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Abstract

The genetic structure of populations is commonly used to infer connectivity among distant populations. Accounting for connectivity is essential in marine spatial planning and the proper design and management of Marine Protected Areas (MPAs), given that their effectiveness depends on the patterns of dispersal and colonisation between protected and non-protected areas. Here, we present a spatially explicit coupled metapopulation gene flow model that simulates the effect of demographic fluctuations on the allele frequencies of a set of populations. We show that in closed populations, regardless of population growth rate, the maintenance of genetic diversity at the initial saturating population density increases with species life expectancy as a result of density-dependent recruitment control. Correlatively, at low initial population density, the time at which a population begins to loose its genetic diversity is driven by recruitment success – the larger the recruitment success, the quicker the genetic drift. Different spatial structures of connectivity established for soft bottom benthic invertebrates in the Gulf of Lions (NW Mediterranean, France) lead to very different results in the spatial patterns of genetic structuration of the metapopulation, with quick genetic drift in sites where the local retention rate was larger than 2%. The effect of recruitment failure and the loss of key source populations on heterozygosity confirm that transient

demographic fluctuations have a major effect on the maintenance of genetic diversity in a metapopulation. This study highlights the role of intra-specific settlement limitations due to lack of space when the effective number of breeders approaches saturating capacity, causing a strong reduction of effective reproduction. The present model allows to: 1) disentangle the relative contribution of local demography and environmental connectivity in shaping seascape genetics, and 2) perform *in silico* evaluations of different scenarios for marine spatial planning.

Keywords: connectivity, metapopulation, seascape genetics, spatially explicit model, sedentary benthic invertebrates, Gulf of Lions.

Introduction

Connectivity strongly impacts the dynamics and persistence of marine populations because it influences key processes, from short-term demography to long-term evolution, including resistance to disturbances and environmental changes. Therefore, understanding the development and maintenance of connectivity patterns is essential in marine spatial planning and the proper design and management of Marine Protected Areas (MPAs) (Palumbi 2004; Botsford *et al.*, 2009). However, the quantitative study of connectivity and the identification of connectivity bottlenecks that could compromise the ability for populations to recover after disturbances are current scientific challenges (Pineda 2000; Burgess *et al.*, 2014).

For sedentary marine benthic species, understanding connectivity translates into understanding larval dispersal, given that it is during the pelagic larval stage that the exchange of individuals and genes among populations takes place (Pineda *et al.*, 2007; Cowen and Sponaugle 2009). Hence, to analyse the patterns of connectivity among populations and study, in more detail, the mechanisms that shape and maintain connectivity patterns through time, the dispersal of organisms should be taken into account (Hastings and Botsford 2006; Aiken and Navarrete 2011).

Different approaches have been undertaken to try to quantify dispersal and connectivity in marine ecosystems. Population genetics has been one of the most frequently used tools to provide direct and indirect measures of connectivity in several marine species (Hellberg *et al.*, 2002; Saenz-Agudelo *et al.*, 2009; Lowe and Allendorf 2010). The dispersal potential of the larvae produced by sedentary benthic species has usually correlated with gene flow and was even used as a predictor of population genetic structure (Siegel *et al.*, 2003).

Using estimates of Pelagic Larval Duration (PLD) as a proxy to measure realised dispersal distance, several studies showed that the correlation between PLD and genetic Isolation by Distance (IBD) was consistent with the expectation that migration was the key factor driving the genetic structuration of marine populations (Hellberg 1996; Gilg and Hilbish 2003). However, recent data set reviews (Riginos *et al.*, 2011; Selkoe and Toonen 2011; Faurby and Barber 2012) have now shown that the relationship between PLD and genetic structuration might be weaker than previously thought.

The influence of several processes (e.g. larval behaviour, availability of suitable habitat, and biotic interactions) can cause the patterns of population connectivity to be different from what would be expected from flow integration along PLD alone (Pineda 2000; Guizien *et al.*, 2006). Among these processes, settlement or recruitment regulation of dispersal alters gene flow (Pineda *et al.*, 2007), in addition to the local demography. This temporal and spatial variability in the strength of gene and demographic flow due to bottlenecks during larval settlement and recruitment implies that there is a set of interacting processes operating across various scales, which introduces complexity into the system. Therefore, inferring connectivity by only one disciplinary approach (either genetic or bio-physic) does not provide the necessary information to properly describe population persistence in a network of MPAs (Hastings and Harrison 1994; Marko and Hart 2011).

It is therefore necessary, in order to efficiently inform conservation management decisions, to use tools that integrate bio-physical models with information regarding local

demographic traits driving genetic linkages that result from larval exchange among populations (Werner *et al.*, 2007; Cowen and Sponaugle 2009). However, detecting the genetic signatures caused by the transient dynamics of demographic changes remains a challenge (Alcala *et al.*, 2013).

Although genetic models incorporating larval dispersal and demographic parameters have recently been developed to quantify connectivity among marine populations (Galindo *et al.*, 2006; Kool *et al.*, 2009; Munroe *et al.*, 2012), they have some limitations when attempting to understand connectivity patterns among non-equilibrium populations. The model Galindo *et al.* (2006) developed, for example, does not take into account demographic variability, as population sizes were kept constant over time. The approach Kool (2009) developed, although allowing changes in population size, does not account for overlapping generations but, more importantly, being an Individual-Based Model (IBM), population sizes were limited to 10 individuals. Recently, Munroe *et al.* (2012), presented a modelling case study for Eastern oysters in Delaware Bay (USA) in which they relaxed population size limitations and assigned each population its own demographic dynamics.

In an attempt to generalise Munroe *et al.'s* (2012) approach, we present a spatially explicit coupled demographic gene flow model that assesses the effect of demography variability on allele frequencies in a marine metapopulation of sedentary benthic species with realistic population densities. The seascape model was used to examine how: 1) demographic traits influence the development of genetic structure in closed populations over time, 2) different patterns of connectivity affect the loss or maintenance of genetic diversity in a metapopulation, 3) fluctuations in local population density help maintain genetic diversity over time and 4) local population stability impacts genetic diversity to provide relevant information for conservation decisions in the Gulf of Lions, France.

