A Review on Poultry Coccidiosis

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ABSTRACT
This review paper was done with the objectives of reviewing poultry coccidiosis and highlighting its economic impact on poultry production sector. Coccidiosis is the most important protozoan disease of poultry which is caused by the intracellular parasite of genus Emeria and resulting in significant economic losses worldwide. This disease is endemic in most of the tropical and subtropical regions of the world and transmitted through feco-oral route. It commonly affects young chickens and chickens managed under intensive system. Poultry coccidiosis is characterized by bloody diarrhea, ruffled feathers, dehydration and paleness of comb clinically and thickening of intestine, hemorrhage and necrotic enteritis at the specific site of intestine of chickens during necropsy depending on the species of Emeria involved. Diagnosis is based on clinical signs, coprology and post mortem examination routinely though various biochemical and molecular methods have also been used in recent years. Different anticoccidial and sulfa drugs are available for treatment and prevention although drug resistance becomes a major problem recently. It can be prevented and controlled by good managemental practices, prophylaxis, vaccination and immunization of chickens, selection of genetically resistant chickens and use of natural feed additives. Coccidiosis causes huge economic losses mainly due to production losses and costs of treatment or prevention. It is concluded that poultry coccidiosis is still the most important protozoan disease of poultry and have a great economic impact worldwide; and good managemental practices including good hygiene and bio security measures should be applied for the control and prevention of the disease.

Keywords: Bloody diarrhea, Economic importance, Emeria, Intensive, Poultry coccidiosis, Young chicken.

INTRODUCTION
The name poultry refers to all domestic birds such as chickens (domestic fowl), turkeys, ducks, geese, guinea fowls, ostriches and others, which are mainly kept for the production of meat and egg for human consumption. Among these, chickens are the most important species, adapted globally to various climatic conditions where human being lives and play a significant role in supplying animal origin protein to improve the nutrition of human being (Chauhan & Roy, 2007).

Poultry constitutes an important component of agricultural and household economy in the developing world and play important role to enable the landless poor farmers move out of poverty (Gueye, 2005). In Africa, village poultry contributes over 70% of poultry products and 20% of animal protein intake. In East Africa, over 80% of human population lives in rural areas and over 75% of these households keep indigenous chickens (Kitalyi, 1998; Matawork, 2016). Ethiopia has huge population of chickens estimated to be 56.53 million with indigenous, hybrid and exotic breeds of chickens representing 94.31%, 3.21% and 2.49%, respectively (CSA, 2017).

Diseases, predation, malnutrition, improper housing and poor management system are main constraints for the improvement of local poultry production systems in rural Ethiopia. Newcastle disease, coccidiosis, salmonellosis, chronic respiratory disease, and nutritional deficiency are the prevalent diseases of chickens in Ethiopia. Poultry mortality due to disease is estimated between 20-50% but can go as high as 80% during time of epidemics. Among those diseases, the parasitic protozoan disease coccidiosis is economically important and prevalent in chicken farm in Ethiopia (CSA, 2004).

Coccidiosis is recognized as the major parasitic disease of poultry and is caused by protozoan parasites of genus Emeria. The disease seriously impairs the growth and feed utilization of infected birds resulting in loss of productivity. It is one of a serious poultry parasitic disease that infects the epithelial lining of the intestine of poultry throughout the world (Conway & Mckenzie, 2007). Most Emeria species affect birds between 3 and 18
weeks of age and can cause high mortality in young chickens (Morris & Gasser, 2006).

Infection by coccidia in sufficient number to produce clinical manifestations of disease is called clinical coccidiosis (Conway & Mckenzie, 2007). Immunosuppressive diseases such as infectious bursal disease and Marek's disease are the major infectious diseases that increase susceptibility to viral, bacterial and parasitic diseases and interfere with acquired vaccine immunity (Hoer, 2010).

Coccidiosis is endemic in most of the tropical and subtropical regions where ecological and management conditions favor an all year round development and propagation of the causal agent (Obasi et al., 2006). It remains the most economically significant parasitic infection of the poultry worldwide (McDougald, 2003). In Ethiopia, coccidiosis is endemic in different regions and affects poultry production seriously (Safari et al., 2004).

Coccidiosis is a problem which needs a deep and thorough investigation and subsequent monitoring so as to boost production and productivity. More knowledge of the etiology and population dynamics of mixed coccidial infections in commercial poultry production is therefore needed (Haug et al., 2008). Moreover, with the increasing interest in poultry production evidenced by the proliferation of poultry farms, it is pertinent to continually evaluate the prevalence, frequencies of the different Eimeria species and management issues associated with common poultry diseases such as coccidiosis in any given zone (Etuk et al., 2004). Therefore, the objectives are to review poultry coccidiosis and to highlight its economic impact on poultry production.

