## **Online Methods**

*Conceptual model*

We consider the scenario of two parapatric species inhabiting demes in two different habitats. These species exchange migrants at a low level, but reproductive isolation via local adaptation (i.e. extrinsic postzygotic isolation and immigrant inviability) is strong enough to prevent substantial introgression. We imagine that these two species must also cope with constant adaptation to a shared oscillating “climate” optimum. This climatic optimum does not not directly affect the degree of local adaptation and/or reproductive isolation, i.e. reproductive isolation is completely independent of the direct effects of climate. The climate oscillation continues for a long initial burn in period, during which alleles conferring adaptation to climate accumulate in each species. After this period, the oscillation ends and the climatic optimum begins rapidly increasing at a constant rate, as is expected under projections of anthropogenic climate change.

We hypothesize that if the rate of change in the climatic optimum is sufficiently high, selection for migrant alleles conferring increased climate tolerance will overwhelm the negative fitness effects of linked reproductive isolation alleles. This will cause the erosion of reproductive isolation between species and increase the chance of speciation reversal. Importantly, we expect this outcome even when the strength of ecological selection mediating reproductive isolation itself is orthogonal to the strength of climate-mediated selection.

*Model details*

We implemented the above conceptual model as a genetically explicit Wright-Fisher model in SLiM 3.025. As in all Wright-Fisher models, population sizes are constant, all fitness is relative and extinction is impossible. The details of our implementation are depicted graphically in Supplementary Figure 1 and a list of simulation parameters and their values are detailed in Supplementary Table 1. We simulated two diploid populations of constant size *Ne,* with a constant migration rate of *m* proportionmigrants per generation. Each individual was initialized with 99999 genetic loci contained on a single chromosome with a uniform recombination rate of *r* between loci. We initially scaled the recombination rate so that the entire genome was 100 cM in length, but also explored varying recombination rates up a genome size of 1000 cM. We modelled extrinsic isolation in the two populations as *lEX* divergently selected alleles at loci evenly spaced across the chromosome, with each population fixed for a different allele. Divergently selected alleles imposed a fitness cost of *sRI* when not found in their home population/habitat, modelling extrinsic postzygotic isolation.

In addition to extrinsic postzygotic isolation, we also modelled intrinsic postzygotic isolation using two-locus Bateson-Dobzhansky-Muller incompatibilities26-28. These epistatic incompatibilities were modelled as a fitness cost of *sRI* scaled by the number of negatively-interacting pairs of alleles from each population (Supplementary Table 2). When testing the effects of BDMs, we maintained a constant number of total reproductive isolation loci, but varied the proportion of loci that were extrinsic or BDM loci (*l)*. We also explored the effect of the total number of RI loci (i.e. the genetic architecture of RI *per se*) on the potential for adaptive introgression/hybridization. To keep the total magnitude of RI similar between simulations, we always co-varied *sRI* so that the *sRI* x *l* was held constant.To allow for fine-scale view of introgression, we tracked ancestry was using 100 neutral alleles initially fixed between the populations, spread evenly across the genome. All alleles of selective/phenotypic effect were codominant with dominance = 0.5.

In addition to reproductive isolation, individual fitness also depended on their phenotypic distance from a climatic optimum. This optimum was initially 0, and during the burn in period oscillated from -5 to 5 (in arbitrary units) every 500 generations based on the formula: *sin*(π \**generation* / 500) /5. The individual phenotype was determined by alleles at QTL-like climate loci which could appear via mutation at all sites other than RI or ancestry tracking loci (i.e. 99899 - *l*sites). Climate QTL mutations occurred at a rate per locus per sample per generation and their phenotypic effect was drawn from a gaussian distribution with a mean of zero and a standard deviation of *QTLSD*. Conceptually, these QTL climate alleles modify whether an individual is “hot” (positive effects) or “cold” (negative effects) adapted.

The first step of the simulations was a burn-in of 10*Ne* generations to simulate the generation of standing genetic variation under normal climatic conditions. At the end of the burn-in period, the complete state of each replicate simulation was saved. Each simulation was then continued under both a “control” and climate change scenario for an additional 100 generations. In the control scenario, the environmental oscillation continued as normal. In contrast, under the climate change scenario the phenotypic optimum increased by a rate of Δ each generation without oscillation. In each generation we recorded the average degree of reproductive isolation, mean fitness, the mean and standard deviation of the climate phenotype and the amount of introgressed ancestry for each population. Reproductive isolation (RI) was calculated accounting for the extrinsic and BDM loci. For extrinsic loci, RI was the difference in fitness for an average individual in their home habitat vs. the foreign habitat. For BDMs, since fitness penalties occur only in F1 hybrids and beyond, we calculated the expected average magnitude of BDM fitness costs based on Hardy-Weinberg expectations in F1s. Finally, for each simulation we report the mean introgressed ancestry and reproductive isolation between the start and end of control and test scenarios, as well as the mean rate of phenotypic change in Haldanes for the test scenario. A Haldane is a measure of evolutionary change in log mean trait value in units of standard deviation of that log trait29. All formulas used in the simulation are presented below and all code for underlying simulations is available at <https://github.com/owensgl/adaptive_introgression>.

