



FEATHER DAMAGE DUE TO MYCOTIC INFECTIONS IN WILD TURKEYS

Authors: Davidson, William R., Shotts, Emmett B., Teska, Jeff, and Moreland, David W.

Source: Journal of Wildlife Diseases, 25(4) : 534-539

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-25.4.534>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

FEATHER DAMAGE DUE TO MYCOTIC INFECTIONS IN WILD TURKEYS

William R. Davidson,¹ Emmett B. Shotts,² Jeff Teska,² and David W. Moreland³

¹ Southeastern Cooperative Wildlife Disease Study, Department of Parasitology,
College of Veterinary Medicine and School of Forest Resources,
The University of Georgia, Athens, Georgia 30602, USA

² Department of Medical Microbiology, College of Veterinary Medicine,
The University of Georgia, Athens, Georgia 30602, USA

³ Louisiana Department of Wildlife and Fisheries, P.O. Box 15570,
Baton Rouge, Louisiana 70895, USA

ABSTRACT: Wild turkeys (*Meleagris gallopavo*) from Pearl River Wildlife Management Area, St. Tammany Parish and from adjacent St. Helena Parish, Louisiana (USA) were observed to have broken and frayed rectrices. The condition was noted in 21% of 90 wild turkeys harvested by hunters during the springs of 1985 through 1988 from the Pearl River Wildlife Management Area. Damage to feathers ranged from mild to severe. Histologic and microbiologic study of five birds disclosed colonization and invasion of the rachis sheath and pulp by fungi of the genera *Aspergillus*, *Curvularia*, *Cladosporium*, *Dactylella*, *Exophiala*, *Helminthosporium* and *Trichophyton* and by *Streptomyces*. Sterilized normal rectrices from wild turkeys were inoculated with these organisms and subsequently developed damage that was histologically compatible with field cases. The condition was diagnosed as a multiple etiology mycosis. Successful colonization and invasion of experimentally inoculated feathers required addition of moisture and elevation of relative humidity within the cultures. The apparent high moisture requirements of the fungi suggest that late winter and early spring flooding may be a probable predisposing factor for this condition.

Key words: Wild turkey, *Meleagris gallopavo*, mycosis, *Curvularia* sp., *Dactylella* sp., *Exophiala* sp., *Helminthosporium* sp., *Trichophyton* sp., feather damage.

INTRODUCTION

During the spring turkey hunting season of 1985, personnel with the Louisiana Department of Wildlife and Fisheries (LDWF; Baton Rouge, Louisiana 70895, USA) noted a single wild turkey (*Meleagris gallopavo*) with frayed and broken tail feather (rectrices) among approximately 20 turkeys harvested on the Pearl River Wildlife Management Area (WMA; St. Tammany Parish, Louisiana, USA). From 1986 through 1988, 18 additional turkeys harvested on Pearl River WMA had similarly damaged tail feathers. In 1987, one of an unknown number of turkeys harvested from adjacent St. Helena Parish also had damaged tail feathers. This report describes the findings of an investigation on the etiology of this condition.

MATERIALS AND METHODS

In 1986, a single, air-dried tail fan from one of the turkeys was submitted for examination to the Southeastern Cooperative Wildlife Disease Study (SCWDS; College of Veterinary

Medicine, The University of Georgia, Athens, Georgia 30602, USA). In 1987, refrigerated or frozen tail fans, the rest of the skin, blood or viscera from four turkeys harvested by hunters were submitted for examination. Three of these turkeys were from Pearl River WMA (approximately 30°16'N, 89°45'W), and one was from St. Helena Parish. In 1987, a moribund turkey with damaged tail feathers was caught by hand on Pearl River WMA, and the refrigerated intact carcass along with blood and tracheal and cloacal swabs were submitted.

Tail feathers were examined grossly and with aid of magnification (7 to 42×), and the extent and severity of damage were recorded. Pieces of feather shafts were preserved in 10% neutral-buffered formalin solution, embedded in paraffin, sectioned, and stained with hematoxylin and eosin (H&E) and periodic acid-Schiff (PAS) stains. Direct scrapings from the surface of feather sheaths and from damaged and intact feather pulp were cleared with lactophenol-cotton blue and examined microscopically.

