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QUANTITATIVE GENETICS OF SPRINT RUNNING SPEED AND SWIMMING ENDURANCE IN LABORATORY HOUSE MICE (*MUS DOMESTICUS*)

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Abstract.—We tested the hypothesis that locomotor speed and endurance show a negative genetic correlation using a genetically variable laboratory strain of house mice (Hsd:ICR: *Mus domesticus*). A negative genetic correlation would qualify as an evolutionary "constraint," because both aspects of locomotor performance are generally expected to be under positive directional selection in wild populations. We also tested whether speed or endurance showed any genetic correlation with body mass. For all traits, residuals from multiple regression equations were computed to remove effects of possible confounding variables such as age at testing, measurement block, observer, and sex. Estimates of quantitative genetic parameters were then obtained using Shaw's (1987) restricted maximum-likelihood programs, modified to account for our breeding design, which incorporated cross-fostering. Both speed and endurance were measured on two consecutive trial days, and both were repeatable. We initially analyzed performances on each trial day and the maximal value. For endurance, the three estimates of narrow-sense heritabilities ranged from 0.17 to 0.33 (full **ADCE** model), and some were statistically significantly different from zero using likelihood ratio tests. The heritability estimate for sprint speed measured on *trial day 1* was 0.17, but negative for all other measures. Moreover, the additive genetic covariance between speeds measured on the two days was near zero, indicating that the two measures are to some extent different traits. The additive genetic covariance between speed on *trial day 1* and any of the four measures of endurance was negative, large, and always statistically significant. None of the measures of speed or endurance was significantly genetically correlated with body mass. Thus, we predict that artificial selection for increased locomotor speed in these mice would result in a decrease in endurance, but no change in body mass. Such experiments could lead to a better understanding of the physiological mechanisms leading to trade-offs in aspects of locomotor abilities.

Key words.—Behavior, body mass, cross-fostering, domestication, genetic correlation, heritability, locomotion, maternal effects, physiology, relaxed selection, trade-off.

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The concept of constraints on adaptive evolution has received considerable attention from evolutionary biologists in recent years. Although numerous kinds of constraints have been defined (e.g., developmental, ecological, mechanical, physiological, phylogenetic: see discussions in Maynard Smith et al. 1985; Arnold 1992), identification of genetically-based constraints is of prime importance for making predictions about limits on the direction and rate of adaptive microevolution (Rose et al. 1987; Arnold 1992). Genetic constraints are defined herein as aspects of the inheritance of traits that limit "the potential for natural selection to result in the most direct ascent of the mean phenotype to an optimum" (Loeschcke 1987, p. 1).

For polygenic traits, quantitative genetic analyses can be used to study genetic constraints. Quantitative genetics comprises statistical methods that partition the observed, within-population phenotypic variation of a continuously distributed trait (or traits) into components attributable to a causal model of shared additive and nonadditive genetic and environmental effects. Various parameters can be estimated, but two are of particular interest: narrow-sense heritability (h^2, the proportion of additive genetic variance to total phenotypic variance) and genetic correlation (r_A, the standardized covariance between additive genetic effects for two traits). Heritabilities and genetic correlations can be estimated directly from sets of relatives obtained from breeding designs or indirectly from the results of artificial selection experiments (Falconer 1989; Boake 1994).

Genetic constraints for quantitative traits can be identified as a lack of heritability for a single trait or by the nature of genetic correlations between traits. Only traits with substantial h^2 can respond rapidly to selection (Falconer 1989; Boake 1994 and references therein). Thus, barring an input of new genetic variation, the absence of heritable genetic variation for a trait would constitute a constraint on adaptive evolution. Most studies, however, find evidence for ample additive genetic variance for various behavioral, morphological, and physiological traits, and even for major components of fitness, both for laboratory and wild populations (Mousseau and Roff 1987; Roff and Mousseau 1987; Boake 1994 and references therein).

Genetic covariances (typically presented as correlations) can slow the rate at which selection can move a multivariate phenotypic mean value for a population towards its optimum value (Lande 1988; Arnold 1992). Genetic correlations are not fixed entities and can themselves be altered by selection (Lande 1984; Brodie 1992). For example, correlational selection (i.e., selection favoring particular trait associations [Lande and Arnold 1983]) can lead to the formation of genetic covariance between traits, either by favoring pleiotropic mutations (one gene affects multiple traits) or by creating linkage disequilibrium (nonrandom association of alleles at different loci). The relative constancy of genetic variance-covariance matrices is important for long-term prediction of response to selection (Lande 1988; Barton and Turelli 1989).

The primary objective of the present study was to deter-

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mine whether locomotor speed and endurance exhibit a negative additive genetic covariance in a random-bred strain of laboratory house mice. Sprint speed and endurance are ecologically relevant measures of locomotor abilities (Djawdan and Garland 1988; Bennett and Huey 1990; Djawdan 1992; Garland and Losos 1994). Various lines of evidence (see Discussion) suggest that locomotor speed and endurance are subject to a physiological trade-off (see also Garland 1988, 1994a; Sorci et al. 1995). This physiological trade-off may be reflected as a negative genetic correlation (cf. Rose et al. 1987; Garland 1988). A negative genetic correlation would qualify as an evolutionary constraint, because both speed and endurance are expected to be under positive directional selection in nature (e.g., Jayne and Bennett 1990).

Our second goal was to test the hypothesis that locomotor abilities are positively genetically correlated with body mass. Body size is one of the most labile features of mammalian evolution (Damuth and Macfadden 1990). Many aspects of physiology are strongly correlated with body size (references in LaBarbera 1989; Garland and Carter 1994), including some aspects of locomotor abilities. Selection on locomotor abilities might, therefore, be an important factor affecting body size evolution, or vice versa.

Theoretical arguments suggest that maximal sprint running speed should either scale positively with body mass (e.g., $M^{0.17}$, dynamic similarity model; $M^{0.25}$, elastic similarity model; $M^{0.40}$, static similarity model) or be independent of mass (i.e., scale as M^0, geometric similarity model) (references in Schmidt-Nielsen 1984; Astrand and Rodahl 1986). Empirically, maximal running speed has been shown to be nonlinearly related to body mass across 143 species of mammals, with species of intermediate size being the fastest (Garland 1983; Garland et al. 1988; cf. Chappell 1989). Among species within Rodentia, speed is not clearly related to body size, at least if the large and slow beaver is included in analyses (Djawdan and Garland 1988; Garland et al. 1988). For humans, maximal running speed is independent of body height within age groups (Astrand and Rodahl 1986).

To our knowledge, no explicit theoretical prediction exists for the scaling of endurance with body mass (cf. Schmidt-Nielsen 1984; Garland 1994b). Available comparative data indicate that maximal aerobic speed (the speed at which the maximal rate of oxygen consumption is reached and, therefore, an index of stamina) is proportional to $M^{0.19}$ across 39 species of mammals (Garland et al. 1988). Within the rodent family Heteromyidae, running endurance is positively correlated with body mass across four bipedal species, but not across four quadrupedal species (Djawdan 1992). Despite the intriguing phenotypic studies of locomotor performance, information on the genetic covariance between body size and locomotor abilities is not yet available for any organism.

Materials and Methods

Strain History and Animal Husbandry

We studied the random-bred Hsd:ICR strain of house mice (*Mus domesticus*) (see also Friedman et al. 1992; Hayes et al. 1992; Dohm 1994; Dohm et al. 1994; Richardson et al. 1994; Garland et al. 1995; Dohm et al., unpubl.). This ICR strain is genetically heterogeneous (Hauschka and Mirand 1973), and has served as a model system for numerous quantitative genetic analyses (e.g., Riska et al. 1984; additional references in Dohm 1994). Hsd:ICR mice are descendents of 100 female and 30 male Swiss-Webster albino mice purchased in 1947 for the Institute for Cancer Research in Philadelphia, Pennsylvania (Hauschka and Mirand 1973). All Swiss-Webster mice, in turn, are descendents of 9 noninbred mice (2 male, 7 female) brought to the United States from Switzerland in 1926 (Hauschka and Mirand 1973; Dohm 1994, ch. 1). Several lines of ICR mice were established at the Institute for Cancer Research following a selection and husbandry program to increase litter size and weaning success (Hauschka and Mirand 1973). These lines were then made available to the research community. Additional lines were subsequently developed at Charles River Laboratories (Wilmington, Massachusetts). In 1983, Harlan Sprague Dawley purchased ICR mice from the Charles River Laboratories (V. L. White, Harlan Sprague Dawley, Indianapolis, Indiana, pers. comm., October 1994). The ICR strains are now maintained in 10 different closed colonies, each consisting of several hundred to thousands of individuals, at Harlan Sprague Dawley facilities throughout the United States (V. L. White, pers. comm., October 1994). Additional details of the history of the strain are available in Dohm (1994).

