

Parental Effects in *Plantago Lanceolata* L. III. Measuring Parental Temperature Effects in the Field

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Abstract:

To determine the evolutionary importance of parental environmental effects in natural populations, we must begin to measure the magnitude of these effects in the field. For this reason, we conducted a combined growth chamber–field experiment to measure parental temperature effects in *Plantago lanceolata*. We grew in the field offspring of controlled crosses of chamber-grown parents subjected to six temperature treatments. Each treatment was characterized by a unique combination of maternal prezygotic (prior to fertilization), paternal prezygotic, and postzygotic (during fertilization and seed set) temperatures. Offspring were followed for three years to measure the effects of treatment on several life-history traits and population growth rate, our estimate of fitness.

Parental treatment influenced germination, growth, and reproduction of newborns, but not survival or reproduction of offspring at least one year old. High postzygotic temperature significantly increased germination and leaf area at 17 weeks by approximately 35% and 2%, respectively. Probability of flowering and spike production in the newborn age class showed significant parental genotype \times parental treatment interactions. High postzygotic temperature increased offspring fitness by approximately 50%. The strongest contributors to fitness were germination and probability of flowering and spike production of newborns. A comparison of our data with previously collected data for chamber-grown offspring shows that the influence of parental environment on offspring phenotype is weaker but still biologically meaningful in the field.

The results provide evidence that parental environment influences offspring fitness in natural populations of *P. lanceolata* and does so by affecting the life-history traits most strongly contributing to fitness. The data suggest that from the perspective of offspring fitness, natural selection favors parents that flower later in the flowering season in the North Carolina Piedmont when it is warmer. Genotypic-specific differences in response of offspring reproductive traits to parental environment suggest that parental environmental effects can influence the rate of evolutionary change in *P. lanceolata*.

Key words: Fitness, flowering phenology, germination, life-history evolution, maternal effects, parental effects, *Plantago lanceolata*, temperature.

Article:

Many studies provide empirical evidence that parental environment and sometimes also grandparental environment can influence an individual's phenotype. In plants, ancestral environments can influence an individual's seed size, germination, growth, flowering time, or male sterility (e.g., see reviews by Rowe 1964; Schaal 1984; Roach and Wulff 1987; Gutterman 1992; Wulff 1995; Donohue and Schmitt 1998; Rossiter 1998). Also, offspring responses to ancestral environments are genetically based (e.g., Schneeberger and Cullis 1991; Wulff and Bazzaz 1992; Platenkamp and Shaw 1993; Schmid and Dolt 1994; Wulff et al. 1994; Case et al. 1996; Sultan 1996; Byers et al. 1997; Mazer and Wolfe 1998). Because these parental environmental effects influence the phenotypic expression of traits contributing to individual fitness, these effects may influence the

evolution of natural populations. They may alter the direction and rate of natural evolutionary change (e.g., Schaal 1984; Riska et al. 1985; Antonovics and Schmitt 1986; Roach and Wulff 1987; Kirkpatrick and Lande 1989; Schmitt et al. 1992; Lacey 1996; Mousseau and Fox 1998).

The evolutionary importance of parental environmental effects is still questioned, however, because empirical support is limited. Whereas many studies of parental effects have been conducted in controlled environments, few have measured parental effects in field experiments (for plants: Biere 1991; Schmitt et al. 1992; Galloway 1995; Donohue and Schmitt 1998; Thiede 1998). Therefore, there is little evidence that parental effects observed under controlled conditions are expressed in natural populations. Also, only one field study (Donohue and Schmitt 1998) has attempted to measure the impact of parental environment on offspring fitness. Until we establish that the parental environment influences offspring phenotype and fitness in natural habitats, the evolutionary importance of these effects will continue to be debated.

For this reason we conducted a combined growth chamber—field experiment using *Plantago lanceolata* L. We sought to answer the following questions. Does parental environment affect offspring life-history traits and fitness when offspring are grown in field conditions? Are the offspring traits most affected by parental environment those that most influence offspring fitness in the field? When during the parental generation does parental environment most influence field-grown offspring? Which parent transmits the parental effects? What do field-collected data suggest about the role of parental temperature effects in the evolution of life-history patterns in *P. lanceolata*?

Our experiment complements Lacey's (1996) study, which measured the strength of prezygotic (i.e., prior to anthesis) and postzygotic (i.e., during anthesis and offspring seed maturation on the maternal parent) temperature effects on off-spring phenotype in *P. lanceolata*. Lacey observed that parental environment significantly influenced offspring germination, growth, and onset of flowering when parental and offspring generations were grown in growth chambers. We grew offspring from the same crosses and temperature treatments used by Lacey (1996) and asked how parental temperature treatment would influence these same traits when offspring are grown in field plots. Also, we determined whether parental temperature affected within-season flowering phenology, year of first flowering, or both in field-grown offspring. These components of onset of flowering could not be separately measured in the growth-chamber experiment.

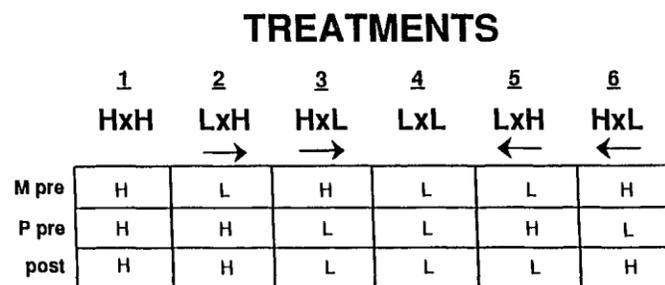


FIG. 1. Experimental design describing the six temperature treatments by maternal prezygotic temperature (M pre), paternal prezygotic temperature (P pre), and postzygotic temperature (post) for *Plantago lanceolata*. The arrow indicates the direction of transfer between temperatures (i.e., chambers) to effect fertilization and seed set. L, low temperature; H, high temperature. See Materials and Methods for additional explanation.

