

A Survey of Parasites and Diseases of Pen-raised Wild Turkeys

- L. F. Schorr,¹ W. R. Davidson, V. F. Nettles,** *Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia, Athens, GA 30602.*
- J. E. Kennamer,** *National Wild Turkey Federation, P.O. Box 530, Edgefield, SC 29824.*
- P. Villegas,** *Poultry Diagnostic Research Center, College of Veterinary Medicine, University of Georgia, Athens, GA 30602.*
- H. W. Yoder,** *Southeast Poultry Disease Laboratory, Agriculture Research Service, U.S. Department of Agriculture, Athens, GA 30605.*

Abstract: Evaluations of health status were made on 119 pen-raised wild turkeys (*Meleagris gallopavo*) by complete necropsy, serological, and microbiological testing, blood smear examinations, subinoculation trials, and parasite identification. At least 33 species of parasites including 9 protozoans, 14 helminths, and 10 arthropods were found. Infectious disease agents isolated or identified histopathologically were avian pox virus, *Mycoplasma gallisepticum*, and *Aspergillus fumigatus*. Serologic testing disclosed antibodies to infectious bursal disease virus-2, *M. gallisepticum*, *M. meleagridis*, and *Salmonella* spp. Based on an epidemiologic evaluation of the disease risks, we conclude that the release of pen-raised wild turkeys without proper consideration for disease prevention should be discouraged or prohibited.

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Wild turkey restoration in the United States has been one of the most noteworthy successes of wildlife management (Lewis 1987). Although the original range of the wild turkey covered most of the United States, excessive hunting and loss of habitat caused rangewide declines in numbers. Between 1900 and 1950, entire populations were eliminated from some states and numbers reached all time lows. As a result of restocking programs, the turkey has reoccupied much of its original range and now occurs in every state except Alaska (Lewis 1987).

¹Present address, National Wildlife Health Research Center, 6006 Schroeder Rd., Madison, WI 53711.

Numerous ineffective attempts have been made to replenish wild turkey populations by releasing pen-raised stock. The major turning point in wild turkey restoration was the movement away from pen-raised birds to use of live-trapped native wild turkeys for restocking (Mosby and Handley 1943, Lindzey 1967, Bailey and Rinell 1968, Lewis 1987). The low success of pen-raised birds in the wild has led many professional wildlife managers to doubt if these birds have the necessary "wildness" for survival. Furthermore, most biologists fear that the pen-raised birds harbor parasites or diseases which could be detrimental to wild turkeys and other wildlife. Parasites and diseases in pen-raised birds also could be responsible for early mortality of released birds.

Release of pen-raised turkeys remains a common practice on private land and hunting preserves. This activity is conducted solely in the private sector since state wildlife management agencies have abandoned the practice (Lewis 1987). Releases on private property are controversial, and most wildlife agencies view them as a threat to native wild turkeys. Despite frequent mention of disease potentials, only limited speculative and circumstantial evidence is available on this subject. For example, Wunz (1971) noted that histomoniasis (blackhead disease) was responsible for excessive mortality of pen-raised wild turkeys in Perry County, Pennsylvania, and Powell (1965) implied that the release of pen-raised turkeys in Volusia County, Florida, initiated a decimating outbreak of avian pox in native wild turkeys.

The purpose of this study was to evaluate pen-raised wild turkeys for important parasites or diseases that: (1) may be introduced into native wild turkeys or other species by the release of infected pen-raised stock, (2) may cause mortality of pen-raised wild turkeys *per se*, or (3) could be of potential significance to the domestic poultry industry.

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Methods

From November 1984 through January 1985, pen-raised "wild" turkeys were acquired from 12 locations (Fig. 1) by the NWTF and the respective state fish and wildlife agency. The turkeys were acquired in a manner that would simulate someone buying the birds for release into the wild. An attempt was made to obtain a general history on the birds at the time of purchase.

The necropsy protocol was designed to examine each turkey for numerous parasites and diseases. After a blood sample was obtained, the birds were killed by cervical dislocation, and their weight and sex recorded. Age was determined by plumage characteristics (Leopold 1943). Two thin blood slides were made, fixed with methanol, and stained using Giemsa stain. Approximately 10,000 red blood cells per slide were scanned for hemoparasites under oil immersion at 1,000X.

Fresh serums were tested for antibodies to *Mycoplasma gallisepticum*, *M. synoviae* and *M. meleagridis* by the rapid plate agglutination (RPA) test and the hemagglutination inhibition (HI) test (Yoder 1980). Plate test readings of +3 or +4 were considered positive, and titers > 1:40 were considered positive on the HI test. Tracheal swabs and sections of trachea were cultured for *Mycoplasma* by methods described by Yoder (1980).

