Theor Appl Genet (1994) 88:604–613

D. Hébert · S. Fauré · I. Olivieri

Genetic, phenotypic, and environmental correlations in black medic, *Medicago lupulina* L., grown in three different environments

Received: 25 February 1993 / Accepted: 9 September 1993

Abstract We have investigated the relationship between phenotypic and genetic correlations among a large number of quantitative traits (36) in three different environments in order to determine their degree of disparity and whether phenotypic correlations could be substituted for their genetic counterparts whatever the environment. We also studied the influence of the environment on genetic and phenotypic correlations. Twenty accessions (full-sib families) of *Medicago lupulina* were grown in three environments. In two of these two levels of environmental stress were generated by harvesting plants at flowering and by growing plants in competition with barley, respectively. A third environment, with no treatment, was used as a control with no stress. Average values of pod and shoot weight indicate that competition induces the highest level of stress. The genetic and phenotypic correlations among the 36 traits were compared. Significant phenotypic correlations were obtained easily, while there was no genetic variation for 1 or the 2 characters being correlated. The large positive correlation between the genetic and phenotypic correlation matrices indicated a good proportionality between genetic and phenotypic correlations matrices but not their similarity. In a given environment, when only those traits with a significant genetic variance were taken into account, there were still differences between genetic and phenotypic correlations, even when levels of significance for phenotypic correlations were lowered. Consequently, it is dangerous to substitute phenotypic correlations for genetic correlations. The number of traits that showed genetic variability increased with increasing environmental stress, consequently the number of significant genetic correlations also increased with increasing environmental stress. In contrast, the number of significant phenotypic correlations was not influnced by the environment. The structures of both phenotypic and genetic matrices, however, depended on the environment, and not in the same way for both matrices.

Key words Genetic correlations · Phenotypic correlation · Environmental correlations · Stress · *Medicago lupulina* L.

Communicated by P. M. A. Tigerstedt

D. Hébert · S. Fauré · I. Olivieri (✉)
Institut National de la Recherche Agronomique, Station de Génétique et d'Amélioration des Plantes de Montpellier, Domaine de Melgueil, F-34130 Mauguio, France

Introduction

Genetic correlations arise from pleiotropy, or linkage disequilibrium, or both (Falconer 1981). As they determine how traits will change in relation to each other, an understanding of genetic correlations among life history traits is crucial for an understanding of coordinate evolution through correlated responses to natural selection (Falconer 1981). In the context of plant breeding, a certain comprehension of genetic correlations is particularly useful for performing indirect artificial selection on characters that show low heritability and/or are difficult to measure (Gallais 1990). In addition, the need to change the mean of one particular trait in a population, while inducing little or no change in other traits makes it necessary to obtain some knowledge of the genetic correlations among traits in order to avoid undesirable correlated responses (Gallais et al. 1983; Huang et al. 1990).

Some authors have studied genetic correlations among traits; however, such determinations have usually been limited to a few characters and mostly only to life history traits (Service and Rose 1985; Shaw 1986; Groeters and Dingle 1987; Gebhart and Stearns 1988; Newman 1988; Scheiner et al. 1991). Furthermore, in most of these experiments genetic correlations have been examined in only one environment (Cheverud 1982; Mitchell-Olds 1986; Kohn and Atchley 1988; Harding et al. 1990). Consequently, despite those studies on genetic correlations, little is known about the influence of environment on these correlations. Several authors, however, have suggested that the genetic correlations among characters may be altered by the envi-

ronment (Cuartero and Cubero 1982; Giesel et al. 1982; Via 1984; Service and Rose 1985; Giesel 1986; Shaw 1986; Groeters and Dingle 1987; Van Noordwijk and Gebhardt 1987; Clarker and Keith 1988; Gebhardt and Stearns 1988; Newman 1988; Falkenhagen 1989), but there have been no detailed studies of the nature or extent of such environmental effects.

The estimation of genetic correlations is often difficult because it requires a large sample of genotypes or related individuals. On the other hand, phenotypic correlations are more easily calculated because they require moderate sampe sizes and no individual filiation. Moreover, they show smaller standard errors than the genetic correlations. Because genetic correlations are difficult or impossible to measure in many cases or species, it is important to determine whether genetic and phenotypic correlations are similar in order to be able to substitute the phenotypic correlations for genetic ones.

Cheverud (1988) studied the relationship between genetic and phenotypic correlations of 41 experimental studies. He found that "much of the dissimilarity between phenotypic and genetic correlation estimates seems to be due to imprecise estimates of genetic correlations." Consequently, he concluded that "when reliable genetic estimates (correlations) are unavailable, phenotypic correlations ... may be substituted for their genetic counterparts in evolutionary models of phenotypic evolution." Willis and Coyne (1990) reexamined the data that Cheverud used and found no support for Cheverud's assertion that phenotypic correlations reflect genetic correlations.

In the study described here we investigated the relationship between phenotypic and genetic correlations in a large number of traits (36) in three different stress-inducing environments in order to determine their degree of disparity and whether phenotypic correlations can be substituted for their genotypic counterparts, whatever the environment. To reach this end, the influence of environment on genetic and phenotypic correlations was also studied.

Materials and methods

Plant material

The material consisted of 20 full-sib families of *Medicago lupulina* L. (Leguminosae), a partially domesticated species used for fodder and grazing. As the species is autogamous (Lesins and Lesins 1979), a full-sib family was considered to be a single genotype. These genotypes were considered to be a representative sample of the species since they differed in domestication (4 cultivated genotypes/16 wild genotypes), in geographic origin (California, Denmark, Spain and different provinces of France), in degree of disturbance of collection site (from pasture land to follow land) and in life-cycle (7 annuals, 13 perennials).

Experimental design

The experiment was located at Montpellier in the south of France. Plants were established in pots, in a greenhouse, during the fall of 1988 and then transplanted to the field in February 1989. Three different environments that induced various levels of stress were created by two treatments: (1) control, with no treatment; (2) harvesting environment, in which plants were harvested at flowering; (3) competition environment, in which each medic plant was surrounded by barley plants sown just after the black medics had been transplanted to the field.

The experimental design was a split plot with environments as whole plots and genotypes as subplots, and two replicates ('blocks') of the design. Genotypes were randomized within each plot. Plants were arranged in rows(= subplots) with a distance of 1.5 m between rows and 1 m between plants within each row. Each subplot consisted of 5 plants of the same genotype. Thus, within a plot, there was covariation between genotype and micro-environment so that it was useless to consider individual data. Instead, for each character we used only the mean value per subplot.

