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A COMPARISON OF GENETIC AND PHENOTYPIC CORRELATIONS

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Abstract.—Genetic variances and correlations lie at the center of quantitative evolutionary theory. They are often difficult to estimate, however, due to the large samples of related individuals that are required. I investigated the relationship of genetic- and phenotypic-correlation magnitudes and patterns in 41 pairs of matrices drawn from the literature in order to determine their degree of similarity and whether phenotypic parameters could be used in place of their genetic counterparts in situations where genetic variances and correlations cannot be precisely estimated. The analysis indicates that squared genetic correlations were on average much higher than squared phenotypic correlations and that genetic and phenotypic correlations had only broadly similar patterns. These results could be due either to biological causes or to imprecision of genetic-correlation estimates due to sampling error. When only those studies based on the largest sample sizes (effective sample size of 40 or more) were included, squared genetic-correlation estimates were only slightly greater than their phenotypic counterparts and the patterns of correlation were strikingly similar. Thus, much of the dissimilarity between phenotypic- and genetic-correlation estimates seems to be due to imprecise estimates of genetic correlations. Phenotypic correlations are likely to be fair estimates of their genetic counterparts in many situations. These further results also indicate that genetic and environmental causes of phenotypic variation tend to act on growth and development in a similar manner.

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The level and pattern of correlation, or covariance, between traits has repeatedly been investigated by evolutionary biologists and is of special concern for those interested in the basis and role of morphological integration in evolution (Huxley, 1932; Olson and Miller, 1958; Cheverud, 1982, 1988*a*, 1988*b*; Reyment et al., 1984). It has become apparent to evolutionary biologists over the past several years that, due to pleiotropy and linkage disequilibrium, knowledge of genetic correlations (and covariances) is crucial for an understanding of coordinate evolution through correlated responses to selection (Lande, 1979; Falconer, 1981; Cheverud, 1982, 1984). Genetic correlations and variances are difficult to measure even in the best circumstances, however, and are utterly impossible to measure in many instances, such as in paleontology or in neontological studies of rare, difficult-to-breed species. If genetic- and phenotypic-correlation levels and patterns are typically similar to one another and if genetic and phenotypic variances are often proportional to one another (or there is little variation in heritability among traits), perhaps evolutionary inferences can be made from phenotypic correlations and variances alone, thus providing a basis for some level of evolutionary inference, even in the absence of genetic data. In this report, I compare the level and pattern of genetic and phenotypic correlation in order to determine the practical importance of obtaining genetic-correlation estimates for the purpose of making inferences about the coordinated evolution of traits. If phenotypic values can be substituted for genetic ones in some instances, the growing body of evolutionary quantitative-genetic theory can be applied more widely than it is today.

There are two reasons one might expect genetic and phenotypic correlations to be similar. First, under the usual additive model, phenotypic correlations are the sum of genetic and environmental components, the causal correlations being weighted by the relative importance of heritable and nonheritable effects, respectively:

$$r_P = h_X h_Y r_G + e_X e_Y r_E \qquad (1)$$

where h represents the square root of the heritability, e represents the square root of the proportion of phenotypic variance due to environmental factors, and r is a correlation with subscripts P, G, and E representing phenotypic, genetic, and environmental correlations, respectively, for traits X and Y (Falconer, 1981). Thus, if both her-

itabilities approach one, genetic and phenotypic correlations must be similar by definition, and phenotypic correlations may be substituted for their genetic counterparts in evolutionary analysis. Unfortunately, heritabilities are rarely high.

If genetically and environmentally based phenotypic variations are produced by similar disruptions of developmental pathways, genetic and environmental correlations should be similar (Cheverud, 1984), given that the pattern of developmental relationships among traits structures the pattern of correlation. Since phenotypic correlations are the weighted sums of genetic and environmental components, genetic and phenotypic correlations in this situation will also be similar, regardless of the level of heritability. This hypothesis must be tested using similarity of phenotypic- and genetic-correlation estimates, rather than estimated genetic-environmental similarity because environmental correlations are routinely computed as differences between phenotypic- and genetic-correlation estimates from the relationship defined in Equation (1). Environmental correlations between traits are typically not estimated directly, due to the difficulty in identifying and directly measuring all the important environmental factors affecting trait variation and covariation. Even so, particular sources of environmental variation, such as maternal effects, are sometimes directly estimated (Falconer, 1981).

