PREVALENCE, INTENSITY, AND PATHOGENICTY OF BLOOD PARASITES IN SPRING MIGRANT LANDBIRDS

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By

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ABSTRACT

Diseases may have a profound effect on the persistence and evolution of populations by altering energetic condition or reproductive success of individuals. For example, it has been hypothesized that hematozoan infections may drive the evolution of sexual dichromatism in birds (Hamilton & Zuk 1982). Ecological studies have provided evidence in support of parasite-mediated sexual selection by demonstrating that hematozoan infection results in reduced reproductive success. Few studies, however, have investigated the effects of hematozoa on migrating landbirds. My research contributes to our knowledge of hematozoan parasites and their effects on migratory birds by: (1) developing a semi-automated method to quantify hematozoan intensity, (2) determining interspecific differences in prevalence and intensity of infection at a northerly migratory stopover site, and (3) elucidating pathogenic effects of hematozoans on migrating landbirds.

Intensity of hematozoan infection is infrequently quantified because accurate calculations require visual counts of parasites relative to the number of erythrocytes counted. Manual quantification of erythrocytes can be circumvented using ImageJ software (National Institute of Health) to count erythrocyte nuclei from digital images. I compared intensity of infection calculated from traditional visual erythrocyte counts to

that calculated from digital erythrocyte counts. I found that the ratio of microscope erythrocyte counts to image erythrocyte counts did not differ between two randomly selected groups (mean difference=0.14, SE=0.07, $F_{1, 34}$=2.15, P=0.15), indicating that image erythrocyte counts can accurately estimate the number of erythrocytes within larger microscopic fields of view. Intensity of hematozoan infection calculated from manual quantification of 2,000 erythrocytes was 0.46 times lower (bootstrap P=0.02) than intensity calculated from digital quantification of 50,000 erythrocytes. I contend that digital quantification of hematozoa infection offers a rapid and more accurate method to quantify infections of varying intensity.

Using digital quantification techniques, I examined whether hematozoan infection was associated with extent of prealternate molt, refueling performance, energetic condition, and arrival timing during migratory stopover. Blood samples were collected from Yellow-rumped Warblers (*Dendroica coronata*, n = 519), Magnolia Warblers (*Dendroica magnolia*; n = 385), and Yellow Warblers (*Dendroica petechia*; n = 216) from mid-April through May of 2004 and 2005 in northwestern Ohio. I found that 36.3% of Magnolia Warblers, 12.7% of Yellow Warblers, and 58.3% of Yellow-rumped Warblers were infected with one or more genera of hematozoa. Fewer Yellow-rumped Warblers (2.1%) were infected by *Plasmodium* spp. than Magnolia (8.9%) or Yellow Warblers (4.4%) (Fisher's Exact P<0.01), while *Leucocytozoon* spp. infected fewer Yellow Warblers (0.0%) than Magnolia (8.9%) and Yellow-rumped Warblers (5.8%)

(Fisher's Exact $P<0.01$). Interspecific differences in prevalence and increased prevalence at a northerly stopover site suggests that relapse occurs during spring migration.

Akaike Information Criterion and multi-model averaging provided evidence that late arrival (prevalence $\beta=0.13$, SE=0.01, RVI=0.98; intensity $\beta=0.13$, SE=0.03, RVI=1.00), and reduced energetic condition (intensity $\beta=-0.27$, SE=0.12, RVI=1.00) at stopover sites were associated with higher probability and intensity of infection in Yellow-rumped Warblers. In contrast, probability and intensity of infection in migrant Magnolia and Yellow Warblers were not significantly associated with either energetic condition or capture date. Extent of prealternate molt and refueling performance were not associated with hematozoan infection in migrant Magnolia and Yellow-rumped Warblers. However, intensity of *Haemoproteus* or *Plasmodium* infection was higher in second year Magnolia and Yellow-rumped Warblers ($\beta=-0.98$, SE=0.52, RVI=0.98; $\beta=-1.23$, SE=0.34, RVI=1.00 respectively) than after second year individuals. This study provides evidence that for some migrating landbird species, events during one stage of the annual cycle could influence parasitization, energetic condition, migratory timing, and reproductive performance in subsequent stages.

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CHAPTER 1

INTRODUCTION

Historically, avian hematozoa were thought to have little impact on individuals or populations because highly virulent parasites are less likely to be transmitted to new hosts if they induce mortality (Anderson & May 1979; Herman 1944; Toft 1991). Early studies of avian hematozoan therefore resulted in host-parasite checklists (Bennett et al. 1982; Coatney 1936, 1937; Herman 1944) and catalogues of prevalence (Greiner et al. 1975). Hamilton and Zuk's (1982) theory of parasite-mediated sexual selection generated renewed interest into the study of blood-born parasites. Under this model, bright plumages provide a heritable and honest signal of individual health and/or immunocompetence. Subsequent research has attempted to quantify the pathogenicity and understand the epidemiology of avian hematozoa.

Hamilton and Zuk's (1982) theory of parasite-mediated sexual selection in birds is based upon the assumption that parasites directly or indirectly influence an individual's reproductive success. Recent experimental and observational studies have demonstrated that parents infected by hematozoa are more likely to have later clutch initiation (Allandar & Bennett 1995), smaller clutches (Marzal et al. 2005), reduced hatching success (Marzal et al. 2005; Sanz et al. 2001), higher rates of clutch desertion (Sanz et al. 2001), and reduced fledging success (Dunbar et al. 2003; Marzal et al. 2005; Merino et

al. 2000). Furthermore, hematozoan infections are associated with lower nestling body mass (Merino et al. 1996), and lower parental body mass (Dawson & Bortolotti 2000; Merino et al. 2000). However, other studies have observed a positive correlation between prevalence and brood size (Davidar & Morton 1993; Nordling et al. 1998; Ots & Horak 1996) indicating that 'high quality' individuals may be able to afford the cost of immunosuppression and parasitism associated with increased reproductive effort (Drent et al. 2003; Raberg et al. 1998).

Avian hematozoa may also lower reproductive success by causing infected individuals to delay migration. Parasitized Barn Swallows (*Hirundo rustica*) (Moller et al. 2004) and Pied Flycatchers (*Ficedula hypoleuca*) (Ratti et al. 1993) arrive on the breeding grounds later than unparasitized individuals. Reproductive performance of late migrants will be reduced if they are unable to obtain high quality territories, acquire high quality mates, or complete nesting early (Drent et al. 2003; Smith & Moore 2005). The costliness of migration and pre-alternate molt ensures that only the 'fittest' individuals are able to possess bright plumage, remain healthy (immunologically or energetically), and arrive early on breeding grounds (Gustafsson et al. 1994; Marra et al. 1998; Ninni et al. 2004; Ratti et al. 1993).

Studies investigating epidemiology of avian hematozoa have demonstrated that prevalence varies geographically (Blanco et al. 2001; Greiner et al. 1975; Sol et al. 2000; Young et al. 1993) and seasonally (Deviche et al. 2001b; Garvin et al. 2003; Schrader et al. 2003; Weatherhead & Bennett 1991). Birds are thought to suppress infections to a latent (i.e. dormant) state until elevated levels of hormones associated with stress, migration, or breeding condition induce relapse (Allander & Sundberg 1997; Gustafsson

et al. 1994; Valkiunas et al. 2004). Therefore, avian hematozoa are more prevalent during the breeding season in nearctic regions where immunosuppression and competent vectors occur (e.g. Deviche et al. 2001b; Greiner et al. 1975; Young et al. 1993). Interspecific differences in prevalence are often attributed to species-specific characteristics of avian hosts (e.g. immunology, geography, nesting height, and habitat preferences) (Deviche et al. 2001a; Greiner et al. 1975). For example, Tella et al. (1999) found that avian hematozoa were more prevalent in raptor species that bred in forested habitats and had shorter embryonic development and larger geographic ranges.

Magnolia Warbler (*Dendroica magnolia*), Myrtle Warbler (*Dendroica coronata*), and Yellow Warbler (*Dendroica petechia*), the focal species of this investigation, breed in northern coniferous forests, mixed coniferous-deciduous forests, and wet-deciduous thickets, respectively. Northern forests have streams and moist, organic soil that black flies (simulids) and sand flies (*Culicoides*), vectors of *Haemoproteus* and *Leucocytozoa*, require to breed (Greiner et al. 1975; Murphree & Mullen 1991). Stagnant-marsh habitats provide breeding habitat for mosquitoes (*Culex* and *Aedes* species), vectors of *Plasmodium*.

LITERATURE REVIEW

Life History and Spatial Distribution of Avian Hematozoa

Avian hematozoans are a phylogenetically and geographically diverse group of protozoa that utilize the blood stream for growth and reproduction (Atkinson & Van

Riper 1991). The most prevalent order of these parasites (Hemosporidia) infect at least 68% of the world's avian species (Atkinson & Van Riper 1991). In North America, it was estimated that at least 36.3% of all individual birds are infected with one or more species of *Haemoproteus* (19.5%), *Leucocytozoa* (17.7%), *Trypanosoma* (3.9%), *Plasmodium* (3.8%), or *Microfilaria* (3.1%) (Greiner et al. 1975). The spatial distribution of hematozoan prevalence, defined as the percentage of infected individuals in the population, is determined by complex interactions between the parasite, vector, avian host, and environment.

Genera of Hemosporidia commonly found in birds (*Haemoproteus*, *Leucocytozoa*, and *Plasmodium*) undergo several stages of reproduction in avian hosts and arthropod vectors (Figure 1.1). Arthropod vectors of *Haemoproteus*, *Leucocytozoa*, and *Plasmodium* include louse flies (Hippoboscidae) or sand flies (Ceratopogonidae), black flies (Simulidae), and mosquitoes (Culicidae), respectively (Atkinson 1999). These dipterans transmit sporozoites, the infective stage of the parasite, to avian hosts during the process of taking a blood-meal (Fig. 1.1 A). Once in the avian bloodstream, sporozoites migrate into the visceral organs to intracellularly grow and asexually produce merozoites (Fig. 1.1 B). This period of development and reproduction within host tissues (the prepatent phase) lasts approximately 14 days before the merozoites migrate out of the visceral organs to invade circulating erythrocytes at the start of the patent phase (Atkinson & Van Riper 1991; Garvin et al. 2003). During the patent phase, merozoites asexually produce gametocytes, which are infective to dipteran vectors. After gametocytes are ingested by vectors, hemospridians undergo several stages of sexual and asexual reproduction in the midgut and salivary glands (Fig. 1.1 C). From the salivary

gland, hemosporidians are transmitted to a new avian host as sporozoites during the process of taking a blood-meal (Atkinson 1999; Atkinson & Van Riper 1991).

Through phylogenetic or evolutionary constraints, arthropod vectors appear less able to produce infective parasites than their complimentary avian species (Atkinson 1988; Atkinson et al. 1983; Garvin & Greiner 2003a). Atkinson (1988) found that the development of *Haemoproteus meleagridis* sporozoites occurred in relatively few experimentally infected *Culicoides edini* (60%) or other *Culicoides* species (less than 25%). Because parasites within vectors undergo several stages of sexual and asexual reproduction, arthropods that ingest gametocytes may not always produce sporozoites. The interplay between species-specific sporozoite development and successful parasite transmission has been well studied in central Florida (Atkinson et al. 1988; Atkinson et al. 1983; Garvin & Greiner 2003a). There, only three of fifty *Culicoides* species were ornithophylic, abundant, and able to support the production of sporozoites (Atkinson 1988; Atkinson et al. 1983; Garvin & Greiner 2003a). A high degree of transmission still occurs because competent vectors, arthropods that successfully transmit sporozoites to birds, comprise 35.3% of the total *Culicoides* population (all species combined) and exhibit spatial and temporal overlap with Blue Jay (*Cyanocitta cristata*, Garvin & Greiner 2003a) and Wild Turkey (*Meleagris gallopavo*, Atkinson et al. 1988) roosting and nesting behavior.

