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(*Peromyscus Maniculatus*)**

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EFFECTS OF THE PARASITE *EIMERIA ARIZONENSIS* ON SURVIVAL OF DEER MICE (*PEROMYSCUS MANICULATUS*)¹

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Abstract. Few experimental studies have documented the negative effect of parasites on fitness of hosts under natural or seminatural conditions. We studied the effect of *Eimeria arizonensis* (Protozoa) on recruitment, winter survival, and change in body mass in deer mice (*Peromyscus maniculatus*). In field observations, the presence of *E. arizonensis* was negatively related to recruitment in females and to over-winter survival in males. Experimental manipulation of *E. arizonensis* in large field enclosures also showed that infection negatively affected over-winter survival of males. There was no relationship between the body mass of deer mice and the presence of either natural or experimental infection. Thus, the mechanism through which *E. arizonensis* affects survival remains unclear.

Key words: deer mice; *Eimeria arizonensis*; host–parasite interactions; host survival; *Peromyscus maniculatus*.

INTRODUCTION

A major goal of ecology is to understand the factors that affect the distribution and abundance of animal populations (Krebs 1994). Sinclair (1989) defined a limiting factor as any factor that causes changes in reproduction or survival, whereas regulating factors must be density dependent. The factors that have received the most attention by ecologists include birth rate, emigration, predation, and competition (Sinclair 1989). However, mathematical models and laboratory studies have suggested that parasites and other pathogens may also be important in determining the population densities of animals (Anderson and May 1979, May and Anderson 1979, Scott and Anderson 1984, Scott 1987).

Empirical studies of parasites on host populations are desirable, but are often logistically difficult. Thus, most researchers have examined the effects of parasites on the fitness of individual hosts. There is ample correlative evidence from the field that parasites from many taxa (e.g., protozoans, nematodes, trematodes, and arthropods) have a negative impact on reproduction and/or survival in a variety of hosts, including fish (McPhail and Peacock 1983), reptiles (Schall 1983), mammals (Festa-Bianchett 1989), mollusks (Goater et al. 1989, Lim and Green 1991), insects (Arnqvist and Maki 1990, Burgett et al. 1990), and birds (Johnson et al. 1991). Ideally, controlled field experiments should be conducted to examine negative associations between parasitism and host fitness. However, experimental manipulations of parasite levels under field conditions have been accomplished in only a few host–parasite

systems (e.g., Hudson 1986, Hudson et al. 1992a, b, Lehmann 1993, Møller 1993).

Scott and Dobson (1989) suggested that directly transmitted parasites (i.e., without intermediate hosts) that have known pathology would be the most likely to have a detectable effect on hosts. Many eimerians (intestinal protozoans in the class Coccidia) meet these criteria. They are transmitted directly through ingestion of contaminated feces and are known to be highly pathogenic in poultry and livestock (see Long 1982). We studied the effect of *Eimeria arizonensis* on survival of individual deer mice (*Peromyscus maniculatus rubidus*) using both field observations and field and enclosure experiments.

Eimeria arizonensis infections last 10–12 d, and up to 10⁷ oocysts are shed in the feces during this period (Fuller et al. 1995). Because infections are of relatively short duration, are easy to detect using noninvasive methods, and individual hosts become infected repeatedly, this system is ideal for examining the dynamics of infections and the effect of infections on individual hosts (Fuller 1994). Deer mice are ideal host subjects because they can be readily trapped, recaptured, and sampled for parasites in the field.

Because many eimerians are pathogenic, we hypothesized that *E. arizonensis* would have a negative impact on survival of deer mice. Although survivorship is difficult to measure in small mammals under field conditions, length of residency in a population has frequently been used as a measure (e.g., Boonstra et al. 1980, Lehmann 1992). We predicted that recruitment and over-winter survival of deer mice would be adversely affected by *E. arizonensis*. We tested effects on recruitment by observing whether free-living, unmanipulated animals that were infected when they first entered a population were less likely to become residents than animals that were uninfected when they entered a population. We tested effects on over-winter

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survival in two ways: first, we determined whether animals that did not reappear in spring (and presumably died over the course of the winter) had more frequent natural infections the previous year than animals that reappeared in spring, and second, we conducted experimental infections in field enclosures and compared survivorship in control and treatment plots.