Methods

A seascape genetic model for benthic invertebrates with a pelagic larval phase

To simulate the transient demographic effect on local allele frequencies arising from environmental variability modulating connectivity through time, a seascape genetic model was developed. The seascape model relies on the forcing of a spatially explicit dynamical model of allele probability by a spatially explicit metapopulation model (Guizien *et al.*, 2014, adapted from Hastings and Botsford 2006) (Fig. 1). The combination of these models allows us to project the development of a population genetic structure through time, resulting from demography dynamics driven by local survival and larval transfer on a regional scale.

The spatially explicit metapopulation model simulates the population density $N_i(t)$ at each site *i* at any time *t* resulting from the balance of adult survival and input of new recruits resulting from reproduction of adults present in the previous time step. Therefore, N_i represents the adult census size per unit surface, and it is equal to the effective number of breeders per unit surface for the next reproductive event (as defined by Waples *et al*., 2013). It is important to note that such formulation implicitly hypothesized that age at first reproduction and the time lapse between reproductive events are both equal to the model time step, ensuring that effective number of breeders does not vary with population age structure. Larval transfer rate among sites is estimated from a set of realistic connectivity matrices obtained using the larval dispersal model (Guizien *et al.*, 2012), and recruitment success (inter-specific competition or predation) is limited by saturating population density (intra-specific competition) (Eq. 1 and 2) (Guizien *et al.*, 2014).

$$N_i(t+1) = min\left(G(t)\,N_i(t), N_{max}\right) \qquad (1)$$

$N_i(t+1)$ = Vector of spatial density of individuals at each site at time *t+1* [individual per m^2]

$N_i(t)$ = Vector of spatial density of individuals at each site at time *t* [individual per m^2]

N_{max}= Saturating population density based on space limitation [individual per m^2]

with the transfer matrix G(t) defined by:

$$G_{ji}(t) = a_i C_{ji}(t) b_j + s_{jj} \delta_{ji} \qquad (2)$$

a_i= Propagule production rate at site *i* [larvae per adult]

$C_{ji}(t)$= Propagule transfer rate from site *i* to site *j* multiplied by the ratio of surface of site *i* to surface of site *j* [no units]

b_j= Recruitment success at site *j* [adults per larva]

s_{jj}=Adult survivorship rate at site *j* [no units]

δ_{ji}= Kronecker symbol (1 when i=j, 0 otherwise)

Maximum population growth rate (P_i) of a closed population in site *i* is then defined by:

$$P_i = a_i b_i + s_{ii} \qquad (3)$$

The time it takes for a population at any initial density N_o to reach its saturating density N_{max}, from any given initial density was estimated as:

$$T = \frac{\ln\left(\frac{N_{max}}{N_o}\right)}{\ln(P_i)} \qquad (4)$$

In the seascape genetics model, the frequency of each allele *k* is calculated at any given time in every site *i*, as a result of mixing between the surviving individuals with allele *k* in site *i* and the input of new recruits with allele *k*, coming from any other site (Eq. 5). Given the large number of larvae produced by each adult and the high larval survival values assigned (10%) compared to estimated regional retention rates (3%), genetic drift due to larval mortality is neglected and, thus, larval mortality is simulated as a proportional and uniform loss that is applied to the larval pool with allele *k* produced at each site. The effect of genetic drift is therefore simulated by the random sampling of the alleles lost during two different periods in the life cycle: larval dispersal and recruitment. Simplifying assumptions were that: 1) mating takes place randomly every year among diploid organisms, 2) allele frequencies are identical among sexes, 3) generations are discrete but overlapping, 4) populations are not age-structured, 5) given the relatively short period of time considered in the simulations (100 years), mutation is assumed to be negligible compared to genetic drift and migration, and thus set to zero and, 6) selection can be neglected.

During larval dispersal, the pool of larvae with allele *k* produced by each site, remaining in the region (accounting for regional retention rate), is randomly sampled before being re-distributed among the destination sites according to the connectivity matrix (Eq. 6 and 7). The number of larvae with allele *k* that will survive dispersal is computed by sorting out the rank of the allele carried by the surviving larvae. During recruitment, saturating density limits the number of arriving larvae that can be recruited at each site, therefore, a random subset of alleles carried by the pool of larvae arriving at each site is actually recruited.

$$p_{ik(t+1)} = \frac{\left(s_{ii} N_{i(t)} p_{ik(t)}\right) + b_j \sum_j \frac{C_{ij}}{\sum_l C_{lj} \frac{S_l}{S_j}} n'^{\,i}_{jk}}{N_{i(t+1)}} \qquad (5)$$

where n'_{jk} is a random number between 0 and n_{jk}, with the constraint that $\sum_k n'_{jk}{}^i = n'_j{}^i$

$$n'_j{}^i = A_j a_j \left(\sum_l C_{lj} \frac{S_l}{S_j} \right) \quad (6)$$

$$n_{jk} = p_{jk} A_j a_j \quad (7)$$

$p_{ik(t+1)}$= Frequency of allele *k* at site *i* at time *t+1* [no units]

$p_{ik(t)}$= Frequency of allele *k* at site *i* at time *t* [no units]

$p_{jk}(t)$= Frequency of allele *k* at site *j* at time *t* [no units]

S_i = Surface area of site *i* [m^2]

A_j = Density of individuals in site *j* [individual per m^2]

$n'_{jk}{}^i$= Number of larvae produced per unit surface in site *j* having allele *k* that remain in the region and will survive until sexual maturity in site *i*

$n'_j{}^i$= Number of larvae produced per unit surface in site *j* that remain in the region and survive until sexual maturity in site *i*

n_{jk}= Number of larvae with allele *k* produced in site *j* per unit surface

Test cases

The simulations were designed to evaluate the changes in allele frequencies due to variations in species demographic parameters and connectivity structure. Although this modelling approach applies to any sedentary species with a reproductive dispersive stage, to test the

193 sensitivity of the model, we focused on marine benthic species with four different life expectancies
194 (2, 5, 10 and 20 years) that reproduce each year. The values of minimum recruitment success were
195 varied in order to test the effect of maximum population growth in the maintenance of genetic
196 diversity over time. Minimum recruitment success was also the same in all populations (10%),
197 although it was limited by saturating spatial density, which was set to Nmax=6250 individuals/m^2
198 per population.

