J. evol. Biol. 2: 95–107 (1989)

1010-061X/89/02095-13 $ 1.50 + 0.20/0

The genetics of phenotypic plasticity I. Heritability

Samuel M. Scheiner and Richard F. Lyman

Department of Biological Sciences, Northern Illinois University, DeKalb, Illinois 60115, USA

Key words: Drosphila melanogaster; heritability; phenotypic plasticity; thorax size; variance components.

Abstract

Methods for estimating the genetic component of phenotypic plasticity are presented. In the general case of clonal replicates or full-sibs raised in several environments, the heritability of plasticity can be measured as the ratio of the genotype-environment interaction variance to the total phenotypic variance. In the special case of only two environments plasticity also can be measured as the difference among environments in genotype or family means. In that case, the heritability of plasticity can be measured as either a ratio of variance components or as the slope of a parent-offspring regression. The general measure suffers because no least-square standard errors have been developed, although they can be calculated by maximum-likelihood or bootstrapping techniques. For the other two methods least-square standard errors can be calculated but require very large experiments for statistical significance to be achieved. The heritability measures are compared using data on plasticity of thorax size in response to temperature in *Drosophila melanogaster*. The heritability estimates are all in close agreement. Models of the evolution of phenotypic plasticity have treated it as a trait in its own right and as a cross-environment genetic correlation. Although the first approach is the one used here, neither one is preferred.

Introduction

How species evolve in response to complex environments is receiving increasing attention (e. g., Hedrick, 1986; Schlichting, 1986). One way in which an individual can adapt to a complex environment is to be phenotypically plastic. That is, the individual's developmental program alters in response to the external environment.

Wright (1931) has suggested that individual adaptability, phenotypic plasticity, is "perhaps the chief object of selection." In order to study how adaptation through phenotypic plasticity can occur information on genetic variation in plasticity is needed.

In this paper we develop and compare methods of quantifying the genetic component of phenotypic plasticity. Previous studies have shown that phenotypic plasticity is a heritable trait which varies among populations and species (Schlichting, 1986). Yet, there have been no recent attempts to quantify what portion of the measured amounts of phenotypic plasticity is heritable. Knowing the heritability is important if we are to make predictions about the response of a population to environmental variation. For example, suppose that in a complex environment there was equal selection for individuals to specialize on particular environments or to increase their plasticity. If the plasticity of a trait had a greater heritability than the trait itself we might expect the primary evolutionary response to be an increase in the plasticity of individuals. Conversely, if plasticity had a low heritability we might expect specialized individuals to evolve.

Our purpose is two-fold. First, we present definitions of plasticity and the heritability of plasticity. In assembling this information we present new definitions as well as generalize measures previously developed. Second, we use data on the plasticity of thorax size in *Drosphila melanogaster* to compare and cross-validate the various measures of heritability. This compilation and synthesis is intended as a guide in experimental design and data analysis.

Measuring phenotypic plasticity

Scheiner and Goodnight (1984) showed that the common verbal description of phenotypic plasticity as the change in phenotype caused by a change in the environment (Bradshaw, 1965) translates into the sum of the environmental variance and genotype-environment interaction variance in a two-way analysis of variance. Plastic variance was defined as:

$$\sigma^2_{PL} = \sigma^2_E + \sigma^2_{G \times E} \tag{1}$$

where σ^2_E is environmental variance and $\sigma^2_{G \times E}$ and is genotype-environment interaction variance. Plasticity was defined as:

$$\sigma^2_{pl} = \frac{\sigma^2_{PL}}{\sigma^2_P} \tag{2}$$

where σ^2_P is total phenotypic variance. We make a correction here to the definitions provided by Scheiner and Goodnight (1984). For the case of plasticity measured in a half-sib mating design a factor of 2 was left out of the estimate of $\sigma^2_{G \times E}$. If the dominance-environment interaction is small, the correct estimate of plasticity is:

$$\sigma^2_{pl} = \frac{\sigma^2_E + 2(\sigma^2_{S \times E} + \sigma^2_{D \times E})}{\sigma^2_P} \tag{3}$$

where $\sigma^2_{S \times E}$ is the sire-environment interacton variance and $\sigma^2_{D \times E}$ is the dam-environment interaction variance. If the dominance-environment interaction is

large, the correct estimate of plasticity is:

$$\sigma^2_{pl} = \frac{\sigma^2_E + 4\sigma^2_{S \times E}}{\sigma^2_P}. \tag{4}$$