Tail feathers from affected turkeys were used for fungal and bacterial isolation. One to 2 cm pieces of the feather shafts were placed on the center of Sabouraud Dextrose Agar plates (GIBCO Laboratories, Madison, Wisconsin 53711, USA) and on Blood Agar Base (BBL Microbi-

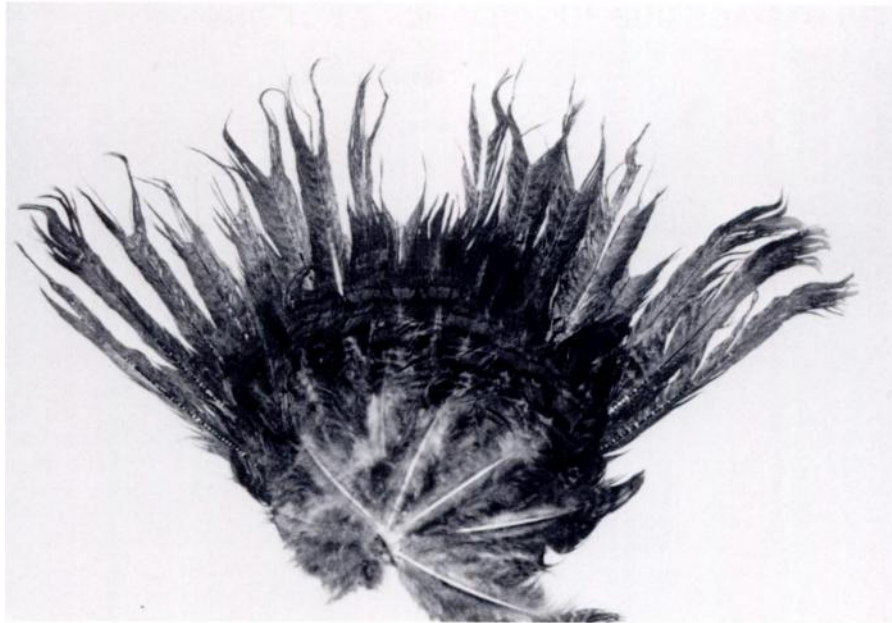


FIGURE 1. Tail feathers from a wild turkey with severe damage due to mycotic infections.

ology Systems, Cockeysville, Maryland 21030, USA) supplemented with 5% bovine blood. All plates were incubated at 25, 30 and 37 C. Isolated bacterial colonies were subcultured and identified by standard methods. Fungal isolates from artificial media were inoculated individually onto the ventral groove of approximately 8 cm sections of sterilized (autoclaved at 121 C for 15 min) normal wild turkey rectrices. These feather sections were maintained in dry glass petri dishes and observed daily for evidence of fungal growth. Following 7 days of incubation at room temperature (~25 C) under conditions of minimal moisture (relative humidity (RH) ~70%), a piece of sterile filter paper moistened with sterile distilled water was added to each dish (RH ~100%). After addition of the moistened filter paper, cultures were incubated for an additional 3 to 4 wk. Sections of uninoculated feathers maintained in separate dishes but under the same conditions served as controls. Sections of artificially inoculated and control feathers were processed for histologic examination as described above.

All available major visceral organs were processed for histopathologic examination as described above and stained with H&E. Since the moribund condition of one turkey from Pearl River WMA suggested disease processes in addition to feather damage, a detailed necropsy with radiographic, histologic, microbiologic and parasitologic examinations was conducted to determine the cause of its condition.

RESULTS

Rectrices from all five cases had similar feather damage, although the extent of the damage ranged from only a few to all (Fig. 1) rectrices. Typically, the rachis was broken 5 to 15 cm from the distal end (Fig. 2), and the ventral surface of the rachis was split longitudinally for a distance of 1 to 5 cm proximal to the break (Fig. 3). The feather pulp in the damaged region was fragmented with a tan or gray discoloration. Loss of fragmented feather pulp was common, and in some feathers only a hollow, split rachis remained. Feathers in which the distal ends were not missing often were bent 5 to 10 cm from the distal end. Inspection of bent feathers disclosed small longitudinal splits along the ventrum of the rachis, often accompanied by swelling of the pulp within the rachis. In some instances, small (0.5 to 1 mm) holes through the ventral rachis sheath were present and small sections of pulp were missing.

