Research

Oikos 116: 975–985, 2007
doi: 10.1111/j.2007.0030-1299.15705.x,
Copyright © Oikos 2007, ISSN 0030-1299
Subject Editor: Kathy Suding, Accepted 2 March 2007

Life-history consequences of vegetative damage in scarlet gilia, a monocarpic plant

Alison K. Brody, Mary V. Price and Nickolas M. Waser

Alison K. Brody (akbrody@uvm.edu), Mary V. Price and Nickolas M. Waser, Rocky Mountain Biological Laboratory, P.O. Box 519, Crested Butte, CO 81224, USA. AKB also at: Dept of Biology, Univ. of Vermont, Burlington, VT 05405, USA. MVP and NMW also at: Dept of Biology, Univ. of California, Riverside, CA 92521, USA.

Although herbivory can occur throughout a plant's life, little is known about relative fitness impacts of damage at different life stages. In the long-lived monocarpic wildflower, *Ipomopsis aggregata* (scarlet gilia), for example, the response to browsing by ungulates in the year of flowering has been studied extensively, whereas damage and its fitness consequences during the preceding years of vegetative growth remain largely unexplored. As part of a long-term demographic study of *I. aggregata*, we mapped 5324 individual seedlings belonging to two annual cohorts in three natural populations, and followed these plants throughout their lives. Of the 30.4% of plants that survived past seedling stage, 15.3% suffered observable damage to the apical meristem of their single rosette of leaves, usually resulting in multiple rosettes in vegetative plants and multiple flowering stalks in plants that survived to flower. Vegetative damage reduced survival to flowering by ca 30%, and slowed growth, leading to an average delay in flowering of over one yr relative to undamaged plants. On average, damaged plants were 136% larger than undamaged members of their cohort in the summer before flowering, but produced only 86% as many flowers, and achieved 78% the overall fitness compared to undamaged plants when damage effects were integrated across their life history. Previously published studies from the same sites suggested that plants suffering browsing during only the year of flowering, achieved 47% of the fitness of unbrowsed plants. Our results indicate that plants are better able to recover from vegetative damage than from loss of their initial reproductive effort, perhaps because of the longer time over which recovery is possible in the former case. These results underscore the importance of studying the consequences of damage across the entire life history.

Plants may experience damage from herbivores, pathogens, or physical disturbance at any stage of their lives, and the consequences can be variable, ranging from outright mortality to altered life history and changes in vegetative or floral architecture. Although conventional wisdom suggests that damage will be detrimental even if it is sublethal, in some cases plants appear able to tolerate damage with little effect on final fitness (''compensation''), or even to enjoy an increment in fitness (''overcompensation''). Which outcome occurs may depend critically not only on the nature of damage but on its timing in the life cycle, the life history of the plant, and the environmental context (Louda and Potvin 1995, Huhta et al. 2000, Stinchcombe 2002a, Warner and Cushman 2002, Ehrlén 2003, Boege 2005, Boege and Marquis 2005).

For long-lived perennial plants, damage in the early stages – if not lethal – can induce changes in resource allocation from growth to defense (Stamp 2003, Farnsworth 2004), slow the time to reach reproductive maturity (Ehrlén 2003), and/or damage can cause changes in plant architecture that may affect photosynthetic efficiency (Strauss and Agrawal 1999, Tiffin 2000), interactions with pollinators (Mothershead and Marquis 2000, Gómez 2003), or alter a plant's ability to resist further damage and tolerate (compensate for) the initial damage. In later stages, particularly when damage occurs to reproductive tissue, there may be little time for plants to recover from such damage and tolerance may be impossible (Juenger and Bergelson 1998, 2000). If damage occurs early in a plant's life history, there may be ample time to recover lost tissue,

but replacement may come at a cost to future reproduction either by delaying the onset of flowering and/or by reducing investment in flowers or seeds.

Although a number of studies compare different plant responses to herbivory over leaf age or plant stage (Karban and Thaler 1999, Loney et al. 2006, Shiojiri and Karban 2006), or compare herbivory on young vs adult leaves (reviewed by Boege and Marquis 2005), there is a notable lack of studies that integrate the effects of damage over the entire life-history from seedling to reproductive adult (Ehrlén 2003). Our knowledge of plant response integrated over the life-history is important not only because plant response in terms of resource allocation to defense versus growth may change, but also the long-term fitness effects of early stage damage are missed if one only examines responses to adult-stage herbivory. Characterizing the response to damage throughout ontogeny, and its effect on the full life-history, is critical to our understanding of the ecological and evolutionary responses of plants to herbivores and for predicting whether and how tolerance will evolve (Boege and Marquis 2005).

Scarlet gilia (*Ipomopsis aggregata*, Polemoniaceae) provides an example of extreme variation in tolerance to herbivory incurred by flowering-stage plants. Elongating flowering stalks are subject to browsing, primarily by ungulates. Browsed plants respond to the loss of the apical meristem by producing multiple flowering stalks instead of the normal single stalk. Relative reproductive success of browsed plants has been reported to vary widely among populations, ranging from considerable overcompensation (Paige and Whitham 1987, Gronemeyer et al. 1997, Paige 1999), to equal compensation or extreme undercompensation (Bergelson and Crawley 1992a, 1992b, Juenger and Bergelson 1997, 2000, Paige 1999, Freeman et al. 2003, Sharaf and Price 2004). The degree of compensation has been attributed to a number of factors, including plant size, seasonal timing and severity of browsing, temperature and length of growing season, competitive environment, and resource availability (Maschinski and Whitham 1989, Juenger and Bergelson 1997, 2000, Paige 1999, Freeman et al. 2003, Sharaf and Price 2004). Larger plants appear to be able to tolerate damage to a greater degree than smaller ones (Sharaf and Price 2004, Juenger et al. 2005), plants browsed early in the season are more tolerant than those browsed later (Freeman et al. 2003, Juenger et al. 2005), and plants with greater resources available to them or living in an environment with few competitors are better able to tolerate browsing than those with limited resources (Maschinski and Whitham 1989, but see Juenger and Bergelson 1997).

