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HOW CLOSELY CORRELATED ARE MOLECULAR AND QUANTITATIVE MEASURES OF GENETIC VARIATION? A META-ANALYSIS

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Abstract.—The ability of populations to undergo adaptive evolution depends on the presence of quantitative genetic variation for ecologically important traits. Although molecular measures are widely used as surrogates for quantitative genetic variation, there is controversy about the strength of the relationship between the two. To resolve this issue, we carried out a meta-analysis based on 71 datasets. The mean correlation between molecular and quantitative measures of genetic variation was weak ($r = 0.217$). Furthermore, there was no significant relationship between the two measures for life-history traits ($r = -0.11$) or for the quantitative measure generally considered as the best indicator of adaptive potential, heritability ($r = -0.08$). Consequently, molecular measures of genetic diversity have only a very limited ability to predict quantitative genetic variability. When information about a population's short-term evolutionary potential or estimates of local adaptation and population divergence are required, quantitative genetic variation should be measured directly.

Key words.—Allozymes, genetic diversity, heritability, phenotypic variance, quantitative genetics.

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Evolutionary and conservation genetics are concerned with the ability of populations to evolve in response to environmental change. This evolutionary response to selection depends on the heritability of the trait(s) and the strength of selection (Franklin 1980; Lande and Barrowclough 1987), although genetic correlations among traits and epistasis may confound long-term projections (Lande 1982). Unlike molecular traits, polygenic (quantitative) traits do not exhibit discrete phenotypes. Quantitative traits vary continuously, due to both the contributions of many loci and environmental effects on those loci. Dominance, epistasis, and pleiotropy further complicate genetic architectures, the expression of polygenic traits, and the maintenance of genetic variation for those traits. Despite the fact that quantitative characters are what selection acts on, the majority of information for wild populations, and almost all for endangered species, is for allozymes and DNA markers (Frankham 1995; Hard 1995; Haig and Avise 1996).

It is widely assumed that the various measures of genetic diversity are positively and strongly correlated (see Soulé et al. 1973; O'Brien et al. 1985; Allendorf and Leary 1986; Houle 1989). However, widely differing opinions about the magnitude of the relationship have been expressed and the extent of the correlation remains controversial. Strong genetic-phenetic correlations are prevalent in the literature (e.g., Soulé and Yang 1973; Briscoe et al. 1992; Soulé and Zegers 1996; Waldman and Andersson 1998). Further, Coyne and Orr (1997) have shown that genetic distances between *Drosophila* species determined from allozymes are correlated with their degree of reproductive isolation. Conversely, Lewontin (1984) and Lynch (1996) have pointed out that correlations are likely to be low. Butlin and Tregenza (1998) analyzed data for 20 species and found the correlation between heterozygosity, as measured by molecular markers, and the coefficient of variation for additive genetic variation of sexually selected traits to be nonsignificant. Patterson et al. (1993) found the relationship between molecular and morphological phylogenies to be weak.

A linear relationship is expected between mean heterozygosity and the variance for a polygenic trait, provided that all gene action is additive (for further details, see Soulé and Yang 1973; Falconer 1989). Therefore, a strong correlation can be expected between the two measures of genetic diversity, based on simple population genetic theory.

Despite this theory, molecular markers may not provide results equivalent to quantitative genetic assays due to nonadditive genetic variation, differential selection, different mutation rates, low statistical power, environmental effects on quantitative characters, and impact of regulatory variation. We elaborate on each of these below.

Quantitative traits associated with fitness typically have large amounts of dominance and epistatic genetic variance (Mather 1973; Crnokrak and Roff 1995). The conversion of nonadditive variation to additive variation in bottlenecked populations (Bryant et al. 1986; Lopez-Fanjul and Villaverde 1989; Wade et al. 1996; Armbruster et al. 1998; Cheverud et al. 1999) can cause departures from the expectation of linearity, lowering the correlation between molecular and quantitative measures of genetic variation.

The degree to which molecular markers can be used to infer the amount of genetic variation in quantitative traits depends in part on the extent to which genetic variation is maintained through selection versus drift, and on what form selection takes. Genetic variation for (nearly) neutral traits is predicted to correlate well with allozyme heterozygosity, because both are affected similarly by population size. Conversely, the relationship may be weaker or absent for polygenic traits under directional selection. This possible dichotomy is important. Traits related to fitness are the ones that concern evolutionary biologists most. Evolutionarily important processes, such as adaptation and speciation, are driven primarily by selection (Futuyma 1998; Rundle et al. 2000).

