Notes and Comments

Are Tolerance, Avoidance, and Antibiosis Evolutionarily and Ecologically Equivalent Responses of Plants to Herbivores?

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Plants possess numerous traits that reduce the potentially detrimental effect of herbivores. These traits, referred to as "defense traits," fall into two broad categories, resistance traits, which reduce herbivory, and tolerance traits, which reduce the impact of herbivory on fitness. Plant resistance can be further divided into avoidance, which affects herbivore behavior (i.e., recognition or preference for a particular plant), and antibiosis, which reduces herbivore performance (Painter 1958; Futuyma 1983). Examples of avoidance traits include egg mimics in *Passiflora* sp. that reduce oviposition by *Heliconius* (Williams and Gilbert 1981) and may also include feeding inhibitors or, conversely, reduced concentrations of feeding or oviposition stimulants (Fraenkel 1959; Jermy 1966; Feeny et al. 1983). Examples of antibiotic resistance traits are numerous and include alkaloids, cardenolides, cyanogens, fouranocumarins, and glucosinolates (reviewed in Rosenthal and Berenbaum 1991; Bernays and Chapman 1994). Lack of evidence for correlations between host preference and herbivore performance suggest that these two resistance strategies often may be independent (Thompson 1988; Thompson et al. 1990; Pilson 1992; but see Via 1986). The distinction between preference and performance, although incorporated into models of the evolution of herbivorous insects (Georghiou 1972; Gould 1984; Lockwood et al. 1984; Kennedy et al. 1987), has not bean incorporated into previous models of the evolution of plant defense.

Likewise, previous models predicting the evolution of resistance (Rhoades and Cates 1976; Simms and Rausher 1987; Herms and Mattson 1992) or tolerance (Fineblum and Rausher 1995; Abrahamson and Weis 1997; Tiffin and Rausher 1999) have not generally included the effects of plant characters on herbivore populations (but see Adler and Karban 1994). The potential for plant traits that affect herbivore preference and performance to alter the temporal and spatial dynamics of herbivore populations has been shown by numerous empirical and theoretical studies (Rhoades 1983; Edelstein-Keshet 1986; Harrison and Karban 1986; Edelstein-Keshet and Rausher 1989; Power 1991; Adler and Karban 1994; Morris and Dwyer 1997; Thaler 1999; Underwood 1999). Tolerance does not directly affect herbivore preference or performance and therefore herbivore dynamics may respond very differently to tolerance and resistance traits. Moreover, since herbivores are likely the evolutionary force that selects for tolerance and resistance, one may expect that differential effects of defense traits on herbivores may result in these traits having different evolutionary dynamics. Analytical results of a phenotypic model by Roy and Kirchner (in press) show that traits conferring antibiosis resistance to pathogens are more likely to be maintained in a population than traits conferring tolerance to pathogens. The model presented in this note differs from Roy and Kirchner's model by having an explicit, although simple, genetic basis and by extending their analyses, including both avoidance and antibiosis resistance, herbivores that are not monophagous, and antibiosis mechanisms that do not completely prevent herbivores from reproducing.

The evolutionary response of plants to herbivores may also affect the ecology of plants and herbivores. Because herbivory may reduce reproductive output (Janzen 1969; Rockwood 1973; Rausher and Feeny 1980; Marquis 1984; Raffa and Berryman 1987), plant traits that reduce herbivore loads may result in increased reproductive output, thereby altering plant population dynamics. Alternatively,

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Table 1: Symbols and their definitions

Symbol	Definition
α	Degree to which the population level intensity of herbivory is reduced by the *AB* allele
β_X	Degree to which herbivore damage experienced by genotype X is reduced due to the physiological properties of X
h	Function describing the size of the herbivore population
η_X	Herbivore's relative preference for plants of genotype X
ρ_X	Function describing the relative preference of the herbivore for an individual plant carrying allele X
D	Herbivore load
H_0	The size of the herbivore population on initial plants
W_0	Fitness of genotype I in the absence of herbivores
W_X	Fitness of genotype X in the presence of herbivores
X	Genotype T (tolerance), AV (avoidance), AB (antibiotic), or I (initial)
C_X	Fitness costs associated of genotype X
p_X	Frequency of the X allele

defense mechanisms may reduce reproductive output because of allocation costs associated with these traits (Berenbaum et al. 1986; Simms 1992; Bergelson 1994; Mauricio and Rausher 1997; Stowe 1998). Reproductive output may, in turn, alter the community dynamics of pathogens and herbivores that use seeds or seedlings as a resource (Janzen 1969; Harper 1977). Plant defense traits may also directly affect herbivores by reducing the size of herbivore populations or by altering herbivore movement and choice of host plants.

In order to gain a better understanding of the evolutionary response of plants to herbivores, I present a simple model to address the following questions: First, under what conditions will avoidance, antibiosis, and tolerance alleles invade and fix in a population? Second, are stable genetic polymorphisms expected to be maintained for these traits? Third, what effect do these three evolutionary responses to herbivores have on mean fitness of the plant population at equilibrium? And, finally, what effect do evolutionary changes in a plant population have on the intensity of herbivory?

