THE COSTS, EFFECTS, AND HOST RESPONSE TO PARASITISM IN A MODEL COSMOPOLITAN AVIAN HOST SPECIES: ROCK PIGEON (*COLUMBA LIVIA*)

by

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(Under the Direction of C. Ronald Carroll)

ABSTRACT

Emerging diseases are rapidly becoming a global conservation issue, and traditional approaches to understanding transmission among in-situ populations often involved investigating hosts in the context of a single pathogen. Yet it is clear now that understanding disease dynamics within ecological frameworks must address hosts as simultaneously infected with multiple organisms. Helminths are among the most common parasites of vertebrates, yet their impact on host populations are underappreciated due to their typical subclinical effects. Conversely, helminths may indirectly play a role in the spread of other virulent pathogens. Yet before these interactions are investigated, the consequences of parasitism in non-native and polymorphic organisms must be addressed. This study examines free-ranging urban Rock Pigeons naturally exposed to gastrointestinal helminths, blood parasites and avian paramyxovirus-1 virus (APMV-1) in Atlanta, Georgia to examine implications of parasite enemy release, melanism and body condition as drivers of pathogen susceptibility.

INDEX WORDS: Rock Pigeons, Helminths, Melanism, Parasite Distribution, Congeners, Prevalence, Hemoparasites, APMV-1

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DEDICATION

Para mi Mamá, Papito, Luis, Jimmy, Irene, Michael, Ada, Frankie, y mis hijos: Poohbear, Belle, y Bianca.

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CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

"The future will most surely bring about the recognition of more and more diseases of birds. I have often made the analogy of disease as a whole being similar to a giant onion. As we peel away each layer of disease, there is another layer of disease lying underneath."

Phil D. Lukert (1995)

Parasite-host evolutionary and ecological relationships

As the mechanisms of evolutionary and ecological host-parasite relationships continue to garner scientific investigation, the singular association between hosts and their associated parasites further unfolds new layers of complexity. These shifting views of the host-parasite liaison from a fundamentally isolated system between two organisms in a continuous evolutionary arms race have accumulated evidence that the cross-sectional analysis of the dynamics between a host and a parasite elucidate only a fractal image of such interactions. In this thesis, a parasite is defined as any extracellular or intracellular organism adapted to meet its life history requirements through the physiological resources of a host species, such that the consequences to the host include either loss of reproductive or survival fitness, increased morbidity or mortality (Clayton and Moore 1997; Poulin 1998).

In the context of a host as an ecosystem for a parasite (Sprent 1963; Guégan et al. 2005), empirical and theoretical frameworks have uncovered evidence of resource

competition between intra-host concomitant parasite infections (Lello et al. 2004; Graham 2008), intermediary parasite virulence as balancing selection for parasite transmission (Anderson and May 1979; May and Anderson 1983; Levin 1996; Jäkel et al. 2001; Friesen et al. 2006; Shirreff et al. 2011), and trophic cascading effects in the context of the host habitat's immune system as a source of parasite depredation (Pedersen and Fenton 2007; Harris et al. 2012). The unique asynchronous co-adaptation of hosts with lengthier lifespans and short-lived parasite populations ripples into continuous fitness responses for both the parasite and parasitized. Given that, the successive outcomes are robust genotypic consequences of variation through successive generations (Hamilton and Zuk 1982). To further compound the complex effects parasites elicit upon their hosts, parasites with direct intra-host life cycles inevitably exhibit multiple adaptive and generalized generations, consuming a myriad of differentiated physiological resources (Combes 2000).

Among host population thresholds, such as Rock Pigeons (*Columba livia*), the model organism of this study, R_0 represents the level of transmission, or secondary cases of infection, emanating from a single-pathogen infectious host (Lloyd-Smith et al. 2005). Inversely, as R_0 increases, H_T, which represents the population size required to sustain infection, subsequently decreases (Dobson and Merenlender 1991). Yet, these model parameters do not account for the synergistic and antagonistic interactions of parasite richness within coinfected hosts (Cox 2001; Booth et al. 2008; Telfer et al. 2008). Nor does the model address multi-host, multi-pathogen systems where parasites mediate competition among community hetero-specifics (Park 1948; Hudson and Greenman 1998; Poulin 1999). The extrinsic effects exerted by parasites upon community and population dynamics, while previously underappreciated, have received greater attention

in recent years (Poulin 1999). It has now become clear that host regulation by parasitism is a density-dependent function, inversely decreasing host fecundity or recruitment as parasite density increases (Scott and Dobson 1989). For instance, Red Grouse of the Scottish moors (*Lagopus lagopus scoticus*) with high intensities of the cecal nematode (*Trichostrongylus tenuis*) demonstrated increased depredation, reduced fecundity from females, reduced male breeding territoriality, and overall cyclic grouse population oscillations (Hudson 1986; Hudson et al. 1992; Hudson et al. 1998; Fox and Hudson 2001). In another example, the native United Kingdom red squirrel (*Sciurus vulgaris),* is a mammalian system that has endured significant invasive consequences via the introduction of non-native sympatric species. These consequences include localized epidemiological extinctions, dwindling population sizes and ecological niche displacement from exotic grey squirrels (*Sciurus carolinensis),* which serve as the reservoir for the introduced parapoxvirus (Tompkins et al. 2002; Thomas et al. 2003; Tompkins et al. 2003). These parasite-mediated population density effects are not limited to terrestrial organisms. An experimentally manipulated system of *Daphnia magna*, a freshwater water flea, independently challenged with six microparasites established that treated Daphnia hosts not only displayed reduced survivorship, but also inhibited fecundity, population density, and in the case of more virulent microparasites, exacerbated both host and microparasite extinction rates in contrast to control cohorts (Ebert et al. 2000). In a shared parasite model system, the Ring-necked Pheasant (*Phasianus colchicus*) demonstrated ecological host exclusion of a conspecific galliform, the Grey Partridge (*Perdix perdix*) due to helminth infectiousness from the more competent pheasant reservoir host. The high host competence of the pheasant and its subsequently higher intensity egg shedding rates of the nematode *Heterakis gallinarum*

subsequently negatively influenced partridge fecundity, body mass, morbidity, and mortality with effects scaling up to spatial ecosystem dynamics for partridge and pheasant co-existence (Tompkins et al. 1999; Tompkins et al. 2000; Tompkins et al. 2001). Parasitism may also result in suboptimal host condition, resulting in proximal mortality from depredation as opposed to direct mortality through parasitism. A combination of qualitative and quantitative assessments found that predation attempts by Red-tailed Hawks (*Buteo jamaicensis*) were more successful when prey with higher parasite loads were pursued as opposed to prey with lower parasite loads (Temple 1987). While these examples supply ample evidence that parasites exert regulatory effects upon host populations, conversely, host populations may respond to these selective pressures through greater fitness in subsequent generations. This response was demonstrated in a factorial mesocosm of *Aedes sierrensis* mosquito larvae randomly exposed or control treated with the cilate parasite *Lambornella clarki* at two larval densities, high or low (Washburn et al. 1991). Resource availability manipulated by the mesocosms' energetic requirements from the high or low densities of larvae established that parasitized high-density larvae first suffered a population trough, reducing the initially imposed nutrient limitations. The remaining population, which was no longer under the selection pressure of resource and intraspecific competition, emerged with greater fitness attributes, such as larger body size and fewer infected individuals in subsequent generations. Such is the paradox of parasitism; that while parasites may exert deleterious effects upon their hosts, these effects may generate phenotype plasticity in subsequent host generations through continuous responses scaling up to the population and community level.

Host-parasite fitness trade-offs

Habitats are inextricably linked to parasite success; as a high quality external

habitat not only provides an abundance of susceptible hosts, but also facilitates the host's body condition and resource availability for exploitation via the parasite (Sprent 1963). The extension of metapopulation theory to spatial epidemiological theory suggests that as habitat resources are aggregated, thus so are the hosts harboring parasites (Hudson and Dobson 1997; Singh et al. 2004; Calabrese et al. 2011). In this framework, colonization of hosts via infection, host mortality, morbidity and immunity to subsequent infections are comparable to host metapopulation movement through fragmented islands for disease. Although parasites are ubiquitous in nature, they are still limited by the availability of susceptible host patches to ensure continued transmission (Hess 1996; Grenfell and Harwood 1997; Rodríguez and Torres-Sorando 2001). Thus, hosts with strong immunity to a particular pathogen may serve as sink habitats, whereas hosts with limited immunity to a particular pathogen may serve as a source habitat. To add an even further layer of transmissive heterogeneity, within host subpopulations, the greatest intensity of parasites are often aggregated within a small number of individuals. Subsequently, these parasite aggregations upwell into survivorship consequences for hosts harboring the highest intensities (Agarwal 1990; Dobson and Merenlender 1991; Poulin 1993; Shaw and Dobson 1995; Jaenike 1996; Goater and Holmes 1997; Morand and Guégan 2000). This phenomenon, widely accepted by disease ecologists as the 20/80 rule, accords that 20% of hosts will generally harbor 80% of a particular pathogen and will assume responsibility for at least 80% of disease transmission (Woolhouse et al. 1997; Perkins et al. 2003; Hawley and Altizer 2011). This pattern explains the inherent variability demonstrated by host species sampled for evidence of parasites, magnifying the disparities between pathogen prevalence and intensity of infection (Poulin 1993). Parasite aggregation has a number of bidirectional effects upon host-parasite

communities, such as epidemiology and the genetic polymorphisms of both pathogens and hosts. These bidirectional effects are particularly influential in the cost trade-offs for evolving resistance to a particular pathogen, which further generates host heterogeneity (May and Anderson 1983; Hellriegel 2001). Furthermore, host polymorphisms in the innate and adaptive immune system, heterogeneity in pathogen exposure, susceptibility to parasite establishment, and parasite fecundity all influence the degree of parasite aggregation. Yet, of all the variables that pathogens must contend with, it is the host's immunological response in reducing parasite fecundity which has the strongest consequences for transmission to conspecifics, thus continuing the seemingly infinite cycle of selection and polymorphism (Galvani 2003). This cycle of selection is documented in the indirect role of parasites to alter host behavior, phenotypes, and body condition, correlated with both humoral and cell-mediated immunity to pathogen invasion (Gonzalez et al. 1999; Poulin 1999; Navarro et al. 2003; MacColl and Chapman 2010). It is the latter two variables, which will be addressed in this thesis.

CHAPTER 2

ENEMY RELEASE: THE PREVALENCE, DIVERSITY, AND INTENSITY OF ROCK PIGEON (COLUMBA LIVIA) PARASITISM IN ATLANTA, GEORGIA IN CONTRAST TO A BIOGEOGRAPHICAL ANALYSIS OF PARASITES AMONG NATIVE AND ASSUMED RANGES [1]

[1] Ayala, A.J., S.M. Hernandez, V.O. Ezenwa and P.J. Miller. To be submitted to *Avian Diseases*.

ABSTRACT

Originally transported via trade routes primarily for nutritional and cultural purposes, Rock Pigeons (*Columba livia*) are among the first recorded avian translocations, dating back to the Neolithic Age. Full genomic sequencing of domesticated pigeon breeds and partial genomic sequencing of free-ranging Rock Pigeons from the United States provides further evidence that not only did Rock Pigeons likely originate from Mesopotamia, but also that domesticated breeds have consistently interbred with wild Rock Pigeons since the initial phase of captivity. We assessed prevalence, intensity and diversity data collected among a population of free-ranging Atlanta, Georgia Rock Pigeons in conjunction with an extensive literature review (Table A1) to examine the degree of rock pigeon parasite release of this now globalized species among native and introduced ranges. Results indicated that likely as a result of time of invasion within the introduced New World range, parasite inversion, as opposed to parasite release, occurred through the accumulation of novel parasites in non-native ranges.

INTRODUCTION

Not to be confused with incidence, parasite prevalence is defined as the cross-sectional, quantitative number of hosts infected with a particular pathogen at a particular point in time (Wobeser 2006). Intensity is parameterized as the sum of a single species of parasite detected within a single host (Bush et al. 1997). Parasite diversity (Bush et al. 1997) is a far more difficult quantitative measure of pathogen abundance, as it is highly dependent upon species, sampling effort and empirical objectives, latitudinal, temporal, climatic, and spatial constraints. Generalities in terms of macroparasite diversity have been attempted, such as the concept that on average, each bird species will be infected with a mean of "three cestodes, two trematodes, three nematodes, and one

acanthocephalan" (Dobson et al. 2008). Yet what of the microparasites, especially viruses, where single-stranded RNA viruses may mutate into novel strains at several orders of magnitude faster than double-stranded DNA viruses due to polymerase and nucleotide base substitution errors (Duffy et al. 2008). Not only are new viral diseases discovered on an average of one per year, but RNA viruses account for at least a third of these discoveries, and disproportionately represent the greatest threats amongst emerging diseases (Woolhouse 2002; Howard and Fletcher 2012). Given that, it appears unlikely that every pathogen harbored by a given host in a single sampling point will be identified.

Anthropogenic pressures from human encroachment, habitat fragmentation, biodiversity loss, the introduction of non-native species and their corresponding pathogens are some of the causal effects attributed to emerging infectious diseases (Daszak et al. 2000; Woolhouse 2002; Bradley and Altizer 2007). A classic example of the decimating effects of emergent diseases into naïve populations are the introduction of avian malaria and avian poxvirus, which devastated the endemic avifaunal populations of Hawaii (van Riper III et al. 1986; van Riper III et al. 2002). More recently, the introduction of the ornithophilic mosquito vector *Culex quinquefasciatus* has been implicated in the transmission of a particularly virulent form of the blood parasite *Plasmodium* sp. into the Galapagos Penguin (*Spheniscus mendiculus*). This finding is a dramatic concern for conservation due to the Galapagos Penguin's limited geographic range, small effective population size, and depauperate major histocompatibility complex (MHC) class II diversity (Bollmer et al. 2007; Levin et al. 2009). The 1999 establishment of a middle-eastern strain of West Nile virus in New York City spread to the West Coast within four years, reducing avian communities throughout the United States (Lanciotti et al. 1999; Reisen et al. 2004). Mortality was most critical in American

Crows (*Corvus brachyrhynchos*), whose populations were decimated by an estimated 65% (Caffrey et al. 2005). Newcastle disease virus (avian paramyxovirus-1), genus *Avulavirus* of the family *Paramyxoviridae*, a potentially virulent and economically devastating poultry disease, was first identified in the United States in 1943 (Beach 1944; Miller and Afonso 2011). Now endemic in North America, the third major viral panzootic spilled over into the columbiformes in 1984, whilst the rapidly continuing evolution of this RNA virus is currently classified into one class I genotype and sixteen class II genotypes (Pearson et al. 1987; Alexander 1988; Miller et al. 2010; Diel et al. 2012a; Courtney et al. 2013). As of 2009, approximately 241 avian species representing 27 taxonomic orders were identified as susceptible to at least one genotype of the virus (Alexander 2009). The prospective virulence and host-switching propensities of this genetically labile RNA virus to free-ranging birds has such potential biodiversity costs, that serological investigations of Newcastle disease virus in backyard chicken flocks were implemented, yielding high prevalence for the disease in Mesoamerica (Hernandez-Divers et al. 2006; Hernandez-Divers et al. 2008). .

With an introduction to the ramifications of novel, invasive avian parasites, the question still remains as to what the ramifications *are* of novel, invasive avian hosts. For example, one tenet of the enemy release hypothesis, "pathogen release" is proposed as a mechanism facilitating the successful invasion of exotic species into new, often distant geographic areas (Mitchell and Power 2003; Torchin et al. 2003; Perkins et al. 2008). More importantly, successful establishment of an exotic species appears to be most effective during the initial stage of invasion period for parasite release (Kvach and Stepien 2008). Although the concept of "parasite release" has not garnered ubiquitous support in the literature (Colautti et al. 2004; Ishtiaq et al. 2006; MacColl and Chapman

2010), a number of empirical and theoretical studies provide a substantial platform supporting this hypothesis from a broad range of taxa (Torchin and Mitchell 2004). A common garden experiment performed on native and non-indigenous plant congeners demonstrated that native plants profited less from beneficial soil microbes while simultaneously suffering more from the effects of pathogenic microbes (Agrawal et al. 2005). Among marine taxa, an exotic crab species (*Hemigraspus sanguineas* and *Carcinus maenas*) established in the northeastern United States with invasion periods differing within an order of magnitude of one another not only had a statistically significantly diminished parasite richness, but also exhibited greater fitness measures from the loss of castrating parasites from their native ranges (Blakeslee et al. 2009). Two species of Eurasian gobies detected in the Great Lakes region in 1990 had a maximum 28% parasite species richness in contrast to their native ranges (Kvach and Stepien 2008). Similar results have been exhibited in the cosmopolitan non-native populations of *Bufo marinus*, having lost 31 to 43 of their native helminth parasite richness, with current helminth diversity attributed primarily to infections acquired in their new ranges (Barton 1997).

Birds, arguably one of the most studied (Mora et al. 2011) and relocated vertebrate taxa (Cassey et al. 2004), have also demonstrated support for the parasite release hypothesis. House Sparrows (*Passer domesticus*) exhibited a highly reduced blood parasite prevalence (Lima et al. 2010), and fewer phylogenetic lineages of *Haemoproteus* sp. and *Plasmodium* sp. than their native congeners in Europe (Marzal et al. 2011). European Starlings (*Sturnus vulgaris*), one of the most successful avian invaders, reportedly carried only nine parasites in new ranges as opposed to the 44 species in their native locales (Torchin et al. 2003; Cassey et al. 2004). Even over sixty

years ago, in a survey of blood parasites among New Zealand exotic and native birds, *Haemoproteus* sp. was absent from the non-native Rock Pigeon (*Columba livia*) and Silvereye (*Zosterops lateralis*) (Laird 1950).

Important factors in quantifying the degree of parasite release include time since invasion, the number of subsequent invasions into the same area, intermediate host availability, and whether the composition of community richness in the new range includes sympatric species with generalist parasites (Torchin and Mitchell 2004; Torchin and Lafferty 2009; Blakeslee et al. 2012). Conversely, non-indigenous hosts will not only carry their own native parasite flora to new ranges, but it is hypothesized that these hosts may benefit in their establishment into new locales via domestication and propagation by humans prior to escape as free-ranging populations (Mack et al. 2000; Mitchell and Power 2003). For these reasons, Rock Pigeons serve as an excellent and tractable model system to evaluate the potential role of the "parasite release tenet" as a mechanism for successful establishment, utilizing field collected data on both macroparasites and microparasites of a wild pigeon population in Atlanta, Georgia**.**

The study system

Although Darwin came to fame for his theory of natural selection, the first chapter of the *Origin of Species* is dedicated to his artificial selection experiments on domestic English pigeon breeds. From that work, he mounted irrefutable evidence that all such birds were derived from the Old World Rock Dove, *Columba livia* (Darwin 1859; Secord 1981). In North America, wild pigeons were first translocated by French and English colonists into Nova Scotia between 1603 and 1608, for utilization as pets and for consumption (Schorger 1952; Erickson 2008). Peridomestic North American flocks established themselves by the 17th century via escapees and releases, and centuries of

consistent interbreeding with domestic birds resulted in the currently recognized polymorphic variants (Johnston and Johnson 1989; Johnston 1992; Hodges Jr. 2010). Five distinct 'electromorph' phenotypes determined by allozyme expression were recognized by Johnston and Janiga (1995), while today the Cornell Laboratory of Ornithology recognizes seven visually distinct free-ranging pigeon morphs in North America: Red, Checker, Pied, White, Spread, Blue-bar and Red-bar (Cornell 2009; Stern and Dickinson 2010; Feinstein 2011). The feeding habits of wild pigeons are primarily granivorous, although trash, manure, and handouts from humans are not refused (Williams and Corrigan 1994; Sol et al. 1998). In Georgia, the pigeon breeding season is year-round, although the breeding season peak is likely concurrent with that of other resident passerine species (spring/summer) due to extended daylight periods (Riddle 1971; Hodges Jr. 2010). Like most avifauna, wild pigeons are socially monogamous, forming life-long pairs, and will build a platform nest in which females usually lay two eggs (Williams and Corrigan 1994; Gayathri et al. 2004; Hodges Jr. 2010). Hatchlings emerge 17-19 days after synchronous egg laying, and nestlings fledge approximately 32 days after hatching (Patel 1936; Abs 1983; Johnston and Janiga 1995). Bi-parental care comprises both sexes performing incubation, brooding the hatchlings, and feeding their offspring 'crop milk' from which both paternal and maternal antibodies (IgA and IgC) are transferred to the nestlings (Patel 1936; Abs 1983; Engberg et al. 1992; Short 1993; Baer 1999). The assimilated parental antibodies protect the nestlings for an average of 27 days, just prior to fledging from the nest (Gibbs et al. 2005). In summation, the extended breeding season, opportunistic foraging ecology, and adaptation from ancestral cliff roosting in the Old World to breeding on the high-rise buildings and highway bridges of the New World facilitated the wild pigeon's overwhelming success in urban areas (Tietz

Marques et al. 2007).

In the United States, Rock Pigeons are now regarded as an urban pest, held responsible for over a billion dollars per year in damages, and associated with over 50 pathogens and parasites (Haag-Wackernagel 2005; Pimentel et al. 2005; Pinkerton et al. 2008). Unfortunately, it is not widely understood that only a small fraction of the parasites to which pigeons are susceptible are zoonotic (Himebaugh and Gopalakrishnan 2011). In Georgia alone, wild pigeons are estimated to number approximately 190,000 birds, concentrated primarily within the metro Atlanta area (Rich et al. 2004; Hodges Jr. 2010). The overall density of pigeons in Georgia has declined since 1996 as a result of a state-wide culling program which euthanizes approximately 5,000 individuals per year; a consequence of the misconceptions on the potential for disease transmission to humans (USDA 2009; Hodges Jr. 2010). Ironically, field experiments examining the strategic culling of wildlife for disease control have concluded that culling may instead perturb equilibrium host transmission rates, increasing epidemiological risk factors for disease outbreaks (Choisy and Rohani 2006; Streicker et al. 2012).

Since it has been become clear that free-ranging pigeons are susceptible to multiple, concomitant infections of both microparasites and macroparasites in the Atlanta, Georgia metro area (Allison et al. 2004), a wide body of literature in conjunction with field collected data examines the intensity, diversity, prevalence, and parasite release and species richness among New World and Old World Rock Pigeons.

