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Modeling the Impact of Plant Toxicity on Plant–Herbivore Dynamics

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Numerous empirical studies over the past two decades have documented substantial effects of plant toxins on diet choice and feeding behavior of herbivores, but analytical models have failed thus far to incorporate toxin-mediated effects of browsing on plant population dynamics. We study a mathematical model that incorporates plant toxicity in the functional response of plant–herbivore interactions. The model also includes a Lotka–Volterra type competition between plants. The model exhibits a rich variety of complex dynamics including Hopf bifurcation and period-doubling bifurcations. Differences in dynamical behavior stem from interspecific differences in plant biology and strategies for growth and defense as well as variation in responses of herbivores to toxins. Analyses suggest that for realistic parameter values, herbivores are capable of promoting coexistence of plant species by ameliorating competitive effects and hence enhancing biodiversity.

KEY WORDS: Mathematical model; bifurcation; plant-herbivore dynamics; functional response; plant toxicity.

1. INTRODUCTION

Three groups of chemicals interact to control mammalian herbivory. They are nutrients (nitrogen, phosphorous, carbohydrates, etc.), digestion inhibitors (fiber, tannins), and toxins [30,31]. Nutrients increase forage intake because they are required by herbivores and often are limiting in the

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food supply [33]. Digestion inhibitors reduce herbivory by slowing the rate of digestion [26,30]. Toxins reduce herbivory by satiating the herbivore's detoxification system, i.e., the herbivore either stops eating a poisonous food or dies. Poisoning is uncommon because consumption of a toxic food usually stops before lethal poisoning occurs.

Over the past two decades, chemically mediated interactions between plants and herbivores have been the focus of intensive research in ecology, evolutionary biology and resource management (for reviews, see [21,25,27,31]. Although most of this research has emphasized insect–plant interactions [16], studies of plant–mammal interactions have been the most cited (1995 Science Citation Index (ISI) Most Cited Papers—[6,13]) and provided much of the foundation of current plant defense theory, for example, the growth rate hypothesis [5,6,13] and the carbon/nutrient balance hypothesis [5]. Unfortunately, prior models have not explicitly incorporated toxicity effects on the dynamics of mammalian herbivores and plants. Here, we use results from research on plant–mammal interactions as the empirical basis for developing a model that extends the scope of plant defense theory by incorporating chemically mediated effects of browsing on vegetation dynamics.

Holling [19,20] proposed the concept of a functional response of a predator to prey abundance. In brief, a functional response is the instantaneous change in the rate of intake of prey by a predator in response to prey abundance. The functional response is characterized by a monotonic increase in prey consumption as prey biomass increases up to the biomass at which the predator's ability to eat more prey becomes satiated. Thereafter, prey consumption is constant irrespective of further increase in prey abundance. In most cases the cause of satiation is believed to be the amount of time it takes the herbivore to mechanically handle plant biomass (bite, chew, ruminate) [1,2,22,23,28]. Alternatively, satiation could be induced by toxins with amelioration by dietary nutrients [31]. In this paper, we model a toxin-dependent functional response to explore how chemically mediated mammalian herbivory can affect vegetation dynamics.

This article is concerned with ecosystem consequences of chemically mediated mammalian herbivory. A functional response model [19,20] of plant–herbivore interactions is built. Unlike all previous functional response models of plant-mammal interactions (e.g., [2,22,23,28], this model explicitly incorporates satiation by toxins.

2. THE MODEL

In the case of plant–herbivore interactions, mechanical handling is not the only plant trait constraining the rate of biomass consumption.

The diversity and concentrations of toxic plant defenses [18] and the mix of nutrients [32,33] in plant tissue are often more important determinants of consumption rate than mechanical handling (see reviews by [5,7,8,10,25,31]). This is because the rate of intake of plant biomass no longer increases after satiation by a specific toxin, irrespective of the abundance of plant biomass containing the satiating toxin. Thus, a functional response in a plant–herbivore system also can be caused by the mix of toxins and nutrients in plant tissue [7,8,10,31].

2.1. Model Formulation

We first derive a functional response which incorporates toxin satiation when there is a single plant. Consider the case when there is no toxin. Let N denote the number of units (or briefly, number) of plant twigs available, T_s denote the searching time, and e denote the encounter rate per unit of twigs. Then the total number of twigs encountered in time T_s is $T_s eN$. If h denotes the time a herbivore spends handling (time taken to bite, chew, and process) one unit of twigs, then the total handling time is $T_h = hT_s eN$. Then, traditionally, the consumption rate has been defined as

$$E = \frac{T_s eN}{T_s + T_h} = \frac{eN}{1 + heN} =: f(N). \tag{1}$$

To investigate the impact of plant toxicity on the herbivore–plant interactions, in this paper, we call E the *effective* encounter rate. The actual (or effective) consumption rate should be lower than E due to the toxicity.

From the meaning of h we see that $1/h$ represents the maximum number of twigs a herbivore may consume per unit of time in the absence of toxicity ($1/h$ is in fact the asymptote of the function $f(N)$ as $N \to \infty$). However, when toxin is present the actual consumption slows as the number of twigs encountered (and consumed) increases. Consequently, the maximum amount of toxin-containing twigs a herbivore can consume per unit time, denoted by G, is smaller than $1/h$. This suggests that the toxin-adjusted consumption rate, denoted by $\mathcal{C}(N)$, should have a lower asymptote or a smaller maximum value than that of $E(N)$ (or $f(N)$). To derive a such function, notice that the ratio $\mathcal{C}/E$ should be a decreasing function of E with its value close to 1 for small E and close to zero when E approaches some threshold value. One simple function that can be used to reflect this relationship between $\mathcal{C}$ and E is shown in Figure 1 and can be described as:

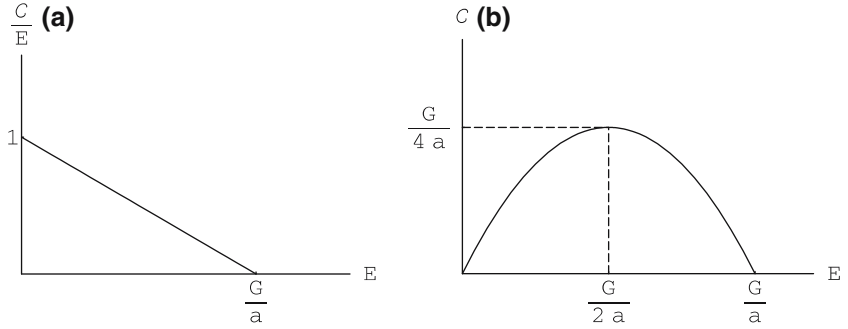

Figure 1. (a) The ratio $\frac{\mathcal{C}}{E}$ vs. the effective encounter rate; (b) the consumption rate vs. the effective encounter rate.

