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Adaptation and the 'shifting balance'

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Summary

Wright proposed that there is a 'shifting balance' between selection within demes, random drift, and selection between demes at different 'adaptive peaks'. We investigate the establishment and spread of new adaptive peaks, considering a chromosome rearrangement, and a polygenic character under disruptive selection. When the number of migrants (Nm) is small, demes fluctuate independently, with a bias towards the fitter peak. When Nm is large, the whole population can move to one of two stable equilibria, and so can be trapped near the lower peak. These two regimes are separated by a sharp transition at a critical Nm of order 1. Just below this critical point, adaptation is most efficient, since the shifting balance greatly increases the proportion of demes that reach the global optimum. This is so even if one peak is only slightly fitter than the other ($\Delta\bar{W} \approx 1/N$), and for both strong and weak selection ($Ns \ll 1$ or $Ns \gg 1$). Provided that Nm varies sufficiently gradually from place to place, the fitter peak can be established in regions where $Nm \approx 1$, and can then spread through the rest of the range. Our analysis confirms Wright's argument that if selection, migration and drift are of the same order, the 'shifting balance' leads to efficient evolution towards the global optimum.

1. Introduction

Wright's (1931, 1932) theory of the 'shifting balance' has received considerable attention, both as a mechanism for establishing new adaptations, and as a process of divergence. Wright illustrated his theory by means of the 'adaptive landscape': a graph of mean fitness against allele frequencies, or any other set of characteristics which describe the state of the population (see Provine, 1986, and Wright, 1988, for differing interpretations). The complex relation between fitness and genotype makes it likely that this surface has many peaks and valleys. Selection tends to increase mean fitness, and so will push a population uphill, towards some local optimum. Wright (1931, 1932) pointed out that stochastic fluctuations such as random sampling drift can cause a shift between such local peaks, allowing the population to evolve towards the global optimum.

Wright saw the shifting balance primarily as leading to efficient adaptation, despite the difficulty inherent in optimization across a rugged surface. The question on which we concentrate in this paper is whether the shifting balance will indeed lead to a significant bias in favour of 'superior' peaks. In a following paper, we will extend the treatment to find whether such a bias may be caused by group, as well as individual, selection. Before developing a general analysis of selection, migration and drift in Wright's island model, we first comment on the kinds of selection and population structure required for the 'shifting balance' to operate.

Evolution does not necessarily increase the mean fitness. This is, for instance, the case where the effects of mutation are included. When gene flow, frequency-dependence, recombination and linkage disequilibrium are taken into account, the dynamics of a population cannot be described by any general optimization principle (Akin, 1979; Hastings, 1981). However, the population will be pushed towards one or other of a set of simple equilibria, limit cycles, or chaotic attractors. It may be that stochastic transitions between these alternative states are important in evolution towards a well adapted state, and in the divergence of populations into reproductively isolated species. For simplicity, we will use the term 'adaptive peak', while recognizing that the process may generally involve the establishment and spread of alternative attractors.

Once a higher peak is reached by a single deme or

a small group of demes, it must then spread to the rest of the population. This may occur in three ways. First, whole demes might go extinct, and be replaced by colonists from demes in a different state. Second, gene flow between demes can overcome selection, and cause the certain spread of the new peak. Finally, the influx of migrants from demes carrying a different peak makes it more likely that the deme will shift to that peak as a result of random fluctuations.

Lande (1979) showed that if the probabilities of extinction and colonization are independent of the state of the deme, then the rate at which new peaks are established throughout the whole population is the same as the rate of establishment in each deme. (This result rests on the assumption that empty sites are colonized by individuals from only a single deme.) There is a precise analogy with the neutral theory, in which the rate of establishment of neutral mutations in a population equals the mutation rate. If one adaptive peak causes a lower rate of extinction, or a higher rate of colonization, then that peak will tend to spread. Such interdemic selection can greatly increase the chance of fixation of an underdominant chromosome mutation, and can greatly reduce its expected time to fixation (Lande, 1985). Note that here, interdemic selection is the only factor which introduces a bias in favour of one or other peak: in contrast with the case developed in this paper, the relative fitness of genotypes within demes has no direct influence.

Wright (1931, 1932) suggested that interdemic selection may allow the *deterministic* spread of a new gene or combination of genes. If emigration from those demes which have attained the higher adaptive peak is sufficiently strong relative to selection, it can pull neighbouring demes to the new state. This does not require that demes at fitter peaks send out more migrants, as Wright assumed. Crow *et al.* (1990) show that very low rates of migration can overcome selection, even when the new adaptive peak consists of fixation for a specific gene combination that loses its advantage when broken up by recombination. Barton (1992) argued that this phenomenon reflects the power of migration relative to selection, and is not due to the higher fitness of the new peak: even deleterious gene combinations can be established by a moderate rate of immigration. Thus, a better-adapted peak can only spread differentially by this mechanism if it causes a substantial increase in the number of emigrants, or if it has a relatively large domain of attraction. (The domain of attraction is relevant here, because the outcome depends on whether the mixed population produced by migration will then move towards the new peak.) Moreover, gene flow must initially be low enough for the new peak to be established, but must later be high enough for it to spread deterministically. These conditions can be satisfied with a constant population structure, if the new peak has a sufficient advantage over the old. For example, drift can establish a new peak in some region of an essentially continuous two-dimensional population; if this peak has a sufficient advantage, and the region is sufficiently large, then it can spread deterministically through the whole population (Rouhani & Barton, 1987; Barton & Rouhani, 1991).

A new adaptive peak can also spread through a sequence of stochastic transitions, in which migration from demes in the new state triggers shifts in neighbouring demes. Lande (1985) compared this process with random extinction and recolonization. Lande took as his example the spread of an underdominant chromosome rearrangement through the island model, or through a one-dimensional circle of demes. In Lande's model, the new arrangement gains an advantage in two ways. First, its increased fitness as a homozygote reduces the threshold frequency above which it will be fixed, and so makes it more likely that neighbouring demes will shift to the new state by chance. Second, demes fixed for the new homozygote are fitter, and so may send out more migrants. This is a form of group selection; it makes demes fixed for the new arrangement more likely to trigger a stochastic shift, and also more likely to colonize empty sites. Lande concluded that the new karyotype is more likely to gain an advantage through individual than through group effects; however, his conclusion will only hold when spread by stochastic transitions is frequent relative to extinction/recolonization.

We concentrate here on spread by a series of stochastic transitions, and examine two simple cases: fixation of an underdominant chromosome rearrangement, and the shift of a polygenic character under disruptive selection. Both can be described by movement up a landscape, though this is not simply one of mean fitness. We begin by describing a general method for deriving the distribution of allele frequencies, or of a quantitative character, in the island model, and derive approximations which hold either when most demes are near one or other adaptive peak, or when selection is weak relative to drift within demes. We show how, with moderate numbers of migrants, a slight asymmetry between the peaks can be greatly amplified: however, if the number of migrants (Nm) rises above a critical value, then the new peak cannot become common.

2. The method

We consider the island model, in which each of many demes exchanges migrants with a common pool. For a given state of the migrant pool, the diffusion approximation can be used to follow the evolution of the distribution of allele frequencies across demes. At any instant, the allele frequency in the migrant pool, $\bar{p}$, must equal the mean of this distribution, $\langle p \rangle$. In general, $\bar{p}$ is weighted by the contribution of each deme, which may in turn be a function of its genetic composition. For simplicity, we assume here that the

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number of migrants contributed by each deme is independent of genotype frequencies (i.e. selection is soft, and acts entirely within rather than between demes).

One can write down the multivariate distribution of states of the whole collection of demes. However, we show in Appendix 1 that as the number of demes becomes large, the mean across demes, which determines the migrant pool, converges to a definite equilibrium value. Thus, the solution in the limit of an infinite number of demes can be found by solving a pair of equations: a diffusion equation for the distribution of allele frequencies (given the state of the migrant pool) and a (trivial) equation relating the state of the migrant pool to this distribution. Although the state of each deme fluctuates randomly, the distribution across a large ensemble of demes changes deterministically, and converges to a fixed equilibrium.

In general, such equations are intractable. However, the equilibrium can be found from Wright's (1937) distribution. This requires the assumption that frequency-dependence and linkage disequilibria are negligible, so that the effects of migration, mutation, selection and drift can be described by a potential function, proportional to the mean fitness. The same method gives the distribution of the mean of a quantitative character, provided that the genetic variance is constant (Lande, 1976). In a single population, this is a good approximation if large numbers of loci are involved, and if selection is weak enough relative to recombination that linkage disequilibrium can be neglected. A more troublesome requirement is that migration must not increase the genetic variance. We return to this question in the discussion.

The *dynamics* of the island model can also be solved if demes spend most of their time near one or other adaptive peak. The system then evolves on two timescales. The distribution rapidly settles into one which is clustered around the adaptive peaks; the system can then be described by the proportion of demes at the various peaks, which will change slowly as a result of occasional shifts between peaks. The increase in the frequency of the different adaptive peaks can then be followed in the same way as the change in frequency of different alleles within a deme. This approximation was used by Lande (1985) to model the spread of a new chromosome arrangement through a set of islands, or through a one-dimensional chain.

3. A polygenic character under disruptive selection

Consider a continuous trait, with mean z; the additive genetic variance, v, and the phenotypic variance, V, are assumed to be constant. Disruptive selection acts such that the log mean fitness is:

$$\ln(\bar{W}) = -\frac{s}{8\Omega^4}\left((\Omega^2-z^2)^2+\frac{4\alpha\Omega}{3}(z-2\Omega)(z+\Omega)^2\right). \quad (1)$$

This quartic polynomial has peaks at $z=-\Omega$ and $+\Omega$, separated by a valley at $-\alpha$. The left-hand peak has log mean fitness $(2s\alpha/3)$ lower than the right-hand peak. The drop from the inferior peak down to the valley is $s(1-\alpha)^3(1+\alpha/3)/8$; this is the barrier that must be overcome by random drift if the population is to shift from the lower to the higher peak. The mean fitness as a function of the population mean derives from the relation between individual fitness and phenotype, x. When selection is weak (as we will assume throughout), and the character follows a Gaussian distribution with phenotypic variance V, the individual fitness is:

$$W(x) = -\frac{s}{8\Omega^4}\left((\Omega^2-x^2)^2-6V(\Omega-x)^2+3V^2 + \frac{4\alpha\Omega}{3}((x-2\Omega)(x+\Omega)^2-3Vx)\right). \quad (2)$$

[One can easily see that eqn (2) leads to eqn (1), with the approximation that $\ln(E[\exp(-sf(x))]) \approx -sE[f(x)]$, where $E[\,]$ is the expectation; see Rouhani & Barton, 1987. It is convenient to choose a simple form for $\bar{W}$, rather than for W.] Equation (1) is the same fitness function as was used by Barton & Rouhani (1991), except that the location of the peaks $(\pm\Omega)$ has been introduced explicitly, rather than being scaled to ± 1. The coefficient s differs by a factor 4 from the model used by Rouhani & Barton (1987), in order to allow direct comparison with underdominance.

We assume that the size of each deme is fixed, and independent of its genetic composition; a constant fraction m is exchanged with the migrant pool in every generation. Since we assume that selection is weak, the mean can be approximated by a continuous function of time, $z(t)$. The expected rate of change of the mean under selection and gene flow is:

$$\frac{\partial z}{\partial t} = v\frac{\partial \ln(\bar{W})}{\partial z} - m(z-\bar{z}). \quad (3a)$$

These deterministic dynamics can be described by a single potential function, which combines the effects of selection and gene flow (Rouhani & Barton, 1987):

$$\frac{\partial z}{\partial t} = v\frac{\partial U}{\partial t}, \quad \text{where } U = \ln(\bar{W}) - \frac{m}{2v}(z-\bar{z})^2. \quad (3b)$$

Sampling drift causes random fluctuations in z, with variance v/N, where N is the effective population size. Adding these gives a stochastic differential equation for z. The equilibrium distribution of z across demes, conditional on the mean among migrants, $\bar{z}$, is (Gardiner, 1983):

$$\psi(z|\bar{z}) = C\exp(2NU) = C\bar{W}^{2N}\exp\left(-\frac{Nm}{v}(z-\bar{z})^2\right). \quad (4)$$

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(C is a normalization constant, chosen so that the distribution integrates to 1.) In the absence of migration, $\psi(z|\bar{z})$ would follow Wright's stationary distribution, and would be clustered around the adaptive peaks. Migration introduces a Gaussian factor, which pulls the distribution towards the mean of the migrant pool, $\bar{z}$.

