

**UNIVERSITY OF MINNESOTA**

This is to certify that I have examined this copy of a master's thesis by  
**Courtney Linda Amundson**  
and have found that it is complete and satisfactory in all respects, and that  
any and all revisions required by the final examining committee have been  
made.

**Dr. Todd William Arnold**,

Faculty Advisor

---

Signature of Faculty Advisor

---

Date

GRADUATE SCHOOL

**The Impacts of Helminthic Parasites on Survival of American  
Coot (*Fulica americana*) Chicks**

A THESIS SUBMITTED TO THE FACULTY OF THE GRADUATE  
SCHOOL OF THE UNIVERSITY OF MINNESOTA

BY

**Courtney Linda Amundson**

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE  
DEGREE OF MASTER OF SCIENCE

January 2007

## ACKNOWLEDGEMENTS

First and foremost I would like to thank Dr. Todd Arnold for taking a chance on a greenhorn that came into his office with several ideas and not a single clue. I'd also like to thank my committee members Dr. Doug Johnson and Dr. Susan Weller for their advice and help with experimental design and the preparation of this manuscript. I would especially like to thank Doug Johnson for help with model selection.

I'd like to thank Delta Waterfowl Foundation, the Dayton-Wilkie Fellowship of the Bell Museum of Natural History and the Chapman Fund of the American Museum of Natural History for field funding, equipment use and stipend support.

I could not have completed my field seasons without the help of the Delta Waterfowl Staff: Dick McDonald, Wanda Gorsuch, and especially Frank Rohwer and Liz Loos thank you for all your help and direction. Fellow graduate students Dan Coulton, Letty Reichart, Travis Quirk and Justin Pitt thank you for making those two summers the best of my life. To my assistants Emily Widi, Kevin Wlock and Andy Olivarez thank you for putting up with my antics, incessant cursing and incompetence...you were fantastic. Thanks to all the people who helped me collect coots: Travis Quirk, Letty Reichart, Dan Coulton, Justin Pitt, Justin Thayer, Kevin Wlock, Dustin Cassidy, Lucas Oligschlager, Phoebe Prather, Curt Francis, Brian Sauer, Ryan (JP's assistant), and especially Talon, Scout and Haylie for doing all the work.

I could not have succeeded in graduate school without the support of my family and friends. I would like to thank my mother, Sandra Ikuenobe, for being my number one cheerleader and my grandparents, Norman Bloom and Lee McNally, for a lifetime of support and nurturing. I'd also like to thank Sadie Fitzgerald, Dawn Lubka and Zack Gontard for their friendship, love, and laughter over the years. Cheers to one day finally finding our happy endings. I'd also like to thank Anthony Pagano for his advice and help with this (and many other) documents. A final thanks to all those who helped me that I forgot to mention, I hope you never read this.

**This thesis is dedicated to the memory of**

**Norman E. Bloom**

**April 4, 1927 – June 14, 1995**

## ABSTRACT

Parasitic infections can lead to decreased body condition, reduced nesting success, and lowered mating success in birds, but little research has been done on the effect of parasites on offspring survival. In 2004 and 2005, I examined the effect that helminthic parasites have on offspring survival in American Coots (*Fulica americana*) by experimentally dosing day-old chicks with the anthelmintic drug fenbendazole (treatment) or sterile water (controls) and measuring their survival to 40 days of age. In 2005, I overlaid the experimental treatment of chicks with an independent experiment providing medicated (anthelmintic) or plain (control) feed to incubating adults to determine if chick survival was further influenced by parasite burdens among parents. I marked hatching chicks using colored nape tags, reobserved them at 10-day intervals from 0 to 50 days of age, and then estimated their survival to 40 days of age using open-population mark-recapture analysis. In both years, offspring survival was a function of anthelmintic treatment. In 2004, chicks receiving fenbendazole had 49.9% (SE = 5.1%) survival to 40 days of age, versus 40.3% (SE = 7.1%) survival for untreated chicks ( $z = 1.84$ ,  $P = 0.065$ ). In 2005, offspring survival to 40 days post-hatch was an additive function of both offspring and parental treatment; cumulative 40-day survival was 54.5% (SE = 7.4) when both parents and chicks were treated, 47.1% (SE = 7.8%) when just chicks were treated, 45.8% (SE = 7.7%) when just adults were treated, and 37.5% (SE = 9.1%) when neither were treated with fenbendazole (chick treatment effect:  $z = 1.80$ ,  $P = 0.07$ ; parent treatment effect:  $z = 1.67$ ,  $P = 0.09$ ). My study provides compelling evidence that helminthic parasites lowered reproductive success in American Coots around Minnedosa, Manitoba.

## TABLE OF CONTENTS

ACKNOWLEDGEMENTS.....	i
DEDICATION.....	ii
ABSTRACT.....	iii
LIST OF TABLES.....	v
LIST OF FIGURES.....	vii
THESIS: The Impacts of Helminthic Parasites on Survival of American Coot ( <i>Fulica americana</i> ) Chicks.....	1
INTRODUCTION.....	1
STUDY AREA AND STUDY SPECIES .....	4
METHODS.....	5
STATISTICAL ANALYSES.....	10
RESULTS.....	16
DISCUSSION.....	27
LITERATURE CITED.....	32

## LIST OF TABLES

<p><b>Table 1.</b> Notation and explanations of all considered Cormack-Jolly-Seber (CJS) models in Program MARK (White1995). .....</p>	13
<p><b>Table 2.</b> Effects of fenbendazole treatment on parasite burdens in 17 adult and 38 immature coots. Means (SE) of adult and immature worms are reported as well as F-statistics, degrees of freedom, and P values from Type III sums of squares. ....</p>	17
<p><b>Table 3.</b> Models of coot chick survival to 40 days of age in relation to fenbendazole treatment and chick age, southwestern Manitoba, Canada, 2004. Models are ranked by differences in Quasi-AIC (QAIC<sub>c</sub>). ....</p>	21
<p><b>Table 4.</b> Number of parameters (k), parameter estimates (Est.), standard errors (SE), and lower (LCI) and upper (UCI) 95% confidence limits of apparent survival (<math>\Phi</math>) and resighting rates (<math>\rho</math>) for American Coot chicks in Minnedosa, Manitoba, Canada in 2004 during each resighting interval (0-10 days = <math>\Phi_1</math>, 11-20 days = <math>\Phi_2</math>, 21-30 days = <math>\Phi_3</math>, and 31-40 days post-hatch = <math>\Phi_4</math>). Cumulative survival<sup>a</sup> of each treatment group (<math>\Phi_{T,C}</math>) is also reported, along with its standard error (SE)<sup>b</sup>. ....</p>	23

**Table 5.** Models of coot chick survival to 40 days of age in relation to fenbendazole treatment and chick age, southwestern Manitoba, Canada, 2005. Models are ranked by differences in Quasi-AIC (QAIC<sub>c</sub>). ..... 25

**Table 6.** Number of parameters (k), parameter estimates (Est.), standard errors (SE), and lower (LCI) and upper (UCI) 95% confidence limits of apparent survival ( $\Phi$ ) and resighting rates ( $\rho$ ) for American Coot chicks in Minnedosa, Manitoba, Canada in 2005 during each resighting interval (0-10 days =  $\Phi_1$ , 11-20 days =  $\Phi_2$ , 21-30 days =  $\Phi_3$ , and 31-40 days post-hatch =  $\Phi_4$ ). Cumulative survival<sup>a</sup> of each treatment group ( $\Phi_{\text{Treatment group}}$ ) is also reported, along with its standard error (SE)<sup>b</sup>. CC = parents control, chicks control; CT = parents control, chicks treated; TT = both parents and chicks treated; and TC = parents treated, chicks control. Survival rates in the second and third intervals ( $\Phi_2$  and  $\Phi_3$ ) were similar and therefore combined in the analysis and subsequent survival estimation. Resighting rate was constant ( $\rho$ ). ..... 26

**LIST OF FIGURES**

**Figure 1.** Mean body condition indices ( $\pm$  SE) of 38 (24 treated, 14 control) collected fledglings in 2005. Differences are nearly significant to a p-value of 0.052. .... 19

**Figure 2.** Effect of fenbendazole and level of brood treatment (all, none, or half of brood members treated; see Methods) on 40-d cumulative survival of coot chicks in 2004 (top) and 2005 (bottom) plus > 95% CI. .... 22

## **Introduction**

Parasites have the potential to reduce host fitness by exploiting limited resources that could otherwise be used for growth, survival, or reproduction (Møller and Erritzoe 2002). In birds, parasites have been shown to influence predation risk, body condition, mate choice, and survival of infected individuals (Hudson et al. 1992, Møller et al. 1999, Navarro et al. 2004).

