linked to the variant of Dutch3128 (Sheble et al., 1986). Subsequently, several related variants either to the types Mass (Egypt /Masse/F/03) or to other European variants [D274 (Egypt/D274/D/89)] or of the Variants Israelis have been identified by the genetic analysis of the IB in this country (Abdel-Moneim et al., 2006, 2012).

In Southern Africa, IBV was isolated in 1980 by Morley and Thomson (1984) and was associated with swollen head syndrome causing severe problems. It was confirmed as a variant that showed to be poorly protected by Massachusetts vaccines (Cook et al., 1999). The only other study done on the detection of the variants of IBV in sub-Saharan Africa is the report of Ducatez et al. (2009), which has characterized a new variant named “IBADAN” in Nigeria and in Niger, which was antigenically different from the other genotypes known.

FACTORS LEADING TO THE EMERGENCE OF NEW STRAINS OF INFECTIOUS BRONCHITIS VIRUS

The emergence and evolution of viral pathogens cause a major problem in the poultry industry. Mutation and recombination processes are involved in the genetic and phenotypic variations of IBV in chickens, leading to the emergence of new variant strains, and give rise to virus population diversity to be modeled by the host, particularly by the
immune system. The consequence is a continuous emergence of new IBV variants with regard to pathotypes, serotypes, and protectotypes.

IBV, like many other RNA viruses, as well as the coronaviruses, has a high error rate during the transcription of its genomes (Lai and Cavanagh, 1997), it creates genetic diversity through rapid replication and large population sizes coupled with a high mutation rate and recombination. Mutations include substitutions, which are the result of a high error rate and limited proofreading capability of the viral RNA-dependent RNA polymerase (RdRp), and insertions and deletions, caused by recombination events or by RdRp stuttering or slippage.

The viral genes encoding the spike S, replicase, and nucleocapsid proteins can be considered the main genomic regions, which indicate the evolution processes of IBV.

Investigations carried out to date have highlighted the role of three factors: (1) lack of RNA polymerase proofreading, leading to replication errors in RNA genomes with mutation at the order of $10^{-4}$; (2) interference of continuous use of live and often multiple attenuated vaccines formulated with different IBV strains; and (3) immune pressure exerted on circulating viruses by the constant presence of partially immune bird populations (Umar et al., 2016).

However, molecular studies have shown that a new serotype or variant can emerge as a result of only a few changes in the amino acid composition in the S1 part of the virus spike protein, while most of the virus genomes remain unchanged (Cavanagh, 2007). This could be due to immunological pressure caused by the widespread use of vaccines, to recombination as a consequence of mixed infections, or to a reduction of dominant serotypes as a result of vaccination, allowing other field strains to emerge (Lee, 2002; Liu et al., 2006).

Due to the high variability and important biological properties of the S1 glycoprotein, antigenic evolution in IBV has been primarily associated with changes in the sequence of the S1 glycoprotein, which contains regions associated with virus attachment to cell receptors and relevant epitopes that induce the production of neutralizing antibodies (Cavanagh et al., 1988). Therefore different serotypes, subtypes, and antigenic variants of IBV are thought to be generated by nucleotide point mutations, insertions, deletions (Kusters et al., 1987), or RNA recombination of S1 gene (Wang and Huang, 2000), resulting in IB outbreaks even in vaccinated chicken flocks.

Actually, serotypic determinants have been identified in the first 395 amino acid region of the S1 subunit, which contains three major hypervariable regions. Amino acid changes within the three S1 glycoprotein hypervariable regions determine the most relevant phenotypic changes, resulting in new serotypes and the induction of non-cross-reacting
VN antibodies (non-cross-protecting). Variants may attain increased virulence, efficient receptor binding, rapid transmission, and persistence in host system causing significant disease in vaccinated flocks of all ages (Wang and Huang, 2000; Dhama et al., 2014).

**DIAGNOSIS TECHNIQUES OF THE EMERGING STRAINS OF INFECTIOUS BRONCHITIS VIRUS**

IBV affects chickens of all ages, involving the respiratory system, also renal and reproductive systems. And because the clinical signs are not specific, the need for differential diagnostic methods was very important to realize. These methods focus on either isolating or detecting the virus itself as well as detecting serum antibodies to it.

