# Coccidiosis in macropods and other species

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**Abstract**

*This presentation will introduce coccidiosis as a disease affecting a wide range of hosts, including a generic life cycle, the epidemiology of coccidiosis, how it can be diagnosed, the pathology coccidia can cause in their hosts, potential treatments and prophylaxis and ways to minimise its effects. Coccidiosis in macropods will be the main focus but examples in other species will be presented to illustrate important characteristics of the disease.*

*Hundreds of different species of Eimeria cause disease in in a very wide range of mammals, birds and reptiles. These coccidia are well described by Brice and Thomas (2018) so there is no need to repeat that here. Most infections are subclinical but clinical signs can include diarrhoea, fever, inappetence, weight loss, emaciation, and death in extreme cases. The virulence of coccidia is enhanced by stressors such as poor nutrition, poor sanitation, overcrowding, weaning, transportation, sudden changes of feed, or severe weather. Coccidiosis is also associated with other infectious agents and immunosuppression.*

*Coccidiosis can cause significant economic losses in cattle, sheep, goats, pigs, poultry and also rabbits, in which the liver as well as the intestine can be affected. Although the infection rate in most species of farm animals is high, the rate of clinical disease is low (5 – 10%), although up to 80% of animals in high-risk groups can show clinical signs. Coccidiosis usually involves acute destruction of intestinal mucosa by protozoa in the genera Eimeria or Isospora. The development of prophylactic drugs against coccidiosis twenty or thirty years ago allowed chickens to be bred intensively. Before, this was not possible due to persistent outbreaks of coccidiosis. Such drug development has not happened in other animal industries nor in relation to Australian marsupials. (Constable on-line; Carmichael 1988).*

## The Life Cycle of Coccidia, Genus Eimeria

Coccidia are strongly host specific and while there is some sharing of parasites between kangaroos and wallabies, many are species specific: they can cause disease in one species but not in others. *Eimeria* and *Isospora* typically complete their life cycles in one host. Some *Isospora* species can infect intermediate hosts. Many species of coccidia affect eastern grey kangaroos, some only causing a mild flu-like illness, but others can cause serious and even fatal disease. Coccidia also affect possums and platypus, but the species involved don't produce clinical disease.

Infected hosts shed unsporulated (therefore non-infective) *Eimeria* and *Isospora* oocysts in their faeces. Under favourable conditions of oxygen, humidity and temperature, oocysts sporulate to

become infective in several days. When the sporulated oocyst is ingested by a susceptible animal, the sporozoites escape from the oocyst, invade the intestinal mucosa and develop intracellularly into multinucleate schizonts (also called meronts). Each nucleus develops into an infective body called a merozoite; merozoites enter new cells and repeat the process. After a variable number of these asexual generations, merozoites develop into either macrogametocytes (females) or microgametocytes (males). These produce a single macrogamete or a number of microgametes in a host cell. After being fertilized by a microgamete, the macrogamete develops into an oocyst. The oocysts have resistant walls and are discharged unsporulated in the faeces, completing the cycle. Oocysts do not survive well below ~30°C or above 40°C; oocysts can survive more than a year at 30 – 40°C. Under suitable conditions, further division occurs in the soil and the oocyst becomes infective. The coccidia are now ready to be ingested and repeat the cycle.

While no work has been done in relation to macropod coccidia, the coccidia species which affect chickens can last up to 20 months in the external environment. Having evolved in a hot, dry country, macropod coccidia species are suspected to last much longer and wet, shady conditions help them survive. They might last years. Other organisms which evolved in Australia are very tough, so coccidia probably are too and have evolved to cope with a wide range of conditions. Once the faeces break down, sporulated oocytes can be dust-borne and can build up in small enclosed areas where animals are housed.

The traditional disinfectants, sulphuric acid and sodium hypochlorite affect coccidia so little they are used to clean them for presentation for research. Ammonia appears to be the most effective disinfectant.