Eimeria arizonensis does not cause obvious pathology in deer mice, i.e., it is not possible to determine whether an animal is infected by visual inspection. We hypothesized that *E. arizonensis* might influence long-term survival by causing mass loss and tested this hypothesis with data from free-living deer mice and from experimental infections in natural and enclosure populations.

MATERIALS AND METHODS

Observations of free-living deer mice in natural populations

A 10 × 12 trapping grid was established in MacDonald Forest (15 km northwest of Corvallis, Benton County, Oregon; elevation 500 m) in March 1990. Sherman live traps were spaced 10 m apart in 1990. Traps were spaced 15 m apart in 1991 and 1992 to increase sample size without increasing trapping effort. Traps were set for two to three consecutive nights at 2–3 wk intervals from 15 April–17 November 1990, 30 March–17 November 1991, and 7 March–12 July 1992. All deer mice were marked individually at first capture and released each morning at the point of capture. Animals were sexed, aged (based on pelage; juveniles were 21–60 d, adults were >60 d), weighed, and adult females were assessed for reproductive condition. Recapture data were used to test whether *E. arizonensis* affected deer mouse recruitment and survival.

In addition to the mark–recapture population, 6–35 (\bar{X} = 19.7) deer mice were trapped monthly from April–September 1990, March–September 1991, and June–July 1992. These animals were captured at different sites in MacDonald Forest each month and (with the exception of two sites) were at least 1 km from the mark–recapture site. We used the same methods and collected the same data as for the mark–recapture animals. However, these animals were killed and examined for helminthes (C. A. Fuller, unpublished data). Mass of these deer mice and mass of each mark–recapture animal at first capture were analyzed for the effect of *E. arizonensis* on body mass in natural populations. Thus, for these analyses, each animal was used only once.

Feces from all deer mice were collected from traps, weighed, and incubated in K₂Cr₂O₇ solution (2.5% w/v) at room temperature for 1 wk. This procedure facilitates the development of *Eimeria* oocysts into the infective or sporulated stage and is necessary for identification to species. Fecal samples were macerated and examined

for the presence of oocysts by sugar flotation (Ash and Orihel 1987). Soiled traps were washed in hot water before they were returned to the field. In 1990 and 1991 traps were also heat sterilized.

Experiments

Eimeria arizonensis cultures and infection levels.—*Eimeria arizonensis* oocysts were obtained from the feces of two deer mice from the MacDonald Forest population in spring 1990. Oocysts were stored at 4°C and fresh oocysts were generated periodically using laboratory-born, *Eimeria*-free deer mice that also originated from the MacDonald Forest population. Oocysts were generated 6 d–4 mo before the initial inoculations, sporulated at room temperature, and refrigerated until used.

We do not know how many oocysts animals ingest under natural conditions. However, Fuller et al. (1995) documented that a deer mouse fecal pellet (produced by animals inoculated with ≥10 000 oocysts) contained as many as 15 000 oocysts. We inoculated enclosure animals with 50 000 and free-living animals with 20 000 oocysts at each inoculation (see *Enclosure and field experiments*).

The ratio of prepatent (after ingestion but before oocysts are shed) to patent period (during which oocysts are shed) is 4:8 d in *E. arizonensis*; thus, it is not possible to determine with certainty which animals had natural infections at the time of the experimental inoculations. Therefore, we randomly assigned animals to treatment and control groups in all experiments but did not assess whether they were shedding oocysts at inoculation.

