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Connecting Herbivore Effects to Population Dynamics of Common Milkweed (*Asclepias syriaca*) Using an Integral Projection Model

A thesis submitted in partial fulfillment of the requirement for the degree of Bachelor of Science in Biology from The College of William and Mary

by

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Accepted for **Honors**

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Abstract

Plant defense mechanisms and their effects on plant performance have been extensively studied in common milkweed (*Asclepias syriaca*). No studies, however, have investigated the relevance of these responses to its population dynamics. Common milkweed are clonal plants that exhibit both an induced defensive chemical response and mechanisms of resource allocation upon herbivore damage. Milkweed population decline has been implicated as a major contributing factor to the decline of monarch butterflies. We examined how herbivores affect the survival, growth, and fecundity of the ramets of common milkweed, and whether those effects were meaningful at the population level. By using an integral projection model to connect individual variation in size and foliar damage to population dynamics, we show that herbivores affect the milkweed’s sexually reproductive output and clonal propagation, both pathways that the population growth rate is sensitive to. Our results provide insight on the herbivore effects that have greater influence on population growth and indicate future directions to improve the currently limited knowledge of how environmental factors drive population dynamics in common milkweed.
Chapter 1

Introduction

Herbivores are known to affect an individual’s growth, reproduction, and survival and are speculated to influence population size and distribution [33]. Plant responses to herbivore damage may include investment in chemical defenses to deter herbivores or a re-allocation of resources away from the site of damage, or a combination of both [35, 25, 27, 2]. While many studies have investigated the variation in plant responses to herbivore damage [26, 8, 44, 27, 9], many fewer have directed attention towards how these plant-herbivore interactions have an effect at the level of the plant population [33].

The integral projection model (IPM) is a powerful tool that can connect variation in individual performance to population level metrics. It is a modification of the matrix population model, which project populations of individuals categorized by discrete stage or age classes, in that the IPM projects populations that are characterized by continuous traits. The basic assumption of this type of population model is that individuals in a continuously size structured population make different contributions to the future state of the population. The IPM is composed of functions that predict an individual’s vital rates, namely survival, growth, and fecundity, from its size or other defining characteristic that explains the observed variation in individual performance. By summing over the size dependent contributions of each individual, IPMs can translate individual level processes to that of the population [15, 12, 11].

While certain characters are strongly representative of an individual’s vital rates, the environment also plays a role in individual performance [28]. There is increasing emphasis on identifying the biotic and abiotic determinants of population dynamics in order to make more accurate predictions about how populations will be altered in rapidly changing environmental conditions [39, 16]. Most of the population models that include environmental covariates incorporate the effects of herbivory [16] but often vary in their results. In tree cholla cactus populations,
insect herbivores decreased an individual’s survival and fecundity and led to decreased population growth rates, though the magnitude of this effect decreased along an increasing elevation gradient [36]. In a monocarpic perennial herb, herbivory increased seed quality, but incorporation of this tolerance did not affect the population growth rate at the observed rates of seed germination, signifying tolerance, though not as a case of over compensation in which the population growth would increase [6]. Grazing on boreal shrubs (Vaccinium myrtillus) had multiplicative effects with resource levels such as soil quality and light yet overall decreased an individual’s growth. At above average levels of grazing, the population growth rate decreased below stability but the population remained increasing at the average low levels of damage, indicating relative tolerance to herbivory [21]. Multiplicative effects of grazing, fire, and harvest led to different results on the population growth rate for a date palm (Phoenix loureiroi). When more than half of the leaves in a genet were removed, survival rates greatly decreased. If a genet experienced light levels of herbivory, at less than 10% removal, growth and clonal reproduction increased, but those vital rates otherwise decreased at high levels. When considered in isolation, grazing and harvest each had negative consequences on the population growth rate, but their interaction counteracted this effect. In the presence of fire disturbance, however, the population growth rate became even lower than with the additive effects of grazing and harvest [32]. It is clear from these examples that individuals vary in their strategies of response upon tissue loss and they can depend on interactions with other environmental factors. Depending on the study system, herbivory leads to fitness consequences that may either decrease, increase, or have no effect on the population growth rate. Because there are no overarching patterns of observed herbivore effects on host population dynamics and few empirical studies that explore this question, further case-specific investigations are necessary.

Upon herbivore damage, common milkweed (Asclepias syriaca) have an induced defensive response in which they increase production of a sticky latex substance with cardenolide compounds around the site of damage [31]. The latex physically hinders the movement of an herbivore’s mouthparts [4] and cardenolides are toxins that disrupt ion transport [29]. These chemicals are harmful to most herbivores, yet some select specialists can withstand their adverse effects. Despite the inadequacy of these secondary metabolites to deter specialist herbivores, the use of the induction mechanism persists in common milkweed and reduces its fitness [5, 14, 50]. To offset these fitness losses, common milkweed are also known to exhibit mechanisms of tolerance by directing resources into the stem [45], which may be linked to observed regrowth after herbivore damage [2]. A milkweed’s decision between these two responses has been posed as a trade-off on the scale of
macroevolutionary trends among closely related milkweed species [23, 3, 38], but within species there is not necessarily a trade-off in resource allocation [2]. Increased plant growth rate does not imply reduction in milkweed resistance traits. Differential combinations of these strategies vary in their fitness consequences and are likely related to spatial variation in herbivore abundance and other environmental factors such as climate [49]. It is less clear what the overall cost or benefit of herbivore damage in common milkweed may be, and whether that impacts population level metrics. Therefore, the objective of this study is to identify how herbivore damage affects an individual’s vital rates and how that translates to population projections. By linking the effect of herbivory on an individual to its effect at the population level, we may better understand how consumers drive population growth and structure across heterogeneous landscapes.

The most popularly known specialist herbivore of common milkweed is the monarch caterpillar, *Danaus plexippus*. Monarch butterflies exclusively lay their eggs on milkweed plants and their caterpillars primarily feed on milkweed leaves [30]. The application of the herbicide glyphosate in Midwestern agricultural fields of glyphosate-resistant corn and soybeans to milkweed populations that typically grow on the outskirts of these fields is causing milkweed population decline [20]. This decline has been implicated as the largest contributing factor to the drastic declines of monarch butterflies in recent years [10, 40]. Monarch larvae are the most studied specialist herbivores of common milkweed, and thus their decline may be representative of population decline in less extensively studied herbivores that rely on milkweed [19], namely the milkweed beetle, *Tetraopes tetraophthalmus*, and the large and small milkweed bugs, *Oncopeltus fasciatus* and *Lygaeus kalmii*. The negative effects on community structure and biodiversity caused by milkweed decline strongly implies that the relationship between herbivores and the common milkweed’s population dynamics is of urgent interest.

Many studies that investigate the effects of herbivory classify it by discrete damage levels [36, 21, 6, 32] to find evidence for or against population level consequences of tissue loss. Here, we quantify the intensity of herbivore damage on each individual and use a continuous covariate distribution of herbivore damage to predict an individual’s vital rates. The IPMs that are described in this thesis thus account for the natural variation of herbivory that is observed in the field, and subsequently provide a closer link on how changes in the intensity of herbivore damage that individuals experience may affect the fate of the population. By extension, as population growth rates may be used as a measure of fitness [22], our population model sheds light on the plant responses that are representative of an individual’s fitness. In studies of herbivore effects on common milkweed, many quantify fruit production as an estimate of fitness [23, 17]. Depending on the extent that sexual
reproduction contributes to the population growth rate, fruit production may or may not be an appropriate fitness measure to test meaningful effects of herbivory. We expect that herbivory will influence the population growth rate if it affects vital rates that the population growth rate is sensitive to.

Common milkweed are perennial and propagate clonally (asexually) and sexually. Throughout the year they have below ground roots with buds, some of which emerge as stems above ground during the growing season. They grow from mid-Spring to mid-Fall, after which the stems senesce and all that remains of the plants are their below ground roots. Although common milkweed are perennial, preliminary analysis revealed that the survival of a stem from one year to the next, characterized by the emergence of a stem in the same location, cannot be predicted from one year to the next by any of the many size-related traits we measured. This is because ramets, or stems, from the same genet, genetically identical entity, can emerge from different root locations each year. Therefore, to model our system, we treat every emerging bud at the beginning of the growing season as a clone. Thus, because we do not have genetic information to infer clonal relationships, we treat this perennial population as an annual population stems. In this respect we are not concerned with the survival and growth of an individual from one year to the next that are typical of IPMs. We are only concerned with sexual and clonal reproduction pathways. This system is represented using a matrix population model (MPM) to project the number of seedlings and number of emergent buds in the population through discrete time. IPMs are nested as elements within this MPM to evaluate the size dependent contributions to sexually reproductive output. This hybrid model has the power of the IPM to predict how vital rates affect reproductive output from individual variation in a continuous size structured population, and the clarity of the MPM to track the relative contributions made to seedlings and emergent buds, whose size distributions at the beginning of the growing season do not depend on that of the previous year.

