FIFTEEN

Speciation

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> Although clearly a major focus of evolutionary biology, the study of speciation has never emerged as a coherent discipline.
>
> —(Harrison 1991, *282)*

Speciation is the process by which ancestral species diverge into two or more descendant lineages. As such, speciation has occurred at each of the branching events that have generated the 8–14 million species thought to exist across the domains of life (Hawksworth et al. 1995; Mora et al. 2011). The theory of speciation has played an important role in the modern development of evolutionary thinking and indeed could be said to have been at the forefront of evolutionary theory since the publication of Darwin's (1859) *On the Origin of Species.*

The Diversity of Reproductive Barriers between Species

In this chapter we review speciation theory and describe how it has provided insights into the fundamental ecological, genetic, and geographic processes causing speciation. Our focus is exclusively on speciation in sexually reproducing organisms, which is commonly conceived of as the evolution of barriers to gene exchange (Mayr 1963; Coyne and Orr 2010). Isolating barriers are often divided into those acting before or after fertilization: Prezygotic barriers occur prior to the formation of a zygote and include ecological, temporal, geographic, behavioral, and mechanical barriers (collectively known as pre-mating barriers), as well as post-mating prezygotic barriers such as gamete interactions and female reproductive tract interactions. Postzygotic barriers include zygotic mortality, hybrid sterility, and hybrid inviability. Barriers resulting from interactions with the biotic or

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Table 15.1. The Theory of Speciation

Domain: The process of speciation through the evolution of reproductive isolation.

Propositions:

1. Barriers to gene flow can occur at both pre- and postzygotic life stages and can be driven by intrinsic and extrinsic factors.
2. Reproductive barriers can evolve owing to divergent evolution between allopatric lineages experiencing little or no gene flow.
3. Intrinsic postzygotic reproductive isolation can result from negative epistatic interactions between loci evolving independently in diverging lineages.
4. Selection to decrease costly hybridization favors the accumulation of prezygotic reproductive isolation between sympatric lineages (reinforcement).
5. Local adaptation to divergent environments can drive pre- and postzygotic isolation between lineages (ecological speciation).
6. Reproductive isolation can evolve between sympatric lineages experiencing gene flow.
7. Sexual selection can drive divergence in mating systems, contributing to reproductive isolation.
8. Genomic changes such as chromosomal rearrangements and polyploidy can contribute to divergence and reproductive isolation

abiotic environment are known as extrinsic barriers, while those that act independently of the environment are known as intrinsic barriers. This categorization of speciation barriers as extrinsic or intrinsic (table 15.1) does not imply that they all cause species divergence, since most speciation events likely involve a combination of such forces. Instead the ideas classify particular barriers to reproduction that together explain a lack of current gene flow between diverging lineages. Although they used different terms, Darwin and the early Darwinians also discussed isolating barriers and theorized how they might arise. However, the major work exploring the evolution of reproductive barriers began during the modern synthesis and has been extended since.

What Is Speciation? What Are Species?

Both empirical and theoretical speciation research ultimately rest on cogent definitions of species, a topic that is fraught with disagreement (Nathan and Cracraft, chap. 6). Nonetheless, we believe that discussions of speciation can proceed productively even in the absence of universal agreement on the definition of species. Species are divergent metapopulation lineages (de Queiroz 2007). However, this fundamental lineage concept of species fails to distinguish species from nonspecies, but instead argues that species as taxa can be recognized by a variety of criteria, including the biological species concept (Mayr 1942), the phylogenetic species concept

(Cracraft 1983), and others. However, if speciation is to be distinguished from within-species evolution, we need a criterion of species rather than a generalized concept of gradual divergence. The conflicting criteria for species (e.g., monophyly, diagnosability, reproductive isolation, evolutionary independence) are manifestations of fundamental genetic and ecological processes that are subject to the vagaries of Earth history, divergence times and rates of divergence via natural selection, and drift or other processes (de Queiroz 2007).

In this chapter, by species we mean divergent forms that can overlap spatially and maintain differences at multiple regions of their genomes. These genomic differences might be maintained via intrinsic incompatibilities or extrinsic, ecological selection. For geographically or temporally separated forms the definition becomes somewhat arbitrary, but we do not believe that this hinders understanding models of speciation. Speciation theory can focus on how species diverge to achieve the ability to overlap spatially or in other words to achieve reproductive isolation—if not across the whole genome then at least at key genes that allow further phenotypic and genetic divergence.

The Goals of Speciation Theory

Speciation theory can help both to explain findings from empirical studies and to provide testable predictions to guide future empirical studies. Speciation theory is built on a large foundation of verbal models based on empirical observations and experimental evidence. Most of these verbal ideas are based on an understanding of how geography (allopatry versus sympatry) and timing (pre- versus postzygotic) influence the evolution of reproductive isolation (proposition 1, table 15.1)—an understanding that does not necessarily depend on mathematics (Turelli et al. 2001). Built on these verbal arguments are many hundreds of mathematical and numerical studies detailing which specific conditions are conducive to the evolution of barriers to reproduction. The sheer number of complex mathematical models can make a broad understanding of general speciation theory challenging (Kirkpatrick and Ravigné 2002). It can also provide an impenetrable shield to empiricists attempting to find broader meaning in their results.

There are many communication difficulties between empiricists and theoreticians in the realm of speciation. For example, empiricists are often interested in and tend to investigate the evolutionary forces acting on existing trait variation. How much reproductive isolation does the trait confer? Is the trait under natural selection or sexual selection? Is there migration between populations or gene flow between species? By contrast, when theoreticians study speciation they generally investigate the evolutionary conditions that allow divergence to occur (Turelli et al. 2001) and are less interested in which traits might contribute to reproductive isolation. How strong does nonrandom mating need to be? How strong does natural selection or sexual selection need to be? Can there be gene flow and if so how much?

Another example of a gap between theory and empirical studies concerns how biologists measure reproductive isolation. Empiricists will often conduct crosses or compare fitness of hybrids and parental types to estimate the degree of isolation between species (Sobel and Chen 2014). By contrast, theoreticians often assume a simple measure of genetic differentiation as an index of a history of isolation, such as when species differ in their allelic identities within a set of polymorphic loci. Gametic association of alleles at different loci between species, also known as linkage disequilibria, provide another common measure of the progress of speciation (see the seminal theoretical papers on speciation of Felsenstein 1981; Kirkpatrick and Ravigné 2002). In models leading to the evolution of reproductive barriers, the reduction of gene flow causes elevated linkage disequilibrium. Without reproductive isolation, in a population characterized by random mating and recombination, linkage disequilibria will decline to zero exponentially with time. As reproductive isolation evolves, gametic associations accumulate between alleles at multiple loci that differentiate species. Strong linkage disequilibrium is also implied by our definition of species as divergent forms that can overlap spatially and maintain differences at multiple regions of their genomes. In contrast, empiricists rarely discuss reproductive isolation in terms of linkage disequilibrium, although other measures of genetic differentiation are sometimes used (Coyne and Orr 1997).

The proliferation of highly specific mathematical theories is likely a result of the complexity of evolutionary factors influencing speciation. Selection, epistasis, gene-by-environment interactions, nonrandom mating, spatial structure, and gene flow are all difficult to model singly, and combining them all into a broad theory of speciation is exceedingly difficult (Kirkpatrick and Ravigné 2002; Gavrilets 2014). Furthermore, it is widely accepted that, in most cases, speciation results from the evolution of many reproductive barriers. Mathematical models do not yet account for the diversity of evolutionary forces affecting these multiple barriers. The solution to this complexity has been to develop models that investigate a particular scenario or set of circumstances for a certain type of barrier to reproduction. Hundreds of these models, both analytical and using numerical simulations, have been published, each exploring specific combinations of evolutionary forces with specific assumptions. In the absence of a general mathematically based theory about speciation, we often fall back on verbal theories to guide our understanding of the evolution of reproductive isolation.

Evolution of Reproductive Isolation in Allopatry

Mayr firmly established the importance of allopatry in speciation, a paradigm that is not strongly challenged today (proposition 2, table 15.1). We currently have methods, albeit indirect and subject to bias, to estimate the proportion of speciation events on a phylogeny that may have occurred in allopatry or involved some sort of sympatry (Barraclough and Vogler 2000; Fitzpatrick and Turelli 2006). Some of these methods implicitly or explicitly assume that allopatry is the expected speciation scenario in the absence of additional evidence—the null model. But it is not obvious that the null model of allopatry should be accepted even if the simple alternative hypothesis of sympatry is rejected. Given the increasing prevalence in the literature of scenarios of speciation involving gene flow at some time or another, lack of evidence for secondary contact or ongoing genetic or behavioral interactions between diverging species is not a solid basis for accepting that species have evolved in allopatry.

The increasing interest in sympatric speciation, reinforcement, porous species boundaries, and other scenarios involving interactions between individuals and populations raises questions: For populations that have diverged in allopatry, is secondary contact necessary to complete the speciation process? What proportion of speciation events have occurred solely in allopatry without some gene flow? The challenges in answering these questions lie largely in the difficulties of reconstructing historical ranges of species accurately on recent or ancient timescales. Although new tools such as niche modeling and geographic information systems permit informed guesses as to the dynamics of species ranges in the past, and fossils occasionally provide insight into whether diverging species experienced sympatry in the past, it is rare that these multiple sources of data allow us to reconstruct the geography of speciation unambiguously.

Theories of Intrinsic Postzygotic Barriers to Reproduction

How postzygotic reproductive isolation could evolve was one of the first conundrums of speciation theory. How do two lineages diverge such that their hybrids are inferior but neither of the two lineages passes through a period of low fitness? This mystery plagued Darwin as he was working through his theories on natural selection and the evolution of species. Darwin (1859) argued that natural selection could not favor hybrid sterility or hybrid inferiority. He argued that rather than being intended and the essence of speciation, as creationists such as Buffon (1753) had postulated earlier, hybrid sterility and inviability were accidental by-products of evolutionary divergence. New insights into this problem were gained during the modern synthesis as a new understanding of genes and population genetics emerged. These theories centered on understanding how genic intrinsic postzygotic reproductive isolation can evolve.