Observations were taken each week during the first year (from February 1989 to July 1989) on 22 characters from which 36 traits were derived (Table 1). These measurements described phenology, vegetative growth, flowering and pod set rate, vegetative yield, inflorescence and pod production, reproductive effort, seed quality and percentage of adult survival after reproduction.

In our discussion the three environments are considered as stress (competition), intermediate stress (harvesting) and nonstress (control) environments because, relative to the control, average pod and shoot weight, the most important agronomic traits of black medic, indicated that competition created a high level of stress, while the harvesting treatment induced a moderate level of stress (Hébert et al. submitted).

Statistical analysis

According to Scheffé (1959) and Dagnélie (1986), the significance level and test power of the analysis of variance (ANOVA) are not very sensitive to non-normality, and the equality of error variances is a secondary hypothesis when sample sizes are equal, as was the case in this experiment. ANOVA (SAS Institute 1985) were thus performd on all 36 characters to test the significance of the genotype effect in each environment.

For each environment the initial ANOVA model was as follows:

$$Y_{ijk} = \mu_j + G_{ij} + b_{jk} + \epsilon_{ijk} \quad (1)$$

where for a given environment j, Y_{ijk} is the phenotype of genotype i in block k, μ_j is the general mean, G_{ij}, $1 \leq i \leq g = 20$ is the random genotype factor, b_{jk}, $1 \leq k \leq b = 2$ is the fixed block factor and ϵ_{ijk} is the residual error.

As in each environment the block effect was not significant for two-thirds of the traits, we pooled this factor into the error. The final model was then:

$$Y_{ijk} = \mu_j + G_{ij} + \epsilon_{ijk} \quad \text{(Table 2)} \quad (2)$$

In each environment, environmental correlations among traits which showed genetic variability were calculated and phenotypic correlations between all 36 characters were estimated (proc MANOVA option PRINTE and proc CORR, respectively, SAS institute 1985). The environmental correlations come from micro-environmental variations in a macro-environment (control, harvesting or competition environment) and represent residual correlations. The significance of phenotypic and environmental correlations is tested by a Student test. The critical value of the phenotypic correlations, which was based on 38 degrees of freedom, was ± 0.31 at $P < 0.05$, ± 0.40 at $P < 0.01$ and ± 0.50 at $P < 0.001$. The critical value of the environmental correlations, which was based on 18 degrees of freedom, was ± 0.43 at $P < 0.05$, ± 0.54 at $P < 0.01$ and ± 0.66 at $P < 0.001$.

In each environment, genetic correlations among those characters which showed significant genetic variability (for description of these traits, see Hébert et al. submitted) were estimated as follows. From the matrix of the sums of products and the sums of squares of the model of variance analysis (Eq. 2), mean products (MP) and mean squares (MS) of the genetic and error factors were calculated. The matrices of

Table 1 Description of the 36 traits used in this study

Trait	Description of trait
Phenological traits	
1	Days to flowering (d[a])
2	Days to end of flowering (d)
3	Days to maximum flowering (d)
4	Days to maximum pod set (d)
5	Days to maximum plant size[b] (d)
6	Days to first pod formation (d)
7	Days to first ripe pod (d)
8	Days when all pods are ripe (d)
9	Flowering duration (d)
10	Interval between flowering and pod setting (d)
11	Pod set duration (d)
Growth	
12	Rate of growth on the major axis[c] ($cm\,d^{-1}$)
13	Rate of growth on the minor axis ($cm\,d^{-1}$)
14	Flowering rate ($n^{d}\,d^{-1}$)
15	Pod set rate ($n\,d^{-1}$)
Vegetative yield	
16	Maximum plant size (cm)
17	Size at flowering (cm)
18	Final vegetative dry matter weight (g)
19	Final vegetative dry matter weight/maximum plant size ($g\,cm^{-1}$)
Inflorescence and pod production	
20	Inflorescence number (n)
21	Average inflorescence number per branch at the begining of flowering (n)
22	Average maximum inflorescence number per branch (n)
23	Average pod number per branch at the begining of pod set (n)
24	Average maximum pod number per branch (n)
25	Total pod number (n)
26	Total pod weight (g)
27	Total pod and shoot weight (g)
28	Viable pod weight (g)
Reproductive effort	
29	Total pod weight/final vegetative dry matter weight
30	Total pod weight/total pod and shoot weight
Seed quality	
31	% of tegumentary dormancy
32	% of embryonic dormancy
33	% of shrivelled seeds
34	% of seed malformation
35	% of viable seeds
36	% of adult survival

[a] d, Number of days from the date of transplantation to the field
[b] Plants had a creeping habit and an elliptical shape. The plant size that was measured was the major axis of the ellipse
[c] As the plant shape was an ellipse, it was described by the major and minor axeses
[d] n, Number

genetic covariances and variances and error covariances and variances were then estimated using the MS or MP expectation formula described in Table 2. Finally, genetic correlations were calculated with the formula:

$$r_G(1,2) = \mathrm{cov}_G(1,2)/[\sigma^2_G(1)\cdot\sigma^2_G(2)]^{1/2},$$

where $\mathrm{cov}_G(1,2)$, $\sigma^2_G(1)$ and $\sigma^2_G(2)$ represent the genetic covariance between, and genetic variances of, traits 1 and 2, respectively.

Through many authors (Reeve 1955; Tallis 1959; Mode and Robinson 1959; Scheinberg 1966; Becker 1984) have studied genetic correlations, there is no direct test of the significance of genetic correlations. Indeed, the problem of significance of genetic correlations is more complex than that of phenotypic or environmental correlations because the genetic correlation coefficients are derived from variance and covariance analyses and are therefore not directly estimated. The statistical significance of estimated genetic correlations was assessed by constructing a test which was based on the approximate standard error of the genetic correlation estimated using the formula of Scheinberg (1966). We applied the modification suggested by Becker (1984), which consists in adding 2 to each degree of freedom. The inferior limit of the confidence interval of each genetic correlation was calculated as:

$$r_{G\min} = |r_G| - t_{(0,975,18)}\cdot[\mathrm{var}(r_G)]^{1/2}$$

where t is the Student test value, and $\mathrm{var}(r_G)$ is the variance of the genetic correlation coefficient. This value was compared to the critical absolute values of the correlation coefficient that were significant at a confidence level of 95%, 99% and 99.9%. If the inferior limit was greater than these critical values then the genetic correlation was considered to be significantly different from zero for the corresponding confidence level. The critical value of the correlation coefficient is as follows:

$$r_c = t_c[(n-2)+t_c^2]$$

where t_c is the Student test value for $n-2$ degrees of freedom and n is the number of genotypes. The significance of the genetic correlations depends on the standard error that varies from estimate to estimate. In most cases, the critical value of the genetic correlations was above ± 0.68.