FIELD MATERIALS AND METHODS

Capture and sampling of wild Rock Pigeons

Rock Pigeons were captured from metro Atlanta, Georgia between May and October 2012. From May – July, pigeons were caught at three urban sites in Fulton County, GA,

while from August – October 2012, pigeons were captured by the USDA-APHIS Wildlife Services personnel in Fulton County as part of their integrated wildlife damage management program (USDA 2009) (Figure 2.1). Pigeons were captured during daylight hours near roost and feeding sites using a combination of hand-nets, drop-nets, mist-netting, and ground traps. During the May-July capture period, all released individuals were banded with a unique four band-color combination to avoid pseudoreplication. For individuals captured twice, collected data was only scrutinized if sampling on the first attempt did not produce analyzable samples.

For each bird, we recorded demographic attributes including age, sex, and breeding status. Birds with light-colored ceres and orange irises but still molting juvenile primaries with partial or fully retained mantle and scapular feathers were classified as hatch-year birds (HY). Juvenile primary molt limit was used in HY birds to provide an approximate number of days since fledging (Kautz and Seamans 1986). Birds with white ceres and orange or red irises not in molt, or undergoing interrupted pre-basic molt in combination with reproductive traits were classified as after hatch-year birds (AHY) (Kautz and Seamans 1986; Blasco-Zumeta 2010).

Breeding status was established using reproductive characteristics, such as the presence of a brood patch, cloacal protuberance, or both (Abs 1983; Pyle 1997). The sex of AHY birds was determined by reproductive characteristics as well as the extent of neck iridescence, where a broad band of purple and green iridescence circling the nape signaled an adult male, and faint iridescence with little to no banding around the nape signaled a female (Blasco-Zumeta 2010). When observed by banded individuals, courtship behaviors were also utilized in sexing (Sol et al. 2000).

We assessed individual body condition using three different indices: weight to

wing chord ratio (Hull et al. 2006), wing-pit fat (Roberts et al. 2005; DeSante et al. 2011), and flight feather wear (Young 1991). Weight to wing chord ratio was calculated as body mass divided by the length of the un-flattened longest primary (Pyle 1997; Hull et al. 2006) while the MAPS (Monitoring Avian Productivity and Survival) protocol (DeSante et al. 2011) was used for fat scoring. Only wing-pit fat was assessed due to the density of plumage at the furculum and stomach. Fat was scored on a seven point scale, with a zero score indicating no yellow subcutaneous fat at the wing-pit and a score of seven indicating a bulging pocket of fat. Flight feather wear was evaluated by examining the outermost 5 primaries (primary numbers 10 – 5) and scored on a 0 - 5 point scale, where zero indicated fresh feathers and a score of five indicated excessively ragged feathers (Young 1991; DeSante et al. 2011). In addition, we recorded the color morph of each bird using morphotypes delineated by the Cornell Lab of Ornithology: Red, Checker, Pied, Spread, White, Blue-bar and Red bar (Johnston and Johnson 1989; Cornell 2009; Feinstein 2011).

Blood and swab sample collection

Up to 1 mL of blood was taken from the brachial vein of each individual using a 25 to 31 gauge needle attached to a 1 mL syringe, and transferred to a 3 mL red-top serum vacutainer tube before clotting commenced (Owen 2011). Blood samples were placed in a cooler with ice packs until transfer to the lab. Once in the lab, vacutainer tubes were tilted overnight and then centrifuged at high speed to harvest serum, with sera stored at -20°C until further analysis (Dovč et al. 2004). Oral and cloacal swabs were collected from each bird by swabbing the mouth four times or the cloaca once for consistency in collecting comparable quantities of viral particles using sterile polyester tipped swabs (Puritan ®). Swabs were then transferred into 2.0 mL cryovials (Corning

®) containing 1.5 mL of Brain-Heart Infusion (BD ®) mixed with the antibiotics Gentamicin and Penicillin, and the anti-fungal Amphotericin-B to inhibit bacterial and fungal growth while simultaneously preserving viral particles. Cryovials were stored in a cooler with ice packs while in the field and then transferred to -80°C immediately upon arrival at the laboratory.

Parasitological analyses

Fecal samples were collected from all birds during field processing to quantify gastrointestinal helminth infections. Samples were stored individually in tubes, kept on ice in the field, refrigerated at approximately 1.6°C, and processed within three days. The number and type of gastrointestinal helminth eggs shed by pigeons were quantified by diluting 0.05 grams of feces in 1.45 mLs of saturated salt solution and calculated using a modified McMaster helminth egg-counting technique (Gordon and Whitlock 1939; Seivwright et al. 2004). Parasite eggs were identified to genus level where possible following Sloss et al. (1994). To evaluate hemoparasite prevalence and intensity, blood smears were made in the field using the protocol of Bennett (1970) with the exception of methanol fixation; smears were then air-dried and transported in a slide box under ambient conditions. Within seven days of collection, all smears were stained with a Wright-Giemsa stain (Camco-Quik Stain II ®)**.** To determine the mean number of erythrocytes per field of view (FOV), ten blood smears were randomly selected from throughout the five-month long field season resulting in a mean erythrocyte count of 255 red blood cells per FOV. Each blood smear was subsequently analyzed for hemoparasites using 100 FOV under a magnification of 100 and 400, for approximately 25,500 red blood cells analyzed per smear (Bromwich and Schall 1986; Martinsen et al. 2008). Using a cell counter, hemoparasites were delineated into genus, while blood

parasites of uncertain taxonomy were tabulated as unknowns. Identification of blood parasites to genus followed an illustrated identification guide presented in Campbell (1995).

Serum samples were analyzed for avian paramyxovirus-1 (APMV-1) antibodies with hemagglutination inhibition assays at the Southeast Poultry Research Laboratory in Athens, GA (SEPRL). Briefly, serum samples were titrated into 96-well plates using two-fold dilutions of phosphate buffered saline and 0.5% washed specific pathogen free chicken red blood cells tested against an 4 HAU units of Newcastle Disease virus (APMV-1) LaSota strain antigen, where an endpoint titer of greater than1:16 is considered positive (Dovč et al. 2004; USDA 2005). APMV-1 was also isolated at the SEPRL via inoculation of embryonating white leghorn chicken specific pathogen free eggs from cloacal and oral swabs, which then underwent two serial passages of allantoic fluid. After viral propagation in eggs, harvested allantoic fluid was tested using hemagglutination tests (HA) to identify samples positive for live NDV. Positive HA samples underwent RNA extraction and PCR using a Superscript III RT-PCR Kit (Invitrogen ®), and subsequently sequenced using Fusion (F) gene specific primers for genotype identification (Miller et al. 2009; Susta et al. 2011; Diel et al. 2012b; Courtney et al. 2013).

To tabulate the ranges of endoparasites relevant to the field study performed in Atlanta, Georgia (helminths, two viruses, and two bacterial species), a survey of 77 literature sources spanning the years 1920 to 2013 presenting data on endoparasite prevalence were reviewed to determine the number of cosmopolitan, non-native, and native parasites. Due to linguistic, spatial, and temporal differentiation in taxonomic classification, endoparasites were cross-referenced within the database the *Encyclopedia*

of Life (http://eol.org/) (Wilson 2003; Blakeslee et al. 2012). When possible, primary sources were consulted, but when language (other than English or Spanish) presented a barrier to the discernment of relevant material, secondary intermediate sources were instead utilized to provide the most reliable results. When multiple samplings of the same endoparasite were detected in either the original or introduced range, the sampling schematic with the largest number of sampled Rock Pigeons was utilized. Due to the large prevalence of papers identifying endoparasites at the genus level only, as a result of unavailable molecular technology from temporal or logistical (funding) restraints, species were analyzed when available and genus was utilized instead for those parasites in which that taxonomic classification was provided. Endoparasite reports providing only presence/absence data instead of sample size and prevalence were excluded from analysis. Native and non-native ranges were identified using the IUCN database (http://www.iucn.org/).

Statistical analyses

All statistical analyses were performed using SAS v. 9.3. A generalized linear model (GLM) accounted for uneven sample sizes in age, season, and sex for all analyses unless otherwise stated. Fixed variables included demographic characteristics, while random effects included the presence or absence of parasites. A Shapiro-Wilk test performed on each endoparasite detected determined whether the intensity of parasitism across sampled Rock Pigeons demonstrated a Gaussian distribution. Non-viral parasites were analyzed using both the raw calculated data to determine intensities using the GLM as well as the $\log_{10}$-transformed data. A Probit logistic model was utilized to analyze viral shedding rates.

RESULTS

Field Study in Atlanta, Georgia

Demographic data yielded 76 birds classifiable as either HY or AHY, with HY birds captured at the highest rate. Out of 78 total birds, only 37 birds could be sexed using available parameters. Of adult birds, only 18 Rock Pigeons evinced discernible reproductive characteristics. Seasons were delineated as summer (June – August), whereas fall included September and October.

The McMaster egg-counting technique yielded fifteen individual genus level helminth taxa; nine nematodes, four cestodes, one trematodes, and one acanthocephalan which could not be identified to further specific Linnaean classification. Of 78 Rock Pigeons sampled, 97.44% shed helminth eggs at the point of capture, with 93.59% of birds shedding at least one nematode, 28.21% shedding at least one cestode, 5.13% shedding the single trematode taxa, and a single bird shed acanthocephalan eggs at 1.28%. For nematodes, *Ascaridia* sp. had the highest helminth prevalence at 84.62%, followed by *Dispharynx* sp. at 44.87%; *Capillaria* sp. at 26.92%, *Trichostrongylus* sp. at 23.08%, *Tetrameres* sp. at 17.95%; *Syngamus* sp. at 12.82%; *Heterakis* sp. at 11.54%; *Allodapa* sp. at 2.56% and *Porrocaecum* sp. at 1.28%. For cestodes, *Choataenia* sp. had the highest prevalence at 12.82%; *Railletina* sp. followed at 10.26%, *Hymenolepis* sp. at 6.41%, with the lowest cestode prevalence detected with *Taenia* sp. at 3.85%. A correlation coefficient network plot is presented in Figure 2.2 to visualize the relationships between helminth correlations (Table A2). The number of different helminth types in which individual birds were infected varied from zero to six taxa. Helminth egg taxa which were not distinguishable were not placed into this analysis. Thus, two birds did shed undistinguishable helminth types, with a total sample size of identifiable helminth taxa diversity remaining at 76 birds. Overall, two birds shed no

helminth eggs at the time of sampling for a prevalence of 2.56%. Rock Pigeons with one discernible helminth taxa had a prevalence of 20.5%; followed by two discernible helminths also at 20.5%. The highest prevalence of diversity was identified in 20 of 76 birds with three helminth taxa for a prevalence of 25.64%. Four distinguishable helminth taxa were detected within 14 individual birds at a prevalence of 17.95%, followed by diversity indices of five helminths in five individual birds at 6.41%. Six distinguishable helminth taxa were detected in three individual birds at 3.85%. Among all helminths, egg shedding ranged in intensity from 0 eggs to 36,720 eggs, with the most overall eggs shed by *Ascaridia* sp. A single bird infected with six helminth taxa shed 38,460 various helminth eggs in 0.05 grams of feces.

Blood parasites were detected at very high levels, with *Haemoproteus* sp. in 98.53% of 68 blood samples analyzed, followed by *Plasmodium* sp. at 91.18%, *Leucocytozoon* sp. in 5.88% of blood smears and Microfilaria in 4.41%. *Haemoproteus* intensity ranged from 0 to 4,452 infected erythrocytes out of 25,500, with the individual bird with the highest intensity averaging 17.5% of infected erythrocytes. *Plasmodium* intensity ranged from 0 to 3,676 infected erythrocytes, with the individual pigeon with the highest intensity averaging 14.7% of infected erythrocytes. Dual blood parasite infection was more common than single infection; of 68 Rock Pigeons, 55 birds (80.1%) had intracellular erythrocyte infections with *Haemoproteus* and *Plasmodium*; in addition, while not counted, it was observed that some red blood cells contained dual intracellular infections with both taxa. For Rock Pigeons that carried only single infections of blood parasites, 8.82% of those infections were *Haemoproteus* alone, whilst only a single bird (1.47%) carried only *Plasmodium*. Detection of Microfilaria and *Leucocytozoon* were never found as non-concomitant blood parasite infections; of the three birds harboring

extracellular microfilariae, two of the birds had triplet infections of Microfilaria, *Haemoproteus* and *Plasmodium*, while a single bird harbored Microfilaria and *Haemoproteus*. All four birds (5.88%) that carried *Leucocytozoon* also carried *Haemoproteus* and *Plasmodium* as triplet infections.

The only gastro-intestinal protozoan analyzed in this study was pooled under the sub-class Coccidia (Phylum: Apicomplexa) due to the lack of molecular analysis and difficulty in differentiation via visual examination. The prevalence of coccidia in 78 Rock Pigeons sampled was detected at 83.33%, with a range of 0 to 61,140 oocytes detected in 0.05 grams of feces. Although West Nile virus was originally a portion of the experimental design, 60 plasma samples collected were not analyzed due to time constraints, but the virus has been detected in Atlanta, Georgia pigeons in a different study, thus those results were utilized as part of the literature review analysis. In the current field study, APMV-1 seroprevalence was identified in 5.63% of 71 sampled Rock Pigeons, while 72 oral and cloacal swabs (divided into 41 sampled birds as one oral and one cloacal swab per pigeon) were pooled for a total 15.28% of Rock Pigeons actively shedding virus. If an individual bird shed virus from both the choanal slit and from the cloaca, the individual was treated as a single sample, and thus analyzed so.

Although seasonal analysis demonstrated no statistical difference in egg shedding for pooled helminth taxa, overall Rock Pigeons did shed more helminth eggs in the summer ($n = 55$, $\bar{x} = 2686.91$ eggs, $\alpha = 0.05$, CI 711.4 – 4662.4; $df = 77$, $F = 1.86$, $p = 0.1772$) (Figure 2.3, Table 2.1). In contrast, fall egg-shedding means ($n = 23$, $\bar{x} = 584.35$, CI -87.24 -1255.94 and ± 1555.06) exhibited a marked decrease (Figures 2.4A, 2.4B, Table 2.2). Interestingly, helminth diversity did statistically increase in the summer as opposed to the fall ($df = 77$, $F = 6.89$, $p = 0.0104$) (Figure 2.5).

Demographic analysis for age demonstrated that pooled helminth egg shedding (HY versus AHY birds) was not statistically significant ($n = 76$, $df = 75$, $F = 0.45$, $p = 0.5065$), but that overall, HY ($n = 48$) birds shed fewer eggs than AHY ($n = 28$) birds. The diversity of helminth taxa between age classes was also statistically insignificant ($n = 76$, $df = 75$, $F = 0.15$, $p = 0.6990$), although the variation as calculated by $\alpha = 0.05$ demonstrated a wider CI for AHY birds (2.04 – 3.10) as opposed to HY birds (2.26 – 3.15). The interaction between age and season for all helminths was also statistically insignificant ($df = 75$, $F = 0.78$, $p = 0.5101$), whilst the same statistical insignificance was also found for the interaction between age, season, and the diversity of helminth taxa ($df = 75$, $F = 2.51$, $p = 0.0655$). Sex did not yield a statistically significant result nor for the diversity of helminth taxa ($df = 36$, $F = 0.32$, $p = 0.57$) (Figure 2.6). Nor was it significant for log-transformed pooled helminth data ($df = 36$, $F = 0.2684$, $p = 0.6077$) or non-log transformed pooled helminth data ($df = 36$, $F = 0.45$, $p = 0.5065$) (Figure 2.7). Coccidia analysis demonstrated a statistically significant effect, with more oocytes detected in the fall as opposed to the summer ($df = 74$, $F = 7.17$, $p = 0.0091$). Surprisingly, a similar analysis did not detect a statistically significant difference in coccidia oocytes in HY birds as opposed to AHY birds using un-transformed data ($df = 72$, $F = 1.99$, $p = 0.1631$), although the effect was significant with the log-transformed data ($df = 74$, $F = 3.7837$, $p = 0.0274$) (Figure 2.8). Overall, HY birds shed more coccidia oocytes than their AHY counterparts ($\bar{x} = 2.22$ and 1.83 log transformed, respectively). The interaction between season and age did produce a statistically significant effect ($df = 72$, $F = 2.99$, $p = 0.0370$) (Figure 2.9). Sex was not statistically significant for coccidia with either transformed or untransformed data ($df = 36$, $F = 0.4399$, $p = 0.5115$, log transformed data).

Blood parasite analysis for both seasons did not yield a statistically significant result with *Haemoproteus* sp. via the un-transformed or log corrected data (df = 67, F = 0.001, p = 0.9960, log-transformed data), nor was a statistically significant difference apparent when the log-transformed data was partitioned by month (df = 67, F = 0.73, p = 0.5716). Although not statistically significant, June demonstrated the fewest log-transformed infected erythrocytes with the highest number in September ($\overline{x}$ of 1.799 and 2.210 respectively) (Figure 2.10A). Sex and age were also not statistically significant with the original or log-transformed data (df = 67, F = 0.3839, p = 0.6827 and df = 30, F = 3.1688, p = 0.0855, log transformed, respectively). Log-transformed *Plasmodium* sp. was not statistically significant when analyzed by season (df = 67, F = 3.58, p = 0.0627), but was statistically significant when seasons were categorized by month (df = 67, F = 3.36, p = 0.0148, log-transformed) (Figure 2.10B). A post-hoc Tukey test identified July ($\overline{x}$ = log of 2.23) with the highest incidence of *Plasmodium* sp. infection and September with the lowest incidence of infection ($\overline{x}$ = log of 1.35), with the means for the remaining three months of sampling not statistically distinctive. Neither sex nor age was statistically significant for log-transformed data (df = 30, F = 0.3054, p = 0.8521 and df = 67, F = 0.3532, p = 0.7038, respectively). Due to subpatent infections and minimal degrees of freedom for microfilaria and *Leucocytozoon* sp., these blood parasites were not analyzed beyond the prevalence data already presented.

APMV-1 analyses yielded a total of 12 birds actively shedding, with 50% shedding during the summer season and 50% shedding during the fall season (df=1 Wald = 2.070, p = 0.1499). Age demonstrated a greater effect, although the model was also not statistically significant. Of the twelve birds shedding virus, eleven of those birds were HY (df = 1, Wald = 3.6152, p = 0.0573).

Literature Review

The survey of 78 primary and secondary sources yielded 69 individual endoparasites spanning both the native and non-native ranges of Rock Pigeons with reported prevalence data and robust sample sizes (see Figure 2.11 for range map). For 69 endoparasites analyzed, 38 parasites were shared between Old World (native) and New World (introduced) pigeons (Table 2.3, Figure 2.12), with 19 endoparasites sequestered only in New World Rock Pigeons, and 19 endoparasites detected in Old World Rock Pigeons only (Table 2.4, Figure 2.12). Of the nineteen Old world parasite taxa, sixteen (84.2%) were classified as cestodes, while in the New world, trematodes dominated (15.8%), followed by an equal representation of cestodes and nematodes (10.5% each, respectively). Furthering this analysis, prevalence data was compared to parasite distributions (New World, Old world or Cosmopolitan). The purpose of this analysis was to test whether a variation in prevalence was equivalent to time since invasion. In theory, host parasite co-evolution within a localized, endemic range should result in the presence of rare, specialist parasites colonizing a limited number of hosts. As a subset of these hosts are moved across large ranges throughout a successive number of transport events, aggregated, rare parasites would be lost, while generalist parasites would be maintained. Over time, continued movement of host subsets (or subsamples of subsamples) would continue this process, until only generalist parasites with the highest prevalence rates would remain. Once the host subpopulation has established itself in its endpoint range, the process of acquiring new parasites would commence. Yet, this endpoint founder population would not only have lost a large degree of parasite diversity, but genetic diversity, as well. Therefore, as new parasites are acquired in non-native ranges from phylogenetically related sympatric species, susceptibility to pathogens may result in

overall high infection rates. Consequently, this sequential analysis rested upon the following assumptions as proposed by (Perkins et al. 2008) modified for this review.

1) Free-ranging rock pigeon parasites found only in the Old World (original range) would include the presence of rare, specialist and/or regulatory parasites with overall minimal prevalence rates due to high fitness costs offset by a more diversified genetic population.

2) Cosmopolitan parasites (parasitizing pigeons in both the New and Old World) would consist of generalist, non-regulatory parasites with increased prevalence rates, which would not interfere with host invasion success.

3) New World only parasites would include novel parasites acquired from sympatric species, but due to reduced genetic variability and demographic stochasticity, hosts surviving the initial lag phase of infection would not exhibit the variation in prevalence rates in relation to their native congeners.

As predicted, while the composition of parasite fauna was distinctly dissimilar between New World and Old World Rock Pigeons, overall prevalence rates between both ranges also contrasted statistically (Pillai's Trace = 0.302, $F(4, 106) = 4.80$, $p = 0.0013$) as expected. To identify whether the mean prevalence of parasites among the three categories: New World (introduced), Cosmopolitan (parasites detected in both non-native and native ranges) and Old World (parasites detected in the native range only) exhibited a significant relationship, a *post hoc* pairwise comparison Scheffe's test was performed. The results of this analysis showed that mean prevalence rates did not contrast statistically between Cosmopolitan and New World parasites, but did contrast significantly for Old World parasites. Furthermore, Old World only parasites exhibited lower mean prevalence rates as than their globalized or New World congeners (Figure

2.13).

DISCUSSION

Significantly increased infection rates, dissimilar parasite fauna and equivalent parasite richness in New World pigeons provides evidence that while parasite escape did occur upon initial colonization, parasite richness over time was replaced. This is not a novel phenomenon, and has been demonstrated among non-native amphibians and fish (Lebarbenchon et al. 2009). Rock Pigeons are an r-selected organism, with an average three year life expectancy and continuous year-round breeding cycle (Johnston and Janiga 1995; Newton 1998). Therefore, it is plausible that initial depauperate genetic variation was restored as a result of rapid population growth since New World invasion almost 400 years ago (Nei et al. 1975). Surprisingly, recent genomic sequencing of two U.S. free-ranging Rock Pigeons separated by almost 2,000 miles displayed a limited genetic variability incongruent with allopatric subpopulations (Shapiro et al. 2013). The resulting dendrogram closely clustered U.S. wild Rock Pigeons with domestic racing pigeons, substantiating the premise that escaped homers provide a constant infusion of genetic material into wild populations (Stringham et al. 2012). Given the reduced allelic diversity among wild pigeon in the United States, the melanistic polymorphisms present in free-ranging urban populations are surprising.

Comparable prevalence rates for Cosmopolitan and New World parasites did not correlate with results found by Blakeslee et al. (2012). In that study, introduced snails (*Ilyanassa obsoleta* and *Littorina saxatilis*) not only demonstrated reduced parasite species richness, but also lower mean prevalence rates of infection in the non-native sampling range. Differences in the results between studies may be attributed to time since invasion, synanthropic and ground foraging ecology of wild Rock Pigeons, in

contrast to aquatic infection dynamics, differences among propagule pressure and number, and the husbandry of pigeons by North American settlers as dispersal agents prior to escape (Mack et al. 2000; Colautti et al. 2004; Simberloff 2009).