From the above definitions we can see that the heritable component of the plastic variation consists of the genotype-environment interaction, or

$$h^2_{pl} = \frac{\sigma^2_{G \times E}}{\sigma^2_P}. \tag{5}$$

This definition has been presented previously in the specific cases of a half-sib mating design (Becker, 1964) and a full-sib mating design (Scheinberg, 1973). See Becker (1984) for the calculation of $\sigma^2_{G \times E}$ with various experimental designs. A discussion of optimal sample sizes for standard diallel and nested design experiments is given by Pederson (1972). He recommends using 3–5 crosses per parent or dams per sire. The number of replicates per full-sib family will be a function of the number of crosses with 5 often being sufficient. Similar considerations will hold for estimates of the heritability of plasticity except that the number of replicates per family will need to be multiplied by the number of environments.

There is one limitation to the use of the above measure of the heritability of plasticity; no least-square estimates of standard errors have been developed for variance components in a mixed cross-nested design experiment. A solution can be found through the use of maximum-likelihood estimation (Shaw, 1987) or bootstrapping techniques (Efron, 1981). However, as will be shown below, these standard errors are often quite large.

Plasticity and heritability of plasticity of necessity can be measured only under experimental conditions since genotypes must be replicated and distributed among environments. Additionally, in nature both genotypes and environments will be random variables while under experimental conditions the latter are usually restricted to a few fixed treatments. Thus, measures of the heritability of plasticity are limited by the extent to which the experimental conditions mimic natural variation. Specifically, the frequency distribution of environments in nature effects the environmental variance and thus the amounts of plastic variance, total phenotypic variance, and trait plasticity. Although the shape of the reaction norms does not change, the amount of genotype-environment interaction variance is a function of environmental frequencies. Thus, the heritability of plasticity is dependent on the distribution of environments. This problem is similar to that faced in extrapolating a measure of trait heritability from a relatively simple laboratory environment to a more complex natural setting. We suggest that the heritability measures defined here have their greatest utility as either strictly relative measures of trait heritability and trait plasticity heritability or for predictions of response to selection under specified experimental conditions. We emphasize that plasticity measures must be specified relative to a given set of environments.

A special case for measuring phenotypic plasticity comes from considering the plasticity of an individual or genotype to be the mean difference in the performance of his/her offspring or clonal replicates reared in two environments. For this special case least-square estimates of the standard errors of heritability have been

developed. The disadvantage of using the mean difference is that it is only defined for plasticity over two environments. The interaction variance can be calculated over any number of environments.

The heritability of cross-environment mean differences is:

$$h^2_{GM} = \frac{\sigma^2_{FM}}{\sigma^2_P} \tag{6}$$

where σ^2_{FM} is the variance among families of genotype mean differences for clonal replicates or the variance among sires of dam mean differences for a half-sib design. Heritability can only be measured for designs involving multiple matings (e. g., half-sib or diallel) since each full-sib family or genotype provides only a single difference measure. The above definition has been provided earlier in the specific case of sexual dimorphism (Eisen and Legates, 1966). We note that this formulation can be extended to any trait which must be measured as an aggregate (e. g., variation in progeny phenotype, mutation rate).

When mean differences are used the total phenotypic variance of the dam or genotype mean differences is a biased estimate of the phenotypic variance of that trait. The correct estimate of the total phenotypic variance requires an addition of the error variance due to genetic, environmental, and measurement variation among full-sibs within each environment or environmental and measurement variation among clonal replicates within each environment. The correct phenotypic variance is given by the formula:

$$\sigma^2_P = \sigma^2_{GM} + \frac{(\sigma^2_{e1} + \sigma^2_{e2})}{1 - 2(n_1 + n_2)} \tag{7}$$

where σ^2_{GM} is the total phenotypic variance of the genotype or dam mean differences, σ^2_{e1} and σ^2_{e2} are the error variances from the one-way ANOVAs for environments 1 and 2, and n_1 and n_2 are the number of replicates per genotype or offspring per dam measured in each environment (modified from Eisen and Legates, 1966). This bias in the estimate of the phenotypic variance declines as the number of replicates per environment increases. Standard errors for h^2_{GM} for balanced and unbalanced designs are given by Becker (1984, pp. 48, 52) but substituting σ^2_P as above.

