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How Do Shellfisheries Influence Genetic Connectivity in Metapopulations? A Modeling Study Examining the Role of Lower Size Limits in Oyster Fisheries

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ARTICLE

How do shellfisheries influence genetic connectivity in metapopulations? A modeling study examining the role of lower size limits in oyster fisheries

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Abstract: Fisheries can potentially alter evolutionary processes such as genetic connectivity and lead to genotypic changes in stocks. Using an individual-based metapopulation genetics model, we examined the possible influence of oyster (*Crassostrea virginica*) fisheries on genetic connectivity. We simulated a range of realistic fishing pressures, with and without a minimum size limit (limit = 63.5 mm), over a range of fishing scenarios including single-area and stock-wide fisheries. Movement of a neutral marker gene provided an indicator of gene transfer between populations. Simulations showed that fishing may alter genetic connectivity. Increasing fishing pressure tended to decrease potential for fished populations to export genes in fisheries with and without size limits. On average, when instantaneous fishing mortality, location, and time period are held constant, fishing unrestricted by size results in a 3.5% lower allele export. Depression of the spawning potential ratio by unrestricted fishing relative to size-limited fishing argues for more conservative fishing mortality targets for unrestricted fisheries. These results demonstrate the importance of considering the influence of fisheries on source–sink dynamics in future management of marine populations.

Résumé : Les pêches peuvent éventuellement modifier des processus évolutionnaires comme la connectivité génétique et ainsi mener à des changements génotypiques dans les stocks. À l'aide d'un modèle de génétique des métapopulations basé sur les individus, nous avons examiné l'influence possible de la pêche à l'huître (*Crassostrea virginica*) sur la connectivité génétique. Nous avons simulé différentes pressions de pêche vraisemblables, avec et sans limite de taille minimum (taille limite = 63,5 mm), pour différents scénarios de pêche dont la pêche limitée à une seule région et la pêche à l'échelle du stock. Les déplacements d'un gène marqueur neutre constituaient un indicateur du transfert de gènes entre populations. Des simulations ont démontré que la pêche peut modifier la connectivité génétique. Une pression de pêche accrue tendait à réduire le potentiel d'exportation de gênes par les populations exploitées dans le cadre de pêches avec ou sans limite de taille. En moyenne, pour un taux de mortalité par pêche instantané, un emplacement et une période de temps constants, la pêche sans limite de taille se traduit par une exportation d'allèles de 3,5 % inférieure à celle de la pêche avec limite de taille. Le potentiel reproducteur relatif plus faible associé à la pêche sans limite de taille par rapport à la pêche avec limite de taille milite en faveur de cibles de mortalité par pêche plus prudentes pour les pêches sans limite de taille. Ces résultats démontrent l'importance de tenir compte de l'influence des pêches sur la dynamique des sources et puits dans la gestion future des populations marines. [Traduit par la Rédaction]

Introduction

Fishing can cause changes in the genetics of exploited populations (reviews: Allendorf et al. 2008; Hutchings and Fraser 2008; Dunlop et al. 2009). This phenomenon, sometimes termed fisheries-induced evolution, occurs when fishing mortality leads to changes in the frequency of certain traits (phenotypes) in the fished stock. Various examples of this phenomenon exist: some include changes in maturation timing (Barot et al. 2004; Olsen et al. 2004; Gårdmark and Dieckmann 2006; Kendall and Quinn 2012), fecundity (Yoneda and Wright 2004; Walsh et al. 2006), body size (Kendall et al. 2009; Olsen et al. 2009), and growth (Ricker 1981; Conover and Munch 2002; Swain et al. 2007; Nusslé et al. 2009). These changes in a fished population can be a result of either phenotypic plasticity, a nonselective response to changes in the environment due to fishing (e.g., density-dependent responses to reduced population), or changes in genotype through selective fishery processes (Heino and Dieckmann 2008; Sharpe and Hendry 2009). These two outcomes are difficult to differentiate and likely act together with many other factors. The evolutionary impact of fisheries is an important question as it relates to ecosystem functioning and resource sustainability (Jørgensen et al. 2007).

Genetic connectivity among populations controls how selected alleles are shared among populations both within and outside of the fished stock (Hendry et al. 2011). The degree of connectivity among populations has direct consequences for species evolution, development of disease resistance, local adaptation, and capacity of a metapopulation to adapt to climate change (Levin 2006; Cowen and Sponaugle 2009; Connolly and Baird 2010). Fishing mortality has the potential to influence genetic connectivity among populations through changes in relative demography of the connected populations. Previous model results have demonstrated that when mortality in a given population is higher than that of adjacent populations, the relative mortality gradient causes decreased overall reproductive

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potential, and thus the ability to export neutral alleles is diminished relative to other populations (Munroe et al. 2012). In their simulations, Munroe et al. (2012) used the same modeling approach employed here to test model sensitivity to various local demographic patterns and larval dispersal patterns. Their simulations demonstrated that changes in neutral alleles over time in an oyster metapopulation are influenced by spatial structure in local demographic characteristics like mortality and population abundance. Fishing mortality is often unevenly distributed over the range of the stock; fisheries may target animals that are more easily accessible (closer to ports), that are outside protected areas, or those with higher growth. By changing the magnitude and spatial patterns of mortality, fishing has the potential to alter the transfer of neutral alleles and ultimately genetic connectivity.

Individual-based models that allow explicit tracking of genetic markers have been used successfully to study fisheries-induced evolution (Dunlop et al. 2007, 2009; Enberg et al. 2009; Wang and Höök 2009). Integration of these genetic models with dynamic metapopulation models provides an important tool for understanding the complexities of genetic connectivity (Epperson et al. 2010; Frank et al. 2011; Lamy et al. 2012). Many adaptive and evolutionary processes of broad interest to fisheries may result from a combination of genetic connectivity and selective forces; however, the activity of fishing may influence genotype even without exerting selective pressure on the population. In this modeling exercise, we simulate neutral allele dynamics in a metapopulation to focus on the dynamics of neutral alleles under the influence of a range of fishing activities. Our goal is to examine the degree to which these fishing activities influence the exchange of alleles among populations even in the absence of selective pressure.

To examine this question, we used oyster fisheries as our model system. Oysters are sessile filter-feeding bivalves. The eastern oyster (*Crassostrea virginica*) is native to the Gulf of Mexico and Atlantic coast of the United States and is the target species of the oyster fishery in those regions. Eastern oysters have a pelagic larval stage lasting 2–4 weeks (Kennedy 1996), after which they attach to hard substrates (often reefs or rocks) where they live as adults. Pelagic larval dispersal is the mechanism for genetic exchange among populations, and dispersal is controlled by hydrodynamics (Bertness et al. 1996; Pineda 1999; Gawarkiewicz et al. 2007; Narváez et al. 2012*a*, 2012*b*), larval swimming behaviour (DiBacco et al. 2001; Metaxas 2001; Shanks and Brink 2005), and larval life span (Grantham et al. 2003).

The fishery for *C. virginica* in the United States is economically important; an estimated 18.2 million lbs (1 lb = 0.454 kg) of meats were landed in 2010, with a value of US$76.2 million (Lowther 2011). Declines in the extent of *C. virginica* reefs in these regions (Beck et al. 2009; Zu Ermgassen et al. 2012) and consequent fishery declines (Mann et al. 1991; Jackson et al. 2001; MacKenzie 2007) are the result of increased disease pressure from two major oyster pathogens, *Perkinsus marinus* and *Haplosporidium nelsoni* (Ford and Tripp 1996), as well as historical overharvest (Rothschild et al. 1994; Wilberg et al. 2011). The current fishery uses a variety of harvest techniques depending on the fishery location and local regulations; techniques include intertidal collection, hand tonging, hydraulic tonging, and dredging. Oyster fisheries can be of two types: one is sometimes called a sack or direct-market fishery that targets large oysters and has a lower size limit. The other type, a seed fishery, harvests oysters of all sizes. The primary goal of the seed fishery is the transplantation of smaller animals to better growing areas or privately maintained leases (Chatry et al. 1983; Dugas 1988; Fegley et al. 2003).

The specific fishery that we used as our model system is the oyster fishery in Delaware Bay. The Delaware Bay oyster fishery is a useful model system in which to examine the influence of fishing pressure and fishery type on genetic connectivity. In Delaware Bay, the commercial oyster fishery is carried out primarily on powered vessels deploying one dredge aft or two dredges abeam. The fishery in Delaware Bay landed 94 470 bushels (1 bushel = 37 L) of oysters in 2011 (Haskin Shellfish Research Laboratory 2012). Like many *C. virginica* stocks along the Atlantic and Gulf coasts of the United States, adult natural mortality rates for oysters in Delaware Bay range from <5% to 55% per year, with elevated rates principally being the product of disease mortality from *Perkinsus marinus* infection (Dermo disease: Ford and Tripp 1996; Bushek et al. 2012). This high natural mortality limits sustainable yield for the fishery. As a consequence, genetic connectivity and the evolution of disease resistance in the metapopulation and how these processes are influenced by the fishery is of interest (Hofmann et al. 2009). Moreover, because these characteristics are representative of most oyster-producing regions along the Gulf of Mexico and Atlantic coasts of the United States, the Delaware Bay case is a broadly applicable example. Unlike many of these areas, however, the oyster populations in Delaware Bay have been continually assessed since 1953 (Ford 1997) and managed sustainably since the early 1960s (Powell et al. 2008), resulting in a 59-year time series that we use here to parameterize these simulations.

The first goal of this study was to use a spatially explicit individual-based eco-genetic numerical model to examine the possible influence of fishing mortality on genetic connectivity. The second goal of this study was to determine how size-selective fisheries (sack fishing) versus size nonselective fisheries (seed fishing) influence genetic connectivity among populations. We hypothesize that these two types of fisheries will influence genetic connectivity differently because in one case (sack fishery), fishing mortality is applied after animals reach maturity, whereas in the other (seed fishery), many animals are removed from the population before first reproduction. It is consequently possible that seed fisheries will exert a greater influence on genetic connectivity than sack fisheries. Assessing genetic connectivity empirically is difficult. Larval tracking is challenging, and genetic data often offer snapshots in time that may be subject to substantial yearly variation. Therefore, we used a metapopulation numerical modeling approach to assess the possible influence of these two fishery types on genetic connectivity.

