

Isolation of *Listeria monocytogenes* from an Eastern Wild Turkey

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tained in captivity where crowding and sanitation may contribute to the disease cycle (Montali, 1976, op. cit.). The organism also is found commonly in wild birds such as sparrows, pigeons, and starlings. According to unpublished diagnostic records from the NWHL, avian tuberculosis is a frequent finding in waterfowl species from some western waterfowl refuges.

Microscopic examination of tissue from the esophagus and intestines revealed that the crane shed organisms into the environment. Although not considered highly contagious, the organism, *M. avium*, is relatively stable in the environment, persisting in the soil for months or even years (Karlson, 1978, *In Mycobacterial Infections of Zoo Animals*, Montali (ed.), Smithsonian Institution Press, Washington, D.C., pp. 21–28). The source of the infection in this case is unknown. Increasing contamination of the environment

presumably would be related directly to an infected individual using the area and shedding *M. avium* in the feces and to organism resistance to environmental degradation. Field observations on the crane prior to its death indicated extensive use of a relatively small area near a grain bin that also attracted other species of birds. Avian tuberculosis and salmonellosis are frequently associated with crowding and/or a contaminated environment and should be recognized as potential dangers where birds congregate.

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Listeria monocytogenes, a bacterium which causes listeriosis, has a worldwide distribution, but only sporadic occurrence (Gray, 1958, *Avian Dis.* 2: 296–313; Gray, 1964, *Proc. N. Am. Wildl. Nat. Res. Conf.* 29: 202–213; Hofstad et al., 1984, *In Diseases of Poultry*, Iowa State University Press, Ames, Iowa, pp. 261–263). The disease has been isolated from at least 42 mammalian and 22 avian species. The most common avian hosts are chickens, canaries and geese, all of which appear to

be the most susceptible (Hofstad et al., 1984, op. cit.). Other species such as grouse, partridges, eagles, sparrows and starlings have been found to be infected (Gray, 1958, op. cit.; Hofstad et al., 1984, op. cit.). The isolation of *L. monocytogenes* from domestic fowl, with mortality up to 40% (Hofstad et al., 1984, op. cit.) has been reported practically worldwide. However, in wild birds isolations have been limited to Europe, with the single exception of an apparently normal snowy owl (*Nyctea scandiaca*) shot in Ontario, Canada (Gray, 1964, op. cit.). The purpose of this report is to document an in-

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fection by *L. monocytogenes* in an eastern wild turkey (*Meleagris gallopavo silvestris*) in Mississippi.

On 14 January 1984 a male eastern wild turkey was found in a field near Kewanee, Lauderdale County, Mississippi in a weakened, dehydrated, emaciated condition. The bird was brought to the Veterinary Diagnostic Laboratory at Jackson and died the next day. At necropsy, the bird weighed 3.14 kg. Gross lesions consisted of atrophy of the breast musculature and an extremely hard, 1 cm spherical mass in one cecal pouch. Numerous yellow fibrinous tags were found in the abdominal air sacs and on the visceral peritoneum. At necropsy, we concluded that the bird had died of diffuse peritonitis and airsacculitis.

Primary cultures were made by inoculating MacConkey agar plates and 5% equine blood agar plates with swabs taken from mesentery and liver and incubating the plates at 37 C under aerobic conditions. Small, flat, butyrous colonies producing a distinct zone of beta-hemolysis were observed on the blood agar plates from the mesenteric swabs after overnight incubation. No isolations were made from the liver swab after incubation for 72 hr. Gram stains of the colonies revealed small Gram positive coccobacilli. The isolate did not produce endospores or capsules. The isolate grew at 4 C and 30 C in brain heart infusion broths; however, motility was detected only in the cultures grown at 4 C. The isolate produced catalase, but not oxidase, and acid, but not gas in 48 hr at 30 C from glucose, lactose, salicin and maltose. No acid was detected from sucrose or mannitol in 7 days at 30 C.

On the basis of colonial, cellular and biochemical characteristics, the isolate was tentatively identified as *L. monocytogenes* (Carter, 1984, *In Diagnostic Procedures in Veterinary Bacteriology and Mycology*, C. C Thomas, Springfield, Illinois, pp. 196–201). This identification was subsequently confirmed by the Na-

tional Veterinary Services Laboratory in Ames, Iowa.