Using the mean difference to measure heritability allows for another, albeit more complex, method of determining heritability, parent-offspring regression. In this technique the full-sib offspring of a parent are raised in two environments as before. However, unlike the usual case, the offspring are being used as the measure of the parent's phenotype. This seems odd, but it occurs because plasticity is a trait of a genotype, not an individual. To obtain the phenotypic scores of the offspring a third generation must be measured. Full-sibs of the F_1 generation are mated, and the F_2 offspring again are raised in two environments. The phenotypic scores of the F_1 generation are the mean differences of the F_2 sibships. The heritability of plasticity is the slope of the parent-offspring regression:

$$OMD = h^2_{pl} PMD + \text{intercept}\,, \tag{8}$$

where PMD is the mean difference in the F_1 offspring of the parental families and OMD is the mean difference in the F_2 offspring of the F_1 families. The standard error

error is the standard error of the regression slope. This method will tend to overestimate the heritability because of the necessity of using inbred F_2 individuals (Becker, 1984, p. 35).

The advantage of this method is that the statistical properties of regression parameters are well understood, unlike the properties of variance components. There are two disadvantages. (1) The amount of work increases because an extra generation is necessary. (2) The power of the test decreases because the power is a function of the number of families not the total number of individuals. One caution, to guard against effects of maternal environment the F_2 progeny should be produced from F_1 individuals raised in both environments, and seperate regressions should be computed. If no maternal effects are found the data can then be pooled.

Beyond the metrics of plasticity dealt with here, three other methods of measurement have been used. Via and Lande (1985, 1987) defined plasticity as the difference in mean expression of a trait in two environments and its genetic component as the genotype-environment interaction variance, expressed for convenience as the cross-environment genetic correlation. Via (1984) presents several methods for calculating the cross-environment genetic correlation for a full-sib design experiment. We note that the use of variance components from a two-way ANOVA to estimate the cross-environment genetic correlation (Yamada, 1962) has been shown to be highly biased (Fernando et al., 1984). Joint regression analysis was developed to compare plasticities among inbred crop strains (Freeman, 1973). As such, it is not useful for evaluating natural populations, and the mathematical foundations of the method have been brought into question (Westcott, 1986). The coefficient of variation of treatment means has been used as a measure of plasticity (Schlichting and Levin, 1984). As proposed, there is no genetic component in that measure.

Materials and methods

We measured the heritability of plasticity of thorax size as affected by temperature in *Drosophila melanogaster*. A population line was established with 301 individuals captured over a one month period in several locations in the DeKalb area. The flies were maintained in mass culture at 21° C for two to three months prior to the start of the experiment.

Sibships were established by collecting 46 males and 138 femalses as newly eclosed adults. The flies were aged for one day. Each male was allowed to mate for 24 h with three females. The females were separated and allowed to oviposit for six days, being given new food vials daily. The food was standard cornmeal-molasses-agar supplemented with live yeast. The vials from alternate days were placed at either 19° C or 25° C such that each male-female combination produced three vials at each temperature. After emergence thorax size was measured on three females from each vial, nine flies per family per temperature, using a LASICO ocular filar and S-4A Auto-processor with a Wild stereomicroscope. The F_2 generation was established by taking a male-female pair from the middle vial of the three at each temperature, or two F_2 pairs for each of the original dams. The females again laid eggs in six vials, three per temperature. Three offspring per vial were measured as before.

For the data analysis we tried to balance the data as much as possible. To do this we eliminated all dams that did not have at least six offspring from at least two vials in each temperature and all sires that did not have at least two dams. Only lines that produced sufficient numbers of F_2 offspring were used. The final data set consisted of 37 sires mated to 88 dams. The F_1 generation consisted of 632 and 606 flies at 19° C and 25° C, respectively. The F_2 generation from F_1 parents raised at 19° C consisted of 505 and 509 flies at 19° C and 25° C, respectively. The F_2 generation from F_1 parents raised at 25° C consisted of 447 and 482 flies at 19° C and 25° C, respectively.

