



Experimentally increased social competition compromises humoral immune responses in house finches

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Abstract

Although social behavior can substantially influence an individual's physiology, few studies have examined whether intraspecific competition compromises individual immunocompetence. We experimentally manipulated the intensity of social competition in captive non-breeding house finches (*Carpodacus mexicanus*) by supplying few (high competition) or many (low competition) feeding sites. We tested whether elevated levels of social competition caused individual changes in aggression rates, humoral immunity, body mass, and baseline and stress-induced corticosterone concentrations. We also examined whether physiological responses to social competition were related to an individual's social status. We found that house finches under high social competition had significantly higher aggression rates, lower antibody responses, and lost more body mass. Within flocks, dominant individuals mounted stronger immune responses in both competition treatments. Our statistical power to detect differences in circulating corticosterone concentrations was low, but we did not find any support for the hypothesis that corticosterone concentrations mediate immunosuppression among or within flocks: baseline and stress-induced corticosterone concentrations did not differ under high and low social competition, were unrelated to individual social status, and did not predict the extent of immunosuppression among individuals. Overall, we documented that two universal components of social behavior, intraspecific competition and social status, modulated the strength of a humoral immune response in house finches.

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Introduction

The interaction between social behavior and individual physiology has been of long-standing interest to ecologists (Christian and Davis, 1964). The plasticity of the vertebrate immune response (e.g., Norris and Evans, 2000) and its responsiveness to social stressors (e.g., Lindström et al., 2005a; Møller et al., 1993) has led to recent interest in immunocompetence as a common physiological link between intraspecific interactions and individual fitness (Lochmiller, 1996; Saino et al., 2000; Svensson et al., 2001). Correlative relationships between population density, physiological stress, and immunocompetence are

best documented in mammals (e.g., Dobrowolska et al., 1974; Geller and Christian, 1982; Wolk and Kozłowski, 1989). Furthermore, Svensson et al. (2001) demonstrated in female lizards that higher territory densities suppress antibody responses, and additional work indicated that stress hormone levels likely mediate this relationship (Comendant et al., 2003). However, the proximate linkages among population density, physiological stress, and immunocompetence remain unclear.

Two types of mechanistic pathways, either direct or indirect, may explain why individuals show suppressed immune responses (Svensson et al., 2001; Tella et al., 2001) or elevated parasite prevalence (Opplinger et al., 1998) at high densities. First, population density may influence immunity directly via increased intraspecific aggression, which can alter circulating levels of hormones. Second, population density may affect immunity indirectly via environmental factors such as food availability, which also change with density (Lochmiller, 1996). In order to demonstrate direct relationships between intraspecific

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competition and immunity, the intensity of social interactions alone must be manipulated while density is held constant. Studies of laboratory and domesticated species have documented that social interactions and aggression in particular can mediate individual immunity (Barnard et al., 1998; De Groot et al., 1999, 2001; Gross and Siegel, 1973). In many cases, the intensity of social aggression is associated with the magnitude of resulting changes in immune parameters (Devoine et al., 2003; Tuchscherer et al., 1998).

In this captive study, we test the impact of social competition on immunocompetence in house finches (*Carpodacus mexicanus*), a highly social species that competes aggressively for feeding sites during the non-breeding season (Thompson, 1960). We held density constant and manipulated the intensity of intraspecific competition to examine its impact on humoral antibody responses to sheep red blood cells (SRBCs), an antigen to which house finches have never been exposed. We also measured concentrations of the hormone corticosterone, which may mediate relationships between social competition and immunity (Comendant et al., 2003) since this hormone both responds to social interactions (e.g., Silverin, 1998) and can act in an immunosuppressive manner (e.g., Apanius, 1998; Nelson et al., 2002). Furthermore, corticosterone concentrations have been linked to social status (Belthoff et al., 1994) and condition (Duckworth et al., 2001) in previous studies of house finches. We tested specifically whether (1) elevated social competition would reduce antibody responses to SRBCs and increase circulating levels of corticosterone concentrations; (2) whether the impact of social competition on immunocompetence and corticosterone concentrations would depend on an individual's social status; and (3) whether the extent of immunosuppression would be inversely correlated with corticosterone concentrations.

