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Short Review

Genetic architecture of fitness and nonfitness traits: empirical patterns and development of ideas

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Comparative studies of the genetic architecture of different types of traits were initially prompted by the expectation that traits under strong directional selection (fitness traits) should have lower levels of genetic variability than those mainly under weak stabilizing selection (nonfitness traits). Hence, early comparative studies revealing lower heritabilities of fitness than nonfitness traits were first framed in terms of giving empirical support for this prediction, but subsequent treatments have effectively reversed this view. Fitness traits seem to have higher levels of additive genetic variance than nonfitness traits — an observation that has been explained in terms of the larger number loci influencing fitness as compared to nonfitness traits. This hypothesis about the larger functional architecture of fitness than nonfitness traits is supported by their higher mutational variability, which is hard to reconcile without evoking capture of mutational variability over many loci. The lower heritabilities of fitness than nonfitness traits, despite the higher additive genetic variance of the former, occur because of their higher residual variances. Recent comparative studies of dominance contributions for different types of traits, together with theoretical predictions and a large body of indirect evidence, suggest an important role of dominance variance in determining levels of residual variance for fitness-traits. The role of epistasis should not be discounted either, since a large number of loci increases the potential for epistatic interactions, and epistasis is strongly implicated in hybrid breakdown.

Keywords: additive genetic variance, dominance variance, fitness, heritability, life history traits, natural selection, sexual selection.

Introduction

Whether one should expect fitness or its major components to exhibit significant heritability has been a matter of considerable controversy in evolutionary biology during the past two decades (Gustafsson, 1986; Charlesworth, 1987; Jones, 1987; Barton & Turelli, 1989; Kirkpatrick & Ryan, 1991; Burt, 1995). The origins of this controversy can be traced back to Fisher's 'Fundamental Theorem of Natural Selection' (Fisher, 1930), according to which 'the rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time' (Fisher, 1930). The standard, and possibly naïve interpretation of Fisher's theorem has been that there should be little or no genetic variation in fitness-related traits, since the alleles conferring highest fitness will be driven quickly to fixation by natural selection (e.g. Jones, 1987). However, as pointed out by several authors, there has been considerable controversy over what Fisher really meant to say, and the implicit assumptions that he made (Frank & Slatkin, 1992). For example, the validity of Fisher's theorem depends on many assumptions such as weak selection, constancy of genotypic fitnesses over time, and absence of migration (Kimura, 1958; Charlesworth, 1987). For example, if the population is divided into many patches and there are spatial differences in fitness between the patches, immigration between patches can maintain some genetic variation in fitness (Charlesworth, 1987). Finally, Fisher was referring to fitness itself, and not to its components. There might be genetic variation for example both for early and late fecundity, but if the genetic correlation between these two components of fitness were negative, there need not be (much) genetic variation in total fitness (Falconer & Mackay, 1996). However, several studies have demonstrated significant genetic variability in total fitness or its major components (e.g. Burt, 1995; Fowler *et al*., 1997).

In recent years, much of the focus in evolutionary quantitative genetics has centred around the question of how genetic variability in fitness and/or its components is maintained (e.g. Kondrashov & Houle, 1994; Fowler *et al*., 1997; Fry *et al*., 1998). According to the mutation–selection balance view, there is a continuous and frequent input of new mutations of which most persist very briefly, and consequently, most of the variation important to evolution and adaptation does not originate from standing variation, but from newly arisen mutations. According to the balancing selection view, on the other hand, genetic variation available to adaptation is not very much a function of mutation pressure, but more a function of existing variation which is protected from selection by opposing or fluctuating selection pressures (e.g. antagonistic pleiotropy or negative frequency dependent selection), and consequently, new mutations have only a minor role in adaptation.

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Both of these issues, the existence of significant heritable variation in fitness or its major components, and mechanisms for the maintenance of genetic variability in traits under directional selection, relate intimately to the broad question as to whether genetic architecture of traits assumed be under strong directional selection (e.g. life history traits, sexually selected traits; *fitness traits*), is fundamentally different from that of traits assumed to be under weak stabilizing selection (e.g. morphological traits; *nonfitness traits*). By genetic architecture we mean the number of loci influencing a trait, and the nature of interactions between alleles within and between loci. The division of traits to fitness and nonfitness traits is necessarily arbitrary, given the paucity of information about the form of selection on traits of any kind in most natural populations. However, it seems plausible that traits such as lifespan and fecundity are likely to be positively correlated with fitness in most organisms, whereas the majority of morphological traits will either be under weak or stabilizing selection. Ideally, one would possess data about each trait's partial correlation with fitness and then treat this as a continuous variable; this approach is possible only for a few exceptionally well-studied natural populations (e.g. Gustafsson, 1986; Merilä & Sheldon, 1999).

