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1 **Modelling the effect of demographic traits and connectivity on the genetic structuration of**
2 **marine metapopulations of sedentary benthic invertebrates**

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9
10 **Abstract**

11 The genetic structure of populations is commonly used to infer connectivity among distant
12 populations. Accounting for connectivity is essential in marine spatial planning and the proper
13 design and management of Marine Protected Areas (MPAs), given that their effectiveness depends
14 on the patterns of dispersal and colonisation between protected and non-protected areas. Here, we
15 present a spatially explicit coupled metapopulation gene flow model that simulates the effect of
16 demographic fluctuations on the allele frequencies of a set of populations. We show that in closed
17 populations, regardless of population growth rate, the maintenance of genetic diversity at the initial
18 saturating population density increases with species life expectancy as a result of density-
19 dependent recruitment control. Correlatively, at low initial population density, the time at which a
20 population begins to lose its genetic diversity is driven by recruitment success – the larger the
21 recruitment success, the quicker the genetic drift. Different spatial structures of connectivity
22 established for soft bottom benthic invertebrates in the Gulf of Lions (NW Mediterranean, France)
23 lead to very different results in the spatial patterns of genetic structuration of the metapopulation,
24 with quick genetic drift in sites where the local retention rate was larger than 2%. The effect of
25 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.

101 **Methods**

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

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

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

121

$$122 \quad N_i(t+1) = \min(G(t) N_i(t), N_{max}) \quad (1)$$

123

124 $N_i(t+1)$ = Vector of spatial density of individuals at each site at time $t+1$ [individual per
125 m^2]

126 $N_i(t)$ = Vector of spatial density of individuals at each site at time t [individual per m²]

127 N_{max} = Saturating population density based on space limitation [individual per m²]

128

129 with the transfer matrix $G(t)$ defined by:

130

131
$$G_{ji}(t) = a_i C_{ji}(t) b_j + s_{jj} \delta_{ji} \quad (2)$$

132

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

134 $C_{ji}(t)$ = Propagule transfer rate from site i to site j multiplied by the ratio of surface of site i

135 to surface of site j [no units]

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

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

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

139

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

141
$$P_i = a_i b_i + s_{ii} \quad (3)$$

142

143 The time it takes for a population at any initial density N_o to reach its saturating density

144 N_{max} , from any given initial density was estimated as:

145

146
$$T = \frac{\ln\left(\frac{N_{max}}{N_o}\right)}{\ln(P_i)} \quad (4)$$

147

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.

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

171

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

173

174
$$n'_j = A_j a_j \left(\sum_l C_{lj} \frac{S_l}{S_j} \right) \quad (6)$$

175

176
$$n_{jk} = p_{jk} A_j a_j \quad (7)$$

177

178 $p_{ik(t+1)}$ = Frequency of allele k at site i at time $t+1$ [no units]

179 $p_{ik(t)}$ = Frequency of allele k at site i at time t [no units]

180 $p_{jk}(t)$ = Frequency of allele k at site j at time t [no units]

181 S_i = Surface area of site i [m²]

182 A_j = Density of individuals in site j [individual per m²]

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

185 n'_j = Number of larvae produced per unit surface in site j that remain in the region and
186 survive until sexual maturity in site i

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

188

189 *Test cases*

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

sensitivity of the model, we focused on marine benthic species with four different life expectancies (2, 5, 10 and 20 years) that reproduce each year. The values of minimum recruitment success were varied in order to test the effect of maximum population growth in the maintenance of genetic diversity over time. Minimum recruitment success was also the same in all populations (10%), although it was limited by saturating spatial density, which was set to $N_{\max}=6250$ individuals/m² per population.

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

The first group consisted of simulations of a closed population (no larval inputs) in which life expectancy was raised from two to 20 years. Groups 2, 3A, 3B, 4A and 4B, consisted of simulations of a metapopulation of a species with a life expectancy of 10 years, distributed among 32 sites along the Gulf of Lions (northwestern Mediterranean) (Fig. 2), in which spatial structure was driven by connectivity only. These groups of simulations correspond to the test case of dominating species of the soft-bottom benthic communities in the Gulf of Lions (polychaetes). The continuous sandy-bottom habitat was described by sites of approximately the same width (~ 20 km) contiguously distributed along the coast spanning the 10 to 30 meters isobaths. Based on these criteria, the areas of the 32 sites ranged from 6.2 to 121 km². Survival and fecundity rates were the same among populations within the metapopulation and were kept constant over time in all of the simulations.

