

Heterozygous advantage and the evolution of female choice

Andrew J. Irwin* and Peter D. Taylor

Department of Mathematics and Statistics, Queen's University, Kingston, Ontario K7L 3N6, Canada

ABSTRACT

The evolution of a gene for female choice of mates with heterozygous advantage at a single locus is discussed. Recent analyses of this problem have been mathematically unclear and misleading. A genetic calculation for a hypothetical one-locus, diallelic species shows that the choice gene is not favoured in a static environment. Computations are presented which show that the choice gene can be favoured in a fluctuating environment where the relative superiority of the homozygotes changes.

Keywords: female choice, fluctuating environment, heterozygote advantage.

INTRODUCTION

It is a common suggestion that increased heterozygosity may be associated with increased fitness. Heterozygosity has been correlated with increased organism viability, developmental stability, growth rate, fecundity and mating opportunities (Brown, 1997; Mitton, 1997). For example, male sulphur butterflies (*Colias* spp.) heterozygous at the phosphoglucose isomerase (PGI) locus have greater flying capacity than many homozygotes. Experienced female butterflies actively choose mates that are heterozygous at this locus (Watt *et al.*, 1986; Andersson, 1994).

Borgia (1979) suggested that if heterozygous advantage occurs at a single locus with two alleles, then a mutant female that recognizes and mates with heterozygous males will leave more progeny than females mating at random. This is surprising, since although overdominance will preserve genetic variability in the population, this variance is not additive and not heritable. Partridge (1983) noted that Borgia's argument fails to account for the relative proportions of the two unequally fit homozygotes in the progeny. She introduced a choice gene at an unlinked locus that causes females to mate with heterozygous males instead of mating randomly. Her calculation showed that the choice gene is neutral. However, Partridge assumed that the probability of a female carrying the choice gene is independent of her genotype at the character locus. Our calculation will show that the choice gene assorts with the least fit genotype and this gives it a selective disadvantage. The one exception arises when the two homozygotes have the same fitness, when the choice gene is neutral. Recently, Mitton (1997) has put forward a model in which the choice gene

* Author to whom all correspondence should be addressed. e-mail: irwin@mast.queensu.ca

appears to be favoured. In addition to the viability selection used by Partridge (and used in the analysis by us), he imposes a threshold selection, which only allows individuals above a fitness threshold to breed. He provides a partial analysis of the effect of a gene for female choice of heterozygotes, claiming that the choice gene can produce a higher proportion of heterozygous offspring than random mating. However, he appears not to analyse the total fitness of a mutant female, which includes both heterosis and the threshold effect, and does not provide a complete genetic analysis of the problem. Because of his two stages of selection, it is difficult to understand how Mitton handles fitness and gene frequencies.

We describe a genetic calculation that shows that a gene for female choice of heterozygous mates is at a disadvantage because it assorts with the least fit genotype. We take advantage of this tendency, placing the gene in a fluctuating environment, and show that the choice gene is at an advantage for environmental periods between two and tens of generations.

MODEL OF THE FEMALE CHOICE GENE FOR HETEROZYGOUS MATES

We assume that an individual's fitness is determined by her genotype at the diallelic over-dominant A locus, that is, heterozygous individuals have greater fitness than homozygous individuals. A second unlinked locus determines how females select their mates. Initially, all individuals mate randomly, but we introduce a rare and dominant mutant allele at this second locus, which causes a female to mate only with heterozygotes with probability ε and randomly with probability $1 - \varepsilon$. We measure the fitness of the choice gene as its change in frequency over one generation. The following calculation shows that, when the choice gene is rare, it will have reduced fitness or will be neutral in a random mating population. The calculation assumes that, in the random mating population, the genotype frequencies at the character (first) locus, p_{AA} , p_{Aa} and p_{aa} , are at equilibrium. We provide a summary of the notation we use in Table 1.

