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Genetic variation in varying environments

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SUMMARY

In order to assess the relationship between genetic and environmental variability, a large natural population of *Drosophila melanogaster* was replicated as eight subpopulations, which were subjected to four different patterns of environmental variation. The environmental variable imposed was presence of 15% ethanol in the culture medium. Experimental treatments of the populations were intended to simulate constant environmental conditions, spatial heterogeneity in the environment, and two patterns of temporal environmental variation with different periodicity (long- and short-term temporal variation). Additive genetic and phenotypic variation in sternopleural and abdominal chaeta number, and body weight, were estimated in two successive years, and measurements were taken of the genotype–environment correlation of body weight and sternopleural bristle score with medium type.

Additive genetic variance of sternopleural chaeta number and of body weight was significantly greater in the three populations experiencing environmental heterogeneity than in the control population, but additive genetic variance of abdominal bristle score was not clearly affected by exposing populations to varying environments. Temporal environmental variation was equally, if not more, efficient in promoting the maintenance of genetic variation than spatial heterogeneity, but the cycle length of the temporal variation was of no consequence. Specific genotype–environment interactions were not present, therefore adaptation to heterogeneous environments is by selection of heterozygosity *per se*, rather than by differential survival of genotypes in the alternate niches.

1. INTRODUCTION

The discovery in the past decade of the ubiquity of enzyme polymorphism in natural populations has encouraged population geneticists to search for systematic forces responsible for the maintenance of this genetic variation. Two alternative proposals have been advanced: the first argues that such variation is for the most part adaptively neutral and maintained by a balance of stochastic processes such as migration and random drift; the second hypothesis states that the polymorphisms are maintained by a form of balancing selection. Although it is not possible to discriminate unambiguously between the two alternatives on the basis of available data in the form of gene frequencies of electrophoretic variants, other evidence demonstrating patterns of gene frequency distribution correlated with

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environmental parameters, and specific genotype–environment interactions in which single environmental correlates of particular gene frequency changes are established, speaks strongly in favour of the maintenance of variation by a form of balancing selection.

What exactly is the 'form' of selective maintenance? Over dominance, frequency-dependent and density-dependent selection have variously been nominated as prospective candidates, but all apparently lack the necessary generality for the maintenance of such ubiquitous polymorphism. Recently, therefore, much attention has been directed towards investigating the intuitively appealing idea that environmental variation in time and space may be sufficient to maintain the observed degree of genetic variability. The theoretical framework is well established (see Felsenstein, 1976, for a comprehensive review), and evidence from natural populations demonstrating positive association between the amount of genetic and environmental variability in many species is certainly suggestive (Nevo, 1978). However, because of the nature of data from natural populations *in situ*, the following ambiguities exist which necessitate caution in interpretation. In any given situation, an observed lack of variance cannot be unequivocally assigned to constancy of environmental factors – variance reduced by stochastic processes can never be eliminated as an alternative hypothesis since generally little or nothing is known of the past history or breeding structure of the community. The use of electrophoretic markers is also ambiguous in this context, for although the evidence generally indicates a form of balancing selection maintaining polymorphism, specific instances of high isozyme variability can always be argued to be adaptively neutral, and even if selection is operating, it is never clear whether it is for electrophoretic alleles or other linked loci. Adequate measures of environmental variability can rarely be employed in the study of natural populations *in situ*, and it is not often possible to identify relevant environmental parameters. The effects of temporal and spatial heterogeneity are of necessity completely confounded in any natural situation, and finally, any associations demonstrated give no indication of causality – are polymorphic populations, by virtue of their polymorphism, more adapted to exploit a greater variety of niches, or does variability of niches enable the survival of different types? Expressed in genetic terms, is heterozygosity *per se* selected in a fluctuating environment, or does diversifying selection for alternate alleles increase polymorphism in a heterogeneous habitat?

A critical experimental evaluation of this question must therefore involve analysis of laboratory populations, as only under controlled conditions will it be possible to circumvent the difficulties associated with the genetic analysis of natural populations *in situ*. It is possible in the laboratory to subject replicate populations of genotypes to systematically varying environmental alterations, and avoid the problems associated with interpretation of electrophoretic data by the measurement of responses of the populations to environmental heterogeneity by scoring meristic traits. The observed phenotypic variance may then be partitioned into genetic, environmental, and genotype–environment interaction components. It is the purpose of this investigation, therefore, to examine experimentally the rela-

tionship (if any) between genetic and environmental variation. Specifically, the following questions will be addressed: is genetic variance maintained in a variable environment? If so, then what is the relationship between the magnitude of genetic variance and the pattern of environmental heterogeneity experienced? i.e. What is the effect on genetic variance of long- and short-term, temporal and spatial environmental variation? What is the mechanism promoting the maintenance of genetic variation? The presence of a genotype–environment interaction component would indicate disruptive selection and fixation of alternate alleles in different environments, rather than selection for heterozygosity *per se*.

