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THE GENETIC ARCHITECTURE OF PLASTICITY TO DENSITY IN *IMPATIENS CAPENSIS*

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Abstract.—Plant responses to crowding may be mediated by resource availability and/or by a specific environmental cue, the ratio of red:far red wavelengths (R:FR) perceived by phytochrome. This study examined the contribution of phytochrome-mediated photomorphogenesis to genetic variation in plastic responses to density in the annual plant *Impatiens capensis*. Inbred lines derived from open and woodland populations were grown under low density, high density, and high density with selective removal of FR wavelengths to block phytochrome-mediated perception of neighbor proximity. Genetic variation in plasticity to density and to the R:FR cue was detected for several traits. Plants grown at high density displayed increased internode elongation; decreased branch, flower, and node production; increased meristem dormancy; and decreased leaf area and specific leaf weight compared to plants grown at low density. Stem elongation responses to density were suppressed when phytochrome perception was blocked at high density. For these phytochrome-mediated traits, a genotype's plasticity to density was strongly correlated with its response to R:FR. Phytochrome-mediated traits were tightly correlated with one another, regardless of the density environment. However, the responses to density of meristem allocation to branching and leaf traits were less strongly phytochrome-mediated. These traits differed in patterns of plasticity, and their genetic correlations often differed across environments. In particular, genetic trade-offs involving meristem allocation to branching were expressed only at low density. The observed density dependence of phenotypic and genetic correlations implies that indirect selection and the potential for correlated response to selection will depend upon the competitive environment. Thus, the differential sensitivity of characters to the R:FR cue can influence the evolution of integrated plastic responses to density.

Key words.—Allocation, density-dependent character correlations, environmental heterogeneity, genetic correlations, phytochrome, red:far red, trade-offs.

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Phenotypic plasticity often involves concurrent responses of several characters to environmental variation. The integration of these plastic responses will determine the environment-specific phenotype that is exposed to selection. The degree of integration will also determine how phenotypic and genetic correlations change with environmental variation thus affecting the expression of life-history trade-offs (Stearns et al. 1991) and the evolutionary potential for response to selection (Lande and Arnold 1983). It is therefore of interest to examine the mechanisms underlying integrated plasticity to ecologically relevant environmental variation. Often, the environments encountered by an organism vary concurrently in several factors. For example, plant responses to crowding or vegetation shade may be mediated either by light availability or by light spectral quality. In such cases, shared sensitivity of different characters to a common environmental factor such as light quality may provide a possible mechanism for integrated plastic responses. This hypothesis leads to the prediction that characters whose plasticity is mediated by a common environmental factor will be more strongly integrated than characters that do not share such sensitivity.

Integrated phenotypic plasticity can occur in response to resource availability (Schmitt 1993; Sultan and Bazzaz 1993a,b,c; Pigliucci and Schlichting 1995; Pigliucci et al. 1995) and in response to environmental cues (Schmitt and Wulff 1993; Dudley and Schmitt 1995; van Hinsberg 1996; van Tienderen and van Hinsberg 1996; van Hinsberg and van Tienderen 1997). It has been argued that plasticity to resources may simply be a passive response to resource limitation and is unlikely to be adaptive, although resource limitation may affect many characters concurrently resulting in apparent "integration" (Schmalhausen 1949; Smith-Gill 1983; Thompson 1991; Schlichting and Pigliucci 1995). In contrast, the activation of specific signal transduction pathways in response to an environmental cue may enable an individual to anticipate a change in the environment and respond to the changing environment in a timely manner (Levins 1968). If several traits are mediated by the same signal transduction pathway, a specific cue may result in an integrated plastic response that may be shaped by selection.

The ability of a single environmental cue, such as light quality, to elicit plastic responses in several different characters has important consequences for the evolution of suites of plastic characters and developmental integration (Schlichting and Levin 1986; Schlichting 1989; van Tienderen 1990; Schlichting and Pigliucci 1995, 1998; van Tienderen and van Hinsberg 1996). If the plastic responses of many characters share a common physiological mechanism, then they are also likely to share a common genetic mechanism. A shared genetic mechanism would influence genetic correlations among characters involved in plastic responses to density and thereby constrain how this suite of characters evolves in response to selection (Lande and Arnold 1983; Schlichting 1989; Stearns et al. 1991). The degree of integration of plastic responses will determine the constancy of character correlations across different environments. If genetic variation for plasticity exists and if different characters have different degrees of plasticity, then the correlations between characters can change with the environment (Stearns et al. 1991), as observed in several species (e.g., Schlichting 1989; Mazer and Schick 1991; Thomas and Bazzaz 1993). The degree to

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which different characters differ in their plastic responses, and consequently the degree to which correlations among characters change with the environment, could depend on the extent to which the plasticities of different characters are mediated by the same mechanism.

