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LIFE-CYCLE COMPONENTS OF SELECTION IN *ERIGERON ANNUUS*: II. GENETIC VARIATION

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Abstract.—Genetic variation for seedling and adult fitness components was measured under natural conditions to determine the relative importance of the seedling stage for lifetime fitness in *Erigeron annuus*. Variation in lifetime reproductive success can result from both the persistent effects of genetic variation expressed among seedlings and from variation in adult fitness components. Analysis of covariance was used to separate the stage specific from the cumulative effects of genetic variance expressed earlier in the life cycle. *E. annuus* produces seeds through apomixis, which allowed measurement of the fitness of replicate genotypes from germination through the entire life cycle. There were significant differences among genotypes for date of emergence, seedling size, survivorship and fecundity, but heritabilities were low, indicating slow response to selection. For all characters, environmental components of variance were one to two orders of magnitude larger than genetic variance components, resulting in broad sense heritabilities less than 0.1. For seedling size and fecundity, all of the genetic variance was in the form of genotype-environment interactions, often with large negative genetic correlations across environments. In contrast, genotypes differed in mean survivorship through one year, but there were no genotype-environment interactions for viability. Genetic differences in viability were primarily expressed as differences in overwinter survivorship. Genotype × environment interactions among sites and blocks were generated early in the life cycle while the genotype × environment interactions in response to competitive environment (open, annual cover, perennial cover) first appeared in adult fecundity. Genetic variation in lifetime fitness was not significant, despite a fourfold difference in mean fitness among genotypes.

Key words.—*Erigeron annuus*, fitness, genetic variation, germination date, seedling size.

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Despite the potential importance of interactions among seedlings for determining fitness, the seedling stage is often bypassed in studies of natural selection. Transplant studies using clonal ramets have yielded much information on genetic variation in plant populations (reviewed by Briggs and Walters, 1984; Endler, 1986; Primack and Kang, 1989), but necessarily ignore the establishment phase of the life cycle. Other studies have followed the demography of naturally occurring seedlings and have shown the importance of variation in emergence date and seedling size for lifetime reproductive success (e.g., Stanton, 1985; Miller, 1987; Kalisz, 1986; Stewart and Schoen, 1987) but these have not addressed the genetic basis of seedling phenotypes and thus the potential response to selection. To the extent that seedling size variation is determined by seedling density (Mithen et al., 1984; Silander and Pacala, 1985) or by the quality of a particular safe site (Harper, 1977; Hartgerink and Bazzaz, 1984; Fowler, 1988) the success of an individual may primarily result from environmental rather than genetic factors. Similarly, maternal effects may be particularly important in reducing the potential response to selection on seedling characters (Schmitt and Antonovics, 1986; Roach and Wulff, 1987; Stratton, 1989). Field estimates of genetic variance for seedling characters are often low (Antonovics and Primack, 1982; Roach, 1985; Mitchell-Olds, 1986; Schmitt and Antonovics, 1986). On the other hand, greenhouse experiments generally find substantial genetic variation for seedling size and growth rate (Schemske, 1984; Clay and Antonovics, 1985; Schwaegerle et al., 1986; Stratton, 1991*a*) indicating that the low heritabilities in the field studies result from the enormous environmental variation rather than a lack of genetic variation.

Selection can be decomposed into a series of episodes that multiplicatively combine to determine lifetime fitness. Genetic vari-

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ation in lifetime fitness will be the cumulative effect of genetic variation expressed at each stage of the life cycle. For example, genetic differences in fecundity may be primarily determined by the genetic effects on fecundity itself, or might equally be determined by genetic differences in seedling size which then indirectly affect fecundity through the correlations between sizes at successive stages. There may be little genetic correlation between juvenile and adult fitness components (Roach, 1986). If fitness differences are primarily the result of genetic variance for characters expressed late in life (e.g., adult survivorship, fecundity) then evolution may be effectively "blind" to events earlier in the life cycle. This is especially true in apomicts where the entire genotype is the object of selection. Conversely, given strong size hierarchies and high seedling mortality, genetic variation in seedling traits may dominate effects on lifetime fitness.

The goals of this experiment were twofold. I examined the ontogeny of lifetime fitness variation among genotypes, to assess the importance of genetic variation expressed at the seedling stage compared to genetic variation in adult fitness components. Second, I tested for habitat-specific variation among genotypes (G × E interactions) that might maintain genetic variation in heterogeneous selective environments. G × E was assessed at two spatial scales and with respect to three experimentally imposed disturbance treatments (corresponding to habitat variation in fields of increasing successional age).

Materials and Methods

Erigeron annuus (Asteraceae) is a common early successional weed native to eastern North America. It is considered a winter annual, but some plants delay reproduction until the second or third summer. *E. annuus* is triploid ($3N = 27$) and apomictic. Despite obligate apomixis, populations of *E. annuus* consist of numerous coexisting genotypes; on average, 7.2 multilocus genotypes can be distinguished by variation at 4 enzyme loci in samples of 18–60 plants (Stratton, 1988). Reproduction occurs solely by wind dispersed seeds and there is little seed dormancy (Stratton, 1988). The apomictic reproduction allows the use of genetically identical seeds to replicate measurements of the fitness of individual genotypes from the seedling stage through the entire life cycle. Male fitness components, which are notoriously difficult to measure, are absent in this species. The use of "clonal" seeds increases the power to detect genetic differences, which is particularly important at the seedling stage when environmental variance is large.