The aim of this review is three-fold. First, to provide a synopsis of current knowledge of empirical patterns of genetic and environmental sources of variability in different types of traits, and second, to provide an overview of the development of the ideas and understanding of the genetic architecture of different types of traits. Although several comparative studies of genetic architecture of different types of traits have appeared during the last decade or so (e.g. Gustafsson, 1986; Mousseau & Roff, 1987; Roff & Mousseau, 1987; Houle, 1992; Crnokrak & Roff, 1995; Pomiankowski & Møller, 1995; Houle *et al*., 1996; Roff, 1996), there appears to be a gap in the integration of nonadditive genetic variance into the broad picture. Hence, our third aim is to bridge this gap and help to identify areas requiring further research in comparative studies of genetic architecture of quantitative traits. We approach all of these questions from the standpoint of how they relate to quantitative genetic variation in natural populations, particularly in relation to current interest in indirect selection on female mating preferences, although in most cases relevant data are only available from laboratory models.

Life history vs. morphological traits

A well-known result from population genetic theory is that directional selection tends to reduce additive genetic variance in a trait, because alleles conferring highest fitness will be driven quickly to fixation by natural selection (e.g. Kimura, 1958). Hence, given that all else is equal, the prediction from this is that traits subject to strong directional selection should have lower levels of additive genetic variance than traits subject to weak stabilizing selection. However, the 'all else equal' assumption here is crucial — in order to arrive at this prediction, one has to assume that factors (e.g. number of loci, distribution of mutational effects) and forces influencing levels of genetic variability are the same for two classes of traits.

Although the idea that different types of traits might differ in levels of heritable variation was raised in several earlier treatments (Kearsey & Kojima, 1967; Falconer, 1989), the issue received wider attention with the publication of several well-known comparative studies of heritability estimates (Gustafsson, 1986; Mousseau & Roff, 1987; Roff & Mousseau, 1987). First, Gustafsson (1986) published results showing that there was a negative relationship between a trait's heritability and the proportion of lifetime reproductive success it explained in a wild population of collared flycatchers (*Ficedula albicollis*). Similar results were obtained by Mousseau & Roff (1987) in their review of data from outbred animal populations, and by Roff & Mousseau (1987) in an extensive review of *Drosophila* literature. Initially, these results were framed as supporting the theoretical prediction that traits closely associated with fitness have lower levels of additive genetic variance than those more remotely associated with fitness.

However, a few years later, Price & Schluter (1991) suggested an alternative explanation for these patterns, namely, that the lower heritabilities of life history traits were not due to their lower levels of additive genetic variance (V_A), but because of their higher environmental variance (V_E). Because heritability is the ratio of additive genetic variance to total phenotypic variance ($h^2 = V_A/V_P$), and V_P is composed of both genetic and environmental sources of variation (i.e. $V_P = V_A + V_D + V_E + V_I$, where V_D = dominance variance, V_I = epistatic variance), it is easy to see how low heritability could result from increased environmental variation. Price & Schluter (1991) suggested that increased V_E of life history traits would occur because many life history traits are complex and influenced by numerous other qualities of individuals, and therefore, capture more environmental variance than morphological traits (Fig. 1). Strong support for this argument was soon provided by Houle (1992), who compared additive genetic variances and residual variances for different traits in *Drosophila* using standardized measures of variability. Since variance scales positively with the mean, Houle (1992) argued that comparison of genetic variances between different traits should be made using mean-standardized measures, such as coefficients of variation (coefficient of additive genetic variation: $CV_A = 100\sqrt{V_A}/\bar{x}$; coefficient of residual variation $CV_R = 100\sqrt{(V_P - V_A)}/\bar{x}$, where $\bar{x}$ is the trait mean). When compared on this standardized scale, life history traits are found to have significantly higher additive genetic variances than morphological traits (Houle, 1992). However, as suggested by Price & Schluter (1991), they seem also to have higher residual variances, and therefore lower heritabilities, than morphological traits (Houle, 1992). Hence, the striking result from Houle's analysis (see also Messina 1993) is that there is actually more additive genetic variance in traits suggested to be under strong directional selection, than in traits suggested be under weak stabilizing selection.

