New Answers for Old Questions: The Evolutionary Quantitative Genetics of Wild Animal Populations

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Abstract

Recent years have seen a rapid expansion in the scope of quantitative genetic analyses undertaken in wild populations. We illustrate here the potential for such studies to address fundamental evolutionary questions about the maintenance of genetic diversity and to reveal hidden genetic conflicts or constraints not apparent at the phenotypic level. Trade-offs between different components of fitness, sexually-antagonistic genetic effects, maternal effects, genotype-by-environment interactions, genotype-by-age interactions, and variation between different regions of the genome in localized genetic correlations may all prevent the erosion of genetic variance. We consider ways in which complex interactions between ecological conditions and the expression of genetic variation can be elucidated, and emphasize the benefits of conducting selection analyses within a quantitative genetic framework. We also review potential developments associated with rapid advances in genomic technology, in particular the increased availability of extensive marker information. Our conclusions highlight the complexity of processes contributing to the maintenance of genetic diversity in wild populations, and underline the value of a quantitative genetic approach in parameterizing models of life-history evolution.

1. INTRODUCTION

Quantitative genetics has an ugly name, but it is a subject that it is integral to an understanding of most evolutionary processes. Evolutionary change within a population requires that any changes in the distribution of phenotypes within a generation—typically due to natural selection—be passed on to subsequent generations via a genetic basis. Quantitative genetics is simply the study of this genetic basis where the trait of interest is quantitative and likely to be influenced by multiple genes and environmental factors, which is probably the great majority of traits in which we might be interested. It may therefore appear heavily descriptive, centered on questions such as, "How much genetic variance?" or "Which genes?". However the answers to these questions underpin a range of important issues in evolutionary biology. In particular, Darwinian natural selection has great intuitive appeal, but presents a paradox: If only the fittest survive, why do less fit genotypes persist? What maintains genetic variance in the face of continuing erosion by natural selection? Is there heritable genetic variance for fitness? What role do trade-offs play in the evolution of life histories? Such questions have widespread implications for all aspects of evolutionary biology (and many aspects of ecology), and addressing them requires knowledge of the genetic architecture underlying phenotypic diversity (Barton & Turelli 1989).

In this review, we focus on insights drawn from quantitative genetic studies of wild animal populations inhabiting natural environments, an area in which there has been much recent activity (Roff 2007). The drive to explore genetic architecture and microevolution in wild populations has a dual motivation. First, much of evolutionary ecology rests on a theoretical foundation provided by classical quantitative genetics, although this is not always realized and critical underlying assumptions frequently remain untested (see discussion in Owens 2006). Phenotypic patterns of covariance are unlikely to provide an accurate representation of underlying genetic patterns, and it is becoming increasingly clear that researchers interested in the evolution of life histories need to delve deeper than consideration of phenotypic associations. Second, although it is undoubtedly easier to explore genetic effects in artificial (domestic or laboratory) populations experiencing controlled conditions, increasing evidence for the impact of environmental conditions on evolutionary trajectories suggests that extrapolation from such studies to a more general evolutionary context may be limited. In this review, to make the case for wild quantitative genetics, we outline recent results supporting these assertions. Returning to the question of what maintains genetic diversity, we show how studies of the quantitative genetics of wild populations have revealed trade-offs that are either not apparent or not estimable at the phenotypic level, and explore factors that contribute to the maintenance of quantitative genetic variance in natural environments.

At a practical level, the increasing interest in the study of quantitative genetics in wild, rather than agricultural or laboratory, study populations has been greatly facilitated by recent methodological advances. On the molecular genetic front, faster and cheaper technology has rapidly accelerated the collection of genetic data with which to determine parentage and hence build pedigrees (Pemberton 2008), or to map individual loci (Slate 2005). On the statistical front, the increased application of more complex mixed model pedigree analyses, specifically the "animal model," has allowed detailed interactions of genetic and environmental effects to be modelled (Kruuk 2004; Nussey et al. 2007). Many long-term studies of wild populations for which pedigree information are available are now exploring the numerous different avenues provided by such data. However, quantitative genetics studies do not require long-term pedigree information—for example, some of the most elegant studies of evolutionary genetics have come from single generation experimental designs (see review in Merilä & Sheldon 2001).

We begin this review with a brief introduction to the relevant methodology (Section 2). In Sections 3–5, we consider evidence for genetically-based trade-offs either between different

components of fitness, or via sexually-antagonistic genetic effects, maternal effects, genotype-environment or genotype-age interactions. In Section 6 we consider the use of a quantitative genetic approach to estimate selection, and in Section 7 we explore potential insights from genomic data. Finally, we discuss the application of the breeder's equation to natural populations (Section 8).

2. METHODOLOGY

A quantitative genetic analysis requires that individuals in a population be measured for one or more phenotypic traits of interest, and that information on the relatedness among those individuals also be available (Falconer & Mackay 1996, Lynch & Walsh 1998). Relatedness, or pedigree, information may be constructed from behavioral observations or from genetic marker data (Pemberton 2008). Phenotype and pedigree data can then be combined in a statistical model to estimate key quantitative genetic parameters such as the magnitude of additive genetic variance (V_A) underlying a trait, its heritability (h^2, the ratio of V_A to total phenotypic variance V_P), and genetic correlations or covariances with other traits. The simplest approaches consider the covariance in the phenotype of parents and offspring (parent-offspring regression), or of groups of siblings (full or half-sib analyses of variance: for details see Falconer & Mackay 1996, chapter 10); these approaches have been adopted by numerous studies to estimate heritability in natural populations (starting with Boag & Grant 1978). Where information on several different types of relative is available, a more powerful approach is to consider covariance between multiple sets of relatives simultaneously, using a form of linear mixed model known as an animal model that includes as a random effect a polygenic additive genetic effect with variance-covariance structure determined by the additive genetic variance and the pedigree (Henderson 1976; Lynch & Walsh 1998, chapters 26 and 27). The animal model approach also allows other components of the phenotypic variance such as common environment or maternal effects to be modelled explicitly, and has become increasingly popular in studies of wild populations in the last decade (Kruuk 2004, Kruuk & Hadfield 2007). Parameter estimation is then typically completed via restricted maximum likelihood; to date, Bayesian approaches have been relatively underexploited in evolutionary biology, but this is changing (O'Hara et al. 2008).

At the individual level, the animal model can also estimate breeding values, or an individual's additive genetic merit, for a given trait (Falconer & Mackay 1996, p. 114). Estimated breeding values (EBVs) can be used, first, to test for temporal trends in underlying genotypes (genetic trends) that may differ from those at the level of the phenotype, or to compare associations between EBVs and fitness as an indication of the genetic basis to selection (Kruuk 2004, Postma 2006). These possibilities are enticing but, as we discuss below, recent work has emphasized the need for caution in their interpretation.

Alternative approaches are available to derive quantitative genetic estimates directly from combinations of phenotypic and genetic marker data, with no (or only limited) explicit pedigree information (Mousseau et al. 1998, Ritland 2000, Thomas 2005). However, despite the great appeal of being able to side-step the need for pedigree information, the uptake of these approaches has been limited, probably owing to reservations about their accuracy (Coltman 2005, Garant & Kruuk 2005). Wild quantitative genetic analyses therefore remain dominated by populations for which some form of pedigree data is available, even if it is acknowledged that there may be some error in pedigree links (Pemberton 2008). These studies typically involve either long-term pedigrees or the use of an experimental approach such as cross-fostering to separate out environmental sources of similarity between relatives from those due to genetic effects (or, ideally, both; Kruuk & Hadfield 2007). The development of a Bayesian approach to incorporate behavioral and spatial

information into the assignment of parentage and allow simultaneous estimation of quantitative genetic parameters should also provide more efficient analyses (Hadfield et al. 2006b). We also consider insights from genomic approaches to understanding quantitative variation, specifically mapping quantitative trait loci (QTL) and loci controlling single locus traits and the anticipated arrival of very high-density marker coverage using single nucleotide polymorphisms (SNPs). We focus on empirical results from studies of wild animal populations living in natural environments, in which mating patterns, food availability, and mortality are all naturally determined.

