# Tipping points in the dynamics of speciation

Patrik Nosil[1]*, Jeffrey L. Feder[2], Samuel M. Flaxman[3] and Zachariah Gompert[4]

**Speciation can be gradual or sudden and involve few or many genetic changes. Inferring the processes generating such patterns is difficult, and may require consideration of emergent and non-linear properties of speciation, such as when small changes at tipping points have large effects on differentiation. Tipping points involve positive feedback and indirect selection stemming from associations between genomic regions, bi-stability due to effects of initial conditions and evolutionary history, and dependence on modularity of system components. These features are associated with sudden 'regime shifts' in other cellular, ecological, and societal systems. Thus, tools used to understand other complex systems could be fruitfully applied in speciation research.**

The speciation process can range from gradual to sudden[1–6]. Here, we draw caricatures of these scenarios. These are not meant to realistically capture the complexity of speciation. Rather, they introduce elements of the on-going debate concerning whether Darwinian gradualism can be reconciled with mounting evidence for rapid evolution, evolutionary gaps, and missing intermediates. Our argument is that resolving this debate will require moving beyond these caricatures because they conflate pattern (genetic and phenotypic change) and process (drivers of change). We outline how this can be achieved using evolutionary theory, genomics, and principles emerging from the study of a wide range of complex, dynamical systems.

Darwin argued that speciation involves the gradual accumulation of differences between populations in small steps[2]. This process can leave an observable and inter-connected 'speciation continuum' of populations varying in differentiation (Fig. 1, Table 1)[7–15]. For example, pea aphid host races vary in levels of population genetic differentiation[11] and natural hybridization between butterfly taxa declines gradually with genetic distance[16]. In modern parlance, many differences in small steps can be interpreted as polygenic, genome-wide changes. Indeed, multiple loci of minor effect underlie many cases of adaptation[17–21], and some cases of reproductive isolation[22,23]. For example, local adaptation of herring[17], cichlid[24], and stick-insect populations involves numerous genome-wide differences[25,26], and multiple loci of modest effect contribute to flowering time differences in maize[21] and sexual isolation between cricket species[22].

However, palaeontologists have long reported the sudden emergence of new taxa in the fossil record. This led influential figures like Simpson, Eldredge, and Gould to highlight the punctuated nature of evolution[1,27–29]. Likewise, modern theory suggests speciation can occur suddenly due to rare founder effects[30–32] or rapid evolution once mutations causing reproductive isolation arise[33,34]. In terms of genetics, speciation as a single evolutionary leap driven by macromutation and 'hopeful monsters' is largely unsupported (polyploidization aside), but major genetic changes do occur[35–37]. For example, major effect loci contribute to differences in bony armour between stickleback populations[38], colour-pattern differences between butterflies[39,40], flower colour in phlox[41], and vision in cichlids[7]. Accordingly, major effect loci or genome re-arrangements can concentrate differentiation into a few genomic regions[4,33,34,39,42–45], as reported between sub-species of crows[45], colour-pattern races of butterflies[38–40,45], and *Drosophila* species[46].

There is a 'many-to-many' relation between the patterns reported above and underlying speciation processes[47]. That is, a given pattern may be explained by the action of many alternative processes, and theoretical expectations become complex once multiple populations and potential gene flow between them is introduced[48–51]. In particular, a pattern of sudden differentiation could be influenced by an abrupt environmental shift, epistatic interactions causing a snowball of intrinsic genetic incompatibilities[33,52,53] (for example, as argued in flies[54] and tomatoes[55]), slight increases in frequency-dependent selection that drive rapid evolutionary branching[56,57], and evolutionary leaps via genome re-arrangement, polyploidization, or founder events[6,30,58]. Of these factors, those that act during speciation are more critical for the divergence process than those that accumulate after speciation is complete[47,59].

Sudden speciation is also compatible with evolution in small steps. For example, sudden evolution can arise when small changes become coupled to each other in a positive feedback loop[60–64] (that is, at some critical threshold a change in a dynamic variable $x$ can increase $y$, which feeds back to increase $x$, and so on). As such, a divergence process involving small changes can suddenly speed up at a 'tipping point' in speciation, at least in theory (Fig. 2; see Table 2 for a glossary)[5]. In this case, speciation emerges as an intrinsic dynamical property of the divergence process, not via a large extrinsic perturbation or trigger. Hence, even when reproductive isolation and genetic differentiation are continuous variables, tipping points cause taxa to generally occupy one of two states: a single species with little differentiation, or two strongly differentiated species.

Such distinct states could be indicative of bi-stability (that is, alternative stable states under similar conditions)[65–67], which arises when evolution is dependent on initial conditions and the sequence of historical events (also called path-dependency or hysteresis). In speciation, initial conditions such as sympatry versus geographic isolation can affect the type of differentiation that builds[5,68], epistasis can cause the historical sequence by which mutations arise to affect evolution[69–71], and drift can affect which variants are lost or established through time[5]. Such factors can affect the reversibility of evolution such that reverting to an original state is difficult, again contributing to bi-stability.

As one example of such dynamics, Flaxman *et al.*[5] modelled divergence with gene flow in terms of the per-locus strength of divergent selection (DS) between ecological environments ($s$), migration rates ($m$), and numbers of genetic loci involved. In this

[1]Department of Animal and Plant Sciences, University of Sheffield, Sheffield S10 2TN, UK. [2]Department of Biology, Notre Dame University, South Bend, Indiana 11111, USA. [3]Department of Ecology and Evolutionary Biology, University of Colorado, Boulder, Colorado 80309, USA. [4]Department of Biology, Utah State University, Logan, Utah 84322, USA. *e-mail: p.nosil@sheffield.ac.uk
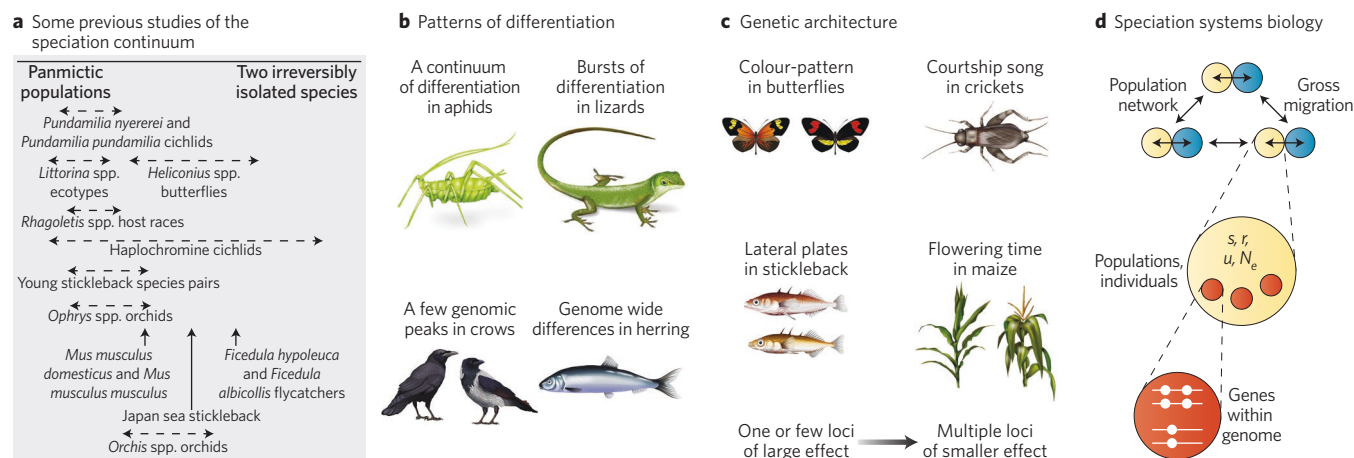
**Figure 1 | Empirical studies of the speciation continuum and the dynamics of speciation. a**, Previous studies of the speciation continuum. Modified from ref. [47], Macmillan Publishers Ltd. **b**, Patterns of differentiation in aphids[11], lizards[105], crows[45] and herring[17]. **c**, Genetic architecture of traits in butterflies[39,40], crickets[22], stickleback[38] and maize[21]. **d**, Overview of a systems biology framework for studying speciation. White lines are chromosomes with circles on them being genetic loci. Red circles are individuals. Blue and yellow circles are populations in different environments. Double-headed arrows represent gross migration and the letters represent evolutionary processes ($s$, selection; $r$, recombination; $u$, mutation; $N_e$, effective population size). Illustrations by R. Ribas.