Differences between genetic- and phenotypic-correlation estimates could arise due to disjunction between patterns of environmental and genetic effects on the developing phenotype and/or due to random sampling error present in estimates of true population values. Actual population genetic correlations are particularly difficult to estimate accurately, because the calculated estimates reported in the literature are really only functions of ordinary statistical estimates of true population values rather than direct statistical estimates. For example, in a quantitative-genetic analysis using a half-sib design, each set of four paternal half-sibs is a sample drawn from all of the father's potential offspring and thus is only an estimate of his additive genetic (breeding) value rather than a measurement of it. The fathers, in turn, are also sampled from the population as a whole (see Arnold [1981] for a brief discussion). There is a two-level sampling problem, because number of offspring per family and number of families are both important in determining the sampling variation of genetic estimates (see Falconer [1981] for optimal balance between family size and number of families). For this reason, quantitative-genetic studies require fairly large sample sizes relative to phenotypic studies, even with standard statistical designs (Klein, 1974). It has even been suggested (Williams, 1962*a*, 1962*b*) that, in particular instances, estimates of genetic variance and covariance not be used in constructing selection indices for agricultural breeding programs because errors in the estimates can be so large that their inclusion may lead to less actual realized economic gain than would have been achieved from using the unmodified economic weights alone. In this situation, poor genetic estimates might actually be misleading.

Gill and Jensen (1968) investigated the probability of obtaining negative heritability estimates (true population heritabilities cannot be negative by definition) as a function of sample size and found this probability to be considerable. Hill and Thompson (1978) determined the probability of obtaining negative semidefinite genetic-covariance or genetic-correlation matrices (matrices with ordinary or partial correlations greater than one or less than negative one and thus containing linear combinations with negative heritability), given certain sample sizes, numbers of traits, and levels of heritability, and found that the probability can approach 100% even for fairly large samples. The probability of obtaining such estimates, corresponding to theoretically impossible sets of genetic covariances, increases dramatically as the number of traits included in the analysis increases but decreases with increasing sample size and heritability of the characters. Indeed, positive definite genetic-correlation matrices are found only rarely in the literature (see Cheverud and Leamy, 1985).

While estimates of individual genetic correlations may not be biased, Hill and Thompson's work (1978; also see Hayes and Hill [1981]) indicates that the overall level

of correlation among sets of traits (as represented by the inverse of the correlation matrix's determinant, its level of integration [Cheverud et al., 1983], the variance of its eigenvalues, or the average squared correlation) will tend towards larger values. Thus, the estimated level of overall genetic covariance or correlation among a set of traits is likely to be extreme, relative to the true population value, due to sampling error when the number of traits is high, the sample size is low, and the heritabilities are low. Likewise, the standard errors considered simultaneously for many related genetic-correlation coefficients are likely to be very high, making the signal-to-noise ratio unfavorable and making it difficult to detect actual population genetic-correlation patterns. The effects of such errors in estimation are much less extreme for phenotypic correlations.

A lack of correlation between genetic- and phenotypic-correlation estimates could be due to lack of precision in estimating genetic correlations. Also, genetic-correlation magnitudes may be extreme, relative to their phenotypic counterparts, due to small sample sizes and/or low heritabilities.

From Equation (1) and the discussion above, it is apparent that the level of heritability potentially affects the similarity of phenotypic- and genetic-correlation levels and patterns in two ways. Higher heritabilities increase the proportional contribution of genetic to phenotypic correlation [see Equation (1)] and thus will result in greater similarity of population phenotypic and genetic correlations. Higher heritabilities will also increase the overall accuracy of genetic-correlation estimates (Klein, 1974; Hill and Thompson, 1978), resulting in greater similarity of phenotypic- and genetic-correlation estimates when population values are similar.

When heritabilities are high, we expect little difference in the overall level and pattern of genetic and phenotypic correlations. When heritabilities are moderate to low and the magnitude and pattern of genetic and phenotypic correlations are similar, it is likely that the effects of genetic and environmental factors are channeled through a common, relatively invariant, developmental system. When heritabilities are moderate to low and the overall level of genetic correlation is greater than phenotypic correlation and genetic correlations follow a different pattern from their phenotypic counterparts, either the genetic parameters are not sufficiently well estimated (see above), or genetically and environmentally based phenotypic variations do not share the same developmental basis.

Previous results on the similarity of genetic and phenotypic correlations have been mixed. It has typically been found that genetic correlations exceed their phenotypic counterparts, although no systematic survey has been attempted since Searle (1961). Some empirical studies have indicated similarity between genetic- and phenotypic-correlation patterns (Bailey, 1956; Hegmann and DeFries, 1970; Leamy, 1977; Atchley et al., 1981; Arnold, 1981), while others have stressed the observed differences (Atchley and Rutledge, 1980; Cheverud, 1982).