Data were obtained from a total of five replicate blocks of mice, each consisting of five founder males and up to 22 founder females and their offspring. Founder mice were weaned at 19 d of age and immediately shipped to Madison from Harlan Sprague Dawley, Inc. (room 202, Barrier A), Indianapolis, Indiana. Mice were toe-clipped for individual identification upon their arrival. Mice were housed in standard clear plastic cages (27 cm long, 17 cm wide, 12.5 cm deep) with metal or wire tops and pine shavings as bedding material. Males were placed in individual cages and females were placed in groups of four or five per cage. Water and food (Purina Rodent Chow, High-Energy Formula) were always available except prior to measurements of basal metabolism (Dohm et al., unpubl.). Photoperiod was 12:12 light: dark, centered at 1300 h (CST), and room temperature was maintained at approximately 21°C.

Breeding Design

Founder mice were paired for breeding one day after the swimming endurance trials (see next section). Each male was paired with four or five randomly selected females. These harems (with males) were left together for 8–11 d, after which males were removed and females were placed in individual cages. We checked for pups once daily (about 1800 h CST) and offspring were immediately weighed and toe-clipped.

We employed a nested breeding design, with cross-fostering (cf. Newman et al. 1989), to allow unique identification of the relative magnitude of direct genetic and environmental effects on individual variation in phenotypic traits. At birth, we cross-fostered up to four pups per dam between two or more dams who had given birth on the same day. Simultaneously, litter sizes were standardized to eight (or fewer) pups per dam. Pups were randomly selected for inclusion with the constraint that sex ratios were equalized. The cross-fostering design included 67 of the 78 families born during this study

(see Results). Pups were weaned at 19 d of age and 4 pups per dam (2 cross-fostered, 2 not cross-fostered) were randomly selected for later measurement. Dohm (1994, ch. 6) provides additional husbandry and breeding details.

Measurements and Measurement Chronology

Throughout, we use *maximal* to refer to measures of performance judged as the highest or best obtained under the conditions specified (cf. Djawdan and Garland 1988; Djawdan 1992; Garland 1994a,b; Garland and Losos 1994 and references therein). We do not present analyses on the averages of the daily maximal values herein because (1) maximal performances are more appropriate measures of physiological limits (see Discussion); and (2) quantitative genetic results were similar (Dohm 1994, ch. 3).

Forced maximal sprint running speed was measured on an 8-m long by 16.5cm wide photocell-timed racetrack, with short-pile artificial grass (plastic) substrate (Djawdan and Garland 1988; Friedman et al. 1992; Dohm et al. 1994; Garland et al. 1995). Twelve sets of photocells spaced at 0.5 m intervals were interfaced to a microcomputer. On each trial day, mice were timed while being chased five times in succession along the racetrack with a cardboard padded meter stick. Between timed runs, mice were carried in a small plastic cup back to the start of the racetrack. For each of these five runs, the single highest speed attained over any 1.0 m interval (three consecutive photocells) was recorded. Trials were conducted on two consecutive days. The highest value from each trial day was assessed for repeatability (calculated as Pearson product-moment correlation because performance showed a statistically significant increase in speed from *trial day 1* to *day 2*, see Results). We then evaluated the quantitative genetic bases of three measures of sprint running speed: the highest speed on *trial day 1*, the highest speed on *trial day 2*, and the single highest (*maximal*) value from either day.

Swimming endurance was measured following standard protocols (Friedman et al. 1992; Dohm et al. 1994 and references therein). We used a swimming protocol rather than a treadmill test to measure endurance because we were unable to obtain repeatable measures of endurance using the treadmill (consistent with previous studies: Friedman et al. 1992; Dohm 1994; and references therein). Moreover, in laboratory rodents, a significant percentage of individuals simply refuse to run to exhaustion on a treadmill. For example, Bedford et al. (1979) noted that about 10% of mature rats will not run on a motorized treadmill, but that these animals show no statistically significant difference in physiological determinants of performance. All mice we have tested to date will swim and attempt to remain at the surface (Friedman et al. 1992; Dohm et al. 1994; this study). Although different muscles are involved (Laughlin et al. 1984), the patterns of recruitment for the different kinds of muscle fibers during exhaustive running or swimming endurance tests are broadly similar (slow-twitch oxidative → fast-twitch, oxidative, glycolytic → fast-twitch glycolytic fibers: Armstrong et al. 1974). Moreover, measures of maximal oxygen consumption obtained during exhaustive swimming protocols are similar to those obtained during treadmill running (Divine Patch and Brooks 1980).

Mice swam in white, five-gallon plastic buckets containing water (depth approximately 30 cm) at a temperature of approximately 31°C (see Table 2), the temperature yielding highest swimming times, as determined in preliminary tests (cf. Friedman et al. 1992). Mice were rinsed in a mild detergent solution (Ivory® liquid) to prevent air bubbles from clinging to fur and a 0.9g weight was attached to their tails to reduce absolute swimming times; without extra weight, many individuals could swim virtually indefinitely. (Added weights represented 3–4% of body mass.) Trials were conducted on three consecutive days, the first consisting of a 10-min training swim without added weight followed on the second and third days by the actual endurance trials. Exhaustion was judged and mice were removed from buckets when they remained under water for eight consecutive seconds (consistent with previous studies, Friedman et al. 1992; Dohm et al. 1994; and references therein). According to data in Calder (1969, p. 1077), this period of time is about one-third of what lab mice can tolerate. Initial and final water temperatures were recorded for each trial and the average was used as a covariate in statistical analyses (see next section). Swimming times from both days were assessed for repeatability (Pearson correlation); times from both days and the single higher value (*maximal*) were all used for quantitative genetic estimates.

Six measures of body mass were analyzed: mass measured at the first and second sprint speed trials; mass measured at the first and second swimming endurance trials; mass at maximal sprint speed; and mass at maximal swimming endurance. Values obtained from the trial days were assessed for repeatability (Pearson product-moment correlation). Phenotypic and genetic correlations of locomotor performance and body mass were estimated for each trial day and the corresponding body mass value.

As part of another study, basal metabolic rate (BMR) and treadmill exercise-induced maximal oxygen consumption ($\dot{V}O_2max$) were determined for the same mice (Dohm 1994; Dohm et al., unpubl.). Measurement chronology for each of the five measurement blocks was as follows: *day* 1: food removed at approximately 1800 (CST); *days* 2–4 BMR (N = 370, groups of 7 mice per d); *days* 5 and 6 sprint speed (N = 347); *days* 7 and 8 $\dot{V}O_2max$ (N = 340); *days* 9–11 swimming endurance (N = 297). Measurements of sprint speed were initiated by 34 days of age (mean ± SD = 38.2 ± 2.34, range 34–44 d). Swimming endurance measurements were completed by the time mice were 52 d of age (mean ± SD = 45.8 ± 2.90, range 41–52). Owing to technical difficulties, swimming endurance was not determined for founder mice of the first and second measurement blocks; similarly, sprint speed was not measured for founder mice of the first block.

