Genetic Variation and Causes of Genotype-Environment Interaction in the Body Size of Blue Tit (*Parus caeruleus*)

Juha Merilä[*] and James D. Fry[†,1]

**Laboratory of Ecology and Animal Systematics, Department of Biology, University of Turku, Turku, Finland and [†]Department of Zoology, Uppsala University, Uppsala, Sweden*

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ABSTRACT

In several studies of natural populations of birds, the heritability of body size estimated by parent-offspring regression has been lower when offspring have developed in poor feeding regimens than when they developed in good feeding regimens. This has led to the suggestion that adaptation under poor regimens may be constrained by lack of genetic variation. We examined the influence of environmental conditions on expression of genetic variation in body size of nestling blue tits (*Parus caeruleus*) by raising full sibs in artificially reduced and enlarged broods, corresponding to good and poor feeding regimens, respectively. Individuals grown in the poor regimen attained smaller body size than their sibs grown in the good regimen. However, there was among-family variation in response to the treatments—*i.e.*, genotype-environment interactions (GEIs). Partitioning the GEI variance into contributions attributable to (1) differences in the among-family genetic variance between the treatments and (2) imperfect correlation of genotypic values across treatments identified the latter as the main cause of the GEI. Parent-offspring regressions were not significantly different when offspring were reared in the good environment (h^2 = 0.75) *vs.* when they were reared in the poor environment (h^2 = 0.63). Thus, there was little evidence that genetic variance in body size was lower under the poor conditions than under the good conditions. These results do not support the view that the genetic potential for adaptation to poor feeding conditions is less than that for adaptation to good conditions, but they do suggest that different genotypes may be favored under the different conditions.

GENOTYPE-environment interaction (GEI) exists when different genotypes have different phenotypic responses to environmental variation (*e.g.*, Stearns 1992; Via 1993, 1994). From the evolutionary point of view, GEI is important for two reasons. First, it can permit populations to evolve ultimately to have the optimum phenotypic mean in different environments, thereby promoting adaptation to heterogeneous environments (Via and Lande 1985, 1987). However, in certain situations, GEI can also slow down the rate of adaptation of a population to a variable environment in the short run (Fry 1996). Second, GEI can lead to maintenance of genetic variation (Gillespie and Turelli 1989), and thereby at least partly help to explain the high genetic variance in many fitness-related traits. Consequently, the amount of genetic variance in the slope of reaction norms (*i.e.*, GEI) may be a strong determinant of the fitness of the population and ultimately of its survival. A lack of variation may lead to a failure to respond adaptively to environmental changes and ultimately to extinction. It is therefore of fundamental importance to investigate the amount and proximate causes of genetic variation in reaction norms in natural populations. Apart from the pioneering studies in amphibians (*e.g.*, Berven 1987; Newman 1988), and a few studies of birds (Gebhardt-Henrich and van Noordwijk 1991, 1994; Price 1991; Smith and Wettermark 1995; Merilä 1996, 1997), there have been almost no attempts to evaluate the significance of GEIs in natural vertebrate populations. The lesson from these studies is mixed: some have found evidence for the presence of GEI (Gebhardt-Henrich and van Noordwijk 1991; Price 1991; Merilä 1997), while others have not (Smith and Wettermark 1995; Merilä 1996). Additional evidence for GEI in natural bird populations comes from studies showing lower heritability estimates of morphological traits when offspring are reared in poor environments (low food supply) than when they are reared in good environments (see Merilä 1997 for a review; Larsson *et al.* 1997). This had led to the suggestion that the potential for adaptation to poor environments is lower than the potential for adaptation to good environments (Larsson 1993; Merilä 1997). However, interpretation of heritability estimates obtained by parent-offspring regressions when offspring are reared in different environments must be made with caution (Lande 1987; Riska *et al.* 1989). If offspring of the same or nearly the same set of parents are studied

Corresponding author: Juha Merilä, Section of Population Biology, Department of Zoology, Uppsala University, Villavägen 9, S-752 36 Uppsala, Sweden. E-mail: juha.merila@zoologi.uu.se

[1] *Present address:* Utah State University, Department of Biology, Logan, UT 84322-5305.

in both good and poor environments, then the denominator of the midoffspring-midparent regression, which is the variance of the midparental values, is the same regardless of offspring environment (Merilä 1997). Any difference in the regressions between good and poor environments must therefore involve the numerator of the regression, which is the covariance of midparent and midoffspring values. When parental and offspring environments are the same, the numerator estimates half of the additive genetic variance (Falconer and Mackay 1996). If parental and offspring environments differ, as they must in at least one case if two offspring environments are involved, then the regression numerator estimates half the additive genetic covariance of the trait between parental and offspring environments. This genetic covariance is the covariance of breeding values for the trait as expressed in the different environments. It may be written as $\rho_{po}\sigma_{Ap}\sigma_{Ao}$, where ρ_{po} is the additive genetic correlation across parental and offspring environments, σ_{Ap} is the square root of the additive genetic variance in the parental environment, and σ_{Ao} is the square root of the additive genetic variance in the offspring environment. If there are two offspring environments, denoted by *o1* and *o2*, any difference in the covariances implies that either $\rho_{po1} \neq \rho_{po2}$ or $\sigma_{Ao1} \neq \sigma_{Ao2}$. Hence, a lower regression in one offspring environment than in the other could reflect a difference in the genetic correlations, not a difference in the additive variances. Either of these inequalities implies that GEI in the formal sense exists, but only in the case of a difference in the additive variances can one conclude that the population has lower potential to adapt to the poor environment than to the good environment. The main aims of this study were (1) to investigate how different environmental conditions (brood size) affect the causal components of phenotypic variance in blue tit body size, (2) to investigate the importance of the component of variance attributable to GEI in determining total phenotypic variance in body size of this species, and (3) to distinguish between the different possible causes of GEI—namely, cross-environment genetic correlations of less than one and differences between genetic variances among environments. In addition, by using cross-fostered nestlings, we asked to what degree the resemblance between offspring and parents might be of environmental origin.

