



HAL
open science

Migration pulsedness alters patterns of allele fixation and local adaptation in a mainland-island model

Flora Aubree, Baptiste Lac, Ludovic Mailleret, Vincent Calcagno

► **To cite this version:**

Flora Aubree, Baptiste Lac, Ludovic Mailleret, Vincent Calcagno. Migration pulsedness alters patterns of allele fixation and local adaptation in a mainland-island model. *Evolution - International Journal of Organic Evolution*, 2023, 77 (3), pp.718-730. 10.1093/evolut/qpac067 . hal-04047090

HAL Id: hal-04047090

<https://hal.inrae.fr/hal-04047090>

Submitted on 27 Mar 2023

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



Distributed under a Creative Commons Attribution - NonCommercial - NoDerivatives 4.0 International License

1 **Running title:** How migration pulsedness impacts gene flow
2 **Type of article:** Original Articles
3 **Number of words in the main text:** ~6431
4 **Number of words in the abstract:** ~200
5 **Number of tables:** 0
6 **Number of figures:** 5
7 **Number of references:** 69

8 **Migration pulsedness alters patterns of allele**
9 **fixation and local adaptation in a mainland-island**
10 **model**

11 **Aubree, Flora¹ Lac, Baptiste¹ Mailleret, Ludovic^{1,2}**
12 **Calcagno, Vincent^{1,*}.**

13 *1. Université Côte d'Azur, INRAE, CNRS, ISA, 06900 Sophia Antipolis, France.*

14 *2. Université Côte d'Azur, INRIA, INRAE, CNRS, Sorbonne Université, BIOCORE,*
15 *Sophia Antipolis, France*

16 *Correspondence author. vincent.calcagno@inrae.fr

17 **Keywords:** temporally variable migration, effective migration rate, gene flow, migra-
18 tion load, selection

19 **Author contributions:** VC and FA designed the research. BL wrote a first version of
20 the codes and made preliminary analyses. FA and VC conducted the research and
21 wrote the manuscript with inputs from LM.

22 **Data accessibility statement:** All simulation files and Java, R and Mathematica scripts
23 are available on Zenodo (DOI 10.5281/zenodo.7133443).

12 Abstract

13 Geneflow across populations is a critical determinant of population genetic struc-
14 ture, divergence and local adaptation. While evolutionary theory typically envisions
15 geneflow as a continuous connection among populations, many processes make it
16 fluctuating and intermittent. We analyze a mainland-island model in which migra-
17 tion occurs as recurrent "pulses". We derive mathematical predictions regarding how
18 the level of migration pulsedness affects the effective migration rate, for neutral and
19 selected mainland alleles. We find that migration pulsedness can either decrease
20 or increase geneflow, depending on the selection regime. Migration increases gene-
21 flow for sufficiently (counter)selected alleles ($s < s_1$), but reduces it otherwise. We
22 provide a mathematical approximation of the threshold selection s_1 , which is veri-
23 fied in stochastic simulations. Migration pulsedness thus affects the fixation rate at
24 different loci in opposite ways, in a way that cannot be described as a change in
25 effective population size. We show that migration pulsedness would generally reduce
26 the level of local adaptation, and introduce an additional genetic load: the "pulsed-
27 ness load". Our results indicate that migration pulsedness can be detrimental to
28 the adaptation and persistence of small peripheral populations, with implications in
29 management and conservation. Our results highlight temporally variable migration
30 as an important process for evolutionary and population genetics.

31 **Keywords:** temporally variable migration, effective migration rate, geneflow, mi-
32 gration load, selection

33 Introduction

34 Geneflow between populations, as a major determinant of evolutionary dynamics,
35 has received considerable interest since almost a century. Depending on its intensity
36 and interactions with other evolutionary forces, geneflow has a range of contrasting
37 effects (e.g. Felsenstein, 1976; Lenormand, 2002; Bürger, 2014; Tigano & Friesen,
38 2016). In a focal population, it can enhance genetic diversity, prevent inbreeding, or
39 on the contrary hamper local adaptation (Gomulkiewicz *et al.*, 1999; Garant *et al.*,
40 2007; Bürger & Akerman, 2011). Across populations, it controls the spatial spread
41 of novel mutations, the maintenance of polymorphisms, the level of population di-
42 vergence and, eventually, the possibility of speciation (e.g. Maynard-Smith, 1966;
43 Johnson *et al.*, 2000; Yeaman & Otto, 2011; Mailund *et al.*, 2012; Rousset, 2013;
44 Feder *et al.*, 2019).

45 The consequences of geneflow have been studied in a range of spatial config-
46 urations, such as two interconnected populations (Maynard-Smith, 1966; Yeaman
47 & Otto, 2011), mainland-island systems (Johnson *et al.*, 2000; Bürger & Akerman,
48 2011) or metapopulations (Slatkin, 1981; Rousset, 2013; Feder *et al.*, 2019). How-
49 ever, temporal variability in the flows of propagules is rarely investigated in theor-
50 etical studies, and those usually consider the process of migration (here used in the
51 sense of dispersal) as constant. This is unfortunate, as migration is often governed
52 by time-fluctuating and potentially highly unsteady phenomena that would make it
53 temporally variable (Peniston *et al.*, 2019).

54 Causes of temporal variability in migration rate are almost endless. Among the
55 most frequently cited phenomena, a first category relates to environmental vari-
56 ations. Geographical barriers can change depending on land bridges (Morris-Pocock
57 *et al.*, 2016; Keyse *et al.*, 2018), sea levels fluctuations (Hewitt, 2000) or habitat
58 fragmentation (Peacock & Smith, 1997). Dispersal can also be affected by variation
59 in oceanic and atmospheric currents (Renner, 2004; White *et al.*, 2010; Smith *et al.*,
60 2018; Benestan *et al.*, 2021; see also Catalano *et al.*, 2020 for a recent attempt to
61 quantify dispersal variability). Last, but not least, dispersal is also affected by ex-
62 treme meteorological or climatic events such as floods and storms (Reed *et al.*, 1988;
63 Boedeltje *et al.*, 2004) that can cause rafting events (Garden *et al.*, 2014; Carlton
64 *et al.*, 2017). These extreme phenomena are bound to become more prevalent with
65 climate change (Masson-Delmotte *et al.*, 2018). A second category includes vari-
66 ations caused by species life-history traits, such as mast seeding, ballooning (Bishop,
67 1990) or various forms of group or clump dispersal (Soubeyrand *et al.*, 2015). And
68 finally, a third category encompasses variation related to dispersal by animal vectors
69 (Yamazaki *et al.*, 2016; Martin & Turner, 2018) or human activities (through bal-
70 last waters for instance, see Carlton & Cohen, 2003). All three categories are known
71 to result in variable dispersal rates, in the form of random fluctuations (Yamazaki
72 *et al.*, 2016), intermittent flows (Hewitt, 2000; Keyse *et al.*, 2018) or episodic bursts
73 of migration (Peacock & Smith, 1997; Reed *et al.*, 1988; Carlton & Cohen, 2003;
74 Reiners & Driese, 2004; Morris-Pocock *et al.*, 2016). The latter form is particularly
75 common and is often referred to as “pulsed migration” (Boedeltje *et al.*, 2004; Boba-
76 dilla & Santelices, 2005; Folinsbee & Brooks, 2007; Smith *et al.*, 2018; Martin &
77 Turner, 2018).

78 The relatively few existing theoretical studies suggest that migration variabil-
79 ity can have important population genetics and adaptive implications. Nagylaki
80 (1979), Latter & Sved (1981) and Whitlock (1992) have all used discrete-time is-
81 land models and considered neutral genetic variation only (infinite alleles model).
82 All three concur in predicting that temporal variability in migration rates should
83 decrease effective geneflow, and thus increase differentiation among populations (see
84 also Rousset, 2013). In a two-population model, Yamaguchi & Iwasa (2013), and
85 following papers, studied the fixation of incompatible mutations and the progress to
86 allopatric speciation, and found that speciation occurred faster with variable migra-
87 tion. Some studies have investigated non-neutral cases in spatially heterogeneous
88 environments, in particular in the context of source-sink population systems. Gag-
89 giotti & Smouse (1996) found that spacing out migration events, while keeping the
90 mean number of migrants constant, decreases the level of genetic variation in the
91 sink population. Rice & Papadopoulos (2009) studied a mainland-island model and
92 suggested that neglecting migration stochasticity would generally lead to overestim-
93 ating the impacts of migration on adaptation. More recently, Peniston *et al.* (2019)
94 extended the results of Gaggiotti & Smouse (1996), investigating the impact of tem-
95 porally pulsed migration on the level of local adaptation in the sink population.
96 They found that spacing out migration events (with a constant mean number of
97 migrants) can either hamper or facilitate adaptation in a harsh sink environment,
98 depending on genetic scenarios. In a different context, Matias *et al.* (2013) studied
99 specific diversity in a metacommunity model, and found that with randomly fluc-
100 tuating migration rates, larger mean dispersal values were needed to produce the
101 same local species richness. Overall, these studies seemingly converge on a negative
102 impact of dispersal variability on effective migration rate. However they remain few
103 and limited in their scope. Most of them underline the need for more attention
104 being given to the consequences of migration variability (Peniston *et al.*, 2019).

105 The present study aims at providing a more comprehensive appraisal of the con-
106 sequences of temporal variability in migration, considering both neutral, beneficial
107 and deleterious alleles, under various forms of selection and levels of dominance.
108 We will consider a mainland-island system where a small island population receives
109 migrants from a large mainland population (Wright, 1931; Felsenstein, 1976; Bürger,
110 2014). In the island population, we will model the dynamics of allele frequencies
111 in continuous time, at one locus, in diploid individuals. We will focus on the case
112 of “pulsed” migration patterns, i.e. geneflow from the mainland occurs as bursts of
113 migration of variable size and frequency (e.g. Rice & Papadopoulos, 2009; Peniston
114 *et al.*, 2019). We explore the range of migration “pulsedness” from continuous migra-
115 tion (independent migration of individuals) to very pulsed migration (rare migration
116 of groups of individuals).