**Table 1 | Examples of empirical studies of different speciation stages for replicate, co-occurring pairs of taxa.**

| Organism | Comparison | Genetic data | Replicates | Results | Reference |
|---|---|---|---|---|---|
| **Fish** | | | | | |
| Cichlid (*Pundamilia*) | Sympatric phenotypes found in different localities within lake Victoria | msat | 5 pairs | $F_{ST}$ = 0.000, 0.002, 0.010, 0.014, 0.026 | 7 |
| Stickleback (*Gasterosteus*) | Parapatric lake–stream pairs in North America, each pair in a different locality | msat | 6 pairs | $F_{ST}$ = 0.05, 0.10, 0.12, 0.14, 0.16, 0.23 | 8 |
| Stickleback (*Gasterosteus*) | Parapatric lake–stream pairs across the globe, each pair in a different locality | WGS | 5 pairs | $F_{ST}$ = 0.09, 0.11, 0.22, 0.22, 0.28 | 9 |
| Lake whitefish (*Coregonus*) | Sympatric dwarf and normal ecotypes within lakes, each pair in a different lake | PGS | 5 pairs | $F_{ST}$ = 0.008, 0.029, 0.049, 0.105, 0.216 | 10 |
| **Plants** | | | | | |
| Sunflowers (*Helianthus*) | Sympatric or parapatric species pairs with different levels of gene flow | PGS | 3 pairs (plus 1 allopatric) | $F_{ST}$ = 0.30, 0.35, 0.51 (0.48 for allopatric pair) | 104 |
| **Insects** | | | | | |
| Pea aphids (*Acyrthosiphon*) | Sympatric populations associated with different host plants in western Europe | msat | 11 sympatric biotypes | An interconnected continuum of differentiation | 11 |
| Mimetic butterflies (*Heliconius*) | Parapatric and sympatric races and species | WGS | 4 pairs | Continuum of differentiation between races and species | 12; see also[13] |
| **Birds** | | | | | |
| Flycatchers (*Ficedula*) | Populations within and between species, with variation in degree of geographic overlap | WGS | 7 pairwise comparisons | $F_{ST}$ ~ 0.1 within species, ~0.3 between species, ~1.0 between distantly related species | 14 |
| **Amphibians** | | | | | |
| Poison frogs (*Ranitomeya*) | Mimetic morphs in three different transition zones | msat | 3 transition zones | Clines for different colour pattern traits, clines varied from offset to coincident; genetic structure varied from present to absent | 15 |

msat, microsatellite loci; WGS, whole genome sequence; PGS, partial genome sequence (for example, from genotyping-by-sequencing). We report quantitative results if the original study tabulated them for genome wide $F_{ST}$, but otherwise give a summary of qualitative findings. Due to different marker types being used, values cannot be easily compared across studies, but within-study variation encapsulates the putative speciation continuum.

model, loci differentiate by the selection they directly experience plus indirect selection stemming from statistical associations (linkage disequilibrium, LD) with other divergently selected loci. When migration was high ($m > s$), sudden speciation and alternative stable states of differentiation occurred and went hand-in-hand with a positive feedback loop (Fig. 2). Below a critical threshold of genome wide DS and between-population LD, differentiation built very slowly due to homogenising migration. However, once a critical level of both was reached, DS and LD entered a positive feedback loop where each enhanced the other, driving a rapid reduction in gene flow and a transition from one species to two. In essence, loci under DS transition from evolving independently to exhibiting

coupled dynamics; out of a mass of a genetically well-mixed population, distinct clusters of genotypes congeal, and rapid, genome-wide differentiation ensues. Due to the role of LD, indirect selection was critical to explaining these dynamics. In Box 1 we use simulations to provide exploratory results on how such coupling in time relates to previous theory on the coupling of multilocus clines in space[72,73]. Similar dynamics likely apply during polygenic adaptation[5,64,74] and the coupling of different reproductive barriers[60,72,75]. Thus, the dynamics we focus on here could be general, and indeed selection and LD are commonly involved in speciation[47].

In contrast to the results described above, divergence with lower migration in the Flaxman *et al.*[5] model was more linear through time and 'gene-by-gene', with individual loci differentiating by the selection they directly experience rather than through strong effects of LD[5]. The issue then is not just whether few or many loci diverge during speciation. Rather, it is whether a few genes diverge first (followed gradually by the rest) versus many genes diverging suddenly and simultaneously once a critical amount of selected, standing variation exists.

Sudden speciation in the aforementioned model occurred without intrinsic genetic incompatibilities, major effect loci, genome re-arrangements, or periods of geographic isolation, though these factors can promote the process. For example, divergence maintained despite migration was often higher when initial differentiation involved a period of geographic isolation than when it did not[5]. This provides one example of bi-stability; two outcomes were possible for the same selection strength, dependent on initial geographic setting. This example also highlights that the divergence process is bi-directional, as differentiation can build, be maintained upon secondary contact, or collapse. Here, we propose a framework for understanding these potentially complex dynamics that draw parallels between speciation and tipping points in other complex systems[65–67]. We then outline approaches to quantify speciation patterns and infer underlying process.

## Connection between speciation and other complex systems
When speciation involves simple evolutionary dynamics driven by few loci then a reductionist approach focused on identifying and studying these individual loci may enhance our understanding of speciation[18,76]. In other cases, speciation may involve many loci and emerge via complex interactions between evolutionary processes[3,5,33]. Such complex phenomena cannot always be understood as the additive combination of their underlying individual parts. Instead, 'systems thinking' may be required that attempts to understand how complex networks exhibit emergent properties not shown by individual nodes in the network[77–79]. Evolutionary studies of complex, population-level processes such as speciation might benefit from such thinking, which remains largely the purview of cell and molecular biologists working below the population level, or ecologists working above it (that is, our analogy is here a heuristic one concerning the study of emergent properties in complex systems, not a direct one-to-one analogy to applications in molecular biology and genomics). Some relevant networks for speciation are genes within genomes, individuals within sub-populations, and sub-populations within a meta-population.