The overall magnitude of correlation within each matrix measured by the average of the squared correlation values (Cheverud 1982). In order to calculate the average of squared correlation values, each individual off-diagonal correlation value was squared, and the squared correlation values were summed and then divided by the number of off-diagonal elements in the matrix. The overall level of correlation within the genetic, phenotypic and environmental correlation matrices was measured using the index of integration (Cheverud et al. 1983). The index is defined as:

$$I = \sum_{i=1}^{p} t_i - 1/[p(p-1)]$$

where t_i is the ith eigenvalue and p is the number of traits. The index will generally take values between 0 and 1, where 0 implies no integration and 1 is perfect integration. Unfortunately, no significance test for the index of integration has yet been developed. Comparison of "patterns" of correlation matrices was made using a Pearson product-moment correlation coefficient (Cheverud 1988). This comparison does not test the similarity between two correlation matrices but the proportionality. Large positive correlations indicate merely that the correlations of the two matrices vary in similar directions, not that the values of the correlations are identical. The average disparity between two correlation matrices was calculated as the mean of the absolute values of the differences between a pair of correlations (Willis and Coyne 1991). Finally, the equality of two correlation matrices was tested using the chi2 test of Jenrich (1970).

To provide a more simple and clear representation of the correlations among characters, correlation networks instead of correlation matrices are presented (Schilchting 1989).

Table 2 Analysis of variance of the genetic effect in each environment (The genetic and residual covariances are obtained by replacing the mean squares (MS) with the mean products (MP))

Source	df	Sum of squares	Mean squares	E(MS)	F
Phenotype	39	SS_P[a]	$MS_p = SS_p/39$		
Genotype	19	SS_G	$MS_G = SS_G/19$	$\sigma^2_r + 2\sigma^2_G$	MS_G/MS_r
Error	20	SS_r	$MS_r = SS_r/20$	σ^2_r	

[a] $SS_P = SS_G + SS_r$

Results and discussion

Genetic correlations

Genetic correlations among certain traits were significantly positive in all three environments, except in the absence of the genetic variability of some of these traits in one environment: genetic correlations (1) among most of the phenological traits (1, 3, 4, 6, and 7), (2) among traits involving vegetative weight (18, 19, and 27), (3) among traits which described inflorescence and pod production (20, 21, 22, 24, 25), (4) between the total pod weight and viable pod weight (26 and 28) and (5) between the two variables which described reproductive effort (29 and 30). The genetic correlation between the percentages of tegumentary and embryonic dormancy (31 and 32) was significantly negative in all environments. Traits which showed highly positive correlations between themselves, whatever the environment, were regrouped in the correlation networks (Fig. 1A–C).

In the control, 45 genetic correlations were calculable, with only 13 being significantly different from zero (Table 3). Estimates of genetic correlations ranged from -1.15 to $+1.05$, with a mean absolute value for all correlations of 0.45. Standard errors varied from near zero to 0.29. Among the significant genetic correlations 5 were positive and 8 were negative (Table 3). Most of the significant genetic correlations which occurred in the control existed in the competition environment but not in the harvesting environment (Fig. 1A–C, Table 4).

In the harvesting environment, 253 genetic correlations were calculable and only 49 were significantly different from zero (Table 3). Estimates of genetic correlations ranged from -1.07 to $+1.04$, which a mean absolute value for all correlations of 0.46. Standard errors varied from near zero to 0.42, but most were between 0.2 and 0.3. Among the significant genetic correlations, 30 were positive and 19 were negative (Table 3).

In the competition environment 406 genetic correlations were calculable, and 85 were significantly different from zero (Table 3). Estimates of genetic correlations ranged from -1.21 to $+1.18$, with a mean absolute value for all correlations of 0.48. Standard errors varied from near zero to 0.55, but most were between 0.2 and 0.3. Among the significant genetic correlations, 50 were positive and 35 were negative (Table 3). Although in the competition and harvesting environments there was significant genetic variability for 22 common traits (Hébert et al. submitted), only 32 significant correlations were identical in the two environments (Table 4).

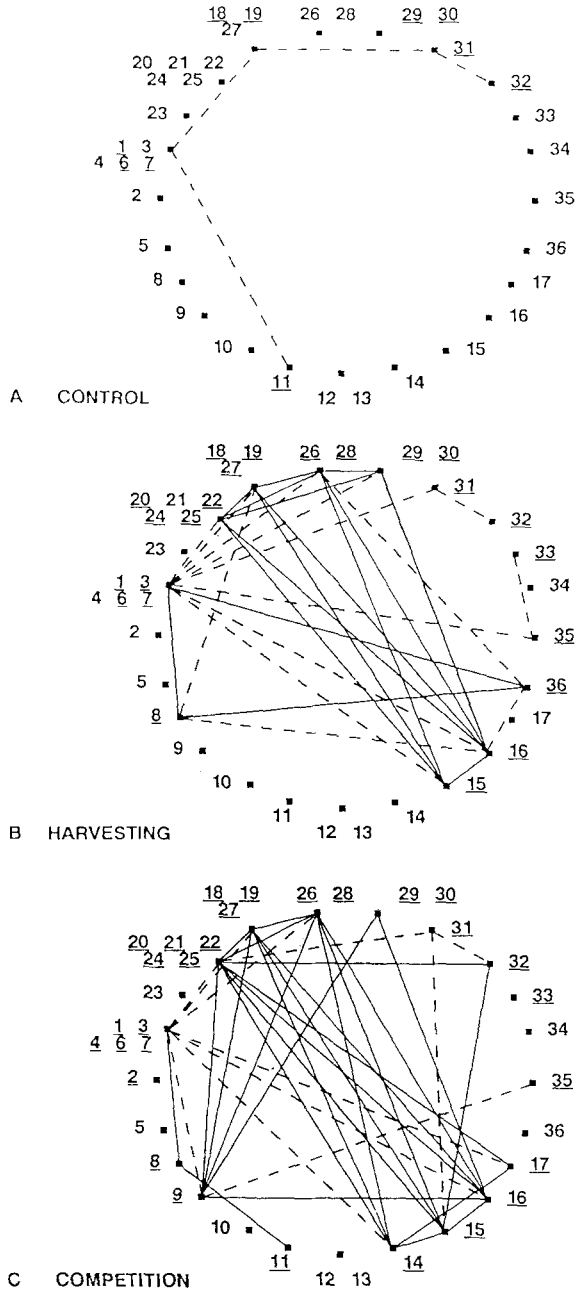


Fig. 1A–C Genetic correlations in control (**A**), harvesting (**B**) and competition (**C**) environments. The correlations among traits which were significantly different from zero at $P < 0.05$, $P < 0.01$ and $P < 0.001$ are represented by *lines* connecting the traits. *Solid lines* indicate positive correlations; *dashed lines*, negative correlations. The traits which are *underlined* show significant genetic variability. The genetic, phenotypic, and environmental correlation matrices and their standard errors or their significance levels are available from the authors upon request