2. MATERIALS AND METHODS

(i) *Population*

Professor A. Prevosti of the University of Barcelona kindly provided a sample of 158 *Drosophila melanogaster* males and 122 females, trapped in an orchard in the Canary Islands. A cage population was established in September 1975 from the wild-caught females and the eggs and larvae present in the vials containing the flies. The cage was allowed to attain equilibrium population density and then in March 1976, 8 replicate population cages were initiated, each with a sample of 500 males and 500 females from the original Prevosti cage.

(ii) *Treatment of population cages*

The environmental variable imposed was presence of alcohol in the culture medium. The 'control' medium was standard Edinburgh agar-molasses-killed yeast medium (UFAW handbook, 1967); the 'alcohol' medium contained 15% absolute ethanol by volume, which was added after the control food had cooled sufficiently to prevent undue evaporation of the ethanol, and then mixed thoroughly. Population cages were maintained by the weekly addition of two half-pint milk bottles containing 100 ml of the appropriate medium, and the concomitant removal of the two bottles added three weeks previously. All cages were maintained at 25 °C, and attained population densities of approximately 3000–4000 individuals.

The cages were subjected to four different patterns of environmental variation, with two replicates of each pattern. Thus there were two control cages, which received weekly two fresh bottles of control (C) medium. Two cages intended to simulate spatial variation in the environment received one bottle to which 15% alcohol was added (A medium) and one bottle of C medium weekly. Two further cages received in alternate weeks two bottles of C medium, then two bottles of A medium – a pattern of short-term temporal variation (i.e. within the lifetime of an individual). Finally, long-term temporal variation in the environment was simulated by changing the medium type every four weeks. It was therefore intended that comparison of the treated cages with the control would indicate whether environmental variability had any effect on genetic variance, whereas comparisons

among treatments would show relative efficiencies of temporal and spatial, long- and short-term patterns in producing the effect. Replicate differences would be the result of genetic drift. (The treatment definitions are only strictly true for the adults; for example, in the 'spatial variation' cages females have a choice of two habitats in which to lay their eggs, but the larvae develop consequently in only that niche.) After one year a sample of 500 males and 500 females was taken from one of the control cages to initiate a new cage, which was then treated as a short-term temporally varying environment. The purpose of this was to investigate whether environmental variability maintains genetic variability at the level initially present in the population, or whether genetic variability can actually be increased under appropriate conditions.

For experimental purposes adult flies were not removed directly from the cages; rather, the population was sampled by allowing animals to lay eggs for 24 h in fresh bottles; thus all population comparisons were made on animals which had developed under the same environmental conditions. Both males and females were collected as virgins from these bottles, and allowed to mature for three days in vials to which a paste of live yeast had been added. As far as possible the age of the animals was controlled to be three days at the time of scoring and subsequent mating.

(iii) *Sternopleural bristle number*

The mean phenotypic variance and additive genetic variance of this character were calculated for each of the populations in two successive years. In the first series of measurements, a sample of approximately 140 males and 140 females from each cage and on each type of medium was scored for the sum of sternopleural chaeta number of left and right sides; the 20 highest scoring and the 20 lowest scoring males and females were selected and mated assortatively. These 40 pairs of flies laid eggs for 72 h on both C and A medium; 40 offspring (10 males and 10 females from each of the two medium types) from each mating were scored for the character. This design gives the most efficient estimate of heritability, calculated from the regression of mean offspring score on midparental value (Hill, 1970). The standard error of the heritability estimate is simply the standard error of the regression coefficient (see Sokal & Rohlf, 1969). An estimate of the genotype–environment correlation is obtained using the 'cross-regressions' of offspring raised on one substrate on parents raised on the alternate medium (Reeve, 1955); the standard error has been derived by Robertson (1959). The mean and phenotypic variance can be estimated from the original population sample. Since this estimate is independent of the heritability estimate, the additive genetic variance can be calculated from the product of the heritability and phenotypic variance, with standard error as given by Mackay (1979).