To estimate fitness, we collected data on age-specific survivorship and fecundity over three years, by the end of which time most offspring had died. Using a matrix model (Caswell 1989), we estimated population growth rate, our measure of fitness, and examined the differences in population growth rate as a function of parental temperature treatment. Then we determined which life-history traits contributed most to population growth rate.

MATERIALS AND METHODS

Study Species

Plantago lanceolata L. (Plantaginaceae), ribwort plantain, is a weedy, temperate, herbaceous perennial introduced into North America from Europe. It grows vegetatively as a rosette and produces small flowers on spikes that arise from axillary buds. Flowering phenology within seasons is both genetically and environmentally determined (e.g., Primack and Antonovics 1981; Wolff 1987; Wolff and van Deldon 1987; Lacey 1996) The species is protogynous, gynodioecious, and self-incompatible (Ross 1973; van Damme 1984).

Plantago lanceolata typically colonizes abandoned crop fields and disturbed places where plant competition for resources is relatively low. It also grows well in mown lawns and persists in hayfields, where competition for space and light can be fierce. The field portion of our experiment mimicked an abandoned crop field.

Experimental Design

Details of the selection, growing conditions, crossing design, and temperature treatments for the parental generation are given in Lacey (1996). Here we summarize features relevant to this experiment. In 1988, five plants were collected from each of two populations (abbreviated D and H) of *P. lanceolata* in Durham, North Carolina. Two populations were used instead of one to increase the genetic diversity of individuals used in the experiment and to eliminate the problem of unknowingly trying to mate relatives. We collected only plants growing more than 1 m apart, and we assumed that all plants were distinct genotypes. We cloned each genotype to produce multiple copies. Cloned individuals of each genotype were transferred at the two- to five-leaf stage to each of two growth chambers in 1989.

The two chambers differed in their temperature regime: either 15°C nights/20°C days or 20°C nights/26°C days. These temperatures approximate the mean monthly temperatures for May and July, respectively, in the North Carolina Piedmont (Teramura et al. 1981) and span the range of mean monthly temperatures over which *P. lanceolata* flowers in the piedmont. All variables other than temperature were held as constant as possible. Both chambers maintained photosynthetically active radiation (PAR) at approximately 500 $\mu\text{M}/\text{m}^2/\text{sec}$. All plants were grown in 50% vermiculite/50% gravel and received 1/4 strength Hoagland's solution once a day. Plants were grown at an 8:16 L:D photoperiod for two months to promote vegetative growth and then at a 16:8 L:D to induce flowering. We reduced unknown idiosyncratic chamber effects by switching plants and temperature settings between chambers each month, although it is impossible to eliminate all chamber effects because each set of plants was subjected to idiosyncratic effects at somewhat different developmental stages.

Genotypes were mated according to a Comstock-Robinson type II mating design (Cockerham 1963). Each of five genotypes from population H was reciprocally crossed with each of four genotypes from population D. One genotype from population D was not used for the experiment because we discovered that it was male sterile. Sixty-two percent of the reciprocal crosses were replicated two to three times. Each replicate reciprocal cross involved a different pair of clones from the same genotypes.

Reciprocal crosses were made within and between chambers to produce six temperature treatments and a total of 240 reciprocal cross \times treatment combinations. Each treatment was characterized by a unique combination of maternal prezygotic, paternal prezygotic, and postzygotic temperatures (Fig. 1). The maternal and paternal prezygotic temperatures were the temperatures under which the maternal and paternal parent, respectively, were grown before pollination. The postzygotic temperature was the temperature during pollination and seed maturation on the maternal parent. Limited space did not permit us to include all possible temperature treatment combinations in our experimental design.

Over several months we performed more than 400 matings: 4 D genotypes \times 5 H genotypes \times 2 reciprocal crosses \times 6 treatments \times 1-3 replicate crosses. Because we were continually moving clones around the chambers for the matings, randomizing reciprocal crosses within a chamber was not practical. We did haphazardly shuffle pollinating and non-pollinating plants in the chambers to help reduce within-chamber position effects. Seeds were collected from each replicate reciprocal cross.

We sowed seeds from each reciprocal cross \times treatment combination into an outdoor plot measuring approximately 10 \times 10 m. Replicates for each reciprocal cross were equally represented. The plot was located in an area that had been used as a vegetable garden for several years prior to our experiment. We plowed the plot approximately one month before sowing seeds. After plowing, plants of all species were allowed to colonize the plot naturally. We mowed the whole plot approximately 15 cm aboveground in the early fall of 1990 and 1991 and at the beginning of 1991 and 1992. Near the middle of each growing season, we mowed only between the rows of experimental plants so that we would not remove spikes from the plants.

Seeds were sown in 19 rows. Neighboring rows were separated by 0.5 m; neighboring planting sites within each row were separated by 0.1 m. Seeds from each of the 240 reciprocal cross \times treatment combinations were sown at six randomly assigned planting sites, for a total of 1440 planting sites. Four seeds were sown per site for 186 reciprocal cross \times treatment combinations. The rest of the combinations had fewer seeds available for sowing. For 36 combinations two to three seeds were sown per site, and for 18 combinations one seed was sown.

Most seeds were sown from 6 to 15 March 1990, and on 16 and 17 March it rained, which initiated germination. The last 71 sites (5% of total) were sown with seeds on 18 March. Although seeds at these last sites did not benefit from the rain on 16 and 17 March and therefore germinated more slowly, we believe that this delay did not affect our results for the following reasons. These sites showed the same final percent germination as did the other sites. Seeds were sown by row, not by treatment or by reciprocal cross. Therefore, all treatments and many reciprocal crosses had a few seeds sown after this first rain. Also, the percentage of sites with late-sown seeds was small. We recorded percent germination at each site at four times from 9 April to 4 June. For each site, after the second census beginning on 24 April and as soon as there was a seedling having at least one true leaf, we thinned seedlings to one per site, retaining the largest seedling.