117 Mathematical predictions for how the level of migration pulsedness should affect
118 the effective migration rate are derived under a low-migration limit. These predic-
119 tions are validated in stochastic simulations with large migration rates and logistic
120 population dynamics on the island. We show that for neutral alleles, migration
121 pulsedness reduces the effective migration rate, thus decreasing the rate of allele
122 fixation, which is an accordance with earlier theoretical results. However, under
123 general scenarios of selection, this conclusion can change quantitatively, and even

124 reverse. We find that if the selection coefficient falls below some negative threshold
125 value s_1 , of which we provide an analytical expression, the qualitative impact of mi-
126 gration pulsedness switches to positive, so that the effective migration rate increases
127 for those deleterious alleles. Moreover, for sufficiently recessive deleterious alleles,
128 migration pulsedness may have a non monotonous effect on the effective migration
129 rate. The interplay of selection, dominance and drift is thus found to play an es-
130 sential role in determining the impact of migration variability on effective migration
131 rate.

132 Our results show that the effect of migration pulsedness is not uniform across
133 loci subject to different selection regimes and dominance levels. Over an entire
134 genome, migration pulsedness effectively homogenizes the fixation rates across loci,
135 in a way that cannot be simply described as a change in effective population size.
136 This has consequences for the dynamics of mean fitness and local adaptation, by
137 creating an additional migration load that we call the “pulsedness load”. Overall,
138 our results highlight migration variability as an important property to be taken into
139 account when studying geneflow and local adaptation. Specifically, pulsed migration
140 patterns could overall be detrimental to the local adaptation and persistence of small
141 peripheral populations.

142 Methods

143 MAINLAND-ISLAND MODEL

144 A large mainland population is connected to a small island population of finite
145 size N , and migrant individuals flow from the former into the latter. We consider
146 arbitrary loci at which some allele A is fixed in the mainland whereas a different
147 allele a was initially fixed in the island population (fig. 1). Individuals are diploid
148 (there are $2N$ genes at any locus) and mate randomly within the island population.
149 The three diploid genotypes AA , Aa and aa have fitness values $1 + s$, $1 + hs$ and
150 1 , with h the degree of dominance and s the selection coefficient, as usually defined
151 (Thurman & Barrett, 2016). Results directly extend to haploids if one considers
152 genic selection ($h = 0.5$) and takes the number of genes to be N . For simplicity,
153 mutation is neglected. The evolution of allele frequencies in the island population
154 is modelled in continuous time (overlapping generations, Moran, 1962).

155 MODELLING OF PULSED MIGRATION

156 Migration occurs as discrete migration events (pulses of migrants) corresponding to
157 the arrival of n individuals into the island population (see Yamaguchi & Iwasa, 2013,
158 for a similar description of migration). The overall intensity of migration is controlled
159 by the migration rate m , the mean number of migrants per unit of time. To keep this
160 mean number of migrants constant, we impose that the more individuals arrive per
161 migration event (the larger n), the less frequent migration events are (Peniston *et al.*,
162 2019). Specifically, the rate at which migration events occur is taken to be m/n . If
163 $n = 1$, individuals migrate independently, corresponding to the classical continuous
164 form of migration. When n exceeds one, migrants arrive more sporadically, but in

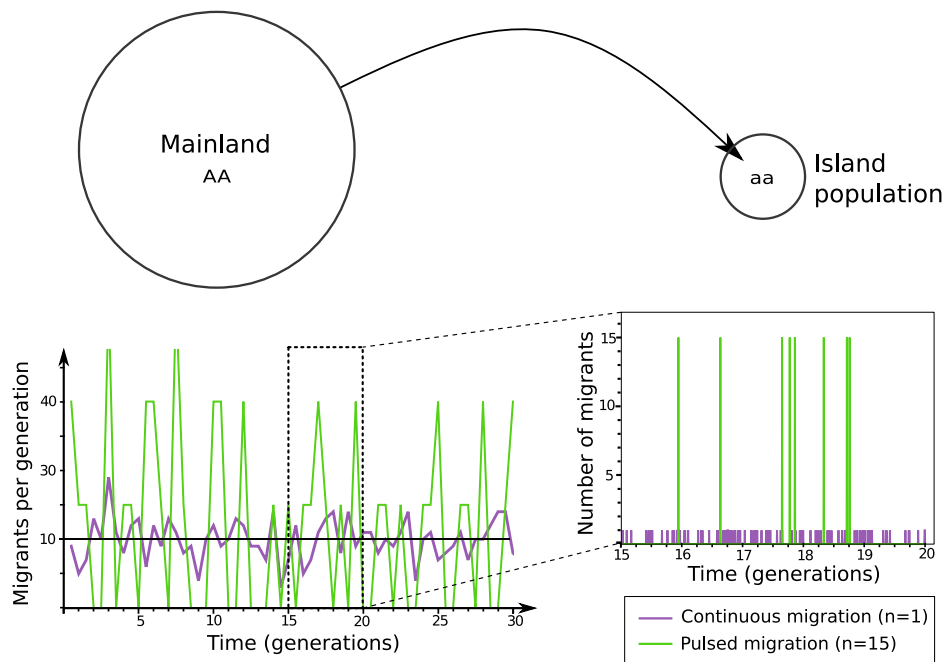


Fig. 1 Number of migrants as a function of time in the continuous case ($n = 1$, independent migration of individuals) and in a pulsed migration case ($n = 15$). The left panel averages the number of migrants over one generation. The right panel is a close-up over 5 generations, showing the arrival of individual migrants at each migration event. Illustrative parameters: $m = 10$, $T = 1$.

165 larger packs. We will thus increase n to make migration more and more pulsed, and
 166 evaluate how this affects predictions compared to continuous migration ($n = 1$).

167 Parameter n controls the degree of migration pulsedness. More precisely, the
 168 number of migration events that occur in one generation time (T) follows a Poisson
 169 distribution with mean mT/n . Therefore mT/n migration events occur on aver-
 170 age, with a variance equal to mT/n . It follows that the variance in the number of
 171 migrants ($n \times mT/n$) per generation is $n^2 mT/n = nmT$. Parameter n thus quanti-
 172 fies the degree of overdispersion (variance inflation) in the number of migrants per
 173 generation, relative to the continuous case. Migration events can range from very
 174 frequent and of small intensity, with minimal temporal variance in the number of
 175 migrants per generation (low n), to more infrequent and intense, with large temporal
 176 variance (large n ; Fig. 1).

177 METRICS OF INTEREST

178 In such a mainland-island model, fixation of mainland alleles in the island eventu-
 179 ally occurs with probability one, but we are interested in how long fixation takes,
 180 depending on the pulsedness level n . In other words, we seek to understand whether
 181 migration pulsedness, all else equal, is favorable or unfavorable to gene flow. We
 182 want to know this for mainland alleles with different selective values (h and s), and
 183 across different population sizes (N). To compare pulsed migration with continuous

184 migration, we will compute the effective migration rate m_e , defined as the migration
185 rate that would be required to produce the same time to allele fixation, all else equal,
186 under continuous migration (i.e. with $n = 1$). This definition is a variant of oth-
187 ers (Wang & Whitlock, 2003; Kobayashi *et al.*, 2008; Rice & Papadopoulos, 2009),
188 adapted to the study of migration pulsedness. The value of m_e is by definition m
189 when $n = 1$, and can deviate from m for larger n : if $m_e > m$, migration pulsedness
190 promotes geneflow, whereas if $m_e < m$, it reduces geneflow. Following Kobayashi
191 *et al.* (2008), we will further compute the geneflow factor, m_e/m : if the geneflow
192 factor is greater than one, migration pulsedness promotes geneflow, whereas if it is
193 less than one, it reduces geneflow.

194 Note that all comparisons are done for a given locus with a given selective regime,
195 not across different selective regimes. Obviously, beneficial alleles go to fixation
196 faster than neutral or maladaptive ones, for any kind of migration. Hence our
197 effective migration rate and geneflow factor are not "relative to a neutral allele", as
198 sometimes used, but rather "relative to continuous migration, for an identical allele"
199 (Kobayashi *et al.*, 2008).

200 STOCHASTIC SIMULATIONS

201 We simulated the island population as a continuous time birth-death-immigration
202 process, using a Gillespie algorithm. The population dynamics of the island popula-
203 tion followed a stochastic logistic model with carrying capacity K (Goel & Richter-
204 Dyn, 1974). Each diploid individual has some basal death rate d , birth rate $b = d$,
205 and density dependence acts through the death rate: total death rate is $d\frac{N^2}{K}$ while
206 total birth rate is bN . Selection occurs at reproduction (fertility selection): repro-
207 ducing individuals are picked at random with odds proportional to their selective
208 values. For computational efficiency, we used an optimized Gillespie algorithm (see
209 Supplementary Information; Section 1 for a full description of the simulation al-
210 gorithm).

211 The island is initially fixed for allele a and at carrying capacity ($N = K$). At the
212 beginning of a simulation, it starts receiving AA migrants from the mainland, under
213 the stochastic migration process described above (Fig. 1). The simulation ends when
214 allele A gets fixed in the island. We conducted 10,000 replicates per parameter set
215 to capture the stochasticity in migration times, population size fluctuations, and
216 genetic drift. Effective migration rates were determined from the observed time it
217 takes for the mainland allele to get fixed in the island, using a pre-computed abacus
218 (S.I.; Section 1.3).