All of these studies, however, have focused on damage that occurs after plants have initiated flowering. In the course of a long-term demographic study of *I. aggregata*, we recognized that some plants suffer damage while they are vegetative rosettes. Vegetative damage rarely appears as the removal of part or all of a leaf, and more often manifests itself as a misshapen rosette, multiple rosettes, or a nub of only a few mm of vegetative tissue attached to a rootstock. In all cases, it is quite distinct from flowering-stage browsing by ungulates that leaves a central stump of tissue subtended by elongating flowering stalks. The recognition of a separate class of vegetative-stage damage presented us the opportunity to compare consequences of damage at different life stages. We ask the following questions: 1) what is the probability of damage in vegetative stage? 2) How does vegetative damage influence survival to flowering, time to reproductive maturity, final fecundity, and (combining these) final fitness? 3) What is the relative impact on reproduction of damage before vs during the flowering year?

Material and methods

Study system

Ipomopsis aggregata is a monocarpic perennial herb of the western USA. The species is common in dry meadows around the Rocky Mountain Biological Laboratory (RMBL) in western Colorado (elevation 3000 m). Plants grow vegetatively for 2–10 years, flower once, and die; iteroparity is very rare (Waser and Price 1989). The flowers are highly self-sterile due to late-acting self-incompatibility (Sage et al. 2006), and are pollinated primarily by broadtailed and rufous hummingbirds (*Selasphorus platycerus* and *S. rufus*), with additional contributions from insects (Price et al. 2005).

Browsing of inflorescences

In the year of flowering, undamaged plants produce a single inflorescence stalk that bears up to several hundred red, trumpet-shaped flowers (Campbell 1989). However, some plants have their elongating inflorescence bitten off by large browsing mammals (Paige and Whitham 1987, Bergelson and Crawley 1992a, Sharaf and Price 2004). Browsing around the RMBL is by ungulates (mule deer, *Odocoileus hemionus* and elk, *Cervus elaphus*), and perhaps also by large rodents (marmots, *Marmota flaviventris*). Such damage usually releases lateral meristems from apical dominance (Aarssen 1995) resulting in replacement of the initial inflorescence by multiple secondary lateral inflorescences (Paige and Whitham 1987) that emerge from below a browsing scar.

Damage in vegetative stage

Previous demographic studies at the RMBL (Waser and Price 1989, Campbell and Waser 2007) indicate that most seedlings emerge soon after snowmelt in the year after they are produced. By the end of this first summer, surviving juveniles appear as a single small rosette of highly-dissected leaves. Vegetative rosettes increase in leaf number and leaf length in subsequent years, and eventually bolt in the flowering year. Plants maintain a single rosette unless the apical meristem is damaged. Those damaged in vegetative stage most often (but not always) develop multiple rosettes from lateral meristems that all emerge from the central root crown. These connected rosettes ultimately may produce inflorescences. Usually the multiple inflorescences appear simultaneously in a single summer, so that the flowering plant resembles those that were browsed in their flowering year, except that it lacks the scar where an original central inflorescence was bitten off. The uncommon instances in which connected rosettes flower in different summers yield most of the rare observations of iteroparity around the RMBL. Although rosette attrition sometimes leaves damaged plants with a single rosette, the damage remains discernable because remaining rosettes emerge laterally from a scarred root crown, giving the plant a markedly disfigured and asymmetrical appearance.

In comparison with damage to inflorescences, whose perpetrators are known for the most part, vegetative damage remains enigmatic. We three authors have collectively studied *I. aggregata* for over 80 person-summers at the RMBL. We have never directly observed vegetative plants being browsed, and have only rarely detected bitten-off leaves during the summer growing season or seen frost-withered apical meristems following spring snowmelt. Damage therefore appears to occur during autumn or winter. Possible agents of vegetative damage include herbivory by insects or by small mammals that remain active during the winter at the soil–snow interface (these include pocket gophers, *Thomomys* spp., and voles, *Microtus* spp.; Juenger and Bergelson 2000), and/or mechanical abrasion of rosettes or root crowns.

Demographic methods

In early July 1995 we established nine permanent study plots in each of two dry meadow sites at the RMBL (Treasury and Bench), and six plots in a third meadow (Vera). The meadows are separated from one another by 150–575 m. Each of the 24 plots consisted of four, 1×1 m subplots, marked at the corners with permanent markers. Using a 1×1 m frame strung into 10×10 cm squares, we mapped to the nearest cm all *I. aggregata* seedlings that were emerging in the plots. This process was repeated in late June 1996, yielding two cohorts of plants.

Mapped plants could be relocated subsequently based on their coordinates within a plot and distinguished from nearby younger recruits, or pre-existing older plants, based on size of the root crown. We returned to re-census in late June each summer from 1996 until no individuals remained alive in 2004. During censuses we scored the life history stage (dead, juvenile, or flowering) of each mapped plant. For juvenile individuals we recorded rosette number and scored size of each rosette as the product of maximum leaf length and total leaf number. For flowering adults we noted the number of inflorescence stalks and counted the total number of flowers produced over the flowering season as an estimate of fecundity. In several years we also noted whether the flowering stalk had been browsed by ungulates. We did not go further to count seeds, because other goals of the long-term study dictated that we not disturb natural seed input to the plots by harvesting fruits.

From 1995 through 2004 we also counted the number of expanded fruits produced by all *I. aggregata* plants within 1 m of each plot, and in each study meadow we counted the number of seeds and expanded fruits produced by five flowering individuals. From these data we estimated the number of seeds that fell naturally into each plot. We also counted the number of seedlings that emerged in each plot from 1995 through 2005.

Life-table parameterization

From our censuses we constructed separate life tables for two vegetative damage categories (damaged, undamaged), three study sites (Treasury, Bench, Vera), and two seedling cohorts (1995, 1996), as well as pooled overall tables for damaged vs undamaged plants. Seeds were scored as age 0, seedlings as age 1, and so on to the maximum age of eight years. We used rosette morphology and juvenile rosette number to classify individual plants as damaged and to pinpoint the age at which damage occurred. Damage causes the production of additional rosettes or it produces an obviously disfigured or stunted rosette. Undamaged plants produce a single, symmetrical rosette. We noted in which census the change in rosette morphology or number occurred and the plant was thereafter classified as "damaged".