The resulting geographical difference in prevalence is thus influenced by the abundance of competent vectors (Blanco et al. 2001; Sol et al. 2000). For example, arctic nesting Rock Ptarmigans (*Lagopus mutus*) and Lapland Longspurs (*Calcarius lapponicus*) are only infected at the southern portion of the range where high latitude

forests provide habitat for arthropod vectors (Bennett et al. 1992). Although total abundance and species richness of dipteran vectors is high in neotropical regions, avian hematozoa are less prevalent in wintering migrant and resident birds (e.g. Garvin & Marra 1991; Rodriguez & Matta 2001; Valkiunas et al. 2003) because populations of competent vectors are lacking (Bennett et al. 1991; Garvin et al. 2004; Young et al. 1993). The larval stage of black flies, a vector of *Leucocytozoa*, require swift streams that are less abundant in neotropical than nearctic regions (Greiner et al. 1975; Scheuerlein & Ricklefs 2004). However, trypanosomes and *Plasmodium* tend to be more abundant in tropical parasite communities because they inhabit a wider variety of arthropod vectors (Bennett 1961; Votypka et al. 2002).

Seasonal Variation of Avian Hematozoa

Prevalence of avian hematozoa varies seasonally, typically being higher during breeding than non-breeding periods. For example, 94% of Gray Catbirds (*Dumetella carolinensis*) captured between 7-21 June in Ohio were infected with *Haemoproteus beckeri,* while 29% of catbirds captured from 22 July – 7 August were infected with *H. beckeri* (Garvin et al. 2003). In Florida, prevalence of hematatozoa in breeding Blue Jays decreased from 47% during June-July to 3% between November-January (Garvin & Greiner 2003b). These examples illustrate that geographic variation of hematozoan prevalence is exacerbated by seasonal fluctuations of prevalence. Seasonal variation of prevalence is also dictated by interactions between the environment, vectors, parasites, and avian hosts.

Co-adaptations between hematozoan parasites and avian hosts results in dramatic differences in prevalence between the breeding and non-breeding seasons. Hemosporidian infections can persist throughout an avian host's lifetime (Atkinson & Van Riper 1991). Most of the year, avian hosts are usually capable of immunologically suppressing infections so that parasites remain inactive within the visceral organs of the host (i.e. latent infection) (Atkinson & Van Riper 1991). When the immune system is compromised through environmental or nutritional stress, parasites are again able to reproduce in the host. Spring relapse, which occurs when a latent infection becomes patent, is thought to be induced by hormones that are associated with migratory or breeding condition (Allander & Sundberg 1997; Garvin et al. 2003; Valkiunas et al. 2004). Relapsing individuals provide a source of infection for immunologically naïve individuals during the summer. When birds are less nutritionally stressed after breeding, they are again able to suppress infection to a latent state until the next spring. Therefore, parasites are more prevalent during the breeding season when increased nutritional stress, immunosuppression, relapse, and vector abundance promotes increased transmission rates.

Since immunosuppression and relapse are influenced by stress, not every individual has the same probability of having gametocytes circulating in the blood stream. Avian biology thus causes prevalence to vary within a population. Experimental studies with food supplementation (Wiehn & Korpimaki 1998) or with clutch size manipulation (Ots & Horak 1996) demonstrate that prevalence is positively correlated with reproductive effort. Similarly, increased levels of reproductive (gonadal) hormones during the breeding season may explain the higher rates of infection in males (Deviche et

al. 2001b; Poulin 1996; Tarof et al. 1997) and in older individuals (Deviche et al. 2001b; Garvin et al. 2003). Alternatively, older individuals may have a higher proportion of infected individuals because they are exposed to more vectors over their lifespan (Deviche et al. 2001a; Sanz et al. 2001b; Weatherhead & Bennett 1991). However, other studies have found no association between prevalence and age (Garvin & Greiner 2003b; Ratti et al. 1993; Rintamaki et al. 1998) or sex (Davidar & Morton 1993; Rintamaki et al. 1998; Schrader et al. 2003).

Climate can also influence avian and vector ecology thus influencing prevalence. Seasonal and geographical variation of precipitation and temperature influence the occurrence of spring relapse by affecting arrival time and the onset of breeding in migratory birds. The subsequent infection of new avian hosts will ultimately depend upon vector abundance which is determined by habitat suitability (Atkinson & Van Riper 1991). Relapse, vector abundance, and decreased host immuncompetance, explains higher prevalence during the summer in temperate regions or during the wet season in tropical areas. Species-specific interactions between parasites and vectors may then influence seasonality of parasite genera within the breeding season. In northern Florida, transmission of *Leucocytozoa* is highest between April and June when simulids are more abundant; while *Plasmodium* transmission primarily occurs from August to December when mosquitoes are more abundant (Atkinson & Van Riper 1991).

Understanding the seasonal and geographic distribution of avian hematozoa is only possible when considering the interrelationships among parasites, vectors, birds, and the environment. Since these protozoan are obligate parasites, they cannot persist without the presence of both hosts and vectors. Avian hematozoa have evolved a

complicated life cycle that exploit both the high vector abundance and host susceptibility that occur during the breeding season. Avian hosts and arthropod vectors vary in abundance temporally and geographically relative to environmental conditions. Species-specific relationships between hosts, parasites, and vectors further influence the distribution of avian hematozoa. Spatial and temporal distributions also can be influenced by the hosts' behavioral response to potential disease causing parasites. When parasites negatively impact host fitness or reproductive success, natural selection and population regulation can occur. These evolutionarily and populationally significant questions warrant further investigation into the interactions between avian behavior and pathogenic parasites.

Avian Biology and Pathogenicity of Avian Hematozoa

Theoretical models have demonstrated that pathogenic parasites can retain high transmission rates while regulating host populations (Anderson & May 1978, 1979; May & Anderson 1978). Recent studies show that moderately pathogenic parasites may regulate host populations to a greater extent than non-pathogenic or highly pathogenic parasites (Hudson & Dobson 1991). Ultimately, parasite-mediated selection is dependant upon the ability of parasites to cause disease (i.e. pathogenicity). Although sexual selection hypotheses are beyond the scope of this study, research into the pathogenicity of avian hematozoa is vital to the understanding of host-parasite interactions.

Pathogenic parasites directly influence birds by lowering host condition or survival. Laboratory experiments demonstrate that avian hematozoa may cause gross

lesions, anemia, and anorexia in experimentally infected birds. Garvin et al. (2003) found evidence of tissue damage after schizogony in juvenile Blue Jays experimentally infected with *Haemoproteus danilewskyi*. In free-range captive Northern Bobwhites (*Colinus virginianus*), high intensity infections resulted in decreased host fitness and occasional mortality (Cardona et al. 2002). Infected birds exhibit anemia because gametocytes weaken, destroy, or cause the auto-immunological destruction of infected erythrocytes (Atkinson & Van Riper 1991; Garvin et al. 2003). Parasitization rarely influences body mass in captive experiments because food is supplied *ad libitum* (Allander & Sundberg 1997; Atkinson & Van Riper 1991; Garvin et al. 2003).

Parasite-mediated mortality and reduced host fitness is evident in some natural populations of breeding birds. By medicating Blue Tits (*Parus caeruleus*) with anti-malarial drugs, Merino et al. (2000) demonstrated that the body mass of parasitized nesting females was lower than that of unparasitized nesting females. Other studies have observed reduced body mass for parasitized Willow Warblers (*Phylloscopus trochilus*) (Bensch & Akesson 2003), American Kestrels (*Falco sparverius*) (Dawson & Bortolotti 2000), and Red-bellied Woodpeckers (*Melanerpes carolinus*) (Schrader et al. 2003) and lower survival rates for parasitized Purple Martins (*Progne subis*) (Davidar & Morton 1993), American Kestrels (Dawson & Bortolotti 2000), Collared Flycatchers (*Ficedula albicollis*) (Nordling et al. 1998), Red-bellied Woodpeckers (Schrader et al. 2003), and Rock Pigeons (*Columba livia*) (Sol et al. 2003). Similarly, intense infections are associated with reduced body mass in Willow Warblers and American Kestrels (Bensch & Akesson 2003; Dawson & Bortolotti 2000), and lower return rates of American Kestrels (Dawson & Bortolotti 2000). However, infected individuals often do not

directly suffer from parasitism in the wild (e.g. Deviche et al. 2001b; Ratti et al. 1993; Rintamaki et al. 2000) because they are able to regulate their body mass or survival at the expense of immediate reproductive success.

Infected parents may be able to mediate the effect of parasitism by trading reproductive success for health, or vice versa. Parental infection in Pied Flycatchers resulted in lower nestling body mass without influencing parental body mass (Merino et al. 1996). Male Pied Flycatchers infected with *Trypanosoma* spp. arrived 2 days later thereby reducing breeding success by an estimated 20% (Ratti et al. 1993). Female Pied Flycatchers infected with *Trypanosoma* had higher clutch desertion rates while females infected with *Haemoproteus balmorali* had lower hatching success (Sanz et al. 2001). By medicating Blue Tits with anti-malarial drugs, Merino et al. (2000) demonstrated that unmedicated parents suffered lower rates of fledgling success. Similarly vaccinated House Martins (*Delichon urbica*) had increased clutch size, hatching success, and fledging success (Marzal et al. 2005). A non-experimental study on Greater Sage-Grouse (*Centrocercus urophasianus*) demonstrated that females infected with *Leucocytozoa* fledged fewer young (Dunbar et al. 2003).

Parasitization is not always associated with reduced energetic fitness or breeding success. Genotypically or phenotypically superior hosts may experience greater levels of stress associated with increased reproductive effort. Because relapse may be induced by stress, individuals in better energetic condition before breeding may have higher infection rates (Bennett et al. 1988; Bensch & Akesson 2003; Dawson & Bortolotti 2000) with increased reproductive effort. Higher rates of infection are often associated with increased reproductive effort via early arrival (Moller et al. 2004), early egg laying dates

(Sanz et al. 2001), low food availability (Wiehn & Korpimaki 1998; Wiehn et al. 1999), or large broods (Davidar & Morton 1993; Ots & Horak 1996; Sanz et al. 2001).

Natural History of Neotropical Migrants

Neotropical migrant songbirds residing on wintering habitats in the Caribbean, Central America, and northern South America migrate north to temperate regions in North America (Moore 2000). More than one third of the of the annual life cycle is completed within stopover habitat (Moore 2000). Migrants typically depart winter habitats in late-February to early-March and arrive on breeding grounds late-May to early June. Competing energetic demands (e.g. provisioning young, pre-basic molt, and preparation for migration) cause the protraction of fall migration from mid-August to mid-October (Norris et al. 2004; Young et al. 1998). During spring migration, songbirds migrate farther and spend fewer days at stopover sites to arrive earlier on breeding territories (Young et al. 1998). Favorable stopover habitat is similar to favorable breeding habitat for most neotropical migrants (Petit 2000).

