Does hybridisation influence speciation?

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As Butlin et al. (2012) emphasise, hybridisation occurs almost inevitably during speciation. Moreover, studying patterns of introgression across the genome, and the spatial structure of hybrid zones, can tell us much about the genetics of divergence and reproductive isolation. Nevertheless, hybridisation may well have a negligible effect on the actual process of speciation. On the one hand, uniformly favourable alleles can spread so easily between diverging populations that the barrier to gene flow due to incipient reproductive isolation hardly affects their divergence. On the other hand, reinforcement and hybrid speciation - where selection within hybrid populations is crucial - may be so rare as to make little overall contribution. This is really a rephrasing of the old arguments that gene flow across a broad two-dimensional habitat may hardly impede divergence, so that parapatric speciation is almost as easy as allopatric (Clarke, 1966; Endler, 1977; Barton and Hewitt, 1981).

Before spelling out the argument in detail, it is important to distinguish two components of speciation: first, the establishment of the alleles that will be responsible for reproductive isolation, and second, the coupling together of independent incompatibilities. Alleles that will lead to reproductive isolation must spread. This spread will be difficult if they cause reproductive isolation from the beginning - for example, an underdominant allele or a set of alleles that are favoured only in combination. However, if reproductive isolation is not expressed initially, as in the Dobzhansky-Muller model (Orr, 1996), then they will not be opposed by selection, and divergence can occur

in a variety of ways.

So, incompatibilities may be established without great difficulty. However, speciation almost always requires multiple independent differences, and so divergent alleles that evolve independently must be coupled together to give two distinct reproductively isolated taxa. Without such coupling, independent incompatibilities would have different distributions, and we may just see a gradual increase in reproductive isolation with geographic distance. Coupling is automatic if divergence is strictly allopatric, or occurs across a single sharp ecotone. More generally, however, it requires some combination of range change, barriers to gene flow, and epistasis. Butlin et al. (2012) discuss this coupling process, suggesting that pre-existing incompatibilities can be recruited to strengthen local divergence that at first evolved as a direct response to a heterogeneous environment. This is just Felsenstein's (1981) "two allele" model, but is distinct from the usual process of reinforcement, in which reproductive isolation evolves as an adaptation. (It is important to distinguish the broad process of coupling from the buildup of linkage disequilibrium due to mixing of distinct populations, which depends on the "coupling coefficient", S/R; compare lines 236 and 290).

As two populations diverge, selection maintains differences at some loci, and impedes gene flow in the surrounding region of genome. If the populations overlap in broad sympatry, or abut in adjacent demes, then further divergence can only occur if selection is stronger than some threshold, proportional to the effective migration rate. This leads to a sieving effect which could produce "islands of speciation", and also favours chromosome rearrangements that reduce recombination, and so help maintain divergence.

However, the situation is quite different in a spatially continuous habitat. Then, very weak selection can maintain clines despite

free gene flow, and so divergence can proceed unimpeded. Pre-existing clines will attract each other, coupling together independently evolved components of reproductive isolation. However, this occurs only if the clines overlap: range change is a more plausible mechanism of coupling. Thus, if divergence occurs across an extensive range, the strength of barriers to gene flow may hardly affect divergence. This view is testable: it would be refuted if diverged loci do cluster in genomic "islands", and by the frequent involvement of chromosome rearrangements. Such observations would suggest divergence under high levels of gene flow (in sympatry or in adjacent demes).

Alternatively, barriers to gene flow could facilitate divergence under the classic Dobzhansky-Muller model, by slowing down favourable alleles for long enough that an incompatible allele arises at another locus, and meets the first allele to maintain a stable incompatibility (Kondrashov, 2002; Navarro and Barton, 2003). However, this effect is confined to a very small region of genome around divergent loci: unless linkage is exceptionally tight, simple geographic distance is more effective than a narrow hybrid zone in impeding the advance of a favourable allele. For example, the toads *Bombina bombing* and *B. variegata* are quite distinct, and only form hybrids within a narrow zone less than ~10Km wide; clines at marker loci imply a genome-wide barrier to gene flow equivalent to B~100Km (Szymura and Barton, 1991). Nevertheless, such a localised barrier would delay even a weakly favoured allele for a negligible time (Pialek and Barton, 1997). Divergence due to spatially heterogeneous selection, or to negative interactions with pre-existing incompatibilities, is insensitive to local barriers to gene flow, and will not lead to clustering of reproductive isolation along the genome.

