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Predation risk, host immune response, and parasitism

C. Navarro,[a] F. de Lope,[a] A. Marzal,[a] and A. P. Møller[b]
[a]Departamento de Biología Animal, Universidad de Extremadura, E-06071 Badajoz, Spain, and
[b]Laboratoire de Parasitologie Evolutive, CNRS UMR 7103, Université Pierre et Marie Curie, 7 quai St. Bernard, Case 237, F-75252 Paris Cedex 05, France

Predation risk may affect the allocation priorities of limiting resources by potential prey. Investment in immune function should receive reduced priority, when hosts are exposed to predators because of the costs of immune function. We tested this hypothesis by randomly exposing adult house sparrows, *Passer domesticus*, to either a cat, *Felis catus*, or a rabbit, *Oryctolagus cuniculus*, for 6 h while assessing their ability to raise a T-cell–mediated immune response to a challenge with phytohemagglutinin. Sparrows exposed to a cat had a significant reduction of, on average, 18% and 36% in T-cell response in two different experiments compared with sparrows that were exposed to a rabbit. In a field experiment with a barn owl, *Tyto alba*, or a rock dove, *Columba livia*, placed next to a nest-box during laying, we found a mean reduction in T-cell–mediated immune response of 20%. In males, the reduction in cell-mediated immune response owing to cat exposure increased with increasing size of the badge, which is a secondary sexual character, but only during the breeding season. In a third experiment, house sparrows were either exposed to a barn own, *T. alba*, or a rock dove, *C. livia*, and development of malarial infections was recorded during the following 6 weeks. Individual sparrows exposed to a predator had a higher prevalence and intensity of *Haemoproteus* malarial infection than did control individuals. Therefore, exposure to predators reduced that ability of hosts to cope with parasitism mediated through effects on immune function. *Key words:* cell-mediated immunity, *Haemoproteus*, house sparrow, malaria, *Passer domesticus*, phytohemagglutinin, sexual selection, T-cell response. *[Behav Ecol 15:000–000 (2004)]*

Predation is an important cause of natural selection, as demonstrated by numerous examples of differences in mean phenotypes between individuals that fall prey to a predator and survivors (for review, see Endler, 1986). Evolutionary trends in defense and offense provide evidence that predation is an important selective interspecific interaction on an evolutionary time scale (Vermeij, 1987). Although predation (and other interspecific interactions) often is studied in isolation, several pieces of evidence suggest that different kinds of interspecific interactions may affect each other.

Predators are usually considered to prey upon individuals of inferior condition. In an empirical break-through, Temple (1987) used a raptor to collect a sample of small mammal prey and compared the parasite loads of prey with a random sample collected with a shotgun. Mammals killed by the predator consistently suffered significantly more from parasitism by trematodes, nematodes and ectoparasites than did the randomly collected mammals (Temple, 1987). A second study by Møller and Erritzøe (2000) compared the phenotype of passerine birds captured by cats with the phenotype of a control group killed by collisions with windows or cars. Birds that had fallen prey to cats consistently had smaller spleen sizes than did birds that had died for other reasons, suggesting that they had weak immune systems. No other traits such as sex or age, month of death, body mass, body condition, liver mass, wing length, or tarsus length differed significantly between prey and nonprey. A third study by Hudson (1986) showed that female red grouse, *Lagopus lagopus*, infected with helminths produced an odor that made them more susceptible to mammalian predation compared with that of uninfected individuals. These results indicate that parasites may play an important role in predator-prey interactions.

Several aspects of the immune system are known to be under the influence of condition (Alonso-Alvarez and Tella, 2001; Chandra and Newberne, 1977; Gershwin et al., 1985; Møller et al., 1998). This condition-dependence is reflected by an increased ability to raise an immune response when provided with a high-quality diet. However, immunity is also directly dependent on current state as reflected by endocrine factors, because both androgens and corticosteroids can affect the ability to produce an immune response (Apanius, 1998; Silverin and Wingfield, 1998; von Holst, 1998). A typical consequence of stress is an increase in circulating levels of glucocorticoid steroids (the so-called adrenocortical response), which eventually can result in immune suppression (Apanius, 1998; Wingfield et al., 1998). Although many different kinds of stressors have been used in experiments to induce an adrenocortical response, to the best of our knowledge there are no studies linking predation to the ability to raise an immune response.