In particular, patterns of character integration can influence the expression of trade-offs in different environments. For example, allocation of meristems either to vegetative or reproductive tissue necessarily imposes a trade-off between these two life-history options, because allocation of a meristem to reproductive tissue precludes further vegetative growth from that structure (Watson 1984; Geber 1990). However, such a developmental constraint will only result in a negative genetic correlation between the two characters if genetic variation in resource allocation is high relative to variation in resource acquisition. High variation in resource acquisition among genotypes can cause positive genetic correlations even among allocation characters that are tightly developmentally linked (van Noordwijk and de Jong 1986; Houle 1991; Stearns et al. 1991). If plasticity alters the relative expression of genetic variation for allocation or assimilation traits across different environments, it may cause environmental changes in expression of life-history and developmental trade-offs.

Plasticity of plants to density provides an ideal system for examining integrated plastic responses to cues and resources and the effects of those responses on genetic architecture. Many plants show great plasticity in morphology and allocation in response to crowding (e.g., Schmitt et al. 1986; Geber 1989; Weiner and Thomas 1992; Thomas and Bazzaz 1993). This response could be due to the combined effects of reduced light availability—a resource—and reduced ratio of red to far red light (R:FR) within vegetation canopies in high density stands—a cue of competitive conditions. Plastic responses to density can be mediated by the R:FR perceived by the phytochrome family of photoreceptors (Smith 1995). A low R:FR acts as a cue that can accurately predict present or future competitive environments under some conditions, because light filtered through or reflected from vegetation is deficient in red light (Smith 1982; Casal and Smith 1989; Schmitt and Wulff 1993). Low R:FR can elicit a photomorphogenic response, referred to as the shade-avoidance response, even before direct competition is present (Ballaré et al. 1990; Smith et al. 1990; Smith and Whitelam 1990). Manipulation of R:FR independently from density allows one to determine the extent to which plastic responses to density are mediated by phytochrome, which uses R:FR as its cue, and to what extent they are mediated by other aspects of the density environment, such as resource availability (Ballaré et al. 1991; Schmitt and Wulff 1993; Dudley and Schmitt 1995, 1996). Such manipulation thereby offers a system in which to investigate how shared sensitivity of traits to an environmental cue (R:FR) influences the integration of plasticity to an ecologically important environmental factor (density).

Here we describe an investigation of the genetic architecture of responses to density in the annual *Impatiens capensis* Meerb. (Balsaminaceae). In this study, inbred lines were grown under different density and light environments in a quantitative genetic experimental design. This design allowed us to dissect genetic variation in plasticity to density into components mediated by light availability and by the R:FR cue perceived by phytochrome. In this study we address the following questions. What is the nature of genetic variation for plasticity to density, light resources, and R:FR? In particular, are responses to density for certain traits similar to their responses to R:FR, suggesting phytochrome mediation of plasticity to density? How do responses to a common R:FR cue influence the integration of plastic responses to density? How do correlations among resource-mediated and cue-mediated traits change with density?

Materials and Methods

The inbred lines of the annual *I. capensis* Meerb. (Balsaminaceae) used for this study were originally collected from two populations in the Haffenreffer Reserve of Brown University in Bristol, Rhode Island (Schmitt 1993). *Impatiens capensis* has a mixed mating system of cleistogamous and chasmogamous flower production, and it is highly selfing under natural conditions. The sunny population is located in a clearing where seedlings emerge in densities up to 3000 plants/m^2, whereas the woodland population grows under a deciduous oak-hickory canopy where maximal seedling densities are 450 plants/m^2 (Dudley and Schmitt 1995). The populations are within 1 km of each other. The lines were maintained by self-pollination under uniform conditions in the greenhouse for six generations. Seeds were collected from up to 20 lines from each population during November 1995 and were stratified at 4°C for four months in water before planting. Up to 20 germinants from each line were planted individually into 2.54-cm pine cells filled with Metromix 350. Each plant, therefore, had equivalent below-ground resources while the above-ground light environment was manipulated independently. Because of unusually low germination success due to refrigeration failure and a fungal pathogen, only 11 lines from the sunny population and nine lines from the woodland population could be used, giving a total of 286 seedlings.

We manipulated density and R:FR in three experimental treatments, referred to as "low density," "high density," and "blocked cue." In the low-density treatment, the pine cells were arranged at a density of 90 plants/m^2 in pine cell racks, and the racks were covered with neutral shade cloth that reduced photosynthetically active radiation (PAR) to 35–40% that of ambient light. In the high-density treatment, pine cells were arranged at a density of 1190 plants/m^2, and the racks were covered with the same neutral shade cloth. In both the high- and low-density treatments, the R:FR above the canopy remained that of ambient light (1.1 before canopy growth). In the blocked-cue treatment, plants were arranged at the same density as the high-density treatment, but the R:FR cue was blocked by artificially raising the R:FR by placing the racks beneath a 0.5 × 0.8 m Plexiglas tray containing 6 L of a 30 g/L copper sulfate solution held within six 1-L clear plastic bags (Ballaré et al. 1991; Dudley and Schmitt 1996). This solution filtered out far red light, increasing the R:FR to eight times that of ambient light (8.8 before canopy growth) and decreasing PAR to 35–40% that of ambient light. Therefore, PAR above the canopy was the

same for the three treatments. The two high-density treatments provide a contrast in which light availability was equivalent but the R:FR cue differed; the neutral-shade treatment had reduced R:FR as the canopy formed and the blocked-cue treatment had a R:FR that remained high. Comparing the high- and low-density treatments shows the response to density itself. These three treatments, therefore, distinguish between a response to density that is due to a combined reduction in light availability and reduced R:FR from that which is due to only a change in the R:FR cue perceived by phytochrome.