We followed these seedlings over the next three growing seasons and recorded survivorship through flowering in the third year, growth in the first year, year of first flowering and flowering time in the first year (if relevant), spike production per plant per year, and seeds per spike in the second year. We used as our estimate of growth total leaf area, which we estimated from the equation: $LA = 0.29(NLW) + 38.29$, where LA is leaf area, N is leaf number, L is length of longest leaf, and W is width of longest leaf. The coefficients for this equation were obtained from a regression analysis of a separate set of plants (see Lacey, 1996). We collected data on spike production per plant for all flowering plants in the first year. Accidental plowing of part of the experimental plot in the third year prevented us from collecting spike data from approximately one-half of the two year olds.

In the second year, we estimated seeds per spike so that we could estimate total seed production per plant per year. To estimate seeds per spike, we collected three representative spikes from each plant. In the laboratory, we counted the number of capsules touching a line drawn along and paralleling the length of the reproductive portion of each spike; we then ranked each spike according to the amount of seed abortion without regard to source of the spike. Small and unswollen seeds were considered aborted.

In an independent analysis of covariance on 140 spikes from a variety of reciprocal cross \times treatment combinations, capsule number along the line paralleling the spike and seed abortion explained 75% of the variation in total seeds per spike (E. P. Lacey, unpubl. data). Therefore, we used these two variables to estimate total seeds per spike (SDSPK) and total seeds per plant. For spikes showing $> 80\%$ seed abortion: $SDSPK = 67.0856 + 5.533CAP$; for spikes with 20—80% seed abortion: $SDSPK = 112.097 + 5.533CAP$; for spikes with $< 20\%$ seed abortion: $SDSPK = 165.911 + 5.533CAP$, where CAP is the number of capsules touching the straight line running the length of the spike. The coefficients for the above equations were obtained from the analysis of covariance (ANCOVA) of the 140 spikes. Finally, we multiplied mean SDSPK per plant by spike number for each year to estimate total seeds per plant per year.

Statistical Analysis of Life-History Traits

Percent germination, total leaf area (our growth measure), percent flowering of newborns (< 1 year old) and one year olds, spike production of newborns and one year olds, and seeds per spike of 1-yr olds were analyzed using

fixed and mixed models of analysis of variance (ANOVA) and ANCOVA (SAS Institute 1985). The independent variables for all models were parental genotype from population D (D), parental genotype from population H (H), parental temperature treatment, and their interactions. For the mixed models D, H, and their interactions were treated as random, and treatment was fixed. ANCOVA included mean seed mass as a covariate. The computation of mean seed mass for each reciprocal cross \times treatment combination is described in Lacey (1996). For analyses of all dependent variables except germination, the dataset was restricted to planting sites where germination occurred. Percent plants flowering in the first and second summers were the only dependent variables that required transformation to achieve normality before analysis. The values for these variables were arcsine transformed.

For all analyses, the reciprocal cross \times treatment combination was the experimental unit. Thus, each cell in a model contained two values maximum, one for each reciprocal cross. For the analyses of germination, leaf area, percent flowering in first and second summers, and spike production, the models contained no empty cells. For the analysis of seeds per spike, there was one empty cell in each of treatments 1 and 4. Conclusions drawn from the fixed model analyses using Type I, III, and IV sums of squares did not differ for any dependent variable. Therefore, we show only Type III mean squares for all variables.

We used fixed models so that one can compare our results with those of other biologists who have used fixed models to analyze their data. Mixed models were used to see if our data could be extended to *P. lanceolata* populations at large. An assumption of mixed models is that individuals selected for an experiment are chosen randomly from a population. Although we haphazardly, rather than randomly, chose individuals, the specific genotypes that we chose were important only in the sense that they were samples of two populations. Comparing the results of ANOVA and ANCOVA models allowed us to determine if a treatment effect was mediated by seed mass or acted independently of mass.

To examine the source of the parental treatment effects, we examined six pairs of fixed-model contrasts: for maternal prezygotic—temperature effects, we compared treatments 1 versus 2 and 3 versus 4; for paternal prezygotic—temperature effects, we compared treatments 1 versus 6 and 4 versus 5; for postzygotic effects, treatments 3 versus 6 and 2 versus 5 (Fig. 1). These contrasts established whether the overall treatment effect was produced by differences in prezygotic and/ or postzygotic temperature and if a prezygotic treatment effect was transmitted maternally and/or paternally. We did not use a Bonferroni procedure to adjust the P-values for the contrasts because each contrast had an a priori reason to be of interest, and only a small fraction of all possible contrasts was examined.

Nonparametric tests were used to analyze the effect of temperature treatment on survivorship and within-season flowering time in newborns, which we define as individuals younger than one year old. To test for differences in survivorship as a function of treatment, we used the Wilcoxon and log-rank tests (SAS Institute 1985), which are appropriate for experiments ending before all experimental individuals die (Pyke and Thompson 1986). To determine if treatment affected time of flowering in newborns, we assigned each of the 156 flowering newborns to one of two categories: flowering or not flowering by 23 June. Then we performed a χ^2 test on the combined treatment groups. We also performed χ^2 tests on six pairs of treatment groups separately. These pairs were those used for the contrasts in the ANOVA and ANCOVA analyses. Pairs were tested separately to determine the source(s) of the treatment effects.