One explanation for this apparently paradoxical finding is that the life history traits have higher input of mutational variance than morphological traits and therefore also have higher additive genetic variances. Houle *et al*. (1996) compiled data on mutational variabilities from published studies, and found that mutational variabilities were much (~six times) higher for life history than for morphological traits. Hence,

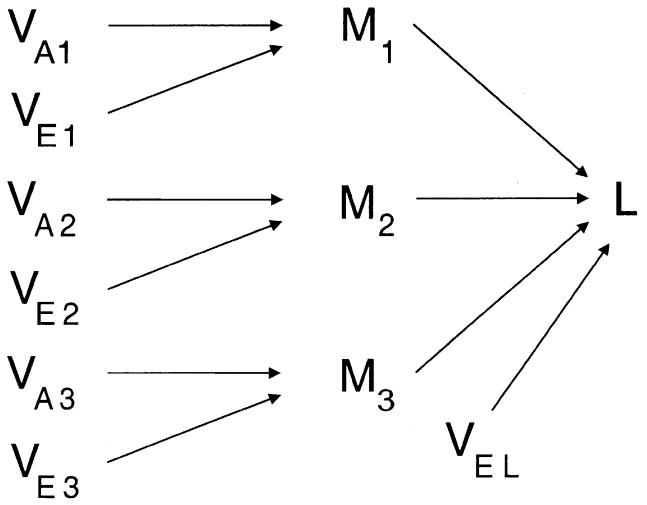

Fig. 1 A schematic illustration of hypothesized difference in genetic architecture of morphological and life history traits. L depicts some life history trait, M_{1-3} refer to morphological traits which all have their underlying additive genetic (V_{Ai}) and environmental (V_{Ei}) components. Variation in life history traits arise as the summed effects of variation in morphological traits, and is in addition influenced by environmental variation influencing the life history trait directly (V_{EL}). For sake of simplicity, correlation between different traits is ignored. Modified from Price & Schluter (1991).

this could explain the higher additive genetic variances for life history traits. In other words, as Houle *et al.* (1996) put it: 'as every locus in the genome must be capable of affecting fitness, we expect that fitness and its components are very large mutational targets'. Thus, the empirical finding that life history traits have higher levels of additive genetic variance than morphological traits could be explained by them being influenced by many loci across which more mutations are accumulated. If one were able to calculate the 'per-locus' additive contribution to fitness traits, this might be lower than for nonfitness traits, but the large number of loci influencing fitness traits leads to greater overall additive variation for fitness traits. Because they also have higher residual variances, their heritabilities are low. However, the picture is not complete since the residual variance includes nonadditive genetic sources of variation, besides environmental ones.

The role of nonadditive genetic variance

Although the early reviews of heritabilities (Gustafsson, 1986; Mousseau & Roff, 1987; Roff & Mousseau, 1987) and coefficients of additive genetic variance (Houle, 1992) did not explicitly distinguish between dominance and environmental variance as a source of residual variance, population and quantitative genetic theories actually predict that dominance contributions to fitness related traits should be high (Fisher, 1958; Frankham, 1991; Crnokrak & Roff, 1995). Because selection is expected to erode additive genetic variation in fitness traits relatively fast, most of the remaining genetic variation in fitness traits should be attributable to dominance variance (Roff, 1997). Consequently, one should also expect that the dominance contribution will be higher for fitness traits than for nonfitness traits. There are two lines of evidence which support this expectation.

First, inbreeding depression (or its converse: heterosis), which by definition owes to dominance or over-dominance, is usually much more pronounced for fitness than nonfitness related traits (e.g. Lynch & Walsh, 1998; DeRose & Roff, 1999). Secondly, Crnokrak & Roff (1995) compared levels of dominance variance in life history and morphological traits and found that levels of dominance variance were much higher for life history than morphological traits. Furthermore, as morphological traits in domesticated animals have often been subject to intense directional selection, they also predicted that the difference in levels of dominance contribution to life history and morphological traits should be less pronounced in domestic, than in outbred populations. This prediction was also confirmed (Crnokrak & Roff, 1995). In the data set compiled by Houle *et al.* (1996), the high residual variance of life history traits was attributed explicitly to high levels of environmental variance, and not to elevated dominance contribution. However, Houle *et al.* (1996) did not detail how the possibility of dominance contribution was excluded, and part of this could still owe to interactive effects. An additional factor which suggests that dominance variance is an important source of phenotypic variation in traits under directional selection comes from artificial selection experiments (Frankham, 1991). Compilation of published studies shows that selection responses are usually asymmetrical, such that selection towards lower fitness results in a much larger response than that towards higher fitness (Frankham, 1991). This is what one should expect if there is significant directional dominance in fitness traits. Finally, it is worth noting that since directional selection is expected to deplete additive genetic variance faster than dominance variance, the prediction is that the ratio of dominance to additive genetic variance should be higher for fitness than for nonfitness traits as confirmed by Crnokrak & Roff (1995; see also Roff, 1997). This finding is not predicted by Price & Schluter's (1991) model.

