



# Infectious Bursal Disease: An Important Challenge in Transforming the Ethiopian Poultry Sector

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## ABSTRACT

Infectious Bursal Disease (IBD) is an acute, highly contagious, immunosuppressive viral disease of young chickens; IBDV predominantly targets B-cells residing in the Bursa of Fabricius (BF) and results in bursal edema, atrophy, and abrogation of antibody responses. Significantly, IBD-induced immunosuppression increases the occurrences of other diseases caused by opportunistic pathogens and, thus, prevents young chickens from responding optimally to vaccines. Owing to its widespread nature, high morbidity and mortality rates, IBD has been given a considerable economic importance both at the international and national levels and is of growing scientific interest. Most of the economic losses associated with IBD are due to its immunosuppressive effects that eventually lead to poor vaccination response, secondary bacterial, viral and protozoan infections and poor performance. As a result, the subclinical and immunosuppressive form of IBD probably is the commonest form that occurs among these birds. There are different reports and complains of disease outbreak from different parts of Ethiopia and many small scale poultry farms are closed due to Gumboro outbreak. Therefore, control of Gumboro disease requires a balanced approach including strict biosecurity measures and rational vaccination schedules to reduce the incidence and impact of IBD in Ethiopian poultry industry.

## INTRODUCTION

Ethiopia has the highest number of livestock population in Africa; the poultry population being 56, 866,719 of which according to the CSA (2014/15) Indigenous 54,510,523(95.86%); Exotic 770,052 (1.35%); Hybrid 1,586,144 (2.79) unfortunately however, the contribution of exotic poultry to the Ethiopian economy is significantly lower than that of other African countries (FAO, 2007). Therefore, intensification and upgrading of the potential of birds will be inevitable to provide surplus products (Hailemariam *et al.*, 2006). However the industry is confronted with a variety of problems, particularly the diseases of viral origin.

Infectious bursal disease (IBD) and Newcastle disease (NCD) have remained as two most important infectious diseases threatening the village chicken and commercial poultry production in most parts of the world (El-Yuguda *et al.*, 2005; El-Yuguda *et al.*, 2009). In Ethiopia, accompanying the intensification of poultry farming; constraints associated with the prevalence of infectious diseases are challenging factors. Among these, infectious bursal disease is the one that become to cause frequent outbreaks and a serious threat and a challenge to the juvenile poultry industry in Ethiopia (Mazengia, 2012).

Infectious Bursal Disease is an acute and highly contagious disease affecting young chickens from 3-6 weeks of age. The disease causes immunosuppression in chickens and rendering them vulnerable to a variety of other infections. Accordingly, chick 1-14 days of age is less sensitive to respond to immunization and, in addition, maternal antibodies usually protect them from any attack by the virus during this period. Birds older than 6 weeks rarely develop sign of disease although they produce antibodies to the virus. After an incubation period of 2-3 days, chicks show clinical signs of distress, depression, ruffled feather, anorexia, diarrhea, trembling and dehydration; usually 30% of them die. The clinical disease lasts for 3-4 days, after which surviving birds recover rapidly. IBDV is excreted in the feces for 2-14 days. On the other hand, when IBDV is newly introduced into a flock, morbidity approaches 100% and mortality may reach as high as 90%. In broilers, immunosuppression is denoted by a high prevalence of viral respiratory infections and elevated mortality due to airsacculitis and colisepticemia during the terminal third of the 6–8-week growing cycle (Ikuta *et al.*, 2001).

Infectious bursal disease (IBD) was first recognized as a distinct clinical entity in 1957 (Cosgrove, 1962). Cosgrove initially described the malady as “avian nephrosis” on account of the tubular degenerative lesions found in the kidneys of infected broiler chickens. The syndrome adopted the name “Gumboro disease” since the first outbreaks occurred in and around the area of Gumboro, Delaware, USA. The first confirmed acute outbreak of IBD in Ethiopia was reported in the months of March and April, 2002 in Commercial poultry farms, Debre Zeit. Since then, unconfirmed post-vaccination

outbreaks have been observed and many farms are closed in different areas of the country and becoming a threat to the poultry industry and no further investigation was carried out to know the nature of the virus and the distribution of the disease (Zelege *et al.*, 2002).