Fitness Estimates

Using our life-history data, we estimated the fitness of each reciprocal cross \times treatment combination. Our fitness measure was the dominant eigenvalue, λ , for a matrix model of population growth:

$$\begin{bmatrix} F_{11} & F_{12} & F_{13} \\ L_{21} & 0 & 0 \\ 0 & L_{32} & 0 \end{bmatrix} \begin{bmatrix} N_1 \\ N_2 \\ N_3 \end{bmatrix} = \lambda \begin{bmatrix} N_1 \\ N_2 \\ N_3 \end{bmatrix}. \quad (1)$$

The dominant eigenvalue is the geometric growth rate of a population at stable age/stage distribution (Caswell 1989). The transition matrix elements were: F_{11} = mean percent newborns flowering \times mean spikes per newborn \times mean seeds per spike \times mean percent seed germination; F_{12} = mean percent one year olds flowering \times mean spikes per one year old \times mean seeds per spike \times mean percent germination; F_{13} = mean percent two year olds flowering \times mean spikes per two year old \times mean seeds per spike \times mean percent germination; L_{21} = mean percent survivorship of newborns to age 1; L_{32} = mean percent survivorship of one year olds to age 2; N_{1-3} = number of individuals in age classes 0-2. We solved for λ by using the explicit solution for a cubic characteristic equation (Selby 1971). Eigenvalues were then analyzed using fixed and mixed models ANOVA and ANCOVA (SAS Institute 1985). The models contained one empty cell in treatments 1 and 4.

Our matrix model assumed three age classes, which seemed reasonable given our survivorship results. Few individuals survived past their second year. The model also assumed that seeds never germinate if they do not germinate in the first year after their production. Studies of seed dormancy *P. lanceolata* report varying amounts of seed dormancy after the first year. Some populations accumulate little to no seed bank, whereas others have a persistent seed bank (Roberts and Boddrell 1984; van Groenendael 1985; Tonsor et al. 1993). Our assumption of no dormancy is consistent with no observed germination in the second year of our experiment. For the two year olds for which we had no data on spike production because of accidental plowing, we assigned a spike number that equalled the mean spike number for all two-year-old plants for which we did collect data. Because fewer than 5% of our plants survived to age 2, few plants were assigned this mean value.

To determine which life-history trait(s) most strongly contributed to fitness, we performed a multiple regression analysis (SAS Institute 1985) of λ on the life-history traits used to compute the elements in our transition matrix. We chose multiple regression rather than elasticity analysis (de Kroon et al. 1986; van Tienderen 1995) because elasticity analysis measures the relative contributions of matrix elements to λ , but not the contributions of individual life-history traits when multiple traits are used to compute the matrix elements. Three of the five nonzero elements in our transition matrix were products of multiple traits. Multiple regression analysis produces a standardized partial linear regression coefficient for each trait. This coefficient measures the sum of the direct contribution of the trait to λ and the indirect contributions arising from correlations among traits. Traits included in the regression model were percent germination, flowering probabilities of newborns and one year olds; seed set of newborns, one year, and two year olds; and survival probabilities of newborns to age 1 and of one year olds to age 2. The flowering probability of two year olds was not included because all two year olds flowered. We performed regression analyses for each treatment separately and for all treatments combined.

RESULTS

Life-History Traits

Most germination occurred within the first 25 days after the rain on 16 March (Fig. 2). From day 25 to our last measurement date, the effects of parental genotype and treatment on germination were quite visible and significant, regardless of statistical model (Table 1A). Also, treatment affected germination independently of seed mass, which itself did not significantly influence germination. The temperature effect was best explained by postzygotic environment (Table 1B). At day 25, one postzygotic contrast was significant, whereas the other was marginally significant. Both contrasts were statistically significant by 38 days after the rain. The mean germination value for the high postzygotic temperature treatments (Fig. 2: solid symbols) exceeded that for the low post-zygotic temperature treatments (Fig. 2: open symbols) by approximately 35%.

Plants in all temperature treatments showed a similar pattern of growth for the first 10 weeks after the rain. After 10 weeks, growth increasingly differed among treatments. By 17 weeks, when we made our last measurements, the mixed-model analyses showed a significant effect of parental genotype but not treatment on leaf area, regardless of whether seed mass was included in the model (Table 1A). However, the contrasts showed a nearly significant postzygotic temperature effect for treatments 3 versus 6 (Table 1B: $P = 0.051$). High temperature increased leaf area. Also, leaf area was positively correlated with seed mass.

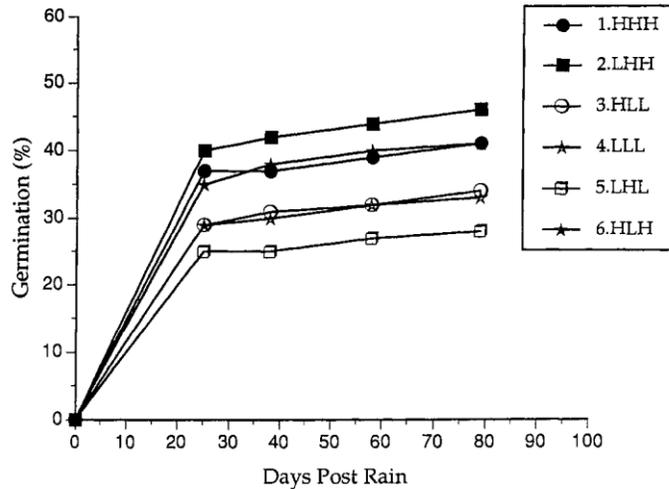


FIG. 2. Mean percent germination of *Plantago lanceolata* shown for each parental temperature treatment over time. The maximum standard error observed for the mean treatment values was 3.5%. The legend shows the maternal prezygotic temperature, paternal prezygotic temperature, and postzygotic temperature, in that order, for each treatment. L, low temperature; and H, high temperature.

There was a marginally significant $D \times$ treatment inter-action for leaf area at 10 weeks that became significant at 17 weeks using the mixed models (Table 1A). Parental genotypes from population D responded differently to temperature treatment. The treatments did not alter the rank order among parental genotypes, but they did modify the relative differences in offspring size among genotypes. The comparison of treatments 1 and 4 is most informative because these two treatments best mimic environments to which natural populations might be subjected (maternal and paternal prezygotic and postzygotic temperatures either all high or all low). At high temperature (treatment 1), parental genotype D2 produced much smaller offspring (mean \pm 2 SE = 86 ± 27) than did genotypes D5 and D7 (offspring means \pm 2 SE = 122 ± 36 and 128 ± 29 , respectively). At low temperature (treatment 4), offspring sizes did not significantly differ.

