Speciation as a positive feedback loop between postzygotic and prezygotic barriers to gene flow

Maria R. Servedio[1,2*] and Glenn-Peter Sætre[3,4]

[1]*Ecology, Behavior, and Evolution (EBE), Division of Biology 0116, University of California, San Diego, La Jolla, CA 92092-0116, USA*
[2]*Department of Biology, CB no. 3280 Coker Hall, The University of North Carolina at Chapel Hill, Chapel Hill, NC 27599-3280, USA*
[3]*Department of Evolutionary Biology, Evolutionary Biology Centre, Uppsala University, Norbyvägen 18d, SE-752 36 Uppsala, Sweden*
[4]*Department of Biology, Division of Zoology, University of Oslo, PO Box 1050, Blindern, N-0316 Oslo, Norway*

Speciation is intimately associated with the evolution of sex-and-reproduction-related traits, including those affecting hybrid incompatibility (postzygotic isolation) and species recognition (prezygotic isolation). Genes controlling such traits are not randomly distributed in the genome but are particularly abundant on the sex chromosomes. However, the evolutionary consequences of the sex linkage of genes involved in speciation have been little explored. Here, we present simulations of a continent–island diploid model that examines the effects of reduced recombination using both autosomal and sex-linked inheritance. We show first that linkage between genes affecting postzygotic and prezygotic isolation leads to a positive feedback loop in which both are strengthened. As species recognition evolves, genes causing hybrid incompatibility will hitchhike along with those improving premating isolation, leading to stronger hybrid incompatibility and thus increased pressure for further preference divergence. Second, we show that this loop effect is generally enhanced by sex linkage, because recombination is eliminated in the heterogametic sex, leading to tighter effective linkage between the two classes of genes and because natural selection is more efficient at sex-linked loci, as recessive alleles are not masked by dominance in the heterogametic sex. Accordingly, hitchhiking can be important in promoting speciation and can also lead to increased postzygotic isolation through adaptive evolution.

Keywords: Haldane's rule; gene flow; hitchhiking; sex linkage; reinforcement; sex chromosome

1. INTRODUCTION

Comparative studies indicate that the X and Z chromosomes, in male and female heterogamy, respectively (hereafter referred to as the sex chromosomes), are enriched with genes associated with sex and reproduction (e.g. Hurst & Randerson 1999; Saifi & Chandra 1999; Wang *et al.* 2001). It is well established that sex-linked genes are involved in the evolution of postzygotic isolation, particularly with respect to hybrid sterility (Coyne & Orr 1998). Incompatibilities that cause hybrid dysfunction typically involve X (Z)–autosomal or X–Y (Z–W) interactions (Turelli & Orr 2000). Mate-recognition systems consist of secondary sexual characteristics in one sex (usually males) associated with a preference for the trait(s) in the other sex (usually females). Both secondary sexual traits and mate preferences have been found to be sex linked in some cases (Reinhold 1998; Ritchie 2000; Noor *et al.* 2001*a*; Iyengar *et al.* 2002; Sætre *et al.* 2003). However, the potential evolutionary consequences of sex linkage of the genes affecting both postzygotic and prezygotic barriers to gene flow have been little explored in speciation theory.

According to the theory of reinforcement, natural selection against the production of unfit hybrids would favour alleles that make interbreeding between the parental types

less likely (Dobzhansky 1940). Both empirical studies (e.g. Noor 1995; Sætre *et al.* 1997) and theoretical models (e.g. Felsenstein 1981; Liou & Price 1994; Servedio 2000) show that reinforcement of prezygotic isolation can occur under certain conditions. One important factor that opposes reinforcement is recombination between alleles affecting mate recognition and those affecting hybrid fitness (Felsenstein 1981; Trickett & Butlin 1994; Servedio 2000). Thus, linkage, including that on the sex chromosomes, between genes affecting assortative mating and genes affecting hybrid fitness is likely to enhance the reinforcement process in hybridizing species (Noor *et al.* 2001*a*; Sætre *et al.* 2003). These ideas, however, neglect to consider whether intrinsic forms of postzygotic isolation, generally thought of as a non-adaptive consequence of divergence without gene flow, increase or are static as premating isolation is evolving during reinforcement. They also do not consider whether linkage on the sex chromosomes or linkage on autosomes would be more effective in promoting reinforcement.