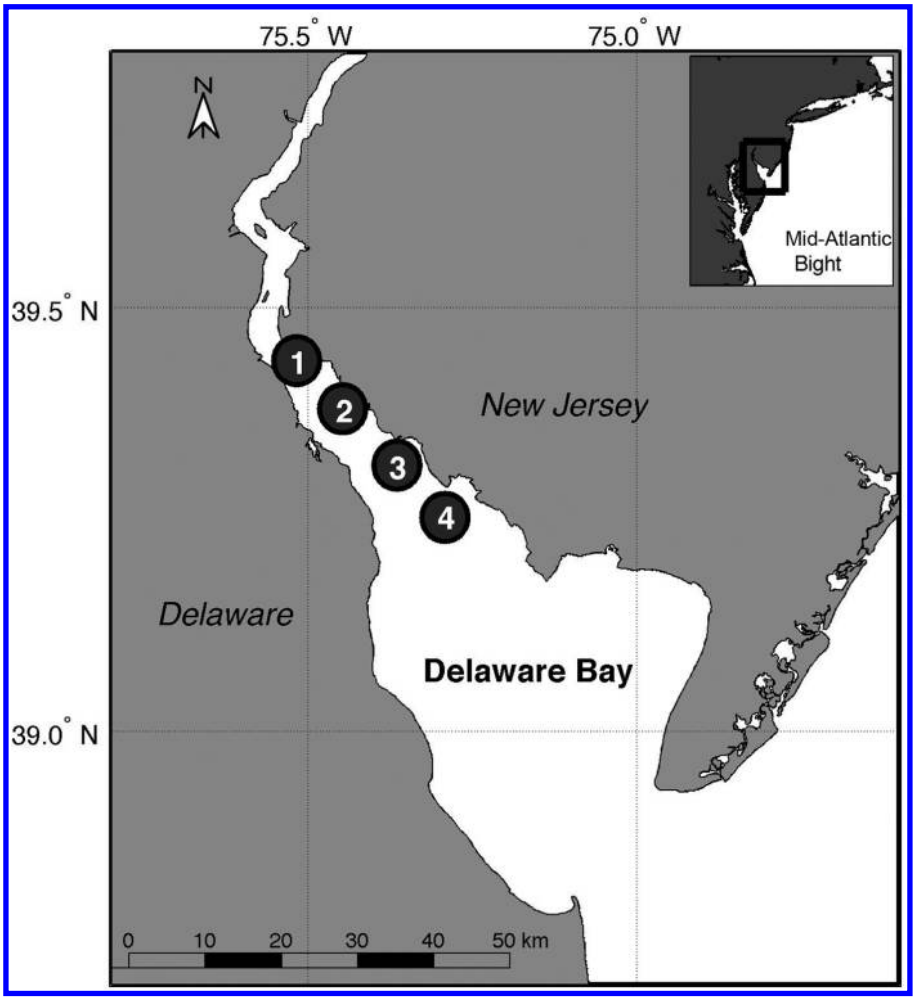
Materials and methods

The model

The Dynamic Population Genetics Engine (DyPoGEn) (Munroe et al. 2012; Powell et al. 2011*a*, 2011*b*) is a numerical model that simulates metapopulation genetic structure and population dynamics. The model incorporates a number of characteristics urged by Lambert (2010) to be included in models of population genetics, including varying population abundance determined by time-varying rates of recruitment and mortality. The model was parameterized to simulate a metapopulation containing four populations of eastern oyster (*Crassostrea virginica*), connected by larval dispersal in Delaware Bay for two time periods: the decade of the 1970s and the decade of the 2000s (further discussion of the populations during these time periods is provided subsequently). Locations of the four populations within Delaware Bay on the Mid-Atlantic coast of the United States are shown in Fig. 1. Additional history of the Delaware Bay oyster population and its fishery is provided in Ford (1997) and Powell et al. (2008, 2009).

Each simulated population is composed of multiple cohorts of oysters, with populations interacting via larval dispersal. Larvae are created from parent pairs via independent assortment of parental genotypes to simulate meiosis and random egg fertilization. Larvae produced in each population can remain within the source population (self-recruitment) or disperse to any of the other populations (dispersal rates shown in Table 1). Many processes in the model (equations described below) depend on a random draw. Random draws (R) use a pseudo-random number generator function (described by Press et al. 1986) from a uniform distribution with a range between 0 and 1. Whenever a normal deviate (N) is required, the gasdev routine of Press et al. (1986) is used to obtain a random deviate from a zero mean, unit variance

Fig. 1. Map of locations of oyster populations in Delaware Bay used in the simulations. Inset shows location of Delaware Bay on the Atlantic Coast of the United States.



normal distribution. Repeat simulations using different sequences of random numbers returned results with only modest variations in scale and trend in initial trials. Consequently, results are provided only for single simulations for each set of parameter values.

The DyPoGEn model has three basic components: (*i*) a post-settlement population dynamics submodel that contains parameterizations for growth, mortality, and reproduction; (*ii*) a larval submodel that contains parameterizations for larval mortality, larval exchange between populations, and early juvenile survival; and (*iii*) a gene submodel that describes each individual in terms of its genetic structure. Additional details of the single population model structure and formulation, on which the metapopulation model is based, are provided in Powell et al. (2011*a*, 2011*b*, 2011*c*). Model processes used in this study pertinent to neutral allele behavior, namely specification of the processes of growth, reproduction, and mortality (see also Munroe et al. 2012), are described below and shown schematically in Fig. 2.

In the population dynamics submodel, the probability of mortality (P_{mort}) is derived from the age of the animal (Age, in years) as

$$P_{\text{mort}} = 0.5\left[1 + \tanh\left(\frac{\text{Age} - \text{MeanAgeMort}}{\text{MeanSpreadMort}}\right)\right] \quad (1)$$

where P_{mort} increases nonlinearly with age such that the rate of increase is low at young and old age and greatest at the mean age of mortality (MeanAgeMort). The range of ages and how steeply the mortality probability approaches 1 is controlled by the denominator of eq. 1 (MeanSpreadMort) (see also examples in fig. 2 in Powell et al. 2011*c*). Juvenile mortality is specified separately as a specific rate applied to recruited animals of age 0.

Fishing mortality is applied to all adults in the population that are larger than the specified lower fishing limit. For these simulations, minimum size limits were set at 63.5 mm (2.5 inches) for the size-limited fishery (sack fishery), consistent with the size frequency of observed landings from the Delaware Bay oyster fishery (Powell et al. 2005) and at 0 mm for the non-size-limited fishery (seed fishery). Fishing mortality is specified by the probability of capture (FishFrac) set in each population. Each individual larger than the fishing size limit is assigned a random value from a uniform distribution between 0 and 1. If the random value is less than FishFrac, the individual is removed from the population by the fishery.

The sex of new recruits is determined by the two-allele system described by Guo et al. (1998). In this system, heterozygotes, those with a dominant male allele *M* and a recessive protandric allele *F* (*MF*) are permanent males; homozygotes (*FF*) are protandric, and the homozygous dominant, *MM*, cannot exist. In each generation, a protandric male is given the chance to convert to a functional female. A conversion probability was obtained from empirical data from Delaware Bay (Powell et al. 2013) using age–length relationships developed by Kraeuter et al. (2007). Powell et al. (2013) modeled the relationship between the fraction of the population that is female, Femfrac, and age as a Gompertz curve:

Table 1. Population characteristics for each of the four populations during two different decades, 2000–2010 and 1970–1980, and larval transfer rates among populations.

	Population 1	Population 2	Population 3	Population 4
Population characteristics of Delaware Bay oysters for the 2000s				
Abundance (millions of oysters)[a]	492	395	868	197
Mean adult mortality fraction[a,f] (%)	8	10	16	26
Juvenile mortality fraction[a,f] (%)	NA[c]	8	23	47
von Bertalanffy growth parameters (Age_0, k, L_∞)[b]	NA[c]	0.2, 0.175, 110	0.2, 0.26, 125	0.2, 0.23, 140
Sex change parameters (α, β, γ)[d]	NA[c]	0.78, –2.6, –0.353	0.74, –5.0, –0.774	0.79, –3.9, –0.653
Population characteristics of Delaware Bay oysters for the 1970s				
Abundance (millions of oysters)[a]	3270	2066	4428	4758
Mean adult mortality fraction[a,f] (%)	11	11	11	11
Juvenile mortality fraction[a,f] (%)	NA[c]	8	23	47
von Bertalanffy growth parameters (Age_0, k, L_∞)[b]	NA[c]	0.2, 0.175, 110	0.2, 0.26, 125	0.2, 0.23, 140
Sex change parameters (α, β, γ)[d]	NA[c]	0.78, –2.6, –0.353	0.74, –5.0, –0.774	0.79, –3.9, –0.653
Larval transfer rates (%) among populations[e]				
Population 1 to:	11	54	27	8
Population 2 to:	6	56	29	9
Population 3 to:	3	40	29	28
Population 4 to:	3	19	14	64

Note: The 2000s conditions were used for parameterization of the 2000s simulations; the 1970s conditions were used for parameterization of the 1970s simulations. Larval transfer rates were used in both time periods.

[a]From Powell et al. 2011*b*; L_∞ in mm, k in years^{-1}.

[b]From Kraeuter et al. 2007.

