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Review

Review on major viral diseases of chickens reported in Ethiopia

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In Ethiopia, the major poultry products come from backyards chickens. But in recent times, more commercialized poultry farms are flourishing having considerable contribution to the supply of poultry products, especially to urban areas. There are also attempts to upgrade the productivity of local chickens through distribution of exotic and cross breeds to the rural areas. These endeavors, however, are hampered from providing the expected benefits due to various constraints, among which viral diseases are of greater concern. Some of the viral diseases are thought to be introduced concurrent with intensification of poultry industry. In addition, the growing numbers of exotic flocks in the backyard system increases the number of birds which are at risk of getting infected with pathogens in the environment. The present review article deals with major viral diseases of chickens, their current status and future challenges to the poultry industry in Ethiopia. Among these, Newcastle disease, infectious bursal disease and Marek's disease become serious threats to poultry production. Due to limited research activities, the epidemiology and the total economic damage caused by this disease are not fully known. Frequent outbreaks and occurrence of new strains for these viral diseases became a challenge to the juvenile poultry industry in Ethiopia.

Key words: Chickens, Ethiopia, infectious bursal disease, Marek's disease, Newcastle disease, viral diseases.

INTRODUCTION

Rural poultry production plays a major role in the economy particularly of developing countries (Sonaiya, 1990). The larger proportion of rural poultry in the national flock population of developing countries makes them worth paying attention to improved management and breeding. Horst (1989) reported that about 80% of the poultry population in Africa is kept in backyard production system. For example, rural poultry constitutes about 99, 88, 80, and 86% of the national poultry production of Ethiopia, Uganda, Tanzania, and Nigeria, respectively (Alamargot, 1987; Kitalyi, 1997). In village systems, farmers keep poultry for diverse objectives. They are raised for purposes of hatching, sale, and home consumption, sacrifices (healing ceremonies) and gifts (Alemu, 1995). Village chicken products are often the only source of animal protein for poor households. Eggs are the source of high quality protein for sick and malnourished children (Horst, 1989; Kitalyi, 1997). About 20% of the eggs produced from backyard chickens are eaten or sold (Spradbrow, 1995; Tadelle, 1996). In most instances after some eggs are retained for hatching, the household consumes a small proportion but most are sold to supplement income (Alemu, 1995; Tadelle, 1996). Meat and eggs are not plentiful in villages and eating meat is undoubtedly a sign of wealth (Veluw, 1985). This simply indicates that income generation from village poultry is more important than consumption for the farmers (Alemu, 1995).

In Ethiopia, village chickens have been reared for a long time for similar purposes. They have contributed to the country's economy. This is not because they are productive but are huge in number (Alamargot, 1987). Constraints which restrict the potential of village chickens in Ethiopia include; low inputs of feeding, poor management, the presence of diseases of various natures and lack of appropriate selection and breeding practices (Alemu, 1995; Ashenafi, 2000; Tadelle and Ogle, 2001). In recent years, however, attempts are underway to enhance poultry productivity and optimize the contribution of chickens to the national economy.

Greater efforts have been made to transform the production system into a more commercialized and intensive large-scale system (Ashenafi, 2000). In addition, exotic breeds and cross-breeds are multiplied in government owned poultry farms and distributed to individual farmers via the extension division of the Bureau of Agriculture and Rural Development to be maintained and produced under the backyard management system. This is thought to improve the livelihood and nutrition of poor farmers and further to contribute to the national economy at large (Tadelle and Ogle, 2001). Accordingly, the Bureau of the Amhara Agriculture and Rural Development (BoARD) schemed poultry development strategy starting from 2003.