The most sophisticated defense system used by hosts against parasites is the immune system. Numerous studies have shown correlations between strength of immune response and prevalence or intensity of infection, and particular major histocompatibility complex haplotypes are associated with reduced levels of infection (Klein, 1990; Roitt et al., 1996; Wakelin, 1996). This also applies to birds, in which studies of chickens have shown that viral diseases, gastrointestinal parasites, Coccidia, *Cryptosporidium*, *Salmonella*, and protozoan blood parasites are affected by host immune responses (Lillehoj, 1991; Sharma et al., 1991).

The first main objective of the present study was to test how perceived predation risk affects the ability of potential prey to produce a response to a challenge of the immune system with a novel antigen. This was done by experimentally exposing individual house sparrows, *Passer domesticus*, to a predator or a control treatment and subsequently measuring the immune response. This was done in aviary experiments and in field

Address correspondence to A.P. Møller. E-mail: amoller@snv.jussieu.fr.
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experiments near occupied nest-boxes. We used exposure to two common predators that typically inhabit breeding sites for house sparrows: a domestic cat, *Felis catus*, and a barn owl, *Tyto alba*. These predators are commonly present in farms where house sparrows are breeding, and they can thus be exposed to such predators throughout the breeding season. Our experimental exposure of house sparrows to predators thus consisted of a short-term exposure compared with the permanent and long-lasting exposure that occurs in many breeding sites. The second objective of the study was to investigate to which extent the effects of the experimental treatment on immune response depended on the expression of a secondary sexual character. The prediction was that males with large secondary sexual characters generally would be in better body condition, because their secondary sexual character is condition-dependent, and that they therefore were able to sacrifice their ability to produce an immune response without compromising their current health status. The third objective was to test the long-term effects of reduced immune response on malarial infection. Previous studies of this host-parasite system have shown a negative relationship between strength of cell-mediated immunity and intensity of malarial infection (González et al., 1998; Navarro et al., 2003).

House sparrows are approximately 30-g passerine birds that are gregarious during and outside the breeding season (Summers-Smith, 1963). Among adults, males differ from females by having a black badge that is partly covered with light feather tips just after the annual moult in September–October (González et al., 2001; Møller and Erritzøe, 1992). Males with larger badges appear to be more dominant (González et al., 2002; Møller, 1987a,b; Solberg and Ringsby, 1997), and they are more successful in becoming mated and in obtaining extrapair copulations than are other males (Møller, 1988, 1990; Veiga, 1993). Males with a large badge produce a stronger T-cell–dependent immune response than do males with small badges during the breeding season, but not outside the breeding season (González et al., 1998, 1999). Males with large badges have a smaller bursa of Fabricius than do average males (Møller et al., 1996). House sparrows with strong T-cell responses also consistently have lower levels of malarial infections than do individuals with weak responses (González et al., 1998; Navarro et al., 2003). House sparrows are very vigilant when foraging to avoid predator attacks (Cramp and Perrins, 1994). Common predators of house sparrows include cats and other mammalian predators, birds of prey, and owls (Cramp and Perrins, 1994).

METHODS

General methods

We captured house sparrows in mist nets during December 2000–January 2001 and September–October 2001 and put them in eight aviaries sized 3.5 × 1.1 × 2.5 m. The aviaries contained food (a mixture of commercial feed for seed eating birds) and water ad libitum. Perches were provided, and each aviary contained two nest-boxes. The first experiment was conducted 5–7 April 2001 and included 33 sparrows (16 males, 17 females); the second experiment was conducted 29 November–16 December 2001 and included 31 sparrows (15 males and 16 females), and the third experiment was conducted 5 March–17 April 2002 and included 96 sparrows (46 males and 50 females). In the two first experiments, the experimental sparrow was placed in an aviary next to a cage containing either a rabbit or a cat.

At the start of the two first experiments, a test of T-cell–mediated immunity was initiated, and the response was measured after 6 h of exposure. The first series of experiments had food available continuously. The second series of experiments had all food removed the evening before the experiment, and food was made available at 0900 h, when the experiment was started. This change in procedure was made to allow video filming of risk taking during exposure for a complementary study.

