Parental Effects in Plantago Lanceolata L. I.: A Growth Chamber Experiment to Examine Pre- and Postzygotic Temperature Effects

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Abstract:

In spite of the potential evolutionary importance of parental effects, many aspects of these effects remain inadequately explained. This paper explores both their causes and potential consequences for the evolution of life-history traits in plants. In a growth chamber experiment, I manipulated the pre- and postzygotic temperatures of both parents of controlled crosses of *Plantago lanceolata.* All offspring traits were affected by parental temperature. On average, low parental temperature increased seed weight, reduced germination and offspring growth rate, and accelerated onset of reproduction by 7%, 50%, 5%, and 47%, respectively, when compared to the effects of high parental temperature. Both pre- and postzygotic parental temperatures (i.e., prior to fertilization vs. during fertilization and seed set, respectively) influenced offspring traits but not always in the same direction. In all cases, however, the postzygotic effect was stronger. The prezygotic effects were more often transmitted paternally than maternally. Growth and onset of reproduction were influenced both directly by parental temperature as well as indirectly via the effects of parental temperature on seed weight and germination. Significant interactions between parental genotypes and prezygotic temperature treatment (G × E interactions) show that genotypes differ in their intergenerational responses to temperature with respect to germination and growth. The data suggest that temperature is involved in both genetically based and environmentally induced parental effects and that parental temperature may accelerate the rate of evolutionary change in flowering time in natural populations of *P. lanceolata.* The environmentally induced temperature effects, as mediated through G × (prezygotic) E interactions are not likely to affect the rate or direction of evolutionary change in the traits examined because postzygotic temperature effects greatly exceed prezygotic effects.

Key words: Germination, growth, life-history evolution, maternal effects, onset of reproduction, parental effects, paternal effects, *Plantago lanceolata*, pre- and postzygotic effects, seed weight, temperature

Article:

Parents can influence the phenotypes of their offspring beyond their direct chromosomal contributions. In many circumstances, the parental environment may transcend the generational barrier to alter the phenotypic expression of fitness traits in offspring (see reviews of plants, Rowe 1964; Schaal 1984; Roach and Wulff 1987; Lacey 1991; insects, Mousseau and Dingle 1991; fishes, Reznick 1991; mammals, Cowley 1991; amphibians, Kaplan 1991; reptiles, Sinervo 1991). In spite of their pervasiveness, many aspects of parental effects remain inadequately explained. For example, we would like to know: (1) When during the parent's lifetime can the environment produce an effect that is transmitted to the off-spring? (2) Are environmentally induced effects transmitted paternally as well as maternally? (3) Are adult as well as juvenile life-history traits affected directly by parental environment? (4) Are some genotypes more strongly affected by ancestral environments than are others? Here I address these questions by presenting the results of the first of several experiments on parental effects in *Plantago lanceolata* L., ribwort plaintain, an herbaceous short-lived perennial plant species. I focus on the temperature under which a parental generation is grown and its impact on the expression of off-spring life-history traits when both parental and offspring generations are grown in controlled environments.

I address not only some causes of parental effects but also their potential evolutionary consequences. Several biologists have hypothesized that parental effects may accelerate, retard, or even alter the direction of evolutionary change in natural populations (Falconer 1965, 1983; Riska et al. 1985; Antonovics and Schmitt 1986; Kirkpatrick and Lande 1989, 1992; Lande and Kirkpatrick 1990). Presently, there is little empirical evidence to support this hypothesis. Therefore, I address two additional questions: (5) Are parental effects actually manifested in natural populations? (6) How would parental effects alter the responses to natural selection for various life-history traits?

Previous studies of maternal/paternal effects suggest that juvenile life-history traits, for example, seed size and germination rate, respond strongly to the parental environment (e.g., Schaal 1984; Stanton 1984; Roach and Wulff 1987; Aarssen and Burton 1990; Schmitt et al. 1992; Platenkamp and Shaw 1993). Fewer studies have shown that adult characters, for example, components of reproduction, may also respond (Edwards and Emara 1970; Schaal 1984; Roach and Wulff 1987; Miao et al. 1991 a,b; Wulff and Bazzaz 1992). Because juvenile traits can strongly influence adult traits (e.g., Gross 1984; Schaal 1984; Stanton 1984; Roach 1986; Wulff 1986a,b), it is unclear whether the parental environment directly affects the adult traits or does so indirectly through the direct effects on juvenile traits. Therefore, in my experiment I measured both the direct and indirect effects of temperature on the juvenile life-history traits, seed weight and germination, and on the adult traits, growth and onset of reproduction.

I also looked for evidence of paternal as well as maternal transmission of the temperature effects. Most parental effects are assumed to be transmitted maternally and, consequently, evidence for "maternal" effects abounds (Roach and Wulff 1987). Often, however, experimental designs have not al-lowed biologists to discriminate between maternal and paternal transmission. For example, biologists may not have controlled for nonrandom pollination or may have used completely selfing species. A few studies provide evidence for the paternal transmission of parental effects (Beddows et al. 1962; Garwood et al. 1970; Aksel 1977; Tilney-Basset 1978; Sears 1980; Szmidt et al. 1987; Wagner et al. 1987; Corriveau and Coleman 1988; Richardson and Stephenson 1992), and recently, biologists have found that parental environment can affect pollen size, viability, and growth rate (e.g., Young and Stanton 1990; Lau and Stephenson 1993). Therefore, it seems timely to explore more explicitly the role of the paternal parent in transmitting parental effects.

A parent passes through four developmental phases during its life, and the environment can, theoretically, initiate a parental effect in each phase. An effect could be produced during: (1) the seed phase, which includes both the time of seed development of the parent while attached to its maternal parent and the time of independent existence between abscission and germination; (2) the vegetative phase; (3) anthesis, or flowering, which includes the gametophytic phase and the final stages of gamete production up to and including fertilization; and 4) fruiting, during which time the parent produces fruits and the offspring (i.e., seeds) are themselves undergoing embryonic development. Phases 1-3 are prezygotic phases relative to the offspring generation, whereas phase 4 is a postzygotic phase. In my experiment, I examined the independent effects of both pre- and postzygotic phases. Additionally, I looked for evidence that offspring genotypes differ in their intergenerational responses to parental temperature, that is, I looked for significant G × (prezygotic) E interactions.

Finally, I chose to manipulate temperature experimentally because temperature is an environmental factor that changes predictably, on average, both over large geographical regions and locally within and among populations. Within populations, individuals often differ genetically in their phenological schedules (Primack and Antonovics 1981; Wolff 1987; Wolff and van Delden 1987). Therefore, individuals may experience different temperature regimes either because they have predictably different phenological schedules or because they grow in different geographical regions. It is likely that temperature functions as a selective agent intragenerationally for *P. lanceolata* (Teramura and Strain 1979; Teramura et al. 1981; Primack and Antonovics 1982; Pons and Van der Toorn 1988). It is therefore worth asking if and how temperature might influence the process of natural selection intergenerationally.

METHODS

Study Species

Plantago lanceolata L. (Plantaginaceae), ribwort plantain, is a weedy, cosmopolitan, herbaceous perennial plant species that was introduced to North America from Europe. It grows vegetatively as a rosette and produces small flowers on spikes that arise from axillary buds. The species is. protogynous, gynodioecious, and self-incompatible (van Damme 1984).

Experimental Design

In August 1988, I collected plants from two populations of *P. lanceolata* in Durham, North Carolina, and cloned them to produce multiple copies of each genotype. The fields from which the plants were collected appeared to be mown occasionally and were approximately 3 km apart. I collected the plants haphazardly except that in order to increase the genetic diversity of the plants selected, I collected only plants growing more than one meter from each other. Each plant was assumed to be a distinct genotype. Rootstock cuttings were grown in 50% vermiculite and 50% gravel in a green-house (26°C day/20°C night; 16 h days) until each had produced 3-5 new leaves. I further divided established cuttings 2-3 additional times in the same way until there were 40— 60 clones of each of five genotypes per population. Because very few cloned individuals died during the whole experiment, clonal selection was minimal.

