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Mechanisms of tolerance to herbivore damage: what do we know?

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Abstract. Identifying mechanisms of tolerance to herbivore damage will facilitate attempts to understand the role of tolerance in the evolutionary and ecological dynamics of plants and herbivores. Investigations of the physiological and morphological changes that occur in plants in response to herbivore damage have identified several potential mechanisms of tolerance. However, it is unlikely that all physiological changes that occur following damage are tolerance mechanisms. Few studies have made direct comparisons between the expression of tolerance and the relative expression of putative mechanisms. I briefly review empirical evidence for some of the better-studied potential mechanisms, including increased photosynthetic activity, compensatory growth, utilization of stored reserves, and phenological delays. For each of these mechanisms I discuss reasons why the relationship between tolerance and these characters may be more complicated than it first appears. I conclude by discussing several empirical approaches, including herbivore manipulations, quantitative trait loci (QTL) analysis, and selection experiments, that will further our understanding of tolerance mechanisms.

Key words: compensation, empirical approaches, herbivory, plant defense, plant–herbivore interaction, tolerance mechanisms

Introduction

Tolerance to herbivore damage may play an important role in the evolutionary and ecological dynamics between plants and herbivores. Because of this importance, tolerance has been subject of recent empirical and theoretical investigation. Much of this attention has focused on understanding the selective pressures acting on tolerance, constraints preventing tolerance from evolving in response to these selective pressures, and environmental factors that limit the expression of tolerance (reviewed in Rosenthal and Kotanen, 1994; Strauss and Agrawal, 1999; Stowe *et al.*, 2000). Less attention has been focused on identifying specific mechanisms of tolerance, which I define as those plant characters that reduce the detrimental effects of herbivore damage on plant fitness. Note that this definition does not imply that herbivores have necessarily been an important selective force in the evolution of these characters.

Identification of tolerance characters will facilitate answering several outstanding questions regarding the evolution and ecology of tolerance. For example, knowledge of mechanisms may be useful in designing more appropriate and powerful experiments to assess the role of herbivores vs. other selective agents such as competition, drought, and fire that may also act to increase tolerance to herbivory. Moreover, measuring specific mechanisms may help in identifying allocation and ecological constraints that limit tolerance from responding to these selective forces. Knowledge of mechanisms may also help in determining the environmental factors that limit the expression of tolerance and thus the ecological environments under which tolerance may most effectively minimize the potentially negative effects of herbivores. Finally identification of mechanisms will allow for a greater understanding of the selective role herbivores have had on plant physiology and morphology.

In this paper I briefly review empirical evidence that supports some of the better-studied putative mechanisms, discuss how these mechanisms may mitigate the effect of herbivore damage on plant fitness, and suggest why the relationship between the proposed mechanism and tolerance may be more complex than it first appears. After briefly reviewing putative mechanisms I suggest directions of future research that are necessary or useful in obtaining a more thorough understanding of tolerance mechanisms. This paper is not intended as a thorough review of the empirical evidence for possible mechanisms of tolerance. I focus on physiological and morphological characters that minimize the effects of herbivory on the fitness of individual plants. Although external factors such as water availability, nutrient availability, and competitive environment will affect the ability of individual plants to tolerate damage (McNaughton, 1979; Maschinski and Whitham, 1989; Whitham *et al*., 1991; Trumble *et al*., 1993) these external factors affect the expression of tolerance by mediating internal mechanisms. By understanding the internal mechanisms of tolerance we will be better able to understand how the environment in which plants are grown may affect the expression of tolerance.

Potential mechanisms of tolerance

Photosynthetic activity

Increased leaf level photosynthetic activity following herbivore damage is perhaps the most often cited mechanism of tolerance (McNaughton, 1979; Crawley, 1983; Whitham *et al*., 1991; Rosenthal and Kotanen, 1994; Strauss and Agrawal, 1999). The presumed role of increased photosynthesis comes from numerous empirical studies showing that partial leaf defoliation can cause increased photosynthetic rates in remaining tissue (reviewed in Welter,

1989). These studies have been conducted using several different plant species, suggesting that compensatory photosynthesis may be a widespread physiological response by plants to leaf removal. Increased photosynthetic activity may not, however, be a universal response to partial defoliation and the photosynthetic activity of some species has been shown to be unaffected (Caldwell *et al*., 1981) or even reduced by partial leaf removal (Zangerl *et al*., 1997). Moreover, empirical data suggest that photosynthetic activity is increased only in response to partial leaf defoliation. Other types of herbivore damage, including damage caused by leaf miners and sucking insects (hoppers and aphids) may have no effect on or actually reduce photosynthetic activity (reviewed in Welter, 1989).

