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MORTALITY IN FLEDGLING GREAT HORNED OWLS FROM BLACK FLY HEMATOPHAGA AND LEUCOCYTOZOONOSIS

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ABSTRACT: Black fly feeding alone and in concert with *Leucocytozoon* spp. infection caused mortality in fledgling great horned owls (*Bubo virginianus*) in the Yukon, Canada 1990 to 1991. These mortalities occurred during a year of food shortage corresponding with a decline in the population of snowshoe hare (*Lepus americanus*), the main prey for great horned owls. We hypothesize an interaction between food availability and the consequences of host-parasite interactions.

Key words: *Bubo virginianus*, anemia, host-parasite interactions, tethering platforms.

INTRODUCTION

Rohner (1994) evaluated the numerical response of great horned owls (*Bubo virginianus*) to the snowshoe hare (*Lepus americanus*) cycle from 1988 to 1993 in the boreal forest of southwestern Yukon, Canada. Populations of snowshoe hare in boreal Canada and Alaska (USA) follow a well documented 8 to 11 year cycle (Krebs et al., 1992). Predators such as the great horned owl which use the snowshoe hare as a primary food source, follow a similar population cycle with a time lag of 1 to 2 years (Rohner, 1995). Snowshoe hare densities in the study area peaked in 1989 to 1990 and began to decline in 1991 (Rohner, 1996). The survival of juvenile owls was very high during 1989 and 1990 but decreased in 1991, the first year of the snowshoe hare population decline (Rohner and Hunter, 1996). Extensive monitoring of nest sites combined with tracking of individuals by radio-telemetry provided us with carcasses of 39 juvenile owls found dead during 1989 to 1991. We observed a variety of causes of death in these carcasses, including trauma and bacterial infections, but the most interesting findings were lesions attributed to feeding by ornithophilic black flies (Diptera: Simuliidae) and in several birds concurrent infections with the blood parasite *Leucocytozoon* spp. (Rohner and Hunter, 1996). The prevalence of parasitism and predation as causes of mortality increased as hare densities decreased (Rohner and Hunter, 1996).

The pathogenicity of ornithophilic black flies is poorly documented. Raptor biologists often observe nestlings tormented by black flies but we were unable to find published reports of clinical consequences or nestling mortality. S. Cain (pers. comm.) observed mortality in red-tailed hawk chicks (*Buteo jamaicensis*) in northern Wyoming (USA) which he attributed to intense black fly feeding causing anemia and occasionally driving chicks from their nests prematurely.

The blood parasite *Leucocytozoon* spp. can be a serious pathogen in immunologically naive captive or domestic waterfowl, turkeys, and chickens which have been introduced into areas enzootic for the parasite (Bennett et al., 1993) but, with the exception of epizootics in juvenile Canada geese (*Branta canadensis*) at the Seney Wildlife Refuge in northern Michigan (USA) (Herman et al., 1975), infection of wild birds with *Leucocytozoon* spp. is rarely associated with clinical disease or mortality. There are many reports of *Leucocytozoon* spp. being found in blood smears from raptorial birds (Peirce, 1981) but documentation of pathogenicity is lacking.

In this paper we describes the necropsy findings from 28 of the juvenile great horned owl carcasses obtained during the

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great horned owl/snowshoe hare study (Rohner and Hunter, 1996) and discusses the role of ornithophilic black flies and *Leucocytozoon* spp. in causing mortality in juvenile owls during the fledging and post-fledging period.

MATERIALS AND METHODS

The study area encompassed 350km^2 of glacial valley near Kluane Lake in the southwestern Yukon, Canada (60°57'N, 138°12'W). Great horned owl nests were located either by triangulating hooting pairs (Rohner and Doyle, 1992) or monitoring females with radio-transmitters. Between 1989 and 1991, 116 nestlings were banded and monitored from 1 wk of age until fledging. As part of a feeding and behavioral study 86 owlets were transferred to tethering platforms at a height of 3.5 m above the ground (Rohner, 1994). Fifty-five owlets (13 naturally fledged, 42 released from the platforms) were equipped with radio transmitters. During the pre-fledging period the owlets were checked two to four times per week, and between fledging and dispersal (post-fledging) the birds were radio-monitored at least once a week.

