# Adaptive introgression during environmental change can weaken reproductive isolation

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**Anthropogenic climate change is an urgent threat to species diversity1,2. One aspect of this threat is the merging of species through increased hybridization3. The primary mechanism for this collapse is thought to be the weakening of ecologically-mediated reproductive barriers, as demonstrated in cases of “reverse speciation”4,5. Here, we expand on this idea and show that adaptive introgression between species adapting to a shared, moving climatic optimum can readily weaken *any* reproductive barrier, including those that are completely independent of climate. Using genetically explicit forward-time simulations, we show that genetic linkage between alleles conferring adaptation to a changing climate and alleles conferring reproductive isolation (intrinsic and/or non-climatic extrinsic) can lead to adaptive introgression facilitating the homogenization of reproductive isolation alleles. This effect causes the decay of species boundaries across a broad and biologically-realistic parameter space. We explore how the magnitude of this effect depends upon the rate of climate change, the genetic architecture of adaptation, the initial degree of reproductive isolation, the degree to which reproductive isolation is intrinsic vs. extrinsic, and the mutation rate. These results highlight a previously unexplored effect of rapid climate change on species diversity.**

One potential effect of global climate change (GCC) is increased interspecies hybridization by, for example, bringing together species ranges or disrupting mating timing4. Such hybridization can cause a common species to subsume a rare species6 or the collapse of multiple species into a single hybrid swarm7. In both cases, species diversity is lost as has been seen in smaller localized environmental shifts5,7.

There is a rich theoretical literature dedicated to the study of the dynamics of interspecific hybridization (reviewed in e.g. 8-10). However there has thus far been poor integration between models of reproductive isolation and models of adaptation to climate change. The fact that introgression can transfer alleles between species has led to the idea that hybridization could facilitate adaptation to GCC through the transfer of adaptive alleles between species, i.e. adaptive introgression. This has traditionally been studied in the context of species/populations with pre-existing differential adaptation to the changing climate variable; for example a warm adapted species transferring alleles to a cold adapted species11. In this example, one species acts as a pool of alleles preadapted to a future climatic optimum. Importantly, in these types of models, introgression is being driven by selection and not demographic processes or perturbations of prezygotic isolation, as seen in other models where climate change drives hybridization.

What has not been appreciated in previous models of adaptation to a changing climate is that during a rapid environmental shift, segregating variation within two reproductively isolated species could theoretically undergo adaptive introgression even if neither species is particularly preadapted to the environmental shift. We propose that climate-induced adaptive introgression could readily occur in most species because (1) the identity of the particular alleles involved in climatic adaptation are likely idiosyncratic in each species/population, and (2) these alleles could, in principle, be globally adaptive under a GCC scenario. Indeed, segregating climate adaptation alleles (or linked blocks of alleles) could easily be strong enough to outweigh the fitness costs of any linked reproductive isolation (RI) alleles. As a side effect, RI alleles could readily be homogenized between species, reducing RI and precipitating the collapse of species boundaries. This scenario dramatically increases the likelihood of GCC-induced introgression from populations differing in altitude or latitude, to nearly any parapatric pair capable of hybridization, even if RI is initially high.

Here, we directly test the role of climate-induced adaptive introgression in degrading reproductive barriers using state-of-the-art forward time population genetic computer simulations (Supplemetary Figure 1, Online Methods). We consider the scenario of two parapatric species inhabiting demes in two different habitats. These species exchange migrants at a low level, but RI 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 directly affect the degree of local adaptation and/or RI, i.e. RI 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 (i.e. climate QTL) 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 then measure the amount of RI lost at the end of the climate change period, in comparison to a control period of the same length. With our simulations we ask three questions; (1) Can climate change drive RI collapse and what factors control its severity? (2) To what extent does introgression facilitate adaptation to climate change? (3) Do the latter two phenomena occur under realistic evolutionary conditions?

