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The evolution of recombination in changing environments

Sarah P. Otto and Yannis Michalakis

The main difficulty with the evolution of recombination is that parents who survive to reproduce have a genotype that works under local environmental conditions; tinkering with their offsprings' genotypes by recombination risks making the situation worse, with little hope of making it better. This idea underlies the Reduction Principle: in a constant environment without mutation, only modifier alleles that reduce recombination can invade a randomly mating population at equilibrium^{1–4}. Lower recombination rates evolve whenever there are genetic associations among loci (linkage disequilibria), whether these are positive or negative^{1–4}.

Recent models of the evolution of recombination have greatly clarified the conditions under which increased recombination can evolve, especially when the population is changing over time^{3,5–10}. It

has long been thought that recombination might help a population keep up with environmental changes by producing novel allelic combinations^{11–13}. It is not true, however, that changing environments always favor increased

Recombination generates under-represented genotypes by breaking down linkage disequilibrium between genes. Recent analyses have specified the conditions under which recombination is favored. These conditions are surprisingly sensitive to the form of selection and environmental change. This quantification makes it possible to use empirical measurements of critical parameters such as the form of epistasis, the rate of mutation, and the frequency of beneficial sweeps to assess different hypotheses for the evolution of recombination.

Sarah P. Otto is at the Dept of Zoology, University of British Columbia, Vancouver, BC, Canada V6T 1Z4 (otto@zoology.ubc.ca); Yannis Michalakis is at Fonctionnement et Evolution des Systèmes Ecologiques, Université Pierre & Marie Curie Paris, CNRS URA 258, 7 quai St Bernard, 75252 Paris CEDEX 05, France (loannis.Michalakis@snv.jussieu.fr).

recombination and the new analyses specify the conditions that must hold for higher rates of recombination to evolve. Here, we highlight some of the important recent results on the evolution of recombination (the interested reader is referred to Refs 4,14–16 for further review).

Directional selection

Studies investigating directional selection are particularly useful in that they differ in only one important respect from those models in which the Reduction Principle holds: the population is not at equilibrium, as beneficial alleles rise in frequency in response to selection. Increased recombination may evolve in populations experiencing directional selection, but for the recombination rate to matter at all requires the existence of linkage disequilibria (such that certain combinations of alleles are found together more often than

expected). There are two ways in which disequilibria may be generated: either directly by selection or stochastically^{14,17}. Recent papers have examined both cases and we shall treat these in turn.