Even for genotypes or species that increase photosynthetic activity following damage compensatory photosynthesis may not be a mechanism of tolerance. Some empirical studies that have found increased photosynthetic activity in response to herbivore damage have not found a relationship between tolerance and the degree to which photosynthetic rate is increased (Nowak and Caldwell, 1984). Rather than mitigating the effects of damage on fitness, increased photosynthetic activity following damage may be necessary to support the synthesis of induced chemical defense (Karban and Baldwin, 1997). Induced defense requires carbon for the synthesis of the enzymes needed to synthesize the defensive compounds, generation of ATP and NADPH needed to support enzymatic activity, a structural component of storage vacuoles needed to store some secondary chemicals, as well as a structural component of the induced defense. If the expression of both tolerance and defense depend on photosynthate produced following damage then competition for a limited amount of photosynthate may result in a physiological tradeoff between tolerance and resistance, which has been predicted by some theoretical models (Van der Meijden *et al*., 1988) and detected in some empirical studies (Fineblum and Rausher, 1995).

Compensatory growth and activation of dormant meristems

Herbivore damage can change plant growth trajectories, allowing plants to replace some, or all, tissue removed by herbivores. Compensatory regrowth has been documented following partial defoliation, but the most thoroughly investigated type of regrowth is the activation of meristems following removal or damage of vegetative or floral meristems (Inouye, 1982; Paige and Whitham, 1987; Prins and Verkaar, 1989; Doak, 1991; Bergelson *et al*., 1996; Mabry and Wayne, 1997). There is little doubt that activation of dormant meristems is involved in tolerance in at least some species. Plants that have only a single meristem such as some palms and some agaves are unable to replace a lost meristem and can be considered completely intolerant to meristem damage.

For the majority of species, however, the question is not whether the activation of meristems is involved but rather what is the relationship between regrowth and tolerance. This relationship remains largely unexplored and it is possible that maximal tolerance may be achieved by a sub-maximal level of regrowth.

An intermediate level of regrowth may be advantageous for several reasons. For example, regrowth of aboveground tissues following defoliation is often associated with reduced root growth (Richards, 1984; Mabry and Wayne, 1997; Zangerl *et al*., 1997). Smaller root volume may limit water and nutrient acquisition and thus reduce plant fitness in environments in which water or nutrients are limiting. Regrowth may also incur fitness costs because meristems activated after damage compete with developing flowers and fruits for water, nutrients, and carbohydrates (Mabry and Wayne, 1997). Finally, plants that activate many meristems may be shorter than genotypes that activate only one or two. If shorter plants attract fewer pollinators then plants that activate many meristems may end up being pollen limited, resulting in limited seed production (Juenger and Bergelson, 2000a). Shorter plants may also have reduced fitness because of shading by neighbors.

When implicating compensatory growth as a mechanism of tolerance care must be taken in how tolerance is measured. If tolerance is measured on the basis of plant size or biomass rather than fitness, then genotypes with highest compensatory regrowth will, by definition, be most tolerant of damage. As such, compensatory growth and tolerance will be positively correlated but compensatory growth is not a mechanism of tolerance. Moreover because damage may alter the relationship between biomass and fitness (Simoes and Baruch, 1991), using biomass as a surrogate for fitness should be done with caution, even if biomass and fitness are tightly correlated in undamaged plants. Likewise, for plants that reproduce clonally biomass and activation of dormant meristems may not be independent of fitness and thus care must be taken when implicating these traits as mechanisms of tolerance.

