Chapter

Management and Control of *Eimeria* Infection in Goats

Saw Bawm and Lat Lat Htun

Abstract

Coccidian parasites of the genus *Eimeira* cause coccidiosis in farm animals, which develop in both the small and the large intestines. Coccidiosis is a major economic concern in many livestock, especially in young animals, as a result of losses caused by clinical infection (diarrhea) and subclinical (poor weight gain in particular) and the required treatment costs. Herein, we summarize geographical distribution of *Eimeria* parasites, their life cycle, pathogenesis, clinical signs, economic losses due to coccidiosis, diagnosis, recent information on control and prevention, and anticoccidial drugs for *Eimeria* infection in goats. With regard to poverty alleviation in most developing agricultural countries, it is important to maintain and develop goat-related industries. Proper management should be used to prevent losses and reduce the productivity from coccidiosis in young animals by: reducing the level of environmental contamination by infectious oocysts; minimizing stress; and avoiding overcrowding.

Keywords: *Eimeria* infection, goats, management, control, anticoccidial drugs

1. Introduction

The world populations of 2.3 billion small ruminants (goat and sheep) which comprise 1.09 billion goats [1] provide vital milk, meat and fiber. Goats (and sheep) are particularly important in Asia and Africa where they account for more than 90% and nearly 70% respectively of the world stocks [2]. Coccidian parasites of the genus *Eimeria* cause coccidiosis in small ruminants, which develop in both the small and the large intestines and specifically affect young animals [3]. In many livestock, especially in young animals, coccidiosis is of great economic importance as a result of losses due to clinical infection (diarrhea) and subclinical (poor weight gain in particular) and the required treatment costs. In goats, the majority of *Eimeria* infections are asymptomatic; notwithstanding, a few species have been linked to diarrhea and hindered growth [3, 4]. The coccidian are intracellular parasites, members of the protistan phylum Apicomplexa, subclass Coccidiasina. The genus *Eimeria* and *Isospora* are homoxenous, they develop both sexually and asexually in the same host [5]. Various *Eimeria* spp. are known to be involved in different ruminant hosts (bovine, ovine, caprine), however, because of the strict specificity of the host, no cross infection take place [3]. The *Eimeria* species do not transmit from animal species to another.

Studies in some countries such as Sri Lanka [6], Iraq [7], Jordan [8], Austria [9], Turkey [10], Saudi Arabia [11], China [12], Brazil [13] and Iran [14, 15] have shown that coccidiosis in goats is an important clinical and subclinical disease that may be linked to serious economic losses, especially under intensive breeding conditions with high animal density and high productivity [16]. Of the 16 *Eimeria* species identified in goats worldwide, *E. arloingi, E. ninakohlyakimovae, E. christenseni*, and *E. caprina* were considered being the most pathogenic species [17–20]. According to reports [3, 21], the common species of *Eimeria* in goats are listed in **Table 1**.

1.1 Geographical distribution

In temperate areas including Europe, *E. ninakohlyakimovae*, *E. arloingi, E. christenseni, E. jolchijevi, E. alijevi, E. caprina and E. caprovina* are the most prevalent *Eimeria* species in goats [22]. In semi-arid zones (Gran Canaria, Spain), the most common *Eimeria* spp. are *E. ninakohlyakimovae, E. arloingi* and *E. alijevi* [4]. In USA (mid-western states), *E. arloingi* (98.8%), *E. christenseni* (58.2%), *E. ninakohlyakimovae* and *E. parva* (33.3%) are the most frequent *Eimeria* spp. found in goats [23]. In dry tropical areas such as Senegal, the common coccidia species are *E. arloingi* (64%) and *E. ninakohlyakimovae* (56%) [24]. Similar findings have been reported in Ghana [25], Nigeria [26], Kenya [27, 28] and Zimbabwe [29]. In dry areas of Sri Lanka, *E. ninakohlyakimovae* (31%) *E. alijevi* (29%) and *E. arloingi* (21%) are the three most prevalent coccidia [6]. In Myanmar, the most common species of *Eimeria* found in goats are *E. arloingi* (25.4%), followed by *E. hirci* (20.7%) and *E. christenseni* (13.9%) [30]. High prevalence of *Eimeria* species infections, *E. arloingi* (64–80%) and *E. christenseni* (60%), have been reported in dry tropical areas of Africa (Senegal) [24], Nigeria [26], and Zimbabwe [29]. The occurrence of mixed-species infection was higher than single-species infection [12, 26, 31]. Coccidia of small ruminants, therefore, exist globally, and it appears difficult to say that there is any specific geographical distribution for one or the other species of coccidia.

