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Postdispersal Seed Predation Limits the Abundance of a Long-Lived Perennial Forb (Lithospermum ruderale)

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Postdispersal seed predation limits the abundance of a long-lived perennial forb (*Lithospermum ruderale*)

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Abstract. Loss of seeds to consumers is common in plant communities, but the degree to which these losses influence plant abundance or population growth is often unclear. This is particularly the case for postdispersal seed predation by rodents, as most studies of rodent seed predation have focused on the sources of spatiotemporal variation in seed loss but not quantified the population consequences of this loss. In previous work we showed that seed predation by deer mice (*Peromyscus maniculatus*) substantially reduced seedling recruitment and establishment of *Lithospermum ruderale* (Boraginaceae), a long-lived perennial forb. To shed light on how rodent seed predation and the near-term effects on plant recruitment might influence longer-term patterns of *L. ruderale* population growth, we combined experimental results with demographic data in stage-based population models. Model outputs revealed that rodent seed predation had a significant impact on *L. ruderale* population growth rate (λ). With the removal of postdispersal seed predation, the projected population growth rates increased between 0.06 and 0.12, depending on site (mean $\Delta\lambda$ across sites = 0.08). Seed predation shifted the projected stable stage distribution of populations from one with a high proportion of young plants to one in which larger adult size classes dominate. Elasticities of vital rates also changed, with germination and growth of seedlings and young plants becoming more important with the removal of seed predation. Simulations varying the magnitude of seed predation pressure while holding other vital rates constant showed that seed predation could lower λ even if only 40% of available seeds were consumed. These results demonstrate that rodent granivory can be a potent force limiting the abundance of a long-lived perennial forb.

Key words: granivory; Lithospermum ruderale; *matrix model;* Peromyscus maniculatus; *population growth rate; population projection; seed predation; small mammal.*

Introduction

As major constituents of ecological communities, plant consumers have the potential to substantially affect plant populations. The circumstances under which that potential is realized, however, remain unclear. We know from many individual-level studies on plants that consumers commonly depress plant size and fecundity, thus reducing the number of available seeds in plant populations (reviewed in Crawley 1989, 1997, Louda et al. 1990, Huntly 1991, Marquis 1992). However, our understanding of how these reductions in the performance of individual plants influence long-term patterns of population growth and plant abundance remains much more limited (reviewed in Gange 1990, Huntly 1991, Louda and Potvin 1995, Crawley 2000, Strauss et al. 2002, Halpern and Underwood 2006, Maron and Crone 2006).

Experimental studies quantifying how consumer-driven reductions in plant performance translate to changes in plant abundance are increasing but are still few in number (reviewed by Maron and Crone 2006). Most studies of consumer impacts on plant abundance have focused on short-lived plants with limited seed dormancy (Louda 1982*a*, *b*, Louda and Potvin 1995, Lennartsson et al. 1998, McEvoy and Coombs 1999, Maron et al. 2002, Rose et al. 2005, Shea et al. 2005). In contrast, the effects of consumers on populations of long-lived perennial plants or plants with seed banks have received far less study despite representing common life-history types in ecosystems worldwide (Maron and Crone 2006 but see Froborg and Eriksson 2003, Knight 2004, Kauffman and Maron 2006, Miller et al. 2009). Plants with these life histories are often assumed to be buffered at the population level from negative effects of consumers because (1) the many reproductive events of long-lived perennials might reduce the importance of seed loss in any one season and (2) seed banks could "store" reproduction, reducing plant vulnerability to current seed loss. Empirical tests of these assumptions, however, remain rare (Crawley 1997, 2000).

In addition, most work that has examined the population-level consequences of herbivory has focused on insect or larger ungulate herbivores (Maron and Crone 2006). Fewer studies have examined how post-

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dispersal seed predators, such as granivorous mice, influence plant abundance (but see Brown and Heske 1990, Kauffman and Maron 2006, Maron and Kauffman 2006). Because insect and ungulate herbivory can simultaneously influence several vital rates (growth, survival, reproduction) whereas postdispersal seed predation likely only influences one vital rate (seed survival), one might assume that postdispersal seed predators have more limited impacts on plant abundance than do other consumers. Moreover, even if postdispersal seed loss is relatively high, this need not lead to lower plant abundance if plant recruitment is more limited by safe sites for germination than by seed availability. That is, the number of microsites favorable to germination or seedling survival may have more influence on the number of seedlings establishing than does the number of available seeds (Harper 1977, Eriksson and Ehrlen 1992, Crawley 2000). In these cases, seed reduction from consumers will have less power to reduce plant abundance (Duggan 1985).

In a previous study we quantified how rodent seed predation by deer mice (*Peromyscus maniculatus*) affected the emergence of seedlings and the establishment of juvenile plants of a long-lived, large-seeded forb, *Lithospermum ruderale* (Boraginaceae). Through rodent exclusion and seed addition experiments, we found that cumulative seedling emergence was over 2.5 times higher in seed addition plots protected from rodent seed predators than in plots exposed to rodents (Bricker et al. 2010). These reductions in seedling emergence remained significant up to two years after seedlings emerged, with no evidence of compensatory density dependence during early life stages (Bricker et al. 2010).

Here, we examine the extent to which changes in recruitment due to rodent seed predation influence population growth rates of *L. ruderale*. This species has large seeds that make it vulnerable to seed predation and seed limitation, yet at the same time, has a long adult life span and seed dormancy (albeit limited) that may buffer it from negative population-level impacts of seed predation. To infer population-level consequences of postdispersal seed predation, we combined results of rodent exclusion and seed addition experiments with demographic monitoring and population modeling. Specifically, we quantified the ambient levels of seed predation and calculated the difference in population growth rate that would occur if rodent seed predation were eliminated. We then examined how seed predation alters population structure (stable stage distribution) and vital rate elasticities. We also simulated what level of seed predation would be required to significantly impact population growth rate in these populations.

