SHORT COMMUNICATIONS

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ESTRADIOL CYPIONATE (ECP) MARKEDLY IMPROVES SURVIVAL OF WILLOW PTARMIGAN IN CAPTIVITY[1]

KATHY MARTIN[2] AND CYNTHIA A. WRIGHT
Division of Life Sciences, Scarborough Campus, University of Toronto, 1265 Military Trail, Scarborough, Ontario, M1C 1A4, Canada

Key words: Estradiol cypionate (ECP); survival in captivity; removal experiments; Willow Ptarmigan; Lagopus lagopus.

Experiments that remove individuals are very useful in addressing questions in population and behavioral ecology (Hannon 1986). If experimental designs require the absence of individuals for relatively short durations there are a number of ecological and ethical reasons to hold the removed animals in captivity. For example, populations that exist at low density or have low fecundity may have difficulty recovering from removal experiments, particularly when older, experienced or high quality individuals are removed. Altering the demographic structure of a population may have behavioral or other consequences that could reduce the reliability of results from studies in future years. Releasing removed individuals elsewhere may be undesirable as they may return to the study site during the experiment, or they may survive and reproduce poorly at the new site due to lack of familiarity with the area, or reduced competitive abilities with individuals residing in the new area. Although it may be desirable to maintain removed animals in captivity, wild birds often survive poorly or suffer loss of body condition in captivity, especially some species of Tetraoninae such as grouse and ptarmigan (West 1968, West and Meng 1968, McEwen et al. 1969, Moss 1972, Pendergast and Boag 1971).

This study was part of a larger investigation that used removal experiments to examine the importance of biparental care in Willow Ptarmigan (*Lagopus lagopus*) for survival and body condition of offspring and breeding females (Martin and Cooke 1987, Martin 1989). In this paper, we report methodologies that improved survival in captivity of Willow Ptarmigan removed during the breeding season. Also, we measured survival in subsequent years of birds placed in captivity.

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[2] Present address: Canadian Wildlife Service, Pacific and Western Region, P.O. Box 340, Delta, British Columbia, Canada V4K 3Y3.

METHODS AND STUDY AREA

The study was conducted on about 10 km^2 of subarctic tundra at La Perouse Bay, 40 km east of Churchill, Manitoba, Canada (58°24′N, 94°24′W). The vegetation, study area, field methods, and general biology of this population of Willow Ptarmigan were described earlier by Jefferies et al. (1979) and Martin (1984). During 1981–1983, pairs of ptarmigan were assigned randomly to control or removal treatments after territories were established. Paired males were removed from territories at the onset of incubation or when their chicks hatched. Replacement males were also removed if they remained with the "widowed" female for three days. During 1982–1983, paired females were removed on the day their eggs hatched.

Most ptarmigan were captured before incubation with an extendable noosing pole (Zwickel and Bendell 1967), and individually marked with one numbered metal band and three color bands. Birds were classified as yearlings (hatched the previous season) or adults (2+ years) by comparing pigmentation on the eighth and ninth primaries, and sexes were distinguished on the basis of plumage, voice and wing chord (Bergerud et al. 1963). We determined territorial and mating status of birds prior to onset of the removals. On the assigned day of removal, birds were captured and their wing chord and body mass recorded. Birds were placed in a cotton banding bag and carried upright to the field camp. Here, they were weighed again to record loss of mass in transport and placed in individual cages. Travel time to the camp ranged from 15 to 90 min. Dates of removal ranged from 8 June to 24 July for males and from 28 June to 25 July for females.

The cages were placed on a small island adjacent to camp and constructed using heavy cord netting and aluminum poles in an elongated triangular design 1 m high with the sides and floors being continuous. Individual cages were made by closing off sections of the tubular structure. During inclement weather, additional cover was provided by placing pieces of plywood on the sides of the cages. Birds were provided with water and fed freshly cut branches of several species of willow (*Salix* spp.) *ad libitum*. We avoided placing males in adjacent cages to reduce visual contact with other males. In accordance with the experimental protocol, birds

TABLE 1. Survival and return in subsequent years of Willow Ptarmigan placed in captivity at La Perouse Bay, Manitoba, 1981–1983.

