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Coccidiosis in poultry-a review

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Abstract: In poultry industry, coccidiosis is one of the diseases that have negative impact on the growth performance of birds. It is the most consistently reported contagious disease in poultry because *Eimeria* species are competent to live for long time in infected birds and the environment, thereby, demanding the attention of poultry producers, poultry disease experts and other beneficiaries. Here, we review the factors affecting the development of these parasites and different strategies used for their control.
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Introduction

Coccidiosis is an intestinal disease, caused by protozoan parasites of genus *Eimeria*, and is one of the most important livestock diseases in the world. It has a high impact in the poultry industry where parasite transmission is favoured by high-density housing of large numbers of susceptible birds (Blake and Tomley, 2014). In poultry industry, it is one of the diseases that have negative impact on the growth performance of birds. Various factors affect the onset of this disease (Fig. 1). Poor management practices such as damp litter promoting oocyst sporulation, unhygienic feeders and drinkers, poor ventilation facilities and high stocking densities can aggravate the clinical infection (Khan *et al.*, 2006). Moreover, it is the most consistently reported contagious disease in poultry because *Eimeria* species are competent to live for long time in infected birds and the environment thereby; demanding the attention of poultry producers, poultry disease experts and other beneficiaries. According to an economic analysis, United States poultry production is facing about US$127 million annual losses because of coccidiosis (Chapman, 2009).

There are seven valid species of chicken coccidia, *E. acervulina*, *E. brunetti*, *E. maxima*, *E. mitis*, *E. necatrix*, *E. praecox*, and *E. tenella* (Shirley, 1986) and all of these have global distribution. Among all the species responsible for coccidiosis, *E. tenella* is the highly pathogenic species that requires continuous prophylactic treatment (Guo *et al.*, 2013). *E. tenella* is also the most prevalent parasite in the poultry farms of Kashmir Valley (Ahad *et al.*, 2014). This parasite invades caecum of the intestine, causing severe damage in birds. Disease is characterized by invasion of epithelial tissues of the intestine and destruction of the intestinal mucosa (Dalloul and Lillehoj, 2006). The use of several drugs, alone or in combination, have been employed in the struggle against avian coccidiosis. However, the emergence of drug resistant strains, especially after a prolonged use of a drug, is a real problem (Abbas *et al.*, 2008; Abbas *et al.*, 2011). The disease directly influences the production potential of infected chicken due to high mortality, retarded growth and poor feed conversion ratio, causing heavy economic losses up to three billion US dollars annually worldwide (Williams, 1999; Dalloul and Lillehoj, 2006). On the other hand, in sub-clinical form, it may ground the birds immuno-compromised and that paves way to secondary disease circumstances (Kabell *et al.*, 2006). Thus, for advantageous farming management of coccidiosis and maintenance of immune functions for maximum performance, growth and production in poultry industry are principal requirements.

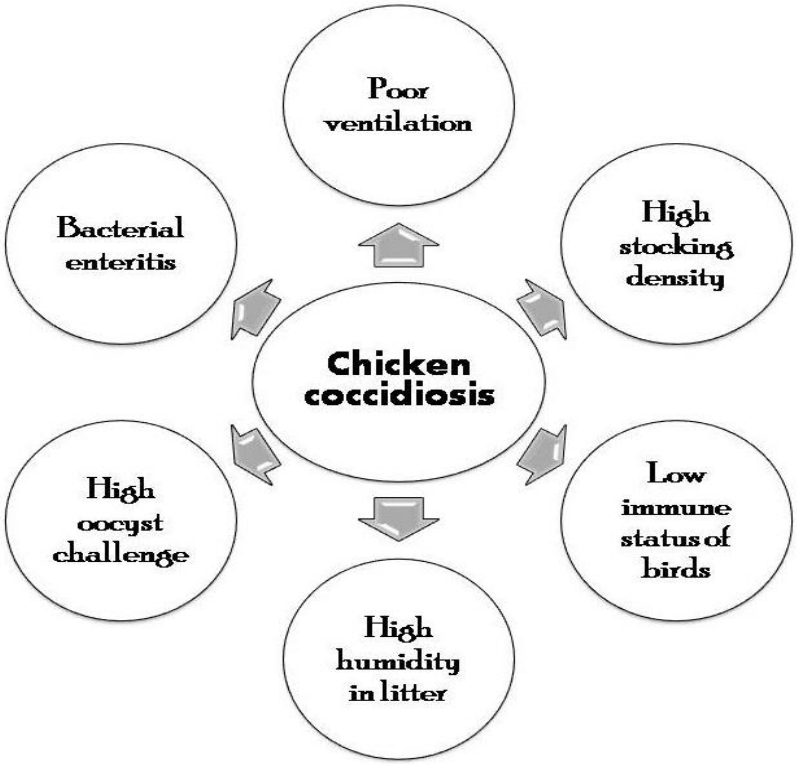

Fig. 1 Various factors affecting chicken coccidiosis

Etiology

Poultry coccidiosis is caused by protozoan parasites of genus *Eimeria*. A number of *Eimeria* species have been recorded from poultry birds (Table 1), each species affecting the particular part of the intestinal tract (McDougald, 1998).