Materials and Methods

In order to test for similarities between genetic and phenotypic correlations, a total of 41 pairs of phenotypic- and genetic-correlation matrices from 23 different studies were collected from the agricultural genetic and evolutionary literature (see Appendix). Animals included range from human to amphipod, while traits analyzed range from morphological to cognitive. Only studies including five or more traits were included in the compilation. This sample of 41 matrix pairs is not exhaustive, nor are the pairs independent of one another. Several well studied populations, such as the rodent populations originating in the University of Wisconsin's animal laboratory (Numbers 1–7, 9–10, 12–19, 27, and 30–31 in the Appendix) are represented multiple times. This lack of independence among the matrix pairs complicates statistical analysis, so that the statistical probabilities reported here are likely to be too low. In these 41 studies, genetic variances and correlations were estimated in a variety of ways using various kinds of relatives and a variety of experimental, and nonexperimental, statistical designs. A compensating advantage of these studies is that all authors published their genetic- and phenotypic-correlation matrices.

The overall magnitude of correlation in each matrix was measured by the average R^2 value; more extreme values are always relatively higher on this scale, as opposed to a strict correlation scale. In order to calculate the average R^2 value, each individual off-diagonal correlation value was squared, and the squared correlations were summed and then divided by the number of off-diagonal elements in the matrix. Squared correlations were used because the intention here is to compare magnitudes of correlation, rather than their patterns. These overall magnitudes were compared by subtracting phenotypic from genetic average R^2's to generate a variable labelled "difference."

The patterns of genetic and phenotypic correlation are compared using a matrix correlation, an element-wise Pearson product-moment correlation between all comparable, nonredundant genetic- and phenotypic-matrix elements. A high positive matrix correlation indicates a strong similarity of correlation pattern. The statistical significance of this matrix correlation is tested with a quadratic-assignment procedure (Dow and Cheverud, 1985; Cheverud and Leamy, 1985; Hubert, 1987; Dow et al., 1987*a*, 1987*b*), sometimes referred to as a Mantel test (Mantel, 1967; Smouse et al., 1986). In quadratic assignment, the observed matrix correlation is compared to an empirically derived distribution based on a null hypothesis of no association between genetic and phenotypic matrices. The distribution of matrix correlations under the null hypothesis is generated iteratively by repeatedly (200 repetitions per comparison) correlating the phenotypic matrix with the genetic matrix, after first having randomized the rows (and corresponding columns) of the genetic matrix.

In order to examine the possibility that differences between genetic- and phenotypic-correlation magnitudes and patterns are due to poorly resolved genetic-correlation estimates, the geometric mean heritability (H) and number of families (N) used in deriving genetic estimates were recorded for each pair of matrices. The reliability of genetic-correlation estimates depends on both of these factors in combination (Turner and Young, 1969). Thus, a measure referred to as effective sample size (N_{es}) was calculated as the product of number of families and heritability and taken as a very rough estimate of the true sample size used in deriving genetic-correlation estimates. The varied designs and estimation techniques make this measure one of the few that are at least coarsely comparable across these diverse studies. Spearman rank-order correlations and Mann-Whitney U tests are used to evaluate the relationship between the reliability of genetic-correlation estimates and differences between genetic- and phenotypic-correlation matrices.

Results

Over the entire set of studies, heritabilities tend to be moderate (see Table 1; simple average $h^2 = 0.35$ [SD = 0.15]) with only six values greater than 0.50. Thus, Equation (1) does not inherently imply close similarity of genetic- and phenotypic-correlation estimates in this sample. The average R^2 values for the set of genetic- and phenotypic-correlation matrices are also presented in Table 1. The average squared genetic correlation over the whole sample is 0.49, while the average squared phenotypic correlation is 0.29, yielding an average difference of 0.20. Phenotypic squared correlations exceed genetic ones in only four cases, and three of these four involve trivial differences. Clearly, squared genetic-correlation estimates are more extreme than their phenotypic counterparts. This may represent a real difference in true population values for the average squared correlations or the effects of small effective sample sizes for genetic estimation.

The difference between average genetic and phenotypic squared correlations is significantly negatively correlated with heritability and effective sample size (see Table 2), indicating that the greater magnitude of squared genetic-correlation estimates is due to lack of precision caused by small samples, although it is also possible that greater similarity of correlation magnitudes with higher heritability is due in small part to the definitional relationship given in Equation (1). These negative correlations are not large, but given the imperfect manner in which the variables used relate to the precision of genetic-correlation estimates, they appear to be considerable. The difference in cor-

TABLE 1. Comparison of pairs of genetic- and phenotypic-correlation matrices. The sources are identified by number in the Appendix. H is the geometric mean heritability for the trait set, R^2_P and R^2_G are the average squared phenotypic and genetic correlations, respectively, "Diff" is the difference between R^2 values, "Corr" is the matrix correlation between phenotypic- and genetic-correlation matrices, N is the number of families used in the study, and N_{es} is the effective sample size (HN).

