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Responding to inflammatory challenges is less costly for a successful avian invader, the house sparrow (*Passer domesticus*), than its less-invasive congener

Abstract When introduced into new regions, invading organisms leave many native pathogens behind and also encounter evolutionarily novel disease threats. In the presence of predominantly novel pathogens that have not co-evolved to avoid inducing a strong host immune response, costly and potentially dangerous defenses such as the systemic inflammatory response could become more harmful than protective to the host. We therefore hypothesized that introduced-populations exhibiting dampened inflammatory responses will tend to be more invasive. To provide initial data to assess this hypothesis, we measured metabolic, locomotor, and reproductive responses to inflammatory challenges in North American populations of the highly invasive house sparrow (*Passer domesticus*) and its less-invasive relative, the tree sparrow (*Passer montanus*). In the house sparrow, there was no effect of phytohemagglutinin (PHA) challenge on metabolic rate, and there were no detectable differences in locomotor activity between lipopolysaccharide (LPS)-injected birds and saline-injected controls. In contrast, tree sparrows injected with PHA had metabolic rates 20–25% lower than controls, and LPS injection resulted in a 35% drop in locomotor activity. In a common garden captive breeding experiment, there was no effect of killed-bacteria injections on reproduction in the house sparrow, while tree sparrows challenged with bacteria decreased egg production by 40% compared to saline-injected controls. These results provide some of the first data correlating variation in immune defenses with invasion success in introduced-vertebrate populations.

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Introduction

Biological invasions are of major conservation concern, second only to habitat destruction as a cause of species extinctions (Glowka et al. 1994; but see Gurevitch and Padilla 2004). Well-known examples of invasive species threats to resident species include zebra mussels (*Dreissena polymorpha*) that foul native mussels in the Great Lakes region of the USA (Ricciardi et al. 1998), and the brown tree snake (*Boiga irregularis*) that has caused the decline or extinction of many of Guam's endemic bird species (Wiles et al. 2003). Recently, much progress has been made toward identifying factors that contribute to the success of such invaders, including the number and size of introduction attempts, region of origin, genetic variation, and life history variables such as fecundity (Kolar and Lodge 2001; Mack et al. 2000). Escape from natural enemies such as native pathogens and parasites has also received much attention as a potential explanation for invader success (Mitchell and Power 2003; Torchin et al. 2003). Invasive animals and plants tend to host a lower diversity of parasites and exhibit a lower prevalence of infection than do native populations (Mitchell and Power 2003; Torchin et al. 2003), even many years after introduction (Cornell and Hawkins 1993; Kennedy and Pojmanska 1996). Because pathogens and parasites are capable of regulating host populations (Hudson et al. 1998), escaping natural enemies has the potential to contribute to the rapid population growth and maintenance of high densities by invasive species. However, invading organisms also encounter novel disease challenges. The potential of

introduced populations to become invasive depends upon how well they make the shift from immune defense against co-adapted pathogens to defense against evolutionarily novel ones (Lee and Klasing 2004; Muller-Scharer et al. 2004).

