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Natural Selection and Random Genetic Drift as Causes of Evolution on Islands [and Discussion]

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Natural selection and random genetic drift as causes of evolution on islands

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SUMMARY

The evolutionary processes responsible for adaptation and speciation on islands differ in several ways from those on the mainland. Most attention has been given to the random genetic drift that arises when a population is founded from just a few colonizing genomes. Theoretical obstacles to 'founder effect speciation' are discussed, together with recent proposals for avoiding them. It is argued that although certain kinds of epistasis can facilitate the evolution of strong reproductive isolation, this favours divergence by selection as much as by random drift.

1. INTRODUCTION

Evolution on islands may differ in many ways from that on the mainland. A novel physical environment, and fewer competing species, may allow unusual adaptive radiations (Schluter 1996). The total number of individuals in the species may be low, giving fewer advantageous mutations, and allowing deleterious mutations to accumulate (Kondrashov 1995). Finally, populations may be founded by one or a few colonists, giving an immediate burst of random drift (Mayr 1954). I will concentrate on this last aspect of evolution on islands, and in particular, on the idea that genetic drift in founder events may lead to the formation of new species. Because random drift is a well-understood and ubiquitous process, its effects are most amenable to theoretical generalities; indeed, most discussions of speciation on islands (verbal and mathematical) have concentrated on drift in founder events. However, this should not lead us to neglect the importance of natural selection: although it is hard to say much in general, its importance in particular cases is clear (for example, see papers by Clarke *et al.*, Grant & Grant, Schluter and Thorpe & Malhotra, this volume).

I will begin by describing the apparent obstacles to the evolution of the reproductive isolation which defines biological species, and the way founder effects have been seen as avoiding these difficulties. I will then summarize general arguments which suggest that random drift (whether in established populations or during founder events) is unlikely to lead to strong reproductive isolation. These arguments were set out in detail by Barton & Charlesworth (1984) and Barton (1989*b*). I then discuss recent models which have been suggested as ways of avoiding these arguments, and which would make it likely that strong reproductive isolation could evolve in a single step. Finally, I consider the kinds of evidence which might tell us whether random genetic drift has contributed to the often dramatic radiations of species on islands.

Before getting mired in the details of particular models, it is important to keep in mind the general questions. Do populations typically contain genetic variation that can be reshuffled so as to give strong reproductive isolation? Do new species form when random genetic drift reshuffles this variation? Does this drift occur during drastic population bottlenecks? The answer to the first question, in particular, is relevant to many alternative mechanisms of speciation, as well as to the likely genetic basis of adaptive evolution.

2. OBSTACLES TO SPECIATION

The origin of species involves a special difficulty, which does not apply to adaptive evolution in general. Suppose that we take species to be 'biological' species; that is, we define a species as a group of individuals which share genetic differences that prevent them exchanging genes with other such species. The evolution of biological species therefore requires the establishment of genotypes which cause some reproductive isolation. The key difficulty is that such genotypes should not be able to invade the population: either they would not be chosen as mates, or if they did mate, they would leave fewer offspring. Thus models for the evolution of reproductive isolation lead to multiple stable equilibria, so that evolution away from one equilibrium state is resisted by natural selection. This is easiest to understand with postzygotic isolation, where recombinant or heterozygous genotypes have lower fitness. Selection then tends to increase mean fitness, driving the population towards alternative 'adaptive peaks'. From this viewpoint, movement from one peak to another requires passage through an unfit intermediate state, and so is resisted by selection. Multiple equilibria also emerge in models where non-

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random mating leads to stable reproductive isolation (e.g. Moore 1979). However, such models usually involve frequency-dependent fitnesses, and hence cannot be described by an 'adaptive landscape'.

The simplest genetic model of reproductive isolation is where heterozygotes at a single locus have reduced fitness; the classic example is where heterozygotes for chromosome rearrangements are partly sterile because of failure in pairing and segregation at meiosis. There are thus two peaks in the graph of mean fitness against allele frequency. Frequency-dependent selection can have a similar effect if it favours the commoner morph. For example, the snail *Partula suturalis* may coil in either the sinistral or dextral direction; coiling direction is determined by a single locus. Because the rarer morph will usually mate with the opposite type, it will both fertilize and be fertilized less efficiently. Hence, just as with underdominant chromosome rearrangements, populations tend to fix one or other morph, and polymorphism is confined to the narrow zones where the alternative morphs meet (Johnson *et al.* 1990; and paper by Clarke *et al.*, this volume). Other examples of alternative equilibria include the production of colicins in bacteria, where the cost of producing the toxin is outweighed by its advantage in killing competitors only above a threshold frequency (Chao 1979) ; the spread of *Wolbachia* through *Drosophila simulans*, where a similar frequency-dependent advantage arises because infected males sterilize their uninfected mates (Turelli & Hoffmann 1995) ; and Müllerian mimicry, where the rarer morph is at a disadvantage because it is not recognized as being distasteful (see paper by Turner & Mallet, this volume). These examples have a simple genetic basis; below, we consider whether more complex models of reproductive isolation necessarily sustain alternative stable equilibria, and if so, how populations may diverge into these alternative states.

3. DEFINING REPRODUCTIVE ISOLATION

The formation of biological species requires genetic differences that prevent gene exchange; that is, the evolution of reproductive isolation. To compare mechanisms of speciation, we need know how much reproductive isolation they produce. This is not straightforward; indeed, much of the theoretical argument over whether founder events can cause strong reproductive isolation depends on how this isolation is measured. There are two difficulties: first, the effect of genetic differences on gene flow depends on just how the divergent populations meet; and second, their eventual contribution to future reproductive isolation is not in general predictable from their immediate effect on gene flow. I have argued that the degree of isolation is best measured by the mean fitness of the population that forms when two divergent populations hybridize (Barton & Charlesworth 1984, 1989*b*). Here, I summarize this argument, and discuss the difficulties with this and with alternative measures.

Suppose that divergent populations meet along a continuous habitat. Selection on those genetic differences responsible for keeping the populations at their different equilibria will maintain a set of clines, which may persist indefinitely. Unless F_1 hybrids are completely inviable or sterile, genes at other loci can flow from one population to the other. However, this exchange will be impeded by the selection on other loci: genes may be eliminated by selection against introgression of the genes with which they are initially associated before they can recombine onto the other genetic background.