$$\frac{\mathcal{C}}{E} = 1 - \frac{\alpha E}{G}, \tag{2}$$

where α is a scaling parameter which is chosen such that the consumption rate $\mathcal{C}$ is bounded by G. Rewriting (2) to express $\mathcal{C}$ as a function of E we have

$$\mathcal{C}(E) = E\left(1 - \frac{\alpha E}{G}\right). \tag{3}$$

To determine the value for α, we notice that the function $\mathcal{C}(E)$ has a maximum $G/(4\alpha)$, which occurs at $E = G/(2\alpha)$ (see Figure. 1(b)). Thus, $\alpha = 1/4$. This value is used in the rest of this paper. We only are concerned about the case in which $C(E)$ is nonnegative, i.e., $E/(4G) \leqslant 1$, which is always true if $G > 1/(4h)$ as $E < 1/h$. Combining this with the fact that $G < 1/h$ we assume in the rest of the paper that

$$\frac{1}{4h} < G < \frac{1}{h}. \tag{4}$$

The consumption rate as a function of the plant abundance is obtained by substituting (1) into (3):

$$\begin{aligned}\mathcal{C}(N) &= f(N)\left(1 - \frac{1}{4G} f(N)\right) \\ &= \frac{eN}{1+eN}\left(1 - \frac{1}{4G}\frac{eN}{1+eN}\right).\end{aligned} \tag{5}$$

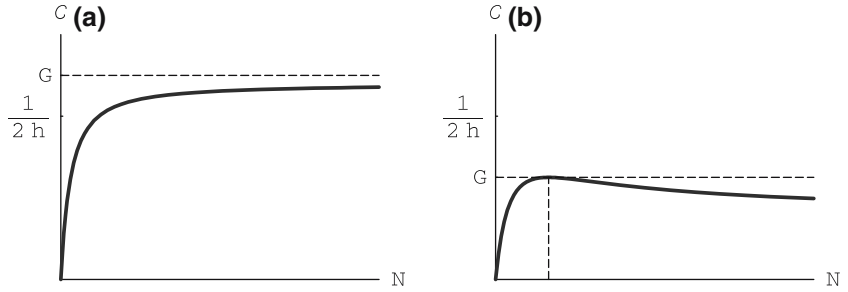

Figure 2. $\mathcal{C}$ vs. the plant abundance N. (a) $G \geqslant \frac{1}{2h}$; (b) $G < \frac{1}{2h}$.

The curve of the function is either monotonically increasing with the asymptote below G if $G \geqslant \frac{1}{2h}$ (see Figure 2(a)) or has a shape similar to the type IV functional response [14] with a maximum value below G and an asymptote less than its maximum value if $G < \frac{1}{2h}$ (see Fig. 2(b)).

Consider a landscape of n plant species and one herbivore population, in which each plant species may have a different level of toxicity and competitive ability for resources, and the herbivore's functional responses to plant abundance are dependent on plant toxicity. The toxin-dependent intake rate of plant i per herbivore can be derived in a similar way as for a single plant species. Let N_i denote the plant abundance of plant species i at time t; $\mathbf{N} = (N_1, N_2, \ldots, N_n)$, with the subscript i denoting the plant species; e_i the encounter rate; h_i the handling time; G_i the maximal amount of toxin of plant species i that can be eaten before an herbivore dies; $E_i = f_i(\mathbf{N})$ the effective encounter rate of species i. Then from (1) and (3) we get

$$E_i = f_i(\mathbf{N}) = \frac{e_i N_i}{1 + \sum_{i=1}^{n} h_i e_i N_i} \tag{6}$$

and

$$\mathcal{C}_i(\mathbf{N}) = f_i(\mathbf{N}) \left(1 - \frac{a_i f_i(\mathbf{N})}{G_i}\right), \quad i = 1, 2, \ldots, n, \tag{7}$$

where $a_i = 1/4$. Using the functional response given in (7), our model for the plant–herbivore interaction is given by the following system of differential equations:

$$\begin{aligned}\frac{dP}{dt} &= \sum_{j=1}^{n} B_j \mathcal{C}_j(\mathbf{N}) P - DP, \\ \frac{dN_i}{dt} &= r_i N_i \left(1 - \frac{N_i + \sum_{j=1, j\neq i}^{n} \beta_{ij} N_j}{K_i}\right) - \mathcal{C}_i(\mathbf{N}) P, \quad i = 1, 2, \ldots, n,\end{aligned} \tag{8}$$

where $P = P(t)$ denotes the number of herbivores at time t; B_i the conversion of consumed plant species i biomass into new herbivores, D the per capita rate of herbivore death due to causes unrelated to plant toxicity, r_i the plant growth rate under the best circumstances in the focal environment, i.e., no competition for resources by plants, β_{ij} the competition parameter which measures the competition intensity of species j against species i, and K_i is the carrying capacity. All parameters and their units are defined in Table 1. A more detailed discussion on the parameter values is given in Section 3 when the simulation results are presented. This model combines the approaches of Holling [19,20], Lundberg [22], Lundberg and Astrom [23].

2.2. Equilibria and their Stability

We now consider the case of two plant species for which $G_i = 1/(2h_i)$, i.e., the function $\mathcal{C}_i(\mathbf{N})$ is a monotonically increasing function with an asymptote G_i (Fig. 2a).

Table I. Parameters, units, and values used in the paper.

Parameter	Unit	Value
G_1	Max units of type 1 toxin-containing twigs a herbivore can consume/day	8 (Figs. 5–7)
G_2	Max units of type 2 toxin-containing twigs a herbivore can consume/day	80 (Figs. 5–7)
h_i $(i = 1, 2)$	Time for handling one unit of type i twigs in the absence of toxin	$1/h_i \leqslant 2G_i$ e.g., 1/32–1/400
e_i $(i = 1, 2)$	Rate of encounter/per unit of twigs	0.0001–0.0005
B_i $(i = 1, 2)$	Conversion constant (herbivore/unit of twigs)	0.00003–0.00006
r_i $(i = 1, 2)$	Max units of new twigs/twig/day	0.01–0.003
K_i $(i = 1, 2)$	Carrying capacity of type i plant	10^4–10^5
β_{ij} $(i, j = 1, 2)$	Competition coefficient	10^{-1}–10
a_i	Scaling constant	1/4
D	Per capita death rate of herbivore	0.00003–0.0002

