Notes and Comments

The Good-Genes and Compatible-Genes Benefits of Mate Choice

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ABSTRACT: Genetic benefits from mate choice could be attained by choosing mates with high heritable quality ("good genes") and that are genetically compatible ("compatible genes"). We clarify the conceptual and empirical framework for estimating genetic benefits of mate choice, stressing that benefits must be measured from offspring fitness because there are no unequivocal surrogates for genetic quality of individuals or for compatibility of parents. We detail the relationship between genetic benefits and additive and nonadditive genetic variance in fitness, showing that the benefits have been overestimated in previous verbal treatments. We point out that additive benefits readily arise from nonadditive gene action and that the idea of "heritable nonadditive benefits" is a misconception. We review the empirical evidence of the magnitude of benefits of good genes and compatible genes in animal populations, and we outline the most promising future directions for empirical research on the genetic benefits of mate choice.

Keywords: female choice, genetic compatibility, indirect benefits, sexual selection.

Introduction

Mate choice, and especially mate choice for indirect genetic benefits, is an important and debated topic in the current research on sexual selection. Choice based on secondary sexual traits that presumably indicate the heritable quality of mates ("good genes") has been rigorously studied both theoretically and empirically (for reviews, see Mead and Arnold 2004; Tomkins et al. 2004; Kokko et al. 2006). More recently it has been recognized that genetic benefits can also be achieved through the choice of mates who are genetically compatible (Zeh and Zeh 1996, 1997; Tregenza and Wedell 2000; Colegrave et al. 2002; Neff and Pitcher 2005). The concept of genetic compatibility rests on the idea that offspring fitness can be increased by choosing a mate with alleles that, when combined with the alleles of the choosing parent, yield a high genetic value for fitness. With the introduction of the idea of genetic compatibility and the new molecular tools available for studying the genetic basis of fitness differences, the study of genetic benefits of mate choice has been revived.

In the past, no distinction was made between additive and nonadditive genetic benefits of mate choice, but the concept of "good genes" referred to any indirect genetic benefits of choice (e.g., Møller and Alatalo 1999). Currently, the "good genes" concept is defined as additive genetic variance in fitness, and the "compatible genes" concept is defined as nonadditive genetic variance in fitness (Neff and Pitcher 2005; Puurtinen et al. 2005). This more precise conceptual framework will facilitate progress in research on mate choice. However, the use of the terms "good genes" and "compatible genes" is still highly ambiguous in current literature. Kempenaers (2007) states that "these terms are often loosely defined or not defined at all" (p. 194) and that "there is a need for clear-cut and generally accepted definitions" (p. 196). Ample confusion in literature is apparent in regard to questions such as how good genes and compatible genes should be understood at the allelic level, how nonadditive gene action and nonadditive genetic variance are related to one another, and what constitutes evidence for mate choice for compatible-genes benefits.

Let us give just two examples to make it clear that there is a dire need for a conceptual revision and unification regarding the benefits of good genes and compatible genes in mate choice. First, is it possible to distinguish "good alleles" and "compatible alleles," as is suggested by Kempenaers (2007)? Consider for a moment the role of deleterious mutations in contributing to the variation of good genes and compatible genes in fitness. The influential "genic capture" hypothesis posits that deleterious mutations are the main cause of good-genes variation in populations (Rowe and Houle 1996; Tomkins et al. 2004). On the other hand, it is well known that deleterious mutations are also the main source of inbreeding depression (Charlesworth and Charlesworth 1999), which is universally accepted to be a case of compatible-genes variation (Tregenza and Wedell 2000; Neff and Pitcher 2005; Puurtinen et al. 2005; Kempenaers 2007). Does this mean that

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the same alleles (mutations) can contribute to variation in both good-genes and compatible-genes variation? We suggest that this is the case and that the labeling of single alleles as "good" or "compatible" is, in most cases, flawed. This topic is addressed in detail in "Allele-Level Interpretation of the Potential Genetic Benefits of Mate Choice."

As a second example, consider the suggestion that genetic dissimilarity of mates could be used as a proxy for the genetic compatibility of the parents (e.g., Mays and Hill 2004; Fossoy et al. 2008; Gillingham et al. 2009). This approach is problematic for two reasons. First, the relationship between genetic dissimilarity and offspring fitness in any particular system cannot be predicted a priori because different species show very different optimal outbreeding distances. Depending on the system being studied, almost any relationship between genetic dissimilarity and reproductive success can be expected. Although fitness usually decreases with genetic similarity of the parents, positive within-population relationships between genetic similarity and reproductive success have been detected in, for example, Peron's tree frogs (Sherman et al. 2008) and *Ambrosia* beetles (Peer and Taborsky 2005), and hump-shaped relationships have been found in, for example, fig wasps (Greeff et al. 2009), blue-gill sunfish (Neff 2004), common lizards (Richard et al. 2009), and humans (Helgason et al. 2008). A further technical difficulty regarding the use of genetic dissimilarity as a proxy for genetic compatibility is that indices of molecular dissimilarity (Queller and Goodnight 1989) and individual heterozygosity are intercorrelated (Roberts et al. 2006), meaning that more heterozygous individuals will, on average, be less similar to the members of their population. When this is the case, genetic dissimilarity of a pair is effectively confounded with the heterozygosity of individuals, making it impossible to draw any distinction between good genes and compatible genes.