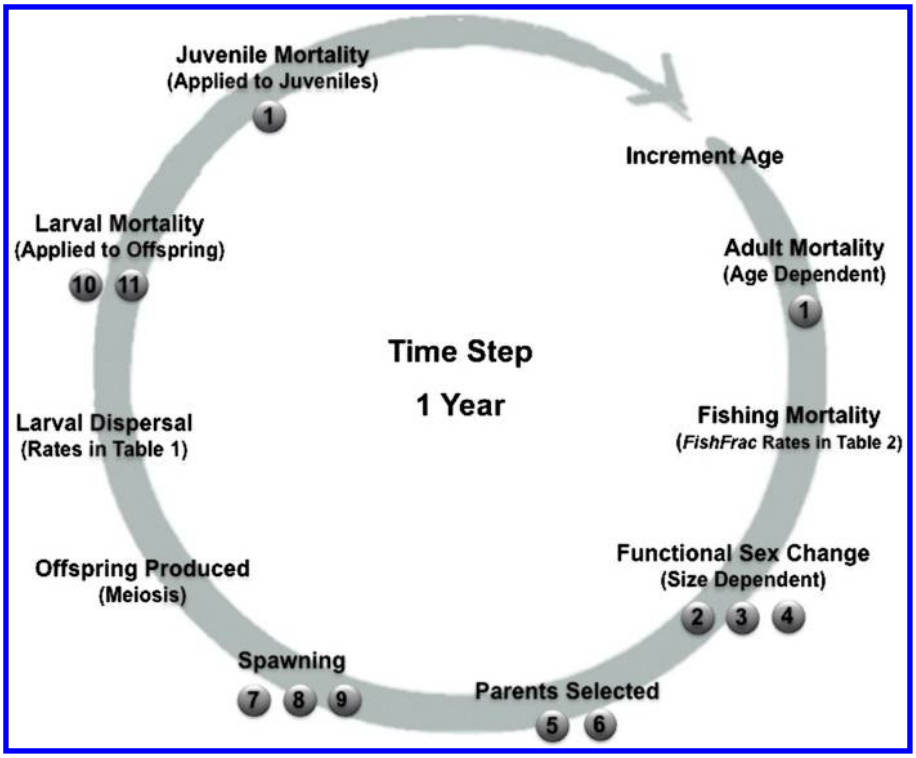
[c]NA, no data available. Used approximated L_∞ from stock assessment data and same juvenile mortality and k as population 2.

[d]From Powell et al. 2012.

[e]From fig. 7*e* in Narváez et al. 2012*a*.

[f]Fraction is equivalent to $1 - e^{-mt}$, where m is the specific mortality rate and t is 1 year.

Fig. 2. Model schematic of processes executed in a single time step (1 year). Numbered circles below each process indicate the equations invoked in that process.



$$\text{Femfrac} = \alpha e^{\beta e^{(\gamma \text{Age})}} \quad (2)$$

where α and β are population-specific parameters (Table 1). The first derivative of eq. 2 gives the rate at which any animal can change from male to female (df) as

$$df = \frac{d\text{Femfrac}}{d\text{Age}} = \alpha\beta\gamma e^{[(\gamma \text{Age}) + (\beta e^{\gamma \text{Age}})]} \quad (3)$$

where γ is a population-specific parameter (Table 1). The probability of conversion ($P_{\text{sex}\Delta}$) is

$$P_{\text{sex}\Delta} = \min\left(1, \frac{df}{1 - \text{Femfrac}}\right) \quad (4)$$

Owing to the age dependency of the probability of sex change, all long-lived protandric individuals eventually become functional females. As all oysters that are protandric begin life as males, all recruits are male. However, some recruits become female prior to first spawning, as appears to be the case in populations from Delaware Bay (Powell et al. 2013).

The fraction of the population parenting each generation (FrParents) is derived from a predefined fraction of parents reproducing each mating season (FracParents set at 0.05% annually), based on estimates of effective population number for oysters in Delaware Bay (Hedgecock et al. 1992; Hedgecock 1994).

$$\text{FrParents} = \text{FracParents} \times 10^{(N\times\text{FracParentsVar})} \quad (5)$$

where the coefficient, FracParentsVar, permits variability to exist in the fraction of parents reproducing. The number of parental pairs (n_{Parents}) is determined as

$$n_{\text{Parents}} = (0.5 \times \text{FrParents} \times \text{LastAnimal}) \quad (6)$$

where LastAnimal is the count of adult animals in the population.

Potential parents are drawn randomly, without replacement, from a list of all animals greater than 1 year of age (Kennedy 1983; Powell et al. 2012) until enough males and females accrue to provide n_{Parents} or until the list of animals is exhausted. Each pair of parents, taken randomly without replacement from the parents' list, produces a number of offspring up to a maximum number, which represents a typical larval settlement (set), at the beginning of the simulation. The number of offspring produced is dependent upon parental age through a mass-based relationship that is described by a von Bertalanffy equation (Fabens 1965; Jensen 1997) that relates size and fecundity to age as

$$M = M_{\infty}[1 - e^{-k(\text{Age}-\text{Age}_0)}]^b \quad (7)$$

where M_{∞} is the maximum mass, and k and Age_0 are population-specific von Bertalanffy parameters (Table 1). The value of M_{∞} is obtained from the adult maximum length, L_{∞}, using an allometric equation that relates mass and length as

$$M = a \times L^b \quad (8)$$

with $a = 0.0003$ and $b = 2$. Note that for oysters, mass scales more nearly with the square of length rather than the cube (Yoo and Yoo 1972; Powell and Stanton 1985).

Equation 7 is applied to fecundity by assuming that oyster spawn is a standard fraction of biomass (Hofmann et al. 1992, 1994). The number of offspring (n_{Off}) produced by a female of a given age and mass is estimated as

$$n_{\text{Off}} = \frac{M_{\infty}}{M_{76}}[1 - e^{-k(\text{Age}-\text{Age}_0)}]^b \text{Max}_{\text{Off}} \quad (9)$$

where M_{76} is the mass of a 76 mm oyster and Max_{Off} is the fecundity of a 76 mm oyster, which can be as much as 60 million eggs per female (Davis and Chanley 1955). For the simulations used in this study, the value of Max_{Off} was set as maximum fecundity of 100 000 eggs per female to reduce computation time. This maximum limit on fecundity has been demonstrated to be robust to allele loss through drift (Powell et al. 2011*c*). Genotypes of the offspring are determined by random combination of haploid genotypes, one from each parent, after meiosis. Recombination can occur during meiosis, in which crossing over of alleles occurs at a randomly chosen locus on each chromosome.

In the larval submodel, all offspring produced are transferred among the populations in the metapopulation using a transfer probability obtained from Lagrangian particle simulations that used an individual-based model of oyster larval growth and behavior that was coupled to a Delaware Bay circulation model (Narváez et al. 2012*a*, 2012*b*). The circulation model is an implementation of the Regional Ocean Modeling System (ROMS; Haidvogel et al. 2000; Shchepetkin and McWilliams 2005). The Delaware Bay circulation model has a horizontal resolution that ranges from 0.2 to 2.1 km and a vertical resolution that ranges from 0.03 to 6.2 m. Details of the configuration and calibration of the implementation for Delaware Bay are given in Wang et al. (2012).

The individual-based model is based on the growth and behavioral models developed for eastern oyster larvae described in Dekshenieks et al. (1993, 1996, 1997). The larval growth model estimates larval growth as a function of temperature, salinity, food supply, and turbidity and was parameterized using laboratory and observational studies (Dekshenieks et al. 1993). Larval vertical migratory behavior depends on salinity (controls time swimming), temperature (controls swimming speed), and larval size (controls swimming and sinking speed) (Dekshenieks et al. 1996). The larval model was implemented as a component of the passive Lagrangian particle tracking module in ROMS; the larval model added vertical velocity to the particles, thereby changing their behavior, and established an end point to the Lagrangian trajectories based on larval size at settlement (Narváez et al. 2012*a*, 2012*b*).

The larval transfer rates used in this study were obtained from connectivity matrices that were constructed from analyses of Lagrangian particle simulations for Delaware Bay (Narváez et al. 2012*a*). The connectivity between oyster reefs in Delaware Bay was determined by the percentage of particles released in an area that settled in either the release region (i.e., self-recruitment) or another region. Populations 1–4 used in this study (Fig. 1) correspond to the Hope Creek (HOP), Arnolds (ARN), Shell Rock (SHR), and Bennies (BEN) oyster beds, respectively, used by Narváez et al. (2012*a*) to calculate larval exchanges (see fig. 7 in Narváez et al. 2012*a*). For this study, only larvae released in these four areas that settled in the four areas were used to calculate transfer rates (Table 1).

The larval recruits were assigned to one of the four areas by biased random draw, the bias being determined by the calculated transfer probabilities. A survival probability in the receiving area, constrained between 0 and 1, was defined as

$$\text{LarvSurv} = (0.5 + 1.5R)\frac{K}{4 \times \text{ReprPerAdult} \times \text{LastAnimal}} \quad (10)$$

where R is a uniform random number that permits individual recruitment events to vary about the broodstock–recruitment relationship, LastAnimal is the total number of animals in the population, ReprPerAdult is the total number of eggs produced by the spawning subset of the adult population scaled to the total number of adult animals in the population, and K is the local carrying capacity, which regulates the number of animals in the population. A full derivation of this equation is provided by Powell et al. (2011*c*). This relationship incorporates the logistic process in which mean recruitment per adult declines as population abundance increases with respect to the local carrying capacity. The ability of oysters to filter water more rapidly than food is resupplied, thereby generating a food limitation (Wilson-Ormond et al. 1997; see also Powell et al. 2013), provides a theoretical basis for this expectation. The probability of death (P) for each individual larva is

(11) $P = 1 - \text{LarvSurv}$

If $R < P$ in a random draw, then the larva dies. If the larva survives to recruit into the destination population, it is given an age of zero.

Model caveats

As with any modeling exercise, a trade-off exists between realism and model simplicity. Genetic connectivity in a metapopulation is a complex biological process. To model this process using realistic parameterizations, the trade-off between realism and model simplicity requires that certain processes and drivers be excluded, either because their relationship with other components is inadequately understood or to simplify the model. In these simulations, we do not include environmental drivers explicitly. Environmental changes (differences in salinity and temperature, for example) are known to influence dispersal (Narváez et al. 2012*a*, 2012*b*), oyster mortality (Gunter 1955; Soletchnik et al. 2007; Bushek et al. 2012), and growth (Kraeuter et al. 2007). Rather than including these environmental drivers explicitly, the model uses differential parameterization of populations and simulations to vary population dynamics consistent with the known range of environmental conditions represented. We used, for example, different mortality rates that are based on known differentials in mortality determined by position in the salinity gradient (Powell et al. 2008), thereby incorporating the influence of salinity implicitly. Additionally, interannual stochasticity is generated in the model through reliance of model processes on a random draw; this compensates for the lack of direct environmental drivers.