The following year an additional series of measurements of sternopleural bristle number were undertaken, utilizing a different design. Here random samples of 200 males and 200 females from each cage were scored for both sternopleural and abdominal chaeta number (the sum of segments 4 and 5 or of 5 and 6, for males

and females, respectively). Of these, a random sample of 50 males and 50 females were mated at random, and 10 male and 10 female offspring scored for both characters in each full-sib family. Only control medium was used throughout this experiment. Population means and phenotypic variances of the two characters, as well as the phenotypic correlation between them, were obtained from the original samples from the population cages. As previously, the offspring–parent regression allows calculation of the heritabilities of both sternopleural and abdominal bristle number, and their genetic correlation; multiplication of the independently obtained phenotypic variance by the heritability gives an estimate of the additive genetic variance.

It may be appropriate to mention at this point that the criterion for a decision of significance of a comparison of either heritability or additive genetic variance is whether the upper and lower limits of the smaller and larger estimates, respectively, overlap. As the distribution of these values is not known, no formal test of significance is available, so these limits are attached by simply ± twice the standard error of the estimate. Even though one cannot produce an exact significance level by this method, non-overlapping ranges thus obtained imply an upper limit to the probability of Type I error to be 0·05.

(iv) *Abdominal bristle number*

A random sample of 400 males and 400 females from each cage was scored for abdominal chaeta number in the first year, and 200 males and 200 females in the second year. Population means and phenotypic variances of the sum of the two segments were obtained from these; in addition, since it is known that the genetic correlation of bristle number between any two abdominal segments is 1, and the environmental correlation 0 (Reeve & Robertson, 1954), the phenotypic correlation of score on two successive segments provides an estimate of the heritability of score of a single abdominal segment. The standard error of this heritability estimate is the standard error of a correlation. The additive genetic variance of the single segment score is then the phenotypic correlation (t) multiplied by the phenotypic variance of the single segment score (σ^2), where σ^2 may be determined from the phenotypic variance of the sum of the two segments (V_p) by the relationship $\sigma^2 = V_p/2(1+t)$. The standard error of the estimate of additive genetic variance is $\sigma^2\sqrt{[(1+t)^2/n]}$, where n is the total number of individuals measured. (I wish to thank Dr W. G. Hill for the derivation of these relationships.)

(v) *Body weight*

This trait was analysed in the second year only, utilizing a design appropriate to the detection of genotype–environment interaction. As this character was expected to be influenced more by environmental conditions than either bristle character, particular care was taken to ensure culture conditions as similar as possible for parents and offspring. Samples of eggs were collected from the population cages by allowing the flies to lay for several hours in small petri dishes filled

with culture medium. Fifty eggs were then transferred, using a stylet probe, on to the surface of either C or A medium in a vial; animals emerging from these vials were used as parents. One hundred and fifty 3-day-old males were weighed to the nearest 0·05 mg – of these, the 40 heaviest and 40 lightest individuals were mated at random to unmeasured females. The females were then allowed to lay approximately 50 eggs in each of 4 vials, 2 of each type of medium. Ten males from each of the four vials were then weighed *en masse*. Twice the regression of son on sire is an estimate of the heritability of the trait on either of the two alternative media, whereas the 'cross-regression' gives an estimate of the genotype–environment correlation, as described above. Additive genetic variance estimates are again the product of phenotypic variance and heritability of the trait.

Table 1. *Sternopleural bristle score*

(Population means ($\bar{X}$), phenotypic variances (V_P), heritabilities (h^2) obtained by offspring–parent regression, and additive genetic variances (V_A) (± standard errors). Two replicates (I, II) are presented for each population. Year 1)