Effect of environment on genetic correlations

The number of potential genetic correlations were the highest in the competition environment, the lowest in the control, and intermediate in the harvesting environment (chi2 test $P < 0.001$, Table 3). Indeed, the genetic correlations could only be estimated for traits that showed genetic variability. The stress induced by the competition and harvesting treatments reduced micro-environmental differences among individuals even more than they reduced differences among genotypes (Hébert

Table 3 Number of phenotypic (P), genotypic (G) and environmental (E) correlations that were potential, significant, significant at $P < 0.05$, $P < 0.01$ and $P < 0.001$ and significant and negative in the control, harvesting and competition environments (We have detailed the number of significant genetic, phenotypic, and environmental correlations at $P < 0.05$, $P < 0.01$ and $P < 0.001$ in this table to show that even when 31 phenotypic correlations among 630, 2 genetic correlations among 45 in the control, 12 genetic correlations among 253 in the harvesting environment and 20 genetic correlations among 406 in the competition environment are expected to be significant by chance alone, many other significant correlations remain)

Matrix	Correlation type	Control	Harvesting	Competition	Chi2
Among the 36 traits					
P	Potential	630	630	630	–
	Significant	260	244	234	2.29^{NS}
	Significant at $P < 0.05$	54	56	55	0.04^{NS}
	Significant at $P < 0.01$	66	83	69	2.56^{NS}
	Significant at $P < 0.001$	140	105	110	7.46*
	Significant and negative	118	107	100	0.70^{NS}
Among only those traits which showed genetic variability in each respective environment					
G	Potential	45	253	406	445.9***
	Significant	13	49	85	53.7***
	Significant at $P < 0.05$	0	16	30	–
	Significant at $P < 0.01$	0	11	15	13.5**
	Significant at $P < 0.001$	13	22	40	–
	Significant and negative	8	19	35	2.2^{NS}
P′	Significant	16	167	207	
	Significant at $P < 0.05$	3	29	44	
	Significant at $P < 0.01$	0	59	60	
	Significant at $P < 0.001$	13	79	103	
	Significant and negative	10	79	83	
E	Significant	14	105	115	
	Significant at $P < 0.05$	5	38	52	
	Significant at $P < 0.01$	2	33	29	
	Significant at $P < 0.001$	7	34	34	
	Significant and negative	9	37	28	

NS, Observed chi2 < theoretical chi2 at $P > 0.05$; *, observed chi2 < theoretical chi2 at $P < 0.05$; **, observed chi2 > theoretical chi2 at $P < 0.01$; ***, observed chi2 > theoretical chi2 at $P < 0.001$

et al. submitted). Consequently, the number of traits having significant genetic variability in the competition and harvesting environments (29 and 22, respectively) was higher than that found in the control (10). Since the number of traits which showed genetic variability increased with increasing environmental stress (Hébert et al. submitted), the number of potential genetic correlations also increased with environmental stress.

The competition environment had the highest number of significant genetic correlations; the control, the lowest, and the harvesting environment, the intermediate (chi2 test $P < 0.001$, Table 3). In the control the percentage of significant genetic correlations among the potential genetic correlations was higher than that found in the two stress environments, but it was not sufficiently higher to compensate for the low number of potential genetic correlations. Consequently, we found that the number of significant genetic correlations increased with increasing environmental stress. This results is in contrast to results obtained from previous studies of Service and Rose (1985), Cuartero and Cubero (1982), Shaw (1986) and Groeters and Dingle (1987), who all showed that the magnitude of genetic correlations decreased with increasing environmental stress, and to the results of Scheiner et al. (1991), who found that the environment had no effect on genetic correlations. However, our result is in agreement with the conclusion of Via (1984), Clarker and Keith (1988) and Falkenhagen (1989) that genetic correlations change with the environment.

In the control the genetic correlations which were significantly different from zero were all significant at $P < 0.001$. Correlations were either close to ± 1 or to 0, but never intermediate (Table 3). The traits which showed genetic variability in the control were either highly correlated or independent. This result is consistent with our observation that the genetic variances were either high or non-existent in the control (Hébert et al. submitted).

The nature of the environmental stress influenced the type of characters that were most often involved in significant genetic correlations. In the harvesting environment in which the stress induced a delay in the development of the reproductive system (Hébert et al. submitted), most of the significant genetic correlations were for phenological traits (1, 3, 4, 6 and 7), while in the competition environment, where the stress decreased reproductive effort and increased plant survival (Hébert

Table 4 Number of common and different correlations for each couple of matrices (*NS* Correlations not significantly different from zero at $P < 0.05$ in both matrices 1 and 2, *S* correlations significantly different from zero at $P < 0.05$, $P < 0.01$ and $P < 0.001$ in both matrices 1 and 2, *S1* correlations significantly different from zero at $P < 0.05$, $P < 0.01$ and $P < 0.001$ in matrix 1 only, *S2* correlations significantly different from zero at $P < 0.05$, $P < 0.01$ and $P < 0.001$ in matrix 2 only, *NS'* Correlations not significantly different from zero at $P < 0.001$, in matrix 1 and at $P < 0.05$, $P < 0.01$ and $P < 0.001$ in matrix 2, *S'* correlations significantly different from zero at $P < 0.001$ in matrix 1, at $P < 0.05$, $P < 0.01$ and $P < 0.001$ in matrix 2, *S1'* correlations significantly different from zero at $P < 0.001$ in matrix 1 only, *S2'* correlations significantly different from zero at $P < 0.05$, $P < 0.01$ and $P < 0.001$ in matrix 2 only)

Matrix 1 / Matrix 2	Total	Number of correlations			
		Common		Different	
		NS	S	S1	S2
P^a_{Cl}/P_H	630	267	143	119	101
P_{Cl}/P_C	630	304	170	92	64
P_{Cn}/P_H	630	300	148	86	96
G_{Cl}/G_H	36	26	6	4	0
G_{Cl}/G_{Cn}	45	32	10	3	0
G_{Cn}/G_H	231	165	32	21	13
P'_{Cl}/G_{Cl}	45	29	13	3	0
P'_H/G_H	253	85	48	120	0
P'_{Cn}/G_{Cn}	406	191	77	130	8
		NS'	S'	S1'	S2'
P'_{Cl}/G_{Cl}	45	32	13	0	0
P'_H/G_H	253	168	43	6	36
P'_{Cn}/G_{Cn}	406	406	56	29	47

[a] Cl, Control; H harvesting environment Cn competition environment

et al. submitted), the majority of the significant genetic correlations were those associated with inflorescence and pod production (20, 21, 22, 24 and 25) (Fig. 1A–B). In the control, the number of significant genetic correlations was too low to be able to determine which type of genetic correlation was the most common.