Comparison		*n*	% survival	G/G_{ran}	*P*
a) Survival during the breeding season					
Injected	Yes	85	84	16.7	0.00
	No	4	0		
Injection route	0.1 mg intramuscular	21	95	15.3[a]	0.00
	0.2 mg intramuscular	43	70		
	0.2 mg combination	21	100		
Sex[b]	Male	59	78	0.2	0.70
	Female	12	71		
Age	Adult	59	81	0.7	0.40
	Yearling	26	88		
b) Return to the study area in subsequent years					
Injection route	0.1 mg intramuscular	20	55	1.2[a]	0.54
	0.2 mg intramuscular	20	70		
	0.2 mg combination	21	62		
Sex	Male	62	64	0.9	0.78
	Female	10	60		
Age	Adult	48	69	1.5	0.23
	Yearling	24	54		

[a] df = 2.
[b] Data from 1982 and 1983 only as no females were removed in 1981.

were released either the day after their eggs hatched or were depredated, or when their hatched offspring had reached two weeks of age. Time in captivity ranged from 1 to 20 days.

Immediately prior to placing ptarmigan in cages, we injected them with either 0.1 or 0.2 mg of 17β-estradiol in cottonseed oil [estradiol cypionate ECP; concentration of 1 mg per ml; Upjohn]. Initial dosage levels of 0.1 mg (0.2 mg/kg of body mass) and 0.2 mg (0.4 mg/kg) were calculated in consultation with the Queen's University veterinarian. We used intramuscular (to promote rapid entry into the system) and subcutaneous (to allow a gradual release of hormone) injections. We initially injected ECP with the purpose of bringing males out of breeding condition by altering the balance of reproductive hormones (Lorenz 1954), thereby reducing the time required to maintain them in captivity. In 1981 we injected birds, kept them in captivity for 7–10 days to allow time for the ECP to take effect, and then released them. This did not alter the behavior of males as desired; birds returned to their territories and mates shortly after release (Martin 1991). However, the ECP injections markedly increased survival of captive birds. In 1982 and 1983, we continued injections of ECP to promote survival in captivity. Four treatments were given to ptarmigan placed in captivity: no injection, 0.1 mg intramuscular, 0.2 mg intramuscular, and 0.2 mg combined (0.1 mg intramuscular and 0.1 mg subcutaneous).

Caged birds were checked visually each day to monitor survival. Immediately prior to their release, we weighed birds to obtain mass loss in captivity. In this analysis, we measured survival, rate of loss of body mass during captivity, loss of body mass in captivity and rates of return of individuals in subsequent years. Also, we examined the influence of dosage and injection route on birds in relation to sex, age and body mass at capture, year of capture and date of capture. Hannon et al. (1988) reported annual differences in timing of breeding for the study site; thus, we adjusted dates of capture to compare effects between years. Here, we standardized annual variation by adjusting dates of capture to an overall mean date of clutch initiation (5 June) for all three years. To dates of capture, we added three days in 1981, six days in 1982, and subtracted eight days for 1983.

Wing chord is a useful measure of size in Willow Ptarmigan (Robb et al. 1992). Body mass of male and female ptarmigan varies over the breeding season in relation to stage of reproduction (Hannon and Roland 1984). We found significant interactions between body mass and wing chord ($r^2 = 0.52$, $n = 100$, $P = 0.0001$) and body mass and adjusted date of capture ($r^2 = 0.10$, $n = 100$, $P = 0.0015$). Thus, we adjusted body mass using analysis of covariance (ANCOVA) with sex as the factor and wing chord and adjusted date of capture as covariates. We used the residuals from this ANCOVA to examine survival in relation to body condition at the time of capture and removal.