Source	H	R^2_P	R^2_G	Diff	Corr	N	N_{es}
1	0.32	0.59	0.61	0.02	0.50*	108	34
2	0.27	0.29	0.65	0.36	0.66*	108	29
3	0.26	0.38	0.67	0.29	0.76*	108	28
4	0.23	0.20	0.97	0.77	0.37	108	24
5	0.30	0.67	0.69	0.02	0.71*	108	32
6	0.42	0.42	0.61	0.19	0.95*	92	38
7	0.31	0.44	0.61	0.17	0.56*	92	28
8	0.13	0.55	0.53	−0.02	−0.14	22	2
9	0.28	0.58	0.55	−0.03	0.97*	345	96
10	0.36	0.59	0.70	0.11	0.95*	345	124
11	0.41	0.44	0.05	−0.39	0.80*	30	12
12	0.24	0.50	0.84	0.34	0.27	108	25
13	0.25	0.61	0.90	0.29	0.56*	108	27
14	0.26	0.53	0.87	0.34	0.08	108	28
15	0.25	0.32	1.17	0.85	−0.25	48	12
16	0.34	0.27	0.37	0.10	0.82*	108	36
17	0.33	0.31	0.64	0.33	0.46	108	35
18	0.27	0.30	0.63	0.33	0.03	60	16
19	0.14	0.27	0.96	0.69	−0.54	60	8
20	0.83	0.34	0.37	0.03	0.62*	27	22
21	0.70	0.08	0.16	0.08	0.50*	31	21
22	0.48	0.21	0.35	0.14	0.72*	22	10
23	0.34	0.19	0.20	0.01	0.74*	21	7
24	0.36	0.23	0.45	0.22	0.51*	18	6
25	0.18	0.21	1.00	0.79	0.37*	12	2
26	0.57	0.11	0.16	0.05	0.81*	300	171
27	0.60	0.31	0.48	0.17	0.77*	92	55
28	0.52	0.02	0.61	0.59	0.26*	55	28
29	0.43	0.16	0.14	−0.02	0.75*	200	86
30	0.40	0.18	0.21	0.03	0.71*	92	36
31	0.45	0.15	0.22	0.07	0.82*	92	41
32	0.55	0.24	0.42	0.18	0.71*	124	68
33	0.46	0.06	0.13	0.07	0.86*	200	92
34	0.39	0.05	0.12	0.07	0.85*	250	97
35	0.26	0.06	0.27	0.21	0.72*	66	17
36	0.37	0.22	0.37	0.15	0.50*	63	23
37	0.32	0.11	0.13	0.02	0.96*	565	180
38	0.25	0.22	0.39	0.17	0.65*	150	37
39	0.41	0.38	0.40	0.02	0.43	1,300	533
40	0.13	0.03	0.11	0.08	0.70*	19	2
41	0.18	0.06	0.27	0.21	0.81*	10	2

* Correlation significantly greater than zero at the 0.05 level, as determined by the quadratic assignment procedure.

relation level is plotted against effective sample size in Figure 1. It is especially notable that the difference between estimates ranges widely when effective size is less than 40. This is not surprising, given that each study includes at least five traits.

TABLE 2. Spearman rank-order correlations between factors affecting the reliability of genetic-correlation estimates (average heritability [H], number of families [N], and effective sample size [N_{es}]) and differences in magnitude (Diff) and pattern (Corr) between genetic- and phenotypic-correlation matrices.

	Diff	Corr
H	−0.39*	0.39*
N	−0.22	0.36*
N_{es}	−0.35*	0.52*

* Correlation significantly different from zero at the 0.05 level.

If the studies are arbitrarily divided in two, so that those with effective sizes less than and greater than 40 are separated ($N = 30$ and $N = 11$, respectively), there is a highly significant disparity ($P < 0.01$, according to Mann-Whitney U test) in squared-correlation difference between the low and high effective-sample-size sets (low set: average difference = 0.25; high set: average difference = 0.06). There is also a slight but statistically significant difference in heritability between sets, with the high effective-sample-size group showing a slightly higher average heritability ($h^2 = 0.44$) than the low effective-sample-size group ($h^2 = 0.33$). However, even given the part-whole relationship between genetic and phenotypic correlations, this small difference in heritability is not sufficient to explain the relatively large difference in squared-correlation magnitude between the two groups of studies. With relatively large samples, the average difference between genetic and phenotypic squared correlations is only about 0.06. Therefore, it is likely that, for actual population values, genetic correlations may be only slightly larger than their phenotypic counterparts.