1.2 Life cycle

Eimeria usually needs only one host in which to complete their life cycle. Within a host's intestinal cells, two stages, schizogony/merogony and gamogony grow up.

Organism	Distribution	Remark	Reference
E. ninakohlyakimovae	USA, European countries	Pathogenic	[3, 21]
E. arloingi	USA, Australia, Asia	Pathogenic	[3, 21]
E. caprina	USA, Spain, Africa	Pathogenic	[3, 21]
E. christenseni	US, Africa, Asia, Australia	Pathogenic	[3, 21]
E. parva	Africa, Asia	Benign	[3, 21]
E. hirci	Africa, Asia, Australia	Benign	[3, 21]
E. jolchijevi	Australia	Benign	[3, 21]
E. apsheronica	Africa	Benign	[3, 21]
E. alijevi	Asia	Benign	[3, 21]
E. caprovina	Africa	Benign	[3, 21]
E. capralis	-	-	[21]
E. charlestoni	-	-	[21]
E. masseyensis	-	-	[21]
E. pallida	-	-	[21]
E. punctata	-	-	[21]

Table 1.
Common species of Eimeria *in goats.*

Sporogony/sporulation, on the other hand, takes place outside of the host within an oocyst protecting infectious sporozoites. The life cycle involves an extracellular oocyst maturation stage (sporogony) as well as a parasitic intracellular stage inside the host with a sexual reproduction accompanied by an asexual [16].

When the unsporulated oocysts pass through the feces, after 2–7 days they become infected depends on *Eimeria* species and environment. The first single cell is divided into four sporoblasts, each of which grows into single sporocyst with two sporozoites [32]. The sporulated oocysts are resistant to adverse environmental conditions. They are able to live for several months or even over a year. Extreme desiccation, direct exposure to the sunlight, however, limits the survival of the oocysts, and temperatures below −30°C or above 63°C are lethal for the oocysts. The oocyst has thick-wall and usually ovoid form [16].

After ingestion by the host, the walls of the oocyst break down, releasing sporozoites from the sporocysts. The sporozoites penetrate the small intestine through an epithelial cell and develop into schizont of the first generation. The schizonts release motile merozoites, which may either initiate a second generation of schizonts or develop into gamont, gametes and then non-sporulated oocysts, which are released with the feces. The schizogony of second generation in the large intestines usually occurs with another generation of merozoites invades epithelial cells and develops the sexual stages, the gametocytes, male (microgametocytes) and female (macrogametocytes). Second-generation schizogony and fertilization of the gametocytes (gametogony) cause functional and systemic lesions of the large intestine [16]. In general, prepatent period for *Eimeria* species in goats is approximately 19 days [33]; however, the prepatent period is 20 days, 14–23 days, 10–13 days and 17–20 days for *E. arloingi*, *E. christenseni*, *E. ninakohlyakimovae* and *E. capria*, respectively [34]. Life cycle of *Eimeria* spp. in goat is shown in **Figure 1**.

