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Will plant vigor and tolerance be genetically correlated? Effects of intrinsic growth rate and self-limitation on regrowth

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Abstract. Plants are known to maintain fitness despite herbivore attack by a variety of damage-induced mechanisms. These mechanisms are said to confer tolerance, which can be measured as the slope of fitness over the proportion of plant biomass removed by herbivore damage. It was recently supposed by Stowe *et al.* (2000) that another plant property, general vigor, has little effect on tolerance. We developed simple models of annual monocarpic plants to determine if a genetic change in components of growth vigor will also change the fitness reaction to damage. We examined the impact of intrinsic growth rate on the tolerance reaction norm slope assuming plants grow geometrically, i.e., without self-limitation. In this case an increase in intrinsic growth rate decreases tolerance (the reaction norm slope becomes more negative). A logistic growth model was used to examine the impact of self-limiting growth on the relationship between intrinsic growth rate and the tolerance reaction norm slope. With self-limitation, the relationship is sensitive to the timing of attack. When attack is early and there is time for regrowth, increasing growth rate increases tolerance (slope becomes less negative). The time limitations imposed by late attack prevent appreciable regrowth and induce a negative relationship between growth rate and tolerance. In neither of these simple cases will the correlation between vigor and tolerance constrain selection on either trait. However, a positive correlation between growth rate and self-limitation will favor fast growth/strong self-limitation in a high-damage environment, but slow growth/weak self-limitation in a low-damage environment. Thus, fundamental growth rules that determine vigor have constitutive effects on tolerance. The net costs and benefits of damage-induced tolerance mechanisms will thus be influenced by the background imposed by fundamental growth rules.

Key words: growth rate, herbivory, plant defense, tolerance

Introduction

Tolerance of herbivory can be defined as the ability of a plant genotype to maintain fitness despite damage. A concept that is sometimes associated with tolerance is that of 'general vigor,' which is the ability of a genotype to perform

well in multiple environments. Vigor manifests as a positive genotypic correlation of fitness across environments (Futuyma and Philippi, 1987). To better understand how tolerance evolves it is important to know if and how it might genetically correlate to vigor. If a correlation exists, direct selection to increase vigor will cause an indirect evolutionary response in tolerance even in populations that never experience herbivory. Equally important, it has been supposed that a negative correlation between growth vigor (in the absence of damage) and tolerance is a sign that tolerance comes at a fitness cost (Simms and Triplett, 1994; Abrahamson and Weis, 1997; Mauricio *et al*., 1997; Tiffen and Rausher, 1999).

Here we present simple models of plant growth to see how tolerance might change as selection acts on components of plant vigor. More specifically, we ask if changes in the genetic 'rules' that govern plant growth in the absence of damage pleiotropically change regrowth ability. Understanding this relationship could aid in interpreting data for signs of special recovery mechanisms and their costs.

To measure tolerance, one subjects clonal replicates of a genotype (or sibs within a family) to an array of damage levels and then measures one or more fitness components on these plants. Regression analysis can be used to parameterize an equation that describes fitness as a function of the proportion of plant mass lost to damage (Simms and Triplett, 1994; Abrahamson and Weis, 1997; Mauricio *et al*., 1997; Stowe, 1998; Tiffin and Rausher, 1999; Hochwender *et al*., 2000). This equation describes the genotype's reaction norm of fitness to the environmental variable 'damage'. The degree of tolerance is measured by the parameters of the function that describe slope and curvature. When fitness does not vary with damage level, tolerance is complete. A downward trend in fitness with increasing damage indicates incomplete tolerance.

Various mechanisms have been proposed to underlie herbivory tolerance (Stowe *et al*., 2000). These include initiation of dormant meristems (Islam and Crawley, 1983; Benner, 1988; Geber, 1990; Bergelson and Crawley, 1992; Rosenthal and Welter, 1995; Lennartsson *et al*., 1997; Juenger and Bergelson, 2000), mobilization of stored resources (Hendrix, 1979; Paige and Whitham, 1987; Hendrix and Trapp, 1989; Bilbrough and Richards, 1993; Paige, 1992, 1999; Hochwender *et al*., 2000), and damaged–induced upregulation of photosynthesis (Wareing *et al*., 1968; Nowak and Caldwell, 1984; Marby and Wayne, 1997; Meyer, 1998). Traits such as these involve the plant's active responses to damage, and as such are not directly involved in normal growth (although the cost of maintaining them can negatively impact growth). General vigor, on the other hand, will include a variety of metabolic and developmental mechanisms to promote growth in the absence of damage.

Will 'normal' growth mechanisms also influence the slope and curvature of the tolerance function? Stowe *et al*. (2000) assert that by defining herbivory

tolerance as the slope of the fitness reaction to herbivory, the effect of general vigor on tolerance is confined to raising or lowering the intercept, or the mean height, of the reaction norm. If the tolerance reaction norm slope is insensitive to vigor, there will be no genetic correlation between the two traits, and each evolves independently of the other. One implication of this view is that tolerance evolves only through damage-induced mechanisms of re-growth. Before we can fully evaluate the contributions of special mechanisms for re-growth, we need a clear notion of how basic growth patterns can influence tolerance.

At the most basic level, two factors can enhance plant vigor: an increase in intrinsic growth rate, and the ability to sustain that growth rate throughout the growing season. Intrinsic growth rate, which we symbolize as ρ, is the potential mass, M, added per unit mass per unit time. When plants are growing at their full potential, the relative growth rate ($dM/dt \times 1/M$), will equal ρ (Blackman, 1919). If this maximum relative growth rate is maintained over the season, growth is exponential (Fig. 1A). When plants are very small, they generally will grow close to this maximum rate, but as they get larger, relative growth rate (hereafter RGR) tends to decline. Declining RGR is caused by a variety of factors: these include self-shading (Horn, 1971; Honda and Fisher, 1978; Niklas, 1988; Ackerly and Bazzaz, 1995; Givnish, 1995), declining nitrogen content and photosynthetic capacity of older leaves (Field, 1983; Hirose and Werger, 1987), or increasing demand for allocation of biomass to non-photosynthesizing support structures (McMahon, 1973; Augspurger, 1984; Brokaw, 1987; Yamamura, 1997; Ackerly, 1999). A decline in RGR caused by these constraints on the plant's internal economy can be called self-limitation and leads to a logistic-like plant growth curve (Fig. 1B). The prevention or diminution of self-limitation is a second component of general vigor.

We used basic growth equations to see how genetic changes in the intrinsic growth rate and in self-limitation change the tolerance reaction norm. We assume that damage reduces plant size, but does not induce changes in any other factor that affects RGR. The models are not intended to predict the optimal degree of vigor or tolerance. Rather they serve as a guide in understanding how a change in plant growth rules can by itself change tolerance reaction norm slope and curvature. In this way, we can see if tolerance can evolve as a correlated trait when selection acts on fundamental growth parameters.

