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Abstract

Nineteen dead wild turkeys were necropsied and 573 live wild turkeys were physically examined for pathological agents in Arkansas between 1992 and 1997 to determine the proximate role disease may play in declining wild populations in Arkansas. Necropsy of the dead wild turkeys identified avian pox and histomoniasis as the most common diseases (16% and 11% of necropsies, respectively). Avian pox was recorded from three major physiographic regions in the state (Ozark Highlands, Ouachita Mountains, Gulf Coastal Plain). One hen died of non-accidental crop impaction, the fifth occurrence observed in the southeastern United States. Another hen died after developing severe, focal necrotic dermatitis caused by a *Penicillium* sp. fungus, the first occurrence observed in wild turkeys. All live wild turkeys appeared free of gross signs of disease. We found diseases in wild turkeys in Arkansas are not uncommon and are more diverse than previously reported. Continued monitoring of disease in wild turkeys is therefore encouraged.

Introduction

Annual harvest of eastern wild turkeys (*Meleagris gallopavo silvestris*) in the Ouachita Mountains of west-central Arkansas suggested a decline in abundance from 1987 to 1996 (Thogmartin, 1998). Nest predation, reproductive effort, and clutch size have been identified as factors influencing this decline (Thogmartin and Johnson, 1999). Furthermore, body mass of adult male and subadult female turkeys in the Ouachita Mountains declined significantly from 1993 to 1996 (9% and 22%, respectively; Johnson et al., 1996). Conversely, populations in the Ozark Highlands of Arkansas were stationary despite poor reproductive success (Badyaev, 1995).

Our objectives in this study were to report on the presence and prevalence of disease in dead wild turkeys in Arkansas. Furthermore, we wished to reexamine the notion provided by Hopkins et al. (1990) that wild turkey populations in Arkansas were "in good health". Our purpose with this study was not so much to detail occurrence of mortality sources in wild turkeys in Arkansas, the epidemiological details of which will be published elsewhere, as much as it was to discuss their potential influence on wild turkey population ecology and management in Arkansas.

Hopkins et al. (1990) examined only live turkeys captured at bait sites in a study of infectious diseases in wild turkeys. This procedure likely excluded infirmed turkeys unable to travel to bait sites. The study by Hopkins et al. (1990) also suffered from an autocorrelation of results; diseases and parasites carried by one individual in a flock are also likely to have infected other individuals in the captured flock, thus potentially confounding true levels of disease prevalence. Our study controlled for potential autocorrelation of disease results by examining independently gathered specimens. However, our survey was also biased because we sampled only dead wild turkeys recovered in a radiotelemetry project and birds submitted to the Arkansas Game and Fish Commission (AGFC) because they appeared in poor condition. Despite this bias, our study complements the efforts of Hopkins et al. (1990) by sampling turkeys more likely to carry disease, thus providing a more complete perspective of disease in wild turkeys in Arkansas.

Methods

Wild turkeys were captured with rocket nets (Bailey et al., 1980) at White Rock (35°37' to 35°46' N, 93°50' to 94°07' W) and Piney Creek (35°37' to 35°49' N, 93°07' to 93°30' W) Wildlife Management Areas in the Ozark Highlands, Arkansas, from January to March 1992 to 1995 and at Muddy Creek Wildlife management Area (34°39' to 34°52' N, 93°30' to 94°00' W) in the Ouachita Mountains from January to March 1993 to 1997. We ascertained sex by breast and tail feather coloration, age (adult or subadult) from shape and barring patterns on the ninth and tenth primary feathers (Larson and Taber, 1980), and body mass to the nearest 0.1-kg. All birds were inspected for clinical signs

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of disease and other abnormalities including unusually low weight, exudations around the eyes and nares, and presence of gross external lesions indicative of avian pox and other diseases (Couvillion et al., 1991). A topical antiseptic spray was applied to abrasions incurred during capture. Turkeys were outfitted with 110-g motion-sensitive radio transmitters (Telonics, Mesa, AZ) and located two or more times weekly for up to 30 months. Transmitters signaling prolonged inactivity were located and carcasses retrieved. Because of scavenging, predation, and our reluctance to approach inactive hens during the nesting season, only 10 carcasses were retrieved in the three study areas. Wild turkeys collected by hunters ($n = 3$) and AGFC personnel ($n = 6$) from throughout the state were also included in this survey.