Serum samples initially were screened for antibodies to *Salmonella* spp. by an RPA test (Anonymous 1979), and any samples with reactivity were retested by the tube agglutination test to distinguish antibodies against *S. pullorum*/*S. typhimurium* (Anonymous 1979). Cloacal swabs and a portion of the mid-intestine were thawed and cultured for *S. pullorum* (Williams et al. 1980) on all birds with suspected antibody test reactions.



Figure 1. Geographic distribution by county of 12 locations from which pen-raised wild turkeys were examined.

A virus neutralization test was utilized to detect antibodies against infectious bursal disease virus (Winterfield 1980). Antibodies for hemorrhagic enteritis were surveyed by the agar gel precipitin test (Domermuth et al. 1972). Serums were tested for titers to avian influenza viruses and paramyxoviruses using a single radial immunodiffusion test by St. Jude's Children's Research Hospital, Memphis, Tennessee. Tracheal and cloacal swabs also were provided to St. Jude's for avian influenza virus isolation attempts by egg inoculation (Hinshaw et al. 1986).

Ectoparasites were collected by brushing the entire body of each bird as it was held over a white enamel tray. Any debris on the tray was washed into a vial containing 70% ethyl alcohol. Ectoparasites were separated and mounted in Hoyer's medium. Lice were identified by R. Gerrish of the National Veterinary Services Laboratory in Ames, Iowa. One chigger specimen was examined by L. Goff, Entomology Department, University of Hawaii, and feather mites were identified by W. Atyeo, Department of Entomology, University of Georgia.

Necropsy procedures and examination of viscera for parasites were done according to the procedures described by Prestwood (1968). Crops and esophagi were examined grossly for nematodes and later digested in pepsin and reexamined (Pritchard and Kruse 1982). The proximal one-third of the trachea was collected for immediate culture attempts for *Mycoplasma* organisms and swabs for microbiological studies were taken of any gross lesions or wounds. Major visceral organs were inspected *in situ* for lesions and representative sections of each organ were preserved in 10% neutral buffered formalin. Tissues were processed by standard histological procedures and stained with hematoxylin-eosin (HE) for microscopic examination. Portions of brain, liver, lung, spleen, mid-intestine, kidney, and trachea were collected aseptically and frozen for later microbiologic studies as required.

Female *Heterakis* were removed from the ceca and stored under refrigeration (4°C) in 1% formalin for subinoculation trials to detect the presence of *Histomonas meleagridis*. Eggs within female *Heterakis* were allowed to embryonate and then were fed to domestic poult according to the protocol described by Horton-Smith and Long (1956). The experimental birds were monitored daily and were killed at 16 days post-infection. Livers and ceca were examined grossly for evidence of histomoniasis, and tissue sections of any lesions were placed into 10% buffered formalin for microscopic examination. The ceca were opened, contents washed in a 100- μ m sieve, and the washings examined through a dissecting microscope for *Heterakis* larvae.

Coccidial oocysts in fecal samples were allowed to sporulate in 2% potassium dichromate, and Sheather's flotations were performed. For species identification, sporulated oocysts were inoculated *per os* into 2- to 4-week-old domestic poults. Feces from inoculated poults was collected and examined for oocysts by sugar flotation. Poults were killed after 6 days, and scrapings from duodenum, jejunum, ileum, and ceca were examined and oocysts measured. Tissue samples from the same regions were fixed in 10% neutral buffered formalin, stained with HE stain and examined microscopically. Examination of fecal samples and the tissue sections was done by M. Ruff, Animal Health Laboratory, U.S. Department of Agriculture,

Beltsville, Maryland, and the results have been described separately (Ruff et al. 1988).

Results

Age and sex of birds varied among the sources sampled. Of 119 turkeys, 42 were males and 77, females. The age distribution for males was 40% adults, 7% subadults and 53% juveniles. Of the females in the sample, 19% were adults, 11% were subadults, and 70% were juveniles. Physical condition ratings for the turkeys were: 23% in excellent condition, 56% in good condition, and 21% in fair condition. Body weights ranged from 1.22 to 11.34 kg.

Background information that accompanied each shipment generally was limited, and attempts were not made to verify the histories reported by the suppliers. Ancestors for 4 flocks were unknown. Three flocks originated from game farms and 3 came from breeding wild gobblers to domestic hens. Two sources indicated the turkeys were derived from eggs taken from the wild. Eleven of the 12 premises had other domesticated fowl including the following species: ducks, geese, chickens, guinea fowl (*Numida meleagris*), ring-necked pheasants (*Phasianus colchius*), golden pheasants (*Chrysolophus pictus*), chukar (*Alectoris graeca*), bobwhite quail (*Colinus virginianus*), pigeon (*Columbia livia*), and mourning doves (*Zenaidura macroura*).

