

# The genetic backburn: using rapid evolution to halt invasions.

Ben L. Phillips<sup>1</sup>, Richard Shine<sup>2</sup>, and Reid Tingley<sup>3</sup>

<sup>1</sup>*School of Biosciences, University of Melbourne*

<sup>2</sup>*School of Environmental and Life Sciences, University of Sydney*

<sup>3</sup>*School of Biosciences, University of Melbourne*

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# 1 Abstract

2 The impact of an invasive species depends upon the extent of area across which it ultimately spreads.  
3 A powerful strategy for limiting impact, then, is to limit spread, and this can most easily be achieved  
4 by managing or reinforcing natural barriers to spread. Using a simulation model, we show that rapid  
5 evolutionary increases in dispersal can render permeable an otherwise effective barrier. On the other  
6 hand, we also show that, once the barrier is reached, and if it holds, resultant evolutionary decreases in  
7 dispersal rapidly make the barrier more effective. Finally, we sketch a strategy – the genetic backburn  
8 – in which low-dispersal individuals from the range core are translocated to the nearside of the barrier  
9 ahead of the oncoming invasion. We find that the genetic backburn — by preventing invasion-front  
10 genotypes reaching the barrier, and hastening the evolutionary decrease in dispersal — can make barriers  
11 substantially more effective. In our simulations the genetic backburn never reduced barrier strength,  
12 however, the improvement to barrier strength was negligible when there was substantial long distance  
13 dispersal, or when there was no genetic variation for dispersal distance. The improvement in barrier  
14 strength also depended on the trade-off between dispersal and competitive ability, with a stronger trade-  
15 off conferring greater power to the genetic backburn.

16     Keywords — *Barrier, Contemporary evolution, Invasive species, Spatial sorting, Targeted gene flow*

## 2 Background

Species ranges are shifting at an unprecedented rate in response to global change, and evolutionary theory provides an important perspective on this phenomenon [Perkins, 2012]. Rates of dispersal and reproduction evolve upwards on expanding range fronts and so attempts to predict rates of range expansion that assume their constancy are likely to under-estimate the rate at which populations spread [Phillips et al., 2010b]. At the same time, theory predicts that trade-offs between dispersal, reproduction, and competitive ability should lead to less competitive phenotypes emerging on the invasion front [Burton et al., 2010]. Finally, once an invasion front encounters an environmental limit to its further spread, theory predicts that traits that increase rates of dispersal will be selected against [Kubisch et al., 2010, Phillips, 2012].

These predictions regarding the evolution of range expansion phenotypes have important implications for managing the spread of invasive species. The impact of any particular invader typically scales with the size of the area it comes to inhabit [Epanchin-Niell and Hastings, 2010]. Thus, once an invasion is underway, our best strategy will often be containment; to slow or limit the extent of spread [Sharov and Liebhold, 1998a]. Unfortunately, localised control efforts aimed at stopping spread are rarely cost-efficient because they are typically directed at low-density populations: detection and reward-for-effort are low. A powerful alternative is to exploit natural landscape barriers that impede spread. Islands, for example, commonly offer refuge from invasive species, at almost no cost to managers. Islands, however, are typically small, so a focus on “mainland” barriers can potentially prevent a much larger area from being colonised. Barrier zones were used to temporarily halt, and successfully slow, the spread of the gypsy moth in the US [Sharov and Liebhold, 1998b], and potentially have application wherever abiotic conditions narrow the available habitat down to “linear” corridors. Examples include invasions along streams and rivers [e.g., Kerby et al., 2005], along coastlines [e.g., Cousens and Cousens, 2011], and other distributional choke-points [e.g., Tingley et al., 2013].

Rapid evolution can influence the effectiveness of barrier zones in two ways. First, increased dispersal rates of the vanguard may render barriers more easily surmountable: for any given barrier zone width, individuals from invasion-front populations are more likely to cross that barrier than are conspecifics from core populations [Travis et al., 2010]. Second, once a barrier is reached, natural selection and spatial sorting rapidly favour individuals of lower dispersal ability, driving dispersal rates downwards

again [Kubisch et al., 2010, Phillips, 2012]. Thus, any barrier that is initially effective may become more effective as time passes because the population abutting the barrier will evolve lower rates of dispersal. Finally, by exploiting the emergent differences in dispersal and competition abilities of core vs frontal populations, managers can potentially make barriers more effective through translocation: by translocating the less dispersive phenotypes of the range core to the near side of the barrier – setting up a genetic backburn – it may be possible to prevent invasion front phenotypes ever encountering the barrier.

Our interest in these issues was stimulated by the spread of invasive cane toads (*Rhinella marina*) through the Australian tropics. The toads are moving from well-watered eastern areas of the continent into severely arid western regions; and within a decade or so, will encounter a narrow strip of coastal habitat where the only water-sources are artificial ponds and stock-watering sites [Florance et al., 2011, Tingley et al., 2013]. Thus, we might be able to prevent toads occupying 268,000km<sup>2</sup> of their potential range if we can prevent them moving down that narrow coastal corridor, and this can be achieved by restricting their access to artificial watering-points [Tingley et al., 2013, Letnic et al., 2014]. Models are encouraging as to the effectiveness of that barrier (if water sources could be eliminated), but those models have not incorporated the possibility of evolutionary responses to the barrier. Potentially, the toads’ capacity for rapid evolutionary shifts in traits that affect rates of dispersal [Phillips et al., 2010b, Brown et al., 2014] might substantially affect the feasibility of a barrier in curtailing the Australian expansion of cane toads.

