

THE ECOLOGY AND GENETICS OF FITNESS IN *CHLAMYDOMONAS*. X. THE RELATIONSHIP BETWEEN GENETIC CORRELATION AND GENETIC DISTANCE

REES KASSEN¹ AND GRAHAM BELL²

Department of Biology and Redpath Museum, McGill University, 1205 Avenue Docteur Penfield,
Montréal, Québec H3A 1B1, Canada

¹E-mail: rkassen@bio1.lan.mcgill.ca

²E-mail: gbell2@maclan.mcgill.ca

Abstract.—A necessary condition for the maintenance of genetic variation in heterogeneous environments is that the relative fitnesses of a collection of genotypes vary as conditions of growth change. This can be detected by estimating the amount of gene-by-environment interaction ($G \times E$) when a range of types are tested across a range of conditions. However it is the sign and magnitude of the genetic correlation, which is a component of $G \times E$, that governs the ultimate fate of variation. Whether genetic variation will be preserved, then, depends on how the genetic correlation changes as a function of the ecological differences among environments and the genetic differences among genotypes. To evaluate this, we assayed the performance of 15 chlorophyte species of known genetic relation in 20 environments. We found that the quantity of $G \times E$ increased as both the environmental variance across environments and the genetic distance increased. Moreover the genetic correlation declined as the environmental variance between pairs of environments and the genetic distance between pairs of genotypes increased. These results suggest that divergent selection will be more likely to maintain genetic variation when environments are strongly contrasted and genotypes widely divergent.

Key words.—*Chlamydomonas*, diversity, genetic correlation, genetic distance, genotype-by-environment interaction.

Received November 13, 1998. Accepted October 26, 1999.

Genetic diversity can be preserved by divergent selection in heterogeneous environments. This requires that relative fitness varies with the conditions of growth, such that each type is well adapted to some sites, but poorly adapted to others. If a range of types is tested across a range of sites, this variation will be expressed as genotype-by-environment interaction, or $G \times E$. This is readily detected and estimated in the laboratory, where $G \times E$ often constitutes a major component of variation for characters associated with fitness (Bell 1990), and it can be studied in the field by implant experiments (Schoen et al. 1994) or reciprocal transplant experiments (Futuyma and Phillippi 1987; Via 1991). Estimates from crop trials and laboratory experiments normally put the quantity of $G \times E$ between 40% and 60% of total genotypic variance, irrespective of whether the genotypes being tested are different species, varieties or strains within a species, or full-sibs (Simmonds 1981; Bell 1997).

The dynamics of variation in a heterogeneous environment depend on the magnitude and the nature of $G \times E$. In the first place, the quantity of $G \times E$ depends on the extent to which conditions at different sites vary: The greater the variation, the more $G \times E$ is expected. When $G \times E$ is generated through the crossing of reaction norms for fitness variation may be stably maintained in the population, with different types being well adapted to different environments. Although a necessary condition, it is by no means a sufficient one (Levene 1953; Gliddon and Strobeck 1975; Maynard Smith and Hoekstra 1980; Via and Lande 1987). Nevertheless, crossing reaction norms or equivalently negative genetic correlations across environments will greatly retard the loss of diversity even if the conditions for stability are not satisfied.

To see more clearly how the nature of $G \times E$ can effect the outcome of selection in heterogeneous environments, consider a collection of genotypes tested in two environments.

Robertson (1959) pointed out that the $G \times E$ they express can be written as:

$$\sigma_{GE}^2 = \frac{1}{2}(\sigma_{G1} - \sigma_{G2})^2 + \sigma_{G1}\sigma_{G2}(1 - \rho_{G1G2}), \quad (1)$$

where σ_{G1} and σ_{G2} are the genetic standard deviations of a character expressed in environments 1 and 2, respectively, and ρ_{G1G2} is the genetic correlation of that character across environments 1 and 2. The character concerned will be taken to be fitness or some attribute closely connected with fitness. This makes it clear how $G \times E$ can be understood as the sum of two components.

The first is the variance of the difference between the amount of overall genetic variation expressed in the two environments, $(\sigma_{G1} - \sigma_{G2})^2$. $G \times E$ will be generated if there is more genetic variance in one environment than the other, because the difference between any two genotypes will then depend on the environments where they are measured. This component will therefore create differences in the rate of response to selection among environments.

The second component involves the cross-environment genetic correlation, ρ_{G1G2} : $G \times E$ will be generated if the collection of genotypes respond inconsistently to environmental variation. When the cross-environment genetic correlation is negative, which implies that relative fitness changes with conditions of growth, selection favors different types in each environment, although with only two environments a maximum of two genotypes can be maintained at equilibrium ($\rho_{G1G2} = -1$ at equilibrium; Levene 1953). When there are more than two environments, selection can support at least as many types as there are environments (Strobeck 1974), although it is not immediately obvious what the cross-environment genetic correlation will be at equilibrium. One suggestion by Dickerson (1955) is that $r_G = -1/(N - 1)$ at equilibrium, where N is the number of environments. The

important point is that a negative cross-environment genetic correlation represents a necessary (but not sufficient) condition for the stable maintenance of diversity and will always retard the loss of diversity. The cross-environment genetic correlation has been shown to decrease as the macroenvironmental variance between pairwise combinations of environments increases (Bell 1992).