While any or all of the interaction among these factors may play a role in the regulating parasite release or parasite inversion at the biogeographical level, prevalence rates are also likely to vary at the regional level, be it a native or assumed range (Ishtiaq et al. 2007). A survey performed on necropsied pigeons from the vicinity of Ankara, Turkey detected up to eight species of helminths among 15% of birds (Gıcık and Burgu 2000). In contrast, a maximum of six helminths in 3.84% of Atlanta birds were identified. Atlanta is located in the rock pigeon's assumed range, while Ankara is part of the native range, as identified by the IUCN. Thus, there are several plausible explanations for this dichotomy in observations. First, the helminth fauna in Ankara may be far more diverse due to climatic and habitat conditions, fostering a richer biodiversity of helminths, intermediate hosts or the preservation of infective larval stages. Secondly, the high prevalence of non-identifiable eggs and larvae (47.44% and 16.67%, respectively) in the Atlanta field study may have contributed to the reduced genus count. Another season for the reduced detection rate of helminth taxa among Atlanta birds may be attributed to differences in experimental design, specifically, McMaster egg counts were utilized in Atlanta birds while intact adult helminths were removed and taxonomically identified through the use of dichotomous keys in Ankara. In addition, although population genetics were not examined, the likelihood that wild pigeons established in Turkey prior to introduction into North America is possible due to the regional proximity of the endemic range, providing for the enemy release hypothesis. This antecedent colonization to North American establishment may have provided a

longer series of generations to develop a greater allelic richness of the MHC. Lastly, severe drought conditions affecting Georgia in 2012 may have impeded the infectious cycles for some helminths, particularly for trematodes, a class of helminths which utilize mollusks as intermediate or definitive hosts (Gérard 2001; Gibson et al. 2002; Karl et al. 2012).

The high prevalence and mean intensities of *Haemoproteus sp.* and *Plasmodium sp.* in Atlanta pigeons may be attributed to fitness trade-offs in breeding strategy in conjunction with the prevalence of the vectors, the Hippoboscid or louse fly for *Haemoproteus* and the ornithophilic mosquito *Culex quinquefasciatus*, a primary vector for avian *Plasmodium* in the southern United States (Campbell 1995; Allander 1997; Calhoun et al. 2007; Davis 2007). Over a three-year trapping period, Davis (2007) found that the prevalence of Hippoboscid flies in Atlanta began to rise in May, peaking in September, with a marked decline in October. *C. quinquefasciatus* followed a similar temporal distribution in prevalence in Tanyard Creek, a combined sewage overflow in Atlanta, approximately four miles from the rock pigeon field sampling sites. Results indicated that prevalence for *C. quinquefasciatus* rose in June, peaking in October, and sharply decreased in November (Calhoun et al. 2007). Prevalence rates for *Haemoproteus* sp. in Northern Florida (Alachua County) in 108 sampled wild Rock Pigeons reached 94%, closely mirroring prevalence rates of Atlanta pigeons at 98.5% (Forrester and Spalding 2003). An extensive search of the literature did not yield reports for *Plasmodium* sp. in Rock Pigeons in the southeastern United States, but did yield reports of *Plasmodium* among sympatric peridomestic passerines in Florida, also present within Atlanta field sites, such as House Sparrows with a prevalence of 30.3% (Marzal et al. 2011), Northern Mockingbirds, Blue Jays, and Brown-headed Cowbirds (*Mimus*

polyglottos, *Cyanocitta cristata* and *Molothrus ater*), unreported prevalence, and Northern Cardinals (Cardinalis cardinalis) with a prevalence of 8% (Forrester and Spalding 2003). *Plasmodium* sp. was also detected in Athens, Georgia, among the same distribution of peridomestic urban birds as far back as the early 1940's (Jordan 1943). Thus, it is altogether possible that Rock Pigeons have been harboring *Plasmodium* in Georgia since the early 20th century, since non-migrants allow chronic parasites to over-winter, but due to lack of sampling, was not detected. The highest *Plasmodium* sp. prevalence reported in Rock Pigeons in a non-native nation, Nigeria, reported an infection rate of 30% over fifty sampled birds (Dadi-Mamud et al. 2012). The lack of a statistically significant difference among blood parasite infections in AHY and HY birds was unexpected, as vectors have experimentally demonstrated higher feeding preferences upon HY pigeons (Kartman 1949). Conversely Sol et al. (2000) found that the intensity of infection did not covary with vector abundance, although AHY birds exhibited a higher prevalence of infection. Surprisingly, although Sol et al. (2000) found that greater numbers of AHY birds tended to be infected with blood parasites, on the other hand, when HY birds did become infected, the infections in HYs exhibited greater intensities of infection than in AHYs. In a follow-up study, Sol et al. (2003) postulated that although some selection occurred in HY birds as a result of elevated intensities of infection, the development the immunity to chronic blood parasitism as HY birds transitioned to AHY birds served as a parsimonious explanation to those observations. This idea concurs with a gross pathological examination of the immune system in juvenile Rock Pigeons, which established that full development of the immune system did not arise until four months of age (Selvaraj and Pitchappan 1988).

APMV-1 shedding rates were extremely high, although the duration of viral

shedding varies across taxa (Seal et al. 2000). Among adult chickens surviving viral challenge, antibodies subsequently provided humoral immunity against subsequent inoculation, and NDV antibodies have a known efficacy of a minimum of 4.5 months (Heuschele and Easterday 1970; Gelb et al. 1987). NDV viral particles excreted with the feces can remain infectious for several months (Ritchie and Carter 1995), and has been documented replicating within chicken macrophages and lymphocytes, systematically inducing cellular apoptosis in addition to necrotizing heterophils (Lam 1996; Qureshi et al. 2000). Chickens challenged with both phylogenetic and antigenic variants of NDV vaccines shed for a week post-inoculation, until day nine when shedding was generally reduced (Miller et al. 2007). Although a paucity of data exists on viral shedding rates of NDV in wild Rock Pigeons, a field study of migratory geese found that avian influenza (AIV) reached proportions of 1 to 3% during the breeding season (Ely et al. 2013). In contrast, the 15.28% viral shedding rate in Atlanta Rock Pigeons is extremely high in comparison. Presently, as the mode of transmission or the reservoir of the virus remains unknown, further examination to disentangle transmission dynamics of APMV-1 in Atlanta Rock Pigeons is in pursuit for investigation.

The documentation of infection rates among exotic avian species is relevant to not only understanding the role of exotics in disease ecology, but also the role that exotic avian species may play in the conservation of avian community species richness. Unlike secondary cavity nesting House Sparrows, platform nest-building Rock Pigeons do not displace native avifauna due to their anthropomorphic site faithfulness and propensity to nest on buildings and highway bridges (Houser 1996; Tietz Marques et al. 2007; Bonter et al. 2010). On the contrary, the role of free-ranging pigeons as a primary food source has been implicated in the conservation of the Peregrine Falcon (*Falco peregrinus*) in

urban sites (López-López et al. 2009). Consumption of wild pigeons by raptors does not come without disease implications, such as the transmission of columbid herpesvirus-1 in Cooper's Hawks (*Accipiter cooperii*) and *Trichomonas* sp. (Boal and Mannan 1999). Interestingly enough, genotyping of *Trichomonas* among raptors and wild pigeons in eastern Spain found that one genotype was primarily detected in wild pigeons, while a second genotype was detected among raptors, although some overlap did occur (Sansano-Maestre et al. 2009). This data may provide evidence for a second look into the responsibility of wild pigeons as transmission agents of novel pathogens into native birds. In light of the results for parasite inversion amongst non-native Rock Pigeons, the literature review data may support that disease transmission into native avifauna may stem from parasites already present in their native range. Furthermore, although the high prevalence and amplified intensities of blood parasites found in Atlanta birds may be a theoretical cause for conservation concern, the high host specificity of *Haemoproteus* sp. and *Plasmodium* sp. make it an unlikely scenario that these malarial parasites exhibit an increased transmission gradient to native Georgia birds (Valkiūnas et al. 2013). Continued surveillance and molecular work performed on the parasites of free-ranging pigeons may provide more substantiated evidence upon the invasion success of exotics, parasite release and gain, and the conservation implications of exotic species establishment into native community biota.

FIGURES AND TABLES

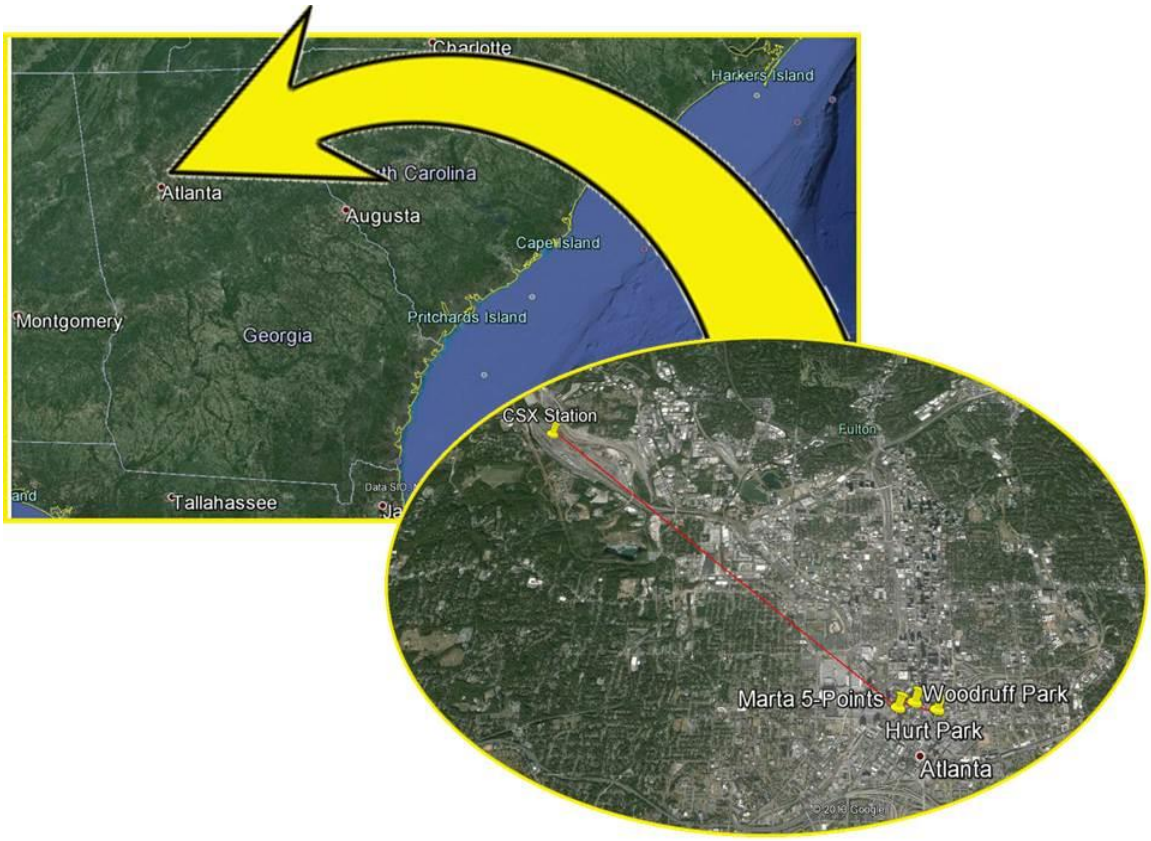


Figure 2.1. Sampling sites for the Atlanta, Georgia 2012 field season. Red lines in the inset figure represent distances in kilometers. The three clustered sites in the south of Fulton county, Georgia (Marta 5 Points, Woodruff Park and Hurt Park had a maximum distance 0.20 km from one another, while the CSX Station site equaled 7.84 km from Marta 5 Points. All sites were treated as subsampling sites for a single population of pigeons due to an average home range of 10 km for wild pigeons (Houser 1996).

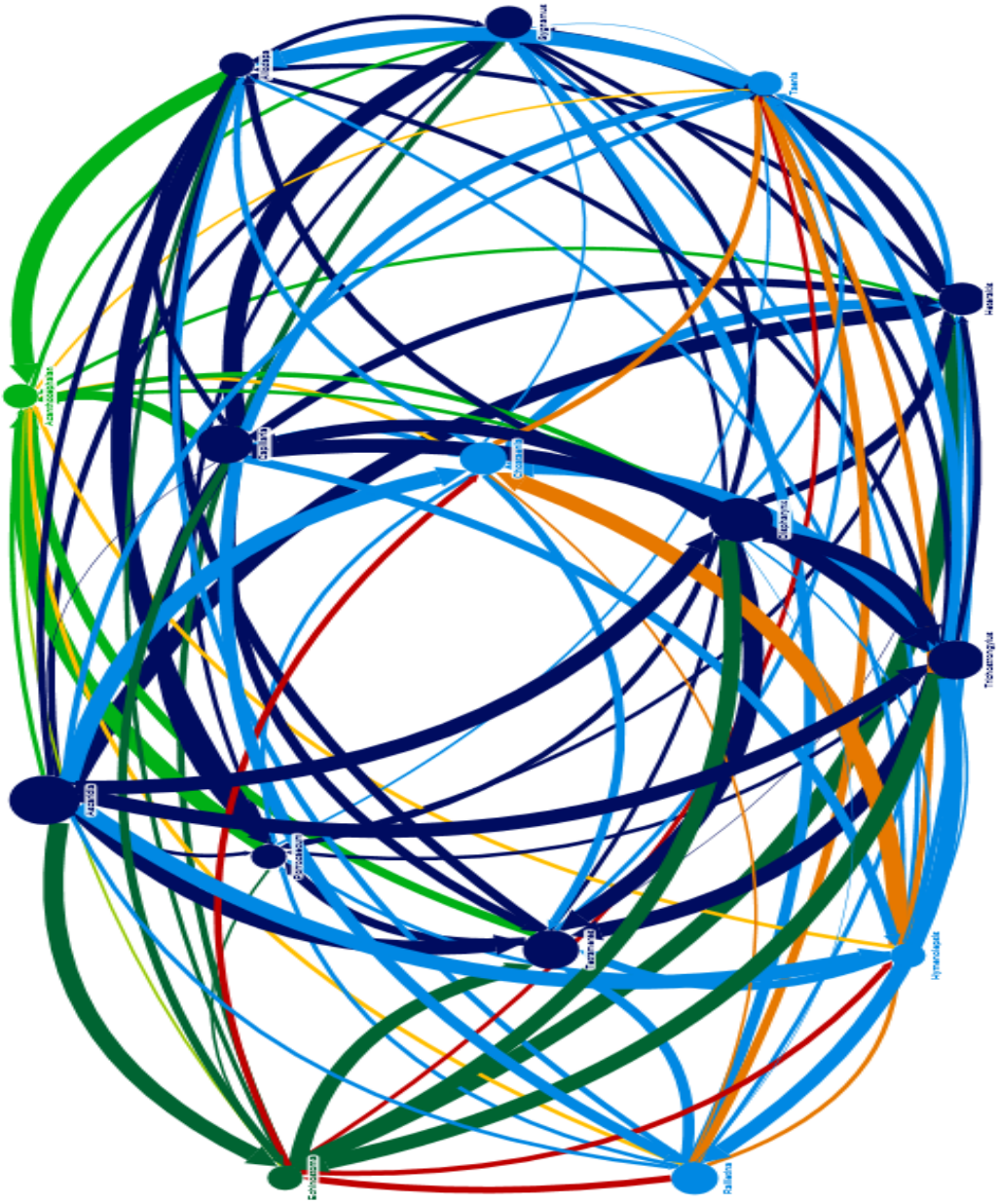


Figure 2.2. Correlation coefficient network plot for Atlanta Rock Pigeon helminths. Node size corresponds to helminth prevalence, while the thicknesses of lines correspond to the degree of correlation among helminth types. Dark blue lines = nematode to nematode correlation, light blue lines = nematode to cestode relationships, orange lines = cestode to cestode relationships, green lines = trematode to nematode relationships, red line = trematode to cestode relationships, light green lines = acanthocephalan to nematode relationships, and yellow lines = acanthocephalan to cestode relationships. **Prevalence:** *Ascaridia* sp. (84.62%), *Echinostoma* sp. (5.13%), *Railletina* sp. (10.26%), *Tetrameres* sp. (17.95%), *Trichostrongylus* sp. (23.08%), *Hymenolepis* sp. (6.41%), *Porrocaecum* sp. (1.28%), *Choataenia* sp. (12.82%), *Dispharynx* (44.87%); *Capillaria* 26.92%), *Allodapa* sp. (2.56%), *Syngamus* (12.82%), *Taenia* sp. (3.85%), *Heterakis* sp. (11.54%), *Acanthocephalan* sp. (1.28%).

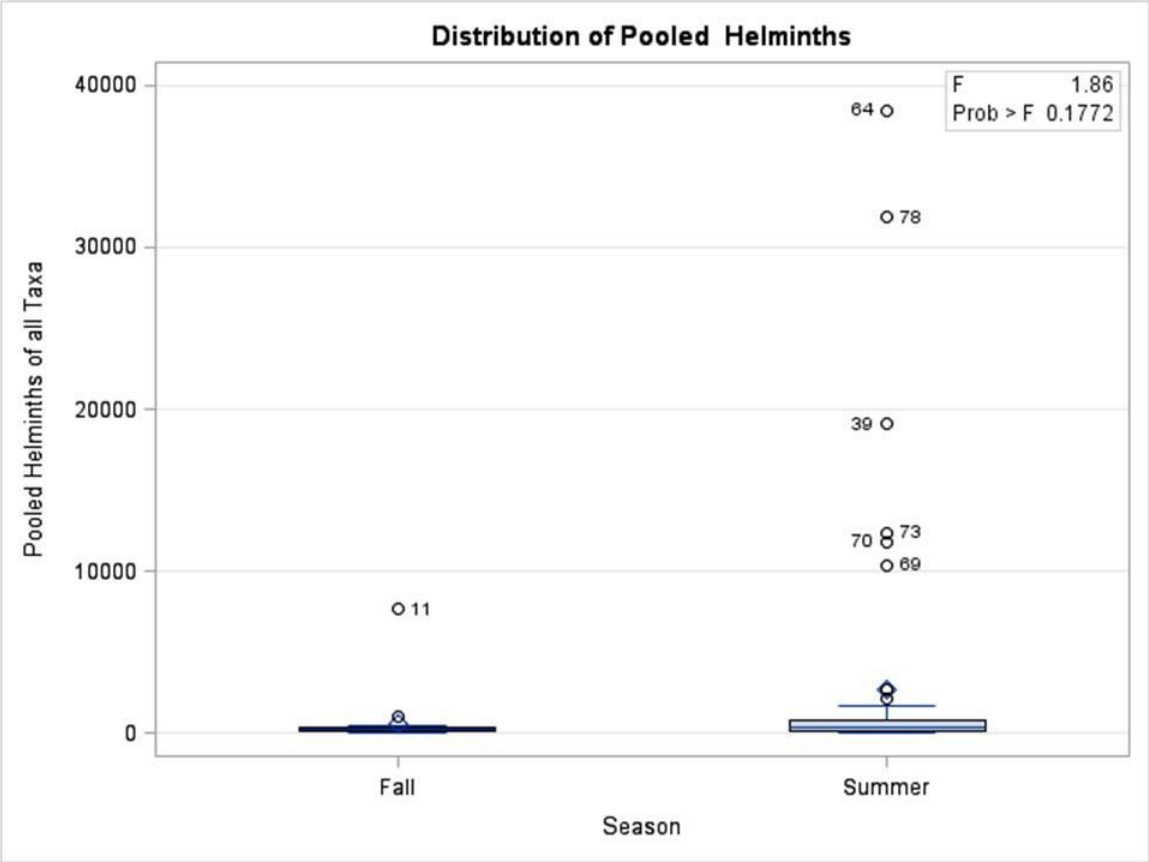
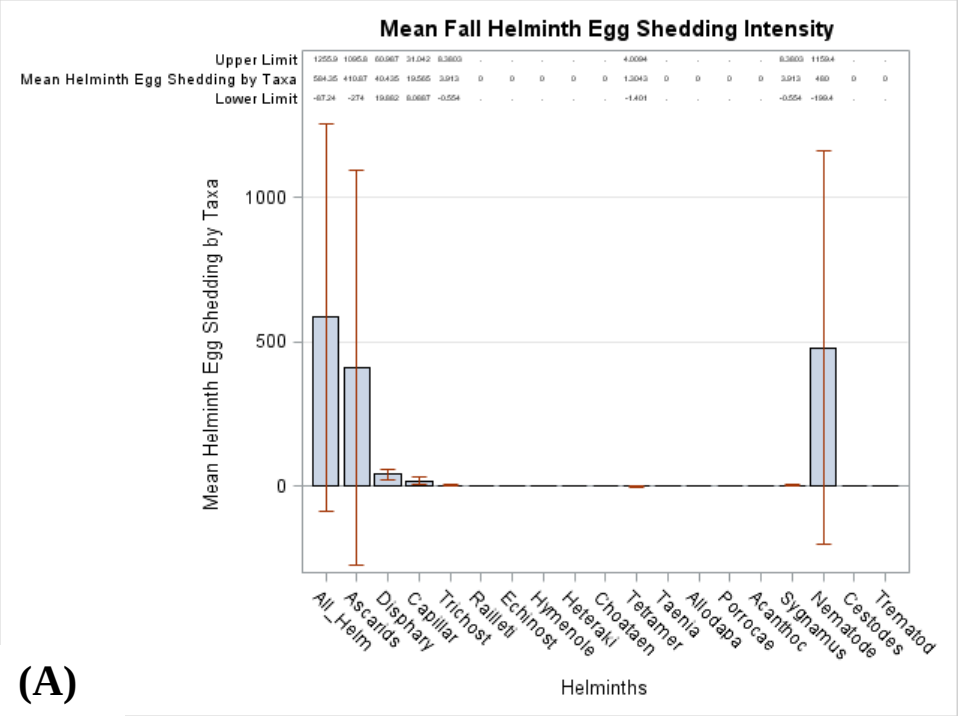
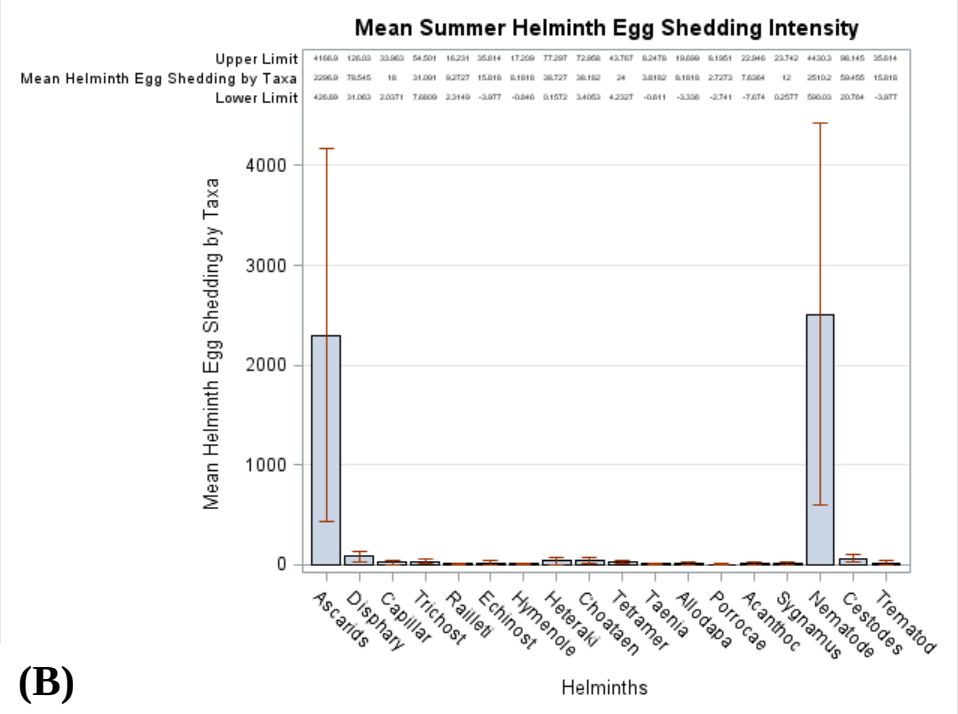


Figure 2.3. Distribution of pooled helminths among Atlanta Rock Pigeons. Non-log transformed data demonstrated no statistically significant seasonal effect among helminth egg shedding, although the summer sampling period exhibited a generalized increase in overall eggs shed.