Here, we develop the ideas that there may be coevolution of postzygotic and prezygotic barriers to gene flow and that this may be enhanced by the sex linkage of genes affecting mate recognition and hybrid viability. Coevolution may occur during reinforcement or sympatric speciation if genes for prezygotic isolation are linked to those controlling postzygotic isolation: as mate choice evolves, genes causing postzygotic isolation will hitchhike along (Maynard Smith & Haigh 1974; Kaplan *et al.* 1989;

Wiehe & Stephan 1993; Begun & Whitley 2000), leading to increased pressure for further premating divergence. First, we examine whether hitchhiking occurs by comparing the rates of spread of alleles causing postzygotic isolation as the linkage between loci controlling prezygotic and postzygotic isolation is tightened. Second, we examine the effects on this process of sex linkage of the genes controlling pre- and postzygotic isolation. Two factors make it likely that genes on the sex chromosomes will evolve together more rapidly than they would if they were on autosomal chromosomes. First, recombination is eliminated in the heterogametic sex, allowing enhanced coevolution of the genes controlling prezygotic and postzygotic isolation. Second, the efficiency of natural selection can be increased at sex-linked loci because recessive alleles are not masked by dominance in the heterogametic sex, leading to stronger effective selection (Charlesworth *et al.* 1987). Both of these effects may lead to increased coevolution in reinforcement as well as to the enrichment of sex-and-reproduction-related genes on the sex chromosomes.

2. THE MODEL

We explore this concept by examining the outcome of secondary contact between two populations that have partially diverged in allopatry. A continent–island model is used to consider a peripheral isolate receiving migrants from a large ancestral population. The properties of continent–island models in the context of reinforcement are fairly well understood (Servedio & Kirkpatrick 1997; Kirkpatrick & Servedio 1999; Servedio 2000).

Individuals are diploid and have four loci, a preference locus P, a locus for a sexually selected trait T and two population-specific background loci M and N. Each locus has two alleles. Continental individuals are fixed for the P_1, T_1, M_1 and N_1 alleles throughout the simulations; evolution occurs only on the island. Island individuals have diverged in allopatry at the trait and background loci, where they carry predominantly T_2, M_2 and N_2 alleles (the genotype $P_1T_2M_2N_2/P_1T_2M_2N_2$ has a starting frequency of 99%). At the start of secondary contact, island individuals carry the preference allele P_1; there is no premating isolation. After an initial equilibrium is reached at the trait and background loci, as a result of migration and selection, the novel preference allele P_2 is introduced at a low frequency (0.001) on the continent, from where it migrates to the island. Reinforcement is considered to have occurred if the P_2 allele spreads on the island, leading to some premating isolation between island individuals and continental migrants.

The life cycle consists of migration, natural selection, sexual selection (mating), recombination and offspring production. In each generation, individuals migrate from the continent to compose a proportion m of the island population.

Natural selection acts at both the trait locus and the background loci. The trait is expressed and selected on only in males. The viabilities of T_1T_1, T_1T_2 and T_2T_2 genotypes in males on the island are $1/(1 + s_T)$, 1, and $1 + s_T$, respectively. This parameterization was chosen to make the sexual-selection model behave similarly to a haploid model where T_1 has viability 1 and T_2 has viability $1 + s_T$ (Gomulkiewicz & Hastings 1990). Postzygotic