1.3 Pathogenesis and pathology

The pathological and clinical outcomes are influenced by a number of factors such as the present *Eimeria* species, infection dose, its replication potential, inflammatory immune and concurrent infections by other pathogens as well as management and related stress. Due to the intracellular localization of all internal

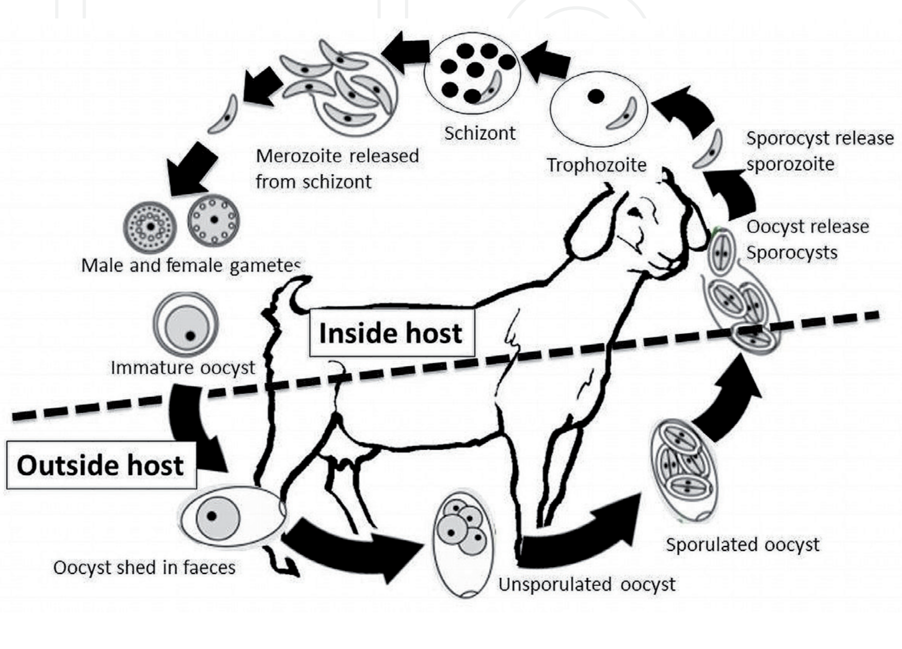

Figure 1.
Life cycle of Eimeria *species in goat. This figure was redrawn based on the picture in Taylor et al. [21].*

developmental stages, significant damage to the intestinal mucosa occurs. The outcome of *Eimeria* infections can vary greatly by parasite species as well as by individual host animal and farm. The damage caused by the parasite infection and replication occurs most during the late schizogony and gamogony [35]. This is due to the multiplication that the parasite undergoes during its first schizogony, leading to an exponential increase in the number of intestinal cells during subsequent multiplication. As a result, most of the damage occurs shortly before oocyst excretion starts in affected animals.

In early infections with *E. ninakohlyakimovae* or *E. caprina* in goat kids are characterized by haemorrhagic enteritis [36]. Polyps in the small intestine may develop as a result of *E. arloingi* infections [18], and *E. apsheronica* induce formation of white nodules in the mucosa that are visible from the serosal surface [37].

1.4 Clinical signs

Eimeria ninakohlyakimovae and *E. caprina* are highly pathogenic species capable of inducing watery to bloody diarrhea [13]. The infections with *E. arloingi* can also lead to watery diarrhea [38]. Acute symptoms may not exist in older animals at all or only in a small numbers of the affected goats following reinfection. Subclinical coccidiosis following infection with pathogenic species can be expected to produce subacute to chronically adverse impacts on the health, feed efficiency, and prolonged productivity [28]. Coccidia can invade and kill hosts' intestinal cells that cause anemia, loss of electrolyte and poor absorption of nutrients.

Most affected goats show diarrhea, poor growth rate, weakness and rough hair coat [12]. The feces are soft, watery and have clumps of mucus and color shifts from brown to yellow or dark tarry [18]. Loss of weight and dehydration are noted. Because the appetite is decreased, the animals' general condition is worsened. In some cases, sudden deaths occur in young animals between 2 and 4 months old without preceding digestive signs [3]. Diarrhea with or without mucus or blood, dehydration, emaciation, fatigue, and death are common clinical signs. But in fact, some goats are constipated and die acutely without diarrhea.