We collected field data from five different populations in Virginia and constructed separate IPMs/MPMs for each. By comparing the results of each model, we asked what properties at each site may explain the observed spatial variation in herbivory effects, population growth rate, and size structure. The results of this study inform us of how spatial variation in herbivore abundance and plant response leads to different effects on common milkweed populations. This highlights the advantage of incorporating a continuous covariate herbivory distribution in IPMs that is used to explain the observed variation in vital rates. The ability to create a direct link between herbivore damage and vital rates allows us to predict how changes in the distribution or intensity of tissue loss may affect the population growth rate and size structure, thus advancing our understanding of the
demographic consequences of herbivory.
Chapter 2

Methods

2.1 Natural History

Common milkweed (*Asclepias syriaca*) are native to North America. They range as far north as New Brunswick, Canada, are spread across the eastern United States, and go as far west as North Dakota and Kansas [49]. The common milkweed is a perennial plant whose adventitious roots extend laterally and produce buds, some of which emerge as ramets, or stems. The ramets that come from the same root system are genetically identical and belong to the same genet. The roots remain underground all year but the stems’ growing season begins with emergence in late April and ends in early September. In June, the stems produce flowers in clusters that are called inflorescences. By the end of the growing season, after pollination, the inflorescences wilt and those flowers that are successfully pollinated develop pods. Each pod contains approximately 103 seeds (unpublished data). These seeds are attached to a cotton-like material that aids in wind dispersal and are released when the pods open in September. The information we recorded and subsequent model construction were guided by the life cycle of common milkweed (Figure 2.1).

![Figure 2.1: Life cycle diagram.](image-url)
2.2 Data Collection

For two years (2014 and 2015) over 2,500 tagged stems were followed across four different field sites in Virginia; each site has multiple transects that were one meter wide and varied in length: Blandy Experimental Farm (BLD), Presquile Wildlife Refuge (PWR), Yorktown National Battlefield (YTB), and Sky Meadows State Park (SKY). We treat BLD as two separate sites, with transects assigned according to whether they were burned in the late fall or not (BLD-Burned or BLD-Unburned). Information on sites and their general transect length characteristics are listed in Table 2.1. All stems were tagged and tracked throughout multiple censuses. A PCA of soil characteristics indicated that in 2013, cation exchange capacity explained most of the variation (47% of variance) in soil quality between sites. This was followed by parts per million of phosphorous (25% variance explained). In 2014, a PCA of soil characteristics indicated that soil pH explained most of the variation (41% of variance) in soil quality between sites, followed by parts per million of magnesium (22% of variance). Though these results differ between years, cation exchange capacity and pH covary as well as parts per million of phosphorous and magnesium and so one year’s results may sufficiently represent that of another year. Along the principal component axes in both years, sites somewhat overlap in their soil characteristics but can generally be distinguished, and there exist a few outlier transects.

<table>
<thead>
<tr>
<th>Site</th>
<th>Abbrev.</th>
<th>Total Transects</th>
<th>Transect Length (m), µ</th>
<th>Transect Length (m), σ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blandy Experimental Farm, Unburned</td>
<td>BLD-U</td>
<td>3</td>
<td>17</td>
<td>1.73</td>
</tr>
<tr>
<td>Blandy Experimental Farm, Burned</td>
<td>BLD-B</td>
<td>3</td>
<td>17.67</td>
<td>12.5</td>
</tr>
<tr>
<td>Presquile Wildlife Refuge</td>
<td>PWR</td>
<td>4</td>
<td>14.88</td>
<td>6.14</td>
</tr>
<tr>
<td>Sky Meadows State Park</td>
<td>SKY</td>
<td>2</td>
<td>14.16</td>
<td>5.41</td>
</tr>
<tr>
<td>Yorktown National Battlefield</td>
<td>YTB</td>
<td>5</td>
<td>18.74</td>
<td>7.81</td>
</tr>
</tbody>
</table>

We visited these sites annually at the beginning and end of the milkweed stems’ growing season (June and September, respectively). We measured (in June) height
to the apical meristem, stem diameter, length and width of the largest leaf, total number of leaves, whether the plant flowered, number of umbels produced, presence of stem damage, and level of herbivory experienced. We quantified a stem’s herbivory first by assigning each leaf on the individual a score on a scale of 0-6 according to percent of leaf tissue removed (0=leaf is whole and intact, 1=1-5% removed, 2=6-25% removed, 3=26-50% removed, 4=51-75% removed, 5=76-99% removed, 6=only the petiole remains). The scores of each leaf on a stem were then averaged to attain an individual’s herbivory score. Upon return in September we recorded each individual’s survival, height, and number of viable and unviable pods produced.

In May 2015 we designated each individual as a seedling or clonal sprout based on size differences and emergence patterns. In previous years, this distinction was not recorded. To determine the individual’s stage (seedling or emergent bud) in a year when it was not recorded, we modeled the relationship between size and stage in 2015 to predict the probability than an individual was a seedling from its size in previous years when it was not recorded. From the 2015 data, the function was modeled from a logistic regression of whether an individual was a seedling (a binary value) on the stem width of the individual in May. We used size in May because that is when the distinction between seedlings and emergent buds was most clear in the field. Sizes in May were not measured in 2014, so a function that predicted an individual’s stem width in May from its stem width in June was also modeled from a regression of May 2015 sizes against June 2015 sizes. With the linear model that predicted reverse stem width transitions from June to May and the logistic model that predicted the probability that an individual was a seedling from its stem width in May, we took the sum of each individual’s seedling probability to obtain a quantity that represented the total number of seedlings expected to have been present in all of the transects of a given site.

Ripe pods were collected from site BLD-U from two separate years and the number of seeds inside were counted. This information was used to calculate the average number of seeds per pod in each year they were collected. Those values were then averaged to attain the number of seeds per pod (103) that would be used in the model for each site.

2.3 Data Preparation

IDs that were recorded more than once in a single census were removed because it was unknown whether the duplicate data came from the same stem. Measurements that were obvious outliers, likely because of a recording error, were also removed. IDs whose traits in June were inconsistent with their traits in September were
2.4 Model Structure

An individual’s size and probability of survival from one year to the next cannot be predicted by any of the size characteristics we measured. Thus we treat this perennial plant as an annual population of stems and individuals described in the model are ramets. In this case, the projected population depends on the output of two reproductive pathways: sexual reproduction and clonal reproduction. Sexual reproduction depends on the production of seeds that establish and emerge as seedlings the following year. Clonal reproduction depends on the above ground emergence of a bud from an existing root system. In this regard we are concerned with the number of seedlings and emergent buds that are recruited to the population each year. The method of treating emergent buds as having emerged from the population of pre-existing roots, and not tracking which genet a ramet is a part of, is a model limitation but is necessary given the life history complexities of our study system. We cannot establish relationships between ramets and genets without sampling for genetic relationships and thus treat emergent buds as a population level property. This needs to be done if one wants to make any inference about the population and is not an uncommon approach to investigating and modeling clonal populations [24, 21, 46].

We constructed a matrix population model with some elements that are integral projection models (IPMs) to project the respective number of seedlings and emergent buds in the population through discrete time. For a more thorough explanation of the functions and their parameters that compose the model, refer to Box 2.1 (page 16). An accompanying proof of how the matrix population model is consistent with an integral projection model can be found in the appendix (A.1). The following text provides a more conceptual overview of the model structure. At time $t$ (June), $S_t$ represents the number of seedlings and $B_t$ represents the number of emergent buds. Relative to their respective number of individuals, the matrix population model tracks the contributions made by seedlings and emergent buds to the number of individuals in each stage at the following time step. The matrix population model, followed by the equations demonstrating the matrix multiplication, are listed below:

\[
\begin{bmatrix}
S_{t+1} \\
B_{t+1}
\end{bmatrix} =
\begin{bmatrix}
a & b \\
p_{em} & p_{em}
\end{bmatrix}
\begin{bmatrix}
S_t \\
B_t
\end{bmatrix}
\]

\[S_{t+1} = aS_t + bB_t\]

\[B_{t+1} = p_{em}(S_t + B_t)\]
a and b are constants that predict the respective number of seeds that establish and emerge as seedlings at time $t+1$ (following June) that are released per seedling or emergent bud. These values are quantified by evaluating integral projection models that apply the probability density distribution of either seedlings or emergent bud sizes, because sizes at the beginning of the growing season do not depend on that of the previous year. The integral projection models use this individual variation in size and therefore vital rates to sum over the varying contributions from individuals in one time step to the per capita number of seedlings in the next time step. $p_{em}$ is the number of buds that emerge per stem, which includes seedlings and emergent buds, the previous year. Contributions from seedlings and emergent buds to each stage at the next time step are added to determine the number of seedlings and emergent buds at the next time step, $S_{t+1}$ and $B_{t+1}$, respectively.