I transferred the cloned individuals of the 10 genotypes at the 2-5 leaf stage to one of two growth chambers on January 24,1989. The chambers differed in their temperature regime: either 20°C days/15°C nights or 26°C days/20°C nights. These temperatures approximate the mean monthly temperatures for May and July, respectively, in Piedmont, North Carolina (Teramura et al. 1981) and span the range of temperatures over which *P. lanceolata* flowers in North Carolina. Each clone was randomly assigned to the high or low temperature chamber such that there was an equal number of clones per genotype in each chamber. All variables other than temperature were held as constant as possible. The plants and temperature settings were switched between chambers each month to minimize any idiosyncratic chamber effect. Both chambers maintained PAR at approximately 500 $\mu m/m^2/sec$. All plants received one-quarter strength Hoagland's solution once a day and were grown under 8-h days for two months to promote vegetative growth. Then daylength was increased to 16 h to induce flowering.

Genotypes were mated using a Comstock-Robinson type II mating design (Cockerham 1963). Each genotype from one population was mated with each genotype from the other population, with each genotype being used both maternally and paternally, but in different crosses. I will use the word "cross" to refer to the mating of a genotype from one population with a genotype from the other population. A "cross" includes the reciprocal crosses. Most reciprocal crosses included 2-3 replicate matings, that is, although the genotypes for the matings and the direction of the cross were the same, different pairs of clones were used for each mating. Seldom was a cloned individual mated more than once. When it be-came apparent during the matings that one genotype was male sterile, that genotype was removed from the experiment.

I mated the genotypes by covering flowering spikes with pollination bags. Each mating produced seeds from only one plant. Matings were carried out over several months in 1989. To reduce the bias in seed weight that might arise from in-creasing resource limitation that is often associated with continued seed production, I periodically removed from all clones unused spikes that were past anthesis. This reduced the possibility that seed production in the pollination bags might be resource-limited because of seed production on older spikes.

I crossed genotypes that were growing in the same chamber and also genotypes that were, until the cross, growing in different chambers. In this way, I measured the importance of three components of the temperature effect: the maternal prezygotic temperature, that is, the temperature under which the mother was growing before pollination, paternal prezygotic temperature, and postzygotic temperature. The post-zygotic temperature effect included the temperature during the final phase of gametophytic development, and consequently, included a small portion of the prezygotic temperature effect. However, the prezygotic temperature effects that I measured were entirely prezygotic.

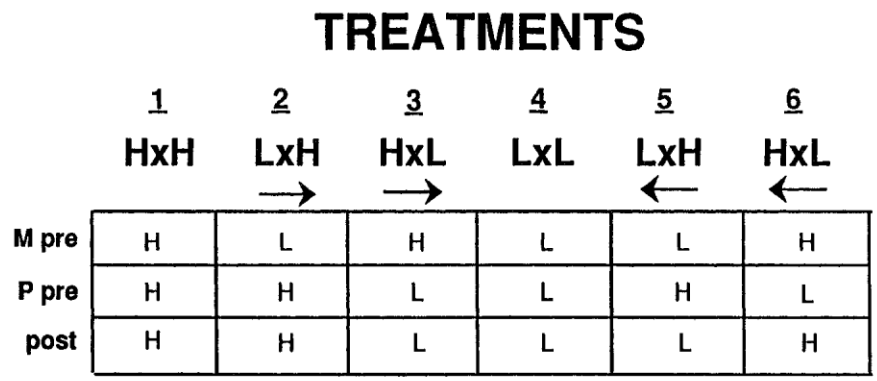

FIG. 1. Experimental design describing the six temperature treatments by maternal prezygotic temperature (M pre), paternal prezygotic temperature (P pre), and postzygotic temperature (post) for *Plantago lanceolata*. The arrow indicates the direction of transfer between temperatures (i.e., chambers)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

My crossing design yielded six temperature treatments for the parental generation (Fig. 1). Each 5 × 4 factorial mating design was replicated for the 6 temperature treatments. Summed over all treatments, there were 120 crosses with two reciprocal crosses per cross plus replicate matings within each reciprocal cross. For two treatments (1 and 4), parents were grown and seeds matured at the same temperature. For two treatments (2 and 6), either the mother or father was grown at high temperature and the other parent, which had been grown at low temperature, was transferred to the high temperature chamber for pollination and seed set. For two treatments (3 and 5), either the mother or father was grown at low temperature, and the other parent, having been grown at high temperature, was transferred to the low temperature for pollination and seed set. Each treatment was characterized by a unique combination of maternal prezygotic, paternal prezygotic, and postzygotic temperatures.

In 1990 I began collecting data. I weighed individually 12 seeds per reciprocal cross per treatment. The replicate matings contributed equally to these 12 seeds, such that if there was only one replicate, all seeds were randomly selected from that replicate; if there were two replicates, six were randomly selected from each replicate, etc. For most reciprocal cross/ treatment combinations, there were at least two replicate matings.

I then measured the percent germination of the weighed seeds plus 12 unweighed seeds for each reciprocal cross/ treatment combination under high and low temperature. The same chambers were used for both generations. Six weighed seeds and six unweighed seeds per combination were randomly assigned to each chamber. All seeds per combination were germinated on moist filter paper in a petri dish. I recorded germination over two weeks and then transplanted to pots seedlings from the three fastest germinating seeds that had been weighed. These seedlings, one per pot, grew in the chamber in which the seed had germinated. If three weighed seeds did not germinate, I transplanted seedlings from unweighed seeds.

Over the next several months, I collected data on growth and onset of reproduction. To measure plant growth, I measured leaf number and length of the longest leaf at approximately 27 and 43 days after germination and also width of the longest leaf at 43 days. These data were used to estimate total leaf area, my measure of plant size. Onset of reproduction was measured in terms of percent progeny flowering by day 73.

The following equations were used to estimate total leaf areas: at 27 days, $LA = 0.55 (N \times L) + 11.41$ (r^2 0.84); at 43 days, $LA = 0.29 (N \times L \times W) + 38.29$ ($r^2 = 0.88$), where LA = leaf area, N = leaf number, L = length of longest leaf, and W = width of longest leaf. These equations were derived from a regression analysis (SAS 1985) performed on a separate set of plants (Lacey, unpubl. data).

Statistical Analysis

Each offspring trait (seed weight, percent germination, leaf area, and onset of flowering) was analyzed with analyses of variance (ANOVA) and covariance (ANCOVA). Percent germination was examined after one and two weeks. Germination was examined twice to obtain information about early germination rate as well as total

germination potential. Loosely speaking, data from week 1 provide a better estimate of early germination rate, and data from week 2 a better estimate of total germination potential prior to winter stratification. Leaf area was examined at approximately 27 and 43 days. Seed weight and leaf area data were log-transformed, and percent germination was arcsine-transformed before per-forming the analyses.

The model for seed weight included as independent variables the parent from each population and the temperature treatment plus all interactions. The model for percent germination also included the temperature at which offspring seeds germinated (referred to hereafter as offspring temperature) and the interactions with offspring temperature. Seed weight was included as a covariate. The models for leaf area and onset of reproduction included all the variables used in the germination analysis and also percent germination after two weeks as a covariate. Seed weight showed a curvilinear relationship with percent germination and onset of flowering, and percent germination showed curvilinear relationships with leaf area and percent flowering. Therefore, for these covariates, I used the quadratic polynomial form of the covariate in the ANCOVA models. For all other covariates I used the linear form.