The analyses of variance were done using SAS procedure GLM (SAS Institute Inc., 1985). Temperature was designated as a random effect contrary to the typical practice of designating treatments as fixed effects. However, in the present case we were estimating the variation in plasticity that would exist if both genotypes and environments were randomly distributed as in a natural population. Variance components for the two-way ANOVA and their standard errors were estimated by restricted maximum-likelihood (REML) methods using BMDP3V (Jennrich and Sampson, 1983). Variance components for the within environment one-way ANOVAs were estimated by least-square methods using procedure NESTED. Least-square methods were used because least-square standard errors were calculated, but REML estimates were virtually identical. All data conformed to the assumptions of analysis of variance. Standard errors for the sib correlations were calculated as by Becker (1984, pp. 52, 59). Regression analyses were done using SYSTAT (Wilkinson, 1984). Standard errors for the regression coefficients were calculated as by Zar (1984, p. 272).

Results

The results of the analyses of variance of the F_1 generation are shown in Table 1. As expected a significant genetic component was found. For the two-way ANOVA the sire-temperature interaction variance was of marginal significance, but the dam-temperature interaction variance was highly significant. Variance components are shown in Table 2. The sire variance components were approximately equal, 4.6 % different, in the two temperatures. The dam variance component was larger than the sire component at both temperatures indicating non-additive genetic and/or maternal effects especially at low temperature. Non-additive/maternal effects especially were also seen in the dam-temperature interaction variance, so plasticities were calculated using only the sire-temperature interaction component. The plasticity of thorax size was 0.260.

The heritability of plasticity was calculated using three methods, variance components from a sib correlation two-way ANOVA (Eq. 5), variance components from an analysis of cross-environment dam mean differences (Eq. 6), and the slope from a parent-offspring regression of dam mean differences (Eq. 8). Note that the sib correlation and parent-offspring regression use different data for their estimates and thus provide a cross-validation of the techniques. Strictly for the purpose of

Table 1. Analysis of variance of thorax size of F_1 flies raised at 19° C and 25° C. Values shown are $\times 10^3$.

A. Analysis within each treatment.

	Temperature					
		Low			High	
Source	*df*	Mean Squares	$P <$	*df*	Mean Squares	$P <$
Sire	36	4.116	0.02	36	3.992	0.02
Dam (Sire)	51	2.268	0.0001	51	2.071	0.001
Error	544	0.937		518	1.156	
Total	631	1.236		605	1.398	

B. Analysis of dam means.

		Temperature							
		Low			High			Difference	
Source	*df*	Mean Squares	$P <$	*df*	Mean Squares	$P <$	*df*	Mean Squares	$P <$
Sire	36	0.593	0.02	36	0.596	0.02	36	0.723	0.06
Error	51	0.307		51	0.319		51	0.453	
Total	87	0.425		87	0.433		87	0.565	

C. Analysis of treatment interactions.

Source	*df*	Mean Squares	Error term	$P <$
Temperature	1	100.829	Sire × Temp	0.0001
Sire	36	5.687	Sire × Temp	0.004
Sire × Temp	36	2.327	Dam × Temp	0.07
Dam (Sire)	51	2.729	Dam × Temp	0.02
Dam × Temp	51	1.502	Error	0.0001
Error	1062	1.044		
Total	1237	1.400		

Table 2. Variance components of thorax size for F_1 flies raised at two temperatures. Values shown are $\times 10^4$.

A. Analysis within each treatment.

	Temperature	
Source	Low Variance	High Variance
Sire	1.13	1.08
Dam (Sire)	1.92	1.39
Error	9.37	11.56

B. Analysis of dam means.

	Temperature		
Source	Low Variance	High Variance	Difference Variance
Sire	1.21	1.17	1.13
Error	3.07	3.19	4.53

C. Analysis of treatment interactions.

Source	Variance
Temperature	1.78
Sire	0.56
Sire × Temp	0.53
Dam (Sire)	0.93
Dam × Temp	0.72
Error	10.41

comparing methods we also calculated the heritability of thorax size at each temperature using the two dam mean techniques, the standard sib correlation technique (Becker, 1984, p. 55), and the standard parent-offspring regression technique (Becker, 1984, p. 103). We realize for these latter estimates that the dam mean measures are redundant. They are presented for comparative purposes only.

