The Condor 112(4):849–853

PREVALENCE OF HEMATOZOA INFECTIONS AMONG BREEDING AND WINTERING RUSTY BLACKBIRDS

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Abstract. The Rusty Blackbird (*Euphagus carolinus*) has declined precipitously over the past several decades, and stressors on both the breeding and wintering grounds are suspected causes. Over 3 years, we collected blood samples from breeding birds in Alaska and Maine and from wintering birds in Mississippi and Arkansas to determine the prevalence of hematozoan infections at different times of the year. The prevalence of hematozoa (percent of birds infected) in Alaska was 44% of 43 birds, lower than previously reported from Newfoundland and Ontario (83% of 23 birds). Blood parasites were found among 67% of 12 Rusty Blackbirds sampled in Maine, not different from the prevalence in Newfoundland and Ontario. *Leucocytozoon* was the most common parasite; few breeding birds were infected with *Plasmodium* or *Trypanosoma* sp. During the winter in Mississippi and Arkansas, the overall prevalence of hematozoa was 49% over three years. In winter as in summer, *Leucocytozoon* was the most commonly encountered parasite ($n = 62$ birds), followed by *Haemoproteus* ($n = 5$), microfilaria ($n = 2$), *Trypanosoma* ($n = 2$), and *Plasmodium* ($n = 1$). The prevalence of hematozoa among wintering Rusty Blackbirds was much higher than expected because winter is generally a time when there are few transmissions and in most birds blood parasites are absent from the peripheral circulation. This high prevalence might indicate a nonseasonal relapse of hematozoan infections among wintering birds; possibly due to high levels of stress, which are known to lower the immune response and trigger nonseasonal relapses.

Key words: Rusty Blackbird, hematozoa, blood parasites, Leucocytozoon, Haemoproteus, Trypanosoma, *microfilaria.*

Prevalencia de Infecciones por Hematozoos entre Individuos Reproductivos e Invernantes en *Euphagus carolinus*

Resumen. Las poblaciones de *Euphagus carolinus* han disminuido marcadamente durante las últimas décadas y se sospecha que entre las causas se encuentran factores de estrés que operan tanto en las áreas de cría como en las de invernada. A lo largo de tres años, recolectamos muestras de sangre de aves que se estaban reproduciendo en Alaska y Maine y de aves que estaban pasando el invierno en Mississippi y Arkansas para determinar la prevalencia de infecciones por hematozoos en diferentes momentos del año. La prevalencia de hematozoos (porcentaje de aves infectadas) en Alaska fue del 44% de 43 aves, un valor menor que el documentado para Newfoundland y Ontario (83% de 23 aves). Se encontraron parásitos sanguíneos en el 67% de 12 individuos muestreados en Maine, lo que no difirió de la prevalencia encontrada en Newfoundland y Ontario. *Leucocytozoon* fue el parásito más común y pocas aves estuvieron infectadas con *Plasmodium* o *Trypanosoma* sp. Durante el invierno, la prevalencia total de hematozoos fue del 49% a lo largo de tres años en Mississippi y Arkansas. Tanto en el invierno como en el verano, *Leucocytozoon* fue el parásito más comúnmente encontrado ($n = 62$ individuos), seguido por *Haemoproteus* ($n = 5$), microfilarias ($n = 2$), *Trypanosoma* ($n = 2$) y *Plasmodium* ($n = 1$). La prevalencia de hematozoos entre individuos invernantes fue mucho mayor que la esperada, debido a que el invierno generalmente es un período en el que ocurren pocas transmisiones y en la mayoría de las aves los parásitos sanguíneos están ausentes de la circulación periférica. Esta alta prevalencia podría indicar una recaída no estacional de las infecciones por hematozoos entre las aves invernantes posiblemente debida a altos niveles de estrés, los cuales reducen la respuesta inmunológica y conducen a recaídas no estacionales.

