Detecting sexually antagonistic coevolution with population crosses

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The result of population crosses on traits such as mating rate, oviposition rate and survivorship are increasingly used to distinguish between modes of coevolution between the sexes. Two key hypotheses, erected from a verbal theory of sexually antagonistic coevolution, have been the subject of several recent tests. First, statistical interactions arising in population crosses are suggested to be indicative of a complex signal/receiver system. In the case of oviposition rates, an interaction between populations (x, y and z) would be indicated by the rank order of female oviposition rates achieved by x, y and z males changing depending upon the female (x, y or z) with which they mated. Second, under sexually antagonistic coevolution females will do 'best' when mated with their own males, where best is defined by the weakest response to the signal and the highest fitness. We test these hypotheses by crossing strains generated from a formal model of sexually antagonistic coevolution. Strains differ in the strength of natural selection acting on male and female traits. In our model, we assume sexually antagonistic coevolution of a single male signal and female receptor. The female receptor is treated as a preference function where both the slope and intercept of the function can evolve. Our results suggest that neither prediction is consistently supported. Interactions are not diagnostic of complex signal–receiver systems, and even under sexually antagonistic coevolution, females may do better mating with males of strains other than their own. These results suggest a reinterpretation of several recent experiments and have important implications for developing theories of speciation when sexually antagonistic coevolution is involved.

Keywords: sexual conflict; arms races; theory; comparative biology; mating rate

1. INTRODUCTION

The evolutionary interests of the sexes at mating typically conflict, and these conflicts may lead to rapid sexually antagonistic coevolution (Parker 1979; Rice & Holland 1997; Chapman *et al.* 2003). Conflicts occur over a wide variety of traits, including mating rate (Rowe *et al.* 1994; Choe & Crespi 1997; Arnqvist & Nilsson 2000), oviposition rate (Chapman *et al.* 1995; Eberhard 1996) and offspring provisioning (Trivers 1972; Haig 2000). Traits apparently coevolving to further the interests of one sex over the other in these conflicts include grasping and anti-grasping structures (Bergsten *et al.* 2001; Arnqvist & Rowe 2002*a*), male seminal signals and female receptors (Chapman 2001; Swanson & Vacquier 2002), reproductive tract morphology (Presgraves *et al.* 1999; Miller & Pitnick 2002; Miller & Pitnick 2003) and mating behaviours (Rowe & Arnqvist 2002). A central prediction of sexually antagonistic coevolution is that these traits will evolve very rapidly, thereby generating divergence in isolated populations and perhaps speciation (Rice 1998; Howard *et al.* 1998; Parker & Partridge 1998; Gavrilets 2000; Gavrilets *et al.* 2001; Gavrilets & Waxman 2002).

There is growing support for this prediction in both laboratory and natural populations. Several experimental evolution studies in *Drosophila* and *Scathophaga* have demonstrated that a variety of reproductive traits are apparently shaped by sexual conflict and evolve very rapidly

(Rice 1996; Holland & Rice 1999; Pitnick *et al.* 2001*a,b*; Hosken *et al.* 2001). Support in natural populations comes primarily from two types of studies. First, comparative studies of traits thought or known to be involved in sexual conflict suggest that their evolution is rapid and divergent (Pitnick *et al.* 1999; Presgraves *et al.* 1999; Westlake *et al.* 2000; Bergsten *et al.* 2001; Arnqvist & Rowe 2002*b*). For example, Arnqvist (1998) reported that male genital shape in insects had evolved much more quickly among species that were susceptible to sexual conflict (groups with polyandrous mating) than those that were not (monandrous). Moreover, these polyandrous groups also appear to speciate more rapidly (Arnqvist *et al.* 2000). A second approach is to determine the pattern and rate of molecular evolution in reproductive proteins. Several studies have found evidence for directional selection and rapid evolution of these proteins (Palumbi & Metz 1991; Aguadé 1999; Swanson *et al.* 2001). For example, Civetta & Singh (1995) reported that *Drosophila* reproductive proteins were about twice as diverse as non-reproductive proteins.