The ventral median groove of the rachis on all damaged rectrices, and usually on all rectrices of affected tail fans, was cov-

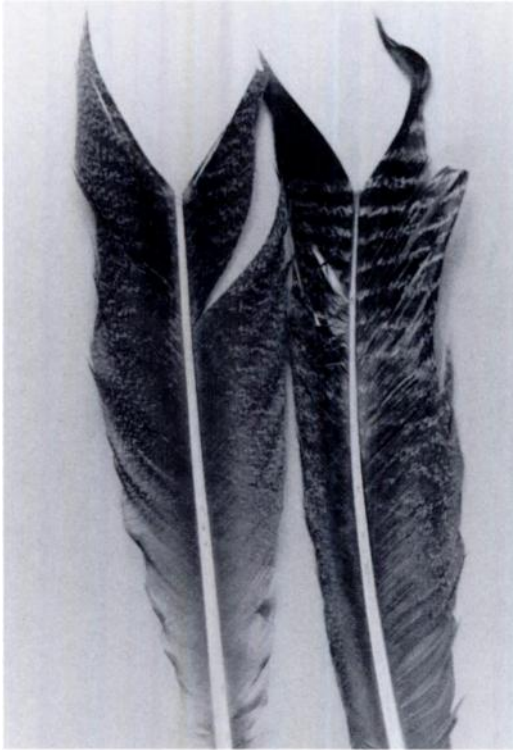


FIGURE 2. Rectrices from a wild turkey with breakage and loss of distal portion of feathers due to mycotic infections.

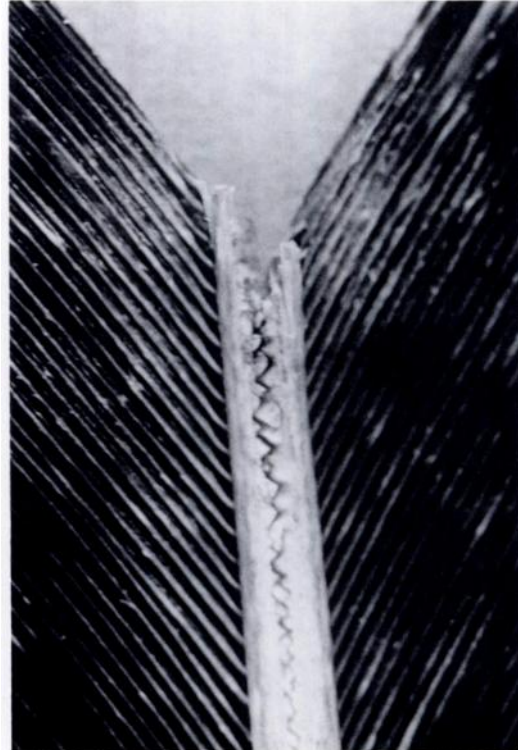


FIGURE 3. Splitting of the sheath proximal to a break in the rachis of a wild turkey rectrix damaged by mycotic infections.

ered with varying amounts of bluish, greenish, gray, black and/or white mold-like debris (Fig. 4). Fresh preparations of this debris disclosed a mat of fungal hyphae. Hyphae were also common in fresh preparations of damaged feather pulp. Long, filamentous, branching hyphae predominated in feather pulp, and branching, beaded or chain-like hyphae with occasional conidia predominated on the surface debris. A mixture of hyphal types and sizes occurred most often. Colonization by fungi and damage to the feathers were generally limited to the distal two-thirds of the rachis and never involved the feather follicles or skin.