Data Analyses

Quantitative genetic parameter estimates for all traits were based on residuals from multiple regression equations. Although many pair-wise correlations among the potential covariates were significantly different from zero, none were large

(Dohm 1994, table IX). Regression equations were obtained by using a stepwise selection algorithm, with entry and removal levels set at $P = 0.05$ and $P = 0.10$, respectively. The reason for using these residuals was to remove possible confounding effects of covariates (e.g., age, time at measurement) and fixed effects that can obscure genetic differences among individuals. Sex, measurement block, and whether an individual was a breeder obtained from Harlan Sprague Dawley or an offspring born in our laboratory (i.e., founder status) were scored as dummy variables. For sprint speed, we also used dummy variables coding for observer; for swimming endurance water temperature was also included as a potential covariate. For time and age at measurement, orthogonal polynomials (e.g., Z-transformed Age^2) were also used to allow for nonlinear associations with the dependent variable. For regressions in which the squared term was found to be statistically significant, but the first-order term was not included in the final stepwise model, we also forced the first-order variable into the equation prior to computing residuals (Montgomery and Peck 1992). Dependent variables were transformed to improve normality and/or homogeneity of variances in relation to body mass, age, etc., and the behavior of residuals from the final multiple regression equations was checked by looking at distributions of standardized residuals and normal probability plots (Montgomery and Peck 1992). Estimation of additive genetic (V_A), dominance genetic (V_D), and postnatal common environmental (V_C) variances for traits was based on the following linear model (expressed in matrix form):

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_a A} + \mathbf{Z_d D} + \mathbf{Z_c C} + \mathbf{IE} \qquad (1)$$

where $\mathbf{y}$ is the vector of observations (e.g., residual maximal sprint speeds for individual mice), $\mathbf{b}$ is the vector of fixed effects, and $\mathbf{A}$, $\mathbf{D}$, $\mathbf{C}$, and $\mathbf{E}$ are the vectors of additive breeding values, dominance deviations, common environmental, and specific environmental effects, respectively. $\mathbf{X}$ is the incidence or design matrix for the grand mean; $\mathbf{Z_a}$, $\mathbf{Z_d}$, $\mathbf{Z_c}$ represent the design matrices relating observations to their respective random effects (i.e., contained in $\mathbf{A}$, $\mathbf{D}$, and $\mathbf{C}$). $\mathbf{Z}$ matrices are partitioned into a null matrix (all zeros) for individuals without records and an identity matrix (diagonal matrix with 1's along the diagonal) for individuals with records. For example, every row of $\mathbf{Z_c}$ is null except for a "1" under the column pertaining to a known nurse dam. The incidence matrix $\mathbf{I}$ is an identity matrix for individual error. This model is referred to as the "full" (**ADCE**) model in the present study. The structural model does not allow for covariances among the additive, dominance, common environment, or specific environmental effects; thus, in effect, we assume these covariances to be negligible. The model described above is usually termed an "animal model" (Meyer 1983), because all individuals are included, whether or not the individual has been measured, in order to provide information on relationships.

The means (expectations) and variances for the model are:

$$\mathrm{E}\begin{vmatrix}\mathbf{y}\\ \mathbf{a}\\ \mathbf{d}\\ \mathbf{c}\\ \mathbf{e}\end{vmatrix} = \begin{vmatrix}\mathbf{Xb}\\ \mathbf{a}\\ \mathbf{d}\\ \mathbf{c}\\ \mathbf{e}\end{vmatrix}, \qquad (2)$$

$$\mathrm{V}\begin{vmatrix}\mathbf{y}\\ \mathbf{a}\\ \mathbf{d}\\ \mathbf{c}\\ \mathbf{e}\end{vmatrix} = \begin{vmatrix}\mathbf{V_{com}} & \mathbf{Z_a A}\sigma_a^2 & \mathbf{Z_d D}\sigma_d^2 & \mathbf{Z_c C}\sigma_c^2 & \mathbf{I}\sigma_e^2\\ \mathbf{AZ}'\sigma_a^2 & \mathbf{A}\sigma_a^2 & 0 & 0 & 0\\ \mathbf{DZ}'\sigma_d^2 & 0 & \mathbf{D}\sigma_d^2 & 0 & 0\\ \mathbf{CZ}'\sigma_c^2 & 0 & 0 & \mathbf{C}\sigma_c^2 & 0\\ \mathbf{I}\sigma_a^2 & 0 & 0 & 0 & \mathbf{I}\sigma_e^2\end{vmatrix} \qquad (3)$$

where E is the expectation, $\mathbf{V_{com}} = \mathbf{Z_a}(\mathbf{A}\boldsymbol{\sigma}_a^2)\mathbf{Z_a}' + \mathbf{Z_d}(\mathbf{D}\boldsymbol{\sigma}_d^2)\mathbf{Z_d}' + \mathbf{Z_c}(\mathbf{C}\boldsymbol{\sigma}_c^2)\mathbf{Z_c}' + \mathbf{I}\boldsymbol{\sigma}_e^2$, and $\boldsymbol{\sigma}_a^2$ is the additive genetic variance, $\boldsymbol{\sigma}_d^2$ is the dominance genetic variance, $\boldsymbol{\sigma}_c^2$ is the postnatal maternal and common environmental variance, $\boldsymbol{\sigma}_e^2$ is the specific environmental variance, and $\mathbf{A}$, $\mathbf{D}$, and $\mathbf{C}$ are the additive, dominance, and post-natal common environmental relationship matrices. Additive variance includes all additive epistatic effects (e.g., V_{AA}, V_{AAA}); and all other epistatic genetic effects (e.g., V_{AD}, V_{DD}) are contained by dominance variance. Dominance variance in the above model is confounded by prenatal maternal effects (dominance variance herein represents the statistical deviation from additive genetic variance [Falconer 1989]). Therefore, estimates of V_D equal dominance genetic variance per se only if epistasis and prenatal maternal effects are absent (see Dohm 1994 for additional details).

Model Fitting

Estimates of quantitative genetic parameters were obtained using Shaw's (1987) maximum-likelihood (ML) programs, "Quercus," running on an HP-9000 minicomputer. Description and rationale for using maximum likelihood-based methods for variance component estimation have been reviewed extensively (e.g., Meyer 1983; Shaw 1987; Searle et al. 1992). In brief, variance component estimation by ML methods are preferred over ANOVA procedures because ML estimates have several desirable statistical properties, including asymptotic normality (i.e., distributions of ML estimates approach the normal as sample size increases) and efficiency (i.e., all information available about relationships is used in an optimal way) (Searle et al. 1992). We used the restricted maximum likelihood (REML) procedure and iterations were continued until the differences in successive likelihoods was less than 0.0001. REML maximizes the part of the likelihood that is independent of the fixed effects (e.g., grand mean). In effect, estimates of the variance components are obtained after adjusting for the fixed effects with linear functions of the data. As noted above, multiple regression equations were used to adjust the data prior to genetic analyses; the REML program used in this study does not allow for linear covariates (e.g., body mass, age).

In addition to determining whether each parameter in the specified model was statistically significant, we evaluated the goodness-of-fit of the structural model to the data. Briefly, a nested submodel was compared to a less constrained model and the fit of the former to the data was assessed. The less-constrained model would be preferred over the nested model if the goodness-of-fit measure is larger than a specified critical value. Thus, the full model is considered to be a hypothesis subject to evaluation by comparing the fit functions to a series of fits for the nested hypotheses.

Univariate Models.—We first used single-character data sets to obtain parameter estimates and model-fit statistics for

the full model (i.e., the model containing all four estimable variance components: $V_A + V_D + V_C + V_E$) and for all nested submodels (**ADE**, **ACE**, **AE**, **CE**, etc.). Nested submodels were compared to the full model by constraining a specified parameter to zero and obtaining a new likelihood. Twice the difference in log-likelihoods (called the deviance score) is distributed approximately as a chi-square with the degrees of freedom taken as the number of parameters constrained to zero. This deviance statistic (hereafter referred to as χ^2) is then viewed as a goodness-of-fit measure (Searle et al. 1992); large χ^2 values indicate poor fit to the data. If the deviance statistic was significant at $P < 0.05$, then the submodel was rejected in favor of model(s) with more parameters.

Models excluding the specific environmental component (e.g., **CD**, **AD**) were not considered because such models predict perfect resemblance between relatives. The "best-fitting model" was identified by comparing the relative differences in χ^2 across the nested models and examining the significance of the variance components estimated within each model.

Bivariate Models.—We constructed models that partitioned the covariation between measures of, for example, sprint speed and endurance, and between the performance measures and body mass, into genetic and environmental sources of covariation. These different models included: (1) **AE** models for both traits, with only additive genetic and specific environmental sources of covariation; (2) **ADE** or **ACE** models for both traits, with additive genetic, specific environmental, and either dominance or common environmental sources of covariation; (3) the **ADCE** or full model; and (4) several reduced models in which one or more variance components is not represented in the univariate model of the other trait (e.g., **AE** model for the first trait and **ADE** model for the second trait; therefore, only additive genetic and specific environmental sources of covariation were specified). Only the additive genetic covariances were tested for statistical significance. For this significance testing, the likelihood of a model with the additive genetic covariance constrained to zero was compared to that of the less-constrained model using 1 df.

