Correlation between Fitness and Genetic Diversity

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Abstract: *Genetic diversity is one of the three forms of biodiversity recognized by the World Conservation Union (IUCN) as deserving conservation. The need to conserve genetic diversity within populations is based on two arguments: the necessity of genetic diversity for evolution to occur, and the expected relationship between heterozygosity and population fitness. Because loss of genetic diversity is related to inbreeding, and inbreeding reduces reproductive fitness, a correlation is expected between heterozygosity and population fitness. Long-term effective population size, which determines rates of inbreeding, should also be correlated with fitness. However, other theoretical considerations and empirical observations suggest that the correlation between fitness and heterozygosity may be weak or nonexistent. We used all the data sets we could locate (34) to perform a meta-analysis and resolve the issue. Data sets were included in the study, provided that fitness, or a component of fitness, was measured for three or more populations along with heterozygosity, heritability, and/or population size. The mean weighted correlation between measures of genetic diversity, at the population level, and population fitness was 0.4323. The correlation was highly significant and explained 19% of the variation in fitness. Our study strengthens concerns that the loss of heterozygosity has a deleterious effect on population fitness and supports the IUCN designation of genetic diversity as worthy of conservation.*

Correlación entre Adaptabilidad y Diversidad Genética

Resumen: *La diversidad genética es una de las tres formas de biodiversidad reconocidas por la Unión de Conservación Mundial (IUCN) que merecen ser conservadas. La necesidad de conservar la diversidad genética de las poblaciones se basa en dos argumentos: la necesidad de diversidad genética para que ocurra la evolución, y la relación esperada entre la heterocigosidad y la adaptabilidad de la población. Debido a que la pérdida de diversidad genética se relaciona con la endogamia, y la endogamia reduce la aptitud reproductiva, se espera una correlación entre heterocigosidad y adaptabilidad de la población. Sin embargo, otras consideraciones teóricas y observaciones empíricas sugieren que la correlación entre adaptabilidad y heterocigosidad puede ser débil o inexistente. Utilizamos todos los conjuntos de datos que pudimos localizar (34) para realizar un meta-análisis y aclarar el asunto. Se incluyeron conjuntos de datos en el estudio siempre y cuando se disponía de medidas de adaptabilidad, o un componente de la adaptabilidad, así como de la heterocigosidad, heredabilidad y/o tamaño de la población para tres o más poblaciones. La correlación media ponderada entre medidas de diversidad genética, a nivel de población, y la adaptabilidad de la población fue 0.4323. La correlación fue altamente significativa y justificaba el 19 % de la variación de la adaptabilidad. Nuestro estudio apoya la hipotesis de que la pérdida de heterocigosidad tiene efecto deletéreo sobre la adaptabilidad de la población y apoya la designación de diversidad genética de IUCN como merecedora de conservación.*

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Introduction

Genetic variation is one of the three levels of biodiversity that the World Conservation Union (IUCN) has recommended for conservation (McNeely et al. 1990). There are two reasons for this recommendation: (1) genetic diversity is required for populations to evolve in response to environmental changes and (2) heterozygosity levels are linked directly to reduced population fitness via inbreeding depression. This leads to the expectation that levels of heterozygosity and fitness, at the population level, will be correlated.

Finite populations lose genetic variation as a consequence of genetic drift and at the same time become inbred. This loss of heterozygosity can be described by the inbreeding coefficient, F, which is related to the amount of genetic variation present, in the absence of mutation and selection, according to the following formula:

$$H_t/H_0 = [1-(1/2N_e)]^t = 1 - F_t, \quad (1)$$

where t is time in generations, N_e is effective population size, and H is the amount of heterozygosity present in the population. The rate at which molecular heterozygosity is lost per generation ($1/2N_e$) also applies, theoretically, to the loss of additive genetic variation (Falconer & MacKay 1996). A strong correlation can therefore be expected between population size and heterozygosity, based on simple population genetic theory. The expected correlation between population size and heterozygosity has been validated at the empirical level (Frankham 1996).

