

Plant Toxicity, Adaptive Herbivory, and Plant Community Dynamics

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ABSTRACT

We model effects of interspecific plant competition, herbivory, and a plant's toxic defenses against herbivores on vegetation dynamics. The model predicts that, when a generalist herbivore feeds in the absence of plant toxins, adaptive foraging generally increases the probability of coexistence of plant species populations, because the herbivore switches more of its effort to whichever plant species is more common and accessible. In contrast, toxin-determined selective herbivory can drive plant succession toward dominance by the more toxic species, as previously documented in boreal forests and prairies. When the toxin concentrations in different plant species are similar, but species have different toxins with nonadditive effects,

herbivores tend to diversify foraging efforts to avoid high intakes of any one toxin. This diversification leads the herbivore to focus more feeding on the less common plant species. Thus, uncommon plants may experience compensatory mortality from herbivory, reducing local species diversity. The compensatory effect of herbivory may inhibit the invasion of other plant species that are more palatable or have different toxins. These predictions were tested and confirmed in the Alaskan boreal forest.

Key words: functional response; plant competition; herbivory; plant toxin; adaptive foraging; mammalian herbivores; boreal forest.

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Author Contributions: This study was conceived by JPB. The original version of the toxin-determined functional response model (TDFRM) was developed by JPB, ZF, and RKS. DLD made a significant modification of the model by incorporating the choice parameter for herbivore's ingestion and provided suggestions for analysis and simulations. DLD, ZF, and RL performed most of the model analysis and simulations. JPB and KK designed field experiments, collected and analyzed data (for parameter estimation). FSC has provided many insightful comments that played an important role in making sure that the model analysis is guided by biological questions and facts. All authors contributed to writing of the manuscript.

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INTRODUCTION

Herbivores can affect plant populations and plant communities by altering plant competition, facilitating the invasion of extant plant communities by new plants (invaders), and modifying the trajectory (rate and outcome) of plant community succession (Crawley 1983; Harper 1977). In response to herbivory, plants have evolved a variety of defenses, including chemicals that are toxic to herbivores (reviewed by Stamp 2003; Dearing and others 2005). In counter-response, many herbivores have evolved offensive tactics to counteract plant defenses (reviewed by Karban and Agrawal 2002; Provenza and others 2003; Villalba and Provenza 2005). General models that incorporate the inter-

play of plant defense and herbivore offense are needed if ecologists are to understand the consequences of these complex plant-herbivore interactions for community dynamics. As an initial step, we consider here how herbivory and plant competition interact to influence community dynamics when plants either do or do not differ in toxicity and when the herbivore adopts a foraging strategy consisting of a variable effort that changes through time to maximize fitness.

The impetus for model development arose from the observation that previous use of the Holling (1959) functional response model for mammalian herbivory did not include plant toxicity (for example, Caughley 1976; Lundberg 1988; Owen-Smith 2002). However, many plant species contain toxins that control the maximum per capita daily biomass intake by herbivores (reviewed by Dearing and others 2005; MacLean and Duncan 2006). To examine the effect of toxicity on feeding rates, we constructed a toxin-determined functional response model (acronym TDFRM) (Feng and others 2008; Li and others 2006; Liu and others 2008). The model adds one parameter to the Holling Type 2 response; G , which describes the negative effect that plant toxins have on the herbivore population's time-dependent per capita intake of plant biomass. This addition allows one to dynamically model how toxin-determined herbivory affects forage consumption and ultimately vegetation dynamics. Thus, the model is a potential tool for resource managers working in ecosystems containing toxic plants.

The TDFRM was initially applied to the 'one herbivore species (P)-one plant species (N)' case (Feng and others 2008). The model in this case has the biomass equations

$$\frac{dN}{dt} = rN \left(1 - \frac{N}{K} \right) - C(N)P, \quad (1a)$$

$$\frac{dP}{dt} = BC(N)P - dP, \quad (1b)$$

where

$$C(N) = f(N) \left(1 - \frac{f(N)}{4G} \right), \quad (1c)$$

and $f(N)$ is the Holling Type 2 functional response, that is,

$$f(N) = \frac{e\sigma N}{1 + h e \sigma N}. \quad (1d)$$

In equations (1a, b), r is the intrinsic growth rate of the plant, K is the plant's carrying capacity,

and B is the conversion constant (herbivore biomass per unit of plant). The toxin-determined functional response, $C(N)$, contains, as one factor, the traditional Holling Type 2 functional response (equation 1d), representing ingestion per unit time derived on the basis of search by the consumer moving at a constant speed through space with randomly distributed prey of biomass density N . The parameter e in equation (1d) is the resource encounter rate, which depends on the movement velocity of the consumer and its radius of detection of food items. The parameter σ ($0 < \sigma < 1$) is the fraction of food biomass encountered that the herbivore ingests, whereas h is the handling time per unit biomass, which incorporates the time required for the digestive tract to handle the item. The second part of the toxin-determined functional response, that is, $1 - (f(N)/4G)$ accounts for the negative effect of toxin, where $G = M/T$. The parameter M is a measure of the maximum amount of toxicant per unit time that the herbivore can tolerate (g toxin kg^{-1} herbivore biomass day^{-3}), and T is the amount of toxin per unit plant biomass (g toxin kg^{-1} plant biomass). Hence, the units of G are $[(\text{g toxin kg}^{-1} \text{ herbivore biomass tolerated day}^{-1}) (\text{g toxin kg}^{-1} \text{ plant biomass})^{-1}]$. G decreases when the concentration of toxin tolerated by the herbivore decreases (high toxicity) or the concentration of toxin in the plant biomass increases. Therefore, the smaller the value of G , the larger the effect the toxin has on intake. If we assume that M is the same for all individuals within an herbivore population, the variation in G will result from the different values of T of the plant species encountered. The factor 4 is just a multiplier that simplifies the form of the peak value of $C(N)$ as a function of N .

When the effect of toxin is very low, that is $1/G < 1$, it can be shown that $C(N)$ is approximately

$$C(N) \cong \frac{e\sigma N}{1 + h e \sigma N + \frac{e\sigma N}{4G}} \quad (2)$$

What this means is that in the limit of low toxin concentration, the effect of toxin is simply equivalent to an increase in handling time by the herbivore; that is, toxin slows down ingestion further than handling time alone. This type of simple increase in handling time would also occur from nontoxic digestion-slowing plant material, such as fiber. However, $C(N)$ was designed so that for smaller values of G (greater toxicity), there can be more severe consequences for an herbivore population. If the fraction of encountered resources ingested, σ , is

assumed to be constant, then over a range of values of G , $1/(4h) < G < 1/(2h)$, $C(N)$ is unimodal, declining to an asymptote after reaching a peak,

$$C(N_m) = G, \text{ at } N_m, \text{ where } N_m = \frac{G}{e\sigma(1/2 - hG)} \quad (3)$$

This decrease in $C(N)$ for $N > N_m$ is the result of the increasing negative impact of plant toxicant, which affects the herbivore physiologically by decreasing its ingestion rate and possibly leading to death. The parameter h , where $1/h$ is the maximum consumption rate by the herbivore in the absence of toxin, influences the effect of G . The larger $1/h$ is, the greater effect that a certain level G will have on population dynamics.