Model

The model follows the fitness and genotype frequencies at a single locus within a single plant population. There are, however, two plant populations included to allow for herbivore shifts away from the focal population. The model makes several simplifying assumptions. First, the host plant is assumed to be an annual with discrete, nonoverlapping generations and a haploid genome. Second, herbivores are assumed to have no effect on plant population size but may select for plant tolerance and resistance characters. Third, plant characters may affect herbivore behavior and population size but do not cause evolutionary change in the herbivore population. Finally, it is assumed that the characteristics of the second plant population (i.e., species) remain unchanged. A haploid model was chosen for ease of analysis.

Tolerance alleles are designated by the subscript T, avoidance alleles by the subscript AV, antibiotic alleles by the subscript AB, and the genotype of the initial population by the subscript I (all symbols are presented in table 1). The frequencies of T, AV, AB, and I alleles are designated by p_T, p_{AV}, p_{AB}, and p_I, respectively. The initial, I, genotype is considered to be nontolerant relative to T alleles and nonresistant relative to AV and AB alleles. For simplicity of notation, the focal plant population is not subscripted. The subscript 2 designates values for plants from one or more different populations or species that are also part of the herbivore's diet.

The basic form of the model is similar to that of Simms and Rausher (1987). The fitness of an individual plant of genotype X, where $X = T, AV, AB,$ or I, within population 1 is equal to

$$W_X = W_0 - C_X - D_X T_X, \tag{1}$$

where W_0 is the fitness of genotype I in the absence of herbivory, C_X is equal to the costs associated with allele X, D_X is equal to the herbivore load experienced by an individual of genotype X within population 1, and T_X is an inverse measure of tolerance to damage; it reflects the degree to which the effects of herbivory are mitigated by allele X. The terms W_0, C_X, and T_X are assumed to be constants. The variable D_X is described by

$$D_X = h(H_0, \alpha, p_{AB})\rho_X(\nu, \eta_X, \eta_2, p_X)\beta_X, \tag{2}$$

where the function $h(H_0, \alpha, p_{AB})$ describes the size of the herbivore population, the function $\rho_X(\nu, \eta_X, \eta_2, p_X)$ describes the relative probability that an individual herbivore feeds on plants of genotype X relative to all other genotypes within the population, and β_X is the rate at which her-

bivores feed on a plant of genotype X. I assume the following linear function for h:

$$h(H_0, \alpha, p_{AB}) = H_0(1 - \alpha p_{AB}), \tag{3}$$

where H_0 is the number of herbivores per plant under the initial conditions and α is a constant between 0 and 1 that reduces the size of the herbivore population because of effects of the AB allele on herbivore reproduction. The function ρ_X is given by

$$\rho_X(\nu, \eta_X, \eta_2, p_X) = \frac{\eta_X}{\left(\nu \sum_{X}^{T, AV, AB, I} p_X \eta_X\right) + (1 - \nu)\eta_2}, \tag{4}$$

where ν is the proportion of all plants that are in population 1, η_X is a measure of the relative preference of herbivores for genotype X, and η_2 is a measure of the relative preference of herbivores for population 2 plants. The expression in the denominator of equation (4) is simply a measure of the overall mean food quality, in terms of preference, of plants available to an herbivore, whereas the numerator is the preference of herbivores for genotype X relative to other genotypes. Since ρ_X describes the relative probability that an herbivore chooses to feed on an individual of genotype X, the numerator of equation (4) need not include the frequency of X individuals within the plant population. In comparison, an equation describing the relative probability that an individual herbivore feeds on plants of genotype X would include the frequency of X individuals within the herbivores' diet breadth, νp_X. In other words, equation (4) is equivalent to the probability that an individual herbivore chooses to feed on plants of genotype X divided by the frequency of genotype X individuals among all plants upon which the herbivore may feed. Substituting equations (2)–(4) into equation (1) gives the following expression for the fitness of genotype X:

$$W_X = W_0 - C_X - H_0(1 - \alpha p_{AB}) \times \left[\frac{\eta_X}{\left(\nu \sum_{X}^{T, AV, AB, I} p_X \eta_X\right) + (1 - \nu)\eta_2}\right] \beta_X T_X. \tag{5}$$

All alleles other than I alleles are assumed to incur costs, $C_T, C_{AV}, C_{AB} > C_I = 0$. Avoidance alleles, which affect only behavior, are defined by being less preferred by the herbivore than other alleles, $\eta_{AV} < \eta_T = \eta_I = \eta_{AB}$. Antibiotic alleles are defined by reducing herbivore intensity on AB genotypes more than on other genotypes through the rate at which herbivores feed on genotype X, $\beta_{AB} < \beta_T = \beta_{AV} = \beta_I$. Tolerance alleles are defined by lower effects of herbivory on fitness, $T_T < T_{AV} = T_{AB} = T_I$. For simplicity, it is assumed that $\eta_T = \eta_{AB} = \eta_I = 1$, $\beta_T = \beta_{AV} = \beta_I = 1$, $T_{AV} = T_{AB} = T_I = 1$, and that the values of all variables range between 0 and 1.