3. REVEALING TRADE-OFFS: THE USE OF MULTIVARIATE QUANTITATIVE GENETICS

3.1. Life-History Trade-Offs and Genetic Correlations

The existence of trade-offs between different components of fitness is fundamental to much of life-history theory. If these constraints are to affect evolutionary processes, which evolutionary ecologists typically assume they will, they must have a genetic basis, requiring the action of either antagonistic pleiotropy or linked genes with antagonistic effects, and the existence of genetic correlations between the traits in question (Lande 1982, Roff & Fairbairn 2007). However, despite the wealth of studies exploring life-history variation in natural populations, and the central role that life-history constraints play for fields such as behavioral ecology (Owens 2006), much of our understanding of the relationships between different components of fitness or between a trait and fitness stems from analyses of phenotypic associations. For example, even estimates of genetic correlations underlying one of the most fundamental trade-offs in life history, that between offspring quality and quantity, are available for only a handful of taxa in wild populations: red squirrels (McAdam & Boutin 2004), Soay sheep (Wilson et al. 2005a), great tits (Garant et al. 2008), and lizards (Sinervo 2000).

This reliance on phenotypic data is only justifiable if phenotypic correlations are assumed to provide a sufficiently accurate estimate of genetic correlations, as has been suggested by some researchers (Cheverud 1988, Roff 1996). However other studies have recommended caution regarding this assumption (e.g., Hadfield et al. 2007, Willis et al. 1991). The phenotypic correlation between two traits arises from both genetic and environmental sources of covariance:

$$r_p = r_g\, h_x\, h_y + r_e\, e_x\, e_y,$$

where r_p is the phenotypic correlation between traits x and y, r_g is their additive-genetic correlation, h_x and h_y are the square roots of their heritabilities, r_e is their environmental correlation, and e_x and e_y are the square roots of $(1 - h_x^2)$ and $(1 - h_y^2)$, respectively (Willis et al. 1991). Any correspondence between r_p and r_g will therefore depend heavily on the heritabilities of the two traits, and on their environmental covariance, all of which are likely to vary with environmental heterogeneity. It therefore seems likely that the correspondence between phenotypic and genetic correlations will be lower in natural populations experiencing less constant environmental conditions than in populations reared under controlled laboratory or agricultural populations. Furthermore, where variation between individuals occurs in both allocation and acquisition of resources, a trade-off in allocation (generating a negative genetic correlation between traits) may be masked by a positive covariance in acquisition of limited resources (de Jong & van Noordwijk 1992, van Noordwijk & de Jong 1986). As a corollary, it may even be that when resources are particularly limited, there should be more variation in resource acquisition, potentially increasing the environmental covariance between traits and causing the phenotypic correlation to diverge from the genetic correlation even further.

Following Roff (1996), we compared published estimates of phenotypic and genetic correlations in natural populations to test how well the phenotypic correlation predicted the genetic correlation. **Figure 1** shows estimates of 282 phenotypic r_p and genetic r_g correlations from 24 studies; because of the small number involved we consider largely descriptive statistics (details of the studies are given in **Supplemental Table 1**; follow the **Supplemental Material link** from the Annual Reviews home page at **http://www.annualreviews.org**). Across all pairwise combinations, the correlation between estimates of genetic and phenotypic correlations was high ($r = 0.735$, $p < 0.001$). The slope of a regression of r_g on r_p was also not different from unity (1.059 ± 0.063SE), and we found no evidence of any difference in the correspondence in correlations between morphological traits versus life-history traits, although the latter were more poorly represented (see legend of **Figure 1** for details; cf. Roff 1996). However, there was considerable disparity between individual

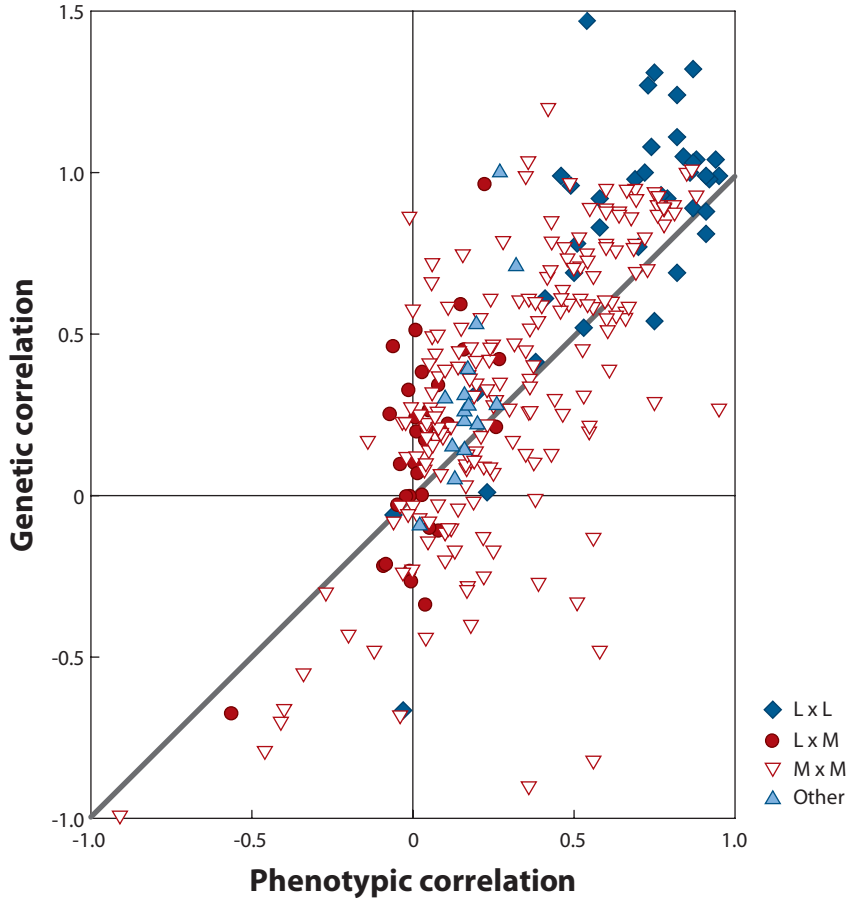

Figure 1

Regression of phenotypic correlations (r_p) on genetic correlations (r_g): estimates from 282 estimates from 24 studies of natural populations. Diagonal line shows 1:1 relationship. Correlations are classified according to whether traits are morphometric (M), life history (L), or other (specifically behavioral or parasite resistance). Results from linear mixed model of r_g regressed on r_p with study as a random effect: intercept $= 0.031 \pm 0.042$SE; slope $= 1.059 \pm 0.063$SE ($F = 280.0$, d.f. $= 1, 277.1$; $p < 0.001$). There was no evidence of any difference between classes (L × L, L × M, M × M or Other) in the ability of r_p to predict r_g (interaction term: $F = 1.52$, d.f. $= 3, 271.7$; $p = 0.210$). See **Supplemental Table 1** for details of studies and traits.

phenotypic and genetic correlations: the absolute disparity ($D = |r_p - r_g|$, Willis et al. 1991) had a mean value of 0.245 (SD 0.222, n = 282) across all comparisons, and was on average equal to 112% of the magnitude of genetic correlation. Genetic correlations were larger in absolute magnitude in 78% of cases where the two were of the same sign (188 out of 241), but it was also possible for substantial phenotypic correlations to have no genetic basis (**Figure 1**).