Impairment of growth is the major sign in subclinical form of coccidiosis. Early signs in acute cases include decreased appetite, listlessness, fatigue and abdominal pain which can be manifested by crying and repeated rising up and lying down. First, the feces may be unpelleted, then pasty, and eventually watery yellowish-green or brown diarrhea. The typical characteristics associated with coccidiosis are diarrhea that may be mucoid or bloody, abdominal pain, tenesmus, loss of appetite, fatigue, weight loss, rough hair coat, dehydration and anemia. Fever, ocular and nasal discharges can occur in the acute disease. Clinical coccidiosis is common in lambs 4–6 weeks old. Acute, bloody diarrhea can occur in severe cases, as a result of extensive damage to the intestinal epithelium [39].

While coccidiosis is self-limiting, the clinical presentation can be exacerbated further by other enteric pathogens. Exposure to low-grade challenges results in development of a strong immunity against to the disease. Successive infections in young animals might lead to the excretion of a large numbers of oocysts in animals, which leads to heavy contamination of houses, pastures or watering places [34].

2. Materials and methods

The present chapter intends to give a comprehensive approach of the importance of *Eimeria* parasite in goats, pathogenesis, clinical signs, diagnosis and economic losses due to coccidiosis. In addition, this chapter aims to explore the recent information on control and prevention, and anticoccidial drugs for *Eimeria* infection in goats. The databases were searched from PubMed and Google Scholar search. Search terms were "*Eimeria* in goats" and "coccidiosis in goats". A total of 130 articles were retrieved from the search. These studies were conduct in different regions of the world with different breeds of goats. Findings of anticoccidial studies carried out in the author's laboratory were also included.

3. Economic impact

Some assumed parameters for estimation of economic losses are summarized in **Table 2**. There are not well documented economic impacts of coccidiosis in small ruminants and no published data for economic losses due to subclinical or clinical disease in tropical regions are available. While subclinical coccidiosis might not be of great importance, it cannot be compared to other infections [5]. Where the high density of animals with high productivity can cause coccidiosis to become an infection of great economic significance in small ruminants [16]. In the case of a mild infection, these losses can be attributed to reduce productivity with no clinical signs.

Global sheep and goats production was being estimated to lose up to $140 million per year [40]. Losses result from mortality, treatment costs for animals with diarrhea, enhanced sensitivity to secondary infections in infected animals, and reduced production efficiency. Beside the acute impacts, a prolonged effect was suggested as regards a lower feed efficiency, less final growth performance, and a reduced reproductive performance for life [41]. Subclinical coccidiosis is believed to cause higher production losses than clinical coccidiosis, since animals are infected and are affected for long-term [42].

No.	Parameters	Possible impact	Reference
1.	Poor feed conversion rate	Poor weight gain	[3, 41]
2.	Poor body weight gain	Poor weight gain	[3, 41]
3.	Weight loss	Poor weight gain	[3, 41]
4.	Intercurrent or concurrent diseases	Secondary infections, the cost of treatment	[16]
5.	Reduced productions (milk, wool, hair)	Reduced income	[3]
6.	Losses due to mortality	Production loss	[16]
7	Reduced carcass quality	Reduced income	[3]
8	Reduced fertility	Production loss	[41]

Table 2.
Assumed parameters for estimation of economic losses due to coccidiosis in goats.