Here our purpose is to clarify the definitions of the genetic benefits of mate choice, starting with the fact that genetic benefits must be measured from the reproductive consequences of mate choice, ideally including the number of offspring produced and their subsequent reproductive success (Kokko et al. 2003). Good-genes benefits are then understood to be the effects on reproduction that are due to the mate's breeding value for fitness, and compatible-genes benefits are understood to be the effects that are due to the genetic interactions of the genes of the parents. These definitions are logical, simple, and intuitive. What is more, these definitions have a clear-cut relationship with the components of quantitative genetic variance in fitness, and thus they offer a way to compile and compare studies with various quantitative genetic designs. Furthermore, because the relationship between the components of quantitative genetic variance and allelic effects is mathematically well understood, it becomes possible to understand how variation at the molecular level contributes to good-genes and compatible-genes benefits of mate choice. After explaining these conceptual issues in detail, we review the empirical evidence of the magnitude of potential good-genes and compatible-genes benefits of mate choice in genetically variable animal populations, and we outline the most promising future directions for research on the genetic benefits of mate choice.

Formal Definition of the Genetic Benefits of Mate Choice

The genetic benefits of mate choice relate to genetic variation in offspring fitness within a population. This is the key to understanding the nature of genetic benefits. If we adopt the conventional female-choice scenario, the question is, which male should a female choose to obtain offspring with the highest genetic value for fitness? Males can vary in their good-genes quality, that is, in their expected reproductive success and the genotypic value of their offspring when they are mated with a random female in the population. Males can also vary in their compatibility with specific females, meaning that there may be interaction effects between males and females that result in variation in reproductive success and the genotypic value of the offspring that is independent of the good-genes qualities of the parents. The larger the variation in either the good-genes effects of the males or the compatible-genes effects, the more a female can gain by choosing a mate with desirable qualities. Hence, the potential genetic benefits of female mate choice are equal to the variation in effects of good genes and compatible genes in the population.

This definition of the potential benefits to mate choice of good genes and compatible genes can be formalized and implemented in a mating design where a sample of males and females from a population are crossed in all possible combinations and the consequences to reproductive success and offspring fitness are recorded (Wedekind et al. 2001; Neff and Pitcher 2005). This cross-classified breeding design, called the North Carolina II (NCII) breeding design (Comstock and Robinson 1948; Lynch and Walsh 1998), is also an illustrative heuristic for understanding how male effects and male-by-female interaction effects are calculated and how fitness variation in a population can be partitioned to components of good genes and compatible genes.

In the NCII breeding design, a number of males and females are mated so that each male breeds with each female. For each pair, the fitness of k offspring is recorded. Fitness of the kth offspring from male i and female j can be expressed as

$$w_{ijk} = \bar{w} + m_i + d_j + I_{ij} + e_{ijk},$$

where $\bar{w}$ is the mean fitness in the population, m_i is the effect of the male i, d_j is the effect of the female j, I_{ij} is the interaction effect of male i and female j, and e_{ijk} is the deviation from the family mean of the kth offspring. Both males and females are assumed to be randomly sampled from a larger population. The effects are defined as

$$m_i = \bar{w}_{i\cdot\cdot} - \bar{w},$$

$$d_j = \bar{w}_{\cdot j\cdot} - \bar{w},$$

$$I_{ij} = \bar{w}_{ij\cdot} - \bar{w} - m_i - d_j,$$

$$e_{ijk} = w_{ijk} - \bar{w} - m_i - d_j - I_{ij}.$$

The estimated effect of an individual male (m_i) is a measure of the good-genes quality of that male, and it is equal to one-half of its breeding value for fitness (Falconer and Mackay 1996, p. 114). The effect of interaction of male i and female j (I_{ij}) estimates the genetic contribution to fitness that cannot be explained by the breeding values of the parents, and it is thus a measure of the genetic compatibility of the pair.

All the effects are independent (i.e., uncorrelated), have zero expectations, and have variance estimates that are equal to s_m^2, s_d^2, s_I^2, and s_e^2, respectively. The total variance in fitness in the offspring generation is estimated as simply the sum of the four variance components: $s_w^2 = s_m^2 + s_d^2 + s_I^2 + s_e^2$. The variance components can be calculated, for example, from mean squares or with maximum-likelihood methods (which is preferred when the data are not balanced; Lynch and Walsh 1998, p. 600).