Small and large scale chicken farms are rapidly growing in Ethiopia. The chicken strains imported are temperate breeds that are less adapted to the heat stress and disease challenges in the country. Accompanying intensification of poultry farming, there is occurrence of epidemics of newly introduced diseases and/or epidemics of endemic diseases. One of the diseases that is of growing concern in poultry is Infectious Bursal disease (Gumboro disease). There are different reports and complains of disease outbreak from different parts of the country and many small scale poultry farms are closed. Although vaccination is given, the disease outbreak is common. Gumboro is the worst and number one problem which causes high mortality by concurrent diseases. Despite the fact that IBD incidences are increasing at alarming rate all over the country, little have been known about it to devise effective control strategies (Zelege, 2005). Therefore, the objective of this paper is to review the current status of Infectious Bursal Disease in Ethiopia.

## Etiology

Infectious bursal disease virus (IBDV) is an etiology of infectious bursal disease “Gumboro disease”, (Mahgoub, 2012; Muller *et al.*, 2012), which belongs to a genus Avibirnavirus (Fauquet *et al.*, 2005), of family Birnaviridae (Delmas, 2011). It is a double strand an RNA virus (dsRNA) virus (Etteradossi and Saif, 2008) and a non-enveloped, icosahedral capsid with bisegmented genome (Wu *et al.*, 2007; Zhu *et al.*, 2008). The larger segment, A, is 3261 nucleotides long and contains two open reading frames (ORF) and encodes four viral proteins designated as VP2, VP3, VP4 and VP5 and also the smaller segment B encodes only VP1 which has polymerase activity (Van den Berg, 2000; Lukert and Saif, 2003). The two viral proteins, VP2 and VP3 are structural proteins which form the viral capsid. The epitopes responsible for the induction of neutralizing and protective antibodies are located on the VP2 protein (Abdel *et al.*, 2001).

## Epidemiology

### *Host range*

Clinical disease occurs solely in chickens but Turkeys, ducks, and ostriches can be naturally and experimentally infected with IBDV serotypes I and II, as evidenced by serological response and isolation; however, the infections are apathogenic. Several other avian species including rooks, wild pheasants, crows, gulls, and falcons, were reported to be susceptible to infection or to possess antibodies against IBDV. Serotype I viruses

affect every breed of chicken, but the most severe clinical signs and lesions and the highest mortality rate have been observed in white leghorns (Etteradossi and Saif, 2008). In fully susceptible flocks, mortality associated with classic strain infections may range from 1-60%, with high morbidity of up to 100% (Muller *et al.*, 2003; Etteradossi and Saif, 2008). In contrast, vvIBDV strains cause mortality of 50-60% in laying hens, 25-30% in broilers, and 90-100% in susceptible SPF leghorns (Van den Berg *et al.*, 2000). According to (Schat and Xing, 2000; Jarosinski *et al.*, 2005; Asif *et al.*, 2007) reports the induction of a high mortality rate after IBDV infection of susceptible chickens with virulent strains correlated with the ability of the bird to mount a rapid systemic cytokine-mediated immune response, which may lead to a shock-like syndrome followed by death.

#### *Physico-chemical nature of the virus*

The virus is non-enveloped and quite resistant to physical and chemical agents, resistant to: pH conditions of 2–11, but it is inactivated at pH 12 (Lukert and Saif, 2003) due to this ability of stability and hardness, it persists in poultry premises even after thorough cleaning and disinfection (Lukert and Saif, 2003), for up to 4 weeks in the bone marrow of infected chickens (Elankumaran *et al.*, 2002). The virus has been shown to remain infectious for 122 days in a chicken house, and for 52 days in feed, water and faeces (Benton *et al.*, 1967).

#### *Route excretion and transmission of the virus*

Infected birds excrete virus in their dropping at least for 14 days (Baxendale, 2002). It is excreted in the faeces and then contaminates water, feed and litter, where it persists and from where it commonly spreads. The most common mode of infection is through the oral route, Conjunctival and respiratory routes may also be involved (Sharma *et al.*, 2000) but the virus is highly contagious so that then disease is transmitted by direct contact with excreting subjects, or by indirect contact with any inanimate or animate (farm staff, animals) contaminated vectors between infected and susceptible flocks (OIE, 2008). The high tenacity of the virus and its resistance to several disinfections and virucidal procedures may contribute to the rapid distribution of the virus (Van den berg *et al.*, 2000; Garriga *et al.*, 2006). IBDV may spread through contaminated equipment (Flensburg *et al.*, 2002; Jackwood and Sommer-Wagner, 2010). There is no evidence to suggest that IBDV is spread via transovarial transmission (Etteradossi and Saif, 2008). No specific vectors or reservoirs of IBDV have been established, but the virus has been isolated from mosquitos (*Aedes vexans*), rats, and lesser mealworms (*Alphitobius diaperinus*) (Etteradossi and Saif, 2008). Viable vvIBD virus was recovered after 2 days from the faeces of a dog that had been fed tissues from experimentally infected chickens, indicating that dogs may act as mechanical vectors for the virus (Pages-Mante *et al.*, 2004).