### 2.5 Integral Projection Model Structure

Elements $a$ and $b$ from the previously described matrix population model are constants that are evaluated from integral projection models. An integral projection model projects how the population changes over time by summing over every individual's contributions, and these contributions depend on an individual's size. Therefore populations that differ in their initial size distribution will make different contributions to the future of the population. This is the basis of what makes $a$ and $b$ different. $a$ represents the number of seedlings that emerge per seedling and $b$ represents the number of seedlings that emerge per emergent bud. The integral projection model structure for each is the same except for the initial size distribution that the population is projected from. $a$ is evaluated using a seedling size distribution and $b$ is evaluated using an emergent bud size distribution. The following goes into greater detail about the integral projection model structure.

Individuals are characterized by their size, $z_h$, and the population is projected in discrete time using a yearly time step from June ($t$) to June ($t+1$). Within this time frame there is an intermediary step in September ($t+\tau$, $\tau < 1$). The population is described by a probability density distribution, $n(z_h, t)$. An individual's herbivory score is represented by $z_\omega$. The covariate herbivory distribution, meaning the probability density distribution to have herbivory score $z_\omega$, is included in the model as $\rho_\omega(z_\omega)$. A given individual’s herbivory score is not correlated to an individual’s size.

For the purpose of generality, the model described here incorporates the effects of herbivory on each function. Refer to Box 2.1 or Figure 3.4 in the Results chapter to see how herbivory effects vary between sites. $p_{es}$ is the estimated number of seedlings that emerge per seed released and $\nu$ is the estimated number of seeds per
pod. Given a size distribution of seedlings or emergent buds, $\rho_S(z_h)$ and $\rho_B(z_h)$, respectively, the integral projection model sums over a kernel that maps size dependent contributions from individuals in one time step to the number of seedlings in the next time step.

The structure of the IPM kernel is determined by whether the number of pods produced in September is best predicted by an individual’s size in June or September. If it is better predicted by an individual’s size in June, the fecundity kernel, $F_t(z_h, z_\omega)$, is composed of survival and fecundity functions of size and herbivory score in June to predict the number of seedlings recruited to the population the following year. This is described by the following formula, which is used to construct both $a$ and $b$:

$$p_{e\nu} \int \int F_t(z_h, z_\omega) \rho_\omega(z_\omega) n(z_h, t) dz_h dz_\omega$$

where in $a$, the population is projected from the seedling size distribution:

$$n(z_h, t) = \rho_S(z_h)$$

and in $b$, the population is projected from the emergent bud size distribution:

$$n(z_h, t) = \rho_B(z_h)$$

If the number of pods produced in September is better predicted by an individual’s size in September, $z_h'$, the fecundity kernel, $F_{t+\tau}(z_h', z_\omega)$, is a function of that September size and June herbivory score. The size distribution in September is mapped by the flowering, survival, and growth kernel, $P_t(z_h', z_h, z_\omega)$, which is a function of size and herbivory score in June to give a probability density of possible sizes that an individual in June that flowered and survived can transition to in September. This is described by the following formula, which is used to construct both $a$ and $b$:

$$\int \int F_{t+\tau}(z_h', z_\omega) n(z_h', z_\omega, t + \tau) dz_h' dz_\omega$$

where in $a$, the population is projected from the seedling size distribution:

$$n(z_h', z_\omega, t + \tau) = \int P_t(z_h', z_h, z_\omega) \rho_\omega(z_\omega) \rho_S(z_h) dz_h$$

and in $b$, the population is projected from the emergent bud size distribution:

$$n(z_h', z_\omega, t + \tau) = \int P_t(z_h', z_h, z_\omega) \rho_\omega(z_\omega) \rho_B(z_h) dz_h$$
2.6 Model Selection

The functions included in the IPM kernels were parameterized from regression analysis of the collected data. Survival within the growing season and whether an individual flowered in June were modeled logistically using a binomial regression. The number of pods produced in September are count data and thus modeled using a Poisson regression. If the growth function was included in the IPM for a given site (listed as "Case 2" in Box 2.1), growth from June to September was visually linear and thus modeled using a linear regression. The stems that enter the sexual reproduction pathway are conditional on whether they flower in June, and therefore the functions following flowering probability should be parameterized from regressions of just the data from the individuals that flowered. This was done for growth and pod production, but not survival because of resulting inadequate data to estimate parameters significant from 0. Refer to the appendix (A.1.3) for an explanation of this decision.

In R, we regressed vital rate responses on the following traits: height to the apical meristem, stem diameter, leaf length, leaf width, and total number of leaves. The use of more than one trait to predict vital rates was also considered. This was accomplished by identifying which trait alone had the lowest AIC, followed by comparing that value to the AIC of that same trait and an additional trait as predictors to see if the model improved. If including an additional trait as a predictor lowered the AIC, we considered whether the application of these two traits to predict vital rates in the model was worth the associated computational complexities. In a similar respect, we evaluated whether the inclusion of herbivory in addition to a plant trait as a predictor improved the model. Interaction effects and correlations between traits were also investigated for subsequent consideration. The most fitting variable(s) to characterize individuals were chosen based on comparatively low AIC values with P-values lower than the significance level 0.05 for the slope associated with those traits.

Per capita seedling emergence from the number of seeds released and emergent buds per stem the previous year are population-level constants in the model. Their values were determined by bootstrapping 3000 samples with replacement from the observed data and using those samples to estimate each parameter. The respective estimates were then averaged from the results of all samples. Those averaged values were used in the model.
2.7 Analysis

The IPMs were numerically analyzed in R by discretizing the kernels into large matrices. The resulting matrix was evaluated using Eigen analysis to determine the population growth rate, $\lambda$. Confidence intervals of $\lambda$ were determined by bootstrapping samples with replacement of stems recorded in the collected data, using that sample to re-parameterize vital rate functions, and subsequently evaluating $\lambda$ each time. We repeated the procedure 800 times for each site to evaluate confidence intervals. Choosing a greater number of samples would not effectively change the results and would thus be unnecessarily time consuming.

Sensitivity of $\lambda$ to herbivory was evaluated by simulating a point mass distribution of herbivory scores in the population at integers in the range of possible scores, [0,6]. This point mass distribution represents a population in which every plant in the field experiences the same average tissue damage. Using the same bootstrapping method as previously discussed, confidence intervals of $\lambda$ were determined for each simulated point mass distribution.

Sensitivity of $\lambda$ to model parameters was evaluated by perturbing model parameters and identifying the change in $\lambda$, representing the rate of change of lambda for each model parameter. For each parameter, its value was increased by 1, $\lambda$ was evaluated, and then the sensitivity was calculated as the absolute change in $\lambda$ per absolute change in parameter value. The perturbation was then divided by 2 and the process of evaluating lambda and its sensitivity was repeated. This continued until smaller perturbations did not cause a noteworthy change in sensitivity. If the proportional change in sensitivity between a perturbation value and its subsequent halved value did not exceed $\epsilon = 0.000001$, then the sensitivity was determined to have converged, and that sensitivity value was used to represent the rate of change of lambda to that parameter. Using that same recorded information for sensitivity, the elasticity of lambda was evaluated. While the sensitivity of lambda to a parameter represents the change in lambda per absolute change in parameter, the elasticity represents the change in lambda per proportional change in parameter. Therefore the elasticity was calculated as such and recorded for each parameter.

2.8 Supplementary Correlation Tests

The clonal reproduction pathway is represented by a constant, $p_{em}$, that is evaluated by averaging the number of buds that emerged in one year per stem present the previous year. In order to understand the environmental factors that may influence the variation in these estimates between transects, sites, and years, we investigated the transect-level correlation between $p_{em}$ and density, average her-
bivore damage, and soil quality in 2013 and 2014. Soil quality is represented by
cat-ion exchange capacity and parts per million of phosphorous. While these two
soil traits are not the ones that explain most of the variance in 2014, they highly
covary with those that do and are thus sufficiently representative of soil quality in
each year.
Box 2.1: Model Formula and Notation