Unfortunately, the key prediction of rapid and divergent evolution of reproductive traits is not exclusive to sexual conflict. In fact, most models of sexual selection, such as the so-called 'Fisher' and 'good genes' processes, make precisely the same prediction (Lande 1981; West-Eberhard 1983; Eberhard 1985, 1996; Panhuis *et al.* 2001). Therefore, without direct knowledge that a given trait is (or has been) shaped by sexual conflict, this sort of evidence does not indicate a primary role for sexually antagonistic coevolution. To address this issue, investigators have begun to build and test a verbal theory that similarly

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assumes rapid and diversifying trait evolution, but claims to have the power to distinguish sexually antagonistic coevolution from other forms of sexual selection (Andres & Arnqvist 2001; Brown & Eady 2001; Knowles & Markow 2001; Hosken *et al.* 2002; Nilsson *et al.* 2002).

The verbal theory follows from the expectation that arms races will commonly result from the coevolution of persistence traits in males and resistance traits in females (Parker 1979; Parker & Partridge 1998; Gavrilets 2000; Gavrilets *et al.* 2001; E. Cameron, T. Day and L. Rowe, unpublished data). From this central tenet, authors have extrapolated to a multi-trait system, where the evolution of female resistance to one manipulative male trait favours evolution of a new male trait to which females do not yet have a defence. It is also assumed that there are many pathways along which males can manipulate females. In short, the theory predicts that sexually antagonistic coevolution will cause females to become more resistant to manipulative traits of males with which they have had a chance to coevolve than to those with which they have not. This contrasts with other models of sexual selection where females may benefit indirectly or directly from those males with exaggerated sexual signals. Here, it is reasoned that females may be more responsive to signals of males with which they have coevolved, because these signals are indicators of some benefit. These contrasting predictions can be tested by crossing closely related strains, and estimating the resistance of females to their 'own' males relative to that of 'other' male strains.

Two central predictions have been derived from this verbal theory. First, because there are multiple pathways with which males can manipulate females it is expected that when strains are crossed, the effectiveness of male manipulations will be dependent upon the female strain to which they are mated. This would generate interactions among male and female strains in their effect upon female mating behaviour. Second, females are expected to be best defended against males with which they have had some opportunity to coevolve (i.e. their 'own'). Thus, if for example males and females have conflicting interests over oviposition rate (with males that can induce a higher rate obtaining higher reproductive success), then females should, on average, have the lowest oviposition rate with their own males.

Several crossing experiments relevant to these ideas have already been conducted (Clark *et al.* 1999; Andres & Arnqvist 2001; Brown & Eady 2001; Knowles & Markow 2001; Hosken *et al.* 2002; Nilsson *et al.* 2002), and further experiments are ongoing. Both the results and interpretation of these experiments are mixed (Chapman *et al.* 2003). Our aim is to explore the verbal theory with a more formal treatment, and thereby provide some guidance for interpreting these studies. Our approach is to create model strains from a formal theory of sexually antagonistic coevolution, and then fully cross these lines and assess the outcome. Specifically, we seek to establish whether the main predictions of this verbal theory hold in a formal theoretical system. First, do we always observe an interaction-free pattern between male genotypes across female genotypes in a simple one-signal model? If not, then interactions are not diagnostic of a complex signal–receiver system. Second, are females that have evolved under sexually
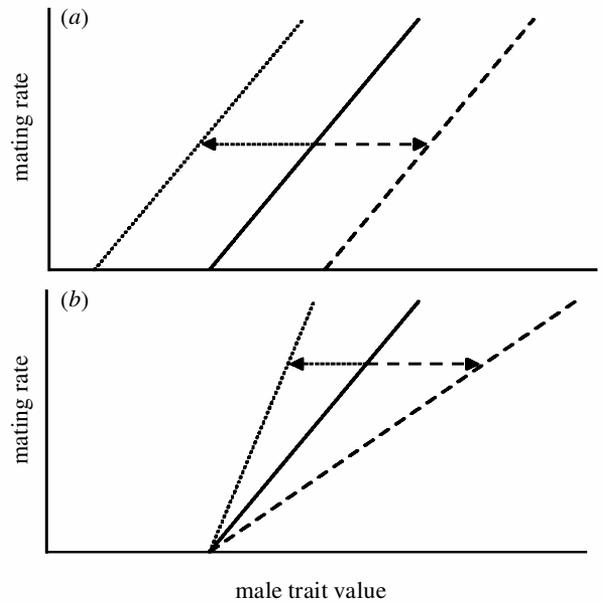

Figure 1. Females may respond to increased male stimulation by reducing the mating rate in one of two ways: (*a*) by increasing the threshold amount of the male trait required to initiate mating or (*b*) by adjusting their sensitivity (slope of the preference function) to the male trait. The female preference function is assumed to be linear in all examples.

antagonistic selection best defended against males of their own strain? We also explore the relationship between the outcome of the signal–receiver interaction (e.g. mating rate) and fitness of the interactors, because the sign of this relationship is viewed as diagnostic of sexual conflict, and has implications for speciation.