MATERIALS AND METHODS

The study species and population: The blue tit (*Parus caeruleus*) is a small, cavity-nesting passerine that inhabits a wide range of habitats, from mixed deciduous forests to urban parks, with a clear preference for rich old deciduous forests (Cramp and Perrins 1993). It is a partial migrant in continental Europe; most juveniles disperse far from their natal areas, while adult birds are territorial and remain on breeding grounds all year round. Blue tits readily accept nest-boxes as breeding sites and, in our population, lay only one clutch annually. Although polygamy is common in some parts of the breeding range (Dhondt 1987), in our study area 99% of the males were monogamous (J. Merilä, unpublished data). The mean clutch size in first clutches is about 11 eggs (range 4–17 eggs), and incubation is by the female alone. The male feeds the female during incubation, and when the young hatch, both parents feed the nestlings. The nestling period is 16–20 days, and the young reach independence about 2 weeks after fledging.

We studied a breeding population of blue tits on the southern part of the island of Gotland (about 57°10′ N, 18°20′ E), off the east coast of Sweden, during April–June 1993–1995. Our study area consisted of 16 wood lots, which were mostly rich deciduous forest dominated by oak (*Quercus robur*) and ash (*Fraxinus excelsior*), sometimes with a dense understory of hazel (*Corylus avellana*). Some parts of the area were mixed coniferous forest, and a few nest-boxes were placed in suburban gardens located close to rich deciduous woodlands.

The data: The data were collected during regular nest-box inspections beginning in late April until all young had fledged by mid-June. For each brood, the date of clutch initiation, clutch size, and the date of hatching were recorded. Except for 1995, when all birds were measured by another person, J.M. measured the tarsus length of all 14-day-old young (to an accuracy of 0.1 mm) using digital calipers as detailed in Merilä (1997). By this time, the nestling tarsus length has usually attained its final size (Dhondt 1982), as confirmed by repeated measurement of individuals first measured as nestlings in 1992 or 1993, and their remeasurement as adults in 1993 or 1994. There was no systematic change in tarsus length after fledging (mean change ± SE = 0.002 ± 0.051 mm, paired *t*-test: $t_{50} = 0.04$, $P = 0.97$), and the repeatability (*i.e.*, intra-class correlation; Sokal and Rohlf 1981; see also Bailey and Byrnes 1990) calculated using nestling and adult measurements was high ($R = 0.83$, $F_{25,26} = 10.48$, $P < 0.001$). However, in 1994, when the growing conditions for nesting tits were suboptimal, nestlings in most broods failed to develop normally. Hence, in 1994, most 14-day-old nestlings were underdeveloped both in terms of plumage (J. Merilä, personal observation) and tarsal growth. The latter was confirmed by repeated measurements of nestlings from 22 families, first measured at day 14 post-hatch, and then remeasured 2–4 days after this. In all different experimental groups, nestlings continued their growth [average increment ±SE: 0.32 ± 0.11 mm, 0.25 ± 0.03 mm, and 0.56 ± 0.15 mm for reduced ($n = 6$), control ($n = 7$), and enlarged ($n = 9$) broods, respectively]. Hence, though nestlings were still growing, there was no evidence that average growth increments would have differed among experimental groups (one-way ANOVA: $F_{2,19} = 1.80$, $P = 0.19$). Comparison of repeated measurements of females measured twice during the breeding season in 1993 (~3 weeks apart) and of adults measured both in 1993 and 1994 (~1 year apart) further confirmed the high repeatability of measurements (1993: $R = 0.88$, $F_{34,35} = 16.07$, $P < 0.001$; 1993–1994: $R = 0.91$, $F_{15,16} = 22.39$, $P < 0.001$). Likewise, the repeatability of measurements in 1995, calculated using the same birds captured twice, was high ($R = 0.89$, $F_{13,14} = 17.58$, $P < 0.001$). However, comparison of birds that J.M. measured in 1993 or 1994 with the measurements made in 1995 revealed that although the measurements were highly correlated ($r = 0.84$, $P < 0.001$), the measurements of tarsus length from 1995 were on average 0.39 mm longer than those made in previous years (paired *t*-test: $t_{38} = 8.12$, $P < 0.0001$). Hence, a correction was made for all measurements made in 1995 by subtracting the average among-year difference (0.39 mm) from all 1995 measurements.

Mothers were captured for the first time when they had incubated eggs for about 9 days (1993–1994), and for the