Utilization of stored reserves

Utilization of storage reserves may be an important mechanism of tolerance for some growth forms such as perennials, biennials, and winter annuals that contain large taproots or other storage organs. The importance of stored reserves may also depend on type of damage. For example, seeds with greater amounts of stored reserves may be more tolerant of partial cotyledon removal than smaller seeds. Several studies have found evidence for mobilization of carbon reserves following grazing (e.g. Danckwerts and Gordon, 1987; Van der Heyden and Stock, 1996) but perhaps the best known empirical support, at least among evolutionary ecologists, for stored reserves being associated with tolerance comes from Van der Meijden *et al*. (1988) who found root–shoot

ratios were significantly positively correlated with regrowth following defoliation among five biennial species. However, other studies have found no evidence that stored reserves are associated with tolerance (Davidson and Milthorpe, 1966; Ryle and Powell, 1975; Richards and Caldwell, 1985), and a study of eight desert perennials detected no clear interspecific relationship between stored carbohydrate and regrowth following clipping (Trlica and Cook, 1971). Stored reserves may not be directly related to tolerance because stored reserves are largely carbon based; whereas herbivory may deplete nitrogen and other nutrients more than carbon (Chapin *et al*., 1990). Alternatively, even in systems where stored reserves are an important component of tolerance, not all stored reserves are likely to be available following damage. As such, there may not be a direct relationship between the total quantity and the availability of stored reserves.

Phenological changes

Changes in plant phenology may be one of the more widespread but less studied effects of herbivore damage. Differential changes in phenology may thus be one of the more widespread mechanisms of tolerance. Empirical evidence for herbivory altering phenology has been collected from several species and for several types of herbivore damage. Partial defoliation, meristem damage, and gall infestation have all been shown to cause delayed growth, flower production, and/or fruit production (Harnett and Abrahamson, 1979; Islam and Crawley, 1983; Marquis, 1988; Bergelson and Crawley, 1992; Juenger and Bergelson, 1997; Mabry and Wayne, 1997; Lennartsson *et al*., 1998; Meyer, 1998b).

Genotypic differences in the length of phenological delay caused by herbivore damage may result in genetic variation for tolerance for several reasons. First, many plants live in seasonal environments where the end of the growing season may limit reproduction. If herbivory causes delays in seed maturation then genotypes that experience the shortest delay following damage may be most tolerant, because only those genotypes will produce seed before the end of the season. Alternatively, if herbivory causes equal delays for all genotypes, but genotypes differ in their time of development, then faster developing genotypes may be most tolerant. Under either of these scenarios, the expression as well as the pattern of selection acting on tolerance will depend upon the length of growing season. During long growing seasons all genotypes may have sufficient time to mature seeds regardless of damage and as such there may be little variation in tolerance and little selection favoring tolerance. In contrast, during short growing seasons delays in seed production may result in damaged plants dying before seed production is complete, resulting in severe reductions in fitness. Under these conditions, there may be considerable variation for

tolerance and those genotypes that experience the shortest delay in reproduction following damage will be most tolerant and selectively favored. Phenological delays may also be related to tolerance because delays cause plants to be exposed to floral herbivores (Pilson, 2000) or miss peak pollinator activity (English-Loeb and Karban, 1992), which may result in reduced seed set, reduced siring success, or a higher proportion of selfed seeds.

Plant architecture at the time of damage

The above mechanisms may generally be viewed as 'active' or induced mechanisms of tolerance in that damage elicits physiological changes that do not occur in undamaged individuals. Tolerance may also result from 'passive' or constitutive mechanisms related to physiology and morphology at the time of damage. Possible constitutive mechanisms of tolerance include root–shoot ratios, stem number or stem rigidity, and proportion of photosynthetic surfaces in stems and fruits.

Root–shoot ratios at the time of damage may contribute to tolerance in several ways. Individuals with high root–shoot ratios may be more tolerant because they are better able to acquire nutrients for regrowth or seed production following loss of nutrients stored in tissue lost to herbivores (Chapin and McNaughton, 1989). Alternatively, fitness of genotypes with low root–shoot ratios may be less affected by herbivore damage if at the time of damage plant photosynthetic capacity is limited more by water than photosynthetic area. Under these conditions, loss of photosynthetic surface may reduce transpiration thereby reducing demands on the root system. As such, loss of some photosynthetic capacity may cause only minor detriments to fitness. Stem number and rigidity may also affect the probability that stem boring insects cause stems to break or fall over when damaged, as well as the number of stems that remain undamaged after a plant is attacked (Rosenthal and Welter, 1995). Finally, photosynthetic activity of reproductive structures may commonly contribute greater than 20% of the carbon needs of developing fruits and seeds (Bazzaz *et al*., 1979). As such, genotypes with a higher proportion of photosynthetic surfaces in stems and fruits may be less dependent on photosynthate produced by leaves and thus more tolerant of folivory.