Methods

Study population

House finches are small (20 g) passerine birds that winter in large flocks throughout North America (Hill, 1993). From January to March 2002, we captured sixty-four finches (1:1 sex ratio) in Tompkins (42°51'N, 76°34'W) and Tioga Counties (42°31'N, 76°19'W), New York, using mist nets and hand-built cylindrical wire-mesh cage traps. Trapping was conducted under permits from the New York State Department of Environmental Conservation (No. LCP 99-039) and the US Fish and Wildlife Service (PRT 802829). At capture, we measured unflattened wing length at the longest primary (0.5 mm), tarsus length (0.1 mm), and mass (0.1 g).

Experimental protocol

We randomly assigned individuals to eight same-sex flocks containing eight individuals (4 male flocks, 4 female flocks). Within a flock, each individual was randomly assigned two uniquely colored leg bands for behavioral observations. Flocks were housed in indoor free-flight rooms (9.5' × 13' × 8') at moderate temperatures (10–18°C) and day length (10L:14D). We provided each flock with a single live conifer, four wooden dowels, a single plastic water dish, and at least one feeder containing ad libitum black oil sunflower seed. The number of feeders that a flock received varied with the social competition treatment (see below). All procedures for maintenance and care of the birds were conducted according to protocol 00–90 approved by the Cornell University Institutional Animal Care and Use Committee (IACUC).

We randomly assigned flocks to one of two treatments: high competition (8 individuals: 1 feeder) or low competition (8 individuals: 4 feeders). We equally distributed the treatments across our single-sex flocks in a two by two factorial design (Table 1). Both competition treatments received ad libitum access to sunflower seeds (i.e., the seed level never fell below the highest feeding hole). However, individuals in the high competition treatment flocks were forced to feed from a single feeder (8 individuals: 1 feeder), while individuals in the low competition flocks had four feeders from which to choose (8 individuals: 4 feeders).

Our experimental protocol for each flock was identical (Table 2). We allowed individuals to acclimate to their flock for 3 days and then quantified dominance behavior for each flock on days 4–6 (see below). On day 6, we collected blood samples for corticosterone levels (see below) and pre-injection antibody levels. We then injected five randomly selected individuals from each flock with SRBCs and three control birds with saline solution (see below). Individuals were left undisturbed for 8 days, with the exception of brief captures on days 9 and 12 to assess morphological condition. There was no mortality during the course of the experiment, and all individuals were released into the wild following the completion of the study.

Quantifying humoral immunity

We intraabdominally injected individuals with 5×10^7 SRBCs (MP Biomedicals, Irvine, CA, USA) suspended in 100 μ l phosphate-buffered saline (Deerenberg et al., 1997). No bruising was observed at the site of injection. For antibody measurement, we collected 100 μ l of blood from the brachial vein prior to and 8 days following injection with SRBCs. We quantified antibody levels blind to individual identity using a base-2 serial dilution hemagglutination assay, subtracting pre-injection antibody levels from post-injection levels. Antibody titers are given as the reciprocal of the highest \log_2 dilution at which an individual's plasma showed positive hemagglutination.


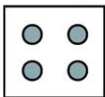
Quantifying corticosterone levels

On days 6 and 14 of the experiment, we took two small (50 μ l) blood samples within 3 min of capture and at 30 min following capture to obtain measurements of baseline (3 min) and stress-induced (30 min) corticosterone concentrations (Romero and Romero, 2002). Individuals were sampled in random sequence during both sampling periods, but due to manpower constraints, we obtained baseline samples from only about half of the individuals in each flock within the necessary 3-min time window. To ensure comparable treatment of all individuals, we bled the remaining individuals in a flock even if the required sampling time was exceeded.