To predict which defense strategies should be most effective against novel pathogens, the costs as well as the benefits of immune defense must be taken into account. The disease-fighting functions of the immune system are well known, but some immune responses are costly in terms of energy, nutrients, and behavioral changes (Bonneaud et al. 2003; Klasing and Leshchinsky 1999). Moreover, strong and/or misdirected immune responses can be damaging or fatal (Mims et al. 2001). This is particularly true of responses involving systemic inflammation, which is characterized by fever, anorexia, dramatic changes in protein and nutrient metabolism, and behavioral changes, and is considered one of the most costly types of immune defense (Klasing and Leshchinsky 1999). Recently, we suggested that an immune defense strategy characterized by a dampened systemic inflammatory response might be most likely to promote invasion success (Lee and Klasing 2004). A moderated systemic inflammatory response could benefit introduced hosts in two ways. First, because many infectious organisms are highly host-specific (Mims et al. 2001), the evolutionarily novel pathogens faced by invading populations will likely often lack the mechanisms necessary to produce significant disease in a new, invading host species. For example, acanthocephalan parasites of the amphipod *Gammarus pulex* also infect the invasive amphipod *Gammarus roeseli*, but the pathological change in behavior that results in increased predation risk in the native amphipod host is not seen in the invasive host (Rigaud and Moret 2003). If introduced-vertebrate hosts experience similar infections by pathogens or parasites that are not able to produce significant disease in the new host, vigorous immune defenses against these challenges could incur high costs with little benefit. Second, in addition to co-opting resources, a naïve host's own overly vigorous or inappropriate systemic inflammatory response against novel pathogens can itself be the cause of severe host illness or mortality (Mims et al. 2001). This is because pathogens that successfully colonize new host species are likely to lack the adaptations that allow more co-evolved pathogens to avoid eliciting a strong immune response (Mansfield and Olivier 2002). As a result, invaders with dampened systemic inflammatory responses might not only avoid unnecessarily allocating resources to defense that could be used for reproduction or growth but could also be more likely to survive novel infections.

Comparisons of phylogenetically related species pairs have long proven useful in understanding ecological variation between species (Cooper 1999; Ghalambor and Martin 2000; Klein et al. 1997; Martin et al. 2000). The high degree of similarity that often exists between closely related species makes it possible to study interspecific variation in a character of interest while minimizing variation due to the multitude of other possible differences between the species. While a single species pair comparison cannot definitively demonstrate relationships between ecological variables, these kinds of data are useful both for generating testable hypotheses and for contributing data to larger-scale comparative analyses. For this study, we compared costs of inflammatory immune responses in introduced-populations of two species that exhibit different degrees of invasion success: the house sparrow (*Passer domesticus*) and its congener; the Eurasian tree sparrow (*Passer montanus*) (hereafter referred to as "tree sparrow"). In North America, the house sparrow is highly invasive while the tree sparrow remains restricted to the region surrounding St. Louis, MO, USA (36°N, 90°W) where it was originally introduced (Long 1981). These two species share several important traits: both are human commensals, gregarious, largely sedentary, and socially monogamous breeders (Summers-Smith 1988).

We hypothesized that immune defense strategies could contribute to the differential invasion success of these two species if introduced tree sparrows mount more vigorous inflammatory responses than do house sparrows. This is because some behavioral and life history attributes that might be important for invasiveness are likely to be sensitive to differences in investment in immune defenses. Here, we evaluate this hypothesis against the background of life history traits of these two sparrows in North America. Tree sparrows in North America probably have lower adult survival rates (35%) (Anderson 1978) than do house sparrows (57%) (Lowther and Cink 1992). In contrast, tree sparrows in Missouri, USA, had higher average reproductive rates (6.7 young-fledged per pair per year) than did house sparrows (4.5 young per pair per year on average) (Anderson 1978). Competitive ability also differs between house and tree sparrows, at least where they co-occur in Europe. Studies in western Europe have shown tree sparrows to be inferior competitors for nesting sites compared to house sparrows (Summers-Smith 1995).

Differences in investment in inflammatory responses could contribute to the differing degrees of invasion success exhibited by house and tree sparrows by causing or increasing disparities in survival or competitive ability between the two species. Reproductive rates might also be differentially affected by investment in inflammatory responses, particularly during disease outbreaks. To understand the effects immune challenges might have on behavior and life history traits, we measured metabolic, behavioral, and reproductive costs of responding to multiple challenges known to elicit inflammatory responses in birds. We report the downstream costs of responding to inflammatory challenges rather than measuring the immune responses directly because our goal here is to address ecological endpoints of immune responses, and because measuring both behavioral and physiological responses during our experiments was not always compatible. This study

provides the first data addressing the hypothesis that the introduced-populations that invest less in systemic inflammatory responses should be the more successful invaders.