This 'hitch-hiking' effect is reflected directly in the sharp clines in marker alleles seen in hybrid zones. For example, the toads *Bombina bombina* and *B. variegata* are separated by a hybrid zone (see figure 1*a* in Szymura & Barton 1991). This concordance makes it unlikely that each locus is responding directly to selection; indeed, these markers are in strong 'linkage disequilibrium' with eachother in hybrid populations, even though they segregate independently in crosses. Rather, the sharp clines reflect the net effects of selection on the rest of the genome. The ratio between the step in these clines and the gradient on either side, $B = \Delta u/(\mathrm{d}u/\mathrm{d}x)$, gives a measure of the strength of the barrier to gene flow (figure 1*a*). This has the dimensions of a distance, and can be thought of as the span of unimpeded habitat which would present the same obstacle to flow of a neutral allele. It applies to any localized barrier, whether genetic or physical, and can be used to find the effect on selected as well as neutral alleles.

A simple model in which linkage disequilibria are generated by the mixing of divergent populations predicts that the gradients in marker alleles should depend primarily on the mean fitness of the population, through the relation $(\mathrm{d}u/\mathrm{d}x) \approx \bar{W}^{-1/r}$ (where r is the harmonic mean recombination rate with the selected loci; see Barton 1986). Hence, the ratio between the barrier strength and cline width is $(B/w) = \bar{W}^{1/r}$, where is the mean relative fitness of the hybrid population (see figure 1*b*).

This relation applies to any model in which genotypes have fixed fitnesses; simulations show that though derived as a weak selection limit, it is accurate up to fairly strong selection (Barton & Gale 1993). However, if selection is strong, the mean fitness of the hybrid population may be much less than that of the ancestral populations connecting the two forms (see below). This is because if hybrids are on average unfit, particular recombinants with high fitness cannot easily be reconstructed. Similarly, if immigrants are rare, the barrier to gene flow depends primarily on the fitness of the first generation hybrids (Bengtsson 1985; Barton & Bengtsson 1986), which may be much lower than that of the hybrids which might form in a continuous habitat, or of the ancestral genotypes. Evidence of this effect is seen in the transition between chromosome races of the grasshopper *Podisma pedestris*. In most places, these meet in a smooth cline, showing no evidence of a barrier. However, across two small streams, gene flow is reduced much more than can be explained by the direct reduction in dispersal. This can be accounted for by a genetic barrier to gene flow due to reduced fitness of F_1 and backcross hybrids (Jackson 1992; Barton & Gale 1993).

Thus, the effect of genetic divergence on the mean

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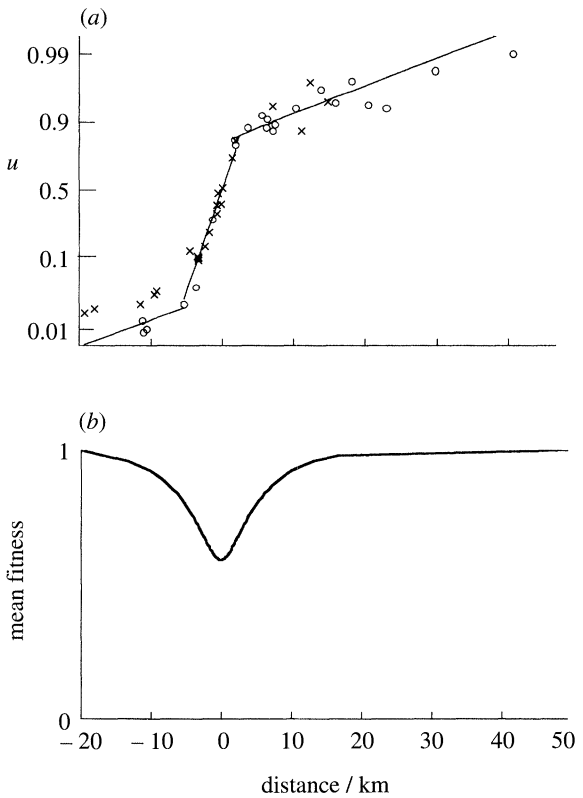

Figure 1. The barrier to gene flow between the toads *Bombina bombina* and *B. variegata*. Symbols show the average frequency, u, of alleles diagnostic for variegata at 5 enzyme loci, across two transects in southern Poland (Szymura & Barton 1991). (Open circles: Przemysl; crosses: Krakow.) The lines show the best fitting cline, which has width $w = (\mathrm{d}u/\mathrm{d}x)_{\max} = 6.0$ km, and corresponds to a barrier $B_v = \Delta u/(\mathrm{d}u/\mathrm{d}x)_v = 51$ km to flow into *variegata*, and $B_b = 260$ km to flow into *bombina*. (*b*) The reduction in mean fitness needed to account for this stepped cline (harmonic mean recombination rate is $r = 0.25$, and assuming selection against heterozygotes at 55 loci, as estimated from these data).

fitness of hybrids, and on gene flow, depends on just how those hybrids are formed. A further difficulty is that the contribution of divergence to eventual speciation may not be reflected by its immediate effect on gene exchange. One could argue that speciation may be completed in allopatry, in which case the flow of genes across continuous hybrid zones is irrelevant. Even if speciation is entirely parapatric, gene flow may hardly slow divergence. If selection against hybrids is strong, as is required for biological speciation, the rate of gene flow depends mainly on F_1 or backcross fitness: one could measure progress to full speciation by the contribution to reducing this fitness. If one assumes that shifts in different sets of genes have multiplicative effects, then one might use the reduction in F_1 or backcross fitness as the measure of isolation, even though the immediate effect on gene flow depends more on the mean fitness of a hybrid population, and may be much smaller. However, effects may well not multiply; by analogy with deleterious mutations (Kondrashov 1988) hybrid fitness may decrease faster than geometrically with divergence.