There are several interesting aspects of these data that could be explored further, but in relation to the reliability of phenotypic correlations, they suggest that estimates of r_p are not a very accurate indicator of individual r_g. The lack of correspondence could conceivably reflect measurement error in the estimates of genetic correlations. However, if the disparity D between r_p and r_g were largely due to measurement error in r_g, we would expect D^2 to be roughly equal to the square of the standard error of r_g (Roff 1996). In these studies, D^2 was significantly greater than the square of the estimated standard errors [for individual estimates, median $D^2 = 0.035$ (IQR 0.009 – 0.090), median $\text{EstSE}^2 = 0.010$ (IQR 0.002 – 0.037); Wilcoxon paired test statistic = 12852.0, n = 279, $p < 0.001$; taking averages per study: Wilcoxon statistic = 14.00, n = 24, $p < 0.001$]. There was also no indication of any association between a study's average D^2 and its average sample size (defined as the number of individuals: correlation = –0.110, n = 24, p = 0.612). These data from natural populations therefore seem to show a stronger indication than Roff's (1996) survey that differences between genetic and phenotypic correlations cannot be entirely explained by sampling error. Note also that where genetic and environmental correlations have been estimated explicitly it is possible for the two to have very different values (e.g., Boag 1983, Robinson et al. 2008b, Wilson et al. 2005a), or even entirely contrasting values (e.g., Hadfield et al. 2007). Finally, in the likely event that estimates of genetic associations are inflated by relatives sharing common environments, estimates of genetic correlations may well be biased by environmental correlations (Kruuk & Hadfield 2007); as a result, true underlying genetic correlations may be even more different from phenotypic correlations than the estimated values suggest.

In further support of the need to consider genetic rather than phenotypic relationships, parameterization of some life-history models requires genetic correlations that cannot be approximated by phenotypic correlations. For example, models of sexual selection and the role of indirect benefits of female choice invoke genetic correlations between traits expressed in different generations, specifically between male trait and offspring fitness, and in different sexes, specifically between male trait and female preference (Kirkpatrick & Barton 1997). Estimates of the relevant parameters are rare for wild populations, presumably in part because assessing female preferences in a nonexperimental setting is difficult. However, recent studies have found no evidence for the necessary positive genetic correlation between male trait and offspring fitness in either a cross-fostering study of blue tits (*Cyanistes caeruleus*, Hadfield et al. 2006a), or an analysis of long-term pedigrees from a collared flycatcher (*Ficedula albicollis*) population (Qvarnström et al. 2006). Furthermore, using an indirect measure of female preference, the latter study also found only a relatively low and nonsignificant correlation between male trait and female preference (Qvarnström et al. 2006). In contrast, there was a significant genetic correlation between male trait (tail length) and provision of direct benefits (nest size) in swallows (Møller 2006). Combined, these studies possibly suggest a need for a revaluation of the relative role of direct versus indirect benefits in mate choice.

Proving the existence of the evolutionary trade-offs that are central to life-history theory therefore requires evidence of antagonistic genetic associations. **Figure 2** contains a schematic outlining two slightly different scenarios that may generate antagonistic genetic associations. We present a general framework that applies to tests for trade-offs between two traits expressed in two different environments, but the arguments apply to simply testing for trade-offs between two different traits (treat environment 1 = environment 2 and ignore the genetic correlation between fitness in different environments), or, as we discuss later, to the same trait in different environments.

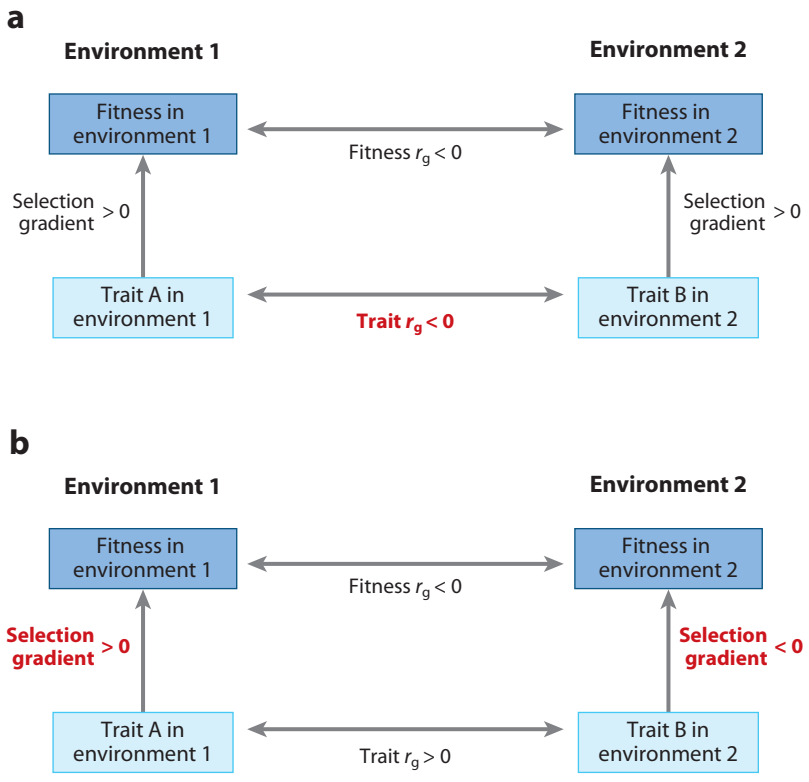

Figure 2

Schematic showing alternative scenarios generating evolutionary trade-offs between two traits expressed in different environments. "Environment" may refer to different environments, different genders, different generations, or different ages. A negative genetic correlation (r_g) between fitness in two environments can arise via two alternative scenarios: (*a*) antagonistic pleiotropy (or antagonistic linkage disequilibrium) generates a negative genetic correlation between two traits A and B, both of which are under positive selection pressures; (*b*) traits A and B have a positive genetic correlation, but antagonistic selection pressures. For generality, we illustrate two traits A and B, but note that this is easily simplified to apply to a single trait expressed in the two different environments. Highlighted text (in *red*) indicate the cause of the antagonistic effects under the two scenarios.

Thus genetically-based trade-offs require either negative genetic correlations between any two phenotypic traits that are selected in the same direction (**Figure 2*a***) or contrasting selection regimes acting on two traits that are positively genetically correlated (**Figure 2*a***). In the following sections, we refer to this framework in relation to tests for trade-offs between traits expressed in different genders, generations, environments, and finally ages.

3.2. Sexually Antagonistic Genetic Variance and Cross-Sex Genetic Correlations

Sexually-antagonistic gene expression is likely to play an important role in the maintenance of genetic diversity, as well as having implications for the indirect benefits models of sexual selection mentioned above. Experimental studies have reported intersexual genetic conflicts in fitness or fitness components under laboratory conditions: for example, in *Drosophila* (Chippindale et al. 2001, Rice & Chippindale 2001) and crickets (Fedorka & Mousseau 2004). It seems likely that the

potential for sexually-antagonistic effects is especially high in polygynous species with pronounced sexual dimorphism, in which reproductive roles differ greatly between the sexes, and tests for sexually-antagonistic genetic variance in wild populations of such taxa are just emerging.

