Effects of Haemoparasite Infection and Food Supplementation in a High Elevation Passerine Bird.

by

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Abstract

Strong circumstantial evidence from laboratory and theoretical studies suggests that sublethal parasite burdens can influence host population dynamics whether acting alone or in conjunction with reduced food availability. The significance of these effects has rarely been examined in natural environments. This study aims to examine the interactive effects of avian malarial haemoparasite infections and food supplementation in a food limited free-living population of Mountain White-crowned Sparrows (*Zonotrichia leucophrys oriantha*) breeding in Gunnison National Forest, Gothic, CO.

To analyze the effects of haemoparasites (*Plasmodium, Leucocytozoon, and Haemoproteus*) and food supplementation on multiple aspects of reproductive success, we analyzed blood smear data (to determine infection status) and manipulated sparrow food availability. During the early breeding season, when natural food availability is especially scarce due to residual snowpack and frequent spring snowstorms, supplemental food was provided on select plots. We quantified the impact of haemoparasite infection and food supplementation on reproductive success by evaluating: daily nest survival (DNS), clutch size, probability of the clutch hatching, probability of the nestlings fledging, and offspring quality.

Our data underscore the impacts of avian malaria on the reproductive success of a free-living avian population where malaria is endemic. Most of the negative effects of malaria infections are observed in young-rearing phase. In addition, we documented a supported strong positive effect of food supplementation, which however was restricted to the incubation phase. There was no effect of infection or food supplementation on clutch size, or on the quality of the fledged (quantified as young mass or tarsus length). Interestingly, we did not detect any interactions between food supplementation and blood parasite coinfection in any metrics of reproductive success, daily nest survival, or offspring quality.

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Introduction

Over the past several decades there has been a surge of interest into the effects of parasitism and disease on natural populations. May and Anderson catalyzed this research movement in the late 1970s with their seminal work on the impact of parasitic organisms on host abundances (May & Anderson 1978; Anderson & May 1978). This was closely followed with Price's (1980) argument regarding the important role that parasites play in natural ecosystems. This research has refined an earlier view that parasites, while ubiquitous, pose only benign threats to their hosts (Price 1980). Indeed, subsequent research has revealed the obscured but pervasive effect of parasites on the host's population size, population cycles, community structure, sexual selection, and secondary sexual traits (Anderson & May 1979; Hamilton & Zuk 1982; Minchella & Scott 1991; Hudson, Newborn & Dobson 1992; Hudson, Dobson & Newborn 1998; Grenfell & Dobson 1995; Albon *et al.* 2002; Vergara *et al.* 2012).

In addition to parasites, food availability is thought to be a major factor regulating host populations (Begon, Harper & Townsend 2006). Both experimental and observational studies have shown that insufficient food availability in vertebrates can lead to growth retardation, attenuated immunocompetence, diminished reproductive performance, and reduced survival (Ankney & Macinnes 1978; Martin 1987; Lochmiller, Vestey & Boren 1993; Lochmiller & Deerenberg 2000; Allen, Gilchrist & Smith 2007; Cox & Cresswell 2014). In addition, it has been shown repeatedly in laboratory settings that malnourished vertebrates are more susceptible to parasitic infection (Ing *et al.* 2000; Koski & Scott 2001). Field studies have corroborated these results, further highlighting the effect of inadequate food availability on susceptibility to parasitism and reproductive performance (Hörnfeldt 1978; Sinclair 1974; Obendorf & McColl 1980; Davidson, McGhee & Nettles 1980; Gulland 1992; Appleby, Anwar & Petty 1993). Heavily parasitized, malnourished individuals are regularly observed in natural environments; however, it is not clear whether malnutrition is the cause of infection or vice versa. At present, few experimental studies have examined these questions in wild populations, and none have evaluated these effects in birds (Murray, Cary & Keith 1997; Ives and Murray 1997; Pedersen and Greives 2008).

Laboratory studies typically evaluate the effect of a single parasite strain or species on a host (Sternberg *et al.* 2011). However, host populations in natural environments frequently harbor multiple different parasite strains and species (Petney & Andrews 1998; Cox 2001; Rigaud & Haine 2005). Previous studies have demonstrated that concomitant parasite infections do not necessarily confer synergistic effects of each infection (Malakar *et al.* 1999; Thomas, Fauchier & Lafferty 2002; Druilhe, Tall &

Sokhna 2005; Haine, Boucansaud & Rigaud 2005; Pedersen & Fenton 2007). The results of these studies have been mixed, with some showing hosts suffering a greater cost from concomitant infections (Read & Taylor 2001), and others showing hosts benefiting from concomitant infections, where parasites competing for similar resources limit one another's growth (Dobson & Barnes 1995; Read & Taylor 2001; Ishii *et al.* 2002).

Avian malaria is a disease with a cosmopolitan distribution caused primarily by three genera of haemosporidian parasites: *Plasmodium*, *Leucocytozoon*, and *Haemoproteus*. The three types of blood parasites are very widespread in birds and are found in every region of the globe excluding Antarctica (Valkiūnas 2005). Currently, 206 species of haemosporidian parasites have been documented to infect wild avian populations (Valkiūnas 2005; Bennett, Peirce & Ashford 1993). This disease can be fatal in early, acute stages, though chronic, sublethal infections appear to have few overt symptoms on the host. However, recent studies have demonstrated that even chronic infections can have detrimental effects on birds. Diminished body condition and immunity, as well as reduced survival, have been documented in avian species harboring chronic haemoparasite infections (Sol, Jovani & Torres 2003; Stjernman, Råberg & Nilsson 2004; Valkiūnas 2005; Bonier *et al.* 2007; Millington *et al.* 2007). Haemoparsite infections have also been documented impacting the reproductive success of avian hosts, however, results of these studies have been mixed, with some providing evidence for a positive effect of haemoparasite infection on reproductive success, and others demonstrating a negative effect (Marzal *et al.* 2005, 2008; Kilpatrick & LaPointe 2006; Tomás *et al.* 2007). Furthermore, it is unclear on which part of the reproductive cycle haemoparasite infections have the most significant impact.

Free-ranging birds will often carry multiple genera of haemosporidian parasites, henceforth referred to as "coinfections" (Marzal et al., 2008; Valkiūnas, Iezhova & Shapoval 2003; Palinauskas et al. 2005). Birds carrying multiple haemoparasite infections may experience anemia, reduced body mass, and survival (Graham *et al.* 2005; Davidar & Morton 2006). However, results from haemosporodian coinfection studies on avian populations have been equivocal, with some studies showing compounding negative effects of coinfection (Evans & Otter 1998), while others failing to show a relationship between coinfection level and various aspects of reproduction (Sanz *et al.* 2001; Marzal *et al.* 2008).

Here, we present data on the effect of haemoparasite coinfections and food supplementation on the reproductive success, daily nest survival, and offspring condition in a free-living population of a temperate zone, high elevation migratory songbird, the Mountain White-crowned Sparrow (*Zonotrichia leucophrys oriantha)*. This study disentangles

impacts on reproductive success by separating and evaluating the three recognized avian breeding phases: egg production, incubation, and raising nestlings to independence (Monaghan & Nager 1997). Additionally, daily nest survival (DNS), the probability that an individual nest survives one day (Dinsmore, White & Knopf 2002), was included as a response variable to provide an all encompassing metric of reproductive success that spans the whole duration of the breeding period. Fitness costs to offspring were analyzed by using nestling mass and tarsus length, two measurements broadly thought to reflect future quality of offspring (Perrins 1964, 1965; Garnett 1981). This study thus aims to discern how coinfection levels and food supplementation impact reproductive success and offspring quality, and how the interaction between coinfection levels and food supplementation impacts reproductive success and offspring quality.

Methods

Study Site - Over the span of six breeding seasons (2000 – 2005) we captured Mountain White-crowned Sparrows (*Zonotrichia leucophrys oriantha*) on four field sites, located along the East River Valley in the Gunnison National Forest, (Gothic, Gunnison County, Colorado, USA, 38° 57' 33" N, 106° 59' 21" W). The four study plots were similar in size (200m by 400 m) and were separated by >200m wide buffers of natural habitat. They were located in alpine meadow habitat that was interspersed by willow thickets (*Salix spp*), bog birch (*Betula glandulosa*), and mountain alder (*Alnus tenuifolia*). Elevation of the plots ranged between 2,900 to 2,990 m asl (Dietz *et al.* 2013).

Focal Population - The Mountain White-crowned Sparrow (*Z. l. oriantha*) is a socially monogamous migratory sparrow that breeds and nests in the alpine meadows of western North America (Morton 2002). The sparrows return to their breeding grounds from their Mexican overwinter sites in mid-May, and construct ground nests at the base of dense brushy vegetation (Morton 2002). A substantial amount of research has been conducted on the species, providing us with a detailed understanding of its' diet, behavior, physiology, and breeding biology (for reviews see Chilton *et al.* 1995; Morton 2002). Additionally, previous studies have documented the presence and transmission of haemosporidian parasites in this species (Murdock, Foufopoulos & Simon 2013; MacDougall-Shackleton *et al*. 2005).

Bird Capture and Sample Collection - During each summer season (May-July) sparrows were trapped daily (06:00am-12:00pm) using millet-baited Potter traps. All sparrows were banded at first capture with a uniquely numbered US Fish and Wildlife Service band (Bird Banding

Laboratory, Patuxent, MD, USA). After a bird was captured, standard morphometric measures were recorded and a blood sample was collected from the brachial vein into heparinized micro-capillary tubes. A small amount of blood from each sample was used to prepare a blood smear on a microscope slide, which was then air-dried and stained using the Fisher Hema 3 Stat pack™ (Fisher Scientific, Middleton, VA, USA). Blood smears were microscopically scanned for 20 minutes at 1,000x magnification to determine haemoparasite infection status (uninfected, single genus infection, two-genera infection, three-genera infection). Previous studies in this and other species have demonstrated that, if conducted for the appropriate period of time, optical microscopy is sufficient to detect the vast majority of haemoparasite infections (Merila & Andersson 1999; Kilpatrick & LaPointe 2006; Gilman, Blumstein & Foufopoulos 2007; Martínez-de la Puente *et al.* 2010; Knutie, Waite & Clayton 2013).

Food Supplementation - Each year, two non-adjacent experimental plots were supplemented daily with millet seed (*Panicum miliaceum*). The two remaining plots did not receive food supplementation and served as controls. To avoid legacy effects, experimental treatment of each plot alternated between years. Supplementation sites were distributed throughout each plot, and the number of supplementation sites on each plot varied according to plot size. Food supplementation site locations on each study plot remained invariant over the duration of the study and seed was provided on a daily basis. Food supplementation sites were placed at the edges of willow thickets and in areas where sparrows forage naturally (Chilton *et al.* 1995; Morton 2002). The same locations were also used to place the Potter traps utilized to capture birds. Early field observations suggested that visitation to food supplementation sites varied widely among individual sparrows. To quantify the effects of food supplementation treatment on an individual bird, we measured frequency of visitation of each bird to the feeding sites. This metric was calculated by dividing the number of times that an individual sparrow was captured at a feeding site over a season, with the total capture effort (number of trap-hours) on that plot.

Drug Administration - Half of the sparrows in the study received an antiprotozal agent. Birds, randomly assigned an odd-numbered FWS band received the antiprotozal treatment, and even numbered bands did not receive treatment. Birds were administered an aqueous solution of sulfadimethoxine (50 mg/kg) and pyrimethamine (1 mg/kg), a solution shown to subdue avian protozal infections (Huchzemeyer 1996; Foufopoulos 1999; Fukui *et al.* 2002). A graduated rubber-tipped syringe was used to administer the drug orally. (See Gilman, Blumstein & Foufopoulos 2007), for complete discussion of drug administration methods).

Nest Searching and Monitoring - Nests were located on the study plots either through opportunistic searches or through targeted observation of parental nest-associated behaviors (McDonald & Greenberg 1991). Once a nest was found, nest location was marked and we recorded the color-band combination of the parents associated with each nest. Nests were monitored every two days until nest failure or fledging. On each nest visit we recorded date, time of visit, number of eggs, nestlings, and fledglings. Nestling mass and tarsus length were measured on Day 7 after hatching.

A nesting attempt was considered complete when all nestlings had either fledged, or otherwise disappeared from the nest, or if parents abandoned the nest before the fledging date. A nest was considered successful if ≥ 1 nestlings fledged. We concluded fledging had occurred if we found the nest cup empty, and we observed parents carrying food to fledglings, or heard fledglings begging for food. A nest was considered to have failed if there were signs of abandonment (dead nestlings or cold eggs) or there were signs of predation (empty nest, damaged nest cup, broken eggs, etc.).

