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Authors: Stuht, John N., Bowerman, William W., and Best, David A.

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Leucocytozoonosis in Nestling Bald Eagles in Michigan and Minnesota

John N. Stuht,[1] **William W. Bowerman,**[2,4] **and David A. Best**[3] [1] Small Animal Clinical Sciences, College of Veterinary Medicine, Michigan State University, East Lansing, Michigan 48823, USA; [2] Gale Gleason Environmental Institute, Lake Superior State University, Sault Sainte Marie, Michigan 49783, USA; [3] U.S. Fish and Wildlife Service, 2651 Coolidge Road, East Lansing, Michigan 48823, USA; and [4] Corresponding author (e-mail: wbowerman@gw.lssu.edu).

ABSTRACT: Thirteen of 21 nestling bald eagles (*Haliaeetus leucocephalus*) examined for blood parasites in Michigan and Minnesota (USA) during June and July 1997 had patent infections of *Leucocytozoon toddi.* No other parasites were seen. The degree of parasitemia was light and varied from 1 to 2 on the Ashford Scale. Several of the infected nestlings appeared to have elevated levels of heterophils in their peripheral circulating blood. One of the infected nestlings also showed signs of severe anemia. We believe this is the first report of *L. toddi* in the bald eagle.

Key words: Anemia, bald eagle, *Haliaeetus leucocephalus,* leucocytosis, *Leucocytozoon toddi,* parasites, survey, threatened species.

The bald eagle (*Haliaeetus leucocephalus*) has been listed as a threatened species in Michigan and Minnesota (USA) under the United States Endangered Species Act (Grier et al., 1983). Its status as a threatened species makes it somewhat difficult to study and there are still aspects of its life history that are poorly understood. Morbidity and mortality factors effecting populations are one of the areas that need further study. The purpose of this investigation was to look for occurrences of blood parasites in nestling bald eagles and to begin to assess the impact of blood parasites on bald eagle populations.

Twenty-one nestling bald eagles from 19 breeding areas in Michigan and Minnesota were sampled during annual banding operations during June and July 1997 (Table 1). Nestlings were from 12 breeding areas within Voyageurs National Park (Minnesota), and six breeding areas in the Upper Peninsula and one from the Lower Peninsula of Michigan. Nestlings were handled in accordance with established guidelines and protocols of the U.S. Fish and Wildlife Service Endangered Species Subpermit No. 96-03 (U.S. Fish and Wildlife Service, Fort Snelling, Minnesota, USA). Nestlings were sexed and aged using the methods of Bortolotti (1984).

Thin blood smears were prepared in the field using a drop of blood from the brachial vein. The smears were air dried immediately and stored in an envelope or slide box to protect them from insects. They were later fixed in absolute methanol and stained with Giemsa in the laboratory. Each slide was examined for parasites in its entirety using low magnification (100×) and for an additional 5 min under oil immersion (970×). Each slide where parasites were not observed was re-examined. The degree of parasitemia was estimated on a logarithmic scale of 0 to 4 following the method of Ashford et al. (1990) as follows: 0 = no parasites seen in the entire smear examined at low power (100×); 1 = fewer than 1 parasite per 100 high power (400×) fields; 2 = 1–10 parasites per 100 high power fields; 3 = 11–100 parasites per 100 high power fields; 4 = more than 100 parasites per 100 high power fields. Parasites were identified by comparing color, morphology and size against descriptions of known species. Blood smears with representative infections (Accession # 88010 and 88011) were deposited at the U.S. National Parasite Collection (USDA, Beltsville, Maryland, USA).

Leucocytozoon toddi was the only blood parasite seen in any of the 21 nestlings examined (Table 1). Blood smears from 14 of the nestlings showed relatively light infection (1–2 on the Ashford Scale). All 12 nestlings from Minnesota were infected, and 2 of 9 nestlings from Michigan, both from breeding areas within Seney National

TABLE 1. Bald eagle nestlings examined for blood parasites in Michigan and Minnesota (USA) during June and July 1997.