Table 1 Species of *Eimeria* with their predilection site in the host

Species	Site of lesions
E. acervulina	Duodenal loop
E. brunette	Lower intestine
E. hagani	Anterior gut
E. maxima	Mid gut
E. mitis	Anterior gut
E. mivati	Duodenal loop to rectum and caecum
E. necatrix	Mid gut
E. praecox	Anterior gut
E. tenella	Caecum

Source: (Foreyt, 2001)

Life cycle of coccidian parasites

The life cycle of *Eimerian* parasites takes about four to seven days to complete. It begins when active "oocysts" (formed of parasites protected with a thick wall capsule) are picked up by the bird and swallowed. The whole life cycle (Fig. 2) consists of two stages:

a) An exogenous stage or sporogony in the external environment.

b) An endogenous stage in the digestive tract of the chicken consisting of three essential phases:

- Excystation or active departure of the sporozoites from the sporocysts
- This is followed by several schizogonies or merogonies
- Gamogony

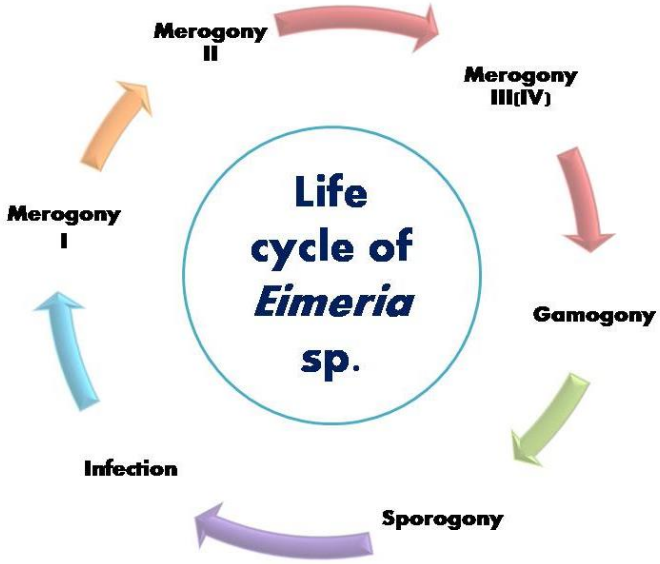

Fig. 2 General life cycle representation of *Eimeria* sp.

(a) Exogenous stage

Oocysts are excreted and released in the external environment with the droppings of infected birds. These oocysts sporulate in the external environment to become infectious. Transformation of sporont into four sporozoites takes about 48 hours at 25-28°C. Each sporocyst contains two sporozoites which are the infecting stages, thus in total eight sporozoites are formed from one oocyst. Sporulation is permitted by the conditions of temperature, moisture and oxygenation, often provided by the litter.

(b) Endogenous stage

The cycle starts again when sporulated oocysts are ingested by chicken. These oocysts are ground in the gizzard mechanically and their shells are destroyed, thereby releasing the sporocysts. In the duodenum excystation occurs. Here trypsin and bile salts dissolve the external wall of the sporocyst releasing sporozoites.

Merogony or asexual multiplication

Sporozoites are mobile and depending on the parasite species, these penetrate the epithelial cells of caecum or the intestine. After entry into the cell, the sporozoite transforms into trophozoite. Its nucleus divides resulting in the formation of schizonts or meronts. The meronts may contain several hundred merozoites and on maturation burst the cells to release merozoites into the lumen of intestine. These merozoites penetrate the neighboring epithelial cells and repeat this asexual multiplication process. Depending on the type of species, two to four successive generations of merogonies may take place.

Gamogony or sexual reproduction

After asexual multiplications, the merozoites invade the cells and develop into microgamonts and macrogamonts. Macrogamonts further grow to form a macrogamete and the microgamont forms a large number of microgametes. These microgametes are mobile having two flagella and fertilize the macrogametes. After fertilization, oocysts are formed which are further eliminated from the intestinal lumen along with the droppings of infected birds. Thus, several days later thousands of oocysts are formed from one oocyst and excreted into the environment. The time from ingestion of oocyst and its first excretion from the host is called prepatent period and varies from four to seven days depending on the type of species of *Eimeria* infecting the birds.