The number of juvenile choice gene carriers of each genotype in the present generation is denoted by the column vector $[\hat{p}_{AA} \ \hat{p}_{Aa} \ \hat{p}_{aa}]'$. The number of juvenile choice gene carriers in the next generation, $[\hat{p}'_{AA} \ \hat{p}'_{Aa} \ \hat{p}'_{aa}]'$, can be calculated from the number in the previous generation, the fitness of each genotype, the rules of the mating system, and the equilibrium number of random mating individuals as follows,

$$\begin{bmatrix} \hat{p}'_{AA} \\ \hat{p}'_{Aa} \\ \hat{p}'_{aa} \end{bmatrix} = (\varepsilon H + (1 - \varepsilon)R) \begin{bmatrix} \hat{p}_{AA} \\ \hat{p}_{Aa} \\ \hat{p}_{aa} \end{bmatrix} \quad (1)$$

Table 1. Fitness and gene frequency notation

	Genotype		
	AA	Aa	aa
Relative fitness	$W_{AA} = 1 - s_1$	$W_{Aa} = 1$	$W_{aa} = 1 - s_2$
Random mater's frequency	$p_{AA} = p^2$	$p_{Aa} = 2p(1 - p)$	$p_{aa} = (1 - p)^2$
Choice mater's frequency	\hat{p}_{AA}	\hat{p}_{Aa}	\hat{p}_{aa}

Note: Frequencies refer to juveniles before selection. Random maters are in Hardy-Weinberg proportions and at equilibrium: the frequency of the A allele is $p = s_2/(s_1 + s_2)$.

where

$$H = \frac{1}{\bar{W}} \begin{bmatrix} \frac{1}{2}W_{AA} & \frac{1}{4} & 0 \\ \frac{1}{2}W_{AA} & \frac{1}{2} & \frac{1}{2}W_{aa} \\ 0 & \frac{1}{4} & \frac{1}{2}W_{aa} \end{bmatrix}$$

$$R = \frac{1}{\bar{W}} \begin{bmatrix} M_A W_{AA} & \frac{1}{2}M_A & 0 \\ M_a W_{AA} & \frac{1}{2}(M_A + M_a) & M_A W_{aa} \\ 0 & \frac{1}{2}M_a & M_a W_{aa} \end{bmatrix}$$

and $M_A = (W_{AA}p_{AA} + \frac{1}{2}p_{Aa})/\bar{W}$ and $M_a = (\frac{1}{2}p_{Aa} + W_{aa}p_{aa})/\bar{W}$ are the probabilities of getting an A or a allele from a random mate. The requirement that the choice gene is rare makes equation (1) linear in the number of rare choice gene carriers. Matrices H and R are transition matrices for genotype frequencies of a female who always chooses a heterozygous mate or mates at random, respectively. The matrix H encodes the selection regime and the mating system for heterozygote mating; the left column specifies the expected number of offspring of each genotype of a juvenile AA female who mates with heterozygous males. First, her numbers are reduced by selection (multiplication by W_{AA}) and, since she mates with a heterozygote, her offspring are either AA or Aa, each with a probability of $\frac{1}{2}$. The centre and right columns are constructed in a similar way for juvenile Aa and aa females with the choice gene. The matrix R encodes the selection regime and the mating system for random mating; the left column specifies the expected number of offspring of each genotype of a juvenile AA female who mates at random. For example, R_{11} first converts the number of juveniles to adults by multiplication by W_{AA} . Homozygous AA offspring can be produced in two ways: by mating with an AA or an Aa mate. The probability an A allele is obtained from a random mate is M_A . The normalization factor of $\bar{W} = (W_{AA}p_{AA} + W_{Aa}p_{Aa} + W_{aa}p_{aa})$ guarantees that the population size remains constant.