Sheather's flotations demonstrated 74 *Eimeria*-infected turkeys. Subinoculating these samples in domestic poulters produced infection in 34 instances. Four additional turkeys that were negative by Sheather's flotation produced *Eimeria* infections by subinoculations. Only 30 fecal samples yielded sufficient oocysts to permit species identification. Species and prevalences among those 30 were *E. meleagritidis* (97%), *E. gallopavonis* (47%), *E. meleagridis* (27%), *E. dispersa* (17%), *E. innocua*/*E. subrotunda* (13%), *E. adenoides* (7%), and an unnamed species (3%). The majority of infected turkeys harbored mixed infections of *Eimeria*. Of the 30 turkeys, 30% were infected only with *E. meleagritidis*, 40% harbored 2 species, 20% had 3 species and 10% were infected by 4 *Eimeria* spp. Lesions attributable to coccidiosis were not noted.

Protozoan blood parasites observed were *Haemoproteus meleagridis* and *Leucocytozoon smithi*. Mixed infections of these were found in 5% of the turkeys. The parasitemia in all infections was low. Although counts were not done, in most cases only < 2 parasites were noted on each blood smear.

Diagnosis of *Histomonas meleagridis* infection was by a combination of methods. Gross examination of livers and ceca at necropsy was the initial opportunity for detection of clinical histomoniasis. Small nodules were noted in the ceca of 9 turkeys. Histologic examination of these nodules revealed only nonspecific typhlitis with lymphoid hyperplasia in all but 1 of these birds. However, in 1 turkey from Winona County, Minnesota, *Histomonas* organisms were demonstrated with HE and periodic acid Schiff (PAS) stained sections indicating subclinical histomoniasis.

Subinoculation trials with embryonated *Heterakis* eggs pooled by sample area

demonstrated subclinical histomoniasis in turkeys from Early County, Georgia. Poults experimentally inoculated with *Heterakis* eggs from the Early County sample developed cecal cores typical of histomoniasis accompanied by inflammation of the cecal mucosa and small focal areas of hemorrhage. Histopathologic examination of ceca from these experimental poults revealed many histomonads. Liver lesions were not evident on gross or histologic examination, and organisms were not found in liver sections from these poults.

Fourteen species of helminth parasites were recovered, of which 10 were located in the intestinal tract (Table 1). Additional species were found in the trachea, under the gizzard lining, in the mucosa of the crop and esophagus and from the body cavity. Significant lesions were not attributed to infections with helminth parasites.

Arthropod parasites including members of Acarina, Mallophaga, and Psocoptera infested 72% of the turkeys. Infestations were not quantified; however, the prevalence of ectoparasites found on the turkeys was determined (Table 1). The turkeys did not appear to have clinical problems as a result of ectoparasite infestations and associated lesions were not found.

Lesions attributable to avian pox were found in 9 turkeys from Early County, Georgia (Table 2). The birds had localized epithelial hyperplasia with the formation of nodules over the head and neck areas. Microscopic examination of lesions disclosed eosinophilic intracytoplasmic inclusion bodies characteristic of pox virus in 1 bird.

Antibody titers ranging from 1:40 to 1:2,560 to infectious bursal disease virus-2 (IBDS-2) were noted in 55% of the turkeys. Turkeys from 2 study areas appeared to be serologically negative for antibodies to IBDS-2; turkeys from Crawford County, Pennsylvania, and Early County, Georgia, had very low titers (<1:20). All turkeys tested negative for antibodies for hemorrhagic enteritis virus, avian influenza viruses, and paramyxoviruses. Isolation attempts for avian influenza viruses also were negative.

Turkeys from 1 of the 12 locations had infectious sinusitis from *Mycoplasma gallisepticum*. Infection was dramatic in turkeys from South Carolina where 7 of 10 birds had ocular discharge in 1 or both eyes and swelling of the infraorbital sinuses. Of 10 turkeys, 7 had positive RPA tests, and HI titers of > 1:40 were recorded in 6 birds. Culture of sinuses and tracheas in these 10 birds resulted in isolation of *M. gallisepticum* from 4 turkeys. Three isolations were from infraorbital sinus swabs and 1 was from a tracheal swab. Histologic examination of respiratory epithelium disclosed a chronic inflammatory response typical of *M. gallisepticum* infection.