Blood samples for corticosterone analysis were refrigerated immediately upon sampling. Within 3 h of sampling, we removed the plasma after centrifugation for 5 min at 7000 g and stored samples at -20°C . All blood samples were labeled using a randomized ordinal system to allow blind analysis. We quantified the plasma corticosterone concentration of each sample using the method described in Wingfield et al. (1992). In brief, corticosterone levels were extracted from small volumes (10–20 μ l) of plasma and detected with a direct radio-immuno assay. The within-assay variation was 8–14% and the between-assay variation was 8%; samples were adjusted for recoveries and the average recovery was 75%.

Our final sample sizes were as follows: baseline day 6 ($n = 42$), stress-induced day 6 ($n = 52$), baseline day 14 ($n = 29$), stress-induced day 14

Table 1
Factorial design for the competition treatment

| | High competition 1 feeder | Low competition 4 feeders |
|---------|--|---|
| |  |  |
| Males | 2 flocks | 2 flocks |
| Females | 2 flocks | 2 flocks |

Circles represent feeders.

Table 2
Experimental Protocol for each flock (SRBCs—sheep red blood cells)

| Day | Protocol |
|-------|--|
| 0 | Randomly assigned individuals to flock |
| 4–6 | Quantified dominance status at feeders |
| 6 | Measured corticosterone concentrations; injected with SRBCs or saline |
| 12–14 | Requantified dominance status at feeders |
| 14 | Measured corticosterone concentrations; final blood sample for SRBC antibodies |
| 15 | Released individuals into the wild |

($n = 33$). We analyzed baseline and stress-induced corticosterone concentrations separately since we frequently did not have both measures for the same individual. We were able to make within-individual comparisons for birds sampled at both days 6 and 14 for baseline corticosterone ($n = 21$) and stress-induced corticosterone ($n = 28$). We found no evidence that keeping birds in captivity altered circulating levels of corticosterone, since corticosterone concentrations closely matched those measured in wild-ranging house finches in the study area at this time of the year (Lindström et al., 2005b).

Behavioral observations

We quantified two measures of social behavior, aggression rate, and dominance status, prior to and following injection with SRBCs. We collected behavioral data from an attached blind observation room and standardized the motivation to feed by removing all food sources 2 h prior to observation. We calculated aggression rates on days 6 and 14 by recording the total number of displacements (wins and losses combined) per individual during a 1-h observation period. We defined displacement as the aggressive replacement of the focal individual by another bird while feeding. We calculated “changes in aggression rate” for each individual as the number of displacements experienced on day 14 minus the number experienced on day 6.

We conducted dominance observations for 1 h per flock across three consecutive mornings (days 4–6 and days 12–14), by recording all pairwise displacements and identifying the winner and loser. For each flock, we observed at least 5 independent displacement interactions between every pairwise combination at the feeder. We assigned an individual as the winner of a dyad if it dominated more than 70% of the pairwise interactions. In dyads where neither individual won more than 70% of the interactions, we quantified the pair as tied. We calculated dominance status for each individual as the proportion of flockmates that it dominated (ties were considered as dominating 0.5 individuals). Thus, an individual with dominance status of 1.0 dominated all other individuals. Individual dominance status at the two time periods (days 6–8 and 12–14) was highly correlated ($n = 64$, $r = 0.93$, $P < 0.0001$), and we used dominance status following injection (days 12–14) for all analyses.

Statistical analyses

We performed all statistical analyses using JMP 5.0 and SAS 9.0 (SAS Institute). We used mixed models, including individual and flock as random effects, to examine (1) the effect of SRBC vs. saline injection on body mass and corticosterone levels, (2) the effect of the competition treatment, dominance status, and sex on multiple dependent variables (antibody response, aggression rate, body mass, corticosterone levels). All two-way interaction terms were initially included but were removed from the final model if not significant. We used general linear models to examine the effect of the competition treatment and dominance status on the paired measures (day 14–day 6 for aggression rate, body mass, corticosterone levels, and dominance status) since within-individual measures inherently control for random sources of variation.