2. MATERIAL AND METHODS

Distinct theoretical strains were obtained by allowing populations to diverge according to the dynamic equations derived in Appendix A (E. Cameron, T. Day and L. Rowe, unpublished data). Briefly, we assumed conflict over mating rate. Ancestral females have an exploitable receptor for mating, and males then evolve some signal that exploits this receptor. As males evolve exaggerated signals, females pay a cost as a result of mating at too high a rate. To reduce these costs, females can evolve to resist the stimulus in one of two ways:

(i) by adjusting the quantity of male stimulation they require for mating (mating threshold); or
(ii) by adjusting their sensitivity to the male trait (i.e. the slope of the preference function) (figure 1).

Note that there is a single signal evolving in this model, in contrast to a complex signal–receptor system with multiple pathways. There are varying degrees of natural selection on all variables: female mating rate, male signal value and the female threshold and sensitivity, with natural selection favouring an intermediate value in each. The strength and sign of sexual selection on male signal value are determined by the combination of the threshold and sensitivity of the female-response curve. Although this model was devised to explore the male–female trait dynamics resulting from conflict over the rate of
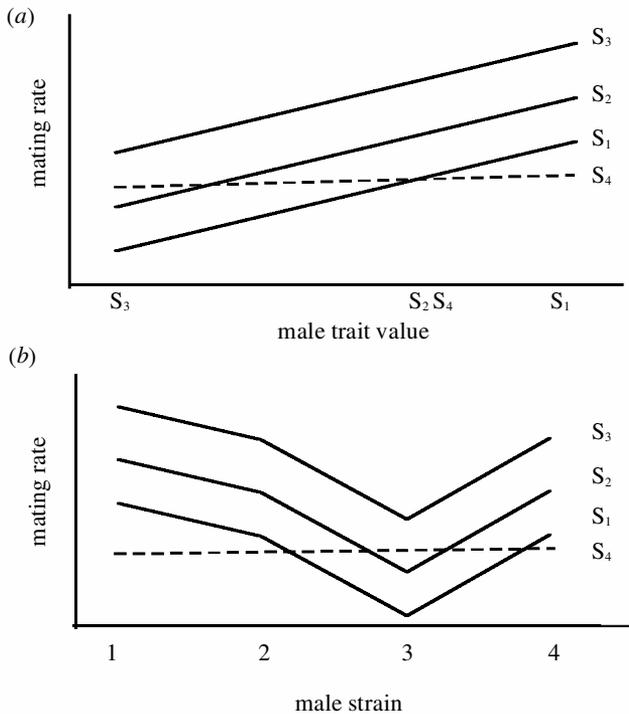
(*a*)

(*b*)

Figure 2. (*a*) Evolved female preference functions for four strains where females respond to males by altering their mating threshold (solid lines, S₁₋₃) or their sensitivity to the male trait (dashed line, S₄). Each line represents the strain-specific female preference function plotted across the distribution of male traits. The male trait value for each of the strains (S₁₋₄) is shown along the *x*-axis. (*b*) Reciprocal crosses between these strains reveal that interactions can arise when females respond to only one male trait but in different ways.

mating, the results are equally applicable to conflicts over other variables (Gavrilets *et al.* 2001).

Different strains were created by varying the initial input parameters of the model (natural selection optima for the female and male traits, and the strength of selection against deviations from these optima) as would be expected if different strains occupied different environments. The parameter values used to generate specific examples (figures 2 and 4) are given in Appendix B. All strains were allowed to diverge for a minimum of 8000 generations (or until equilibrium was reached in simulations). By altering the strength of selection on female thresholds relative to selection on sensitivity, we determined the degree to which either evolved. For example if there was strong selection on the threshold and weak selection on sensitivity, then the threshold would tend to stay at its intermediate optimum and the sensitivity would evolve away from its optimum (E. Cameron, T. Day and L. Rowe, unpublished data). Reciprocal crosses were subsequently performed among the evolved strains to determine both intra- and interstrain mating rates. We present the results graphically in a manner similar to empirical studies (Clark *et al.* 1999) in order to best identify resultant patterns.