Antibodies to *M. meleagridis* were found in turkeys from 2 sample sites. Two birds from Blue Earth County, Minnesota, had positive plate tests (+ 4) and HI titers of 1:80. Of 10 birds samples from Richfield County, Pennsylvania, 1 bird had a positive plate test (+ 3) and an HI titer of 1:80. Attempts to culture *M. meleagridis* from frozen lung tissue from all turkeys in these groups were unsuccessful.

Serum testing for antibodies to *Salmonella* organisms disclosed 24 turkeys with some degree of reactivity on the plate test. Seropositive turkeys were from 8

Table 1. Prevalence, intensity and maximum number of parasites found in 119 pen-raised wild turkeys obtained from 12 locations.

Parasite	Prevalence (%)	Intensity ^a	Maximum ^a
<i>Eimeria adenoides</i>	1.7		
<i>E. dispersa</i>	4.2		
<i>E. gallopavonis</i>	11.8		
<i>E. innocua/E. subrotunda</i>	3.4		
<i>E. meleagridis</i>	6.7		
<i>E. meleagritidis</i>	24.3		
<i>Eimeria</i> sp.	41.1		
<i>Haemoproteus meleagridis</i>	15.9		
<i>Histomonas meleagridis</i>	1.7		
<i>Leucocytozoon smithi</i>	17.6		
<i>Hymenolepis</i> sp.	1.6	1.0	1
<i>Ascaridia dissimilis</i>	17.0	11.5	74
<i>Ascaridia</i> sp.	49.0	12.0	134
<i>Capillaria anatis</i>	32.0	30.1	144
<i>C. annulata</i>	11.0	4.2	12
<i>C. bursata</i>	2.5	1.7	2
<i>C. caudinflata</i>	21.0	65.9	392
<i>C. contorta</i>	25.0	9.3	45
<i>C. obsignata</i>	53.0	95.9	756
<i>C. phasianina</i>	10.0	96.0	415
<i>Capillaria</i> sp.	18.5	19.5	66
<i>Cheilospirura hamulosa</i>	2.5	1.3	2
<i>Heterakis gallinarum</i>	65.5	35.6	267
<i>Heterakis</i> sp.	7.5	4.2	16
<i>Singhilaria hayesi</i>	0.8	1.0	1
<i>Syngamus trachea</i>	6.7	4.0	10
<i>Trichostrongylus tenuis</i>	8.4	12.1	29
<i>Dermoglyphus</i> sp.	0.8		
<i>Megnina ginglymura</i>	72.3		
<i>Neotrombicula richmondi</i>	0.8		
<i>Pterolichus</i> sp.	6.7		
<i>Pterygocrusolichus chanayi</i>	4.2		
<i>Chelopistes meleagridis</i>	18.4		
<i>Chelopistes</i> sp.	31.1		
<i>Menacanthus stramineus</i>	5.0		
<i>Menacanthus</i> sp.	17.6		
<i>Oxylipurus corpulentus</i>	2.5		
<i>O. polytrapezius</i>	2.5		
<i>Oxylipurus</i> sp.	9.2		
Mallophaga nymph	19.3		
<i>Psocid</i> sp.	0.9		

^aBlank indicates parasites not counted.

Table 2. Prevalence of disease agents found in 119 pen-raised wild turkeys obtained from 12 locations.

Disease agent	Prevalence (%)	Type of test
Avian pox virus	6.0	Histology
Infectious bursal disease virus-2	55.0	Serology
<i>Mycoplasma gallisepticum</i>	5.0	Serology
<i>Mycoplasma gallisepticum</i>	3.4	Isolation
<i>Mycoplasma meleagridis</i>	2.5	Serology
<i>Salmonella pullorum</i> / <i>gallinarum</i>	6.7	Serology
<i>Salmonella typhimurium</i>	7.6	Serology
<i>Aspergillus fumigatus</i>	0.8	Histology

collection sites: 3 birds from Iowa, 5 from Missouri; 4 from Maryland; 1 from Winoona County, Minnesota; 5 from Alabama; 3 from Blue Earth County, Minnesota; 2 from North Carolina; and 1 from Georgia. When sera of these 24 turkeys were studied by the tube agglutination test, 12 birds were seropositive and 12 were seronegative. Nine were seropositive for *S. pullorum*/*S. gallinarum* and 8 were seropositive for *S. typhimurium*. Five birds had dual reactions to *S. pullorum*/*S. gallinarum* and *S. typhimurium* antigens. Attempts to isolate salmonellae from cloacal swabs and sections of intestine from the seropositive samples were unsuccessful. Despite negative culture attempts, 1 turkey from Moore County, North Carolina, was considered a suspect paratyphoid (*S. typhimurium*) case based on positive serologic tests and lesions compatible with reported salmonellosis in wild turkeys (Howerth 1985).