2.2.1. Boundary Equilibria

The system (8) has six possible boundary equilibria (i.e., at least one component is zero):

$$\begin{array}{lll} E_0=(0,0,0), & E_1=(0,K_1,0), & E_2=(0,0,K_2), \\ \bar{E}=(0,\bar{N}_1,\bar{N}_2), & \tilde{E}=(\tilde{P},\tilde{N}_1,0), & \hat{E}=(\hat{P},0,\hat{N}_2), \end{array} \tag{9}$$

where

$$\begin{aligned} \bar{N}_1&=\frac{K_1-\beta_{12}K_2}{1-\beta_{12}\beta_{21}}, & \bar{N}_2&=\frac{K_2-\beta_{21}K_1}{1-\beta_{12}\beta_{21}}, \\ \tilde{P}&=r_1\tilde{N}_1\left(1-\frac{\tilde{N}_1}{K_1}\right)\frac{B_1}{D}, & \tilde{N}_1&=\frac{2G_1}{e_1}\left(\frac{1}{\sqrt{1-\frac{D}{B_1G_1}}}-1\right), \\ \hat{P}&=r_2\hat{N}_2\left(1-\frac{\hat{N}_2}{K_2}\right)\frac{B_2}{D}, & \hat{N}_2&=\frac{2G_2}{e_2}\left(\frac{1}{\sqrt{1-\frac{D}{B_2G_2}}}-1\right). \end{aligned} \tag{10}$$

The first four boundary equilibria have exactly the same properties as those present in the classical two-species Lotka–Volterra competition system. The existence and uniqueness of the equilibria $\tilde{E}$ and $\hat{E}$ are given in the following result.

Theorem 1. *Assume that* $G_i \geqslant \frac{1}{2h_i}$ *and that* $D < B_iG_i$, $i=1,2$. *The boundary equilibria* $\tilde{E}$ *and* $\hat{E}$ *given in* (9) *and* (10) *exist and are unique.*

Proof. From the mathematical symmetry of N_1 and N_2 it suffices to prove the result only for $\tilde{E}$. To solve for $\tilde{E}$ we set the right-hand side of Eq. (8) equal to zero and let $N_2=0$. Notice that $\tilde{N}_1$ can be solved by first solving for $x=x(N)$ where

$$x=:f_1(\tilde{N}_1,0)=\frac{e_1\tilde{N}_1}{1+h_1e_1\tilde{N}_1} \tag{11}$$

and x satisfies (from the P equation) the equation

$$B_1x\left(1-\frac{x}{4G_1}\right)=D, \tag{12}$$

which has two roots

$$x_\pm=2G_1\left(1\pm\sqrt{1-\frac{D}{B_1G_1}}\right).$$

Notice that $e_1N_1/(1+h_1e_1N_1) < 1/h_1 \leqslant 2G_1$. Hence, $x < 2G_1$, and consequently, x_+ cannot be a root of Eq. (12). Denote x_- by $\tilde{x}$, i.e.,

$$\tilde{x} = 2G_1\left(1 - \sqrt{1 - \frac{D}{B_1G_1}}\right). \tag{13}$$

Obviously $0 < \tilde{x} < 1$ as $D < B_1G_1$. Substituting (13) for x in (11) (and noticing that $2G_1 = 1/h_1$) we can solve for $\tilde{N}$ and get

$$\tilde{N}_1 = \frac{2G_1}{e_1}\left(\frac{1}{\sqrt{1 - \frac{D}{B_1G_1}}} - 1\right). \tag{14}$$

Using the N_1 equation in Eq. (8) and substituting $\frac{D}{B_1}$ for $\tilde{x}(1 - \frac{\tilde{x}}{4G_1})$ we can solve for $\tilde{P}$ and get

$$\tilde{P} = r_1\tilde{N}_1\left(1 - \frac{\tilde{N}_1}{K_1}\right)\frac{B_1}{D}.$$

Clearly $\tilde{P} > 0$ as $\tilde{N} < K_1$. This finishes the proof. □

We remark that the condition $D < B_1G_1$ has a clear biological interpretation. B_1G_1 and D are the per capita growth and death rates of the herbivore, respectively. Hence, for the herbivore population to survive the growth rate must exceed the death rate.

2.2.2. Stability of the Equilibria

The first four equilibria in (9) represent the steady states at which the herbivore is absent. The stability conditions for these equilibria are similar to those given in the standard Lotka–Volterra competition model provided that the death rate of the herbivore, D, exceeds the critical value, $\min\{D_1, D_2, D_3\}$, where

$$D_1 = B_1c_{10}, \qquad D_2 = B_2c_{20}, \qquad D_3 = B_1\bar{c}_1 + B_2\bar{c}_2 \tag{15}$$

and

$$\begin{aligned} c_{10} &= C_1(K_1, 0) = \frac{e_1K_1}{1+h_1e_1K_1}\left(1 - \frac{e_1K_1}{4G(1+h_1e_1K_1)}\right), \\ c_{20} &= C_2(0, K_2) = \frac{e_2K_2}{1+h_2e_2K_2}\left(1 - \frac{e_2K_2}{4G(1+h_2e_2K_2)}\right), \\ \bar{c}_i &= C_i(\bar{N}_1, \bar{N}_2), \quad i = 1, 2. \end{aligned} \tag{16}$$

More specifically, we have the following result.

Theorem 2. *Let* D_i, $i=1,2,3$ *be defined as in* (15). *Then*

(a) E_0 *is always unstable* (*a saddle*);

(b) E_1 *is locally asymptotically stable* (*l.a.s.*) *if and only if* $\dfrac{\beta_{21}K_1}{K_2}>1$ *and* $D>D_1$;

(c) E_2 *is l.a.s. if and only if* $\dfrac{\beta_{12}K_2}{K_1}>1$ *and* $D>D_2$; *and*

(d) $\bar{E}$ *is l.a.s. if and only if* $\dfrac{\beta_{21}K_1}{K_2}<1$, $\dfrac{\beta_{12}K_2}{K_1}<1$ *and* $D>D_3$.

The proof of Theorem 2 is straightforward. We observe form the above results that, if the death rate of herbivores is high (i.e., $D>D_i$, $i=1,2,3$), then the herbivore population will not persist due to the stability of the steady states at which the herbivore is absent.

We now consider the stability of $\tilde{E}$ and $\hat{E}$, both of which have a positive component of P. Depending on the focus of a study, one can choose different parameters to vary for the bifurcation analysis. For example, the following result uses the growth rates r_1 and r_2 as bifurcation parameters. Recall that the existence of $\tilde{E}$ and $\hat{E}$ requires the conditions $D<B_1G_1$ and $D<B_2G_2$ to be satisfied, respectively. □

Theorem 3. *Let* $D<B_1G_1$ *and* $K_1>1/(e_1h_1)$. *There exist positive constants* R_{21} *and* $D_1^*<B_1G_1$ *such that*

(i) $\tilde{E}$ *is l.a.s. if and only if* $r_2/r_1<R_{21}$ *and* $D>D_1^*$, *and unstable otherwise;*

(ii) *A supercritical Hopf bifurcation occurs at* $D=D_1^*$.