Dobzhansky-Muller Incompatibilities (DMIs)

It was proposed that epistatic interactions between alleles at different genes could result in intrinsic reproductive isolation (proposition 3, table 15.1). Although this idea was initially discussed by W. Bateson (1909), the genetic hypothesis was first clearly developed by Dobzhansky (1937a) and Muller (1942), and so loci that show this pattern are often today referred to as Dobzhansky-Muller incompatibilities (DMIs). In the basic verbal theory, two lineages that diverge in allopatry (or with virtually no gene flow between them) can accumulate neutral or selected substitutions at different genes. Within each lineage, genes evolve without the opposition of intermediate steps or allelic combinations by selection. Then, crosses between these two diverged lineages bring together divergent alleles in combinations never before seen by natural selection. These new combinations of alleles have negative interactions that make hybrids unfit, sterile, or inviable (fig. 15.1).

Based on the negative epistatic mechanism of incompatibility, theory predicts that the numbers of DMIs will accumulate faster than linearly as the number of substitutions differentiating the two species increases. The numbers of DMIs are expected to "snowball" and accumulate with the square of the number of substitutions separating two diverging lineages; if three or more loci generate an incompatibility, the snowballing effect will be even faster (H. A. Orr 1995; H. A. Orr and Turelli 2001). Because each new substitution in one lineage has the potential to interact with any of the previous substitutions, the number of possible interactions between substitutions is much greater than the raw number of substitutions. The beauty of the overall idea is that DMIs will inevitably accumulate between isolated populations as divergence occurs between species, even if neutral

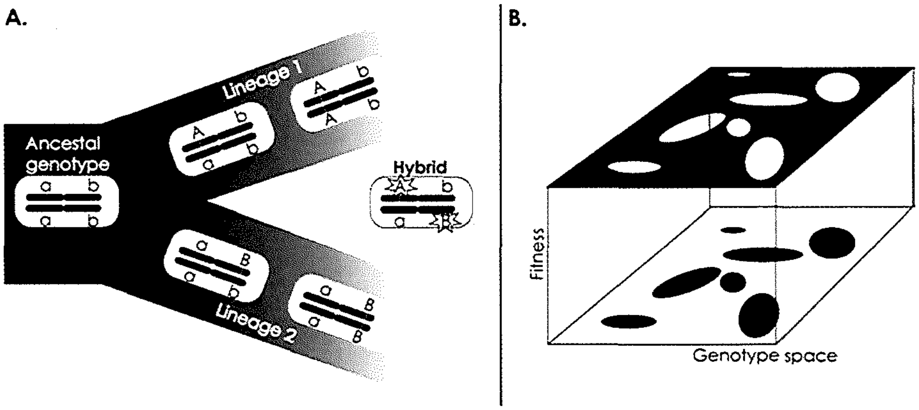

Figure 15.1. Negative epistatic interactions between loci can cause hybrid inviability or sterility. (A) Schematic of the evolution of a Dobzhansky-Muller-incompatibility. Two loci independently evolve in diverging lineages such that one lineage has a mutation in one locus ($a \rightarrow A$) while the other lineage has a mutation in a second locus ($b \rightarrow B$). In the hybrid the two derived alleles may interact to reduce hybrid fitness. (B) A schematic of a holey landscape as described by Gavrilets (1997) in which fitness is either zero or one. The genotypic space is scattered with regions of incompatibilities causing low fitness.

drift within species is the only driving force. In its simplest form, evolution within species is neutral and the field of gene combinations is a "holey landscape": a flat surface representing equal fitness, while some gene combinations are in deep "holes" of low fitness (Gavrilets 1997).

Recent Studies on Incompatibilities

Research in the past two decades has clarified the relationships between DMIs and speciation. The early 1990s was a time of renewed awareness of DMIs and the first empirical characterizations of putative incompatibilities (H. A. Orr 1993; H. A. Orr and Turelli 1996; Presgraves and Orr 1998; Presgraves et al. 2003; Phadnis and Orr 2009). This work frequently focused specifically on hybrid breakdown and attempted to link the hybrid breakdown observed in the laboratory, and sometimes confirmed in nature, with the speciation process. Because they cause hybrid inviability or sterility, DMIs became termed "speciation genes"—a "perhaps unfortunate term" (H. A. Orr 2005). It has subsequently been argued that many such speciation genes might not have been the original drivers of speciation because they could have evolved long after speciation was complete (Via and West 2008; Mallet 2010; K. L. Shaw and Mullen 2011).

Separation of populations in allopatry was classically considered a key requirement for the evolution of DMIs (Orzack and Sober 2001; Coyne and Orr 2004). It is commonly acknowledged that all species segregate deleterious recessive mutations (some causing lethality), and through the same logic recently it has been observed that deleterious incompatibilities between loci can also segregate within populations (Corbett-Detig et al. 2013). Thus, DMIs could play an important role in the distribution of fitness effects not only between species but within species as well. Given that empirical examples of DMIs often reveal positive selection (Tang and Presgraves 2009; Sweigart and Flagel 2015; Tang and Presgraves 2015), the upper surface of the holey landscape (fig. 15.1B) is unlikely to be completely flat for these loci, and this positive selection may counter some level of gene flow. Complete allopatry may not be required.

The verbal theory of the origin of DMIs has been validated not only mathematically (H. A. Orr and Turelli 2001), but also via empirical investigations of gene loci in *Drosophila* that epistatically generate hybrid incompatibility (Presgraves et al. 2003; Brideau et al. 2006; Masly and Presgraves 2007; Tang and Presgraves 2009). Experimental evolutionary studies in yeast have perhaps yielded the clearest picture yet of the relationship between divergent evolution in allopatry and the genic basis of DMIs (Dettman et al. 2007; J. B. Anderson et al. 2010; Stukenbrock 2013), as well as compelling examples of ecological speciation. Although the relevance to natural systems can always be debated, these studies yield sensible and intelligible connections between the genes exhibiting DMIs in hybrids and their role in divergent ecological settings in ways that genetic studies on long-diverged species in nature have not.

The snowball theory of the accumulation of DMIs over time has been tested in animals (Matute et al. 2010) and plants (Moyle and Nakazato 2010), and in a meta-analysis (Gourbière and Mallet 2010), all of which generally confirm the nonlinear accumulation of incompatibilities predicted by the theory. The logic of snowball DMI evolution is compelling and successfully explains Haldane's rule (see below). Two problems may lead to difficulties with tests using comparative data: First, stochastic accumulation of a few incompatibilities initially, each of which may have quite large and variable fitness effects on hybrids (Turelli and Moyle 2007), can result in very noisy changes in hybrid fitness, making the expected curvature hard to detect (Gourbière and Mallet 2010; Turelli et al. 2014). Second, the extent to which other, nonepistatic incompatibilities such as underdominance of chromosome rearrangements, or the effects of additive loci undergoing quantitative genetic adaptation to divergent niches, contribute to hybrid inviability and sterility between species is still unclear (Gourbière and Mallet 2010). In many systems, it is likely that ecological divergence and mate choice contribute more to reproductive isolation between recently diverged species than incidental DMIs (Schemske 2010; Turelli et al. 2014). Overall the prediction and testing of the snowball effect represent major refinements in our understanding of the genetics of incompatibilities, but the extent to which DMIs cause speciation in nature remains unclear.

Haldane's Rule

Perhaps the most famous examples of DMI evolution obey Haldane's rule, which states that if hybrids of one sex suffer most from inviability or sterility, it is most likely the heterogametic sex, the sex with different sex chromosomes (Haldane 1922). The heterogametic sex is the male (XY) in *Drosophila* and mammals, or the female (ZW) in birds and butterflies. Haldane's original observation sparked decades of empirical work investigating the generality of the rule as well as theoretical work attempting to understand the causal mechanism of the pattern. To date, four general theoretical explanations for Haldane's rule have been put forward: dominance theory, faster-male theory, faster-X theory, and meiotic drive (Schilthuizen et al. 2011). Each of these explanations consists of a cluster of models within the constitutive theory of speciation. The genetic scenarios described in these theories are extensions of scenarios in DMI evolution, albeit applied to the heterogametic sex.

The dominance theory posits that homogametic hybrids, for example, male birds (ZZ) or female monkeys (XX), will suffer only from DMIs that are dominant. Those loci may be linked or unlinked to the X or Z sex chromosomes and do not cause a major bias between the sexes. F1 hybrids will additionally suffer from DMIs affected by recessive alleles on the X or Z chromosome that are expressed only in the heterogametic sex, although the interacting partner loci may be dominant and on autosomes. In the flour-beetle *Tribolium*, autosomal loci differing between geographic populations as well as environmental effects influence the expression of Haldane's rule, as well as sex-linked recessives; this suggests that local adaptation to different environments may be a driver of the DMIs involved in Haldane's rule (Wade et al. 1994; Wade et al. 1999).

In addition, faster-male theory proposes that more rapid selective processes occurring in males (e.g., sexual selection on seminal fluids involved in sperm competition) lead to an accumulation of greater hybrid sterility or inviability in males than in females. A third idea, faster-X theory, proposes that the sometimes heterogametic X or Z chromosome evolves faster because advantageous recessive mutations will be exposed to selection in the heterogametic sex more rapidly than in the homogametic sex. Even after incorporating the frequent observation of faster evolution for genes with male-biased expression that presumably underlie male traits subject to sexual selection, recessiveness on the X chromosome was still required to explain Haldane's rule (Turelli and Orr 1995). Both faster male evolution and a more rapid accumulation of DMIs on the X or Z chromosome have been observed in natural systems (e.g., Masly and Presgraves 2007). However, for obvious reasons, faster-male theory cannot explain Haldane's rule in birds and butterflies, whereas dominance theory certainly can. It is unclear how great a role faster-X theory plays in general, but in *Drosophila* there is some evidence for more rapid accumulation of hybrid incompatibility-causing alleles on the X than on the autosomes (e.g., Masly and Presgraves 2007).