The heritability of thorax size at 19° C was estimated as 0.352 and 0.363 using standard parent-offspring regression and sib correlation techniques, respectively (Table 3). At 25° C heritability was estimated as 0.314 and 0.307. These values are comparable to previous estimates (Robertson, 1957, 1960). Heritabilities did not differ significantly between temperatures. As expected the two methods resulted in almost identical estimates. Standard errors of the estimates were small, in part because the experiment was close to optimal design (Falconer, 1981, p. 168). Estimates of heritabilities using dam means gave results very close to those using individual measurements. Values based on sib correlations were slightly inflated relative to the other three measures. This bias comes about because the error mean squares from the one-way ANOVAs were used to estimate the errors in the dam

Table 3. Heritabilities of thorax size and plasticity of thorax size estimated using individual measurements and dam means by the methods of parent-offspring regression, b (SE), and sib correlation variance components, t (SE). Significance levels are based on ANOVA and regression, not the estimated standard errors.

	Individuals		Dam means	
	b	t	b	t
Thorax size				
Low temp.	0.352*** (0.041)	0.363*** (0.034)	0.359** (0.110)	0.393** (0.118)
High temp.	0.314*** (0.038)	0.307*** (0.034)	0.316** (0.116)	0.329* (0.117)
Combined	—	0.160** (0.137)	—	—
Plasticity of thorax size	—	0.150$^+$ (0.110)	0.184 (0.112)	0.193$^+$ (0.116)

$^+ P < 0.07$; $^* P < 0.05$; $^{**} P < 0.01$; $^{***} P < 0.001$

mean measures. The standard errors of the dam mean measures were 2 to 4 times larger than the others mainly due to the 100-fold decrease in sample size going from individuals to means. We also estimated the heritability of thorax size from the combined two-way ANOVA. The heritability was approximately half that estimated in each environment separately due to genetic variation taken up by the interaction component. This last heritability estimates what would occur in a natural population with a variable environment.

Estimates of the heritability of plasticity showed good agreement among the three methods, and they were not statistically significantly different (Table 3). For the regression of dam means there were no significant differences in the slopes for F_1 parents raised in different temperatures, so the data from the two temperatures were pooled. The heritability of plasticity of thorax size was about half that of the heritability of thorax size itself. The heritability estimates were marginally statistically significant for the sib correlation estimates and not significant for the parent-offspring regression estimate.

Discussion

We have presented several methods for measuring the heritability of phenotypic plasticity and shown that estimates using these methods are in good agreement. These methods have various strengths and weaknesses. One weakness brought out by our data is the low power of all of the methods as indicated by the marginal statistical significance of the heritability measures. Plasticity of a trait may often have a lower heritability than the heritability of the mean of the trait. To achieve

statistical significance with the use of dam means may require very large experiments carried out over two generations. A one generation estimate is preferable. Even then, the maximum-likelihood standard errors are large. Shaw (1987) showed that for many breeding designs maximum-likelihood techniques have lower power than least-square techniques. For example, the standard error of the heritability of thorax size for the two environments combined indicated that the estimate was not significantly different from zero (Table 3) despite the fact that the ANOVA showed a statistically significant sire variance component (Table 1).

A critical test for the existence of heritable variation is a selection experiment. We have selected for increased and decreased thorax size mean differences across environments with lines derived from the base population measured here. After 16 generations of selection we have had a significant response. The details of that experiment will be reported in the next paper in this series. This result is further demonstration of the low power in these methods of estimating the heritability of plasticity.

In the study of phenotypic plasticity this paper represents only a first step in the understanding of the genetic basis of this developmental property. Further work is needed in two areas. We discuss below approaches to the integration of phenotypic plasticity into evolutionary models. Empirical data is also needed to guide those models. Future papers in this series will examine such data relating to the effects of selection on the phenotypic plasticity of thorax size in response to temperature and whether there exist genes which affect the plasticity of a trait independent of effects on the mean of that trait.

The integration of phenotypic plasticity into evolutionary models has proceeded from two directions. Lynch and Gabriel (1987) define a parameter, the variance in environmental tolerance, z_2 in their notation, which is equivalent to phenotypic plasticity. Phenotypic plasticity is treated as a trait in its own right, divided into genetic and non-genetic components, and both are permitted to vary. They show that both temporal and spatial heterogeneity will select on phenotypic plasticity. Their treatment of phenotypic plasticity differs somewhat from that presented here. First, their model deals explicitly with fitness. Second, their measure of plasticity is the amount of curvature in a Gaussian normal curve of fitness over an environmental gradient. Our definition of plasticity is more general with no particular shaped curve implied and can be measured over as few as two environments. However, we wish to emphasize the basic similarity of our approaches, treating phenotypic plasticity as a trait with a heritable component. For example, their model could be extended to specific traits which affect fitness such as size. To do this, it is necessary to develop a mapping of trait plasticity onto fitness plasticity. Perhaps this mapping may show how more general shapes of plasticity curves can be resolved with Gaussian fitness functions. Weis and Gorman (1987) have done one such mapping in the case of a linear plasticity response by using analysis of covariance. Implicit in these types of models is the existence of genes affecting the plasticity of a trait. Such genes would have an epistatic effect on genes determining the mean of the trait. Needed are multilocus models exploring this epistasis and empirical information on the existence and nature of such genes.