Seeds from each of the lines were randomly assigned to each treatment across two blocks as soon as no new germinants appeared. Germination date was recorded for all seedlings. Plants were bottom watered as needed and fertilized three times per week with 50 ppm Pete's (Allentown, PA) 20:10:20 NPK fertilizer. After seven weeks, all plants were harvested and the following traits were measured: the height of each node; the number of nodes; numbers of axillary meristems on the primary stem allocated to flowers (primary flowers), branches (primary branches), or quiescent buds (buds); the number of leaves; total leaf area; and specific leaf weight (g dry leaf/cm^2 leaf area).

Data Analysis

A mixed-model multivariate analysis of variance (SAS, Proc GLM), with treatment, population, and block as main effects, family (nested within population) as a random factor, and germination date as a continuous covariate, was used to investigate the effects of treatment and genetic variation for plasticity (genotype by environment interaction). The block $\times$ treatment interaction was pooled with the residual mean square because blocks were arbitrarily established rather than biologically determined, and the treatment interactions with block were thought to be unimportant and not biologically relevant (Newman et al. 1997). The effect of treatment was tested over the interaction with family, and the effect of population was tested over the family term. Because of poor germination success, some genotypes were planted into only two of the three treatments. As a consequence, the analysis presented includes the sample of genotypes grown at high and low density separately from the sample that includes genotypes grown in the two high-density treatments. All characters except the number of primary flowers were included in the MANOVA. All three meristem allocation characters could not be included because they were not independent. Individual ANCOVAS were then performed on each character separately, using the same mixed model.

Plasticities to density and to R:FR were calculated for each genotype by determining the difference between genotypic mean values across environments. Plasticity to density was defined as the phenotype at high density minus the phenotype at low density. Plasticity to R:FR was defined as the phenotype at high density minus the phenotype in the blocked-cue treatment. Genotypic correlations between plasticities were calculated as Pearson correlations between genotype plasticity to density and plasticity to R:FR. *Z*-transformation of correlation coefficients (Sokal and Rohlf 1981), using the adaptation to small sample sizes suggested by Hotelling (1953), was performed to construct 95% confidence intervals (corrected for multiple comparisons). Confidence intervals were constructed to test the hypotheses that correlations were significantly different from 0.5, the value expected if correlations were spurious and variances were equal. Spurious correlations are expected because plasticity to density (high minus low) and plasticity to R:FR (high minus blocked cue) both are functions of the phenotype expressed at high density. As another index of the genetic relationship between plasticity to density and plasticity to R:FR, we calculated genetic correlations between the same character expressed at low density and the blocked-cue treatments from variance component estimates (Fry 1992). This cross-environment correlation reflects the degree to which trait expression is controlled by the same genes in two environments with high R:FR, but differing in resource availability. Standard errors of the estimates based on jackknife resampling (Roff and Preziosi 1994; Roff 1997; Windig 1997) were calculated to test the hypothesis that correlations were significantly different from 1 or -1. Because of the small sample size and occasional problems with negative variance components that rendered genetic correlations nonestimable, correlations were also calculated from genotypic mean values (Via 1984; Geber 1990; Campbell 1997). Although such correlations are biased estimates of genetic correlations (Windig 1997), this method allowed examination of all of the traits.

Pearson phenotypic correlations were calculated among all characters for plants grown at high and low density. The two populations were pooled because of sample size constraints. In addition, genetic correlations among traits within an environment were estimated from variance components using Free-stat (Mitchell-Olds 1989), with significance tests based on permutation tests. Standard errors to test the hypothesis that correlations were significantly different from 1 or -1 were calculated from bootstrap resampling with a bootstrap program supplied by Patrick Phillips (Department of Biology, University of Texas at Arlington). Standard errors from bootstrapping tend to be inflated (Windig 1997), so this represents a conservative test of the hypotheses. Correlations were also calculated using genotypic means for the same reasons as stated above. Variance-covariance matrices calculated from family mean values were compared between high- and low-density treatments using hierarchical common principle components (CPC) analysis (Flury 1988; program available from Patrick Phillips at http://wbar.uta.edu/software/cpc/file/cocreadme.htm). The matrices using all characters at once could not be compared using this method because the number of matrix elements was large compared to the number of observations. To reduce the number of comparisons, sets of functionally related traits were identified a priori: elongation, meristem allocation, and leaf characters. Matrices of a subset of characters in different functional groups were also compared with CPC. The subset, which contained length of the first internode, number of primary flowers and buds, and total leaf area, was chosen a priori. Genetic variance-covariance matrices were not compared in this manner due to negative variance components.

Germination date is expected to influence adult morphology due to its effect on size hierarchy formation, particularly at high density (Schmitt and Ehrhardt 1990). Earlier germinating plants may grow faster and be more likely to sup-

TABLE 1. Means of characters in the three experimental treatments. Backtransformed means are given when data were transformed for analyses. See Table 2 for significance of effects.

