Maintenance of genetic polymorphism under conditions of genotype-dependent growth and size-selective mortality

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Associations between heterozygosity at one or more electrophoretically detected enzyme loci and growth rate have been reported for several species of plants and animals, including several commercially important species of finfish and shellfish. The general pattern is for heterozygotes to grow faster than homozygotes, although there is some variation in growth response even within a species. Regardless of the physiological or biochemical basis of genotype-dependent growth, polymorphism at a locus affecting growth rate in an overdominant manner may be lost if larger individuals have a greater mortality rate than smaller ones. In an exploited population, mortality of this sort is likely to result from size-selective fishing pressure. Using a continuous-time single-locus model of natural selection, we have related the maintenance of polymorphism at a locus to two measures of fishing effort: β, the legal minimum size below which there is no mortality, and f, an instantaneous mortality rate owing to fishing (above the legal minimum size). We considered two different models of fishing mortality. In model 1, fishing mortality above the legal minimum size is constant; in model 2, fishing mortality is a linear function of size (above β). Numerical analysis of model 1 indicates that maintenance of polymorphism requires either a low rate of fishing mortality or a value of β that is close to zero or close to the maximum attainable size. Analysis of model 2 gives similar results, suggesting that the conclusions are not dependent on the exact form of the mortality function.

Key words: heterozygosity, growth, size, mortality.

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Des corrélations entre l'hétérozygosité sur un ou plusieurs locus (loci) d'enzyme détecté par électrophorèse et la vitesse de croissance ont été rapportées chez plusieurs espèces de plantes et d'animaux, y inclus plusieurs espèces de poissons à nageoires et de poissons à carapace d'importance commerciale. La tendance générale est que les hétérozygotes croissent plus rapidement que les homozygotes bien qu'on trouve une certaine variation de croissance même à l'intérieur d'une espèce. Si on fait abstraction de la base physiologique ou biochimique de la croissance liée au génotype, le polymorphisme sur un locus affectant la vitesse de croissance d'une façon surdominante, peut disparaître si les individus plus gros ont un taux de mortalité plus élevé que les plus petits. Chez une population exploitée, ce type de mortalité peut survenir de pressions de pêches sélectives quant à la taille. À l'aide d'un modèle en temps continu et à locus unique de sélection naturelle, nous avons établi un lien entre le maintien du polymorphisme sur un locus et deux mesures liées à la pêche: β, la taille minimum légale en deçà de laquelle il n'y a pas de mortalité, et f, un taux de mortalité instantanée dû à la pêche (au delà de la taille minimum légale). Nous avons étudié deux modèles différents de mortalité dûe à la pêche. Dans le modèle 1, la mortalité dûe à la pêche de spécimens au delà de la taille minimum légale est constante; dans le modèle 2, la mortalité dûe à la pêche est une fonction linéaire de la taille (au delà de β). L'analyse numérique du modèle 1 indique que le maintien du polymorphisme requiert ou bien un faible taux de mortalité dûe à la pêche, ou bien une valeur de β qui est proche de zéro ou près de la taille maximum possible. L'analyse du modèle 2 donne des résultats semblables, ce qui suggère que les conclusions ne dépendent pas de la forme exacte de la fonction mortalité.

Mots clés: hétérozygosité, croissance, taille, mortalité.

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Introduction

The importance of maintaining genetic variation in populations of commercially important organisms can be approached from two viewpoints. First, preservation of genetic variation is necessary to provide a substrate for future selection, either natural or artificial (Franklin 1980). Second, to the extent that heterozygosity is related to such traits as growth, fecundity, and survival, maintenance of genetic variation is necessary to avoid decreasing the mean fitness of the population (Soulé 1980; Smith and Chesser 1981). Previous research on the genetic consequences of wildlife management techniques has emphasized the loss of heterozygosity through random drift in populations whose sizes are reduced by habitat destruction (Franklin 1980) or hunting (Ryman et al. 1981).

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Here, we demonstrate that under certain management regimes polymorphism at a locus may be lost even in populations of effectively infinite size if two conditions are met. First, mortality is size selective as a result of hunting or fishing (in particular, larger individuals must have a greater mortality rate than smaller ones). Second, growth rate is genotype dependent so that heterozygous individuals grow faster than homozygous ones. There is an extensive literature on size-selective mortality in populations of commercially important species of finfish and shellfish (Ricker 1969), which suggests that the first condition is often imposed on the population. In exploited populations, in particular, size-selective mortality can result from harvesting techniques that involve netting or dredging. In such cases, the probability of being caught is proportional to size. Data relevant to the second condition, genotype-dependent growth, have accumulated rapidly in the last 5 years. In several species, heterozygotes appear to grow faster than homozygotes, although there is considerable variation in growth response in relation to heterozygosity even among populations within species (e.g., Ledig et al. 1983; Garton and Stickle 1984; Reinitz 1977; Pierce and Mitton 1982). At least 16 species have been reported to show genotype-dependent growth rate: the list includes plants (*Liatris cylindracea*, Schaal and Levin 1976; *Pinus rigida*, Ledig et al. 1983; *Populus tremuloides*, Mitton et al. 1981), a polychaete worm (*Hyalinoceia tubicola*, Manwell and Baker 1982), bivalve molluscs (*Macoma balthica*, Green et al. 1983; *Mytilus californianus*, Levinton and Fundiller 1975), gastropod molluscs (*Haliotis discus*, Fujino 1978; *Thais haemastoma*, Garton 1983; *T. lamellosa*, Garton and Stickle 1984), fish (*Gambusia affinis*, Smith and Chesser 1981; *Salmo gairdneri*, Reinitz 1977; *Pagrus major*, Taniguchi et al. 1981), other vertebrates (*Ambystoma tigrinum*, Pierce and Mitton 1982; *Ovis aries*, Baker and Manwell 1977), and humans (Bottini et al. 1979).