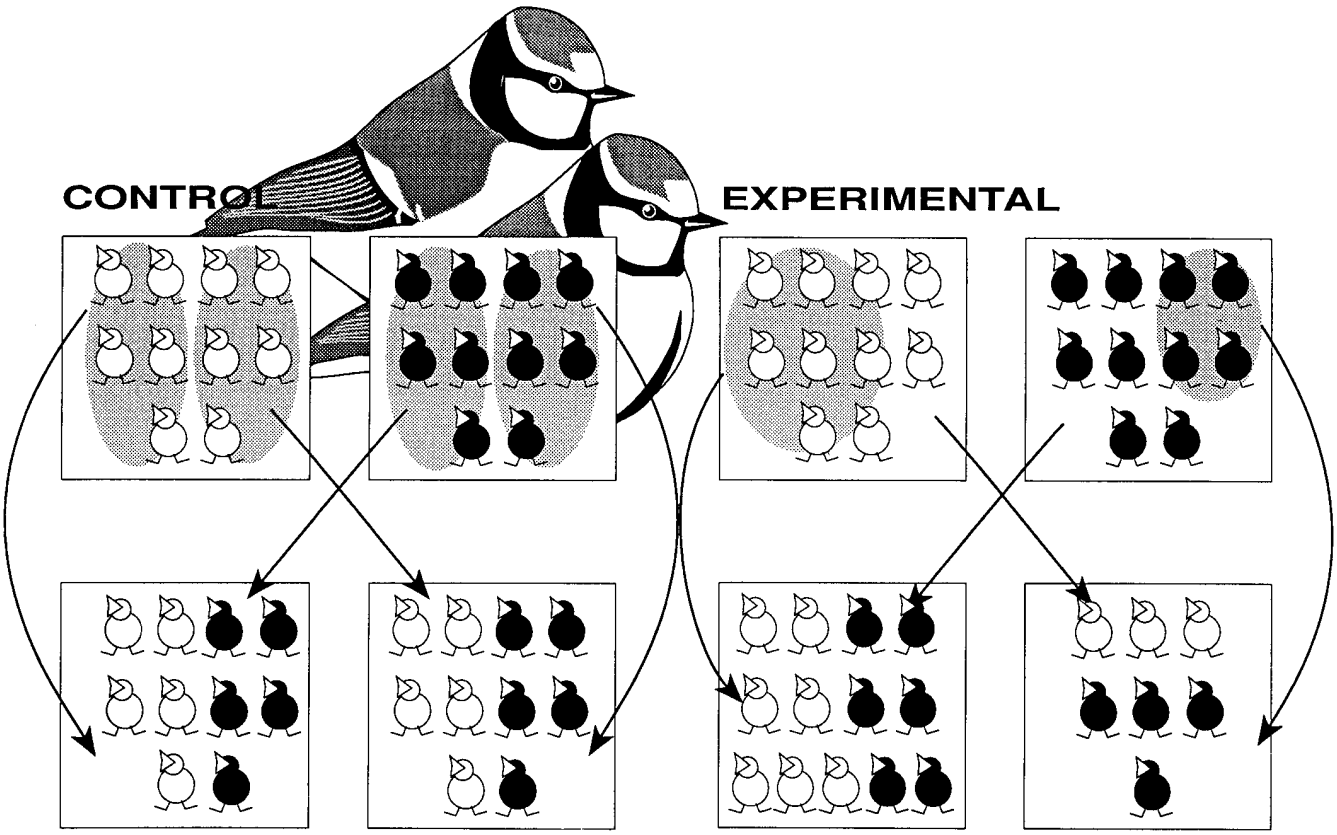

Figure 1.—Schematic representation of the cross-fostering protocol in control (no brood size manipulation) and experimental (brood reduction or enlargement) broods. Each box represents a nest-box, and the arrows indicate the origin (upper row) of the young in the resultant broods (lower row).

second time with the putative father when feeding the 2-wk-old young. Hence, the family relationships are based on the assumptions that (1) the adults feeding the young in a particular nest-box were their true parents and (2) all nestlings originally in the same nest-box were full-sibs. However, as extra-pair paternity (EPP) is known to occur in this species (Kempenaers *et al.* 1992), some of the presumed full-sibs might have been half-sibs not related to their putative father. However, one way to evaluate the possible importance of EPP is provided by comparison of father-offspring and mother-offspring regressions (*cf.* Alatalo *et al.* 1989) because EPP should result in lower father-offspring than mother-offspring regressions.

Cross-fostering and brood size manipulation experiments: To separate genetic and environmental causes of resemblance, we performed reciprocal cross-fostering experiments, creating broods that consisted of approximately equal numbers of nestlings from two different families (Figure 1). As the aim was also to evaluate how different growth conditions might affect heritability estimates and causal components of variance, brood size was simultaneously manipulated by reducing or increasing the original clutch size by about 1/3. This was accomplished by moving about 2/3 of the young from a "reduced" brood to an "enlarged" brood and switching back 1/3 of the young in the recipient (enlarged) nest to the donor (reduced) nest (Figure 1). Hence, both reduced and enlarged broods consisted of foreign and own young in approximately equal numbers. The experimental pairs of nests were created by matching two nests according to their hatching dates and clutch sizes. However, as it was not always possible to match nests by these criteria, some exchanges were made between nests that differed in clutch size by (at most) two eggs, but never between nests that differed in their hatching date. Most exchanges took place among nests situated 200–1500 m apart, but some exchanges took place among nests situated up to 5 km apart. All young were banded with aluminum rings at the age of 6 days; until this age, foreign young were made identifiable by painting their claws or clipping some of the downy feathers on their head. In 1993, 1994, and 1995, 32, 23, and 29 pairs of broods, respectively, were manipulated; hence, in total, 168 families were subjected to experimentation. However, to reduce variation caused by differences in the genetic constitution of the two experimental groups, only those nests where at least one own and one foreign young survived in both nests of the pair (= dyad) were included in the analyses. Therefore, after excluding all dyads where one nest was lost because of predation, death of the whole brood, death of all young from one family, 29, 19, and 25 pairs of experimental broods were available for analyses in 1993, 1994, and 1995, respectively. To check that the transfer itself did not affect the size test nestlings attained, we also performed swaps where young were exchanged without changing the original brood size (Figure 1). Thus, these nests served as controls to evaluate possible effects of moving. Using the same criteria as for experimental broods, 13 and 6 pairs of control broods were available for analysis in 1993 and 1994, respectively. No control broods were created in 1995. There was no difference in initial clutch size between reduced and enlarged broods (Table 1), although clutch size in control broods in 1993, but not in 1994, was significantly smaller than in experimental broods (Table 1). However, reduced broods contained significantly fewer young both immediately after manipulation (day 2 post-hatch) and at day 14 post-hatch, than control

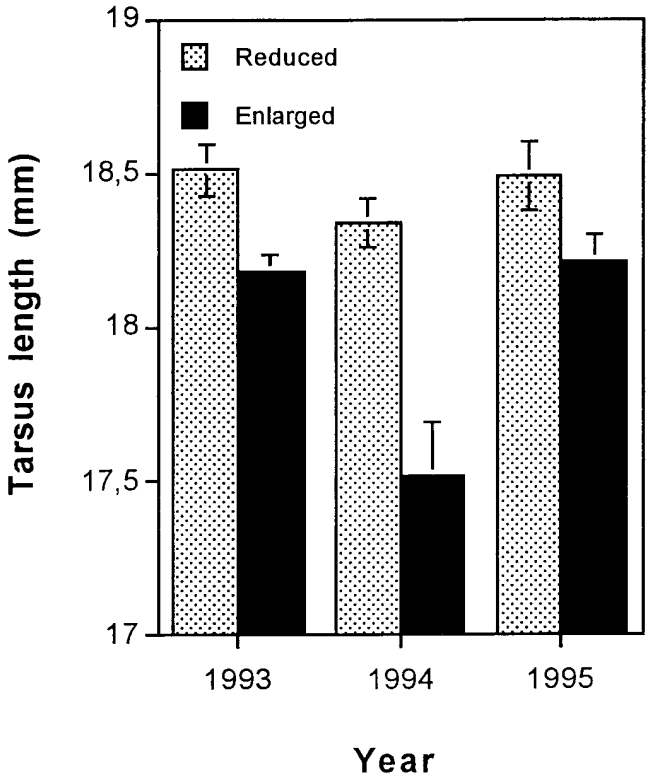

Figure 2.—Mean tarsus length (± SE) of blue tit nestlings in two experimental environments (reduced and enlarged broods) in 3 different study years. Within each year, the two means are based on mean values of two full-sib groups raised either in reduced or enlarged broods. In each of the years, the experiments' main effect was significant (Table 5), but there was also a significant Year × Experiment interaction in the combined analysis (Table 4), showing that the effect of the experiment differed in different years.

broods, while the enlarged broods contained significantly more young than reduced and control broods—except in 1994, when enlarged broods experienced heavy mortality (Table 1). Despite the heavy mortality in 1994, and significantly lower fledging success in 1995 as compared with other years, the results in Table 1 suggest that our manipulations were successful in creating differential growth environments, as attempted.