Proof. (i) The Jacobian matrix at $\tilde{E}$ is

$$\tilde{J}=\begin{pmatrix} 0 & a_{12} & * \\ a_{21} & a_{22} & * \\ 0 & 0 & a_{33} \end{pmatrix}, \tag{17}$$

where "$*$" represents a number that does not affect the eigenvalues of the matrix and

$$a_{12}=\frac{\tilde{P}B_1e_1}{(1+h_1e_1\tilde{N}_1)^2}\left(1-\frac{\tilde{x}}{2G_1}\right),$$
$$a_{21}=-\tilde{x}\left(1-\frac{\tilde{x}}{4G_1}\right),$$

$$a_{22} = \frac{r_1\tilde{N}_1}{(1+h_1e_1\tilde{N}_1)(2+h_1e_1\tilde{N}_1)} \left[-\frac{2}{K_1} + \left(\frac{3h_1e_1}{1+h_1e_1\tilde{N}_1} + \frac{h_1^2e_1^2\tilde{N}_1}{(1+h_1e_1\tilde{N}_1)^2}\right)\left(1-\frac{2\tilde{N}_1}{K_1}\right)\right], \quad (18)$$

$$a_{33} = r_2\left(1-\frac{\beta_{21}\tilde{N}_1}{K_2}\right) - \frac{\tilde{P}e_2}{1+h_1e_1\tilde{N}_1}$$

and $\tilde{x}$ is given in (13). Here, we have used the following equivalent expression:

$$\frac{1}{1+h_1e_1\tilde{N}_1} = 1-\frac{\tilde{x}}{2G_1} = \sqrt{1-\frac{D}{G_1B_1}}.$$

$\tilde{E}$ is locally asymptotically stable if all eigenvalues of $\tilde{J}$ have negative real parts. Clearly, a_{33} is an eigenvalue, and two other eigenvalues are the roots λ of the equation

$$\lambda^2 - a_{22}\lambda - a_{12}a_{21} = 0. \quad (19)$$

Thus, $\tilde{E}$ is l.a.s. iff the following conditions hold:

$$a_{12}a_{21} < 0, \qquad a_{22} < 0, \qquad a_{33} < 0.$$

Clearly $a_{12} > 0$ and $a_{21} < 0$ as $\tilde{x} < 2G_1$. Thus, $a_{12}a_{21} < 0$. Substituting $\tilde{P}$ (see (10)) into the inequality we see that $a_{33} < 0$ is equivalent to the condition

$$\frac{r_2}{r_1} < R_{21} =: \frac{B_1e_2\tilde{N}_1(1-\tilde{N}_1/K_1)}{D(1-\beta_{21}\tilde{N}_1/K_2)}\sqrt{1-\frac{D}{G_1B_1}}, \quad (20)$$

where $\tilde{N}_1$ is given in (10). Notice that the condition (20) is possible only if $\beta_{21}\tilde{N}_1/K_2 < 1$.

To determine the sign of a_{22}, we consider $a_{22} = a_{22}(\tilde{N}_1)$ as a function of $\tilde{N}_1$. Notice that, for any $\tilde{N}_1 > 0$, $a_{22} <$ (or $>$) 0 if and only if $F(\tilde{N}_1) <$ (or $>$) 0 where

$$F(\tilde{N}_1) = -\frac{2}{K_1} + \left(\frac{3h_1e_1}{1+h_1e_1\tilde{N}_1} + \frac{h_1^2e_1^2\tilde{N}_1}{(1+h_1e_1\tilde{N}_1)^2}\right)\left(1-\frac{2\tilde{N}_1}{K_1}\right) \quad (21)$$

is a continuous function of $\tilde{N}_1$ for $\tilde{N}_1 \in [0, K_1]$. Setting $F(\tilde{N}_1) = 0$ we get the quadratic equation for $\tilde{N}_1$:

$$A\tilde{N}_1^2 + B\tilde{N}_1 + C = 0$$

with

$$A = 10h_1^2e_1^2 > 0, \qquad C = 2 - 3h_1e_1K_1 < 0.$$

Hence, the equation $F(\tilde{N}_1) = 0$ has a unique solution $\tilde{N}_1^*$. From

$$F(0) = -\frac{2}{K_1} + 3h_1e_1 > 0$$

and

$$F(K_1) = -\frac{2}{K_1} - \frac{3h_1e_1}{1+h_1e_1K_1} - \frac{h_1^2e_1^2K_1}{(1+h_1e_1K_1)^2} < 0,$$

we know that $\tilde{N}_1^* \in (0, K_1)$. This shows that $a_{22}(\tilde{N}_1) = 0$ has a unique solution $\tilde{N}_1^* \in (0, K_1)$. Note that $\tilde{N}_1 = \tilde{N}_1(D)$ is a continuous increasing function of D with

$$\tilde{N}_1(0) = 0, \quad \tilde{N}_1(\tilde{D}) = K_1, \quad \text{where} \quad \tilde{D} = B_1G_1\left(1 - \frac{1}{(1+\frac{e_1K_1}{2G_1})^2}\right) < B_1G_1.$$

Thus, there exists a unique $D_1^* \in (0, \tilde{D})$ such that $\tilde{N}_1(D_1^*) = N_1^*$. It follows that $a_{22} > 0$ for $D < D_1^*$ and $a_{22} < 0$ for $D > D_1^*$, and $D_1^* < B_1G_1$. Therefore, $\tilde{E}$ is l.a.s. if $D > D_1^*$ and unstable if $D < D_1^*$.