These studies are characterized by a variety of experimental designs, so the results are not readily comparable. However, the overall pattern is consistent with the suggestion that heterozygosity is associated with increased rates of growth, although the explanation for this phenomenon need not be the same in all cases.

The species that has been studied most extensively in this regard is the American oyster, *Crassostrea virginica* (Singh and Zouros 1978, 1981; Zouros et al. 1980; Singh 1982). In this species, heterozygotes for five of seven polymorphic enzyme loci were significantly heavier at 1 year of age than were homozygotes. Multilocus heterozygosity explained about 4% of the total variance in body weight (Foltz et al. 1983). Koehn and Shumway (1982) have provided a physiological explanation for genotype-dependent growth rates in the American oyster in terms of differential oxygen consumption (heterozygotes have lower oxygen consumption weight than do homozygotes). Stanley et al. (1981) looked at the effect on growth in the American oyster of polyploidy induced by blocking meiosis with cytochalasin B. At 2 years of age, polyploids formed by blocking meiosis I were significantly heavier than polyploids formed by blocking meiosis II and also heavier than the diploid (control) oysters (Hidu et al. 1982). These results suggest that overall heterozygosity, rather than polyploidy per se, is the important factor affecting growth rate and provide independent confirmation of the work of Zouros et al. (1980). Additional support for the idea that heterozygous oysters grow faster than more homozygous ones is provided by the breeding experiments of Newkirk et al. (1977) and the interpopulation crosses of Mallet and Haley (1983).

Basic theory

The theory of selection in age-structured populations is described by Charlesworth (1980). The notation used in developing the model is explained in Table 1. Let $n_{ij}(x,t)$ be the number of individuals of genotype ij ($ij = 11, 12, 22$) of age x at time t. The demographic dymanics of the population can be described by a system of three partial differential equations:

$$[1] \quad \frac{\partial n_{ij}}{\partial x} + \frac{\partial n_{ij}}{\partial t} + n_{ij}(x,t)\mu_{ij}(x) = 0 \qquad i,j = 1,2$$

where $\mu_{ij}(x)$ is the age-dependent mortality rate. Equation 1 describes the ageing and death of individuals. Reproduction is described using a boundary condition for $x = 0$:

$$[2] \quad n(0,t) = \int_{x_0}^{\infty} (m_{11}(x,t)n_{11}(x,t) + m_{12}(x,t)n_{12}(x,t) + m_{22}(x,t)n_{22}(x,t))\mathrm{d}x$$

where $n(0,t) = n_{11}(0,t) + n_{12}(0,t) + n_{22}(0,t)$, $m_{ij}(x,t)$ is the expected number of offspring for genotype ij at age x and time t, and x_0 is the age at which reproduction begins. Charlesworth (1980, p. 126) lists some of the assumptions of this formulation.

Let $p(t)$ be the frequency of A_1 alleles among newly formed zygotes. It can be computed using

$$[3] \quad p(t) = \left\{\int_{x_0}^{\infty} \left(m_{11}(x,t)n_{11}(x,t) + \frac{1}{2}m_{12}(x,t)n_{12}(x,t)\right)\mathrm{d}x\right\} \Big/ n(0,t)$$

With random union of gametes

$$[4] \quad \begin{aligned} n_{11}(0,t) &= p^2(t)n(0,t) \\ n_{12}(0,t) &= 2p(t)(1 - p(t))n(0,t) \\ n_{22}(0,t) &= (1 - p(t))^2 n(0,t) \end{aligned}$$