When climate change is rapid, we find that adaptive introgression of climate QTL alleles rapidly drives the homogenization of allele frequencies at linked RI loci between species. Figure 1 visualizes one example simulation where after 100 generations of climate change, RI is degraded to nearly half its original strength (Figure 1a) and introgressed climate QTL alleles are common (Figure 1b). As climate QTL alleles move between populations, RI and neutral alleles hitchhike along with them resulting in substantial genome-wide introgression (Figure 1a & 1c). In contrast, in the control scenario without climate change, RI remains intact and introgression is minimal (Figure 1 e-h).

For a wide range of parameter values we find decreased RI and increased introgressed ancestry under the climate change scenario (Figures 2 and Supplementary Figure 2). When adaptive variation is limited, RI is initially weak, or environmental change is rapid, complete genetic homogenization is likely. In these cases, RI is completely degraded and would clearly represent speciation reversal in a natural system. In other cases, introgression is increased during environmental change, but populations do not completely homogenize (Supplementary Figure 2). In these cases, RI is still eroded between populations (Figure 2). Importantly, we believe our estimates of RI loss are likely conservative, because we do not include any additional factors that would contribute to species collapse (e.g. cases where RI is directly affected by a change in climate).

We found that in the absence of divergent selection intrinsic reproductive isolation (BDM incompatibilities) was unable to maintain RI during the burn-in period. This result is consistent with previous modelling of parapatric speciation12,13. Consistent with their effect in the burn-in period, during climate change, introgression and RI loss is enhanced when RI is purely intrinsic. While other forms of intrinsic isolation that are more resistant to introgression have been suggested14, any intrinsic isolation locus can be weakend by introgression. In contrast the strength of an extrinsic isolation locus is independent of genomic background , and as such we do not expect any form of intrinsic isolation to be more resistant to the adaptive introgression than the extrinsic isolation modelled here (assuming similar genomic architecture). Thus, although we have focused on extrinsic RI, intrinsic RI is also susceptible to adaptive introgression.

The ultimate question of which species are in danger of reverse speciation is dependent on a multitude of interacting factors, but based on our simulations we can highlight several risk factors:

1. For hybridization to be an issue, a potential hybridizing species must be at least in parapatry. Surveys have estimated the percent of species that hybridize with at least one other congener to be around 10-25%, although if climate change disrupts species ranges or premating isolation, that number may increase15.
2. The rate of environment change and the steepness of the changing fitness landscape. Species with broader climate niches will be less susceptible because they will be under weaker selection.
3. The genetic architecture of climate adaptation within species. Species with numerous large effect climate adaptation alleles segregating within their gene pool will be more able to adapt to the changing climate *without* introgressed alleles. Low diversity species will be more susceptible to adaptive introgression.
4. The genetic architecture of reproductive isolation between species. Species with few large effect RI loci will be more resistant to RI decay than species with a more diffuse and polygenic RI architecture. See the Supplementary Discussion for further exploration on the role of linkage and recombination rate.
5. The demographic and life history of the species. Unbalanced population sizes may result in one population harboring more adaptive alleles and lead to unbalanced introgression. Small populations will also be more susceptible to extinction due to the fitness costs of introgressed RI alleles. Features that reduce effective population size, e.g. high variance in reproductive success, are also likely to have reduced diversity of climate adapting alleles.

Our simulations suggest that rapidly changing environments can cause the collapse of species barriers even when the mechanisms of reproductive isolation are independent of climate. We modelled a scenario in which the strength of RI (modelled as divergent selection) is (a) invariant throughout (i.e. not reduced by environmental change itself) and (b) orthogonal to the strength of climate-mediated selection (i.e. extrinsic RI alleles do not affect the climate phenotype). This is an important departure from previous work, in which the collapse of reproductive isolation or “reverse speciation” occurs because RI is itself dependent on the environment (e.g. trophic or sensory niche5).

This difference in modelling approach has several important implications. For one, the collapse of RI we describe here can occur in any population where adaptive introgression is possible (i.e. RI is not absolute and the climate-mediated selective optimum is to some degree shared). This greatly expands both the number of populations that may be susceptible to introgressive collapse and the potential severity of such collapses. For example, adaptive introgression could act in concert with the collapse of climate-mediated reproductive barriers, or a reduction in population sizes, accelerating reverse speciation.