Formalin-fixed tissues from several organs were processed, sectioned at 6 μ m, stained with hematoxylin and eosin and examined microscopically. Sections of liver revealed a chronic cholangitis characterized by circumscribed zones of fibrous tissue around the portal triads. Some of these zones also had a lymphocytic infiltrate. A parasite containing ova suggestive of a trematode was found within a distended bile duct. Small scattered foci of hepatocellular necrosis, each involving about 10 cells in a single plane, were randomly distributed. Most of the hepatocytes were hypertrophied and there was hyperplasia of Kupffer's cells. A single unidentified microfilaria was found in the sinusoids. No Gram positive organisms were found in sections stained by the Brown and Brenn technique (Luna, 1968, *In Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology*, McGraw-Hill Book Co., New York, New York, pp. 222–223). The testicle was aspermatogenic, and there was focal orchitis, interstitial fibrosis and tubular necrosis. Moderate numbers of lymphocytes were in the aorta adventitia. The myocardial lesions reported by others (Davis et al., 1971, *In Infectious and Parasitic Diseases of Wild Birds*, Iowa State University Press, Ames, Iowa, pp. 146–152; Gray, 1958, op. cit.; Hofstad et al., 1984, op. cit.) were not detected in this bird.

Though frequently associated with other diseases, e.g., coccidiosis, avian leukosis, Newcastle disease and salmonellosis (Hofstad, 1984, op. cit.; Gray, 1964, op. cit.), numerous reports of primary *Listeria* infections have been cited by Gray (1964, op. cit.). Listeriosis is most frequently manifested as a septicemia and the bacterium can be isolated from most of the internal viscera, particularly the liver and spleen. Usually, the most conspicuous lesions are massive areas of myocardial degeneration. Yet focal hepatic necrosis

without cardiac alterations is a common finding in naturally infected birds (Gray, 1958, op. cit.). Other frequently encountered lesions include peritonitis and airsacculitis. In acute cases, the necrotic foci tend to be less marked (Gray, 1958, op. cit.). Histologic lesions in the liver of the wild turkey were consistent with those described by Csontos et al. (1955, Acta Vet. Hung. 5: 261-274), as reported by Gray

(1958, op. cit.). They found foci of necrosis without a marked inflammatory reaction in both liver and spleen. In this case, since *L. monocytogenes*, a known pathogen, was isolated from the affected tissue in pure culture, it seems reasonable to ascribe the lesions encountered as well as the wild turkey's death to *L. monocytogenes*.

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***Frenkelia* sp. from the Brain of a Porcupine (*Erethizon dorsatum*) from Alberta, Canada**

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Cysts of *Frenkelia* spp. have been reported in several species of rodents belonging to the families Muridae, Cricetidae, and Chinchillidae (Frenkel, 1956, Ann. N.Y. Acad. Sci. 64: 215-251; Karstad, 1963, Can. Vet. J. 4: 249-251; Krampitz and Rommel, 1977, Berl. Muench. Tieraerztl. Wochenschr. 90: 17-19; Meingassner and Burtscher, 1977, Vet. Pathol. 14: 146-153; Rommel and Krampitz, 1978, Zentralbl. Veterinaermed. Reihe B 25: 273-281). To our knowledge, the present report is the first to document *Frenkelia* as a natural infection in a porcupine.

The life cycle for any *Frenkelia* spp. has only recently been determined (Rommel and Krampitz, 1975, Berl. Muench. Tieraerztl. Wochenschr. 8: 338-340; Krampitz et al., 1976, Z. Parasitenkd. 51: 7-14; Rommel et al., 1976, Z. Parasitenkd. 50: 204-205; Rommel et al., 1977, Z. Parasitenkd. 51: 139-146). Rommel and Krampitz (1975, op. cit.) determined the life cycle and developmental stages of *F.*

glareoli (= *F. clethrionomyobuteonis*) in the buzzard (*Buteo buteo*) definitive host and bank vole (*Clethrionomys glareolus*) intermediate host. Other researchers have added to this information, noting that the parasite is specific for the definitive host, but not the intermediate one (Rommel et al., 1976, op. cit.; Rommel and Krampitz, 1978, op. cit.; Tadros et al., 1980, Trop. Geogr. Med. 32: 86). *Buteo buteo* is also the definitive host for *Frenkelia microti*. The specific name for *Frenkelia* spp. in North America is not known (Frenkel et al., 1979, Z. Parasitenkd. 58: 115-139).

On 13 August 1984 a female porcupine, in good physical condition, was trapped near Calgary, Alberta, Canada by personnel of the Fish and Wildlife Division and submitted to the Veterinary Laboratory, Animal Health Division, Airdrie, Alberta for examination. The porcupine was kept in captivity at the Veterinary Laboratory until 18 August, when it died. The brain was removed at necropsy and preserved in 10% buffered formalin. Portions of the brain tissue were prepared for histological examination. Histological examination re-

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