TABLE 1. Explanations of symbols used in the text

B_{ij}	Maximum size of an individual of genotype ij	$n_{ij}(x,t)$	Number of individuals of genotype ij at age x and time t
β	Legal minimum size	$n^*_{ij}(x)$	Equilibrium number of individuals of genotype ij at age x
$c(t)$	Recruitment rate per unit size per individual at time t	$n(x,t)$	Number of individuals at age x and time t, without regard to genotype
c^*	Equilibrium recruitment rate per unit size per individual	$n^*(x)$	Equilibrium number of individuals of age x, without regard to genotype
$f(y)$, $f_1(y)$, $f_2(y)$	Instantaneous mortality rates owing to fishing	Ω_{ij}	A measure of "aggregate size" of individuals of genotype ij
ij	Genotype (ij = 11, 12, 22)	$p(t)$	Frequency of A_1 alleles at age 0 and time t
K_{ij}	Instantaneous growth rate of an individual of genotype ij	p^*	Equilibrium frequency of A_1 alleles at age 0
$l(x)$	Probability of an individual surviving to age x, without regard to genotype	R^*_{ij}	Total number of offspring produced by individuals of genotype ij at equilibrium
$l_{ij}(x)$	Probability of an individual of genotype ij surviving to age x	$\bar{R}^*$	Average value of R^*_{ij} across genotypes
$l_{ij}(y)$	Probability of an individual of genotype ij surviving to size y	t	Time
$m_{ij}(x,t)$	Expected number of offspring for an individual of genotype ij at age x and time t	$W_{ij}(x)$	Size of an individual of genotype ij at age x
$m^*_{ij}(x)$	Equilibrium expected number of offspring for an individual of genotype ij at age x	$W^{(-1)}_{ij}(y)$	Age of an individual of genotype ij at size y
$\mu_{ij}(x)$	Mortality rate of an individual of genotype ij at age x	x	Age
		x_o	Age at which reproduction begins
		$\tilde{x}_{ij}$	Age at which an individual of genotype ij attains legal minimum size (β)
		y	Size
		Z	Instantaneous natural mortality rate

Genotype-dependent growth

Let the variable y represent size. Growth will be based upon the following gompertz function:

$$[5] \quad y = W_{ij}(x) = B_{ij}(1 - e^{-K_{ij}(x - x_o)}) \qquad x \geq x_o$$

where $W_{ij}(x)$ is the size of an individual of genotype ij and age x, B_{ij} is the maximum size of an individual of genotype ij, K_{ij} is the growth rate of an individual of genotype ij, and x_o is the age at which size is taken to be 0, also age at which reproduction begins. The inverse of the growth function gives an individual's age based on its size:

$$[6] \quad x = W^{(-1)}_{ij}(y) = x_o - K^{-1}_{ij} \ln (1 - (y/B_{ij})) \qquad y \geq 0$$

It should be noted that [5] and [6] are based on the assumption that, prior to age x_o, all genotypes are identical in size.

Size-specific recruitment

Assume that the expected number of offspring for an individual is proportional to its size, irrespective of genotype. Furthermore, assume that the probability that a zygote survives long enough to be counted as a recruit of age x_o is equal to the proportion of individuals lost owing to mortality. In other words, the total number of new recruits balances losses owing to mortality, resulting in a stable population size. Let

[7] $c(t) \equiv$ (expected number of zygotes per unit size per individual) · (probability of a zygote becoming a new recruit)

With this definition we can write

$$[8] \quad m_{ij}(x,t) \begin{cases} = 0 & x < x_o \\ = c(t)W_{ij}(x) & x \geq x_o \end{cases}$$

for the expected number of offspring. This assumption is valid for situations in which reproduction depends on body size or gonad size rather than age.

Size-specific survivorship

First, consider the age-specific mortality rate $\mu_{ij}(x)$. We will assume that there are two sources of mortality: (*i*) a natural mortality rate Z which is independent of size and age, and (*ii*) a fishing mortality rate $f(y)$ which may depend on size. We will also assume that there exists a legal minimum size β below which there is no fishing mortality. To simplify the analysis in terms of age, define

$$[9] \quad \tilde{x}_{ij} = W^{(-1)}_{ij}(\beta) = x_o - K^{-1}_{ij} \ln (1 - (\beta/B_{ij}))$$

where $\tilde{x}_{ij}$ is the age at which individuals of genotype ij attain legal minimum size. Now we can write the mortality rate as a function of age:

$$[10]\quad \mu_{ij}(x)\begin{cases} = Z & 0 \le x < \tilde{x}_{ij} \\ = Z + f(y) = Z + f(W_{ij}(x)) & x \ge \tilde{x}_{ij} \end{cases}$$

To obtain an explicit model, it is necessary to choose an explicit form for the function $f(y)$. We will consider two simple forms for the function $f(y)$, which will be referred to as model 1 and model 2. In model 1, we assume that fishing mortality above the legal size is a constant. One can imagine this situation as being analogous to an idealized situation of net fishing where all individuals below legal size ("mesh size of the net") escape. The fishing mortality rate is analogous to the instantaneous probability of being netted. For model 2, we assume size-dependent mortality, specifically, that the instantaneous probability of being captured is proportional to size for individuals above the legal minimum. The fishing mortality rates are

$$[11]\quad f(y)\begin{cases} = f_1 & \text{for model 1} \\ = f_2 y & \text{for model 2} \end{cases}$$

where f_1 and f_2 are constants. Note that f_1 has units (time)$^{-1}$ and f_2 has units (size·time)$^{-1}$.