As a measure of immune response we used the T-cell–mediated immune response to a challenge with phytohemagglutinin (PHA). This is a standard estimate from the poultry literature of the ability to produce a T-cell–mediated immune response (Dietert et al. 1996; Goto et al., 1978; McCorkle et al., 1980; Parmentier et al., 1993). Injection with PHA results in local activation and proliferation of T cells, followed by local recruitment of inflammatory cells and major histocompatibility complex molecules (Abbas et al., 1994; Goto et al., 1978; Parmentier et al., 1998). House sparrows were injected 0.05 ml of 0.2 mg phytohemagglutinin (PHA-P) in one wing web (patagium) and 0.05 ml physiological water in the other wing web.

We measured the thickness of the patagium injected with PHA and with physiological water before injection and after 6 h, using a pressure-sensitive caliper (Digimatic Indicator ID-C Mitutoyo Absolute cod. 547–301 Japan),with an accuracy of 0.01 mm. This measure has a very high repeatability, as shown previously for the house sparrow (González et al., 1998; Lope F, unpublished data). We have shown in a previous study of house sparrows from the same population that T-cell responses measured after 3, 6, 12, 24, 36, 48, and 72 h are strongly positively correlated, and that there is no further significant increase in T-cell response after 6 h (Navarro et al. 2003). This justifies the use of an interval of 6 h in the present study.

Body mass was recorded with an electronic balance to the nearest 0.01 g at start and after 6 h. The difference in body mass between these two measures was used as an additional variable. We measured tarsus length on the first capture with a digital caliper to the nearest 0.01 mm. Badge size of males was estimated from the length and the width of the total badge (including the part that was hidden by white feather tips) with a ruler to the nearest 0.01 mm after pressing the feathers against the body. All measurements were made by a single experienced person, which reduced the variance in the data owing to interobserver variability.

A blood smear was made from a drop of blood collected from the brachial vein immediately after exposure to the cat or the rabbit. The slide was air dried and then fixed in absolute ethanol and stained with Giemsa for 45 min. Smears were scanned at 1000× magnification and were investigated for presence of *Haemoproteus*, *Leucocytozoon*, Plasmodium, *Trypanosoma*, and microfilaria. The intensity of infection was quantified as the number of *Haemoproteus* blood parasites observed per 2000 red blood cells, following the protocol of Merino and Potti (1995) and Merino et al. (1997).

In the third experiment, house sparrows were randomly either placed near a barn owl, *Tyto alba*, which is a common predator on house sparrows in our study site, or a rock dove, *Columba livia* (a control). This experiment was initiated in spring to coincide with the annual resurgence in blood parasite infections at the beginning of the breeding season. Blood smears (as described above) were taken at the start of the experiment on 5 March and again approximately every 2 weeks on 24 March and on 3 and 17 April. The experiment was terminated after the fourth blood smear had been taken.

In a field experiment, we placed either a barn owl or a rock dove randomly chosen in a cage for 1 h at a distance of 1 m to an occupied nest-box during the early egg laying period in April–May 2002. We subsequently attempted to capture the house sparrows attending the box and injected it with PHA, as described above. The bird was kept in captivity for 6 h after which we estimated PHA response, as described above.