Most offspring survived through their first summer, approximately 70% survived to age 1, but fewer than 5% survived to age 2 in any treatment. Survivorship did not differ significantly among treatments (log-rank test: $\chi^2 = 5.3$, df = 5, $P = 0.38$; Wilcoxon test: $\chi^2 = 5.7$, df = 5, $P = 0.34$).

Parental genotype and treatment influenced flowering and spike production in the newborn age class. Approximately 25% of newborns flowered, and parental genotypes from population D differed significantly in their response to treatment with respect to probability of flowering in both fixed and mixed ANCOVA models (Table 1; Fig. 3A). For example, at low temperature (treatment 4) many fewer newborns of parent D1 flowered than did newborns of D2 and D5 (offspring means \pm 2 SE for D1, D2, and D5 = 14.8 ± 6.3 , 46.3 ± 26.4 , and 34.8 ± 10.2 , respectively). At high temperature (treatment 1), percent offspring flowering did not significantly differ among parental genotypes. Flowering percent-age was positively and significantly correlated with seed mass (Table 1). The main effects of genotype and treatment on percent flowering of newborns were not significant, nor were the contrasts.

The percent of newborns flowering by 23 June ranged from 24% to 62% across treatments (Fig. 3B). Treatment marginally affected percent flowering by 23 June when all treatment groups were combined ($\chi^2 = 9.35$, df = 5, $P = 0.096$). It significantly affected percent flowering when treatments 1 and 6 were compared ($\chi^2 = 4.34$, df = 1, $P = 0.037$). No other χ^2 test showed a significant treatment effect. Treatments 1 and 6 indicate that high paternal prezygotic temperature accelerated flowering in newborns. Treatments 4 and 5 show the same, although insignificant, pattern.

Spike production varied greatly among individual newborns, with a few producing as many as 35 spikes. Treatment means for spike production per newborn were small, however, ranging from one to two spikes per

individual. The main effects of genotype and treatment on spike production were not significant, nor were the contrasts (Table 1). However, parental genotypes did differ significantly in their response to treatment (Fig. 3C). At low temperature (treatment 4), spike production for offspring of genotype D1 was significantly lower than for D5 or D7 offspring (offspring means \pm 2 SE for D1, D5, and D7 = 0.29 ± 0.28 , 1.95 ± 1.29 , and 1.30 ± 1.00 , respectively). At high temperature (treatment 1), there was no significant difference. The D X treatment interaction was significant in the fixed-model ANCOVA and marginal in the mixed-model ANCOVA (Table 1: $P = 0.06$). Also, the fixed-model analysis showed a marginally significant H X treatment interaction ($P = 0.075$).

Parental environmental effects observed in the newborn age class disappeared in one year olds. More than 90% of one year olds flowered, but genotype, treatment, and seed mass did not significantly affect percent flowering (Table 1). Mean treatment values for spike production per individual ranged from 49 to 68 spikes, with some one year olds producing more than 150 spikes per individual, but neither treatment nor seed mass significantly affected spike production, either for one year olds alone or for newborn and one-year-old data combined (Table 1). Mean treatment values for seed number per spike ranged from 247 to 258 seeds per spike, but genotype, treatment, and seed mass did not significantly affect seed number. No contrast was significant for any variable measured on one year olds.

The few individuals that survived to their second year all flowered. Mean spike production of two year olds for which we were able to collect data was 76 spikes per individual.

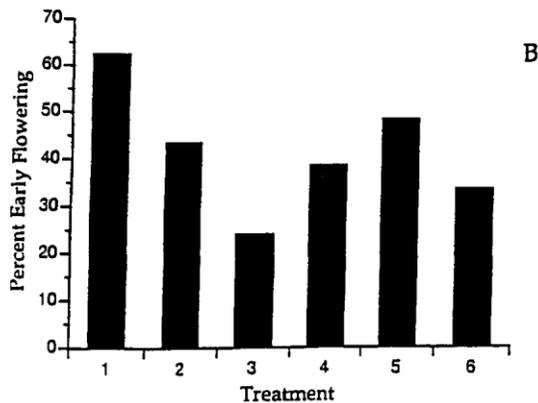
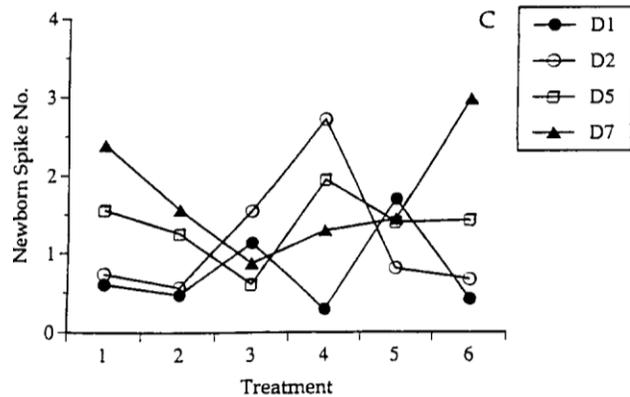
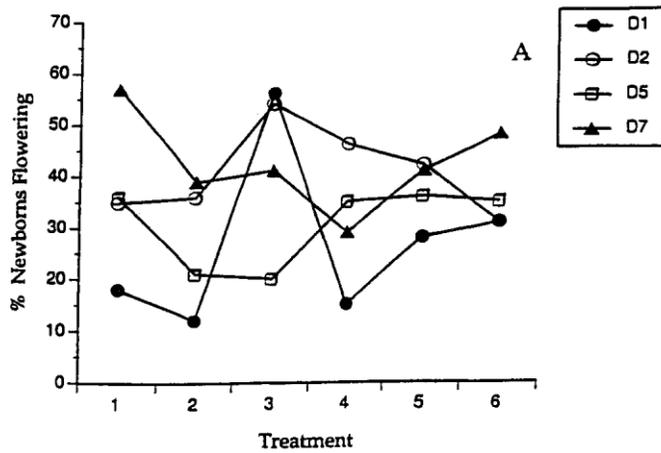
Fitness

Both parental genotype and temperature treatment significantly affected the matrix-model eigenvalue (λ), our fitness estimate, in all statistical analyses (Table 1). The treatment effect is best explained by differences in postzygotic environment (Fig. 4). The mean λ value for the high postzygotic temperature treatments exceeded that for the low treatments by approximately 50%. Surprisingly, seed mass did not significantly affect λ (Table 1).