Now, we will describe the survivorship curves for each model. Survivorship is related to mortality rate by the expression

$$[12]\quad l(x) = \exp\left(\int_0^x \mu(\epsilon)d\epsilon\right) \qquad x \ge 0$$

Substituting [10] into [12] gives

$$[13]\quad l_{ij}(x) \begin{cases} = \exp\left(-\int_0^{\tilde{x}_{ij}} Z d\epsilon\right) & 0 \le x < \tilde{x}_{ij} \\ = \exp\left(-\int_0^{\tilde{x}_{ij}} Z d\epsilon - \int_{\tilde{x}_{ij}}^{\infty} (Z + f(W_{ij}(\epsilon))d\epsilon\right) & x \ge \tilde{x}_{ij} \end{cases}$$

for the genotype-specific survivorship function. Substituting [11] into [13] gives an explicit solution to the survivorship function for each model.

Model 1:

$$[14]\quad l_{ij}(x)\begin{cases} = \exp(-Zx) & 0 \le x < \tilde{x}_{ij} \\ = \exp(-Zx)\cdot\exp(-f_1(x - \tilde{x}_{ij})) & x \ge \tilde{x}_{ij} \end{cases}$$

Model 2:

$$[15]\quad l_{ij}(x)\begin{cases} = \exp(-Zx) & 0 \le x \le \tilde{x}_{ij} \\ = \exp(-Zx)\cdot\exp(-f_2 B_{ij}(x - \tilde{x}_{ij} + K_{ij}^{-1}e^{-K_{ij}(x - x_0)} - K_{ij}^{-1}e^{-K_{ij}(\tilde{x}_{ij} - x_0)})) & x \ge \tilde{x}_{ij} \end{cases}$$

Finally, it is possible to use [5] to express survivorship as a function of size for each model.

Model 1:

$$[16]\quad l_{ij}(y)\begin{cases} = e^{-Zx_0}(1 - (y/B_{ij}))^{K_{ij}^{-1}Z} & 0 \le y < \beta \\ = e^{-Zx_0}(1 - (y/B_{ij}))^{K_{ij}^{-1}(Z+f_1)}(1 - (\beta/B_{ij}))^{-K_{ij}^{-1}f_1} & \beta \le y \le B_{ij} \end{cases}$$

Model 2:

$$[17]\quad l_{ij}(y)\begin{cases} = e^{-Zx_0}(1 - (y/B_{ij}))^{K_{ij}^{-1}Z} & 0 \le y < \beta \\ = e^{-Zx_0}(1 - (y/B_{ij}))^{K_{ij}^{-1}Z}((B_{ij} - y)/(B_{ij} - \beta))^{K_{ij}^{-1}f_2 B_{ij}} e^{f_2 K_{ij}^{-1}(y - \beta)} & \beta \le y \le B_{ij} \end{cases}$$

Note that in both models, survivorship is a function of size *and* genotype.

Equilibrium

At genetic and demographic equilibrium, the genotype numbers, $n_{ij}(x,t)$, remain constant over time. Substituting $\partial n_{ij}/\partial t = 0$ into [1] and solving for the equilibrium age distribution, $n_{ij}^*(x)$, of each genotype yields

[18] $n^*_{ij}(x) = n^*_{ij}(0)l_{ij}(x), \qquad i,j = 1,2$

As one might expect, the age distribution is proportional to the survivorship curve when population size remains constant. Let $m^*_{ij}(x)$ be the age- and genotype-specific expected number of offspring at equilibrium. Equations 2, 3, and 4 become

$$[19] \quad n^*(0) = \int_{x_0}^{\infty} (m^*_{11}(x)n^*_{11}(x) + m^*_{12}(x)n^*_{12}(x) + m^*_{22}(x)n^*_{22}(x))dx$$

$$[20] \quad p^* = \left(\int_{x_0}^{\infty} \left(m^*_{11}(x)n^*_{11}(x) + \frac{1}{2}m^*_{12}(x)n^*_{12}(x)\right)dx\right) \Big/ n^*(0)$$

$$1 - p^* = \left(\int_{x_0}^{\infty} \left(\frac{1}{2}m^*_{12}(x)n^*_{12}(x) + m^*_{22}(x)n^*_{22}(x)\right)dx\right) \Big/ n^*(0)$$

and

$$[21] \quad n^*_{11}(0) = p^{*2}n^*(0)$$
$$n^*_{12}(0) = 2p^*(1 - p^*)n^*(0)$$
$$n^*_{22}(0) = (1 - p^*)^2 n^*(0)$$

Now, consider the total number of offspring produced by each genotype at equilibrium. This quantity is defined as

$$[22] \quad R^*_{ij} = \int_{x_0}^{\infty} m^*_{ij}(x)l_{ij}(x)dx \qquad i,j = 1,2$$

Substituting [21] into [18] and [18] into [20] gives

$$[23] \quad p^* = p^{*2}R^*_{11} + p^*(1 - p^*)R^*_{12}$$
$$(1 - p^*) = p^*(1 - p^*)R^*_{12} + (1 - p^*)^2 R^*_{22}$$

