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PREVALENCE OF *Aspergillus fumigatus* IN FREE-LIVING GOSHAWKS *(Accipiter gentilis atricapillus)*

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Abstract: During the fall migration of 1972 and 1973 unusually large numbers of goshawks *(Accipiter gentilis atricapillus)* were counted at Hawk Ridge in Duluth, Minnesota. These birds were sampled for prevalence of fungi of the genus *Aspergillus*. Fungi of this genus were recovered from 26 of 49 birds (53%) in 1972 and 4 of 45 (7%) birds in 1973. Aspergillosis was confirmed at necropsy in three wild goshawks in 1972, but none in 1973. The disease was further confirmed at necropsy in 8 of 12 (67%) goshawks trapped in the fall and retained for falconry in 1972 and in 2 of 17 (12%) such birds in 1973. We suggest that the stress of intraspecific agonistic behavior in conjunction with a high density of goshawks and greatly reduced prey base may increase the susceptibility of these hawks to aspergillosis.

INTRODUCTION

Pulmonary aspergillosis has been recognized as a disease of avian species since early 1800.[19] The disease is caused by species of fungi in the genus *Aspergillus*. *A. fumigatus* appears to be the most pathogenic species and is most frequently isolated from clinical cases.[5] Aspergillosis commonly is reported in both wild and domestic[2] captive birds, in wild birds recently captured,[8] and among free-living birds of the anseriform, larid, gallinaceous, and passeriform groups[16,19,21] (see Beer[4] for a detailed review). Zinkl[29] reported a large outbreak that killed about 1500 crows *(Corvus brachyrhynchos)* in Nebraska, and Adrian *et al.*[1] reported an epornitic in mallards *(Anas platyrhynchos)* in which 387 birds were found dead.

References to the occurrence of aspergillosis in free-living raptors are few. The species in which the disease has been reported include the barn owl *(Tyto alba)*,[19] golden eagle *(Aquila chrysaetos)*,[28] sparrow hawk *(Accipiter nisus)*,[14] martial eagle *(Polemaetus bellicosus)*,[7] tawny owl *(Strix aluco)*,[15] and bald eagle *(Haliaeetus leucocephalus)*.[6]

Acute and chronic forms of aspergillosis, each having a unique pathogenesis, are reported in the literature. The acute form arises when birds are exposed to an overwhelming dose of spores from a point source that produces miliary granulomas throughout the lungs and air sacs.[1,10] The clinical course is only a few days long, birds die in good flesh, and there is usually no evidence of other concurrent unusual circumstance such as disease, injury or capture. The chronic form is manifested by large granulomas in the lungs, free growing, sporulating mold in the air sacs and airways of the lung, and emaciation. The clinical course may extend over 3 weeks and the disease often accompanies

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capture, injury, or other stressful factors.[2,6,27,29] Profound immunosuppression appears to play a major role in the pathogenesis of the chronic form[2,26] thus allowing the low level background levels of this ubiquitous organism to become pathogenic.

The objectives of this paper are to report the recovery of *Aspergillus* sp. from free-living goshawks and to document *Aspergillus*-associated mortality found in these raptors during migration.

MATERIALS AND METHODS

The prevalence of *Aspergillus* spp. in free-living goshawks was determined by sampling trapped goshawks as they moved through the Duluth, Minnesota region in the fall of 1972 and 1973. The areas now included in the Hawk Ridge Nature Reserve in Duluth are an historic concentration area for migrating raptors.[11] This concentration allows a large sample size to be obtained in a short period of time and the annual hawk counts conducted at Hawk Ridge provide a data base for comparing the sample size to an estimate of the total number of goshawks passing over the area. Exceptionally large flights of goshawks occurred over Hawk Ridge in the fall of 1972 (5,152) and 1973 (3,517).[23,24]

Culturettes[2] were used to swab the inside of the trachea and Sabouraud's[3] agar was used as the culture media. Plates were streaked in the laboratory within 48 h of sampling and then incubated for 48 h at 37 C. *Aspergillus* spp. were microscopically examined and identified to genus and species using keys by Raper and Fennel.[20] The goshawks were banded and released.

