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The genetics of phenotypic plasticity. III. Genetic correlations and fluctuating asymmetries

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Abstract

We examined the relationship of three aspects of development, phenotypic plasticity, genetic correlations among traits, and developmental noise, for thorax length, wing length, and number of sternopleural bristles in *Drosophila melanogaster*. We used 14 lines which had previously been selected on either thorax length or plasticity of thorax length in response to temperature. A half-sib mating design was used and offspring were raised at 19° C or 25° C. We found that genetic correlations were stable across temperatures despite the large levels of plasticity of these traits. Plasticities were correlated among developmentally related traits, thorax and wing length, but not among unrelated traits, lengths and bristle counts. Amount of developmental noise, measured as fluctuating asymmetry and within-environmental variation, was positively correlated with amount of plasticity only for some traits, thorax length and bristle number, and only at one temperature, 25° C.

Introduction

The development of an organism entails the coordination of many events. At least some of those events must be linked to each other so that developmental shifts in one pathway can be compensated for by modifications in other pathways (Adams, 1967; Caswell, 1983). Phenotypic plasticity is the extent to which a

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developmental pathway is modified by external environmental conditions (Bradshaw, 1965). In this paper we address the issue: to what extent does the plasticity of one trait affect other aspects of development? We will explore three facets of development.

The first aspect of development which we address is the extent to which plasticity of one trait alters correlations of that trait with others. It has been proposed that genetic correlations among traits will not be stable across environments if the correlated traits are phenotypically plastic (Clark, 1987). This issue cuts to the heart of our ability to make evolutionary predictions. Natural environments are constantly changing from minute to minute, day to day, and year to year. If genetic correlations are highly dependent on environmental conditions then no experimental measure of genetic correlations will be of use in predicting future population changes. However, if we can discover genetic correlations which are stable in the face of environmental changes and, more important, if we can discover rules governing which correlations are likely to be stable, then we have hope in building predictive evolutionary models. We will address this issue by comparing genetic correlations of plastic traits across environments.

The second aspect of development which we address is the extent to which phenotypic plasticity of one trait is related to phenotypic plasticity of other traits. Is a response to an environmental perturbation systemic or does each part of the phenotype react separately? We will address this issue by measuring correlations of plasticities of genetically and phenotypically correlated traits.

The final aspect which we address is the extent to which phenotypic plasticity is related to a second type of developmental variation, developmental noise. Phenotypic plasticity entails environmentally mediated developmental change. Developmental noise entails changes in developmental pathways due to random, internal events. At bottom, such events may be due to the stochastic nature of molecular processes. Although presented as a dichotomy, we recognise that there is a fuzzy boundary between these two types of variation. At what point should a random, internal change in one part of an organism be considered an external change in another part? It may be impossible experimentally to separate random, internal changes from microenvironmental variation below the resolution of or beyond the control of the experimenter. Be that as it may, such a distinction is still useful. It might be that a genotype that is highly plasticity by necessity is also developmentally "noisy". However, there is no reason why such a linkage must occur. We will address this issue by measuring correlations of plasticity and developmental noise.

We examined plasticity, genetic correlations, and developmental noise of three morphological traits in a set of lines of *Drosophila melanogaster* that differed in their plasticities for one of the traits, thorax length. These lines were the result of selection on thorax size and plasticity of thorax size (Scheiner and Lyman, 1990; see below). They have differentiated with respect to plasticity of thorax size and thus are likely to show effects of the plasticity of one trait on other aspects of development.

We investigated two measures of developmental noise. Fluctuating asymmetry (FA), nondirectional deviations from bilateral symmetry, is a useful measure of

departure from some ideal developmental program (Waddington, 1957; Palmer and Strobeck, 1986). Its usefulness is limited, however, to traits which can be measured independently on the left and right sides of the body. We also measured a second aspect of developmental noise, within-environmental variation among genetically identical or closely related individuals. We assume that genetically identical or closely related individuals raised in a uniform environment experience approximately the same environment and receive approximately the same amount of resources. Variation among such individuals, once genetic effects are removed, will estimate developmental noise. We realize that the above assumption is critical; to the extent that it does not hold no pattern will be discernible. Thus, comparisons of amounts of plasticity and amounts of within-environmental variation are biased against finding a relationship. No such bias exists in the comparison of amounts of plasticity and amounts of FA.