Materials and methods

Experiment 1: measuring the energetic cost of an inflammatory response

We captured house and tree sparrows from sympatric populations in and around St. Louis, Missouri (36°N, 90°W) and Meredosia, Illinois, USA (40°N, 90°W) in July 2002 using mist nets. Food [Kaytee Supreme Wild Bird Seed (Kaytee Products Inc., Chilton, WI, USA) supplemented with hard-boiled chicken eggs] and water were available to the birds ad libitum. To induce an immune response, we injected 100 μl of 1 mg/ml phytohemagglutinin (PHA-P, Sigma L-9017) in saline subcutaneously into the wing web of each treatment bird ($n=6$ tree sparrows, $n=8$ house sparrows) following a standardized method (Smits et al. 1999). The dosage of 100 μg of PHA is an intermediate dosage within the range used in small birds in published studies (Moller et al. 2001; Smits et al. 1999). Control birds ($n=3$ of each species) were injected with saline. PHA injected subcutaneously causes edema and T-cell mediated infiltration of the tissue by granulocytes, macrophages, and lymphocytes (McCorkle et al. 1980). Swelling at the injection site is typically used as an index of cell-mediated immunity (Grasman 2002), but PHA has also been shown to induce a systemic inflammatory response in birds (Adler et al. 2001). We measured metabolic rate continuously following acclimation for 24 h prior to and 48 h following PHA injection using open flow, push-through respirometry (Martin et al. 2003; Withers 1977). Birds were allowed to acclimate to the metabolic chambers (5-l plastic containers) for 24 h prior to the beginning of the experiments. Light cycle, temperature, and relative humidity were equivalent to the conditions that free-living birds were experiencing at that time and location (15L:9D, average of 26.7°C and 60.9% RH). Here, we report the minimum per hour oxygen consumption measured for each bird at three time points (12 h before and 12 h and 36 h after PHA injection) during daylight hours, corrected for (mass)$^{3/4}$. Body mass was measured daily during the experiment to the nearest 0.1 g.

Experiment 2: measuring the behavioral costs of an inflammatory response

We captured house and tree sparrows for this and the following experiment in January 2003, from the same populations as in Experiment 1 (different individuals were used in all experiments). We conducted Experiment 2 in winter to assure that the differences between species we observed in July were not dependent on breeding stage, and used a different immune challenge, lipopolysaccharide (LPS) from *Escherichia coli* (Sigma F8666), to check whether the species differences we saw in Experiment 1 were antigen-dependent. We injected birds intraperitoneally with 100 μl of 0.1 mg/ml LPS in saline ($n=6$ tree sparrows, $n=7$ house sparrows). This dosage is similar to that used to elicit behavioral and reproductive responses from house sparrows in a previous study (Bonneaud et al. 2003). Control birds received saline alone ($n=6$ tree sparrows, $n=4$ house sparrows). In birds, LPS induces a systemic inflammatory response characterized by changes in protein and mineral metabolism (Klasing 1998) and decreased activity and food intake (Bonneaud et al. 2003; Johnson et al. 1993). We monitored locomotor activity for 24 h before (day 0) and 24 h following LPS injections (day 1). Birds were housed in individual cages equipped with an infrared light beam and reflector located between two perches. An automated activity monitoring system (Mini Mitter Co. 1999) recorded an event each time the light beam was broken by a bird moving through it. We calculated change in activity due to LPS injection as percent change in activity from day 0 to day 1. Data are presented in terms of percentages because of the high variability in sensitivity of the automated sensors; because each bird was recorded by the same sensor both days, this should correct for an effect of monitor sensitivity. Throughout the experiment, photoperiod was held constant at 10L:14D.

Experiment 3: measuring the reproductive costs of an inflammatory response

The captive breeding experiment took place in two large indoor aviaries (20′×12′×8′) furnished with wooden dowel perches and nest boxes. Treatment and control birds of each species were kept in separate but adjacent sections of a large aviary, and the number of males within each aviary equaled the number of females. We provided nest boxes in excess of the number of females per group (ten boxes per 5–8 females). Nesting material (hay and chicken feathers) and birdseed supplemented with mealworms (*Tenebrio molitor*), hard-boiled chicken eggs and oranges, and water were available ad libitum. We monitored nests every 3 days during the breeding season (May 15–August 30, 2003) and recorded number of eggs and chicks in each. Two tree sparrow females were found to move between aviaries early in the experiment and we removed them from the aviaries and subsequent data analyses. To mimic a natural infection as closely as possible, we used whole killed-bacteria as the immune challenges in this experiment. We injected treatment females of each species ($n=6$ tree sparrows, $n=7$ house sparrows) intraperitoneally (IP) with killed *E. coli* (American Type Culture Collection#11303)