## **Pathogenesis and pathological lesions**

Following host entry via oral ingestion or inhalation, IBDV may bind to host cell proteins such as N-glycosylated polypeptide(s) expressed on the cell membrane of immature IgM+ B-cells during viral entry process (Luo *et al.*, 2010). A pore forming peptide of the virus (pep46), which is associated with the outer capsid of the IBDV particle, may facilitate viral entry into the cytoplasm of infected cells (Galloux *et al.*, 2007; Galloux *et al.*, 2010). A lipid draft mediated endocytic mechanism was suggested based on the results of an in vitro study to support entry of attenuated IBDV to the cells (Yip *et al.*, 2012).

Generally, the sequellae of IBDV infections such as severity of clinical signs, organ lesions and immunosuppression correlate with the status of immunity, age and genetic background of affected chickens and with the virulence of the infecting virus strain (Van den Berg, 2000). SPF chickens infected with vvIBDV develop an earlier onset of mortality and more severe bursal lesions compared to broiler chickens with MAB and vaccinated chickens (Aricibasi *et al.*, 2010). A massive mast cell influx detected in the bursa of SPF chickens infected with vvIBDV may aggravate bursal lesions as typical indicators of acute hypersensitivity responses were observed in the bursa of such chickens (Wang *et al.*, 2008; Wang *et al.*, 2012a). These cytokine mediated bursal lesions may result in an early onset of severe immunosuppression in younger chickens (Rautenschlein *et al.*, 2007). Highly virulent virus strains could also cause depletion of lymphoid cells in the thymus, spleen and bone marrow (Corley *et al.*, 2001). In long standing cases, there is an increased connective tissue mass in the interfollicular areas replacing the depleted lymphoid tissues (Sharma *et al.*, 2000; Negash, 2004).

According to the virus virulence and pathogenicity, IBD cause more severe or less severe lesions on the bursa of Fabricius and other organs such as: spleen, thymus and kidneys, and may induce immunosuppression and mortality in birds (Sharma, 2000; Van den berg, 2004; Etteradossi and Saif, 2008). Macroscopic lesions are observed principally in the bursa which presents all stages of inflammation following acute infection (Muller, 2003). Autopsies performed on birds that died during the acute phase (three to four days following infection) the bursa reveal initially hypertrophic, oedematous and haemorrhagic and its colour turns from white to cream and a yellow transudate covers its serosa early in infection. The most severe cases are characterized by a major infection of the mucous membrane and a serous transudate, giving the bursal surface a yellowish colour and often accompanied by petechiae and haemorrhages. By the fifth day, the bursa reverts to normal size and by the eighth day becomes atrophied to less than a third of the normal size. Moreover, in the acute form of the disease caused by hyper virulent strains, macroscopic lesions may also be observed in other lymphoid organs (thymus, spleen,

caecal tonsils, Harderian glands, and Peyer's patches) (Etteradossi and Saif, 2008).

Necropsy examination will usually show changes in the bursa of Fabricius such as swelling, oedema, haemorrhage, the presence of a jelly serosa transudate and eventually, bursal atrophy. Pathological changes, especially haemorrhages, may also be seen in the skeletal muscle, intestines, kidney and spleen; however, definitive diagnosis can only be achieved by the isolation and/or specific detection and characterization of IBDV (OIE, 2012).

### Clinical findings of the disease

In infectious bursal disease virus infection severity of clinical signs and immunosuppression correlate with the status of immunity, age and genetic background of affected chickens and with the virulence of the infecting virus strain (Van den Berg, 2000). IBD occurs in both layer and broiler birds and although it has been found in turkeys (Etteradossi and Saif, 2008). The age of maximum susceptibility is between three and six weeks (Muller, 2003) corresponding to the period of maximum bursa development, during which the acute clinical signs are observed. Infections occurring prior to the age of three weeks are generally subclinical and immunosuppressive. Some studies have shown that age of infection is directly related to the degree of immunosuppression, (Ivanyi and Morris., 1976) demonstrated that no immunosuppressive response after 3 weeks of infection despite the manifestation of a clinical disease.