The main purpose of the strategy was to enable farmers to generate income through rearing day-old chickens of two breeds Rhode Isle land Red (RIR) and Lohmann White breeds (LOH) which are hatched and distributed from poultry multiplication centers located at Andasa and Kombolcha. However, it is becoming a growing concern that there is introduction of diseases of various etiologies into several poultry farms concurrent with importation of exotic breeds to backyard chickens. Furthermore, intensification is aggravating the rapid spread of the prevailing infectious diseases between and within poultry farms. And the distribution of these exotic breeds to farmers is creating a great treat to the indigenous backyard chickens (Alamargot, 1987; Zeleke et al., 2005a). Among these threats viral diseases like Newcastle disease (ND), Marek's Disease and infectious bursal disease (IBD) are the major health constraints inflicting heavy losses (Alamargot, 1987; Alemu, 1995; Ashenafi, 2000; Tadelle and Ogle, 2001; Zeleke et al., 2005a, b). Therefore, the objectives of this review article are:

1) To highlight the spread and pathogenesis of major viral diseases of chickens;
2) To compile the distribution and impact of viral diseases of chickens reported in Ethiopia;
3) To indicate the direction of research and development endeavor with regard to the major viral diseases of chickens in Ethiopia.

MAJOR VIRAL DISEASES OF CHICKENS IN ETHIOPIA

Newcastle disease

Newcastle disease (ND) is a highly contagious and the most dreaded disease of chickens, turkeys and many other birds (Hanson, 1978; Nonnewitz, 1986; Chuahan and Roy, 1998; Alexander and Jones, 2001). It is characterized by the lesions in the respiratory tract, visceral organs and brain and causes moderate to severe mortality and morbidity in susceptible flocks. The disease was first reported for the first time on the island of Java, Indonesia (Kraneveld, 1926) and Newcastle up on Tyne, England (Doyle, 1927). It is from this town that the disease has got its name. According to Spradbrow (1987) and Alexander (1988) the disease was recognized in other parts of Asia (Korea, Philippines, India) in the same and subsequent years. However, several reports exist in the literature of disease outbreaks, which predated the year 1926. Hanson (1978) and Alexander (1988) reviewing the literature, stated that ND first occurred in and around sea ports, apparently as a result of commercial activities by sea, and then spread to the interior of the countries much later.

Newcastle disease (ND) is caused by a group of closely related viruses that form the avian *Paramyxovirus* type 1 (APMV-1) serotype. Some serological relationships have been demonstrated between Newcastle disease virus (NDV) and other *Paramyxovirus* serotypes, the most significant being that with viruses of APMV-3 serotype. For many years NDV strains and isolates were considered to form a serologically homogenous group and this has been the basis of vaccination procedures employed for prophylaxis in most countries (Al-Garib, 2003). However, more specific serological techniques most notably monoclonal antibody based serology, have shown the existence of considerable antigenic variation between the different strains of NDV. Based on the disease produced in chickens under laboratory conditions, NDVs have been placed into five pathotypes; viscerotropic velogenic NDVs (VVND) cause a highly severe form of the disease in which hemorrhagic lesions are characteristically present in the intestinal tract, neurotropic velogenic NDVs cause high mortality following respiratory and nervous signs, mesogenic NDVs cause respiratory and sometimes nervous signs with low mortality, lentogenic respiratory NDVs cause mild or inapparent respiratory infection and asymptomatic enteric NDVs cause inapparent enteric infection (Chuahan and Roy, 1998; Alexander and Jones, 2001). Approximately from 800 known avian species, only about 236 species have a record of NDV isolation. The disease is seen most frequently in domestic poultry, including guinea fowl, a species more susceptible than turkey and peafowl (Allan et al., 1973). Ducks, geese, partridge, and quill are relatively resistant (Higgins, 1971; Allan et al., 1973). The most resistant species appear to be aquatic birds, while the most susceptible are gregarious birds forming temporary or permanent flocks (Kaleta and Baldauf, 1988).