To support our argument, we consider how tipping points in speciation relate to those in other complex systems. 'Tipping points' in the narrow sense are cases where positive feedback at an unstable equilibrium causes a rapid shift between alternative states. This usage is applied to critical transitions or sudden 'regime shifts' in a range of complex systems (for example, health: asthma attacks; ecology: population extinction, climate change, shifts in community composition; economics: crash of financial markets)[65–67]. Principles governing tipping points in these systems have emerged[65–67], such as features that make a system prone to system-wide regime shifts. Specifically, increased connectivity (that
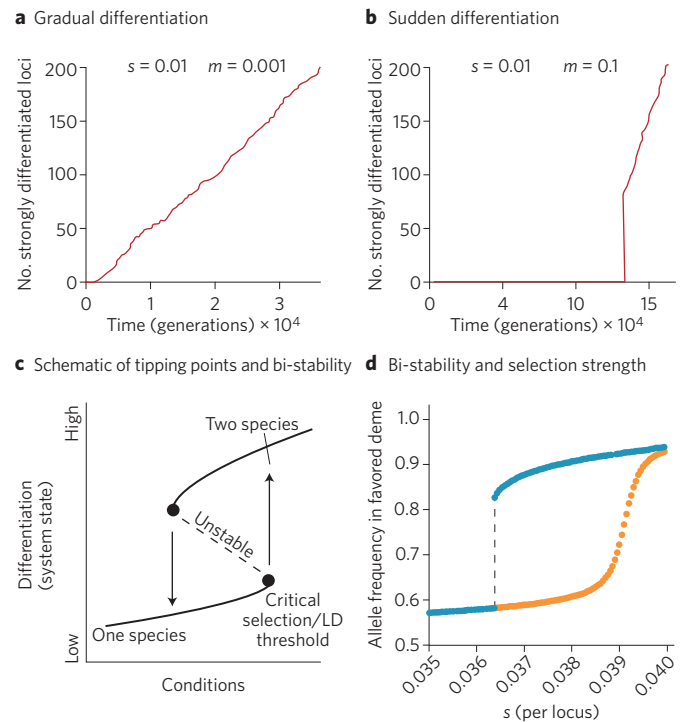


**Figure 2 | Gradual and sudden dynamics of speciation in the model by Flaxman *et al.*[5]. a**, Gradual differentiation occurs when selection is strong relative to migration. Following Hartl and Clark[106], strongly differentiated loci are those with $F_{ST} \geq 0.25$, but results are similar for other cut-offs. ($s$ = strength of divergent selection. $m$ = gross migration rate.) **b**, Sudden differentiation occurs when selection is weak relative to migration, because a critical threshold of divergent selection and genome-wide linkage disequilibrium must be achieved before differentiation can ensue. **c**, Schematic of a tipping point where positive feedback at an unstable equilibrium (dashed line) causes a rapid shift between alternative stable states. Slight changes in conditions (for example, selection strengths and levels of standing genetic variation) can cause the system to switch from one state to the other. An unstable domain predicts alternative states under similar conditions, dependent on the history of events leading to those conditions. **d**, An example of bi-stability in the Flaxman *et al.*[5] model (with the number of divergently selected loci ($L$) = 60 loci, $m = 0.1$). The y-axis shows the local frequency of an allele in the deme in which it is favoured. Within a range of $s$ values, there are two equilibria and the system state depends upon past conditions. This is indicated by the two sets of points, one when initializing populations with maximum divergence (blue) and the other when initializing with no divergence (orange). Panels **a**,**b**,**d** use previously published data from ref. [5] and the Dryad repository[107]. A script for producing the panels is archived in a GitHub repository (https://github.com/flaxmans/NatureEE2017).

is, reduced modularity) of a system network increases the chance of critical transitions. One explanation for this is that local changes in a well-connected system are 'repaired' by neighbouring nodes, buffering the system against local change[65–67]. Thus, observable change does not occur until the entire system hits a threshold that drives a shift to an alternative, system-wide state. In other words, a highly connected system is robust to local perturbation, but prone to system-wide change. In contrast, poorly connected systems allow gradual node-by-node change.

These concepts appear to apply to speciation. For example, sudden dynamics in Flaxman *et al.*[5] were dependent on two types of connectivity. First, sudden transitions from one species to two were only observed in models that allow for the build up of LD, because LD was a key component of the feedback that drives the transition.

**Table 2 | Glossary of key terms and examples of their relevance for speciation dynamics.**

| Term | Definition | Example of relevance |
|---|---|---|
| Indirect selection | Selection on a trait stemming from correlation of the trait with a directly selected trait; the same concept applies to a locus rather than a trait. | Can increase the total selection experienced by a trait (or locus) above that due to direct selection, potentially promoting divergence with gene flow. |
| Tipping point (narrow sense) | A point where a system may flip to an alternative state, involving positive feedback at an unstable equilibrium. | Could explain sudden speciation and gaps between populations and species, and do so without invoking catastrophic events or large external perturbations. |
| Positive feedback | A process in which dynamic variables enhance the changes happening in each other (that is, each increases the other). | At a critical threshold, divergent selection and LD can enter a feedback where each reinforces the other, driving rapid speciation. |
| Bi-stability | A scenario in which a system has two alternative possible stable states under the same conditions (often due to effects of initial conditions and path-dependence in evolution). | Could explain gaps between populations and species. |
| Linkage disequilibrium (LD) | Non-random statistical associations between alleles at different loci. | A core component of some types of positive feedback loops that drive speciation. |
| Sudden dynamics; (non-linear dynamics) | Some parts of speciation occur much faster than others such that differentiation is not uniform through time. | Leads to discontinuous patterns of differentiation and gaps between stages of speciation. |
| Gradual dynamics (linear dynamics) | Near uniform/constant differentiation through time; note that this does not imply slow change, but rather simply a fairly constant rate. | Leads to a well inter-connected speciation continuum, with intermediate states readily observed. |
| Critical transition | Abrupt shift in a system when driving parameters reach a threshold (that is, critical) value; the associated shift from one state to another is sometimes referred to as a 'regime shift'. | Rapid shifts in differentiation at critical thresholds of divergent selection and LD. |
| Gross migration | Movement of individuals between populations (contrasts with effective migration which considers the incorporation of the alleles in those individuals into the local genetic background). | Variation in gross migration rates can affect the dynamics of speciation, and whether tipping points occur. |

Indeed, factors that promote LD, such as increased physical linkage in chromosomal inversions, can promote speciation with gene flow[43,44,80]. LD can be conceptualized as a type of connectivity between genes due to their organisation into individuals (that is, genomes). In other words, genes are connected because offspring are not formed gene-by-gene from a population 'beanbag' of alleles, but rather, parents pass on sets of genes to offspring. Note that we refer here to connectivity of genes in a statistical sense (that is, LD), although connectivity in gene regulatory networks warrants future work.

Second, speciation dynamics were dependent on the gross migration rate connecting populations (relative to the strength of DS). When migration was low, individual loci overcame gene flow via the selection they directly experience, and thus diverged on their own (Fig. 2). In other words, genes had largely independent dynamics and gradual, gene-by-gene divergence ensued. The situation was different with high migration, where loci have difficulty diverging via direct selection. Instead, speciation requires alleles at different loci to develop strong associations, causing them to be selected against as units in migrating individuals (Box 1). This allows even weakly directly selected loci to overcome high gene flow via the combined effects of direct and indirect selection. Thus, connectivity of genes within individuals (in genomes) and among populations (due to migration) can affect the likelihood of sudden change.