The fundamental problem in defining a measure of 'reproductive isolation' is that the effect of any one difference on gene flow depends on what else is different. In the early stages of speciation, hybridization can readily produce a variety of recombinants; the mean fitness of a hybrid population can be increased by selection of the fitter recombinants, and it is this mean fitness that is the most appropriate measure. When hybrids have low fitness, it is likely they will be kept at low frequency, and it is their average fitness that determines the degree of isolation. Both measures must therefore be considered in judging the plausibility of founder effect speciation.

4. DOES RANDOM GENETIC DRIFT CAUSE REPRODUCTIVE ISOLATION?

(*a*) *Models of founder-effect speciation*

If species are maintained by natural selection at stable equilibria, then it is at first hard to see how they could be formed. This difficulty led to the idea that random drift causes speciation, by knocking populations from one equilibrium to another despite the opposition of selection. Mayr (e.g. 1942, 1982) has been the most influential proponent of such ideas. He argues that species cannot change significantly in the main part of their range, because gene flow prevents divergence, and because genes are closely coadapted with eachother. Mayr's model of 'peripatric speciation' proposes that species form as a result of founder events in peripheral isolates; these cause a loss of heterozygosity which supposedly changes selection pressures and triggers 'genetic revolutions'. Carson (1968, 1975) has proposed a similar model of 'founder-flush speciation', which includes the effects of relaxed selection during the rapid increase after colonization. Finally, Templeton (1980) has proposed that founder events may trigger a 'genetic transilience' if a fewer major loci interact with many modifiers of small effect (see paper by Hollocher, this volume).

Mayr has argued that founder events have effects distinct from random drift, and that theories of founder effect speciation originated independently of Wright's theories of a 'shifting balance' between different evolutionary processes. However, Mayr, Carson and Templeton's theories of founder effect speciation all trace back to Wright's ideas (see Provine 1989). Wright proposed the 'shifting balance' primarily as a more efficient means of adaptation, but recognized that it would also lead to reproductive isolation (1940, 1982). The 'shifting balance' differs from the other theories in that it involves a network of partly isolated demes, giving many opportunities for peak shifts to occur by drift and founder effects. A new 'adaptive peak' which arises in one of many trials can spread through the whole population, making reproductive isolation more likely. Theories of founder effect speciation assume that the colonizing population spreads over a large area. A population therefore traces its ancestry back through just a few successive bottlenecks, which greatly reduces the chances that strong isolation will evolve.

There are many difficulties with the particular theories outlined above; these are discussed in reviews by Barton & Charlesworth (1984) and Barton (1989*b*).

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Here, I will concentrate on the general question of whether random genetic drift (especially, in founder events) is likely to lead to significant reproductive isolation.

(*b*) *Populations of steady size*

First, consider the effects of drift in a small isolated population which has a steady effective size of N_e diploid individuals. Quite generally, the rate at which drift knocks the population from one adaptive peak to another is proportional to $\bar{W}^{2N_e}$. Here $\bar{W}$ is the mean fitness of the population at the unstable equilibrium which separates the two peaks, relative to the mean fitness at the original equilibrium. This relation was first derived by Wright (1941), and applies whenever the system can be described by an 'adaptive landscape' (Barton & Rouhani 1987). For example, it gives a good approximation to disruptive or stabilizing selection on a quantitative trait (Lande 1985; Barton 1989*a*), selection against heterozygotes (Lande 1979; Hedrick 1981), or Wagner *et al.*'s (1994) model of epistatic selection on two loci (see below).

Now, we have seen that the reproductive isolation which is generated by a peak shift also depends on mean fitness, being proportional to $\bar{W}^{1/r}$. Hence, shifts which lead to strong isolation are necessarily unlikely. One can make the quantitative argument that because the rate of shifts decreases exponentially with $N_e\Delta\log(\bar{W})$ ($\Delta\log(\bar{W})$ being a measure of the depth of the adaptive valley), reproductive isolation will build up mainly through the gradual accumulation of shifts of magnitude $\Delta\log(\bar{W}) \approx 1/N_e$ (Walsh 1982; Barton 1989*b*).

There is an assumption here, that the mean fitness of the population which forms when divergent populations hybridize is similar to the mean fitness of the 'adaptive valley' which separates the two peaks. The former may in fact be lower, leading to stronger reproductive isolation. This distinction is crucial to models which allow strong isolation to evolve with high probability; we consider it in detail below.

(*c*) *Founder events*

Suppose now that there is a drastic bottleneck, as when a new population is founded. During this brief period, selection is negligible compared with drift, because there will be too little time for it to change allele frequencies appreciably. Therefore, the chance of a shift can be approximated by the chance that the new population will have drifted into the domain of attraction of a new equilibrium. (This approximation overestimates the chance of a shift, because selection will tend to impede divergence.)

The problem can be further simplified, because the exact sequence of population sizes during the bottleneck is irrelevant: under the diffusion approximation, the distribution of allele frequencies afterwards depends on a single parameter, which we can take to be the reduction in genetic variance, $(1\text{-}F)$. The mean of an additive quantitative trait will be normally distributed with variance $2FV_g$, where the genetic variance is reduced from V_g to $(1\text{-}F)V_g$ (Barton & Charlesworth 1984). Similarly, the distribution of allele frequency after the bottleneck depends primarily on F, and the initial frequency (figure 2). Thus, the net effect of a bottleneck can be measured by observing the loss of neutral variability: the detailed demography of the bottleneck is irrelevant, provided that drift is concentrated in a short period. Templeton has suggested that a bottleneck which involves a severe reduction in 'variance effective size', but little reduction in 'inbreeding effective size', is favourable to speciation, because it allows drift in allele frequencies and yet little loss of variation. There is a fundamental mistake here, because both drift to a new adaptive peak and loss of variation, are due to random changes in allele frequency, and cannot be disentangled. For example, if a population grows from two diploid individuals sampled from a large pool, the inbreeding effective size is much greater than the variance effective size in the first generation (∞ versus 2), because the founders are unrelated, but have a large variance in allele frequency. Figure 2 shows that for given F, the distribution of allele frequencies, and hence the chance of a shift, is almost independent of whether growth is rapid or slow, and hence of the transient discrepancy between inbreeding and variance effective sizes.