During 1990 and 1991, 28 juvenile great horned owl carcasses were located. These were placed in individual plastic bags, labelled (band number, location, age, approximate time of death), frozen and shipped to the Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada. Each bird was necropsied and, if carcass preservation warranted, selected tissues (lung, liver, spleen, kidney, and heart; in some birds sections of skin and eyelid, brain, and digestive tract) were fixed in 10% buffered formalin, embedded in paraffin, sectioned at 6 μ and stained with hematoxylin and eosin for light microscopic examination.

Specimens of black flies were carefully removed from these owls from areas where lesions were most numerous such as eyelids and auricular openings. The condition of individual flies varied according to the state of preservation of the host owls. Most were in an advanced state of decay and badly fragmented. Identifications were possible only for the most completely preserved individuals. Genitalia were cleared with lactic acid and examined at up to 130 under a stereomicroscope.

Identification of black flies was further hindered by a lack of recent keys. The only taxonomic treatments for northwestern North America are long out of date (Stone, 1952; Sommerman, 1953) and include fewer than half of the 76 species now known from Alaska and the Yukon Territory (Currie, 1997). Further, many northwestern species have not been formally described, or belong to unresolved sibling complexes; flies from these latter complexes require examination of the polytene chromosomes of the larvae for species-level identification. Adults were identified to the lowest taxonomic-level possible through comparisons with confirmed material in the Royal Ontario Museum (Toronto, Ontario, Canada) and the Canadian National Collection of Insects (Ottawa, Ontario, Canada). Voucher specimens were deposited in the entomological collection of the Royal Ontario Museum, with a voucher specimen number of ROM 979999.

RESULTS

A cause of death was established for 23 of the 28 carcasses. Five carcasses were too badly autolysed for a meaningful necropsy and three additional carcasses were partially scavenged. Histologic evaluation was carried out on tissues from 17 birds. The quality of the histologic sections was sometimes poor due to autolysis or freezing but tissues from many of the birds were surprisingly well preserved.

Nine owlets less than 10 wk of age were examined. Six birds died of anemia and severe dehydration and had extensive skin lesions attributed to black fly feeding. Four of these had histologic lesions of concurrent *Leucocytozoon* spp. infection. One bird had a localized cellulitis and septicemia related to an injury sustained on the tethering platform. No diagnosis was established for the remaining two birds.

Fourteen owlets were between 10 and 14 wk of age. Of these, seven birds were dehydrated, anemic and had severe skin lesions attributed to black fly feeding. Five birds had concurrent *Leucocytozoon* spp. infections. Four birds died from trauma and one died as a result of cloacal impaction. No diagnosis was established for the two remaining birds in this age group.

Five of the owlets were older than 14 wk of age. Four of these were killed on the highway and no diagnosis was reached on the last bird. Three of the four road-killed owls had megaloschizonts in liver

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and myocardium but no evidence of parasitemia.

The skin lesions related to black fly feeding were found mainly in unfeathered areas such as the eyelids, cere, corners of the beak, auricular openings, jugular groove and ventral surfaces of the patagium. The eyelids were edematous with multiple raised, erythematous areas surrounding small puncture sites. The adjacent feathers were caked with dried blood. Black flies often were found associated with the lesions or within the surrounding feathers. Sixty-five black flies were counted on one individual. Microscopically, the skin lesions were characterized by increased vascularization, localized edema, subcutaneous hemorrhage, considerable local tissue necrosis, inflammation, and thrombosis of adjacent capillaries.

Forty-five intact female black flies were recovered from owl carcasses. These represented just a small portion of the total number of black flies associated with the owls, but were selected on the basis of their complete state of preservation. The 45 intact females belong to four taxa as follows: *Helodon* (*Distosimulium*) *pleuralis* (Malloch), $n = 1$; *Helodon* (*Parahelodon*) *decemarticulatus* (Twinn), $n = 3$; *Simulium* (*Eusimulium*) *aureum* Fries complex, $n = 3$; and *Simulium* (*Eusimulium*) *canonicolum* (Dyar and Shannon) complex, $n = 38$.