4. Diagnosis

There are different diagnostic methods available for specific identification of *Eimeria*. They are based mainly on clinical observations. The most direct, definitive, and cost-effective method of diagnosis continues to be the microscopic examination of diarrhoeal or bloody feces from young animals [42]. Traditional methods are primarily based upon microscopic oocysts morphological features, parasite biology, clinical signs in animals affected, and typical macroscopic lesions assessed by lesion scores [44]. In general, fecal samples are collected and analyzed

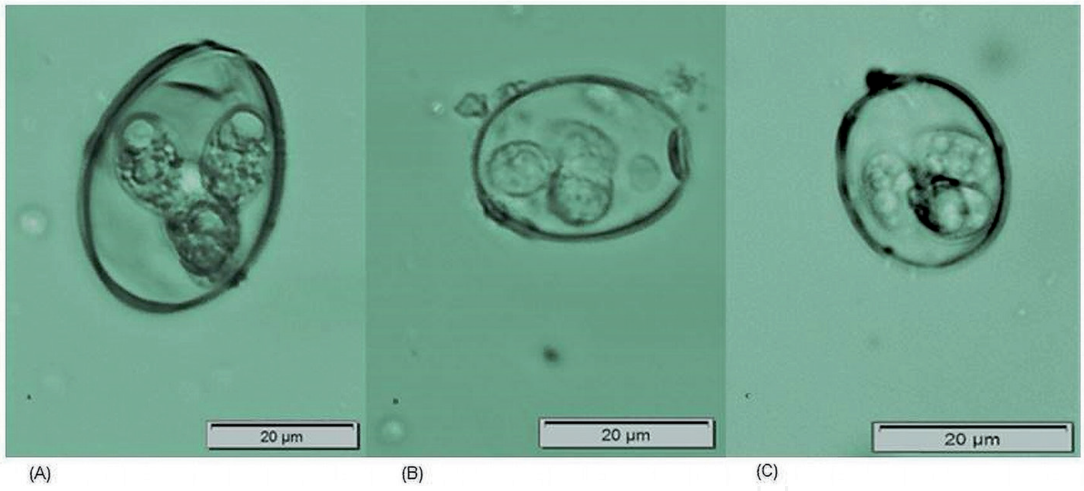

Figure 2.
Oocysts of E. christenseni *(A),* E. arloingi *(B) and* E. hirci *(C) from goats in Myanmar identified by Bawm et al. [30].*

for the presence of *Eimeria* oocysts (**Figure 2**). Fecal flotation enables oocysts to concentrate and increases their sensitivity significantly. Moreover, species-level identification should be performed. As many of the *Eimeria* species are either not or mildly pathogenic, it is possible to determine whether the *Eimeria* oocysts found are associated with a clinical disease or only an accidental finding and whether the cause of this disease requires more study. Pooled fecal samples can be used for monitoring purposes, while clinically ill should be individually sampled so that the excreted amount of *Eimeria* oocysts can be easily estimated. Accurate quantification of oocyst excretion in individual samples from animals in the focus groups can be helpful. There are several quantitative flotation methods available, with the McMaster counting technique being the most common method one [8].

However, the morphological approach is not entirely accurate due to the prevalence of intraspecies heterogeneity, as natural *Eimeria* infections are typically mixed with more than one species and some species have confounding characteristics [43, 44]. In addition, morphological observations are very labour-intensive in conjunction with fecal analysis and require a professional method of classification. In order to overcome the limitations of conventional methods [45–47], molecular techniques have been identified as useful for species identification or classification of this genus and have further demonstrated the phylogenetic location of each *Eimeria* species and phylogenetic clades [48, 49]. Molecular characterization of *Eimeria* goat species has been recorded in Australia [50], India [51], Iran [52] and Myanmar [30].

5. Prevention and control

Coccidiosis in ruminant is usually controlled by a combination of good management and treatment with anticoccidial drugs or prophylactics [42]. In general, effective control of coccidiosis is not based on the complete removal of *Eimeria* from the affected premises. It is considered neither possible nor useful to avoid contact between the naïve hosts and *Eimeria* for the operation of this parasite. Low-dose infections are generally not linked to disease, and low-dose infections are beneficial to the host because they allow the host to develop a protective, non-sterile immunity and protect against future infections. Therefore, instead of pathogen eradication, the emphasis of ruminant coccidiosis management lies in reducing the infection burden to uncritical levels and endemic stability [42].

