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Condition-dependent sexual selection can accelerate adaptation

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ABSTRACT

Sexual selection is responsible for much of the spectacular natural diversity of mating traits. It is unclear, however, how this powerful evolutionary force affects the evolution of traits unrelated to mating. Four recent theoretical studies have argued that sexual selection might increase the rate of adaptation, but each relies on the assumption of a substantial positive covariance between male condition (non-mating fitness) and display. Here we demonstrate, with an explicit genetic model, that sexual selection itself can easily lead to the evolution and maintenance of this covariance. This process occurs through the evolution of condition-dependent male display and the resultant transfer of genetic variance for condition into variance in male display. We also track the effect of the covariance between condition and display on the rate of adaptation. Our results demonstrate a powerful synergy between natural and sexual selection that can elevate population mean fitness. Moreover, this synergy can greatly accelerate the rate of adaptation, making the feedback between natural and sexual selection a particularly potent force in changing environments. This has important implications for several key evolutionary processes, including the evolution of sex, sexual conflict and speciation.

Keywords: Fisher process, good genes, individual-based model, simulation.

INTRODUCTION

Sexual selection has generally been viewed as reducing population mean fitness (Haldane, 1932; Lande, 1980; Kirkpatrick, 1982; Grafen, 1990b; Kirkpatrick and Ryan, 1991; Price *et al.*, 1993; Tanaka, 1996; McLain *et al.*, 1999; Gavrilets *et al.*, 2001; Houle and Kondrashov, 2001). Elaborate display traits clearly cost their bearers, these costs can reduce female or offspring fitness (by reducing direct benefits or paternal care), and evolved female preferences can also reduce female fitness. Recently, however, it has been demonstrated that sexual selection can have a beneficial effect on the evolution of non-sexual traits. Sexual selection can enhance the purging of genetic load, thereby providing a considerable

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advantage to sexual reproduction (Agrawal, 2001; Siller, 2001), improving population mean fitness. Similarly, sexual selection can enhance adaptation by increasing the spread of beneficial alleles (Proulx, 1999, 2001, 2002; Whitlock, 2000). Each of these conclusions, however, is based on the assumption that male display is an honest indicator of male condition (i.e. display is condition-dependent or display intensity and condition show positive genetic covariance). Early theoretical objections to this assumption in the form of the 'lek paradox' (Borgia, 1979) have led many to question the validity of such 'good genes' arguments (Taylor and Williams, 1982; Kirkpatrick, 1996; Kirkpatrick and Barton, 1997). The lek paradox states that strong directional selection on male display can remove variance in display. Without variance in display, the covariance between display and condition that is necessary for good genes models will not develop.

The process of 'genic capture' provides one potential resolution to the lek paradox (Rowe and Houle, 1996) with unanticipated consequences for population mean fitness. This process is also a hidden assumption of models where sexual selection purges load (Agrawal, 2001; Siller, 2001) and, indeed, of good genes sexual selection models generally. If condition is defined broadly as the amount of resources available for allocation to fitness-enhancing traits (not related to mating success), then most genes contribute to condition in some way, and there should be abundant genetic variance in condition. When male displays are costly to produce and investing in display increases mating success, then we expect condition-dependent display to evolve. In other words, we do not expect display traits to be purely 'Fisherian' (i.e. traits that evolve solely due to their genetic covariance with preference), but instead that they will evolve dependence on condition. This is true because after genetic variance is exhausted at the loci initially involved in display, males will be selected to alter their display based on their genetic quality at other loci. So genic capture resolves the lek paradox by providing abundant genetic variance for display traits through condition. The evolution of condition dependence produces genetic covariance between display and condition and this is what makes good genes models of sexual selection work [including the models of Agrawal (2001) and Siller (2001)].

Here we demonstrate that, in addition to solving the lek paradox, genic capture can also have the effect of enhancing adaptation in fitness components unrelated to mating. To some, this may sound like an obvious restatement of good genes sexual selection models. We believe that genic capture, as demonstrated here, makes clearer the mechanism by which good genes sexual selection can improve population mean fitness, as well as drawing attention to adaptation in traits unrelated to mating. More rapid and more extensive adaptation can have important consequences for speciation and for resolving sexual conflicts of interest. By exposing genetic variance in traits unrelated to mating to a combination of natural and sexual selection, condition-dependent sexual selection has the effect of increasing equilibrium mean fitness, as well as the speed at which the population is able to attain this equilibrium. This latter effect can result in a pronounced positive effect of sexual selection on population adaptation, especially when environmental conditions are fluctuating.

