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SHORT COMMUNICATION

The red queen coupled with directional selection favours the evolution of sex

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Abstract

Why sexual reproduction has evolved to be such a widespread mode of reproduction remains a major question in evolutionary biology. Although previous studies have shown that increased sex and recombination can evolve in the presence of host–parasite interactions (the 'Red Queen hypothesis' for sex), many of these studies have assumed that multiple loci mediate infection vs. resistance. Data suggest, however, that a major locus is typically involved in antigen presentation and recognition. Here, we explore a model where only one locus mediates host–parasite interactions, but a second locus is subject to directional selection. Even though the effects of these genes on fitness are independent, we show that increased rates of sex and recombination are favoured at a modifier gene that alters the rate of genetic mixing. This result occurs because of selective interference in finite populations (the 'Hill–Robertson effect'), which also favours sex. These results suggest that the Red Queen hypothesis may help to explain the evolution of sex by contributing a form of persistent selection, which interferes with directional selection at other loci and thereby favours sex and recombination.

Introduction

Sexual reproduction is a costly process, and yet most organisms reproduce sexually, at least occasionally (Bell, 1982). Many previous models aimed at explaining the ubiquity of sex have focused on one form of selection that might favour sex, including host–parasite interactions (Hamilton, 1980; Peters & Lively, 1999; Otto & Nuismer, 2004; Gandon & Otto, 2007; Salathé *et al.*, 2008), epistatic selection acting against deleterious alleles (Feldman *et al.*, 1980; Kondrashov, 1984; Charlesworth, 1990; Barton, 1995), epistatic selection favouring beneficial alleles (Charlesworth, 1993; Barton, 1995), selection to bring together beneficial alleles in finite populations (Otto & Barton, 1997, 2001; Barton & Otto, 2005; Roze & Barton, 2006), or selection to eliminate deleterious alleles from finite populations (Keightley & Otto, 2006). Nevertheless, taking a pluralist approach that incorporates multiple forms of selection may allow for the evolutionary maintenance of sex under conditions that would not be permissive otherwise, if each selective force were considered separately (West *et al.*, 1999). One of the classic examples of the success of a pluralist approach explored host–parasite interactions within a genome subject to deleterious mutations, finding that the accumulation of deleterious mutations in asexual lineages through Muller's ratchet is more severe when host–parasite interactions simultaneously occur (Howard & Lively, 1994). Similarly, combining deleterious and beneficial mutations in finite populations increases the selective interference among loci (the Hill–Robertson effect; Hill & Robertson, 1966), generating stronger selection for sex and recombination than either type of mutation alone (Peck, 1993; Hartfield *et al.*, 2010), although the net effect is generally less than the sum would predict (Hartfield *et al.*, 2010). In this article, we combine directional selection for advantageous alleles with host–parasite interactions to explore whether sex and recombination can be favoured by their interaction.

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If beneficial alleles appear infrequently, they may rarely co-occur with beneficial alleles at other loci; with only one locus segregating for a beneficial allele, sex and recombination become irrelevant to the dynamics of selection in a haploid organism. Similarly, the genetic basis of interactions between a host and a parasite may often be mediated by a major locus determining resistance or infection; again, the Red Queen cannot favour the evolution of sex and recombination with only a single haploid locus underlying the genetic interaction. Although neither form of selection would favour sex and recombination on its own under these conditions, the combination may. Here, we explore whether an advantage to sex and recombination arises from uncoupling beneficial alleles from alleles involved in antagonistic Red Queen interactions between a host and a parasite, when there is only a single haploid locus underlying each form of selection.

In finite populations, recombination can bring together beneficial alleles harboured by different individuals, providing an evolutionary advantage to sex and recombination known as the 'Fisher–Muller hypothesis' (Morgan, 1913; Fisher, 1930; Muller, 1932). Analytical models have subsequently shown that modifiers that increase the rate of sex and recombination are, on average, indirectly selected because they increase the chance that beneficial alleles fix within a population (Otto & Barton, 1997) and because they reduce the negative linkage disequilibrium that arises when selection occurs among a finite sample of individuals (Barton & Otto, 2005; Roze & Barton, 2006). That is, sex and recombination have been shown to increase in frequency within a population because they reduce the selective interference between a focal beneficial allele and whatever low fitness alleles might happen to lie in its genetic background.