For the proof of part (ii), we notice from the part (i) that $a_{22} = 0$ at D_1^*. Hence, Eq. (19) has a pair of pure imaginary roots for $D = D_1^*$, and complex roots for D near D_1^* with the real part $\Re\lambda = a_{22}$. Notice also that

$$\begin{aligned}\left.\frac{\partial \Re\lambda}{\partial D}\right|_{D=D_1^*} &= \frac{r_1\tilde{N}_1^*}{(1+h_1e_1\tilde{N}_1^*)(2+h_1e_1\tilde{N}_1^*)} \left.\frac{\partial F}{\partial \tilde{N}_1}\right|_{\tilde{N}_1=\tilde{N}_1^*} \left.\frac{\partial \tilde{N}_1}{\partial D}\right|_{D=D_1^*} \\ &= \frac{-r_1\tilde{N}_1^*}{(1+h_1e_1\tilde{N}_1^*)(2+h_1e_1\tilde{N}_1^*)} \times \left(\frac{2}{K_1}\left(\frac{3h_1e_1}{1+h_1e_1\tilde{N}_1^*} + \frac{h_1^2e_1^2\tilde{N}_1^*}{(1+h_1e_1\tilde{N}_1^*)^2}\right)\right. \\ &\quad \left. + \left(\frac{2h_1^2e_1^2}{(1+h_1e_1\tilde{N}_1^*)^2} + \frac{2h_1^3e_1^3\tilde{N}_1^*}{(1+h_1e_1\tilde{N}_1^*)^3}\right)\left(1 - \frac{2\tilde{N}_1^*}{K_1}\right)\right) \frac{\partial \tilde{N}_1}{\partial D}\Big|_{D=D_1^*} < 0\end{aligned}$$

since $\frac{\partial \tilde{N}_1}{\partial D} > 0$, $0 < \tilde{N}_1^* < K_1$ and $1 - 2\tilde{N}_1^*/K_1 > 0$ (see (21) and notice that $F(\tilde{N}_1^*) = 0$). From the part (i) we also know that $\tilde{E}$ is l.a.s. for $D > D_1^*$ and unstable for $D < D_1^*$. Therefore, a supercritical Hopf bifurcation occurs at $D = D_1^*$. This finishes the proof of Theorem 3. □

From the mathematical symmetry of the N_1 and N_2 equations the following result can be proved in the similar way as for Theorem 3.

Theorem 4. *Let $D < B_2G_2$ and $K_2 > 1/(e_2h_2)$. There exist positive constants R_{12} and $D_2^* < B_2G_2$ such that (i) $\hat{E}$ is l.a.s. if and only if $r_1/r_2 < R_{12}$ and $D > D_2^*$, and unstable otherwise; and (ii) a supercritical Hopf bifurcation occurs at $D = D_2^*$.*

The proof of Theorem 4 is very similar to that of Theorem 3. The constant R_{12} is

$$R_{12} = \frac{B_2 e_1 \hat{N}_2 (1 - \hat{N}_2/K_2)}{D(1 - \beta_{12}\hat{N}_2/K_1)} \sqrt{1 - \frac{D}{G_2 B_2}},$$

where $\hat{N}_2$ is given in (10). Notice that the condition $r_1/r_2 < R_{12}$ is possible only if $\beta_{12}\hat{N}_2/K_1 < 1$.

Let $D_4 = B_1G_1$ and $D_5 = B_2G_2$. If $D < \min_{1 \leqslant i \leqslant 5}\{D_i\}$, then all six boundary equilibria exist, and E_0, E_1, E_2, and $\bar{E}$ are unstable. If the condition $R_{12} < \frac{r_1}{r_2} < \frac{1}{R_{21}}$ holds as well, then $\tilde{E}$ and $\hat{E}$ are also unstable. In this case, coexistence of the two plants may be expected.

2.2.3. The Interior Equilibrium

It is very difficult to obtain an analytic expression for the interior equilibrium E^* due to the high nonlinearity of the equations. Nevertheless, we have derived equations whose positive root(s) can be used to determine E^*. Setting the right hand side of (8) equal to zero and eliminating the variable P^* we obtain the following equations for N_1^* and N_2^*:

$$\begin{aligned} \mathcal{F}_1(N_1^*, N_2^*) =: & \sum_{i,j=1, i\neq j}^{2} \frac{B_i e_i N_i^* (2 + h_i e_i N_i^* + 2h_j e_j N_j^*)}{2(1 + h_1 e_1 N_1^* + h_2 e_2 N_2^*)^2} - D = 0, \\ \mathcal{F}_2(N_1^*, N_2^*) =: & \sum_{i,j=1, i\neq j}^{2} (-1)^j r_i e_j \left(1 - \frac{N_i^* + \beta_{ij} N_j^*}{K_i}\right) \\ & (2 + 2h_i e_i N_i^* + h_j e_j N_j^*) = 0. \end{aligned} \tag{22}$$

Each of Eq. (22) defines a curve in the (N_1, N_2) plane, and an intersection of the two curves is an interior equilibrium, as shown in the two figures on the left of Fig. 3 (the circle and triangle indicate the stability and instability, respectively). The figures on the right are produced by AUTO [15] showing that the solutions either converge to the interior equilibrium (top right) or diverge (bottom right).

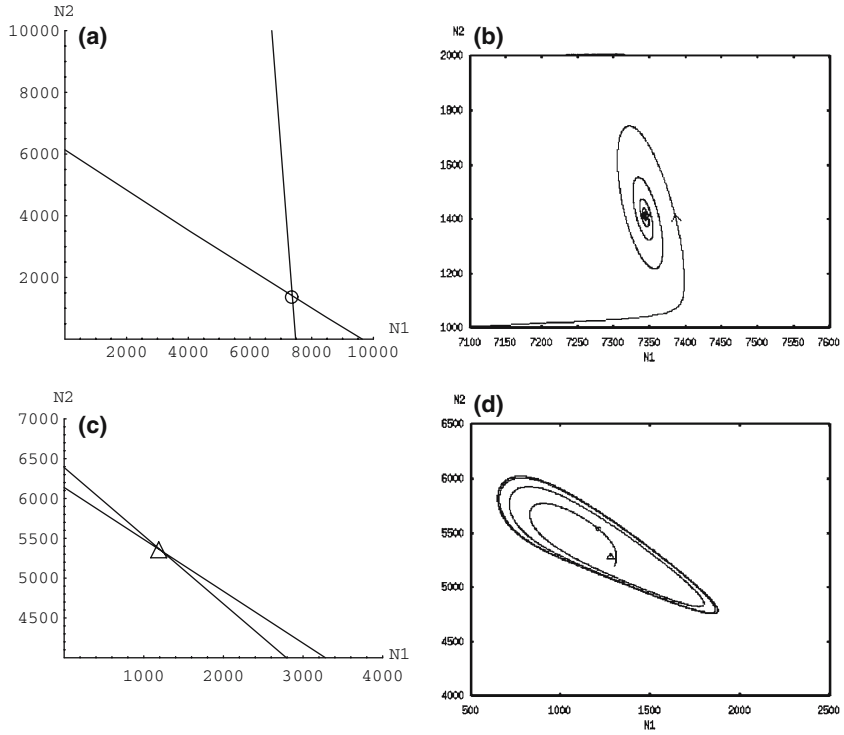

Figure 3. The figures on the left show the interior equilibrium E^* as an intersection of the two curves of equations (22). The figures on the right illustrate the stability of E^* (top) and instability of E^* (bottom) under different conditions.