For each ANOVA and ANCOVA, the reciprocal cross by treatment combination was used as the experimental unit. For all dependent variables, the full models contained a few empty cells (maximum = 3 for any one model). Conclusions drawn from the fixed model analyses using Type I, III, and IV sums of squares (SAS 1988) did not differ. In this paper, I present Type III sums of squares.

There were two reasons for choosing reciprocal cross by treatment as the experimental unit. First, progeny of a replicate mating within a reciprocal cross are statistically not independent of each other (e.g., they are produced by the same mother). Therefore, data on progeny derived from the same replicate mating were pooled. Second, space limitations prevented me from collecting data for each replicate mating separately. However, for seed weight and germination, all replicate matings per reciprocal cross were represented as equally as possible, for example, seed weights and germination were measured on a sample of seeds drawn from all matings.

Results of both fixed and mixed ANOVA and ANCOVA models are presented. I show the fixed-model results so that one can compare my results with those of other biologists studying parental effects. Most have used fixed models to analyze their data. The mixed-model analyses are included to see if the data could be extended to *P. lanceolata* populations at large (question 5 above). An assumption of mixed models is that individuals selected for an experiment are chosen randomly from a population. Although I haphazardly chose plants for cloning, the specific genotypes that I used are important only in the sense that they were samples of two populations.

I chose Scheffe's (1959) model for the mixed-model analyses because it does not require the assumption that inter-actions between fixed and random effects be independent of random main effects. Scheffe's (1959) algorithm was used to construct a table of expected mean squares for a balanced four-way factorial design with factors A and B fixed and factors C and D random. This and a similar table for a balanced three-way factorial design with one fixed factor (given in Scheffe 1959) were used to estimate the F-statistics and *P* values associated with the independent variables in each model. Where necessary, the Sattherthwaite approximation (Neter et al. 1985) was used to determine the denominator degrees of freedom for determining the *P* value associated with an *F* value.

To determine the source of the parental treatment effects, I examined six pairs of contrasts: for maternal prezygotic-temperature effects, I compared treatments 1 versus 2 and 3 versus 4 (Fig. 1); for paternal prezygotic-temperature effects, I compared treatments 1 versus 6 and 4 versus 5; for post- zygotic effects, treatments 3 versus 6 and 2 versus 5. These contrasts were first performed using the full fixed-effects model to establish whether or not the overall treatment effect was produced by differences in prezygotic and/or postzygotic temperature (question 1) and if the treatment effect was trans-mitted maternally and/or paternally (question 2). I 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.

In addition, I compared single pairs of treatments using a mixed-model design. For these comparisons I used only two treatments in each analysis (e.g., treatments 1 and 2, treatments 3 and 4, etc.); so there were six analyses. These allowed me to explore whether or not a pre- or postzygotic effect would be observed in a larger population (question 5). I only partially achieved this goal because some of the mixed-model analyses yielded negative *F* statistics, which are difficult to interpret (Searle et al. 1992).

The maternal and paternal prezygotic contrasts were also used to determine whether or not genotypes differ in their intergenerational response to parental temperature, that is, I looked for significant G × (prezygotic) E interactions (question 4). The postzygotic contrasts were not included in these analyses because these postzygotic effects confound intra-generational and intergenerational parental effects.

TABLE 1. ANOVA results for seed weight. Factors in the models are genotype from population D (D), genotype from population H (H), parental temperature (T), and all interactions. The r^2 value for the full model was 0.52. The contrasts show the significance levels for the fixed-model (FM) and mixed-model (MM) analyses. T was the only source variable included in the post-zygotic contrasts. Significance levels: † = $P < 0.10$, * = $P < 0.05$, ** = $P < 0.01$, *** = $P < 0.001$, ns = $P \geq 0.10$.

A. Full model

Source	df	MS	Mixed-Model denominator	Fixed *F*	Mixed *F*
D	3	0.0441	MS_{DH}	2.26†	6.19**
H	4	0.0252	MS_{DH}	1.19	3.27*
DH	12	0.0066	MS_E	0.37	0.37
T	5	0.0635	$MS_{DT} + MS_{HT} - MS_{DHT}$	3.25**	2.20
DT	15	0.0260	MS_{DHT}	1.42	1.61†
HT	20	0.0192	MS_{DHT}	0.93	1.06
DHT	60	0.0166	MS_E	0.88	0.88
error	116	0.0189		—	—

B. Contrasts

		Maternal pre-zygotic				Paternal pre-zygotic				Post-zygotic			
		trt. 1 vs. 2		3 vs. 4		1 vs. 6		4 vs. 5		2 vs. 5		3 vs. 6	
Source	df	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM
T	1	ns	ns	ns	ns	ns	ns	ns	ns	*	ns	†	ns
DT	3	ns	ns	ns	ns	ns	†	†	ns	—	—	—	—
HT	4	ns	ns	ns	ns	ns	†	ns	ns	—	—	—	—
DHT	12	ns	ns	ns	ns	ns	ns	ns	ns	—	—	—	—

RESULTS

Seed Weight

Seed weights varied from approximately 0.8 to 3.1 mg over all treatments. In the fixed-model ANOVA, parental temperature was the only main factor that significantly affected seed weight (Table 1A). Its effect was relatively small, however. In comparing treatments 1 (high) and 4 (low), which differed in both pre- and postzygotic temperatures, there was only a 7% difference in mean seed weight (Fig. 2). In the mixed-model ANOVA, parental genotypes, but not temperature, significantly influenced seed weight (Table 1A).

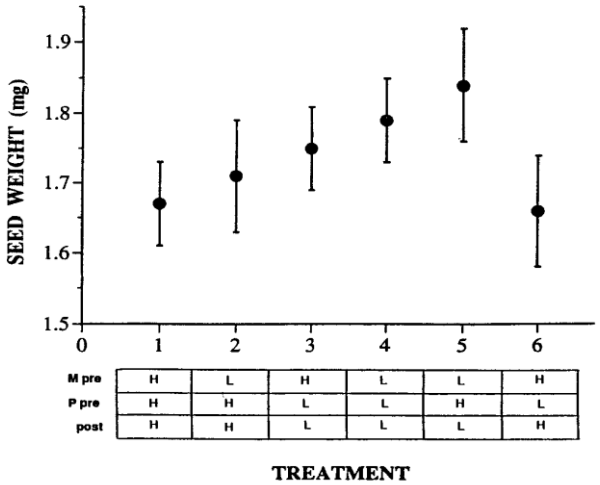

FIG. 2. Mean seed weight (± 2 SE) for each parental temperature treatment. M pre = maternal prezygotic temperature, P pre = paternal prezygotic temperature, post = postzygotic temperature, L = low temperature, and H = high temperature.

The temperature effect was caused by differences in post-zygotic temperature (Fig. 2, Table 1B). Low postzygotic temperature produced heavier seeds (Fig. 2: treatments 2 versus 5, 3 versus 6). Altering prezygotic temperature produced no noticeable change in weights. Several of the genotype × prezygotic temperature interactions were marginally significant (Table 1B).

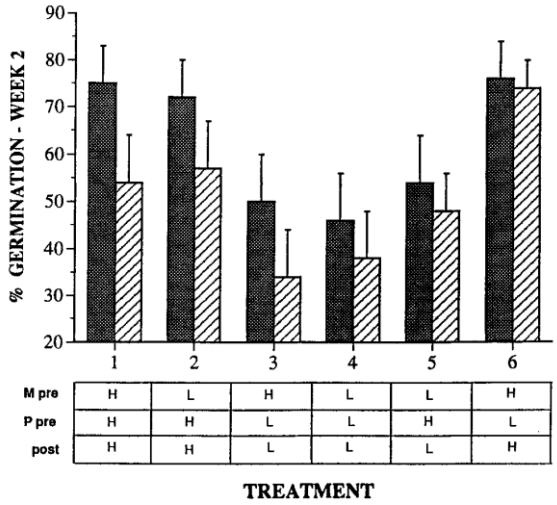

FIG. 3. Mean percent germination (± 2 SE) at the end of week 2, shown for each parental temperature treatment. High offspring temperature is indicated by the darkly shaded bars; low offspring temperature is indicated by the striped bars. L = low temperature, and H = high temperature, M pre = maternal prezygotic temperature, P pre = paternal prezygotic temperature, post = postzygotic temperature.