Here we investigate these issues with a simulation model. Although motivated by the toad example, we frame the model in general terms and do not incorporate specific aspects of cane toad biology and life history. Our purpose is to investigate the general issue of dispersal evolution around spread barriers, and how this might affect management outcomes and strategies. Using the model, we examine whether:

1. rapid evolution on the invasion front generates invasion front phenotypes that are more capable of breaching a barrier zone;
2. once a barrier is reached, rapid evolution make barriers more effective over time; and
3. translocating individuals from the core of the range to the near side of the barrier can establish a "genetic backburn" that prevents invasion front phenotypes reaching the barrier and so makes barriers substantially more effective.

## 3 Methods

To investigate these ideas, we built a spatially-explicit simulation model that tracks a population spreading through space. The population is composed of sexually hermaphroditic individuals (obligately outcrossing) with discrete generations. The choice to make individuals hermaphrodites allows us to incorporate sexual recombination, but without the computational overhead of tracking non-reproducing individuals (i.e., males). All simulations were executed in R [Team, 2014] and the code is available at <https://github.com/benflips/GBB>.

### 3.1 Population dynamics

All individuals in the population have a maximum density-independent rate of reproduction,  $R_{max}$ , modified by density dependence, described using the Beverton-Holt model [Beverton and Holt, 1958], which yields their expected reproductive output,  $E(W_i)$ :

$$E(W_i) = \frac{R_{max}}{1 + \alpha_i N}$$

Here  $\alpha$  determines the strength of competition in the system, and this is where we introduce individual variation in reproductive output. We set  $\alpha_i = \frac{R_{max}-1}{N^* w_i}$ . In this formulation  $N^*$  represents the carrying capacity that would be achieved if all individuals achieved a fecundity of  $R_{max}$ , and  $w_i$  ( $0 \leq w_i \leq 1$ ) is a fitness modifier that causes some individuals to be more competitive at carrying capacity than others (see Evolutionary dynamics section, below).

Finally, we introduced demographic stochasticity into the model by drawing each individual's expected fecundity from a poisson distribution:  $W_i \sim \text{Pois}(E(W_i))$ .

### 3.2 Spatial dynamics

We set this population up on a 1D lattice of contiguous cells (with a single reflective boundary constraining  $x \geq 0$ ). Each cell takes up a length of 1 unit on the lattice, and local dynamics plays out within each cell on the lattice. Individuals disperse in continuous space, but are aggregated to the cell for the purposes of local dynamics.

99 Immediately after birth (and the death of parents), individuals disperse according to their individual  
 100 dispersal phenotype,  $d_i$ . Dispersal is treated as a stochastic process with each individual displacing ac-  
 101 cording to a draw from a scaled t-distribution. This distribution has a shape parameter,  $v$ , (corresponding  
 102 to the familiar degrees of freedom) controlling the kurtosis, and a scale parameter (analogous to  $\sigma$  in  
 103 the Gaussian distribution) affecting the standard deviation. As  $v \rightarrow \infty$ , the distribution converges on  
 104 a Gaussian. Thus, by specifying particular values for  $v$ , we can use the scaled t-distribution to control  
 105 the degree of long distance dispersal in the model. Each individual's new location was drawn from the  
 106 scaled t-distribution with a mean equal to the individual's current location in space,  $x_i$  and  $\sigma = e^{d_i}$ . In  
 107 almost all that follows we treated dispersal as Gaussian ( $v \rightarrow \infty$ ), but we also briefly explore how relaxing  
 108 this assumption (and allowing long distance dispersal) alters the effectiveness of a genetic blackburn (see  
 109 below). Individuals that disperse beyond the bounds of the lattice (i.e.,  $x < 0$ ) are reflected back into  
 110 the bounded space. Offspring inherit their parent's location, and so commence dispersal from that point  
 111 (rather than the centre of the cell).

### 112 **3.3 Evolutionary dynamics**

113 We assumed that individuals choose a mate at random from within their cell. Each individual carries  
 114 with it 20 independently-segregating diploid loci that contribute to a quantitative trait, the individual's  
 115 dispersal phenotype,  $d_i$ . There are two alleles possible at each locus: one that is neutral with regard to  
 116 dispersal, and the other which increases the  $E(d_i)$  of the individual by an effect size,  $m$ , that is constant  
 117 across all loci.

118 Under a simple quantitative genetic model, the total phenotypic variance in a population,  $V_T = V_g + V_e$ ,  
 119 where  $V_g$  is the genetic variance (attributable to genetic differences between individuals), and  $V_e$  is the  
 120 environmental variance (attributable to the effect of environment on the expression of each individual's  
 121 genotype). In our model (which assumes no dominance or epistasis), the heritability of the trait,  $h^2$  is  
 122 the proportion of the total phenotypic variance that is attributable to genetic variance:  $h^2 = \frac{V_g}{V_T}$ . We  
 123 set the initial heritability to a value of 0.3 [a common value for real traits, see Roff, 1997] and the total  
 124 variance in the dispersal trait,  $V_T = (\log 1.5)^2$ . This variance value was chosen to give us computationally  
 125 tractable rates of population expansion. These values determined our value of  $V_g$ , and, in combination  
 126 with the number of loci, and our initial allele frequencies, determined our effect size,  $m$ .

