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Genetic and environmental components of phenotypic variation in immune response and body size of a colonial bird, *Delichon urbica* (the house martin)

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Directional selection for parasite resistance is often intense in highly social host species. Using a partial cross-fostering experiment we studied environmental and genetic variation in immune response and morphology in a highly colonial bird species, the house martin (*Delichon urbica*). We manipulated intensity of infestation of house martin nests by the haematophagous parasitic house martin bug *Oeciacus hirundinis* either by spraying nests with a weak pesticide or by inoculating them with 50 bugs. Parasitism significantly affected tarsus length, T cell response, immunoglobulin and leucocyte concentrations. We found evidence of strong environmental effects on nestling body mass, body condition, wing length and tarsus length, and evidence of significant additive genetic variance for wing length and haematocrit. We found significant environmental variance, but no significant additive genetic variance in immune response parameters such as T cell response to the antigenic phytohemagglutinin, immunoglobulins, and relative and absolute numbers of leucocytes. Environmental variances were generally greater than additive genetic variances, and the low heritabilities of phenotypic traits were mainly a consequence of large environmental variances and small additive genetic variances. Hence, highly social bird species such as the house martin, which are subject to intense selection by parasites, have a limited scope for immediate microevolutionary response to selection because of low heritabilities, but also a limited scope for long-term response to selection because evolvability as indicated by small additive genetic coefficients of variation is weak.

Keywords: additive variance, coloniality, environmental variance, heritability, parasites, T cell response.

Introduction

A growing number of studies has demonstrated the fitness costs imposed by parasites on their hosts (reviewed by Møller, 1997). In particular, highly social hosts appear to suffer from the detrimental effects of parasites (e.g. Alexander, 1974; Møller *et al*., 1993), although social hosts appear to invest more in antiparasite defences, such as an efficient immune system, than less social species (Møller & Erritzøe, 1996). Apart from behavioural responses (Hart, 1997), the immune system is probably the most efficient defence that hosts have evolved to counter the detrimental effects of infectious diseases and parasites (Wakelin, 1996). The ability to mount an efficient immune response against parasites is under both environmental and genetic control. Low nutritional status negatively affects the development of organs involved in immune responses (Møller *et al*., 1998) and decreases the level of cell-mediated immune function (Gershwin *et al*., 1985; Lochmiller *et al*., 1993). Because altricial birds depend entirely on food brought by their parents during growth, provisioning rate may strongly influence immunocompetence (Saino *et al*., 1997). Recent work on the barn swallow (*Hirundo rustica*) and the magpie (*Pica pica*) has shown that

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hatching date correlates negatively with aspects of immunocompetence (Saino *et al*., 1997; Sorci *et al*., 1997a). Hence, food availability, which is known to decrease during the breeding season in many birds (e.g. Lack, 1966), seems to mediate certain immune responses.

Variation in immune responses may also have genetic components. Heritable variation in host resistance to parasites can be estimated by comparing resemblance between relatives in the intensity of parasitic infestations or immune responses, but such studies are rare and likely to be confounded by common environment effects (Sorci *et al*., 1997b). To date only a few field studies have investigated the role of genetic factors in host resistance against ectoparasites in natural populations. Using a partial cross-fostering experiment, Møller (1990) showed resemblance between male parent barn swallows (*Hirundo rustica*) and their offspring in susceptibility to the tropical fowl mite (*Ornithonyssus bursa*). Moreover, nestlings that were cross-fostered in another nest resembled their true parents more than their foster parents with respect to mite intensities. These results strongly suggest that there is heritable variation in host resistance to tropical fowl mites in barn swallows. Boulinier *et al*. (1997) found a positive correlation between parent and offspring parasite load in a population of kittiwakes (*Rissa tridactyla*) infested with a tick (*Ixodes uriae*), suggesting heritable variation in host susceptibility. A third study demonstrated significant heritability of faecal egg counts of an gastrointestinal nematode in Soay sheep (*Ovis aries*) (Smith *et al*., 1999). Two final studies showed additive genetic variation in T cell immune responsiveness to an artificial antigen in cross-fostered barn swallow and cross-fostered great tit (*Parus major*) nestlings (Saino *et al*., 1997; Brinkhof *et al*., 1999), implying that cell-mediated immunity has a genetic basis under field conditions.

The ability to raise a defence against parasites can only evolve if the trait is subject to selection, and if it is determined partially by additive genetic variation. Strong directional selection tends to deplete additive genetic variation (Fisher, 1930). Consistent with this idea, phenotypic traits closely related to fitness have been shown to have lower heritabilities than morphological and other traits supposed to be less closely related to fitness (Mousseau & Roff, 1987; Price & Schluter, 1991). Hence, if immunocompetence permits birds to cope with the detrimental effects of parasites, we should predict a low heritability of immune defence in bird species under intense directional selection caused by parasites. Because parasites might be particularly virulent and hence impose particularly strong selection pressures on their hosts in highly colonial species (Brown & Bomberger-Brown, 1986), where elevated rates of horizontal transmission and multiple infections occur frequently (Møller & Erritzøe, 1996), we may predict a low heritability of immune function in colonial species.