Sample sizes differed for variables measured because complete data were not available for all individuals. *G*-tests and two-way or multi-way contingency analyses were done using log likelihood chi-squares; if the samples were small we performed sampled randomization tests ($n = 500$ tests, Sokal and Rohlf 1981) and reported G_{ran} values. Continuous variables were analyzed using analysis of variance (SAS Inst. 1987). To examine the source of heterogeneity in survival, numerically-ordered net body mass loss categories were compared using 2 × 2 contingency analysis, and adjacent categories that were homogeneous were pooled and compared to the succeeding category (Bishop et

TABLE 2. Survival of male Willow Ptarmigan in captivity that were injected with 0.2 mg of estradiol cypionate (ECP), intramuscular only, at La Perouse Bay, Manitoba 1981–1983.

Comparison	*n*	% survival	Fate	Mean ± SE	*F*	*P*
Mass loss in transport (g)	27	70	Lived	13.7 ± 2.5	0.3	0.57
			Died	16.2 ± 3.4		
Body mass residuals (g)[a]	42	74	Lived	−4.7 ± 5.9	0.005	0.94
			Died	−5.5 ± 8.8		
Wing chord (mm)	42	74	Lived	204.2 ± 0.9	1.3	0.27
			Died	206.3 ± 1.8		
Date of capture[b]	42	74	Lived	183.3 ± 2.0	11.0	0.002
			Died	171.4 ± 2.3		

[a] Body mass adjusted for date of capture and wing chord.
[b] Date of capture (Julian) adjusted for annual differences in timing of breeding.

al. 1975). Statistical comparisons were omitted when sample sizes were small. Unless otherwise noted, values are presented as mean ± standard error.

RESULTS

Factors influencing survival in captivity. Eighty-nine (76 males, 13 females) of the 100 birds removed during the 1981 to 1983 breeding seasons were placed in captivity. The four birds (three males, one female) that did not receive injections of ECP died. All uninjected males died during the fourth day of captivity; the female died during the second day. However, only 14 (16%) of the birds injected with ECP died in captivity. The injection route and amount of ECP administered influenced survival. Birds given intramuscular injections of 0.2 mg had lower survival than the other two injection regimes (Table 1a). Survival of captive birds was not related to either sex or age class (Table 1a). The mean time in captivity of 61 injected males that survived was 10.9 ± 0.5 days, range 1–20, significantly longer than 11 injected males that died (mean = 4.7 ± 0.6 days, range 2–7, $F = 27.1$, $P = 0.0001$). Thus, all mortality occurred during the first week of captivity.

The mean adjusted body mass at time of removal for 86 males was 602.3 ± 3.8 g and for 14 females was 506.1 ± 9.7 g. We examined whether residuals of adjusted body mass at time of capture, net mass loss, or percent loss of body mass during the first week in captivity influenced survival in captivity. For injected males, residuals of body mass at time of removal did not differ in relation to survival (survived: mean = −1.1 ± 4.2 g, $n = 61$; died: mean = −5.5 ± 8.8 g, $n = 11$, $F = 0.17$, $P = 0.68$). The three uninjected males that died had body mass residuals of −3.1, +5.9 and +10.6, respectively, and thus were not in worse body condition at the time of capture than other males removed. The uninjected female had a body mass residual of −30.4.

Net loss of body mass in captivity (i.e., mass at capture − mass at release) for injected males was related to survival; 60 males that survived lost an average of 76.1 ± 6.5 g and nine that died lost an average of 148.3 ± 22.4 g ($F = 14.7$, $P = 0.0003$). Data for injected females in captivity were limited but showed a similar pattern to males; two females that died lost an average of 127.5 ± 37.5 g and 10 females that survived lost a mean of 36.0 ± 16.8 g. We categorized net mass loss by 50 g increments to ascertain how the amount of mass lost might influence survival. Although we observed overall heterogeneity in percent survival with net mass loss ($G_{ran} = 20.3$, df = 5, $P = 0.001$), sequential contingency tests of adjacent net mass loss categories showed that survival in captivity did not decline until birds had lost more than 150 g (i.e., about 25% of their body mass, $G_{ran} = 18.9$, df = 1, $P < 0.0001$, Fig. 1).