incompatibilities are produced by epistatic interactions at the background loci (Cabot *et al.* 1994; Wu & Palopoli 1994). We assume that hybrid genotypes that are further from either pure parental genotype have lower fitness (Coyne 1985; Davis *et al.* 1994; Hollocher & Wu 1996). Pure genotypes (e.g. $M_1N_1M_1N_1$) are therefore assigned a fitness of 1, genotypes with one incompatible allele (e.g. $M_1N_1M_1N_2$) have a fitness of $1 - hs_E$, where $0 < h < 1$ and $s_E > 0$, and genotypes with two pairs of alleles that are incompatible (e.g. $M_1N_2M_1N_2$ and $M_1N_1M_2N_2$) have a fitness of $1 - s_E$. Runs were performed with $h = 0.4$ and $h = 0.6$, where a single incompatibility behaves as if it is partly recessive compared with two incompatibilities, and with $h = 1.0$, where a single incompatibility behaves as if it is fully dominant.

Females that have the preference allele P_1 prefer to mate with males that have a T_1 allele, while P_2 females tend to prefer T_2 males. The exact frequencies of matings are shown in table 1; these mating frequencies for diploids were also chosen to make the sexual-selection model behave similarly to a haploid model with comparable assumptions (Gomulkiewicz & Hastings 1990). Recombination occurs before gametes are produced, at a rate of r_{ij} between loci i and j. The order of genes on the chromosome is assumed to be PTMN.

We also consider a model of sex linkage that assumes that all four loci are located on the Z chromosome, patterned after the recent findings of sexual-trait and postzygotic-isolation alleles on the Z chromosome in flycatchers (Sætre *et al.* 2003) and Z-linkage of preference alleles in the moth *Utetheisa ornatrix* (Iyengar *et al.* 2002). Males are therefore diploid while females are haploid. Fitness at the background loci in females is set for each run so that pure haploid genotypes (e.g. M_1N_1) have the same fitness as pure diploid genotypes, and recombinant haploid genotypes (e.g. M_1N_2) have the same fitness as diploid genotypes with two incompatibilities. This type of fitness pattern would result if there was dosage compensation. The fitnesses of alleles at the trait locus do not change, as the trait is expressed only in diploid males. Haploid P_1 and P_2 females have the same mating preferences as P_1P_1 and P_2P_2 females (respectively) in the diploid simulations (see table 1). Recombination occurs only in males. Although all of the loci are assumed to be on the same chromosome, we allow a variety of recombination rates as high as 0.5 to examine the effects of tightening linkage.

The models were analysed by iterations of the recursion equations using programs written in C. The initial equilibrium (before the introduction of P_2) was obtained by running each simulation until the relative changes per generation in the frequencies at the T, M and N loci were less than 10^{-12}. After the introduction of P_2 the simulations were considered to be at equilibrium when the change per generation at the P locus was less than 10^{-10}.

We measured the final equilibrium value of the introduced preference allele P_2. This provides an indication of the amount of premating isolation present at equilibrium. We also measured the increase in the frequency of the population-specific background island genotype (measured on haploid chromosomes), ΔM_2N_2, as the difference in the frequencies of the M_2N_2 genotype before and after the introduction of P_2. Our results were qualitatively identical when the relative increase in M_2N_2 was

Table 1. Mating frequencies between male and female genotypes.

(The frequency of each type of mating is obtained by multiplying the frequencies of the male and female genotypes after natural selection by the corresponding entry in the table. The parameters α_1 and α_2 are defined as greater than 0. The factors z_0, z_1 and z_2 are defined such that the frequencies of all matings involving a given female genotype sum to that genotype's frequency among mated females. (Adapted from Kirkpatrick & Servedio 1999).)

female genotype	male genotype		
	T_1T_1	T_1T_2	T_2T_2
P_1P_1	$\dfrac{1+\alpha_1}{z_0}$	$\dfrac{1}{z_0}$	$\dfrac{1}{z_0(1+\alpha_1)}$
P_1P_2	$\dfrac{1}{\dfrac{z_1}{2}\left(\dfrac{1}{1+\alpha_1}+(1+\alpha_2)\right)}$	$\dfrac{1}{z_1}$	$\dfrac{1}{\dfrac{z_1}{2}\left((1+\alpha_1)+\dfrac{1}{1+\alpha_2}\right)}$
P_2P_2	$\dfrac{1}{z_2(1+\alpha_2)}$	$\dfrac{1}{z_2}$	$\dfrac{1+\alpha_2}{z_2}$

considered instead, by dividing ΔM_2N_2 by the value of M_2N_2 before the introduction of P_2.

