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Post-settlement viability in the American oyster (*Crassostrea virginica*): an overdominant phenotype

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SUMMARY

The degree of heterozygosity as determined by electrophoretic analysis of three or four polymorphic loci correlates positively with survival in age groups of the American oyster (*Crassostrea virginica*) collected as spat from two different natural populations. The phenomenon is shown to operate in ages from 2 weeks (post-settlement) to 3 years and appears to be general in populations of marine molluscs. The most likely explanation for this result is that heterozygosity improves survival through its effect on growth (heterozygotes grow faster). The effects of individual loci on viability are independent of each other. A direct involvement of the enzyme polymorphisms is the most probable genetic interpretation of the data, but associative overdominance cannot be excluded.

1. INTRODUCTION

Reports of higher heterozygote viabilities abound in the literature. Inbreeding studies almost invariably produce strong correlations between degree of inbreeding and loss of viability (e.g. Dobzhansky, Spassky & Tidwell, 1963; Levene *et al.* 1965; Schull & Neel, 1965). In such studies a substantial fraction of the genome becomes homozygous and, therefore, the direct effect of heterozygosity at individual loci cannot be clearly established. Studies which have reported correlations between allozyme heterozygosity and viability in outbreeding populations remain relatively few and include those of Tinkle & Selander (1973) on the lizard *Sceloporus graciosus*, Mitton & Koehn (1975) on the marine fish *Fundulus heteroclitus*, Schaal & Levin (1976) on the perennial herb *Liatris cylindracea*, Watt (1977) on *Colias* butterflies and Black & Salzano (1981) on humans.

One group of organisms which has proved particularly suitable for population genetic studies is that of marine molluscs. These organisms are relatively easy to collect in large numbers, they can be marked and studied *in situ*, they can be aged (in most instances) and they are exposed to environmental conditions which may vary markedly on a micro- or macro-geographical scale.

As a result of these advantages, there has been a growing literature on the population genetics of marine molluscs, and several observations of general interest

have emerged. The work of Koehn and his associates, for example, has provided a clear example of a gene frequency cline maintained by a balance between gene flow and differential survival among genotypes for the leucine aminopeptidase locus (Koehn, Newell & Immermann, 1980). Significant and persistent deviations of genotypic frequencies from the Hardy–Weinberg expectations have been observed in many species of marine molluscs (see Zouros & Foltz, 1982, for a recent review), which suggests the frequent occurrence of selective forces causing post-settlement micro-geographical differentiation, and a case of heterozygosity-dependent growth has been established in the American oyster (Singh & Zouros, 1978; Zouros *et al.* 1980).

Several population studies of marine molluscs have shown that shell size is positively correlated with degree of enzyme heterozygosity (Koehn, Turano & Mitton, 1973; Tracey, Bellet & Gravem, 1975; Levinton & Fundiller, 1975). One difficulty with most of these studies is that the animals' ages were not accurately determined and, therefore, one cannot know whether the observed differences in shell size are due to differential age, differential growth or a combination of the two. In this paper we report on the levels of enzyme heterozygosity among age cohorts of the American oyster and conclude that viabilities are influenced by the degree of heterozygosity.

2. MATERIALS AND METHODS

As described in earlier papers (Singh & Zouros, 1978; Zouros *et al.* 1980), spawning of the American oyster in the Atlantic provinces of Canada occurs once a year, in mid-July. Settling of larvae occurs about 3 weeks later. The animals used in this study were collected as settling larvae (spat) from two localities: Ellerslie Inlet in Prince Edward Island (P.E.I.) and the River Denys in Cape Breton, Nova Scotia.

(i) *Age classes from P.E.I.* One cohort was collected on scallop shells in August 1976. The animals were kept in a floating tray suspended in the Ellerslie inlet. After 1 year a random sample was drawn from the tray and the animals were individually weighed and assayed electrophoretically for seven enzyme loci. The correlation of weight with individual degree of heterozygosity has been reported in a previous publication (Zouros *et al.* 1980). In this paper, we will refer to this cohort as the 1-year-old class from P.E.I.