The standard 'Fisherian' model of evolution treats adaptation as a response to selection acting on individual loci. By contrast, Sewall Wright (e.g. Wright, 1931) stressed the role of interactions between loci in determining evolutionary trajectories. In quantitative genetic terms the contribution of interactions between alleles at different loci to determining phenotypic variation is termed epistasis. While there is still considerable debate about the relative merits of these two models of evolution (Coyne *et al.*, 1997; Wade & Goodnight, 1998), this debate does not bear on the issue of how important epistasis is in contributing to phenotypic variation (Fenster *et al.*, 1997). Epistasis is difficult to quantify in natural populations, but a number of observations suggest that epistasis may contribute disproportionately to variance in fitness traits. First, if one accepts the argument (based both on logic and comparisons of mutational variances) that fitness traits are affected by more loci than nonfitness traits, epistasis is likely to be more important for fitness variation simply

because there are many more interactions possible between a greater number of loci (Lynch & Walsh, 1998). Secondly, analysis of line-crosses provides a useful means to investigate epistatic contributions to phenotypic variance, since the presence of nonadditive gene action will result in departure of the phenotypes of F_1 offspring of two crossed lines from the average of the parental generation. In many cases, these analyses reveal substantial epistasis for quantitative traits, even though very few studies have been able to estimate the contribution of epistatic interactions involving more than two loci (Lynch & Walsh, 1998). Some studies reveal substantial contributions of epistasis to variation in fitness traits (e.g. Falconer, 1989), or fitness itself (Armbruster *et al*., 1997). Thirdly, hybrid breakdown, which often involves disproportionately large effects on fitness traits, provides strong evidence for the importance of epistasis in natural populations, since the production of F_1 and F_2 hybrid offspring disrupts potentially coadapted gene complexes (Lynch, 1991).

To sum up, there seem to be clear differences in genetic architecture between fitness and nonfitness traits. Fitness traits have lower heritability but more additive genetic variance, and higher levels of residual variance than nonfitness traits. Furthermore, there is evidence to suggest that fitness traits are also subject to high levels of dominance variance, and that epistasis may contribute disproportionately to their phenotypic variance. Given that there appear to be clear differences in genetic architecture between fitness and nonfitness traits, when fitness traits are represented by life-history traits, the question becomes whether these patterns apply also to sexually selected traits, since these should also represent a class of traits that have been subject to strong directional selection.

Sexually selected traits

Sexually selected traits often are, or have been, subject to strong directional selection (Andersson, 1994), and one should therefore probably expect to see similarities between genetic architecture of sexually selected and life history traits. However, despite the immense interest the study of sexual selection has attracted (Andersson, 1994), data concerning the quantitative genetics of sexually selected traits are less extensive. Pomiankowski & Møller (1995) compiled 38 heritability estimates of sexually selected characters, and compared these to estimates of heritability of homologous characters in females, and characters suggested to be under stabilizing selection in the same species. These comparisons revealed that the heritabilities for sexually selected traits were actually higher than those of traits suggested to be subject to stabilizing selection. Sexually selected characters had much higher CV_As than nonsexually selected characters, whereas residual variances were very similar for all types of trait. To explain the high CV_As of sexually selected traits, Pomiankowski & Møller (1995; see also Turner, 1995) proposed that strong sexual selection has favoured evolution of modifier genes which have (i) increased the number of genes influencing these traits, as well as (ii) increased the average contribution of loci to phenotypic variation in sexually selected traits. While this is a possibility, Rowe & Houle (1996) suggested an alternative, but simpler, explanation for high CV_A in sexually selected traits. Their explanation was founded on the assumption that the expression of sexually selected traits is highly condition-dependent, meaning that the degree of expression of male traits covaries with male condition (Andersson, 1994). If there is genetic variation in condition, and this variation owes to the effects of many genes, then the high CV_A could be explained by a similar argument to that for the high CV_A of life history traits — since more genes are involved, sexually selected traits are larger mutational targets than nonsexually selected traits, and are therefore, genetically more variable than nonsexually selected traits. However, by this argument one should also expect higher CV_R for sexually selected traits than for nonsexually selected traits, something not found in the data set compiled by Pomiankowski & Møller (1995).