The fate of the mutant choice gene can be discovered by examining the eigenvectors corresponding to the largest eigenvalue of the matrix $\varepsilon H + (1 - \varepsilon)R$. The dominant right eigenvector is the equilibrium distribution of genotypes among carriers of the choice gene and the dominant eigenvalue has the interpretation of the growth rate of the choice gene at equilibrium. For all selection coefficients where the homozygotes are different ($s_1 \neq s_2$) and the heterozygote is the most fit ($s_1 > 0$ and $s_2 > 0$), the dominant eigenvalue can be written as:

$$\lambda = 1 - \frac{(s_1 - s_2)^2(s_1(1 - s_2) + s_2(1 - s_1))}{4(s_1 + s_2)(s_1(1 - s_2) + s_2)}\varepsilon^2 + O(\varepsilon^3) \quad (2)$$

An examination of the mutant growth rate λ shows that the choice gene is at a disadvantage when weak ($\varepsilon \sim 0$). As ε increases from 0, the growth rate of mutants decreases below the growth rate of wild-type individuals. The equilibrium distribution of mutant genotypes is given by the dominant eigenvector:

$$\hat{\mathbf{p}} = \begin{bmatrix} \hat{p}_{AA} \\ \hat{p}_{Aa} \\ \hat{p}_{aa} \end{bmatrix} = \begin{bmatrix} \left(\frac{s_2}{s_1 + s_2}\right)^2 \\ \frac{2s_1s_2}{(s_1 + s_2)^2} \\ \left(\frac{s_1}{s_1 + s_2}\right)^2 \end{bmatrix} + \varepsilon \left(\frac{s_2^2(1 - s_1) - s_1^2(1 - s_2)}{(s_1 + s_2)^3} \right) \begin{bmatrix} -s_1 \\ (s_2 - s_1) \\ s_1 \end{bmatrix} + O(\varepsilon^2) \quad (3)$$

When the mutant has no effect ($\varepsilon = 0$), this is just the standard Hardy-Weinberg distribution; but as the mutant strength increases (ε increases from 0), the distribution of the mutant changes. Since one homozygous genotype has greater fitness than the other (e.g. AA is superior to aa if $s_2 > s_1$), we can see from the equilibrium genotype distribution that, among carriers of the choice gene, the proportion of superior homozygous genotypes is reduced and the proportion of inferior homozygous genotypes is increased compared to the wild population ($s_2^2(1 - s_1) - s_1^2(1 - s_2) > 0$). This change in distribution lowers the growth rate for the mutants compared with wild-type individuals. A wild female receives male gametes which are of superior type A with probability $p > \frac{1}{2}$, but a mutant which preferentially mates with heterozygotes has a lower probability of getting a superior male gamete because heterozygote males produce A and a gametes in equal proportion. Keeping in mind that the genotype distributions among mutant and wild-type individuals differ, an average mutant female will produce less fit offspring than an average wild female. Two examples will illustrate these calculations.

Anti-symmetric case

The anti-symmetric case, where $s_2 = 1 - s_1$, was examined by Mitton (1997). We use his values for the selection coefficients, $s_1 = 1/5$ and $s_2 = 4/5$; the AA homozygote is superior to the aa homozygote in this case. The equilibrium genotype distribution of mutants is

$$\begin{bmatrix} \hat{p}_{AA} \\ \hat{p}_{Aa} \\ \hat{p}_{aa} \end{bmatrix} = \begin{bmatrix} \frac{16}{25} - \frac{252}{625} \varepsilon \\ \frac{8}{25} + \frac{189}{625} \varepsilon \\ \frac{1}{25} + \frac{63}{625} \varepsilon \end{bmatrix} + O(\varepsilon^2) \quad (4)$$

and the relative growth rate is $\lambda = 1 - \frac{51}{700} \varepsilon^2 + \frac{459}{17,500} \varepsilon^3 + O(\varepsilon^4)$. Turning on heterozygote preference (increasing ε above 0) has the effect of reducing the abundance of AA genotypes among mutants (from $\frac{16}{25}$ to $\frac{16}{25} - \frac{252}{625} \varepsilon$) in favour of the less fit aa genotype (which increases from $\frac{1}{25}$ to $\frac{1}{25} + \frac{63}{625} \varepsilon$) as well as the heterozygote. The growth rate of the mutant population decreases as the strength of the mutant gene increases because $-\frac{51}{700} \varepsilon^2 < 0$. The fitness of a random mutant is lower than a random wild individual and the choice gene is at a disadvantage.