Materials and methods

Selection history

The flies used in this analysis resulted from 16 generations of selection on thorax size and plasticity of thorax size. There were 7 selection regimes, increased and decreased plasticity, increased and decreased thorax size at 19° C, increased and decreased thorax size at 25° C, and random selection (control), with two replicates of each regime for a total of 14 lines. A family selection scheme was used. See Scheiner and Lyman (1990) for the details of the selection experiment.

After 16 generations of selection, stock cultures of each line were established in half-pint milk bottles. After mass mating for at least one generation a half-sib mating scheme was established within each line by collecting males and females as newly eclosed adults. One day after eclosion, each male was allowed to mate for 24 h with three females. The females were separated and allowed to oviposit for six days in vials, being given new 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.

Five traits were measured on emerged offspring, thorax length, left and right wing lengths, and number of sternopleural bristles on the top row of the left and right sides. Thorax length was measured on three etherized 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 number of sternopleural bristles was counted at the same time. Wing length was measured for one female from each vial, three flies per family per temperature, by removing the wings, mounting them on glass slides, and measuring them with the ocular filar. Fluctuating asymmetry of wing length and bristle number was measured as the absolute difference of the left and right sides of an individual. Palmer and Strobeck (1986) suggested that measures of FA using the square of the difference were better than those using the

absolute value of the difference. However, becuase our aim was to compare amounts of plasticity and FA, we used absolute differences so that the two types of traits would have the same units. Preliminary analyses indicated no significant differences in conclusions when the square of the differences were used. Swain (1987) argued that meristic characters, such as numbers of bristles, may give misleading results in analyses of FA because shifts in mean counts relative to underlying thresholds can result in large changes of asymmetry even if the amount of developmental noise remains constant. However, in the present case mean counts changed very little between temperatures and among lines (see below). Thus, our use of FA of bristle number to measure developmental noise is likely to be robust. Plasticity differs from the other traits in that it is not a trait of an individual, but a trait of a genotype. Plasticity was measured as the difference in full-sib family means for flies raised at 19° C and 25° C (Scheiner and Lyman, 1989).

Statistical analyses

For the analyses we tried to balance the data as much as possible. To do this we eliminated all dams that did not have at least three offspring measured for thorax length or bristle number in each temperature and all sires that did not have at least two dams. Final sample sizes were 574, 587, and 589 half-sib families measured for thorax length, wing length, and bristle number, respectively. There were 2.6, 2.7, and 2.7 dams per sire, and 7.5, 1.9, and 6.5 offspring per full-sib family per temperature, for a total of 12345, 3220, and 11244 flies measured for each trait, respectively, from the 19° C treatment and 12264, 3232, and 11126 flies measured for each trait, respectively, from the 25° C treatment.

All analyses were done with SAS (SAS Institute Inc., 1985). Variance and covariance components of thorax length, wing length, bristle number, and FAs were calculated using procedure NESTED. The model used was:

$$Z_{ijkl} = \mu + l_i + s_{ij} + d_{ijk} + e_{ijkl}$$

where Z_{ijkl} is the individual measure, μ is the overall mean, l_i is the line effect, s_{ij} is the sire effect nested within line, d_{ijk} is the dam effect nested within sire, and e_{ijkl} is the offspring effect nested within dam. Narrow-sense heritabilities averaged over lines were calculated as:

$$h^2 = 4\sigma_S^2/(\sigma_S^2 + \sigma_D^2 + \sigma_R^2)$$

where σ_S^2 is the sire (half-sib) variance component, σ_D^2 is the dam (full-sib) variance component, and σ_R^2 is the residual variance component. A similar model was used for the calculation of covariances and additive genetic correlations.

Since plasticity was measured as the difference in full-sib family means, correlations of plasticity and FA were calculated using the mean FA of each family. The following model was used:

$$Z_{ijkl} = \mu + l_i + s_{ij} + d_{ijk}$$

where d_{ijk} is the dam (full-sib) mean effect nested in sires. The heritability of plasticity was estimated as:

$$h^2 = \sigma^2_{DM}/\sigma^2_P$$

where σ^2_{DM} is the variance among sires of dam mean differences and correcting the total phenotypic variance, σ^2_P, as given by Scheiner and Lyman (1989, p. 98 equation 7).

Standard errors for heritabilities and genetic correlations were calculated as by Becker (1984). Statistical tests with correlations were done as by Zar (1984, p. 310 and p. 313) but substituting the calculated standard errors.