All research was conducted using the appropriate federal, state, and institutional permits: a federal banding permit (#2328), a Colorado Fish and Wildlife Service License (09TRb1094), and University Committee on Use and Care of Animals at the University of Michigan (Permit Number: 09077).

Statistical Analysis

Nest Survival - Daily nest survival (DNS) is the daily survival probability of a nest to survive one day, while the nest was under observation (Dinsmore *et al.* 2002) . We modeled nest success using standard nest survival models (Mayfield 1961, 1975; Dinsmore, White & Knopf 2002; Rotella, Dinsmore & Shaffer 2004) implemented in the program MARK (White & Burnham 1999) with RMark interface (Laake & Rexstad 2007) for the statistical and programming package R (R Development Core Team 2013). Using these models, we tested for the effects of blood parasite coinfection level, food visitation frequency, drug administration, individual bird identity, site, and year. The DNS models explicitly included distinct variables for each sex, as males and females, have separate roles during reproduction (Morton 2002). These model results were then used to evaluate the cumulative impact of the aforementioned variables on nest survival over the total period of use of a nest (30 days).

Reproductive Success – Three additional measures of reproductive success (clutch size, probability of an egg hatching, and probability of a nestlings fledging) were modeled with generalized linear mixed models (GLMM), using the statistical computing software R (R Core Team 2013). GLMM

with a log-link function and assuming Poisson distributions were used to analyze clutch size, and GLMM with a logit-link function and a binomial distribution were used to analyze the probability of an egg hatching, as well as the probability of a nestling fledging. To examine the effect of avian blood parasites and food supplementation on the three aforementioned reproductive success measures we created an *a priori* candidate model set for each response variable. All models included the variables year and identification number as random effects to account for potential pseudoreplication (Burnham & Anderson 2002). For clutch size and probability of an egg hatching, the *a priori* candidate model sets included in addition site, blood parasite coinfection level, and female food visitation frequency, as males are not known to play an significant role in the egg-laying or incubation stages of reproduction (Morton 2002). For the proportion of hatchlings that fledge, the *a priori* candidate model set included year, site, as well as food visitation frequency and blood parasite coinfection for both the males and the females, since both parents are involved in raising the nestlings (Morton 2002).

Offspring Quality – Young tarsus length (in *mm*) and young tarsus mass (in *g*) were modeled using linear mixed-effects models (LMEM). To examine the effect of avian blood parasites and food supplementation on the young mass and tarsus length we created an *a priori* candidate model set for each response variable. Every model included the variables year and identification number as random effects. The *a priori* candidate model sets for young mass and tarsus length included food visitation frequency, blood parasite coinfection level, drug administration, number of fledglings per nest, site, and year.

Akaike's information criterion for small sample sizes (AIC_c) was used to identify the most parsimonious model and evaluate model support in each model candidate set (Burnham & Anderson 2002). We selected the most parsimonious model as the model with the lowest AIC_c score. Models with $\Delta AIC_c < 2.0$ units of the most parsimonious model were considered equally supported (Burnham & Anderson 2002). Model Akaike weights (w_i) were calculated for each model and were used to infer the probability of the model being the best model in the candidate model set (Burnham & Anderson 2002). Relative variable importance was calculated by summing model Akaike weights for every model that included the variable.

Results

Haemoparasite Occurrence - We located and followed 235 sparrow nests over six breeding seasons (2000 – 2005). We scored blood smears from 113 females and 137 males for the presence of haemoparasites. Three genera of haemosporidian parasites: *Plasmodium*, *Leucocytozoon*, and

Haemoproteus, were present in our study population. 88% and 89% of the females and males respectively, were infected with at least one genus of blood parasite. Of the infected females 51% harbored a single parasite genus, 36% were infected with two parasite genera, and 12% were infected with all three parasite genera. Of the infected males, 57% were infected with one parasite genus, 38% were infected with two parasite genera, and 8% were infected with three parasite genera.

Antimalarial drug treatment was effective in reducing the haemosporidian infections in the study population (Fisher's exact test: χ^2=109.55, p = $2.2*10^{-16}$). Since the antiprotozal agent was not completely effective in clearing haemosporidian infections, blood smear data, instead of treatment status, was used to determine infection level for each individual.

Food Supplementation - Sparrows regularly visited the designated food supplementation locations to consume millet seed. Visitation to food supplementation sites was determined by the number of times a bird was trapped over the course of the summer at these sites, corrected for trapping effort–this number varied widely between individual birds. Thus, while the average female food visitation frequency was 0.0023 ± 0.0004 *captures/trapping hour*; value range varied from 0-0.0172 *captures/ trapping hour* (n=164). Average Male food visitation frequency is 0.0031 *captures/ trapping hour* ± 0.0006; Range: 0-0.0268, n=181. Additionally, food supplementation did not impact coinfection status in females (Fisher's exact test: χ^2=6.26, p = 0.10) nor males (Fisher's exact test: χ^2=4.37, p = 0.23). Thus, birds consuming seed did not have a significantly different number of coinfections than birds not consuming seed.

Estimates of Daily Nest Survival - The most parsimonious model (w_i = 0.28) analyzing DNS included Female food visitation ($Food_{FEM}$), Female ID number (ID_{FEM}), Site, and Year (Table 1A). One additional model (w_i = 0.11) was within $\Delta AIC_c \leq 2$ of the most parsimonious model, and added Number of Female blood parasite coinfections ($Coinfection_{FEM}$) to the most parsimonious model. When calculating the relative importance of individual fixed effect variables we found strong support for effects of Female food visitation (relative variable importance = 0.73) and some support for Number of Female blood parasite coinfections (0.36). Female food visitation was strongly and positively related to DNS [in the most parsimonious model, $\hat{\beta}_{Food_{FEM}}$ = 0.27 (1 SE=0.16, 95% CL = -0.05, 0.56) on the logit scale (Fig. 1)]. The model that included Number of Female blood parasite coinfections was equally supported, in the second best model $\hat{\beta}_{Coinfection_{FEM}}$ = -0.06 (1 SE = 0.14, 95% CL=-0.33, 0.21) and $\hat{\beta}_{Food_{FEM}}$ = 0.28 (1 SE=0.16, 95% CL = -0.04, 0.60), on the logit scale.

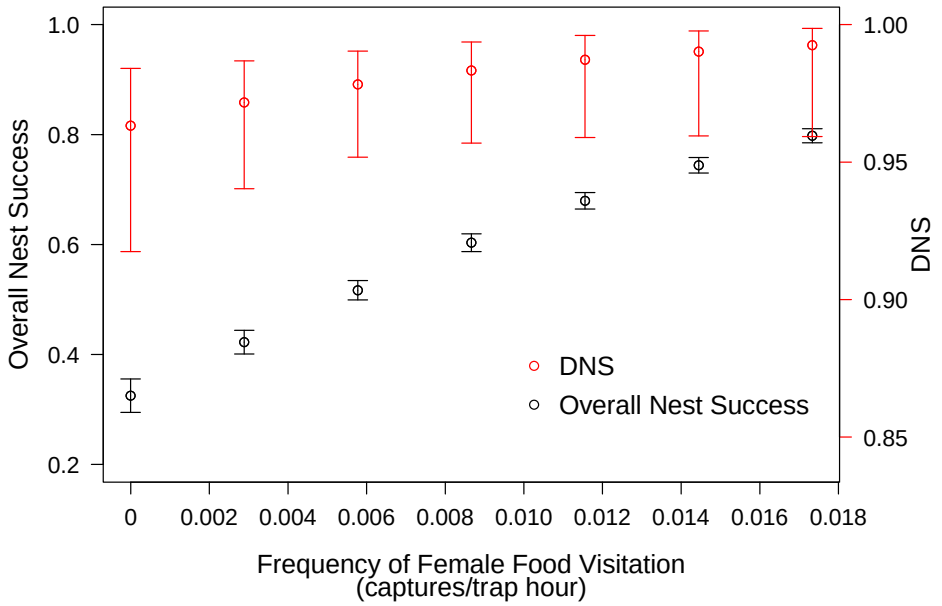

Figure 1. White-crowned Sparrow overall nest success and daily nest survival (with 95% confidence levels) for the Female food visitation frequency (*captures/ trap hour)*.

Overall Nest Success over the whole period of nest use was determined using the formula:

$$Overall\ Nest\ Success = (DNS)^{Number\ of\ Days\ of\ Nest\ Use}.$$

Consequently, the true impact of Female food visitation on sparrow reproduction becomes fully apparent when considering the total breeding period, 30 days (Fig. 1). Indeed whereas the range of DNS across the Female food visitation frequency gradient appears narrow (0.96-0.99), the corresponding overall Nest success varies much more widely (0.33-0.80).

Clutch Size – The model that received the strongest support (w_i = 0.38) from the GLMM analysis on clutch size was the null model (Table 1B). Two additional models received support within $\Delta AIC_c \leq 2$. The second best model (w_i = 0.15) included Female drug administration ($Drug_{FEM}$), as well as Female ID number, and Year. The third best model (w_i =0.14) included Female food visitation in addition to Female ID number, and Year (Table 1B). Relative importance of individual fixed effect variables was calculated, and some support was found for the effects of antimalarial drug administered ($Drug_{FEM}$: relative variable importance = 0.29), for the extent of food visitation ($Food_{FEM}$: 0.28), and for a negative impact of infections ($Coinfection_{FEM}$: 0.27). In the second best model $\hat{\beta}$

$Drug_{FEM}$ = 0.07 (1 SE = 0.12, 95% CL = - 0.17, 0.31). In the third best model $\hat{\beta}\ Food_{FEM}$ = -0.007 (1 SE = 0.02, 95% CL = -0.04, 0.02).

Probability of an Egg Hatching – The best model (w_i = 0.33) describing the probability of an egg hatching included: Female food visitation, in addition to Year, Site, Female ID number, (Table 1C). A second best model (w_i = 0.16) within $\Delta AIC_c \leq 2$ was similar to the most parsimonious model but excluded Site. Relative importance of individual fixed effect variables was calculated and strong support was found for Female food visitation (relative variable importance = 1.0), while some support was found for Female drug administration (0.29) and Number of Female blood parasite coinfections (0.31). Strong support was found for models that included Female food visitation, which had a positive relationship to probability of hatching ($\hat{\beta}\ Food_{FEM}$ = 0.37 (1 SE = 0.10, 95% CL = 0.19, 0.60) (Fig. 2.A), on the logit scale. The second best model, also gave similar results for Female food visitation ($\hat{\beta}\ Food_{FEM}$ = 0.34 (1 SE = 0.10, 95% CI = 0.16, 0.57), on the logit scale.

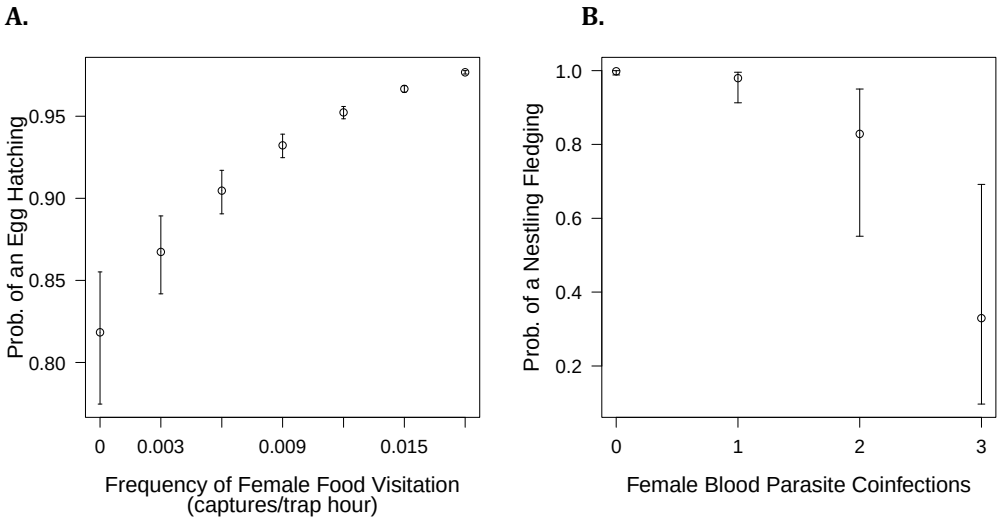

Figure 2A. (Left) The probability of a White-crowned Sparrow egg hatching (with 95% confidence levels) for the Female food visitation frequency (*captures/ trap hour).*
Figure 2B. (Right) The probability of a White-crowned Sparrow nestling fledging (with 95% confidence levels) as a function of the Number of Female blood parasite coinfections.