The mean of z across the whole ensemble, which we denote by $\langle z \rangle$, can be found by integrating $z\psi(z|\bar{z})$, using eqn (4). The crucial step is to note that in our model, $\langle z \rangle$ must equal the mean in the migrant pool, giving the equation $\langle z \rangle = \bar{z}$. This can be solved to find the joint mean at equilibrium, and hence the whole distribution. In Appendix 1, we show how this solution is approached as the number of demes becomes large.

(i) *Multiple stable states above a critical rate of gene flow*

The population evolves in two qualitatively different ways, depending on the number of migrants exchanged per generation. When Nm is small, the mean of the distribution, $\langle z \rangle$, depends only weakly on the mean of the migrant pool, $\bar{z}$. The slope of the graph of $\langle z \rangle$ against $\bar{z}$ is shallow, and so there is only one equilibrium (Fig. 1*a*; $Nm = 0{\cdot}5\,(v/\Omega^2)$, $\alpha = 0{\cdot}01$). The demes fluctuate independently of each other, and approach one or other peak. The frequency of the alternative peaks approaches an equilibrium ratio, with an intermediate value which somewhat favours the fitter peak. The graph becomes steeper as Nm increases, and so two other equilibria appear (Fig. 1*b*; $Nm = (v/\Omega^2)$, $\alpha = 0{\cdot}01$). The set of demes will be predominantly near either one or the other adaptive peak; this is because migration is now strong enough that the system behaves as one. Note that when the population is in *either* state, individual demes can still shift stochastically from one peak to the other, and the equilibrium distribution still has two peaks (Fig. 2*c*). However, when most demes are at one of the two peaks, migration is common enough to make shifts away from that peak unlikely.

When they exist, the two outer equilibria are stable, whilst the intermediate equilibrium is unstable. This is shown in Appendix 1, by finding the distribution with n demes, and taking the limit of large n. We will also confirm our intuition below, by using the approximation that the demes are almost always close to one or other peak. This will hold when selection is strong relative to drift ($Ns \gg 1$).

As the advantage of the superior peak increases, demes are more likely to be found in its vicinity; above some threshold, the lower two equilibria disappear, leaving a single equilibrium in which the higher peak predominates. This threshold is shown in Fig. 1*c* ($Nm = (v/\Omega^2)$, $\alpha = 0{\cdot}022$ for $Ns = 30$). Thus, there are two qualitatively different, and sharply separated, regimes: with high asymmetry and low gene flow, there is a single equilibrium, whilst with low asymmetry and high gene flow, there are two stable equilibria. Before developing approximations that will give us a better analytic understanding of the process, we briefly discuss the efficiency of adaptation in these two regimes.

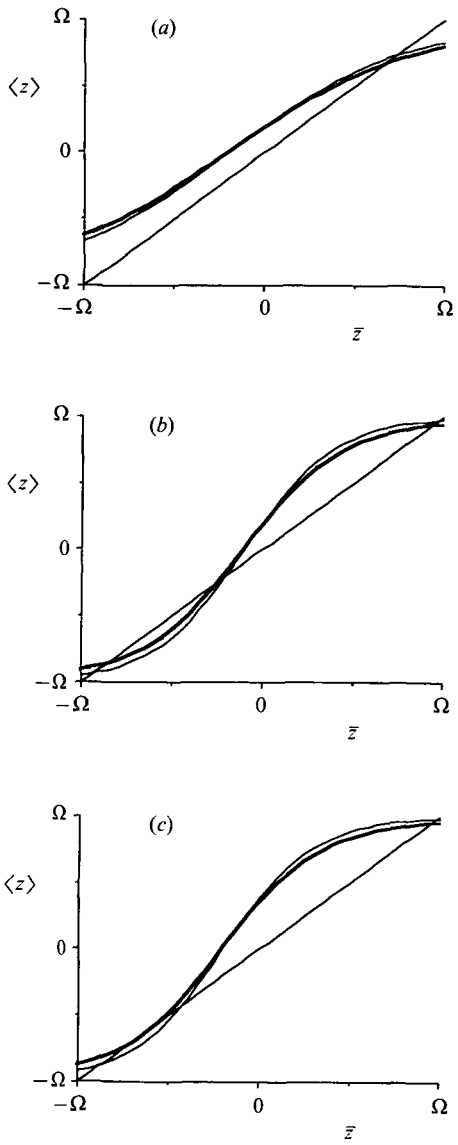

Fig. 1. (*a*) Graph of the mean across demes ($\langle z \rangle$) as a function of the mean in the migrant pool ($\bar{z}$), for disruptive selection on a polygenic character. Here, the number of migrants is $Nm = 0{\cdot}5\,(v/\Omega^2)$, and the asymmetry is $\alpha = 0{\cdot}01$; $Ns = 30$. There is a single equilibrium, given by the intersection with the diagonal, $\langle z \rangle = \bar{z}$. (*b*) When the number of migrants increases to $Nm = (v/\Omega^2)$, two new equilibria appear. (*c*) When asymmetry is increased above $\alpha = 0{\cdot}02$, there is again only a single equilibrium, biased towards the fitter peak. In each graph, the heavy curve gives the exact $\langle z \rangle$, calculated by numerical integration, and the light curve gives the Gaussian approximation, neglecting terms of order m/s. This is the approximation which gives eqn (8*a*); the more accurate approximation of eqn (8*b*) is indistinguishable from the exact curve.

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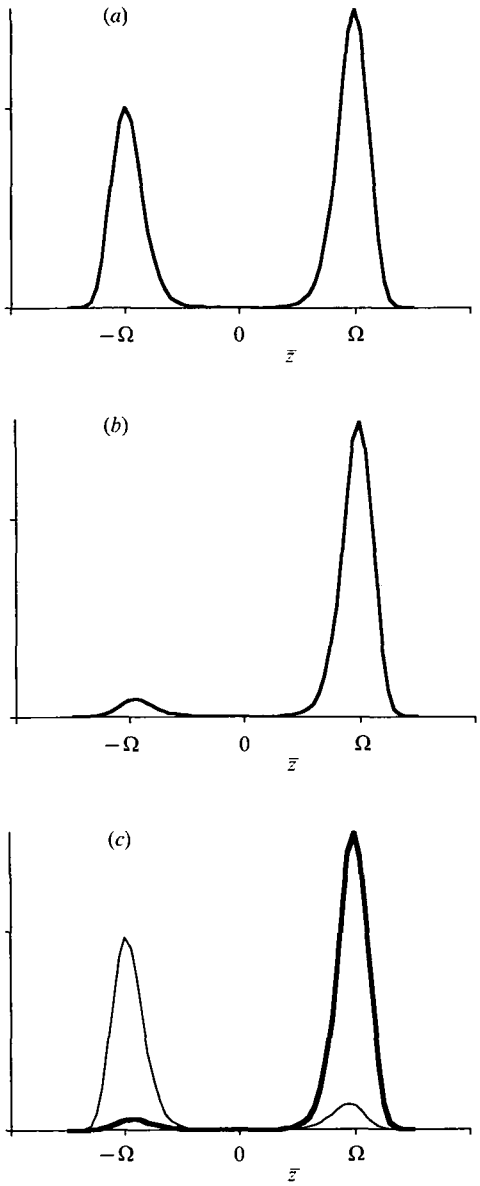

Fig. 2. (*a*) The equilibrium distributions with no gene flow, and asymmetry $\alpha = 0{\cdot}01$; there is a slight bias to the higher peak. (*b*) When the number of migrants is just below the critical point ($Nm = 0{\cdot}7\,(v/\Omega^2)$, Nm,crit = $0{\cdot}74\,(v/\Omega^2)$), there is a much stronger bias. (*c*) Above the critical point ($Nm = 0{\cdot}8\,(v/\Omega^2)$), there are two alternative stable equilibria: one is given by the heavy curve, the other by the light curve. The distribution is given by eqn (4), with $\bar{z}$ given by the solution to eqn (8*a*).

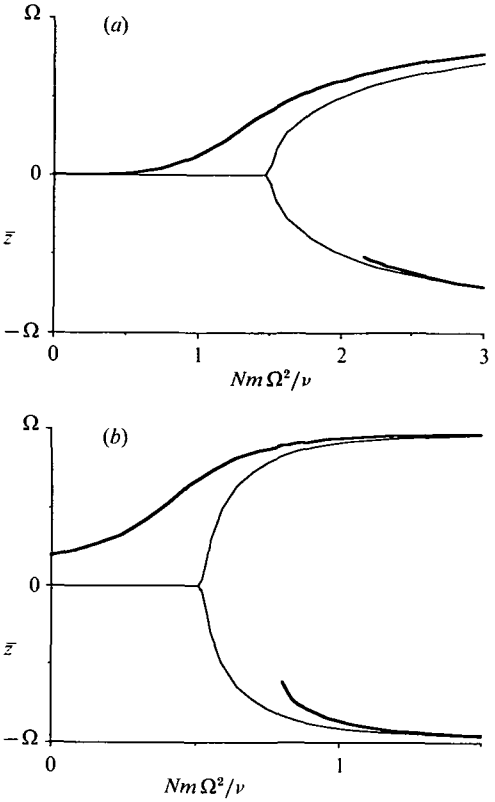

Fig. 3. The mean at equilibrium ($\langle z \rangle = \bar{z}$), as a function of the (scaled) number of migrants ($Nm\Omega^2/v$), for disruptive selection on a quantitative character. (*a*) The limit of weak selection [$Ns \ll 1$; eqn (10)]. The light curve gives the symmetric case, where the critical number of migrants is $Nm_{\text{crit}} = 1{\cdot}50\,(v/\Omega^2)$. The heavy curve is for asymmetry $\alpha = 0{\cdot}1$; the critical number of migrants is then $Nm_{\text{crit}} = 2{\cdot}17\,(v/\Omega^2)$. (*b*) Strong selection ($Ns = 30$), calculated using the approximation of eqn (8*b*). The light curve is the symmetric case [$Nm_{\text{crit}} = 0{\cdot}539\,(v/\Omega^2)$ exactly, and $0{\cdot}536\,(v/\Omega^2)$ from eqn (8*b*)], whilst the heavy curve is for asymmetry $\alpha = 0{\cdot}01$ [$Nm_{\text{crit}} = 0{\cdot}804\,(v/\Omega^2)$ exactly, and $0{\cdot}801\,(v/\Omega^2)$ from eqn. (8*b*)].

(ii) *Adaptation is most efficient at intermediate levels of gene flow*

In the absence of gene flow, the demes will fluctuate independently between the two peaks, and will approach an equilibrium distribution in which the fitter peak is more common; the ratio between the frequencies of the peaks is approximately $(\overline{W}_2/\overline{W}_1)^{2N} = \exp(4Ns\alpha/3)$ [eqn (4); Fig. 2*a*]. Because the fitter peak is commoner, the mean in the migrant pool will be biased towards it; thus, as the number of migrants increases, it becomes still more common, further enhancing the bias produced by migration.

