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Mate choice for non-additive genetic benefits: A resolution to the lek paradox

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ABSTRACT

In promiscuous mating systems, females often show a consistent preference to mate with one or a few males, presumably to acquire heritable genetic benefits for their offspring. However, strong directional selection should deplete additive genetic variation in fitness and consequently any benefit to expressing the preference by females (referred to as the lek paradox). Here, we provide a novel resolution that examines non-additive genetic benefits, such as overdominance or inbreeding, as a source of genetic variation. Focusing on the inbreeding coefficient f and overdominance effects, we use dynamic models to show that (1) f can be inherited from sire to offspring, (2) populations with females that express a mating preferences for outbred males (low f) maintain higher genetic variation than populations with females that mate randomly, and (3) preference alleles for outbred males can invade populations even when the alleles are associated with a fecundity cost. We show that non-additive genetic variation due to overdominance can be converted to additive genetic variation and becomes "heritable" when the frequencies of alternative homozygous genotypes at fitness loci deviate from equality. Unlike previous models that assume an infinite population size, we now show that genetic drift in finite populations can lead to the necessary deviations in the frequencies of homozygous genotypes. We also show that the "heritability of f," and hence the benefit to a mating preference for non-additive genetic benefits, is highest in small populations and populations in which a smaller number of loci contribute to fitness via overdominance. Our model contributes to the solution of the lek paradox.

1. Introduction

Evolutionary and behavioral ecologists have long been interested in mate choice and particularly in understanding why females of many species are highly selective when it comes to mating (Darwin, 1871; Bateson, 1983; Andersson, 1994; Kokko et al., 2003; Neff and Pitcher, 2005). In resource-based mating systems, it has been relatively straightforward to show that females prefer males that provide the most resources such as food, shelter, parental care, and protection from predators (reviewed in Møller and Jennions, 2001). However, there are many other mating systems in which females receive no resources from males (non-resource-based mating systems), yet females still express a preference among males. One such mating system that has attracted considerable attention is the lek.

Lek mating systems typically involve a congregation of displaying males, and females visiting the lek often show congruence in their mating preference for one or a few males that have the most elaborate display (e.g. secondary sexual trait; Höglund and Alatalo, 1995). Presumably, choosy females receive a genetic benefit ("good genes") that increases the survivorship or mating success of their offspring as compared with the genes provided by less desirable males (reviewed in Kokko et al., 2003; Mead and Arnold, 2004). Empirical research on lek mating systems, as well as other non-resource-based mating systems, have confirmed the association between mate preference and increased offspring viability, although the fitness effects appear small at only a few percent (Møller and Alatalo, 1999; Jennions et al., 2001; also see Kirkpatrick and Barton, 1997). Furthermore, strong directional selection on the "best" males should deplete the underlying additive genetic variation in the secondary sexual trait and more generally in fitness. Consequently, after several generations, there should be little fitness benefit to expressing the preference by females; this is referred to as the lek paradox (Kirkpatrick and Ryan, 1991).

Early resolutions to the lek paradox have focused on genetic mutations as a mechanism to maintain additive genetic variation in fitness (Pomiankowski and Møller, 1995; Rowe and Houle, 1996; reviewed in Tomkins et al., 2004; Kotiaho et al., 2008). For example, Rowe and Houle (1996) postulated that additive genetic variation could be maintained if the secondary sexual trait was condition dependent. Condition dependence implies that expression of the trait is linked to an individual's condition, which would be influenced by many loci. Thus, condition dependence could

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provide a large mutational target for the trait. Pomiankowski and Møller (1995) instead proposed that sufficient numbers of modifier loci underlying the secondary sexual trait could similarly increase the mutational target of the trait and maintain additive genetic variation. However, these resolutions have been challenged because mutation alone may not be sufficient to maintain variation in fitness (Møller and Alatalo, 1999; Jennions et al., 2001; Tomkins et al., 2004), especially to compensate for the cost of expressing the preference (Taylor and Williams, 1982; Kirkpatrick and Barton, 1997; but see Reynolds and Gross, 1990).

Recently, it has been speculated that mate choice for non-additive genetic benefits could provide a resolution to the paradox (Neff and Pitcher, 2005; Reid, 2007; Hoffman et al., 2007). Such benefits depend on non-additive genetic variation, which involves the interaction between homologs at a gene locus (over- or under-dominance) or the interaction between gene loci (epistasis). However, this variation is expected to be heritable in only specific circumstances (Lynch and Walsh, 1998; for an example see Mitton et al., 1993) and theoretical analyses indicate that preference alleles for non-additive genetic benefits are unlikely to evolve, particularly when the preference is associated with a fitness costs such as reduced fecundity (Irwin and Taylor, 2000; Lehmann et al., 2007).

(Irwin and Taylor, 2000; Lehmann et al., 2007) used mathematical models to examine the evolution of female preferences for non-additive genetic benefits conveyed by heterozygous males (i.e. overdominance). Both models assume an infinite population size and examine a single preference gene and a single viability gene, each with two alleles. Both the models show that the preference allele is neutral when there is symmetric overdominance at the viability locus (i.e. the fitness of the heterozygote is greater than that of the homozygotes, and the fitness of both homozygotes are equal). In this case, both viability alleles are equally common in the population at Hardy–Weinberg equilibrium, and regardless of whether a female mates randomly or only with heterozygous males, she will always generate offspring that are half heterozygous and half homozygous. In contrast, when there is asymmetric overdominance (i.e. the fitness of one homozygote is lower than the other homozygote), mating with heterozygous males leads to an increased production of the less fit homozygote, as compared with random mating, and the preference allele is actually selected against even though the female produces more heterozygous offspring than a randomly mating female (Irwin and Taylor, 2000).

