

Genetic demarcation of geographical distribution by hybrid zones

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Abstract.

Two models of coadaptation are examined in terms of their effect on hybridising taxa in a contact zone. It is shown that the intrinsic selective forces generated by differential coadaptation of the hybridising taxa are sufficient to maintain a stable, narrow hybrid zone, in the absence of extrinsic selective forces such as those provided by an ecotone.

One of the models is shown to be capable of generating a stable, narrow hybrid zone which moves in one direction. It is suggested that this movement of a hybrid zone will give rise to apparent unidirectional introgression across the zone. Consequently the point of maximal marker heterozygosity will not correspond to the point of minimum population mean fitness. Eight natural examples of asymmetrical hybrid zones are discussed in the light of the model.

Introduction

The narrowness and apparent temporal stability of secondary contact hybrid zones has intrigued evolutionary biologists at least since the classical study of Meise (1928) on the hybrid zone between hooded and carrion crows in Europe. A commonly postulated hypothesis for the maintenance of such zones is that the hybridising taxa are differentially coadapted. Disruption of the coadapted parental gene complexes in the F₂ and backcross generations causes a reduction in fitness, which is sufficient to maintain a narrow and stable zone (Mayr, 1963). Unfortunately, very little effort has been made to develop this proposal beyond the stage of verbal description (which is almost a truism) and consequently understanding of the phenomenon has stood still.

The principal problem is a lack of basic information about the mechanism of coadaptation and the way in which related taxa might evolve different systems of coadaptation. A possible means of circumventing this problem is to develop rigorous genetic models of coadaptation and to examine these models in the context of hybrid zones. There are several objections to this approach. First, genetic models of coadaptation involve few genes, whereas very many genes may be

involved in nature. However, results from oligogenic models may not differ qualitatively from those derived from polygenic models; and in any case, it is likely that simple models will facilitate our understanding of the potentially more complex natural situation. A second objection is that hybrid zones may be primary zones (Endler, 1977), not secondary contact zones, as these analyses will assume. However, it is universally accepted that some hybrid zones are the results of secondary contacts. This objection, therefore, challenges only the generality, rather than the validity of the results obtained from the models. A third possible objection is that the analysis of the models has ignored extrinsic selection gradients which could be created by environmental variation. In reply to this, it must be pointed out that a major difficulty of practical hybrid zone studies has been the identification of environmental variation, which might be involved in maintaining the zone. Any model which can simulate a narrow zone in the absence of extrinsic fitness gradients must, therefore, be helpful to our understanding of the natural situations.

The models

The heuristic basis of the coadaptation models

used is due to Maynard Smith (1958). Suppose that in taxon 1, two steps in a developmental or biochemical process are controlled by two loci, such that the reaction rates of both steps are fast. In a closely related taxon, taxon 2, two different alleles cause both steps to be slow. Derived offspring of these taxa, bearing unmatched combinations of steps, such as fast/slow, fast/intermediate or slow/intermediate are sterile or inviable; that is, they have fitness of zero. Only two variants of this model which differ in the mode of gene action are considered here. In the first variant, the alleles at both loci are codominant. In the second variant, the alleles at both loci in taxon 1 are dominant to the corresponding alleles in taxon 2. The loci are assumed to be autosomal and unlinked. A consideration of the effects of linkage, sex linkage and other patterns of dominance will be presented elsewhere (Moran, in prep.).

(1) The PQ or symmetrical breakdown model.

The alleles P,Q in taxon 1 are codominant with alleles p,q in taxon 2.

The F2 phenotype and fitness matrix is as follows:

PQ	Pq	pQ	pq	X
fast fast (T1) 1	fast int 0	int fast 0	int int (F1) 1	PQ
	fast slow 0	int int 1	int slow 0	Pq
		slow fast 0	slow int 0	pQ
			slow slow (T2) 1	pq

The parental, T1 and T2, and F1 phenotypes are indicated. The top row of this matrix and the right hand column represent the results of backcrosses of the F1 to taxon 1 and to taxon 2 respectively. The backcrosses are equally affected by hybrid breakdown.

(2) The AB or assymetrical breakdown model.

A,B alleles in taxon 1 are dominant to a,b alleles in taxon 2. The F2 phenotype and fitness matrix in this case is quite different from that of the PQ model.

