

Avian Coccidiosis

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Abstract: Coccidiosis is a disease that is caused by protozoan parasites of the genus *Eimeria*, developing within the intestine of most domestic and wild animals and birds. The disease is distributed worldwide and primarily affects young animals that are raised under various management systems avian coccidiosis, is caused by several species of the protozoan parasite *Eimeria*. This parasite invades epithelial tissues of the intestine, causing severe damage in birds and as a result, significant economic losses. The main problem with *Eimeria* infections is that they are caused by more than one species that attack different regions of the intestine. Coccidiosis is responsible for major losses due to mortality and primarily to the poor performance of sub clinically infected animals which costs the livestock industry many millions of dollars. Diagnosis and genetic characterization of different species of *Eimeria* are central to the prevention and control of coccidiosis. The classical parasitological methods of diagnosis are labor intensive and therefore costly. Different diagnostic tools such as fecal flotation techniques, morphological identification, molecular biology, biochemistry, cell biology and immunology have been used to diagnose coccidiosis. These methods have generally had major limitations in the specific diagnosis of coccidiosis and identification of *Eimeria* species. The traditional approaches are unreliable.

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1. Introduction

Coccidiosis is a disease that is caused by protozoan parasites of the genus *Eimeria*, obligate intracellular parasites, developing within the intestine of most domestic and wild animals and birds. These parasites can infect and multiply within the mucosal epithelia in different parts of guts via oral route. The disease causes gut damage (i.e., inflammation, hemorrhage, diarrhea, etc.), morbidity, and mortality of the affected host. Coccidia species infect chickens, canines, felines, rabbits, cattle, sheep and goats, swine, geese, mice, rats, frogs, guinea pigs and man, among others (**Radostitis et al., 2007; Yatusевич, 2006**). The disease is responsible for major losses due to mortality and primarily to the poor performance of sub clinically infected animals which costs the livestock industry many millions of dollars. Coccidiosis is most economical important in poultry production worldwide. Each year, over 50 billion chickens are raised as a source of meat, accounting for over one-third of protein food for humans (**Taylor et al., 2007**). Coccidiosis annually causes a global loss of over 2.4 billion US dollars in the poultry industry, including poor growth performance, replacement of chicks, and medication Very important is the finding that almost 70 percent of this estimated cost is due to subclinical coccidiosis, by impact on weight gain and feed conversion rate. One of the reasons for these remarkable findings is probably the difficult diagnosis

of subclinical coccidiosis, (**Sorensen et al., 2006; Williams, 1999**)

Infection of animals occurs after the ingestion of these oocysts along with water and / or food contaminated with fecal matter under favourable conditions, oocysts sporulate and become infective. Infection can be readily spread throughout an entire population of animals, such as, sheep and cattle in a feed lot or pasture, chickens in pens, and, canines and felines, through the release of coccidian oocysts in fecal waste, followed by oocyst ingestion by uninfected animals In young animals, the ingestion of oocysts attached to the mother's teats, soil or bedding, ingestion by licking their own hair or even ingestion through contact with oocyst-contaminated feeders can be of high significance in disease transmission (**Lima, 2004; Meskerem et al., 2013**). Control of coccidiosis should be relayed on accurate diagnosis. In recent years, different diagnostic tools such as fecal flotation techniques, morphological identification, molecular biology, biochemistry, cell biology and immunology have been used to expand greatly our knowledge of these parasites and the disease they cause (**Chapman et al., 2013**)

Etiology

The coccidian are members of the protistan phylum Apicomplexa, subclass Coccidiasina, intracellular parasites, characterized at some stage of the life cycle by a typical apical complex of organelles

at one end of the organism. Members of the genus *Eimeria* are homoxenous with sexual and asexual development taking place in a single host. The most prevalent species of *Eimeria* that cause coccidiosis in cattle are *Eimeria. bovis*, *E. zuernii*, and *E. auburnensis*. The species of *Eimeria* that affects chickens are *E. acervulina*; *E. brunetti*; *E. hagani*; *E. maxima*; *E. mivati*; *E. mitis*; *E. necatrix*; *E. praecox*; and *E. tenella* among which the most economically important species in poultry are *E. tenella*, *E. acervulina*, and *E. maxima* (Taylor *et al.*, 2007; Urquhart *et al.*, 2003).

Life cycle

The protozoan *Eimeria* parasites have a complex life cycle with one part outside the host and another part in the intestinal tract, mainly within the epithelial cells. The cycle start from the unsporulated oocysts shed into the environment in the faeces of infected animals. Oocysts are thick walled structures, adjusted to survival outside the host that serve to transfer the parasite to new hosts. Given the correct environmental conditions (warmth, oxygen and moisture), the oocysts sporulates and becomes infective. Sporulated oocysts can remain infectious for relatively long periods and can persist in the environment. After ingestion by a new host, the oocyst wall is cracked in the crop of the host under the influence of bile salts and gravel that is also ingested to facilitate digestion (Allen and Fetterer, 2002).

