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PLANT-ANIMAL INTERACTIONS - ORIGINAL RESEARCH

Intraspecific competition facilitates the evolution of tolerance to insect damage in the perennial plant *Solanum carolinense*

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Abstract Tolerance to herbivory (the degree to which plants maintain fitness after damage) is a key component of plant defense, so understanding how natural selection and evolutionary constraints act on tolerance traits is important to general theories of plant–herbivore interactions. These factors may be affected by plant competition, which often interacts with damage to influence trait expression and fitness. However, few studies have manipulated competitor density to examine the evolutionary effects of competition on tolerance. In this study, we tested whether intraspecific competition affects four aspects of the evolution of tolerance to herbivory in the perennial plant *Solanum carolinense*: phenotypic expression, expression of genetic variation, the adaptive value of tolerance, and costs of tolerance. We manipulated insect damage and intraspecific competition for clonal lines of *S. carolinense* in a greenhouse experiment, and measured tolerance in terms of sexual and asexual fitness components. Compared to plants growing at low density, plants growing at high density had greater expression of and genetic variation in tolerance, and experienced greater fitness benefits from tolerance when damaged. Tolerance was not costly for plants growing at either density, and only plants growing at low density benefited from tolerance when undamaged, perhaps due to greater intrinsic growth rates of more tolerant genotypes. These results suggest that competition is likely to facilitate the evolution of tolerance in *S. carolinense*, and perhaps in other plants that regularly experience competition, while spatio-temporal variation in density may maintain genetic variation in tolerance.

Keywords Compensatory responses · Costs of tolerance · Phenotypic plasticity · Plant–herbivore interactions · *Solanum carolinense*

Introduction

Most plants receive at least some damage from herbivores and some are able to maintain fitness despite substantial tissue loss. This capacity, termed tolerance to herbivory (Stowe et al. 2000), has been attributed to a combination of intrinsic traits, such as plant size or growth rate, and plastic responses to damage, hereafter referred to as compensatory responses (Hochwender et al. 2000; Tiffin 2000; Weis et al. 2000; Stevens et al. 2008). To understand how the evolution of tolerance fits into general theories of plant–herbivore interactions, such as coevolution and optimal defense theory (reviewed by Juenger and Lennartsson 2000; Fornoni 2011), it is necessary to explain why plants are not completely tolerant. Many studies have focused on identifying constraints or measuring selection on traits conferring tolerance to herbivory (e.g., Tiffin and Rausher 1999; Juenger and Bergelson 2000; Weinig et al. 2003), but these aspects of the evolution of tolerance may be greatly affected by environmental context. For example, biotic and abiotic factors have been shown to affect the phenotypic expression of tolerance to herbivory (Hartnett 1989; Maschinski and Whitham 1989; Huhta et al. 2000; Rand 2004; reviewed by Hawkes and Sullivan 2001; Wise and

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Abrahamson 2007), constraints on its evolution (Hochwender et al. 2000; Stinchcombe 2002; Siemens et al. 2003), and its adaptive value (Lennartsson et al. 1997; Juenger and Bergelson 2000; Juenger et al. 2000).

Plant density (intra- or interspecific) is one aspect of the environment that likely has important effects on the evolution of herbivore tolerance, because competition and damage can interact to affect plant trait expression and fitness (Maron 2001; Del-Val and Crawley 2004; Rand 2004; but see Rees and Brown 1992; Suwa et al. 2010). Competition may affect plant fitness, and thus influence tolerance, through altered resource levels (reviewed by Schmitt and Wulff 1993; Casper and Jackson 1997), allelopathy (Muller 1966; Singh et al. 1999), apparent competition (e.g., Muller 1966; Dangremond et al. 2010), or interactive effects of above- and belowground competition (Cahill 1999). Although most prior studies have manipulated only resource levels (but see Siemens et al. 2003), directly manipulating intra- or interspecific density is necessary to determine the overall evolutionary effects of competition, not just those mediated by resource limitation.

There are at least three general ways in which competition, including associated changes in resource availability, could inhibit or facilitate adaptive evolution of tolerance. First, competition might affect the average expression of traits conferring tolerance, and selection can only act on the expressed phenotype. Competition may reduce tolerance to herbivory if it limits resources needed for regrowth or reproduction after damage (Maschinski and Whitham 1989; Huhta et al. 2000), or if plastic responses to competition inhibit the expression of compensatory responses, as may occur for other defense traits (reviewed by Cipollini 2004). Alternatively, competition may facilitate tolerance to herbivory if responses to competition also provide fitness benefits when plants are damaged by herbivores. For example, increased belowground allocation (i.e., higher root:shoot ratio) is a typical response of perennial plants to competition (Cheplick and Gutierrez 2000; Berendse and Moller 2009). As this may also lead to greater compensation after herbivory (van der Meijden et al. 1988; Hochwender et al. 2000; but see Stevens et al. 2008), increased expression of tolerance under competition seems likely for some perennials.