The focal species of this study, Magnolia Warbler, Yellow Warbler, and Myrtle Warbler, are migratory birds that differ in distribution and habitat preferences (Hall 1994; Hunt & Flaspohler 1998; Lowther et al. 1999). Magnolia Warblers typically breed and forage in dense second-growth coniferous stands of spruce (*Picea* spp.) or eastern hemlock (*Tsuga canadensis*) throughout the northeastern United States and east of the Canadian Rockies (Hall 1994). Yellow Warblers inhabit wet, deciduous thickets of willows (*Salix* spp.) and shrubland habitats throughout the United States and Canada

(excluding the southern United States) (Lowther et al. 1999). Myrtle Warblers breed in mature mixed coniferous-deciduous forests from the northeastern United States and Canada to Alaska (Hunt & Flaspohler 1998). Myrtle Warblers are able to migrate earlier because of their generalist diet and more northerly wintering range (Hunt & Flaspohler 1998).

Timing of Migration and Prevalence of Avian Hematozoa

Intraspecific variation in the timing of spring migration may be the result of age, sex, and phenotypic plasticity. Most male neotropical migrant songbirds including Barn Swallows (Saino et al. 2004), Wilson's Warblers (*Wilsonia pusilla*) (Young et al. 1998), and American Redstarts (*Setophaga ruticilla*) (Smith & Moore 2003) migrate earlier than females to acquire high quality breeding territories. Even within sex classes, early arrival increases reproductive performance by providing the opportunity for individuals to sequester high quality territories, acquire high quality mates, and complete nesting earlier (Drent et al. 2003; Smith & Moore 2005). Older individuals migrate earlier through experience and improved energetic condition (Drent et al. 2003; Smith & Moore 2003; Young et al. 1998). Early migration is risky and energetically costly because inclement weather in combination with low food abundance increases the risk of mortality (Drent et al. 2003; Ninni et al. 2004). Therefore, it is difficult to predict energetic condition from arrival date on the breeding grounds (Drent et al. 2003; Marra et al. 1998; Ninni et al. 2004; Smith & Moore 2003).

Individuals in poor energetic condition on the wintering grounds appear to have delayed departure schedules because they are less able to afford the cost of early migration (Drent et al. 2003; Marra et al. 1998). If migratory schedules are influenced by energetic condition, then infection from avian hematozoa may influence arrival of neotropical migrants on breeding territories. Male Pied Flycatchers parasitized by *Trypanosoma* (Ratti et al. 1993) and male Barn Swallows parasitized by *Haemoproteus prognei* (Moller et al. 2004) arrived later on breeding grounds. However, Davidar and Morton (1993) found that *H. prognei* infection had no effect on the arrival date of Purple Martins. Rintamaki et al. (1998) demonstrated that southern populations of Willow Warblers infected with *H. belopolskyi* migrated earlier than northern populations infected with *Leucocytozoa phylloscopus*.

Preliminary evidence suggests that prevalence of avian hematozoa during migratory stopover is typically lower than prevalence in breeding areas (Rintamaki et al. 1997; 1999; 1998; 2000; Smith et al. 2004). For example, 13% of spring migrating Redstarts (*Phoenicurus phoenicurus*) and 48% of breeding Redstarts were infected with *Leucocytozoa* or *Trypanosoma* (Rintamaki et al. 1999). *Haemoproteus, Leucocytozoa,* and *Trypanosoma* (about a 4:3:1 ratio) infected 18.6% of Willow Warblers (Rintamaki et al. 1998), 18.5% of European Robbins (*Erithacus rubecula*), 23.8% of Lesser Whitethroats (*Sylia curruca*), and 13.2% of Redstarts (Rintamaki et al. 1997) during spring migration in southern Finland. Smith et al. (2004) found that in New Mexico, 41% of hatch-year Sharp-shinned Hawks (*Accipiter striatus*) were infected with *Haemoproteus* and/or *Leucocytozoa* (about a 3:1 ratio). Further information about

prevalence, intensity, and condition of migrants is needed to fully assess the effect of hematozoa during migratory periods and throughout the avian lifecycle.

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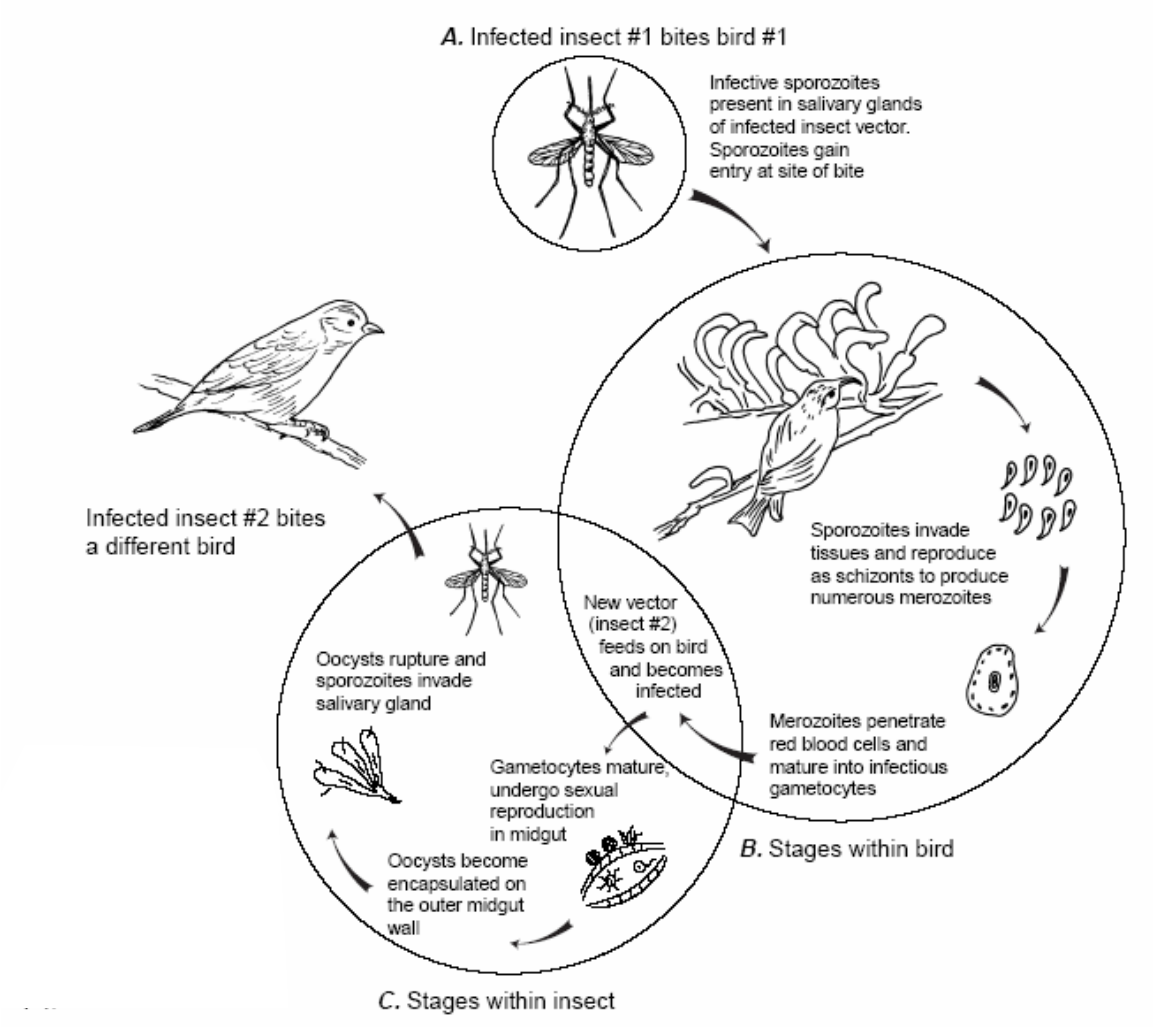

Figure 1.1. From Atkinson and Van Riper (1991). The complex general life cycle of hemosporidian parasites begins with (A), an infected insect biting a susceptible bird. Separate infectious and developmental stages occur in (B), the bird host, and (C) the insect vectors.

CHAPTER 2

QUANTIFICATION OF HEMATOZOA IN BLOOD SMEARS: A COMPARISON OF DIGITAL AND MANUAL TECHNIQUES

Until recently, avian hematozoa were detected and quantified from thin blood smears. Recent studies have demonstrated that Polymerase Chain Reaction (PCR) based analyses are up to ten times more likely to detect hematozoan infections than visual inspection of blood smears using microscopy (Durrant et al. 2006). Discrepancies between parasite detection using PCR and blood smear analyses indicate that prevalence cannot be accurately quantified from blood smears. Prevalence data from smear analysis would instead denote the presence of one or more parasites within the number of erythrocytes scanned (e.g. ≥1 parasite/ 100,000 erythrocytes). In addition, frequent false negatives from smear analysis (i.e. parasites not detected on blood smears when PCR-based analyses detect parasites) suggest that parasite densities may often be lower than previously suspected.

Fedynich et al. (1995) proposed that intensity of hematozoan infection (number of parasites per standard number of erythrocytes) is more ecologically relevant than presence or absence of hematozoa. Subsequent research has provided evidence that hematozoan intensity is inversely related to energetic condition (Dawson & Bortolotti

2000), return rates (Dawson & Bortolotti 2000), and reproductive success (Allandar & Bennett 1995; Marzal et al. 2005; Merino et al. 2000). However, infection intensity is infrequently quantified because manual erythrocyte counts require approximately 30 minutes per 2,000 erythrocytes (Godfrey Jr. et al. 1987). Methods quantifying parasites per 100 fields of view or parasites per unit scanning time (e.g. 10 minutes) have been shown to be imprecise and irreproducible (Godfrey Jr. et al. 1987); therefore intensity of infection can only be accurately quantified in relation to the number of erythrocytes scanned.

To alleviate tedious erythrocyte quantification, Gering and Atkinson (2004) devised an automated method to count erythrocyte nuclei from digital images using ImageJ software (National Institute of Health). However, intensity cannot be calculated using digital image analysis without first correcting for area differences between digital images and microscopic fields of view. Slide micrometers are not precise enough to accurately estimate area, but the ratio of microscope erythrocyte count to image erythrocyte count (hereafter the field:image ratio) can be estimated from repeated erythrocyte counts. Here I demonstrate that manual quantification of intensity of infection within 2,000 erythrocytes is less accurate than digital quantification of intensity within 50,000 erythrocytes. I contend that digital image analysis of blood smears may provide a relatively easy and accurate method to quantify infections of varying intensity.

Yellow-rumped Warblers (*Dendroica coronata*; n=209), Magnolia Warblers (*Dendroica magnolia*; n=204), and Yellow Warblers (*Dendroica petechia*; n=216) were captured using 2.6 x 12 meter mist in northwestern Ohio during the spring migratory period of 2004 and 2005 (16 April to 3 June). I used a sterile 27-gauge needle to

puncture the brachial vein and collect 10-30 μL of blood in prehepernized microhematocrit capillary tubes. A drop of blood was immediately placed on two clean glass slides to prepare blood smears. At the end of each day, blood smears were fixed in 100% methanol for 1 minute.