<table>
<thead>
<tr>
<th>Notation</th>
<th>Definition</th>
<th>Notation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>$t$</td>
<td>Time, June</td>
<td>$\rho_S(z_h)$</td>
<td>Seedling size distribution</td>
</tr>
<tr>
<td>$t+\tau$</td>
<td>Time, September</td>
<td>$\rho_B(z_h)$</td>
<td>Emergent bud size distribution</td>
</tr>
<tr>
<td>$t+1$</td>
<td>Time, June following year</td>
<td>$p_{em}$</td>
<td>Buds that emerge per capita</td>
</tr>
<tr>
<td>$S_t$</td>
<td>Number of seedlings, time $t$</td>
<td>$p_{es}$</td>
<td>Seedlings that emerge per capita</td>
</tr>
<tr>
<td>$B_t$</td>
<td>Number of emergent buds, time $t$</td>
<td>$\nu$</td>
<td>$\text{Avg. number of seeds per pod (-103, same value used at all sites)}$</td>
</tr>
<tr>
<td>$z_h$</td>
<td>Plant size, height, time $t$</td>
<td>$s(z_h,z_\omega)$</td>
<td>$\text{Pr[Survive to } t+\tau\text{]}$</td>
</tr>
<tr>
<td>$z'_h$</td>
<td>Plant size, height, time $t+\tau$</td>
<td>$f(z_h,z_\omega)$</td>
<td>$\text{Pr[Flower, time } t\text{]}$</td>
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<tr>
<td>$z_\omega$</td>
<td>Herbivory score, time $t$</td>
<td>$G(z'_h</td>
<td>z_h,z_\omega)$</td>
</tr>
<tr>
<td>$\rho_\omega(z_\omega)$</td>
<td>Covariate herbivory distribution</td>
<td>$p(z_h,z_\omega)$</td>
<td>Number of pods produced</td>
</tr>
<tr>
<td>$n(z_h,t)$</td>
<td>Size distribution, time $t$</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

— Model Formulation —

$$
\begin{pmatrix}
S_{t+1} \\
B_{t+1}
\end{pmatrix} =
\begin{pmatrix}
a & b \\
\rho_{em} & \rho_{em}
\end{pmatrix} \begin{pmatrix}
S_{t} \\
B_{t}
\end{pmatrix}
$$

$a$ and $b$ are constants evaluated from integral projection models that estimate the number of seedlings that establish and emerge from the population. They differ in their initial size distributions, $n(z_h,t)$ at time $t$. In $a$, $n(z_h,t) = \rho_S(z_h)$ and in $b$, $n(z_h,t) = \rho_B(z_h)$.

[Case 1] If pod production is best predicted by height in June, $a$ and $b$ are given by

$$
p_{es}\nu \int s(z_h,z_\omega)f(z_h,z_\omega)p(z_h,z_\omega)\rho_\omega(z_\omega)n(z_h,t)dz_hdz_\omega
$$

[Case 2] If pod production is best predicted by height in September, $a$ and $b$ are given by

$$
p_{es}\nu \int p(z_h,z_\omega)n(z'_h,z_\omega,t+\tau)dz_hdz_\omega
$$

These equations are a general representation of the model, though the functions that compose the IPMs vary in whether they include herbivory or not. Herbivory was included if (i) it lowered the AIC from regression analysis by 2 or more compared to a regression of the vital rate against height alone, and (ii) its associated slope was significantly different from 0 using significance level 0.05. The below table specifies the functions and their parameters. If a
parameter is 0, then herbivory was not included in the function based on the described criteria and the parameters were determined from a regression of the vital rate on height alone.

<table>
<thead>
<tr>
<th>Description</th>
<th>Function</th>
<th>Parameters</th>
</tr>
</thead>
</table>
| **Probability of Survival**  | $\text{logit}(p_s(z_h, z_\omega)) = \alpha_s + \beta_s z_h + \beta_s z_\omega$ | $\begin{array}{ccc} 
\alpha_s & \beta_s & \beta_{s_\omega} \\
BLD-U & -1.92 & 0.04 & -0.41 \\
BLD-B & -1.84 & 0.06 & -2.19 \\
PWR & -3.23 & 0.03 & 0 \\
SKY & -1.21 & 0.03 & -2.87 \\
YTB & -2.99 & 0.06 & 0 \\
\end{array}$ |
| **Probability of Flowering** | $\text{logit}(f(z_h, z_\omega)) = \alpha_f + \beta_f z_h + \beta_f z_\omega$ | $\begin{array}{ccc} 
\alpha_f & \beta_f & \beta_{f_\omega} \\
BLD-U & -4.31 & 0.09 & -1.25 \\
BLD-B & -7.39 & 0.14 & -5.19 \\
PWR & -8.00 & 0.10 & 1.82 \\
SKY & -5.36 & 0.10 & -1.73 \\
YTB & -5.89 & 0.06 & 0 \\
\end{array}$ |

**Case 1**

| Pod Production               | $p(z_h, z_\omega) = \exp(\alpha_p + \beta_{p_\omega} z_h + \beta_{p_\omega} z_\omega)$ | $\begin{array}{ccc} 
\alpha_p & \beta_{p_\omega} & \beta_{p_\omega} \\
SKY & -2.07 & 0.02 & 0 \\
YTB & -6.36 & 0.08 & -1.79 \\
\end{array}$ |

**Case 2**

| Growth Probability Density  | $G(z_h', z_h, z_\omega) = \alpha_g + \beta_{g_\omega} z_h + \beta_{g_\omega} z_\omega + \mathcal{N}(0, \sigma)$ | $\begin{array}{ccc} 
\alpha_g & \beta_{g_\omega} & \beta_{g_\omega} & \sigma \\
BLD-U & 81.12 & 0.38 & -7.02 & 16.17 \\
BLD-B & 40.34 & 0.70 & -26.92 & 13.73 \\
PWR & 15.92 & 0.90 & -7.89 & 6.52 \\
\end{array}$ |
| Pod Production               | $p(z_h', z_\omega) = \exp(\alpha_p + \beta_{p_\omega} z_h' + \beta_{p_\omega} z_\omega)$ | $\begin{array}{ccc} 
\alpha_p & \beta_{p_\omega} & \beta_{p_\omega} \\
BLD-U & -0.74 & 0.02 & 0 \\
BLD-B & -6.03 & 0.06 & 0 \\
PWR & -1.03 & 0.01 & 0 \\
\end{array}$ |

**Population Level Estimates**

<table>
<thead>
<tr>
<th>Site</th>
<th>$p_{es}$</th>
<th>$p_{em}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>BLD-U</td>
<td>7.9e-4</td>
<td>1.70</td>
</tr>
<tr>
<td>BLD-B</td>
<td>1.9e-4</td>
<td>1.63</td>
</tr>
<tr>
<td>PWR</td>
<td>5.23e-5</td>
<td>1.17</td>
</tr>
<tr>
<td>SKY</td>
<td>2.0e-3</td>
<td>0.96</td>
</tr>
<tr>
<td>YTB</td>
<td>3.3e-4</td>
<td>0.80</td>
</tr>
</tbody>
</table>
Chapter 3

Results

3.1 Vital Rate Regressions

Regressions that used different stem traits to explain vital rate responses were compared to determine which trait(s) would characterize individuals in the model. Guided by comparatively low AIC values, P-values lower than the significance level 0.05 for the slope associated with size in regression summaries, and relatively greater accuracy in field measurement, an individual’s height to the apical meristem was chosen as the explanatory size variable in the model. The use of more than one size-related trait was also considered, but the disadvantage of a more computationally complicated model outweighed the benefits of a model that only slightly improved in accuracy. Each size-related trait is moderately to highly correlated to the others, and thus the predictions made by one trait sufficiently represent the predictions made by another. Figure 3.1 displays plots of each vital rate response against an individual’s height using data from site BLD-U. It is a visual confirmation that an individual’s height is a strong predictor of its vital rates. The June size distributions of seedlings \( \rho_S(z_h) \) and emergent buds \( \rho_B(z_h) \) in the model were assigned based on height observed in the field (Figure 3.2).

Our collected data show that every site varies in its range and distribution of herbivory scores that an individual experiences (Figure 3.3). Therefore while the covariate herbivory distribution is consistently modeled as lognormal, the mean and standard deviation differ for each site and thus the herbivory distribution is represented differently in each site’s model. The level of herbivore damage an individual experiences, determined by the covariate herbivory distribution (Figure 3.2), was included as an explanatory variable in a function when the AIC value decreased by at least 2 upon inclusion of herbivory and the P-value of its associated slope was lower than the significance level 0.05. Based on our criteria, sites vary in their vital rates that include herbivory (Box 2.1, Figure 3.4). There is no vital rate
Figure 3.1: Vital rate regressions from field collected data. Black dots represent the observed height (cm) and vital rate responses of stems that were recorded and tracked over time. Orange lines are the predictions of vital rate responses from height using results from regression analysis. The data and regressions shown here are from site BLD-U. Survival and Flowering response values are only 0 or 1; the spread of points around those binary values is just for visualization purposes.

in which all sites did or did not include herbivory as an explanatory variable. For each vital rate, we explain which sites include herbivory in their model (Section 3.2).

When interpreting Figure 3.4, it is important to remember that the scale of slope estimates varies because of the relative magnitude of each response variable. The growth function has higher slope estimates because it involves a greater absolute change in stem height between June and September. The probability of survival and the probability of flowering both range in response from 0 to 1. Considering these similar scales, herbivory generally has a larger effect on probability of flowering than probability of survival but comparison of these vital rates within sites reveals that the effect of herbivory on flowering is not consistently larger than
3.2 Herbivory Effects

3.2.1 Survival

The survival of an individual to the time of pod production is modeled as a function of height in June and, depending on the site, herbivory. Herbivory decreased survival at 3 of the 5 sites (BLD-U, BLD-B, SKY) and had its greatest effect on survival at SKY. We did not detect an effect of herbivory on survival at PWR and YTB.

