Black-flies and *Leucocytozoon* spp. as Causes of Mortality in Juvenile Great Horned Owls in the Yukon, Canada

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ABSTRACT.—Black fly feeding and infection with the blood parasite *Leucocytozoon* spp. caused mortality in juvenile Great Horned Owls (*Bubo virginianus*) in the Yukon, Canada during 1989-1990. The mortality occurred during a year of food shortage corresponding with the crash in snowshoe hare (*Lepus americanus*) populations. We postulate that the occurrence of disease was mediated by reduced food availability.

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 Kluane Lake area of southwestern Yukon, Canada. The survival of juvenile owls was very high during 1989 and 1990, both years of abundant hare populations. Survival decreased in 1991, the first year of the snowshoe hare population decline (Rohner and Hunter 1996). Monitoring of nest sites combined with tracking of individuals by radiotelemetry provided us with carcasses of 28 juvenile owls found dead during 1990 and 1991 (Rohner and Doyle 1992). Although we observed a variety of causes of death in these carcasses including trauma and bacterial infections, 13 of the 28 owls died from severe anemia, dehydration and extensive skin lesions attributed to feeding by ornithophilic black flies (Diptera: Simulidae) and in several birds, internal lesions caused by heavy concurrent infections with *Leucocytozoon* spp. (Rohner and Hunter 1996, Hunter *et al.* 1997). This was the first record of black flies from Great Horned Owls and provided evidence of black flies as potential pathogens for young owls (Hunter *et al.* 1997).

Black flies are abundant throughout northwestern North America. Of the 70 species of black flies identified from Alaska, USA and the Yukon Territory, Canada, 36 percent are ornithophilic, 39 percent mammalophilic and 25 percent autogenous (Currie 1997). Numerous female black flies were obtained from the carcasses of the juvenile owls, but only 45 of these were sufficiently well preserved for identification. They belonged to four taxa as follows: *Helodon* (*Distosimulium*) *pleuralis* (Malloch), 1; *Helodon* (*Parahelodon*) *decemarticulatus* (Twinn), 3; *Simulium* (*Eusimulium*) *aureum* Fries complex, 3; and *Simulium* (*Eusimulium*) *canonicolum* (Dyar and Shannon) complex, 38 (Hunter *et al.* 1997).

Black flies are pool feeders that penetrate the skin and produce small craterous lesions using a slashing or biting action involving the stylets and labium (Sutcliffe and McIver 1984). Black fly saliva containing anticoagulants, enzymes and histamine, is mixed with the blood preventing clotting until it is ingested by the fly. Black fly bites cause localized tissue damage and if the number of feeding flies is sufficient, they may produce a blood-loss anemia. In addition, the host's reaction to fly attacks may include systemic illness, allergic reactions or even death; these reactions presumably mediated by histamine. 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 hysteria, systemic disease, 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

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chickens tormented by black flies. We could only find a single report of black fly induced pathology in raptorial birds. S. Cain (unpubl. data) observed mortality and premature evacuation of nests by Red-tailed Hawk (*Buteo jamaicensis*) chicks in Wyoming (USA) due to black fly attacks. Black flies are also the main vector for the blood parasite *Leucocytozoon* spp.

Although *Leucocytozoon* spp. can be a serious pathogen in immunologically naive captive or domestic waterfowl, turkeys and chickens (Bennett *et al.* 1993), rarely has it been associated with clinical disease or mortality in wild birds (Herman *et al.* 1975). *Leucocytozoon* spp. infection is common in raptorial birds (Peirce 1981) but documentation of clinical disease and mortality is lacking. The fledgling owls in our study had massive parasitemias and widespread tissue lesions associated with concurrent *Leucocytozoon* spp. infection which we believe contributed to the mortality.

Interestingly, mortality from parasitism only occurred in 1991, the year of the crash in snowshoe hare populations. We have no reason to believe that the owls were not equally parasitized in the previous years of the study, yet in these years clinical disease or mortality due to parasitism was not observed.

We suspect that almost every northern Great Horned Owl chick is exposed to black flies and that most become infected with *Leucocytozoon* spp. Under normal conditions and during years of adequate food supply, as occurred in 1989 and 1990, young birds were able to successfully fight infection, recover from the anemia and fledgling survival remained high. We propose that the occurrence of clinical disease and mortality in 1991 was mediated by reduced food availability and possibly other factors.

Most wildlife managers recognize that disease is endemic in wildlife populations, but there is a tendency to think of disease in terms of individual mortality events or isolated dieoffs. Our understanding of the subclinical effects of disease on individuals or populations is very limited. There is an increasing number of reports demonstrating that subclinical disease in wildlife may affect biological processes such as predator avoidance (Hudson *et al.* 1992, Temple 1987) or reproductive success by influencing mate selection (discussed by Clayton 1991), clutch size (Rohner 1994, Korpimäki *et al.* 1993), or basic behaviors such as nest defense activity (Ilmonen pers. comm.).

That subclinical disease can affect reproductive performance, life history strategies, and survival in wild bird populations is not surprising if one considers the large body of information available in the domestic poultry literature. In growing commercial poultry, subclinical disease, regardless of the etiologic agent, results in decreased growth rates, reduced feed consumption and feed conversion rates, retarded physiologic maturation leading to slow feathering patterns, and reduced immune competence etc., without causing overt clinical disease or mortality. Any of these sorts of physiologic changes induced by subclinical diseases in wild species must reduce the chances of juvenile survival. In adult breeder or commercial laying hens, the first indication of subclinical disease is a reduction or complete cessation of egg production. Parasites and most pathogens compete for critical resources and during periods resource decline, for example during a period of declining food availability, the host maintains itself at the expense of reproduction or offspring survival. This strategy makes sense in species with a relatively long reproductive life span.

In our Great Horned Owl study, black fly feeding, the effects of infection with *Leucocytozoon* spp., and likely other unrecognized subclinical disease occurrences all compete for host resources. We suspect that even in years when mortality due to parasitism is low, black fly feeding may influence other aspects of Great Horned Owl behavior such as nest site selection, timing of nesting, and roost site selection of fledglings. The recognition and biologic importance of subclinical disease has received little attention by researchers, yet may yield interesting and important biological information about the life history strategies of wild species.

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