Although loss of body mass in captivity did not influence survival until birds had lost more than 150 g, the rate of mass lost, especially during the first week in captivity was related to survival. Birds that died in captivity lost a greater percentage of their initial body mass during the first week in captivity than survivors (mean % mass loss during week 1: died—22.5 ± 0.03, range 8–37%, $n = 11$; lived—14.0 ± 0.01, range 2–32%, $n = 58$). Although there was extensive overlap in percent of mass lost in both groups, birds that died in captivity lost body mass at a faster rate during the first week in captivity than birds that lived (Fig. 2).

Of the three injection routes used, only the 0.2 mg intramuscular injection resulted in a significant reduction from 100% survival (Table 1a). Thus, we restricted our analysis of survival in relation to morphometric traits, date of capture and mass loss in transport to males given intramuscular injections of 0.2 mg ECP (Table 2). Survival of birds given 0.2 mg ECP did not differ in the three years ($G_{ran} = 4.8$, df = 2, $P = 0.10$), so years were pooled for further analysis. No relationship was found between survival and adjusted body mass at capture, wing chord, or amount of mass lost during transport to the camp (Table 2). However birds that died were removed significantly earlier in the season (Table 2).

Survival of captive birds to the subsequent year. We examined whether captivity influenced the return of released birds to the study site in subsequent years. During 1981 to 1983, 57% of 49 removed males and 57% of 123 unmanipulated paired males returned to the study area the next year ($G = 0.0008$, df = 1, $P = 0.98$); 60% of 10 females placed in captivity returned, compared to 38% of 125 unmanipulated hens ($G = 1.8$, df = 1, $P = 0.18$). We found no relationship between likelihood of returning and the three injection routes; likewise, return rates did not vary with the sex or age class of removed birds (Table 1b). Net mass loss in captivity of ptarmigan that lived and returned the next year was 68.1 ± 7.9 g ($n = 45$), while individuals that did not return in subsequent years had lost an average

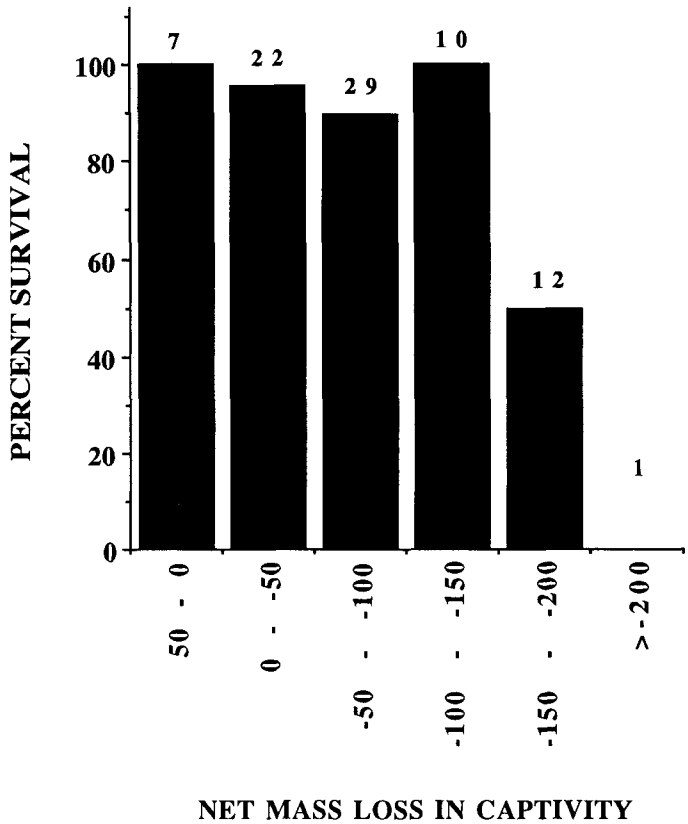

FIGURE 1. Survival in captivity in relation to net mass loss (50 g increments) by Willow Ptarmigan injected with estradiol cypionate (ECP) at La Perouse Bay, Manitoba, 1981–1983.

of 74.4 ± 10.4 g ($n = 25$, $F = 0.23$, $P = 0.63$). Hence, if birds survived captivity, they suffered no reduction in survival after their release.