	Substrate	$\bar{X}$ (± S.E.)	h^2 (± S.E.)	V_P	V_A (± S.E.)
			Control		
I	C	18·666 (0·123)	0·416 (0·030)	4·026	1·675 (0·126)
	A	17·811 (0·113)	0·422 (0·057)	3·450	1·456 (0·232)
II	C	18·336 (0·108)	0·377 (0·044)	3·111	1·173 (0·169)
	A	19·339 (0·122)	0·385 (0·045)	3·912	1·506 (0·219)
			Spatial variation		
I	C	18·658 (0·129)	0·583 (0·035)	4·460	2·600 (0·273)
	A	18·122 (0·132)	0·628 (0·053)	3·452	2·168 (0·284)
II	C	20·320 (0·142)	0·589 (0·041)	5·494	3·236 (0·358)
	A	19·130 (0·163)	0·699 (0·076)	4·832	3·378 (0·510)
			Temporal variation, short-term		
I	C	19·230 (0·158)	0·624 (0·059)	6·576	4·103 (0·527)
	A	19·952 (0·160)	0·654 (0·044)	6·825	4·464 (0·486)
II	C	18·830 (0·140)	0·595 (0·037)	5·244	3·120 (0·333)
	A	19·201 (0·142)	0·588 (0·037)	5·413	3·183 (0·342)
			Temporal variation, long-term		
I	C	19·005 (0·158)	0·637 (0·047)	6·805	4·335 (0·490)
	A	18·615 (0·145)	0·623 (0·049)	5·592	3·484 (0·407)
II	C	19·097 (0·139)	0·673 (0·048)	5·182	3·487 (0·390)
	A	20·715 (0·165)	0·579 (0·039)	7·261	4·204 (0·460)

3. RESULTS

(i) *Sternopleural bristle number*

Population means and phenotypic variances of sternopleural chaeta score are presented for each year separately in Tables 1 and 2. No significant difference in mean is observed according to substrate, between replicates, treatments, or successive years. Phenotypic variances are not significantly different between substrate or replicates within treatments, but there is a marked treatment effect in that both temporally varying cages have an increased phenotypic variance

compared to either control or spatially heterogeneous cages; this pattern persists over time. Such an increase is attributable to either an increase in genetic variance, or an increase in environmental variance, or both.

Estimates of heritability and additive genetic variance are given, for the first and second years, in Tables 1 and 2, respectively. Both sets of data exhibit the same pattern of results. Heritabilities are not significantly different between sub-

Table 2. *Sternopleural bristle score*

(Population means ($\bar{X}$), phenotypic variances (V_P), heritabilities (h^2) obtained by offspring–parent regression, and additive genetic variances (V_A) ($\pm$ standard errors). Year 2)

	$\bar{X}$ ($\pm$ S.E.)	h^2 ($\pm$ S.E.)	V_P	V_A ($\pm$ S.E.)
		Control		
I	19·133 (0·013)	0·287 (0·095)	4·217	1·210 (0·409)
II	18·843 (0·091)	0·487 (0·128)	3·347	1·630 (0·444)
		Spatial variation		
I	18·000 (0·103)	0·645 (0·072)	4·210	2·716 (0·359)
II	19·765 (0·097)	0·767 (0·072)	3·763	2·886 (0·339)
		Temporal variation, short-term		
I	19·118 (0·137)	0·765 (0·067)	7·540	5·632 (0·643)
II	18·478 (0·113)	0·751 (0·057)	5·138	3·859 (0·398)
		Temporal variation, long-term		
I	19·313 (0·137)	0·765 (0·101)	7·491	5·731 (0·858)
II	19·130 (0·124)	0·836 (0·045)	6·187	5·172 (0·458)
		Control/temporal variation, short-term		
I	19·510 (0·103)	0·320 (0·121)	4·253	1·361 (0·523)

strates or between replicates within populations, but are significantly higher in the three populations exposed to environmental variation than the control. This could not have occurred with constant additive genetic variances, unless the phenotypic variances had been reduced in the variable populations. In fact, we have seen that the opposite happened; therefore the additive genetic variance estimates (V_A) obtained indicate even more clearly the increased genetic variance in the environmentally varying cages, relative to the control. Spatially heterogeneous populations have approximately twice, and temporally varying cages approximately three times the additive variance of the control; these differences are significant for both years, although the magnitude of the difference is greater in the second year. Furthermore, the comparison of spatial environmental variation to temporal heterogeneity, although significant only for the second year, suggests that the former is less effective in the maintenance of genetic variability. There is no difference in the level of V_A between the two populations undergoing temporal variation of different periodicity.

Since an estimate of the initial level of genetic variation present was not made, the above results may be interpreted either as an initially high level of genetic variance declining in the control populations and remaining constant in the populations exposed to varying conditions, or as an initially low level of genetic variation

remaining constant in the control populations and increasing in environmentally variable populations. To clarify this ambiguity an additional cage which had been under control conditions for one year, and hence had known phenotypic and genetic variances, was transferred to conditions of short-term temporal variation for the second year. An increase of genetic variance in the second year would therefore support the second interpretation, whereas if the variance remains at the control level the first interpretation would be supported. The heritability and additive genetic variances observed in the second year for this population were equivalent to those of the two control cages (Table 2); thus, environmental variation can maintain genetic variation only at a level initially present in the population, and does not appear to increase genetic variation by differentially selecting genotypes specifically adapted to the different niches.

