

Male killing can select for male mate choice: a novel solution to the paradox of the lek

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In lekking species, intense directional selection is applied to aspects of the male genotype by female choice. Under conventional quantitative genetics theory, the expectation is that this will lead to a rapid loss in additive genetic variance for the trait in question. However, despite female choice, male variation is maintained and hence it pays females to continue choosing. This has been termed the ‘paradox of the lek’. Here we present a theoretical analysis of a putative sex-role-reversed lek in the butterfly *Acraea encedon*. Sex-role reversal appears to have come about because of infection with a male-killing *Wolbachia*. The bacterium is highly prevalent in some populations, such that there is a dearth of males. Receptive females form dense aggregations, and it has been suggested that males preferentially select females uninfected with the bacterium. As with more conventional systems, this presents a theoretical problem exactly analogous to the lek paradox, namely, what maintains female variation and hence why do males continue to choose? We model the evolution of a male choice gene that allows discrimination between infected and uninfected females, and show that the stable maintenance of both female variation and male choice is likely, so long as males make mistakes when discriminating between females. Furthermore, our model allows the maintenance, in a panmictic population, of a male killer that is perfectly transmitted. This is the first model to allow this result, and may explain the long-term persistence of a male killer in *Hypolimnas bolina*.

Keywords: male killer; sex ratio; lek; mate choice; *Acraea encedon*; *Hypolimnas bolina*

1. INTRODUCTION

Standard population genetics theory would predict that intense directional selection imposed by female choice will lead to a rapid loss of additive genetic variance for the male trait in question. Should this occur, the benefits of female choice disappear. This presents us with the ‘paradox of the lek’, namely, why do females continue to choose between males when the genetic benefits of choice are so small?

In the most convincing resolution of the paradox (Pomiankowski & Møller 1995), the authors turn the problem around. Rather than asking what maintains female choice in the absence of male variation, they ask what maintains variation despite strong directional selection? They argue that intense directional selection will favour modifiers that increase the number of genes and the average contribution of a locus to phenotypic variance in males. Continued variability of the male trait is hence expected in their model and consequently so is the maintenance of female choice—the paradox disappears. To back up their case, they present data showing that additive genetic variance is in fact higher in sexually selected traits and not lower as the traditional argument would predict. Here we present an alternative solution to the lek paradox inspired by the biology of *Acraea encedon* and its male-killing bacterium.

Certain Ugandan populations of the butterfly *Acraea encedon* are infected with the male-killing bacterium *Wolbachia*. As is typically the case with this bacterial group, infection of new individuals does not occur horizontally (i.e. to conspecifics that the host encounters), but

vertically, via the mother’s eggs. One consequence of this is that bacteria finding themselves in a male have effectively zero fitness (there is no transmission via sperm) (O’Neill *et al.* 1997; Werren 1997). In *A. encedon*, and numerous other hosts (Hurst *et al.* 1996; Hurst 1993; Majerus *et al.* 1998), bacterial pathogenesis is sex limited such that the *Wolbachia* will only kill their host if they find themselves in a male (Jiggins *et al.* 1998, 2000a). Killing occurs at the egg stage, so infected females typically produce a clutch in which only half the eggs hatch, these are almost all females. While this is an act of suicide for the parasite, bacterial fitness in males is zero anyway, so they have nothing to lose. However, their clonal relatives in the dead males’ sisters probably benefit through female host cannibalism of their dead brothers (Hurst & Majerus 1993).

One remarkable feature of the *Wolbachia* infection of *A. encedon* is the high prevalence of the bacteria in females (78–100%) (Jiggins *et al.* 2000b). As a consequence, many populations have a marked shortage of males. What is unique about this system though, is that bacterial prevalence is correlated with a change in the butterfly’s mating system. Under normal circumstances, males seek out and compete for matings with females that are dispersed around larval food plants. In populations where the male killer is at high frequency, however, females form dense aggregations in grassy areas near prominent landmarks (e.g. trees). At these sites, up to 351 female butterflies have been found congregated in a small arena measuring roughly 10 m × 20 m (Jiggins *et al.* 2000b).

Several pieces of evidence suggest that females aggregate at these sites in order to attract mates. First, in mark–release–recapture experiments, virgin females were more likely to remain at these sites than mated females,

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suggesting that females congregate in order to mate. Second, aggregating females exhibit a range of mate-attracting behaviours (Jiggins *et al.* 2000b).