Other proposed mechanisms leading to Haldane's rule include accumulation of suppressors of meiotic drive on the sex chromosomes. Early statements of this model (Frank 1991; Hurst and Pomiankowski 1991) envisioned suppressors of meiotic drive accumulating on the sex chromosomes, causing rapid co-evolutionary arms races that increase divergence and incompatibilities between species. Empirical results consistent with this model have appeared, mostly in *Drosophila*, but a number of caveats apply, not the least of which is the inability of the theory to explain the accumulation of meiotic drive suppressors on sex chromosomes as opposed to autosomes (McDermott and Noor 2010). In conclusion, meiotic drive and a bias toward faster sex chromosome evolution, together with faster male evolution in male-heterogametic species, all play a role in Haldane's rule incompatibilities in *Drosophila*, but linkage of a recessive allele to an asymmetrically inherited genomic component (the dominance theory), usually a sex chromosome, is required for the very widespread obedience to the rule (Turelli and Moyle 2007).

Reinforcement and the Evolution of Prezygotic Reproductive Isolation

Alfred Russel Wallace (1889) was the first to lay out a coherent argument that reproductive isolation could evolve by natural selection, even though, in earlier correspondence, he had seemingly accepted Darwin's (1868) arguments against it. His idea, sometimes termed the Wallace effect (N. A.

Johnson 2008), was elaborated and refined during the modern synthesis, and when applied to the evolution of assortative mating, is today termed reinforcement. The general verbal theory asserts that two lineages, after diverging in allopatry, produce hybrids that may suffer from postmating incompatibilities when the two meet in secondary contact. The cost of producing hybrids or of mating with the other lineage will create a selection pressure to increase prezygotic reproductive isolation. Reinforcement is therefore the evolution of prezygotic reproductive isolation via selection to reduce the costly effects of hybridization (proposition 4, table 15.1; fig. 15.2).

The history of reinforcement research is marked by controversy (N. A. Johnson 2008). During the modern synthesis, Dobzhansky (1937a, 1940) championed the theory of reinforcement. He and others performed empirical studies documenting patterns of reproductive isolation consistent

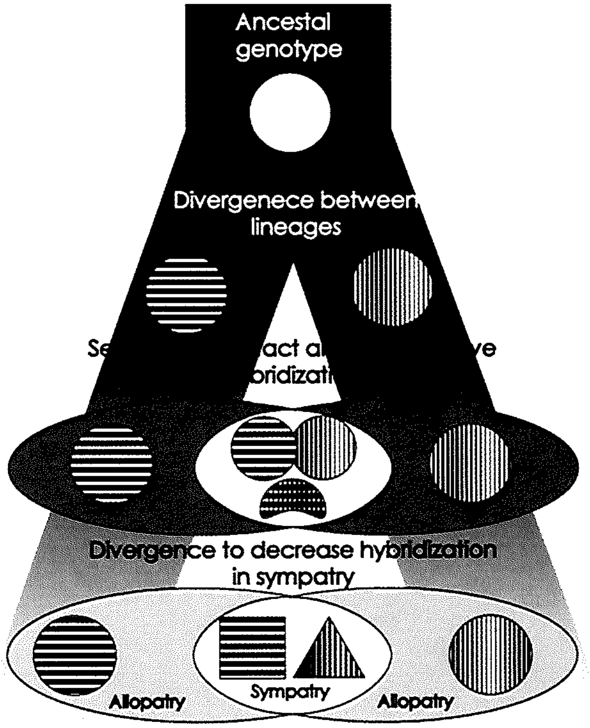

Figure 15.2. A schematic of the process of reinforcement. Two lineages diverge from a common ancestor and, in secondary contact, produce maladaptive, sterile, or inviable hybrids. This creates selection to decrease hybridization and leads to further divergence between the lineages in sympatric populations.

with reinforcement and performed experimental evolution to demonstrate the feasibility of the process. On the other hand, although Ernst Mayr (1942, 1963, 1970) believed that mating behavior evolved to aid species recognition, he argued that evolution of divergent mating behavior was normally achieved in allopatry, rather than by a process of reinforcement. Theoretical results accumulated that appeared to support the feasibility of reinforcement (Maynard Smith 1966; Dickinson and Antonovics 1973). However, by the 1980s additional theory suggested that gene flow and recombination would often prevent a buildup of linkage disequilibrium between divergently selected loci that caused postzygotic isolation and loci that affected mating behavior (Felsenstein 1981; Spencer et al. 1986; Butlin 1987). This sort of recombination may lead one species to go extinct because, for example, it acquires alleles that cause it to be attracted to the other species and to produce offspring with reduced fertility or viability.

Despite theoreticians' doubts, empiricists continued to report examples of reproductive isolation that likely evolved owing to reinforcement. Testing for reinforcement was popular in the 1980s and was reinvigorated by landmark studies in *Drosophila* and other groups, confirming key predictions of the reinforcement model, including a greater propensity to mating assortatively in areas of sympatry with sister species and a decrease in fitness of interspecific hybrids (Noor 1995). The identification of specific genes contributing to reinforcement in natural populations has also reinvigorated this model (Hopkins and Rausher 2011), and the measurement of reinforcing selection in a natural system has demonstrated the role that it can play in speciation (Hopkins and Rausher 2012).

Accompanying the abundance of empirical examples of reinforcement has been a deluge of theoretical studies investigating the feasibility of reinforcement as a function of the strength of selection, the extent of gene flow, and the genetic architecture of traits (Liou and Price 1994; Servedio and Kirkpatrick 1997; Barton and de Cara 2009). Clearly reinforcement suffers acutely from the problem faced by most speciation theory—too many specific models and little general theoretical framework. Nevertheless, there is currently a general understanding that reinforcement can occur within a wide range of parameters (N. A. Johnson 2008). As discussed below, selection can be strong enough to maintain high linkage disequilibrium in the face of gene flow and recombination. Reinforcement has thus emerged as an increasingly viable hypothesis for species divergence in natural systems, particularly when researchers seek it in appropriate study systems and use a carefully controlled experimental design.

One of the reasons why reinforcement may be more successful in nature than the models of Felsenstein (1981) and others would predict is that genetic divergence, for instance in ecological adaptation, can often have an indirect or pleiotropic effect that can lead to assortative mating. For example, divergence in ecological niche may entail habitat choice, but because individuals tend to mate with others in the same location, this divergence will also affect mating behavior (Diehl and Bush 1989). Any ecologically divergent trait that also affects mating behavior can thus improve the chances of speciation (Servedio et al. 2011).

The direct experimental evidence for reinforcement, however, does not answer questions about the generality of reinforcement; for these, comparative studies are required. Perhaps the most comprehensive dataset that exists is that compiled by Coyne and Orr (1997) for *Drosophila* species. Taking genetic divergence between species pairs as a surrogate for time since separation, the study examined levels of premating and postmating variation as measured in captivity. The study compared sympatric and allopatric species pairs and showed that in both, postmating isolation evolved at roughly the same rate. Premating isolation in currently allopatric species evolved at a similar rate to postmating isolation, but in sympatry premating isolation seems to have evolved much faster than postmating barriers. The greater rate of assortative mating evolution in pairs of sympatric species argues that reinforcement was likely pervasive.

Recently, Coyne and Orr's data were updated and reanalyzed in the hopes of finding some patterns in the causes of premating isolation. Curiously, in recently evolved pairs of sympatric species, there was no evidence that the strength of postmating incompatibilities, host plant differences (a surrogate for ecological postmating isolation), or X-chromosome size, which is expected to be correlated with increased rapidity of DMI evolution via Haldane's rule, had any effects on mating isolation (Turelli et al. 2014). And yet around half of all recently evolved species overlap spatially, show strong mating isolation, and are probably separated in some way ecologically. These results suggest that underestimates of the strength of postmating isolation in sympatric species are caused by inaccuracies of laboratory assays and shifts in host plant use. Although geography is undoubtedly involved, "the pervasiveness of the reinforcement pattern and the commonness of range overlap for close relatives indicate that speciation in *Drosophila* is often not purely allopatric" (Turelli et al. 2014, 1176).

Reproductive Isolation through Local Adaptation to Divergent Environments

Ecological Speciation

In the past twenty years, the role of ecology in speciation has garnered renewed attention, with diverse scenarios coalescing under the title of "ecological speciation" (proposition 5, table 15.1). During the modern synthesis, ecological and habitat differences were found to be widespread among closely related species in nature, and ecological divergence formed a key component of the process of speciation (Turesson 1922; Clausen et al. 1939; Mayr 1963; Schluter 2000; Rundle and Nosil 2005; Nosil 2012). The notion that ecological adaptation drives divergence and eventually speciation has a strong and consistent history in the plant literature (Lowry 2012), although research in animals has been increasing. The more recent increase in appreciation of ecology in speciation was driven in part by new statistical tools that allowed the measurement of natural selection in the wild and the fitness of hybrid individuals in the habitats of the parental species as well as intermediate habitats (Hoekstra et al. 2001; Kingsolver et al. 2001; Ramsey et al. 2003; Wang et al. 2013). Ecological speciation has also become more popular because of the ability to employ data from both the field and experiments in the lab to test hypotheses about mechanisms (Lowry et al. 2008a). Finally, the recent focus on ecological speciation (Nosil 2012) has been driven largely by spectacular findings from natural, nonmodel systems, pointing to mechanisms whereby adaptation to novel environments or niches ultimately results in the evolution of pre- or postzygotic isolating mechanisms in nature (Rundle et al. 2000; Hawthorne and Via 2001; Jiggins et al. 2001; H. D. Bradshaw and Schemske 2003; Lowry et al. 2008b). These systems demonstrate ecological causes of divergent selection; evidence for ecological differences with pleiotropic effects on assortative mating; and linkages between ecological selection and genes driving reproductive isolation (Rundle and Nosil 2005).

The theoretical foundations for ecological speciation can be traced back to Darwin's principle of divergence. In general, empirical studies (e.g., host shifts of Rhagoletis fruit flies from hawthorn to apple, G. L. Bush 1969; Feder et al. 1988; McPheron et al. 1988; Berlocher 2000; Feder et al. 2003) have played a more prominent role in the development of the field of ecological speciation than has mathematical theory (M. R. Orr and Smith 1998; Schemske 2010; Nosil 2012). However, the two approaches are complementary, with hybrid sterility studies emphasizing genetic mechanisms of incompatibility and ecological genomics emphasizing the generation of incompatibilities via ecological adaptation. Although some argue that the focus on ecological speciation is hardly new (Harrison 2012), it is arguably an advance over the more restricted search for speciation genes if only because it explicitly incorporates ecological and behavioral drivers of divergence and their consequences for genomic divergence and incompatibility.