Table 3. *Genotype–environment correlations* ($\pm$ *standard error*) *of sternopleural bristle score on control and alcohol media*

	Replicate	
Population	I	II
Control	0·925 (0·013)	0·785 (0·039)
Spatial variation	0·999 (0·000)	0·737 (0·035)
Temporal variation		
Short-term	0·861 (0·018)	1·014*
Long-term	0·950 (0·007)	0·955 (0·005)

* Standard error undefined. All standard errors given here must be taken as approximations only, as the Robertson (1959) formula is not appropriate as the correlations approach unity.

This demonstration of an association between genetic and environmental variability does not, however, give an indication of causality – it is possible that either diversifying selection increases polymorphism in a heterogeneous habitat, or that heterozygosity *per se* is selected in a fluctuating environment. The genotype–environment correlation measures the extent to which the same genes are operating in each niche, and may be used to distinguish the two modes of adaptation to a variable environment. If microdifferentiation and specific adaptation to each habitat is the major factor promoting increased genetic variance in the variable environments, then the genotype–environment correlation would be expected to decrease in the treated populations, reflecting selection for divergent constellations of genotypes preferred in the alternate environments. If, however, the genotype–environment correlation is similar in both constant and variable populations, this would indicate that the same genotypes are selected in the different niches, and one must propose that these are heterozygous genotypes to account simultaneously for greater genetic variance observed in environmentally variable populations.

Estimates of the genotype–environment correlation between sternopleural bristle scores on the two media (Table 3) average 0·9 overall but are, with one exception, significantly different from one. However, the variation of the estimates of the correlation between replicates, within treatments, is greater than the overall

between-treatment variation. Since the observed pattern is not one of substantially lower genotype–environment correlations in the variable populations than the constant environment, we have no evidence of disruptive selection, and specific adaptation to each niche has not created the differences between populations in additive genetic variance. Although to some extent different genes affect bristle number in the two environments, these differences are not instrumental in determining the adaptation of the populations to different patterns of environmental variation; we may therefore hypothesize that it is heterozygous genotypes which are preferred in the variable habitats.

Table 4. *Abdominal bristle score*

(Population means ($\bar{X}$) and phenotypic variances (V_P) of sum of scores of 2 terminal segments, phenotypic variances (σ^2) of single segment score, heritabilities (h^2) obtained by correlation of score of adjacent terminal abdominal segments, and additive genetic variances (V_A) (± standard errors). Year 1)

	$\bar{X}$ (± S.E.)	V_P	σ^2	h^2 (± S.E.)	V_A (± S.E.)
			Control		
I	35·594 (0·125)	12·492	4·170	0·498 (0·031)	2·076 (0·165)
II	35·658 (0·124)	12·257	4·053	0·512 (0·030)	2·075 (0·161)
			Spatial variation		
I	37·904 (0·125)	12·591	4·197	0·500 (0·031)	2·099 (0·166)
II	37·445 (0·141)	15·628	4·832	0·617 (0·028)	2·982 (0·201)
			Temporal variation, short-term		
I	38·907 (0·138)	14·036	4·554	0·541 (0·030)	2·464 (0·183)
II	36·398 (0·125)	12·544	4·107	0·527 (0·030)	2·165 (0·164)
			Temporal variation, long-term		
I	37·766 (0·109)	9·441	3·515	0·343 (0·033)	1·206 (0·131)
II	36·915 (0·110)	9·680	3·349	0·445 (0·032)	1·491 (0·130)

(ii) *Abdominal bristle number*

Population means and phenotypic variances of abdominal chaeta score are presented for each year separately in Tables 4 and 5. There are no significant differences in either mean or variance between replicates, treatments or successive years, although it appears that the long-term temporal variation cages are somewhat less variable than the others. This pattern is in direct contrast with that observed for the sternopleural bristle scores; here the similarity of the phenotypic variances indicates that greater additive variance in variable populations than the control could be accommodated only by a concomitant and corresponding reduction in the environmental variance.