Future directions

Perhaps the greatest limitation to our understanding of tolerance mechanisms is that few studies have directly investigated the relationship between the expression of tolerance and the expression of putative mechanisms. Rather, putative mechanisms have been identified by documenting physiological changes

that occur following damage. As discussed above, the relationship between these changes and tolerance is not necessarily clear. The basic question that needs to be addressed when investigating mechanisms is what is the relationship between the expression of tolerance and the expression of putative mechanisms? e.g. Are more tolerant genotypes the same genotypes that exhibit the greatest levels of compensatory photosynthesis? or activate the greatest number of meristems following damage? and for continuous types of damage, Is there a relationship between levels of damage and increased, or decreased, expression of mechanism? Moreover, because tolerance is a relative measure (the tolerance of a genotype or species can only be quantified in relation to other genotypes or species, Rausher (1992a)) experiments establishing a trait as a correlate of tolerance need to use groups of related individuals. For each of these groups it is necessary to measure the level of tolerance and the expression of the specific trait. It is not sufficient to simply compare the physiological status of two groups of plants – one group that receives damage and one group that does not.

Direct comparisons between tolerance and putative mechanisms have been made almost exclusively in interspecific or interpopulation studies (e.g. Caldwell *et al.*, 1981; Van der Meijden *et al.*, 1988; Rosenthal and Welter, 1995; but see Juenger and Bergelson, 2000b). Interspecific comparisons are useful for identifying possible mechanisms but because species are likely to differ in numerous traits and experience different selective environments, mechanisms responsible for interspecific differences are not necessarily the same traits that are responsible for intraspecific differences (Rausher, 1984; Via, 1990). Rather, traits contributing to interspecific differences may be fixed within populations and the lack of genetic variation means that such traits will not be responsible for genetic differences in the expression of tolerance within populations.

Empirical study of tolerance mechanisms is limited to relatively few types of damage and has been conducted primarily in herbaceous systems. In particular, the majority of studies have focused on the effects meristem removal and artificial defoliation imposed during a short period of time have on plant growth and fitness of herbaceous annuals and short-lived perennials. Although imposed, discrete damage may appropriately mimic some types of naturally occurring damage (i.e. grazing) a large percentage, if not a majority of naturally occurring herbivore damage is probably more dispersed and occurs during much longer periods of time. Dispersed damage is generally less detrimental to plants than concentrated damage (Cook and Stoddard, 1960; Mauricio *et al.*, 1993; Meyer, 1998b) and may have more subtle effects on plant physiology and thus elicit different responses. Moreover, tolerance to underground tissue has received little attention (but see Houle and Genevieve, 1996). Of course, the focus on short-lived herbaceous plants is understandable given the practical necessities of empirical work, nevertheless it is worth realizing that

the relative importance of different mechanisms may depend on life history and the mechanisms employed by long lived trees may be much different than the mechanisms employed by herbaceous annuals.

Mechanisms of tolerance are also likely to be interrelated. Just as plant resistance is likely to be a function of specific chemicals, nutrient balance, and morphological characters, tolerance is likely to be a function of numerous physiological interactions and these mechanisms may not be independent. For example, compensatory growth that results from increased photosynthetic rate, activation of dormant meristems, or utilization of storage supplies may result in delayed flowering and fruit set (e.g. Bergelson and Crawley, 1992; Mabry and Wayne, 1997). Alternatively, the expression of tolerance mechanisms to some types of damage may be suppressed by the expression of mechanisms of tolerance to other types of damage. For example, meristem activation in response to meristem damage may be suppressed by carbon limitations resulting from reduced photosynthetic area caused by defoliation (Meyer, 1998a). Because of the interrelatedness of mechanisms, studies that examine numerous mechanisms may provide greater insight into the physiological and morphological basis of tolerance than studies examining only single mechanisms. For experiments in which multiple potential mechanisms are measured, path analysis provides a statistical approach to disentangle the relative effects different mechanisms have on the expression of tolerance (Kingsolver and Shemske, 1991; Scheiner and Callahan, 1999).