Our logic above focused on simple spatial settings (example, population pairs), but could be extended to complex networks of many sub-populations (that is, a meta-population)[81], for which speciation is an emergent systems-level property (Fig. 1). Nodes in the network are sub-populations and connections between nodes are edges representing gross migration. Each node can have a series of properties (for example, population size, selective regime, dispersal rate, genetic architecture, recombination landscape, etc.). Reproductive isolation and genetic differentiation emerge when considering sets of populations in the network (for example, those that are ecologically divergent or connected in particular ways by migration), but are not exhibited by individual populations.

Another phenomenon studied in regime shifts is their propensity to exhibit 'early warning signs'[65–67]. Early warning signs are statistical signals that occur when a system is nearing a tipping point, but before a critical transition. Examples are increased variance and autocorrelation, slow return to previous state upon small perturbation ('critical slowing down'), and 'flickering' between alternative states when perturbations are sufficiently large to push a system temporarily back and forth between states. It is unclear if such signs apply to speciation, but we suspect some signals should precede a drastic shift, such as the initial appearance of subsets of loci with elevated LD. Work in this area is warranted as it might allow populations near tipping points (that are poised for greater differentiation) to be identified and compared to those far from tipping points. With this framework in place, we turn to empirical studies of speciation dynamics.

## Patterns and processes of speciation

Here, we consider empirical patterns of genetic differentiation during speciation, and inference of underlying processes.

**Quantifying gradual versus sudden patterns.** We focus on genetic differentiation because it can be measured in a wide range of systems and can reflect reproductive isolation. In this context, genetic differentiation is best considered with spatial setting in mind, for example compared among taxa in a similar spatial setting (for example, multiple pairs of adjacent lake and stream ecotypes of stickleback)[82]. This is because reproductive isolation is only tested to the extent that geographic proximity allows for potential gene flow[6,30,47,58,83,84], and a continuum of differentiation under isolation-by-distance need not be indicative of a continuum of reproductive isolation. Despite our focus on genetic differentiation, our logic applies to experimental estimates of reproductive isolation, which could be used as another measure of degree of speciation.

Inferring patterns of differentiation during speciation is challenging for at least four reasons (Fig. 3). First, high replication is required. This is because whenever data are sparse there is danger
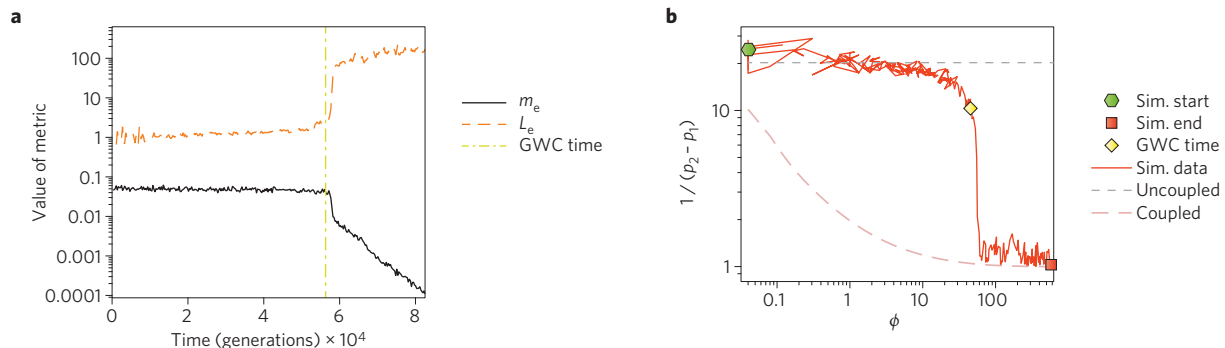
**Box 1 | Exploratory results on the relation between multilocus coupling in time versus space.**

Multilocus cline theory[72,73] makes predictions about the shape of allele-frequency clines in space by determining the conditions in which clines at different barrier loci will be coupled (that is, acting as a multilocus selected unit) or uncoupled (loci evolving independently)[108]. Coupling is promoted by increasing the number of barrier loci ($L$), increasing the strength of selection per locus ($s$), or decreasing the recombination probability between neighbouring loci ($r$). The effects of these key drivers can be encapsulated by the summed coupling coefficient, $\phi$, defined[73] as $\phi = (L - 1)s/r$.

Though most previous theory considers equilibrium patterns in space, temporal dynamics of coupling have also been studied[5]. However, theories in space versus time are poorly connected, and non-equilibrium conditions have proven difficult to study analytically. We show here exploratory results relating temporal dynamics and critical transitions observed in stochastic, forward-time simulations to metrics commonly used in multilocus cline theory. The figure shows results with $s = 0.02$ and gross migration rate $m = 5\%$ between two demes (that is, discrete

space). Additional parameter combinations and details are in the Supplementary Information.

New, divergently selected mutations arise continuously in our simulations, causing $\phi$ to increase over time because $L$ increases and $r$ decreases as a greater number of variable sites become packed into a genome of fixed size. The actual degree of coupling between loci at any point in time can be quantified by the 'effective number of loci'[72], $L_e$, computed as the number of loci with selection coefficient $s$ that would need to be perfectly coupled (that is, in complete linkage disequilibrium) to produce the observed average allele frequency difference between demes at a given time. $L_e = 1$ when loci are evolving independently (each locus acts as one independent locus characterized by $s$) and increases as loci become coupled. Our simulations highlight two key points about evolutionary dynamics when $m > s$: (1) genome-wide congealing (GWC) in time is associated with nonlinear shifts in reproductive isolation and coupling ($L_e$), and (2) there may be a critical value of $\phi$ that defines a tipping point between undifferentiated and differentiated populations (see panels **a** and **b**, respectively).



**a**, Example time series (one simulation run) showing the effective migration rate, $m_e$ (solid line), and effective number of loci, $L_e$ (orange dashed line), through the genome-wide congealing (GWC) transition (yellow dash-dot line). $m_e$ is a population-level measure of reproductive isolation, defined as the proportion of a deme's reproduction from immigrants[109]. **b**, Relationship between a discrete-space analogue of cline width—the inverse of allele frequency difference between demes—and $\phi$ (time-implicit parametric plot from same simulation run as **a**; solid line: simulation results; dashed and dotted lines: deterministic expectations for completely coupled and uncoupled sites, respectively; circle: start time; square: end time; diamond: time of GWC.) Random fluctuations in the analogue to cline width ($y$-axis) arise due to the effects of genetic drift. See Supplementary Information for detailed methods.

of inferring discontinuities when they don't exist, an issue long discussed with respect to inferences from the fossil record[1,27–29]. Second, the full distribution of differentiation should be sampled to avoid false inference of a continuum. For example, an inferred continuum among weakly to moderately differentiated ecotypes could actually be part of a strong discontinuity between ecotypes and well-differentiated species. Third, the build-up of differentiation within a single lineage should ideally be analysed[33]. Combining data from distantly related lineages can be problematic, for example when sudden change within each of two different lineages appears gradual when data from the two are combined. This presents an empirical difficulty for studying speciation because individual lineages may not exhibit variation in all stages of speciation. Indeed, studies of the speciation tend to span a modest portion of the speciation continuum[47] (Fig. 1). Fourth, not all loci necessarily couple and differentiate simultaneously, with divergently selected loci differentiating before neutral ones[4,85]. Thus, neutral loci may exhibit gradual differentiation for long periods of time and assessment of whether divergence is gradual or sudden can depend on whether phenotypes, neutral loci, or adaptive loci are examined[7].