Methods

Study system

Experiments and demographic monitoring took place at three study sites (Blackfoot, Bandy, and Kleinschmidt) dispersed over ~30 km of semiarid grasslands in the Blackfoot Valley of western Montana, USA. The plant community in these grasslands is dominated by native perennial bunchgrasses (*Festuca idahoensis* and *Festuca scabrella*) and scattered sagebrush (*Artemisia tridentata*) and includes a high diversity of native forbs. Exotic species are present at these sites but generally occur at very low densities.

The focal plant species, *Lithospermum ruderale*, is a long-lived native perennial forb that is a common but not dominant member of the grassland plant community. It begins aboveground growth in late April to early May and flowers between May and early July. Plants reproduce only by sexual reproduction, producing relatively heavy seeds (seed mass, 0.0211 ± 0.005098 g, mean $\pm$ SD), which fall from the plants as they mature in August and September. Seeds have a thick seed coat, lack eliasomes, and are not dispersed by ants.

Deer mice (*Peromyscus maniculatus*) are the main postdispersal seed predators at our sites. Several other small-mammal species are present (including montane voles, *Microtus montanus*; northern pocket gophers, *Thomomys talpoides*; Columbian ground squirrels, *Spermophilis columbianus*; and (rarely) yellow-pine chipmunks, *Tamias amoenus*; shrews, *Sorex* sp.; and hares, *Sylvagus nutallii*), but these animals are primarily herbivorous, florivorous, insectivorous, or inactive when seeds are dispersed at the end of the summer. We have observed no visible damage from insect herbivory on *L. ruderale*. The leaves of the plant are covered in stiff hairs, which may deter herbivory, and we have observed no noticeable pre- or postdispersal seed damage by insects (i.e., no larvae observed in opened seeds, and in seed offering experiments, no seeds appear to be removed or damaged by insects).

Seed predation and dormancy experiments

In order to estimate rates of seed predation and seedling germination, we carried out seed addition experiments using small-mammal exclosures built in the fall of 2002. At each site, one 10×10 m control plot was paired with a nearby 10×10 m small-mammal exclosure, within which we established a series of seed addition plots. A detailed description of the small-mammal exclosures is given in Bricker et al. (2010). In 2004 and 2005, we added locally collected seeds to 0.25×0.25 m subplots in each small-mammal exclosure and control plot pair, making five seed addition subplots in each site and treatment combination. We had no replication of particular seed densities or rodent exclosure treatments within our sites because our primary interest was to understand impacts of postdispersal seed predation across a wide spatial distribution of sites, rather than to compare differences between the sites. In 2004 we added seeds at densities of 50, 100, 200, and 300 seeds to new 0.25×0.25 m subplots in and out of rodent exclosures at each site. In 2005 seeds were added at just one density; each site had one pair of seed addition subplots (one in exclosure and one in control)

with 100 added seeds. At each site, we followed and recorded the emergence and survival of seedlings and young plants from both seed cohorts during the spring and summer of 2005, 2006, and 2007 (see Bricker et al. 2010 for details). These data were used to calculate rates of seedling emergence and the impacts of seed predation on recruitment.

To examine rates of seed survival in the soil seed bank, we buried bags containing locally collected *L. ruderale* seeds at each site in the late summer of 2004 and 2005. Bags were 5 × 5 cm, made of 3-mm fiberglass mesh, and buried 1–2 cm deep in the soil. Each bag contained 25 seeds. In August of 2005, 2006, and 2007, we excavated seeds buried the previous year and counted the number of original seeds that remained intact. In 2005 and 2006, half of the seeds left intact were buried again to estimate survival to two years. Seeds missing or visibly damaged were considered removed from the seed bank through either germination or decay. These data, together with the germination data from seed addition and rodent exclusion experiments, were used to estimate parameters related to seed survival in the soil seed bank.

Demographic monitoring

In the spring of 2005, we established permanently marked 0.5 m wide belt transects through naturally occurring *Lithospermum ruderale* populations at each site, extending the transects until they included at least 120 adult (non-seedling) individuals (total transect lengths = 75–100 m at each site). We marked and measured the size of all *L. ruderale* plants, including seedlings, on each transect. We monitored marked plants from spring 2005 through summer of 2007, which yielded three years of demographic data and two transitions. Size measures were taken in May (spring census) and August (summer census). During the spring census, we measured plant size, recorded whether each individual flowered or not, and marked any new seedlings. We recorded mortality of any marked plants in both spring and summer censuses, but only counted plants as dead if they did not appear in the next spring census. At the summer census we estimated fecundity by counting the number of seeds on each plant.

We estimate the size of plants by measuring the diameter of the plant at its widest point and the diameter perpendicular to that. We multiplied these two measures together to generate an index of canopy area. Canopy area provided the most explanatory power in logistic regression models to predict survival and flowering for adult plants based on size, site, year, and site × size interaction (for survival, $R^2 = 0.1370$, $P < 0.0001$; for flowering, $R^2 = 0.5385$, $P < 0.001$).

Selecting size metric and size class boundaries

Plants were divided into stages based on size and life stage. Seeds and seedlings represent distinct, time-bounded biological states, and are therefore treated as stage classes (one- and two-year-old seeds, and seedlings). Plants that are older than seedlings (one year or more) were divided into size classes based on canopy area. We used logistic regression to determine size class boundaries based on the relationship between plant area, survival, and probability of flowering (Morris and Doak 2002). We also used visual inspection of the graphical data to examine the minimum size for seed production and rates of fecundity vs. size. Based on these patterns we divided adult (non-seedling) plants into three size classes based on their canopy area: small ($\leq$12 cm^2), medium ($>$12 cm^2 to $\leq$50 cm^2), and large ($>$50 cm^2).