It should be noted that the appropriate estimate of the potential good-genes benefits of female mate choice is the standard deviation, rather than the variance, of the male effects m_i in the population, $s_m = (s_m^2)^{1/2}$. In the same way, the appropriate estimate of the potential compatible-genes benefits is the standard deviation of the interaction effects I_{ij} in the population, $s_I = (s_I^2)^{1/2}$. Standard deviations are preferred for comparisons because they scale linearly with absolute fitness differences (i.e., the potential benefits of mate choice), whereas variances scale with the second power of fitness differences. Unless the potential good-genes and compatible-genes benefits are identical in magnitude, comparison of variance components will result in gross under- or overestimation of the relative magnitudes of the potential benefits. Comparison of standard deviations, however, always yields an unbiased estimate for the relative magnitudes of the potential benefits of good genes and compatible genes.

It is also worth noting that the magnitude of the potential good-genes benefits (s_m) of mate choice is equal for all females. This is because s_m is the standard deviation of male effects calculated over all females and is thus independent of female identity. However, the magnitude of compatible-genes benefits (s_I) is not necessarily equal for all females. This is because s_I is the standard deviation of interaction effects of all females with all males, and thus it gives the expected (i.e., average) magnitude of potential compatible-genes benefits of mate choice. For any individual female, the potential compatible-genes benefit is the standard deviation of the interaction effects of that specific female with all the males. Thus, for some females, the compatible-genes benefit of mate choice is likely to be larger than the expected value, while for other females, the benefit is likely to be smaller.

Quantitative Genetic Interpretation of the Genetic Benefits of Mate Choice

Recent studies have equated good-genes benefits with the amount of additive genetic variance in fitness and compatible-genes benefits with the amount of nonadditive genetic variance in fitness (Neff and Pitcher 2005; Puurtinen et al. 2005). However, a closer examination reveals that this definition is overly simplistic. In fact, the potential benefits from both good genes and compatible genes are much smaller than previously thought. The exact relationship between components of quantitative genetic variation in fitness and the potential benefits of mate choice is determined by interpreting the variance in offspring fitness due to male effects (s_m^2), female effects (s_d^2), interaction effects (s_I^2), and within-family variance (s_e^2) in terms of hypothetical causal components of variance. To keep the equations reasonably simple, we work with effect variances and not with the standard deviations (which must be used, however, when magnitudes of the effects are compared; see above). For the sake of clarity, variance from environmental sources (common and special environmental effects) is not included in the equations. Assuming no sex linkage, and ignoring more-than-two-loci interactions and cytoplasmic genes, the causal components contributing to the estimated variance components are

$$s_m^2 \simeq \frac{1}{4} V_A + \frac{1}{16} V_{AA}, \quad (1)$$

$$s_f^2 \simeq \frac{1}{4} V_A + \frac{1}{16} V_{AA}, \quad (2)$$

$$s_I^2 \simeq \frac{1}{4} V_D + \frac{1}{8} V_{AA} + \frac{1}{8} V_{AD} + \frac{1}{16} V_{DD}, \quad (3)$$

$$s_e^2 \simeq \frac{1}{2} V_A + \frac{3}{4} V_D + \frac{3}{4} V_{AA} + \frac{7}{8} V_{AD} + \frac{15}{16} V_{DD}, \quad (4)$$

where V_A is additive variance, V_D is dominance variance,

V_{AA} is additive × additive epistatic variance, and V_{AD} and V_{DD} are additive × dominance and dominance × dominance epistatic variances, respectively (Lynch and Walsh 1998). If maternally inherited cytoplasmic genes also contribute to the variance in offspring fitness, the equations are slightly more complex:

$$s_m^2 \simeq \frac{1}{4} V_A + \frac{1}{16} V_{AA}, \tag{5}$$

$$s_f^2 \simeq \frac{1}{4} V_A + \frac{1}{16} V_{AA} + V_C + \frac{1}{4} V_{AC}, \tag{6}$$

$$s_I^2 \simeq \frac{1}{4} V_D + \frac{1}{8} V_{AA} + \frac{1}{8} V_{AD} + \frac{1}{16} V_{DD} + \frac{1}{4} V_{AC} + \frac{1}{4} V_{DC}, \tag{7}$$

$$s_e^2 \simeq \frac{1}{2} V_A + \frac{3}{4} V_D + \frac{3}{4} V_{AA} + \frac{7}{8} V_{AD} + \frac{15}{16} V_{DD} + \frac{1}{2} V_{AC} + \frac{3}{4} V_{DC}, \tag{8}$$

where V_C is the variance of additive effects of the cytoplasmic genes and V_{AC} and V_{DC} are the variances of the epistatic gene effects between cytoplasmic and nuclear genes (additive and dominance epistasis, respectively; Lynch and Walsh 1998). As the above equations show, genetic variance involving cytoplasmic genes contributes to the female component of variance and to the interaction variance, that is, to compatible genes. Recent empirical studies have detected sizable contributions of cytoplasmic genes to within-population genetic variance (Rand et al. 2001; Maklakov et al. 2006; Dowling et al. 2007, 2008).