199 Six groups of simulations were performed (Table 1). All simulations were run for 100
200 iterations, one iteration being the time lapse between two reproductive events. The initial genetic
201 structure was described by ten different alleles present simultaneously in every site. In order to
202 remove the potential bias due a specific initial condition for the genetic structure, simulations were
203 repeated at least 100 times with different and random initial conditions of allele frequencies.

204 The first group consisted of simulations of a closed population (no larval inputs) in which
205 life expectancy was raised from two to 20 years. Groups 2, 3A, 3B, 4A and 4B, consisted of
206 simulations of a metapopulation of a species with a life expectancy of 10 years, distributed among
207 32 sites along the Gulf of Lions (northwestern Mediterranean) (Fig. 2), in which spatial structure
208 was driven by connectivity only. These groups of simulations correspond to the test case of
209 dominating species of the soft-bottom benthic communities in the Gulf of Lions (polychaetes). The
210 continuous sandy-bottom habitat was described by sites of approximately the same width (~ 20
211 km) contiguously distributed along the coast spanning the 10 to 30 meters isobaths. Based on these
212 criteria, the areas of the 32 sites ranged from 6.2 to 121 km^2. Survival and fecundity rates were the
213 same among populations within the metapopulation and were kept constant over time in all of the
214 simulations.

215 Larval transfer between the 32 sites was computed from Lagrangian larval dispersal
216 simulations based on regional hydrodynamic simulations performed with the coastal circulation
217 model SYMPHONIE (Marsaleix *et al.*, 2008). Larval dispersal was simulated by releasing larvae

218 at the average site depth (20 m). Connectivity matrices containing larval transfer probabilities were
219 produced for 20 sets of 10-day long spawning periods in 2004 and 2006 with an average PLD of
220 four weeks (Guizien *et al.*, 2014). A total of 20 different connectivity matrices were used for this
221 analysis.

222 Simulations of Group 2 were performed to evaluate the effect of the distinct connectivity
223 structure on heterozygosity. In this case, connectivity was set deterministically using the same
224 connectivity matrix every year (either one of the 20 variants) and, thus, simulations were only run
225 100 times.

226 Groups 3A, 3B, 4A and 4B were performed in order to test the effect of recruitment (3A
227 and 3B) and habitat loss (4A and 4B) on metapopulation size and the maintenance of
228 heterozygosity within a metapopulation over time. Metapopulation size was represented as average
229 regional coverage, defined as the ratio between regional average population density and saturating
230 density. Connectivity was set stochastically using a different connectivity matrix among the 20
231 variants every year and, thus, simulations were run 500 times to reach the convergence of the mean
232 when combining stochasticity in connectivity and initial condition. Group 3A simulated gene flow
233 among all populations, and recruitment success was constant every year. Group 3B explored a
234 scenario where recruitment failure (no recruitment) occurred every three years. Group 4A
235 simulated the loss of four populations (sites 17, 18, 19 and 20). These populations were identified
236 as being essential for the regional persistence of species at minimum recruitment success in
237 metapopulation modelling, comparing the vulnerability to habitat loss of four ports in the Gulf of
238 Lions (Guizien *et al.*, 2014). Nonetheless, it is important to mention that for the present
239 simulations, the values of minimum recruitment success and fecundity were set to ensure long-
240 term persistence of the metapopulation after removing those four sites, unlike in Guizien *et al*.
241 (2014). Group 4B evaluated a scenario where only one population (site 21) was lost from the
242 metapopulation. This site was characterised by consistently showing the highest values of

243 betweenness in the metapopulation when applying graph theory analysis to connectivity matrices
244 (Costa, *pers. comm.*).
245
246 *Metrics and statistics*
247 Heterozygosity and allelic richness are two measures of genetic diversity commonly used
248 in the population genetics literature, and in the present study both were used. Heterozygosity (He_i),
249 was estimated taking into account all individuals at each site *i* over time (Eq. 7).
250
251 $$\square_{\square}(\,)\sum_{\square}^{\square}(\square_{\square}(\,))^{\square} \qquad (7)$$
252
253 where $p_{ik}(t)$ = frequency of the *k*th allele of *n* alleles in site *i* at time *t* [no units].
254
255 However, heterozygosity and allelic richness describe the state of genetic diversity of a
256 population at a particular time and not its dynamics. In order to measure the speed at which genetic
257 diversity varied as a function of demographic parameters (regulating genetic drift) and
258 connectivity structure, new metrics describing the initial and average slope of the evolution of
259 genetic diversity over time are introduced: 1) Allele Drifting Time (Td), which is the moment at
260 which allelic diversity starts decreasing, meaning the time when the first allele is lost from the
261 population or the metapopulation. 2) Allele Fixation Time (Tf), which is the time lapse between
262 the first loss of an allele (Td) and the fixation of any allele (Fig. 3).
263 These metrics were defined on allelic richness rather than heterozygosity to avoid
264 ambiguity in defining genetic diversity reference points and given the known sensitivity of allelic
265 richness to the loss of rare alleles (Allendorf 1986).
266 $$\square_{\square}(\,)\sum_{\square}^{\square}(\square_{\square}(\,))^{\square}p_{ik}(t)$$

Results

Do differences in demographic parameters affect genetic diversity in closed populations?

Figures 4A and 4B display the loss of genetic diversity in closed populations at initial saturating capacity as a function of the ratio between mortality (being complementary to unity of survival) and recruitment success. This ratio, having the dimensions of larvae/adult/year, depicts the origin of recruitment regulation (*sensu* by Hixon *et al.,* 2002) and informs on the relative contribution of intrinsic vs. extrinsic drivers: high values indicate recruitment regulation due to low recruitment success driven by inter-specific competition or predation, while low ratio values indicate recruitment regulation due to space limitation at a high population density (intra-specific competition). The observed relationship between the loss of genetic diversity (measured as Td) and recruitment success in these simulations, as it is defined in Equation 2, also reflects the relationship between Td and local retention in the case of an isolated population.