Several population genetic models have dealt with condition-dependent sexual selection (particularly relevant models include: Grafen, 1990a,b; Iwasa and Pomiankowski, 1994; Agrawal, 2001; Houle and Kondrashov, 2001; Siller, 2001). All of these models are equilibrium models that ignore the advantages of sexual selection which accrue before the equilibrium is reached (a pre-equilibrium advantage). None examine the effect of condition-dependent sexual selection on the rate and extent of adaptation in non-mating traits. Only Iwasa and Pomiankowski (1994) examined the evolution of condition

dependence in detail (as opposed to assuming it has already evolved). However, this model suffers from an internal constraint – the genetic variance–covariance matrix relating display, preference and condition is assumed to be constant. This assumption introduces an internal inconsistency in that the extent to which display depends on condition can evolve but the genetic covariance between display and condition cannot. If condition dependence evolves, we do not expect the covariance between display and condition to remain constant. Two recent models (Agrawal, 2001; Siller, 2001) show a positive effect of sexual selection on population mean fitness. These models implicitly assume genic capture is occurring rather than letting it evolve, and they focus on sexual selection helping to pay the twofold cost of sex rather than examining the evolution of costly displays and preferences. The results we present here show that it is important to include the temporal dynamics of selection and the evolution of covariance because only examining equilibrium conditions can underestimate the extent of co-evolution between display, preference and condition. Our model is designed to avoid what we consider weaknesses of previous population genetic models while answering the following questions:

1. How does genic capture operate?
2. How can condition-dependent sexual selection accelerate adaptation and increase population mean fitness?

Understanding how genic capture works and how it can solve the lek paradox is the key to understanding how sexual selection can accelerate adaptation. For this reason, we begin by developing a model within which genic capture can operate. Rowe and Houle (1996) provided a model for how genic capture can solve the lek paradox. Here we demonstrate mechanistically how genic capture operates. Our model shows how genetic variances and covariances change dynamically as condition dependence evolves. This is a genetically explicit model (as opposed to a traditional quantitative genetic model), and we can allow genetic variances and covariances to evolve rather than making assumptions about them that may introduce unrealistic constraints. Using this model we show how genic capture operates, solving the lek paradox and allowing display traits to become more elaborate than would otherwise be possible. We then demonstrate how this same process can lead to faster and more extensive adaptation in condition (and underlying traits). To demonstrate the fitness advantage populations with condition-dependent sexual selection have over those without it, we change the environmental optimum for a trait that contributes to condition and compare the rate and extent of adaptation between population types. Changing the environmental optimum also allows us to demonstrate the potential for a substantial pre-equilibrium advantage to condition-dependent sexual selection. Finally, we demonstrate how this pre-equilibrium advantage can cause the advantage of condition dependence to be underestimated, especially in fluctuating environments. We end by discussing our results in the context of equilibrium models and some of the implications of our results for several key evolutionary processes, including the evolution of sex, sexual conflict and speciation.

METHODS

We use an individual-based genetic model, where we allow genetic variances and covariances between traits to evolve. The model has four basic traits: (1) a resource acquisition trait (g) that determines condition; (2) a trait that specifies the extent to which

display trait expression depends on male condition (b); (3) a trait representing the condition-independent part of male display (z); and (4) the intensity of female mating preference (y). All traits are determined by multiple, additive loci (see Simulation details below). Trait g can be thought of as one trait or as a composite of several traits that determine fitness unrelated to mating success. Condition (C) and male display trait (T) are derived from the first three basic traits in such a way that when there is no condition-dependent trait expression ($\bar{b} = 0$), $\bar{T} = \bar{z}$. Otherwise, T has condition-dependent and condition-independent components (see Simulation details below).

To clearly demonstrate the effect that the evolution of condition dependence can have on the evolution of fitness as well as on the evolution of male display and female preference, we compared simulations with female choice where condition dependence was allowed to evolve with simulations where it was prevented from evolving (the Fisher process only), as well as with simulations in which there was no sexual selection at all.

Simulation details

We use a diploid additive genetic system with unlinked loci (C code available upon request). Alleles at each locus added either 1 or 0 to each trait (e.g. the phenotypic value of a trait coded for by five diploid loci could range from 0 to 10). Alleles were initially assigned from a random normal distribution, centred on half of trait maximum (except trait b, which started with all loci set to zero). There were 20 loci for g, 5 for z (because we assume there are more genes contributing to g than T; Rowe and Houle, 1996), 5 for y and 20 for b. Mutation occurred at 0.02 mutations/locus/generation (with 100 loci, that means 2 mutations/individual/generation). These numbers of loci and the rate of mutation were chosen to balance competing goals of minimizing computation time and avoiding fixation of traits with too low a mutation rate while still being able to demonstrate the lek paradox. Using realistic lower mutation rates would require using realistic much higher numbers of loci. Not only do we not know what the realistic numbers of loci are for these traits, particularly for condition, the numbers are likely to be too large to deal with on available computers.

