COCCIDIOSIS AND CRYPTOSPORIDIOSIS IN GOATS

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Carlisle – April 2017.

INTRODUCTION

Infections with apicomplexan coccidian parasites belonging to the genera, *Eimeria* and *Cryptosporidium* are common in goats. Coccidiosis is one of the most important diseases of kids in the first few months of life and cryptosporidiosis is often associated with neonatal diarrhoea. All kids may become infected, but the presence of infection does not necessarily lead to the development of clinical signs of disease as in many situations low levels of challenge will stimulate protective immune responses. Heavily contaminated environments predispose to disease with disease dependent on husbandry and management factors, particularly hygiene and stocking densities.

COCCIDIOSIS

As in other ruminants, coccidiosis is caused by species of *Eimeria*, of which nine species have been identified in goats in the UK (Norton 1986). Coccidia are normally present in goats of all ages and cause no clinical signs as immunity is quickly acquired, and maintained by continuous exposure to re-infection. However, modern goat husbandry systems may alter the delicate balance between immunity and disease with profound consequences for young kids.
It was thought for many years that the species of coccidia affecting sheep and goats were the same. Rigorous cross-transmission studies, however, have shown that although morphologically similar, the species of coccidia of sheep and goats are unique, host-specific species with only one exception *(Eimeria caprovina)*. Thus, there is no transmission of infection between sheep and goats, or goats and any other species of animals.

Life Cycle

Infection occurs following the ingestion of sporulated oocysts. From the oocyst, sporozoites emerge in the small intestine and invade host cells in the gut mucosa. There then follows several merogony stages of which the first produces giant meronts which release large numbers of merozoites that re-invade the mucosa and produce a second generation of small meronts. These produce merozoites that invade the epithelium and produce male and female gamonts, which give rise to oocysts which pass out with the faeces. Once outside the body, the parasite divides to form a fully sporulated an infective oocyst. The life cycle takes between 2-4 weeks depending on the species of *Eimeria.* Sporulation outside the body may be complete in a day or two under ideal conditions, but can take several weeks in cold weather. Each stage of individual coccidia species has its preferences as to which cells and which parts of the gut

it infects. Those infecting the posterior parts of the intestine tend to be more harmful.

Pathogenesis

Development of disease is dependent on levels of oocyst contamination and parasite challenge. As seen in lambs, the most pathogenic species of coccidia are those that infect and destroy the crypt cells of the large intestinal mucosa. This is because in ruminants, the small intestine is very long, providing many host cells that provide the potential for enormous parasite replication with minimal damage. If the absorption of nutrients is impaired, the large intestine is, to some extent, capable of compensating. Coccidia that invade the large intestine are more likely to cause pathological changes, particularly if large numbers of oocysts are ingested over short periods. Here, the rate of cellular turnover is much lower and there is no compensation effect from other regions of the gut (Taylor 2002). In heavily infected kids, the mucosa becomes completely denuded resulting in severe haemorrhage and impaired water resorption leading to diarrhoea, dehydration and death.

Clinical and Pathological Signs

Clinical signs vary from loss of pellet formation, weight loss, anorexia and diarrhoea (with or without blood). On post-mortem, lesions may be seen in the small and/or large intestine or there may be little to see beyond thickening and petechiation of the bowel, but mucosal scrapings will reveal masses of gamonts and oocysts. Giant meronts may be seen in the mucosa of the small intestine as pin-point white spots, but unless they are in vast numbers they cause little harm. The most pathogenic stages are the gamonts.

Little is known about the pathogenicity of different species in goats. *Eimeria ninakohlyakimovae* is the most pathogenic species.