Parameter estimation

We used the vital rates from demographic monitoring and our rodent exclusion experiments to construct stage-based matrix models of the general form of $\mathbf{N}_{t+1} = \mathbf{A}\mathbf{N}_t$, where $\mathbf{N}$ is a vector of the number of individuals in each size class (subscripted to denote an annual time step). The transition matrix $\mathbf{A}$ is made up of matrix elements (a_{ij}) representing the stage-specific transition rates calculated from vital rates (germination, survival, growth, fecundity, and seed predation (Table 1). Fig. 1 shows a life cycle diagram illustrating the stages and transitions comprising the matrix model. Table 2 shows the matrix structure built from those vital rates.

We estimated survival probability for the three adult size classes using the two-step process outlined in Morris and Doak (2002). That is, we first ran a logistic regression of survival on canopy area (ln-transformed) using the whole data set. When there were significant differences between sites (based on Type III SS from logistic regression in PROC LOGISTIC; SAS Institute 2004), we generated separate regression equations for each site. We then used the fitted regression equation to calculate survival for each class based on the median size of individuals in that class at each site. We calculated seedling survival with counts from demographic monitoring data at each site, combining the three years of data to generate the estimate.

We used the same procedure to estimate the probability of flowering, creating a logistic regression relating canopy size and flowering. We used this fitted logistic regression equation to generate a probability of flowering for the medium and large size classes based on the median size of individuals in that class. Individuals in the small adult class were never observed to flower and therefore have no probability of flowering in the models. Fecundity was calculated from the average number of seeds produced by a flowering plant in each size class. The probability of surviving plants transitioning between classes was calculated directly from field counts, as the proportion of surviving individuals in each size class transitioning to each of the other size classes (or staying in the same class).

We estimated seedling emergence rates and rates of dormancy in the soil seed bank using data from seed addition and buried seed bag experiments. In these

TABLE 1. Mean vital rates for each site in semiarid grasslands in the Blackfoot Valley of western Montana, USA.

Vital rate symbol	Vital rate description	Blackfoot	Bandy	Kleinschmidt
Pg1	probability of a seed germinating in first year	0.0078	0.0080	0.00980
Pg2	probability of seed germinating in second year (applies only to seed 1 class)	0.4284	0.2955	0.1907
Pd1	probability of seed dying in seed bank in first year	0.6678	0.3524	0.3954
Pd2	probability of seed dying in seed bank in second year	0.0118	0.2516	0.4880
Pm1	probability of seed being eaten by mice in year 1	0.6000	0.99999	0.8928
Pm2	probability of seed being eaten by mice in year 2	0	0	0
Surv_Sdlg	seedling survival	0.6634	0.2564	0.5915
Surv_SmAd	small adult survival	0.8300	0.7775	0.7765
Surv_MedAd	medium adult survival	0.9475	0.9455	0.9395
Surv_LgAd	large adult survival	0.9912	0.9855	0.9878
SmAd_SmAd	small adult stasis	0.7899	0.75	0.8254
SmAd_MedAd	transition: small adult to medium adult	0.2101	0.25	0.1746
SmAd_LgAd	transition: small adult to large adult	0	0	0
MedAd_SmAd	transition: medium adult to small adult	0.05405	0.05172	0.07792
MedAd_MedAd	medium adult stasis	0.7162	0.4655	0.7013
MedAd_LgAd	transition: medium adult to large adult	0.2297	0.4828	0.22078
LgAd_SmAd	transition: large adult to small adult	0	0.008264	0.007576
LgAd_MedAd	transition: large adult to medium adult	0.01941	0.02479	0.0530
LgAd_LgAd	large adult stasis	0.9806	0.9669	0.9394
Flprob_MedAd	medium adult flowering probability	0.02424	0.07843	0.06479
Flprob_LgAd	large adult flowering probability	0.9272	0.78471	0.8434
Fec_MedAd	medium adult fecundity (of flowering ind'ls)	1.000	2.375	0.25
Fec_LgAd	large adult fecundity (of flowering idl's)	12.52	12.68	8.310

experiments, many seeds germinated in the second spring after they were produced, but few germinated in the first spring. This meant that rates of seedling emergence and seed predation could not be measured directly, because some unknown number of seeds initially added to the plots could die in the seed bank during the first year, making the size of the seed pool available to germinate in the second spring unknown. To circumvent this problem, we estimated seedling emergence using a mechanistic model of seedling emergence and survival. In cases where some model parameters cannot be estimated empirically, maximum-likelihood modeling can provide a way to parameterize these vital rates based on the available empirical data (Kauffman and Maron 2006). We generated maximum-likelihood estimates of: (1) the probability of a seedling emerging in year 1 (i.e., the spring immediately following the summer in which it was produced) (Pg1), (2) the probability of a seedling emerging in year 2 (Pg2), (3) the probability of a seed dying in year 1 (Pd1), (4) the probability of a seed dying in year 2 (Pd2), (5) the probability of a seed being eaten by rodents in year 1 (Pm1), and (6) the probability of a seed being eaten by rodents in year 2 (Pm2) (Table 3). The seed stages were age-based, and seeds were forced to progress through the age structure at each time step and did not live past three years old, which was consistent with the survival of seeds in the buried seed bag experiments. We did not have enough data to separately estimate a probability of germinating in three-year-old seeds; thus the values for Pg1 (seeds germinating after one year of dormancy or two years after they were produced) were the same as Pg2 (seeds germinating after two years of dormancy or three years after they were produced).