(A)

Figure 2.4A. Mean fall season egg shedding. Nematodes and sub-taxa such as *Ascaridia* sp., *Dispharynx* sp., *Capillaria* sp., *Trichostrongylus* sp., and *Tetrameres* sp., were the only helminth eggs detected in the fall.



(B)

Figure 2.4B. Mean summer season egg shedding. Although nematodes dominated in prevalence during the summer as well, the species richness of parasites was more diverse for eggs detected.

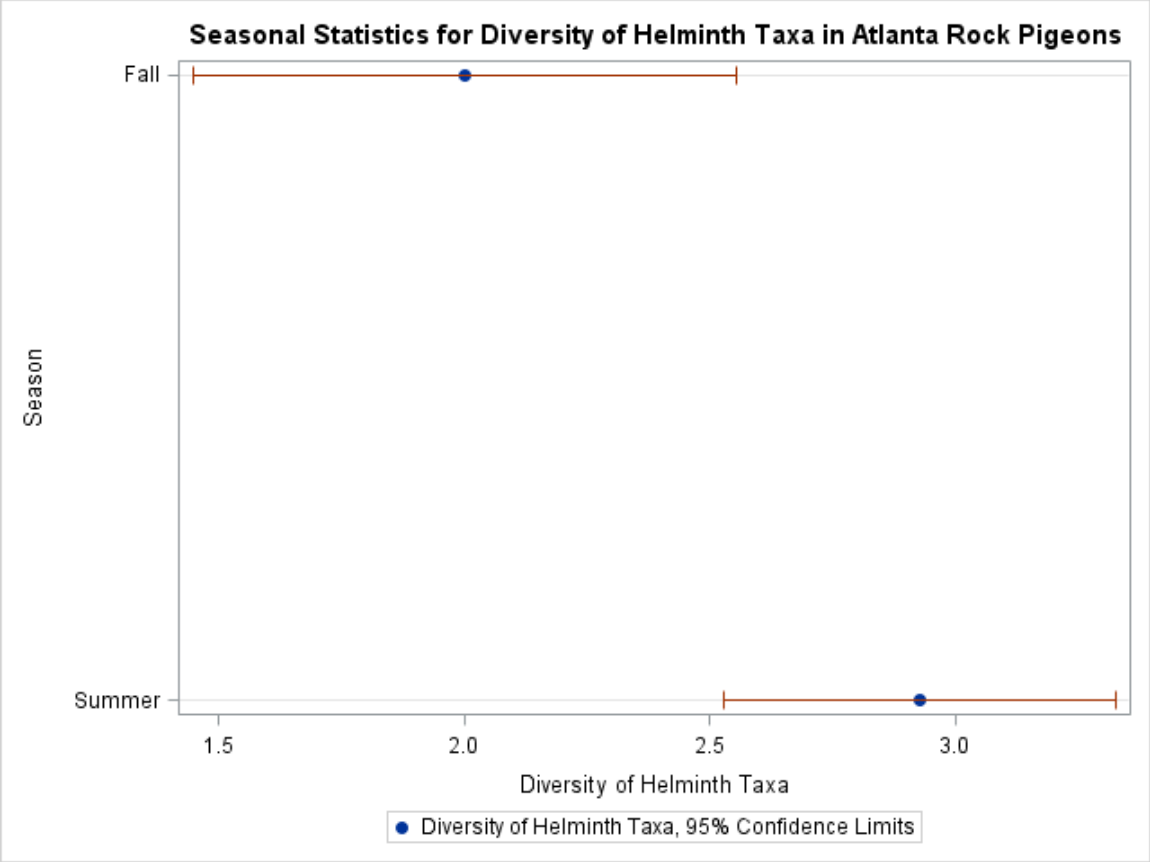


Figure 2.5. Seasonal helminth diversity. Pigeons hosted a statistically significantly greater helminth richness in the summer as opposed to the fall (df = 77, F = 6.89, p = 0.0104).

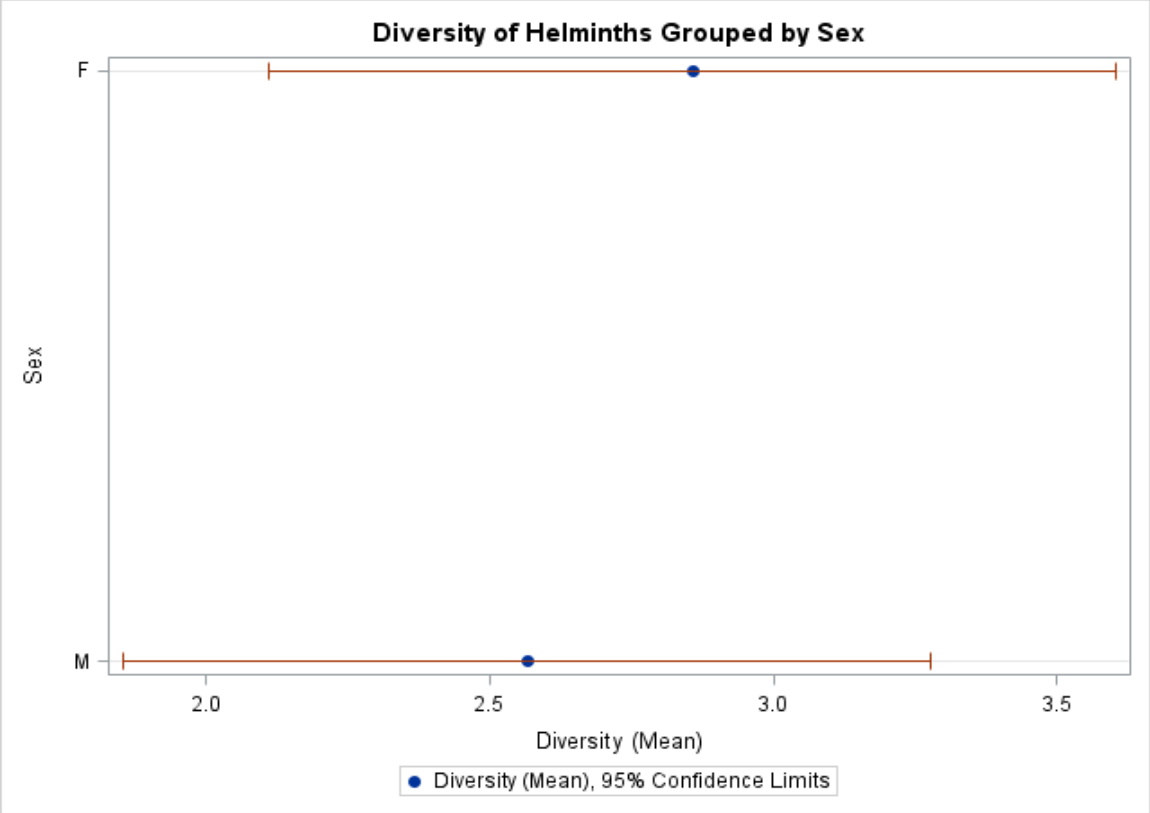


Figure 2.6. Diversity of helminths by gender. Likely due to the low degrees of freedom, helminth diversity did not covary by gender, although females tended to carry a greater richness of helminths than their male counterparts (df = 36, F = 0.32, p = 0.57)

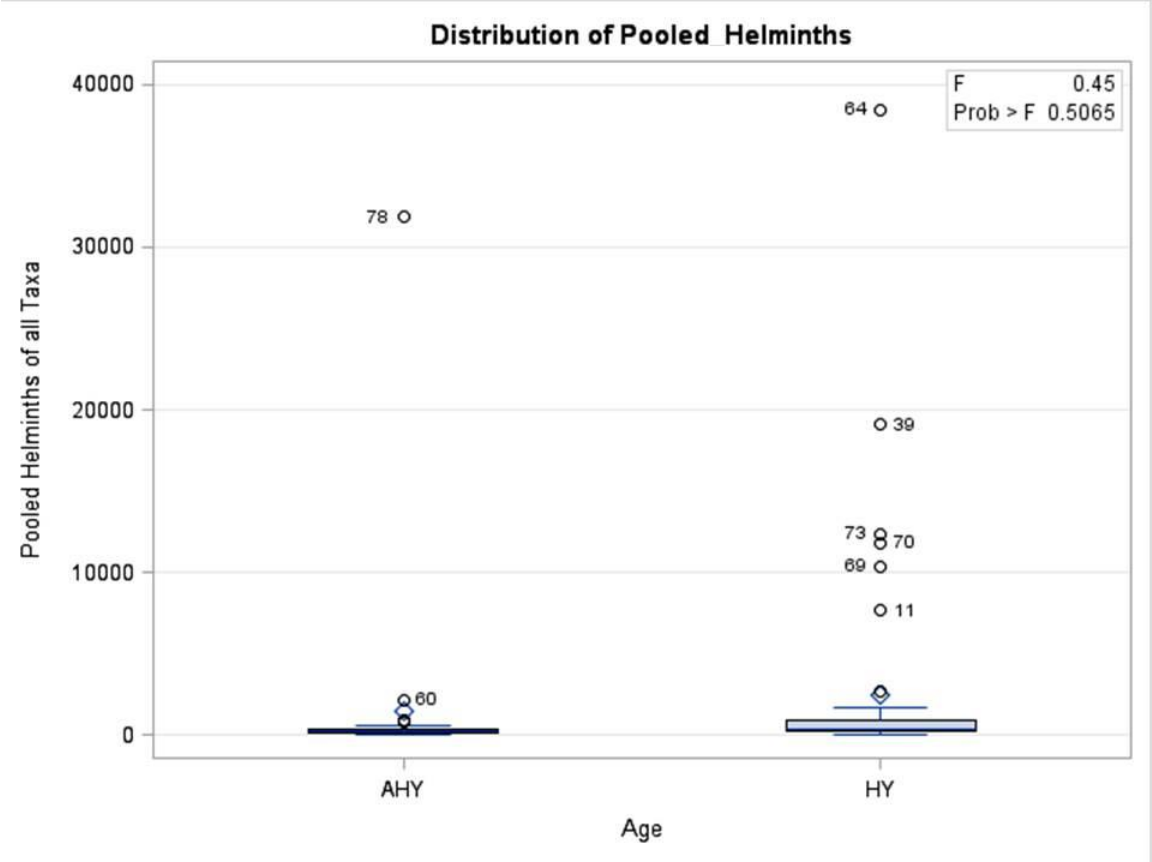


Figure 2.7. Helminth distribution among age classes. No statistical differences among age classes of birds were evident, although HY birds did generally shed more eggs. The variance in the data is more pronounced for HY birds, demonstrating that host heterogeneity may be influenced by age.

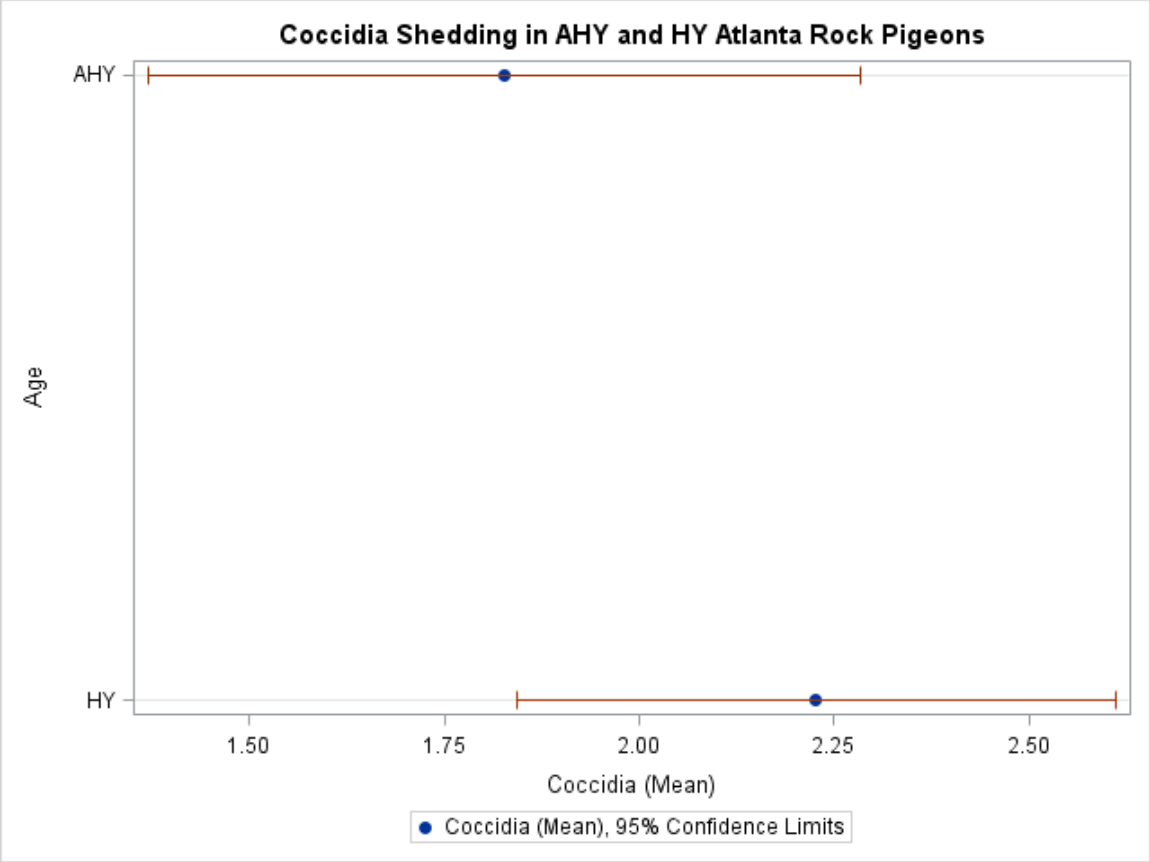


Figure 2.8 Dynamics of coccidia shedding according to age. Utilizing log-transformed data, HY bird statistically shed more coccidia oocytes than their AHY counterparts (df = 74, F = 3.7837, p = 0.0274).

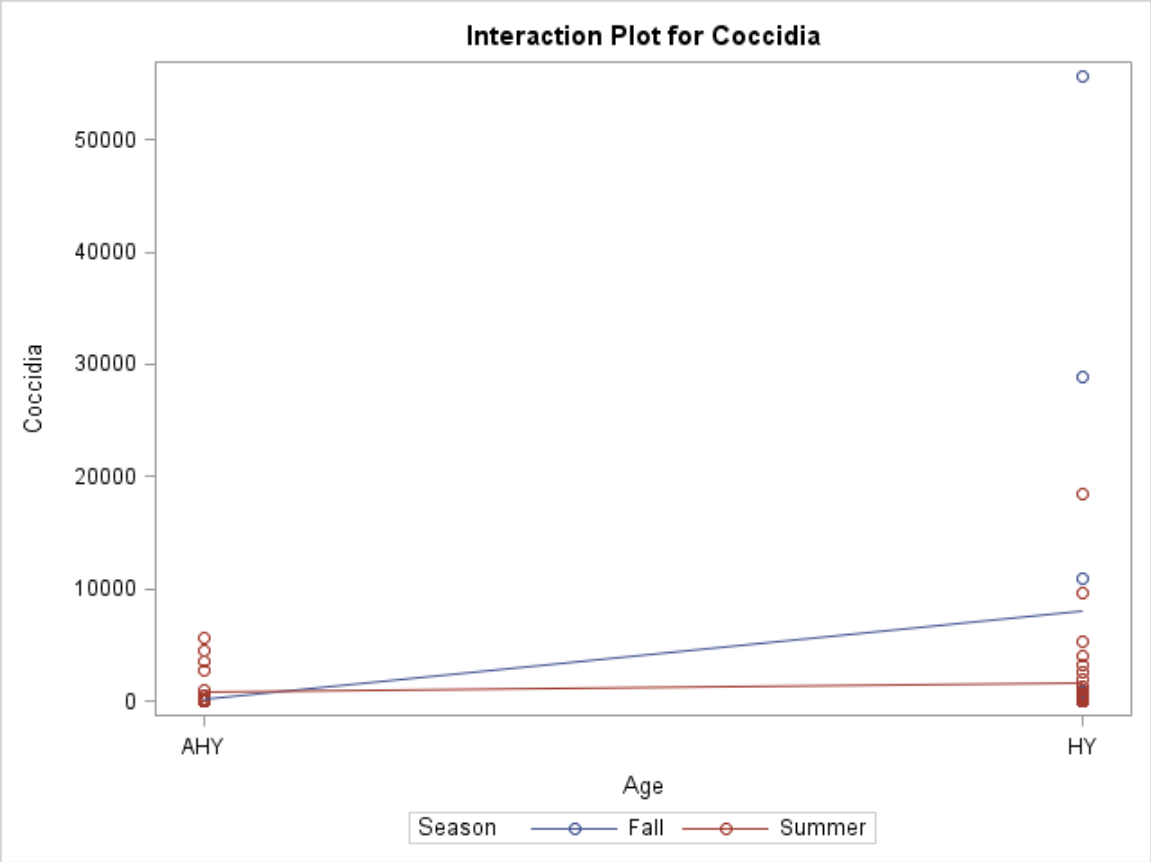


Figure 2.9. Interaction of Coccidia Shedding by Age and Season. The interaction between season and age demonstrates that AHY birds statistically shed more Coccidia oocytes in the fall, while HY birds tended to shed relatively equal densities of oocytes each season (df = 72, F = 2.99, p = 0.0370)

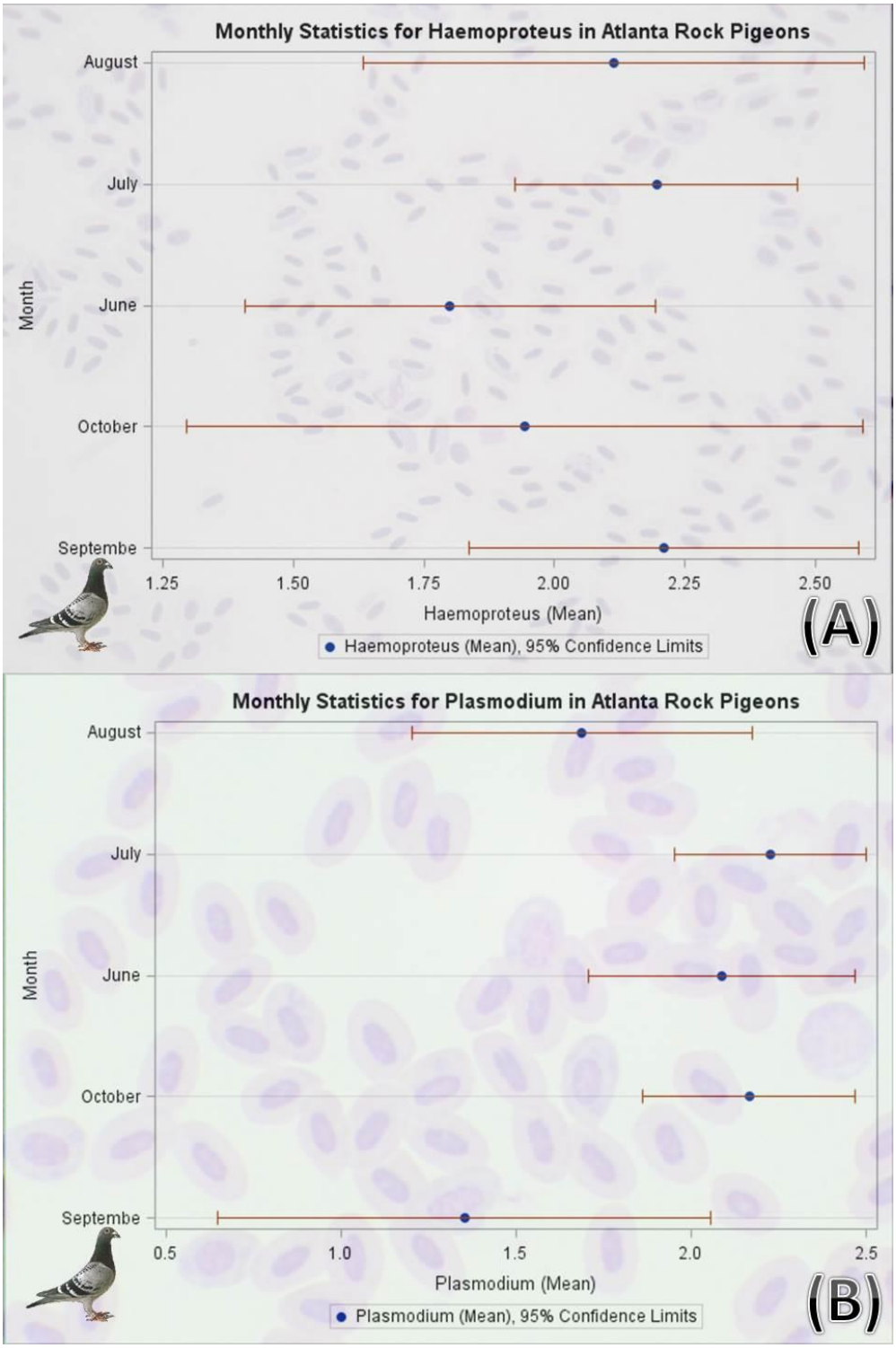


Figure 2.10. A,B. *Haemoproteus* sp. and *Plasmodium* sp. Monthly Distributions Of Infection. *Haemoproteus* sp. was not statistically significant according to season or month, while *Plasmodium* sp. was statistically significant with an increased intensity in June and the lowest intensity in September**.**

Table 2.1. Helminth summer descriptive statistics for Atlanta Rock Pigeons.