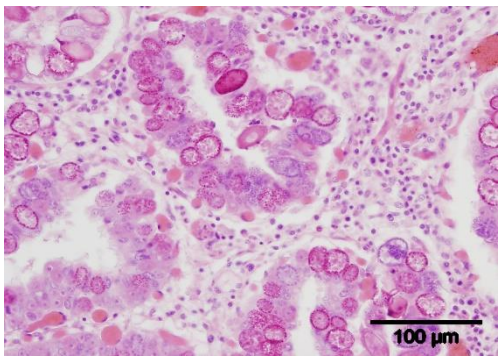

Fig.1 Gamonts of *E. ninakohlyakimovae*

This species and *E. caprina* can cause widespread denudation of the mucosa in the upper and lower large intestine, respectively (Gregory and Norton 1986). Histological sections may show loss of epithelial cells in the crypts of the mucosa, which may lead to electrolyte imbalance manifest as hyperkalaemia, hyponatraemia or hypophosphataemia. Other species that may be pathogenic are *E. christenseni* and *E. hirci* (Taylor et al. 2016). *Eimeria arloingi* is probably the most commonly encountered coccidia causing polyp formation and hyperplasia of the mucosa.

Epidemiology

Adult animals are highly resistant to the disease, but not totally resistant to infection. As a result, small numbers of parasites manage to complete their life cycle throughout the goats' life and usually cause no detectable harm. Wild goats (or those under extensive systems of management) with kids at foot, will produce small numbers of oocysts and these offer the kids an opportunity to acquire a protective resistance. The fact that they browse and move from place to place prevents the build-up oocysts, or limits the level of exposure to infective oocysts. The domestic goat kid in contrast, is born into a potentially heavily contaminated environment, and where numbers of sporulated oocysts are high, so disease often occurs. The dam, although possibly the original source of infective oocysts in the environment, may not be responsible for the heavy levels of contamination that kids may encounter. The source is often the kid itself, which following an initial infection in the first few days of life, may produce millions of oocysts within its own environment. The kid may then face potentially lethal doses of infective oocysts three weeks later when its natural resistance is at its lowest. Later born kids introduced into the same environment are immediately exposed to heavy oocyst challenge. Under unhygienic, overcrowded conditions, the kids will be exposed to and ingest a sizeable proportion of this infection and will develop severe disease and may even die from the infection. Under less crowded, more hygienic conditions, the infective dose ingested by kids will be lower, they may show moderate, slight or no clinical signs and develop an immunity to reinfection, but they in turn will have multiplied the infection a million-fold. Stress factors, such a poor milk supply, weaning, cold-weather and transport, will reduce any resistance the kid may have had, and can exacerbate the condition.

Management factors associated with the development of high-levels of infection and the development of disease:

a) pens not cleaned on a regular basis;
b) kids kept in overcrowded conditions;
c) kids housed together in different age groups.

Coccidiosis may be seen either indoors or out on pasture. Indoor coccidiosis develops in goats in an exactly comparable manner to that seen in lambs in the lambing pens. A major problem in goat herds is that, to ensure a constant year-round milk supply, kidding often takes place over an extended period. If the same pens are used constantly for successive batches of kids, or if young kids are added to a pen already housing older kids, then the later born kids are immediately exposed to heavy challenge and can show severe coccidiosis within

the first few weeks of life.

Coccidiosis can also occur in kids at pasture in the same way. On well-maintained pastures, where the grass is long, the oocysts lying on the soil surface will not be available for ingestion in large numbers by the kids. Under these circumstances the kids will ingest low levels of infection and develop immunity with time. However, under conditions where the grass is short, i.e. during dry weather in spring, summer or autumn or on heavily stocked, overgrazed pastures the oocysts will become available for ingestion in large numbers and coccidiosis will develop. On permanent pastures grazed the previous year, kids turned out in the spring may be exposed to oocysts that have over-wintered on the pasture.

Diagnosis

The diagnosis of coccidiosis in goats should be based on history, clinical and pathological signs as well as faecal oocyst counts and the species of coccidia present. Counts of faecal oocysts identified to species can help to complete the picture, but oocyst numbers may be grossly misleading when considered in isolation. Healthy animals may pass more than a million oocysts per gram of faeces whereas in a kid dying of coccidiosis the count may be <10,000opg. High counts of non-pathogenic species could mask significant numbers of *E. ninakohlyakimovae,* for instance, and give the impression that the abundant species was the abundant species.

DIAGNOSIS OF COCCIDIOSIS IN GOATS

Diagnosis should be based on **history**, **clinical signs** (severe diarrhoea in young kids), **post-mortem findings** (inflammation, hyperaemia and thickening of caecum) **supported by** oocyst counts and **speciation** (*E. ninakohlyakimovae, E. caprina;* possibly *E. christenseni* and *E. hirci* predominate).