Full-sib analyses: We used three different types of full-sib analyses to investigate the relative importance of genetic and environmental factors in determining nestling size. First, following Merilä (1996, 1997) we performed mixed-model analyses of variance on the data from both offspring environments. In the full model, the main effects were Year and Dyad within Year (both random), Experiment (reduced or enlarged broods, a fixed effect), and Box of origin within Dyad and Year (random). We also analyzed reduced models for each of the different years to reveal possible among-year heterogeneity in different effects. Second, we performed nested, random-effect ANOVAs for each offspring environment separately, using Box of rearing and Box of origin (nested within Box of rearing) as factors. In this analysis, the box-of-origin effect estimates 1/2 of the additive genetic variance (V_A), plus 1/4 of the dominance variance and maternal effects if present. Third, we used the control broods to perform a two-way nested (random effects) analysis of variance (ANOVA) following Atchley and Rutledge (1980) to investigate GEI in response to differences between nest-boxes within dyads. In this analysis, the terms Box of rearing, Box of origin and their interaction were nested within Dyads. Atchley and Rutledge (1980) give a fuller account of the biological interpretation of the effects in this model.

All full-sib analyses were performed using type III sums-of-squares as obtained from PROC GLM in SAS (SAS Institute, Inc. 1989). The variance components for all effects were calculated by equating the observed mean squares with expected mean squares; and, if they occurred, negative variance components were set to zero. The "variance" explained by the experimental environment was also calculated, although the environments were considered a fixed effect as a result of their nonrandom sampling. The estimates based on type III sums-of-squares were usually in good agreement with estimates derived by restricted maximum-likelihood methods.

Cross-environment genetic correlations: The genetic correlation across environments quantifies the degree to which expression of a trait in one environment shares a heritable genetic basis with the expression of the same trait in a different environment (Via and Lande 1985; Falconer and Mackay 1996). There are two ways of estimating the cross-environment genetic correlation from the experimental design used in this study. First, cross-environment genetic correlations in each year can be estimated as

$$r_g = \frac{V_{BOXO,RED+ENL}}{\sqrt{(V_{BOXO,RED} \times V_{BOXO,ENL})}} \quad (1)$$

where $V_{BOXO,RED+ENL}$ is the estimated variance component due to Box of origin from the ANOVA with both environments (Table 4 or 5), and $V_{BOXO,RED}$ and $V_{BOXO,ENL}$ are the estimated variance components from the ANOVAs on the single-environment (reduced or enlarged broods) data, respectively (Table 6; Fry 1992). Second, another estimate of the cross-environment correlation can be obtained by partitioning the Box of origin × Experiment interaction from the mixed-model ANOVA (see above) into components attributable to heterogeneity of genetic variance and lack of genetic correlation between environments (Robertson 1959, p. 478). Rearranging Robertson's equation gives

$$r_g = \frac{(V_{BOXO,RED+}V_{BOXO,ENL})/2 - V_{BOXO \times EXP}}{\sqrt{(V_{BOXO,RED} \times V_{BOXO,ENL})}} \quad (2a)$$

Here, the numerator includes the average of the genetic variances in the two environments $[(V_{BOXO,RED} + V_{BOXO,ENL})/2]$, and the GEI variance ($V_{BOXO \times EXP}$) estimated by the Experiment × Box of origin interaction (Table 4 or 5). The denominator is the same as in the first method. This equation can be also written as (Robertson 1959)

$$V_{BOXO \times EXP} = 0.5(\sqrt{V_{BOXO,RED}} - \sqrt{V_{BOXO,ENL}})^2 \quad (2b, \text{first term})$$
$$+ (1 - r_g)\sqrt{(V_{BOXO,RED} \times V_{BOXO,ENL})} \quad (2b, \text{second term})$$

where the first term is the component of the GEI variance attributable to difference in the genetic variances, and the second term is the component attributable to the cross-environment correlation being less than one. By using r_g from Equation 2a and substituting variance components into Equation 2b, it is possible to evaluate the relative importance of the two possible causes of GEI by comparing the magnitude of these two terms.

Parent-offspring regressions: Parent-offspring regressions (using both midparental and single-parent values) were used to compare heritability estimates for tarsus length in the reduced and enlarged offspring environments (Falconer and MacKay 1996). Because the same set of parental values was used for offspring in the two environments, we used multivariate regression (SAS Institute Inc. 1989) to take into account

TABLE 1

Mean clutch size, day 2 and day 14 brood sizes and fledging success (percentage of nestlings at day 2 fledged) in different experimental groups of blue tits

	Reduced	Control	Enlarged
Clutch size[a]			
1993	11.86 ± 0.24 29	10.67 ± 0.57 26	11.41 ± 0.22 29
1994	11.50 ± 0.47 19	10.69 ± 0.57 12	11.61 ± 0.41 19
1995	11.64 ± 0.23 25	—[e]	11.68 ± 0.21 25
Brood size at day 2[b]			
1993	7.69 ± 0.21 29	9.88 ± 0.31 26	14.17 ± 0.32 29
1994	7.31 ± 0.69 19	9.67 ± 0.64 12	13.50 ± 0.75 19
1995	8.44 ± 0.52 25	—[e]	14.52 ± 0.53 25
Brood size at day 14[c]			
1993	7.41 ± 0.25 29	9.42 ± 0.38 26	13.28 ± 0.39 29
1994	6.62 ± 0.64 19	9.17 ± 0.67 12	8.56 ± 0.74 19
1995	6.76 ± 0.57 25		12.40 ± 0.66 25
Fledging success[d]			
1993	0.96 ± 0.02 29	0.95 ± 0.02 26	0.94 ± 0.01 29
1994	0.91 ± 0.03 19	0.94 ± 0.02 12	0.66 ± 0.06 19
1995	0.79 ± 0.03 25	—[e]	0.86 ± 0.03 25

All values are brood means ± SE. n, number of broods.

[a] ANOVA (excluding control broods): Year $F_{2,136} = 0.06$, $P = 0.93$; Exp. $F_{1,136} = 0.18$, Year × Exp. $F_{2,136} = 0.62$, $P = 0.54$; excluding 1995: Year $F_{1,124} = 0.05$, $P = 0.82$; Exp. $F_{2,124} = 4.50$, $P = 0.013$; Year × Exp. $F_{2,124} = 0.36$, $P = 0.69$.