Although these finite-population models have focused on the benefits of recombination when the genetic background is subject to directional selection (selecting for or against mutants), a similar advantage should arise when neighbouring loci are cycling over time due to antagonistic interactions between species, such as between hosts and parasites (May & Anderson, 1983). In fact, if these cycles occur over very long periods of time, previous mathematical results based on a QLE approximation ('quasi-linkage equilibrium', a technique that assumes that genetic associations equilibrate rapidly relative to allele frequency change) would continue to apply (e.g. Barton & Otto, 2005). Here, we explore moderately strong selection between hosts and parasites, where we expect faster cycles that invalidate the QLE approximation.

Two evolutionary problems arise when directional selection and host–parasite selection act upon neighbouring loci. First, beneficial alleles may fail to fix unless they spread by recombination to all genetic backgrounds at the host–parasite locus. Specifically, a beneficial allele can be lost if it arises on a chromosome carrying the currently disadvantageous host–parasite allele; even if it arises with the currently advantageous host–parasite allele, the beneficial allele can be lost or its spread slowed once the host–parasite cycle changes direction. Second, if the beneficial allele does succeed in fixing, it might drag along with it only a subset of alleles at the host–parasite locus, reducing the ability of the focal species to keep up in the Red Queen race. These possibilities were emphasized by Peck (1993), who used a model of negative frequency-dependent selection within a single species to mimic Red Queen dynamics between species, finding that mean fitness was substantially lower in the absence of sex. Here, we determine whether such selective interference between loci subject to host–parasite selection and directional selection favours the evolution of increased rates of sex and recombination, using an explicit two-species model with genetically variable recombination rates.

Methods

We simulated the evolution of sex and recombination in a haploid host species, involving three loci in the host and one locus in an interacting haploid parasite species, with two possible alleles at each locus. The host loci consisted of a host–parasite interaction locus (**H** with alleles *H/h*), a directionally selected locus (**B** with alleles *B/b*) and a modifier of recombination (**M** with alleles *M/m*). In the parasites, we tracked a single locus (**P** with alleles *P/p*), which interacts with the host **H** locus according to a matching-alleles model (which is equivalent to the inverse-matching-alleles model with only a single haploid locus underlying host–parasite interactions; Otto & Michalakis, 1998). Host genotypes were assigned a fitness based on the frequency at which they encounter each type of parasite within a generation. Matching between a host and a parasite (*P* with *H* or *p* with *h*) caused a loss in fitness of α_h in the host and a gain in fitness of α_p in the parasite. Specifically, the average fitness for *H*- and *h*-bearing hosts was $\omega_H = 1 - \alpha_h(x_P)$ and $\omega_h = 1 - \alpha_h(x_p)$, respectively, where x_P and x_p represent the frequency of the *P* and *p* alleles in parasites. At the **B** locus, the fitness of individuals carrying the novel *B* allele was higher by an amount β relative to individuals carrying *b*. Epistasis was assumed absent on a multiplicative scale, so that the overall fitness of *HB* individuals, for example, was $(1 + \beta)(1 - \alpha_h(x_P))$.

Recombination among the host loci was genetically variable and controlled by the modifier locus **M**. Specifically, the recombination rate between **H** and **B** depended on the modifier genotype during a transient diploid phase in the hosts (with diploids formed from randomly uniting haploid gametes) and was set to r_{mm}, r_{Mm} or r_{MM}. The recombination rate between the **M** and **B** loci was set to $R = 0.01$ without cross-over interference, but it should be emphasized that recombination between these two loci is only relevant in *M/m* heterozygotes (as measured by *R*) and that there could be genetic variation

in the rate of recombination between **M** and **B** without affecting the dynamics. It should also be emphasized that, in haploid models, a modifier that alters the frequency of sex is mathematically equivalent to a modifier that alters the rate of recombination, but where r_i and R are interpreted as the probability of reproducing sexually times the rate of recombination and where the amount of cross-over interference is properly adjusted (e.g. see Gandon & Otto, 2007). Because we track only one locus in a haploid population of parasites, the parasite may be sexual, with a diploid phase outside of the host, or asexual without affecting the dynamics.

Individuals went through a lifecycle involving a census, selection affecting the fertility of each individual, mutation at the host **H** and parasite **P** loci (at rate 10^{-3}, both forwards and backwards) and reproduction, followed by multinomial sampling of N adult hosts that would make up the next generation. The simulations were written in R by E.E.H. (R Development Core Team 2009) and are available at dryad; a subset of the results were checked against an independently written program by S.P.O. (all results were consistent, not shown). The host population size was N = 10 000 individuals, except where noted. In the main text, we assumed that the parasite population was so large that random genetic drift could be ignored; this assumption is reasonable when considering parasites that can infect multiple host species or that can reproduce outside of a host, so that the effective population size of parasites is much larger than that of the focal host species. Similar results were obtained in simulations where the parasite population size was identical to the host population size, with each host exposed to one and only one parasite (Fig. S1).