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

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

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

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

betweenness in the metapopulation when applying graph theory analysis to connectivity matrices (Costa, *pers. comm.*).

Metrics and statistics

Heterozygosity and allelic richness are two measures of genetic diversity commonly used in the population genetics literature, and in the present study both were used. Heterozygosity (H_e), was estimated taking into account all individuals at each site i over time (Eq. 7).

$$H_e(t) = \frac{1}{n} \sum_{k=1}^n (p_{ik}(t))^2 \quad (7)$$

where $p_{ik}(t)$ = frequency of the k th allele of n alleles in site i at time t [no units].

However, heterozygosity and allelic richness describe the state of genetic diversity of a population at a particular time and not its dynamics. In order to measure the speed at which genetic diversity varied as a function of demographic parameters (regulating genetic drift) and connectivity structure, new metrics describing the initial and average slope of the evolution of genetic diversity over time are introduced: 1) Allele Drifting Time (T_d), which is the moment at which allelic diversity starts decreasing, meaning the time when the first allele is lost from the population or the metapopulation. 2) Allele Fixation Time (T_f), which is the time lapse between the first loss of an allele (T_d) and the fixation of any allele (Fig. 3).

These metrics were defined on allelic richness rather than heterozygosity to avoid ambiguity in defining genetic diversity reference points and given the known sensitivity of allelic richness to the loss of rare alleles (Allendorf 1986).

$$H_e(t) = \frac{1}{n} \sum_{k=1}^n (p_{ik}(t))^2$$

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 T_d) and recruitment success in these simulations, as it is defined in Equation 2, also reflects the relationship between T_d 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 (T_d/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 T_d/Re for the lowest values of the ratio between mortality and recruitment success. Such minimum T_d/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 (T_f/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 lose its genetic diversity in terms of population turnover (adimensionalising T_d 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 losing 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/m²) shows that it would take twice as long for a population with an initial density of 50 individuals/m² compared to a population with an initial density of 1000 individuals/m² (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

to the maintenance of genetic diversity, measured as T_f (Fig. 5). While in fast growing populations, the maintenance of genetic diversity is not sensitive to initial density, in slow growing populations, the maintenance of genetic diversity lasts longer and is more variable when the initial population size is small (50 individuals/m²) compared to when it is at saturating density. Such sensitivity to initial population size is amplified at small life expectancies.

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

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

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

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

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

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

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

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

On a local scale, high local retention, which helps maintain a species in isolated protected marine areas, accelerates genetic drift and as a result, accelerates the reduction of genetic diversity. At the network level, the site having the most important role in heterozygosity maintenance (highest betweenness) did not correspond to those sites identified as essential for regional persistence in the metapopulation. The combination of these findings poses a question for management: in order to protect species persistence and genetic diversity, should we be seeking a 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.

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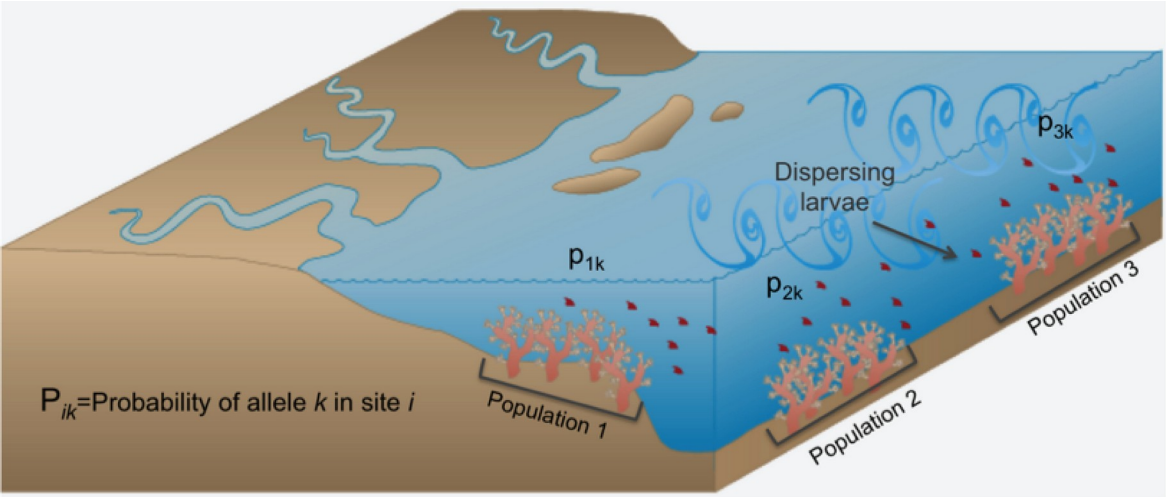
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	Group 1	Group 2	Group 3A	Group 3B	Group 4A	Group 4B
Connectivity	NO	D	S	S	S	S
Initial population density (Individuals/m ²)	50, 1000, Nmax	50	50	50	50	50
Fecundity	1000	2000	2000	2000	2000	2000
Life expectancy	2,5,10 and 20 years	5 years	10 years	10 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)