Leucocytozoon spp. infections were characterized as mild or severe depending on numbers of circulating gametocytes observed within vessels and the numbers of schizonts or megaloschizonts observed in tissues. Birds with mild infections had a low level parasitemia based on observation of *Leucocytozoon* spp. gametocytes within vessels and occasional schizonts or megaloschizonts within tissues, usually the liver. Five owlets in the 10 to 14 wk of age group, had marked parasitemia and *Leucocytozoon* spp. gametocytes occupied virtually every erythrocyte in vessels. These birds had numerous schizonts within endothelial cells of the lung and megaloschizonts in liver, heart muscle and spleen.

All of the owlets with *Leucocytozoon* spp. infections had black fly related skin lesions. Birds less than 10 wk of age had mild *Leucocytozoon* spp. infections and two of the birds in this age group had severe skin lesions related to black fly feeding but no evidence of *Leucocytozoon* spp. infection on histology. Severe *Leucocytozoon* spp. infections were only observed in owls between 10 to 14 wk of age.

DISCUSSION

Based on our findings, black fly feeding alone in great horned owls under 10 wk of age and in concert with *Leucocytozoon* spp. infections in owls 10 to 14 wk of age was determined as the cause of death in 13 of the 28 owl carcasses examined. Three additional road-killed owls had histologic evidence of *Leucocytozoon* spp. infection but were in good body condition.

The owl chicks examined in this study had dozens of bite wounds on unfeathered areas of skin of the face, underside of the wings and inguinal areas caused by black fly feeding. These lesions were characterized by localized edema, tissue necrosis and thrombosis of vessels. In some of the owls the eyelids were swollen closed.

This is the first record of black flies from great horned owls. Black flies are pool feeders (as opposed to channel feeders like the mosquito) that penetrate the skin and produce small craterous lesions using a slashing or biting action involving the stylets and labium which lacerate capillaries to initiate blood flow (Sutcliffe and McIver, 1984). In addition to direct blood loss and localized skin trauma, black fly attacks may initiate systemic illness, allergic reactions or even death. Harwood and James (1979) refer to a systemic reaction to black fly bites in humans known as "black fly fever" characterized by headaches, fever, nausea, adenitis, generalized dermatitis and allergic asthma. In Alberta and Saskatchewan (Canada), intense black fly attacks have caused widespread vascular le-

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sions and mortality in cattle (Fredeen, 1969; Harwood and James, 1979). The pathogenicity of black flies for birds has not been well established. Edgar (1953) reported egg production drops in laying chickens tormented by black flies. S. Cain (pers. comm.) observed mortality and premature evacuation of nests by red-tailed hawk chicks in Wyoming (USA) due to black fly attacks.

The attraction of female black flies to bird corpses was previously reported by Lowther and Wood (1964), who collected over a thousand individuals of *Simulium* (*Eusimulium*) *euryadminiculum* Davies from a single common loon (*Gava immer*). Females of *S. euryadminiculum* were evidently attracted to a substance in the uropygial oil. Black flies collected from corpses of great horned owls are probably attracted to similar odiferous substances, although the uropygial gland of raptors is not as markedly developed as in waterfowl. Most black flies were concentrated on specific parts of the body; thus other chemical or visual clues might serve to orient females once a host is located. However, none of the specimens had evidence of blood feeding because the most important clues for initiation of engorgement (body heat and CO_2) are lacking from dead owls.

The deteriorated condition of the flies prevented us from establishing which species served as vectors of *Leucocytozoon* spp. to owls. Two of the taxa collected, *Helodon decemarticulatus* and members of the *S. aureum* complex, have already been implicated as vectors of *Leucocytozoon berestneffi* to ravens (*Corvus corax*) (Khan and Fallis, 1971). Females of *H. pleuralis* and *S. canonicolum* complex have not been evaluated for *Leucocytozoon* spp.

Evaluation for *Leucocytozoon* spp. infection was limited to formalin-fixed tissue sections. We were able to identify only circulating elongate gametocytes within blood vessels and stages of schizogony within tissues. Unfortunately, blood smears were not obtained from nestlings or birds tethered on platforms.

Leucocytozoon spp. can produce anemia by the destruction of infected erythrocytes (Atkinson and Van Riper, 1991). Erythrocytes may be removed from the circulation by cells of the reticulo-endothelial system because of parasite-induced red cell deformity (Maley and Desser, 1977), immune-mediated processes or because of increased erythrocyte osmotic fragility caused by a suspected anti-erythrocytic factor thought to be produced during hepatic schizogony (Kocan, 1968).