DISCUSSION

Injections of estradiol cypionate (ECP) strongly enhanced survival of Willow Ptarmigan in captivity. Similar responses were observed for both sex and age classes. Injections of ECP also had the advantage of showing no subsequent effects on survival after birds were released. Injected birds that survived captivity had a similar probability of returning the following year as ptarmigan that were not removed.

Only birds given 0.2 mg intramuscular injections had significant mortality, and within this group, there was an interaction with date of capture. Ten of 11 males that died after being given 0.2 mg intramuscular injections were removed during the first half of the experimental period (mid to late June). This mortality may have been due to increased stress occurring earlier in the breeding season when males were still involved in energetically demanding activities. In our study, we removed birds well after the seasonal peak of testosterone production associated with the acquisition of territories and mates (Hannon and Wingfield 1990), but birds would still have had levels well above post-breeding condition. As the removal period progressed, decreasing levels of testosterone may have facilitated the ability of males to adjust to captivity. If so, we predict that mortality rates would be higher for birds removed before onset of incubation.

Date of capture was not the principle cause for mortality in captivity, otherwise we would have observed mortality among all injection regimes for birds removed early in the season. Only 1 bird died after an intramuscular injection of 0.1 mg, and it was removed late in the experimental period. No mortality occurred after combination injections of 0.2 mg. In addition, all uninjected birds were removed in the latter half of the experimental period.

The 0.2 mg intramuscular injections resulted in lower survival than the other two injection routes. Jones et al. (1967) administered subcutaneous injections of estrone and 17β-estradiol in corn oil and dimethyl sulfoxide (DMSO) at dosages of 2, 4, and 8 mg/kg/day to 6½ week old cockerals (*Gallus domesticus*). They found that injections in corn oil were absorbed into the blood more slowly than those using DMSO as the vehicle. Also, accumulations of oil were found at the injection site three days after administration. Presum-

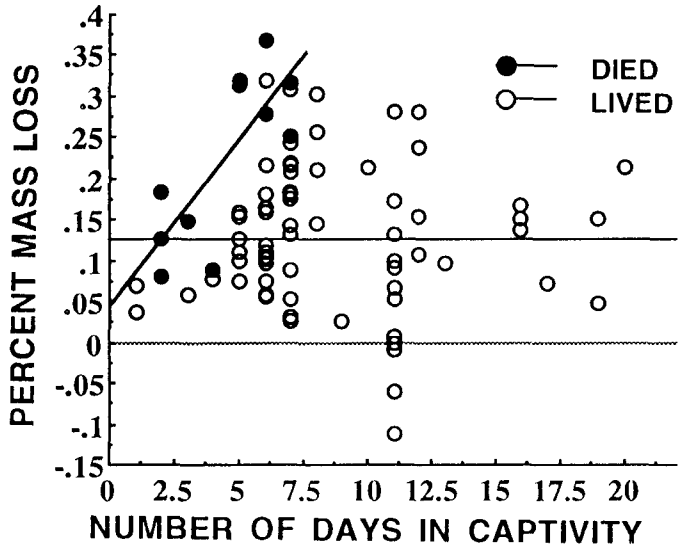

FIGURE 2. Percent loss of body mass for Willow Ptarmigan injected with estradiol cypionate (ECP) in relation to survival during captivity at La Perouse Bay, Manitoba, 1981–1983. Died: $r^2 = 0.60$, $P = 0.005$; $Y = 0.04X + 0.44$, $n = 11$ (9 males, 2 females) Lived: $r^2 < 0.0001$, $P = 0.99$; $Y = -0.00002X + 0.13$, $n = 69$ (60 males, 9 females).