Spradbrow (1988), reviewing the 1985 animal health yearbook of the Food and Agriculture Organization of the United Nations (FAO), summarized that lentogenic or mesogenic strains of NDV were present in most countries of Asia, Africa, and Europe, and in the USSR. More than one third of the countries of Asia and one fifth of the countries of the world acknowledged the presence of the velogenic strains of the virus. In contrast, the countries of Oceania were relatively free of NDV. Some countries

recognized the presence of the avirulent strains of the virus, while many of the island states were apparently free from all pathtypes of NDV. A sequence of events following introduction of NDV into chickens is initiated by multiplication of the virus at the site of introduction. Initially the virus replicates in the mucosal epithelium of the upper respiratory and intestinal tracts. Then follows release of the virus into the bloodstream, a second cycle of multiplication occurs in visceral organs producing a secondary viremia. This leads to infection of other target organs such as lungs, intestine and central nervous system (Murphy et al., 1999). Signs of the disease and liberation of the virus into the environment are associated with the second release of the virus into the bloodstream. An exception occurs with large airborne exposures where the virus replicates in and is disseminated from the epithelium of the respiratory tract (Beard and Hanson, 1984).

Pathologic changes associated with ND vary greatly from bird to bird, flock to flock and from one geographic region to another. Gross lesions vary depending on virus and may also be absent. Cadavers of birds that died because of virulent NDV, usually have a dehydrated appearance. These lesions are often particularly prominent in the proventriculus, small intestine and ceca. These organs are markedly hemorrhagic which apparently results from necrosis of the intestinal wall or lymphoid tissues, such as cecal tonsils (Chuhhan and Roy, 1998; Alexander and Jones, 2001). Little evidence of gross lesions is found in the central nervous system even in birds showing neurological signs prior to death. Gross pathological lesions are usually present in the respiratory tract in birds with respiratory illness. They consist predominantly of hemorrhagic lesion and congestion of the trachea and in addition air sacculitis may be evident. Egg peritonitis is often seen in laying hens affected with virulent NDV (Beard and Hanson, 1984; Murphy et al., 1999). Histopathologically, hyperemia, edema, hemorrhage, and other changes in blood vessels are found in various organs. In general, in most tissues and organs involved, the lesions include hyperemia, necrosis, cellular infiltration, and edema. Lesions in the central nervous system are characterized by nonpurulent encephalomyelitis (Beared and Hanson, 1984; Al-Garib, 2003). For virus isolation in the head, spleen and long bones from acutely sick birds should be collected after disinfection in flame. The brain, bone marrow and spleen tissues are crushed into pieces using pestle and mortar with 5 mL broth, 2000 I.U. of penicillin and 5 mg of streptomycin. In addition to virus isolation other tests conducted to confirm the disease include: hemagglutination test, hemagglutination inhibition (HI) test, virus neutralization test, fluorescent antibody technique (FAT) and complement fixation test (CFT) (Chuahan and Roy, 1998).

Effective control of Newcastle disease requires good sanitation, management, quarantine, an appropriate vaccination program, and monitoring system, including serotyping and pathogenicity testing of isolated virus (Meulemans, 1988). According to Hanson (1978) a minimum of 70% of flocks in high risk areas must be included in sanitary and combined vaccination programs if control is to be effective. Vaccination against ND can be performed using either live or inactivated vaccines (Meulemans, 1988).The effectiveness of ND vaccines in the control of the disease, whether under closed commercial, semi-closed intensive, or under free range rural systems in tropical countries, depends on the virulence of the field strain, immunological state of the birds and the method of vaccine application (Meulemans, 1988).

The results of vaccine trials in Ethiopia showed that conventional (HB1 and La Sota) and the thermostable NDV-I_2 vaccines give similar antibody response and protection against challenge when given via the ocular and the drinking water route. The oral application of NDV-I_2 was also shown to be effective with barley as vaccine carrier, if barley was pre treated by parboiling (Nasser, 1998).