Prevention and Control

Kids particularly at risk from coccidiosis are those kept indoors on damp bedding, or those on contaminated heavily stocked pastures particularly in cold wet weather. The incidence of disease can be reduced through avoidance of overcrowding and stress, and attention to hygiene.

Outbreaks of clinical coccidiosis can appear suddenly and may prove troublesome to resolve as they often occur on heavily stocked farms, particularly where good husbandry and management are lacking. If deaths are occurring, early confirmation of the diagnosis is vital and should be based on history, post mortem examination and intestinal smears. Affected animals should be medicated and moved to uncontaminated pens or pasture as soon as possible. There are no drugs licensed specifically for the treatment of coccidiosis in goats. Decoquinate (Deccox) is used as a feed additive for the treatment of clinical disease in both lambs and calves. Diclazuril (Vecoxan, Elanco) and toltrazuril (Baycox, Bayer, Tolracol, Krka UK) are available for the treatment of coccidiosis

in lambs. Little information is available on the efficacy of these compounds in goats, although studies in Gran Canaria, Spain have shown dose rates of 1-2mg/kg diclazuril to be effective against natural infections of coccidia in goat kids 2-4 weeks of age (Ruiz et al. 2012).

Where these are used off license, all kids in a group should be treated as even those showing no symptoms are likely to be infected. Severely infected animals that are diarrhoeic and dehydrated may require oral or intravenous re-hydration. Where non-specific symptoms of weight loss or ill-thrift are present, it is important to investigate all potential causes and seek laboratory confirmation. If coccidiosis is considered significant, much can be done through advice on management and instigation of preventative measures outlined above. Batch rearing of kids of similar ages, limits the build-up and spread of oocysts and allows targeting of treatment to susceptible age groups during the danger periods.

CRYPTOSPORIDIOSIS

Parasites of the genus, *Cryptosporidium* are small intracellular parasites that occur throughout the animal kingdom and have been reported in many species of mammals, birds, reptiles and fish (Fayer and Ungar 1986). The most important is *C. parvum,* which infects a wide range of mammals and is highly prevalent in ruminants, particularly young calves, lambs and kids. The life cycle of *C. parvum* is essentially like that described for coccidia but, unlike *Eimeria* spp., the oocysts are fully sporulated and are immediately infective when excreted.

Epidemiology

In goats, infection with *C. parvum* is seen in young kids in the first few weeks of life. The first kids to be born often become infected without showing clinical signs but become sources of infection for kids that follow. Infection spreads rapidly, and later-born kids can become so heavily infected that clinical disease results. In many instances where *Cryptosporidium* is diagnosed in animals, it appears that infections usually originate from the same host species. The primary route of infection is mainly by the direct animal-to-animal faecal-oral route. Infection in these cases is likely to occur through grooming, nuzzling, coprophagy, or by faecal soiling by direct contact with infected animals. Infection is often exacerbated by the presence of a range of bacterial and viral pathogens such as rotavirus, coronavirus, *Salmonella* spp. and enteropathogenic strains of *Escherichia coli*. Clinical signs where they do occur include profuse watery diarrhoea, abdominal pain, unthriftiness and dehydration, which if left untreated can lead to death.

Treatment and Control

There are no specific therapies for cryptosporidiosis and treatment relies on the use of supportive antidiarrhoeals and fluid therapy for animals showing dehydration.

OVERALL CONCLUSIONS

Coccidiosis is one of the most important diseases in kids during the first few months of life and cryptosporidiosis is often associated with neonatal diarrhoea. Heavily contaminated environments due to husbandry and management factors, particularly hygiene and stocking densities, predispose to both these diseases. There are no drugs licensed specifically for the treatment of coccidiosis in goats, although products licensed for use in lambs may be used under the cascade, although little information is available on the efficacy of anticoccidials in goats. There are no specific therapies for cryptosporidiosis and treatment relies on the use of supportive antidiarrhoeals and fluid therapy for animals showing dehydration.

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