The patterns of genetic- and phenotypic-correlation estimates seem broadly similar, with an average matrix correlation across the studies of 0.57 (see Table 1). As determined by quadratic-assignment procedures, 78% of the matrix correlations are statistically significantly different from zero at the 5% level. Higher matrix correlations are significantly associated with higher heritabilities, sample sizes, and effective sample sizes (see Table 2 and Figure 2), as expected from Equation (1) and considerations of estimate precision. Better estimated genetic-corre-

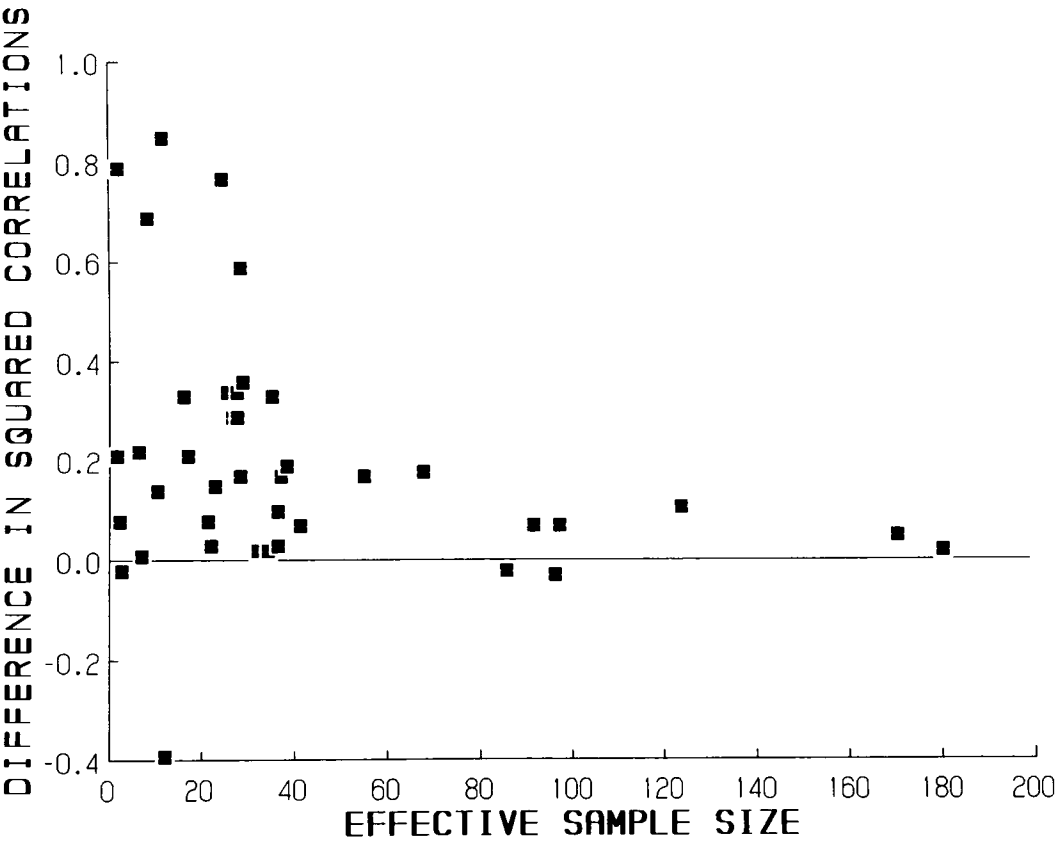

FIG. 1. Difference between average genetic and phenotypic coefficients of determination (R^2) compared to the effective sample size for 41 pairs of genetic- and phenotypic-correlation matrices.

lation matrices are more similar in pattern to their phenotypic counterparts than are poorly estimated matrices. Again dividing the sample into those studies with effective sizes greater than and less than 40, there is a highly significant difference ($P < 0.001$) in the level of matrix correlation (low-set matrix correlation = 0.48; high-set matrix correlation = 0.81) between the groups. The best estimated genetic-correlation matrices are quite similar to their phenotypic counterparts, sharing about 64% of the variation in correlation values. It seems likely that actual population genetic- and phenotypic-correlation matrices are also quite similar, although not necessarily identical.