Infectious bursal disease

Infectious bursal disease (IBD) is a highly contagious immunosuppressive infection of immature chickens caused by a double stranded RNA virus in the genus *Avibirnavirus* (Faragher, 1972; Dobos et al., 1979; Muller et al., 1979; Lukert and Saif, 1997). Infectious bursal disease also known as Gumboro disease was first recognized by Cosgrove (1962) as a clinical entity, in 1957 in USA. Allan et al. (1973) reported that IBD virus (IBDV) infections at an early age were immunosuppressive. The existence of a second serotype was reported in 1980 (McFerran et al., 1980). Control of IBD viral infection has been complicated by the recognition of “variant” strains of serotype 1 IBDV that were found in the Delmarva poultry producing areas in the US (Rosenberger et al., 1985). Infectious bursal disease (IBD) also known as Gumboro disease is caused by infectious bursal disease virus (IBDV) that belongs to the genus *Avibirnavirus* of the family *Birnaviridae*.

Two serotypes of the virus are recognized and designated as serotype 1 and 2. Only serotype 1 appears to be pathogenic to chickens (Murphy et al., 1999). Antigenic and pathogenic variant strains have been documented. The range in virulence of strains in serotype 1 varies from mild to intermediate to intermediate plus. Very virulent strains of IBDV also exist (Chuahan and Roy, 1998). One of the most interesting features of IBDV is its ability to remain infectious for a very long period of time and its resistance to commonly used disinfectants. Infectious bursal disease is usually a disease of three to six week old chickens. But an early subclinical infection before three weeks of age was also observed (Lukert and

Saif, 1997). All breeds are affected but severe reactions with highest mortality rate were observed in White Leghorn (Lukert and Saif, 1997). Chowdhury et al. (1982) observed higher mortality rate (70 to 80%) in Fayoumi breed as compared to that in White Leghorn (40%) during field outbreaks. There is no report of egg transmission of IBDV. Infected birds excrete virus in their dropping at least for 14 days (Baxendale, 2002). Chickens are the only known avian species to develop clinical disease and distinct lesions when exposed to IBDV (Lukert and Saif, 1997). The most common mode of infection is through the oral route. Conjuctival and respiratory routes may also be involved (Sharma et al., 2000). Following oral infections, the virus replicate in gut associated macrophages and lymphoid cells. From there, the virus enters the portal circulation leading to primary viremia. Within 11 h of infection, viral antigen can be detected in bursal lymphoid cells. Large amount of the virus released from the bursa produce a secondary viremia, resulting in localization in other tissues. IBDV causes severe immune suppression in young chickens by its lymphocytolytic effects on surface IgM bearing B-cells.

Furthermore, immune suppression may be associated with the effect of the virus on T-cells and macrophages (Lukert and Saif, 1997; Murphy et al., 1999). Infectious bursal disease virus replication in target organ mainly in the bursa of Fabricius leads to extensive lymphoid cell destruction in bursal follicles. Early in the infection process, the bursa becomes edematous, hyperemic and creamy in color with prominent longitudinal striations. Later on, lymphoid follicles of the bursa become totally necrotic and in surviving birds the follicles will be devoid of lymphoid cells (Chuahan and Roy, 1998; Baxendale, 2002). Highly virulent virus strains could also cause depletion of lymphoid cells in the thymus, spleen and bone marrow. Depletion of B-cells from the medullary areas results in cystic cavities that are obvious under light microscopy. In long standing cases, there is an increased connective tissue mass in the interfolicular areas replacing the depleted lymphoid tissues (Sharma et al., 2000; Negash, 2004). Classical IBD is characterized by acute onset, relatively high morbidity and low flock mortality in 3 to 6 week old-broilers or replacement pullets. Diagnostic lesions include muscle hemorrhages and bursal enlargement (Hanson, 1978; Chuahan and Roy, 1998; Baxendale, 2002). The dramatic impact of a very virulent IBD virus can be reduced by biosecurity methods that are cleaning and disinfection, since the virus is very stable for months.

It is largely excreted through feces hence contaminated litter, feed and water have to be burnt or buried deep under the lime cover. Besides this other measures are; lower stocking densities, increasing intervals between flocks and complete removal of organic waste between batches.