127 At initialisation, allele frequencies at all loci were set to 0.5, and alleles were assigned to individuals

128 stochastically from this expectation using a binomial distribution. The total additive effect of all alleles  
 129 within an individual generate the individual's expected dispersal phenotype,  $E(d_i)$ , and the variance in  
 130  $E(d_i)$  is the additive genetic variance,  $V_g$ . We wanted the mean dispersal phenotype to be independent  
 131 of  $V_g$  and achieved this by zero-centering the distribution of genotype values and then adding a value,  $\mu$   
 132 that determined the mean  $E(d_i)$ ,

$$E(d_i) = \sum_{L=1}^{20} \sum_{j=1}^2 (A_{i,j,L} m) - 20m + \mu$$

133 ,

134 where  $A$  denotes allelic values. Throughout, we set  $\mu = \log(2)$ .

135 The individual's realised dispersal phenotype is, however, subject to a random environmental effect,  
 136 drawn from a normal distribution, such that  $d_i \sim N(E(d_i), \sqrt{V_e})$ , where  $V_e$  is the environmental variance.  
 137 This environmental variance is determined at the initialisation of the simulation such that the initial  
 138 heritability ( $h^2$ ) of  $d_i$  is 0.3: i.e.,  $V_e = \frac{V_g(1-h^2)}{h^2}$ .  $V_e$  thereafter remains constant across space, time, and  
 139 individuals.

140 The trait value,  $d_i$  affects individual fitness at carrying capacity through a trade-off with  $w_i$  such that  
 141  $w_i = e^{-ke^{d_i}}$ , where  $k$  is a constant (set to 0.2 throughout). Thus individuals that tend to disperse further  
 142 have reduced fitness at high population density.

### 143 3.4 Simulated scenarios

144 In all that follows we have set the following demographic parameters as constant:  $R_{max} = 5$ ;  $N^* = 200$ .

#### 145 3.4.1 The evolution of dispersal during spread

146 To obtain estimates of dispersal phenotypes that evolve within the range core and at the expanding  
 147 edge, we simulated the spread of this population over 50 generations. These phenotypes (as well as  
 148 basic information such as distance spread) were recorded over 20 replicates. We took the thousand  
 149 most spatially-advanced individuals and the thousand least spatially-advanced individuals from each  
 150 simulation, and pooled these individuals across replicates to estimate resultant gene frequencies on both  
 151 the expanding front and in the core of the population. We also used these samples to examine the  
 152 resultant differences in dispersal kernel between front and core.

### 153 3.4.2 The effectiveness of barriers to core vs frontal genotypes

154 Once we had estimates of gene frequency from core vs frontal populations, we used these two sets of  
155 frequencies to examine how the effectiveness of a barrier varies between core and frontal populations. To  
156 do this, we set up a population in 30 contiguous cells and allowed this population to spread towards a  
157 barrier set to be 5 units distant. Individuals dispersing into the barrier region were “killed”, but individ-  
158 uals dispersing past the barrier region were allowed to survive. To ensure that gene frequencies remained  
159 approximately constant over time, we removed the dispersal-fitness trade-off (i.e., we set  $k = 0$ ), and,  
160 rather than have individual-level inheritance, drew allelic values for new individuals from the population-  
161 level gene frequencies already established. We ran each of these populations over 50 generations, and  
162 recorded in which generation (if any) the barrier was breached. We defined “breached” as the existence  
163 of  $> 5$  individuals on the far side of the barrier. We ran these simulation tests for both sets of genotypes  
164 (frontal vs core), across barriers of size 1 to 50 units, with 20 replicate simulations per genotype/barrier  
165 combination.

### 166 3.4.3 The effect of rapid evolution once the barrier is reached

167 Here we simply compared the results of the above simulations with identical simulations in which the  
168 dispersal-fitness trade-off was present and in which inheritance was set back to individual-level (as de-  
169 scribed in Results).

### 170 3.4.4 The effect of a genetic backburn

171 In this scenario, we examined three barrier widths (10, 15 and 20 units) such that the barrier could be  
172 crossed by invasion front phenotypes, but not as easily by the low-dispersal phenotypes that characterise  
173 the range core (see section 4, Fig. 3). We then simulated an invasion under identical conditions to those  
174 described in section 3.4.1 above (that is, individual based heritability, and a trade-off in place). After  
175 50 generations of spread, we established our barrier 10 units from the invasion front, and introduced  
176 individuals with range core gene frequencies (at 20% of carrying capacity) to the uncolonised region  
177 abutting the barrier (between the barrier and the oncoming invasion). We then followed the population  
178 for a further 50 generations to determine whether the barrier was breached. We replicated this scenario  
179 20 times for each barrier width, and calculated the proportion of scenarios in which the population was  
180 halted at the barrier.



Several key parameters likely have a bearing on the effect of the genetic backburn. These parameters include the spatial extent across which we introduce new individuals, and the strength of the trade-off between fitness and dispersal. To assess variation in these parameters, we varied the spatial extent of the introduction, and the strength of the trade-off. We ran simulations spanning a range of extents from 0 (i.e., no backburn) to 10 units out from the barrier, and across a range of trade-off strengths ranging through  $k = 0$  (no trade-off) through  $k = 0.1$  (a weak trade-off), and  $k = 0.2$  (the default trade-off strength). Finally, long-distance dispersal can have powerful effects on invasions: causing them to accelerate, and making them harder to stop at landscape barriers. We briefly investigated how long distance dispersal affects the genetic backburn by running simulations at default levels for the trade-off ( $k = 0.2$ ), but with the kernel's shape parameter,  $v$ , varying through  $v = \{10, 7, 5\}$  which corresponds to excess kurtosis values of 1, 2, and 3 respectively.