Lehmann et al. (2007) showed that the preference allele can be selected for when there is symmetric overdominance and a biased mutation rate at the viability locus. The biased mutation rate is required to ensure that the two homozygotes are not equally frequent in the population. In this case, females who mate only with heterozygous males produce more heterozygous offspring than females who mate randomly. As in the asymmetric overdominance scenario, the choosy females still produce more offspring of the less common homozygous genotype than randomly mating females. However, in this case, the fitness of the choosy females is not reduced relative to females who mate randomly because the two homozygotes have equal fitnesses. The heritability of sire heterozygosity increases as the frequencies of the alternative homozygous genotypes deviate from equality. As such, this mechanism is opposite to the conversion of dominance variance to additive variance discussed by other models (Willis and Orr, 1993; Van Buskirk and Willi, 2006). Although biased mutation rates at fitness loci may be common in many populations (e.g. Lin et al., 1998; Lynch et al., 1999; Vassilieva et al., 2000), Lehmann et al. (2007) suggested that preferences for heterozygous males can evolve only under restrictive conditions.

Here, we use dynamic models to determine if non-additive genetic variation associated with overdominance effects can be inherited from parent to offspring and if preference alleles for non-additive genetic benefits can evolve even when they are associated with a fitness cost. We use dynamic models because they allow us to incorporate key evolutionary forces including genetic drift, which is expected to be influential in small populations but has been largely ignored in previous modeling approaches (Irwin and Taylor, 2000; Lehmann et al., 2007). Genetic drift should have the same effect as the biased mutation rate modeled by Lehmann et al. (2007) with respect to the relative frequencies of the alternative homozygous genotypes at a viability locus. The models also allow us to examine the effects of natural and sexual selection on population genetic variation in fitness.

2. Model

2.1. Inheritance of *f*

Our models were programmed using the C++ computer language. We focus on the inbreeding coefficient *f*, which is one important non-additive genetic effect (Falconer and Mackay, 1996). The inbreeding coefficient represents the proportion of homologous alleles in a diploid individual that are identical by descent; *f* can range from 0 (outbred, no homologs are identical by descent) to 1 (inbred, all homologs are identical by descent). We assume that natural selection via inbreeding depression follows a negative linear function (Crnokrak and Roff, 1999). Thus, our approach is analogous to modeling the inheritance of heterozygosity under an overdominance model of selection.

We first simulated a population with a stable population size of *N* and a 1:1 sex ratio. Each generation, females either selected mates randomly or based on one of two preference functions for less inbred males (males with lower *f*) defined by the equations: (1) $Pr_{mating} = 1-f$ or (2) $Pr_{mating} = 1-f^{1/4}$. The first preference function is linear with respect to *f* and reflects a moderate intensity of sexual selection. The second function is non-linear (declining exponentially) and reflects a high intensity of sexual selection. The mating system was assumed to be polygynous such that a male could mate with multiple females. Each generation, offspring were generated following Mendelian inheritance patterns at either 10, 100, or 1000 fitness loci. Inbreeding depression was modeled using the function: survivorship = 1–0.818*f*, which was derived in Crnokrak and Roff (1999) for homeotherms (similar results were found when the function for poikilotherms was instead used; data not shown). For comparison, other sets of simulations assumed no inbreeding depression and omitted the natural selection function.

Novel genetic variation was introduced by mutations following the infinite alleles model (IAM) with a rate of μ mutations per locus per generation. Thus, each mutation gave rise to a unique allele and any two equivalent alleles were identical by descent. The populations were allowed to "evolve" for 5000 generations to ensure a mutation-drift-selection equilibrium was achieved (Shriver et al., 1993). Because we used the IAM, the inbreeding coefficient *f* for each individual—i.e. the proportion of homologs that were identical by descent—could easily be calculated from the proportion of loci that were homozygous. The "heritability of *f*" was estimated from the slope of the regression of mid-parent *f* (calculated for all pairs of males and females) and expected offspring *f* (Falconer and Mackay, 1996). We considered parameters of population size $N = \{50, 100, 1000\}$ and mutation rate $\mu = \{10^{-2}, 10^{-4}\}$. The mutation rates include the upper and mean rate expected for higher eukaryotes (e.g. assuming a genome size of ca. 10^4 genes and 0.1–100 mutations per genome per

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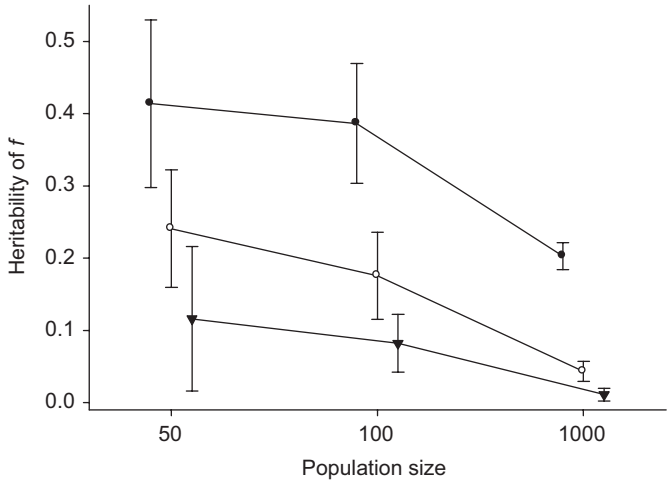