In areas where management practices to reduce virus concentration are used, the disease trends to occur at a later age, and immunosuppressive form of infection is reduced (Stwart-Brown and Grieve, 1992). Administration of inactivated vaccines to breeder hens induces long-standing and high levels of antibodies in the hatched chicks. But in some areas where very virulent IBD virus has caused significant losses the producers do not adopt inactivated vaccination. But intensive live virus vaccination program is used in the hatched chicks from the unvaccinated breeder hens. Such chicks escape the strong risk of immunosuppressive form of the disease (Chuahan and Roy, 1998).

Marek's disease

The causative agent for Marek's disease (MD) is a cell associated lymphotropic herpesvirus. Due to its lymphotrpic nature, MD virus (MDV) was originally classified in the family *Herpes viridae* as a member of subfamily Gammaherpesvirinae (Chuahan and Roy, 1998). However, on the basis of genomic organization, MDV is currently classified with the viruses of subfamily Alphaherpesvirnae. Three serotypes of MDV and related Herpes viruses have been defined. Serotype 1 includes all the pathogenic or oncogenic strains of these viruses. Serotype 2 includes naturally non-attenuated strains of MDV. Serotype 3 includes turkey herpesvirus (HVT), the non-oncogenic MDV related virus isolated from turkey. New pathtypes have been emerging indicating continuous evolution of MDV towards greater virulence (Venugopal et al., 2001). The pathogenesis of MD is complex, with infection occurring throughout the respiratory route from inhalation of poultry house dust contaminated with the virus. After an early cytolytic infection mainly of the B-lymphocytes in the bursa, spleen and thymus, at 3 to 5 days post infection, the virus infects activated T-lymphocytes, mainly of the $CD4^+$ phenotypes. The infection in the T-lymphocytes becomes latent at 6 to 7 days post infection and the virus is spread throughout the body by the infected lymphocytes that persist as a cell-associated viremia. A secondary cytolytic infection occur in the feather follicle epithelium form about ten days after infection, from where infectious cell-free virus is produced and shed into the environment in feather debris and dander. The lately infected T- lymphocytes are subsequently transformed leading to the development of lymphomatous lesions in visceral organs. The main target cells for transformation in natural infections are $CD4^+$ T-cells, although the virus also has the potential to transform $CD8^+$ T-cells (Murphy et al., 1999; Venugopal et al., 2001).

The characteristic gross lesions in the classical form of MD are the enlargement of one or more peripheral nerves. The most commonly affected nerves that are easily seen on post mortem examination are the bracial and sciatic plexus and nerve trucks, celiac plexus, abdominal vagus and intercostals nerves. The affected

nerves are grossly enlarged, and often are three or four times their normal thickness (Chuahan and Roy, 1998. The normal cross-striated and glistering appearance of the nerves is lost; they have grayish or yellowish appearance and are edematous. Lymphomas are present in some chronic form of the disease most frequently as small, soft, grey, tumors in the ovary, kidney, heart, liver and other tissues. In the acute form, the typical lesion is widespread, diffuse lymphomatous involvement of visceral organs such as liver, spleen, ovary, kidney, heart and proventriculus. Sometimes lymphomas are also seen in the skin around the feather follicles and in the skeletal muscle. The liver enlargement in young birds is usually moderate compared to the adult birds. In acute cytolytic form of the disease caused by some virulent isolates, the thymus and buras of Fabricius may disappear completely due to extensive atrophic changes. The peripheral nerves in both classical and acute form of the disease are affected by proliferative inflammatory or minor infiltrative changes (Murphy et al., 1999; Venugopal et al., 2001). The clinical signs, combined with post-mortem findings, will confirm the diagnosis of Marek' disease in most cases, and, most importantly, rule-out other diseases. Enlargement of nerves such as the sciatic nerve are commonly seen at post-mortem. Changes in one or more internal organs may also be observed (Chuahan and Roy, 1998; Venugopal et al., 2001).