Character	Low density		High density		Blocked cue	
	Sunny	Woodland	Sunny	Woodland	Sunny	Woodland
Elongation						
Hypocotyl (cm)	3.1	3.3	5.0	5.8	3.6	3.9
First internode (cm)	3.7	4.0	12.3	11.4	4.1	4.1
Second internode (cm)	4.7	6.6	8.7	8.6	3.6	4.6
Final height (cm)	18.3	22.8	37.0	40.9	14.1	18.7
Meristem and resource allocation						
No. of nodes	5.5	5.9	4.3	4.8	4.2	4.8
No. of primary flowers	5.1	8.2	2.8	4.3	2.7	5.7
No. of primary branches	8.2	6.1	0.7	2.2	1.8	2.9
No. of buds	1.5	1.4	4.5	3.1	3.8	2.4
Leaf characters						
Total leaf area (cm^2)	95.6	101.5	38.5	42.5	33.5	55.2
No. of leaves	12.6	14.4	7.7	8.5	7.8	9.2
Specific leaf weight (g/cm^2)	1.4	1.4	0.6	0.7	0.8	0.8

press later germinants. To control for this possible effect on the genetic relationships among traits, genetic correlations were recalculated as before using the residuals of a series of regressions of the characters on germination date (Geber 1990).

RESULTS

Genetic Variation in Reaction Norms to Density and R:FR

MANOVA detected a significant density effect (F = 137.30; df = 10, 9; $P < 0.001$) and a significant effect of R:FR on plant morphology (F = 104.74; df = 10, 7; $P < 0.001$). Plants grown at high density had longer hypocotyls, first and second internodes, and were taller by the time of harvest than plants grown at low density (Tables 1, 2). Node, flower, and branch production was reduced at high density and more meristems remained dormant as quiescent buds. Plants at high density had lower specific leaf weight than plants at low density. They also had less leaf area, due to a reduction in the number of leaves. Stem elongation responses to density were suppressed in the blocked-cue treatment (Tables 1, 2), evidence that phytochrome plays an important role in the plasticity of these traits to density. There was no significant main effect of R:FR on meristem allocation or leaf traits, suggesting that their response to density was not strongly phytochrome mediated.

MANOVA detected a nearly significant family main effect on plant morphology across densities (high and low density F = 1.30; df = 190, 102; P = 0.07) and a significant effect across R:FR treatments (high density and blocked cue F = 1.57, df = 190, 84; $P < 0.01$). Individual analyses of covariance revealed significant main effects of family across densities for hypocotyl length, first internode length, and meristem allocation to flower production and significant population effects for hypocotyl length, meristem allocation to flowering, and number of nodes (Table 2). However, MANOVA population effects were not significant (F = 1.29, df = 10, 10; $P > 0.05$). Analysis of individual traits across R:FR treatments detected significant main effects of family for hypocotyl length and meristem allocation to flowers and buds and significant differences between populations in hypocotyl length, meristem allocation to flowers and branches, meristem dormancy, number of nodes, total leaf area, and number of leaves (MANOVA population effect F = 3.24, df = 10, 10; $P < 0.05$).

Significant genotype-by-treatment interactions in the MANOVA revealed genetic variation within populations for plastic responses to density (F = 1.47; df = 180, 1226; $P < 0.001$). Individual analyses of covariance detected significant genetic variation for plasticity to density for primary branch production and number of leaves (Table 2). Despite the crossing of reaction norms for other characters, large standard errors prevented the interactions from being significant. There was some evidence of population differentiation in plasticity to density, as indicated by a significant population-by-treatment interaction for number of primary branches and nearly significant interactions for length of the second internode and number of quiescent buds (MANOVA F = 1.71; df = 10, 9; $P > 0.05$). In all cases, the sunny population exhibited greater plasticity than did the woodland population.

MANOVA revealed no significant genetic variation within populations for plasticity to R:FR (F = 1.12; df = 160, 1078; $P > 0.05$), but individual ANCOVAS detected evidence for genetic variation for plasticity to R:FR for first and second internode lengths, number of nodes (nearly significant), total leaf area, and number of leaves (Table 2). Only specific leaf weight showed a significant difference in plasticity to R:FR between populations, with the sunny population being slightly more responsive, but this difference was quite small (MANOVA F = 0.65; df = 10, 7; $P > 0.05$).

Genetic Correlations across Environments and Correlations among Plasticities

Plasticities to density were strongly correlated with plasticities to R:FR for stem elongation (Table 3), suggesting that the same phytochrome-mediated genes are involved in both

TABLE 2. Analysis of covariance of plant characters. Effect of germination date is not shown. Geno, genotype (family) effect; G × T, genotype-by-treatment interaction; P × T, population-by-treatment interaction.