Season	N	Helminth Taxa	Lower 95% CL	Upper 95% CL	Mean	Std Dev	Std Error
Summer	55	Pooled Helminths	711.4637832	4662.35	2686.91	7307.32	985.3188266
		Ascarids	426.8862453	4166.93	2296.91	6917.35	932.7358792
		Dispharynx	31.0630902	126.0278188	78.5454545	175.6408181	23.6834031
		Capillaria	2.0370887	33.9629113	18	59.0480031	7.9620311
		Trichostrongylus	7.6808549	54.5009633	31.0909091	86.5955419	11.6765405
		Railletina	2.3148784	16.2305762	9.2727273	25.7376037	3.4704578
		Echinostoma	-3.9771564	35.61352	15.8181818	73.2244371	9.8735811
		Hymenolepis	-0.8458133	17.2094497	8.1818182	33.3938844	4.5028305
		Heterakis	0.1571945	77.2973509	38.7272727	142.673605	19.238105
		Choataenia	3.4053076	72.9583287	38.1818182	128.6409144	17.3459374
		Tetrameres	4.2326506	43.7673494	24	73.1209044	9.8596207
		Taenia	-0.6114413	8.2478049	3.8181818	16.3855073	2.2094213
		Allodapa	-3.3358172	19.6994536	8.1818182	42.604595	5.7448024
		Porrocaecum	-2.7405799	8.1951253	2.7272727	20.2259959	2.7272727
		Acanthocephalan	-7.6736237	22.9463509	7.6363636	56.6327884	7.6363636
		Sygnamus	0.2576648	23.7423352	12	43.4357763	5.8568789
		Nematodes	590.0278996	4430.34	2510.18	7102.79	957.7404135
		Cestodes	20.764077	98.1450139	59.4545455	143.1189376	19.2981536
		Trematodes	-3.9771564	35.61352	15.8181818	73.2244371	9.8735811

Table 2.2. Helminth fall descriptive statistics for Atlanta Rock Pigeons.

Season	N	Helminth Taxa	Lower 95% CL	Upper 95% CL	Mean	Std Dev	Std Error
Fall	23	Pooled Helminths	-87.2448396	1255.94	584.3478261	1553.06	323.8349907
		Ascarids	-274.0162244	1095.76	410.8695652	1583.8	330.2447966
		Dispharynx	19.8822138	60.9873514	40.4347826	47.5278159	9.9102347
		Capillaria	8.0886544	31.0417803	19.5652174	26.5395521	5.5338792
		Trichostrongylus	-0.5542026	8.3802895	3.9130435	10.3305066	2.1540595
		Railletina	.	.	0	0	0
		Echinostoma	.	.	0	0	0
		Hymenolepis	.	.	0	0	0
		Heterakis	.	.	0	0	0
		Choataenia	.	.	0	0	0
		Tetrameres	-1.400704	4.0093997	1.3043478	6.2554324	1.3043478
		Taenia	.	.	0	0	0
		Allodapa	.	.	0	0	0
		Porrocaecum	.	.	0	0	0
		Acanthocephalan	.	.	0	0	0
		Sygnamus	-0.5542026	8.3802895	3.9130435	10.3305066	2.1540595
		Nematodes	-199.4307764	1159.43	480	1571.18	327.6144461
		Cestodes	.	.	0	0	0
		Trematodes	.	.	0	0	0

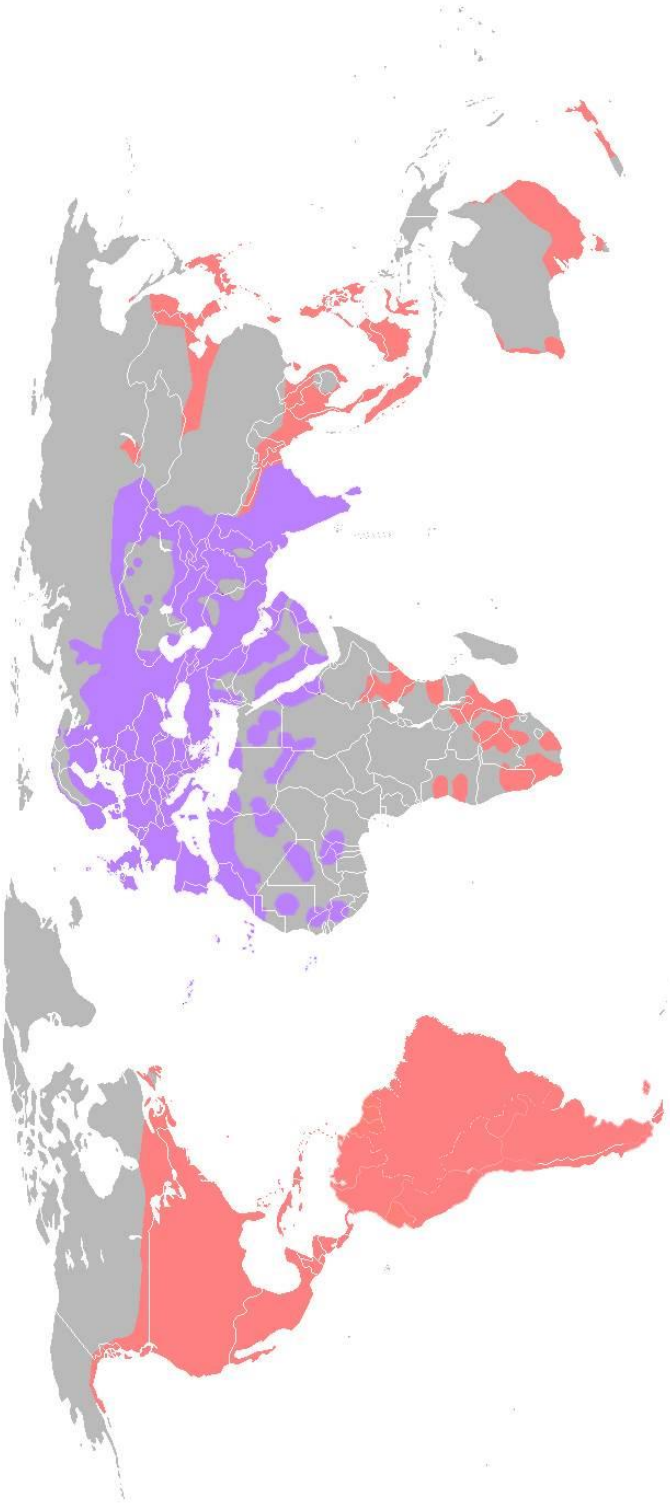


Figure 2.11. Endemic and non-native range of Rock Pigeons. Pink areas represent areas of introduction modified to include areas of establishment as identified by review of the literature and the IUCN, while purple areas represent the assumed native range as reported by the IUCN. Modified from http://maps.iucnredlist.org/map.html?id=106002444.

Parasites Shared In Old World and New World				
Range Distribution	**Frequency**	**Percent**	**Cumulative Frequency**	**Cumulative Percent**
Not shared	38	55.07	38	55.07
Shared (Overlap)	31	44.93	69	100.00

Table 2.3. Distribution of endoparasites among New and Old World pigeons. The overlap of parasites found in both native and non-native ranges did not equal the distribution of parasites found across disparate ranges.

Parasites found in New World, Old World or Both Ranges				
Location	**Frequency**	**Percent**	**Cumulative Frequency**	**Cumulative Percent**
Cosmopolitan	31	44.93	31	44.93
New	19	27.54	50	72.46
Old	19	27.54	69	100.00

Table 2.4. Frequency Table of Rock Pigeon Parasites Distributed Across Native, Cosmopolitan and Assumed Ranges.

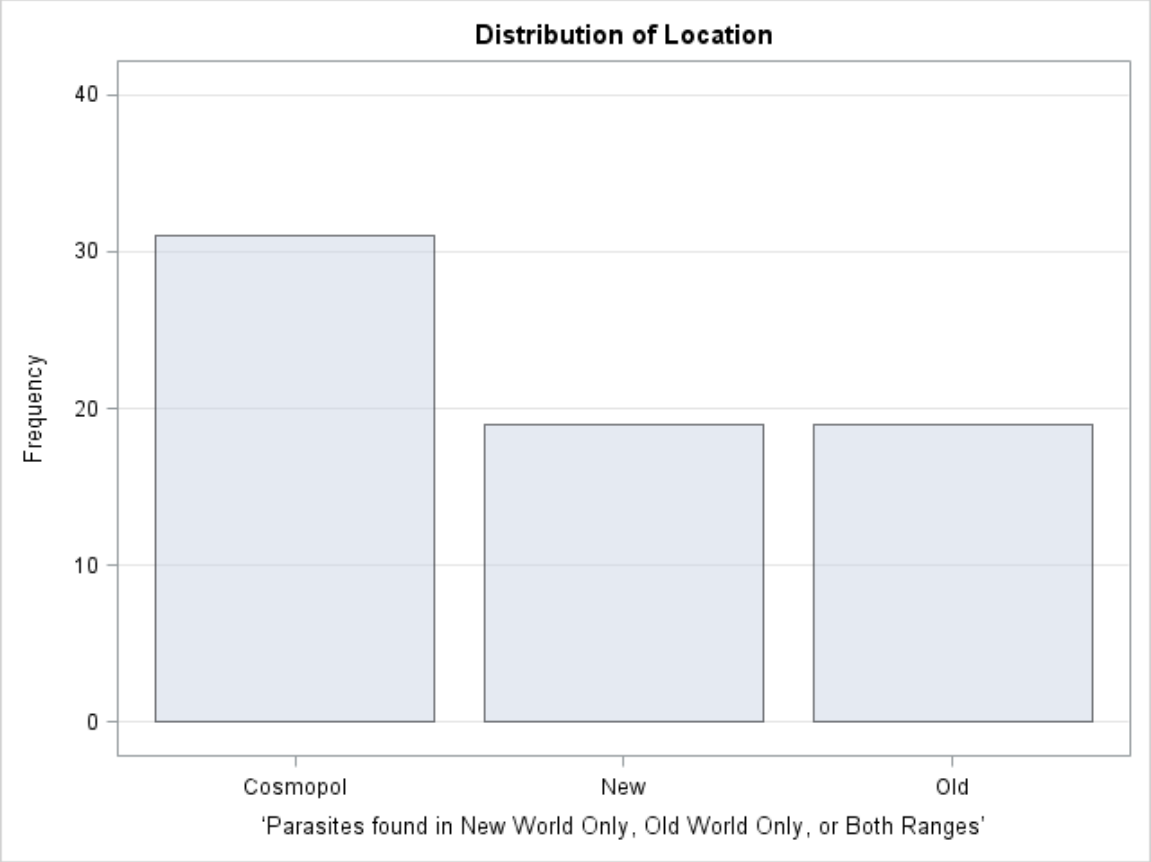


Figure 2.12. Distribution of selected endoparasites distributed across ranges. The Cosmopolitan range encompasses endoparasites harbored by pigeons in both the New and Old World, while parasites found in the New World category or in the Old World category do not overlap.

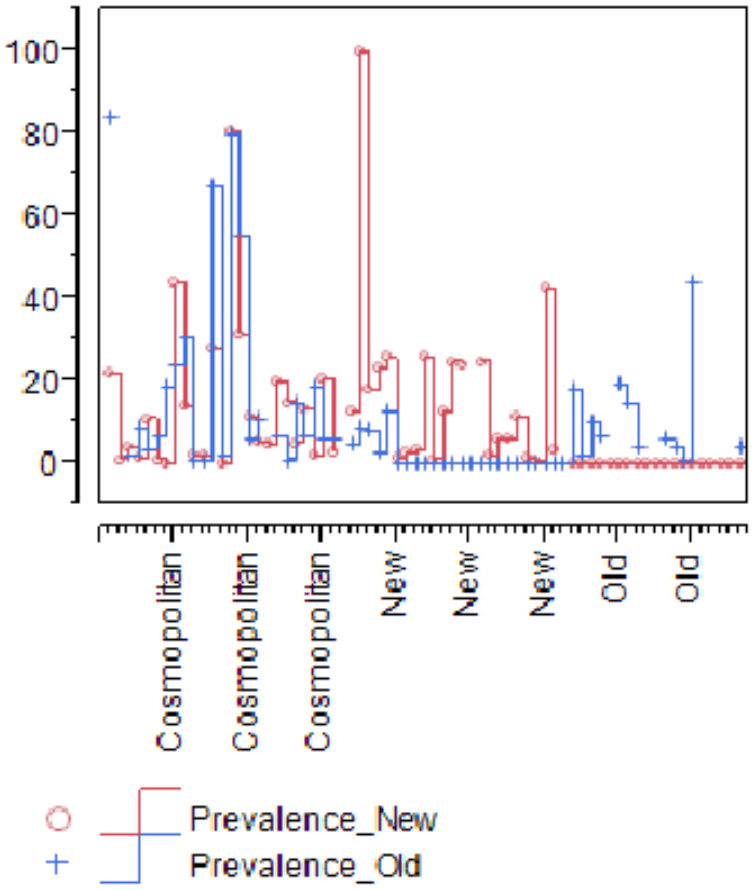


Figure 2.13. Overlay plot for parasite release MANOVA. Pillai's Trace (0.302), F = 4.80, p = 0.0013 for prevalence rates among New World, Old World and Cosmopolitan Parasites strongly suggest that while some degree of parasite inversion, as opposed to release, has occurred, intensities of infection are still generally increased among assumed and the Cosmopolitan range hosting generalist parasites than for Old World parasites.

CHAPTER 3

MELANISM, BODY CONDITION, AND PARASITE SUSCEPTIBILITY IN A POPULATION OF ATLANTA, GEORGIA ROCK PIGEONS [2]

[2] Ayala, A.J., C.R. Becker, S.M. Hernandez, and V.O. Ezenwa. To be submitted to *Avian Diseases.*

ABSTRACT

Melanism and plumage polymorphisms in birds have fascinated avian naturalists and ecologists for centuries. Here we present potential selective advantages for maintaining phenotypic diversity among wild Rock Pigeons in an urbanized sampling location in response to parasitism. For all helminths tested, only *Ascaridia* sp. and pooled nematodes demonstrated a statistically significant effect for reduced disease prevalence among eumelanistic pigeons. Blood parasite analysis did not yield statistically significant effects in response to melanism, although melanistic birds conversely harbored increased mean intensities of *Haemoproteus* sp. and *Plasmodium* sp. To further investigate potential covariates among melanistic and paler birds, body condition, nutritional status, and hematocrits were also assessed.

INTRODUCTION

Hosts, Parasites, Melanism and Body Condition

The epidemiological consequences of parasites may be strong evolutionary drivers, as selection pressures between hosts and parasites are dualistic in nature (Hudson 2005). As parasites exert selective forces upon host populations, hosts respond to parasitism via immune and fitness trade-offs, maintaining vigor in heterozygosity across normally distributed populations (Morand 2000). Conversely, as parasite selective forces favor one life-history trait, such as immunity, that investment may come at a cost to reproduction, growth or other metabolic processes (Sheldon and Verhulst 1996; Lochmiller and Deerenberg 2000). Host phenotypes may also be shifted as a result of parasitism, and if the phenotypic change is directly related to genotype expression, then parasite mediated phenotypes may induce heritable traits (Poulin and Thomas 1999). The role of melanism in parasite susceptibility has also long been recognized as dualistic in

nature. Parasitism may exert selective pressure upon light-colored individuals, while surviving host generations therefore express darker-colored phenotypes (Roulin 2004). The caveat is that the genetic mechanism controlling this process has not been isolated within each model system. Invertebrates are widely used to examine the relationships between melanism and immunity (Wilson et al. 2001; Rolff and Siva-Jothy 2003; Cotter et al. 2004; Armitage and Siva-Jothy 2005; Lee et al. 2008), but this provides a special conundrum for vertebrate ecoimmunologists as insects have only an innate immune system while vertebrates have the added complexity of an adaptive immune system (Jacobs and Zuk 2011). Among tested insect taxa, the enzyme phenyloxidase is a crucial constituent of color synthesis and is thus considered the key player in host parasite susceptibility (Cotter et al. 2008; González-Santoyo and Córdoba-Aguilar 2012). For instance, melanistic greater wax moths (*Galleria mellonella*) exhibited increased resistance to the fungal pathogen *Beauveria bassiana* in contrast to non-melanistic cohorts, where resistance was measured by survivorship rates among color phases (Dubovskiy et al. 2013). Melanistic African armyworm larvae (*Spodoptera exempta*) exposed to the ectoparasitoid *Euplectrus laphygmae* and *Beauveria bassiana* exhibited a number of superior life-history attributes in contrast to their paler counter parts. These attributes included increased phenyloxidase activity, increased survivorship when challenged with *Beauveria bassiana*, and increased melanization (e.g. cuticle circulation, thereby rendering the eggs un-viable) of *Euplectrus laphygmae* eggs (Wilson et al. 2001).

Within vertebrates, pleiotropic effects of the highly conserved Melanocortin-1-receptor (MC1R) gene, is hypothesized to exercise regulation of immune function and the expression of coloration (Mundy 2005; Ducrest et al. 2008). MC1R has been isolated from felids (Eizirik et al. 2003), rock pocket mice (*Chaetodipus intermedius*) (Nachman

et al. 2003), canids (Owczarek-Lipska et al. 2013), 2013) and humans (Hattori et al. 2010). MRC1 has also been isolated in a variety of avifauna, with point mutations for polymorphic expression arising independently among the Aves order (Baião et al. 2007). Given that, plumage polymorphisms among bird species serve as ideal models in which to test trade-offs between melanism and immune function. A seminal paper by Hamilton and Zuk (1982) postulated that blood parasites exude a selective effect strong enough to advertise the optimality of avian male mates via color-based ornamentation during the breeding season. Thus, investment in immune function does not come without cost, as both pigmentation and immunity are metabolically expensive. Reproductive output and sexual ornamentation, among other life-history traits, may be diminished as physiological resources are re-allocated to immune function as a result of pathogen assault (Saino et al. 1997; Norris and Evans 2000; Hanssen et al. 2003; Hanssen et al. 2005).

Color may also be influenced by nutritional and physiological status. In carotenoid-based pigmentation, body mass and nutritional condition have also been associated with the efficacy of immune function (Tella et al. 2001). For instance, House finches (*Carpodacus mexicanus*) develop a deeper intensity of carotenoid pigmented feathers as a consequence of foraging efficiency (Hill 1990). As the pale yellows to bright reds are not synthesized *de novo,* but must instead derive from diet, more colorful males are more attractive to potential mates. Carotenoid pigments may be more than a signal of efficient foraging; they may also directly affect the immune response. The role of carotenoid based bill coloration in Zebra Finches (*Taeniopygia guttata*) in immunocompetence was further investigated by McGraw and Ardia (2003). Zebra Finches artificially supplemented with carotenoid pigments exhibited increases in both cell-mediated and humoral immunity in contrast to the control group as challenged via the

phytohemagglutinin antigen (PHA) and the sheep red blood cell (SRBC) tests. Removal of the helminth parasite *Trichostrongylus tenuis* from Red Grouse increased plasma concentrations of circulating carotenoids, necessary for sexual signaling in males (Martínez-Padilla et al. 2007). Surprisingly, this is not a phenomenon limited to birds or simply status signaling; in fighting fish *(Betta splendens)* concentrations of carotenoid pigmentation have also positively correlated to immune function (Clotfelter et al. 2007). However, as carotenoids are not a function of genetically driven phenotype plasticity, diet derived melanism does not provide an analogous explanation for the relationship between melanism, heritability, and parasite susceptibility (Jacobs and Zuk 2011).

Male Common Yellowthroats (*Geothlypis trichas*) exhibit both a melanistic black face mask, as well as a carotenoid-based yellow bib, during the breeding season, although in juvenile or hatch-year (HY) birds the black mask is relatively non-existent (Sibley 2000; Roy 2009). Like most neotropical migratory warblers, male Common Yellowthroats undergo a yearly pre-basic and pre-alternate molt (Francis and Wood 1989; Pyle 1997). Longitudinal analysis of breeding success in response to concentrations of IgY among disparate breeding populations (Central and Atlantic flyway) found that while carotenoid coloration correlated to humoral immunity of breeding males in New York, the size of the melanistic facial mask correlated to humoral immunity in Wisconsin (Dunn et al. 2010). Experimental removal of *Leucocytozoon* sp., a highly lethal blood parasite within the Anatidae, in color polymorphic Tawny Owls (*Strix aluco*), found that among anti-malarial treated owls, the paler grey owls experienced profound benefits through increases in body mass as opposed to their darker brown cohorts (Shutler et al. 1999; Karell et al. 2011). In the context of chroma-ecoimmunology, the results of this study infer that brown Tawny Owls did not benefit as

greatly from their grey counterparts due to already elevated immune resources.

Body condition has often correlated with immunocompetence (Saino et al. 1997; Owen and Moore 2006; Ardia et al. 2010; Wilcoxen et al. 2010), and ultimately nutrition (Alonso-Alvarez and Tella 2001). Thus, costly trade-offs between life-history traits and immune defense are often physiologically necessary during host parasite establishment (Gonzalez et al. 1999). For instance, the sample size of House Finches exhibiting clinical signs of *Mycoplasma gallisepticum* in molt was not only substantially smaller, but they also exhibited increased numbers of circulating eosinophils, associated with parasitic infections and tissue damage (Campbell 1995; Davis et al. 2004). Thus, analogy suggests that pigeons with increased parasite burdens would demonstrate poorer body condition, as well as smaller growth bars, as a result of resource reallocation to parasite invasion.