Since AB alleles reduce the size of the total herbivore population, through the parameter α, AB alleles may affect herbivore intensity on all genotypes. For example, the size of an insect herbivore population may be limited by the number of larvae that pupate, and AB may reduce survival of larvae to pupation. Alternatively, if the size of the herbivore population is independent of plant characteristics, for example, if population size is limited by overwintering sites, then $\alpha = 0$ and the AB allele will have no effect on the herbivore population. The parameter β_X differs from α in that it affects only the herbivore intensity on genotype X. For example, AB plants may experience less intense herbivory because larvae that feed on AB genotypes die before causing damage equal to the damage larvae would cause on a different genotype.

Analyses

I present three series of analyses. The first series examines the conditions under which T (tolerance), AV (avoidance), and AB (antibiotic) alleles are expected to invade, to fix, and to be maintained in a population. These analyses are first conducted when each trait is encoded for by alleles segregating at independent loci (i.e., no genetic covariance between traits) and then when alleles cosegregate at a single locus (i.e., negative genetic covariance between traits). The second series of analyses examines the effect these alleles have on the mean fitness of the plant population they invade. The final series of analyses examines the effects of each of the three alleles on the intensity of herbivory experienced by the plant population they invade and on the second plant population. The effects of an increase or decrease in the frequency of T, AV, and AB alleles on mean fitness in the plant population and on the intensity of herbivory are qualitatively similar whether alleles segregate at independent loci or at a single locus. For this reason, the second and final series of analyses are presented only for alleles segregating at independent loci.

Invasion, Fixation, and Maintenance of Genetic Variation

Tolerance. When T, AV, and AB are encoded by alleles at independent loci, the expected evolutionary dynamics of each trait can be determined by comparing the fitness of the defense trait with the fitness of the I allele. A new allele is expected to increase in frequency in a population of initial, I, alleles if $W_X > W_I$. Using equation (5) to describe

the fitness associated with T and I alleles and the fact that $p_T + p_I = 1$, the condition for $W_T > W_I$ is equivalent to

$$C_T < H_0\left[\frac{1}{\nu + (1-\nu)\eta_2}\right](1 - T_T). \tag{6}$$

The right side of this inequality is equivalent to the benefit of tolerance (i.e., the effects of herbivory on initial plants minus the effects of herbivory on tolerant plants), whereas the left side is simply the costs of tolerance. Therefore, as expected, tolerance is favored if the costs of tolerance are less than the benefits. Equation (6) is independent of allele frequencies, and thus the condition necessary for an allele to invade ($p_T \approx 0$) is equivalent to the condition for fixation ($p_T \approx 1$). Consequently, if a T allele invades it will be fixed, and, in this two-allele system, genetic dimorphisms will not be maintained (fig. 1*A*).

Avoidance. At an *AV* locus, a new allele is expected to increase in frequency when $W_{AV} > W_I$, which is equivalent to

$$C_{AV} < H_0\left[\frac{1 - \eta_{AV}}{\nu(p_{AV}\eta_{AV} + p_I) + (1-\nu)\eta_2}\right]. \tag{7}$$

This inequality is dependent upon the frequency of the *AV* allele. In order for an *AV* allele to invade, this condition must be met when $p_{AV} \approx 0$, that is, when

$$C_{AV} < H_0\left[\frac{1 - \eta_{AV}}{\nu + (1-\nu)\eta_2}\right]; \tag{8}$$

whereas, for fixation to occur, this inequality must be met when $p_{AV} \approx 1$, that is, when

$$C_{AV} < H_0\left[\frac{1 - \eta_{AV}}{\nu\eta_{AV} + (1-\nu)\eta_2}\right]. \tag{9}$$

Since, by definition, $\eta_{AV} < 1$, equation (9) will be true whenever equation (8) is true. Therefore, if an *AV* allele