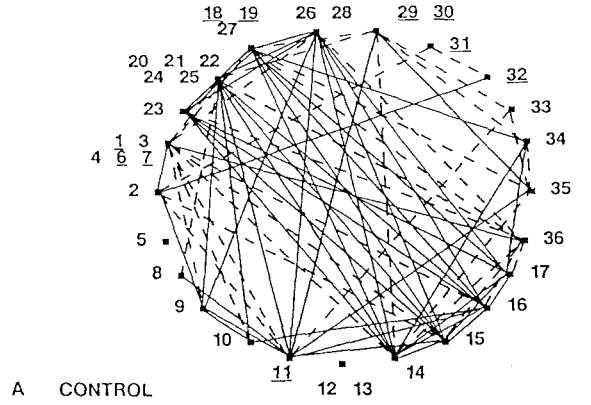
None of the genetic correlations which were significant in all three environments changed sign from one environment to another. This result contradicts previous findings of Giesel (1986), Gebhardt and Stearns (1988) and Newman (1988); namely, that genetic correlations become negative under stress environments.

The influence of the environment on the genetic correlations may have consequences for artificial multivariate selection. The control, which was an environment that allowed a high phenotypic expression without minimizing the micro-environmental variations, showed few genetic correlations. Consequently, this environment and more generally environments without stress and with no way to minimize the micro-environmental variations may favor multicharacter artificial selection when no genetic correlation between characters is required and when heritabilities are high enough. Conversely, selection under stressful conditions might be a way to increase the efficiency of selection, either through a direct increase of heritability or through an increase of genetic correlations between characters under selection showing a low heritability and other characters showing a high heritability.

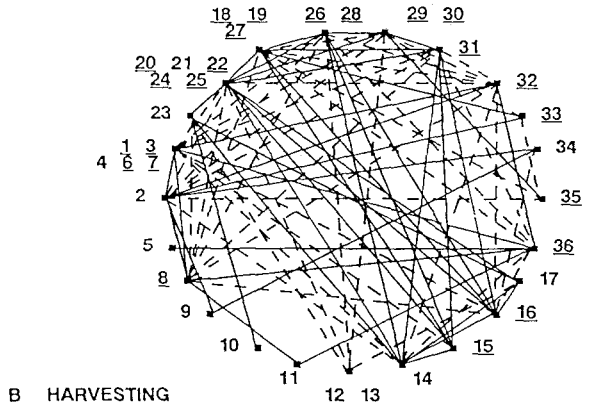
Phenotypic correlation matrices

Phenotypic correlations among the 36 traits are shown in Fig. 2A–C and described in Table 3.

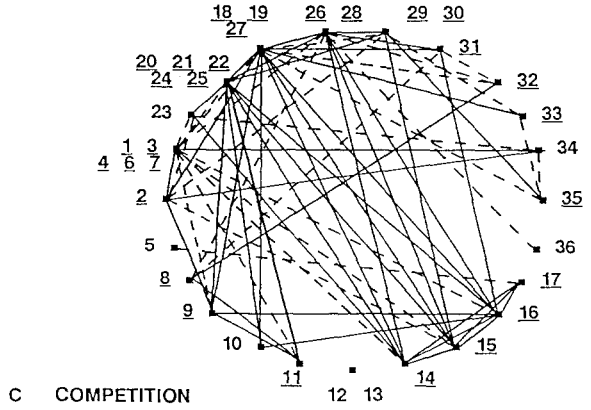
Fig. 2A–C Phenotypic correlations among all the 36 traits in control (**A**), harvesting (**B**) and competition (**C**) environments. The correlations among traits which were significantly different from zero at $P < 0.05$, $P < 0.01$ and $P < 0.001$ are represented by *lines* connecting the traits. *Solid lines* indicate positive correlations; *dashed lines*, negative correlations. The traits which are *underlined* shows significant genetic variability.



A CONTROL



B HARVESTING



C COMPETITION

Effect of the environment on the phenotypic correlation matrices

The number of significant phenotypic correlations was not significantly different in the three environments but tended to be higher in the control (chi2 test $P > 0.05$, Table 3). The correlations among the phenotypic correlation matrices in the three different environments were positive and large (Table 5). This result, which shows the proportionality of the phenotypic matrices, was confirmed by the tests of equality of correlation matrices of Jenrich, which were non significant. The control showed the highest number of phenotypic correlations that were significantly different from zero at $P < 0.001$ (chi2 test $P < 0.08$, Table 3). Since stress reduced the phenotypic expression, it also reduced the magnitude of the phenotypic correlations. This result is in agreement with those of Cuartero and Cubero (1982), Murphy et al. (1983) and Scheiner et al. (1991), but in contrast to that of Primack and Antonovics (1981).

The significant phenotypic correlation between days when all pods are ripe (8) and percentage of embryogenic dormancy (32) was the only one that changed sign from one environment to another (cut to competition environment). The sign of the phenotypic correlations was not influenced by the environment. This is in contrast to results obtained by Gebhardt and Stearns (1988).

The basic structure of the three phenotypic correlation matrices was given by the structure of the matrix of the competition environment (Fig. 2A–C). The phenotypic correlation matrix of the competition environment had a large number of correlations which were common with those of the matrices of the control and harvesting environment (Table 4). The matrix of the harvesting environment showed slightly more correlations oriented towards the percentages of tegumentary and embryonic dormancy and adult survival rate (31, 32 and 36), days when all pods are ripe (8) and rates of growth on the major and minor axes (12 and 13) than did the matrix of the competition environment (Fig. 2B–C).