Simulations

An individual genotype is defined by a complement of 10 chromosome pairs with four genes per chromosome. Each gene is defined by two alleles, *A* and *B*. Gene transfer among the populations was observed by initializing the model with 100% of the individuals in one population being homozygous *BB* at a particular locus, while the initial individuals in the remaining three populations were all homozygous *AA* at the same locus. This allows tracking of allele frequencies of the *B* allele to follow the movement of neutral alleles from one population through the metapopulation over time. A series of simulations was conducted for conditions parameterized for the 1970s and 2000s. The simulations allow fishing of all oysters in the population (seed fishery) or only those 63.5 mm and larger (sack fishery) at different exploitation levels depending on the simulation. The fishing fractions (FishFrac) set for each population in each of the simulations are listed in Table 2 and are described further below.

The base case simulations were parameterized to allow the four simulated populations to have characteristics of the Delaware Bay populations for two time periods: the decades of the 1970s and 2000s. Data from annual stock assessments of oysters in Delaware Bay (Powell et al. 2009; Haskin Shellfish Research Laboratory 2012) document distinctive oyster population dynamics such as differences in local population abundances and mortality rates for the four simulated populations during the 1970s compared with the 2000s. Both the abundances and mortality rates of the four populations were relatively equivalent among all four populations during a period from ca. 1970 to 1985 in contrast with the strong up-estuary to down-estuary gradient in mortality and biased abundance favoring population 3 in the 2000s (Table 1). Larval transfer rates among populations, von Bertalanffy growth rates, probabilities of juvenile and adult mortality, and carrying capacity are specified for each population independently as outlined in Table 1 (also described in Munroe et al. 2012). Population abundances were maintained sufficiently high to minimize the influence of drift (Powell et al. 2011*c*) that might otherwise influence the results from simulations of genetic connectivity (e.g., Gandon and Nuismer 2009). Note in particular that the gradient in natural mortality in the 2000s (Table 1) was produced by Dermo disease (caused by the parasite *Perkinsus marinus*; Ford and Tripp 1996), which increases in severity with increasing salinity. Disease mortality was inconsequential in the 1970s. The analogous gradient in juvenile mortality is driven by the downbay increase in predators of juvenile oysters. Note also the differential growth rates among the populations, such that oysters in lower salinity express slower growth and longer life span (Kraeuter et al. 2007). Thus, these four populations diverge in important attributes of population dynamics, including growth, mortality, and population density.

The series of simulations performed included both seed and sack fishing, both simulated either as single population fisheries (fishing only allowed in one population) or fisheries exploiting the entire metapopulation (stock-wide fisheries) and each simulated for both time periods (1970s and 2000s). The fractions (FishFrac) allowed in each of the single-population fisheries resulted in the removal of 2%, 10%, 16%, and 30% of the population annually for the seed fishery and 4%, 20%, 35%, and 42% of the population

Table 2. Fishing fractions (FishFrac) set for each population in each of the simulations run for both the 1970s and 2000s regimes.

	FishFrac Pop. 1	FishFrac Pop. 2	FishFrac Pop. 3	FishFrac Pop. 4
No fishing: base case	0	0	0	0
Single-population fishing simulation				
Fish1	0.04	0	0	0
Fish2	0	0.04	0	0
Fish3	0	0	0.04	0
Fish4	0	0	0	0.04
MedFish1	0.20	0	0	0
MedFish2	0	0.20	0	0
MedFish3	0	0	0.20	0
MedFish4	0	0	0	0.20
HiFish1	0.35	0	0	0
HiFish2	0	0.35	0	0
HiFish3	0	0	0.35	0
HiFish4	0	0	0	0.35
VHiFish1	0.42	0	0	0
VHiFish2	0	0.42	0	0
VHiFish3	0	0	0.42	0
VHiFish4	0	0	0	0.42
VLowSeed1	0.02	0	0	0
VLowSeed2	0	0.02	0	0
VLowSeed3	0	0	0.02	0
VLowSeed4	0	0	0	0.02
LowSeed1	0.10	0	0	0
LowSeed2	0	0.10	0	0
LowSeed3	0	0	0.10	0
LowSeed4	0	0	0	0.10
MedSeed1	0.16	0	0	0
MedSeed2	0	0.16	0	0
MedSeed3	0	0	0.16	0
MedSeed4	0	0	0	0.16
Seed1	0.30	0	0	0
Seed2	0	0.30	0	0
Seed3	0	0	0.30	0
Seed4	0	0	0	0.30
Whole-stock fishing simulation				
LowFish	0.04	0.04	0.04	0.04
HighFish	0.39	0.39	0.39	0.39
LowSeed	0.04	0.04	0.04	0.04
HighSeed	0.39	0.39	0.39	0.39

Note: Fishing fraction is the annual probability of capture set in each population. For each individual larger than the fishing size limit (63.5 mm for the sack fishery; 0 mm for the seed fishery), a random draw (*R*) is made. For $R \leq$ FishFrac, the individual is removed from the population by the fishery; for $R >$ FishFrac, the individual remains in the population.

annually for the sack fishery; fractions investigated in both seed and sack stock-wide fisheries resulted in the removal of 4% and 39% of the population annually.

Parameterization for the fishing fractions used was based on values obtained from the literature. Harding et al. (2010), calculating oyster seed fishing for the Piankatank River between 1998 and 2009, approximated 30% of the stock annually. Oyster sack fishing rates reported in Delaware Bay approximated 4% of the stock annually (Powell et al. 2008) and are reported to have ranged from 21% to 72% in the Chesapeake Bay (Rothschild et al. 1994; Jordan et al. 2002; Jordan and Coakley 2004). Thus, our matrix of 72 simulations ([(8 single-population rates × 4 populations) + 4 stock-wide rates] × 2 regimes) included both time periods and a range of either seed or sack fishing fractions in addition to the base cases.

Analysis

Metapopulation allele frequency was calculated as the fraction of animals in all four populations possessing a *B* allele in a given locus at a given time; change in allele frequency was calculated as the frequency in the metapopulation at the end of the simulation (100 years) minus the frequency at simulation year 1. The effect of the fishery relative to the nonfishing base case was calculated as the difference between the change in metapopulation allele frequency in the base case minus the change in metapopulation allele frequency for the same allele in the fished case.

Parameterization of the model specified the fraction of animals removed from the population by the fishery in a year (number fished/number in the population). This is distinct from the instantaneous fishing mortality rate, *F*, that is used in fisheries. *F* is not specified in the model but can be obtained from the simulation output as follows:

$$\text{Bio}_t = \text{Bio}_0 e^{-Ft} \tag{12}$$

where Bio_t is the biomass of the population at time t and Bio_0 is the initial biomass. One time step is equal to 1 year, and when considering only fishing mortality, Bio_t is equal to Bio_0 minus the fished biomass. Letting $t = 1$ and solving for F gives

$$F = -\ln\left(1 - \frac{\text{Bio}_{\text{fish}}}{\text{Bio}_0}\right) \tag{13}$$

where Bio_{fish} is the biomass removed by fishing in 1 year. Arguably, the fishing mortality rate could be calculated based on biomass of the fishable stock only (rather than the entire population) or based on the numbers of individuals rather than the biomass. In this case we chose to use the biomass of the entire population because the two types of fisheries being studied here can then be compared with one consistent metric.

Fishing mortality rate (*F*) was calculated for all single-population fishery simulations. Pairs of seed and sack simulations were identified for each regime and population fished based on nearly equivalent computed values of *F*. A posteriori comparisons generated in this way were necessary because the fraction fished for the entire population for the sack fishery varied based on the relative abundance of animals in the unfished and fished size classes, and thus an equivalent *F* could not be stipulated a priori for the simulation. These paired simulations permitted tests of the influence of seed versus sack fishing on the change in allele frequency, under otherwise constant conditions (e.g., regime, fishing location, fishing mortality rate). Fishing mortality rates and change in allele frequency were non-normal in their distribution; therefore nonparametric Wilcoxon signed-rank tests were performed using the R statistical package (R Development Team 2007) to test for differences in the fishing mortality rate (*F*) and in the difference in allele frequency relative to the base case for the pairs of simulations.

Spawning potential ratio (SPR) (see Goodyear 1993) was calculated for each of the pairs of simulations. Spawning potential was calculated annually for the fished population as

$$\text{SPR} = \frac{\text{total reproduction in a given population}}{\text{total recruitment}} \tag{14}$$

Mean SPR was calculated as

$$\text{SPR}_{\text{mean}} = \frac{\text{spawning potential of the fished population}}{\text{spawning potential of the unfished population}} \tag{15}$$

Finally, because relative abundances in the populations can influence genetic connectivity (Munroe et al. 2012), the mean proportional abundance of oysters in each population was compared with the abundance in the entire metapopulation for each simulation. Years 20–100 of the 100-year simulation were used for calculation of mean SPR and mean proportional abundance to avoid model initialization effects on population size frequency.

Results

The frequency of the neutral marker allele within each population was dynamic. In a population where that marker allele is initiated, the frequency begins at 100%, then drops to some equilibrium level in 15–30 years as the nonmarker allele mixes into this population (Fig. 3). In populations other than where the allele is initiated, the frequency of the marker begins at zero and increases to an equilibrium level as the marker allele is mixed into the population (Fig. 3). The rate of change and the equilibrium frequency differed among populations (local demographic rates), time period (1970s versus 2000s), and, depending on the fishing mode (seed versus sack), rate and location. The metapopulation frequency for those marker alleles was also dynamic and reached the same equilibrium level that each of the populations did; the metapopulation marker allele frequency over time is shown with the shaded areas in Fig. 3.