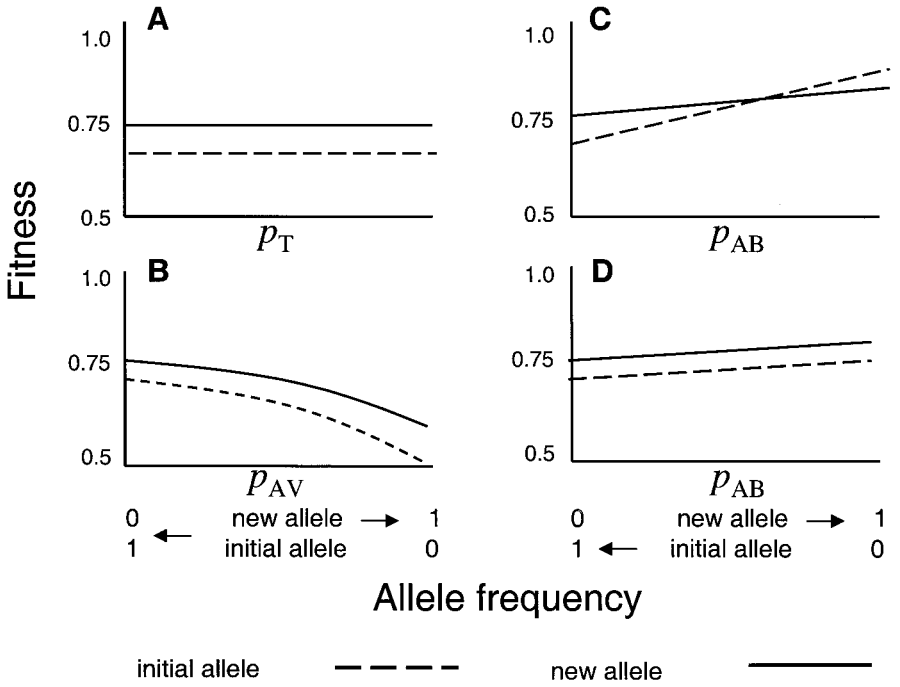

Figure 1: Examples of the fitness of tolerance (T), avoidance (AV), and antibiosis (AB) genotypes (*solid line*) after invading a population of nonresistant, nontolerant initial alleles (*dashed line*). *A*, comparison of tolerance and initial alleles when $T_T = 0.5$ and $T_I = 1$; *B*, comparison of avoidance and initial alleles when $\eta_{AV} = 0.5$ and $\eta_I = 1$; *C*, comparison of antibiosis and initial alleles when $\alpha = 0.5$, $\beta_{AB} = 0.5$, and $\beta_I = 1$; and *D*, comparison of antibiosis and initial alleles when $\alpha = 0.2$, $\beta_{AB} = 0.5$, and $\beta_I = 1$. Fitness values were calculated using equation (5). For all plots, $W_0 = 1$, $D = 0.3$, and $C_T = C_{AV} = C_{AB} = 0.1$.

invades it will be fixed, and a stable dimorphism will not be possible (fig. 1*B*).

Antibiosis. At an *AB* locus, a new allele is expected to increase in frequency when $W_{AB} > W_I$, which, given that $p_{AB} + p_I = 1$, is equivalent to

$$C_{AB} < H_0(1 - \alpha p_{AB})\left[\frac{1}{\nu + (1 - \nu)\eta_2}\right](1 - \beta_{AB}). \quad (10)$$

In order for an *AB* allele to invade, equation (10) must be true when $p_{AB} \approx 0$, that is, when

$$C_{AB} < H_0\left[\frac{1}{\nu + (1 - \nu)\eta_2}\right](1 - \beta_{AB}), \quad (11)$$

and *AB* is expected to fix when $p_{AB} \approx 1$, that is, when

$$C_{AB} < H_0(1 - \alpha)\left[\frac{1}{\nu + (1 - \nu)\eta_2}\right](1 - \beta_{AB}). \quad (12)$$

Unless $\alpha = 0$, the condition for an *AB* allele to fix is more restrictive than the condition for the allele to invade. A stable dimorphism is thus possible when $W_{AB} = W_I$, which yields an equilibrium frequency of

$$\hat{p}_{AB} = \frac{1}{\alpha} - \frac{C_{AB}[\nu + (1 - \nu)\eta_2]}{\alpha H_0(1 - \beta_{AB})}. \quad (13)$$

Therefore, unlike the situation for *T* and *AV* alleles, a stable dimorphism between *AB* and *I* alleles is possible (fig. 1*C*).

Tolerance, Avoidance, and Antibiosis Alleles Cosegregate. The above analyses describe the expected dynamics when *T*, *AV*, and *AB* traits are determined by independent loci. Slightly different dynamics are expected when two defense alleles cosegregate at a single locus. The analyses for each pair of cosegregating alleles are similar. As such, I present the equations describing the fate of a *T* allele invading a population of *AV* alleles. Then I present the results, but not the equations, for *T* and *AB* alleles and *AV* and *AB* alleles cosegregating.