A number of important points are apparent from the quantitative interpretation of the genetic benefits of mate choice. First, the variance in offspring fitness due to males—that is, good-genes variance (s_m^2)—is a function of the additive genetic variance (and a small portion of additive × additive epistatic variance) for fitness in the population, but it captures only one-quarter of the overall V_A. This is because only half of the genes come from the male and only half of his genes are passed on to the offspring. Both theoretical arguments and empirical evidence suggest that the narrow-sense heritability (V_A/V_P) of fitness is low (Gustafsson 1986; Charlesworth 1987; Mousseau and Roff 1987; Roff and Mousseau 1987; Kruuk et al. 2000; Teplitsky et al. 2009). Hence, it is likely that the good-genes benefits of female choice (which are $s_m = [(1/4)V_A + (1/16)V_{AA}]^{1/2}$) are generally rather low.

Second, if there is variance in cytoplasmic effects, females contribute more to genetic variance in offspring fitness than do males. This is because cytoplasmic effects increase the magnitude of the variance component due to females but not the variance component due to males (cf. eqq. [5], [6]). This is interesting, because in such cases, males have more to gain from mate choice in terms of indirect genetic benefits than do females. Also note that nongenetic maternal effects on offspring performance are commonly very large, and they inflate the maternal variance component further (e.g., Hunt and Simmons 2002). In our equations, only variation from genetic sources is depicted.

Third, the variance due to compatible genes (s_I^2) is a function of the nonadditive genetic variances (see eqq. [4], [7]). Because fitness is a complex trait influenced by most, if not all, genes in the genome, nonadditive interactions affecting fitness are also expected to be common (Merilä and Sheldon 1999). Furthermore, directional selection is expected to erode additive genetic variance but not to have a great impact on the amount of nonadditive genetic variance. Thus, nonadditive genetic variance is expected to be a significant source of genetic variation in fitness and life-history traits that are closely associated with fitness. Indeed, empirical studies have found that nonadditive genetic variation is ubiquitous in life-history traits and that life-history traits have relatively more nonadditive genetic variation than, for example, morphological traits (Crnokrak and Roff 1995; Roff and Emerson 2006). Note, however, that at the most, only one-quarter of the nonadditive genetic variance will be expressed as differences among family means (the coefficients of dominance and epistatic genetic variances in eqq. [3] and [7] are all one-quarter or less).

Fourth, excluding additive variation from cytoplasmic genes (V_C), there is always more genetic variance within a group of siblings than among siblings from different families (the coefficients of all variance components, excluding V_C in eqq. [4] and [8], are one-half or more, meaning that one-half or more of variance from each source is among full siblings in a family). Because genetic variance within families is large compared with genetic variance between families, it is reasonable to say that the benefits of mate choice are not highly predictable. In fact, it is impossible to accurately predict the genotypic value of a single offspring from the genotypes of its parents. This point is obvious if you contrast the phenotypic variation among human full siblings with the variation among monozygotic twins.

We must point out an important yet unappreciated problem in many quantitative genetic designs analyzing dichotomous traits like fertilization success or survivorship. These analyses often employ family means as observations, with the result that the total phenotypic (and genetic) variance is underestimated and, thus, heritability is overestimated. The problem arises because the variance of the means is less than the variance of the individual observations (var-

iance of the mean of n measures is one-nth $[1/n]$ of the true variance calculated from the individual observations). When total phenotypic variance is underestimated, heritability and other genetic effects are (often grossly) overestimated, with genetic effects apparently explaining well over 100% of the phenotypic variance (e.g., Pitcher and Neff 2007; Wedekind et al. 2008), which of course is not possible. Fortunately, these mistakes do not affect the magnitudes of male, female, and male-by-female interaction variance components, making comparisons of potential good-genes and compatible-genes benefits still possible (table 1). Also, unbiased estimates of heritability are easily derived by calculating the phenotypic variance from individual observations, instead of using family means.

Allele-Level Interpretation of the Potential Genetic Benefits of Mate Choice

The concepts of good genes and compatible genes are defined in the context of a population of diploid genotypes, but there is also obvious interest to assign potential good-genes and compatible-genes benefits to the phenotypic effects of specific alleles and their interactions (e.g., Pitcher and Neff 2006). In theory, it is possible to calculate the contributions of specific alleles to different components of quantitative genetic variance in fitness and, hence, to good-genes and compatible-genes benefits of mate choice. However, the gene-level interpretation of genetic benefits of mate choice is not as straightforward as it might seem at the outset. An unfortunate and seemingly unshakable misconception among researchers is to associate additive quantitative genetic variance with additive gene action. However, it is exceedingly important to understand that additive quantitative genetic variance in no way implies additive gene action. According to Falconer and Mackay (1996, p. 128), "the concept of additive variance does not carry with it the assumption of additive gene action; and the existence of additive variance is not an indication that any of the genes act additively (i.e., show neither dominance or epistasis).… Additive variance can arise from genes with any degree of dominance or epistasis." This important nonequivalence of gene action and genetic variance can also be stated the other way around: loci with nonadditive effects (dominance and/or epistasis) can and do result in additive quantitative genetic variance (Hill et al. 2008).