Although vaccines are commonly used in the commercial poultry industry, small numbers of doses cannot be purchased for use in backyard flocks. For backyard flocks, the best protection against Marek's disease is obtained by buying, from a commercial source, birds that have been correctly vaccinated. Vaccination alone will not prevent Marek's disease. Particularly for commercial flocks, it is important to have good biosecurity to ensure that vaccinated chicks will develop immunity before they are subjected to a severe challenge of virus. For example, chicks need to be reared separately so that they are free from the infected fluff and dust of older birds. Standard hygiene measures are also important, including a thorough clean-out and disinfection of sheds and equipment between batches of chicks with a disinfectant effective against viruses. Good nutrition and maintenance of freedom from other diseases and parasites are also very important. These practices will help maintain the flock's health and to ensure that the birds have optimum resistance against Marek's disease infection (Murphy et al., 1999; Venugopal et al., 2001).

CURRENT STATUS OF MAJOR VIRAL DISEASES OF CHICKENS IN ETHIOPIA

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. In addition, the intensification and dissemination of exotic breeds to villages has created chicken population, which is susceptible to the major viral diseases. The loss due to viral diseases like Newcastle disease and infectious bursal disease is escalating in recent years. Some farmers have even given up rearing poultry because of increasing disease problems (Edwards, 1992; Tadelle, 1996; Ashenafi, 2000; Zeleke et al., 2005a). Newcastle disease is mentioned as the major disease problem in commercial poultry farms and village chickens in most parts of the country. The disease has many different local names in different areas and the most common one is *"Fengele"* (Edwards, 1992; Tadelle, 1996; Nasser, 1998; Ashenafi, 2000; Halima et al., 2007), which means sudden dorsal prostration and signifies the acuteness and severity of the disease. In 1995, an outbreak of ND in the surrounding areas of Debre Zeit, Nazareth and Addis Ababa killed almost 50% of the local/indigenous chickens (Nasser, 1998) (Table 1). A serological survey conducted in six villages from central Ethiopia (non-vaccinated birds) revealed a high prevalence (43.68%) of NDV antibodies (Ashenafi, 2000). Another study conducted in village chickens in the southern and rift valley districts of Ethiopia indicated that ND is endemic in the area. Higher seroprevalence rates (22.51%) of NDV antibodies were verified in all the dry areas of the rift valley and a prevalence of 14.13% was reported in some parts of the wet southern districts (Zeleke et al., 2005a) (Table1).

In Ethiopia, outbreaks of ND usually happen at the beginning of the main rainy season (end of May and beginning of June). Nevertheless, this seasonal pattern seems to have changed after the 1984 to 1986 villagenization programs were launched, and it has become a problem throughout the year, although it is still more serious at the beginning of the main rainy season (Tadelle and Jobre, 2004). Nasser (1998) has also reported the occurrence of the disease all year round. Ashenafi (2000), on the other hand, reported that few clinical cases of ND during the dryer months of the year even if the seroprevalence of antibodies against NDV remained high throughout the year like reports by other studies (Tadelle and Jobre, 2004). It is possible to say that currently there are no low risk areas for ND remaining in Ethiopia. The disease has already become endemic in village poultry population and thus it recurs every year inflicting heavy losses (Tadelle and Jobre, 2004). It has been indicated that velogenic strains of NDV are widely distributed throughout the country (Nasser, 1998). But in general the epidemiology of ND in the village poultry in Ethiopia is poorly understood and there is no appropriate investigation and control strategy designed against the disease. This is due to lack of disease monitoring capacity in the Veterinary Services Department of the Ministry of Agriculture and Rural Development (Tadelle and Jobre, 2004).

Farmers start to consider, therefore, losses due to diseases as normal and natural (Tadelle, 1996; Nasser,

Table 1. Summary of occurrences of Newcastle disease infectious bursal disease and Marek's disease in certain areas in Ethiopia.