Probability of a Nestling Fledging - The most parsimonious model (w_i = 0.37) analyzing the probability of a nestling fledging included Year, Female ID number, and Number of Female blood parasite coinfections (Table 1D). The second best model (w_i = 0.20) (within $\Delta AIC_c \leq 2$) included in addition Female drug administration. Relative importance of individual fixed effect variables was calculated and strong support was found for Number of Female blood parasite coinfections (relative variable importance = 0.97). Some support was also found for Female drug

administration (0.36). Models that included Number of Female blood parasite coinfections, were strongly supported ($\hat{\beta}\ Coinfection_{FEM}$ = -2.29 (1 SE = 0.90, 95% CL = -4.77, -0.88), in the most parsimonious model on the logit scale (Fig. 2.B). Number of Female blood parasite coinfections had a negative effect on probability of a nestling fledging in all models where it was incorporated. In the second best model, Female drug administration received some support, $\hat{\beta}\ Drug_{FEM}$ = 1.43 (1 SE = 1.29, 95% CL = -1.31, 4.46).

Offspring Condition – No model was clearly superior for young tarsus length (Table 1E). The best model (w_i = 0.11) included Number of male blood parasite coinfections, Number of fledglings, Site, Male ID number, and Year, followed closely by the null model (w_i = 0.10). Seven additional models received support within $\Delta AIC_c \leq 2$. In the most parsimonious model $\hat{\beta} Coinfection_{MALE}$ = -0.44 (1 SE = 0.30, 95% CL= -1.05, 0.18).
Of the models considered for Young body mass, no model was clearly superior. The most parsimonious model for Young body mass was the null model. Six additional models received support within $\Delta AIC_c \leq 2$ (Table 1F).

Discussion

This is the first study to evaluate the effects of haemoparasite blood infections and food supplementation on reproductive success in an avian system. Our results indicate that avian haemoparasites have important negative effects on the reproductive success of a free-living avian population and that these impacts manifest themselves mostly in the young-rearing phase. We also documented a positive effect of food supplementation during the incubation phase and on DNS. Interestingly, there was no effect of disease or food supplementation on young mass or young tarsus length. And surprisingly, we did not detect any interaction between food supplementation and blood parasite coinfection on any metrics of reproductive success.

While the majority of studies of haemosporidians infecting domestic birds have documented the high mortality rates of these parasites (Valkiūnas 2005), other observational studies of wild birds have offered equivocal results as to whether malaria has overt detrimental effects on avian hosts (Davidar & Morton 1993; Tomás *et al.* 2005, 2007; Kilpatrick & LaPointe 2006; Bensch *et al.* 2007; Marzal *et al.* 2008; Martínez-de la Puente *et al.* 2010; Knutie *et al.* 2013). These latter studies in conjunction with the fact that most chronic infections involve very low parasitemias, may have led to the notion that avian malaria infections are generally of little consequence to avian hosts (Bennett, Peirce & Ashford 1993).

In contrast, our results show that avian malaria infections (*Plasmodium, Leucocytozoon, and Haemoproteus*) can have important detrimental effects on the reproductive success of a free-living avian population where malaria is endemic. The deleterious effects of haemoparasite infections on reproduction become increasingly more severe in individuals carrying single, double, or triple blood parasite coinfections. Our results demonstrate that the most negative consequences of malaria infections occur during the young-rearing phase, specifically, in lowering the probability of a nestling fledging.

Mixed haemoparasite infections can be found regularly in free-ranging populations (Valkiūnas, Iezhova & Shapoval 2003; Palinauskas *et al.* 2005). Studies evaluating the effect of multiple infections on host condition have yielded mixed results (Mosquera & Adler 1998; Taylor, Mackinnon & Read 1998; De Roode *et al.* 2003). The host may have a significantly harder time mounting an immune response when faced with multiple concomitant infections (Combes 2001). In this study, uninfected females had a 0.99 probability of fledging their nestlings, individuals infected with a single infection had a 0.97 probability, individuals with a double coinfection had a 0.83 probability, and individuals with a triple coinfection had a 0.39 probability, demonstrating the detrimental reproductive success costs of an avian malaria infection. Thus, the effects of concomitant infections on the probability of fledging nestlings are not additive, but exponential.

Birds harboring multiple infections experienced more pronounced negative fitness consequences than healthy individuals or those with single infections. Malarial infections have been shown to have direct detrimental effects on host condition (Desser & Bennett 1993; Swinnerton *et al.* 2005; Valkiūnas 2005). Haemoparasite infections often decrease hematocrit, obstruct cerebral capillaries, and cause inflammation of skeletal muscle tissue (Valkiūnas 2005, Atkinson 2008). Additionally, negative effects on food provisioning rates, predation avoidance, and overall parental care abilities have been shown in infected individuals (Merino *et al*. 2000; Tomás *et al.* 2007; Møller & Nielsen 2007; Knowles, Palinauskas & Sheldon 2010). Malaria infections, even in chronic stages, cause an up-regulation of the immune system (Atkinson 2008). Recent research has shown that mounting an immune response is demanding of host energy and other resources, and the finite energy reserves of the individual impose energetic constraints on the host (Sheldon & Verhulst 1996; Ilmonen, Taarna & Hasselquist 2000; Zuk & Stoehr 2002). While the complex nature of the immune system presents a challenge for quantifying both the direct energetic burden, as well as the indirect costs associated with an immune response, several studies have suggested the existence of a trade-off between an immune response and reproductive performance (Folstad & Karter 1992; Gustafsson *et al*. 1994;

Sheldon & Verhulst 1996). Studies analyzing the trade-off between immune defense and reproductive effort use experimental challenge techniques of the immune system (Ilmonen, Taarna & Hasselquist 2000; Martin, Scheuerlein & Wikelski 2002; Ardia 2005), as well as experimental manipulation of parental effort (Hõrak, Ots & Murumägi 1998; Nordling *et al.* 1998; Ardia 2005) to demonstrate that immune responses and life-history decisions are constrained by a finite amount of nutrients and energy. Thus, haemoparasites can impact reproduction both directly by undermining the very function of the tissues needed to reproduce, as well as indirectly, by forcing tradeoffs e.g. between immunity and reproduction, that ultimately reduce the amount of resources available for reproductive investment.

Until recently, it has been assumed that the incubation period is a time of reduced energy expenditure, compared to other breeding periods (Williams 1996, Monaghan & Nager 1997). Incubation within the thermally neutral zone incurs relatively low energetic expenditures, however, once outside that thermal neutral zone, incubating birds may experience steep increases in metabolic rate in the range of 19-50%, above the baseline (Williams 1996). Additional energetic demands occur after a bird returns from a foraging bout and must rewarm the eggs to an optimal temperature (Williams 1996). This is a particularly acute issue for White-crowned sparrow females attempting to keep themselves and their eggs warm during frigid mountain nights (Morton 2002), with temperatures which in our field sites always drop below 0^{o}C. Our results do not suggest that malarial infections have a pronounced impact on the incubation stage. This departs from two other studies that have documented a negative effect of avian malaria on the incubation stage of reproduction (Marzal *et al.*, 2005; Knowles *et al.,* 2010). Our results do however suggest that food supplementation plays a critical role in clutch incubation and hatching success. More specifically, we find a monotonic increase in hatching success across the whole range of food supplementation frequencies (Fig. 2.A). Females nesting in food supplemented plots have stable access to free calories, which are likely to be beneficial during the energetically expensive process of incubation under frigid conditions. Thus, it is reasonable to expect that females who visit food supplementation sites more frequently will have more energy to devote to incubation, and therefore have a higher probability of hatching the eggs.

In this study population individuals with a triple haemoparasite coinfection fledged, on average, 1.9 less offspring than uninfected individuals. Thus, the reproductive cost of a malaria infection for the White-crowned sparrow population can be quantified as almost two less offspring per nest (Fig. 3). Additionally, food supplemented birds hatched, on average, 0.5 additional eggs than individuals that did not receive food

supplementation. There is a clear temporal component to the impacts of food supplementation and haemoparasite infection on the reproductive cycle. The beneficial effects of food are seen early in the reproductive cycle, whereas the deleterious effects of disease are observed later. Thus, the positive effects of food are observed early in the summer when temperatures are colder and suitable food sources are scarce and covered by snowpack (Morton 2002). The negative impacts of disease occur late in the reproductive cycle when blood infections, after being subpatent during the summer, recrudesce (Lapointe, Atkinson & Samuel 2012). Furthermore, during the nestling phase, millet is not a sufficient food source for sparrows, as the young require a high protein arthropod diet instead (Searcy, Peters & Nowicki 2004).

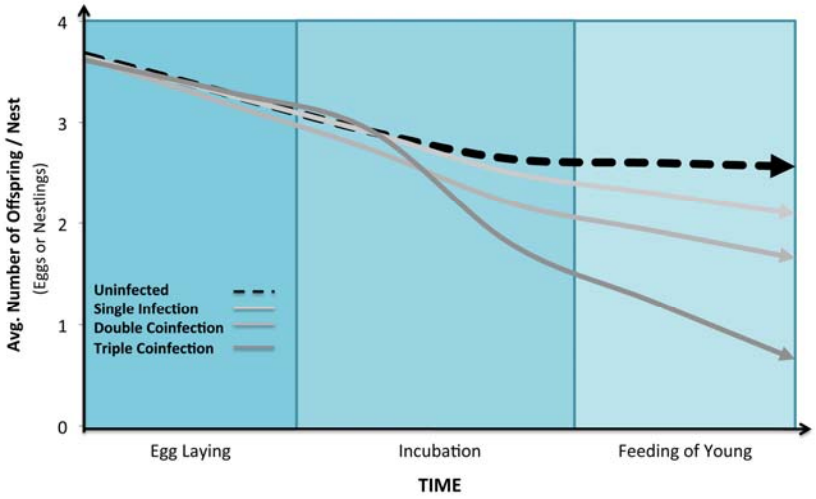

Figure 3. Average cumulative impacts (per nest) of malaria infection on the reproduction of white crowned sparrows across successive stages of the breeding season.

Our data show that food supplementation has a positive effect on DNS. Parents in food supplemented sites may spend less time foraging for food, and more time defending their nest, thereby increasing DNS rates because of reduced nest predation (Arcese & Smith 1988). There was modest support for Number of Female blood parasite coinfections on DNS. Studies evaluating the effect of malaria on avian survival have presented mixed results, with some studies demonstrating no effect of infection on DNS (Kilpatrick & LaPointe 2006; Lachish *et al.* 2011), and others showing infection to have a negative effect on DNS (Marzal *et al.* 2008). One reason for the lack of a strong effect may be due to the fact that the observed negative effect of malaria is focused on one stage of reproduction, the young-rearing phase, while DNS considers the whole reproductive period (for most of which there is no effect of malaria). Therefore, it is possible, that the negative effect of malaria is diluted to the point where it drops below levels needed to achieve significance. In addition, it may stem from the fact that the study birds appear to be,

despite their infection status, in very good condition as they are able to successfully complete reproduction.

Our results did not provide support for effects of blood parasite coinfection or food supplementation on clutch size. Because egg production is considered a non-trivial energetic cost to the female, many studies have focused on the effects of food availability on clutch size (Ewald & Rohwer 1982; Davies & Lundberg 1985; Slagsvold & Lifjeld 1988, 1990; Morton 2002). Interestingly, these studies did not show an effect of food supplementation on clutch size, suggesting that it is not tightly reliant on food supply (Daan *et al.* 1989; Rowe, Ludwig & Schluter 1994). When it comes to the impacts of infection on clutch size, relatively little information exists. One study (Marzal *et al.* 2005) showed that uninfected house martins (*Delichon urbica)* had 18% larger clutch size than conspecifics infected with avian malaria. While the energetic costs of egg production are not insignificant, they are considerably less than the costs incurred during the young rearing phase (Williams 1996). It is thus reasonable to expect a much greater toll of malarial infection and food supplementation on the latter, more energetically costly, stages of reproduction. Our present study demonstrates that the more energetically demanding breeding phases, the incubation and young rearing phases, are impacted by food supplementation and avian malaria infection respectively.