The Model System

Overall, we examined dynamics between melanism, nutrition, and body condition utilizing novel techniques in a population of Rock Pigeons in downtown Atlanta, Georgia. Due to widely studied and easily discernible color polymorphisms, Rock Pigeons (*Columba livia*) are an excellent model system to assess correlations between melanism, body condition, nutrition, and parasite susceptibility. A prior correlative study in wild pigeons examining immunocompetence across degrees of melanism found that darker morph individuals mounted a stronger response to phytohemagglutinin (PHA) challenge, and overall had reduced blood parasite intensities (Jacquin et al. 2011). The PHA test has been reliably and widely used with numerous taxa under field conditions, including birds, as a measure of T-cell mediated (acquired) immunity (Tella et al. 2008). Therefore, we predicted that darker morph birds would harbor a decreased intensity of parasites, exhibit increased body condition controlled for size, and have wider mean

growth bars in collected retrices (tail feathers) as an indicator of nutritional status.

MATERIALS AND METHODS

Capture and sampling of wild Rock Pigeons

Samples were analyzed from wild Rock Pigeons were captured within metro Atlanta, Georgia between May and October 2012. From May – July, pigeons were caught at three urban sites in Fulton County, GA, while from August – October 2012, pigeons were captured by the USDA-APHIS Wildlife Services personnel in Fulton County as part of their integrated wildlife damage management program (USDA 2009) (Figure 2.1). Pigeons were captured during daylight hours near roost and feeding sites using a combination of hand-nets, drop-nets, mist-netting, and ground traps. During the May-July capture period, all released individuals were banded with a unique four band-color combination to avoid pseudoreplication. For individuals captured twice, collected data was only scrutinized if sampling on the first attempt did not produce analyzable samples.

For each bird, we recorded demographic attributes including age, sex, body condition and color morph. We assessed individual body condition using two different indices: weight to wing chord ratio (Hull et al. 2006) and ptilochronology (Grubb 1989). Weight to wing chord ratio was calculated as body mass divided by the length of the un-flattened longest primary (Pyle 1997; Hull et al. 2006) while the Grubb (2006) protocol was used in ptilochronology measurement of growth bar widths. For ptilochronology, the right outermost tail feather (retrix 6) was collected for analysis of feather growth bar width, a well-accepted metric of recent nutritional status (Grubb 1989; Jenkins et al. 2001; Grubb 2006). Each feather was individually placed in Ziploc bag to avoid consumption by feather lice, and recorded with the bird's ID, morph type, capture date,

age, and sex. Feathers were then refrigerated to discourage ectoparasite consumption. Color morphs were categorized for of each bird using morphotypes delineated by the Cornell Lab of Ornithology: Red, Checker, Pied, Spread, White, Blue-bar and Red bar (Johnston and Johnson 1989; Cornell 2009; Feinstein 2011). Finally, wing images were collected for photographic analysis using the protocols of Jacquin et al. (2011) to categorize birds as either dark or light, where birds expressing greater than 50% melanism upon the surface area of both wings fell into the dark (melanistic) category.

Blood and swab sample collection

Up to 1 mL of blood was taken from the brachial vein of each individual using a 25 to 31 gauge needle attached to a 1 mL syringe, and immediately transferred to heparinized capillary tubes for hematocrit (Hct) analysis (Morton 1994). Capillary tubes were placed in 15 mL conical tubes upright in a blood vacutainer tube rack until transfer to the lab, where tubes were spun for 10 minutes with a Zipocrit micro-hematocrit centrifuge (Lab Essentials®) (Gayathri et al. 2004; Gayathri and Hegde 2006).

Oral and cloacal swabs were collected from each bird by swabbing the mouth four times or the cloaca once for consistency in collecting comparable quantities of viral particles using sterile polyester tipped swabs (Puritan ®). Swabs were then transferred into 2.0 mL cryovials (Corning ®) containing 1.5 mL of Brain-Heart Infusion (BD ®) mixed with the antibiotics Gentamicin and Penicillin, and the anti-fungal Amphotericin-B to inhibit bacterial and fungal growth while simultaneously preserving viral particles. Cryovials were stored in a cooler with ice packs while in the field and then transferred to -80°C immediately upon arrival at the laboratory.

Parasitological analyses

Fecal samples were collected from all birds during field processing to quantify

gastrointestinal helminth infections. Samples were stored individually in tubes, kept on ice in the field, refrigerated at approximately 1.6°C, and processed within three days. The number and type of gastrointestinal helminth eggs shed by pigeons were quantified by diluting 0.05 grams of feces in 1.45 mLs of saturated salt solution and calculated using a modified McMaster helminth egg-counting technique (Gordon and Whitlock 1939; Seivwright et al. 2004). Parasite eggs were identified to genus level where possible following Sloss et al. (1994). To evaluate hemoparasite prevalence and intensity, blood smears were made in the field using the protocol of Bennett (1970) with the exception of methanol fixation; smears were then air-dried and transported in a slide box under ambient conditions. Within seven days of collection, all smears were stained with a Wright-Giemsa stain (Camco-Quik Stain II ®)**.** To determine the mean number of erythrocytes per field of view (FOV), ten blood smears were randomly selected from throughout the five-month long field season resulting in a mean erythrocyte count of 255 red blood cells per FOV. Each blood smear was subsequently analyzed for hemoparasites using 100 FOV under a magnification of 100 and 400, for approximately 25,500 red blood cells analyzed per smear (Bromwich and Schall 1986; Martinsen et al. 2008). Using a cell counter, hemoparasites were delineated into genus, while blood parasites of uncertain taxonomy were tabulated as unknowns. Identification of blood parasites to genus followed an illustrated identification guide presented in Campbell (1995).

APMV-1 was isolated at the Southeast Poultry Research Laboratory in Athens, GA (SEPRL) via inoculation of embryonating white leghorn chicken specific pathogen free eggs from cloacal and oral swabs, which then underwent two serial passages of allantoic fluid. After viral propagation in eggs, harvested allantoic fluid was tested using

hemagglutination tests (HA) to identify samples positive for live NDV. Positive HA samples underwent RNA extraction and PCR using a Superscript III RT-PCR Kit (Invitrogen ®), and subsequently sequenced using Fusion (F) gene specific primers for genotype identification (Miller et al. 2009; Susta et al. 2011; Diel et al. 2012b; Courtney et al. 2013).

Ptilochronology analysis in the lab utilized a novel blacklight technique for grayscale retrices. Retrices were individually attached to blackboard squares by the rachis using entomology pins, with the top 2/3 of each feather delineated for measurement as suggested by Grubb (1989). Growth bars in the top 2/3 of each feather were measured using 0.01 mm electronic calipers. Within a darkroom, light-colored growth bars (from growth during nocturnal periods) were visually enhanced for measurement capabilities in the UV-A blacklight spectrum (315-400 nanometers) in contrast to dark-colored growth bars (indicative of daytime growth). Since one daytime growth bar and one night-time growth bar is equivalent to a day's worth of growth, each nocturnal and diurnal growth bar was added together to represent a single 24-hour growth bar. Finally, the mean growth bar width for each rock pigeon was calculated for statistical analysis.

Statistical analyses

All statistical analyses were performed using SAS v. 9.3. Only helminths and blood parasites with a prevalence greater than 20% were analyzed. Although AMPV-1 shedding rates exhibited a prevalence below 20% (actual prevalence 15.28%), it was analyzed to examine potential effects on body condition. Non-parametric Wilcoxon Rank Sums tests were used in color morph analysis due to the non-Gaussian distribution of parasite intensity. Non-parametric Loess regression analyses for blood parasites were

instituted to account for non-linearity in response to body condition metrics. For all other analyses, a generalized linear model was utilized.

RESULTS

Color morph analysis using Wilcoxon Rank Sum-Tests (two-tailed) revealed statistically significant results for egg shedding of *Ascaridia* sp. ($H = 9.1161$, $df = 1$, $p = 0.0025$) (Figure 3.1) and pooled nematodes ($H = 8.9253$, $df = 1$, $p = 0.0028$) (Figure 3.2). Melanistic birds shed a mean rank of 34.36 of ascarid eggs, while light-colored birds shed a mean rank of 51.06 eggs. Among pooled nematodes, melanistic birds shed a mean rank of 34.39 eggs, while light-colored birds shed a mean rank of 50.98 eggs. Several helminth parasites showed no effect of melanism on egg shedding. *Dispharynx* sp. analysis revealed no statistically significant result ($H = 0.0006$, $df = 1$, $p = 0.9810$), with mean egg shedding ranks similar; melanistic birds scored 39.46 and light colored birds scored 39.58. *Capillaria* sp. did not reveal a statistically significant result ($H = 0.5138$, $df = 1$, $p = 0.4978$), nor did *Trichostrongylus* sp. ($H = 0.8168$, $df = 1$, $p = 0.3661$), although for all non-significant results, light colored birds shed higher mean ranks of eggs as opposed to their dark morph counterparts.

For counts of blood parasites, *Haemoproteus* sp. was not statistically significant between color morphs ($H = 0.0725$, $df = 1$, $p = 0.7878$), nor was *Plasmodium* sp. ($H = 0.7310$, $df = 1$, $p = 0.3926$). Melanistic birds were also not statistically in better body condition according to weight-to-wing chord as opposed to their light colored counterparts ($df = 76$, $F = 0.9111$, $p = 0.3429$). Analysis of covariance between color morph and weight-to-wing chord was statistically significant for *Haemoproteus* sp. ($df = 66$, $F = 3.86$, $p = 0.0134$) (Figure 3.4), suggesting that light morph pigeons demonstrated a significant decrease in body condition (weight to wing chord ratio) in contrast to their

dark morph counterparts**.** No significant effect was apparent for the same interaction for Plasmodium sp. (df = 66, F = 0.48, p = 0.6993).

Ptilochronology analysis alone was not predictive for mean numbers of any of the above hemoparasites parasites tested (*Plasmodium* sp. df = 53, F = 2.1267, p = 0.1491; *Haemoproteus* sp. df = 53, F = 0.0504, p = 0.8233). Nor did any statistically significant results occur due to *Ascaridia* sp. infection (df = 53, F = 0.8782, p = 0.6126), pooled nematodes (df = 54, F =0.0509), *Dispharynx* sp. (df = 53, F = 0.6702, p = 0.7310), *Capillaria* sp. (df = 54, F = 0.5633, p = 0.4562), or *Trichostrongylus* sp. (df = 54, F = 0.0001, p = 0.9985). Surprisingly, there was also no correlation between growth bar width and weight-to-wing chord (df = 53, F = 0.0024, p = 0.9610).

Weight-to-wing chord was highly correlated to *Haemoproteus* sp. infection (df = 66, F = 10.1474, p = 0.0022) (Figure 3.5), although the same response was not evident for *Plasmodium* sp. (df = 66, F = 1.1504, p = 0.2874). Multiple regression revealed that weight-to-wing chord and mean growth bar width were significant predictors for *Haemoproteus* sp. (df = 52, F = 2.8054, p = 0.0493) (Figure 3.6), *Plasmodium* sp. (df = 52, F = 4.20, p = 0.0206) (Figure 3.7), and APMV-1 (df = 52, F = 3.35, p = 0.0432) (Figure 3.8). No significant effects were determined for weight-to-wing chord or interaction effects with mean growth bar widths for any of the helminth species tested with ≥ 20% prevalence.

Due to the low sample size of hematocrits, only descriptive statistics were performed. Mean Hct values for 22 tested pigeons equaled 53.24 ± 4.92 (CI 51.07 – 55. 43), while the mean growth bar width of 55 analyzed retrices during ptilochronology analysis equaled 5.3 mm ± 1.42 (CI 4.91 – 5.68).

CONCLUSION

Fifty-four melanistic Rock Pigeons and twenty-four light colored Rock Pigeons were recovered from the metro Atlanta area. All color morphs were represented with the exception of the White morph; Checkers were the most prominent (43%), followed by Blue-bars (27%), Spreads (14.1%), Reds (1.28%), Red-bars (1.28%) and Pieds (1.28%). The remaining birds were hybrids of the assorted polymorphisms (Figure 3.3). Literature comparisons of the plumage variation among free-ranging pigeons populations showed that melanistic birds are increasingly found in densely populated cities as opposed to their rural counterparts (Obukhova 2001; Čanády and Mošanský 2013). The degree of eumelanin in pigeons (the pigment responsible for black and brown phenotypes) (Haase et al. 1992; Ito and Wakamatsu 2003) does not vary with age, and thus was not analyzed as a covariate (Johnston and Janiga 1995). Jacquin et al. (2013) reported that mean blood parasite prevalence among lighter and melanistic birds among urbanized Parisian birds were statistically similar, although less-urbanized paler birds exhibited fewer numbers of blood parasites. The urbanized blood parasite data from Jacquin et al. (2013) concur with the results of this study for blood parasites analyzed without covariates (e.g. intensities of blood parasites were not predicted by color morph alone), although a wider diversity of parasites were analyzed for metro Atlanta birds.

Haag-Wackernagel et al. (2006) followed the recruitment probabilities among phenotypes of wild pigeons in Vienna, Austria, and found that while Blue-bars (light morph) birds had an increased birth rate, they also had reduced recruitment rates in contrast to melanistic counterparts. Potential reasons for these observations included the probability that melanistic birds breed year-round in Europe in densely populated areas, while the wild-type Blue-bar is more constricted in seasonal breeding efforts, preferring rural areas with more predators. Parasitism was not broached in the discussion.

Although in cities, it appears that melanistic pigeons have a selective advantage in terms of disease; wild type Blue-bars may offset that selective advantage as a result of decreased predation. As raptors establish within urbanized areas, depredation on pigeons increases (Boal and Mannan 1999). Peregrine falcons, a recent urban colonizer and the swiftest of all avian predators equally attacked all pigeon plumage variants, with the exception of the wild type Blue-bar. Experimental manipulation of the plumage on released Rock Pigeons demonstrated that the white rump patch found only on the wild-type blue bar conferred a selective advantage during aerial pursuit (Palleroni et al. 2005). Thus, while melanistic birds may convey an immune advantage, paler birds may deflect that advantage via reduced depredation.

Interaction effects between mean growth bar width and weight-to-wing chord as a measure of body condition for blood parasites indicate that a larger sample size of retrices may have also served as a predictor for blood parasite intensity. The significant result between color morphs (dark versus light), *Ascaridia* sp. and pooled nematodes may have also been a function of sample size, as ascarids and nematodes exhibited the greatest prevalence among helminths in this study. As demonstrated by the stabilization of population cycles via an anti-helminthic treatment for the cecal nematode *Trichostrongylus tenuis* in Red Grouse, nematodes incur both morbidity and mortality in free-ranging birds (Hudson et al. 1998). Furthermore, although the roles of the MC1R gene and parasite pleotropic interactions have not been investigated in pigeons, it has been extensively studied in the Eleonora's Falcon (*Falco eleonorae*), Tawny Owls, and Bananaquits (*Coereba flaveola*) (Mundy 2005; Gangoso et al. 2011; Karell et al. 2011); although MacColl et al. (2013) found a contrasting result with no correlation between the amount of eumelanin and parasitism in Bananaquits.

Hematocrit values among metro Atlanta pigeons were increased in contrast to wild Rock Pigeons maintained in aviary conditions ($\overline{x}$ of 53.25 $\geq$ $\overline{x}$ of 47.2) (Gayathri et al. 2004). A feasible explanation for this difference may be due to handling time in the field, the seasonal bias of the sampling season (summer and fall), as well as the likelihood of limited water resources due to the 2012 southeastern drought. Due to limited data on the hematocrit values of pigeons, it is unknown whether the value of 53.25 is clinically abnormal, although Gayathri and Hegde (2006) reported a Hct variance of 42 to 54 among wild pigeons maintained in an outdoor aviary. The mean growth bar retrix widths measured in the sampling pool of Atlanta pigeons averaged 5.3 mm, well below the 7.6 mm measured by (Wood 1950). Although habitat or sample size were not indicated, Wood (1950) found that Rock Pigeon growth bars measured up to 7.6 mm, attributed to their constant foraging habits. Since Wood is the only other author reporting growth bar widths in Rock Pigeons, it is unknown as to why the growth bars among Atlanta birds were smaller. One possibility may be attributed to the poor diet quality of Atlanta pigeons, which were frequently observed consuming bread, chicken, donuts, and potato chips as opposed to their optimal granivorous diet. In a diet manipulation study of captive male White-throated Sparrows (*Zonotrichia albicollis*), Jenkins et al. (2001) established a strong relationship between feather regrowth rate and diet quality.

Future research into the isolation of the MC1R in wild pigeons may confer further insight into the extensive variety of plumage polymorphisms and selective pressures maintaining color morphs in free-ranging pigeons. In addition, the use of ptilochronology in disease studies may provide another measurable factor, (e.g. nutrition), to understanding the effects of parasitism on body condition.

FIGURES

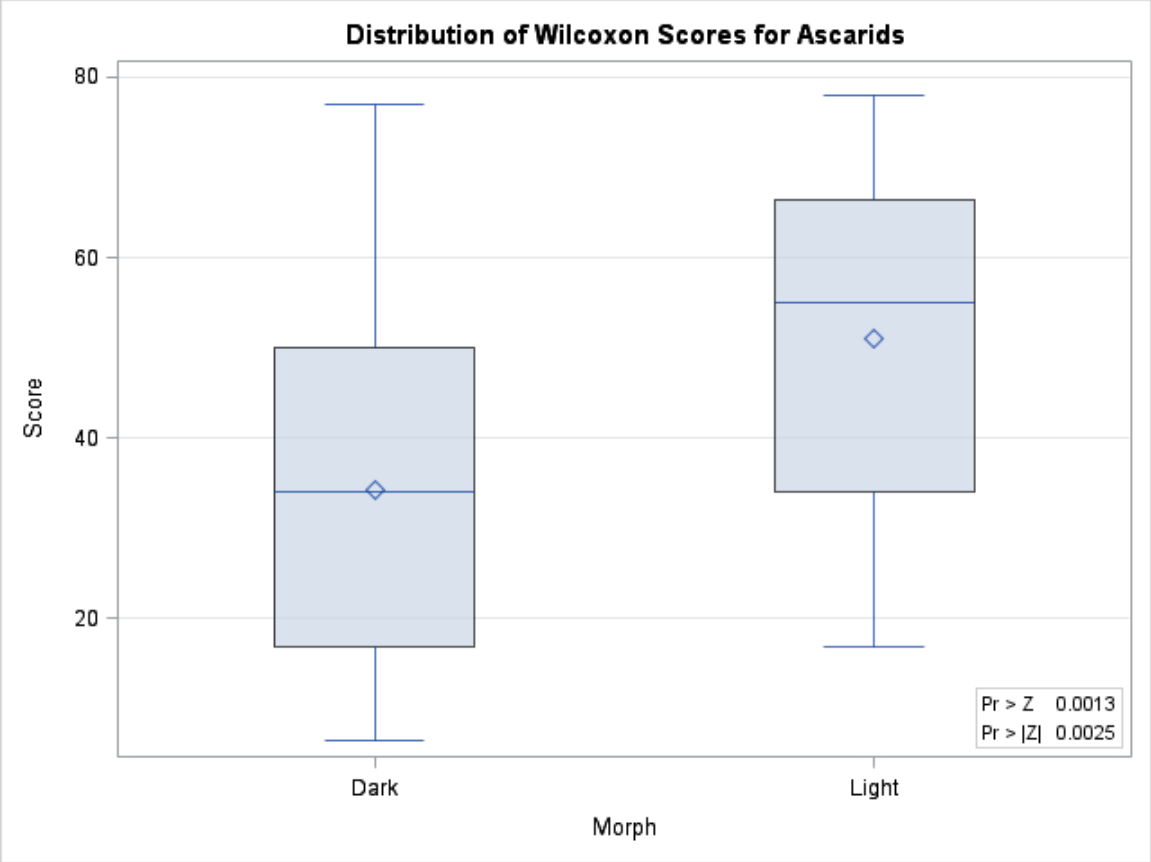


Figure 3.1. Wilcoxon Rank Sum-Test for *Ascaridia* sp. shedding and color morphs (dark versus light). Dark morph pigeons shed statistically fewer Ascarid eggs than their light colored counterparts (H = 9.1161, df = 1, p-value on the figure on the lower right hand side).

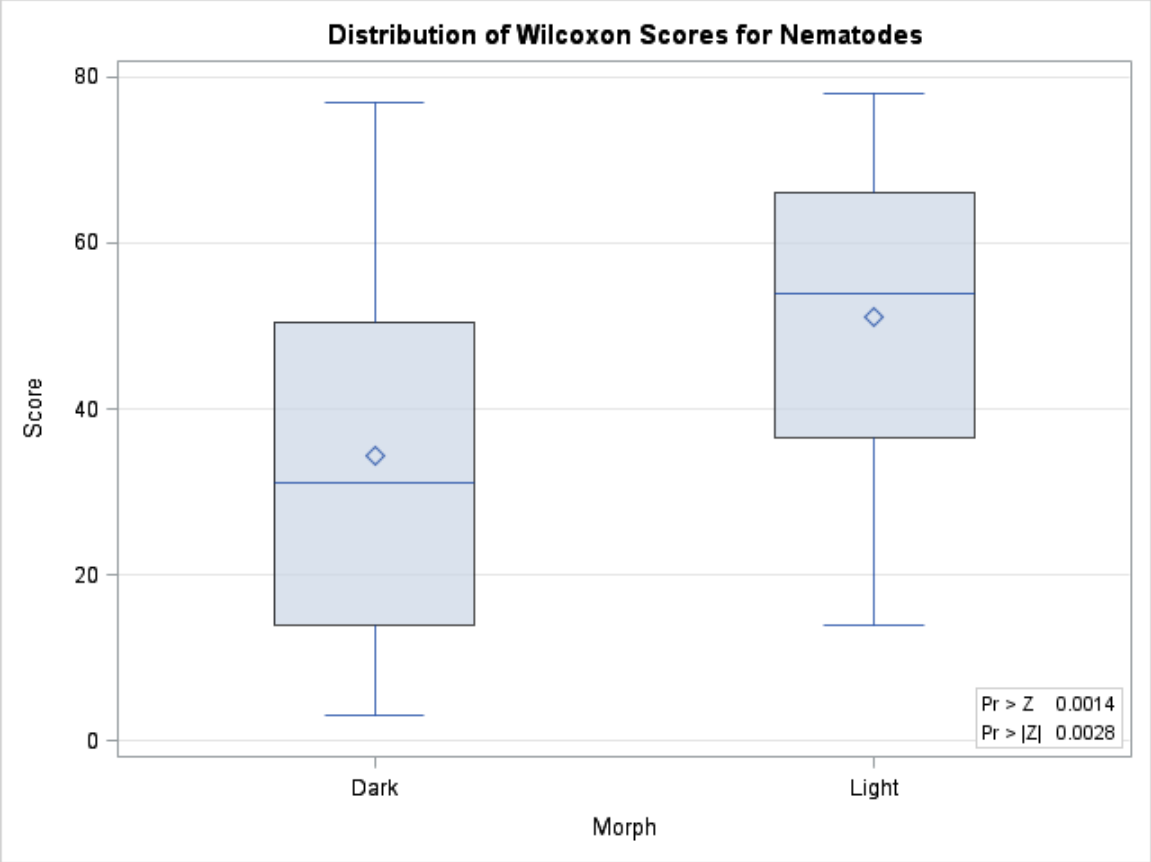


Figure 3.2. Wilcoxon Rank Sum-Test for nematode shedding and color morphs (dark versus light). While separate analyses for *Dispharynx* sp., *Capillaria* sp., and *Trichostrongylus* sp. did not yield statistically significant results individually, pooled nematode analysis which included *Ascaridia* sp. yielded a significantly reduced number of eggs shed by melanistic (dark) pigeons than their paler counterparts (H = 8.9253, df = 1, p-value on lower right hand corner of figure).