To explore the parameter space under which adaptive introgression mediates RI collapse, we systematically varied the following parameters: mutation rate (*μ*), migration rate (*m*), strength of divergent selection (*sRI*), the number of divergently selected loci (*nRI*), the proportion of BDMs (*prBDM*)*,*the standard deviation of QTL effect sizes (*QTLSD*) the recombination rate (*r*), and the rate of climate change (Δ). We varied each parameter independently and kept the other parameters at a default value known to permit a low level of introgression in preliminary tests (Supplementary Table 1). Each parameter set was replicated 100 times. All analyses were carried out in R 3.5.130 and plotting was done using ggplot231.

Finally, while our primary goal was testing the detrimental effects of hybridization, we also examined the potential *beneficial* effects of climate change induced introgression, i.e. to what degree introgression facilitates adaptation. To do this, we ran simulations varying the rate of climate change with (*m*=0.01) or without (*m*=0) migration. At the last generation (gen=10,100), we compared the average climate phenotype to the current phenotypic optimum. We defined “adaptational lag” as the difference between these values divided by the rate of climate shift. This represents how many generations behind the current generation that the population is adapted to. For example, assume the optimum increases by 2 per generation and is currently 100, if the average phenotype is 90, then the adaptational lag is 5 (e.g. (100 - 90) / 2).

*Fitness calculations*

All symbols used in the following equations are described and compiled in Supplementary Table 3. Fitness in this model is determined by how well the sample’s phenotype matches the current climate optimum as well as the genotypes of reproductive isolation alleles. During the initial burn-in of *b* generations, the climate optimum oscillates slightly above and below zero with an amplitude of *a* and a frequency of *f* Eq. 1, and then increases linearly during the climate shift period by units per generation Eq. 2.

(1)

(2)

The fitness effect of the climate match is calculated by first calculating each sample’s climate phenotype by summing the effect sizes, *Q,* of each copy of a QTL allele present in the sample. In this way, QTLs are all additive and co-dominant (dominance=0.5). The fitness effect of this phenotype is determined by a gaussian function with a mean of the current climate optimum and a standard deviation of Eq. 3. Samples with climate phenotypes distant from the optimum have reduced relative fitness.

(3)

Along with climate adaptation, fitness is also determined by the alleles at reproductive isolation loci which are either extrinsic or intrinsic epistatic. In most simulations, all RI loci are extrinsic, except in simulations designed to test the effect of Bateson-Dobzhansky-Muller (BDM) incompatibilities. The effect of *l* extrinsic RI loci is determined by summing the counts of non-local alleles (*gaway)* divided by 2 (for co-dominance) Eq. 4.

(4)

BDM loci were initialized as randomly selected pairs. In each pair, both populations are initially fixed for different alleles in the simulation. One locus is set as the derived state (*A*) in population 1 and the other as the derived state (*B*) in population 2. Negative interactions occur when both derived alleles are present in a single diploid individual and are equally deleterious in all combinations (Supplementary Table 2). Thus each BDM pair can produce epistasis counted as 0 or 2, and this is summed for each individual Eq. 5.

(5)

The total sum of divergently selected alleles and BDM epistasis are treated as independent alleles with selection coefficients of and are multiplicatively added Eq. 6. Importantly, this puts BDM and extrinsic alleles on the same scale, so they are comparable. Although this model results in diminishing returns in terms of absolute fitness, relative fitness scales correctly.

(6)

The two measures of fitness are combined to create the fitness measure of each sample Eq. 7.

(7)

*Reproductive isolation calculations*

To see if rapid shifts in the phenotypic optimum can lead to reverse speciation, we measured average reproductive isolation during the climate shift period. We operationally defined reproductive isolation as the difference in fitness between an average migrant individual vs. an average non-migrant (i.e. the difference in expected “home” vs. “away” fitness). This is determined purely based on the extrinsic and BDM RI alleles, and does not include climate QTL alleles.

For extrinsic loci, we can think of the reproductive isolation in terms of a representative individual being transported from its own population to the other population and measuring its relative fitness. We calculate the average “home” fitness by measuring the proportion of foreign alleles and applying it to Eq. 6 to get an expected fitness penalty. This is averaged for both populations Eq. 8.

(8)

“Away” fitness was calculated in a similar way, but using the proportion of home alleles when calculating the expected fitness penalty Eq. 9.

(9)

Reproductive isolation from BDMs are more complicated because BDMs have no effect in generation 0 after migration (i.e. before breeding). Their effect only appears after mating in F1s and beyond which have combinations of alleles from both populations. Thus, for BDMs, we estimate expected RI based on Hardy-Weinberg expectations of genotype frequencies assuming mating within the population, and mating between populations (Supplementary Tables 4 & 5). From this expectation, we estimated the expected amount of BDM epistasis (Eq 10 & 11). These formulae assume that BDM loci interacting pairs are unlinked and segregate independently.