A second, related, way in which competition might influence the evolution of tolerance is by altering the expression of genetic variation in tolerance (Maschinski and Whitham 1989; Huhta et al. 2000; Rand 2004). If the effects of competition on abiotic conditions (such as light or nutrients) or biotic interactions (such as pollination) strongly limit fitness (reviewed by Stowe et al. 2000), this could minimize fitness differences among genotypes, thereby reducing expressed genetic variation in tolerance and limiting the opportunity for selection. Alternatively, variation in plant fitness may increase under stress (Stanton et al. 2000), including high intraspecific density (Miller et al. 1994; Winn and Miller 1995), which could increase the opportunity for selection on tolerance. It is also possible that competition may alter the rank order of tolerance among genotypes between low and high intraspecific density if genotypic fitnesses are differentially sensitive to density (sensu Shaw 1986). This may manifest as a trade-off in tolerance among competitive environments, which would constrain the evolution of greater tolerance under spatial or temporal variation in competitor density.

Finally, the competitive environment may affect the evolution of tolerance by altering the adaptive landscape, or the relationship between traits conferring tolerance and fitness in each herbivore environment. Competition may affect the adaptive value of tolerance (i.e., the relative fitness benefit that tolerance provides after damage) when herbivores are present, or the expression of evolutionary costs of tolerance (i.e., negative fitness effects of tolerance in the absence of damage) when herbivores are absent. For example, if the fitness of smaller plants is affected more by damage than that of larger plants (Hendrix 1979), traits that confer tolerance, particularly compensatory responses, may provide more benefits, and thus be most adaptive in a competitive environment. While this has been shown to be the case under nutrient limitation (Hochwender et al. 2000), no previous study has examined the effect of competitor density on the adaptive value of tolerance. Competition may also affect the expression of costs of tolerance, which act as evolutionary constraints. Costs of tolerance have been found to vary among nutrient environments (Hochwender et al. 2000) and competitor densities (Siemens et al. 2003), but general conclusions about the effects of competition on costs of tolerance have not yet emerged. Studies examining competitive effects via nutrient limitation have found opposing effects of manipulating nutrient levels on costs of tolerance (Hochwender et al. 2000; Stevens et al. 2007).

We investigated effects of competitive environment on factors that affect the evolution of herbivore tolerance in the clonal perennial *Solanum carolinense*. We manipulated insect damage and intraspecific density in a fully-factorial greenhouse study and asked the following questions: (1) How does intraspecific competition affect the average phenotypic expression of tolerance in *S. carolinense*? (2) Does competition affect the expression of genetic variation in tolerance? (3) Are traits conferring tolerance more or less adaptive at high versus low intraspecific density? (4) Are there costs of tolerance, and how are they affected by the competitive environment?

Materials and methods

Study species

Solanum carolinense (Carolina horsenettle) is a clonal perennial native to the southeastern United States (Imura 2003; Miyazuki 2008). It typically occurs in disturbed sites, such as old-fields and roadsides, and is considered a serious agricultural pest in the United States and is invasive elsewhere (Hackett et al. 1987; Frank 1990; Miyazuki 2008). Asexual reproduction occurs via roots and underground shoots (Wehtje et al. 1987; Miyazuki 2008), and seeds are produced from buzz-pollinated flowers. In this experiment, we used nine clonal lines of *S. carolinense* obtained from five populations in north Florida and Georgia and propagated for 5 years in the greenhouse in a 3:1 mix of Fafard Professional 3 Mix (Conrad Fafard, Agawam, MA, USA) and coarse sand; this same mix was used for our experiment.

We manipulated the presence of two herbivores that naturally colonize *S. carolinense* in north Florida: the specialist chrysomelid *Leptinotarsa juncta* (false potato beetle) and the generalist noctuid moth *Spodoptera exigua* (beet armyworm). We used *L. juncta* larvae from a laboratory colony derived from field-caught insects, and *S. exigua* larvae from Benzon Research (Carlisle, PA, USA). In natural populations, herbivore damage affects *S. carolinense* fitness (Wise and Sacchi 1996), and there can be genetic variation in tolerance to herbivory (Wise et al. 2008).

Experimental design

To investigate how competition affects the evolution of tolerance, we used a fully-crossed greenhouse experiment manipulating intraspecific plant density (high or low), herbivore damage (present vs. absent), and *S. carolinense* genotype (nine clones known to differ in tolerance; S. Halpern and N. Underwood, unpublished field data). We first grew plants of the 9 focal genotypes, plus 29 other genotypes to use as neighbors in the high density treatment, by planting 1.5 g root cuttings in 475-mL pots in February 2008. When cuttings had grown a shoot with several leaves, we established density treatments by transplanting plants into 1.68-L pots. Low density pots received one focal plant, while high density pots received the focal plant surrounded by four neighbors of approximately equal size, each of a different genotype. To keep initial plant sizes similar across treatments, we transplanted in two balanced temporal blocks on 5 and 18 March. After transplantation, we assigned each pot to a spatial block (greenhouse bench); focal genotypes were replicated evenly among temporal and spatial blocks, density, and damage treatments, and treatment combinations were evenly divided among blocks. We fertilized every 7 days by watering until soil saturation with 20-10-20 water-soluble fertilizer (Peters Professional; Scotts-Sierra Horticultural Products, Marysville, OH, USA) at a concentration of 300 ppm nitrogen.