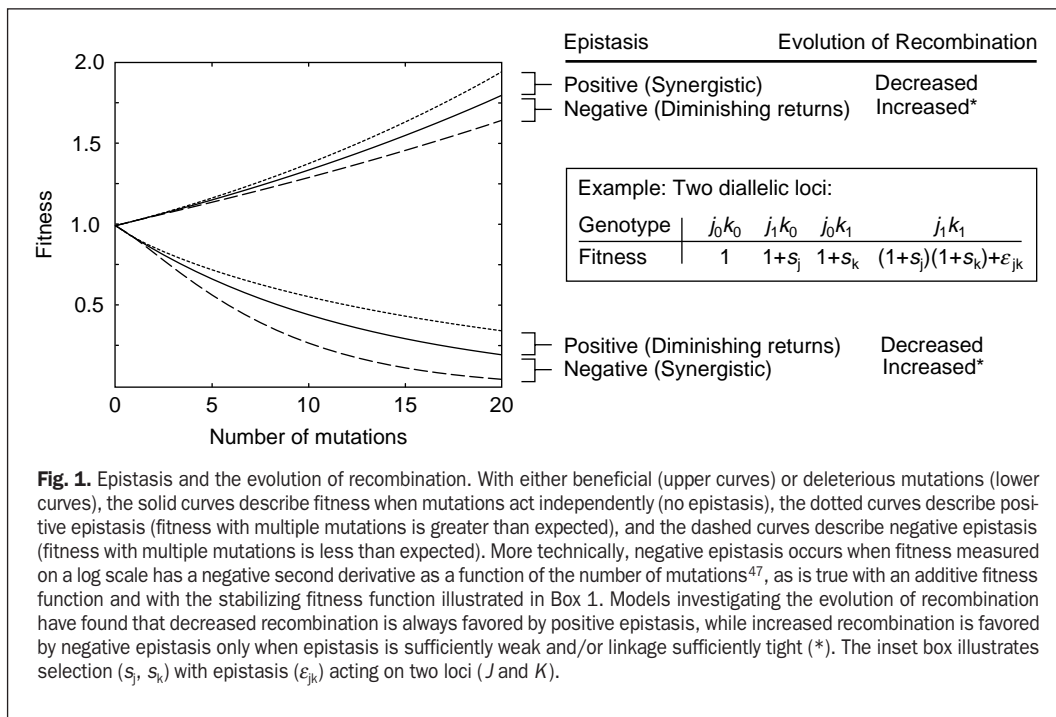


Fig. 1. Epistasis and the evolution of recombination. With either beneficial (upper curves) or deleterious mutations (lower curves), the solid curves describe fitness when mutations act independently (no epistasis), the dotted curves describe positive epistasis (fitness with multiple mutations is greater than expected), and the dashed curves describe negative epistasis (fitness with multiple mutations is less than expected). More technically, negative epistasis occurs when fitness measured on a log scale has a negative second derivative as a function of the number of mutations⁴⁷, as is true with an additive fitness function and with the stabilizing fitness function illustrated in Box 1. Models investigating the evolution of recombination have found that decreased recombination is always favored by positive epistasis, while increased recombination is favored by negative epistasis only when epistasis is sufficiently weak and/or linkage sufficiently tight (*). The inset box illustrates selection (s_j, s_k) with epistasis (ϵ_{jk}) acting on two loci (J and K).

increases recombination will generate more variable offspring. If epistasis is strongly negative, however, the average fitness of recombinant offspring above the mean and recombinant offspring below the mean will be lower than the average fitness of non-recombinant offspring nearer the mean. Therefore, with strong negative epistasis, the immediate fitness effects of increased recombination are disadvantageous³. Of course, having produced some offspring that are fitter than average, the modifier allele will gain a long-term advantage as long as it stays associated with the fittest genotypes, but it will do so only if its linkage to them is tight³. In summary, for increased recombination rates to evolve, there must be a short-term

Disequilibrium generated by selection

It has long been known^{18,19} that selection will not generate disequilibrium if selection acts independently (multiplicatively) at all loci. In this case, the fitness of an individual is the product of the fitness induced by selection at each locus and there is no epistasis (see Fig. 1). If, however, the selective effects of an allele depend on which alleles are present at other loci (i.e. epistasis is present), disequilibrium is generated between interacting loci^{18,20}. In the simplest situation presented in Fig. 1 (upper curves), each additional favorable mutation increases fitness by a larger and larger fraction under positive epistasis and by a smaller and smaller fraction under negative epistasis. Selection with positive epistasis produces more individuals carrying a large number of favorable alleles than expected based on current allele frequencies, generating positive disequilibrium within the population^{18,20}. Conversely, selection with negative epistasis produces fewer individuals with an extreme number of favorable alleles and more individuals near the mean, corresponding to negative linkage disequilibrium. It turns out that the form of epistasis is a critical determinant of whether or not evolution will favor increased recombination.

Modeling the evolution of recombination in the presence of directional selection with epistasis presents a formidable challenge, as it requires analysing non-equilibrium dynamics with at least three loci. Recent papers by Charlesworth⁶ (Box 1) and Barton³ (Box 2) have risen to this challenge using different approaches but achieving compatible results. Their results demonstrate that increased recombination rates can evolve under directional selection, but only if (1) epistasis among pairs of loci tends to be negative and if (2) either epistasis is weak relative to the strength of selection or the initial linkage between the modifier and fitness loci is tight. The requirement for negative epistasis makes sense: only then are extremely good genotypes underrepresented in the population so that recombination can increase their production. The second requirement is less intuitive, but can be explained by considering the fate of offspring of an individual carrying a modifier that increases recombination. If epistasis is negative, selection will have reduced the amount of variance in the population and a modifier allele that

advantage to increasing recombination (true only with weak negative epistasis) and/or a substantial long-term advantage (true only when epistasis is negative and linkage is tight).

Disequilibrium generated stochastically

In the above deterministic models, each beneficial allele within a population will be maintained forever, regardless of how rare the allele becomes. Many theories for recombination, however, focus on the ability of recombination to protect beneficial alleles from loss. For example, Fisher¹² argued (p. 122) that, in the absence of recombination, only those beneficial alleles that fall ‘upon the one individual whose descendants are destined ultimately to survive’ will be maintained in the population; all others will be lost. More generally, Hill and Robertson²¹ found that, within finite populations, selection at a locus increases the amount of random genetic drift and decreases the efficiency of selection at linked loci. To properly assess such effects, models must be constructed that include genetic drift and that allow allelic variants to be lost due to sampling.

In a second landmark paper, Barton⁵ analysed the effect of recombination on the fate of new beneficial alleles. He showed that the average probability of fixation of a new beneficial allele is raised by recombination as long as there is genetic variance for fitness at other loci. This occurs because, with tight linkage between fitness loci, selection acts on the level of the haplotype and therefore the dynamics of a beneficial allele will be governed more by the genomic background on which it arises and less by its own selective advantage. With loose linkage, however, selection at the locus itself becomes more important and drowns out the noise caused by selection at other loci.

