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Effects of parasitic infection on mate sampling by female wild turkeys (*Meleagris gallopavo*): should infected females be more or less choosy?

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Investigations of parasite-mediated sexual selection have concentrated on the effects of parasites on males. Differences in female susceptibility to parasitic infection may also cause variation in reproductive behavior. I propose two alternative hypotheses to explain how infected females may alter their mate sampling behavior. In the first hypothesis, infected females sample fewer prospective mates because chronic parasitic infection imposes energetic costs that limit the time and calories that a female can expend in mate searching. A novel alternative hypothesis is that females recognize their own susceptibility to infection and thus invest more time searching for a male phenotype that indicates he offers genes complementary to her genome. In recombination, these good genes would allow her offspring to better resist parasites despite their mother's susceptibility. I examined the mate sampling behavior of experimentally infected wild turkey hens when presented with an array of males, and compared them to control hens. Infected females did not invest more time assessing individuals, did not wait longer to choose a male, nor were they less likely to solicit during the trial. They did differ from control females in that they visited more males before soliciting copulation and exhibited different preference functions for snood length. These results suggest that females are not so energetically restricted by latent coccidia infection that they must hurry to find a mate. Instead, it appears that infected females assess a larger set of males as prospective mates, perhaps to increase the opportunity to obtain complementary genes for parasite resistance. *Key words:* mate sampling, sexual selection, parasitism. *[Behav Ecol 15:687–694 (2004)]*

Investigations of how female parasitism might cause variation in female choice are rare (Lopez, 1999; Pfennig and Tinsley, 2002; Poulin, 1994; Simmons, 1994) relative to the numerous studies of the effects of male parasitism on mating success (for review, see Møller et al., 1999). A general conclusion from female parasitism studies is that the costs of infection reduce choosiness of females, resulting in random mating with respect to male sexual characters. On the surface, it seems logical to predict that females suffering from deleterious infections might be too weary to assess males thoroughly, thus weakening sexual selection. However, if we accept the notion that healthy females should obtain good genes for parasite resistance from their mates, should not good genes selection be even more important to the mating decisions of females susceptible to parasitism? Although infection imposes costs that may limit female actions, disease also provides information to the female about her own genetic condition. Natural selection may favor infected females that compensate for a genetic "bad job" by finding particular male genotypes that complement theirs such that offspring survival is greater than if she mated randomly.

Mate choice for genetic compatibility differs from typical good genes selection in that individual females should differ in their preferences (Tregenza and Wedell, 2000). With regard to female choice for immune genes, Brown (1997) suggested that females should mate disassortatively to ensure that their offspring are capable of broad immunological surveillance (Doherty and Zinkernagel, 1975). Thus, each female must consider her own genotype before knowing which male genotype is a complementary recombinatorial match. Patterns of female mating in house mice (*Mus musculus*; Potts et al., 1991) and stickleback fish (*Gasterosteus aculeatus*; Reusch et al., 2001) are consistent with this hypothesis. Why then do parasitized females generally appear to choose mates at random? The apparent absence of choosiness in previous studies of this question (Lopez, 1999; Pfennig and Tinsley, 2002; Poulin, 1994) may be a methodological artifact of the dichotomous choice design typical of mate choice experiments (Wagner, 1998). Presumably, wild diseased females have an array of males from which to choose to find the best genetic match. In dichotomous choice experiments, captive females can only choose between two males. Neither of these two males may be a genetically suitable complement to the female's genome, and as a result, she might mate randomly. Zuk et al. (1990) showed a similar effect in paired-choice tests of red junglefowl (*Gallus gallus*), whereby females mate at random when neither male has the threshold ornament value. Clearly, female preference for genetic compatibility is difficult to detect in the absence of genetic data from the female and males, as well as knowledge of the fitness consequences to offspring bearing different allelic combinations. Nevertheless, if one considers other aspects of female mating decisions, such as mate searching strategies, an effect of parasitism on female choice might be detected behaviorally.