## 4 Results

### 4.1 Evolution during spread

Over 50 generations of spread, dispersal values consistently evolved upwards on the expanding range edge (see, for example, Fig. 1). If we compare individuals closest to the expanding front with individuals from the population core, then it is clear that the dispersal kernels of the two populations have diverged during spread (Fig. 2).

### 4.2 The effectiveness of barriers to core vs frontal genotypes

Given our observation that dispersal kernels are different between frontal and core populations, we would expect the two populations to differ in their response to a barrier. Figure 3A shows this effect: the dispersal kernel of core populations is more readily stopped by small barriers.

### 4.3 The effect of rapid evolution once the barrier is reached

If, however, we don't force the kernels to be constant through time, but allow rapid evolution of core vs frontal dispersal kernels as the invasion front encounters the barrier, we see a marked reduction in the tendency for the barrier to be breached. Comparing Figure 3 panels A and B, we can see that when

206 evolution is allowed (panel B), the tendency for the barrier to be breached is markedly lower than when  
207 evolution is excluded (panel A).

#### 208 4.4 The effect of a genetic backburn

209 We now examine the effect of a genetic backburn on barrier strength. Figure 4 shows a single realisation of  
210 this scenario: a realisation in which the barrier remains intact, and invasion front phenotypes remain rare  
211 to non-existent immediately adjacent to the barrier. Figure 5 shows that, under particular conditions,  
212 translocating individuals from the core of the range to the nearside of the barrier can markedly improve  
213 the capacity of the barrier to halt the further spread of the population. In one scenario (the 15-unit  
214 barrier,  $k = 0.2$ ), the genetic backburn increased the strength of the barrier from a 20% chance of success  
215 to an 80% chance. As we would expect from intuition, the extent of the genetic backburn (i.e., the extent  
216 across which translocations are made back from the barrier) affects the result: larger backburns have a  
217 larger effect on barrier strength, but this improvement in barrier strength rapidly asymptotes. The other  
218 clear result from this set of simulations is that the trade-off between dispersal and fitness also has a strong  
219 effect on the efficacy of a genetic backburn. The backburn becomes less effective as the trade-off between  
220 dispersal and fitness is weakened (Fig. 5). Finally, our results show that long distance dispersal has a  
221 powerful influence on barrier strength (supplementary material). When long distance dispersal is allowed,  
222 all barriers become less effective, and this decrease in effectiveness is closely related to the degree of long  
223 distance dispersal: as the dispersal kernel becomes more kurtotic, barriers decrease in their effectiveness.  
224 Long distance dispersal also appears to mute the effect of a genetic backburn: although barriers still tend  
225 to become more effective under a backburn scenario, the degree of improvement in barrier strength is  
226 much more modest when long-distance dispersal is a major feature (supplementary material).

## 227 5 Discussion

228 Using a spatially-explicit individual-based model, we have shown that (i) dispersal ability can evolve  
229 upwards at expanding range margins, such that invasion-front individuals are more capable of overcoming  
230 landscape barriers than are individuals from the population core; (ii) landscape barriers that are capable  
231 of initially halting spread are rendered more effective by rapid evolution; and (iii) a genetic backburn  
232 — translocating individuals from the population core in advance of a landscape barrier — can further

233 increase the barrier’s effectiveness. In all cases these are proof of concept results; clearly, there is an  
234 infinite parameter space that could be explored. Our aim is to highlight the powerful role that rapid  
235 evolution might play in both helping and, with careful management, hindering, the spread of an invasive  
236 population.

237 Our first result — that invasion leads to the evolution of increased dispersal ability on the invasion  
238 front — is already well established. Numerous empirical and theoretical results concur that dispersal  
239 rates should and, in fact do, rapidly increase on invasion fronts [reviewed in Phillips et al., 2010b]. These  
240 evolutionary increases in dispersal can happen rapidly — on time scales of relevance to management —  
241 and can lead to invasions that accelerate over time [Perkins, 2012, Travis and Dytham, 2002, Phillips  
242 et al., 2008]. Thus, rapid evolution tends to make it more difficult to manage invasive species as an  
243 invasion progresses.

244 As well as causing invasions to accelerate, rapid evolution also potentially makes invasions more  
245 difficult to stop. As has been pointed out previously [Travis et al., 2010], dispersal barriers will become  
246 less effective as invasion front populations evolve increased dispersal. Although we do not include the  
247 possibility in our model, the increased growth rates that evolve in vanguard populations [Phillips, 2009]  
248 also may render them more capable of establishing on the far side of a barrier: they grow quickly from  
249 small numbers and so are less susceptible to stochastic extinction. Again, rapid evolution appears to act  
250 against managers’ efforts to limit the spread and impact of an invader.

251 Rapid evolution may not, however, be the management nightmare it currently appears to be. Instead,  
252 the rapid evolution elicited by dispersal barriers can aid managers to halt the spread of invaders. To  
253 begin with, it is clear that when an invasion front encounters a barrier, evolutionary forces conspire to  
254 drive dispersal rates downwards. Again, this is not a new result [e.g., Kubisch et al., 2010, Phillips, 2012],  
255 but our model suggests that the evolutionary shifts can happen surprisingly rapidly and can have a large  
256 impact on the effectiveness of a barrier. Our model, using a realistic value of heritability, showed that  
257 rapid evolution causes a spread barrier to be much more effective than consideration of frontal dispersal  
258 phenotypes alone would suggest (cf panels A and B, Fig. 3). While we would predict that dispersal should  
259 evolve downwards following the attainment of a barrier, the large impact on the probability of barrier  
260 breach seems surprising. If, however, we remember that the first phenotypes to encounter the barrier (and  
261 so die in it) will be the most dispersive phenotypes, it becomes clear that the most dispersive individuals  
262 in the population will have been suffering substantially lower fitness for a number of generations before

263 densities abutting the barrier become high. Many of the most dispersive genotypes therefore will have  
264 been weeded out of the frontal population before the population abutting the barrier reaches high density.  
265 Thus, landscape barriers and control efforts that effectively acts as barriers [such as efforts to contain the  
266 spread of Gypsy Moth: Sharov and Liebhold, 1998a] act to lower dispersal rates and so make barriers  
267 more effective over time.