To our knowledge, data of sufficient scale to resolve these issues are sparse. For example, studies of speciation (Table 1), including our own work in *Timema* stick insects[25], *Lycaeides* butterflies[86], and *Rhagoletis* flies[87], have highlighted the quantitative nature of the divergence process[47]. However, existing work does not generally overcome the difficulties described above, precluding strong inferences about the dynamics of divergence along this continuum. Highly replicated studies of differentiation across the speciation continuum are now required to test whether the transition from weak to strong differentiation occurs gradually or suddenly. Even if time since divergence cannot be inferred, the overall distribution of differentiation can be informative. For example, bi-modal distributions imply gaps in the speciation continuum and potentially sudden dynamics. Observational studies could be supplemented with experiments mimicking secondary contact between populations, testing directly whether gene flow itself (that is, reproductive isolation) changes gradually or suddenly as genetic differentiation increases. Another possibility concerns the fossil record. When fossils contain information on multiple traits that are known to exhibit independent genetic control (for example, in extant relatives)[88,89],

**a** Problems with low replication

**b** Problems with not sampling broadly across the distribution

● Not sampled   ● Sampled

**c** Problems with lineage-specific variation

● Species 1   ● Species 2

**d** Differential timing of coupling ('time lag')
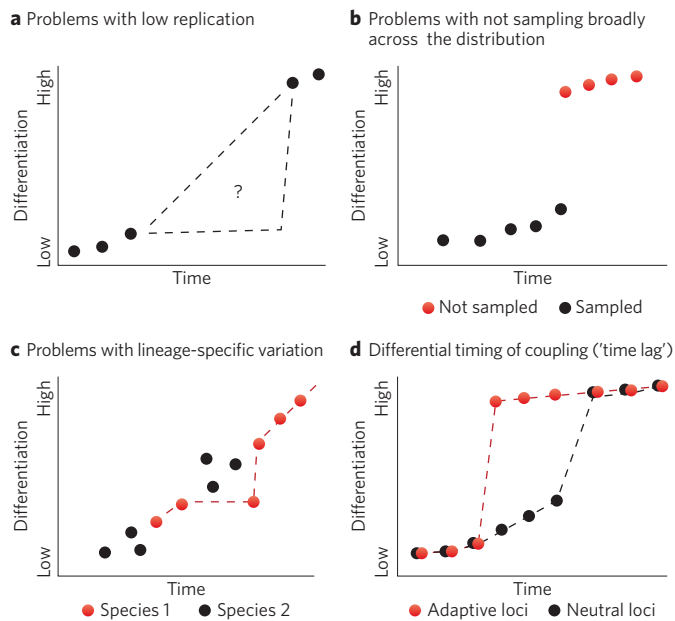
● Adaptive loci   ● Neutral loci

**Figure 3 | Difficulties with quantifying patterns of differentiation during speciation. a**, With low replication it can be difficult to extrapolate between data points in a manner that unambiguously distinguishes gradual from sudden change. **b**, If both extremes of the distribution of differentiation are not sampled, a continuum might be falsely inferred (as for the sampled taxa shown with black points). **c**, Each dot represents data from a different population pair. When data from different species are combined true discontinuities within a species (red, dotted line) might be hidden, leading to the erroneous conclusion of gradual dynamics. Specifically, if a line were drawn through all the points shown (red and black) then the pattern would look continuous, despite it being discontinuous within one of the species. **d**, Adaptive and neutral loci might differentiate at different points in the speciation process such that patterns of speciation are dependent on the type of loci examined.

the dynamics by which the evolution of different traits becomes coupled could be analysed through time. Of course, experimental evolution in the lab could directly quantify speciation dynamics[90]. However, this may not be as simple as it sounds, as many systems with levels of sex and recombination appropriate for tests of most speciation models are not amenable to long-term laboratory studies.

**Quantifying genetic changes.** Speciation can involve genetically localised or genome-wide changes. Under the genic model of speciation, some genetic regions become resistant to gene flow (that is, exhibit reproductive isolation) before others[85,91]. This leads to one or a few localised 'genomic islands' of differentiation, which grow through time as speciation progresses[42,47]. Eventually, effects of reproductive isolation evolve to become genome wide, as implied by the aforementioned theories of coupling and congealing of differentiation across loci[5,68,72], and classic views of biological species[24,30]. An unresolved empirical issue is how readily and why the transition to genome-wide differentiation occurs.

Approaches for quantifying the genetic changes involved in adaptation and speciation have been covered elsewhere[18,42,47,76,92–94]. We briefly re-iterate two core points that are most relevant for speciation dynamics. First, integrative approaches that combine ecology, experiments, population genomics, and genetic mapping could yield more robust inferences than studies relying on one approach alone. In particular, observational genome scans and genetic mapping studies identifying genetic regions associated with population differentiation can be supplemented with manipulative transplant or experimental evolution studies to test if these regions are subject

to divergent selection. Although such experimental approaches are not without limitations, recent studies highlight their potential[25,26,92,95–99]. For example, studies of stick-insects and flies show that genetic regions responding to experimentally induced divergent selection correspond to those which are highly differentiated between natural populations[25,99,100].

Second, quantifying the true distribution of genetic changes involved in speciation is challenging because of the relative ease of detecting large effect changes (that is, leading to overestimation of the distribution of effect sizes). Nonetheless, recent analytical advances for quantifying genetic architecture *per se* can help alleviate this problem. Consider the example of genome-wide association (GWA) mapping. Classic GWA methods analyse contributions of individual genetic variants to phenotypic variation, one at a time[101]. These methods are suited for detecting loci of large effect but not for quantifying the architecture of polygenic, complex traits. In contrast, recently developed whole-genome regression or polygenic modelling approaches consider the joint influence of all genetic variants, and relatedness among individuals, to quantify genetic architecture[101]. Polygenic modelling is aligned in spirit with quantitative genetics and in wide use in artificial breeding, but not fully exploited in evolutionary studies[101,102]. Once patterns of differentiation and associated genetic changes are in hand, one can turn to the difficult task of inference of underlying speciation processes.

**Inferring process.** It is clear that speciation dynamics are parameter-dependent and can involve non-linear, emergent properties. Thus, inferring process will require information on selective regimes, migration, recombination, and the underlying genetic architecture of traits driving speciation, including effect sizes, linkage relationships, and epistatic interactions. Such data can allow patterns of differentiation to be more readily interpreted in light of theoretical predictions. It is also relevant to test whether speciation coincided with a bottleneck, founder event, or abrupt environmental shift. Approaches for inferring these factors have been covered elsewhere[47,83], so we focus on the topics of feedback and bi-stability here.