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The amount of genetic variation present in a population may also differ between molecular markers and quantitative traits, due to the higher mutational input for characters influenced by many loci. Thus, polygenic traits should retain more genetic variability at low population sizes and recover variability more rapidly after a population bottleneck than allozyme loci (Lande and Barrowclough 1987; Lynch 1996).

Statistical power differs between quantitative genetic and molecular measures, although both may be low. Assuming a random sample of unlinked loci, the expected correlation between genomic heterozygosity in the population and that in the sample is equal to the square root of the proportion of loci sampled (Chakraborty 1981). Lewontin (1984, p. 115) stated that, ''differences between species and populations in quantitative characters cannot be compared with differences in gene frequencies at individual loci, because the power of statistical tests to discriminate populations are vastly different for the two kinds of characters'' (but see Felsenstein 1986; Rogers 1986). However, with the advent of highly variable loci, such as microsatellites, this lack of power for molecular markers may be ameliorated to some extent (Hedrick 1999).

Narrow-sense heritabilities and measures of phenotypic variation are environment dependent, whereas molecular markers are not. The expression of quantitative traits is generally quite plastic with respect to environmental effects (Mather 1973). This is likely to reduce correlations with molecular markers when heritabilities or phenotypic variances are used.

Mutations in regulatory genes and gene rearrangements may account for a large proportion of the differences in anatomy, physiology, behavior, and ecology of species (King and Wilson 1975). Such differences and the extent to which they influence genetic variation for quantitative traits are not measured with commonly employed molecular markers. In fact, there are a number of examples of species with large amounts of phenotypic variation existing under extremely varied ecological conditions, but having little allozyme variation (e.g., Simonson 1976; Cherry et al. 1978; Jain et al. 1980; Moran and Kornfield 1993; Cheverud et al. 1994). Whereas mammals have generally been evolving morphologically and taxonomically at a faster rate than other vertebrates, they have the lowest allozyme heterozygosities of any of these groups (Bush et al. 1977).

Despite a body of work, both theoretical and empirical, there is controversy regarding the strength of the relationship between molecular and quantitative measures of genetic diversity (Hard 1995). It is not clear whether there is a weak relationship between quantitative genetic variation and single locus diversity or whether the low power of individual studies is obscuring a strong relationship. The issue of whether a loss of heterozygosity, as measured by molecular markers, corresponds to a proportionate reduction in quantitative genetic variation is a question central to many concerns in population biology. Thus, it is imperative that the strength of the relationship between levels of quantitative genetic variation and molecular measures of genetic variation be quantified. For example, neutral theory is applied to management protocols for endangered species of animals in captivity (Ballou and Lacy 1995). In addition, heterozygosity as measured with molecular markers is used routinely as a surrogate for fitness and as an indirect estimate of the amount of genetic variation for polygenic traits in a population (Allendorf et al. 1997).

Given the problems of statistical power, meta-analysis provides an excellent way of examining the strength of the correlation between molecular and quantitative measures of genetic variation. The primary purpose of meta-analyses is to accumulate knowledge across studies and to ameliorate problems associated with a lack of statistical power in individual studies (Hunter and Schmidt 1990). Meta-analyses have been used to investigate a wide range of issues in population biology (e.g., Griffith et al. 1989; Gurevitch et al. 1992; Arnqvist and Wooster 1995; Britten 1996).

The aim of this study was to quantify the magnitude of the correlation between molecular and quantitative measures of genetic variation. We test the null hypothesis that molecular measures of genetic variation accurately reflect quantitative genetic variation. Also, we assess whether the relationship differs for (1) life-history versus morphological traits; and (2) alternative measures of quantitative genetic variation.

Materials and Methods

Key word searches were conducted using Biological Abstracts CD ROM, Biosis, and Medline. These formal searches of databases were supplemented by searches through references of papers obtained from the databases and by using papers or unpublished datasets that were already known to us. Any data that could be found, where three or more populations were measured for both molecular heterozygosity and a measure of variation for at least one quantitative trait, were used. It must be emphasized here that all correlations are among populations. Papers where genetic distances were calculated separately for both morphological and molecular measures were also used. Correlations between the two distance matrices were calculated. The 71 datasets are identified and described in the Appendix.