[b] ANOVA (excluding control broods): Year $F_{2,136} = 2.38$, $P = 0.09$; Exp. $F_{1,136} = 241.63$, $P < 0.001$; Year × Exp. $F_{2,136} = 0.15$, $P = 0.86$; excluding 1995: Year $F_{1,124} = 1.22$, $P = 0.27$; Exp. $F_{2,124} = 104.11$, $P < 0.001$; Year × Exp. $F_{2,124} = 0.12$, $P = 0.88$.

[c] ANOVA (excluding control broods): Year $F_{2,136} = 12.56$, $P < 0.001$; Exp. $F_{1,136} = 103.31$, $P < 0.001$; Year × Exp. $F_{2,136} = 7.25$, $P < 0.001$; excluding 1995: Year $F_{1,124} = 22.51$, $P < 0.001$; Exp. $F_{2,124} = 34.11$, $P < 0.001$; Year × Exp. $F_{2,124} = 12.74$, $P < 0.001$.

[d] ANOVA (excluding control broods): Year $F_{2,136} = 17.71$, $P < 0.001$; Exp. $F_{1,136} = 8.01$, $P < 0.01$; Year × Exp. $F_{2,136} = 11.98$, $P < 0.001$; excluding 1995: Year $F_{1,124} = 23.74$, $P < 0.001$; Exp. $F_{2,124} = 18.05$, $P < 0.001$; Year × Exp. $F_{2,124} = 12.93$, $P < 0.001$.

[e] No control broods were created in 1995.

the correlation between the offspring of the same parents in the two environments. The multivariate regression procedure was used to test two null hypotheses (1) that the two slopes are equal and (2) that both of the slopes equal zero. We did not perform parent-offspring regressions for control broods, as the sample sizes were too small for any reasonable inference. To combine data from different study years, we first transformed midoffspring and midparent values to zero mean within each of the study years, leaving the variances unchanged. As the single-parent-midoffspring estimates of heritability can be biased by assortative mating or unequal variances between the two sexes, these possible sources of bias were checked. There was no assortative mating with respect to tarsus length in any of the study years when analyzed separately (1993: $r = -0.01$, $n = 55$, $P > 0.10$; 1994; $r = -0.14$, $n = 30$, $P > 0.10$; 1995: $r = 0.10$, $n = 45$, $P > 0.10$) or together ($r = 0.03$, $n = 130$, $P > 0.10$), and the variances of male and female tarsus length were homogeneous ($F_{129,129} = 1.103$, $P = 0.29$).

All analyses were performed with the SAS statistical package (SAS Institute, Inc. 1989).

RESULTS

Effects of cross-fostering: To check that the exchange of young among nests did not affect the size they attained, we first compared the mean tarsus length of the fostered and nonfostered full sibs that were raised in control broods. There was no evidence that fostered and nonfostered young differed in tarsus length in either of the years for which data on control broods were available (Table 2). Hence the moving itself did not affect the size young attained.

Full-sib analyses of control broods: Two-way nested ANOVAs performed for control broods revealed that under "normal" conditions, environmental (Box of rearing, Dyad) and GEI effects (Box of rearing × Box of origin) were not significant and always accounted for

TABLE 2

Tarsus length (mm) of nonfostered (home) and fostered (away) full-sib blue tits reared in control broods

	1993	*n*	1994	*n*
Home	18.36 ± 0.08	26/126	18.26 ± 0.12	12/49
Away	18.51 ± 0.07	26/131	18.24 ± 0.10	12/63

All values are brood means ± SE. In 1995, no control broods were created. There was no difference in tarsus length of home or away-reared young (repeated measures ANOVA: Year $F = 2.43$, $P = 0.13$; home/away $F = 2.65$, $P = 0.11$; interaction $F = 1.88$, $P = 0.18$). *n*, number of families/nestlings.

TABLE 3

Analysis of variance of tarsus length of blue tits in cross-fostered control broods

Source	1993			1994			1993 + 1994		
	d.f.	*F*	Var. (%)	d.f.	*F*	Var. (%)	d.f.	*F*	Var. (%)
Year	—	—	—	—	—	—	1	1.65	1.1
Dyad	12	0.80	0.0	5	0.97	2.0	17	0.85	0.0
Box of rearing (Dyad)	13	1.57	3.8	6	3.16°	0.0	19	1.74	2.0
Box of origin (Dyad)	13	3.98**	21.7	6	14.36*	22.6	19	5.10***	21.9
Box of rearing × Box of origin (Dyad)	13	0.94	0.0	6	0.23	0.0	19	0.71	0.0
Error	205		74.5	80		75.4	285		75.0
Model	51	2.41***		23	1.98*		75	2.38***	

The analysis is a two-way nested ANOVA with Box of rearing, Box of origin, and their interaction all nested within dyads using type III sums-of-squares. Since this is a random-effects model, the effects of Box of rearing and origin are tested against the interaction and the interaction against error. The effect of Dyad is tested against a complex error term (SAS Institute, Inc. 1989). In the combined analysis, all effects are nested within years. Var. (%), percentage of variance accounted for by each of the factors in the model. °$P < 0.10$, *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

less than 5% of the phenotypic variance in body size (Table 3). Indeed, the only significant effect in these analyses was that attributable to Box of origin (Table 3), which is assumed to estimate 1/2 of the additive genetic variance (Atchley and Rutledge 1980).