GENERAL BACKGROUND

Coccidiosis is among the most common diseases of poultry caused by protozoan parasite of Eimeria species in spite of advances made in prevention and control through chemotherapy, management and nutrition. It causes massive destruction of the epithelial cells and characterized by droopiness, paleness of the comb, diarrhea and occasional appearance of blood in droppings. The death rate can be quite high, both in chicks and in adults (Fanatico, 2006).

Etiology:

Poultry coccidiosis is caused by the intracellular protozoan parasite of Eimeria species in the kingdom Protozoa, phylum Apicomplexa, class Coccidia, order Eucoccidiorida, family Eimeridae and genus Eimeria (Taylor et al., 2007).

Nine coccidian (Eimeria) species are identified as causative agents of poultry coccidiosis but only seven of them have been reported to be pathogenic (Kahn, 2008). Eimeria necatrix (E. necatrix) and Eimeria tenella (E. tenella) are the most pathogenic Eimeria species. Eimeria acervulina (E. acervulina), Eimeria maxima (E. maxima) and Eimeria mivati (E. mivati) are common and slightly to moderately pathogenic while Eimeria brunetti (E. brunetti) is uncommon but pathogenic when it does occur. Eimeria mitis (E. mitis), Eimeria praecox (E. praecox) and Eimeria hagani (E. hagani) are relatively non-pathogenic species (Soulsby, 2002).

The morphology of coccidia oocysts is similar for most Eimeria species. They are ellipsoidal or circular shaped with a thick cell wall and sporocysts. Majority of Eimeria oocysts have ovoid shape. E. maxima (30.5 x 20.7 m) is the largest while E. mivati (15.6 x 13.4 m) and E. mitis (15.6 x 14.2 m) are the smallest as compared to other species of Eimeria. E. tenella, E. maxima, E. acervulina, E. hagani and E. burnetti are ovoid while E. necatrix is oblong (Clark & Blake, 2012).

Eimeria species are very host specific and sites of development is intestine (epithelial cells of the intestinal villi or cells of the crypts) (Varghese, 2004). The species of genus emeria which affect chickens and the site of development in intestine of affected chickens are illustrated in Table 1.

| Table 1. Species of Eimeria and their predilection site in the host. |
|-----------------------------|-----------------------------|
| **Eimeria species** | **Predilection site** |
| E. acervulina | Upper small intestine |
| E. brunetti | Lower small intestine and rectum |
| E. maxima | Middle and lower small intestine |
| E. necatrix | Middle and entire small intestine |
| E. tenella | Cecum |
| E. mivati | Upper small intestine |
| E. mitis | Anterior gut |
| E. hagani | Anterior gut |
| E. praecox | Anterior gut |

Source: Foreyt (2001)

Life Cycle:

Eimeria species follow a typical coccidian life cycle with both exogenous and endogenous developmental stages (Barta, 2001) as shown in Fig.1. The life cycle of all Eimeria species involves two or more generation of an asexual development known as shizogony, then merogony and followed by sexual phase gametogony which results in the formation of oocysts (Kaufman, 1999). Under proper conditions of temperature and moisture the oocyst develops within one to two days to form a sporulated oocyst which is capable of infecting other chicken (Fanatico, 2006).
The sporulated oocyst possesses four sporocysts that each sporocyst contains two infective sporozoites (the infective agent) within them. These infective oocysts can remain viable in the litter for an extended period (months to years, under ideal conditions) until transmitted feco-orally to a definitive host. The endogenous phase of the eimerian life cycle is initiated when a bird has ingested a sporulated oocyst (Barta, 2001). Chickens pick up the sporulated oocyst by pecking on the ground or in litter used for bedding in the poultry house (Fanatico, 2006).

The sporulated oocyst which is the infective stage of this enteric protozoan parasite is ingested and the action of mechanical and chemical factors in the gut (bile salt and trypsin) leads to the release of sporocysts and then sporozoites in the duodenal lumen of chickens (Jones et al., 1996).

Excystation results in the opening of the anterior cap of sporocysts to release infective sporozoites. Interactions of the apical complex with the plasma membrane of epithelial cells allow sporozoites to penetrate the host cell (Grimwood & Smith, 1996).