For the statistical analyses, we used square root transformations of corticosterone levels, arc-sin square root transformation of dominance status, and log transformations of changes in mass to meet the assumptions of

normality. For all figures and tables, we present untransformed means and standard errors for ease of interpretation.

Results

SRBC injection

Birds injected with SRBCs or saline did not significantly differ in changes in mass, aggression rate, or dominance status following injection ($F_{1,54} < 1.77$; $P > 0.19$; Mixed Model including flock (random), individual (random), competition treatment, dominance, sex, SRBC vs. saline). Furthermore, saline and SRBC-injected birds had equivalent baseline ($F_{1,28} = 0.04$; $P = 0.84$) and stress-induced corticosterone concentrations at day 14 ($F_{1,23} = 0.25$; $P = 0.62$; Mixed Model including flock (random), individual (random), competition treatment, dominance, sex, SRBC vs. saline).

Competition treatment effects

The competition treatment successfully intensified aggressive interactions within the flock (Fig. 1): birds in high competition flocks maintained the same number of aggressive interactions at the feeder from days 6 to 14 while birds in the low competition treatment showed a significant decrease in aggression rate over time ($F_{1,63} = 5.22$; $P = 0.03$; GLM including treatment, flock, dominance, and sex). The competition treatment resulted in individual changes in mass throughout the experiment (Fig. 2a; $F_{1,63} = 4.90$; $P = 0.03$; GLM including treatment, flock, dominance, and sex): birds in the high competition treatment decreased in body mass from day 6 to 14 (mean \pm SE = $-0.26 \text{ g} \pm 0.13$) while birds in the low competition treatment maintained a constant mass (mean \pm SE = $0.06 \text{ g} \pm 0.13$).

The competition treatment suppressed antibody titers to SRBCs (Fig. 2b; $F_{1,28} = 5.63$, $P = 0.024$; Mixed Model including flock (random), individual (random), treatment,

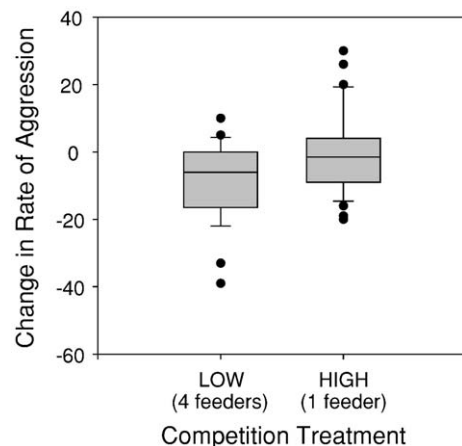


Fig. 1. The competition treatment was associated with changes in the rate of aggression (day 12–day 6) that each individual experienced at the feeder. Box plots show the median (center line), 25th percentile (lower line), and 75th percentile (upper line). Whiskers indicate the 10th and 90th percentiles.

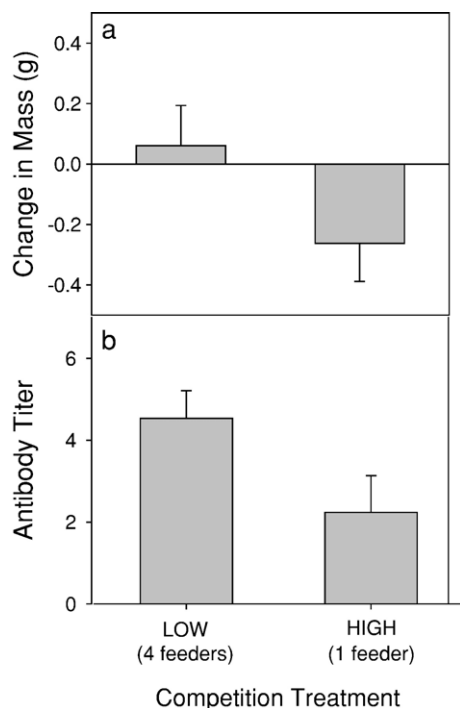


Fig. 2. The competition treatment influenced (a) within-individual changes in mass and (b) the strength of antibody response to sheep red blood cells. Error bars represent one standard error around the mean.