Full-sib analyses of experimental broods: We first investigated factors affecting offspring size by performing a four-factorial, mixed-model ANOVA, which revealed that only the main effect of Box of origin was significant, estimating 1/2 of the additive genetic covariance of tarsus length across the two offspring environments, whereas year, experiment, and dyad effects did not account for any significant proportion of variance (Table 4). All of the interactions in the model were significant (Table 4). The significant Year × Experiment interaction shows that although nestlings raised in enlarged broods attained shorter tarsi than their full-sibs raised in reduced broods in each of the 3 study years (Figure 2), the magnitude of this effect differed between the years. The significant Dyad(Year) × Experiment effect can be interpreted as a common-environment effect of Box of rearing, as each experimental treatment (reduced or enlarged) is represented by a single rearing box per dyad. Hence, there was between-dyad heterogeneity in the success of the experiment in affecting tarsus length. The significant Experiment × Box of origin interaction suggests that there were differences among families in how the young responded to different experimental conditions—*i.e.*, genotype-environment interactions, or equivalently, genetic variation in slopes of reaction norms (Table 4). However, this effect accounted for less than 5% of all phenotypic variance in tarsus length (Table 4). To investigate these effects further, we repeated the analysis for each of the study years separately. These analyses revealed that in each of the study years, the brood-size manipulations (= experiment) had a significant effect on offspring tarsus length (Table 5). Likewise, genetic influences were strong in all study years, as revealed by the significant effect of box of origin (Table 5). There was statistical evidence

TABLE 4

Analysis of variance of tarsus length in cross-fostered blue tit broods

Source	d.f.	MS	*F*	Var.	Var. (%)
Year	2	15.03	1.86	0.021	2.9
Dyad (year)	70	2.36	1.12	0.017	2.3
Experiment	1	52.52	7.83	0.097	13.4
Box of origin (year,dyad)	73	1.31	3.09***	0.115	15.8
Year × Experiment	2	7.06	6.32***	0.035	4.9
Dyad × Experiment	70	1.22	2.88***	0.103	14.2
Experiment × Box of origin (dyad × year)	73	0.42	1.37*	0.030	4.1
Error	1070	0.31		0.307	42.3
Model	289	1.93	6.30***		

Results of mixed-model ANOVA examining environmental (year, experiment) and genetic (nest of origin) influences on offspring tarsus length. Var. is the variance component calculated from the expected means squares, and Var. (%) gives variance as a percentage of the total. *$P < 0.05$, ***$P < 0.001$.

for significant GEI (Box of origin × Experiment interaction) only in 1993, although a similar tendency ($P < 0.10$) was observed in 1995 (Table 5). In all cases, however, the variance components associated with the Experiment × Box of origin interactions were small compared to the Box of origin main effects (Tables 4 and 5). There were also significant Experiment × Dyad interactions in two of the three years. The effect of Dyad was never significant, as could be expected since the families were paired at random with respect to their phenotypic size.

Full-sib heritabilities: To estimate environment-specific heritabilities and variance components from the full-sib data, we performed separate ANOVAs for each of the experimental environments (Table 6). Box of origin variance components, reflecting 1/2 the additive genetic variance plus dominance and maternal-effect contributions (if present), were significant and fairly similar between the two treatments (Table 6). However, residual (error) variance components were always much higher in the enlarged than in the reduced environment. Box of rearing variance components, reflecting common-environment effects, varied considerably but tended to be larger in the enlarged environment (Table 6). Consequently, estimated heritabilities were always higher in the reduced environment than in the enlarged environment (Table 6).

Cross-environment correlations: Cross-environment genetic correlations (r_g) estimated using Equation 1 (see materials and methods) were 0.65, 0.82, and 0.91 for 1993, 1994, and 1995, respectively. These estimates are in good agreement with those obtained with Equation 2a, which gave 0.64, 1.0, and 0.89 for 1993, 1994, and 1995, respectively. Notably, the lowest estimates occurred in 1993, when there was evidence for significant GEI from the mixed-model ANOVA (Table 5). Furthermore, combining the data from all years gives estimates of cross-environment r_g of 0.80 and 0.82 with Equations 1 and 2a, respectively. Solving the two terms in Equation 2b and expressing them as a percentage of the total GEI variance (Tables 4 and 5), the proportional contributions of cross-environmental correlation terms to GEI variance were 82%, 0%, 83%, and 87% in 1993, 1994, 1995, and combined data, respectively. Hence, these estimates indicate that the cause of significant GEI from mixed-model ANOVA in 1993 (nearly significant in 1995 and in combined data) was lack of genetic correlation between the environments, and not differential expression of genetic variance. If so, we would expect that genetic variances for the two offspring environments estimated by parent-offspring regression should be similar (see introduction).

Parent-offspring regressions: Before examining whether parent-offspring regressions would reveal the same difference in heritability estimates between the two experimental environments as the full-sib analyses did, or whether offspring-parent covariances differed

TABLE 5

Analysis of variance of nestling tarsus length for each of the three study years separately

Source	1993				1994				1995			
	d.f.	*F*	Var.	Var. (%)	d.f.	*F*	Var.	Var. (%)	d.f.	*F*	Var.	Var. (%)
Dyad	28	0.76	0.000	0.0	18	1.31	0.071	8.5	24	1.22	0.034	5.9
Experiment	1	14.53***	0.042	8.1	1	18.58***	0.148	17.7	1	6.69*	0.029	4.9
Nest of origin (Dyad)	29	2.19*	0.078	14.0	19	2.60*	0.080	9.6	25	5.21***	0.189	32.1
Experiment × Dyad	28	1.47	0.028	5.5	18	6.63***	0.138	16.5	24	2.76**	0.079	13.5
Experiment × Nest of origin	29	1.68*	0.049	9.5	19	0.85	0.000	0.0	25	1.47°	0.029	4.9
Error	484		0.033	62.9	214		0.397	47.7	378		0.228	38.7
Model	115	2.99***			75	7.05***			99	7.05***		

° $P < 0.10$, * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

TABLE 6

Causal components of phenotypic variance and broad-sense heritability estimates (h^2) of blue tit tarsus length from full-sib analyses in two experimental environments (reduced *vs.* enlarged broods)

	Reduced								Enlarged							
	1993		1994		1995		All		1993		1994		1995		All	
Source	Var.	%	Var.	%	Var.	%	Var.	%	Var.	%	Var.	%	Var.	%	Var.	%
Box of rearing	0.000	0	0.000	0	0.146°	24	0.039	9	0.016	4	0.560***	48	0.089°	18	0.136***	23
Box of origin	0.166*	41	0.092**	28	0.274***	46	0.180***	41	0.076***	16	0.103*	9	0.157***	32	0.114***	19
Error	0.237	59	0.239	72	0.182	30	0.221	50	0.372	80	0.497	43	0.244	50	0.346	58
Total	0.403	100	0.331	100	0.603	100	0.441	100	0.464	100	1.160	100	0.491	100	0.596	100
h^2	0.82		0.56		0.91		0.81		0.32		0.12		0.63		0.38	
n	58/215		38/121		50/152		146/488		58/385		38/169		50/326		146/880	

The data were analyzed with a nested, random-effects ANOVA, where the effect of Box of origin was nested within the Box of rearing. For the combined data, among-year variation in offspring size was first partitioned out by nesting all other factors within years (in all calculations, variance attributable to year effects omitted). Variance components (Var.) were obtained using type III sums-of-squares. h^2, heritability estimate, calculated as $2 \times$ Var.(Box of origin)/Var.(total); n, number of families/number of nestlings. ° $P < 0.10$, * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

for offspring grown in different environments, we first investigated whether there was any suggestion that a shared environment between parents and offspring might have increased their resemblance. By regressing the midoffspring tarsus length of cross-fostered offspring against the tarsus length of their biological parents and foster parents, we found no similarity between the offspring and foster-parent tarsus lengths, while in both experimental environments, resemblance between offspring and their biological parents was high and significant (Table 7). Likewise, there was no suggestion that EPP would have been frequent (*cf.* Alatalo *et al.* 1989), as the contributions of both sexes of parents to offspring size were about equal (Table 8). Therefore, in our subsequent tests, we have combined the cross-fostered and home-grown young into the same analyses to increase the power of the tests.