Model construction and results

We evaluated the effect of damage on the growth trajectory of plants following one of two very simple growth models, one for geometric (which is equivalent to exponential) and the other for logistic growth. Self-limitation on growth is absent in the first of these models, and present in the second. The models

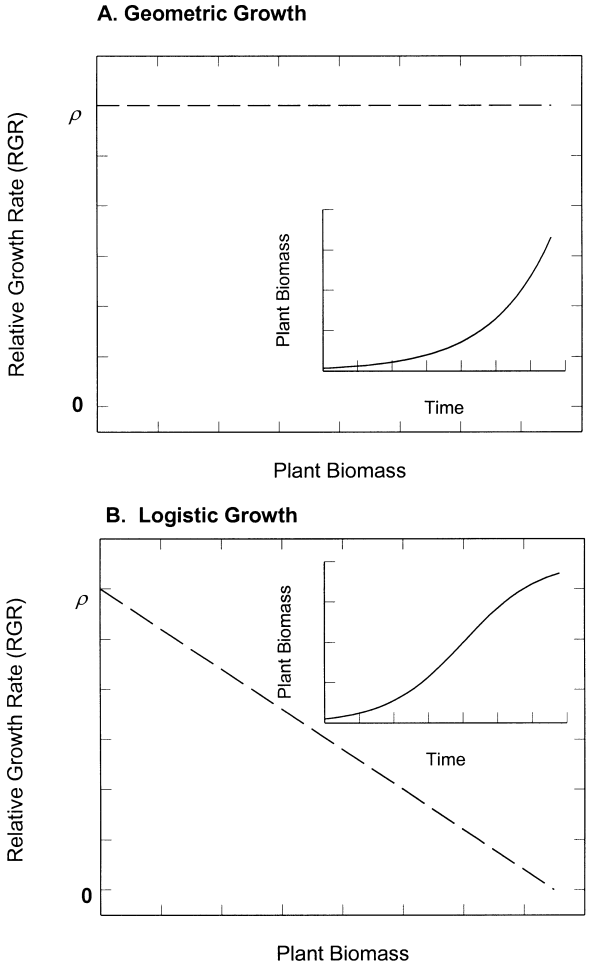

Figure 1. The change in plant relative growth rate as a function of plant size. A) With geometric growth, RGR remains constant and equal to the intrinsic growth rate (ρ). B) With logistic growth, relative growth rate declines linearly with size. The reduction in growth rate of large plants can be caused by factors such as self-shading or increased proportionate allocation to non-productive support tissue. Both damaged and undamaged clones of a given genotype show the same relationship between relative growth rate and size.

emulate the growth of an annual monocarpic plant whose reproduction is initiated at the close of the growing season by external cues that portend a lethal environmental event such as frost or drought. The basic equations of these models are adapted from theoretical studies on the evolution of plant life histories and on the evolution of defense (e.g., Coley *et al.*, 1985; Iwasa and Cohen, 1989; Amir and Cohen, 1990; Iwasa and Kubo, 1997; De Jong and Van der Meijden, 2000). Models follow the growth of productive tissues in an

annual plant over the course of the growing season. By productive tissue, we mean photosynthesizing leaves and the roots and vascular tissue needed to supply them with nutrients and water. We assume no storage and an allocation to structural support tissue that is small enough to ignore. The model plants act like clusters of leaves with minimal stem and root.

We modeled growth when a single herbivory event reduces the productive tissue mass at a specific point in the growing season. After damage, plants regrow by exactly the same growth rules as before herbivory. In other words, we examined how changing intrinsic growth rate and self-limitation affects tolerance in the absence of any damage-induced change in the growth rules.

We simplified assumptions about flowering and seed set to focus on growth rate and self-limitation. We assume that all seeds result from self-pollination, so that maternal and paternal components of fitness are perfectly correlated. We make the additional assumption that a fixed proportion of productive biomass is reallocated to seed production at the end of the season.

Effect of intrinsic growth rate on tolerance without self-limitation

The geometric model assumes that plants grow at a constant rate until they receive a flowering cue. Geometric growth is a discrete time version of exponential models for plant growth (see Blackman, 1919; Hilbert *et al.*, 1981; De Jong and Van der Meijden, 2000). Assume that plant mass increases according to the recursion equation,

$$M_{t+1} = M_t + \rho M_t = M_t(1 + \rho) \tag{1}$$

where M_t stands for plant biomass at time t and ρ is the intrinsic growth rate. Size of the plant at the end of the growth season ($t = n$) is thus

$$M_n = M_0(1 + \rho)^n. \tag{2}$$

In this discrete time model, RGR is defined as $\Delta M/M_t$, that is, the change in size over a time interval divided by the size at the beginning of the interval. An essential feature of geometric growth is that RGR is independent of plant size and at all times is equal to ρ (Fig. 1). Metabolically, ρ is related to net photosynthetic rate.

When herbivores attack, they remove proportion D of productive biomass. After attack, genotypes resume growth at their characteristic intrinsic rate, ρ. The final size of a damaged plant (M'_n) is thus determined by size at time of attack ($t = h$), intensity of damage, and growth rate, such that

$$M'_n = (1 - D)M_h(1 + \rho)^{n-h}. \tag{3}$$

Size at time of attack, M_h, will equal $M_0(1 + \rho)^h$, so that

$$M'_n = (1 - D)M_0(1 + \rho)^h(1 + \rho)^{n-h} = (1 - D)M_0(1 + \rho)^n. \tag{4}$$

This equation shows that when initial size and season length are held constant, final size at any given damage level will increase with increased intrinsic growth rate. Note that when growth is geometric the timing of damage does not affect final size.

To illustrate the effects of herbivory on final size we simulated genotypes that differed in growth rate in a season of 30 time units, with damage imposed at $t = 20$. Figure 2 illustrates growth trajectories for three different growth rate

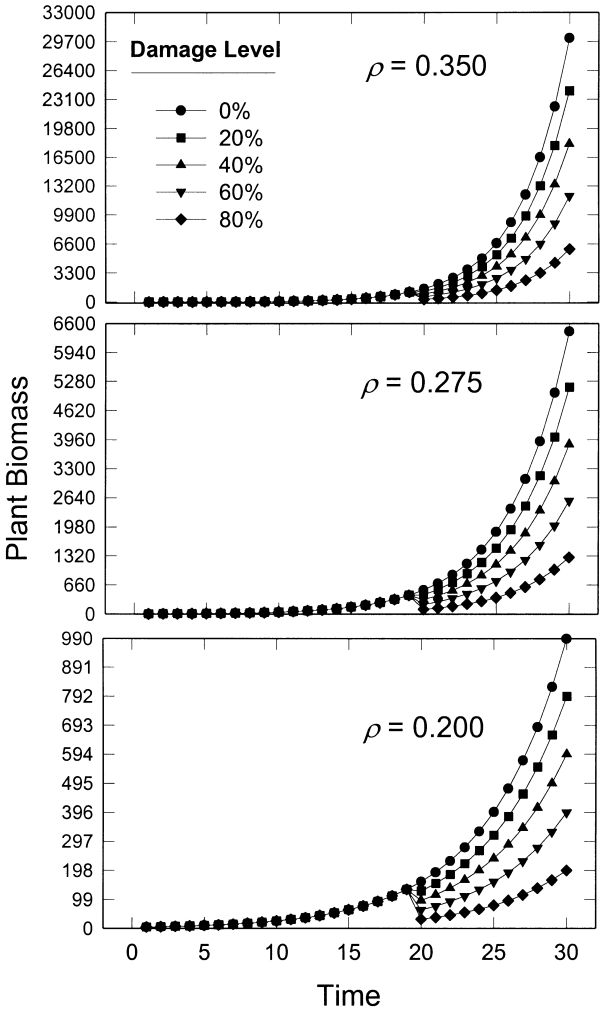

Figure 2. Each panel depicts geometric plant growth through time of replicate individuals of the same growth rate genotype, as affected by differing proportions of tissue removed by herbivory. Note the difference in scale on the three *y*-axes.

genotypes exposed to a gradient of damage. Increasing the growth rate increases final size, as would be expected for a season of set length.