Helminths are primarily intestinal parasites consisting of trematodes (flukes), cestodes (tapeworms), nematodes (roundworms) and acanthocephalans (thorny-headed worms) (Clayton and Moore 1997). Aquatic birds acquire helminthic parasites by primarily ingesting infected invertebrates, especially snails (Cole and Friend 1999). They may also ingest free-floating larval parasites incidentally when feeding or drinking. Reduced food intake is the primary cause of debilitation due to infection by helminthic parasites, which can lead to decreased body condition and suppression of the host's natural defenses against parasites (Chowdhury and Tada 1994). External factors such as severe weather, territorial competition, wounds, food scarcity, or illness can exacerbate parasitic infections and lead to mortality (Davis et al. 1971).

American Coots are known to harbor a wide variety of helminthic parasites, including at least four species of trematodes, three cestodes, two nematodes, and two acanthocephalans (Roudabush 1942, Kinsella 1973, Eley 1976, McKindsey et al. 1994). Infection rates and burdens are highly variable, but there is evidence that immature coots have higher helminthic infection rates than adults (McKindsey et al. 1994). As the breeding season progresses, juveniles stay closer to their nests where infection rates of snails (an important component of juvenile diets) are higher than in open water where

adults typically congregate (McKindsey et al. 1994). Juvenile coots may also be more susceptible to parasites than adults; McKindsey et al. (1994) showed that exposure to a common trematode (*Cyclocoelum mutabile*) resulted in higher infection rates in juveniles than adults.

Research in avian parasitology historically focused on the presence and species composition of parasites (LaPage 1962, Shaw 1978, Kocan et al. 1979), but recent experiments have begun to focus on the fitness consequences of helminthic parasite burdens to breeding birds (Navarro et al. 2004). Hanssen et al. (2003) examined the effects of anthelmintics (broad-spectrum drugs that kill many species of helminths) on body condition, nesting success, and return rates of adult female Common Eiders (*Somateria mollissima*) in northern Norway over a two-year period. Body condition did not improve after treatment, but anthelmintics improved return rates (a proxy for survival) from 18 to 69% among unsuccessful breeders. Newborn and Foster (2002) treated wild Red Grouse (*Lagopus lagopus scoticus*) with anthelmintic grit and found that treated adults had 34% fewer worms than did grouse receiving plain grit. Medicated grit did not significantly affect clutch size, hatching success, or adult survival, but did result in higher offspring survival, presumably because treated adults were in better condition, which allowed them to provide better care to their offspring (Newborn and Foster 2002). Righi and Gauthier (2002) found that semi-captive Greater Snow Goose (*Chen caerulescens atlantica*) goslings treated with an anthelmintic had similar growth rates as controls, but untreated goslings were observed feeding significantly more often, suggesting an effect of helminths on host foraging behavior. A study of the impacts of parasites and body condition on juvenile survival in Ross' Geese (*Chen rossii*) found that

anthelmintics increased survival of all goslings in 1997 and increased survival of large plump goslings in 1998 (Slattery and Alisauskas 2002: Fig. 3). Anthelmintics are widely used in poultry production, and a study of free-range chickens found that chicks treated with an anthelmintic had significantly higher weight gain after 10 weeks than did control chicks (Skallerup et al. 2005). But not all studies have observed a positive effect of anthelmintics; newly hatched Eurasian Oystercatcher (*Haematopus ostralegus*) chicks treated with a combination of levamisole, oxyelozanide, and ivermectin had lower survival than controls, possibly due to an immunosuppressive effect of the anthelmintics (Van Oers et al. 2002). Based on the collective results of these studies (notwithstanding Van Oers et al. 2002), there seems to be compelling evidence that reducing parasite burdens can have a beneficial effect on breeding success, either by improving the condition of brood-rearing parents or by improving the fitness of their chicks.

I investigated the effects of parasites on reproduction in American Coots by experimentally providing anthelmintic drugs (active ingredient fenbendazole) to newly hatched chicks (2004-2005) and incubating adults (2005) and documenting the subsequent survival of chicks to 40 days of age. I predicted that breeding adults receiving fenbendazole would have lower parasite burdens, leading to improved body condition, thereby enhancing their ability to care for dependent offspring, and thus improving the survival of their offspring relative to untreated controls. I further predicted that chicks receiving fenbendazole would have lower parasite burdens, be better able to compete for limited food resources, and thereby have higher survival than untreated control chicks.

## **Study Area**

I conducted my research at the Delta Waterfowl Research Station, located approximately 9 km southwest of Minnedosa, Manitoba, Canada (50°10'N, 99°47'W) during the 2004-2005 breeding seasons. Minnedosa has a high density of 0.1 to 2.0 ha semipermanent and permanent wetlands that provide ideal nesting habitat for coots in all but extreme drought years (Kiel 1955, Arnold 1990, Reed 2000). Importantly, the wetlands have narrow fringes of emergent vegetation that can be easily searched for nests and large central expanses of open water that facilitate brood observations. My study area included approximately 16 different wetlands in 2004 and 31 wetlands in 2005. A detailed description of the Minnedosa area can be found in Stoudt (1982).

## **Study Species**

American Coots are highly territorial and remain on the same portion of the same wetland throughout nesting and brood rearing (Arnold 1990), which greatly facilitates monitoring of individual pairs and their offspring. They lay large clutches of 8-12 energy-dense eggs, and both parents assist with incubation and brood rearing (Alisauskas and Arnold 1994). Clutches hatch asynchronously, typically over 4-10 days, and the semi-precocial chicks are able to leave the nest within hours of hatching, but they are critically dependent on their parents for food during the first few weeks of life (Desrochers and Ankney 1986, Driver 1988). The diet of young coots consists primarily of aquatic invertebrates for the first 2-3 weeks of life, after which plant materials become increasingly important (Driver 1988). Young coot chicks lack the motor skills needed to capture aquatic invertebrates on their own, so they accompany their foraging parents who

feed them on aquatic invertebrates and submerged plant materials (Ryan and Dinsmore 1979, Desrochers and Ankney 1986). When broods are large or food supplies are scarce, parents may resort to preferential feeding of older offspring, which can lead to starvation among younger, less-competitive offspring (Lyon et al. 1994). Coots usually hatch more chicks than they can successfully rear, so virtually all broods experience at least some chick mortality (Lyon et al. 1994). Hence, if parasite burdens reduce the foraging ability of parents or the ability of chicks to compete for parental care, even if only modestly, it could have a pronounced effect on chick survival.

## **Methods**

### *Experimental Design*

I conducted systematic overwater nest searches of the emergent wetland vegetation on roadside wetlands with basins that were entirely visible from the road beginning approximately 20 May 2004 and 5 May 2005 (the 2004 nesting season was delayed by two snowstorms in early May), and continuing weekly until the end of June. Most nests were found during egg laying, and initiation dates were estimated by back dating, assuming 1 egg was laid per day (Arnold 1994). Eggs were floated to determine incubation stage and anticipated date of hatch (Hays and LeCroy 1971) for nests first located after clutch completion. Nests were rechecked at least once per week to determine if they were still active.

I used Panacur (active ingredient fenbendazole) to treat helminth infestations in adult and hatchling coots. Fenbendazole has several potential advantages over alternative anthelmintics (Newborn and Foster 2002). First, it is a safe drug—birds given 100 times

the recommended dosage in clinical trials have displayed no adverse symptoms (Hudson 1992). It is the most commonly used anthelmintic in zoo animals and is generally free of side effects when given to young, sick, or debilitated animals (Adams 2001). Second, it is a versatile, broad-spectrum anthelmintic that is effective against eggs, adults, and larval stages of several species of helminths (Adams 2001), although a full dose of 50mg/kg is needed to see ovicidal and larvicidal effects. It has the greatest chemotherapeutic effect when administered during the intestinal (1<sup>st</sup> week) or migratory (2<sup>nd</sup>-3<sup>rd</sup> weeks) phases of parasite infestation, which corresponds to when I treated chicks. Third, it is palatable and can be administered in the diet with no associated loss of appetite (Adams 2001). Finally, even low concentrations are effective, as partial doses will cause at least some damage to gastrointestinal worms (Hudson 1992). However, there have been a few reports of adverse effects when fenbendazole was administered during egg laying or molt (Howard et al. 1999). The timing of my treatment therefore recognized both of these concerns: fenbendazole was available to adults only after egg laying was complete, and chicks were treated 2 weeks before they began developing adult plumage.

In 2005, nests were checked daily beginning the 10<sup>th</sup> day after nest initiation and continuing until laying was complete. Clutches were deemed complete if they had < 9 eggs on the 10<sup>th</sup> day, or as soon as an egg was not laid for 2 consecutive days. The first nest to finish laying was randomly assigned as a parental treatment or control group and all subsequent wetlands were alternated as treatments or controls. Parental treatment was assigned by wetland rather than by nest because I could not be certain that nesting adults from the same wetland would not utilize feeding platforms in an adjacent pair's territory (Arnold 1994), hence I wanted all pairs on each wetland to receive the same treatment.