We estimated germination rate (p_1) for the 1996 cohort by dividing 1996 seedling counts by estimated seed input in late summer 1995. Because from 1996 to 2005 this rate was a strong positive function of the previous winter's precipitation, we could estimate 1995

germination rates from site-specific linear regression of germination rate on winter precipitation.

Because our initial censuses occurred soon after germination, when no living seedlings showed visible signs of vegetative damage, we could not determine whether a seedling that disappeared in its first year had been damaged. Hence we conservatively assumed that seedling survival to age 2 (p_2) was equivalent for undamaged and damaged plants and calculated it as the total number of age two individuals, whether damaged or not, divided by the total number of seedlings. For subsequent ages we estimated age-specific survival (see next paragraph) and fecundity separately for damaged and undamaged plants in each cohort and study site. To construct life tables, we followed Lenski and Service (1982), pooling across individual life histories for each group. Survival (p_x) was estimated by dividing number of survivors to age x by the number alive the previous year. Fecundity (m_x) was calculated as the total number of flowers produced by a group of age x, divided by the number of surviving individuals in that group.

The procedure just described unavoidably underestimates effects of vegetative damage on lifetime fitness to the extent that such damage kills plants before they can be observed in the next annual census. Figure 1 depicts in schematic form the transitions of plants from undamaged to damaged and from live to dead, across the interval between sequential censuses. Inspection of the figure illustrates that it is impossible to determine the cause of mortality for previously undamaged plants that disappear, a problem that could be reduced but not eliminated if more frequent censuses were feasible (which they were not in this case). We return to this point in the Discussion. Figure 1 also illustrates that new individuals enter the category of damaged plants that we observed in each census, just as others leave this category due to mortality. In other words, damaged plants comprised a series of identifiable cohorts damaged at age 1 (in the seedling year), age 2, and so on. Estimates of age-specific survival for damaged plants were calculated as the weighted average of survival across these damage cohorts. For example, survival from 1997 to 1998 at a given site for damaged members of the 1995 seedling cohort was the average of survival of individuals from 1997 to 1998 that first exhibited damage in the 1996 census, and those that first exhibited damage in the 1997 census, weighted by numbers in those two cohorts observed in the 1997 census.

We used the IML procedure of SAS 8.2 to calculate fitness as the finite rate of population increase (λ) for each life table, using nine age classes and a birth-pulse, postbreeding-census formulation. This measure of fitness incorporates not only survival and fecundity, but age at flowering. Earlier flowering increases the value of λ, all else equal, as is reasonable for a fitness measure except under unusual circumstances (Hogendyk and Estabrook 1984, McGraw and Caswell 1996). We estimated fecundity as the number of flowers produced per flowering individual, which is a reasonable relative measure given that seed

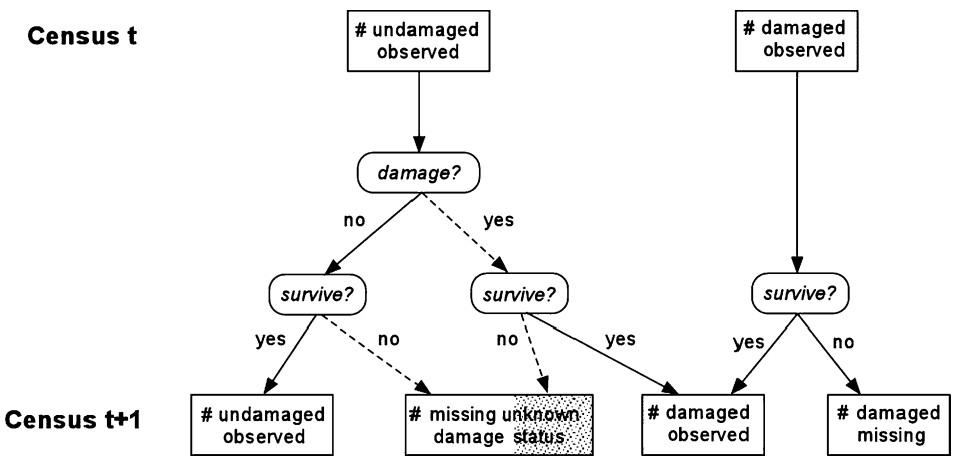

Fig. 1. A schematic showing possible transitions between two successive censuses of plants in the field. In our case censuses were annual, but conclusions would apply for censuses at shorter or longer intervals. Undamaged plants observed in one census can escape damage, live, and appear in the next census. Alternatively, they can experience damage and survive to appear as newly damaged plants in the next census. These transitions are knowable and are depicted with solid arrows. A major ambiguity arises, however, in that previously-undamaged plants which disappear between censuses may or may not have been damaged before they died. Thus the box labelled "# missing damage status unknown" contains contributions from background mortality due to causes other than damage (unshaded), and additional mortality due to damage (shaded); these relative contributions are unknowable and are depicted with dashed arrows.

number increases linearly with flower number (Campbell 1998).

Analyses

We analyzed damage frequencies and survival to flowering using contingency analysis, and age at flowering, size pre-flowering, fecundity, and λ using ANOVA. We treated site and cohort as random effects and vegetative damage (yes or no) as a fixed effect. When full models yielded negative variance component estimates, we simplified models progressively, removing highly insignificant interaction terms and those that produced negative variance estimates. Because growth rates were age-specific, we used paired-t-tests (2 cohorts $\times$ 3 sites $\times$ 2 ages = 12 pairs) to compare the change in size for damaged plants in the year of damage with growth of undamaged plants of the same age. We restricted our comparisons to growth in the two post-seedling years because sample sizes were very small for other age classes. All ANOVA and contingency analyses were run in JMP ver. 5 (SAS Inst.).