If we agree that the genetic benefits of mate choice are measured in terms of quantitative genetic variance in reproductive success, then all the claims asserting heritable nonadditive genetic benefits of mate choice must be seen as misconceptions arising from the erroneous association of nonadditive gene action with nonadditive genetic variance (Reid 2007; Neff and Pitcher 2008, 2009; Fromhage et al. 2009). Claiming heritable nonadditive genetic variance is equal to claiming additive nonadditive genetic variance, which is a logical contradiction and pure nonsense. The misconception of heritable nonadditive benefits aside, studies looking at the heritability of heterozygosity and associated fitness benefits do, however, offer an interesting gene-level perspective to the potential role of mutation and genetic drift in maintaining additive genetic variance in fitness (see Lehmann et al. 2006; Kotiaho et al. 2008; Neff and Pitcher 2008; Fromhage et al. 2009).

What is the relationship between allele effects and components of quantitative genetic variance? Provided that the frequencies and phenotypic effects of all alternative alleles and their interactions are known, calculating the resulting variance components becomes a relatively straightforward mathematical exercise (e.g., see chaps. 4 and 5 in Lynch and Walsh 1998). However, because the additive and nonadditive components of genetic variance are affected by allele frequencies, it does not make sense to label an allele a "good allele" or a "compatible allele" (see Kempenaers 2007). One allele can contribute to additive genetic variation (good-genes variation), to nonadditive genetic variation (compatible-genes variation), or to both, depending on its frequency in the population (see fig. 1 for a simple example of one diallelic locus with complete dominance). Thus, while it is possible to estimate the contribution of specific alleles to potential good-genes and compatible-genes benefits, it must be remembered that these estimates apply only to the allele frequencies in the studied population; were the alleles to be introduced to another population, the effects would be different.

An example of nonadditive gene action resulting in additive genetic variance and good-genes benefits of mate choice is seen in the inbred song sparrow population living on Mandarte Island (British Columbia; Reid 2007). In this population, females can produce offspring with a low inbreeding coefficient and high fitness by choosing males with a large song repertoire (song repertoire size is negatively correlated with the mean kinship of the male with the female population). As the island population suffers from inbreeding depression, the novel alleles carried by occasional immigrants and their offspring will result in higher fitness in the offspring generation, even when the immigrants (or the immigrants' offspring) mate at random in the population. While the immigrant alleles are probably nearly neutral in the larger mainland population, in the inbred island population their effects translate to additive genetic variance in fitness and thus to good-genes benefits (Kotiaho et al. 2008). The idea that local inbreeding can result in a preference for noninbred males and in additive genetic benefits in the local population was first suggested by Reinhold (2004), and a similar idea has recently been proposed by Neff and Pitcher (2008, 2009), although they postulate overdominance in fitness, rather

Table 1: Relative magnitude of the potential compatible-genes benefits

Species, population, trait measured	$s_l/(s_l + s_m)$	N	Range	Reference
Ascidian (*Puyra stolonifera*):				
Natural population:				
Fertilization success	.55	1	…	Marshall and Evans 2007
Survival	.44	1	…	Marshall and Evans 2007
Sea urchin (*Heliocidaris erythrogramma*):				
Natural population (eastern Australia):				
Fertilization success	.77	2	.53–1.00	Evans and Marshall 2005
Natural population (western Australia):				
Fertilization success	.28	1	…	Evans et al. 2007
Survival	1.00	1	…	Evans et al. 2007
Metamorphosis	.13	1	…	Evans et al. 2007
Polychaete (*Galeolaria caespitosa*):				
Natural population:				
Fertilization success	.28	1	…	Marshall and Evans 2005
Sea lamprey (*Petromyzon marinus*):				
Natural population:				
Survival	1.00	1	…	Rodríguez-Muñoz and Tregenza 2008
Chinook salmon (*Oncorhynchus tsawytscha*):				
Natural population:				
Survival	.50	1	…	Pitcher and Neff 2007
Size or weight	.89	1	…	Pitcher and Neff 2007
Lake trout (*Salvelinus namaycush*):				
Hatchery population:				
Development rate	.71	1	…	Pakkasmaa and Jones 2002
Size or weight	.14	2	.00–.29	Pakkasmaa and Jones 2002
Brown trout (*Salmo trutta*):				
Natural population:				
Survival	.00	1	…	Jacob et al. 2007
Arctic charr (*Salvelinus alpinus*):				
Second-generation cultivated stock:				
Growth rate	.29	3	.22–.36	Nilsson 1992
Size or weight	.29	10	.00–.42	Nilsson 1992
Condition index	.29	5	.00–.41	Nilsson 1992
Development rate	.39	2	.37–.41	Nilsson 1992
Cultivated brood fish founded from wild spawners:				
Survival	1.00	1	…	Huuskonen et al. 2003
Atlantic herring (*Clupea harengus*):				
Natural population:				
Size or weight	.37	5	.27–.45	Bang et al. 2006
Atlantic cod (*Gadus morhua*):				
Natural population:				
Survival	1.00	1	…	Rudolfsen et al. 2005
Whitefish (*Coregonus* sp.):				
Natural population (1):				
Survival	.35	2	.00–.70	Wedekind et al. 2001
Natural population (2):				
Survival	.60	4	.28–.99	Wedekind et al. 2008
Cough's spadefoot toad (*Scaphiopus couchii*):				
Natural population:				
Size or weight	.33	4	.00–1.00	Newman 1988
Development rate	.13	2	.00–.26	Newman 1988
Common frog (*Rana temporaria*):				
Two natural populations (1):				
Size or weight	.66	4	.00–1.00	Sommer and Pearman 2003