It may be difficult to distinguish whether sudden differentiation is due to small changes in an individual variable having large effects or a true feedback loop (that is, between dynamic variables). For example, a change in either DS or LD could increase genetic differentiation, without invoking feedback. Changes without feedback predict bi-modality only in one response variable. In contrast, a feedback predicts missing intermediates in both variables being measured. Ultimately, tests could be devised for whether each component of a feedback loop directly strengthens the other. In terms of bi-stability, two states should be observable under similar conditions, that is, those representing an unstable intermediate domain. In some cases, the historical sequence of events will have led to strong differentiation, but in other cases they will have not. The same prediction applies for gene flow upon experimental secondary contact. In principle, populations lying in the unstable domain could be perturbed to an alternative state, for example by manipulating levels of LD via gene flow or other factors. These examples suggest that even if contemporary populations largely sit in one or two domains of differentiation, it may still be possible to study transitions between them.

## Concluding remarks

Implementation of the ideas outlined here will require substantial effort, as it implies the need to generate data on patterns of differentiation, multiple evolutionary processes, and genetic architecture in a wide range of sub-populations, and better frameworks for comparisons among studies. However, until this is done it will be impossible to know how useful systems-level thinking and tipping points will be for understanding the dynamics of speciation, or if the hunt for individual genes driving speciation will largely suffice[53,59,103]. We

predict that the inherently multi-locus and multi-faceted nature of speciation makes tipping points and initial conditions of broad importance for understanding the process. An open question is how evolutionary tipping points might affect ecological systems (for example, communities and ecosystems)[65–67], leading to interactions between evolutionary and ecological dynamics.

## References

1. Gould, S. J. *The Structure of Evolutionary Theory* (Belknap Press, 2002).
2. Darwin, C. *On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life* (John Murray, 1859).
3. Gavrilets, S. Perspective: Models of speciation: What have we learned in 40 years? *Evolution* **57,** 2197–2215 (2003).
   **A review of theoretical models of speciation, with a focus on those utilizing the framework of genetic incompatibilities.**
4. Gavrilets, S. & Losos, J. B. Adaptive radiation: Contrasting theory with data. *Science* **323,** 732–737 (2009).
5. Flaxman, S., Walchoder, A., Feder, J. L. & Nosil, P. Theoretical models of the influence of genomic architecture on speciation. *Mol. Ecol.* **23,** 4074–4088 (2014).
6. Coyne, J. A. & Orr, H. A. *Speciation* 1st edn (Sinauer Associates, 2004).
7. Seehausen, O. *et al.* Speciation through sensory drive in cichlid fish. *Nature* **455,** 620–623 (2008).
   **A demonstration of speciation via natural and sexual selection in cichlid fish, with emphasis on different degrees of differentiation during the process.**
8. Berner, D., Grandchamp, A. C. & Hendry, A. P. Variable progress toward ecological speciation in parapatry: stickleback across eight lake-stream transitions. *Evolution* **63,** 1740–1753 (2009).
9. Feulner, P. G. D. *et al.* Genomics of divergence along a continuum of parapatric population differentiation. *PLoS Genet.* **11,** e1005414 (2015).
10. Gagnaire, P.-A., Pavey, S. A., Normandeau, E. & Bernatchez, L. The genetic architecture of reproductive isolation during speciation-with-gene-flow in lake whitefish species pairs assessed by rad sequencing. *Evolution* **67,** 2483–2497 (2013).
11. Peccoud, J., Ollivier, A., Plantegenest, M. & Simon, J. C. A continuum of genetic divergence from sympatric host races to species in the pea aphid complex. *Proc. Natl Acad. Sci. USA* **106,** 7495–7500 (2009).
12. Martin, S. H. *et al.* Genome-wide evidence for speciation with gene flow in *Heliconius* butterflies *Genome Res.* **23,** 1817–1828 (2013).
13. Kronforst, M. R. *et al.* Hybridization reveals the evolving genomic architecture of speciation. *Cell Rep.* **5,** 666–677 (2013).
14. Burri, R. *et al.* Linked selection and recombination rate variation drive the evolution of the genomic landscape of differentiation across the speciation continuum of Ficedula flycatchers. *Genome Res.* **25,** 1656–1665 (2015).
15. Twomey, E., Vestergaard, J. S., Venegas, P. J. & Summers, K. Mimetic divergence and the speciation continuum in the mimic poison frog *Ranitomeya imitator*. *Am. Nat.* **187,** 205–224 (2016).
16. Mallet, J., Beltran, M., Neukirchen, W. & Linares, M. Natural hybridization in heliconiine butterflies: the species boundary as a continuum. *BMC Evol. Biol.* **7,** 28 (2007).
17. Lamichhaney, S. *et al.* Population-scale sequencing reveals genetic differentiation due to local adaptation in Atlantic herring. *Proc. Natl Acad. Sci. USA* **109,** 19345–19350 (2012).
18. Rockman, M. V. The QTN program and the alleles that matter for evolution: all that's gold does not glitter. *Evolution* **66,** 1–17 (2012).
   **A perspective piece arguing that much of adaptation and evolution may stem from multiple loci with alleles of small effect.**
19. Yang, J. *et al.* Genome partitioning of genetic variation for complex traits using common SNPs. *Nat. Genet.* **43,** 519–544 (2011).
20. Carneiro, M. *et al.* Rabbit genome analysis reveals a polygenic basis for phenotypic change during domestication. *Science* **345,** 1074–1079 (2014).
21. Buckler, E. S. *et al.* The genetic architecture of maize flowering time. *Science* **325,** 714–718 (2009).
22. Ellison, C. K., Wiley, C. & Shaw, K. L. The genetics of speciation: genes of small effect underlie sexual isolation in the Hawaiian cricket Laupala. *J. Evol. Biol.* **24,** 1110–1119 (2011).
23. Fishman, L., Kelly, A. J. & Willis, J. H. Minor quantitative trait loci underlie floral traits associated with mating system divergence in Mimulus. *Evolution* **56,** 2138–2155 (2002).
24. Brawand, D. *et al.* The genomic substrate for adaptive radiation in African cichlid fish. *Nature* **513,** 375–381 (2014).
25. Soria-Carrasco, V. *et al.* Stick insect genomes reveal natural selection's role in parallel speciation. *Science* **344,** 738–742 (2014).
26. Gompert, Z. *et al.* Experimental evidence for ecological selection on genome variation in the wild. *Ecol. Lett.* **17,** 369–379, http://dx.doi.org/10.1111/ele.12238 (2014).
27. Eldredge, N. & Gould, S. J. in *Models in Paleobiology* (ed. Schopf, T. J. M.) 82–115 (1972).
28. Simpson, G. G. *Tempo and Mode in Evolution* (Columbia Univ. Press, 1944).
29. Simpson, G. G. *The Major Features of Evolution* (Columbia Univ. Press, 1953).
30. Mayr, E. *Animal Species and Evolution* (Harvard Univ. Press, 1963).
31. Mayr, E. in *Evolution as a Process* (eds Huxley, J., Hardy, A. C. & Ford, E. B.) (Allen & Unwin, 1954).
32. Barton, N. H. & Charlesworth, B. Genetic revolutions, founder effects, and speciation. *Annu. Rev. Ecol. Syst.* **15,** 133–164 (1984).
33. Gavrilets, S. *Fitness Landscapes and the Origin of Species* Vol. 41 (Princeton Univ. Press, 2004).
34. Kirkpatrick, M. & Barton, N. Chromosome inversions, local adaptation and speciation. *Genetics* **173,** 419–434 (2006).
35. Dietrich, M. R. Richard Goldschmidt: hopeful monsters and other 'heresies'. *Nat. Rev. Genet.* **4,** 68–74 (2003).
36. Chouard, T. Evolution: Revenge of the hopeful monster. *Nature* **463,** 864–867 (2010).
37. Goldschmidt, R. *The Material Basis of Evolution* (Yale Univ. Press 1940).
38. Colosimo, P. F. *et al.* Widespread parallel evolution in sticklebacks by repeated fixation of ectodysplasin alleles. *Science* **307,** 1928–1933 (2005).
   **An empirical study showing repeated adaptation to freshwater via a gene of large effect.**
39. Nadeau, N. J. *et al.* Population genomics of parallel hybrid zones in the mimetic butterflies, *H. melpomene* and *H. erato*. *Genome Res.* **24,** 1316–1333 (2014).
40. Nadeau, N. J. *et al.* Genomic islands of divergence in hybridizing *Heliconius* butterflies identified by large-scale targeted sequencing. *Phil. Trans. R. Soc. B* **367,** 343–353 (2012).
41. Hopkins, R. & Rausher, M. D. Pollinator-mediated selection on flower color allele drives reinforcement. *Science* **335,** 1090–1092 (2012).
42. Feder, J. L., Egan, S. P. & Nosil, P. The genomics of speciation-with-gene-flow. *Trends Genet.* **28,** 342–350 (2012).
43. Noor, M. A. F., Grams, K. L., Bertucci, L. A. & Reiland, J. Chromosome inversions and the reproductive isolation of species. *Proc. Natl Acad. Sci. USA* **98,** 12084–12088 (2001).
44. Rieseberg, L. H., Whitton, J. & Gardner, K. Hybrid zones and the genetic architecture of a barrier to gene flow between two sunflower species. *Genetics* **152,** 713–727 (1999).
45. Poelstra, J. W. *et al.* The genomic landscape underlying phenotypic integrity in the face of gene flow in crows. *Science* **344,** 1410–1414 (2014).
46. McGaugh, S. E. & Noor, M. A. F. Genomic impacts of chromosomal inversions in parapatric Drosophila species. *Phil. Trans. R. Soc. B* **367,** 422–429 (2012).
47. Seehausen, O. *et al.* Genomics and the origin of species. *Nat. Rev. Genet.* **15,** 176–192 (2014).
48. Orr, H. A. The population genetics of adaptation: the distribution of factors fixed during adaptive evolution. *Evolution* **52,** 935–949 (1998).
49. Orr, H. A. The genetic theory of adaptation: A brief history. *Nat. Rev. Genet.* **6,** 119–127 (2005).
50. Yeaman, S. & Otto, S. P. Establishment and maintenance of adaptive genetic divergence under migration, selection, and drift. *Evolution* **65,** 2123–2129 (2011).
51. Yeaman, S. & Whitlock, M. C. The genetic architecture of adaptation under migration-selection balance. *Evolution* **65,** 1897–1911 (2011).
   **Theoretical work on the evolution of genetic architecture when populations are exchanging genes with one another, contrasting with population genetics theory based on evolution within single, isolated populations.**
52. Orr, H. A. The population-genetics of speciation — the evolution of hybrid incompatibilities. *Genetics* **139,** 1805–1813 (1995).
53. Orr, H. A. The genetic basis of reproductive isolation: Insights from *Drosophila*. *Proc. Natl Acad. Sci. USA* **102,** 6522–6526 (2005).
54. Matute, D. R., Butler, I. A., Turissini, D. A. & Coyne, J. A. A test of the snowball theory for the rate of evolution of hybrid incompatibilities. *Science* **329,** 1518–1521 (2010).
55. Moyle, L. C. & Nakazato, T. Hybrid incompatibility "snowballs" between solanum species. *Science* **329,** 1521–1523 (2010).
56. Dieckmann, U. & Doebeli, M. On the origin of species by sympatric speciation. *Nature* **400,** 354–357 (1999).
57. Dieckmann, U., Doebeli, M., Metz, J. A. J. & Tautz, D. *Adaptive Speciation* (Cambridge Univ. Press, Cambridge, 2004).
58. Mayr, E. *Systematics and the Origin of Species* (Columbia Univ. Press, 1942).
59. Nosil, P. & Schluter, D. The genes underlying the process of speciation. *Trends Ecol. Evol.* **26,** 160–167 (2011).