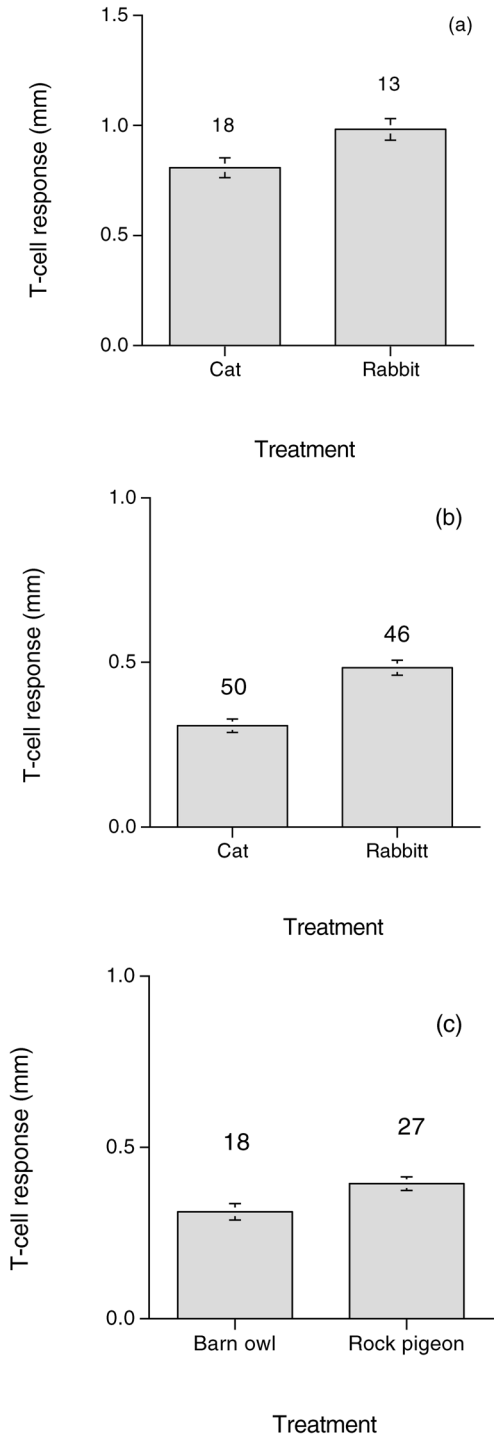

Figure 1
T-cell response (in millimeters) of house sparrows in relation to experimental treatment: Exposure to a cat or a rabbit in the first experiment (a), the second experiment (b), and the field experiment (c). Numbers are number of individuals.

Statistical methods

We investigated the effects of experimental treatment in ANOVAs and ANCOVAs with treatment as a factor, and phenotypic traits of individuals as covariates. Sex was used as an additional factor in these analyses. Intensity of blood parasitism was $\log_{10}(x + 1)$-transformed to conform with assumptions of tests based on normally distributed variables. All analyses were made by using JMP.

The significance level was set to 5%. All values reported are means (SE).

Table 1
One-way ANOVA for phenotypic response of house sparrows in relation to predation treatment

Response variable	df	MS	*F*	*p*
T-cell response	1	0.232	6.714	.015
Skin thickness	1	0.013	0.520	.477
Change in body mass	1	1.285	1.492	.232
Residual	29			

RESULTS

Predation risk and immune response

The two experimental groups did not differ significantly in phenotype for any trait in either experiment. Thus, there was no inadvertent bias in the data. Treatment had a significant depressing effect on T-cell response in both experiments (Figure 1 and Table 1). In the field experiment, T-cell response was also significantly reduced in house sparrows exposed to the barn owl compared with controls (Figure 1). The mean T-cell response was 18% smaller in the cat than in the rabbit treatment in the first experiment, on average 36% smaller in the second experiment, and on average 20% smaller in the field experiment. This contrasts with the thickness of the skin before injection, which did not differ significantly between treatments (Table 1). Similarly, there was no significant change in body mass in relation to treatment (Table 1). The effect on T-cell response remained significant after Bonferroni adjustment for multiple tests. The effect size for the first experiment, expressed as Pearson's correlation coefficient, was 0.43; in the second experiment, 0.50; and in the field experiment, 0.36. These can be considered intermediate to strong effects, sensu Cohen (1988).

We entered treatment, sex, all covariates, and all two-way interactions in a stepwise model. Three- and higher-order interactions could not be included because of small sample sizes. This procedure revealed no significant effects other than treatment for the first and the second experiment, independent of whether a forward- or a backward-elimination procedure was used.

There was a significant badge size and treatment by badge size effect on T-cell response in the first experiment (badge size: $F = 12.56$, df = 1,11, $p = .0046$, slope = 0.003 [0.001]; badge size by treatment: $F = 18.37$, df = 1,11, $p = .0013$, slope = −0.010 [0.002]). This implies that males with larger badges had a stronger T-cell response, and this effect differed between treatments. There was a clearly negative relationship in the cat treatment but a positive relationship in the rabbit treatment (Figure 2). This effect was not significant in the second experiment ($p = .099$), although it went in the same direction (Figure 2). However, a combined probability test revealed an overall significant badge by treatment interaction ($\chi^2 = 17.92$, df = 4, $p < 0.01$).