Consider, for example, the bias caused by a slight asymmetry, $\alpha = 0{\cdot}01$. This corresponds to a fitness difference between the peaks which is only $16\alpha/((1-\alpha)^3(3+\alpha)) = 5{\cdot}3\,\%$ of the depth of the valley separating them. Fig. 2 shows the distributions across the demes for increasing migration, for selection strong relative to drift ($Ns = 30$). Fig. 3*b* is a graph of the population mean at equilibrium, as a function of the scaled number of migrants ($Nm\Omega^2/v$), for the same strong selection. The light line shows the symmetric case $\alpha = 0$, whilst the heavy line shows the effect of a slight asymmetry, $\alpha = 0{\cdot}01$. With no gene flow, this asymmetry would give a mean over the whole population of $0{\cdot}20\Omega$ (Fig. 2*a*, and left side of Fig. 3*b*). This bias increases with the number of migrants, to $0{\cdot}90\Omega$ when $Nm\Omega^2/v = 0{\cdot}7$ (Fig. 2*b*). However, when gene flow rises above a threshold at $Nm = 0{\cdot}74\,(v/\Omega^2)$, another stable equilibrium appears (Fig. 2*c*). Now, if the majority of demes are near the inferior peak, migration makes it unlikely that the superior peak can

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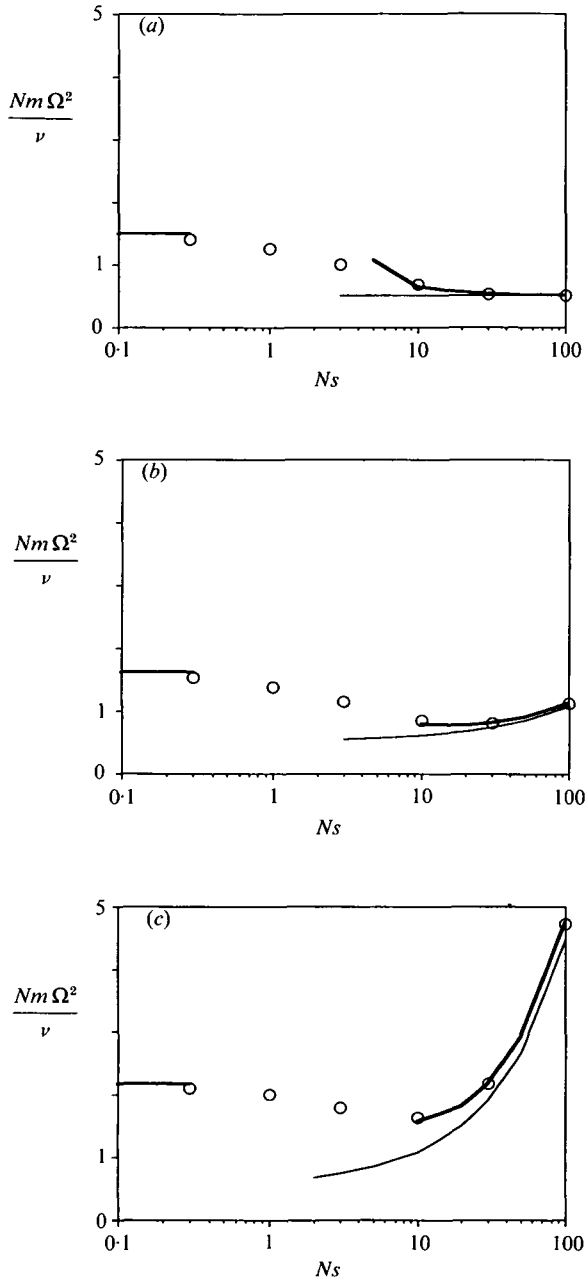

Fig. 4. The critical number of migrants, scaled as $Nm_{crit}\Omega^2/v$, as a function of the strength of selection Ns. (*a*), (*b*), (*c*) are for $\alpha = 0$, 0·01, 0·1 respectively. The circles give exact calculations, derived by numerical integration of eqn (5). The horizontal line on the left is derived from the approximation for small Ns [eqn (10)], whilst the light line on the right is from the Gaussian approximation for large Ns [eqn (8*a*)]. The heavy curve on the right is from the more accurate approximation which includes terms of order $1/Ns$ [eqn (8*b*)]. This breaks down at Ns less than around 10.

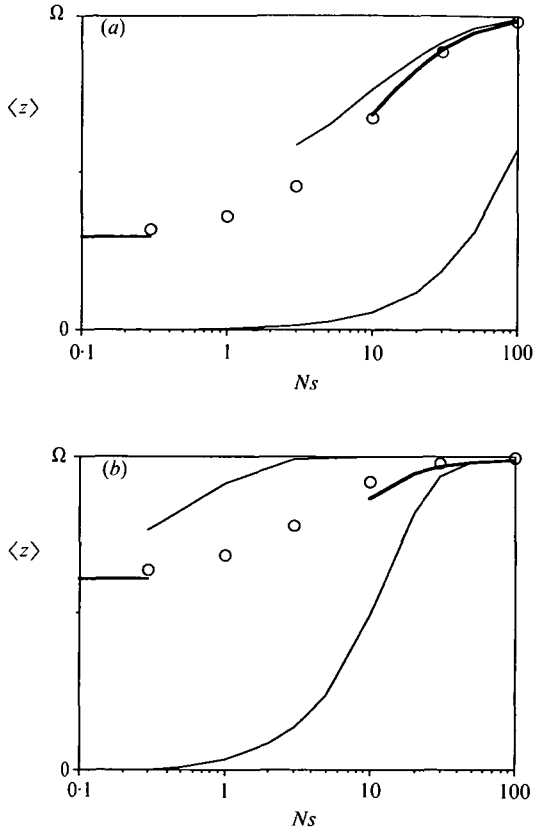

Fig. 5. The mean over all demes ($\bar{z} = \langle z \rangle$) at the critical point Nm_{crit}, as a function of the strength of selection, Ns, for asymmetry; (*a*) $\alpha = 0{\cdot}01$, (*b*) $\alpha = 0{\cdot}1$. This is the greatest bias towards the fitter peak which can be achieved by a population which starts at the lower peak. Symbols are as for Fig. 4. The light curve (lower right) is the mean which would be achieved with no gene flow.

be established. Adaptation is therefore most efficient when Nm is just below the critical point.

Fig. 4 shows this critical number of migrants as a function of selection (Ns); the circles give exact results, whilst the solid lines give various approximations (discussed below). As asymmetry increases (Fig. 4*a–c*), the critical number of migrants also increases. However, it is remarkable that this number is of the same order over the whole range of selection and asymmetry; it becomes large only for moderate asymmetry and very strong selection (Fig. 4*c*), in which case peak shifts are extremely rare (Fig. 6).

The overall mean at this critical migration rate is shown in Fig. 5; this is the largest possible bias towards the fitter peak which can be achieved by a population which starts at the lower peak. This is compared with the overall mean in the absence of gene flow. The bias increases with selection, and approaches Ω even for slight asymmetry ($\alpha = 0{\cdot}01$, Fig. 5*a*) when Ns becomes greater than 30. However, this bias will be approached very slowly when selection is strong, since peak shifts are rare. The improvement caused by the 'shifting balance' is more impressive for weak selection: even in the limit of small Ns, when selection within demes is ineffective, gene flow allows selection to act on the whole ensemble, and can produce a strong bias to the fitter genotype without the population being trapped on a suboptimal peak (left of Fig. 5*a*, *b*).

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(iii) *A general expression for the critical migration rate*

When gene flow is lower than the critical value, a change in the mean of the migrant pool, $\bar{z}$, has little effect on the mean of the distribution, $\langle z \rangle$: the slope of the graph of $\langle z \rangle$ against $\bar{z}$ is less than 1, so that it intersects the diagonal, $\langle z \rangle = \bar{z}$, once, and there is only one equilibrium (Fig. 1*a*). The critical rate of gene flow, above which there are two stable states, occurs when $\partial\langle z \rangle/\partial\bar{z} = 1$ at $\langle z \rangle = \bar{z}$ (Fig. 1*c*). Now, from eqn (4), the mean is:

$$\langle z \rangle = \int z\psi(z)\,dz = \frac{\int z\,\bar{W}^{2N}\exp\left(-\frac{Nm}{v}(z-\bar{z})^2\right)dz}{\int \bar{W}^{2N}\exp\left(-\frac{Nm}{v}(z-\bar{z})^2\right)dz}. \quad (5)$$

Differentiating eqn (5) with respect to $\bar{z}$ shows that $\partial\langle z \rangle/\partial\bar{z}$ is proportional to the variance of z across demes, $\mathrm{var}(z)$:

$$\frac{\partial\langle z \rangle}{\partial\bar{z}} = 2\frac{Nm}{v}\mathrm{var}(z). \quad (6)$$

(It is important to distinguish the between-deme variance, $\mathrm{var}(z)$, from the additive genetic variance within demes, v.) Setting $\partial\langle z \rangle/\partial\bar{z} = 1$, we find that the critical number of migrants is $Nm_{\mathrm{crit}} = v/(2\,\mathrm{var}(z))$. This is half the ratio between the genetic variance within populations (v), and that between populations ($\mathrm{var}(z)$). When selection is strong and symmetric, the two peaks are concentrated with equal probability around $-\Omega$ and Ω, $\mathrm{var}(z) = \Omega^2$, and so $Nm_{\mathrm{crit}} = v/(2\Omega^2)$. With weaker selection, or asymmetry, the variance between demes will be smaller, and the critical number of migrants higher. (Counter-examples exist, but seem contrived.) Note that this result is independent of the model of selection. Quite generally, the critical number of migrants above which the whole population shifts as one is somewhat greater than twice the genetic variance ($2v$), divided by the square of the distance between the peaks $(2\Omega)^2$.

(iv) *The two-state approximation for large Ns*

Exact calculation of the equilibrium distribution requires numerical integration of eqn (4). This is slow: in the following sections we develop approximations which give insight into the key parameters, and which allow rapid calculations. When selection is strong enough that demes are usually near one or other adaptive peak, the distribution can be approximated by the sum of two Gaussians, which can be integrated explicitly. This gives an analytic expression for the relation between the mean of the migrant pool, and the mean of the consequent distribution. Our aim will be to show that this relation applies to any distribution of polygenic traits or allele frequencies which clusters around alternative adaptive peaks, and does not depend on details of the model of disruptive selection.

The Gaussian approximation can be made in several ways. The most precise would be to find the maxima of the distribution [eqn (4)]; the variance of the distribution is approximated by $-1/(\partial^2\log(\psi)/\partial z^2)$ at these maxima. Migration will pull the maxima away from the peaks of mean fitness, at $\pm\Omega$; their exact position must be found by numerical solution of the cubic equation obtained by setting the derivative of eqn (4) to zero. A simpler approximation is to find the location of the maxima to first order in m/s:

$$z_- = \Omega\left(-1+\frac{m(1+\bar{z})}{s(1-\alpha)}\right)+O((m/s)^2). \quad (7a)$$

$$z_+ = \Omega\left(1-\frac{m(1-\bar{z})}{s(1+\alpha)}\right)+O((m/s)^2). \quad (7b)$$

Finally, the maxima can be taken to be at $-\Omega$, $+\Omega$, and terms of order (m/s) can be neglected. This is accurate to leading order in $(1/Ns)$. Then, migration only influences the outcome through its appearance in the exponent, through the factor $\exp(-(Nm/v)(z-\bar{z})^2)$ in eqn (5). This approximation will be accurate when selection is strong, relative to both drift and migration ($Ns \gg 1$, $Ns \gg Nm$), as is necessary if the demes are to cluster around distinct adaptive peaks.

On the simplest assumption, that the distribution is tightly clustered around peaks at $\pm\Omega$, we can use eqn (5) to obtain a simple expression for the relation between the mean across demes, and the mean in the migrant pool:

$$\langle z \rangle = \Omega\tanh\left(N\Delta\ln(\bar{W})+2\frac{Nm\,\Omega\bar{z}}{v}\right). \quad (8a)$$

Here, $\Delta\ln(\bar{W})$ is the difference in log mean fitness between the peaks: for the present model, $\Delta\ln(\bar{W}) = 2s\alpha/3$. This relation applies in the limit of large Ns, and is independent of the precise form of selection, $\bar{W}(z)$.

In Appendix 2, we generalize to cover the evolution of many characters, and to allow for changes in genetic variance with changes in the mean: this is necessary, for example, where we are following allele frequencies rather than polygenic characters, since then, $v = pq$. The general expressions [eqns (A 2.2), (A 2.3)] have the same form as eqn (8*a*), implying a similar critical value of Nm.