One mycotic infection was observed in a juvenile female turkey from Snyder County, Pennsylvania. At necropsy, a 2 cm × 1 cm × 3 mm thick white plaque surrounded by a gelatinous exudate was found in the air sac overlying the right kidney. Microscopic examination of the air sac revealed chronic focal airsacculitis with septate branching fungi. In addition to HE stain, PAS stain was used to make a histologic diagnosis of aspergillosis.

A variety of gross lesions was noted at necropsy. Most of these lesions were chronic inflammatory reactions for which the etiologic agent could not be confirmed by histopathologic examination of the lesions or other tissues. The following histologic diagnoses were made: ulcerative dermatitis ($N = 11$), hepatic fibrosis/lymphoid hyperplasia/microgranulomas ($N = 8$), pulmonary lymphoid hyperplasia/pneumonia ($N = 6$), capture myopathy ($N = 7$), epicarditis/myocarditis ($N = 2$), diphtheritic epiglottitis ($N = 2$), and single instances of airsacculitis, diphtheritic ingluvitis, tarsal tendonitis, and retained ovum.

Several physical characteristics were noted that were not typical of bonafide wild turkeys. Turkeys from Minnesota and Georgia had very obvious white feather coloration on the primary and tail feathers. In addition to white coloration, birds from Georgia were very large and heavily bodied with short, stocky legs. Deviation

of the keel bone was noted in the birds from the same 2 areas. Several birds from North Carolina exhibited greenish discolored muscular tissue on the wing tips attributed to prior trauma.

Discussion

The majority of the parasites and diseases identified from pen-raised turkeys in this study also have been reported from bonafide wild and domesticated turkey populations. For the agents in question, the mode of transmission is either direct or by intermediate or transport hosts that are readily available in vast areas of wild turkey habitat. Therefore, it would appear that cross-transmission among the 3 types of turkeys should be considered feasible.

More important than transmissibility is the question of pathogenicity. Pen-raised wild turkeys and domestic turkeys are raised in confinement by similar husbandry techniques, and thus, they could have essentially the same epidemiological status and susceptibility to the parasites and diseases found in this study. However, the implications of parasites and diseases found in pen-raised wild turkeys to bonafide wild turkey populations are less certain. For example, many of the known pathogens of domestic turkeys occur in wild turkeys but have not been associated with clinical disease. In such cases, morbidity may not occur because the dilution effect of vast areas of free range prevents the potential wild turkey host from receiving an overwhelming dose of the pathogen.

For each agent, an estimation of the potential impact on wild turkey populations was made. Parasites and diseases that were considered to have a low impact can be divided into 2 subcategories. First, there are organisms not considered problems because they have not been shown to cause sickness or death in any type of turkey. This group includes *Ascaridia* sp., *Capillaria anatis*, *C. bursata*, *C. caudinflata*, *C. phasianina*, *Cheilospirura hamulosa*, *Chelonistes* sp., *Dermoglyphus* sp., *Hymenolepis* sp., *Megnina ginglymura*, *Menacanthus* sp., *Neotrombicula richmondi*, *Oxylipеurus* sp., *Psocid* sp., *Pterolichus* sp., *Singhifilaria hayesi* and infections bursal disease virus-2.

A second group of organisms with at least some pathogenicity, particularly for domestic turkeys, includes *Eimeria* spp., *Haemoproteus meleagridis*, *Leucocytozoon smithi*, *Capillaria annulata*, *C. contorta*, *C. obsignata*, and *Trichostrongylus tenuis*. These organisms are not considered significant threats to wild populations because they occur in wild turkey populations without known impact. Thus, adverse consequences to wild turkeys from release of pen-raised birds infected with these agents should be minimal. Nevertheless, high concentrations of infected pen-raised birds could produce conditions suitable for disease production when intermingling with wild birds.

The remaining parasites and diseases were considered potential threats to wild turkey populations. They included *Histomonas meleagridis*, *Syngamus trachea*, avian pox virus, *Mycoplasma gallisepticum*, *M. meleagridis*, and *Salmonella* spp.

If transferred from pen-raised turkeys to their wild counterparts, these pathogens could result in sickness or death.

Histomoniasis can be a significant disease at game farms. Reports from the Pennsylvania Game Commission (PGC) indicate that blackhead was one of the major causes of death following release of pen-raised turkeys into the wild (Roberts 1954, Snyder 1952). The PGC also has suggested that pen-raised turkeys introduced histomoniasis into wild turkey populations with resultant losses of recruitment of poults (Wunz 1971).