Feng and others (2008) considered two possible alternative behavioral responses by an herbivore, which can lead to significantly different outcomes when the density of its food plant is high and the toxin concentration is also high. In the first alternative, the herbivore is assumed unable to control its ingestion fraction, ϕ , even if it is ingesting large amounts of toxin. For this case, the herbivores can potentially overeat and suffer deleterious effects from the toxin. Population extinction is possible in this case. We suspect that unregulated intake of toxic plant biomass is rare, but its occurrence is evidenced by the recent death of several hundred elk (*Cervus elaphus*) feeding on the foliose lichen (*Xanthoparmelia chlorochroa*) containing the toxin (+)-usnic acid (Dailey and others 2008). In the second case considered by Feng and others (2008), the herbivore is able to control the fraction of resources encountered that it ingests, $\phi(t)$, to reduce chronic poisoning. In this case, the model predicts that the herbivore population avoids the threat of extinction from poisoning, although population size is reduced. Adaptively minimizing poisoning is the usual mammal foraging behavior (Boyle and others 2005; Provenza and others 2003).

The results of Feng and others (2008) indicate that the TDFRM can reasonably describe a variety of possible dynamics of an herbivore population feeding on a single plant species. However, it is more typical for mammalian herbivores to feed on a variety of plants (Freeland and Janzen 1974; Westoby 1974, 1978). Our goal here is to investigate the effect of toxicants when the herbivore acts as a generalist that has a choice of plant species to feed on. In this case, ϕ is generalized to ϕ_i to describe the relative effort exerted on feeding on each particular plant species i . Two situations will be considered. One situation is when the toxins in the plants are

distinct enough to be noninteractive, so that the amount of one toxin that an herbivore can tolerate is not affected by the presence of the other toxin. The other extreme situation is when the negative effects of the two toxins are perfectly additive in their negative effects. Intermediate levels of interaction can occur as well, but will not be modeled here, and nutrient-toxin interactions will also be ignored.

Our general objective is to first examine how the dynamics of two competing plant species is affected when herbivory is added but there are no anti-herbivore toxins, and next how the dynamics is affected when different concentrations of either noninteracting or additive toxins are included in the two different plant species. A body of empirical research shows that consumers often feed in a positive frequency-dependent manner, such that the more abundant prey species is disproportionately represented in the diet (for example, Gendron 1987). Such positive frequency-dependent feeding may result naturally from the forming of a search image for the more abundant prey type, which may be adaptive (Holling 1959). Theoretical studies indicate that such behavior by the consumer can contribute to species coexistence (for example, Comins and Hassell 1976; Levin and Segel 1982). Thus, from such studies we expect that, when herbivores forage adaptively on plants, they should tend to promote coexistence through a certain amount of switching to more abundant prey, thus preventing the extinction of inferior plant competitors. However, these studies did not take into account the effect of plant toxins. We will examine the effects that plant toxins have on this traditional view of the role of herbivores on plant communities. We organize our study around some questions and hypotheses concerning how plant toxins change plant-herbivore dynamics:

1. When toxins are present in different concentrations in the competing plants, we hypothesize (Hypothesis 1) the effects of toxins can cause a reversal in the behavior of the herbivore, toward concentrating foraging effort on the rarer plant species (thus acting in a depensatory manner), when either the toxins are noninteracting or the rarer species is less toxic.
2. In successional processes in the presence of herbivores, we hypothesize (Hypothesis 2) that less defended fast-growing species will generally be replaced over time by more defended slower-growing species.
3. We hypothesize (Hypothesis 3) that the presence of a resident species that is highly toxin-defended will generally inhibit the invasion of a

new species unless the invading species is itself highly toxic.

We test these hypotheses through the TDFRM model extended to multiple plant species, and through information from empirical studies that have been done in the past.

METHODS: MODEL DESCRIPTION

To cover the case where there is one herbivore and two plant types, equations (1a-d) can be extended. In the first version of the extended equations below it is assumed that the two plant species have different, noninteracting toxins, and that the plants compete intra- and inter-specifically:

$$\frac{dN_1}{dt} = r_1 N_1 \left(1 - \frac{(N_1 + c_{12}N_2)}{K_1} \right) - \frac{e_1 \sigma_1 N_1 P}{1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2} \left(1 - \frac{e_1 \sigma_1 N_1}{4G_1(1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2)} \right) \quad (4a)$$

$$\frac{dN_2}{dt} = r_2 N_2 \left(1 - \frac{(N_2 + c_{21}N_1)}{K_2} \right) - \frac{e_2 \sigma_2 N_2 P}{1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2} \left(1 - \frac{e_2 \sigma_2 N_2}{4G_2(1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2)} \right) \quad (4b)$$

$$\frac{dP}{dt} = \frac{B_1 e_1 \sigma_1 N_1 P}{1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2} \left(1 - \frac{e_1 \sigma_1 N_1}{4G_1(1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2)} \right) + \frac{B_2 e_2 \sigma_2 N_2 P}{1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2} \left(1 - \frac{e_2 \sigma_2 N_2}{4G_2(1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2)} \right) - m_p P. \quad (4c)$$

In these equations N_1 , N_2 , and P are the biomass densities of edible components of plant species 1 and 2, and of the herbivore, respectively. The parameters and their units are as follows (without the subscript $i = 1$ and 2, which represents plant species i): G is a measure of the effect the toxin has on the herbivore, defined earlier as having the units $[(\text{g toxin kg}^{-1} \text{ herbivore biomass tolerated day}^{-1}) (\text{g toxin kg}^{-1} \text{ plant biomass})^{-1}]$; r is the intrinsic growth rate of plant (day^{-1}); K is the carrying capacity of the plant (kg m^{-2}); c_{ij} is the coefficient of competitive effect of plant species j on i ; B is the conversion constant (herbivore biomass

per unit of plant biomass consumed, kg kg^{-1}); and m_p is the per capita death rate of herbivore unrelated to plant toxicity (day^{-1}).