Fig. 4A shows that the number of reproductive events after which the population begins to loose its genetic diversity (Td/Re) increases with the ratio between mortality and recruitment success. This demonstrates a reduction of genetic drift when recruitment success decreases for any given life expectancy. However, genetic drift is inhibited at high levels of recruitment success, as depicted by low and almost constant Td/Re for the lowest values of the ratio between mortality and recruitment success. Such minimum Td/Re is of the order of magnitude of the number of reproductive events in a life expectancy. For a recruitment frequency of 0.18, short-lived species (two years) could start loosing their genetic diversity after three years, while it would take around 14 years for a long-lived species (20 years). This reflects the effect of genetic drift inhibition by the reduction of reproductive events due to the lack of space caused by the presence of adults as life expectancy increases.

Similarly, the maintenance of genetic diversity, measured by the number of reproductive events before allele fixation (Tf/Re), in closed populations at an initial saturating density increases

with life expectancy, regardless of whether the population growth rate is low (<2.5) or high (>2.5) (Fig. 5). This confirms that genetic drift is limited by population inertia due to recruitment inhibition as a result of space limitation, as neither fecundity nor recruitment success affects the relationship.

However, when dealing with overlapping generations, it must be taken into account that the reduction of genetic drift by space limitation during the first population turnover decreases with species longevity. This is exhibited by the different hierarchies between life expectancy curves when representing the time at which the population starts to loose its genetic diversity in terms of population turnover (adimensionalising Td by the life expectancy of the species) (Fig. 4B). Species with short life expectancies begin to experience a reduction of their genetic diversity after a higher number of population turnovers than do long-lived species, as fewer reproductive events occur during a short life expectancy compared to during a long one. This result confirms that genetic drift can be controlled by effective reproduction (which may be limited by long life expectancy or low recruitment success) and its frequency along the species lifespan in a context of finite population size.

Different initial population densities also affect the time at which a population starts loosing genetic diversity (Fig. 6A) – for any value of the ratio between mortality and recruitment success, the lower the initial density of the population, the slower the loss of genetic diversity. This result points out that genetic drift is slower when considerable space is available for recruitment compared to at saturating density where space availability is regulated by mortality. Quantifying space availability by the time it will take a population to reach saturating density (6250 individuals/m^2) shows that it would take twice as long for a population with an initial density of 50 individuals/m^2 compared to a population with an initial density of 1000 individuals/m^2 (Fig. 6B). Allele drifting time increases linearly with the time necessary to reach saturating density.

The population growth rate triggers the effect of initial population density when it comes

317 to the maintenance of genetic diversity, measured as Tf (Fig. 5). While in fast growing
318 populations, the maintenance of genetic diversity is not sensitive to initial density, in slow growing
319 populations, the maintenance of genetic diversity lasts longer and is more variable when the initial
320 population size is small (50 individuals/m^2) compared to when it is at saturating density. Such
321 sensitivity to initial population size is amplified at small life expectancies.

322 Overall, in closed populations, genetic drift is the quickest when the population is at
323 saturating density with high recruitment success, slowing down at low recruitment success or when
324 population size is lower than saturating density. This result demonstrates the importance of
325 transient demography in regulating the speed of genetic drift.

326

327 *Do different patterns of connectivity affect the maintenance of genetic diversity in a*
328 *metapopulation?*

329 Simulated heterozygosity patterns among populations forming a metapopulation varied
330 according to each connectivity matrix. The two most contrasting spatial patterns with low
331 heterozygosity values either in the west (A) or in the east (B) of the Gulf of Lions are shown in
332 Fig. 7. A plausible explanation is the different spatial patterns of local retention (defined as the
333 proportion of larvae produced locally that remain in the same spatial unit) vs. import (estimated as
334 the proportion of all larvae produced among all sites that settled within the focal site). For
335 connectivity matrix 1, the ratio between local retention and import was higher in the west than in
336 the east (0.074 and 0.027, respectively). On the contrary, for connectivity matrix 2 the values of
337 the same ratio were higher in the east compared to the west (0.069 and 0.0085, respectively).
338 However, simulations demonstrated that the relationship between Td and import do not exhibit any
339 trend with no linear correlation ($R^2 = 0.011$; $P < 0.01$) (Figure S1). Similarly, Td was not linearly
340 correlated to local retention rate values obtained from the analysis of the 20 connectivity matrices
341 ($R^2 = 0.031$; $P > 0.05$). However, Td was delimited by a piecewise function enabling the

discrimination between two groups (Fig. 8) – populations with low levels of local retention (< 2%) that show high variability in the time they begin to loose their genetic diversity (Td/Le ranging from 0.2 to 50) and populations with high local retention (> 2%) where genetic drift is much quicker and less variable (Td/Le ranging from 0.2 to 12). This indicates that the speed of genetic drift is regulated locally only at a high local retention rate (larger than 2%) while, for local retention rates lower than 2%, the strength of genetic drift depends on the regional structure of connectivity. According to the direction of the transfer and the genetic characterisation of the contributing populations, genetic drift will then be accelerated or slowed down in the focal population.

How does local population stability affect heterozygosity in a metapopulation?

When looking into the variation of the average heterozygosity in the metapopulation through time for a species with a life expectancy of 10 years, it becomes clear that changes in population density have a major effect on the loss of genetic diversity in a metapopulation (Fig. 9). The rate at which a metapopulation reaches a stable regional coverage influences its possibility to maintain genetic diversity over time, even in the presence of migration.

Overall, we observe that as soon as regional coverage reaches 80%, heterozygosity values decrease rapidly and genetic diversity is lost. Scenario A shows how, with constant recruitment success, the effect of migration among populations homogenises allele frequencies over time and leads to the fixation of the same allele in all populations over a relatively short period of time. Most interestingly, in Scenario B, we find that high variability in metapopulation regional coverage due to recurrent failures in recruitment actually helps maintain genetic diversity over time. Stochastic connectivity leading to demographical fluctuations in populations enables the maintenance of genetic diversity.

On the other hand, Scenario C exemplifies how, in the Gulf of Lions, removing

populations (sites 17–20) responsible for bringing demographic persistence to the metapopulation at minimum recruitment success divides it into an eastern and western group, causing a slightly faster decrease of genetic diversity in the latter group than in the former. However, Scenario D shows how the loss of only one site, identified as essential for maintaining the integrity of the connectivity graph, indeed disrupts the genetic connectivity of the metapopulation by augmenting the differentiation between the eastern and western groups in terms of the loss of heterozygosity, with drifting starting earlier in the eastern part. In Scenarios C and B, recruitment success was set in order to ensure that the loss of sites within the metapopulation would still allow for species persistence. It should be noticed that in both scenarios, a similar constant metapopulation size was maintained, even if not at saturating density. The effect of migration in this case will also lead to the loss of genetic diversity in the metapopulation over time, although at a slower rate.