Inbreeding leads to inbreeding depression in virtually all species studied thus far (e.g., Wright 1977; Charlesworth & Charlesworth 1987; Frankham 1995; Lynch & Walsh 1998; Crnokrak & Roff 1999; Hedrick & Kalinowski 2000), and inbreeding depression is directly related to the inbreeding coefficient, F (Falconer & MacKay 1996). Thus, the amount of genetic variation a population contains is predicted to correlate with current fitness and, in the case of heritabilities, with evolutionary potential (Franklin 1980; Ralls & Ballou 1986; Soulé 1986; Frankham 1995; Lande 1995).

Despite this theory, the correlation between fitness and levels of genetic variation may be weak or nonexistent because of (1) the neutrality of molecular markers used to estimate heterozygosity, (2) nonadditive genetic variation (in the case of heritabilities), and (3) the purging of deleterious alleles due to increased selection against homozygotes.

A body of literature suggests that allozyme heterozygosity is a good measure of population fitness and adaptive potential (e.g., Garten 1976; Soulé & Wilcox 1980; Beardmore 1983; Allendorf & Leary 1986; Houle 1989). Others (e.g., Hedrick & Miller 1992; Reed & Frankham 2001) caution that such molecular genetic data generally reflect only a small portion of the genome and thus may not be a good indicator of adaptive genetic differences. Because molecular markers are selectively neutral, or nearly so, they may lose genetic variation more rapidly than loci concerned with fitness. In a recent meta-analysis, Reed and Frankham (2001) found that the correlation between heterozygosity at molecular markers and heritabilities is zero. Thus, although molecular markers may be useful for assessing the extent of genetic drift, loss of molecular variation does not necessarily imply a loss of fitness or adaptive potential.

Quantitative traits associated with fitness typically have a larger proportion of their total genetic variance in the form of dominance and epistatic variance than in the form of traits less closely associated with fitness (Mather 1973; Falconer & MacKay 1996; DeRose & Roff 1999). Empirical studies of morphology, behavior, and life history indicate that additive genetic variance, and therefore heritabilities, can remain high or even increase despite severe reductions in population size (Bryant et al. 1986; Lopez-Fanjul & Villaverde 1989; Bryant & Meffert 1993; Wade et al. 1996; Armbruster et al. 1998; Cheverud et al. 1999). This increase in the heritability of a trait is thought to occur via the conversion of nonadditive genetic variation to additive genetic variation (Bryant et al. 1986), perhaps as an evolved response to fluctuations in population size through time (Reed 1998). If this effect is widespread, the correlation between fitness and population size will not be linear and the underlying correlation with genetic variation will be weak.

Most of the alleles likely to be lost in small populations are generally neutral or slightly deleterious (Kimura 1983). Deleterious alleles, in mutation-selection balance, are probably responsible for at least half the genetic variation in fitness (Charlesworth & Hughes 2000). Selection tends to purge the population of deleterious recessive alleles (Lande & Schemske 1985; Charlesworth & Charlesworth 1987) and therefore in theory can create inbred populations with a higher fitness than their outbred progenitor. Thus, inbred populations with less genetic diversity would have higher fitness, and the correlation between the two would be negative in these populations.

This is true only if the population is not kept small enough for long enough to allow the fixation of deleterious alleles to occur (Reed & Bryant 2000). The purging of deleterious alleles during inbreeding will obscure the correlation between heterozygosity and fitness. This is particularly true for correlations based on molecular markers. However, the effects of purging appear to be rather weak (McCall et al. 1994; Ballou 1997; Lacy & Ballou 1998; Byers & Waller 1999; Reed & Bryant 2001).

Thus, there is controversy over (1) whether genetic variation should correlate with population fitness, (2) what types of genetic measures are most predictive of fitness, and (3) whether population size itself is impor-

tant to fitness. This controversy is central to genetic concerns in conservation biology. Resources are being used to perform assays of molecular genetic diversity. Conservation decisions are being made based on these assays without clear evidence of the correlation between these markers and populations fitness. We explored, through meta-analysis, how well molecular data, quantitative genetic data, and population size correlate with population fitness.

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 & Schmidt 1990). For meta-analysis to be successful, two things are necessary: the findings must be conceptually comparable and the studies must be configured in similar statistical forms (Lipsey & Wilson 2000). The correlations between fitness and genetic variation we present here fit both criteria. Although the data available are somewhat heterogeneous, the distinct subcategories of findings can be valuable for permitting comparisons among them (Lipsey & Wilson 2000).