268 Our simulations suggest that assessing barrier strength against invasion front phenotypes will be a  
269 conservative assessment indeed. For example, models of the effectiveness of an arid barrier in stopping  
270 further spread by invasive cane toads in Australia (using invasion front dispersal phenotypes but without  
271 allowing for rapid evolution) estimated a less than 10% probability of toads breaching a 100 km-wide  
272 barrier [Fig. 6 in Tingley et al., 2013]. Our model, showing that barriers are rendered more effective by  
273 rapid evolution, implies an even more encouraging scenario. Although we do not model the case of toads  
274 explicitly, the inclusion of evolutionary processes into the planning of that barrier may substantially  
275 modify the predicted benefits of a given management strategy, and in doing, may change the relative  
276 priority of alternative strategies.

277 While rapid evolution will, without any effort from a manager, render a barrier more effective over  
278 time, we also investigate a more active scenario, in which a manager translocates low-dispersal individuals  
279 from the range core to the barrier region. This "genetic backburn" strategy ensures that invasion front  
280 phenotypes do not reach the barrier. Instead, they encounter the biotic barrier of fitter, less dispersive  
281 genotypes. Our simulations suggest that this strategy can, in the right circumstances, dramatically  
282 improve the strength of a barrier. In situations where a barrier may not be quite wide enough to stop  
283 an invasion front, a barrier can, nonetheless, be rendered effective by asking it to stop the less dispersive  
284 individuals from the range core instead.

285 Our results also demonstrate that the effectiveness of a genetic backburn will depend on the degree  
286 to which invasion front phenotypes are less fit when placed in competition with phenotypes from the  
287 range core. While our model suggests that the genetic backburn can improve barrier strength even in  
288 the absence of a trade-off between fitness and dispersal, this improvement is more modest than scenarios  
289 in which a strong trade-off exists. Although the evolution of increased dispersal during invasion appears  
290 to be a robust expectation in nature, it is less clear that this necessitates a trade-off with fitness at high  
291 density (as instituted in our model). Certainly, dispersal-competition trade-offs occur in nature [e.g.,  
292 Jakobsson and Eriksson, 2003, Cadotte et al., 2006], though they are not ubiquitous [e.g., Limberger and

Wickham, 2011]. If, however, we accept that increased dispersal comes at some cost, then paying that cost from competitive ability is the optimal strategy on an invasion front where conspecific density (and hence competition) is low. Given sufficient time, then, an optimal strategy on the invasion front would be to reallocate resources from competitive ability and towards dispersal [Burton et al., 2010]. How often, and indeed whether, this optimal strategy emerges on invasion fronts [where stochastic forces often lead to non-optimal phenotypes dominating: Peischl et al., 2015, 2013, Phillips, 2015], is yet to be clarified with empirical studies.

In our simulations, the effectiveness of a landscape barrier was very sensitive to the degree of long-distance dispersal. This is not a surprising result: if long distance dispersal is allowed, then landscape barriers will always be more prone to failure. Our results also suggest, however, that the improvement offered by a genetic backburn is greatly diminished under long-distance dispersal scenarios. The genetic backburn never appears to reduce a barrier’s effectiveness, but the improvement in barrier strength can become negligible under long-distance dispersal (supplementary material). This result likely arises because under long distance dispersal as implemented here the barrier can be breached even by range core phenotypes. Thus introducing those phenotypes to the nearside of the barrier does not ensure the barrier is protected from phenotypes that can cross it. The extent to which this is a problem in the real world will depend on the true shape of the dispersal kernel for the species of interest. The degree of long distance dispersal determines how ‘fat’ the kernel’s tail is. Because long-distance dispersal is rarely observed, robustly quantifying the shape of the tail of dispersal kernels is notoriously difficult [e.g., Clark, 1998]. Mechanistic considerations of dispersal, however, suggest that, at large distances, dispersal kernels will usually be exponentially bounded [Petrovskii and Morozov, 2009]. That is, kernels may be fat-tailed to some distance before morphing to have rapidly-decaying tails, shaped like the tails of a Gaussian distribution [Petrovskii and Morozov, 2009]. The t-distribution (implemented here) does not have this property, and so likely overstates the reality of long distance dispersal at large distances. Our results, then, should be seen as confirmation that long distance dispersal is important, but as with all of the above, system-specific models would need to be developed to understand the sensitivity of specific systems.

With regard to specific applications, the use of genetic backburn as a management tool would likely make many managers nervous. Managers have to deal with public perception, and explaining to the public the value of introducing an invasive species ahead of its current invasion front would be challenging.