We did not detect any effects of blood parasite coinfection or food supplementation on young mass or tarsus length. Past studies evaluating the effect of avian malaria on offspring quality have produced mixed results; some studies documenting an effect of malaria on offspring quality (Knowles, Palinauskas & Sheldon 2010) and others showing no effect (Marzal *et al.* 2005; Knutie, Waite & Clayton 2013). Similarly, studies focusing on the effect of food supplementation on offspring quality have presented equivocal results (Arcese & Smith 1988; Richner 1992; Wiebe & Bortolotti 1994; Wiehn & Korpimaki 1997; Marzal *et al*. 2008). One explanation for the absence of the aforementioned effect is that infected parents were still able to devote sufficient energy to not compromise the quality of offspring or that the shortcomings of an infected parent were counterbalanced by the second parent. Preliminary information (Lombardo 2010) suggests that haemoparasite infection may simply not alter provisioning rates of the parents, allowing the offspring to develop at the same rate of offspring born to healthy parents.

Equally important to what we found were the things we did not detect: There was no interaction between food supplementation and blood parasite coinfection for any of the five response variables evaluated. Additionally, food supplementation did not influence the coinfection level of the birds, and coinfection level did not affect the eating habits of the

birds. It would have been reasonable to expect either that infected parents would visit food supplementation sites more often or that food supplementation would have ameliorated the impacts of infection on the reproduction. None of these were observed. One possible explanation is that the high carbohydrate energy the sparrows received from the millet was not the appropriate food to help fend off infection. The millet seed supplemented to the birds, while a carbohydrate-rich source of energy (Issoufou, Mahamadou & Guo-Wei 2013), did not contain all vitamins and minerals essential for mounting an effective immune response (Cunningham-Rundles 2002).

Our results indicate that avian haemoparasites have a significant negative impact on the reproductive success of a free-living avian population. However, the true impact of haemoparasite infections on the population is likely to be much larger, since we have ample evidence that prevalence of haemoparasites is higher in the non-breeding population (JF, unpublished data). This study is solely evaluating the reproductive success of individuals that establish a territory and build a nest. It is probable that many individuals fail to progress past the two aforementioned stages, and are thus excluded from the analysis. Therefore, it is likely that the true impact of haemoparasite infections surpass the effects shown here, given that many infected birds don't even get to the stage of establishing a territory and attracting a mate. Thus, our results should be taken as a conservative estimate of the costs of haemoparasite infection on reproductive success in avian populations.

Table 1.

A.	Dependent Variable: Daily Nest Survival	K	ΔAIC_c	w_i
	$Year + ID_{FEM} + Site + Food_{FEM}$	11	0.00	0.28
	$Year + ID_{FEM} + Site + Food_{FEM} + Coinfection_{FEM}$	12	1.82	0.11
	$Year + ID_{FEM} + Site + Food_{FEM} + Drug_{FEM}$	12	2.02	0.10
	$Year + ID_{FEM} + Site + Coinfection_{FEM}$	11	3.20	0.06
	$Year + ID_{FEM} + Site + Drug_{FEM}$	11	3.26	0.05
	$Year + ID_{FEM} + ID_{MALE} + Site$	11	3.28	0.05
	$Year + ID_{FEM} + Site + Food_{FEM} + Coinfection_{FEM} + Food_{FEM} * Coinfection_{FEM}$	13	3.34	0.05
	S(.)	1	3.51	0.05
	$Year + ID_{FEM} + Site + Food_{FEM} + Coinfection_{FEM} + Drug_{FEM}$	13	3.82	0.04
B.	Dependent Variable: Clutch Size	K	ΔAIC_c	w_i
	$Year + ID_{FEM} + 1$	3	0.00	0.38
	$Year + ID_{FEM} + Drug_{FEM}$	4	1.87	0.15
	$Year + ID_{FEM} + Food_{FEM}$	4	1.99	0.14
	$Year + ID_{FEM} + Coinfection_{FEM}$	4	2.21	0.13
	$Year + ID_{FEM} + Food_{FEM} + Drug_{FEM}$	5	3.94	0.05
C.	Dependent Variable: Probability of Hatching	K	ΔAIC_c	w_i
	$Year + ID_{FEM} + Site + Food_{FEM}$	7	0.00	0.33
	$Year + ID_{FEM} + Food_{FEM}$	4	1.50	0.16
	$Year + ID_{FEM} + Site + Food_{FEM} + Coinfection_{FEM}$	8	2.03	0.12
	$Year + ID_{FEM} + Drug_{FEM}$	8	2.36	0.10
	$Year + ID_{FEM} + Food_{FEM} + Drug_{FEM}$	5	3.71	0.05
	$Year + ID_{FEM} + Food_{FEM} + Coinfection_{FEM}$	5	3.73	0.05
D.	Dependent Variable: Probability of Fledging	K	ΔAIC_c	w_i
	$Year + ID_{FEM} + Coinfection_{FEM}$	4	0.00	0.37
	$Year + ID_{FEM} + Coinfection_{FEM} + Drug_{FEM}$	5	1.21	0.20
	$Year + ID_{FEM} + Coinfection_{FEM} + Food_{FEM}$	5	2.43	0.11
	$Year + ID_{FEM} + ID_{MALE} + Coinfection_{FEM} + Coinfection_{MALE}$	6	3.64	0.06
	$Year + ID_{FEM} + Food_{FEM} + Coinfection_{FEM} + Drug_{FEM}$	6	3.73	0.06
E.	Dependent Variable: Young Tarsus Length *(mm)*	K	ΔAIC_c	w_i
	$Year + ID_{MALE} + Site + Fledge + Coinfection_{MALE}$	9	0.00	0.11
	$Year + ID_{FEM} + ID_{MALE} + Fledge + 1$	5	0.17	0.10
	$Year + ID_{FEM} + Site + Fledge + Drug_{FEM}$	9	0.58	0.08
	$Year + ID_{FEM} + Site + Fledge + Coinfection_{FEM}$	9	0.62	0.08
	$Year + ID_{MALE} + Site + Fledge + Food_{MALE}$	9	1.23	0.06
F.	Dependent Variable: Young Mass *(g)*	K	ΔAIC_c	w_i
	$Year + ID_{FEM} + ID_{MALE} + Fledge + 1$	5	0.00	0.19
	$Year + ID_{MALE} + Fledge + Food_{MALE}$	6	1.40	0.09
	$Year + ID_{FEM} + Fledge + Drug_{FEM}$	6	1.52	0.09
	$Year + ID_{FEM} + Fledge + Coinfection_{FEM} + Drug_{FEM}$	7	1.65	0.08
	$Year + ID_{FEM} + Fledge + Coinfection_{FEM}$	6	1.70	0.08

Table 1. Summary of model selection results for: (**A**) Daily Nest Survival, (**B**) Clutch Size, (**C**) Probability of Hatching, (**D**) Probability of Fledging, (**E**) Young Tarsus Length (*mm*),

(**F**) Young Mass (g) of White-crowned Sparrows breeding in Colorado. Models are ranked in order of increasing ΔAIC_c. Number of parameters (K), and AIC_c weights (w_i) are given for each model. Variables in models included Female food visitation frequency ($Food_{FEM}$), Male food visitation frequency ($Food_{MALE}$), Number of Female blood parasite coinfections ($Coinfection_{FEM}$), Number of male blood parasite coinfections ($Coinfection_{MALE}$), the interaction between Female Food visitation and Female Blood parasite coinfection ($Coinfection_{FEM}$ * $Food_{FEM}$), the interaction between Male Food visitation and Male Blood parasite coinfection ($Coinfection_{MALE}$* $Food_{MALE}$), Female Drug Administration ($Drug_{FEM}$), and Male Drug Administration ($Drug_{MALE}$) Number of fledglings per nest (Fledge), Site, Female ID (ID_{FEM}), Male ID (ID_{MALE}), and Year. For sub-tables **A-D** models with a ΔAIC_c score ≤ 4 were included and for sub-tables **E-F** the table includes the five models with the lowest ΔAIC_c score. For complete list of models included in the comparison see Appendix Section.

References

Albon, S.D., Stien, A., Irvine, R.J., Langvatn, R., Ropstad, E. & Halvorsen, O. (2002) The role of parasites in the dynamics of a reindeer population. *Proceedings of the Royal Society B: Biological Sciences*, 269, 1625–1632.

Allen, P., Gilchrist, H.G. & Smith, J.N.M. (2007) Effects of nest habitat, food and parental behavior on shorebird nest success. *Condor*, 109, 15–31.

Anderson, R.M. & May, R.M. (1978) Regulation and stability of host-parasite population interactions. *Journal of Animal Ecology*, 47, 219-247.

Anderson, R.M. & May, R.M. (1979) Population biology of infectious diseases: Part I. *Nature*, 280, 361–367.

Ankney, C.D. & Macinnes, C.D. (1978) Nutrient reserves and reproductive performance of female Lesser Snow Geese. *The Auk*, 95, 459–471.

Appleby, B.M., Anwar, M.A. & Petty, S.J. (1999) Short-term and long-term effects of food supply on parasite burdens in Tawny Owls, *Strix aluco*. *Functional Ecology*, 13, 315-321.

Arcese, P. & Smith, J.N.M. (1988) Effects of population density and supplemental food on reproduction in song sparrows. *Journal of Animal Ecology*, 57, 119-136.

Ardia, D.R. (2005) Individual quality mediates trade-offs between reproductive effort and immune function in tree swallows. *Journal of Animal Ecology*, 74, 517–524.

Atkinson, C.T. (2008) Avian Malaria. *Parasitic Diseases of Wild Birds* (eds C.T. Atkinson & D.B. Hunter), pp. 35-53. Wiley-Blackwell, Oxford, UK.

Begon, M., Harper, J.L. & Townsend, C.R. (2006) *Ecology: Individuals, Populations and Communities*, 3rd ed (eds J.L. Harper and C.R. Townsend). Wiley-Blackwell, Cambridge, Massachusetts.

Bennett, G.F., Peirce, M.A., & Ashford, R.W. (1993) Avian haematozoa: mortality and pathogenicity. *Journal of Natural History*, 27, 993-1001.

Bensch, S., Waldenström, J., Jonzén, N., Westerdahl, H., Hansson, B., Sejberg, D. & Hasselquist, D. (2007) Temporal dynamics and

diversity of avian malaria parasites in a single host species. *Journal of Animal Ecology*, 76, 112–122.

Bonier, F., Martin, P.R., Sheldon, K.S., Jensen, J.P., Foltz, S.L. & Wingfield, J.C. (2007) Sex-specific consequences of life in the city. *Behavioral Ecology*, 18, 121–129.

Burnham, K.K.P. & Anderson, D.R.D. (2002) *Model Selection and Multimodel Inference: A Practical Information-Theoretic Approach*, 2nd edn. Springer-Verlag, New York , NY.

Chilton, G., Baker, M.C., Barrentine, C.D. & Cunningham, M.A. (1995) White-crowned Sparrow (*Zonotrichia leucophrys*). The birds of North America. Number 183.

Combes, C. (2001) *Parasitism: The Ecology and Evolution of Intimate Interactions*. University of Chicago Press, Chicago, Illinois.

Cox, F.E. (2001) Concomitant infections, parasites and immune responses. *Parasitology*, 122, S23–S38.

Cox, D.T.C. & Cresswell, W. (2014) Mass gained during breeding positively correlates with adult survival because both reflect life history adaptation to seasonal food availability. *Oecologia*, 174, 1197–1204.

Cunningham-Rundles, S. (2002) Evaluation of the effects of nutrients on immune function. *Nutrition and Immune Function* (eds P.C. Calder, C.J Field & H.S Gill), pp. 21-40. CABI Publishing, Wallingford, UK.

Daan, S., Dijkstra, C., Drent, R. & Meijer, T. (1989) Food supply and the annual timing of avian reproduction. *Proceedings of the International Ornithological Congress*, 19, 392-407.

Davidar, P. & Morton, E.S. (1993) Living with Parasites: Prevalence of a Blood Parasite and Its Effect on Survivorship in the Purple Martin. *The Auk*, 110, 109–116.

Davidar, P. & Morton, E.S. (2006) Are multiple infections more severe for Purple Martins (*Progne subis*) than single infections? *The Auk*, 123, 141–147.

Davidson, W.R., McGhee, M.B. & Nettles, V.F. (1980) Haemonchosis in white-tailed deer in the southeastern United States. *Journal of Wildlife Diseases*, 16, 499-508.

Davies, N.B. & Lundberg, A. (1985) The influence of food on time budgets and timing of breeding of the Dunnock *Prunella modularis*. *Ibis*, 127, 100–110.

Desser, S.S. & Bennett, G.F. (1993) The genera of *Leucocytozoon*, *Haemoproteus*, and *Hepatocystis*. *Parasitic Protozoa* (ed P.J. Kreier), pp. 273–308. Academic Press, London, UK.

Dietz, M., Murdock, C., Romero, L., Ozgul, A., Foufopoulos, J. (2013) Distance to a road is associated with reproductive success and physiological stress response in a migratory landbird. *The Wilson Journal of Ornithology*, 125, 50–61.