The system of two equations in [23] reduces to

$$[24] \quad p^*(R^*_{11} - R^*_{12}) + R^*_{12} - 1 = 0$$
$$p^*(R^*_{12} - R^*_{22}) + R^*_{22} - 1 = 0$$

These two equations hold true for all $0 < p^* < 1$ if $R^*_{11} = R^*_{12} = R^*_{22} = 1$. If the numbers of offspring from all three genotypes are not equal, then $R^*_{ij} \neq 1$ and the solution to [24] is

$$[25] \quad p^* = \frac{1 - R^*_{12}}{R^*_{11} - R^*_{12}} = \frac{1 - R^*_{22}}{R^*_{12} - R^*_{22}}$$

Recall from [8] that $m_{ij}(x,t) = c(t)W_{ij}(x)$. Because $m^*_{ij}(x)$ is constant through time, $c(t) = c^*$ where c^* is a constant. Therefore, we can write

$$[26] \quad R^*_{ij} = c^*\Omega_{ij}$$

where

$$\Omega_{ij} = \int_{x_0}^{\infty} W_{ij}(x)l_{ij}(x)dx$$

Substituting [26] into [25] and cross multiplying gives

$$[27] \quad c^*(\Omega_{12} - \Omega_{22}) - c^{*2}\Omega_{12}(\Omega_{12} - \Omega_{22}) - c^*(\Omega_{11} - \Omega_{12}) + c^{*2}\Omega_{22}(\Omega_{11} - \Omega_{12}) = 0$$

Solving [27] for c^*, one gets

$$[28] \quad c^* = \frac{2\Omega_{12} - \Omega_{11} - \Omega_{22}}{\Omega^2_{12} - \Omega_{11}\Omega_{22}}$$

Substituting [26] into [25], one can also write

$$[29] \quad p^* = \frac{(1/c^*) - \Omega_{22}}{\Omega_{12} - \Omega_{22}}$$

Using [28] in [29] and simplifying, one has

$$[30] \quad p^* = \frac{\Omega_{12} - \Omega_{22}}{2\Omega_{12} - \Omega_{11} - \Omega_{22}}$$

which is equivalent to the result obtained using the classical discrete generation formulation (Roughgarden 1979; Charlesworth 1980). Finally, consider the average value for the expected number of offspring per individual, which will be denoted by $\bar{R}^*$. It is calculated using

$$[31] \quad \bar{R}^* = p^{*2}R^*_{11} + 2p^*(1 - p^*)R^*_{12} + (1 - p^*)^2 R^*_{22}$$

Substituting [26], [28], and [30] into [31], it is easy to show that $\bar{R}^* = 1$. This result gives an alternative to [28] which is

$$[32] \quad 1/c^* = p^{*2}\Omega_{11} + 2p^*(1 - p^*)\Omega_{12} + (1 - p^*)^2\,\Omega_{22}$$

Assumptions

Before we apply the theory developed in the preceeding section to the problem of how fishing pressure as measured by legal minimum size may affect maintenance of genetic polymorphism, it is proper that we state explicitly the assumptions on which the theory is based. Some of the assumptions (single locus with two alleles, large population size, etc.) are common in population genetics theory. Here, we discuss those assumptions which are likely to be controversial or problematical.

Random mating

Gametes are assumed to combine at random with respect to the age, size, and genotype of the parent. In species where the males shed their gametes into the water column, this assumption may be a good approximation. In species which mate assortatively on the basis of size or age, genotypic frequencies among the zygotes may differ significantly from the Hardy–

Weinberg expectations (e.g., Zouros and Foltz 1984). In this instance, size-selective fishing mortality has the potential for disrupting mating patterns, with population genetic consequences that can only be answered with a more complex model.

Reproduction proportional to size

This assumption is critical to our models, since it is the larger size of the heterozygotes that allows them to produce more offspring. If no relationship exists between size and fertility for individuals of the same age, then our model is inappropriate. There is some evidence for bivalve molluscs (Bayne et al. 1983; Shafee and Lucas 1980; Thompson 1979) that fecundity is a linear or nearly linear function of body size, although age was not considered as a covariate in these analyses.

Same age at first reproduction

The parameter x_0 in our models represents the age at which reproduction begins. It is assumed to be the same value for all three genotypes. Parameter x_0 also marks the age at which we begin measuring growth. Therefore, prior to age x_0, we assume that all genotypes of the same age are identical in size. For a species with a planktonic larval stage, x_0 might correspond to the age at metamorphosis. If some genotypes begin reproduction at an earlier age, but the differences among genotypes are small relative to the duration of the reproductive period, then this assumption may be a convenient simplification.

Constant natural mortality rate

In the absence of fishing, we have assumed that the instantaneous probability of dying is independent of size and age. If mortality rate is a function of age (e.g., senescence), this should not have an important qualitative effect on our models, since all genotypes age at the same rate. However, if natural mortality is a function of size (e.g., smaller individuals are more vulnerable to predators), then the different genotypes will have different survivorship curves owing to differential rates of growth. In either case, one might expect size-dependent fishing mortality to reduce the likelihood of a polymorphism maintained by genotype-dependent growth. We assume that violations of this assumption will have a negligible effect on the genetic equilibrium of the population, compared to the effect of fishing mortality.