Manuscript received 30 July 2010; accepted 10 August 2010.
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The Condor, Vol. 112, Number 4, pages 849–853. ISSN 0010-5422, electronic ISSN 1938-5422. DOI: 10.1525/cond.2010.100143

INTRODUCTION

Wild birds and their parasites have undergone a long period of mutual co-evolution and subsequent adaptation (Loye and Zuk 1991). In most free-living wild birds, outright mortality from blood-parasite infections occurs infrequently (Bennett et al. 1993, Valkiunas 2005), with the notable exception of naïve hosts (Greiner 1991, Atkinson et al. 1995). However, the other component of pathogenicity involves the energetic costs of the immune response by the host, which may become more visible when animals' resources are limited (Weber and Stilianakis 2007). Davidar and Morton (1993) reviewed the potential effects of hematozoan infections on the survival and fitness of birds and suggested that nutritional stress, lowered mating success, and increased susceptibility to predation are some possible additional indirect effects on infected birds. Therefore, there is increasing interest in ecological immunology and quantifying the cost of fighting diseases at the expense of other life-history traits (Sheldon and Verhulst 1996, Merino et al. 2000). Garvin et al. (2003) stressed the importance of understanding the epizootiology of the avian host–parasite relationship for species that are in decline.

Hematozoa of the genera *Leucocytozoon*, *Haemoproteus*, and *Plasmodium* (Sporozoa: Haemosporida) are common among North American birds (Greiner et al. 1975). The life cycle of these haemosporidians is a two-stage process involving development within both an insect and bird host. Transmission is by an insect vector, typically a blood-feeding dipteran, within which the parasite reproduces sexually (Atkinson 1999). Within an infected bird host, the parasite moves between organs and the circulatory system over the course of the year. After a bird is infected there is an initial prepatent period in which the hematozoan reproduces asexually in various organs of the host. The acute phase occurs when a portion of the parasite's population moves into the peripheral circulation, where it is available to insect vectors for transmission (Valkiunas 2005). The disease enters the chronic phase with the hematozoa residing within internal organs. Relapse occurs when the parasites move again from the visceral organs back to the peripheral circulation and is thought to be stimulated in the spring by the hormonal changes and stresses associated with the onset of breeding (Applegate 1970, Garvin et al. 2003). It is thought that among birds wintering in the temperate zones, blood parasites are transmitted primarily during the breeding season, when the abundance of parasites in the blood is greatest (Bennett and Cameron 1974, Garvin et al. 2003, 2004), vectors are most numerous (Bennett and Cameron 1974), and the stresses and energy costs of breeding reduce immunocompetence (Sheldon and Verhulst 1996). The prevalence of hematozoa among birds wintering in temperate zones is typically lowest during the nonbreeding season (Bennett et al. 1982, Barnard and Bair 1986) when vectors are not present. Thus, rarely are birds migrating to temperate zones sampled for hematozoa during the winter.

On the basis of surveys in both the breeding season and winter, the Rusty Blackbird (*Euphagus carolinus*) has declined precipitously over the past 50 years (Sauer et al. 1996, 2008), yet it is the least studied of North American blackbirds (Avery 1995). The cause of this decline remains unknown, but stressors on both the boreal breeding and temperate wintering grounds have been suggested, with losses of winter habitat a leading candidate (Greenberg and Droege 1999). During both the breeding season and winter the prevalence of hematozoa in the Rusty Blackbird is poorly known. Greiner et al. (1975) reported 19 infected birds in a sample of 23 across North America for a prevalence of 83%. Bennett et al. (1974) found all 20 of his Rusty Blackbirds in insular Newfoundland infected, while Clarke (1946) found no infections among three birds sampled in Ontario. Greiner et al. (1975) cited both of these papers, so presumably they included both samples, although the totals do not match. Williams and Bennett (1977) reported a low prevalence (13%, 1 of 8 birds infected) in nonbreeding Rusty Blackbirds from Maryland, but it is not clear when the samples were taken.

In this study we examined the prevalence of hematozoan infection of the Rusty Blackbird from blood samples collected from adults nesting at the extremes of the breeding range in Alaska and Maine and from adults and juveniles wintering in the species' core winter range in Arkansas and Mississippi. We were interested in identifying geographic areas or seasons with high prevalence of blood parasites, as these may indicate locations or times of the year at which Rusty Blackbirds are subjected to stressors that limit their immunity to infection. Such an indication would apply particularly to winter populations, in which we did not expect much exposure to these parasites because of the dormancy of insect vectors available to transmit the disease.