3.2.2 Fecundity

The probability that an individual produces inflorescences in June is modeled as a function of height in June at every site. We detected an effect of herbivory on vital rates. With all of this in consideration, it is difficult to concede local trends in the effects of herbivory on vital rates.
Figure 3.3: Horizontal box plots for each site that summarize the distribution of individual average herbivory scores.

The flowering probabilities at all sites except for YTB. Of the sites that do include herbivory (BLD-U, BLD-B, SKY, PWR) in the flowering probability function, herbivory decreased flowering probability at BLD-U, BLD-B, and SKY, and increased flowering probability at PWR. Herbivory had the greatest effect on survival at BLD-B.

The number of pods produced by a stem is modeled as a function of height in September in sites BLD-U, BLD-B, and PWR and as a function of height in June in sites SKY and YTB. Herbivory decreased pod production in YTB. We did not detect an effect of herbivory on pod production at the other sites (BLD-U, BLD-B, PWR, SKY).

3.2.3 Growth

The growth of stems from June to September is only included as a function in the sites whose pod production is best predicted by stem height in September: BLD-U,
Figure 3.4: Herbivory slope estimates at each site for different vital rates. Orange bars represent slope estimates that were significantly different from zero (significance level $\alpha = 0.5$) and therefore included in the function predicting the respective vital rate. Gray bars represent slope estimates that were not significantly different from zero. Black line segments on the bars represent 95% confidence intervals on the estimate. Striped bars are used when the function for that vital rate is not included in the model for a given site.

BLD-B, and PWR. Of those sites, herbivory decreased growth in all of them, with the greatest effect on BLD-B.

### 3.3 Population Level Metrics

Four of the five sites have increasing population size (BLD-U, BLD-B, PWR, SKY) and one has decreasing population size (YTB, Figure 3.5). Analysis of the population growth rate’s sensitivity to herbivory reveals that the population growth rates at sites BLD-U and SKY decrease as the intensity of herbivory increases until approximately a mean herbivory score of 3, at which it does not qualitatively change (Figure 3.6). Increasing herbivory does cause the population growth rate
at SKY to change from increasing to decreasing population size. The other sites (BLD-B, PWR, YTB) have population growth rates that do not effectively change as herbivory increases.

Cross-site comparison reveals that the population growth rate is consistently, and by relatively large magnitude, most sensitive to the number of seedlings that emerge per seed released the previous year (Figure 3.7a). There is no absolute consistency in the order of parameters that follow, but all sites’ sensitivity results do include the same ones: pod production, per capita emergence of sprouts from stems the previous year, probability of survival, and probability of flowering. Therefore, while the population growth rate is most sensitive to seedlings that emerge per seed released, which is part of the sexual reproduction pathway, it is also sensitive to other parameters that affect both the clonal and sexual reproduction pathways.

Consistent in each site, the population growth rate is most elastic to the number
of buds that emerge per stem the previous year (Figure 3.7b). This elasticity has almost the same value in each site, ranging from approximately 0.939 to 0.995. The order of the following parameters that the population growth rate is most elastic to varies, though parameters associated with pod production and sprout size are consistently included in the top 5. Sites whose models include the growth function had the population growth rate elastic to at least one growth related parameter. The elasticity of the population growth rate to the parameters that follow the per capita emergence of sprouts from stems vary in effect size relative to that of per capita sprout emergence. PWR and BLD 2 elasticities are negligible compared to the elasticity to per capita sprout emergence. BLD 1, YTB, and SKY have population growth rates whose elasticities level off but still have a noticeable effect size compared to per capita sprout emergence.

3.4 Transect Level Environmental Relationships to Buds Per Stem

The transect-level average herbivory score in 2014 is strongly, positively related ($\rho = -0.813$, $P = 0.0043$) to the number of buds that emerged in 2015 per stem in 2014 in all sites except for YTB, which is an outlier population (Figure 3.8). There was only one year of data available to estimate this correlation. Transect-level density estimates in 2013 and 2014 were not related to buds that emerged the following year per stem in the respective year of density estimates. Herbivory in a given year was not related to density in that year. Transect-level cat-ion exchange capacity and parts per million of phosphorous, the two estimates of soil quality that explain most variation between all samples taken in 2013, are not related to buds that emerge in a given year per stem in the previous year. Across all transects, average herbivory increased by about 150% from 2014 to 2015. The number of emergent buds per stem in a transect are not related from one year to the next. Density estimates in a transect are not related from one year to the next.
Figure 3.6: Population growth rate sensitivity to herbivory. Changes in the population growth rate and are recorded at each level of herbivore intensity, which represents cases in which each stem experiences the same mean level herbivore damage. Confidence intervals are too small to be seen. The purple points represent the population growth rate results at each herbivory score integer. The green points represent the population growth rate if each individual were to experience the mean damage level observed and recorded in the field. The orange line represents stable population size over time.
Figure 3.7: Sensitivity and elasticity of population growth rate to model parameters. (a) The five model parameters for each site that the population growth rate is most sensitive to, in decreasing order. (b) The five model parameters for each site that the population growth rate is most elastic to, in decreasing order.

**Key**
- $p_e$: Seedlings that emerge per capita
- $p_m$: Buds that emerge per capita
- $\beta_{s}$: Slope; survival probability
- $\beta_{f}$: Slope; flowering probability
- $\alpha_k$: Intercept; growth probability density
- $\beta_{p}$: Slope; pod production
- $\mu_k$: Mean; emergent bud size
- $\sigma_k$: Std. Deviation; emergent bud size
Figure 3.8: Correlation between transect level buds per stem and herbivory. Blue dots represent data from transects at all sites except for YTB. Orange dots represent data points from YTB. The dashed line is used to visually distinguish results of the outlier population from all other sites. When transects from all sites are evaluated, the correlation coefficient is near zero and not significant. When transects from all sites except for YTB are evaluated, the correlation is strong, negative, and significant.
Chapter 4

Discussion

4.1 Linking Environmental Factors to the Population Growth Rate

The similarities and differences between parameter estimates for each site, and thus population growth rates, may be explained by environmental properties and other factors that affect them. As shown in Figure 3.7 in the results section, the population growth rate is sensitive and elastic to parameters in both the clonal and sexual reproduction pathways. As such, environmental factors that may influence either of these pathways are possible explanations for spatial variation in population growth rate.

At all sites, the population growth rate is most sensitive to total seedling establishment and emergence per seed released the previous year. Anthropogenic interference that affects the ability of seeds to establish and emerge may thus have a large effect on the population growth rate. As was mentioned earlier, Blandy Experimental Farm is treated as two separate sites, BLD-U and BLD-B, because one of the meadows from which data was collected is burned in the late fall every three years. During data collection in June 2015, we observed an unusually high number of seedlings in the unburned meadow but none in the burned meadow. The burned meadow has less interspecific competition, a slighter layer of litter, and presumably warmer ground temperatures from the burning, all of which would suggest more favorable conditions for seedling emergence than the unburned meadow. Since seedling emergence did not occur, and the transects from each meadow have similar soil properties with one exception, it is probable that the meadow gets burned before the seeds are deep enough in the ground to be protected, subsequently destroying the opportunity for the seeds to establish. Mowing at Yorktown Battlefield (YTB) in the fall may have similar effects to burning at BLD-B. If stems are mowed
before their pods release their seeds, the per capita establishment and emergence of seedlings from seeds will be largely reduced. Another possibility is that mowing reduces seedling emergence because it adds a layer of thatch that decreases light that is necessary for growth. Cases of interference vary between sites, attributing to some of the differences between their population growth rates.

Density estimates are highly variable between transects and are not correlated between years. Though we could not detect an effect of density on clonal reproduction, we have evidence that density negatively affects an individual’s vital rates (unpublished data) and should thus be included in the model. Such incorporation would equip the IPM with greater predictive capacity, especially because it is directly related to population size [16].

The effects of herbivory on vital rate responses and per capita estimates may also explain spatial variation in the population growth rate. This is both a matter of spatial variation in levels of herbivore intensity and the effects that herbivory has on plant responses. Depending on where in the model herbivory has an effect, and whether that model component is meaningful at the population level, herbivory may be an environmental factor that drives common milkweed population dynamics.