Symmetric case

Another interesting example is the symmetric case where the two homozygotes are equally fit, that is $s_1 = s_2$ (and $p = \frac{1}{2}$ independent of the value of s). The equilibrium genotype distribution of the juvenile choice gene carriers is

$$\begin{bmatrix} \hat{p}_{AA} \\ \hat{p}_{Aa} \\ \hat{p}_{aa} \end{bmatrix} = \begin{bmatrix} \frac{1}{4} \\ \frac{1}{2} \\ \frac{1}{4} \end{bmatrix}$$

and the growth rate is 1. The equilibrium genotype distribution and growth rate are independent of the effect of the choice gene. The probability that a male gamete is A is $\frac{1}{2}$ whether the female mates randomly or prefers heterozygous mates because the two homozygotes are equally abundant. The mutant gene is neutral because the fitness of the two homozygotes is the same.

TEMPORAL VARIATION IN FITNESS

Charlesworth (1988) has shown that mate choice for heterozygotes can evolve in a temporally fluctuating environment, but he did not examine the case where the heterozygote has superior fitness. Mitton (1997) has investigated the evolution of female choice of males with heterozygous advantage in a fluctuating environment. Fitnesses are determined by several overdominant loci. Mitton simulated small populations of individuals and his selection eliminated all individuals with fitness below a threshold. His results are quite noisy; sometimes the gene for female choice of heterozygous males spreads through the population, and sometimes the gene is eliminated from the population. No analysis of the conditions which favour the choice gene, beyond the need for environmental variability, was given.

In this section, we modify the model presented in the previous section to allow for temporal variation in fitness, and determine the conditions that favour a mutant choice gene in an infinite population. We imagine that the variation in environment arises from a periodic change, such as a seasonal change in the environment (hot/cold, wet/dry), a successional change in the local ecology, or perhaps as a result of a host–parasite interaction. For example, the parasite may specialize by attacking preferentially the more abundant homozygote (the heterozygote is immune to attack).

Although the choice gene is at a disadvantage (unless $s_1 = s_2$) when the genotype frequencies are at equilibrium, the results from the previous section suggest a way the choice gene could be at an advantage. Consider a population where the AA genotype has greater fitness than the aa genotype and the choice gene assorts with the less fit a allele. Suppose a change in the environment arises which gives the aa individuals a fitness greater than that of the AA individuals. The very assortment with the aa genotype that hurt the choice gene initially might now favour it in the new environment. In a few generations, the choice gene may again be at a disadvantage as it comes to assort with the A allele. We will need another environmental change for the choice gene to be at an advantage again. Periodic oscillations in the environment can provide a mechanism to favour the evolution of the choice gene. The timing of these oscillations is important. If they come too infrequently, the environment will appear nearly static to the choice gene, and it will be at a disadvantage. Faster changes should eventually bring a benefit to the choice gene, but oscillations that are too fast may eat away at this advantage.

The gene flow matrix for this time-dependent problem has a form similar to the matrix in equation (1), but wild gene frequencies and selection coefficients vary as well as the mutant gene frequencies. We use numerical calculations to analyse this problem because of the algebraic difficulties. A seasonal environmental change is modelled with a gradual change in the homozygote selection coefficients that both oscillate between s_1 and s_2 , but 180° out of phase. The time-dependent selection coefficient of period τ for the AA homozygote is

$$S_1(t) = s_1 + (s_2 - s_1) \left(\frac{1 - \cos(2\pi t/\tau)}{2} \right)$$

and the selection coefficient for the aa homozygote is

$$S_2(t) = s_2 + (s_1 - s_2) \left(\frac{1 - \cos(2\pi t/\tau)}{2} \right)$$

where s_1 and s_2 are the initial selection coefficients. The fitness of the Aa heterozygote remains constant at 1 and the initial population is at genotypic equilibrium at the character locus with rare mutants.

