***Technical report*: Histomoniasis (blackhead) in commercial and backyard poultry**

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Abstract:

Histomoniasis (blackhead) is a protozoal disease caused by *Histomonas meleagridis* that affects mainly the liver and ceca of turkeys and other gallinaceous birds, including chickens, peafowl, and quail. Transmission of blackhead occurs by direct contact with infected birds or infected feces, as well as indirect infection via ingestion of infected cecal worms (*Heterakis gallinarum*) or earthworms that contain infected cecal worms. Clinical signs develop in 6-12 days, and occur most commonly at 11 days post-infection. They include listlessness, emaciation, unkempt feathers, and yellow, sulfur-colored droppings. The name "blackhead" is somewhat of a misnomer, as typical presentations of diseased turkeys do not include cyanosis of the head. Gross lesions are characterized by pathognomonic, necrotic target lesions in the liver and caseous cecal cores. Histopathologic lesions are characterized by numerous round to ovoid, faintly eosinophilic to golden-brown PAS-positive bodies, ranging from 10-20 µm in diameter, surrounded by a halo, within macrophages or inflammatory cellular debris in H&E stained tissue sections. Since there are no chemotherapeutic products that are approved and available for treatment of histomoniasis in the U.S., control strategies are focused on prevention. Routine deworming, cleaning of premises, and practicing good biosecurity, such as avoiding multiple species of poultry in a flock (especially chickens and turkeys), are key measures for prevention and control.

Introduction:

Histomoniasis is a protozoal disease that affects mainly the liver and ceca of turkeys and other gallinaceous birds, including chickens, peafowl, and quail (4). Also known as blackhead disease or enterohepatitis, histomoniasis continues to be a cause of sporadic but severe disease in commercial and backyard turkey flocks. Losses are more common but less severe in chickens and game bird flocks (9).

Histomoniasis in turkeys was first described in 1895. In 1920, the causative agent was described as a flagellated, ameboid protozoal organism, *Histomonas meleagridis*, by Dr. E.E. Tyzzer of Harvard University. In the same year, Graybill and Smith at Rockefeller Institute established that the cecal worm *Heterakis gallinarum*, was the carrier of the histomonads (6). Since then, annual mortality losses in turkeys were estimated to exceed $2 million, and although the severity of the disease is not as high in chickens, losses from morbidity and mortality are estimated to be higher in chickens due to the frequency of infection and the sheer number of birds involved (5).

Transmission:

Transmission of blackhead occurs by direct contact between infected and susceptible birds or by contact with infected feces. Outbreaks in turkey flocks can spread through in 1-2 weeks, with approximately 80-100% morbidity and mortality.

The role of the cecal worm *Heterakis gallinarum* as in intermediate host has been well-described in literature. Histomonads are found within intestinal epithelial cells of young cecal worms, and female worms are thought to become infected with the histomonads during copulation, incorporating the protozoa into the eggs prior to shell formation. Infected eggs pass in feces where they may be ingested directly by birds or by earthworms who may serve as a transport host. Ingestion of contaminated feces, cecal worms or cecal worm eggs, or earthworms by the bird results in transport of the parasite to the cecum where flagellated trophozoites directly infect or are released from the nematode egg, multiply in the cecal lumen, and penetrate the cecal wall. The trophozoites then loses the flagella and becomes amoeboid. Within 2-3 days, the histomonads enter into the bloodstream and are carried to the liver via the hepatic-portal system. In the liver and cecal tissues, the cells divide and grow, forming necrotic lesions that are observed upon gross examination. Due to the fragile trophozoite stage of the histomonads, which cannot survive for extended periods of time outside of any of its hosts, the organism would be unable to survive passage through the stomach if not within a nematode egg or an

earthworm. Therefore, fecal-oral transmission is not thought to be an important route of transmission unless the acidity of the proventriculus was neutralized.