This is the conventional manner of interpreting $G \times E$, as a function of the environments in which a given collection of genotypes are tested (Cockerham 1963; Andersson and Shaw 1994; Cooper and DeLacy 1994; Wu and Stettler 1997). However, an equally appropriate interpretation is to see $G \times E$ as a function of the genotypes used to evaluate a collection of environments. This is not a new idea (e.g., Comstock and Moll 1963; Dickerson 1963; Simmonds 1981), although it appears to have been less widely appreciated than the conventional interpretation outlined above.

Suppose that we consider any two genotypes whose fitness we have measured across a range of environments, which is the converse of the previous situation. If they respond differently to these environments, the outcome will be $G \times E$. As before, this has two components that can be analyzed in the same way by relabeling genetic variances and correlations as environmental variances and correlations, respectively. Thus, Robertson's equation becomes:

$$\sigma_{GE}^2 = \frac{1}{2}(\sigma_{E1} - \sigma_{E2})^2 + \sigma_{E1}\sigma_{E2}(1 - \rho_{E1E2}). \quad (2)$$

Now σ_{E1} and σ_{E2} are the environmental standard deviations of a character expressed by genotypes 1 and 2, respectively; ρ_{E1E2} is the cross-genotype environmental correlation of that character across the two genotypes. The first component is the variance of the difference between the environmental standard deviations of the two genotypes: $G \times E$ will be generated if one is more responsive to environmental variation, because the difference between any two environments will then depend on which genotypes are used to measure them. The environmental variance of a genotype is a measure of its phenotypic plasticity so any nonzero quantity for this component represents genetic variation in plasticity, some genotypes being more (or less) plastic than others.

The second component involves the cross-genotype environmental correlation. We might expect that two genotypes that are very similar—perhaps because they are very closely related—are likely to respond in parallel to environmental variation, so that the cross-genotype environmental correlation will be close to +1. As the genotypes become more dissimilar, they are more likely to respond differently over any given range of conditions, so the environmental correlation will fall. It might even become negative for genotypes that are very dissimilar. This would then constitute a second rule governing the quantity of $G \times E$: The cross-genotype environmental correlation tends to fall as the genetic variance increases.

However, environmental variances and correlations are not very useful in understanding the maintenance of diversity because the theory requires that we know the genetic correlation to predict the outcome of selection. Moreover, there is no straightforward transformation that allows us to convert

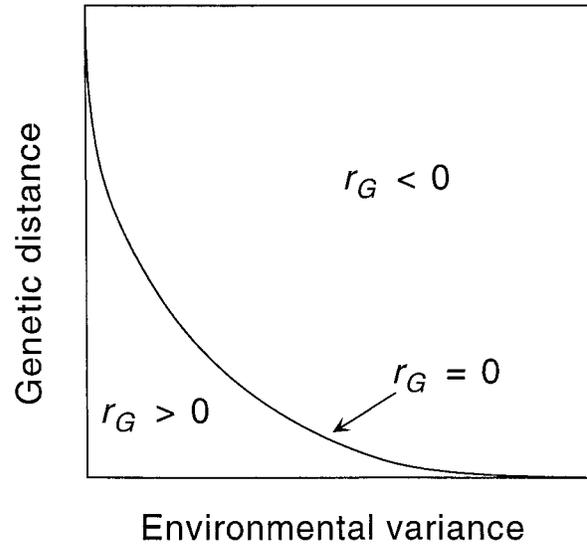


FIG. 1. The genetic correlation in relation to the similarity among genotypes and environments. See explanation in the text.

an environmental correlation into a genetic correlation, so we must find a way to recast this analysis in terms of a cross-environment genetic correlation.

The simplest method, which is also due to Robertson (1959), is to calculate for every pair of genotypes the product-moment correlation across all pairs of environments. Because each genotype-environment pair gives a value for the cross-environment genetic correlation of either -1 or $+1$, the average across all environments for any given pair of genotypes is an estimate of the cross-environment genetic correlation between them. We expect that as genotypes diverge the genetic correlation between them will decrease.

This analysis can be taken one step further. By considering a pair of genotypes in a pair of environments, we can define a genetic similarity between the genotypes and an environmental similarity between the environments. For all pairs of genotypes and environments that fall within some defined range of genetic and environmental similarity, we can calculate the average genetic correlation using Robertson's method. The result thus represents the expected outcome of selection among a set of genotypes of given similarity within a particular environmental context. The schematic in Figure 1 outlines our expectation: The cross-environment genetic correlation should decrease as both genotypes and environments become less similar. Consequently, one should be able to define an isocline where $r_G = 0$. On one side of this isocline ($r_G > 0$) either genotypes or environments are too similar for diversity to be maintained; diversity can only be supported by divergent selection on the other side of the isocline ($r_G < 0$), where either genotypes or environments are sufficiently dissimilar. We emphasize again that this is only a necessary, but not sufficient, condition for the maintenance of diversity.

In this study we examine this interpretation of the nature and quantity of $G \times E$. We are specifically concerned with that component of $G \times E$, the cross-environment genetic correlation, that governs whether diversity could be preserved in a heterogenous environment. Our results are relevant to situations where selection sorts from among an initially di-

verse set of types in a heterogenous environment, as in the model of Levene (1953) and its descendants (Dempster 1955; Maynard Smith and Hoekstra 1980; Gliddon and Strobeck 1975). The types may be asexual genotypes within a population or species within a community because the theory requires no formal distinction between the two (e.g., Van Tien-deren 1997).