To assess genetic variation for fitness components, I planted seeds from 10 electrophoretically distinct genotypes of *E. annuus* into replicate 1 m^2 plots at two sites and under three experimentally imposed levels of disturbance. The two sites, Stony Brook campus (SB) and the Weld Preserve (WP) were approximately 10 km apart. The three disturbance treatments included bare tilled soil ("Open"), tilled soil with sparse cover of summer annuals ("annual"), and natural vegetation with small 10 × 10 cm disturbances ("perennial") (Stratton, 1992). The disturbance treatments were imposed prior to planting and the vegetation was allowed to develop naturally throughout the rest of the experiment. Details of the disturbance treatments and plot preparation are given in Stratton (1992); here I concentrate on the genetic details.

Seeds from 10 genotypes of *E. annuus* were collected from a single population at WP. All genotypes could be distinguished electrophoretically by variation at 4 polymorphic enzyme loci. Seeds from the 10 genotypes were sown in a common outdoor garden and two replicate plants of each were used to generate seed for this experiment. The two replicate seed families were necessary to distinguish true genetic differences from possible maternal environmental effects. Thus all of the seedlings in the two families of each genotype were genetically identical by descent from a common, field-collected, grandparent. Electrophoresis was used simply to confirm that the genotypes were distinct at at least one locus.

The 10 genotypes were planted in four cells within each plot (two maternal families × two replicates). The genotypes were randomly assigned to positions arranged in a checkerboard design within each plot. The seed bank of *Erigeron annuus* is small com-

pared to other early successional weeds, but some seeds do remain viable in the soil (Stratton, 1988). To eliminate seeds of unknown parentage, I removed a plug of soil from each 10 cm cell prior to planting and refilled the hole with autocloaved soil. Greenhouse tests showed that the sterilization treatment was effective: no seedlings emerged in control pots of autoclaved soil.

I scattered 600–900 seeds over the 10 cm cell, using a 30 cm cardboard tube to keep seeds from blowing into neighboring squares during planting. Seeds were planted between 31 July and 2 August 1987. This was just after the peak seed dispersal of natural *Erigeron annuus* (but still within the normal period of seed dispersal) and so minimized the immigration of unknown seeds into the plots. The plots were censused weekly for emergence and survivorship from 15 August to 6 September. All seedlings that emerged during a census week were defined as a germination cohort.

A subset of 2,160 of the surviving seedlings were permanently marked and monitored for seedling growth, winter and spring survivorship, and fecundity. I measured the rosette diameter (mm) and recorded survivorship at four stages of the life cycle: October 1987, March and July 1988, and July 1989. Finally, I harvested all reproductive individuals and estimated fecundity in July 1988 and July 1989. Adult size was measured as the number of stems, mean basal stem diameter (mm) and mean height (cm). Seed production was estimated by the number of heads produced times the number of florets per head (Stratton, 1992).

Viability selection was divided into five components: preestablishment (germination–September), fall (September–October), winter (October–March), spring (March–July), and year-2 (July 1988–July 1989) survivorship. Survivors of a particular selection episode were assigned a fitness value of one, otherwise fitness was zero for that episode. Lifetime fitness was defined for each individual as the product of fitnesses over each selection episode (viability and fecundity). Most estimates of lifetime fitness were zero. The fitness of plants that delayed reproduction until year 2 was discounted by the estimated population growth rate in each 1 m^2 plot (Stratton, 1992).

Data Analysis.—I tested genetic differences in viability using a log-linear categorical analysis with survivorship over the period of interest as a dependent variable (SAS, 1988, Procedure CATMOD). Parameters were estimated using the maximum likelihood option and nonsignificant high order interactions were sequentially dropped to find the simplest model without significant residual variation. Terms that were contained within a significant interaction were always kept in the model. Because the maximum likelihood method requires cell sizes of at least 20 for reliable parameter estimates (SAS, 1988), I lumped adjacent blocks within sites (SB1-2, WP3-4, WP5-6) and I did not test for differences in viability among families within genotypes (maternal environmental effects). Maternal effects were tested for rosette diameter and fecundity and were never significant (below). Log-linear categorical models and logistic regressions were also used to test for variation among genotypes in germination date and the timing of reproduction. Log-linear models are the appropriate tests for frequency data and have much more power than analysis of variance on proportions (Caswell, 1986). However, they do not yield estimates of variance components and thus can not be directly related to equations for evolutionary change. When differences are significant, relative viabilities can be used to predict the outcome of selection among these 10 genotypes. However, I also calculated (approximate) variance components using SAS (1988) Procedure VARCOMP (REML option) to allow direct comparisons of viability and fecundity selection episodes.

Genetic variation for seedling size and fecundity was tested with analyses of variance and covariance (SAS, 1988, Procedure GLM). Measurements of October and March rosette diameter within each 10 cm cell were not independent, so I reduced the data set to one observation per cell using the mean value over all plants within each cell. For ANCOVA, cell means were calculated separately for each germination cohort. Fecundity and fitness showed no dependence within cells (F tests; $P > 0.50$) so all observations were included. The disturbance treatments were considered fixed effects when calculating expected mean

TABLE 1. Log-linear analysis of genetic and environmental effects on emergence date. The chi-square values show tests for dependence of emergence cohort ($n = 4$) on each effect. Thus the degrees of freedom are those of the interaction between each effect and emergence cohort. Higher order interactions were not significant and were deleted to yield the simplest model that adequately fits the data.