Germination

Approximately 50% of the seeds germinated in week 1. Germination slowed thereafter (Fig. 3). The fixed-model ANCOVA of both weeks' data showed that parental genotype, temperature, and offspring temperature significantly influenced germination (Table 2A). High parental temperature greatly increased germination (Fig. 3). Averaged over off-spring temperatures, mean germination in treatment 1 (high) surpassed that in treatment 4 (low) by 47% and 53% after weeks 1 and 2, respectively (Fig. 3). Also, high offspring temperature increased germination. These main effects were significant even when seed weight was not included as a covariate. In week 2, the parental temperature effect was significantly influenced by offspring temperature (Table 2A). When the low and high offspring temperature groups were analyzed separately, parental genotypes and temperature still significantly influence germination.

Both paternal prezygotic and postzygotic temperatures contributed significantly to the parental temperature effect in the fixed-model ANCOVA for both weeks and in the mixed-model ANCOVA for week 1 (Table 2B—D). High postzygotic temperature increased germination (Fig. 3: treatments 2 versus 5, 3 versus 6). The effect of paternal temperature depended on maternal prezygotic and postzygotic temperatures. Under low maternal pre- and postzygotic temperatures, high paternal temperature increased germination (Fig. 3: treatment 4 versus 5); under high maternal and postzygotic temperatures, it decreased germination (treatment 1 versus 6). In week 2, a significant main effect of paternal temperature was detected in only one of eight contrasts, which suggests that paternal effects diminish with time (Table 2C—D).

Significant G × (prezygotic) temperature interactions were detected for both weeks in the mixed-model analyses (Table 2B—D: e.g., treatment 4 versus 5). These interactions can be attributed both to change in variance across the treatments for families from population H (Fig. 4A: treatment 4 versus 5) and to change in rank order of families from population D (Fig. 4B: treatment 4 vs. 5).

Growth

Twenty-seven days after I began germinating seeds, offspring had from 0-7 true leaves; the longest leaf for any plant measured 6.5 cm. The fixed- and mixed-model ANCOVAs showed that parental genotypes, temperature, and offspring temperature all significantly influenced offspring size (Table 3A). Temperature influenced leaf area independently of the effects of seed size and germination on leaf area; it also significantly influenced leaf

area when covariates were not included in the model. The realized effect of parental temperature was small, however. Averaged over offspring temperatures, mean leaf area for plants in treatment 1 (high) was only 5% higher than it was for plants in treatment 4 (Fig. 5). By day 43, all parental temperature, genotype, and offspring temperature effects had weakened.

TABLE 2. ANCOVA results for percent germination after one and two weeks. A) Full models, B) Contrasts for germination after one week, C) and D) Contrasts for germination after two weeks for high and low temperature, respectively. Factors in the models are genotype from population D (D), genotype from populaton H (H), parental temperature (T), offspring temperature (O), and all interactions. Seed weight (sd. wt.) was included as a covariate. The r^2 values for the full models at weeks 1 and 2 were 0.74 and 0.76, respectively. The error df = 225 for week 2. Significance levels: † = $P < 0.10$, * = $P < 0.05$, ** = $P < 0.01$, *** = $P < 0.001$, ns = $P \geq 0.10$, a = negative F value for which no significance level could be assigned.

A. Full model

			Week 1			Week 2		
Source	df	Mixed-Model denominator	MS	Fixed F	Mixed F	MS	Fixed F	Mixed F
D	3	MS_{DH}	5.3656	48.30***	30.18***	5.8340	52.93***	25.52***
H	4	MS_{DH}	5.9853	53.88***	33.67***	6.0020	54.46***	26.26***
DH	12	MS_E	0.1778	1.6†	1.6†	0.2286	2.07*	2.07*
T	5	$MS_{DT} + MS_{HT} - MS_{DHT}$	2.5505	22.96***	11.85***	2.9924	27.15***	19.38***
DT	15	MS_{DHT}	0.1000	0.90	1.58	0.1034	0.94	1.54
HT	20	MS_{DHT}	0.1784	1.61†	2.82**	0.1184	1.07	1.76*
DHT	60	MS_E	0.0633	0.57	0.57	0.0671	0.61	0.61
O	1	$MS_{DO} + MS_{HO} - MS_{DHO}$	0.9242	8.32**	11.97*	3.5252	31.99***	21.82**
DO	3	MS_{DHO}	0.0473	0.43	1.98	0.1392	1.26	2.98
HO	4	MS_{DHO}	0.0538	0.48	2.25	0.0689	0.63	1.48
DHO	12	MS_E	0.0239	0.21	0.21	0.0467	0.42	0.42
TO	5	$MS_{DTO} + MS_{HTO} - MS_{DHTO}$	0.0541	0.49	1.35	0.1948	1.77	7.03*
DTO	15	MS_{DHTO}	0.0498	0.45	1.48	0.0469	0.43	1.16
HTO	20	MS_{DHTO}	0.0239	0.22	0.71	0.0212	0.19	0.52
DHTO	60	MS_E	0.0337	0.30	0.30	0.0404	0.37	0.37
sd. wt.	1		0.0144	0.13	0.13	0.0021	0.02	0.02
error	228		0.1111	—	—	0.1102	—	—

B. Contrasts (week 1)

		Maternal pre-zygotic				Paternal pre-zygotic				Post-zygotic			
		trt. 1 vs. 2		3 vs. 4		1 vs. 6		4 vs. 5		2 vs. 5		3 vs. 6	
Source	df	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM
T	1	ns	ns	ns	ns	***	*	†	ns	*	ns	***	**
DT	3	ns	ns	ns	ns	ns	ns	ns	ns	—	—	—	—
HT	4	ns	ns	ns	†	ns	ns	ns	†	—	—	—	—
DHT	12	ns	ns	ns	ns	ns	ns	ns	ns	—	—	—	—

C. Contrasts (week 2—high T)

		Maternal pre-zygotic				Paternal pre-zygotic				Post-zygotic			
		trt. 1 vs. 2		3 vs. 4		1 vs. 6		4 vs. 5		2 vs. 5		3 vs. 6	
Source	df	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM
T	1	ns	ns	ns	ns	ns	ns	ns	ns	***	†	***	*
DT	3	ns	ns	ns	ns	ns	†	ns	ns	—	—	—	—
HT	4	ns	ns	ns	ns	ns	ns	ns	ns	—	—	—	—
DHT	12	ns	ns	ns	ns	ns	ns	ns	ns	—	—	—	—

D. Contrasts (week 2—low T)

		Maternal pre-zygotic				Paternal pre-zygotic				Post-zygotic			
		trt. 1 vs. 2		3 vs. 4		1 vs. 6		4 vs. 5		2 vs. 5		3 vs. 6	
Source	df	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM
T	1	ns	ns	ns	ns	***	a	ns	ns	*	ns	***	a
DT	3	ns	ns	ns	ns	ns	ns	ns	*	—	—	—	—
HT	4	ns	ns	ns	ns	ns	ns	ns	**	—	—	—	—
DHT	12	ns	ns	ns	ns	ns	ns	ns	ns	—	—	—	—

The treatment effect at 27 days was caused primarily by postzygotic temperature differences and marginally by differences in paternal temperature (Table 3B). High postzygotic and paternal temperatures yielded plants with greater leaf area (Fig. 5: treatments 2 versus 5, 1 versus 6). Additionally, genotypes differed in their response to maternal prezygotic temperature at 27 days (Table 3B: treatments 1 versus 2, 3 versus 4). Maternal temperature changed the rank order of families between treatments (Fig. 6: treatment 3 versus 4).