Histologic examination of feathers also revealed fungal hyphae on the rachis surface and in the feather pulp (Fig. 5). Hyphae penetrated the sheath of the rachis, disrupting and separating the middle mal-

pighian layer. Hyphae extending into the feather pulp grew in the intercellular matrix and only rarely occurred in the pulp cell chambers. Colonization by fungi was concentrated along the ventral groove of the rachis, and subsequent invasion and damage consistently occurred along the lateral margins of the ventral groove. A second common site of heavy colonization was the surface of the rachis at the point of attachment of the feather barbs; however, invasion was less pronounced at this location. Splits in the sheath were not present at the point of barb attachment. In heavily damaged feathers, invasion and disruption of the lateral portions of the rachis sheath also were noted, but in no instance was the dorsum of the feather affected.

Seven genera of fungi and one actinomycete were isolated on artificial media

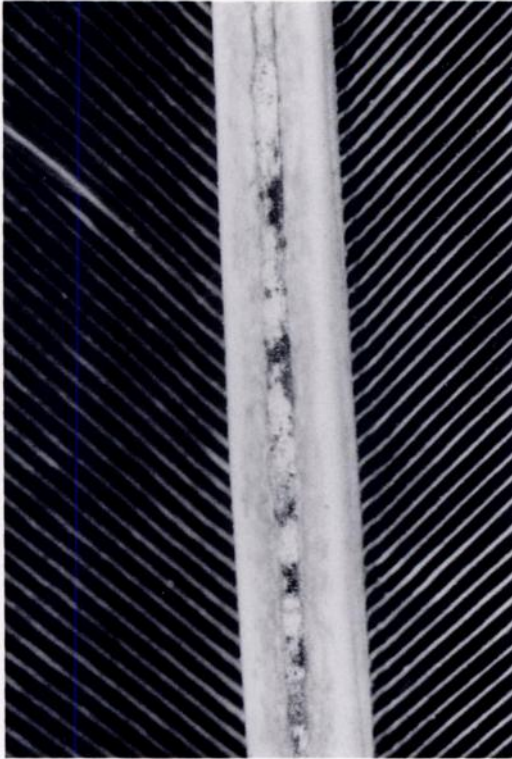


FIGURE 4. Fungal colonies along the ventral groove of a wild turkey rectrix.

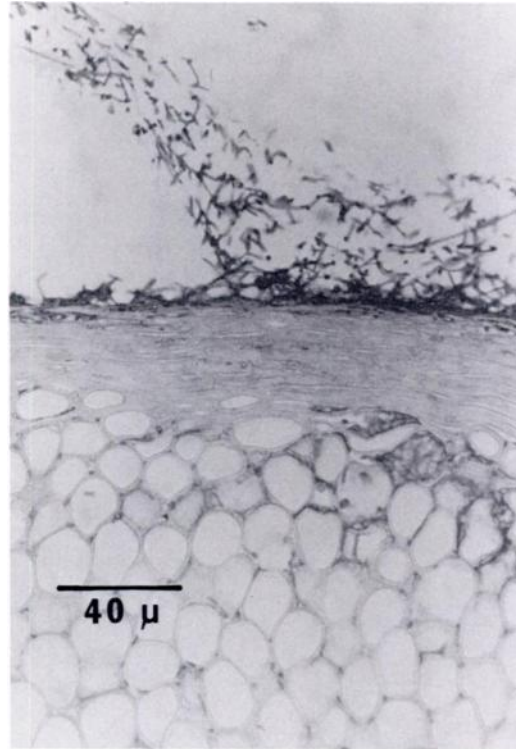


FIGURE 5. Mat of fungal hyphae on the sheath surface with extension of hyphae into the pulp cavity of a wild turkey rectrix. H&E.

from damaged feathers, including species of *Aspergillus*, *Cladosporium*, *Curvularia*, *Dactylella*, *Exophiala*, *Helminthosporium*, *Trichophyton* and *Streptomyces*. When inoculated on sterilized, dry sections of normal turkey tail feathers, none of these isolates produced visible growth within 5 days. Following the addition of moistened filter paper, however, all isolates grew profusely on feather sections.