Table 1 (*Continued*)

Species, population, trait measured	$s_I/(s_I + s_m)$	N	Range	Reference
Development rate	.24	4	.00–.54	Sommer and Pearman 2003
Two natural populations (2):				
Development rate	.51	2	.46–.56	Laurila et al. 2002
Size or weight	.46	4	.00–1.00	Laurila et al. 2002
Growth rate	.44	2	.42–.46	Laurila et al. 2002
Moor frog (*Rana arvalis*):				
Three natural populations:				
Survival	.45	4	.35–.41	Merilä et al. 2004
Developmental anomalies	.51	2	.51–.52	Merilä et al. 2004
Development rate	.62	2	.57–.67	Merilä et al. 2004
Size or weight	.68	4	.66–.69	Merilä et al. 2004
Two natural populations:				
Development rate	.24	4	.00–.67	K. Räsänen and A. Laurila, unpublished manuscript
Size or weight	.36	4	.00–.57	K. Räsänen and A. Laurila, unpublished manuscript
Pool frog (*Rana lessonae*):				
Natural population:				
Size or weight	.29	2	.00–.59	Semlitsch 1993
Development rate	.48	1	…	Semlitsch 1993
Spring peeper (*Hyla crucifer*):				
Natural population:				
Size or weight	.38	2	.23–.53	Travis et al. 1987
Growth rate	.59	1	…	Travis et al. 1987
Development rate	.62	1	…	Travis et al. 1987
Green tree frog (*Hyla cinerea*):				
Natural population:				
Growth rate	.16	2	.04–.29	Blouin 1992
Development rate	0	1	…	Blouin 1992
Size or weight	0	1	…	Blouin 1992
Quacking frog (*Crinia georgiana*):				
Natural population:				
Fertilization success	1	1	…	Dziminski et al. 2008
Survival	1	3	1.00–1.00	Dziminski et al. 2008
Growth rate	1	1	…	Dziminski et al. 2008
Developmental anomalies	1	1	…	Dziminski et al. 2008
Size or weight	1	1	…	Dziminski et al. 2008
Flour beetle (*Tribolium castaneum*):				
Synthetic lab population:				
Development rate	.69	1	…	Dawson 1965
Flour beetle (*Tribolium confusum*):				
Synthetic lab population:				
Development rate	.68	1	…	Dawson 1965

Note: The equation $s_I/(s_I + s_m)$ estimates the magnitude of compatible-genes benefits relative to the sum of compatible-genes benefits and good-genes benefits; values closer to 0 mean that good-genes benefits are larger, and values closer to 1 mean that compatible-genes benefits are larger (see text for definitions of symbols). Information is grouped by the type of life-history trait measured, N is the number of estimates in a study that have been arithmetically averaged, and range gives the lowest and highest estimates in the study. Negative variance components have been set to 0 in calculations.

than recessive deleterious mutations, as the cause of inbreeding depression.

The theoretical considerations and the example above make it clear that estimates of genetic benefits of mate choice can be obtained only from populations where the allele and genotype frequencies are similar to those in populations where mate choice actually takes place. What this means is that estimates obtained from inbred lines (e.g., Ivy 2007; Bilde et al. 2008) will not accurately measure the genetic benefits in wild populations, although they

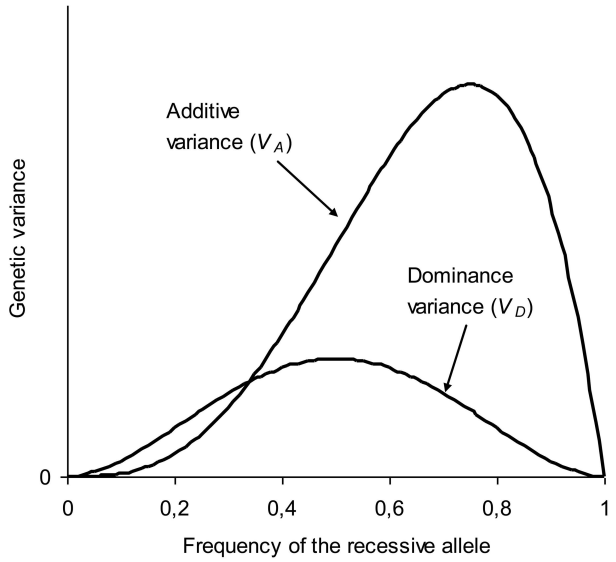

Figure 1: Additive and nonadditive (dominant) quantitative genetic variance arising from a diallelic locus with complete dominance. Even though the alternative allele is completely recessive, genetic variance arising from the locus is mostly additive at intermediate or high frequencies of the recessive allele. Adopted from figure 8.1 of Falconer and Mackay (1996).

do give valuable insight into the genetic architecture of traits.