Under natural conditions, females may use a number of possible search methods to find a suitable mate (Janetos, 1980). Mate sampling, or the way that females search among males to find one that meets their mating criteria, is thought to be costly (Pomiankowski, 1987) and thus may reflect parasitism. I propose two alternative hypotheses, "reducing immediate costs" and "obtaining complementary genes," to explain how parasitic infection of females may affect female mate sampling in species without paternal care. In the reducing immediate costs hypothesis, diseased females may

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not be able to afford the investment of caloric energy necessary for physically searching for males, because immune reactions are energetically costly (Martin et al., 2003). Also, the opportunity costs of assessing prospective mates may be greater for infected females because they may need to compensate for parasitic damage by foraging more or by seeking specific nutrients that enable immune protection (e.g., protein; Lochmiller et al., 1993). If mate sampling is more costly for infected females, they may sample fewer males before soliciting copulation, thus mating more quickly. Alternatively, under the obtaining complementary genes hypothesis, if infected females react to their state by seeking male genes that will, in complement to their own genes, create disease-resistant offspring, they will probably sample more males than do healthy females, and require more time to view males before they choose one for copulation. I test these hypotheses experimentally in a host-parasite system involving wild turkeys (*Meleagris gallopavo*) and their deleterious, intestinal protozoal parasites, the eimerian coccidia (Apicomplexa: Eimeriidae).

Coccidia infest the cells lining the digestive tract, causing in domestic poultry lesions that result in reduced weight gain (Bressler and Gordeuk, 1951), loss of essential nutrients (Ruff et al., 1974), production of free radicals (Allen, 1997), lower testosterone titers in males (Ruff, 1988), and reduced fecundity in both sexes (Bressler and Gordeuk, 1951; Ogbuokiri and Edgar, 1986). Coccidiosis causes poorer carotenoid-based plumage coloration in wild species (Hill and Brawner, 1998; McGraw and Hill, 2000). Seven species of coccidia are found in turkeys, and their pathogenicity ranges from minimal to extreme (Edgar, 1986). These parasites are transmitted via oocysts that are excreted with the feces of the host. The oocysts must sporulate under the appropriate environmental conditions (high oxygen, lower than body temperatures) before becoming infective (Long, 1982) and, thus, are unlikely to be transmitted sexually. Transmission occurs when another turkey inadvertently ingests the sporulated oocyst while foraging. The sporozoites released from the oocyst reproduce asexually within cells, producing merozoites that burst from those cells, each to infect a new cell. Both domestic and wild turkeys are very susceptible to infection when they are young but appear to develop parasite species- or strain-specific immunological resistance as they age (Chapman, 1996; Davidson and Wentworth, 1992). Nevertheless, adult turkeys may harbor latent infections of coccidia that flare up when the host individual is stressed (Aiello and Mays, 1998). Very little is known of the epidemiology of coccidiosis in free-living wild turkeys (Davidson and Wentworth, 1992). Prestwood et al. (1971, 1973) suspected that pathogenic strains of coccida may contribute to the 56–73% mortality that occurs among wild turkey poults in their first few weeks after hatching (Vanglider, 1992). Prestwood et al. (1971) reported a 50% incidence of *Eimeria* oocysts among 123 5–13-week-old poults collected in Alabama, Arkansas, Mississippi, and West Virginia but a much lower prevalence (17%) in juveniles and adults. Kozicky (1948) found oocysts in 40% of the adults' droppings he collected in Pennsylvania. Neither study reported the numbers of oocysts shed in the feces. In north Florida, male wild turkeys show considerable individual differences in mean coccidia burden (range = 10–30,000 oocysts/g of feces; Buchholz, 1995). Such variation in *Eimeria* resistance is associated with immunogenetic and other heritable differences in domestic fowl (*Gallus gallus*; Johnson and Edgar, 1982; Lillehoj et al., 1989). Given the considerable major histocompatibility complex (Mhc) variation in wild turkeys (Zhu et al., 1996), and Mhc-associated immunocompetence (Buchholz et al., 2004) and susceptibility to bacterial and viral diseases in domestic turkeys (Nestor et al. 1996), resistance to coccidia might be heritable in this species as well.

Coccidia reduce the quality of male ornamentation in wild turkeys, indirectly making males less attractive mates (Buchholz, 1995; data not shown). Nothing is known of the effects of infection on female behavior. My null hypothesis is that parasitic infection does not affect mate sampling at all. Alternatively, females may either become less able to invest in male assessment due to the deleterious effects of infection or become choosier to maximize their lifetime fitness by producing offspring that are not as susceptible to disease.

METHODS

Study subjects

Wild turkey poults were hatched from the artificially incubated eggs of 15 pairs of wild turkeys maintained at the Department of Biology's Avian Research Facility at the University of Mississippi Field Station in Lafayette County, Mississippi, USA. To reduce the effects that rearing familiarity within treatments might have on mate sampling at maturity, the poults were repeatedly regrouped. The poults were mixed from staggered hatch groups of various parentage into early rearing groups (0–2 weeks), then pooled into two preinfection groups (2–6 weeks), and finally randomly assigned at 6–8 weeks of age to two postinfection subgroups for each treatment. Rearing groups could always hear, and usually see, one another. At about 4 months of age, treatment subgroups were combined so infected and control groups were housed in two abutting cages, such that they could see and hear one another. At 7–8 months of age, all males were removed from the group cages and housed individually, out of sight from the females. The stretched length of each male's fleshy frontal process, or "snood," was measured when they were approximately 11 months old. Male snood length is known to be sexually selected in wild turkeys (Buchholz, 1995, 1997).