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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.

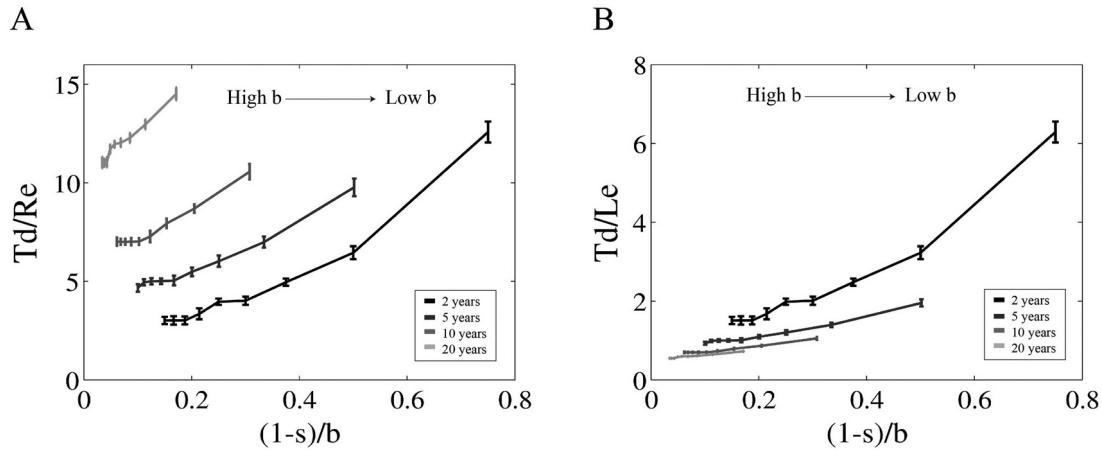


Figure 2. Effect of different life expectancies on the loss of genetic diversity, measure as allele drifting time (T_d), for an isolated population at initial saturating capacity (6250 ind. m^{-2}). Adimensionalizing allele drifting time by the number of reproductive events (T_d/Re) (a) represents the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

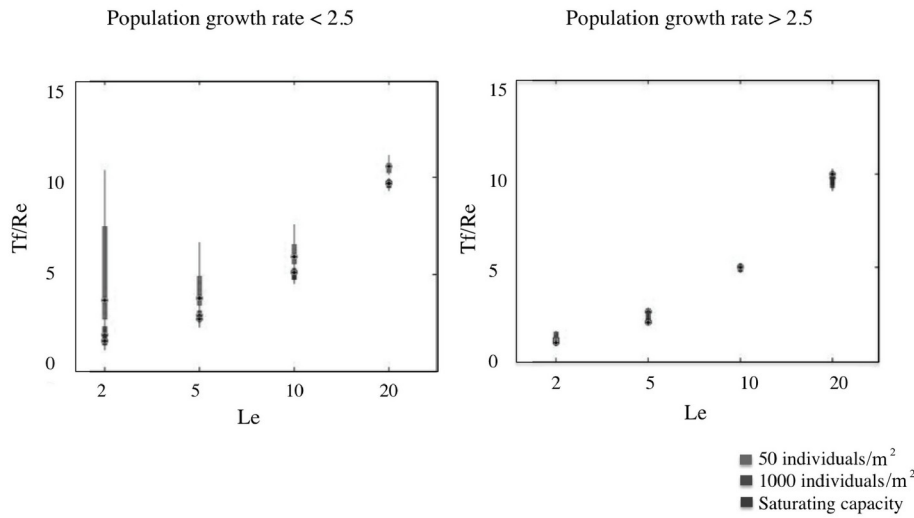


Figure 3. Effect of population growth rate over allele fixation time (T_f) for three different initial conditions of population abundance (50, 1000, and 6250 ind. m^{-2}) as a function of species life expectancy (Le) in a closed population.