Southeastern Cooperative Wildlife Disease Study (SCWDS) personnel in Athens, Georgia, conducted necropsies. All carcasses were sent to them frozen. Condition of the animal at time of recovery influenced the type of necropsy performed (Davidson et al., 1985). All specimens were examined for gross external and internal lesions. Relative physical condition was determined by a visual estimation of fat bodies; for instance, lack of subcutaneous or internal fat reserves resulted in a "fair" rating. Swabs of gross lesions were taken for bacterial cultures and tissues were examined for microscopic lesions when warranted. Radiographs were taken when trauma was suspected.

Results And Discussion

No obvious signs of disease were observed in any of 573 wild turkeys (Ozark Mountains, $n = 298$; Ouachita Mountains, $n = 275$) captured at bait sites. Because this was a post-hoc study conducted ancillary to an investigation into wild turkey population dynamics, we did not investigate subacute diseases in live-caught birds. Sub-clinical diseases may manifest into epidemics after long periods of low level infection, as Davidson et al. (1980) suggested for avian pox in northern bobwhite (*Colinus virginianus*). This also may have been observed by Lutz and Crawford (1987) when

Table 1. Wild turkeys ($n = 19$) submitted to the Southeastern Cooperative Wildlife Disease Study from Arkansas, 1992 - 1996, by source of mortality, sex, age, and county area. A = adult, S = subadult, P = poult. Radio indicates whether wild turkey was radio collared.

	Source of Mortality	Sex	Age	Radio	County Location	Season of Mortality	Parasite Infection	Physical Condition	Comments
Disease									
1	Avian Pox	F	A	N	Montgomery	Autumn	*Heterakis* spp.	Poor	Legally harvested by hunter; emaciated; prominent lesions
2	Avian Pox	F	S	N	Desha	Spring	None	Fair	
3	Avian Pox*	M	A	N	Johnson	Spring	None	N/A	
4	Crop Stasis	F	A	Y	Pope	Winter	*H. gallinarum*, lice	Fair	≥26% mass loss (1.4 kg) in 26 days since capture
5	Histomoniasis	M	S	N	Yell	Summer	Histomonads	Fair	
6	Histomoniasis	M	P	N	Johnson	Spring	Numerous	Fair	4 gastro-intestinal parasite spp.; sustained trauma
7	Necrotic Dermatitis[b]	F	A	Y	Montgomery	Winter	None	Poor	*Heterakis* and *Ascaridia* spp.
8	Fibrinous Perotinitis and Salpingitis	F	A	N	Johnson	Spring	None	Excellent	*E. coli, S. aureus,* and *Enterococcus* infection of oviduct
9	Unknown[c]	M	A	Y	Montgomery	Summer	Numerous	Fair	Enteric disease possible mortality source
10	Unknown Chronic Disease	M	A	Y	Franklin	Summer	None	Poor	Emaciated; secondary condition: gout tophi in kidney sections

[a]Secondary infection of septicemia, contributing to emaciated condition. [b]Severe focal necrotic dermatitis caused by *Penicillium* sp. fungus, at site of trauma.

Table 1. Continued.

	Source of Mortality	Sex	Age	Radio	County Location	Season of Mortality	Parasite Infection	Physical Condition	Comments
Accident									
11	Trauma	M	A	Y	Montgomery	Spring	None	Good	Mortality due to lightning striking roost tree
12	Trauma (Crop Impaction)	F	A	Y	Montgomery	Winter	None	Good	≥28% mass loss (1.2 kg) in 337 days since capture
13	Trauma	M	A	N	Unknown	N/A	N/A	N/A	
Anthropogenic									
14	Gunshot (Legal Harvest)	F	A	N	Chicot	Fall	None	Poor	Evidence of healed gunshot wounds
15	Gunshot (Legal Harvest)	M	A	N	Yell	Spring	Tapeworm (1)	Good	Unrecovered by hunter
16	Gunshot (Legal Harvest)	M	A	Y	Yell	N/A	N/A	N/A	Lesions from previous gunshots
17	Gunshot (Poach)	M	A	N	Yell	Summer	Nematodes (4)	Good	Unrecovered by poacher
18	Capture-related Myopathy	F	A	Y	Yell	Winter	None	Good	Stress-related biochemical changes
19	Capture-related Injury	F	A	Y	Scott	Winter	None	Good	Traumatic injuries resulting from capture

[c]Approximately 100 cecal worms (*Heterakis* spp.), 15 roundworms (*Ascaridia disimilis*), and 25-30 tapeworms (*Raillietina* and *Metroliasthes* spp.) observed in gastrointestinal tract. Emaciated state possibly related to loss of nutrients due to intestinal disease rather than reduced food intake.

they reported avian pox lesions in a small number of captured Merriam's wild turkeys (*M. g. merriami*) in Oregon. Regardless, gross signs of disease were not apparent in live-caught turkeys in Arkansas, similar to turkeys studied by Hopkins et al. (1990).