	Low and high density					High density and blocked cue				
	Treatment	Geno	Population	Block	G × T	P × T	Treatment	Geno	Population	Block	G × T	P × T
Hypocotyl	188.9***	2.5*	10.6**	0.2	0.9	1.6	91.9***	3.6**	10.6**	0.0	0.9	0.0
First internode	240.2***	4.1**	0.0	0.1	0.9	0.4	124.5***	1.1	0.5	0.0	1.9[1,*]	0.7
Second internode	7.6**	1.1	0.0	2.9[+]	1.1	4.0[+]	33.3***	0.6	0.1	7.2**	1.9[1,*]	1.1
Height	39.6***	1.5	1.7	3.5[+]	1.4	0.1	78.0***	1.3	1.7	4.8[+]	1.8	0.1
Primary flowers	45.1***	2.6*	9.0**	18.1***	1.3	0.5	0.2	2.6*	11.4**	5.3*	1.3	0.6
Primary branches	147.8***	1.4	0.9	7.7**	2.3*	19.2***	1.3	1.1	13.5***	17.9***	1.0	0.0
Buds	61.5***	1.7	3.6[+]	12.5***	1.2	4.0[+]	0.8	2.5*	6.6*	0.1	1.2	0.0
No. of nodes	41.5***	1.7	5.6*	22.6***	1.3	0.0	3.4[+]	1.8	5.3*	18.1***	1.5[1,+]	0.2
Leaf area	62.2***	1.8	0.0	19.1***	1.1	0.0	0.7	1.0	5.2*	10.5**	1.9[1,*]	1.0
No. of leaves	79.5***	1.7	2.7	7.9**	1.7*	0.3	0.3	1.2	6.0*	12.2***	1.7*	0.4
Specific leaf weight	148.1***	0.8	3.4	3.4[+]	1.3	2.0	1.2	1.8	2.8	16.2***	1.0	4.6[1,*]

[1] Effect not significant when the subsample of genotypes that were grown in all three treatments were analyzed in the same model.
*** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$; [+] $P < 0.1$.

TABLE 3. Correlations (r) between plasticities to density and plasticities to R:FR (left) and correlations across low-density and blocked-cue environments based on variance component estimates (right). Correlations based on genotypic mean values are in parentheses. Boldface in left column indicates the correlation is significantly different from 0.5 after sequential Bonferroni corrections ($\alpha = 0.05$). Significance of the character correlations across environments are based on the significance of the genotype effect in analysis of variance (Fry 1992). Boldface of the variance component estimates indicates that the correlation is significantly different from zero after sequential Bonferroni corrections ($\alpha = 0.05$). NE, nonestimable due to negative variance components.

Character	Plasticities	Low, blocked cue
Elongation		
Hypocotyl	0.78	1.00[1,*] **(0.69**)**
First internode	**0.96**	0.81* (0.53*)
Second internode	**0.96**	1.00[1,**] (0.54*)
Final height	**0.92**	1.00[1,*] (0.47[+])
Meristem and resource allocation		
No. of nodes	0.67	1.00[1] (0.19)
Primary flowers	0.77	**1.00[1,***] (0.82***)**
Primary branches	**−0.27**	−1.00[1] (−0.51*)
No. of buds	0.07	0.82 (0.13)
Leaf characters		
Total leaf area	0.44	1.00[1] (−0.02)
No. of leaves	0.40	0.97 (0.06)
Specific leaf weight	0.59	NE (−0.16)

[1] Estimate exceeded 1 or −1 and was rounded off.
*** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$; [+] $P < 0.1$.

responses. However, for other traits, phytochrome pathways appeared to be less important for the expression of plasticity to density. The correlation for primary branch production was negative and significantly less than the expected spurious correlation of 0.5, suggesting a possible trade-off in direction of plasticity to density and to R:FR.

Genetic correlations across low-density and blocked-cue environments (i.e., across similar R:FR environments differing in resource availability) were consistent with these results (Table 3). However, there was little power to detect tablewide significance after sequential Bonferroni corrections. All elongation characters displayed strongly positive genetic correlations between the low-density and blocked-cue treatments (not significantly different from one), suggesting a common genetic basis for expression of these traits in similar R:FR environments regardless of resource levels. As for correlations between plasticities to density and R:FR, a different pattern was apparent for primary branch production. This trait displayed a large negative genetic correlation between the low-density and blocked-cue treatments (marginally significant based on genotypic mean values), again suggesting a genetic trade-off in meristem allocation to branching between high- and low-resource environments. The lack of genetic variation in plasticity of meristem allocation to flowers was reflected in a large positive genetic correlation across environments. Leaf characters showed nonsignificant correlations across environments with similar R:FR, suggesting that the difference in resource environment between the two treatments influences responses more strongly than does the similarity in R:FR.

TABLE 4. Genetic correlations among traits of plants grown in high (above diagonal) and low (below diagonal) density based on variance component estimates. Values in parentheses are correlations based on genotypic mean values. Boldface indicates significance after sequential Bonferroni correction ($\alpha = 0.05$ based on 55 comparisons). Hyp, length of hypocotyl (cm); Int 1, length of first internode (cm); Int 2, length of second internode (cm); 1° Fl, number of primary flowers; 1° Br, number of primary branches; SLW, specific leaf weight (g/cm^2); NE, nonestimable due to negative variance components.