Although we have framed our discussion in the context of climate change, our results are applicable to any strong, consistent, and shared selective event. These events include any environmental or ecological disturbance that alters the shared selective landscape of species such that they are sufficiently displaced from their selective optima, i.e. selection is sufficiently strong. One such event that has been studied in natural systems is eutrophication, which has been suggested to have caused speciation reversal in European lake whitefish5. Thus far, this reversal has been attributed to changes in RI as a direct result of ecological and/or behavioural changes. However, if eutrophication exerts a common selective pressure on a group of parapatric species (e.g. mediated through changes in water chemistry) introgression could become adaptive and contribute to the collapse of species boundaries. Similarly, ocean acidification could be a strong source of shared selection and may induce introgression between previously well isolated species16.

While we focus our discussion on how introgression can lead to species merging together, it is likely that the adaptive introgression of climate QTL also increases the chance that one or both species can adapt to a changing environment and avoid extinction. We cannot directly address this question in our model (see Supplementary Discussion) but we do see that when all introgression is prevented the lag between the current phenotype and the current optimum increases (Figure 3). This is consistent with the larger total gene pool of adaptive variants available when gene flow is possible. We see this effect most strongly when climate change is rapid, suggesting the benefits of introgression mainly occur when adaptation is most challenging.

A strong shared selection pressure is ultimately the key mediator of the collapse of RI we observed. Was the magnitude of simulated selection necessary to cause this collapse realistic? One way to assess this is to measure the magnitude of the phenotypic response to selection in our simulations and compare it to estimates from natural systems. In our case, the phenotypic response to selection ranged from 0.01-0.06 Haldanes (standard deviations per generation) (Supplementary Figure 3). This is in line with the magnitude of phenotypic response observed in both natural and anthropogenically-induced selection17. Further, this is well below the theoretical threshold of 0.1 Haldanes thought to result in an unsustainable long-term response to selection (for Ne = 50018,19).

Another way of assessing the realism of our scenarios is comparing the selection coefficients of climate QTL in our simulations with values measured in empirical studies. We measured selection coefficients in our example simulation by comparing relative fitness values for samples with and without each locus (Supplementary Methods). Only 0.7% of introgressed climate QTL loci had selection coefficients > 1, again well within the range of natural estimates20. Thus, the strength of selection we modelled was in no way extreme nor would it necessarily lead to the extinction of the populations under natural conditions. It is also worth noting that the estimated rate of phenotypic change in wild populations due to future GCC is thought to be at least as large as the rates we described here, and are projected to likely exceed 0.1 Haldanes in many cases21,22. In sum, the global strength of phenotypic selection simulated here was not unrealistically high, and if anything represents a conservative adaptive scenario.

Hybridization is a double-edged sword under rapid environmental change. It can provide species access to a larger pool of adaptive alleles but these alleles may be linked to RI alleles, weakening RI and potentially leading to speciation reversal. Importantly, our work highlights the dangers of hybridization for a much wider pool of species, not just those on range margins or with existing porous species boundaries. In the longer term, we predict that specific cases of speciation reversal should be linked to climate change but we also predict effects other than speciation reversal. One core prediction of our model is that alleles conferring adaptation to a shared climate will be more likely to introgress between species. Although identifying all the loci underlying climate adaptation is challenging, recent work by Exposito-Alonso et al.23 highlights progress towards this goal. Such an approach can be combined with sequencing data in related species to identify where introgression is most likely to occur. Our results also suggest that climate change should cause hybrid zones to become increasingly porous as climate adaptation alleles move between species and that this effect would be stronger in regions with more dramatic climate change (e.g. arctic regions24). This prediction could be tested by resampling previously studied hybrid zones or by comparing contemporary samples to museum and herbarium samples. Confirmation of these predictions would show that climate adaptation is occurring through a larger multi-species gene pool and be a warning sign for the future homogenization of these species.

*Author contributions*

G.O and K.S designed the study, created the model, analysed the results and wrote the paper.

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*Competing Interests*

The authors declare no competing interests.

## References

1. Thomas C.D., Cameron A., Green R.E., Bakkenes M., Beaumont L.J., Collingham Y.C., Erasmus B.F., De Siqueira M.F., Grainger A., Hannah L. & Hughes L. 2004. Extinction risk from climate change. Nature. 427(6970):145.