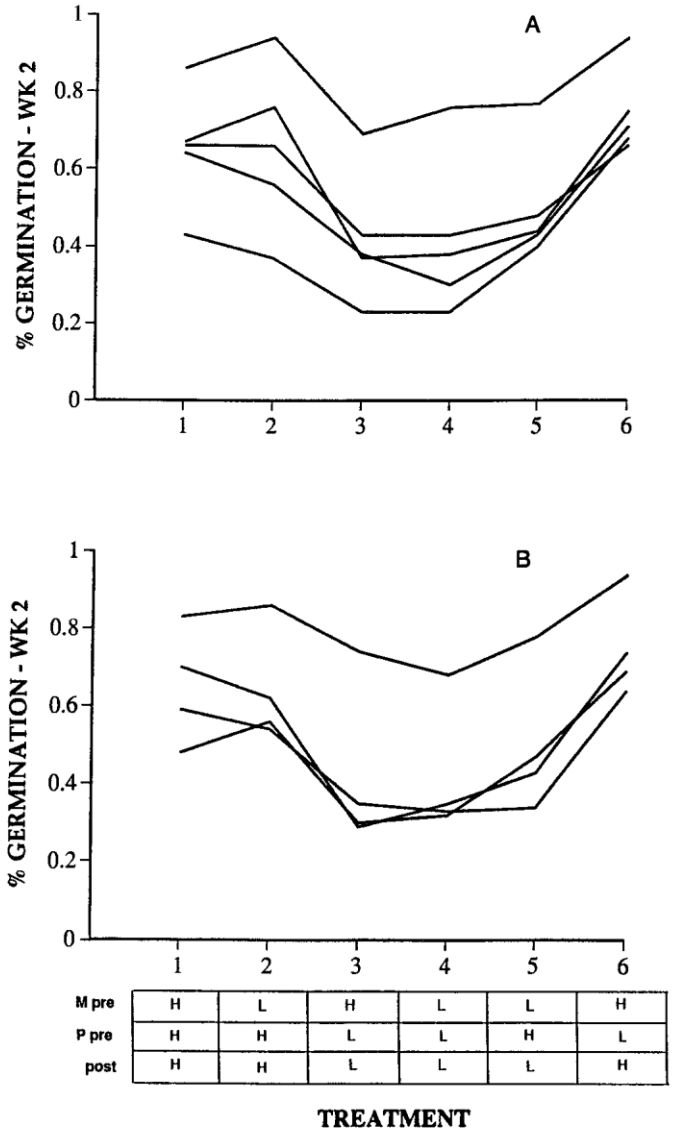

FIG. 4. Reaction norms of germination at two weeks for (A) offspring of the five genotypes from population H and (B) offspring of the four genotypes from population D grown under the same six temperature treatments. L = low temperature, and H = high temperature, M pre = maternal prezygotic temperature, P pre = paternal prezygotic temperature, post = postzygotic temperature.

Onset of Flowering

Seventy-three days after I had begun seed germination, 45% of the experimental plants had produced flowering spikes (Fig. 7). Both the fixed- and mixed-model analyses showed that parental genotype, temperature, and offspring temperature significantly influenced onset of flowering and did so independently of the significant correlation between flowering and juvenile traits, seed weight and germination (Table 4). Low temperature accelerated flowering. Averaged over offspring temperatures, mean percent plants flowering for treatment 4 was 47% higher than the mean for treatment 1 (Fig. 7). Offspring temperature produced an effect opposite to that of parental temperature; high offspring temperature accelerated flowering.

Fixed-model contrasts showed that maternal and paternal prezygotic and postzygotic temperature treatments all contributed to the significant treatment effect (Table 4B). Low postzygotic and paternal temperatures accelerated flowering, whereas low maternal prezygotic temperature delayed flowering (Fig. 7). The sources of the treatment effect could not be detected in the mixed-model contrasts, possibly because no one source was strong enough to be detected after I had subset the data for the mixed-model contrasts. Neither the fixed- nor mixed-model contrasts detected any significant G × (prezygotic) temperature interactions.

DISCUSSION

Characterization of Parental Effects

Parental temperature affected all the life-history traits that I examined in *P. lanceolata.* Low temperature increased seed weight, retarded germination and growth rates, and accelerated flowering when compared to the effects of high temperature. The relative changes in seed weight and growth were small, less than 8%. Consequently, it seems unlikely that parental temperature effects would influence individual fitness strongly via these traits in natural populations. Many experimental studies have shown that survival and reproduction, components of fitness, are correlated with seed weight (e.g., Gross 1984; Stanton 1984; Winn 1985; Wulff 1986c). However, seed weights varied from 30% to more than 400% in these studies. The studies provide little evidence that an 8% change in seed weight would alter fitness.

In contrast, the effects of parental temperature on germination and onset of flowering were large. Low parental temperature increased germination and accelerated flowering by approximately 50%. Previous studies examining the fitness consequences of germination and flowering times suggest that such differences are likely to produce biologically meaningful effects in natural populations (e.g., Rathcke and Lacey 1985; Biere 1991a). For example, a few day's difference in emergence time can strongly alter biomass and fecundity in *P. lanceolata* (Miller 1987). If we can extrapolate my results to natural populations, then parental temperature should affect fitness through its effect on germination and flowering.

TABLE 3. ANCOVA results for leaf area at approximately 27 and 43 d. Factors in the models are genotype from population D (D), genotype from population H (H), parental temperature (T), offspring temperature (O), and all interactions. Seed weight (sd. wt.) and percent germination at two weeks (pct. germ.) were included as covariates. The r^2 values for the full models at 27 and 43 d were 0.76 and 0.64, respectively. Denominators used for the calculations of the mixed-model F statistics are shown in Table 2. The error df = 204 for 43 d. Significance levels: † = $P < 0.10$, * = $P < 0.05$, ** = $P < 0.01$, *** = $P < 0.001$, ns = $P \geq 0.10$, a = negative F value for which no significance level could be assigned.

A. Full model

		27 days			43 days		
			Fixed	Mixed		Fixed	Mixed
Source	df	MS	F	F	MS	F	F
D	3	0.4914	61.38***	311.82***	0.7588	24.77***	21.00***
H	4	0.0196	2.45*	12.45***	0.0671	2.19†	1.86
DH	12	0.0016	0.20	0.20	0.0361	1.18	1.18
T	5	0.0368	4.60***	8.56†	0.0738	2.41*	3.14
DT	15	0.0046	0.58	0.52	0.0315	1.03	0.92
HT	20	0.0087	1.09	0.97	0.0264	0.86	0.77
DHT	60	0.0090	1.12	1.12	0.0344	1.12	1.12
O	1	0.9059	113.16***	55.58**	0.0909	2.97†	0.72
DO	3	0.0157	1.97	6.53**	0.1224	4.00**	8.78**
HO	4	0.0030	0.37	1.23	0.0172	0.56	1.23
DHO	12	0.0024	0.30	0.30	0.0139	0.46	0.46
TO	5	0.0031	0.39	0.34	0.0353	1.15	2.07
DTO	15	0.0108	1.35	1.82†	0.0285	0.93	1.15
HTO	20	0.0044	0.56	0.75	0.0198	0.65	0.80
DHTO	60	0.0059	0.74	0.74	0.0247	0.81	0.81
sd. wt.	1	0.0711	8.89**	8.89**	0.2678	8.74**	8.74**
germ	1	0.0371	4.63*	4.63*	0.2177	7.11**	7.11**
error	203	0.0080	—	—	0.0306	—	—