The toxin-determined functional response in the model (equations 4a-c) is

$$C_i(N_1, N_2) = \frac{e_i \sigma_i N_i}{1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2} \left(1 - \frac{e_i \sigma_i N_i}{4G_i(1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2)} \right), \quad i = 1, 2. \quad (5)$$

The functional response $C_i(N_1, N_2)$ starts with the Holling Type 2 functional response (first factor in equation 5) for foraging on two species, which we denote by $h(N_1, N_2)$, and is extended to include the effects of each of the two plant species containing its own toxin. Small values of G represent a strong effect of toxicity on herbivore foraging. The second factor in equation (5), which accounts for the negative effect of toxin, can be written more concisely as

$$1 - \frac{f_i(N_1, N_2)}{4G_i}$$

In the limit that $1/G_i \sim 1$, the effect of the toxicant can be viewed as purely a slowdown in feeding rate (see equation 2). When $1/G_i > 1$, on the other hand, the TDFRM can represent a more serious deterioration of the herbivore's ability to feed or survive. In the extreme alternative to noninteractive toxins, that is, the case in which the toxins are the same, or are additive, a different toxin-determined functional response is used, denoted by $C_i(N_1, N_2)'$ where

$$\tilde{C}_i(N_1, N_2) = \frac{e_i \sigma_i N_i}{1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2} \left(1 - \frac{(e_1 \sigma_1 N_1 / G_1) + (e_2 \sigma_2 N_2 / G_2)}{4(1 + h_1 e_1 \sigma_1 N_1 + h_2 e_2 \sigma_2 N_2)} \right), \quad i = 1, 2. \quad (6)$$

In this case, ingestion of a toxin from one plant species negatively affects the feeding rate on both species.

The parameters c_{11} and c_{12} are the fractions of effort or time applied to foraging for the respective types of plant species. This differs from the 'one plant species' case, where c_i represented the fraction of encountered items of prey biomass that are ingested. We assume that the herbivore feeds selectively and optimally at each instant (obtaining the maximum possible energy intake rate) by varying $\sigma_1(t)$ and $\sigma_2(t)$ through time, depending on

which selection levels optimize the herbivores immediate growth rate, as long as $o_1(t) + o_2(t) < 1$. Therefore, the herbivore can change its feeding strategy rapidly by adjusting its foraging to maximize its overall fitness. It is unlikely that perfect adaptive foraging is possible in real-world situations. It may be difficult to discern differences in prey, and various contingencies may interfere with optimal selection. However, some degree of adaptive foraging probably occurs in herbivore foraging. The ability of an herbivore to change its feeding strategy through time may have important implications for plant competition, plant succession, community composition, and the capability of a new species to invade. We will use the model (equations 4a-c) and its variation (that is, with $C_i(N_1, N_2)$ being replaced by $C_i(N_1, N_2)$, as given by (6)) to address the questions raised at the end of the "Introduction" section.

METHODS: EMPIRICAL DATA

Although the present study did not involve the collection of new data, empirical observations from a number of earlier investigations, including those of some of the present authors, such as the Alaska Bonanza Creek Long-Term Ecological Research Project (BNZ LTER), are used to test the model results. Some experimental studies allow testing of the hypothesis that the compensatory effect of foraging that favors species coexistence in the absence of toxins may be reversed when toxins are present. One such study is an experimental study by Bergvall and others (2006) with fallow deer (*Dama dama*). This study used food pellets treated with hydrolyzable tannin to simulate plants with lower versus higher defenses. This experiment can address the question of whether a less chemically defended plant would be consumed to a greater extent if it is grown at low density among another abundant species than if it is grown among its own kind.

Feeding experiments and herbivore exclusion experiments are also relevant to the hypothesis that herbivory can lead succession toward more heavily defended and often slower-growing species. Such experimental studies in the boreal forest have been performed in the winter on mammalian herbivores such as snowshoe hare (*Lepus americanus*) and moose (*Alces alces*) to determine whether the per capita daily intake of browse (woody twigs, bark) is related to successional state (feeding experiments; see Bryant 1981; Bryant and others 1983a) and if selective browsing can alter the rate and trajectory of forest succession (exclusion experiments; see

Pastor and Naiman 1992; Kielland and others 2006; Butler and others 2007). Similar information is available from the work of Clay in temperate grasslands (Clay and Holsa 1999; Clay 2001; Clay and others 2005).

RESULTS

Plant Species Competition Only

The outcome of the pure competition model (equations 4a-b, with $P = 0$) is well known; it is determined completely by the competition coefficients for the case when $K_1 = K_2$. If $c_{12} < 1 < c_{21}$, species 1 always eliminates species 2.

Addition of Herbivory to Plant Competition

Equations (4a-c), when the effect of toxins is set to zero ($1/G = 0$), reduce to well-known equations for two plant species and an herbivore with a Holling Type 2 functional response. We used this model to address the question of whether herbivores in the absence of plant toxins prevent the extinction of an inferior competitor. This model has been investigated for fixed values of relative foraging effort, o_1 and o_2 (see supplementary material-Appendix 1). The results depend on the parameter values. It can be shown that when $c_{21} \gg c_{12}$ (that is, species 1 is a much stronger competitor) and the consumption rate by the herbivore on species 1 is not too high, then it is likely that species 2 will still be excluded by species 1 even in the presence of the herbivore. But threshold values occur where sufficiently high feeding rates on the competitively superior species enable mutual coexistence or even exclusion of the superior competitor. Specifics for that situation are provided in supplementary material-Appendix 1, but here we focus on herbivores that can forage adaptively, by adjusting their feeding effort, $o_1(t)$ versus $o_2(t)$, to optimize instantaneous energy intake. In agreement with earlier modeling studies (for example, Murdoch and Oaten 1975), adaptive feeding can promote plant species coexistence (see, for example, the simulation in Figure 1, where species 2 can coexist with species 1 although the former is competitively inferior). Such complete switching of feeding effort in response to changes in relative abundance of plants would be impossible to achieve in the real world. However, even a small tendency toward frequency-dependent consumption will tend to protect the rarer species.

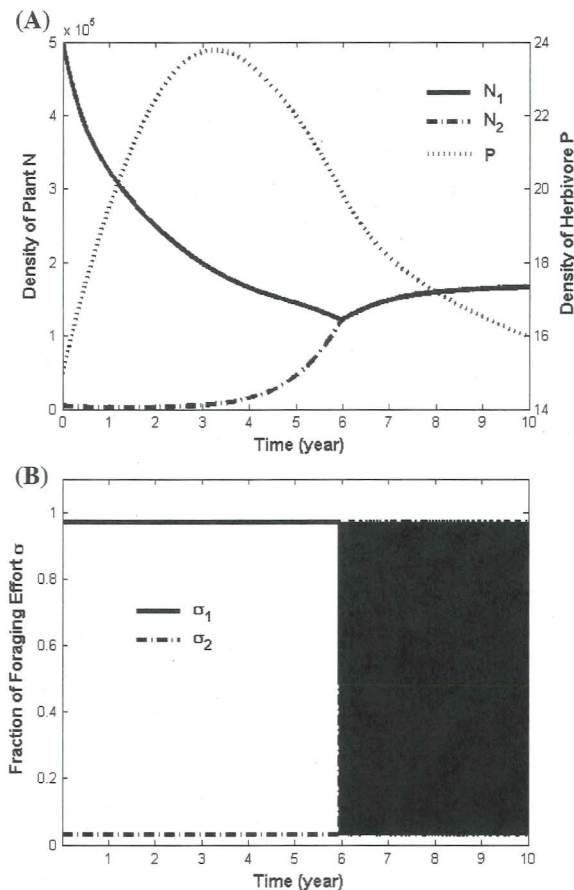


Figure 1. The Holling Type 2 response for the case of optimal feeding with $\sigma_1(t) + \sigma_2(t) \leq 1$. It shows that the two species coexist. We observe that in the beginning $\sigma_1 = 1$, $\sigma_2 = 0$ because the initial density of species 1 is higher and species 1 is decreasing. Once the density of species 2 exceeds that of species 1, the consumption constants switch to $\sigma_1 = 0$, $\sigma_2 = 1$, and the switches continue to occur (the switches are so frequent that it appears like a black area in the plot σ versus t). N_1 and N_2 refer to biomasses of edible twigs. Parameter values same as Figure 2, but without toxicity.