Dinsmore, S.J., White, G.C. & Knopf, F.L. (2002) Advanced techniques for modeling avian nest survival. *Ecology*, 83, 3476–3488.

Dobson, R.J. & Barnes, E.H. (1995) Interaction between *Ostertagia circumcincta* and *Haemonchus contortus* infection in young lambs. *International Journal for Parasitology*, 25, 495–501.

Druilhe, P., Tall, A. & Sokhna, C. (2005) Worms can worsen malaria: Towards a new means to roll back malaria? *Trends in Parasitology*, 21, 359–362.

Evans, M. & Otter, A. (1998) Fatal combined infection with *Haemoproteus noctuae* and *Leucocytozoon ziemanni* in juvenile snowy owls (*Nyctea scandiaca*). *The Veterinary Record*, 143, 72–76.

Ewald, P.W. & Rohwer, S. (1982) Effects of supplemental feeding on timing of breeding, clutch-size and polygyny in red-winged blackbirds. *Journal of Animal Ecology*, 51, 429–450.

Folstad, I. & Karter, A.J. (1992) Parasites, bright males, and the immunocompetence handicap. *The American Naturalist*, 139, 603-622.

Foufopoulos, J. (1999) *Host-Parasite Interactions in the Mountain Spiny Lizard Sceloporus jarrovi*. University of Wisconsin, Madison.

Fukui, D., Murata, K., Bando, G., Kosuge, M. & Yamaguchi, M. (2002) Pyrimethamine/sulfadoxine treatment against mixed avian-haematozoa infections in imported Tengmalm's owls (*Aegolius funereus*). *Journal of Japaneese Vetrinary Medicine Association*, 55, 673-678.

Garnett, M.C. (1981). Body size, its heritability and influence on juvinile survival among great tits, *Parus major*. *Ibis*, 123, 31-41.

Gilman, S., Blumstein, D.T. & Foufopoulos, J. (2007) The effect of hemosporidian infections on White-Crowned Sparrow singing behavior. *Ethology*, 113, 437–445.

Graham, A.L., Lamb, T.J., Read, A.F. & Allen, J.E. (2005) Malaria-filaria coinfection in mice makes malarial disease more severe unless filarial infection achieves patency. *The Journal of Infectious Diseases*, 191, 410–421.

Grenfell, B.T. & Dobson, A.P. (1995) *Ecology of Infectious Diseases in Natural Populations*. Cambridge University Press, Cambridge, UK.

Gulland, F.M.D. (1992) The role of nematode parasites in Soay sheep (*Ovis aries, L.*) mortality during a population crash. *Parasitology*, 105, 493-503.

Gustafsson, L., Nordling, D., Andersson, M.S., Sheldon, B.C. & Qvarnström, A. (1994) Infectious diseases, reproductive effort and the cost of reproduction in birds. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 346, 323–331.

Haine, E.R., Boucansaud, K. & Rigaud, T. (2005) Conflict between parasites with different transmission strategies infecting an amphipod host. *Proceedings of the Royal Society B: Biological Sciences*, 272, 2505–2510.

Hamilton, W.D. & Zuk, M. (1982) Heritable true fitness and bright birds: a role for parasites? *Science*, 218, 384–387.

Hõrak, P., Ots, I. & Murumägi, A. (1998) Haematological health state indices of reproducing Great Tits: A response to brood size manipulation. *Functional Ecology*, 12, 750–756.

Hörnfeldt, B. (1978) Synchronous population fluctuations in voles, small game, owls, and tularemia in northern Sweden. *Oecologia*, 32, 141-152.

Huchzemeyer, F.W. (1996) *Plasmodium durae in Domestic Turkeys (Meleagris gallopavo) and Swainson's Francolin (Francolinus swainsoni): Characteristics, Pathology and Chemotherapy*. University of Pretoria, South Africa.

Hudson, P.J., Dobson, A.P. & Newborn, D. (1998) Prevention of population cycles by parasite removal. *Science*, 282, 2256–2258.

Hudson, P.J., Newborn, D. & Dobson, A.P. (1992) Regulation and stability of a free-fiving host-parasite system: *Trichostrongylus tenuis* in Red Grouse. I: Monitoring and parasite reduction experiments. *Journal of Animal Ecology*, 61, 477–486.

Ilmonen, P., Taarna, T. & Hasselquist, D. (2000) Experimentally activated immune defence in female pied flycatchers results in reduced breeding success. *Proceedings of the Royal Society B: Biological Sciences*, 267, 665–670.

Ing, R., Su, Z., Scott, M.E. & Koski, K.G. (2000) Suppressed T-helper 2 immunity and prolonged survival of a nematode parasite in protein-malnourished mice. *Proceedings of the National Academy of Sciences of the United States of America*, 97, 7078–7083.

Ishii, T., Takatsuka, J., Nakai, M. & Kunimi, Y. (2002) Growth characteristics and competitive abilities of a nucleopolyhedrovirus and an entomopoxvirus in larvae of the Smaller Tea Tortrix, *Adoxophyes honmai* (Lepidoptera: Tortricidae). *Biological Control*, 23, 96–105.

Issoufou, A., Mahamadou, E.G. & Guo-Wei, L. (2013) Millets: Nutritional composition, some health benefits and processing - A review. *Emirates Journal of Food and Agriculture*, 25, 501–508.

Ives, A.R. & Murray, D.L. (1997) Can sublethal parasitism destabilize predator-prey population dynamics? A model of snowshoe hares, predators, and parasites. *Journal of Animal Ecology*, 66, 265-278.

Kilpatrick, A.M. & LaPointe, D.A. (2006) Effects of chronic avian malaria (*Plasmodium relictum*) infection on reproductive success of Hawaii Amakihi (*Hemignathus virens*). *The Auk*, 123, 764–774.

Knowles, S.C., Palinauskas, V. & Sheldon, B.C. (2010) Chronic malaria infections increase family inequalities and reduce parental fitness: Experimental evidence from a wild bird population. *Journal of Evolutionary Biology*, 23, 557–569.

Knutie, S.A., Waite, J.L. & Clayton, D.H. (2013) Does avian malaria reduce fledging success: An experimental test of the selection hypothesis. *Evolutionary Ecology*, 27, 185–191.

Koski, K.G. & Scott, M.E. (2001) Gastrointestinal nematodes, nutrition and immunity: breaking the negative spiral. *Annual Review of Nutrition*, 21, 297–321.

Laake, J., Rexstad, E. (2007) RMark-an alternative approach to building linear models. Appendix C *in* Program MARK: a gentle introduction (eds E. Cooch & G.C White). Colorado State University, Fort Collins, USA.

Lachish, S., Knowles, S.C., Alves, R., Wood, M.J. & Sheldon, B.C. (2011) Fitness effects of endemic malaria infections in a wild bird population: The importance of ecological structure. *Journal of Animal Ecology*, 80, 1196–1206.

Lapointe, D.A., Atkinson, C.T. & Samuel, M.D. (2012) Ecology and conservation biology of avian malaria. *Annals of the New York Academy of Sciences*, 1249, 211–226.

Lochmiller, R. & Deerenberg, C. (2000) Trade-offs in evolutionary immunology: Just what is the cost of immunity? *Oikos*, 88, 87–98.

Lochmiller, R.L., Vestey, M.R. & Boren, J.C. (1993) Relationship between protein nutritional-status and immunocompetence in northern bobwhite chicks. *The Auk*, 110, 503–510.

Lombardo, M. (2010). *Parental provisioning rates in relation to flashy head ornaments and fitness in White-Crowned Sparrows* (*Zonotrichia leucophrys oriantha*). Honors Thesis, University of Michigan.

MacDougall-Shackleton, E.A., Derryberry E. P., Foufopoulos, J., Dobson A.P. & Hahn, T.P. (2005). Parasite-mediated heterozygote advantage in an outbred songbird population. *Biology Letters*, 1, 105-107.

Malakar, R., Elkinton, J., Hajek, A. & Burand, J. (1999) Within-host interactions of *Lymantria dispar* (Lepidoptera: lymantriidae) nucleopolyhedrosis virus and *Entomophaga maimaiga* (Zygomycetes: Entomophthorales). *Journal of Invertebrate Pathology*, 73, 91–100.

Martin, T.E. (1987) Food as a limit on breeding birds: A life-history perspective. *Annual Review of Ecology and Systematics*, 18, 453–487.

Martin, L.B., Scheuerlein, A. & Wikelski, M. (2002) Immune activity elevates energy expenditure of house sparrows: a link between direct and indirect costs? *Proceedings of the Royal Society B: Biological Sciences*, 270, 153–8.

Martínez-de la Puente, J., Merino, S., Tomás, G., Moreno, J., Morales, J., Lobato, E., García-Fraile, S. & Belda, E.J. (2010) The blood parasite *Haemoproteus* reduces survival in a wild bird: a medication experiment. *Biology Letters*, 6, 663–665.

Marzal, A., Bensch, S., Reviriego, M., Balbontin, J. & De Lope, F. (2008) Effects of malaria double infection in birds: One plus one is not two. *Journal of Evolutionary Biology*, 21, 979–987.

Marzal, A., De Lope, F., Navarro, C. & Møller, A.P. (2005) Malarial parasites decrease reproductive success: An experimental study in a passerine bird. *Oecologia*, 142, 541–545.

May, R.M. & Anderson, R.M. (1978) Regulation and stability of host-parasite population interactions: II. Destabilizing processes. *Journal of Animal Ecology*, 47, 249-267.

Mayfield, H.F. (1961) Nesting success calculated from exposure. *Wilson Bulletin*, 73, 255-261.

Mayfield, H.F. (1975) Suggestions for calculating nest success. *Wilson Bulletin*, 87, 456-466.

McDonald, M.V. & Greenberg, R. (1991) Nest departure calls in female songbirds. *Condor*, 93, 365-373.

Merila, J. & Andersson, M. (1999) Reproductive effort and success are related to haematozoan infections in blue tits. *Ecoscience*, 6, 421–428.

Merino, S., Moreno, J., Sanz, J.J. & Arriero, E. (2000) Are avian blood parasites pathogenic in the wild? A medication experiment in blue tits (*Parus caeruleus*). *Proceedings of the Royal Society B: Biological Sciences*, 267, 2507–2510.

Millington, O.R., Gibson, V.B., Rush, C.M., Zinselmeyer, B.H., Phillips, R.S., Garside, P. & Brewer, J.M. (2007) Malaria impairs T cell clustering and immune priming despite normal signal 1 from dendritic cells. *PLoS Pathogens*, 3, 1380–1387.

Minchella, D.J. & Scott, M.E. (1991) Parasitism: A cryptic determinant of animal community structure. *Trends in Ecology & Evolution*, 6, 250–254.

Møller, A.P. & Nielsen, J.T. (2007) Malaria and risk of predation: a comparative study of birds. *Ecology*, 88, 871–881.

Monaghan, P. & Nager, R.G. (1997) Why don't birds lay more eggs? *Trends in Ecology and Evolution*, 12, 270–274.

Morton, M. (2002) *The Mountain White-crowned Sparrow: migration and reproduction at high altitude*. *Studies in Avian Biology Number 24*. Cooper Orinthological Society, Camarillo, California.

Mosquera, J. & Adler, F.R. (1998) Evolution of virulence: a unified framework for coinfection and superinfection. *Journal of Theoretical Biology*, 195, 293–313.

Murdock, C.C., Foufopoulos, J. & Simon, C.P. (2013). A transmission model for the ecology of an avian blood parasite in a temperate ecosystem. *PLoS ONE*, 8, 1-14.

Murray, D.L., Cary, J.R. & Keith, L.B. (1997) Interactive effects of sublethal nematodes and nutritional status on snowshoe hare vulnerability to predation. *Journal of Animal Ecology*, 66, 250-264.

Nordling, D., Andersson, M., Zohari, S. & Gustafsson, L. (1998) Reproductive effort reduces specific immune response and parasite resistance. *Proceedings of the Royal Society B: Biological Sciences*, 265, 1291–1298.

Obendorf, D.L. & McColl, K. (1980) Mortality in little penguins (*Eudyptula minor*) along the coast of Victoria, Australia. *Journal of Wildlife Diseases*, 16, 251-260.

Palinauskas, V., Markovets, M.Y., Kosarev, V. V, Efremov, V.D., Sokolov, L. V. & Valkiūnas, G. (2005) Occurrence of avian haematozoa in Ekaterinburg and Irkutsk districts of Russia. *Ekologija*, 4, 8–12.