Clinical signs:

Clinical signs in histomoniasis develop 6-12 days post-infection, and occur most commonly at 11 days post-infection, and include listlessness, emaciation, unkempt feathers, and yellow, sulfur-colored droppings in the later stages of the disease when liver function is severely damaged, and bile pigments are excreted out via the kidneys (7). The name “blackhead” is somewhat of a misnomer, as typical presentations of diseased turkeys do not include cyanosis of the head (4). The incubation period varies with the infective dose, and infections from worm eggs require longer incubation period versus direct transmission. Turkeys become infective to other turkeys within 2-3 days post-infection (5).

In addition, bacterial flora are thought to be important contributors for the development of clinical histomoniasis and characteristic lesions. Lesions of histomoniasis cannot be produced in germ-free turkeys or chickens unless bacteria, such as *Clostridium perfringens, Escherichia coli,* or other mixed cultures, are present (10).

Gross and histopathologic lesions:

Gross lesions are observed mainly in the liver and cecum. The characteristic liver lesions (Figure 1) are described as round, depressed, target-like areas of necrosis that are yellow to gray, green, or red. The size of the lesions vary greatly, but are typically 1-2 cm in diameter, and may coalesce to form larger areas of necrosis. The ceca are most commonly bilaterally enlarged, with thickening of the cecal walls. The ceca often contain caseous cores with ulceration of the cecal mucosa, which may lead to perforation and cause peritonitis. Small, pale pink to whitish cecal worms ranging from 0.5-1.5 cm in length may be observed in the cecum (Figure 2).

Histopathologic changes in the liver are characterized by multifocal hepatic necrosis with numerous intralesional trophozoites of *Histomonas meleagridis* with mild, predominantly lymphocytic

inflammatory infiltrates in the early stages of the disease. As the lesion progresses, macrophages and numerous multinucleated giant cells are the predominant cell types, with individualized or clusters of trophozoites in the parenchyma or within the cytoplasm of macrophages.

In the earlier stages of infection in the cecum, the lamina propria and submucosa are expanded by densely lymphohistiocytic infiltrates extending into the muscularis. Cecal cores are histologically represented as sloughed, ulcerated epithelium that may almost extend into the serosa, fibrin, erythrocytes, and leukocytes characterized by a predominantly macrophagic population. Intermixed with the cellular infiltrates are organisms consistent with trophozoites of *Histomonas meleagridis*. The trophozoites appear in H&E stained tissue sections as round to ovoid, faintly eosinophilic to golden-brown bodies, ranging from 10-20 µm in diameter, usually surrounded by a halo (lacunae). The trophozoites can be demonstrated more easily in periodic-acid-Schiff (PAS) stained slides (Figure3) (3).

Diagnosis:

Diagnosis can be made on the basis of clinical signs and characteristic lesions in the liver and/or cecum. Typical, well-developed target lesions in the liver +/- cecal lesions are pathognomonic for the disease. Although diagnosis is not difficult using clinical signs, gross lesions, and histopathologic findings, polymerase-chain reaction (PCR) tests are highly accurate in identification of *Histomonas meleagridis*.

Prevention and treatment:

Since there are no chemotherapeutic products that are approved and available for treatment of histomoniasis in the U.S., control measures are focused on prevention.

Rearing turkeys in close proximity to chickens tends to be a common source of cecal worm ova, which serves as an intermediate host and harbors the protozoal organisms. A study by Chute and Chute (1969) describes that young chickens were 16 times as effective as mature chickens in hosting cecal worms (1), and with their subsequent research testing eight species of gallinaceous birds (1973), the Chinese ringneck pheasant to be the best host for cecal worms, followed by chickens and guinea fowl. The

effectiveness of the turkey as a host for cecal worms in this study is almost negligible (2). Due to the hardy nature of the heterakid eggs, recurrence of histomoniasis is common in affected flocks, and range rotation is not a practical solution. Raising turkeys indoors tends to reduce outbreaks of blackhead, but can exacerbate the extent of outbreak by facilitating spread of the cecal worm ova by direct contact. Nitarsone (Histostat-50) is the only approved product in the U.S. for blackhead. Although it may be effective as a preventative, the use of nitarsone as a treatment is ineffective. Coccidiosis caused by *Eimeria tenella* in chickens has been identified in literature as a contributing factor, as the severity of the lesions as well as morbidity of the flock was increased with the presence of both parasites. Results from this study suggest that prevention strategies to control coccidiosis in breeder and layers are important in blackhead infections (8).