Fig. 1. The heritability of the inbreeding coefficient *f*. The plot summarizes the slope of the regression line between mid-parent and offspring *f* given a randomly mating population of size 50, 100, or 1000 breeders that experiences inbreeding depression. Filled circles denote a mutation rate of 10^{-4} at 100 fitness loci, open circles denote a mutation rate of 10^{-2} and 100 fitness loci, and filled triangles denote a mutation rate of 10^{-2} at 1000 fitness loci. Error bars represent ± 1 SD and are based on 25 replicate populations that evolved for 5000 generations. The data points are offset for clarity and the *x*-axis is not linear.

generation; Drake et al., 1998). Twenty-five populations were modeled for each combination of parameters providing at total of 2700 populations (25 populations $\times$ 3 N s $\times$ 2 μs $\times$ 3 mating preference functions $\times$ 2 natural selection functions $\times$ 3 numbers of fitness loci).

Because we expected that the correlation between parent and offspring *f* would depend in part on deviations from equality in the frequencies of alternative homozygous genotypes (Lehmann et al., 2007), for a subset of the parameter sets ($N = \{50, 100\}$, $\mu = \{10^{-2}, 10^{-4}\}$, Natural selection function, random mating, 100 fitness loci) we calculated the standard deviation in the frequency of each possible homozygous genotype at each locus. We then took the mean standard deviation across all loci and used an ANCOVA controlling for population size and mutation rate to determine if there was in fact a positive correlation between the mean deviation and the magnitude of the correlation between parent and offspring *f*.

2.2. Preference evolution

We modified the previous model to examine the evolution of a mate preference for non-additive genetic benefits. For the first 5000 generations, the population evolved with inbreeding depression but with females mating randomly. Next, the

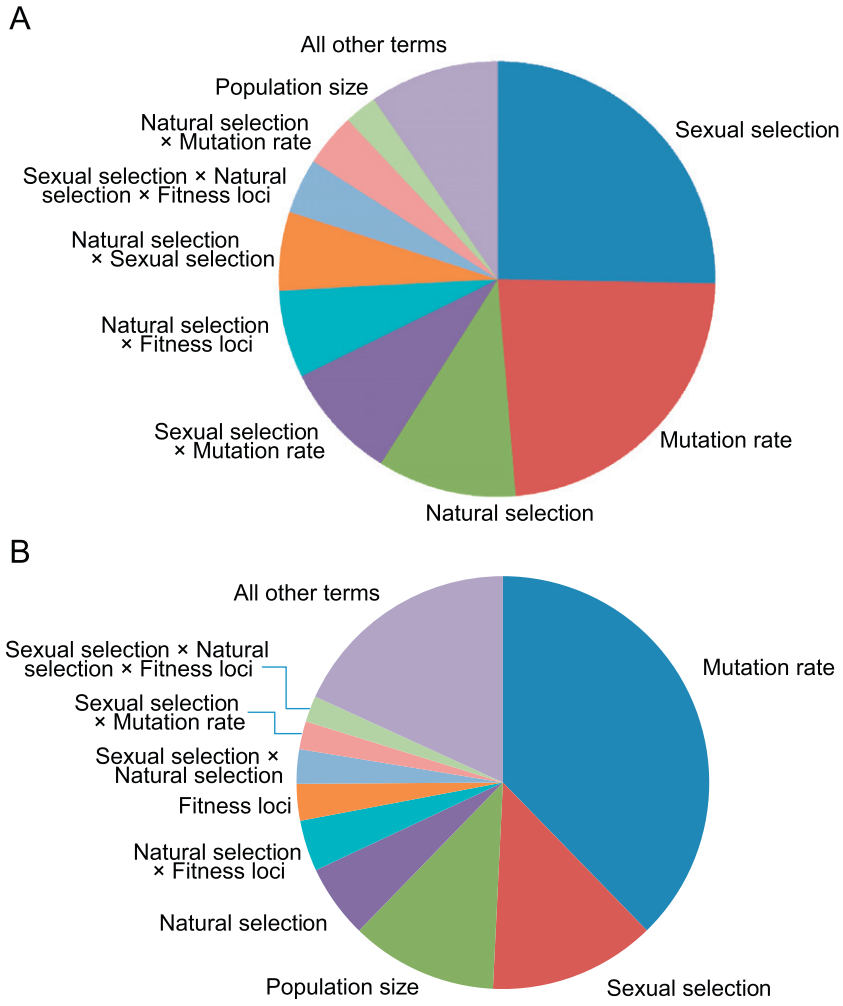

Fig. 2. Mixed-model ANOVA results. The pie charts show the proportion of the total sum of squares assigned to sexual selection via female preference, natural selection via inbreeding depression, mutation rate, population size, and number of fitness loci on (A) the heritability of *f* and (B) the mean population *f*. See Table S1 for further details.