4.2 Spatial Variation in Herbivory Effects and Population Level Consequences

The effect of herbivory on an individual’s survival, growth, and fecundity varies spatially. Comparison of herbivory effects on a vital rate between sites shows that there is no vital rate that herbivory consistently has an effect on. The results get closest to consistency in the effect of herbivory on an individual’s flowering probability (Figure 4), in which herbivory is detected to have an effect on 4 out of the 5 sites (BLD-U, BLD-B, SKY, PWR). Still, of those 4 sites, herbivory decreases flowering probability in 3 sites (BLD-U, BLD-B, SKY) and increases flowering probability in the other site (PWR). It is therefore difficult to determine trends of herbivory effects among all sites. Our results align with other studies that find negative effects of herbivory on an individual’s growth and fecundity [23, 1, 5, 17, 50].

Different effects of herbivory at different sites could be attributed to differences in the composition of the herbivore community. Studies have shown that different herbivores cause different responses in the host plant which could explain spatial variation in herbivore effects on different vital rates. For example, foliar damage caused by monarch larvae leads to a higher reduction in photosynthetic rates of
common milkweed than damage caused by tussock moth larvae \textit{(Euchaetes egle \textit{L.})} [14]. In the case of milkweed latex production, dogbane leaf beetles \textit{(Chrysochus auratus)} were found to elicit higher latex production than by monarch larvae [47]. Growth of monarch larvae is negatively affected if they feed on a previously damaged leaf [47, 17]. Thus monarch avoidance of damaged leaves may alter herbivore community structure. If different herbivores that cause milkweed tissue loss elicit different plant responses and there is spatial variation in the composition of the herbivore community, then we can expect spatial variation in herbivore effects such as demonstrated in our results.

Red milkweed beetles are a good example of how spatial variation in community structure can subsequently affect vital rate responses. Experiments have shown that certain environmental factors can mediate their abundance and that based on their life cycle stage, they have unique effects on milkweed responses [1, 17].

The larvae of red milkweed beetles feed on below ground milkweed roots and such root herbivory is known to reduce a host’s biomass, fruit production, and fruit mass [1]. Root herbivory by red milkweed beetle larvae have different effects on plant performance than their adults above ground. Further investigation of what affects their relative abundance would allow for a more refined understanding of how their respective damage influences milkweed performance that the population growth rate is sensitive to. The attraction of red milkweed beetles to milkweed patches that are surrounded by grasses is of special interest because we know that our site YTB competes with grasses. It has the lowest (and decreasing) population growth rate and the highest levels of natural herbivory that we observed among all of the sites. It is possible that the high levels of herbivory are correlated with the surrounding grasses. We have not quantified the relative abundance of different herbivores but from observation can attest to the high levels of red milkweed beetles. More than a matter of herbivory levels, red milkweed beetles and competition with grasses at YTB might also affect clonal reproduction. It was found that independently, grass competition reduces clonal propagation, characterized by the number of new stems observed after a year, and root herbivory increases clonal propagation [1]. The multiplicative effect of grass competition and root herbivory, though, reduces clonal propagation at a greater magnitude than either of the additive effects of those factors alone [1]. Depending on the levels of grass competition and root herbivory at a given transect in YTB, there could be a multitude of effects on clonal propagation. This is a possible explanation for YTB as an outlier population in the relationship between herbivore damage and the estimate of buds per stem (clonal pathway) (Figure 3.8).

Levels of herbivore intensity and its effects on vital rate responses vary spatially. It is possible that environmental factors that mediate community interactions lead
to spatial variation in community structure. Depending on the relative abundance of herbivores present, milkweed responses to herbivory may vary, and thus may explain why we cannot identify trends in herbivory among all populations.

Even though these effects are not consistent in each site, we know that herbivory affects vital rates and the number of emergent buds per stem. Vital rates compose the integral projection model that predicts sexually reproductive output while emergent buds per stem is a population level estimate that determines clonal reproduction. By quantifying the relative contributions of the components that make up each recruitment pathway to population dynamics, we know where in the model to expect herbivory effects to influence the population growth rate.

While we have shown that herbivory does have an effect on sexually reproductive output, our model does not indicate that herbivory has a meaningful effect on the population growth rate. This is likely because in our current model, herbivory only has an influence on the sexual reproduction pathway. While the population growth rate is sensitive to parameters in this pathway, even at low herbivory they have a negligible effect because of the low values of their current estimates. Specifically, the number of seedlings that emerge per seed released in each site is typically on the order of $10^{-3}$ to $10^{-5}$ and therefore seedlings make up a small proportion of the population. This is typical of clonal and long lived plants [43]. Population models for individuals capable of both sexual and asexual reproduction demonstrate that survival between discrete time steps and clonal propagation contribute most to population dynamics [24, 21, 32].

Even though sexual reproduction makes little contribution to the population growth rate, it is still vital for dispersal and maintains genetic diversity in a population [48] and we know that herbivory decreases sexually reproductive output in common milkweed. This contrasts the expectation that plants that are under stress will allocate more resources to sexual reproduction [5]. Sexual reproduction allows for recombination to produce offspring genotypes that may have greater fitness in the environmental conditions that the parent experiences [37]. There exists empirical evidence that the stress of herbivore damage may cause a greater allocation of resources to sexual reproduction in perennial plants that reproduce sexually and asexually [18]. It is possible that herbivory causes a greater allocation of resources to sexual reproduction in PWR because it increases the probability that an individual flowers. There also exists a gap in the literature about how herbivores might affect seed quality. Other species are known to have compensatory mechanisms in which herbivory increases seed quality [6], thereby increasing its probability of establishment, but this possibility remains unexplored in common milkweed. Nonetheless, the overall effect of herbivory on different vital rates decreases the number of pods an individual will produce. Under the current assumption that
there is an average number of seeds per pod that is unaffected by herbivory, then herbivory decreases the number of seeds released. SKY has the highest estimate of per capita seedling emergence among all of the sites and its per capita bud emergence is below a one to one ratio of buds that emerge per stem. An increase in herbivore intensity at SKY causes the population growth rate to decrease below stable population size (Figure 3.6). If the estimate of seedling emergence per seed released was to increase at other sites, herbivore damage would have an effect on seed production that would be more easily detectable in the population growth rate as well. High levels of herbivory almost completely diminish contributions of the sexual reproduction pathway. This threatens genetic diversity and the population growth rate is reduced to the estimate of per capita bud emergence in the clonal reproduction pathway.

4.3 Considerations on per capita bud emergence

Currently, the model does not incorporate a causal relationship between herbivore damage and the per capita estimate of buds that emerge the following year, but we do have evidence of a strong, negative correlation between them in 4 of our 5 sites (BLD-U, BLD-B, PWR, SKY). We cannot however rule out the possibility that the effect of herbivory is confounded with the effect of another factor on bud production. Still, our transect-level estimates of density and soil quality are not related to emergent buds per stem or herbivory. Therefore to the best of our knowledge, herbivores are the drivers of the observed variation in clonal propagation. Herbivory is known to decrease asexual reproduction in other clonal species as well [46]. Contradictory to this, there is evidence that clonal propagation decreases when conditions are good and increases in times of stressful environmental conditions so that genets persist even when ramets die [13, 41]. This framework aligns with the trend observed in common milkweed that populations in higher stress environments have higher clonal propagation [49], though this is not relevant when considering plastic responses to changing environmental conditions instead of population level differences. It is possible that there exist thresholds of stress levels that determine when ramets invest more or less in clonal propagation, but this has not been studied.

Across all sites, transect-level herbivory scores increased by about 150% from 2014 to 2015. The winter preceding the summer of 2015 was more mild than the winter in 2014, and so warming climate is a likely explanation for increasing herbivore intensity [7]. Since we have evidence that increasing herbivore intensity reduces clonal reproduction, and clonal reproduction in turn contributes to the majority of population growth, the inclusion of this effect is a crucial next step in the model.
if we want a meaningful understanding of how herbivores affect population level metrics in common milkweed.

The decreasing order of population growth rates for our sites match the decreasing order of per capita bud emergence from stems. This makes sense because the population growth rates are most elastic to this parameter and the sexual reproduction pathway makes minimal contributions at its current estimates. BLD-U and BLD-B have population growth rates that are not significantly different from each other. Their similar population growth rates are matched by almost identical estimates for per capita bud emergence. These two sites do not however have similar parameters for pod production and per capita seedling establishment and emergence. This indicates that either (i) these parameters vary but together cause a similar number of seedlings to emerge the following year or (ii) the sexual reproduction pathway, at its current parameter estimates, plays a negligible role in affecting the population growth rate. The aforementioned possibilities may generally imply the same thing when almost no seedlings are recruited to the population. Regardless, consistent among our study sites, clonal propagation determines the population growth rate. This necessitates an examination of the potential drivers of this estimate.

