

“Evo-Devo” and the Conundrum of Sympatric Speciation

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Evolution is a spatiotemporal dynamical process, albeit an extremely complex one—too complex to yield to the type of mathematical arguments that over the past 100 years have revolutionized our understanding of the physical universe. Yet facets of evolution are revealed by mathematical models (Turelli et al. 2001), most recently those that address the question of how speciation is affected by the links between evolutionary and developmental biology. We are at the very beginning of elucidating how genotypes are translated into phenotypes through genetic expression, intracellular biochemical pathways, cellular signaling, organismal growth, and physiology—all of which may be influenced by environmental factors. How this translation occurs has a bearing on the question of speciation.

Rudimentary models linking phenotypes to genes in the context of postzygotic mechanisms of speciation (i.e., those occurring after mating and fertilization) are now being explored. The simplest consider unbranched chains or pathways of n genetic loci. In these chains, the amount of product produced by genes at locus 1 regulates the amount of product produced by genes at locus 2 through a promoter site associated with locus 2, and so on with loci 3 to n . The phenotype of the organism is assumed to be represented solely by the amount of product produced by genes at locus n . Gene effects in such systems are not additive, as they are in most models used in population genetics, but are nonlinear.

In unbranched chain models, the fitness of a phenotype under strong directional selection is ultimately determined by how well gene product j binds

with gene promoter site $j + 1$ for all loci $j = 1, \dots, n - 1$. Adam H. Porter and Norman A. Johnson are pioneers in the application of such “developmental genetic” models to questions of speciation (Porter and Johnson 2000). In a paper published recently in *Evolution*, they applied such a model to the question of speciation when selection favors identical new phenotypes in two populations that are only partially isolated with regard to gene flow (Porter and Johnson 2002). Gene flow is modeled by a parameter m that represents the probability that an individual in either population will migrate to the other population. Porter and Johnson contrast the results with those they obtained using a standard two-locus, two-allele Dobzhansky–Muller (D-M) genetic model with epistatic interaction. Their conclusion is that although both types of model allow postzygotic speciation to occur at surprisingly high levels of gene exchange, there are important differences that illustrate the significance of incorporating development in evolutionary models.

Dobzhansky–Muller models and developmental genetics models should not be thought of as alternatives. Rather, the unbranched-chain gene regulation model is a plausible scheme for providing the postzygotic speciation mechanism that is simply decreed in the D-M framework. This two-locus system with alleles A and a at locus 1 and alleles B and b at locus 2 is defined by incompatibilities existing between alleles a and b : Specifically, as assumed for purposes of comparison by Porter and Johnson, the five genotypes that do not include both a and b (that is, $AABB$, $AaBB$, $aaBB$, $AABb$, and $AAbb$) are

taken to be equally fit, while the four genotypes that do include both a and b (that is, $AaBb$, $Aabb$, $aaBb$, and $aabb$) are assumed to be inviable. The primary difference between Porter and Johnson’s developmental genetics model and their application of the D-M model is that the former has a fitness function that changes quantitatively with mutational changes to genotypes, so that phenotypes are subject to directional selection. The latter is qualitative with regard to fitness and does not include mutational changes to alleles, only changes in their relative frequency.

Porter and Johnson (2002) report results of simulations involving genetic changes in two populations of the same size N , with N varying among simulations ($N = 25, 50, 100, 250, 500$, and 1000) for different levels of gene flow m . The work responds to a call by Turelli and colleagues (2001) “to understand how much gene flow is needed to inhibit the accumulation of D-M incompatibilities.” Porter and Johnson’s results demonstrate that in both developmental genetic and standard D-M models, “speciation” over the span of 2000 generations can be expected to occur around 50 percent of the time if the gene flow between the two populations is limited to fewer than 0.1 percent of individuals, on average, in each generation (i.e., $m < 0.001$). They define speciation using the *ad hoc* criterion that the average fitness of F_1 hybrids of the two populations must be less than 10 percent of maximum fitness for the two populations to be considered separate species. Their analysis is robust in that they obtain similar results if their speciation threshold criterion is reduced from 10 percent to only 0.1 percent.

Not unexpectedly, Porter and Johnson find that the probability of speciation in a straightforward D-M model falls dramatically with an increase in the migration parameter m . More insightful are their findings that speciation is independent of population size N , that speciation occurs around 50 percent of the time when the probability that any individual migrates between the two populations is less than 0.1 percent, and that speciation still happens more than 5 percent of the time when the probability that any individual migrates between the two populations exceeds 1 percent. This latter case translates into 10 individuals on average swapped between each population each generation, which Porter and Johnson (citing Sewall Wright) note is a rate considered high enough to thoroughly mix neutral alleles.

The results from Porter and Johnson's developmental genetics model are more complex, the primary difference being that a population size effect (i.e., dependence on the value of N) and chain length effect (the number of loci in the regulatory pathway) are now evident. Speciation rates over 2000 generations are comparable to those in the D-M model when gene flows are less than 0.01 percent, and both models predict no speciation when gene flows are greater than about 5 percent. Between gene flows of 0.01 and 5 percent, a distinct population size effect is evident in the developmental genetics model: For migration rates around 1 percent, speciation rates in populations of size 25 are around 5 to 20 percent, while speciation rates in populations 10 times larger are around 37 to 47 percent. The analysis of the developmental genetics model is complicated by the fact that population extinction rates are much

higher in smaller populations. Additionally, chain length in the developmental genetics model affects speciation because the contribution of each locus in determining the overall phenotype is reduced when there are more loci in the regulatory pathway. Selection pressure on many mutations is thus lowered and more easily swamped by gene flow. The result is a reduced rate of speciation. Nevertheless, because incipient genetic divergence is harder to reverse in the developmental genetics model than in the D-M model, Porter and Johnson conclude that "development...facilitates the interactions among genes and gene products that make speciation more likely—even in the face of a strong gene flow."

Postzygotic isolation resulting from the type of hybrid inviability inherent in a D-M formulation or arising in a Porter and Johnson-type developmental genetics model is the stuff of speciation. The current challenge in a mathematical analysis of speciation, however, is not to model speciation in general but to model the relative importance of pre- and postzygotic mechanisms in facilitating sympatric speciation (Turelli et al. 2001, Via 2001). Two recent theoretical studies indicate that assortative mating arising in genomes containing loci for both ecological and mate preference traits is sufficient, under disruptive selection, for the occurrence of sympatric speciation (Tregenza and Butlin 1999). These models can now be extended to include developmental genetics components to see how rates of speciation are facilitated by postzygotic factors. Such extensions provide a means for assessing Porter and Johnson's conclusion about the significance of including developmental pathways in genetic models.

No one should doubt that pre- and postzygotic mechanisms acting in concert will result in enhanced speciation rates, whether under allopatric, parapatric, or sympatric conditions. Nor should anyone doubt that sympatric speciation rates will be enhanced by considerations of how the mosaic structure of most ecological communities can lead to spatial segregation of sympatric phenotypes (a condition that has been termed *heteropatry*). Consequently, it is still unclear to what extent incorporating a developmental genetics component in complex population models supports Porter and Johnson's contention that besides "act[ing] as a constraining force on evolutionary change...development can also act, equally importantly, as a creative factor in evolutionary change." Their contributions do, however, represent a necessary step forward in our quest to better understand how the interplay of genetics and development modulates speciation.

References cited

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