Sporozoites penetrate directly into host intestinal epithelial cells in the gut lining in various regions, depending on the species of Eimeria. Within these host cells, the sporozoite develops into a trophozoite (a growing stage that absorbs nutrients from the host) that grows larger and divides asexually to form numerous merozoites (known as merogony) (Allen & Fetterer, 2002). Merozoites lyse out of the original infected host intestinal epithelial cell to infect new intestinal epithelial cells, completing a second cycle of merogony (Innes & Vermeulen, 2006).

It should be noted that each species of Eimeria has a predetermined number of cycles of merogony (ranging between 2 and 4) that lead to characteristic prepatent periods (McDonald & Shirley, 2009). At the conclusion of the last merogonic cycle, the resulting merozoites enter new intestinal epithelial cells and initiate the sexual phase of the life cycle known as gametogenesis (Jones et al., 1996). These merozoites invade cells and develop into either macrogametes or microgametes. The macrogametocyte gives rise to a single female macrogamete whereas the male gametocyte matures and ruptures, releasing a large number of minute biflagellate micro-gametes. A thickened wall forms around the macro-gamete, forming a zygote when the macro-gamete is fertilized by micro-gamete. This stage is the young or immature oocyst (Conway & McKenzie, 2007).

The resultant diploid zygote produced upon fertilization builds a heavy resistant oocyst wall around itself and enters the intestinal lumen (Fayer, 1980). Sporulation occurs within the young oocyst to produce sporocysts, within which the infective sporozoites develop and contaminate the environment to begin a new cycle (Levine, 1973).

The life cycle is quite rapid with prepatent period of about 4-5 days but there may be variation within species. The degree varies with species but optimally may result in hundreds of thousands or even millions of oocysts produced from one ingested oocyst (Jordan et al., 2002).

**Epidemiology:**

The epidemiology of coccidiosis is a timely issue to be established for determining the potential risk.
factors and species causing the diseases, and subsequent design of preventive production system, agro-ecology and level of and control regimen, which is suiting the local management. Under farming conditions, it is impossible to produce a coccidia free environment (Jordan et al., 2002).

High animal density confined on a small space, high relative humidity, high air temperature, different (especially different age) categories of birds at same place, feed change, quality of feed, as disease were as high as 80% usually occurring in the form of outbreaks (Safari et al., 2004). The disease contributed to be a problem with prevalence rate of 50.8% and 11% in deep litter intensive system and backyard poultry production systems, respectively (Kinunghi et al., 2004). The previous and recent studies in different areas of Ethiopia indicated that poultry coccidiosis is highly prevalent and widely distributed disease as explained in Table 2.

Risk factors:
The possible risk factors associated with the outbreak of coccidiosis are the absence of disposal of litters from leaking pipes, absence of all-in-all-out system, the prevalence of stressors (such as change in diets and concurrent infections) and extensive use of coccidiostats (Mersha et al., 2009). Factors contributing to outbreaks of clinical coccidiosis include litter moisture exceeding 30%, immune suppression, suboptimal inclusion of anticoccidials in feed and environmental and managemental stress (Singla et al., 2007).

The severity of coccidiosis is dependent on both species of Eimeria and the size of the infecting dose of oocysts. The key factors in the epidemiology of coccidiosis are presence of sporulated oocysts in the environment for long period (Jordan et al., 2002). Most Eimeria species affect birds between 3 and 18 weeks of age and can cause high mortality in young chicks (Al-Natour et al., 2002).

Pathogen factors:
The oocysts are extraordinary resistant to environmental stressful factors and disinfectants, remaining viable in the litter for many months.