The Pearson product moment correlation coefficient, *r,* was the common metric used in this study. The correlation coefficients were not transformed (Hunter and Schmidt 1990). Correlations were based on weighted averages, thus giving more strength to the results of larger experiments. Sample size was determined using a nested (hierarchical) design (Sokal and Rohlf 1995) according to the following formula:

$$[(AK_1N_1 - 1) + (AK_2N_2 - 1)]^{0.5}, \quad (1)$$

where A is the number of populations, K the number of polymorphic loci or quantitative traits (subscripts 1 and 2, respectively) assayed, and N is the mean number of individuals used for each assay, respectively. The degrees of freedom for a correlation are determined solely by the number of populations, but the actual power of the correlation to reveal the underlying relationship also depends on the standard errors surrounding the point estimates used in the correlation. Thus, this nested approach to determining sample sizes was deemed appropriate. Alternative weighting methods (number of populations, polymorphic loci, quantitative traits) did not alter the conclusions of the study.

The molecular assays used in this study were predomi-

TABLE 1. Mean correlation coefficients (r), between molecular and quantitative genetic variation under different weighting methods. A regression was performed and an r^2-value is given, showing the amount of variance in the correlation coefficient that is explained by the various measures of sample size. None of the regressions are significant.

Weighting method	Mean r ± SE	r^2
Unweighted	0.185 ± 0.051	NA
Median	0.161	NA
Nested	0.217 ± 0.050	0.016
Populations	0.228 ± 0.048	0.025
Loci	0.280 ± 0.050	0.025
Traits	0.249 ± 0.048	0.022

nantly based on allozyme electrophoresis (60 of 71 datasets as detailed in the appendix). The quantitative genetic measures, varied, however. In fact, three very distinct types of data were used: (1) correlations based on heritabilities or other measures reflecting levels of additive genetic variance and therefore short-term adaptive potential: This correlation is the most important to conservation concerns. The heritability category includes 15 measures of heritability, two measures of additive genetic variance, and two measures of the coefficient of variation for additive genetic variation; (2) correlations based on the coefficient of variation or other measures of phenotypic variation: The phenotypic category includes 22 measures of the coefficient of variation for quantitative traits and five measures of phenotypic variation itself; and (3) Correlations (n = 22) based on measures of genetic distance (typically Mahalanobis phenetic distances or Q_{ST} with Nei's [1987] genetic distances): Matrices corresponding to molecular and quantitative genetic data were formed and their results correlated. This provides a measure of the general level of agreement between molecular and quantitative data in assigning genetic distances between pairs of populations. If selection on quantitative traits differs from that on molecular markers, the difference in selection regimes is expected to be evident in the lack of correlation between the distance matrices (Slatkin 1987; Spitze 1993). Unlike other measures used in this study, genetic distances are based on trait means rather than variances.

Statistical tests were carried out comparing the type of trait under examination, life historical versus morphological; quantitative genetic measure used, genetic distance, heritability, or phenotypic variance; and whether the correlation was presented in the original paper. The data and subsets thereof were normally distributed (Shapiro-Wilk test). Thus, all statistical testing was done using analysis of variance.

RESULTS

The weighted mean correlation coefficient for the 71 datasets was 0.217 (Table 1). This is considered to be a small effect, but the correlation was significantly different from

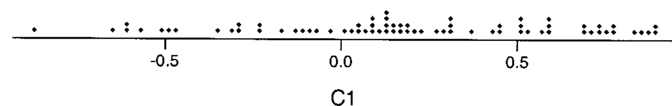

FIG. 1. Distribution of correlation coefficients (r) for the 71 datasets used in the meta-analysis.

TABLE 2. Comparison of mean weighted correlation coefficients (r), between molecular and quantitative genetic variation for life-history versus morphological traits. They differed, as indicated by an F-test (F = 14.39; df = 1, 69; $P < 0.0003$).

Trait	N	Mean r ± SE
Life history	19	−0.110 ± 0.098
Morphological	52	0.311 ± 0.052

zero. The correlations had a median of 0.161. Correlations between molecular and quantitative variation were highly variable ranging from −0.88 to 0.90, with a standard deviation of 0.433 (Fig. 1, Table 1). The mean correlation was little affected by different methods of weighting, ranging from 0.22 to 0.28 (Table 1).

The median number of populations, quantitative traits, and polymorphic loci used in the studies were seven, seven, and eight, respectively. Linear regressions based on various measures of sample size were carried out. The number of populations used in a study, the number of quantitative traits used in a study, and the number of polymorphic loci screened were each able to explain no more than 2.5% of the variance in correlation coefficients (Table 1) and were nonsignificant.