Condition was a Gaussian function of an individual's g phenotype:

$$C = \mathrm{e}^{-(g - g_{\mathrm{opt}})^2/2\omega_C^2} \tag{1}$$

where g_{opt} is the optimal g phenotype and ω_C, how strongly C scales with $g - g_{\mathrm{opt}}$, was set at 20. Though there will be directional selection for increased resource acquisition, individual traits are expected to experience stabilizing selection in particular environments (e.g. feeding morphology should be tuned to gather the most abundant food type).

Once condition dependence evolved ($b > 0$), display trait increased as a linear function of relative condition and condition dependence. For computational ease, we supposed that if condition dependence was maximal, the condition-dependent contribution to display would range linearly from the maximum possible to zero depending on condition. If condition dependence phenotype was less than maximal, the contribution to display would range from something less than the maximum to zero and some range of low condition would contribute nothing to display. Male trait was therefore calculated in terms of the deviation of g from its optimum ($\delta_g = |g - g_{\mathrm{opt}}|$) in the following way. When $b - \delta_g > 0$,

$$T = z + b - \delta_g \tag{2}$$

and when $b - \delta_g \leq 0$,

$$T = z \tag{3}$$

Since the range of possible b phenotypes was the same as for g, the above function produced the largest contribution to T when g was at its optimum.

The probability of male survival depended on both his condition and his level of trait expression in the following way:

$$P_{\mathrm{sm}} = C\mathrm{e}^{-T^2/2C\omega_T^2} \tag{4}$$

where ω_T was set at 20. This made the display trait (T) a condition-dependent handicap (Maynard Smith, 1985), where the strength of natural selection scaled negatively with condition.

The probability of female survival depended on her condition and her preference intensity:

$$P_{\mathrm{sf}} = C\mathrm{e}^{-y^2/2\omega_y^2} \tag{5}$$

where ω_y was set at 30. Each surviving female produced one son and one daughter initially. If these offspring survived, they became potential parents in the next generation. We kept the number of mothers constant (at 1000) by randomly choosing females and allowing them to have an additional daughter until there were 1000 (this amounts to soft selection on females). Equations (4) and (5) result in directional selection for increased C.

The probability of females choosing to mate with a male having a given level of display from among all surviving males was (according to an open-ended, psychophysical preference function; Lande, 1981; Higashi *et al.*, 1999):

$$P_{\mathrm{pf}} = \frac{\mathrm{e}^{\alpha Ty}}{\Sigma_T p(T)\mathrm{e}^{\alpha Ty}} \tag{6}$$

where female preference efficiency $\alpha = 0.2$ and $p(T)$ is the number of males with display trait value T.

Analysis of simulation output

After 150 generations, we shifted the natural selection optimum g from 10 to 30. The first optimum was set to let the genetic system reach equilibrium. Shifting to the second optimum mimics a change in the environmental optimum for the resource acquisition trait g. Traits were considered to be in evolutionary equilibrium when their values changed less than 0.05% of their maximum possible values between generations. We estimated the rate of change of traits by dividing equilibrium trait values (the extent, as compared to trait value of zero) by the time it took to reach these values. We repeated each set of simulations 10 times and we report medians. We compared rates and extents of evolution for equilibrium values using the Wilcoxon rank-sum test (equivalent to Mann-Whitney U) with continuity correction.

To estimate the effect of sexual selection on the evolution of condition, we assumed that our measure of average condition was equivalent to net reproductive rate (R_0) in a

population that grows according to the following rule ($N(t) = N(0)\ \Pi_{i=0}^{t} C(i)$, where $N(t)$, population size at time t, is a function of the product of mean condition at each time i) when released from density constraints. This model of population growth assumes that costs paid by males for producing elaborate displays do not affect population growth rate (Agrawal, 2001; Siller, 2001) but that changes in condition do affect population growth rate. We calculated the *product* of mean condition for each population ($\Pi_{i=0}^{t} C(i)$). Then we found the proportional difference in this product between a population in which condition dependence was allowed to evolve and one not experiencing sexual selection (relative fitness advantage of condition dependence). This is equal to the proportional difference between the geometric means for the two types of populations. To measure the pre-equilibrium effect of condition dependence (after it has evolved), we focused on the proportional difference from the generation when we switched the optimum for g (150) until mean relative fitness reached its equilibrium value in the population without sexual selection. The proportional difference in *mean* condition between the two types of populations in the generation when the population without sexual selection reached its equilibrium value was taken as the *equilibrium* advantage to sexual selection (equivalent to the estimate of Siller, 2001, for our populations). The proportional difference in the *product* of mean condition one generation earlier is the additional advantage during the period where the populations are approaching equilibrium. By looking at the proportional difference in mean condition between zero and 150 generations, we can evaluate how the relative fitness advantage of condition dependence evolves as condition dependence itself is evolving.