Results

A total of 561 offspring from 78 litters were born between 11 January 1989 and 6 February 1990 (see also Hayes et al. 1992; Dohm 1994). The proportion of pairings producing litters did not differ significantly among measurement blocks (Pearson $\chi^2 = 8.022$, df = 4, $P = 0.091$). Mean litter size at birth was 9.44 (range 2–16 pups; Hayes et al. 1992, Table 1 and Fig. 4; Dohm 1994). Body mass of offspring at birth ranged from 1.02 to 2.12 g. Results from multiple regression analyses indicated that dam body mass and her age at the birth of her litter, taken together, accounted for 33.3% and 37.4% of the variation in litter size and in total litter mass, respectively (Hayes et al. 1992, Table 2). As noted in the Materials and Methods, the cross-fostering design included 67 of the 78 families born during this study. These 67 dams successfully weaned 513 offspring (98%; litter size was standardized at up to eight pups per dam). We did not measure offspring from the 11 families not included in the cross-fostering protocol.

TABLE 1. Descriptive statistics for locomotor performance traits and body mass measured on random-bred, genetically variable Hsd: ICR house mice (*Mus domesticus*).

Traits	*N*	Mean	SD	Min	Max
Sprint speed (m/s), trial 1					
Females	200	1.26	0.224	0.77	2.00
Males	147	1.25	0.251	0.59	2.37
Sprint speed, trial 2					
Females	200	1.38	0.253	0.84	2.24
Males	147	1.31	0.260	0.71	2.10
Maximal sprint running speed[a]					
Females	200	1.42	0.227	0.94	2.24
Males	147	1.38	0.248	0.71	2.37
Body mass on maximal sprint trial					
Females	200	22.12	2.305	13.23	39.25
Males	147	26.41	2.967	18.22	34.05
Swimming endurance (min), trial 1					
Females	162	11.4	26.09	0.45	278.2
Males	135	6.7	6.69	0.32	35.1
Swimming endurance, trial 2[b]					
Females	160	11.2	16.00	0.33	134.7
Males	135	8.7	16.14	0.35	165.9
Maximal swimming endurance[c]					
Females	162	15.1	27.24	0.72	278.2
Males	135	10.4	16.30	0.42	165.9
Body mass on maximal endurance trial					
Females	162	22.62	2.001	17.61	29.31
Males	135	28.29	2.841	19.86	35.95

[a] Mean age (± SD) at maximal sprint speed was 38.8 (± 2.44), range 34–45 d.
[b] Two mice were not given a second swimming test because each swam for more than 60 min on the first trial.
[c] Mean age (± SD) at maximal swimming endurance was 45.3 (± 3.00), range 40–52 d.

Descriptive Statistics and Variability

Descriptive statistics for speed and endurance are given in Table 1. Coefficients of variation (CV) were greater for endurance (e.g., 82–188% for maximal swimming endurance) than for forced sprint speed (e.g., 14–21% for maximal sprint speed). Log transformations were used for all measures of sprint running speed and swimming endurance because distributions were positively skewed. Square-root, power, and other transformations did not improve normality as compared to the log transformation.

Repeatability

Measurements for all traits were repeatable ($P < 0.0001$) between trial days. Pearson product-moment correlations were 0.486 for log-transformed speed (0.519 for nontransformed), 0.639 for log swimming endurance, and 0.942 and 0.987 for log body mass over the sprint speed and swimming endurance trial days, respectively. Correlations of the trait residuals (see below; all log-transformed prior to regression analyses) were similar (speed, 0.515; swimming endurance,

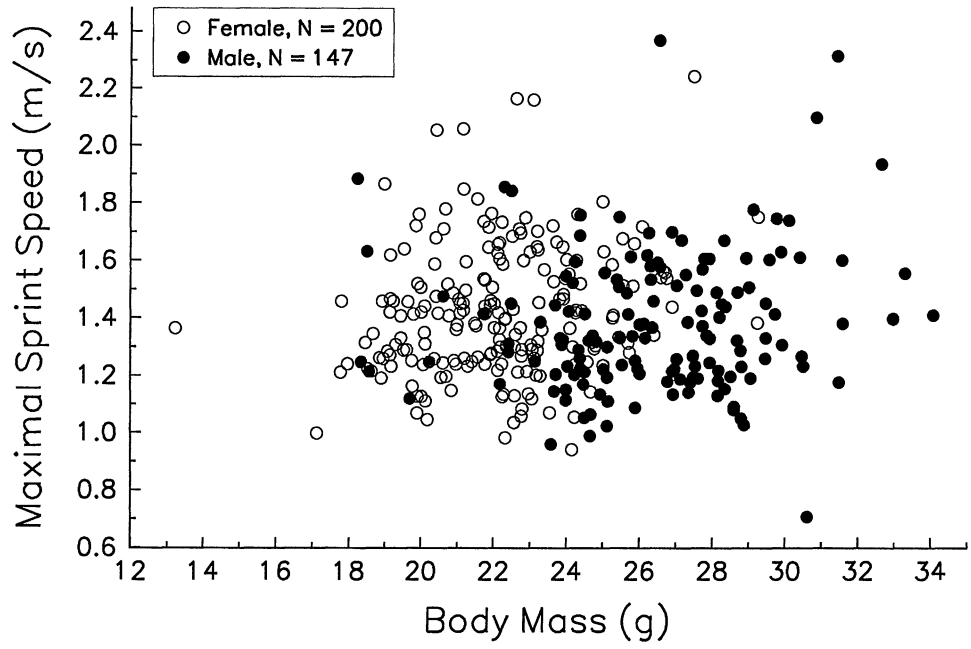

FIG. 1. Maximal sprint running speeds of 347 random-bred Hsd:ICR house mice in relation to body mass. The effects of other covariates (see Table 2) are not controlled. We did not exclude from analyses the sprint speed data for a male (mass 30.6 g) that was an outlier following log transformation (see Fig. 3 of residuals), because the mouse's performance was consistent (0.59 m/s on trial 1 versus 0.71 m/s on the second day), our notes did not indicate that the mouse "refused" to run, and he performed similarly for different observers on the two trial days.

0.616; body mass @ speed, 0.905; body mass @ endurance, 0.970).

Small, but statistically significant increases between *trials 1* and *2* were found for sprint speed (not transformed; median = 5.3%, range = −55.4–40.1%; paired $t = 7.36$, df = 346, $P < 0.001$), body mass @ speed (not transformed; median = 2.3%, range = −15.9–45.3%; paired $t = 9.90$, df = 346, $P < 0.001$), and body mass @ endurance (not transformed; median = 0.83%, range = −7.9–10.5%; paired $t = 6.99$, df = 294, $P < 0.001$). Sixty-three percent (219/347) ran faster on *trial day 2*. A subset of the mice were also tested for sprint speed on a third consecutive day. This addition of a third day for sprint speed did not significantly improve repeatability and so was discontinued ($r = 0.586$ between *trials 2* and *3*; mean (± *SD*) speed on *trial 3* = 1.42 ± 0.283 m/s, $N = 47$). Swimming endurance increased slightly (log-transformed; mean = 2.9%, range = −90.5–163.7%) from *trial 1* to *trial 2*, but the difference was not statistically significant (paired $t = 1.37$, df = 294, $P = 0.171$); 56% (164/295) of the mice swam longer on the *trial day 2*. (Hereafter, we discuss results for transformed variables only, unless stated otherwise.)

Removing the Contributions of Significant Covariates Prior to Genetic Analyses

Body mass was not significantly correlated with sprint speed (Fig. 1) nor with swimming endurance (Fig. 2). However, additional covariates explained about 25% and 11% of the variance in log sprint speed and log swimming endurance, respectively (Table 2; covariates of body mass are also listed). For example, male founder mice and mice tested at older ages tended to be faster sprinters than were the offspring and younger mice. Both male and female founder mice tended to swim for longer times than did offspring. Toe-clipping had a small (coefficient of determination, $r^2 < 0.03$), statistically significant negative effect on sprint speeds, but did not affect swimming times (Table 2). Total number of toes clipped ranged from 1 to 6, with no more than 2 toes clipped per foot (Dohm 1994, Appendix II). Distributions of computed residuals for both log maximal and log average sprint running speed were not significantly skewed, but remained kurtotic (Dohm 1994). All log swimming endurance measures were approximately normally distributed (Dohm 1994).