ably the accumulated oil contained portions of unabsorbed estrogens. If Willow Ptarmigan also experienced low ECP absorbance at the subcutaneous injection site then the 0.1 mg intramuscular and 0.2 mg combined injections may have resulted in equivalent doses of ECP. An injection of 0.1 mg subcutaneous might have resulted in a mortality rate similar to uninjected birds. Since we did not give 0.1 mg subcutaneous only injections, we cannot evaluate the possibility that the differential survival observed for injections of 0.2 mg intramuscular and 0.2 mg combined may be explained by the slow absorption of ECP subcutaneously. Because the dosages we used were low compared to other studies involving estradiol injections (Jones et al. 1967, Balnave 1971, Balthazart and Deviche 1977, Bruce and Anastassiadis 1977, Rosebrough et al. 1982), we would not have predicted that birds given ECP injections of 0.2 mg intramuscular would have lower survival than those given 0.1 mg intramuscular. Wild birds may be more sensitive than domestic species to hormone administrations.

Body mass at capture and net mass losses of less than 150 g were unrelated to survival in captivity. However during the first week in captivity, ptarmigan that died lost a greater percentage of their body mass more rapidly than survivors. Perhaps birds that died were unable to reverse the trend for rapid mass loss in captivity. Pendergast and Boag (1971) also found Spruce Grouse (*Canachites canadensis*) that died in captivity lost mass quickly before death. Given the relationship shown in Figure 1, and the lack of relationship between survival and initial body condition of birds at the time of removal, it is most likely that rapid loss of body mass did not cause the mortality of ptarmigan directly, but was perhaps a consequence of other lethal factors.

Possible mechanisms whereby ECP improves survival in captivity. Our study demonstrates that injections of 17β-estradiol in cottonseed oil (ECP) greatly improved survival of Willow Ptarmigan in captivity. Given the goals of our study and the remoteness of the field site, we were not able to determine the physiological and endocrinological mechanisms involved. However, we can present several suggestions as to how ECP might have enhanced survival of Willow Ptarmigan in captivity. In birds, lipids are an important energetic fuel and the liver contributes significantly to the synthesis of fatty acids (Goodridge and Ball 1967, Griminger 1986). Estrogen compounds enhance hepatic lipid synthesis in birds (Griminger 1986) and treatments with estrogens not only increase the overall mass of the liver, but also increase liver and blood lipid levels (Balnave 1971, Bruce and Anastassiadis 1977, Rosebrough et al. 1982). As well, Jackson et al. (1971) found a five fold increase in blood linoleic acid and non-essential fatty acids after administration of estradiol to immature female chickens. Balnave (1971) found similar results with immature pullets. If this occurs in Willow Ptarmigan, then injections of 17β-estradiol could have had a compound effect by enhancing lipid synthesis and increasing essential fatty acids (EFAs) levels in the blood.

Essential fatty acids (EFAs) must be obtained in the diet and one of the principal EFAs for Willow Ptarmigan is linoleic acid (West and Meng 1968, Balnave 1971, Jackson et al. 1971, Griminger 1986). Cottonseed oil (the 17β-estradiol carrier for ECP) contains various fatty acids and glycerides composed chiefly of palmitic, oleic and linoleic acids (Whiteley 1949, Sax and Lewis 1987). Evans et al. (1977) fed laying domestic hens diets containing crude cottonseed oil and

observed increases of linoleic acids in the plasma. Thus, cottonseed oil might have produced similar effects by providing injected birds with a source of readily available EFAs in the blood.

Captivity may result in modified lipid metabolism in several ways. During initial captivity, lipid stores may be reduced rapidly due to increases in metabolic rate associated with the stress of capture and removal. Thus, an ECP injection might have a dual positive effect by providing captive birds with enhanced lipid metabolism and a source of EFAs and readily available triglycerides from the cottonseed oil to be used as fuel during initial adjustment to captivity. Willow Ptarmigan do not have large amounts of depot fat at any time (West and Meng 1968, Myrberget and Skar 1976, Thomas 1982), and thus may be prone to EFA deficiency when under stress.