For each line, within-environment, non-genetic variance components were estimated using two models. If the among dam variance was greater than the among sire variance we used the model:

$$\sigma^2_W = \sigma^2_R - (3\sigma^2_D - \sigma^2_S).$$

If the among sire variance was greater than the among dam variance we used the model:

$$\sigma^2_W = \sigma^2_R - 2\sigma^2_S$$

(Becker, 1984, p. 56). Sequential Bonnferoni procedures were used throughout the analyses with a nominal α of 0.05 to correct the critical values for multiple tests (Rice, 1989).

Results

Means and heritabilities

In order to examine the relationship of plasticity and other aspects of development, we first determined amounts of plasticity, FA, and additive genetic variation. As expected, temperature had a significant effect on thorax length, wing length, and bristle number (Table 1). Flies were larger at lower temperatures resulting in plasticities for the traits of thorax length and wing length. The ratio of wing length to thorax length also changed across temperatures from 2.126 ± 0.046 at 19° C to 1.978 ± 0.044 at 25° C. Significant heritabilities were measured for all of these traits in both temperatures with the values for wing length being about twice those for thorax length (Table 2). As with thorax length, the heritabilities of plasticity of wing length were about one-third the heritabilities of the trait. There were no significant differences in lengths, plasticities or heritabilities for left and right wings. Thus, FA of wing length was small and did not differ significantly between temperatures. We found no additive genetic variation for FA of wing length.

For sternopleural bristle number, more bristles were found at higher temperature. Again, left and right sides did not differ significantly. Fluctuating asymmetries did differ significantly among temperatures ($t = 10.83$, $P < 0.0001$) with more variation being found at the higher temperature. Heritabilities for bristle traits were generally

Table 1. Means and standard deviations (SD) of traits for individuals raised at 19° C and 25° C. Plasticity was measured as the difference among full-sibs raised at the two temperatures.

	Temperature								
	19° C			25° C			Plasticity		
Trait	Mean	SD	n	Mean	SD	n	Mean	SD	n
Thorax length (mm)	1.077	0.027	12345	1.053	0.031	12264	0.0243	0.0186	1606
Left wing length (mm)	2.300	0.051	3220	2.095	0.055	3232	0.2062	0.0524	1670
Right wing length (mm)	2.300	0.051	3220	2.096	0.054	3232	0.2050	0.0525	1670
FA wing length (mm)	0.0120	0.0112	3200	0.0131	0.0128	3232	—	—	—
Left bristle number	3.022	0.164	11244	3.073	0.299	11126	0.0447	0.1522	1673
Right bristle number	3.022	0.165	11244	3.080	0.309	11126	0.0525	0.1582	1673
FA bristle number	0.0437	0.2031	11244	0.1050	0.3158	11126	—	—	—

low. In contrast to the length traits, the heritabilities of plasticity of bristle number were of a similar magnitude to the heritabilities of bristle number itself. The heritabilities did not differ significantly between left and right sides, however they did differ significantly between temperatures for left bristle number ($Z = 2.80$, $P < 0.005$). Heritabilities for FA of bristle number were marginally significant.

Phenotypic correlations

We measured phenotypic correlations to determine which traits were developmentally linked. As expected, thorax length and wing lengths were strongly phenotypically correlated and did not differ either among temperatures or among left and right sides (Table 3). The correlation between left and right wing lengths differed significantly from 1. FA of wing length was significantly correlated with only two traits, thorax length and left wing length, and only at 25° C (0.059 ± 0.018 and -0.095 ± 0.018, respectively). As there was little variation for FA of wing length, correlations with this trait are uninformative and not discussed further. In contrast

Table 2. Heritabilities (± SE) for individuals raised at 19° C and 25° C; standard errors were calculated as by Becker (1984, p. 60). Heritabilities of plasticity were based on full-sib family means; standard errors were calculated as described in Scheiner and Lyman (1989). Significance levels were determined by ANOVA.