219 MATHEMATICAL APPROXIMATION

220 To allow analytical progress, we make the standard assumption that population reg-
221 ulation is fast, so that population size is effectively constant at N in the island. How-
222 ever, we cannot employ the equally standard method of diffusion approximations.
223 Indeed, the latter would require n to be smallish relative to N , *de facto* preventing
224 the description of pulsed migration patterns (see Kimura, 1962; Yamaguchi & Iwasa,
225 2013). We rather use an alternative approximation, assuming that migration events

226 are rare enough, so that genetic drift and selection result in allele fixation before the
227 next migration event occurs.

228 With these assumptions, at each migration event, n homozygous AA migrants
229 arrive into the island population containing either N aa individuals (if fixation
230 did not occur yet) or N AA individuals (if it did). Following every migration
231 event, population regulation quickly takes population size back to N . Until fixation
232 of the mainland allele, each migration event brings the latter in initial frequency
233 $f = \frac{2n}{2n+2N} = \frac{n}{N+n}$, and the outcome of the particular migration event is either
234 fixation (success), or disappearance (failure) (see also Yamaguchi & Iwasa, 2013).
235 The probability of success can be well approximated by the fixation probability of
236 an allele in initial frequency f in a closed population of size N_e , $u(f, N_e, s, h)$. Ex-
237 pressions of u are well-known (Kimura, 1962; Whitlock, 2003). For simplicity,
238 we will typically consider N_e is equal to N in computations, even though N_e could
239 often be smaller than population size, including in our simulations (owing to demo-
240 graphic fluctuations). To simplify notations, we'll write interchangeably $u(f)$ or
241 $u(f, N_e, s, h)$.

242 From this, we can derive the probability that the mainland allele has not yet
243 swamped the island after a given time, and the mean time before it does, from an
244 exponential distribution with rate $-u(f)m/n$ (S.I.; section 2.2).

245 PARAMETER VALUES

246 The island population should be sufficiently isolated, otherwise it will immediately
247 be swamped by the mainland and gene flow is a trivial issue. A famous rule-of-thumb
248 is the so-called “one migrant per generation rule” (Mills & Allendorf, 1996, see also
249 Blanquart *et al.*, 2013), stating that migration is strong if more than one migrant
250 enters the population every generation. Without loss of generality, we'll set the basal
251 death rate as $d = N$ in our simulations, so that the generation time (defined as the
252 average time for N deaths to occur, see Moran, 1962) is $T \approx 1$. Since the mean
253 number of migrants per generation is mT , this means that, roughly speaking, m
254 should not exceed 1. In turn, our mathematical approximation requires the average
255 time between migration events (n/m) to be much larger than the average time to
256 fixation after one event. Using classical theory (Kimura & Ohta, 1969; Whitlock,
257 2003; Otto & Whitlock, 2013), we can show that this always holds if $m \ll \frac{1}{4N}$.
258 This is a sufficient condition: the approximation should hold well even for larger
259 migration rates (S.I.; section 2.1).

260 We will consider four scenarios regarding the intensity of migration in simula-
261 tions: (i) *very low* migration ($m = 0.001$); (ii) *low* migration ($m = 0.1$); (iii) *medium*
262 migration ($m = 1$), and (iv) *strong* migration ($m = 10$). The first scenario will be
263 used to check that simulations do converge to our mathematical approximation. The
264 three other scenarios span the one migrant per generation rule by one order of mag-
265 nitude in both directions. The last scenario (strong migration) is the one illustrated
266 in fig. 1.

267 The level of migration pulsedness n will be systematically varied between 1 and
268 N , the latter being an extreme case such that migration pulses temporarily double
269 the local population size and take migrants at 50% frequency. Selection parameters
270 will also be systematically varied in plausible ranges: s between -0.1 and 0.1 (Martin

271 & Lenormand, 2015; Thurman & Barrett, 2016), and h between 0 and 1. The
272 population size N will be varied between 50 and 200 (Palstra & Ruzzante, 2008;
273 Peniston *et al.*, 2019). With such parameters, the average proportion of immigrant
274 individuals per generation ($m/(N + m)$) varies between 0.0005 and 0.2.

275 Results

276 MATHEMATICAL PREDICTIONS

277 *General criterion for the impact of migration pulsedness*

278 Under our mathematical approximation, the rate of fixation of a mainland allele is
279 the product of m/n and the probability of success $u\left(\frac{n}{N+n}\right)$ of a particular migration
280 event (see above), and the mean time to fixation is the inverse of this rate. By
281 definition of the effective migration rate, we must have $m_e u\left(\frac{1}{N+1}\right) = \frac{m}{n} u\left(\frac{n}{N+n}\right)$.
282 It follows that in order to have a geneflow factor greater than one ($m_e > m$, i.e.
283 migration pulsedness increases geneflow), we must have:

$$u\left(\frac{n}{N+n}\right) > nu\left(\frac{1}{N+1}\right) \quad (1)$$

284 This condition yields a simple graphical criterion to determine whether migration
285 pulsedness promotes or decreases geneflow, based on the shape of the u function
286 corresponding to any particular selective regime. It also provides an operational
287 way to derive quantitative mathematical predictions for specific types of genetic
288 variation, as we'll proceed to do now.

289 *Neutral variation*

290 At a locus where the mainland allele is neutral, we can apply criterion (1) using the
291 fixation probability of a neutral allele, which is well known to be its initial frequency:

$$u\left(\frac{n}{N+n}\right) = \frac{n}{N+n} \quad (2)$$

292 In that case, it is straightforward to see that criterion (1) is never met, for any n .
293 Note that in the above equation, N is the actual census population size, not N_e , even
294 if they differ. We thus predict that migration pulsedness should invariably decrease
295 the effective migration rate, i.e. decrease geneflow. The more pulsed migration is,
296 the larger the effect is.

297 *Selected variation*

298 In the general case of selection with dominance, an expression for function u is:

$$u(f) = \frac{\int_0^f G(x)dx}{\int_0^1 G(x)dx} \quad (3)$$

299 with $G(x) = \exp[2N_e s((2h - 1)x^2 - 2hx)]$ (Kimura, 1962).

300 Note that in the expression of $G(x)$, the effective population size N_e matters, if
301 different from the census population size N .

302 This expression allows much greater flexibility in the shape of the function. Func-
303 tion $u(f)$ is well known to be either strictly concave, strictly convex, or convex-
304 concave with an inflexion point, depending on s and h . A particular case is the
305 additive case (frequency-independent selection in haploids, or codominance in dip-
306 loids, i.e. $h = 1/2$), for which a simpler explicit solution exists (Kimura, 1962;
307 Maruyama, 1970; Whitlock, 2003).

308 Mathematical predictions based on numerical analyses for all s and h values are
309 presented in fig. 2. Even though eq. (3) is much less mathematically tractable, we
310 can still make some analytical progress. Criterion (1) can be viewed as a compar-
311 ison to linearity for function $u[f(n)]$ (S.I.; section 2.3): if initially (for low levels of
312 pulsedness, i.e. n close to 1), the function is concave, then the criterion will not be
313 met. If on the contrary it is convex, then the criterion will be met. We can there-
314 fore determine whether making migration pulsed increases or decreases gene flow by
315 studying the curvature of $u[f(n)]$ at $n = 1$. Since f is a concave function of n , we
316 know that $u[f(n)]$ will be more prone to being concave than $u(f)$.

317 Differentiating $u[f(n)]$ twice at $n = 1$, using eq. (3) and $f = n/(N + n)$, we
318 find after some calculations (S.I.; section 2.3) that migration pulsedness increases
319 the effective migration rate (promotes gene flow) if:

$$s < s_1 = -\frac{(N + 1)^2}{2N_e N(hN + 1 - h)} \quad (4)$$

320 This threshold selection coefficient is always negative. In the special case of
321 additivity ($h = 1/2$), if population size is large and no different from the effective
322 population size, we obtain the approximation $s_1 \approx -1/N$. This becomes $s_1 \approx$
323 $-1/2N$ for fully dominant alleles, whereas for fully recessive alleles, we get the quite
324 extreme value $s_1 \approx -1/2$. These results are in close agreement with the numerics
325 and with stochastic simulations under a very low migration scenario (fig. 2).

326 We remark that the values of n at which an initially positive effect of pulsedness
327 would revert to negative are usually unrealistically large (thick green curves in sub-
328 panels of fig. 2; see also S.I.; section 2.6). Therefore, eq. (4) in practice means that
329 for sufficiently deleterious alleles ($s < s_1$), migration pulsedness increases the effect-
330 ive migration rate, instead of reducing it as it does for neutral alleles. It reduces
331 gene flow, as for neutral alleles, for slightly deleterious alleles ($s_1 < s < 0$), and for
332 all beneficial alleles ($s > 0$).

333 The condition $s < s_1$ is sufficient, but there still is the possibility that function
334 $u(n)$ is first convex, then switches to concave to such an extent that criterion (1) is
335 met for some larger n value, even though it was not for small values. In such circum-
336 stances, the consequences of migration pulsedness further depend on the value of n :
337 it would generally decrease the effective migration rate, except for some intermedi-
338 ate range of n values, for which it would on the contrary increase it. This situation
339 requires function $u(f)$ to have a pronounced sigmoidal shape, which is known to
340 occur only for recessive deleterious alleles.

341 Accordingly, as can be seen in fig. 2, for sufficiently recessive alleles, increasing s
342 above s_1 may not immediately turn the effects of migration pulsedness from positive

343 to negative, but rather there can be an intermediate range (s_1, s_2) of values for
344 which migration pulsedness has mixed effects depending on its intensity. One can
345 determine the level of dominance at which this bifurcation occurs (h_c), by remarking
346 it is the point ($h, s_1(h)$) at which the second inflexion point of function $u(n)$ collides
347 with the first one (located at $n = 1$). In other words, it is the point at which function
348 $u(n)$ has a null third derivative at $n = 1$ while verifying the condition $s = s_1$ (S.I.;
349 section 2.4). After some calculations, we get the following condition:

$$h < h_c = \frac{N - 1}{3N - 1} \quad (5)$$

350 Note that this depends on the census population only, not on the effective pop-
351 ulation size. If population size is large, we get the approximation $h \approx 1/3$, in close
352 agreement with the numerics (fig. 2).