B. Contrasts (27 d)

		Maternal pre-zygotic				Paternal pre-zygotic				Post-zygotic			
		trt. 1 vs. 2		3 vs. 4		1 vs.6		4 vs. 5		2 vs. 5		3 vs. 6	
Source	df	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM
T	1	ns	ns	ns	a	†	ns	ns	a	**	ns	ns	a
DT	3	ns	ns	ns	ns	ns	ns	ns	ns	—	—	—	—
HT	4	*	†	ns	ns	ns	ns	ns	ns	—	—	—	—
DHT	12	ns	ns	*	**	ns	ns	ns	ns	—	—	—	—

Postzygotic temperature effects are stronger and more pervasive than are prezygotic effects in *P. lanceolata.* Postzygotic temperature influenced all traits examined, whereas prezygotic temperature significantly influenced only germination and onset of flowering. Also, where the prezygotic effect differed from the postzygotic effect, as it did for germination and flowering, the postzygotic effects prevailed, i.e., the overall effect resembled the postzygotic effect. The data suggest that a prezygotic effect may moderate but does not change a postzygotic effect. Robertson et al. (1962) observed a similar pattern in peas.

The patterns of response to parental temperature with respect to seed weight and germination in *P. lanceolata* resemble those reported for other species grown in controlled environments. Decreasing temperature during seed development appears to slow maturation such that mature seeds are larger (Stearns 1960; Robertson et al. 1962; Siddique and Goodwin 1980, Wulff 1986a) and germinate later (Koller 1962; Sawhney and Naylor 1979, Siddique and Goodwin 1980; Gutterman 1980/81, 1983). This general effect may explain why seeds from woodland populations are larger than are seeds from field populations in some species (*e.g.,* Winn 1985). Postzygotic temperatures are likely to be lower in shaded than in open habitats.

When Alexander and Wulff (1985) first reported the existence of parental effects in *P. lanceolata,* they ascribed the effects to the mother. My data suggest that this may be premature. Prezygotic effects were more often transmitted paternally than maternally. Maternal prezygotic temperature influenced flowering, whereas paternal temperature influenced early germination, growth, and flowering. The postzygotic effects may have been mediated by the maternal parent. However, temperature could instead have affected offspring embryonic development directly.

Historically, the term "maternal effects" has been used to embrace all parental effects regardless of the pathway of transmission (maternal versus paternal). This may create problems because different types of "maternal" effects may produce different evolutionary consequences (Lande and Kirkpatrick 1990). Lumping all types of parental effects together obscures these differences. Paternal effects have traditionally been assumed to be minimal (Roach and Wulff 1987), even though experimental designs often have not been capable of detecting them (Milligan 1991). My data and those of others (e.g., Freeman and Vitale 1985; Schlichting 1986; Bertin 1988, Young and Stanton 1990; Richardson and Stephenson 1992) suggest that paternal transmission should not be ignored. Collectively the studies suggest restricting the use of "maternal effects" to cases in which an effect is known to be maternally transmitted. "Parental effects" can be used to embrace both maternal and paternal effects and those effects for which the pathway of transmission is unknown. This practice would at least help remind us that our knowledge of transmission pathways is quite limited.

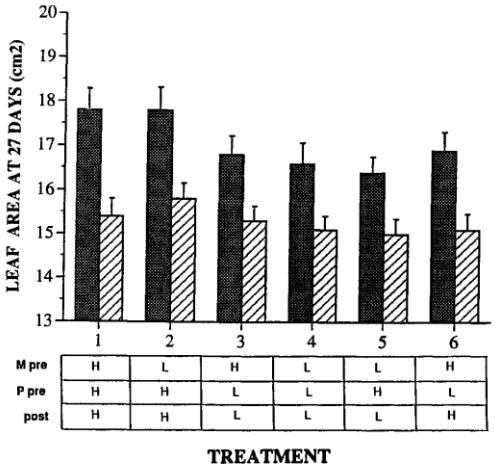

FIG. 5. Mean estimated leaf area (± 2 SE) at approximately 27 days shown for each parental temperature treatment. High offspring temperature is indicated by the the darkly shaded bars; low offspring temperature is indicated by the striped bars. L = low temperature, and H = high temperature, M pre = maternal prezygotic temperature, P pre = paternal prezygotic temperature, post = postzygotic temperature.

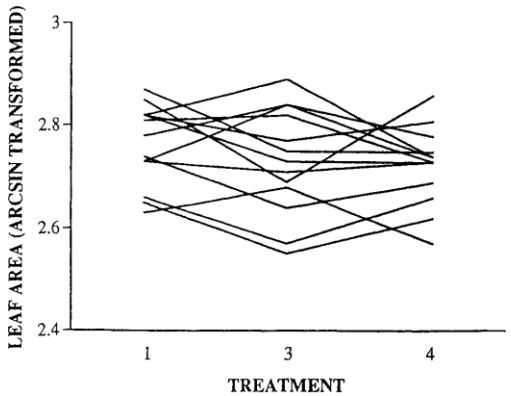

FIG. 6. Reaction norms of estimated leaf area at 27 days for offspring of a sample of 13 different parental crosses for three of the six temperature treatments. Pre- and postzygotic temperatures were all high in treatment 1; they were all low in treatment 4. Treatment 3 differed from treatment 4 in that the maternal parent was grown under high prezygotic temperature.

Additionally, biologists have often equated a "maternal" effect with an intergenerational effect. This may be inappropriate when postzygotic effects are involved. In the absence of additional information about the mechanism by which the environment produces a postzygotic effect, one can correctly conclude that an intergenerational effect (e.g., intergenerational phenotypic plasticity) exists only if one can demonstrate prezygotic effects or postzygotic effects that are, themselves, transmitted across generations. The observed prezygotic effects in *P. lanceolata* are unambiguously intergenerational. Intergenerational effects have also been detected in several other plant species (e.g., Rowe 1964; Miao et al. 1991b; Schneeberger and Cullis 1991; Platenkamp and Shaw 1993). In these cases experiments were carried over at least three generations.

There are several hypothetical mechanisms by which the environment could produce an intergenerational effect. First, the environment could affect a gamete's probability of uniting with another gamete to produce an offspring. Recent studies have shown that the parental environment, most often nutrient availability, can affect number of seeds sired (e.g., Young and Stanton 1990; Lau and Stephenson 1993), pollen viability (e.g., Freeman and Vitale 1985), pollen size (e.g., Lau and Staphenson 1993) and pollen germination and growth rate (e.g., Schlichting 1986; Bertin 1988). In some cases, the response of pollen to the parental environment differs among genotypes or lineages (e.g., Gawel and Robacker 1986; Elgersma et al. 1989). Whether or not the environment is altering the fitness of a gametophyte (or gamete) by changing either's environment or genome, it is effectively influencing gametophytic (or gametic) selection, which would manifest itself in the next generation.