The Evolution of Reproductive Isolation in Sympatry

Speciation in sympatry, or in the presence of gene flow, has been far more controversial than speciation in allopatry (proposition 6, table 15.1). In his monumental work *Animal Species and Evolution*, Mayr's (1963) emphasis on allopatric speciation led many in the field to discount the possibility of sympatric speciation. Mayr's insistence, longevity, and strong influence on the field led to the idea that allopatric speciation should be a strong null hypothesis that should generally be accepted, unless a clear set of tests favor sympatric speciation or speciation in the face of substantial gene flow (Coyne and Orr 2004). Many complex patterns of phenotypic variation, including ring species (Moritz et al. 1992; Irwin et al. 2001; Alcaide et al. 2014), appear compatible with allopatric speciation, or at least with parapatric speciation accompanied by limited gene flow. But even Mayr (2002) eventually admitted the possibility and occasional occurrence in some groups (especially freshwater fishes) of sympatric speciation. As stated previously, it is inappropriate to accept allopatric speciation as a null model merely because it cannot be rejected. Additionally, recent findings of cryptic introgression between phenotypically distinct species in many groups, such as hominins (Green et al. 2010), birds (Rheindt and Edwards 2011), and butterflies (S. H. Martin et al. 2013), suggest that more complex speciation scenarios than simple isolation in divergence may often be warranted.

Others, particularly those working with host-specific parasitic or phytophagous insects, have long argued that there are simply too many species to be explained entirely by the slow grind of Earth's geography. Additionally, the evolution of ecological niches can create mating barriers between derived and ancestral populations (G. L. Bush 1975b, 1975a; White 1978; P. W. Price 1980). Pleiotropy between locally adapted traits and reproductive isolating barriers will be abundantly present in the adaptation of, for example, a parasitic insect to a new host. This debate in the speciation literature has sparked theoretical investigations of how reproductive isolation evolves with gene flow between diverging lineages.

Models of Sympatric Speciation

We can trace the origin of quantitative models of sympatric speciation to Maynard Smith's (1966) model involving two niches, the establishment of a stable polymorphism for the ecological (or postmating isolation) trait, and the subsequent evolution of premating reproductive isolation. This classic paper was not so much an argument for sympatric speciation as an outline of the conditions under which it might happen. The most favorable situation proposed by Maynard Smith was what he called pleiotropism: a single-locus ecological trait causing habitat selection that also has a pleiotropic effect on mating behavior. Maynard Smith raised and immediately dismissed the idea in three lines as very unlikely. Yet in modern and perhaps less clear terminology "pleiotropism" is none other than a "magic trait," an ecologically adapted trait that also acts as a barrier to reproduction. Today this seems rather more likely than hitherto (Servedio et al. 2011), and it was the basis of earlier verbal arguments for sympatric speciation (G. L. Bush 1975b, 1975a). Felsenstein (1981), in a highly influential paper, also regarded pleiotropy as an unlikely one-allele model, giving the Maynard Smith model as an example. Felsenstein pointed out that recombination between loci conferring divergent ecological advantages in specific habitats and loci specifying assortative mating could make sympatric speciation difficult because disequilibria between the loci would be degraded. By the 1980s, many considered sympatric speciation to be virtually impossible.

Consider the following argument. Suppose m is the rate of gene flow between two incipient species, and after every gene flow event introgressed genomes recombine randomly. Magic trait models assume that $m < 0.5$ between divergently adapted genotypes, which favors speciation because divergence due to natural selection becomes easier than in panmictic populations. However, the greatest challenge is to model speciation in a single population with panmixia, where $m = 0.5$ initially between the incipient species. For this reason, models of sympatric speciation usually start with panmixia.

A recent innovation in modeling the combination of population ecology and evolutionary dynamics is known as adaptive dynamics. Traditional population genetic models have often ignored population growth, possibly to the detriment of their realism. Adaptive dynamics models of speciation suggest a relatively wide set of conditions under which sympatric speciation might take place (Dieckmann and Doebeli 1999). Adaptive dynamics theories typically propose a phenotypic model of evolutionary divergence based on demography and ecological competition. Speciation is envisaged as a branching process where one population splits into two phenotypic clusters as a result of ecological pressures (Metz 2011). Unlike population genetic models, most adaptive dynamics theory does not explicitly include genetic considerations or reproductive isolation. Rather, it explicitly models population growth and the demography of ecological competition as drivers of divergence. Darwin (1859) proposed his own principle of divergence based on Malthusian ideas of geometric population growth and ecological competition that drove divergence in very much the same vein, and the adaptive dynamics community therefore claim that they are modeling Darwin's ideas more closely than traditional population genetics models (Metz 2011). Dieckmann and Doebeli's (1999) innovation to the adaptive dynamics literature was to add a simple quantitative genetic model of an ecological phenotype and genetic assortative mating to the adaptive dynamics models. The reason that branching took place was that competition for the available resource spectrum drove the populations apart, with the populations acquiring premating isolation as a result of reinforcement.

However, the adaptive dynamics model may be unrealistic. In the Dieckmann and Doebeli (1999) model, branching took place because the model allowed mutations only of small effect so that there was better exploitation of the resource when there were two isolated panmictic species than if one was occupying the center of the resource. If variable effect mutations were allowed, a single population perfectly exploiting the Gaussian resource would be able to evolve that could not be invaded by another species (Polechová and Barton 2005). The Dieckmann and Doebeli model predicts that linkage disequilibrium between the ecological and mating loci will develop, but it requires an initial chance deviation from linkage equilibrium on which selection is able to act. This model provides an interesting case in which random drift might kick-start a higher-order selective process.

Today's many models of sympatric speciation can be viewed as extensions of a simple and very early single-population model of migration/selection balance (Haldane 1930, 1932). Haldane showed that a one-locus polymorphism will be maintained provided that divergent selection is greater than gene flow (fig. 15.3). The same will be true for more loci, although the overall selection will now act on multilocus genotypes, and a buildup of linkage disequilibrium comes into play, so that recombination as well as gene flow is important. Although we have highlighted the influential although contested contribution of Dieckmann and Doebeli (1999), many others before and since also have shown that sympatric speciation

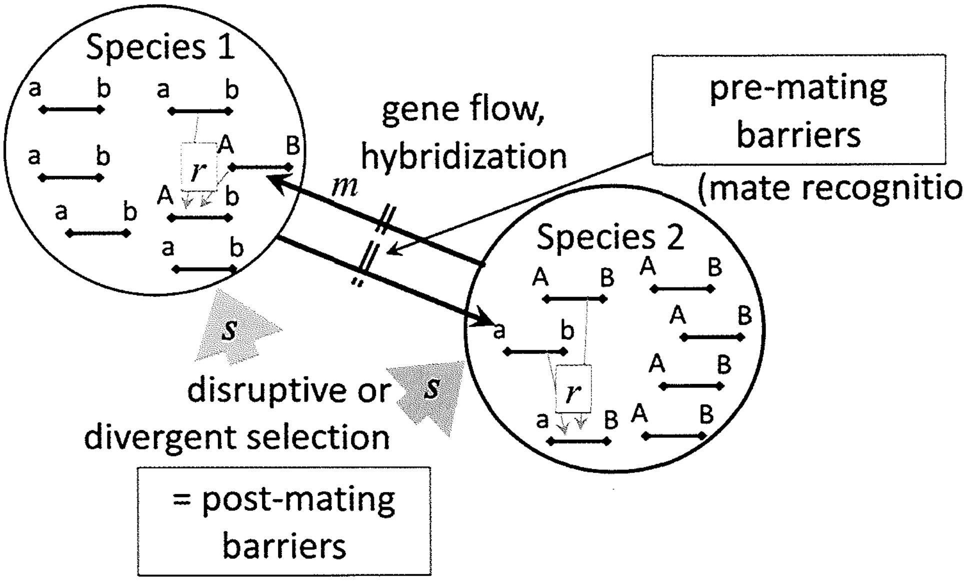

Figure 15.3. Simplified diagram of speciation with gene flow. Selection (s) for divergently selected loci A, B in two different ecological niches allows some differentiation to take place, maintaining a balanced polymorphism (due to migration-selection balance) and linkage disequilibrium of adapted loci between niches. This is opposed by gene flow (m) and recombination (r) that can produce less-well-adapted or even deleterious intermediates (i.e., DMIs, see fig. 15.1A) within each niche. Such polymorphisms must be selected strongly enough to avoid swamping by gene flow: only if $s > m$ will such a polymorphism exist. A second step in most models of sympatric speciation is reinforcement: the evolution of assortative mating can then further reduce gene flow. The combination of these two steps can then allow more weakly selected divergent loci to evolve, which in turn causes an increase in cumulative selection against migrants and recombinants and further selection for reinforcement.

is possible under a wide variety of circumstances (Maynard Smith 1966; Diehl and Bush 1989; Fry 2003). It is clear that sympatric speciation can occur in theory; the only remaining questions are whether the models are realistic and how often sympatric speciation occurs in nature (Coyne and Orr 2004).

Sexual Selection and the Evolution of Reproductive Barriers

Sexual selection is differences in mating success among members of one sex. Advantages of mate choice may be direct (e.g., ensuring parental assistance) or indirect (e.g., superior fitness of offspring). There are two major flavors of indirect sexual selection, usually termed good genes models or runaway models. In good genes models, the offspring have superior fitness in general, while in runaway models, the offspring merely have superior mating success that may conflict with ecological fitness (Kirkpatrick and Ryan 1991). Speciation by reinforcement is a form of good genes sexual selection (proposition 7, table 15.1). Some models of mate choice, such as those envisioning an avoidance of "bad genes" (Iwasa et al. 1991), may capture certain aspects of reinforcement as well.