Blood smears were stained for 10 minutes with Jenner's Stain pH ~7 and 60 minutes with Giemsa Stain pH ~7 (The Ohio State University Reagent Lab, Columbus, OH, USA). An Olympus BX40F microscope mounted with a Spot RT Color digital camera (Diagnostics Instruments Inc., Sterling Heights, MI, USA) was used to examine blood smears at 1000x under oil immersion. Digital photographs of each examined field of view were taken using Spot Advanced software (automatic color balance, automatic exposure). To reduce spurious quantification of erythrocytes from particulate and other non-nucleic erythrocyte matter, images were taken with gain = 2 (compare Figure 2.1a and 2.1b). Because hematozoans may be heterogeneously distributed throughout blood smears (Godfrey Jr. et al. 1987), examination began at a randomly selected starting position where the smear formed a relatively homogeneous monolayer of cells. Parasites counts and image captures were conducted until at least 50,000 erythrocytes (150 to 175 fields of view) were scanned. Thirty-six slides (n=7 Magnolia Warblers; n=27 Yellow-rumped Warblers, n=2 Yellow Warbler) with at least 1 intracellular parasite (n=29 *Haemoproteus* spp., n=2 *Plasmodium spp*, n=5 unidentified *Haemoproteus* or *Plasmodium* spp.) were randomly selected for manual counts of erythrocytes and parasites within at least 2,000 erythrocytes (4 to 11 fields of view).

Quantification of nucleated erythrocytes from digital images was performed using ImageJ software (National Institute of Health) (Gering & Atkinson 2004). Images series

were imported (Figure 2.1b), converted to 8-bit formats, and subjected to automatic thresholding (Figure 2.1c) (Gering & Atkinson 2004). The "Analyze Particles" function was used to count erythrocyte nuclei as objects between 1000 and 4200 pixels in size (Figure 2.1d) (Gering & Atkinson 2004). Nuclei along edges of digital images were excluded to avoid overdispersion (Godfrey Jr. et al. 1987). Intensity of infection is reported as number of parasites per 2,000 erythrocytes (Godfrey Jr. et al. 1987).

Statistical analyses were performed using R Version 2.2.1 (R Development Core Team 2005). Field:image ratios were calculated for each of 36 slides by dividing manual erythrocyte counts by erythrocyte counts using ImageJ. Intensity of infection from image analysis was calculated using the equation: (# parasites) * 2000 / (# erythrocytes from images) / (mean field:image ratio). To test if field:image ratios could be consistently calculated from erythrocyte counts, slides were randomly divided into two groups and analyzed using a one-way ANOVA. A bootstrap analysis of mean field:image ratio was performed to calculate 95% confidence intervals for theoretical samples of 1 to 25 slides. Scaled differences of intensity (difference/mean) between manual erythrocyte quantification (2,000 erythrocytes ; n=32) and digital erythrocyte quantification (50,000 erythrocytes) were calculated for each slide to reduce heteroscedacity. A bootstrap analysis of scaled differences of intensity was used to determine if manually quantified intensity was different than digitally quantified intensity.

The mean field:image ratio for 36 slides was 2.99 (SE=0.05). Field:image ratios did not differ between randomly selected groups (Figure 2.2a; mean difference=0.14, SE=0.07, $F_{1, 34}$=2.15, P=0.15), thus demonstrating that erythrocytes from digital images can be consistently extrapolated to larger microscopic fields of view. Bootstrap analysis

of mean field:image ratios revealed that 95% confidence bounds approached asymptotic lines and ceased exponential reduction at approximately 10 slides (Figure 2.2b). Mean scaled difference of intensity from bootstrap analysis was -0.46 less than zero (P=0.02, 95% CI -0.91 to -0.02); therefore infection intensity from manual quantification of 2,000 erythrocytes was significantly lower than intensity from digital quantification of 50,000 erythrocytes.

I have provided evidence that field:image ratios can be consistently calculated from simultaneous digital and manual quantification of 2,000 erythrocytes from 10 slides (Figure 2.2a and b). Digital erythrocyte counts can then be used in conjunction with manual parasite counts to quantify intensity of hematozoan infection. Because intensity from manual counts of 2,000 erythrocytes was 0.46 times lower than intensity from digital counts of 50,000 erythrocytes (bootstrap P= 0.02), I argue that digital quantification offers a more accurate method to quantify intensity. I suggest that low intensity infections (e.g. <2 parasites per 2,000 erythrocytes) cannot be accurately quantified manually because heterogeneity often results in the absence of parasites (11 of 21 slides) or occasionally the overrepresentation of parasites within 2,000 erythrocytes (manual intensity – digital intensity > 1 for 5 of 21 slides). Digital quantification of 50,000 erythrocytes requires approximately the same amount of time necessary to manually count 2,000 erythrocytes (i.e. 30 to 40 minutes). Therefore, digital quantification of erythrocytes with simultaneous manual quantification of hematozoa in blood smears offers an accurate and repeatable means to quantify hematozoan intensity.

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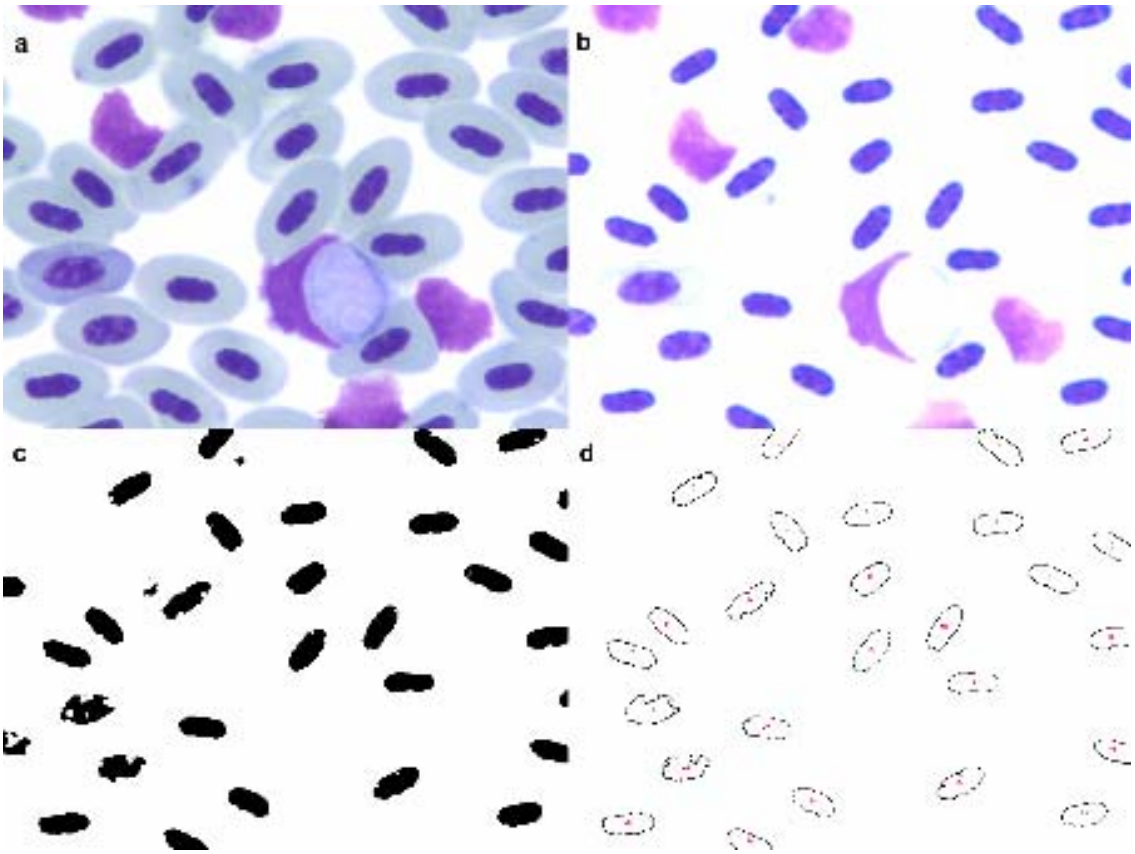

Figure 2.1. One quarter of a digital image from a single avian blood smear (1000x under oil immersion) demonstrating erythrocyte quantification using ImageJ software.
(a) image gain = 1, (b) image gain = 2, (c) image b after 8-bit conversion and automatic thresholding, (d) image c after "analyze particle" function identified erythrocyte nuclei between 1000 to 4200 pixels in size.

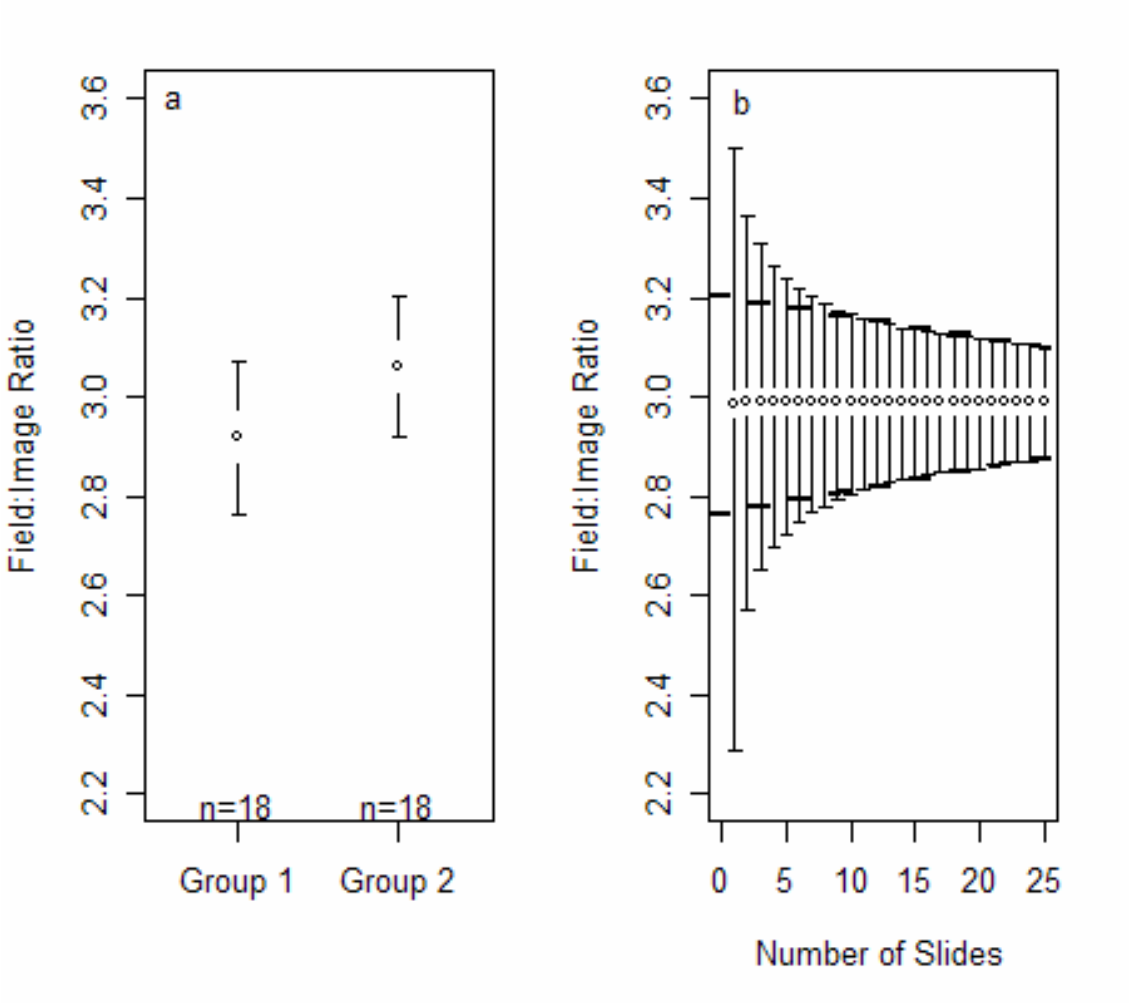

Figure 2.2. Mean field:image ratio (manual erythrocyte count/ image erythrocyte count) with 95% confidence intervals. (a) Mean field:image ratio was not different between randomly selected groups (mean difference=0.14, SE=0.07, $F_{1, 34}$=2.15, *P*= 0.15). (b) Bootstrap calculations across theoretical samples of 1 to 25 slides. Dashed lines approximate horizontal asymptotes.