These general considerations show that for strong isolation to evolve during a founder event, there must be initial variation in traits or genes that can cause isolation by reducing hybrid fitness. Thus, founder effect speciation is unlikely in models of discrete loci where alleles are initially held at low frequency in a mutation-selection balance. This argument suggests that the most favourable cases are either balanced polymorphisms where epistatically interacting alleles are held at high frequency, or polygenic variation involving many genes. Carson (1968, 1975) has put forward verbal models in which alternative coadapted combinations of genes segregate. The simplest representation of this scheme involves two loci, with polymorphism being maintained by overdominance, and epistasis allowing two alternative equilibria which differ in the sign of linkage disequilibria (Charlesworth & Smith 1982). The expected reproductive isolation produced by this model is proportional to the non-additive variance in fitness in the base population. Although there are few direct measurements of the components of fitness variance (Burt 1995), comparisons of the effects of chromosomes extracted from male and from female *Drosophila* (which differ in that only the latter have undergone recombination show that the non-additive variance in viability and fertility is small (a few percent; Mukai & Yamaguchi 1974; Charlesworth & Charlesworth 1975). Under this model, the amount of reproductive isolation generated by a bottleneck is constrained by the standing variation in fitness associated with epistatically interacting genes. We consider below whether this is a general constraint.

Shifts are more likely to occur if they are based on the random drift of a quantitative trait with high genetic variance. If this variation is additive, we can make a general prediction about the effect of a bottleneck. In generation t, the genetic variance is

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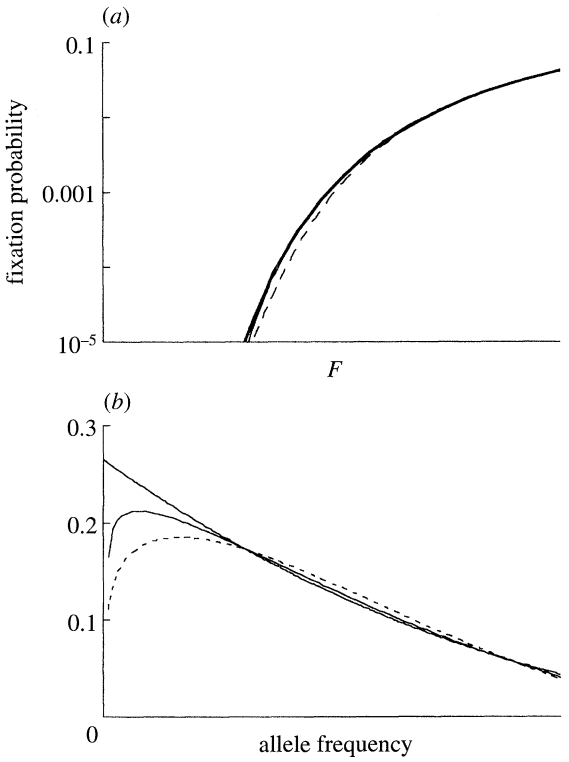

Figure 2. Accuracy of the diffusion approximation for the distribution of allele frequencies after a bottleneck. (*a*) The probability that an allele initially at frequency $p_0 = 0.05$ will be fixed after a bottleneck which causes inbreeding F. On the diffusion approximation (heavy line), the fixation probability is determined solely by F (Crow & Kimura 1970). The dashed line shows the fixation probability for a population which grows geometrically from two diploid individuals, calculated assuming Wright-Fisher sampling, for a range of growth rates. A light line shows exact results for a population growing from four diploid individuals, but is indistinguishable from the diffusion approximation. (*b*) The distribution of allele frequencies for $F = 0.555$. The heavy line gives the diffusion approximation, the light line a population increasing by 17.4% per generation, in the sequence 4, 5, 6, 6, 8, 9, 10, 12....., and the dashed line growth by 50% per generation, in the sequence 2, 3, 4, 7,....

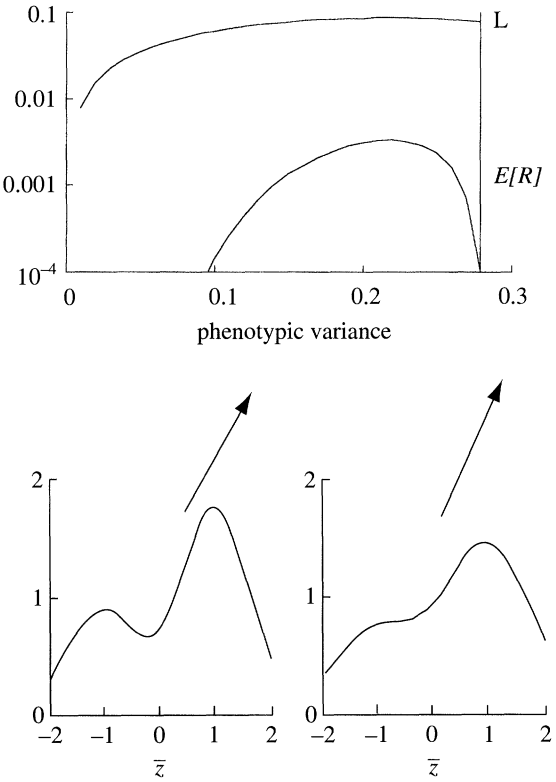

Figure 3. The relation between the expected reproductive isolation, $E[R]$, caused by an extreme bottleneck ($F = 1$), the standing genetic load, L, and the phenotypic variance, $V_g + V_e$. Disruptive selection acts on an additive trait, z, determined by infinitely many unlinked loci. Individual fitness is $W = (Ae^{-(z-1)^2/2V_s} + e^{-(z+1)^2/2V_s})$, as in Kirkpatrick (1982). There are potentially two peaks of width $\sqrt{V_s}$, one near $z = -1$, and one near $z = +1$ which is higher by a factor A. Environmental and genetic contributions to z are assumed to follow Gaussian distributions, so that mean fitness is given by the above formula, with V_s replaced by $V_s + V_g + V_e$. In this example, $A = 2$, $V_s = 0.3$, and $h^2 = V_g/V_p = 50\%$. Reproductive isolation is defined by the ratio between initial mean fitness and mean fitness in the adaptive valley, $R = -\log_e(\bar{W}_p/\bar{W}_h)$. The standing load is defined by the ratio of initial mean fitness to that in the absence of genetic variation.

reduced to $V_{t+1} = V_t(1-1/2N_t)$, and the variance of the mean around its original value increases by V_t/N. The cumulative effect of drift over many generations is to reduce the genetic variance by a factor

$$(1\text{-}F) = \prod_t (1-1/2N_t),$$

and to give a variance of the mean $2FV_0$. Thus, regardless of the exact sequence of population sizes, the mean will vary in proportion to the net inbreeding. A shift of more than 2 standard deviations, or $2\sqrt{(2FV_0)}$ is unlikely; thus, even with complete inbreeding, the mean is unlikely to shift more than $2\sqrt{2}$ genetic standard deviations.