3. RESULTS

(a) *Do interactions result only when the signal–receptor system involves multiple male stimuli and female receptors?*

To test this hypothesis we created four strains and crossed them. Strains were derived from a common ances-

tor, but evolved to different equilibria because they had varying degrees of natural selection applied to their female preference threshold and sensitivity and varying natural selection optima of the male and female traits. Strains 1, 2 and 3 had fixed sensitivities (equivalent to very strong natural selection on sensitivity), varying natural selection optima of the male trait and female threshold values and varying degrees of selection against deviations from these optimal female threshold values. This resulted in three equilibrium preference functions varying only in their threshold (figure 2*a*, solid lines). By contrast, strain 4 had a fixed threshold (equivalent to very strong selection on the threshold) and weak selection on sensitivity. Consequently, strain 4 evolved a sensitivity that differed from the other three strains. The male trait values that evolved are also shown on the horizontal axis of figure 2*a*.

We have allowed only one signal to evolve in a simple, quantitative manner. The verbal theory suggests that when these strains are crossed this should result in male genotypes being ranked similarly across female genotypes. In our example, this would mean that the mating rate induced by males from the four strains would achieve the same ranking regardless of the female strain with which they were crossed. Results for mating rate when the four strains are fully crossed are shown in figure 2*b*. When females were permitted to respond to males only by altering their mating threshold, the result was as predicted: female strains ranked similarly among males and no interactions were observed (figure 2*b*, solid lines). These lines in figure 2*b* are parallel because females that rank low in resistance with one male strain rank low with all male strains (figure 1).

However, when crosses were performed involving strains where females evolved a difference in their sensitivity to male stimulation the prediction was not upheld. Instead, it was easily possible to obtain patterns of interactions among strains that evolved. This can be seen in figure 2*b*, where the dashed line represents a strain in which female sensitivity evolved, and it is crossing the solid lines where females had evolved different thresholds. These results demonstrate that a complex signal–receptor system is not necessary to generate interactions among strains evolving under sexual conflict. Here, the interactions observed among strains result from antagonistic coevolution of a simple one-signal system.

The pattern of interactions depicted in figure 2*b* is not an atypical result of our simulations. More generally, in a single signal–receptor system no male × female interactions can occur when only the mating threshold evolves. In this case, the shape of the female preference function remains constant and it is only its relative position that varies. Female strains with a low threshold will mate at a higher rate with all male strains than those with a high threshold. By contrast, interactions between the relative success of male genotypes across female genotypes are possible whenever the shape of the female-response curve differs between divergent strains (figure 3).

(b) *In the presence of interactions, are females always better defended against males of their own strain?*

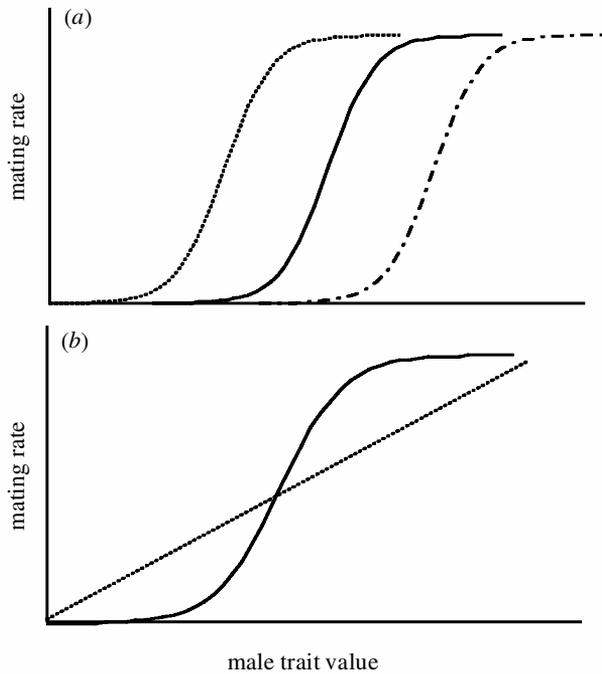If this prediction is upheld, then females will achieve their lowest mating rate when crossed with males of their

Figure 3. Female-response functions where only the mating threshold evolves (*a*) will not result in male × female strain interactions among male genotypes. However, when the sensitivity of the preference function evolves (*b*), the curves may intersect. If trait values (*y*) of male strains span both sides of the intersection point of response curves, then interactions will be observed. Imagine two male strains: s_1, which has a trait value y_1 just to the left, and s_2, which has a trait value y_2 just to the right of the intersection of the two female-response curves in (*b*). Females from the strain represented by the solid line will tend to have higher mating rates than females from the dashed line strain when crossed to males from strain S_2. However, the opposite is true when females of these two strains are crossed with strain S_1 males.