### Table 2: Prevalence of coccidiosis in different areas of Ethiopia.

<table>
<thead>
<tr>
<th>Site of study</th>
<th>No of examined</th>
<th>Method of examination</th>
<th>Prevalence</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hawassa</td>
<td>384</td>
<td>Fecal</td>
<td>65.1%</td>
<td>Muluken &amp; Liuel (2017)</td>
</tr>
<tr>
<td>Bahir Dar</td>
<td>384</td>
<td>Fecal and direct smear respectively</td>
<td>40.6%</td>
<td>Abebe &amp; Mekonnen (2016)</td>
</tr>
<tr>
<td>Mekelle</td>
<td>384</td>
<td>Fecal</td>
<td>20.3%</td>
<td>Brhane &amp; Nibret (2016)</td>
</tr>
<tr>
<td>Gondar</td>
<td>407</td>
<td>Fecal and post mortem</td>
<td>53.6%</td>
<td>Belaynew et al. (2016)</td>
</tr>
<tr>
<td>Yabello</td>
<td>384</td>
<td>Fecal</td>
<td>19.3%</td>
<td>Addis &amp; Endale (2016)</td>
</tr>
<tr>
<td>Kombolcha</td>
<td>582</td>
<td>Fecal and post mortem</td>
<td>48.5%</td>
<td>Bereket &amp; Abdu (2015)</td>
</tr>
<tr>
<td>Adama</td>
<td>394</td>
<td>Fecal and post mortem</td>
<td>56.3%</td>
<td>Ermias &amp; Mekonnen (2015)</td>
</tr>
<tr>
<td>Dire Dawa</td>
<td>384</td>
<td>Fecal</td>
<td>42.7%</td>
<td>Migbaru &amp; Abdi (2015)</td>
</tr>
<tr>
<td>Central</td>
<td>190</td>
<td>Clinical, fecal and post mortem</td>
<td>25.8%</td>
<td>Ashenafi et al. (2004)</td>
</tr>
<tr>
<td>Ethiopia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gondar</td>
<td>384</td>
<td>Fecal</td>
<td>43%</td>
<td>Hadas et al. (2014)</td>
</tr>
<tr>
<td>Addis Ababa</td>
<td>384</td>
<td>Fecal</td>
<td>23.1%</td>
<td>Alemayehu et al. (2012)</td>
</tr>
<tr>
<td>Debre zeit</td>
<td>300</td>
<td>Post mortem and histopathological</td>
<td>71.7%</td>
<td>Dinka &amp; Yacob (2012)</td>
</tr>
</tbody>
</table>

well as all other factors that compromise resistance to the disease and general health status of the birds plays an important role in distribution and prevalence of coccidiosis (Hofstad, 1984).

Infected chickens shed oocyst for several days or weeks. Coccidial oocysts are normally introduced in to new facilities through contaminated equipment or vehicles coming from other poultry operations (Conway & Mckenzie, 2007).

Geographical distribution:
Coccidiosis is a widespread disease in growing chickens around the world that can seriously restrict the development of poultry production (Conway & Mckenzie, 2007). The disease is endemic in most of the tropical and subtropical regions where ecological and management conditions are favorable for sporulation of oocyst and development the causative agent (Obasi et al., 2006).

Poultry coccidiosis, caused by *E. acervulina, E. necatrix, E. maxima, E. tenella, E. mivati and E. brunetti*, is endemic in all parts of the Ethiopia and affects mainly young growing birds. In the past years coccidiosis used to be the most important cause of mortality in all farms. Incidences of the
Eimeria species are omnipresent and can survive in infected birds and the environment for long times (McDougal, 2003). Hence, their viability outside the host for long time is crucial regarding the course of the disease and lack of cross immunity between species of Eimeria predisposes birds to infection and disease outbreaks caused by different species (Yun et al., 2000).

Due to the short prepatent period of the parasite and its high biotic potential, the number of oocysts in the litter rises rapidly. Poultry coccidia have high capacity to reproduce within the host; this leads to a rapid increase to success and the subsequent high level of the parasite within the susceptible host and subsequently high level of contamination of the environment (Jordan et al., 2002).

**Host factors:**

Young chickens are very susceptible to coccidiosis and outbreaks usually occur when birds are between 3 and 8 weeks of age. But birds can be infected at any time, if never exposed before (Fanatico, 2006). Age of the bird at the time of the first infection and number of passages of the infection as well as on ability of the bird to develop proper specific immune response are important factors for the occurrence of poultry coccidiosis (Hofstad, 1984).

**Environmental and management factors:**

The frequency of occurrence of coccidiosis in all farms studied may be attributed to the presence of favorable environmental conditions for oocysts sporulation. Deep litter under poor sanitation could provide optimal temperature and relative humidity for sporulation of oocysts (Mersha et al., 2009). High incidence of coccidiosis is usually observed in poultry managed under intensive management system like deep litter due to increased likelihood of high oocysts accumulation in the litters (Dakpogan & Salifou, 2013).

Management of poultry houses plays a momentous function in the spread of coccidiosis because coccidial oocysts are omnipresent and are easily spread in the poultry house environment. It is very complex to keep chickens coccidia free, especially under current intensive rearing conditions (Adhikari et al., 2008). Prevalence varied by management but not vary by flock size while bad management, such as wet litter that encourages oocyst sporulation, contaminated drinkers and feeders, bad ventilation, and high stocking density can worsen the clinical signs (Hadipour et al., 2011).