Table 5 Correlations among the genetic (G) and phenotypic (P) correlation matrices in the control, harvesting and competition environments (All the correlations among matrices are significantly different from zero at $P < 0.001$)

		Control		Harvesting		Competition	
		P	G	P	G	P	G
Control	P	1	0.96 *45*[a]	0.79 *630*	–	0.83 *630*	–
	G	–	1	– *36*	0.91	– *45*	0.95
Harvesting	P	–	–	1	0.97 *253*	0.82 *630*	–
	G	–	–	–	1	–	0.88 *231*
Competition	P	–	–	–	–	1	0.92 *406*
	G	–	–	–	–	–	1

[a] Number of correlations (in italics)

The matrix of the control showed slightly more correlations involving pod set duration (11), size at flowering (17) and percentage of adult survival rate (36) than did the matrix of the competition environment (Fig. 2A, C). We found that the environment affected the structure of the phenotypic correlation matrices, but no trend was detected, results which are in agreement with Schilting (1989) and Falkenhagen (1989).

Comparison of the genetic and phenotypic correlation matrices (among the 36 traits)

In general, there were many more significant phenotypic correlations than significant genetic correlations in all three environments, but this was specially conspicuous in the control environment (chi2 test $P < 0.001$, Table 3). The main reason for this difference between phenotypic and genetic correlation matrices was that genetic correlations among traits with no genetic variability were undefined.

The environment had no influence on the number of significant phenotypic correlations, which is opposite to that found for the number of significant genetic ones. The influence of the environment on the structure of the phenotypic correlation matrices was different from that on the structure of the genetic correlation matrices (Figs. 1A–C and 2A–C).

The disparity between the phenotypic and genetic correlation matrices was most marked in the control and the least marked in the competition environment, therefore the similarity between the phenotypic and genetic correlation matrices increased with increasing environmental stress, but still remained too low to allow for the substitution of genetic correlations by phenotypic ones, as has been proposed by Cheverud (1988) and not recommended by Willis and Coyne (1990). As the phenotypic correlations differed from the genetic correlations principally because the genetic correlations among traits which had no genetic variability were undefined, the genetic and phenotypic correlations among only those traits which had a significant genetic variance in each respective environment were compared (i.e. 10 traits in the control, 23 in the harvesting environment, and 29 in the competition environment). As the phenotypic correlations are linear combinations of genetic and environmental correlations, the environmental correlations were also studied.

Comparison between genetic, phenotypic and environmental correlation matrices among only those traits that showed genetic variability in each respective environment

Phenotypic and environmental correlations among only those traits which showed genetic variability in each respective environment are shown in Figs. 3A–C and 4A–C and described in Table 3.

There were more significant phenotypic and environmental correlations than genetic correlations in the harvesting and competition environments (chi2 test $P < 0.01$) but not in the control (chi2 test $P > 0.50$, Table 3). The magnitudes of the significant phenotypic and environmental correlations was similar to that of the genetic correlations, but as the critical phenotypic and environmental values (± 0.31 and ± 0.43, respectively) were lower than the critical genetic value (± 0.68), more phenotypic and environmental correlations were significant. The phenotypic correlations overestimated the number of significant genetic correlations, as was found when all 36 characters were considered. Whatever the environment, all of the significant genetic correlations were represented by the significant phenotypic correlations but not necessarily with exactly the same value and same level of significance (Table 4, S2). Therefore, if the phenotypic correlations are substituted for the genetic ones, the phenotypic correlations will overestimate the degree of significance of the significant genetic correlations. To avoid the overestimation of the degree of significance and the number of the genetic correlations by the phenotypic ones we took into account only those phenotypic correlations that were significant at $P < 0.001$. Then, the number of significant phenotypic correlations remained larger than that of the significant genetic correlations in all environments except the control (Table 3), and some significant genetic correlations were not represented by significant phenotypic correlations (Table 4, S2').

In contrast to the genetic correlations, the phenotypic correlations showed no clear influence of the environment on the type of characters that were very often involved in significant phenotypic correlations (Fig. 3A–C). This result is in agreement with those of Cuartero and Cubero (1982). The environmental correlations showed just such an influence, but only in the harvesting environment where most of the environmental correlations were for phenological traits (Fig. 4A–C). The sign of the phenotypic correlations was the same as that of the genetic correlations. This is in contrast with the result of Mitchell-Olds (1986).

We also compared the overall magnitudes and integration indexes of the genetic phenotypic and environmental correlation matrices in the different environments. The following comparisons are only indicative as no statistical test is available. In all three environments, the overall magnitude of the genetic correlation matrix was higher than that of the phenotypic and environmental correlation matrices (Table 6). The phenotypic correlation structure was less integrated than the genetic correlation structure but more integrated than the environmental one (Table 6).

Correlations between genetic, phenotypic and environmental correlation matrices were calculated in order to quantify the proportionality of the matrices in each environment. The phenotypic correlations were proportional to the genetic correlations (Table 5). Therefore, the genetic and phenotypic correlations varied in similar