Another cohort was collected from the same locality in August 1979. A random sample of scallop shells containing oyster spat was submerged in a laboratory tank at the Ellerslie Fisheries Research Station. The animals were provided with a continuous flow of filtered sea water and fed cultured phytoplankton until they were large enough for electrophoresis. This size, approximately 0·5 cm in shell diameter, was reached in about 3 weeks. A random sample from this population provided the 3-week-old age class.

Another set of spat-containing scallop shells in 1979 provided the 2-month-old age class. Immediately after removal from the water, each scallop shell was divided into nine cells by drawing two vertical and two horizontal lines with indelible ink. Using a microscope and dissecting needle, we removed all oyster spat except one, located approximately in the centre of each of the nine cells. Sixty-two

scallop shells were treated in this way, producing a population of 558 oyster spat. The spat were then treated as described for 3-week-old oysters, and returned to the inlet for an additional 6 weeks (no planktonic larvae existed in the inlet at that time). At the end of this period, 265 (47·5 %) of the original 558 spat survived and were processed for electrophoresis.

(ii) *Age classes from Cape Breton.* Spat were collected during July in 1977, 1978 and 1979. The resulting cohorts were kept in the water until August 1980. A random sample was drawn from each cohort to provide the material for the 3-, 2-, and 1-year-old age classes. The 3-year-old class may have included a few individuals from 1976.

Table 1. *Single-locus statistics in three age cohorts from Prince Edward Island*

(a, Three-week-old cohort; b, 2-month-old cohort; c, 1-year-old cohort; N, sample size; H_o, observed heterozygosity; D, index of heterozygote deficiency defined as $D = (H_o - H_e)/H_e$, where $H_e = 1 - \Sigma_i p_i^2$ and p_i is the frequency of the ith allele.)

	Lap-2			*Pgi*			*Pgm*		
Allele	a	b	c	a	b	c	a	b	c
2	0·41	0·53	0·48	0·83	0·81	0·79	0·27	0·20	0·20
3	0·43	0·31	0·38	—	—	—	0·40	0·43	0·58
4	0·15	0·13	0·12	0·16	0·18	0·20	0·31	0·32	0·20
Others	0·01	0·03	0·02	0·01	0·01	0·01	0·02	0·05	0·02
N	497	257	1803	499	265	1764	369	247	1735
H_o	0·21	0·26	0·34	0·28	0·30	0·31	0·24	0·28	0·38
D	−0·64	−0·57	−0·45	−0·03†	−0·03†	−0·07	−0·64	−0·60	−0·34

† Not significantly different from zero.

(iii) *Electrophoresis.* The techniques of horizontal starch-gel electrophoresis are described by Singh & Zouros (1978) and Zouros *et al.* (1980). The 3-week- and 2-month-old age classes were scored for the following enzymes: leucine aminopeptidase-2 (*Lap-2*), phosphoglucose isomerase (*Pgi*) and phosphoglucomutase (*Pgm*). The 1-year-, 2-year- and 3-year-old cohorts from Cape Breton were scored for four enzymes: *Lap-2*, *Pgi*, glutamate oxaloacetate transaminase (*Got-1*) and esterase-3 (*Est-3*).

3. RESULTS

Table 1 displays one-locus statistics for the three age classes from P.E.I. The heterogeneity in allele frequencies among the three classes is tested in Table 3(*a*) and found to be significant for all three loci. No clear trends could be seen in the change of allele frequencies with age, therefore no meaningful interpretation can be given for this heterogeneity. When observed heterozygosities are compared, it can be seen that they increase with age for all three loci. This increase is tested for significance in Table 3 (*b*) and found to be significant for *Lap-2* and *Pgm*, but not for *Pgi*. In these tests, all homozygote classes were pooled to form one class, and similarly for the heterozygotes. In addition to the increase of heterozygosity with age, it should be noted that observed heterozygosities are lower than the Hardy–Weinberg expectations. This phenomenon, which we shall refer to as

heterozygote deficiency, is most pronounced among young animals, and its magnitude steadily declines with age.