2. Hoffmann A.A. & Sgrò C.M. 2011 Climate change and evolutionary adaptation. Nature. 470(7335):479.

3. Todesco M., Pascual M.A., Owens G.L., Ostevik K.L., Moyers B.T., Hübner S., Heredia S.M., Hahn M.A., Caseys C., Bock D.G., Rieseberg L.H. 2016. Hybridization and extinction. Evolutionary Applications. 9(7):892-908.

4. Chunco AJ. 2014 Hybridization in a warmer world. Ecology and Evolution. 4(10):2019-2031.

5. Vonlanthen P., Bittner D., Hudson A.G., Young K.A., Müller R., Lundsgaard-Hansen B., Roy D., Di Piazza S., Largiadèr C.R. & Seehausen O. 2012. Eutrophication causes speciation reversal in whitefish adaptive radiations. Nature. 482(7385):357.

6. Oliveira R., Godinho R., Randi E. & Alves P.C. 2008. Hybridization versus conservation: are domestic cats threatening the genetic integrity of wildcats (*Felis silvestris silvestris*) in Iberian Peninsula?. Philosophical Transactions of the Royal Society of London B: Biological Sciences. 363(1505):2953-2961.

7. Taylor E.B., Boughman J.W., Groenenboom M., Sniatynski M., Schluter D. & Gow J.L. 2006. Speciation in reverse: morphological and genetic evidence of the collapse of a three‐spined stickleback (*Gasterosteus aculeatus*) species pair. Molecular Ecology.15(2):343-355.

8. Abbott, R., Albach, D., Ansell, S., Arntzen, J. W., Baird, S. J., Bierne, N., ... & Butlin, R. K. 2013. Hybridization and speciation. Journal of evolutionary biology, 26(2), 229-246.

9. Barton N. H., 2013. Does hybridization influence speciation? Journal of Evolutionary Biology. 26(2):267–269.

10. Seehausen, O. 2013. Conditions when hybridization might predispose populations for adaptive radiation. Journal of Evolutionary Biology, 26(2), 279-281.

11. Gómez J.M., González-Megías A., Lorite J., Abdelaziz M. & Perfectti F. 2015. The silent extinction: climate change and the potential hybridization-mediated extinction of endemic high-mountain plants. Biodiversity and Conservation. 24(8):1843-1857.

12. Barton, N. & Bengtsson, B.O. 1986. The barrier to genetic exchange between hybridising populations. Heredity. 57(3):357-376.

13. Bank C., Bürger R. & Hermisson J. 2012. The limits to parapatric speciation: Dobzhansky–Muller incompatibilities in a continent–island model. Genetics. 191(3):845-863.

14. Lindtke, D. & Buerkle, C.A. 2015. The genetic architecture of hybrid incompatibilities and their effect on barriers to introgression in secondary contact. Evolution. 69(8):1987-2004.

15. Mallet J. 2005. Hybridization as an invasion of the genome. Trends in Ecology & Evolution. 20(5):229-237.

16. Pespeni M.H., Sanford E., Gaylord B., Hill T.M., Hosfelt J.D., Jaris H.K., LaVigne M., Lenz E.A., Russell A.D., Young M.K. & Palumbi S.R. 2013. Evolutionary change during experimental ocean acidification. Proceedings of the National Academy of Sciences. 110(17):6937-6942.

17. Hendry A.P., Farrugia T.J. & Kinnison M.T. 2008. Human influences on rates of phenotypic change in wild animal populations. Molecular Ecology. 17(1):20-29.

18. Lynch, M., & R. Lande. 1993. Evolution and extinction in re­sponse to environmental change. Pp. 234-250 in P. M. Kareiva, J. G. Kingsolver, and R. B. Huey, eds. Biotic interactions and global change. Sinauer, Sunderland, Mass.

19. Bürger R. & Lynch M. 1995. Evolution and extinction in a changing environment: a quantitative‐genetic analysis. Evolution. 49(1):151-163.

20. Kingsolver, J.G., Hoekstra H.E., Hoekstra J.M, Berrigan D., Vignieri S.N., Hill C.E., Hoang A., Gibert P., & Beerli P. 2001. The strength of phenotypic selection in natural populations. The American Naturalist. 3(157):245-261.