MATERIALS AND METHODS

Study Species and Genetic Distances

We studied 13 species from the chlorophyte genus *Chlamydomonas* and two species from related genera. The species were obtained from the University of Texas Culture Collection of Algae (UTEX), except for three of the *C. reinhardtii* strains, which were from our own laboratory stocks. The species are listed in the Appendix along with their UTEX accession numbers. The genus *Chlamydomonas* has been undergoing extensive taxonomic revision, so both the current and the former taxonomic designations are given in the Appendix.

Genetic distances were obtained from phylogenetic trees published by Buccheim et al. (1996), who provide details on taxon sampling, the methods used to obtain the sequences, and the programs used to calculate the genetic distances. Briefly, the species chosen for sequencing represent the nearly the entire range of biochemical and morphological characters traditionally used to distinguish species of *Chlamydomonas* (Ettl 1976; Schlosser 1984). Genetic distances were calculated for the nuclear-encoded small subunit rRNA and the chloroplast-encoded large subunit rRNA sequences using Kimura's (1980) two-parameter model of nucleotide change.

Environments

In October 1996, we collected soil samples from 10 habitats at the Seed Farm and Morgan Arboretum of MacDonald Campus, McGill University, Quebec. The habitats were agricultural fields (soybean, clover, alfalfa, and corn), second-growth forests (mixed deciduous-coniferous, beech-maple, larch), and pond-side vegetation. Two sites were sampled within each habitat, giving a total of 20 different environments. A preliminary analysis of the performance of all genotypes in each site revealed substantial variation among sites nested within habitats, so we treat each site as a separate environment in the analysis (nested ANOVA; habitats: $F = 55.24$, $df = 9$, $P < 0.0001$; sites nested within habitats: $F = 7.80$, $df = 10$, $P < 0.0001$).

Measuring Performance

Approximately 50 ml of wet soil from a single site were placed in a "teabag" of Nitex fabric (pore diameter $\approx 10 \mu\text{m}$) and allowed to soak in 1200 ml of distilled water overnight at 4°C. This infusion was not autoclaved, because this would alter its chemical composition and nutrient status. We inoculated 100- μl samples from liquid preinoculation cultures (grown in standard Bold's medium; see Harris 1989) into 20 ml of soil-water infusions. In our experience, about 10% of uninoculated infusions will grow green algae, but the growth of native algae is almost always suppressed by the

rapid growth of the *Chlamydomonas* inoculum. We never observed the filamentous or flake-like forms that usually appear in uninoculated cultures, and all the experimental cultures were green suspensions without appreciable fungal or bacterial growth. The cultures were grown in screw-top glass tubes kept in racks under constant illumination at room temperature ($25 \pm 1^\circ\text{C}$). We monitored changes in cell density every two days by recording transmittance at 665 nm using a Bausch and Lomb (Rochester, NY) Spec-20 digital spectrophotometer. There were two replicates set out as separate randomized blocks. Thus, for the experiment as a whole, there were 15 species in each of 20 environments and two replicates giving a total of 600 cultures. There was one missing value caused by a broken tube. Our measure of performance was the carrying capacity, K , of each culture, which was estimated as the maximum cell density over the one month period of the experiment. K would be sufficient to describe fitness in environments that remain undisturbed over long periods of time.

Analysis

The species \times sites matrix was analysed by a two-way analysis of variance (ANOVA) with species and sites treated as a random effects, using PROC GLM in SAS version 6.12. We then calculated three measures for every pairwise combination of environments and genotypes as follows.

Genetic parameters

(1) *The amount of overall $G \times E$.*—This is a variance component estimated by equating expected with observed mean squares from the analysis of variance.

(2) *The cross-environment genetic correlation of growth.*—This was calculated as the Pearson product-moment correlation coefficient of growth in one environment with growth in the other, over all genotypes. A correlation of +1 would imply a consistent relationship among genotypes with respect to growth regardless of environment, and thus the same ranking in every environment and the same expected outcome of selection. The degree to which the correlation departs from +1 thus measures the inconsistency of response by genotypes to environmental variation.

(3) *The variance of the genetic standard deviations in each pair of environments.*—This was calculated using $\frac{1}{2}(\sigma_{G1} - \sigma_{G2})^2$ from equation (1). A value of zero implies that the genetic variation expressed in the two environments is the same; a value greater than zero implies that one environment expresses more genetic variation than the other.

Environmental parameters

(4) *The amount of overall $G \times E$.*—Again, this is a variance component estimated by equating expected with observed mean squares from the ANOVA.

(5) *The cross-genotype environmental correlation of growth.*—This was calculated as the Pearson product-moment correlation coefficient of growth by one genotype with growth by the other, over all environments. A correlation of +1 would imply a consistent relationship among environments with respect to growth for each pair of genotypes mea-

TABLE 1. Analysis of variance for the entire experiment treating the sites within each habitat as different environments. All effects are random.

Source	df	MS	F	P	Variance components
Block	1	30,189	14.63	0.0002	94
Species	14	49,594	12.78	0.0001	1143
Sites	19	123,889	31.93	0.0001	4000
Species \times sites	266	3879	1.88	0.0001	908
Error	299	2064			2064

sured. The degree to which the correlation departs from +1 is a measure of the extent to which the ranking of environments with respect to growth differs between pairs of genotypes.

(6) *The variance of the environmental standard deviations between all pairs of genotypes.*—This was calculated using $\frac{1}{2}(\sigma_{E1} - \sigma_{E2})^2$ from equation (2). A value of zero implies that the two genotypes do not differ in their responsiveness to environmental variation; values greater than zero indicate the degree to which the two genotypes differ in their responsiveness.