**Transmission:**

Transmission of poultry coccidiosis is through feco-oral route. Coccidiosis is transmitted by direct and indirect contact with the droppings of infected birds. It could also be spread between birds by the consumption of food or drinking water contaminated by faeces containing the infective stage of the coccidia which are known as sporulated oocysts (Chapman, 2002).

Fecal contamination of vehicles and personnel can spread the infection to other farms. Oocysts are distributed within the poultry building, inside and outside the house by invertebrates and vermin whilst mechanical ventilation systems serve to scatter the oocysts outside the house (Taylor et al., 2007).

People are important vectors of coccidian in disseminating oocysts, which could be carried over by manure clinging to shoes or by utensils carried about from one pen to the other. Flies, beetles, cockroaches, rodents, pets and other birds have also been incriminated as mechanical vector (Charlton, 2006).

**Pathogenesis:**

Infection is through ingestion of sporulated oocysts by oral route, with contaminated feed and/or water. After ingestion, infectious oocysts excyst, liberating the infective form called the sporozoit. Sporozoits infect epithelial cells of the intestine. Transfer of the sporozoits up to the locus of the primary lesion is with the help of intraepithelial lymphocytes (Daszak, 1999).

The parasites invade the lining of the intestine and cause tissue damage, lowered feed intake, poor absorption of nutrients from the feed, dehydration, and blood loss (Fabiyi, 2000). The tissue damage can also expose the bird to bacterial infections, like Clostridium and Salmonella. Diseases that suppress the bird’s immune system may act with coccidiosis to produce a more severe problem. For example, Marek’s disease may interfere with the development of coccidiosis immunity and infectious bursal disease may exacerbate a coccidia infection (Julie, 1999).

Pathogenesis of the infection is influenced by species of the coccidium, concurrent diseases and nutritional factors. *E. necatrix* and *E. tenella* are the most pathogenic in chickens because schizogony occurs in the lamina propria and crypts of epithelium of the small intestine and ceca, respectively, and causes extensive hemorrhage. Most species develop in epithelial cells lining the villi (Kahn, 2008).

**Clinical Signs:**

Poultry coccidiosis affects birds in both clinical and sub-clinical forms. The clinical form of the disease manifests through prominent signs of mortality, morbidity, diarrhea or bloody feces, dehydration, lowered feed intake, weight loss,
paleness, huddling, ruffled feathers, and depression (Julie, 1999; Taylor et al., 2007). The chicken affected with coccidia become tired and weak, young birds often have blood in the faces after 4-5 days, later their eyes are closed and their wings hang down and many birds die (Julie, 1999).

The occurrence of clinical coccidiosis is directly related to the number of sporulated oocysts ingested by a bird at one time, the pathogenicity of the Eimeria species, the age of the infected chicken and the management system. Chickens suffering from coccidiosis quickly become less productive and poor performers. Laying hens will experience a reduction in rate of egg production (Nematollahi et al., 2009). The sub-clinical coccidiosis manifests mainly by poor weight gain and reduced efficiency of feed conversion and gives rise to highest proportion of the total economic loss (Taylor et al., 2007).

**Necropsy Findings:**

The lesions of coccidiosis depend on the degree of inflammation and damage to the intestinal tract. They include thickness of the intestinal wall, mucoid to blood-tinged exudates, petechial hemorrhages, necrosis, hemorrhagic enteritis and mucous profuse bleeding in the ceca (McDougald & Hu, 2001).

Coccidiosis causes a thickening of the intestines which make them feel like sausage. There may be light colored spots on the surface of the gut, and inside the gut hemorrhages and streaks. The type and locations of lesions in the gut indicates the species of genus Eimeria (Fanatico, 2006) as explained in Table 3.

Histopathologically, the wall of the gut is thickened indicating retention of fluid (edema). There may be blood in the lumen of the gut indicating blood loss (hemorrhage), or merely retention of an excessive amount of blood in the tissue (hyperemia). There is also infiltration with various body reactions and the development of immune response (Marquardt et al., 2000).

**Diagnoses:**

The diagnosis of coccidiosis is based on clinical signs, coprology and pathomorphological and pathohistological analysis (Conway & Mckenzie, 2007). In recent years, various biochemical and molecular methods have also been used (Morris & Gasser, 2006). Clinical findings like the presence of faces with blood dysentery and diarrhea suggests coccidiosis (Vegad, 2004). Although serology is the predominant method of disease monitoring in commercial poultry, examination of blood smears, bone marrow and clinical chemistry values is rarely done (Wakenell, 2010).