The large numbers of circulating gametocytes and the numerous schizonts and megaloschizonts observed in tissue sections of five owlets were evidence of significant *Leucocytozoon* spp. infection. Although there were focal areas of necrosis in liver and spleen associated with megaloschizonts, there was little associated inflammatory response observed. Schizonts were very numerous in pulmonary capillary endothelium and combined with the large numbers of circulating *Leucocytozoon* spp. gametocytes may have altered the normal pulmonary blood flow and affected pulmonary function as has been suggested in *Leucocytozoon smithi* infections in turkeys (Siccardi et al., 1974).

Mortality from anemia, attributed to black fly feeding and concurrent *Leucocytozoon* spp. infection, was observed only in owls tethered on platforms. The tethering of owls on platforms 3.5 m above the ground may have interfered with their normal black fly avoidance behavior compared to untethered individuals that could choose the location and elevation of roost sites. This may have increased black fly exposure and subsequent *Leucocytozoon* spp. transmission (Rohner and Hunter, 1996). The importance of the tethering process in influencing mortality is difficult to determine. Mortalities due to parasitism were infrequent on the platforms in 1989 ($n = 0$) and 1990 ($n = 3$), years of abundant food supply, but in 1991 the first year of the snowshoe hare decline, ten owlets died from parasitism (Rohner and Hunter, 1996). Presumably tethered birds were ex-

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posed to black flies in each of the three years. The necropsy sample was strongly skewed towards tethered birds because the tethering process facilitated observation and carcasses were located more quickly.

We have evidence that naturally fledged owls were also infected with *Leucocytozoon* spp. Only six carcasses were examined and because of autolysis or scavenging, tissues from only two of these were suitable for histologic examination. Both were road kills but had numerous parasitic schizonts in liver and myocardium.

The ecological consequences of blood parasite infections are poorly understood (Desser and Bennett, 1993). Korpimäki et al. (1993) reported that *L. ziemanni* reduced clutch size in female Tengmalm's owls (*Aegolius funereus*) in Finland. This effect was only observed in a year of moderate to low food availability, and the authors postulated that parasites may compete for critical resources of the female owls necessary for egg-laying.

We suspect that almost every northern great horned owl chick is exposed to black flies and many subsequently become infected with *Leucocytozoon* spp. We propose that under normal conditions and during years of high food supply, as occurred in 1989 and 1990, the young owls are able to successfully fight infection and recover from the anemia caused by the parasites and fledgling survival remains high. Decreased fledgling survival due to black fly feeding and *Leucocytozoon* spp. infection coincided with the 1991 decline in the snowshoe hare cycle (Rohner and Hunter, 1996). The consequences of host-parasite interactions are likely mediated by food availability and other factors. The role of tethering platforms on the prevalence of disease during the period of food shortage is unclear, but researchers using tethering platforms to enhance data collection in raptor studies should be aware of the potential risk arising from exposure to black flies and *Leucocytozoon* spp.

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LITERATURE CITED

ATKINSON, C. T., AND C. VAN RIPER, III. 1991. Pathogenicity and epizootiology of haematozoa: *Plasmodium*, *Leucocytozoon*, and *Haemoproteus*. *In* Bird-parasite interactions, L. J. Loye and M. Zuk (eds.). Oxford University Press, Oxford, England, pp. 19–48.

BENNETT, G. P., M. A. PEIRCE, AND R. W. ASHFORD. 1993. Avian haematozoa: Mortality and pathogenicity. Journal of Natural History 27: 993–1001.

CURRIE, D. C. 1997. Black flies (Diptera: Simuliidae) of the Yukon, with reference to the black fly fauna of northwestern North America. *In* Insects of the Yukon, H. V. Danks and J. A. Downes (eds.). Biological Survey of Canada (Terrestrial Arthropods), Ottawa, Canada, in press.

DESSER, S. S., AND G. F. BENNETT. 1993. The genera *Leucocytozoon*, *Hemoproteus* and *Hepatocystis*. *In* Parasitic protozoa, Vol. 4, 2nd ed., J. P. Kreier (ed.). Academic Press, London, England, pp. 273–305.