After clutch completion, a 60x60 cm floating platform was placed approximately 10-15 m from each nest. Platforms were supplied with 2 kg of cracked corn that was either treated (coated with 60 mL vegetable oil mixed with fenbendazole powder) or untreated (coated with 60 mL vegetable oil only). The treated corn was dosed at 5.7g/kg Panacur (5% active ingredient, fenbendazole), which would provide a therapeutic dose of 50 mg/kg of fenbendazole if each coot ate approximately 200 g of corn per day. Even if treated coots ate all the medicated corn in a single day, the highest potential dose rate (250 mg/kg) was well within the safety range for fenbendazole (Hudson 1992). Feeding platforms were removed after the first chick hatched to prevent parents from feeding chicks food from the platforms. Mean availability of feeding platforms to adults was 14 days (range: 8-20 days,  $n = 55$ ). Coots were observed feeding from the platforms on several occasions (see also Arnold 1994), but few platforms had been emptied before food replacement occurred on day 7.

In both years, the first nest to hatch in each wetland was randomly assigned to 1 of 4 treatment groups: 1) full brood treated (treatment, T), 2) every other chick treated starting with the first chick as a treatment (alternating,  $A_t$ ) or 3) the first chick as a control (alternating,  $A_c$ ), or 4) no chicks treated (control, C). Alternating treatment groups were used to determine if there was a greater advantage to being treated if half of your siblings were not, or a greater disadvantage to not receiving treatment if half of your siblings did (i.e., is condition relative to your broodmates more important than absolute condition?). Subsequent nests on each pond were assigned to the next treatment group in a fixed rotation (T,  $A_t$ ,  $A_c$ , C). Experimental coot nests were visited daily throughout the estimated hatching period and any pipped eggs were collected and individually marked

with their nest and egg numbers. Eggs were placed in an artificial incubator and allowed to hatch in captivity. Newly hatched chicks were weighed ( $\pm 0.1$  g), measured (tarsus length,  $\pm 0.05$  mm), and individually color-marked with nape-tags consisting of two plastic beads on a brass safety-pin (Reed 2000). Chicks were treated orally with 0.05cc of fenbendazole (diluted to 20 mg/mL) at a dosage rate of 50 mg per kg of body mass. Control chicks were given an equivalent dose of sterile water. Treatments and sham controls were administered using a 1 cc sterile syringe with a modified extended tip to allow the syringe to reach past the glottis to ensure the liquid was not aspirated. Marked chicks were returned to their nest bowls within 12 hours of hatching.

Re-observations of marked chicks began 5 days after the first chick hatched and continued until 44 days after all chicks had hatched. I subdivided the sampling effort into 5 age-specific time periods: 1-10 days, 11-20 days, 21-30 days, 31-40 days, and 41-50 days old. I was only interested in measuring survival to 40 days of age, but I needed to extend the capture-recapture matrix by one additional interval in order to measure resighting rate during the final interval. Forty days provided enough time to assess the impact of parental effort on offspring survival, without extending into later stages of development when chicks were more likely to be foraging on their own (Desroschers and Ankney 1986). Chick observations were conducted using a 15-60x spotting scope from a parked vehicle and lasted 1 hour or until all marked chicks were identified. Broods were observed on a weekly rotation in 2004 and every 3 days in 2005, with successive observation periods alternating between morning and afternoon. Nests where no chicks were seen were revisited every other day (every 3 days in 2005) until chicks were seen, or determined to have moved to an adjacent wetland, or have died.

### *Parasite Burdens*

Before laying commenced in early May and after brood observations were completed in late July 2005, I shot an opportunistic sample of adults and chicks in order to document parasite burdens. Pre-laying adults were collected to establish that adult coots in my study area were subject to helminthic infection and to determine baseline infection levels. Post-brooding adults were collected from territories of experimental nests and were assumed to have consumed either medicated or control feed, although there was no way to verify that the unmarked adults were parents to experimental chicks.

In March 2006, coots were thawed, measured (13 external morphological measurements as described in Alisauskas and Ankney 1987), weighed ( $\pm 10$  g with a Pesola scale), and dissected within 48 hours of thawing. Birds were sexed via visual examination of the gonads, spleens were removed and weighed ( $\pm 0.01$  g), and internal organs were examined for helminthic parasites using a 6x magnifying glass. Most helminths in coots are found in the small intestine, caeca, and colon and so analysis focused on these areas (Roudabush 1942, Eley 1976). After all organs were visually inspected for parasites, the intestines were removed just below the gizzard, cut lengthwise, and the contents were expelled into a Petri dish, scraping the mucosa with the blunt end of a scalpel to remove any attached worms. Intestinal contents were then refrigerated overnight. The next day, contents of the intestines were systematically examined in the Petri dish over a gridded light box and all adult worms were counted and removed for verification under a 10x microscope.

After the intestinal contents were examined, the contents of the dish were mixed and a 3 g sample was taken for centrifugal flotation and examination under a 10x

microscope. Samples were mixed with Sheather's sugar solution (specific gravity = 1.28), strained using a tea strainer, and then poured into a 15cc centrifuge tube (Dryden et al. 2005). Eggs per 3 g of intestinal contents were counted using the Wisconsin Sugar Floatation Egg Counting Technique (Averbeck 2002). I also took a small sample (approx. 1 g) of intestinal contents and conducted a direct smear to look for eggs of parasite species that do not readily float in centrifugal flotation (Averbeck 2002, Dryden et al. 2005).

### *Statistical Analyses*

Mark-resighting data were analyzed with Cormack-Jolly-Seber open population models in Program MARK (White and Burnham 1999) to estimate apparent survival ( $\phi$ ) and recapture ( $\rho$ ) rates of chicks. Apparent survival was defined as the probability that an individual alive at age  $i$  survives until age  $i+1$  and does not permanently emigrate from the study population. Most losses represented mortality rather than emigration, because coot chicks are incapable of flight before about 50 days of age (Brinkhof 1997, pers. obs.) and they rarely move between wetlands prior to fledging (pers. obs.; T. Arnold, unpubl. data). Recapture rates were defined as the probability that an individual that has not emigrated and is alive at age  $i$  is seen during that interval. Recapture rates are important for coots because chicks spend considerable time concealed in emergent vegetation and broodmates are rarely all together (Horsfall 1984, Lyon 1991, Reed 2000). Encounter histories for each chick were coded to comprise 6 occasions starting with day 0 (release day), 1-10 days, 11-20 days, 21-30 days, 31-40 days, and 41+ days post-release. If a chick was seen at any time during a particular age-interval (e.g. on day 3 for the 1-10

interval) it was coded as observed (1), whereas chicks that were not seen during the interval were coded as not observed (0). Data for 2004 were coded into 2 attribute groups (chicks treated, chicks untreated), whereas data for 2005 were coded into 4 attribute groups ( $\pm$  chick treatment,  $\pm$  adult treatment). Because treatment protocols differed between years (i.e., adults received food supplements  $\pm$  Panacur in 2005), data from each year were analyzed separately.

The most general Cormack-Jolly-Seber (CJS) model that allowed survival and recapture rates to vary independently among all combinations of treatment groups and age classes (i.e., post-release intervals) was used to examine overdispersion in the data (Burnham and Anderson 1998). I conducted a bootstrap goodness-of-fit test in program MARK to estimate a variance inflation factor ( $\hat{c}$ ) by dividing the observed deviance of the most general model by the mean deviance from 1,000 bootstrap simulations (Cooch and White 2006). Model-fitting decisions were based on Akaike's Information Criterion corrected for sample size and overdispersion (QAIC<sub>c</sub>) (Burnham and Anderson 1998). The best-supported model was determined by the lowest QAIC<sub>c</sub> value and highest Akaike weight ( $w_i$ ) (Burnham and Anderson 1998). I also calculated  $\Delta$ QAIC<sub>c</sub> values as the difference between QAIC<sub>c</sub> of the current model versus the model with minimal QAIC<sub>c</sub>. Any model that had  $\Delta$ QAIC<sub>c</sub> < 2 was presumed to have strong support (Burnham and Anderson 1998).

To achieve the most powerful tests of treatment effects on survival, I first fit reduced-parameter models of resighting rates and age-related variation in survival rates (Lebreton et al. 1992). Survival and resighting rates were first treated as time-invariant (i.e., constant across all 4 age intervals), and then hybrid models allowing pooling of

adjacent intervals were examined (Table 1). I did not expect anthelmintic treatment to influence resighting rates so I also evaluated models that treated resighting rates as equivalent between treatment groups.