The Effects of Noninteractive Plant Toxins

Our Hypothesis 1 is that plant toxins can alter the herbivore-mediated community dynamics. Above we observed that, in the absence of toxin, the plant species that is in lower abundance will be favored by herbivory, as herbivores will concentrate their efforts on the more abundant plant species (positive frequency dependence). Some analytic results are again possible for the case of fixed relative efforts σ_1 and σ_2 (see Supplementary material-Appendix 1). But again here we focus on herbivores foraging adaptively ($\sigma_1(t)$ and $\sigma_2(t)$

change through time) in a community where plants contain toxins. The effects of the toxins are assumed not to be additive, so that the effect of the toxicant of a given species is negative only on the feeding on that species.

Assume that plant species 1 is the resident and species 2 is introduced in small numbers. Assume further that the competition coefficients of the two species are set equal; $c_{12} = c_{21} = 0.9$. Simulations of the TDFRM show that, when the herbivore is able to vary its foraging effort through time to maximize fitness, species 2, starting from a low density, cannot invade, if G_1 is sufficiently small (that is, the resident, species 1, is sufficiently toxic) (Figure 2). Here, although the initial ratio of the invader to resident is very small, $N_2/N_1 = 0.01$, the foraging effort remains greater on species 2 than on species 1. The population of species 2 is pushed rapidly toward extinction (Figure 2B). Notice that the relative feeding effort on species 2, $\sigma_2(t)$, remains high even as the population N_2 rapidly declines toward zero (Figure 2C). This is in contrast to the model of adaptive herbivory with no plant toxin effects, in which the herbivore switches back and forth to maintain its effort on the more common plant species (Figure 1). This result supports our Hypothesis 1 that, when plant toxicity is considered, under some conditions adaptive herbivory can result in compensatory effects, favoring exclusion of the less abundant species.

As in the case where no plant toxins are involved, it is unlikely that herbivores in the real world would be able to adjust their feeding perfectly in response to toxins to maximize fitness, but some tendencies in this direction are revealed by empirical research. The experimental study by Bergvall and others (2006) with fallow deer (*Dama dama*) supports this model prediction. The authors demonstrated that when the less defended plant (represented by low tannin pellets) was associated with more defended plants (represented by high tannin pellets), the less defended plant was eaten more than if it was growing among its own kind. Even if a less abundant invader is relatively toxic, switching from a highly toxic resident to the invader is still predicted to occur if the invader's toxins and those of the more abundant resident are nonadditive. The result is also consistent with Heady's (1964) conclusion: "Observations indicate that some species (for example, the toxic monoterpene containing big sage *Artemisia tridentata*; Stoddart and Smith 1955) are grazed heavily (by livestock) when they occur in small quantities throughout a 'better' forage, whereas in dense stands the use is light." This toxin-mediated compensatory effect imposes relatively

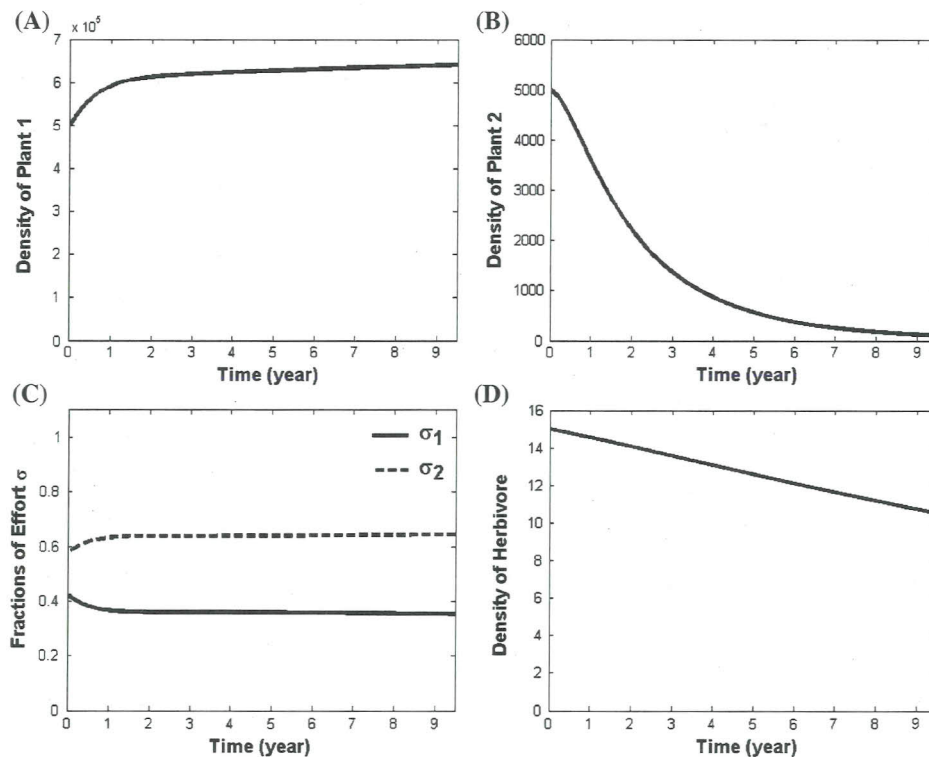


Figure 2. Simulation results of the TDFRM for an adaptively foraging herbivore when the resident species (1) is more toxic than the invading species (2): $G_1 = 35$, $G_2 = 60$. Initial plant densities are $N_{1,0} = 10^5$ and $N_{2,0} = 10^3$. Species 2 is eliminated. Parameter values used for this figure are: $c_{12} = 0.9$, $c_{21} = 0.9$, $r_1 = r_2 = 0.007$, $K_1 = K_2 = 7 \times 10^5$, $B_1 = B_2 = 3.4 \times 10^{-5}$, $e_1 = e_2 = 0.0007$, $h_1 = h_2 = 0.008$, $m_p = 0.0013$. N_1 and N_2 refer to biomasses of edible twigs.

stronger herbivory on the less abundant invader, thereby reducing its survival, and in the process, reducing plant species diversity. We have named this compensatory process "the rare plant effect." Subsequent studies on white-tailed deer (*Odocoileus virginianus*; Augustine and others 1998) and moose (*Alces alces*; Edenius and others 2002) support the prediction.