Pedersen, A.B. & Fenton, A. (2007) Emphasizing the ecology in parasite community ecology. *Trends in Ecology and Evolution*, 22, 133–139.

Pedersen, A.B. & Greives, T.J. (2008) The interaction of parasites and resources cause crashes in a wild mouse population. *Journal of Animal Ecology*, 77, 370-377.

Perrins, C. (1964). Survival of young swifts in reation to brood-size. *Nature*, 201, 1147-1148.

Perrins, C. (1965). Population fluctuations and clutch-size in the great tit, *Parus major L. Journal of Animal Ecology*, 34, 601-647.

Petney, T.N. & Andrews, R.H. (1998) Multiparasite communities in animals and humans: frequency, structure and pathogenic significance. *International Journal for Parasitology*, 28, 377–393.

Price, P.W. (1980) *Evolutionary Biology of Parasites.* Princeton University Press, Princeton, New Jersey.

R Development Core Team. (2013) R: a language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. http://www.R-project.org

Read, A.F. & Taylor, L.H. (2001) The ecology of genetically diverse infections. *Science*, 292, 1099–1102.

Richner, H. (1992) The effect of extra food on fitness in breeding carrion crows. *Ecology*, 73, 330-335.

Rigaud, T. & Haine, E.R. (2005) Conflict between co-occurring parasites as a confounding factor in manipulation studies? *Behavioural Processes*, 68, 259–262.

De Roode, J.C., Read, A.F., Chan, B.H.K. & Mackinnon, M.J. (2003) Rodent malaria parasites suffer from the presence of conspecific clones in three-clone *Plasmodium chabaudi* infections. *Parasitology*, 127, 411–418.

Rotella, J.J., Dinsmore, S.J. & Shaffer, T.L. (2004) Modeling nest-survival data: a comparison of recently developed methods that can be implemented in MARK and SAS. *Animal Biodiversity and Conservation*, 27, 187-205.

Rowe, L., Ludwig, D., & Schluter, D. (1994) Time, condition, and the seasonal decline of avian clutch size. *The American Naturalist*, 143, 698-722.

Sanz, J.J., Arriero, E., Moreno, J. & Merino, S. (2001) Interactions between hemoparasite status and female age in the primary reproductive output of pied flycatchers. *Oecologia*, 126, 339–344.

Searcy, W.A., Peters, S. & Nowicki, S. (2004) Effects of early nutrition on growth rate and adult size in song sparrows *Melospiza melodia*. *Journal of Avian Biology*, 35, 269–279.

Sheldon, B.C. & Verhulst, S. (1996) Ecological immunology: costly parasite defences and trade-offs in evolutionary ecology. *Ecology*, 5347, 317–321.

Sinclair, A.R.E. (1974) The natural regulation of buffalo populations in east Africa. *African Journal of Ecology*, 12, 185-200.

Slagsvold, T. & Lifjeld, J.T. (1988) Ultimate adjustment of clutch size to parental feeding capacity in a passerine bird. *Ecology*, 69, 1918–1922.

Slagsvold, T. & Lifjeld, J.T. (1990) Influence of male and female quality on clutch size in tits (*Parus* Spp.). *Ecology*, 71, 1258–1266.

Sol, D., Jovani, R. & Torres, J. (2003) Parasite mediated mortality and host immune response explain age-related differences in blood parasitism in birds. *Oecologia*, 135, 542–547.

Sternberg, E.D., Lefèvre, T., Rawstern, A.H. & de Roode, J.C. (2011) A virulent parasite can provide protection against a lethal parasitoid. *Infection, Genetics and Evolution*, 11, 399–406.

Stjernman, M., Råberg, L. & Nilsson, J.A. (2004) Survival costs of reproduction in the blue tit (*Parus caeruleus*): a role for blood parasites? *Proceedings of the Royal Society B: Biological Sciences*, 271, 2387–2394.

Swinnerton, K.J., Peirce, M.A., Greenwood, A., Chapman, R.E. & Jones, C.G. (2005) Prevalence of *Leucocytozoon marchouxi* in the endangered Pink Pigeon *Columba mayeri*. *Ibis*, 147, 725–737.

Taylor, L.H., Mackinnon, M.J. & Read, A.F. (1998) Virulence of mixed-clone and single-clone infections of the rodent malaria *Plasmodium chabaudi*. *Evolution*, 52, 583–591.

Thomas, F., Fauchier, J. & Lafferty, K.D. (2002) Conflict of interest between a nematode and a trematode in an amphipod host: test of the "sabotage" hypothesis. *Behavioral Ecology and Sociobiology*, 51, 296–301.

Tomás, G., Merino, S., Martínez, J., Moreno, J. & Sanz, J.J. (2005) Stress protein levels and blood parasite infection in blue tits (*Parus caeruleus*): a medication field experiment. *Annales Zoologici Fennici*, 42, 45–56.

Tomás, G., Merino, S., Moreno, J., Morales, J. & Martínez-de la Puente, J. (2007) Impact of blood parasites on immunoglobulin level and parental effort: a medication field experiment on a wild passerine. *Functional Ecology*, 21, 125–133.

Valkiūnas, G. (2005) *Avian Malaria Parasites and Other Haemosporidia*. CRC Press, Boca Raton, Florida.

Valkiūnas, G., Iezhova, T.A. & Shapoval, A.P. (2003) High prevalence of blood parasites in hawfinch *Coccothraustes coccothraustes*. *Journal of Natural History*, 37, 2647–2652.

Vergara, P., Mougeot, F., Martinez-Padilla, J., Leckie, F. & Redpath, S.M. (2012) The condition dependence of a secondary sexual trait is stronger under high parasite infection level. *Behavioral Ecology*, 23, 502–511.

White, G.C. & Burnham, K.P. (1999) Program MARK: survival estimation from populations of marked animals. *Bird Study*, 46 (supplement), 120-139.

Wiebe, K.L. & Bortolotti, G.R. (1994) Food supply and hatching spans of birds: energy constraints or faculative manipulation. *Ecology*, 75, 813-823.

Wiehn, J. & Korpimaki, E. (1997) Food limitation on brood size: experimental evidence in the Eurasian kestrel. *Ecology*, 78, 2043-2050.

Williams, J.B. (1996) Energetics of avian incubation. *Avian Energetics and Nutritional Ecology* (eds C. Carey), pp. 375-416. Chapman & Hall.

Zuk, M. & Stoehr, A.M. (2002) Immune defense and host life history. *The American Naturalist*, 160, S9–S22.

Appendices

The following appendices contain the complete model sets for all six variables evaluated (Appendix 1 through Appendix 6). Akaike's information criterion for small sample sizes (AIC_c) was used to identify the most parsimonious model and evaluate model support in each model candidate set (Burnham and Anderson 2002). We selected the most parsimonious model as the model with the lowest AIC_c score. Models with ΔAIC_c <2.0 units of the most parsimonious model were considered equally supported. Model Akaike weights (w_i) were calculated for each model and were used to infer the probability of the model being the best model in the candidate model set (Burnham and Anderson 2002). Variable weights were generated by summing model Akaike weights for every model that included the variable. Number of parameters for each model was included (K).

Appendix 1: Summary of model selection results for Daily Nest Survival of White-crowned Sparrows breeding in Colorado. Models are ranked in ascending order by ΔAIC_c. Number of parameters (K), and AIC_c weights (w_i) are given for each model. Variables in models included Female food visitation frequency ($Food_{FEM}$), Male food visitation frequency ($Food_{MALE}$), Number of Female blood parasite coinfections ($Coinfection_{FEM}$), Number of Male blood parasite infections ($Coinfection_{MALE}$), the interaction between food visitation and blood parasite coinfection ($Food_{FEM}$ * $Coinfection_{FEM}$ or $Food_{MALE}$ * $Coinfection_{MALE}$), Site, and Year. S(.) designates a constant daily nest survival model. The lowest AIC_c value was 422.32.

Dependent Variable: DNS	K	ΔAIC_c	w_i
Year + ID_{FEM} + Site + $Food_{FEM}$	11	0.00	0.28
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$	12	1.82	0.11
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Drug_{FEM}$	12	2.02	0.10
Year + ID_{FEM} + Site + $Coinfection_{FEM}$	11	3.20	0.06
Year + ID_{FEM} + Site + $Drug_{FEM}$	11	3.26	0.05
Year + ID_{FEM} + ID_{MALE} + Site	11	3.28	0.05
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	13	3.34	0.05
S(.)	1	3.51	0.05
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$	13	3.82	0.04
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Drug_{FEM}$ + $Food_{FEM}$ * $Drug_{FEM}$	13	4.01	0.04
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$	13	4.04	0.04
Year + ID_{FEM} + Site + $Coinfection_{FEM}$ + $Drug_{FEM}$	12	5.17	0.02
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	14	5.36	0.02
Year + ID_{FEM} + $Food_{FEM}$	8	5.68	0.02
Year + ID_{FEM} + Site + $Coinfection_{FEM}$ + $Coinfection_{MALE}$	13	7.01	0.01
Year + ID_{FEM} + ID_{MALE} + Site + $Drug_{FEM}$ + $Drug_{MALE}$	13	7.11	0.01
Year + ID_{FEM} + ID_{MALE} + $Food_{FEM}$ + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$	15	7.51	0.01
Year + ID_{FEM} + $Drug_{FEM}$	8	7.61	0.01
Year + ID_{FEM} + $Coinfection_{FEM}$	8	7.63	0.01
Year + ID_{FEM} + $Food_{FEM}$ + $Drug_{FEM}$	9	7.65	0.01
Year + ID_{FEM} + $Food_{FEM}$ + $Coinfection_{FEM}$	9	7.68	0.01

$Year + ID_{FEM} + ID_{MALE} + Site + Food_{FEM} + Food_{MALE} + Drug_{FEM} + Drug_{MALE}$	15	8.05	0.00
$Year + ID_{FEM} + Food_{FEM} + Coinfection_{FEM} + Food_{FEM} * Coinfection_{FEM}$	10	9.45	0.00
$Year + ID_{FEM} + Coinfection_{FEM} + Drug_{FEM}$	9	9.59	0.00
$Year + ID_{FEM} + ID_{MALE} + Food_{FEM} + Food_{MALE}$	10	9.62	0.00
$Year + ID_{FEM} + Food_{FEM} + Drug_{FEM} + Food_{FEM} * Drug_{FEM}$	10	9.65	0.00
$Year + ID_{FEM} + Food_{FEM} + Coinfection_{FEM} + Drug_{FEM}$	10	9.66	0.00
$Year + ID_{FEM} + ID_{MALE} + Site + Coinfection_{FEM} + Coinfection_{MALE} + Drug_{FEM} + Drug_{MALE}$	15	10.79	0.00
$Year + ID_{FEM} + ID_{MALE} + Site + Drug_{FEM} + Drug_{MALE}$	10	10.84	0.00
$Year + ID_{FEM} + ID_{MALE} + Coinfection_{FEM} + Coinfection_{MALE}$	10	11.42	0.00
$Year + ID_{FEM} + Site + Food_{FEM} + Coinfection_{FEM} + Drug_{FEM} + Food_{FEM} * Coinfection_{FEM}$	11	11.46	0.00
$Year + ID_{FEM} + ID_{MALE} + Site + Food_{FEM} + Food_{MALE} + Coinfection_{FEM} + Coinfection_{MALE} + Drug_{FEM} + Drug_{MALE}$	17	11.48	0.00
$Year + ID_{FEM} + ID_{MALE} + Coinfection_{FEM} + Coinfection_{MALE} + Drug_{FEM} + Drug_{MALE}$	12	14.52	0.00
$Year + ID_{FEM} + Site + Food_{FEM} + Food_{MALE} + Coinfection_{FEM} + Coinfection_{MALE} + Food_{FEM} * Coinfection_{FEM} + Food_{MALE} * Coinfection_{MALE}$	14	16.98	0.00
$Year + ID_{FEM} + Site + Food_{FEM} + Food_{MALE} + Coinfection_{FEM} + Coinfection_{MALE} + Drug_{FEM} + Drug_{MALE} + Food_{FEM} * Coinfection_{FEM} + Food_{MALE} * Coinfection_{MALE}$	16	20.64	0.00

Appendix 2: Summary of model selection results for probability of a nestling fledging (*Prob. Fledge*) of White-crowned Sparrows in Gothic, Colorado. Models are ranked in ascending order by ΔAIC_c. Number of parameters (K), and AIC_c weights (w_i) are given for each model. Variables in models included Female food visitation frequency ($Food_{FEM}$), Male food visitation frequency ($Food_{MALE}$), Number of Female blood parasite coinfections ($Coinfection_{FEM}$), Number of Male blood parasite coinfection ($Coinfection_{MALE}$), the interaction between food visitation and blood parasite coinfection ($Food_{FEM}$*$Coinfection_{FEM}$ or $Food_{MALE}$ *$Coinfection_{MALE}$), Site, and Year. The lowest AIC_c value was 140.69.