After modeling age-specific variation in resighting and survival rates and treatment effects on resighting rates, I evaluated treatment effects on survival using the best-supported model. In addition to testing for survival differences among treatment groups, I also evaluated link functions that considered treatment effects as additive rather than factorial (Lebreton et al. 1992). For example, if there were 4 age-classes for survival in the 2004 analysis, evaluation of an independent treatment effect would require 4 additional parameters (allowing for independent treatment effects at each age class), whereas an additive model would require only 1 additional parameter and treatment effects would be constrained to be of similar magnitude across all 4 age classes. In addition to treatment-by-age additivity, I allowed chick versus adult treatments in 2005 to behave in a potentially additive fashion (i.e., like a 2-way ANOVA with no interaction effect). Additive effects were modeled using the design matrix in Program MARK (Cooch and White 2006). In addition to assessing the contribution of treatment effects to  $\Delta\text{QAIC}_c$ , I also calculated Z-tests to evaluate treatment effects using a more traditional hypothesis-testing framework.

I incorporated model selection uncertainty into my estimates of survival rates by model averaging over all candidate models in Program MARK. Sometimes several models seem plausible, based on  $\Delta\text{QAIC}_c$  values (especially for values under 2) and model averaging is a formal way to base inference on more than a single model (White and Burnham 1999). Akaike weights ( $w_i$ ) are used to calculate the weighted mean of

Table 1. Notation and explanations of variables considered in Cormack-Jolly-Seber (CJS) models in Program MARK (White 1995).

Variables	Sample notation <sup>1</sup>	Explanation
Null	$\Phi_{(\cdot)}, \rho_{(\cdot)}$	Survival (or resighting) rates are constant over all age-classes and attribute groups
Chick age	$\Phi_{(1,2,3,4)}$	Separate estimates of $\Phi$ for each age class: 1 = 1-10, 2 = 11-20, 3 = 21-30, and 4 = 31-40 days old
	$\Phi_{(1,2,3-4)}$	Pooled estimates of $\Phi$ for ages 21-40 days
	$\Phi_{(1,2-4)}$	Pooled estimates of $\Phi$ for ages 11-40 days
Chick treatment	$\Phi_{\text{Chick}}$	Separate survival rates for chicks treated versus not treated with anthelmintics (Panacur)
	$\Phi_{(\text{Chick}*\text{Brood})}$	Model allowing survival to vary depending on whether all broodmates received the same treatment (i.e., Trt vs. Alt <sub>c,t</sub> vs. Ctrl)
Adult treatment	$\Phi_{\text{Adult}}$	Separate survival rates for chicks with parents that were treated vs. not treated with anthelmintics
Interactions	$\Phi_{(1,2,3,4)*\text{Chick}}$	Multiplicative effects of age and chick treatment (e.g. global model for 2004)
	$\Phi_{(1,2,3,4)*\text{Chick}*\text{Adult}}$	Multiplicative effects of age, chick treatment, and adult treatment (e.g., global model for 2005)
	$\Phi_{(1,2,3,4)+\text{Chick}+\text{Adult}}$	Additive effects of age, chick treatment, and adult treatment

<sup>1</sup> The first line gives examples for both local survival ( $\Phi$ ) and resighting rates ( $\rho$ ). Thereafter all examples are for  $\Phi$ , but the same notation also applies to variation in  $\rho$ .

survival estimates over all considered models. Model averaging produces unconditional estimates of variances and standard errors.

Cumulative survival estimates were estimated by multiplying survival estimates in each resighting interval ( $\Phi_1 \cdot \Phi_2 \cdot \Phi_3 \cdot \Phi_4$ ). Variances were estimated using the Delta method (Cooch and White 2006) and are reported as  $\pm$  SE except in figures where error bars are represented either as  $\pm$  2SE or  $\pm$  95% CI. Fledging rates for each treatment group were calculated by multiplying mean initial brood size by cumulative survival to 40 days of age in both 2004 and 2005.

To test whether chick survival rates were dependent on competitive superiority (Lyon et al 1994), I used a dummy variable (“Brood,” with values of 0 or 1) to code for whether all broodmates had received the same fenbendazole treatment (0; i.e. all treatments, or all controls) or whether half of broodmates had received the alternative treatment (1). I used this variable as an individual covariate in the design matrix of Program MARK to code chicks into four different groups and I predicted (based on Lyon et al. 1994) that survivorship would exhibit the following pattern: treated (partial brood) > treated (whole brood) > control (whole brood) > control (partial brood).

To see if parasite burdens of adults increased over the breeding season, I compared adult and immature helminth abundance (defined as: adults, oocysts, and larvae combined) between pre-laying adults and post-laying adults using a Student’s t-test (SAS Institute Inc., 2001). To determine if fenbendazole treatment influenced parasite burdens, I conducted a multivariate analysis of variance (MANOVA; SAS Institute 2001) where adult and immature (eggs + larvae) helminths were treated as two separate response variables and fenbendazole treatment (treated, control) and sex (male,

female) were used as predictor variables. I analyzed chicks and adults separately because treatment regimens for chicks (direct oral dosing) and adults (indirect dosing in feed) were different.

I conducted a Principal Components Analysis (PCA; SAS Institute 2001) of 10 (adults) and 13 (chicks) external morphological measurements to derive an index of structural size for each coot (Alisauskas and Ankney 1987). For adults I excluded total length due to missing values and wing chord and wing length because some adult coots were molting when collected). Morphometric measurements were log transformed and PCA scores were derived from the covariance matrix (Alisauskas and Ankney 1987). I then regressed body mass on PC1 scores and used the residuals from this regression as an index of body condition (Alisauskas and Ankney 1987). Coots with positive body condition scores (greater than scores predicted by the linear trend line) were considered to be in “good” condition (higher mass relative to size), whereas coots with negative (less than those predicted by the linear trend line) scores were considered to be in “poor” condition (lower mass relative to size). To determine if structural size (PC1) and body condition were influenced by anthelmintic treatment or parasite burdens, I used a least-squares linear model with body condition or PC1 as the response variable and parasite abundance (combined adults, larvae, and eggs), fenbendazole treatment, age (days of age for chicks), and sex as predictor variables. I conducted separate analyses for adults and chicks. I predicted that coots that had been treated with fenbendazole and/or had lower parasite burdens would be larger and/or in better body condition.

## Results

Mayfield nest success was 67.7% ( $n = 56$ ) in 2004 and 89.7% ( $n = 118$ ) in 2005. The hatching period of experimental nests ranged from 7 June to 12 July (35 days, mean = 21 June) in 2004 and from 25 May to 17 June (23 days, mean = 7 June) in 2005. A blizzard on 13 May 2004 (total snowfall = 34 cm) destroyed many early-season nests and greatly delayed the onset of hatch. In 2004, mean clutch and brood sizes were about 1 egg and chick smaller than in 2005 (data are means  $\pm$  SE; clutch size: 2004,  $8.85 \pm 0.15$ , 2005,  $9.86 \pm 0.10$ ; brood size: 2004,  $7.88 \pm 0.20$ ; 2005,  $8.89 \pm 0.13$ ).

### *Parasite burdens and body condition:*

I collected data on parasite burdens, body condition, and body size from 38 chicks (23 treated), 6 pre-breeding adults, and 17 post-brooding adults (9 treated) in 2005. Several additional birds had to be eliminated from final analyses due to incomplete data: 16 found birds were too severely damaged and 16 birds that had been found dead were removed from analyses since they did not have known ages (chicks) or were too badly decomposed.

Six adult coots collected pre-nesting had mean parasite burdens of  $2.16 \pm 0.54$  adult worms and  $1.67 \pm 0.49$  immatures, which did not differ from the  $1.38 \pm 0.56$  adults and  $2.13 \pm 0.69$  immature worms found among post-brooding control adults (adult parasites:  $t = 0.99$ ,  $df = 12$ ,  $p = 0.34$ , immature:  $t = 0.51$ ,  $df = 12$ ,  $p = 0.62$ ).

In a MANOVA testing for differences in parasite abundance in relation to fenbendazole treatment, treated chicks had fewer adult and immature parasites than did control chicks, but this effect was not seen among adult coots (Table 2).

Table 2. Effects of fenbendazole treatment on parasite burdens in 17 adult and 38 immature coots. Means (SE) of adult and immature worms are reported as well as F-statistics, degrees of freedom, and P values from Type III sums of squares.