Empirical Estimates of the Potential Genetic Benefits

A number of studies have been performed in genetically variable animal populations from which it is possible to obtain estimates of both good-genes and compatible-genes effects on life-history traits. We have compiled a list of these studies, estimated the magnitude of the compatible-genes effects in relation to the good-genes effects, and included information about the type of life-history trait measured (table 1). Studies were obtained from a search of the Web of Science using "diallel" and "nonadditive" as search terms. Further references were obtained from a search of the literature cited in relevant publications and by contacting people known to use NCII designs in their work. Studies were chosen on the basis of the following criteria: (1) The population of study animals was a natural population or a genetically diverse managed population that could be assumed to have near-natural genetic composition. (2) Some life-history trait had been estimated from the offspring generation. (3) Authors had calculated the relevant male and male-by-female variance components, or they provided other information from which these variance components could be calculated (see Lynch and Walsh 1998, p. 600). Because in this analysis we were interested only in the genetic benefits of mate choice, we did not analyze the female component of variation, which can be affected by both genetic effects and maternal provisioning.

We found 24 studies altogether reporting 110 estimates for good-genes and compatible-genes variance (table 1). As can be seen in table 1, the proportion of compatible-genes effects in relation to the sum of good-genes and compatible-genes effects varies widely both within and between studies. Some of this variation undoubtedly reflects variation in the true genetic effects, but some is probably also due to differences in study designs and analytical methods. Nevertheless, the mean of all the estimates is 0.46, which suggests that the overall magnitude of compatible-genes effects is comparable to the magnitude of good-genes effects. However, not a single study on compatible genes has examined fitness beyond the very early stages of offspring development. Genetic trade-offs between fitness components weaken the correlation between single fitness components and total fitness, which includes the number of offspring produced and the survival and reproductive success of the offspring (Kokko et al. 2003; Hunt et al. 2004). Thus, the data in table 1 should be taken as only preliminary evidence for the relative importance of compatible-genes benefits. It will be interesting to see whether future studies will reveal increased importance of compatible genes, as could perhaps be expected from the general observation that fitness traits have relatively more nonadditive variation than do nonfitness traits (Crnokrak and Roff 1995; Roff and Emerson 2006).

As table 1 shows, most studies examining the potential compatible-genes benefits have been conducted on species with external fertilization. This has lead to an underrepresentation of many taxonomic groups in the estimation of the potential compatible-genes benefits. However, the analysis can also be performed using species with internal fertilization by setting up several smaller NCII breeding designs where, for example, two males are mated to two females in all four possible combinations (Dawson 1965; Lynch and Walsh 1998). Studies with internally fertilizing species are more complex, because factors such as female age and possible differential investment may influence offspring fitness (Kotiaho et al. 2003). These effects must be controlled for statistically or by using a fertilization method where such effects can be excluded (e.g., artificial inseminations with a mix of sperm from different males; Miller et al. 1963).

Implications for Sexual Selection

Mate choice for good-genes and compatible-genes benefits seems at first to imply very different mating patterns. If mate choice was mainly for good genes, all females should

be congruent in their choice of males, preferring the male with the best good genes. In contrast, if mate choice was primarily for compatible genes, different females should prefer different males. However, good-genes and compatible-genes benefits are not mutually exclusive; as defined here, good-genes and compatible-genes benefits are uncorrelated. Because the benefits are not correlated, females can obtain maximal offspring fitness by optimizing their mate choices with respect to both good-genes and compatible-genes benefits (Colegrave et al. 2002; Roberts and Gosling 2003; Neff and Pitcher 2005; Puurtinen et al. 2005). However, if the benefits of one or the other are larger, more weight in mate-choice decision should (on average) be given to the factor that confers larger fitness benefits. Indeed, a study of major histocompatibility complex (MHC)–congenic laboratory mice has shown such adjustment of mate-choice criteria depending on the variability in cues reflecting good-genes and compatible-genes benefits (Roberts and Gosling 2003).