Discussion

The present study highlights that genetic drift increases considerably with settlement and post-settlement limitations due to space availability when the effective number of breeders is approaches saturating capacity, and causes a strong reduction of effective population size.

Early authors had already shown how changes in genetic diversity are related to effective population size (Wright 1931; Kimura 1955). Nonetheless, it has been demonstrated theoretically that life cycle processes, like overlapping generations (Jorde and Ryman 1995), and demographic parameters can also have an effect on the amount of temporal allele frequency change and should be taken into account (Ryman 1997). Our results highlight the role of life cycle processes, other than age at first reproduction (Lee *et al*., 2011), in reducing effective population size and increasing the effect of genetic drift. In closed populations, recruitment intensity regulates the speed of genetic drift on genetic diversity, demonstrating the relevance of transient demography in shaping the genetic structuration of marine species. In open populations, genetic drift is slowed

down as a result of larval exchange. The effect of recruitment failure and the loss of key source populations on heterozygosity confirm that transient demographic fluctuations, remaining above the risk of regional extirpation, actually help maintain genetic diversity over time.

Recently, several meta-analyses and empirical studies evaluating the relationship between PLD and population genetic differentiation across different marine species indicated a weak correlation between the two (Selkoe and Toonen 2011; Faurby and Barber 2012). In some cases, the high uncertainty in the estimates of larval dispersal and measures of population differentiation were suggested as the main causes for the unexpected results, while other studies suggested that differential mortalities and levels of abundance among populations were more relevant (Munroe *et al.,* 2012). The results from our simulations support these findings, while further demonstrating that considering other biological parameters and the inclusion of transient dynamics caused by other demographic parameters, such as life expectancy, fecundity, local retention and recruitment success, would help explain better the variability of genetic connectivity in a metapopulation.

Dawson *et al. (*2014), found that differences in fecundity, population size and PLD could account for the differences in population genetic structure between eight co-distributed rocky intertidal invertebrate species in the eastern North Pacific. They found that population size was positively related to population genetic structure and they attributed the variability among species to differences in recruitment and demography. Furthermore, they highlighted that the differences in migration potential found between theoretical and empirical data could be attributed to the role of genetic drift and natural selection. Here, we corroborate their hypotheses by demonstrating how the strength of genetic drift is influenced by recruitment success, local retention and fluctuations in population density due to habitat fragmentation and recruitment.

Our results confirm that genetic drift is controlled by effective reproduction, as it is observed when genetic diversity decreases due to a reduction of recruitment success caused by space limitation, rather than changes in the number of reproductive adults in the population. This is

417 particularly the case when the filtering during reproduction applies to the fate of the larvae and not
418 on the adults contributing to reproduction. We demonstrate that a long life expectancy and low
419 recruitment success regulate the temporal variation of allele frequencies and reduce the loss of
420 genetic diversity over time. This provides an alternative explanation to empirical evidence of
421 strong genetic structure in long-lived species, when recruitment success is limited by space
422 availability. In fact, it has already been shown how a long-lived species, such as the red coral
423 *Corallium rubrum*, can exhibit patterns of strong genetic differentiation at small spatial scales (tens
424 of metres) in the Mediterranean Sea, suggesting that this could result from recruitment regulation
425 by low population turnover (Costantini *et al.*, 2007).

426 Population regulation occurs when at least one demographic rate is density-dependent
427 (Hixon *et al.*, 2002). Clear evidence of genetic drift accelerating due to recruitment regulation is
428 observed in our simulations for closed populations at the initial saturating density. Furthermore,
429 the evaluation of the ratio between mortality and recruitment success allowed for disentangling the
430 relative contribution of intrinsic (intra-specific competition) vs. extrinsic drivers (inter-specific
431 competition or predation).

432 In open systems, it was already suggested that the amount of local retention could have a
433 strong influence on the relative importance of demographic vs. connectivity parameters in the
434 dynamics of metapopulations (Figueira 2009). Our results confirm those findings and indicate that
435 local retention rate values larger than 2% diminish the influence of the connectivity structure over
436 genetic drift, as genetic drift accelerates in those populations close to saturating capacity. This is
437 particularly relevant for the spatial planning of MPAs. Whether an MPA is created for the
438 conservation of biodiversity or for fishery management, its main purpose is to maintain the
439 persistence of its populations. In the case of marine metapopulations, local retention is considered
440 the currency of persistence: the higher the local retention, the closer the population is to its
441 saturating capacity (Burgess *et al.*, 2014). Nonetheless, it is important to note that, as Figueira

(2009) suggested, under the same flow structure, differences in PLD among species could cause distinct patterns of local retention for different species in the same place, making biodiversity conservation efforts even more difficult. In any case, the loss of genetic variability through genetic drift at a high local retention rate can diminish future adaptability to a changing environment, which might be detrimental to local conservation efforts, strongly advocating for a network of MPAs formed by sites with lower local retention rate.

The maintenance of genetic diversity in the metapopulation over at least 100 years in the case of regular recruitment failure, creating regional demographic fluctuations, was not unexpected. These results, incorporating realistic spatially explicit connectivity matrices, extend previous theoretical studies that found that demographic instability and fluctuations in migration produce major changes in the evolution of genetic variation and population differentiation (Whitlock 1992). Demographic disturbances due to population bottlenecks have been shown to induce strong genetic drift. In the present study, demographic disturbances around saturating capacity can reversely reduce the effect of genetic drift. Furthermore, our results suggests that there is a potential for opposing effects to population disturbances depending on the condition of the population prior to the disturbance: 1) accelerating genetic drift when population census size is high with recruitment limitation, or 2) decelerating genetic drift when population census size is low with no recruitment limitation.

Given that disturbances can cause variations in key demographic and biological processes, disturbance history may be one of the major drivers shaping the patterns of genetic diversity in many natural populations (Banks *et al.,* 2013). The pattern of genetic diversity maintenance over time observed in our simulations has been reported in an empirical study on natural populations of frogs, where no losses of genetic diversity after a disturbance were found when survival was high or when recovering populations recruited many individuals from multiple sources (Spear *et al.*, 2012). In addition, Larson *et al.* (1984) suggested, based on a survey of 22

467 species of salamanders, that historical influences are much more important in describing genetic
468 variation patterns than is recent migration.