Heritability is defined as the ratio of additive genetic variance to total phenotypic variance (Falconer & Mackay, 1996), and a low heritability may thus arise from a low additive genetic variance, a high environmental variance, or a combination. An alternative measure of evolutionary potential to selection is the additive genetic coefficient of variation (Charlesworth, 1984; Houle, 1992). This measure of evolvability standardizes the additive genetic variance with respect to the mean value of the trait. Secondary sexual characters and life history traits, which supposedly are closely associated with fitness, differ markedly among species and hence demonstrate a strong realized potential for evolutionary change. Yet, life history traits and similar characters have low heritabilities (Mousseau & Roff, 1987). Calculation of the additive genetic coefficient of variation revealed high evolvability for these traits, suggesting that standardized measures of genetic variability are large despite intense directional selection (Houle, 1992; Pomiankowski & Møller, 1995).

In this study we first estimate environmental and genetic components of variation of immune responses and morphological traits in a Spanish population of a highly colonial bird species, the house martin (*Delichon urbica*). Secondly, we report phenotypic and additive genetic coefficients of variation for these characters. The fitness cost of parasitism for house martins by an haematophagous ectoparasite, the house martin bug (*Oeciacus hirundinis*), has previously been demonstrated to be of considerable importance in this population (de Lope *et al*., 1993, 1998; Møller *et al*., 1994). To be able to separate environmental from genetic factors, we performed a partial cross-fostering experiment by exchanging half of the offspring between pairs of nests after hatching.

Materials and methods

Study organism and study site

The house martin is a small colonial passerine bird feeding exclusively on small insects caught on the wing, and it is therefore very sensitive to adverse meteorological conditions (Bryant, 1975). There is no sexual dimorphism in external morphology. Old nests may be used in successive years. The house martin bug [*Oeciacus hirundinis* (Hemiptera, Cimicidae)] is the most important haematophagous ectoparasite found in the study site (de Lope *et al*., 1993). The field study took place in Badajoz, Extremadura, Spain, during the 1996 breeding season,

in a colony of approximately 600 pairs located on a student dormitory at the university campus.

Manipulation of loads of nest parasites

We manipulated the parasite load of nests in order to determine the effects of parasites on environmental and genetic components of phenotypic variation of nestling immune responses and morphology. The experiment was carried out in natural nests that are usually aggregated. When house martins arrived from their winter quarters a pyrethrin solution (0.91%) was used to fumigate all nests. In order to reduce future ectoparasite migration between nests, we artificially created units of 1–5 nests by removing old nests between these units at the beginning of the breeding season. The average distance between nearest neighbouring units was 40 cm. For the experiment only one randomly chosen nest was used from each unit. When clutches were completed, a sample of 50 randomly chosen nests were each experimentally infested with 50 house martin bugs extracted from old house martin nests. Fifty other nests were fumigated with a pyrethrin solution every second day throughout the breeding season until the second clutch fledged. Previous experiments using this technique have shown no negative effects of the mild pesticide on reproduction and growth of the host (de Lope *et al*., 1993, 1998; Møller *et al*., 1994).

Cross-fostering experiment

A partial cross-fostering experiment was performed during the first clutch. Hatching date was determined by daily examination of nests. Only pairs of nests (dyads in the following) in which entire broods hatched within 24 h were used for this experiment. When all eggs had hatched a randomly chosen half of the nestlings was exchanged between two nests with similar hatching date and different parasite treatment (sprayed and infested). The same number of chicks was transferred between nests, unless the number of nestlings was uneven, in which case a number below one half was transferred. In order to be able to classify the nestlings with respect to their nest of origin and their nest of rearing, chicks were made identifiable by means of cutting the end of a claw, which consists of dead tissue. Because we were also interested in the effect of parasites during the second clutch, parasite loads were estimated by removing the nests and counting the parasites present in the nest material soon after nestlings from the second brood had fledged. This estimate was considered an approximation. House martin bugs were categorized in two age classes, juveniles and adults. We also recorded the number of fleas that had colonized the nests.

When nestlings were 15 days old, they were ringed with an individually numbered aluminium ring, their tarsus length measured to the nearest 0.01 mm with a digital calliper and their wing length measured to the nearest mm with a ruler. Nestlings were weighed with a precision of 0.1 g using a Pesola spring balance. Simultaneously we collected a small blood sample in a microcapillary from the brachial vein. One drop of blood was used for a blood smear for subsequent leucocyte counts and detection of blood parasites. Microcapillary tubes were centrifuged for 10 min at 14 000 r.p.m. in order to estimate the haematocrit value which is the proportion of total blood volume occupied by erythrocytes after centrifugation. An age of 15 days was chosen because that is when nestling body mass peaks and tarsus reaches its final length in the study area (F. de Lope, unpubl. data). Chicks fledged when they were 22–30 days old in this population.