Dependent Variable: Probability of Fledging	K	ΔAIC_c	w_i
Year + ID_{FEM} + $Coinfection_{FEM}$	4	0.00	0.37
Year + ID_{FEM} + $Coinfection_{FEM}$ + $Drug_{FEM}$	5	1.21	0.20
Year + ID_{FEM} + $Coinfection_{FEM}$ + $Food_{FEM}$	5	2.43	0.11
Year + ID_{FEM} + ID_{MALE} + $Coinfection_{FEM}$ + $Coinfection_{MALE}$	6	3.64	0.06
Year + ID_{FEM} + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$	6	3.73	0.06
Year + ID_{FEM} + ID_{MALE} + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$	7	4.60	0.04
Year + ID_{FEM} + ID_{MALE} + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$	6	4.94	0.03
Year + ID_{FEM} + ID_{MALE} + $Food_{FEM}$ + $Drug_{MALE}$	6	4.97	0.03
Year + ID_{FEM} + ID_{FEM} + ID_{MALE} + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$	7	6.21	0.02
Year + ID_{FEM} + ID_{MALE} + $Coinfection_{FEM}$ + $Drug_{FEM}$ + $Drug_{MALE}$	7	6.36	0.02
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	7	6.38	0.02
Year + ID_{FEM} + Site + $Coinfection_{FEM}$	7	6.46	0.01
Year + ID_{FEM} + ID_{MALE} + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	8	7.13	0.01
Year + ID_{FEM} + ID_{MALE} + Site + $Coinfection_{FEM}$ + $Food_{FEM}$ + $Drug_{MALE}$	7	7.62	0.01
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$	8	7.92	0.01
Year + ID_{FEM} + ID_{MALE} + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$ + $Drug_{MALE}$	8	9.11	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{MALE}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	8	10.35	0.00
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$	9	10.80	0.00
Year + ID_{FEM} + ID_{MALE} + 1	4	10.86	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Coinfection_{FEM}$ + $Coinfection_{MALE}$	9	10.98	0.00
Year + ID_{FEM} + $Food_{FEM}$	4	11.19	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$ + $Drug_{MALE}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	9	12.00	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Coinfection_{FEM}$ + $Drug_{MALE}$	9	12.09	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$	10	12.20	0.00
Year + ID_{FEM} + ID_{MALE} + $Coinfection_{MALE}$ + $Drug_{FEM}$	6	12.29	0.00
Year + ID_{FEM} + $Food_{FEM}$ + $Drug_{FEM}$	5	13.55	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Coinfection_{FEM}$ + $Drug_{FEM}$ + $Drug_{MALE}$	10	13.72	0.00
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	10	13.80	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$	10	13.90	0.00
Year + ID_{FEM} + ID_{MALE} + $Coinfection_{MALE}$	7	14.86	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	11	15.07	0.00
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{MALE}$	10	15.08	0.00
Year + ID_{FEM} + ID_{MALE} + $Food_{FEM}$ + $Food_{MALE}$	6	15.62	0.00
Year + ID_{FEM} + ID_{MALE} + $Food_{MALE}$ + $Drug_{FEM}$	6	15.66	0.00
Year + ID_{FEM} + ID_{MALE} + $Drug_{FEM}$ + $Drug_{MALE}$	6	15.70	0.00
Year + ID_{MALE} + $Coinfection_{MALE}$	4	15.77	0.00

Year + ID_{FEM} + $Food_{FEM}$ + $Drug_{FEM}$ + $Food_{FEM}$ * $Drug_{FEM}$	6	15.97	0.00
Year + ID_{MALE} + $Food_{MALE}$	4	16.00	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$ + $Drug_{MALE}$	11	16.87	0.00
Year + ID_{FEM} + ID_{MALE} + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	7	16.95	0.00
Year + ID_{FEM} + Site + $Food_{FEM}$	7	16.97	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$	11	17.11	0.00
Year + ID_{FEM} + ID_{MALE} + $Food_{MALE}$ + $Drug_{FEM}$ + $Food_{MALE}$ * $Drug_{FEM}$	8	17.33	0.00
Year + ID_{FEM} + ID_{MALE} + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Food_{MALE}$ * $Coinfection_{MALE}$	8	17.50	0.00
Year + ID_{FEM} + ID_{MALE} + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	8	17.56	0.00
Year + ID_{MALE} + $Coinfection_{MALE}$ + $Drug_{MALE}$	5	17.60	0.00
Year + ID_{MALE} + $Food_{MALE}$ + $Coinfection_{MALE}$	5	17.68	0.00
Year + ID_{FEM} + ID_{MALE} + $Food_{FEM}$ + $Food_{MALE}$	7	18.09	0.00
Year + ID_{FEM} + ID_{MALE} + $Food_{FEM}$ + $Drug_{FEM}$ + $Drug_{MALE}$	7	18.11	0.00
Year + ID_{MALE} + Site + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$	11	18.17	0.00
Year + ID_{FEM} + ID_{MALE} + $Food_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	7	18.29	0.00
Year + ID_{MALE} + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Food_{MALE}$ * $Coinfection_{MALE}$	6	18.49	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$	12	18.68	0.00
Year + ID_{MALE} + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$	6	19.52	0.00
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Drug_{FEM}$	8	19.71	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$ + $Drug_{MALE}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	12	20.19	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$	12	20.31	0.00
Year + ID_{MALE} + Site + $Food_{MALE}$	7	20.50	0.00
Year + ID_{FEM} + ID_{MALE} + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$ + $Food_{MALE}$ * $Coinfection_{MALE}$	9	20.55	0.00
Year + ID_{MALE} + Site + $Coinfection_{MALE}$	7	20.67	0.00
Year + ID_{MALE} + Site + $Drug_{MALE}$	7	21.14	0.00
Year + ID_{FEM} + ID_{MALE} + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$ + $Food_{MALE}$ * $Coinfection_{MALE}$	7	21.17	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$	10	21.24	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	10	21.69	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{MALE}$ + $Drug_{FEM}$	9	21.91	0.00
Year + ID_{FEM} + Site + $Drug_{FEM}$	7	21.93	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$	9	21.99	0.00
Year + ID_{MALE} + Site + $Food_{MALE}$ + $Coinfection_{MALE}$	8	22.05	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Drug_{FEM}$ + $Drug_{MALE}$	9	22.15	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Coinfection_{MALE}$ + $Drug_{FEM}$	8	22.61	0.00
Year + ID_{MALE} + Site + $Coinfection_{MALE}$ + $Drug_{MALE}$	8	22.83	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	13	23.86	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$ + $Food_{MALE}$ * $Drug_{MALE}$	11	24.21	0.00
Year + ID_{MALE} + Site + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$	9	24.27	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	11	24.38	0.00
Year + ID_{FEM} + ID_{MALE} + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$	11	24.41	0.00
Year + ID_{MALE} + Site + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Food_{MALE}$ * $Coinfection_{MALE}$	9	24.42	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Drug_{FEM}$ + $Drug_{MALE}$	10	24.70	0.00

Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$ + $Drug_{FEM}$	10	24.73	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	10	24.91	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Food_{MALE}$ * $Coinfection_{MALE}$	14	26.00	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$ + $Food_{MALE}$ * $Coinfection_{MALE}$	10	26.64	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$ + $Food_{FEM}$ * $Coinfection_{FEM}$ + $Food_{MALE}$ $Coinfection_{MALE}$	14	27.38	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Drug_{FEM}$ + $Drug_{MALE}$ + $Food_{FEM}$ * $Drug_{FEM}$	11	27.84	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	11	27.86	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$ + $Food_{MALE}$ * $Coinfection_{MALE}$	12	28.09	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$ + $Food_{FEM}$* $Coinfection_{FEM}$ + $Food_{MALE}$ * $Coinfection_{MALE}$	15	29.33	0.00
Year + ID_{FEM} + ID_{MALE} + Site + $Food_{FEM}$ + $Food_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$ + $Food_{FEM}$ * $Drug_{FEM}$ + $Food_{MALE}$ * $Drug_{MALE}$	13	29.77	0.00
Year + ID_{FEM} + Site + $Coinfection_{FEM}$ + $Food_{FEM}$	7	37.06	0.00
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	8	39.65	0.00

Appendix 3: Summary of model selection results for probability of an egg hatching (*Prob. Hatch*) of White-crowned Sparrows. Models are ranked in ascending order by ΔAIC_c. Number of parameters (K), and AIC_c weights (w_i) are given for each model. Variables in models included Female food visitation frequency ($Food_{FEM}$), Number of Female blood parasite coinfection ($Coinfection_{FEM}$), the interaction between food visitation and blood parasite coinfection ($Coinfection_{FEM} * Food_{FEM}$), Site, and Year. The lowest AIC_c value was 226.12.

Dependent Variable: Probability of Hatching	K	ΔAIC_c	w_i
$Year + ID_{FEM} + Site + Food_{FEM}$	7	0.00	0.33
$Year + ID_{FEM} + Food_{FEM}$	4	1.50	0.16
$Year + ID_{FEM} + Site + Food_{FEM} + Coinfection_{FEM}$	8	2.03	0.12
$Year + ID_{FEM} + Drug_{FEM}$	8	2.36	0.10
$Year + ID_{FEM} + Food_{FEM} + Drug_{FEM}$	5	3.71	0.05
$Year + ID_{FEM} + Food_{FEM} + Coinfection_{FEM}$	5	3.73	0.05
$Year + ID_{FEM} + Site + Food_{FEM} + Coinfection_{FEM} + Food_{FEM} * Coinfection_{FEM}$	9	4.27	0.04
$Year + ID_{FEM} + Site + Food_{FEM} + Coinfection_{FEM} + Drug_{FEM}$	9	4.49	0.04
$Year + ID_{FEM} + Site + Food_{FEM} + Drug_{FEM} + Food_{FEM} * Drug_{FEM}$	9	4.82	0.03
$Year + ID_{FEM} + Food_{FEM} + Drug_{FEM} + Food_{FEM} * Drug_{FEM}$	6	5.36	0.02
$Year + ID_{FEM} + Site + Food_{FEM} + Coinfection_{FEM} + Food_{FEM} * Coinfection_{FEM}$	6	5.80	0.02
$Year + ID_{FEM} + Food_{FEM} + Coinfection_{FEM} + Drug_{FEM}$	6	6.00	0.02
$Year + ID_{FEM} + Site + Food_{FEM} + Coinfection_{FEM} + Drug_{FEM} + Food_{FEM} * Coinfection_{FEM}$	10	6.78	0.01
$Year + ID_{FEM} + Food_{FEM} + Coinfection_{FEM} + Drug_{FEM} + Food_{FEM} * Coinfection_{FEM}$	7	8.14	0.01
$Year + ID_{FEM} + 1$	3	12.83	0.00
$Year + ID_{FEM} + Drug_{FEM}$	4	14.90	0.00
$Year + ID_{FEM} + Coinfection_{FEM}$	4	15.00	0.00
$Year + ID_{FEM} + Site + Coinfection_{FEM}$	7	15.95	0.00
$Year + ID_{FEM} + Site + Drug_{FEM}$	7	16.03	0.00
$Year + ID_{FEM} + Coinfection_{FEM} + Drug_{FEM}$	5	17.13	0.00
$Year + ID_{FEM} + Site + Coinfection_{FEM} + Drug_{FEM}$	8	18.26	0.00

Appendix 4: Summary of model selection results for clutch size (*clutch)* of White-crowned Sparrows. Models are ranked in ascending order by ΔAIC_c. Number of parameters (K), and AIC_c weights (w_i) are given for each model. Variables in models included Female food visitation frequency ($Food_{FEM}$), Number of Female blood parasite coinfections ($Coinfection_{FEM}$), the interaction between food visitation and blood parasite coinfection ($Food_{FEM}$*$Coinfection_{FEM}$), site, and year. The response variable *clutch* was the number of eggs laid in each nest. The lowest AIC_c value was 371.95.