Methods

We conducted key word searches of *Biological Abstracts* (CD ROM), *BIOSIS*, and *MEDLINE*. These formal searches of databases were supplemented by searches of the literature cited sections of papers obtained from the databases and by examining papers and unpublished data known to us. We included data from studies in which fitness or a component of fitness was measured for three or more populations and in which heterozygosity, and/or heritability, and/or population size were measured. It must be emphasized that the correlations considered are all among populations, not among individuals. Plants considered primarily selfing were excluded from the study. The 34 data sets used in our analysis are described in appendices 1 and 2.

The Pearson product-moment correlation coefficient, r, was the common metric used in this study. The correlation coefficients were not transformed (Hunter & Schmidt 1990). The traits considered for analysis were normally distributed (Shapiro-Wilk test). Thus, all statistical testing was done with parametric tests.

Overall correlations were based on weighted averages, which lend more credence to the results of larger experiments. Weightings were determined according to the following formula:

$$[(A-2)\,N]^{0.5}, \qquad (2)$$

where A is the number of populations and N is the mean number of individuals used for the fitness assay per population. The degrees of freedom for a correlation are determined solely by the number of populations (A), 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 (Reed & Frankham 2001). We used the sample size from the fitness test because the sampling methods for the various surrogates (heritability, heterozygosity, or population size) were too heterogeneous to be compared.

We used analyses of variance to compare (1) the fitness surrogate used (heterozygosity, heritability, or population size), (2) the fitness measure used (total fitness [number of adult progeny produced or population growth rate] or a component thereof), (3) taxonomic classification (invertebrate, plant, or vertebrate), and (4) whether or not fitness was measured in the natural environment. All statistical tests were carried out with JMP software (version 3.0; SAS Institute) on weighted means and variances.

In some instances the data sets we used provided more than one fitness component. In these cases we used the fitness component expected to most closely correlate with total fitness (i.e., fecundity or survival measures were given preference over fluctuating asymmetry or individual growth rates). In cases where there were multiple, equally valid fitness measures, we used the mean correlation of all fitness measures. Details of the fitness measures we used are given in Appendix 1. For correlations between fitness and heritabilities in the narrow sense, where heritabilities for the population were calculated for more than one trait, the mean heritability of all the traits was used. Details of the traits for which heritabilities were calculated are given in Appendix 2.

Results

The weighted mean correlation coefficient between fitness and genetic diversity for the 34 data sets was moderate, with a mean of 0.432 ± 0.0577; (Fig. 1). Twenty-eight of the 34 correlations were positive. The overall weighted mean correlation was significantly different from zero. The correlations among fitness and its surrogates were highly variable (SD = 0.336; Fig. 1). The median correlation was 0.440.

The correlations with fitness, among the various surrogates, did not differ from one another. Current population size had the lowest correlation, in absolute terms (mean $= 0.354 \pm 0.111$, $n = 11$) but was still significantly different from zero. The correlation between heritability and fitness was the highest (mean $= 0.509 \pm 0.134$, $n = 6$) and was significantly different from zero. The correlation between molecular heterozygosity and fitness was moderate (mean $= 0.447 \pm 0.081$, $n = 17$) and significantly different from zero.

The "file drawer" problem (i.e., unpublished data) is a

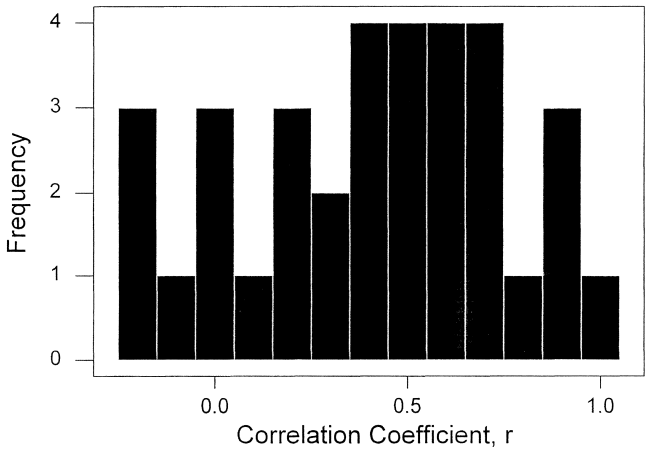

Figure 1. Distribution of correlations between reproductive fitness and genetic diversity (or a surrogate thereof). The distribution's weighted mean is 0.432 and the median is 0.440.