We imposed damage treatments after plants had grown for approximately 6 weeks and had reached the 5–7 leaf stage. We measured initial size for each focal plant (stem height, leaf number, and length of longest leaf); average initial size of plants in the two damage treatments did not differ. Each focal genotype was replicated 2–8 times per density/damage treatment combination (replication varied because the proportion of root cuttings that sprouted differed among genotypes) for a total of 223 plants (1 plant died before treatment). On 14 April (block 1) and 29 April (block 2), we applied damage treatments to each temporal block over the course of 7 days. For damaged plants, we tied breathable mesh bags that contained either four 3–4th instar *L. juncta* larvae or five 3–4th instar *S. exigua* larvae over two leaves per plant; each plant received both herbivore species. We allowed insects to consume approximately 50 % of each leaf and then transferred the bag to another leaf on the same plant, adding new larvae if necessary, until all fully expanded leaves on the plant were damaged. This damage amount is above the natural average of 27 % leaf area lost per plant, but well within the maximum damage intensity of 79 % observed in natural populations (S. Halpern, personal observation). After repeated bagging, some leaves still had >50 % leaf area remaining, and we cut them with sterile scissors to standardize damage at 50 % per plant. For undamaged plants, we tied breathable mesh bags without insects around two leaves and rotated them every 2 days to control for leaf handling/shading. Neighboring plants in the high density treatments were not damaged or handled.

After damage, we surveyed each focal plant every 5 days for the total number of expanded flowers; this interval ensured that every flower was counted (K. Barrows, personal observation). We marked each flower on its petiole with a permanent black marker to avoid recounting; marking the petiole had no effect on flower duration or morphology (D. McNutt, personal observation). Flowers were counted until the plant stopped flowering or began senescing. Upon completion of flowering, we harvested focal plants, separated their roots and shoots, dried them in a drying oven for 48 h at 65 °C, and weighed them separately. From the time of initial planting, focal plants lived from 114 to 139 days and flowered, on average, for 35 days.

Plant fitness and tolerance measures

We used total biomass, which in perennials typically correlates with increased growth rate, reproduction, and

survival (Arendt 1997), as the primary estimate of fitness for each plant; no fruit was produced because flowers are self-incompatible. Because flowers withered and fell off as the plant matured, we estimated flower biomass by multiplying the number of flowers produced by the average weight of ten flowers haphazardly harvested from plants in a separate experiment. We then summed estimated flower mass, root mass, and shoot mass to obtain total biomass. Because *S. carolinense* can reproduce both sexually and asexually, and these modes of reproduction may contribute differentially to tolerance among competitive environments, we also analyzed the separate fitness components of flower number, above-ground dry biomass (stems and leaves), and below-ground dry biomass (roots).

For total fitness and for each fitness component, we defined tolerance as the difference between the average fitness measures of damaged (D) and undamaged (U) plants of the same clonal line (Strauss and Agrawal 1999). While some studies have used ratio (D/U) or other measures of tolerance (see Strauss and Agrawal 1999), the difference measure is advantageous because it is defined over its entire range and is comparable to measuring tolerance as the slope of a regression of fitness on amount of damage. For analyses of costs, benefits, and trade-offs, which all occurred within density treatments, we calculated tolerance using untransformed fitness because these analyses address evolutionary questions, where genotypes' absolute fitnesses are biologically relevant. Using untransformed fitness, when damage is measured on a proportional scale, can create the appearance of differences in tolerance when none exist if groups of plants differ in size at time of damage (Wise and Carr 2008). In our study, however, plants within density treatments were very similar in size and leaf number at the time of damage, making both proportional and absolute damage equal among damage treatments and genotypes; thus, untransformed fitness is appropriate (Wise and Carr 2008). For the analysis of effects of density on tolerance expression and genetic variation in tolerance (i.e., models that included density as a factor, described below), fitness was log-transformed, avoiding this problem.

Statistical analyses

To test for effects of competition (density) on tolerance expression and for genetic variation in tolerance (questions 1 and 2), we performed four fixed-effects, type III ANOVAs using SAS PROC GLM (SAS Institute 2010); each measure of fitness (total biomass and each fitness component (root mass, shoot mass, and flower number)) was the response variable in a separate model. All response variables were log-transformed, after which they met the assumptions of ANOVA (see Table 1). All models included temporal block, genotype, damage, density, and their interactions as factors; temporal block was considered fixed because it was used to standardize the initial size at transplanting. We also treated genotype as fixed because we used information about performance in the field to identify genotypes with an expected range of tolerance to damage. Two-, three-, and four-way interactions between block and other predictors were never significant and were dropped from the models. To differentiate between competitive effects on tolerance due to intrinsic growth versus compensatory traits, we also included plant height at the time of damage as a covariate in all models. This measure was positively correlated with length of the longest leaf ($r = 0.70$), leaf number ($r = 0.58$), and final undamaged biomass at low density (an estimate of intrinsic growth,

Table 1 Univariate analyses testing for genetic variation in tolerance and whether its expression changes with density

Source	*df*	Total mass		Root mass		Shoot mass		Flower number	
		F	*P*	*F*	*P*	*F*	*P*	*F*	*P*
Block	1	25.85	**<0.0001**	40.57	**<0.0001**	8.89	**0.0033**	0.01	0.93
Density	1	242.01	**<0.0001**	197.66	**<0.0001**	184.49	**<0.0001**	172.4	**<0.0001**
Damage	1	1.2	0.27	1.61	0.21	0.38	0.54	0.18	0.68
Height	1	34.82	**<0.0001**	37.86	**<0.0001**	20.01	**<0.0001**	3.77	**0.054**
Density × damage	1	6.14	**0.01**	12.4	**0.0005**	1.29	0.26	0.08	0.78
Genotype	1	6.62	**<0.0001**	6.29	**<0.0001**	6.37	**<0.0001**	21.34	**<0.0001**
Density × genotype	8	2.71	**0.008**	2.92	**0.004**	2.53	**0.01**	7.86	**<0.0001**
Damage × genotype	8	2.78	**0.006**	3.29	**0.002**	2.27	**0.02**	1.57	0.14
Density × damage × genotype	8	2.82	**0.006**	3.76	**0.0004**	1.78	0.08	2.01	**0.05**