The genetic parameters (1–3) were regressed against the standard deviation of the difference in mean performance within each environment (σ_{E1-E2} , environmental standard deviation), obtained from the variance of mean performance of all genotypes in a pair of environments (from the method of Finlay and Wilkinson 1963). This quantity thus represents the macroenvironmental variance displayed by a collection of genotypes tested in two environments, as opposed to the microenvironmental variance that arises through deviations between replicate cultures as a result of accidents of development and other sources of uncontrolled error. The environmental parameters (4–6) were regressed against the genetic distances obtained from Buccheim et al. (1996). Standard deviations of the variance parameters (1, 3, 4, and 6) were analyzed because these fit a linear least-squares regression better than the variances themselves. Because these analyses make use of factorial combinations of environments or genotypes, conventional parametric statistics are not applicable. We tested the significance of the regression slopes using Manly's (1992) randomization procedure ($n = 10,000$ randomizations).

RESULTS

Statistical Analysis of the Species \times Site Matrix

An analysis of variance for the entire experiment is shown in Table 1. There was substantial variance among species and among environments. The species \times site interaction variance component was similar in magnitude to the among-species variance component. Block effects were also significant. Therefore, the analyses that follow have been adjusted by equating all values to the mean for both replicates through the addition (or subtraction) of a constant.

Analysis of Genetic Parameters

The quantity of $G \times E$ interaction increased as the variance of growth conditions between pairs of sites increased (Fig.

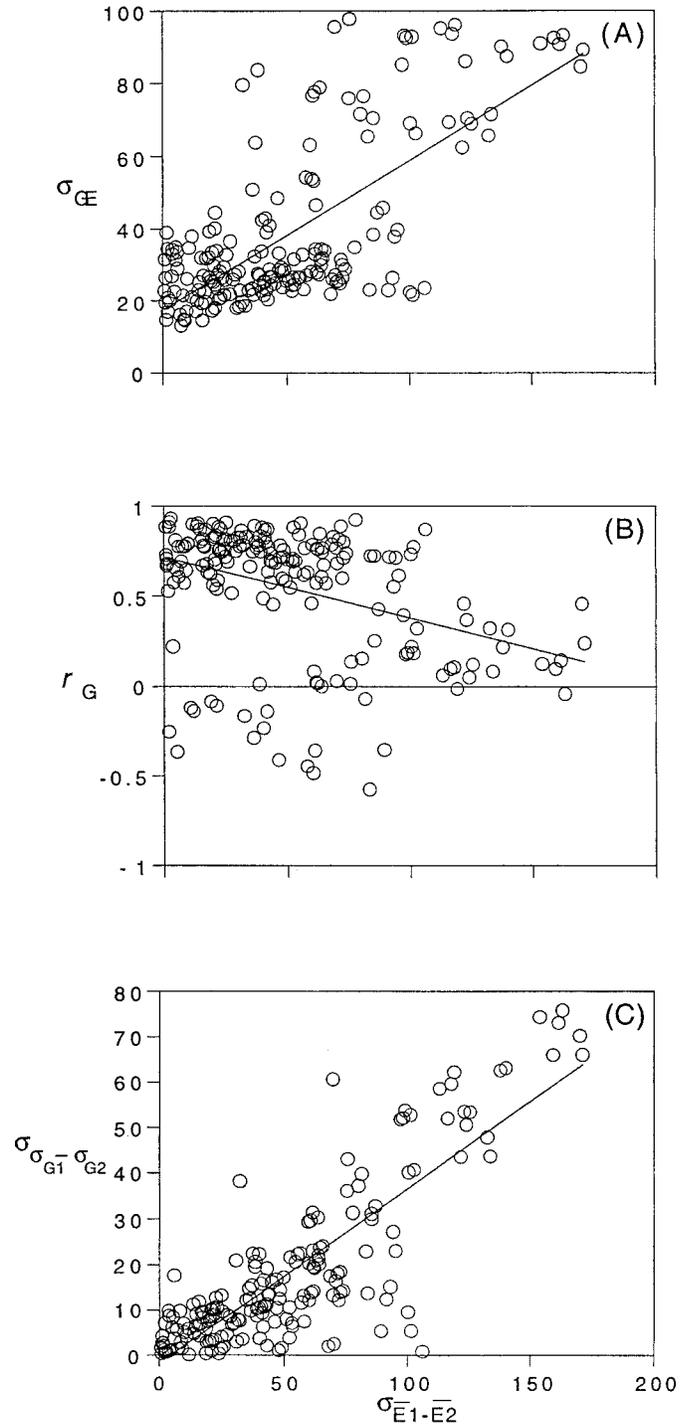


FIG. 2. Analysis of genetic parameters. Genotype-by-environment interaction increases with increasing mean environmental standard deviation (A). The genetic correlation decreases (B) and the variance of genetic standard deviations (C) increases with increasing environmental standard deviation.

2A; $r^2 = 0.51$, $P < 0.0001$), suggesting that the performance of genotypes becomes more different as conditions of growth become more different.

The cross-environment genetic correlation decreased as the mean environmental standard deviation increased (Fig. 2B;

$r^2 = 0.38$, $P \leq 0.0014$). Thus, the increase in $G \times E$ interaction observed above is due in part to reaction norms that cross within the range of environments tested. The mean genetic correlation among all 190 pairwise combinations of sites was 0.54, which is substantially higher than has been found in previous experiments using chemically defined media (Bell 1990, 1992). One site gave negative genetic correlations for nearly all site combinations. Removing it improves the relationship substantially, although it does not change the overall pattern of the results ($r^2 = 0.60$, mean $r_G = 0.62$).