Figure 3 shows that genotypes with high growth rates are less tolerant of damage than those with low rates, i.e., the slope of the tolerance function is more negative. Following from Equation (4), the slope of the tolerance reaction norm for any genotype is $-DM_n$. Given that final size, M_n, is a function of intrinsic growth rate, ρ, growth vigor has a scaling effect on the tolerance reaction norm slope. Vigorous growers lose more fitness on an absolute scale because they have more fitness to lose.

Removing the scaling effect (all fitness values within a genotype made proportionate to fitness in the absence of damage) eliminates genotypic differences (Fig. 4). Within a genotype, all replicates are the same size at the time of attack, and all re-grow at the same ρ. Therefore, according to Equation (3), the final size of damaged and undamaged plants is due only to differences in D. Formally, the final size of a defoliated plant, relative to an undamaged one will follow the ratio

$$M'_n/M_n = (1-D)M_h(1+\rho)^{n-h}/M_h(1+\rho)^{n-h} = (1-D). \quad (5)$$

For example, a 20% loss in biomass to herbivory produces a 20% reduction in fitness, making the fitness ratio 0.80.

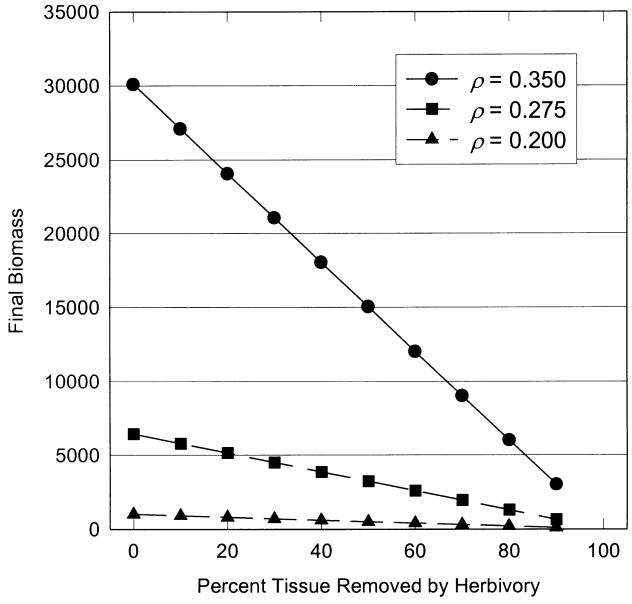

Figure 3. Norms of reaction of plant fitness to a damage gradient for three growth-rate genotypes that all express geometric growth. Damage is scaled as proportion of tissue lost to herbivory.

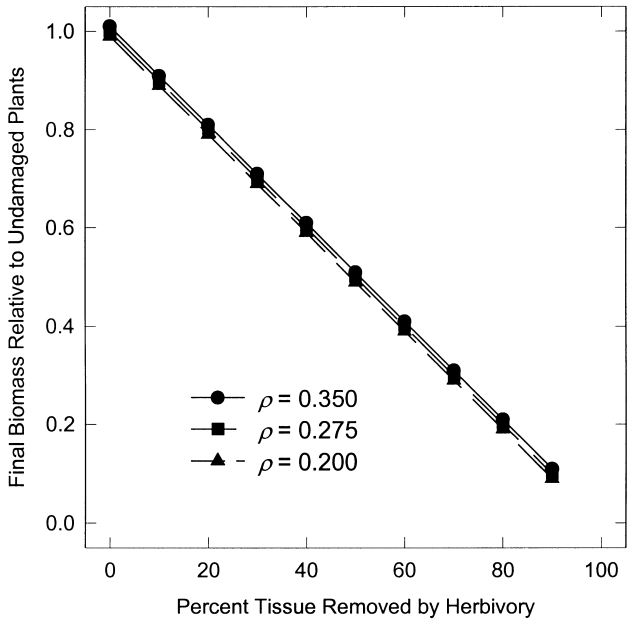

Figure 4. Norms of reaction of relative plant fitness to a damage gradient for the same three growth-rate genotypes portrayed in Figures 2 and 3. Here, fitness of each genotype is standardized to the maximum it can achieve in the absence of damage. Damage is scaled as proportion of tissue lost.

In summary, intrinsic growth rate affects the tolerance reaction norm in two ways when growth is geometric (Fig. 3). Increasing ρ raises the intercept of the fitness function and it also decreases the slope (makes it more steeply negative). That is, as this component of vigor increases, tolerance decreases, creating a negative genetic correlation between the two traits. For the geometric case, we can reject the conjecture by Stowe *et al*. (2000) that a change in growth vigor will affect only the reaction norm intercept. Next, we consider the impact of intrinsic growth rate when plants are self-limited.

Effect of intrinsic growth rate on tolerance under self-limitation

Few, if any, plants maintain geometric growth throughout their vegetative phase. Our second model represents the frequently observed case of a sigmoid plant growth curve (Hunt, 1982). When the plant is small, its RGR is close to its geometric potential. However, as the plant grows its RGR (that is, $\Delta M/M_t$) declines.

The model assumes logistic growth, i.e., a linear decline in relative growth rate with size (Fig. 1B). Sigmoid growth curves will also result from growth rate declines that are non-linear and that are functions of age rather than size.

In the logistic formulation we employ, the decline in RGR can be readily interpreted as the result of self shading (Iwasa and Cohen, 1989; Iwasa and Kubo, 1997; Weis and Hochberg, 2000) or other forms of local resource depletion which are a function of plant size.

Logistic growth is modeled by the recursion equation,

$$M_{t+1} = (M_t + \rho M_t)/(1 + \theta M_t) = \{M_t(1 + \rho)\}/(1 + \theta M_t) \qquad (6)$$

where M, t and ρ are as above, and θ is a constant describing the effects of self-limiting growth. Metabolically, θ reflects the respiratory costs caused by reduced photosynthetic activity in some leaves. Self-shading or other types of local resource depletion can cause a decline in net photosynthesis. When θ is zero the logistic model reduces to the geometric. In a season of sufficient length plants reach a final size of ρ/θ, that is, the size at which respiration balances photosynthesis.

As for the geometric case, the final size of a damaged plant under logistic growth rules depends on its size at time of attack, the proportion of damage, and intrinsic growth rate. But the self-limitation constant enters the equation, such that

$$M'_n = \{(1 - D)M_h(1 + \rho)^{n-h}\} / \prod_{t=h}^{n}(1 + \theta M_t) \qquad (7)$$

where Π stands for the product over all time intervals from the time of herbivory, h, to the end of the season, n. The effect of self-limitation during the post-attack period makes final size dependent on the amount of time available for recovery. Since early attack allows more time for recovery, timing of attack will affect final size. In contrast, timing has no effect in the geometric case.

We illustrate the effect of damage on simulated growth curves in Figure 5. The same three growth rates were used here as for the geometric case. As before, the season lasted 30 time units, with herbivory imposed at $t = 20$. We set θ to 0.001 for all cases. Inspection of these growth curves shows that RGR increases immediately after damage but again declines as plants regain size. Intrinsic growth rate, ρ, is unchanged by damage. However, damage reduces size and thus relieves plants from self-limitation; say, as plants are relieved from self-shading. Re-growth of leaves re-imposes self-shading and causes relative growth rates to decline again. The temporary boost in RGR may be sufficient to allow recovery from modest damage levels.