Block interactions were never significant, and thus were dropped from the final model. All predictors were treated as fixed. Total mass, root mass, and shoot mass were log transformed, error $df = 177$. Flower number was log transformed, error $df = 181$

Significant values ($P < 0.05$) shown in bold

$r = 0.64$). In these analyses, we interpret a significant damage effect on fitness as evidence of either undercompensation (reduced fitness with damage) or overcompensation (higher fitness with damage). A significant damage × density interaction indicates that intraspecific competition affects tolerance. A significant genotype × damage interaction indicates there is genetic variation in tolerance, while a genotype × damage × density interaction signifies that the presence of competitors affects the expression of this variation.

While significant genotype × damage × density interactions in the above analyses indicate that competitor density affects the expression of genetic variation in tolerance, they do not differentiate between the two possible ways that density may affect variation: the rank order of tolerance may differ between density environments (genotypic norms of reaction across densities cross), and/or the amount of genetic variation expressed may be different between density environments (the slopes of reaction norms differ but may not cross). To distinguish between these possibilities, we first examined whether the rank order of tolerance values changed across density environments by calculating a Spearman's rank test to determine whether there was a correlation between tolerance in high density and low density treatments for genotypic means of total biomass and each fitness component. A negative correlation in this analysis indicates changes in the relative ranks of tolerance, suggesting a trade-off between tolerance at high versus low competitor density. We next conducted two tests for differences in genetic variation among density environments. First, to determine whether genetic variation in tolerance differed significantly from zero within each density treatment, we used type III ANOVAs on the data from each density treatment separately to test the fixed effects of block, genotype, damage, and their interactions on total biomass and each fitness component in four separate models, each including initial height as a covariate. In these models, a significant genotype × damage interaction is interpreted as genetic variation in tolerance. Because these analyses were performed within density treatments, log-transformation was not necessary to avoid measuring damage and fitness on different scales (Wise and Carr 2008), so we only transformed fitness measures when required to meet model assumptions (see Table 2). Second, to determine if there were differences in the total amount of genetic variation in tolerance expressed at the two competitor densities, we used a Levene's test to test for homogeneity of variance of genotypic tolerance between density treatments. Data were not transformed in this analysis to preserve unequal variances, and the Levene's test is not sensitive to departures from normality.

To determine whether tolerance was adaptive at each density (question 3), we calculated the covariance between tolerance and damaged fitness, where a positive covariance indicates a fitness benefit of tolerance when damage occurs. We used genotypic means for both variables, and analyzed data separately by density treatment and tolerance measure. Because tolerance is calculated using damaged fitness, we corrected for artifactual covariance between tolerance and damaged fitness following the method of Tiffin and

Table 2 Univariate analyses testing for genetic variation in tolerance and whether its expression changes when plants are growing at low density or high density of competitors

Source	*df*	Total biomass		Root mass		Shoot mass		Flower number	
		F	*P*	*F*	*P*	*F*	*P*	*F*	*P*
Low density treatment									
Block	1	44.39	**<0.0001**	53.92	**<0.0001**	11.71	**0.0009**	0.16	0.69
Damage	1	4.08	**0.047**	4.16	**0.044**	1.95	0.17	0.00	0.99
Height	1	21.34	**<0.0001**	24.67	**<0.0001**	6.28	**0.01**	0.79	0.38
Genotype	8	10.48	**<0.0001**	6.88	**<0.0001**	12.95	**<0.0001**	22.34	**<0.0001**
Damage × genotype	8	0.53	0.83	0.82	0.59	0.22	0.99	1.24	0.29
High density treatment									
Block	1	8.92	**0.004**	11.06	**0.001**	4.74	**0.03**	0.07	0.79
Damage	1	3.27	0.07	7.49	**0.008**	0.57	0.45	0.42	0.52
Height	1	22.83	**<0.0001**	21.80	**<0.0001**	15.92	**0.0001**	3.91	**0.05**
Genotype	8	3.53	**0.001**	3.77	**0.0008**	3.03	**0.005**	3.23	**0.003**
Damage × genotype	8	3.28	**0.003**	4.49	**0.0001**	2.20	**0.04**	3.01	**0.005**

All predictors were treated as fixed effects. Low density: error $df = 88$ (all biomass measures) and 89 (flower number); response transformations were square root (flowers). High density: error $df = 87$ (total and shoot biomass), 88 (root biomass), 90 (flower number); all response variables were log-transformed

Significant values ($P < 0.05$) shown in bold

Rausher (1999) using SAS code provided by Stinchcombe (2005). We used bootstrapping to estimate 95 % confidence intervals (CI) for corrected covariances by resampling with replacement 10,000 times (Stinchcombe 2005). If the 95 % CI did not overlap zero, we considered the covariance to be significantly different from zero.

To determine whether there were costs to tolerance (question 4), we examined the covariance between tolerance and undamaged fitness, where a negative covariance indicates a cost. We used similar methods as those testing for the adaptive value of tolerance, including correcting the covariance estimates for artifactual covariance and estimating 95 % CI for corrected covariances using bootstrapping. Because a combination of plant mortality and low sample size led to an unbalanced distribution of biomass measures for one genotype, we removed this line when performing both the adaptive value and costs of tolerance analyses.