Another parameter that needs refinement in the model is the estimate of emergent buds per seedling. Presently, estimates for per capita bud emergence are the same for seedlings and emergent buds. Yet emergent buds are generally of larger size than seedlings and should thus have more resources available to allocate to clonal bud production and emergence than seedlings do. Analysis of data that was collected for a separate experiment reveals that there is size dependence in the number of buds produced (unpublished data). We do not have that data on a per capita basis and so it is not of a form that is useful to the model. Results from another study agrees with our inferred relationships, though, as common milkweed root-to-shoot ratio was positively correlated with the number of root buds below ground [49]. Collection of data to properly evaluate this size dependent estimate will allow for greater accuracy in the variable contributions that seedlings versus emergent buds can make to the projected population. Yet while survival within a season only affects the sexual reproduction pathway in our model, it would be interesting to see how this effect on the ramet affects bud production below ground, likely through its effect on below ground biomass.

The focus on above versus below ground biomass could subsequently allow for a connection in the model between ramet survival and bud production. Currently, survival within the season only affects the sexual reproduction pathway yet above ground senescence likely has an effect on below ground biomass. A link between ramet survival and bud production could refine the model by allowing for slightly
more realistic predictions.

Seedlings cannot reproduce sexually for their first two years [45]. The model parameter that represents seedling contributions to seedlings at the next time step, \( a \), is negligible because the distribution of smaller seedling sizes does not lead to meaningful pod production. The treatment of seedlings as emergent buds through clonal reproduction, on the other hand, permits seedlings to be of larger size that can make more meaningful contributions to seedling counts in the following time step. Although the sexual reproduction pathway currently makes little difference in population growth, a modification must be made in the model if the estimate of per capita seedling emergence was to increase because there would be an unrealistic recruitment of seedlings that in their second year could make the same contributions to the population as ramets from a genet that is many years older.

As it has been previously discussed, we have evidence that herbivores decrease per capita bud emergence. Since herbivores generally decrease sexually reproductive output as well, it is clear that our populations do not have the compensatory ability to offset fitness losses from all herbivore damage. Our results are concordant with a previous experiment at BLD that compared patch-level estimates of clonal reproduction between a control patch with natural herbivory and a patch in which above ground beetles were removed [34]. This experiment found that the control patch that experienced foliar damage had a significant reduction in clonal reproduction compared to the patch with no herbivores. This could be attributed to other factors that were not measured, but it does align with trends that we found at the same site. However, these results do not distinguish between the effects of root herbivores and foliar herbivores. This question remains relevant.

Herbivore effects on clonal reproduction is interesting when considering previous studies that followed local changes in plant quality following tissue loss. Red milkweed beetles that caused foliar damage above ground sequentially increased performance of its larvae, whose life stage feeds on milkweed roots [17]. This suggests an increase in below ground root quality as a host’s response to above ground herbivory. Below ground herbivores then cause nitrogen to be allocated away from the roots and into the stem [45]. Because red milkweed beetles that are above and below ground facilitate each others’ survival through their reallocation of resources, it is possible that above ground herbivore abundance is representative of below ground herbivore abundance. In this case, our measures of above ground tissue loss sufficiently represent the effects of below ground herbivores that decrease root quality. Still, direct effects of all below ground herbivores on bud production should be quantified to establish tighter relationships between below ground herbivory and bud production.

A comparative greenhouse experiment on how subterranean herbivores affect
below ground biomass indicated that an increase in larvae caused a decrease in root growth [34]. This follows our predictions given the negative correlation that we found between herbivory and per capita bud emergence. Nonetheless, experiments from this publication should be repeated with slight modifications (or different methods of analysis should be done if the data are available) because there were some flaws in the experimental design. These shortfalls were mainly that some estimates were not scaled to make proper comparisons (such as absolute change in biomass or additional number of ramets in a patch). We could distinguish between the effects of above and below ground herbivores on bud production in the model by either including them as separate covariates with separate effects. Another approach could be to identify which variables that are currently in the model may predict the abundance of below ground herbivores and accordingly incorporate their effects on vital rates or per capita estimates. By distinguishing between above and below ground herbivores in the model, we would be able to gain better insight into how different pieces of the herbivore community vary in their effect on the population growth rate.

4.4 Model Limitations and Future Considerations

We know that herbivory can have an effect on every vital rate and that there is spatial variation in the vital rates that it has an effect on. Although we have evidence that herbivory decreases pod production, this effect is only detected in one site (YTB). This could be a simple matter of spatial variation in the response, whether by means of population genetic differences and/or environmental factors. It may also be a matter of the design of the study. It is possible that there is a loose connection between the damage recorded in June and responses recorded later in the year. It needs to be verified whether herbivore damage at one point in the growing season is representative of the damage later on. If the intensity of damage is variable throughout the season, then it may be that the damage recorded in June makes weak predictions for estimates taken during another census. For example, the effect of herbivory on pod production, a rate that is recorded in September, is only detected at one site (YTB). Perhaps if herbivore damage were recorded in September as well, regression analysis using that variable would detect an effect on the vital rate. In a similar respect, we relate herbivory in one year to the number of buds that emerge the following year. This is a year long time step, and a ramet’s bud production mechanism might be affected by herbivore damage later in the season. With this in mind, another study on common milkweed [17] did detect an effect of herbivory on pod production when they quantified it in June. The effect of herbivory in this study was much stronger than ours, as it reduced about 33%
of pod production when levels of herbivory were kept at 10% or less of leaf tissue removed. It is therefore possible that the effect of herbivory on pod production varies by site and is not a matter of when the census is taken.

With the exception of YTB, herbivory scores at each site range from none to low or intermediate levels of damage, with most individuals having scores between 0 and 1. While we do detect herbivory effects at our sites, there may still exist limitations in the regressions to predict the vital rate responses of an individual to a higher intensity of tissue damage. Regressions were not analyzed from higher levels of damage because of limitations in the natural level of herbivory observed in the field. Responses in the clonal pathway may also be different for higher levels of herbivory. For example, there may exist response thresholds in bud production related to the extent of environmental stress that an individual is experiencing. To accommodate this shortcoming, future work could include experimental manipulation of vital rate response to tissue damage in the greenhouse or experimental manipulation in the field. Experimental manipulation of herbivore damage in the field must be considered with caution, though, because artificial interference may not elicit the natural responses that are intended for study [23, 14]. Another approach may be to create one population model for all of the sites together. In such a case the data would be analyzed using a linear mixed effect model with random effect by site. This would allow us to detect whether herbivory has a greater effect than analysis of our sites alone indicated, and it would allow us to ask questions about Virginia’s common milkweed populations as a whole.

4.5 Insights from population growth rate on measures of fitness

Studies that investigate common milkweed response to environmental stress relate a ramet’s fitness to its increased growth, biomass, or pod production [23, 1, 5, 17, 50]. Pod production is positively correlated with size, and size (biomass) is positively correlated with bud production. Since bud production (clonal pathway) largely determines the population growth rate, a measure of fitness [22], biomass and pod production are seemingly reasonable measures of fitness when studying ecological interactions of common milkweed. Yet in light of the previous discussion of resource re-allocation upon herbivory, total biomass needs to be decoupled into above and below ground biomass measurements (or root-to-shoot ratio) to effectively infer what the effects will be on clonal versus sexual reproduction (where increase in clonal reproduction increases fitness). Still, this may not be generalized to all populations. A study showed that higher latitude populations exhibited a
higher root-to-shoot ratio than lower latitude plants, likely because they have a shorter growing season and rely more on clonal propagation than sexual reproduction to withstand winter stress and have earlier phenology [49]. While this may be true, our study populations in Virginia are closer to this study’s lower latitude populations in North Carolina, and still exhibit low effectiveness in sexually reproductive capacity. More research is required to decouple investment in clonal versus sexual reproduction and how seed quality, probability of seed establishment, and probability of seedling emergence might confer fitness benefits that are or are not related to above or below ground biomass.
Chapter 5
Conclusion

Our study links the effects of individual response of common milkweed to herbivore damage to population dynamics at five separate sites. We found that herbivores almost always decrease an individual’s probability of within-season survival, probability of producing inflorescences, growth rates within the season, and pod production. These effects vary spatially, which could be attributed to combinations of different factors: interactions with other environmental stresses, the composition of the herbivore community, or population genetic differences. The effects of herbivory on an individual’s response translates to a decrease in both sexual and asexual reproductive output. At its current estimates, the emergence of buds from pre-existing roots contributes most to the population growth rate. Although it was not included in our model, we have evidence that increasing herbivore damage is connected to decreasing bud emergence. More research is necessary to disentangle how above versus below ground herbivory elicits different responses regarding clonal propagation in common milkweed. Our research establishes the need to improve the current knowledge on the relationships between root herbivory and bud production, identifies how other environmental factors such as competition in combination with herbivory drive common milkweed population dynamics, and provides a general framework to test for meaningful effects of herbivory on common milkweed responses.
References


REFERENCES


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Appendix A

Appendix

A.1 Proof for MPM with IPM elements

S refers to Seedlings
B refers to Emergent Buds
$S \rightarrow S$: Seedling contributions to Seedlings
$S \rightarrow B$: Seedling contributions to Emergent Buds
$B \rightarrow S$: Emergent Bud contributions to Seedlings
$B \rightarrow B$: Emergent Bud contributions to Emergent Buds