Midparent-midoffspring regressions performed using the same set of biological parents for the two groups of full sibs raised in different environments revealed that the heritability estimates were lower in the "poor" offspring environment than in the "good" offspring environment, although not significantly so (Table 9). There was no evidence for nonlinearity in parent-offspring resemblance (*cf.* Gimelfarb and Willis 1994), as the quadratic components in each of the regressions (*cf.* Table 9) were far from significant ($F \leq 0.94$, $P > 0.34$, in all cases). However, even when the data from different years were combined, there was no significant difference in the regression slopes between the two treatment groups (Table 9).

DISCUSSION

Genotype-by-environment interaction: In this study, we found evidence for significant GEI in body size of nestling blue tits in 1 of the 3 study years and in the combined data. Hence, although the overall variance due to GEI always accounted for less than 10% of all phenotypic variance, there appeared to be some genetic variation in the sensitivity to environmental effects

TABLE 7

Regressions of cross-fostered (mid)offspring tarsus length on midparent and midfoster-parent tarsus length in two offspring environments

	Midparent		Midfoster-parent	
Manipulation	b	n	b	n
Reduced	0.73 ± 0.21**	63	−0.06 ± 0.19	62
Enlarged	0.45 ± 0.19*	67	−0.11 ± 0.18	67

All b values are ± SE.

The estimates have been obtained after standardizing all values to zero mean to account for differences in means between different study years. b, slope of the regression; n, number of families. * $P < 0.05$, ** $P < 0.01$.

TABLE 8

Analysis of midoffspring tarsus length in relation to father's and mother's tarsus length in nestling blue tits

	Reduced			Enlarged		
Source	d.f.	*F*	*b*	d.f.	*F*	*b*
Year	2	3.4*		2	24.16***	
Father	1	16.29***	0.40 ± 0.10	1	8.60**	0.31 ± 0.11
Mother	1	15.76***	0.42 ± 0.10	1	13.77***	0.41 ± 0.11
Error	125			124		

The data were analyzed with ANCOVA in which the year term was introduced to the model to account for variation in offspring tarsus length between the years. All *b* values are ± SE. *b*, slope of the regression. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

among different blue tit families. Dissection of the GEI variance into its causal components (Robertson 1959) revealed that the interaction was apparent due primarily to the genetic correlation between the reduced and enlarged environments being less than one, and not due to lowered additive genetic variance in the enlarged environment. Hence, given the equal genetic variances under poor and good environments, the results reported here do not support the view that the genetic potential for adaptation under poor feeding conditions in natural bird populations is less than the genetic potential for adaptation under good feeding conditions (*cf.* Merilä 1997). Still, however, it is worth emphasizing that the lower mean fitness in poor environments might tend to make selection in poor environments less important in determining how the population evolves (Kawecki *et al.* 1997).

The presence of GEI may allow adjustment of development toward the trajectory that maximizes fitness in a particular environment (*e.g.*, Stearns 1992). For example, under poor feeding conditions, it may be advantageous to cease growth at an early stage and allocate the available energy to maintenance instead of to further growth. This is what has been called an "adaptive flexible strategy," but a similar phenotypic response may also take place as a simple consequence of stress, without any fitness benefits (Kearsey and Pooni 1996). Likewise, an "inflexible" or highly canalized genotype that produces the same phenotype irrespective of environmental conditions may be the most fit in certain situations, but it can be difficult to demonstrate that its fitness is higher than that of a more flexible genotype. Although we cannot address the possible adaptive significance of the observed GEI in this study, the bottom line is that genotypes differing in the shape and/or orientation of their reaction norms have the potential to influence the course and pace of evolution in heterogeneous environments (Via and Lande 1987; Gillespie and Turelli 1989). Hence, if different blue tit genotypes are favored under different environmental conditions, there should be no severe genetic constraints for adaptation to each of the environments.

Heritability of body size: The heritability estimates obtained from offspring-parent regressions were very similar to those obtained by Dhondt (1982) in a study of Belgian blue tits ($h^2 = 0.70 \pm 0.27$), and to that reported earlier from this same population using a different data set ($h^2 = 0.81 \pm 0.17$); Merilä and Wiggins 1995). Although these h^2 values for tarsus length are in the upper range reported for birds in any population (see Boag and van Noordwijk 1987; Merilä 1997 for reviews), we found no evidence that the offspring-parent

TABLE 9

Comparison of heritability estimates (h^2) of blue tit tarsus length in reduced and enlarged broods in different study years as revealed by midparent-midoffspring rergressions

	Reduced			*Enlarged*					
	h^2	cov_{OP}	*n*	h^2	cov_{OP}	*n*	V_P	F_{equal}	F_{zero}
1993	0.61 ± 0.12**	0.0715	55	0.49 ± 0.15**	0.0569	55	0.1160	0.42	17.25***
1994	0.87 ± 0.29**	0.0637	30	0.76 ± 0.52	0.0541	29	0.0687	0.07	4.76*
1995	0.85 ± 0.21**	0.1282	45	0.72 ± 0.22**	0.1036	45	0.1430	0.31	6.81**
All	0.75 ± 0.12***	0.0850	130	0.63 ± 0.14***	0.0716	129	0.1130	0.72	21.74***

All h^2 values are ± SE. cov_{OP}, midoffspring-midparent covariance; *n*, number of families; V_P, phenotypic variance of midparent values; F_{equal}, multivariate test-statistics for the null hypothesis of equality of slopes; F_{zero}, multivariate test statistics for testing the null hypothesis that both slopes are equal to zero. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

resemblance was due to genotype-environment correlation, as there was no resemblance between offspring and their foster parents. Such an environmentally induced similarity among parents and offspring could arise, for example, if the largest parents occupied the best territories and thereby enhanced the growth performance of their offspring. However, apart from a few notable exceptions (James 1983; Larsson and Forslund 1992), there is little evidence for such effects from natural bird populations, as none of the cross-fostering studies performed so far have found any indications of genotype-environment correlation in body-size traits (Smith and Dhondt 1980; Alatalo and Lundberg 1986; Wiggins 1989; Gustafsson and Merilä 1994; Smith 1994).