The reaction of final size to the damage gradient is plotted in Figure 6. Three differences from the geometric model are notable. First, the reaction norms are not linear; increasing damage from 0 to 10% has virtually no effect on fitness, whereas increasing damage from 80 to 90% has a large adverse effect. The second important difference is that tolerance tends to increase with growth rate – the reverse of the geometric case. Increased growth rate causes a flattening of

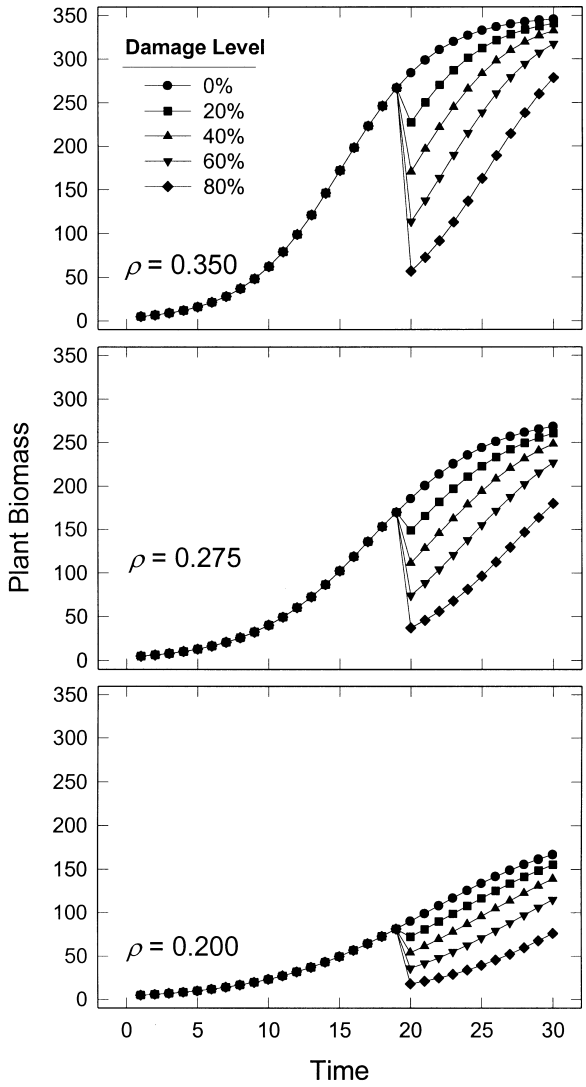

Figure 5. Each panel depicts logistic plant growth through time of replicate individuals of the same growth-rate genotype, as affected by differing proportions of tissue removed by herbivory.

the tolerance function at the lower damage levels. (In the extreme case, 100% damage will reduce final size to zero regardless of growth rate since, by definition, the plants are killed.) In this example, the genotype with the highest growth rate lost 20 biomass units (compared to the undamaged condition) when afflicted with 50% damage, whereas 40 units were lost by the slowest genotype at that damage level. Fast-growing plants approach their size plateau by the end of the season even when moderately damaged, whereas slow

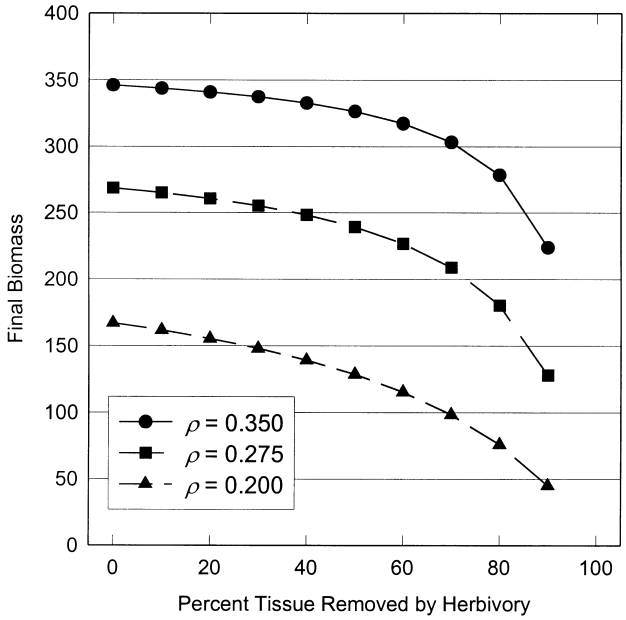

Figure 6. Norms of reaction of plant fitness to a damage gradient for three growth-rate genotypes that all express logistic growth. Herbivore attack occurs at time interval 20. Damage is scaled as proportion of tissue lost to herbivory.

growing plants with moderate damage are still well short of their asymptotic size at season's end.

The time available for recovery changes the tolerance reaction norm slope and curvature when growth is self-limiting (Fig. 7). It is crucial to note here that if the growing season is sufficiently long, and there is no further damage, all plants reach the asymptotic size of ρ/θ for any damage level less than 100%. When growing season has a limited duration intrinsic growth rate can determine if a plant has time to recover. When ρ is high, early-damaged plants may have enough time to catch up with their undamaged counterparts. This makes reaction norms flatter over low damage levels (although they take a sharp downturn as damage approaches 100%). However, if the intrinsic growth rate is slow, time is more limiting on recovery, and tolerance reaction norms are steeper. High growth rates do not flatten tolerance reaction norms when attack is very late. At the extreme, when attack occurs at the very end of the season, Equation (7) reduces approximately to the geometric model, i.e., $M'_n = (1 - D)M_h$. The later that attack occurs in the season, the more that an increase in ρ will decrease (make more negative) the tolerance reaction norm slope.

Logistic growth breaks the linear scaling effect of growth rate. This causes fast-growing plants to suffer proportionately less from a given amount of damage than do slow growers (Fig. 8), provided attack does not occur at the

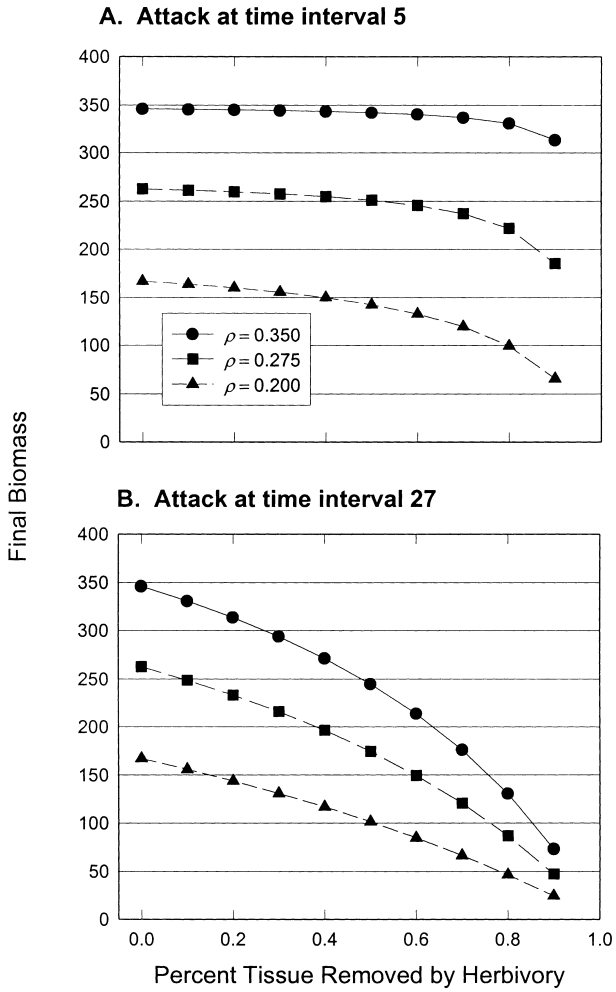

Figure 7. Norms of reaction of plant fitness to a damage gradient for three growth-rate genotypes that all express logistic growth. Same growth rates as in Figure 6, but, A) herbivore attack at time 3 and B) attack at time 27.

end of the season. Tolerance functions are more nearly parallel in the logistic case (Fig. 6) and this indicates that tolerance is less sensitive to growth rate than with geometric growth. With logistic-like growth and early attack, the assertion by Stowe *et al.* (2000) is approximately true.