Each panel in Fig. 1 shows the genotypic frequencies at the character locus for the mutant and wild subpopulations over many generations in a changing environment. At $t = 0$, the frequencies are at the equilibrium described by equation (3): the wild individuals are at Hardy-Weinberg equilibrium, and the mutant individuals have a perturbed distribution, with a bias in favour of the less fit homozygote. The ratio of the growth rates for choice gene carriers to wild individuals, λ_c/λ_r , is shown as the top curve in each panel. This ratio changes and is recomputed each generation. When the ratio is larger than 1, the choice gene is increasing in frequency within the population; when the ratio is less than 1, the frequency of the choice gene is declining.

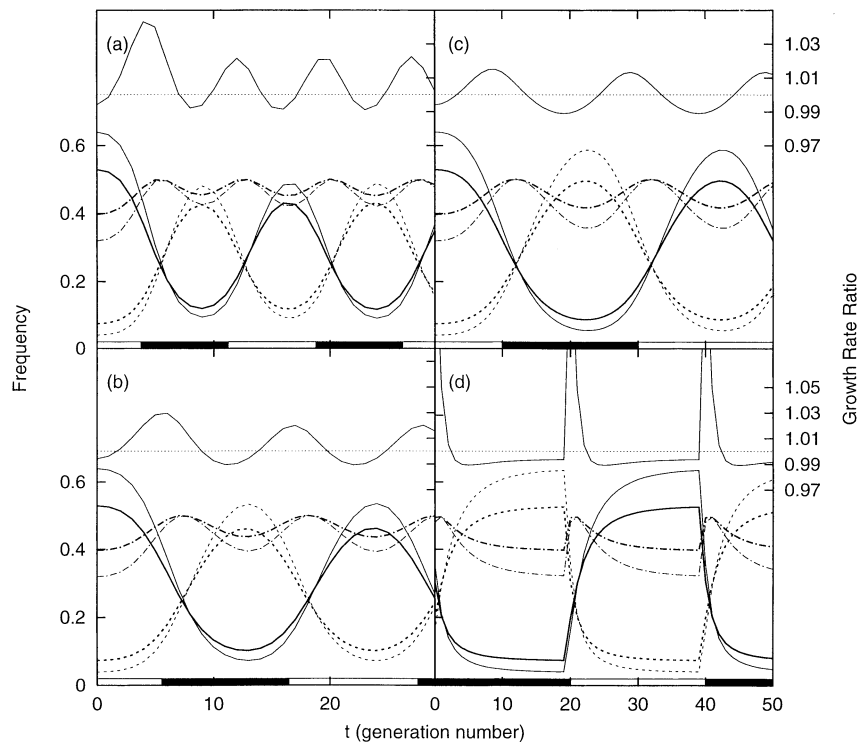


Fig. 1. Gene frequencies and the ratio of growth rates for wild and mutant (choice) genes. Each panel shows AA (solid line), Aa (dash-dotted line) and aa (dashed line) gene frequencies for wild (thin lines) and mutant (thick lines) genes against generation number. The top line, using the right-hand scale, shows the ratio of mutant to wild gene growth rates, λ_c/λ_r , for each generation. A horizontal dotted line is drawn at $\lambda_c/\lambda_r = 1$, dividing the intervals where the choice gene is favoured and disfavoured. The bar across the bottom of each panel shows which homozygote is superior. A white bar means AA is more fit than aa, and a black bar means aa is more fit than AA. Each panel has different parameters: (a) $s_1 = 0.2$, $s_2 = 0.8$, $\varepsilon = 0.3$, $\tau = 15$; (b) the same except $\tau = 22$; (c) the same except $\tau = \tau_{crit} = 40$, an oscillation period which puts the choice gene at a disadvantage; and (d) the same as (c) except the selection coefficients vary according to the step-function described in the text.