METHODS

Matsuoka et al. (2010) located nesting Rusty Blackbirds in boreal wetlands at Elmendorf Air Force Base and the U.S. Army's Fort Richardson near Anchorage, Alaska. The birds were captured in mist nets placed near their nests from 29 May to 26 June, 2007–2009. In Maine, from 29 May to 25 June 2007, Powell et al. (2010) captured nesting Rusty Blackbirds in mist nets. During the winters of 2005–2006 through 2007–2008, from mid-December to early March, birds were captured in mist nets at 12 locations across 3200 km^2 in Mississippi and Arkansas. Eleven capture sites were located near Greenville, Mississippi, and included Washington, Sharkey, and Yazoo counties. The one additional capture site was near Johnson, Washington County, Arkansas. Birds were captured in three different winter habitats—bottomland hardwood forests, forest fragments along creeks, and pecan orchards—and were sometimes lured into mist nets with bait consisting of boiled eggs mixed with corn meal and cracked corn.

TABLE 1. Prevalence of hematozoan infections among breeding (Maine and Alaska) and wintering (Mississippi and Arkansas) Rusty Blackbirds.

State	Year	Total birds	Total infected	Number of birds with specific infections				
				Leucocytozoon	*Haemoproteus*	*Plasmodium*	*Trypanosoma*	Microfilaria
Maine	2007	12	8	7		1	2	
Alaska	2007	15	5	5				
	2008	21	12	11			1	
	2009	7	2	2				
Mississippi	2005–2006	50	30	26	2		1	1
	2006–2007	50	24	22	1	1	1	
	2007–2008	51	20	17	2			1
Arkansas	2006–2007	5	1	1				

Blood samples were obtained from each bird by puncture of the ulnar vein with a sterile 27-gauge hypodermic needle, allowing the blood to pool, and then collecting the blood in a heparinized capillary tube. A small drop of blood was then smeared into a thin layer across the surface of a glass slide. After the blood dried it was immediately fixed in methanol (Alaska) or 80% ethanol (Mississippi and Arkansas) and later stained with Giemsa stain at the end of the field season (Bennett 1970). The stained smears were examined under 100× magnification for *Trypanosoma* sp. and microfilaria and under oil immersion (1000× magnification) for *Haemoproteus* sp., *Leucocytozoon* sp., and *Plasmodium* sp. Each smear was examined under 100× magnification throughout its entire suitable area and at 1000× magnification for 10 min or until 50 fields were viewed under oil immersion (approximately 10 000 erythrocytes). Protozoan parasites were identified to genus.

The prevalence of blood parasites is equivalent to the percentage of birds detected with one or more parasites, but this estimate is conservative because a low-level infection might be overlooked by microscopy. Thus our estimates of prevalence are likely lower than those estimated with more sensitive techniques such as genetic markers. For samples from the breeding grounds, we used a chi-squared test to test for differences in prevalence of hematozoa infection between Alaska and Maine and among years in Alaska. We also used chi-squared tests to compare levels of prevalence in Alaska and Maine to those of reported from Canada by Greiner et al. (1975). For winter samples, we used a chi-squared test to test for difference in prevalence by year and month, defining the level of significance as $\alpha = 0.05$.

RESULTS

Blood smears were collected from a total of 43 nesting Rusty Blackbirds from Alaska. The percentage of birds infected with hematozoa (prevalence) by year was 5 of 15 (33%) in 2007, 12 of 21 (57%) in 2008, and 2 of 7 (29%) in 2009. Prevalence did not vary significantly by year ($\chi^2 = 2.9$, $P = 0.08$) and was 44% for the three years combined. Eighteen of these birds were infected with *Leucocytozoon*, one with *Trypanosoma*. In Maine, 8 of 12 birds (67%) were infected with hematozoa. Seven of the birds were infected with *Leucocytozoon*, two with *Trypanosoma*, and one with *Plasmodium*. One bird had infections of both *Leucocytozoon* and *Trypanosoma*; another had infections of both *Leucocytozoon* and *Plasmodium* (Table 1). The prevalence rate reported by Greiner et al. (1975) from eastern Canada, 19 of 23 birds (83%), was higher than that we found in Alaska ($\chi^2 = 9.1$, $P = 0.003$) but similar to that in Maine ($\chi^2 = 1.1$, $P = 0.19$).

