# COCCIDIOSIS IN POULTRY

**Lisa D Schmidt**, of SVS Laboratories, Hamilton, discusses one of the world's most economically damaging poultry diseases.

## WHY IS COCCIDIOSIS IMPORTANT?

For decades, coccidiosis has been reported as one of the most economically important diseases of poultry worldwide due to decreased production, increased mortality and the cost of prophylaxis and treatment. Most disease is caused by organisms within the genus *Eimeria*, which are obligate intracellular protozoa. In chickens there are nine described *Eimeria* species, and in turkeys there are seven species; however, not all are pathogenic. In addition to being species specific, most coccidia infect specific areas of the intestinal tract (see Table 1) resulting in enteritis and typhlitis. An exception is renal coccidiosis reported in geese and ducks.

## HOW DOES IT SPREAD?

*Eimeria* spp have a direct, but complicated, life-cycle. In brief, the organisms are spread by the faecal-oral route when birds ingest sporulated oocysts from infected feed, water, litter or soil. Released sporozoites from the

TABLE 1:

**Important pathogenic Apicomplexa (coccidia)**

| Species | Coccidia | Region of intestine affected |
|---|---|---|
| **Chickens** | *E. acervuline* | Duodenum |
| | *E. necatrix*[a]*/maxima* | Jejunum |
| | *E. brunette* | Ileum, caeca, rectum and cloaca |
| | *E. tenella*[a] | Caeca |
| **Turkeys** | *E. meleagridis* | Caeca |
| | *E. adenoides*[b] | Caeca, ileum |
| | *E. meleagrimitis* | Jejunum, duodenum |
| | *E. gallapovonis* | Ileum, colon |
| **Turkeys/quail** | *E. dispersa* | Jejunum |
| **Geese** | *E. anseris/nocens*[c] | Duodenum, jejunum |
| **Geese, ducks** | *E. truncata* | Kidney |
| **Ducks** | Tyzzeria perniciosa | Distal jejunum, ileum |

[a,b] *Most pathogenic species in chickens and turkeys, respectively*
[c] Eimeria nocens *is often a concurrent infection with* Eimeria anseris



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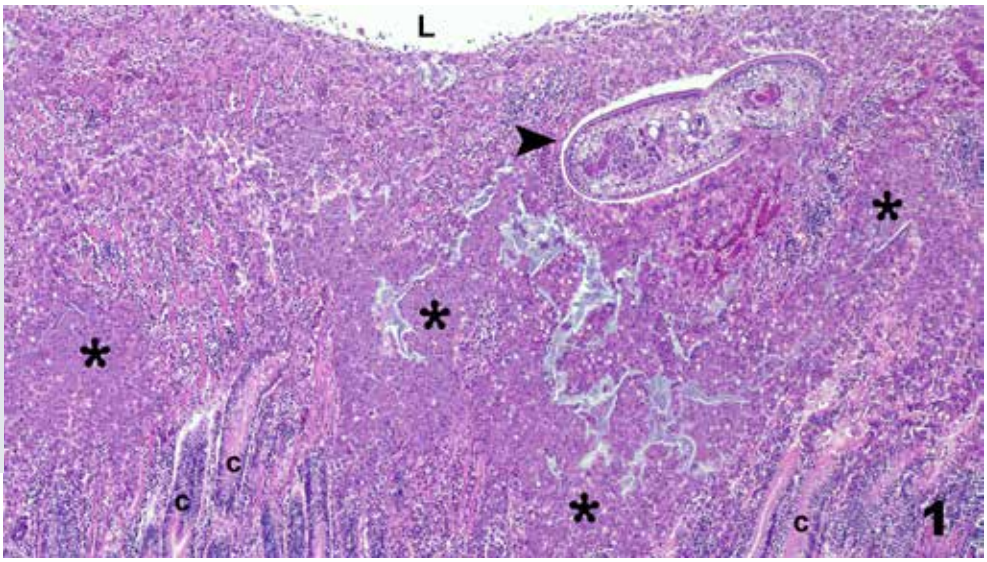