CHAPTER 3

ENERGETIC CONDITION AND ARRIVAL TIMING ASSOCIATED WITH HEMATOZOAN INFECTION IN SPRING MIGRANT WOOD WARBLERS (PARULIDAE)

INTRODUCTION

Avian hematozoa are a phylogenetically and geographically diverse group of protozoans that utilize the blood stream for growth and reproduction (Atkinson & Van Riper 1991). The most prevalent order of these parasites (Hemosporidia) infect at least 68% of the world's species and 36% of North American birds (Atkinson & Van Riper 1991; Greiner et al. 1975). Current hematozoan research has attempted to determine if bright plumages provide a heritable and honest signal of individual health (e.g. hematozoan infection) and/or immunocompetence (Hamilton & Zuk 1982). The Hamilton-Zuk Hypothesis assumes that hematozoan infections are pathogenic to avian hosts. Ecological studies have subsequently demonstrated that hematozoan infection reduced passerine reproductive success via delayed clutch initiation in Great Tits (*Parus major*) (Allandar & Bennett 1995) and increased probability of clutch desertion in Pied Flycatchers (*Ficedula hypoleuca*) (Sanz et al. 2001). Similarly, hematozoan infection

was associated with reduced clutch size, hatching success, and fledging success in Blue Tits (*Parus caeruleus*) (Merino et al. 2000), Pied Flycatchers (*Ficedula hypoleuca*) (Sanz et al. 2001), and House Martins (*Delichon urbica*) (Marzal et al. 2005).

Despite extensive research into the epidemiology and pathogenicity of avian hematozoa, little work has been conducted on migrating birds. Preliminary evidence suggests that avian hematozoans are less prevalent during migratory periods than the breeding season (Garvin et al. 2003; Garvin et al. 2006; Rintamaki et al. 1999). Infection has not been found to differ between age and sex classes during spring migration (Rintamaki et al. 1999; Rintamaki et al. 1998; Smith et al. 2004) even though differences are often present throughout the breeding season (Deviche et al. 2001b; Garvin et al. 2003; Garvin & Greiner 2003b). Moreover, infection did not appear to be associated with reduced energetic condition in migrant Sharp-shinned Hawks (*Accipiter striatus*) (Smith et al. 2004) or migrant wood warblers captured in Louisiana (Garvin et al. 2006).

I suggest that hematozoan infection may not be associated with reduced energetic condition because migrant landbirds may mediate the energetic cost of infection through the modification of migratory schedules. Delayed or protracted migration would allow parasitized individuals to encounter phenologically advanced stopover sites that may have greater food availability (Drent et al. 2003; Ninni et al. 2004), thereby enabling parasitized individuals to meet higher energetic demands. Infected Barn Swallows (*Hirundo rustica*) (Moller et al. 2004) and Pied Flycatchers (Ratti et al. 1993) have been shown to arrive later on breeding grounds, but the effect of infection or intensity on the arrival timing of migrants on stopover sites has not yet been studied.

Hematozoan infection could also influence energetic condition prior to migrant departure and/or migrant refueling performance at stopover sites. Extrapolation of the Hamilton-Zuk Hypothesis (Hamilton & Zuk 1982) provides the argument that hematozoan infection during the non-breeding season could reduce energetic condition and result in a reduction in the extent of prealternate molt. Alternatively, hematozoan infection could reduce migrant landbirds' ability to forage *en-route*. Measurement of plasma lipid metabolite concentrations offers an analytical method to quantify refueling performance of migrating birds (Cerasale & Guglielmo 2006; Guglielmo et al. 2005; Zajac et al. 2006). Either scenario would result in late arrival via delayed departure (Norris et al. 2004) and/or longer stopover periods.

To determine the prevalence, intensity, and pathogenicity of avian hematozoa during spring migration I sampled Yellow-rumped Warblers (*Dendroica coronata*), Magnolia Warblers (*Dendroica magnolia*), and Yellow Warblers (*Dendroica petechia*) at stopover sites in northwestern Ohio. These three species are susceptible to hematozoan infection (Deviche et al. 2001a; Greiner et al. 1975; Weatherhead et al. 1991), abundant during spring migration, and represent both temperate and Nearctic-Neotropical migrants. By measuring the extent of prealternate molt, arrival date, energetic condition, and lipid metabolite concentrations, I will quantify past and present levels of fitness to elucidate the effects of hematozoa on migrating landbirds.

MATERIALS AND METHODS

Field Collection

Yellow-rumped Warblers (n = 519) and Magnolia Warblers (n = 385) were captured at stopover sites in Northwestern Ohio during the spring migratory period of 2004 and 2005 (11 April to 3 June). Yellow Warblers (n = 216) were captured during the same period, but only in 2004. To capture birds I used 2.6 x 12 m mist nets during the first 7 hours after sunrise but occasionally until dusk. Each bird was banded with an individually numbered USGS aluminum leg band before age and sex was determined using methodologies described by Pyle et al. (1987). Time, day, and location of capture were recorded. Metatarsus, unflattened wing chord, and mass were measured with calipers (0.01 mm accuracy), wing rules (0.5 mm accuracy), and digital scales (0.1 g accuracy), respectively. Approximately 15 µL of blood were collected via brachial veinipuncture and a drop of blood was immediately placed on two clean glass slides to prepare blood smears. In 2005, 40 to 60 µL of blood were collected within 10 min. of capture for future quantification of plasma lipid metabolites. At the end of each day, blood smears were fixed in 100% methanol for 1 min. and whole blood was centrifuged at 2,000x g for 10 min. before storing plasma at -80°C in liquid nitrogen.

In 2005, the extent of breast streaking (i.e. prealternate molt) was quantified for Magnolia and Yellow-rumped Warblers using digital photography and ImageJ software (National Institute of Health) (Gering & Atkinson 2004). Individuals were held in an upright posture with the wingtips, tail, and tibia tarsus between the index and middle fingers. Digital photographs were taken in front of a white background and adjacent to a

metric ruler at a distance of 0.5 m. Photographs were imported into ImageJ, adjusted to a standard size, and cropped to isolate breast feathers. Breast streaking was quantified using the "analyze particle" function of ImageJ to count the number of pixels with a "threshold" value less than 70 (Gering & Atkinson 2004).

Laboratory Analysis

Blood smears were stained with Jenner's Stain pH ~7 (10 min.) and Giemsa Stain pH ~7 (60 min.). Smears were digitally photographed (Spot RT Color, Diagnostics Instruments Inc.) and examined at 1000x under oil immersion. Examination began at a randomly selected starting point and proceeded lengthwise until at least 50,000 erythrocytes (150 to 175 fields) were intensively scanned for hematozoan parasites. Avian erythrocytes were quantified from digital photographs using ImageJ (Gering & Atkinson 2004). Intensity of infection (i.e. number of parasites per 2000 erythrocytes) was calculated from manual parasite counts and digital erythrocyte counts. For the purposes of this study, individuals for which no parasites were seen are reported as "uninfected", though infection may occur at undetectable levels (Kirkpatrick & Suthers 1988).

To determine refueling performance from plasma lipid metabolites, plasma triglycerides concentrations were assayed in 400 μL flat-bottomed wells using a microplate spectrophotometer (Guglielmo et al. 2005; Guglielmo et al. 2002). Four μL of plasma, 240 μL of Trinder reagent A (SIGMA, USA), and 40 μL of Trinder reagent B (SIGMA, USA) were used to sequentially measure glycerol and total triglyceride by end point assay as described by Guglielmo et al. (2005; 2002).

Statistical Analysis

Statistical analyses were performed using R Version 2.2.1 (R Development Core Team 2005). Principle component analysis (PCA) of tarsus and wing length was used to depict body size of Yellow-rumped, Magnolia, and Yellow Warblers. Energetic condition scores (Condition) were created from the residuals of weight regressed against PCA scores of body size. Capture date was centered for each year to create an arrival index (Day) that would remove mean arrival differences. Intensity of infection was natural log-transformed to improve normality. Multiple regression was used to model the effects of Year (where 2004 = 0 and 2005 =1), Day, Condition, Age (where after second year = 1 and second year = 0) and Sex (where Male = 1 and Female = 0) on ln(intensity) of *Haemoproteus* spp. and *Plasmodium* spp. infection. Logistic regression (log-log link) was used to model the probability of infection (regardless of parasite genera) in relation to Day, Condition, Age, and Sex.

I used an information theoretic approach to evaluate the relative effect of arrival Day, Condition, Age, and Sex on intensity or probability of infection (Burnham & Anderson 2002). Model selection was performed using Akaike's Information Criterion (AIC). Analyses were conducted separately for Magnolia, Yellow-rumped, and Yellow Warblers to accommodate separate life history characteristics. An *a priori* set of nested candidate models (n = 20) was created under the full model: Year + Condition*Age*Sex + Day*Age*Sex (where D*A*S denotes D + A + S + D*A + A*S + D*S +D*A*S). I reasoned *a priori* that the effect of Condition and Day on intensity or probability of infection would not be appreciably different between years, therefore Year was not included as an interaction term. An interaction between Day and Condition was not

included (post hoc $r < 0.17$ for all species) because I believed that migrant mass exhibits consistent variability throughout migration. Year was included in all models as an indicator variable to account for annual variation. Model sets were minimized by applying interaction terms to all variables when interaction terms were included in the model. A null model was created by modeling the response variable against year and a random number (1 to 100). Models with $\Delta AIC_c < 7$ were averaged and the relative variable importance (RVI) was calculated to obtain multimodel inference (Burnham & Anderson 2002).

Breast streaking and triglyceride levels could not be included in the aforementioned AIC analysis because these measurements were taken in 2005 only. To determine if breast streaking (Streak) was related to probability and intensity of infection, a one-way ANOVA was performed to compare the most parsimonious model from AIC against a model containing the additional term Age*Sex*Streak. A linear mixed-effects model was used to evaluate the effect of intensity on plasma triglyceride concentrations (Gillies et al. 2006). Triglyceride concentrations were regressed by residuals from AIC model averaging while capture location was modeled as a random effect to account for variation between sites (Guglielmo et al. 2005).

RESULTS

A total of 518 Yellow-rumped Warblers, 383 Magnolia Warblers, and 205 Yellow Warblers were sampled during spring 2004 and 2005. Prevalence of avian hematozoa in Magnolia, Yellow-rumped, and Yellow Warblers during stopover were 36.3%, 58.3%,

12.7% respectively (Table 3.1). Most infected individuals were parasitized by *Haemoproteus* spp. (63.2%) or *Plasmodium* spp. (11.8%) which together comprised 78.6% of all infections. Prevalence of *Haemoproteus* spp. and *Trypanosoma* spp. were significantly different between Magnolia Warblers (10.7% and 9.7% respectively), Yellow-rumped Warblers (47.3% and 5.4%), and Yellow Warblers (4.4% and 0.5%) (Fisher Exact $P<0.01$ and $P<0.05$ respectively, Table 3.1). *Plasmodium* spp. infected fewer Yellow-rumped Warblers (2.1%) than Magnolia (8.9%) or Yellow Warblers (4.4%) (Fisher's Exact $P<0.01$), while *Leucocytozoon* spp. infected fewer Yellow Warblers (0.0%) than Magnolia (8.9%) and Yellow-rumped Warblers (5.8%) (Fisher's Exact $P<0.01$, Table 3.1).