Verbal theory predicts that divergent mating preferences and sexual signals arising in allopatry could impede the merging of species upon secondary contact (Ritchie 2007; Safran et al. 2013). Sexual selection may also accelerate speciation in sympatry (Higashi et al. 1999), but unambiguous empirical examples of the process are rare or nonexistent. While sexual selection can promote speciation, it may impede speciation in sympatry (Kondrashov and Shpak 1998; Kirkpatrick and Nuismer 2004) or in peripheral isolates (Servedio and Bürger 2015).

Sexual selection as a driver of speciation has been tested in comparative studies of birds (T. Price 1998; Edwards et al. 2005; T. Price 2008), fish such as cichlids (Seehausen et al. 1999; Seehausen and van Alphen 1999; A. B. Wilson et al. 2000), and in insects, with a group of flightless Hawaiian crickets being a prominent example (Mendelson and Shaw 2005). Learning and phenotypic plasticity of mate choice can facilitate speciation in some situations (Verzijden et al. 2012), but can also retard it (Nonaka et al. 2015). More recently, little correlation between sexual dimorphism and diversification rates has been found in comparative studies (Morrow et al. 2003; Ritchie 2007). Distinguishing between sexual selection and selection on traits other than mate choice as drivers of speciation can be challenging; Safran et al. (2013) suggest that focusing on traits that have clear functions in fecundity and survival rather than in mate choice may help.

Genomic and Chromosomal Barriers to Reproduction

Plants, animals, and fungi all exhibit enormous amounts of chromosomal variability (Deakin and Ezaz 2014; Baack et al. 2015; Manicardi et al. 2015) that may play a role in speciation (proposition 8, table 15.1). Often variation occurs among populations or species, and connections between chromosomal variation, instability, and speciation have been postulated (A. C. Wilson et al. 1975; White 1978). Chromosomal rearrangements and inversions between species are potent inhibitors of hybridization, given the challenges they pose to normal pairing during meiosis (Dobzhansky 1937a; White 1973; Lande 1984). Yet the loss of fitness during meiosis can provide a strong barrier to the evolution and fixation of these very same chromosomal rearrangements in natural populations (Lande 1983; Baker and Bickham 1986). The challenges of explaining the fixation of chromosomal rearrangements has led to an extensive literature, with recent work suggesting that some types of rearrangements may not require extreme levels of genetic drift or overdominant selection to become established (Kirkpatrick and Barton 2006; Kirkpatrick 2010). The importance of genetic drift in chromosomal evolution and speciation, especially founder-effect speciation, is today generally deprecated (Barton and Charlesworth 1984; Coyne and Orr 1997, 2004). However, the possibility of evolution through Wright's (1982) shifting balance process, particularly in chromosomal evolution (Barton and Rouhani 1991), deserves to be reexamined (Coyne and Orr 1997).

Polyploidy

The abundance of examples of ploidy differences between closely related plant species has led researchers to consider chromosomal mechanisms of speciation a major force in plant speciation (Baack et al. 2015). Because hybrids between a tetraploid and its diploid ancestor usually produce infertile triploid hybrids, polyploidy can be an instantaneous mechanism of speciation. Whole-genome duplications are inferred to have occurred in many lineages across the eukaryotic tree of life; in plants, the correspondence between polyploidy and recent speciation is particularly widespread. The possibility that changes in ploidy can drive speciation in plants is increased in cases where species can produce offspring by selfing (autopolyploidy), which in a novel polyploid would ameliorate the problem of finding a mate. Polyploidization can also occur in conjunction with hybridization (allopolyploidy). Disagreement exists as to whether allopolyploidy (Soltis and Soltis 2009) or autopolyploidy (Ramsey and Schemske 2002) is the more common mechanism. A recent survey found approximate parity between the two modes: although autopolyploids are produced more rapidly, they also seem to be more prone to extinction (Barker et al. 2016). The greater abundance of even versus odd chromosome numbers, among other evidence, suggests that approximately 15 percent of angiosperm and 31 percent of recent fern species evolved as a result of chromosome doubling (Otto and Whitton 2000; T. E. Wood et al. 2009). Macroevolutionary analyses of comparative genomics suggest an association between polyploidization and the diversification of plants (Jiao et al. 2011; Jiao et al. 2012; Tank et al. 2015). While whole genome duplication may have contributed to evolutionary flexibility and diversification deep in the tree of life, recent polyploidy events, in contrast, generally seem to slow down rates of diversification (T. E. Wood et al. 2009; Mayrose et al. 2011).

Inversions and Local Adaptation

Another type of genomic rearrangement that shows conspicuous connections with speciation is chromosomal inversion. Inversions maintain their distinctness between species by suppressing recombination, and may create fixed divergent adaptations between populations or species, even in the presence of gene flow, explaining their tendency to accumulate between species (Kirkpatrick and Barton 2006). Inversions that reduce recombination have increasingly been found to harbor genes underlying species differences and reproductive isolation (Noor et al. 2001; Lowry and Willis 2010). In recent years, genomic dissection of a number of species that exhibit chromosomal differences or striking intraspecific polymorphisms have revealed that inversions are hotspots of evolutionary novelty, sometimes harboring multiple loci that work in concert to promote reproductive isolation (Joron et al. 2011; Kunte et al. 2014; Küpper et al. 2016; Lamichhaney et al. 2016a; Tuttle et al. 2016). Polymorphic inversions represent an evolutionary alternative to speciation, since each inversion morph may allow exploitation of divergent environments in the face of potential gene flow. However, such polymorphisms could also have the potential to lead to speciation, perhaps when they become geographically isolated and additional genome-wide divergence accumulates.

Conclusion

Almost thirty years ago, Harrison (1991, 282) wrote, "Although clearly a major focus of evolutionary biology, the study of speciation has never emerged as a coherent discipline." Speciation research remains a wonderfully, if sometimes frustratingly, diverse subfield within evolutionary biology. Even today, researchers from systematists to geneticists to ecologists, publishing in a wide array of journals, purport to study speciation, often without reference to major lines of research within the field of speciation.

The focus of speciation research has changed over time, often adopting new paradigms or approaches as technical or conceptual advances have been made, or sometimes spearheaded by key publications (e.g., Maynard Smith 1966; Felsenstein 1981) or new genomic technologies. The most recent trend in speciation research is one of integration: How synthetic is a single study or research program, and how many levels in the hierarchy of mechanisms, from molecular to ecological, are spanned? Does the model system chosen for study lend itself to both ecological and genetic manipulation and investigation? These are the questions that drive the choice of species and topics in speciation research today.

Building on research agendas laid out during the modern synthesis, a new generation of experimental studies, using the latest tools of genomics and high-throughput phenotyping, has emerged in the past twenty years that focuses on the details of genetic incompatibilities or reinforcement, or that links fitness in the wild with allelic variation at specific genes. We believe that both descriptive (biogeographic/historical) and experimental research have valuable contributions to make to our understanding of speciation. Because speciation is in essence a historical event, experimental studies necessarily have to consider what is known of the particular history of the organisms being studied, and to ask how this history might constrain the details of genetic or ecological interactions observed in that study system today.

Despite the overwhelming evidence for the role of allopatry in speciation, this chapter has emphasized the importance of interactions among lineages found together in either primary or secondary sympatry. We have also emphasized the importance of verbal theories and the two-way flow of information between empirical and theoretical studies of speciation. Many mathematical theories of speciation understandably often envision very simple genetic mechanisms for some extrinsic traits, such as models that envision a single locus controlling mate or habitat choice (Kirkpatrick and Ravigné 2002). Such models, while necessarily simplistic, are useful in so far as they can envision a collection of processes that could result in speciation in the quantitative genetic limit of single genes controlling single traits. We agree with Turelli et al. (2001, 330) that "given the complexity of speciation, mathematical theory is subordinate to verbal theory and generalizations about data." Empirical studies of speciation are the most useful means of determining the relative strength and roles of intrinsic and extrinsic forces in speciation in nature, and arbitrate as to which mechanisms of speciation are most prevalent. In particular, the recent flush of genomic studies of speciation (Seehausen et al. 2014) has provided a detailed window into the genetic processes and genomic restructuring that accompany, or cause, speciation. Ultimately, empirical, historical, theoretical, and experimental studies will all be essential for evaluating the relative roles of selective versus random processes in speciation, whether at the level of genomes, individuals, or populations.

Speciation in the past was regarded as a key element in evolutionary theory: "Without speciation there would be no diversification of the organic world" (Mayr 1963, 621). However, Mayr's ideas conflicted somewhat with the uniformitarian Darwinian view that the processes we see operating within species, if extended, will lead naturally toward speciation. Notwithstanding Mayr's great contributions to the field, theories of speciation today tend to revert to a more Darwinian view of divergence. Speciation is seen instead as part of a continuum of diversification, with the species boundary no longer as clearly defined as it seemed to zoologists of the 1960s and 1970s (Nathan and Cracraft, chap. 6). Today we can more clearly perceive that within-species processes of natural and sexual selection, and likely some stochastic processes, can drive prezygotic isolation directly, by altering mating behavior, or by driving evolution in different directions in each lineage, leading indirectly to epistatic or other incompatibilities. Thus, today's theory of speciation would perhaps have been somewhat unexpected to earlier generations, but it is nonetheless coherent and well-formed, in contrast to Harrison's (1991) earlier view of the field. Our theory of speciation (table 15.1) thus successfully bridges theories of microevolution and of macroevolution, yet without needing to establish any clear hiatus between the two.

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References

Alcaide, M., E. S. C. Scordato, T. D. Price, and D. E. Irwin. 2014. Genomic divergence in a ring species complex. Nature 511:83-85.

Anderson, J. B., J. Funt, D. A. Thompson, S. Prabhu, A. Socha, C. Sirjusingh, J. R. Dettman, L. Parreiras, D. S. Guttman, A. Regev, and L. M. Kohn. 2010. Determinants of divergent adaptation and Dobzhansky-Muller interaction in experimental yeast populations. Current Biology 20:1383-1388.

Baack, E., M. C. Melo, L. H. Rieseberg, and D. Ortiz-Barrientos. 2015. The origins of reproductive isolation in plants. New Phytologist xxx:000.