Tolerance when intrinsic growth rate and self-limitation are correlated

The relationship between tolerance and general vigor can be radically modified by a positive genetic correlation between growth rate and self-limitation. This

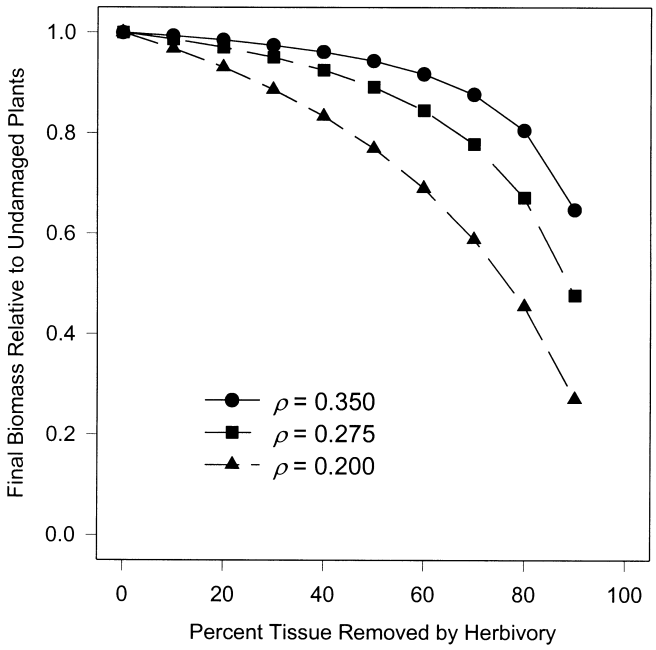

Figure 8. Norms of reaction of relative plant fitness to a damage gradient for the same three growth-rate genotypes portrayed in Figures 5 and 6. Here, fitness of each genotype is standardized to the maximum it can achieve in the absence of damage. Damage is scaled as proportion of tissue lost.

type of correlation constrains plants either to grow quickly to a small plateau size, or slowly to a larger plateau. As a possible example, consider a gene that controls water use. Plants that are extravagant in their water use will be able to photosynthesize at a high rate when they are small. They may consume more water per unit leaf mass, but because total mass is small, they extract only a small portion of the available soil water. As they grow, however, water consumption comes into balance with water supply and growth stops. A genotype with more conservative water use will grow slower, but will be able to sustain that growth longer because water consumption and supply come into balance at a larger plant size. The tolerance reaction norms for these two genotypes could intersect (Fig. 9). The water-conservative genotype would achieve a larger size in a damage-free environment, but at high damage levels, its slow growth rate could preclude plants from approaching their plateau size in the time available for recovery. The water-extravagant genotype, by contrast, would plateau at a smaller size in the undamaged condition. If leaf mass is removed by herbivory, total water consumption will go down. The photosynthetic rates of the surviving leaves can increase because they are relieved

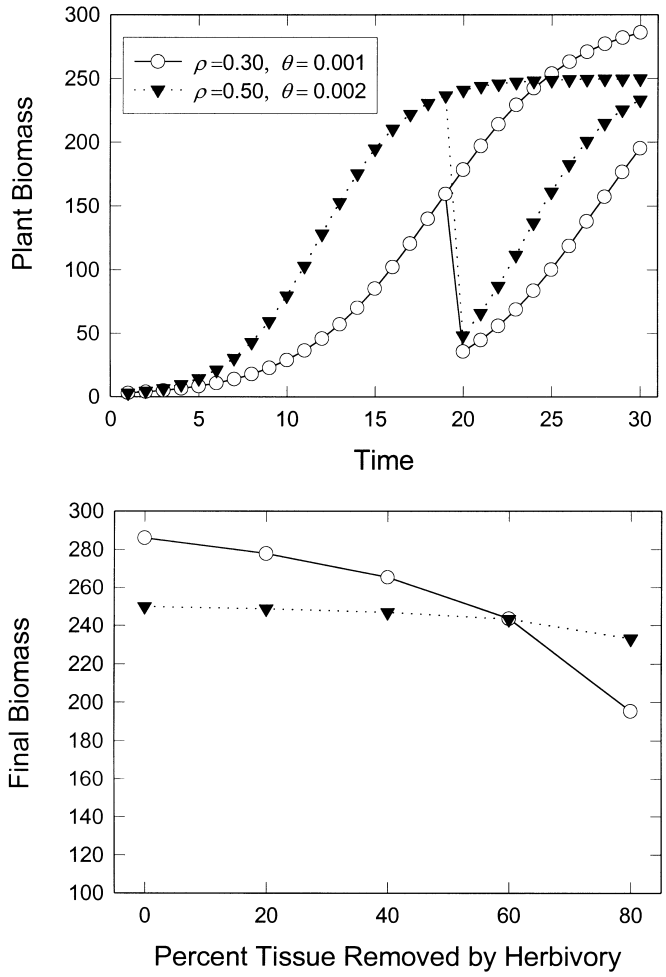

Figure 9. Damage levels can change the relative fitness of vigor genotypes when there is a positive genetic correlation between intrinsic growth rate and self-limitation. A) growth trajectories for a slow-growth/weak-limitation genotype and a fast-growth/strong-limitation genotype. Trajectories for zero or 80% damage shown. B) tolerance reaction norms for the two contrasting genotypes. The slow-growth/weak-limitation combination is favored in low-damage environments but the fast-growth/strong-limitation one is favored under high-damage conditions.

from water limitation. Re-growth will continue at a fast rate until consumption balances supply, or until the season ends. The trade-off between intrinsic growth rate and self-limitation radically changes relative fitness of the two genotypes between low damage and high damage environments (Fig. 9). When damage is absent, it is better to grow slowly to the larger size. When damage is severe, the re-growth benefits conferred by a high intrinsic growth rate outweigh the costs of self-limitation. Thus, when the components of general vigor

have conflicting effects on fitness, their influence on tolerance can be decisive in determining the course of adaptation.

Discussion

The simple models presented here show that two components contributing to plant vigor, the intrinsic growth rate and the degree of self-limited growth, can affect tolerance of herbivory. Stowe *et al.* (2000) asserted that changes in growth rate would change the intercept of the tolerance reaction norm but have scant effect on its slope or shape. Our results show that this assertion is approximately true in some circumstances, but not all. Here we discuss first the correlations among growth rate, self-limitation and the tolerance reaction norm as revealed in these models, and then how less stringent assumptions can affect our conclusions.