Variant IBDV strains do not produce overt clinical signs, but cause immunosuppression, which is the most significant economic losses, result from subclinical infections and may cause mortality due to secondary opportunistic infections in immune compromised birds (Van den Berg *et al.*, 2000; Rodriguez, 2002; Etteradossi and Saif, 2008). In contrast, vvIBDV strains cause mortality of 50-60% in laying hens, 25-30% in broilers, and 90-100% in susceptible SPF leghorns (Van den Berg, 2000). In fully susceptible flocks, mortality associated with classic strain infections may range from 1-60%, with high morbidity of up to 100% (Van den Berg, 2000; Muller, 2003).

The disease has an acute and per acute course. The incubation period is very short 2-3 days. However, (OIE, 2004) recommends an incubation period of 7 days for regulatory purposes. Virus excretion can begin as early as 24 hours after infection. Mortality will peak and recede usually in a period of 5-7 days (OIE, 2012). Accompanying symptoms include the disease has been described worldwide (Van den Berg *et al.*, 2000) as acute onset of depression, trembling, white watery diarrhea, ruffled feathers, severe prostration, vent picking, vent feathers soiled with urates, anorexia, dehydration, and elevated water consumption.

### Laboratory Diagnosis

Clinical disease due to infection with the IBDV can usually be diagnosed by a combination of characteristic signs and post-mortem lesions. Laboratory confirmation of disease, or detection of subclinical infection, can be carried out by demonstration of a humoral immune response in unvaccinated chickens or by detecting the presence of viral antigen or viral genome in tissues. In the absence of such tests, histological examination of bursa may be helpful. Antigen-capture enzyme-linked immunosorbent assays (ELISAs) based on plates coated with IBDV-specific antibodies have also been described for the demonstration of IBDV antigens in bursal homogenates. The reverse transcription polymerase chain reaction (RT-PCR) with specific primers may be used to detect viral genomic RNA in the bursa of Fabricius (OIE, 2012). Generally a preliminary diagnosis can usually be made based on flock history, clinical signs and post-mortem (necropsy) examinations.

#### *Serological diagnosis.*

Serological tests such as AGID, ELISA, and VNT for detecting antibodies are used for monitoring vaccine responses and might be additional information for diagnosis of infection of unvaccinated flocks (OIE, 212). The enzyme linked immuno sorbent assay (ELISA) is the most commonly used test for the detection and quantification of IBDV antibodies to check response to vaccination, natural field exposure and decay of maternal antibody titer (Lukert and Saif, 2003). It is economical, simple, and quick tests a large number of samples at the same time and is adaptive to automation to computer software (Lukert and Saif, 2003). Viral antigens can be demonstrated by the agar-gel precipitin assay or by the antigen-capture enzyme-linked immunosorbent assay (AC-ELISA) (Islam *et al.*, 2001a). The VN titers accurately correlate with protection of chickens against IBDV (Knoblich *et al.*, 2000). Differentiation of classic and variant strains has been made by using ELISA and monoclonal antibodies (Sapats *et al.*, 2005). However, these methods may not be as rapid and sensitive as molecular methods (Jackwood, 2004).

#### *Virological diagnosis*

Infectious bursal disease virus may be detected in the bursa of Fabricius of chicks in the acute phase of infection, ideally within the first three days following the appearance of clinical signs (OIE, 2012). The viral antigens specific to IBDV may be detected by direct and indirect immunofluorescence (Abdel-Alim and Saif, 2001) or by immuno peroxidase staining (Cho *et al.*, 1987) in the bursal follicles of infected chickens between the fourth and sixth day after infection, Immunofluorescence (Macdonald, 1980) and electron microscopy (McNulty *et al.*, 1979) of the infected cell culture or embryonated eggs are valuable tools for monitoring the growth of IBDV particularly those strains lacking pronounced cytopathic effect (CPE).