	Temperature		
Trait	19° C	25° C	Plasticity
Thorax length	0.339*** ± 0.043	0.325*** ± 0.043	0.105*** ± 0.025
Left wing length	0.693*** ± 0.087	0.648*** ± 0.082	0.279*** ± 0.063
Right wing length	0.686*** ± 0.086	0.655*** ± 0.082	0.295*** ± 0.064
FA wing length	0.005 ± 0.056	−0.026 ± 0.054	—
Left bristle number	0.038* ± 0.018	0.122*** ± 0.024	0.055** ± 0.022
Right bristle number	0.069*** ± 0.018	0.084*** ± 0.023	0.052* ± 0.023
FA bristle number	0.039* ± 0.019	0.027+ ± 0.018	—

+P < 0.10; *P < 0.05; **P < 0.01; ***P < 0.001

to wing length, left and right bristle numbers were only moderately correlated. Individuals with more bristles tended to have a greater FA of bristle number. This result was expected as individuals tend to lose or gain one or two bristles only. Correlations between length measures and bristle numbers were generally weak but significantly different from 0, except for wing lengths and bristle numbers at 19° C. In general, phenotypic correlations were of greater magnitude at 25° C than 19° C (12 of 15, $P < 0.035$) although pairwise comparisons were significant for only 4 pairs of correlations: thorax length and left bristle number, thorax length and FA of bristle number, left and right bristle number, and left bristle number and FA of bristle number.

Genetic correlations

Comparisons of genetic correlations across temperatures addresses the issue of the extent to which plasticity alters correlations across environments. First we determined which traits were genetically correlated. Within temperatures, length measures were highly genetically correlated (Table 3). The genetic correlations of left and right wing lengths were not significantly different from 1. The correlations between wing lengths and thorax length were large (0.73 – 0.74) and did not differ significantly for left and right wings. Because there was no additive genetic variance for FA of wing length, genetic correlations with this trait were undefined. The

Table 3. Phenotypic (above diagonal) and additive genetic (below diagonal) correlations (± SE) for individuals raised at 19° C (top) and 25° C (bottom). Standard errors for phenotypic correlations were calculated as by Zar (1984, p. 310) and significance levels were determined from standard tables. Standard errors for genetic correlations were calculated as by Becker (1984, p. 124) and significance levels were determined as by Zar (1984, p. 310) but using the calculated standard errors. Significance levels for comparisons between correlations measured at different temperatures were determined as by Zar (1984, p. 313) but using the calculated standard errors for genetic correlations. Pairs which differed significantly are shown in boldface.

	Thorax length	Left wing length	Right wing length	Left bristle number	Right bristle number	FA bristle number
Thorax	—	0.820* ± 0.018	0.818* ± 0.018	**0.041* ± 0.009**	0.054* ± 0.009	**0.068* ± 0.009**
length	—	0.822* ± 0.018	0.835* ± 0.018	**0.082* ± 0.009**	0.076* ± 0.009	**0.107* ± 0.009**
Left						
wing	0.732* ± 0.057	—	**0.985* ± 0.018**	0.035 ± 0.018	0.032 ± 0.018	0.075* ± 0.018
length	0.728* ± 0.058	—	**0.975* ± 0.018**	0.089* ± 0.018	0.079* ± 0.018	0.102* ± 0.018
Right						
wing	0.733* ± 0.057	1.001* ± 0.003	—	0.039 ± 0.018	0.036 ± 0.018	0.074* ± 0.018
length	0.740* ± 0.056	1.001* ± 0.004	—	0.090* ± 0.018	0.082* ± 0.018	0.104* ± 0.018
Left						
bristle	−0.003 ± 0.173	−0.119 ± 0.141	−0.102 ± 0.140	—	**0.183* ± 0.009**	**0.527* ± 0.009**
number	−0.319 ± 0.113	−0.315 ± 0.123	−0.294 ± 0.122	—	**0.420* ± 0.009**	**0.408* ± 0.009**
Right						
bristle	0.154 ± 0.136	−0.412 ± 0.479	−0.470 ± 0.519	1.198* ± 0.328	—	0.489* ± 0.009
number	0.257 ± 0.132	−0.338 ± 0.165	−0.345 ± 0.164	1.026* ± 0.103	—	0.471* ± 0.009
FA						
bristle	0.115 ± 0.181	−0.247 ± 0.204	−0.221 ± 0.200	0.901* ± 0.193	1.250* ± 0.241	—
number	0.182 ± 0.192	−0.067 ± 0.389	−0.134 ± 0.411	1.365* ± 0.388	0.967* ± 0.262	—

* Significantly different from 0 based on a sequential Bonnferoni procedure.

Table 4. Full-sib family mean (above diagonal) and additive genetic (below diagonal) correlations (± SE). Standard errors for family mean correlations were calculated as by Zar (1984, p. 310) and significance levels were determined from standard tables. Standard errors for genetic correlations were calculated as by Becker (1984, p. 116) and significance levels were determined as by Zar (1984, p. 310) but using the calculated standard errors.