Measures of immunocompetence and immune responses

T cell response As a measure of one component of immunocompetence we used the intensity of T-lymphocyte response, which is a cell-mediated *in vivo* immune response to an injection of 15-day-old nestlings with 0.625 mg of phytohemagglutinin (PHA-P, Sigma Chemical Co., St. Louis, MO, USA) in 0.125 mL of phosphate-buffered saline (PBS) in the middle of the wing web (patagium). PHA has a mitogenic effect on T-lymphocytes and the injection stimulates macrophage infiltration and dense perivascular accumulation of lymphocytes (Stadecker *et al*., 1977; McCorkle *et al*., 1980). In the other wing we injected a similar volume of PBS as a control. Twenty-four hours later the thickness of the wing web at the inoculation sites was measured with a digital calliper to the nearest 0.01 mm. The change in thickness in the wing where PHA was injected minus the change in the control wing was used as a measure of immunocompetence. The response to inoculation of PHA has been shown to be a reliable indicator of one component of immunocompetence (see Lochmiller *et al*., 1993). All values were measured three times. We then computed the mean and used these values for calculations.

Immunoglobulin assay Immunoglobulins are a heterogeneous class of serum proteins that are the source of antibody proteins involved in the humoral response to a large spectrum of parasite infestations (Wakelin, 1996). Immunoglobulins were assayed by densitometric analysis after electrophoretic separation of plasma proteins on agarose gels (Paragon SPE Kit, Beckman). Densitometric analysis was performed with a computer image

analysis procedure (Gelanalyst, Eidosoft). The relative titre of immunoglobulins was expressed as the ratio between the area of the densitometric profile corresponding to the immunoglobulin region and the total area of the densitometric profile. As shown in a previous study, within-blood sample repeatability of immunoglobulin titres was high (for more details, see Saino & Møller, 1996).

Leukocyte count Blood smears were air-dried, fixed in absolute methanol and stained by the May–Grunwald–Giemsa staining method. Leucocytes and red blood cells were counted at 1000× magnification. In each smear we counted the number of leucocytes per 10 000 red blood cells ('no. of leucocytes'). To obtain the total number of leucocytes circulating in the blood, we used the number of leucocytes per 10 000 red blood cells multiplied by the haematocrit value (hereafter 'absolute counts'), following the method described by Dufva & Allander (1995).

Statistical analyses

Body condition was defined as the residuals from the regression of body mass on tarsus length. The results of the cross-fostering experiment were analysed using a mixed-model nested ANOVA using type III sum-of-squares, following the design described by Merilä (1996, 1997) and Merilä & Fry (1998). The effect of nest of origin (random effect) is nested within each dyad. Each dyad consisted of two broods that were partially exchanged. The term 'dyad' (random effect) reflects any temporal variation faced by offspring among pairs of nests during the breeding season. The term 'rearing' (fixed effect) measures the effect of the parasite treatment. The direction of the effect was evaluated by the least-squares means. A significant rearing-by-origin interaction (random effect) would indicate that the young responded differently to different environments, in this case to different parasite loads. This implies a genotype-by-environment interaction. Including the interaction between rearing and dyad in the analyses does not change the results.

We calculated phenotypic means and variances based on our field data. Variance components were estimated with the SAS VARCOMP Procedure using maximum likelihood estimates. All variance components due to dyad, nest of rearing and the error variance term sum up to the environmental variance (V_E). The variance component resulting from the term nest of origin estimates half the additive genetic variance ($\frac{1}{2}V_A$), but also includes one quarter of dominance variance ($\frac{1}{4}V_D$) and maternal effects if present (V_M) (Merilä, 1996). The rearing-origin (nested in dyad) interaction term estimates the variance component of the genotype-by-environment interaction (V_{GE}). By summing $V_E + V_A + V_{GE}$, we obtained the total phenotypic variance V_P.

The heritability is defined as $h^2 = V_A/V_P$. As maternal effects and dominance variance are included in the V_A term, the heritability as presented here is an approximation.

We calculated phenotypic and additive genetic coefficients of variation (CV_P and CV_A, respectively) as $CV = \sqrt{V}/X$, where X is the mean character value (Charlesworth, 1984).

All statistical analyses were performed using SYSTAT (Wilkinson, 1992), except for the nested ANOVA and the VARCOMP procedure, which were performed with SAS (SAS Institute, 1990). All P-values reported are two-tailed.