Blood smears were collected from a total of 156 wintering Rusty Blackbirds in Arkansas and Mississippi. Prevalence by year was 30 of 50 birds (60%) in the winter starting in 2005, 25 of 55 birds (45%) in 2006, and 20 of 51 birds (39%) in 2007. The infection rate did not vary significantly by year ($\chi^2 = 1.9$, $P = 0.17$) and was 49% for the years combined. As on the breeding grounds, *Leucocytozoon* was by far the most common pathogen, found in 66 birds. *Haemoproteus* was present in five birds, *Trypanosoma* in five, and *Plasmodium* in two. Two birds were also infected with microfilaria (Table 1). The percentage of wintering birds infected with hematozoa varied by month ($\chi^2 = 9.2$, $P = 0.03$) with infection rates lowest in February (34% of 50 birds), intermediate in December (50% of 14 birds) and January (52% of 80 birds), and highest in March (70% of 10 birds).

DISCUSSION

Studies of blood parasites of nonbreeding birds are few. Two studies of birds wintering within the temperate zone estimated monthly rates of prevalence in Vermont (Barnard and Bair 1986) and in France (Bennett et al. 1982). The prevalence of hematozoa among birds wintering in Vermont averaged 15%, five times lower than in the summer, when prevalence reached a high of 78% in late July (Barnard and Bair 1986). In the birds sampled in France, Bennett et al. (1982) found a much lower prevalence, but during the breeding season prevalence (10%) was 10 times higher than during winter (1.0%). Both studies found prevalence among birds to be lowest in winter, rising

sharply in April, peaking in late summer, and dropping precipitously in fall. Similarly, Rintamäki et al. (1999) found that the prevalence of *Leucocytozoon* and *Trypanosoma* infections was higher in Common Redstarts (*Phoenicurus phoenicurus*) breeding in northern Finland in June and July (48%) than in redstarts passing through southern Finland during spring migration in May (13%).

The high prevalence of hematozoa we found among wintering Rusty Blackbirds (49%) was unexpected and might be evidence of a relapse—it is less likely to be the result of new infections acquired during the winter months. A relapse is the reappearance of haemosporidians in the blood after a period of latent infection. Valkiunas (2005) identified two types of relapse: seasonal and nonseasonal. A seasonal relapse occurs in the spring and is most likely triggered by the breeding-season activities. The nonseasonal relapse is poorly understood; Valkiunas (2005) suggested stresses and associated decrease of immunity as possible causes. Barrow (1963) noted a behavioral aspect to relapse, showing that in waterfowl relapses of *Leucocytozoon simondii* could be induced at any time of the year by aggressive interactions. The high prevalence of hematozoans in overwintering Rusty Blackbirds suggests a nonseasonal relapse. The traditional winter habitats of the Rusty Blackbird are swamps wooded with hardwoods, riparian zones, and river bottomlands, habitats whose reduction has continued since European settlement (Greenberg and Droege 1999). Conversions of habitat or changes in hydrology may be forcing Rusty Blackbirds to feed in less optimal habitat where competition for preferred foods between the sexes, age groups, or with other species of blackbirds is lowering the birds' immune response and causing a subsequent relapse of a chronic but latent infection.