dominance, and sex). Individuals in the high competition treatment mounted significantly lower antibody responses (mean \pm SE = 2.24 ± 0.79) than birds in the low competition treatment (mean \pm SE = 4.53 ± 0.84). Furthermore, birds in the high competition treatment were significantly less likely to mount an immune response (titer ≤ 0) than birds in the low competition treatment ($n = 32$, $\chi^2 = 7.17$, $P = 0.008$).

The social competition treatment had no detectable effect on average corticosterone levels (baseline or stress-induced) at either time period ($F_{1,4,94} < 0.39$, $P > 0.54$; Figs. 3a, b; Mixed Model including flock (random), individual (random), treatment, dominance, and sex). Retrospective power analyses indicated that our power to detect treatment effects on absolute corticosterone concentrations was inadequate (baseline day 6: 6.0%; stress-induced day 6: 6.6%; baseline day 14: 15.6%; stress-induced day 14: 41.4%), due in large part to substantial unexplained variation among individuals: covariance parameter estimates from the Mixed Models revealed that random variation among individuals accounted for 44–57% of the variation in absolute corticosterone concentrations, while flock only accounted for 0–21% of the observed variation. In order to minimize the effects of intraindividual variation on our ability to detect treatment effects, we also examined changes in corticosterone within individuals for which we had complete baseline ($n = 21$) or stress-induced ($n = 28$) samples from both time periods. The competition treatment did not predict changes in baseline corticosterone concentrations from days 6 to 14 (treatment: $F_{1,20} = 0.09$; $P = 0.76$; GLM including flock, dominance, sex), but stress-induced corticosterone concentrations tended to increase from day 6 to day 14 in

high competition flocks (treatment: $F_{1,27} = 3.86$; $P = 0.06$; GLM including flock, dominance, sex).

Status and sex-related effects

Consistent with previous studies of dominance behavior in this species (McGraw and Hill, 2000a,b), measures of body size (wing length, tarsus length, mass) and condition (mass/tarsus) did not predict dominance status (linear regression: $n = 64$, all $r^2 < 0.03$, all $P > 0.17$) across individuals.

An individual's dominance status was a significant predictor of antibody titer to SRBCs (Fig. 4; $F_{1,28} = 5.63$, $P = 0.025$; Mixed Model including flock (random), individual (random), treatment, and sex). This relationship was similar in both treatment groups, as there was no significant interaction between dominance status and social competition treatment on antibody response when the interaction term was included in the model ($F_{1,27} = 0.19$, $P = 0.66$; Mixed Model including flock