The life-history traits most influencing λ were traits expressed during the first growing season: germination, probability of flowering in newborns, and seed set in newborns (Table 2). Seed set in one year olds also significantly affected λ , but to a lesser extent. The relative contributions of life-history traits to λ varied across treatments, however, the general pattern is consistent with the results of the regression analysis for all treatments combined.

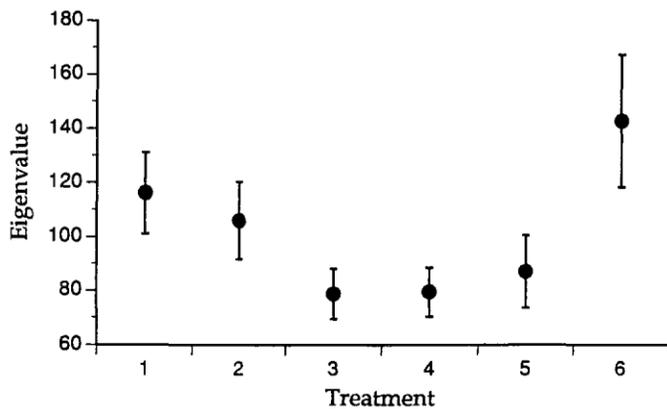
TABLE 1. (A) ANCOVA results for *Plantago lanceolata*. Variables are germination at 25 days after rain, leaf area at 17 weeks, percent flowering in newborns and one-year-old age classes, spike production of newborns and newborns plus one year olds combined, and population growth rate (λ). Factors in the models were parental genotype from population D (D), genotype from population H (H), parental temperature (TRT), all interactions, and mean seed mass (SDMASS), included as a covariate. The r^2 values for the full models of the above variables were 0.72, 0.62, 0.59, 0.51, 0.61, 0.47, and 0.55, respectively. Error degrees of freedom for germination, leaf area, percent flowering and spike production, and λ were 111, 107, 108, and 91, respectively. (B) Contrasts show the significance levels for parental temperature effects using fixed models. If a mixed-model significance level differed from that of the fixed model, both are shown with the former in parentheses. † $0.05 < P < 0.10$; * $0.01 < P \leq 0.05$; ** $0.001 < P \leq 0.01$; *** $P < 0.001$; ns, $P \geq 0.10$.

A. Full model					% Flowering		Spike production			
Source	df	Mixed-model denominator	Germination MS	Leaf area MS	Newborns MS	One year olds MS	Newborns MS	One year olds MS	λ	
D	3	$MS_{DH} + MS_{DTRT} - MS_{DHTRT}$	0.324***(*)	14,505 ^{ns} (*)	0.212 [†] (ns)	0.015	4.887	8260 ^{ns} (ns)	55,063***(***)	
H	4	$MS_{DH} + MS_{HTRT} - MS_{DHTRT}$	0.395***(*)	10,268 ^{ns} (*)	0.039	0.041	5.102	2366	33,292***(**)	
DH	12	MS_{DHTRT}	0.086***	2140	0.121	0.046	5.119 ^{ns} (ns)	1441	4997	
TRT	5	$MS_{DTRT} + MS_{HTRT} - MS_{DHTRT}$	0.120***(*)	3022	0.061	0.036	2.095	1201	23,213*	
D X TRT	15	MS_{DHTRT}	0.006	3280 ^{ns} (*)	0.185*	0.026	5.455 [†] (†)	1629	6315	
H X TRT	20	MS_{DHTRT}	0.014	1456	0.077	0.024	4.107 [†] (ns)	1392	4108	
D X H X TRT	60	MS_E	0.015	1616	0.097	0.047	3.116	2444	4836	
SDMASS	1	MS_E	0.006	32,091***(***)	0.484*	0.005	2.107	567	6504	
Error			0.019	1789	0.090	0.045	2.628	2671	8561	
B. Contrasts					Maternal prezygotic		Paternal prezygotic		Postzygotic	
Variable	Source	df	1 vs. 2	3 vs. 4	1 vs. 6	4 vs. 5	2 vs. 5	3 vs. 6		
Germination	TRT	1	ns	ns	ns	†	***	*		
Leaf area	TRT	1	ns	ns	ns	ns	ns	†		



A pre	H	L	H	L	L	H
P pre	H	H	L	L	H	L
post	H	H	L	L	L	H

FIG. 3. Percent flowering and spike production in offspring of *Plantago lanceolata* shown for each parental temperature treatment. (A) Reaction norms for newborns of parental genotypes from population D. Maximum standard errors observed for treatments 1–6 were 12.6, 13.3, 15.6, 13.2, 11.3, 13.1, respectively. (B) Percent flowering newborns that flowered by 23 June. (C) Reaction norms for spike production in newborns of parental genotypes from population D. Maximum standard errors observed for treatments 1–6 were 0.87, 0.73, 0.63, 1.34, 0.94, and 1.01, respectively. L, low temperature; H, high temperature; M pre, maternal prezygotic temperature; P pre, paternal prezygotic temperature; post, postzygotic temperature.



M pre	H	L	H	L	L	H
P pre	H	H	L	L	H	L
post	H	H	L	L	L	H

FIG. 4. Mean eigenvalues (± 1 SE) for offspring of *Plantago lanceolata* shown by parental temperature treatment. Abbreviations are identified to those in Figure 3.