Legal minimum size

We have assumed no fishing mortality below the legal minimum size. More realistically, one might expect some fishing-induced mortality to individuals below the legal minimum. In this respect, model 2 is likely to be a better representation than model 1, because in model 2 the mortality rate owing to fishing increases with the size of the individual. Any fishing-induced mortality which affects individuals below the legal minimum size would be qualitatively similar to reducing the legal minimum size.

Constant population density

Our analysis has been focused upon equilibrium conditions. In particular, we have ignored completely the potential effects of fishing on changes in population density. In our models we have assumed that recruitment always balances mortality, resulting in a stable equilibrium population size. This was accomplished by making the constant which relates reproduction to size a function of time [7]. Therefore, our models assume that reproduction will always be sufficient to offset losses owing to mortality. While this might be true for low to moderate rates of fishing, this assumption might be questioned when populations are being heavily exploited. One should remember that our models were designed to address genetic questions and not such topics as extinction of the population or maximum sustainable yield. Also, our models provide no information on the rate of approach to genetic equilibrium.

Overdominance for growth rate

Throughout this paper, we have assumed that heterozygotes have an advantage in growth rate over homozygotes. This assumption seems reasonable considering the increasing evidence for overdominance in growth rate in a variety of species. This evidence is based on allozyme variation. Although many authors support the view that the allozymes are themselves the agents of overdominance, associative overdominance (linkage of the various allozymes with different detrimental mutations) can not be excluded. Favro et al. (1979, 1980) have studied the effects of size-selective fishing when heterozygotes are intermediate in growth rate between the two homozygotes (codominance).

No life history differences between the sexes

Implicit in our models is the assumption that males and females do not experience different growth rates or different age- or size-dependent mortalities. In other words, our model assumes a constant sex ratio at all ages and sizes. In simultaneous hermaphrodites (e.g., some *Pecten* species), this assumption is probably valid. For protandrous hermaphrodites, this assumption is less satisfactory. Buroker (1983) has reviewed the evidence concerning size-dependent changes in sex ratio in oysters (*Crassostrea gigas*). Given the strong influence of the environment on sex ratios in oysters, it may be difficult to include sex differences in our models without greatly increasing their complexity.

Effect of fishing pressure on genetic variation

We can now address the question of how fishing pressure as measured by fishing mortality, $f(y)$, and

legal minimum size, β, affect the maintenance of genetic polymorphism. To answer this question, we will (*i*) compute Ω_{ij} from [26] for each genotype, (*ii*) compute p^* from [30] using the Ω_{ij}'s, and (*iii*) determine values of fishing pressure (parameters f_1 or f_2, and β) that allow a stable polymorphism.

Solutions to Ω_{ij} *(model 1)*

$$\Omega_{ij} = \int_{x_0}^{\infty} W_{ij}(x) l_{ij}(x) dx$$

Substituting [14] gives

$$\Omega_{ij} = \int_{x_0}^{\tilde{x}_{ij}} W_{ij}(x) e^{-Zx} dx + \int_{\tilde{x}_{ij}}^{\infty} W_{ij}(x) e^{-Zx} e^{-f_1(x-\tilde{x}_{ij})} dx$$

Substituting [5] and solving gives

$$[33]\quad \Omega_{ij} = e^{-Zx_0} B_{ij} (Z^{-1} - (K_{ij} + Z)^{-1} - (Z^{-1} - (f_1 + Z)^{-1})(1 - (\beta/B_{ij})) K_{ij}^{-1} Z + ((K_{ij} + Z)^{-1} - (f_1 + K_{ij} + Z)^{-1}) \times (1 - (\beta/B_{ij}))^{K_{ij}^{-1} Z + 1})$$

The following special cases are worth noting: If $f_1 \equiv 0$ or $\beta \geq B_{ij}$, then

$$[34]\quad \Omega_{ij} = e^{-Zx_0} B_{ij} (Z^{-1} - (K_{ij} + Z)^{-1})$$

If $\beta = 0$, then

$$[35]\quad \Omega_{ij} = e^{-Zx_0} B_{ij} ((f_1 + Z)^{-1} - (f_1 + K_{ij} + Z)^{-1})$$

For these two cases, if $K_{11} = K_{12} = K_{22}$, then

$$[36]\quad p^* = \frac{B_{12} - B_{22}}{2B_{12} - B_{11} - B_{22}}$$

Stable polymorphism is only possible if $B_{11} < B_{12} > B_{22}$. If $B_{11} = B_{12} = B_{22}$, then [34] gives

$$[37]\quad p^* = \frac{(K_{12} - K_{22})(K_{22} + Z)^{-1}}{(K_{12} - K_{11})(K_{11} + Z)^{-1} + (K_{12} - K_{22})(K_{22} + Z)^{-1}}$$

and [35] gives

$$[38]\quad p^* = \frac{(K_{12} - K_{22})(K_{22} + f_1 + Z)^{-1}}{(K_{12} - K_{11})(K_{11} + f_1 + Z)^{-1} + (K_{12} - K_{22})(K_{22} + f_1 + Z)^{-1}}$$

For [37] and [38], a stable polymorphism is only possible if $K_{11} < K_{12} > K_{22}$. Thus, in the absence of fishing [34] or in the absence of a legal minimum size [35], polymorphism requires that the heterozygote be larger in size than either homozygote for any given age.