Maximum-likelihood estimates for the seed parameters were based on the observations (experimental outcomes) listed in Table 3. Within each set of data from seed burial and seed addition experiments at a given site and year, we constructed a joint probability function based on a binomial distribution for the

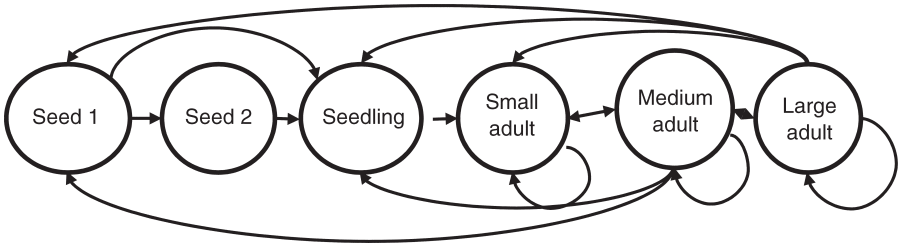

FIG. 1. Life cycle diagram for *Lithospermum ruderale*, from semiarid grasslands in the Blackfoot Valley of western Montana, USA, showing stages and transitions used in the matrix models. Arrows indicate possible transitions of individuals between stages and the reproductive contributions of plants in a given stage. Arrows from adult plants to seeds or seedlings represent the contribution of new seedlings to the population through seed production of larger plants.

TABLE 2. Matrix structure for matrix including and without seed predation.

	Sd1	Sd2	Seedling	Small adult
Including seed predation				
Sd1	0	0	0	0
Sd2	(1 − Pg2 − Pd2) × (1 − Pm2)	0	0	0
Seedling	Pg2 × (1 − Pm2)	Pg2 × (1 − Pm2)	0	0
Small adult	0	0	Surv_Sdlg	Surv_SmAd × SmAd_SmAd
Medium adult	0	0	0	Surv_SmAd × SmAd_MedAd
Large adult	0	0	0	0
Without seed predation				
Sd1	0	0	0	0
Sd2	0(1 − Pg2 − Pd2)	0		0
Seedling	Pg2	Pg2		0
Small adult	0	0	Surv_Sdlg	Surv_SmAd × SmAd_SmAd
Medium adult	0	0	0	Surv_SmAd × SmAd_MedAd
Large adult	0	0	0	0

Note: Sd1 refers to seeds produced in time t that remain dormant in the seed bank in year $t + 1$; Sd2 refers to seeds that were produced in time t but remain dormant in the seed bank in year $t + 2$.

probability of each of the observed events. The general form of the binomial probability function is

$$\text{probability}(p)\text{of } k \text{ events in } N \text{ trials} = p^k(1-p)^{N-k}\frac{N!}{k!(N-k)!}.$$

Within a data set the final factorial term is a constant, so we dropped that term from the probability functions and wrote the probability of the observed number of events (k) out of (N) number of trials (e.g., $k =$ number of seeds that survive, out of N seeds buried in seed bags), as

$$p^k(1-p)^{N-k}.$$

This generated six joint likelihood functions within each data set (one for each observation). We used the solver function in Microsoft Excel (version 2003; Redmond, Washington, USA) to maximize the sum of the natural log of these likelihood functions by changing the values of the six estimated parameters. The likelihood functions for each of the observations are given in Table 3. The maximum-likelihood solutions created a set of estimated parameters that were most likely, given the data, for each set of observations, and thus avoided the problem of impossible combinations of rates that could occur if trying to calculate the parameters directly from each of the separate experiments. At each site we used data from four pairs of seed addition plots (the four different seed densities, inside and outside of rodent exclosures) from 2004, and the data from the 100-seed addition plots in 2005. We used likelihood ratio tests to test the significance of site and year by comparing models including site or year to those that did not, for each parameter.

Our ability to parameterize the seed predation rates in years one and two separately was very low. Models that assumed that all seed predation occurred in year one (second year seed predation set to zero) performed significantly better in likelihood ratio tests ($\chi^2 = 41.87$, $P < 0.001$). This is consistent with field observations that most seeds are consumed in the first fall and winter after they are released from the plant. In our models, therefore, all seed predation occurs in the first year.

Model structure and simulations

We created separate matrices for each site. Models projecting population growth with rodent seed predation ("with mice") included the probability of seeds being eaten and the effects of this on seedling emergence (Pm1, Pm2); those projecting population growth in the absence of seed predation ("no mice") did not (Table 2). With only two transitions, our ability to incorporate temporal variation into matrices was extremely limited. Within each site, we calculated a mean for each vital rate by combining the observations across multiple years. We used these matrices to calculate the elasticity of vital rates and the stable age distribution, with and without mice, at each site (Morris and Doak 2002). Table 4 shows the parameterized matrices.

To test the robustness of the model results to parameter uncertainty and examine the significance of the change in λ due to removing rodent seed predation, we calculated growth rates as a bootstrap analysis based on resampling from the original data sets. For each vital rate, we resampled with replacement from the original data, and calculated that vital rate at each bootstrap iteration from the resampled data (McPeek and Kalisz 1993). For vital rates relating to seedling and adult survival, flowering, fecundity, and transitions between stages, the raw data for the bootstrapping came from the measurements of individual plants on demographic transects at each site. For seed-related vital rates (germination, survival in seed bank, and probability of being eaten by rodents), the data array for the bootstrapping was made up of the five maximum-likelihood estimates for that site at each iteration. We used these bootstrapped vital rates to calculate population growth

TABLE 2. Extended.

Medium adult	Large adult
FlrProb_MedAd × Fec_MedAd × (1 − Pg1 − Pd1) × (1 − Pm1)	FlrProb_LgAd × Fec_LgAd × (1 − Pg1 − Pd1) × (1 − Pm1)
0	0
FlrProb_MedAd × Fec_MedAd × Pg1 × (1 − Pm1)	FlrProb_LgAd × Fec_LgAd × Pg1 × (1 − Pm1)
Surv_MedAd × MedAd_SmAd	Surv_LgAd × LgAd_SmAd
Surv_MedAd × MedAd_MedAd	Surv_LgAd × LgAd_MedAd
Surv_MedAd × MedAd_LgAd	Surv_LgAd × LgAd_LgAd
FlrProb_MedAd × Fec_MedAd × (1 − Pg1 − Pd1)	FlrProb_LgAd × Fec_LgAd × (1 − Pg1 − Pd1)
0	0
FlrProb_MedAd × Fec_MedAd × Pg1	FlrProb_LgAd × Fec_LgAd × Pg1
Surv_MedAd × MedAd_SmAd	Surv_LgAd × LgAd_SmAd
Surv_MedAd × MedAd_MedAd	Surv_LgAd × LgAd_MedAd
Surv_MedAd × MedAd_LgAd	Surv_LgAd × LgAd_LgAd

rate (λ) in the presence of small mammals. We calculated the mean of these 1000 iterations, and 95% confidence intervals of λ by ordering the 1000 estimates of λ and selecting the 25th and the 975th values as the lower and upper confidence limits, respectively.