Infectious disease did, however, cause death for ten recovered wild turkeys (Table 1). Not surprisingly, diseased birds were in worse physical condition than those dying by trauma or by anthropogenic means ($\chi^2 = 9.01$, $P < 0.005$). Infectious disease is common in wild turkeys (Davidson and Wentworth, 1992), and incidence of disease in wild turkeys as well as their associated vectors has been widely reported for the Southeastern and Midwestern U.S. (e.g., Davidson et al., 1985; Castle and Christensen, 1990; Luttrell et al., 1991; Fedynich and Rhodes, 1995).

Three carcasses exhibiting evidence of gross lesions of avian pox were recovered; none of the wild turkeys captured and examined by Hopkins et al. (1990) were infected with avian pox. Avian pox is the most commonly reported disease in wild turkeys in the southeastern U.S. (Davidson et al., 1985) and in Arkansas, pox was identified in three physiographic regions, the Ozark Highlands, Ouachita

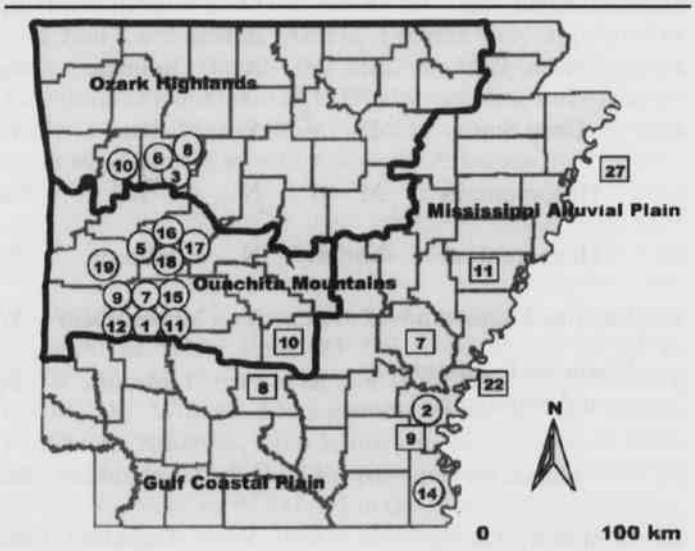

Fig. 1. County map of Arkansas depicting numbered locations (in circles) of recovered dead wild turkeys listed in Table 1, 1992 to 1997. Boxed numbers are specimens described in Maxfield et al. (1963).

Mountains, and Gulf Coastal Plain (Fig. 1). Its negative consequences to fecundity may be important in limiting population growth (Thompson et al., 1997).

This study is the first to report necrotic dermatitis associated with a *Penicillium* sp. The *Penicillium* sp. fungus cooccurred with *Stalphylococcus hyicus, S. aureus, Klebsiella oxytoca,* and *Bacillus* sp. bacteria in an infection of the wing. While it is not clear whether this infection was a direct source of mortality (there were additional infections of the spleen, kidney, and duodenum), it likely was severely debilitating and thus the main contributor to mortality. Mycoses (fungal infections) in general are rare in wild turkeys (Hopkins et al., 1990). Davidson et al. (1985) reported one respiratory case of fungal infection in turkeys and believed it to be unique (Davidson and Wentworth, 1992). Hopkins et al. (1990) reported several infections of turkeys by *Aspergillus fumigatus,* a common soil fungus.

Bacterial infection may be a more frequent source of natural mortality in wild turkeys than previously demonstrated (Davidson and Wentworth, 1992). A single case of salpingitis and fibrinous peritonitis (inflammation of the oviduct and body cavity) resulted from infection by *Staphylococcus aureus, Eschericia coli,* and *Enterococcus* spp. This syndrome is uncommon in wild turkeys; Davidson et al. (1985) reported one case of salpingitis. *Eschericia coli* is normally found in the intestinal tract and operates opportunistically following stress, internal trauma, or other infection (Woodard et al., 1993).