The lack of independence of matrix pairs and the potential lack of comparability of the measured family-size variable among various designs, calls into question the precise probabilities reported above. However, all of the trends reported concerning the similarity of genetic and phenotypic correlations were also evident within three sets (Numbers 1–5, 12–19, and 21–25 in the Appendix) of related matrix pairs. Within each of these sets, a single population (although sample sizes were not the same for each genetic matrix) and common design were used to obtain genetic estimates.

DISCUSSION

The overall level of integration evident in estimated genetic-correlation matrices tends to be higher than in the paired phenotypic matrices, and genetic correlations show a moderate degree of similarity in pattern to their phenotypic counterparts. The effective sample size is significantly correlated with the differences between matrix pairs in both pattern and magnitude, however, indicating that some of the inconsis-

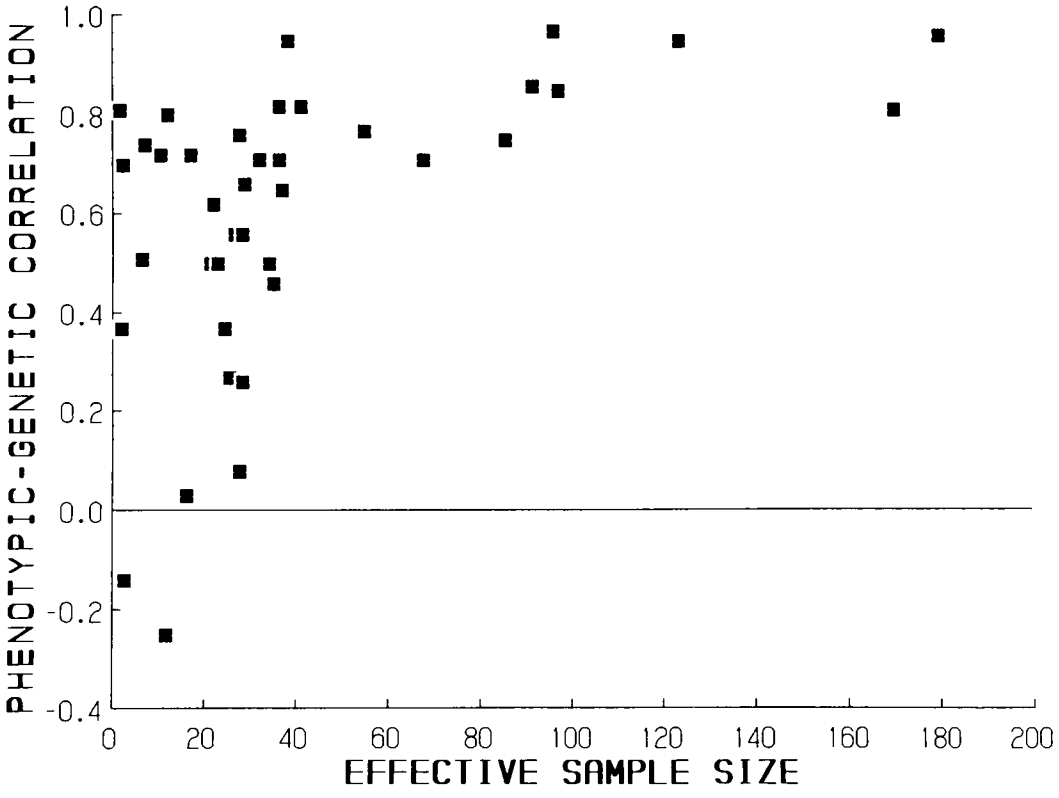

FIG. 2. Matrix correlation between paired genetic- and phenotypic-correlation matrices compared to the effective sample size.

tency between matrices is due to inaccurate estimates of genetic-correlation parameters. It seems likely that actual population values for genetic and phenotypic correlations are quite similar. The level of similarity in actual population values may be grossly determined from the studies that provide the most accurate genetic estimates. For these studies, genetic-correlation estimates slightly exceed their phenotypic counterparts (R^2 difference = 0.06) and show a similar pattern of correlation (matrix correlation = 0.81).

The broad similarity of genetic- and phenotypic-correlation matrices indicates that genetic and environmental effects on development typically produce similar patterns of phenotypic variation. This is probably the physiological basis for common phenocopies of genetic mutants and points out the importance of investigating environmentally generated teratologies for studies of development and evolution (Waddington, 1961). Most environmentally caused phenotypic variants should have genetic counterparts and vice versa.

The generality of these results depends on the character of the studies included in the analysis. Most matrices contained traits of a single type, morphometric, reproductive, or behavioral, with a predominance of morphometric studies in the sample. It is possible that concentration on other kinds of traits, which are not typically subjected to stabilizing selection, or on matrices containing mixtures of morphological, behavioral, and life-history traits, might produce different results. However, in these instances, the number of traits per study is often low (less than five) making it difficult to define a correlation pattern.