The equality of genetic variation across pairs of sites, which is measured by the variance of the genetic standard deviations expressed in each pair of environments, and the mean environmental standard deviation are shown in Figure 2C. The relationship is strongly positive ($r^2 = 0.71$, $P \leq 0.0001$). Removing the site with consistently negative correlation does not improve the relationship ($r^2 = 0.71$). A large portion of the increase in $G \times E$ is therefore attributable to changes in the amounts of genetic variance expressed in different environments, implying that at least some genes have environment-specific effects.

Analysis of Environmental Parameters

The quantity of $G \times E$ expressed by pairs of genotypes increased for both measures of genetic distance (Fig. 3A; $r^2 = 0.14$, $P \leq 0.0002$; we report only the results for nuclear genetic distance because the chloroplast data always gave comparable results).

Figure 3B shows the relationship between the cross-genotype environmental correlation and genetic distance. The cross-genotype environmental correlation is close to +1 among the most similar genotypes in this experiment (strains of the same species) and declined toward zero as the genetic distance increased ($r^2 = 0.32$, $P \leq 0.0001$). Removing the same site as before does not change the overall relationship, nor does it substantially improve the fit ($r^2 = 0.36$).

The variance of environmental standard deviations expressed by pairs of genotypes, which measures the similarity in responsiveness, increased with genetic distance (Fig. 3C; $r^2 = 0.11$, $P \leq 0.0006$). Thus, the increase in $G \times E$ interaction that we observed was generated by both the inconsistency with which species rank environments and by differences in the overall breadth of adaptation.

Relationship between Genetic Distance and Genetic Variance

In a manner analogous to the analysis of genetic parameters above, it should be possible to use the variance between the mean performance of two genotypes across all environments as a measure of genetic divergence (just as the variance of the mean of all genotypes in two environments is a measure of the extent to which their conditions of growth differ). We plotted this variance in mean performance for each pair of genotypes against nuclear genetic distance. This was positive, as expected, but very weak and only marginally significant ($r^2 = 0.03$, $P \leq 0.07$; data not shown). This generated a correspondingly weak relationship between the variance of genotype mean performances and both $G \times E$ (positive, P

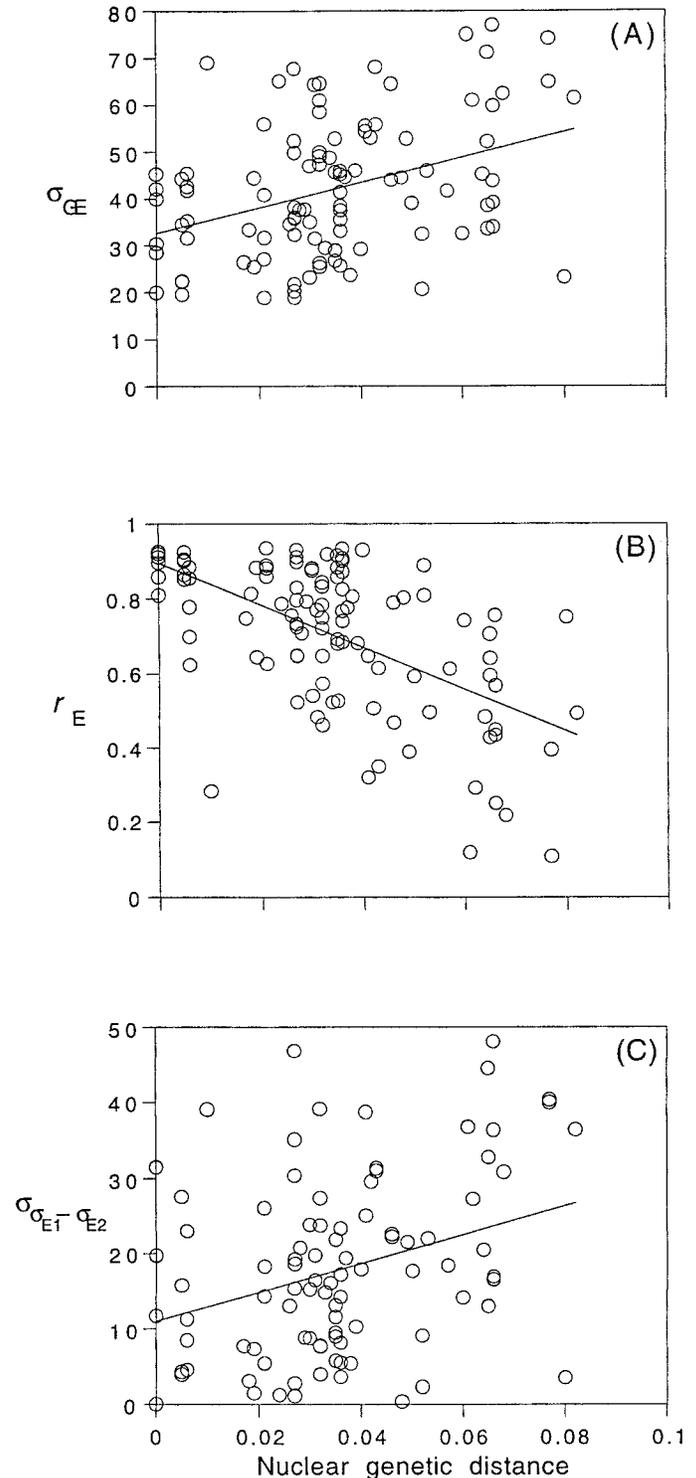


FIG. 3. Analysis of environmental parameters. Genotype-by-environment interaction increases with increasing genetic distance (A). The environmental correlation decreases (B) and the variance of environmental standard deviations increases (C) with increasing genetic distance.