Results

Effects of parasite treatment on parasite load

The number of adult bugs per nest on the day following fledging of offspring from the second brood was (mean ± SE) 38.3 ± 10.6 ($N = 39$) for sprayed nests and 177.8 ± 29.4 for infested nests ($N = 41$) (t-test on log-transformed data, $t = -6.32$, d.f. = 78, $P < 0.001$). The number of juvenile bugs was 11.4 ± 4.1 for the spray treatment group and 153 ± 62.2 for the infested group (t-test on log-transformed data, $t = -6.85$, d.f. = 78, $P < 0.001$). Fleas were also found in most nests. For sprayed nests, the number was 3.2 ± 0.64 vs. 46.2 ± 7.5 for the parasite group. This difference was highly significant (t-test on $\log_{10}$-transformed data, $t = -9.55$, d.f. = 78, $P < 0.001$). Thus parasite treatment had a clear effect on both house martin bugs and fleas. There was a strongly positive relationship between the number of juvenile and adult bugs [linear regression on log-transformed data: $F = 252.81$, d.f. = 1, 78, $r^2 = 0.764$, $P < 0.001$, slope (SE) = 0.920 (0.057)], which provides evidence for local reproduction of parasites.

None of the blood smears showed infection with blood parasites.

Phenotypic and genetic variation in the partial cross-fostering experiment

A total of 45 pairs of nests was exchanged. As a consequence of desertion or complete mortality occurring in 10 nests, only 34 pairs of nests could be used in the statistical analyses. Results of the nested ANOVA on morphological characters and haematocrit are reported in Table 1. Dyads showed significant differences for all measures. These differences may be interpreted to be the result of temporal variation during the breeding season. Additive genetic differences are represented by the

Table 1 Results of a mixed-model nested ANOVA with morphological measures and haematocrit of nestling house martins as dependent variables and dyad, nest of origin (nested within Dyad) and nest of rearing as factors

	d.f.	SS	MS	*F*	*P*
Body mass					
Dyad	33	880.22	26.67	12.06	< 0.001
Origin (Dyad)	34	63.70	1.87	0.85	0.707
Rearing	1	2.05	2.05	0.93	0.336
Rearing*Origin (Dyad)	65	390.66	6.01	2.72	< 0.001
Error	139	307.30	2.21		
Body condition					
Dyad	33	7.330	0.222	13.03	< 0.001
Origin(Dyad)	34	0.580	0.017	1.00	0.476
Rearing	1	0.051	0.051	3.03	0.084
Rearing*Origin (Dyad)	65	3.044	0.046	2.75	< 0.001
Error	139	2.369	0.017		
Tarsus length					
Dyad	33	18.80	0.569	3.21	< 0.001
Origin (Dyad)	34	6.871	0.202	1.14	0.295
Rearing	1	0.765	0.765	4.31	0.039
Rearing*Origin (Dyad)	65	12.262	0.188	1.06	0.377
Error	139	24.677	0.177		
Wing length					
Dyad	24	3799.19	158.30	5.84	< 0.001
Origin (Dyad)	25	1433.86	57.35	2.12	0.004
Rearing	1	37.98	37.98	1.40	0.239
Rearing*Origin (Dyad)	48	3027.39	63.07	2.33	< 0.001
Error	104	2817.62	27.09		
Haematocrit					
Dyad	33	0.135	0.004	2.79	< 0.001
Origin (Dyad)	34	0.080	0.002	1.60	0.031
Rearing	1	0.0007	0.0007	0.53	0.466
Rearing*Origin (Dyad)	65	0.121	0.001	1.27	0.121
Error	137	0.202	0.001		

'origin' factor, and the effect of house martin bugs by the 'rearing' factor. Parasite treatment significantly reduced tarsus length, whereas the other variables were unaffected by the presence of parasites. The additive genetic factor was significant for wing length and haematocrit, suggesting significant heritable variation only for these two traits. The presence of a genotype-by-environment interaction was indicated by a statistically significant origin-by-rearing interaction for body mass (Fig. 1), body condition and wing length, showing that some siblings performed better in parasite-free nests while others performed better in parasitized nests.

Results of partial cross-fostering on the different measures of immune response are summarized in Table 2. Dyads showed significant differences for all measures. The origin factor was nonsignificant for all measures of immune response. The parasite treatment significantly increased the T cell response, the number of leucocytes and absolute counts of leucocytes circulating in the blood and decreased immunoglobulin production.

Variance components and heritabilities are reported in Table 3. Environmental variances are generally considerably larger than the additive genetic variances with the variance ratio ranging from 4.7 to 146.6 for different traits (Table 3). The heritability estimates were consistently small (ranging from 0 to 0.156 for different traits) and significant only for wing length and for haematocrit (Tables 1,2,3).