Hence, the data from both life history and sexually selected traits seem to point in the same direction, namely, that there are generally higher levels of additive genetic variation in traits under directional selection than in traits under stabilizing selection. This may be explained by their being larger mutational targets. However, as pointed out by several authors (Alatalo *et al*., 1997; Griffith *et al*., 1999) the data concerning sexually selected traits are relatively sparse and full of uncertainties. For example, of the 38 estimates used by Pomiankowski & Møller (1995) review, 10 were of birds, of which only two had controlled for possible environmental correlation between parents and offspring, meaning that overestimation of CV_As (and concomitant underestimation of CV_Rs) are quite likely. This is suggested, for example, by the fact that while the heritability estimate for house sparrow (*Passer domesticus*) badge size given by Pomiankowski & Møller is 0.60, a recent study by Griffith *et al*. (1999) involving cross-fostering suggests the heritability is much lower (and not significantly different from zero). If we add to these considerations the fact that there appear to be publication biases in heritability estimates, perhaps owing to a recent 'paradigm shift' (Alatalo *et al*., 1997), it would be prudent to refrain from generalizations about the genetic architecture of sexually selected traits for the time being. We also note that there seems to be very little information about the size of nonadditive genetic components of variance for sexually selected traits.

Genetic correlations

The question of whether the genetic architecture of fitness and nonfitness related traits are fundamentally different in terms of their genetic correlation structure has long been a subject of interest (e.g. Houle, 1991; Roff, 1996). One reason to believe that there should be a difference is based upon the popular argument (e.g. Falconer & Mackay, 1996; Roff, 1997) that different components of fitness should become negatively correlated (i.e. show antagonistic pleiotropy), because those alleles that contribute positively for both traits (e.g. early and late fecundity) will be quickly driven to fixation, whereas alleles that contribute negatively to both traits will be quickly eliminated. Therefore, alleles that contribute positively to one and negatively to another trait will segregate for longer at intermediate frequencies (i.e. equivalent effects on total fitness). A related logical argument goes that, since most

mutations are likely to be deleterious, we can assume that most pleiotropic mutations are also harmful, and the next commonest class of mutations would be those with a negative effect on one, and positive effect on the other trait (Prout, 1980; Roff, 1997).

There has been controversy over whether these predictions are overly simplistic, since direct estimates of pleiotropic effects of mutations are few, and the nature of the pleiotropy is not always clear (Houle, 1998). Consequently, different theoretical treatments differ widely in their predictions as to whether fitness components should become negatively correlated or not (e.g. Houle, 1991). The outcome of these models turns out to be very sensitive to assumptions about mutation process, genetic variation in ability to acquire resources (Houle, 1991), as well as about the amount of dominance variance in fitness components (Curtsinger *et al*., 1994). The current resolution is that the conditions for evolution of antagonistic pleiotropy are quite restrictive, and this has cast doubt on the possibility that balancing selection arizing from antagonistic selection would be important in maintaining genetic variation in fitness related traits. In the hope of clarifying the situation, Roff (1996) recently reviewed 1872 empirical estimates of genetic correlations and compared their sign and magnitude depending on whether these correlations were estimated between different life history and morphological traits. There were significantly more negative genetic correlations between life history than between other types of traits, although most of the life history traits were still positively correlated (Roff, 1996). The data are not very conclusive, but they seem to conform to the prediction that traits subject to strong selection in the same direction are more likely to be negatively genetically correlated.

Finally, antagonistic pleiotropy was previously a strong candidate for a mechanism which would help to explain how genetic variation in traits subject to directional selection could be maintained, but theoretical treatments then suggested that the conditions in which antagonistic pleiotropy could work were very restrictive (Curtsinger *et al*., 1994; see also: Hedrick, 1999). However, as pointed out by Roff (1997), one of the main reasons that Curtsinger *et al*. (1994) concluded that antagonistic pleiotropy was not likely to play a major role in maintaining fitness was that high levels of dominance variance are required in order for their model to work: 'If antagonism of fitness components often plays a role in maintaining polymorphisms, then the dominance variance for fitness components should, on average, be about half as large as the additive genetic variance for those same fitness components' (Curtsinger *et al*., 1994). However, as shown by Crnokrak & Roff (1995), 70% of the available estimates show that the ratio of dominance variance to additive genetic variance is higher than 0.5. Hence, this suggests that the chapter is not closed yet, and that antagonistic pleiotropy remains a viable model for explaining maintain genetic variation in fitness-related traits (Roff, 1997).