**FIGURE 1**: Small intestinal villi are effaced by necrosis and myriad coccidian organisms (*) in various stages of development. Concurrent nematodiasis (arrowhead) and secondary bacterial infection are present in this bird. C: Small intestinal crypts; L: Lumen.

oocysts initiate asexual replication, followed by sexual reproduction, which produces thousands of new oocysts. The unsporulated oocysts are shed in the faeces and sporulate within 24 to 48 hours. Wet litter and warm temperatures (21°C to 32°C) promote sporulation and precipitate disease outbreaks. Additional risk factors for infection include: host genetics, nutritional factors, concurrent host disease and immunosuppression, coccidian species and previous exposure to the organism(s). Veterinarians and animal care staff can also spread disease because coccidia are readily transported on boots, shoes, clothing, crates, vehicle wheels and other animals and insects.

## WHAT ARE THE CLINICAL SIGNS?

While protective immunity can develop in response to moderate and continuing infection, birds of any age can be affected, and the severity of infection is related to the number of oocysts ingested. Subclinical infection is common and is associated with decreased performance such as decreased weight gain, increased feed conversion ratio and decreased egg production, depigmentation due to decreased absorption of carotenoids, and increased risk of secondary infections, especially with *Clostridia* spp. In clinical infections, the birds have diarrhoea and dehydration, with high morbidity and mortality. Diarrhoea may be watery, mucoid and/or haemorrhagic. Infection progresses rapidly, and by the time the flock is examined the birds may have ruffled feathers, anaemia, weakness, listlessness and/or somnolence.

## HOW IS IT DIAGNOSED?

A combination of clinical signs, appearance and location of post-mortem lesions, histology, and microscopic features of the oocysts on direct faecal or mucosa scraping examinations are used to diagnose coccidiosis. Faecal flotation can also be carried out; however, faecal oocyst count may not correlate with the severity of infection, and an absence of oocysts does not rule out severe infection. Polymerase chain reaction (PCR) assays are also used currently in research settings to speciate coccidia.

## WHAT SHOULD I LOOK FOR DURING A NECROPSY?

Necropsies, when possible, should be performed on freshly (<1hr) dead birds to minimise post-mortem changes. Post-mortem haemostatic congestion, haemoglobin imbibition and freeze-thaw artefacts distort the colour of the intestinal loops, making it difficult to localise the lesions within the gastrointestinal tract. Lesions of coccidiosis will vary from red and white pinpoint spots to plaques seen on the serosal and

TABLE 2:

### Samples to submit to the diagnostic lab for the diagnosis of coccidiosis

| Sample | Test |
|---|---|
| **Fresh SI[a], caeca and colon** | **1.** Mucosal scraping (direct examination)<br>**2.** Bacterial culture<br>**3.** PCR |
| **Formalin fixed SI, caeca and colon** | Histopathology |
| **Excreta** | Faecal flotation, direct examination, bacterial culture |
| **Fixed and fresh major organs[b]** | Histopathology, bacterial or fungal culture |

[a] *Small intestine*
[b] *Major organs include: lung, heart, liver, spleen, kidney, proventriculus, ventriculus, bursa of Fabricius (in neonate/juvenile animals), air sacs (if pathology present), proximal tibia (growth plate, and bone marrow).*