Source	*df*	Chi-square
Site	3	121.42***
Disturbance	6	41.76***
Site•Disturbance	6	59.22***
Genotype	27	112.79***
Site•Genotype	27	44.50*
Residual	108	104.47

* $P < 0.05$; *** $P < 0.001$.

squares; all other effects were considered random. High mortality over the course of the experiment resulted in an unbalanced design, so I used type III sums of squares throughout. Nonsignificant interactions were pooled with the error (following rules given by Sokal and Rohlf, 1981) by removing the terms from the model and recomputing mean squares. Rosette diameter and fecundity were log-transformed prior to analysis, which resulted in a normal distribution of residuals. Lifetime fitness could not be transformed to normality. I computed restricted maximum likelihood estimates of variance components using SAS (1988) procedure VARCOMP, considering disturbance treatments to be fixed effects. Broad sense heritabilities are given by the proportion of total variance explained by the genetic main effect. Note that when cell means are analyzed, the within-cell variance must be added back in to estimates of the total phenotypic variance (Becker, 1985). Standard errors for variance components were not calculated since they are unknown for highly unbalanced designs (Mitchell-Olds and Rutledge, 1986), but significance levels from the ANOVAS show differences from zero.

Two aspects of genetic variance are of interest: the total genetic variance for each character and the stage-specific components of variance (genetic variation expressed during each selection episode). The cumulative genetic variation in fecundity may partially result from the continued effects of size variation generated early in the life cycle. It is the cumulative genetic variation that governs the response to selection, but stage-specific contributions describe when the genetic variation is expressed. To assess the stage-specific genetic components of seedling size variation, I used a series of analyses of covariance corresponding to the sequential stages of the life cycle. Stage-specific tests of October rosette diameter control variation in germination date; stage-specific tests for genetic variation in March rosette diameter include both germination date and October diameter as covariates; and stage-specific contributions to fecundity are tested with all three previous characters used as covariates. These tests measure the incremental growth between censuses and may be considered tests of growth rate rather than tests of size per se. A similar approach using sequential analyses of covariance has been used by Jordan (1986) and Kalisz (1989).

Genetic correlations (broad sense) were computed as the correlation of genotype means for characters that showed significant genetic variance. This has the advantage of having well-known significance tests, but the power is low since the correlations are based on a sample size of 10. Genetic correlations less than ± 0.58 could not be distinguished from zero.

RESULTS

Time to Emergence.—Variation in emergence date showed highly significant effects of both environment and genotype (Table 1). Most seedlings emerged in the first cohort, two weeks after planting, with another emergence peak in cohort 3. Relatively more seedlings emerged in cohort 1 at the Weld Preserve than at Stony Brook (67% vs 54%) which resulted in a difference in mean emergence time between sites of about two days. Genetic differences were of about the same magnitude as the between site variation (Table 1). The difference in mean time to emergence between the extreme genotypes averaged 3.2 days. In addition there was a smaller, but still significant ($P < 0.02$), genotype-site interaction for emergence date. The (approximate) genetic variance for emergence time was 0.018 compared to 0.0004 for the genotype × site interaction. Genotypes did not differ in mean emergence

TABLE 2. Genetic and environmental effects on survivorship over 5 viability selection episodes. Shown are chi-square values from the maximum likelihood log-linear analysis. There were no significant genotype-environment interactions for viability. Pairs of adjacent blocks (SB1–2, WP3–4, WP5–6) were lumped to maintain sufficient sample sizes for the maximum likelihood analysis. The residual term tests the significance of higher order interactions (G × E) and can be used as a measure of lack of fit for the entire model.

		Viability selection episode						
Source	*df*	Establishment $N = 6,251$	Fall 2,344	Winter 1,605	Spring 678	Year-1 3,087	Year-2 345	Lifetime 3,028
Block	2	161.99***	202.61***	32.55***	2.18	77.09***	4.33	102.1***
Disturbance	2	46.35***	31.49***	24.44***	1.31	26.81***	3.69	28.9***
Block• Disturbance	4	33.41***	72.90***	123.68***	17.51**	51.20***	4.96	42.1***
Genotype	9	15.20 (0.08)	9.19	19.22*	9.09	17.32*	11.92	14.5 (0.10)
Residual	72	67.30	83.32	77.25	75.57	61.40	68.38	69.00

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

time in the three disturbance treatments. The genetic variance was nearly two orders of magnitude smaller than the environmental variance components, yielding a (broad-sense) heritability of emergence date of only 0.03.

Viability.—Patterns of preestablishment mortality among genotypes showed a complex interaction between genotype, emergence date, and site. Overall, there was a marginally significant ($P = 0.08$) dependence of viability during establishment on genotype (Table 2) but when emergence date was also included, no simple interaction between genotype, environment, and survivorship adequately fit the data (not shown). In cohort 1 there was a significant genotype × disturbance treatment interaction ($\chi^2 = 30.12$, $P < 0.02$) as well as an interaction between genotype and blocks ($\chi^2 = 43.95$, $P < 0.0001$). No genetic effects on viability during establishment were significant for cohorts 2–4. As a result, there was no genetic correlation between emergence date and early survivorship within plots (mean $r_g = 0.01$). Fall survivorship (September to October) showed no dependence on genotype or on genotype-environment interactions, nor were there genetic effects on spring or year-2 survivorship (Table 2).

In contrast, the 10 genotypes differed significantly in survivorship over the winter (Table 2). Mean winter survivorship ranged from 0.45 to 0.62 among genotypes. The winter viability differences were consistent across sites and disturbance treatments (there was no significant G × E). Although winter survivorship was highly dependent on October rosette diameter (logistic regression; $\chi^2 = 164.7$, $P < 0.0001$), the genetic variation in viability remained significant when October size and emergence date were included in the model ($\chi^2 = 33.69$, $P < 0.0001$).