Predation risk and malarial infection

Only the *Haemoproteus* malarial parasite was commonly recorded in the house sparrows, as in previous studies

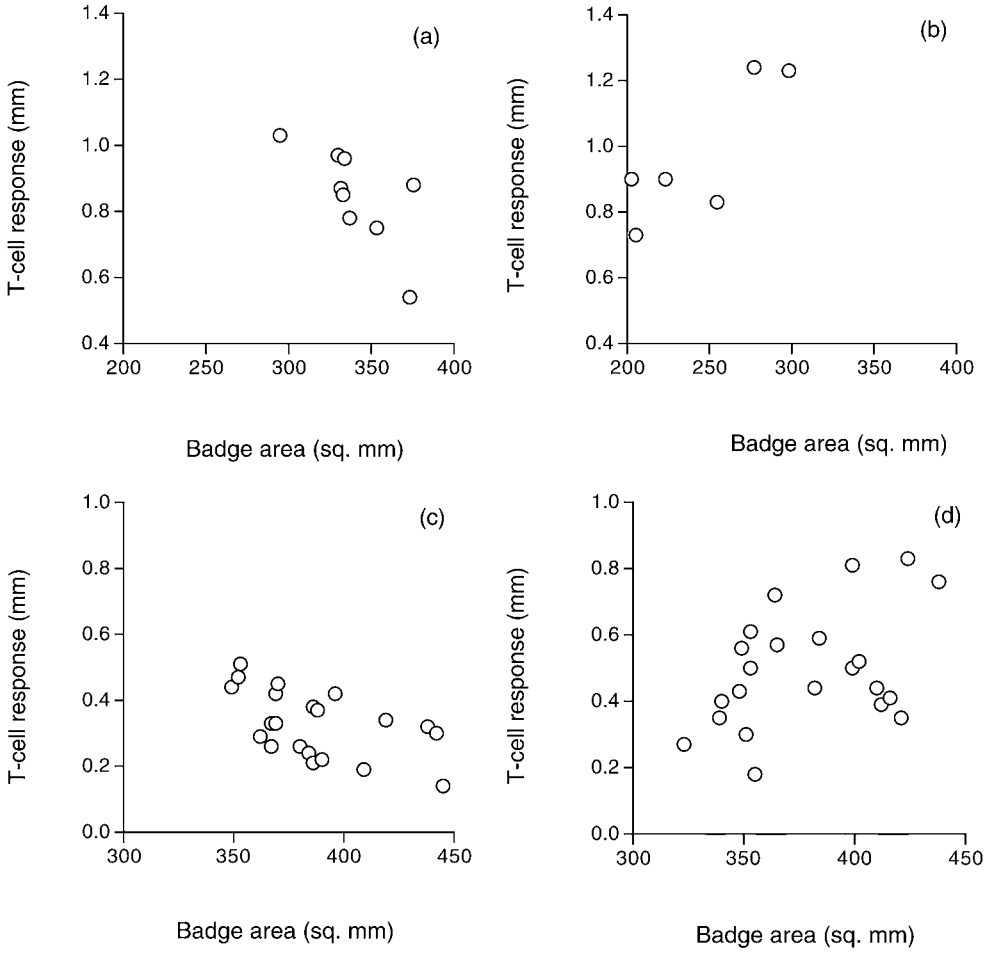

Figure 2
T-cell response (in millimeters) of male house sparrows being exposed to a cat (a, c) or a rabbit (b, d) in relation to their badge size (in millimeters squared). (a, b) The first experiment; (c, d) the second.