3. BIFURCATION ANALYSIS

A bifurcation occurs when the stability of an equilibrium or a periodic solution switches as the bifurcation parameter passes through a critical point. We have found several bifurcations in the last section. In this section, more complex bifurcations are demonstrated thorough numerical simulations. In our first example, we choose r_1 to be the bifurcation parameter and examine how the stability of the equilibria may change when r_1 is varied.

Assume that $D < B_2G_2$. Rewrite the condition $\dfrac{r_1}{r_2} < R_{12}$ in terms of r_1:

$$r_1 < \frac{r_2 B_2 e_1 \hat{N}_2(1-\hat{N}_2/K_2)}{D(1-\beta_{12}\hat{N}_2/K_1)}\sqrt{1-\frac{D}{G_2B_2}}, \tag{23}$$

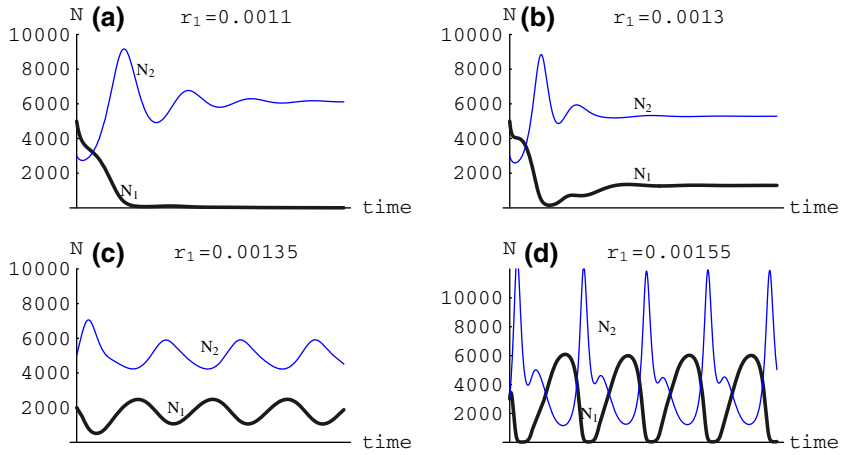

Figure 4. Time plots showing the bifurcations as r_1 increases.

where

$$\hat{N}_2 = \frac{2G_2}{e_2}\left(\frac{1}{\sqrt{1-\frac{D}{B_2G_2}}}-1\right).$$

If all parameters are fixed except r_1, then the right-hand side of (23) defines a critical value, r_{c1} (c for critical), such that $\hat{E}$ is stable if $r_1 < r_{c1}$, and unstable if $r_1 > r_{c1}$. This is confirmed by our numerical simulations which are shown in Fig. 4. This figure illustrates one scenario in which the species 1 is assumed to be more toxic (i.e., $G_1 = 80 > G_2 = 8$), but may have a slower growth rate (e.g., $r_2 = 0.015$ and $r_1 < r_2$). A more detailed discription on parameter values and units is given in the discussion of Fig. 5. It shows three critical points r_{ci}, $i = 1, 2, 3$. When $r_1 = 0.0011 < r_{c1} \approx 0.0012$, species 1 goes extinct due to the stability of $\hat{E}$ (Fig. 4(a)). For $r_1 = 0.0013 > r_{c1}$, $\tilde{E}$ is unstable and the two species coexist in the form of a stable interior equilibrium E^* as long as r_1 is not too large (Fig. 4(b)). If we continue to increase r_1, the interior equilibrium loses its stability and a stable periodic solution emerges. For example, when $r_1 = 0.00135$ a stable periodic solution occurs (Fig. 4(c)), indicating that there exists another critical point $r_{c2} \in (0.0013, 0.00135)$ at which a Hopf bifurcation occurs. When r_1 is further increased a period doubling bifurcation occurs at $r_{c3} \approx 0.0015$, i.e., when r_1 passes through r_{c3} the periodic solution becomes unstable and a stable period-2 solution appears (Fig. 4(d)).

Note that the coexistence of the two plant species exhibited in Fig. 4 will not be possible for the selected parameter values if the herbivore population is absent. To see this more clearly, we remove the P equation by assuming that $P = 0$ in the system (8). Then we obtain the standard Lotka–Volterra competition model. One of the outcomes predicted by the Lotka–Volterra model is competitive exclusion, which will occur if one of the two conditions holds:

$$\frac{\beta_{21} K_1}{K_2} > 1, \qquad \frac{\beta_{12} K_2}{K_1} > 1. \tag{24}$$

The second condition is indeed satisfied by the parameter values used in Fig. 4. Plant species 1 will be excluded in the absence of herbivores, i.e., $N_1(t) \to 0$ as $t \to \infty$. However, from Fig. 4, we see that this competitive exclusion occurs only when the growth rate of species 1 is small, i.e., $r_1 < 0.0011$ (Fig. 4(a)). Species 1 is able to coexist with species 2 when $r_1 > 0.0011$ either at a steady state (Fig. 4(b)) or in an oscillatory fashion (Fig. 4(c) and (d)).

Of course bifurcations also can be studied for parameters other than the growth rate r_i. For example, let the competition parameter β_{12} be the bifurcation parameter. Figure 5 shows several bifurcations. For demonstration purposes, we have chosen the parameter values that capture some of the characteristics of two plant species, black spruce (*Picea mariana*, hereafter Pima), and resin birch (*Betula resinifera*, hereafter Bere), referred to as species 1 and 2, respectively. These plants are browsed by snowshoe hares *Lepus americanus* which may ingest a different amount of toxin-containing twigs of species i.