The three kinds of Gaussian approximation perform comparably, and become more accurate as Ns increases. Fig. 1 compares the exact results [heavy curve; eqn (5)] with those from the simplest Gaussian approximation [light curve; eqn (8*a*)], for $Ns = 30$. However, though the error in $\langle z \rangle$ is small, the consequent error in the position of the equilibrium is larger, especially near the critical point (see Figs 4, 5). To get an adequate approximation near the critical

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point, we must include terms of order $(1/Ns)$. This requires both that we approximate the positions of the peaks at $z_\pm$ using eqn (7), and that we allow for skew and kurtosis in the shape of the peaks by expanding around $z_\pm$ as

$$\psi(z) = (1 + a\zeta^3 + b\zeta^4 + O(\zeta^5)) \exp(-c\zeta^2/2),$$

where $z = z_\pm + \zeta$. This gives:

$$\langle z \rangle = \gamma\Omega\left[\bar{z}\frac{m\Omega^2}{sv} + \tanh\left(N\Delta \ln(\bar{W}) - \frac{\alpha}{2} + 2\gamma\frac{Nm\Omega\bar{z}}{v}\right)\right], \tag{8b}$$

where

$$\gamma = \left[1 - \frac{1}{4Ns}\left(3 + \frac{4Nm\Omega^2}{v}\right)\right].$$

The factor γ reduces the dependence of $\langle z \rangle$ on $\bar{z}$, and so reduces the mean at the critical point: it is caused by the skew of the adaptive landscape towards the centre. This approximation works very well for $Ns > 10$, but fails to give a solution for weaker selection. In the next section, we develop an approximation for this opposite limit.

Equation 8*b* cannot be solved for the general case. However, an approximation can be found when selection is almost symmetric. In the limit of small $Ns\alpha$, and including terms of order $1/Ns$:

$$Nm_{crit} = \frac{1}{2}\left(1 + \frac{3}{4Ns} + (Ns\alpha)^{\frac{2}{3}}\right), \tag{9a}$$

$$\bar{z} = 2\left(1 + \frac{5}{4Ns}\right)(Ns\alpha)^{\frac{1}{3}}. \tag{9b}$$

This dependence on the cube root of the controlling parameter is typical of this kind of bifurcation (a cusp catastrophe; Poston & Stewart, 1978). As a consequence, a very small asymmetry can give a large bias. For example, with $Ns = 30$, and $\alpha = 0{\cdot}001$, eqn (9) gives $\bar{z} = 0{\cdot}65$ at $Nm_{\text{crit}} = 0{\cdot}56$; this compares with $\bar{z} = 0{\cdot}55$ at $Nm_{\text{crit}} = 0{\cdot}59$, from eqn (8*b*).

(v) *Interpretation in terms of a modified adaptive landscape for small Ns*

When Ns is small, selection within demes is ineffective: the distribution $\bar{W}^{2N}$ is almost flat. However, if migration is high enough ($m > s$), the system will behave as one panmictic population, and disruptive selection will push it towards either $-\Omega$ or $+\Omega$. Here, we show analytically that, as with strong selection, selection can produce a strong bias towards the higher peak, even when all the demes start at the lower peak. As before, this process is most effective just below a critical number of migrants.

We approximate $\bar{W}^{2N}$ by $\exp(2N\log(\bar{W})) \approx 1 + 2N\log(\bar{W})$. Then, from eqn (5):

$$\langle z \rangle - \bar{z} \approx$$

$$\frac{\int (z-\bar{z})(1 + 2N\log(\bar{W}))\exp\left(-\frac{Nm}{v}(z-\bar{z})^2\right)dz}{\int (1 + 2N\log(\bar{W}))\exp\left(-\frac{Nm}{v}(z-\bar{z})^2\right)dz}$$

$$\approx 2N\int (z-\bar{z})\log(\bar{W})\exp\left(-\frac{Nm}{v}(z-\bar{z})^2\right)\sqrt{\left(\frac{Nm}{\pi v}\right)}dz$$

$$\approx \frac{v}{m}\frac{\partial \bar{W}^*}{\partial \bar{z}} \tag{10}$$

where

$$\bar{W}(\bar{z})^* = \int \bar{W}(z)\exp\left(-\frac{Nm}{v}(z-\bar{z})^2\right)\sqrt{\left(\frac{Nm}{\pi v}\right)}dz,$$

and $\log(\bar{W})$ has been approximated by $(\bar{W} - 1)$.

Since the equilibria are where $\bar{z} = \langle z \rangle$, we see that these correspond to the stationary points of the graph of $\bar{W}^*$ against $\bar{z}$, where $\partial \bar{W}^*/\partial \bar{z} = 0$. This function is just the adaptive landscape that would be produced by adding $(v/2Nm)$ to the phenotypic variance. When the number of migrants is large, this addition is small, and so the modified adaptive landscape still has two peaks: there are thus two stable equilibria. However, once the number of migrants falls below a critical value, the increased variance smooths the two peaks into one, so that the ensemble of demes has a single equilibrium. This qualitative change is similar to that described by Kirkpatrick (1982), who analysed the effects of changes in phenotypic variance on the evolution of a quantitative character under disruptive selection. This modification of the adaptive landscape has a simple interpretation: if selection is ignored altogether, the variance between demes is just $(v/2Nm)$ (Lande, cited in Lofsvold, 1988). Hence, $\bar{W}^*$ is the adaptive landscape for the whole population, allowing for the extra variance introduced in a balance between drift and gene flow.

For the model defined by eqn (1), eqn (10) gives:

$$\langle z \rangle - \bar{z} = \frac{Nsv}{8\Omega^4 Nm}\left[2(\Omega^2 - \bar{z}^2)(\bar{z} + \alpha\Omega) - \left(\frac{v}{Nm}\right)(3\bar{z} + \alpha\Omega)\right] \tag{11}$$

(The approximation can be made in another way. The distribution

$$\bar{W}^{2N}\exp\left(-\frac{Nm}{v}(z-\bar{z})^2\right)$$

has a single peak when $Nm \gg Ns$; approximating this peak by a Gaussian, and retaining terms of order $[(Ns/Nm)$, gives the first term of eqn (11)].

The form of eqn (11) is independent of the strength of selection. Thus, a large bias can be produced even when selection is weak relative to drift within individual demes (see Fig. 3*a*). However, when selection becomes very weak ($Ns \ll Nm$, 1), the difference between population mean and migrant pool

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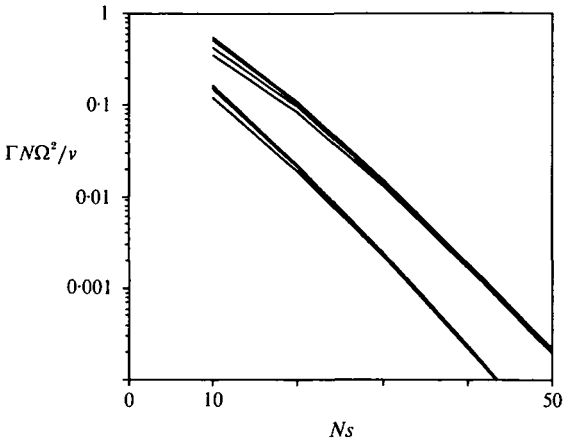

Fig. 6. The transition rate, Γ, as a function of Ns. This is scaled relative to the effective population size, N, and plotted on a log scale; thus, $\Gamma N\Omega^2/v = 0{\cdot}01$ corresponds to a timescale of $100N\Omega^2/v$ generations. Asymmetry is $\alpha = 0{\cdot}01$, and $Nm = 0{\cdot}5(v/\Omega^2)$, as in Fig. 1*a*). The upper set of lines gives the transition rates from lower peak to higher ($\bar{z} = -1$ to $+1$), and the lower set of lines gives the rate of transitions in the opposite direction. Each cluster consists of three lines, corresponding to exact numerical integration, and two approximations: first, that the equilibrium distribution is Gaussian, and second, that the peaks are at $+1$ and -1 (see text). In all cases, the overall mean $\bar{z}$ is approximated by the solution to eqn (8*a*). The approximations converge for large Ns.

($\langle z\rangle - \bar{z}$) becomes small, suggesting that approach to equilibrium may be slow.

In the symmetric case ($\alpha = 0$), the critical rate of gene flow is $Nm_{\text{crit}} = 1{\cdot}5(v/\Omega^2)$, which is higher than when selection is strong ($Nm_{\text{crit}} = 0{\cdot}5(v/\Omega^2)$ for large Ns). Some insight can be gained by considering very slight asymmetry:

$$Nm_{\text{crit}} = \frac{3}{2}\left(1+3\left(\frac{\alpha}{3}\right)^{\frac{2}{3}}\right), \tag{12a}$$

$$\bar{z} = 2\left(\frac{\alpha}{3}\right)^{\frac{1}{3}}. \tag{12b}$$

As with strong selection, the bias at the critical point is proportional to the cube root of the asymmetry, and so can be large. For example, if $\alpha = 0{\cdot}001$, $\bar{z} = 0{\cdot}14$ at $Nm_{\text{crit}} = 1{\cdot}52$, which is within 1 % of values calculated directly from eqn (11).

The critical rates of gene flow, and the corresponding bias to the higher peak, are shown for the limit of small Ns on the left of Figs 4, 5. The bias becomes much larger when Ns is large. However, the bias would also be much larger in the *absence* of gene flow when selection is strong (lower curve in Fig. 5): it is more impressive that gene flow can amplify even weak selection. As we show in the next section, the rate of approach to equilibrium is very slow when Ns is large, since peak shifts are then unlikely: the 'shifting balance' may therefore be a more effective mechanism of adaptation when selection is moderate (in the range $0{\cdot}01 < Ns < 10$, say).

(vi) *The dynamics*

When demes are usually near one or other peak, the state of the whole population can be described by a single variable – for example, the overall mean, $\langle z\rangle = \bar{z}$. If the population starts in some arbitrary distribution $\psi(z)$, it will rapidly move to cluster around the adaptive peaks. The mean will then change slowly as demes make occasional shifts between the peaks. In the limit of strong selection ($Ns \gg 1$), the rates of transition, Γ, can be found by numerical integration over the equilibrium distribution [eqn (5*b*) of Barton & Rouhani, 1987]. This integral can be further approximated by supposing that the distribution ψ is Gaussian [eqn (5*c*) of Barton & Rouhani, 1987], and by neglecting the slight change in the position of the peaks of the distribution caused by gene flow (see Appendix 2). For the model of disruptive selection [eqn (1)], this gives:

$$\Gamma(-\to+) = \left(\frac{Ns}{2\pi}\right)\sqrt{(2(1+\alpha))}(1-\alpha)$$
$$\times\exp\left[-\frac{Ns}{12}(1-\alpha)^3(3+\alpha)+\frac{Nm\Omega}{v}(1-\alpha)\right.$$
$$\left.(\Omega(1+\alpha)+2\bar{z})\right]. \tag{13a}$$

$$\Gamma(+\to-) = \left(\frac{Ns}{2\pi}\right)\sqrt{(2(1-\alpha))}(1+\alpha)$$
$$\times\exp\left[-\frac{Ns}{12}(1+\alpha)^3(3-\alpha)+\frac{Nm\Omega}{v}(1+\alpha)\right.$$
$$\left.(\Omega(1-\alpha)-2\bar{z})\right]. \tag{13b}$$

Various Gaussian approximations are compared in Fig. 6. This shows that the simplest approximation, given above, performs well. Our previous work has shown that the Gaussian approximation gives a close fit to simulations (Barton & Rouhani, 1987*a*, *b*; Barton, 1989). Figure 6 shows that peak shifts occur at an appreciable rate, provided that Ns is less than about 50. The rates depend very strongly on the asymmetry, especially when Ns is large.