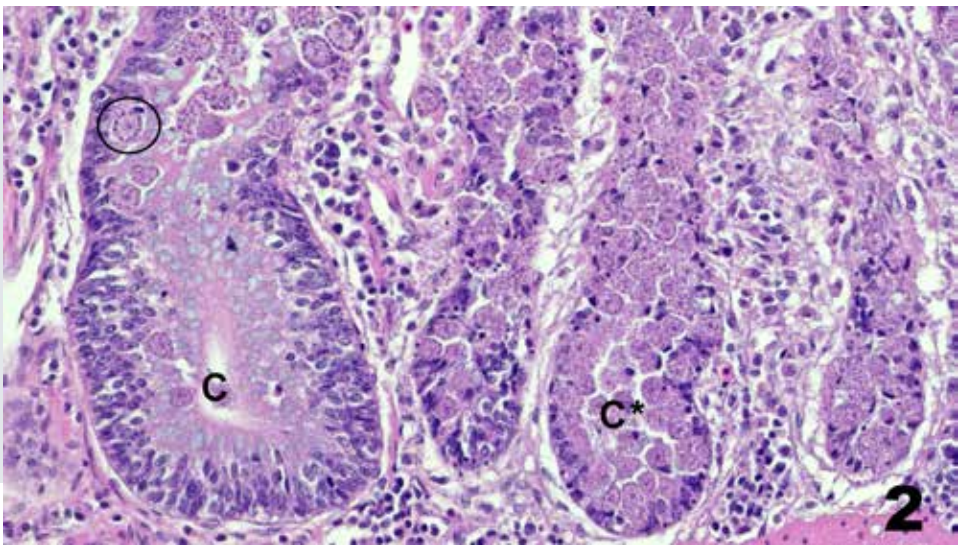


**FIGURE 2**: Intracytoplasmic macrogametes with characteristic peripheral eosinophilic granules (one organism circled) are present within enterocytes. One intestinal crypt (C) has very few intracellular macrogametes compared to adjacent crypts where most enterocytes have intracellular macrogametes (C*).

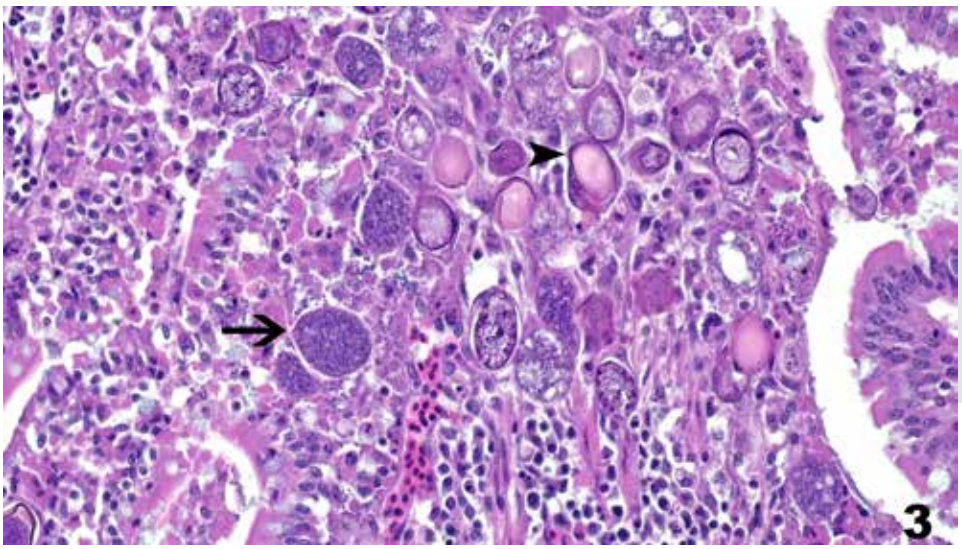


**FIGURE 3**: Microgametes (arrow) with numerous basophilic nuclei, macrogametes, and oocysts (arrowhead) free within the small intestinal lamina propria.

mucosal surfaces. The intestinal loops may be dilated up to 2.5 times normal, with or without mural thickening. More severe coccidiosis may present with fibrinous or fibrinonecrotic membranes covering the mucosal surface, or have caseous cores that are composed of clotted blood, tissue debris and oocysts that fill the lumina. Alternatively, lumina may be filled with fluid, mucus and/or blood. Tissues should be collected throughout the intestinal tract, focusing on pathologic lesions. Additional tissues should be collected (see Table 2) to rule out concurrent disease(s), including necrotic enteritis, ulcerative enteritis, salmonellosis and histomoniasis. It is also important to rule out other causes of necrotising enteritis and typhlitis, such as salmonellosis and clostridial infections.

Histologically, lesions containing coccidian organisms are pathognomonic. Tissue damage by coccidia includes coagulative necrosis, sloughing of the mucosa and loss of villi. Intracellular microgametes and macrogametes within crypt enterocytes, or extracellular organisms and oocysts, may also be seen (see Figures 1-3). Overall, histologic changes cause reductions in the absorptive surfaces, with resultant decreased nutrient absorption, dehydration, blood loss and increased susceptibility to secondary infections. VS

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