After all the average fitness values are compiled, an estimated RI score is calculated using Eq. 12. This RI score represents the *average fold higher fitness in the home population compared to the other (away) population*. If there is no RI or populations are completely admixed, RI will equal 1, representing equal fitness in either environment.

*Selection coefficient visualization*

To understand the scale of fitness effects in the simulation, we collected individual genotype values and sample fitness for each sample in each generation during the climate shift period (i.e. generation 10001 to 10100). We then calculated *s* using Equation 13. This equation compares the mean fitness for samples homozygous for the mutation with samples homozygous for the wild-type allele and is normalized by the mean fitness for wild-type samples. We required that each group needed to have at least 2 individuals for *s* to be calculated.

In this case, *s* is the realised fitness effect which includes the effect of linked loci. We plotted mutations with intermediate allele frequencies (0.1 < frequency < 0.9) because at frequencies closer to 0 and 1, mutations are most often found in first generation migrants, and have more extreme variation in fitness (Figure 1d).

*Data availability*

All code for underlying simulations are available at <https://github.com/owensgl/adaptive_introgression>.

## **Supplementary Discussion**

*Introgression and extinction*

Here we’ve focused on the possibility of species collapse, but another more common predicted effect of climate change is extinction. In our model, population sizes are constant and fitness is relative, so extinction is impossible. Despite this, our results do have implications for the likelihood of extinction.

If RI alleles contribute to local adaptation (i.e. are extrinsic), then the collapse of RI must result in the spread of fitness reducing maladaptive non-local loci, similar to the concept of linkage drag in plant breeding32. The net effect of the introgression is positive in our climate change scenario, since the maladaptive introgressed alleles are linked to positive climate QTL alleles, but the overall fitness of the population is lower than it would be if it adapted *without* introgression (Figure 1). In this way, adaptive introgression makes populations better adapted to the changing climate, but less adapted to their home niche. In the real world, this may be reflected in reduced population size or growth rate and could increase the chance of extinction. This effect is dependent on the amount and type of RI loci swept to fixation by linkage with beneficial climate QTL, which in itself will depend on the speed of fixation and amount of recombination. Thus stronger selection, smaller population size or reduced recombination rate will all increase linkage drag. Note that if RI is intrinsic, this doesn’t necessarily apply because intrinsic RI loci are only detrimental based on interactions with other loci and may have no fitness penalty if all RI alleles are homogenized by introgression.

*Linkage and the genetic architecture of climate adaptation*

A key aspect of our model is that while RI loci occur at predefined intervals in the genome, climate-sensitive alleles can arise at any other locus in the genome. This allows for climate-sensitive alleles to become readily linked to RI-causing alleles and eventually introgress if the combined effect of positively selected climate alleles exceeds the deleterious effect of the linked RI allele. The incidental establishment of this linkage within the two adapting populations is a fundamental cause of later introgressive collapse. This is supported by our simulations that varied the number of RI loci and also incidentally varied the average degree of linkage between all climate-sensitive loci and all RI loci. We found that in simulations with more RI loci, and therefore a higher probability of linkage between RI and climate-sensitive loci, there was greater loss of reproductive isolation. Thus, a key question is whether such linkage could plausibly be established in a natural population.

Several lines of evidence suggest that this is likely to be true. First, the genetic architecture of adaptation to a changing climate is likely to closely resemble the architecture of local adaptation in general, i.e. a large number of small effect alleles with a smaller number of large effect loci (reviewed in 33). This idea is directly supported by recent work showing that climatic adaptation in conifers is underlain by large number of loci scattered throughout the genome, with the majority of these showing modest phenotype-environment correlations34. Secondly, recent analyses of large human datasets support the idea that most complex traits (of any kind) are probably determined by a large number of small-effect loci found nearly everywhere in genome along with a handful of “core genes”35. Thus, given that the genetic architecture of RI is itself likely to be highly polygenic (further discussed in 36), it seems highly plausible that linkage between climate-sensitive alleles and RI alleles can readily occur in natural populations.

*The role of recombination rate*

Recombination rate is thought to play a key role in mediating patterns of divergence and introgression in natural populations37-39. Specifically, regions of high recombination are thought to be less resistant to gene flow because of decreased linkage between alleles conferring RI. We see this in our simulations, as simulations with higher recombination rates have greater amounts of introgression in both control and climate change scenarios (Supplementary Figure 2). Interestingly, we observe two different patterns for RI loss (Figure 2). Under control scenarios, higher recombination results in more RI loss because it allows mildly deleterious foriegn RI loci to separate from their haplotype and drift to high frequency. Under climate change, high recombination results in less RI loss because in this case RI loss is driven by adaptive introgression of climate QTL dragging foriegn RI loci; high recombination is more likely to free these linked RI loci.

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