In order for a *T* allele to invade a population of *AV* alleles, it is necessary that $W_T > W_{AV}$ when $p_T \approx 0$, that is, when

$$C_{AV} - C_T > H_0\left[\frac{T_T - \eta_{AV}}{\nu\eta_{AV} + (1 - \nu)\eta_2}\right], \quad (14)$$

whereas the condition for the *T* allele to fix is

$$C_{AV} - C_T > H_0\left[\frac{T_T - \eta_{AV}}{\nu + (1 - \nu)\eta_2}\right]. \quad (15)$$

If the benefits of tolerance, T_T, are greater than the benefits of avoidance, η_{AV}, then the right sides of the above inequalities are positive. The condition for invasion is thus more restrictive than fixation ($\rho_T \approx 1$ and $p_{AV} \approx 0$) and a *T* allele that invades will be fixed. If the benefits of tolerance are equal to the benefits of avoidance ($T_T = \eta_{AV}$), then the condition for invasion is equal to the condition for fixation and when a *T* allele invades it fixes. In contrast, if the benefits of tolerance are less than the benefits of avoidance, then the right sides of equations (14) and (15) are negative and the condition for invasion is less restrictive than the condition for fixation. The evolutionary dynamics thus depend on the relationship between the costs of the *T* and *AV* alleles. If $C_T \leq C_{AV}$, then a *T* allele that invades will be fixed; whereas, if $C_T > C_{AV}$, then it is possible for equation (14) to be true and equation (15) to be false. Under these conditions, a stable dimorphism between *T* and *AV* is possible.

The dynamics of a new *AV* allele in a population of *T* alleles simply mirror the dynamics of a new *T* allele in a population of *AV* alleles. When the benefits of *AV* are greater than the benefits of *T* ($\eta_{AV} > T_T$), the condition for invasion is less restrictive than the condition for fixation. Therefore, the *AV* allele will either fix or be maintained in equilibrium with the *T* allele, depending on the relative costs associated with those alleles. In contrast, if the benefits of *AV* are equal to or less than the benefits of *T*, then the criteria for invasion are equal to or greater than, respectively, the condition for fixation; and if an *AV* allele invades it will be fixed.

Similar dynamics are expected when *T* and *AB* alleles cosegregate. When $T_T > \beta_{AB}$, the condition for a *T* allele to invade is less restrictive than the condition for the allele to be fixed. As such, stable dimorphisms between *T* and *AB* alleles are possible, but only if $C_T < C_{AB}$ (if $C_T \geq C_{AB}$, the *T* allele can not invade). If, however, $T_T \leq \beta_{AB}$, then the condition for invasion is equal to or greater than the condition for fixation and a *T* allele that invades will be fixed. The dynamics of a new *AB* allele in a population of *T* alleles mirror the dynamics of a new *T* allele evolving in a population of *AB* alleles. If β_{AB} is greater than or equal to T_T, the condition for the *AB* allele invading will be

more or equally restrictive, respectively, than the condition for fixation. Therefore, if an *AB* allele invades, it will be fixed. In contrast, if $\beta_{AB} < T_T$, the condition for invasion is less restrictive than the condition for fixation and a stable dimorphism is possible, but only when $C_{AB} > C_T$.

The dynamics of cosegregating *AB* and *AV* alleles also depend on their relative costs and benefits. If the benefits of avoidance are greater than the benefits of antibiosis ($\eta_{AV} > \beta_{AB}$), then the condition for an *AV* allele to invade a population of *AB* alleles is less restrictive than the condition for fixation. Therefore, a stable dimorphism is possible, but only if $C_{AV} < C_{AB}$ (if $C_{AV} \geq C_{AB}$, the *AV* allele will not invade). In contrast, if $\eta_{AV} = \beta_{AB}$ or $\eta_{AV} < \beta_{AB}$, then the condition for an *AV* allele to invade is equal to or greater than the condition for fixation, respectively, and if an *AV* allele invades it will be fixed. As expected, the dynamics of an *AB* allele that invades a population of *AV* alleles mirror the dynamics of an *AV* allele invading a population of *AB* alleles.

Effect of T, AV, *and* AB *Alleles on Mean Fitness of the Plant Population*

Tolerance. The effect of a new allele on mean population fitness can be determined by comparing mean population fitness before invasion with mean population fitness at equilibrium. Evaluating equation (1) with $p_I = 1$ provides the mean fitness of the initial population before the invasion of a new allele. Comparing the fitness of the initial population to the mean population fitness at equilibrium after a *T* allele invades, or W_T when $p_T = 1$ (since if *T* invades it fixes), reveals that whenever *T* is able to invade it will increase the population's mean fitness (fig. 1*A*).

Avoidance. The effect of an *AV* allele on mean fitness is slightly more complicated than the effects of a *T* allele and depends on the diet breadth of the herbivore. Mean population fitness after invasion is equal to W_{AV} when $p_{AV} = 1$, since if *AV* invades it fixes. Mean fitness following invasion and fixation of *AV* is greater than the mean fitness of the initial population, W_I when $p_I = 1$, that is, when

$$C_{AV} < H_0\left[1 - \frac{\eta_{AV}}{\nu\eta_{AV} + (1 - \nu)\eta_2}\right]. \tag{16}$$

If the herbivore is a specialist, then $\nu = 1$, the expression in parentheses in equation (16) is equal to 0, and, given the presence of costs, equation (16) can never be true. Therefore, an *AV* allele that invades will reduce the mean fitness of the plant (fig. 1*B*). This decrease is due to the fact that the average herbivore load is the same when *I* is fixed and when *AV* is fixed, but, in the latter case, there is an additional cost associated with the *AV* allele. Alternatively, if the herbivore is not a strict specialist, then $\nu < 1$ and the benefits of the *AV* allele may persist after fixation. As shown below, the reason the effect of *AV* on mean fitness depends on the diet breadth of the herbivore is that the *AV* allele can cause a shift in the intensity of herbivory from the population in which the *AV* allele evolves to a second plant population (i.e., species).