Coefficients of variation

We calculated the phenotypic and the additive genetic coefficients of variation for all characters (Table 3). Phenotypic coefficients of variation ranged from 0.0011

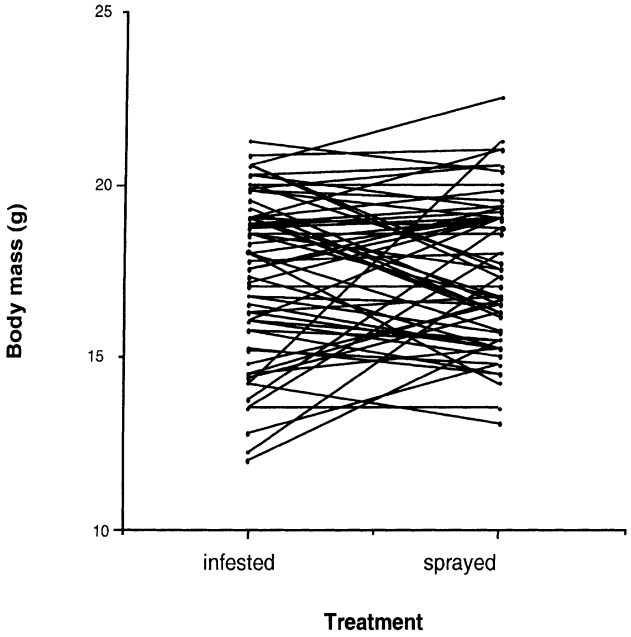

Fig. 1 Genotype-by-environment interaction for body mass of sibling house martins reared in two different environments. Lines connect mean values for siblings in the two environments.

for haematocrit to 0.482 for leucocyte counts with a median value of 0.110 (Table 3).

The additive genetic coefficients of variation ranged from 0 for body mass to a maximum of 0.118 for leucocyte counts with a median value of 0.029 (Table 3).

Discussion

Environmental and genetic components of phenotypic variation

Experimental manipulation of parasite load of house martin nests combined with partial cross fostering provided clear evidence for strong environmental effects on nestling size and immune response (Tables 1–3). This finding is what was predicted based on the hypothesis that highly colonial species generally encounter high degrees of environmental fluctuations compared to solitarily breeding relatives (Lack, 1968).

Components of phenotypic variation can be partitioned if siblings are reared in different environments, as in several partial cross-fostering experiments (Møller, 1990; Gebhardt-Heinrich & van Noordwijk, 1991; Merilä, 1997). However, this experimental approach

Table 2 Results of a mixed-model nested ANOVA with four different measures of immune function of nestling house martins as dependent variables and dyad, nest of origin (nested within Dyad) and nest of rearing as factors

	d.f.	SS	MS	*F*	*P*
T cell response					
Dyad	28	90.25	3.22	3.57	< 0.001
Origin (Dyad)	29	24.09	0.83	0.92	0.585
Rearing	1	4.36	4.36	4.84	0.029
Rearing*Origin (Dyad)	55	45.64	0.83	0.92	0.628
Error	112	100.98	0.90		
Immunoglobulins					
Dyad	33	0.451	0.013	2.06	0.002
Origin(Dyad)	34	0.263	0.007	1.17	0.266
Rearing	1	0.029	0.029	4.39	0.038
Rearing*Origin (Dyad)	65	0.424	0.006	0.98	0.521
Error	134	0.890	0.006		
No. of leucocytes					
Dyad	23	251 395.81	10 930.25	1.80	0.026
Origin (Dyad)	24	125 156.99	5 214.87	0.86	0.651
Rearing	1	34 909.87	34 909.87	5.76	0.018
Rearing*Origin (Dyad)	45	275 737.04	6 127.48	1.01	0.472
Error	88	533 372.50	6 061.05		
Absolute no. of leucocytes					
Dyad	23	474 948 453	20 649 932	2.20	0.004
Origin (Dyad)	24	184 972 725	7 707 196	0.82	0.699
Rearing	1	75 648 133	75 548 133	8.08	0.005
Rearing*Origin (Dyad)	45	483 817 396	10 751 497	1.15	0.287
Error	87	815 014 198	9 367 979		

Table 3 Phenotypic (V_P), environmental (V_E) and additive genetic (V_A) components of variation, the additive genetic coefficient of variation (CV_A), the phenotypic coefficient of variation (CV_P), and the heritability (h^2) for morphology and immune response variables in nestling house martins