353 We remark that the minimal level of pulsedness required to get an increased
354 effective migration rate is typically quite large (40–100; thin green curves in the
355 subpanels of fig. 2). Therefore, in practice, this situation is essentially similar to
356 that of neutral alleles (migration pulsedness reduces the effective migration rate).

357 To summarize, migration pulsedness should increase the effective migration rate
358 for sufficiently maladapted and not-too-recessive alleles ($s < s_1$), whereas it should
359 reduce the effective migration rate for slightly maladapted alleles, neutral alleles and
360 beneficial alleles.

361 CONFRONTING PREDICTIONS WITH STOCHASTIC SIMULATIONS

362 The above mathematical predictions were well verified in stochastic simulations in
363 which the overall rate of migration m was allowed to take larger values (fig. 3). To
364 facilitate comparisons across parameter sets, we follow the usual practice of plotting
365 results in terms of $N_e s$ rather than s , to reflect the effective strength of selection
366 relative to drift (Wright, 1931; Lande, 1994).

367 Qualitatively, mathematical predictions hold almost perfectly in the low and
368 medium migration scenarios. In other words, migration pulsedness could either
369 increase or decrease the effective migration rate, depending on the type of selective
370 regime, in the direction predicted. In the strong migration scenario, predictions
371 also held except that the space of selective regimes for which migration pulsedness
372 increases the effective migration rate was shifted to lower s values (i.e. s_1 gets more
373 negative).

374 Results did not depend importantly on population size, provided one scales n
375 with N . This is again consistent with mathematical predictions: the left-hand side
376 of eq. 1 is unchanged if $N_e s$ and n/N (and thus f) are both kept constant (see
377 eq. 3). The right-hand side changes very little: its relative variation with N is of
378 order less than $1/N^2$ (see S.I.; section 2.7). Therefore, mathematical predictions are
379 almost invariant for given values of $N_e s$ and n/N .

380 In quantitative terms, the impact of migration pulsedness on the effective mi-
381 gration rate (the geneflow factor) was very close to the one predicted by our math-
382 ematical approximation in all cases, except for deleterious alleles under the strong
383 migration scenario (fig. 3). In the latter case, the observed increase in geneflow could
384 be much smaller than the one predicted. In the most extreme example, a predicted

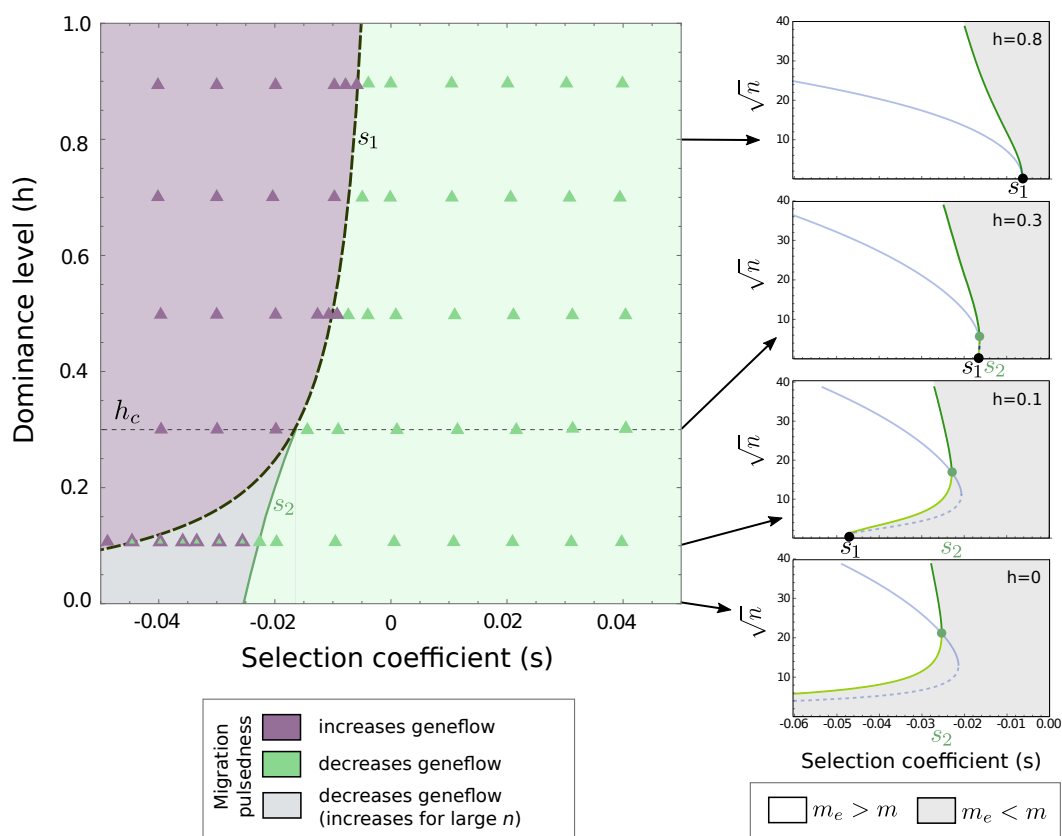


Fig. 2 Mathematical predictions. For all selection coefficient s and dominance h combinations, the plane is partitioned according to the impact of migration pulsedness on effective migration rate (geneflow). Predictions were computed numerically (from eq. (1) and (3)). Triangles show the observed result in stochastic simulations under the very low migration scenario ($m = 0.001$), to check consistency with mathematical analysis. The dashed curve is the mathematical expression for s_1 (eq. (4)). Subpanels are cross-sections of the main panel at four levels of dominance ($h = 0.8, 0.3, 0.1$ and 0), showing n values that increase (white) or decrease (gray) the effective migration rate. Note the square-root y-scale. The blue curves show the n values that maximize (solid) or minimize (dotted) geneflow. Other parameters: $N = N_e = 100$.

385 six-fold increase in effective migration rate turned out to be slightly more than a
 386 two-fold increase with $m = 10$ (fig. 3). Overall, large migration rates (i.e. beyond
 387 the one-migrant-per-generation rule) shift predictions towards a decrease in effective
 388 migration rate: more negative s values are required to obtain the same impact as
 389 for low migration rates. They otherwise have little qualitative impact. Remark that
 390 these quantitative differences are most important for large pulsedness levels (n);
 391 results for low n values, and thus the value of s_1 , are in much closer agreement with
 392 mathematical predictions (fig. 3). Similar conclusions hold for other values of the
 393 dominance level h (see S.I.; fig. S1).

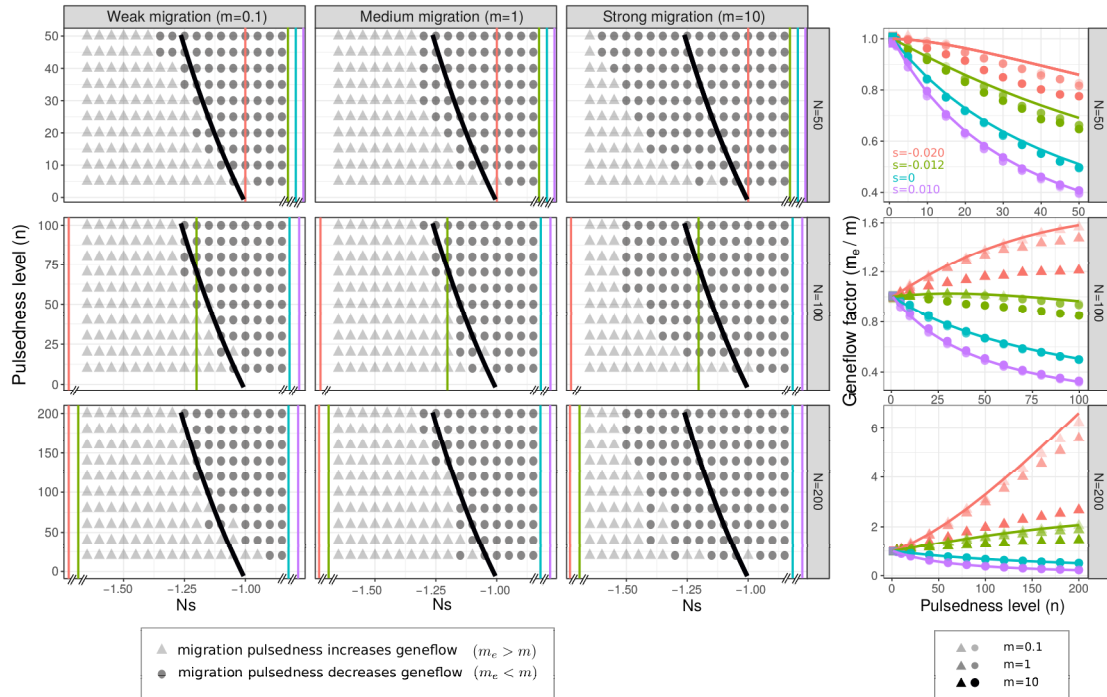


Fig. 3 Results of stochastic simulations. *Left:* Effect of migration pulsedness on effective migration rate, for n between 1 and N , and for a range of Ns values. The solid black lines represent the mathematical prediction, obtained numerically from eq. (1) and (3). Results are provided for the tree scenarios of migration intensity (columns), and for three population sizes (rows). *Right:* Geneflow factor (m_e/m) as a function of n , for different selection coefficients s and for the three migration scenarios. The s values used are also positioned in the left panels as vertical lines. Symbols are the values obtained in stochastic simulations, and solid lines are mathematical predictions, obtained as before. Other parameters: $h = 0.5$.