	Hyp	Int 1	Int 2	Height	No. of nodes
Hyp		0.38 (0.23)	NE (−0.11)	0.69 (0.04)	−0.06 (0.05)
Int 1	**1.00**[1]******* (0.49*)		NE **(0.69***)**	0.76 **(0.76***)**	0.15 (0.42$^+$)
Int 2	**1.00**[1]******* **(0.71**)**	**0.79***** **(0.62**)**		NE **(0.95***)**	NE **(0.71***)**
Height	**1.00**[1]******* **(0.78***)**	**0.59***** (0.42$^+$)	**0.95***** **(0.89***)**		0.29 **(0.81***)**
No. of nodes	0.41 (0.27)	−0.26 (−0.35)	0.46 (0.19)	0.64$^+$ (0.47$^+$)	
1° Fl	0.36 (0.17)	0.01 (−0.15)	0.39** (0.27)	**0.51***** (0.46$^+$)	0.88** (0.52*)
1° Br	−0.26 (−0.01)	−0.23 (−0.14)	**−0.42***** (−0.31)	**−0.42***** (−0.27)	**−0.77***** (−0.27)
No. of buds	−0.88 (−0.20)	−0.09 (−0.29)	−0.36 (−0.28)	−0.58* (−0.14)	−0.80 (0.06)
Leaf area	1.00 (0.50*)	1.00[1] (−0.24)	0.67 (0.13)	1.00[1] (0.40)	−0.36 (0.34)
No. of leaves	0.54$^+$ (0.35)	−0.21 (−0.33)	0.40* (0.29)	**0.63***** (0.42$^+$)	0.81* (0.42$^+$)
SLW	0.81 (0.41)	1.00[1]* (0.15)	0.08 (0.14)	0.10 (0.38)	−0.42 (0.26)

[1] Estimate exceeded 1 or −1 and was rounded off.
*** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$; $^+$ $P < 0.1$.

Density-Dependent Correlations among Characters

Strong positive genetic correlations were found among the strongly phytochrome-mediated elongation characters within both environments (Table 4). However, hypocotyl length was significantly correlated with the other elongation characters only at low density. The family mean correlation matrices for elongation traits differed significantly between density treatments, as indicated by CPC analysis (χ^2 for matrix equality = 64.51, $P < 0.001$). The matrices were not proportional ($\chi^2 = 20.72$, $P < 0.05$), and they did not share common principle components ($\chi^2 = 18.35$, $P < 0.01$), in large part due to the weaker relationship between the hypocotyl and the other elongation traits at high density.

The relationships among allocation characters also differed significantly across density treatments, as indicated by CPC analysis of family mean correlations ($\chi^2 = 60.63$, $P < 0.001$ for equality; $\chi^2 = 60.50$, $P < 0.001$ for proportionality). At low density, we observed significant negative genetic correlations of allocation to branches with second internode length, height, allocation to flowers, and number of nodes; in contrast, at high density, all of these correlations were positive (significantly so for allocation to branches vs. flowers). Thus, trade-offs for these traits were apparent only at low density. A negative genetic correlation between allocation to flowers and quiescent buds was apparent in both density environments, although it was significant only at low density (Table 4).

The relationships among leaf characters differed significantly across the two density treatments ($\chi^2 = 31.63$, $P < 0.001$ for equality; $\chi^2 = 30.37$, $P < 0.001$ for proportionality; $\chi^2 = 19.44$, $P < 0.01$ for common principle components). Specific leaf weight was significantly positively correlated with number of leaves at high density but not at low density (Table 4).

The relationships among characters in different functional groups differed significantly across the two density treatments ($\chi^2 = 65.63$, $P < 0.001$ for equality; $\chi^2 = 65.05$, $P < 0.001$ for proportionality). The difference in the covariance structure based on family means was due in part to a marginally significantly positive correlation between leaf area and length of the first internode at high density, but a negative correlation at low density. None of the genetic correlations among these characters were significant, however, and some were nonestimable (Table 4). Genetically based trade-offs were found between leaf number and number of branches and buds (marginally significant) at low density. Leaf characters were strongly positively correlated with several elongation and meristem characters at high density based on family mean correlations, whereas few of these correlations were strong at low density, and many were negative.

No genetic correlations were significantly different from

TABLE 4. Extended.

1° Fl	1° Br	No. of buds	Leaf area	No. of leaves	SLW
0.18 (0.21)	0.68 (0.05)	−0.78* (−0.62**)	NE (−0.09)	0.60 (0.11)	NE (0.19)
−0.46 (0.03)	0.31 (0.18)	0.07 (0.35)	NE (0.50*)	0.27 (0.61**)	NE (0.24)
NE (0.16)	NE (0.45+)	NE (0.50*)	NE **(0.82***)**	NE **(0.79***)**	NE (0.44+)
−0.31 (0.35)	1.00[1] (0.57*)	0.00 (0.29)	NE **(0.90***)**	0.97 **(0.89***)**	NE (0.59*)
0.69 (0.61**)	0.43 (0.49*)	−0.44 (0.10)	NE **(0.80***)**	NE **(0.79***)**	NE **(0.79***)**
	0.80* (0.62**)	−0.70+ (−0.44+)	NE (0.59*)	0.71 (0.52*)	NE (0.67**)
−0.96*** **(−0.83***)**		−0.62 (−0.33)	NE **(0.80***)**	NE (0.64**)	NE (0.64**)
−0.75*** (0.07)	0.25 (−0.05)		NE (0.11)	NE (0.08)	NE (−0.19)
1.00[1] (0.16)	−0.52 (0.30)	−1.00[1] (0.20)		NE **(0.89***)**	NE (0.67**)
0.78*** (0.35)	−0.31** (0.04)	−0.68* (−0.30)	1.00[1] (0.69**)		NE (0.54*)
−0.66 (0.13)	0.28 (0.03)	0.31 (0.24)	−1.00[1] (0.28)	−0.56 (−0.01)	