A.1.1 Seedling Recruitment

Theorem A.1.1. \( P_t(z_h', z_h, z_\omega) \) is a kernel that projects the population from June (t) to September \((t + \tau)\). \( F_{S_{t+\tau}}(z_h', z_\omega) \) is a kernel that predicts pod production in September. Let \( B_t \) and \( S_t \) represent the number of emergent buds and seedlings in the population at time \( t \), respectively. Then:

\[
S_{t+1} = aS_t + bB_t
\]

\[
a = p_{es} \nu \int \int \int F_{S_{t+\tau}}(z_h', z_\omega) P_t(z_h', z_h, z_\omega) \rho_\omega(z_\omega) \rho_S(z_h) dz_h dz_h' dz_\omega
\]

\[
b = p_{es} \nu \int \int \int F_{S_{t+\tau}}(z_h', z_\omega) P_t(z_h', z_h, z_\omega) \rho_\omega(z_\omega) \rho_B(z_h) dz_h dz_h' dz_\omega
\]
Proof.

\[ n_t^S(z_h, z_\omega) \equiv S_t \rho_S(z_h) \rho_\omega(z_\omega) \]

\[ n_{\tau}^{S\to S}(z_h', z_\omega) = \int P_t(z_h', z_h, z_\omega) n_t^S(z_h) \rho_\omega(z_\omega) dz_h \]

\[ = \int P_t(z_h', z_h, z_\omega) S_t \rho_S(z_h) \rho_\omega(z_\omega) dz_h \]

\[ = S_t \int P_t(z_h', z_h, z_\omega) \rho_S(z_h) \rho_\omega(z_\omega) dz_h \]

\[ n_{t+1}^{S\to S}(z_h') = P_{es} \nu \int \int F_{S_{t+\tau}}(z_h', z_\omega) \rho_S(z_h) n_{\tau}^{S\to S}(z_h', z_\omega) dz_h' dz_\omega \]

\[ = P_{es} \nu S_t \int \int F_{S_{t+\tau}}(z_h', z_\omega) \rho_S(z_h) [\int P_t(z_h', z_h, z_\omega) S_t \rho_S(z_h) \rho_\omega(z_\omega) dz_h' dz_\omega] \]

\[ = P_{es} \nu \rho_S(z_h) \int \int F_{S_{t+\tau}}(z_h', z_\omega) \rho_S(z_h) [\int P_t(z_h', z_h, z_\omega) S_t \rho_S(z_h) \rho_\omega(z_\omega) dz_h' dz_\omega] \]

\[ S_{t+1}^{S\to S} = \int n_{t+1}^{S\to S}(z_h') dz_h' \]

\[ = a S_t \]

where \( a = P_{es} \nu \int \int F_{S_{t+\tau}}(z_h', z_\omega) P_t(z_h', z_h, z_\omega) \rho_\omega(z_\omega) \rho_S(z_h) dz_h' dz_\omega \)

since \( \int \rho_S(z_h') dz_h' = 1 \)
The same logic applies to $B_t \to S_{t+1}$

$$n_t^B(z_h, z_\omega) = B_t \rho_B(z_h) \rho_\omega(z_\omega)$$

$$n_t^B(z_h, z_\omega) = \int P_t'(z'_h, z_h, z_\omega) n_t^B(z_h) \rho_\omega(z_\omega) dz_h$$

$$= \int P_t'(z'_h, z_h, z_\omega) B_t \rho_S(z_h) \rho_\omega(z_\omega) dz_h$$

$$= B_t \int P_t'(z'_h, z_h, z_\omega) \rho_B(z_h) \rho_\omega(z_\omega) dz_h$$

$$n_t^B(z_h, z_\omega) = P_{es\nu} \int F_{S_{t+1}}(z'_h, z_\omega) \rho_B(z_h) n_t^B(z'_h, z_\omega) dz_h dz_\omega$$

$$= P_{es\nu} B_t \int F_{S_{t+1}}(z'_h, z_\omega) \rho_B(z_h) dz_h$$

$$= P_{es\nu} \rho_B(z_h) \int F_{S_{t+1}}(z'_h, z_\omega) dz_h$$

$$= P_{es\nu} \rho_B(z_h) \rho_B(z_h) dz_h$$

$$S_{t+1} = n_t^B(z_h, z_\omega) dz_h$$

$$= a B_t$$

where $b = p_{es\nu} \int F_{S_{t+1}}(z'_h, z_\omega) P_t(z'_h, z_h, z_\omega) \rho_\omega(z_\omega) \rho_B(z_h) dz_h dz'_h dz_\omega$

since $\int \rho_B(z'_h) dz'_h = 1$

Together, $S_{t+1} = a S_t + b B_t$

\[ \square \]

### A.1.2 Emergent Bud Recruitment

**Theorem A.1.2.** $F_{B_t}$ is the kernel that projects clonal recruits in June $(t + 1)$ from the population in June $(t)$. Let $B_t$ and $S_t$ represent the number of emergent buds and seedlings in the population at time $t$, respectively. Then:

$$B_{t+1} = p_{em}(B_t + S_t)$$
Proof.

\[ n_{t+1}^B \equiv B_{t+1} \rho_B(z_h^n) + S_{t+1} \rho_S(z_h^n) \]

\[ n_t^S(z_h) \equiv S_t \rho_S(z_h) \]

\[ S_t = \int n_t^S(z_h) dz_h \] and

\[ B_{t+1}^{\rightarrow B} = \int n_{t+1}^{\rightarrow B}(z_h^n) dz_h^n \]

where \( n_{t+1}^{\rightarrow B}(z_h^n) = \int F_{Bt}(z_h^n) n_t^S(z_h) dz_h \), by (6)

\[ = p_{em} \rho_B(z_h^n) \int n_t^S(z_h) dz_h, \] by (7)

\[ = p_{em} \rho_B(z_h^n) S_t \]

thus \( B_{t+1}^{\rightarrow B} = \int p_{em} \rho_B(z_h^n) S_t dz_h^n \)

\[ = p_{em} S_t \int \rho_B(z_h^n) dz_h^n \text{ where } \int \rho_B(z_h^n) dz_h^n = 1 \]

\[ B_{t+1}^{\rightarrow B} = p_{em} S_t \]

The same logic follows for the number of emergent buds at time \( t \) that contribute to the number of emergent buds at time \( t + 1 \):

\[ n_t^B(z_h) \equiv B_t \rho_B(z_h) \]

\[ B_t = \int n_t^B(z_h) dz_h \] and

\[ B_{t+1}^{\rightarrow B} = \int n_{t+1}^{\rightarrow B}(z_h^n) dz_h^n \]

where \( n_{t+1}^{\rightarrow B}(z_h^n) = \int F_{Bt}(z_h^n) n_t^B(z_h) dz_h \), by (6)

\[ = p_{em} \rho_B(z_h^n) \int n_t^B(z_h) dz_h, \] by (7)

\[ = p_{em} \rho_B(z_h^n) B_t \]

thus \( B_{t+1}^{\rightarrow B} = \int p_{em} \rho_B(z_h^n) B_t dz_h^n \)

\[ = p_{em} B_t \int \rho_B(z_h^n) dz_h^n \text{ where } \int \rho_B(z_h^n) dz_h^n = 1 \]

\[ B_{t+1}^{\rightarrow B} = p_{em} B_t \]
The contributions from seedlings and emergent buds at time $t$ to the number of emergent buds at time $t + 1$ sums the total number of emergent buds at time $t + 1$.

$$B_{t+1} = B_{t+1}^{B} + B_{t+1}^{S} = p_{em}B_{t} + p_{em}S_{t} = p_{em}(B_{t} + S_{t})$$

\[\square\]

A.1.3 Model Selection for Survival Function

The survival, growth, and pod production functions compose the sexual reproduction pathway and they depend on each other for their progression. In June, an individual has a certain probability of flowering. Given that it flowers in June, it has a certain probability of surviving to September and growing to a certain size. The number of pods that this flowering individual produces either depends on height in June or height in September. Because of the condition that an individual flowers, survival, growth, and pod production functions should be parameterized by regressions using the data collected of just flowering individuals. This was done to parameterize growth and pod production. Survival was instead still parameterized from all of the stems. Similar to the probability of flowering, larger individuals in June had a higher probability of surviving the growing season. When individuals that did not flower were excluded from regressions of survival on size, larger individuals that mostly survived remained in the data set, and the analysis could not produce estimates that were significant from zero. This is likely because it could not detect the dependence of survival on size given the lack of variation in size and survival response. Therefore, survival was regressed against size for all stems, including those that did and did not flower. This is demonstrated in Figure A.1 below.
Figure A.1: Survival for individuals of a given size that did and did not flower.