In wild populations, estimates of additive genetic variance for a range of traits differ between the sexes, suggesting substantial sexual differences in genetic architecture (e.g., Brommer et al. 2007, Coltman et al. 2005, Jensen et al. 2003). Perhaps surprisingly, however, there has been relatively little work to date on cross-sex correlations in any trait, let alone in fitness, in natural populations, despite the critical role they play in determining the evolution of any trait with sexual dimorphism (Lande 1980). From the data available, cross-sex genetic correlations in natural populations are high (close to unity) for standard morphological traits (**Table 1**), in accordance with reviews of

Table 1 Estimates of cross-sex, within-trait genetic correlations published in wild populations, for (*a*) traits under natural selection, (*b*) secondary sexual traits, and (*c*) measures of fitness

Cross-sex genetic correlations			
Species	**Trait**	**Correlation (SE)**	**Reference**
(*a*) Traits under natural selection			
Collared flycatcher (*Ficedula albicollis*)	Tarsus length	1.00 (0.22)	(Merilä et al. 1998)
House sparrow (*Passer domesticus*)	Tarsus length	1.00 (0.00)	(Jensen et al. 2003)
	Wing length	0.89 (0.24)	
	Bill depth	0.76 (0.35)	
	Bill length	0.98 (0.19)	
	Body mass	1.00 (0.00)	
	Body condition	0.69 (0.46)	
Bighorn sheep (*Ovis canadensis*)	Body mass	0.63 (0.30)	(Poissant et al. 2008)
Soay sheep (*Ovis aries*)	Body mass	0.79 (0.25)	(Robinson et al. 2008b)
	Parasite resistance	0.84 (0.27)	
Red deer (*Cervus elaphus*)	Birth mass	0.90 (V_A),	L. Kruuk, unpubl. data
		0.98 (V_{MG}[a])	
	Jaw length	1.00 (0.00)	
(*b*) Secondary sexual traits			
Bighorn sheep (*Ovis canadensis*)	Horn volume	0.24 (0.28)	(Poissant et al. 2008)
Soay sheep (*Ovis aries*)	Horn length[b]	0.49 (0.20)	(Robinson et al. 2008b)
Swallow (*Hirundo rustica*)	Tail length	0.51 (0.16)	(Møller 1993)
(*c*) Fitness measures			
Collared flycatcher (*Ficedula albicollis*)	Annual RS[c]	0.23 (0.35)	(Qvarnström et al. 2006)
	Lifetime RS[d]	–0.85 (0.59)	(Brommer et al. 2007)
Red deer (*Cervus elaphus*)	Annual fitness p_{ti}[e]	–0.95 (0.42)	(Foerster et al. 2007)
	Annual reproduction[f]	–1.38 (0.42)	
	Annual survival[f]	–0.45 (0.89)	
	Lifetime RS[d]	–0.48 (0.44)	

[a]Maternal genetic effects; SEs not available.
[b]Normal-horned individuals only.
[c]Annual reproductive success.
[d]Lifetime reproductive success.
[e]Using "delifed" fitness, p_{ti}: annual fitness incorporates reproduction and survival (Coulson et al. 2006b).
[f]Annual reproduction and survival component of delifed fitness p_{ti} (Coulson et al. 2006b).

estimates from artificial populations (J. Poissant, unpublished data; Roff 1997, chapter 7). For secondary sexual traits, there is a suggestion that cross-sex correlations are lower and more likely to be significantly less than unity (**Table 1**). Furthermore, evidence is emerging for significantly negative cross-sex genetic correlations for fitness, or components of fitness, in some systems (**Table 1**). Although substantially more results are required to confirm these patterns, there are clear indications of the existence of sexually-antagonistic genetic variance in natural populations.

In addition to knowledge of the cross-sex genetic architecture, a full understanding of the sex-specific selection pressures acting on focal traits is a critical component of tests for sexually-antagonistic effects. As with the conflicts described above, the potential for evolutionary conflict between the sexes can manifest itself in different ways: either through antagonistic cross-sex genetic correlations (within or between traits) for traits that are positively correlated with fitness (**Figure 2*a***, with "environment" now referring to gender), or through opposing selection pressures (**Figure 2*b***); both scenarios result in negative genetic correlations between male and female fitness, indicating intralocus conflict within genes affecting fitness (Rice & Chippindale 2001). The scenario outlined in **Figure 2** can refer either to the same trait expressed in each sex or to different traits (e.g., a male secondary sexual trait and female fecundity). Negative cross-sex genetic correlations for traits such as morphology seem unlikely, but under a more realistic scenario, there may be antagonistic pleiotropic effects on different traits each under positive selection in the two sexes: for example, it is conceivable that certain alleles would be associated with enhanced male fighting success but reduced female fecundity due to their effects on levels of hormones such as testosterone. However data on cross-trait, cross-sex genetic correlations are even scarcer (though see Poissant et al. 2008, Qvarnström et al. 2006).

To our knowledge, no studies to date have provided information on the complete suite of genetic correlations and selection pressures outlined in **Figure 2**, but recent work has investigated cross-sex genetic architecture and selection pressures affecting the secondary sexual traits of horns in two wild sheep populations. In bighorn sheep (*Ovis canadensis*) inhabiting the Canadian Rocky Mountains, although horn size showed significant V_A in both sexes, there is no evidence that either cross-sex genetic correlations or current selection pressures are different from zero (Poissant et al. 2008). In a feral population of Soay sheep (*O. aries*) on St Kilda, Scotland, there is a positive cross-sex genetic correlation in horn length among normal-horned individuals (Robinson et al. 2008b), but also a suggestion of sexually-antagonistic selection pressures (Robinson et al. 2006), reflecting the scenario in **Figure 2*b***.

In summary, analyses of sexually-differentiated genetic architecture in wild populations have provided interesting suggestions of the existence of sexually-antagonistic effects. However empirical results are still extremely scarce, and important questions regarding the genetic architecture of sexually-differentiated traits remain to be explored, for example in relation to sex linkage (Kirkpatrick & Hall 2004).

3.3. Maternal Effects

Genetic trade-offs may also occur across generations. In particular, maternal effects—the impact of a mother's phenotype on the phenotype of her offspring, over and above the direct effects of the genes they inherit from her—have the potential to introduce evolutionary constraints, as optimal levels of maternal investment are likely to differ between mothers and offspring. If there are antagonistic covariances between maternal and offspring genetic effects on a trait (for example, if genes with a direct effect of causing large body size in an individual also, on average, cause relatively poor maternal performance, perhaps in terms of in utero investment or lactation), these will dampen any response to selection on the trait (Kirkpatrick & Lande 1989) and hence have the

potential to contribute to the maintenance of genetic variance. A genetic basis to many maternal effects has been demonstrated in many animal breeding and plant breeding studies (e.g., Thiede 1998, Wilson & Réale 2006). Studies of nondomestic animal species have more often treated maternal effects as purely environmental sources of variation, but evidence is accumulating for a significant heritable genetic basis to a range of different maternal effect traits in wild populations (reviewed in Råsånen & Kruuk 2007). This genetic basis to maternal effects provides ample potential for genetic trade-offs via antagonistic pleiotropic effects on both offspring and maternal traits (or the equivalent linkage disequilibrium). Several recent studies of wild populations have adopted quantitative genetics analyses to elucidating the evolutionary significance of maternal effects.