The parasitized treatment groups were created by infecting each chick per os with approximately 12,000 oocysts of turkey coccidia in a variable volume of palatable sucrose solution. This dose is based on the reasonable scenario of a chick consuming 0.25 g of feces from a heavily infected adult (Buchholz, 1995). Uninfected control chicks were given a sham infection of sucrose solution only. Coccidia species in the oocyst cultures included deleterious strains of *Eimeria adenoeides* alone, or with *E. meleagrimitis*, and *E. gallopavonis* as provided by P. Augustine (Parasite Biology, Epidemiology and Systematics Laboratory, US Department of Agriculture). The coccidia caused the near cessation of feeding and drinking for several days, accompanied by diarrhea and bloody stool. Although the parasitized chicks were listless, none died in the weeks immediately after infection. Normal feeding resumed within 10 days postinfection. The wire flooring of their brooder cages prevented immediate reinfection via fecal consumption. The sexes of the chicks were not known at the time of treatment, making it necessary to equalize the sample sizes of the treatment groups when they could be sexed at 3 months of age. Two slightly older females, previously uninfected, were infected and added to the treatment group at this time. The control group received medicated water (0.020% amprolium) to impair replication of coccidia in their gastrointestinal tracts should they somehow ingest some oocysts (Lindsay and Blagburn, 2001). Control hens showed no clinical signs of infection during rearing, and pooled fecal samples from their pens did not contain oocysts. I did not monitor the coccidia burden of individual hens before the trials because the species-specific circadian cycles of the coccidia (Boughton, 1988; Hudman et al., 2000), require a 24-hour collection of feces to accurately summarize total coccidia burden in adult wild turkeys (Buchholz, 1995).

Isolating individual hens is very stressful to the individual and also disrupts flock dominance hierarchies, endangering the isolated hen when she is reintroduced to her group. Consequently, individual coccidia burdens were assessed after the mating trials were completed. After all hens were tested, the anticoccidial medication of the control group was stopped to allow any latent infections to be detected. Two weeks later, each hen was placed in a large, well-ventilated, wax-coated cardboard box (specifically designed to transport wild turkeys safely; National Wild Turkey Federation) for 24 h. The accumulated feces in each box were mixed together, weighed, suspended in 2.5% potassium dichromate, and later examined for oocysts using standard quantitative methods (Buchholz, 1995).

Apparatus

Mate sampling trials were conducted when the birds were 10–11 months old. The sampling arena was an artificial lek consisting of a row of 16 male cages bordering a long corridor that females could walk along during the trials (Figure 1). The fronts of the male cages were covered with poultry netting, allowing females a clear view of each male, but solid dividers between cages prevented males from visual and physical interaction. The lighting was a combination of indirect sunlight and incandescent artificial light. Because female wild turkeys are greatly disturbed by handling, the 29 females were trained to walk from their group cages to the sampling corridor. Before the individual trials, each treatment group was gently herded en masse in one direction along the row of males to familiarize them with the arena and the position of the males, but they were not allowed time to view each male carefully. This process was repeated a second time by herding the females in the opposite direction so that the first male seen in the first visit was the last seen in the next visit. No females solicited during these habituation parades.

During the trials each study subject was moved to the sampling row of males by allowing her to walk out of her group pen of her own volition, with barriers placed to direct her to the arena some 12 m away. All females entered the same end of the arena and then were gently encouraged to walk to the opposite end of the male row by an investigator slowly walking behind her. Escorting each female down the sampling corridor ensured that all knew that 16 males were present. When the female reached the last male, I exited the arena and observed from hidden observation ports behind the males and at either end. It was not logistically possible for me to record the display rate of all 16 males because females often moved rapidly. During the sampling trials, I confirmed that each male did court when a female was present. Males must court to be solicited by females, but display rate is not associated with mating success (Buchholz, 1995). In separate male choice trials, I demonstrated that males do not display differently to infected females (data not shown).