Disease	Location	Period/year	Mortality rates (%)	Prevalence/ Incidence (%)	References
Newcastle disease	Central highlands of Ethiopia (Dembi, Shola and Lemlem poultry farms)	1983-1995	14.90	58.50	Nasser, 1998
	State poultry farms of Ethiopia	1983-1995	2.3	-	Nasser, 1998
	Central high lands of Ethiopia	1999-2000	43.7	-	Ashenafi, 2000
	Rift valley	2004-2005	22.51	-	Zeleke et al., 2005a
	Wet highland districts	2004-2005	14.13	-	" "
	Bahir Dar District	October 2007-April 2008	-	29.60	Mazengia et al., 2010
	Farta District	October 2007-April 2008	-	21.70	" "
Infectious bursal disease	Debre Zeit overall (mortality)	2004-2005	49.89	-	Zeleke et al., 2005b
	Layer (morality)	2004-2005	25.08	-	" "
	Broiler (mortality)	2004-2005	56.09	-	" "
	Debre Zeit (seroprevalence)	2004-2005	-	93.30	" "
	Andasa poultry farm (young mortality)	Oct-Nov 2006	72	-	Woldemariam and Wossene, 2007
	Andasa poultry farm (adult mortality)	Oct-Nov 2006	7	-	" "
	Andasa poultry farm (seroprevalence)	Oct-Nov 2006	-	100	" "
	Bahir Dar	October 2007-April 2008	38.9	29.40	Mazengia et al., 2009, 2010
	Farta	October 2007-April 2008	17.40	21.70	" "

1998) and they fail to report outbreaks to the veterinary authorities. Infectious bursal disease is another newly emerging disease of chickens in Ethiopia. This disease has been speculated to be introduced concurrent with the increased number of commercial poultry farms flourishing in the country (Zeleke et al., 2002; Woldemariam and Wossene, 2007). The report of introduction of IBD in the country has come only recently where chickens with clinical signs and lesions of IBD have been shown to be positive for anti-IBDV antibody (Zeleke et al., 2005b) (Table 1). Report of IBD outbreak in Debre Zeit in 20 to 45 days old broiler and layer chickens indicated that the mortality rate of the disease in different poultry houses ranges from 45-50%. The overall mortality however was 49.89%. Broiler mortality due to IBD was 56.09%, while 25.08% for layer chickens and the seroprevalence of IBDV antibody was 93.30% (Zeleke et al., 2005b) (Table 1). Case report from Andasa poultry farm, northwest Ethiopia, indicated that the overall mortality of birds due to IBD was 72% in young (1 to 70 days of age) and 7% in adults (> 70 days old), showing young birds are more susceptible than adults. In this study a 100% seroprevalence has been shown in the study population (Woldemariam and Wossene, 2007). These reports indicate that the disease is present in many parts of the country and is disseminating at a faster rate in recent years. 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, there is not any endeavor towards the implementation of cost effective control strategies of IBD. But there is recommendation from the Federal Ministry of Agriculture and Rural Development that regional states should implement vaccination against IBD to combat the loss of poultry at this stage (Mazengia et.al 2010).

Future challenges and prospects regarding the control of major viral diseases of chickens in Ethiopia

Among the different food sources, poultry products contribute significantly to the country's food demand. With the increasing population of the country, there is an increasing demand for the supply of food. Under the prevailing management

situations, it may be difficult to fulfill these demands in a short time. Therefore, intensification and upgrading of the potential of birds will be inevitable to provide surplus products (Hailemariam et al., 2006). In line with this aim different chicken strains have been introduced into this country. The chicken strains imported are temperate breeds that are less adapted to the heat stress and disease challenges in the country. Accompanying with the intensification of poultry farming, there are occurrences of epidemics of newly introduced diseases and/or epidemics of endemic diseases like ND and IBD (Zeleke et al., 2005a, b). These diseases have incurred considerable economic loss to the country. At present, the most important challenge at the door is the failure of early diagnosis and reporting of the different poultry diseases when they occur and this has hindered the success of control mechanisms implemented in some parts of the country (Tadelle and Jobre, 2004).