Heritability and Univariate Model-Fitting

Estimates of narrow-sense heritabilities for all traits are reported in Table 3. Complete results of all genetic models tested, estimates of variance components, and standard errors for variance components are provided in Dohm (1994, tables XI–XV).

Sprint Running Speed.—General results of the model-fitting approach can be summarized as follows (Dohm 1994, table XI). First, the three-component models (**ACE**, **ADE**, and **DCE**) generally fit the data better than did the full (**ADCE**) model (i.e., the χ^2 values for each comparison were all less than the critical value of 3.841). Second, estimates of V_A, and therefore of heritability, were negative in four of six three-component models (Table 3). The negative estimates generally accounted for only a small proportion of the total variance (mean 12%), so they were probably indicative of high sampling variation rather than invalidity of the models per se (cf. Searle et al. 1992; Shaw and Platenkamp 1993;

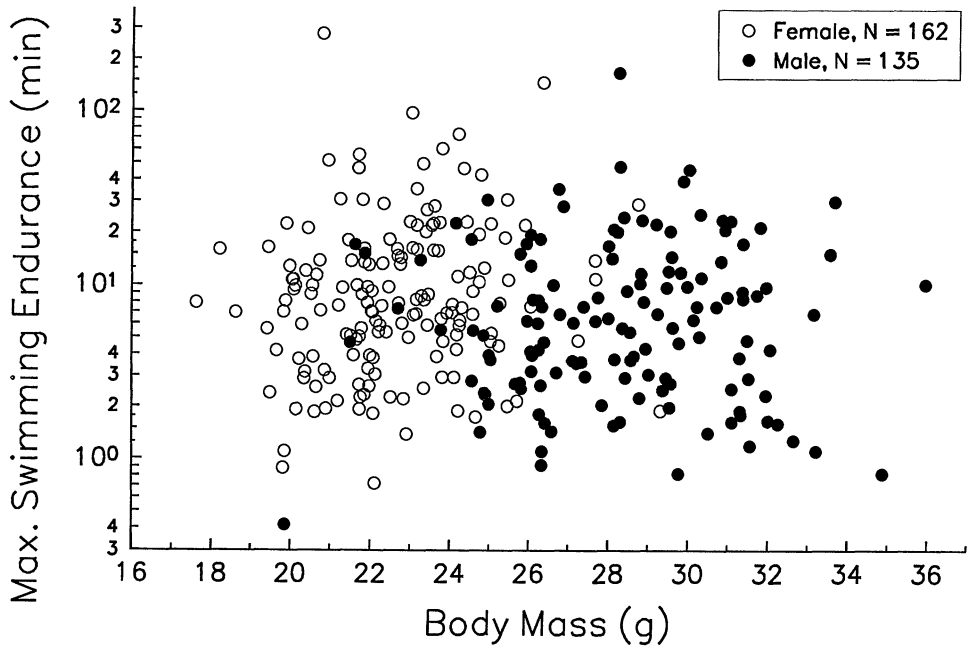

FIG. 2. Maximal swimming endurance times of 297 Hsd:ICR mice in relation to body mass. The effects of other covariates (see Table 2) are not controlled.

see also Dohm 1994). Third, additive genetic effects cannot be removed from the model for sprint speed on *trial day 1* without worsening the fit. In contrast, common environmental sources (**CE** model) contributed significantly to differences among individuals for maximal sprint speeds, whereas additive and dominance genetic effects were negligible (Dohm 1994). Lastly, no estimate of h^2 is available for sprint speed on *trial day 2* because all models yielded negative estimates of V_A (Table 3); furthermore, the **E** model was not rejected for *trial day 2* (Dohm 1994).

For sprint running speed on *trial day 1*, further nested comparisons (see Dohm 1994) indicated that: (1) the component for common environment (**C**) can be eliminated from the model without significantly worsening the fit; (2) additive genetic effects cannot be eliminated from the model; and (3) the **AE** model fits the data better than does the **DE** model (i.e., slightly higher log-likelihood score and χ^2 for test of **AE** versus **E** is statistically significant). Narrow-sense heritability estimates were consistent (approximately 0.17) across all four models (Table 3).

Family resemblance for *maximal* sprint running speed may be more related to shared environment rather than to either additive or dominance genetic effects (Table 3). The **ADE** model showed significant deterioration as compared to the

TABLE 2. Significant ($P < 0.05$) covariates for body mass and for organismal performance traits. Values are partial r^2 in percent. For traits in which the second-order term (Age^2, $Temp^2$), but not first-order term (Age, Temp), were found to be significant, we forced the first-order terms into the model before obtaining residuals. Residuals from the multiple regression equations were used for estimation of quantitative genetic parameters. Body mass was not a significant covariate of either sprint speed or swimming endurance; measurement block 5 and number of toes clipped[2] were not significant covariates for any trait and so are not listed.

Trait	Sex	Sex x founder	Founder	Age	Age^2	Measurement block[a] 2	3	4	No. of clipped toes	Observer[b]	Water temp.[c]	Multiple R^2
Sprint running speed												
log trial 1	—	3.1	—	<0.1	8.6	—	—	2.6	2.4	—		16.7
log trial 2	2.3	3.2	—	0.5	9.8	1.1	1.5	2.8	1.9	—		23.1
log maximal	2.2	2.6	—	0.5	10.6	1.6	2.1	—	2.6	2.7		25.0
Body mass @ max	40.0	—	2.0	11.9	—	—	—	1.9	—	—		55.8
Swimming endurance												
log trial 1	—	—	5.3	—	—	4.1	—	—	—	—	—	9.4
log trial 2	—	—	1.9	—	—	3.0	—	—	—	—	—	4.9
log maximal time	1.2	—	3.6	—	—	4.5	—	—	—	—	1.7	11.1
Body mass @ max	57.8	—	1.0	5.0	0.5	—	2.4	7.2	1.3			66.4

[a] Coded as four dummy variables (measurement block 1 = 0, blocks 2, 3, 4, 5 coded as 1).
[b] Sprint speed observer effect, coded as three dummy variables (A,A = 0, combinations of A,B B,A B,B were coded as 1).
[c] Mean water temperature (± SD) was 30.8 (± 0.88), range 27.9–32.6°C.

TABLE 3. Standardized estimates of variance components from univariate genetic models. Data were residuals from multiple regression equations. We used single-character data sets to obtain parameter estimates and model-fit statistics for the ADCE model (i.e., the model [1] containing all four estimable variance components: $V_A + V_D + V_C + V_E$) and for all nested submodels (e.g., [2] ADE, [3] ACE, [4] AE), using Shaw's (1987) REML program modified to account for the cross-fostering breeding design. Tests of statistical significance of various components and of a particular model's goodness-of-fit were conducted as follows. First, we obtained parameter estimates for the full ADCE model. Second, we proceeded to constrain one or more parameters to zero and obtained new parameter estimates under the reduced, nested submodel (e.g., AE). The goodness-of-fit for the reduced model relative to the fit of the full model was then assessed by comparing the log-likelihoods. Twice the difference in log-likelihoods is distributed approximately as a chi-square (X^2). Each test has 1 df; critical X^2 values are 3.84 at $P \leq 0.05$ and 2.71 at $P \leq 0.10$. Components as a proportion of the total phenotypic variance are: h^2, narrow-sense heritability = additive genetic variance; d^2, dominance genetic [plus prenatal maternal] variance; c^2, postnatal maternal effects and common environmental variance; e^2, error variance; NA, not applicable. (See text for further explanations and Dohm [1994: Tables X–XIV, pp. 110, 112–119] for nonstandardized estimates of variance components, model fit statistics, and significance levels of other models.)

