Coccidiosis in the North Island Brown Kiwi: MVSc Thesis Proposal

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CAPTIVE REARING OF KIWI

Kiwi numbers are in decline. In the wild, chick survival rate has been shown to be as low as 2% due to predation by introduced mammals. Mustelids, cats and rats are the main perpetrators, feeding on eggs and defenceless chicks.

In the past 10 years, there has been intensive captive rearing of kiwi chicks from wild born eggs that has increased chick survivability to around 40%. Under the "Operation Nest Egg" programme (ONE), eggs are removed from their wild nest and are artificially incubated to hatching. Chicks are raised in predator-free enclosures until they reach a suitable release weight (usually around 1000–1200grams), at which they are considered able to fend off cats and mustelid predators. The grown chicks are then released back to the site from where they were removed as eggs.

The marked elevation in density and the high turnover of chicks in captive situations increases the risk of exposure to disease. ONE facilities are likely to increase in importance as more kiwi are raised in intensive conditions.

COCCIDIA AND KIWI

Coccidia appear to be a natural parasite of the North Island brown kiwi (*Apteryx mantelli)*, having been described in both free living and captive birds. To date, this disease has only been seen in the North Island species of brown kiwi. Both intestinal and renal coccidial forms have been identified.

Coccidiosis is one of the main limiting factors of successful rearing of kiwi in captivity. It is primarily a disease of juvenile kiwi, and is most commonly seen in captive rearing institutions. Compared to their wild counterparts, the densities of chicks in captive institutions are significantly increased, increasing the environmental contamination with coccidial oocysts. The continual turnover of juvenile kiwi through incubators and brooders greatly increases the risk of exposure to infective oocysts.

One third of all juvenile North Island brown kiwi entered on the New Zealand National Wildlife Pathology Database ("Huia") over the past 13 years showed histopathological signs of coccidial infection, some incidental, others lethal. To date, nineteen cases of coccidial infection have been reported. Around 80% of these were juvenile birds, and the majority of these were from captive

institutions. A survey looking at free-living kiwi found that 81% of coccidial infected kiwi were less than two years old.

Epizootiology of Avian Coccidia

Little is known about the type of intestinal or renal coccidia infecting kiwi. Preliminary examination indicates this to be an *Eimeria* species, and it is unknown whether the coccidian infecting the gastrointestinal tract is the same as that affecting the renal parenchyma. No work has been done to determine the coccidial life cycle of either form.

Intestinal Coccidia

All previously described forms of avian intestinal coccidia have a direct life cycle, and the following descriptions are extrapolated from other species. Mature oocysts are ingested by the potential host where the digestion process results in the release of sporozoites. These invade the host's intestinal epithelial cells and undergo schizogony. The parasite reproduces asexually forming several generations of schizonts then ultimately merozoites. Each of these stages enters new host cells. Sexual reproduction results in male and female gametocytes. Fertilised macrogametocytes form a thick wall to develop into oocysts that are shed in the faeces. Sporulation occurs in the external environment.
Clinical disease occurs when the rupture and destruction of intestinal epithelial cells occurs at a rate greater than can be replaced by the regenerating intestinal cells.

Renal Coccidia

Renal coccidiosis in kiwi was first reported in 1978. Kidney coccidial infections are common in waterfowl where, in general, adult birds are subclinically infected and infections in young birds may be extremely pathogenic. In ducks, unsporulated oocysts are passed with the urine and, once matured, are ingested by a suitable host. It is unknown how the coccidia move from the gastrointestinal to the renal tract.

Clinical signs

Coccidial infections in kiwi, like other species, appear to occur both with and without clinical disease. Clinical signs specific to either intestinal or renal forms are yet to be established. In general, it appears low to moderate infestations may show no outward signs of disease. Heavier coccidial infections causing subsequent mortality may be preceded by a period (1-2 days up to 3 weeks) of decreased growth rate, anorexia and weight loss. Other affected kiwi may succumb to the disease without visible clinical signs, or clinical signs of such a short duration that they aren't detected.

Diagnosis

Diagnosis of gastrointestinal coccidial infections in kiwi has been achieved by flotation and detection of large numbers of faecal oocysts in association with clinical signs of disease. There are potential complications associated with this method of diagnosis. In other species, infected birds may have the disease before large numbers of oocysts are present in the faeces. For example, in chickens, the most damaging stage of *Eimeria tenella* are the second generation schizonts that occur before oocysts occurs before oocysts development, so death may occur prior to excretion of detectable oocysts.

Renal coccidial infections are more difficult to detect. In studies looking at renal coccidiosis in waterfowl, oocysts are passed with the urine. However, routine collection of the urinary fraction of the droppings is difficult. Diagnosis of renal coccidial infections generally relies upon the detection of urinary oocysts included with faecal floatations. At necropsy, confirmation of both forms of coccidiosis is confirmed by histopathology.

Pathology

Intestinal

In early or moderate alimentary tract infections, there may be hyperaemia of the intestinal mucosa. Excessive intestinal luminal fluid may be present. In more severe cases, the mucosa may become congested and the intestinal wall hyperplastic. An abundance of both asexual and sexual stages of coccidia may be seen within the intestinal epithelial cells with an associated inflammatory change.

Renal

In severe infections of young birds, the kidneys may be markedly swollen, pale and covered with military white streaks. Similar gross lesions are reported in severely infected young waterfowl. Histological changes include disruption of renal tubules and extensive accumulations of mixed populations of inflammatory cells. The epithelial cells contain many sexual stages of coccidia (gametocytes) and oocysts. This is consistent with findings from other avian species, where mostly only sexual stages have been reported
In waterfowl, older birds have focal lesions without clinical significance.

Prevention, Control and Treatment

Prevention relies upon hygiene and ensuring dirt floors and leaf litter are replaced between chicks. Oocysts are extremely resistant in the environment, and aren't susceptible to most disinfectants.

The aim for control of coccidiosis should be to reduce the initial exposure of birds to coccidial oocysts, whilst allowing some exposure to the coccidia. Since coccidia are a natural parasite of wild kiwi, this is an important process in the production of immunity.
To date, affected birds have been treated with toltrazuril ("Baycox"). The dose rate and efficacy of this product in kiwi is yet to be established.

Objectives of this Research Project

1. Determine the incidence and prevalence of coccidiosis in wild and captive populations of juvenile kiwi throughout New Zealand
2. Determine the age distribution of coccidiosis in juvenile North Island Brown Kiwi
3. Determine the efficacy of the current treatment for coccidia ("Baycox")
4. Determine methods that aid in the ante-mortem identification of renal coccidiosis
5. Determine variations in blood parameters of coccidial infected versus uninfected juvenile kiwi
6. Describe pathological changes associated with renal and gastrointestinal coccidial infection

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