	Plasticity thorax length	Plasticity left wing length	Plasticity right wing length	Plasticity left bristle number	Plasticity right bristle number	FA bristle number–19° C	FA bristle number – 25° C
Plasticity thorax length	—	0.450* ± 0.025	0.457* ± 0.025	0.024 ± 0.025	0.058 ± 0.025	−0.033 ± 0.025	0.041 ± 0.025
Plasticity left wing length	0.889* ± 0.117	—	0.952* ± 0.024	−0.027 ± 0.025	−0.003 ± 0.025	0.022 ± 0.025	0.038 ± 0.024
Plasticity right wing length	0.886* ± 0.116	0.995* ± 0.011	—	−0.032 ± 0.025	−0.005 ± 0.025	0.010 ± 0.025	0.021 ± 0.024
Plasticity left bristle number	−0.299 ± 0.214	−0.260 ± 0.204	−0.189 ± 0.199	—	0.450* ± 0.024	−0.244* ± 0.024	0.407* ± 0.024
Plasticity right bristle number	0.037 ± 0.214	0.034 ± 0.209	0.013 ± 0.207	0.927* ± 0.227	—	−0.176* ± 0.024	0.482* ± 0.024
FA bristle number – 19° C	0.047 ± 0.500	0.286 ± 0.565	0.439 ± 0.658	0.268 ± 1.168	−0.135 ± 0.842	—	0.143* ± 0.024
FA bristle number – 25° C	0.026 ± 0.255	0.197 ± 0.253	0.168 ± 0.250	2.175 ± 1.661	1.060 ± 0.724	2.013 ± 4.127	—

* Significantly different from 0 based on a sequential Bonnferoni procedure.

correlations of left bristle number, right bristle number, and FA of bristle number did not differ significantly from 1. Only one correlation between a length measure and a bristle number differed significantly from 0, thorax length and left bristle number at 25° C. In part the lack of significant correlations reflects the low power of the test when heritabilities are low. However, at best we can conclude that the genetic correlations were small because the correlations among the bristle traits themselves were significant despite their low heritabilities.

Genetic correlations measured in the two temperatures did not differ significantly for any pair of traits. Again, to some extent the lack of difference reflects the low heritabilities of the bristle traits. No pattern in magnitudes of the correlations across temperatures was found (7 of 14 larger at 25° C), unlike the phenotypic correlations.

Correlations among plasticities

Correlations among plasticities indicate the extent to which response to an environmental perturbation is systemic. The pattern of correlations among plasticities of traits (Table 4) followed that of the correlations among the traits themselves. The plasticity of thorax length was moderately phenotypically correlated with the plasticity of wing length and strongly genetically correlated. The genetic correlation differed significantly from 1. Plasticities of left and right wings were highly correlated although both the phenotypic and genetic correlations differed significantly from 1. Plasticities of left and right bristle numbers were moderately phenotypically correlated and strongly genetically correlated although differing significantly from 1. None of the plasticities of lengths was significantly correlated with the plasticities of bristle number.

If plasticities are correlated we expect that selection causing differences among lines in the plasticity of thorax length would create differences in the plasticities of other traits. There were significant differences among lines for plasticity of left wing lenth ($F_{13,587} = 21.56$, $P < 0.0001$), right wing length ($F_{13,587} = 15.55$, $P < 0.0001$), left bristle number ($F_{13,589} = 6.44$, $P < 0.0001$), and right bristle number ($F_{13,589} = 10.83$, $P < 0.0001$). As predicted based on the genetic correlations, among line correlations of plasticities of thorax length and wing length differed significantly from 0 (left wing: $r = 0.479$, $P < 0.05$; right wing: $r = 0.583$, $P < 0.05$) while correlations with plasticities of bristle number were not significantly different (left side: $r = 0.210$, $P < 0.5$; right side: $r = 0.350$, $P < 0.5$). As expected, correlations between left and right sides differed significantly from 0 for both plasticities of wing length ($r = 0.979$, $P < 0.0001$) and plasticities of bristle number ($r = 0.947$, $P < 0.0001$).

Plasticity and developmental noise

Correlations of amounts of plasticity and amounts of developmental noise indicate the extent to which these two types of developmental variation are

independent. We found that plasticity and noise were correlated, but the results were dependent on the environment of development. Conclusions were identical for the two measures of developmental noise, fluctuating asymmetry and within-environmental variation.