Finally, we examined the effect of density on one possible trait conferring tolerance, the post-damage allocation of resources below or above ground (below: aboveground biomass ratio, hereafter B:A) calculated as root biomass/(shoot + estimated flower biomass). To test sources of variation in B:A, we used a univariate type III ANOVA to examine the fixed effects of damage, density, genotype, and their interactions on final (natural log-transformed) B:A. We included temporal block and initial plant height as covariates; initial plant height was removed from the model because it had no effect. In this analysis, a significant density by damage interaction indicates that the effect of damage on final B:A differs with competitor density.

Results

Effects of competitor density on phenotypic expression of tolerance (question 1)

On average, plants showed undercompensation to herbivory, as herbivory reduced all fitness components (average total tolerance $= -0.38 \pm 0.75$ g; average root tolerance $= -0.34 \pm 0.45$ g; average shoot tolerance $= -0.06 \pm 0.31$ g; average flower tolerance $= -0.21 \pm 2.3$ flowers). For total biomass, competition significantly affected tolerance to damage (damage × density interaction; Table 1). Specifically, plants at high density overcompensated for damage, while plants at low density undercompensated (Fig. 1). Of the three fitness components, competition significantly affected root tolerance (Table 1), with overcompensation at high density and undercompensation at low density (Fig. 1). Competition did not significantly affect tolerance measured as shoot mass or flower number (Table 1; Fig. 1).

Effects of density on expression of genetic variation in tolerance (question 2)

There was significant genetic variation in tolerance (damage × genotype interaction; Table 1), and the expression of this genetic variation was affected by density (density × damage × genotype interaction; Table 1). One way that competition affected genetic variation in tolerance was by altering the relative tolerance values of genotypes; there was a negative correlation between tolerance in the two competitive environments for total fitness ($\rho = -0.75$, $P = 0.02$), and root biomass ($\rho = -0.68$, $P = 0.05$) but not for shoot biomass ($\rho = 0.2$, $P = 0.61$) or flower number ($\rho = -0.008$, $P = 0.90$). For total biomass and all fitness components, we found significant genetic variation in tolerance at high density, but not low density (compare damage × genotype interactions; Table 2). For total fitness, there was marginal support for a difference in the amount of genetic variation in tolerance expressed in the high density ($\bar{X} = 1.17$, $\sigma^2 = 13.99$) and low density ($\bar{X} = -1.94$, $\sigma^2 = 1.93$) environments (Levene's test,

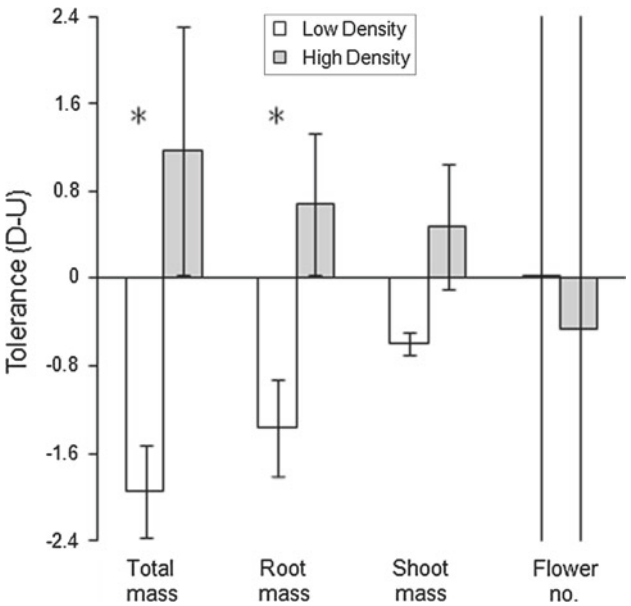

Fig. 1 Level of tolerance at low and high densities for total biomass, root biomass, shoot biomass, and flower number. Tolerance is calculated for each genotype as average damaged fitness − average undamaged fitness ($D-U$) and averaged across genotypes at each density. *Error bars* are mean ± SE; *error bars* that overlap zero indicate complete tolerance (no effect of damage on fitness). *Bars* that do not overlap zero indicate either undercompensation or overcompensation. *Asterisk* indicates that tolerance level is affected by density, determined by a density × damage interaction ($P < 0.05$; see Table 1)

$F_{1,16} = 3.57$, $P = 0.08$). Among fitness components, there was greater genetic variation in tolerance expressed at high density for shoot biomass ($F_{1,16} = 5.48$, $P = 0.03$), but not for root biomass ($F_{1,16} = 1.16$, $P = 0.30$) or flower number ($F_{1,16} = 0.08$, $P = 0.78$; Fig. 2).

Effects of density on the adaptive value and costs of tolerance (questions 3 and 4)

For plants growing at high competitor density, tolerance positively covaried with fitness after damage for total fitness and all fitness components except flower number, indicating that tolerance is adaptive for damaged plants at high density (Table 3). At low density, tolerance did not significantly covary with fitness after damage for any fitness measure (Table 3).

There were no significant costs of tolerance detected at either density. At high density, the bootstrapped 95 % CI of corrected covariance overlapped 0 for each of the fitness measures (Table 4). At low density, however, we found significant benefits to tolerance when plants remained undamaged; tolerance positively covaried with undamaged fitness as measured by total biomass, root biomass, and shoot biomass, but not flower number (Table 4).