3. RESULTS

Tighter linkage, simulated by decreasing recombination rates, generally leads to an increase in the amount of premating isolation at equilibrium. This is true both when all pairs of contiguous loci have the same recombination rates and when one of the incompatibility loci recombines freely. It holds for both autosomal and sex-linked inheritance when incompatibilities act as though they are partially recessive (figure 1*a*) and fully dominant (figure 1*b*). With sex-linked inheritance, free recombination at the incompatibility loci always increased the amount of reinforcement. This can be a large effect (e.g. figure 1). This effect was smaller, but still present, when $h = 0.4$ (not shown), and when selection against hybrids was very weak ($s_E = 0.01$, $h = 0.6$ and 1.0, not shown). When inheritance is autosomal, free recombination of one of the incompatibility loci has a variable effect.

One of the primary findings of this study is that the species-specific background genotype (M_2N_2), which produces incompatibilities in hybrids, also generally displays a greater increase in frequency with tighter linkage of prezygotic and postzygotic isolation (with partially recessive incompatibilities, figure 2*a*; with fully dominant incompatibilities, figure 2*b*). The patterns of spread of the M_2N_2 genotype often closely mimic those found for the frequency of P_2 at equilibrium. This indicates that the process of reinforcement has caused the incipient species to differ more from one another, strengthening the possibility of postzygotic incompatibilities in a cross-population mating.

Two additional lines of evidence (besides the pattern shown in figure 2) suggest that this process can be attributed to hitchhiking, resulting from linkage, of the postzygotic-isolation alleles with the premating-isolation alleles P_2 and T_2, which are both increasing in frequency. This evidence comes from examining the frequencies at the end of the simulations of the N_2 and M_2 alleles. The order of genes on the chromosome is PTMN, so, first, we would expect the frequency of M_2 to be greater than that of N_2

if the postzygotic-incompatibility alleles were spreading by hitchhiking with the sexual-selection alleles. This effect can be seen in the increase in the difference between the frequencies of the M_2 and N_2 alleles in a characteristic set of runs as r_{MN} increases when r_{MT} is less than 0.5 in table 2. Second, when there is free recombination between the sexual-selection alleles and the postzygotic-incompatibility alleles ($r_{MT} = 0.5$), we would expect no difference between the frequencies of the M_2 and N_2 alleles; this effect can also be seen in table 2.

The magnitude of the change in M_2N_2 is much greater (as high as 0.05 with sex linkage) with very weak selection against hybrids ($s_E = 0.01$, not shown), most probably owing to the fact that there is much more variation maintained at the M and N loci under these conditions. With very weak selection the amount of change in the M_2N_2 genotype sometimes decreases slightly with more reinforcement in autosomal runs, although it shows a comparable pattern to strong selection when there is sex linkage. Under these conditions, the increase in mating preference is probably driven much more through its associations with the male trait allele than through its associations with alleles for postzygotic isolation (M. R. Servedio, unpublished data).

The second novel finding is that sex linkage often dramatically increases the spread of P_2 and the increase in the frequency of M_2N_2. This occurs both with incompatibilities acting as partly recessive and as fully dominant (figures 1 and 2). With strong selection (e.g. figures 1 and 2) this effect was more prominent with higher recombination rates (above 0.01). With weak selection (not shown) the effect is instead very strong when recombination rates are low (0.1 or less) and it is not seen with high recombination (above 0.2, there is instead a very weak effect in the opposite direction). Generally speaking, sex linkage therefore promotes the evolution of both premating and postmating isolation in this model.