60. Servedio, M. R. & Saetre, G. P. Speciation as a positive feedback loop between postzygotic and prezygotic barriers to gene flow. *Proc. R. Soc. B* **270,** 1473–1479 (2003).

61. Crespi, B. J. Vicious circles: positive feedback in major evolutionary and ecological transitions. *Trends Ecol. Evol.* **19,** 627–633 (2004).
**A review of how positive feedback may drive major biological transitions, in cases ranging from speciation to social evolution.**

62. Schwander, T., Vuilleumier, S., Dubman, J. & Crespi, B. J. Positive feedback in the transition from sexual reproduction to parthenogenesis. *Proc. R. Soc. B* **277,** 1435–1442 (2010).

63. Lehtonen, J. & Kokko, H. Positive feedback and alternative stable states in inbreeding, cooperation, sex roles and other evolutionary processes. *Phil. Trans. R. Soc. B* **367,** 211–221 (2012).

64. Rasanen, K. & Hendry, A. P. Disentangling interactions between adaptive divergence and gene flow when ecology drives diversification. *Ecol. Lett.* **11,** 624–636 (2008).
**An overview of the interactions, and potential feedback, between adaptive divergence and gene flow.**

65. Scheffer, M. *et al.* Early-warning signals for critical transitions. *Nature* **461,** 53–59 (2009).

66. Scheffer, M., Carpenter, S. R., Dakos, V. & van Nes, E. H. Generic indicators of ecological resilience: inferring the chance of a critical transition. *Ann. Rev. Ecol. Evol. System.* **46,** 145–167 (2015).

67. Scheffer, M. *et al.* Anticipating critical transitions. *Science* **338,** 344–348 (2012).
**A review of critical transitions and tipping points in complex systems, with an emphasis on the potential to predict tipping points in advance using early warning signs.**

68. Kirkpatrick, M. & Ravigné, V. Speciation by natural and sexual selection: Models and experiments. *Am. Nat.* **159,** S22–S35 (2002).

69. Carneiro, M. & Hartl, D. L. Adaptive landscapes and protein evolution. *Proc. Natl Acad. Sci. USA* **107,** 1747–1751 (2010).

70. Poelwijk, F. J., Kiviet, D. J., Weinreich, D. M. & Tans, S. J. Empirical fitness landscapes reveal accessible evolutionary paths. *Nature* **445,** 383–386 (2007).

71. Weinreich, D. M., Delaney, N. F., DePristo, M. A. & Hartl, D. L. Darwinian evolution can follow only very few mutational paths to fitter proteins. *Science* **312,** 111–114 (2006).

72. Barton, N. H. Multilocus clines. *Evolution* **37,** 454–471 (1983).

73. Kruuk, L. E. B., Baird, S. J. E., Gale, K. S. & Barton, N. H. A comparison of multilocus clines maintained by environmental adaptation or by selection against hybrids. *Genetics* **153,** 1959–1971 (1999).