We initialized the simulations by sampling allele frequencies p and h randomly between 0 and 1 and allowing the host–parasite dynamics at the **H** locus to proceed for approximately ten host–parasite cycles, at which point the modifier allele that causes a higher rate of recombination, M, was assumed to be segregating at a frequency of 0.5 (i.e. M alleles were randomly assigned to half of the hosts). This population configuration was then saved (as a 'burn-in' of the simulations). For each burn-in, 10 000 replicate introductions of the B allele were performed. In each case, a B allele was placed on a randomly chosen genetic background and followed until it was lost or fixed within the population, at which point the final frequency of the modifier was recorded. The final modifier frequency was averaged across replicates for each burn-in, and this average was treated as a single data point. This process was repeated using 500 burn-in simulations.

Results and discussion

On average, recombination rates rose over the course of the simulations (Fig. 1), especially for the more strongly selected beneficial alleles (β was varied from 0.01, 0.025, 0.05, 0.1, 0.2, 0.5, 0.9). Sign tests indicated that, in a large population of 10 000 individuals, the modifier allele that increased recombination rose significantly in frequency in 42 of the 63 parameter sets investigated (marked by symbols in Fig. 1 using an uncorrected α-level of 0.05; one-sample t-test) and never declined significantly. Similar results were observed in populations consisting of 1000 and of 5000 individuals (in both cases, significant increases were observed in 16 of the 21 parameter sets; with N = 1000, a significant decline was also observed at β = 0.025, but the decline

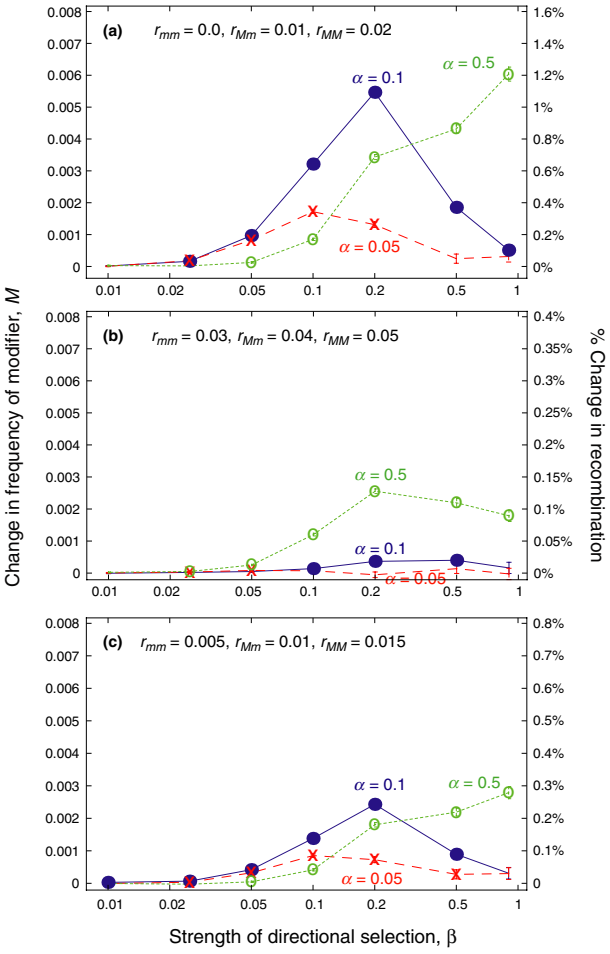

Fig. 1 Change in the frequency of the modifier allele that increases recombination, M, at the point when the favoured allele, B, becomes fixed or lost from the population (± one standard error across burn-ins). The recombination rate between **M** and **B** loci was set to 0.01 and that between **H** and **B** depended on the modifier genotype, with $\{r_{mm}, r_{Mm}, r_{MM}\}$ set to (a) {0.0, 0.01, 0.02}, (b) {0.03, 0.04, 0.05} and (c) {0.005, 0.01, 0.015}. Number of burn-ins = 500; replicates per burn-in = 10 000; population size = 10 000; initial modifier frequency = 0.5. Curves describe all of the data; cases where the change in modifier frequency was significantly different from zero are also indicated by points: red × 's with $\alpha_h = \alpha_p = 0.05$, blue closed circles with $\alpha_h = \alpha_p = 0.1$ and green open circles with $\alpha_h = \alpha_p = 0.5$.