Change in metapopulation allele frequency is used here to describe the potential for a population to act as a source of alleles for the metapopulation as a whole. When population abundances remain constant relative to one another through time (as was the case for the base case simulations here) an increase in the metapopulation allele frequency over time results from the ability of the population from which the allele originates to export that allele to other populations; thus that population acts like a source for that allele. Conversely, a decrease in the metapopulation allele frequency would result from the inability of the population from which the allele originates to export that allele to other populations; thus that population acts like a sink. The change in metapopulation allele frequency varied with the simulated time period (1970s versus 2000s), fishing pressure, type of fishery (seed versus sack), and the population in which the marker allele was initially present (local demographic rates). Change in metapopulation allele frequency ranged from a maximum increase of 0.80 for the HighSeed simulation (baywide 39% seed fishery) for the population 4 allele during the 1970s to a decrease of –0.33 for the Seed3 simulation (single-population 30% seed fishery) for the population 3 allele during the 2000s (Figs. 4, 5).

Simulations based on each of the two time periods showed distinct differences in the change in allele frequency in the metapopulation over time for the range of simulated conditions (Figs. 4, 5). The base case (no fishing) simulations generally showed the opposite pattern in the 1970s versus the 2000s (black bars in Figs. 4, 5). In the 1970s simulations, alleles initially present in populations 1 and 2 decreased in frequency in the metapopulation over time, while those in populations 3 and 4 increased in fre-

Fig. 3. Allele frequency over time for the neutral allele marker in the 2000s. Columns from left to right show the influence of fishing on the frequency of the neutral allele initiated in population 1 with fishing in populations 1 through 4. Upper panels show allele frequencies under sack fishing; lower panels show allele frequencies under seed fishing. Lines show the allele frequency within each individual population (shown in different shades of grey over time; solid lines indicate base case, dashed lines indicate lowest fishing rate, and dotted lines indicate highest fishing rate. The shaded areas show the allele frequency in the metapopulation, overlaid with the darkest color representing the base case, medium indicating the lowest fishing, and lightest indicating highest fishing.

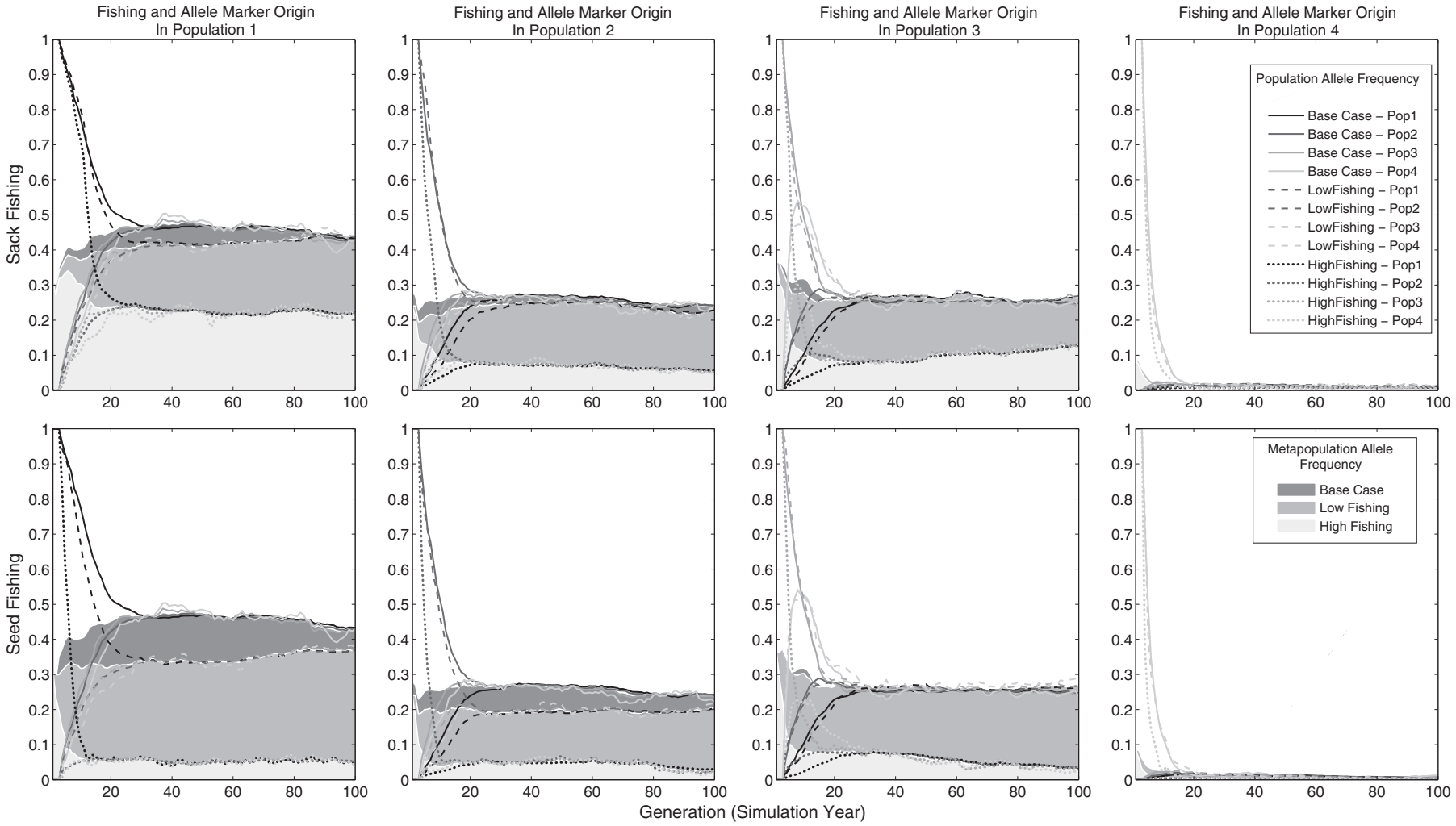
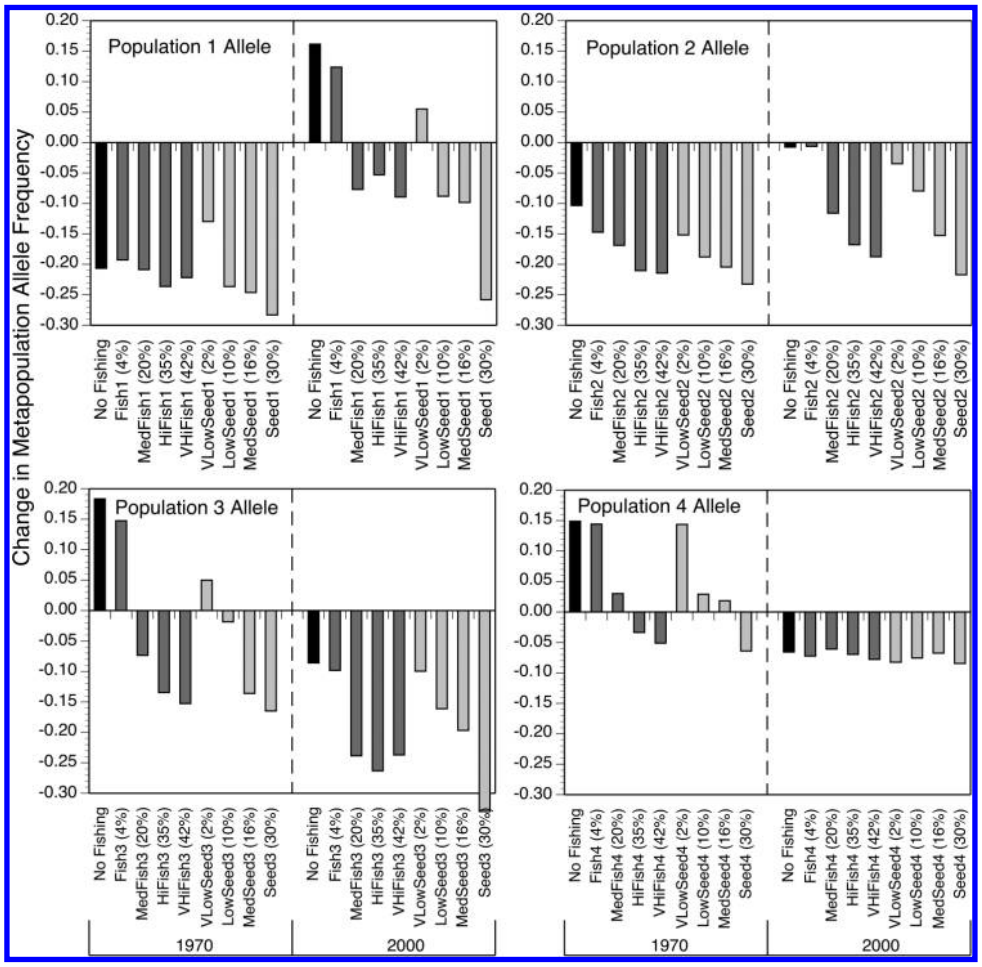


Fig. 4. Change in simulated allele frequency in the metapopulation for the marker allele (the neutral *B* allele originally fixed in the indicated population, but absent from the remaining three) for each of the four populations over 100 generations for fisheries restricted to a single population (only one of the four regions shown in Fig. 1). Black bar indicates the base case (no fishing) for each time period. Dark grey bars indicate sack fishery simulations; light grey bars indicate seed fishery simulations. For all fishing simulations, each panel shows the population for which the fishery was colocated in the population with the neutral marker allele source (all animals in the population *BB* initially). *X* axes labels are defined in Table 2; regime characteristics are defined in Table 1.



quency. The opposite pattern was evident in the 2000s simulations (see also Munroe et al. 2012 for further discussion of the influence of regime shift on genetic connectivity).