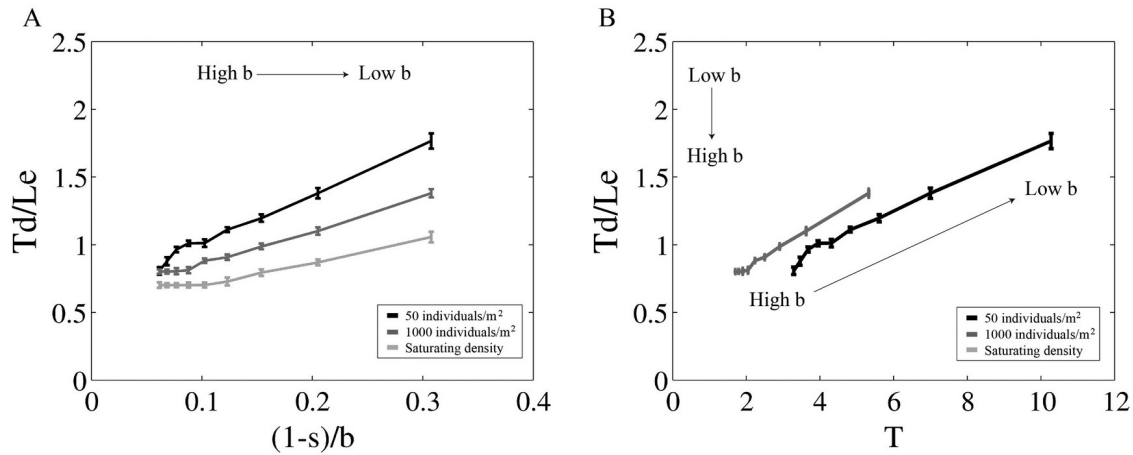


Figure 4. Effect of different initial population abundances (50, 1000, and 6250 ind. m⁻²) on the loss of genetic diversity, measured as allele drifting time (T_d), as a function of (a) the ratio between mortality and recruitment success $[(1-s)/b]$, and (b) the time to reach saturating capacity (T). 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 losing genetic diversity in an isolated population.

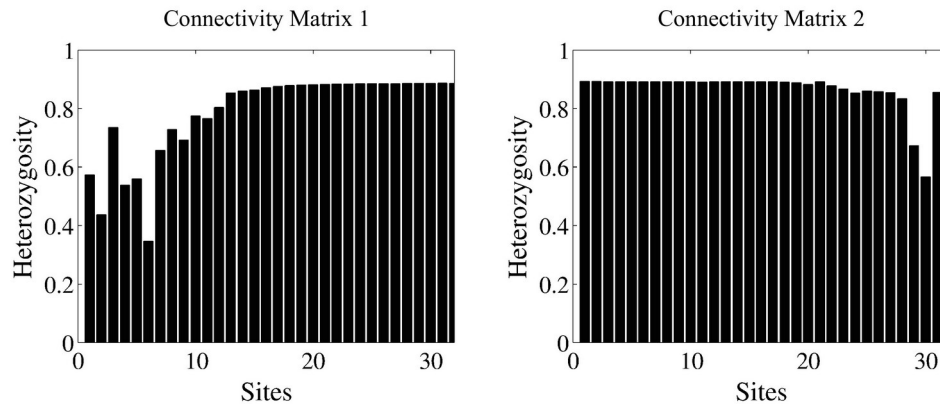


Figure 5. Spatial pattern of genetic diversity caused by distinct connectivity structures. Variability of heterozygosity for 32 sites forming a metapopulation for a species with a life expectancy of 5 years.