Transmission

The transmission or spread of coccidia is very easily accomplished. Chicken become infected with coccidia oocysts that have been shed in the droppings of infected chicken. These oocysts are thick-walled structures which are passed out in the feces (droppings). They become infective once they sporulate after a few days and may survive for long periods depending on environmental factors such as temperature and moisture. Infection of coccidiosis

starts with consumption of sporulated oocysts. Chicken pick them up by pecking on the ground or in litter used for bedding the house. Oocysts can survive many weeks in the soil outdoors—as long as 600 days (Farr and Wehr, 1949). Once the oocysts are ingested they broke down in the gizzard and releasing the sporozoite form of the coccidia, which enter the intestinal lining and start multiplication. Chicken may be infected with multiple species of coccidia at the same time. The source of the infection varies and depends on the technology used in the poultry industry. In a flock, disease may spreads by direct, as well as indirect contact (Williams, 2002). Oocysts that are infectious could be distributed by dust, equipments, people, rodents, insects as well as wild birds. Distribution and prevalence is influenced by several factors: high animal density, air temperature, relative humidity, different categories of birds (especially different age) at same place, feed change, quality of feed, other factors that compromise resistance to the disease and general health status of the birds (Chapman, 2014). Onset of the disease depends on the age of the bird at the time of the first infection and number of routes of the infection, as well as on ability of the bird to develop proper specific immune response (Ilic *et al.*, 2003).

Epidemiology

Coccidiosis is a disease common in poorly managed farms where the hygiene standards are compromised (Musa *et al.*, 2010). Damp litter conditions and warmth of 25-30ºC, favour the sporulation of oocysts (David, 2000). Oocyst sporulation occurs under optimal conditions of temperature (21-30ºC), adequate moisture and oxygen and become infective within 1-2 days (Trees, 1999; Etuk *et al.*, 2004; Musa *et al.*, 2010). Following the ingestion of high doses of the sporulated oocysts sudden outbreak of coccidiosis can occur over a short period of time by non-immune young (3-8 week old) birds (Musa *et al.*, 2010). Most birds get infection in the early few weeks of life but birds of any age are susceptible to coccidiosis (Chookyonix *et al.*, 2009). Coccidiosis has been reported about 3 days following ingestion of large numbers of sporulated oocysts (Urquhart *et al.*, 1996) and under field condition, the incubation period was reported to be 5 days for intestinal coccidiosis while it was 5-6 days for that of caecal coccidiosis (Chookyonix *et al.*, 2009; Musa *et al.*, 2010). In addition, the infected and recovered adult birds shed oocysts in their faeces thus contaminating feed and water (Trees, 1999; Musa *et al.*, 2010). Additionally, oocysts can survive outside the host for up to 2 years and have the capacity to resist low temperatures, dry weather conditions and many of the disinfectants (David, 2000). The highest incidence of coccidiosis is during spring and fall, especially when weather is moderate and humid. The incidence is significantly smaller during extreme cold, and during hot and dry weather conditions (Razmi and Kalideri, 2000; Ahad *et al.*, 2014). Clinical disease can be prevented by continuous adding of the anticoccidials in feed. However, persistence of the sub clinical disease is always a possibility. According to Razmi and Kalideri (2000), sub clinical forms of the disease depend on the size of the flock.

Pathology

The disease is a complex association and its pathogenicity depends on the parasite species involved, number of oocysts ingested, host sensitivity, and the environmental situation. The parasitic development affects the physiology of the host and is accompanied by rupture of the intestinal cells. However, pathogenicity varies in accordance with the species of *Eimeria* involved. Invariably a dose of 5 x 10^6 oocysts of *E. acervulina* produces intestinal lesions and weight loss; however, deaths are rarely observed. In comparison, a dose of 5 x 10^4 oocysts of *E. tenella* or *E. necatrix* is sufficient to kill most susceptible chicken.

The pathogenicity of coccidia depends largely on the successful replication of developing parasites inside the host. The pathogenic process starts during schizogonic phase of the parasite development. During first generation of schizonts the pathogenic process is insignificant but is at high level during the second generation of schizonts. Continuous multiplication of the oocysts results in inflammation, mucus desquamation, capillary rupture and hemorrhage. Initial signs of coccidian infection include decreased feed and water consumption, decreased egg production, pigmentation loss, weight loss, slow growth, poor feed conversion, bloody diarrhea, and high mortality. In severe infections, much of the mucosal epithelium is sloughed off and nutrient absorption is compromised (Jeurissen *et al.*, 1996; McDougald and Reid, 1997; Yun *et al.*, 2000). The severity of infection depends on the health conditions of the bird and the number of oocysts ingested. Chicken will usually develop immunity quickly, thus self-limiting the infection. However, immunity to one species will not prevent infection with another species; there is no cross-protective immunity. Coccidiosis goes hand in hand with gut diseases, because it damages the gut and allows bacteria to enter and cause secondary infections (Fanatico, 2006).