Prevention is mainly based on the management of herds, including hygienic measures, since no vaccine is available. Coccidiosis outbreaks are a herd-level problem that is driven by stress. Infected animals need a responsive immune system to prevent severe disease. Therefore, minimizing or eliminating stressors like diet changes, harsh climate conditions, crowding, frequent shipping, animal grouping and exposure to other disease pathogens is a crucial part in preventing disease [16]. It is important to ensure adequate nutrition and appropriate uses of anticoccidial drugs. Colostrum uptake is important for newborn ruminants as it protects them from pathogens. Although the protection against *Eimeria* is not efficient, the protection from other pathogens supports and prevents problems in the animals' immune system.

Proper hygiene and minimizing predisposing factors in the environment are important for the control strategies of coccidiosis [53]. Pens for lambing and kidding should be kept clean, and bedding should be disposed when old or infested with oocysts. The washing and disinfecting of the buildings must be done with boiling water under pressure and gaseous ammonia [54].

All steps that minimize the amount of fecal contamination on hair coats should be routinely applied. Feed and water troughs should be high sufficient to prevent heavy fecal contamination. Feeding animals on the ground should be avoided, particularly when overcrowding. The regular rotation of pastures for parasite control will also assist in the controls of coccidial infection [53].

6. Anticoccidial drugs

With respect to available anticoccidials, they are usually supplied as feed additives, in drinking water, or in feed supplements such as salt. As a result, their use is often most feasible in weaned animals. However, anticoccidial prevention may need to start in the first weeks of life depending on the procedure [3]. In general, coccidiostats and coccidiocidal drugs are available. The development of internal coccidia stages is inhibited by coccidiostats, while coccidiocidal drugs kill the parasites. Drugs of both modes of action are currently available on the market. The best time to administer a prophylactic treatment would allow for the infection to develop but not for full parasite development. An appropriate treatment given following or prior to infection by the onset of oocyst release and appearance of symptoms is known as metaphylaxis. Anticoccidial therapy would therefore be optimally applied after infection, for the most important ruminant *Eimeria* species, approximately 14 days after infection, when the first and second merogonies develop. The main advantage of metaphylaxis over prophylaxis is the development of immunity and protection against reinfection, which is unlikely to make any subsequent anticoccidial treatments necessary. In general, it is important to apply prophylactic and metaphylactic treatment on the basis of a herd or animal group, as infected animals cannot be determined and all animals living in the same contaminated environment would be exposed to *Eimeria* infections. Continued use of coccidiostats reduces the number of oocysts passed through the feces over time, but may also lead to selection for resistance and therefore a regular monitoring of the treated animals is needed [16].

Anticoccidial drugs belong to one of two categories [55, 56, 63]:

1. Polyether antibiotics or ionophores produced by the *Streptomyces* spp. or *Actinomadura* spp. Ion gradients across the parasite cell membrane are interrupted by these drugs:

    a. Monovalent ionophores (monensin, narasin, salinomycin)

   b. Monovalent glycosidic ionophores (maduramicin, semduramicin)

   c. Divalent ionophore (lasalocid)

2. Synthetic compounds produced by chemical synthesis with a particular mode of action, which are often referred to as "chemicals":

   a. Inhibition of parasite mitochondrial respiration (decoquinate, clopidol)

   b. Inhibition of the folic acid pathway (sulfonamides)

   c. Competitive inhibition of thiamine uptake (amprolium)

   d. Inhibition of respiratory chain enzymes and nuclear division of protozoan (e.g., diclazuril, halofuginone, nicarbazin, robenidine)

Polyether ionophores inhibit the growth of sporozoites by increasing the concentration of intracellular Na^+ ions. They also accelerate the activity of Na+/K+/ATPase [57] and affect merozoites by inducing the breakup of the cell membrane [58]. Monensin was the first antibiotic to show an anticoccidial effect at reasonable concentrations, allowing it to be used in feed [59]. It can act as an effective anticoccidial agent for coccidiosis caused by *E. crandallis*, *E. christenseni* and *E. ninakohlyakimovae* in goats kept in confined space [60].