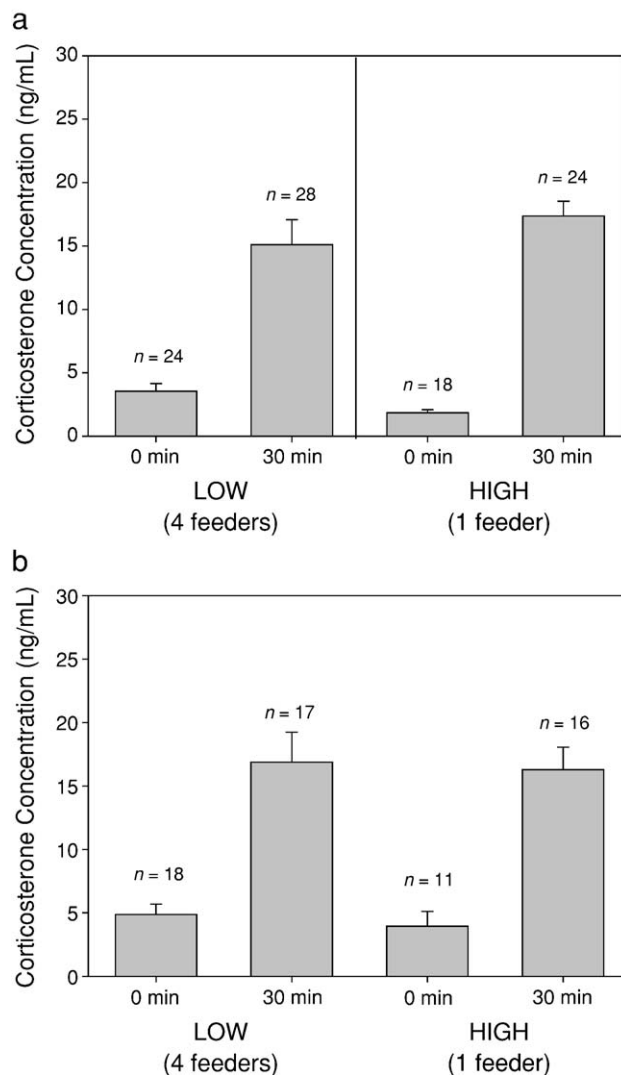


Fig. 3. The social competition treatment did not influence absolute levels of baseline and stress-induced corticosterone concentrations (ng/ml) on day 6 (a) or day 14 (b). Sample sizes are shown above. Error bars represent one standard error around the mean.

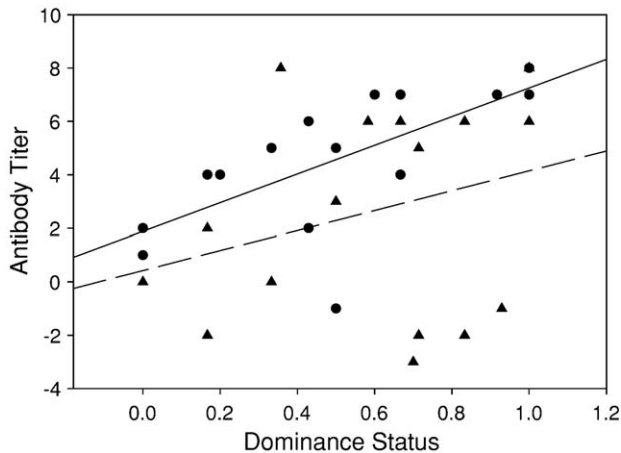


Fig. 4. Individual dominance status predicted the strength of antibody response to sheep red blood cells in both low (circles) and high (triangles) competition flocks. Regression slopes are plotted separately for the low (solid line) and high (dashed line) competition treatments.

(random), individual (random), treatment, dominance, and sex). Dominance status did not predict within-individual changes in mass or aggressive interactions from day 6 to day 12 (mass: $F_{1,63} = 0.02$, $P = 0.98$, aggressive interactions: $F_{1,63} = 0.38$, $P = 0.54$; GLM including flock, treatment, and sex). Sex was not a significant predictor of antibody titer $F_{1,28} = 1.32$, $P = 0.26$ or within-individual changes in mass or aggressive interactions (mass: $F_{1,63} = 0.01$, $P = 0.91$, aggressive interactions: $F_{1,63} = 0.53$, $P = 0.47$).

Dominance status did not predict either baseline measure of corticosterone concentrations or stress-induced corticosterone concentrations at day 14 ($F_{1,25-32.6} < 2.57$, all $P > 0.12$; Mixed Model including flock (random), individual (random), treatment, and sex). However, dominance status, sex, and their interaction predicted stress-induced corticosterone concentrations at day 6 (dominance: $F_{1,43.7} = 10.05$, $P = 0.0028$; sex: $F_{1,15.9} = 8.78$, $P = 0.009$; sex*dominance: $F_{1,43.2} = 14.5$, $P = 0.004$; Mixed Model including flock (random), individual (random), and treatment).