4. DISCUSSION

Taken together, the further spread with tighter linkage of the island preference and of the island genotype at the incompatibility loci constitutes evidence for a positive
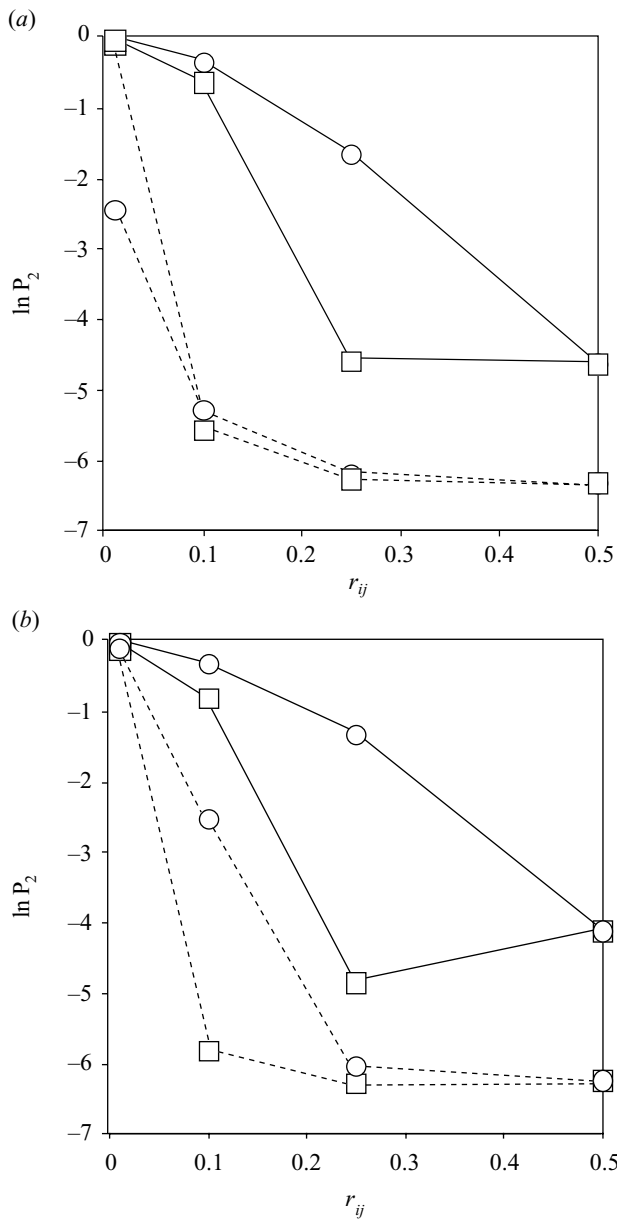
Figure 1. The premating-isolation allele P_2 spreads further at equilibrium as recombination rates decrease. Premating isolation evolves further with sex-linked inheritance (solid lines) than with autosomal inheritance (dashed lines). Squares indicate similar recombination rates between all loci ($r_{PT} = r_{MT} = r_{MN}$). Circles indicate free recombination between the background loci that produce incompatibility in hybrids ($r_{PT} = r_{MT}$ but r_{MN} is fixed at 0.5). Parameter values are $s_T = 0.2$, $s_E = 0.5$, $m = 0.001$ and $\alpha_1 = \alpha_2 = 0.1$. (*a*) Incompatibilities act as though they are partially recessive ($h = 0.6$). (*b*) Incompatibilities act as though they are fully dominant ($h = 1.0$).