We also estimated the advantage to sexual selection, in terms of increased mean condition, when the environment fluctuates by letting the optimal g vary through time (comparing data from an arbitrary one of the 10 simulations for each type of population). We assumed that optimal g fluctuates between 10 and 30 at various frequencies. Then we calculated the advantage to sexual selection based on what proportion of the time the population spent in equilibrium. This allowed us to estimate the frequency of environmental fluctuations below which underestimates of the advantage to condition-dependent sexual selection would be most dramatic.

RESULTS

When sexual selection was excluded, costly male display (T) slowly decreased from its starting level towards zero (dropping from 5.0 to 4.3 in 300 generations on average; Fig. 1a). When sexual selection was included but with no condition dependence (just the Fisher process), male display evolved to mutation selection balance near its maximum, driving the variance in the display trait to near zero (Fig. 1b), and illustrating the lek paradox. Comparing T when condition dependence was allowed to evolve to when only the Fisher process was included, T evolved farther [median extent of evolution: 46.73 as compared to 9.68; Wilcoxon rank-sum test (WRS): $Z = 3.74$, $P = 0.0002$] and faster (median rate of evolution: 3.02 as compared to 1.61; WRS: $Z = 3.75$, $P = 0.0002$) than when just the Fisher process was included (Fig. 1a). The variance in T stayed at higher levels when condition dependence was allowed to evolve than when sexual selection was excluded (Fig. 1b).

Female preference was more intense when condition dependence was allowed to evolve than when it could not evolve (median y of 6.987 compared to 5.227; WRS: $Z = 3.36$, $P = 0.0008$). Female preference evolved away from zero due to a correlated response to

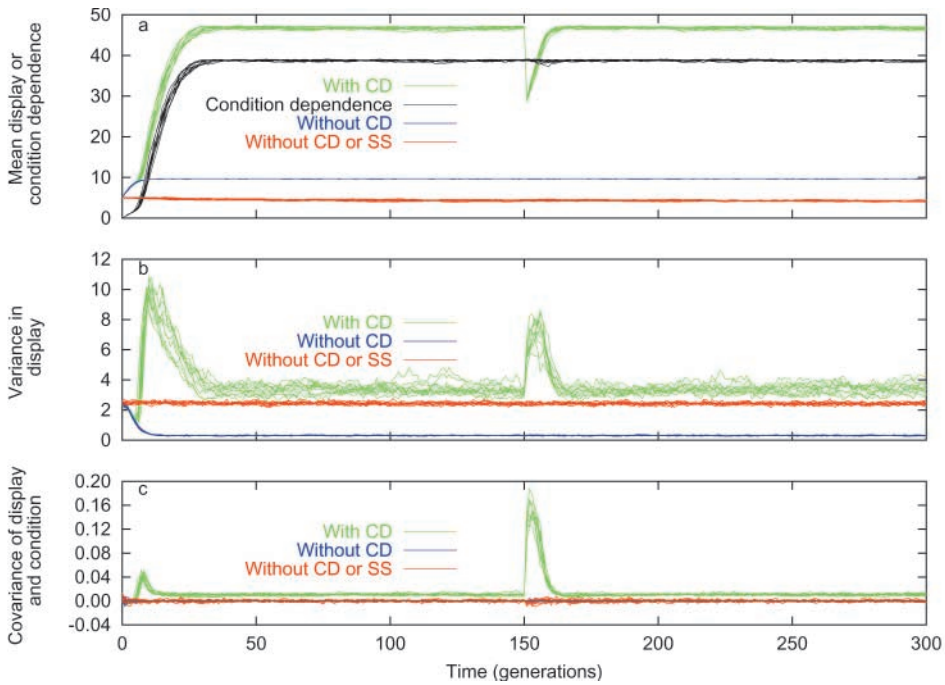

Fig. 1. Evolution with and without condition dependence (CD) and sexual selection (SS). (a) Mean male display trait (T) slowly decreases without SS, quickly fixes for a value near its maximum (10) with SS but without CD, and evolves farther and faster (once b increases) with CD and SS. (b) Variance in T remains steady without SS, quickly decays to near zero with SS but without CD, and reaches higher levels with spikes when the resource acquisition trait (g) is evolving to a new optimum with CD and SS. (c) Covariance between display and condition hovers around zero when either there is no SS or when it is not condition-dependent. The low variance in T keeps the covariance closer to zero when there is SS alone. When there is SS and CD, the covariance is consistently positive, demonstrating how the evolution of CD solves the lek paradox.

selection for increased display (positive covariance between display and preference; Fig. 2a), and as condition dependence evolved, due to an additional transient correlated response to selection on condition [transient because this covariance disappears once condition trait g and display reach mutation selection balance (Fig. 2b)]. These two correlated responses result in a self-reinforcing evolutionary escalation of preference and condition (i.e. non-mating fitness) in much the same way as occurs between preference and male display in traditional models of the Fisher process (Andersson, 1994). This predicted covariance between condition and preference is in accord with recent empirical observations (Bakker *et al.*, 1999).