This result puts two constraints on the likely level of reproductive isolation. First, the genetic variance must be high enough to include unfit intermediate genotypes if a shift is to be likely; this implies a standing genetic load. Second, if the phenotypic variance is too high, it is unlikely that there will be two distinct 'adaptive peaks': above a critical variance, enough individuals will approach the fittest phenotype that the population will move to that state under selection alone (Kirkpatrick 1982). In the simplest model of disruptive selection on a single additive trait, the genetic variance is constrained to a narrow range: if it is too small, the expected isolation produced in a bottleneck is very small, whereas if it is too large, there is only a single adaptive peak. Within this range, the expected reproductive isolation is smaller than the standing genetic load. In the example shown in figure 3, disruptive selection favouring optima at ± 1 only allows alternative equilibria if the phenotypic variance is less than 0.279; however, if the variance is much smaller than this threshold, the chance of a shift is small, even after an extreme bottleneck. The expected isolation is at most 3.9% of the standing load.

The discussion so far has centred on the simplest

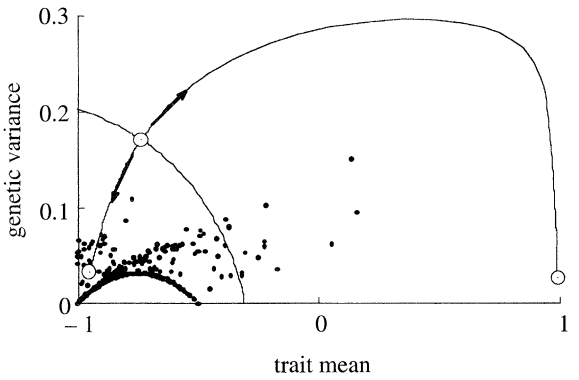

Figure 4. Shifts in an additive trait under disruptive selection. Selection acts as in figure 3, with $V_s = 0.3$, $A = 2$, $V_e = 0.1$. Mutation at a net rate $U = \Sigma\mu = 0.03$ maintains genetic variance at 16 loci, each segregating for two alleles with effect $\alpha = 0.25$. There are two stable equilibria at $\bar{z} = -0.952$, $V_g = 0.0332$, and $\bar{z} = +0.990$, $V_g = 0.0268$; these are separated by an unstable equilibrium at $\bar{z} = -0.737$, $V_g = 0.171$ (open circles). The heavy line separates the domains of attraction of the two equilibria, whilst the arrows show paths away from the unstable equilibrium. These are calculated using the rare alleles approximation. This was checked by numerical iterations of the 16 allele frequencies. These showed that the outcome is determined almost entirely by the initial $(\bar{z}, V_g)$; the domains calculated in this way are separated by a line close to that shown, but slightly flatter. The points show the outcome of 1000 bottlenecks, in which 50% of genetic variation was lost ($F = 0.5$); the distribution was calculated using the diffusion approximation. Shifts occurred in 13 cases, and resulted in reproductive isolation $R = \log_e(\bar{W}_p/\bar{W}_h) = 0.0531$. This compares with a load due to genetic variation around the initial optimum of $L = 0.0337$; $E[R]/L = 0.020$.

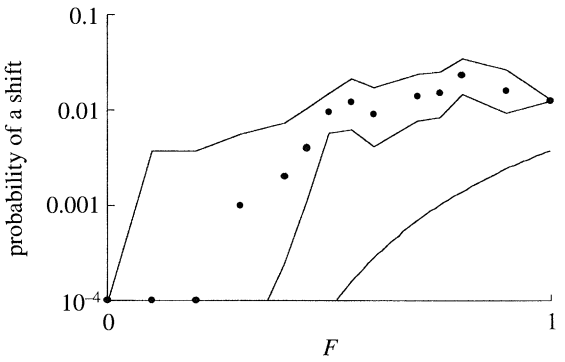

Figure 5. The probability of shifts to a new peak, for an additive trait under disruptive selection. Parameters are as in figure 4. Each values is calculated from 1000 trials, for varying F (solid circles); the thin lines show 95% confidence intervals. The maximum rate found was 23/1000 for $F = 0.8$; this corresponds to $E[R]/L = 0.035$. The curve on the lower right shows the probability of a shift calculated ignoring fluctuations in genetic variance.

model, in which random changes in genetic variance are ignored, and the trait is assumed to be additive. Relaxing these assumptions introduces considerable complications, because the outcome depends on the genetic basis of the trait, rather than on purely phenotypic arguments. First, consider fluctuations in variance. Whitlock (1995) has shown that in a bottleneck, genetic variance may by chance increase substantially. Enough individuals may then approach the fitter optimum to trigger a shift. Whitlock (1995) assumed that the distribution of allelic effects at each locus is Gaussian, and included no mechanism for maintaining the initial variation. However, if variation is maintained by mutation, it is likely that each locus is near fixation, so that variation is due to rare alleles (Turelli 1984). Figure 4 shows the evolution of the mean and variance of an additive trait, based on 16 loci, assuming the rare alleles approximation (Barton & Turelli 1987). In a population of steady size, drift is most likely to cause a shift by taking the population across the unstable equilibrium which lies between the alternative equilibria; this involves a substantial increase in genetic variance (from 0.033 to 0.171). However, a founder event is likely to cause less increase in variance (see simulated scatter in figure 4) This is because a shift is most likely to occur along the shortest route to the domain of attraction of the new state, rather than by making an excursion via the unstable state. Nevertheless, allowing for fluctuations in variance does increase the chance of a shift. For example, using the same parameters as figure 4, a shift is unlikely for $F < 0.4$, and rises to around 1% for larger F (figure 5). It is an order of magnitude larger than would be the case ignoring fluctuations in variance (curve at lower right of figure 5). However, allowing for changes in genetic variance also reduces the strength of isolation produced, because a hybrid population can recover fitness by increasing its variance to encompass the fitter phenotypes (figure 4). The expected reproductive isolation is at most 3.5% of the standing genetic load, similar to the value of 3.9% calculated neglecting fluctuations in variance.