concern with meta-analysis. The distribution of correlations in this study did not display the funnel shape indicative of a lack of publication bias (Light & Pillemer 1984; Palmer 2000). However, a regression of weighting factor on the correlation coefficient was not significant ($r^2 = 0.043$, $p = 0.238$) and the slope of the line was positive.

There were no significant differences in the weighted correlation whether fitness was measured in the natural environment or in a common environment (i.e., laboratory or greenhouse), whether fitness itself was measured or a component of fitness ($F = 2.698$, $p = 0.110$) or taxonomic grouping (i.e., invertebrate, plant, or vertebrate) ($F = 0.752$, $p = 0.480$). The number of loci used in molecular-marker studies was not significantly related to the correlation between fitness and heterozygosity ($r^2 = 0.003$, df = 15, $p = 0.831$).

Discussion

Our major finding was that commonly used surrogates for fitness—heterozygosity, population size, and quantitative genetic variation—were positively and significantly correlated with population fitness. They explained, however, only 15–20% of the variation in fitness. This positive correlation is in agreement with theory. If inbreeding and drift are important constituents of current population fitness, then heterozygosity and population size should be positively correlated with fitness among populations of a species. Despite the noise inherent in a meta-analysis such as this, the correlations were surprisingly high.

The correlation of heritabilities with current fitness is of particular interest to conservation and evolutionary biologists. Heritabilities provide estimates of a population's ability to respond to selective pressures posed by the changing environment. The link between heritability and fitness may be obscured by the conversion of nonadditive to additive genetic variance during reductions in population size or by additive genetic variance having been expended in past efforts to adapt to the current environment. We found the correlation between heritability and population fitness to be large.

Levels of heterozygosity, as measured by molecular markers, correlate significantly with population size (Frankham 1996). Thus, there is an expectation that heterozygosity will also correlate with fitness. But because the markers themselves are generally considered neutral and the total sample relative to the genome is small, this correlation may not be strong. Reed and Frankham (2001) found the correlation between heritability and molecular heterozygosity to be zero. The significant and positive correlation between heterozygosity and fitness found here suggests that heterozygosity, through its link with population size, can give some indication of population fitness.

Of the surrogates tested, population size should have the most direct link to fitness. But because most of the estimates of population size encompassed only 1 year and contemporary population size is often a poor surrogate for effective population size, the long-term effects of fluctuating population size on eroding fitness and genetic variation may not be apparent. Thus, heterozygosity or levels of quantitative genetic variation may give a better estimate of long-term effective population size than a one-time count of individuals. Despite this drawback, population size did correlate significantly with fitness.

All three individual surrogates had highly significant and positive correlations with fitness. However, comparisons among subsets of the data were hampered by the small number of data sets available for analysis and the uneven number of studies among the surrogates (heritability, heterozygosity, and population size). The power of our meta-analysis to determine differences among the various surrogates was very weak (estimated Type II error rate approximately 80%). Total fitness and components of fitness should have differed in the magnitude of their correlations because of trade-offs among different components of fitness (e.g., seed weight and seed number, growth rate and survival). Despite a trend in that direction, however, the number of studies was too few to permit concrete conclusions.

The conclusions we can draw from this study are that (1) fitness and future adaptability are reduced in smaller populations of plants and animals, due to drift and inbreeding depression and (2) commonly used surrogates for fitness—heritabilities, heterozygosity, and population size—significantly correlate with fitness and explain 15–20% of the variation in fitness.