Implications

What are the implications of recent findings concerning the genetic architecture of fitness traits for studies of natural populations? We discuss this in relation to the question of whether selection can act on female mate preferences via the indirect (i.e. genetic) effects of those preferences on fitness of offspring. Not surprisingly, this field has been influenced heavily by the development of ideas about genetic sources of variation in fitness (e.g. Charlesworth, 1987; Kirkpatrick & Ryan, 1991; Andersson, 1994; Pomiankowski & Møller, 1995; Kirkpatrick & Barton, 1997). Initial scepticism about the feasibility of indirect selection was fuelled by the observation that the heritability of fitness did indeed seem to be very small: few studies were able to demonstrate any significant additive genetic effect on fitness (Burt, 1995), but this probably had more to do with technical difficulties (e.g. controlling for confounding variation, large standard errors associated with the estimates) than with the absence of an effect. However, empirical studies of natural or seminatural populations by behavioural ecologists, less constrained by population genetic theory in their approach to this question, have repeatedly produced results that are consistent with additive genetic variance in fitness driving the evolution of female mating preferences. A recent meta-analysis of 22 such studies suggests that the conclusion that female mating preferences can increase the fitness of their offspring through purely genetic means is robust, although the average effect size is small, corresponding to approximately 1.5% of the total phenotypic variance in fitness (Møller & Alatalo, 1999). Under these conditions, selection on female mating preferences is weak, and can easily be swamped by direct selection (Kirkpatrick & Barton, 1997), although in many cases direct and indirect selection are likely to act in similar directions. These findings agree well with the emerging picture of additive genetic variation for fitness described above. Although the additive genetic contribution to phenotypic variation in fitness is small, and hence the potential indirect fitness benefits of mate choice small, there is no absence of genetic variation to maintain selection for directional preferences.

The finding that nonadditive genetic variance for fitness may be substantial has not yet been fully integrated into empirical work on sexual selection. If epistasis and dominance contribute substantially to fitness variation, as suggested above, there are a number of implications for selection on mate choice behaviour of females. First, the genetic effect of a male on a female's offspring will not be independent of the female's genotype, suggesting that different females might have different optimal mates. Recent work arguing for the importance of genetic compatibility in sexual selection (e.g. Zeh & Zeh, 1996) is based on the idea of interactions between male and female genotypes, although the arguments are often couched in terms of the effects being due to selfish genetic elements. This perspective could be widened to include general epistasis and dominance. Second, epistasis will tend to increase the variance in offspring characters more for females that mate multiply than for females that mate singly. Strong selection on characters closely related to fitness will tend to magnify the fitness consequences of an increase in the variance of fitness, even if there is no change in mean fitness of offspring produced by females mating multiply (which must be true if mating is random). Multiple mating by females is frequently observed in a wide range of taxa (Birkhead & Møller, 1998), and in many cases appears indiscriminate. Perspectives gained from studies

of nonadditive genetic variance for fitness may illuminate this phenomenon, and also contribute to explaining recent empirical studies that have revealed fitness-benefits for females of mating with multiple mates (e.g. Tregenza & Wedell, 1998).

Conclusions

In conclusion, the data suggest that fitness traits have low heritabilities, and consequently most of the variation in fitness in nature is not likely to be inherited by offspring from parents. This however, does not mean that the heritability of fitness would be zero, or that 'good genes' models of sexual selection are implausible (Møller & Alatalo, 1999). However, the current data strongly suggest that environmental and nonadditive genetic effects are the most important determinants of fitness in nature. Despite their low heritability, fitness traits harbour high absolute levels of additive genetic variation, possibly because they capture genetic variation and accumulate mutations from many loci. Consequently, genetic variability in fitness-related traits should not became easily depleted. Given the evidence for the importance of dominance variance in contributing to fitness, antagonistic pleiotropy remains a viable mechanism for the maintenance of genetic variation in traits under strong directional selection. Finally, as a historical reflection, we note that it is quite remarkable to look back and see how comparative studies of genetic architecture of different types of traits started from the prediction that traits most closely associated with fitness would have the lowest levels of genetic variability, and have ended up showing the opposite. Against this historical background, it would be unwise to suggest that the controversy over causes of variation in genetic architecture has been resolved. Much of the evidence for differential functional architecture of morphological and life history traits, for example, is circumstantial, and more direct approaches are required to establish these differences firmly.

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