These observations are reminiscent of the common male phenomenon of lek formation. Classical leks are situations in which '... males aggregate when advertising for females, females are unimpeded in their choice of mates, and males are thought to make no contribution to the females' effort besides their sperm.' (Ryan 1997, p. 179). In such circumstances, it is females that are the 'in-demand' sex, as they have the slower reproductive rate. Hence, with many males laid out before them, females can afford to be choosy about whom they mate with.

In a highly female-biased population it is likely that the balance of which is the 'in-demand' sex will swing towards males. In such situations, it is expected that males rather than females will exercise choice when selecting a partner (Emlen & Oring 1977). One piece of evidence suggesting that males may use the sites to discriminate between infected and uninfected females is that uninfected females are more likely to be mated than infected females (Jiggins *et al.* 2000b). Assuming that males are choosing, this system poses a theoretical problem that is exactly analogous to the paradox of the lek. Namely, can the male-killing bacterium be maintained in a population in which males are discriminating against it?

At first sight this seems rather unlikely. Assuming that a choice gene can invade, its presence in the population confers a mating disadvantage on infected females relative to uninfected. The prevalence of the bacterium will therefore decrease, and consequently males with the choice gene will find it increasingly easy to realize the benefits of choice (because uninfected females will be easier to find and hence mate searching will be less costly). Such a positive feedback process could well lead to loss of the male killer (MK) altogether. If this is the case, then male mate choice is likely to be a rather transient phenomenon.

Our models are able to explain both the invasion and maintenance of a male choice gene coupled with retention of the male-killing bacterium. Crucially, male discrimination must have some degree of inaccuracy if choice is to be maintained.

2. THE SIMPLE MODEL

Fisher's explanation for the near ubiquity of the 1:1 sex ratio rests on the expectation that if the population deviates from equal numbers of males and females, any individual that produces an excess of the rarer sex will gain an advantage (provided investment in males and females is equal) (Fisher 1930). If this change is heritable then the gene responsible will spread, returning the population to equal proportions of males and females. A host population infected with a male-killing bacterium will by necessity be female biased, and so for similar reasons, most autosomal modifiers that promote male production will be favoured by selection (primary sex-ratio compensation is an exception). Attempts to model host genes that resist cytoplasmic sex-ratio distorters have been made previously (Randerson *et al.* 2000; Caubet *et al.* 2000; Werren 1997, and references therein; Uyenoyama & Feldman 1978).

(a) *Male killer only*

We specify a model of male killing similar to those that have been proposed previously (Randerson *et al.* 2000; Freeland & McCabe 1997; Hurst 1991). MK transmission is entirely vertical (mother to egg), with a proportion α of a mother's eggs being infected. (All parameters are listed in Appendix A.) There is no horizontal transmission (i.e. infectious transmission to conspecifics encountered by the host) and the bacteria do not enter sperm. All males infected with the bacterium die as eggs and their fitness is redistributed amongst the rest of the surviving brood. The MK benefits from this because it is mainly females (i.e. transmitters of the bacterium) that receive the fitness handout. We assume this 'fitness compensation' to be a function of the amount of male death that occurs and hence of α . If the death of males is perfectly compensated amongst the rest of the brood (i.e. no fitness is wasted), then the fitness augmentation received by survivors is equal to the term in brackets in equation (1). We assume fitness compensation to be a proportion ϕ of this theoretical maximum, so the compensation received by survivors as a consequence of male death is

$$\varphi = 1 + \phi \left(\frac{1}{1 - \alpha/2} - 1 \right) = 1 + \frac{\phi\alpha}{2 - \alpha}. \quad (1)$$

Note that this is an increasing function because as α and hence male death increases, the amount of fitness to be distributed increases, while brood fitness is shared amongst an increasingly small number of individuals.

In addition, we assume that the MK has a direct effect on adult female fitness U . There is evidence that the male-killing bacteria *Rickettsia* imposes a viability cost on females of its host *Adalia bipunctata* (Hurst *et al.* 1994). However, in *A. encedon* there is some suggestion that the bacteria may in fact benefit female hosts (Jiggins *et al.* 2000a). Either scenario can be incorporated depending on which operator precedes the U -parameter: positive values of U refer to a fitness cost, while negative values refer to a fitness benefit.