After I left the arena, the hen was given 5 min to become accustomed to her surroundings, and then her movements in front of the male cages were recorded. Each female visit to the section of the walkway immediately in front of a male's cage was counted as a sampling event. Each 5-s period that the female stayed in a male's zone was tallied as an additional sampling event. Sampling events were multiplied by 5 s to calculate the total time a female visited each male. Visits in which the female ate the male's food for more than 5 s, directed her attention to objects outside of the aviary, or foraged in the grassy strip opposite the male cages were subtracted from the total trial length to measure the proportion of trial time each female spent mate sampling. Trials ended when a female solicited a male by exhibiting the diagnostic "crouching" behavior, or after more than 35 min had passed without solicitation of a male. The movements of two females (both infected) that ran up and down the arena's length, alarm calling, were excluded from analysis because they did not appear to be examining the males. The observer was aware of the treatment condition of each hen; however, the standardized methodology of recording female position and actions should have prevented any inadvertent biasing of the data. In addition, the data were examined to ensure that the linear nature of the arena was not biasing the results by causing females to stay near (or keep away from) the males near the entrance/exit at either end.

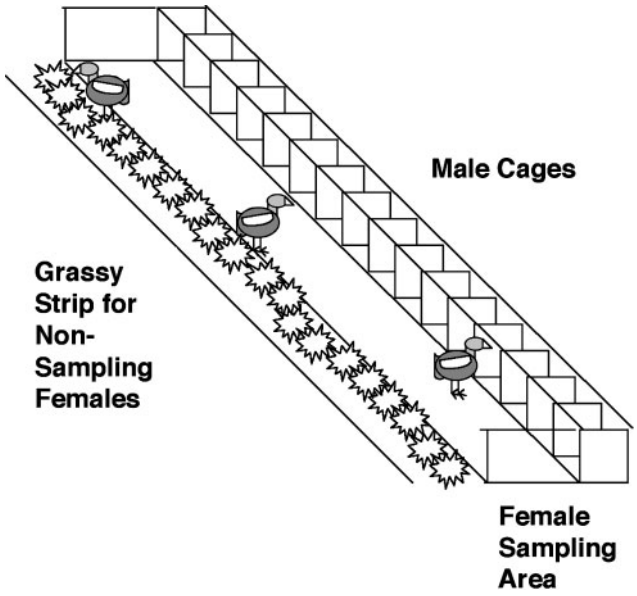

Figure 1
Arena to observe mate sampling by wild turkey hens (not to scale). Males are housed individually in sixteen 2 × 1.5-m pens. Females are able to inspect males by walking along a 1.8-m-wide corridor running the length of the males' pens. When females faced or entered the grassy strip (i.e., away from the males) they were recorded as not sampling (e.g., the upper most female in the figure). An "old field" habitat was visible to the females through the poultry netting wall behind the grassy strip. Each female was tested individually.

Statistical analysis

The activity rate of each female was calculated as the number of times she crossed the divider between male cages, divided by trial length. An index of a female's mating indecision was calculated as the number of times she returned to inspect the male she spent the most time with, divided by trial length. Four dependent variables indicated variation in female sampling behavior: proportion of trial time spent sampling males, number of males visited, mean time with sampled males, and the time preference function for male snood length. The number of males visited was regressed onto trial length, and the residuals were used as the dependent variable in subsequent analyses. The ornament preference function (Wagner, 1998) of each female is represented by a Spearman's correlation coefficient (rho, corrected for ties) of the proportion of sampling time she spent with each male relative to the preferred male ornament in a previous study, male stretched snood length (Buchholz, 1995). The variables were inspected for normality and for homogeneity of variances between treatments. The data for proportion of trial time sampling required arcsine transformation to achieve a normal distribution. The ornament preference function values were

Table 1
Multivariate and univariate ANOVAs in female sampling behavior

	MANOVA			ANCOVA					
				%T samp		T with male		No. sampled	
Independent variables	Wilk's λ	$F_{3,18}$	p	$F_{1,20}$	p	$F_{1,20}$	p	$F_{1,20}$	p
Coccidia infection	0.589	4.20	.021	0.80	.38	6.41	.02	5.51	.03
Mate solicitation	0.488	6.29	.004	0.23	.63	11.42	.003	2.72	.12
Body condition	0.838	1.16	.353	0.27	.61	0.24	.63	3.11	.09
Infect × solicitation	0.918	0.54	.662	0.86	.36	0.57	.46	0.18	.68
Infect × condition	0.562	4.68	.014	3.39	.08	0.69	.42	0.46	.51
Solicit × condition	0.638	3.41	.040	3.43	.08	0.25	.62	1.52	.23

%T samp, proportion of time spent sampling males; T with male, mean time female spent visiting a sampled male; No. sampled, number of males sampled during the trial.

bimodally distributed and could not be transformed appropriately. Therefore, the preference rho's are analyzed separately with nonparametric statistics. I suspected that an individual female's body condition might affect her sampling behavior, even if she is uninfected. A female body condition index (BCI) was calculated as the residual values of a regression of body mass on tarsus length.