Enclosure experiment.—Free-living deer mice were live-trapped and placed in four 50 × 50 m (two treatment and two control) field enclosures in the fall of 1993. There were 100 live traps (10 × 10 grid with 5-m spacing) in each enclosure and each enclosure was surrounded by a 1 m high, corrugated sheet-metal fence above ground and a 0.5-m fence below ground. Thus, animals were exposed to aerial but not terrestrial predators and could not escape from enclosures. The dominant vegetation was alfalfa; deer mice were not given water or additional food but were provided with nest boxes (20 in each enclosure). Animals in treatment enclosures were inoculated with oocysts in water, and animals in control enclosures were inoculated with water one to three times at 5-wk intervals. The majority of animals were first inoculated in September 1993 (55 adults and 15 juveniles), given a second infection in October 1993, and a third infection in November 1993 at 5-wk intervals. A second group of animals was first inoculated in October 1993 (18 adults and 4 juveniles) and given a second infection in November. Animals were trapped as in field studies and fecal samples were collected directly from the animals. Fecal samples were weighed to the nearest 0.01 g and oocysts were quantified by counting subsamples of known volume in a

MacMaster's counting chamber (Weber Scientific International, Limited, Sussex, England; Dunn and Keymer 1986).

Field experiment.—Adult male deer mice from the mark-recapture population were used to examine the effect of *E. arizonensis* on mass change after the observational study had ended. In addition, a second mark-recapture grid was established ≈ 1.5 km from the first. Animals were lightly anaesthetized with halothane and inoculated by stomach intubation two times at 3-wk intervals (October and November 1992) or once (November 1992). Animals were released within 1 h of inoculations and were free living throughout the experiment. Traps were set overnight on days 5 or 6 post-inoculation (PI), i.e., days of peak oocyst output for *E. arizonensis* (Fuller et al. 1995), and animals were weighed and released the following morning. Feces from traps were weighed, and oocysts were quantified as in the enclosure experiment.

Statistical analysis

To test for effects of parasites on recruitment, we compared the number of animals that remained in the population and those that disappeared with a three-way Mantel-Haenszel χ^2 analysis (factors = sex, infection status, and year). This test factors out the effect of year on infection rate (Wilkinson 1990). To test for effects on over-winter survival, data from field observations within each year were compared by Mann-Whitney *U* test. Data from the enclosure experiments were compared by ANOVA (N = two treatment and two control enclosures). Mass change in naturally infected and uninfected animals was compared by regression analysis. We tested the significance of season and parasites with an ANCOVA, where the covariate was the quadratic parameter "date." This parameter reflected mass increases early in the trapping season and mass decreases late in the trapping season. Because some groups of animals (e.g., lactating females) were only present during a few months, seasonal mass change may not have been important in those groups. Thus, if the *P* levels of seasonal parameters were >0.10 , the parameters were not included and a Student's *t* test was used. Data sets from adult males, nonreproductive, pregnant, and lactating females and juveniles were analyzed separately. For experiments on mass change (enclosure and field), differences in mass change between parasitized and unparasitized animals were compared by Student's *t* test. Differences among enclosures within treatments were not significant; therefore, we pooled data within treatments. Data are presented as means ± 1 SE. In all tests, one-tailed probabilities are given unless stated otherwise. Alpha levels of 0.05 were considered to be statistically significant.

RESULTS

Establishment of residency

Field observations.—Deer mice were considered to be residents if they were present in at least four trapping