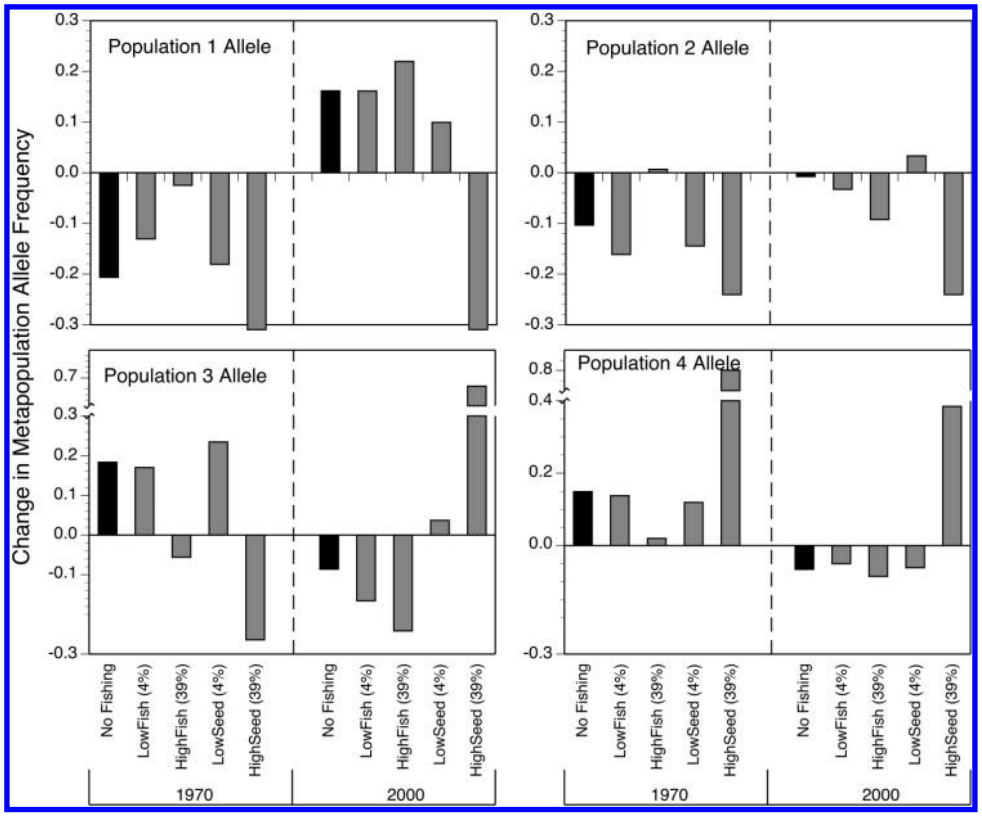
Single-population fishing

With the exception of two simulations (Fish1 and VLowSeed1 in the 1970s and Fish2 and MedFish4 in the 2000s), all simulations in which fisheries are limited to a single population (one of the four areas shown in Fig. 1), fishing a population generates a more negative change in allele frequency within the metapopulation relative to the base case, in which no fishing occurs (solid black bars, Fig. 4). In general, higher fishing pressure creates a larger difference in allele frequency within the metapopulation when compared with the no-fishing base case regardless of the type of fishery (Fig. 4). With some exceptions (VHiFish1 in 1970; HiFish1, VHifish3, Fish4, and all Seed4 simulations in 2000), increasing fishing pressure generates a stepwise decrease in the change in metapopulation allele frequency for a neutral allele originally present within the fished populations. Within each time period and for each fished population, the largest decrease in metapopulation allele frequency is observed for the highest fishing fraction for the seed fishery (SeedX at 30%, Fig. 5).

Paired comparisons

A total of 19 seed–sack pairs of simulations were included in the pairwise comparisons. Pairs were chosen such that no significant difference existed between the fishing mortality rates (F) for the pairs ($p = 0.56$, $t = 0.59$, df = 18; Fig. 6). The difference between the change in metapopulation allele frequency for the fishing simulation compared with the nonfishing base case was significantly different between seed and sack fishing. On average for these pairs, in which fishing mortality (F), population fished, and regime are held constant, seed fishing results in a 3.5% lower allele frequency compared with sack fishing. A one-sided paired t test showed that the difference was greater (generating a more negative value) for seed fishing than it was for sack fishing ($p = 0.025$, $t = -2.1$, df = 18; Fig. 7). Initially, in each simulation, the *B* allele is located in one population only, and thus a decrease in the metapopulation frequency over time indicates a loss of that allele from that population and (or) failure to export that allele to other populations. Seed fishing leads to a greater decrease in metapopulation *B* allele frequency for neutral alleles, effectively a greater decrease in export of the *B* allele from its single population source, relative to a nonfishing scenario than sack fishing, when fishing mortality (F), population, and regime are held constant. Further, for a given fished population contributing relatively

Fig. 5. Change in simulated allele frequency in the metapopulation for the marker allele (the neutral *B* allele originally fixed in the indicated population, but absent from the remaining three) for each of the four populations over 100 generations for fisheries covering the whole stock (fishing allowed in all four regions; Fig. 1). Black bar indicates the base case (no fishing) for each time period. Grey bars indicate stock-wide fishery simulations. *X* axies labels are defined in Table 2; regime characteristics are defined in Table 1.



more or less to the total abundance of the metapopulation and for a given fishing mortality rate (*F*), seed fishing tends to cause a greater decrease in the *B* allele frequency, engendered by a greater reduction in export of the *B* allele, than sack fishing. This trend is illustrated in Fig. 8; looking horizontally across the two panels, the left (sack) panel has lighter contours than the equivalent abundance on the right (seed) panel, indicating a lower allele frequency of the *B* allele at the end of the simulation. This trend is generally true regardless of the proportional contribution of the fished population to the entire metapopulation.

Comparing these paired values, we see that seed fishing ranges from a 6.3% higher allele frequency for population 1 in the 1970s to a 19.8% lower allele frequency for population 2 in the 2000s, a span of 26.1%, whereas the interquartile range was only 6.5%. Thus, a few simulation pairs contributed much of the range in effect (Fig. 7). A single simulation pair generated an increase in allele frequency from the fished population (1970 population 1, shown with ⊙ in Fig. 7); in this simulation FishFrac was set at 0.02 (2% annually) in the seed fishery (VLowSeed1, Table 2) and 0.04 (4% annually) in the sack fishery (Fish1, Table 2), while natural adult mortality was set at 11% for all populations (1970s parameters, Table 1).

Spawning potential ratio (SPR) shows a decreasing trend with increasing fishing mortality rate for both types of fisheries (Fig. 9). For 16 out of 19 pairs, a sack fishery generates a higher SPR than a seed fishery. On average, over all 19 pairs, a sack fishery generates an SPR that is 0.12 greater than that of a seed fishery when fishing mortality (*F*), population, and regime are held constant.

Whole-stock fishing

The impact of stock-wide fishing on the change in frequency of a neutral allele initiated in a given population is less consistent

Fig. 6. Instantaneous fishing mortality rates (*F*) for paired seed–sack simulations. Dark bars show the computed *F* for seed simulations; light bars show the computed *F* for sack simulations. Pairs of bars are consistent for regime and population fished as indicated by the *X* axis label. Results of the paired *t* test showing no significant difference in *F* between pairs are shown in the top left inset.

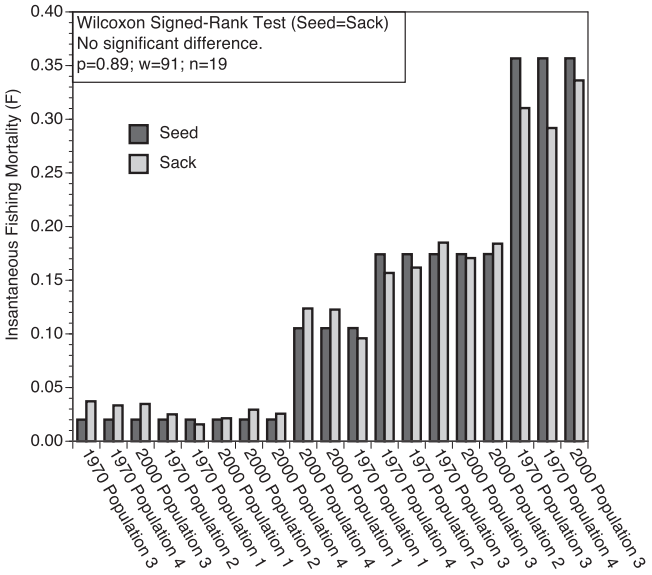
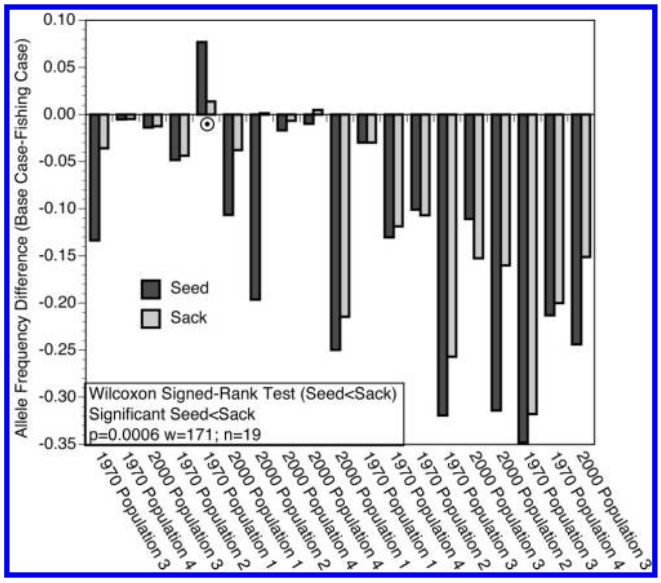


Fig. 7. Difference in allele frequency compared with the base case (base case change in allele frequency minus fishing case change in allele frequency) for paired seed–sack simulations. Dark bars show the allele frequency difference for seed simulations; light bars show the allele frequency difference for sack simulations. Pairs of bars hold regime and population fished consistent as indicated by the *X* axis label. Results of the paired *t* test showing that seed simulation allele frequency is significantly less than sack simulation allele frequency are shown in the bottom left inset. The only pair to generate an increase in allele frequency from the fished population (1970 population 1) is indicated (⊙).



than that for a single population fishery colocated with the source of the neutral allele (Fig. 5). Sack fishing, at both 4% and 39%, decreases the frequency of the neutral *B* allele in the metapopulation compared with that in the nonfishing base case for all source locations for the *B* allele marker, with the exception of the case where the *BB* marker was initially present in population 1. Relative to the nonfishing base case, seed fishing decreases the frequency of the allele marker if it originated from the upbay populations (populations 1 and 2), yet increases the frequency of the allele marker if it originated from the downbay populations (populations 3 and 4) relative to the nonfishing case. In all whole-stock fishing simulations, the greatest deviation for each marker allele in each regime, either positive or negative, from the nonfishing base case is observed for the HighSeed simulation, indicating that a high rate of seed fishing has the greatest potential to alter genetic connectivity.