There has been considerable interest in the observation that additive genetic variance can increase substantially following a bottleneck (Bryant *et al.* 1986, 1993, 1995). The simplest explanation is the chance increase of recessive alleles that were rare in the base population (Willis & Orr 1993), though epistasis may also contribute. The key question for founder effect speciation is whether such non-additive inheritance makes it more likely that a founder population will shift into the domain of a new equilibrium. The variance of the mean following a bottleneck is increased above the additive expectation of $2FV_g$ only if the bottleneck is strong (figure 6, from Barton 1989*b*). Moreover, the most plausible reason why recessives should initially be rare is that they are deleterious; the population would then tend to return to its original state.

5. AVOIDING THE OBSTACLES TO SPECIATION

As Darwin realized, in considering the origin of complex adaptations such as the eye, two distinct morphs may be connected by a relatively fit set of intermediates (chapt. 6, Darwin 1859). This possibility aids speciation as well as adaptation, because it allows populations to diverge to incompatible states without

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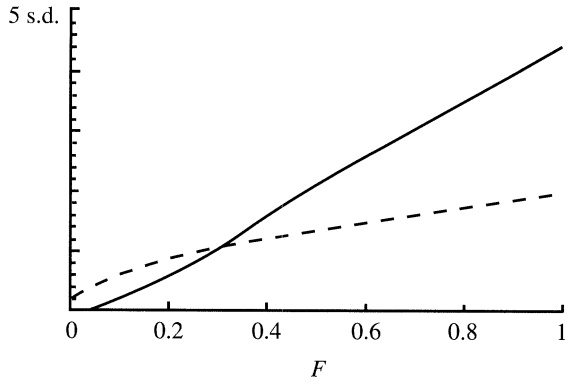

Figure 6. The largest likely trait (defined as a shift of 2 standard deviations in the mean) after a bottleneck, measured in phenotypic standard deviations. ($h^2 = 50\%$). The graphs show predictions for additive traits ($2\sqrt{(2FV_g)}$) and for traits based on recessive alleles, initially at 10%. Solid line = recessive; dashed line = additive.

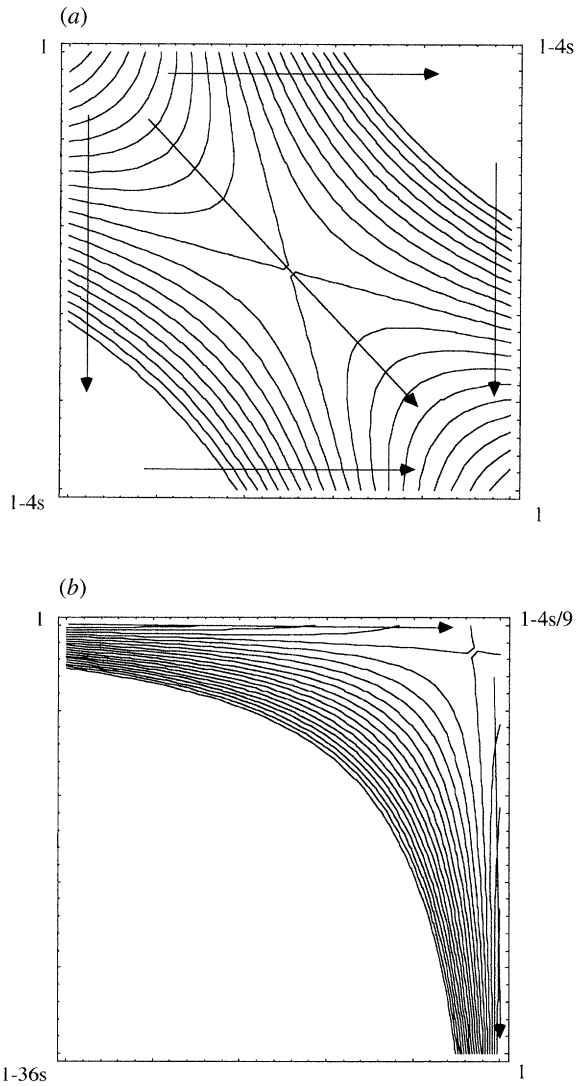

Figure 7. The mean fitness as a function of allele frequencies, for Wagner *et al.*'s (1994) model. Contours are spaced at intervals 0.05. (*a*) Stabilizing selection on an additive trait ($\beta = 0$); the F_1 has fitness 1. (*b*) Stabilizing selection on a non-additive trait ($\beta = -4/3$). The F_1 now has fitness $1-16s/9$. Arrows show the alternative paths by which a shift may occur.

suffering low fitness. It can be appreciated by contrasting F_2 or backcross hybrids with the genotypes connecting the parental populations, which (if ancestral alleles have not been lost) will be contained within the set of F_2 genotypes. On the whole, randomly produced hybrids have never been tested by selection, and may therefore have much reduced fitness; the ancestral genotypes make up a small fraction of those hybrids, and may have high fitness. Indeed, recombinant genotypes may be fitter than either parent, and may spawn 'hybrid species' if they can rise to high frequency (Rieseberg 1995). In this section, I consider how far evolution along ridges in the adaptive landscape aids the evolution of strong reproductive isolation, and whether it makes founder effect speciation more or less likely than alternative mechanisms.

Those who have invoked the need for random drift to overcome natural selection have not accepted that extant genotypes are connected by fit intermediates. Wright argued, in his theory of the 'shifting balance' (1932), that populations could not find paths of increasing fitness which would allow them to escape inferior 'adaptive peaks'. Similarly, Mayr (1942) held that 'coadaptation' would prevent significant change in large populations. The idea that obstacles to speciation can be avoided by evolution along ridges in the adaptive landscape has a long pedigree, having been proposed in a formal two-locus model by Dobzhansky (1937), and developed by (among others) Muller (1942), Bengtsson & Christiansen (1983) and Nei *et al.* (1983). It has recently received somewhat more attention; in particular, Gavrilets and Hastings (1995) and Wagner *et al.* (1994) have used it to argue that random drift may readily lead to strong reproductive isolation.