	V_P	V_E	V_A	CV_A	CV_P	h^2
Wing length	63.34	46.46	9.908	0.066	0.168	0.156
Tarsus length	0.252	0.219	0.020	0.013	0.045	0.079
Body mass	6.309	5.069	0	0	0.144	0
Haematocrit	0.0021	0.0016	0.0003	0.0004	0.0011	0.143
Immunoglobulins	0.0079	0.0075	0.0004	0.057	0.255	0.051
T cell response	1.181	1.173	0.0083	0.029	0.352	0.007
Leukocytes	6080.99	4882.99	361.29	0.118	0.482	0.059

does not account for any maternal effects influencing the quality of eggs, and the results should be viewed with this caveat in mind. Although maternal effects may cause significant bias in heritability estimates, that is not necessarily the case. A study of heritability of morphological characters in the barn swallow based on paternity analyses of offspring revealed no resemblance between extra-pair offspring and the foster father, while resemblance between offspring and the biological father was highly significant (N. Saino *et al*. unpubl. data). Because the attending female always was the mother, this study provides little evidence of strong maternal effects related to the phenotype of the mate. The present study showed little heritable variation in morphology and immune response variables in the house martin (Tables 1–2). This contrasts with two studies in the closely related, semicolonial barn swallow and the territorial great tit, in which results from partial cross-fostering experiment demonstrated heritable variation in cell-mediated immunity (Saino *et al*., 1997; Brinkhof *et al*., 1999). Because heritability is the ratio between the additive genetic variance and the total phenotypic variance (Falconer & Mackay, 1996), it could be argued that differences in the experimental design between these three studies render the comparisons irrelevant because parasite treatment could increase environmental variance. However, environmental and phenotypic variances were also increased artificially in the two other studies. In the study on the great tit ectoparasites were added in some nests and in the study on barn swallows, brood size was experimentally manipulated. Only analyses of pairs of closely related species that differ in degree of sociality would allow an assessment of whether small heritabilities in colonial species are a general rule. If that turned out to be the case, it would have important consequences for rates of evolution in relation to social organization.

Several characters demonstrated genotype–environment interactions with some nestlings performing better in parasitized nests, while others performed better in parasite-free environments. This was the case for body mass, body condition and wing length (Table 1). Such differences in performance among genotypes in different environments may be an important factor maintaining additive genetic variance in a population (Falconer & Mackay, 1996).

Intense directional selection, as in the case of parasite impact on host fitness in highly social species, will tend to reduce the level of additive genetic variance (Falconer & Mackay, 1996). The apparently small heritabilities of immune responses in the house martin are in accordance with this prediction. The morphological characters studied demonstrated additive genetic variability in a few cases, as shown by the origin variance component being statistically significant (Table 1). The largest estimated heritability for morphology was 0.156 for wing length with smaller estimates for tarsus length and body mass (Table 3). These findings are interesting given the generally high heritabilities recorded for morphological traits in free-living populations of birds (review in Cooke & Buckley, 1987). Because morphological traits are not supposed to be subject to equally strong directional selection as immune response variables, the similarity of heritability of morphology and immune response traits is not consistent with persistent directional selection resulting in reduced heritabilities.

The additive genetic coefficient of variation has been hypothesized to provide an appropriate measure of evolvability, because it scales the additive genetic variance to the mean phenotypic value, and because it avoids the influence of the environmental variance that may otherwise strongly affect heritability estimates (Charlesworth, 1984; Houle, 1992). Additive genetic coefficients of variation for the house martin had a mean value of 4.7% (Table 3). This compares to a mean value of 8.4% for bristle number in *Drosophila* and 11.9% for fecundity in *Drosophila* (Houle, 1992). Secondary sexual characters in a range of different species had a mean value of 17% (Pomiankowski & Møller, 1995).

Coloniality and environmental variance

Coloniality is presumed to be associated with unpredictable environments (Danchin & Wagner, 1997), which might have important consequences for the development of morphology and immune function in nestling birds. This social mode of reproduction is supposed to occur mainly in species faced with spatially and temporally unpredictable food supplies that therefore are economically indefensible (Lack, 1968). Thus, high levels of environmental variation may be predicted to occur in highly social organisms living under environmentally variable conditions. The findings of the present study are in accordance with the general assumption that environmental fluctuations encountered by colonial bird species are considerable.

Parasite virulence has recently attracted considerable attention because of the importance of parasites for the evolution of sex, sexual selection, and host life-history evolution (Bull, 1994; Ewald, 1994; Garnett & Antia, 1994; Frank, 1996). The relative or absolute rate of horizontal as opposed to vertical transmission of parasites may cause increased levels of virulence (Ewald, 1983; Bull *et al*., 1991; Herre, 1993). The frequency of multiple infections with parasite strains of different genetic origin due to intense competition for limiting host resources may result in the evolution of increased virulence (Knolle, 1989). Interestingly, coloniality increases opportunities both for horizontal transmission of parasites and the probability of multiple infections because (i) colonial species on a regular basis encounter conspecifics that may transmit parasites, (ii) colonial species frequently re-use nest sites that may hold parasite populations that build up over the years, and (iii) the use of common foraging grounds may provide ample opportunities for infective stages of parasites to be transmitted to intermediate hosts that are consumed by the colonial species (Møller & Erritzøe, 1996). A comparative study of investment in immune function provided indirect evidence for increased virulence of parasites of colonial breeding bird species, because the relative size of two immune defence organs was consistently larger in colonial species than in closely related solitary species (Møller & Erritzøe, 1996).