**Sampling**

Samples appropriate to the form of IB observed must be obtained as soon as signs of clinical disease are apparent. Laryngotracheal swabs from live birds or tracheal and lung tissues from fresh carcasses from diseased birds should be collected. Also, kidney, oviduct, or proventriculus samples from birds with nephritis could be used for laboratory diagnosis of IBV. All samples should be placed in virus transport medium containing penicillin (10,000 IU/mL) and streptomycin (10 mg/mL) and kept in ice and then frozen (OIE, 2013).

**Virus Isolation**

Specific pathogen-free embryonated chicken egg (SPF-ECE) is recommended for primary isolation of IBV. Those embryonated eggs used for virus isolation should originate preferably from SPF chickens or from breeder sources that have been neither infected nor vaccinated with IBV. Suspensions of tissues (10%–20% w/v) are prepared in sterile phosphate buffered saline. After being clarified by low-speed centrifugation and filtration through bacteriological filters, 0.1–0.2 mL of sample supernatant is inoculated into the allantoic cavity of 9- to 11-day-old embryos (Delaplane, 1947). Eggs are candled daily for 7 days with mortality within the first 24 hours being considered nonspecific death. Normally, the allantoic fluids of all eggs are pooled after harvesting 3–6 days after infection; this pool is diluted 1/5 or 1/10 in antibiotic broth. Blind passage into another set of eggs for up to a total of three to four passages is conducted. The last passage is left for 7 days to screen
the presence of pathognomonic embryonic changes consisting of stunted and curled embryos with feather dystrophy (clubbing) and urate deposits in the mesonephros on the second to fourth passage. Isolation of IBV must be confirmed by serum neutralization or reverse transcription polymerase chain reaction (RT-PCR) (OIE, 2013; Delaplane, 1947).

Methods for Identification

The initial tests performed on IBV isolates are directed at eliminating other viruses from diagnostic consideration. Chorioallantoic membranes from infected eggs are collected, homogenized, and tested for avian adenovirus group 1 by immunodiffusion or PCR (OIE, 2013). Group 1 avian adenovirus infections of commercial chickens are common, and the virus often produces stunted embryos indistinguishable from IBV-infected embryos. Furthermore, IBV exerts hemagglutination (HA) activity only after phospholipase C treatment of concentrated virus-infected allanto-amniotic fluids (Bingham et al., 1975).

Genetic-based tests (RT-PCR or RT-PCR–RFLP) are used commonly to identify an isolate as IBV. Also, in situ hybridization can be used to detect viral nucleic acid (Collisson et al., 1990).

IBV can also be detected using immunofluorescence or immunoperoxidase on the tracheal or kidney section from the field isolates or on the chorioallantoic membrane from the inoculated embryos (Abdel-Moneim et al., 2009). Other techniques may be used, for example, cells present in the allantoic fluid of infected eggs may be tested for IBV antigen using fluorescent antibody tests (Clarke et al., 1972). Furthermore, serotyping of IBV isolates and strains has been done using HA inhibition (King and Hopkins, 1984). However, nonspecific reactions or lower sensitivity especially in field samples may occur (Benyeda et al., 2010), for that we needed a reliable technique as and VN tests in chick embryos (Dawson and Gough, 1971) and also as enzyme-linked immunosorbent assay. This technique is quick, inexpensive, and sensitive, which is suitable for screening a large number of samples, IBV diagnosis, and serotype identification as well (Karaca and Syed, 1993). It has been proved useful in grouping and differentiating strains of IBV (Ignjatovic et al., 1991).

Treatment and Vaccination

There is no specific antiviral therapy available to control IBV field infection, but we can reduce the effect of the complicating bacterial infections by using antimicrobial therapy. Also reduced mortalities in nephrogenic strains can be achieved by reducing the protein
concentrations in ration, providing electrolytes in drinking water, and using diuretics (Abdel-Moneim, 2017). So, the only solution we have left is vaccination.

