

Popular Article

Coccidiosis In Small Ruminants

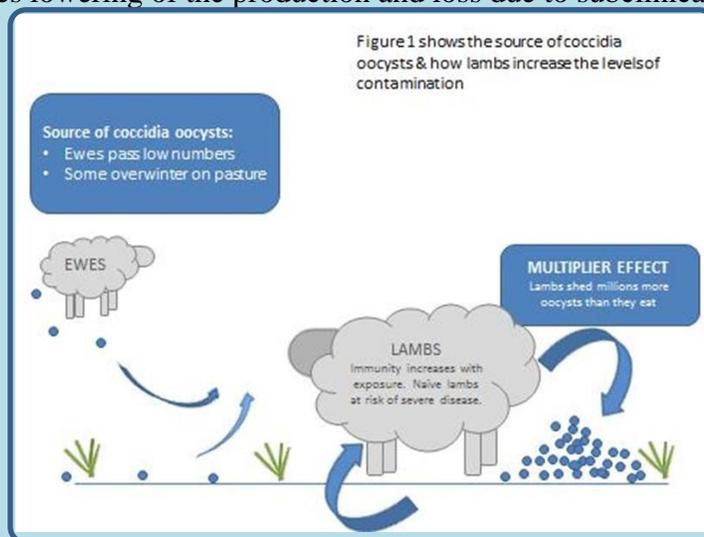
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Introduction

Coccidiosis is a common protozoan parasitic disease causing severe economic loss to the livestock sector. It is caused by a coccidian parasite of the genus *Eimeria*. There are several species of *Eimeria* found to affect sheep and goat, among them *E.ovinodalis* and *E.ahsata* are highly pathogenic in sheep whereas *E.arlongi*, *E.christenseni* and *E.ninakohlykimove* are found to be more pathogenic in goat. Most of the species of *Eimeria* are reported to be host specific with no cross infection between sheep and goat (Engidow *et al.*,2015). It is considered as an intracellular parasite with more affinity towards epithelial cells of the intestine. During their lifecycle they undergo a series of asexual reproductive stages followed by sexual stage resulting in oocyst formation. The infected animal passed out oocyst and result in contamination of feed, water and soil. The patent period for coccidiosis is 3 to 10 days but it depends on the host species, species, infective dose of the oocyst, condition and age of the host and other factors. Coccidiosis is an economically important because it causes lowering of the production and loss due to subclinical diseases.

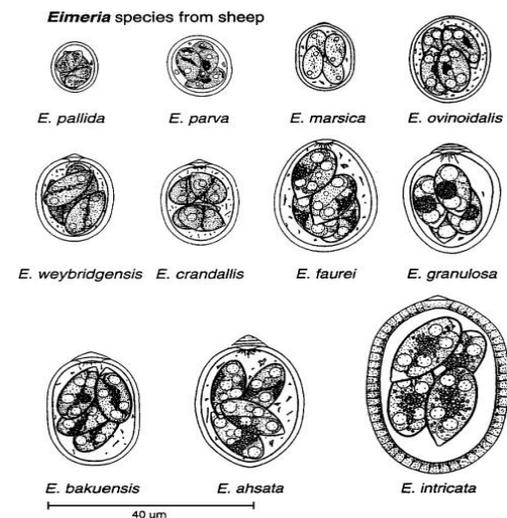
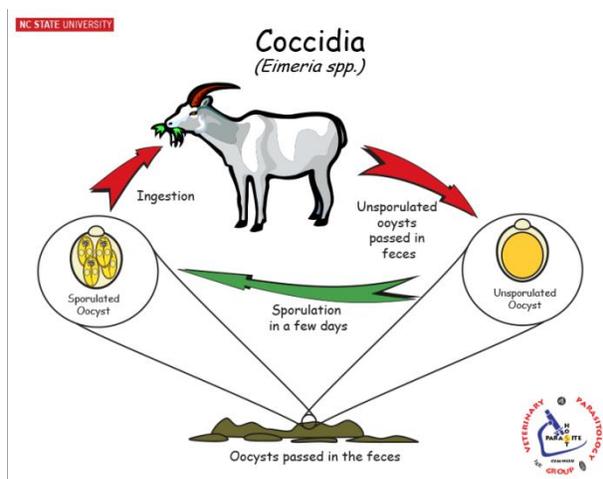
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(Source: <https://www.nadis.org.uk/disease-a-z/sheep>)