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moderate female preference allele for less inbred males was introduced with a mutation rate of 10^{-3}. The reciprocal mutation at the same rate took the preference allele back to random mating. Thus, there was no bias in mutation rate for or against the preference allele. The preference allele was assumed to be dominant to the random mating homolog. The population was allowed to evolve for an additional 5000 generations and the proportion of females expressing the mating preference was determined. We examined the same parameter set as above for N and μ, but also included a cost to the preference in terms of reduced fecundity of $C = \{0, 0.005, 0.01\}$; the intermediate value represents a cost commonly assumed in other models (e.g. Lehmann et al., 2007). We considered 50 populations for each parameter set and report the results for the simulations involving 100 fitness loci (qualitatively similar results were found with 10 or 1000 fitness loci).

All statistics were performed using JMP (v. 4). Means are reported plus or minus one standard deviation.

3. Results

The simulation revealed that non-additive genetic variation such as f can be inherited from parent to offspring. Generally, the heritability of f increased with decreasing population size, mutation rates and numbers of fitness loci (Fig. 1). For example, under the random mating scenario with natural selection via inbreeding depression, a mutation rate of 10^{-2}, and a population size of 50, the heritability of f was 0.27 ± 0.16 for 10 loci, 0.24 ± 0.08 for 100 loci, and 0.12 ± 0.10 for 1000 loci. Introduction of either preference function also reduced the heritability (Fig. 2A, Table S1); for the population of 50 individuals noted above, with 100 fitness loci, the heritability dropped by about 0.05 to a value of 0.19 ± 0.09 when the moderate strength preference function was included. Conversely, omitting both the preference function and natural selection led to higher heritability; the heritability was 0.28 ± 0.10 for the same population of 50 individuals and 100 fitness loci. Based on the sum of squares from a mixed-model ANOVA, the top three factors affecting heritability were sexual selection, mutation rate, and natural selection (Fig. 2A). We also found that there was a positive relationship between the heritability and the mean standard deviation in the frequency of homozygous genotypes across the fitness loci (ANCOVA, mean standard deviation term: $r^2 = 0.19$, $F_{1,95} = 10.7$, $P = 0.002$). Thus, as expected, the heritability also depended on the relative frequencies of homozygous genotypes at each fitness locus.

Using a mixed-model ANOVA, we found that female preference, natural selection, mutation rate, population size, and number of fitness loci each independently affected population genetic variation as measured by the mean inbreeding coefficient f (Figs. 2B and 3). Inclusion of either preference function or the inbreeding depression function reduced the population mean f and thus increased genetic variation. These effects were greater at the lower mutation rate of 10^{-4} (Fig. 3). Generally, the preference (sexual selection) had contributions to genetic variation similar in magnitude as population size and greater in magnitude than natural selection (inbreeding depression; Figs. 2B and 3). There also were significant interactions between female preference, natural selection, mutation rate, population size, and the number of fitness loci indicating that the effect of each of these parameters on population genetic variation varied across the parameters tested (Table S1). Examining the mean number of unique alleles per locus in the population provided similar results to the f parameter (data available from the authors).

A preference allele for outbred males (low f) was able to invade into populations even when the allele was associated with a 0.5% or 1% reduction in fecundity (Figs. 4 and 5). In small populations

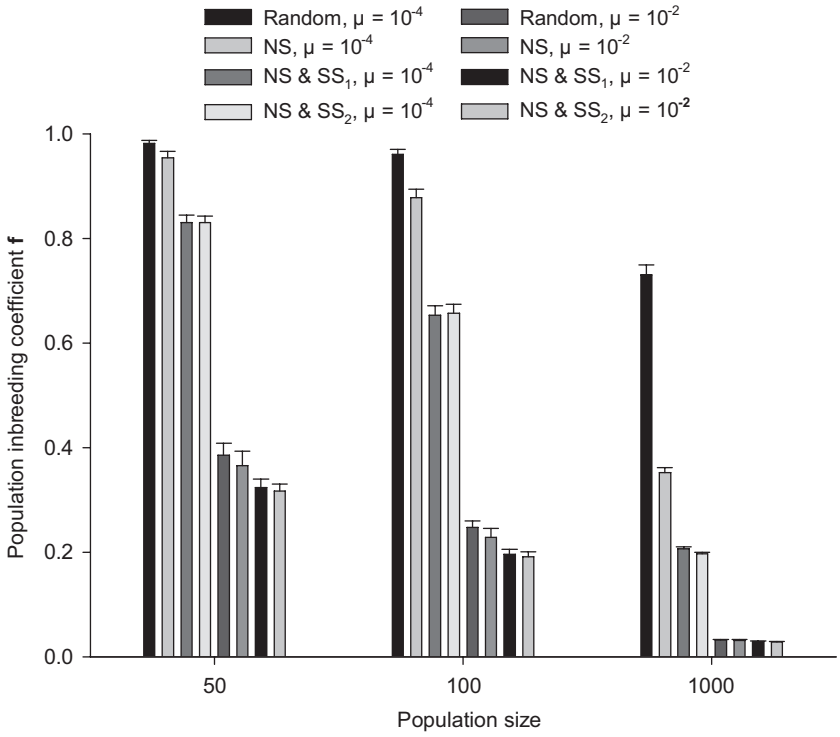

Fig. 3. The effects of female mating preference and natural selection on the population inbreeding coefficient f. The simulated populations incorporated random mating and survival (random), random mating and natural selection via inbreeding depression (NS), or natural selection and sexual selection (SS) via a female mating preference (moderate intensity, NS & SS_1, or high intensity, NS & SS_2). Two mutation rates were considered at 100 fitness loci. Error bars represent 1 SD and are based on 25 replicate populations that evolved for 5000 generations.