We characterized the contributions of individual demographic variables to the difference in fitness between damaged and undamaged plants in a life-table response analysis. This analysis first calculates sensitivity, the partial derivative of λ to a change in an element of a reference population projection matrix (Caswell 2001). Contributions of individual matrix elements to the effect of damage on λ are then determined by the element-by-element product of the matrix of differences between the damaged projection matrix and the reference matrix, multiplied by the matrix of sensitivities for the reference matrix. We used the undamaged projection matrix as the reference. All matrix manipulations were performed with the IML procedure of SAS ver. 8.2.

Results

Vegetative damage rates

We mapped 5324 seedlings (age 1) across the three permanent study sites. Of these, 3705 failed to reappear at age 2 (1 year post-emergence). Because we could not assign these plants to damaged/undamaged categories (Fig. 1), our analyses of effects of damage on plant fates beyond the seedling year are based on 1619 plants that survived to age 2, representing 30.4% of the seedlings initially mapped.

Of the plants that survived to age 2, 247 (mean = 15.3%, range 8–23% across site-cohort combinations), were visibly damaged in vegetative state at some point thereafter. Damage rate did not vary between the 1995 and 1996 seedling cohorts (Wald $\chi^2 = 0.782$, DF = 1, $p = 0.377$), but did vary across sites ($\chi^2 = 6.181$, DF = 2, $p = 0.046$), and with the site $\times$ cohort interaction ($\chi^2 = 13.109$, DF = 2, $p = 0.001$).

Growth trajectories

Damaged and undamaged plants did not differ in size in the census before damage occurred (paired comparison of means for cohort–site–age damaged; 11 pairs, mean difference in size = 0.013 cm, $t = 0.004$, $p = 0.997$), indicating that damage was not size-selective. However, damaged plants grew less over the year following damage than did undamaged plants of the same age, cohort, and site (paired comparison for means of cohort–site–age; 12 pairs, mean difference in growth = 12.60 cm, $t = 4.691$, $p = 0.0007$).

Although damaged plants grew less in the year they were damaged, they grew larger before they flowered for both cohorts at all sites, albeit to different extents (site $\times$ cohort $\times$ damage interaction $F_{2,353} = 3.279$, $p = 0.039$). When this interaction term was removed from the model, rosette size in the pre-flowering year was significantly greater for damaged (120.5 cm) than undamaged plants (88.5 cm; $F_{1,355} = 9.262$, $p = 0.0025$).

Survival to flowering

Of the 1619 plants that reached age 2 (one year past emergence as seedlings), 449 flowered in that year or survived to flower in a subsequent summer. Damaged plants survived to flowering only 69.6% as well on average as undamaged plants (20.2% survival vs 29.1%), a statistically significant difference (Wald $\chi^2 = 4.314$, $p = 0.039$). In addition, the site $\times$ damage category interaction was significant ($\chi^2 = 8.333$, $p = 0.016$); a far smaller fraction of damaged plants survived to flower at two sites (Treasury, Bench), but a slightly larger fraction survived at the third site (Vera; Table 1).

Age at reproduction

Vegetative damage delayed the age at flowering over all sites and cohorts (Table 1). Expressed as least squares means, the ages at flowering for undamaged and damaged plants were respectively 2.02 and 3.23 years ($F_{1,444} = 98.004$, $p < 0.0001$). Cohort and site also affected age to reproduction, but did not interact significantly with vegetative damage.

Table 1. Summary of means for 6 life history variables for three sites (Bench, Treasury, Vera), two seedling cohorts (1995, 1996), and two levels of vegetative damage (undamaged = "U", damaged = "D").

Variable	Bench				Treasury				Vera			
	1995		1996		1995		1996		1995		1996	
	U	D	U	D	U	D	U	D	U	D	U	D
Growth rate	30.78	17.81	35.31	31.58	75.80	68.36	60.82	28.39	44.92	19.78	32.08	35.02
Size pre-flowering	78.07	127.88	67.65	83.76	107.72	153.30	93.07	113.40	121.06	124.56	64.33	126.77
% survival	22.5	17.1	31.4	18.8	37.9	20.9	43.6	16.7	15.6	26.5	20.6	20.9
Age at flowering	3.68	4.61	3.31	4.76	2.80	3.78	2.49	3.88	2.930	4.58	2.93	3.66
Flowers	62.85	70.84	69.31	46.56	126.98	102.57	113.61	123.26	110.14	104.42	76.71	67.68
λ	0.836	0.519	0.907	0.676	1.372	0.828	1.025	0.654	0.605	0.684	0.787	0.873

Growth rate = change in size (cm of leaf length) during the year of first damage (weighted average of means for ages 2 and 3). Pre-flowering size = rosette size (cm of leaf length) in the year prior to flowering (least-squares mean). % survival = percentage of 2-year-old plants that survived to flower. Age at flowering = age in year of flowering (least-squares mean). Flowers = total flowers produced (least-squares mean). λ = finite rate of population increase.

Fecundity

Plants that were damaged in vegetative stage and survived to flower produced on average only 85.8% as many flowers as their undamaged counterparts (79.53 flowers vs 92.74 flowers). This difference was not statistically significant overall ($p = 0.592$), largely because there was a pronounced, nearly significant ($p = 0.056$), site × damage interaction. Damaged plants produced fewer flowers at Bench and Treasury, but more flowers at Vera (Table 1), a fecundity effect parallel to the pattern of survival to flowering just described.

Overall fitness effect of vegetative damage

Using an overall life table pooled across sites and cohorts (Fig. 2), and assuming that each flower produced a single seed on average, λ for visibly damaged plants was only 77.6% as large as that for undamaged plants ($\lambda = 0.726$ vs 0.936, respectively). This fitness difference was not significant statistically ($F_{1,2} = 1.851$, $p = 0.307$) in a mixed-model ANOVA of λ values calculated individually for each site-cohort combination. The site × damage interaction approached significance ($p = 0.170$), because damaged plants achieved slightly higher λ values than undamaged plants at one site (Vera) as opposed to substantially lower values at the other two sites (Table 1). The final ANOVA model included damage category as a fixed effect, and the site × damage interaction as a random effect, but omitted cohort, cohort × damage, and site as random effects, since they returned negative variance component estimates when included in the model.