The correlation between vigor and tolerance

It is reasonable to suspect that the intrinsic growth rate of a plant also affects the rate at which it re-grows from damage (Coley *et al.*, 1985; De Jong and Van der Meijden, 2000). For instance, Sun (1992) compared rate of recovery from trampling in eight pasture species and found that it correlated with growth rate in the un-trampled condition. In a similar way growth rate could influence the fitness reaction norm with respect to herbivore damage. Selection for faster growth will change the fitness reaction to damage by elevating the intercept of the function. There is no mathematical necessity that factors affecting the intercept of a reaction norm also affect its slope (De Jong, 1990), but they may co-vary for functional reasons. We conclude that this functional link exists for the tolerance reaction norm.

If plants could grow at their maximum rate indefinitely, the relationship between proportion of tissue lost to herbivory and fitness would be linear. The faster the growth rate, the steeper this relationship (Fig. 3), which would create a negative genetic correlation between growth rate and tolerance. Increased growth rate would increase general vigor, and thus improve performance at all damage levels. Hence, selection would favor increased growth rate despite the loss of tolerance.

Self-limitation generally prevents maximum growth rates from being sustained (Hunt, 1982). Comparison between Figures 2 and 5 show that final size for a given growth rate genotype can be reduced by orders of magnitude by introducing a term for self-limiting growth. Self-limitation alters the tolerance reaction norm (compare Figs 3 and 6) by inducing a curvature. Low levels of damage have proportionately less impact on final size than higher levels of

damage (see Osterheld, 1992). This type of curvature could be expected if special mechanisms for compensatory growth were induced by damage (McNaughton, 1979; Hilbert *et al.*, 1981). However, the logistic model shows that fundamental plant growth rules also cause a curvature in the tolerance reaction norm. The decelerating RGR of intact plants allows moderately damaged plants to catch up. Compensatory mechanisms, when they exist, act on top of this fundamental pattern.

When growth is self-limiting, an increase in intrinsic growth rate tends to increase both vigor and tolerance (Fig. 6). Higher intrinsic growth rate will let a damaged plant achieve more re-growth in the time available. Early attack affords more time for recovery, and so may cause little loss of fitness. Late attack, in contrast, may offer no time for recovery, in which case final size becomes a linear function of the proportion of tissue removed by damage (just as in the geometric case). Thus, the genetic correlation between intrinsic growth rate and tolerance slope will change over the season from positive to negative. Any factor selecting for faster intrinsic growth rate will tend to increase early tolerance and decrease late tolerance.

The selective advantage of a change in the components of vigor will be composed of the direct effect that each has on fitness regardless of damage, and the indirect fitness effect through tolerance. The growth models suggest that as long as growth rate and self-limitation are not themselves genetically correlated, the direct fitness effects will outweigh the indirect effects. Comparing reaction norms within Figures 3 or 5, an increase in growth rate leads to increased fitness at all damage levels – the advantage of faster growth in high damage environments is quite similar to that in low damage environments. Comparison between Figures 3 and 5 shows that release from self-limitation likewise provides a fitness advantage at all herbivory levels. Tolerance reaction norms do not cross in these cases, and so there can be a single genotype that is most fit at all points along the herbivore damage gradient. Selection in environments at one damage level will increase the ability to grow across all damage levels. This has a counter-intuitive implication. The more the reaction norms resemble the geometric case, such as for late damage defoliation under logistic growth, the more that selection to increase vigor in high-damage environments can cause the evolution of lower tolerance.

A contrasting situation arises when intrinsic growth rate and self-limitation are positively genetically correlated. This correlation can cause the tolerance reaction norms to cross, so that some combinations of growth rate and self-limitation will be favored in low-damage environments, while other combinations are favored in high-damage environments (Fig. 9). In this situation herbivory can tip the selective balance in favor of growth rate over reduced self-limitation. Here the indirect fitness effects of the vigor components can become as strong as the direct effects.

Simplified models and complex realities

The growth models presented here are admittedly cartoon versions of plant growth. Nevertheless, simplified models of plant growth and herbivory have been useful in advancing our understanding of more complex realities (e.g. Hilbert *et al*., 1981; Coley *et al*., 1985; Osterheld and McNaughton, 1991; Osterheld, 1992; De Jong and Van der Meijden, 2000). Our goal was to see if there are any fundamental reasons to suspect that changes in general growth vigor can by themselves change the tolerance reaction norm slope or curvature. To do this we stripped growth rules down to their simplest form. Here we discuss a few ways in which relaxing these simplifications may affect the predicted relationship between vigor and tolerance. Will the complexities of plant growth make tolerance reaction norms more closely resemble the geometric or the logistic extremes we present? Throughout this section, we make the realistic assumption that RGR is in some degree diminished as the plant grows (Hunt, 1982). We maintain our focus on the effects of vigor on tolerance by assuming no damage-induced recovery mechanisms.

A primary assumption is that herbivores damage productive tissue (folivory). The influence of other kinds of damage may influence plant growth differently. Damage to vascular tissue can kill branches and the leaves they hold. In this case, the models should still be relevant. Other damage types can change RGR by means other than reducing the amount of productive tissue. Damage to active meristems temporarily removes resource sinks; RGR may temporarily decline while dormant meristems are activated. Meristem activation increases the capacity for re-growth in some cases (Islam and Crawley, 1983; Benner, 1988; Geber, 1990; Bergelson and Crawley, 1992; Rosenthal and Welter, 1995; Lennartsson *et al*., 1997; Juenger and Bergelson, 2000), and the altered crown structure caused by increased branching may also affect self-limitation. Attack by sap-sucking or by galling insects will reduce RGR by imposing new resource sinks, rather than by directly reducing the size of the production machinery. Attack by these enemies may not release plants from self-limitation unless they shed leaves to match the diminished supply of resources. Although these attack types change plant size and RGR in different ways, the models may be helpful in understanding tolerance against them. To the extent that damaged plants grow by the same rules as undamaged ones, final sizes will be affected by the elements in Equation (7): size after attack, intrinsic growth rate, self-limitation strength, and time remaining in the growing season.

We have assumed that fitness is directly proportional to final biomass. This isometric relationship makes fitness a linear function of size, with a zero intercept. If instead one assumes a minimum size to reproduce, the function remains linear but has a negative intercept. This type of relationship between

size and fitness gives the same reaction norm slope and curvature as the isometric case (although the intercept is lowered). One could also assume an exponential increase in fitness with size. If so, the loss of the first aliquot (say, 10%) of biomass would have a greater fitness effect than loss of the next 10%, and so on. We examined this kind of relationship using exponents less than two, that is, within the range for typical allometric relationships. With no or weak self-limitation an exponential relationship causes weakly concave tolerance reaction norms. When self-limitation is stronger reaction norms have a slightly steeper slope and weaker curvature. Thus, an allometric relationship between final size and fitness can shift reaction norms for early attack toward those observed for late attack. In turn this shift can weaken or reverse the positive correlation between vigor and tolerance.

Our most restrictive assumption concerns the morphology and life history embodied in Equations (1) and (6). We assume that allocation to storage and support tissue is small enough to ignore. Heavy allocation to these types of structures would lower the post-attack RGR in two ways. Removing leaf mass would lower net photosynthesis of the productive tissue without changing the respiratory demands of the non-productive tissue. If growth rules, including allocation patterns, are unchanged by attack, the RGR in productive tissue would not return all the way to the level predicted by the mass of the surviving leaves. This brings up another assumption, that all standing leaf biomass remains capable of photosynthesizing at maximum rate. Unless herbivores preferentially eat senescent leaves, the lower intrinsic growth rate of senescent leaves will limit post-attack RGR (Woledge, 1986; Gold and Caldwell, 1990) and make recovery more limited by time. Consequently, tolerance reaction norms would be more like those the model predicts for late attack (Fig. 7), in which tolerance is slightly decreased by higher intrinsic growth rates.