TABLE 2. Standardized partial linear regression coefficients for the multiple regression of λ on the life-history traits used to compute the transition matrix elements for the calculation of λ in *Plantago lanceolata*. Coefficients ($P < 0.05$) are shown for the regression analyses of each temperature treatment alone and for all treatments combined. Statistically significant coefficients ($P < 0.05$) are boldfaced. Marginally significant coefficients ($0.05 \leq P < 0.10$) are not. Insignificant coefficients ($P \geq 0.10$) are not shown. G, percent germination; F_0 and F_1 , probabilities of flowering as a newborn and a one year old, respectively; S_0 , S_1 , and S_2 , seed set of a newborn, one year old, and two year old, respectively; L_1 and L_2 , probabilities of survival of a newborn to age 1 and of a one year old to age 2, respectively.

Trait	Treatment						Combined
	1	2	3	4	5	6	
G	0.27	0.30	0.59	0.51	0.25	—	0.31
F_0	0.32	0.22	0.21	0.26	0.25	0.47	0.36
F_1	-0.20	—	-0.32	—	—	—	—
S_0	0.35	0.64	0.37	0.35	0.46	0.47	0.40
S_1	0.30	—	0.27	—	0.16	—	0.11
S_2	—	—	—	—	0.29	—	—
L_1	0.12	—	0.28	—	—	—	0.08
L_2	—	—	—	—	-0.21	—	—

DISCUSSION

Growth-chamber and greenhouse studies of plants have reported large parental environmental effects on many off-spring traits (e.g., see reviews in Rowe 1964; Roach and Wulff 1987; Gutterman 1992; Wulff 1995; Donohue and Schmitt 1998; Rossiter 1998), and these studies have provided the empirical basis for the argument that parental environmental effects influence the evolution of natural plant populations. If we are to understand the evolutionary importance of parental environmental effects in natural populations, however, we need to determine if effects observed in artificial environments are expressed in natural environments. Recent

studies have shown that offspring environment can influence the magnitude of parental effects (Stratton 1989; Schmitt et al. 1992; Schmid and Dolt 1994; Donohue and Schmitt 1998; Thiede 1998). Also, we need to measure the effect of parental environment on offspring fitness.

The results of our experiment show generally that parental environment, primarily postzygotic environment, influences offspring fitness and the life-history traits most contributing to fitness in field-grown offspring of *P. lanceolata*. First, we discuss the parental environmental effects on germination, growth, and flowering time and consider our results in light of Lacey's (1996). She raised offspring from the same reciprocal cross \times treatment combinations in growth chambers. Next we discuss the fitness effects. Finally, we discuss the possible role of parental temperature effects in the evolution of life-history patterns in natural *P. lanceolata* populations.

Germination, Growth, and Flowering Time Comparisons

Four general conclusions can be drawn from the comparison of Lacey's (1996) and our data. First, the influence of parental environment on offspring phenotype is weaker in natural habitats than in artificial habitats. For *P. lanceolata*, this weakening manifested itself as a decline in both number and magnitude of treatment effects in field-grown offspring. High parental temperature increased germination by approximately 53% in the growth chamber, but only by 35% in the field. Size at 5 weeks in treatment 1 (high temperature) exceeded size in treatment 4 (low temperature) in chamber-grown offspring by 5%. In our experiment, the size difference was smaller (1% at 7 weeks, 2% at 17 weeks). The significant main effect of parental treatment on flowering time in chamber-grown offspring was not significant for field-grown offspring. Prezygotic effects detected in growth chambers were largely undetected in the field. We suspect that increased heterogeneity of the offsprings' immediate environment in the field explains this weakening. Our results suggest that "large" effects in an artificial environment may not be so large in nature.

Second, growth-chamber data for onset of flowering may be somewhat misleading for perennial species such as *P. lanceolata*. Lacey observed that low paternal prezygotic temperature accelerated flowering. We observed that low paternal prezygotic temperature retarded within-season flowering of newborns. We suspect that the confounding of first-flowering year and within-season flowering time in an artificial environment contributed to this discrepancy. In general, the results argue strongly for conducting more empirical studies in field conditions so that we can better assess the magnitude and type of parental environmental effects in natural populations.

Third, even though parental environmental effects may be weaker, they do influence the phenotypic variation for some traits in the field. Parental treatment strongly influenced germination. We also detected genotype-specific differences in response to parental treatment for growth and newborn reproduction. These parental genotype \times parental environment interactions are best explained by changes in the relative differences in offspring growth and flowering among parental genotypes and could be evolutionarily important (to be discussed later). The differences were measurable when comparing treatments 1 and 4, which mimic natural spatial and temporal temperature change better than do the other treatments. The genotype-specific differences in response to parental treatment for growth became increasingly significant with time. If treatment was influencing growth rate, then one would expect this effect to be detected later rather than earlier in plant growth. This effect might also appear later if treatment affected adult development, such as onset of flowering, which may be coupled to growth. Resource limitation likely produces a trade-off between flowering and vegetative growth (e.g., Stearns 1989; Reznick 1992).

Fourth, postzygotic environment more strongly influences offspring phenotype than does prezygotic environment. Only postzygotic environment significantly influenced germination and growth in both growth-chamber and field experiments. Postzygotic effects could be produced by environmentally induced parental effects (environmentally induced phenotypic change truly transmitted across generations); direct environmental modification of early offspring ontogeny (an intragenerational process); gametophytic/gametic selection; repeatable nonrandom mutation; and indirect genetic effects, in which case offspring phenotype is determined not only by the Mendelian transmission of parental genes but also by the genes' effect on postzygotic environment (reviewed by Lacey 1998). Which of these is responsible for the effects that we observed is

unclear; in general, designing experiments to differentiate between the possible causes is difficult. Regardless of the cause, the postzygotic environment, used in its broadest sense to include all the above possibilities, clearly influences offspring phenotype.