Once ingested by the host, the oocysts undergo a process of excystation. The sporozoites penetrate into an epithelial cell of the small intestine to transform into a schizont. Two asexual multiplication cycles (schizogonies) occur in the small intestine only, or in the small then large intestines, according to the *Eimeria* species. Eventually, the schizonts penetrate the epithelial cells (sexual phase or gamogony) that lead to the production of gamonts, gametes and then non sporulated oocysts that are released with the faecal matter (Graat *et al.*, 1997; Williams, 2005).

Clinical signs

Clinical signs of coccidiosis are due to destruction of the intestinal epithelium and, frequently, the underlying connective tissue of the mucosa. This may be accompanied by hemorrhage into the lumen of the intestine, catarrhal inflammation, and diarrhea. Clinical signs include diarrhea, fever, inappetence, weight loss, emaciation, and in extreme cases, death. However, many infections are subclinical (Aitken, 2007; Bujmehrani, 2011; Thebo *et al.*, 2007).

Pathogenesis

The pathogenesis and life cycles of coccidia must be understood in order to visualize their effects upon the host. Coccidia are obligate intracellular parasites whose development within the cytoplasm of epithelial cells results in the hyperplasia or death of each cell

that is parasitized. Mechanisms and degree of tissue damage depend on the species of *Eimeria* involved, the size of the infective dose of oocysts, stress, and various host-related factors including age, physical condition, genetic susceptibility, and degree of immunity that has developed from previous low levels of infection. (Urquhart *et al.*, 2003; Bhatia *et al.*, 2004).

The pathogenesis begins with infection of a cell in the intestinal mucosa, by a sporozoite released from a sporocyst in the lumen of the gut. When ingested by the host, the wall of the oocyst breaks down and the sporocysts are released. The sporozoites penetrate into an epithelial cell of the small intestine to transform into a first generation schizont. The schizonts produce motile merozoites, which may initiate another generation of schizonts in other intestinal cells or become gamont, gametes and then non sporulated oocysts that are released with the faecal matter. The second generation schizogony occurs usually in the large intestines followed by the release of another generation of merozoites, which invade epithelial cells and produce the sexual stages, the macrogametocyte and the microgametocyte. The second generation schizogony and fertilization of the macrogametocyte by the microgametocyte (gametogony) are the stages of the life cycle that cause functional and structural lesions of the large intestine (Khan *et al.*, 2011; Kaya, 2004).

Diagnosis

As indicated, next to the fact *Eimeria* are very effective parasites, one of the main reasons coccidiosis is still a major problem, is the difficult diagnosis. Diagnosis of coccidia is clinically non-specific. Several techniques have been developed for quantitative identification of *Eimeria* species (Allen and Fetterer, 2002).

Oocysts can be identified in feces by salt or sugar flotation methods. In some laboratories today simple flotation and light microscopy is still used as the method of detecting *Eimeria* species oocysts, but is not recommendable due to the low sensitivity (Taylor *et al.*, 2007).

Simple flotation and microscopical investigation is usually sufficient to identify *Eimeria* oocysts, but species differentiation is needed if they are to be associated as a cause of diarrhoea (Conway and McKenzie, 2007; Joyner and Long, 2008). *Eimeria* species may be distinguished on basis of their size and shape. Morphological differentiation by light microscopy is still gold standard for *Eimeria*, however, their reliability suffer from species cross reactivity. Unfortunately, these approaches can be unreliable, particularly because there can be an “overlap” in the sizes of oocysts of different species and multiple species of *Eimeria* can simultaneously

infect the host (Tafti and Mansourian, 2008; Umar *et al.*, 2014).

In routine diagnostics the gold standard is currently fluorescent antibodies specific for antigens on the oocysts surface (IFAT, Indirect Fluorescent Antibody Technique) that allow them to be easily identified in a fluorescence microscope. Though highly specific and sensitive, the method does not reveal morphological details well, and is thus often backed up by staining methods. The acid fast contrast staining method is used in many variations.

Methods of identifying species with PCR from faecal samples have been developed and are accurate, but are still time consuming and lack the important ability to estimate the infection intensity. The same problem can be noted for the immunochromatographic rapid assays (rapid tests) that qualitatively give good results but no quantitative data. Since all farms have coccidia present to larger or lesser extent the value of these tests is questionable (Conway and McKenzie, 2007; Brian, 2009).