The cumulative survivorship through two years showed no significant difference among genotypes. Genetic variation in viability was present through the first year, yielding 50% differences in mean one-year survivorship among genotypes (Table 2, range = 0.16–0.24). However the nonsignificant trend in second-year survivorship among genotypes was opposite to that seen in year one. The overall rank correlation between mean first and second-year survivorship of the 10 genotypes was -0.55 ($P < 0.1$), so there was only a 26% difference in survivorship among extreme genotypes after two years (range = 0.15–0.19).

Seedling Size.—For both October and March rosette diameter, environmental components of variation were one to two orders of magnitude greater than the genetic variance components. Most of the environmental variation occurred on a small scale, within the 1 m^2 plots (error) and among blocks within sites (Table 3). Maternal environmental effects (families within genotypes) were not significant for either size or fecundity.

There were no significant differences among genotypes in mean seedling size, but significant genotype × environmental interactions (G × E) for rosette diameter were present in both October and March (Table 3). In October, most of the G × E variance

TABLE 3. Analysis of variance for seedling rosette diameter and seed production, testing patterns of cumulative variation in each character. Variance components are maximum likelihood estimates from a separate analysis (SAS, 1988, Procedure VARCOMP). Disturbance was considered a fixed effect. Adjusted fecundity has been corrected for the estimated population growth rate in each plot. Higher order G × E were never significant and were pooled with the error. The total variance includes the variance within cells. Terms in the denominator mean square used for each F test are given in Table 4.

Source	*df*	October rosette diameter		March rosette diameter	
		MS	Variance	*MS*	Variance
Site	1	5.223	0	0.840	0
Block(Site)	4	20.264***	0.182	1.826***	0
Disturbance	2	33.150***	—	12.230***	—
Site•Disturbance	2	8.409*	0.083	2.272	0.073
Block(Site)•Disturbance	8	1.111**	0.027	1.898***	0.097
Genotype	9	0.335	0	0.179	0
Genotype•Site	9	0.363	0	0.626*	0.003
Genotype•Disturbance	18	0.325	0.003	0.289	0
Genotype•Block(Site)	36	0.460**	0.017	0.266	0
Family(Genotype)	10	0.261	0	0.281	0
Error		0.270	0.270	0.302	0.298
Error *df*		500		322	
Model	99	2.120***		0.804***	
R-square		0.32		0.29	
Total variance			0.797		0.665

Source	*df*	Seed production		Fitness	
		MS	Variance	*MS*	Variance
Site	1	25.292	0.594	57.036	0
Block(Site)	4	9.881***	0.392	342.265***	0.688
Disturbance	2	2.451*	—	306.016***	—
Site•Disturbance	2	2.150	0.154	131.443*	0.272
Block(Site)•Disturbance	8	0.839	0.031	29.058*	0.165
Genotype	9	1.902	0.020	9.527	0
Genotype•Site	9	0.691	0	11.084	0.006
Genotype•Disturbance	18	0.953*	0.019	8.924	0
Genotype•Block(Site)	36	0.761*	0.057	9.233	0
Family(Genotype)	10	0.309[a]	0.003	7.546	0
Error		0.526	0.520	12.165	12.057
Error *df*		339		2,046	
Model	98	3.995**		31.860***	
R-square		0.69		0.11	
Total variance			1.790		13.188

[a] Pooled with error mean square.
* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

occurred at a small spatial scale, among blocks within sites (Table 3). When variation in emergence time was controlled (Table 4), there was also a significant genotype × site interaction for stage-specific fall growth. Reaction norms for mean October and March rosette diameter across sites show large reversals in the rank order of seedling size. Genotypes with the largest mean October rosette diameter at Stony Brook were among the smallest at the Weld Preserve and vice versa (Fig. 1). This resulted in a negative genetic correlation across sites for October rosette diameter ($r = -0.28$; ns). The mean genetic correlation among blocks was also negative (Table 5).

March rosette diameter showed a pattern of genetic variation similar to that in October, with a large component due to the genotype-environment interaction and no average differences among genotypes (Table 3). In March most of the G × E occurred among sites rather than within and the negative genetic correlation among sites was much larger (Fig. 2, Table 5). However, no stage-specific genetic effects were observed

TABLE 4. Analysis of covariance for seedling rosette diameter and seed production, testing stage-specific genetic and environmental effects. Variance components are separate maximum likelihood estimates using the residuals from regressions on the covariates. The total variance includes the phenotypic variance within each cell. Abbreviations for terms in the denominator mean square correspond to the first letter of each effect.

Source	*df*	October rosette diameter		March rosette diameter	
		MS	Variance	*MS*	Variance
Emergence date	1	216.957***	—	2.515***	—
October diameter	1	—	—	51.299***	—
Site	1	0.765	0	9.432	0.009
Block(Site)	4	12.025***	0.057	4.629*	0.034
Disturbance	2	26.138**	—	1.353	—
Site•Disturbance	2	2.672	0	0.579	0
Block(Site)•Disturbance	8	1.629	0.024	1.496***	0.047
Genotype	9	0.235	0	0.146	0
Genotype•Site	9	0.919*	0.005	0.199	0
Genotype•Disturbance	18	0.195	0	0.250	0.004
Genotype•Block(Site)	36	0.348(*)	0	0.159	0.001
Family(Genotype)	10	0.329	0.002	0.218	0.002
Error		0.246	0.247	0.174	0.188
Error *df*		936		504	
Model	98	4.249***		1.978***	
R-square		0.65		0.69	
Total variance			0.401		0.350