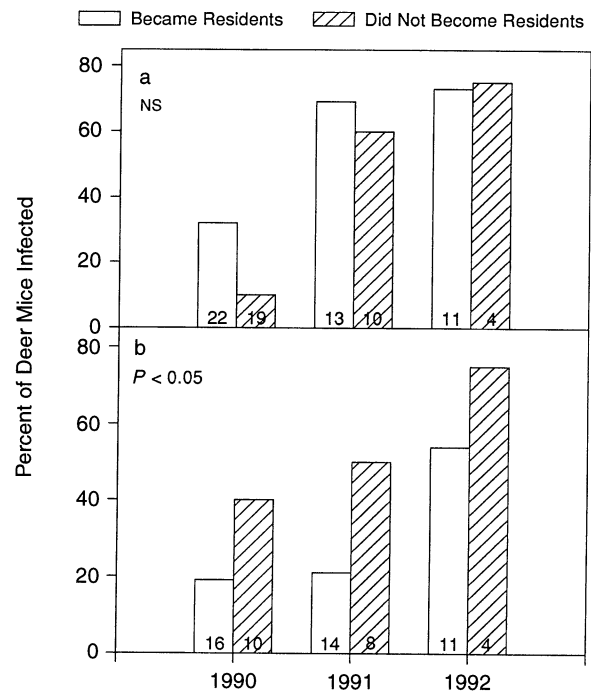


FIG. 1. The frequency of *Eimeria arizonensis* infections among newly marked males (a) and females (b) that became residents or did not become residents in the mark-recapture population. Numbers in columns are sample sizes.

sessions (a minimum of 6 wk). Males that became residents in the population were infected during their first captures as frequently as males that did not become residents (Fig. 1a; Mantel-Haenszel $\chi^2 = 1.841$, $df = 1$, $P > 0.10$). In contrast, females that became residents were infected significantly less often when they first appeared than females that did not become residents (Fig. 1b; Mantel-Haenszel $\chi^2 = 3.566$, $df = 1$, $P < 0.05$).

Over-winter survival

Field observations.—Only residents (see *Establishment of residency*) that were present in one of the last two trapping sessions of each year were included in this analysis. Males that survived the winter had been infected significantly less frequently the previous year than males that disappeared. This pattern was clear in the spring of both 1991 and 1992 (Fig. 2; 1990–1991: $U = 36.5$, $N = 14$, $P < 0.05$; 1991–1992: $U = 12$, $N = 7$, $P = 0.05$). Females that survived the winter of 1990 were not infected less frequently than females that disappeared (Fig. 2; $U = 17$, $N = 14$, $P > 0.2$). There were not enough resident females during the last two trapping sessions of 1991 to conduct a statistical analysis ($N = 4$). However, the mean proportion of infections is presented in Fig. 2b for comparison.

Enclosure experiment.—As in field observations, males in treatment populations were significantly less likely to survive the winter than males in the control

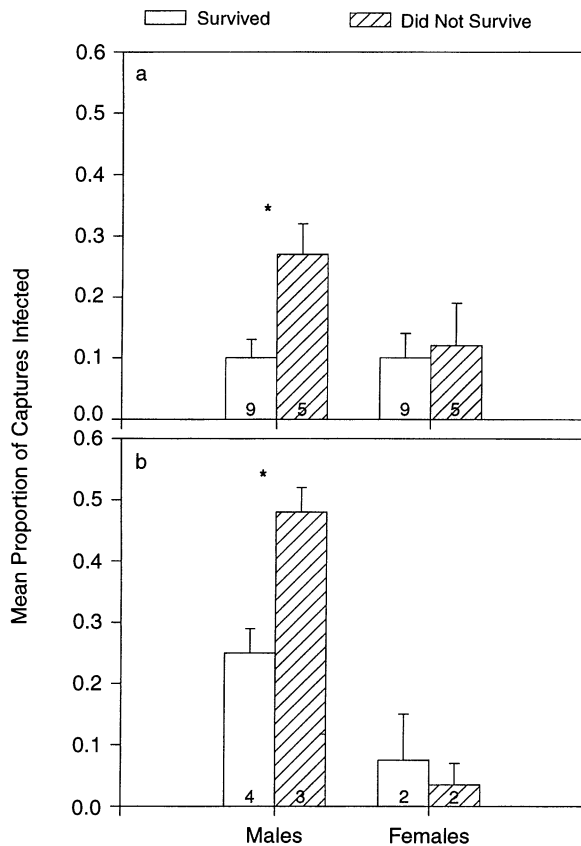


FIG. 2. Proportion (mean + 1 SE) of captured free-living deer mice infected with naturally occurring *Eimeria arizonensis* in 1990 (a) and 1991 (b), shown separately for mice that survived and did not survive the following winter. Numbers in columns are sample sizes. The asterisks indicate significant differences at the 0.05 level.

populations (ANOVA: $F = 40.65$; $df = 1,2$; $P < 0.025$). There were no significant differences in survival between control and treatment populations for females ($F = 0.004$; $df = 1,2$; Fig. 3).