Diagnosis based on characteristic lesions

Gross lesions

Gross lesions of coccidiosis are variable by host species, parasite species, and intestinal location. Lesions vary from proliferative in sheep and goats to necrotic-hemorrhagic in cattle, avian species, dogs and cat. In small ruminants, coccidial-induced enterocyte hyperplasia results in nodule formation and thickening of the intestinal wall can cause reduction in food absorption, emaciation, serous atrophy of fat, diarrhea, and dehydration. Gross lesions, even in heavily infested animals, may be minimal to absent. The minimal to moderate changes are observed as thickening of the intestinal mucosa associated with a few scattered whitish 1-2 mm in diameter non-pedunculated plaques or nodules. The large schizonts of some species are sometimes grossly visible as well (Mohammad, 2012).

Histo-pathological lesions

Although the main histopathological lesion of coccidiosis is hyperplastic or proliferative enteritis, the pathological changes vary in detail according to the species concerned. In most cases, loss of surface epithelial cells and villous atrophy are associated with first generation schizonts, while crypt destruction or hyperplasia is associated with gamonts. Histopathological lesions in case of caecal form revealed loss of epithelial tissue, congestion of blood vessels which indicated disruption followed by leakage of blood, severe muscular oedema, necrosis of submucosa, loss of villi, disruption of caecal mucosa, cluster of oocysts and marked haemorrhage, necrosis of caecal mucosa and lymphoid cells showing hyperplasia (Soomro *et al.*, 2001; Khan *et al.*, 2011).

Characteristic lesions to some of *Eimeria* species

Generally, it is agreed upon that from the species recognized in broiler chickens, the most pathogenic are *E. acervulina*, *E. maxima* and *E. tenella*. The latter is, amongst broiler farmers, the best known. It infects the caeca and because of its deep development in the mucosa and subsequent widespread damage with distinct gross lesions and loss of blood in the faeces.

On the other hand, when performing field necropsies on a larger scale, *E. tenella* appears to be the least prevalent of the three species mentioned. Also, the damage is being limited to the caeca, relative less important parts of the gut with regard to digestion and absorption, thus effects on growth and feed conversion rate (Joyner and Long, 2008; McDougald and Fitz-Coy, 2008).

The presence of clusters of large schizonts in the caecum is pathognomonic for *E. tenella*. *E. brunetti* oocysts are indistinguishable from those of *E. praecox*, *E. tenella* and *E. necatrix* based on size alone but the location in the lower gut and the appearance of the lesions could be used as reliable indicators. Light infections of *E. brunette* are overlooked easily unless careful attention is paid to the lower small intestine. Histopathology of *E. brunetti* reveals schizonts on the fourth day of infection (Jolley and Bardsley, 2006; Mathis, 2005).

The predilection site for *E. acervulina* is upper part of small intestine (duodenum) and in heavier infections also more caudal, interfering even with the ability for *E. maxima* to develop. The characteristic lesions include thickening of mucosal walls and 'white ladder lesions' produced by dense foci of gamonts and oocysts and a watery exudate are likely to be found in this case. *Eimeria maxima* cause petechiae, thickened walls and a pink exudate in the midgut. To assess the level of damage caused by these two species, lesion scoring can be performed. An important debate is still on going on what levels are to be considered clinical (and requiring treatment) and what levels are subclinical. Some consider lesions higher than 1.5 per species as indicative for clinical disease, and levels below as subclinical, not (Williams, 2005; Bowman *et al.*, 2003).

Eimeria acervulina affects the upper part of the small intestine. The characteristic lesions are small red spots and white bands on it. Microscopically duodenum of experimentally infected chicken with *E. acervulina* showed presence of hyperplastic changes in the epithelial mucosa with activation of goblet cells, sometimes there was epithelial desquamation. The lamina propria was infiltrated with inflammatory cells, accompanied with hemorrhagic areas. The histological finding of the middle part of small intestine of naturally infected balady chicks with *E. necatrix* showed its characteristic coagulative necrosis and focal hemorrhagic areas and deeply embedded

gametocyte in tunica muscosa and serosa (**Quiroz-Castañeda and Dantán-González, 2015; Thebo et al., 2007**).

Eimeriatenella: swollen caeca, thickened intestinal walls, dark colouring of damaged intestine containing a core of necrotic tissue and blood. *Eimerianecatrix*: mid-gut, the wall will show 'ballooning', white spots and petechiae form characteristic 'salt and pepper' lesions and there will be haemorrhage into the lumen. The mid-intestine was distended and exhibited pinpoint red and white spots visible from both the serosal and mucosal side. Large schizonts (>50 /im in diameter) were found in smears from the affected area, and 17 to 22 /im long oocysts were observed in the caeca. In case of *Eimeria brunette* numerous petechiae is apparent in the lower small intestine and the rectum (**Bujmehrani, 2011; Jalila et al., 1998**).

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