Similar results are obtained for the age cohorts from Cape Breton. One-locus statistics are given in Table 2, and heterogeneity tests in Table 3. Again, no general pattern could be discerned in the change of allele frequencies with age, but observed heterozygosities increased with age in most instances. Significant heterozygote deficiencies were also observed, but in general the deficiencies are lower than those

Table 2. *Single-locus statistics in three age cohorts from Cape Breton*

(a, One-year-old cohort; b, 2-year-old cohort; c, 3-year-old cohort (rest of notation as in Table 1).)

	Lap-2			*Pgi*		
Allele	a	b	c	a	b	c
1	0·16	0·11	0·14	—	—	—
2	0·21	0·29	0·20	0·67	0·65	0·68
3	0·55	0·51	0·52	—	—	—
4	0·07	0·08	0·12	0·33	0·34	0·32
Others	0·01	0·01	0·02	—	0·01	—
N	209	204	203	209	209	206
H_o	0·42	0·31	0·42	0·44	0·48	0·53
D	−0·32	−0·50	−0·36	0·00†	0·04†	0·22
	Got-1			*Est-3*		
Allele	a	b	c	a	b	c
1	0·01	0·09	0·14	0·15	0·12	0·09
2	0·70	0·65	0·57	0·18	0·16	0·15
3	—	—	—	0·47	0·59	0·65
4	0·29	0·26	0·29	0·18	0·10	0·09
Others	—	—	—	0·02	0·03	0·02
N	200	209	204	200	209	203
H_o	0·40	0·45	0·61	0·34	0·42	0·32
D	−0·07†	−0·10†	0·07	−0·50	−0·30	−0·41

† Not significantly different from zero.

observed in the young age classes from P.E.I. This observation is consistent with the notion that heterozygote deficiencies diminish with age.

With few exceptions, all animals were scored for all three or four loci (depending on locality). An index can be attached to each animal which will vary from 0 to 3 or from 0 to 4, depending on the number of loci for which the animal was a heterozygote. We can then ask how this index of individual heterozygosity changes with age. Table 4 classifies the number of individuals in each age class according to degree of individual heterozygosity. Percentages (relative frequencies) are given in parentheses. The frequency of animals with low individual heterozygosity (0 to 1) tends to decrease with age, and conversely for animals with high individual heterozygosity (2 or more). It appears that degree of individual heterozygosity is correlated with viability. A simple way to illustrate this correlation with our data is to compute an index which measures the relative

Table 3. *Homogeneity tests of allele frequencies and of homozygote/heterozygote frequencies among three age cohorts of oysters from two localities in the Atlantic provinces of Canada*

(The G statistic (Sokal & Rohlf, 1969) was used in these tests. D.F. = degrees of freedom, P = probability of homogeneity.)

		(*a*) allele frequency tests			(*b*) homozygote/heterozygote test		
Locality	Locus	G	D.F.	P	G	D.F.	P
P.E.I.	*Lap-2*	55·21	8	0·001	31·32	2	0·001
	Pgi	11·72	4	0·019	1·84	2	0·399
	Pgm	137·03	10	0·001	39·55	2	0·001
Cape Breton	*Lap-2*	21·37	8	0·006	7·07	2	0·029
	Pgi	0·81	2	0·667	3·31	2	0·191
	Got-1	60·79	4	0·001	19·26	2	0·001
	Est-3	35·44	8	0·001	5·08	2	0·079

Table 4. *Distribution of individual heterozygosities among the three age cohorts from each locality*

(The test of heterogeneity gives G (6 D.F.) = 49·60, $P \cong 0$ for P.E.I. and G (8 D.F.) = 10·33, $P = 0{\cdot}24$ for Cape Breton. N = number of heterozygous loci.)