### Embryo inoculation

The inoculation of bursal homogenates from IBDV infected chickens per the chorioallantoic membrane of 9-10 days old embryonated SPF (Specific-pathogen-free) chicken eggs is the most sensitive diagnostic method for virus isolation. The most sensitive route of inoculation is the CAM; the yolk sac route is also practicable (OIE, 2012). It is important especially for Wild-type IBDV, usually not replicating in conventional cell culture, can also be regenerated by the reverse genetics approach, but can grow in embryonated chicken eggs (Brandt *et al.*, 2001; Islam *et al.*, 2001c). Some strains grow well in embryos (Brandt *et al.*, 2001; Islam *et al.*, 2001c) but are not readily adapted to grow in CEF (Chicken embryo fibroblasts) or CEK (Chicken embryo kidney) (Lee and Lukert, 1986). Variant viruses however, do not kill the embryos but cause embryo stunting, discoloration, splenomegaly and hepatic necrosis (Lukert and Saif, 2003).

### Cell culture

A filtered homogenate of the bursa of Fabricius is inoculated in nine- to eleven-day-old embryonated eggs originating from hens free of anti-IBDV antibodies. Primary cell cultures of CEF, bursa (CEB) and CEK have been used to propagate the virus (Boot *et al.*, 2000). The cloacal bursa and spleen are used for the isolation of the virus (Lukert and Saif, 2003; Muller *et al.*, 2003). The virus can be found in other organs such as the thymus, liver and bone marrow but in significantly low quantities than in the bursa (Etteradossi and Saif, 2008; Elankumaran *et al.*, 2002; Kabell *et al.*, 2005). The inoculum for virus isolation is prepared by homogenizing the tissue sample in antibiotic containing buffer (PBS) that is centrifuged to remove larger tissue particles and is used for inoculating embryonated eggs and tissue culture (Lukert and Saif., 2003).

### Molecular characterization

The classical methods for molecular characterization and differentiation of IBDV field isolates include reverse transcriptase polymerase chain reaction (RT-PCR) and restriction fragment length polymorphism (RFLP), nucleotide sequence analysis, and quantitative real time RT-PCR (qRT-PCR) (Jackwood, 2004; Wu *et al.*, 2007a). Nowadays, reverse transcription-polymerase chain reaction (RT-PCR) is a molecular tool frequently applied in IBDV diagnosis. RT-PCR in combination with restriction enzyme analysis allows the rapid identification of vvIBDV (Zierenberg *et al.*, 2001). Nucleotide sequencing of RT-PCR products is widely used for further characterization of IBDV strains (Zierenberg *et al.*, 2000; Islam *et al.*, 2001a). The VP2 gene of IBDV contains variable region which suggests the potential of this region for differentiation of IBDV strains (Jackwood, 2004). RT-PCR followed by digestion with multiple restriction enzymes or RFLP (Zierenberg *et al.*, 2001) and nucleotide sequencing of VP2 gene (Lin *et al.*, 1993) have been used for differentiation of IBDV strains.

The molecular differentiation of IBDV strains using VP2 has been improved by use of labelled probes in real-time RT-PCR (Jackwood and Sommer, 2005). In

recent years detection of nucleotide variation has been facilitated by application of melt curve analysis. A TaqMan qRT-PCR and melting curve analysis can be used to trace mutations in the hVP2 region (Jackwood *et al.*, 2003) this method allows comparing sequences between field and vaccinal strains (Jackwood and Sommer., 2002; Gao *et al.*, 2007). It determines a single nucleotide polymorphism in VP2 (Wu *et al.*, 2007a). Genetic typing according VP2 sequence of IBDV has been widely used as a means of tracing the spread of IBDV and virulence change (Jackwood and Sommer-Wagner., 2007).

### Treatment

No therapeutic treatment has been found to have an effect on the course of the viral infection; however birds may be helped with drugs to treat symptoms so as to control secondary agents and the effects of immunosuppression (Muller, 2003).