Using a similar stochastic model, we subsequently showed that a modifier allele that increases recombination can be selected because it increases the fixation probability of advantageous alleles, even if there are no epistatic interactions among loci⁸. A modifier allele that increases recombination increases the chance that new beneficial alleles will fix and, as these alleles rise to fixation, they carry the modifier allele along with them by a process known as genetic

hitchhiking. The strength of selection acting on a modifier of recombination decreases rapidly, however, as a function of the current level of recombination within the genome for two reasons: other loci then have less influence on the fixation probability of a beneficial allele and the modifier only receives a short hitch before recombining away from the beneficial allele. Therefore, while increasing the probability of fixation of beneficial alleles can favor increased recombination, selection for recombination is only substantial when linkage is tight or when beneficial alleles arise frequently throughout the genome. This latter condition suggests that recombination rates need not be static but may rise during periods of rapid environmental change and intense selection^{15,22}.

Mutation–selection balance

Increased recombination rates may also evolve in models where the progress of directional selection is opposed by the continuous appearance of deleterious mutations^{4,9,23}. Barton³ synthesized our mathematical understanding of the evolution of recombination under different selection regimes, demonstrating that the same general conditions are required for recombination to evolve with directional selection and with a mutation–selection balance. For increased recombination to be favored at a mutation–selection balance, there must again be negative epistasis between deleterious alleles (see Fig. 1, lower curves) and this epistasis must be weak or linkage between the modifier and fitness loci must be tight (see Box 2). Whether or not epistasis is weak enough relative to selection now depends on the mutation rate^{3,23}. With negative epistasis, every additional mutation within the genome experiences stronger and stronger direct selection. That is, when the mutation rate is high, the population is pushed further from the genotype with highest fitness, increasing the strength of directional selection relative to the strength of epistasis and increasing the likelihood that increased rates of recombination will evolve.

Under mutation–selection balance, the strength of selection acting to increase recombination is proportional to (1) the square of the genome wide mutation rate, (2) the effect of the modifier on recombination, and (3) the amount of negative epistasis and is inversely proportional to (4) the recombination rates among loci³. For epistasis among mutant alleles to have a large influence on the evolution of recombination, therefore, relatively high mutation rates or tight linkage throughout the genome are required.

Fluctuating selection

Fluctuating selection, with changes over time in the direction of selection, may also favor increased recombination. Fluctuating selection arises naturally in many models of species interactions (see next section), but will also occur in response to climatic and seasonal fluctuations. Surprisingly, models that have incorporated fluctuating selection coefficients tend to be less favorable to the evolution of recombination than models of directional selection. Nevertheless, two cases in particular have been shown to favor the evolution of increased recombination.

When selection fluctuates rapidly, there is little sustained directional selection and the mean phenotype changes little over time. In this case, selection for decreased recombination is favored as in a constant environment *unless* the fluctuations involve changes in the sign of epistasis^{2,3,24}. That is, the allelic combinations that are favored in one generation must be disfavored in the near future³. In the simplest case of a symmetrical two-locus model where allele frequencies are held constant but genotypic fitnesses vary sinusoidally over time (i.e. the sign of epistasis varies over time), Sasaki

Box 1. A quantitative genetics model of recombination

Charlesworth⁶ investigated a quantitative genetics model under stabilizing selection whose optimum (θ) changes every generation by $\Delta\theta$. In this model, fitness of genotype x is given by:

$$e^{-(x-\theta_n)^2/(2V_s)}$$

where θ_n is the optimum in the n^{th} generation and V_s measures the width of the fitness function acting on the genotype (when selection is weak, V_s is large). Regardless of changes in θ_n , the genotypic variance remains constant under stabilizing selection. For example, in a freely recombining haploid population with genome wide mutation rate U , the genotypic variance is $2UV_s$ (Ref. 6). Consequently, the mean but not the variance of the frequency distribution of genotypic values (solid curves) changes over time following shifts in the fitness function (dashed curves). In this example, three generations are shown (0, 40 and 80, as marked at the top of each curve), using parameters values: $\Delta\theta=0.05$, $V_s=20$, $U=0.01$.

While the genotypic variance does not depend on $\Delta\theta$, it does depend critically on the mating system and recombination rate within a population. The equilibrium genetic variance rises as a function of the recombination rate, because genotypes achieving the optimum using different combinations of alleles are mixed together to form progeny that fall both below and above the optimum. This increased variance due to recombination improves the ability of a population to track changes in the optimum. By calculating the effect of recombination on the genetic variance and phenotypic mean of a population, Charlesworth⁶ determined the fate of a modifier of recombination, paying particular attention to two important cases. A modifier that increases recombination can spread in a non-recombining population if approximately