However, the existence of potential good-genes and compatible-genes benefits does not guarantee that mate choice is always optimized with respect to these benefits. Indeed, mate choice for compatible-genes benefits presents some difficulties, because the choice for genetic compatibility requires that females know their own genotype and the genotypes of their prospective mates. Moreover, because the precise combination of genes in each zygote is affected by random segregation and crossing over in both parents during meiosis, precopulatory mate choice will not result in predictable offspring fitness, as we showed in "Quantitative Genetic Interpretation of the Genetic Benefits of Mate Choice." However, if compatible-genes benefits are mostly due to few linked loci, like the MHC, adaptive mate choice for compatible-genes benefits is facilitated. Indeed, some evidence exists for such choice on the basis of dissimilarity in the MHC, but not all studies report consistent effects (Tregenza and Wedell 2000; Penn 2002; Roberts and Gosling 2003). Another empirically supported case of compatibility choice is avoidance of mating with close kin (Pusey and Wolf 1996). Inbreeding typically results in lowered offspring fitness because recessive deleterious alleles are expressed in the homozygous state in the inbred progeny, and thus it is one form of genetic incompatibility (Tregenza and Wedell 2000). Interestingly, the MHC seems to also be important in this type of compatibility choice because it plays a key role in vertebrate kin recognition (Penn 2002).

Postcopulatory female choice presents a possibility to circumvent some of the difficulties in obtaining compatible-genes benefits. If the female, or indeed the egg, could detect the haploid genotype of the sperm, compatibility choice would be easier because the difficulties arising from segregation and crossing-over are not present at the gametic level (Birkhead and Pizzari 2002). Also, selective abortion of incompatible zygotes can result in compatible-genes benefits if abortion of incompatible zygotes releases resources or parental care that can be directed to the provisioning of higher-quality zygotes (Birkhead and Møller 1998). At the moment, there is only limited evidence for postcopulatory selection on the basis of the haploid genotype of sperm in species with males and females (Wedekind et al. 1996; Rülicke et al. 1998; Simmons 2005; Firman and Simmons 2008). However, haploid sperm choice is well documented in sessile hermaphroditic species, where it probably evolved for avoidance of self fertilization (Birkhead and Pizzari 2002).

Since the introduction of Darwin's theory of sexual selection (Darwin 1871), there has been intense debate over the importance of mate choice in sexual selection. Although the current paradigm is heavily focused on the importance of female choice and genetic benefits, the role of male-male competition in sexual selection is often difficult to exclude (Kotiaho and Puurtinen 2007). Interestingly, choice based on compatible-genes benefits presents a case of mate choice that cannot be explained by male-male competition. Thus, demonstrations of mate choice for compatible-genes benefits provide the least ambiguous evidence for the existence of active mate choice for genetic benefits. Curiously, however, choice for compatible-genes benefits is not sexual selection sensu stricto. The classic textbook definitions of sexual selection stress that sexual selection is a process whereby some individuals of a given sex have greater mating success than others; that is, there is mating bias, and differences in the mating success are related to the expression of a secondary sexual trait (Darwin 1871; Andersson 1994). Choice for compatible-genes benefits does not generate mating bias, nor does it involve a secondary sexual trait. Mate-choice behavior and physiological traits involved in detection of compatibility can, however, evolve to exploit compatible-genes benefits, and compatibility may thus play an important role in the evolution of reproductive systems. Indeed, sexual selection was narrowly defined for historical reasons: it was Darwin's attempt to negate the arguments that natural selection could not explain the evolution of traits that decrease individual survival. The dichotomy of natural and sexual selection has long since been understood to be illusory: selection acts on the reproductive fitness to which both survival and mating success contribute.

Concluding Remarks

The two main questions that remain to be addressed in the future are the magnitude of the indirect genetic benefits and whether mate choice for these benefits actually occurs. Many empirical studies have shown that mate choice can increase at least some offspring fitness components (e.g.,

Boake 1985; Reynolds and Gross 1992; Norris 1993; Petrie 1994; Alatalo et al. 1998; Wedell and Tregenza 1999), but only a handful of studies have estimated the net fitness consequences of mate choice including survival, fecundity, and mating success of offspring (Jones et al. 1998; Fedorka and Mousseau 2004; Head et al. 2005; Qvarnström et al. 2006; Rundle et al. 2007). Even less is known about the fitness effects of sexual selection for compatible-genes benefits, because no studies focusing on genetic compatibility have tracked the fitness of offspring beyond the very early stages of development.

The most important unanswered question is whether potential genetic benefits determine the mating patterns observed in natural populations. There are formidable challenges in determining whether the observed mating patterns are the result of active mate choice for genetic benefits, sensory exploitation, male-male competition, or mate choice for direct benefits (Kotiaho and Puurtinen 2007). Bearing in mind the difficulties associated with inferring the ultimate reasons for observed mating patterns, there are nevertheless insights to be gained from studies that simultaneously assess mating patterns and the genetic benefits of mate choice. One possible experimental setup that could reveal the role of genetic benefits in determining mating patterns would be to record the mate preferences of a group of males and females in behavioral tests and then assess the good-genes and compatible-genes benefits for the same group by executing a NCII breeding design and recording offspring fitness (Casalini et al. [2009] performed this type of study, but with maternal half-sibs only). With information from behavioral trials and NCII, it would be possible to determine whether mate choice correlates with good-genes or compatible-genes benefits or whether it can actually be optimized with respect to both good-genes and compatible-genes benefits, which would yield the maximal offspring fitness.

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