First we consider developmental noise as measured by FA. The plasticity of bristle number was moderately, but significantly, phenotypically correlated with FA of bristle number (Table 4). As plasticity can only be measured as the mean of a set of full-sibs, these correlations were done with the mean FA of the full-sib family raised at each temperature. For bristle number the correlations differed significantly among temperatures (left side: $Z = 20.06$, $P < 0.0001$; right side: $Z = 20.96$, $P < 0.0001$) with sibships that were more plastic having less FA at 19° C and more FA at 25° C. The FAs themselves were only weakly, but significantly, correlated. Among line correlations differed significantly from 0 for the plasticity and FA of bristle number at 25° C (left side: $r = 0.851$, $P < 0.0001$; right side: $r = 0.852$, $P < 0.0001$) but not for the plasticity and FA of bristle number at 19° C (left side: $r = 0.366$, $P < 0.2$; right side: $r = 0.473$, $P < 0.1$). The among line correlation of FAs of bristle number differed significantly from 0 ($r = 0.734$, $P < 0.01$). None of the genetic correlations between plasticity and FA of bristle number differed significantly from 0 (Table 4). Again, this may have been due to the weak power of these tests due to the low heritabilities of the traits. Because there was no additive genetic variation for FA of wing length, correlations could not be calculated.

Second we consider developmental noise as measured by within-environmental variance. Among line correlations were estimated between the plasticity of each trait and the within-environmental variance at 19° C and 25° C. Of the 10 correlations (5 traits, 2 environments) only 3 were significant, plasticities and within-environmental variances at 25° C of thorax length, left bristle number, and right bristle number ($r = 0.699$, $r = 0.802$, and $r = 0.882$, respectively). Correlations were not significantly different from 0 for within-environmental variances of wing length at 25° C or for any within-environmental variances at 19° C. For bristle number, within-environmental variances were greater at 25° C than 19° C (left side: 0.069 vs 0.027, $t = 3.867$, $P < 0.0001$; right side: 0.071 vs 0.023, $t = 4.634$, $P < 0.0001$), but did not differ between left and right sides within temperatures.

Discussion

Stability of genetic correlations

We found that genetic correlations between traits of thorax length and wing length were unchanged across temperatures. This genetic correlation is also stable across populations. Several workers have measured this correlation in a variety of stocks from around the world over several decades (Reeve and Robertson, 1953; Tantawy, 1959; Tantawy et al., 1964; Tantawy and Rakha, 1964; Tantawy and El-helw, 1966; Tantawy and Tayel, 1970). Values ranged from 0.75 to 0.84 on unselected stocks and from 0.70 to 0.96 on lines which had undergone selection on

either wing length or thorax length. The mean correlation measured in this study, 0.73, falls within this range. Interestingly the range of heritabilities measured in the above and additional studies (Robertson, 1959, 1962; Cowley et al., 1986; see summary in Roff and Mousseau, 1987, Fig. 5) is much greater, 0.15 to 0.50 for thorax length and 0.08 to 0.67 for wing length. The values in the present study fall in the middle of the range for thorax length, 0.34 at 19° C and 0.33 at 25° C, but are at the extreme end for wing length, 0.69 at 19° C and 0.65 at 25° C. The greater variability of heritability measures is not unexpected. The estimation of heritability includes the residual variance, which is highly dependent on experimental conditions. On the other hand, the estimate of the genetic correlation contains only genetic variance and covariance components and would be less subject to experimental variability.

Is there a functional explanation for the stability of this genetic correlation? Thorax length and wing length may be functionally related in three ways. First, they may be coupled by pleiotropy. For example, a gene affecting cell size might change both wing size and thorax size. Second, they may be epigenetically coupled since the flight muscles run within and are attached to the thorax. Larger wings might require larger muscles which in turn would result in the development of a larger thorax. Third, they may be in linkage disequilibrium. Stabilizing selection on wing loading, the ratio of wing surface area to body weight, would produce a constant wing length to thorax length ratio. To maintain this ratio would require that alleles for a longer thorax be in disequilibrium with alleles for a longer wing. In two previous studies, one comparing 11 species of *Drosophila* (Starmer and Wolf, 1989) and one comparing strains of *D. melanogaster* collected from different regions (Robertson, 1959), a strong phenotypic correlation of thorax and wing length was found. We concur with Robertson (1959) and Starmer and Wolfe (1989) that there appears to be stabilizing selection on wing loading.