Baker, R. J., and J. W. Bickham. 1986. Speciation by monobrachial centric fusions. Proc. Natl. Acad. Sci. USA 83:8245-8248.

Barker, M. S., N. Arrigo, A. E. Baniaga, Z. Li, and D. A. Levin. 2016. On the relative abundance of autopolyploids and allopolyploids. New Phytologist 210:391-398.

Barraclough, T. G., and A. P. Vogler. 2000. Detecting the pattern of speciation from species-level phylogenies. American Naturalist 155:419-434.

Barton, N. H., and B. Charlesworth. 1984. Genetic revolutions, founder effects, and speciation. Annu. Rev. Ecol. Syst. 15:133-164.

Barton, N. H., and M. A. de Cara. 2009. The evolution of strong reproductive isolation. Evolution 63:1171-1190.

Barton, N. H., and S. Rouhani. 1991. The probability of fixation of a new karyotype in a continuous population. Evolution 45:499-517.

Bateson, W. 1909. Heredity and variation in modern lights, Pages 85-101 *in* A. C. Seward, ed. Cambridge, Cambridge University Press.

Berlocher, S. H. 2000. Radiation and divergence in the *Rhagoletis pomonella* species group: inferences from allozymes. Evolution 54:543-557.

Bradshaw, H. D., and D. W. Schemske. 2003. Allele substitution at a flower locus produces a pollinator shift in monkeyflowers. Nature 426:176-178.

Brideau, N. J., H. A. Flores, J. Wang, S. Maheshwari, X. Wang, and D. A. Barbash. 2006. Two Dobzhansky-Muller genes interact to cause hybrid lethality in *Drosophila*. Science 314:1292-1295.

Buffon, C. d., Georges-Louis Leclerc. 1753. Description de la partie du Cabinet qui a rapport à l'histoire naturelle du cheval. L'asne, Pages 377-403 Histoire Naturelle, Générale et Particuliére, avec la Description du Cabinet du Roy. Paris.

Bush, G. L. 1969. Sympatric host race formation and speciation in frugivorous flies of the genus *Rhagoletis* (Diptera, Tephritidae). Evolution 23:237-251.

Bush, G. L. 1975a. Modes of animal speciation. Annu. Rev. Ecol. Syst. 6:339-364.

Bush, G. L. 1975b. Sympatric speciation in phytophagous parasitic insects, Pages 187-206 *in* P. W. Price, ed. Evolutionary Strategies of Parasitic Insects and Mites. New York, Plenum.

Butlin, R. 1987. Speciation by reinforcement. Trends in Ecology and Evolution 2:8-12.

Clausen, J., D. D. Keck, and W. M. Hiesey. 1939. The concept of species based on experiment. American Journal of Botany 26:103-106.

Corbett-Detig, R. B., J. Zhou, A. G. Clark, D. L. Hartl, and J. F. Ayroles. 2013. Genetic incompatibilities are widespread within species. Nature 504:135-137.

Coyne, J. A., N. H. Barton, and M. Turelli. 1997. Perspective: a critique of Sewall Wright's shifting balance theory of evolution. Evolution 51:643-671.

Coyne, J. A., and H. A. Orr. 1997. "Patterns of speciation in *Drosophila*" revisited. Evolution 51:295-303.

Coyne, J. A., and H. A. Orr. 2004, Speciation. Sunderland, Mass., Sinauer Associates.

Coyne, J. A., and H. A. Orr. 2010. Speciation: a catalogue and critique of species concepts. Reprinted from Appendix of *Speciation*, (Sinauer 2004), pp. 27, 447- 472. *in* A. Rosenberg, and R. Arp, eds. Philosophy of Biology. An Anthology. Chichester, Wiley-Blackwell.

Cracraft, J. 1983. Species concepts and speciation analysis. Current Ornithology 1:159-187.

Darwin, C. R. 1859, On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life. London, John Murray.

Darwin, C. R. 1868. Letter 6033 - Darwin, C. R. to Wallace, A. R., [21 Mar 1868], Pages - Darwin Correspondence Project. Cambridge, Cambridge University.

de Queiroz, K. 2007. Species concepts and species delimitation. Syst. Biol. 56:879-886.

Deakin, J. E., and T. Ezaz. 2014. Tracing the evolution of amniote chromosomes. Chromosoma 123:201-216.

Dettman, J. R., C. Sirjusingh, L. M. Kohn, and J. B. Anderson. 2007. Incipient speciation by divergent adaptation and antagonistic epistasis in yeast. Nature 447:585-588.

Dickinson, H., and J. Antonovics. 1973. Theoretical considerations of sympatric divergence. American Naturalist 107:256-274.

Dieckmann, U., and M. Doebeli. 1999. On the origin of species by sympatric speciation. Nature 400:354-357.

Diehl, S. R., and G. L. Bush. 1989. The role of habitat preference in adaptation and speciation, Pages 345-365 *in* D. Otte, and J. A. Endler, eds. Speciation and its Consequences. Sunderland, Mass., Sinauer.

Dobzhansky, T. 1937, Genetics and the Origin of Species. New York, Columbia Univ. Press.

Dobzhansky, T. 1940. Speciation as a stage in evolutionary divergence. American Naturalist 74:312-321.

Edwards, S. V., S. B. Kingan, J. D. Calkins, C. N. Balakrishnan, W. B. Jennings, W. J. Swanson, and M. D. Sorenson. 2005. Speciation in birds: genes, geography, and sexual selection. Proc. Natl. Acad. Sci. USA 102:6550-6557.

Feder, J. L., C. A. Chilcote, and G. L. Bush. 1988. Genetic differentiation between sympatric host races of the apple maggot fly *Rhagoletis pomonella*. Nature 336:61-64.

Feder, J. L., J. B. Roethele, K. Filchak, J. Niedbalski, and J. Romero-Severson. 2003. Evidence for inversion polymorphism related to host race formation in the apple maggot fly, *Rhagoletis pomonella*. Genetics 163:939-953.

Felsenstein, J. 1981. Skepticism towards Santa Rosalia, or why are there so few kinds of animals? Evolution 35:124-138.

Fitzpatrick, B. M., and M. Turelli. 2006. The geography of mammalian speciation: mixed signals from phylogenies and range maps. Evolution 60:601-615.

Frank, S. A. 1991. Divergence of meiotic drive-suppression systems as an explanation for sex- biased hybrid sterility and inviability. Evolution 45:262-267.

Fry, J. D. 2003. Multilocus models of sympatric speciation: Bush versus Rice versus Felsenstein. Evolution 57:1735-1746.

Gavrilets, S. 1997. Evolution and speciation on holey adaptive landscapes. Trends in Ecology and Evolution 12:307-312.

Gavrilets, S. 2014. Models of speciation: where are we now? Journal of Heredity 105:743-755.

Gourbière, S., and J. Mallet. 2010. Are species real? The shape of the species boundary with exponential failure, reinforcement, and the "missing snowball". Evolution 64:1-24.

Green, R. E., J. Krause, A. W. Briggs, T. Maricic, U. Stenzel, M. Kircher, N. Patterson, H. Li, W. Zhai, M. H. Fritz, N. F. Hansen, E. Y. Durand, A. S. Malaspinas, J. D. Jensen, T. Marques-Bonet, C. Alkan, K. Prüfer, M. Meyer, H. A. Burbano, J. M. Good, R. Schultz, A. Aximu-Petri, A. Butthof, B. Höber, B. Höffner, M. Siegemund, A. Weihmann, C. Nusbaum, E. S. Lander, C. Russ, N. Novod, J. Affourtit, M. Egholm, C. Verna, P. Rudan, D. Brajkovic, Z. Kucan, I. Gusic, V. B. Doronichev, L. V. Golovanova, C. Lalueza-Fox, M. de la Rasilla, J. Fortea, A. Rosas, R. W. Schmitz, P. L. F. Johnson, E. E. Eichler, D. Falush, E. Birney, J. C. Mullikin, M. Slatkin, R. Nielsen, J. Kelso, M. Lachmann, D. Reich, and S. Pääbo. 2010. A draft sequence of the Neandertal genome. Science 328:710-722.

Haldane, J. B. S. 1922. Sex ratio and unisexual sterility in hybrid animals. Journal of Genetics 12:101-109.

Haldane, J. B. S. 1930. A mathematical theory of natural and artificial selection. 6. Isolation. Proceedings of the Cambridge Philosophical Society 26:220-230.

Haldane, J. B. S. 1932, The Causes of Evolution. London, Longmans, Green & Co.

Harrison, R. G. 1991. Molecular changes in speciation. Annu. Rev. Ecol. Syst. 22:281-308.

Harrison, R. G. 2012. The language of speciation. Evolution 66:3643-3657.

Hawksworth, D. L., M. T. Kalin-Arroyo, P. M. Hammond, R. E. Ricklefs, R. M. Cowling, and M. J. Samways. 1995. Magnitude and distribution of biodiversity, Pages 107-192 *in* V. H. Heywood, and R. T. Watson, eds. Global Biodiversity Assessment. Cambridge, Cambridge University Press.

Hawthorne, D. J., and S. Via. 2001. Genetic linkage of ecological specialization and reproductive isolation in pea aphids. Nature 412:904-907.

Higashi, M., G. Takimoto, and N. Yamamura. 1999. Sympatric speciation by sexual selection. Nature 402:523-526.

Hoekstra, H. E., J. M. Hoekstra, D. Berrigan, S. N. Vignieri, A. Hoang, C. E. Hill, P. Beerli, and J. G. Kingsolver. 2001. Strength and tempo of directional selection in the wild. Proc. Natl. Acad. Sci. USA 98:9157-9160.

Hopkins, R., and M. D. Rausher. 2011. Identification of two genes causing reinforcement in the Texas wildflower *Phlox drummondii*. Nature 469:411.

Hopkins, R., and M. D. Rausher. 2012. Pollinator-mediated selection on flower color allele drives reinforcement. Science 335:1090-1092.

Hurst, L. D., and A. Pomiankowski. 1991. Causes of sex ratio bias may account for unisexual sterility in hybrids: a new explanation of Haldane's Rule and related phenomena. Genetics 128:841-858.

Irwin, D. E., S. Bensch, and T. D. Price. 2001. Speciation in a ring. Nature 409:333-337.