The results of this study also point out the importance of using large sample sizes in quantitative-genetic studies, especially if multiple characters are being considered. It seems that an effective sample size of at least

40 should be used. Crudely, for a set of characters with a geometric mean heritability of 0.33, at least 120 families seem to be indicated. In laboratory experiments with standard lab-bred species, this can be accomplished by using more space and money. Those working with other species do not always have the option of very large samples, however, due to difficulties in breeding and raising the organisms in the laboratory or due to the limited population size and amount of information available for feral populations. In these cases, new techniques, such as the use of symmetric squared differences (Grimes and Harvey, 1980; Bruckner and Slanger, 1986*a*, 1986*b*) or restricted maximum-likelihood estimation techniques (Shaw, 1987), designed to make the most of the genetic relatedness available in a population will prove helpful, although larger samples will always be desirable. Matrix manipulations, such as matrix "bending" (Hayes and Hill, 1981), may also be useful if they can generally increase the accuracy of genetic-correlation estimates. Further theoretical statistical work in both of these areas will help improve the utility of quantitative genetics for evolutionary studies.

Given the general similarity of genetic- and phenotypic-correlation matrices, it seems that phenotypic correlations may be substituted for genetic ones when genetic correlations are unavailable or not precisely estimated. While substitution of phenotypic for genetic correlations will certainly lead to errors in evolutionary inference in specific instances, the use of phenotypic correlations may be justified as being the best available estimates of genetic correlations in some cases. They may even be more precise than genetic-correlation estimates derived from small samples.

When evolutionary inferences are desired, one could substitute phenotypic values for the genetic ones identified in the following equation from Lande (1979),

$$\mathbf{R} = \mathbf{G}\beta \qquad (2)$$

where $\mathbf{R}$ is a vector of changes in means, $\mathbf{G}$ is the genetic-variance/covariance matrix, and β is the selection gradient defining direct selection on traits. Such a substitution will allow retrospective studies of differentiation by natural selection and prediction of response to hypothetical or imposed selection regimes with phenotypic data. Phenotypic- or genetic-variance/covariance matrices are related to their respective correlation matrices by the following transformation,

$$\mathbf{C} = \mathbf{V}^{-1}\mathbf{S}\mathbf{V}^{-1} \qquad (3)$$

where $\mathbf{C}$ is the correlation matrix, $\mathbf{S}$ is the variance/covariance matrix, and $\mathbf{V}^{-1}$ is the inverse of a diagonal matrix of standard deviations. The results presented here indicate that, when patterns of genetic and phenotypic correlation are fairly similar and genetic and phenotypic variances are proportional ($\mathbf{V}_G\mathbf{V}_G' = k\mathbf{V}_P\mathbf{V}_P'$, where k is the heritability and there is little or no variation in heritability among traits), the phenotypic-variance/covariance matrix may be substituted for the genetic-variance/covariance matrix after a proportional reduction based on the level of heritability. If more information on variation in the level of heritability among traits is available (heritabilities are more precisely estimated than genetic correlations), the constant k may be replaced by a diagonal matrix, $\mathbf{K}$, with the individual trait heritabilities along the diagonal, when reconstructing the genetic-variance/covariance matrix from phenotypic values.

For example, morphometric character sets tend to have average heritabilities of about 0.30–0.40 (see Table 1) and rarely show statistically significant differences among traits. Thus, the genetic-variance/covariance matrix may be roughly estimated by $0.35\mathbf{P}$ where 0.35 represents a typical level of heritability for morphometric traits and $\mathbf{P}$ is the phenotypic-variance/covariance matrix. Life-history traits tend to have lower heritabilities than morphometric traits (Falconer, 1981; Gustaffson, 1986), and thus a lower level of heritability should be used in estimating genetic variances and covariances given phenotypic life-history statistics. The very rough estimates proposed here are bound to introduce error relative to full quantitative-genetic analysis but may actually reduce error in evolutionary inferences drawn from purely phenotypic data. Also, while the overall magnitude and pattern of genetic and phenotypic correlation

represented in a matrix may be similar, it is quite possible that individual pairs of values could be quite different from one another. It is impossible, based on phenotypic data alone, to determine which correlations within a phenotypic matrix are good and which ones are bad estimates of the corresponding genetic correlation.

