CHROMOSOMAL REARRANGEMENTS, RECOMBINATION SUPPRESSION, AND SPECIATION: A REVIEW OF RIESEBERG (2001)

The causal link between chromosomal rearrangements and speciation events is still considered by many to be unclear. Whether new arrangements arise after speciation to reinforce species' boundaries or, instead, serve as the primary isolation mechanism in speciation, is some what of an enigma to evolutionary biologists. Furthermore, the exact mechanism, by which these two outcomes arise, remains uncertain. Shedding new light on this quandary, Rieseberg (2001) offers a new theoretical mechanism by which these changes might facilitate speciation and/or provide a barrier to gene flow after a speciation event. Her contention arises from the observation that chromosomal rearrangements affecting specific loci may in fact inhibit recombination, thereby, extending there range of influence within the genome. While her theoretical mechanism is still largely untested, its ability to strengthen some current models – which otherwise seem less parsimonious, by relying too heavily on reduced heterospecific fitness – seems a compelling reason to further explore the theory.

Many traditional models of chromosomal speciation depend greatly upon the existence of small isolated populations, where drift can play a major role in determining the fixation of a new rearrangement. Furthermore, these models assume that these underdominant mutations impair hybrid fertility and/or viability, thus, generating species boundaries. These models often implore drift alone to engender fixation of mutations in very small populations, rendering them unlikely forces in speciation. None the less, many of the models presented do offer observational evidence to support their particular mechanisms. Rieseberg's main critic against these models, though, is that reduced gene flow between population appears to strongly enforce species

boundaries and that reduced gene flow can be produced without the total hybrid sterility or inviability inherent in these models.

The importance of this observation becomes more evident when one examines the differential effects of chromosomal rearrangements across organism types. To date, it has been shown that these types of mutations more strongly affect plant hybrid fertility/viability than animal hybrids. This conclusion stems from two complimentary lines of reasoning: [1] Sterility in plants is more likely because the male gamete often expresses more of its genome; meaning that, if a chromosomal abnormality is present in the pollen the plant gamete itself has a higher tendency to abort. This does not occur in animal sperm. Secondly [2], the XY type sex determination in animals has led to a very genetically sparse Y-type chromosome. The degenerate Y-chromosome facilities genetic, but not chromosomal, hybrid sterility. Even in plant populations, which use this system for sex determination, the degradation of the Y-chromosome is less apparent. The observation that sterility/inviability of plant hybrids is more often chromosomal, whereas sterility/inviability of animal hybrids is more often genetic constrains the current models, which depend so much upon this reduced hybrid fitness.

From these considerations, Reiseberg offers a more simplistic model to reduce gene flow after a chromosomal rearrangement, than heterospecific fitness. She observes that these rearrangements can strongly effect recombination without rendering the offspring infertile. If the rearrangement is within or linked to an isolating locus of the genome, then reduced gene flow will occur as a result of a lack of recombination. Chromosomal rearrangements can function in this way even when they do not affect hybrid fitness. This theory, however, is still limited because reduced gene flow itself may not cause speciation; furthermore, it is only with an accumulation of many neutral or weakly underdominant rearrangements, linked to isolation loci, that a larger

portion of the genome can be affected. One limitation of this model, however, is that is does not help determine whether the arrangement occurred before or after speciation. However, unlike many of the present models, it does allow for speciation in sympatry or parapatry, as well as, reinforcement of allopatrically derived species – which have come together in secondary contact. Reiseberg's new theoretical mechanism proves to be both interesting and plausible. Given further empirical data, it may serve as the new standard model of chromosomal speciation.

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