The results shown in Figure 2 change substantially, however, if species 1 is not too toxic (G_1 is not too small). In such cases, avoidance of toxin in species 1 is not so critically important for the herbivore, and its efforts can be more strongly determined by the relative population abundances of the two plant species. Some specific scenarios are considered here, each with $c_{12} = c_{21}$. In one scenario, $G_1 = 50$ and $G_2 = 35$ (more toxic) (Figure 3). In this case, species 2 is able to both invade and achieve higher densities than species 1, by virtue of its higher toxicity. In fact, species 1 goes to extinction.

In a further scenario, two species were allowed to start growing from very small values, simulating invasion of a bare plot of land, and it was assumed that $r_2 < r_1$ and $G_2 < G_1$, so that species 1 grows faster but species 2 is more toxic. The simulations (Figure 4) show that species 1 initially increased to a high value instantaneously on the scale of the figure, but was eventually pushed to lower values by the slow growing, heavily defended species 2. Al-

though in this case both plant species persisted, it can be shown that in other cases species 1 will go to extinction. This result corroborates our Hypothesis 2 that the less defended fast-growing species can be replaced over time by more defended slower-growing species.

Natural history observations and experiments on winter foraging of snowshoe hare and moose tend to confirm these results. The per capita daily intake rates of browse (woody twigs, bark) of both species are largely determined by browse toxins (reviewed by Bryant and Kuropat 1980; Bryant and others 1991). In general, these herbivores feed selectively on rapidly growing early successional species (usually deciduous), show moderate avoidance of mid-successional species, and show strongest avoidance of late successional evergreens such as spruce (*Picea* spp.) and Labrador tea (*Ledum* spp.). Accordingly, consumption of the mid-successional species such as the alders (*Alnus* spp) is much lower than the intake of early successional species, for example, willow (*Salix* spp.) and slightly higher than the intake of late successional evergreens (Bryant 1981; Bryant and others 1983a, b; Kielland and Bryant 1998; Kielland and others 2006; Butler and others 2007). These observations led to the resource availability hypothesis of plant defense-slow-growing species are more defended than fast-growing species (Bryant and others

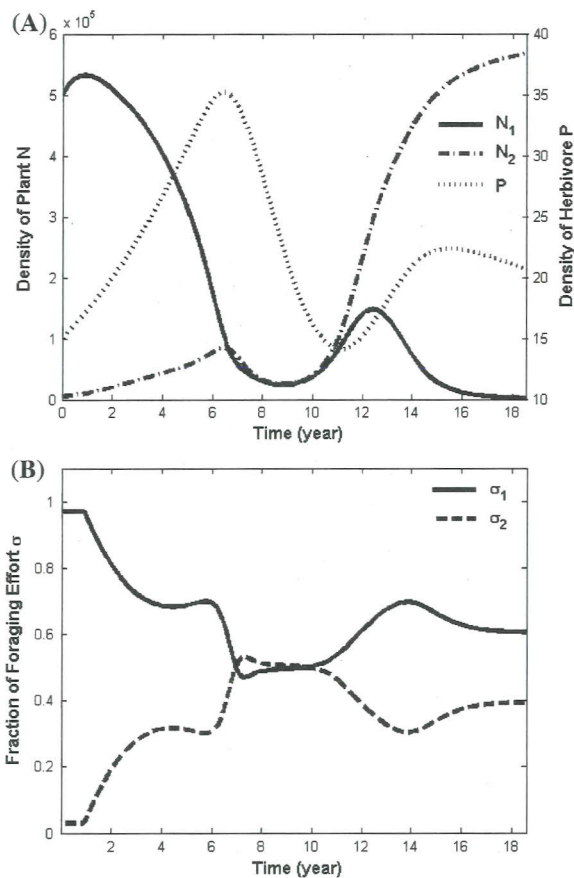


Figure 3. Simulation results for the TDFRM for an adaptively foraging herbivore when the competition coefficients are equal $c_{12} = c_{21}$ and the resident species 1 is less toxic ($G_1 = 50$) than in the scenario in Figure 2, and other parameter values being the same except where stated. $G_1 = 50$, $G_2 = 35$, $c_{12} = 0.9$, $c_{21} = 0.9$. N_1 and N_2 refer to biomasses of edible twigs, and should be multiplied by 5 in the figure.

1983a; Coley and others 1985). Kielland and others (2006) summarize the browsing research, which focused on the willow-alder transition occurring early in primary succession on the Tanana River Floodplain. On the floodplain, recently deposited sediment is initially colonized by willow and balsam poplar with willow being most abundant (Chapin and others 2006). Alder (*A. tenuifolia*) subsequently invades the willow thickets, leading to a willow-alder transition. Increasing dominance of alder also initiates a collapse in wildlife's food supply because alder and the later successional evergreens that replace willow are so toxic that they are hardly eaten (Bryant and Kuropat 1980; Bryant and others 1983a, b).

Further model studies on species invasion reveal additional aspects of toxin-influenced dynamics. In

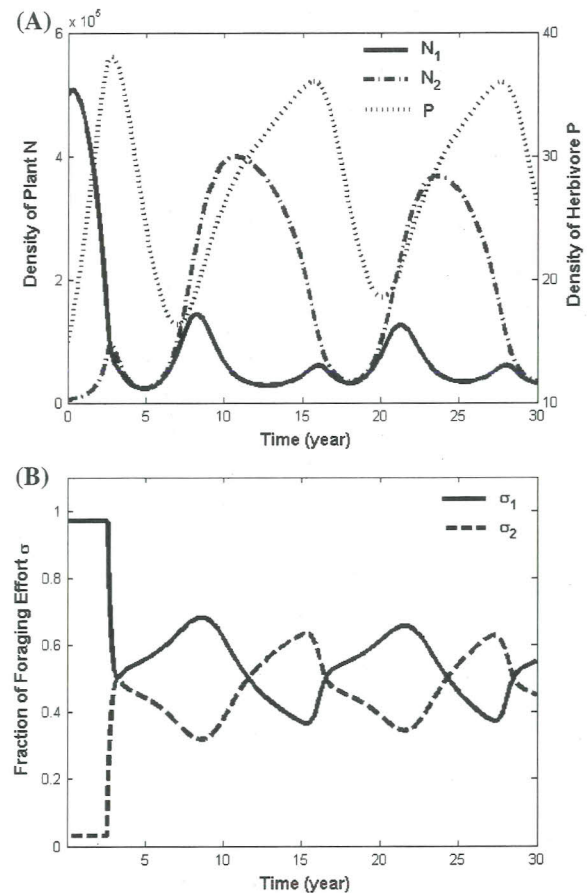


Figure 4. Simulation results for the TDFRM for an adaptively foraging herbivore when the competition coefficients are equal $c_{12} = c_{21}$. In this case, species 1 is assumed to have a higher growth rate; $r_1 = 0.009 > r_2 = 0.007$, but species 2 is more toxic; $G_1 = 110 > G_2 = 40$. Other parameters values are the same.