Intensity and probability of infection in Yellow-rumped Warblers were most parsimoniously explained by models containing Year, Condition, Day, Age, and Sex as explanatory variables (Akaike Weight $\omega_i=0.82$ and $\omega_i=0.48$ respectively, Table 3.2). However, the average linear regression model revealed that 95% confidence intervals encompassed zero for both Year ($\beta=0.45$, SE=0.25) and Sex ($\beta=-0.21$, SE=0.28), despite high relative variable importance for the latter (RVI=0.69). Therefore, intensity of Yellow-rumped Warbler infection is best explained by Condition ($\beta=-0.27$, SE=0.12, RVI=1.00), Day ($\beta=0.13$, SE=0.03, RVI=1.00), and Age ($\beta=-1.23$, SE=0.34, RVI=1.00) ($R^2 = 0.29$, Table 3.3, Figures 3.1 and 3.2). The average logistic regression model resulted in 95% confidence intervals for Condition ($\beta=-0.10$, SE=0.06, RVI=0.98), Age ($\beta=-0.03$, SE=0.14, RVI=0.98), and Sex (logistic $\beta=-0.03$, SE=0.14, RVI= 0.82) encompassing zero (Table 3.3). Prevalence in Yellow-rumped Warblers is therefore best

explained by year (β=0.77, SE=0.16) and arrival date (β=0.13, SE=0.01, RVI=0.98) (Table 3.3, Figure 3.3).

Intensity and prevalence of infection in Magnolia and Yellow Warblers were not related to energetic condition, capture date, or sex of the individual. Candidate models for Magnolia Warblers with Δ AIC_c values less than seven included six models for intensity and six models for prevalence resulting in comparatively low Akaike weights (intensity $\omega_i \leq 0.38$, prevalence $\omega_i \leq 0.30$, Table 3.4). Similarly, candidate models for Yellow Warblers resulted in low Akaike weights (intensity $\omega_i \leq 0.58$, prevalence $\omega_i \leq 0.26$, Table 3.5) because five intensity models and nine prevalence models possessed Δ AIC_c values less than seven. Furthermore, high variability resulted in 95% confidence intervals encompassing zero for Condition, Day, and Sex (Tables 3.6 and 3.7). Second year Magnolia Warblers tended to have more intense infections than older individuals (β=−0.98, SE=0.52, RVI=0.98). Conversely, after second year Magnolia Warblers were more likely to be infected than second year individuals (β=0.69, SE=0.19, RVI=0.98), whereas after second year Yellow Warblers were less likely to be infected than second year individuals (β=−1.35, SE=0.59, RVI=1.00).

Breast streaking and triglyceride concentrations were not associated with intensity or probability of infection in either Magnolia or Yellow-rumped Warblers. Linear and logistic regression models including Streak*Age*Sex were not significantly better than models with the lowest AIC score (ANOVA $P>0.1$). Residuals from linear and logistic model averaging did not explain triglyceride concentrations in Yellow-rumped Warblers despite accounting for capture location ($P=0.80$ and $P=0.85$ respectively). Likewise, prevalence was not associated with triglyceride levels in Magnolia Warblers ($P=0.88$).

The relationship between infection intensity and triglyceride concentrations in Magnolia Warblers could not be determined because only 5 of 21 individuals were infected with *Haemoproteus* or *Plasmodium*.

DISCUSSION

This study documents prevalence and intensity of hematozoans at northerly stopover sites and provides evidence that for some landbird species, relapse or transmission may occur while birds are actively migrating. In northwestern Ohio, prevalence of avian hematozoa for three species of wood warblers during migratory stopover (12% to 58%) was higher than prevalence than for migrating wood warblers at stopover sites in coastal Louisiana (4% to 26%) (Garvin et al. 2006). Spring relapse could explain higher prevalence in northwestern Ohio because both migration and relapse are associated with elevated stress (Navarro et al. 2004; Valkiunas et al. 2004) and breeding hormones (Deviche & Parris 2006; Valkiunas et al. 2004). Alternatively, transmission may occur at southerly stopover sites when vectors of *Haemoproteus* become abundant after February (Atkinson et al. 1988; Garvin & Greiner 2003a). A better understanding of the ecology of vectors and their avian hosts is needed to determine whether *en-route* transmission or spring relapse causes elevated prevalence at northerly stopover sites.

Total prevalence differed significantly among Magnolia (36.3%), Yellow-rumped (58.3%), and Yellow Warblers (12.7%) captured in northwestern Ohio. Interspecific differences in total prevalence were largely due to differences in the prevalence of

Haemoproteus among avian species (Table 3.1). Because migrant warblers migrate in close geographic proximity to conspecifics (Petit 2000), I believe that genera-specific differences in prevalence were not to due *en-route* transmission. The finding that *Plasmodium* was more prevalent in Yellow Warblers (4.9%) than Yellow-rumped Warblers (2.1%) (Table 3.1) would reflect differences in prevalence during the breeding season if infection at stopover sites resulted from spring relapse. Because Yellow Warblers typically breed in a variety of deciduous, shrub-dominated habitats near water (Lowther et al. 1999) while Yellow-rumped Warblers breed in mature mixed coniferous-deciduous forests (Hunt & Flaspohler 1998), Yellow warblers should be exposed to higher densities of *Plasmodium*-infected mosquitoes (*Aedes* spp. and *Culex* spp.) (Atkinson & Van Riper 1991). Similarly, breeding habitat preferences would result in Magnolia Warblers (Hall 1994) and Yellow-rumped Warblers (Hunt & Flaspohler 1998) being exposed to higher densities of *Leucocytozoa* and *Trypanosoma*-infected black flies (*Simulim* spp.) (Atkinson & Van Riper 1991) than Yellow Warblers (Lowther et al. 1999). These habitat preferences during the breeding season explain my finding that *Leucocytozoa* and *Trypanosoma* were more prevalent in Magnolia and Yellow-rumped Warblers than Yellow Warblers (Table 3.1).

Unlike previous studies of migrant wood warblers (Garvin et al. 2006), hematozoan infections differed between second year and after second year individuals for each of the three species I studied (Tables 3.3, 3.6, and 3.7). Second year individuals were more heavily parasitized than after second year Magnolia and Yellow-rumped Warblers. Younger birds may experience heightened stress levels during their first northward migration and this may advance the timing of relapse and increase the

intensity of hematozoan infections (Valkiunas et al. 2004). Higher prevalence in after second year Magnolia Warblers may simply be the result of increased exposure to vectors throughout numerous breeding seasons (Deviche et al. 2001b). In Yellow Warblers, the presence of breeding individuals and low infection rates (26 of 205 individuals sampled) would likely lead to spurious conclusions about age-related prevalence.

More importantly, results from this study indicate that avian hematozoa are pathogenic for some species of migrating songbirds. Energetic condition and capture date were negatively related to intensity of infection in Yellow-rumped Warblers (Tables 3.2 and 3.3). Prevalence was associated with later arrival of migrant Yellow-rumped Warblers but was not related to energetic condition. These results suggest that infected migrants could delay the initiation of migration or extend the migratory period to preserve their energetic condition. Because reproductive performance was reduced for American Redstarts (*Setophaga ruticilla*) (Norris et al. 2004; Smith & Moore 2005) and Pied Flycatchers (Ratti et al. 1993) arriving later on breeding grounds, this study provides evidence that migratory events could influence parasitization, energetic condition, or reproductive performance during the subsequent breeding period.

My results provide little explanation about why hematozoa are pathogenic for some species of migrating wood warblers and not others. Susceptibility in terms of prevalence and pathological effects may be ecologically or genetically regulated. Early migrants such as the Yellow-rumped Warbler may not be able to afford the costs of parasitization or immunosuppression if they migrate before temperate stopover sites have developed phenologically. Similarly, migrants could experience more intense infections and deleterious consequences if individuals are exposed to new infections in the southern

United States prior to migratory departure (Atkinson & Van Riper 1991; Hunt & Flaspohler 1998). However, previous studies have suggested that some avian species may simply be more genetically resistant or susceptible to hematozoan infection (Deviche et al. 2001a; Tarof et al. 1997). Studies encompassing a broader variety of avian species and stopover areas are needed to elucidate pathological differences amongst avian hosts.

Recent research on the ecology of neotropical migrant landbirds has focused on the importance of considering a variety of factors that influence migrants at different stages of their annual life cycles (Moore 2000). Declines of neotropical migrant populations have been attributed to the loss of wintering, stopover, and breeding habitat (Moore 2000). Loss of habitat may force neotropical migrants into suboptimal habitats where they are exposed to increased predation risks, increased competition, and reduced food availability (Marra et al. 1998; Petit 2000). Migrant Barn Swallows (Saino et al. 2004) and American Redstarts (Marra et al. 1998) that used suboptimal winter habitat were shown to be in poorer energetic condition and departed later from wintering grounds (Marra et al. 1998; Saino et al. 2004). While hematozoan infection was not associated with the extent of prealternate molt in this study, individuals using suboptimal wintering habitat may be more susceptible to spring relapse. Because prevalence and intensity of hematozoan infection increases with predation risk (Navarro et al. 2004), stress (Valkiunas et al. 2004), and reproductive effort (Wiehn & Korpimaki 1998), avian hematozoa may compound the deleterious effects of habitat loss by delaying migration thereby reducing reproductive performance.

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Parasite Genera	Magnolia Warbler	Yellow-rumped Warbler	Yellow Warbler	Total
Haemoproteus spp.	**10.7%**	**47.3%**	**4.4%**	26.7%
Plasmodium spp.	8.9%	**2.1%**	4.9%	5.2%
Haemoproteus spp. or *Plasmodium* spp.	20.9%	51.5%	9.8%	33.4%
Leucocytozoa spp.	8.9%	5.8%	**0.0%**	5.9%
Trypanosoma spp.	**9.7%**	**5.4%**	**0.5%**	6.0%
Microfilaria spp.	0.3%	0.6%	0.0%	0.4%
Multiple Infections	5.0%	7.3%	0.0%	5.2%
Unknown	2.1%	3.1%	1.0%	2.4%
Total Prevalence	**36.3%**	**58.3%**	**12.7%**	42.2%
Sample Size	383	518	205	1106

Table 3.1. Prevalence of avian hematozoa in Magnolia, Yellow-rumped, and Yellow Warblers during migration in northwestern Ohio, late April–May of 2004 and 2005. Percentages in bold are significantly different from other species based on pairwise Fisher's Exact tests ($P<0.05$).