The model structure assumes that all productive parts are damaged proportionately, but in reality herbivores tend to prefer some plant parts to others. Such uneven damage should lower post-attack RGR. Leaf removal lowers the amount of photosynthesizing tissue without reducing the respiratory demands of non-photosynthesizing tissue. Root damage lowers water and nutrient supplies to leaves, causing their photosynthetic rate to drop in proportion to their respiratory needs. In either case, damage requires that a greater proportion of production be allocated to maintenance. Plants often respond to damage by shedding excess roots or leaves to maintain a balance between the two tissue types (e.g. Windel and Franz, 1979). One solution is to define damage as not only the tissue directly consumed by the herbivore, but also the tissue shed in response to consumption. An immediate shedding response would thus conform to the simplified models. A lagged shedding response would limit the time for regrowth and again make tolerance reaction norms for early attack more like those we predict for late attack.

We assumed that plants grow vegetatively, then convert biomass to propagules. This is an extreme form of the 'big-bang' strategy for monocarpic annuals (Amir and Cohen, 1990; Kozlowski, 1992). A more realistic assumption would be that the growth of reproductive structures is fueled in whole or in part by current photosynthesis rather than strictly by resorption and reallocation from vegetative structures. When current photosynthesis is used for reproduction, plants will reduce or curtail allocation to new productive tissue at flowering. This strategy will make the impact of vigor on tolerance sensitive to the allocation rules that govern flower and seed production. It also makes tolerance sensitive to flowering date, both in relation to the timing of season's end and the timing of attack.

If damage occurs before flowering, while plants are growing close to their intrinsic rate, tolerance reaction norms are likely to resemble those for the geometric case. Damage that occurs after the start of flowering can have a variety of effects, depending on the rules that govern the allocation trade-off between growth of new productive tissue and reproduction. The tolerance reaction norm to damage after anthesis will also depend on whether flowering starts when plants reach a particular size, or at a particular time. When allocation rules are inflexible (e.g., flowering occurs at a set time and the proportion of photosynthate devoted to reproduction is constant or increases with time) release from self-limitation may have little effect on RGR, and reaction norms will always resemble those we predict for late attack. If the rules are flexible (e.g., initiation of flowering and the allocation to reproduction are determined by vegetative size) reaction norms could resemble those we predict for middle to late attack, depending on the timing of attack.

Conclusions

We show, in principal, that an evolutionary change in plant general vigor in growth can alter the fitness reaction to damage. This result reinforces the argument that tolerance is a complex property of genotypes composed of constitutive and damage-induced components. There has been much interest in damage-induced recovery mechanisms (Trumble *et al*., 1993; Rosenthal and Kotanen, 1994; Strauss and Agrawal, 1999; Stowe *et al*., 2000). Indeed, these may be the most important adaptive pathways for plant populations faced with predictable and intense herbivory. However, the selective value of a gene for a damage-induced reaction will depend on how it enhances or antagonizes the fitness effects of the underlying constitutive rules of growth.

Our models predict that two extreme cases will produce correlations between growth vigor and tolerance with opposing signs – negative for geometric growth and a tendency for positive with logistic growth. These contrasting

correlations could affect how costs constrain the evolution of damage-induced recovery mechanisms. Consider a hypothetical induced recovery mechanism that imposes a cost on intrinsic growth rate. If fundamental growth rules resemble the logistic model, this cost would reduce both general vigor and constitutive tolerance of the inducible genotype. Thus, for a given cost, the benefit of an induced recovery mechanism would need to overcome the fitness lost by a reduction in fundamental growth rate and its concomitant reduction in constitutive tolerance. In contrast, as fundamental growth rules approach the geometric case, an induced recovery mechanism can be favored at a lesser benefit/cost ratio.

Plants do not operate by the extreme rules in these models. Plant RGR slows with size, and age, and plants seldom reproduce solely by reallocating resources from productive to reproductive structures. However, our consideration of model assumptions suggests that more realistic growth rules will yield tolerance reaction norms that resemble the late-attack logistic case (Fig. 7B). Of the simulations we present, this case reveals the weakest response of reaction norm slope to intrinsic growth rate. Thus, while tolerance reaction norm slopes and curvatures are correlated with vigor in the two extreme growth models explored here, more realistic growth rules probably lead to substantially weaker vigor–tolerance correlations. Hence, the assertion by Stowe *et al.* (2000) that tolerance evolves independently of general plant vigor, while not inevitable, may be approximately correct under many natural conditions.

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References

Abrahamson, W.G. and Weis, A.E. (1997) *Evolutionary Ecology across Three Trophic Levels: Goldenrods, Gallmakers, and Natural Enemies*. Princeton University Press, Princeton, NJ.

Ackerly, D. (1999) Self-shading, carbon gain and leaf dynamics: a test of alternative optimality models. *Oecologia* **119**, 300–310.

Ackerly, D.D. and Bazzaz, F.A. (1995) Leaf dynamics, self-shading and carbon gain in seedlings of a tropical pioneer tree. *Oecologia* **101**, 289–298.

Amir, S. and Cohen, D. (1990) Optimal reproductive effort and the timing of reproduction of annual plants in a randomly varying environment. *J. Theor. Biol.* **147**, 17–42.