(*a*) *Do ridges in the 'adaptive landscape' favour divergence by drift?*

Two difficulties arise. First, although ridges in the 'adaptive landscape' make divergence by random drift easier, they also make divergence by selection alone more likely. This objection applies particularly where slight changes in the parameters can remove the adaptive valley altogether. In the example of figure 3, founder effects produce the greatest isolation when the phenotypic variance is close to the threshold at which the two peaks merge into one. Thus, slight changes in conditions could cause divergence with no need for random drift to oppose selection (cf. Kirkpatrick 1982). Moreover, the existence of ridges in the fitness surface aids founder effect speciation less than it aids divergence by drift in stable populations, or in response to fluctuating selection. This is because divergence in a founder event is most likely to take a direct path: the population will not have time to seek out a tortuous path of high fitness (for example, compare the ridge connecting the peaks in figure 4 with the outcome of founder events).

Wagner *et al.* (1994) present an example in which the existence of a path of high fitness aids the evolution of reproductive isolation. They consider stabilizing selection on a quantitative trait, determined by two

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loci with two alleles. If the trait is additive, the optimal phenotype can be obtained by fixing either ($U_1U_1V_2V_2$) or ($V_1V_1U_2U_2$); there are two peaks in the 'adaptive landscape' (figure 7*a*). Allowing epistasis changes the relative fitnesses of the less fit homozygotes, such that the population can shift to a new adaptive peak without passing through a deep adaptive valley. For example, with epistasis of strength $\beta = -4/3$ (the value analysed by Wagner *et al.*), the F_1 heterozygote has fitness reduced by $(1–16s/9)$, but the homozygote ($U_1U_1U_2U_2$) has fitness $(1–4s/9)$ (figure 7*b*). The interpretation in terms of stabilizing selection on a non-additive trait is not essential: the model is really one in which the location and height of the ridge can be tuned by the parameter β. With $\beta = -1$, all genotypes homozygous for U_1U_1 or U_2U_2 (or both) have maximum fitness, as in the models of Dobzhansky (1937) and Bengtsson & Christiansen (1983).

A small population can readily drift along the ridge, even when the F_1 and F_2 have low fitness (e.g. figure 7*b*); in the extreme case, the F_1 could be completely sterile, and drift could cause full speciation. However, when β is close to -1, the 'adaptive valley' is very shallow, and slight changes in selective conditions could also readily lead to isolation. This kind of model therefore favours speciation by any means, rather than by drift in particular. Moreover, this kind of model is relatively insensitive to founder effects: the initial variation is low, being sustained by mutation, and the chance of a shift depends on the domains of attraction of the alternative states, which are independent of epistasis. This is illustrated in figure 8, which shows the chances that a shift will occur following a founder event in which half the genetic variation is lost ($F = 0.5$), as a function of mutation. Unless mutation is frequent ($\mu/s > 0.1$), it is most likely that one mutation will fix first, followed by evolution up to the new peak (see arrows in figure 7). (The same is true for drift in a stable population: Wagner *et al.* (1994) found that the rate of shifts scales with μ rather than μ^2.) The rate is 4.5-fold higher for the non-additive model; however, this is solely because the selection against one allele is weaker along the ridge, allowing it to become more common. This contrasts with a steady population, where the existence of a ridge gives an overwhelming advantage to the non-additive model when selection is strong (Wagner *et al.* 1994).

Gavrilets & Hastings (1995) also discuss a variety of models in which drift can lead to strong isolation, as measured from first generation hybrids. One class of their models relies on ridges in the landscape, and is similar to Wagner *et al.*'s (see figures 1 & 5 therein). However, they introduce another class of model, in which divergence is due to the accumulation of mutations which arise after the founder event. For example, they suppose that at one locus, polymorphism is maintained by symmetric overdominance. A second locus is fixed for an allele which is favoured when associated with heterozygotes at the first locus. However, if the polymorphism is lost in a founder event, a new allele is favoured at the second locus; if this is fixed, it can lead to strong isolation. This model is close to Mayr's proposal that different alleles can become favourable in the homozygous state that may follow a founder event. However, it is hard to see why the changed genetic background that follows a founder event should be more likely to trigger divergence than changes due to any alteration in gene frequencies. For example, the analogue of Gavrilets & Hastings' (1995, figure 3*b*) model would be one where a new allele at the second locus is favoured following a substitution (for whatever reason) at the first.

(*b*) *Are ridges in the adaptive landscape consistent with strong isolation?*

The second difficulty is that the fit intermediates may be reconstructed after hybridization, leading to the collapse of reproductive isolation. This process can be observed directly. For example, F_1 males from the cross between *Chorthippus parallelus parallelus* and *C. p. erythropus* are sterile, yet natural hybrids are fertile (figure 9, from Virdee & Hewitt 1994). The mechanism by which hybrid fitness can be recovered is understood in the case of chromosome rearrangements. For example, races of the shrew *Sorex araneus* have mainly metacentric karyotypes. Because these involve different chromosome combinations, meiosis is severely disrupted in F_1 hybrids. However, in natural hybrid populations, acrocentric chromosomes are found at high frequency; because simple heterozygotes between fused and unfused chromosomes segregate regularly, there is little loss of fertility, and hence little barrier to gene flow (Searle 1986).