The rate of change of the mean is:

$$\frac{\partial\bar{z}}{\partial t} = \{(\Omega+\bar{z})\Gamma(-\to+)-(\Omega-\bar{z})\Gamma(+\to-)\}. \tag{14a}$$

This can be simplified by defining Γ^* as the geometric mean transition rate ($\sqrt{[\Gamma(-\to+)\Gamma(+\to-)]}$), and noting that the ratio $\Gamma(-\to+)/\Gamma(+\to-)$ equals the ratio of abundances of the peaks at equilibrium, $(\Omega+\bar{z}_{\text{eq}})/(\Omega-\bar{z}_{\text{eq}})$. Then $\bar{z}_{\text{eq}}$ is given by eqn (8*a*). Thus:

$$\frac{\partial\bar{z}}{\partial t} = \frac{2(\bar{z}_{\text{eq}}-\bar{z})\Gamma^*}{\sqrt{1-\bar{z}_{\text{eq}}/\Omega}}. \tag{14b}$$

The condition that the intermediate equilibrium be stable is that the differential of eqn (14*b*) with respect to $\bar{z}$ be negative; note that $\bar{z}_{\text{eq}}$ depends on $\bar{z}$. This

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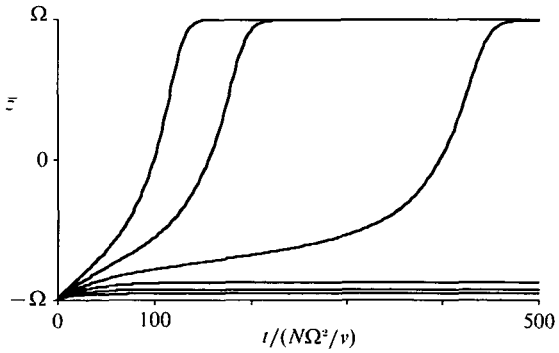

Fig. 7. The change in overall mean through time, on the assumption that demes are usually near one or other adaptive peak, Initially, all demes are at the lower peak; $Nm = (v/\Omega 2)$, $Ns = 30$. For these parameters, the critical asymmetry is $\alpha = 0{\cdot}022$; below this value, most demes stay near the original peak (lower curves: $\alpha = 0$, 0·01, 0·02), whilst above, most demes shift to the higher peak within $\approx 100N\Omega^2/v$ generations (upper curves: $\alpha = 0{\cdot}03$, 0·04, 0·05).

shows that the stability of equilibria depends on whether $\partial\bar{z}_{eq}/\partial\bar{z} = \partial\langle z\rangle/\partial\bar{z}$ is greater than or less than 1, at least where the two-state approximation applies. This supports eqn (6), and the derivation in Appendix 1. Fig. 7 shows the spread of the superior peak though time, for various asymmetries. When asymmetry is below the threshold, the superior peak cannot spread to high frequency; when it is above the threshold, it spreads to almost all the demes. Spread occurs moderately rapidly when $Ns = 30$, taking about $100N\Omega^2/v$ generations (Fig. 7). In general, the timescale increases exponentially with Ns.

4. Selection against heterozygotes

We now consider selection against heterozygotes at a single locus; variation is maintained by recurrent mutation. The method is essentially the same as for the polygenic case. However, if rates of mutation and migration are low ($N\mu, Nm \approx 1$ or less), the distribution of allele frequencies will follow a Gamma distribution rather than a Gaussian; the calculations must be modified accordingly (cf. Barton & Rouhani, 1987). If mutations are unique, the population never reaches an equilibrium. However, the probability of establishment and spread of a unique underdominant allele can be derived from the equilibrium formula for two alleles by taking the limit of low mutation rate.

For an island model receiving migrants at a rate m, from a migrant pool with allele frequency $\bar{p}$, Wright (1937) showed that the allele frequency distribution is:

$$\psi(p) = Cp^{4N\mu+4Nm\bar{p}-1}\, q^{4Nv+4Nm\bar{q}-1}\, \bar{W}^{2N} \qquad (15)$$

Mutation occurs at a rate μ from Q to P, and v from P to Q. We define the fitnesses of genotypes (QQ, PQ, PP) as 1, $1-s+\alpha s$, $1+2\alpha s$; selection is assumed to be weak ($s \ll 1$). The parameter α causes an asymmetry in fitness of the homozygotes, and can vary between

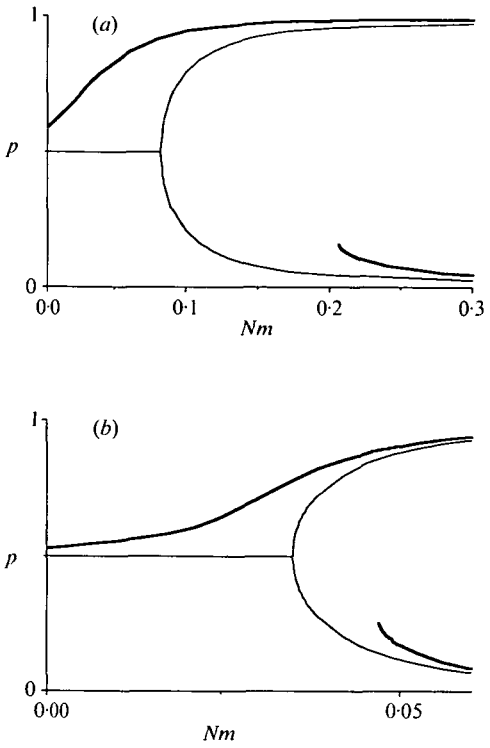

Fig. 8. The overall mean allele frequency ($\langle p\rangle = \bar{p}$), as a function of the number of migrants (Nm), for selection against heterozygotes; $N\mu = 0{\cdot}01$. (*a*) Weak selection ($Ns = 1$), using the approximation of eqn (23*a*). The light curve gives the symmetric case, where the critical number of migrants is $Nm_{crit} = 0{\cdot}0876$ (exactly), compared with 0·0828 from eqn (23*a*), and 0·0707 from eqn (25*b*). The heavy curve is for asymmetry $\alpha = 0{\cdot}1$; the critical number of migrants is then $Nm_{crit} = 0{\cdot}237$ (exactly), compared with 0·206 from eqn (23*a*), and 0·051 from eqn (25*a*) (that approximation breaks down because $N\mu$ is not small compared with Nm). (*b*) Strong selection ($Ns = 30$), calculated using the approximation of eqn (18*b*). The light curve is the symmetric case ($Nm_{crit} = 0{\cdot}0351$ exactly, 0·0350 from eqn (18*b*), and 0·0305 from eqn (21*b*), whilst the heavy curve is for asymmetry $\alpha = 0{\cdot}001$ ($Nm_{crit} = 0{\cdot}0461$ exactly, 0·0427 from eqn (18*b*), and 0·00559 from eqn (21*a*): again this approximation breaks down because $N\mu$ is not small compared with Nm).

$\alpha = 0$ and $\alpha = 1$; this is the same scale as in our model of disruptive selection. The selection coefficient is also comparable: the deterministic dynamics of $(p-q)$ are the same as those of z if s here is identified with (sv/Ω^2) for disruptive selection.

The mean of this distribution, $\langle p\rangle$, can be found by numerical integration. It depends on the mean of the migrant pool, $\bar{p}$, in essentially the same way as for the polygenic case (cf. Fig. 1). There are again two qualitatively different regimes. Below a certain critical number of migrants, the demes scatter towards different adaptive peaks, and the allele frequency amongst migrants evolves towards a single intermediate value. Above the critical number, all the demes tend to shift together to one or other adaptive peak. Fig. 8 shows the transition between the regimes (cf. Fig. 3). Note that as before, the behaviour is qualitatively similar for weak and strong selection

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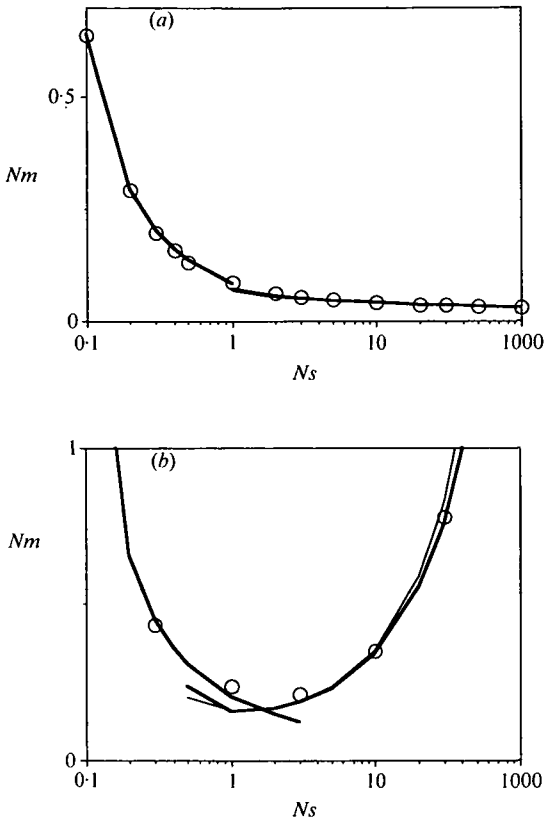

Fig. 9. The critical number of migrants, Nm, as a function of the strength of selection, Ns, for selection against heterozygotes; $N\mu = 0{\cdot}01$. (*a*), (*b*) Asymmetry $\alpha = 0$, $0{\cdot}1$ respectively. The circles give exact calculations, derived by numerical integration of eqn (13). (*a*) The heavy curve gives the small Ns approximation [eqn (23*a*)] for $Ns \leqslant 1$, and the large Ns approximation [eqn (18*a*)] for $Ns \geqslant 1$; the more accurate approximation of eqn (18*b*) is indistinguishable in this range. (*b*) The heavy curve on the left gives the approximation for small Ns [eqn (23*a*)], whilst the heavy curve on the right gives the leading-order approximation for large Ns [eqn (18*a*)]. The light curve on the right is from the more accurate approximation which includes terms of order $1/Ns$ [eqn (18*b*)]. Both the strong selection approximations break down for Ns less than around 1.

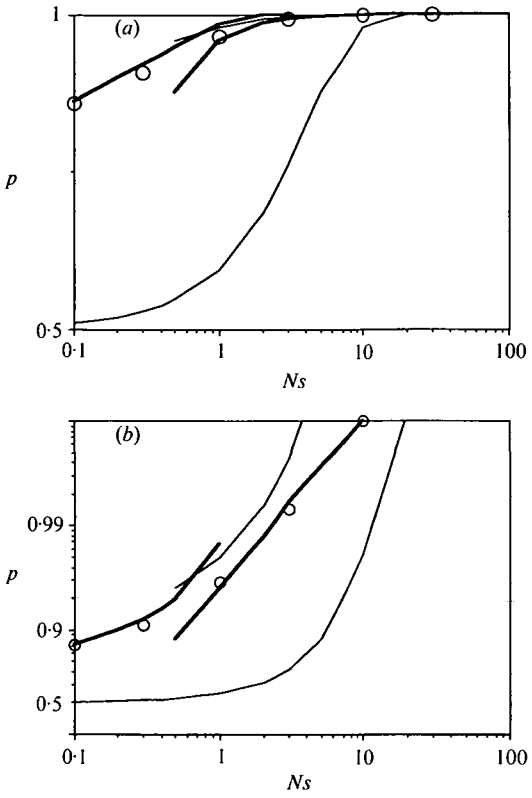

Fig. 10. (*a*) The mean allele frequency over all demes ($\bar{p} = \langle p \rangle$) at the critical point Nm_{crit}, as a function of the strength of selection, Ns, for asymmetry $\alpha = 0{\cdot}1$, and mutation rate $N\mu = 0{\cdot}01$. This is the greatest bias towards the fitter peak which can be achieved by a population which starts at the lower peak. Symbols are as for Fig. 9. The light curve on the lower right is the mean which would be achieved with no gene flow. (*b*) The same graph plotted on a log scale to expand the region near fixation.

(Fig. 8*a*, *b*), but that when selection is strong ($Ns = 30$, Fig. 8*b*), a very slight asymmetry ($\alpha = 0{\cdot}001$) can cause a strong bias.