Figure 2. The increase in the species-specific genotype at the incompatibility loci, M_2N_2, is greater at equilibrium when recombination rates are lower. Symbols and parameters are as in figure 1. (*a*) Incompatibilities act as though they are partially recessive ($h = 0.6$). (*b*) Incompatibilities act as though they are fully dominant ($h = 1.0$).

feedback loop of prezygotic and postzygotic isolation. The first finding, that linkage of the genes controlling assortative mating and hybrid incompatibility promotes the reinforcement process, is in agreement with previous models (e.g. Felsenstein 1981; Servedio 2000). Because hybrids are partially viable and are fertile, recombination would tend to break down the association between alleles affecting hybrid fitness and alleles promoting prezygotic isolation. Thus, linkage acts to oppose the tendency of
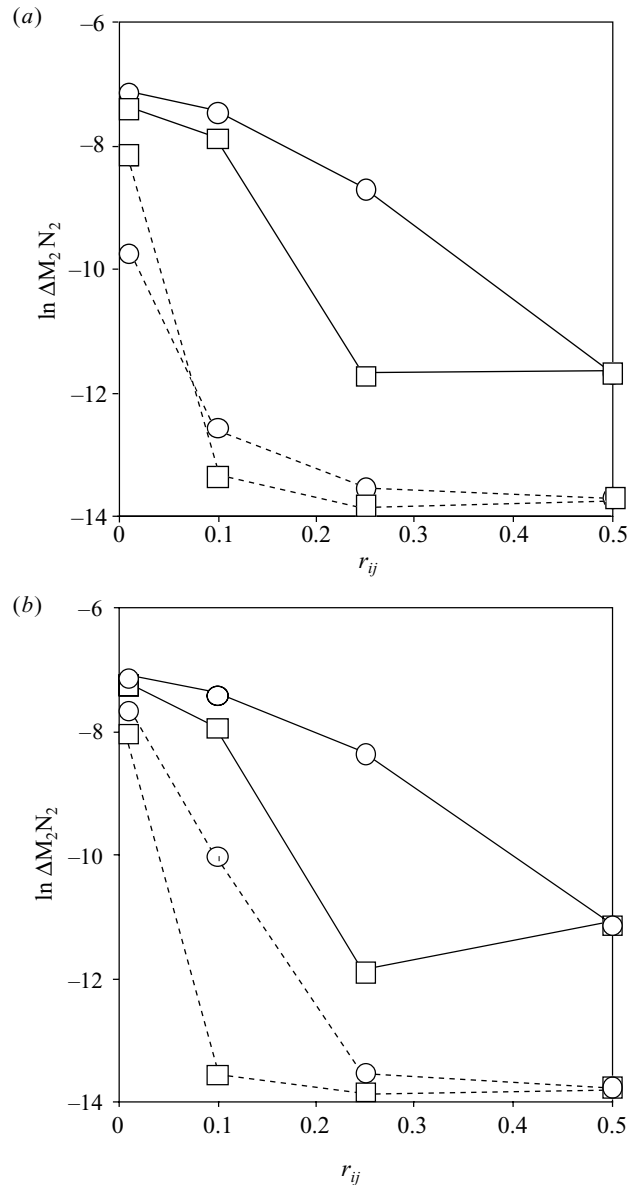
isolation alleles to become randomly combined with the alleles that reduce hybrid fitness.

Our second finding, which completes the positive feedback loop, is that the species-specific background genotype (M_2N_2), which produces incompatibilities in hybrids, also generally displays a greater increase in frequency with tighter linkage. Increasing the frequency of M_2N_2 can be interpreted as strengthening the species identity of the island population. In other words, a mating between an individual on the island and a newly arrived continental migrant will have a greater likelihood of producing offspring with incompatibilities. Increasing M_2N_2 therefore increases postzygotic isolation. The fact that there is a greater increase in the frequency of M_2N_2 with tighter linkage suggests that postzygotic isolation increases at least in part because of genetic hitchhiking, through the linkage

Table 2. Final frequencies of N_2 and M_2.