The parameter values are either estimated by using experimental/field data or are selected for the purpose of demonstration. For example, In the empirical paper by Bryant *et al.* (in press), G_i was estimated by using T_i (the toxin content of plant) and M_i (toxin satiation), and $G_i = M_i/T_i$ $(i = 1, 2)$. T_i was calculated using OG resin content ($T_1 = 20\%$ for spruce and $T_2 = 6\%$ for birch), and M_i was calculated using the mass of resin consumed/day in the resin-chow trials ($M_1 = 1.0$ g resin/hare/day for spruce and $M_2 = 2.7$ g resin/hare/day for birch). Converting to the appropriate units we obtain $G_1 = 8$ and $G_2 = 80$ (see Table 1 for the unit). For r_i we adopted the data from Chapin *et al.* [12] in which the unit is grams dry mass/seedling. The seedlings were about 3 months old and grown in pots. These data give us an index of growth rate for the whole plants, which will be in the range $0.001 < r_i < 0.003$. We have considered two scenarios: (i) $G_1 < G_2$ and $r_1 < r_2$ and (ii) $G_1 < G_2$ and $r_1 > r_2$. The first scenario assumes that the more toxic plant (type 1) has a lower growth rate while the second scenario assumes that the more toxic plant has a higher

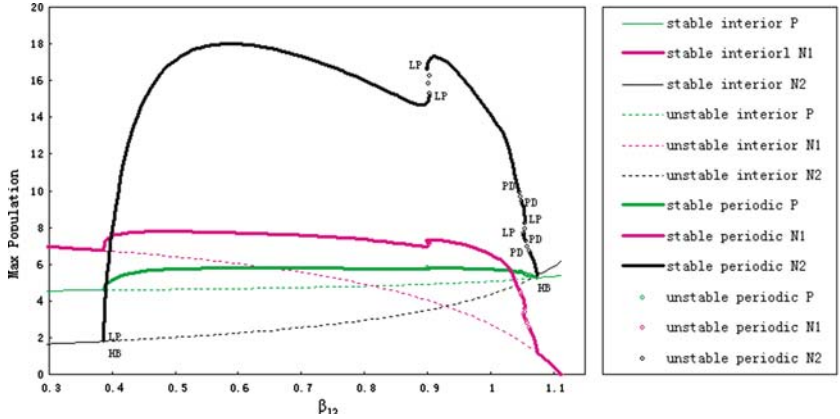

Figure 5. Bifurcation diagram calculated using AUTO for the case when $G_1 < G_2$ and $r_1 < r_2$. It shows two bifurcation points (HB). There are also period doubling bifurcations (PD) and limit points (LP).

growth rate. For illustration purposes we have chosen $r_1 = 0.00167$ and $r_2 = 0.0028$ for the first case and $r_1 = 0.00167$ and $r_2 = 0.0028$ for the second case. h_i is chosen to be equal to $1/(2G_i)$, $K_1 = 10^4$ and $K_2 = 10^5$ (these values may vary from one environment to another). We do not have data for other parameters which have been chosen to be in reasonable ranges. $D = 0.00003$ for Fig. 5 and 0.00014 for Fig. 7. $B_1 = 0.000034$ and $B_2 = 0.000056$ for both Figs. 5 and 7. $e_1 = 0.0001$, $e_2 = 0.0005$ and $\beta_{21} = 0.3$ for Fig. 5, and $e_1 = e_2 = 0.0005$ and $\beta_{21} = 0.75$ for Fig. 7. β_{12} is chosen to be the bifurcation parameter.

Figure 5 is a bifurcation diagram generated using AUTO [15]. For $\beta_{12} < 0.38$ or $\beta_{12} > 1.1$ there is a stable interior equilibrium. This interior equilibrium becomes unstable when β_{12} passes through the Hopf bifurcation (HB) points, $\beta_{c1} \approx 0.38$ and $\beta_{c2} \approx 1.1$, and stable periodic solutions appear. As β_{12} continues to change (increasing from the left branch of periodic solutions and decreasing from the right branch of periodic solutions) other bifurcations occur including period doubling (PD) bifurcations and limit point (LP) bifurcations. Figure 6 presents four time plots to illustrate some of these bifurcations. The top two plots are for the cases in which β_{12} is slightly greater than 0.38 and slightly less than 1.1, respectively. The bottom two plots are for the case in which β_{12} is between 0.38 and 1.1 but near the PD bifurcation points.

Figures 5 and 6 are for the case in which species 1 is more toxic (i.e., $G_1 < G_2$) but has a slower growth rate (i.e., $r_1 < r_2$). Other bifurcations are also possible if we consider different scenarios. Figures 7 and 8 are for the

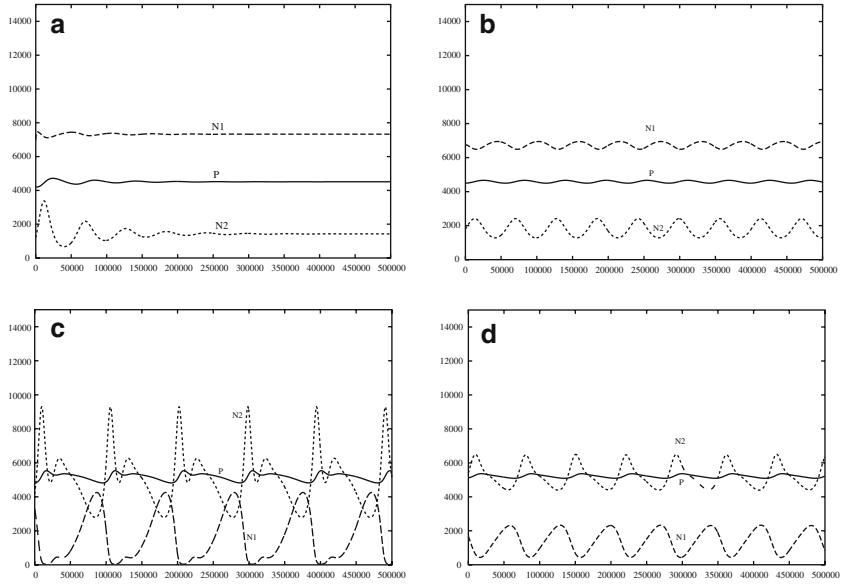

Figure 6. Time plots for various values of β_{12} chosen according to the diagram in Figure 5.

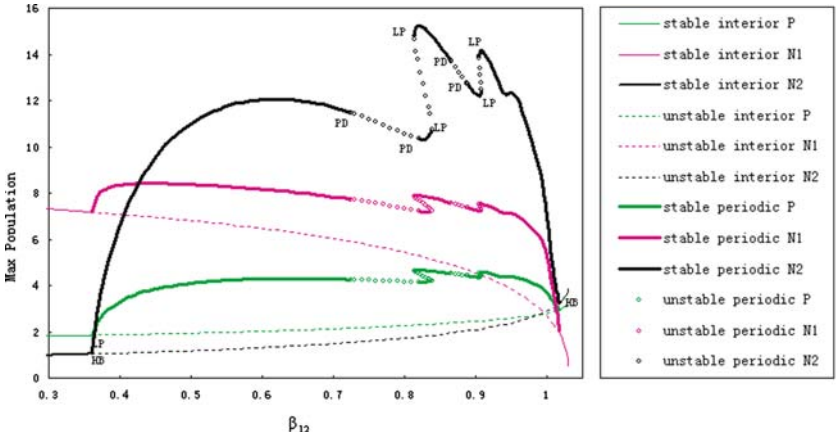

Figure 7. Bifurcation diagram for the case $G_1 < G_2$ and $r_1 > r_2$. The labels have the same meanings as in Figure 5.