Antibiosis. The mean fitness following fixation of *AB* is greater than the mean fitness of the initial population when

$$C_{AB} < H_0\left[\frac{1}{\nu + (1 - \nu)\eta_2}\right][1 - \beta_{AB}(1 - \alpha)]. \tag{17}$$

Equation (17) will be true whenever an *AB* allele is able to invade. Therefore, the invasion of an *AB* allele will result in a higher mean fitness in the population, regardless of whether that allele is maintained in a stable dimorphism (fig. 1*C*) or goes to fixation (fig. 1*D*).

Effect of T, AV, *and* AB *Alleles on the Intensity of Herbivory*

Tolerance. The herbivore load experienced by plants in population 1 after each allele invades can be determined by evaluating the intensity of herbivory in population 1 (eq. [2]) at equilibrium. Before tolerance or resistance alleles invade, the intensity of herbivory experienced by the initial population is equal to

$$D = H_0\left[\frac{1}{\nu + (1 - \nu)\eta_2}\right]. \tag{18}$$

This is also equal to the intensity of herbivory after the *T* allele invades, indicating that *T* alleles do not affect the overall intensity of herbivory experienced in population 1 (fig. 2*A*). Moreover, because *T* alleles do not affect the size or behavior of the herbivore population, changes in the frequency of *T* within population 1 do not affect the intensity of herbivory experienced in population 2 (appendix).

Avoidance. After fixation of an *AV* allele, the intensity of herbivory experienced by plants in population 1 is equal to

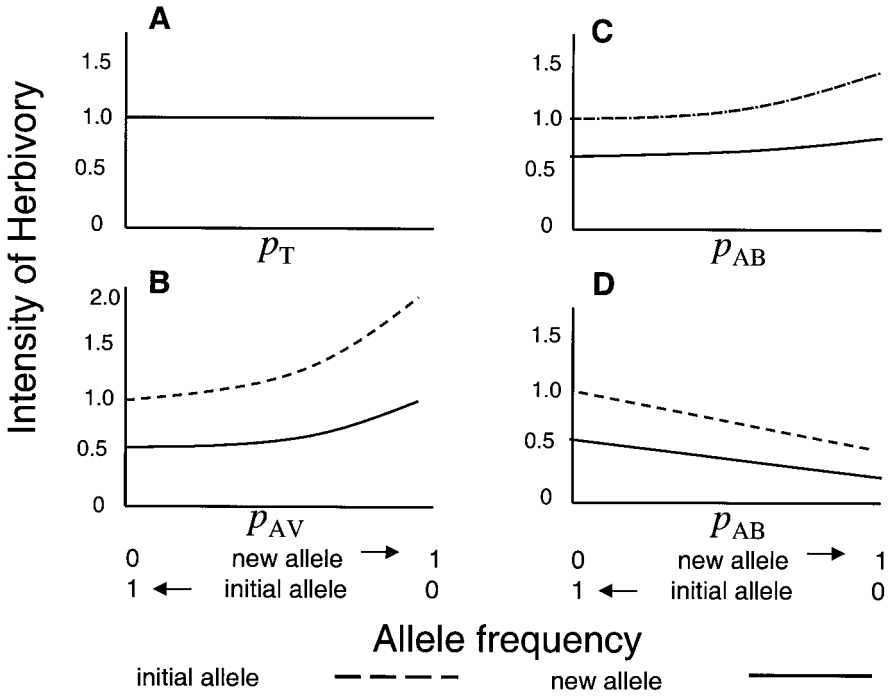

Figure 2: Examples of the intensity of herbivory following the invasion of tolerance (*T*), avoidance (*AV*), and antibiosis (*AB*) alleles (*solid line*) into a population of nonresistant, nontolerant initial alleles (*dashed line*). *A*, comparison of tolerance and initial alleles when $\nu = 1$, $\eta_T = \eta_I = 1$, and $H_0 = 1$; *B*, comparison of avoidance and initial alleles when $\nu = 1$, $\eta_{AV} = 0.5$, $\eta_I = 1$, and $H_0 = 1$; *C*, comparison of avoidance and initial alleles when $\nu = 0.5$, $\eta_{AV} = 0.5$, $\eta_I = \eta_2 = 1$, and $H_0 = 2$; and *D*, comparison of antibiosis and initial alleles when $\nu = 1$, $\eta_{AB} = \eta_I = 1$, $\alpha = 0.5$, $\beta_{AB} = 0.5$, $\beta_I = 1$, and $H_0 = 1$; the same plot is produced if $\nu = 0.5$ and $H_0 = 2$. Values were calculated using equation (2).