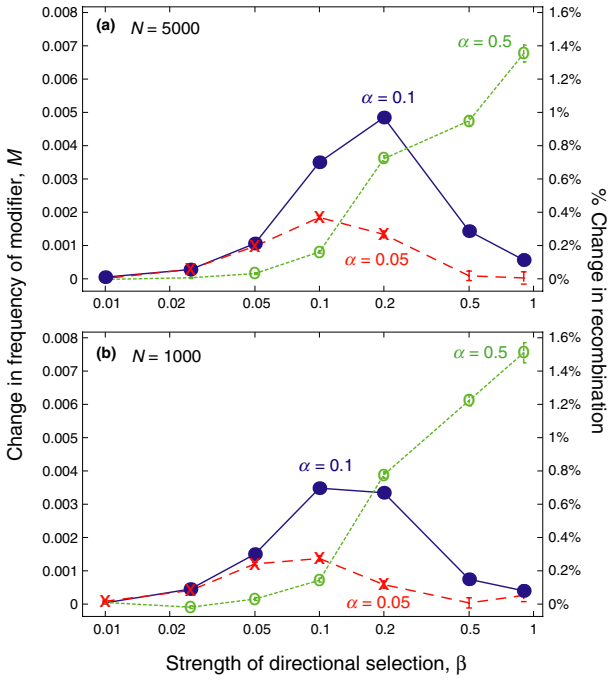

Fig. 2 Change in the frequency of the modifier allele with smaller population sizes. The parameters are identical to those used in Fig. 1c, where the population size was 10 000, except with (a) 5000 individuals or (b) 1000 individuals. Again, the symbols indicate significant changes in the frequency of the modifier increasing recombination.

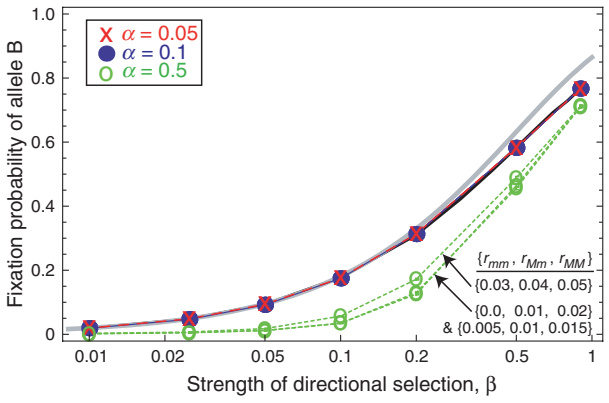

Fig. 3 Probability of fixation of the beneficial allele, *B*. The recombination rate between **M** and **B** was set to 0.01 and that between **H** and **B** depended on the modifier genotype, with either $\{r_{mm}, r_{Mm}, r_{MM}\} = \{0.0, 0.01, 0.02\}$, $\{0.03, 0.04, 0.05\}$ or $\{0.005, 0.01, 0.01\}$. Results were similar for the three sets of recombination rates explored, so they are superimposed in this figure and distinguished only where they differ visually. For comparison, the upper grey curve illustrates the fixation probability based on Kimura's diffusion approximation in the absence of host–parasite interactions; the black curve illustrates the fixation probability based on one-locus simulations (**B** locus only), which provides a more accurate estimate when directional selection is strong (black curve is virtually superimposed on the results obtained with weaker host–parasite selection: red × 's with $\alpha_h = \alpha_p = 0.05$ and blue closed circles with $\alpha_h = \alpha_p = 0.1$).

was modest and was most likely a type I error; Fig. 2). The evolutionary change at the modifier locus was generally weaker when selection on the hosts and parasites was weak (red curves with $\alpha_h = \alpha_p = 0.05$). The pattern was not, however, monotonic with respect to host–parasite selection. Rather, the modifier frequency changed most when the strength of selection at the two loci was more equally matched (i.e. the blue curves with $\alpha_h = \alpha_p = 0.1$ peaked at smaller values of β than the green curves with $\alpha_h = \alpha_p = 0.5$). This result is consistent with previous work that found faster evolution of recombination when linked beneficial alleles experienced roughly similar selection pressures (see Fig. 3c in Otto & Barton, 1997). Although the percentage increase in recombination appears small, these figures average over many cases where the favourable *B* allele is lost soon after it is introduced. If we condition our results on the fixation of the *B* allele, the percentage rise in recombination is much more substantial (Fig. S2), especially for intermediate values of β. This makes sense because if the *B* allele is lost soon after it arises, Hill–Robertson effects disappear. The per cent increase in recombination observed in Fig. S2 is comparable to simulation results obtained by Otto & Barton (2001), who considered directional selection acting on two loci in the presence of a modifier of recombination (see their Figs 2a and 3a; these figures are most directly comparable to our Fig. S2 because Otto & Barton introduce the favourable alleles at a higher frequency than we do, and so the alleles are more likely to fix).