Figure 4. (*a*) Evolved female preference functions for three strains where females respond to males by altering their mating threshold and their sensitivity to the male trait. Each line represents the strain-specific female preference function (S_{1-3}) plotted across the distribution of male traits. The male trait value for each of the strains (S_{1-4}) is shown along the *x*-axis. (*b*) Crosses among these three strains demonstrate that the mating rate need not be lowest for intrapopulation matings. In this particular example, the mating rate is not lowest for intrapopulation matings in *any* of the strains.

own strain. The three theoretical strains used to test this hypothesis were generated from a model where females were permitted to respond to the male stimulus by adjusting both the threshold and sensitivity of their preference function simultaneously. Each strain was derived from a common ancestor, but the strength of natural selection acting on the two parameters of the preference function varied as follows: strain 1 evolved with natural selection on both threshold and sensitivity; strain 2 evolved with natural selection on sensitivity only; and strain 3 evolved free of natural selection on either threshold or sensitivity. Equilibrium preference functions and male trait values for each strain are shown in figure 4*a*, and the mating rates resulting from fully crossing the strains are shown in figure 4*b*.

Contrary to the verbal theory, the female response (mating rate) is not weakest for intrastrain matings. In fact, in this particular simulation, females never had the lowest mating rate when crossed with their own males. More generally, it can be seen from inspection of figure 3*a* that in crosses where the preference functions intersect, whether females are more or less responsive to males of their own strain will depend only on which side the intersection point each of the male's traits falls.
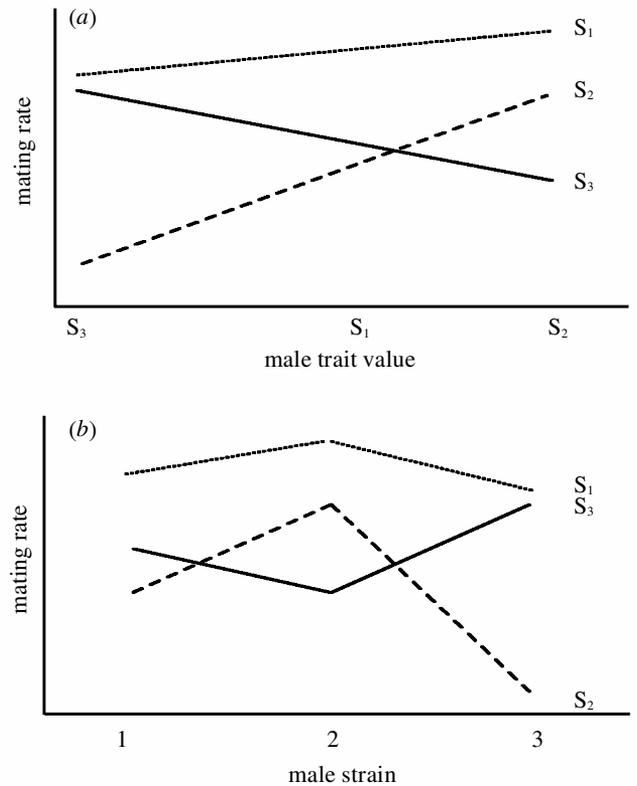
4. DISCUSSION

To determine whether sexually antagonistic coevolution, rather than a more traditional model of coevolution (i.e. good genes), is responsible for the observed rapid evolution of signal–receiver systems (and dimorphisms in general), a simple diagnostic tool would be useful. Recent verbal theory suggests that the pattern resulting from crossing recently diverged strains provides such a tool. Our results suggest that these predictions are not supported. In § 4*a* we discuss the implications of these results for the interpretation of past empirical studies. We also point to implications for related theories of speciation. Finally, we highlight the difficulties involved with attempting to distinguish among competing mechanisms of male/female coevolution by inspection of emergent patterns that result from unobserved coevolutionary processes.

(a) *Inferring details about signal receiver systems from population crosses*

It has been suggested that when strains are fully crossed, the presence or absence of interactions reveals something about the structure of the signal–receiver system. Specifically, it has been argued that the rank effectiveness of the signal from any one male strain would be independent of

female strain when the signal–receiver system involves only one signal and one receptor (the female preference; Andres & Arnqvist 2001). Alternatively, if the rank effectiveness of male signals depends upon the female strain with which they are crossed, then a complex multi-signal and receiver system is indicated (see also Clark *et al.* 1999; Hosken *et al.* 2002; Nilsson *et al.* 2002).