**Predisposing Factors**

Coccidiosis is universal, most commonly in young animals housed or confined in small areas contaminated with oocysts. Most animals acquire Eimeria or Isospora infections between 1 month and 1 year of age. Older animals are usually resistant to clinical disease but can have sporadic inapparent infections. Although generally young animals are more susceptible than adults, older animals never previously exposed to coccidia can also be badly affected. In other words, no previous exposure can lead to serious disease in adults. Clinically healthy, mature animals can be sources of infection to young, susceptible animals. Normally, the parasite enters its host, reproduces, and drops out. Disease only occurs if something throws this out of sequence.

Some strains of coccidia from different geographical locations might be more virulent than others and cold, wet areas appear to be worse for susceptibility to coccidiosis than hot dry areas.

Stress factors are a very significant in mammals and birds, including in marsupials. Although many people believe eastern grey kangaroos stress very easily, all macropods can stress easily which trigger increased susceptibility. Only small changes can cause oocyst production to flare into clinical disease.

- poor nutrition, unsuitable foods and changes in diet increase the susceptibility to coccidiosis - always change diets gradually
- movement and transportation
- handling or disturbance by strangers; even mild harassment by other animals or children
- weather extremes: wind, rain, heat, cold
- crowding or overcrowding. Clinical disease is suspected to often follow confinement of animals and as this has been proven in the case of poultry, there is no reason to suspect macropods are different. This can be as simple as enclosing an area with a fence, as in a Game Park, which, while they always try to provide ideal conditions, do not always provide ideal environments.
- noise or movement.
- faecal contamination - the more oocytes ingested, the more likely disease will occur. Move animals off contaminated ground. Sanitation is very important, especially where many animals are housed together (e.g., wildlife rehabilitation centres, catteries and kennels) and prophylactic treatment might be required. Faeces should be removed frequently to prevent faecal contamination of feed and water. Disinfect runs, cages and utensils daily.

**Pathogenesis**

Macropods are host to many easily identified coccidia species, but little information is available on which coccidia species cause actual disease, or at what stage in their life cycle this occurs. Other species of coccidia infest the liver, particularly of rabbits, but the importance of these parasites in macropods is not known.

Some species of coccidia live on the surface of the inner lining of the intestine and cause no problem. They are sloughed off naturally with normal body wear and are of biological interest only. Only parasites which burrow deep into the cells of the intestinal wall and rupture the nucleus of these cells cause significant disease. This occurs when massive production of schizonts ruptures the gut wall, releasing enormous numbers of schizonts, which invade more cells within the gut wall, continuing the process of rapid asexual reproduction. Cell ruptures kill massive numbers of cells. Clinical signs are due to destruction of the intestinal lining (epithelium and often the underlying mucosa). This can cause bleeding into the intestinal lumen, catarrhal inflammation, and diarrhoea. Other signs can include discharge of blood or tissue, tenesmus (straining to defecate) and dehydration.

A single organism can multiply massively, destroying so many cells that the host can die within three or four days after ingestion. When coccidia invade the intestinal wall and cause the cells to slough, huge ulcer can form, causing acute pain and the external surface of the intestine to become red and swollen. Coccidia do not produce toxins – affected animals die of dehydration, due to fluid loss and of shock, caused by the extreme pain. The presence of coccidia oocysts in faeces does not mean the animal has coccidiosis and, conversely, animals can die with few

coccidia in their faeces because the immature stages can destroy so much tissue the animal dies before the mature forms of coccidia even develop.

However, very numerous oocysts in faeces is generally associated with disease. Oocyst counts can be 80 to 90 thousand per gram of faeces: 400 - 450,000 per teaspoon of faeces. If you have macropods, you will have coccidia oocysts in the soil on your property.

**Diagnosis:**
Oocysts can be identified in faeces by salt or sugar flotation. Finding numerous oocysts of pathogenic species is diagnostic (>100,000 oocysts/g of faeces in severe outbreaks), but diarrhoea can precede the heavy output of oocysts by 1–2 days and can continue after the oocyst discharge returns to low levels, so oocysts might not be identified in a single faecal sample; multiple faecal examinations from one animal or single faecal examinations of all animals housed together might be required. The number of oocysts in faeces is influenced by the stage of infection, age and immune status of the host, prior exposure, consistency of the faecal sample, and the method of examination. Thus, the results must be related to the clinical signs. The finding of numerous oocysts of a nonpathogenic species concurrent with diarrhoea does not constitute a diagnosis of clinical coccidiosis.