Absence of research-based investigation approaches resulted in lack of knowledge of the prevalent strain of viruses and information on the overall epidemiological patterns of the different viral diseases. This has been posing a challenge especially for the success of vaccines used at these times (Wit and Baxendale, 2004). In diseases like MD, even if successful vaccination programs have been implemented, it remained an economically important disease basically because of the combined effect of relatively high vaccination costs, a continued evaluation of the virus to a more virulent strain and the early challenge by MDV before vaccine-induced protection (Schat, 2004). Lack of integrated approaches for the control of predisposing diseases has led to the ineffectiveness of vaccination programs. A good example is failure of ND 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, 1996; Tadelle and Ogle, 2001; Hailemariam et al., 2006). In Ethiopia, lack of coordinated implementation of importation policy and quarantine services have opened the door to the introduction of many poultry diseases that have not been reported previously like IBD. Birds are imported without health certificates and are not declared whether they were vaccinated against major viral diseases of chicken or not (Zeleke et al., 2005a, b).

Another challenge in the protection of chickens against major pathogens is that chickens are challenged with the viruses, as well as other pathogens, as soon as they are placed on the farms, which is in general within 1 to 3 days after hatching. This will be before the time of vaccine-induced protection even if we vaccinate day old chicks (Sharma and Burmester, 1982). Thus, this directs the vaccination of embryos 3 days before hatching. In Ethiopia, lack of coordinated implementation of importation policy and quarantine service has opened the door to the introduction of many poultry disease that have not been reported previously like IBD. Birds are imported without health certificate and are not declared whether they were vaccinated against major viral diseases of chicken or not (Chuan and Roy, 1998; Baxendale, 2002). In the future, there is a tendency of growing poultry industry and there are some promising research outputs that could help effective control of viral diseases threatening this sub-sector. Production of safe and effective vaccines are underway to minimize the effects of these agents. In recent times, an immune complex (ICX) vaccine against IBD is prepared.

In addition to improved safety, the ICX vaccine is known to induce early immunity when administered to embryos as evidenced by higher antibody titers or higher levels of protection. Antigens preserved in the form of ICX are believed to maintain humoral immune responses for a long duration (Tew et al., 1980; Jerurissen et al., 1998) and ICX vaccines are also believed to be insensitive to maternal antibody and thus can also be administrated to day-old chicks irrespective of the level of maternal antibody (Jerurissen et al., 1998). The research of Sharma and Burmster (1982) has brought the idea of *in ovo* vaccination into the vaccine technology. *In ovo* vaccination or embryo vaccination is the administration of vaccines into fertile chicken embryos at incubation day 18 to 19 after drilling a hole via the eggshell. The embryos may ingest the surrounding amnion with the vaccine and thus the antigen can stimulate the immune competent cells of gut-associated lymphoid tissues. This is known to induce the development of an earlier and a more solid protective immunity. It is also plausible that the highly efficient administration of the *in ovo* vaccine reduces the percentage of chicks that are not properly vaccinated, thus reducing the number of chicks at risk. Currently, *in ovo* administered vaccines are available for ND and IBD (Jerurissen et al., 1998).

CONCLUSIONS AND RECOMMENDATIONS

Newcastle disease, infectious bursal disease and Marek's disease are among major viral diseases of chickens in Ethiopia. They are highly contagious serious threats of the poultry industry. Although, these diseases are introduced recently to the country, they cause significant losses to both commercial poultry farms as well as rural poultry production. Although there are some studies that indicated poultry diseases of viral origin became endemic throughout the country, information on the epidemiology and as well as the occurrence of new strains of these diseases s scanty. The following points are recommended as they are important to design strategies to control and prevent these diseases:

1) In depth studies should be done on investigation of the epidemiology of these viral diseases;

2) Knowledge on the use of vaccines against this disease should be exploited, so as to have cost effective prevention methods; and
3) Attempts should emphasize on the identification of local viral strains to design cost effective vaccine.

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