Intensity				Prevalence			
Model	K	Δ AICc	ω_i	Model	K	Δ AICc	ω_i
Y + C + D + A + S	**7**	**0.00**	**0.48**	**Y + C + D + A + S**	**6**	**0.00**	**0.82**
Y + C*A + D*A	**8**	**1.30**	**0.25**	Y + C + D + A	5	3.34	0.15
Y + C*A*S + D*A*S	**14**	**1.65**	**0.21**	Y + C*A + D*A	7	7.28	0.02
Y + C + D + A	6	4.19	0.06	Y + C*A*S + D*A*S	13	12.05	0.00
Y + C + A + S	6	36.11	0.00	Y + D + A + S	5	49.29	0.00
Y + C*A	6	52.66	0.00	Y + D + A	5	52.39	0.00
Y + C + A	5	53.43	0.00	Y + C + A + S	4	52.46	0.00
Y + C + D + S	6	92.38	0.00	Y + D*A	5	54.29	0.00
Y + C*S + D*S	8	94.24	0.00	Y + C + D + S	5	56.24	0.00
Y + C + D	5	106.51	0.00	Y + C*S + D*S	7	58.86	0.00
Y + D + A + S	6	107.56	0.00	Y + C + D	4	65.37	0.00
Y + D + A	5	112.71	0.00	Y + C*A	5	70.16	0.00
Y + D*A	6	112.77	0.00	Y + C + A	4	70.53	0.00
Y + C*S	6	131.29	0.00	Y + D + S	4	105.51	0.00
Y + C + S	5	131.32	0.00	Y + D*S	5	107.03	0.00
Y + C	4	153.56	0.00	Y + C + S	4	109.85	0.00
Y + D + S	5	202.80	0.00	Y + C*S	5	111.88	0.00
Y + D*S	6	202.87	0.00	Y + D	3	114.84	0.00
Y + D	4	216.77	0.00	Y + C	3	136.28	0.00
Y + Random	4	265.44	0.00	Y + Random	3	195.35	0.00

Table 3.2. Akaike Information Criterion results for Yellow-rumped Warblers with K = number of parameters in model and ω_i = Akaike weight. Models based on linear regression of natural log infection intensity (Intensity) and logistic regression of infection status (Prevalence) against independent variables with Y = year, C = condition index, D = date of first capture centered by year, S = sex (male = 1, female = 0), and A = age (after second year = 1, second year = 0). Models with delta AIC_c scores < 2 are shown in bold text.

	Intensity			Prevalence		
Variable	Beta hat	95% CI	RVI	Beta hat	95% CI	RVI
Intercept	2.17	1.50 , 2.83		-0.64	-1.02 , -0.26	
Year	0.45	-0.05 , 0.95		**0.77**	**0.45 , 1.09**	
Cond	**-0.27**	**-0.51 , -0.04**	**1.00**	-0.10	-0.22 , 0.02	0.98
Day	**0.13**	**0.08 , 0.19**	**1.00**	**0.09**	**0.07 , 0.12**	**0.98**
Age	**-1.23**	**-1.91 , -0.55**	**1.00**	-0.03	-0.33 , 0.27	0.98
Sex	-0.21	-0.75 , 0.34	0.69	0.01	-0.27 , 0.29	0.82
Cond:Age	0.27	-0.38 , 0.92	0.46			
Day:Age	-0.05	-0.17 , 0.07	0.46			
Cond:Sex	-0.02	-0.13 , 0.10	0.21			
Age:Sex	-0.04	-0.36 , 0.27	0.21			
Day:Sex	0.01	-0.04 , 0.06	0.21			
Cond:Age:Sex	-0.14	-0.67 , 0.39	0.21			
Day:Age:Sex	0.01	-0.05 , 0.08	0.21			

Table 3.3. Yellow-rumped Warbler coefficients (Beta hat), 95% confidence intervals (95% CI), and relative variable importance (RVI) based on averaging linear regression (Intensity) and logistic regression (Prevalence) models with Δ $AIC_c < 7$.

Intensity				Prevalence			
Model	K	Δ AIC_c	ω_i	Model	K	Δ AIC_c	ω_i
Y + C + A	**5**	**0.00**	**0.38**	**Y + C + A**	**4**	**0.00**	**0.30**
Y + C + A + S	**6**	**1.59**	**0.17**	**Y + C + D + A**	**5**	**0.47**	**0.24**
Y + C + D + A	**6**	**1.95**	**0.14**	**Y + C + A + S**	**5**	**1.07**	**0.18**
Y + C*A	6	2.30	0.12	**Y + C*A**	**5**	**1.89**	**0.12**
Y + C + D + A + S	7	3.09	0.08	**Y + C + D + A + S**	**6**	**1.98**	**0.11**
Y + C*A + D*A	8	3.10	0.08	Y + C*A + D*A	7	3.18	0.06
Y + D*A	6	8.58	0.01	Y + C*A*S + D*A*S	13	12.72	0.00
Y + C + S	5	10.32	0.00	Y + D + A	4	14.67	0.00
Y + D + A	5	10.42	0.00	Y + D*A	5	14.96	0.00
Y + C + D + S	6	10.70	0.00	Y + D + A + S	5	15.13	0.00
Y + D + A + S	6	10.81	0.00	Y + C + S	4	27.53	0.00
Y + C*S	6	11.92	0.00	Y + C*S	5	29.56	0.00
Y + C	4	12.26	0.00	Y + C + D + S	5	29.58	0.00
Y + C + D	5	13.22	0.00	Y + C	3	29.71	0.00
Y + C*A*S + D*A*S	14	14.13	0.00	Y + C + D	4	31.69	0.00
Y + C*S + D*S	8	14.50	0.00	Y + C*S + D*S	7	33.70	0.00
Y + D + S	5	18.99	0.00	Y + D + S	4	42.11	0.00
Y + D*S	6	20.40	0.00	Y + D*S	5	44.14	0.00
Y + D	4	22.15	0.00	Y + Random	3	45.84	0.00
Y + Random	4	22.40	0.00	Y + D	3	46.07	0.00

Table 3.4. Akaike Information Criterion results for Magnolia Warblers with K = number of parameters in model and ω_i = Akaike weight. Models based on linear regression of natural log infection intensity (Intensity) and logistic regression of infection status (Prevalence) against independent variables with Y = year, C = condition index, D = date of first capture centered by year, S = sex (male = 1, female = 0), and A = age (after second year = 1, second year = 0). Models with delta AIC_c scores < 2 are shown in bold text.

Intensity				Prevalence			
Model	K	Δ AICc	ω_i	Model	K	Δ AICc	ω_i
C + A	**4**	**0.00**	**0.58**	**C + A**	**3**	**0.00**	**0.26**
C + A + S	5	3.83	0.09	**C + A + S**	**4**	**0.63**	**0.19**
C*A	5	3.85	0.08	**C + D + A**	**4**	**1.63**	**0.12**
D + A	4	3.91	0.08	**C*A**	**4**	**1.78**	**0.11**
C + D + A	5	4.11	0.07	**D + A**	**3**	**1.83**	**0.10**
C	3	5.57	0.04	C + D + A + S	5	2.16	0.09
D*A	5	7.18	0.02	D + A + S	4	2.53	0.07
D + A + S	5	8.01	0.01	D*A	4	3.77	0.04
Random	3	8.56	0.01	C*A + D*A	6	5.33	0.02
C + D	4	8.59	0.01	C*A*S + D*A*S	12	10.72	0.00
C + D + A + S	6	8.75	0.01	D	2	25.85	0.00
C + S	4	8.82	0.01	C + D	3	26.21	0.00
D	3	10.32	0.00	D + S	3	26.34	0.00
C + D + S	5	12.34	0.00	C	2	26.35	0.00
C*S	5	12.54	0.00	C + S	3	26.45	0.00
D + S	4	13.57	0.00	C + D + S	4	26.61	0.00
C*A + D*A	7	14.80	0.00	Random	2	28.07	0.00
D*S	5	17.10	0.00	D*S	4	28.27	0.00
C*S + D*S	7	21.11	0.00	C*S	4	28.53	0.00
C*A*S + D*A*S	10	39.90	0.00	C*S + D*S	6	30.71	0.00

Table 3.5. Akaike Information Criterion results for Yellow Warblers with K = number of parameters in model and ω_i = Akaike weight. Models based on linear regression of natural log infection intensity (Intensity) and logistic regression of infection status (Prevalence) against independent variables with Y = year, C = condition index, D = date of first capture centered by year, S = sex (male = 1, female = 0), and A = age (after second year = 1, second year = 0). Models with delta AIC_c scores < 2 are shown in bold text.

	Intensity			Prevalence		
Variable	Beta hat	95% CI	RVI	Beta hat	95% CI	RVI
Intercept	2.94	1.91 , 3.97		-1.31	-1.66 , -0.96	
Year	0.12	-0.92 , 1.15		**0.55**	**0.19 , 0.91**	
Cond	0.34	-0.47 , 1.16	0.98	-0.02	-0.32 , 0.28	1.00
Age	-0.98	-2.01 , 0.05	0.98	**0.69**	**0.32 , 1.05**	**1.00**
Day	0.00	-0.04 , 0.05	0.31	0.01	-0.02 , 0.03	0.41
Sex	0.13	-0.41 , 0.67	0.25	-0.02	-0.17 , 0.13	0.29
Cond:Age	-0.01	-0.34 , 0.33	0.20	0.01	-0.11 , 0.14	0.18
Day:Age	0.01	-0.04 , 0.07	0.08	0.00	-0.01, 0.01	0.06

Table 3.6. Magnolia Warbler coefficients (Beta hat), 95% confidence intervals (95% CI), and relative variable importance (RVI) based on averaging linear regression (Intensity) and logistic regression (Prevalence) models with $\Delta AIC_c < 7$.

	Intensity			Prevalence		
Variable	Beta hat	95% CI	RVI	Beta hat	95% CI	RVI
Intercept	1.89	0.85 , 2.93		-1.69	-2.30 , -1.08	
Age	0.68	-1.29 , 2.65	0.90	**-1.35**	**-2.51 , -0.20**	**1.00**
Cond	1.37	-0.27 , 3.00	0.86	-0.05	-0.33 , 0.23	0.13
Day	0.01	-0.03 , 0.05	0.16	0.31	-0.38 , 1.00	0.78
Sex	0.04	-0.23 , 0.30	0.09	0.01	-0.03 , 0.05	0.44
Cond:Age	0.07	-0.42 , 0.56	0.08	0.00	-0.01 , 0.01	0.06
Day:Age				0.12	-0.39 , 0.62	0.35

Table 3.7. Yellow Warbler coefficients (Beta hat), 95% confidence intervals (95% CI), and relative variable importance (RVI) based on averaging linear regression (Intensity) and logistic regression (Prevalence) models with Δ $AIC_c < 7$.