Slide[a]	Date	State	Latitude/longitude	Age[b]	Sex	Weight[c]	Parasites[d]	Ashford scale	Breeding area name
556	6/04/97	MI	43°10′N, 84°00′W	39	M	—	NPS	0	Shiawassee SGA
559	6/09/97	MI	46°10′N, 86°30′W	39	M	3.4	NPS	0	Baldy Lake
561	6/10/97	MI	46°00′N, 86°20′W	41	M	3.4	NPS	0	Thunder Lake
562	6/10/97	MI	46°00′N, 86°20′W	46	F	4.3	NPS	0	Thunder Lake
282	6/10/97	MI	45°50′N, 86°30′W	49	M	3.6	NPS	0	Fishdam River
283	6/10/97	MI	45°50′N, 86°30′W	51	M	3.4	NPS	0	Fishdam River
41563	6/11/97	MI	46°20′N, 84°10′W	31	M	2.6	NPS	0	Sugar Island
95035	6/26/97	MN	48°30′N, 93°00′W	55	M	3.9	L	2	Northland Is. II
95036	6/26/97	MN	48°30′N, 93°00′W	43	M	3.1	L	1	N. Windigo Point
95037	6/26/97	MN	48°20′N, 92°50′W	50	F	4.3	L	1	Moxie Is. W.
95038	6/26/97	MN	48°10′N, 92°20′W	60	F	4.0	L	1	Mukooda G
95039	6/27/97	MN	48°20′N, 92°40′W	47	M	3.6	L	1	Sullivan Bay
95040	6/27/97	MN	48°30′N, 92°40′W	47	M	3.2	L	1	Brown's Bay
95041	6/27/97	MN	48°30′N, 92°50′W	42	M	3.4	L	1	Shelland Is.
95042	6/27/97	MN	48°30′N, 92°50′W	40	F	3.5	L	1	Fox Is. II
95043	6/28/97	MN	48°30′N, 92°50′W	42	F	3.4	L	1	W. Fox Is.
95044	6/28/97	MN	48°30′N, 93°00′W	45	M	3.2	L	2	Kocinski Is.
95046	6/28/97	MN	48°20′N, 93°00′W	47	F	4.0	L	1	Gold Portage
95047	6/28/97	MN	48°30′N, 93°00′W	53	F	4.4	L	1	W. Squaw Franks
SC-2	7/01/97	MI	46°10′N, 86°00′W	37	M	1.8	L	1	Seney NWR C-2
SC-1	7/01/97	MI	46°10′N, 85°50′W	25	F	2.5	L	1	Seney NWR D

[a] 95044 and SC-1 deposited with the U.S. National Parasite Collection (Accession #88010 and 88011).
[b] Age in days.
[c] Weight in kg.
[d] NPS = No parasites seen; L = *Leucocytozoon toddi*.

Wildlife Refuge (Table 1), were infected. Seven of eight females and seven of 13 males were infected. There was no obvious relationship between age or weight and infection rate. Several of the infected nestlings appeared to have a leucocytosis of some degree. Heterophil numbers seemed unusually high in these birds. Due to a lack of baseline data, we could not judge the condition more thoroughly.

The blood from the infected nestling at Seney Site C-2 (Table 1) was thin and watery as well and typical of a bird with severe anemia. This bird was well below normal growth-weight curves when compared to other birds of its age and sex (Bortolotti, 1984), but otherwise appeared healthy. We did not have the opportunity to more fully examine this nestling to rule out some concurrent problem other than infection with *L. toddi* that may have contributed to its condition.

The prevalence data as a whole may be misleading and the differences noted were probably due to our sampling chronology and not a true difference in prevalence. None of the seven nestlings examined before 11 June showed infection, whereas all 14 examined after this date were infected. This suggests that many of the nestlings were examined before patency or before transmission began. This would explain what appears to be a significant difference between prevalence in Michigan and Minnesota, and between males and females. It is possible, however, that further study may show that *L. toddi* is indeed not transmitted at some of the sites in Michigan. The lack of obvious relationship between prevalence and age or weight also was most likely due to the limited temporal and spatial nature of our sampling procedure. One would normally expect the infection rate to be higher in older nestlings.