We assume for the purposes of deriving the MK invasion and equilibrium conditions that the population is infinite and panmictic with discrete generations. Recursion equations are expressed in terms of infected and uninfected females since all breeding males are uninfected. The proportion of infected adult females is p , while the proportion of uninfected adult females is q . W is the sum of the right-hand sides.

$$Wp' = p\alpha(1 - U)\varphi, \quad (2)$$

and

$$Wq' = p(1 - \alpha)\varphi + q. \quad (3)$$

The invasion conditions for MK are found when

$$\left. \frac{dp'}{dp} \right|_{p=0} > 1,$$

is satisfied, i.e. when

$$U < \frac{\alpha\varphi - 1}{\alpha\varphi}. \quad (4)$$

The equilibrium value of MK (p^*) is found by solving for $p' = p$, which gives

$$p^* = \frac{1 - \varphi\alpha + \varphi\alpha U}{1 - \varphi + \varphi\alpha U}. \quad (5)$$

This is a stable equilibrium. As reported previously (Hurst 1991), perfect transmission of a MK theoretically leads to fixation of the MK ($p^* = 1$ when $\alpha = 1$), otherwise p and q are maintained in polymorphism ($p^* < 1$ when $\alpha < 1$). Fixation of a MK would lead to population extinction because of the severe shortage of males.

3. INVASION OF THE CHOICE GENE

We propose a modifier of male choice that allows discrimination between infected and uninfected females. Although the mechanism for such choice is hypothetical, one could imagine a situation in which males use chemical cues to single out uninfected females. The potential advantages for such a gene are large, especially if the MK is at a high frequency. Males with the choice gene will be able to ensure that half of their offspring are male—the ‘in-demand’ sex.

We assume an autosomal, dominant choice gene that acts in males, but has no effect on females. There are two rounds of mate choice. In the first round, a male with the gene will encounter female genotypes (N.B. we use this term to refer to the presence or absence of both the choice gene and the bacterium) at their frequency in the population. If he encounters an uninfected female he accepts her and mates. If on the other hand he encounters an infected female he rejects her and moves on to the second round of mating. In the second round he accepts any female he encounters. We assume that the act of rejection and passage to the second round imposes a cost of extra mate searching, c ; there is a cost associated with a second bite at the cherry. Males without the choice gene always accept the first female they meet in the first round, hence they never suffer the cost of mate searching. One could imagine numerous other ways in which to model male choice and with no data on how males may be choosing this is perhaps as good as any. It seems unlikely that alterations, such as allowing multiple rounds of mating, would significantly change the qualitative predictions of the model, but this remains to be proven.

Generations are discrete and both sexes mate only once. Although the latter assumption is unlikely to be met in the field, it is necessary to prevent the model from becoming overly complicated. For convenience at this stage we also assume an infinite panmictic population. This provides the most permissible conditions for choice gene invasion because male mate selection in the first round of mating has no effect on the distribution of available female genotypes in the second round; the second bite at the cherry is as good as the first. We also assume at this stage that males and females do not compete with each other (males and females have a separate mean fitness W_m and W_f , respectively).

A further aspect of the model is that males may not be able to distinguish perfectly between infected and uninfected females. The parameter m describes the proportion

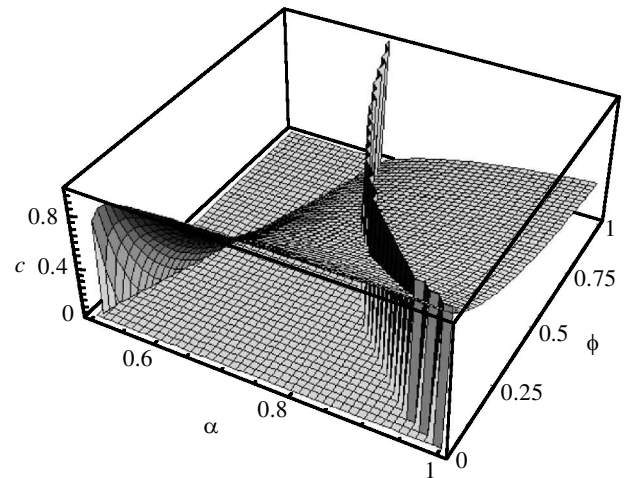


Figure 1. Invasion conditions for the simple choice gene (infinite population size) when $U = 0$ and $m = 0$. The vertical sheet represents the invasion conditions for the MK with parameter combinations to the right of the sheet allowing invasion. The other sheet represents invasion conditions for the choice gene. Invasion is permitted below the sheet. Note that for all parameter space in which the MK can invade, the choice gene can invade also. The choice gene is capable of invading in much of the parameter space despite a high cost of mate searching.