EDGAR, S. A. 1953. A field study of the effect of black fly bites on egg production of laying hens. Poultry Science 32: 779–780.

FREDEEN, F. J. H. 1969. Outbreaks of the black fly *Simulium articum* (Malloch) in Alberta. Quaestiones Entomologicae 5: 341–72.

HARWOOD, R. F., AND M. T. JAMES. 1979. Gnats, black flies, and related forms. *In* Entomology in human and animal health, 7th. ed. MacMillan Publishing Co., New York, pp. 148–149.

HERMAN, C. M., J. H. BARROW JR., AND I. B. TARSHIS. 1975. Leucocytozoonosis in Canada geese at the Seney National Wildlife Refuge. Journal of Wildlife Diseases 11: 404–411.

KHAN, R. A., AND A. M. FALLIS. 1971. Speciation, transmission, and schizogony of *Leucocytozoon* in corvid birds. Canadian Journal of Zoology 49: 1363–1367.

KOCAN, R. M. 1968. Anemia and mechanism of erythrocyte destruction in ducks with acute *Leucocytozoon* infections. The Journal of Protozoology 13: 465–468.

KORPIMÄKI, E., H. HAKKARAINEN, AND G. F. BENNETT. 1993. Blood parasites and reproductive success of Tengmalm's owls: Detrimental effects

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on females but not on males? Functional Ecology 7: 420–426.

KREBS, C. L., R. BOONSTRA, S. BOUTIN, M. DALE, S. HANNON, K. MARTIN, A. R. E. SINCLAIR, J. N. M. SMITH, AND R. TURKINGTON. 1992. What drives the snowshoe hare cycle in Canada's Yukon? *In* Wildlife 2001: Populations, D. McCullough and R. Barrett (eds.). Elsevier, London, England, pp. 886–896.

LOWTHER, J. K., AND D. M. WOOD. 1964. Specificity of a black fly, *Simulium euryadminiculum* Davies, toward its host, the common loon. Canadian Entomologist 96: 911–913.

MALEY, G. J., AND S. S. DESSER. 1977. Anemia in *Leucocytozoon simondi* infections. 1. Quantification of anemia, gametocytemia, and osmotic fragility of erythrocytes in naturally infected Pekin ducklings. Canadian Journal of Zoology 55: 355–358.

PEIRCE M. A. 1981. Current knowledge of the haematozoa of raptors. *In* Recent advances in the study of raptor diseases, J. E. Cooper, and A. G. Greenwood (eds.). Chiron Publications, Keighley, West Yorkshire, England, pp. 15–19.

ROHNER, C. 1994. The numerical response of great horned owls to the snowshoe hare cycle in the boreal forest. Ph.D. thesis, Department of Zoology, University of British Columbia, Vancouver, British Columbia, Canada, pp. 42–43, 145–147.

———. 1995. Great horned owls and snowshoe hares: What causes the time lag in the numerical response of predators to cyclic prey? Oikos 74: 61–68.

———. 1996. The numerical response of great horned owls to the snowshoe hare cycle: Consequences of non-territorial "floaters" on demography. Journal of Animal Ecology 65: 359–370.

———, AND F. I. DOYLE. 1992. Methods of locating great horned owl nests in the boreal forest. Journal of Raptor Research 26: 33–35.

———, AND D. B. HUNTER. 1996. First-year survival of great horned owls during a peak and decline of the snowshoe hare cycle. Canadian Journal of Zoology 74: 1092–1097.

SICCARDI, I. W., H. O. RUTHERFORD, AND W. T. DERIEUX. 1974. Pathology and prevention of *Leucocytozoon smithi* infection of turkeys. Avian Diseases 18: 21–32.

SOMMERMAN, K. M. 1953. Identification of Alaskan black fly larvae (Diptera: Simuliidae). Proceedings of the Entomological Society of Washington 55: 258–273.

STONE, A. 1952. The Simuliidae of Alaska (Diptera). Proceedings of the Entomological Society of Washington 54: 69–96.

SUTCLIFFE J. F., AND S. B. MCIVER. 1984. Mechanics of blood-feeding in black flies (Diptera:Simuliidae). Journal of Morphology 180: 125–144.

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