Correlations reported here, in combination with the

typical population sizes of endangered species, suggest that many populations have reduced fitness as a result of inbreeding depression and genetic drift. The concern over genetic variation is particularly warranted in light of the fact that endangered species typically have lower levels of heterozygosity than related nonendangered species (Frankham 1995; Haig & Avise 1996). The loss of adaptive genetic variation and inbreeding depression puts wildlife populations at an increased risk of extinction. This increase can occur as a result of reduced reproductive fitness due to inbreeding depression, or from a failure to track the changing abiotic and biotic environment of the population as a result of the loss of genetic variation through drift. Several studies of natural populations provide clear examples of inbred populations for which migration, to counter inbreeding depression and drift, was a remedy for low reproductive fitness (Vrijenhoek 1994; Heschel & Paige 1995; Westemeier et al. 1998; Madsen et al. 1999).

Our results strengthen concerns over the loss of genetic diversity in endangered populations of plants and animals. Not only does heterozygosity level relate to evolutionary potential, but our study validates its correlation with current population fitness. Natural populations need to be kept at a size sufficient to retain genetic diversity and minimize their risk of extinction.

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Appendix 1. **Data sets used in the analyses of correlations between genetic diversity and reproductive fitness.**

Species	r[a]	n[b]	*Surrogate*[c]	*Measure*[d]	*Taxon*	*Reference*
Arnica montana	0.378	18	*N*	fitness	plant	Luijten et al. 2000
Bicyclus anynana	0.518	14	b^2	fitness	invertebrate	Oosterhout 2000
Bicyclus anynana	0.860	20	*H*	fitness	invertebrate	Oosterhout 2000
Bicyclus anynana	0.510	16	b^2	component	invertebrate	Saccheri et al. 1996
Bicyclus anynana	0.607	28	*H*	component	invertebrate	Saccheri et al. 1999
Bolitotherus cornutus	−0.010	29	*H*	component	invertebrate	Whitlock 1993
Bombus terrestris	0.550	11	*H*	component	invertebrate	Liersch & Schmid-Hempel 1998
Bufo calamita	0.572	14	*H*	component	vertebrate	Rowe et al. 1999
Drosophila melanogaster	0.369	17	b^2	fitness	invertebrate	Gilligan 2001
Drosophila melanogaster	0.173	17	*H*	fitness	invertebrate	Gilligan 2001
Drosophila melanogaster	0.388	33	b^2	fitness	invertebrate	D. H. Reed, unpublished
Gentiana pneumonanthe	0.268	12	*H*	component	plant	Oostermeijer et al. 1995
Gentiana pneumonanthe	0.111	12	*N*	component	plant	Oostermeijer et al. 1995
Gentianella germanica	0.69	20	*H*	component	plant	Fischer & Matthies 1998*a*
Gentianella germanica	0.60	09	*N*	component	plant	Fischer & Matthies 1998*a*
Ipomopsis aggregata	0.303	09	*N*	component	plant	Heschel & Paige 1995
Limnanthes floccosa	0.738	12	*N*	component	plant	Dole & Sun 1992
Lychnis viscaria	−0.062	16	*H*	component	plant	Lammi et al. 1999
Lychnis viscaria	−0.208	16	*N*	component	plant	Lammi et al. 1999
Musca domestica	0.60	17	b^2	component	invertebrate	Bryant et al. 1999
Musca domestica	0.88	33	*H*	component	invertebrate	Bryant et al. 1999
Musca domestica	0.731	20	b^2	fitness	invertebrate	Reed 1998
Musca domestica	0.365	20	*H*	fitness	invertebrate	Reed 1998
Nassella pulchra	0.170	22	*H*	component	plant	Knapp & Rice 1998
Peromyscus maniculatus	0.73	12	*H*	component	vertebrate	Meagher 1999
Petrogale lateralis	0.998	08	*H*	component	vertebrate	Eldridge et al. 1999
Picea glauca	0.173	19	*H*	component	plant	Furnier et al. 1991
Rana temporaria	0.008	23	*H*	component	vertebrate	Hitchings & Beebee 1997
Rana temporaria	−0.024	23	*N*	component	vertebrate	Hitchings & Beebee 1997
Salvia pratensis	−0.183	07	*N*	component	plant	Ouborg & van Treuren 1995
Senecio integrifolius	0.897	26	*N*	component	plant	Widén 1993
Silene regia	0.492	18	*N*	component	plant	Menges 1991
Swainsona recta	−0.241	09	*H*	component	plant	Buza et al. 2000
Swainsona recta	0.208	09	*N*	component	plant	Buza et al. 2000