$$D = H_0\left[\frac{\eta_{AV}}{\nu\eta_{AV} + (1 - \nu)\eta_2}\right]. \tag{19}$$

If the herbivore is a strict specialist, then $\nu = 1$ and the intensity of herbivory experienced by *AV* individuals is equal to the intensity of herbivory experienced by *I* individuals before the *AV* allele invaded. Only when *AV* and *I* alleles are both in the population is there a difference in the intensity of herbivory they experience (fig. 2*B*). If the herbivore is not a strict specialist, then $\nu < 1$ and the fixation of the *AV* allele results in lower herbivore pressure in population 1 (fig. 2*C*) but higher herbivore loads on plants in population 2 (appendix). This occurs because *AV* alleles cause a redistribution of herbivores away from plants carrying the *AV* alleles.

Antibiosis. After an *AB* allele invades, the intensity of herbivory experienced in population 1 is equal to

$$D = H_0(1 - \alpha p_{AB})\left[\frac{1}{\nu + (1 - \nu)\eta_2}\right]. \tag{20}$$

This is less than the intensity of herbivory experienced by the initial population whenever $\alpha < 1$, regardless of whether the herbivore is a specialist or generalist. But, unlike *AV* alleles that cause increased intensity of generalist herbivores in population 2, the invasion of *AB* alleles results in

$$D_2 = H_0(1 - \alpha p_{AB})\left[\frac{\eta_2}{\nu + (1 - \nu)\eta_2}\right], \tag{21}$$

which is always less than D_2 before the invasion of the *AB* allele. Thus an antibiotic allele invading a population will reduce generalist herbivore loads in both the population in which the allele invades as well as other populations (i.e., species) that are part of the herbivores' diet breadth (fig. 2*D*). This is because *AB* genotypes reduce the size of

the herbivore population but do not cause a redistribution of herbivores.

Discussion

Evolutionary Dynamics of Defense Traits

Analysis of this model demonstrates that tolerance, avoidance, and antibiosis traits may have different evolutionary dynamics within populations. In particular, when these traits are determined by alleles segregating at independent loci, the conditions necessary for tolerance and avoidance alleles to invade a population are equal to or more restrictive than the conditions necessary for these alleles to become fixed. In contrast, the relative fitness benefits of antibiotic genotypes decrease as an antibiotic allele increases in frequency. Hence, when defense traits are not genetically correlated, frequency-dependent selection makes stable dimorphisms between antibiotic alleles and nonresistant, nontolerant alleles possible. If the results from this simple model extend to more complex quantitative genetic, coevolutionary models, then, when defense traits are not genetically correlated, a balance between costs and benefits may be sufficient to explain the maintenance of genetic variation for antibiotic resistance (Ayala and Campbell 1974; May and Anderson 1983; Roy and Kirchner, in press) but not to explain the maintenance of genetic variation for avoidance or tolerance.

The conclusion that costs and benefits are not sufficient to explain maintenance of genetic variation for avoidance may, however, be dependent on herbivore behavior. Some types of herbivore behavior may be expected to lead to stable dimorphisms between avoidance and initial alleles. For example, herbivores may disperse when avoidance alleles reach a high frequency, and dispersion of herbivores may reduce herbivore loads. Alternatively, increased frequency of avoidance genotypes is expected to cause crowding onto nonresistant individuals. Because of random herbivore movement or herbivore response to crowding, herbivore aggregation on nonresistance plants may result in higher than expected amounts of damage on avoidance genotypes growing nearby. Although these scenarios were not explored in the analyses, either may be expected to reduce the relative benefits associated with avoidance alleles. A reduction in benefits may lead to stable dimorphisms between avoidance and nonresistant alleles. Thus, herbivore behavior may be important in determining the evolution of resistance alleles.

Negative genetic covariances between defense traits may also result in tolerance, avoidance, and antibiosis alleles being maintained at intermediate frequencies, at least under some conditions. This result seemingly contradicts the results of the analytical model of Finelbum and Rausher (1995). Their model showed that a negative genetic covariance between tolerance and resistance leads to the fixation of either tolerance or resistance traits but not both. The results from this model are, however, consistent with those predictions when the costs of tolerance are equal to the costs of resistance, an assumption made in their model. In the analyses presented here, stable dimorphisms between different defense traits are possible only if the allocation costs associated with different defense strategies are unequal. Empirical data suggest that negative genetic covariances between tolerance and resistance may be found in some (van der Meijden et al. 1988; Fineblum and Rausher 1995; Stowe 1998) but not all systems (Simms and Triplett 1994; Mauricio et al. 1997; Tiffin and Rausher 1999). There are, however, few if any data available to determine the relative magnitude of allocation costs associated with different defense strategies. As such, it is not possible to assess how often negative genetic covarience between defense traits may contribute to the maintenance of genetic variation for these traits.