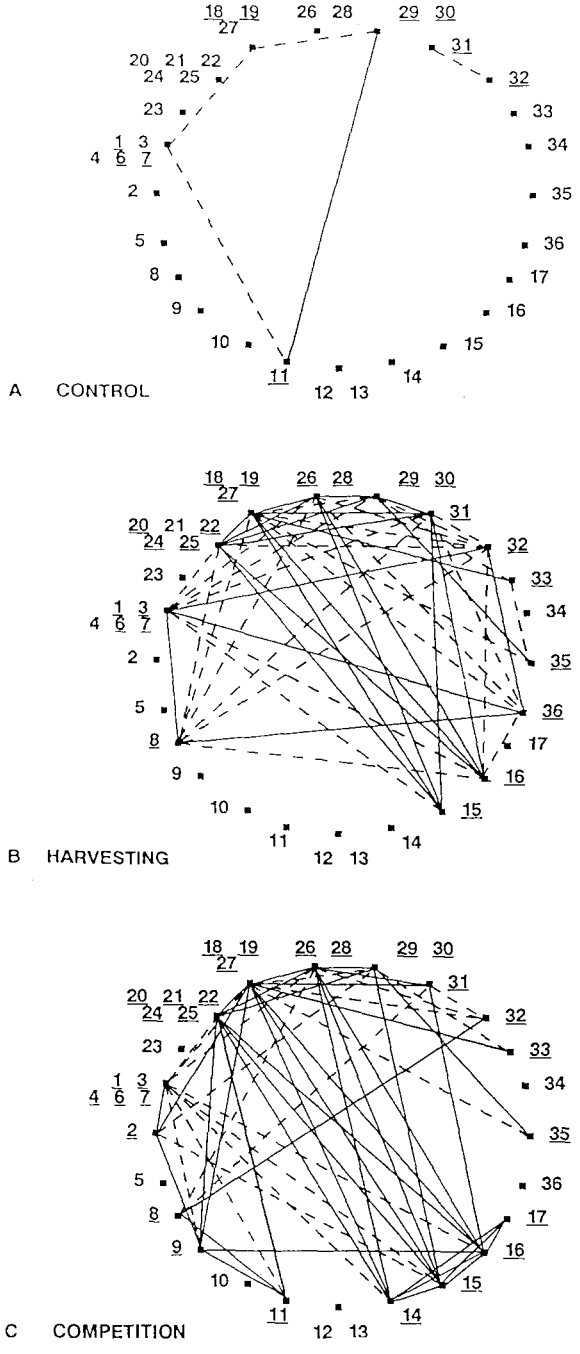


Fig. 3A–C Phenotypic correlations among traits which showed genetic variability in control (**A**), harvesting (**B**) and competition (**C**) environments

direction although they were not identical. This result is in agreement with those of Roff and Mousseau (1987) and Kohn and Atchley (1988). The average disparities between the phenotypic and genetic correlation matrices also showed that the genetic correlation matrix was different from the phenotypic correlation matrix in each environment, especially in the competition environment (Table 7).

In the three environments the significance of the genetic correlations and the structure of the genetic

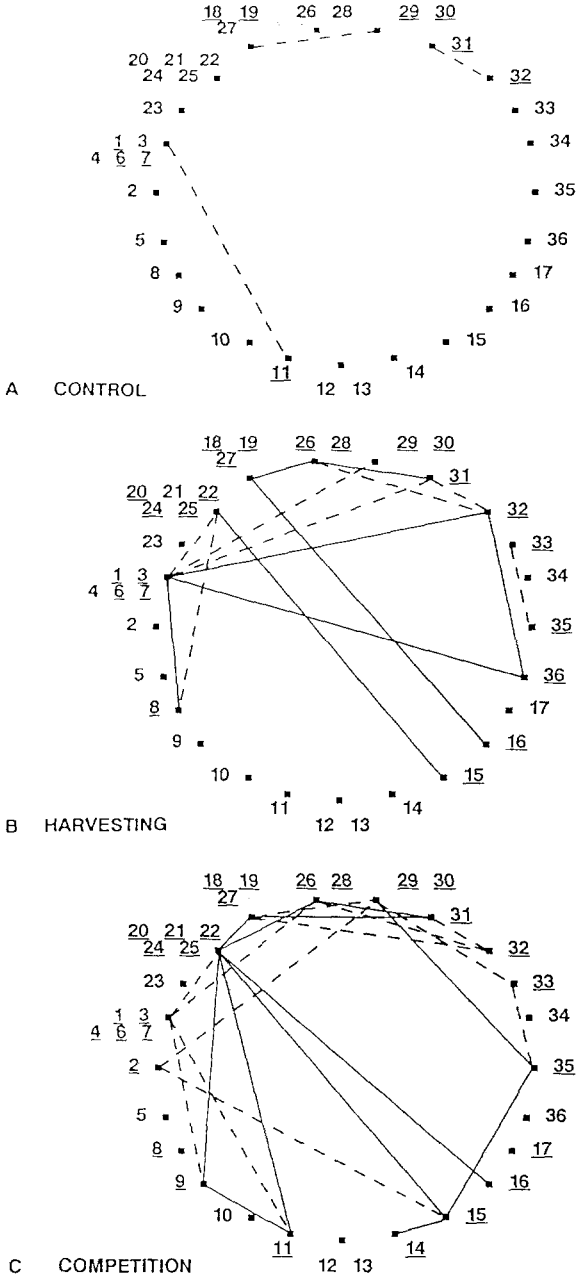


Fig. 4A–C Environmental correlations among traits which showed genetic variability in control (**A**), harvesting (**B**) and competition (**C**) environments

correlation matrix were not consistent with those of the phenotypic correlations among those traits that showed genetic variability. Even in the control where the phenotypic correlation matrix had a pattern similar to that of the genetic correlation matrix, the phenotypic correlation matrix still overestimated the number of significant correlations (Tables 3, 4). Our results confirm the conclusion of Willis and Coyne (1991) and Harding et al. (1990) that the phenotypic correlation cannot be used in the place of a genotypic correlation due to a lack of correspondence between phenotypic and genetic correlation matrices for traits that show genetic variability.

Table 6 Overall magnitudes and integration indexes of the genetic (G), phenotypic (P') and environmental (E) correlation matrices in the control, harvesting and competition environments among only those traits which showed genetic variability in each respective environment

Matrix		Control *45*[a]	Harvesting *253*	Competition *406*
Overall	G	0.45	0.46	0.48
	P'	0.36	0.41	0.37
Magnitude	E	0.34	0.39	0.34
Integration	G	0.34	0.23	0.32
	P'	0.24	0.18	0.20
Index	E	0.19	0.13	0.17

[a] Number of correlations (in italics)

Table 7 Average disparity between phenotypic (P') and genetic (G) correlation matrices in the control, harvesting and competition environments

	Control	Harvesting	Competition
Average disparity	0.13	0.12	0.15

One main reason evoked to explain why the pattern of the phenotypic correlation matrix is different from that of the genetic correlation matrix has been that the environmental correlations are important and may differ in sign from the genetic correlations (Cuartero and Cubero 1982; Giesel 1982; Harding et al. 1990). In our experiment, the environmental correlations were less important than the genetic ones: in each environment the overall magnitude of the environmental correlations was smaller than that of the genetic correlations (Table 6); moreover, the number of significant environmental correlation at $P < 0.001$ was not significantly different from that of the genetic correlations (Table 3, chi2 test $P > 0.05$). The number of significant negative environmental correlations was not significantly different than of the genetic correlations, except in the harvesting environment (Table 3, chi2 test $P < 0.05$). Some environmental correlations had a sign opposite to that of the genetic correlations, but these were never significant. Thus, the corresponding phenotypic correlations were lower than the genetic correlations, but they generally still remained significant.

We found that the disparity between phenotypic and genetic correlations when those traits which showed genetic variability were considered increased with increasing environmental stress.

Even if in one environment the phenotypic correlation matrix is equivalent to that of the genetic correlation matrix, because the genetic correlation matrix is influenced by the environment the extrapolation of this result to any environment is possible only if the phenotypic correlation matrix is also influenced by the environment in the same way as the genetic correlation matrix. In our study, the influence of the environment on

the phenotypic correlation matrix differed from its influence on the genetic correlation matrix when either all of the traits or only those which showed genetic variability in each environment were considered. This result is in agreement with those of Cuatero and Cubero (1982) and Scheiner et al. (1991).