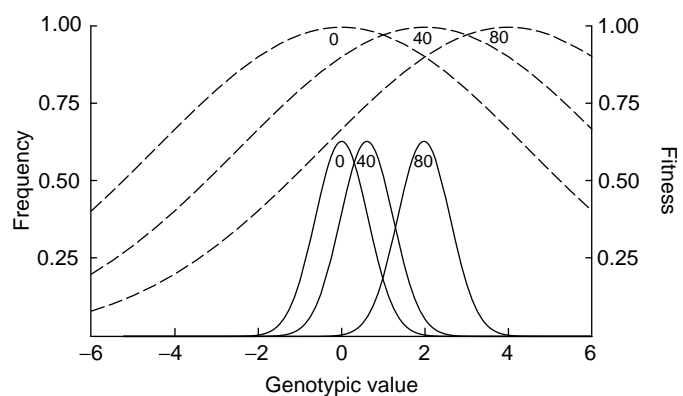
$$|\Delta\theta| > V_{G(\text{No})}^{3/2} (\sqrt{2}V_s)$$

where $V_{G(\text{No})}$ is the genetic variance in a non-recombining population. Conversely, a freely recombining population will be stable against the invasion of modifiers that decrease recombination if

$$|\Delta\theta| > V_{G(\text{Free})} \sqrt{3V_s}$$

where $V_{G(\text{Free})}$ is the genetic variance maintained under free recombination. Both of these conditions may be satisfied if the optimum changes rapidly enough, but the first tends to be far easier to satisfy. Consequently, under intermediate rates of change of the environment, recombination may be expected to evolve toward an intermediate level.

This model may appear odd in that variance remains constant regardless of how rapidly the environment changes. This feature relies on the assumption of an infinite population with an infinite number of loci subject to stabilizing selection. Kondrashov and Yampolsky⁴⁵ showed by simulation that, in a finite population, the genetic variance can increase over time under stabilizing selection with a changing optimum, as rare alleles become common and contribute more variability to the population. Nevertheless, their conclusions⁷ concerning the evolution of recombination did not differ qualitatively from those of Charlesworth⁶. When other fitness functions are used, variance does change over time. For example, under directional truncation selection, genetic variance is rapidly depleted, increasing the advantage of recombination^{6,46}.

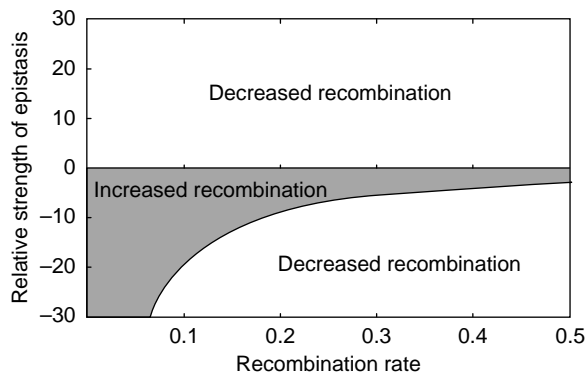


and Iwasa²⁵ found that recombination rates evolve to about $2\pi/T$, where T is the period of the epistasis cycle, so that high rates of recombination are favored only when epistasis changes sign frequently. While such rapid shifts in the sign of epistasis are unlikely to be caused by fluctuations in the physical environment, they may be common with biotic interactions.

Box 2. A general multi-locus model of recombination

By assuming that modifier alleles only weakly affect recombination rates, Barton³ was able to simplify multi-locus dynamical equations to obtain general results concerning the evolution of recombination. When selection and epistasis are extremely weak relative to recombination, decreased recombination rates are favored. This is because the selective environment is nearly constant on the time scale over which recombination acts (confirming the Reduction Principle). With stronger directional selection, however, modifier alleles that increase recombination can be favored, but only if the weighted average value of $\Psi = \epsilon_{jk} [s_j s_k (1/r_{ij} + 1/r_{ik} - 1) + \epsilon_{jk}]$ is negative, where s_j , s_k , and ϵ_{jk} measure the strength of selection (s) and epistasis (ϵ) acting on loci J and K and where r_{ij} and r_{ik} are the recombination rates between a modifier locus (I) and loci J and K . This condition is shown graphically for a three-locus model, where the order of the loci is assumed to be IJK . Only within the shaded region will an allele that increases recombination be favored. Here, the x -axis gives the amount of recombination between adjacent loci. The y -axis gives the strength of epistasis measured relative to the product of the selection coefficients at loci J and K [$\epsilon_{jk}/(s_j s_k)$]. The region in which recombination is favored is identical whether the beneficial alleles are sweeping through the population or are held at a mutation-selection balance³. This condition is accurate only for sufficiently loose linkage and weak epistasis and can overestimate the advantage of recombination in other cases (see Barton³ for further details). For increased recombination to evolve (i.e. for Ψ to be negative), there must be negative epistasis and this epistasis must be weak relative to selection, especially if the loci are already loosely linked.