A MANCOVA was used to examine the effects and interaction of the factors (female infection and female solicitation) and the covariate (female BCI) on mate sampling. The multivariate test results were compared with univariate ANOVA results to explore whether statistical significance is owing to common variation shared by the dependent variables or owing to separate relationships of the dependent variables with the factors (SAS, 1998). To explore how the parasitic burden of the turkey hens as adults might affect female sampling differently than their previous infection history as poults, I repeated the multivariate and univariate ANOVAs using as the infected group females that were actively shedding oocysts, and the remaining females as the control. Games/Howell tests were used for robust post hoc examination of means differences while controlling for experiment-wide type I error. I assume that the response of each female is statistically independent. Some female sibships were more numerous in one treatment group than the other, but the number of biased families (and individuals in those families) was equivalent in each group, and therefore, bias in parentage should not be more likely to affect one treatment than the other. Statview 5.0 was used to calculate the statistics (SAS, 1998). Probability values are two-tailed unless otherwise noted. Mean ± SE is reported.

RESULTS

Thirteen infected and 14 control females responded by walking calmly along the sampling corridor. Eighteen females solicited copulation. No solicitation bias in favor or against males at the ends of the arena occurred ($y = 1.664 - 0.232x + 0.014x^2$, $R^2 = .03$, $F_{2,15} = 0.18$, $p = .84$). Treatment did not affect the likelihood of a female to solicit copulation (62% infected, 71% control; $\chi^2 = 0.30$, df = 1, $p = .57$), nor was the time until soliciting significantly different between infected females (16.7 ± 4.7 min) and control females (14.5 ± 4.3; $t = 0.35$, df = 16, $p = .73$). Female treatment groups did not differ in the rate of activity (males per minute: infected, 3.36 ± 0.59; control, 4.25 ± 1.16; $t = -0.70$, df = 19, $p = .50$) or in the number of times they reexamined a favored male (revisits per minute: infected, 0.43 ± 0.08; control, 0.73 ± 0.19; $t = -1.56$, df = 19, $p = .14$). Multivariate and univariate ANOVA results are summarized in Table 1. Shared variation in the female sampling variables was explained by both female infection status and solicitation during the trial. Examination of individual ANOVA tables revealed that infection affected both the variation in the time that females visited each male and the total number of males sampled. The treatment means indicate that infected females sampled 2.2 more males than did uninfected females (Figure 2). Females that solicited visited individual males for 30-s shorter periods than did females that did not solicit. Infection and female solicitation showed no significant interaction, thus the effect of infection on female sampling did not depend on whether she solicited. Body condition did not have a significant direct effect on mate sampling but had a significant multivariate interaction with the dependent variables. The univariate analyses suggested ($p = .08$) that the proportion of trial time that a female spent sampling might interact with female condition differently, depending on the factor category. Closer inspection of the relationship between the proportion of time sampling and body condition showed negative regression slopes for control (−0.45) and nonsoliciting (−0.20) females, and positive slopes for infected (0.20) and soliciting (0.42) females, but these associations were weak (all $R^2 < .08$) and statistically not significant (all $p > .05$).

Overall, the test females had negative mean preference functions (sign test, $p = .05$), largely owing to the sampling behavior of infected females (−0.23 ± 0.09) rather than uninfected hens (−0.09 ± 0.08). If only soliciting females are considered, the ornament preference functions of infected females remained negative (−0.28 ± 0.1) and were significantly different than those of control hens (0.0 ± 0.1) (Mann-Whitney U test, $U = 15$, $U^1 = 65$, $z = -2.234$, $p = .026$). When both treatments were combined, females were more likely to solicit males with longer than average snoods ($\chi^2 = 5.54$, df = 1, $p < .05$). All the females that solicited shorter-than-average-snood males were infected (or 50% of the infected hens who solicited copulation). Thus, infected females appear to mate at random with respect to male snood length, whereas the uninfected control females showed a significant preference for longer than average snoods (100% of the soliciting control hens; binomial test, one-tailed $p = .01$).