Analyses of maternal effects may focus entirely on the phenotype of the offspring, and define a trait of "maternal performance" as the average impact of an individual mother on her offspring (following Wilham 1972). Taking this approach, large amounts of data on the covariance between offspring trait and maternal performance, defined as the direct-maternal genetic covariance, are available: a review of cattle and sheep data concluded that these are generally negative, though concerns about effects of statistical artifacts remain to be explored (Wilson & Réale 2006). However, similar estimates from nondomestic species are still scarce and inconclusive: an estimate of the direct-maternal genetic correlation for offspring body mass is positive in red squirrels (McAdam et al. 2002), but negative (though nonsignificant) for offspring birth weight and birth date in a feral population of Soay sheep (Wilson et al. 2005a).

An alternative approach is to more explicitly consider two separate traits measured in both offspring and mothers, with the maternal trait having a quantifiable impact on the offspring trait (following Kirkpatrick & Lande 1989). Thus maternal provisioning and offspring elicitation of food show a positive genetic correlation in great tits (Kölliker et al. 2000), but there was a negative genetic correlation between maternal body mass and relative maternal expenditure, assessed as the ratio of offspring to maternal mass, in bighorn sheep (Réale & Festa-Bianchet 2000). Despite the vast body of work that has been conducted on determinants of laying date (which can be considered as a maternal effect) in wild bird populations, we could not find estimates of genetic correlations between it and offspring traits in the literature; as mentioned above, estimates of genetic correlations between avian clutch size and offspring size are similarly rare (though see Garant et al. 2008).

Thus, although generalizations as to the nature of the impact of maternal effects in wild populations are not yet possible, results available to date indicate that maternal effects may have a substantial genetic basis and that, in some cases, there is evidence for antagonistic pleiotropy. Again, however, in the context of identifying trade-offs (**Figure 2**), a full understanding of patterns of selection acting on different generations is also required. Theoretically, maternal effects can have a substantial impact on evolutionary trajectories (Kirkpatrick & Lande 1989), but surprisingly little attempt has yet been made to fully parameterize these models using data from natural environments (Råsånen & Kruuk 2007, although see McAdam & Boutin 2004). We discuss maternal effects further below in relation to their interaction with environmental conditions.

4. GENOTYPE-BY-ENVIRONMENT INTERACTIONS

Quantitative genetic models typically assume that genetic and environmental effects contribute additively to an individual's phenotype. However, it is also well established that genetic and environmental effects can interact such that the effect of any given environment on the phenotype is itself dependent on an individual's genotype (Barton & Turelli 1989). These genotype-by-environment (G×E) interactions have been extensively demonstrated in laboratory experiments

(for recent examples, see Danielson-Francois et al. 2006, Valdar et al. 2006), and in studies of livestock and aquaculture species in a wide range of taxa (e.g., Maniatis & Pollott 2002, N'Dri et al. 2007). Furthermore, G×E may play a critical role in shaping phenotypic evolution in natural populations that, almost without exception, live in heterogeneous environments. This is because, in the presence of G×E, additive genetic variance for a trait of interest will vary across environmental conditions (and r_g among environments will be less than one). Equivalently, G×E means that the phenotypic response to a changing environment will differ among individuals such that phenotypic plasticity may itself be thought of as a heritable trait (discussed further below).

Most attempts to characterize G×E in wild animal populations have involved the comparison of genetic parameters for environment-specific subtraits (i.e., a single phenotypic trait measured under differing environmental conditions). This allows various questions to be addressed such as whether changing V_A facilitates faster selection responses in some conditions than others or whether evolutionarily important trade-offs occur across environmental conditions: a negative genetic correlation between performance in different environments implies the lack of a single optimal genotype (**Figure 2*a***); alternatively, a positive genetic correlation across environments may impose a trade-off if selection on a trait fluctuates in sign across those environments (**Figure 2*b***). Note that, as before, the scenario in **Figure 2** can represent either two subtraits defined as the same trait expressed in different environments or two entirely different traits.

In one of the first studies to consider environment-specific traits, Larsson (1993) found that heritabilities of body-size traits in barnacle geese (*Branta leucopsis*) were higher when growth conditions were good and not significantly different from zero under harsher conditions; genetic correlations also showed environmental sensitivity. Subsequent studies using this approach have defined discrete environments on the basis of naturally occurring heterogeneity in food abundance (Ernande et al. 2004), temperature (Uller et al. 2002), or population density (Garant et al. 2005).

G×E can also be tested for by manipulating environmental conditions in otherwise natural systems. In particular, cross-fostering experiments in passerine birds have been used in conjunction with sib-analyses to explore the impact of the nest environment on genetic parameters for size and growth traits (e.g., Kunz & Ekman 2000, and see review in Merilä & Sheldon 2001). Typically, cross-fostering is coupled with brood size manipulation such that environmental conditions in the form of feeding regime for nestlings are experimentally altered (e.g., Gebhardt-Henrich & van Noordwijk 1991, Merilä & Fry 1998). An alternative form of manipulation was recently performed by Charmantier et al. (2004), in which experimentally deparasitized broods of blue tits (*Parus caerulus*) showed higher V_A for tarsus length relative to untreated broods. It is worth considering however that estimates of G×E interactions from cross-fostering experiments have the potential to be confounded by differences in the effect of the treatment owing to other brood characteristics (i.e., Brood×E interactions, for example, owing to differences in age, however slight, between broods in a dyad; Hadfield & Owens 2006, Kruuk & Hadfield 2007).

In a recent meta-analysis of genetic parameters estimated across heterogeneous environments, Charmantier & Garant (2005) reported an emergent trend of higher heritabilities under more favorable conditions. As well as truly natural populations, this meta-analysis also included parameters from laboratory studies where animals had been wild-collected less than five generations previously. The suggestion of a difference in h^2, which was statistically significant for morphological but not for life-history traits, indicates that environmental conditions can have important consequences for predicted responses to selection. Nevertheless, because h^2 is dependent on V_P (and hence on all components of variance, not just V_A), this is not evidence of systematic and widespread G×E. A corresponding analysis of V_A was hampered by a lack of standard error reporting, although the data indicated that V_A was higher under better conditions in 65% of cases included ($n = 40$ traits; Charmantier & Garant 2005).

Environmental conditions may often show continuous, rather than discrete, variation, and an alternative approach to that outlined above lies in the use of "infinite dimensional" or "function-valued" traits (Kirkpatrick 1997), whereby an individual's phenotype is modelled as a continuous function of an environmental variable (E). With pedigree information available, the phenotype can be decomposed so that the breeding value is also modelled as a function of E, for example, in a so-called random regression animal model (Nussey et al. 2007). This technique has been widely used by animal breeders for analyses of longitudinal data (e.g., size, milk yield) with genetic effects modelled as covariance functions of age (Meyer & Kirkpatrick 2005). However, where repeated measurements are made on individual animals under differing environmental conditions, it is also a useful tool for exploring G×E in natural systems, particularly if environmental parameters of interest vary continuously (Nussey et al. 2007). For example, in Soay sheep *Ovis aries*, modeling the maternal additive genetic effect as a polynomial function of annual environmental quality (defined as the average neonatal survival) shows that genetic variance for birth weight is higher in good years (Wilson et al. 2006). Conversely, positive directional selection, which acts on birth weight through differential lamb viability, is actually weaker under good conditions, such that there is environmentally induced covariance between the strength of selection and the trait heritability (Wilson et al. 2006). In this case the covariance is negative, reducing the expected rate of phenotypic evolution (predicted as the product of h^2 and the selection differential S; Falconer & Mackay 1996, chapter 11), although there may also be instances where environmental heterogeneity induces positive covariance.