Effects of Defense Traits on Plant and Herbivore Communities

The analyses also demonstrate that defense traits may have very different effects on the plant population in which they evolve, the herbivores that select for these traits, and other plant and herbivore communities. When tolerance alleles invade a population, they are expected to increase that population's mean fitness but to have no effect on the community of herbivores that selects for tolerance. Although a change in mean fitness is not expected to affect evolution within a population, such a change may have important effects on interspecific competition as well as pathogens and other organisms that exploit seeds as a resource. Moreover, if plant population size is affected by mean fitness, then the increased mean fitness associated with tolerance alleles may also increase the size of the herbivore populations.

Unlike tolerance alleles, the effects of avoidance alleles depend on the diet breadth of the herbivore. When plants are attacked by specialist herbivores, avoidance alleles may actually lower the mean fitness of the plant population. This reduction in mean fitness occurs because, when an avoidance allele is fixed in a population, plants experience the costs associated with that allele but no benefit. There is no benefit after an avoidance allele fixes because these alleles confer benefits only when more preferred plant genotypes (i.e., initial genotypes) are present. In contrast, when avoidance alleles fix in response to an herbivore that is not a strict specialist, these same alleles may increase the mean fitness of the plant population in which they evolve. This occurs because avoidance alleles cause a shift

in herbivore pressure toward the alternative herbivore host(s). Because avoidance alleles are more likely to fix than antibiosis alleles and because avoidance traits cause shifts in herbivore pressures between hosts, it is possible that avoidance traits are more important in causing host shifts than antibiosis traits. These results may help to explain the observation that the host range of many insects appears to be determined largely by herbivore behavior rather than performance (Futuyma 1983).

Finally, an antibiotic allele that is able to invade a population will reduce the size of the herbivore population, resulting in higher mean fitness in the plant population. Similar to avoidance, antibiosis alleles are also expected to affect herbivore pressure in plant populations (i.e., species) in which the antibiosis does not evolve. Unlike avoidance, however, antibiosis alleles are expected to reduce rather than increase the herbivore pressure experienced by other host plants. Also unlike avoidance, the benefits associated with antibiosis are not lost after these alleles become fixed.

Tolerance, avoidance, and antibiosis are all mechanisms plants may evolve to minimize the potentially detrimental effect of herbivores. The model and analyses show that these traits are not equivalent in the evolutionary dynamics they exhibit or in their effects on plant and herbivore communities. These results suggest that a fuller understanding of plant-herbivore interactions will require empirical studies to determine the relative importance of tolerance, avoidance, and antibiosis in the response of plants to selection exerted by herbivores and to determine how these traits affect plant and herbivore population dynamics.

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APPENDIX

Effect Defense Traits Evolving in One Plant Population Have on Herbivore Loads in a Second Plant Population

In this appendix, I present an equation that describes the herbivore load experienced by individuals in population 2. I then show that when tolerance (T), avoidance (AV), and antibiosis (AB) alleles invade population 1, they cause no change, an increase, and a decrease, respectively, in herbivore loads in population 2. Herbrivore loads experienced by individuals in population 2 can be described by modifying equation (2) to

$$D_2 = h(H_0, \alpha, p_{AB})\rho_2(\nu, \eta_X, \eta_2, p_X) = H_0(1 - \alpha p_{AB})\beta_2\left[\frac{\eta_2}{\left(\nu \sum_{X}^{T, AV, AB, I} p_X\eta_X\right) + (1 - \nu)\eta_2}\right]. \quad \text{(A1)}$$

The herbivore load experienced in population 2 under initial conditions can be calculated by evaluating this equation with $p_I = 1$, which is equivalent to

$$H_0\beta_2\left[\frac{\eta_2}{\nu + (1 - \nu)\eta_2}\right]. \quad \text{(A2)}$$

Since it is assumed that $\eta_I = \eta_T$, equation (A2) also describes the herbivore load experienced in population 2 after the T allele invades and goes to fixation. In contrast, if an AV allele fixes in population 1, then herbivore loads in population 2 are equal to

$$H_0\beta_2\left[\frac{\eta_2}{\nu\eta_{AV} + (1 - \nu)\eta_2}\right]. \quad \text{(A3)}$$

Since , by definition, $\eta_{AV} < 1$, (A3) > (A2) and herbivore loads experienced in population 2 will increase as a result of the AV allele invading population 1. Finally, when an AB allele invades population 1, herbivore loads in population 2 are equal to

$$H_0(1 - \alpha p_{AB})\beta_2\left[\frac{\eta_2}{\nu + (1 - \nu)\eta_2}\right]. \quad \text{(A4)}$$

Since $\alpha p_{AB} > 0$, (A4) < (A2), which indicates that an AB allele invading population 1 reduces herbivore loads experienced by population 2.

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