This difficulty can be avoided in two ways. First, ancestral alleles may have been lost: in the *Sorex* example, hybrid fertility is ameliorated by acrocentric chromosomes which are not found elsewhere in the species and might in principle not have been regenerated by mutation. Second, if divergence has gone so far that the initial hybrids are very unfit, then it may not be possible to establish fit recombinants: in the limit where F_1 hybrids are completely infertile or inviable, speciation is complete. As discussed above, the outcome depends on how the divergent populations meet. A trickle of gene flow is less likely to allow the

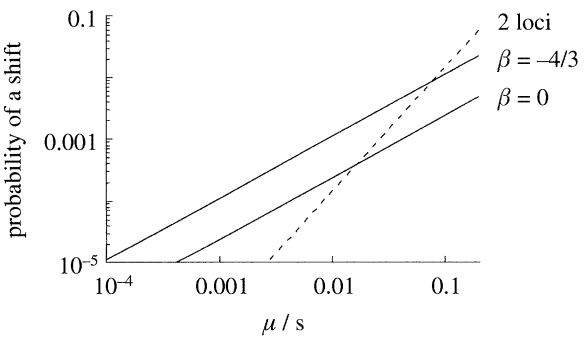

Figure 8. The chance that a founder event ($F = 0.5$) will trigger a peak shift in Wagner *et al.*'s (1994) model, plotted against the ratio between mutation and selection (μ/s). The dotted line shows the chance that both loci fix the rare allele; this is the same for $\beta = 0$, $\beta = -4/3$. The two solid lines show the chance that one locus will fix, followed by a shift at the second locus. This is higher for the case $\beta = -4/3$.

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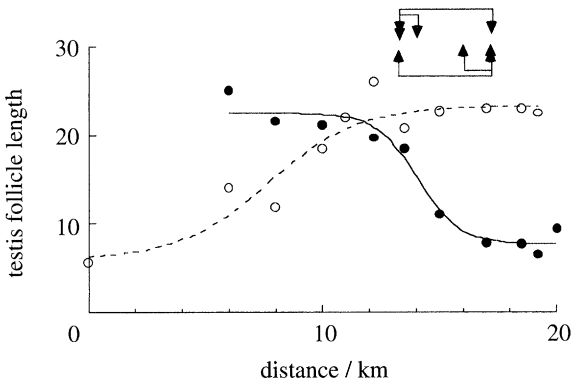

Figure 9. Male fertility in crosses between grasshoppers from the hybrid zone between *Chorthippus parallelus parallelus* and *C. p. erythropus*, as measured by testis follicle length (data from Virdee & Hewitt 1994). Filled circles show the fertility of sons from mothers crossed to *C. p. parallelus* fathers, plotted against the position from which the females were collected; open circles are for crosses to *C. p. erythropus* fathers. Females from the centre of the hybrid zone give fertile sons for both sires.

establishment of fit recombinants than free hybridization in a continuous hybrid zone; however, even there, strong selection can prevent the establishment of particular genotypes. There is, however, a chance that drift in a partly isolated hybrid population can establish a fit recombinant, perhaps leading to 'hybrid speciation' (McCarthy *et al.* 1995; Rieseberg 1995). Because hybrid populations may have an exceptionally high standing load and high non-additive genetic variance, these may be the most favourable sites for founder-effect speciation.

6. CONCLUSIONS

There are strong theoretical constraints on the degree of reproductive isolation which is likely to be produced by a sudden founder event. The genetic variation present in the population must be reshuffled so as to substantially reduce the fitness of hybrids with other populations. Hence, the expected degree of isolation is limited by the initial non-additive variation in fitness, and the standing genetic load. It may be that evolution is facilitated by the existence of sequences of fit genotypes. However, these are more favourable to selective mechanisms of divergence than to founder effects, where drift overrides selection. The fit intermediates may also be reconstructed if diverging populations meet before speciation is complete, leading to the breakdown of isolation.

These theoretical arguments suggest what kinds of evidence are relevant. The effect of a bottleneck depends primarily on the fraction of genetic variance which is lost (F). Although founder effects can cause a loss of allozyme and DNA variation (e.g. Baker & Moeed 1987; Janson 1987), there is no evidence of a drastic reduction in the classic example of the Hawaiian *Drosophila* (Sene & Carson 1977; De Salle & Templeton 1988). The way genes interact to determine fitness can be investigated by examining the inheritance of fitness in the base population and in species crosses. Haldane's Rule and the large effects of the sex chromosomes indicate that isolation is due to interactions between recessive alleles (Turelli & Orr 1995) ; the dissection of hybrid fitness in *Drosophila* is just beginning to disentangle the relative fitnesses of recombinant genotypes and to isolate the genes involved (Wu & Palapoli 1993). The strongest evidence, however, is the most straightforward: bottlenecks induce little reproductive isolation in artificial populations of *Drosophila* (Rice & Hostert 1993). Indeed, artificial selection can produce divergence at least as easily as drift (Cohan & Hoffmann 1989; Cohan *et al.* 1989), and the classic examples of speciation on islands involve adaptive radiation in response to a novel physical and biotic environment (Grant 1986; and see papers by Clarke *et al.*, Grant & Grant, Schluter and Thorpe & Malhotra, this volume).

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Discussion

J. Mallet (*Galton Laboratory, University College London, U.K.*) It seems to me that your measure of reproductive isolation includes only postmating isolation. Do your results on the improbability of speciation in founder populations change when you consider premating isolation? Coexistence in sympatry is the only good test of results of speciation; and $\bar{W}$hybrids/$\bar{W}$pure tells one extremely little about whether coexistence will occur.

N. Barton. This question raises two issues. First, how much does premating isolation reduce gene flow? Divergence in mating system can lead to alternative stable equilibria in the same way as does postmating isolation; indeed, narrow hybrid zones often involve sexually selected characters (Barton & Hewitt 1985). If the dynamics of such divergence can be described by a potential function analogous to $\bar{W}$, then strength of the barrier to gene flow will be proportional to $\bar{W}^{1/r}$; the effective rate of recombination r will be reduced in so far as non-random mating reduces heterozygosity. Thus the arguments over the plausibility of founder effect speciation apply to premating isolation as well as postmating, at least qualitatively. The second issue concerns coexistence in sympatry. This requires both sufficient assortative mating, and divergence into different ecological niches (i.e. the use of different limiting resources, such that the rarer genotypes gain an advantage). It is much harder to say anything general about how likely founder events are to trigger such ecological divergence than about their effect on gene flow. If frequency dependent selection maintains abundant polymorphism in response to diverse resources, then founder effects could more easily lead to sympatry. However, the novel ecological conditions experienced by a colonizing population seem to me more important than random drift.

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