Several points can be made concerning the results of this analysis. First, while genetic correlations play a central role in theories of phenotypic evolution, they are difficult to estimate accurately. More theoretical work needs to be done on the statistical analysis of quantitative-genetic data from natural populations in order to improve the precision of genetic-variance and genetic-correlation estimates. Phenotypic correlations between characters may be estimated with much greater precision and are also much simpler to obtain than are their genetic counterparts. When genetic correlations are well estimated, they tend not to be very different in either magnitude or pattern from their phenotypic counterparts. Thus, when reliable genetic estimates are unavailable, phenotypic correlations and scaled variances may be substituted for their genetic counterparts in evolutionary models of phenotypic evolution. From the analysis presented here, this would seem to be an acceptable solution to serious problems involved in obtaining genetic estimates in many instances. While the substitution of phenotypic for genetic parameters in evolutionary studies will undoubtedly introduce error and can only lead to provisional solutions, relative to situations where precise genetic-correlation estimates are available, substitution will increase the rigor of evolutionary inferences drawn from phenotypic data compared to current ad hoc methods. These results do not indicate that quantitative-genetic studies are unnecessary but, rather, that quantitative evolutionary theory can be cautiously applied even when one is so unfortunate as to only have phenotypic data available.

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Appendix

The following is a list of sources for paired phenotypic- and genetic-correlation matrices in the present study. For each reference, type of organism, type of trait, and number of traits are also given.

1) Leamy and Cheverud, 1984; mouse; age-specific weights; 6 traits
2) Leamy and Cheverud, 1984; mouse; age-specific head lengths; 6 traits
3) Leamy and Cheverud, 1984; mouse; age-specific trunk lengths; 6 traits
4) Leamy and Cheverud, 1984; mouse; age-specific trunk circumferences; 6 traits
5) Leamy and Cheverud, 1984; mouse; age-specific tail lengths; 6 traits
6) Cheverud et al., 1983; rat; age-specific weights; 6 traits
7) Cheverud et al., 1983; rat; age-specific tail lengths; 6 traits
8) Trail et al., 1971; cattle; age-specific weights; 8 traits
9) Riska et al., 1984; male mouse; age-specific weights; 9 traits
10) Riska et al., 1984; female mouse; age-specific weights; 9 traits
11) Mavrogenis et al., 1980; sheep; age-specific weights; 6 traits
12) Cheverud and Leamy, 1985; mouse; 17-day live-body traits; 5 traits
13) Cheverud and Leamy, 1985; mouse; 24-day live-body traits; 5 traits
14) Cheverud and Leamy, 1985; mouse; 31-day live-body traits; 5 traits
15) Cheverud and Leamy, 1985; mouse; 38-day live-body traits; 5 traits
16) Cheverud and Leamy, 1985; mouse; 45-day live-body traits; 5 traits
17) Cheverud and Leamy, 1985; mouse; 52-day live-body traits; 5 traits
18) Cheverud and Leamy, 1985; mouse; 59-day live-body traits; 5 traits
19) Cheverud and Leamy, 1985; mouse; 66-day live-body traits; 5 traits
20) Grant, 1983; finch; live-body traits; 5 traits
21) D. Fong and D. Culver, unpubl.; amphipod; 20-day live-body traits; 8 traits
22) D. Fong and D. Culver, unpubl.; amphipod; 40-day live-body traits; 8 traits
23) D. Fong and D. Culver, unpubl.; amphipod; 60-day live-body traits; 8 traits
24) D. Fong and D. Culver, unpubl.; amphipod; 80-day live-body traits; 8 traits
25) D. Fong and D. Culver, unpubl.; amphipod; 100-day live-body traits; 8 traits
26) Black, 1982; human; live-body traits; 27 traits
27) Leamy and Atchley, 1984; rat; skeletal traits; 19 traits
28) Cheverud and Buikstra, 1981; macaque; skeletal traits; 13 traits
29) Leamy, 1977; mouse; skeletal traits; 18 traits
30) Atchley et al., 1981; rat; skeletal traits; 18 traits
31) Atchley, 1983; rat; skeletal traits; 9 traits
32) Koch, 1978; cattle; production traits; 10 traits
33) Smith et al., 1962; pig; production traits; 32 traits
34) Smith and Ross, 1965; pig; production traits; 26 traits
35) Mason et al., 1972; cattle; production traits; 16 traits
36) Benyshek and Little, 1982; cattle; reproductive traits; 6 traits
37) Burfening et al., 1978; cattle; reproductive traits; 6 traits
38) O'Ferrall, 1976; sheep; reproductive traits; 8 traits
39) Plomin and DeFries, 1979; human; behavioral traits; 5 traits
40) Arnold, 1981; inland garter snake; behavioral traits; 12 traits
41) Arnold, 1981; coastal garter snake; behavioral traits; 12 traits