Economic importance

Coccidiosis is one of the most economically important diseases of poultry that play inhibitory role in the growth of poultry production. It is a complex disease of poultry caused by different species of *Eimeria*, affecting the birds in both clinical and sub-

clinical forms. The clinical form of the disease manifests through prominent signs of mortality, morbidity, diarrhoea or bloody faeces, and 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 losses (Williams, 1998; 1999; Dalloul and Lillehoj, 2006). Globally cost of coccidiosis to the poultry industry has been estimated to exceed US $ 2 billion per annum (Fornace *et al.*, 2013). It is also estimated that the economic losses due to the disease is about US $ 450 million with additional US $ 100 million due to medication in the United States alone (Maikai *et al.*, 2007). These estimates include the costs of prophylactic in-feed medication, alternative treatments (e.g., with amprolium) if the medications fail, and losses due to mortality, morbidity, and poor feed conversions of birds that survive outbreaks.

On agricultural basis the impact of the disease is typically monitored in quantitative terms. In poultry industry, these terms include for example lost revenues; costs of vaccination/prevention, eradication, decontamination and restocking. These have been referred to as negative inputs (Thrusfield, 1995). In developing countries, poultry mortalities due to diseases are estimated in between 20% to 50% but they can rise as high as 80% during epidemics (Alamargot, 1987; Alemu, 1995). During the last few years poultry sector occupies about 80% of the total production of meat. Chicken meat alone accounts for 30% of protein food consumed by humans (Belova *et al.*, 2012). This is gaining utmost importance among all sectors of livestock. Utilization of chicken meat is rising high because of the good feed conversion ratio. Poultry industry experience losses in the form of mortalities, coccidiostats cost, reduced weight gains, reduced market value of affected birds, culling, delayed off take and reduced egg production. An outbreak of coccidiosis has a very high economic impact on the flock as well as for the poultry producer as treatment alone cannot prevent the economic losses (Majero, 1980).

Control

Role of Poultry House Management

Management of the poultry birds is very difficult because of the ubiquitous nature of coccidial oocysts. These oocysts are easily dispersed in the surroundings of poultry farm and have such a large reproduction potential, thus becoming very difficult to keep chicken coccidia free, especially under intensive rearing conditions. Besides, the sporulation rate of oocysts is very fast, as a result of which the number becomes very large within a stipulated period of time. At the same time they can be damaged by bacteria, other organisms, and ammonia present in the litter and their viability begins to diminish after 3 weeks (Williams, 1995). In United States, the ventilation is maintained properly and removing of the caked litter is done on regular basis. On the other hand, in most European countries and Canada a thorough cleanout is done between the flocks. In addition the spread of coccidial infection can also be minimized by following the bio-control measures such as requiring caretakers to change clothes between houses.

Role of vaccines

Vaccines also play significant role in controlling the coccidial infection in poultry. Vaccination emerges as a tool of control because of the resistance against anticoccidial drugs (Augustine *et al.*, 2001). From immunological point of view vaccination proved to be the simplest and cheapest way to achieve immuno-prophylaxis. In that mode, the immune system is pre-activated and natural infection that causes a secondary immune reaction gets faster and better reaction in comparison to the primary immune reaction (Dimitrijevi and Ilic, 2003). For coccidiosis control, both live as well as recombinant vaccines can be used (Lillehoj and Trout, 1993). A perfect vaccine will stimulate long lasting immunity against all epitopes in the coccidia structure. But, at the same time the vaccination requires high expertise and care particularly while dealing with live vaccines.

Role of anticoccidials

In poultry industry the successful and effective exploitation of anticoccidial feed additives played a key role in the growth of the poultry by improving the quality of poultry products to the consumer. A number of anticoccidial agents are used such as amprolium, clopidol decoquinate, lasalocid, salinomycin, narasin, and maduramycin (Jeffers, 1997). Commonly polyether ionophores are reported to play a major role in the anticoccidial programs in commercial broilers, either alone or in shuttle programs with chemical anticoccidial drugs (Long and Millard, 1978; McDougald, 1982, 1990). However, there are increasing concerns about rising levels of drug resistance (Chapman, 1997; Abbas *et al.*, 2008; Abbas *et al.*, 2011). In addition, modern synthetic drugs used for curing coccidian disease also cause a number of side effects. There are increasing concerns over the presence of drug residue in the poultry products which is un-desirable for the consumers.

Conflict of interest

The authors declare there are no known conflicts of interest associated with this publication.

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