Source	*df*	Seed production		Terms in denominator mean square
		MS	Variance	
Emergence Date	1	0.595	—	error
October Diameter	1	2.008*	—	error
March Diameter	1	6.095***	—	error
Site	1	32.455	1.009	B(S), G*B(S), S*G
Block(Site)	4	7.498***	0.377	G*B(S)
Disturbance	2	1.460*	—	error
Site•Disturbance	2	2.592	0	B(S)*D
Block(Site)•Disturbance	8	0.601	0.017	error
Genotype	9	1.482	0.023	S*G, G*B(S), F(G)
Genotype•Site	9	0.465	0	G*B(S)
Genotype•Disturbance	18	0.814*	0.022	error
Genotype•Block(Site)	36	0.573	0.069	error
Family(Genotype)	10	0.499	0	error
Error		0.441	0.513	
Error *df*		301		
Model	100	4.025***		
R-square		0.75		
Total variance			2.030	

(*) $P < 0.07$; * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

for rosette growth between October and March (Table 4). The significant environmental effects during this interval show the presence of nonrandom phenotypic variation in winter growth (they were not merely dormant), but neither the genetic main effect nor the G × E interactions for growth between October and March were significantly different from zero. Rather, the (cumulative) genotype × site interaction for March rosette diameter (Table 3) reflects the continuing expression of differences that arose during the fall.

Seed Production and Lifetime Fitness.— There was large genetic variation in the timing of reproduction (Table 6): among plants that survived the first year, the proportion that reproduced as annuals ranged from 0.46 to 0.80 among genotypes. The environmental variation was large, so the heritability of flowering time was only 3%. The probability of a plant flowering during the first year was

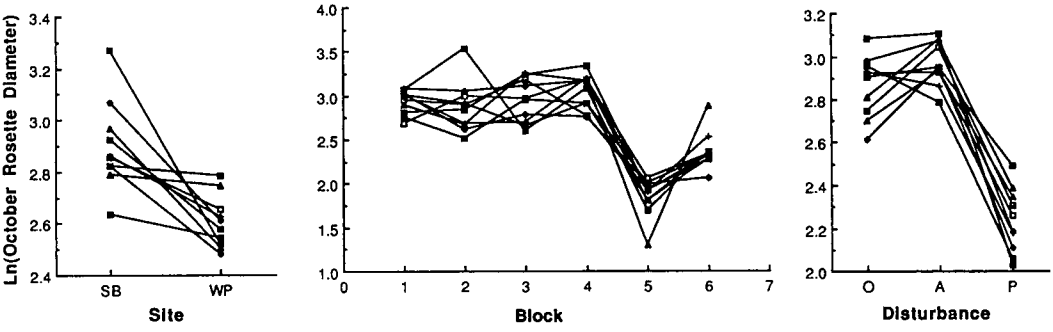

FIG. 1. Norms of reaction for October rosette diameter across sites, blocks, and disturbance treatments. Lines connect genotype means expressed in each environment.

highly dependent on March rosette diameter (logistic regression, $\chi^2 = 109.2$, $P < 0.0001$) but the genetic differences in the probability of flowering remained (and statistical significance increased) when seedling size was included ($\chi^2 = 29.03$, $P < 0.001$).

The cumulative genetic variation in seed production was again dominated by significant genotype-environment interactions (Table 3). There was still a significant spatial component to the G × E, primarily among blocks within sites (over a scale of 10–50 m, Fig. 3), as was observed for seedling size. The mean genetic correlation between October diameter and fecundity (within plots) was 0.40. In addition, there was significant variation among genotypes in fecundity with respect to the competitive environment (Genotype × Disturbance, Table 3). The genotype × disturbance interaction resulted entirely from stage-specific variation expressed in adults (Table 4), since the genotype × disturbance interaction was not present in earlier episodes. The significant genotype × environment interactions show that the genetic correlation across the disturbance treatments was significantly less than 1.0, but the mean genetic correlation among disturbance treatments was $r_g = 0.29$ and all pairwise genetic correlations were positive (0.12, 0.34, 0.40 for comparisons O-A, O-P, A-P). Thus the genotypes differed in their relative fitness among the three treatments, but there were not large reversals in the rank order of fecundity. The mean genetic correlation among all plots was near zero (Table 5). The estimate of genetic variance for fecundity was nearly as large as the interaction variance (Tables 3 and 4), although the genetic main effect was not significant when tested over the appropriate interactions. There was more than a twofold difference in average fecundity among genotypes (range 16.3×10^3 to 37.8×10^3). Again, the environmental components of variation were much larger than the genetic effects, however.

There was no significant genetic variation or G & E for lifetime fitness (Table 3), despite the fourfold difference in genotype means (Fig. 4, squares). Relative lifetime fitnesses ranged from 0.40 to 1.73. Significance levels must be considered only ap-

TABLE 5. Genetic correlations across environments. Shown are the mean pairwise correlations of genotype means for the same character expressed in different environments. Large negative correlations imply reversals in the rank order of genotypes in different habitats. The number of pairwise comparisons included in each mean is $N(N-1)/2$.