The mean weighted correlation was lower for life-history than for morphological traits, as predicted (Table 2). The correlation for morphological traits was weak to moderate (r = 0.31 ± 0.05 SE) and significantly different from zero. Conversely, the correlation between molecular measures and life history traits was not significantly different from zero, and the direction of the correlation was negative (r = −0.11 ± 0.10).

Correlations differed significantly among the types of quantitative measures used (Table 3). Correlations between molecular heterozygosity and the amount of phenotypic variance (typically the coefficient of variation) was moderate and significantly different from zero (r = 0.36 ± 0.07). The correlation between measures of genetic distances was weak (r = 0.22 ± 0.08), but still significantly different from zero. The correlation between the heritability of a trait and heterozygosity was not significantly different from zero, and the direction of the correlation was negative (r = −0.08 ± 0.11).

The file-drawer problem (unpubl. data) is a concern with meta-analysis. The distribution of correlations displayed the funnel shape indicative of a lack of publication bias (Light and Pillemer 1984). However, papers that self-reported the relationship had higher correlations (r = 0.265 ± 0.057, n = 47) than those where the correlation was not reported but was calculated as part of this study (r = 0.002 ± 0.112, n = 24, $F_{1, 69}$ = 4.22, $P < 0.05$). Perhaps there is additional data that we could not locate because the authors did not

TABLE 3. Comparison of mean weighted correlation coefficients (r), between molecular and quantitative genetic variation for different measures of quantitative genetic variation. The correlations differ significantly, as indicated by an F-test (F = 5.69; df = 2, 69; $P < 0.0052$).

Quantitative measure	N	Mean r ± SE
Genetic distance	26	0.217 ± 0.077
Heritability	19	−0.081 ± 0.107
Phenotypic	26	0.356 ± 0.073

wish to address the question of the correlation between molecular and quantitative measures of genetic variation or because they found no significant effect and therefore did not report it. Thus, an already weak relationship should be viewed as the maximum likely correlation between the two measures.

The data was further subdivided based on the use of allozymes exclusively versus other forms of molecular markers, taxon, self-compatible versus self-incompatible plants, and wild versus laboratory or greenhouse populations. None of these tests yielded significant effects.

Discussion

The major finding of this study is that the correlation between assays of molecular marker diversity and quantitative trait variation is weak. At best, molecular measures only explain 4% of the variation in quantitative traits. Most disturbingly, the relationship is weakest for the measures of greatest interest to evolutionary and conservation biologists, those associated with life-history traits and heritabilities.

If allozyme polymorphisms are determined primarily by drift, the amount of heterozygosity present may reflect patterns of quantitative genetic variance in traits unrelated to fitness more closely than for fitness or life-history traits. The results of this study confirm these expectations. Allozymes, or other forms of neutral or nearly neutral molecular markers, are unlikely to accurately predict patterns of variation in quantitative traits when selection, rather than drift, is the primary force acting (e.g., local adaptation, speciation).

The correlation of most concern to conservation and evolutionary biologists—that between molecular markers and heritabilities—was not significantly different from zero ($r = -0.08$) and was significantly less than the correlation with phenotypic variance. The correlation between molecular diversity and phenotypic variance was moderate in effect ($r = 0.36$) and significantly different from zero. This genetic-phenetic correlation has been argued to be positive because of its linear relationship with heterozygosity (Soulé and Yang 1973) or negative (Lerner 1954) due to the loss of developmental stability brought about by lowered heterozygosity. Correlations based on measures of genetic distance were weak ($r = 0.22$) but significantly different from zero. Again, allozymes or other forms of neutral molecular markers, are unlikely to provide conservation or evolutionary biologists with reliable information on a population's evolutionary potential or to accurately reflect population differentiation and local adaptation.

Why are the correlations so low? Six factors reducing the correlations were described in the introduction, namely nonadditive genetic variation, differential selection, impact of regulatory variation, different mutation rates, low statistical power, and environmental effects on quantitative characters.

Correlations were lower for life-history characters than morphological characters and lower for heritabilities than for phenotypic variance. This suggests that selection is the most important factor influencing the strength of the correlation between quantitative and molecular measures of genetic variation. Nonadditive genetic variation could also contribute to the difference between correlations involving life-history traits and morphological traits, but the levels of nonadditive genetic variation are themselves a reflection of selection on characters (Mather 1973; Crnokrak and Roff 1995). The impact of variation in regulatory genes is difficult to evaluate, but is likely to be associated with selective differences. In the long term, differences in mutation rates would be expected to result in lower correlations for life-history traits than for morphological characters, because fitness characters are influenced by a greater number of loci (Houle et al. 1996; Merilä and Sheldon 2000). Differences in correlations were in accord with this prediction. However, differences in mutation rates cannot explain the lower correlation for heritabilities. Statistical power does not seem to be the major issue, because correlations showed little relation with sample size, at least with the sample sizes usually employed in such studies. Overall, differences in selective forces are likely the major factor leading to low correlations between molecular and quantitative measures of genetic variation.