Sexual selection acted on condition and condition dependence indirectly through their joint effects on display. These effects combined with the effects of natural selection for increased condition. Once condition dependence develops, there is persistent positive covariance between display and condition (Fig. 1c). The combined effects of natural and sexual selection brought about by condition dependence reduced the variance in the condition-related trait g compared with sexual selection without condition dependence

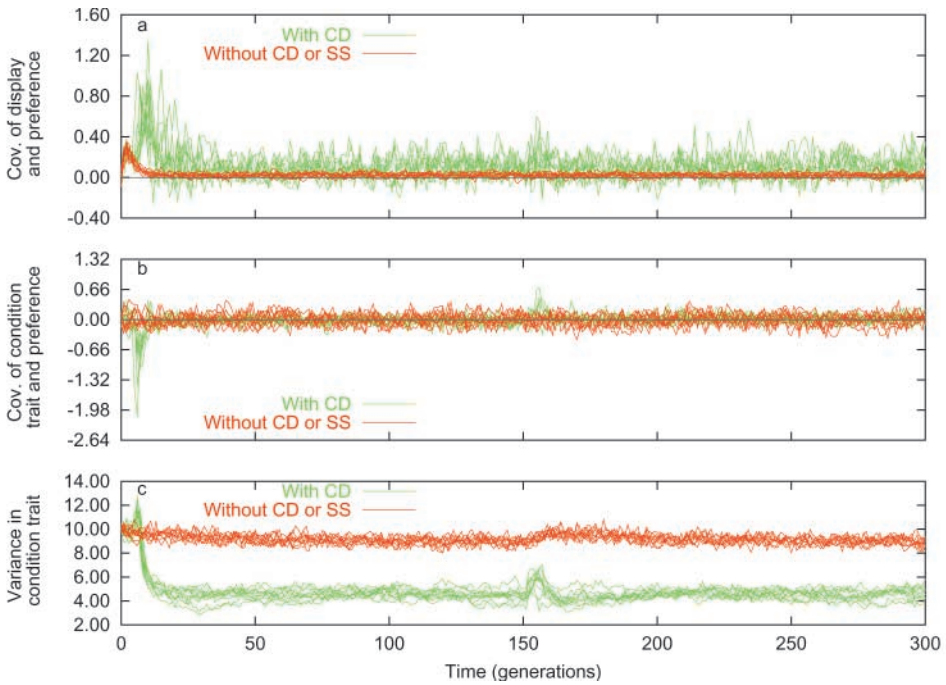

Fig. 2. Covariance between preference and (a) display or (b) condition (g); (c) variance in g. Covariance between preference and g is shown rather than condition to show the effect on the underlying trait g. Negative covariance between this trait and preference translates into positive covariance between condition and preference because initially reductions in g (from initial value of 20 towards optimum of 10) increase condition. Data for simulations without sexual selection (not shown) were either centred on zero (a, b) or matched data for simulation without condition dependence (b, c).

(Fig. 2c). To measure the rate and extent of adaptation, we calculated the mean condition (mean fitness) of our simulated population in each generation:

$$\frac{\sum_i^N C_i}{N}$$

where i is the index for N individuals. As expected, there was no difference in the extent or the rate of adaptation when there was no sexual selection as compared to when sexual selection was not condition-dependent (see Fig. 3a; median extent of adaptation: condition-independent = 0.93, no sexual selection = 0.93; WRS: $Z = 0.57$, $P = 0.57$; median rate of adaptation: condition-independent = 0.03, no sexual selection = 0.027; WRS: $Z = 1.64$, $P = 0.10$). However, the rate of adaptation of condition or mean fitness was dramatically higher when condition dependence was allowed to evolve (see Fig. 3a; median rates of adaptation: condition-dependent = 0.076, no sexual selection = 0.027; WRS test comparing rates: $Z = 3.76$, $P = 0.0002$). The extent of adaptation was also much higher with condition-dependent sexual selection than without it (see Fig. 3a; median extent of adaptation: condition-dependent = 0.99, no sexual selection = 0.93; WRS test comparing extent: $Z = 3.08$, $P = 0.0002$). So condition-dependent sexual selection allowed the resource acquisition trait to evolve more rapidly and get closer to its optimum.