	Mean genetic correlation among:			
	Sites $N = 2$	Blocks 6	Disturbance 3	All plots 18
October diameter	−0.28	−0.08	−0.03	0.004
March diameter	−0.74	−0.10	−0.20	−0.050
Fecundity	0.51	0.06	0.29	0.0001

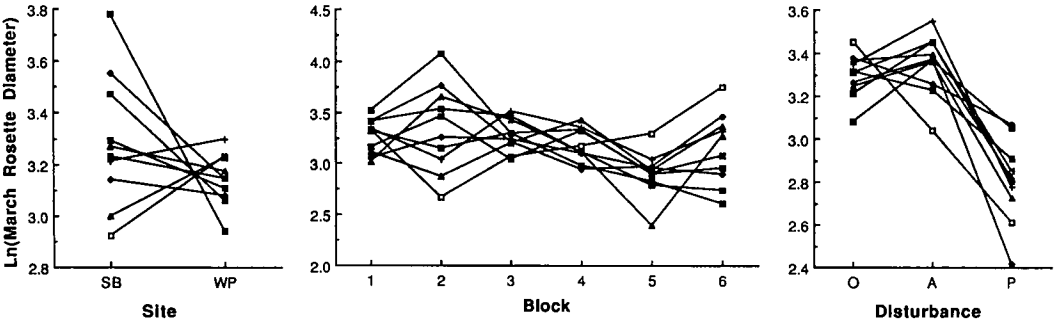

FIG. 2. Norms of reaction for March rosette diameter across sites, blocks, and disturbance treatments. Lines connect genotype means expressed in each environment.

proximate, because the normality and homoscedasticity assumptions of ANOVA were violated, but nonparametric Kruskall-Wallis tests within plots (SAS 1988, PROC NPAR1WAY) also yielded no significant differences among genotypes (not shown).

A simple measure of the degree of habitat specialization among genotypes is the variance in fitness across environments (Fig. 5). Genotypes with high variance in relative fitness may be considered specialists, having high fitness in only a limited subset of environments. Those with low variance in fitness are relatively buffered against changes in the environment and follow a generalist strategy. This measure of specialization is very similar to stability parameters developed for crop species (Shukla, 1972; Lin et al., 1986). The variance in relative fitness was significantly heterogeneous among the 10 genotypes ($F_{max} = 36.8$; $P < 0.01$). For example, genotype 5 had the highest mean fitness but only moderate variance in fitness across environments, showing that it was a well-adapted, generalist genotype (Fig. 5).

TABLE 6. Genetic variation in the timing of reproduction (annual or biennial strategy). The maximum likelihood log-linear analysis tests for independence between genetic or environmental factors, and whether or not the plant reproduced during year 1. There were no significant G × E.

Source	*df*	Chi-square
Site	1	91.42***
Disturbance	2	77.39***
Genotype	9	26.07**
Residual	43	49.99

*** $P < 0.001$; ** $P < 0.01$.

In contrast, genotype 10 had extremely high variation in relative fitness across plots, showing a large sensitivity to habitat variation, although there was no obvious pattern with respect to disturbance treatment, site, or other environmental factors. Others, such as genotype 3, had uniformly low fitness. The heterogeneity of variance in relative fitness appears robust, as it remains significant even if genotypes with the highest and lowest variances are deleted.

DISCUSSION

The observed genetic variation for the measured fitness components was small relative to environmental variation and may not have been detectable in a conventional experiment using half sibs. The use of replicate genotypes allowed detection of genetic variation among seedlings as being significantly greater than zero, but the broad-sense heritabilities are quite low, usually less than 5%. Low levels of genetic variation are expected for major components of fitness, which are by definition under strong directional selection (Gustaffson, 1986; Mousseau and Roff, 1987). Other experiments have shown that genetic variation is present among these *Erigeron* genotypes. Broad-sense heritabilities for the same characters measured in the greenhouse range from 0.2 to 0.5 (Stratton, 1991), well within the range observed for sexual species.

Greenhouse and field estimates of genetic variance are much more similar when scaled to the mean instead of the total variance. The average genetic coefficient of variation (CV_g = square root genetic variance/mean) for October rosette diameter within plots

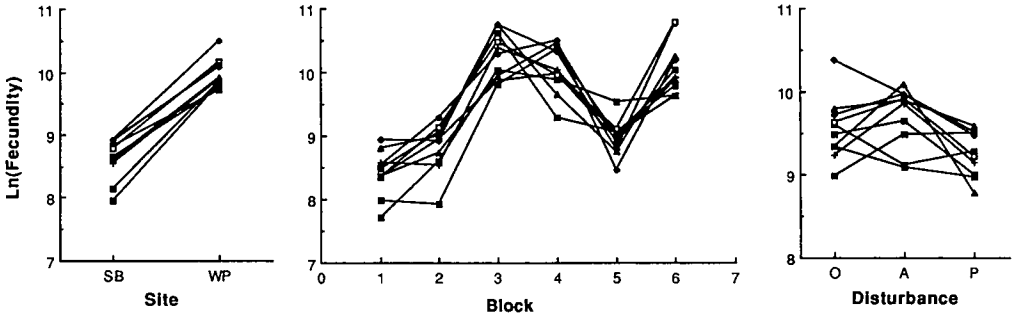

FIG. 3. Norms of reaction for fecundity across sites, blocks, and disturbance treatments. Lines connect genotype means expressed in each environment.

(5.2%) was similar to that for two-month rosette diameter in the greenhouse (6.5%, Stratton, 1991*a*). Note that the response to selection on fitness itself, or a multiplicative component of fitness, depends only on the genetic variance, not the heritability (Fisher, 1930; Arnold and Wade, 1984, eq. 20), so the coefficient of variation is a useful measure of genetic variation in fitness components (D. Houle, pers. comm). The genetic coefficients of variation (within plots) for fecundity and winter survivorship were 3.2% and 37.5%, respectively. Thus the low heritabilities in the field are not due to a lack of genetic variation, but rather reflect the extremely large environmental variance components.