Examination of feces after experimental inoculations revealed that in 69 of 70 treatment inoculations, infected animals shed *E. arizonensis* oocysts on days 5–

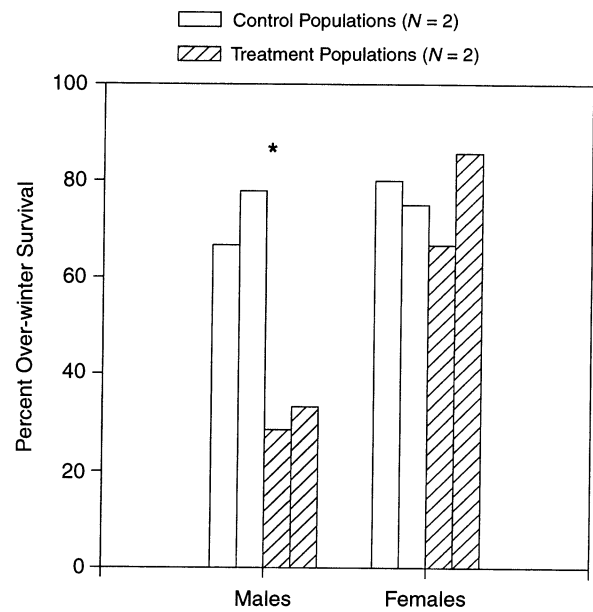


FIG. 3. Percent of population that survived the winter in control and treatment enclosure populations. Control deer mice were sham inoculated with water and treatment deer mice were inoculated with 5×10^5 sporulated oocysts one to three times in the fall. * indicates significant differences at the 0.05 level; $N = 2$.

6 PI, whereas animals shed oocysts in only 5 of 67 sham inoculations. Moreover, treatment animals generally shed $>10\,000$ oocysts per gram feces, whereas all 5 sham-inoculated animals shed <1000 oocysts per gram feces.

Changes in mass associated with E. arizonensis infection

Field observations.—Parasitized lactating females weighed significantly less than uninfected lactating females. There were no significant differences in mass between infected and uninfected animals in any other group (Table 1).

Enclosure experiment.—There were no significant differences in mass change for adult males or for non-

TABLE 1. Mass of infected and uninfected deer mice in field observations. In analyses where quadratic parameters were included to account for seasonal variation in mass (adult male, pregnant female, and nonreproductive male), mass = estimated mass. For these analyses, the reported SE are too low because masses were estimated in groups, thus artificially decreasing variances.

	Infected		Uninfected		Student's <i>t</i>
	Mass (g) ($\bar{X} \pm SE$)	<i>N</i>	Mass (g) ($\bar{X} \pm SE$)	<i>N</i>	
Juvenile	12.3 ± 0.6	14	11.6 ± 0.2	111	1.101
Adult male	17.3 ± 0.1	56	17.2 ± 0.1	115	0.230†
Pregnant female	19.1 ± 0.2	9	20.1 ± 0.1	27	1.147†
Lactating female	16.2 ± 0.6	6	18.2 ± 0.6	18	1.803‡
Nonreproductive female	15.2 ± 0.2	16	16.2 ± 0.1	54	1.370†

† Partial *t* values generated by SYSTAT (Wilkinson 1990).
‡ One-tailed $P < 0.05$.