1 if positive or from −1 if negative, based on bootstrapped standard error estimates, although power was low for this analysis. Correlations based on residuals of germination date did not differ substantially from standard correlations for any trait (not shown).

Phenotypic correlations generally reflected the same patterns as the genetic correlations, except that some trade-offs observed in the genetic analysis were not apparent in the phenotypic analysis, presumably due to positive environmental correlations (Table 5). Phenotypic correlations were stronger at high density than at low density, except for correlations involving quiescent bud number, which were weak in both environments. The phenotypic correlation between branch and flower allocation was negative at low density but positive at high density, a result consistent with the genetic correlation analysis. Forty-two of 55 correlations were significant at high density by Bonferroni criteria ($\alpha = 0.05$), whereas only 16 were significant at low density. At high density, three correlations were negative, and at low density 10 were negative. Covariance relationships differred significantly between high and low density (equality $\chi^2 = 645.22$, df = 66, $P < 0.0001$; proportionality $\chi^2 = 588.71$, df = 65, $P < 0.0001$; common principle components $\chi^2 = 204.08$, df = 55, $P < 0.0001$).

DISCUSSION

The results of this study demonstrate that genetic variation in plasticity to density can be dissected into phytochrome-mediated responses to the R:FR cue and responses to other environmental factors (such as resource availability) that vary with density. These different mechanisms for plasticity had different effects on character integration within and across environments. Traits sharing strong phytochrome-mediated sensitivity to the R:FR cue displayed similar plasticities to density and were tightly correlated with one another regardless of the density environment. Traits for which the response to density was not strongly phytochrome mediated differed in patterns of plasticity, and genetic correlations involving these traits often differed across environments. In particular, genetic trade-offs in allocation traits were expressed only at low density. Thus, as predicted, characters with plasticity mediated by a common signal were more strongly integrated across environments than characters that did not share such sensitivity. The observed density dependence of phenotypic and genetic correlations implies that patterns of indirect selection and the potential for correlated response to selection will depend upon the competitive environment.

Impatiens capensis displayed strong plasticity to density and, for some characters, strong plasticity to the R:FR cue. As previously observed (Dudley and Schmitt 1995), the stem elongation response to density was sensitive to R:FR and therefore was phytochrome mediated, but the responses to density of leaf traits and some meristem allocation characters were less strongly phytochrome dependent.

The degree to which plasticity of different characters is determined by the same phytochrome-mediated mechanism

TABLE 5. Phenotypic correlations among traits within high (above diagonal) and low (below diagonal) density. Boldface indicates significance after sequential Bonferroni correction ($\alpha = 0.05$, 55 comparisons). Abbreviations are as in Table 4.

	Hyp	Int 1	Int 2	Height	No. of nodes	1° Fl	1° Br	No. of buds	Leaf area	No. of leaves	SLW
Hyp		**0.41*****	0.20*	**0.37*****	**0.29****	**0.44*****	0.17*	−0.15	0.26*	**0.34*****	0.25*
Int 1	0.23*		**0.68*****	**0.72*****	**0.60*****	**0.56*****	**0.26****	**0.36*****	**0.75*****	**0.67*****	**0.51*****
Int 2	**0.46*****	**0.61*****		**0.90*****	**0.77*****	**0.65*****	**0.48*****	0.13	**0.94*****	**0.82*****	**0.77*****
Height	**0.56*****	**0.40*****	**0.87*****		**0.89*****	**0.73*****	**0.63*****	0.07	**0.88*****	**0.87*****	**0.79*****
No. of nodes	0.29**	−0.19	0.28**	**0.55*****		**0.69*****	**0.64*****	0.06	**0.83*****	**0.89*****	**0.78*****
1° Fl	0.21*	−0.15	0.11	0.26**	0.32**		**0.51*****	−0.08	**0.72*****	**0.73*****	**0.65*****
1° Br	0.10	−0.05	0.08	0.09	0.19	**−0.45*****		−0.21*	**0.57*****	**0.66*****	**0.63*****
No. of buds	−0.12	−0.25*	−0.29**	−0.15	0.16	0.06	0.01		0.18⁺	0.12	0.03
Leaf area	**0.38*****	0.08	**0.46*****	**0.59*****	**0.53*****	0.23*	**0.51*****	0.16		**0.90*****	**0.73*****
No. of leaves	0.29**	−0.18	0.30**	**0.48*****	**0.44*****	0.28**	0.08	−0.05	**0.60*****		**0.76*****
SLW	0.23*	0.03	0.22*	0.25*	**0.32****	0.11	0.21*	0.08	0.24*	0.18⁺	

*** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$; ⁺ $P < 0.1$.

could influence the degree to which these characters and their plasticity can evolve independently from one another (Schlichting and Levin 1986; Schlichting 1989; van Tienderen 1990; Schlichting and Pigliucci 1995, 1998; Pigliucci 1996; van Tienderen and van Hinsberg 1996; van Hinsberg 1997). In most cases the characters whose responses were strongly mediated by the R:FR cue also had strong genetic correlations with each other in both density environments. Therefore, the common physiological mechanism of mediation by phytochrome appears to reflect a common genetic mechanism for the response of these traits. Thus, the shade avoidance syndrome may evolve as a suite of characters controlled by the same developmental pathway. The one exception to this pattern involved correlations of hypocotyl length with the other elongation characters, which were much stronger at low density than at high density. A possible explanation for this result is that the light environment changes over time in dense stands as the canopy closes. Early germinants experience noncompetitive conditions at the time of hypocotyl elongation, but later germinants and later internodes experience the reduced irradiance and R:FR typical of competitive conditions. Consequently, the weak correlations between hypocotyl length and other elongation traits at high density probably result from differences in the environment the plant experiences at different developmental stages, rather than from differences in photomorphogenic mechanisms.

Characters that did not share the common mechanism of mediation by the R:FR cue were more labile in correlation structure across environments. This result suggests that differential plasticity of different characters, due in part to different genetically based physiological mechanisms of plastic responses, can cause genetic correlations among such characters to vary with the environment, as suggested by Stearns et al. (1991). In particular, the expression of genetic trade-offs involving allocation characters was density dependent. At low density, allocation to branches was negatively correlated with height, number of nodes, and meristem allocation to flowers. At high density, however, these trade-offs were not apparent; in fact, genotypes with more branches also produced more primary flowers. It is interesting to note that trade-offs are often thought to be stronger under conditions of low resource availability or more competitive conditions (e. g., Adams 1967; Reznick 1985; Biere 1995; Kawano and Hara 1995), but in this study the opposite was observed; the trade-offs were apparent only under the high-resource, low-density environment. More negative phenotypic correlations were also observed under low density than high density. Phenotypic integration was stronger at high density, indicated by the stronger phenotypic correlations, as it has been shown to be in other plant systems (Kawano and Hara 1995). These results are probably due to exaggeration of variation in individual growth rates by asymmetric competition for light (Stanton 1985; Weiner 1985, 1990; Schmitt et al. 1986; Weiner and Thomas 1986; Scheiner 1989). If certain genotypes are more vigorous than others, asymmetric competition will intensify these genetic differences in growth rate under competitive conditions. Such genetically based variation in general vigor or resource acquisition can cause positive genetic correlations for many characters even if there are physiolog-

ical trade-offs (van Noordwijk and de Jong 1986; Stearns et al. 1991; Houle 1991). At low density, however, genetic variation in resource allocation may be larger relative to variation in resource acquisition, so trade-offs in allocation become apparent. If so, density-dependent expression of allocation trade-offs may be common in systems where resource competition is asymmetric. More generally, if genetic variation in resource acquisition is only expressed when resources are limiting, then genetic trade-offs are more likely to be detected in high-resource environments.

Although plasticity integration appears to be facilitated by shared sensitivities to an environmental cue, integration can apparently occur through other mechanisms as well. In this system, genetic correlations among characters within functional groups were often strong, indicating strong integration. Similarly, Waitt and Levin (1993) found that functionally or developmentally related characters had similar plasticities in *Phlox dummondii* and were strongly correlated. Integration was also apparent between characters within different functional groups, similar to results of Schlichting (1986; Schlichting and Pigliucci 1998), who found in *Phlox* that plasticities were sometimes strongly correlated between characters of different functional groups.

The result that the magnitude and direction of correlations between some traits depend on density indicates that the evolution of these characters may be strongly density dependent. Not only does direct selection vary with density (Dudley and Schmitt 1996), but indirect selection may also vary with the changing phenotypic correlation matrix (Lande and Arnold 1983). Moreover, the response to selection is also expected to vary due to the different genetic architecture at high and low density. Therefore, density influences not only the evolution of mean phenotypes of individual characters, but also the relationship among characters. Various studies in other systems have also documented that phenotypic and genetic correlations can vary with the environment (e. g., Service and Rose 1985; Schlichting 1989; Holloway et al. 1990; Mazer and Schick 1991; Thomas and Bazzaz 1993; Pigliucci et al. 1995; van Tienderen and van Hinsberg 1996) and that genetic correlations strongly influence correlated responses to selection (e.g. Hillesheim and Sterns 1992; Kelly 1992; Scheiner and Istock 1992; van Tienderen and van Hinsberg 1996; van Hinsberg 1998).

Shared sensitivity to a common environmental cue can influence the manner in which genetic architecture changes with the environment and consequently can influence the evolution of integrated plasticity. In many organisms, plastic responses to density can involve both developmental responses to environmental signals (such as R:FR or pheromones) and growth responses to resource competition. Thus, the patterns of density-dependent genetic architecture reported here may be general for a wide range of plants and animals.

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