Random regression models can also be viewed as formulations of the individual reaction-norm perspective commonly used to study phenotypic plasticity (Nussey et al. 2007). For example, in the simplest case an individual's phenotype might be assumed to follow a linear reaction norm over an environmental parameter E. In this case the phenotype for any value of E can be defined by two individually-specified parameters, an intercept (interpreted as the phenotype at $E = 0$) and a slope (interpreted as plasticity). Researchers have been interested in whether there is among individual variation in plasticity and, if so, whether this has a genetic component (G×E). If the latter condition is met then plasticity can itself be thought of as a heritable trait that may respond to selection.

The question of whether or not a phenotypic trait shows G×E is therefore equivalent to asking whether plasticity in the trait has a heritable basis of variation. This perspective has recently been applied to studies of plasticity in several vertebrate populations. For example, individual phenotypic plasticity of lay date in response to spring temperature was found to be both heritable (i.e., a G×E) and under positive selection in great tits (*Parus major*), leading to the expectation that the plastic response should evolve to help alleviate the current mismatch between the timing of reproduction and peak food availability (Nussey et al. 2005). Evidence of genetic variance for seasonal plasticity in body mass has also been reported in bighorn sheep (*Ovis candensis*, Pelletier et al. 2007). In contrast, similar analyses detected no significant heritability for lay date plasticity in collared flycatchers (*Ficedula albicollis*, Brommer et al. 2005) or common gulls (*Larus canus*, Brommer et al. 2008).

It is important to note that although studies of G×E can be framed in terms of either reaction norms or environment-specific trait expression, the two approaches are in reality equivalent (Roff 1997, chapter 6). This equivalence is particularly apparent under a random regression animal model because, for a given functional form of reaction norm, the genetic covariance matrix of associated parameters (e.g., intercept and slope for a linear reaction norm) necessarily defines a corresponding **G** matrix of environment-specific traits. As a result, conclusions regarding reaction norms can equally be restated in terms of environment-specific traits: the presence of genetic variance for plasticity (i.e., reaction norm slope) necessarily means changing V_A for environment-specific traits.

The vast majority of studies of genotype-by-environment interactions in nature have considered only a single phenotypic trait (albeit often defined as a set of environment-specific subtraits for analytical purposes). However, a more complete understanding of the prevalence and evolutionary implications of G×E necessitates a multivariate approach, because genetic correlations among traits, as well as levels of V_A, are likely to change with environmental conditions (Sgrò & Hoffmann 2003). This was attempted by Garant et al. (2008), who used data from a long-term study of great tits (*Parus major*) to estimate **G** matrices for a set of three reproductive traits (lay date, clutch size, and egg mass). By partitioning data collected over 40 years into two periods, an earlier cooler period and a later warmer one, they tested for effects of global warming on genetic architecture. Estimates of **G** were similar in the two environments and the researchers consequently concluded that there was no evidence for G×E in these traits (Garant et al. 2008). The impact of environmental heterogeneity on genetic correlations has also been examined in Soay sheep, with genetic correlations among horn length, body weight, and parasite resistance tending to be lower in better environments, both within and between sexes (Robinson et al. 2008b). Furthermore, the genetic correlation between first year horn growth and lifetime fitness (a measure of selection, see below) was negative for lambs born in poor environments, but positive for those born in better conditions (Robinson et al. 2008a). This fluctuating selection arises because though high investment in horn growth yields increased breeding success, it is also associated with a short-term survival cost in this system that may not be affordable in adverse environments.

The concept that trade-offs between performance in different environments should prevent the erosion of genetic diversity is intuitively highly appealing, although there has been considerable theoretical debate over the potential for G×E interactions to maintain V_A (e.g., Gillespie & Turelli 1989, Turelli & Barton 2004). To date, empirical evidence for antagonistic G×E interactions is also limited (Roff 1997). Note that the existence of G×E is not sufficient to conclude that cross-environment genetic correlations will actually be antagonistic: G×E may simply reflect changes in the magnitude of V_A. Where they have been quantified, estimates of cross-environment correlations in natural populations are frequently positive and close to unity (e.g., Garant et al. 2003, 2008, Merilä & Fry 1998, Wilson et al. 2006). However, these studies have been largely restricted to morphological traits, and fitness or fitness components may show entirely different patterns (cf. **Table 1**). Evidence of fluctuating selection across different environmental conditions (Grant & Grant 2002, Robinson et al. 2008a) generates an expectation of negative cross-environment correlations in fitness (**Figure 2*b***), but to our knowledge these have not yet been explicitly calculated for any population in a natural environment. Given the likely complexity of such environmentally-based trade-offs, it may be that we are only now in a position to investigate them fully.

Overall, it is clear that G×E can occur in wild animal populations and that it has major implications for phenotypic evolution. However, more empirical studies are needed before we are able to assess either its ubiquity or typical effect size. G×E interactions may also have important implications for the interpretation of temporal trends in breeding values (e.g., Garant et al. 2004, Merilä et al. 2001), as their presence may be sufficient to generate a change in EBVs regardless of whether or not microevolution has occurred. Furthermore, as with other applications of quantitative genetic models, concerns exist about statistical power and potential biases in parameter estimation. We consider these two issues in more depth in the **Supplemental Discussion**.

5. GENOTYPE-BY-AGE INTERACTIONS

For many traits phenotypic distributions change as animals get older, and selection can also show systematic variation with age. Furthermore, the availability of repeated records made on individuals at different ages or ontogenetic stages allows the possibility of genotype-by-age (G×A) interactions

to be scrutinized. Age can be viewed as an intrinsic environmental variable such that G×A can be tested for using both the character state approach (analyzing age-specific traits) or the reaction norm approach by estimating V_A for parameters describing individual ontogenies (e.g., growth curve parameters). These two approaches have been used to test for changes in heritability over ontogeny (e.g., Réale et al. 1999), persistence of maternal effects into adulthood (e.g., Wilson et al. 2005b), and evolutionary constraints arising from among-age genetic correlations (Charmantier et al. 2006b).

Two types of phenotypic trait have been examined in particular. First, a large number of studies have estimated genetic (co)variance structures for size traits across ontogeny (e.g., Badyaev & Martin 2000, Björklund 1997). Second, there is growing interest in exploring the quantitative genetics of senescence by estimating genetic parameters for survival and reproductive traits (including metrics of annual fitness) in iteroparous animals (e.g., Charmantier et al. 2006b). For example, the antagonistic pleiotropy model of senescence (Williams 1957) states that genes having detrimental effects in late life will be maintained by selection if they have beneficial effects earlier. This can simply be viewed as a trade-off between the early and late life environments and leads to the prediction that negative genetic correlations should occur between fitness (or fitness components) expressed at different ages (**Figure 2*b***). The empirical results of these studies have been the subject of recent reviews to which we refer the interested reader for more details (see Charmantier et al. 2006a, Wilson et al. 2008). However, it is clear from this work that constancy of genetic parameters with age cannot and should not be assumed in many cases, and there is also some support for genetically-based trade-offs across ages. Thus, explicit consideration and estimation of G×A effects are vital to understanding the evolution of trait ontogenies.