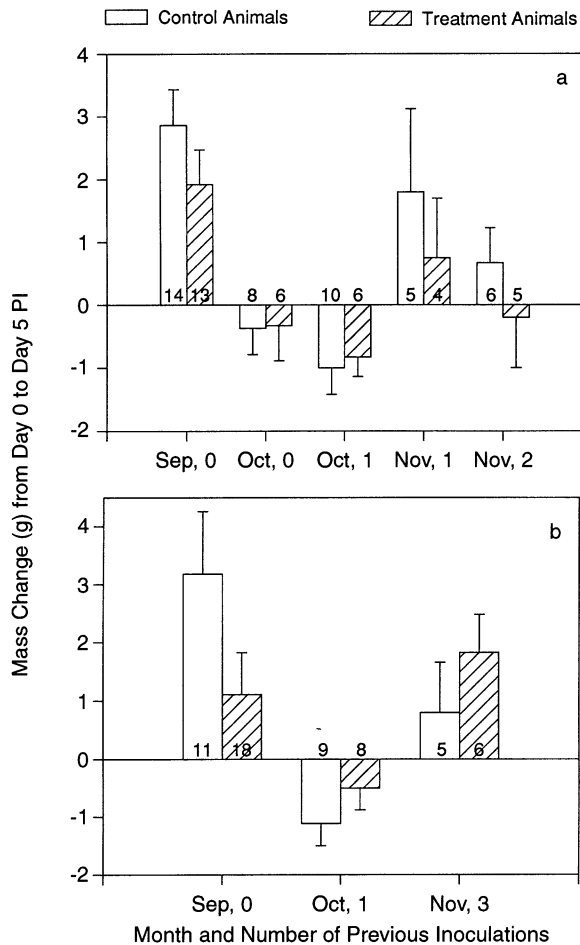


FIG. 4. Mass change from Day 0 to Day 5 post-inoculation (PI) of enclosure deer mice inoculated with water (control) or 5×10^5 sporulated *Eimeria arizonensis* oocysts (treatment) one to three times. Error bars show 1 SE. There were no significant differences in mass change for either males (a) or females (b) in any month and regardless of the number of times inoculated. Numbers at the base of columns are sample sizes.

reproductive adult females in the enclosure experiment whether mass was analyzed separately or pooled by month (i.e., ignoring the number of times animals had been inoculated; Fig. 4a, b). The sample size of other age/sex classes was insufficient for analysis.

Field experiment.—There were no significant differences in mass change from inoculation to day 5/6 PI between control and treatment animals in October (\bar{X} change = -0.15 ± 0.4 g for parasitized and -0.37 ± 0.2 g for unparasitized males; $t = -0.49$, $df = 20$) or in November (\bar{X} change = -2.18 ± 0.4 g for parasitized and -0.75 ± 0.6 g for unparasitized males; $t = -1.50$, $df = 21$) 1992.

Examination of feces after experimental inoculations revealed that in all 29 treatment inoculations, infected animals shed *E. arizonensis* oocysts, whereas only 5 of 16 sham-inoculated animals shed oocysts. Moreover,

all animals inoculated with *E. arizonensis* shed $>10\,000$ oocysts/g feces, whereas 4 of the 5 sham-inoculated animals shed <1000 oocysts/g feces.

DISCUSSION

We tested three predictions of the hypothesis that *E. arizonensis* has a negative impact on deer mouse survival. As predicted, we found that the frequency of infection was negatively associated with two measures of survivorship: establishment of residency and over-winter survival. However, males and females were not affected in the same way. Females infected with naturally occurring *E. arizonensis* when they first appeared in the population were less likely to become residents than females that were uninfected, but we did not detect a similar trend in males. On the other hand, once a male became a resident, the frequency with which he was infected over the remainder of the year was negatively associated with over-winter survival both in naturally occurring and experimental infections. In this case, we did not detect a similar trend in females. Sample sizes in our studies of over-winter survival were small in both field observations and enclosure experiments, but differences were significant in all cases. This suggests that *E. arizonensis* strongly affects survival.

Although it is common for males and females of a host species to have different rates of infection (Alexander and Stimson 1988, Bundy 1988), it is unclear why *E. arizonensis* would affect different aspects of deer mouse life history. In the study of recruitment, females that did not persist were infected as frequently as males, whether the males persisted or not. Females that *did* persist were infected less frequently than other animals. This pattern suggests that new females may be more affected by infections than new males. However, dispersal among males is common. It may be that frequent movement among males swamped the effect of the parasite. In observations of over-winter survival, males that did not reappear in spring were infected more frequently than all other animals. Although males and females in the enclosure experiment were inoculated with equal frequency, it is not known how often they became reinfected between November (the last experimental inoculation) and March (collection). Thus, the probability of surviving the winter could be a function of the number of times animals were infected.