Model	Test	X^2	h^2	d^2	c^2	e^2	X^2	h^2	d^2	c^2	e^2	X^2	h^2	d^2	c^2	e^2
		Sprint speed, trial 1					*Sprint speed, trial 2*					*Maximal sprint speed*				
1. ADCE	NA	NA	.169	.214	−.078	.695	NA	−.224	.689	.080	.455	NA	−.068	−.211	.295	.984
2. ADE	vs 1	0.95	.158	.095		.747	1.16	−.219	.819		.400	7.71	−.054	.322		.732
3. ACE	vs 1	0.56	.195		−.055	.860	3.34	−.120		.178	.942	0.30	−.107		.263	.844
4. AE	vs 2	0.12	.173			.827	6.95	−.038			1.038	0.90	.040			.960
	vs 3	0.51					4.76					8.32				
		Swimming endurance, trial 1					*Swimming endurance, trial 2*					*Maximal swimming endurance*				
1. ADCE	NA	NA	.165	.893	.143	−.201	NA	.326	.889	−.064	−.151	NA	.297	.730	.028	−.055
2. ADE	vs 1	3.04	.177	1.010		−.187	1.28	.328	.792		−.120	0.10	.296	.774		−.070
3. ACE	vs 1	6.29	.320		.226	.454	7.59	.494		.009	.497	3.26	.401		.128	.471
4. AE	vs 2	8.96	.361			.639	6.31	.494			.506	4.99	.426			.574
	vs 3	5.70					0.01					1.82				
		Body mass @ sprint speed trial 1					*Body mass @ sprint speed trial 2*					*Body mass @ maximal sprint speed*				
1. ADCE	NA	NA	.149	.875	.093	−.117	NA	.177	.820	.072	−.069	NA	.126	.826	.117	−.069
2. ADE	vs 1	1.68	.155	.961		−.116	1.26	.179	.889		−.068	2.86	.132	.926		−.058
3. ACE	vs 1	7.04	.276		.196	.528	7.10	.355		.151	.494	7.07	.277		.204	.519
4. AE	vs 2	10.45	.308			.692	9.81	.386			.614	10.65	.318			.682
	vs 3	5.09					3.97					6.44				
		Body mass @ endurance trial 1					*Body mass @ endurance trial 2*					*Body mass @ maximal endurance trial*				
1. ADCE	NA	NA	.325	.657	−.023	.041	NA	.387	.482	.013	.118	NA	.310	.570	.021	.099
2. ADE	vs 1	0.12	.324	.636		.040	0.04	.387	.493		.120	0.10	.310	.593		.097
3. ACE	vs 1	4.18	.468		.018	.514	2.32	.476		.043	.481	2.80	.424		.067	.509
4. AE	vs 2	4.11	.471			.529	2.63	.483			.517	3.42	.436			.564
	vs 3	0.05					0.34					0.73				

full model ($\chi^2 = 7.479$). The **CE** model, but not the **AE** or **DE** models, fit significantly better than did the model with only the specific environmental component. Narrow-sense heritability was estimable only for the **AE** model ($h^2 = 0.04$, Table 3).

Swimming Endurance.—Considering **ADCE** models first, dominance genetic variance (plus prenatal maternal effects, if present) comprised greater than 70% of total phenotypic variance for swimming endurance (Dohm 1994, table XII). Additive genetic variance explained a small, but statistically significant fraction of total phenotypic variance. Estimates of the specific environmental component (**E**) were always negative when the dominance component was also included in the model (Table 3). Similar to our findings for sprint speed, however, these negative estimates generally accounted for a relatively small fraction (< 20%) of the total variance. Negative estimates for the specific environmental variance can be obtained when the source population is highly inbred (R. G. Shaw, pers. comm., August 1994), but that is not the case for these animals (see Materials and Methods and Dohm [1994]). Negative estimates for **E** can also be obtained when the trait is highly variable and/or when the pedigree sample size is relatively small (cf. Searle et al. 1992, p. 61), both of which apply to our data.

Results of model-fitting indicated that additive and dominance genetic variances contributed significantly to total phenotypic variance for swimming endurance on both trial days and for maximal performance. Furthermore, models fitting only specific (random) environmental effects did not fit the data well, as compared with either the **CE**, **DE**, or **AE** two-factor models, thus confirming models of "family resemblance" for swimming endurance. Estimates of narrow-sense heritability for *maximal* endurance ranged from 0.30 to 0.43 depending on the variance components included in the model (Table 3).

Body Mass.—For body mass recorded on sprint speed and endurance trial days, variance component estimates and model fit statistics are provided in Dohm (1994, tables XIII and XIV). Body mass was modestly heritable (Table 3) and, as expected from earlier studies with Hsd:ICR mice (e.g., Riska et al. 1984), heritability increased slightly as the mice aged between speed and endurance trials. Estimates of h^2 were never negative for any measure of body mass. Moderate to high levels of dominance variance were also evident for all body mass measures (Table 3).

Phenotypic and Genetic Covariance and Correlations

Additive and nonadditive genetic, common environmental, and environmental covariances are reported in Dohm (1994, tables XVII, XVIII). All additive genetic covariance estimates between measures of swimming endurance and *trial day 1* sprint running speed were large and negative; most were statistically different from zero at $P < 0.1$. Across the four models (**ADCE**, **ADE**, **ACE**, **AE**), both signs and magnitudes of the additive genetic covariance estimates between speed and endurance tended to be very consistent. In general, additive genetic covariances between sprint running speed and swimming endurance were negative (26/36 models), but only those involving speed on *trial day 1* were statistically different from zero (at $P < 0.1$). No phenotypic correlation between measures of sprint running speed and endurance was significantly different from zero (e.g., speed *trial day 1* versus swimming *trial day 1*, $r_P = -0.028$; see also Fig. 3).

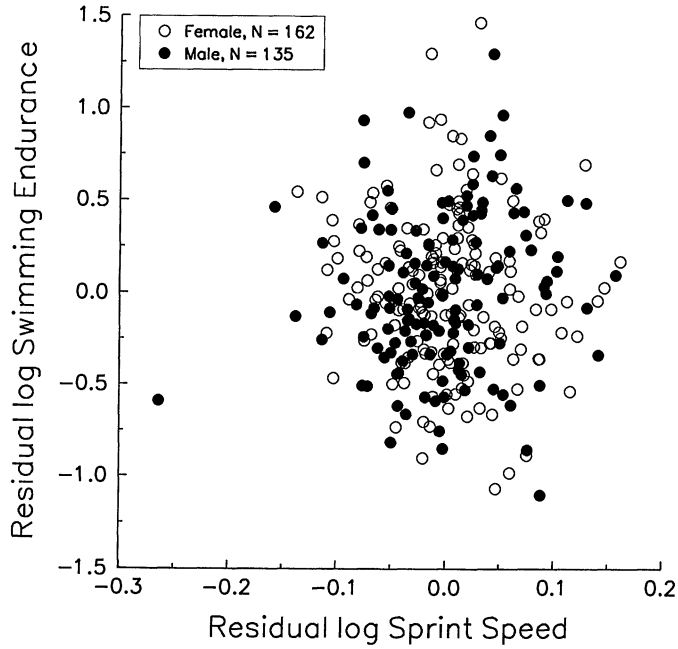

FIG. 3. Scatterplot depicting absence of phenotypic correlation between maximal swimming endurance residuals and maximal sprint running speed residuals ($r_P = 0.002$).

Phenotypic and additive genetic correlations between the consecutive trial days for swimming endurance ($r_P = 0.610$; from **ADCE** × **ADCE** model, $r_A = 1.161$) were all positive and large; however, no tests of significance were available for the genetic correlation because the matrix became singular when the additive genetic covariance path was constrained to zero. For sprint speed, no genetic correlation can be reported because the heritability estimates for sprint speed on *trial day 2* were negative (Table 3). When estimable, the additive genetic covariance between the two traits was very small and not statistically different from zero (e.g., under **ADE** × **AE** model, $COV_A = -0.96$, $\chi^2 = 0.073$, $P > 0.5$).

As expected, both phenotypic (see *Repeatability* above) and genetic correlations between the measures of body mass on consecutive days were positive and large (e.g., body mass at speed trials: $r_A = 0.948$; body mass at swimming trials: $r_A = 0.986$ [no test of significance was available because the matrix became singular when the path for additive genetic covariance was constrained to zero]). The phenotypic correlations between measures of body mass and the locomotor traits (Figs. 1–2), however, tended to be small and not statistically different from zero (body mass × sprint speed ranged from 0.002–0.050; body mass × swimming endurance ranged from 0.005–0.134). Only one of the correlations between body mass and a performance trait was significant at $P < 0.05$ ($r_P = 0.134$, between body mass and swimming endurance on *trial day 2*). All additive genetic covariance estimates between body mass and sprint running speed measures were small and positive; none were statistically dif-

ferent from zero at $P < 0.1$. Between body mass and swimming endurance, estimates of additive genetic covariance were both positive and negative and none was statistically different from zero. Similarly, the genetic correlations between body mass and the performance traits were not statistically significant (e.g., r_A for body mass × speed, *trial day 1* = 0.53, $\chi^2 = 1.253$; r_A for body mass × maximal swimming endurance = −0.23, $\chi^2 = 0.124$).