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($N = 50$ individuals), the preference allele appeared to drift with the populations repeatedly returning to a state where all females expressed the preference for less inbred males (Figs. 4A, 5A, and C). As expected given the influence of the preference on population f, there was a negative correlation between the mean population f and the proportion of females expressing the preference (Spearman's $r < -0.63$). Thus, f was lower when a greater proportion of females in the population were expressing the preference. In large populations ($N = 1000$ individuals), the fecundity cost reduced the frequency of the preference allele and consequently the proportion of females expressing the preference at any given time. For example, at a mutation rate of 10^{-4} and when there was no cost associated with the preference allele, about 80% of females expressed the preference at equilibrium; when there was a cost of 0.5%, this proportion dropped to just over 40% of females (Fig. 4B). This difference in frequency was less apparent at the higher mutation rate of 10^{-2} (Fig. 5B). The preference allele, however, was still consistently able to invade into the population. Simulations involving either 10 or 1000 fitness loci provided similar results; a recessive preference allele also was able to invade populations (data available from the authors).

4. Discussion

We have used dynamic models to show that non-additive effects such as those relating to the inbreeding coefficient f can be inherited from parent to offspring. We found that the heritability of f is greatest in populations characterized by a small number of breeders, a low mutation rate, and a small number of loci affecting

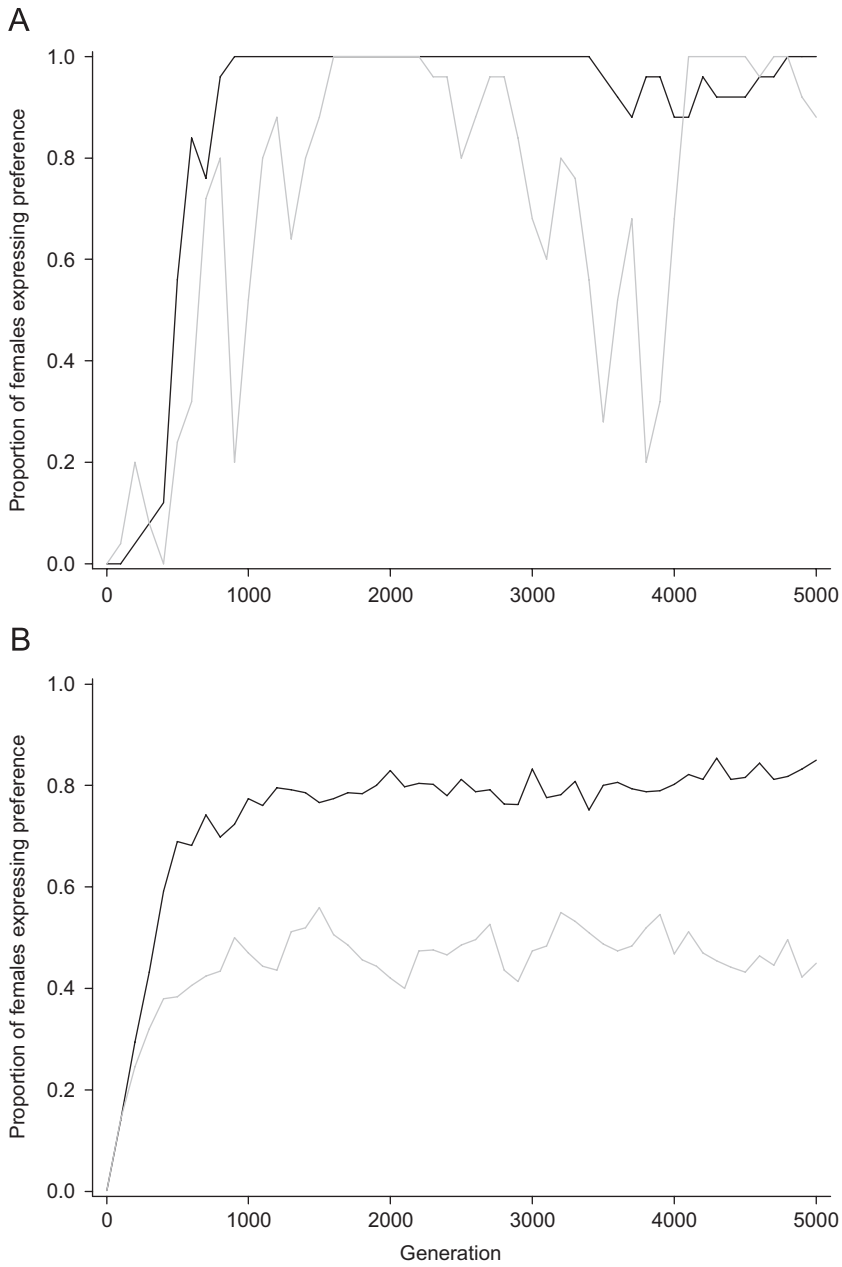

Fig. 4. The evolution of a female preference allele for less inbred males. Plots show the proportion of females expressing the preference in populations of size (A) 50 individuals or (B) 1000 individuals. Black lines denote no cost and gray lines denote a cost of a 0.5% reduction in female fecundity associated with the expression of the preference. Each line represents median values based on 50 replicate populations with a mutation rate of 10^{-4} at 100 fitness loci.