the next scenario, $G_1 = 50$ and $G_2 = 60$, the slightly higher toxicity of species 1 keeps it superior in biomass to species 2 (Figure 5). In this case, the herbivore feeding and the relative abundance of the plant species interact in a way that produces oscillations of all species on a time scale of several years. (The biomass values in these plots refer to edible twigs only, not whole plants.) Note that the changes in $\sigma_1(t)$ versus $\sigma_2(t)$ through time are not large. Recall in the case of herbivory without toxins, the herbivore shifts completely to expending foraging effort on only one species or the other; whereas, here there is only a modest change in foraging effort between the plant species. In the final scenario, the competition coefficients are reduced, $c_{12} = c_{21} = 0.5$, and the system stabilizes with both plant species persisting, with the more toxic species (species 1) being more abundant (Figure 6).

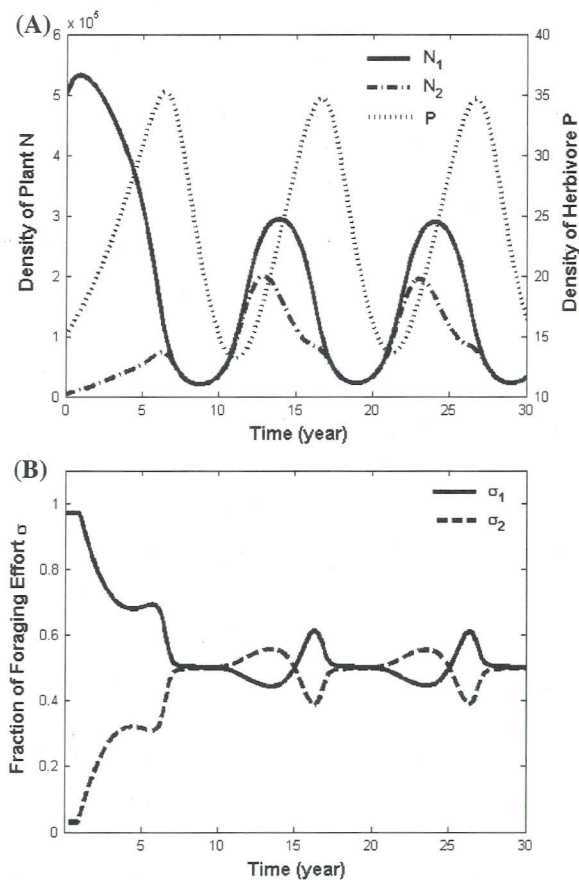


Figure 5. Simulation results for the TDFRM for an adaptively foraging herbivore when the competition coefficients are equal $c_{12} = c_{21}$ and the resident species 1 is less toxic ($G_1 = 50$) than in the scenario in Figure 2, and other parameter values being the same except where stated. $G_1 = 50$, $G_2 = 60$, $c_{12} = 0.9$, $c_{21} = 0.9$. N_1 and N_2 refer to biomasses of edible twigs.

The effects of relative toxicity levels of the two species on invasion can be summarized. Invasion is more likely when G_1 increases (resident species becomes less toxic) (Figure 7A). There is a threshold level G_1^* for species 1 below which invasion by species 2 is impossible. Simulations (not shown here) also suggest that the threshold value of G_1^* decreases with increasing initial density of species 2 ($N_{2,0}$). That is, the greater the initial density of the invader, the more toxic must be species 1 if invasion is not to occur (Figure 7B), as larger numbers reduce the probability of depensatory herbivory on the invader. These model results support our Hypothesis 3 that the occurrence of toxin in the resident species and the relative amounts of toxins in the resident and invader can determine the ability of an introduced species to successfully invade in a system with herbivory.

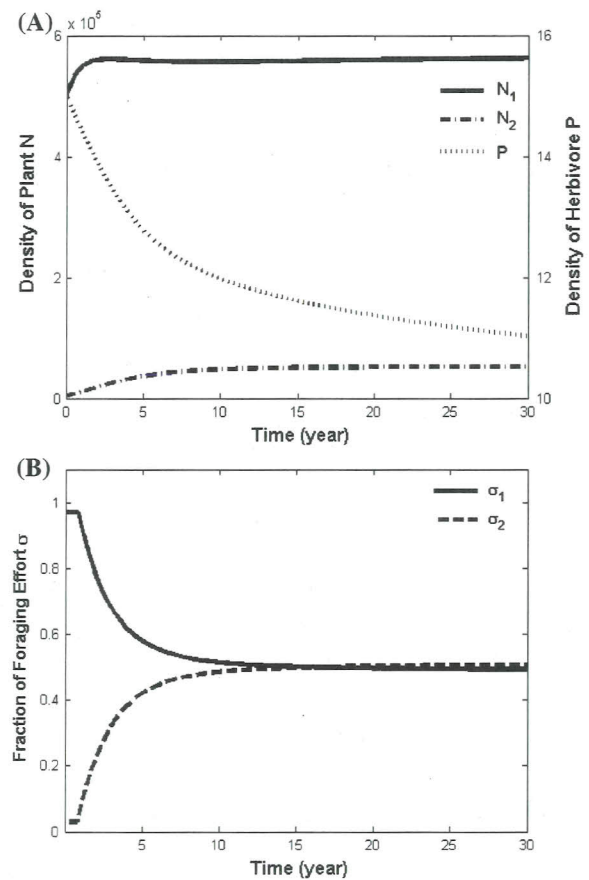


Figure 6. Simulation results for the TDFRM for an adaptively foraging herbivore when the competition coefficients are equal $c_{12} = c_{21}$ and the resident species 1 is less toxic ($G_1 = 50$) than in the scenario in Figure 2, and other parameter values being the same except where stated. $G_1 = 50$, $G_2 = 60$, $c_{12} = 0.5$, $c_{21} = 0.5$, $m_p = 0.0018$. N_1 and N_2 refer to biomasses of edible twigs.