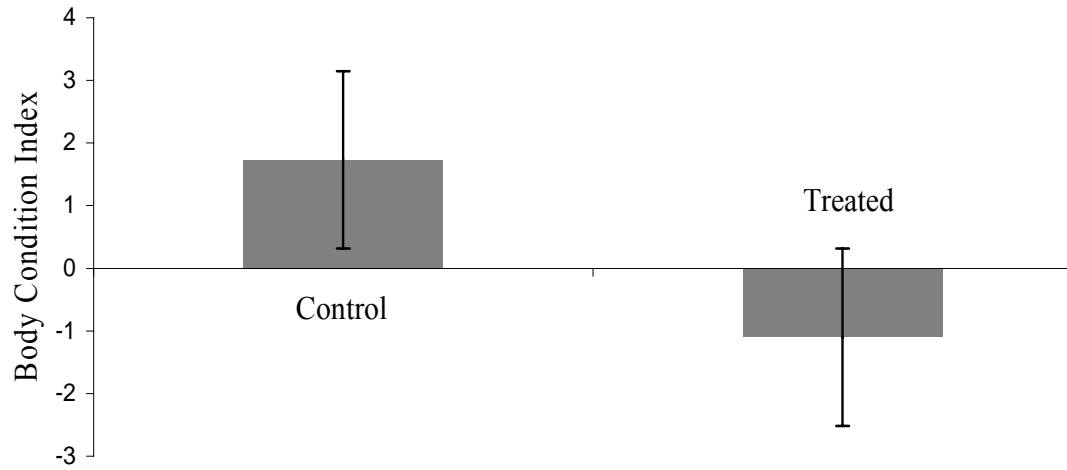
Age Group:	Treated	Control	F	df	P
Adult coots					
Adult worms	2.00 (0.50)	1.38 (0.53)	0.73	1,15	0.41
Immature worms	3.00 (0.72)	2.13 (0.77)	0.69	1,15	0.42
Overall effect	5.00 (0.69)	3.51 (0.66)	0.78	2,14	0.48
Juvenile coots					
Adult worms	2.26 (0.85)	5.40 (1.05)	5.40	1,36	0.026
Immature worms	1.17 (0.25)	2.00 (0.31)	4.35	1,36	0.044
Overall effect	3.43 (0.72)	7.40 (0.88)	3.43	2,35	0.044

A second MANOVA testing for the effects of treatment group, parasite abundance and chick age (correlated with PC1 and so included in analyses) in relation to body condition of adults and chicks, and body size in chicks yielded few insights. In chicks, neither body size nor condition was influenced by treatment group, chick age, or parasite abundance in either sex, although treatment effect was almost significant predicting *worse* condition of treated chicks (Figure 1). In addition, although females had significantly lower body condition than males ( $F = 7.27$ ,  $df = 1,12$ ,  $p = 0.02$ ), treatment group and parasite abundance did not appear to influence body condition of adult coots.

#### *Chick survival -- 2004*

I collected survivorship data from 322 coot chicks from 38 nests in 2004. Three additional broods were censored from analysis because they were unobservable due to thick vegetative cover. Only 13 chicks were resighted during the 41-50 day age interval (vs. 90 - 121 chicks in each of the prior four age intervals), which was too few for reliable parameter estimation, so I excluded the 5<sup>th</sup> resighting interval from my mark-resighting analysis in 2004. The bootstrap goodness-of-fit test on model  $\Phi_{(1,2,3,4)*Chick}$ ,  $\rho_{(1,2,3,4)*Chick}$  revealed little evidence of overdispersion ( $\hat{c} = 1.16$ ), but I nevertheless included this slight variance-correction factor for subsequent modeling. The best-fitting model for the effects of age on resighting probability ( $\rho_{(1,2,3,4)}$ ) recognized all four age intervals as unique (i.e. no parameter reduction occurred), whereas the 2<sup>nd</sup> through 4<sup>th</sup> intervals could be pooled for estimating survival ( $\Phi_{(1,2-4)}$ , yielding a 5.46 reduction in QAIC<sub>c</sub> versus model  $\Phi_{(1,2,3,4)}$ ). There was no evidence that chick treatment affected resighting rates

Figure 1. Mean body condition indices ( $\pm$  SE) of 38 (24 treated, 14 control) collected fledglings in 2005. Differences are nearly significant to a p-value of 0.052.



(2.90 or 5.24 increases in QAIC<sub>c</sub>, depending on whether I treated it as an additive or multiplicative effect), so I considered resighting rates to be equal between treatment groups.

The top-fitting model ( $\Phi_{(1,2-4)+\text{Chick}}$ ,  $\rho_{(1,2,3,4)}$ ) recognized an additive effect of fenbendazole treatment on chick survival over all age classes (Table 3). Although this model was the top-ranked model based on QAIC<sub>c</sub>, the treatment effect was not quite significant based on a conventional Z-test ( $\beta = 0.412$ ,  $\text{SE} = 0.224$ ,  $Z = 1.84$ ,  $P = 0.07$ ). The model recognizing independent effects of fenbendazole treatment on each age class ( $\Phi_{(1,2-4)*\text{Chick}}$ ,  $\rho_{(1,2,3,4)}$ ) had nearly identical parameter estimates, but because it had a 2-unit penalty for an additional parameter, it performed slightly worse than the model recognizing no treatment effects. A model that posited different treatment effects depending on whether all broodmates had been treated similarly ( $\Phi_{(1,2-4 + \text{Chick}*\text{Brood})}$ ,  $\rho_{(1,2,3,4)}$ ) fit less well than the additive treatment model, but was a slight improvement over the model recognizing no treatment effects (Table 3). Survival estimates from this model suggested there was a greater benefit of receiving fenbendazole when half versus all of your broodmates were similarly treated, whereas control chicks had similar survival regardless of brood composition (Figure 2<sub>top</sub>).

Cumulative survival to 40 days of age was higher for chicks that were treated with fenbendazole ( $\Phi_{0-40} = 0.499$ ,  $\text{SE} = 0.051$ ) than for untreated control chicks ( $\Phi_{0-40} = 0.403$ ,  $\text{SE} = 0.071$ ) (Table 4). Parents of treated chicks fledged 3.93 ( $\pm 0.41$ ) young whereas parents of control chicks fledged an average of 3.18 ( $\pm 0.33$ ) young in 2004.

TABLE 3. Models of coot chick survival to 40 days of age in relation to fenbendazole treatment and chick age, southwestern Manitoba, Canada, 2004. Models are ranked by differences in Quasi-AIC (QAIC<sub>c</sub>).

Model <sup>a</sup>	$\Delta\text{QAIC}_c^b$	$w_i^c$	$K^d$	QDev <sup>e</sup>
$\{\Phi_{((1,2-4) + \text{Chick})}, \rho_{(1,2,3,4,5)}\}^f$	0.00	0.33	7	1173.76
$\Phi_{\text{Chick} + \text{Brood} + (1,2-4) + \text{Interaction}},$ $\rho_{(1,2,3,4,5)}$	1.06	0.20	9	1170.72
$\Phi_{(1,2-4)}, \rho_{(1,2,3,4,5)}$	1.33	0.17	6	1177.13
$\Phi_{\text{Chick} * (1,2-4)}, \rho_{(1,2,3,4,5)}$	1.87	0.13	8	1173.58
$\Phi_{\text{Chick} * (1,2-4)}, \rho_{((1,2,3,4,5) + \text{Chick})}$	2.90	0.08	9	1172.55
$\Phi_{(1,2,3,4)}, \rho_{(1,2,3,4,5)}$	3.37	0.06	7	1177.13
$\Phi_{\text{Chick} * (1,2-4)}, \rho_{((1,2,3,4,5) * \text{Chick})}$	5.24	0.02	12	1168.68
$\{\Phi_{\text{Chick} * (1,2,3,4)}, \rho_{(\text{Chick} * (1,2,3,4,5))}\}^g$	10.70	0.00	15	1167.87

<sup>a</sup> See Table 1 for explanation of model notation.

<sup>b</sup> Difference in QAIC<sub>c</sub> relative to model with the lowest value.

<sup>c</sup> Relative model weight (weights sum to 1.00) as evidence of being the actual best-approximating model.

<sup>d</sup> Number of parameters in each model.

<sup>e</sup> Model deviance

<sup>f</sup> QAIC<sub>c</sub> for best-fit model = 1187.934

<sup>g</sup> Global model, not included in model averaged estimates due to an inestimable parameter

Figure 2: Effect of fenbendazole and level of brood treatment (all, none, or half of brood members treated; see Methods) on 40-day cumulative survival of coot chicks in 2004 (top) and 2005 (bottom) plus > 95% CI.