We found only limited support for the prediction. In those cases where the female preference function for the male signal was allowed to evolve its threshold response level only (figure 1), then no interactions occurred. Thus, under these assumptions, our analysis supports the suggestion by Andres & Arnqvist (2001) that single signal–receiver systems do not lead to interactions in strain crosses. However, when females were allowed to evolve their sensitivity to the male trait, interactions often resulted. Interactions occur because the preference functions of any two strains may evolve to intersect. An intersection of preference functions guarantees interactions, provided that the mean male trait values for the crossed strains span the intersection point of the preference functions (figure 3). There is considerable evidence that such interactions can occur when populations are crossed (e.g. Clark *et al.* 1999; Andres & Arnqvist 2001; Brown & Eady 2001; Hosken *et al.* 2002; Nilsson *et al.* 2002), but our results suggest that this fact cannot be taken as indicative of a complex signal–receptor system unless one assumes that female preference functions (or the receptor) can only evolve in threshold rather than sensitivity. We note, however, that several comparative studies have demonstrated that the sensitivity of female preference functions readily evolves (Morris *et al.* 1996; Basolo 1996, 1998; see also Rosenthal & Servedio 1999), while other studies have revealed that preference functions are quite plastic (Berglund 1993; Hedrick & Dill 1993; Wagner 1998). Therefore, an assumption of fixed slopes over evolutionary time is unlikely to hold in general.

A second idea from the verbal theory is that, in the presence of interactions, females should be least responsive to those males with which they evolved. This prediction has been tested in several species but with mixed results (Andres & Arnqvist 2001; Brown & Eady 2001; Knowles & Markow 2001; Hosken *et al.* 2002; Nilsson *et al.* 2002). In crosses of our model strains this prediction was not upheld: females may or may not be least responsive to those males with which they have coevolved (figure 4).

(b) *Comparison with other models of sexual selection and conflict*

Our model has only one male signal, and a preference function with two parameters (threshold and sensitivity). Therefore, we cannot be certain whether patterns more closely aligned with the verbal theory would result from a more complex multi-signal–receiver system (see Arak & Enquist 1993, 1995). However, for now, we cannot simply assume that the issues raised here will dissolve in a more complex system. A reasonable expectation is that the more complex the signal–receiver system, the less likely it is that one can make inferences about processes driving the evolution of the system.

We have chosen a model of sexually antagonistic coevolution to make our simulations most relevant to the original

verbal model. However, we could have generated these same patterns if we had used one of the more conventional models of sexual selection (e.g. 'Fisher' or good genes'), by including the possibility of intersecting female preference functions. Indeed, in such models of sexual selection it is possible to get any equilibrium combination of female sensitivity to the male trait and the male trait value itself, given the right parameter values (e.g. natural selection on the relevant traits). For example, within a simple Fisher model (Lande 1981), one can easily choose strains from two 'lines of equilibrium' that cross and male trait values that lie on either side of this intersection. These are the conditions that will produce male × female strain interactions, and allow females to be either most responsive to their own or to inter-strain males. Therefore, these features are not diagnostic of past sexually antagonistic coevolution.

(c) *The effects of inter-population crosses on female fitness and gene flow*

It is often assumed that fitness is a monotonic function of the value of the trait over which conflict exists. For example, in the present model this would mean that the lowest rate of mating yields the greatest fitness for females. More generally, however, there is likely to be an optimum for females such that female fitness decreases with mating either above *or below* this optimum (Arnqvist & Nilsson 2000). Thus, the *y*-axis (corresponding to the response variable discussed in § 3a,b) in graphs commonly generated to detect interactions among genotypes may be bidirectional rather than unidirectional. This has significant implications for the interpretation of results from crosses.

Intersexual conflict over mating rate can be expected whenever a difference between the optimal mating rates exists between the sexes. Over evolutionary time, selection should favour male–female trait pairs that result in the population lying between the two optima (in the conflict zone; figure 5). Outside this zone there is no discrepancy between male and female interests, and therefore there is evolutionary cooperation between the sexes (Parker 1979; Parker & Partridge 1998). This reasoning suggests that intrapopulation matings will probably fall within the conflict zone, but there is no reason to expect the same of interpopulation matings. When interpopulation crosses result in mating rates falling outside this zone of conflict, female fitness can be either increased or reduced relative to intrapopulation crosses (figure 5). One consequence is that comparisons of the fitness of females in inter- versus intrapopulation crosses is not in itself diagnostic of sexual conflict. Female fitness in interpopulation crosses may be either greater or less than in intrapopulation crosses.