Iwasa, Y., A. Pomiankowski, and S. Nee. 1991. The evolution of costly mate preferences. II. The "handicap" principle. Evolution 45:1431-1442.

Jiao, Y., J. Leebens-Mack, S. Ayyampalayam, J. Bowers, M. McKain, J. McNeal, M. Rolf, D. Ruzicka, E. Wafula, N. Wickett, X. Wu, Y. Zhang, J. Wang, Y. Zhang, E. Carpenter, M. Deyholos, T. Kutchan, A. Chanderbali, P. Soltis, D. Stevenson, R. McCombie, J. Pires, G. Wong, D. Soltis, and C. dePamphilis. 2012. A genome triplication associated with early diversification of the core eudicots. Genome Biology 13:R3.

Jiao, Y., N. J. Wickett, S. Ayyampalayam, A. S. Chanderbali, L. Landherr, P. E. Ralph, L. P. Tomsho, Y. Hu, H. Liang, P. S. Soltis, D. E. Soltis, S. W. Clifton, S. E. Schlarbaum, S. C. Schuster, H. Ma, J. Leebens-Mack, and C. W. dePamphilis. 2011. Ancestral polyploidy in seed plants and angiosperms. Nature 473:97-100.

Jiggins, C. D., R. E. Naisbit, R. L. Coe, and J. Mallet. 2001. Reproductive isolation caused by colour pattern mimicry. Nature 411:302-305.

Joron, M., L. Frézal, R. T. Jones, N. L. Chamberlain, S. F. Lee, C. R. Haag, A. Whibley, M. Becuwe, S. W. Baxter, L. Ferguson, P. A. Wilkinson, C. Salazar, C. Davidson, R. Clark, M. A. Quail, H. Beasley, R. Glithero, C. Lloyd, S. Sims, M. C. Jones, and J. Rogers. 2011. Chromosomal rearrangements maintain a polymorphic supergene controlling butterfly mimicry. Nature 477:204-208.

Kingsolver, J. G., H. E. Hoekstra, J. M. Hoekstra, D. Berrigan, S. N. Vignieri, C. E. Hill, A. Hoang, P. Gibert, and P. Beerli. 2001. The strength of phenotypic selection in natural populations. The American Naturalist 157:245-261.

Kirkpatrick, M. 2010. How and why chromosome inversions evolve. PLoS biology 8.

Kirkpatrick, M., and N. Barton. 2006. Chromosome inversions, local adaptation and speciation. Genetics 173:419-434.

Kirkpatrick, M., and S. L. Nuismer. 2004. Sexual selection can constrain sympatric speciation. Proceedings of the Royal Society of London B: Biological Sciences 271:687-693.

Kirkpatrick, M., and V. Ravigné. 2002. Speciation by natural and sexual selection. American Naturalist 159:S22-S35.

Kirkpatrick, M., and M. J. Ryan. 1991. The evolution of mating preferences and the paradox of the lek. Nature 350:33-38.

Kondrashov, A. S., and M. Shpak. 1998. On the origin of species by means of assortative mating. Proceedings of the Royal Society of London B: Biological Sciences 265:2273-2278.

Kunte, K., W. Zhang, A. Tenger-Trolander, D. H. Palmer, S. P. Mullen, and M. R. Kronforst. 2013. *doublesex* is a mimicry supergene. Nature xxx:000-.

Küpper, C., M. Stocks, J. E. Risse, N. dos Remedios, L. L. Farrell, S. B. McRae, T. C. Morgan, N. Karlionova, P. Pinchuk, Y. I. Verkuil, A. S. Kitaysky, J. C. Wingfield, T. Piersma, K. Zeng, J. Slate, M. Blaxter, D. B. Lank, and T. Burke. 2016. A supergene determines highly divergent male reproductive morphs in the ruff. Nature Genetics 48:79-83.

Lamichhaney, S., G. Fan, F. Widemo, U. Gunnarsson, D. S. Thalmann, M. P. Hoeppner, S. Kerje, U. Gustafson, C. Shi, H. Zhang, W. Chen, X. Liang, L. Huang, J. Wang, E. Liang, Q. Wu, S. M.-Y. Lee, X. Xu, J. Hoglund, X. Liu, and L. Andersson. 2016. Structural genomic changes underlie alternative reproductive strategies in the ruff (*Philomachus pugnax*). Nature Genetics 48:84-88.

Lande, R. 1983. Chromosomal rearrangements in speciation. Heredity 50:214.

Lande, R. 1984. The expected fixation rate of chromosomal inversions. Evolution 38:743-752.

Liou, L. W., and T. D. Price. 1994. Speciation by reinforcement of premating isolation. Evolution 48:1451-1459.

Lowry, D. B. 2012. Ecotypes and the controversy over stages in the formation of new species. Biol. J. Linn. Soc. 106:241-257.

Lowry, D. B., J. L. Modliszewski, K. M. Wright, C. A. Wu, and J. H. Willis. 2008a. The strength and genetic basis of reproductive isolating barriers in flowering plants. Philosophical Transactions of the Royal Society B: Biological Sciences 363:3009-3021.

Lowry, D. B., C. R. Rockwood, J. H. Willis, and D. Schoen. 2008b. Ecological reproductive isolation of coast and inland races of *Mimulus guttatus*. Evolution 62:2196-2214.

Lowry, D. B., and J. H. Willis. 2010. A widespread chromosomal inversion polymorphism contributes to a major life-history transition, local adaptation, and reproductive isolation. PLoS Biology 8:e1000500.

Mallet, J. 2010. Why was Darwin's view of species rejected by 20th Century biologists? Biology and Philosophy 25:497-527.

Manicardi, G. C., A. Nardelli, and M. Mandrioli. 2015. Fast chromosomal evolution and karyotype instability: recurrent chromosomal rearrangements in the peach potato aphid *Myzus persicae* (Hemiptera: Aphididae). Biol. J. Linn. Soc. 116:519-529.

Martin, S. H., K. K. Dasmahapatra, N. J. Nadeau, C. Salazar, J. R. Walters, F. Simpson, M. Blaxter, A. Manica, J. Mallet, and C. D. Jiggins. 2013. Genome-wide evidence for speciation with gene flow in *Heliconius* butterflies. Genome Research 23:1817-1828.

Masly, J. P., and D. C. Presgraves. 2007. High-resolution genome-wide dissection of the two rules of speciation in *Drosophila*. PLoS Biology 5:e243.

Matute, D. R., I. A. Butler, D. A. Turissini, and J. A. Coyne. 2010. A test of the snowball theory for the rate of evolution of hybrid incompatibilities. Science 329:1518-1521.

Maynard Smith, J. 1966. Sympatric speciation. American Naturalist 100:637-650.

Mayr, E. 1942, Systematics and the Origin of Species. New York, Columbia Univ. Press.

Mayr, E. 1963, Animal Species and Evolution. Cambridge, Mass., Harvard University Press.

Mayr, E. 2002. Interview with Ernst Mayr. BioEssays 24:960-973.

Mayrose, I., S. H. Zhan, C. J. Rothfels, K. Magnuson-Ford, M. S. Barker, L. H. Rieseberg, and S. P. Otto. 2011. Recently formed polyploid plants diversify at lower rates. Science 333:1257.

McDermott, S. R., and M. A. F. Noor. 2010. The role of meiotic drive in hybrid male sterility. Philosophical Transactions of the Royal Society B-Biological Sciences 365:1265-1272.

McPheron, B. A., D. C. Smith, and S. H. Berlocher. 1988. Genetic differences between host races of *Rhagoletis pomonella*. Nature 336:64-66.

Mendelson, T. C., and K. L. Shaw. 2005. Rapid speciation in an arthropod. Nature 433:375-376.

Metz, J. A. J. 2011. Thoughts on the geometry of meso-evolution: collecting mathematical elements for a post-modern synthesis, Pages 193-231 *in* F. A. C. C. Chalub, and J. F. Rodrigues, eds. The Mathematics of Darwin's Legacy, Mathematics and Biosciences in Interaction. Basel, Springer.

Mora, C., D. P. Tittensor, S. Adl, A. G. B. Simpson, and B. Worm. 2011. How many species are there on Earth and in the ocean? PLoS Biology 9:e1001127.

Moritz, C., C. J. Schneider, and D. B. Wake. 1992. Evolutionary relationships within the *Ensatina eschscholtzii* complex confirm the ring species interpretation. Syst. Biol. 41:273-291.

Morrow, E. H., T. E. Pitcher, and G. Arnqvist. 2003. No evidence that sexual selection is an 'engine of speciation' in birds. Ecol. Lett. 6:228-234.

Moyle, L. C., and T. Nakazato. 2010. Hybrid incompatibility "snowballs" between *Solanum* species. Science 329:1521-1523.

Muller, H. J. 1942. Isolating mechanisms, evolution and temperature, Pages 71-125 *in* T. Dobzhansky, ed. Biological Symposia Volume 6. Temperature and evolution. Isolating mechanisms. Genetic control of Embryonic development. Biological Symposia. A Series of Volumes Devoted to Current Symposia in the Field of Biology. Lancaster, Pennsylvania, Jacques Cattell Press.

Nonaka, E., R. Svanbäck, X. Thibert-Plante, G. Englund, and Å. Brännström. 2015. Mechanisms by which phenotypic plasticity affects adaptive divergence and ecological speciation. The American Naturalist 186:E126-E143.

Noor, M. A. F. 1995. Speciation driven by natural selection in *Drosophila*. Nature 375:674-675.

Noor, M. A. F., K. L. Grams, L. A. Bertucci, and J. Reiland. 2001. Chromosomal inversions and the reproductive isolation of species. Proc. Natl. Acad. Sci. USA 98:12084-12088.

Nosil, P. 2012, Ecological Speciation. Oxford, Oxford University Press.

Orr, H. A. 1993. Haldane's rule has multiple genetic causes. Nature 361:532-533.

Orr, H. A. 1995. The population genetics of speciation: the evolution of hybrid incompatibilities. Genetics 139:1805-1813.

Orr, H. A. 2005. The genetic basis of reproductive isolation: insights from *Drosophila*. Proc. Natl. Acad. Sci. USA 102:6522-6526.