Fig. 9 shows the way the critical number of migrants depends on the strength of selection; exact results from numerical integration are shown as circles, whilst the curves are approximations, developed below. These curves are for low mutation rates ($N\mu = 0{\cdot}01$), which seems most likely in nature. In the symmetric case, Nm_{crit} is around 1 when selection is weak, and declines as selection becomes stronger (Fig. 9*a*). With asymmetry $\alpha = 0{\cdot}1$ (Fig. 9*b*), Nm_{crit} has a minimum at $\approx 0{\cdot}2$. The corresponding bias towards the fitter peak is shown in Fig. 10*a*, and on a log scale in Fig. 10*b*. Note that $\bar{p}$ is close to 1 for strong selection, and is much higher than in the absence of gene flow (lower light curve) even with weak selection. Comparison of Figs 5 and 10 suggests that adaptation via the 'shifting balance' is more effective with disruptive selection on discrete alleles than with disruptive selection on a quantitative trait. However, note that when mutation between the alleles is rare, equilibrium will be approached slowly ($t \approx 1/\mu$).

(i) *A general expression for the critical migration rate*

Proceeding as for eqn (6), we find that the critical value occurs when:

$$\frac{\partial \langle p \rangle}{\partial \bar{p}} = 1 = 4Nm_{crit} \langle (p - \bar{p}) \ln (p/q) \rangle. \qquad (16a)$$

The critical number of migrants is inversely proportional to the expectation of $(p - \bar{p}) \ln (p/q)$, taken across the whole population. This does not have as simple an interpretation as in the polygenic case. However, when the demes cluster around peaks which are close to fixation, at ϵ_-, $1 - \epsilon_+$, say, eqn (16*a*) reduces to:

$$Nm_{crit} \approx \frac{1}{4\langle \bar{p} \ln (1/\epsilon_-) + \bar{q} \ln (1/\epsilon_+) \rangle}. \qquad (16b)$$

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This suggests that the critical number of migrants may be small (0·1, say), but depends only logarithmically on the location of the adaptive peaks. We confirm below that this is so in the limit of strong selection.

(ii) *Approximation for large Ns*

When selection is strong ($Ns \approx 1$ or more) the distribution is peaked near the boundaries. It can be approximated by a gamma distribution, which is obtained by taking the deterministic dynamics to be linear near the boundary. This gamma approximation accurately predicts the transition rates when selection is strong ($Ns \gg 1$), and reduces to the simpler Gaussian form when migration and mutation are common (Barton & Rouhani, 1987). To leading order in μ/s and m/s, the present model gives the location of the peaks of the distribution as:

$$p_- = \frac{\mu + m\bar{p}}{s(1-\alpha)}, \quad p_+ = 1 - \frac{\nu + m\bar{q}}{s(1+\alpha)}. \tag{17a}$$

Integrating over the gamma distribution gives the probabilities of being near one or other peak as $\{a_-/(a_- + a_+),\ a_+/(a_- + a_+)\}$, where:

$$a_- = \Gamma(4N\mu + 4Nm\bar{p})\,(4Ns(1-\alpha))^{-(4N\mu + 4Nm\bar{p})} \exp(-2Ns\alpha), \tag{17b}$$

$$a_+ = \Gamma(4N\nu + 4Nm\bar{q})\,(4Ns(1+\alpha))^{-(4N\nu + 4Nm\bar{q})} \exp(+2Ns\alpha). \tag{17c}$$

Since small asymmetries have a large effect on the occupancy of the two peaks, we need only consider $\alpha \ll 1$; then, the factors $(1-\alpha)$, $(1+\alpha)$ can be dropped. Since we assume that the peaks are near fixation, we take $p_- = 0$, $p_+ = 1$: this is correct to leading order in μ/s, m/s. With these approximations, the average across the whole population is:

$$\langle p - q \rangle = \tanh\left[N\Delta \ln(\bar{W}) + 2Nm(\bar{p} - \bar{q}) \log(4Ns) - \tfrac{1}{2}\log\left(\frac{\Gamma(4N\mu + 4Nm\bar{p})}{\Gamma(4N\nu + 4Nm\bar{q})}\right)\right]. \tag{18a}$$

This expression is similar to eqn (8*a*). The effect of gene flow depends primarily on $2Nm\log(4Ns)$, rather than on $2Nm\Omega^2/v$. This suggests that for given $Ns\alpha$, the critical migration rate may be low ($\approx 1/\log(4Ns)$) when selection is strong, as suggested by eqn (16*b*) above, and by Fig. 9*a*. There is also a third term, which includes the effects of mutation. If mutation rates are higher than migration rates ($4N\mu$, $4N\nu \gg 4Nm\bar{p}$, $4Nm\bar{q}$), this is approximately constant, and merely introduces a bias towards the allele with lowest mutation rate. If, as is more likely, mutation rates are low, the third term can dominate: it is this term which ensures that with no mutation, there are always stable equilibria at $\bar{p} = 0$ and 1, corresponding to fixation or loss through the whole population.

Eqn 18*a* is of order 1 in $(1/Ns)$, if we take the asymmetry α to be of order $(1/Ns)$. It predicts the critical number of migrants accurately down to $Ns \approx 1$ (Fig. 9), but overestimates the bias towards the fitter peak (upper light curve in Fig. 10). The same pattern was seen for the analogous approximation of eqn (8*a*). A better approximation is obtained by keeping terms of order $(1/Ns)$, as in the derivation of eqn (8*b*):

$$\langle p - q \rangle = \frac{m}{s}(\bar{p} - \bar{q}) + \frac{(\mu - \nu)}{s} + \left(1 - \frac{m + \mu + \nu}{s}\right) \times \tanh\left[N\Delta \ln(\bar{W}) + 2N(m(\bar{p} - \bar{q}) + (\mu - \nu)) \log(4Ns) - \tfrac{1}{2}\log\left(\frac{\Gamma(4N\mu + 4Nm\bar{p})}{\Gamma(4N\nu + 4Nm\bar{q})}\right) - 2N\alpha(m + \mu + \nu)\right] \tag{18b}$$

Since this is not much more complicated than eqn (18*a*), and since it is accurate even for $Ns \approx 1$ (Fig. 10*b*), we will use it throughout.

(iii) *The fate of individual mutations*

When there are many mutations per generation ($4N\mu \gg 1$), Stirling's approximation can be applied; this gives the formula that would be obtained using the general Gaussian procedure described in Appendix 2. It is more likely that mutations are rare. The two regimes of high and low migration can then be understood in terms of the fate of individual mutations.

Consider a population fixed for the less fit homozygote, QQ. If a single P allele is introduced into a deme by mutation, it has some small probability Γ_+ of being fixed in that deme, despite its continual dilution by immigrants from the rest of the population. After local fixation, its fate depends on whether it is more likely to infect another deme, or to be lost. Let the probabilities of spread and loss be λ_+, λ_- per generation; these define a Markov process, in which the number of demes fixed for the new arrangement increases or decreases at these rates (see Slatkin, 1980; Lande, 1985). The new allele is certain to be lost if $\lambda_- > \lambda_+$, and has probability $(1 - \lambda_-/\lambda_+)$ of spreading to high frequency if $\lambda_- < \lambda_+$. The rates λ_-, λ_+ are related to the probabilities of fixation of a P allele in a deme fixed for QQ (Γ_+), and of a Q allele in a deme near fixation for PP (Γ_-). For simplicity, assume that migration is rare ($4Nm \ll 1$). This ensures that the chance per generation that a PP deme will fix for Q is proportional to the number of immigrants carrying the Q allele, $2Nm$: $\lambda_- = 2Nm\,\Gamma_-$. The chance that a PP deme will trigger fixation of another deme can be found as follows. The frequency of the P allele in the migrant pool is $(1/k)$, where k is the (very large) number of demes. The average number of P alleles entering any one deme is $2Nm/k$, and so the chance of fixation of that deme is $2Nm\,\Gamma_+/k$. Summing over all demes gives $\lambda_+ = 2Nm\,\Gamma_+$ (see Slatkin, 1980). If the number of migrants is high ($4Nm \approx 1$), λ will be lower than $2Nm\,\Gamma$ (see Barton & Rouhani, 1987).

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This argument shows that when a deme at the new adaptive peak is more likely to shift back than to infect another deme ($\Gamma_- > \Gamma_+$), the new peak cannot spread to high frequency. If the number of migrants were small, then the fixation probabilities (Γ) would be independent of the state of the whole population, and would reflect the relative fitnesses of the two adaptive peaks. Only the fitter peak could then increase from low frequency, and there would be a single equilibrium. However, when the number of migrants is high, the common peak is likely to swamp the rare invader: the fixation probabilities reflect the current state of the population, and there will be two alternative stable equilibria, each close to fixation. The critical migration rate separating these two regimes is that which gives $\Gamma_- = \Gamma_+$.

The low-mutation limit could be obtained explicitly from the fixation probabilities. However, one can get the same result more simply by taking the limit of eqn (18*b*) as $4N\mu$ tends to zero. Consider first the equilibrium where the P allele is rare ($\bar{p} \approx \mu \ll 1$). Then:

$$\langle p \rangle = \left(\bar{p} + \frac{\mu}{m}\right)\left[\frac{m}{s} + \left(1 - \frac{m}{s}\right)\phi_-\right], \tag{19}$$

where

$$\phi_- = \frac{4Nm\,\Gamma(4Nm)\,e^{\alpha(4Ns-4Nm)}}{(4Ns)^{4Nm}}; \phi_- = a_+/(\bar{p}a_-)$$

in the limit of low mutation and small $\bar{p}$ [eqn (17)], and so is proportional to the relative chances that a deme will be at one or other peak.

Eqn (19) has the equilibrium solution:

$$\bar{p} = \frac{\mu}{(1-\phi_-)}\left[\frac{1}{s-m} + \frac{\phi_-}{m}\right]. \tag{20}$$

This lower equilibrium only exists when $\phi_- < 1$; then, an allele introduced at low frequency will stay rare, at a frequency proportional to the mutation rate. Above the threshold $\phi = 1$, it can increase from low frequency to spread through the whole set of demes. The critical migration rate cannot be calculated explicitly from $\phi_- = 1$; however, when $Ns \gg 1$, we have approximately:

$$Nm_{\text{crit}} = \frac{Ns\alpha}{(\log(4Ns) + \alpha + \gamma)} \quad \text{where } \gamma = 0{\cdot}577\ldots \text{is Euler's constant.} \tag{21a}$$

This was derived on the assumption that $\bar{p}$ is small, which will only be the case with moderate asymmetry. In the symmetric case, eqn (18*b*) leads directly to:

$$Nm_{\text{crit}} \approx \sqrt{\left(\frac{N\mu}{2(\log(4Ns) + \gamma)}\right)} \quad (\alpha = 0). \tag{21b}$$

As expected from eqn (16*b*), the critical number of migrants decreases with $\log(4Ns)$ in both cases, for fixed $Ns\alpha$. These approximations also correctly predict the monotonic decrease of Nm_{crit} with Ns in the symmetric case [eqn (21*b*), Fig. 9*a*), and the minimum for intermediate Ns [eqn (21*a*), Fig. 9*b*). For the mutation rate $N\mu = 0{\cdot}01$ used in Figs 8–10; eqn 21*b* is accurate down to $Ns = 5$, but eqn (21*a*) only converges for $Ns\alpha \ll \log(4Ns)$.