Discussion

A Genetic Trade-Off between Speed and Endurance

Our study apparently is the first to report evidence for negative additive genetic correlation for measures of locomotor abilities in mammals. Genetic correlations between performance traits (or between performance traits and body mass) apparently have not been estimated for either greyhounds or racehorses. (For reptiles, see Garland [1988, 1994a]; Scorci et al. [1995] and references therein).

We found evidence for large, negative additive genetic covariance (correlation) between measures of swimming endurance and sprint running speed measured on the first of two trial days, but not for any other measure of speed (see also Dohm [1994]). Only 8 of 36 estimable additive genetic covariances between speed and endurance were statistically different from zero (almost 4 would be expected to be significant by chance alone at $P = 0.10$); all of these covariances were negative and involved sprint speed on *trial day 1*. Because maximal sprint speeds (the fastest run from either day of the sprinting trials) were mostly *trial day 2* values (65% of individuals were faster on second day), it is not surprising that maximal sprint speed shows a pattern similar to speed on *trial day 2*. Nonetheless, the overall picture of additive genetic covariance between speed and endurance is clear; of the 36 possible estimates of additive genetic covariance, 26 were negative (Dohm 1994, table XVII). If additive genetic covariances between average performances for swimming endurance and sprint speed are also considered, 52 of 64 estimates were negative (Dohm 1994). (These values are not independent, of course.) Moreover, estimates of additive genetic covariance under the different models were very consistent. Mean (± SE) estimates of additive genetic covariance between speed and endurance under the different models were as follows: −19.6 ± 6.57 (**ADCE**); −22.0 ± 6.20 (**ADE**); −17.0 ± 5.23 (**ACE**); −21.5 ± 4.93 (**AE**). One-way analysis of variance indicated no significant differences among all four models ($F_{3,58} = 0.158$, $P = 0.924$) nor between the **ADCE** and **AE** models ($F_{1,29} = 0.054$, $P = 0.818$). Our results thus support the hypothesis of a trade-off between these contrasting locomotor abilities (see below).

Although we found evidence for large negative genetic covariance (and, therefore, genetic correlations) between some measures of sprint speed and endurance, phenotypic correlations were virtually zero (Fig. 3). The general explanation for this apparent paradox is that there need not be a relationship between genetic and phenotypic correlations. In this strain of mice, a trade-off between these two aspects of locomotor performance was manifested at the genetic and not the phenotypic level. Phenotypic correlations are sometimes found to be similar to genetic correlations, especially for large data sets (Cheverud 1988; cf. Willis et al. 1991). Not uncommonly, however, estimates of phenotypic and genetic correlations are strikingly dissimilar. Examples can be found for a variety of traits among a variety of taxa (e.g., Lynch et al. 1988; Garland et al. 1990; Sorci et al. 1995). Our results illustrate the potential pitfalls in extrapolating from the phenotypic to the genetic level, as has been noted by numerous authors (e.g., Falconer 1989; Willis et al. 1991). An alternative explanation is that the trade-off may not be manifested at the phenotypic level because of the different levels of phenotypic variation for the two traits (van Noordwijk and de Jong [1986]); swimming endurance was six to nine times more variable than was sprint speed (Results and Table 1). Previous studies have also found similar magnitudes of variation for running speed and endurance in heteromyid rodents (e.g., compare Djawdan 1992, Table 1 to Djawdan and Garland 1988, Table 1).

Larger Mice Did Not Have Greater Performance Capacities

The signs of all phenotypic correlations between endurance and body mass were positive; however, only one was significantly different from zero (body mass × *trial day 2* swimming endurance). Similarly, the signs of the genetic correlations between swimming endurance and body mass tended to be positive, but we could not reject the null hypothesis of zero correlation. For sprint speed and body mass, only half of the phenotypic correlations were positive and none was significantly different from zero. The signs of genetic correlations between measures of sprint speed and body mass were all positive, but none approached statistical significance. Thus, we found little evidence to suggest that selection on body mass alone would cause changes in locomotor abilities, nor that selection for enhanced locomotor performance would necessarily cause correlated changes in body size.

On the other hand, Falconer (1977, p. 20) has noted that mice selected for large size tend to be "less reactive" than smaller mice and body mass has been reported to be inversely related to activity in the open-field arena within some strains of mice (e.g., C3H, but not C57BL and BALB/c, Thiessen 1961). Similarly, DeFries and Hegmann (1970) also reported negligible genetic correlation between body mass and activity during open-field tests of mice (parents and offspring derived from crosses of C57BL and BALB/c strains). (Genetic coupling between body mass and other behaviors in house mice have also been reported [e.g., Lynch et al. 1988].) Interestingly, a trend of increasing body size has been noted for Japanese racehorses over the past two decades; body mass correlated positively with measures of racing performance (e.g., time of finish, earnings) (Tsunemoto et al. 1992). We have not found any comparable reports for greyhounds. As noted in the Introduction, interspecific comparisons generally suggest a positive correlation between endurance and body mass for other mammals (Djawdan 1992; see also Garland et al. 1988).

Speed Measured on Consecutive Days as Different Traits

The "same" trait measured at different times may also be considered as different traits. For example, body mass measured at different ages can be studied by fitting growth equa-

tions and then analyzing parameters of those equations (e.g., Riska et al. 1984). Alternatively, multiple measures of body mass can be treated as different characters, with separate heritabilities computed for each and genetic correlations computed between body masses measured at each different time (e.g., Rutledge et al. 1973).

In the present study, we measured both speed and endurance on two consecutive days. This was done for several reasons. First, we wanted to increase the probability of observing a maximal or near-maximal performance for each individual (Garland and Losos 1994). Second, we wished to estimate the repeatability of each trait, because repeatability sets an upper limit to heritability if contributions of maternal effects are negligible (e.g., Falconer 1989). If we could not measure performance repeatably, then it would be difficult to demonstrate either heritability or genetic correlation. Both speed ($r_P = 0.52$) and endurance ($r_P = 0.62$) turned out to be repeatable, as we have found in previous studies of house mice (Friedman et al. 1992; Dohm et al. 1994; Garland et al. 1995). As might be expected, the estimate of the genetic correlation between endurance on *trial days 1 and 2* exceeded +1, indicating that, in terms of additive genetic effects, the two trials measure the same trait.

In contrast, for sprint speed on *trial days 1 and 2*, the genetic covariance was small and not statistically different from zero. (We could not test the hypothesis that the additive genetic correlation between sprint speed for the two days was different from 1, because this correlation was not defined.) Moreover, statistically significant V_A estimates were obtained for sprint speed on *trial day 1*, but not for the other measures of sprint speed (Table 3). This unexpected result suggests that speed measured on two consecutive days may not index the same trait. First, for sprint speed on *trial day 2*, a hypothesis of no family resemblance was sufficient as the **E** model could not be rejected (Dohm 1994). Second, less than 20% of total phenotypic variance for sprint speed on *trial day 1* was accounted for by dominance, whereas for sprint speed on *trial day 2*, substantial variance (80%) was explained by dominance genetic variance (Table 3). One reason to attempt transformations of scale for quantitative genetic data is to minimize nonadditive effects (Falconer 1989). However, distributions for sprint speed on the two days were virtually identical (Dohm 1994), suggesting that scale differences are not likely to account for the different contributions of dominance variance (cf. discussion in Falconer 1989, p. 296).

Alternatively, the absence of genetic correlation between the two measures of sprint speed may be related to the number of families and individuals measured. Gill and Jensen (1968) found that the probability of obtaining negative variance component estimates (using nested ANOVA) and, therefore, heritability, may be high. For example, if the true heritability of sprint speed on *trial day 2* was 0.1, then the probability of obtaining negative heritability may be as much as 30% (using equations in Gill and Jensen [1968] and sample size of 5 dams per sire, 4 offspring per dam). Similar cautions have been made for the probability of obtaining nonpositive definite matrices when two or more traits are considered simultaneously (Cheverud 1988). Although we have not performed power calculations using the maximum likelihood methods for this data set, our REML estimates are expected to be comparable to ANOVA estimates because of the nearly balanced number of offspring measured per dam and sire.