of occasions on which a male with the choice gene makes a mistake in the first round (i.e. either rejecting an uninfected female or accepting an infected female). Hence, $m = 0$ corresponds to perfect discrimination between infecteds and uninfecteds, $m = 0.5$ corresponds to an absence of choice, and $0.5 < m < 1$ would refer to choice for infected females. The recursion equations for the system and invasion conditions for the choice gene are reported in the electronic Appendix B, which can be found at The Royal Society Web site.

(a) ‘Perfect’ choice

We first consider the specific case of perfect discrimination ($m = 0$). Under these conditions, it is clear from figure 1 that as long as the MK can invade, the choice gene can invade also. What is more, very often it can do so despite a massive cost (c) of mate searching. Simulations were performed in order to determine the ultimate outcome of the system. In these the MK was allowed to reach its equilibrium frequency, then the choice gene was introduced at low frequency in males. If any genotype reached very low frequency (i.e. 10^{-6}), it was set to zero and the system was allowed to run to its equilibrium point. These simulations reveal that once the choice gene has invaded the host population, it almost always removes the MK completely. Results in which the choice gene and the MK are maintained in polymorphism only begin to appear when the cost of mate searching is of the order of 0.2 or greater, and even then they are rare. So, it appears that this simplest of models is unlikely to explain the results obtained from the *A. encedon* system. Although the presence of a MK can promote the evolution of male choice, if discrimination between infecteds and uninfecteds is perfect then the MK is exceedingly unlikely to be maintained in the population (let alone at the sort of frequencies we see in *A. encedon* populations).

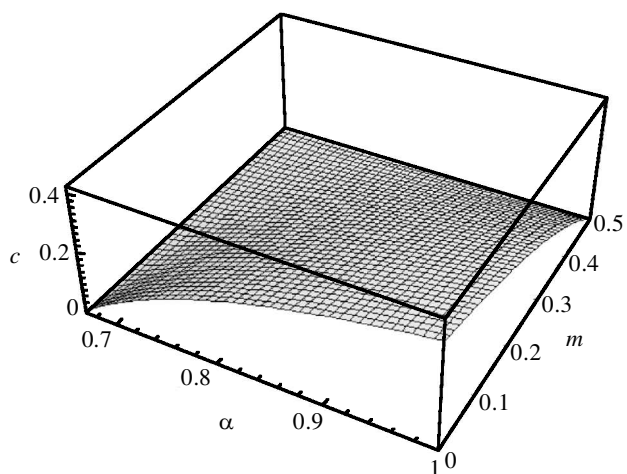


Figure 2. Invasion conditions for the simple choice gene (infinite population size) when $U = 0$ and $\phi = 0.5$. Only values of α for which the MK can itself invade are shown. Note that for all values of m below 0.5 the choice gene can invade if costly (this corresponds to some degree of discrimination between infected and uninfected females). Also note that as the frequency of mistakes in male choice increases (i.e. increasing m) invasion conditions become more stringent.

(b) Error-prone choice

Figure 2 shows the invasion conditions for the choice gene when $0 < m < 0.5$. This corresponds to some degree of error in discrimination between infected and uninfected females. It is clear that the choice gene is still capable of invading the population, although as the probability of errors increases, the invasion conditions become more stringent. An extensive scan of parameter space revealed that when $0 < m < 0.5$, results in which both the MK and the choice gene are maintained in the population are much more frequent. What is more, as choice becomes more inaccurate (i.e. increasing m) the proportion of results in which the MK is at high frequency increases. The model therefore predicts that for maintenance of the MK at high frequency, the frequency of male mistakes must be high.

(c) The effect of male sampling in the first round of mating

We have so far assumed that male choice in the first round of mating has no effect on female genotype frequencies in the second round. This would only be the case in an infinite population. We now consider the more realistic case of finite population size and hence the effect of male sampling. Let N_t be the total population size. We assume that this is invariable, but that the number of males and females (respectively N_y and N_x) can change (N.B. $N_y + N_x = N_t$). This amounts to a model in which an excess of juveniles are produced by each of the different male–female combinations, but that these individuals then compete for N_t spaces as adults in the environment. This assumption seems reasonable as long as the population is not so female biased that there are not enough males to maintain the population size.