394 GENOME-WIDE CONSEQUENCES: MEAN FITNESS AND THE PULSEDNESS LOAD

395 The above results have established that migration pulsedness should affect different
 396 loci in different ways, depending on their selective regime. Overall, it will promote
 397 geneflow (promote fixation) of deleterious or maladapted alleles, especially if not too-
 398 recessive, whereas it will reduce geneflow (slowdown fixation) of slightly deleterious,
 399 neutral and beneficial alleles, especially recessive ones.

400 As pulsedness promotes the fixation of maladapted alleles and opposes the fixa-
 401 tion of beneficial alleles, its overall effect is to homogenize geneflow (fixation rates)
 402 across loci with different selective regimes in a genome. Such a homogenizing effect
 403 could at first be regarded as similar to a decrease in effective population size: demo-
 404 graphic fluctuations or other sources of variability that decrease N_e would reduce
 405 the strength of selection and also homogenize fixation rates across different loci.

406 However, simply decreasing population size, in a model with continuous migra-
 407 tion, cannot reproduce the patterns observed with migration pulsedness (fig. 4).
 408 Even if adjusting potentially unknown parameters (m or the time since onset of
 409 migration), in order to match exactly the fixation rate of neutral alleles, a reduction

410 in N_e can either fit the fixation patterns observed for maladapted alleles, or those of
 411 beneficial alleles, but never fits both (fig. 4). In other words, the change in N_e that
 412 is required to mimic the consequences of migration pulsedness is not identical across
 413 loci differing in their selection regimes. Therefore, the consequences of migration
 414 pulsedness cannot be interpreted as some reduction in effective population size.

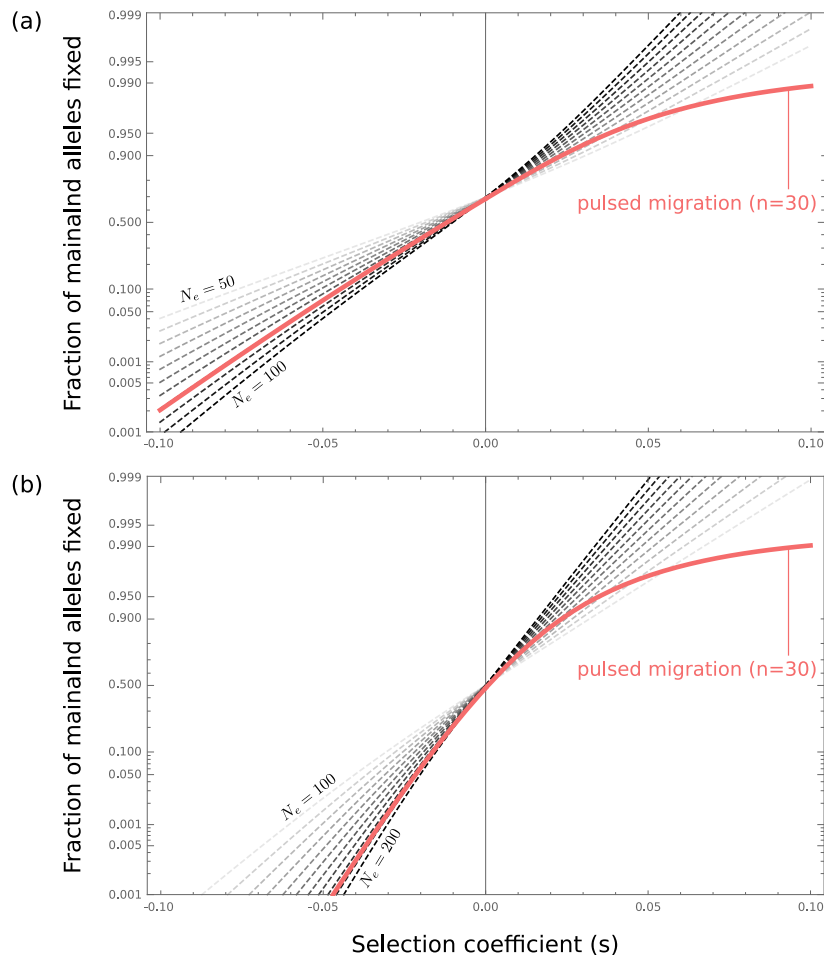


Fig. 4 Probability of fixation of different alleles varying in their selection coefficient, under pulsed migration ($n = 30$; red curve), and under continuous migration with different effective population sizes (gray dashed curves). (a) $N = 100$ and (b) $N = 200$. The time since the onset of migration (t) was set at an intermediate value so that about 50% of neutral alleles have gone to fixation in the pulsed case: $t = 150$. To minimize the difference between pulsed and continuous curves, the time was adjusted for the continuous cases, so as to match the neutral fixation rate: (a) $t = 120$ and (b) $t = 140$. If using the actual times, the discrepancies between the two migration scenarios are even more pronounced. The curves are from the mathematical model with parameters $m = 1$ and $h = 1/2$. Note the logistic scale for the y-axis.

415 One can extrapolate the fitness consequences of migration pulsedness over the

416 entire genome, assuming a large number of independent loci, each with its particular
417 selection regime. The distribution of h and s values over loci would vary greatly
418 across organisms and contexts, but for illustration we can assume some plausible
419 distribution of fitness effects (DFE) with $h = 1/2$, such as the one proposed in
420 (Martin & Lenormand, 2015, S.I.; section 3.1). This DFE, illustrated in fig. 5a,
421 features an excess of deleterious (maladapted) alleles, representative of situations
422 where migration from the mainland mostly decreases fitness on the island. At any
423 time, the mean fitness \bar{w} in the island population is the product of the fitness
424 contributions of all loci, depending on their fixation status (see S.I.; section 3.2).

425 Following the onset of immigration, the island population mean fitness evolves
426 through time, asymptotically settling at its equilibrium value, when all mainland
427 alleles have fixed, and thus controlled by the DFE. Under recurring migration, re-
428 gardless the level of pulsedness, beneficial (adapted) alleles tend to go to fixation
429 first, followed by neutral alleles, and ultimately deleterious (maladapted) alleles.
430 These dynamics of allele fixation are shown in fig. 5b, with the FDE of fixing
431 alleles, at two different times, is shown as inserts. Introducing migration pulsedness
432 slows down the fixation of beneficial and neutral variation, which decreases mean
433 fitness in the island population, compared to what it should be under continuous
434 migration. Conversely, it hastens the fixation of deleterious (maladapted) alleles
435 with $s < s_1$, which is also detrimental to mean fitness (see fig. 5a). As a result, mi-
436 gration pulsedness causes mean fitness to be lower than under continuous migration
437 (fig. 5c), and we call this fitness deficit (i.e. additional genetic load) the "pulsedness
438 load". If most fitness variation is driven by adaptation to local habitat conditions,
439 the pulsedness load translates into lower local adaptation. It must be stressed that
440 if one simply extrapolated the fact that migration pulsedness decreases the effective
441 migration rate, as is observed for neutral alleles, to the entire genome, one would
442 reach the opposite prediction: migration pulsedness would *increase* mean fitness at
443 any time.

444 As shown in fig. 5d, the pulsedness load can readily cause a 10% increase in the
445 genetic load, compared to what is under continuous migration. Interestingly, slightly
446 deleterious alleles with selective values in the range $(s_1, 0)$ have reduced fixation rates
447 under pulsed migration, which has the opposite effect of boosting mean fitness: this
448 contributes negatively to the pulsedness load. As a consequence, the pulsedness load
449 might transiently decline (and possibly become negative), at the time when most
450 alleles getting to fixation fall into this category (fig. 5d). Generically, the dynamics
451 of the pulsedness load thus follows three consecutive phases over time, driven by
452 the fixation of different categories of alleles (fig. 5a). The relative importance and
453 timing of the three phases of course depends on the exact DFE.

454 Discussion

455 Temporal variability in migration is probably pervasive in nature. Plants, fungi,
456 birds, mollusks and other marine invertebrates are most represented in the literat-
457 ure about variable dispersal, with many other taxa presumably concerned, includ-
458 ing hominoids (Folinsbee & Brooks, 2007). A common form of migration temporal
459 variability is migration pulsedness, i.e. the positively correlated migration of indi-