Effects of density and damage on final B:A

Plants grown at high density had a 1.6× larger B:A at harvest under competition (Fig. 3). The effect of damage on final B:A differed across plant densities (damage × density, $F_{1,178} = 5.05$, $P = 0.026$); at low density final B:As decreased by 7 % in damaged plants, while at high density B:A increased by 5 % with damage (Fig. 3). There was also genetic variation in the effect of damage on final B:A (damage × genotype, $F_{8,178} = 3.07$, $P = 0.003$).

Discussion

Our results suggest that intraspecific competition is likely to facilitate the evolution of tolerance in *S. carolinense*. Plants growing at higher density had greater phenotypic expression of tolerance (due to root overcompensation; Fig. 1), more genetic variation in tolerance (Fig. 2), and thus greater opportunity for selection. Moreover, at high density, tolerance provided fitness benefits for damaged plants (Table 3), and was not costly (Table 4). Other studies have also found that the evolution of tolerance may be affected by the environment (reviewed by Núñez-Farfán et al. 2007); however, none of the previous studies examined the evolutionary effects of intraspecific competition.

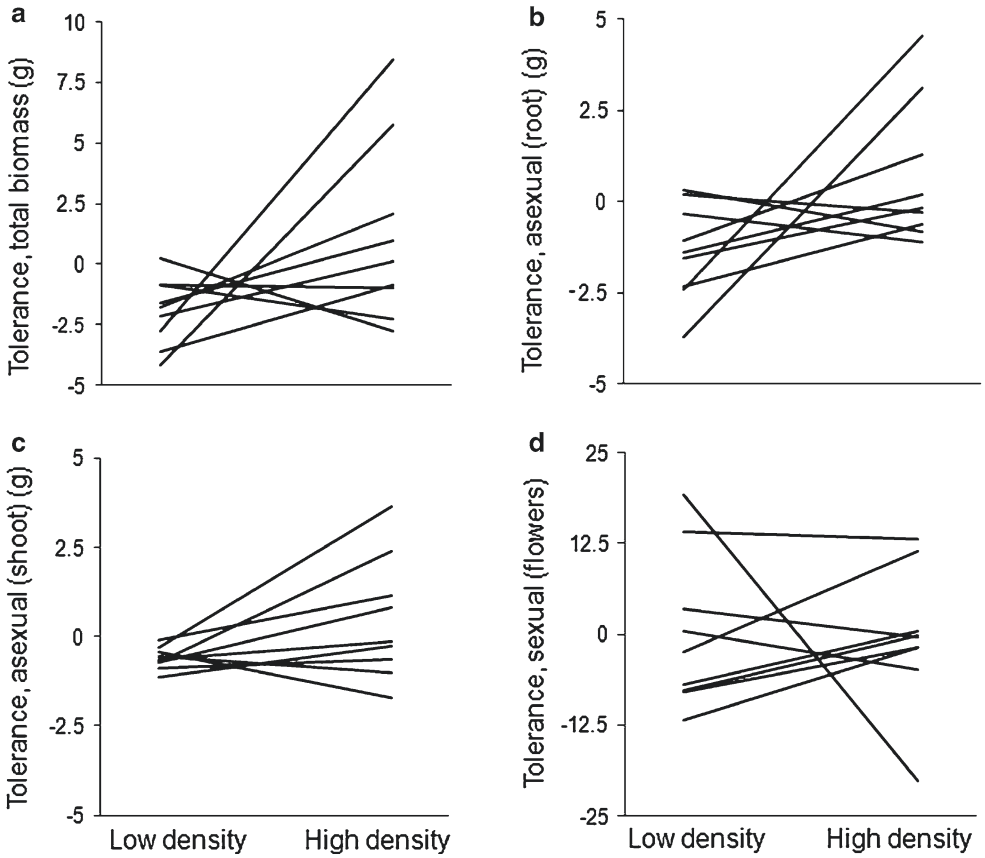

Fig. 2 Reaction norms for tolerance of nine clonal lines grown at low and high competitor densities. Tolerance was measured as the difference between damaged and undamaged individuals at each density for **a** total biomass, **b** root biomass, **c** shoot biomass, and **d** flower number

Table 3 Adaptive value of tolerance, measured as the covariance between damaged fitness and tolerance (damaged fitness − undamaged fitness) at low and high density

Density	Fitness measure	Artifact	Corrected covariance	2.5 % CI	97.5 % CI
Low	Total mass	−2.20	2.14	−1.37	4.65
Low	Flower no.	−40.73	193.24	−30.51	400.34
Low	Root mass	−1.14	2.08	−0.02	3.23
Low	Shoot mass	−0.27	0.10	−0.12	0.35
High	Total mass	−1.71	8.21	**0.52**	**17.49**
High	Flower no.	−13.13	47.14	−1.21	99.88
High	Root mass	−0.43	1.97	**0.15**	**4.25**
High	Shoot mass	−0.43	2.03	**0.12**	**4.06**

95 % CI for corrected covariances that significantly differ from zero shown in bold

Table 4 Costs of tolerance, measured as the covariance between undamaged fitness and tolerance (damaged fitness − undamaged fitness) at low and high density

Density	Fitness measure	Artifact	Corrected covariance	2.5 % CI	97.5 % CI
Low	Total mass	−5.12	2.94	**0.16**	**6.66**
Low	Flower no.	−40.95	66.90	−84.00	229.75
Low	Root mass	−2.66	1.77	**0.18**	**3.71**
Low	Shoot mass	−0.72	0.45	**0.04**	**0.98**
High	Total mass	−1.24	0.29	−3.00	4.21
High	Flower no.	−9.79	−40.66	−102.30	13.75
High	Root mass	−0.30	−0.08	−0.71	0.52
High	Shoot mass	−0.34	−0.14	−0.78	1.39

95 % CI for corrected covariances that significantly differ from zero shown in bold

Our study suggests that these effects should be further explored, given the ubiquity of competition in natural populations and its fitness consequences through pathways other than resource limitation.