Some previous studies have demonstrated that sexual conflict can drive speciation through the rapid divergence of male–female signal–receptor systems in different populations (Gavrilets 2000; Gavrilets & Waxman 2002). Other studies have suggested that sexual conflict can enhance or inhibit speciation depending upon who gains the upper hand in the conflict (Parker & Partridge 1998). For example, if males gain the upper hand, then their success at manipulating females will increase gene flow between populations thereby inhibiting speciation, whereas if females can gain the upper hand then the reverse will be true (Parker & Partridge 1998). Still there
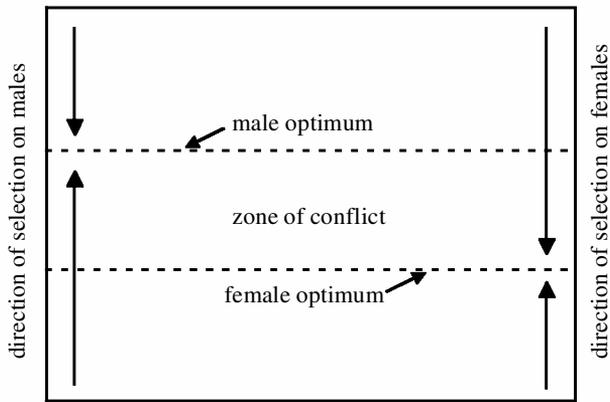
Figure 5. When optima differ between the sexes, selection will tend to drive trait values into the zone of conflict. Within this zone, selection on females opposes selection on males. Outside this zone, below the female optimum or above the male optimum in this figure, there is no longer any conflict between the sexes. Matings between males and females that have coevolved in the same population are expected to fall within the conflict zone, but there is no such expectation when males and females from different populations are mated.

have been other suggestions that the effect of sexual conflict on speciation will change over time as populations diverge, initially enhancing but eventually inhibiting the possibility of gene flow between diverging populations.

The theoretical results presented here suggest it is unlikely that we will be able to make predictions about speciation without detailed knowledge of how the relevant preference functions are evolving. In general, the extent to which gene flow will occur between populations depends on how females have evolved to respond to male stimuli. If, for example, females have evolved a low sensitivity to differences in male stimuli, then this will probably increase gene flow between populations regardless of who (if either party) has the upper hand in the conflict, as this will promote indiscriminate mating by females. Alternatively, evolutionary changes in the threshold responses of females might well decrease gene flow between populations. Even this latter prediction is not straightforward, however, because the extent to which interpopulation gene flow will occur depends on the evolved female-response functions of both populations as well as the male traits that have evolved in both populations. Thus, it seems unlikely that any sort of simple prediction regarding the relationship between sexual conflict and speciation will be possible.

5. CONCLUSIONS

Our results suggest that few reliable inferences can be made about the detailed processes and pathways underlying male–female coevolution of signal–receiver systems from the outcome of population crosses alone. Specifically, the presence or absence of interactions, and the female response to intra- versus inter-population matings, cannot be used to infer attributes of the signal–receiver system, or to distinguish sexually antagonistic coevolution from a number of other models of sexual selection. More generally, pattern is a poor predictor of process. Notably, similar conclusions have been made in the past regarding

earlier attempts to distinguish processes of sexual selection from emergent patterns. For example, it is now understood that among-population correlation between male trait exaggeration and strength of female preference does not imply a primary role of the 'Fisher' process (Houde & Endler 1990; Kirkpatrick & Ryan 1991). Similarly, the presence of condition dependence of preferred male traits does not imply a primary role of the 'good genes' process (Rowe & Houle 1996). We suggest that careful studies of signals and receptors (the preference) and the consequence of the outcome of their interaction for male and female fitness are required first. Recent studies of these signal-receptor pathways are promising (Chapman 2001; Swanson & Vacquier 2002). Once these features of the system are better understood, it may then be possible to make predictions about the outcome of population crosses and then to test them.