	Prince Edward Island		
N	3 weeks old	2 months old	1 year old
0	146 (40·4%)	87 (36·7%)	449 (27·6%)
1	166 (46·0%)	107 (45·2%)	730 (44·8%)
2	44 (12·2%)	38 (16·0%)	393 (24·1%)
3	5 (1·4%)	5 (2·1%)	57 (3·5%)
Total	361	237	1629
	Cape Breton		
N	1 year old	2 years old	3 years old
0	29 (14·5%)	29 (13·9%)	16 (7·9%)
1	68 (34·0%)	66 (31·6%)	57 (28·1%)
2	62 (31·0%)	66 (31·6%)	71 (35·0%)
3	35 (17·5%)	38 (18·2%)	48 (23·6%)
4	6 (3·0%)	10 (4·7%)	11 (5·4%)
Total	200	209	203

change in the frequency of a heterozygosity class over an age transition. Thus, if $P_{i,n}$ is the frequency of individuals of heterozygosity i ($i = 0, 1, 2, 3$, or $i = 0, 1, 2, 3, 4$) in age-class n ($n = 1, 2, 3$), then the relative viability of class i in the transition from age n to age $n+1$ will be

$$V = \frac{P_{i,n+1} - P_{i,n}}{P_{i,n}}. \tag{1}$$

Clearly, V is subject to large sampling error (whose calculation requires knowledge of the covariance of $P_{i,n}$ and $P_{i,n+1}$) and is not appropriate for statistical testing.

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Such tests can be done by using the multinomial distribution, as was done in Table 3. But V can be useful as a comparison index, and as such is shown in the upper part of Fig. 1. It can be seen that multiply-heterozygous genotypes have, in general, higher relative viabilities. Also, at P.E.I. the slope of V versus individual

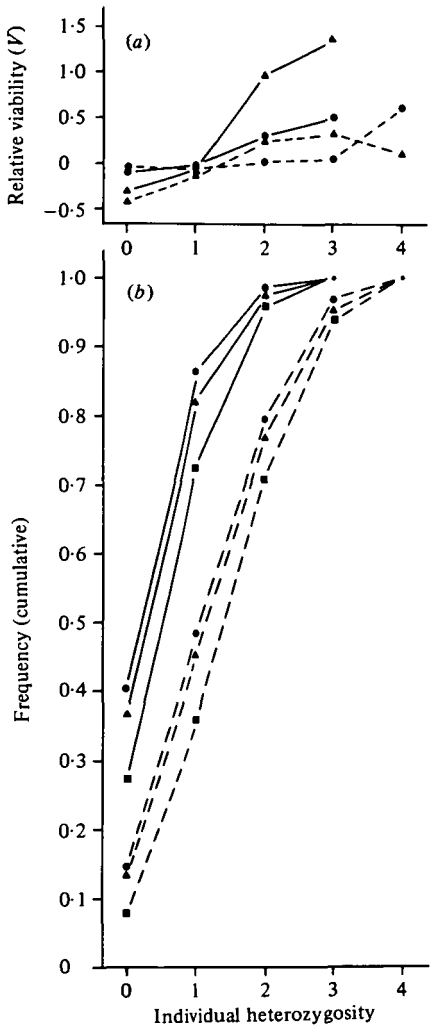

Fig. 1. (*a*) Relative viability between the youngest and the intermediate age class (●) and between the intermediate and the oldest age class (▲) as a function of degree of individual heterozygosity for oysters from Prince Edward Island (solid lines) and Cape Breton (broken lines). (*b*) Cumulative frequency distribution of animals of given degree of individual heterozygosity for the youngest (●), intermediate (▲) and oldest (■) age classes from Prince Edward Island (solid lines) and Cape Breton (broken lines).

heterozygosity is larger in the second transition (2 months to 1 year) than in the first (2 weeks to 2 months). This result may be due to the longer time interval between the second and third age classes than between the first and second age classes. At Cape Breton, the transitions are equally spaced in time and the slopes appear to be of the same magnitude.

The effect of increased viability of multiply-heterozygous individuals on the cumulative distribution of heterozygosity is shown in the lower part of Fig. 1. It can be seen that in a cohort the frequency of individuals with a heterozygosity equal to or higher than a given value increases with the age of the cohort.