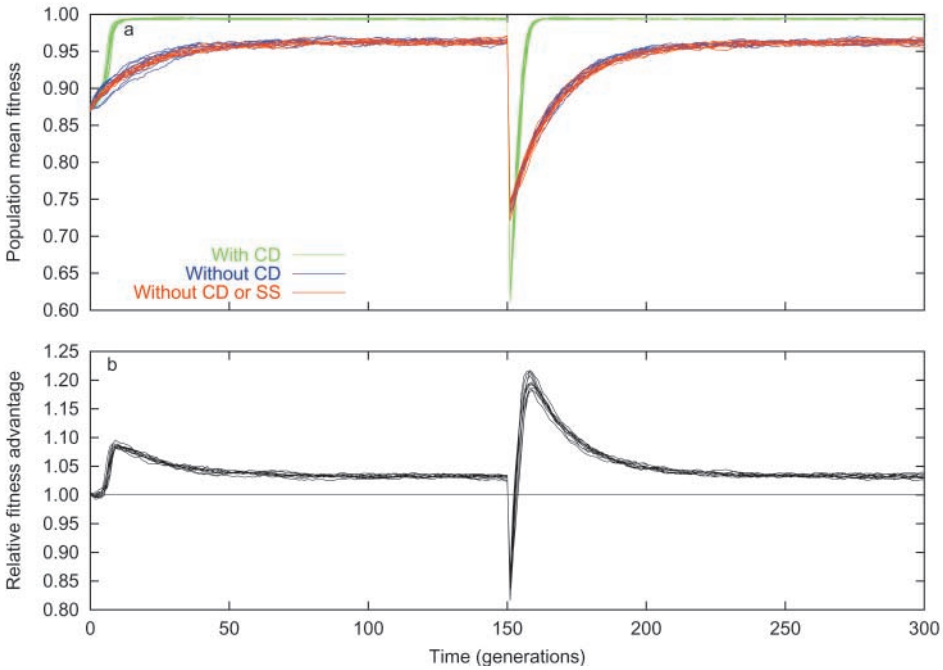

Fig. 3. Population mean fitness and the relative advantages of condition-dependent sexual selection. Zero to 150 generations shows what happens while condition dependence is evolving; 150–300 generations shows what happens after the optimimum for the condition trait (g) shifts from 10 to 30. (a) Evolution of population mean fitness (mean condition; assuming density-independent population growth). With condition dependence (CD) and sexual selection (SS), population mean fitness evolves farther and faster than when there is no SS. There is a larger decrease in mean fitness in populations with SS and CD after the change in optimum g because, immediately after the shift, g is farther from its optimum in this population than in either of the others. (b) Relative fitness advantage of SS with CD, measured as the proportional difference in average condition when CD is allowed to evolve versus when there is no SS (in a given generation). The largest relative fitness advantage occurs before equilibrium (between 150 and 200 generations). This element of the potential advantages of CD and SS for population mean fitness is missing from equilibrium analyses.

Importantly, with a simple model of population growth, the total fitness advantage due to sexual selection is largest before equilibrium is reached for male display (between 150 and 200 generations; Fig. 3b). This means that before equilibrium is reached, for our model parameters, a population with condition-dependent sexual selection could grow as much as 20% faster than one with no sexual selection. After equilibrium, the advantage is only 5% (see Methods for details). Particularly if the optimum for traits contributing to condition shifts regularly, estimates of the advantage can be improved by considering how the rate of adaptation in condition changes with time. For our simulated populations, if the environmental optimum g shifts less frequently than every five generations, using only equilibrium estimates of the advantage causes underestimation of the total fitness advantage due to sexual selection. These results suggest that past theory considering the fitness benefits of sexual selection at equilibrium may have considerably underestimated its power by ignoring pre-equilibrium advantages (Agrawal, 2001; Siller, 2001).

DISCUSSION

Our results demonstrate the capture of genetic variance in condition into sexual display using a mechanistic model that allows the evolution of condition-dependent display. These results demonstrate how condition-dependent sexual selection can solve the lek paradox. Once condition dependence evolved, variance in male display trait stayed at a higher level, leaving room for the covariance between male display and condition that makes good genes models work. Interestingly, the male display trait evolved farther and faster than it would have without this capture. Evolving farther was purely a result of having more loci in our additive model and not the result of condition-dependent sexual selection removing more of the alleles that do not increase the display trait (P.D. Lorch and S. Proulx, unpublished results). It would be interesting to allow linkage to evolve and to examine different forms of epistasis to determine whether condition-dependent sexual selection could have effects on display independent of the number of loci. Evolution of display occurred more quickly because of the self-reinforcing feedback between sexual and natural selection arising from the positive covariances between preference, display and condition.