Publication bias is a concern in meta-analyses. In this case, any biases are likely to be in the direction of underreporting of nonsignificant or negative correlations. Consequently, the already weak relationship between molecular and quantitative measures of genetic variation found in this study is likely to be inflated.

Our findings have important implications in evolutionary and conservation genetics. The low correlation between molecular and quantitative genetic variation means that molecular measures alone cannot be relied upon to reflect evolutionary potential of populations, especially for life-history traits. For example, endangered species have been shown to have low genetic diversity, as measured by molecular means (see Frankham 1995; Haig and Avise 1996). However, there is insufficient data for quantitative characters to evaluate the issue for them. Consequently, it is unclear if evolutionary potential is reduced in endangered species compared to related nonendangered species.

Molecular data, however, have numerous uses in evolution and conservation (example, determining homology from analogy, parentage and kinship, hybridization, mating systems, population structure, dispersal rates, gene flow, and effective population size). Methods for performing quantitative genetic analyses on natural populations for which pedigrees are unknown have been developed based upon the use of DNA markers (Ritland 1996; Lynch 1999). Field studies have already produced exciting results, for example, the first two estimates of the heritability of lifetime reproductive success in natural populations (Kruuk et al. 2000; Merilä and Sheldon 2000).

Our results strengthen concerns as to whether molecular measures of genetic diversity reflect adaptive difference among populations (see Hedrick and Savolainen 1996; Karhu et al. 1996; Crandall et al. 2000). Such concerns extend to the use of molecular markers to resolve taxonomic uncertainties for allopatric populations (Hard 1995) and the classification of evolutionarily significant units (Crandall et al. 2000). Molecular markers are widely used in these contexts, but there are broad overlaps between genetic distances for populations with different levels of reproductive isolation (see Nei 1987; Coyne and Orr 1989; Avise and Johns 1999).

Conclusions

Molecular markers primarily reflect the effects of genetic drift and do not accurately reflect adaptive evolutionary processes, evolutionary potential, or differentiation among populations brought about by natural selection. Given the weak correlation between molecular and quantitative genetic variation, it is imperative that quantitative trait variation be directly assessed when studying processes involving selection.

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APPENDIX

Dataset summary.