The results of studies on competition at the BNZ LTER tend to confirm the above model results. In 1987 (year 12 in Figure 8), the BNZ LTER established seven pairs of exclosures and browsed control plots in the mid-colonization stage (sensu Chapin and others 2006) of floodplain succession (experimental design in supplementary material-Appendix 2) to test the hypothesis that selective browsing by moose and hare contributes to the willow-alder transition (Kielland and others 2006) (for details, see supplementary material-Appendix 2). We parameterized the TDFRM for the Tanana Floodplain system and computed the expected ratio of alder/willow from 1987 to 2007 for both the browsed control plots and the unbrowsed exclosure plots. (For discussion of measurement of alder/willow ratio, see supplementary material-Appendix 2). We also used the Holling Type 2 model to simulate browsing's effect

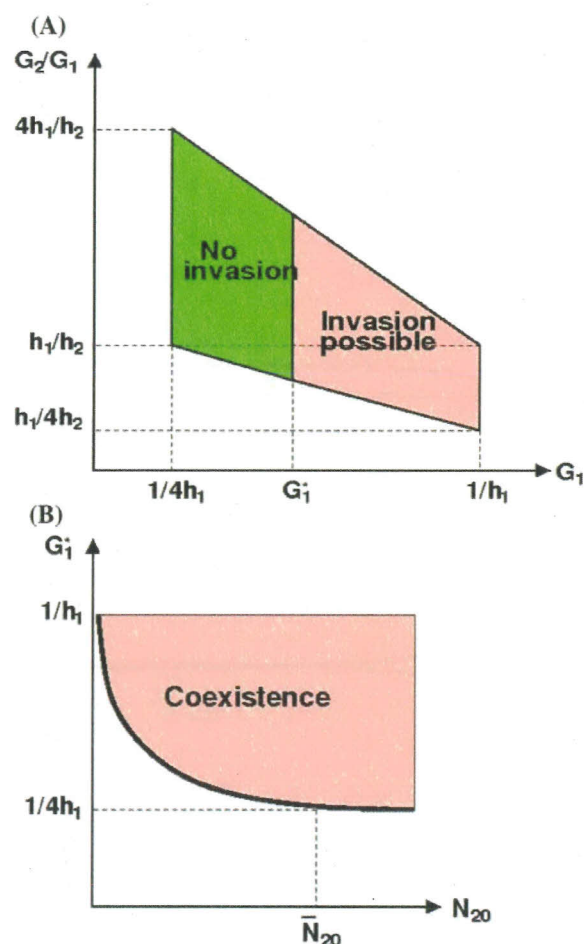


Figure 7. (A) Range of toxicity values over which an invader (N_2) can succeed when the resident species (N_1) is at equilibrium with the herbivore. Simulation results (with an adaptively foraging herbivore) show that the toxicity level of the resident species G_1 can affect the invasion. G_1 is bounded by $1/4 h_1$ and $1/h_1$ (Feng and others 2008), and the colored regions denote the biologically feasible ranges of G_1 and G_1/G_2 . For a given set of parameter values, there is a threshold toxin level G_1^* such that invasion is possible only for $G_1 > G_1^*$. Moreover, for each given $G_1 > G_1^*$, there is a range for the ratio G_2/G_1 in which invasion is possible (the upper and lower bounds in the red region). The threshold value of G_1^* increases with the growth rate r_1 . (B) The threshold value G_1^* decreases with the initial density of species 2 ($N_{2,0}$). (Refer colour figure online).

on the ratio. The simulation results and their fit to the measured ratios of alder/willow are presented in Figure 8. The TDFRM (thick solid line in Figure 8) very accurately simulated changes in community composition, whereas the H2FRM could not be fit reasonably to the data. In the presence of herbivores, the more toxic alder continued increasing in abun-

dance relative to the less toxic willow. In the absence of herbivores (exclosure), the two plant species approached an equilibrium at a lower alder:willow ratio. In other words, herbivores speeded the elimination of the less toxic early successional willow.

We hypothesize that toxin-determined selective herbivory by mammals has resulted in invasion of more toxic plants during plant succession in taiga forest (Bryant and Chapin 1986; Kielland and Bryant 1998; Kielland and others 2006; Butler and others 2007), the southern boreal forest (Pastor and Naiman 1992), and temperate grasslands (Clay and Holo 1999; Clay 2001; Clay and others 2005). Our theoretical study and the supportive results from the literature indicate that when toxic plants are involved, the distribution of levels of toxicity of neighboring plants profoundly affects plant community dynamics. TDFRM further predicts that the herbivore's foraging strategy can have a dramatic effect on the competitive outcome for plant communities containing toxins. Thus, TDFRM provides a framework for predicting the interactive effects of plant defense and herbivore offense on community trajectories.

The Effects of Additive Plant Toxins

To examine the alternative limiting case, in which the toxins of the two plant species are perfectly additive, the functional response given by equation (6) is substituted into equations (4a-c). In this case, the ingestion of a toxin from one plant species negatively affects the feeding rate on both species. We present only a few results to indicate the general trend that inclusion of an interactive effect of the toxins has on the dynamics. Two specific cases that were simulated above are now reconsidered using equation (6). In the first case, using the noninteracting function (equation 5), the resident species excluded the rare, less toxic invading species (Figure 2). When equation (6) is substituted, however, the two species are able to coexist (see Figure A3 in supplementary material-Appendix 3). A second case is that shown in Figure 3 for the noninteracting toxins, in which the invading species is more toxic and is able to exclude the resident. When this simulation is repeated using equation (6) for additive toxins, the two plant species are able to coexist (see Figure A4 in supplementary material-Appendix 3). Thus, the additive toxins do not show the same effect of noninteracting toxins in reversing the effect herbivore compensation on the competing plants. Instead, the situation is tilted toward herbivore-facilitated coexistence.