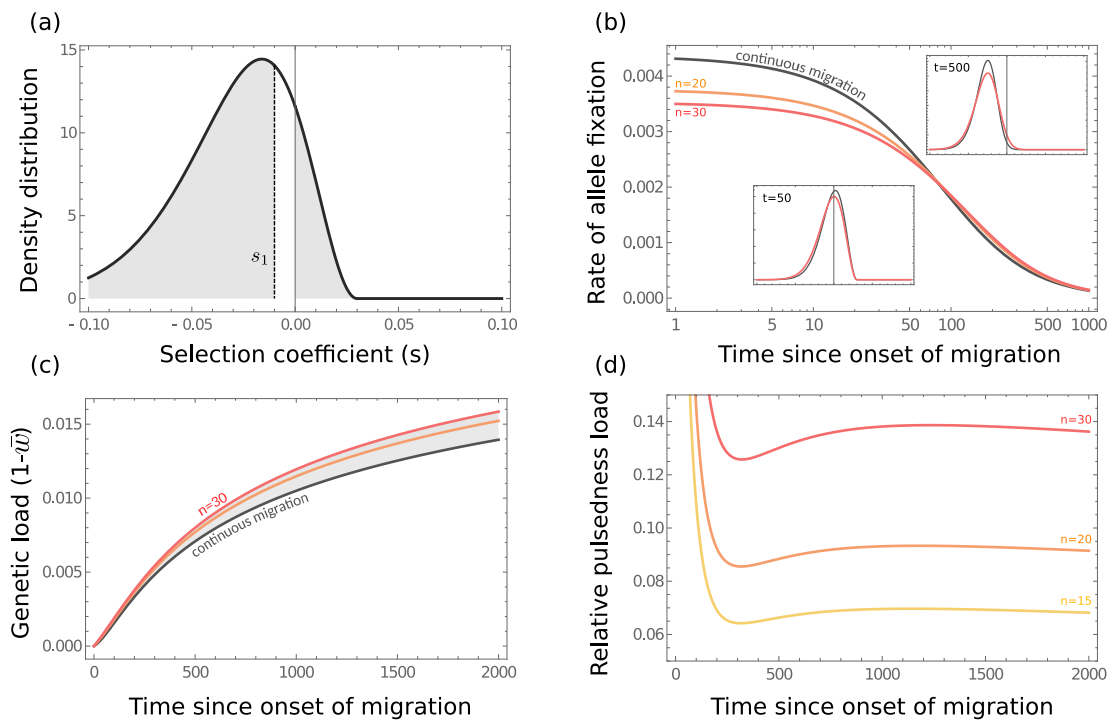


Fig. 5 The pulsedness load. (a) An illustrative distribution of fitness effects (DFE) for mainland alleles across loci. Most alleles are slightly deleterious or neutral, some are beneficial (Martin & Lenormand, 2015). Three important classes of alleles are distinguished: $s < s_1$, $s_1 < s < 0$ and $s > 0$. (b) The instantaneous rate of fixation (fixation flux) of mainland alleles as a function of the time since onset of migration, for continuous migration and two levels of migration pulsedness. The two inserts show the DFE of alleles that are currently going to fixation, at times 50 and 500, for continuous migration and pulsed ($n = 20$ and $n = 30$) migration. (c) Genetic load ($1 - \bar{w}$) as a function of time since onset of migration, for continuous and pulsed migration. The excess of genetic load caused by migration pulsedness (the pulsed load) is highlighted as the shaded area. (d) Value of the pulsedness load (relative to the genetic load with continuous migration) as a function of time since onset of migration, for three different levels of migration pulsedness. Results are from the mathematical model with parameters $h = 1/2$, $m = 1$ and $N = 100$.

460 viduals, producing pulses of migration interspersed among periods of low or absent
 461 migration. However, classical evolutionary theory largely rests on the assumption of
 462 constant migration rates (e.g. Johnson *et al.*, 2000; Yeaman & Otto, 2011; Mailund
 463 *et al.*, 2012; Rousset, 2013; Peniston *et al.*, 2019). Considering the growing evidence
 464 for non-constant migration processes, it is important to gain a general theoretical
 465 understanding of their evolutionary consequences.

466 We here proposed a novel approach and derived mathematical predictions re-
 467 garding how the level of migration pulsedness should impact gene flow (the effective
 468 migration rate) at loci subject to an arbitrary selection regime. This is an import-

469 ant difference from earlier existing studies, that usually focused on neutral variation
470 alone, or on some specific forms of selection. Though we here addressed pulsed
471 migration patterns (Yamaguchi & Iwasa, 2013; Peniston *et al.*, 2019), we can reas-
472 onably expect our predictions to apply more broadly to other forms of variable mi-
473 gration patterns, intended as a temporal overdispersion in the number of migrants
474 per generation. The latter is quantified by parameter n in our model.

475 Our main finding is that the effect of migration pulsedness on geneflow depends
476 not only quantitatively, but also qualitatively, on the selective regime considered.
477 We find that migration pulsedness should decrease the effective migration rate (re-
478 duce geneflow) for alleles that are neutral or beneficial. However, for sufficiently
479 deleterious (maladapted) alleles, the effect can be opposite. We found that there
480 exists some threshold selection coefficient s_1 , of which we derived a mathematical
481 expression, below which migration pulsedness on the contrary increases the effective
482 migration rate (increases geneflow). The value of s_1 increases with the dominance
483 level h , so that an increase in geneflow with pulsedness is more likely for dominant
484 alleles, and much less likely for recessive alleles.

485 The effect of migration pulsedness is therefore not homogeneous across different
486 loci over the genome that experience different selection regimes. Migration pulsed-
487 ness increases geneflow, and thus increases the speed and probability of fixation, for
488 deleterious (maladapted) alleles, but it does the opposite for beneficial alleles. This
489 homogenizes fixation rates over the genome. Such a homogenization of fixation
490 rates across loci experiencing contrasted selection regimes could also be brought
491 about by a reduced effective population size, i.e. a greater importance of drift rel-
492 ative to selection. However, we showed that a simple change in effective population
493 size, retaining a continuous mode of migration, cannot adequately reproduce the
494 fixation patterns created by pulsed migration. The action of migration pulsedness
495 therefore leaves a signature that, at least in principle, could be distinguished from
496 other forms of variability that reduce effective population size (random population
497 fluctuations, random inter-individual fecundity variations, biased sex-ratios). The
498 signature is more subtle. It is more comparable to that of deterministic processes
499 such as directional changes of population size through time (Otto & Whitlock, 1997).
500 In particular, an increased fixation of deleterious alleles and concomitant decreased
501 fixation of beneficial alleles is analogous of what is predicted for shrinking popula-
502 tions (Otto & Whitlock, 1997). Our results also suggest that migration pulsedness
503 would generate a relative excess of dominant deleterious alleles, and a relative defi-
504 cit of recessive deleterious alleles (see fig. 2), which adds an extra dimension to its
505 genomic signature.

506 Another consequence of migration pulsedness is that, by promoting the fixation
507 of deleterious (maladapted) alleles, while slowing down the fixation of beneficial
508 (adaptive) alleles, it overall reduces mean fitness and the level of local adaptation.
509 We showed that migration pulsedness creates an additional genetic load, on top of
510 the migration load expected under a continuous migration of similar intensity. This
511 so-called "pulsedness load" can be non-negligible, and may easily represent a 10%
512 increase in the genetic load, or even more, for plausible distributions of fitness effects
513 and pulsedness values.

514 Our results generalize some existing results and are compatible with them. Most

515 importantly, our predictions for neutral alleles are consistent with the few earlier
516 population genetics studies, even though they used entirely different modelling ap-
517 proaches (Nagylaki, 1979; Latter & Sved, 1981; Whitlock, 1992; Rousset, 2013). We
518 predict, as they did, that temporal variation in migration rates always decreases the
519 effective migration rate for neutral alleles.

520 Even fewer studies have so far considered non-neutral variation. Peniston *et al.*
521 (2019) investigated the consequences of pulses of migration in a mainland island
522 model, like us, but they specifically considered source-sink situations: the island
523 population was initially not viable on its own, and had to adapt in the face of
524 maladaptive geneflow. This is quite a different scenario, as we here assumed that
525 the island population was viable, not a sink. Our results thus complement the latter
526 study, and we find, as they did, that migration pulsedness can importantly affect
527 the dynamics of local adaptation. Even though direct comparisons are difficult,
528 we remark that among all the scenarios they considered, for the one closest to
529 ours, Peniston *et al.* (2019) report that more pulsedness migration hampers local
530 adaptation, which is consistent with our predictions.

531 Yamaguchi & Iwasa (2013) and subsequent papers considered parapatric speci-
532 ation through the accumulation of neutral mutations between two isolated popula-
533 tions, until prezygotic isolation develops. They used a formalism similar to ours,
534 but were not specifically interested in migration variability. They did not vary the
535 latter, and for simplicity they kept the number of migrants per migration event
536 (n) at very low values. Their results thus cannot be compared to ours. However,
537 we can use our results to predict the consequences for the build-up of reproductive
538 isolation between isolated populations. Indeed, if we extend the range of selection
539 regimes we've considered to incompatible mutations causing reproductive isolation
540 (Dobzhanski-Muller – DM – mutations; Gavrilets (2004)), we can see those as the
541 limit where $s \rightarrow 0^-$, and $h \rightarrow \infty$ (strong overdominance). For instance, $s = -0.001$
542 and $h = 10000$ describes a mutation that cause a -0.1 fitness disadvantage, only
543 in heterozygotes. Extrapolating fig. (2) to those values, we would conclude that
544 migration pulsedness should slightly decrease geneflow for them. This may seem
545 paradoxical, as such mutations obviously benefit from arriving in higher frequency.
546 However, their probability to go to fixation by chance is almost zero, unless their
547 frequency reaches or exceeds 50%, a value that would require unrealistic large values
548 of n . We can tentatively posit that migration pulsedness should not much affect the
549 accumulation of DM incompatibilities, even though establishing this would require
550 a dedicated study.

551 Most of our predictions can be understood in terms of the trade-off between fewer
552 migration events (rate m/n), which reduces geneflow, and higher initial frequency
553 after an event ($n/(N+n)$), which promotes geneflow. Increasing the pulsedness level
554 n decreases the number of migration events in inverse proportion, but the initial
555 frequency at each event increases less than linearly ($n/(N+n)$). In general, and for
556 neutral alleles in particular, the net effect of increasing n is therefore detrimental
557 to geneflow. Intuitively, this is because concentrating the arrival of alleles in time
558 causes those mainland alleles to compete more among themselves, which does not
559 help their fixation over local alleles. It is only when the fact of arriving in larger
560 initial frequency is very beneficial to fixation that this effect can overcome that of

561 a decreased number of migration events. This is the case for sufficiently deleterious
562 alleles, whose chance of establishing is smallish from a low initial frequency, but
563 much better if in higher frequency, owing to genetic drift. We have shown that
564 sufficiently deleterious here means $s < s_1$. The beneficial effect of a larger initial
565 frequency is of course strengthened by positive frequency-dependent selection: this
566 is why the value of s_1 increases with the level of dominance h in a diploid context,
567 as it generates positive frequency-dependence at the allele level. An increase in
568 geneflow with migration pulsedness would similarly be promoted by other sources of
569 positive frequency-dependence, such as aposematic color signalling (Endler, 1988):
570 migration pulsedness could probably promote the successful migration of foreign
571 aposematic morphs.