The epizootiology of hematozoan disease is dependent upon the number and availability of vectors and, given the milder climate of Mississippi and Arkansas, the possibility exists that hematozoa might be transmitted actively during the winter. In northern Florida, Stacey et al. (1990) found that *Leucocytozoon smithi* was transmitted to Wild Turkeys (*Meleagris gallopavo*) from January through early April but that *Haemoproteus meleagridis* was not transmitted during the winter because of a lack of vectors. O'Dell and Robbins (1994), however, found no evidence of winter transmission of hematozoa to Wood Ducks (*Aix sponsa*) in Missouri and suggested that suitable insect vectors were absent or that other factors inhibited transmission. While it cannot be discounted, winter transmissions have not been demonstrated in a passerine bird overwintering in temperate regions. Even in the neotropics, Garvin et al. (2004) found no evidence of local transmissions of blood parasites between October and March among American Redstarts (*Setophaga ruticilla*) overwintering in Jamaica. Evidence to date does not support active transmission of *Leutocytozoon* as a factor contributing to the high prevalence we found among wintering Rusty Blackbirds. If suitable vectors were present in Mississippi and Arkansas and active transmission were occurring, then we should see an increase in infections through the winter, provided that birds infected earlier on the summer grounds are not dying at an increased rate. Instead, the prevalence of hematozoan infections remained constant from December through January and decreased in February, with a slight increase in March. However, the slight increase in prevalence we detected from the small sample of birds captured in March might be due to either new transmissions resulting from the spring emergence of insect vectors or a seasonal relapse triggered by the onset of migration. More information is needed to determine whether the high prevalence of hematozoa among wintering Rusty Blackbirds is the result of a relapse. If the prevalence is low among fall migrants and increases among wintering birds, then a nonseasonal relapse (Valkiunas 2005) might be suspected. Ideally, prevalence should be monitored through the winter at single site since it may have varied among the multiple sites at which we sampled. Information about body condition and stress hormones and variation in habitat use by sex and age class should be recorded and analyzed. Such information might help explain patterns in disease prevalence (Weatherhead and Bennett 1991).

Both intra- and interspecific variation in the prevalence of blood parasites is well documented (Merilä et al. 1995), and factors contributing to this variation are quite varied (Deviche et al. 2001). It was not clear why the prevalence of hematozoan infections among Rusty Blackbirds in Alaska (44%) is less than half that reported from Newfoundland (100%; Bennett et al. 1974) and intermediate in Maine (67%). We sampled breeding Rusty Blackbirds for blood parasites earlier in Alaska and Maine (May and June) than Bennett et al. (1974) sampled them in Newfoundland (May–August). Thus the pattern may reflect in part an increase in prevalence through the breeding season. However, the prevalence of hematozoa we found among breeding Rusty Blackbirds is also higher than the 24% reported among other North American blackbirds (Grenier et al. 1975) and also contrary to the general pattern in North America, of prevalence higher in western Canada and subarctic Alaska than in other regions of the U.S. and Canada (Greiner et al. 1975). Rusty Blackbirds breeding in the eastern section of their range may be particularly susceptible to hematozoan infections because of differences in vectors' densities or lowered immunity due to environmental stressors such as high exposures to methylmercury (Edmonds et al. 2010). *Leucocytozoon*, the primary blood parasite found among breeding and wintering Rusty Blackbirds, has been found to cause severe anemia, weight loss, and mortality among some juveniles (Greiner 1991, Bennett et al. 1993, Atkinson 1999), and additional study is need to clarify the causes and effects of the high prevalence in both the eastern breeding range and winter range of the Rusty Blackbird. Such research may provide important clues to the factors contributing to the species' decline.

ACKNOWLEDGMENTS

We thank Luke Powell for providing samples from Maine and Jason Luscier for providing samples from Arkansas. We also thank Russell Greenberg and two anonymous reviewers who provided valuable comments on an earlier draft of the manuscript. We thank the USDA Forest Service, Center for Bottomland Hardwoods, and the Theodore Roosevelt National Wildlife Refuge Complex for providing housing, and logistical support. The Theodore Roosevelt Complex, Leroy Percy State Park, Delta National Forest, Delta Research and Extension Center, and Frederick Ballard and other farmers kindly permitted our work on their refuges and land. We very much thank our field assistants for collecting blood smears. Assistants, travel, and material were supported by the U.S. Fish and Wildlife Service, the U.S. Geological Survey, the Canadian Wildlife Service, Friends of the National Zoo, the Max Planck Institute for Ornithology, Andechs, the German Ethological Society, the Society for Tropical Ornithology, and the Arthur-von-Gwinner Foundation.

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