Functionally related traits appear to be the most likely candidates for stable genetic correlations. We conducted a study (Scheiner et al., 1989) of genetic correlations among life history traits in *D. melanogaster* and found that correlations among parts of the fecundity schedule were consistent across environments and negative between early and late fecundity, but correlations between fecundity and lengevity were not consistent. A comparison with previous studies found that these conclusions held across a large number of populations and types of experiments. We concluded that consistent genetic correlations are expected between functionally related traits, such as parts of the fecundity schedule, but not more distantly related traits. A number of workers have looked at the stability of genetic correlations across environments (Baker and Cockrem, 1970; Giesel et al., 1982; Murphy et al., 1983; Via, 1984; Service and Rose, 1985; Giesel, 1986; Groeters and Dingle, 1987; Lynch et al., 1988; Falkenhagen, 1989). All found at least some genetic correlations to differ across environments but no distinction was made between functionally related and unrelated traits.

Recently, concerns have emerged about the stability of genetic correlations among environments (Service and Rose, 1985). In particular, Clark (1987) stated that "the observation of different genetic covariance structures under different

laboratory conditions strongly indicates that estimates of genetic correlation observed in the lab have little bearing in the expression of the genetic variation in the field." He suggested that genetic correlations should be done under field conditions. We propose, however, that the problem needs to be turned on its head; we need to discover those correlations that are stable under a wide range of conditions. In this experiment we found no effect of rearing temperature on genetic correlations of morphological traits. This result is especially noteworthy because these traits exhibited substantial amounts of genotype-environment interaction variance, precisely the conditions which would alter correlations according to Clark. We reiterate our previous conclusion that functionally related traits may be those most likely to show stability of genetic correlations across environments.

Correlations of plasticities

We found that the plasticity of one trait was related to the plasticity of another trait only if the two traits were otherwise genetically and phenotypically correlated. The plasticity of thorax length was correlated with the plasticity of wing length but neither were correlated with the plascticity of bristle number. The plasticities of left and right side bristle numbers were correlated, indicating that the lack of correlation with the length traits was likely due to a general developmental independence of the two types of traits. Even though the bristles, the top row of sternopleurals, were on the thorax, their development was apparently independent of other parts of the thorax. We conclude that the amount of plasticity is not a general property of a genotype but is specific to a trait or trait complex. Similar conclusions were reached in studies of eye size and wing-vein length in *D. melanogaster* (Thompson and Rook, 1988) and various growth and morphological traits in *Danthonia spicata* (Scheiner and Goodnight, 1984), species of *Phlox* (Schlichting, 1986), and *Nicotiana rustica* (Perkins and Jinks, 1973).

Plasticity and developmental noise

There was no general relationship between the plasticity expressed by a genotype and the within-environment developmental stability expressed by that genotype. However, some regularities were found. Significant correlations were found only for flies raised at 25° C. Most of the significant effects involved sternopleural bristle number. Wing length was not developmentally "noisy". It might be expected that there is stronger selection for flies to have equal sized wings than to have equal number of bristles on the left and right sides. Plasticity of thorax length and the amount of within-environmental variation were significantly correlated among lines, in contrast to wing length. Thus, despite the strong phenotypic and genetic correlation between thorax length and wing length the two traits have different developmental patterns.

Plasticity and developmental noise have been found to vary independently in most previous studies. Selection on plasticity in *D. melanogaster* did not result in

changes in FA of scutellar bristle number (Kindred, 1965), wing-vein length (Scharloo et al., 1972), or eye size (Waddington, 1960). Santiago et al. (1989) found no correlation between plasticity and within-environment variation for fecundity in *D. melanogaster*. Bagchi and Tyama (1983) increased within-environmental variability in *Arabidopsis thaliana* by exposure to radiation but differences among lines were independent of the amount of phenotypic plasticity. In *Nicotiana rustica* amount of within-environmental variation was positively correlated with amount of phenotypic plasticity for flowering time but not for the traits of growth rate, leaf length, or final height (Perkins and Jinks, 1973). However, in one study the within-family coefficient of variation increased following selection on plasticity of eye size (Waddington and Robertson, 1966). Additionally, Garcia-Vazquez and Rubio (1988) selected for increased FA in scutellar bristle number and found that the plasticity of bristle number in response to temperature also increased.