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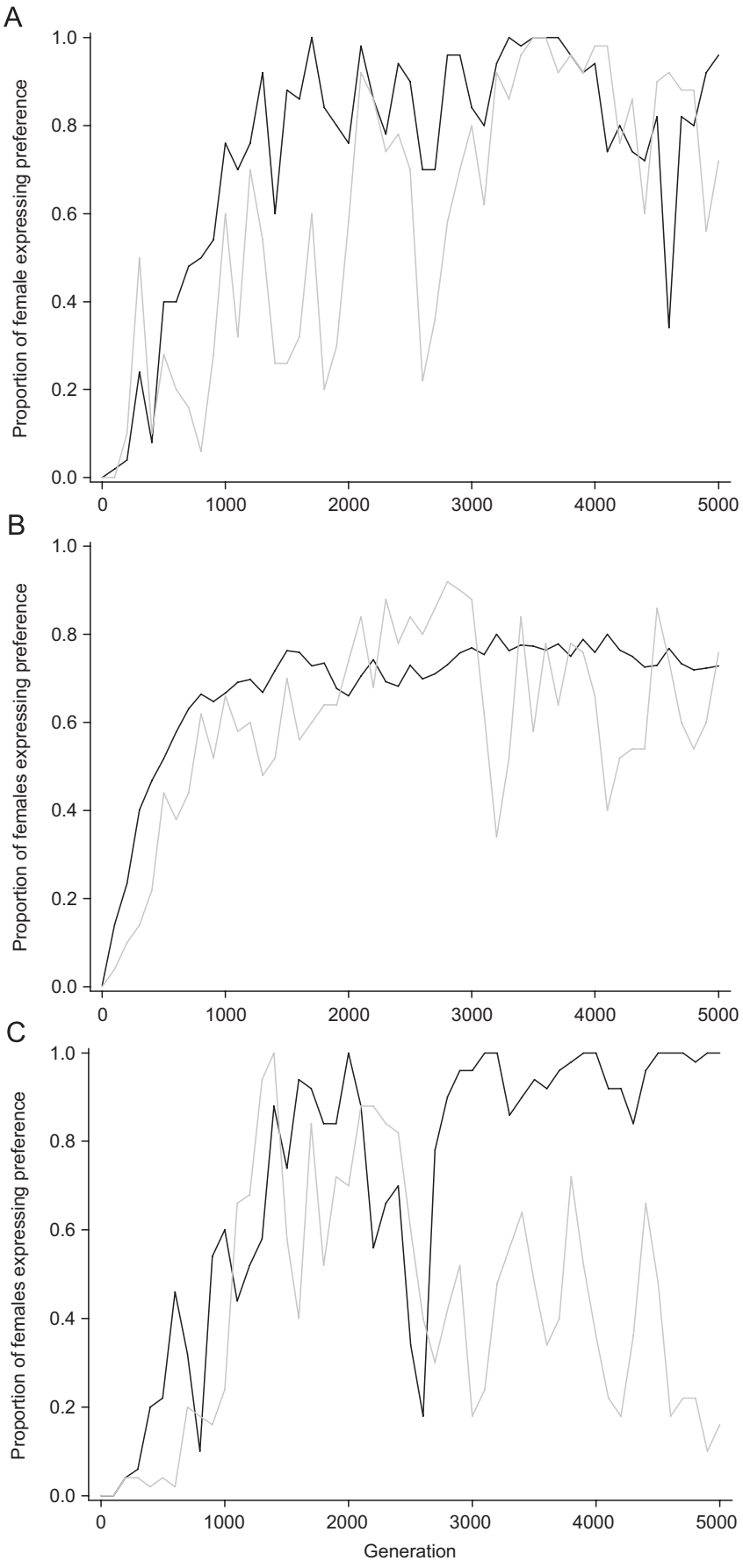

Fig. 5. The evolution of a female preference allele for less inbred males. Plots show the proportion of females expressing the preference in populations with parameters (A) population size $N = 50$, mutation rate $\mu = 10^{-2}$, and preference fecundity cost $C = 0$ (black line) or $C = 0.5\%$ (gray line), (B) $N = 1000$, $\mu = 10^{-2}$, and $C = 0$ (black line) or $C = 0.5\%$ (gray line), and (C) $N = 50$, $C = 1\%$, and $\mu = 10^{-4}$ (black line) or $\mu = 10^{-2}$ (gray line). Each line represents median values based on 50 replicate populations and 100 fitness loci.

fitness due to inbreeding depression as modeled by overdominance. We also found that a female preference for outbred males can invade small populations even when associated with a fecundity cost of 1%. Furthermore, the preference actually increases population genetic variation. Given that many of the mating systems from which the lek paradox originally emerged are small (Table 1), our model suggests that in these populations the heritability of f is at least 0.12 (range = 0.12–0.41). This value is similar in magnitude to empirical estimates for the heritability of life history traits (e.g. Van Buskirk and Willi, 2006). Thus, mate choice for non-additive fitness benefits may be a major yet largely overlooked component of sexual selection.