If we assume that the increase of heterozygosity with age is due to selection, then this selection can be either of the form of overdominance (the heterozygote viability exceeds the viabilities of both homozygotes) or of the form of selection favouring the homozygote for the less frequent allele (with the heterozygote having an intermediate viability). In terms of the maintenance of genetic variation in populations, these are rather different types of selection, and distinguishing between them is important in population genetic studies. One way of providing evidence for one or the other type is to compare the relative viability (V) of any two types of homozygotes with the relative viability of the corresponding heterozygote. Because of the sensitivity of V to sample size, we have employed this comparison only for the two most common alleles. This analysis yielded six comparisons for the P.E.I. age groups (three loci × two age transitions) and eight comparisons for Cape Breton (four loci × two age transitions). Of these, four P.E.I. comparisons and six Cape Breton comparisons produced a higher V for the heterozygote. Given that in each comparison the probability that the heterozygote viability will exceed both homozygote viabilities is 1/3, the probability of seeing this event in 10 out of 14 trials by chance alone is 0·004.

Finally, we note that selective explanations of multi-locus observations depend critically on the assumption of independent segregation of one-locus genotypes in the population. In the present study, the effect of a locus on viability was studied by classifying each individual as homozygous or heterozygous for the locus in question. Therefore, the problem is reduced to testing whether the distribution of multiple-locus heterozygosity is independent of the marginal (one-locus) distributions. Chi-square contingency tests of all possible pair-wise combinations of loci produced results consistent with the null hypothesis of random association. This result suggests that the effects of individual loci on viability are additive and, therefore, that one-locus selection models may provide an adequate description of the selective process involved.

4. DISCUSSION

Our survey includes six age classes of the American oyster sampled as spat from two localities. Five different enzyme loci were used as indicators of overall heterozygosity. The results indicate that heterozygotes have higher survival expectancies. This phenomenon does not appear to be age-specific (it was shown to operate at least up to the age of 3 years) nor to depend on the locality of sampling. Across the various enzyme loci, the direction of the effect appears to be the same, but its magnitude clearly varies.

A review of the literature indicates that evidence for higher heterozygote viabilities can be found in many studies of marine bivalves. We have attempted to summarize the most relevant information in Table 5. This table covers nine reported cases of genetic differences among size or age classes in five species of marine molluscs. The genetic differences are either in allele frequency or in the

Table 5. *Reported cases of association of survival with heterozygosity in marine molluscs*

Species	Locus	Comment	Source
Modiolus demissus	*To*	D increases with shell length	Koehn *et al.* (1973)
M. californianus	*Lap*	Size 1–2 mm, $D = -0{\cdot}51$; size 9–10 cm, $D = -0{\cdot}08$	Tracey *et al.* (1975)
	Pgi	Size 1–2 mm, $D = -0{\cdot}39$; size 9–10 cm, $D = -0{\cdot}30$	
M. edulis	*Got*	Size 5–9 mm, $D = -0{\cdot}70$; size 1–5 cm, $D = 0{\cdot}08$	Johnson & Utter (1975)
M. californianus	*Lap*	r (correlation of shell length with D) = 0·9	Levinton & Fundiller (1975)
Mya arenaria	*Lap*	r (correlation of common allele frequency with subclass size) < 0	Levinton & Fundiller (1975)
M. edulis	*Lap*	Minor allele frequency increases with shell size; D increases with shell size	Koehn *et al.* (1976)
Modiolus demissus	*To*	Small animals: $D = -0{\cdot}06$; large animals: $D = 0{\cdot}17$	Chaisson *et al.* (1976)
C. gigas	*Mp*	2 years old: $H = 0{\cdot}42$; 4 years old: $H = 0{\cdot}49$; 6 years old: $H = 0{\cdot}49$	Buroker (1979)
C. gigas	*Cat*	Survival of homozygote 43 % of heterozygote	Fujio *et al.* (1979)

magnitude of heterozygote deficiency (measured by D). The table does not include cases in which the correlation between heterozygosity and shell size clearly is due to differential growth as opposed to differential survival (e.g. in the American oyster: Zouros *et al.* 1980). In most previous studies of marine bivalves, the two effects cannot be separated due to lack of control over the age structure of the population. The present study is not limited in this way, because the age of each animal is known.