This estimate of λ and its confidence intervals includes variance from all of the vital rates simultaneously. To estimate the difference (and confidence limits around that difference) in growth rate due specifically to small-mammal seed predation ($\Delta\lambda$), we created a bootstrapping routine where at each iteration, two matrices were built from the resampled vital rates. One matrix included seed predation while the other did not. For the two matrices all vital rates were the same, except that one included seed predation, while the other did not. At each iteration we calculated λ of both matrices and generated a metric of the change in growth rate ($\Delta\lambda$) as

$$\Delta\lambda = \lambda_{\text{no-mouse}} - \lambda_{\text{mouse}}$$

to reflect the increase in growth rate that would occur

TABLE 3. Observations and parameters used in maximum-likelihood functions to estimate seed-related vital rates and likelihood functions for maximum-likelihood estimates of seed-related vital rates.

Observations, parameters, and likelihood functions	Symbol
Observations	
Number of seeds added to seed addition plots	Ns
Number of seedlings in year 1 in rodent-excluded plots	Ks1
Number of seedlings in year 2 in rodent-excluded plots	Ks2
Number of seedlings in year 1 in rodent-accessible (control) plots	Kr1
Number of seedlings in year 2 in rodent-accessible (control) plots	Kr2
Number of seeds in bags, at start	Nb
Number of seeds in bags still alive in year 1	Kb1
Number of seeds from bags still alive in year 2	Kb2
Estimated parameters	
Probability of germinating in year 1	Pg1
Probability of germinating in year 2	Pg2
Probability of dying in year 1	Pd1
Probability of dying in year 2	Pd2
Probability of being eaten by mice in year 1	Pm1
Probability of being eaten by mice in year 2	Pm2
Likelihood functions	
Likelihood for Ks1	$Pg1^{Ks1} \times (1 - Pg1)^{(Ns-Ks1)}$
Likelihood for Kb1	$(1- Pg1 - Pd1)^{Kb1} \times (Pg1 + Pd1)^{(Nb-Kb1)}$
Likelihood for Ks2	$(Pg2^{Ks2}) \times (1 - Pg2)^{(Ns\times(1-Pd1-Pg1)-Ks2)}$
Likelihood for Kb2	$(1 - Pg2 - Pd2)^{Kb2} \times (Pg2 + Pd2)^{(Kb1-Kb2)}$
Likelihood for Kr1	$[(1 - Pm1) \times (Pg1)]^{Kr1} \times [1 - (1 - Pm1) \times (Pg1)]^{(Ns-Kr1)}$
Likelihood for Kr2	$[(1 - Pm2) \times (Pg2)]^{Kr2} \times [1 - (1 - Pm2) \times (Pg2)]^{(Ns\times(1-Pd1-Pg1)-Kr2)}$

TABLE 4. Parameterized matrices for each site built from vital rates presented in Table 1.

	Seed 1	Seed 2	Seedling	Small adult	Medium adult	Large adult
Blackfoot						
Seed 1	0	0	0	0	0.0031 (*0.0079*)	1.5070 (*3.7674*)
Seed 2	0.5598	0	0	0	0	0
Seedling	0.4284	0.4284	0	0	0.0001 (*0.0002*)	0.0362 (*0.0906*)
Small adult	0	0	0.6634	0.6556	0.0512	0
Medium adult	0	0	0	0.1744	0.6786	0.0192
Large adult	0	0	0	0	0.2177	0.9720
Bandy						
Seed 1	0	0	0	0	0.000000001 (*0.1191*)	0.0001 (*6.3643*)
Seed 2	0.4529	0	0	0	0	0
Seedling	0.2955	0.2955	0	0	0.0000000001 (*0.0015*)	.000000001 (*0.076*)
Small adult	0	0	0.2564	0.5832	0.0489	0.0081
Medium adult	0	0	0	0.1944	0.4402	0.0244
Large adult	0	0	0	0	0.4565	0.9530
Kleinschmidt						
sd1	0	0	0	0	0.0010 (*0.0096*)	0.4469 (*4.1693*)
sd2	0.3213	0	0	0	0	0
sdlg	0.1907	0.1907	0	0	0.00001 (*0.0002*)	0.0074 (*0.0687*)
smad	0	0	0.5915	0.6409	0.0732	0.0075
medad	0	0	0	0.1356	0.6589	0.0524
lgad	0	0	0	0	0.2074	0.9280

Note: For matrix elements that differ in the presence of seed predators, the value in the absence of seed predation is shown italicized and in parentheses.

with the exclusion of rodent seed predation. As with the λ estimates, we calculated a mean and 95% confidence intervals for $\Delta\lambda$ based on the 1000 bootstrap iterations.

In order to examine how populations might respond to varying levels of seed predation, we calculated $\Delta\lambda$ across a range of simulated predation intensities. These projections were done using the same bootstrapping methods described previously, with the exception of the mouse predation term (Pm1). In these simulations, we bootstrapped for the probability of seed predation using an array of 10 numbers, with the proportion of 1's (seed eaten) and 0's (not eaten) varying to reflect a mean probability of being eaten between 0.1 and 1. We calculated a mean and 95% confidence interval for the value of $\Delta\lambda$, at each simulated rate of seed predation, for each site.