21. Gienapp P., Leimu R. & Merilä J. 2007. Responses to climate change in avian migration time—microevolution versus phenotypic plasticity. Climate Research. 35(1-2):25-35.

22. Merilä, J., & Hoffmann, A.A. 2016. Evolutionary Impacts of Climate Change. In *Oxford Research Encyclopedia of Environmental Science*. Oxford University Press.

23. Exposito-Alonso M., Vasseur F., Ding W., Wang G., Burbano H.A., & Weigel, D. 2018. Genomic basis and evolutionary potential for extreme drought adaptation in *Arabidopsis thaliana*. Nature ecology & evolution. 2(2):352.

24. Osborne E., Richter-Menge J., & Jeffries M. 2018. Arctic Report Card 2018, https://www.arctic.noaa.gov/Report-Card.

## Figure Captions

**Figure 1 | An example simulation with Δ = 1.5, illustrating climate driven adaptive introgression.**

Panels a-d present the test climate change scenario, while e-h are the control scenario. (a & e) The upper half is the average introgressed ancestry for each species (purple and green) and the average reproductive isolation between populations (black). The lower half is the ancestry for neutral loci during the post-burn-in period at 20 generation intervals. The top and bottom parts of this portion represent species 1 and 2 respectively. (b & f) The allele frequency trajectory for introgressed climate QTL color coded by QTL strength. Color codes QTL effect; -ve phenotypic effect (blue), +ve effects (light red) or large +ve effect (>2, dark red). (c & g) The allele frequency trajectory for introgressed RI alleles (d & h) The distribution of selection coefficients on QTL loci per species per generation. Color groups represent QTL with -ve phenotypic effect (blue), +ve effects (light red) or large +ve effect (>2, dark red). Plot is filtered to only include loci with allele frequency < 0.9 and > 0.1.

**Figure 2 | The effect of simulation parameters on RI loss.**

The average reproductive isolation at generation 10,100 for climate change (dotted dark) and control simulations (solid light), while varying individual parameters. The shaded area encompasses 95% of the simulations. RI is defined as the home fitness advantage which is the fold fitness advantage for the average sample in its home environment compared to the alternate environment based on divergent selection and BDM loci. A value of 1 means equal fitness in both environments and there is no RI. The dashed line is the initial and maximum level of RI for each simulation. Individual parameters were varied to show the effect of (a) climate QTL effect size standard deviation, (b) optimum shift per generation (delta), (c) migration rate, (d) climate QTL mutation rate, (e) proportion of RI loci that are BDM instead of extrinsic, (f) the recombination rate, (g) the fitness effect of each RI loci and (h) the number of RI loci.

**Figure 3 | Hybridization enhances adaptation at high rates of climate change.**

The adaptational lag at the final generation for simulations with (m=0.01, solid light) and without migration (i.e. hybridization) (m=0, dotted dark). The shaded area encompasses 95% of the simulations. Adaptational lag is defined as the phenotypic optimum minus the phenotypic mean divided by the rate of climate change, and represents how many generations behind the changing optimum that the population is.

## **Online Methods**

*Model details*

We implemented our 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 between 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 (*BDMpr*)*,*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>.

## References

25. Haller B.C. & Messer P.W. 2018. SLiM 3: Forward genetic simulations beyond the Wright–Fisher model, Molecular Biology and Evolution. msy228

26. Bateson W. 1909. Heredity and variation in modern lights. Darwin and Modern Science. 85–101

27. Dobzhansky, T. H. 1936. Studies on hybrid sterility. II. Localization of sterility factors in Drosophila pseudoobscura hybrids. Genetics. 21(2): 113.

28. Muller, H.J. 1942. Isolating mechanisms, evolution, and temperature. Biol. Symp. 6:71–125.

29. Gingerich P.D. 1993. Quantification and comparison of evolutionary rates. American Journal of Science, 293(A):453-478

30. R Core Team (2018). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. URL https://www.R-project.org/.

31. Wickham H. 2016. ggplot2: elegant graphics for data analysis. Springer.