Lifecycle

The lifecycle of coccidia consist of two phases: exogenous phase and endogenous phase. The un-sporulated oocyst passed out through the faeces undergo sporulation in the environment. Endogenous phase starts with the ingestion of the sporulated oocyst which will then undergoes excystation in the intestine and form sporozoites. Sporozoites enter in to the intestinal epithelial cells where they form trophozoites and then to schizonts. The schizonts consist of many merozoites which will enter in to the neighboring epithelial cells and produce secondary schizonts. These schizonts produce second generation merozoites. These merozoites penetrate the large intestinal epithelial cells and form micro and macrogamonts which initiate the sexual development. Fusion of micro and macrogamete result in zygote formation and it forms a wall around that and leads to oocyst formation. Oocyst will be released in the lumen and passed out through the faeces (Lefevre, 2010).



(Adopted from North Carolina State University, Veterinary Parasitology)

(Courtesy: Small Ruminant Research Journal, <https://doi.org/10.1016/j.smallrumres.2011.10.02>)

Clinical signs

The infected animals exhibit signs like fever, depression, loss of appetite, yellow to dark watery diarrhea sometimes with clumps of mucus, progressive dehydration and emaciation and anemia.

Hematological studies reported reduction in RBC count and Hemoglobin count as well as increase in leukocyte count. Reduction in glucose and alkaline phosphatase level observed in biochemical studies (Singh *et al.*, 2016).

Gross and histopathology of coccidiosis in small ruminants

The lesions were predominantly noticed in jejunum and ileum and sometimes in caecum also. Macroscopically, there will be white to yellow colored raised pinpoint to larger nodular lesions may be evident in the mucosal layer. Other than that, there will be oedema, and congestion of serosa and mucosa (Sathish *et al.*,2015). In advanced cases the entire affected mucosa might be thickened due to mucosal hypertrophy and adenomatous like changes, cerebriform or gyrate pattern of projections and depressions could be evident in the serosal surface of the intestine (Khodakaram Tafti *et al.*,2008).

In Histopathology, the affected villi and crypts could be distended with developing stages of *Eimeria* and due to inflammatory reaction. The various developmental stages of *Eimeria* like micro and macrogametocytes, young and first generation schizonts with stage of blastophore formation, early stage of compartmentalization, advanced stage of compartmentalization, early and mature second generation with merozoites and immature oocyst can be observed in the affected portions. The large schizonts with numerous merozoites arranged in whorl pattern can also be seen (Khodakaram Tafti *et al.*,2008; Sathish *et al.*,2015). Local hypertrophy, hyperplasia of the villi, villous blunting, inflammatory cell infiltration in the lamina propria also reported (Sayin *et al.*,1980).

Sever non hemorrhagic typhlocolitis is a characteristic lesion in *E. ninakohlykimove* with a mortality rate of 30% in kids (Koudela and Bakowa,1998).

Treatment

Supportive care should be given to animals in acute cases. Broad spectrum antibiotics are advised to prevent secondary bacterial septicemia. Drugs commonly used for the treatment include sulfonamide, nitrofurans and quinolones (Engidow *et al.*, 2015).

Control measures

Good management techniques should be followed to prevent outbreaks of coccidiosis and minimize the effects of sub-clinical coccidiosis. Management should be aimed at reducing the fecal-to-oral transmission of the pathogen. Good sanitation and hygiene are essential. Maternity areas should be kept clean and dry. Lambing and kidding jugs



Diarrhoea and staining with fresh blood is highly suggestive of coccidiosis
(Source: <https://www.nadis.org.uk/disease-a-z/sheep>)

should be cleaned between litters. Pens should not be overcrowded. They should be kept dry and well-bedded. No feed should be fed on the ground or the floor of a pen. Feeders should be elevated or located on the outside of the pen. Water receptacles should be kept clean and free from fecal matter. Control measures include avoid overcrowding, provide clean bedding materials, ensure proper colostrum intake.

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