Our findings of different heritability estimates for traits measured on two consecutive days are not unprecedented, however. Based on both phenotypic and genetic evidence, many researchers interpret activity measured during open-field tests on days 1 and 2 as different traits (e.g., Whimbey and Dennenberg 1967; Russell and Williams 1973). In a recent example with mice, the genetic correlation for activity in an open-field test was negative between the first and second days, but large and positive between day 2 and all subsequent trial days (van der Staay et al. 1990).

Even given that speed measured on *trial day 1* and *2* indexed different aspects of the phenotype, it is not obvious why genetic correlations with endurance should be negative for *trial day 1*, but zero or positive for *trial day 2*. This pattern is all the more surprising when one considers that speeds on *trial day 2* were slightly faster for most mice, and thus would seem to better represent physiological differences among individuals. We note, however, that the heritability for the average of the two maximal sprint speeds was also very low ($h^2 = -0.01$) (Dohm 1994). Moreover, all additive genetic covariances between swimming endurance and average sprint speed performance were negative (Dohm 1994), which supports our findings of a negative trade-off between sprint speed and swimming endurance. Regardless, our estimates of heritabilities and genetic correlations should themselves be viewed as hypotheses, subject to further testing by, for example, artificial selection experiments (cf. Boake 1994; Garland and Carter 1994).

Comparisons of Heritabilities with Values for Horses and Dogs

For this population of random-bred laboratory house mice, Table 3 indicates that measures of residual forced sprint running speed (e.g., for *trial day 1*, $h^2 < 0.2$) were less heritable than were measures of residual log swimming endurance. Overall, our h^2 estimates for the ICR mice are similar to published estimates of various indices of racing ability in the American Quarter horse (weighted mean $h^2 = 0.24$, for distances between 201 and 796 m: Buttram et al. 1988) and thoroughbreds ($h^2 = 0.36$, for distances of 2400 and 2800 m: Gaffney and Cunningham 1988). For Irish greyhounds, Ryan (1975) reported h^2 estimates of about 0.23 over distances of about 400 m.

Comparisons of our genetic results with those for racing dogs and horses are problematic for several reasons. First, breeding programs employed by owners of racing horses are generally undertaken for reasons other than estimation of quantitative genetic parameters. Thus, heritability estimates reported for horses are probably inflated by genotype-environment correlations, assortative mating, and because racehorses represent a selected, rather than random-breeding population (Langolis 1980; cf. Falconer 1989). Second, speeds analyzed for horses constitute average speeds over the entire length of a race, not maximal speeds attained during any segment. In the present study, we analyzed the fastest 1.0 m segments along a timed length of 5.5 m. Third, most studies

of horses report that motivational and/or competitive differences among races have large effects on racing performance (Buttram et al. 1988; Oki et al. 1994). In contrast, all of our mice were tested singly and on the same track. Many of the foregoing concerns can also be raised for racing greyhounds (Ryan 1975). Finally, estimates of genetic correlations for locomotor abilities have not been published for dogs or horses.

Evolutionary Significance of a Trade-Off between Speed and Stamina in Hsd:ICR Mice

In nature, we expect that both sprint speed and endurance should generally be subject to positive directional selection. All else being equal, individuals that are faster and/or capable of longer periods of sustained activity should tend to have higher Darwinian fitness (e.g., Jayne and Bennett 1990; see also Bennett and Huey 1990; Garland et al. 1990; Garland and Losos 1994). In nature, therefore, a negative genetic correlation between speed and endurance would qualify as an evolutionary constraint on adaptive microevolution, because it would be opposite in sign to the (presumed) prevailing action of natural selection. Studying quantitative genetics of wild organisms can be problematic (Boake 1994), however, so we have used a random-bred strain of laboratory house mice as a model system.

An immediate question is whether domestic and/or laboratory organisms are appropriate for making inferences about evolution in wild organisms. Wild and domestic/laboratory strains of the same species typically differ for some traits but not for others (Boice 1973; Dohm et al. 1994; Richardson et al. 1994; Garland et al. 1995, and references therein), so the answer will likely depend on the trait in question. In previous studies, we found that wild house mice from a commensal Wisconsin population, bred and raised in our laboratory, sprinted about twice as fast as did Hsd:ICR mice (Dohm et al. 1994; Garland et al. 1995). Behavioral differences between wild and lab mice were implicated as the predominate cause of the difference in sprint speed, because we did not find parallel differences in a series of suborganismal traits thought to affect speed (e.g., gastrocnemius muscle mass, muscle fiber composition: Dohm et al. 1994; Garland et al. 1995). The same wild and lab mice had similar stamina, although the difference was statistically significant when body mass was used as a covariate (Dohm et al. 1994). In the present study, with a much larger sample size, we did not find strong evidence for a relationship between endurance and body mass, so we favor the interpretation that these wild and lab mice do not differ in endurance. (Levels of variation for sprint speed and swimming endurance were also similar between these wild and lab mice [see Figures in Dohm et al. 1994; Garland et al. 1995].) If the reduction in sprint speed of Hsd:ICR lab mice as compared with a wild population does indeed reflect genetic changes in behavior as opposed to morphology/physiology, and if the negative genetic correlation between speed and endurance is based on morphology/physiology as opposed to behavior, then these lab mice may indeed serve as a reasonable model for making inferences about the evolution of locomotor abilities in wild house mice.

On the other hand, we note that the alleles affecting sprint speed and/or endurance may differ between laboratory and wild house mouse populations. The laboratory strains ancestral to Hsd:ICR mice were started during the latter part of the 19th century (see Dohm 1994, ch. 1), so hundreds of generations of captive breeding have occurred, ample time for new mutations to accumulate that affect sprint speed and/or endurance. Genetic differences between wild and laboratory populations may also have been present before or arisen during the divergence of domestic and wild populations. Thus, whether our finding of a negative genetic correlation and, therefore, a trade-off between speed and stamina is a general feature of house mice, of rodents, or perhaps even of mammals, can only be addressed by evaluating the genetic architecture for these traits in additional strains of house mice (including wild populations), other species of rodents, and so forth (cf. Lynch 1992; Lynch 1994).

In addition to the present results, several other lines of evidence suggest that a genetically based, physiological trade-off between speed and stamina may be a general feature of mammalian exercise physiology. Among species of mammals, cheetahs are noted as champion sprinters (Garland 1983), but apparently possess only a pedestrian capacity for sustained aerobic performance (Taylor et al. 1974). Among individual humans, good sprinters are usually not good marathon runners, and physiological differences between sprinters and distance runners are cited as one explanation. Highly trained sprinters tend to have higher percentages of fast-twitch (i.e., rapid contracting) muscle fibers, whereas marathon runners tend to have high percentages of slow oxidative fibers (Komi 1984; Astrand and Rodahl 1986). In horses, the composition of muscle fiber types correlates with reported ratings (excellent versus poor) for endurance events (Rivero et al. 1993). Existing data for house mice, however, have not shown a correlation between sprint speed and fiber types (Garland et al. 1995). Muscle fibers are relatively plastic, dynamic structures (e.g., the isoform expression can change in response to different functional demands [Pette and Staron 1993]). Nevertheless, the distribution of fiber types in mammalian muscle appears to be partly genetically determined (e.g., humans: Komi [1984]; mice: Hooper [1978]; Nimmo et al. [1985]). Moreover, breeds of horses and dogs that have been selected for racing performance tend to differ in muscle fiber type composition from nonracing breeds (Snow and Harris 1985).

During the domestication process, we believe that laboratory strains of mice were directly or indirectly selected for reduced escape behavior (references in Dohm 1994, ch. 1, 2). Sprint speed, as measured with our protocol, may have diminished as a correlated response to selection on escape behavior. Alternatively, lab mice may simply have experienced relaxed selection on sprint speed. However, quantitative rate tests (e.g., Turelli et al. 1988) suggest that the divergence between wild and ICR mice observed for sprint speed (Dohm et al. 1994; Garland et al. 1995) is too great to be attributed to mutation and random genetic drift (unpubl. results).

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