Effects of competition on the phenotypic expression of tolerance

We found greater phenotypic expression of tolerance at high density, which was likely due to a compensatory response. At low density, undamaged biomass covaried positively with tolerance (Table 4), suggesting that intrinsic growth traits contributed substantially to tolerance in the absence of competition. In contrast, plants

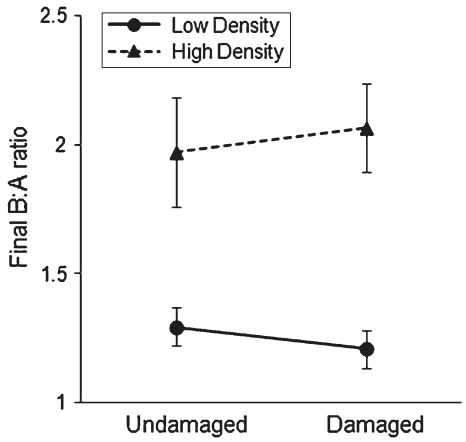

Fig. 3 Ratio of final belowground:aboveground biomass (B:A) for undamaged versus damaged plants grown at high and low competitor densities. B:A was calculated as root biomass/(shoot + estimated flower biomass). The slopes of these lines are significantly different in a univariate ANOVA, damage × density interaction $F_{1,178} = 5.05$, $P = 0.03$

overcompensated at high density even after controlling for the effects of initial size (i.e., intrinsic growth). Increased tolerance at high density has been attributed to an increased ability of damaged plants to catch-up when the growth of neighbors is constrained by competition (Hilbert et al. 1981). However, this hypothesis cannot explain the relative root and total biomass overcompensation we observed at high density, which likely resulted from a plastic shift in resource allocation not induced at low density. The limiting resource model of Wise and Abrahamson (2005) also predicts greater tolerance under limited resources given certain conditions; our data cannot address this model because we did not identify or manipulate specific resources limiting growth in each environment.

One trait likely mediating compensatory ability under competition is the relative allocation of resources below and aboveground, since B:As in perennials are often affected by both damage (e.g., Fowler and Rausher 1985; Bruna and Ribeiro 2005) and competition (e.g., Gurevitch et al. 1990; Berendse and Moller 2009). We found that B:A increased more with damage at high density than at low density (density × damage interaction; Fig. 3). Any effects of damage on B:A in this study were probably not simply due to the damage treatment reducing aboveground biomass (and thus increasing B:A); at low density, damage actually decreased the final B:A by 7 % (Fig. 3). The observed interactive effect of damage and competition on B:A may be the result of either adaptive phenotypic plasticity and/or effects of stress on plant development. For example, competition may alter wound signaling involved in adaptive plastic responses to damage; in the congener *S. nigrum*, damage resulted in downregulation of systemin precursors and greater tolerance in the presence versus

absence of conspecifics (Schmidt and Baldwin 2009). Alternatively, plants under the combined stresses of herbivory and competition may have been reduced to an earlier point along a fixed developmental trajectory; root:shoot ratio often decreases as plants mature (Evans 1972; Coleman et al. 1994). Since we did not measure B:A across plant development, we cannot distinguish between the two possibilities. Although studies of other species have also found changes in relative allocation to roots due to an interaction of damage and competition or nutrient availability, results are not consistent; root biomass can either increase (Huhta et al. 2000) or decrease (Stevens et al. 2008) with damage under competition. These differences in effects of damage on below- versus aboveground allocation may be due to experimental methods (e.g., timing of biomass measurements after damage) or other aspects of the environment, such as the type or density of competitor (e.g., intra- vs. interspecific), the location of the study (e.g., greenhouse vs. field), or the identity of the damaging herbivore. For example, without competitors, Wise and Cummins (2006) found decreased *S. carolinense* root biomass in response to folivory by a leaf-sucking lace bug, while we found increased biomass in response to chewing damage by *L. juncta* and *S. exigua* under competition.

Competition and the evolutionary potential of tolerance

Two of our results suggest that competition increases the opportunity for selection on traits conferring tolerance in *S. carolinense*. First, we observed genetic variation in tolerance only in the presence of competitors (Table 2). Second, the expression of genetic variation in tolerance via shoot biomass, which may contribute to both sexual and asexual reproduction in plants (Arendt 1997), was significantly greater under competition (Fig. 2c). Although we included only nine genotypes in this study, we selected them to encompass a wide range of tolerance, so it is notable that this variation was not expressed at low density. While large natural populations may contain genetic variation in tolerance even at low density (Tiffin and Rausher 1999; Fornoni and Núñez-Farfán 2000; Juenger and Bergelson 2000), our clonal lines originated from several different populations, so the genotypes in our experiment were likely more variable than in any single population. Regardless, these results add to evidence that intraspecific competition often increases the amount of variation expressed in plant traits (Miller et al. 1994; Winn and Miller 1995; but see Shaw 1986).