(González et al., 1999; Navarro et al., 2003). Other blood parasites such as *Trypanosoma* had prevalences less than 2%. Initial prevalence and intensity of *Haemoproteus* infection at the start of the experiment did not differ significantly between treatments (prevalence: $G = 3.806$, df = 3, $p = .400$; intensity: $F = 2.68$, df = 1,72, $p = .106$). Prevalence subsequently increased to 58% in the predator treatment but remained low at 15% in the control treatment ($G = 15.204$, df = 3, $p = .0022$) (Figure 3a). Similarly, intensity of infection (no. malarial parasites per 2000 red blood cells) increased significantly in the predator treatment but remained constantly low in the control treatment (Figure 3b). In a repeated-measures ANOVA, the intensity of infection was strongly affected by treatment, as were the date effect and the date by treatment interaction effect on intensity of infection (Table 2). The effect size measured in terms of Pearson's correlation coefficient was 0.38.

DISCUSSION

The main findings of this study were that (1) the presence of a predator reduced cell-mediated immune response considerably, both in an aviary and in the field; (2) this reduction depended in males on the size of a secondary sexual character; and (3) increased predation risk was associated with an increase prevalence and intensity of malarial infection with parasites of the genus *Haemoproteus*. We will discuss each of these three findings and their implications.

The main finding was that it was possible to reduce cell-mediated immune response considerably with short-term exposure to a predator. This reduction occurred both in the aviary, where house sparrows had little opportunity to escape, and in the field, when the predator was presented near the nest-box. This provides the first experimental demonstration of a direct relationship between predation risk and immune function. The reduction in T-cell response associated with experimental treatment was, on average, 18% in comparison to controls in the first experiment, 36% in the second experiment, and 20% in the field experiment (Figure 1). This equals a reduction by 1 SD from the mean value of the control group in the two experiments. This reduction contrasts with the difference in skin thickness of the same spot of the patagium before treatment, which was very small and far from significant (Table 1). Similarly, body mass did not change significantly in response to treatment. Effects of sex and malarial infection did not change the conclusion that exposure to a potential predator caused a decrease in T-cell response. Measured in term of effect size, the three experiments reported here showed strong effects (sensu Cohen, 1988), explaining 16–25% of the variance.

We hypothesize that the suppression of T-cell response caused by one experimental treatment, but not the other, was owing to an adrenocortical response (Apanius, 1998; Wingfield et al. 1998). Obviously, this suggestion has to be explicitly tested. An increase in corticosterone levels is usually expected to result in depressed immune function within days or weeks of stress, rather than hours (Apanius, 1998). However, the present experiment clearly shows that house sparrows can experience a rapid reduction in immune reaction in response to exposure to a potential predator.

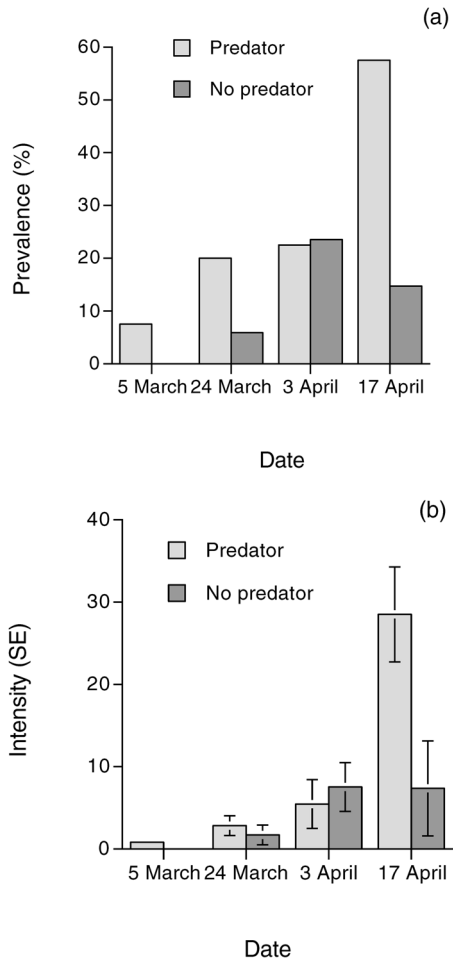

Figure 3
Prevalence (a) and intensity (b) of infection with *Haemoproteus* blood parasites in relation to date and treatment. Error bars are SE. Sample sizes are 40 for predator exposure and 34 for control treatment.