These estimates of genetic variation and genotype-environment interactions are broad-sense measures of genetic variance. They include an unknown mixture of additive, dominance and epistatic variance components. However, they are free of contamination by maternal environmental effects, because multiple seed families were used. Moreover, broad sense measures are the appropriate variance components to use for predicting the response to selection in obligately asexual species, because the entire genotype is transmitted from parents to offspring.

Although differences in lifetime fitness among genotypes were not significant, that does not mean fitnesses were truly equal. Standard errors were large, despite large sample sizes, as a result of the many plants with zero fitness. In general, correlations among fitness components were not negative, so differences in fitness increased over time. The genotype with the highest two-

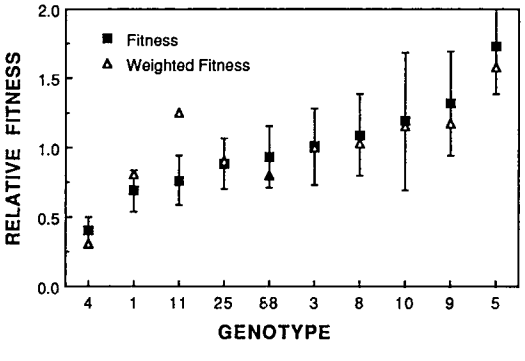

FIG. 4. Relative lifetime fitness (±SE) for 10 genotypes of *Erigeron annuus*. Fitnesses are relative to the overall mean fitness in the entire experiment. Squares show the experimental estimates of relative fitness; triangles show relative fitness weighted by the frequency distribution of environments in nearby *Erigeron* populations (see text).

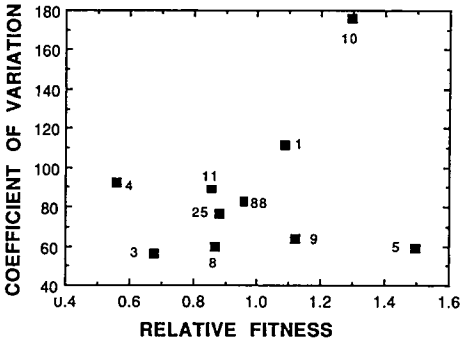

FIG. 5. Coefficient of variation for lifetime relative fitness across 18 plots versus mean relative fitness. Relative fitnesses were computed separately for each plot. Genotypes with high coefficients of variation for fitness across environments may be considered specialists.

year survivorship had a mean viability 1.26× higher than that of the genotype with the lowest mean survivorship. There was a 2.3× range in mean fecundity among genotypes and a 4.3× range of mean lifetime fitness among the 10 genotypes. However, the variance also increased with time and the test for variation in lifetime fitness had little power. Significant variation in major components of fitness (seed production, 1-year survival) and the fourfold variation in relative fitness, suggest that the fitness differences may be real. The lack of significant genetic variation for fitness was not simply the result of discounting the fecundity of second-year plants. Similar results are obtained if unweighted seed production is used (analysis not shown).

Genetic effects were most important during the first year, and there was significant variation in emergence date, survivorship, growth, and fecundity over that interval. These genotypes differ in seed size (Stratton, 1991) but there were no significant genetic correlations between mean seed size and mean performance in the field. After one year of growth, no genetic effects could be detected for survivorship or fecundity of the second year plants. This may be partly a result of smaller sample sizes during the second year. Nevertheless, there was a clear fitness advantage for early reproduction: the mean fitness of plants that reproduced in year 1 was 6.2 times higher than the fitness of plants flowering in year 2 (10,938 versus 1,755). It is thus important to measure lifetime fitness of the entire cohort. Had the experiment stopped after a single growing season, the genetic variance for survivorship and fecundity would have been overestimated. Instead, random variation (independent of genotypes) in the viability and fecundity of second year plants reduced the correspondence between genotype and fitness. Plants that delayed reproduction tended to be the smaller plants, in the later cohorts that would have had low expected fecundity during year 1 (Stratton, 1992). Delayed reproduction reduced the intensity of selection against those genotypes.

Genotype-Environment Interactions and Maintenance of Genetic Variation.—A central question for ecological genetics is the role of spatial heterogeneity and genotype-environment interactions for the maintenance of genetic variation (Felsenstein, 1976; Hedrick et al., 1976; Slatkin, 1978; Via and Lande, 1985; Gillespie and Turelli, 1989). Single locus models have shown spatial variation in fitness to be ineffective for the maintenance of variation in haploid or asexual organisms (Gliddon and Stroebeck, 1975) unless there is a precise matching of relative fitnesses. For polygenic characters, Via and Lande (1985) concluded that G × E will seldom maintain additive variance, because in the long run the population will achieve the optimum phenotype. In their two-environment models, G × E will maintain genetic variation only when the genetic correlation across environments is ±1.0. Gillespie and Turelli (1989) have shown that environmental heterogeneity is a more effective force maintaining variation in a model that assumes heterozygotes have reduced variance (and hence remain closer to the optimum phenotype). Heterozygote superiority is ineffective in apomicts, however. In all cases, it is clear that negative correlations across environments increase the effectiveness of G × E for the maintenance of variation. For G × E to be effective at maintaining variation, the rank order of fitness must be reversed across environments such that the differences in relative fitness in each environment cancel. In general, the response to simultaneous selection in multiple environments will cease if the mean genetic correlation across environments is less than $-1/(n-1)$ (Dickerson, 1955; Via and Lande, 1985).