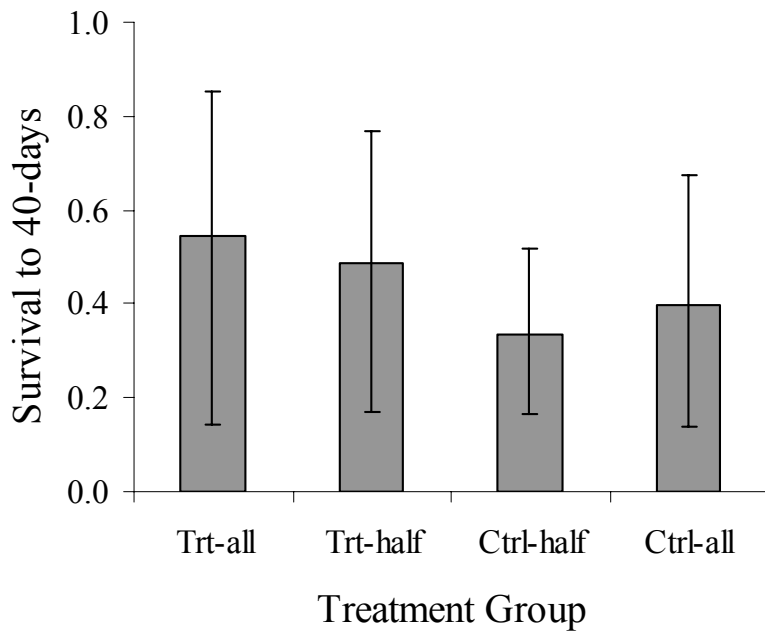
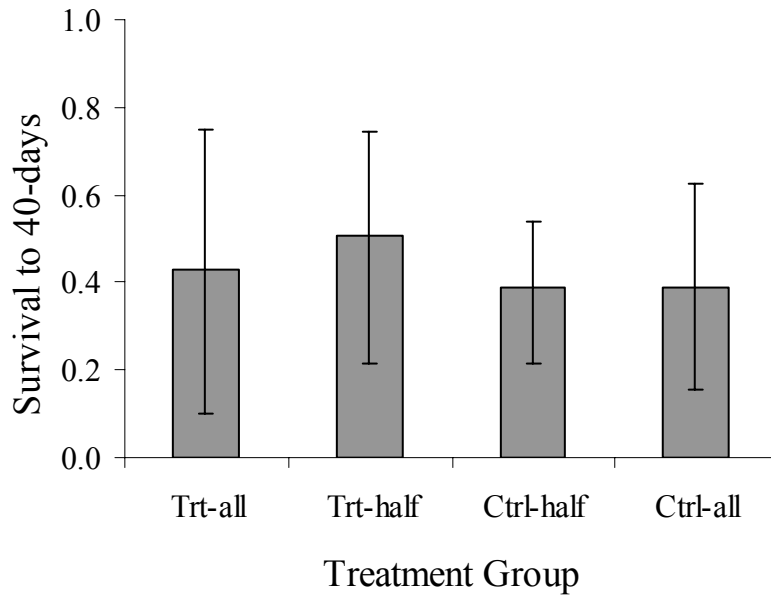


TABLE 4: Number of parameters ( $k$ ), parameter estimates (Est.), standard errors (SE), and lower (LCI) and upper (UCI) 95% confidence limits of apparent survival ( $\Phi$ ) and resighting rates ( $\rho$ ) for American Coot chicks in Minnedosa, Manitoba, Canada in 2004 during each resighting interval (0-10 days =  $\Phi_1$ , 11-20 days =  $\Phi_2$ , 21-30 days =  $\Phi_3$ , and 31-40 days post-hatch =  $\Phi_4$ ). Cumulative survival<sup>a</sup> of each treatment group ( $\Phi_{T,C}$ ) is also reported, along with its standard error (SE)<sup>b</sup>.

	$k$	Est.	SE	LCI	UCI
Treated	$\Phi_{(1)}$	0.620	0.042	0.535	0.699
	$\Phi_{(2-4)}$	0.930	0.029	0.847	0.969
	$\Phi_T$	0.499	0.051		
Control	$\Phi_{(1)}$	0.550	0.054	0.575	0.738
	$\Phi_{(2-4)}$	0.901	0.040	0.809	0.932
	$\Phi_C$	0.403	0.071		
T,C	$\rho_{(1)}$	0.480	0.041	0.400	0.567
	$\rho_{(2)}$	0.620	0.042	0.530	0.700
	$\rho_{(3)}$	0.760	0.047	0.655	0.840
	$\rho_{(4)}$	0.610	0.061	0.485	0.723

<sup>a</sup> Cumulative survival was calculated as  $(\Phi_{(1)} \cdot \Phi_{(2-4)}^3)$ .

<sup>b</sup> Overall SE was calculated using the Delta Method (Cooch and White 2006)

### *Chick survival -- 2005*

I collected survivorship data from 340 chicks from 40 nests in 2005. Four additional broods were censored due to inability to resight the brood (3) or investigator-induced abandonment (1). The bootstrap goodness-of-fit test on the full model ( $\Phi_{(1,2,3,4)*Chick*Adult}$ ,  $\rho_{(1,2,3,4)*Chick*Adult}$ ) revealed modest evidence of overdispersion ( $\hat{c} = 1.66$ ). The best-fitting model for resighting probability recognized constant sightability across all treatment groups and age classes ( $\rho = 0.566$ ,  $SE = 0.027$ ). Estimated survival probabilities for intervals 2 (ages 11-20) and 3 (ages 21-30) were often 100%, so I pooled these 2 intervals to help reduce the number of inestimable parameters (Table 5), consequently yielding an 8.18 reduction in QAIC<sub>c</sub> (model  $\Phi_{((1,2-3,4,5)*Chick*Adult)}$ ,  $\rho_{(.)}$ ) versus model  $\Phi_{((1,2-3,4,5)*Chick*Adult)}$ ,  $\rho_{(.)}$ ). The top-fitting model (Table 5:  $\Phi_{Adult + Chick + (1,2-3,4,5)}$ ,  $\rho_{(.)}$ ) recognized additive effects of fenbendazole treatment on chicks and adults over all age classes.

Although this model was the top-ranked model based on QAIC<sub>c</sub> (Table 5), neither the chick treatment effect ( $\beta = 0.444$ ,  $SE = 0.247$ ,  $Z = 1.80$ ,  $P = 0.07$ ) nor the adult treatment effect ( $\beta = 0.413$ ,  $SE = 0.247$ ,  $Z = 1.68$ ,  $P = 0.09$ ) were significant based on Z-tests, although both results were in the predicted direction of higher survival among fenbendazole treated birds. The model that considered different treatment effects depending on whether or not all broodmates had been treated similarly fit less well than the additive treatment model, but was a slight improvement over the model recognizing no treatment effects (Table 6). However, results from this model did not support my a priori predictions, with both treatment and control chicks surviving better when the entire brood was treated similarly (Figure 2<sub>bottom</sub>).

TABLE 5. Models of coot chick survival to 40 days of age in relation to fenbendazole treatment and chick age, southwestern Manitoba, Canada, 2005. Models are ranked by differences in Quasi-AIC (QAIC<sub>c</sub>).

Model <sup>a</sup>	ΔQAIC <sub>c</sub> <sup>b</sup>	w <sub>i</sub> <sup>c</sup>	K <sup>d</sup>	QDev <sup>e</sup>
{Φ <sub>Adult + Chick + (1,2-3,4), ρ(.)</sub> } <sup>f</sup>	0.00	0.26	7	1037.86
Φ <sub>Chick + (1,2-3,4), ρ(.)</sub>	0.76	0.18	6	1040.66
Φ <sub>Adult + (1,2-3,4,5), ρ(.)</sub>	1.21	0.14	6	1041.11
Φ <sub>Adult + (Chick * Brood) + (1,2-3,4,5), ρ(.)</sub>	1.32	0.14	9	1035.09
Φ <sub>(1,2-3,4,5), ρ(.)</sub>	1.46	0.13	5	1043.40
Φ <sub>Adult * Chick + (1,2-3,4,5), ρ(.)</sub>	1.80	0.11	8	1037.62
Φ <sub>Adult * (1,2-3,4,5), ρ(.)</sub>	4.63	0.03	9	1038.41
Φ <sub>Chick * (1,2-3,4,5), ρ(.)</sub>	6.13	0.01	9	1039.90
Φ <sub>(Adult + Chick) * (1,2-3,4,5), ρ(.)</sub>	15.51	0.00	17	1032.71
Φ <sub>(Adult + Chick) * (1,2,3,4,5), ρ(.)</sub>	23.69	0.00	21	1032.47
{Φ <sub>(Adult * Chick) * (1,2,3,4,5), ρ(Adult * Chick) * (1,2,3,4,5)}</sub> <sup>g</sup>	42.90	0.00	36	1019.31

<sup>a</sup> See Table 1 for an explanation of model notation.

<sup>b</sup> Difference in QAIC<sub>c</sub> relative to model with the lowest value.

<sup>c</sup> Relative model weight (weights sum to 1.00) as evidence of being the actual best-approximating model.

<sup>d</sup> Number of parameters in each model.

<sup>e</sup> Model deviance

<sup>f</sup> QAIC<sub>c</sub> for best-fit model = 1052.01

<sup>g</sup> Global model.

Table 6: Number of parameters ( $k$ ), parameter estimates (Est.), standard errors (SE), and lower (LCI) and upper (UCI) 95% confidence limits of apparent survival ( $\Phi$ ) and resighting rates ( $\rho$ ) for American Coot chicks in Minnedosa, Manitoba, Canada in 2005 during each resighting interval (0-10 days =  $\Phi_1$ , 11-20 days =  $\Phi_2$ , 21-30 days =  $\Phi_3$ , and 31-40 days post-hatch =  $\Phi_4$ ). Cumulative survival<sup>a</sup> of each treatment group ( $\Phi_{\text{Treatment group}}$ ) is also reported, along with its standard error (SE)<sup>b</sup>. CC = parents control, chicks control; CT = parents control, chicks treated; TT = both parents and chicks treated; and TC = parents treated, chicks control. Survival rates in the second and third intervals ( $\Phi_2$  and  $\Phi_3$ ) were similar and therefore combined in the analysis and subsequent survival estimation. Resighting rate was constant ( $\rho$ ).