To allow the calculation of N_x and N_y after changes in genotype frequencies in each generation, we now assume that males and females do not have separate mean fitnesses, but that there is a single population mean

fitness. Hence, the recursions for the more complicated model are the same as those for the simple model, except that W_x and W_y are replaced by a population mean fitness W (the sum of the relative fitnesses of all male and female genotypes). A further change from the simple model is that female genotype frequencies in the second round of mating (reported in electronic Appendix B) are now affected by male sampling in the first round. The model was investigated by simulation.

It is evident that invasion conditions for the choice gene are more stringent in a finite population than for the simple model. Intuitively this is sensible because the benefits of male choice are reduced. In the second round, some proportion of the uninfected females will have already been removed by mating in the first round, so the chance of meeting a ‘desirable’ female in the second round is reduced compared with the previous model. So the second bite at the cherry is not as good as the first. Despite this reduced likelihood of invasion however, the choice gene is still capable of invading and persisting in polymorphism with the MK. Figure 3*a–d* summarizes the parameter space in which we obtain results, where the choice gene and the MK both persist in polymorphism. If discrimination is perfect (i.e. $m = 0$), then the MK is always ousted from the population by the choice gene. However, if there is even a slight amount of error (i.e. $m > 0$), then both the MK and choice gene are retained at equilibrium, although for very low values of m the MK will be at very low frequency. The model predicts that invasion of the choice gene followed by persistence of the MK is most likely when male discrimination is imperfect, but with few mistakes, and when the cost of mate searching is low. For maintenance of both the MK and the choice gene at high frequency, the model predicts that male mistakes must be fairly common, the MK should impose a low cost (or preferably a benefit) to females and the cost of mate searching should be low.

(d) 100% transmission

Up to this point we have only considered MKs that are not transmitted to all of a female’s eggs. What happens if the MK has perfect vertical transmission ($\alpha = 1$)? Can the choice gene invade and reach an equilibrium at which both the MK and the choice gene are maintained? This arises as an interesting question when we consider the case of the butterfly *Hypolimnas bolina*, a MK host (see §4) (Hurst 1993).

When $\alpha = 1$, so long as the MK can invade it will go to fixation in the population under conditions of panmixis. Hence, if a choice gene is going to have any effect on the MK’s dynamics then it only has a short time-window in which to invade before the population goes extinct. An alternative sequence of events would be that a MK with a very efficient (but not perfect) vertical transmission prompted the evolution of a male choice gene and then subsequently improved its transmission to $\alpha = 1$. This is more likely because it does not require the evolution of male choice in a very short time-period.

For convenience we investigated the first scenario because it was possible to address it using our existing model. Simulations were set up similar to those above, except that the choice gene was introduced after an