AB	Ab	aB	ab	X
fast fast (T1) 1	fast fast 1	fast fast 1	fast fast (F1) 1	AB
	fast slow 0	fast fast 1	fast slow 0	Ab
		slow fast 0	slow fast 0	aB
			slow slow (T2) 1	ab

In the top row of this matrix, it can be seen that all progeny of backcrosses to taxon 1 survive. However, the right hand column shows that only half of the progeny from backcrosses to taxon 2 survive. There is distinct asymmetry in the pattern of breakdown in this case because of the mechanism of gene action in which A,B alleles are dominant to a,b.

Methods

Two types of analysis have been used:

(1) A multilocus generator program was used to determine the unstable equilibrium frequencies of both P,Q and A,B alleles, given initial complete coupling of alleles as found in the parental taxa. The program deterministically tracks gene frequencies from any given initial frequency. The behaviour of alleles at neutral loci, with various levels of linkage to the P or Q, or A or B loci, was also examined but is not reported in detail here.

(2) A two-locus multidemic simulation program, which deterministically follows changes in gene frequency due to selection and migration in 100 contiguous demes, was used to examine these models in a secondary contact situation. A simulation was initiated with demes 0 to 50 consisting only of taxon 1 and demes 51 to 100 containing only taxon 2. Migration levels of 10%, that is 5% in either direction, and 20% were used, and the frequency of P,Q or A,B alleles in each deme was followed for up to 400 generations. In this way, it was possible to determine whether an abrupt transition in gene frequencies persisted, and also whether the position of the gene frequency transition was stable.

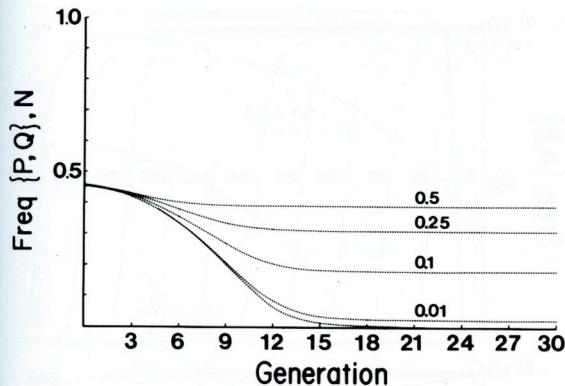


Fig. 1—Illustration of the changes in gene frequency induced by the PQ model of breakdown. The initial frequency of P, Q, N alleles in this case is 0.45. The level of linkage of the neutral, N, loci, with the P or Q loci is indicated in the body of the graph.

Results

The PQ system

The unstable equilibrium frequency of P, Q alleles, with or without complete coupling of P with Q and p with q as an initial condition, is 0.5. Any divergence from this frequency leads to rapid elimination of the less common alleles. Figure 1 illustrates gene frequency behaviour when there are initially 0.45 PQN/PQN and 0.55 pqn/pqn.

P, Q alleles, in this case, are rapidly eliminated. It is also obvious from this figure that the behaviour of alleles at the neutral loci depends on the level of their linkage with one or other of the interactive loci. Unlinked neutral alleles (N, 0.5) are only slightly depressed from their initial frequency of 0.45 to 0.39 at nine generations, by which stage they have lost their initial linkage disequilibrium with the P or Q loci. By contrast, tightly linked neutral alleles (N, 0.1) are displaced from 0.45 to 0.02 by generation 18. No further perturbation is obvious at this point since P, Q alleles are by then reduced to a negligibly low frequency. Perturbations of the neutral allele frequencies are, of course, dependent on changes in P, Q frequency and linkage disequilibrium between the neutral and the selected loci.

Simulated secondary contact (Fig. 2) demonstrates that the PQ model produces a stable and static narrow hybrid zone. The position of the gene frequency equivalence point, which in this case is also the unstable equilibrium frequency point, can be seen by interpolation to be midway between demes 50 and 51 after frequencies in neighbouring demes have stabilised. Different