Wild turkeys appear to be very susceptible to histomoniasis. Mortality resulting from histomoniasis was responsible for 10% of all sick or dead wild turkeys examined during a 13-year period in the southeastern United States (Davidson et al. 1985). During a 1-year period in Mississippi, 3 wild turkeys with histomoniasis were found, and it was suggested that histomoniasis is a widespread cause of mortality (Hurst 1980). This conclusion, although based on few observations, should be considered valid since most turkeys debilitated by histomoniasis probably are taken by predators and are never found.

The infected turkeys in this study appeared to have subclinical histomoniasis since they did not have severe gross lesions. Had the birds been kept alive, clinical disease might have developed. Regardless of their eventual fate, such birds should be considered potential sources for spreading *H. meleagridis*-infected cecal worms upon release. This situation could result in transmission to bonafide wild turkeys and other susceptible galliform birds such as bobwhite quail or ruffed grouse.

Gapeworms, *Syngamus trachea*, were found in pen-raised turkeys from Baltimore County, Maryland. Poults are more susceptible to gapeworm infections than adult birds, and although *Syngamus* is encountered infrequently in the wild, it is usually in young birds. Disease associated with this nematode is due to inflammation and obstruction of the trachea with resultant suffocation (Ruff 1984). Wunz (1971) reported *S. trachea* as a cause of mortality in released game farm birds. On this basis, *S. trachea* should be considered as a potentially pathogenic nematode that could cause mortality in wild poults.

Lesions attributable to avian pox virus on the head and neck of 9 turkeys from Early County, Georgia, were considered important. Lack of inclusion bodies in all but 1 of these birds suggested that these turkeys had chronic infections that were healing. Still, all of these birds should be considered potential sources of virus since the virus may exist in non-clinical, latent infections in some birds (Tripathy and Hanson 1975). As importantly, some sloughing scabs probably contained virus even though inclusion bodies were not detectable.

Avian pox viruses are prevalent worldwide and vary in host range and virulence. In domestic turkeys, the morbidity and mortality from pox infections are dependent upon the virulence of the virus and the form of infection. Cutaneous infection (dry pox) generally affects the head and feet without serious damage. Conversely, the diphtheritic form (wet pox) affects the mucus membranes of the eye or oral cavity and produces greater debilitation. Most mortality occurs when pox pro-

duces diphtheritic infection on the conjunctival or oral mucosa, which in turn causes blindness, starvation, or asphyxia.

Avian pox is an important disease for wild turkeys. This viral infection was the most prevalent disease observed in clinically ill turkeys from the southeastern United States (Davidson et al. 1985). Wild turkey die-offs from avian pox have been reported in Alabama (Davis 1966) and Florida (Forrester, unpubl. data). Of particular significance to this study is the decline of a wild turkey population in Florida from avian pox that was thought to have been introduced by release of infected pen-raised turkeys (Powell 1965). Since avian pox viruses are relatively stable in the environment and can be transmitted mechanically by contact with infected birds or by mosquitoes, release of pen-raised turkeys with active infections should be regarded as a significant disease threat.

Identification of *Mycoplasma gallisepticum* (MG) infections was probably the most alarming finding of this study. *Mycoplasma gallisepticum* is a well-known pathogen in domestic poultry that can cause economic loss (Yoder 1978). The status of mycoplasmosis in wild turkeys has been a recent topic of concern for the Wildlife Disease Association (WDA 1985), the International Association of Fish and Wildlife Agencies (Nettles 1984) and the United States Animal Health Association (Nettles and Thorne 1982). Reports of mycoplasmosis in wild or semi-wild turkeys in several states led to an accelerated effort to prevent potential exposure of native wild turkey populations in other areas to this disease (Hensley and Cain 1979, Amundson 1981, Davidson et al. 1982, Jessup et al. 1983).

The MG infections diagnosed in wild turkeys are thought to have originated from contact with infected free-ranging domestic poultry (Davidson et al. 1982, Jessup et al. 1983). Many disease specialists maintain that backyard poultry and pen-raised galliform game birds are major sources of *Mycoplasma* organisms (WDA 1985). Transmission of MG is by direct contact with a carrier bird, in airborne dust, contaminated equipment, or by egg transmission. Considering the methods of transmission, close confinement of pen-raised birds would increase the rate of transmission and prevalence of MG infections.