	$k$	Est.	SE	LCI	UCI
CC	$\Phi_1$	0.522	0.074	0.380	0.661
	$\Phi_2 \Phi_3$	1.00	0.00	1.00	1.00
	$\Phi_4$	0.718	0.104	0.482	0.875
	$\Phi_{\text{CC}}$	0.375 <sup>a</sup>	0.091 <sup>b</sup>		
CT	$\Phi_1$	0.603	0.057	0.488	0.708
	$\Phi_2 \Phi_3$	1.00	0.00	1.00	1.00
	$\Phi_4$	0.781	0.085	0.574	0.904
	$\Phi_{\text{CT}}$	0.471	0.078		
TT	$\Phi_1$	0.661	0.057	0.543	0.761
	$\Phi_2 \Phi_3$	1.00	0.00	1.00	1.00
	$\Phi_4$	0.826	0.071	0.644	0.925
	$\Phi_{\text{TT}}$	0.545	0.074		
TC	$\Phi_1$	0.590	0.058	0.473	0.698
	$\Phi_2 \Phi_3$	1.00	0.00	1.00	1.00
	$\Phi_4$	0.776	0.085	0.570	0.901
	$\Phi_{\text{TC}}$	0.458	0.077		
P	$\rho$	0.566	0.027	0.513	0.618

<sup>a</sup> Overall group survival was calculated by multiplying survival estimates of all 4 resighting intervals ( $\Phi_1 * \Phi_2 * \Phi_3 * \Phi_4$ ).

<sup>b</sup> Overall SE was calculated using the Delta Method (Cooch and White 2006)

Based on model-averaged results, cumulative survival to 40-days of age was highest when both chicks and adults were treated with fenbendazole ( $\Phi_{0-40} = 0.545$ , SE = 0.074). If either the chicks or the adults had been treated, survival was intermediate (Chicks treated:  $\Phi_{0-40} = 0.471$ , SE = 0.078; adults treated:  $\Phi_{0-40} = 0.458$ , SE = 0.077). But if neither chicks nor adults received anthelmintics, then cumulative survival was only 0.375 (SE = 0.091) (Table 6). Fledging rates were highest for treated adults with treated chicks ( $4.85 \pm 0.33$ ), followed by control adults with treated chicks ( $4.19 \pm 0.29$ ), treated parents with control chicks ( $4.07 \pm 0.28$ ), and the lowest rates were for control parents with control chicks ( $3.33 \pm 0.23$ ).

## **Discussion**

Coots in this study had high nest success in both years and most nests fledged at least one chick. As a consequence of delayed nesting in 2004, mean clutch and brood sizes were about 1 egg and chick smaller than in 2005. Experimental treatment in adults and chicks was successful in increasing fledging success, but condition of chicks and adults and size of chicks appeared unaffected by anthelmintic treatment.

### *Parasite burdens*

After treating chicks at hatching with fenbendazole, I found an average of 3.4 total parasites per chick when chicks were collected at 45 days of age, whereas untreated chicks had slightly more than twice as many total parasites ( $\bar{x} = 7.4$ ). This difference was especially pronounced for adult worms, which averaged 2.3 among treated chicks versus

5.0 among control chicks. Although total parasite burdens were low in both groups, my results were consistent with parasite burdens documented by Eley (1976).

In contrast to chicks, parasite burdens did not differ between treated and control adults collected 45 days post-treatment, and in fact average parasite burdens were non-significantly higher among adults that received fenbendazole. Since adults were not visibly marked, it is possible that my sample of adults may have included non-breeding adults or adults that had only incidental access to medicated feed. It is also possible that 45 days post-treatment was too late to see anthelmintic effects in adults, and burdens may have reestablished to control levels (unlike for juvenile coots, most parasites in adult coots consisted of oocysts or larval worms, rather than adult worms). However, if this were the case I would have expected to see similar results in chicks, since chicks were also treated 45 days prior to collection, on average.

#### *Effects of treatment on offspring survival and fledging success*

I found that survival of coot chicks increased with anthelmintic treatment of both parents and offspring. Broods that were fully treated with fenbendazole fledged from 0.7 to 0.9 more chicks per brood than did untreated broods in 2004 and 2005 (representing 19-24% increases in fledging success). Treating parents with fenbendazole in 2005 increased fledging success by an additional 0.7 to 0.8 chicks per brood. For broods in 2005 in which both parents and chicks were treated, fledging success increased by 46% over that observed in untreated broods.

Since chicks ingest mainly invertebrates early in life (Driver 1988), parasite transmission could occur almost immediately after hatch. I expected that by reducing

parasite burdens early in life, a large part of the benefit would be observed at later ages when control chicks began suffering the effects of more advanced parasitic infections (i.e. from adult worms). However, survival advantages were observed through the first 40 days of life, suggesting that reduction of parasites resulted in an immediate benefit to treated chicks, which may be a consequence of higher mortality in younger chicks (e.g. Chouinard 2000) and since survival often reaches a plateau among older offspring.

My results suggest that even small differences in parasite burdens can influence fledging success in coots. Helminths can cause anorexia, disrupt gastro-intestinal (GI) tract motility and secretions, and damage the walls of the GI tract (Chowdhury and Tada 1994). Reduced food intake along with the increased metabolic needs of parasitized animals may lead to decreased body condition and further suppression of the host's natural defenses against parasites (Chowdhury and Tada 1994).

Since coot chicks compete with their broodmates for parental resources, an increase in health or condition of individual chicks should allow them to compete more effectively against their broodmates, thereby increasing their relative prospects of survival (Lyon et al. 1994). Chicks in better condition may be better able to beg for food, survive periods of non-feeding (parental avoidance), or alleviate the negative effects of asynchrony (Lyon et al. 1994). Following Lyon's rationale, I hypothesized that treating half the chicks in a brood with an anthelmintic would increase the survival of treated chicks even more than treating all the chicks in a brood, since treated chicks within partially treated broods would have a competitive advantage over their untreated siblings. Similarly, I predicted that untreated chicks in partially treated broods would have even lower survival than chicks in completely untreated broods, since the untreated chicks in

partially treated broods would be at a competitive disadvantage to their treated siblings. Although there was some support for models that differentiated between broods where some versus all of the chicks had been treated with fenbendazole, the differences in survival did not fully support my a priori prediction of competitive hierarchies within broods. In 2004, treated siblings had much higher survival rates than control siblings from mixed broods (i.e., the treatment effect was greater within broods than across broods), and treated chicks from mixed broods had higher survival than treated chicks from fully treated broods (i.e., this evidence was supportive), but control chicks from mixed broods did not differ from control chicks in fully untreated broods. In 2005, control chicks from mixed broods survived less well than control chicks from fully untreated broods (this was supportive), but treated chicks from mixed broods survived less well than chicks from fully-treated broods, and treatment effects were stronger among broods than within broods (i.e. most evidence was contrary to the competitive advantage hypothesis).

Møller and Saino (2004) recently reviewed the role of the immune system in bird survival. Their meta-analysis found that survivorship in young birds was positively correlated with the ability to mount a strong immune response, as measured by t-cell immunity (Moller and Saino 2004). Although I did not measure immune response functions or parasite burdens in very young chicks, their explanation is consistent with what I observed in terms of enhanced survivorship among all ages of coot chicks. I hypothesized that reducing parasites in coot chicks may have served multiple functions, alleviating parasite-derived taxation of their immune systems and increasing food consumption and nutrient absorption, thereby increasing the probability of survival to 40

days post-hatch. However, evidence of better nutritional condition was contrary to my hypothesis, with no effect of anthelmintics treatment on size or condition of chicks or adults and an almost significant *negative* effect of treatment on condition of chicks. Despite this lack of nutritional benefit, chicks survived better when either they or their parents or both they and their parents were treated with an anthelmintic.

I recommend that future investigations of anthelmintic treatment in coots include markers for both chicks and adults, and that coots be examined for parasite burdens sooner following treatment. Gross examination of intestinal contents and internal organs suggests parasite burdens in coots are low (Roudabush 1942, Eley 1976, this study); however detailed histopathic and serological examination could provide insight into total parasite burdens in coots and which parasites are most abundant and have the most impact in coots during late summer.