RESULTS

We estimated the ambient levels of seed predation by deer mice, averaged across years, to be 60% at Blackfoot, 89% at Kleinschmidt, and 99% at Bandy (Table 1). *Lithospermum ruderale* populations exposed to this level of postdispersal seed predation are relatively stable at all three sites, as our population growth estimates suggest that populations are likely neither growing nor shrinking dramatically (Bandy $\lambda = 0.966$, 95% CI = 0.932–0.992; Kleinschmidt $\lambda = 0.980$, 95% CI = 0.950–1.01; Blackfoot $\lambda = 1.08$, 95% CI = 0.981–1.18). However, when population growth was simulated in the absence of postdispersal seed predation, population growth rate of *L. ruderale* increased significantly at all three sites ($\Delta\lambda$ for Blackfoot = 0.0702, Bandy = 0.1169, Kleinschmidt = 0.0555; Fig. 2). Although populations at the three sites differed in their projected responses to seed predation, the 95% confidence intervals around the estimate of the difference between populations with and without seed predation ($\Delta\lambda$) did not overlap zero for any of the sites, indicating a significant impact of rodent seed predators on population growth rates across the sites (Fig. 2).

As one might expect, we found that the influence of postdispersal seed predation on plant population growth ($\Delta\lambda$) increases as we simulate increasing levels of seed predation (Fig. 3). Interestingly, however, seed predation rates do not have to be extraordinarily high for population-level impacts to appear. For example, even if only 30–40% of available seeds are eaten, our simulations reveal that this is sufficient to reduce the growth

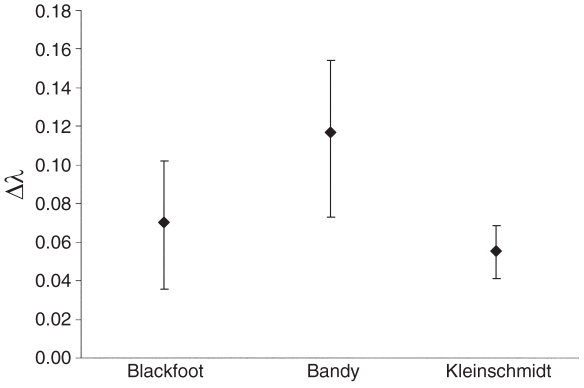

FIG. 2. Difference in population growth rate ($\Delta\lambda$) due to release from seed predation by small mammals (λ without mice $-$ λ with mice) at each of the three sites. Error bars show 95% confidence intervals generated by 1000 bootstrap iterations.

rate of *L. ruderale* populations (meaning the 95% confidence intervals for the $\Delta\lambda$ no longer overlap zero).

The projected stable stage distributions for the three sites shifted with the exclusion of seed predators (Fig. 4). In the absence of seed predation, the proportion of seeds and younger plants in the population became much higher than at natural levels of seed predation, when larger size classes dominated. Elasticity analysis showed that the vital rates with the largest elasticity values were the survival and stasis of individuals in the large-adult stage class (Fig. 5). The elasticity of these vital rates was lower in the presence of small-mammal seed predation, although they remained relatively high. With the exclusion of seed predators, the elasticity of reproductive vital rates (flowering probability and fecundity) increased, as did the importance of germination and seed survival (Fig. 5). The effects on both stable stage distribution and elasticities of vital rates were most dramatic at sites with the highest ambient levels of seed predation.

Discussion

Seed predation clearly has great potential to impact population growth in short-lived plants that have minimal seed banks, as recruitment for plants with this life history relies directly on seed production each year (Brown and Heske 1990, Crawley 2000). Our results, however, suggest that this potential is not restricted solely to plants where population growth is highly sensitive to current seed production. Using demographic data for *Lithospermum ruderale*, we found low elasticity values for seeds, as has been commonly found for other long-lived perennial plants (Silvertown et al. 1993, Franco and Silvertown 2004), suggesting that there is limited scope for consumer-driven changes in seed availability to alter plant abundance and population dynamics. Yet seed predation in our system was of sufficient magnitude to reduce population growth of *L. ruderale*. Furthermore, the elasticity of different vital rates shifted (sometimes dramatically) with the inclusion or exclusion of seed predation in simulations. In the absence of seed predation, vital rates related to reproduction, germination, and growth of small plants increased relative to the importance of survival and stasis of older, larger plants. The fact that this biotic interaction shifted the elasticity values in the population suggests that we should be cautious in concluding that species interactions affecting low-elasticity vital rates will not affect population dynamics. Rather, it may also be important to take into account the variation possible in those vital rates (Wisdom et al. 2000, Kauffman and Maron 2006).

Seed predators are generally assumed to have greater impacts when populations are limited more by seeds than by microsites (Harper 1977). Systems that have represented most of the work on plant population responses to seed predation have been those where we might expect seed availability to be more limiting than