The occurrence of clinical disease in the population sampled as opposed to simple recovery of the organism was partially confirmed by data collected from goshawks found disabled in the wild or trapped for falconry and subsequently submitted to a raptor research and rehabilitation program at the University of Minnesota.[9]

RESULTS

There was a significant difference between the number of positive cultures in 1972 (53%) and number of positive cultures in 1973 (7%) (see Table 1). *A. fumigatus* was present in all positive cultures, while *A. terreus* and *A. niger* were found in mixed culture with *A. fumigatus* in 4 plates in 1972 and 3 plates in 1973.

Three case histories confirmed the occurrence of aspergillosis in free-living goshawks in 1972:

1. On 22 October 1972, an adult female goshawk was submitted with a gunshot wound received the previous day. She died that evening and aspergillosis was confirmed at necropsy.
2. On 26 October 1972, an adult female goshawk was presented with multiple injuries from a gunshot wound received the previous day. On 27 October she showed signs of respiratory difficulty. Intravenous therapy with Amphotericin B[4] was ineffective and the bird died on 29 October. Chronic aspergillosis was confirmed by gross and microscopic examination at necropsy.
3. An adult female goshawk was found perched close to the ground during the last week of October, 1972, and although not injured, she was unable to fly and died 6 days later. Chronic aspergillosis was confirmed at necropsy.

No similar cases were received during the fall of 1973.

[2] Culturette® Scientific Products, 1430 Waukegan Rd., McGaw Park, Illinois 60085, USA.

[3] Sabouraud's agar, Difco Laboratories, Detroit, Michigan 48232, USA.

[4] Fungizone, E. R. Squibb Co., Princeton, New Jersey 08540, USA.

TABLE 1. The Recovery Rate of *Aspergillus* sp. in Goshawks trapped at Duluth, Minnesota, Fall, 1972, 1973.

Age Class	Total No. and (%) Trapped in Each Age Class for the Season	Total No. and (%) Sampled	No. of Samples and (%) of sample Positive
Hatching Year	59 (15.1)	9 (18.4)	3 (33)
Second Year	165 (42.2)	22 (44.9)	13 (59)
1972			
After Second Year	167 (42.7)	18 (36.7)	10 (55)
Total	391 (100)	49 (100)	26 (53)
Hatching Year	41 (6.3)	5 (8.9)	0
Second Year	37 (5.7)	11 (19.6)	2 (18)
1973			
After Second Year	572 (88.0)	40 (71.4)	2 (5)
Total	650 (100)	56 (100)	4 (7)

Aspergillosis was not encountered in goshawks during the winter of 1972-73. An adult goshawk was captured at the Cedar Creek Natural History Area and its movements monitored by automatic radio-telemetry from 22 February 1973 to 16 March 1973. The bird was recaptured and examined; no clinical signs of aspergillosis were evident. The goshawk weighed only 12 g less than at the time of first capture. Another adult goshawk was captured on 14 March 1973, and held for 13 days for treatment of an injured wing. No clinical signs of disease were observed while the bird was in captivity. It was released and tracked by radio-telemetry for seven days before moving out of tracking range. The fall and winter of 1973-74 yielded no further encounters with aspergillosis in free-living goshawks.

Several goshawks captured for falconry purposes in Minnesota were submitted for examination or treatment in 1972 and 1973. The prevalence of aspergillosis-related mortality occurring within six weeks of capture was notably different in the two years. In 1972, twelve goshawks were presented for examination. Eight died and all eight were diagnosed as cases of aspergillosis at necropsy. In 1973, seventeen goshawks were examined. Four died within a few weeks of capture and in only two cases was aspergillosis confirmed. *A. fumigatus* was identified in all 10 cases.