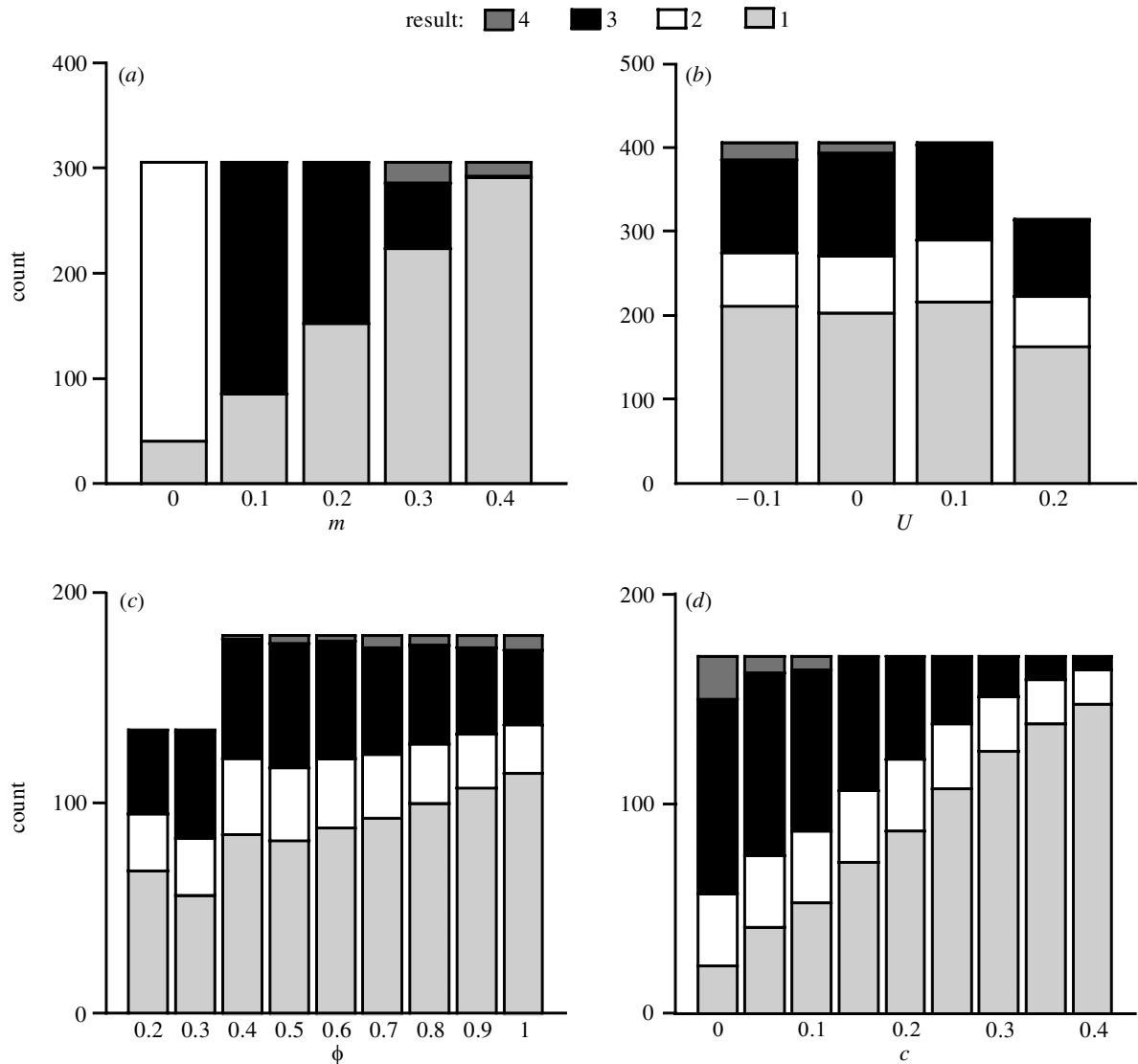


Figure 3. (a–d) Results of a parameter scan for the more complicated choice model (incorporating the effects of male sampling of female genotypes in the first round of mating). Simulations were conducted so that the MK reached its equilibrium frequency before introduction of the choice gene at low frequency. The system was again allowed to reach equilibrium and the result was classified as one of the following: 1, failure of the choice gene to invade; 2, invasion of the choice gene followed by loss of the MK; 3, maintenance of both choice gene and MK in the population at polymorphic frequencies; 4, polymorphism with both the MK and choice gene at high frequency (i.e. more than half of males exhibiting choice, and the MK frequency is in excess of 60%). The following parameter values were used in all possible combinations (the empirical estimate of $\alpha = 0.95$ from the closely related MK in *Acraea encedana* was used throughout; Jiggins *et al.* 2000a): m , range 0–0.4, step 0.1; U , range –0.1–0.2, step 0.1; ϕ , range 0.2–1, step 0.1; c , range 0–0.4, step 0.05 (all ranges inclusive). Ignoring those combinations for which the MK cannot invade, this gives 1534 combinations. In (a–d) the ‘count’ label on the y -axis refers to the number of each class of result for each value of the focal parameter.

arbitrary value of 20 generations and not when the MK had reached its equilibrium frequency. The results are presented in figure 4. In a number of cases, the system had not reached equilibrium after 500 000 generations. These were considered as results in which the MK and the choice gene are maintained. Although there are generally fewer results than for $\alpha = 0.95$, in which both the MK and choice gene are at high frequency, it is clear that persistence of both is a common result. So, the evolution of male choice for uninfected females could lead to the long-term survival of a host population infected with a MK that would otherwise have sent it extinct. To the best of our knowledge this is the only model that allows

population maintenance when vertical transmission is perfect (and is unaffected by host genotype) in the absence of population structure (but, see Heuch 1978; Heuch & Chanter 1982).