Regular fecal sampling from the cage litter of the female groups confirmed the efficacy of the treatment; oocysts occurred in pooled samples from the infected female cage but were not detected in the control cage. The fecal samples collected over a 24-h period from each infected female 2 weeks after the behavioral trials showed that five of them were actively shedding oocysts (1091 oocysts/g ± 595). Two weeks after withdrawal of the amprolium treatment, two of the

control females were shedding coccidia in low numbers (47 oocysts/g ± 24), revealing that they were harboring nascent infections that had been suppressed by the anticoccidial. Shedding females did not have different time preference functions for snood length (Mann-Whitney U test, $U = 32.5$, $U^1 = 77.5$, $z = -1.40$, $p = .16$) Two of the five hens shedding oocysts solicited copulation, both from males of below-average snood length. Multivariate ANCOVA using the five females that had active infections as the infected group, and the remaining females as the control did not result in a significant effect of shedding on mate sampling (Wilk's $\lambda = 1.0$, $F_{3,18} = 0.65$, $p = .59$). Nor were there statistically significant effects of oocyst shedding on the dependent variables in univariate ANOVAs (proportion of time sampling: $F_{1,20} = 1.07$, $p = .31$; mean time with a male: $F_{1,20} = 0.01$, $p = .91$; number of males sampled: $F_{1,20} = 1.23$, $p = .28$).

DISCUSSION

Previous studies of the effects of female parasitism on mate choice in dichotomous choice tests concluded that sexual selection is weakened by female infection in guppies (*Poecilia reticulata*; Lopez, 1999), spadefoot toads (*Scaphiopus couchii*; Pfennig and Tinsley, 2002), and the upland bully (*Gobiomorphus breviceps*; Poulin, 1994). In these studies, infected females appeared to mate at random or were less choosy, whereas uninfected females exhibited preferences for males with higher-quality ornaments. In similar studies in which females were given a dichotomous choice of odors from infected and uninfected (or less infected) males, parasitized salamander (*Plethodon angusticlavius*) females also chose male stimuli at random (Maksimowich and Mathis, 2001), and coccidia-infected female mice (*Mus musculus*) were less extreme in their bias (Kavaliers et al., 1998) than were healthy females. The present study differs from these in that young females were infected with a deleterious parasite to which they can acquire resistance as they get older. Although the coccidia were actively replicating in a few of the infected turkeys, it appeared that most had either eliminated the infection or suppressed reproduction (and thus tissue damage) by the parasite. As adults, the infected females had information about their own susceptibility to coccidia without being heavily burdened by the parasite at the time of mating. Good genes models of intersexual selection would predict that a female should choose a mate whose genes, in recombination with her own, will spare her offspring the harmful effects of parasitism. The control females, on the other hand, were given false information about their genetic condition because the anticoccidial medication prevented coccidia replication and damage. The control females were predicted to be no more or less choosy than in previous dichotomous choice trials (Buchholz, 1995). Unlike the dichotomous choices provided in previous studies, the females tested in the present study were presented with numerous potential mates.

Under these experimental conditions, I detected behavioral differences between control and infected females in the way that they sampled males. Although not all infected hens chose the same male phenotype (longer snoods) preferred by all of the control hens, the data on mate sampling suggest that this was not the result of females being so burdened by infection that they solicited copulation haphazardly. Two lines of evidence suggest that the changes in female behavior as a result of their infection history were not so they could reduce the immediate costs of mate sampling. The activity rate of infected females was not less than control hens; in fact, they sampled more individual males than did healthy females. Infected hens did not mate more quickly than did control birds. If infected females are trying to reduce the costs of mate