**Detection of oocyst in feces:**

Floatation method with saturated salt or sugar solution is important for detection oocysts in feces of infected birds. It can be a useful indicator of subclinical infection. Concentration floatation technique is also used for the collection of Eimeria oocysts from intestinal content of chickens. Eimeria oocysts isolation depends on the measurements of oocysts by using a calibrated ocular micrometer at 400x magnification (Conway & Mckenzie, 2007). Oocyst size, shape, and minimum sporulation time are helpful in identification of Eimeria species (Arakkhazaeli et al., 2011).

**Examination of gross lesions:**

Diagnosis of coccidiosis in chicken is best accomplished by post mortem examination of representative number of birds (Hadipour et al., 2011). The characteristics of the observed lesions such as its location on the intestinal tract, its appearance and severity, the nature of intestinal contents and other associated gross changes can be useful in establishing a diagnosis (Conway & Mckenzie, 2007).

**Microscopic examination of lesion:**

Developmental stages of coccidia can be seen in smears taken from suspected lesion. Oocysts, schizonts or gametocysts are easily seen and can be pathogenic for certain species of coccidia. The

<table>
<thead>
<tr>
<th>Eimeria species</th>
<th>Location</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>E. necatrix</td>
<td>Middle intestine</td>
<td>Severe hemorrhage with mucoid discharge whitish and red spot in wall of intestine</td>
</tr>
<tr>
<td>E. maxima</td>
<td>Middle intestine</td>
<td>Distended intestine with hemorrhage spots, mucoid discharge</td>
</tr>
<tr>
<td>E. brunetti</td>
<td>Lower half of intestine</td>
<td>Thin walled intestine, mucoid on necrotic discharge, distension of intestine</td>
</tr>
<tr>
<td>E. tenella</td>
<td>Ceca</td>
<td>Severe hemorrhage with white red spots in wall of intestine</td>
</tr>
<tr>
<td>E. acervulina</td>
<td>Upper intestine</td>
<td>Whitish spots on wall on serous surface hemorrhage streak and whitish lesions on intestinal surface, mucoid enteritis</td>
</tr>
<tr>
<td>E. praecox</td>
<td>Duodenum</td>
<td>No lesion but slightly hemorrhagic appearance on intestinal surface of duodenum slight mucoid discharge</td>
</tr>
</tbody>
</table>

**Table 3:** Some characteristic lesions of Eimeria infection during post mortem examination.

**Source:** Saxema et al. (1998)
information from microscopic examination should be combined with other observations such as gross lesions and identification of individual species (Mcdougald & Reid, 1997).

**Molecular diagnosis:**
During recent years, there have been significant advances in the development of molecular-diagnostic tools (Lien et al., 2007). Molecular techniques have some advantages over traditional methods in that they rely only on the genomic sequence of the Eimeria species. Several Polymerase Chain Reaction (PCR) based assays using primers that specifically target different regions of the Eimeria genome have been described (Ogedengbe et al., 2011).

**Differential Diagnosis:**
Intestinal coccidiosis may be confused with necrotic enteritis, haemorrhagic enteritis, or other enteric diseases. Caecal coccidiosis may be confused with histomoniasis and salmonellosis due to their similar lesions (Hafez, 1997).

**Treatment:**
Anticoccidial drugs available for use finely or various combination are amprolum, clopidol, diclazuril, ethopabate halofuginonane and ionophores (monensin, lasalocid, narasin, maduramicin, nicarbasin, robenidine), and sulphaquinoxaline. Amprolum is structurally similar to and is a competitive antagonist of thiamine (vitamin B) because rapidly dividing coccidian has relatively high requirements for thiamine; nitrobemzamibles exerts their greatest coccidiostatic activity against the asexual stages (Kahn, 2008). While ionophores are anticoccidials commonly used in the large-scale industry and they alter the function of the cell membrane and rupture the parasite. Ionophores also have antibacterial action and help to prevent secondary gut diseases (Fanatico, 2006).

In the successful treatment of an outbreak of coccidiosis the aim is to treat birds already affected and at the same time allow sufficient merogonous development in the clinically unaffected birds to stimulate their residence (Taylor et al., 2007). Anticoccidials are commonly withdrawn from broilers within 3-7 days before slaughter to meet regulatory requirements and to reduce production cost; because of broilers have varying susceptibility infection at this point, the risk of coccidiosis outbreaks is increased with longer withdrawal (Kahn, 2008).