[a] *Correlation coefficient,* r.
[b] *Weighting factor,* n.
[c] *Heritability,* h^2; *heterozygosity,* H; *population size,* N.
[d] *Component is fitness component.*

Appendix 2. **Further information on what was measured as fitness, number of populations used in the study (populations), and number of loci and traits used in molecular markers and heritability studies (miscellaneous).**

Species	r	*Fitness*	*Populations*	*Miscellaneous**	*Reference*
Arnica montana	0.378	total fitness	14		Luijten et al. 2000
Bicyclus anynana	0.518	total fitness	15	h^2 for 5 wing color/pattern	Oosterhout 2000
Bicyclus anynana	0.860	total fitness	15	7 allozyme loci	Oosterhout 2000
Bicyclus anynana	0.510	egg hatching	19	h^2 for 9 wing color/pattern	Saccheri et al. 1996
Bicyclus anynana	0.607	egg hatching	19	8 allozyme loci	Saccheri et al. 1999
Bolitotherus cornutus	−0.01	body size	65	5 allozyme loci	Whitlock 1993
Bombus terrestris	0.550	parasite prevalence	15	relatedness among colonies	Liersch & Schmid-Hempel 1998
Bufo calamita	0.572	survival and larval size	6	25 microsatellite loci	Rowe et al. 1999
Drosophila melanogaster	0.369	total fitness	10	h^2 for 2 bristle counts	Gilligan 2001
Drosophila melanogaster	0.173	total fitness	10	4 allozyme loci	Gilligan 2001
Drosophila melanogaster	0.388	total fitness	38	h^2 for fitness	D. H. Reed, unpublished
Gentiana pneumonanthe	0.268	seed weight	10	7 allozyme loci	Oostermeijer et al. 1995
Gentiana pneumonanthe	0.111	seed weight	10		Oostermeijer et al. 1995
Gentianella germanica	0.69	seed no. and flower no.	11	23 RAPD phenotypes	Fischer & Matthies 1998*a*
Gentianella germanica	0.60	total fitness	23		Fischer & Matthies 1998*a*
Ipomopsis aggregata	0.303	seed mass and germination %	10		Heschel & Paige 1995
Limnanthes floccosa	0.738	seed set	8		Dole & Sun 1992
Lychnis viscaria	−0.062	germination % and seed mass	11	6 allozyme loci	Lammi et al. 1999
Lychnis viscaria	−0.208	germination % and seed mass	11		Lammi et al. 1999
Musca domestica	0.60	emergence %	8	h^2 for wing and scutellum length	Bryant et al. 1999
Musca domestica	0.88	emergence %	31	2 allozyme loci	Bryant et al. 1999
Musca domestica	0.731	total fitness	16	h^2 for clutch size	Reed 1998
Musca domestica	0.365	total fitness	16	2 allozyme loci	Reed 1998
Nassella pulchra	0.170	seed biomass	10	8 banding phenotypes	Knapp & Rice 1998
Peromyscus maniculatus	0.73	parasite load	9	8 allozyme loci	Meagher 1999
Petrogale lateralis	0.998	with young %	3	10 microsatellite loci	Eldridge et al. 1999
Picea glauca	0.173	tree height at 19 years	22	6 allozyme loci	Furnier et al. 1991
Rana temporaria	0.008	survivorship	13	19 allozyme loci	Hitchings & Beebee 1997
Rana temporaria	−0.024	survivorship	13		Hitchings & Beebee 1997
Salvia pratensis	−0.183	flowering % and surviving %	4		Ouborg & Van Treuren 1995
Senecio integrifolius	0.897	seed set and germination %	6		Widén 1993
Silene regia	0.492	germination %	23		Menges 1991
Swainsona recta	−0.241	germination % × survival	5	7 allozyme loci	Buza et al. 2000
Swainsona recta	0.208	germination % × survival	5		Buza et al. 2000

**Heritability,* h^2*; RAPD, random amplified polymorphic DNAs.*