These covariances also resulted in faster and more extensive adaptation in condition. Our results are the first to show how this sort of feedback develops as condition dependence evolves. Although the details of the rate and extent of adaptation are undoubtedly dependent on our chosen genetic architecture and parameters, we expect this general acceleration of adaptation to be important in nature for three reasons: (1) most genes are expected to have an effect on condition; (2) strong sexual selection is likely to lead to condition dependence (Rowe and Houle, 1996; Jennions *et al.*, 2001); and (3) condition dependence as we have defined it means that natural and sexual selection will combine to increase condition.

We believe that the genetically explicit simulation model used here has several advantages over other approaches. We were able to allow variances and covariances to develop as condition dependence evolved, rather than assuming that they were positive and constant, for example. Simplifying assumptions about variances and covariances, necessary to make quantitative genetic models tractable, were not necessary for our simulations. As condition dependence evolved, variance in display and covariance between display and preference were not constant (Figs 1a,b, 2a). With our approach, we could watch the consequences of linkage disequilibrium between display and preference develop, then decay and then increase again as condition dependence developed (see first 20 generations of Fig. 2a). Surprisingly, the spread of preference- and condition-related alleles occurred even with large fluctuations in the covariance between female preference and male trait.

The benefits of this explicitly genetic approach are tempered by the need to make specific assumptions (e.g. about genetic architecture, mutation rates); however, we do not feel that they affect the qualitative results we have presented. These sorts of genetically explicit models provide a tractable way to study complex sexual selection models without having to assume that traits are at equilibrium. This last point gave us insight that we might not otherwise have gained about what happens before equilibrium is reached.

Our results demonstrate the potential for a large pre-equilibrium advantage of condition-dependent sexual selection that has been missed in equilibrium models of sexual selection. This potentially large advantage may lead to underestimates of the importance of sexual selection as a force for improving population mean fitness, particularly in fluctuating environments. An example where such an underestimate may matter outside the field of sexual selection comes from theory for the evolution of sex in fluctuating environments

(reviewed in Otto and Michalakis, 1998). Current theory states that sex (and the genetic recombination that accompanies it) can evolve in response to fluctuating environments only under a narrow set of conditions, unlikely to be met in nature. The critical conditions for recombination to be favoured are that, although the environmental optimum is fluctuating, there must be strong net directional selection and weak negative epistasis. Kondrashov and Yampolsky (1996) showed that, under conditions that will select for increased recombination, fluctuations in the optimum must be so large that 30% of the population must die or fail to reproduce. Condition-dependent sexual selection, as modelled here, improves a species' ability to track a fluctuating environmental optimum. This should improve the ability of a host to out-evolve its parasites, for example, but it also reduces the net amount of directional selection in fluctuating environments (mean phenotype is closer to the optimum, on average). More work clearly needs to be done to determine whether condition-dependent sexual selection makes the evolution of sex and recombination more or less likely in fluctuating environments.

Although sexual selection is more often thought of as working in opposition to natural selection, the net consequences of these two forces on population fitness have received little attention. One context in which they have received attention is that of sexual conflict. When sexual selection involves sexual conflict, one might expect population fitness to decline (Holland and Rice, 1998; Gavrilets *et al.*, 2001). Empirical support for this decline is conflicting. Promislow *et al.* (1998) showed that after 17 generations where female *Drosophila melanogaster* were either allowed to choose mates (promiscuous treatment) or were randomly assigned single mates (monogamous treatment), the offspring of promiscuous females had higher adult survivorship and body size than offspring of monogamous females. In a similar selection experiment on the same species (after 47 generations), Holland and Rice (1999) showed that monogamous females had higher net reproductive rates than promiscuous females. In contrast, Holland (2002) saw no decrease in promiscuous female fitness relative to that of those with imposed monogamy (after 36 generations). Only Promislow *et al.* (1998) saw the increase in female fitness that we anticipate if condition-dependent sexual selection improves population mean fitness. However, it is unclear from all of these experiments whether either sexual selection or sexual conflict was actually occurring. Only the opportunity for sexual selection was manipulated. If, as demonstrated in our model, preferred traits evolve condition dependence, then the positive effects on fitness we see here will oppose those negative effects of conflict. The prevalence of condition dependence (Rowe and Houle, 1996; Jennions *et al.*, 2001) suggests that the magnitude of proposed negative effects caused by sexual conflict may currently be overestimated. More generally, positive effects on fitness of sexual selection will oppose the negative fitness effects of costly displays and preferences. More work needs to be done to understand the relative importance of positive and negative effects of sexual selection on population mean fitness (Blows, 2002; Cordero and Eberhard, 2003; Kokko *et al.*, 2003). Kokko *et al.* (2003) begin this process by explicitly modelling the effects of direct and indirect costs (through offspring) of female mate preferences on evolutionarily stable preference intensity.