If the environmental component of phenotypic variation for immune response variables is large and additive genetic variances are small in colonial species, as suggested by the present study, and if heritabilities are therefore small, such species will have little immediate opportunity to evolve more efficient antiparasite defences against their parasites. Hence, we might expect that colonial species will suffer a double disadvantage compared to solitarily breeding species: because their parasites generally will be virulent, parasites will become established readily in nests because of efficient transmission, and low heritabilities will prevent a strong, immediate response to selection. If the magnitude of additive genetic coefficients of variation reflects the scope for microevolutionary response to selection, and hence the evolvability of phenotypic characters (Charlesworth, 1984; Houle, 1992), these findings indicate that the ability of social hosts such as the house martin to respond to the selection pressure from parasites may be smaller than that of less social species.

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References

ALEXANDER, R. D. 1974. The evolution of social behaviour. *Annu. Rev. Ecol. Syst*., **5**, 325–383.

BOULINIER, T., SORCI, G., MONNAT, J. Y. AND DANCHIN, E. 1997. Parent-offspring regression suggests heritable susceptibility to ectoparasites in a natural population of kittiwake *Rissa tridactyla*. *J. Evol. Biol*., **10**, 77–85.

BRINKHOF, M. W. G., HEEB, P., KÖLLIKER, M. AND RICHNER, H. 1999. Immunocompetence of nestling great tits in relation to rearing environment and parentage. *Proc. R. Soc. B*, **266**, 2315–2322.

BROWN, C. R. AND BOMBERGER-BROWN, M. 1986. Ectoparasitism as a cost of coloniality in cliff swallows (*Hirundo pyrrhonota*). *Ecology*, **67**, 1206–1218.

BRYANT, D. M. 1975. Breeding biology of house martin *Delichon urbica* in relation to aerial insect abundance. *Ibis*, **120**, 180–216.

BULL, J. J. 1994. Perspective: Virulence. *Evolution*, **48**, 1423–1437.

BULL, J. J., MOLINEUX, I. J. AND RICE, W. E. 1991. Selection of benevolence in a host – parasite system. *Evolution*, **45**, 875–882.

CHARLESWORTH, B. 1984. The evolutionary genetics of life histories. In: Shorrocks, B. (ed.) *Evolutionary Ecology*, pp. 117–133. Blackwell Scientific Publications, Oxford.

COOKE, F. AND BUCKLEY, P. A. (eds) 1987. *Avian Genetics*. Academic Press, London.

DANCHIN, E. AND WAGNER, R. 1997. The evolution of coloniality: the emergence of new perspectives. *Trends Ecol. Evol*., **12**, 342–347.

DE LOPE, F., GONZÁLEZ, G., PÉREZ, J. J. AND MØLLER, A. P. 1993. Increased detrimental effects of ectoparasites on their bird

hosts during adverse environmental conditions. *Oecologia*, **95**, 234–240.

DE LOPE, F., MØLLER, A. P. AND DE LA CRUZ, C. 1998. Parasitism, immune response and reproductive success in the house martin *Delichon urbica*. *Oecologia*, **114**, 188–193.

DUFVA, R. AND ALLANDER, K. 1995. Intraspecific variation in plumage coloration reflects immune response in great tit (*Parus major*) males. *Funct. Ecol.*, **9**, 785–789.

EWALD, P. W. 1983. Host-parasite relations, vectors, and the evolution of parasite severity. *Ann. Rev. Ecol. Syst.*, **14**, 465–485.

EWALD, P. W. 1994. *The Evolution of Infectious Disease*. Oxford University Press, Oxford.

FALCONER, D. S. AND MACKAY, T. F. C. 1996. *Introduction to Quantitative Genetics*, 4th edn. Longman, New York.

FISHER, R. A. 1930. *The Genetical Theory of Natural Selection*. Clarendon Press, Oxford.

FRANK, S. 1996. Models of parasite virulence. *Q. Rev. Biol.*, **71**, 37–78.

GARNETT, G. P. AND ANTIA, R. 1994. Population biology of virus–host interactions. In: Morse, S. S. (ed.) *The Evolutionary Biology of Viruses*, pp. 51–73. Oxford University Press, New York.

GEBHARDT-HEINRICH, S. G. AND VAN NOORDWIJK, A. J. 1991. The genetical ecology of nestling growth in the great tit. I. Heritability estimates under different environmental conditions. *J. Evol. Biol.*, **4**, 341–362.

GERSHWIN, M. E., BEACH, R. S. AND HURLEY, L. S. 1985. *Nutrition and Immunity*. Academic Press, Orlando.

HART, B. L. 1997. Behavioural defence. In: Clayton, D. H. and Moore, J. (eds) *Host-Parasite Evolution, General Principles and Avian Models*, pp. 59–77. Oxford University Press, Oxford.