At $t = 0$, the mutant gene is at a disadvantage, but after one or more generations, the choice gene is favoured, then gradually loses this advantage as the population structure changes in response to the changing environment. A short environmental period ($\tau = 15$, Fig. 1a) makes the gene frequencies vary rapidly as the selection coefficients change due to the environmental forcing. Longer environmental periods (Figs 1b,c) increase the temporal variation in gene frequencies and create longer time intervals where the choice gene is at a disadvantage ($\lambda_c/\lambda_r < 1$). Changing the selection coefficients with a step-function, so that $S_1(t)$ changes from s_1 to s_2 in one generation, makes the gene frequencies change rapidly after the change in selection coefficients (Fig. 1d).

The evolutionary success of the choice gene is determined by the limit of the product of the ratio of the choice to random growth rates over a complete period of the environmental oscillation,

$$\frac{\Lambda_c}{\Lambda_r} = \lim_{t \rightarrow \infty} \prod_{k=1}^{\tau} \frac{\lambda_{c,t+k}}{\lambda_{r,t+k}}$$

which we call the periodic growth ratio. The growth rates of the choice and wild genes in the k th generation are $\lambda_{c,k}$ and $\lambda_{r,k}$ respectively. The mutant choice gene is favoured if the periodic growth ratio is greater than 1 and will be eliminated from the population if the periodic growth ratio is less than 1. Our computations show that in all cases the dynamics of the periodic growth ratio as a function of the environmental period can be described quite simply. For an environmental period of one generation ($\tau = 1$), the periodic growth ratio is less than 1; the environment appears constant to the gene, and the female choice gene is at a disadvantage. As the environmental period increases, the periodic growth ratio increases to a maximum greater than 1, and then decreases. The value of the period at which the mutant gene becomes disadvantageous, or the periodic growth ratio is less than 1, is called the critical period, τ_{crit} (Fig. 2).