All eight genera colonized the surface of inoculated feather sections, and all exhibited some degree of invasion of the keratinized rachis sheath. Five fungal isolates (*Curvularia* sp., *Dactylella* sp., *Exophiala* sp., *Helminthosporium* sp., and *Trichophyton* sp.) exhibited greater ability to invade and disrupt the rachis sheath. Disruption of the rachis sheath and pulp in inoculated feather sections was histologically similar to that occurring in field cases

(Fig. 6). In no instance, however, did inoculated feather sections develop visible splitting or holes in the feather sheath. None of the control feather sections exhibited any fungal growth.

Bacteriologic cultures of feathers produced isolates of *Pseudomonas aeruginosa*, *P. fluorescens*, *P. maltophilia*, *P. paucimobilis*, *Bacillus* spp., *Staphylococcus aureus*, *S. sciuri*, and *Enterobacter aerogenes*. Sections of visceral organs did not contain significant lesions except for the moribund turkey caught by hand on Pearl River WMA in 1987. Diagnostic evaluation of this case disclosed severe multifocal ulcerative colitis and extensive myopathy. *Clostridium perfringens* was isolated from intestinal lesions and visceral organs, and large gram-positive bacterial rods were demonstrated histologically in the centers of intestinal ulcerations. The

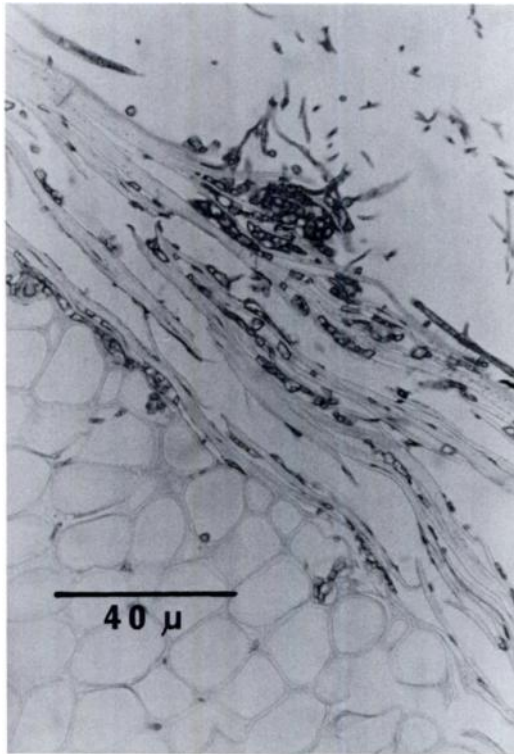


FIGURE 6. Disruption and separation of the malpighian layer of a wild turkey rectrix experimentally inoculated with fungi. H&E.

primary diagnosis in this turkey was ulcerative enteritis due to *C. perfringens*, and the bird's moribund condition was not considered to be related to feather damage.

DISCUSSION

Histologic examination of feathers from field cases strongly suggested a mycotic etiology for the condition. This was further supported by experimental inoculation of normal feathers, which led to colonization and invasion of the feathers by fungi isolated from field cases. Based on the similarity of natural and experimental cases, we conclude that the condition was of mycotic origin. The role of bacteria in damage to feathers in field cases is not clear; however, under experimental conditions, the various fungi alone produced microscopic damage identical to that in field cases.

Which specific fungi were responsible for the condition of the feathers from field cases is unclear, since all species of fungi inoculated on normal feathers exhibited some degree of invasiveness. Based on their apparently greater ability to invade and damage feathers inoculated experimentally, we believe that *Curvularia* sp., *Dactylella* sp., *Exophiala* sp., *Helminthosporium* sp., and/or *Trichophyton* sp. may be more important than the other species in development of the condition. At present, however, the condition probably is best categorized as a multiple etiology mycosis.

A large number of mycotic organisms have been isolated from birds (Pugh, 1964, 1965; Rees, 1967a, b; Hubalek, 1976, 1978; Tudor, 1983), including many species of keratinophilic or keratinolytic fungi from the feathers (Pugh, 1965; Rees, 1967a, b). The reports of these fungi from the feathers of birds do not, however, generally mention invasion of or damage to the feathers. Exceptions are the notations by Pugh (1964, 1965) that *Arthroderma curreyi* might "be implicated in the rather ragged appearance of blackbirds in early summer," a report by Sartory (1942) that *Aspergillus fumisalordes* variety *roseus* damaged feathers in pigeons, and a report by Tudor (1983) that *Paecilomyces* sp., *A. phoenicis*, *A. candidus*, *Mucor circinelloides*, *Rhizopus arrhizus*, *Penicillium chrysogenum* and *P. cyclopium* were associated with feather-pulling in captive pigeons and psittacine birds.