(The recombination rate between the loci causing hybrid incompatibilities, N and M, was varied for each of three different recombination rates between the M and T and the P and T loci (the order of loci on a chromosome is PTMN). The frequencies of the M_2 and N_2 alleles at the end of each simulation are recorded.)

r_{MT} (= r_{PT})	r_{MN}	frequency of M_2	frequency of N_2
0.1	0.5	0.90728	0.88337
0.1	0.1	0.91234	0.90137
0.1	0.001	0.92173	0.92159
0.25	0.5	0.88348	0.87574
0.25	0.25	0.88532	0.88037
0.25	0.001	0.90143	0.90141
0.5	0.5	0.87161	0.87161
0.5	0.001	0.89119	0.89119

of these loci with those involved in premating isolation. When this hitchhiking occurs, the increased postzygotic isolation can then drive further evolution of premating isolation through reinforcement. This effect constitutes a positive feedback loop of prezygotic and postzygotic isolation that increases with tighter linkage.

The increase in postzygotic isolation is itself not a necessary consequence of reinforcement, which implies that alleles for prezygotic isolation must become associated with those for postzygotic isolation, but does not imply that the latter must increase in frequency. To our knowledge, this effect has not been recognized in previous theoretical treatments of reinforcement, although it may have been occurring. Intrinsic postzygotic incompatibilities have been generally thought to develop as a by-product of selection and/or drift when populations are isolated from one another (e.g. Hayashi & Kawata 2002; Porter & Johnson 2002). Explicit theoretical observations of the build-up of Dobzhansky–Muller incompatibilities with gene flow are sparse in the literature (but see Gavrilets *et al.* 1998; Gavrilets 1999; Porter & Johnson 2002). We found that the alleles causing these incompatibilities increased in frequency, despite gene flow, under all sets of assumptions regarding strength and pattern of linkage, dominance pattern and inheritance (sex linked or autosomal). The effect can occur even without hitchhiking (e.g. when $r_{ij} = 0.5$). This can be explained simply: gene flow is effectively reduced as premating isolation strengthens, so the migration–selection balance at adaptive allele combinations would change accordingly. The hitchhiking demonstrated in this study would enhance this evolution of postmating isolation and the feedback loop that results.

Although the positive feedback loop presented here occurs because an increase in frequency of a single interacting pair of alleles causes postzygotic isolation, we suggest that a similar effect may occur by the substitution, possibly successive, of multiple sets of alleles causing postzygotic isolation. This possibility, which would be the most interesting biologically, warrants further investigation. With the evolution of a pair of alleles, the loop effect in this study terminates in equilibrium frequencies of both the postmating and the premating isolation alleles. With multiple sets of alleles it is possible that the evolution of both premating and postmating isolation can be driven even further. It is also possible that, as genetic incompatibilities build up at some loci, the level of gene flow could be reduced at closely linked loci, likewise potentially

easing the conditions for further genetic divergence (see Barton & Bengtsson 1986). Because this type of gene-flow reduction is very porous (Barton & Bengtsson 1986), however, the development of further premating isolation, such as by the feedback loop presented in this study, would probably be a more effective way to reduce gene flow.

The further spread of both prezygotic and postzygotic isolation is more pronounced when one of the loci causing hybrid incompatibility recombines freely. This finding is consistent with the effects of reducing recombination rates between incompatibility loci in previous models of reinforcement in haploids (Felsenstein 1981; Servedio 2000), but had not been found in diploids (Felsenstein 1981). This probably occurs because increased recombination results in a greater fraction of F_2 or backcross offspring being unfit, which increases the selection for premating divergence. We found the effects of free recombination between these alleles to be particularly large when these loci are sex linked. In nature, hybrid dysfunction, particularly sterility, generally follows Haldane's rule in that the fitness of hybrids of the heterogametic sex is most severely affected (Haldane 1922). Empirical and theoretical evidence indicates that this is caused by epistasis between partially recessive sex-linked loci and autosomal loci (Turelli & Orr 1995, 2000; Coyne & Orr 1998). In our sex-linked runs we do not have autosomes. However, the pattern of freely recombining incompatibility loci may suggest what would happen if one of the incompatibility genes were autosomal (it increases the chance of incompatible alleles co-occurring in hybrid progeny). Thus, we note that the scenario in our model that most closely mimics the fitness scheme producing Haldane's rule is the one that yields the highest increase of prezygotic and postzygotic isolation.