Evidence of *M. meleagridis* (MM) in pen-raised wild turkeys also was alarming. In the domestic poultry industry, MM causes economic losses of \$9.4 million per year from hatchability losses and the costs of egg treatment (Carpenter et al. 1981). *Mycoplasma meleagridis* has not been reported in free-ranging wild turkeys and this study is the first report of MM seropositive pen-raised wild turkeys. Although MM potentially could enter wild turkey populations where wild and pen-raised birds interbreed, it does not appear to be a major disease threat compared to MG.

The serum rapid plate tests, conducted for detection of possible pullorum and fowl typhoid reactors, used antigens shared by *S. pullorum* and *S. gallinarum*, the respective causative agents of these diseases. The rapid plate test alone is equivocal, however, because antibodies to paratyphoid salmonellae such as *S. typhimurium* as well as other bacteria may produce false positive results (Williams et al. 1980). Results of tube agglutination tests suggested that most of the plate test reactors to

pullorum/fowl typhoid antigens likely were false positives since only 9 samples reacted to pullorum/fowl typhoid antigens by the tube test. The tube test also may give false positives, and in the absence of confirmatory organism isolations, must be interpreted with caution. Because all attempted salmonellae isolations were negative, the ultimate status in regard to salmonellae infections must be considered undetermined. The tremendous economic importance of all salmonellae infections to the domestic poultry industry, however, is sufficient reason for further clarification of the status of these agents in pen-raised wild turkeys.

When evaluating the findings of this study, it is important to recognize 2 factors relative to disease detection and epidemiology. First, the diseases detected represent the minimal number present in pen-raised turkeys. Examination of more turkeys from other sources would have a good probability of disclosing diseases not present in the birds examined. Second, this study represents what was present at a particular point in time in the groups of birds examined. Resampling of the same premises might reveal the presence of additional diseases or that some diseases are no longer present.

This documents that pen-raised wild turkeys harbor infectious agents that are potential threats to free-ranging wild turkeys and domestic turkeys. These agents also are capable of causing substantial mortality in released penned birds. Therefore, release of pen-raised wild turkeys without proper consideration for disease prevention should be discouraged, if not prohibited.

Literature Cited

- Amundson, T. 1981. Disease surveillance plans for wild turkey reintroduction program in Wisconsin. *Am. Assoc. Wildl. Vet. Newsletter* 6:4-5.
- Anonymous. 1979. The national poultry improvement plan and auxiliary provisions. U.S. Dep. Agric., Agric. Res. Serv. Publ. ARS-NE-32-2, Beltsville, Md. 49pp.
- Bailey, R. W. and K. T. Rinell. 1968. History and management of the wild turkey in West Virginia. *W. Va. Dep. Nat. Resour. Bul. 6.*, Charleston. 59pp.
- Carpenter, T. E., R. K. Edson, and R. Yamamoto. 1981. Decreased hatchability of turkey eggs caused by experimental infections with *Mycoplasma meleagridis*. *Avian Diseases* 25:151-156.
- Davidson, W. R., V. F. Nettles, C. E. Couvillion, and E. W. Howerth. 1985. Diseases diagnosed in wild turkeys (*Meleagris gallopavo*) of the southeastern United States. *J. Wildl. Diseases* 21:386-390.
- Davidson, W. R., V. F. Nettles, C. E. Couvillion, and H. W. Yoder. 1982. Infectious sinusitis in wild turkeys. *Avian Diseases*. 26:402-405.
- Davis, J. R. 1966. Internal and external parasites and diseases of wild turkeys. *Ala. Dep. Conserv., Annu. Progress Rep.*, Montgomery. 28pp.
- Domermuth, G. H., W. B. Gross, R. T. Dubose, C. S. Douglass, and C. B. Rebusch, Jr. 1972. Agar gel diffusion precipitin test for hemorrhagic enteritis of turkeys. *Avian Diseases* 16:852-857.
- Hensley, T. S. and J. R. Cain. 1979. Prevalence of certain antibodies to selected disease-causing agents in wild turkeys in Texas. *Avian Diseases* 23:62-69.