Effect on Fitness

Whether parental environmental effects on individual life-history traits are evolutionarily important depends in large part on their cumulative effect on offspring fitness. It seems intuitively possible that small effects on individual traits might interact with each other multiplicatively and synergistically to produce large fitness effects. In contrast, large effects on individual traits might offset each other in such a way that the cumulative effect of parental environment on fitness is minimal. Parental treatment strongly affected off-spring fitness in *P. lanceolata*. The fitness of offspring averaged over the high postzygotic temperature treatments exceeded average fitness for the low postzygotic treatments by almost 50%.

Parental environment also influenced the most important fitness components: germination, probability of flowering as a newborn, and spike production of newborns. Germination was strongly affected by treatment averaged over genotypes. The responses to treatment with respect to probability of flowering and spike production in newborns were genotype specific. Overall, it appears that parental environment can influence offspring fitness by altering the phenotypic expression of life-history traits that contribute to fitness.

Our data provide some of the strongest evidence thus far that parental environmental effects (used in the broadest sense) influence the evolution of natural plant populations. Two other studies have experimentally manipulated an environmental factor to measure the strength of parental environmental effects in field populations of plant species (Schmitt et al. 1992; Donohue and Schmitt 1998), but to our knowledge, ours is the first to combine the effects of multiple life-history traits to estimate the fitness effect while also controlling for paternity. Given that our experimental plot mimicked a disturbed or recently abandoned site, where *P. lanceolata* typically grows, the data suggest that parental environment influences offspring fitness in many natural plantain populations. The fact that the mixed-model analyses detected significant treatment effects and parental genotype \times treatment interactions also supports this conclusion. The observed effects on our small sample of genotypes will likely manifest themselves in larger populations.

Evolution of Life-History Pattern

Our parental temperature treatments were artificial in the sense that they lacked the variability and unpredictability that typify natural temperature changes during the flowering season of *P. lanceolata* in the North Carolina Piedmont. The experiment does, however, suggest how real parental temperature patterns might affect the intensity and direction of selection for alternative life-history patterns in natural populations.

First, parental environmental effects may strongly influence the selection for flowering phenology within seasons in *P. lanceolata*. Flowering occurs from May into August in the North Carolina Piedmont and temperature, on average, increases during that time (Teramura 1978). Onset of flowering is partially genetically controlled in *P. lanceolata* (e.g., Primack and Antonovics 1981; Wolff 1987; Wolff and van Del-den 1987; Lacey 1996), and flowering phenology appears to be under strong selection in many species (e.g., Schmitt 1983; Schemske 1984; Rathcke and Lacey 1985). Natural selection is a multigenerational process, however, in that parental fitness is determined by both the quantity and quality of off-spring (e.g., Lacey and Pace 1983; Donohue and Schmitt 1998; Wade 1998). In spite of this, fitnesses associated with different flowering times are typically measured only in terms of offspring quantity. Our data indicate that offspring quality should not be ignored. The data suggest that offspring produced late in the flowering season, when it is warmer, may be much more fit (of higher quality) than are offspring produced early, when it is cooler. From the perspective of offspring fitness, selection should favor individuals flowering in July and August, when it is hot, and select against individuals flowering in May and June, when it is cooler. We would like to know how flowering time affects offspring quantity as well as quality. At this point, we lack such information for *P. lanceolata*. It is possible that parents flowering late in the season set more seeds than do parents flowering early, which should intensify the

selection pressure for later flowering. In contrast, early flowering parents may set more seeds, offsetting the reduction in offspring quality. The possibility of such a trade-off is being examined.

Second, our data suggest that postzygotic temperature also strongly influences germination. Given the increase in temperature throughout the flowering season, we would predict that offspring seed germination should be positively correlated with parental flowering time. Low postzygotic temperature slows seed maturation and/or reduces germination in a number of species (e.g., Koller 1962; Robertson et al. 1962; Gutterman 1980, 1981, 1983; Siddique and Goodwin 1980; Wulff 1986). In some inbred lines of *Avena fatua*, decreasing postzygotic temperature increases dormancy, regardless of the temperature during germination (Sawhney and Naylor 1979). Low postzygotic temperature also may induce dormancy in *P. lanceolata*. The offspring used in Lacey's (1996) and our experiments received no cold treatment before planting. If we had subjected the offspring to cold before sowing, then the treatment effect on germination might have disappeared. This needs to be tested.

The evolution of a life-history pattern is determined not only by the genetic variation for traits constituting the pattern, it is also determined by the environment in which selection occurs. If environment affects the phenotypic overlap among genotypes, then the rate of response to selection will vary across environments (Sultan 1987; Schlichting 1989; Schmitt 1995). If environment alters the relative ranking of genotypes, then identical selection pressures will select for different genotypes in different environments. If environment alters the structure of genetic correlations among traits, then identical selection pressures will produce different evolutionary responses of correlated traits (Gupta and Lewontin 1982; de Jong 1990; Stearns 1989; Stearns et al. 1991).

Parental environmental effects potentially influence the evolution of natural populations for all the above reasons (Antonovics and Schmitt 1986; Kirkpatrick and Lande 1989; Stratton 1989; Biere 1991; Schmitt et al. 1992; Platenkamp and Shaw 1993; Lacey 1996). Growth-chamber and green-house studies that control for paternity have shown that off-spring responses to parental environment differ among parental genotypes in several plant species (Platenkamp and Shaw 1993; Schmid and Dolt 1994; Case et al. 1996; Lacey 1996; Sultan 1996; Mazer and Wolfe 1998). Our study, which also controls for paternity, shows that genotypic-specific differences can be detected in the field for two life-history traits that strongly contribute to offspring fitness: probability of flowering and spike production of newborns. For both traits, offspring response to parental temperature differed among parental genotypes. Differences were greater at low temperature than at high temperature. These results suggest that the response to selection on these traits may be more rapid in cooler habitats.

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