6. USING QUANTITATIVE GENETICS TO DESCRIBE SELECTION PRESSURES

Although analyses of selection typically focus on within-generation change, and may therefore appear independent of the genetic basis of phenotypic diversity, a quantitative genetic framework can provide a comprehensive description of selective processes not necessarily apparent at a phenotypic level. In particular, a phenotypic selection gradient relates different values of phenotype to fitness (Lande & Arnold 1983), but the resulting estimate will give a misleading expectation of cross-generational responses to selection if there are environmental variables simultaneously affecting both the trait of interest and fitness (Fisher 1958, p. 138; Price et al. 1988; Rausher 1992). Under such conditions, fitness differences may be largely associated with only the environmental component of the trait. A quantitative genetic approach then becomes essential for disentangling the impact of unmeasured environmental variables or "invisible traits" (Hadfield 2008). This may be done by comparing selection gradients on the phenotypic values with those associated with some measure of genotypic value (Rausher 1992), for example family means or, as in several recent studies of pedigreed wild animal populations, using estimated breeding values (e.g., Kruuk et al. 2002, Sinervo & McAdam 2008; see review in Postma 2006). A more direct approach is to compare phenotypic and genetic covariances between fitness and the trait (van Tienderen & de Jong 1994). This is also more robust, as there may be substantial differences between true and estimated breeding values (Postma 2006): Hadfield (2008) has shown that the use of individual estimated breeding values from pedigrees typical of wild populations can generate severely biased estimates of genotypic-level selection. The bias is avoided if the invisible trait implications are evaluated from the genetic correlation between the trait and fitness in a multivariate quantitative genetic analysis (Hadfield 2008). A related issue is the "invisible fraction": those individuals who die before a trait is measured or expressed (Grafen 1988). If this mortality is in any way nonrandom

in relation to genes affecting the trait of interest, selection affects the distribution of the trait in a manner that cannot be assessed at the phenotypic level, and estimates of selection are biased unless the missing data process is modelled explicitly (Hadfield 2008). The genetic component of the invisible fraction can be at least partially assessed from the phenotypes of surviving relatives, such that the genetic correlation between the trait in question and survival prior to the trait can provide key information with which to model the missing data mechanism (Hadfield 2008).

To date, to our knowledge, two studies have presented both selection gradients on breeding values and genetic correlations with fitness. Considering antler size in red deer, Kruuk et al. (2002) tested for environmental covariance inflating phenotypic selection estimates and reached the same conclusions from both approaches: estimates of phenotypic selection on antler size are increased by an environmental covariance between antler size and fitness (the invisible trait). Sinervo & McAdam (2008) used data from side-blotch lizards to investigate selection on the invisible fraction, considering selection via juvenile mortality prior to reproduction in both males and females in relation to clutch size, a trait expressed only in females. Interestingly, in this system, selection is sexually-antagonistic (see above), with genetic associations between clutch size and juvenile survival being positive in females and negative in males. In both cases, the breeding value approach involved much lower standard errors—making it dangerously appealing, but also reflecting the misleading aspect that selection gradients on EBVs ignore both the uncertainty associated with their prediction and their nonindependence between relatives. However, as we discuss further in the **Supplemental Discussion**, there may also be problems associated with calculating genetic correlations between a trait and fitness when the latter shows non-normal distributions.

Finally, note that analyses of selection necessarily require estimates of fitness, and that the questions of how best to measure fitness, and how best to then incorporate the resulting estimates into multivariate analyses, are also issues about which there is much ongoing debate (see **Supplemental Discussion**).

7. GENOMIC APPROACHES TO STUDYING MICROEVOLUTION

The above analyses have largely focused on evolutionary insights gained from combining phenotypic data with pedigree information. However, understanding microevolutionary processes in natural populations should, in principle, be greatly aided by identifying the genes or genomic regions that explain fitness variation. By typing a panel of molecular markers, quantitative trait loci or QTL can be mapped (and their magnitude quantified) by identifying markers that cosegregate with variation at a focal trait. In controlled crosses, the statistical framework for mapping QTL is well established and has been applied in numerous species and settings (Andersson 2001, Mackay 2001). In wild populations, where controlled matings are impossible or undesirable, an alternative approach is to test for the presence of a QTL at a given genomic location using a mixed effects model (animal model) framework (reviewed in Slate 2005). Here the QTL effect is fitted as a random effect, in addition to the model term describing a polygenic (V_A) effect, and likelihood ratio tests are then used to compare models.

This approach was first used in a wild population to map QTL for birth weight in red deer (Slate et al. 2002). Subsequently, additional mapping studies have been reported in a feral population of Soay sheep, where a panel of microsatellite markers has been used to identify genes underlying variation in quantitative traits such as body size and birth weight (Beraldi et al. 2007b) and parasite resistance (Beraldi et al. 2007a). These studies have provided the first estimates of the genomic location and phenotypic magnitude of QTL, but it is questionable to what extent they

have as yet provided new insights into microevolutionary processes. To a large degree the early mapping studies are descriptive (asking, "How many loci?", "What magnitude are their effects?"). Estimating the distribution of QTL effects is of fundamental interest in evolutionary genetic research, but from wild populations there are currently insufficient data to use approaches described elsewhere, which take account of the fact that only the largest QTL can be estimated and that these estimates may be upwardly biased (Hayes & Goddard 2001, Otto & Jones 2000). Further studies (possibly including typing an independent set of animals to confirm QTL) are needed to establish whether these loci have consistent effects in different environments, whether they are responsible for genetic correlations, and whether they are responding to selection. In principle genetic correlations and G×E at specific QTL can be estimated in exactly the same way as outlined above for polygenic variation.

Attempts to map genes for single-locus traits have been more successful in Soay sheep with genes controlling coat color, coat pattern, and horn type polymorphisms mapped (Beraldi et al. 2006) and, in the case of coat color, the causative mutation identified (Gratten et al. 2007). Subsequent typing of the coat color mutation in ~2500 sheep has suggested that an unexpected decline in the frequency of dark-coated sheep is explained by close linkage (and linkage disequilibrium) between loci affecting body size and lifetime fitness within the vicinity of the coat color gene (Gratten et al. 2008). In this instance, the local genetic correlation between body size and fitness was negative, whereas overall phenotypic selection on body size was positive. In other words, a molecular approach was required to dissect the genetic correlation in this specific genomic region, which in turn could explain the counterintuitive decline in the frequency of dark coats.

It is likely that mapping studies will soon be conducted in other wild vertebrates. Linkage maps of complete or partial genomes have recently been reported for pedigreed wild populations of collared flycatchers (Backström et al. 2006) and great reed warblers (Hansson et al. 2005), and we are aware of similar efforts being carried out in bighorn sheep, great tits, and song sparrows. Until recently, microsatellites have been the marker of choice, often exploiting resources generated in model or economically important organisms closely related to the focal organism (e.g., domestic cattle markers have been used to build maps in wild ruminants). One of the most exciting developments in recent years has been the development of massively parallel high-throughput genome sequencing (Margulies et al. 2005). It is now feasible to generate hundreds of thousands, or even millions, of DNA sequences rapidly and cost effectively. These can be exploited to mine single-nucleotide polymorphisms (SNPs), which in turn can be typed at a large scale (thousands of markers) at a considerably lower (10–100 fold) cost per genotype than microsatellites. Effectively, any wild vertebrate population can now be mapped with SNPs at very high-density marker coverage. A preliminary analysis of domestic sheep SNPs indicates that two thirds of markers are also segregating in Soay sheep. Therefore, the 60K sheep SNP chip currently being assembled by the International Sheep Genomics Consortium (**http://www.sheephapmap.org**) will result in a platform by which 40,000 SNPs can be typed in Soay sheep. What does this mean for studies of microevolution in wild populations?

First, the much higher marker density that is now feasible means that it will soon be possible to identify markers that are in linkage disequilibrium (LD) with most of the genes that explain fitness variation. Therefore, it might be possible to track changes in allele frequency (and to evaluate the relative effects of selection and drift). Second, markers known to be in LD with causative mutations can be modeled as fixed effects, which will make testing for dominance deviations (i.e., dominance genetic variance), G×E, and G×G (epistasis) easier. One cautionary note, however, is that very large sample sizes (possibly thousands rather than hundreds of individuals) may be required to distinguish markers that are truly in LD with fitness loci from Type I errors.