- Hinshaw, V. W., V. F. Nettles, L. F. Schorr, J. M. Wood, and R. G. Webster. 1986. Influenza virus surveillance in waterfowl in Pennsylvania after the H5N2 avian influenza outbreak. *Avian Diseases* 30:207-212.
- Horton-Smith, C. and P. L. Long. 1956. Studies in histomoniasis. *Parasitology*. 46:79-90.
- Howerth, E. W. 1985. Salmonellosis in a wild turkey. *J. Wildl. Diseases* 21:433-434.
- Hurst, G. A. 1980. Histomoniasis in wild turkeys in Mississippi. *J. Wildl. Diseases* 16:357-358.
- Jessup, D. A., A. J. DaMussa, R. Lewis, and K. R. Jones. 1983. *Mycoplasma gallisepticum* infection in wild type turkeys living in close contact with domestic fowl. *J. Am. Vet. Med. Assoc.* 183:1245-1247.
- Leopold, A. S. 1943. The molts of young wild and domestic turkeys. *Condor* 45:133-145.
- Lewis, J. B. 1987. Success story: wild turkey. Pages 31-43 in H. Kallman, C. P. Agee, W. R. Goforth, and J. P. Linduska, eds. *Restoring America's wildlife*. U.S. Dep. Int., Fish and Wildl. Serv., Washington, D.C.
- Lindzey, J. S. 1967. Highlights of management. Pages 245-259 in O. H. Hewitt, ed., *The wild turkey and its management*. The Wildl. Soc., Washington, D.C.
- Mosby, H. S. and C. O. Handley. 1943. *The wild turkey in Virginia: Its status, life history and management*. Va. Comm. Game Inland Fish., Richmond. 281pp.
- Nettles, V. F. 1984. Report of the Fish and Wildlife Health Committee. *Proc. Internatl. Assoc. Fish and Wildl. Agencies* 74:89-98.
- and E. T. Thorne. 1982. Annual report of the wildlife Diseases Committee. *Proc. Annu. Conf. U.S. Animal Health Assoc.* 86:64-65.
- Powell, J. A. 1965. *The Florida wild turkey*. Fla. Game Fresh Water Fish Comm., Tech. Bul. 8, Tallahassee. 27pp.
- Prestwood, A. K. 1968. Parasitism among wild turkeys (*Meleagris gallopavo silvestris*) of the Mississippi delta. Ph.D. Diss., Univ. Ga., Athens. 68pp.
- Pritchard, M. H. and G. O. W. Kruse. 1982. *The collection and preservation of animal parasites*. Univ. Neb. Press, Lincoln. 122pp.
- Roberts, H. A. 1954. Investigations of the frequency and kinds of diseases and parasites found among wild turkeys in Pennsylvania. Pa. Game Comm., Qrtly. Prog. Rep., Fed. Aid Proj. W-46-R, Harrisburg. pp. 24-27.
- Ruff, M. D. 1984. Nematodes and acanthocephalans. Pages 614-648 in M. S. Hofstad, H. J. Barnes, B. W. Calnek, W. M. Reid, and H. W. Yoder, eds., *Diseases of poultry*, 8th ed. Iowa State Univ. Press, Ames.
- , L. F. Schorr, W. R. Davidson, and V. F. Nettles. 1988. Coccidiosis in pen-raised wild turkeys. *J. Wildl. Diseases* 24:711-714.
- Snyder, R. L. 1952. Ability of spring release wild turkeys to survive and adapt to a natural environment. Pa. Game Comm., Qrtly. Prog. Rep., Fed. Aid Proj. W-46-R, Harrisburg. pp. 19-25.
- Tripathy, D. N. and L. E. Hanson. 1975. Immunity to fowl pox. *Am. J. Vet. Res.* 36:541-544.
- WDA. 1985. Wildlife Disease Association advisory statement on disease monitoring in wild turkeys. *Wildl. Disease Newsletter (Suppl., J. Wildl. Diseases)*. 21(2):1-3.
- Williams, J. E., E. T. Mallinson, and G. H. Snoeyenbos. 1980. Salmonellosis and arizonosis. Pages 1-8 in B. Hitchner, C. H. Domermuth, H. G. Purchase, and J. E. Williams, eds., *Isolation and identification of avian pathogens*, 2nd ed. Am. Assoc. Avian Pathol., College Station, Texas.

- Winterfield, R. W. 1980. Infectious bursal disease. Pages 79–80 in B. Hitchner, C. H. Domermuth, H. G. Purchase, and J. E. Williams, eds., Isolation and identification of avian pathogens, 2nd ed. Am. Assoc. Avian Pathol., College Station, Texas.
- Wunz, G. 1971. Evaluation of game-farm and wild trapped turkeys in Pennsylvania. Pa. Game News 1:20–44.
- Yoder, H. W., Jr. 1978. *Mycoplasma gallisepticum* infection. Pages 236–250 in M. S. Hofstad, H. J. Barnes, B. W. Calnek, W. M. Reid, and H. W. Yoder, eds., Diseases of poultry, 7th ed. Iowa State Univ. Press, Ames.
- . 1980. Mycoplasmosis. Pages 40–42 in B. Hitchner, C. H. Domermuth, H. G. Purchase, and J. E. Williams, eds., Isolation and identification of avian pathogens, 2nd ed. Am. Assoc. Avian Pathol., College Station, Texas.