Injections of ECP could decrease aggression and consequently decrease stress and the levels of associated glucocorticoids. Lorenz (1954) observed decreased aggression and cessation of fighting in domestic fowl after administration of estrogens and attributed this to an indirect decrease in androgen production. A reduction in stress reduces metabolic costs associated with stress, possibly allowing birds to remain calm and shunt energy to adjusting to captivity. To utilize this technique more effectively, future research should be directed at determining whether survival in captivity is enhanced primarily due to the estradiol, the cottonseed oil, or whether these function in a cooperative manner. If the vehicle (cottonseed oil) can enhance survivorship alone, it may be possible to eliminate the steroid altogether, thus avoiding other potential complications of hormone administration.

From an ecological and ethical point of view it is desirable that captive animals from removal studies are returned to their environment and that effects of captivity are minimized. Our technique of injecting low dosages of ECP (17β-estradiol in cottonseed oil) greatly enhanced survival in captivity for Willow Ptarmigan, and may have similar effects for other bird species that are difficult to maintain in captivity. These injections are easy to perform in the field and appear to lack hazardous long-term effects. This technique has potential utility for field researchers, wildlife management agencies and conservation institutes concerned with the temporary maintenance of wild birds in captivity.

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DOES BROWN-HEADED COWBIRD EGG COLORATION INFLUENCE RED-WINGED BLACKBIRD RESPONSES TOWARDS NEST CONTENTS?[1]

JOSEPH C. ORTEGA,[2] CATHERINE P. ORTEGA[2] AND ALEXANDER CRUZ
Department of Environmental, Population, and Organismic Biology, University of Colorado, Campus Box B-334, Boulder, CO 80309-0334

Key words: Brown-headed Cowbird; Red-winged Blackbird; Molothrus ater; Agelaius phoeniceus; *accepters; rejecters; brood parasitism; Colorado.*

In North America, the Brown-headed Cowbird (*Molothrus ater*) is known to parasitize 221 species (Friedmann et al. 1977, Friedmann and Kiff 1985). In Colorado, Brown-headed Cowbirds parasitize Red-winged Blackbirds (*Agelaius phoeniceus*) to varying degrees depending upon year, location, and habitat (Hanka 1979, Ortega and Cruz 1988, Ortega 1991).

Brood parasites often are detrimental to the reproductive efforts of their hosts; therefore, natural selection should favor host behaviors that deter brood parasitism (Rothstein 1975, 1990; Rohwer and Spaw 1988). Such behaviors or defense mechanisms include: (1) aggression toward adult parasites, (2) abandoning parasitized nests, (3) constructing a new nest over parasitized clutches, (4) puncturing parasitic eggs, and (5) removing parasitic eggs (Rothstein 1975, 1990; Ortega and Cruz 1988; Rohwer and Spaw 1988). Although brood parasitism by Brown-headed Cowbirds can significantly decrease the reproductive output of host species (Mayfield 1965, Walkinshaw 1972, Marvil and Cruz 1989, but see Weatherhead 1989, Ortega 1991), Red-winged Blackbirds are nevertheless an "accepter" species of cowbird eggs and also readily accept cowbird egg models (Rothstein 1975, Ortega and Cruz 1988). That is, in contrast to "rejecter" species (see Rothstein 1975), "accepter" species are those species in which nearly all individuals accept eggs that are nonmimetic (Rothstein 1975).

Examining how various parameters (e.g., egg shape, size, color, and maculation) influence responses towards cowbird eggs is basic to understanding the evolution of host-parasite interactions and could aid in the potential management of cowbirds. Ortega and Cruz

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[2] Present address: Department of Biology, Fort Lewis College, Durango, CO 81301-3999.