High-density marker scans may also facilitate alternative approaches to estimating breeding values (Meuwissen 2007). If the loci underlying trait variation are in LD with a marker, then in theory it is possible to estimate individual breeding values from marker genotypes alone (Meuwissen et al. 2001). So-called genomic selection (GS) methods for estimating breeding values have attracted considerable attention in the animal breeding literature (summarized by Meuwissen 2007 and addressed by other papers in the same issue of *Journal of Animal Breeding & Genetics*), as it is thought they may lead to faster rates of animal improvement than do conventional methods. GS-EBV estimation is a two-stage process: Markers in LD with causative loci are identified in stage 1 and then are used in a different set of individuals to estimate breeding values (stage 2). In the same way that animal breeders introduced the animal model to evolutionary biologists, the GS-EBV approach may cross over to applications in wild populations. In this way, one of the limitations of gene mapping studies in the wild—the inability to predict breeding values—may be overcome. Tracking changes in marker genotypes over time may also provide a reliable means of distinguishing microevolutionary change from the effects of G×E in generating phenotypic trends (see **Supplemental Discussion**).

Finally, as well as improving the accuracy of parentage assignment during pedigree construction, high-density marker scans may revitalize attempts to infer relationships between individuals in unpedigreed populations. As discussed previously, there has been a low uptake of marker-based approaches to estimating quantitative genetic parameters in the absence of pedigree data, largely owing to scepticism about the reliability of marker-based estimators of relatedness (Coltman 2005, Garant & Kruuk 2005, Thomas 2005). However, when thousands of markers are typed, accuracy should be much higher. A related approach has also recently been described by (Visscher et al. 2006). They show how high-density marker scans can be used to estimate quantitative genetic parameters entirely within families (e.g., sibships), such that the impact of confounding environmental variables may be reduced and non-additive genetic variance components can be estimated. The conceptual difference of this new approach relative to pedigree-based methods is that the markers are used to estimate realized rather than predicted identity-by-descent sharing between particular sets of known relatives.

In summary, marker-based approaches to studying microevolution are in their infancy. Most empirical studies from natural populations have been descriptive (analogous to the first quantitative genetic studies of these populations where variance components were reported). The next challenges will be to: (*a*) investigate genetic correlations and G×E at the level of the locus, (*b*) track evolutionary responses to selection via allele frequency changes, and (*c*) predict breeding values from high-density marker scans. The ability to marry quantitative genetic theory (which utilizes means and variances of focal traits) with population genetics (which focuses on allele frequencies at relevant genes) may soon become a reality.

8. THE BREEDER'S EQUATION

We have reserved for last a discussion of the breeder's equation, the prediction of the response to selection as the product of the heritability of a trait and the selection acting on it (Falconer & Mackay 1996, chapter 11). In multivariate form, this is given as: $\Delta \mathbf{z} = \mathbf{G}\mathbf{P}^{-1}\mathbf{s}$, where $\Delta \mathbf{z}$ is a vector of the change in trait means between generations, $\mathbf{G}$ is the genetic variance-covariance matrix, $\mathbf{P}^{-1}$ is the inverse of the phenotypic variance-covariance matrix, and $\mathbf{s}$ is the vector of selection differentials (or alternatively, $\Delta \mathbf{z} = \mathbf{G}\boldsymbol{\beta}$ where $\boldsymbol{\beta}$ is a vector of selection gradients; Lande & Arnold 1983). This provides an attractive framework within which to test quantitative predictions. With artificial selection, the univariate version generates accurate enough predictions of the response

of a single trait (Falconer & Mackay 1996, chapter 11), although the extension to two traits is less reliable (Roff 2007).

In wild populations, however, predictions from the breeder's equation typically break down entirely (Merilä et al. 2001). There may be numerous explanations for the lack of correspondence between expectation and observation; these usually assume inaccurate measurement of either selection or genetic variance, changing environmental conditions, or an incomplete picture of all traits under selection (Merilä et al. 2001; see also Barton & Turelli 1989, Hadfield 2008, Wilson et al. 2006). Even for a rare case of artificial selection being applied to a wild population, phenotypic change showed little correspondence to the theoretical predictions (Postma et al. 2007). There is also increasing realization that predictions of evolutionary change need to incorporate population demography, particularly with the overlapping generations and varying cohort sizes typical of many of the populations mentioned here (Coulson et al. 2006a,b).

Thus, though being able to test quantitative predictions is highly appealing, for studies of natural selection in heterogeneous environments we may have to acknowledge that the breeder's equation is too simplistic a representation. Combined with concern as to the interpretation of trends in breeding values (see **Supplemental Discussion**), it is probably fair to conclude that we are currently less confident about predicting or understanding temporal trends in phenotypic trends than we might have thought we were 10 years ago. Clearly this is disappointing as, from a management or conservation viewpoint, quantitative genetics is most useful in relation to being able to understand or predict phenotypic trends. However, given the current activity in the area, this is hopefully a conclusion that will be overturned swiftly as we learn how to incorporate a battery of new tools—ranging from SNP technology to the incorporation of population demography—into tests and predictions for microevolutionary change.

SUMMARY POINTS AND FUTURE ISSUES

1. Studies of the genetic basis of quantitative traits in natural populations can provide valuable insights into evolutionary processes and into the evolutionary trade-offs that may contribute to the maintenance of genetic diversity, for example, via sexually-antagonistic effects, maternal effects, or G×E interactions. Phenotypic correlations may not give an accurate representation of the underlying genetic associations that are critical to much life-history theory, and a quantitative genetic analysis becomes essential when testing assumptions underlying many models in evolutionary ecology.
2. Evidence for changes in evolutionary parameters over both environments and ontogeny is now so strong that their constancy cannot be assumed, especially across a long-term study. However, the relative novelty of the animal model techniques to evolutionary analyses has revealed methodological issues that still need careful consideration. In particular, sensible specification of models is required, statistical power may be limited, and trends in estimated breeding values may be open to alternative interpretations (see **Supplemental Discussion**).
3. Although we have focused largely on techniques for dissecting components of phenotypic diversity, a full understanding of evolutionary processes also requires analyses of selection patterns. It is possible to quantify selection within a quantitative genetic framework, and estimates of the genetic correlation between trait and fitness provide a robust test for evolutionarily relevant selection.

4. Many interesting patterns only become apparent once multi- rather than univariate analyses are undertaken. To date, however, quantitative genetic studies of wild populations have not yet moved far beyond bivariate analyses. Canonical analyses of multitrait G matrices may give considerably greater insights into limitations and constraints on genetic variance, particularly when combined with more thorough explanations of patterns of nonlinear selection (Blows 2007).
5. We hope that future directions in the study of the quantitative genetics of wild populations will include a wider diversity of taxa and traits (for example, considering the genetics of behavioral traits, Stirling et al. 2002) and of the genetic basis of ecological or social interactions within a population (for example, Bijma et al. 2007).
6. Use of genomic data is likely to open up a wealth of new avenues of investigation and may also offer alternative approaches to estimating traditional quantitative genetic parameters such as breeding values.
7. Studies of the quantitative genetics of natural populations have revealed the extent to which ecological conditions affect evolutionary processes. The challenge now is to understand how microevolutionary change within a population impacts ecological processes.

DISCLOSURE STATEMENT

The authors are not aware of any biases that might be perceived as affecting the objectivity of this review.

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