Several studies of vertebrate hosts have found negative associations between host survivorship and parasites in naturally occurring infections (Boonstra et al. 1980, Hibler et al. 1982, Ross et al. 1989, Arnqvist and Maki 1990, Gulland and Fox 1992, Hudson et al. 1992b, Lehmann 1992). However, other observational studies have not found any correlation between parasitism and host survivorship (Goater et al. 1989, Moss et al. 1990, Johnson et al. 1991). Keith et al. (1986) found evidence that only one of six parasites examined

affected snowshoe hare (*Lepus americanus*) survival. Of the few controlled field experiments to test the effect of parasites on host survival, some (Hudson et al. 1992a, Lehmann 1992, Trout et al. 1992) concurred with earlier observations of negative effects, whereas others did not (Samson et al. 1987).

Eimeria arizonensis does not cause obvious pathology in deer mice. Therefore, we examined mass loss as a mechanism through which *E. arizonensis* might influence deer mouse survival. We observed significant mass differences between parasitized and unparasitized lactating females, but in no other groups. Because females use more energy during lactation than during any other part of the reproductive cycle (Bronson 1989), they may be especially susceptible to adverse effects of parasites during this period. There also were no differences in mass change between treatment and control animals in any experimental infections. Unfortunately, experiments took place too late in the year to include reproductive females.

Other investigators have examined mass loss as a possible mechanism for parasite-induced reduction in survival and/or reproductive success in birds and mammals (Moore and Bell 1983, Keith et al. 1986, Howe 1992, Lehmann 1992, Booth et al. 1993). Of these, only Booth et al. (1993) studying Rock Doves (*Columba livia*) and Keith et al. (1986) studying snowshoe hares (*L. americanus*) found significant negative associations between mass change and parasitism. Others have examined changes in metabolic rate (Munger and Karasov 1989, Booth et al. 1993), organ pathology (Gellar and Christian 1982, Watkins et al. 1991), and blood abnormalities (Wiger 1977).

None of the studies discussed above addresses the direct mechanism of host mortality. Among other things, parasitized animals may starve, or be cannibalized or eaten more readily than unparasitized animals (Wiger 1977, Temple 1987, Hudson et al. 1992b). In our study, it is unclear which factors affected host mortality. Parasitized females recruited into the population less frequently than unparasitized females. Because female *Peromyscus* generally settle in or near their natal home range (Wolff and Lundy 1985, Wolff et al. 1988), it seems unlikely that females in our study dispersed and more likely that they died. In winter deer mice were typically 20–25% lighter than at other times of the year (C. A. Fuller, *personal observation*) indicating possible nutritional stress in over-wintering animals. However, some of this loss may be due to seasonal changes in reproductive condition (Millar 1989; C. A. Fuller, *personal observation*). Predators were observed both in the field (short-tailed weasels, *Mustela erminea*) and near enclosures (falcons and hawks). It is possible that parasitized deer mice were less able to avoid predation than unparasitized animals due to poorer general condition (Wiger 1977, Scott 1988, Lefcort and Eiger 1993). Adverse weather may enhance the negative effects of parasites (de Lope et al. 1993).

It is becoming increasingly clear that a wide variety of parasites negatively affect survival of free-living hosts. In addition, several observational and experimental studies have documented negative effects of parasites on host reproduction (Schall 1983, Durnin 1993, Lafferty 1993, Møller 1993). In some cases, these factors appear to be density dependent (Scott 1988). Thus, empirical evidence suggesting that parasites in natural populations have the potential to affect host abundance is beginning to accumulate. Field experiments at the level of the host population are needed to determine whether the effects on reproduction and survival act in a density-independent, a density-dependent compensatory manner, or to regulate host abundance.

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