Estimates of heritability obtained by correlations of scores of two successive abdominal segments (Tables 4 and 5) are also remarkably constant between replicates, populations, and years, but again the estimates are lower (but not significantly) for the long-term temporally variable populations. Additive genetic variances computed from these data reflect the same trend; with these estimates

Table 5. *Abdominal bristle score*

(Population means ($\bar{X}$) and phenotypic variances (V_P) of sum of scores of 2 terminal segments, phenotypic variances (σ^2) of single segment score, heritabilities (h^2) obtained by correlation of score of adjacent terminal abdominal segments, and additive genetic variances (V_A) ($\pm$ standard error). Year 2)

	$\bar{X}$ ($\pm$ S.E.)	V_P	σ^2	h^2 ($\pm$ S.E.)	V_A ($\pm$ S.E.)
			Control		
I	34·773 (0·178)	12·648	4·236	0·493 (0·044)	2·088 (0·236)
II	36·325 (0·174)	12·059	4·147	0·454 (0·045)	1·883 (0·228)
			Spatial variation		
I	37·160 (0·162)	10·462	3·578	0·462 (0·045)	1·653 (0·197)
II	37·100 (0·170)	11·520	4·111	0·401 (0·046)	1·649 (0·221)
			Temporal variation, short-term		
I	40·203 (0·186)	13·778	4·693	0·468 (0·044)	2·196 (0·259)
II	36·395 (0·178)	12·605	4·250	0·483 (0·044)	2·053 (0·236)
			Temporal variation, long-term		
I	36·543 (0·163)	10·670	3·940	0·354 (0·047)	1·395 (0·209)
II	37·300 (0·152)	9·205	3·342	0·377 (0·046)	1·260 (0·179)
			Control/temporal variation, short-term		
I	36·485 (0·168)	11·317	3·951	0·432 (0·045)	1·707 (0·215)

Table 6. *Abdominal bristle score*

(Phenotypic variances (V_P), heritabilities (h^2) obtained by offspring–parent regression, and additive genetic variances (V_A) ($\pm$ standard errors) of sum of scores on adjacent terminal segments. Year 2)

	h^2 ($\pm$ S.E.)	V_P	V_A ($\pm$ S.E.)
		Control	
I	0·523 (0·107)	12·648	6·615 (1·429)
II	0·418 (0·129)	12·059	5·041 (1·594)
		Spatial variation	
I	0·492 (0·084)	10·462	5·147 (0·954)
II	0·463 (0·094)	11·520	5·334 (1·144)
		Temporal variation, short-term	
I	0·417 (0·092)	13·778	5·745 (1·334)
II	0·473 (0·108)	12·605	5·962 (1·427)
		Temporal variation, long-term	
I	0·396 (0·085)	10·670	4·225 (0·953)
II	0·325 (0·113)	9·205	2·992 (1·063)

the persistent suggestion that the long-term temporally varying populations are less variable than the others is significant in the first year. A similar pattern emerges when heritability of the sum of scores on adjacent terminal segments is estimated from the regression of offspring on parent in Year 2 (Table 6). Additive genetic variances here are not significantly different between replicates or among populations. The population which was initially treated as a control population, and then subjected to short-term environmental variation in the second year, also showed heritabilities and phenotypic variances similar to those of the other populations.

Given the totally different responses of the two characters, abdominal and sternopleural bristle number, to environmental heterogeneity, it is of interest to know to what extent the two traits are controlled by the same genes, as shown by the genetic correlation between them. Phenotypic and genetic correlations between abdominal and sternopleural chaeta score (Table 7) are for the most part small and positive, not significantly different from zero or from each other. The two bristle characters thus show virtual genetic independence, and are free to respond to selection in divergent manners.

Table 7. *Phenotypic* (r_p) *and genetic* (r_G) *correlations* ($\pm$ *standard error*) *of sternopleural and abdominal bristle scores*

Population	Replicate	r_p ($\pm$ S.E.)	r_G ($\pm$ S.E.)
Control	I	0·093 (0·050)	0·155 (0·180)
	II	0·082 (0·050)	0·067 (0·201)
Spatial variation	I	0·070 (0·050)	0·096 (0·097)
	II	−0·066 (0·050)	0·140 (0·096)
Temporal variation, short-term	I	0·197 (0·049)	0·202 (0·095)
	II	0·081 (0·050)	0·074 (0·093)
Temporal variation, long-term	I	0·131 (0·050)	0·014 (0·119)
	II	0·138 (0·050)	0·140 (0·095)
Control/temporal variation, short-term	I	0·186 (0·049)	0·099 (0·322)