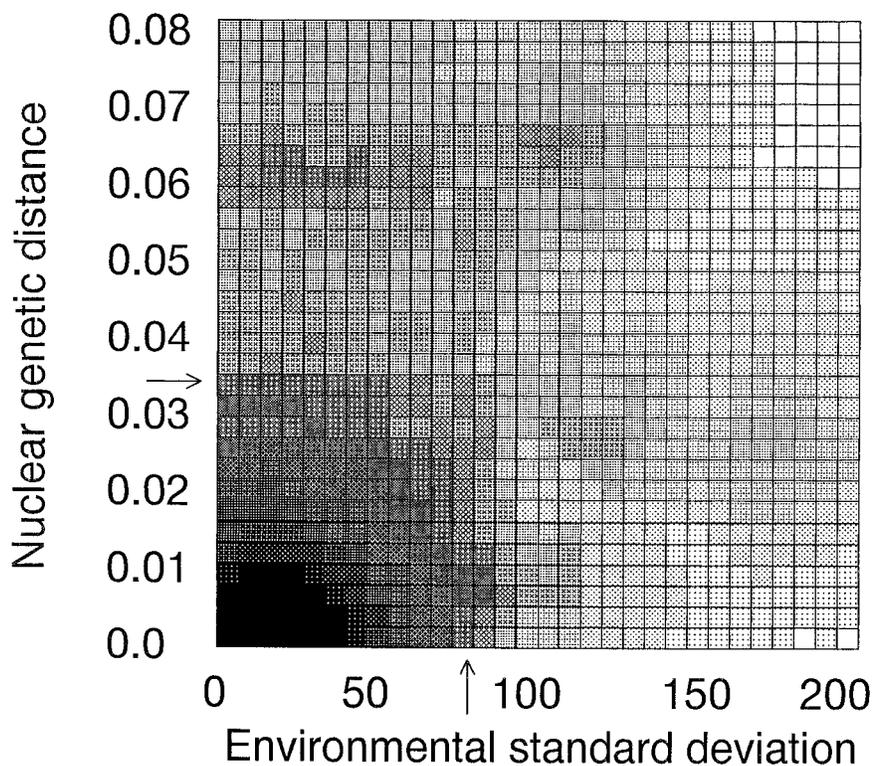


FIG. 4. The genetic correlation in relation to the similarity among genotypes and environments. Dark regions represent positive correlations; light regions are negative correlations. The arrows mark the points at which the isocline of zero genetic correlation crosses the environmental and genetic axes. The genetic correlation between pairwise combinations of genotypes and environments decreases as a function of the mean environmental standard deviation and the genetic distance.

≤ 0.07) and cross-genotype environmental correlation (negative, $P \leq 0.43$).

The Response of Genetic Correlation to Phylogenetic and Ecological Divergence

We calculated the cross-environment genetic correlation of each pairwise combination of species and sites to examine the effects of both genetic divergence and ecological disparity on the genetic correlation. This produces approximately 19,000 possible combinations, each of which gives a value for the genetic correlation of +1, 0, or -1. We then divided each axis into 20 units at equal intervals and calculated the average genetic correlation within each of the 400 regions. Regions containing fewer than 30 datapoints were discarded and the remainder (166 regions) plotted by Kriging (tension = 0.5; SYSTAT 1996) to produce Figure 4, which can be compared with the expected outcome depicted in Figure 1.

DISCUSSION

$G \times E$ interaction increased in a regular fashion with increasing mean environmental standard deviation and increasing genetic distance. Figure 4 illustrates how the cross-environment genetic correlation, which is that component of the $G \times E$ interaction that reflects changes in the ranking of genotypes across environments, changes as environments become more different and genotypes diverge: The cross-environment genetic correlation decreases along the diagonal from the lower left region toward the upper right region, as

expected. The line of zero genetic correlation crosses the environmental axis at an environmental standard deviation of approximately 80 units, representing a coefficient of variation of 61.5%, and the genetic axis at a distance of approximately 0.035 substitutions per base pair.

This result has a simple interpretation. Selection in the region to the left of the isocline, where the genetic correlation is positive, is expected to lead to the loss of genetic diversity. Selection in the region to the right of the isocline, where the cross-environment genetic correlation is negative, may lead to the maintenance of genetic diversity or at least greatly impede the rate at which it is lost. This suggests that selection will be least effective at removing variation either when environments are strongly contrasted or when genotypes are widely divergent. Diversity will tend to be maintained, therefore, when either environments or genotypes are sufficiently different.