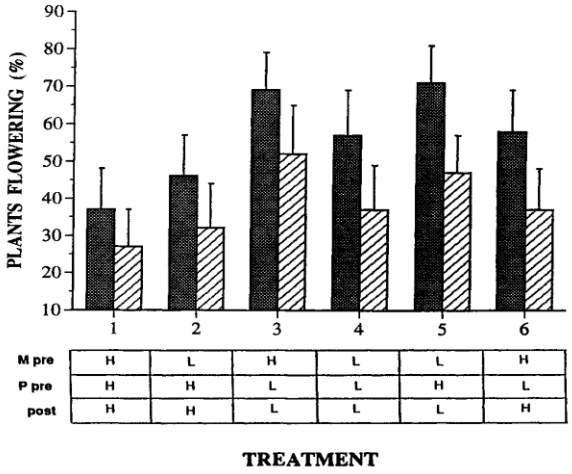

FIG. 7. Mean percent of offspring having reproductive spikes (± 2 SE) at 73 days shown for each parental temperature treatment. High offspring temperature is indicated by the the darkly shaded bars; low offspring temperature is indicated by the striped bars. L = low temperature, and H = high temperature, M pre = maternal prezygotic temperature, P pre = paternal prezygotic temperature, post = postzygotic temperature.

TABLE 4. ANCOVA results for percent plants with reproductive spikes at 73 d. Factors in the models are genotype from population D (D), genotype from population H (H), parental temperature (T), offspring temperature (O), and all interactions. Seed weight (sd. wt.) and percent germination at two weeks (germ) were included as covariates. The r^2 value for the full model = 0.70. Denominators used for the calculations of the mixed-model F statistics are shown in Table 2. Significance levels: † = $P < 0.10$, * = $P < 0.05$, ** = $P < 0.01$, *** = $P < 0.001$, ns = $P \geq 0.10$, a = negative F value for which no significance level can be assigned.

A. Full model

Source	df	MS	Fixed F	Mixed F
D	3	6.1311	28.49***	14.00***
H	4	3.7066	17.23***	8.46**
DH	12	0.4380	2.04*	2.04*
T	5	1.4985	6.96**	8.81**
DT	15	0.1959	0.91	0.85
HT	20	0.2035	0.95	0.89
DHT	60	0.2293	1.07	1.07
O	1	5.0688	23.56***	10.77*
DO	3	0.2529	1.18	1.45
HO	4	0.3926	1.82	2.24
DHO	12	0.1749	0.81	0.81
TO	5	0.2058	0.96	1.28
DTO	15	0.1537	0.71	0.98
HTO	20	0.1638	0.76	1.05
DHTO	60	0.1562	0.73	0.73
sd. wt.	1	1.5667	7.28**	7.28**
germ	1	0.5802	2.70†	2.70†
error	208	0.2152		

B. Contrasts

		Maternal pre-zygotic				Paternal pre-zygotic				Post-zygotic			
		trt. 1 vs. 2		3 vs. 4		1 vs. 6		4 vs. 5		2 vs. 5		3 vs. 6	
Source	df	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM	FM	MM
T	1	ns	ns	*	ns	*	ns	ns	ns	*	a	*	ns
DT	3	ns	ns	ns	ns	ns	ns	ns	ns	—	—	—	—
HT	4	ns	ns	ns	ns	ns	ns	ns	ns	—	—	—	—
DHT	12	ns	ns	ns	ns	ns	ns	ns	ns	—	—	—	—

Alternatively, the postzygotic environment could directly alter the genome of the embryonic sporophyte, thereby affecting sporophytic selection. Environmentally induced heritable changes have been observed in several species (Durrant 1962; Cullis 1977, 1981; Schneeberger and Cullis 1991). The environment can alter nuclear DNA (Schneeberger and Cullis 1991; Matzke and Matzke 1993), and it may conceivably also alter organelle inheritance through either the mother or father. Corriveau and Coleman (1988) found no evidence for paternal transmission of plastids in *P. lanceolata,* however, recent data suggest that 20-50% of the angiosperms exhibit biparental inheritance of plastid DNA (Tilney-Bassett 1978; Sears 1980; Corriveau and Coleman 1988).

Finally, the environment may produce nongenetic changes in the embryo, endosperm, and/or pollen that affect offspring phenotype. These changes might explain the results of many studies reporting significant effects of parental environment on seed and seedling characters (e.g., Rowe 1964; Roach and Wulff 1987; Stratton 1989; Aarssen and Burton 1990; Biere 1991a,b; Miao et al. 1991 a,b; Schmitt et al. 1992; Philippi 1993; Platenkamp and Shaw 1993). The evolutionary implications of nongenetic changes are less clear. These changes have typically been hypothesized to be very short-lived, both within the offspring generation and over generations (Roach and Wulff 1987). However, if a nongenetic change persists over multiple generations, as will be discussed later, and affects fitness, its evolutionary effect may be profound.

The alternative mechanisms by which the environment may produce an intergenerational effect leads us to consider another aspect of parental effects that has not been adequately explored. This is the degree to which the parental environment directly affects adult phenotype in offspring. A few studies have shown that parental effects can persist into the adult phase of a progeny's life to influence yield and fitness components, for example, growth rate, flowering phenology, seed set (e.g., Aksel 1977; Singh and Murty 1980; Alexander and Wulff 1985; Hanson and Conde 1985; Wulff 1986a,b; Miao et al. 1991a,b). My results show that parental temperature affected adult leaf area and onset of flowering.

The question is whether parental effects influence these adult traits directly or only indirectly because of their influence on juvenile traits. Parental effects affect adult traits via both pathways in *P. lanceolata* (see also Wulff and Bazzaz 1992). Postzygotic temperature strongly influenced seed weight, and vegetative growth and onset of flowering were significantly correlated with seed weight. However, even after removing the correlations with seed weight and germination, parental temperature still influenced growth and onset of reproduction. It seems likely that either gametophytic selection occurred or that the environment modified genes controlling adult traits. Recent studies have shown that parental environment can affect gene amplification and gene activity in *Petunia,* maize, tobacco, and flax (Schneeberger and Cullis 1991; Matzke and Matzke 1993).

Studies showing that parental effects are expressed in field conditions, that is, in natural populations, are scarce. Individuals have almost always been grown in greenhouses and growth chambers (exceptions are Schmitt et al. 1992; Biere 1991a). In the absence of field studies, one can still use mixed-model analyses to determine if the parental effects observed with a small sample of genotypes would likely manifest themselves in naturally larger populations. My results suggest that they would for germination, growth, and onset of reproduction. The phenotypes of these traits were significantly affected by parental temperature treatment either alone or in interaction with genotype. Surprisingly, the data provide no evidence that seed size would be affected in natural populations, even though temperature strongly affected seed weight in the fixed-model analysis of this and Alexander's and Wulff's (1985) experiment.

My mixed-model analyses also showed that genotypes differed in their germination and growth responses to parental prezygotic temperature. Prezygotic temperature influenced the rank order of families for germination and growth. For germination, it also affected the mean differences among families. A few other studies have detected G x (ancestral) E interactions (e.g., Durrant and Timmis 1973; Alexander and Wulff 1985; Platenkamp and Shaw 1993). However, because pre- and postzygotic effects were combined in these studies, direct comparisons with my data are not possible. These genotype-specific responses to parental temperature could be viewed as intergenerational phenotypic plasticity, but alternatively they may reflect gametophytic selection.

Potential Consequences of Parental Effects

Recent theoretical (e.g., Riska et al. 1985; Gimelfarb 1986; Kirkpatrick and Lande 1989, 1992; Lande and Price 1989; Lande and Kirkpatrick 1990) and empirical studies (e.g., Antonovics and Schmitt 1986; Janssen et al. 1988; Kaplan 1991; Mousseau 1991; Mousseau and Dingle 1991; Reznick 1991; Sinervo 1991; Schmitt et al.,1992; Platenkamp and Shaw 1993; Schluter and Gustafsson 1993) suggest that parental effects may influence the evolution of life-history traits in a variety of organisms. The evolutionary consequences are still poorly understood, however. I chose to examine the intergenerational effects of temperature because temperature, on average, changes relatively predictably both temporally and spatially, on small and large scales. Within populations of *P. lanceolata*, on average, it increases through the flowering season (Teramura 1978). Also, shaded populations are subjected to cooler temperatures than are neighboring unshaded populations (Teramura 1978). On a large scale, mean temperature during the growing season decreases with increasing latitude and altitude. Therefore, any parental effects mediated by temperature should be fairly predictable and should persist over multiple generations. The evolutionary consequences are likely to differ from those produced by parental effects that are caused by an environmental factor that varies unpredictably over space and time.