(iii) *Body weight*

Population means and variances of body weight are given in Table 8; this character was analysed in the second year only. Neither means nor phenotypic variances are significantly different between substrate, replicate, or treatment; however, there are significant differences in additive genetic variances among the populations, which must have been accompanied by concomitant reductions in either environmental or non-additive genetic variance. Further examination of Table 8, in which heritabilities and additive genetic variances are presented for each population, indicates that heritabilities do not vary significantly between replicate or substrate within treatments, but spatially varying populations have heritabilities twice as large as the controls and both temporally varying populations three times as large. The additive genetic variances reflect similar significant changes.

Estimates of the genotype–environment correlation between body weights on the two media average greater than one (Table 9). On the whole, it must be concluded that no evidence exists for correlations being differentially reduced in populations undergoing environmental variation, compared to the control value. The same genes determine the character in each environment; it does not appear that diversifying selection maintains genetic variation for this character by selecting alternate genotypes in the two environments.

Table 8. *Body weight*

(Population means ($\bar{X}$), phenotypic variances (V_P), heritabilities (h^2) obtained by offspring–parent regression, and additive genetic variances (V_A). Standard errors attached to the estimates of additive genetic variance are all of the order of 10^{-6}; the observed differences between populations are therefore significant. Year 2)

	Substrate	$\bar{X}$ (± S.E.)	h^2 (± S.E.)	V_P	V_A
		Control			
I	C	0·907 (0·0065)	0·035 (0·098)	0·0063	0·0002
	A	0·951 (0·0070)	0·284 (0·073)	0·0074	0·0021
II	C	0·910 (0·0073)	0·268 (0·065)	0·0081	0·0022
	A	0·928 (0·0069)	0·202 (0·119)	0·0072	0·0015
		Spatial variation			
I	C	0·903 (0·0076)	0·458 (0·114)	0·0086	0·0039
	A	0·943 (0·0072)	0·478 (0·129)	0·0078	0·0037
II	C	0·992 (0·0091)	0·439 (0·113)	0·0124	0·0054
	A	0·911 (0·0071)	0·439 (0·119)	0·0076	0·0033
		Temporal variation, short-term			
I	C	0·912 (0·0058)	0·697 (0·138)	0·0050	0·0035
	A	0·954 (0·0076)	0·651 (0·071)	0·0087	0·0057
II	C	0·939 (0·0064)	0·632 (0·117)	0·0062	0·0039
	A	0·891 (0·0075)	0·594 (0·140)	0·0085	0·0050
		Temporal variation, long-term			
I	C	0·863 (0·0072)	0·623 (0·103)	0·0078	0·0049
	A	0·952 (0·0078)	0·541 (0·081)	0·0092	0·0050
II	C	0·888 (0·0072)	0·618 (0·091)	0·0078	0·0048
	A	0·908 (0·0066)	0·666 (0·185)	0·0066	0·0044

Table 9. *Genotype–environment correlations* (± *standard error*) *of body weight on control and alcohol media*

	Replicate	
Population	I	II
Control	1·525*	1·264*
Spatial variation	0·934 (0·023)	0·975 (0·009)
Temporal variation		
Short-term	0·938 (0·012)	1·063*
Long-term	1·065*	1·038*

* Standard error undefined.

4. DISCUSSION

These experiments were conducted under controlled laboratory conditions; therefore, the environmental treatment imposed was the agent determining differences in level of genetic variability between populations. Each population was initiated from a sufficiently large sample of the base population to ensure adequate replication of the original constellation of genotypes, and to preclude genetic drift as a source of the differential response. We are now able to utilize the

experimental evidence to assess empirically the questions initially formulated concerning the relationship between genetic and environmental variation. Is genetic variance maintained in a variable environment? The answer is: 'yes, sometimes'. Three quantitative characters were analysed, and three patterns of response to environmental heterogeneity were observed. These patterns can be described in terms of the associations between environmental variability and the phenotypic, additive genetic, and environmental variances of the three metric traits. The phenotypic and additive genetic variances of sternopleural bristle number are substantially and significantly greater in populations experiencing spatial and temporal environmental variation than in the control populations, while the environmental variance is equivalent in all populations; for this character, genetic variance is certainly maintained under environmentally varying conditions. Additive genetic variance of body weight similarly exceeds the level of control populations in the three variable populations; however, the phenotypic variance of body weight is equivalent in each population, so that the environmental and/or non-additive genetic variance is consequently reduced in the variable populations. Yet a third pattern is that determined for the second bristle character, abdominal chaeta number – neither phenotypic, additive, nor environmental variances are consistently affected by exposing populations to varying environments. The opposing responses of the two bristle characters are particularly intriguing; although they have been shown to be genetically uncorrelated, and thus capable of divergent responses to selection, *Drosophila* bristles fulfil the same functional requirement in that they are sensory receptors, and therefore intuitively should perceive the same environmental cues irrespective of their location on the animal.