In our study, two of 19 (11%) turkeys exhibited histomoniasis (blackhead disease), a disease caused by *Histomonas meleagridis.* Blackhead is the second most commonly reported disease in wild turkeys and represents about 12% of turkey accessions to SCWDS (Davidson and Wentworth, 1992). Blackhead has been reported in numerous studies of wild turkeys (Kozicky, 1948; Amundson, 1985; Davidson et al., 1985) including turkeys in Arkansas (Hopkins et al., 1990). Emaciation and reduced activity are common clinical signs associated with advanced cases of blackhead disease. Blackhead can reduce growth in young birds (Woodard et al., 1993) and cause early cessation of laying in adult hens (Davis et al., 1971), suggesting this pathogen may affect wild populations by increasing risk of predation and reducing fecundity.

Data regarding diversity, geographic distribution, and prevalence of parasites in wild turkeys of Arkansas is incomplete. Maxfield et al. (1963) reported gastrointestinal helminths in 76 wild turkeys from southeastern Arkansas (Fig. 1). In our survey, at least seven of 17 examined birds were infected with endoparasites; partial scavenging precluded examination for parasite infection in two birds. In our survey, one adult male turkey from Montgomery County was parasitized by *Brachylaema* sp., extending observed incidence of this trematode parasite northwest into the Ouachita Mountains. Maxfield et al. (1963) reported *Brachylaema* sp. infection in one of 45 turkeys in Desha county in southeastern Arkansas. *Ascaridia disimilis* (nematode), *Heterakis gallinarum* (nematode), *Metroliasthes lucida* (cestode), and *Raillientina* spp. (cestodes), four helminths observed in our study, were also common in the viscera of birds examined by Maxfield et al. (1963). Parasite burden may alter reproductive performance, intraspecific competition for food resources, and opportunities for mating (Hudson and Dobson, 1991), but the helminth species observed in our study are not generally associated with severe consequences to otherwise healthy animals.

Trauma is another leading source of mortality in wild turkeys (Davidson and Wentworth, 1992) and in this study three carcasses exhibited injuries suggesting mortality related to trauma. Fatal injuries included fractured bones in wings, keel, beak, and neck, and crop impaction. Crop impaction often occurs when ingested vegetation is blocked in the esophagus due to trauma in the neck area. However, rare cases of crop impaction are not accident-related, but instead may have their origins in lead poisoning or genetic malfunction (Davidson et al., 1985). Our single case of crop impaction (or crop stasis) was believed to be a non-accident related case (SCWDS necropsy report 52-94). SCWDS reported only four other incidences of crop stasis not attributed to accidental trauma in over 250 wild turkeys necropsied between 1975 and early 1994 (Davidson et al., 1985; SCWDS necropsy report 52-94).

Several authors have suggested turkeys that die from gunshots and are unrecovered during the hunting season may vary between 7 and 30% of all hunting season mortalities (Mosby and Handley, 1943; Bailey and Rinell, 1967; Everett et al., 1978). Three of 19 birds (15.8%) in this study exhibited evidence of healed gunshot wounds. Two of three were eventually harvested, likely within the same season they received the initial wound. The full impact of this disturbance to population dynamics has not been evaluated in Arkansas, but it may be high. One of three birds radiographed in New York, for instance, was positive for shot (S. D. Roberts, pers. comm.), whereas in Washington, 15% of examined pheasants (Phasianus sp.) were carrying shot (9% of females, 32% of males; D. S. Galbreath and E. S. Dziedzic, unpub. rept.).

Conclusions

Results from our survey suggest disease is not uncommon and more diverse in wild turkeys of Arkansas than previously reported. The prevalence of disease, however, has yet to be fully ascertained due to biased collection methods in this and past studies. Without more comprehensive collections from captured wild turkeys for bacterial and viral

isolation, we cannot know if sub-clinical infection by avian pox, histomoniasis, or other diseases may be limiting reproduction in the Interior Highlands of Arkansas. Mortality from infectious diseases (avian pox and histomoniasis) comprised half of all diseased birds and approximately one-quarter of all necropsies in this study. Continued research on the extent of these diseases in Arkansas is therefore warranted. We recommend a more systematic inspection of wild turkey pathologies in Arkansas. We urge the testing of blood, feces, and throat swabs of all captured wild turkeys.

Wild turkeys captured for translocation should be tested for disease prior to introduction to new areas to prevent dissemination of pathogens into unaffected flocks (Maxfield et al., 1963; Hopkins et al., 1990). Greater knowledge of disease occurrence within source and recipient populations in Arkansas should also aid in managing disease outbreaks. Lastly, dead birds should continue to be necropsied periodically to aid in monitoring disease outbreaks in Arkansas.

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