In this model, the strength of selection for recombination equals the weighted average of $-\delta r/r_{jk} \Psi p_j (1-p_j) p_k (1-p_k)/r_{jk}$, where $\delta r/r_{jk}$ measures the proportional increase in recombination caused by the modifier, p_j and p_k measure the frequency of beneficial alleles at loci J and K , and r_{jk} is the probability that recombination occurs somewhere between loci J and K (Ref. 3). The amount of selection for increased recombination is, therefore, likely to be weak, unless linkage is tight or directional selection is strong and persistent.



Fluctuations occurring over a longer time scale will generally create sustained directional selection, causing changes in the mean phenotype over time (e.g. Fig. 2). In this case, as with directional selection, there must be weak negative epistatic interactions between currently favored alleles for increased recombination rates to evolve³. Fluctuating selection tends to be less effective than directional selection, however, for a number of reasons⁶. Since the mean phenotype lags behind the optimal phenotype, there are points in time when the optimum passes through the mean of the population (dots in Fig. 2) and at these points decreased recombination is favored. (This may be avoided if selection occurs on many traits and does not retrace its steps⁷.) Even so, as the direction of selection switches, selection weakens relative to the strength of epistatic interactions, reducing the advantage of recombination. Consequently, the conditions under which increased recombination is favored are rather stringent. For example, if there is stabilizing selection with a fluctuating optimum as illustrated in Fig. 2, recombination is favored only when the amplitude (A) of fluctuations is high and selection is strongly stabilizing (i.e. low V_S) such that approximately $A^2 > V_S$ (Refs 6,7). The problem with this condition

is that it leads to a very high fitness cost to the population. As shown in Fig. 2, the phenotypic mean tends to lag 90° behind the optimum in this model (unless the period is long and selection relatively strong), causing the average fitness to be quite low whenever the optimum fluctuates widely⁶. For instance, Kondrashov and Yampolsky⁷ found that more than 30% of the population must die or fail to reproduce due to fluctuating selection alone before free recombination is favored. Given this high fitness cost, fluctuating selection seems unlikely to explain the maintenance of high rates of recombination.

Parasite-mediated fluctuating selection

The potential of host-parasite interactions to generate fluctuating selection that could favor the evolution of increased recombination has long been recognized²⁶ and has been championed in a number of papers by Hamilton²⁷⁻²⁹. At first, it might appear that host-parasite models should add little to the results on fluctuating selection reviewed in the previous section. Host-parasite models exhibit one essential difference, however, in that changes in the genetic system of the host or parasite can cause changes to their realized fitness functions. With higher rates of recombination, a host can more effectively out-evolve its parasites and experience milder selection.

Nee³⁰ examined the evolution of recombination in a simplified two-locus model similar to that of Sasaki and Iwasa²⁵, but where fitness is imposed by the coevolutionary dynamics between a host and a parasite (see Fig. 3). In this model, allele frequencies remain constant, but the sign of epistasis varies over time depending on the parasites present. This model, while biologically unrealistic, can be readily analysed³⁰ and does capture the essential advantage of recombination in host-parasite models, that of recombining common genotypes to produce rare combinations of alleles that may be less susceptible to parasites. Nee's³⁰ results are fundamentally different from those found in the non-parasite models with fluctuating selection. In particular, the main effect of increased recombination is to change the phase relationship between host and parasite. The more a host recombines, the further out of phase the host is with its parasite (and the nearer the host stays to its maximum fitness). In contrast, in the models of Sasaki and Iwasa²⁵ and Charlesworth⁶, the phase relationships are fairly insensitive to the degree of recombination. In addition, whether or not recombination raises mean fitness depends only weakly on the period of the host-parasite cycle³⁰, but depends strongly on the period of fluctuating selection in single species models^{3,25}.

Although the model of Nee is highly simplified, it does suggest that evolutionary biologists must model host-parasite dynamics directly and avoid drawing inferences about coevolutionary dynamics from single species models. Unfortunately, it is difficult at this stage to determine the exact conditions under which increased recombination will be favored in host-parasite models, let alone to determine the strength of selection on a modifier of recombination. This is largely because most host-parasite models examine the evolution of sex and not the evolution of recombination. Looking for insight from models of the evolution of sex suggests many factors that may play an important role and raises many questions for future study.

All host-parasite models have one thing in common: there is negative frequency-dependent selection. Hosts with common resistance-genes constitute a favorable environment for the selection of parasites able to overcome their defenses. When such parasites become common, hosts that were previously rare but are immune to the currently dominant

parasites are at an advantage. Different host–parasite models vary, however, in almost every other possible dimension. For example, Fig. 3 illustrates some of the various ways in which host–parasite interactions have been modeled. Comparing the host–parasite models suggests three distinct factors that favor the evolution of sex and that might also favor the evolution of recombination.