Monensin fed prophylactically at 20 g per ton of feed for 28 days decreases shedding of oocysts and improves feed conversion. However, high monensin levels make the feed unpalatable and toxic [53, 60]. It is believed that toxic effects in the horse, cattle, dogs, cats, rats, avian species and goat are mediated by interference with cell membranes ion gradients, inducing mitochondrial disruption and thus depleting of cellular energy [61, 62]. The documented toxic effects include heart toxicity, muscle degeneration and neuropathy, the latter is shown by myelin and ataxia [62].

Quinolones, pyridones, alkaloids, guanidines, thiamine analogues, and triazine derivatives are examples of synthetic anticoccidial drugs. Triazines inhibit nuclear division of protozoan thus interfere with the development of schizonts and gametocytes [63]. Decoquinate (0.5 mg/kg BW) and lasalocid at a dose of 25-100 mg/kg feed can be used to treat coccidiosis from weaning to market [53]. Sulfonamides at dosage rates of 25 to 35 mg/kg BW for at least 15 days are effective against coccidiosis in small ruminants. The combination of chlortetracycline and sulfonamide has provided protection in lambs. Other drugs include monensin (20 and 16 g/ton of feed for sheep and goats, respectively), toltrazuril (20 mg/kg BW as a single oral dose) and diclazuril (2 mg/kg BW as a double oral dose) [34, 53, 64].

Amprolium in feed is also used to treat the disease in goats (100 mg/kg BW for 21 days) and sheep (50 mg/kg BW for 21 days) [53]. Amprolium is structurally related to thiamine, and it is believed to be associated with a competitive inhibition of successful transportation of thiamine into parasite [65]. The production of oocyst in lambs has proved to be decreased when given as an in-feed medicine and clinical coccidiosis outbreaks have been successfully controlled by single drenching [66]. Young *et al.* [65] also stated that reductions in oocyst production have been detected in goats.

Diclazuril and toltrazuril have shown in several studies to decrease production of oocysts in natural and artificial *Eimeria* infections when orally given to young cattle, pigs, or lambs prior to the onset of clinical signs [67–71]. The molecules of decoquinate, toltrazuril, and diclazuril act on the whole coccidial cycle and allows curative as well as preventive action [53, 72]. According to our recent study [73], among the treated group of goats with monensin, toltrazuril, and amprolium, the percentage reduction in the number of fecal oocyst in the toltrazuril- treated group was found to be observed the highest (92%).

Resistant problems have been reported for some anticoccidial drugs, such as arprinocid and quinolone buquinolate [74]. In the field trials, toltrazuril resistance did not exist in at least five consecutive drug exposures [75]. The polyether ionophores became the drug of choice in 1972 and, as of today, are now the most commonly used drugs in poultry. Although ionophores resistance is likely to develop slowly due to their specific mode of action, resistance development in synthetic drugs with a specific mode of action appears to be faster, involving genetic mechanisms [76].

7. Conclusion

In this review, we highlight the management and control of *Eimeria* parasite in goats, the causative agent of coccidiosis, which is of great economic importance as a result of losses due to clinical diseases (diarrhea) and subclinical (poor weight gain) and the required treatment costs. We summarize the geographical distribution of *Eimeria* parasites, their life cycle, pathogenesis, clinical signs, economic losses due to coccidiosis, diagnosis, recent information on control and prevention, and anticoccidial drugs for *Eimeria* infection in goats. With regard to poverty alleviation in most developing agricultural countries, it is important to maintain and develop goat-related industries. Proper management should be used to prevent losses and reduced productivity from coccidiosis in young animals by reducing the level of environmental contamination by infectious oocysts, minimizing stress, and avoiding overcrowding [3]. It is essential to be aware of the problem and to implement control strategies, such as the maintenance of hygienic conditions and use of anticoccidial drugs.

Conflict of interest

The authors declare no conflict of interest.

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