G × E in Relation to Environment

Our study confirms previous reports (Bell 1992, 1997) that the cross-environment genetic correlation of growth decreases with increasing environmental variance. In natural environments, the environmental variance of physical factors such as soil nutrient status or pH increases with distance (Palmer 1990; Lechowicz and Bell 1991; Bell et al. 1993). Moreover, the environmental variance of plant growth itself likewise increases with distance (Bell and Lechowicz 1992). It seems reasonable to infer that the genetic correlation of growth

among sites in natural environments will tend to decrease with the distance between sites, although a direct demonstration of this would be welcome. If so we can conclude that diversity will tend to be preserved as environmental heterogeneity increases. No stronger statement can be supported because the maintenance of diversity, or the rate at which it is lost, will also depend on the magnitude of the cross-environment genetic correlation (Dickerson 1955; Via and Lande 1987), the amount of gene flow between sites (Endler 1973), the frequency of different kinds of site (Maynard Smith and Hoekstra 1980), and the manner of population regulation (Levene 1953, Dempster 1955).

G × E in Relation to Genotype

Our second conclusion, that the cross-environment genetic correlation of growth declines as genotypes become less similar or less closely related, is not as familiar, at least in population genetics. In community ecology, a similar proposition—the concept of limiting similarity—has been debated at length (see Abrams 1983). It was originally argued that species must occupy substantially different niches or must differ substantially in size or some other ecologically important attribute to coexist. The least difference that would reduce competition to the point where coexistence was possible was called the “limiting similarity” (MacArthur and Levins 1967). Our results operationalize the concept of limiting similarity through the use of the cross-environment genetic correlation: The isocline of $r_G = 0$ represents limiting similarity in an environment with a given degree of heterogeneity, in the sense that types that are more similar cannot coexist.

Although the pattern that we found was quite clear, it was not exactly what might have been expected. The genetic correlation of growth across environments might be expected to decline with the genetic variance, because this expresses the phenotypic difference among genotypes. The cross-environment genetic correlation would then also decline as genetic distance increased, but this would be an indirect effect caused by the phenotypic divergence of less closely related taxa, and would therefore be weaker. In fact, we observed a clear effect of ancestry, over large genetic distances, whereas the phenotypic effect was much weaker. The reason for this is that a large genetic variance implies a large difference in the mean fitnesses over environments of two given genotypes, one being on average much more fit than the other. It is therefore unlikely that the type with the lower mean fitness should be the more fit in either environment. The strong relationship with genetic distance may imply that the genetic correlation is most likely to be negative among types that have similar mean fitness across environments, but markedly different physiological attributes. This is close to the original concept of limiting similarity.

G × E in Relation to Genotype and Environment

The main importance of our study is that it defines the degree of similarity among environments and among genotypes that is required to support distinct types within an area. The conclusion that arbitrarily similar types can coexist seems fragile, even in a deterministic world, because similar

types are likely to express positive genetic correlation for growth across environments and diversity will then be eliminated rapidly by selection. Coexistence instead requires a divergence of phenotypic attributes sufficient at least to generate negative genetic correlation of fitness across environments. The extent of divergence that is required cannot be defined uniquely because it is conditional on the amount of environmental heterogeneity: only very different types can coexist when there is little environmental variance, whereas the requisite difference will decline as environmental variance increases. It will also depend on gene flow and other community properties. Empirical generalizations that apply to natural populations and communities will therefore be difficult to obtain, as Abrams (1983) pointed out, but the properties of $G \times E$ in this microbial system may justify a renewed investigation of limiting similarity.

ACKNOWLEDGMENTS

We thank T. Bernhardt and M. Richard for assistance with programming. Thanks also to K. Tallon for technical assistance and B. Latta for discussion. The paper was substantially improved by the criticisms of S. Via, M. Lynch, and three anonymous reviewers. This work was supported by a Natural Sciences and Engineering Research Council (NSERC) Canada Operating Grant to GB and an NSERC Postgraduate Fellowship to RK.

LITERATURE CITED

- Abrams, P. 1983. The theory of limiting similarity. *Annu. Rev. Ecol. Syst.* 14:359–376.
- Andersson, S., and R. G. Shaw. 1994. Phenotypic plasticity in *Crepis tectorum* (Asteraceae): genetic correlations across light regimes. *Heredity* 72:113–125.
- Bell, G. 1990. The ecology and genetics of fitness in *Chlamydomonas*. I. Genotype-by-environment interaction among pure strains. *Proc. R. Soc. Lond. B* 240:295–321.
- . 1992. The ecology and genetics of fitness in *Chlamydomonas*. V. The relationship between genetic correlation and environmental variance. *Evolution* 46:561–566.
- . 1997. Selection: the mechanism of evolution. Chapman and Hall, Toronto.
- Bell, G., and M. J. Lechowicz. 1991. The ecology and genetics of fitness in forest plants. I. Environmental heterogeneity measured by explant trials. *J. Ecol.* 79:663–685.
- Bell, G., M. J. Lechowicz, A. Appenzeller, M. Chandler, E. Deblois, L. Jackson, B. Mackenzie, R. Preziosi, M. Schallenberg, and N. Tinker. 1993. The spatial structure of the physical environment. *Oecologia* 96:114–121.
- Buccheim, M. A., C. Lemieux, C. Otis, R. R. Gutell, R. L. Chapman, and M. Turmel. 1996. Phylogeny of the Chlamydomonadales (Chlorophyceae): a comparison of ribosomal RNA gene sequences from the nucleus and chloroplast. *Mol. Phyl. Evol.* 5: 391–402.
- Cockerham, C. C. 1963. Estimation of genetic variances. Pp. 53–94 in W. D. Hanson and H. F. Robinson, eds. *Statistical genetics and plant breeding*. National Research Council Publication no. 982. National Academy of Sciences, Washington, DC.
- Comstock, R. E., and R. H. Moll. 1963. Genotype-environment interactions. Pp. 164–196 in W. D. Hanson and H. F. Robinson, eds. *Statistical genetics and plant breeding*. National Research Council Publication no. 982. National Academy of Sciences, Washington, DC.
- Cooper, M., and I. H. Delacy. 1994. Relationships among analytical methods used to study genotypic variation and genotype-by-environment interaction in plant breeding multi-environment experiments. *TAG* 88:561–572.