Species	r	Trait[1]	Measure[2]	Weight	Source
Avena barbata	0.90	LH	GD	62	Price et al. 1984
Bicyclus anynana	−0.30	M	H	75	Oosterhout 1998
Bicyclus anynana	0.07	M	H[a]	81	Saccheri et al. 1996, 1999
Carduelis chloris	0.71	M	GD	60	Merilä 1997
Carduelis chloris	0.50	M	GD	60	Merilä 1997
Clarkia williamsonii	−0.30	LH	GD	51	Price et al. 1984
Clupea harengus	0.59	M	P	110	Ryman et al. 1984
Cottus	0.73	M	P	247	Strauss 1991
Cottus	0.19	M	P	241	Strauss 1989
Daphnia obtusa	0.16	LH	H	79	Spitze 1993
Daphnia pulex	−0.17	LH	H[b]	100	Lynch et al. 1999
Dipodomys	0.06	M	GD	88	Schnell et al. 1978
Dipodomys	0.29	M	GD	88	Schnell et al. 1978
Dipodomys	−0.09	M	GD	88	Schnell et al. 1978
Dipodomys	0.11	M	GD	88	Schnell et al. 1978
Dolichopoda	−0.30	M	GD	70	Allegrucci et al. 1987
Drosophila melanogaster	0.89	M	H	52	Briscoe et al. 1992
Drosophila melanogaster	0.30	M	H	39	R. Frankham, unpubl. data
Drosophila melanogaster	0.21	M	H	27	D. Gilligan, unpubl. data
Drosophila melanogaster	0.31	M	H	27	D. Gilligan, unpub. data
Drosophila melanogaster	0.69	M	H	26	Lopez-Fanjul and Hill 1973
Fringilla coelebs	0.68	M	P	145	Dennison and Baker 1991
Gentiana pneumonanthe	−0.49	LH	P	42	Oostermeijer et al. 1995
Gypsophila fastigiata	0.59	M	GD	53	Prentice 1992
Hordeum jubatum	−0.23	LH	GD	165	Price et al. 1984
Hordeum spontaneoum	0.37	M	P	66	Nevo t al. 1979
Hordeum vulgare	−0.13	LH	GD	33	Price et al. 1984
Mullus barbatus	0.08	M	GD[c]	66	Mammuris et al. 1998
Mullus barbatus	0.05	M	GD[c]	124	Mammuris et al. 1998
Mullus barbatus	0.09	M	P[c]	124	Mammuris et al. 1998
Mullus barbatus	0.30	M	P[c]	66	Mammuris et al. 1998
Multiple	−0.04	LH	H	57	Butlin and Tregenza 1998
Multiple	−0.36	LH	H	63	Butlin and Tregenza 1998
Mus musculus	0.09	LH	P	37	Schnell and Selander 1981
Musca domestica	0.53	M	H	24	Bryant et al. 1999
Musca domestica	0.14	LH	H	41	Reed 1998
Musca domestica	0.57	M	P	100	Bryant 1984
Nassella pulchra	0.46	M	GD	54	Knapp and Rice 1998
Oncorhynchus gorbuscha	0.51	M	GD	104	Beacham and Withler 1985
Oncorhynchus gorbuscha	0.27	M	GD	104	Beacham and Withler 1985
Pararge aegeria	−0.51	M	P	46	Berwaerts et al. 1998
Pararge aegeria	0.52	LH	P	42	Berwaerts et al. 1998
Passarella iliaca	−0.07	M	P	114	Zink et al. 1985
Peromyscus maniculatus	0.02	M	GD	52	Aquadro and Kilpatrick 1981
Peromyscus maniculatus	0.12	M	GD[d]	67	Ashley and Willis 1989
Peromyscus maniculatus	0.18	M	GD[d]	25	Ashley and Willis 1989
Peromyscus maniculatus	0.82	M	P	52	Aquadro and Kilpatrick 1981
Peromyscus polionutus	0.01	M	GD	112	Schnell and Selander 1981
Petrorhagia prolifera	0.22	M	GD	32	Lönn and Prentice 1990
Phlox drummondii	0.42	M	GD	85	Schwaegerle et al. 1986
Phlox drummondii	−0.48	LH	H	40	Schwaegerle et al. 1986
Phlox drummondii	−0.61	M	H	76	Schwaegerle et al. 1986
Raphanus sativus	0.75	M	GD	211	Rabbani et al. 1998
Rattus rattus	−0.60	M	GD	90	Patton et al. 1975
Rattus rattus	0.77	M	P	90	Patton et al. 1975
Salvia pratensis	0.16	LH	P	130	Ouborg and Van Treuren 1995
Scabiosa	0.84	M	H	35	Waldmann and Andersson 1998
Scabiosa	0.14	LH	H	35	Waldmann and Andersson 1998
Sigmodon hispidus	−0.57	M	P	57	McClenaghan and Gaines 1981
Thomomys bottae	0.87	M	P	84	Soulé and Zegers 1996
Thomomys	0.58	M	P	131	Soulé and Zegers 1986
Thomomys bottae	0.14	M	P	135	Zink et al. 1985
Tupinambis	0.13	LH	P	21	Colli et al. 1998
Tupinambis	−0.22	LH	P	26	Colli et al. 1998

APPENDIX
Continued.

Species	*r*	Trait[1]	Measure[2]	Weight	Source
Tupinambis	−0.12	M	P	27	Colli et al. 1998
Urocyon littoralis	0.78	M	GD[b]	84	Wayne et al. 1991
Urocyon littoralis	0.45	M	P[b]	84	Wayne et al. 1991
Uta stansburiana	0.73	M	P	67	Soulé 1973
Wyeomyia smithii	−0.66	LH	H	81	Armbruster et al. 1998
Wyeomyia smithii	−0.88	LH	H	57	Armbruster et al. 1998
Zonotrichia capensis	0.17	M	P	73	Yezerina et al. 1992

[1] LH, life-history trait; M, morphological trait.

[2] GD, genetic distance; H, additive genetic variance measure; P, phentotypic variability. The molecular measure used was allozymes, except for the studies indicated by a subscript, where they were (a) minisatellites and allozymes; (b) mtDNA; (c) RAPDs; (d) mtDNA and allozymes.