The extent to which increased recombination evolved was typically higher when linkage between the beneficial mutation and the host–parasite locus was tighter, all else being equal. This is illustrated by a comparison of Fig. 1a,b. In both panels, the strength of the modifier was the same ($r_{MM} - r_{mm} = 0.02$), but the change in modifier frequency was generally much lower when the average rate of recombination was raised (from $\bar{r} = 0.01$ in panel a to $\bar{r} = 0.04$ in panel b). The only case where substantial changes in recombination continued to be observed was when selection was very strong ($\alpha_h = \alpha_p = 0.5$, green curves), in which case, substantial genetic associations could continue to accumulate despite the more frequent recombination. As expected, modifiers having a weaker effect on the rate of recombination changed by a proportionately smaller amount. Relative to Fig. 1a, the average rate of recombination was the same in Fig. 1c ($\bar{r} = 0.01$), but the strength of the modifier was halved (from $r_{MM} - r_{mm}$ of 0.02 to 0.01). Consequently, the evolutionary change in recombination rate was approximately halved.

The extent to which selective interference reduces the fixation probability of a beneficial allele depends on

the advantage of this allele relative to the strength of the host–parasite interactions. As illustrated in Fig. 3, beneficial alleles are much less likely to fix when host–parasite interactions are strong, especially when the average recombination rate is low. In these cases, the beneficial allele can initially establish and yet still be lost from the population if associated with an allele at the interaction locus that allows infection by the current or near future population of parasites (Peck, 1993). Proportionately, however, this interference has less of an effect on the fixation probability of strongly beneficial alleles (right-hand side of Fig. 3).

The results of this study contrast with recent simulations using only loci involved in host–parasite interactions (Kouyos *et al.*, 2007, 2009). When the selected loci interacted multiplicatively, sex and recombination were both strongly selected against. This occurred because the loci tended to cycle in lock step, with strong linkage disequilibrium of constant sign (e.g. involving cycles between *HB* and *hb*), which strongly selected against sex and recombination regardless of population size. Conversely, when multiple genes mediated the host–parasite interaction in a strongly epistatic way (e.g. matching must occur at all loci for infection to proceed), sex and recombination were favoured as long as selection was sufficiently strong (see also Otto & Nuismer, 2004), but then population size played little role in the outcome. It is important to stress, however, that these previous models have assumed that multiple loci contribute equally to resistance (but see Agrawal & Otto, 2006), whereas many parasites have evolved to express a single-antigen locus (Donelson, 1995; Barbour & Restrepo, 2000; Kusch & Schmidt, 2001) and many resistance traits in plants are attributed to single Mendelian genes (Staskawicz *et al.*, 1995). The results of our work demonstrate that Red Queen dynamics can still favour increased recombination, even when only one haploid locus mediates the interaction between a host and a parasite, as long as directional selection is present at neighbouring loci.

Our results demonstrate that increased sex and recombination evolve in the presence of host–parasite interactions and directional selection because genetic mixing uncouples the fate of favourable alleles from the specific host–parasite allele with which it arises. Although the change in frequency of the modifier was modest in all of the simulations, this was partially due to the fact that the beneficial allele was often lost soon after it arose, causing the modifier to become neutral. Considering only cases where the *B* allele eventually fixed within the population, much larger changes in recombination were observed (Fig. S2). A major advantage of this pluralistic explanation for sex and recombination is that genes mediating host–parasite interactions tend to exhibit high levels of polymorphism over prolonged periods of time (such as the highly polymorphic MHC loci in animals, e.g. Messaoudi *et al.*, 2002; and resistance *R* genes in plants, e.g. Mauricio *et al.*, 2003), and so would induce Hill–Robertson interference on surrounding sites under selection. This avoids a limitation of the Fisher–Muller hypothesis, which requires beneficial alleles to appear close enough together in time and in linkage to affect each other's spread, as noted by Peck (1993). Thus, interactions between directional selection and host–parasite interactions would continually act on genetic variation for recombination and sex, helping to maintain genetic mixing within a population.

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Supporting information

Additional Supporting Information may be found in the online version of this article:

Figure S1 Change in the frequency of the modifier allele that increases recombination, *M*, when coevolving with a finite population of parasites.

Figure S2 Change in the frequency of the modifier allele that increases recombination, *M*, conditional on the fixation of the *B* allele.

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