- Dempster, E. R. 1955. Maintenance of genetic heterogeneity. Cold Spring Harbor Symp. Quant. Biol. 20:25–32.
- Dickerson, G. E. 1955. Genetic slippage in response to selection for multiple objectives. Cold Spring Harbour Symp. Quant. Biol. 20:213–224.
- . 1963. Biological interpretation of the genetic parameters of populations. Pp. 95–107 in W. D. Hanson and H. F. Robinson, eds. Statistical genetics and plant breeding. National Research Council Publication no. 982. National Academy of Sciences, Washington, DC.
- Endler, J. 1973. Gene flow and population differentiation. Science 179:243–250.
- Ettl, H. 1976. Die Gattung *Chlamydomonas* Ehrenberg. Nova Hedwigia 49:1–1122.
- Finlay, K. W., and G. N. Wilkinson. 1963. The analysis of adaptation in a plant-breeding programme. Aust. J. Agric. Res. 14: 742–754.
- Futuyma, D. J., and T. E. Philippi. 1987. Genetic variation and covariation in responses to host plants by *Alsophila pometaria* (Lepidoptera: Geometridae). Evolution 41:269–279.
- Gliddon, C., and C. Strobeck. 1975. Necessary and sufficient conditions for multiple-niche polymorphism in haploids. Am. Nat. 109:233–235.
- Harris, E. 1989. The *Chlamydomonas* sourcebook. Academic Press, Toronto.
- Kimura, M. 1980. A simple method for estimating evolutionary rate of base substitution through comparative studies of nucleotide sequences. J. Mol. Evol. 16:111–120.
- Lechowicz, M. J., and G. Bell. 1991. The ecology and genetics of fitness in forest plants. II. Microspatial heterogeneity of the edaphic environment. J. Ecol. 79:687–696.
- Levene, H. 1953. Genetic equilibrium when more than one ecological niche is available. Am. Nat. 87:331–333.
- MacArthur, R. H., and R. Levins. 1967. The limiting similarity, convergence and divergence of coexisting species. Am. Nat. 101: 377–385.
- Manly, B. F. J. 1992. RT 1.02: a program for randomization testing. University of Otago, Dunedin, New Zealand.
- Maynard Smith, J., and R. Hoekstra. 1980. Polymorphism in a varied environment: how robust are the models? Genet. Res. 35: 45–57.
- Robertson, A. 1959. The sampling variance of the genetic correlation coefficient. Biometrics 15:469–485.
- Palmer, M. W. 1990. Spatial scale and patterns of species-environment relationships in hardwood forest of the North Carolinas piedmont. Coenoses 5:79–87.
- Schlosser, U. 1984. Species-specific sporangium autolysins (cell-wall-dissolving enzymes) in the genus *Chlamydomonas*. Pp. 409–418 in D. Irvine and D. John, eds. Systematics of the green algae. Academic Press, London.
- Schoen, D. J., G. Bell, and M. J. Lechowicz. 1994. The ecology and genetics of fitness in forest plants. IV. Quantitative genetics of fitness components in *Impatiens pallida* (Balsaminaceae). Am. J. Bot. 81:232–239.
- Simmonds, N. 1981. Genotype (G), environment (E), and GE components of crop yields. Exp. Agric. 17:355–362.
- SYSTAT. 1996. SYSTAT: statistics, Vers. 6.1. SYSTAT, Inc., Evanston, IL.
- Van Tienderen, P. H. 1997. Generalists, specialists, and the evolution of phenotypic plasticity in sympatric populations of distinct species. Evolution 51:1372–1380.
- Via, S. 1991. The genetic structure of host plant adaptation in a spatial patchwork: demographic variability among reciprocally transplanted pea aphid clones. Evolution 45:827–852.
- Via, S., and R. Lande. 1987. Evolution of genetic variability in a spatially variable environment: effects of genotype-environment interaction. Genet. Res. 49:147–156.
- Wu, R., and R. F. Stettler. 1997. Quantitative genetics of growth and development in *Populus*. II. The partitioning of genotype × environment interaction in stem growth. Heredity 78:124–134.

Corresponding Editor: E. Martins

APPENDIX

List of taxa used in this study.

Current taxon name	Former taxon name (if applicable)	UTEX number
<i>Chlamydomonas reinhardtii</i>		1010, 1952, M3-54 A ₂ *, M4-81B ₁ *
<i>C. culleus</i>	<i>C. frankii</i>	1057
<i>C. spheroides</i>		221
<i>C. debaryana</i>	<i>C. komma</i>	579
<i>C. callosa</i>		624
<i>C. moewusii</i>	<i>C. eugametos</i>	9
<i>C. mutabilis</i>		578
<i>C. applanata</i>	<i>C. humicola</i>	225
<i>C. mexicana</i>		730
<i>C. segnis</i>	<i>C. pallidostigmatica</i>	1905
<i>C. debaryana</i>	<i>C. agloëformis</i>	231
<i>Chlorogonium elongatum</i>		11
<i>Haematococcus lacustris</i>		16

* These taxa were isolated from MacDonald Campus, McGill University, Quebec.