We also found changes in the rank order of tolerance among genotypes growing at high versus low densities, such that no genotype had uniformly high tolerance across density environments. If genotypes in natural populations also vary in tolerance expression among competitive environments, and if the constraints we found on the expression of tolerance act similarly in the field, this genotype-by-environment interaction is expected to maintain genetic variation in tolerance when competitor densities vary. *S. carolinense* is commonly found in intermediate to frequently disturbed habitats, which may lead to significant temporal variation in competitor densities. The observed trade-off between tolerance at high and low densities is likely driven by a negative correlation between traits conferring tolerance in each environment, including those associated with morphology, physiology, or growth. For example, intrinsic growth rate could be negatively correlated with traits underlying compensatory ability. Adaptive plant responses to stress (including herbivory) are often associated with low intrinsic growth rates (reviewed by Chapin et al. 1993; Arendt 1997), and the relative fitness effects of intrinsic growth traits and compensatory ability are known to differ among resource levels (Hochwender et al. 2000).

Competition affects costs and benefits of tolerance

We tested whether intraspecific competition changed the adaptive landscape of tolerance, or the relationship between fitness and traits conferring tolerance. We predicted competitor density would affect this relationship for both damaged plants (where tolerance is expected to be adaptive) and undamaged plants (where tolerance is expected to be costly); the combination of these competitor effects should result in different evolutionary trajectories or optima across competitive environments for traits conferring tolerance. In our study, competition affected when tolerance was adaptive, or positively correlated with fitness after damage; as predicted, we found that tolerance was adaptive only at high density (Table 3). While it has been shown that the competitive environment can alter selection on other plastic traits in plants (Weinig 2000; McGoey and Stinchcombe 2009; Boege 2010), few studies have documented changes in the adaptive value of tolerance due to variation in factors associated with competition (Hochwender et al. 2000). There are at least two mechanisms by which competition could affect the adaptive value of tolerance. First, because the benefits and costs of tolerance can vary among plant life stages (Boege et al. 2007), shifts in growth trajectories and developmental timing due to competition could affect the net fitness benefit of tolerance, especially when damage occurs at only one developmental stage, as in this study. Second, competition (and associated effects on plant size) may influence the adaptive value of tolerance via the differential effects of intrinsic growth and compensatory traits on fitness after damage. In low density environments, fitness after damage may be more a function

of intrinsic growth traits if larger plants suffer less from tissue loss (Hendrix 1979). At high density, competition may constrain plant size so that compensatory responses to damage contribute more to fitness, as has been shown under nutrient limitation (Hochwender et al. 2000). Therefore, compensatory traits may be most adaptive at high density, which is what we found in this experiment (Table 3).

We did not detect fitness costs of tolerance among undamaged plants in either competitive environment (Table 4). In fact, in the absence of competition, we found that traits that enhanced tolerance also improved fitness in the absence of herbivores. These results are striking because previous studies have found tolerance to be either costly (Siemens et al. 2003) or to have no effect on undamaged fitness (Hochwender et al. 2000; Stevens et al. 2007) when plants are grown in non-competitive or high nutrient environments. It is likely that traits resulting in greater tolerance at low density are positively associated with intrinsic growth, due to the significant positive covariance of undamaged biomass (an estimate of intrinsic growth) and tolerance in this environment (Table 3). High intrinsic growth is predicted to be beneficial even when plants remain undamaged, since they often result in increased size or reproduction in low stress environments (Arendt 1997).

Conclusions: expected outcomes of natural selection on tolerance

If the expression and evolutionary constraints on tolerance shown in our greenhouse study are also similar in the field, we can use the results of our study to predict how tolerance may evolve in *S. carolinense* and other disturbance-adapted perennial plants whose populations face spatial or temporal variation in herbivory and competition. First, we predict that increased tolerance should evolve when populations commonly experience high density environments with herbivores, the conditions under which tolerance positively correlates with fitness and is not constrained by reduced genetic variation or trade-offs. Additionally, herbivore pressure can be positively related to plant density in some systems (Root 1973), which should increase selection pressure on plant defenses. Second, we predict that spatio-temporal variability in competition may lead to the maintenance of genetic variation in tolerance due to the trade-off we observed between tolerance at high and low densities (Fig. 2a). Finally, we predict that tolerance via asexual reproduction, rather than sexual reproduction, is most likely to evolve in response to variation in density, because both the expression of the root biomass component of the tolerance phenotype and its genetic variation was most consistently affected by competition. Like many perennials, asexual reproduction from rhizomatous growth is the dominant mode of recruitment for *S. carolinense*. Because there is a strong correlation between aboveground biomass and root biomass in the previous year (N. Underwood and S. Halpern, unpublished data) and between stem size 1 year and asexual progeny the following year (N. Underwood and S. Halpern, in review), the greatest effects of tolerance on fitness may be seen in subsequent years. To date, nearly all cost of tolerance studies in perennials (including this one) have been performed over one growing season (Hochwender et al. 2000; Siemens et al. 2003; Stevens et al. 2007; Manzaneda et al. 2010; Hakes and Cronin 2011; but see Huhta et al. 2009), emphasizing the need for evolution of tolerance studies that span multiple years.

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