The highest frequency of P which can be reached by a population initially at the lower peak is when Nm is just below the critical point. The upper equilibrium is given by the analogue of eqn (20):

$$\bar{p} = 1 - \frac{\nu}{(1-\phi_+)}\left[\frac{1}{s-m} + \frac{\phi_+}{m}\right] \approx 1 - \frac{\nu\phi_+}{m(1-\phi_+)}$$

for $s \gg m$ (22*a*)

where

$$\phi_+ = \frac{4Nm\,\Gamma(4Nm)\,e^{-\alpha(4Ns-4Nm)}}{(4Ns)^{4Nm}}.$$

Now,

$$\phi_+ = e^{-2\alpha(4Ns-4Nm)}\phi_- \approx e^{-8\alpha Ns}\phi_- = (\bar{W}_-/\bar{W}_+)^{4N}\phi_-.$$

At the critical point, $\phi_- = 1$, and so the maximum bias to the fitter peak can be written as:

$$\bar{p} \approx 1 - \frac{\nu}{m[(\bar{W}_+/\bar{W}_-)^{4N} - 1]} \tag{22b}$$

This compares with the bias that would be achieved with no gene flow:

$$\bar{p} \approx \frac{\mu}{[\mu + \nu(\bar{W}_-/\bar{W}_+)^{2N}]}. \tag{22c}$$

Gene flow produces a strong bias, for two reasons. First, the ratio of mean fitnesses is raised to the power $(4N)$, rather than $(2N)$, which amplifies the asymmetry. Second, eqn (22*b*) has the form $(1 - \nu C)$, which is close to 1 for low mutation rates: the fitter peak spreads through the whole ensemble with the aid of migration, and approaches fixation. In the absence of gene flow, the peaks approach intermediate frequencies, in a balance between mutation in the two directions. Because low mutation rates ensure that demes approach fixation, the bias towards the fitter peak is much stronger than was the case for disruptive selection (cf. Figs 3 and 8, or Figs 5 and 10).

(iv) *Approximations for small Ns*

When selection within demes is weak relative to sampling drift ($Ns \ll 1$), we can proceed as for eqn (10), and approximate $\bar{W}^{2N}$ *by* $(1 - 4Nspq + 2Ns\alpha(p-q))$. This leads to:

$$\langle p - q \rangle = (\tilde{p} - \tilde{q}) + \frac{8Ns\,\tilde{p}\tilde{q}C}{(C+1)(C+2)}\left[(\tilde{p} - \tilde{q}) + \alpha\left(1 + \frac{2}{C}\right)\right] \tag{23a}$$

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where

$$C = 4N(m+\mu+\nu), \quad \tilde{p} = \frac{(\bar{p}+\mu/m)}{(1+(\mu+\nu)/m)}, \quad \tilde{q} = 1-\tilde{p}.$$

For $\bar{p} \approx \mu \ll 1$, this reduces to:

$$\langle p \rangle = \left(\bar{p}+\frac{\mu}{m}\right)(1-\omega), \tag{23b}$$

where

$$\omega = \frac{4Ns\,4Nm}{(4Nm+1)(4Nm+2)}\left[1-\alpha\left(1+\frac{1}{2Nm}\right)\right].$$

The solution is:

$$\bar{p} = \frac{\mu}{m}\left(\frac{1}{\omega}-1\right). \tag{24}$$

This solution exists provided that $0 < \omega < 1$; the former condition gives a critical number of migrants:

$$Nm_{crit} = \frac{\alpha}{2(1-\alpha)}. \tag{25a}$$

As for eqn (21*a*), this was derived on the assumption that the lower equilibrium is close to zero, which requires that there be sufficient asymmetry. In the symmetric case, eqn (23*a*) leads directly to:

$$Nm_{\text{crit}} = \sqrt{\frac{\mu}{2s}}. \tag{25b}$$

In the asymmetric case, the overall mean at the critical migration rate [eqn (25*a*)] is:

$$\bar{p} = 1-\frac{\nu(1+\alpha)}{2s\alpha^2}. \tag{26}$$

As with strong selection, these approximations fail for the mutation rate $N\mu = 0{\cdot}01$ used in Figs 8–10: they converge in the biologically reasonable limit where $N\mu$ tends to zero. Equations (21*a*) and (25*a*) confirm the patterns found in the numerical results. Since asymmetry acts through $Ns\alpha$, a slight asymmetry can cause a large bias when selection is strong. With moderate asymmetry, the critical migration rate is around 1, though it falls to $\approx \sqrt{\mu}$ in the symmetric case. The main contrast with disruptive selection is that if Nm is low enough that the fitter peak can spread from low frequency, it will spread almost to fixation ($\bar{p} \approx 1-\nu C$).

5. Discussion

Wright's 'shifting balance' allows a set of demes to escape from inferior adaptive peaks, and so evolve towards the global optimum. Our analysis has shown that the 'shifting balance' can ensure that the population becomes concentrated around the highest peak, even when that peak is only slightly above the alternatives. This process is most effective just below a critical number of migrants ($Nm_{\text{crit}} \approx 0{\cdot}1-1$). If the number of migrants is slightly higher, then migration from other demes prevents the spread of the new adaptive peak, whilst if it is lower, there is less bias towards the fitter peak. When selection is very weak, or very strong, convergence to equilibrium will be slow. However, there is a wide range of selection strengths over which the process can operate within a reasonable time ($0{\cdot}01 \ll Ns \ll 10$, say). The similarity between the results for quantitative traits and for selection against heterozygotes suggests that these patterns extend to any form of selection that sustains alternative adaptive peaks.

This analysis is one application of a general technique for understanding interactions between genes or demes, and can be extended in several other directions. In a future paper, we will deal with the 'third phase' of the shifting balance, by allowing the number of emigrants from a deme to increase with its mean fitness. Our results show that unless the relation between migration rate and mean fitness is very steep, this form of interdemic selection has little effect. Another possibility is to model joint fluctuations of population size and allele frequencies, so that the process of extinction and recolonization emerges naturally from the model. This offers an analytic approach to 'metapopulation' models (Gilpin & Hanski, 1991) with explicit population dynamics. We anticipate that the number of migrants will play a similar key role in this ecological context.

The island model is unrealistic for most organisms. Evolution in a one- or two-dimensional continuum could be treated in two ways. First, the population might be divided into regions, each consisting of large numbers of demes. Within each region, migration occurs at random, as in the island model. Migration also occurs between neighbouring regions, and could be approximated by diffusion if it is sufficiently localised. This is essentially a stepping-stone model in which the elements are *sets* of demes, and the variables are *distributions* of allele frequencies. If there is clinal variation across the whole population, this can be seen as a model of a 'mosaic' hybrid zone (Rand & Harrison, 1989).

This 'gradient-island' model might be a good approximation to a two-dimensional grid of demes if migration were very leptokurtic: immigrants would then come from a large enough area that random fluctuations in the migrant pool could be neglected, and the equilibrium distribution within each region derived as a function of the mean of that pool. It is instructive to make the analogy with the Ising model, which models a magnetic crystal in which atoms can align their spins either up or down, and neighbouring spins tend to align despite random thermal fluctuations. A mean field approximation, equivalent to our method used here, is successful even when interactions are only between nearest neighbours (Feynman, 1972). The analogy can be taken further when selection is strong, since then, demes are in one

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of two states, just as spins can take two directions. Results carry over directly for disruptive selection, but require some modification with selection against heterozygotes to allow for mutation.

The model of stabilizing selection on an additive polygenic trait can be combined with the island model. This case can be closely approximated by assuming that the mean of the character is close to the optimum. Stabilizing selection then acts to reduce the variance, and is equivalent to selection against heterozygotes at each locus (Wright, 1935). There are then two regimes. If Nm is greater than the critical value derived above, all demes will be close to fixation for the same allele at each locus. Gene flow will then have little effect on the genetic variance. However, if Nm is just below the critical point, demes will be near fixation for different alleles at any given locus. Gene flow can then maintain very much more variation than could mutation alone. If $N\mu \ll Nm \ll Ns\beta^2$, $V_g = (2m/s)$ per locus, as compared with $(4\mu/s)$ per locus with no gene flow (here, stabilizing selection has strength s, $W = \exp(-s(z-z_{opt})^2/2)$, and β is the effect of a single allele).

In the analysis of disruptive selection on a quantitative trait, we ignored the inflation of the variance by migration. It seems likely that the worst case would be where gene flow maintains all the variance, as described above. However even then, the difference in variance as the mean changes will be small, provided that there are many loci. This is because when Nm is below the critical point, roughly equal proportions of loci will be near fixation for '+' and for '−' alleles (assuming that changes in the mean are small compared with the possible range of the character, from all '−' to all '+'). Hence, migration acts like steady mutation. When Nm is above the critical point, most demes are at the same state for each locus. Therefore, there will only be an inflation of genetic variance due to migration at a small fraction of loci. This makes peak shifts more likely, but should be small if large numbers of loci are involved.

It has long been realised that the number of migrants, Nm, plays a crucial role in determining the distribution of neutral allele frequencies (Wright, 1932; Slatkin, 1987). For example, the relation $F_{st} = 1/(1+4Nm)$ shows that if Nm is small, most variation will be between rather than within demes. However, the dependence on Nm in the 'shifting balance' is much stronger, since there is a sharp transition between two qualitatively different regimes. Moreover, the similarity between our results for strong and weak selection, and for discrete alleles and quantitative traits, suggests that in a population with Nm in the range 0·01 to 1, the 'shifting balance' will be effective for all loci for which selection can maintain alternative adaptive peaks.

Kauffman & Johnsen (1992) have shown that phase transitions between ordered and disordered behaviour occur in a polygenic model of coevolution, and argue that in general, adaptation is most efficient near such transitions. This model, while very much simpler, supports their argument. Kauffman & Johnsen suggest that evolution may lead to fitness surfaces which bring the population close to the phase transition, thus maximising its rate of adaptation. In the present model, it is hard to see that individual selection would cause the appropriate number of migrants to evolve. It seems much more plausible that Nm varies across the species' range with the environment, and that conditions appropriate to the 'shifting balance' are found only in a small fraction of marginal demes. The large body of experimental work which infers Nm from patterns of gene frequencies suggests that Nm is much greater than 1 in most of the populations studied (Slatkin, 1987).

This is not a serious problem for the 'shifting balance'. If Nm is close to the critical value in some small region, then the fitter peak will be established there. It will initially be able to spread rather easily, because near the critical point, the lower equilibrium is only marginally stable. It will spread to occupy the whole range if its advantage is strong enough to overcome the flux of genes out of regions where Nm is larger. There is an analogy with optimisation by 'simulated annealing' (Kirkpatrick *et al.*, 1983), where a system is started at high temperature (or low Nm), and gradually cooled (slowing near any phase transition) until it 'freezes' into the optimal state. Here, Nm varies in space rather than in time.

This process can be described using the 'gradient-island' model, by adding a diffusion term to the expressions for $(\langle z\rangle - \bar{z})$: though this does not necessarily give the correct dynamics, it does show whether a balance between selection and a density gradient is possible. For example, with weak disruptive selection, adding diffusion between regions at a rate σ^2 to eqn (23*a*) shows that the fitter peak can spread if the density gradient is less than

$$\partial(\log(Nm)/\partial x = (\alpha/\sigma)\sqrt{[sv/(2Nm\Omega^2)]}.$$

Even if this condition is not often satisfied, random fluctuations in population structure may allow the fitter peak to advance past local obstacles. This model quantifies one explanation of the observation that evolutionary novelties tend to arise in peripheral isolates (Mayr, 1963).

Many open questions remain. Does the 'shifting balance' tend to maximise mean fitness, or do adaptive peaks spread for other reasons? Can fluctuations in selection play the same role as sampling drift? Can any intrinsic advantage of one peak over another overcome random processes, such as the chance extinction and recolonisation of large areas? Our analysis provides a general analytic technique that we hope will clarify these questions.

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Appendix 1

A finite number of demes

Here, we derive the distribution across a finite set of demes coupled together by migration. The same method is developed in more detail by Barton (1989), who dealt with n genes, coupled together by stabilizing selection. The mathematics for the two cases are almost identical.