Sex linkage almost invariably dramatically promoted the evolution of prezygotic and postzygotic isolation in our model compared with the case when all loci were autosomal. With strong selection against hybrids, this is particularly true when there is significant recombination. In general, the rapid evolution of sex chromosomes is expected to occur if favourable mutations are partially or fully recessive, because they would not be masked by dominance in the heterogametic sex (Charlesworth *et al.* 1987). Such recessivity would be expected if incompatibilities had large effects (Kacser & Burns 1981; Hartl *et al.* 1985). A second source of rapid sex-chromosome evolution in our model may be the lack of recombination in

the heterogametic sex, which will reduce the overall chance that the genes of interest will recombine. This would occur not only because of increased linkage between the preference genes and the genes controlling incompatibilities, but also because of higher linkage between the preference genes and the trait genes, which has been shown to enhance reinforcement (Servedio 2000). We cannot compare the scenarios of partially recessive and fully dominant incompatibilities directly in our model because by changing dominance we simultaneously change the overall strength of the postzygotic incompatibilities. The greater effect of sex linkage cannot be solely an effect of recessive incompatibilities, however, as fully dominant cases also showed more evolution with sex linkage. The absence of recombination in the heterogametic sex therefore has a significant effect in promoting prezygotic and postzygotic isolation in the sex-linked runs. This effect may be a significant factor promoting speciation in taxa, such as *Drosophila*, that lack crossing over even in autosomes in the heterogametic sex. The effects of sex linkage found here would depend heavily, of course, on lower recombination rates of traits that are sex linked than of traits that are autosomal; recombination rates have been found to be very heterogeneous within and between chromosomes in general, so this assumption may not always hold (e.g. Noor *et al.* 2001*b* and citations therein).

Empirical findings in *Ficedula* flycatchers are consistent with the speciation-loop hypothesis presented here. In these species reinforcement has been shown to operate through a sympatric character displacement in male plumage traits that help species recognition (Sætre *et al.* 1997). Both hybrid sterility and male plumage traits are influenced by sex-linked genes in these birds (Sætre *et al.* 2003). These flycatchers are found in two classes of hybrid zone: in a continental cline where reinforcement operates (Sætre *et al.* 1997), and on recently colonized islands where reinforcement is much less pronounced (Sætre *et al.* 1999). Interestingly, hybrid fertility is significantly higher on the islands than in the cline (Sætre *et al.* 1999), as would be predicted from the speciation-loop hypothesis. Further empirical studies, on both this and other model systems, are needed, however, to test the hypothesis critically.

In conclusion, we demonstrate that linkage between genes controlling traits associated with mate recognition and those controlling hybrid incompatibilities can lead to a positive feedback loop in which both prezygotic and postzygotic isolation are promoted, and that these effects are further enhanced by sex-linked inheritance. These results have a number of important implications. This feedback loop may lead to the increased accumulation of both of these types of genes on the sex chromosomes, accounting in part for the finding of this pattern in empirical studies. We additionally explicitly demonstrate adaptive evolution resulting from hitchhiking, a phenomenon that has been generally considered only to reduce genetic variation around selection events (Nachman 2001). Furthermore, this study suggests a role for selection, through hitchhiking, in the evolution of postzygotic isolation that is less specialized than that postulated previously (e.g. parental investment (Coyne 1974); soft selection (Johnson & Wade 1995); kin selection (Leibowitz 1994); and chromosomal rearrangements (Navarro & Barton 2003)), and

demonstrates that postzygotic isolation may develop despite gene flow (e.g. Gavrilets *et al.* 1998; Gavrilets 1999; Porter & Johnson 2002). Finally, although we assume that the initial divergent selection for reproductive characters is caused by pre-existing postzygotic isolation, the positive feedback loop present in the model may ease the conditions for sympatric speciation. Postzygotic isolation may build up during the sympatric speciation process, accelerating and easing the conditions for speciation to proceed.

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