With negative frequency-dependent selection, rare genotypes have, on average, a fitness advantage over more common genotypes. By definition, when linkage disequilibrium is present in a population, some genotypes are rarer than expected. By breaking down disequilibrium, sex and recombination generate these rare genotypes and can benefit from this association. This is the primary process acting in Nee’s³⁰ model.

A second process that contributes to the evolution of sex in many host–parasite models is that, by destroying linkage disequilibrium, allele frequencies can change more rapidly in the face of selection, increasing the likelihood that the host will be able to evade parasitic attack. As in the models of directional selection, recombination will only improve the response to selection if there is negative linkage disequilibrium, and hence negative epistasis, among currently favorable alleles. Indeed, Bell and Maynard Smith found that free recombination evolved in the organism subject to negative epistasis (parasites subject to stabilizing selection) but not in the organism subject to positive epistasis (hosts subject to disruptive selection)³¹. Host–parasite models commonly generate negative epistasis (e.g. those described in Fig. 3), in that the advantage of having resistant interactions at two loci is often less than the advantage expected from resistance at one locus.

Finally, a third process at work is that sex and recombination can generate genotypes that are currently *absent* within a population. Once a genotype is lost in an asexual population, there may be a substantial delay before it is regenerated by mutation or migration. If the absent genotype becomes the fittest possible one, an asexual population will suffer a reduced mean fitness during this time. This mechanism has been called the ‘gene-storage effect’²⁸, since sex uses alleles currently ‘stored’ in other genotypes to regenerate a lost genotype. This gene-storage advantage of sex is functionally equivalent to Muller’s ratchet³² in non-coevolutionary models. The difference between the two is that in coevolutionary models the fittest genotype depends on the composition of the parasite population and therefore changes over time, while in Muller’s ratchet the relationship between fitness and genotype remains constant. In host–parasite models, the ‘ratchet’ will click more frequently if the population is small and there are a large number of loci and if the parasites are efficient at eliminating host genotypes from a population (that is, are highly transmissible³³ or cause severe damage^{33,34}).

In summary, the results of any given host–parasite model will depend on the form of genetic interactions between hosts and parasites (a matter of active debate^{35–38}), on the way these interactions are summed across loci, on the size of the population and on the number of loci. The extent of directional selection is particularly important in that, if directional selection is too strong (as in the gene-for-gene model described in Fig. 3), polymorphism can be lost and cycling will end. In this case, all of the above advantages of sex and recombination will cease, as has been observed in gene-for-gene models³⁴ and in models with a highly structured host population^{39,40} (a.k.a. the Friedman effect). In addition, the advantages of sex and recombination will depend on the extent to which alternative mechanisms exist to generate genotypic diversity (e.g. mutation²⁸ or migration^{39,40}).

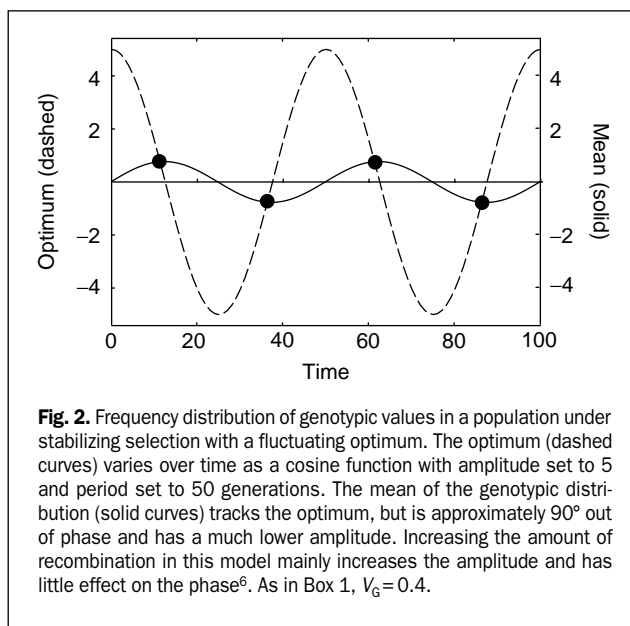


Fig. 2. Frequency distribution of genotypic values in a population under stabilizing selection with a fluctuating optimum. The optimum (dashed curves) varies over time as a cosine function with amplitude set to 5 and period set to 50 generations. The mean of the genotypic distribution (solid curves) tracks the optimum, but is approximately 90° out of phase and has a much lower amplitude. Increasing the amount of recombination in this model mainly increases the amplitude and has little effect on the phase⁶. As in Box 1, $V_G = 0.4$.