The second major finding of the study was that T-cell response covaried with the size of a secondary sexual character in male house sparrows, but this relationship depended on treatment (Figure 2). Although male house sparrows with large secondary sexual characters had stronger T-cell responses in the control group, the relationship was the opposite in the cat treatment. Thus, there was a statistically significant interaction between treatment and secondary sexual character in April but not in November–December. The positive relationship between badge size and T-cell response has previously been reported for male house sparrows during the breeding season (González et al., 1999), but not during the nonbreeding season (González et al., 1998, 1999). Hence, our findings replicate these previous results. Studies of house sparrows and other bird species have shown that males with larger secondary sexual characters have higher concentrations of circulating testosterone but lower concentrations of circulating corticosterone (González et al., 2001; Peters et al., 2001; Saino and Møller, 1995; Saino et al., 2002). These findings suggest that males with large secondary sexual characters in general should be less susceptible to stress than are males with small characters. The findings of the present study may suggest that this relationship varies seasonally, with low susceptibility of males with large secondary sexual characters to the effects of stress during the breeding season and high susceptibility during the nonbreeding season.

Table 2
Repeated-measures ANOVA for intensity of malarial infection of house sparrows in relation to predation treatment and date

Response variable	MS	df	F	p
Treatment	18.321	1	7.520	.0077
Subjects	2.436	72		
Date	13.444	3	13.012	<.0001
Date × treatment	8.052	3	7.793	<.0001
Date × subject	1.033	216		

Intensity of infection was log-transformed.

Alternatively, males with large secondary sexual characters have high levels of corticosterone when encountering a predator but low levels otherwise, whereas males with small secondary sexual characters consistently have high levels.

The T-cell response used in the present study is known to be associated with malarial infection in two different bird species, including the house sparrow (González et al., 1998; Hõrak et al., 2001), and T-cell response correlates positively with probability of survival in birds, including house sparrows (Christe et al., 1998, 2000; González et al., 1998; Hõrak et al., 1999; Merino et al., 2000; Soler et al., 1999). Because the reduction in cell-mediated immune response occurred in response to short-term exposure to a predator, it was a priori unclear whether such a reduction would affect parasite infections. However, short-term exposure to a predator significantly increased the prevalence and intensity of infection with malarial blood parasites (Figure 3). Differences in prevalence and intensity of infection between treatments may arise because intensity of infections increased to levels that allowed detection or because individuals became infected. We cannot easily distinguish between these two alternatives. Infections with blood parasites typically surge during spring to reach peak levels. These levels are at least two orders of magnitude higher than is the detection level, which is around one parasite per 2000 red blood cells. Given that prevalence differed between treatments, even at peak level of infection during spring, we can infer that sparrows exposed to predators must have become infected after exposure. To the best of our knowledge, this study provides the first successful experimental manipulation of infection with a blood parasite. This effect is likely to have been mediated by suppression of cell-mediated immunity in sparrows exposed to a predator. Chickens with reduced cell-mediated immune response have been shown experimentally to have higher susceptibility to infection with hematozoan blood parasites than do controls (Lillehoj, 1991). Although we did not measure the fitness consequences of immunosuppression in the present study, we can speculate that the increased level of infection with blood parasites is likely to result in reduced survival prospects. Previous studies in our house sparrow population have shown that individuals infected with *Haemoproteus* have an increased risk of mortality (González et al., 1999).

Parasites interact with their hosts in coevolutionary relationships whereby parasite exploitation selects for antiparasite defenses in hosts, which in turn select for more efficient parasite exploitation (see Thompson, 1994). Theoretical studies of host-parasite coevolution often only consider parasites and hosts and disregard other selective agents that may interact with the host-parasite relationship. The present study implies that predators cause reduced immune response in their prey. In other words, predators may interact indirectly with parasites through their effects on immune function in hosts. There are additional ways in which such indirect selection may affect host-parasite interactions. For example,

sexual selection for extravagantly adorned males in prime condition may select for increased investment in immune function (Møller et al., 1999), and such selection may have indirect consequences for host-parasite interactions, and thereby for predator-prey interactions. The hypothesis that bright male sexual coloration may signal unprofitable prey (Baker and Parker, 1979) suggests that males are signaling their ability to escape predators, and that this is the reason why females prefer brightly colored males. It seems possible that such a mechanism may have evolved through selection by predators of prey with poor health status and hence with poor immune function.

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