323 In none of our simulations, however, did implementation of a genetic backburn decrease a barrier's  
 324 effectiveness. Thus the manager probably does not risk making a barrier less permeable. If the barrier  
 325 fails despite the backburn, the risk is that the species may have reached the far side of the barrier a little  
 326 earlier than it otherwise would have. This risk is offset against the possibility of permanently excluding  
 327 the invasive species from the same area. Nonetheless, implementation of a genetic backburn would require  
 328 careful consideration of the specific system at hand. In order of priority we would suggest:

- 329 1. A careful consideration of the dispersal capacity of the species in the core of its range. If the species  
 330 exhibits capacity for very long distance dispersal, then any barrier may be ineffective.
- 331 2. Demonstration that dispersal shows evolved differences between range core and range front.
- 332 3. Ideally, demonstration that range core phenotypes outcompete invasion front phenotypes.

333 These latter two pieces of evidence may be assessed by translocating range core individuals to a  
 334 recently colonised area and monitoring the invasion (or failure) of range core phenotypes at the intro-  
 335 duction locality, compared with a control location. We note that in our motivating example – that of  
 336 the cane toad's invasion of northern Australia – these pieces of evidence are largely in hand. Cane toads  
 337 disperse via the physical movement of adults, and so likely have an exponentially-bounded kernel [e.g.,  
 338 Schwarzkopf and Alford, 2002]; cane toads have evolved to become substantially more dispersive on their  
 339 invasion front [Phillips et al., 2008, 2010a]; and recent evidence suggests that there are fitness costs to  
 340 having done so [Brown et al., 2007, Hudson et al., 2015]. Although a field demonstration of the competi-  
 341 tive superiority of range core phenotypes has not been undertaken, there are strong indications from the  
 342 decay of dispersal rate behind the invasion front that range core phenotypes outcompete those on the  
 343 invasion front [Lindström et al., 2013, Perkins et al., in review].

344 More generally, the suite of evolutionary forces that fashion highly-dispersive individuals at the in-  
 345 vasion front is wider than those applying in a spatially equilibrial population. At an expanding range  
 346 edge, processes such as mutation surfing [Klopfstein et al., 2006, Travis et al., 2007], enhanced kin com-  
 347 petition [Kubisch et al., 2013], spatial sorting [Shine et al., 2011], and enhanced spatial selection [Perkins  
 348 et al., 2013] come into play. Mutation surfing can undermine fitness directly [Peischl et al., 2015, 2013,  
 349 Phillips, 2015], while spatial sorting, enhanced spatial selection, and kin competition work to fashion a  
 350 phenotype that is adept at rapid dispersal, even if that dispersal enforces compromises in traits that  
 351 enhance fitness in equilibrium populations [Burton et al., 2010]. By translocating range-core individuals

352 to the range-edge, we eliminate the evolutionary conditions of the invasion front (the density gradient  
353 that drives spatial sorting as well as the high relatedness driving kin-competition effects), and bring  
354 invasion front phenotypes into direct conflict with fitter range-core phenotypes. Our model shows that  
355 doing this can make barriers to spread substantially more effective. This genetic backburn strategy is  
356 a new example of how “targeted gene flow” might be profitably used to achieve conservation outcomes  
357 [Aitken and Whitlock, 2013, Kelly and Phillips, 2016]. Currently such strategies are being investigated  
358 primarily in the context of mitigating the impact of climate change on biodiversity [Aitken and Whitlock,  
359 2013, Hoffmann and Sgrò, 2011], but our results hint that the careful movement of heritable variation  
360 may have much broader application.

## 361 **6 Data accessibility**

362 All the code used to execute the model and run simulations is available online at <https://github.com/benflips/GBB>.

## 363 **7 Competing interests**

364 We have no competing interests to declare.

## 365 **8 Author contributions**

366 The original idea was conceived jointly. Phillips carried out model development and initial coding; Tingley  
367 cross-checked code; Phillips conducted all simulations; all authors contributed to drafting the manuscript.

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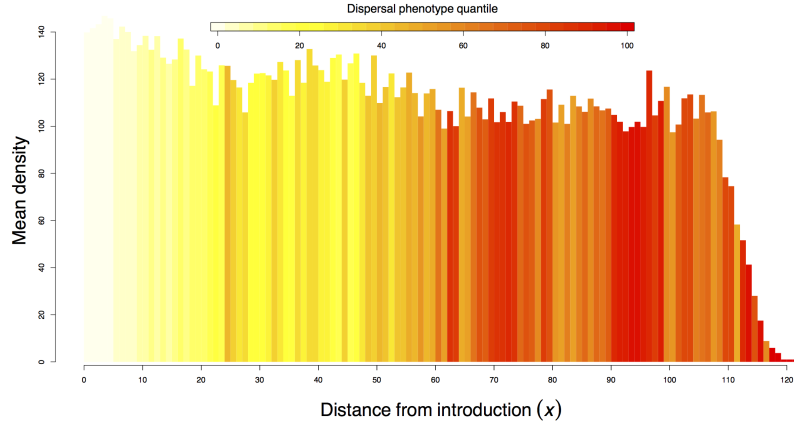


Figure 1: A single realisation of spread after 50 generations. The height of the bars represents density, and the colour represents the quantile of the dispersal phenotype,  $(d_i)$ .