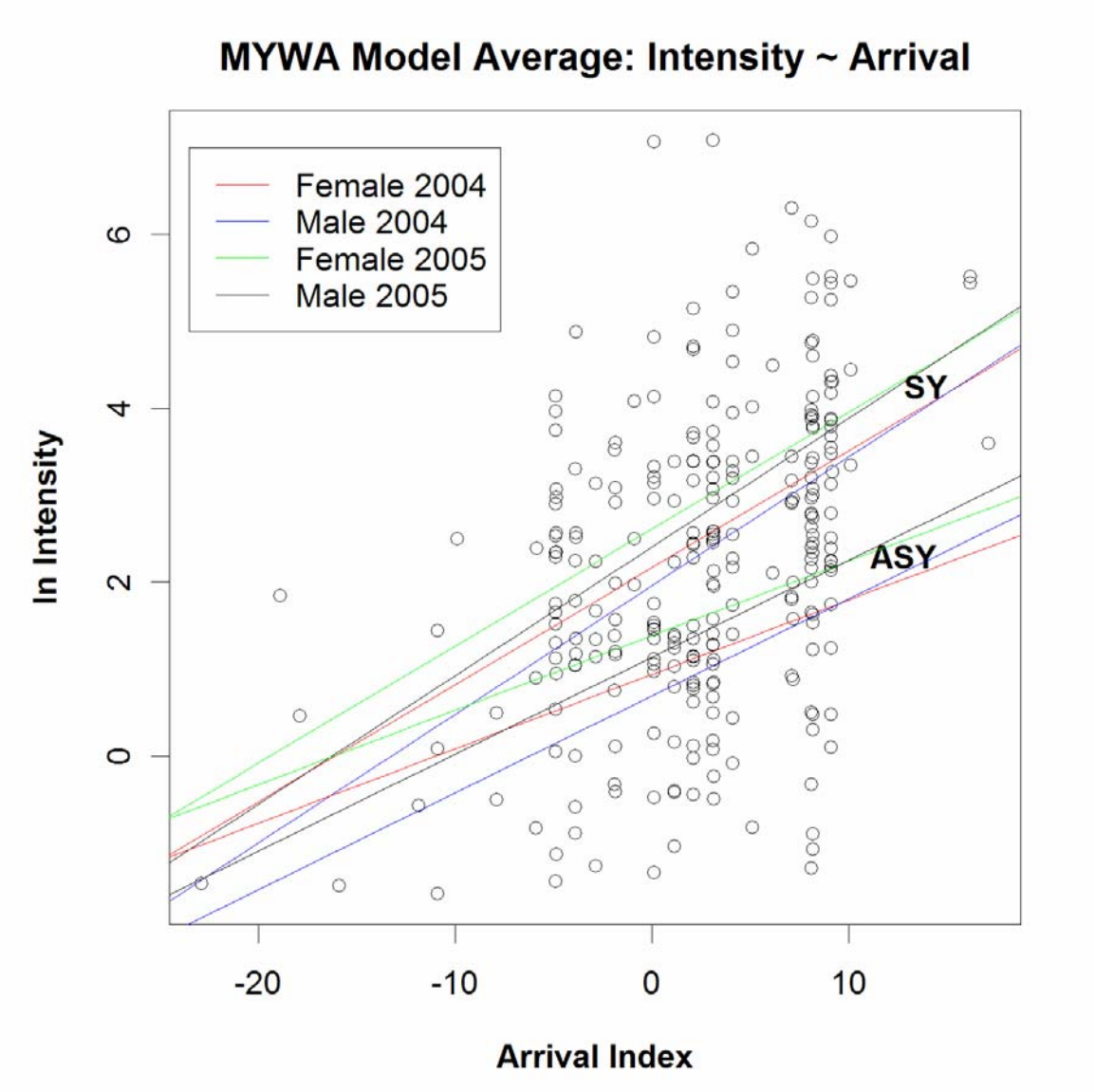

Figure 3.1. Average Yellow-rumped Warbler linear regression model when condition index equals zero. ln(Intensity) = 2.17 + 0.45*Year + 0.13*Day – 1.23*Age – 0.21*Sex – 0.05*Day*Age – 0.04*Age*Sex + 0.01*Day*Sex + 0.01*Day*Age*Sex.

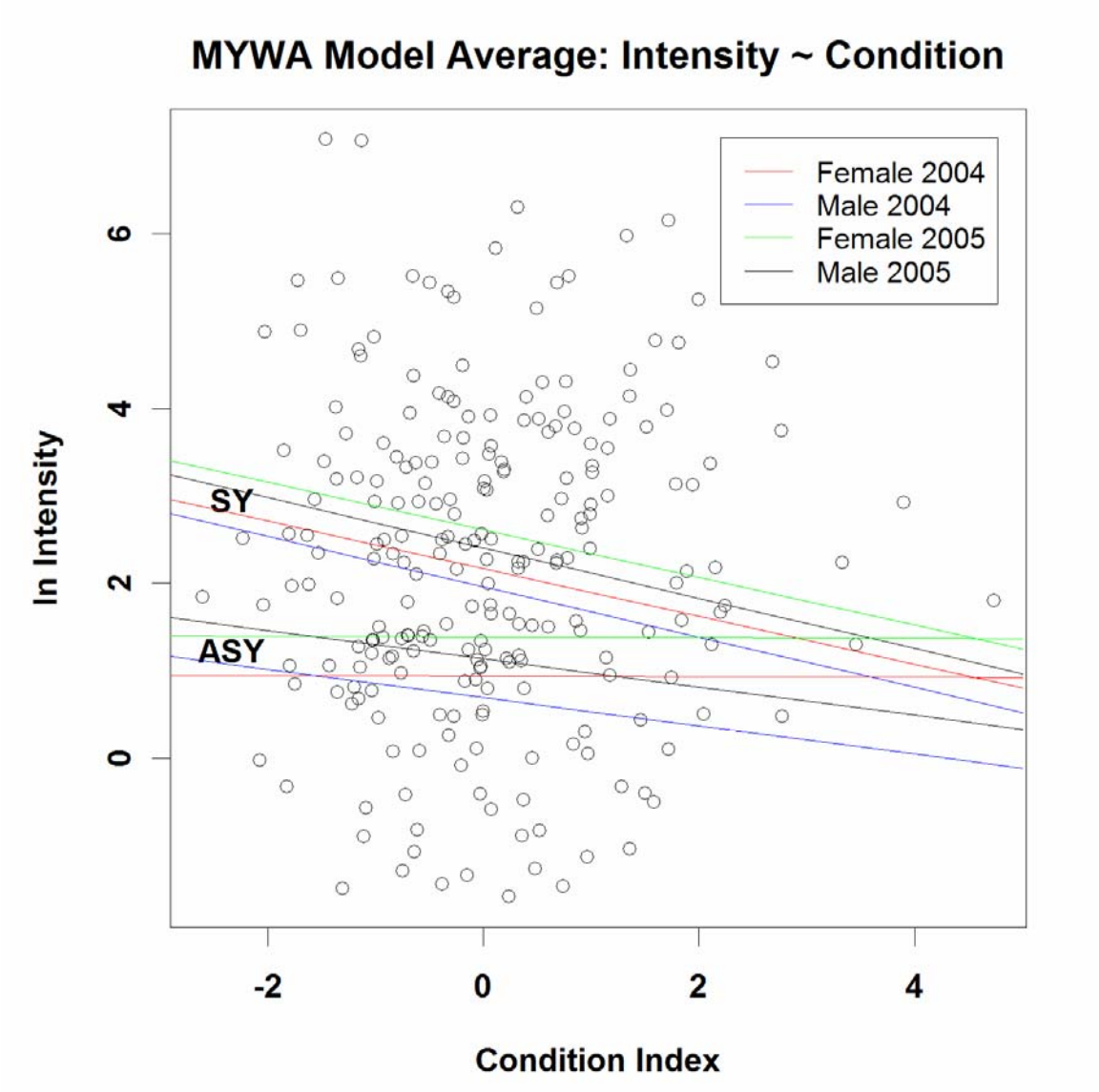

Figure 3.2. Average Yellow-rumped Warbler linear regression model when arrival index equals zero. ln(Intensity) = 2.17 + 0.45*Year – 0.27*Condition – 1.23*Age – 0.21*Sex + 0.27*Condition*Age – 0.04*Age*Sex – 0.02*Condition*Sex – 0.02*Condition*Age*Sex.

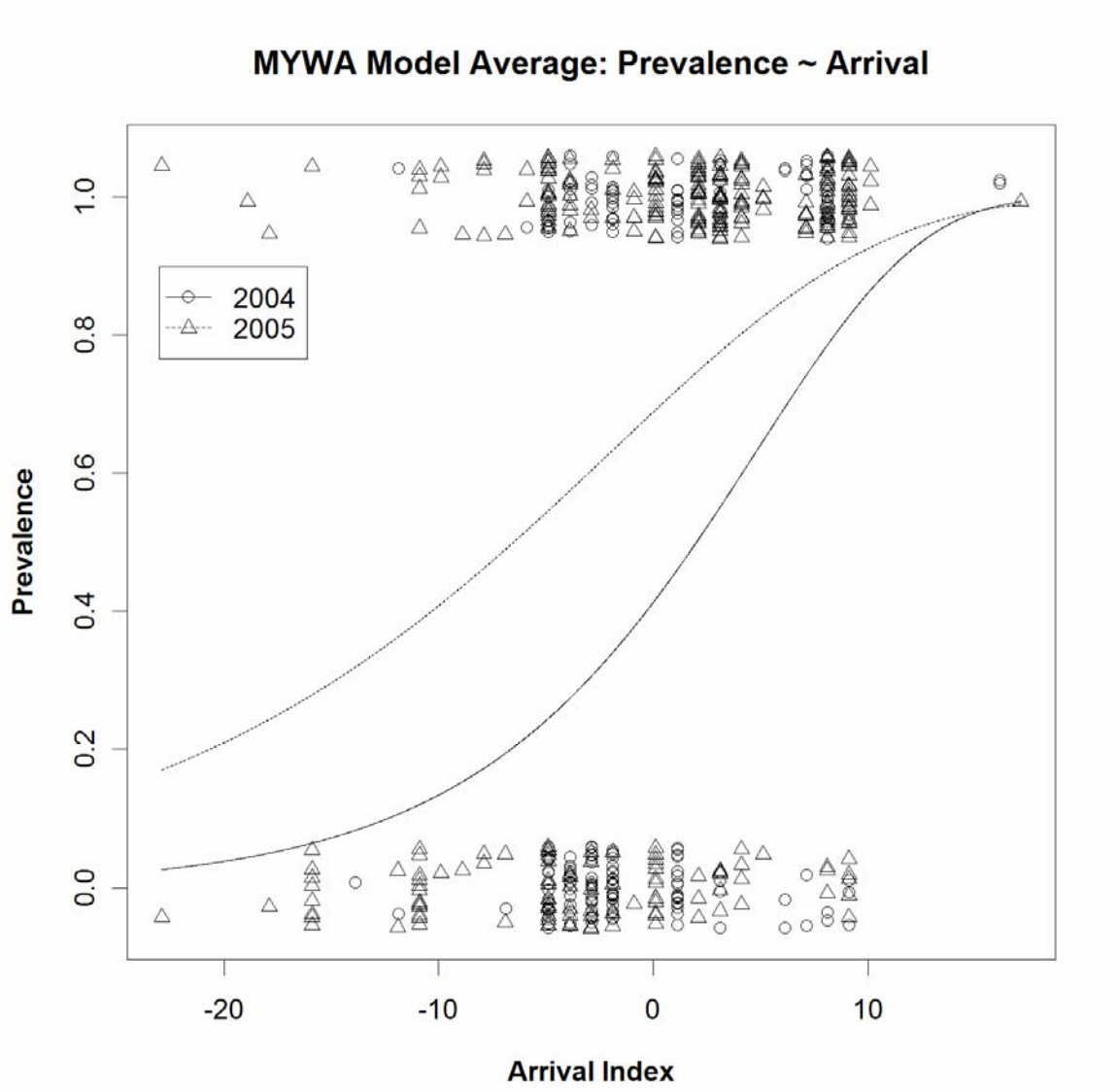

Figure 3.3. Fitted values from logistic regression of infections status (0 or 1, prevalence) by year and arrival index for Yellow-rumped Warblers. Prevalence is jittered to make temporal effects more evident.

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