Suppose that there are n demes, with means z_i ($i = 1,\ldots,n$), and mean fitnesses $\bar{W}_i$. Generalizing eqn (3*a*), the deterministic dynamics are:

$$\frac{\partial z_i}{\partial t} = v\frac{\partial \ln(\bar{W}_i)}{\partial z_i} - m(z_i - \bar{z}), \quad \text{where } \bar{z} = \frac{1}{n}\left(\sum_{i=1}^{n} z_i\right). \tag{A 1.1a}$$

These can be described by a potential, U, which is a function of the states of the whole set of demes [cf. eqn (3*b*)]:

$$\frac{\partial z_i}{\partial t} = v\frac{\partial U}{\partial z_i}, \quad \text{where } U = \sum_{i=1}^{n}\left(\ln(\bar{W}_i) - \frac{mn}{2v(n-1)}(z_i-\bar{z})^2\right). \tag{A 1.1b}$$

The equilibrium distribution of the whole set $\mathbf{z} = \{z_1, z_2, \ldots, z_n\}$, is given, as in eqn (4), by $\psi(\mathbf{z}) = C\exp(2NU)$, where N is the effective deme size. The mean across demes fluctuates randomly, with a distribution that can be derived from $\psi(\mathbf{z})$. We now show that (subject to a stability condition), the mean across demes converges to the fixed value calculated above using $\langle z\rangle = \bar{z}$.

We use the substitution:

$$\sum_{i=1}^{n}(z_i-\bar{z})^2 = \sum_{i=1}^{n}(z_i-z^*)^2 - n(\bar{z}-z^*)^2, \tag{A 1.2}$$

where z^* is for the moment an arbitrary reference point. Then:

$$\psi(\mathbf{z}) = C\prod_{i=1}^{n}\left(\bar{W}_i^{2N}\exp\left(-\frac{Nm}{v}(z_i-z^*)^2\right)\right) \exp\left(+\frac{n^2Nm}{v(n-1)}(\bar{z}-z^*)^2\right). \tag{A 1.3}$$

The product is over terms analogous to eqn (4): each gives the distribution of z_i, *given* that migrants have mean z^*, which we denote by $\psi(z_i|z^*)$. Now, the distribution of the mean across demes, $\psi(\bar{z})$, can be found by integrating eqn (A 1.3) across $\mathbf{z}$, with the constraint $(1/n)\Sigma_{i=1}^{n} z_i = \bar{z}$. The last term remains unchanged, whilst the first term reduces to the distribution of the mean of n independent variables, each with distribution $\psi(z_i|z^*)$. By the Central Limit Theorem, this converges to a Gaussian with mean

$$\langle z_i\rangle = \int z_i\psi(z_i|z^*)\,dz_i,$$

and variance

$$\mathrm{var}(z_i)/n = \left(\int (z_i-\langle z_i\rangle)^2\psi(z_i|z^*)\,dz_i\right)\Big/ n.$$

Now, set $z^* = \langle z\rangle$; since all demes must follow the same distribution in this model, we can drop subscripts. Then, the distribution of $\bar{z}$ reduces to the form:

$$\psi(\bar{z}) = \exp\left(-\frac{n}{2\,\mathrm{var}(z)}(\bar{z}-\langle z\rangle)^2 + \frac{n^2Nm}{v(n-1)}(\bar{z}-\langle z\rangle)^2\right). \tag{A 1.4}$$

This is a Gaussian with mean $\langle z\rangle$, and variance:

$$\mathrm{var}(\bar{z}) = \frac{1}{n\left(\dfrac{1}{\mathrm{var}(z)} - \dfrac{2nNm}{v(n-1)}\right)}. \tag{A 1.5}$$

Provided that $nNm/(n-1) < v/(2\,\mathrm{var}(z))$, this converges to zero as the number of demes becomes large: the distribution across demes then approaches that where the mean is fixed at the solution to $\bar{z} = \langle z\rangle$, as assumed above. The condition on Nm can be understood from eqn (6); since $\partial\langle z\rangle/\partial\bar{z} = 2(Nm/v)\,\mathrm{var}(z)$, it reduces to $\partial\langle z\rangle/\partial\bar{z} < 1$, confirming the heuristic argument used above.

Appendix 2

Generalization to multivariate evolution, and changing genetic variance

We now show that the relationship in eqn (8*a*) gives a general description of the competition between alternative adaptive peaks in the island model. Suppose that selection acts on a vector of variables, $\mathbf{z}$. We first consider the simplest case, where the matrix of genetic variances and covariances, $\mathbf{v}$, remains constant. This would be a good approximation if $\mathbf{z}$ represents the means of a set of polygenic traits, determined by large numbers of loci, and under weak selection. As for the case of a single trait under disruptive selection, we must make the restrictive assumption that migration itself does not cause significant changes in variance (see Discussion). With these assumptions, the system can be described by a potential,

$$U = \ln(\bar{W}) - m(\mathbf{z}-\bar{\mathbf{z}})^T\mathbf{v}^{-1}(\mathbf{z}-\bar{\mathbf{z}})/2 \quad \text{[cf. eqn (3}b\text{)]}.$$

The exact solution could be found by numerical integration over the distribution $\exp(2NU)$. However, a simple approximation can be applied, as above, by approximating the distribution around each peak by a Gaussian, and discarding terms of order (m/s).

We develop this approximation by first considering the distribution in the absence of gene flow. The

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probability of being near an adaptive peak at $\pm\Omega$ is denoted by $\exp(2NF_{\pm})$, which is defined as the integral of $\exp(2NU)$ over the domain of attraction of the peak. Under the Gaussian approximation:

$$F = \ln(\bar{W}) + \frac{S}{2N}, \quad \text{where } S = \tfrac{1}{2}\ln\left(\left(\frac{\pi}{N}\right)^n \frac{1}{\det(\mathbf{D})}\right) \tag{A 2.1}$$

Here, n is the number of variables – in other words, the dimension of the adaptive landscape. **D** is the matrix of second differentials of the log mean fitness at the peak ($\mathbf{D}_{ij} = -\partial^2 \ln(\bar{W})/\partial z_i \partial z_j$). Equation (A 1) is equivalent to eqn (14*b*) in Barton & Rouhani (1987); note that in eqn (11) in Barton (1989), the probability of being near some peak labelled m is denoted by Z_m/Z, where $Z = \Sigma_m Z_m$. Here, $Z_m = \exp(2NF)$.

F is approximately equal to the log mean fitness, but includes a second term that takes account of fluctuations away from the peak. The notation used here is derived from an analogy with thermodynamics (see Barton, 1989, p. 64). The log mean fitness is analogous to (minus) the energy, E. Wright's distribution $\exp(2N\ln\bar{W})$ is analogous to Boltzmann's distribution, $\exp(-E/kT)$. Thus, the number of genes in the population ($2N$) is analogous to an inverse temperature ($1/kT$): small population size corresponds to high temperature, since random fluctuations are large, and low fitness (i.e. high energy) states become more likely. The difference between the energy (here, the log mean fitness) and the free energy (here, F) is the product of entropy and temperature (here, $S/2N$). The entropy, S, is a measure of the number of available states which surround the adaptive peak: if the peak is flat, the curvature **D** will be small, the entropy will be large, and the peak is more likely to be occupied.

If the effects of gene flow are now included, by including the term $[m(\mathbf{z}-\bar{\mathbf{z}})^T \mathbf{v}^{-1}(\mathbf{z}-\bar{\mathbf{z}})/2]$ in the potential, the probability of occupying the peak will now be proportional to

$$\exp(2NF^*) = \exp(2N\ln(\bar{W})^* - m(\mathbf{z}-\bar{\mathbf{z}})^T \mathbf{v}^{-1}(\mathbf{z}-\bar{\mathbf{z}})/2 + S^*).$$

Now, the peak of the distribution will be perturbed slightly, from $\pm\Omega$ to $\mathbf{z}^*$; migration will also perturb the mean fitness at the peak ($\bar{W}^*$), and the size of the surrounding region [$\exp(S^*)$]. However, if migration is weak relative to selection, these effects can be ignored: to leading order in Ns and (m/s), gene flow only affects the exponent, through the term $\exp[-m(\mathbf{z}-\bar{\mathbf{z}})^T \mathbf{v}^{-1}(\mathbf{z}-\bar{\mathbf{z}})/2]$. So, approximating $\mathbf{z}_-$ by $-\Omega$, and $\mathbf{z}_+$ by Ω, the mean of the distribution is:

$$\langle \mathbf{z} \rangle = \frac{[-\Omega \bar{W}_-^{2N} e^{-m(-\Omega-\bar{\mathbf{z}})^T \mathbf{v}^{-1}(-\Omega-\bar{\mathbf{z}})/2} + \Omega \bar{W}_+^{2N} e^{-m(\Omega-\bar{\mathbf{z}})^T \mathbf{v}^{-1}(\Omega-\bar{\mathbf{z}})/2}]}{[\bar{W}_-^{2N} e^{-m(-\Omega-\bar{\mathbf{z}})^T \mathbf{v}^{-1}(-\Omega-\bar{\mathbf{z}})/2} + \bar{W}_+^{2N} e^{-m(\Omega-\bar{\mathbf{z}})^T \mathbf{v}^{-1}(\Omega-\bar{\mathbf{z}})/2}]}$$

$$= \Omega \tanh[N\Delta \ln(\bar{W}) + 2Nm\Omega^T \mathbf{v}^{-1}\bar{\mathbf{z}}]. \tag{A 2.2}$$

This is a multivariate generalization of eqn (8*a*), but is still restricted to constant genetic variance, **v**. The difference between the entropies at either peak (ΔS) has been ignored (as in the one-dimensional case described above), as being negligible compared with the leading term, $N\Delta \ln(\bar{W})$.

The next step is to allow the genetic variance to be a function of the dynamic variables, **z**. The vector **z** could now represent a set of allele frequencies. The genetic variance is then $\mathbf{v}_{ii} = p_i q_i/2$; this determines both the response to selection and the variance of fluctuations due to sampling drift. (In order that the population moves up gradients of mean fitness, with metric **v**, selection must be weak enough that linkage disequilibria are negligible.) **z** could also represent the means of a set of quantitative characters, or even, the means, variance and higher moments of the set of characters. In the latter case, the Gaussian or rare-allele approximation must be used to describe the evolution of the system (Barton & Turelli, 1987).

If the variables **z** represent a set of allele frequencies (p, q), the effects of gene flow can be described by a potential $m[\bar{p}\ln(p) + \bar{q}\ln(q)]$; this case is elaborated above. In general, however, gene flow cannot be described by any potential if the genetic variance changes. We can nevertheless extend eqn (A 2.2) by considering gene flow as introducing a small perturbation away from a system which *is* described by potential. As before, consider the case of no gene flow. Then, the population is most likely to shift from one peak to the other along a particular trajectory, which is a ridge in the adaptive landscape. The problem can be reduced to one in a single dimension by integrating over the $(n-1)$ dimensions orthogonal to this most likely path (see Barton & Rouhani, 1987). When a small amount of gene flow is included ($m \ll s$), the most likely path between the two peaks will follow almost the same trajectory: the dynamics therefore need only be followed along this path. Label points along the trajectory by ζ, the peaks being at $\zeta = \pm\Omega$. The expected rate of change due to migration is $-m(\zeta - \bar{\zeta})$, and the variance in ζ is $v(\zeta)$; this is a projection of the full matrix **v** onto the most likely path. The probability of making a transition can be found by integrating along the most likely path (see Rouhani & Barton, 1987). Since the relative probabilities of being near one or other adaptive peak at equilibrium depend on the ratio between the transition rates in either direction, this method gives the equilibrium distribution. Hence, the mean value of ζ over the whole set of demes is:

$$\langle \zeta \rangle = \Omega \tanh\left[N\Delta \ln(\bar{W}) - Nm \int_{-\Omega}^{\Omega} \frac{(\zeta - \bar{\zeta})}{v(\zeta)} d\zeta\right]. \tag{A 2.3}$$

This expression applies to any system for which the Gaussian approximation holds, provided that selection is strong relative to drift and gene flow ($Ns \gg 1$, Nm). In general, the mean fitness $\bar{W}$ may be modified to take account of other evolutionary forces, such as

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mutation. Above, where we consider selection against heterozygotes, we must make a further generalization to cover the case where the adaptive peaks are close to fixation, so that the distribution is far from Gaussian.

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