74. Hendry, A. P. Selection against migrants contributes to the rapid evolution of ecologically dependent reproductive isolation. *Evol. Ecol. Res.* **6,** 1219–1236 (2004).

75. Barton, N. H. & de Cara, M. A. R. The evolution of strong reproductive isolation. *Evolution* **63,** 1171–1190 (2009).
**A theoretical model showing how different reproductive barriers can become coupled in their effects to drive strong reproductive isolation.**

76. Rausher, M. D. & Delph, L. F. Commentary: When does understanding phenotypic evolution require identification of the underlying genes? *Evolution* **69,** 1655–1664 (2015).

77. Kitano, H. Systems biology: A brief overview. *Science* **295,** 1662–1664 (2002).
**A brief overview of systems thinking and emergent properties in biology.**

78. Novikoff, A. B. The concept of integrative levels and biology. *Science* **101,** 209–215 (1945).

79. Benfey, P. N. & Mitchell-Olds, T. Perspective: From genotype to phenotype: Systems biology meets natural variation. *Science* **320,** 495–497 (2008).

80. Hoffmann, A. A. & Rieseberg, L. H. Revisiting the impact of inversions in evolution: from population genetic markers to drivers of adaptive shifts and speciation? *Annu. Rev. Ecol. Evol. Syst.* **39,** 21–42 (2008).

81. Hanski, I. A. Eco-evolutionary spatial dynamics in the Glanville fritillary butterfly. *Proc. Natl Acad. Sci. USA* **108,** 14397–14404 (2011).

82. Berner, D., Grandchamp, A.-C. & Hendry, A. P. Variable progress toward ecological speciation in parapatry: stickleback across eight lake-stream transitions. *Evolution* **63,** 1740–1753 (2009).

83. Pinho, C. & Hey, J. Divergence with gene flow: models and data. *Ann. Rev. Ecol. Evol. System.* **41,** 215–230 (2010).

84. Mallet, J., Meyer, A., Nosil, P. & Feder, J. L. Space, sympatry and speciation. *J. Evol. Biol.* **22,** 2332–2341 (2009).

85. Wu, C. The genic view of the process of speciation. *J. Evol. Biol.* **14,** 851–865 (2001).

86. Gompert, Z. *et al.* Admixture and the organization of genetic diversity in a butterfly species complex revealed through common and rare genetic variants. *Mol. Ecol.* **23,** 4555–4573 (2014).

87. Powell, T. H. Q. *et al.* Genetic divergence along the speciation continuum: the transition from host race to species in *rhagoletis* (Diptera: tephritidae). *Evolution* **67,** 2561–2576 (2013).

88. Bell, M. A. Implications of a fossil stickleback assemblage for Darwinian gradualism. *J. Fish Biol.* **75,** 1977–1999 (2009).

89. Purnell, M. A., Bell, M. A., Baines, D. C., Hart, P. J. B. & Travis, M. P. Correlated evolution and dietary change in fossil stickleback. *Science* **317,** 1887–1887 (2007).

90. Bell, G. Experimental macroevolution. *Proc. R. Soc. B* **283,** 20152547 (2016).

91. Mallet, J. A species definition for the modern synthesis. *Trends Ecol. Evol.* **10,** 294–299 (1995).

92. Barrett, R. D. H. & Hoekstra, H. E. Molecular spandrels: tests of adaptation at the genetic level. *Nat. Rev. Genet.* **12,** 767–780 (2011).

93. Feder, M. E. & Mitchell-Olds, T. Evolutionary and ecological functional genomics. *Nat. Rev. Genet.* **4,** 651–657 (2003).

94. Cruickshank, T. E. & Hahn, M. W. Reanalysis suggests that genomic islands of speciation are due to reduced diversity, not reduced gene flow. *Mol. Ecol.* **23,** 3133–3157 (2014).

95. Barrett, R. D. H., Rogers, S. M. & Schluter, D. Natural selection on a major armor gene in threespine stickleback. *Science* **322,** 255–257 (2008).

96. Pespeni, M. H. *et al.* Evolutionary change during experimental ocean acidification. *Proc. Natl Acad. Sci. USA* **110,** 6937–6942 (2013).

97. Anderson, J. T., Lee, C.-R., Rushworth, C. A., Colautti, R. I. & Mitchell-Olds, T. Genetic trade-offs and conditional neutrality contribute to local adaptation. *Mol. Ecol.* **22,** 699–708 (2013).

98. Anderson, J. T., Lee, C. R. & Mitchell-Olds, T. Strong selection genome-wide enhances fitness trade-offs across environments and episodes of selection. *Evolution* **68,** 16–31 (2014).

99. Egan, S. P. *et al.* Experimental evidence of genome-wide impact of ecological selection during early stages of speciation-with-gene-flow. *Ecol. Lett.* **18,** 817–825 (2015).

100. Michel, A. P. *et al.* Widespread genomic divergence during sympatric speciation. *Proc. Natl Acad. Sci. USA* **107,** 9724–9729 (2010).

101. Zhou, X., Carbonetto, P. & Stephens, M. Polygenic modeling with Bayesian sparse linear mixed models. *PLoS Genet.* **9,** e1003264 (2013).

102. Hill, W. G. Applications of population genetics to animal breeding, from Wright, Fisher and Lush to genomic prediction. *Genetics* **196,** 1–16 (2014).

103. Orr, H. A. & Presgraves, D. C. Speciation by postzygotic isolation: forces, genes and molecules. *Bioessays* **22,** 1085–1094 (2000).

104. Renaut, S. *et al.* Genomic islands of divergence are not affected by geography of speciation in sunflowers. *Nat. Commun.* **4,** 1827 (2013).

105. Harmon, L. J., Schulte, J. A., Larson, A. & Losos, J. B. Tempo and mode of evolutionary radiation in iguanian lizards. *Science* **301,** 961–964 (2003).

106. Hartl, D. L. & Clark, A. G. *Principles of Population Genetics* 4th edn (Sinauer, 2007).

107. Flaxman, S. M., Wacholder, A. C., Feder, J. L. & Nosil, P. Data from: Theoretical models of the influence of genomic architecture on the dynamics of speciation. *Dryad Digital Repository* http://dx.doi.org/10.5061/dryad.kc596 (2014).

108. Abbott, R. *et al.* Hybridization and speciation. *J. Evol. Biol.* **26,** 229–246 (2013).

109. Vuilleumier, S., Goudet, J. & Perrin, N. Evolution in heterogeneous populations from migration models to fixation probabilities. *Theor. Popul. Biol.* **78,** 250–258 (2010).

## Acknowledgements

## Author contributions

P.N., S.M.F., J.F. and Z.G. conceived the project and wrote the paper. S.M.F. ran the simulations and analysed them.

## Additional information

**Supplementary information** is available for this paper.

**Reprints and permissions information** is available at www.nature.com/reprints.

**Correspondence** should be addressed to P.N.

**How to cite this article:** Nosil, P., Feder, J. L., Flaxman, S. M. & Gompert, Z. Tipping points in the dynamics of speciation. *Nat. Ecol. Evol.* **1,** 0001 (2017).

## Competing interests

The authors declare no competing financial interests.