Previous researchers have proposed several potential resolutions to the lek paradox (reviewed in Tomkins et al., 2004; Kotiaho et al., 2008). These resolutions are based on the expectation that sexual ornaments are expressed in proportion to male genetic condition (e.g. Rowe and Houle, 1996; Houle, 1998); mutation being able to create substantial new genetic variation in sexually selected traits (e.g. Pomiankowski and Møller, 1995; Petrie and Roberts, 2007); direct selection being more important than indirect selection (e.g. Reynolds and Gross, 1990); and maternal phenotypes (e.g. habitat selection behavior) influencing the condition and expression of secondary sexual traits in sons (i.e. indirect genetic effects, Miller and Moore, 2007). The solution proposed by Rowe and Houle (1996) arguably has received the most support; however, this solution still relies on mutation to maintain sufficient additive genetic variation, which may not be sufficient to counter act the costs of mating preferences in small populations. Conversely, our model, which deals with non-additive genetic variation and its conversion to heritable variation, is most applicable in small populations and may provide an alternative solution to the paradox.

Why is there a correlation between parent and offspring f? Several empirical studies have found such a correlation including in, for example, song sparrows, *Melospiza melodia* (Reid et al., 2006; for an example with heterozygosity see Hoffman et al., 2007). Lehmann et al. (2007) argued that such inheritance occurs in populations characterized by a biased mutation rate. The biased mutation rate ensures that homozygotes fixed for alternative alleles are not equally frequent in the population. As an example, consider a single locus with two alleles, *A* and *a*. Suppose that *AA* and *aa* individuals are 25% common and *Aa* individuals are 50% common in the population. Under random mating, both homozygous and heterozygous females will always on average generate equal proportions of homozygous and heterozygous offspring (Table 2). Consequently, there will be no heritability of the non-additive effect (i.e. heterozygosity in this case).

In contrast, a biased mutation rate will lead to the *A* and *a* alleles differing in frequency in the population and one of the two homozygous genotypes will be more common than the other (say *AA* is 0.49 common and *aa* is 0.09 common; Table 2). Females who mate only with heterozygous males still produce an average of 50% heterozygous offspring. However, because one of the homozygous genotypes is more common in the population, females who instead mate randomly produce fewer than 50% heterozygous offspring (42% in our scenario) and consequently, heterozygosity becomes "heritable." Biased mutation rates may be common in many populations, particularly at fitness loci (e.g. Lin et al., 1998; Lynch et al., 1999; Vassilieva et al., 2000). Furthermore, we have shown that genetic drift can have the same effect as a biased mutation rate with respect to the frequency of

Table 1
Examples of lek mating systems

Species	Lek size	Reference
Sage grouse (*Centrocercus urophasianus*)	2–200	Bradbury et al. (1989)
Black grouse (*Tetrao tetrix*)	3–17	Höglund and Alatalo (1995)
Cock-of-the-rock (*Rupicola rupicola*)	25–61	Trail (1987)
Manakins (*Pipra filicauda, Pipra pipra, Lepidothrix coronota, Chiroxiphia pareola*)	3–19	Loiselle et al. (2007)
Tungara frog (*Physalaemus pustulosus*)	44–425	Ryan et al. (1981)
Tephritid fruit flies (*Ceratitis capitata, Bactrocera dorsalis*)	2–20	Shelly (2001)

Data include species name and lek size as an estimate of population size.

Table 2
Proportion of heterozygous offspring produced by females that mate either randomly or only with heterozygous males (non-random)

Female genotype	Male genotype			Proportion heterozygous offspring
Homozygous genotypes equally common				
Random	*AA* (0.25)	Aa (0.50)	aa (0.25)	**0.5**
AA (0.25)	**0** (0.25 × 0.25 × 0)	**0.0625** (0.25 × 0.5 × 0.5)	**0.0625** (0.25 × 0.25 × 1)	0.125
Aa (0.50)	**0.0625** (0.5 × 0.25 × 0.5)	**0.125** (0.5 × 0.5 × 0.5)	**0 0625** (0.5 × 0.25 × 0.5)	0.25
aa (0.25)	**0.0625** (0.25 × 0.25 × 1)	**0.0625** (0.25 × 0.5 × 0.5)	**0** (0.25 × 0.25 × 0)	0.125
Non-random	*AA* (0.25)	Aa (0.50)	Aa (0.25)	**0.5**
AA (0.25)	–	**0.125** (0.25 × 1 × 0.5)	–	0.125
Aa (0.50)	–	**0.25** (0.5 × 1 × 0.5)	–	0.25
aa (0.25)	–	**0.125** (0.25 × 1 × 0.5)	–	0.125
Homozygous genotypes unequally common				
Random	*AA* (0.49)	Aa (0.42)	aa (0.09)	**0.42**
AA (0.49)	**0** (0.49 × 0.49 × 0)	**0.1029** (0.49 × 0.42 × 0.5)	**0.0441** (0.49 × 0.09 × 1)	0.147
Aa (0.42)	**0.1029** (0.42 × 0.49 × 0.5)	**0.0882** (0.42 × 0.42 × 0.5)	**0 0189** (0.42 × 0.09 × 0.5)	0.21
aa (0.09)	**0.0441** (0.09 × 0.49 × 1)	**0.0189** (0.09 × 0.42 × 0.5)	**0** (0.09 × 0.09 × 0)	0.063
Non-random	*AA* (0.49)	Aa (0.42)	Aa (0.09)	**0.5**
AA (0.49)	–	**0.245** (0.49 × 1 × 0.5)	–	0.245
Aa (0.42)	–	**0.21** (0.42 × 1 × 0.5)	–	0.21
aa (0.09)	–	**0.045** (0.09 × 1 × 0.5)	–	0.045

Numbers in parentheses denote the frequency of matings between females and males of the given genotypes and either random or non-random mating; the third number in the series is the proportion of heterozygous offspring produced from the mating. The right-hand column provides the row sums and the bold and underlined number is the total for the given scenario. Note that symmetric overdominance as we have modeled can reduce the frequencies of homozygotes within the population, but does not change the relative proportion of the two homozygotes.