Solutions to Ω_{ij} *(model 2)*

This model is more complex. An explicit solution for Ω_{ij} can be obtained by substituting $u = B_{ij} e^{-K_{ij}(x - x_0)}$ and $-K_{ij}^{-1} u^{-1} du = dx$ into the integral $\Omega_{ij} = \int_{x_0}^{\infty} W_{ij}(x) l_{ij}(x) dx$. The solution is

$$[39]\quad \Omega_{ij} = e^{-Zx_0} B_{ij} (Z^{-1}(1 - (1 - \beta/B_{ij})^{K_{ij}^{-1} Z}) - (Z + K_{ij})^{-1}(1 - (1 - \beta/B_{ij})^{K_{ij}^{-1} Z + 1})) + e^{-Zx_0} (1 - \beta/B_{ij})^{K_{ij}^{-1} Z} e^{f_2 K_{ij}^{-1} (B_{ij} - \beta)} K_{ij}^{-1} \quad \text{(an infinite series)}$$

where an infinite series $= \sum_{h=0}^{\infty} \frac{(-1)^h (f_2 K_{ij}^{-1})^h (B_{ij} - \beta)^h (\beta + B_{ij}(K_{ij}^{-1}(Z + f_2 B_{ij}) + h)^{-1})}{h!(K_{ij}^{-1}(Z + f_2 B_{ij}) + h + 1)}$

This infinite series will always converge. Here again, when $f_2 \equiv 0$ or $\beta = B_{ij}$, one obtains [34].

Numerical results (model 1)

To evaluate the effect of fishing pressure on the maintenance of genetic polymorphism, we chose specific numerical values for all parameters except f_1 and β. For these numerical values, we searched for values of f_1 and β that represented the "dividing line" between stable polymorphism and fixation of one of the alleles. This occurs when $\Omega_{12} = \min(\Omega_{11}, \Omega_{22})$. Because absolute size is arbitrary, we set the maximum size of heterozygotes, B_{12}, at 1.0. Heterozygotes were assigned a 5% advantage in both maximum size and rate of growth (K_{12}). Research on oysters (Zouros et al. 1980; Singh 1982) has consistently observed an average growth advantage for heterozygotes of 5–7%. The absolute magnitudes of K_{12} and f_1 are less important than their magnitudes relative to Z. For example, doubling both K_{12} and Z will give, for each β, a critical value of f_1 that is twice its original value. Therefore, in presenting our results we have given the ratios K_{12}/Z and f_1/Z rather than the actual values of K_{12}, f_1, and Z that were used in the computations. A large ratio of K_{12}/Z represents a situation in which the growth rate exceeds the mortality rate and most individuals grow to

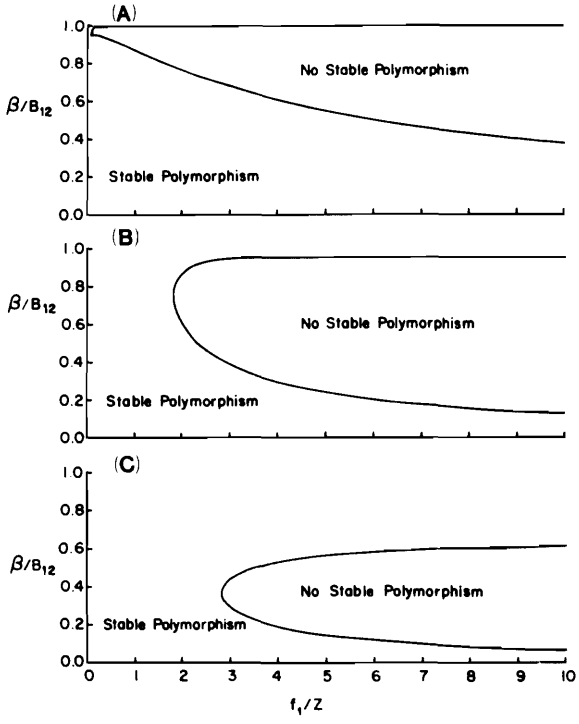

FIG. 1. Critical values of legal minimum size (β/B_{12}) under model 1 separating stable polymorphism from fixation as a function of fishing mortality (f_1/Z) for (A) $K_{12}/Z = 5.0$, (B) $K_{12}/Z = 1.0$, and (C) $K_{12}/Z = 0.5$. See text for details of the model.