572 Our mathematical predictions held very well in stochastic simulations for differ-
573 ent population sizes, and when the overall migration rate (m) was large. However, in
574 the latter case the possibility for migration pulsedness to increase geneflow is shifted
575 towards more deleterious alleles, i.e. requires more stringent conditions than at lower
576 migration rates. This can be understood intuitively in terms of mass-effects: with
577 large migration rates, the influx of mainland alleles is strong enough to increase their
578 frequency in the island on its own, regardless of the local selection-drift dynamics. In
579 other words, deleterious alleles "pile-up" over successive migration events, and this
580 can be an important driver of their eventual fixation. The latter effect is virtually
581 unaffected by migration pulsedness; it solely depends on the average rate m . As a
582 consequence, the positive impact of migration pulsedness that we have identified,
583 and explained just before, is in some sense diluted and gets relatively weaker. This
584 is not the case for beneficial alleles, which get fixed after a small number of migra-
585 tion events, so that the "piling-up" effect does not significantly contribute to their
586 fixation. Further mathematical investigations allowing for large migration would be
587 needed to validate this interpretation.

588 The finding that migration pulsedness all else equal favors the fixation of de-
589 leterious alleles and reduces local adaptation has direct implications for biological
590 conservation, population management and island biology. For instance, popula-
591 tion reinforcement programs usually takes the form of periodic releases of groups
592 of individuals, whose frequency and intensity must both be optimized. Our res-
593 ults suggest that rare introductions of relatively large numbers of individuals, i.e.
594 pulsed introduction patterns, would be more detrimental to population fitness and
595 local adaptation, relative to more continuous fluxes of individuals, possibly com-
596 promising population viability and persistence. Extending our reasoning to island
597 communities (Cowie & Holland, 2006), this also suggests that sporadic but poten-
598 tially intense bouts of immigration, as brought about by rafting and human-driven
599 invasions, could favour the establishment of relatively maladapted mainland species
600 within communities competing for similar resources.

601 Of course, these impacts would combine with other effects, in particular demo-
602 graphical effects. It has been shown for instance that in small populations subject
603 to Allee effects, pulsed migration patterns can be favourable to population estab-
604 lishment and persistence (Rajakaruna *et al.*, 2013; Bajeux *et al.*, 2019). Similarly,
605 Peniston *et al.* (2019) found that migration pulsedness could sometimes favour local
606 adaptation and persistence in populations that are demographic sinks (see also Gag-

607 giotti & Smouse, 1996). These examples underline how complex the consequences
608 of dispersal can be when demo-genetics effects are taken into account (Garant *et al.*,
609 2007). It would be interesting to extend our model considering a broader range
610 of demographic scenarios, and see how predictions are affected. For instance, the
611 decrease in mean fitness through time as mainland alleles fix in the island popula-
612 tion could have a negative impact on the local population. A decrease in N could
613 have various impacts on the probabilities of fixation. At the simplest level, it would
614 increase the n/N ratio, which would make the effects of pulsedness more and more
615 pronounced. It would also increase the importance of drift, i.e. decrease $N_e s$, which
616 would shift the value of s_1 . How exactly this would impact the overall dynamics of
617 the pulsedness load remains to be determined. At a more genetic level, we assumed
618 for simplicity that alleles at different loci were unlinked and behaved independently.
619 This would not always be the case, and it would be interesting to study the role
620 of migration pulsedness for pairs of linked mutations that are in strong linkage dis-
621 equilibrium among migrant individuals.

622 We conclude, along the lines of Peniston *et al.* (2019), by advocating for tests of
623 predictions on migration pulsedness in empirical systems with well-designed experi-
624 mental set-ups. We add that available data on migration flows, e.g. from monitoring
625 of oceanic rafting, individual tracking of dispersal routes, or marine and aerial cur-
626 rent models (Ser-Giacomi *et al.*, 2015; Lagomarsino Oneto *et al.*, 2020), combined
627 with genomics data, may also provide opportunities for testing such predictions
628 in the wild. Understanding the consequences of ecological variability is increas-
629 ingly important in the present context of climate and habitat changes, and the
630 spatio-temporal variability in migration patterns probably deserves to receive more
631 attention.

632 References

- 633 Bajeux, N., Grognard, F. & Mailleret, L. (2019) Influence of the components of propagule
634 pressure, Allee effects, and stochasticity on the time to establish introduced populations.
635 Journal of Theoretical Biology, **471**, 91 – 107.
- 636 Benestan, L., Fietz, K., Loiseau, N., Guerin, P.E., Trofimenko, E., Rühs, S., Schmidt, C.,
637 Rath, W., Biastoch, A., Pérez-Ruzafa, A., Baixauli, P., Forcada, A., Arcas, E., Lenfant,
638 P., Mallol, S., Goñi, R., Velez, L., Höppner, M., Kininmonth, S., Mouillot, D., Puebla,
639 O. & Manel, S. (2021) Restricted dispersal in a sea of gene flow. Proceedings of the
640 Royal Society B: Biological Sciences, **288**, 20210458.
- 641 Bishop, L. (1990) Meteorological Aspects of Spider Ballooning. Environmental
642 Entomology, **19**, 1381–1387.
- 643 Blanquart, F., Kaltz, O., Nuismer, S.L. & Gandon, S. (2013) A practical guide to measuring
644 local adaptation. Ecology Letters, **16**, 1195–1205.
- 645 Bobadilla, M. & Santelices, B. (2005) Variations in the dispersal curves of macroalgal
646 propagules from a source. Journal of Experimental Marine Biology and Ecology, **327**,
647 47–57.
- 648 Boedeltje, G., Bakker, J.P., Ten Brinke, A., Van Groenendael, J.M. & Soesbergen, M.
649 (2004) Dispersal phenology of hydrochorous plants in relation to discharge, seed release
650 time and buoyancy of seeds: the flood pulse concept supported. Journal of Ecology, **92**,
651 786–796.
- 652 Bürger, R. (2014) A survey of migration-selection models in population genetics. Discrete
653 & Continuous Dynamical Systems - B, **19**, 883–959.
- 654 Bürger, R. & Akerman, A. (2011) The effects of linkage and gene flow on local adaptation:
655 A two-locus continent–island model. Theoretical Population Biology, **80**, 272–288.
- 656 Carlton, J.T., Chapman, J.W., Geller, J.B., Miller, J.A., Carlton, D.A., McCuller,
657 M.I., Treneman, N.C., Steves, B.P. & Ruiz, G.M. (2017) Tsunami-Driven Rafting:
658 Transoceanic Species Dispersal and Implications for Marine Biogeography. Science, **357**,
659 1402–1406.
- 660 Carlton, J. & Cohen, A. (2003) Episodic Global Dispersal in Shallow Water Marine Organ-
661 isms: The Case History of the European Shore Crabs *Carcinus Maenas* and *C-Aestuarii*.
662 Journal of Biogeography, **30**, 1809–1820.
- 663 Catalano, K.A., Dedrick, A.G., Stuart, M.R., Puritz, J.B., Montes Jr., H.R. & Pinsky, M.L.
664 (2020) Quantifying dispersal variability among nearshore marine populations. Molecular
665 Ecology, **n/a**.
- 666 Cowie, R.H. & Holland, B.S. (2006) Dispersal is fundamental to biogeography and the
667 evolution of biodiversity on oceanic islands. Journal of Biogeography, **33**, 193–198.
- 668 Endler, J.A. (1988) Frequency-dependent predation, crypsis and aposematic coloration.
669 Philosophical Transactions of the Royal Society of London. B, Biological Sciences, **319**,
670 505–523.

- 671 Feder, A.F., Pennings, P.S., Hermisson, J. & Petrov, D.A. (2019) Evolutionary Dynamics in
672 Structured Populations Under Strong Population Genetic Forces. G3: Genes, Genomes,
673 Genetics, **9**, 3395–3407.
- 674 Felsenstein, J. (1976) The theoretical population genetics of variable selection and migra-
675 tion. Annual Review of Genetics, **10**, 253–280. PMID: 797310.
- 676 Folinsbee, K.E. & Brooks, D.R. (2007) Miocene hominoid biogeography: pulses of dispersal
677 and differentiation. Journal of Biogeography, **34**, 383–397.
- 678 Gaggiotti, O. & Smouse, P. (1996) Stochastic Migration and Maintenance of Genetic Vari-
679 ation in Sink Populations. American Naturalist, **147**, 919–945.
- 680 Garant, D., Forde, S. & Hendry, A. (2007) The multifarious effects of dispersal and gene
681 flow on contemporary adaptation. Functional Ecology, **21**, 434–443.
- 682 Garden, C., Currie, K., Fraser, C. & Waters, J. (2014) Rafting dispersal constrained by an
683 oceanographic boundary. Marine Ecology Progress Series, **501**, 297–302.
- 684 Gavrilets, S. (2004) Fitness landscapes and the origin of species (MPB-41). Princeton
685 University Press.
- 686 Goel, N.S. & Richter-Dyn, N. (1974) Stochastic models in biology. Academic Press., New
687 York, San Francisco, London.
- 688 Gomulkiewicz, R., Holt, R.D. & Barfield, M. (1999) The Effects of Density Dependence
689 and Immigration on Local Adaptation and Niche Evolution in a Black-Hole Sink Envir-
690 onment. Theoretical Population Biology, **55**, 283–296.
- 691 Hewitt, G. (2000) The Genetic Legacy of the Quaternary Ice Ages. Nature, **405**, 907–913.
- 692 Johnson, K.P., Adler, F.R. & Cherry, J.L. (2000) Genetic and phylogenetic consequences
693 of island biogeography. Evolution, **54**, 387–396.
- 694 Keyse, J., Treml, E.A., Huelsken, T., Barber, P.H., DeBoer, T., Kochzius, M., Nuryanto,
695 A., Gardner, J.P.A., Liu, L.L., Penny, S. & Riginos, C. (2018) Historical divergences
696 associated with intermittent land bridges overshadow isolation by larval dispersal in
697 co-distributed species of *Tridacna* giant clams. Journal of Biogeography, **45**, 848–858.
- 698 Kimura, M. (1962) On the probability of fixation of mutant genes in a population. Genetics,
699 **47**, 713–719.
- 700 Kimura, M. & Ohta, T. (1969) The average number of generations until fixation of a
701 mutant gene in a finite population. Genetics, **61**, 763–771.
- 702 Kobayashi, Y., Hammerstein, P. & Telschow, A. (2008) The neutral effective migration
703 rate in a mainland-island context. Theoretical Population Biology, **74**, 84 – 92.
- 704 Lagomarsino Oneto, D., Golan, J., Mazzino, A., Pringle, A. & Seminara, A. (2020) Timing
705 of fungal spore release dictates survival during atmospheric transport. Proceedings of
706 the National Academy of Sciences, **117**, 5134–5143.
- 707 Lande, R. (1994) Risk of population extinction from fixation of new deleterious mutations.
708 Evolution, **48**, 1460–1469.