APPENDIX A

Male and female fitness (W_m and W_f) are described by

$$W_f = [1 - a(\psi - P_{opt})^2] + [1 - s_{x_1}(x_1 - \theta_{x_1})^2]$$
$$+ [1 - s_{x_2}(x_2 - \theta_{x_2})^2], \tag{A 1a}$$

$$W_m = \lceil 1 + 2b\psi \rceil + [1 - s_y(y - \theta_y)^2], \tag{A 1b}$$

where x_1 represents a female trait determining the quantity of male stimulation required for mating (a decrease in the value of x_1 corresponds to an increase in the mating threshold), x_2 represents a female trait determining sensitivity to the male stimulus, and y represents the male stimulus. The parameters s_{x_1}, s_{x_2} and s_y control the strength of natural selection on the female and male traits and θ_{x_1}, θ_{x_2} and θ_y specify the optimal value of these traits under natural selection. P_{opt} represents the optimal female mating rate, a is a parameter scaling fitness consequences of deviating from this optimum (Gavrilets 2000), b is a measure of the strength of sexual selection on males, and ψ is a function describing the rate of mating and is defined as

$$\psi(x_1, x_2, y) = x_2 y + x_1. \tag{A 2}$$

Given equations (A 1), a quantitative-genetic model (Lande 1976, 1980, 1981) for the evolutionary dynamics of these three traits (in continuous time) can be derived using the approximations outlined in Iwasa *et al.* (1991; see also Taylor 1996) to obtain

$$dx_1/dt = v_{x_1}(-a(\psi - P_{opt}) - s_{x_1}(x_1 - \theta_{x_1})), \tag{A 3a}$$

$$dx_2/dt = v_{x_2}(-ay(\psi - P_{opt}) - s_{x_{21}}(x_2 - \theta_{x_2})), \tag{A 3b}$$

$$dy/dt = v_y(bx_2 - s_y(y - \theta_y)). \tag{A 3c}$$

Additive genetic variances of the female and male traits are v_{x_1}, v_{x_2} and v_y, respectively. Indirect evolutionary responses due to selection on genetically correlated traits

are not included in equations (A 3), but these have no effect on the evolutionary equilibria that are observed (E. Cameron, T. Day and L. Rowe, unpublished data).

APPENDIX B

The input parameter values for figure 2 were as follows:

(i) S_1: $v_{x_1} = 0.05$; $v_y = 0.05$; $a = 0.1$; $b = 0.5$; $P_{opt} = 0$; $y_{initial} = 2$; $x_{1\,initial} = 2$; $x_2 = 1$; $\theta_y = 7$; $\theta_{x_1} = 0$; $s_y = 0.5$; $s_{x_1} = 0.25$;

(ii) S_2: $v_{x_1} = 0.05$; $v_y = 0.05$; $a = 0.1$; $b = 0.5$; $P_{opt} = 0$; $y_{initial} = 2$; $x_{1\,initial} = 2$; $x_2 = 1$; $\theta_y = 5$; $\theta_{x_1} = 0$; $s_y = 0.5$; $s_{x_1} = 0.5$;

(iii) S_3: $v_{x_1} = 0.05$; $v_y = 0.05$; $a = 0.1$; $b = 0.5$; $P_{opt} = 0$; $y_{initial} = 2$; $x_{1\,initial} = 2$; $x_2 = 1$; $\theta_y = 0$; $\theta_{x_1} = 2$; $s_y = 0.5$; $s_{x_1} = 0.5$;

(iv) S_4: $v_{x_1} = 0.05$; $v_y = 0.05$; $a = 0.1$; $b = 0.5$; $P_{opt} = 0$; $y_{initial} = 2$; $x_1 = 1$; $x_{2\,initial} = 1$; $\theta_y = 7$; $\theta_{x_1} = 1$; $s_y = 1$; $s_{x_2} = 1$.

The input parameter values for figure 4 were as follows:
$v_{x_1} = 0.05$; $v_{x_2} = 0.05$; $v_y = 0.05$; $a = 0.5$; $b = 0.5$; $P_{opt} = 0$; $y_{initial} = 2$; $x_{1\,initial} = 0.000\,000\,000\,01$; $x_{2\,initial} = 1$; $\theta_y = 2$; $\theta_{x_1} = 0$; $\theta_{x_2} = 1$; $s_y = 0.5$; S_1: $s_{x_1} = 0.5$, $s_{x_2} = 0.5$; S_2: $s_{x_1} = 0$, $s_{x_2} = 0.5$; S_3: $s_{x_1} = 0$, $s_{x_2} = 0$.

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