Contributions to damage effects on λ

Demographic perturbation analysis (Caswell 2001) showed that the difference in λ between damaged and undamaged plants was largely due to fecundity at age 3 (F_3), followed by substantially smaller contribution from fecundity at age 2 (Table 2). Separate analyses by cohort and site (not shown) presented a parallel picture: in 6 of 6 individual analyses, F_3 made the largest contribution.

Correlation between vegetative damage and flower browsing

For 217 of the 449 plants that flowered we recorded whether they suffered browsing of their elongating central inflorescence stalk. Of these, 36 (16.6%) were damaged in vegetative state, 104 (47.9%) were browsed

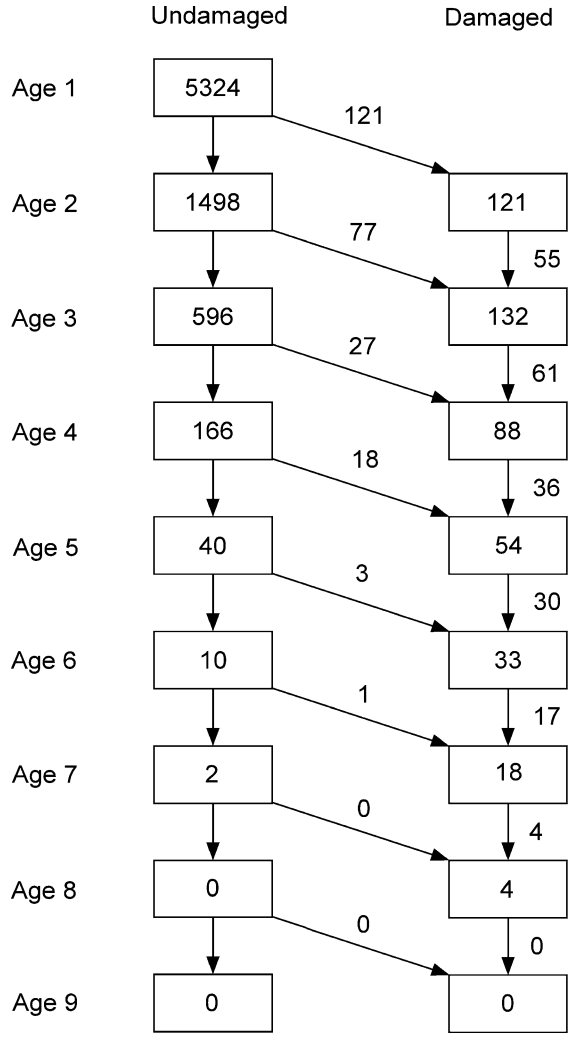

Fig. 2. A more explicit overall life cycle diagram for our *I. aggregata* plants, summarizing the fates of those that suffered observable damage in vegetative phase, vs no damage, pooled across sites and cohorts. 5324 plants were first mapped as seedlings (age 1), and were followed until they flowered and died or died without flowering (transitions to flowering are not shown). Numbers of individual plants surviving between annual censuses, and moving from undamaged to damaged categories, are given next to arrows indicating these transitions. As shown in Fig. 1, we cannot know that all loss of plants that were undamaged in a given census was independent of subsequent damage occurring before the next censuses. We have conservatively assigned all such loss to the ''undamaged'' category in calculating life tables, except that we assigned an equal probability for the transitions from age 1 to 2 for both ''undamaged'' and ''damaged'' categories, as explained in text. A subset of the mortality is due to flowering and dying (*I. aggregata* is monocarpic). These deaths are accounted for in the analyses as transitions to adult-stage.

in the year of flowering, and 15 (6.9%) suffered both types of damage. The probability of inflorescence browsing was statistically independent of vegetative damage (Wald $\chi^2 = 0.675$, DF = 1, $p = 0.412$).

Discussion

Damage to the apical meristem of the vegetative rosette is an appreciable risk in our populations of *I. aggregata.* In our 1995 and 1996 study cohorts, about one of every six plants that survived past the seedling stage suffered observable vegetative damage. Damage levels may be higher in some years; in 2006, for example, one of every four plants was visibly damaged in a survey in two of these same sites (A. K. Brody, unpubl.). Vegetative damage influences the architecture of plants, usually leading to multiple rosettes in the vegetative stage, and multiple flowering stalks in flowering plants, and less frequently causing misshapen single rosettes. As we found in this study, damage also alters life history. Visibly damaged plants survived to flower less frequently than undamaged ones, grew more slowly immediately after damage, and took substantially longer to reach flowering adulthood. Those that did survive to flower were larger in the summer before flowering than undamaged members of their cohort, but produced fewer flowers on average. Damage incurred an overall fitness cost of about one quarter.

Several damage effects were not statistically significant (i.e. effects of damage on flower number and λ), because the combined results average over opposing responses in different populations, i.e. tolerance to damage ranged from undercompensation in two populations to slight overcompensation in a third. That tolerance can vary with circumstances is well understood for herbivory in the flowering stage, with *Ipomopsis* again serving as an example. For *I. arizonica,* a close relative of *I. aggregata*, the response to flower browsing ranged from undercompensation to overcompensation, and tolerance was enhanced by reduced competition from conspecifics and by nutrient addition (Maschinski and Whitham 1989). Similarly, Juenger and Bergelson (1997) were able to enhance tolerance (although not to the level of overcompensation) by adding pollen and nutrients to clipped *I. aggregata* plants. Reviewing such evidence, Whitham et al. (1991) concluded that plants tolerate damage better in ''benign'' environments with few competitors and high levels of soil nutrients. Our results for vegetative damage mirror these earlier findings for inflorescence damage, in the sense that the three study sites may not be equally benign. Canonical discriminant analysis

Table 2. Age-specific survival and fertility for damaged and undamaged plants, pooled across sites and cohorts. $F_x = m_x \times p_x$; $p_x = L_x/L_{x-1}$ in this post-breeding, birth-pulse formulation. Sens = sensitivity of lambda to change in an element of the pooled projection matrix for undamaged plants (the reference matrix). Contribution = contribution of each matrix element to the difference in lambda between pooled life histories for undamaged and damaged plants.