Not all attempts to correlate shell size with degree of heterozygosity at enzyme loci have succeeded. In a study involving over a hundred sampling localities and several thousand specimens, Koehn, Milkman & Mitton (1976) have scored six loci in *Mytilus edulis*, but have reported shell size-heterozygosity correlation only for the *Lap* locus. The other loci either had no effect on growth or survival, or their effect was minor and had been obscured by environmental variation.

Negative correlations between heterozygosity and shell size have been reported by Wilkins (1978) and Beaumont (1982). Wilkins has studied the distribution of heterozygosity at the *Pgi* locus in shell size classes from three populations of the scallop *Pecten maximus*. He observed that mean individual heterozygosity was higher in the smaller classes than in the larger ones. A likely explanation considered by Wilkins is that the animals constituting the large shell classes may be heterogeneous in age. If allele frequencies vary among yearly recruits, then a 'Wahlund effect' is likely to appear when animals of different age are combined to form one sample, and thus produce an apparent heterozygote deficiency. Another explanation is that heterozygous scallops may grow faster and, as a result, be more likely to be taken by fishermen once their size is above the legal minimum. (Wilkins's samples came from beds which had been subject to continuous commercial fishing.) This preferential removal of heterozygotes will not occur in young age cohorts in which all individuals are below the legal minimum size. Beaumont (1982) examined the heterozygosity levels at a non-specific protein locus (*Pt-A*)

and at the *Pgm-2* locus in *Chlamys opercularis*, the queen scallop. Even though no clear trend can be seen in his study, several comparisons have produced lower heterozygosity levels among classes of larger animals. Beaumont proposes population mixing as the most likely explanation for this observation.

The design of our experiment precludes population mixing as an explanation of the difference in degree of heterozygosity among age classes. The increase in heterozygosity among older animals in the P.E.I. samples may perhaps reflect a year effect (1976 versus 1979) or a treatment effect (fed versus non-fed) rather than an age effect as such. A year effect could also account for the pattern of heterozygosity among age classes in the Cape Breton oysters. The year-effect explanation would require that, in each locality, the earlier years were more favourable for the survival of heterozygotes than the later years. We think that genotypic-specific mortality is a more plausible explanation for these results, although a change over time in some environmental factor affecting survival of heterozygotes cannot be ruled out. Given that negative correlations of heterozygosity with shell size have only rarely been reported for populations of bivalve molluscs, genotypic-specific mortality also appears to be a more general explanation. The regularity in the observed trend toward increased heterozygosity with age favours some form of balancing selection. Of the various common types of balancing selection, our results seem to exclude frequency-dependent selection, because there has been no increase in the low-frequency alleles with age. However, we cannot distinguish between unconditional overdominance and balancing selection resulting from varying selection coefficients due to short-term fluctuations of the environment (Gillespie & Langley, 1974). Although the evidence for selection is very strong, these data provide no escape from the perennial argument that the actual targets of selection are not the allozyme variants but rather some other components of the genome. Such an explanation would involve associative overdominance caused by linked detrimental mutations (Ohta, 1971). The associative overdominance hypothesis has been discussed in some detail by Zouros *et al.* (1980) in regard to the effect of heterozygosity on growth. If one discounts this hypothesis, then one needs a model that will explain overdominance in molecular terms. Such models have been proposed by Schwartz & Laughner (1969) and Fincham (1972). A hypothesis based on differences in energy expenditure during enzyme biosynthesis has been proposed by Berger (1976). An energetic argument also has been made by Koehn & Shumway (1982) to explain different levels of oxygen uptake among homozygous and heterozygous oysters.