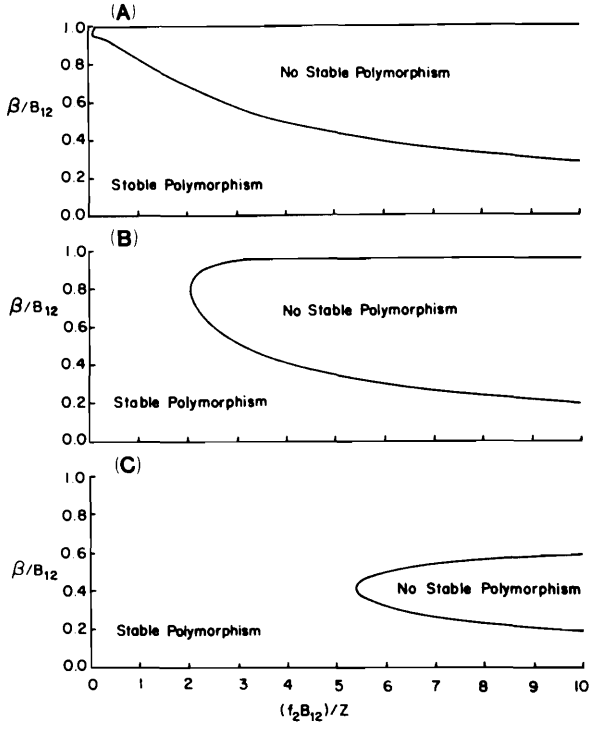

FIG. 2. Critical values of legal minimum size (β/B_{12}) under model 2 separating stable polymorphism from fixation as a function of fishing mortality (f_2B_{12}/Z) for (A) $K_{12}/Z = 5.0$, (B) $K_{12}/Z = 1.0$, and (C) $K_{12}/Z = 0.5$.

a large size before dying. In contrast, a low K_{12}/Z ratio represents a situation in which the natural mortality rate is large and few individuals grow to a large size. The K_{12}/Z values of 0.5, 1.0, and 5.0 can be thought of as representing environmental conditions which are harsh, moderate, and benign, respectively. The ratio f_1/Z is a dimensionless quantity which represents the rate of fishing mortality relative to the natural mortality rate. In a similar fashion, we have expressed the legal minimum size as the ratio β/B_{12}, because the absolute size of the animals is arbitrary. The following table summarizes this information:

Parameter	Value(s)
x_o	0.0
B_{12}	1.00
B_{11}, B_{22}	0.95
K_{12}/Z	0.5, 1.0, 5.0
K_{11}/K_{12}, K_{22}/K_{12}	0.95

The results of the analysis appear in Fig. 1. There is a family of curves, each curve representing a different value of the ratio K_{12}/Z. In harsh environments (Fig. 1C), fishing mortality must be large to have an impact on the genetic composition of the population, even when this mortality is scaled to the natural mortality rate. In this case, the polymorphism is most vulnerable to fishing mortality at intermediate values of β/B_{12}. A similar situation obtains for moderate environments (Fig. 1B). In benign environments (Fig. 1A), there is a monotonic relationship between the critical values of β and f_1, for values of β/B_{12} less than 0.95. For large values of β/B_{12}, a small amount of fishing pressure can result in decreased polymorphism. This is because the faster-growing heterozygotes reach the legal minimum size more quickly than slower-growing homozygotes. Formally, this is a case of underdominance, which is known not to result in stable polymorphism, but in fixation of one or the other allele depending on the initial perturbation from equilibrium. The difference in the ages at legal size, $\max(\tilde{x}_{11}, \tilde{x}_{22}) - \tilde{x}_{12}$, grows larger as β gets closer to the maximum size B_{12}. In the extreme case where $B_{12} > \beta > B_{11} = B_{22}$, only heterozygotes are subject to fishing mortality because homozygotes never reach the legal minimum size. Conversely, as β goes to zero, all genotypes reach legal size at approximately the same age, and, therefore, the dif-

ferential effects of fishing mortality on genotypes are small. Thus, in benign environments genetic polymorphism can be maintained in the population even when mortality due to fishing is high, provided the legal minimum size is low enough. We repeated the analysis for growth rates representing 1% advantage for heterozygotes and obtained similar results. The results are also similar when growth rates are set equal across genotypes and only maximum size varies between homozygotes and heterozygotes, and vice versa.

Numerical results (model 2)

For model 2, heterozygotes were again assigned a 5% advantage in growth rate and maximum size. As before, we have expressed K_{12} relative to Z. The major difference in model 2 is that the instantaneous mortality rate, f_2, gives the amount of fishing mortality per unit size. In presenting the numerical results, we have taken this size dependence into account by multiplying f_2 by B_{12} before dividing by Z. As before, the result is a dimensionless quantity which represents the rate of fishing mortality relative to the natural mortality rate. The parameter values used are the same as in model 1. The results of the analysis are given in Fig. 2. As before, we have presented a family of curves representing three values of K_{12}/Z. Qualitatively, the results are very similar to model 1, which suggests that our conclusions are fairly robust.

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