approximately 2 weeks after egg-laying began, and *Salmonella choleraesuis* serotype *typhimurium* (ATCC# 700720) 6 weeks later. We used two types of bacteria so that immunological memory induced by the first injection would not affect the birds' responses to the second injection. The bacterial concentrations we used (1×10^4 and 1.5×10^6 bacteria in 100 μl saline, respectively) are at the low end of the range of live bacterial concentrations that have been shown to cause infection when injected into chickens (Holt 1993; Johnson et al. 2001). We injected control females twice with physiological saline ($n=5$ tree sparrows, $n=6$ house sparrows). Because prolonged observations were disruptive to breeding, we were not able to assign individual females to particular nest boxes. Therefore, we estimated individual egg production as eggs per nest box. While this meant that we were unable with certainty to assign parentage to each egg, conspecific brood parasitism is relatively rare in house sparrows (Kendra et al. 1988) and we could find no mention in the literature of it occurring in tree sparrows. In the three cases in which a single female had clearly used more than one nest box (i.e., more nest boxes were used than there were females), we combined the data for two boxes with no overlap in their laying dates to represent a single female.
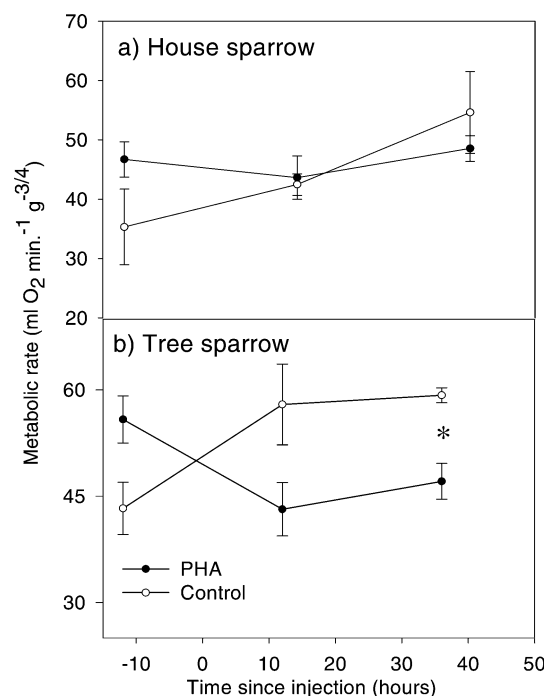
Data analysis

In all experiments, to test for the effects of treatment we compared treatment groups of each species with their conspecific control group, rather than comparing treatment groups between species. Comparing treatment and control birds within each species allowed us to control for any species-specific effects not due to treatment (such as response to captivity or any disturbances). We tested for species and treatment effects on metabolic measurements in Experiment 1 using repeated measures ANOVA. During one 24-h period, due to errors in recording the species, the identity of the individual birds was not known; in this case, we used the species mean for that time period (five house sparrow and two tree sparrow time points, baseline day before injection). Three other time points (2 tree sparrows, one at 12 h and one at 36 h, and one house sparrow at 12 h) were lost due to problems with the respirometry equipment and were replaced with the mean value for that group at that time point. Metabolic rates at individual time points were compared between treatment and control groups using post-hoc *t*-tests. We compared activity measurements in treatment and control house and tree sparrows in Experiment 2 using Mann–Whitney *U*-tests because the data were non-normal. We tested for species and treatment effects on breeding parameters in Experiment 3 using an ANOVA when the data were normally distributed; otherwise we used a Kruskall–Wallis test. For all statistics we used SPSS 10.0 (SPSS 1999).