What is the relationship between genetic variance and pattern of environmental heterogeneity experienced? The two characters for which an association between genetic and environmental variance has been demonstrated (sternopleural chaeta number and body weight) show two different responses to the environmental variability to which they were exposed. Additive genetic variance for body weight is similar among the three environmentally varying populations, whereas additive genetic variance for sternopleural bristle number is significantly less in the spatially heterogeneous populations than in either of the populations experiencing a pattern of long- or short-term temporal variability. This is in contrast to predictions from all theoretical studies, which uniformly agree temporal variation should be much less effective than spatial heterogeneity in promoting the maintenance of genetic variability (Felsenstein, 1976). Bryant (1976), however, has argued that spatial variability is an entirely predictable component of environment and that spatially maintained genetic variation may therefore be a transitional state toward speciation. Temporal environmental variation, which represents an uncertainty provoking a more general rather than a specific genetic response, should therefore more often be associated with genetic variance than spatial heterogeneity. The results here are in accord with this prediction. Theory also predicts that periodicity of temporal environmental variation determines its relative efficiency, long cycles being less effective than short-term environmental variation (Hedrick, 1974,

1976). Environmental grain is also theoretically important, genetic variation being less likely in fine- than coarse-grained environments (Gillespie, 1974; Strobeck, 1975; Templeton, 1977). Neither of these effects was observed in this study, however; additive genetic variance was the same in both long-term (coarse-grained) and short-term (fine-grained) temporally varying populations.

By what mechanism is genetic variability preserved in heterogeneous environments? Genotype–environment interactions between the environmental variable and the character would indicate that it is selection of alternative genotypes in the different niches. If, however, the genotype–environment correlation does not depart significantly from one, the genetic variation would be general and not a character-specific response to environmental uncertainty. The latter was the response observed, it appears that selection for heterozygosity *per se*, rather than for specialization to the two environmental states, allows maintenance of genetic variation in populations experiencing environmental heterogeneity. The observations of both a general response to environmental variability and of temporal variation being equally, if not more, efficient than spatial variation in the maintenance of additive genetic variance (despite theoretical considerations) are in accord with Bryant's (1976) suggestion that it is the temporal element of instability to which the populations are responding. The discovery of a general response to a varying environment implies also that the functional interaction of a particular character with the varying environmental parameter is not critical in the determination of the response. While not providing a mechanistic explanation for the different patterns of response of the two bristle characters to the same environmental heterogeneity, this observation does eliminate one source of confusion concerning the outcome – the assumption that because the two characters are responsible for perceiving the same environmental stimuli, they should behave similarly to variation in these stimuli. This has been shown to be inapplicable; specific interaction of a character with the environmental variable is not a determinant of the maintenance of genetic variation of that character.

The question of what exactly is being selected as the environment varies remains unanswered. One feature common to all experimental work on the subject is the generality of the effect of environmental variation on the maintenance of genetic variation. Whether the environmental variables are temporal and/or spatial, long- or short-term, the genetic variance of electrophoretic, karyotypic, or quantitative characters is greater in variable populations than controls (Beardmore, Dobzhansky & Pavlovsky, 1960; Beardmore, 1961; Beardmore & Levine, 1963; Long, 1970; Powell, 1971; McDonald & Ayala, 1974; Powell & Wistrand, 1978). These results all indicate that selection may be for heterozygotes; at the enzyme level, the presence of several active gene products may confer adaptive biochemical diversity in the face of varying environmental conditions. However, the rationale for investigating selection in variable environments was both to circumvent theoretical problems and the lack of experimental evidence for heterosis as a cause of selective maintenance of polymorphisms. From the results of this experiment, one may speculate that heterosis may only be adaptive in variable environments;

the next stage is to measure heterozygote advantage, not under constant conditions, but in the face of environmental uncertainty.

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