Via and Lande (1985) take a different approach in their model for the evolution of a trait expressed in two environments. In this model the traits expressed in each environment are treated as different traits, and a cross-environment genetic correlation is measured (Robertson, 1959; Yamada, 1962; Becker, 1964; Eisen and Legates, 1966). Plasticity evolves through changes in the mean expression of the trait in each environment, and this change is unbounded. The genetic component of the plasticity, the cross-environment correlation, is assumed to be constant. This assumption is an extension of the standard assumption of all similar models that the genetic variance/covariance matrix is constant through time and was shown to hold in the restricted case of additive gene action and panmixia (Via and Lande, 1987). There is a direct connection between the approach of Via and Lande and that presented here; the cross-environment correlation is a transformation of the genotype-environment interaction variance (Yamada, 1962). In terms of our definition of plasticity, their model is the equivalent of letting the environmental variance evolve while holding the genotype-environment interaction variance constant.

We disagree with the statement of Via and Lande (1985) that "the correlation format is also more useful mathematically, because estimates of genotype-environment interaction cannot be incorporated into any current theory of evolution." As indicated above, the solution to this problem is to consider the plasticity of a trait as a trait itself (Lynch and Gabriel, 1987). Treating plasticity as a trait in its own right allows for possibilities not considered by Via and Lande. For example, a trait can evolve as a correlated response to selection on the plasticity of that trait or there can be joint evolution on the plasticities of different traits (Scheinberg, 1973). In the model of Via and Lande such interactions of traits and plasticities would be contained in the covariance terms, which are not permitted to evolve in their system. Genes for the plasticity of a trait may be separate from those responsible for the expression of that trait. Thus, they may evolve separately, and models need to incorporate those possibilities. Models have been developed of the evolution of variable progeny (e. g., Jaenike, 1978; Price and Waser, 1982; Kaplan and Cooper, 1984) and the source of that variability could be phenotypic plasticity in response to an individual's social environment (Fagen, 1987). Finally, the cross-environment correlation is inherently a pairwise measure treating each environment as a discrete entity. As the number of environments multiply, the number of correlation coefficients increase by the square of that number. In contrast, variance components or measures of fitness function curvature can encompass an entire environmental gradient in a single number. We favor neither one type of model nor the other. They are different ways of viewing the same phenomenon. Both methods can be used within current evolutionary theories depending upon the problem at hand.

Acknowledgments

We thank Fred Cohan and Bill Etges for advice on experimental design, Roberta Caplan for invaluable assistance, Brent Jacobsen and Art Weis for supplying flies, and Roberta Caplan and Art Weis for comments on the manuscript. Funding was provided by National Institutes of Health GM 38201 and Northern Illinois University.

References

Becker, W. A. 1964. Heritability of a response to an environmental change in chickens. Genetics 50: 783–788.

Becker, W. A. 1984. Manual of Procedures in Quantitative Genetics. Academic Enterprises, Pullman, WA.

Bradshaw, A. D. 1965. Evolutionary significance of phenotypic plasticity in plants. Adv. Genet. 13: 115–155.

Efron, B. 1981. Nonparametric estimates of standard error: The jackknife, the bootstrap and other methods. Biometrika 68: 589–599.

Eisen, E. J., and J. E. Legates. 1966. Genotype sex interaction and the genetic correlation between the sexes for body weight in *Mus musculus*. Genetics 54: 611–623.

Fagen, R. 1987. Phenotypic plasticity and social environment. Evol. Ecol. 1: 263–271.

Falconer, D. S. 1981. Introduction to Quantitative Genetics, 2nd ed. Longman, London.