Results

Experiment 1: metabolic effects of PHA challenge

Phytohemagglutinin injection did not affect night-time minimum hourly oxygen consumption during the first (0–9 h) or second (24–33 h) nights following injection in tree or house sparrows. During daytime hours, PHA injection had a significantly different effect on metabolic rate in tree and house sparrows (repeated measures ANOVA, species × treatment $F_{1,16}=5.942$, $P=0.027$). Daytime metabolic rate was not significantly different between house sparrow groups (time × treatment term $F_{2,18}=2.070$, $P=0.155$, Fig. 1a). In tree sparrows, PHA caused a 26% decrease in metabolic rate at 12 h following injection and a 21% decrease at 36 h relative to saline-injected controls; (time × treatment term $F_{2,14}=12.222$, $P=0.001$, Fig. 1b). Post-hoc tests showed that PHA-injected tree sparrows had nearly significantly lower metabolic rates at 12 h ($t_7=2.351$, $P=0.051$) and significantly lower metabolic rates at 36 h post-injection ($t_7=3.270$, $P=0.014$) relative to conspecific controls. Lower daytime metabolic rates of PHA-injected tree sparrows relative to controls with no difference in nighttime metabolic rate is consistent with a difference in daytime locomotor activity, and suggests that tree sparrows exhibited more marked

Fig. 1 Daytime metabolic rate in **a** house and **b** tree sparrows injected with PHA or saline (control). Injection took place at 0 h. The effect of treatment differs significantly between species (see text). *Bars* represent one standard error; *asterisks* denote significant differences
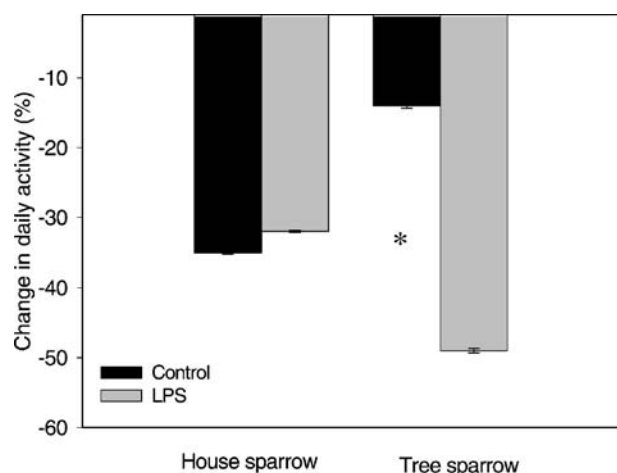
"sickness behavior" than did house sparrows in response to PHA. Body mass was unaffected by treatment in both species.

Experiment 2: effects of LPS on locomotor activity

Total daily locomotor activity was significantly reduced in tree sparrows injected with LPS relative to conspecific saline-injected controls over the 24 h following injection (Mann–Whitney test, $U = 5.0$, $P = 0.041$). There was no effect of LPS on house sparrow activity ($U = 12.0$, $P = 0.705$, Fig. 2). There was no change in body mass due to treatment in either species.

Experiment 3: reproductive effects of killed-bacteria injections
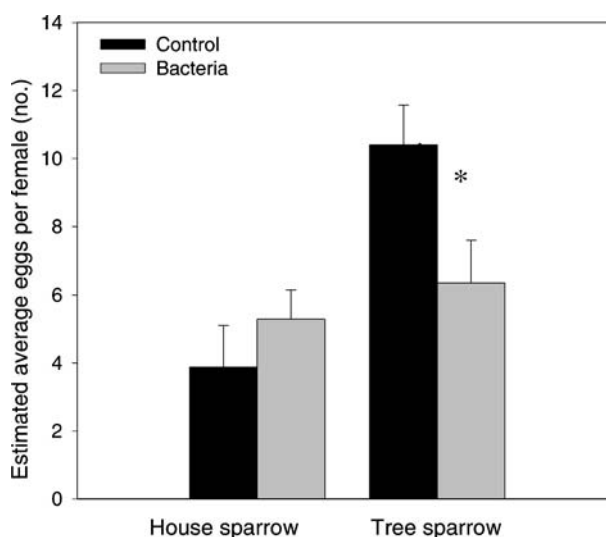
In tree sparrow females, but not in house sparrow females, bacterial injections caused a significant decrease in egg production relative to that of conspecific saline-injected controls (ANOVA: dependent variable eggs per female; species × treatment $F_{1,22} = 5.451$, $P = 0.029$; independent samples t-test on treatment effect within species: house sparrows, $t_{13} = -0.911$, $P = 0.379$; tree sparrows, $t_9 = 2.299$, $P = 0.047$; Fig. 3). This was due to a tendency for lower total number of clutches laid by bacteria-injected tree sparrows (though this difference was not significant, see Table 1). Hatching and fledging rates were low in both species and did not differ significantly between treatment and control birds of either species (Table 1).