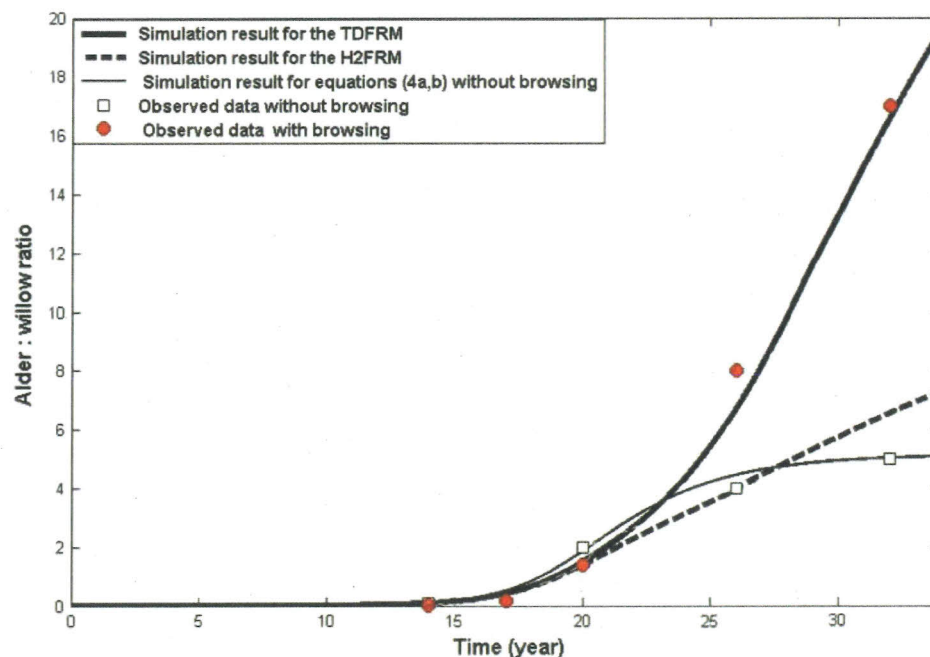


Figure 8. Comparison of simulations in which fits of the toxin-determined functional response (TDFRM) and the Holling Type 2 functional response (H2FRM) are made to data on primary succession in the Alaska Bonanza Creek LTER. Simulations of both functional responses are fit to measured ratio alder/willow in plots with versus without browsing by hares and moose. See text for explanation. The parameter values used are: $r_1 = 0.0016$, $r_2 = 0.0017$, $K_1 = 50000$, $K_2 = 140000$, $c_{21} = 0.17$, $c_{12} = 0.2$, $e_1 = e_2 = 0.0001$, $h_1 = 0.01$, $h_2 = 0.06$, $G_1 = 98$, $G_2 = 5$, $B_1 = 0.00034$, $B_2 = 0.0003$, $d = 1/(3.5 \times 365)$.

DISCUSSION

We have used particular functional responses to analyze the effect of herbivores on the outcome of plant competition in both the absence and presence of toxins and when the herbivore forages adaptively to maximize fitness. In this section we discuss general theoretical conclusions of the results, based on examples that were parameterized to depict a range of possible outcomes to encourage ecologists to generate hypotheses about specific plant-herbivore systems.

As is well known, the model of pure plant-plant competition without herbivory predicts that for equal K , the better competitor always wins. However, adaptively foraging herbivores are able to counteract the effects of competition. By switching to whichever plant species is more easily found, herbivores can in many cases prevent the rarer plant's local extinction, thereby facilitating coexistence (Figure 1). This should occur as long as the rare species is not at too much of a competitive disadvantage. Of course, it is unlikely that any herbivore will have the instantaneous knowledge of slight differences in the relative availability of two forage species that is assumed in the model. An

herbivore's choices will be contingent on its hunger status and what resources are immediately available, as reflected in some models of mammalian herbivores in heterogeneous environments (for example, Hobbs and others 2003; Spalinger and Hobbs 1992). But even if frequency-dependent switching lags somewhat behind the current relative plant biomasses, it may increase the viability of the rarer species. This result seems reasonable, because herbivory that is focused on the abundant species would release the rare species from competition.

When plants contain toxins, the nature of herbivore effects on plant community dynamics can change dramatically. High toxin levels in the resident species 1 (low G_1) can impede successful establishment of invading species 2, because the herbivore must limit its intake of species 1, and will tend to feed in a negative frequency-dependent way by switching some of its effort to species 2, despite the low abundance of the species 2 population (Figure 2). The $1 - (f_1(N_1, N_2)/4G_1)$ (actor in the species 1 portion of the functional response) depresses the feeding rate on species 1, resulting in a larger relative effort ($o_2(t)$ versus $o_1(t)$) on species 2. Species 2 may to some extent overcome

the indirect negative effect (through increased herbivory) of the toxins of its competitor if it is toxic itself (Figs. 3, 4, 5, and 6). It can also be shown that in many cases species 2 may also overcome this compensatory effect if it both is introduced in high enough numbers (supplementary material-Appendix 1) and is sufficiently toxic. The ratio G_2/G_1 (relative toxicity) is an important determinant of invasion success for an inferior competitor (Figure 7A), and the probability of successful invasion increases with the initial population size of the introduced species.

The TDFRM makes three general predictions. The first is that under conditions of substantial adaptive herbivory, high toxin levels in a plant strongly favor its dominance, in terms of higher biomass levels in the community, over species with lower levels of toxicity. This can be true even though herbivory is often considered to occur in a positive frequency-dependent way; that is, focused on the species that is more abundant to a greater extent than would be expected from random encounters. Instead, when an abundant species is highly toxic, the fraction of effort that is directed at a less abundant or rare species, $o_2(t)$, may remain disproportionately high even though the population biomass of that species is declining rapidly toward zero. The second general prediction is that, in a community of fast-growing species with low toxicity and slow-growing species with high toxicity, and in which there is also significant herbivory, the fast-growing species will dominate in biomass initially, but will be outcompeted by the high-toxicity species in the long run. The third general prediction is that, when the resident species of a community are highly toxic to herbivores, invasion by new species with low toxicity or nonadditive toxins will be difficult, but highly toxic species may be able to invade. All of these predictions appear to be confirmed by empirical data. The model makes other predictions as well. The model simulations suggest that oscillatory behavior is common (Figure 4). Strong competition coefficients, c_{ij} , appear to contribute to such oscillations.

When the toxins are additive rather than interactive, that is, when the functional response of equation (6) is substituted for equation (5), then the effects of toxicity are weakened. In the two examples that were considered, where the noninteracting toxins led to a reversal of the herbivore compensation effect, such that the less toxic species was excluded, in the case of additive toxins this did not occur. This is a fairly intuitive result. The toxin of each species decreased feeding on both species. This did not decrease the advantage to the herbi-

vore of feeding on the less toxic plant species. However, the higher toxicity of one plant species has the effect of reducing the feeding rate on both plant species. This works to the advantage of a species that is pushed to low levels. Under those circumstances the herbivore must feed heavily on the more toxic species, which has the effect of weakening the overall food intake of the herbivore and protecting the rare, less toxic species as a result.

We have considered only certain ideal cases in this paper. In particular, we assumed that the herbivore is able to forage adaptively to optimize intake at all times, as ratios of biomasses of the plant species changed. This should be relaxed in future work to foraging that is adaptive, but suboptimal. We have also assumed only nonadditive or completely additive toxins, whereas in most cases there may be some intermediate level of synergism. We have not explicitly addressed nutrient-toxin/energy interactions. However, we recognize their importance, and furthermore, suggest that consideration of the interactions can lead to further experimental tests of the model. Such interactions can ameliorate a target plant's toxicity (for example, Provenza and others 2003; Villalba and Provenza 2005). The amelioration is operationally equivalent to increasing the G value of the target toxic plant. In essence, when the nutrient/energy spectrum of the vegetation in which a toxic plant grows reduces the ratio of dietary toxicants:nutrients/energy below that which the animal cannot exceed, the toxic plant's G value effectively increases. The increase will reduce the target plant's potential to invade the vegetation, or if the target plant is rare, accelerate its removal from the vegetation. Conversely, if the background vegetation decreases the toxic target plant's G value (effectively increases the plant's critical ratio toxicants:nutrients), the effect should be opposite in direction. The predictions could be tested by introducing at different densities a toxic plant into vegetation that either ameliorates its toxicity or enhances its toxicity.

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