There is no doubt that there are cecal worms and other vermin, such as earthworms, that are involved in the pathogenesis, so the flock needs to be on an aggressive deworming program with benzamidazole-type anthelmintics in turkeys and other domestic poultry or a Hygromix feed program in chickens (*Personal communication*: Dr. Bret Rings, Cobb-Vantress, Inc.). Aggressive clean out of litter and fecal material that harbor organisms and routine disinfection of the enclosure and waterers to reduce bacterial load is also important. The administration of effective anthelmintics at least 1 week prior to expected times of outbreak is crucial to controlling outbreaks within susceptible flocks. In order to assess effective deworming strategies, routine fecal exams in backyard flocks with previous blackhead outbreaks are recommended.

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Figure 1: Multifocal to coalescing necrotic target lesions in the liver of a turkey with histomoniasis.

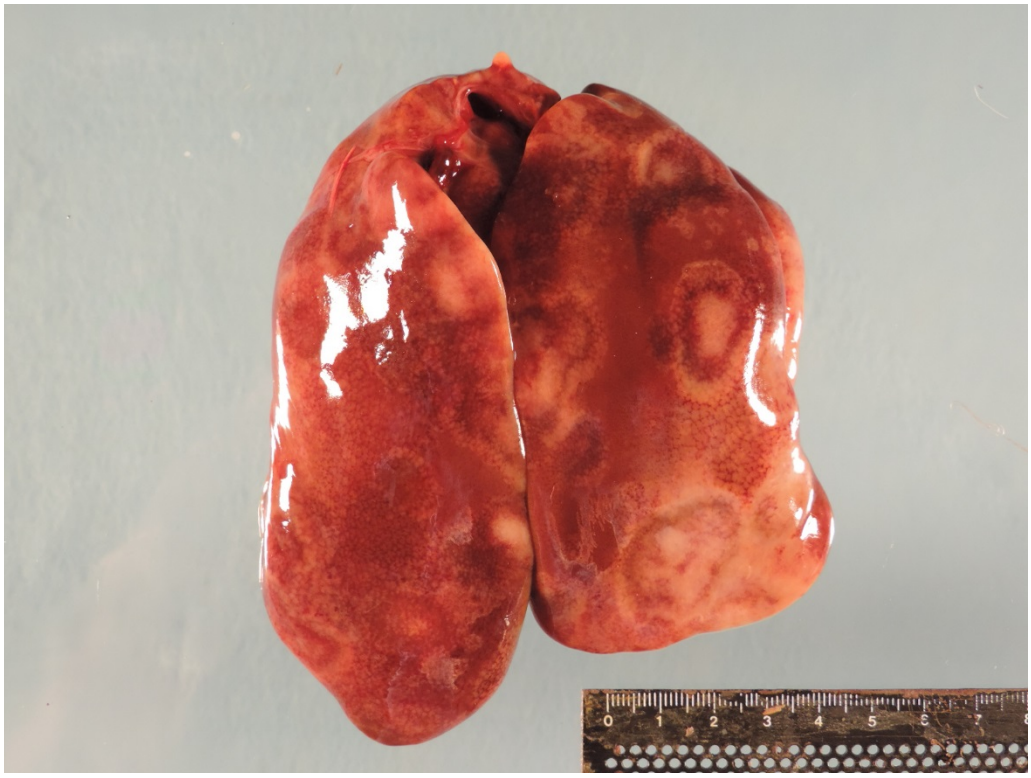
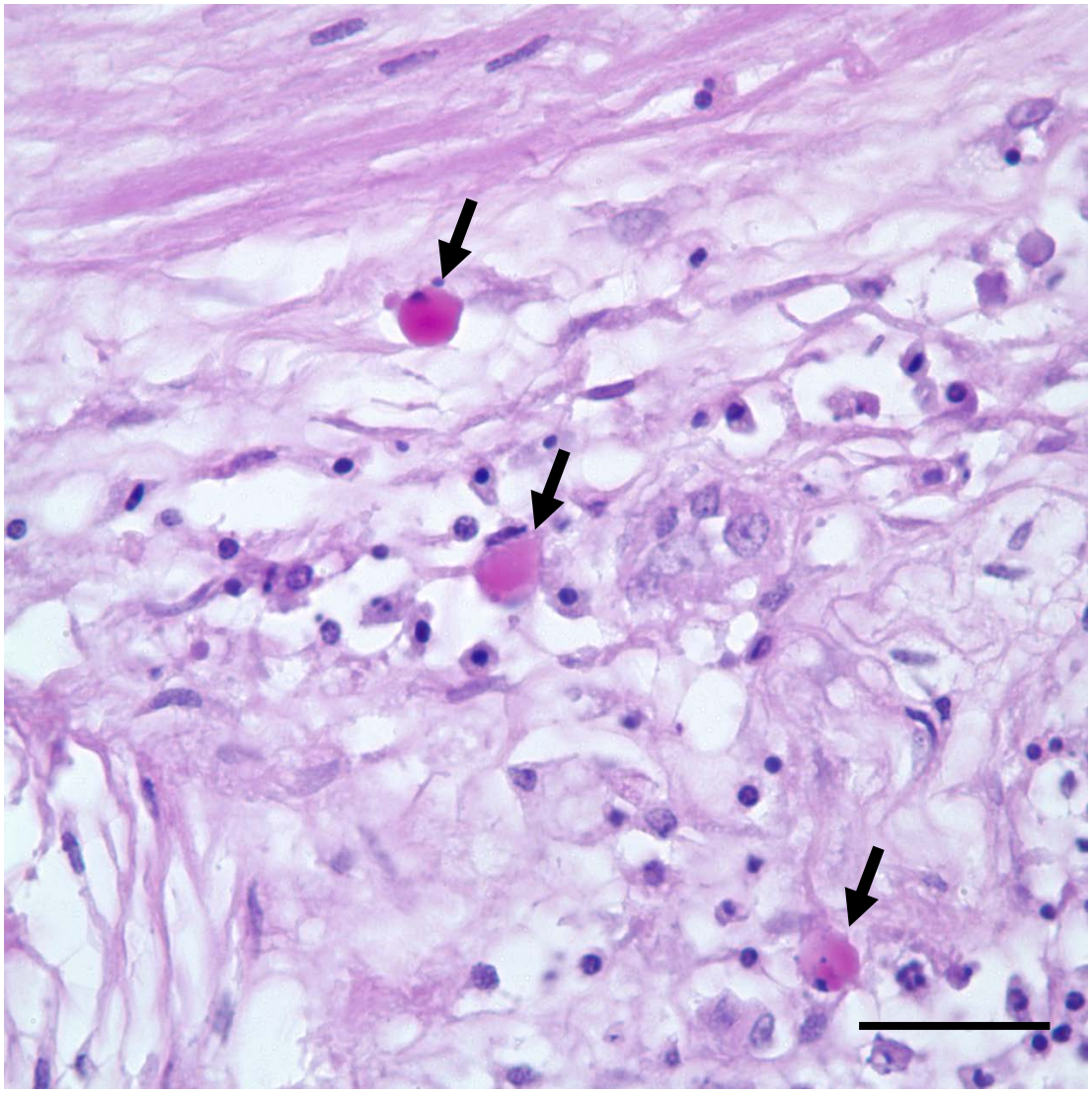


Figure 2: Multiple pale whitish cecal worms (arrows) within a necrotic cecal lumen

143 Figure 3: PAS-positive histomonads (arrows) in the cecum; bar = 50 µm, 100x magnification, Periodic

144 acid-Schiff (PAS) stain.



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