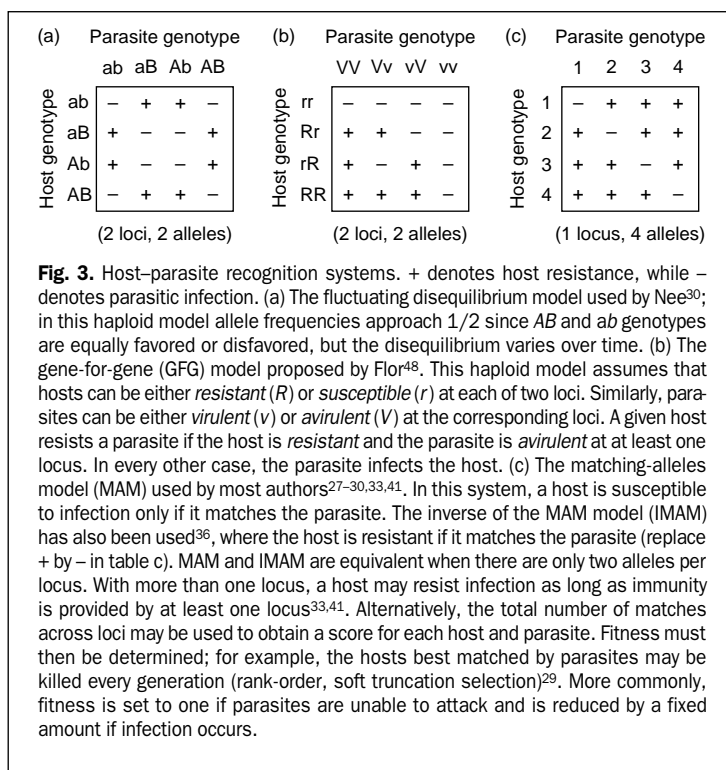


Fig. 3. Host–parasite recognition systems. + denotes host resistance, while – denotes parasitic infection. (a) The fluctuating disequilibrium model used by Nee³⁰; in this haploid model allele frequencies approach 1/2 since AB and ab genotypes are equally favored or disfavored, but the disequilibrium varies over time. (b) The gene-for-gene (GFG) model proposed by Flor⁴⁸. This haploid model assumes that hosts can be either *resistant* (R) or *susceptible* (r) at each of two loci. Similarly, parasites can be either *virulent* (v) or *avirulent* (V) at the corresponding loci. A given host resists a parasite if the host is *resistant* and the parasite is *avirulent* at at least one locus. In every other case, the parasite infects the host. (c) The matching-alleles model (MAM) used by most authors^{27–30,33,41}. In this system, a host is susceptible to infection only if it matches the parasite. The inverse of the MAM model (IMAM) has also been used³⁶, where the host is resistant if it matches the parasite (replace + by – in table c). MAM and IMAM are equivalent when there are only two alleles per locus. With more than one locus, a host may resist infection as long as immunity is provided by at least one locus^{33,41}. Alternatively, the total number of matches across loci may be used to obtain a score for each host and parasite. Fitness must then be determined; for example, the hosts best matched by parasites may be killed every generation (rank-order, soft truncation selection)²⁹. More commonly, fitness is set to one if parasites are unable to attack and is reduced by a fixed amount if infection occurs.

Concluding remarks

The common feature shared by all models reviewed here is that they consider the evolution of recombination in an environment which is changing in some way. Under directional selection for beneficial mutations and mutation–selection balance against deleterious mutations, increased recombination is favored in the presence of negative epistasis when linkage is initially tight^{3,6}. These forms of selection favor the initial increase of recombination from zero, but their power fades as recombination increases and disappears when epistasis is strong relative to selection^{3,6}. Variation among loci in the sign and strength of epistatic interactions will further limit the conditions under which recombination is able to evolve⁹. This can be seen from the figure in Box 2: even if the average strength of epistasis falls within the shaded region, variability in the amount of epistasis between loci will cause many pairs of loci to fall outside

of this region, reducing or even eliminating selection for increased recombination. Therefore, even though these processes might explain the evolution of higher rates of recombination in specific regions of the genome, it seems unlikely that epistasis is generally of the right form to promote the evolution and maintenance of recombination throughout the genome.

Despite the intuitive appeal of the idea that fluctuations in the direction of selection should favor increased recombination, the mathematical models reviewed here agree, overall, that this is unlikely^{3,6,7,25}. Indeed, unless the direction of selection changes at very specific time intervals the benefits conveyed by recombination in allowing populations to track a shifting optimum do not compensate for the disadvantages of breaking down beneficial gene combinations.

In sharp contrast, models where fluctuating selection is imposed by coevolutionary interactions between hosts and parasites allow the relatively easy evolution of sex, and perhaps of increased recombination, as long as polymorphism is preserved^{27-30,33,41}. The discrepancy between fluctuating selection and host-parasite models may stem from the fact that, in the former, the fitness function over time is independent of how the organism reacts while, in coevolutionary models, the very reaction of an organism affects the fitness function in future generations. It will be difficult to assess whether this contention is valid or not until we better understand the importance of different mechanisms favoring sex and recombination in host-parasite models.