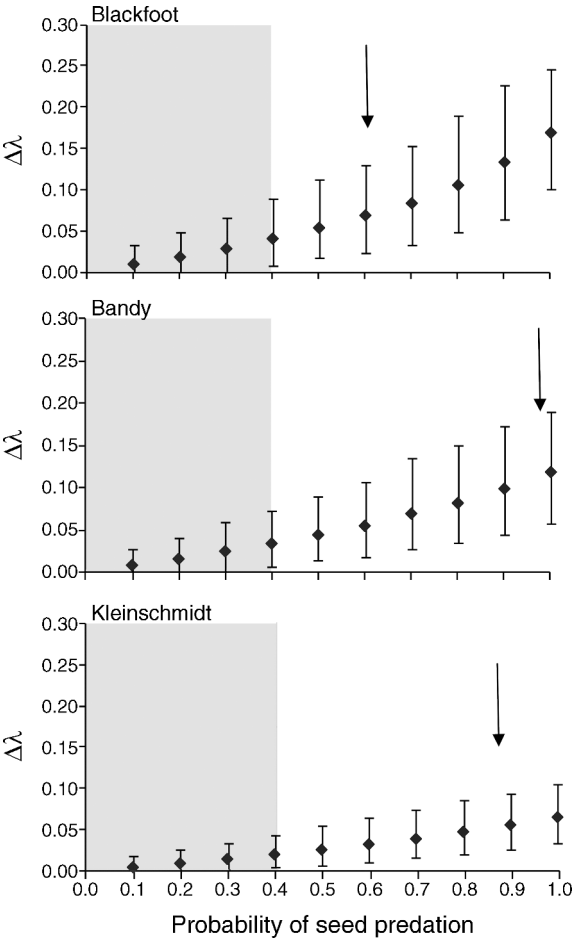

Fig. 3. The difference in population growth rate ($\Delta\lambda = \lambda$ without mice $-$ λ with mice) calculated across a range of simulated seed predation intensities. Arrows indicate the natural level of seed predation estimated for each site. The shaded area represents the range of tolerable seed predation, defined as the level at which the 95% confidence limits for $\Delta\lambda$ overlap zero, indicating no difference in population growth between projections with and without seed predation.

microsites—generally, desert and dune systems, and planted prairie restorations, where seeds were added to initially bare ground (Edwards and Crawley 1999, Howe and Brown 2000, Howe and Brown 2001, Howe and Lane 2004, Howe et al. 2006). Greater cover of vegetation has been shown to inhibit seedling germination (Eriksson and Ehrlen 1992, Reader 1993), leading to the expectation that communities with denser vegetation will be less seed limited, and more site limited. This work complements what has been done in these other systems, showing that even in a higher cover environment, species may exhibit population-level responses to postdispersal seed predation.

Historically, postdispersal seed predators have often been assumed to have little impact on plant abundance, due in part to the assumption that any population has some surplus of seeds beyond what is needed to

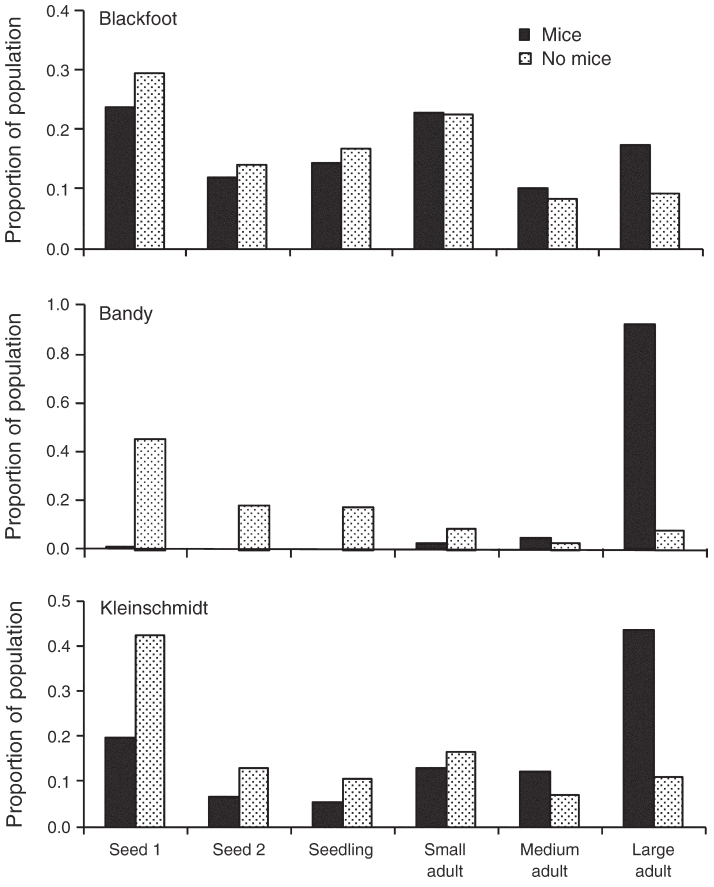

FIG. 4. Stable stage distributions projected from the matrix for each site, with and without rodent seed predation. The relative proportion of larger plants is dramatically lower when small-mammal seed predation is prevented.

maintain the population. Furthermore, it has often been assumed that seed removal rates need to be extremely high in order to affect population growth or plant density. Harper (1977) encapsulated this idea by suggesting that a population could sustain tolerable levels of seed predation, wherein a certain number of seeds that were "doomed to die" from various other causes (as seeds or at later stages), could be eaten by consumers without substantially altering the numbers of adults in the population. In the *L. ruderale* populations we studied, however, the ambient levels of postdispersal seed predation do not appear to fall within the range of "tolerable" seed predation. Moreover, based on our simulations, even reductions in seed availability of ~40% (well below what we estimated) would be sufficient to suppress population growth rates. This result is similar to what Louda and Potvin (1995) found working on predispersal seed predation by insects. More specifically, Louda and Potvin (1995) modified a version of Harper's original model, and showed that only a very minimal amount of seed predation (if any) might be tolerable and that this was well below the levels observed in their system. In fact, the levels of seed predation that do not cause some reduction in plant density or population growth may generally be very small. The level of seed predation required to lower population growth will likely vary by population, and depend heavily on the demographic rates of the population considered.

Our results also demonstrate the utility of incorporating experimental data on consumer impacts on a particular demographic transition in demographically based stage-structured population models. For many species, particularly in areas with short growing seasons or harsh conditions, a slower life-history pattern is common, typified by long-lived adult stages, variable fecundity, and slow growth. These common life-history features often make direct observation of changes in abundance due to experimental manipulation of consumer pressure unfeasible in an experimental time frame. Combining experiments, demography, and population models can be a valuable approach for estimating interaction strength (Maron et al. 2010).