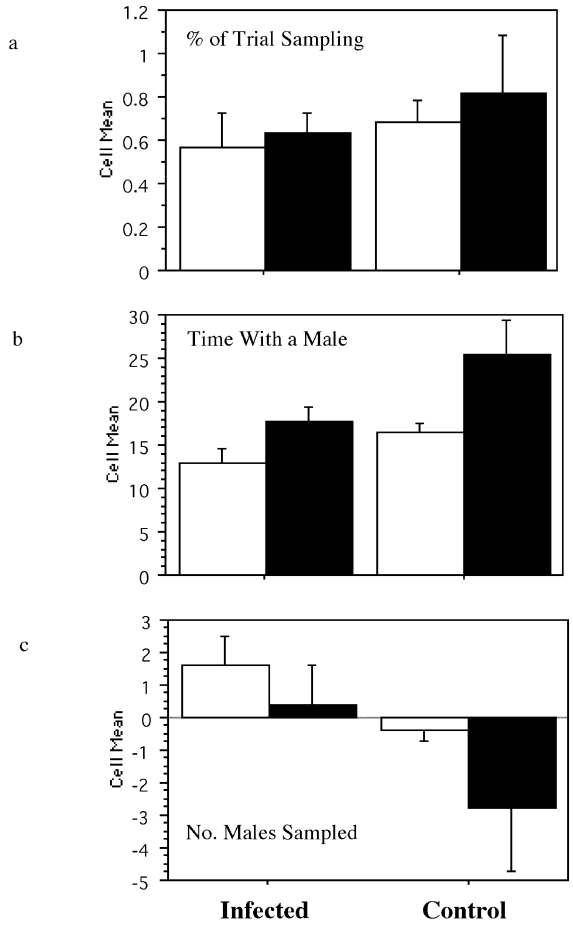

Figure 2
Soliciting females had a significantly lower mean "time with a male" than did nonsoliciting females. Infected females visited significantly more males than did control females. White bars are females that solicited copulation. Black bars are nonsoliciting females. Standard error bars are provided. (a) Proportion of trial time spent sampling (not significant). (b) Mean time sampling individual males ($p < .05$ for soliciting versus nonsoliciting). (c) Number of males sampled ($p < .05$ for infected versus uninfected). Games/Howell post hoc tests were used to determine significance of mean differences while controlling the testwide p level to 0.05.

sampling, they should have visited few males, quickly. Infected hens did behave in one way that would be reflective of females that were trying to reduce the costs of mate sampling: they seemed to spend less time examining each individual male.

Support for the obtaining complementary genes hypothesis is evidenced by the sampling behavior of infected females; they examined a greater range of males and, in soliciting hens, spent a greater proportion of their time examining shorter-snooded males. If infection does not alter the cognitive template against which sampling females compare males, they should have preferred the same male ornament as did the control females and visited short- and long-snooded males for equal amounts of time. Contrary to my predictions for the obtaining complementary genes hypothesis, infected females did not spend a greater amount of time inspecting every male. Classic parasite-mediated sexual selection (Hamilton and Zuk, 1982) might predict that infected hens would show a stronger preference for the parasite-indicating male trait than resistant birds. This did not occur. Mate choice for genetic compatibility (Tregenza and Wedell, 2000), on the other hand, pre-

dicts that females use information about their own genome to select a uniquely compatible partner, such that females do not agree on a "best" male phenotype. In summary, my results suggest that coccidia infection has long-term impacts on female mating that are more consistent with compatibility tactics than with the reduction of sampling costs or directional selection for parasite resistance alleles. My conclusions with respect to coccidia infection and female sampling behavior are clearly speculative, so before discussing the evolutionary implications of the changed mating tactics of infected females, nonsexual hypotheses should be considered.

Because parasitic infection may reduce the fear mechanisms of hosts in a variety of contexts (Adams and Fell, 1997; Kavaliers et al., 1998), it may be unreasonable to conclude that the behavior of infected females has an adaptive explanation under sexual selection. I address three nonsexual hypotheses that might explain changes in mate sampling by parasitized females: First is neuropathology. Parasitic infection may cause nerve damage that impairs the sensory or cognitive mechanisms of females. Infected females might not be able to distinguish between males of different ornament quality, or they might not be able to remember which males are superior in appearance. Second is parasite avoidance. Diseased females might not assess all the available males to avoid exposure to additional parasites that might be accumulated during male sampling (Pfennig and Tinsley, 2002). Third is parasite manipulation. The parasite may alter the behavior of the female to facilitate transmission of infection to new hosts (Møller, 1993).