**Clinical Signs:**
- if the parasite is benign there might be no signs of disease
- failure to thrive, decreased growth rate, or poor weight gain
- persisting diarrhoea, which can be mild or prolific
- blood in the faeces (not always present) can be caused by other diseases, e.g., salmonellosis
- might be oocysts in the faeces, but not always
- might be fragments of muscle or intestinal lining in the faeces
- great pain and shock

Although recovered animals are suspected to carry the pathogen, this can be advantageous if it stimulates immunity in the next generation. Coccidiosis can, however, develop in recovered animals.

**Treatment:**
The life cycles of *Eimeria* and *Isospora* are self-limiting and end spontaneously within a few weeks unless reinfection occurs. Prompt medication can slow or inhibit reinfection, shortening the length of illness, reducing oocyst discharge, haemorrhage and diarrhoea, and lessening the likelihood of secondary infections and death. Isolate and treat sick animals individually to avoid exposing other animals. However, the efficacy of treating clinical coccidiosis has not been demonstrated for any drug, although it is widely accepted that treatment is effective against reinfection and should facilitate recovery.

**Anti-Coccidial Drugs**

Most currently available anti-coccidial drugs, such as the sulfonamides, are prophylactic - they help prevent the disease, but once an animal has developed coccidiosis, the only drug that might cure it is toltrazuril plus an effective antibiotic (Vogelnest and Portas 2009). If an animal recovers after treatment for coccidiosis, it might not have had coccidiosis. The available prophylactic drugs inhibit asexual division by repressing the number of coccidia produced. To be effective, prophylactic drugs must be used before and during periods of change or stress. The best protection is to keep susceptible animals on prophylactic drugs continuously.

**Immunity**

Coccidia species differ in their ability to stimulate an immune response. While they might reduce the number of coccidia, circulating antibodies play only a minor role in protecting an animal. The main reaction is cell-mediated in the gut wall. Coccidia are host-specific with no cross-immunity between species of coccidia. That is, immunity to one type of coccidian does not provide resistance to another species of coccidian. The best option appears to be to expose young animals to very low levels of virulent oocysts to help develop a significant level of immune-protection.

The Australian poultry industry breeds 350,000,000 birds a year and is worth $2.7 billion. The American industry even more. Despite enormous financial investment, a cure is still not available.

**Prevention:**

Prevention is based on limiting the sporulated oocysts ingested by young animals so that a mild infection is established to induce immunity but not clinical signs. Young, susceptible animals should be kept in clean, dry, sanitary conditions. Minimise stresses (e.g., weaning, sudden changes in feed, transportation). Preventive coccidiostats are recommended when animals are under increased risk of coccidiosis.

**Summary**

Eastern grey kangaroos are more prone to coccidiosis than other kangaroos. Field outbreaks can be triggered by stressors such as overcrowding, flooding, inclement weather, malnutrition, dietary changes, feed contaminated with faeces (on ground). Observe animals vigilantly for signs of stress. Any changes, no matter how minor, should be gradual and always observe the strictest hygiene - feed should never be placed on the ground but in containers, to prevent faecal contamination. Outbreaks can be precipitated by any of the stressors such as strangers, changes to feeding or routine, transporting or relocating, confinement, lack of cleanliness or even by the stress of separation.

**References:**


Carmichael, I, 1998, **Coccidiosis**, A talk at the Marsupial Society of Australia Inc. General Meeting, Thursday 21st May 1998.

Constable, PD, **Overview of coccidiosis**, MSD Veterinary Manual, Merck & Co., Inc., Kenilworth, NJ, USA, https://www.msdvetmanual.com/digestive-system/coccidiosis/overview-of-coccidiosis

Cox, F.E.G., 1998. **Control of coccidiosis: lessons from other sporozoa**, *International Journal of Parasitology*, **28**(1): 165-179.

Brice B, Thomas G., 2018, **Cocccidian Tales**, *In*: Proceedings of the Australian Wildlife Rehabilitation Conference 2018, Sydney, Australia. Pp. ??

Vogelnest L, Portas T, 2008, **Macropods**. *In*: Medicine of Australian mammals, eds L Vogelnest, R Woods, CSIRO Publishing: Melbourne, pp 133-225.