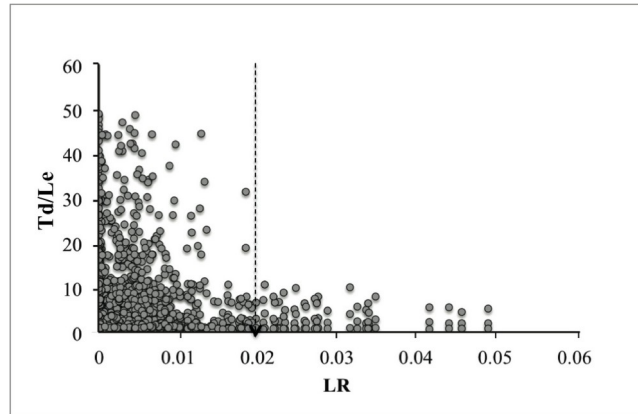


Figure 6. Relationship between local retention (LR; defined as the proportion of larvae produced in one population that remain in the same population) and the number of population turnovers to start losing genetic diversity (T_d/Le), in a metapopulation over time. Values correspond to 20 connectivity 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.

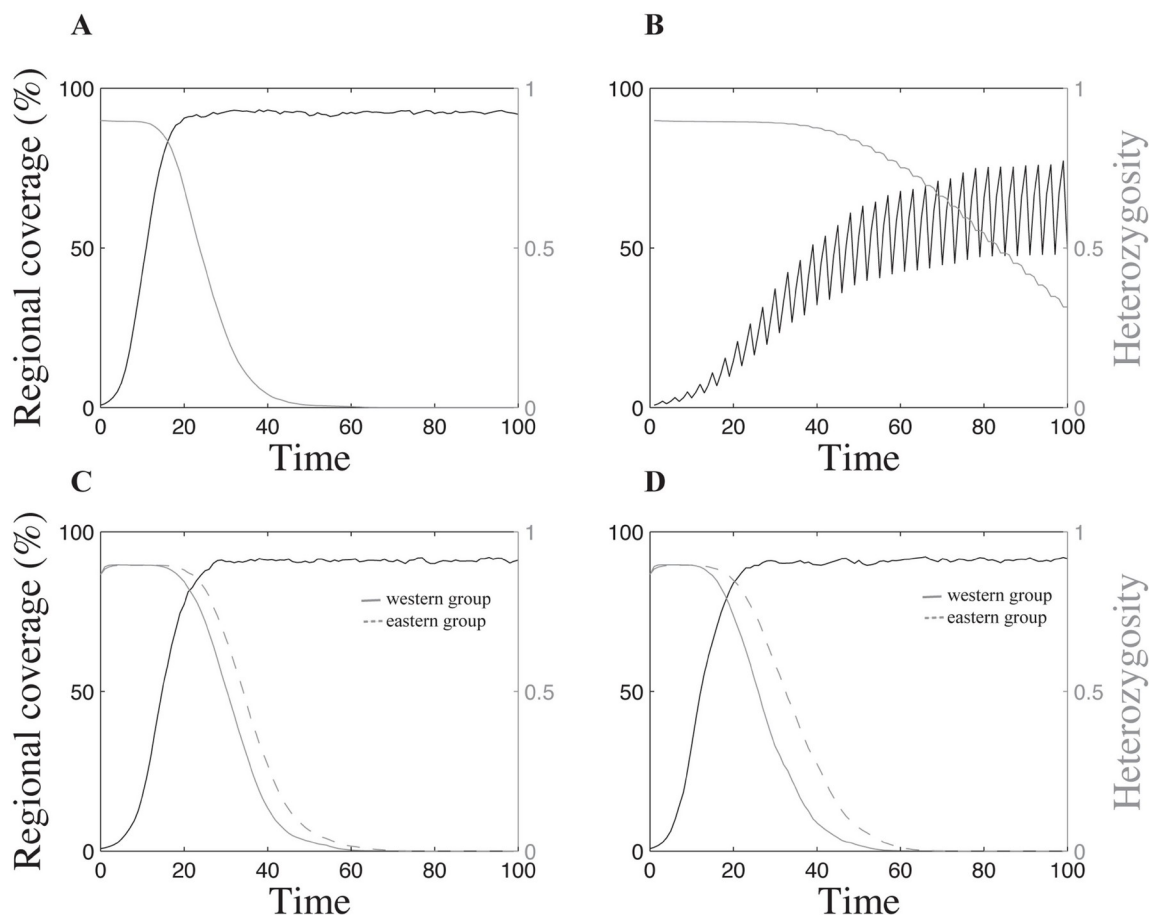


Figure 7. Effect of fluctuations in local population stability on heterozygosity over time. Scenarios: (a) Simulates gene flow among all of the populations forming a metapopulation with constant recruitment success every year. (b) Simulates recruitment failure (no recruitment) every 3 years. (c) Simulates 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.