Augspurger, C.K. (1984) Light requirements of neotropical tree seedlings: a comparative study of growth and survival. *J. Ecol* **72**, 777–795.
Belsky, A.J. (1986) Does herbivory benefit plants? A review of the evidence. *Am. Nat.* **127**, 870–892.
Benner, B.L. (1988) Effects of apex removal and nutrient supplementation on branching and seed production in *Thlaspi arvense* (Brassicaceae). *Am. J. Bot.* **75**, 645–651.
Bergelson, J. and Crawley, M.J. (1992) The effects of grazing on the performance of individuals and populations of scarlet gilia, *Ipomopsis aggregata*. *Oecologia* **90**, 435–441.
Bilbrough, C.J. and Richards, J.H. (1993) Growth of sagebrush and brittlebush following simulated winter browsing: mechanisms of tolerance. *Ecology* **74**, 481–492.
Blackman, V.H. (1919) The compound interest law and plant growth. *Ann. Bot.* **33**, 353–360.
Brokaw, N.V.L. (1987) Gap-phase regeneration of three pioneer species in a topical forest. *J. Ecol.* **75**, 9–19.
Coley, P., Bryant, J.P. and Chapin III, F.S. (1985) Resource availability and plant antiherbivore defense. *Science* **230**, 895–899.
De Jong, G. (1990) Quantitative genetics of reaction norms. *J. Evolutionary Biol* **3**, 447–468.
De Jong, T. and Van der Meijden, E. (2000) On the correlation between allocation to defense and regrowth in plants. *Oikos* **88**, 503–508.
Field, C.B. (1983) Allocating leaf nitrogen for the maximization of carbon gain: leaf age as a control on the allocation program. *Oecologia* **56**, 341–347.
Futuyma, D.J. and Philippi, T.E. (1987) Genetic variation and covariation in responses to host plants by *Alsophila pometaria* (Lepidoptera: Geometridae). *Evolution* **41**, 269–279.
Geber, M.A. (1990) The cost of meristem limitation in *Polygonum arenastrum*: negative genetic correlations between fecundity and growth. *Evolution* **44**, 799–819.
Givnish, T.J. (1995) Plant stems: biomechanical adaptation for energy capture and influence on species distributions. In B.L. Gartner (ed.) *Plant Stems: Physiology and Functional Morphology*. Academic Press, New York, NY, pp. 3–49.
Gold, W.G. and Caldwell, M.M. (1990) The effect of the spatial pattern of defoliation on regrowth of a tussock grass, III. Photosynthesis, canopy structure and light interception. *Oecologia* **82**, 12–17.
Hendrix, S.D. (1979) Compensatory reproduction in a biennial herb *Pastinaca sativa* following insect *Depressaria pastinacella* defloration. *Oecologia* **42**, 107–118.
Hendrix, S.D. and Trapp, E.J. (1989) Floral herbivory in *Pastinaca sativa*: do compensatory responses offset reductions in fitness? *Evolution* **43**, 891–895.
Hilbert, D.W., Swift, D.M., Deteling, J.K. and Dyer, M.I. (1981) Relative growth rates and the grazing optimization hypothesis. *Oecologia* **51**, 14–18.
Hirose, T. and Werger, M.J.A. (1987) Maximization of daily canopy photosynthesis with respect to the leaf nitrogen allocation pattern in the canopy. *Oecologia* **72**, 520–526.
Hochwender, C., Marquis, R. and Stowe, K. (2000) The potential for and constraints on the evolution of compensatory ability in *Asclepias syriaca*. *Oecologia* **122**, 361–370.
Honda, H. and Fisher, J.B. (1978) Tree branch angle: maximizing effective leaf area. *Science* **199**, 888–889.
Horn, H.S. (1971) *The Adaptive Geometry of Trees*. Princeton University Press, Princeton, NJ.
Hunt, R. (1982) *Plant Growth Curves: The Functional Approach to Plant Growth Analysis*. Edward Arnold, London, UK.
Islam, Z. and Crawley, M.J. (1983) Compensation and regrowth in ragwort (*Senecio jacobeae*) attacked by the cinnabar moth (*Tyria jacobeae*). *J. Ecol.* **2**, 828–843.
Iwasa, Y. and Cohen, D. (1989) Optimal growth schedule of a perennial plant. *Am. Nat.* **133**, 480–505.
Iwasa, Y. and Kubo, T. (1997) Optimal size of storage for recovery after unpredictable disturbances. *Evolutionary Ecol.* **11**, 41–65.
Juenger, T. and Bergelson, J. (2000) The evolution of compensation to herbivory in scarlet gilia, *Ipomopsis aggregata*: herbivore-imposed natural selection and the quantitative genetics of tolerance. *Evolution* **54**, 764–777.
Kozlowski, J. (1992) Optimal allocation of resources to growth and reproduction: implications for age and size at maturity. *Trends Ecol. Evol.* **7**, 15–19.

Krupnick, G.A., Weis, A.E. and Campbell, D.R. (1999) The consequences of floral herbivory for pollinator services to *Isomeris arborea*. *Ecology* **80**, 125–134.

Lennartson, T., Toumi, J. and Nilsson, P. (1997) Evidence for an evolutionary history of overcompensation in the grassland biennial *Gentianella campestris* (Gentianaceae). *Am. Nat.* **149**, 1147–1155.

Marby, C.M. and Wayne, P.W. (1997) Defoliation of the annual herb *Abutilon theophrasti*: mechanisms underlying reproductive compensation. *Oecologia* **111**, 225–232.

Mauricio, R., Rausher, M.D. and Burdick, D.S. (1997) Variation in the defense strategies of plants: Are resistance and tolerance mutually exclusive? *Ecology* **78**, 1301–1311.

McMahon, T. (1973) Size and shape in biology. *Science* **179**, 1201–1204.

Meyer, G.A. (1998) Patterns of defoliation and its effects on photosynthesis and growth of goldenrod. *Functional Ecol.* **12**, 270–279.

Niklas, K.J. (1988) The role of phyllotactic pattern as a ''developmental constraint'' on the interception of light by leaf surfaces. *Evolution* **42**, 1–16.

Nowak, R.S. and Caldwell, M.M. (1984) A test of compensatory photosynthesis in the field: implications for herbivory tolerance. *Oecologia* **61**, 311–318.

Osterheld, M. (1992) Effect of defoliation intensity on aboveground and belowground relative growth rates. *Oecologia* **92**, 313–316.

Osterheld, M. and McNaughton, S.J. (1991) Effect of stress and time for recovery on the amount of compensatory growth after grazing. *Oecologia* **85**, 305–313.

Paige, K. (1992) Overcompensation in response to mammalian herbivory: from mutualistic to antagonistic interactions. *Ecology* **73**, 2076–2085.

Paige, K. (1999) Regrowth following ungulate herbivory in *Ipomopsis aggregata*: geographic evidence for overcompensation. *Oecologia* **118**, 316–323.

Paige, K.N. and Whitham, T.G. (1987) Overcompensation in response to mammalian herbivory: the advantage of being eaten. *Am. Nat.* **129**, 407–416.

Rosenthal, J.P. and Welter, S.C. (1995) Tolerance to herbivory by a stemboring caterpillar in architecturally distinct maize and wild relatives. *Oecologia* **102**, 146–155.

Simms, E.L. and Triplett, J.K. (1994) Costs and benefits of plant responses to disease: resistance and tolerance. *Evolution* **48**, 1973–1985.

Stowe, K.A. (1998) Experimental evolution of resistance in *Brassica rapa*: correlated response of tolerance in lines selected for glucosinolate content. *Evolution* **52**, 703–712.

Stowe, K.A., Marquis, R.J., Hochwender, C.G. and Simms, E.L. (2000) The evolutionary ecology of tolerance to consumer damage. *Ann. Rev. Ecol Systemat.* **31**, 565–595.

Sun, D. (1992) Trampling resistance, recovery and growth rate of 8 plant species. *Agri. Ecosyst. Environ.* **15**, 265–273.

Tiffen, P. and Rausher, M.D. (1999) Genetic constraints and selection acting on tolerance to herbivory in the common morning glory, *Ipomoea purpurea*. *Am. Nat.* **154**, 700–716.

Trumble, Y.T., Kolodny-Hirsch, D.M. and Ting, I.P. (1993) Plant compensation for arthropod herbivory. *Ann. Rev. Entomol.* **38**, 93–119.

Wareing, P., Khalifa, M. and Terharne, K. (1968) Rate-limiting processes in photosynthesis at saturating light intensities. *Nature* **220**, 453–457.

Weis, A.E. and Hochberg, M.E. (2000) The diverse effects of intraspecific competition on the selective advantage to resistance: a model and its predictions. *Am. Nat.* **156**, 276–292.

Windel, P.N. and Franz, E.H. (1979) The effects of insect parasitism on plant competition: greenbugs and barley. *Ecology* **60**, 521–529.

Woledge, J. (1986) The effect of age and shade on the photosynthesis of white clover leaves. *Ann. Bot.* **57**, 257–262.

Yamamura, K. (1997) Optimality in the spatial leaf distribution of the weed *Portulaca oleracea* L. Ecological Modeling **104**, 133–143.