Another difficulty with the type of observation reported here is that the number of loci studied is small compared to all loci that may affect the phenotypic character in question. In an outbreeding population, an individual heterozygous for one such locus will be homozygous for many other loci affecting the same character. Thus, individual heterozygosity measured at a few randomly selected loci is not expected to be strongly correlated with total (genic) heterozygosity. This problem has been raised by Eanes (1978) and Kat (1982), and was investigated by Mitton & Pierce (1980) and Chakraborty (1981). Chakraborty's analytic result is that the correlation between heterozygosity measured at r loci and heterozygosity for all n loci affecting a phenotypic character is roughly is equal to $(r/n)^{\frac{1}{2}}$, but it

is also affected by the mean and variance of heterozygosity at the n loci in the population. In our study, r is three or four and, therefore, no detectable correlations between sample heterozygosity and viability are expected unless the number of loci affecting viability is about 100 and the average population heterozygosity at these loci is less than 0·10.

The foregoing argument assumes that the examined loci represent a random sample of all loci affecting viability. It may be argued that the enzyme loci surveyed here do not represent such a sample, but rather a sample strongly biased toward loci with a high degree of polymorphism. The number of gene loci that may affect viability must be fairly large. Lewontin (1974, p. 44) estimates that the number of loci capable of mutating to lethals in the *Drosophila* genome is between 800 and 1300. The number of loci at which viability modifiers (not lethals) may occur could be much larger, if one assumes a continuous distribution of viability for new mutants (Crow, 1972). However, most of this variation will be subject to purifying genic selection which will keep the frequency of deleterious alleles per locus at risk in low levels. This frequency is estimated at 0·001 for recessive lethals, and it can be as high as 0·05 for heterotic deleterious alleles (Lewontin, 1974, p. 80). Thus, the majority of viability-affecting loci are expected to be monomorphic. The polymorphic loci represent a small subset, and from this subset we have selected the most variable ones: electrophoretically monomorphic loci or loci with low heterozygosities (less than 0·2) are not included in this study or in our study of growth (Zouros, Singh & Miles, 1980). The total number of enzyme loci likely to foster large amounts of variation in natural populations may not be large. In spite of considerable effort by many researchers working with a variety of organisms, the number of highly variable enzyme loci in electrophoretic surveys has not increased substantially, and methods of improved scoring of polymorphisms have failed to uncover polymorphisms for enzyme loci which were initially classified as monomorphic or nearly monomorphic (Ayala, 1982). Two-dimensional electrophoresis has severely reduced the estimates of overall heterozygosity in humans (McConkey, Taylor & Phan, 1979), *Drosophila* (Leigh-Brown & Langley, 1979) and mice (Racine & Langley, 1980). A small number of highly polymorphic (heterotic) loci affecting important fitness components such as viability is, also, more in line with the conventional concept of segregational genetic load. It may, then, be not totally unexpected that a small sample from this limited set of loci will show a detectable correlation with the fitness trait in question.

In previous studies we have established that heterozygotes for the enzyme loci employed in this study grow faster within the same age cohort. The present result of higher viability of heterozygotes can be explained as a consequence of differential growth. Larger animals of the same age may be less vulnerable to predation or to death due to extreme fluctuations in environmental conditions. Even if predation or death due to physical factors are not related to size, a positive pleiotropy between growth and viability may exist, although in many organisms, and in particular in fish, the opposite is true (Jones, 1958). Levinton & Fundiller (1975) appear to support a similar explanation for the differential growth and survival observed among various *Lap* genotypes of *Mytilus californianus* and *Mya arenaria*.

Finally, we return to the observation that an overall heterozygote deficiency is present in most age classes of the American oyster. Because this phenomenon is most pronounced among young oysters (3 weeks after settlement), its origin must be sought in the larval population. It can result either from population mixture or from some form of selection (other hypotheses such as inbreeding or null alleles appear very unlikely; see Zouros *et al.* 1980). Population mixture tells us nothing about viability selection during the larval stage. The hypothesis of selection will necessitate either dominance for the deleterious allele or underdominance for viability. In either case, the selection regime during the dispersal (pre-settlement) stage must be different from the one acting after settlement. This possible reversal of viability of various genotypes, which has been observed in other organisms (Clegg, Kahler & Allard, 1978), may be a general characteristic of marine molluscs. Its implications were examined by Zouros & Foltz (1982).

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