Dependent Variable: Clutch Size	K	ΔAIC_c	w_i
Year + ID_{FEM} + 1	3	0.00	0.38
Year + ID_{FEM} + $Drug_{FEM}$	4	1.87	0.15
Year + ID_{FEM} + $Food_{FEM}$	4	1.99	0.14
Year + ID_{FEM} + $Coinfection_{FEM}$	4	2.21	0.13
Year + ID_{FEM} + $Food_{FEM}$ + $Drug_{FEM}$	5	3.94	0.05
Year + ID_{FEM} + $Coinfection_{FEM}$ + $Drug_{FEM}$	5	4.10	0.05
Year + ID_{FEM} + $Food_{FEM}$ + $Coinfection_{FEM}$	5	4.26	0.05
Year + ID_{FEM} + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$	6	6.25	0.02
Year + ID_{FEM} + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	6	6.53	0.01
Year + ID_{FEM} + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	7	8.54	0.01
Year + ID_{FEM} + Site + $Drug_{FEM}$	7	8.57	0.01
Year + ID_{FEM} + Site + $Food_{FEM}$	7	8.61	0.01
Year + ID_{FEM} + Site + $Coinfection_{FEM}$	7	8.91	0.00
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Drug_{FEM}$	8	10.72	0.00
Year + ID_{FEM} + Site + $Coinfection_{FEM}$ + $Drug_{FEM}$	8	10.99	0.00
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$	8	11.06	0.00
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$	9	13.24	0.00
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	9	13.51	0.00
Year + ID_{FEM} + Site + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$ + $Food_{FEM}$ * $Coinfection_{FEM}$	10	15.71	0.00

Appendix 5: Summary of model selection results for fledging tarsus length of White-crowned Sparrows breeding in Colorado. Models are ranked in ascending order by ΔAIC_c. Number of parameters (K), and AIC_c weights (w_i) are given for each model. Variables in models included Female food visitation frequency ($Food_{FEM}$), Number of Female blood parasite coinfections ($Coinfection_{FEM}$), Number of fledglings per nest (Fledge), Site, Female ID (ID_{FEM}), Year. The lowest AIC_c value was 256.11.

Dependent Variable: Young Tarsus Length *(mm)*	K	ΔAIC_c	w_i
$Year + ID_{MALE} + Site + Fledge + Coinfection_{MALE}$	9	0.00	0.11
$Year + ID_{FEM} + ID_{MALE} + Fledge + 1$	5	0.17	0.10
$Year + ID_{FEM} + Site + Fledge + Drug_{FEM}$	9	0.58	0.08
$Year + ID_{FEM} + Site + Fledge + Coinfection_{FEM}$	9	0.62	0.08
$Year + ID_{MALE} + Site + Fledge + Food_{MALE}$	9	1.23	0.06
$Year + ID_{FEM} + Fledge + Drug_{FEM}$	6	1.25	0.06
$Year + ID_{MALE} + Fledge + Coinfection_{MALE}$	6	1.73	0.05
$Year + ID_{MALE} + Site + Fledge + Food_{MALE} + Coinfection_{MALE}$	10	1.87	0.04
$Year + ID_{MALE} + Site + Fledge + Drug_{MALE}$	9	1.94	0.04
$Year + ID_{FEM} + Site + Fledge + Food_{FEM}$	9	2.04	0.04
$Year + ID_{FEM} + Fledge + Food_{FEM}$	6	2.06	0.04
$Year + ID_{MALE} + Fledge + Food_{MALE}$	6	2.07	0.04
$Year + ID_{FEM} + Site + Fledge + Coinfection_{FEM} + Drug_{FEM}$	10	2.33	0.03
$Year + ID_{MALE} + Fledge + Food_{MALE} + Coinfection_{MALE}$	7	2.59	0.03
$Year + ID_{MALE} + Fledge + Drug_{MALE}$	6	2.82	0.03
$Year + ID_{FEM} + Fledge + Food_{FEM} + Drug_{FEM}$	7	2.92	0.03
$Year + ID_{FEM} + Fledge + Coinfection_{FEM}$	6	3.16	0.02
$Year + ID_{MALE} + Site + Fledge + Coinfection_{MALE} + Drug_{MALE}$	10	4.17	0.01
$Year + ID_{MALE} + Fledge + Coinfection_{MALE} + Drug_{MALE}$	7	4.25	0.01
$Year + ID_{FEM} + Fledge + Coinfection_{FEM} + Drug_{FEM}$	7	4.39	0.01
$Year + ID_{FEM} + Site + Fledge + Food_{FEM} + Coinfection_{FEM}$	10	4.69	0.01
$Year + ID_{FEM} + Site + Fledge + Food_{FEM} + Drug_{FEM}$	10	4.69	0.01
$Year + ID_{MALE} + Site + Fledge + Food_{MALE} + Drug_{MALE}$	10	5.12	0.01
$Year + ID_{MALE} + Fledge + Food_{MALE} + Drug_{MALE}$	7	5.18	0.01
$Year + ID_{FEM} + Fledge + Food_{FEM} + Coinfection_{FEM}$	7	5.25	0.01
$Year + ID_{MALE} + Fledge + Food_{MALE} + Coinfection_{MALE} + Drug_{MALE}$	8	5.77	0.01
$Year + ID_{MALE} + Site + Fledge + Food_{MALE} + Coinfection_{MALE} + Drug_{MALE}$	11	6.33	0.00
$Year + ID_{FEM} + Fledge + Food_{FEM} + Coinfection_{FEM} + Drug_{FEM}$	8	6.35	0.00
$Year + ID_{FEM} + Site + Fledge + Coinfection_{FEM} + Coinfection_{MALE}$	11	6.82	0.00
$Year + ID_{FEM} + Site + Fledge + Coinfection_{FEM} + Drug_{FEM}$	11	6.88	0.00
$Year + ID_{FEM} + ID_{MALE} + Fledge + Drug_{FEM} + Drug_{MALE}$	8	7.93	0.00
$Year + ID_{FEM} + Fledge + Coinfection_{FEM} + Coinfection_{MALE}$	8	8.49	0.00
$Year + ID_{FEM} + ID_{MALE} + Site + Fledge + Drug_{FEM} + Drug_{MALE}$	11	8.93	0.00
$Year + ID_{FEM} + ID_{MALE} + Site + Fledge + Food_{MALE} + Coinfection_{FEM} + Coinfection_{MALE}$	12	8.97	0.00
$Year + ID_{FEM} + ID_{MALE} + Fledge + Food_{MALE} + Coinfection_{FEM} + Coinfection_{MALE}$	9	9.93	0.00
$Year + ID_{FEM} + ID_{MALE} + Fledge + Food_{MALE} + Drug_{FEM} + Drug_{MALE}$	9	11.31	0.00
$Year + ID_{FEM} + ID_{MALE} + Site + Fledge + Food_{MALE} + Drug_{FEM} + Drug_{MALE}$	12	13.44	0.00
$Year + ID_{FEM} + ID_{MALE} + Fledge + Coinfection_{FEM} + Coinfection_{MALE} + Drug_{FEM} + Drug_{MALE}$	10	14.19	0.00

Year + ID_{FEM} + ID_{MALE} + Site + Fledge + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	13	14.91	0.00
Year + ID_{FEM} + ID_{MALE} + Fledge + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	11	17.53	0.00
Year + ID_{FEM} + ID_{MALE} + Site + Fledge + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	14	19.33	0.00

Appendix 6: Summary of model selection results for fledging masses of White-crowned Sparrows breeding in Colorado. Models are ranked in ascending order by ΔAIC_c. Models are ranked in ascending order by ΔAIC_c. Number of parameters (K), and AIC_c weights (w_i) are given for each model. Variables in models included Female food visitation frequency ($Food_{FEM}$), Number of Female blood parasite coinfection ($Coinfection_{FEM}$), number of fledglings per nest (Fledge), Site, Female ID (ID_{FEM}), Year. The lowest AIC_c value was 246.53.

Dependent Variable: Young Mass *(g)*	K	ΔAIC_c	w_i
Year + ID_{FEM} + ID_{MALE} + Fledge + 1	5	0.00	0.19
Year + ID_{MALE} + Fledge + $Food_{MALE}$	6	1.40	0.09
Year + ID_{FEM} + Fledge + $Drug_{FEM}$	6	1.52	0.09
Year + ID_{FEM} + Fledge + $Coinfection_{FEM}$ + $Drug_{FEM}$	7	1.65	0.08
Year + ID_{FEM} + Fledge + $Coinfection_{FEM}$	6	1.70	0.08
Year + ID_{MALE} + Fledge + $Drug_{MALE}$	6	1.77	0.08
Year + ID_{FEM} + Fledge + $Food_{FEM}$	6	1.78	0.08
Year + ID_{MALE} + Fledge + $Food_{MALE}$ + $Drug_{MALE}$	7	2.91	0.04
Year + ID_{MALE} + Fledge + $Coinfection_{MALE}$	6	2.92	0.04
Year + ID_{FEM} + Fledge + $Food_{FEM}$ + $Drug_{FEM}$	7	3.26	0.04
Year + ID_{FEM} + Fledge + $Food_{FEM}$ + $Coinfection_{FEM}$ + $Drug_{FEM}$	8	3.52	0.03
Year + ID_{FEM} + Fledge + $Food_{FEM}$ + $Coinfection_{FEM}$	7	3.79	0.03
Year + ID_{MALE} + Fledge + $Food_{MALE}$ + $Coinfection_{MALE}$	7	4.65	0.02
Year + ID_{MALE} + Fledge + $Coinfection_{MALE}$+ $Drug_{MALE}$	7	4.72	0.02
Year + ID_{MALE} + Site + Fledge + $Drug_{MALE}$	9	4.76	0.02
Year + ID_{FEM} + Site + Fledge + $Drug_{FEM}$	9	6.12	0.01
Year + ID_{FEM} + Site + Fledge + $Food_{FEM}$	9	6.37	0.00
Year + ID_{FEM} + Site + Fledge + $Coinfection_{FEM}$	9	6.38	0.00
Year + ID_{MALE} + Fledge + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$	8	6.38	0.00
Year + ID_{FEM} + ID_{MALE} + Fledge + $Drug_{FEM}$ + $Drug_{MALE}$	8	6.77	0.00
Year + ID_{MALE} + Site + Fledge + $Food_{MALE}$	9	6.98	0.00
Year + ID_{MALE} + Site + Fledge + $Coinfection_{MALE}$	9	7.38	0.00
Year + ID_{FEM} + Site + Fledge + $Coinfection_{FEM}$ + $Drug_{FEM}$	10	7.56	0.00
Year + ID_{MALE} + Site + Fledge + $Food_{MALE}$ + $Drug_{MALE}$	10	7.64	0.00
Year + ID_{MALE} + Site + Fledge + $Coinfection_{MALE}$+ $Drug_{MALE}$	10	8.17	0.00
Year + ID_{FEM} + Fledge + $Coinfection_{FEM}$ + $Coinfection_{MALE}$	8	8.45	0.00
Year + ID_{FEM} + Site + Fledge + $Food_{FEM}$ + $Drug_{FEM}$	10	8.69	0.00
Year + ID_{FEM} + ID_{MALE} + Fledge + $Food_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	9	9.34	0.00
Year + ID_{FEM} + Site + Fledge + $Food_{FEM}$ + $Coinfection_{FEM}$	10	9.44	0.00
Year + ID_{FEM} + ID_{MALE} + Fledge + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$	9	10.39	0.00
Year + ID_{FEM} + Site + Fledge + $Coinfection_{FEM}$ + $Drug_{FEM}$	11	10.55	0.00
Year + ID_{MALE} + Site + Fledge + $Food_{MALE}$ + $Coinfection_{MALE}$	10	11.08	0.00
Year + ID_{FEM} + ID_{MALE} + Fledge + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	10	11.19	0.00
Year + ID_{FEM} + ID_{MALE} + Site + Fledge + $Drug_{FEM}$ + $Drug_{MALE}$	11	11.43	0.00
Year + ID_{MALE} + Site + Fledge + $Food_{MALE}$ + $Coinfection_{MALE}$ + $Drug_{MALE}$	11	11.87	0.00
Year + ID_{FEM} + ID_{MALE} + Fledge + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	11	14.87	0.00

Year + ID_{FEM} + Site + Fledge + $Coinfection_{FEM}$ + $Coinfection_{MALE}$	11	15.03	0.00
Year + ID_{FEM} + ID_{MALE} + Site + Fledge + $Food_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	12	15.83	0.00
Year + ID_{FEM} + ID_{MALE} + Site + Fledge + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	13	18.19	0.00
Year + ID_{FEM} + ID_{MALE} + Site + Fledge + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$	12	19.35	0.00
Year + ID_{FEM} + ID_{MALE} + Site + Fledge + $Food_{MALE}$ + $Coinfection_{FEM}$ + $Coinfection_{MALE}$ + $Drug_{FEM}$ + $Drug_{MALE}$	14	24.09	0.00