469 Previous studies in the region identified the spatial structuration of population
470 vulnerability to habitat loss driven by hydrodynamical connectivity. Four sites essential for species
471 regional persistence at minimum recruitment success were identified from metapopulation
472 modelling around the port of Sète (sites 17–20, Guizien *et al.*, 2014). However, analysis of the
473 same connectivity graph used in the metapopulation model depicted those four sites as high
474 bridging centrality, spanning across the two subclusters of the region. Another site (site 21) was
475 instead identified for maintaining network integrity, as depicted by the highest betweeness (Costa,
476 *pers. comm*). Here, we show that both the loss of four essential populations and the loss of the
477 highest betweenness site divide the metapopulation into two genetically distinct subpopulations
478 (eastern and western). Nevertheless, the loss of only site 21 leads to a higher level of genetic
479 differentiation in the metapopulation. This result suggests that the highest betweeness of
480 hydrodynamical connectivity graph could be interpreted for relaying gene flow and maintaining
481 genetic connectivity, similar to how Rozenfeld *et al*. (2008) interpreted the highest betweenness of
482 genetic distance. This example encourages further investigation of the potential of graph analysis
483 of hydrodynamical connectivity matrices to identify sites important for conservation of genetic
484 diversity.

485 On a local scale, high local retention, which helps maintain a species in isolated protected
486 marine areas, accelerates genetic drift and as a result, accelerates the reduction of genetic diversity.
487 At the network level, the site having the most important role in heterozygosity maintenance
488 (highest betweeness) did not correspond to those sites identified as essential for regional
489 persistence in the metapopulation. The combination of these findings poses a question for
490 management: in order to protect species persistence and genetic diversity, should we be seeking a
491 compromise between preserving populations that are low in terms of diversity but are also

demographically stable vs. highly diverse but unstable populations? The present model, by combining demography and gene flow in realistic conditions allows to 1) disentangle the relative contribution of local demography and environmental connectivity in shaping seascape genetics, and 2) perform *in silico* evaluations of different scenarios for marine spatial planning.

Acknowledgments

This work was (co-)funded through a MARES Grant. MARES is a Joint Doctorate programme selected under Erasmus Mundus coordinated by Ghent University (FPA 2011-0016). Check www.mares-eu.org for extra information. This work was also partly funded by the French National Program LITEAU IV of the Ministère de l'Ecologie et de l'Environnement Durable under project RocConnect—Connectivité des habitats rocheux fragmentés du Golfe du Lion (PI, K. Guizien, Project Number 12-MUTS-LITEAU-1-CDS-013). We would like to thank Proof-reading-service.com for English edition and corrections of the manuscript, and Dr. Lorenzo Bramanti for an early critical review of the manuscript.

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Table 1. Settings of six simulation groups. Connectivity, when present, was set either deterministically (D) using the same matrix every year (either one of the 20 variants) or stochastically (S) picking a different matrix among the 20 variants every year. Disturbance scenarios were only applied to the groups with stochastic connectivity.

	Group 1	**Group 2**	**Group 3A**	**Group 3B**	**Group 4A**	**Group 4B**
Connectivity	NO	D	S	S	S	S
Initial population density (Individuals/m^2)	50, 1000, Nmax	50	50	50	50	50
Fecundity	1000	2000	2000	2000	2000	2000
Life expectancy	2,5,10 and 20 years	5 years	10 years	10 years	10 years	10 years
Number of simulations	100	100	500	500	500	500
Disturbance	NO	NO	NO	Recruitment failure	Habitat loss (source sites)	Habitat loss (betweenness)

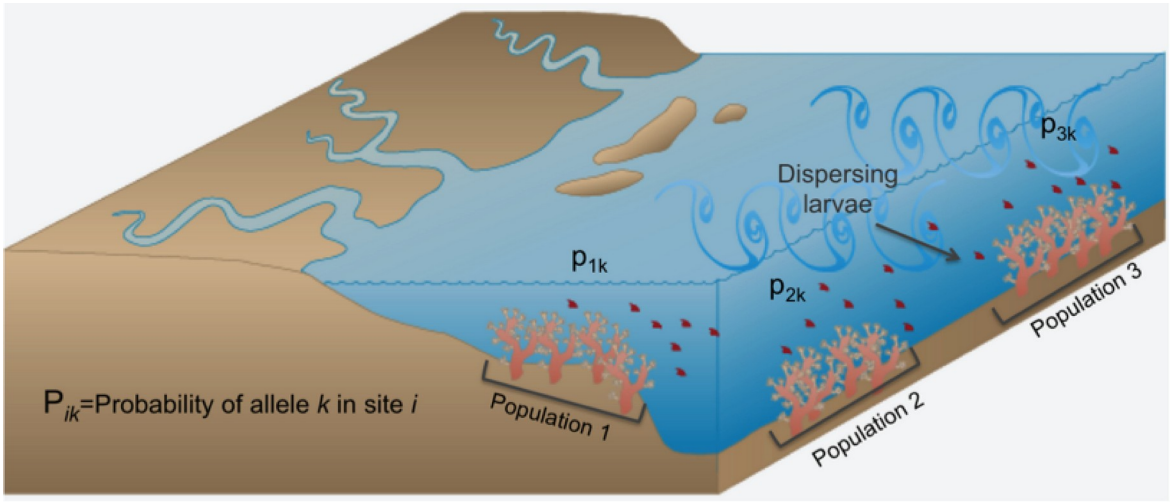

Figure 1. Schematic representation of seascape genetic model. The frequency of each allele k is calculated at any given time in every site i, as a result of mixing between the surviving individuals with allele k in site I and to the input of new recruits with allele k, coming from any other site.