Our results are in disagreement with those from many earlier studies (Mitchell-Olds 1986; Roff and Mousseau 1987; Khon and Atchley 1988; Cheverud 1982) because these authors have compared the magnitude, sign and pattern of phenotypic and genetic correlations only among traits that show genetic variability, and only in one environment. Consequently, they have considered only particular cases and have not compared the effect of the environment on the phenotypic and genetic correlations.

Acknowledgements The text was greatly improved and received constructive criticism from J. D. Thompson. We thank G. Gintzburger, V. Perrot, M. Angevain, and especially J. M. Prosperi for providing most of the accessions used in this experiment. Finally, we would like to thank M. Angevain, M. Vabre, C. Raynaud, B. Serrate and F. Lechevallier for very helpful field assistance. Results presented here constitute part of the PhD thesis of the first author. Financial support for this study was provided by the European Economic Community (contract no. 6019, INRA 4202A, AGRIMED program, attributed to Paul Mansat, head of the laboratory).

References

Becker WA (1984) Quantitative genetics. Washington State University Press, Seattle, Wash.

Cheverud JM (1982) Phenotypic, genetic and environmental morphological integration in the cranium. Evolution 36:499–516

Cheverud JM, Rutledge JJ, Atchley WR (1983) Quantitative genetics of development: genetic correlations among age-specific trait values and evolution of ontogeny. Evolution 37:895–905

Clarker AG, Keith LE (1988) Vaiation among extracted lines of *Drosophila melanogaster* in triacylglycerol and carbohydrate storage. Genetics 119:595–607

Cuartero J, Cubero JI (1982) Phenotypic, genotypic and environmental correlation in tomato (*Lycopersicon esculentum*). Euphytica 31:151–159

Dagnélie P (1986) Théorie et méthodes statistiques, vol 2, 2nd edn. Les presses agronomiques de Gembloux, Gembloux, France

Falconer JS (1981) Introduction to quantitative genetics, 2nd edn. Longman, New York

Falkenhagen ER (1989) Influences of testing sites on the genetic correlations in open-pollinated family traits of *Pinus elliotti* in South Africa. Theor Appl Genet 77:873–880

Gallais A (1990) Théorie de la sélection en amélioration des plantes. (Collection sciences agronomiques.) Masson, Paris

Gallais A, Vincourt P, Bertholleau JC (1983) Etude de critères de sélection chez le maïs fourrage. Héritabilities, corrélations génétiques et réponse attendue à la sélection. Agronomie 3: 751–760

Gebhardt MD, Stearns SC (1988) Reaction norms for developmental time and weight at eclosion in *Drosophila mercatum*. J Evol Biol 1:335–354

Giesel JT (1986) Genetic correlation structure of life history variables in outbred, wild *Drosophila melanogaster*: effects of photoperiod regime. Am Nat 128:593–603

Giesel JT, Murphy PA, Manlove MN (1982) The influence of temperature on genetic interrelationship of life history traits in a population of *Drosophila melanogaster*: what tangled data sets we weave. Am Nat 119:467–479

Groeters FR, Dingle H (1987) Genetic and maternal influences on life history plasticity in response to photoperiod of milkweed bugs (*Oncopeltus fasciatus*). Am Nat 129:332–346

Harding J, Huang H, Byrne T, Huang N (1990) Quantitative analysis of correlations among flower traits in *Gerbera hybrida Compositae*. 1. Genetic and environmental correlations. Theor Appl Genet 80:552–558

Huang H, Harding J, Byrne T, Huang N (1990) Quantitative analysis of correlations among flower traits in *Gerbera hybrida Compositae*. 2. Direct and mean correlated respose to selection. Theor Appl Genet 80:559–563

Jenrich RI (1970) An asymptotic chi2 test for the equality of two correlation matrices. J Am Stat Assoc 65:904–912

Kohn LA, Atchley WR (1988) How similar are genetic correlation structures? Data from mice and rats. Evolution 42:467–481

Lesins KA, Lesins I (1979) *Genus Medicago* (Leguminosae), a taxogenetic study. Dr. W. Junk, The Hague

Mitchell-Olds T (1986) Quantitative genetics of survival growth in *Impatiens capensis*. Evolution 40:107–116

Mode JC, Robinson HF (1959) Pleitropism and the genetic variance and covariance. Biometrics 15:518–537

Murphy PA, Giesel JT, Manlove MN (1983) Temperature effects on life history variation in *Drosophila simulans*. Evolution 37:1181–1192

Newman RA (1988) Adaptative plasticity in development of *Scaphopus couchii* tadpoles in desert ponds. Evolution 42:774–783

Primack RB, Antonovics J (1981) Experimental ecological genetics in Plantago. V. Components of seed yield in the ribwort plantain *Plantago lanceolata* L. Evolution 35:1069–1079

Reeve ECR (1955) The variance of the genetic correlation coefficient. Biometrics 11:357–374

Roff DA, Mousseau T (1987) Quantitative genetics and fitness: lessons from *Drosophila*. Heredity 58:103–118

SAS Institute (1985) SAS/STAT Guide for personal computers. SAS Institute Inc, Cary, N. C.

Scheffé H (1959) The analysis of variance. John Wiley and Sons, New York

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

Scheiner SM, Caplan RL, Lyman RF (1991) The genetics of phenotypic plasticity. III. Genetic correlations and fluctuating asymmetries. J Evol Biol 4: 51–68

Schilchting CD (1989) Phenotypic plasticity in *Phlox*. II. Plasticity of chatacter correlations. Oecologia 78:496–501

Service PM, Rose MR (1985) Genetic covariation among life-history components: the effect of novel environments. Evolution 39:943–945

Shaw GR (1986) Response to density in a wild population of the perennial herb *Salvia lyrata*: variation among families. Evolution 40:492–505

Tallis GM (1959) Sampling errors of genetic correlation coefficients calculated from analyses of variance and covariance. Aust J Agric Res 10:35–43

Van Noordwijk AJ, Gebhart M (1987) Reflections on the genetics of quantitative traits with continuous environmental variation. In: Loeschke V(ed) Genetic constraints on adaptative evolution. Springer, Berlin Heidelberg New York

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

Willis JH, Coyne JA (1991) Can one predict the evolution of quantitative characters without genetics? Evolution 45:441–444