In the three wild goshawks and the 10 captive goshawks examined the gross and microscopic lesions were consistent with chronic aspergillosis. Furthermore, the clinical course of the disease among the 10 captive birds was characterized by

severe emaciation and pronounced respiratory distress.

DISCUSSION

We suggest that the prevalence of *A. fumigatus* and related fungi in 53% of the invading goshawks captured, the detection of aspergillosis in 3 dead wild birds, and aspergillosis-associated mortality in 67% of the recently captured goshawks examined in 1972, compared to the 1973 results of a 7% recovery in migrating birds, no wild birds found dead or dying of aspergillosis and only 2 of 17 recently captured goshawks dying of aspergillosis, provides direct evidence for an epizootic of aspergillosis in goshawks in 1972.

Beer[4] reported the results of a similar survey among various species of wildfowl. The recovery rate ranged from 7% to 15% over a 3 year period in which the oral pharynx of 1188 pink-footed geese was sampled. He observed no clinical indications of disease in any of the birds he sampled. Our intratracheal recovery rate of 7% in 1973, in which sample there was very little evidence of clinical aspergillosis even among those goshawks subjected to the rigors of handling for falconry purposes, compares favorably with the background level reported by Beer[4] among the vegetation-feeding geese. Thus the 53% of recovery rate among wild goshawks in 1972 and the mortality rate of 67% among birds retained for falconry, for whom the stresses of capture and handling were essentially similar to the goshawks retained in 1973, becomes highly significant. Simply stated, if the goshawk has clinical aspergillosis the growth of the mold in the airways greatly increases the chances of recovering spores from the trachea. Our opinion, based on the high rate of recovery of *A. fumigatus*, is that a large number of goshawks migrating in 1972 had clinical aspergillosis.

In all goshawks upon which postmortem examination was conducted, the lesions were characteristic of chronic aspergillosis. Clues to the stresses that led to the development of aspergillosis in 1972 and to the difference in recovery rate of *Aspergillus* spp. during the two years of the study are found in the circumstances surrounding the periodic invasions of goshawks.

Goshawk invasions appear to coincide with and may be caused by reduced numbers of prey (especially snowshoe hare and ruffed grouse) in the breeding range of the hawks. Prey levels fluctuate from high to low over a ten year period.[12,13,23] Trapping results for both years show a greater proportion of "second year" and "after second year" (adults) goshawks in the invading population than would be expected if the age structure was representative of a normally producing population in which a majority of first year birds would be predicted.[19,20]

Since all animals, including goshawks, are continually exposed to the ubiquitous *Aspergillus* spp. spores,[2] we believe the stress of agonistic interactions, reduced prey availability, and migration may have increased the susceptibility of the birds to infection. Malnutrition has been a suspected contributing factor in outbreaks of aspergillosis in wild birds[25] and von Faber[26] has reviewed a wide variety of stressors such as muscular fatigue, cold, heat, restraint, crowding, and social rank that are capable of rapidly altering the normal physiologic state of a bird and potentially compromising its defense mechanisms.

Not all of the invading goshawks would be likely to succumb. Those birds with greater genetic resistance to aspergillosis,[3] greater tolerance of social strife, and greater ability to capture prey would survive the fall and winter and return to the breeding range.[18] These migration survivors and the goshawks which remained on the breeding range formed the majority of the 1973 breeding

population. The continued lack of prey[22] apparently resulted in very low nest success which was reflected in the small proportion of juveniles in the 1973 fall migration and a generally smaller invasion than in 1972.[17,24]

Eighty-eight percent of the goshawks trapped in 1973, and about 75% of those trapped by Mueller *et al.*[19] were "after second year" birds. These birds had already survived the social stress brought about by the low prey of the previous year. Some had probably migrated. We suggest these "proven" individuals were more resistant to the rigors which stressed the 1972 birds and caused increased susceptibility to aspergillosis. Thus the incidence of the disease was lower in the 1973 migrants which was reflected by a lower aspergillosis-associated mortality in recently captured birds and by a lower recovery rate of the organism from the sampled population of goshawks.

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