### Prevention and Control

Infectious bursal disease virus is both highly contagious and very resistant to inactivation, which accounts for its persistent survival on poultry farms, despite disinfection (Van den Berg *et al.*, 2000; Etteradossi and Saif, 2008) so that it requires strict hygienic and managerial practice. Therefore, even with strict biosecurity programs (e.g. 'down time' between broods, all-in/all-out production, cleaning and disinfection of the premises and equipment) is vital for prevention of IBDV infection but also vaccination is especially important to reduce the incidence and impact of IBD in the poultry industry (Van den Berg *et al.*, 2000; Etteradossi and Saif, 2008). Rational vaccination schedules and strict biosecurity measures were indicated in many reports as essential tools for the control of IBD (Farooq *et al.*, 2003). Immunization of chickens with high quality vaccines is the primary method of control of many poultry infectious diseases; However IBDV is resistant to a large variety of disinfectants and is environmentally very stable but mainly controlled by vaccination (Van den Berg, 2000; Muller *et al.*, 2003; Dacic *et al.*, 2008) with a proper vaccination schedule. Vaccines developed against the Gumboro disease were effective for about 25 years. The first vaccines to prevent IBD in broilers and replacement pullets were prepared by adaptation of field isolates in embryonated eggs (Edgar and Cho, 1965; Edgar and Cho 1973).

### Economic Importance of IBD

The economic impact of IBD in fowl is serious and influenced by strain of virus, susceptibility and breed of flock; inter current primary and secondary pathogens, and environmental and managerial factors. Clinical IBDV leads to direct losses due to high mortality, in addition, condemnation of carcasses due to skeletal muscle, thigh and pectoral muscle haemorrhages can be

an important cause of economic losses (McFerran, 1993, van den berg, 2004). Indirect losses in Gumboro disease arise due to the severe immunosuppression of broilers and egg laying hens and their increased predisposition for other diseases and vaccination failure (Van den berg, 2004). Thereby, as a consequence, they result delayed growth, reduced weight gain, greater food conversion, longer fattening, lesser production values, increased mortality and lower quality of products observed (Sharma, 2000). The occurrence of vvIBDV has increased the economic importance of the disease. Until 1987, the strains of the virus were of low virulence, causing less than 2% mortality, and vaccination was able to satisfactorily control the disease. However, the occurrence of vvIBDV has led to vaccination failures, and increased mortality and morbidity (van den Berg, 2000). In 80% of the OIE member countries, acute clinical disease due to IBDV has been reported (van den Berg, 2000).

The presence of disease may also limit opportunities in the market place, either locally or internationally, and hinder the adoption of improved technologies, be they improved breeds, better management systems or more efficient processing and marketing methodologies. There would be further loss of income for an extended period because of the stamping-out policy. The disruption to the flow of product and decreased production may cause job losses on farms and in service and associated industries, depending on the time it takes to bring the outbreak under control. Even a small outbreak would result in dislocation of the industry and its normal marketing patterns. An uncontrolled outbreak would markedly increase production costs because of the impact of the disease and the need for continuing control measures (Van den berg, 2004).

### The status of IBD in Ethiopia

In Ethiopia IBD is prevalent in various areas (Zelege *et al.*, 2005) causing high mortality ranging from 49.89% to 72% in chicken (Woldemariam and Wossene, 2007; Tesfaheywet and Getnet, 2012). Infectious Bursal Disease is a newly emerging disease of chicken in Ethiopia, as described by Zelege *et al.*, (2005) the disease has been speculated to be introduced concurrent with the increased number of commercial state and private poultry farms flourishing in the country. Research and case reports coming from various regions of the country indicated that viral diseases are posing a growing threat to the young poultry industry flourishing in the country (Alamargot, 1987; Zelege *et al.*, 2002; Zelege *et al.*, 2005; Woldemariam and Wossene, 2007; Mazengia *et al.*, 2009). Therefore infectious diseases like IBD are becoming real threats to chicken production (Alamargot 1987; Zelege *et al.*, 2005). Frequent outbreaks and occurrence of new strains of infectious bursal disease became a challenge to the juvenile poultry industry in Ethiopia (Mazengia, 2012). Over the past few years, 25 to 75% of the deaths/losses in exotic

and cross chickens have been associated with infectious bursal disease (Zelege *et al.*, 2002; Woldemariam and Wossene, 2007).