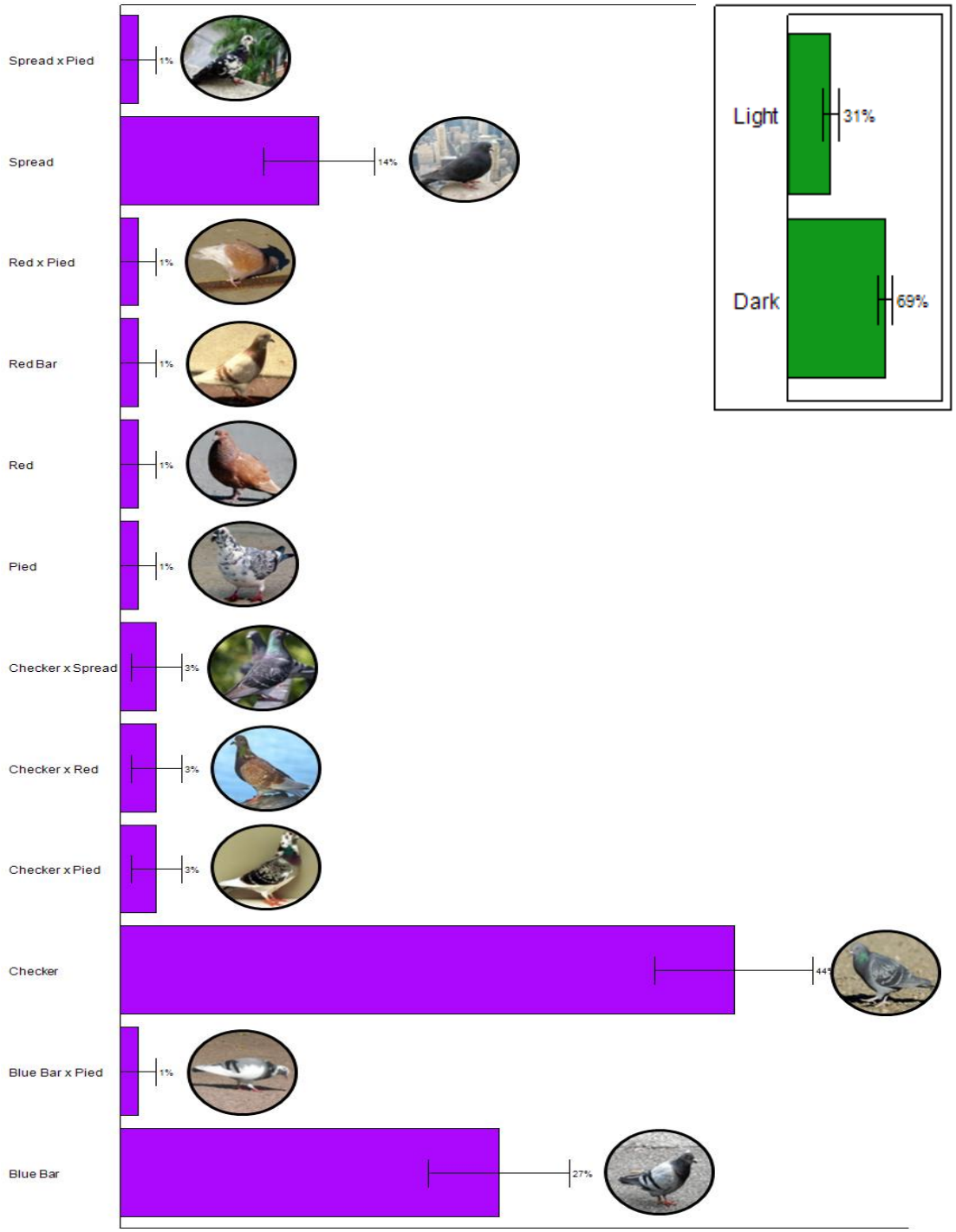


Figure 3.3. Color morphs of wild Rock Pigeons captured in Atlanta, Georgia. All plumage polymorphisms as identified by the Cornell Laboratory of Ornithology were represented during sampling except for the White morph. Six hybrids were also identified. Checkers (Dark) were captured at the highest frequency at 44%, followed by the wild type variant (Light) Blue-bar (27%), (Dark) Spreads (14%), Checker-Hybrids (2%), where melanism varied according to the degree of wing pigmentation, one (Light) Checker-Pied (1%), one (Dark) Spread-Pied (1%), followed by (all Light) Red-Pied, Red-bar, Red, and Pied (1%).

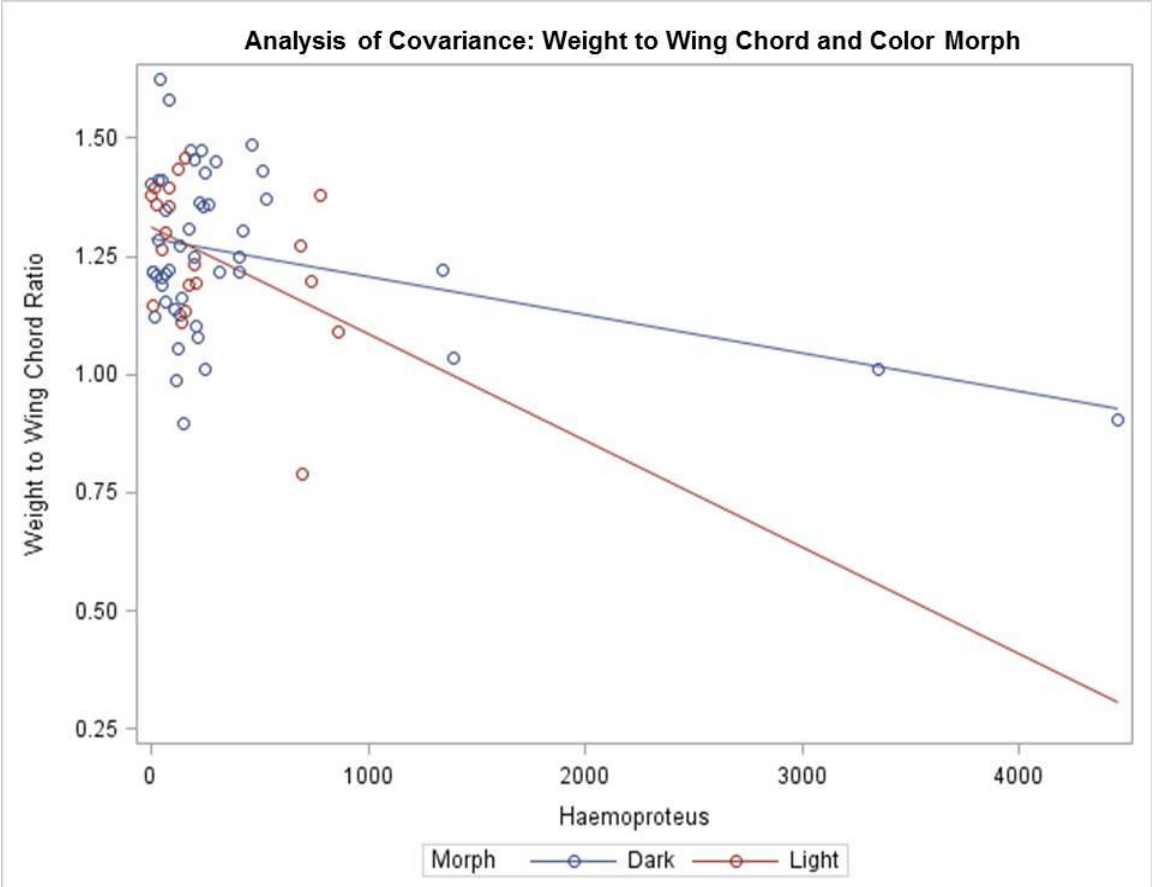


Figure 3.4. ANCOVA for *Haemoproteus* sp., color morph, and weight to wing chord. Light morph pigeons demonstrated a significant decrease in body condition (weight to wing chord ratio) in contrast to their dark morph counterparts as the intensity of *Haemoproteus* sp. increased (df = 66, F = 3.86, p =0.0134).

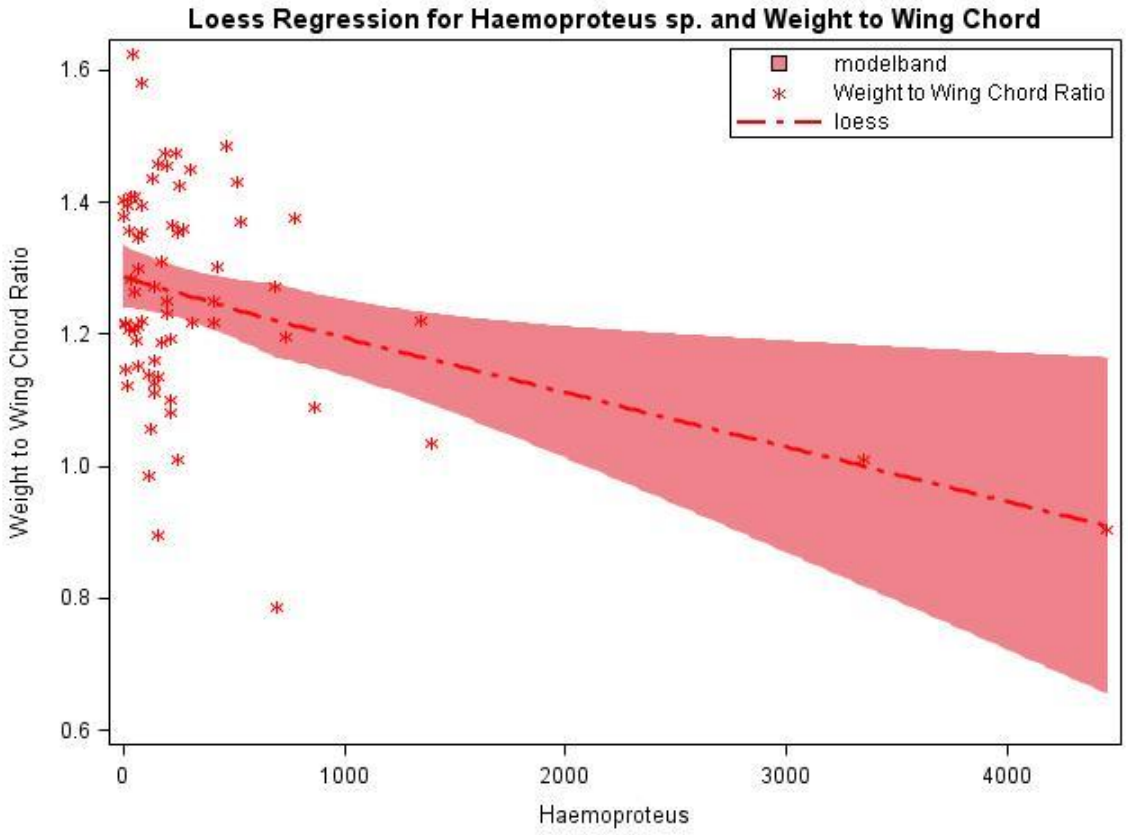


Figure 3.5. Regression for *Haemoproteus* sp. and weight to wing chord. Body condition significantly declines with intensity of *Haemoproteus* sp. infection, although as the variance increases, the weighted sum of the square errors for body condition also increases due to non-linearity (df = 66, F = 10.1474, p = 0.0022).

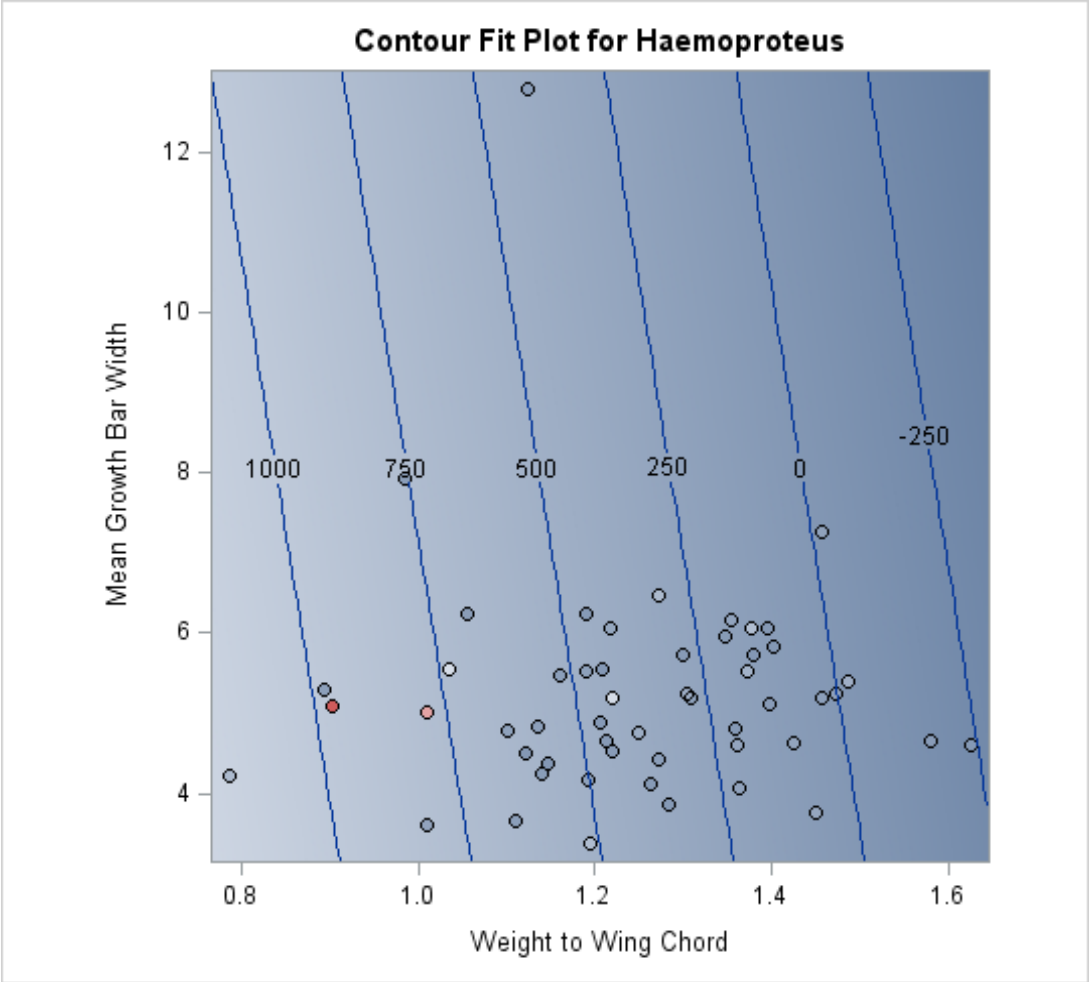


Figure 3.6. Multiple regression contour plot for *Haemoproteus* sp. with mean growth bar width and weight to wing chord as predictor variables. *Haemoproteus* sp. infection has the highest aggregation of infected erythrocytes in Rock Pigeons with a weight to wing chord ratio ≤ 1.1 (poor to very poor body condition) and a mean growth bar width just above 5.5 mm.

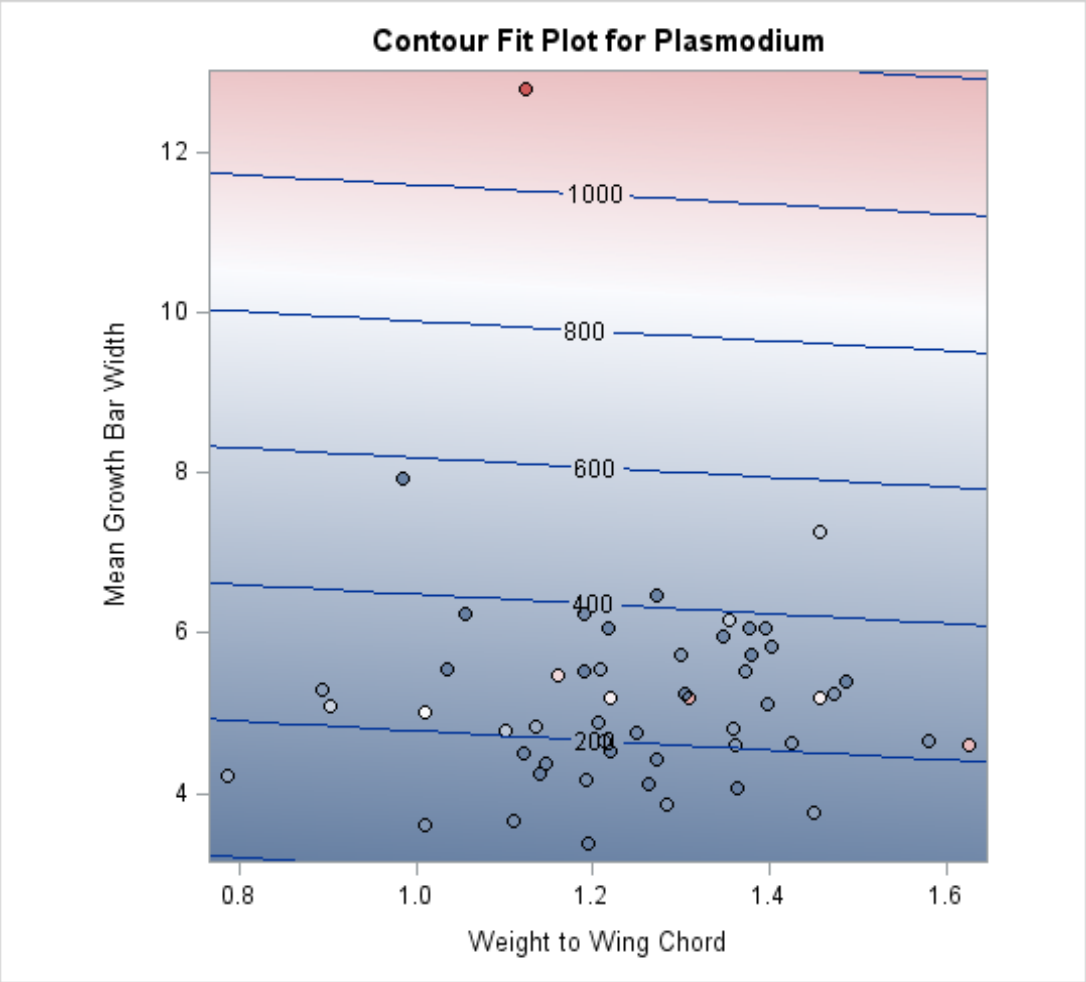


Figure 3.7. Multiple regression analysis for *Plasmodium* sp. with mean growth bar width and weight to wing chord as predictor variables. Mean intensities of Plasmodium sp. aggregated at just above 1.2 for weight to wing chord ratios, where ≤ 1.26 falls into the poor body condition category. Growth bar widths correlated between 5 and 6.5 mm for *Plasmodium* sp. intensity.

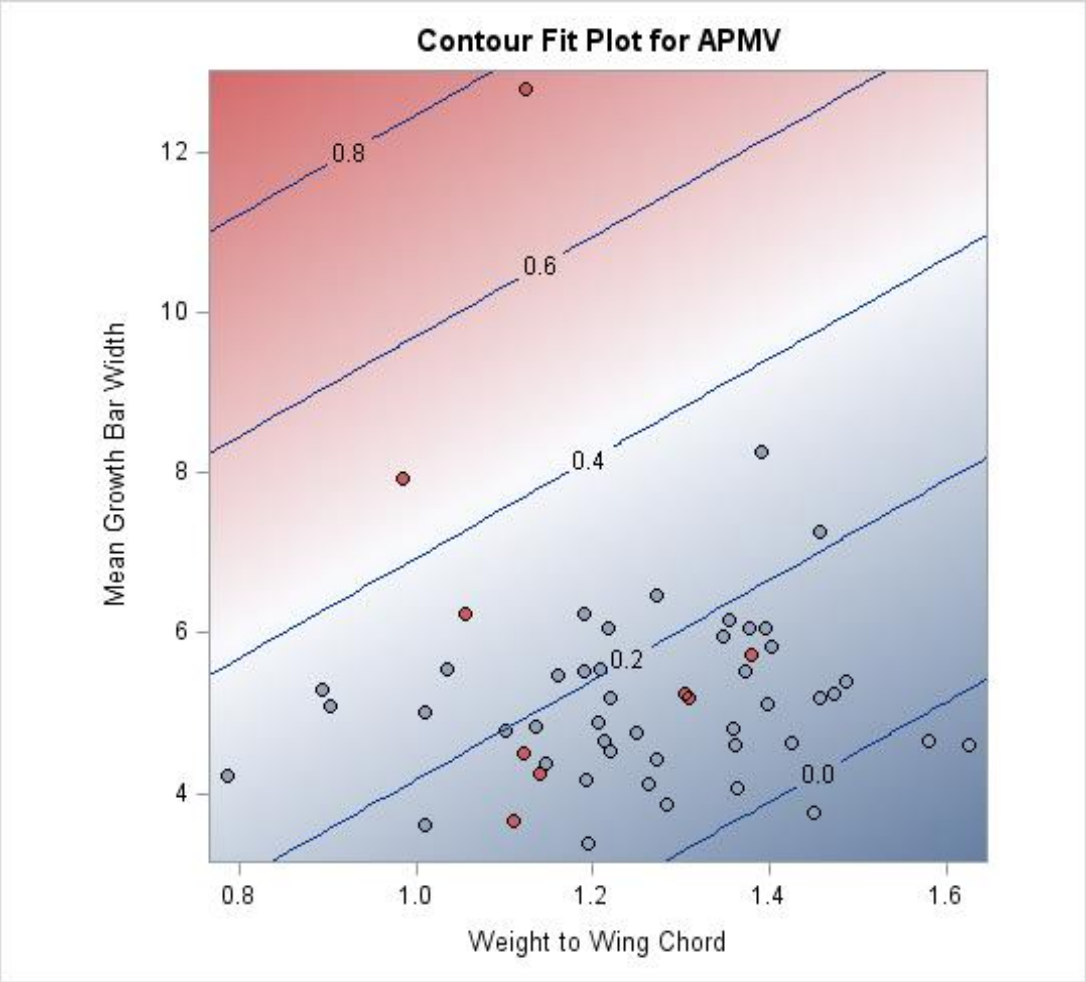


Figure 3.8. Multiple regression analysis for APMV-1 with mean growth bar width and weight to wing chord as predictor variables. Mean intensities of APMV-1 aggregated between 1.2 and 1.4 for weight to wing chord ratios, where ≤ 1.26 falls into the poor body condition category. Growth bar widths correlated between 5.0 and 6.0 mm for APMV-1 intensity.