The genotype × environment interactions for October and March seedling rosette diameter showed the appropriate reversals in rank across sites. Genotypes that were largest at SB were smallest at WP and vice versa. However, none of the correlations across environments were sufficiently negative to halt the response to selection (Via and Lande, 1985). There were many fewer reversals in rank among genotypes for characters more closely related to fitness. With respect to viability, genotype × environment interactions were not observed during any viability selection episode. G × E among disturbance treatments and blocks were present for fecundity, but the genetic correlations across competitive environ-

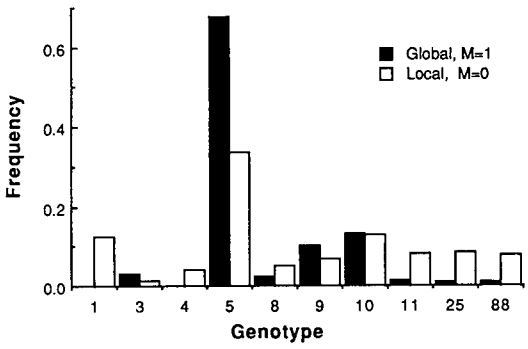

FIG. 6. Projected changes in genotype frequencies after six generations given the measurements of lifetime fitness from this experiment. All genotypes had equal initial frequencies (0.10) at generation 0. Solid bars: Population size held constant over the entire populations (global density regulation) and complete mixing of seeds every generation (m = 1). Open bars: Population size constant for each 1 m^2 plot (local density regulation) and no migration between plots (m = 0).

ments were positive. Miller and Schemske (1990) also found positive genetic correlations among competitive treatments in *Brassica,* despite significant G × E. Characters that increase fecundity in one environment tend to increase fecundity in others as well. Thus, negative correlations across environments were not observed for fitness components and genotype-environment interactions are probably not sufficient for maintaining genetic variation in *E. annuus.*

Changes in genotype frequencies may in fact be quite rapid, given the estimated lifetime fitnesses. Population projections show that genotype 5 may reach 68% abundance within six generations (Fig. 6, solid bars). Changes of this magnitude would be important even in the short lived early-successional communities occupied by *E. annuus.* However, the predicted dynamics depend critically on assumptions of gene flow and population regulation, for which no data are currently available. For example, genotype 1 would rapidly go extinct if the environment varies on a fine scale relative to seed dispersal and density regulation (Fig. 6, solid bars), but ranks third in abundance if plots evolve independently (Fig. 6, open bars).

In the presence of G × E, relative fitnesses depend critically on the frequency of each environment in nature (Lewontin, 1974; Gupta and Lewontin, 1982). This has long been appreciated for single locus models (e.g., Maynard Smith and Hoekstra, 1980) but it is rarely addressed in studies of quantitative genetic variation. Experiments that estimate G × E are necessarily confined to only a tiny fraction of the possible environments into which a seed may fall. For maximum statistical power, equal numbers of plots are usually put in each major habitat. However, predictive statements about the outcome of selection must be made with reference to the actual frequency of environments in the population of interest. This is almost never known. As an ad hoc estimate of the relative frequency of environments, I used data from a vegetation analysis of nearby *Erigeron* populations (unpubl. data). Each of 160 0.25 m^2 quadrats was classified as one of the six site/disturbance combinations based on the percent open ground and the relative percent cover of co-occurring species. The classification must be considered extremely approximate. When fitnesses are weighted by the approximate frequency of environments, the relative fitness of genotype 11 increases dramatically (Fig. 4, triangles). Other genotypes show little change in rank, indicating that the 18 plots are a reasonably adequate sample of environments. In general, however, there may be little correspondence between weighted and unweighted fitness estimates, especially when fitness is measured in only a few plots.

The chance distribution of clones among early colonists may have a larger effect on genetic diversity within populations than the intrinsic adaptedness of the genotypes present. For all characters measured, the environmental components of variance were at least an order of magnitude larger than the genetic variance components. Plastic variation in seedling size and fecundity in response to variation in microsites was by far the most important determinant of individual reproductive success. The magnitude of phenotypic selection on seedlings varied on a local scale in this experiment (Stratton, 1992), indicating local environmental heterogeneity. Phenotypic plasticity (e.g., the ability to increase fecundity in response to a favorable environment) represents an al-

ternative to genetic specialization and may be favored when the environment varies locally and unpredictably in space and time (Bradshaw, 1965). Large spatial and temporal fluctuations in the environment are especially common in the early successional communities occupied by *E. annuus* (Bazzaz, 1979).

Components of Selection.—Significant genetic variation was detected for the seedling characters emergence date and October rosette diameter, both of which affect phenotypic variation in lifetime fitness (Stratton, 1992). As a result, some of the genetic differences in major fitness components (viability, fecundity) are determined very early in the life cycle. Mortality is concentrated in the early stages of the life cycle: 42% of all seedlings die during the first month after emergence (Stratton, 1992). But the genetic effects on survivorship during establishment were small compared to genetic variation in winter survivorship. There was little genetic correlation between emergence date and survivorship, despite strong phenotypic effects. So, although mortality during establishment is demographically important, it is a minor component of genetic variation in fitness. Similarly, phenotypic variation in winter survivorship was strongly dependent on October rosette diameter (Stratton, 1992), but the pattern of genetic variation in winter survivorship (average differences, no G × E) was very different from the pattern of genetic variation in size (large G × E, no main effect). Thus much of the genetic variation in winter survivorship was not determined by genetic effects operating through seedling size. Genetic variation in seedling size did determine much of the genetic variation in fecundity, however. The spatial variation in relative size (G × E among blocks) first appeared in October rosette diameter and persisted throughout the life cycle. Using the square of the genetic correlation as a measure of determination, October rosette diameter determined 16% of the genetic variation in fecundity. G × E across disturbance treatments appeared only in adults, so stage specific genetic variation was also important and accounted for the bulk of the fecundity variation.

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