Gumboro disease was first reported in 2002 in Ethiopia at privately owned commercial poultry farm in which 45-50% mortality rate was documented and diagnosed first in commercial poultry (Zelege *et al.*, 2005b) and thereafter in a government-owned poultry multiplication center (Woldemariam and Wossene, 2007) and a commercial broiler farm (Chanie *et al.*, 2009) with serological tests. In addition to the above different serological studies molecular characterization of the Ethiopian IBD virus isolates was done for the first time in 2005 from the samples collected from Kombolcha Poultry Multiplication Center, and in commercial and breeding poultry farms in Ethiopia between 2009 and 2011 (Jenbreie *et al.*, 2012). In both cases the samples were processed at the National Veterinary Institute, Ethiopia, for virus isolation using chicken fibroblast cell culture, and the positive isolates were submitted to OIE IBD Reference Laboratory, AFSSA, France, for further antigenic and genomic characterization, and were identified as virulent classical viruses and very virulent IBD virus. In all cases the situation of the disease at small scale commercial flocks, and back yard poultry farms indicate the disease is widely distributed in the country, Moreover, chicken traders also suffer from huge financial losses due to IBDV mortality in chicken, particularly those who buy young aged chicken and rear them for several weeks after purchase (Zelege *et al.*, 2005).

Prior to February 2006, the health measures at the government owned poultry multiplication and distribution centres with the exception of Bonga and Bedelle, all the centres were devastated by the outbreak of Infectious Bursal Disease i.e. Gumboro disease (Yilma, 2007) and a commercial broiler farm (Chanie *et al.*, 2009). On the other hand most of the researchers (Degefu *et al.*, 2010, Mazengia *et al.*, 2012) in Ethiopia performed serological surveyed in different parts of the country and documented results indicates that IBD is a threat on both backyard chickens and commercial chickens. Currently, IBD is the most important threat to poultry production in the country (Zelege *et al.*, 2005) and widely distributed in all regions in the backyard chickens, commercial farms and poultry multiplication centres. The disease has since spread to all investigated commercial farms and multiplication centres occurring at an average outbreak rate of 3-4 farms per year.

The studies summarized above indicates the presence of the disease in many parts of the country and is despite the fact that IBD incidences are increasing at alarming rate all over the country where commercial poultry production is intensified and even in the backyard chickens, and also a serious problem for the poultry industry of Ethiopia. This disease has incurred considerable economic loss to the country and has been posing a challenge especially for the success of vaccines used at this time (Wit and Baxendale, 2004). A good example is failure of NCD vaccination in areas

where there is no integrated approach for the control of IBD (Wit and Baxendale, 2004; Woldemariam and Wossene, 2007). On top of this, most control strategies designed in the country do not take into consideration the local chickens, and this may lead into the failure of most strategies (Tadelle and Ogle, 2001; Hailemariam *et al.*, 2006). Considering the significant economic losses associated with IBDV, the development and evaluation of new generation IBDV vaccine are important to minimize the effects of these agents and design suitable preventive and control measures this tendency of growing poultry industry (Mazengia, 2012).

## CONCLUSION AND RECOMMENDATIONS

Infectious Bursal Disease (IBD) is an acute and highly contagious disease affecting young chickens from 3-6 weeks of age. The disease causes immunosuppression in chickens and rendering them vulnerable to a variety of other infections. Early subclinical infections are the most important form of the disease because of economic losses. They cause severe, long-lasting immunosuppression due to destruction of immature lymphocytes in the bursa of Fabricius, thymus, and spleen.

Infectious bursal disease is one of the important diseases of poultry with huge loss in young flock. It continues to be a major problem of chickens in poultry rearing areas. It has been documented in Ethiopia before one decade and considered to be endemic in many parts of the country. The high morbidity and mortality of the IBD outbreak observed in a particular study on chicken flocks in Debre zeit and other outbreaks observed in the country could be a potential indicator for the presence of acute IBD in Ethiopia, which might be due to the newly emerged strain of vvIBDV. IBD Vaccines are widely used to control IBD but there are, still, post-vaccination outbreaks. The more frequent reason for outbreaks in vaccinated flocks could be incorrect application of the vaccine. Although first observed about ten years ago, IBD continues to pose an important threat to the growing commercial poultry industry in the country.

- As the disease established itself and became endemic in our country, molecular characterization of the field strains of the virus involved in outbreaks for the subsequent production of effective vaccine to the targeted pathogens should be initiated.
- Appropriately staffed and equipped diagnostic laboratories should be established for timely diagnosis of poultry diseases and accurate titration of level of maternal antibodies.
- Efforts should be initiated to bring the prevalence of IBD as low as possible by adoption of authentic vaccination schedules in many intensive poultry farms.
- Better hygienic and strict bio-security measures should be applied for successfully overcoming the

predisposing factors which act as conducive media for the emergence of outbreaks of IBD.

- Molecular epidemiology of IBDV virus should be studied with a planned interval to assess the antigenic diversity of the IBDV virus.

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