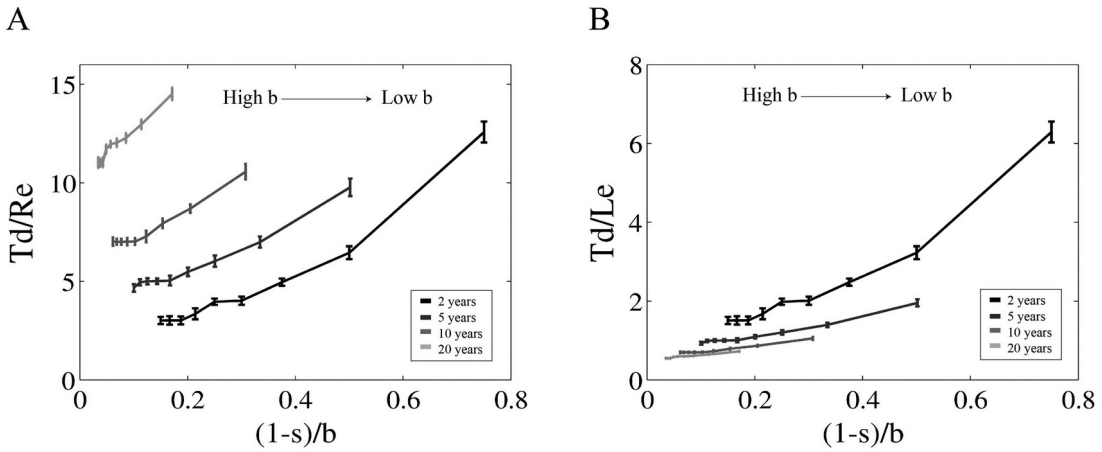

Figure 2. Effect of different life expectancies on the loss of genetic diversity, measure as allele drifting time (T d), for an isolated population at initial saturating capacity (6250 ind. m 22). Adimentionalizing allele drifting time by the number of reproductive events (T d /Re) (a) represents the differences in the number of generations it takes for a species with different life expectancies to start loosing its genetic diversity as a function of the ratio between mortality and recruitment success. In contrast, adimentionalizing allele drifting time by the life expectancy (T d /Le) of a species (b) provides a different vision corresponding to population turnover.

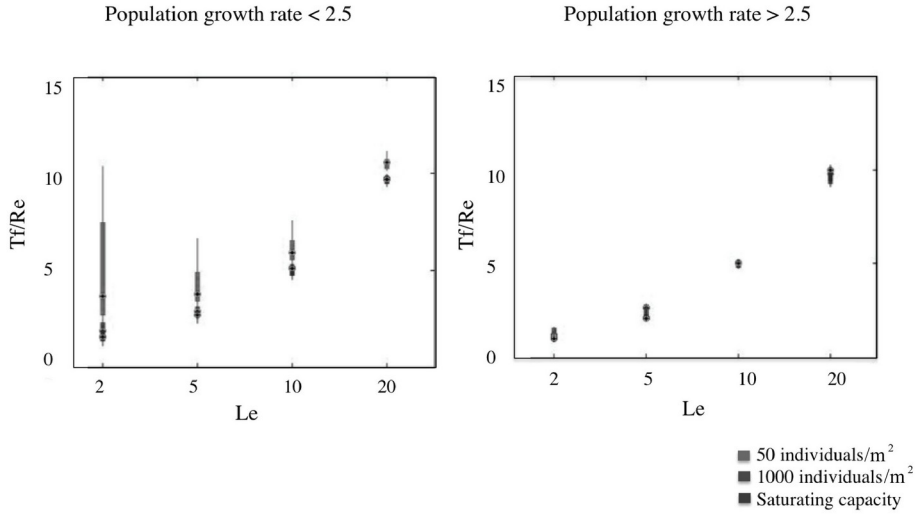

Figure 3. Effect of population growth rate over allele fixation time (T f) for three different initial conditions of population abundance (50, 1000, and 6250 ind. m^{-2}) as a function of species life expectancy (Le) in a closed population.

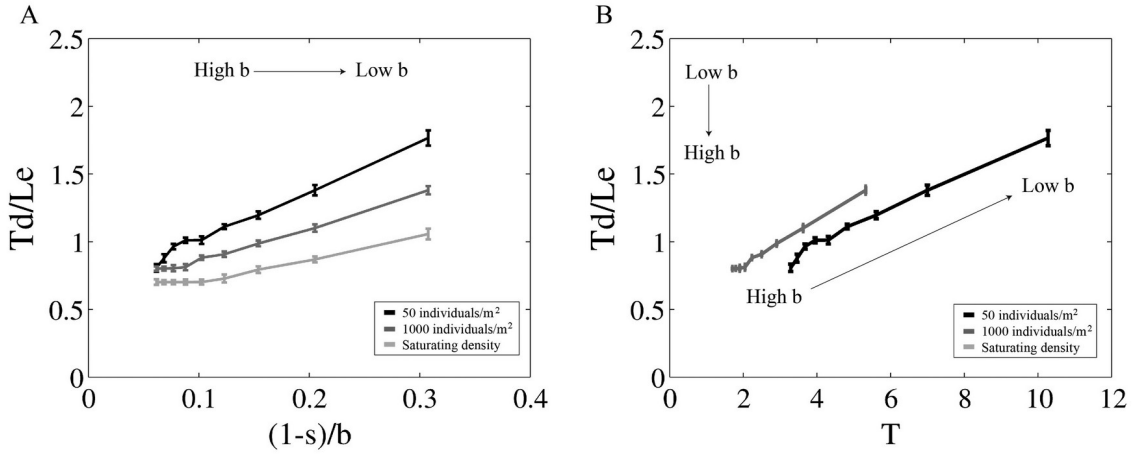

Figure 4. Effect of different initial population abundances (50, 1000, and 6250 ind. m^{-2}) on the loss of genetic diversity, measured as allele drifting time (T d), as a function of (a) the ratio between mortality and recruitment success [(1-s)/b], and (b) the time to reach saturating capacity (T). T d is adimensionalized by the life expectancy (T d /Le) of a species that lives 10 years, thus representing the number of population turnovers to start loosing genetic diversity in an isolated population.