Obviously my experiment cannot mimic the natural environment, or even the temperature changes to which plants are naturally subjected. In real populations temperatures vary more than in a growth chamber. They are also less predictable. My experiment can, however, suggest how real temperature patterns might affect natural populations and can suggest hypotheses that can later be tested in a more natural setting.

The data suggest that temperature is involved in two kinds of parental effects: those that are genetically based and those that are environmentally induced. Evidence for genetically based effects comes from three observations about the onset of flowering in *P. lanceolata.* First, onset of flowering is partially genetically controlled (Primack and Antonovics 1981; Wolff 1987; Wolff and van Delden 1978; data presented here). Second, temperature, on average, increases during the flowering season in natural populations (Teramura 1978). Third, I observed a positive correlation between parental temperature and offspring flowering time. Together these data suggest that parents influence the flowering time of their progeny not only by the direct transmission of Mendelian genes controlling flowering time but also by determining the temperature environment during flowering and seed development. In quantitative genetic terms, a single character with a partly Mendelian basis exerts a parental effect on itself in the next generation. This type of effect differs from that produced by other environmental factors that change spatially throughout the population but change independently of the genotypes constituting the population.

This genetically based temperature effect should accelerate any response to selection among families for flowering time in *P. lanceolata.* Here it is useful to imagine a hypothetical population comprised of three families that genetically differ in flowering time (Fig. 8a). In my experiment the main effect of low parental temperature was to accelerate flowering in the offspring. This result suggests that early-flowering families (e.g., family A in Fig. 8a) should produce offspring that flower even earlier, and/or that late-flowering families (e.g., family C) should produce offspring that flower even later. Such an effect should increase the positive assortative mating within the population and increase the mean difference in flowering times among families, facilitating selection among families and accelerating the response to selection.

In general, whether a parental effect accelerates or retards the response to selection depends on both the extent to which offspring phenotype is determined by the parental effect and by the direction of the parental effect. If offspring phenotype is completely determined by the mother, then the response to selection will be retarded because one is actually selecting among mothers and not among offspring (Antonovics and Schmitt 1986; Mazer 1987). If, however, offspring phenotype is determined both by its own genotype and by a parental effect, a situation that may be more common, then the direction of the parental effect will determine the rate of the response. For example, when low parental temperature accelerates flowering, as in *P. lanceolata,* parental temperature should reduce the phenotypic overlap among families (Fig. 8a), thereby accelerating a response to

selection. Alternatively, when low temperature retards flowering, one would expect an increase in phenotypic overlap among families, thereby retarding the response to selection.

Environmentally induced parental temperature effects are produced by changes in temperature regime that are associated with habitat change. Such effects could be produced by changes in shading, altitude, or latitude. In all cases, temperature changes independently of the genotypes constituting the populations. In terms of flowering time in our hypothetical population, lowering temperature should shift the phenotypic distributions for flowering time to the left for all families (Fig. 8b). It is not clear that this effect would alter the response to selection for a particular character, like flowering, in a population, because all families are affected. However, such shifts could alter the gene flow between neigh-boring shaded and unshaded populations.

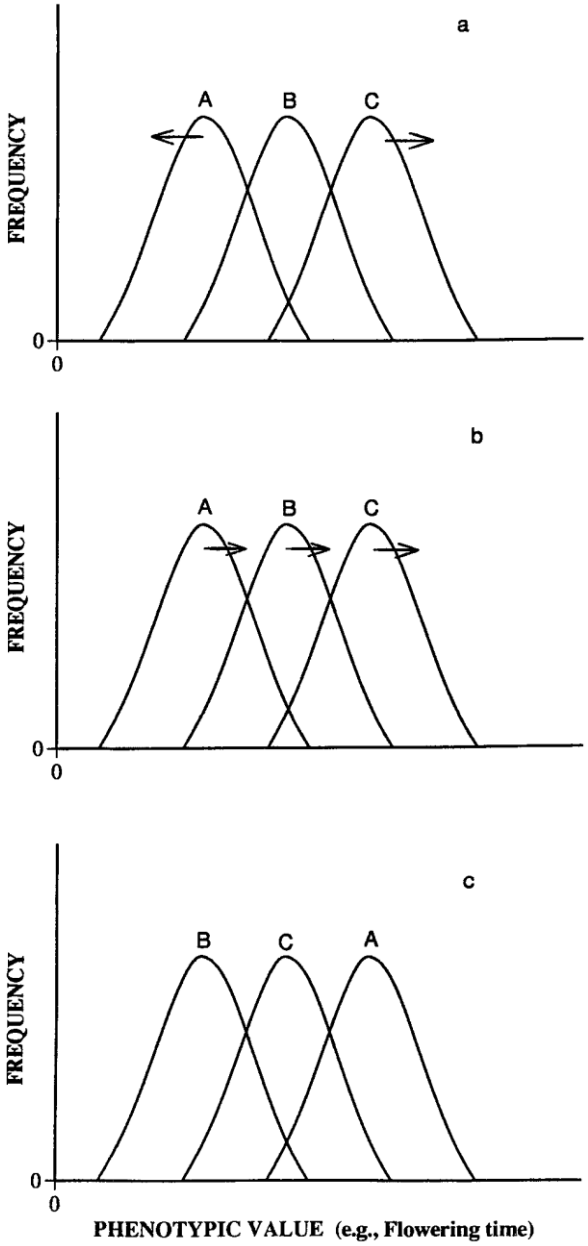

FIG. 8. The phenotypic distributions of three genetically different families (A, B, and C) that constitute a hypothetical population, and the influence of parental temperature effects on these distributions: (a) A genetically based temperature effect shifts the phenotypic distribution of family A to the left and/or that of family C to the right, thereby increasing the mean phenotypic differences among families; (b) An environmentally induced temperature effect shifts the distributions of all families to the right (shown) or left (not shown); and (c) An environmentally induced temperature effect changes the rank order of phenotypic distributions for the families from those shown in Figure 8a. See Discussion for further details.

Environmentally induced temperature effects could have a stronger evolutionary impact when genotypes differ in their response to parental temperatures. Such genotypic-specific responses could change the rank order of families and/or the among-family variance for fitness traits across environments. When rank order changes across environments, then different families could be evolutionary favored in the different environments. Also, the response to selection among families will be rapid in environments producing the large among-family variance but slow in environments producing the small variance.

At this point, my data provide little evidence that the intergenerational, environmentally induced temperature effects influence the evolution of *P. lanceolata.* Differences among and rank orders of families for treatments 1 and 4 were nearly equivalent in spite of the significant G × (prezygotic) E interactions in germination and growth (e.g., see Fig. 5). The reason is that postzygotic temperature more strongly influenced offspring germination and growth than did prezygotic temperature. However, Alexander and Wulff (1985) did detect significant intergenerational G × E interactions when pre-and postzygotic effects were combined, which suggests that intergenerational postzygotic interactions need further study.

In conclusion, evidence suggests that parental temperature effects could alter the direction and rate of evolutionary change in *P. lanceolata.* First, the magnitude of parental influence on offspring phenotype is large for some life-history traits. Second, the data suggest how acceleration, retardation, and directional change in evolution could occur. Parental effects could theoretically produce both genetic and nongenetic phenotypic changes in offspring. Even "short-lasting" non-genetic effects could persist long enough to influence the evolutionary divergence or convergence of populations found in habitats with different temperature regimes.

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