HERRE, E. A. 1993. Population structure and the evolution of virulence in nematode parasites of fig wasps. *Science*, **259**, 1442–1445.

HOULE, D. 1992. Comparing evolvability and variability of quantitative traits. *Genetics*, **130**, 195–204.

KNOLLE, H. 1989. Host density and the evolution of parasite virulence. *J. Theor. Biol.*, **136**, 199–207.

LACK, D. 1966. *Population Studies of Birds*. Clarendon Press, Oxford.

LACK, D. 1968. *Ecological Adaptations for Breeding in Birds*. Chapman & Hall, London.

LOCHMILLER, R. L., VESTEY, M. R. AND BOREN, J. C. 1993. Relationship between protein nutritional status and immunocompetence in northern bobwhite chicks. *Auk*, **110**, 503–510.

McCORKLE, F., OLAH, I. AND GLICK, B. 1980. The morphology of the phytohemagglutinin-induced cell response in the chicken's wattle. *Poultry Sci.*, **59**, 616–623.

MERILÄ, J. 1996. Genetic variation in offspring condition: an experiment. *Funct. Ecol.*, **10**, 465–474.

MERILÄ, J. 1997. Expression of genetic variation in body size of the collared flycatcher under different environmental conditions. *Evolution*, **51**, 526–536.

MERILÄ, J. AND FRY, J. D. 1998. Genetic variation and causes of genotype–environment interaction in the body size of blue tit (*Parus caeruleus*). *Genetics*, **148**, 1233–1244.

MØLLER, A. P. 1990. Effects of a haematophagous mite on the barn swallow (*Hirundo rustica*): a test of the Hamilton Zuk hypothesis. *Evolution*, **44**, 771–784.

MØLLER, A. P. 1997. Parasitism and the evolution of host life history. In: Clayton, D. H. and Moore, J. (eds) *Host-Parasite Evolution: General Principles and Avian Models*, pp. 105–127. Oxford University Press, Oxford.

MØLLER, A. P. AND ERRITZØE, J. 1996. Parasite virulence and host immune defense: host immune response is related to nest reuse in birds. *Evolution*, **50**, 2066–2072.

MØLLER, A. P., ALLANDER, K. AND DUFVA, R. 1993. Parasites and the evolution of host social behavior. *Adv. Stud. Behav.*, **22**, 65–102.

MØLLER, A. P., DE LOPE, F., MORENO, J., GONZÁLEZ, G. AND PÉREZ, J. J. 1994. Ectoparasites and host energetics: house martin bugs and house martin nestlings. *Oecologia*, **98**, 263–268.

MØLLER, A. P., CHRISTE, P., ERRITZØE, J. AND MAVAREZ, J. 1998. Condition, disease and immune defence. *Oikos*, **83**, 301–306.

MOUSSEAU, T. A. AND ROFF, D. A. 1987. Natural selection and the heritability of fitness components. *Heredity*, **59**, 181–198.

POMIANKOWSKI, A. AND MØLLER, A. P. 1995. A resolution of the lek paradox. *Proc. R. Soc. B*, **260**, 21–29.

PRICE, T. AND SCHLUTER, D. 1991. On the low heritability of life-history traits. *Evolution*, **45**, 853–861.

SAINO, N. AND MØLLER, A. P. 1996. Sexual ornamentation and immunocompetence in the barn swallow. *Behav. Ecol.*, **7**, 227–232.

SAINO, N., CALZA, S. AND MØLLER, A. P. 1997. Immunocompetence of nestling barn swallows in relation to brood size and parental effort. *J. Anim. Ecol.*, **66**, 827–836.

SAS INSTITUTE. 1990. *SAS User's Guide: Statistics*. SAS Institute, Cary, NC.

SMITH, J. A., WILSON, K., PILKINGTON, J. G. AND PEMBERTON, J. M. 1999. Heritable variation in resistance to gastro-intestinal nematodes in an unmanaged mammal population. *Proc. R. Soc. B*, **266**, 1283–1290.

SORCI, G., MØLLER, A. P. AND BOULINIER, T. 1997b. Genetics of host–parasite interactions. *Trends Ecol. Evol.*, **12**, 196–199.

SORCI, G., SOLER, J. J. AND MØLLER, A. P. 1997a. Reduced immunocompetence of nestlings in replacement clutches of the European magpie (*Pica pica*). *Proc. R. Soc. B*, **264**, 1593–1598.

STADECKER, M. J., LUKIC, M., DVORAK, A. AND LESKOWITZ, S. 1977. The cutaneous basophil response to phytohemagglutinin in chickens. *J. Immunol.*, **118**, 1564–1568.

WAKELIN, D. 1996. *Immunology to Parasites*, 2nd edn. Cambridge University Press, Cambridge.

WILKINSON, L. 1992. *Systat: Statistics, Version 5.2*. Systat Inc., Evanston, IL.