Fernando, R. L., S. A. Knights, and D. Gianola. 1984. On a method of estimating the genetic correlation between characters measured in different experimental units. Theor. appl. Genet. 67: 175–178.

Freeman, G. H. 1973. Statistical methods for the analysis of genotype-environment interactions. Heredity 31: 339–354.

Hedrick, P. W. 1986. Genetic polymorphism in heterogeneous environments: A decade later. Ann. Rev. Ecol. Syst. 17: 535–566.

Jaenike, J. 1978. An hypothesis to account for the maintenance of sex within populations. Evol. Theory 3: 191–194.

Jennrich, R., and P. Sampson. 1983. General mixed model analysis of variance, pp. 413–426. *In* W. J. Dixon (ed.), BMDP Statistical Software. University of California Press, Berkeley, CA.

Kaplan, R. H., and W. S. Cooper. 1984. The evolution of developmental plasticity in reproductive characteristics: An application of the "adaptive coin-flipping" principle. Am. Nat. 123: 393–410.

Lynch, M., and W. Gabriel. 1987. Environmental tolerance. Am. Nat. 129: 283–303.

Pederson, D. G. 1972. A comparison of four experimental designs for the estimation of heritability. Theor. appl. Genet. 42: 371–377.

Price, M. V., and N. M. Waser. 1982. Population structure, frequency-dependent selection, and the maintenance of sexual reproduction. Evolution 36: 35–43.

Robertson, A. 1959. The sampling variance of the genetic correlation coefficient. Biometrics 15: 469–485.

Robertson, F. W. 1957. Studies in quantitative inheritance XI. Genetic and environmental correlation between body size and egg production in *Drosophila melanogaster*. J. Genet. 55: 428–443.

Roberston, F. W. 1960. The ecological genetics of growth in *Drosophila* 2. Selection for large body size on different diets. Genet. Res. 1: 305–318.

SAS Institute Inc. 1985. SAS User's Guide: Statistics, Version 5 Edition. SAS Institute Inc., Cary, NC.

Scheinberg, E. 1966. The sampling variance of the correlation coefficients estimated in genetic experiments. Biometrics 22: 187–191.

Scheinberg, E. 1973. The design and analysis of genetic experiments to modify genotype-environment interaction. I. Single pair matings and selection using one simple quantitative attribute measured in two environments. Can. J. Genet. Cytol. 15: 635–645.

Scheiner, S. M, and C. J. Goodnight. 1984. The comparison of phenotypic plasticity and genetic variation in populations of the grass *Danthonia spicata*. Evolution 38: 845–855.

Schlichting, C. D. 1986. The evolution of phenotypic plasticity in plants. Ann. Rev. Ecol. Syst. 17: 667–693.

Schlichting, C. D., and D. A. Levin. 1984. Phenotypic plasticity of annual phlox: Tests of some hypotheses. Am. J. Bot. 71: 252–260.

Shaw, R. G. 1987. Maximum-likelihood approaches applied to quantitative genetics of natural populations. Evolution 41: 812–826.

Via, S. 1984. The quantitative genetics of polyphagy in an insect herbivore. II. Genetic correlations in larval performance within and among host plants. Evolution 38: 896–905.

Via, S., and R. Lande. 1985. Genotype-environment interaction and the evolution of phenotypic plasticity. Evolution 39: 505–522.

Via, S., and R. Lande. 1987. Evolution of genetic variability in a spatially heterogeneous environment: effects of genotype-environment interaction. Genet. Res. 49: 147–156.

Weis, A. E., and W. L. Gorman. 1987. Effects of host-plant variation on the expression of genetic variance in the goldenrod gall fly. Bull. ecol. Soc. Am. 68: 443.

Westcott, B. 1986. Some methods of analyzing genotype-environment interaction. Heredity 56: 243–253.

Wilkinson, L. 1986. SYSTAT: The System for Statistics. SYSTAT, Inc., Evanston, IL.

Wright, S. 1931. Evolution in Mendelian populations. Genetics 16: 97–159.

Yamada, Y. 1962. Genotype by environment interaction and genetic correlation of the same trait under different environments. Jap. J. Genetics 37: 498–509.

Zar, J. H. 1984. Biostatistical Analysis, 2nd ed. Prentice-Hall, Inc., Englewood Cliffs, NJ.

Received 27 January 1988;
accepted 31 May 1988.
Corresponding Editor: G. Wagner