4. DISCUSSION

We have demonstrated that, in theory, the presence of a MK at equilibrium in its host population can select for the evolution of male choice for uninfected females. Such a choice gene gains its advantage because by frequently allying itself with the uninfected cytotypic, it ensures that in the next generation it will be represented in a brood

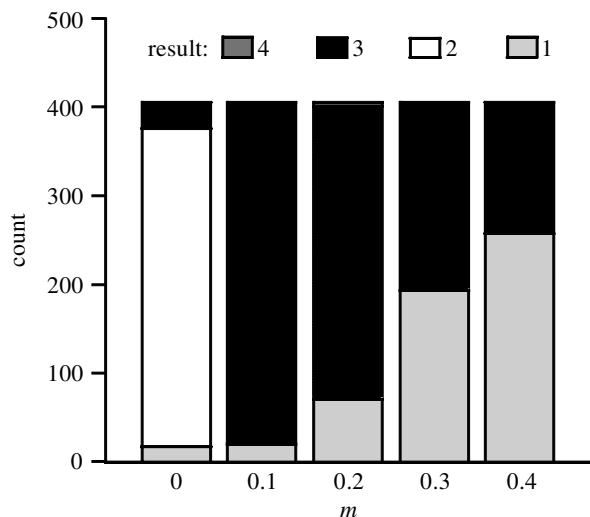


Figure 4. Results of a parameter scan for the more complicated choice model when $\alpha = 1$ (i.e. perfect vertical transmission). The data are similar to those in figure 3, but the range of parameter combinations scanned is slightly larger. In this case the values of the U -parameter considered were extended to 0.4. This gives an extra 491 parameter combinations for which the MK can invade. This was done because a MK with perfect transmission can invade even if it imposes a high cost. Note that retention of the MK and the choice gene is a common result, although maintenance at high frequency is less likely than for $\alpha = 0.95$.

with a 1:1 sex ratio, as opposed to one in which most of the males have been killed. In a female-biased population, the gene's presence in males allows it to take advantage of their increased reproductive success as the 'in-demand' sex. The conditions for the invasion of such a choice gene are benign, despite the incorporation of male mistakes and a cost of mate searching. Furthermore, as long as there are mistakes in male choice the MK is maintained in the population, which preserves the benefits of male discrimination. We thus provide a novel and simple solution to the lek paradox.

(a) *Relevance to the *Acraea encedon* system*

What are the model's predictions regarding the particular case of *A. encedon*? Observations suggest that in populations exhibiting female-aggregating behaviour, the MK tends to be at high frequency (Jiggins *et al.* 2000*b*). As is evident from figure 3*a-d*, although results in which both the MK and the choice gene are retained are common, those in which both are at high frequency are less so. This is because, in many cases, when the choice gene invades it brings down the frequency of the MK appreciably. However, there are cases in which the choice gene goes almost to fixation without making much of an impression on the MK. The model predicts that for both the choice gene and the MK to be at high frequency at equilibrium, male mistakes should be common, the cost of mate searching should be low and the MK should impose a low cost, or be beneficial to females. The first of these predictions has yet to be tested, the second is probably true given that females aggregate and the third has been suggested by previous work (Jiggins *et al.* 2000*a*).

(b) *The paradox of the female lek*

The strongest prediction from the models is that both the MK and the choice gene will be retained in the population as long as there are mistakes in male choice. This is the case even if there is no cost to the extra mate searching involved in rejecting an infected female in favour of one that is uninfected. Hence, if male choice is inaccurate then the MK cannot be 'chosen out' of the population. This is an intriguing result because it provides a simple solution to the lek paradox. In general terms, this is the expectation that by the act of mate selection, the choosy sex rapidly eliminates all population variability for the trait in question. Once this occurs, choice is no longer beneficial, and hence if the act of choosing has any associated costs then the gene will be lost (Andersson 1994; Pomiankowski & Møller 1995). In our model, the presence of male mistakes prevents the MK from being eliminated, so the paradox of choice without variation does not arise. Mistakes effectively result in a frequency-dependent cost to choice; as the choice gene invades and in the process pushes down the frequency of the MK, choosy males are more likely to encounter uninfected females in the first round. Hence, there comes a point at which the MK is so uncommon that it is detrimental to be choosing. This is because choosers will sometimes reject 'desirable' females and suffer the cost of mate searching. At this point, the fitness of choosers and non-choosers is equivalent.