CHAPTER 4

CONCLUDING THOUGHTS AND FUTURE DIRECTIONS

The overarching purpose of this thesis was to disentangle certain complex mechanisms driving host-parasite relationships in the ecological framework of a globalized avian model system. As the implications of concomitant infections in wild hosts are increasingly recognized (Cox 2001; Tompkins et al. 2011), addressing multi-faced variables such as host demographics and the role of exotic species in are necessary in unveiling potential confounding patterns of disease transmission. Wild Rock Pigeons serve as an excellent model in which to test the role of co-infection among avian species due to their susceptibility to numerous pathogens (Tudor 1991). Yet, as an exotic species that ranges throughout a large portion of the world, this presents a particularly tricky challenge in determining whether results uncovered in one region are reproducible in another. This issue presents a new variable for understanding disease transmission within the interface between veterinary medicine, ecology, and public health; since pathogen infectiousness and host susceptibility may be driven by synergistic effects wholly unrelated among different populations. The enemy release hypothesis is particularly suited to this question; if the presence of one host parasite exerts intrinsic effects upon the transmissibility of another parasite, what are the implications for transmission if the host-parasite community is altered? Demographic effects also serve as ecological variants among populations. For instance, the role of melanism as a potential regulator of immune function is strongly supported in birds (Mundy 2005; Ducrest et al. 2008; Gangoso et al. 2011), yet geographically isolated populations exposed to diversified

systems of parasite fauna may express phenotypic differences as a result. Consequentially, correlative studies such as this one may draw potential parallels for patterns and processes of pathogens among avian hosts, but conclusivity remains elusive.

Specifically, my master's research centered upon three primary themes (1) the effects of helminth co-infection and virus susceptibility (2) the role of parasite release in an introduced avian species in Georgia and (3) the role of avian host heterogeneity parameterized by demographic variables such as body condition, melanism, and nutrition in parasite presence or absence. For each general question, I attempted to draw correlative relationships from a limited sample of a complex avian host system, wild pigeons in metro Atlanta, Georgia, to broad ecological disease phenomena.

The most significant results of chapter two were multi-faceted. First, I identified two novel helminths in pigeons (*Allodapa* sp. and *Porrocaecum* sp.) which had not been previously identified in the literature. Secondly, I also identified the presence of *Plasmodium* sp. which has never been documented among wild Rock Pigeons in Georgia. Lastly, I also found that the parasite fauna of pigeons in Georgia, their non-native range, have undergone parasite inversion in contrast to the parasites found in their native region, which according to the review of the literature, have lost nineteen parasites from their native ranges, but have since gained nineteen in their new range over the last four hundred years. In chapter three, ptilochronology was utilized for the first time as a response variable to parasitism. It is an interesting dichotomy that chronic parasitism with helminths with body condition as a covariate was not statistically significant, although chronic blood parasitism and acute viral parasitism with body condition as a covariate was significant. Thus, it appears that intracellular parasites may induce greater costs to overall body condition in wild pigeons than extracellular parasites. Linear

regression lines for helminth parasitism indicated that while mean growth bar widths did decline in response to helminths, it is very likely as a result of feather sample size (n = 55), that this result was not statistically significant. Future work amalgamating the empirical representation of nutrition in birds as represented by ptilochronology should be further investigated, since molt is not only energetically costly (Murphy and Taruscio 1995), but also necessary for optimal flight performance (Williams and Swaddle 2003). For pigeons, parasitism which may inhibit flight performance may be of significance due to high rates of depredation.

Although Chapter 2 suggested that while parasite inversion has occurred in wild pigeons in the New World, similar results may not be reproducible in other model systems due to the unique synchronous history pigeons have with man (Blackburn et al. 2009). In addition, the global culling practices of pigeons as a pest species may facilitate the transmission of pathogens to re-colonizers of culled areas (Donnelly et al. 2003). Thus as culling practices continue; pathogen accumulation and potential host shifts in wild pigeons may be occurring more rapidly than in non-culled species. While Chapter 3 found results concurrent with pleiotropic effects of immunity and melanism, this study suffers from a lack of experimental manipulation in contrast to correlation, in addition to an unidentified mechanistic explanation for those effects.

Clearly, although this work laid the foundation for novel approaches to the understanding of disease dynamics in wild birds, more concrete work should be pursued. The identification of the transmission origin of avian paramyxovirus-1 among Atlanta pigeons is an extremely interesting research goal, considering the intense prevalence of the shedding rates among the sample size collected. Furthermore, empirically testing the use of the blacklight method for ptilochronology may provide an inexpensive yet

powerful tool for researchers utilizing growth bars for nutritional analysis in birds with grayscale retrices. A final goal is to further pursue analysis into the effects of co-infection in wild pigeons, as the question remains whether chronic parasitism induces susceptibility to acute parasitism, or does acute parasitism induce susceptibility to chronic parasitism in wild birds? Close collaboration with governmental entities such as the SEPRL has provided a framework for more established research studies and findings. A key future direction for this work will be to develop sounder, more empirically driven ecological investigations to elucidate pathogen transmission dynamics in wild birds.

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APPENDIX A

SUPPORTING INFORMATION FOR CHAPTER 2

METHODS

Literature review included in the study

A review of 78 sources provided 69 endoparasites to analyze the number of parasites lost and gained since the Rock Pigeon's time of invasion (early 1600's) into the New World.

Table A1. Endoparasite, range, distribution, and sources.

Endoparasite	Range	Distribution	Source
Acanthocephalan sp.	Not Shared	New	This study; Friend and Franson 1999
Allodapa sp.	Not Shared	New	This study
Amoebataenia cuneata	Not Shared	New	Adang 2007; Adang et al. 2008
APMV-1	Shared	Cosmopolitan	This study; Friend and Franson 1999; Toro et al. 1999; Ciglar Grozdanić 2000; Vučemilo et al. 2003; Allison et al. 2004; Toro et al. 2005
Aporina delafondi	Shared	Cosmopolitan	Coletta 1958; Jochen 1962; Toro et al. 1999; Dehlawi 2006; Smith 2008; Smith and Fedynich 2012
Ascaridia columbae	Shared	Cosmopolitan	Irwin-Smith 1920; Miller 1935; Jochen 1962; Walcott 1969; Da Silva et al. 1990; Boado et al. 1992; Toro et al. 1999; Gıcık and Burgu 2000; Mushi et al. 2000; Oliveira et al. 2000; Foronda et al. 2004; Senlik et al. 2005; Adang 2007; Adang et al. 2008; Gül et al. 2009

Table A1. Continued.

Endoparasite	Range	Distribution	Source
Ascaridia galli	Shared	Cosmopolitan	Adang 2007; Adang et al. 2008; Msoffe et al. 2010
Brachylaima mazzanti	Not Shared	New	Da Silva et al. 1990
Capillaria annulata	Not Shared	New	Toro et al. 1999
Capillaria columbae	Shared	Cosmopolitan	Miller 1935; Wehr 1939; Jochen 1962; Walcott 1969; Da Silva et al. 1990; Boado et al. 1992; Toro et al. 1999; Gıcık and Burgu 2000
Capillaria obsignata	Shared	Cosmopolitan	Gkithkopoulos and Liakos 1987; Boado et al. 1992; Toro et al. 1999; Senlik et al. 2005; Basit et al. 2006; Tanveer et al. 2011; Al-Barwari and Saeed 2012
Capillaria sp.	Shared	Cosmopolitan	This study; Irwin-Smith 1920; Miller 1935; Boado et al. 1992; Dovč et al. 2004; Tietz Marques et al. 2007; Sari et al. 2008; Gül et al. 2009; Eljadar et al. 2012
Chlamydophila psittaci	Shared	Cosmopolitan	Friend and Franson 1999; Mushi et al. 2001; Dovč et al. 2004
Choataenia sp.	Not Shared	New	This study; Zahrani et al. 2012
Coccidia	Shared	Cosmopolitan	This study; Boado et al. 1992; Friend and Franson 1999; Mushi et al. 2000; Sari et al. 2008; Opara et al. 2012
Cotugnia cuneata	Not Shared	New	Musa et al. 2011
Dispharynx nasuta	Shared	Cosmopolitan	Gıcık and Burgu 2000; Smith 2008; Smith and Fedynich 2012
Dispharynx spiralis	Shared	Cosmopolitan	Coletta 1958; Da Silva et al. 1990; Toro et al. 1999; Mushi et al. 2000; Foronda et al. 2004
Echinostoma columbae	Not Shared	New	Miller 1935
Echinostoma paraulum	Not Shared	New	Miller 1935

Table A1. Continued.

Endoparasite	Range	Distribution	Source
Echinostoma revolutum	Not Shared	New	Musa et al. 2011
Eimeria	Shared	Cosmopolitan	Jochen 1962; Walcott 1969; Gkithkopoulos and Liakos 1987; Dovč et al. 2004; Foronda et al. 2004; Bandyopadhyay et al. 2006; Tietz Marques et al. 2007; Gül et al. 2009; Radfar et al. 2011; Eljadar et al. 2012; Opara et al. 2012
Gongylonema ingluvicola	Not Shared	New	Toro et al. 1999
Hadjelia truncata	Shared	Cosmopolitan	Radfar et al. 2011; Naem et al. 2013
Haemoproteus columbae	Shared	Cosmopolitan	Kartman 1949; Jochen 1962; Knisley and Herman 1967; Walcott 1969; Bennett and Peirce 1990; Graczyk et al. 1994; Quek et al. 1999; Mushi et al. 2000; Oliveira et al. 2000; Sol et al. 2000; Rodríguez and Matta 2001; Forrester and Spalding 2003; Foronda et al. 2004; Öz and Turut 2007
Haemoproteus sp.	Shared	Cosmopolitan	This study; Dranzoa et al. 1999; Deviche et al. 2001; Shurulinkov and Golemansky 2002; Adlard et al. 2004; Padilla et al. 2004; Valkiūnas et al. 2005; Tietz Marques et al. 2007; Dadi-Mamud et al. 2012; Opara et al. 2012
Heterakis sp.	Shared	Cosmopolitan	This study; Jochen 1962; Adang 2007; Adang et al. 2008; Gül et al. 2009; Eljadar et al. 2012
Hymenolepis cantaniana	Not Shared	New	Adang 2007; Adang et al. 2008

Table A1. Continued.

Endoparasite	Range	Distribution	Source
Hymenolepis carioca	Shared	Cosmopolitan	Adang 2007; Adang et al. 2008; Zahrani et al. 2012
Leucocytozoon sp.	Not Shared	New	This study; Earle and Little 1993; Dadi-Mamud et al. 2012
Microfilaria	Shared	Cosmopolitan	This study; Pande et al. 1962; Earle and Little 1993; Ngoented 2000
Ornithostrongylus quadriradiatus	Not Shared	New	Le Roux 1926; Vigueras 1929; Cuvillier 1937; Jochen 1962; Walcott 1969; Tongson et al. 1975; Da Silva et al. 1990
Plasmodium sp.	Shared	Cosmopolitan	This study; Dranzoa et al. 1999; Al-Barwari and Saeed 2012; Dadi-Mamud et al. 2012; Opara et al. 2012
Porrocaecum sp.	Not Shared	New	This study
Raillietina bonini	Shared	Cosmopolitan	Da Silva et al. 1990; Gıcık and Burgu 2000; Smith 2008; Al-Barwari and Saeed 2012; Smith and Fedynich 2012
Raillietina cesticillus	Shared	Cosmopolitan	Boado et al. 1992; Adang 2007; Diakou et al. 2013
Raillietina echinobothrida	Shared	Cosmopolitan	Boado et al. 1992; Gıcık and Burgu 2000; Senlik et al. 2005; Adang 2007; Adang et al. 2008; Msoffe et al. 2010; Musa et al. 2011; Zahrani et al. 2012; Diakou et al. 2013
Raillietina magninumida	Shared	Cosmopolitan	Adang 2007; Radfar et al. 2011
Raillietina tetragona	Shared	Cosmopolitan	Gkithkopoulos and Liakos 1987; Boado et al. 1992; Adang 2007; Adang et al. 2008; Msoffe et al. 2010; Al-Barwari and Saeed 2012; Zahrani et al. 2012; Diakou et al. 2013
Salmonella sp.	Shared	Cosmopolitan	Gregurić et al. 1991; Friend and Franson 1999; Toro et al. 1999; Vučemilo et al. 2003; Dovč et al. 2004

Table A1. Continued.

Endoparasite	Range	Distribution	Source
Sarcocystis calchasi	Shared	Cosmopolitan	Friend and Franson 1999; Wünschmann et al. 2011
St. Louis Encephalitis virus	Not Shared	New	Gruwell et al. 2000; Allison et al. 2004
Syngamus sp.	Shared	Cosmopolitan	This study; Campbell 1935; Boado et al. 1992; Friend and Franson 1999; Sari et al. 2008; Gül et al. 2009; Smith and Fedynich 2012; Mohammad et al. 2013
Tanaisia bragai	Not Shared	New	Maldonado and Hoffman 1941; Da Silva et al. 1990
Tetrameres americana	Not Shared	New	Smith 2008; Smith and Fedynich 2012
Tetrameres columbicola	Not Shared	New	Simpson et al. 1984
Toxoplasma gondii	Shared	Cosmopolitan	Mushi et al. 2001; Yan et al. 2011
Trichimonas gallinae	Shared	Cosmopolitan	Jochen 1962; Walcott 1969; Gkithkopoulos and Liakos 1987; Friend and Franson 1999; Toro et al. 1999; Vučemilo et al. 2003; Dovč et al. 2004; Robinson et al. 2010; Radfar et al. 2011; Al-Barwari and Saeed 2012; Opara et al. 2012
Trichostrongylus sp.	Shared	Cosmopolitan	This study; Irwin-Smith 1920; Vučemilo et al. 2003
West Nile virus	Shared	Cosmopolitan	Allison et al. 2004; Calistri et al. 2010
Aonchotheca sp.	Not Shared	Old	Foronda et al. 2004
Capillaria caudinflata	Not Shared	Old	Gkithkopoulos and Liakos 1987
Cotugnia columbae	Not Shared	Old	Al-Barwari and Saeed 2012
Cotugnia digonopora	Not Shared	Old	Radfar et al. 2011; Al-Barwari and Saeed 2012; Zahrani et al. 2012
Cotugnia majdoubii	Not Shared	Old	Magzoub et al. 1980

Table A1. Continued.

Endoparasite	Range	Distribution	Source
Cotugnia polyacantha	Not Shared	Old	Al-Barwari and Saeed 2012
Cotugnia satpuliensis	Not Shared	Old	Al-Barwari and Saeed 2012
Hymenolepis sphenocephala	Not Shared	Old	Gıcık and Burgu 2000
Killigrewia chandigarhensis	Not Shared	Old	Duggal and Gupta 1987
Raillietina fragilis	Not Shared	Old	Duggal and Gupta 1987
Raillietina carpohagi	Not Shared	Old	Al-Barwari and Saeed 2012
Raillietina fuhrmanni	Not Shared	Old	Al-Barwari and Saeed 2012
Raillietina georgensis	Not Shared	Old	Gıcık and Burgu 2000
Raillietina micracantha	Not Shared	Old	Foronda et al. 2004
Raillietina perplexa	Not Shared	Old	Dehlawi 2006
Raillietinia canabia	Not Shared	Old	Magzoub et al. 1980
Raillietinia zahratis	Not Shared	Old	Magzoub et al. 1980
Retinometra serrata	Not Shared	Old	Dehlawi 2006
Tetrameres fissispina	Not Shared	Old	Foronda et al. 2004

Table A2. Correlation Coefficient Matrix for Helminths from Atlanta Rock Pigeons captured in 2012. Coefficients highlighted in red are statistically significant.

	Ascarids	Dispharynx	Capillaria	Trichostrongylus	Railletina	Echinostoma	Hymenolepis	Heterakis	Choataenia	Tetrameres	Taenia	Allodapa	Porrocaecum	Acanthocephalan	Syngamus
Ascarids	1	0.31743	-0.0032	0.47518	-0.03141	0.58418	0.23439	0.47395	0.42239	0.25131	0.13757	-0.10091	-0.04243	-0.04627	-0.03258
		0.0046	0.9779	<.0001	0.7948	<.0001	0.0389	<.0001	0.0001	0.0265	0.2297	0.3794	0.7122	0.6875	0.7771
Dispharynx	0.31743	1	0.16524	0.78562	-0.00758	0.17751	-0.01558	0.07321	-0.04422	0.65021	0.36547	-0.10935	0.02977	-0.07296	-0.05137
	0.0046		0.1483	<.0001	0.9475	0.12	0.8923	0.5241	0.7007	<.0001	0.001	0.3406	0.7956	0.5256	0.6551
Capillaria	-0.0032	0.16524	1	0.31427	0.08829	-0.06198	-0.07432	-0.08029	-0.04387	-0.07557	0.59118	-0.03804	-0.07075	0.04287	0.0926
	0.9779	0.1483		0.0051	0.4421	0.5898	0.5178	0.4847	0.7029	0.5108	<.0001	0.7409	0.5382	0.7094	0.42
Trichostrongylus	0.47518	0.78562	0.31427	1	0.15085	0.36091	-0.01986	0.03722	0.00748	0.59431	0.57597	-0.0681	0.07575	-0.05095	-0.03587
	<.0001	<.0001	0.0051		0.1874	0.0012	0.8629	0.7463	0.9482	<.0001	<.0001	0.5536	0.5098	0.6578	0.7552
Railletina	-0.03141	-0.00758	0.08829	0.15085	1	-0.05446	-0.02393	-0.02852	0.01337	-0.08388	0.25458	-0.04655	-0.05868	-0.04848	-0.03414
	0.7848	0.9475	0.4421	0.1874		0.6358	0.8352	0.8043	0.9075	0.4653	0.0245	0.6857	0.6099	0.6734	0.7667
Echinostoma	0.58418	0.17751	-0.06198	0.36091	-0.05446	1	-0.03741	0.24853	-0.04514	0.14384	0.02006	-0.06625	-0.03561	-0.02943	-0.02072
	<.0001	0.12	0.5898	0.0012	0.6358		0.745	0.0282	0.6948	0.209	0.8616	0.5644	0.7569	0.7982	0.8571
Hymenolepis	0.23439	-0.01558	-0.07432	-0.01986	-0.02393	-0.03741	1	-0.04339	0.29808	0.08222	-0.04223	-0.02625	0.04952	-0.03331	-0.02345
	0.0389	0.8923	0.5178	0.8629	0.8352	0.745		0.706	0.008	0.4742	0.7135	0.8196	0.6668	0.7722	0.8385
Heterakis	0.47395	0.07321	-0.08029	0.03722	-0.02852	0.24853	-0.04339	1	0.10398	-0.00772	0.00862	-0.07722	-0.04457	-0.03683	-0.02593
	<.0001	0.5241	0.4847	0.7463	0.8043	0.0282	0.7061		0.365	0.9465	0.9403	0.5016	0.6984	0.7489	0.8217
Choataenia	0.42239	-0.04422	-0.04387	0.00748	0.01337	-0.04514	0.29808	0.10398	1	-0.06092	-0.04304	0.13005	-0.04863	-0.04018	-0.02829
	0.0001	0.7007	0.7029	0.9482	0.9075	0.6948	0.008	0.365		0.5962	0.7083	0.2564	0.6724	0.7269	0.8058
Tetrameres	0.25131	0.65021	-0.07557	0.59431	-0.08388	0.14384	0.08222	-0.00772	-0.06092	1	-0.04345	-0.04677	-0.02769	0.07514	0.07872
	0.0265	<.0001	0.5108	<.0001	0.4653	0.209	0.4742	0.9465	0.5962		0.7056	0.6843	0.8098	0.5132	0.4933
Taenia	0.13757	0.36547	0.59118	0.57597	0.25458	0.02006	-0.04223	0.00862	-0.04304	-0.04345	1	-0.05386	-0.03637	-0.03464	-0.02439
	0.2297	0.001	<.0001	<.0001	0.0245	0.9616	0.7135	0.9403	0.7063	0.7056		0.6396	0.7519	0.7633	0.8321
Allodapa	-0.10091	-0.10935	-0.03804	-0.0681	-0.04655	-0.06625	-0.02625	-0.07722	0.13005	-0.04677	-0.05386	1	-0.07138	-0.05898	-0.04153
	0.3794	0.3406	0.7409	0.5536	0.6857	0.5644	0.8196	0.5016	0.2564	0.6843	0.6396		0.5346	0.608	0.7181
Porrocaecum	-0.04243	0.02977	-0.07075	0.07575	-0.05868	-0.03561	0.04952	-0.04457	-0.04863	-0.02769	-0.03637	-0.07138	1	0.29802	-0.02232
	0.7122	0.7958	0.5382	0.5098	0.6099	0.7569	0.6668	0.6984	0.6724	0.8098	0.7519	0.5346		0.008	0.8462
Acanthocephalan	-0.04627	-0.07296	0.04287	-0.05095	-0.04848	-0.02943	-0.03331	-0.03683	-0.04018	0.07514	-0.03464	-0.05898	0.29802	1	0.74886
	0.6875	0.5256	0.7094	0.6578	0.6734	0.7982	0.7722	0.7489	0.7269	0.5132	0.7633	0.608	0.008		<.0001
Syngamus	-0.03258	-0.05137	0.0926	-0.03587	-0.03414	-0.02072	-0.02345	-0.02593	-0.02829	0.07872	-0.02439	-0.04153	-0.02232	0.74886	1
	0.7771	0.6551	0.42	0.7552	0.7667	0.8571	0.8385	0.8217	0.8058	0.4933	0.8321	0.7181	0.9462	<.0001	

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