Leucocytozoon toddi is a common blood parasite of falconiformes. It has been recorded in at least 38 species of birds from around the world (Greiner and Kocan, 1977), including the African fish eagle (*Haliaeetus vocifer*) and golden eagle (*Aquila chrysaetos*). We believe that this is the first report of *L. toddi* in the bald eagle. The only blood parasites previously reported from bald eagles are *Plasmodium polare* (Greiner et al., 1981) and *P. elongatum* (Nayar et al., 1998). We suspect that the method we used for detecting blood parasites was not sensitive enough for us to find any of the other parasites commonly seen in the blood of birds. It is often necessary to use isodiagnosis, or other methods to detect infections outside of peak parasitemias (Herman, 1968). It is likely that a more comprehensive survey would produce a more accurate picture of the true occurrence of blood parasites in the bald eagle.

Despite the common occurrence of *L. toddi,* there appears to be relatively little known about many basic aspects of its life history (Greiner and Kocan, 1977). Schizogony is unknown, but may be multiform similar to *L. simondi* (Peirce and Marquis, 1983). The vector is unknown but may be a black fly (Simuliidae) (Greiner and Kocan, 1977) or a *Culicoides* spp. (Ashford et al., 1991). In all likelihood it is a black fly. The only species of *Leucocytozoon* known to be transmitted by *Culicoides* spp. may belong to another genus entirely (Bennett et al., 1965). These are particularly important bits of information on the life history of *L. toddi* that need further clarification if we are to better understand the impacts of this parasite on its host and host populations. All species of the genus *Leucocytozoon* that have been shown to have a multiform schizogony like *L. simondi* appear to be pathogenic to some degree (Fallis and Desser, 1974). Equally important is more information on vectors. Their abundance and emergence pattern will determine to a great degree what effect *L. toddi* will have on its host. A heavy infection at a young age is more likely to be harmful than a light infection at any age.

It is unfortunate that we were unable to examine the eagle nestlings more thoroughly. The mere presence of *L. toddi* tells us very little about the effects of the parasite on its host. Though leucocytosis and anemia are both conditions that are commonly associated with leucocytozoonosis in birds (Fallis and Desser, 1974), they can be caused by other conditions as well. Typically, with leucocytozoonosis, anemia and leucocytosis accompany morbidity and mortality, but not always (Kocan and Clarke, 1966; Fallis and Desser, 1974; Herman et al., 1975; Hodge et al., 1981). Anemia can also be caused by nutritional factors or by simple blood loss alone. Hunter et al., (1997) recently reported fatal anemia in fledgling great horned owls (*Bubo virginianus*) from black fly hematophaga. Not much is known about hematophaga in birds though Fallis and Desser (1974) suggested that any birds confined to nests in the canopy that are unable to seek shelter from biting insects are at high risk of excessive hematophaga by black flies. They are also at risk of acquiring heavy infections of *Leucocytozoon* sp. as well. These conditions certainly apply to the Seney National Wildlife Refuge where ornithophilic black flies are present from April to September in most years, and Voyageurs National Park where black flies and other blood feeding insects are known to be abundant at times.

Although most investigators believe that *L. toddi* is harmless (Ashford et al., 1990, 1991), Peirce and Marquiss (1983) found that chicks of sparrow hawks (*Accipiter nisus*) in Scotland (UK) with very high parasitemias of *L. toddi* may become temporarily listless from anemia. Although the eagle nestling that we examined with signs of anemia from a breeding area in Seney did not have a large number of *L. toddi* parasites in its peripheral circulating blood at the time we obtained a blood sample, it is very difficult to evaluate parasitemia without some supporting

information. *Leucocytozoon simondi* for example can kill goslings (Herman et al., 1975) and ducklings (Hodge et al., 1981) without detectable parasites in peripheral circulating blood. Toyne and Ashford (1997) speculate that this might explain why they found only light infections of *L. toddi* in a study of nestling goshawks (*Accipiter gentilis*). Heavy infections if they occurred, may have killed nestlings before patency.

As is often the case with so many parasites, only well controlled experimental infections can answer the question of whether or not they are harmful to their hosts. The difference between what appears to be a benign parasitism and pathogenesis depends entirely on the nature of the interaction between the host, parasite and the environment. This interaction is complex and unique to each site where the parasite and host occur. If the schizogony of *L. toddi* is shown to be strain dependent like it is for the schizogony of *L. simondi* (Desser and Ryckman, 1976; Desser et al., 1978) then *L. toddi* may well prove to be a harmless parasite to a host in one location and pathogenic to the same host in another.

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