Another question posed by conventional lekking species is: Why do 'undesirable' members of the chosen sex opt to congregate at leks when they have little chance of mating? One possible answer is that it pays 'undesirable' individuals to congregate because they benefit from occasional errors in discrimination by choosers (Kokko 1997). Although our models do not address this issue directly, we note that error in male discrimination is an essential component of the maintenance of variation in our model. Choice mistakes could well be an important general force maintaining leks.

(c) *The maintenance of perfect male killers*

Classical models of male killing predict that if the MK is transmitted perfectly to the entire brood (i.e. $\alpha = 1$), then it will rapidly go to fixation (Hurst 1991). In the process this will eliminate its host population due to the extreme dearth of males. This result applies even if one considers the evolution of host resistance. One class of resistance gene that has been modelled is that which acts in males to prevent bacterial killing. Invasion of such a gene always results in either fixation or loss of the MK when $\alpha = 1$ (Randerson *et al.* 2000). A further class of resistance genes that acts in females to reduce bacterial transmission to the eggs is able to maintain a perfectly transmitted MK at polymorphic frequencies. However, in this case, α no longer equals unity (Randerson *et al.* 2000). How then can we make sense of empirical evidence for a long-term infection of the butterfly *Hypolimnas bolina* with a perfectly transmitted male-killing cytoplasmic element?

It has been known for some time that the butterfly *Hypolimnas bolina* has a maternally inherited sex-ratio (SR) condition in which SR females give rise to all-female broods (Clarke *et al.* 1983; Hurst 1993). On a

number of the West Fijian islands, Simmonds found biased sex ratios due to the presence of a polymorphism for SR and normal females (Simmonds 1923, 1926, 1928). He also found that the SR condition was associated with high egg mortality and early larval mortality. Later work has established that the individuals killed are all males (Clarke *et al.* 1975). By revisiting sites at which the SR polymorphism was previously documented, Clarke *et al.* (1983) showed that in some instances, the polymorphism has existed for at least 150 generations. Assuming that the male-killing trait does indeed achieve 100% vertical transmission, this poses a theoretical problem. As described above, previous models of male killing in a panmictic population, predict that a MK with perfect vertical transmission will rapidly go to fixation, sending its host population extinct in the process (Hurst 1991). Our results suggest a novel explanation for the stable persistence of the SR condition over time. It is clear that provided male choice appears before the MK goes to fixation, it can prevent population extinction and allow the stable maintenance of the MK at polymorphic frequencies. This would manifest itself as a polymorphism for SR females and normal females. Clarke *et al.* (1983, p.230) suggest however that male mate choice is unlikely, stating that 'The butterflies flew and mated freely in our heated greenhouses and there was no indication that males mated preferentially with bisexual or unisexual females, or with those of any particular pattern.' This appears to be just an assertion however, not backed up by any controlled mate-choice experiments, so it is possible that they simply did not notice any mate choice that was in fact occurring (particularly if it was rather inaccurate).

At present it is not clear how general the phenomenon of male mate choice is in male-killing systems. However, there is some evidence from *Armadillidium vulgare* that females not infected with a feminizing *Wolbachia* are more likely to be mated than those that are infected (J. Moreau and T. Rigaud, personal communication). Moreover, empirical and theoretical work on meiotic drive in stalk-eyed flies has suggested that mate choice may be an important factor in the population dynamics of other types of sex-ratio distorter (Lande & Wilkinson 2000; Pomiankowski & Hurst 1999; Reinhold *et al.* 1999; Wilkinson *et al.* 1998).

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APPENDIX A. LIST OF PARAMETERS

α , proportion of eggs from an infected female that contain the male-killing bacterium.

c , cost of mate searching, i.e. the cost suffered by males that reject a female in the first round of mating and move to the second round.

ϕ , proportion of dead male fitness that is redistributed amongst the rest of the brood.

φ , fitness compensation received by survivors in MK broods.

U_j , cost imposed by the MK on infected females as adults.

m , proportion of occasions on which a male makes an error (in either direction) when discriminating between infecteds and uninfecteds in the first round of mating.

p , frequency of the MK in females.

q , frequency of the uninfected cytotypic in females.

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