**Live Attenuated Vaccine**

Live attenuated vaccines are the first-generation IBV vaccines used to control IBV infection in the field. Currently, live IBV vaccines are normally attenuated by multiple repeat passage of a virulent virus in SPF-ECEs (Cavanagh, 2003). However, extensive passage should be avoided to prevent the reduction in immunogenicity. There is an evidence that some attenuated vaccines showed increase in virulence after back passage in chickens (Hopkins and Yoder, 1986). These vaccines are commercially available for application via drinking water or by coarse spray at 1 day or within the first week of age. Live vaccination of 1-day-old chicks induced a rapid decline in maternally derived antibodies due to binding and partial neutralization of vaccine viruses (Mondal and Naqi, 2001). Since the duration of immunity following live attenuated vaccines is short, booster vaccination is carried out with the same or combinations of other strains, 2–3 weeks after prime vaccination (Cavanagh, 2003). Given the nature of these live attenuated vaccines, further passage beyond the master seed stock must be kept to a minimum to prevent potential loss of immunogenicity. The stocks must be grown in SPF chicken eggs to prevent the introduction of other potential pathogens (OIE, 2013).

Most of the commercially available live attenuated vaccines are derived from virulent strains such as Massachusetts-based M41 serotype and the Dutch H52 and H120 strains, although some strains with regional or local impact have been used in different parts of the world (Sasipreeyajan et al., 2012).

For logistics and economic reasons, some commercially available live attenuated IBV vaccines have been combined with other virus vaccines such as those against Newcastle disease virus, Marek’s disease virus, and infectious bursal disease virus. However, it is not clear whether the combination may influence immune response to the combined antigen (Vagnozzi et al., 2010), and if excess IBV component is present, IBV may interfere with other virus response such as NDV response (Thornton and Muskett, 1975).

Some of the limitations of live attenuated viral vaccines include reversion to virulence, tissue damage, and interference by MDA. Tissue damage due to live vaccines may lead to pathological disorders or secondary bacterial infections, especially in day-old chick (Tarpey et al., 2006). And it has been found that H52 and H120 IBV vaccines induce considerable pathology in the trachea (Zhang et al., 2010). And potential recombination between vaccine strains and virulent field strains may lead to the emergence of new IBV serotypes (Lee et al., 2010; McKinley et al., 2008).
**Inactivated or Killed Vaccines**

Inactivated or killed vaccines have been used either alone or in combination with live attenuated IBV vaccines (Finney et al., 1990). These vaccines are administered by injection to layers and breeders at point of lay (13–18 weeks of age). Inactivated IBV vaccines have poor efficacy unless the chickens have previously been primed by vaccination with a live virus vaccine (OIE, 2013). And since inactivated vaccines do not replicate, they are unlikely to revert and cause pathological effects. Being injectable, administration of killed vaccines is either difficult or impracticable in large poultry setting. Likewise, issues of injection-site reactions may also lead to carcass rejection or reduction in value (Cook et al., 2012).

**Recombinant Vaccines**

Recombinant DNA vaccines have been enhanced to target multiple serotypes and their efficacy has been improved using delivery vectors, nano-adjuvants, and in ovo vaccination approaches. Although most recombinant IB DNA vaccines are yet to be licensed, it is expected that these types of vaccines may hold sway as future vaccines for inducing a cross-protection against multiple IBV serotypes (Jackwood, 1999).

**CONCLUSION**

Even with spending huge amounts of money to control IB, outbreaks involving classical and newly emerging virus serotypes are constantly reported. Though live attenuated vaccines are still common in the field, these vaccines provide only a little or partial cross-protection occurs between vaccine strains and new field which require the development of new vaccines as the recombinant vaccines. There is no doubt that newer generation vaccines such as the recombinant vector DNA vaccines, plasmid DNA vaccines, and multiepitope vaccines may stand as future alternatives as these vaccines have potential to deliver numerous antigens, thus producing broad-based antibody and cell-mediated immune response against numerous serotypes and also, the requirement of new strategies for vaccination.

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