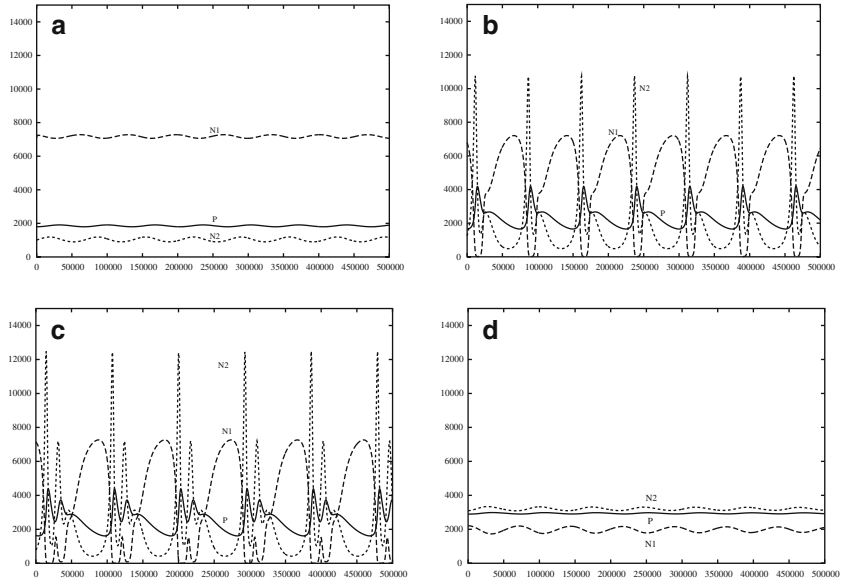

Figure 8. Time plots corresponding to Figure 7.

case in which the plant species 1 is still more toxic but has a higher growth rate, i.e., $G_1 < G_2$ and $r_1 > r_2$. Although most of the dynamics of the two scenarios are similar, Fig. 7 and 8 exhibit some differences in the dynamics compared to those shown in Figs. 5 and 6.

4. DISCUSSION

We have modified the standard Holling type II functional response to incorporate reduced consumption by herbivores due to plant toxins. The dependence of an herbivore's functional response on plant toxicity has not been considered in previous models for plant–vertebrate herbivore interactions, despite the acknowledged role of plant toxins in diet choice by herbivores from a variety of taxa and ecological contexts [4,5,17,18,24]. Thus, the addition of toxicity within the analytical framework of a functional response provides an opportunity for enhanced realism with which to predict population-level behavior.

Our analysis of the modified system described by the toxin-mediated functional response revealed a rich array of possible behaviors spanning the spectrum from damped oscillations to complicated cyclical dynamics through Hopf bifurcations and period-doubling bifurcations. Differences

in dynamical behavior stem from two sources. First, interspecific differences in plant growth rates, competitive ability, and carrying capacity interact to influence dynamics irrespective of herbivory. Evidence from the tropical rain forest and northern taiga forest suggests that slow-growing plants invest relatively more in toxic defenses than do fast growing plants [5,6,12]: a "fast" life history places a premium on rapid growth and early reproduction, versus a "slow" life history emphasizing tolerance to stressful conditions. In the former strategy, investment in growth provides plants an enhanced capacity to replace tissues eaten by herbivores (compensatory growth), whereas in the slow-growth strategy, enhanced chemical defense against herbivory is emphasized.

A second source of dynamical complexity stems from variation in herbivore responses to plant toxins. Changes by herbivores in the handling time, threshold capacity, or conversion into offspring of toxin-containing plant material can alter equilibrial relationships of 2-species plant communities by magnifying or diminishing the costs of herbivory for plants that vary in toxin content. Feeding trials with snowshoe hares suggested that considerable intra-specific variation exists in sensitivity to plant toxins; hares from northern latitudes were capable of consuming more toxin-containing twigs than hares from southern latitudes [29]. The implications for plant communities of such geographic variation in herbivore tolerance warrant closer inspection.

Our analysis also demonstrates the possible influence of herbivores on the diversity of plant communities. For example, we compared the outcomes of two models of plant competition, one of which included herbivores. For the same set of parameters, the model without herbivores predicted competitive exclusion, whereas the model with herbivores predicted coexistence of the plant species. Thus, herbivores may be able to ameliorate the consequences of interspecific competition by virtue of their responses to plant toxins. Note that our discussion has focused on comparisons of two plant species, but the level of biological organization is somewhat arbitrary. With modifications to allow for interbreeding, our model could be applied to genotypes within a single species, raising the interesting possibility that herbivores may be capable of maintaining distinctive "defense" genotypes in a plant population.

One of the major results of a quarter century of research on chemically mediated interactions between woody plants and vertebrate herbivores has been the empirical verification of the Freeland and Janzen hypothesis [18]: Lipophilic plant toxins determine the intake of many woody plants by vertebrate herbivores (e.g., [4,17,24]). The verification of the Freeland and Jazen hypothesis has provided a sound foundation for starting the mathematical analysis of toxin-regulated plant–herbivore

interactions (Bryant *et al.*, in press) using the functional response approach of [19,20,22]. The preliminary analysis of Bryant *et al.* (in press) predicts that toxicity differences among plants cause differences in the plant biodiversity of ecosystems, and these predictions have been experimentally verified.

Competition among plants is a major driver of plant biodiversity, both at the level of plant species and at the level of plant chemical genotypes. Our model has for the first time coupled the Lotka–Volterra competition equations to a toxin-regulated functional response, thereby greatly extending the theoretical scope and application of chemical ecology. This model has led to the testable prediction that the dynamical behavior of ecosystems can be controlled by the interaction between toxin-regulated herbivory and plant competition. We are now in the process of mathematically analyzing how the ability of slowly growing [3,11] and chemically defended [5,6,13] plants to persist at very low-resource availabilities affects the defense-competition dynamics. The model presented in the current paper will for the first time allow the mathematical analysis of herbivory's effects on ecosystems where Lotka–Volterra competition may be expected (e.g., some agroecosystems), and the mathematical variants we are now exploring will allow similar analysis of the effects of herbivory in natural ecosystems where the ability of some plants to persist in low resource environments may override Lotka–Volterra competition. Such analyses will be of importance to ecologists and resource managers faced with understanding plant–herbivore interactions in a world where invasive plants and herbivores are a significant and growing problem, where changing atmospheric chemistry is expected to alter the biochemistry and palatability of many plant species, and where climate warming is expected to cause unparalleled migrations of both plants and herbivores.

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