While our approach allowed us to forecast the long-term population-level consequences of rodent seed predation on *L. ruderale*, there are several important caveats to bear in mind. First, we used deterministic population models despite the fact that some vital rates differed between years. Having only three years of demographic data (two transitions) limited our ability to incorporate temporal variation in the matrix projections. However, for models with fewer than five years of

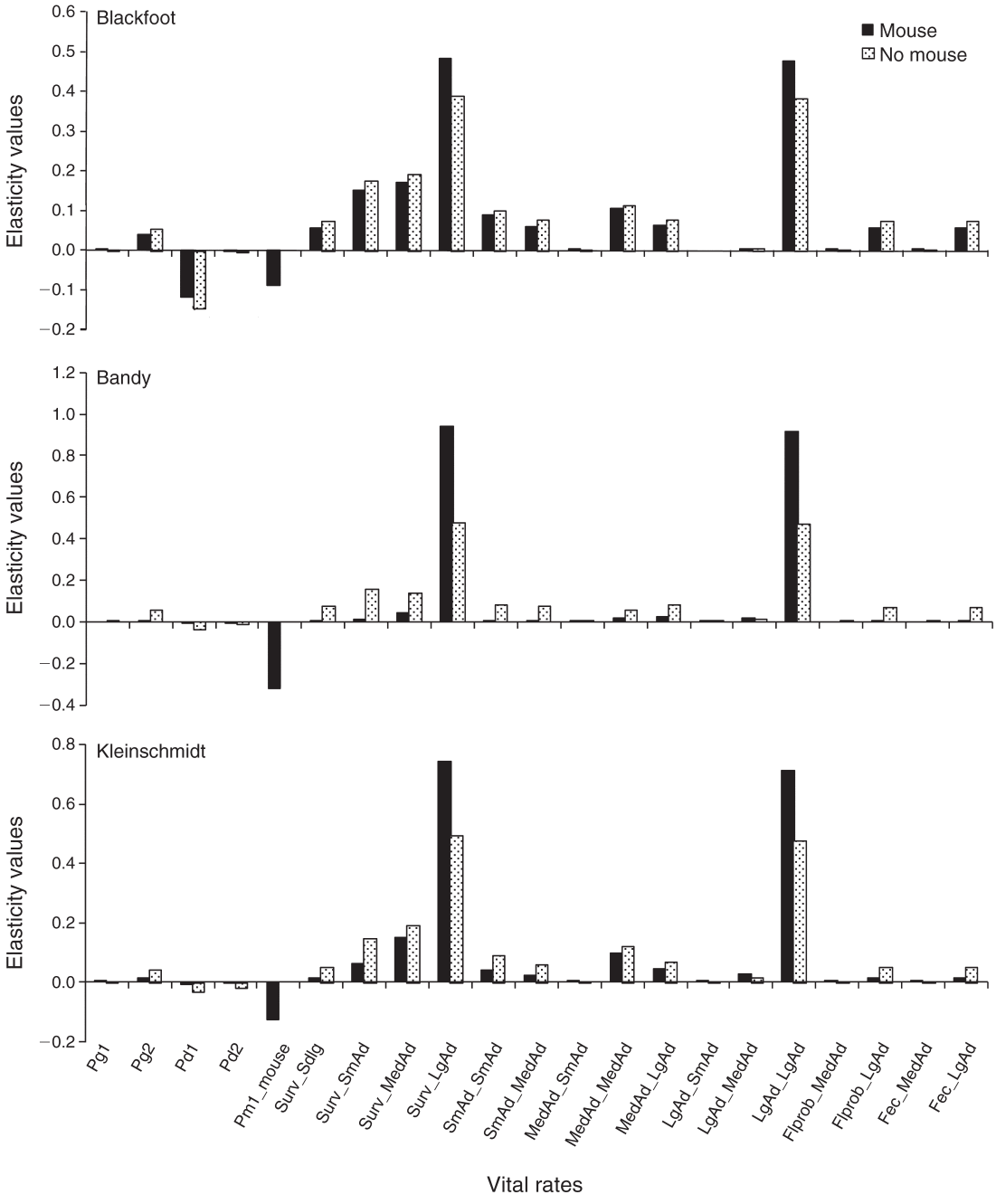

FIG. 5. Elasticities of the vital rates making up the matrix for each site. Vital rates are described in Table 1.

data, a deterministic model can be more accurate than a stochastic model based on limited data (Doak et al. 2005). Beyond these considerations, our primary goal was not to generate an extremely precise estimate for *L. ruderale* population growth rate, but to determine how postdispersal seed predation might change population growth. Furthermore, by bootstrapping from resampled data from both years for all vital rates, we incorporated temporal variation in vital rates into our estimates for how seed predation altered λ. Second, our population model does not incorporate density dependence. Clearly the density of individuals in a finite space will at some point begin to cause decreases in the performance of individual plants, which can feed back to depress population growth. In previous work we tested for negatively density-dependent seedling survival and growth in seed addition plots, and found no differences with density (Bricker et al. 2010). A central challenge for the future is to determine how best to incorporate density dependence into population models that esti-

mate consumer impacts on plant population growth/abundance (Halpern and Underwood 2006). In this system, however, while we recognize that density dependence could occur at other life stages, *L. ruderale* plants are fairly sparsely distributed. This means there would need to be a dramatic increase in plant abundance due to release from rodent seed predation, before density dependence began to cause declines in plant performance.

Our results demonstrate that seed predation by deer mice in this grassland community substantially affects the population structure and abundance of *L. ruderale*. Removing seed predation leads to shifts in the stage structure of the populations, changes in elasticity values, and higher projected population growth. Thus, ambient levels of seed predation in these grassland communities are sufficient to keep abundance of *L. ruderale* lower than it would be without rodent seed consumers. More broadly, our work suggests that for large-seeded species inhabiting grassland ecosystems, postdispersal seed predation may be an important but often overlooked interaction that can limit plant abundance. This will be particularly true in cases where compensatory mechanisms (such as strong density dependence at seedling or other life stages) are relatively weak. Our understanding of the general importance of seed loss to plant abundance could be greatly enhanced by increased focus on understanding the strength of these compensatory mechanisms (sensu Garren and Strauss 2009).

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