Protozoal Management in Turkey Production

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The two turkey protozoa that cause significant animal welfare and economic distress include various *Eimeria* species of coccidia and *Histomonas meleagridis* (McDougald, 1998). For coccidia, oral ingestion of the organism allows for colonization and replication while fecal shedding passes the organism to another host. With *Histomonas,* once one turkey is infected it can pass *Histomonas* to its flock mates by cloacal contact. Outbreaks of coccidiosis followed by *Histomonosis* (blackhead disease) is commonly seen in the field but the relationship between the protozoa is not understood. Turkey fecal moisture, intestinal health and behavior changes due to coccidiosis could be increasing horizontal transmission of *Histomonas*.

Clinical signs of coccidiosis, like macroscopic lesions in the intestines, are not necessarily evident but altered weight gain and feed conversion are (Madden and Ruff, 1979; Milbradt et al., 2014). Birds can become more vocal. Depending on the infective dose, strain of coccidia and immune response of the turkey, intestinal irritation leading to diarrhea can occur (Chapman, 2008; McDougald, 2013). Birds are also more susceptible to other infectious agents. This is potentially due to the damage the coccidia can cause on the mucosal lining of the intestines but studies on this interaction are limited (Ruff et al., 1981; Milbradt et al., 2014). Coccidia sporozoites penetrate the turkey intestinal mucosa and utilize the intestinal tract for replication and survival. Of the seven coccidia *Eimeria* species known for infecting turkeys, four are considered pathogenic (*E. adenoeides, E. gallopavonis*, *E. meleagrimitis* and *E. dispersa)* (Chapman, 2008; McDougald, 2013; Milbradt et al., 2014). The pathogenic strains are utilized for vaccine development to aid in a nonmedicated option for coccidia immunity.

Coccidia acquired immunity and epidemic outbreaks are based on coprophagy, or fecal consumption. If a flock is vaccinated, it is done on day of hatch where either a spray or gel application is given to poults for oral administration of (live) oocysts. This allows for the initial interaction between the poult's gut and the oocysts so that early immunity can be developed through production. Once the oocysts reach a certain level of maturity while in the turkey gut, they are expelled from the intestinal lining and shed in the feces. Coccidia sporozoites have a protective coating known as the oocyst wall that can withstand drastic changes in the external environment so this organism can survive for extended periods outside of the host (McDouglad, 2013). Coprophagy and the ground pecking behavior commonly seen with turkeys allows for the ingestion of oocysts by the flock so that acquired immunity occurs (Chapman, 2008). Currently, the turkey industry has limited coccidia vaccine options because (1) there is only one commercial vaccine available (2) producers can use an autogenous vaccine but this is not tested or well controlled (3) turkeys do not feather peck as often, altering the dosage of a gel- administered vaccine. These three variations could cause a rolling coccidia infection instead of specific cycles.

The addition of anticoccidials (ionophores) in feed, as an alternative to vaccination, have been shown successful in controlling coccidia outbreaks (Milbradt et al., 2014). However, turkeys are highly susceptible to ionophore toxicity and a common indication of toxicity is decreased feed intake with increased litter consumption (Chapman, 2008). The increased litter consumption can increase oocyst intake (leading to a rolling infection) and increases the potential of consuming other parasites, like *Heterakis gallinarum*.

Heterakis gallinarum, the common reservoir of *Histomonas*, is found in poultry litter. The *Heterakis* egg acts as a protection mechanism for *Histomonas* and the eggs can survive in the environment for multiple years (Cupo and Beckstead, 2019). The consumption of litter contaminated with *Histomonas* infected *Heterakis* eggs is the start of an outbreak in a turkey facility but not how *Histomonas* becomes epidemic (Hu and McDougald, 2003). Without the natural encapsulation of the egg, it is hypothesized that very few, if any, histomonads can survive the chemical and physical challenges presented while in the digestive tract (Hu, et al., 2006). In an experimental model, the oral inoculation of only *Histomonas* failed to cause blackhead disease in poults while a cloacal inoculation from the same culture led to 60-80% of poults showing signs of infection (Fudge, et al., 2019). This study suggests that oral consumption of only *Histomonas* will not propagate disease.

Cloacal drinking of *Histomonas* will cause blackhead disease (Hu, et al., 2004; Hu and McDougald, 2003). Physical stimulation of the cloacal vent, heavy panting or vocalization can lead to muscle contractions of the turkey anus to draw material from the environment into the ceca. In experimental models, dropping liquid containing *Histomonas* on the turkey vent lead to the uptake of the culture and blackhead disease. Also, uninfected poults will contract *Histomonas* if they are in physical contact with an infected bird. This is because a behavior commonly seen in (sick) turkeys is huddling. This behavior has been noted with coccidiosis and blackhead disease (Hu, et al., 2006; Chapman, 2008). The huddling behavior allows for one bird's defecation to encounter more of the flock's population.

Cloacal drinking can more easily occur if the material taken up has a fluid consistency (Ferket and Veldkamp, 1999). Wet droppings are commonly seen in turkey rearing facilities with a healthy or sick flock. With cocci specifically, outbreaks in turkeys can lead to an increase in fecal moisture. The wet fecal droppings in combination with cloacal drinking can allow for cloacal-cloacal contact to transfer *Histomonas* (Hu, et al., 2006). However, it is important to note that litter quality is not indicative of a *Histomonas* outbreak. In the field, dry or wet litter can be seen while a flock is suffering from blackhead disease.

Behavioral changes in turkeys during coccidiosis including litter/ fecal consumption, wet feces, constant cheeping, and huddling can increase the likelihood that a flock will later break with *Histomonas*. Litter consumption increases the potential of turkeys consuming a carrier of *Heterakis gallinarum* which will start a blackhead disease outbreak. Gut irritation and diarrhea from the infected bird will allow for more fluid movement of *Histomonas*-contaminated droppings. Huddling will make the transfer of droppings between birds more likely. Turkey vocalization stimulates the movement of the cloacal vent, leading to reverse peristalsis bringing *Histomonas* contaminated droppings into the ceca. Establishment in the ceca will cause blackhead disease in a turkey, leading to mortality.

Turkeys fail to have an effective immune response to *Histomonas* (Powell, et al., 2009). This protozoan colonizes in the ceca then eventually migrates to the liver through the hepatic-portal vein. During the first few days of infection, *Histomonas* invades the cecal mucosa but there are no gross pathological lesions. By day 4, the ceca start to become inflamed. Around day 7, the ceca will have a caseous core that develops in the center and the liver will start to become necrotic. At 10 to 14 days after infection, turkeys will suffer from liver failure and succumb to the disease (McDougald, 2005; McDougald, 2013). Currently, the time period that turkeys are contagious with *Histomonas* is not well defined.

A correlation between coccidia then *Histomonas* outbreaks have been shown in the field while experimental inoculations of both protozoa vary. One interaction of cocci then *Histomonas* infections in turkeys lead to more severe lesions related to blackhead disease (Vignale- Pollock, et al., 2020). Other research models have failed to increase blackhead severity following coccidiosis in turkeys, but this could be due to the timing of the inoculations. Many of the coccidiosis outbreaks are not indicative by macroscopic lesions or mortality and so it can be difficult to diagnose severity of the outbreak. Initial mortality due to blackhead disease is low so diagnosis usually does not occur until after most of the population is infected. Targeting poults suffering from blackhead disease during the initial infection instead of after horizontal transmission occurs will decrease the spread of this parasite. Two weeks after poults are showing signs of coccidiosis, it is suggested to post any mortality to determine if blackhead disease has started in the flock.

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