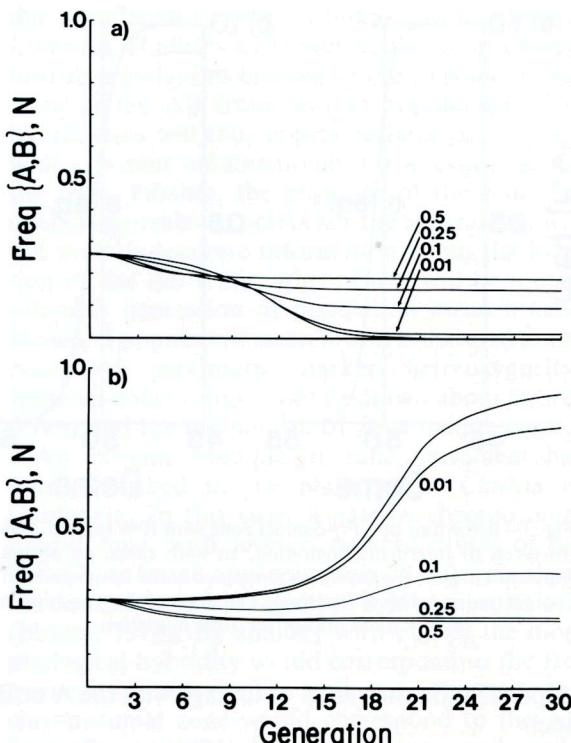


Fig. 2—Illustration of the changes in gene frequency induced by the AB model of breakdown. Note that the unstable frequency of A, B alleles lies between 0.26 (a) and 0.27 (b). Neutral loci are labelled as in Figure 1.

rates of migration affect the width but not the position of the midpoint of the zone.

The AB system

The unstable equilibrium frequency of A, B alleles, given initial complete coupling of A with B and a with b, is 0.267. (The unstable frequency has only been estimated to three decimal places in this case, whereas it has been determined exactly for the PQ model.) The behaviour of A, B alleles, from initial frequencies of 0.26 and 0.27 respectively, is illustrated in Fig. 3. a and b respectively. In the former case, A, B alleles are rapidly eliminated, so that by generation 18, only a negligible frequency of these alleles is present in the population. In the latter case, by contrast,

A, B alleles rapidly increase in frequency. However, the efficiency of selection against a, b alleles is very low, when A, B alleles are very common, because of the masking effect of the dominant alleles. Hence, the approach to fixation of A, B alleles slows markedly after a frequency of about 90% has been attained. As with the PQ system, the extent of perturbation of N alleles is

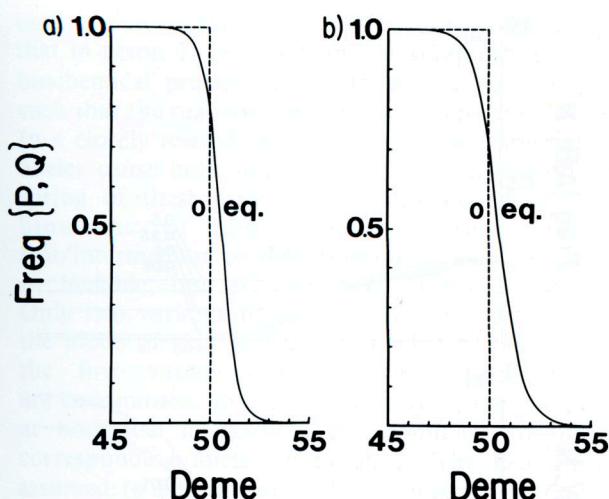


Fig. 3—Behaviour of a PQ contact zone with 10% (a) and 20% (b) levels of interdemic migration. In both cases, an abrupt transition in gene frequency is maintained at the initial point of contact (indicated by dotted line). The slope of the transition is slightly more abrupt for 10% migration.

dependent on their level of linkage with the A or B loci.

Simulated secondary contact (Fig. 4, a,b) shows that the AB model of breakdown, like the PQ system, is capable of maintaining an abrupt gene frequency transition or hybrid zone. However, in marked contrast to the PQ system, the hybrid zone shows a constant tendency to move so that ab/ab populations are replaced by AB/AB. Both the width of the zone at any given time, and the rate of movement of the zone, are dependent on the rate of interdemic migration.

Discussion

A simple difference in the mode of gene action between the PQ and AB models has been shown to produce drastically different results at the level of hybridising populations. On the one hand, the PQ model with codominant gene action produces a static hybrid zone. On the other, the AB system with dominance of A,B alleles over a,b alleles creates a zone which moves continuously at a rate dependent on the interdemic migration rate. Even though the geographical location of the hybrid zone is not constant, the zone is stable in the sense that an abrupt gene frequency transition is maintained.