Discussion

Introduced species leave many native pathogens behind when transported to a new area, but they also encounter novel disease challenges (Torchin et al. 2003). We hypothesized that an immune defense strategy characterized by dampened systemic inflammatory responses might favor invasiveness, because these responses are costly, and have the potential to be overly vigorous and misdirected when elicited by novel pathogens, and therefore dangerous (Lee and Klasing 2004). Here, we show that inflammatory immune challenges cause no measurable metabolic or behavioral changes or reproductive decrease in individuals from an invasive house sparrow population, while the same challenges are behaviorally costly and negatively affect reproductive output in introduced and less-invasive tree sparrows. Our results suggest that when the North American populations of house and tree sparrows that we studied experience infections in nature, the magnitude of inter-specific differences in immune defense costs could be great enough to result in differences in population growth rates, either indirectly via behavioral differences that could impact survival or competition for limited resources such as nest sites, or by directly affecting reproductive output. If the patterns we have shown in these two populations are a general phenomenon, differential investment in costly immune defenses could contribute to the house sparrow's greater invasion success in North America relative to the tree sparrow. More generally, this study provides some of the first evidence that immune defenses might be involved in mediating invasion success of introduced-vertebrates (see also Moller and Cassey 2004).

Fig. 2 Change in locomotor activity in house and tree sparrows over the 24 h following LPS or saline injection (control). Values are given as the percent change relative to the day prior to challenge. *Bars* represent one standard error; *asterisks* denote significant differences

Fig. 3 Estimated number of eggs laid per female house and tree sparrow injected with killed-bacteria or saline (control.) *Bars* represent one standard error; *asterisks* denote significant differences

Table 1 Breeding parameters in house sparrows and tree sparrows injected with killed-bacteria or saline

	House sparrow			Tree sparrow		
	Bacteria-injected	Control		Bacteria-injected	Control	
Sample size (n)	7	8		6	5	
Hatch rate (%)	20.4 ± 10.6	27.8 ± 12.7	$U=19.5, P=0.836$	2.1 ± 2.1	7.98 ± 4.09	$U=9.0, P=0.205$
Fledge rate (%)	41.7 ± 25.0	53.3 ± 29.1	$U=5.5, P=0.853$	0	50.0 ± 28.9	$U=0.5, P=0.346$
Clutch size	2.49 ± 0.56	2.5 ± 0.75	$t_{13}=-0.012, P=0.090$	1.97 ± 0.31	2.05 ± 0.14	$t_9=-0.223, P=0.829$
Number of clutches	2.6 ± 0.48	1.4 ± 0.74	$t_{13}=1.794, P=0.096$	3.2 ± 0.4	4.4 ± 0.51	$t_9=-1.929, P=0.086$

Sources of variation in responses to immune challenges

A number of variables could contribute to the immunological differences between house and tree sparrows that we observed. If our hypothesis is correct and dampened inflammation is in fact favorable for invaders, then the differences we show here could be the result of post-introduction evolution of the house sparrow toward a defense strategy that is more appropriate in the new environment, and the lack of such evolution in the tree sparrow population. The tree sparrow's initial population size in the USA was small (between 20 and 40 birds) (Long 1981), and the lack of genetic variation could have constrained immune defense evolution. Alternatively, the differences we observed could reflect different phenotypic responses to a similar infection history. Perhaps house sparrows have more plastic immune defenses than do tree sparrows, and can more easily adopt a defense strategy characterized by low investment in inflammation. Finally, infection histories themselves might have differed between the house and tree sparrows we studied, and could have affected their responses. More extensive captive breeding studies could allow us to distinguish between these alternative mechanisms.