Corticosterone concentrations, aggression rate, and immunity

Corticosterone concentrations (baseline or stress-induced) at days 6 and 14 did not predict the strength of antibody response to SRBCs ($F_{1,12-19} < 2.45$, all $P > 0.14$; Mixed Model including flock (random), individual (random), treatment, dominance, and sex, and dominance*sex). Furthermore, within-individual changes in baseline or stress-induced corticosterone levels did not predict the strength of antibody response to SRBCs ($F_{1,9-12} < 0.29$, all $P > 0.62$).

Discussion

Competition treatment effects

Our manipulation of the number of food access points created treatment-level differences in the intensity of aggressive

interaction and had noticeable effects on individual house finch physiology in only 14 days. The high competition treatment compromised both individual body mass and the strength of antibody responses to sheep red blood cells: house finches in flocks forced to use a single food source lost more body mass and mounted significantly lower antibody titers than individuals in flocks that fed at multiple sources. Although social competition has been largely overlooked in studies of individual immunity in wild vertebrates (Saino et al., 2000; Svensson et al., 2001), our results suggest that the extent of aggression experienced in a social group or population can have important implications for individual condition and immunocompetence. In fact, the higher infection rates associated with larger groups sizes in many animal systems (Alexander, 1974; Brown and Brown, 1986; Côté and Poulin, 1995; Hoogland, 1979; Krause et al., 1999; Poulin, 1999) and the greater relative investment in immune organs in social species (Møller et al., 2001; Wilson et al., 2003) may result in part from the effects of social competition on immunocompetence.

Social status effects

Individual social status predicted the strength of antibody response within flocks, with more dominant individuals mounting stronger responses. A positive relationship between dominance status and immunocompetence was also detected in a previous study in red jungle fowl (Zuk and Johnsen, 2000), and the strength of this relationship varied with an individual's social context (solitary or group housed). Another study of greenfinches found differences in virus clearance rates between high and low status individuals (Lindström, 2004). Contrary to our initial predictions, our social competition treatment did not reveal context-dependent relationships between dominance and immunity: the impact of social status on antibody response was equivalent in the high and low competition treatments. Overall, our results suggest that subordinate individuals in hierarchical social groups may suffer greater costs of parasite and pathogen infection. In fact, previous studies have documented higher levels of parasitism in subordinate individuals in the wild (Ezenwa, 2004; Rubenstein and Hohmann, 1989; but see Halvorsen, 1986).

Corticosterone as mechanism

Aggressive interactions such as those experienced during intraspecific competition are known to mediate concentrations of glucocorticoids such as corticosterone in vertebrates (Wingfield, 1994), and these hormones also have well-documented immunosuppressive effects (Apanius, 1998). However, our results lend little support to the idea that plasma corticosterone concentrations downregulated humoral immune responses in the high competition treatment. Although our corticosterone results should be interpreted with substantial caution due to low statistical power, none of our four measures of absolute corticosterone concentrations increased significantly in the high competition flocks (Fig. 3). Interestingly, within-individual changes in stress-induced corticosterone concentrations, which

control for the considerable variation present among individuals, tended to be higher under elevated social competition, but this result was not quite statistically significant. Finally, all of the corticosterone measures were unrelated to the magnitude of antibody response, which would provide the most robust support for a role of corticosterone in down-regulating immunity at high social competition. Overall, our low statistical power and limited sample sizes do not allow us to discount a role for glucocorticoid hormones in the observed immunosuppression under high social competition.