One of the striking findings of this study was the conspicuous disagreement of heritability estimates from full-sib and parent-offspring analyses. While the estimates from full-sib analyses and parent-offspring regressions were more similar when based on offspring grown in reduced broods, full-sib estimates obtained using offspring grown in enlarged broods were much lower than equivalent estimates from parent-offspring regressions. This contrasts with the observation that full-sib estimates of heritability usually exceed parent-offspring estimates (*e.g.*, Larsson 1993; Merilä and Gustafsson 1993). However, the low full-sib estimates of heritability in enlarged broods resulted from much higher environmental—and, hence, total phenotypic variances (and possibly genetic variances)—in enlarged broods compared with reduced broods and the parents. This suggests that either much selection occurs between fledging and first breeding, and/or that growth of smaller nestlings continues after fledging. It is quite likely that both processes will account for part of the higher phenotypic variance in enlarged broods, as some growth was observed in the poor weather year (1994) after the nestlings were measured, and selection against small-sized nestlings has been repeatedly observed in this (Nur 1984) as well as in other phylogenetically and ecologically closely related species (Alatalo *et al.* 1990; Lindén *et al.* 1992).

It is worth noting that the estimated midparent-midoffspring covariances tended always to be higher for offspring in the reduced environment than for offspring in the enlarged environment (Table 9). This suggests either that (1) the additive genetic variance is higher in the reduced environment and/or that (2) the genetic correlation between parental and offspring environment is higher when offspring are in the reduced environment than when they are in the enlarged environment (see Introduction). For both environments, the estimated covariances were always smaller than the corresponding Box of origin variance components from the full-sib analyses (Table 5). This difference could have a variety of explanations. If additive genetic variances are the same in each environment, and if maternal and dominance effects are not present, then the parent-offspring covariances should be smaller than the full-sib variance components by a fraction equal to the genetic correlation between parent and offspring environment (see Introduction). Alternatively, the Box of origin variance components could be inflated by dominance or maternal effects. It is also possible that additive genetic variances are lower among parents than among offspring because selection has reduced the variance among parents.

Confounding factors: There is ever-increasing evidence that EPP is common in many socially monogamous birds, including the blue tit. Kempenaers *et al.* (1992) found that about 31% of the blue tit nests (11% of all young) contained nestlings sired by extra-pair males. In theory, this should lead to lowered father-offspring resemblance as compared with mother-offspring resemblance (Alatalo *et al.* 1989; but see Gebhardt-Henrich and Nager 1991). We found no indications that EPP was common in our population, as the contributions of both putative parents to offspring-parent resemblance were similar. Hence, unless we assume strong maternal effects that increased the resemblance between mothers and their offspring, there is no reason to suspect that EPP has biased our estimates of heritability. Indeed, it is possible that EPP is much more frequent among the Belgian blue tits, which also show much higher frequency (≈11%; Dhondt 1987) of polygamy than that observed in our population (<1% of pairs; J. Merilä, unpublished data). In any case, as the treatments in this study were allocated randomly relative to EPP, it should not affect our inferences about relative magnitudes of genetic variance in these environments.

Maternal and common-environment effects in the form of genotype-environment correlations are a potential source of bias in all quantitative genetic studies. As discussed earlier, we found no evidence for genotype-environment correlations between parents and their offspring, but we cannot exclude the possibility that our full-sib estimates of genetic variances could include a component of maternal or early common-environment effects. As the young were swapped when they were 2 days old, effects of early caretaking and prehatching maternal effects, if present, have been included in our estimates of genetic variances and correlations from the full-sib data. However, such effects are, according to our best knowledge, likely to be small. Likewise, full-sib estimates also include a component of dominance variance, if such was present. Again, empirical data to date indicates that if such effects are present, they are typically small (Clayton *et al.* 1957; Mousseau and Roff 1987).

Offspring size and number: Our results also bear relevance to discussions of one of the major trade-offs in life history evolution: namely, that between offspring size and number (Stearns 1992). In organisms with parental care, this trade-off is assumed to arise because of the inability of parents to provide adequate food for the young in large broods (Stearns 1992). Our results show that parents caring for large broods were less suc-

cessful in providing food for the offspring than those caring for smaller broods. If we assume that the size attained by the offspring at the end of the nestling period predicts their future survival prospects (Lindén *et al.* 1992), then the observed among-family variation in the response to the two environments means also that the optimal brood size for different parental genotypes may differ depending on the environmental conditions. This might at least partly help to explain the large individual variation in blue tit clutch size (4–17 eggs, *e.g.*, Merilä and Wiggins 1995).

We further found that the experiment had different effects on offspring size in different years, the effect being most pronounced in the year when the weather conditions were particularly bad for breeding (1994). The 1994 breeding season was cold and rainy (mean temperature and total rainfall during the first two weeks of June: 11.0° and 21.1 mm), whereas 1993 and 1995 were warmer—and except for 1995, less rainy (1993: 13.5° and 0.2 mm; 1995: 13.4° and 62.2 mm). As the weather will affect the timing and abundance of caterpillar larvae, which are the main food source of tits (Cramp and Perrins 1993), we believe that the experiment-year interaction in offspring size was caused by between-year differences in food availability, so that the effect of the experiment became amplified when the feeding conditions were particularly bad.

In conclusion, our results demonstrate a weak but statistically significant GEI in body size of nestling blue tits. This interaction, representing genetic variation in sensitivity to environmental effects, was explained mainly by the genetic correlation between the two environments being less than unity. Hence, our results give no support for the view that the amount of genetic variance expressed in poor environments is necessarily less than that expressed in good environments—and, consequently, that the genetic potential for adaptive evolution differs between poor and good environments. Further, our results, together with circumstantial evidence from several other studies of natural bird populations (reviewed in Merilä 1997), suggest that GEIs may be common in natural vertebrate populations—and, consequently, that the relative fitness of different genotypes may vary according to prevailing environmental conditions.

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