## Literature Cited

- Adams, H.R. ed. 2001. Veterinary Pharmacology and Therapeutics. 8<sup>th</sup> ed. Ames: Iowa State University Press, 1201p.
- Alisauskas, R.T. and C.D. Ankney. 1987. Morphometric correlates of age and breeding status of American Coots. *Auk* 104(4): 640-646.
- Alisauskas, R.T. and T.W. Arnold. 1994. American Coot. Migratory Shore and Upland Game Bird Management in North America. Thomas C. Tacha and Clait E. Braun, editors. p. 127-143.
- Arnold, T.W. 1994. Effects of supplemental food on egg production in American Coots. *Auk* 111: 337-350.
- Arnold, T.W. 1990. Food limitation and the adaptive significance of clutch size in American coots (*Fulica americana*) Unpublished Ph.D. diss., University of Western Ontario, London, Ontario.
- Averbeck, G. 2002. Diagnostic Parasitology Techniques. University of Minnesota Press St. Paul, Minnesota p. 31.
- Brinkhof, M.G. 1997. Seasonal decline in body size of coot chicks. *Journal of Avian Biology* 28(2): 117-131.
- Burnham, K. P. and D. R. Anderson. 1998. Model selection and inference: a practical information-theoretic approach. Springer-Verlag, New York, NY.
- Chinouard. 2000. Mallard (*Anas platyrhynchos*) duckling survival in central California Unpublished Master's thesis, Humboldt State University, Humboldt, California.
- Chowdhury, N. and I. Tada. Ed. 1994. Helminthology. New Delhi: Narosa Publishing House 373p.
- Clayton, D.H. and J. Moore ed. 1997. Host-Parasite Evolution: General Principles and Avian Models. Oxford: Oxford University Press 473p.
- Cole, R.A. and M. Friend. 1999. Field Manual of Wildlife Diseases: Birds. (eds M. Friend and J.C. Fransen) USGS Biological Resources Division, 426 p.
- Cooch, E. and G.C. White. 2006. Program MARK: A Gentle Introduction. 4<sup>th</sup> ed. [www.phidot.org/software/mark/docs/book](http://www.phidot.org/software/mark/docs/book).
- Davis, J.W., R.C. Anderson, L. Kastad, and D.O. Trainer. 1971. Infectious and parasitic diseases of wild birds, 1<sup>st</sup> ed. Iowa State University Press, Ames.

- Desrochers, B.S. and C.D. Ankney. 1986. Effect of brood size and age on the feeding behavior of adult and juvenile American Coots (*Fulica americana*). *Canadian Journal of Zoology* 64: 1400-1406.
- Dryden, M.W., P.A. Payne, R. Ridley and V. Smith. 2005. Comparison of common fecal flotation techniques for the recovery of parasite eggs and oocysts. *Veterinary Therapeutics* 6(1): 15-28.
- Driver, E.A. 1988. Diet and behaviour of young American Coots. *Wildfowl* 39: 34-42.
- Eley, T.J. 1976. Helminth parasites in American Coots from the lower Colorado river. *California Fish and Game* 62(2): 156-157.
- Foreyt, B. 2001. *Veterinary Parasitology: reference manual*, 5<sup>th</sup> ed. Ames: Iowa State University Press viii, 235p.
- Hanssen, S.A., I. Folstand, K.E. Erikstad and A. Oksanen. 2003. Costs of parasites in common eiders: effects of antiparasite treatment. *Oikos* 100(1): 105-111.
- Horsfall, J.A. 1984. Brood reduction and brood division in coots. *Animal Behaviour* 32: 216-225.
- Howard, L.L. et al. 1999. Benzimidazole toxicity in birds. *Am. Assoc. Zoo Vet. Annu. Proc.* 36. 1999. ("Proceedings of the American Association of Zoo Veterinarians Annual Conference held in Columbus, Ohio, October 9-14, 1999"). **Notes:** abstract only
- Hays H. and M. Lecroy. 1971. Field criteria for determining incubation stage in eggs of the common tern *Wilson Bulletin* 83(4): 425-429.
- Hudson, P.J. 1992. *Red Grouse Population Studies*. The Game Conservancy, Fordingbridge.
- Hudson, P.J., D. Newborn and A.P. Dobson. 1992. Regulation and stability of a free-living host-parasite system, *Trichostrongylus tenuis* in red grouse. I. Monitoring and parasite reduction experiments. *Journal of Animal Ecology*, 61: 477-486.
- Kiel W.H. 1955. Nesting studies of the coot in southwestern Manitoba. *Journal of Wildlife Management* 19(2): 189-198.
- Kinsella, J.M. 1973. Helminth parasites of the American Coot *Fulica americana americana* on its winter range in Florida. *Proceedings of the Helminthological Society of Washington* 40: 240-242.
- Kocan, A.A., M.G. Shaw and P.M. Morgan. 1979. Some parasitic and infectious diseases in waterfowl in Oklahoma. *Journal of Wildlife Diseases* 15(1): 137-141.

- LaPage, G. 1962. A list of the parasitic protozoa, helminthes and arthropoda recorded from species of the family anatidae (ducks, geese, and swans). *Wildlife Diseases* 26: 109p.
- Lebreton, J.D., K.P. Burnham, J. Clobert and D.R. Anderson. 1992. Modeling survival and testing biological hypotheses using marked animals: a unified approach with case studies. *Ecological Monographs* 62: 67-118.
- Lyon, B.E., J.M. Eadie, and L.D. Hamilton. 1994. Parental choice selects for ornamental plumage in American Coot chicks. *Nature* 371: 204-243.
- Lyon, B.E. 1991. Brood parasitism in American Coots: avoiding the constraints of parental care. *Acta XX Congressus Internationalis Ornithologia* Vol. II p.1023-1030.
- McKindsey, C.W., J.K. Goring and J.D. McLaughlin. 1994. In vivo and in vitro studies on the viability and the infectivity to coots, *Fulica americana* of *Cyclocoelum mutabile metacerariae* from three species of snails. *Canadian Journal of Zoology* 72(7): 1186-1190.
- McLaughlin. 1986. The biology of *Cyclocoelum mutabile* (Trematoda) infections in American Coots. *Proceedings of the Helminthological Society of Washington* 53: 177-181.
- Moller, A.P. and N. Saino. 2004. Immune response and survival. *Oikos* 104(2): 299-304.
- Moller, A.P. and J. Erritzoe. 2002. Coevolution of host immune defence and parasite-induced mortality: relative spleen size and mortality in altricial birds. *Oikos* 99: 95-100.
- Moller, A.P., Christe, P. and Lux, E. 1999. Parasite-mediated sexual selection: effects of parasites on host immune function. *Quarterly Review of Biology* 74: 3-20.
- Newborn, D. and R. Foster. 2002. Control of parasite burdens in wild red grouse *Lagopus lagopus scoticus* through the indirect application of anthelmintics. *Journal of Applied Ecology* 39: 909-914.
- Navarro, C. F. de Lope, A. Marzal and A.P. Moller. 2004. Predation risk, host immune response, and parasitism. *Behavioral Ecology* 15(4): 629-635.
- Reed, W. R. 2000. Maternal effects in the American coot: consequences for offspring growth and survival. PhD Dissertation, Iowa State University, Ames, IA.

- Righi, M. and G. Gauthier. 2002. Natural infections by internal cestodes: variability and effect on growth in greater snow goose goslings (*Chen caerulescens atlantica*). Canadian Journal of Zoology 80(6): 1077-1083.
- Roudabush, R. K. 1942. Parasites of the American Coot (*Fulica americana*) in Iowa. Iowa State College Journal of Science 41: 437-441.
- Ryan, M. R., and J. J. Dinsmore. 1979. A quantitative study of the behavior of breeding American Coots. Auk 96: 704-713.
- SAS Institute. 2001. SAS/STAT User's Guide, version 6, 4<sup>th</sup> ed. SAS Institute Inc. Cary, North Carolina.
- Skallerup P., L.A. Luna, M.V. Johansen et al. 2005. The impact of natural helminthic infections and supplemental protein on growth performance of free-ranging chickens on smallholder farms in El Sauce, Nicaragua. Preventative Veterinary Medicine 69(3-4): 229-244.
- Slattery, S.M. and R.T. Alisauskas. 2002. Use of the Barker model in an experiment examining covariate effects on first-year survival in Ross's Geese (*Chen rossii*): a case study. Journal of Applied Statistics 29(1-4): 497-508.
- Stoudt, J.H. 1982. Habitat use and productivity of canvasbacks in southwestern Manitoba. 1961-1972. U.S. Fish and Wildlife Service Special Sci. Report. Wild. No. 248 iv + 29p.
- Van Oers, K., D. Heg, and S.L.D. Quenec'hdu. 2002. Anthelmintic treatment negatively affects chick survival in the Eurasian oystercatcher *Haematopus ostralegus*. Ibis 144: 509-517.
- White, G.C. 1995. Multi-species investigations: consulting services for mark-recapture analysis. Colorado Division of Wildlife 14p.
- White, G.C. and K.P. Burnham. 1999. Program MARK: survival estimation from populations of marked animals. Bird Study 46, supplement 120-138.