Some of the genera of fungi which we isolated are known to produce various cutaneous infections in mammals and other animals; however, we were unable to locate reports that any of these genera, except for members of the genus *Aspergillus* (Tudor, 1983), specifically damaged only the feathers of living birds. The genera *Aspergillus*, *Trichophyton* and *Cladosporium*, however, are among the more common genera of keratinophilic fungi associated with birds (Pugh, 1965; Hubalek, 1978; Tudor, 1983).

We believe that environmental factors

on Pearl River WMA may have been an important predisposing factor in development of this condition. Pearl River WMA is located along the flood plain of the Pearl River, and the area often floods during late winter and early spring. In 1985, 1986 and 1987, there was extensive flooding immediately prior to the spring turkey hunting seasons, which were from mid-March through most of April. In 1987, the water did not recede until about 1 wk after the hunting season opened. During two of these three years, the prevalence of damaged tail feathers among harvested wild turkeys was high (nine of 24 or 37% in 1986 and seven of 19 or 37% in 1987). In contrast, during the drier spring of 1988 there was no flooding, and only two of 27 (7%) turkeys with slightly damaged tail feathers were noted. The low prevalence of feather damage recorded for 1985 (1 of approximately 20) was due, at least in part, to only casual observations prior to detection of the single severely affected turkey, which was noted after the hunting season was well in progress. One of us (DWM) has observed turkeys wading in water during these flooding episodes. The downward position of the tail could result in it becoming wet, either during wading or by frequent contact with wet vegetation, while the remainder of the feathers stay drier. This may enhance fungal growth and invasion only of the tail feathers. This concept also is supported by the fact that there was no visible fungus growth on experimentally inoculated feathers until available moisture and relative humidity were increased in the culture chambers.

The absence of holes and splits in the sheath or breakage of the rachis in the experimentally inoculated feathers may have been related to the short duration of

the experimental trial or, more likely, to the lack of physical forces acting upon the feathers. Experimentally inoculated feathers became more pliable and tended to bend or crease easily. We suggest that the physical forces exerted on the rectrices of live turkeys, such as during flight, intraspecific aggressive behavior, or courtship displays, are important in the damage process. Bending of the rachis at specific points weakened by fungal invasion could lead to the development of holes, splitting, and eventually breakage of the feather. The consistent pattern of breakage approximately two-thirds of the distance along the length of the feather also suggests that a physical factor, such as wind resistance during flight, is being applied across all rectrices.

LITERATURE CITED

- HUBALEK, Z. 1976. Interspecific affinity among keratinolytic fungi associated with birds. *Folia Parasitologica* 23: 267-272.
- . 1978. Coincidence of fungal species associated with birds. *Ecology* 59: 438-442.
- PUGH, G. J. F. 1964. Dispersal of *Arthroderma curreyi* by birds, and its role in the soil. *Sabouraudia* 3: 275-278.
- . 1965. Cellulolytic and keratinophilic fungi recorded on birds. *Sabouraudia* 4: 85-91.
- REES, R. G. 1967a. Keratinophilic fungi from Queensland. II. Isolations from feathers of wild birds. *Sabouraudia* 6: 14-18.
- . 1967b. Keratinophilic fungi from Queensland. III. Isolations from feathers of domestic fowls. *Sabouraudia* 6: 19-28.
- SARTORY, A. 1942. *Aspergillus fumigatus* var. *roseus* n. sp. pathogenic to pigeon feathers. *Academie des Sciences Colon* 214: 564-565.
- TUDOR, D. C. 1983. Mycotic infection of feathers as the cause of feather-pulling in pigeons and psittacine birds. *Veterinary Medicine/Small Animal Clinician* 78: 249-253.

Received for publication 12 December 1988.