Butlin et al. (2012, line 368…) argue that the rate of hybridisation may be far higher than the rate of mutation, and so may be an important source of novel variation. However, this is a misleading analogy: until isolation is nearly complete, we can

think of the set of diverging populations as essentially one population; hybrid zones separating diverging taxa will reduce gene flow, and so impede adaptations that are favoured everywhere. Overall, the supply of variation is only slightly reduced. Viewed from the opposite perspective, gene flow across hybrid zones allows faster adaptation than if it were absent, simply because the total gene pool is then larger than in any one fragment. However, these effects will not be large, unless fragments occupy a small fraction of the total range. The process of adaptation may hardly be affected by weak subdivision of the whole set of populations. For example, in *Heliconius* butterflies, alleles that change the warning pattern have passed between species: the pool of variation available for adaptation is wider than that held within any one species. Similarly, *Drosophila persimilis* and *D. pseudoobscura* are quite distinct, and yet exchange genes often enough that the ancestry of most of the genome does not reflect this species barrier (Noor et al., 2000). In such cases, adaptation will proceed no faster than in a single gene pool, consisting of all the hybridising species.

Complex alleles, consisting of tightly linked changes, may pass easily across hybrid zones, or between 'species', but this does not imply that hybridisation accelerates adaptation. In large populations, complex alleles can be constructed rapidly, by successive mutation, with no need for recombination: Karasov et al. (2010) show how this has happened several times in the evolution of pesticide resistance in *Drosophila melanogaster*.

Hybridisation leads directly to reproductive isolation in two ways: via selection for reinforcement of reproductive isolation, and through hybrid speciation. Pre-zygotic isolation will evolve as an adaptation to reduce the production of unfit hybrids (Wallace, 1889; Dobzhansky, 1940). Butlin et al. (2012) review the evidence for such reinforcement, which comes almost entirely from cases of broad overlap rather than from narrow hybrid zones. This is consistent with theoretical arguments that

modifiers of mate choice should only be able to evolve within hybrid zones if they carry little cost, and cause strong isolation (Sanderson. 1989). Thus, the feedback between reinforcement and further isolation (Butlin et al., 2012, lines 714...) may only occur in the final stages of speciation, once broad sympatry has already been achieved.

Hybridisation does have a distinct effect, in that it introduces sets of alleles characteristic of the divergent population. Loosely linked alleles that are favoured only in combination may remain together for a few generations, but it takes extremely strong selection and/or tight linkage for these to be established. This is the process that drives "hybrid speciation": hybrid genotypes can be established as distinct populations that are reproductively isolated from either parent species. Allopolyploid speciation is common in plants, and hybrid speciation may occur in a similar way when clonal reproduction or selfing predominate. However, it is hard for a hybrid species to be established in an outcrossing sexual population. Such cases can readily be detected, and the distribution of blocks of genome derived from either parent tell us much about their origin (e.g. Ungerer et al., 1998). Nevertheless, "hybrid speciation" (in the strict sense) is rare, and (polyploidy excepted) hardly influences the process of speciation overall.

So, we can understand the role of hybridisation in speciation in a rather simple way: individual alleles flow freely across most of the genome, even when selection maintains differences at many loci that contribute to reproductive isolation. The key point of this note - which is implicit in Butlin et al.'s (2012) review - is that if divergence is driven by selection, and if the population is spread over an extensive range, then the rate of divergence is hardly affected by what happens within narrow hybrid zones, or by occasional migrants: in that sense, hybridisation hardly influences speciation.

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