Discussion

We examined the possible influence of an oyster fishery on genetic connectivity in a metapopulation using an individual-based numerical model capable of resolving the population in terms of the genotypes of its constituent individuals. We used simulations that included a range of realistic seed and sack fishing pressures from the literature (Rothschild et al. 1994; Jordan et al. 2002; Jordan and Coakley 2004; Powell et al. 2008; Harding et al. 2010) and spatially and temporally explicit demographic parameters from a well-studied oyster metapopulation in Delaware Bay (Kraeuter et al. 2007; Powell et al. 2009; Bushek et al. 2012; Haskin Shellfish Research Laboratory 2012). Simulations covered a range of possible fishing scenarios, including fisheries restricted to single areas and stock-wide fisheries, and used movement of a neutral allele marker as a proxy for genetic connectivity. These simulations demonstrate the ability of fishing to influence genetic connectivity among fished populations of oysters. In general, increasing fishing pressure tends to decrease the neutral allele output potential of the fished stock. This result is true for both seed and sack fisheries; however, seed fisheries diminish the export potential of neutral alleles more than a comparable sack fishery.

The trend generated by our simulations, that increasing fishing pressure tends to decrease the genetic output potential of the fished population, is supported by empirical studies. In one example, Miller et al. (2009) showed that a population of abalone that had collapsed owing to high fishing pressure had higher genetic diversity than comparable populations under lower fishery pressure. The authors originally hypothesized the opposite outcome, that high fishing pressure leading to a crashed population should result in reduced genetic diversity. They suggested that this counterintuitive result stems from the diminished capacity of the heavily fished population to produce local recruits, thus allowing genetically diverse immigrant recruits to enter the population. This agrees with our results, which suggest that fishing pressure tends to reduce the ability of a population to export alleles.

The difference in allele frequency in the simulation output between seed and sack fishing (Fig. 7) should depend on life history characteristics such as spawning frequency and maturation timing. For the oyster stock used for model parameterization here, oysters spawn only once in a season; this is true for oysters from Delaware Bay. The species has the ability to spawn twice in a season if conditions are appropriate, an event that is commonplace for oyster stocks in the Gulf of Mexico (Hopkins 1954; Hayes and Menzel 1981; Hofmann et al. 1994). It is possible that this increased spawning frequency could accentuate the difference between seed and sack fishing on allele frequencies because a fishery with a lower size limit (sack fishing) may allow more spawning events to occur before animals are subject to fishing pressure. Conversely, oysters from the Gulf of Mexico also experience faster growth rates owing to the higher temperatures and abundance of food (Butler 1952; Hayes and Menzel 1981) that would allow them to grow into fishable size classes faster, thereby eliminating the additional spawning events that would otherwise widen the gap between allele frequencies in populations harvested with seed versus sack fisheries.

In addition to examining how other oyster fishery demographics from other regions might influence population connectivity, this model could be applied to other fished stocks wherein fishing has possibly acted in a selective manner (Heino and Dieckmann 2008; Sharpe and Hendry 2009). As an example, an extensive 90-year Norwegian dataset documents reduction in variability in body size in Atlantic cod (Olsen et al. 2009). Similarly, a nearly 60-year dataset demonstrates trends in size and age-at-maturity in sockeye salmon from Alaska (Kendall et al. 2009). Assuming that the observed changes in phenotype are a result of changes in gene frequency, these long-term datasets, along with fishery data for those stocks, could be used in a model like this one to examine potential drivers and the role of ecological characteristics for these documented phenotypic changes.

Arguably, the impact of the fishery should scale with the proportion of the stock exploited. In these simulations, for high rates of instantaneous fishing mortality ($F > 0.3$), as the proportion of the metapopulation subjected to fishing pressure increases, allele frequency of the marker allele decreases sharply (Fig. 8). However, at intermediate fishing mortalities ($0.1 < F < 0.25$), the decrease in frequency of the marker allele gets stronger as the proportion of the metapopulation in the fishery increases up to a point. Then at a point where the source population for the marker allele contributes approximately 25% to metapopulation abundance, the decrease in marker allele frequency begins to weaken. This transition point holds for both the seed and sack fisheries and implies that a fishery utilizing 25% or more of the overall abundance of the stock begins to spread over a large enough proportion of the population to compensate slightly for the neutral allele frequency

Fig. 8. Contour plots of the difference in allele frequency compared with the base case (base case change in allele frequency minus fishing case change in allele frequency) over the range of instantaneous fishing mortality rates (*F*) and percentage of metapopulation abundance for all simulations where fishing was restricted to a single population. Left panel shows sack fishing simulations; right panel shows seed fishing simulations.

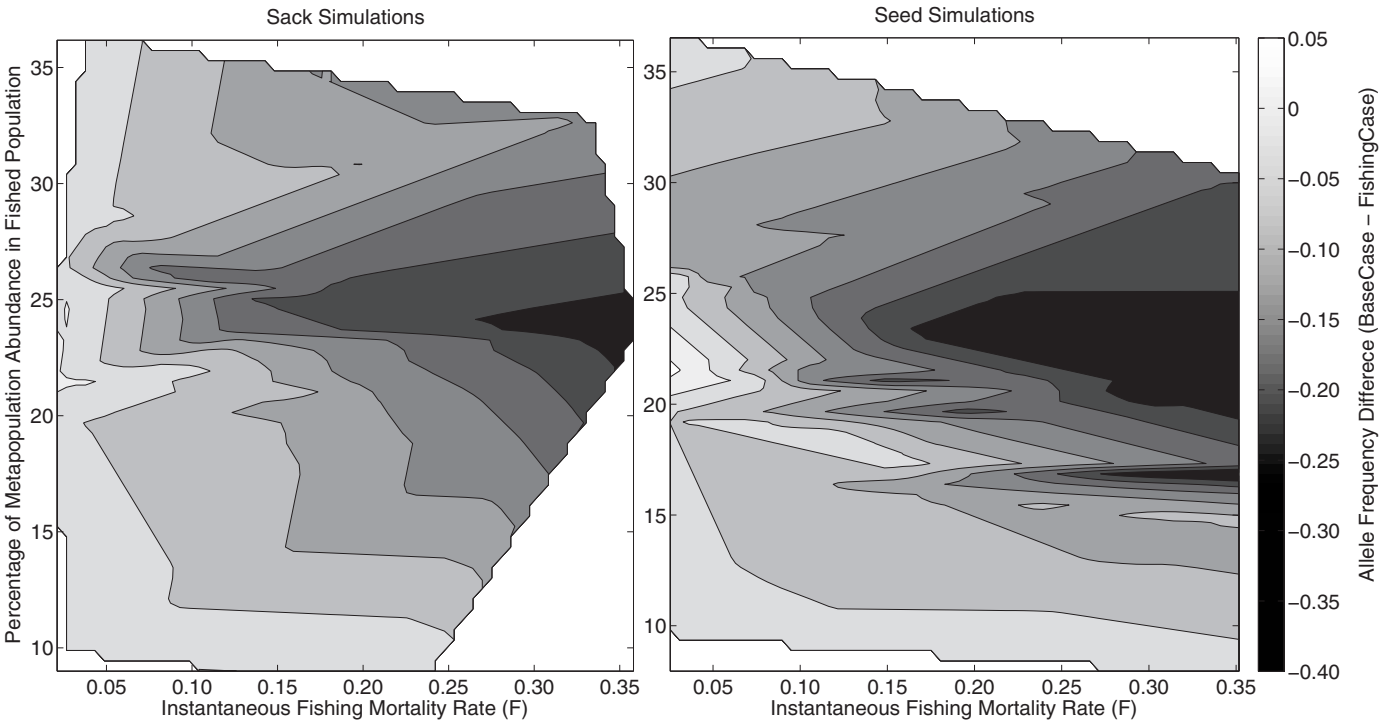
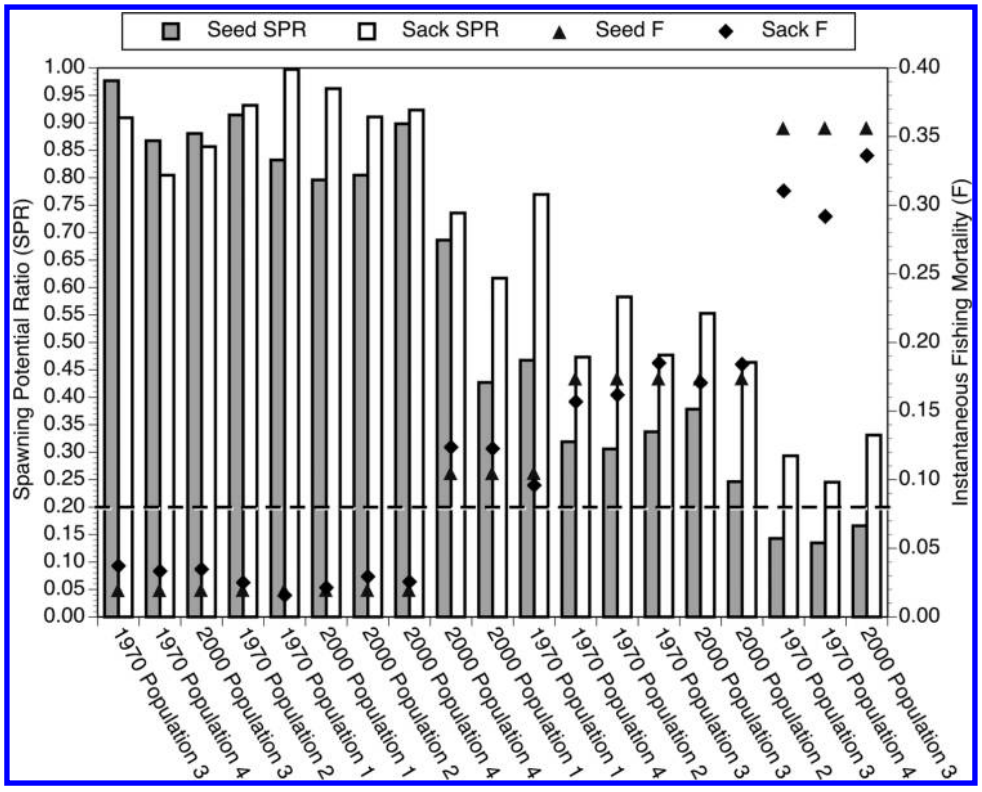


Fig. 9. Spawning potential ratio (SPR) and instantaneous fishing mortality rate (*F*) for paired seed–sack simulations. Shaded bars show the SPR for seed simulations; open bars show the SPR for sack simulations. Pairs of bars are consistent for regime, population fished, and instantaneous fishing mortality rate as indicated by the *X* axis label. Solid triangles show *F* for seed simulations; solid diamonds show *F* for the sack simulations. Horizontal dashed line delineates SPR = 0.2.



depression created when a small portion of the stock is fished. It should be noted that this compensation is not very steep; at F = 0.2, going from 25% to 30% of the stock fished generates an approximate 10% increase in allele frequency (Fig. 8).