Of course, hybrid zones are not observed in nature in terms of P,Q or A,B allelic frequencies. At best a limited amount of information on coadaptational differentiation can be obtained from experimental hybridisation. Instead, hybrid

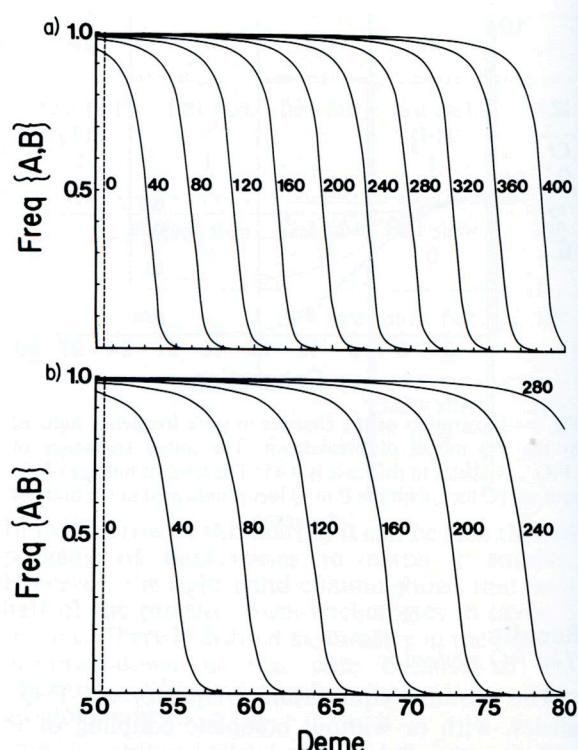


Fig. 4—Behaviour of an AB contact zone with 10% (a) and 20% (b) levels of interdemic migration. In this case, although an abrupt transition in gene frequency is maintained, the position of the transition is not, and it forms a moving gene frequency front. The position of the front is shown at intervals of 40 generations. Note that both the slope of the front and the rate at which it moves are influenced by the level of interdemic migration.

zones are analysed by using morphological, electrophoretic or chromosomal markers. What effect will selected loci such as the PQ loci or the AB loci have on marker loci which have no role in the maintenance of the hybrid zone? For simplicity, such marker loci will be treated as neutral loci, although they could conceivably be selected by extrinsic selective forces. Unfortunately, this problem could not be analysed at the hybrid-zone level because the available multidemic simulation program was capable of simulating only two genetic loci. However, the results of the multilocus generator analysis have provided sufficient information (Moran in prep. a,b) to give partial answers to this question.

It is clear that perturbation of neutral gene frequencies is related to the intensity of linkage between the neutral and selected loci. Tightly linked neutral loci are perturbed to a greater extent and for a longer period of time than are loosely linked neutral loci (see, for example, Figs 1 and 3). For

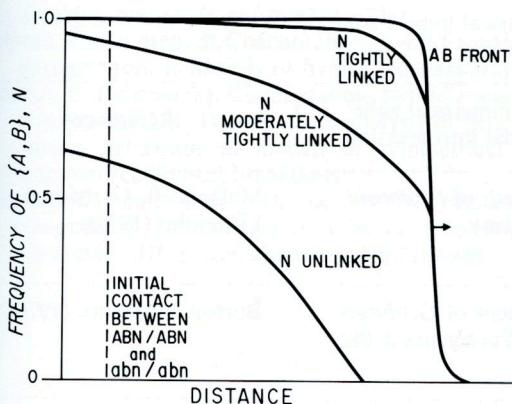


Fig. 5—Postulated effect of a moving gene frequency front of the AB type on neutral loci. It is proposed that the movement of the AB front will give rise to apparent one way introgression of n alleles from the abn taxon into the ABN taxon. The extent of this apparent introgression will be determined by the level of linkage of the neutral loci to the A or B loci, with unlinked neutral alleles introgressing at the fastest rate. Such introgression really represents a trail of the movement of the AB front.

the PQ system, this means that loosely linked neutral loci will develop less abrupt gene frequency transitions more quickly than tightly linked neutral loci, but in all cases the gene frequency equivalence points of the neutral loci will coincide with the equivalence point for the P and Q loci. Thus unlinked neutral loci will introgress fastest, but in all cases, introgression will be equal in both directions. Hence, symmetrical gene frequency transitions will persist for all loci. Further, the position of the equivalence point for the marker loci will reveal the position of the unstable point for the selected loci, a point of considerable biological significance.