The first two nonsexual explanations for random mating of parasitized females do not appear to explain the results of the present study. The impacts of coccidia on domestic poultry have been studied in great detail, but host neuronal damage is never mentioned as a consequence of infection (Long, 1981; McDougald et al., 1986). The infected females in the present study did not show signs of being confused or cognitively disabled. For example, parasitized hens did not dither and reexamine their favored male more often than did control females. They also did not seem to be avoiding the risk of becoming infected by additional parasites; infected females inspected more males, a result opposite to that predicted by the parasite avoidance hypothesis. The third nonsexual explanation for random mating by infected females is that the parasite is manipulating its host to enable it to spread to new host individuals. There are several reasons why I do not favor this hypothesis for the present study system. First, parasitic manipulation seems to be more common in parasites with indirect life cycles wherein they must pass through several host species before reproducing (see Moore, 1984). Second, although infected hens did visit more males, they rarely defecated during the trials (Buccholz R, personal observation) and therefore would not be effective transmitters of coccidia to the males in this context. Only five of the infected females were actively shedding oocysts during the trial period, and oocyst production was not associated with changes in female sampling behavior. Third, the time preference functions of the parasitized hens show that they spent more time with shorter-snooded males. Short-snooded males are the most heavily infected individuals in the wild (Buchholz, 1995). It may seem, therefore, that females are being manipulated to stay near males that are innately more susceptible to infection. However, already infected males might not be the best hosts for this parasite. Given the overdispersion typical of parasites, some highly ornamented males are both susceptible and immunologically naive (Poulin and Vickery, 1996) and would make good habitat for coccidia sporozoites. Oocysts ingested by already infected hosts, on the other hand, face intense competition for undamaged tissue in the gut (Edgar, 1986). They also have to survive the acquired immune defenses of a host already immunized by *Eimeria* antigens (Chapman, 1996). Finally, even if coccidia are manipulating hen movements, this manipulation is not necessarily incongruent with adaptive mate choice by the female turkey. Coccidia are not sexually transmitted, and thus, the parasite's fitness is not affected by which male a female chooses, as long as she continues to visit the home ranges of additional males. In conclusion, there is no clear evidence that the significant changes observed in the sampling behavior of infected turkey hens is the result of parasite damage, avoidance, or manipulation.

In many previous studies of the effects of parasitism on female choice, the choosy females suffered greatly from the disease organism during the trials themselves. For example, Lopez (1999) infected guppies with blood-sucking gill ectoparasites that probably impeded oxygen exchange in addition to causing anemia. Pfennig and Tinsley (2002) observed the effects of infections of monogenean parasites in the urinary bladder, also blood feeders, on the mating patterns of spadefoot toads. Poulin (1994) examined the effects of a trematode that disrupts muscle and organ function when it encysts in large numbers in the tissues of upland bullies. Infected females exhibited reduced activity in guppies and bullies, but not in spadefoot toads. These investigators rightly chose to use parasites that the respective hosts regularly encounter in the wild, and they used host individuals with naturalistic numbers of parasites. However, the study of the effects of greatly burdensome parasites may not represent the majority of host-parasite associations. Many parasites of vertebrates probably reproduce and are active only during brief immunological windows (Schad et al., 1997) that occur when the host is young or stressed or invests energy in alternative fitness-enhancing activities, such as parental care (Richner et al., 1995). Assuming that parasitic infection is not an immediate threat to survival, diseased females should attempt to increase their reproductive success, just as parasitized males do (Kavaliers et al., 1997).

Previous alternative explanations for apparently random mate choice in diseased females have not considered that different females may use different choice criteria to obtain different types of benefits, depending on their individual genotype, previous rearing history, and present environment (Jennions and Petrie, 1997). In studies of mate choice by parasitized females, the subjects may have been looking for male characters other than those presented by investigators in the dichotomous choice trials. In the wild, females have the option to accept greater search costs when their mating criteria are not met. Satin bowerbird (*Ptilorhynchus violaceous*) females, for example, increase mate searching when preferred male phenotypes are unavailable (Uy et al., 2000). The ways in which females search and decide on a mate have different costs, depending on how many males are visited. The combination of sampling strategy and the number of males inspected affects the mean fitness of males (Janetos, 1980). Wagner (1998) described how the experimental design of mating trials can skew our understanding of how sexual selection affects the rate and direction of the evolution of male secondary sexual characters. If mate sampling by parasitized females is not random, as my observations of female behavior in an artificial wild turkey lek suggest, then the results of some previous studies of this question in which females were given dichotomous choices may be open to alternative explanations. Mate choice experiments should give females a choice of multiple males and monitor not just choice but female sampling as well. Varying the sampling costs to females may also be informative. Studying the subclinical effects of so-called benign parasites might provide results more broadly

applicable to host decision-making processes than would new investigations of females heavily infected with debilitating disease. If most parasites are not acutely harmful, then not all infected females should be forced into hurried mate choice. It will be difficult to design studies to test the genetic complementarity hypothesis as it applies to parasitized females. Unfortunately, in this study the sample size of soliciting infected females is probably too small, and the variation in the major histocompatibility complex of wild turkeys too great (Zhu et al., 1996) to determine if the infected hens were attempting to produce young of greater immunogenetic heterozygosity. The lower variability seen in parasitized compared with control hens for activity and reexamination rates suggests that infection equally impacts different genotypes affecting premating movements. However, this would not necessarily mean that infected females will all prefer the same male characteristic. In the future, naturalistic experiments testing mate sampling and choice by females of known parasite-resistance genotypes may be a productive approach towards understanding why parasitized females do not seem to prefer more ornamented, or healthier, males.

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