Age (x)	Undamaged lambda = 0.936				Damaged lambda = 0.722		Contribution	
	p_x	$sens_{px}$	F_x	$sens_{Fx}$	p_x	F_x	p_x	F_x
1	0.010	3.111	0	0.332	0.010	0	0	0
2	0.304	0.843	1.54	0.035	0.304	0	0	0.055
3	0.419	0.113	18.14	0.012	0.455	4.99	−0.004	0.154
4	0.292	0.030	7.45	0.005	0.462	8.48	−0.005	−0.000
5	0.2700	0.004	4.80	0.000	0.4093	0.950	−0.001	0.001
6	0.2700	0.002	1.08	0.000	0.556	7.46	−0.000	−0.003
7	0.2220	0	5.00	0.000	0.515	0	0	−0.000
8	0	0	0	0.000	0.2225	0	0	−0.000
9	0	0	0	0	0	0	0	

indicated that the sites differ in five soil properties (Wilks' lambda = 0.511, $F_{8,48} = 2.396$, $p = 0.029$), and individually for soil depth ($p = 0.015$) and nitrate level ($p = 0.010$). Vera, the site in which damaged plants achieved slightly higher fitness, differs from the other two in having deeper soils with higher levels of nitrate, ammonia, and potassium. Understanding the range in expressions of tolerance will require a better understanding of variation in environmental factors, and of how plants respond to such environmental factors throughout development.

In a previously published study of inflorescence browsing at the RMBL in the same three study sites used here, plants with their elongating inflorescences clipped to mimic ungulate herbivory achieved significantly lower seed set than unclipped controls (Sharaf and Price 2004). Similarly, in other studies in nearby populations, the fitness of clipped inflorescences was significantly lower than unclipped plants or those that were naturally browsed by deer (Freeman et al. 2003). In populations further afield from those we studied, the response to clipping of the central inflorescence has been more variable, ranging from the production of more flowers (and, presumably, seeds) than unclipped or unbrowsed counterparts (Paige and Whitham 1987, Paige 1999) to significant fitness reductions (Bergelson and Crawley 1992a, 1992b, Bergelson et al. 1996, Freeman et al. 2003, Sharaf and Price 2004, Becklin and Kirkpatrick 2006). Although a purely observational study has obvious limitations – in particular, naturally-imposed herbivory is inherently biased by environmental variables that affect both fitness and herbivores (Tiffin and Inouye 2000), we did not clip *I. aggregata* plants for several reasons. First, and most importantly, the response of *I. aggregata* to clipping is not always akin to the response of plants to natural damage (Paige 1999, Freeman et al. 2003, Sharaf and Price 2004). In each of these cases, clipped plants at various sites in Colorado produced significantly fewer seeds than naturally browsed counterparts. Second, care must be taken to adequately mimic the type of damage inflicted by herbivores (Paige 1999, Inouye and Tiffin 2003) and because vegetative damage has not been studied before in this species, we had no a priori expectation of how many plants (if any) would be damaged as rosettes, the intensity of damage (i.e. how much tissue would be removed), how many of those would survive damage, and whether plants damaged in one year would be subsequently damaged in following years. Thus we could not impose levels or frequency of damage that would be realistically comparable to natural damage.

Comparisons of our results to those of others cited above and, in particular, to those of Sharaf and Price (2004) conducted in the same study sites, suggest a greater fitness consequence of flowering-stage than vegetative-phase damage. Plants damaged in vegetative phase achieved 78% of the fitness of undamaged plants – compared to clipped, adult (i.e. bolting) plants achieving 47% the fitness of controls (Sharaf and Price 2004). What might explain this apparent difference in tolerance to the two types of damage? Certainly the avenues available for plants to recover from damage differ in the two situations. Plants damaged as juveniles potentially have months or years in which to regain lost tissue and to accumulate the additional resources needed for flowering. In contrast, those damaged at the onset of flowering have only a matter of weeks in which to compensate.

It seems to us unsurprising, then, that inflorescence damage would have a stronger detectable fitness effect than vegetative damage. However, this conclusion ignores the fact that plants damaged in vegetative stage are smaller on average than those damaged while flowering, and potentially experience considerable damage-related mortality (inflorescence browsing rarely kills the plant outright). At this point we repeat a major caveat noted earlier (Methods): because we could not

detect individuals that were damaged after one census and disappeared before the next, we assigned all such disappearance to the ''undamaged'' category (or virtually all, in the case of the seedling to year 2 transition). Thus we may seriously underestimate the actual frequency of vegetative damage (it may exceed our estimate of ca 15%) and its consequences. A thought experiment may help to illustrate the point. If we modify the life tables by assuming that 25% of the plants that disappeared between seedlings and year 2 were damaged, rather than ca 7.5% that corresponds to our pooled estimate of survival for this transition (Methods), λ estimates for damaged plants are lower than those for undamaged plants at all sites (including Vera), and the overall statistical significance of damage on λ is now $p = 0.066$. Furthermore the pooled estimate of λ for damaged plants is 0.981 and for damaged is 0.569, or 58% as large – now much closer to the estimated 47% fitness of plants damaged in the summer of flowering. In summary, conclusions of overall damage frequency, fitness consequences, and relative tolerance to types of damage are (understandably) sensitive to the assumption one makes about los desaparecidos – the disappeared ones. Our assignment of all of them to ''undamaged'' is probably very conservative, but to an unknown – and unknowable – degree. Exact quantitative conclusions are problematic in the face of this unavoidable empirical limitation.