Our experimental design might have influenced the results in ways that were unrelated to true species differences in inflammatory responses. In the captive breeding experiment, because we had only one treatment and control group of each species, each in a separate aviary section, we cannot completely rule out the potential effect of aviary. However, for several reasons, we believe that this was unlikely to have strongly affected the results. Control and treatment birds of each species were housed within one large aviary that was divided into half by a thin nylon mesh curtain. Therefore, environmental conditions should have been identical. In addition, the birds in adjacent aviaries could see and hear each other; so any social influences attributable to vocalizations or visual perception of conspecific numbers were shared.

Throughout the captive breeding study, house and tree sparrows exhibited overall low laying, hatching, and fledging rates relative to free-living sparrows (Summers-Smith 1988). A low reproductive rate during the first year in captivity is the rule rather than the exception for captive birds (E. Gwinner, personal communication). This could be due to the stress of captivity, and at least in our case due to inadequate food for nestlings. These factors should have been constant between control and treatment birds and should not have affected the relative differences in reproduction due to bacteria injection between groups. However, stress and immune responses might interact differently in the two species. Differences in stress-immune interactions could produce differences in inflammatory responses in the wild, and could also lead to exaggerated species differences when exposed to captivity stress. Additionally, our measures of investment in systemic inflammation were at the relatively coarse level of metabolic, behavioral and reproductive effects. It is possible that North American house sparrows do in fact mount vigorous systemic inflammatory responses, but that the associated behavioral changes have somehow become decoupled from the inflammatory mechanisms. Alternatively, our results might reflect an overall downregulation of inflammation in North American house sparrows. Additional indices of the systemic inflammatory response, such as acute phase proteins produced by the liver during systemic inflammation, could help distinguish between these two possibilities.

In other house sparrow populations, energetic, reproductive, and behavioral costs of immune defenses associated with inflammation have been demonstrated (Bonneaud et al. 2003; Martin et al. 2003). In contrast to our results, Martin et al. (2003) reported significant elevation in resting metabolic rate in North American house sparrows following PHA injection. However, that experiment was carried out at a different time of year (January and February); we conducted Experiment 1 with birds during the summer (July), a time when PHA swelling in temperate house sparrows is at its lowest (Greenman et al. 2005; Martin et al. 2004). Interestingly, Bonneaud et al. (2003) demonstrated that immune challenge with LPS causes reductions in reproductive output and locomotor activity in a European population of the house sparrow, while we could find no such effects of the same immune challenge on activity in individuals from a North American population of this species. Similar to the differences between house and tree sparrows, this pattern could reflect differences in phenotypic responses to past infections, or evolutionary change of North American house sparrows' immune defenses post-

introduction; again, it would be possible to distinguish between these scenarios with more extensive captive breeding studies.

Conclusions

Many factors could contribute to the house sparrow's greater invasion success in North America compared with the tree sparrow. Most often invoked are the direct competition between the two species and the differences in the introduction effort (Long 1981; Summers-Smith 1995). In general, dispersal ability, high reproductive rates, ability to compete with native species for resources, and association with humans are thought to be important predictors of the spread of non-native birds (Duncan et al. 1999; Oconnor 1986; Sakai et al. 2001). Here, we do not suggest that differences in immune defenses are an alternative explanation for the contrasting introduced distributions of the house and tree sparrow, but rather an overlooked and potentially important contributing factor. While further research encompassing additional species as well as additional house and tree sparrow populations is needed to increase our understanding of the potential role of the immune system in invasion success, these results show that the study of immune defense strategies could provide a promising avenue of research that could shed light on processes underlying the dynamics of biological invasions.

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