The emergence of drug resistance strains of coccidia presents a major problem. Continuous use of anticoccidial drugs leads to increased incidence of drug resistant strain development which results in reduced activity of the drug against the agent (Roy, 2007).

**Control and Prevention:**
Prevention of poultry coccidiosis can be achieved much easier than treatment. Coccidiosis can be prevented by good managemental practices (Ashenafti et al., 2004). It can be controlled mainly by drugs and also an effective vaccine is now available for breeders or layer replacements. Drugs have been very important in controlling coccidiosis but the emergency of anticoccidial drug resistance has affected the use of fullness of the drugs. The possibility that drugs may not always be relied up on to control coccidiosis has led to an interest in other means of control (Vegad, 2004). Apart from the use of drugs, control is now based on hygiene, vaccine and genetics. But genetics is a theoretical strategy not in practical use (Jordan et al., 2002).

**Management:**
Prevention of avian coccidiosis is based on a combination of good management and the use of anticoccidial compounds in the feed or water. Litter should always be kept dry and special attention should be given to litter near water fonts or feeding troughs (Taylor et al., 2007). Good ventilation, dry and clean litter, cleaning and decontamination of drinkers and feeders, and proper stocking density in poultry farms are the key managemental practices for prevention of the disease (Ashenafti et al., 2004).

Special care is needed in rainy season when moisture is prevalent along with suitable temperature for sporulation of oocysts. In case of clinical outbreaks, it is essential to remove and isolate the clinically affected birds because they excrete of oocysts every day, thus endangering the health of other birds (Roy, 2007).

**Prophylaxis:**
The prophylactic drugs used for prevention of coccidiosis are coccidiostats. An effective coccidiostat should inhibit the schizogenic stage and allow immunity to develop. The drugs available for use finely or various combination are amprolum, ionophores and sulphaquinoxaline (Kahn, 2008). Sulphaquinoxaline was the first drug administered in the feed continuously and at lower doses (McDougald, 2003).

The use of anticoccidial agents depends on the type of management concerned (Taylor et al., 2007). Antibiotics and increase level of vitamin A and K are sometimes used in the ration to improve rate of recovery and prevent secondary infection (Kahn, 2008).
**Vaccination and immunization:**

The intensive use of anticoccidial drugs which has led to the development of resistance, the public concern of chemical residues in poultry products and pollution of the environment has stimulated research for alternative control methods such as applying a vaccine early in life or development of new drugs (Yim et al., 2011).

The two types of available vaccines for immunization of chickens are attenuated and virulent vaccines (Chapman et al., 2002). Attenuated vaccines lack a part of the life cycle (less asexual reproductive cycles) of the original strain they were derived from, and as a consequence have a lower reproductive and pathogenic potential. This is a major advantage towards performance of virulent coccidial vaccines, but because of the lower reproductive potential of attenuated vaccines, production costs are significantly higher. The virulent vaccines are vaccines consisting of anticoecidal-sensitive strains and others made of more or less resistant strains. The main advantage of the live anticoecidal-sensitive strain vaccines is their ability to alter the level of resistance in a certain coccidial population (Mathis & Broussard, 2006).

Live vaccines comprising attenuated or virulent oocysts of various Eimeria species have offered a practical alternative to anticoecidal drugs for the sustainable control of coccidiosis in chickens, and in fact several such vaccines have been commercially available in the world market. However, Eimeria species induces solid immunity to homologous challenge and immune variation, as documented in *E. maxima*, may provide the basis for the lack of cross protective immunity among geographically isolated strains (McDonald & Shirley, 2009).

Numerous vaccination strategies have been attempted to control avian coccidiosis. Paracox vaccine has now been developed and used in chickens kept for breeding purpose and egg laying of non-caged system in Europe and many other countries (Shirley & Harvey, 2000).

Attenuated vaccines are produced mainly by either passaging through embryonated eggs, such as *E. tenella* in Livacox vaccines, or by selection for precocity, such as the other species of Livacox vaccines and the Paracox vaccines (Arabkhazaeli et al., 2014). A live attenuated vaccine is available as an alternative to coccidiosis for the control of coccidiosis in chickens. This consists of selected precocious strains of each of the pathogenic species of coccidia that affect poultry; these strains show rapid development in vivo with minimal damage to the intestine but stimulate an effective immunity. Various other vaccines have also become available in several countries using either live or attenuated strains of coccidian (Taylor et al., 2007).