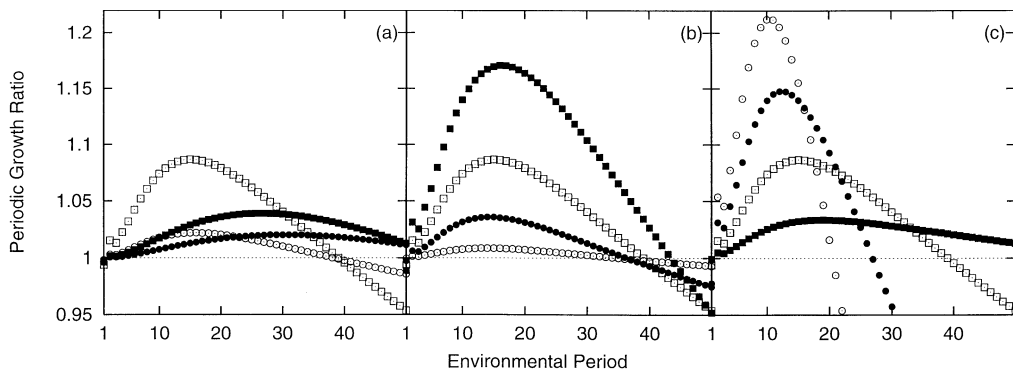


Fig. 2. Periodic growth ratio λ_c/λ_r as a function of environmental period. The choice gene is at an advantage for fast environmental periods corresponding to a few generations, and is at a disadvantage for sufficiently long environmental periods. (a) Data for four sets of selection coefficients: $s_1 = 0.1$, $s_2 = 0.4$ (■); $s_1 = 0.2$, $s_2 = 0.8$ (□); $s_1 = 0.1$, $s_2 = 0.3$ (●); $s_1 = 0.3$, $s_2 = 0.6$ (○). (b) Selection coefficients from the anti-symmetric case, $s_2 = 1 - s_1$, where $s_1 = 0.1$ (■), $s_1 = 0.2$ (□), $s_1 = 0.3$ (●) and $s_1 = 0.4$ (○). (c) Varying the strength of the mutant gene changes the amplitude of the periodic growth ratio: $s_1 = 0.2$, $s_2 = 0.8$, and $\epsilon = 0.1$ (■), $\epsilon = 0.3$ (□), $\epsilon = 0.6$ (●), $\epsilon = 1.0$ (○).

Both the selection coefficients and the probability of heterozygote choice have an impact on the period at which the maximum periodic growth ratio is attained, and the critical period. Results for a variety of selection coefficients show that the maximum periodic growth ratio and the environmental period at which this maximum is attained vary widely (Fig. 2a). The open squares correspond to the selection coefficients used in Fig. 1. Increasing both of the selection coefficients has the effect of increasing the maximum periodic growth ratio and reducing the period at which this maximum is attained. In the anti-symmetric case, where increasing the fitness of one homozygote is matched by an equivalent decrease in the fitness of the other homozygote ($s_1 = 1 - s_2$; Fig. 2b), the critical periods tend to be similar as the selection coefficients change. The maximum periodic growth ratio decreases as the selection coefficients approach $\frac{1}{2}$, because as the homozygotes become more similar in fitness, there is less possibility of variability in the genotype frequencies. Changing the strength of the mutant gene (varying ε from 0.1 to 1) without changing the selection coefficients increases the amplitude of the periodic growth ratio and decreases τ_{crit} (Fig. 2c).

Our results show that periodic environmental disturbances covering a large temporal range favour the choice gene, even if the homozygotes have fitnesses only slightly lower than that of the heterozygote. A contour plot of the critical oscillation period τ_{crit} is shown in Fig. 3. The critical oscillation period increases with the strength of the mutant (ε) and usually increases with decreasing selection coefficient. Although the contours are close together at small selection coefficients, indicating regions of rapid change, τ_{crit} changes very slowly over a wide range of selection coefficients, and is generally close to 60 generations for a weak mutant ($\varepsilon = 0.1$; Fig. 3a) and 20 generations for a strong mutant ($\varepsilon = 1$; Fig. 3b).

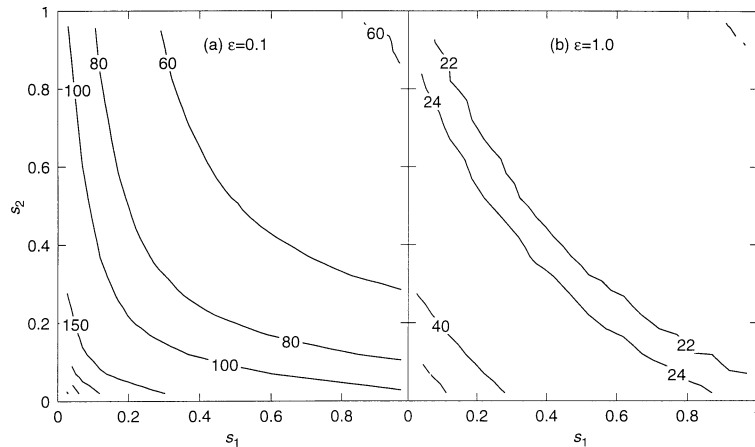


Fig. 3. Critical environmental period τ_{crit} shown as contours for selection coefficients $0 < s_1, s_2 < 1$. Environmental periods shorter than τ_{crit} will allow the mutant choice gene to spread and longer environmental periods put the mutant gene at a disadvantage. The results for a weak mutant gene ($\varepsilon = 0.1$, a) and a strong mutant ($\varepsilon = 1$, b) are similar, except τ_{crit} is larger for the weak mutant. The surfaces, especially panel (b), are nearly flat except near the boundary. Contours are drawn through the line $s_1 = s_2$, but there is no environmental period that will benefit the mutant gene at these points; fine detail near this line is not shown.