For the AB system, the situation is considerably more complex. The probable course of events is represented diagrammatically in Fig. 5. As the AB zone moves, displacing ab populations, the unlinked neutral markers will quickly lose their initial linkage disequilibrium with the A and B loci. Consequently, N alleles will be left behind and n alleles will appear at increasingly higher frequencies behind the moving AB front. Tightly linked neutral loci will lose their initial linkage disequilibrium with the A or B loci very slowly, and consequently will follow the movement of the AB front closely; although given sufficient time they also will be left behind by the moving front. Neutral loci with intermediate levels of linkage will, of course, show intermediate patterns of change. Thus, there will appear to be variable introgression of n alleles into AB populations, with variable penetration of the "introgressant" alleles,

due to different patterns of linkage for each locus. Likewise, N alleles will never appear to introgress into ab populations because of the constant movement of the AB front into ab populations. The hybrid zone will thus appear to be asymmetrical, with apparent unidirectional introgression across the zone. Further, the positions of the gene frequency equivalence points for the neutral loci will not provide accurate information about the location of the AB front, where there will be a considerable depression of population mean fitness. Hence, if population mean fitness is assessed at the point of maximum marker heterozygosity, spurious conclusions could be drawn about hybrid fitness and the mechanism of zone maintenance.

An extreme example of zone movement has been described in the plant genus *Clarkia* in California. In this case, a narrow chromosomal hybrid zone, with severely reduced fitness of the hybrids, is found approximately 70 miles south of the centre of a broad region of hybrid morphology (Bloom, 1976). By analogy with Fig. 5, the morphological hybridity would correspond to the frequency transition for unlinked N, and the chromosomal zone would correspond to the AB front. Bloom (1976), however, has assumed northward movement of superior morphological genes, with the chromosomal zone marking the point of initial contact.

Several other examples of asymmetrical introgression, in some cases associated with observed zone movement, are listed in Table 1. Hybrid-zone movement, due to asymmetrical hybrid breakdown, could explain the properties of all of these zones; although most of the observed zone movements have been attributed to climatic or other ecological changes.

Acknowledgements

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TABLE I. Examples of asymmetrical hybrid zones.

Direction of introgression	Reason for asymmetrical zone and unidirectional introgression	References
<i>Pseudophryne semimarmorata</i> ↓ <i>Pseudophryne bibroni</i>	Observed southward movement of <i>P. bibroni</i> into <i>P. semimarmorata</i> territory.	McDonnell, Gartside & Littlejohn (1978).
<i>Gymnorhina hypoleuca</i> ↓ <i>Gymnorhina tibicen</i>	Observed southward movement of <i>G. tibicen</i> (which is picking up <i>G. hypoleuca</i> genes in the process).	Burton & Martin, (1976).
<i>Quiscalus quiscale versicolor</i> ↓ <i>Quiscalus quiscale quiscale</i>	Observed northward movement of <i>Q. q. quiscale</i> over a 30-year to 40-year time span.	Yang & Selander (1968).
<i>Drosophila arizonensis</i> ↓ <i>Drosophila mojavensis</i>	Heterosis for second and third chromosomes of <i>D. arizonensis</i> in genetic background of <i>D. mojavensis</i> .	Mettler (1969), Nagle & Mettler (1969).
Torresian (<i>Caledia captiva</i>) ↓ Moreton (<i>Caledia captiva</i>)	Asymmetrical hybrid breakdown in embryonic stages of F2 and backcross progeny.	Moran & Shaw (1977), Moran (1979).
<i>Mus musculus domesticus</i> ↓ <i>Mus musculus musculus</i>	Proposed either: (1) net northward dispersal, or (2) lesser intensity of selection against introgressant alleles in <i>M. m. musculus</i> .	Hunt & Selander (1973).
<i>Uroderma bilobatum</i> 2n = 38 ↓ <i>Uroderma bilobatum</i> 2n = 48	Proposed phenotypically adaptive effects of genes within 2n = 38 rearrangements.	Baker, Bleier & Atchley (1976). Baker (in press).
<i>Clarkia speciosa polyantha</i> ↓ <i>Clarkia nitens</i>	Proposed superior fitness of the <i>C. s. polyantha</i> genotype.	Bloom (1976).

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