**Selection of genetically resistant chickens:**

Selection of poultry for genetic resistance to coccidiosis is a promising method of control (Charlton, 2006). Disease control based on host natural resistance becomes an attractive alternative approach. This approach to control has not been developed, partly due to the efficacy and availability of drugs and partly due to the overriding priorities in breeding programmers. However, interest in this strategy is increasing as modern technologies of genetic manipulation developed (Jordan et al., 2002).

**Natural feed additives:**

A number of natural products or feedstuffs have been tested as anticoecidal dietary additives. It has been reported that antioxidant-rich plant extracts have potential benefits in treating coccidial infections (Allen & Fetterer, 2002). There have been some very good results with feeding oregano extract or essential oil to chickens, with evidence that oregano essential oil exerted an anticoecidal effect against *E. tenella*, with treated chickens showing body weight gains and feed conversion ratios no different to the uninfected control group. However, one study found that oregano may only be effective in birds that were not already vaccinated against coccidia (Batungbacal et al., 2009).

In Ethiopia, Adamu and Chaiwat (2013) have reported the protective effect of *Moringa stenopetala* leaf supplemented diets on *E. tenella* infected broiler chickens. Their experimental result confirmed that a diet supplemented with *Moringa stenopetala* leaf powder significantly reduced the oocyst count of *E. tenella* infected chickens similarly to those supplemented with amprolium when compared to the control chickens. In addition, a diet supplemented with Moringa leaf powder reduced the cecal lesion scores of *E. tenella* infected chickens similarly to those supplemented with amprolium.

**Economic Importance of Poultry Coccidiosis:**

Poultry coccidiosis is recognised as the parasitic disease with the greatest economic impact on poultry industries worldwide (Allen & Fetterer, 2002). The impact of disease on animal agriculture is typically assessed in quantitative terms. In poultry industry, the negative inputs including lost revenues, costs of vaccination/prevention, eradication, decontamination and restocking are the main impacts of the disease in poultry agricultural sector (Thrusfield, 2005).

The most problematic disease in the poultry industry worldwide is coccidiosis, mainly due to
This protozoan disease is responsible for great worldwide economic losses to the poultry industry with an estimated world annual loss of more than 3 billion USD (Dkhil, 2013). These estimates include the costs of prophylactic in feed medication for broilers and broiler breeders, alternative treatments if medication fails and losses due to mortality, morbidity, impaired growth rate, temporary reduction of egg production in layers and poor feed conversion of chickens that survive outbreaks (Asadi et al., 2015).

In Ethiopia coccidiosis is identified as a cause of direct and indirect losses in all farms. Losses occurred in the form of mortalities, coccidostats cost, reduced weight gains, reduced market value of affected birds, culling, delayed off take and reduce egg production. Previous studies conducted in the country showed that coccidiosis contributes to 8.4% and 11.86% losses in profit in large and small-scale farms, respectively (Kinunghi et al., 2004; Safari et al., 2004). In Ethiopia, average total losses were estimated as 898.8 and 5,301.8 Ethiopian Birr per farm in small- and large-scale farms, respectively (Kinunghi et al., 2004).

In conclusion, In general, poultry coccidiosis is still important and most prevalent protozoan parasitic disease of chickens that affects poultry production seriously and results huge annual economic loss worldwide including Ethiopia in spite of advances are made in control and prevention of the disease. It is caused by host specific, site specific and non cross protective protozoan parasites of genus Eimeria and affects the intestinal portion of the alimentary tract of chickens. This enteric parasitic disease is more common in chickens managed under intensive production system; and affects more frequently young growing chickens and chickens which never exposed before. Even though various control measures have been attempted against the disease, prophylactic use of anticoccidial drugs was widely used control approach which has resulting a problem of drug resistance in recent time. Eventually, it is concluded that agent, host, environmental and managerial risk factors for the occurrence of the disease and problem of drug resistance should be taken into account in designing the control and preventive program against this disease.

Hence, based on the above conclusion, for the control and prevention of poultry coccidiosis proper hygienic and bio security measures should be implemented, and prophylactic anticoccidial drugs should be provided with appropriate time and recommended dose. Moreover, cage housing system should be practiced in intensive production system instead of deep litter housing system to reduce the risk of accumulation of coccidian oocytes and occurrence of the disease, different age groups of chickens should not be reared in the same house and recommended stocking density should be practiced, and all-in and all-out system of production should be practiced in intensive farms.

REFERENCES