CONCLUSIONS

There is confusion in the literature surrounding the evolution of female choice for mates heterozygous at an overdominant locus. Our analysis shows that a rare gene for female choice of heterozygous males at a single locus is not favoured in a randomly mating population in a static environment. The gene is neutral when the two homozygotes have the same fitness and is at a disadvantage otherwise. When AA is superior to aa ($s_1 < s_2$), the mutant behaviour of the choice gene causes it to assort non-randomly with the three genotypes, appearing more often with Aa and aa and less often with AA as shown by the dominant eigenvector $\hat{\mathbf{p}}$ of the gene-flow matrix (equation 3). This occurs because, in heterozygote mating, exactly 50% of the male gametes have the a allele, whereas in random mating, less than 50% of the male gametes have the a allele. Aa is more fit than AA and aa is less fit than AA, so this redistribution has both an advantage and a disadvantage. The growth rate of the choice gene at equilibrium shows that the disadvantage of this redistribution outweighs the advantage (equation 2). A nice illustration is given by the simpler anti-symmetric case (equation 4). In the symmetric case, where the homozygotes are equally fit ($s_1 = s_2$), the difference between the two kinds of matings no longer exists and the choice gene is neutral. This rules out this hypothetical mechanism for the evolution of female choice of heterozygotes. The disadvantage is not a result of a fitness cost associated with mate choice.

This tendency for the choice gene to associate with the least fit genotype holds the key to constructing a situation in which the choice gene will be at an advantage. Fluctuations in the environment that give aa individuals a fitness greater than the fitness of AA individuals means that the association with the aa genotype which hurt the choice gene initially now favours it in the new environment. In a few generations the choice gene will again be at a disadvantage as it comes to assort with the A allele. Periodic oscillations in the environment perpetuate this periodic benefit to the choice gene. If the oscillations are frequent enough, the benefit to the choice gene overcomes the periodic disadvantage to the gene and provides a mechanism to favour the evolution of the choice gene. Our computations show that the choice gene is favoured under a wide range of environmental variations. Environmental oscillations ranging from one to tens of generations favour the choice gene. Longer environmental periods put the choice gene at a disadvantage. There is a wide range of biologically plausible environmental fluctuations and genetic parameters that will favour the evolution of a gene for female choice of overdominant heterozygous males. The results suggest that even very long environmental periods (60 or more generations) can play an important evolutionary role.

ACKNOWLEDGEMENTS

We thank Z. Finkel and D. Queller for helpful comments. This work was supported by a grant from the Natural Sciences and Engineering Research Council of Canada. A.I. was partially supported by a Queen's Graduate Fellowship.

REFERENCES

- Andersson, M. 1994. *Sexual Selection*. Princeton, NJ: Princeton University Press.
- Borgia, G. 1979. Sexual selection and the evolution of mating systems. In *Sexual Selection and Reproductive Competition in Insects* (M.S. Blum and N.A. Blum, eds), pp. 19–80. New York: Academic Press.

- Brown, J.L. 1997. A theory of mate choice based on heterozygosity. *Behav. Ecol.*, **8**: 60–65.
- Charlesworth, B. 1988. The evolution of mate choice in a fluctuating environment. *J. Theor. Biol.*, **130**: 191–204.
- Mitton, J.B. 1997. *Selection in Natural Populations*. Oxford: Oxford University Press.
- Partridge, L. 1983. Non-random mating and offspring fitness. In *Mate Choice* (P. Bateson, ed.), pp. 227–256. Cambridge: Cambridge University Press.
- Watt, W.B., Carter, P.A., and Donohue, K. 1986. Females' choice of 'good genotypes' as mates is promoted by an insect mating system. *Science*, **233**: 1187–1189.