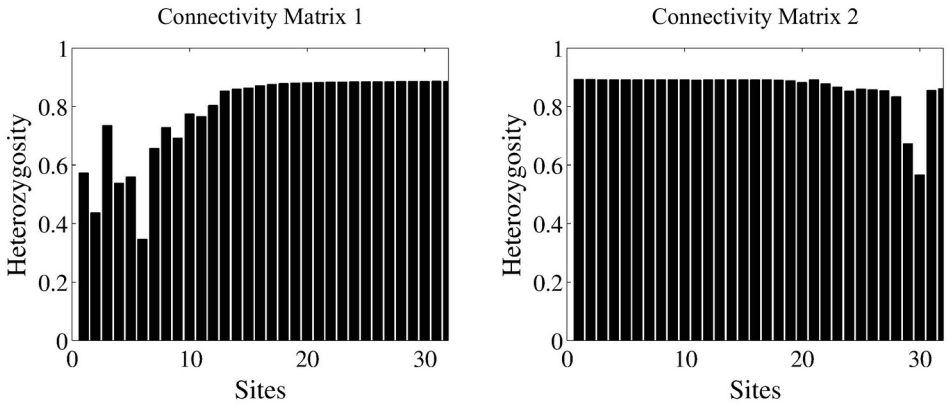

Figure 5. Spatial pattern of genetic diversity caused by distinct connectivity structures. Variability of heterozygosity for 32 sites forming a metapopulation for a species with a life expectancy of 5 years.

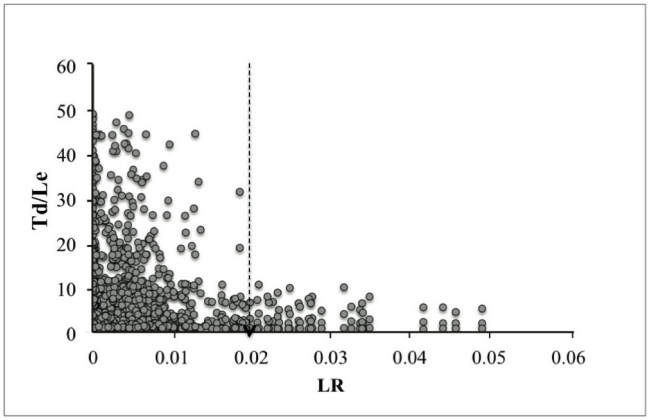

Figure 6. Relationship between local retention (LR; defined as the proportion of larvae produced in one population that remain in the same population) and the number of population turnovers to start loosing genetic diversity (T d /Le), in a metapopulation over time. Values correspond to 20 connectivity matrices, and species with three different life expectancies (2, 5, and 20 years). Dashed arrow indicates the threshold at which the effect of LR on T d /Le begins to decrease.

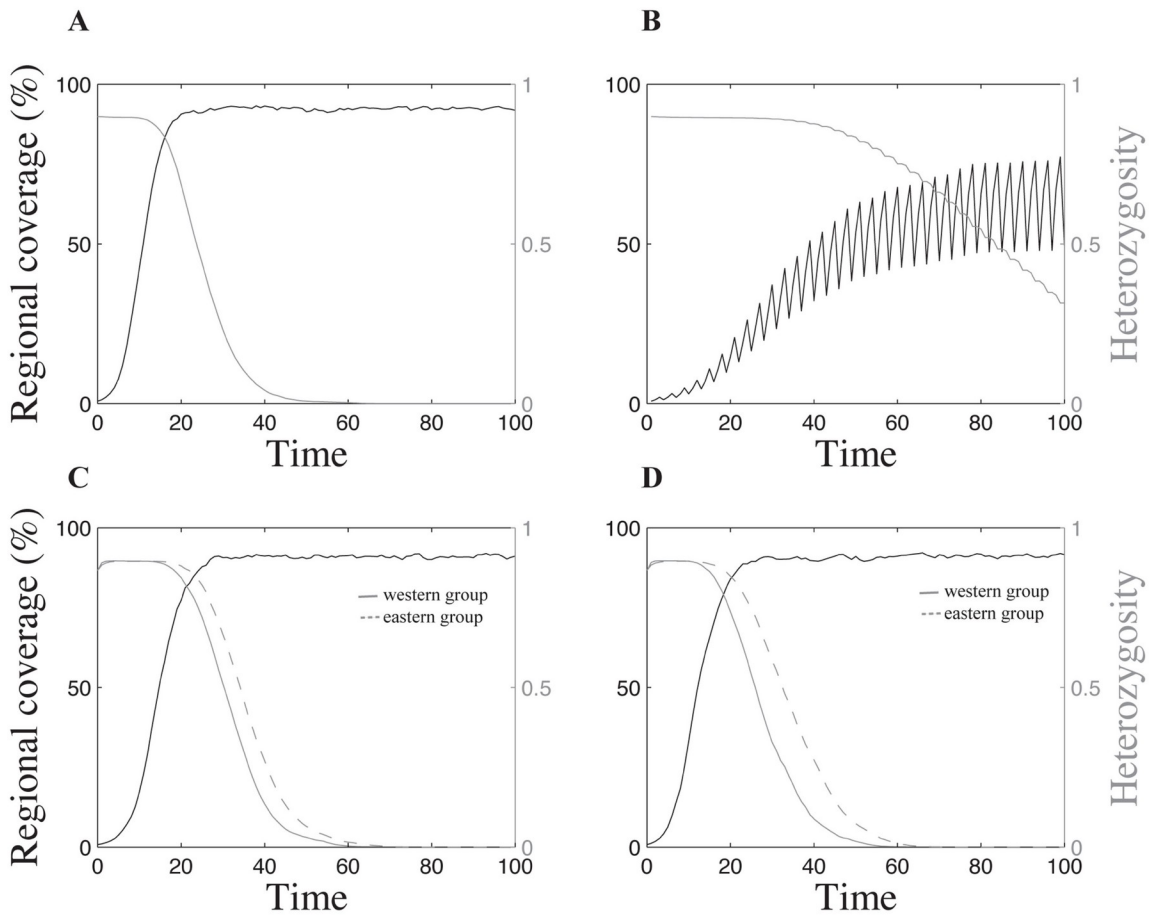

Figure 7. Effect of fluctuations in local population stability on heterozygosity over time. Scenarios: (a) Simulates gene flow among all of the populations forming a metapopulation with constant recruitment success every year. (b) Simulates recruitment failure (no recruitment) every 3 years. (c) Simulates the loss of four source sites (sites 17– 20) in the metapopulation, while keeping recruitment success constant every year. (d) Simulates the loss of one key population (site 21) for maintaining genetic connectivity in the metapopulation.