How do our qualitative results compare to those from the few other studies of damage to plants at different life stages? Ehrlén (2003) reported that vegetative damage to *Lathyrus* reduced survivorship, growth, and the probability of flowering, much as we found in *I. aggregata.* In morning glory (*Ipomoea*), herbivore damage to mature leaves did not affect plant fitness, while damage to cotyledons reduced fitness indirectly by reducing plant size (a correlate of seed set; Stinchcombe 2002b). Comparisons across life-stages of several grassland species showed that plants tolerate damage to adult leaves significantly more than damage to cotyledon and young leaves, although tolerance was not strictly linear with leaf age (del-Val and Crawley 2005). Vegetative damage to *Erysimum strictum* (a strict biennial) reduced all fitness measures in the subsequent flowering year (Piippo et al. 2005). Tolerance may increase when plants flower (Boege and Marquis 2005), although this may apply more to polycarpic (iteroparous) plants than to monocarpic ones (taking the original comparison above for *I. aggregata* at face value). In one of few studies to examine the link between herbivory on juveniles and population dynamics, Stanforth et al. (1997) suggested that early herbivory increased time to reproduction and was likely to reduce persistence for the endangered Pitcher's thistle, *Cirsium pitcheri*. Our results were similar, in that vegetative damage always increased time to reproduction. However, it must be made clear that we are not comparing leaf herbivory across different age classes. Ungulate herbivores remove the flowering stalk of adult-stage *I. aggregata* plants. Whereas the damage we describe is to rosette leaves. While these comparisons across studies are intriguing, overall it seems premature to generalize about how damage at different life stages, and tolerance to it, express themselves.

As alluded to in the Introduction, much heat, and some light, has ensued from the published discussion of tolerance to damage, i.e. compensation, in the genus *Ipomopsis.* The changes in plant architecture that we report here may shed a bit of additional light. Both vegetative and flowering-stage damage in *I. aggregata* produce similar multiple-stalked inflorescences, which may influence how we should interpret studies of browsing that do not explicitly recognize this potential source of ambiguity in the timing of damage. Early reports of overcompensation (Paige and Whitham 1987, Paige et al. 2001) made no mention of using presence of a browsing scar on the central inflorescence stalk as a criterion for determining whether multiple-stalked plants were flower-browsed or not. If the ''browsed'' category in these studies included plants that had been damaged in vegetative phase, then any apparent beneficial effect of ungulate browsing on fitness could be an overestimate: the true fitness of vegetatively browsed plants should incorporate the consequence of reduced survival to and delay in flowering, or any other fitness consequences that apply in the sites and years in question. The possibility that this potential source of error affected early reports of overcompensation in *I. aggregata* is supported by statements that browsed plants did not flower later in the season than unbrowsed plants (Paige and Whitham 1987). Subsequent studies have noted that plants that were damaged prior to the year of flowering (discerned by the lack of a browsing scar) flower essentially synchronously with undamaged plants, whereas plants whose elongating inflorescences have been clipped flower as much as 30 days later than undamaged plants (Juenger and Bergelson 2000, Freeman et al. 2003, Sharaf and Price 2004).

Ehrlén (2003) has emphasized that final fitness effects of damage depend both on how strongly damage affects different components of fitness, and on how strongly each fitness component affects an integrated measure such as λ. It follows that a complete understanding of the impacts of herbivory and other forms of damage will require an integrated fitness accounting of damage throughout the life cycle. The effort necessary for this accounting in long-lived species such as *I. aggregata* should be amply repaid by the improved insight it yields into ecological and evolutionary facets of plant tolerance to damage.

Acknowledgements – Diane Campbell joined us in the larger demographic study from which this work is derived, and provided encouragement and advice throughout. Essential help in the field came from Heidi Albright, Regula Bollier, Rachel Freeman, Dawn Graydon, Ben Koch, Ursula Lang, Isaac Leader, Twila Paterson, Brook Peterson, Holly Prendeville, Kim Rand, Alta Price, Andrea Snow, Anne Valdenaire, Joe Vineis and Elliott Wilkinson. Denise Massart contributed the thoughtful assistance needed to get the study started, and billy barr provided data on winter precipitation at the RMBL. We were supported by the U. S. National Science Foundation (DEB 9805034 and DEB 9806501) and the Univ. of California, Riverside Academic Senate.

References

Aarssen, L. W. 1995. Hypotheses for the evolution of apical dominance in plants: implications for the interpretation of overcompensation. – Oikos 74: 149–156.

Becklin, K. M. and Kirkpatrick, H. E. 2006. Compensation through rosette formation: the response of scarlet gilia (*Ipomopsis aggregata*: Polemoniaceae) to mammalian herbivory. – Can. J. Bot. 84: 1298–1303.

Bergelson, J. and Crawley, M. J. 1992a. The effects of grazers on the performance of individuals and populations of scarlet gilia, *Ipomopsis aggregata*. – Oecologia 90: 435–444.

Bergelson, J. and Crawley, M. J. 1992b. Herbivory and *Ipomopsis aggregata*: the disadvantages of being eaten. – Am. Nat. 139: 870–882.

Bergelson, J. et al. 1996. Regrowth following herbivory in *Ipomopsis aggregata*: compensation but not overcompensation. – Am. Nat. 148: 744–755.

Boege, K. 2005. Influence of plant ontogeny on compensation to leaf damage. – Am. J. Bot. 92: 1632–1640.

Boege, K. and Marquis, R. J. 2005. Facing herbivory as you grow up: the ontogeny of resistance in plants. – Trends Ecol. Evol. 20: 441–448.

Campbell, D. R. 1998. Variation in lifetime male fitness of *Ipomopsis aggregata*: tests of sex allocation theory. – Am. Nat. 152: 338–353.

Campbell, D. R. 1989. Inflorescence size: test of the male function hypothesis. – Am. J. Bot. 76: 730–738.

Campbell, D. R. and Waser, N. M. 2007. Evolutionary dynamics of an *Ipomopsis* hybrid zone: confronting models with lifetime fitness data. – Am. Nat. 169: 298–310.

Caswell, H. 2001. Matrix population models. – Sinauer Associates, Inc.

Del-Val, E. K. and Crawley, M. J. 2005. Are grazing increaser species better tolerators than decreasers? An experimental assessment of defoliation tolerance in eight British grassland species. – J. Ecol. 93: 1005–1016.

Ehrlén, J. 2003. Fitness components versus total demographic effects evaluating herbivore impacts on a perennial herb. – Am. Nat. 162: 798–810.

Farnsworth, E. 2004. Hormones and shifting ecology throughout plant development. – Ecology 85: 5–15.

Freeman, R. S. et al. 2003. Flowering phenology and compensation for herbivory in *Ipomopsis aggregata*. – Oecologia 136: 394–401.