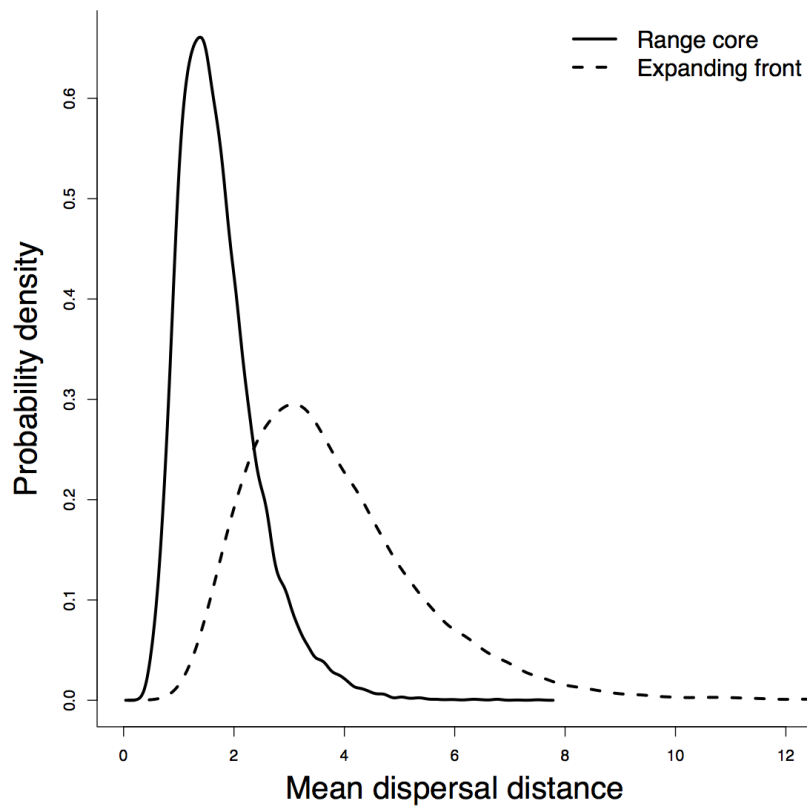


Figure 2: Dispersal kernels evolving in the core and at the leading edge of the range after 50 generations of spread. The y-axis, probability density, can be thought of as a scaled frequency value.

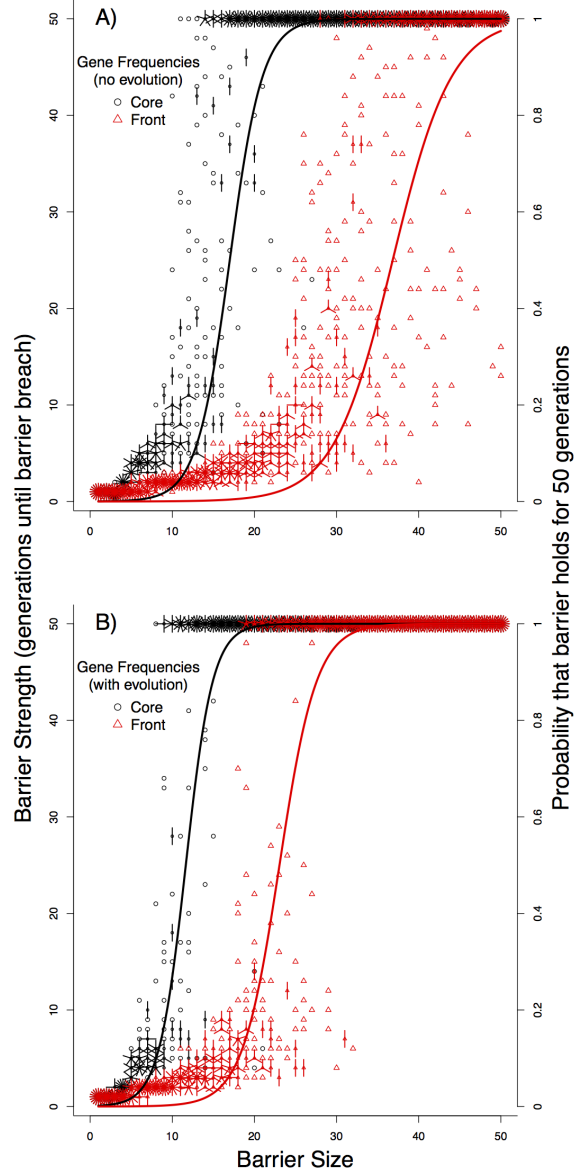


Figure 3: The capacity to breach a dispersal barrier varies between core and frontal populations, and is eroded by rapid evolution. Panel A shows the situation in which gene frequencies remain constant over time (i.e., there is no evolution). Panel B shows the situation in which gene frequencies evolve in response to encountering the barrier. Each point is a replicate simulation, (with overlapping points denoted by "petals" around each point) recording the breach time for that simulation. The lines represent logistic curves fitted to each dataset (modelling the probability of barrier breach within 50 generations). Each simulation ran over 50 generations, so this was the maximum number of generations a barrier could be observed to be effective.