It may, of course, be the case that a complete picture of the evolution of recombination can only be gained by studying several mechanisms acting simultaneously. Recent studies have steered in this direction, illustrating that several mechanisms can act synergistically and favor increased recombination over and beyond what each mechanism can do singly. For example, with both host-parasite interactions and Muller's ratchet operating, Howard and Lively³³ found that sex was even more likely to evolve, because host-parasite interactions kept clones from becoming so common that Muller's ratchet would grind to a halt. Similarly, Peck¹⁰ showed that beneficial mutations could be incorporated much more easily into a sexual population than into an asexual population when there were frequent deleterious mutations. This occurs because a beneficial mutation that arises on a haplotype bearing many deleterious mutations is very likely to be lost in an asexual population, since recombination cannot place the beneficial mutation onto a better genetic background. Indeed, there is no reason why a single mechanism should explain all the observed patterns of recombination: all organisms are subject to parasitic attack, deleterious mutations, and beneficial mutations.

Experimental studies have begun to accumulate showing that the mechanisms discussed within this paper do, under certain circumstances, lead to the evolution of recombination. Directional selection acting on traits other than recombination has led to higher recombination in a number of cases (reviewed in Refs 15 and 22). These experiments indicate that either there is widespread negative epistasis among pairs of loci³ or there is substantial disequilibrium generated stochastically within populations⁸, resulting in selection for increased recombination. Similarly, recent experimental studies have shown that sex may be adaptive in the face of antagonistic coevolutionary interactions⁴² and may be important in the elimination of deleterious mutations from the genome⁴³. Conversely, reductions in the frequency of sex have been observed under fluctuating selection⁴⁴. These experiments are not definitive and require evaluation in a wider variety of species. Furthermore, there is need for experiments

that evaluate more than one hypothesis at a time to determine their relative strengths (e.g. Ref. 43). Nevertheless, each such experiment brings us closer to assessing the importance of different hypotheses for recombination, an assessment aided by recent advances in the evolutionary theory of recombination.

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Evolutionary explosions and the phylogenetic fuse

Alan Cooper and Richard Fortey

George Gaylord Simpson, in *Tempo and Mode of Evolution*¹, made a pioneering attempt to describe patterns of speciation as deduced from the fossil record. Numbers of species within major groups of organisms clearly fluctuated throughout their history. It was evident that there were times when many new major taxa appeared over a geologically short time period; other times when a background rate of origination and extinction prevailed. One of the patterns Simpson identified was a rapid evolutionary ‘burst’ which was preceded in some cases by an attenuated ‘tail’ – a greatly extended, but often obscure earlier history before the group of organisms in question expanded into prolific evolutionary creativity. Probably the best example of this pattern was provided by the mammals and birds. Both groups have a prolonged but poorly known period of evolution during the Mesozoic when many ‘archaic’ groups are observed – the birds exemplified by arguably the most famous fossil of them all, the Jurassic *Archaeopteryx*². After the disappearance of the dinosaurs at the end of the Cretaceous,

A literal reading of the fossil record indicates that the early Cambrian (c. 545 million years ago) and early Tertiary (c. 65 million years ago) were characterized by enormously accelerated periods of morphological evolution marking the appearance of the animal phyla, and modern bird and placental mammal orders, respectively. Recently, the evidence for these evolutionary ‘explosions’ has been questioned by cladistic and biogeographic studies which reveal that periods of diversification before these events are missing from the fossil record. Furthermore, molecular evidence indicates that prolonged periods of evolutionary innovation and cladogenesis lit the fuse long before the ‘explosions’ apparent in the fossil record.

Alan Cooper is at the Dept of Biological Anthropology, Oxford University, Oxford, UK OX2 6QS;
Richard Fortey is at the Dept of Palaeontology, The Natural History Museum, Cromwell Road, London, UK SW7 5BD.

the Palaeocene and Eocene Epochs saw the appearance of many of the ancient sister-groups of the kinds of mammals and birds that still prosper today – and many more extinct kinds besides. Stanley subsequently showed³ that the rate of mammalian evolution during the early Tertiary was, indeed, exceptionally fast, and it has become customary to describe such phases as ‘radiations’, or even as ‘explosions’ – an analogy of unbridled creation inevitably invoking chain reactions. The first appearance of many animal phyla in the Cambrian is often described in similar terms as the Cambrian ‘evolutionary explosion’⁴.

Although such evolutionary radiations have been extensively studied, the earlier histories that preceded them have not received quite the same attention. There has been an assumption that the first appearance of familiar groups in the fossil record does closely approximate their time of origin –

that is, the rocks provide a correct narrative of the time of first appearance of taxa. On this account, ‘true’ birds are largely a post-Cretaceous radiation⁵, and the Cambrian was