Gómez, J. M. 2003. Herbivory reduces the strength of pollinator-mediated selection in the Mediterranean herb *Erysimum mediohispanicum*: consequences for plant specialization. – Am. Nat. 162: 242–256.

Gronemeyer, P. A. et al. 1997. The effects of herbivory on paternal fitness in scarlet gilia: Better moms also make better pops. – Am. Nat. 150: 592–602.

Hogendyk, C. G. and Estabrook, G. F. 1984. The consequences of earlier reproduction in declining populations. – Math. Biosci. 71: 217–235.

Huhta, A. P. et al. 2000. Tolerance of *Gentianella campestris* in relation to damage intensity: an interplay between apical dominance and herbivory. – Evol. Ecol. 14: 373–392.

Inouye, B. D. and Tiffin, P. Measuring tolerance to herbivory with natural or imposed damage: a reply to Lehtila. – Evolution. 57: 681–682.

Juenger, T. and Bergelson, J. 1997. Pollen and resource limitation of compensation to herbivory in scarlet gilia, *Ipomopsis aggregata*. – Ecology 78: 1684–1695.

Juenger, T. and Bergelson, J. 1998. Pairwise versus diffuse natural selection and the multiple herbivores of scarlet gilia, *Ipomopsis aggregata*. – Evolution 52: 1583–1592.

Juenger, T. and Bergelson, J. 2000. The evolution of compensation to herbivory in scarlet gilia, *Ipomopsis aggregata*: herbivore-imposed natural selection and the quantitative genetics of tolerance. – Evolution 54: 764–777.

Juenger, T. et al. 2005. Scarlet gilia resistance to insect herbivory: the effects of early season browsing, plant apparency, and phytochemistry on patterns of seed fly attack. – Evol. Ecol. 19: 79–101.

Karban, R. and Thaler, J. S. 1999. Plant phase change and resistance to herbivory. – Ecology 80: 510–517.

Lenski, R. E. and Service, P. M. 1982. The statistical analysis of population growth rates calculated from schedules of survivorship and fecundity. – Ecology 63: 655–662.

Louda, S. M. and Potvin, M. A. 1995. Effect of inflorescence-feeding insects on the demography and lifetime fitness of a native plant. – Ecology 76: 229–245.

Loney, P. E. et al. 2006. How does ontogeny in a *Eucalyptus* species affect patterns of herbivory by brushtail possums? – Funct. Ecol. 20: 982–988.

Maschinski, J. and Whitham, T. G. 1989. The continuum of plant responses to herbivory: the influence of plant association, nutrient availability, and timing. – Am. Nat. 134: 1–19.

McGraw, J. B. and Caswell, H. 1996. Estimation of individual fitness from life-history data. – Am. Nat. 147: 47–64.

Mothershead, K. M. and Marquis, R. J. 2000. Indirect effects of leaf herbivores on plant-pollinator interactions in *Oenothera macrocarpa* (Onagraceae). – Ecology 81: 30–40.

Paige, K. N. 1999. Regrowth following ungulate herbivory in *Ipomopsis aggregata*: geographic evidence for overcompensation. – Oecologia 18: 316–323.

Paige, K. N. and Whitham, T. G. 1987. Overcompensation in response to mammalian herbivory: the advantage of being eaten. – Am. Nat. 129: 407–416.
Paige, K. N. et al. 2001. Overcompensation through the paternal component of fitness in *Ipomopsis arizonica*. – Oecologia 128: 72–76.
Piippo, S. et al. 2005. Resource availability at the rosette stage and apical dominance in the strictly biennial *Erysimum strictum* (Brassicaceae). – Can. J. Bot. 83: 405–412.
Price, M. V. et al. 2005. Temporal and spatial variation in pollination of a montane herb: a seven–year study. – Ecology 86: 2106–2116.
Shiojiri, K. and Karban, R. 2006. Plant age, communication, and resistance to herbivores: young sagebrush plants are better emitters and receivers. – Oecologia 149: 214–220.
Sage, T. L. et al. 2006. Self-sterility in *Ipomopsis aggregata* (Polemoniaceae) is due to prezygotic ovule degeneration. – Am. J. Bot. 93: 254–262.
Sharaf, K. E. and Price, M.V. 2004. Does pollination limit tolerance to browsing in *Ipomopsis aggregata*? – Oecologia 138: 396–404.
Stamp, N. 2003. Out of the quagmire of plant defense hypotheses. – Q. Rev. Biol. 78: 23–55.
Stanforth, L. M. et al. 1997. Insect herbivory on juveniles of a threatened plant, *Cirsium pitcheri*, in relation to plant size, density and distribution. – ÉcoScience 4: 57–66.
Stinchcombe, J. R. 2002a. Can tolerance traits impose selection on herbivores? – Evol. Ecol. 15: 595–602.
Stinchcombe, J. R. 2002b. Fitness consequences of cotyledon and mature-leaf damage in the ivy leaf morning glory. – Oecologia 131: 220–226.
Strauss, S. Y. and Agrawal, A. A. 1999. The ecology and evolution of plant tolerance to herbivory. – Trends Ecol. Evol. 14: 179–185.
Tiffin, P. 2000. Mechanisms of tolerance to herbivore damage: what do we know? – Evol. Ecol. 14: 523–536.
Tiffin, P. and Inouye, B. D. 2000. Measuring tolerance to herbivory: accuracy and precision of estimates made using natural versus imposed damage. – Evolution 54: 1024–1029.
Warner, P. J. and Cushman, J. H. 2002. Influence of herbivores on a perennial plant: variation with life history stage and herbivore species. – Oecologia 132: 77–85.
Waser, N. M. and Price, M. V. 1989. Optimal outcrossing in *Ipomopsis aggregata*: seed set and offspring fitness. – Evolution 43: 1097–1109.
Whitham, T. G. et al. 1991. Plant responses to herbivory: the continuum from negative to positive and underlying physiological mechanisms. – In: Price, P. W. et al. (eds), Plant–animal interactions: evolutionary ecology in tropical and temperate regions. John Wiley and Sons.