- 709 Latter, B.D.H. & Sved, J.A. (1981) Migration and Mutation in Stochastic Models of Gene
710 Frequency Change. II. Stochastic Migration with a Finite Number of Islands. J. Math.
711 Biology, **13**, 95–104.
- 712 Lenormand, T. (2002) Gene Flow and the Limits to Natural Selection. Trends in Ecology
713 and Evolution, **17**, 183–189.
- 714 Mailund, T., Halager, A.E., Westergaard, M., Dutheil, J.Y., Munch, K., Andersen, L.N.,
715 Lunter, G., Prüfer, K., Scally, A., Hobolth, A. & Schierup, M.H. (2012) A New Isola-
716 tion with Migration Model along Complete Genomes Infers Very Different Divergence
717 Processes among Closely Related Great Ape Species. PLOS Genetics, **8**, 1–19.
- 718 Martin, C.H. & Turner, B.J. (2018) Long-distance dispersal over land by fishes: extremely
719 rare ecological events become probable over millennial timescales. Proceedings of the
720 Royal Society B: Biological Sciences, **285**, 20172436.
- 721 Martin, G. & Lenormand, T. (2015) The fitness effect of mutations across environments:
722 Fisher’s geometrical model with multiple optima. Evolution, **69**, 1433–1447.
- 723 Maruyama, T. (1970) Effective number of alleles in a subdivided population. Theoretical
724 Population Biology, **1**, 273–306.
- 725 Masson-Delmotte, V., Zhai, P., Pörtner, H.O., Roberts, D., Skea, J., Shukla, P., Pirani, A.,
726 Moufouma-Okia, W., Péan, C., Pidcock, R., Connors, S., Matthews, J., Chen, Y., Zhou,
727 X., Gomis, M., Lonnoy, E., Maycock, T., Tignor, M. & Waterfield, T. (2018) IPCC, 2018:
728 Global Warming of 1.5C. An IPCC Special Report on the impacts of global warming
729 of 1.5C above pre-industrial levels and related global greenhouse gas emission pathways,
730 in the context of strengthening the global response to the threat of climate change,
731 sustainable development, and efforts to eradicate poverty. Working Group I Technical
732 Support Unit, Bern, Switzerland.
- 733 Matias, M.G., Mouquet, N. & Chase, J.M. (2013) Dispersal stochasticity mediates species
734 richness in sourcesink metacommunities. OIKOS, **122**, 395–402.
- 735 Maynard-Smith, J. (1966) Sympatric Speciation. The American Naturalist, **100**, 637–650.
- 736 Mills, L.S. & Allendorf, F.W. (1996) The One-Migrant-per-Generation Rule in Conserva-
737 tion and Management. Conservation Biology, **10**, 1509–1518.
- 738 Moran, P.A.P. (1962) The statistical processes of evolutionary theory. Clarendon Press;
739 Oxford University Press.
- 740 Morris-Pocock, J.A., Anderson, D.J. & Friesen, V.L. (2016) Biogeographical barriers to
741 dispersal and rare gene flow shape population genetic structure in red-footed boobies
742 (*Sula sula*). Journal of Biogeography, **43**, 2125–2135.
- 743 Nagylaki, T. (1979) The Island Model with Stochastic Migration. Genetics, **91**, 163–176.
- 744 Otto, S.P. & Whitlock, M.C. (1997) The probability of fixation in populations of changing
745 size. Genetics, **146**, 723–733.
- 746 Otto, S.P. & Whitlock, M.C. (2013) Fixation probabilities and times. eLS. American
747 Cancer Society. ISBN 978-0-470-01590-2.

- 748 Palstra, F.P. & Ruzzante, D.E. (2008) Genetic estimates of contemporary effective popu-
749 lation size: what can they tell us about the importance of genetic stochasticity for wild
750 population persistence? Molecular Ecology, **17**, 3428–3447.
- 751 Peacock, M.M. & Smith, A.T. (1997) The effect of habitat fragmentation on dispersal
752 patterns, mating behavior, and genetic variation in a pika (*Ochotona princeps*) meta-
753 population. Oecologia, **112**, 524–533.
- 754 Peniston, J.H., Barfield, M. & Holt, R.D. (2019) Pulsed Immigration Events Can Facilitate
755 Adaptation to Harsh Sink Environments. The American Naturalist, **194**, 316–333.
- 756 Rajakaruna, H., Potapov, A. & Lewis, M. (2013) Impact of stochasticity
757 in immigration and reintroduction on colonizing and extirpating populations.
758 Theoretical Population Biology, **85**, 38–48.
- 759 Reed, D.C., Laur, D.R. & Ebeling, A.W. (1988) Variation in Algal Dispersal and Recruit-
760 ment: The Importance of Episodic Events. Ecological Monographs, **58**, 321–335.
- 761 Reiners, W.A. & Driese, K.L. (2004) Transport Processes in Nature: Propagation of
762 Ecological Influences Through Environmental Space, vol. 2. Cambridge University Press.
- 763 Renner, S. (2004) Plant Dispersal across the Tropical Atlantic by Wind and Sea Currents.
764 International Journal of Plant Sciences, **165**, S23–S33.
- 765 Rice, S.H. & Papadopoulos, A. (2009) Evolution with Stochastic Fitness and Stochastic
766 Migration. PLOS ONE, **4**, 1–12.
- 767 Rousset, F. (2013) Genetic structure and selection in subdivided populations (MPB-40),
768 vol. 40. Princeton University Press.
- 769 Ser-Giacomi, E., Rossi, V., López, C. & Hernández-García, E. (2015) Flow networks: A
770 characterization of geophysical fluid transport. Chaos: An Interdisciplinary Journal of
771 Nonlinear Science, **25**, 036404.
- 772 Slatkin, M. (1981) Fixation Probabilities and Fixation Times in a Subdivided Population.
773 Evolution, **35**, 477–488.
- 774 Smith, T.M., York, P.H., Broitman, B.R., Thiel, M., Hays, G.C., van Sebille, E., Putman,
775 N.F., Macreadie, P.I. & Sherman, C.D.H. (2018) Rare long-distance dispersal of a marine
776 angiosperm across the Pacific Ocean. Global Ecology and Biogeography, **27**, 487–496.
- 777 Soubeyrand, S., Sache, I., Hamelin, F. & Klein, E.K. (2015) Evolution of dispersal in
778 asexual populations: to be independent, clumped or grouped? Evolutionary Ecology,
779 **29**, 947–963.
- 780 Thurman, T.J. & Barrett, R.D.H. (2016) The genetic consequences of selection in natural
781 populations. Molecular Ecology, **25**, 1429–1448.
- 782 Tigano, A. & Friesen, V.L. (2016) Genomics of local adaptation with gene flow. Molecular
783 Ecology, **25**, 2144–2164.
- 784 Wang, J. & Whitlock, M.C. (2003) Estimating Effective Population Size and Migration
785 Rates From Genetic Samples Over Space and Time. Genetics, **163**, 429–446.

- 786 White, C., Selkoe, K.A., Watson, J., Siegel, D.A., Zacherl, D.C. & Toonen, R.J. (2010)
787 Ocean currents help explain population genetic structure. Proceedings of the Royal
788 Society B: Biological Sciences, **277**, 1685–1694.
- 789 Whitlock, M.C. (1992) Temporal fluctuations in demographic parameters and the genetic
790 variance among populations. Evolution, **46**, 608–615.
- 791 Whitlock, M.C. (2003) Fixation Probability and Time in Subdivided Populations. Genetics,
792 **164**, 767–779.
- 793 Wright, S. (1931) Evolution in Mendelian Populations. Genetics, **16**, 97–159.
- 794 Yamaguchi, R. & Iwasa, Y. (2013) First Passage Time to Allopatric Speciation. Interface
795 focus, **3**, 20130026.
- 796 Yamazaki, Y., Naoe, S., Masaki, T. & Isagi, Y. (2016) Temporal variations in seed dispersal
797 patterns of a bird-dispersed tree, *Swida controversa* (Cornaceae), in a temperate forest.
798 Ecological Research, **31**, 165–176.
- 799 Yeaman, S. & Otto, S.P. (2011) Establishment and Maintenance of Adaptive Genetic
800 Divergence under Migration, Selection, and Drift. Evolution, **65**, 2123–2129.