Genetic basis of plasticity

We previously (Scheiner and Lyman, 1990) presented three genetic models of plasticity: (1) plasticity is a function of homozygosity, the overdominance model (Lerner, 1954; Gillespie and Turelli, 1989), (2) plasticity is a function of differential expression of the same gene in different environments, the pleiotropy model (Falconer, 1981; Via and Lande, 1985, 1987; Via, 1987), or (3) plasticity is due to genes that determine the magnitude of response to environmental effects which interact with genes that determine the average expression of the character, the epistasis model (Lynch and Gabriel, 1987; Jinks and Pooni, 1988; Scheiner and Lyman, 1989). Based on the direct response to selection on the plasticity of a character, correlated responses to selection on the character itself, changes in genetic variance during selection, and changes in the cross-environment genetic covariance, we concluded that plasticity was most likely due to epistasis. These models make two additional predictions with regard to correlations among traits and developmental noise. (1) Plasticities of other traits: the overdominance model predicts that the plasticity of all traits will be positively genetically correlated because plasticity is a function of the number of heterozygous loci; the pleiotropy model predicts that plasticities will be genetically correlated to the extent that the traits are correlated because the same loci are responsible for both the mean of the trait and plasticity; the epistasis model predicts that plasticities will be genetically correlated to the extent that there is a common developmental basis for plasticity because the plasticity loci are independent of the loci controlling the mean of the trait. (2) Developmental noise: the overdominance model predicts a positive correlation between plasticity and developmental noise because both are a function of the number of heterozygous loci; the pleiotropy model assumes no correlation because the distribution of allele effects in the two environments are independent; the epistasis model makes no prediction.

As in the previous paper, the strongest negative evidence comes with regard to the overdominance model. Plasticities of some traits were correlated but not others.

Developmental noise and plasticity were correlated only for some traits and only in some environments.

Again, the evidence differentiating the pleiotropy model and the epistasis model is more equivocal. As predicted by the pleiotropy model, since thorax length and wing length were strongly genetically correlated, their plasticities were also strongly correlated. However, the correlation might also be predicted on epigenetic grounds based on the developmental relation between thorax length and wing length in accord with the epistasis model. The genetic correlations of the plasticities of thorax length and wing length were significantly larger than the genetic correlations of the traits themselves ($Z \geqslant 3.52$, $P \leqslant 0.0002$, Tables 3 and 4). Unfortunately, there is no statistical theory to predict how much of the genetic correlation among plasticities is a simple mathematical artifact of the correlation of the traits. The significantly larger correlation of the plasticities suggests to us that additional genetic factors, ie. epistatic interactions between genes determining the mean of a trait and genes determining trait plasticity, may be involved.

The correlation of developmental noise and plasticity in some traits violates the assumptions of the pleiotropy model. The underlying mathematical model upon which the pleiotropy model is built explicitly assumes that the within-environmental variation of a character is independent of the mean value of the character (Via and Lande, 1987). We found that lines which were more plastic were also more variable at 25° C for both thorax length and bristle number. For bristle number there was also a phenotypic, although not genetic, correlation between plasticity and FA. Three studies have also found correlations between trait means and amounts of FA (Bradley, 1980; Soule and Cuzin-Roudy, 1982; Leary et al., 1985) Two studies failed to find a correlation (Mather, 1953; Thoday, 1958) and one study failed to find a correlation between the trait mean and amount of within-environmental variation (Bagchi and Iyama, 1983). This diversity of results cannot be incorporated into the pleiotropy model.

Although the epistasis model does not make any prediction with regard to the relationship of plasticity and developmental noise, thus the data presented here neither support nor refute it, this model can incorporate a variety of relationships. Lynch and Gabriel (1987) differentiate between random variation among genotypes and genetic variation for plasticity (e_1 and g_2 in their notation). Although they assume that the two are uncorrelated, more complex models assuming a correlation can be developed. In particular, different traits can be treated differently including positive, negative, and zero correlations. Similarly, Scheinberg (1973) illustrated how two plasticities could coevolve, given a genetic correlation, independent of selection on trait means.

The results presented here and in our companion paper (Scheiner and Lyman, 1990) lend support to the notion that there are genes which control developmental variation separate from those which determine the mean value of a particular character. Clearly, the system is complex however, as each trait (thorax length, wing length, and bristle number) showed a different relationship among overall amounts of variation, amounts of plasticity, and amounts of developmental

noise, and all were temperature dependent. Evolutionary models are needed which can encompass the entirety of this diversity.

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