Orr, H. A., and M. Turelli. 1996. Dominance and Haldane's Rule. Genetics 143:613-616.

Orr, H. A., and M. Turelli. 2001. The evolution of postzygotic isolation: accumulating Dobzhansky-Muller incompatibilities. Evolution 55:1085-1094.

Orr, M. R., and T. B. Smith. 1998. Ecology and speciation. Trends in Ecology and Evolution 13:502-506.

Otto, S. P., and J. Whitton. 2000. Polyploid incidence and evolution. Annu. Rev. Genet. 34:401-437.

Phadnis, N., and H. A. Orr. 2009. A single gene causes both male sterility and segregation distortion in Drosophila hybrids. Science 323:376-379.

Polechová, J., and N. H. Barton. 2005. Speciation through competition: a critical review. Evolution 59:1194-1210.

Presgraves, D. C., L. Balagopalan, S. M. Abmayr, and H. A. Orr. 2003. Adaptive evolution drives divergence of a hybrid incompatibility gene between two species of *Drosophila*. Nature 423:715-719.

Presgraves, D. C., and H. A. Orr. 1998. Haldane's rule in taxa lacking a hemizygous X. Science 282:952-954.

Price, P. W. 1980, Evolutionary Biology of Parasites: Monographs in Population Biology, v. 15. Princeton, New Jersey, Princeton University Press.

Price, T. 1998. Sexual selection and natural selection in bird speciation. Philosophical Transactions of the Royal Society of London Series B-Biological Sciences 353:251-260.

Price, T. 2008, Speciation in Birds. Greenwood Village, Colorado, Roberts & Co.

Ramsey, J., H. D. Bradshaw, and D. W. Schemske. 2003. Components of reproductive isolation between the monkeyflowers *Mimulus lewisii* and *M. cardinalis* (Phrymaceae). Evolution 57:1520-1534.

Ramsey, J., and D. W. Schemske. 2002. Neopolyploidy in flowering plants. Annu. Rev. Ecol. Syst. 33:589-639.

Rheindt, F. E., and S. V. Edwards. 2011. Genetic introgression: an integral but neglected component of speciation in birds. Auk 128:620-632.

Ritchie, M. G. 2007. Sexual selection and speciation. "Annu. Rev. Ecol. Evol. Syst." 38:79-102.

Rundle, H. D., L. Nagel, J. W. Boughman, and D. Schluter. 2000. Natural selection and parallel speciation in sympatric sticklebacks. Science 287:306-308.

Rundle, H. D., and P. Nosil. 2005. Ecological speciation. Ecol. Lett. 8:336-352.

Safran, R. J., E. S. C. Scordato, L. B. Symes, R. L. Rodríguez, and T. C. Mendelson. 2013. Contributions of natural and sexual selection to the evolution of premating reproductive isolation: a research agenda. Trends Ecol. Evol. 28:643-650.

Schemske, D. 2010. Adaptation and the origin of species. American Naturalist 176:S4-S25.

Schilthuizen, M., M. C. W. G. Giesbers, and L. W. Beukeboom. 2011. Haldane's rule in the 21st century. Heredity 107:95-102.

Schluter, D. 2000, The Ecology of Adaptive Radiation. New York, Oxford University Press.

Seehausen, O., R. K. Butlin, I. Keller, C. E. Wagner, J. W. Boughman, P. A. Hohenlohe, C. L. Peichel, G.-P. Saetre, C. Bank, A. Brannstrom, A. Brelsford, C. S. Clarkson, F. Eroukhmanoff, J. L. Feder, M. C. Fischer, A. D. Foote, P. Franchini, C. D. Jiggins, F. C. Jones, A. K. Lindholm, K. Lucek, M. E. Maan, D. A. Marques, S. H. Martin, B. Matthews, J. I. Meier, M. Most, M. W. Nachman, E. Nonaka, D. J. Rennison, J. Schwarzer, E. T. Watson, A. M. Westram, and A. Widmer. 2014. Genomics and the origin of species. Nat Rev Genet 15:176-192.

Seehausen, O., P. J. Mayhew, and J. J. M. van Alphen. 1999. Evolution of colour patterns in East African cichlid fish. J. Evol. Biol. 12:514-534.

Seehausen, O., and J. J. M. van Alphen. 1999. Can sympatric speciation by disruptive sexual selection explain rapid evolution of cichlid diversity in Lake Victoria? Ecol. Lett. 2:262-271.

Servedio, M. R., and R. Bürger. 2015. The effects of sexual selection on trait divergence in a peripheral population with gene flow. Evolution 69:2648-2661.

Servedio, M. R., and M. Kirkpatrick. 1997. The effects of gene flow on reinforcement. Evolution 51:1764-1772.

Servedio, M. R., G. S. Van Doorn, M. Kopp, A. M. Frame, and P. Nosil. 2011. Magic traits in speciation: 'magic' but not rare? Trends in Ecology and Evolution 26:389-397.

Shaw, K. L., and S. P. Mullen. 2011. Genes versus phenotypes in the study of speciation. Genetica 139:661-.

Sobel, J. M., and G. F. Chen. 2014. Unification of methods for estimating the strength of reproductive isolation. Evolution 68:1511-1522.

Soltis, P. S., and D. E. Soltis. 2009. The role of hybridization in plant speciation. Annu. Rev. Plant Biol. 60:561-588.

Spencer, H. G., B. H. McArdle, and D. M. Lambert. 1986. A theoretical investigation of speciation by reinforcement. American Naturalist 128:241-262.

Stathos, A., and L. Fishman. 2014. Chromosomal rearrangements directly cause underdominant F1 pollen sterility in *Mimulus lewisii–Mimulus cardinalis* hybrids. Evolution 68:3109-3119.

Stukenbrock, E. H. 2013. Evolution, selection and isolation: a genomic view of speciation in fungal plant pathogens. New Phytologist 199:895-907.

Sweigart, A. L., and L. E. Flagel. 2015. Evidence of natural selection acting on a polymorphic hybrid incompatibility locus in *Mimulus*. Genetics 199:543-554.

Tang, S., and D. C. Presgraves. 2009. Evolution of the *Drosophila* nuclear pore complex results in multiple hybrid incompatibilities. Science 323:779-782.

Tang, S., and D. C. Presgraves. 2015. Lineage-specific evolution of the complex *Nup160* hybrid incompatibility between *Drosophila melanogaster* and its sister species. Genetics 200:1245-1254.

Tank, D. C., J. M. Eastman, M. W. Pennell, P. S. Soltis, D. E. Soltis, C. E. Hinchliff, J. W. Brown, E. B. Sessa, and L. J. Harmon. 2015. Nested radiations and the pulse of angiosperm diversification: increased diversification rates often follow whole genome duplications. New Phytologist 207:454-467.

Turelli, M., N. H. Barton, and J. A. Coyne. 2001. Theory and speciation. Trends in Ecology and Evolution 16:330-343.

Turelli, M., J. R. Lipkowitz, and Y. Brandvain. 2014. On the Coyne and Orr-igin of species: effects of intrinsic postzygotic isolation, ecological differentiation, X chromosome size, and sympatry on *Drosophila* speciation. Evolution 68:1176-1187.

Turelli, M., and L. C. Moyle. 2007. Asymmetric postmating isolation: Darwin's corollary to Haldane's Rule. Genetics 176:1059-1088.

Turelli, M., and H. A. Orr. 1995. The dominance theory of Haldane's rule. Genetics 140:389-402.

Turesson, G. 1922. The genotypical response of the plant species to the habitat. Hereditas 3:341-347.

Tuttle, Elaina M., Alan O. Bergland, Marisa L. Korody, Michael S. Brewer, Daniel J. Newhouse, P. Minx, M. Stager, A. Betuel, Zachary A. Cheviron, Wesley C. Warren, Rusty A. Gonser, and Christopher N. Balakrishnan. 2016. Divergence and functional degradation of a sex chromosome-like supergene. Current Biology 26:344-350.

Verzijden, M. N., C. ten Cate, M. R. Servedio, G. M. Kozak, J. W. Boughman, and E. I. Svensson. 2012. The impact of learning on sexual selection and speciation. Trends in Ecology and Evolution 27:511-519.

Via, S., and J. West. 2008. The genetic mosaic suggests a new role for hitchhiking in ecological speciation. Mol. Ecol. 17:4334-4345.

Wade, M. J., N. A. Johnson, and Y. Toquenaga. 1999. Temperature effects and genotype-by-environment interactions in hybrids: Haldane's Rule in flour beetles. Evolution 53:855-865.

Wade, M. J., N. A. Johnson, and G. Wardle. 1994. Analysis of autosomal polygenic variation for the expression of Haldane's Rule in flour beetles. Genetics 138:791-799.

Wallace, A. R. 1889, Darwinism. An Exposition of the Theory of Natural Selection with Some of its Applications. London, Macmillan & Co.

Wang, I. J., R. E. Glor, and J. B. Losos. 2013. Quantifying the roles of ecology and geography in spatial genetic divergence. Ecol. Lett. 16:175-182.

White, M. J. D. 1973, Animal Cytology and Evolution. Cambridge, Cambridge, Univ. Press.

White, M. J. D. 1978, Modes of Speciation. San Francisco, WH Freeman.

Wilson, A. B., K. Noack-Kunnmann, and A. Meyer. 2000. Incipient speciation in sympatric Nicaraguan crater lake cichlid fishes: sexual selection versus ecological diversification. Proc. Roy. Soc. B Biol. Sci. 267:2133-2141.

Wilson, A. C., G. L. Bush, S. M. Case, and M. C. King. 1975. Social structuring of mammalian populations and rate of chromosomal evolution. Proc. Natl. Acad. Sci. USA 72:5061-5065.

Wood, T. E., N. Takebayashi, M. S. Barker, I. Mayrose, P. B. Greenspoon, and L. H. Rieseberg. 2009. The frequency of polyploid speciation in vascular plants. Proc. Natl. Acad. Sci. USA 106:13875-13879.

Wright, S. 1982. The shifting balance theory and macroevolution. Annu. Rev. Genet. 16:1-19.