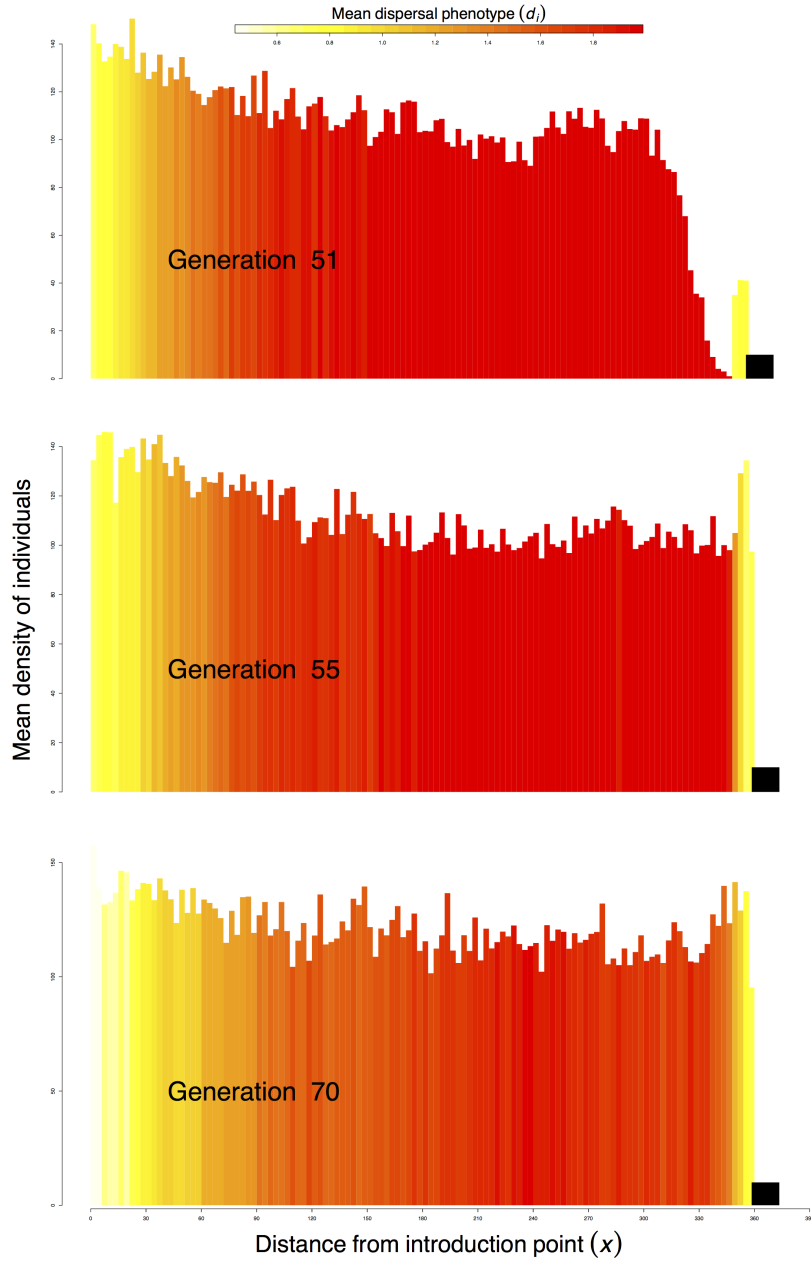


Figure 4: One realisation of a genetic backburn scenario. Following 50 generations of spread, invasion front phenotypes are highly dispersive (indicated by heat colour scale) and at 50 generations, we relocate individuals from the range core (far left) to between the oncoming population and the barrier (indicated by black rectangle at the far right). Figure panels show dynamics over the next twenty generations: the barrier remains intact and less dispersive (range-core) phenotypes invade back into the frontal population. Highly dispersive phenotypes remain rare to non-existent in the immediate vicinity of the barrier.



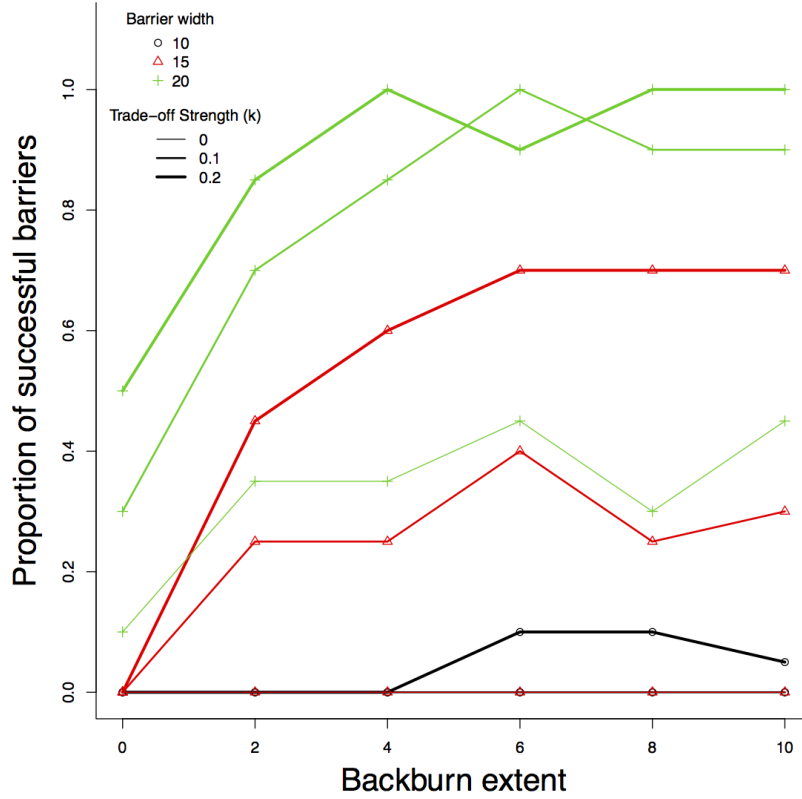


Figure 5: The strength of a barrier can be influenced by a genetic backburn. Here we show three barrier sizes (widths = 10, 15 and 20 units), and the proportion (of 20 simulations) in which, over 50 generations, the barrier stops the invasion. In all cases, the strength of the barrier was improved by a genetic backburn: translocating individuals from the range core to the near side of the barrier. This improvement in barrier strength was, however, dependent on the strength of the trade-off between dispersal and competitive ability. As this trade-off became weaker, the improvement gained by the genetic backburn also diminished.