Implications for management

The differential influence of seed and sack fisheries on genetic connectivity implies that seed fisheries should be managed in a more conservative manner than a comparable sack fishery. Spawning potential ratio is a metric often used to assess the relationship of the exploitation rate to sustainability (Goodyear 1993). Consider, for example, a scenario in which a certain population is the source of genotypes that are unique and (or) valuable to the metapopulation. A seed fishery with an instantaneous fishing mortality of around 0.10 generates a 0.05–0.30 lower SPR in comparison to a sack fishery with the same fishing mortality (Fig. 9).

Brooks et al. (2010) argue that a minimum reference SPR above which overfishing is not occurring and which is applicable across all fisheries cannot be defined; instead, species-specific life histories must be considered to determine an appropriate cutoff on a species and fishery basis. Nonetheless, the authors demonstrate that an SPR of 0.3 is likely suitable for short-lived, early maturing species like oysters. For the series of paired simulations performed here, seven generated a mean SPR ≤ 0.3; five of those seven were seed fishing simulations. In addition, seed fishing scenarios were the only simulations that generated an SPR < 0.2, well below the SPR level identified by Brooks et al. (2010), and therefore likely to create overfishing conditions (Fig. 9). The depression of the SPR by the seed fishery relative to a sack fishery means that seed fisheries have greater potential to limit the exchange of alleles from the source location, and this is likely the origin of the lower rate of allele export observed in the simulations (Fig. 7). By implication, fishing mortality targets for seed fisheries should be set conservatively relative to the limits set for fisheries with a defined size limit.

The change in marker allele frequency between paired seed and sack simulations covers a range of 26.1%. This range includes one pair that generated an increase in allele frequency from the fished population (1970 population 1, shown with a ⊙ in Fig. 7). This pair of simulations was calculated with FishFrac set at 0.02 (2% of the stock annually) in the seed fishery (VLowSeed1, Table 2) and 0.04 (4% of the stock annually) in the sack fishery (Fish1, Table 2), while natural adult mortality was set at 11% for all populations (1970s parameters, Table 1). Thus, fishing rates were well below the natural mortality rates. A possible explanation for this counterintuitive increase in allele frequency under fishing in these two simulations is that the influence of the fishery is negligible in comparison to the influence of the natural mortality rates on genetic connectivity. This highlights the importance of understanding natural mortality rates in fished populations and considering fishing mortality relative to natural mortality.

Another important management consideration highlighted in these simulations is the temporal variability of locations of genetic sources and sinks in Delaware Bay oysters. A temporally dynamic (or adaptively managed) fishery that allows fishery location and rates to respond to stock movement over time is intuitive for mobile resources like migratory fish (Game et al. 2009). Less intuitive is the need for temporally dynamic management for sessile species like oysters. Regime shifts are characterized by a sudden, rapid shift in a biological community (Scheffer and Carpenter 2003; Weijerman et al. 2005) and have been observed commonly in the temporal population dynamics of many marine organisms (Collie et al. 2004; Rothschild and Shannon 2004; Powell et al. 2009). The simulations performed here compared the dynamics of oyster populations under two separate regimes. Differences in simulated source and sink characteristics during these two time periods have implications for the effects of the fishery on genetic connectivity. For example, population 1 in Fig. 5 (upper left panel) acts as a sink for alleles in the 1970s regime, and continues to operate as a sink under fishing pressure. In the 2000s regime, this population shifts to an allele source and remains a source under all but the highest stock-wide fishing mortality (HighSeed). Thus, our results show that regime shifts can alter locations of sources and sinks (see also Munroe et al. 2012) and in doing so can change the influence of fisheries on genetic connectivity over time. Given the importance of local adaptation in many marine species (Sanford and Kelly 2011), the dynamics of changing source and sink may promote or impede the long-term benefits of allele transfer within the metapopulation and allele retention within the local population. The potential for changes in metapopulation connectivity over time should be integrated into fisheries planning and adaptive strategies should be implemented, allowing managers to respond to such changes.

Managers are increasingly being called upon to bring evolutionary principles into management (Hendry et al. 2011). This influence of fisheries on genetic connectivity is an important consideration for management of the evolution of fished stocks. Our simulations show that certain combinations of proportional contribution by a local population to metapopulation abundance and local fishing pressure may alter genotype diversity much more than others. Jørgensen et al. (2007) propose evolutionary impact assessment as an important component of fisheries management, allowing managers to evaluate and mitigate the consequences of fisheries-induced evolution. Our simulations suggest that potential reductions in genetic output from fished populations resulting from fishing mortality, and the way that affects ecological and evolutionary impacts of fishing, should be brought to bear in evolutionary impact assessment. An important caution exists concerning the outputs discussed here in relation to evolutionary impacts of fishing. The simulations performed here use neutral alleles only as markers to track genetic connectivity, and thus selection does not play a role in determining the simulated allele frequencies. Fisheries have been shown to be able to generate changes in genotype, and thus changes in population allele frequency at specific loci, through selective processes (Allendorf et al. 2008; Hard et al. 2008; Heino and Dieckmann 2008; Hutchings and Fraser 2008; Dunlop et al. 2009; Sharpe and Hendry 2009). Our simulations demonstrate that fishing mortality also has the potential to alter the dynamics of neutral allele frequencies among populations. Oysters and some other shellfish are highly polymorphic (Launey and Hedgecock 2001; Zhang and Guo 2010; Wang et al. 2010), and maintenance of this polymorphy is likely to include the transfer of many neutral alleles and should be impacted by fishing as it interacts with genetic connectivity. In these simulations, we have not tested how selective forces such as selective fishing or disease mortality interact or potentially counteract changes demonstrated here in neutral allele frequency made by fishing mortality. This interaction of selective forces with demographically driven changes in genetic connectivity is an important consideration and one that we are currently examining in ongoing research.

Fishing and evolution of disease resistance

The decline of the eastern oyster is in part a result of overfishing (Rothschild et al. 1994; Wilberg et al. 2011). Overfishing in other species has been associated with impacts on genetic variability of the stock (Law 2007; Olsen et al. 2009; Perry et al. 2010); however, evidence that overfishing may have impacted the genetic variability of eastern oyster stocks has not yet been identified. Our simulations demonstrate that heavy fishing on a small portion of the stock in Delaware Bay could potentially reduce the genetic connectivity of that population to the overall stock. This could lead to reduction in those genotypes over time from the metapopulation. This is problematic if those genotypes are valuable now or in the future. Alternatively, if those genotypes have low fitness in other

populations, it could be beneficial to the metapopulation if those genotypes are reduced in frequency.

In Delaware Bay, development of resistance to MSX (*Haplosporidium nelsoni*) in the native population is nearly complete (Ford and Bushek 2012). Development of MSX resistance has been slower, but not inconsequential, in the Chesapeake Bay (Carnegie and Burreson 2011). Development of resistance to Dermo (*Perkinsus marinus*) has been much slower, although some evidence suggests that the process may be ongoing (Powell et al. 2011*c*, 2012). The severity of these two diseases increases with increasing salinity, thus generating an along-estuary mortality gradient that follows the salinity gradient (e.g., Bushek et al. 2012; Ford et al. 1999). In Delaware Bay, this gradient generates a downbay drift of genes from source populations upbay (Munroe et al. 2012). Our simulations agree with the conclusions of Munroe et al. (2012), who found that upbay populations are likely to be source populations under current conditions. Ford and Bushek (2012) show that these populations function as refuges for disease-susceptible genotypes. Thus, our simulations suggest that the mortality gradient generated by disease not only protects susceptible genotypes upbay, but also facilitates their continual importation into downbay populations, thereby restricting the development of resistance to disease. Fishing these upbay populations could reduce their genetic output and facilitate evolution of disease resistance in the metapopulation. This suggestion must be considered within the context of differential population dynamics such as slower growth rates in these upbay (lower salinity) populations and how fishing those populations might influence population persistence.

Conclusions

Incorporation of evolutionary impacts on population genetics is a developing priority in the management of marine resources (Bert et al. 2011; Hendry et al. 2011; Jørgensen et al. 2007). Many of the approaches used to study evolutionary impacts of fishing focus on selective processes in which the fishery changes population genetics through the selection of certain phenotypes (Hard et al. 2008; Hutchings and Fraser 2008; Sattar et al. 2008). Results of simulations conducted in the present study indicate that nonselective fishing can also alter the underlying processes of evolution, namely genetic connectivity. Importantly, in the case of oyster fisheries, seed fisheries (without a lower size limit), have a greater potential to alter genetic connectivity than sack fisheries (with a lower size limit). Consequently, it is essential for the influence of fisheries on source–sink dynamics to be incorporated in the future management of marine populations, as it relates to the ability of a stock to evolve and maintain genetic diversity. Our results emphasize the need to manage a seed fishery more conservatively than a sack fishery. Trends in SPR illustrate the sensitivity of genetic connectivity to the removal of seed.

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