Relationships between social behavior, hormone levels, and immunity can vary substantially with an individual's sex (e.g., Saino et al., 2000) or social rank (e.g., Barnard et al., 1998; Zuk and Johnsen, 2000), but we also found little support for the idea that corticosterone concentrations were related to sex or dominance status in our study. A single corticosterone value was significantly related to dominance status, sex, and their interaction, with subordinate males showing higher corticosterone concentrations at day 6. The single previous study of dominance status and corticosterone concentrations in house finches found comparable results: Belthoff et al. (1994) found no detectable effect of social status on corticosterone levels in group-housed males but found significantly elevated corticosterone concentrations in subordinates housed as dyads with a single dominant male. The group housing of individuals in our study may have obscured any obvious relationship between dominance rank and corticosterone concentrations that may exist under pairwise housing, where a subordinate individual cannot avoid interacting with dominant flockmates.

The mechanisms mediating the cost of immunity in vertebrates remain debatable (Lochmiller and Deerenberg, 2000), but evidence is accumulating that resource or energy-mediated costs may be important in birds (Alonso-Alvarez and Tella, 2001; Klasing, 1998; Martin et al., 2003; Ots et al., 2001; but see Svensson et al., 1998) and other vertebrates (Demas et al., 1997). Our results lend some support to a resource-mediated relationship between social competition and immunity: birds in the high competition treatment decreased significantly in mass over the course of the experiment. However, because the changes in body mass consisted of an average decrease of only 1%, it is unclear if this could explain the lower immune responses. Further detailed study is needed to test whether resource and/or hormone-mediated mechanisms led to the observed immunosuppression under high social competition.

Conclusions

Any effects of social competition on humoral immunity to SRBCs are likely to extend to the many classes of parasites and pathogens that stimulate humoral components of immunity. Humoral immunocompetence to SRBCs predicts long-term survival in barn swallows (Saino et al., 2003), and in some cases, immunity to novel antigens negatively correlates with variation in parasite loads (Poulin, 1996; Schalk and Forbes, 1997). However, little empirical support exists for the idea that humoral immunocompetence reflects meaningful differences in com-

posite pathogen resistance (Adamo, 2004). Furthermore, studies using standardized immune assays assume that higher antibody titers indicate greater pathogen resistance, but detected differences in antibody titer may also reflect adaptive changes in cell distribution across different arms of the immune response (Braude et al., 1999). The fitness consequences of variation in antibody response must be interpreted with caution, and individual resistance to a suite of parasites and pathogens (Adamo, 2004; Owens and Wilson, 1999) must be examined before firm conclusions can be drawn as to how intraspecific competition influences parasite and pathogen dynamics in wild populations.

The house finch is a particularly interesting species for asking questions about social competition, stress levels, and immunocompetence because the eastern North American population of this species was recently colonized by a bacterial pathogen, *Mycoplasma gallisepticum* (MG), which causes debilitating conjunctivitis (Ley et al., 1996). MG infection is most prevalent during seasonal periods of flocking (Altizer et al., 2004), when house finches congregate at backyard feeders and compete intensely for food (Thompson, 1960). Previous studies have found positive relationships between house finch group size and the prevalence of MG (Altizer et al., 2004) which may in part result from variation in the intensity of social interactions during the non-breeding season. Furthermore, house finch mortality from the MG epidemic shows density dependence on both broad geographic (Hochachka and Dhondt, 2000) and experimental scales (Luttrell et al., 1998; Roberts et al., 2001). Two studies that examined MG spread in house finch social groups found quite disparate results that varied with housing density: Luttrell et al. (1998) found high (>40%) MG-associated mortality rates when finches were housed at 26.6 birds/m³, while Roberts et al. (2001) documented only an 8% mortality rate at 1 bird/m³. Although disease spread will occur more quickly at high housing densities, density-dependent disease mortality is also consistent with the idea that social competition at high densities suppresses immunocompetence and disease resistance. Future studies should address to what extent social stress and immunocompetence are more general contributors to patterns of density-dependent pathogen infection in natural populations (e.g., Arneberg et al., 1998).

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