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homozygous genotypes in the population. Indeed, we found that, independent of other population parameters such as population size and mutation rate, the correlation between parent and offspring f is higher when there is greater variation in the frequency of homozygous genotypes at the fitness loci. Because the influence of genetic drift is inversely related to population size, the correlation between parent and offspring f (or heterozygosity) is highest in small populations.

The fitness benefits derived from a female preference for non-additive effects are greatest in small populations. Independent of the intensity of inbreeding depression, the correlation between parent and offspring f is higher in small populations and thus, all else being equal, choosy females are more likely to obtain the non-additive genetic benefits in smaller populations. In addition, despite the increased susceptibility to genetic drift, the preference should be more likely to evolve in small than large populations. Indeed, we found that the preference allele for outbred males obtained a higher frequency in small versus large populations. For example, when associated with a fecundity cost of 0.5%, the preference allele repeatedly went to fixation in populations of 50 individuals whereas only about 80% of females expressed the preference in populations of 1000 individuals. These data indicate that in the latter population the preference allele becomes effectively neutral when about 80% of females express the preference and 20% of females mate randomly. Consequently, there is a genetic polymorphism in mating preference—with some females preferring outbred males and other females mating randomly—that exists in an evolutionary stable state (also see Neff and Pitcher, 2005).

Mate choice for non-additive benefits likely will play a more significant role when fewer loci are involved. We found that the heritability of f was greater when fewer loci contributed to fitness via overdominance. This dependency on loci number probably reflects the stochastic nature of genetic drift—the more loci contributing to fitness, the less likely it is that homozygous genotypes deviate in frequency across all loci. Although we modeled genome-wide effects via f, our model is applicable to any fitness-related trait that is coded by loci showing overdominance effects. Indeed, it is possible that the expression of some secondary sexual traits will be influenced by small numbers of loci displaying overdominance. On the other hand, condition-dependent traits are likely to be influenced by larger numbers of loci in so much as condition captures genome-wide effects (Rowe and Houle, 1996; for an exception see Abzhanov et al., 2006). However, there are few studies addressing the genomic architecture of sexually selected traits and as such it remains unclear as to the numbers of loci that actually contribute to these traits (e.g. Fitzpatrick, 2004; Tatsuta and Takano-Shimizu, 2006; Nadeau et al., 2007; also see Chenoweth and Blows, 2006). Nevertheless, our data indicate that, even though f can be heritable when inbreeding is governed by 1000 fitness loci, non-additive effects will play a larger role in mate choice when smaller numbers of loci are involved. This may be the case for sexually selected traits that are not linked to condition, or when condition is affected by a few loci with strong overdominance effects.

Although the lek paradox originated from non-resource-based mating systems, the paradox can apply to any mating system where there is directional sexual selection. For instance, one important example comes from the inbred population of song sparrows on Mandarte Island, Canada (Reid, 2007). The population consists of an average of only 35 breeding pairs that are socially monogamous with both parents provisioning young (Smith et al., 2006). Nevertheless, females are genetically promiscuous with more than a quarter of the young being sired by extra-pair males (O'Connor et al., 2006). Females select mates in part based on the complexity of males' courtship song; females prefer more complex songs (Reid et al., 2004). Thus, there is directional selection on song complexity and given that song complexity is heritable (Reid et al., 2006), the preference should deplete any underlying additive genetic variation in the trait. However, because outbred males (low f) produce more complex songs, the preference results in females selecting outbred males as mates (Reid et al., 2005). Furthermore, the inbreeding coefficients appear to be inherited as there is a positive correlation between parent and offspring f (Reid et al., 2006). Our model not only corroborates their data showing that in small populations there is a positive correlation between parent and offspring f—i.e. non-additive genetic variation can be converted to additive genetic variation—but also shows that directional preferences can increase population genetic variation in fitness. Thus, it is conceivable that the mating preference for song complexity and hence outbred males not only provides an indirect benefit (reduced inbreeding depression; Keller et al., 1994) but also helps to maintain genetic variation in a perpetually small population.

In conclusion, we have shown that non-additive genetic variation such as f can be inherited from parent to offspring thereby representing a conversion of non-additive genetic variation to additive genetic variation. Furthermore, directional selection on traits showing overdominance fitness effects can actually increase genetic variation in the population. Our results thereby provide a novel resolution to the lek paradox. Additional empirical studies are now needed that examine the genetic architecture of secondary sexual traits. We anticipate that the expression of many secondary sexual traits may have a strong non-additive genetic component.

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Appendix A. Supporting Information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jtbi.2008.05.019.

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