One of the criticisms of the good genes process is that any potential genetic benefits accrued to females must pass through an imperfect covariance between male trait and condition (Kirkpatrick, 1996; Kirkpatrick and Barton, 1997). The same criticism applies to the effects of sexual selection acting on non-mating fitness traits that are the focus here. However, we believe that this effect is still potentially strong for two reasons. First, the

positive phenotypic covariance between condition and display traits is often strong (Jennions *et al.*, 2001). Second, sexual selection is often strong relative to natural selection (Hoekstra *et al.*, 2001; Kingsolver *et al.*, 2001). In our particular formulation, the covariance between display and condition was moderate (Fig. 1c), yet the rate of evolution of condition under condition-dependent sexual selection was three times that when under natural selection alone.

In contrast with our results, Kirkpatrick (1996) found increasing condition dependence of male display by 50% actually led to a small (about 1%) decrease in population mean fitness. As he pointed out, this was due to female preference being drawn away from its natural selection optimum, lowering mean fitness. In his model, male display responded to sexual selection by the evolution of genes directly affecting the trait rather than a change in genes related to condition (Kirkpatrick, 1996, p. 2132). Most of the response in male display in our model occurs through changes in genes related to condition, while genes coding for male display directly are quickly fixed by strong sexual selection (i.e. the lek paradox). Natural and sexual selection combined caused enough genetic load to be purged from condition-related trait loci to increase mean fitness in spite of fitness losses due to costly female preferences. Whether condition-dependent ('good genes') sexual selection increases or decreases population mean fitness appears to depend on the relative importance of load purging and female preference costs, an unresolved empirical question.

Many of the parameters used in previous work, such as that of Iwasa and Pomiankowski (1994), cannot be related to measurable features of real organisms (Houle and Kondrashov, 2001). Our explicit genetic approach avoids this difficulty, but makes it difficult to compare our model directly with traditional quantitative genetic models. The results of Iwasa and Pomiankowski are completely driven by mutation pressure, so their assumption of fixed mutation pressure is particularly suspect. Our genetic model allows mutation pressure to develop naturally; as the display trait becomes more extreme, there are more mutational steps that reduce display than increase display. This means that mutation pressure actually increases as the average display becomes more intense. The actual mutation rate we use is higher than would be expected in nature, but we expect that the qualitative result that genic capture allows an increased rate and extent of evolution for display and condition would not change with realistic mutation rates and realistic numbers of genes for display and condition (unknowns).

Iwasa and Pomiankowski (1994) also consider how multiple display traits evolve simultaneously. In their model, traits that are forbidden to evolve condition dependence ('Fisher' traits) are competed against traits that can evolve condition dependence (handicap traits), and they show that either the two traits can co-exist or the Fisher trait can exclude the handicap trait. Paradoxically, when two handicap traits are evolving, the one that is least sensitive to condition will have the least effect on female preferences and be smaller. But, Iwasa and Pomiankowski show that a trait that is not sensitive at all to condition will exclude one that is. So, there is a qualitative difference between these types of traits. It is unlikely that any trait is incapable of evolving condition dependence; rather, all traits are probably capable of responding in varying degrees to selection on condition dependence. This means that the competition of a Fisher trait against a handicap trait is artificial.

Previous discussions of the capture of variance from condition into display have focused on the role of this process in the maintenance of genetic variance in preferred traits, a requirement for the preference to persist (Rowe and Houle, 1996). Here, we have pointed to

the other side of the covariance coin: the positive covariance between the fitness of mating and non-mating traits, described by condition dependence, can allow sexual selection to accelerate the rate of change of all those covarying traits. Put another way, if sexual selection becomes condition-dependent, on one side of the coin, mutations at an increased number of loci can contribute to the evolution and maintenance of preference. On the other side of the coin, preference itself purges these mutations, potentially improving population mean fitness. Any evolutionary process that hinges upon the rate of adaptation to local conditions can be accelerated by condition-dependent sexual selection. These processes include niche expansion, adaptation to novel environments and speciation. In each case, accelerated purging of genetic load caused by the covariance between display and condition can lead to more rapid and extensive divergence in the relevant ecological traits. So, for example, when speciation depends on divergence of ecologically relevant traits that contribute to reproductive isolation (e.g. bird bill size or shape, fish gill raker length), sexual selection can accelerate this divergence. As a consequence, sexual selection can promote speciation in two ways: differences in mate choice patterns can select for pre-mating isolation (e.g. Turner and Burrows, 1995; Higashi *et al.*, 1999) and condition-dependent sexual selection can increase the rate and extent of evolution in ecologically important traits that can lead to post-mating isolation (Kawecki, 1997; Doebeli and Dieckmann, 2000).

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