Artificial selection may also be useful for identifying tolerance mechanisms. Potential mechanisms may be identified by selecting replicated lines for both increased and decreased levels of tolerance and measuring phenotypic traits that evolve in response to the selection placed on tolerance. It may be particularly interesting to determine if replicated selected lines evolve the same mechanisms, or if multiple evolutionary paths are available for achieving similar levels of tolerance. A reverse approach of selecting on putative tolerance traits, then determining if tolerance evolves in response to this selection, may also be used. Stowe (1998) used a similar approach to determine that glucosinolate production and tolerance appear to be negatively genetically correlated in *Brassica rapa*. Selection conducted in both manners would be particularly strong evidence for implicating traits as tolerance mechanisms.

Phenotypic and genetic manipulations may also be used to identify plant traits that affect the expression of tolerance. Several morphological and physiological phenotypes can be altered through exogenously applied plant growth hormones (e.g. Cippolini and Schulz, 1999) or through mutant or transgenic lines (Schmitt *et al.*, 1995; Purrington and Bergelson, 1999; Tatar, 2000). Of course, these approaches involve several caveats and limitations. For example, exogenously applied hormones may alter the expression of many unmeasured physiological and morphological characters, the process of

transformation may have phenotypic effects independent of the transgene itself, and expression patterns of transgenes may be quite different than the expression of endogeneous genes (Schmitt, 1999). Perhaps more important than these technical limitations is that like interspecific studies and selection analyses transgenic manipulation may often produce phenotypes that are outside of the range of phenotypes seen in nature. Moreover, unlike interspecific studies and selection analyses, which take advantage of extant genetic variation, phenotypic manipulations involving hormone application or genetic mutants may produce phenotypes that are outside of phenotypes obtainable with extant genetic variation. For these reasons, genetic manipulation may be best viewed as a tool for identifying candidate traits and candidate genes. However, in order to understand adaptive evolution in natural populations these approaches should be combined with other approaches that test for polymorphism and a role of these traits or genes in natural populations (Schmitt, 1999). Nevertheless, genetic and phenotypic manipulations clearly provide powerful tools for exploring the role of specific plant characters and identifying genes involved in the expression of tolerance.

The above methods are useful for identifying mechanisms but they do not help in determining whether herbivores impose selection on those characters. One approach for determining that herbivores impose selection on tolerance or tolerance mechanisms is to test whether experimental removal of herbivores causes a change in the pattern of selection acting on these traits (Mitchell-Olds and Shaw, 1987; Wade and Kalisz, 1990; Mauricio and Rausher, 1997). This approach was taken recently by Juenger and Bergelson (2000b) in showing that herbivores alter the pattern of phenotypic selection acting on branch production and time of flower production in *Ipomopsis*. Because Juenger and Bergelson (2000b) also demonstrated that these traits are positively correlated with tolerance, their study provides evidence that herbivores may act as a selective agent on tolerance mechanisms. However, it should be noted that genetic variation for tolerance was not detected in this system and although the presence of herbivores altered the pattern of phenotypic selection herbivores did not appear to alter the patterns of selection estimated using breeding values (Rausher, 1992b). In trying to understand the selective forces acting on tolerance it would also be interesting to manipulate multiple selective agents. Experiments that manipulate multiple selective agents may help to determine the relative importance of herbivores vs. other selective agents, such as competitors and fire, that have also been implicated as selective agents acting on tolerance.

This paper has focused on an organismal approach to studying tolerance mechanisms. Mapping and identification of loci that affect quantitative genetic variation (QTL) in tolerance also provides a potentially powerful complementary approach to understanding mechanisms. The mapping of QTL using

traditional techniques such as random amplified polymorphic DNA (RAPDs) or restriction fragment length polymorphisms (RFLPs) is extremely laborious. However, amplified fragment length polymorphisms (AFLPs) make the generation of linkage maps and identification of QTL feasible in natural populations for which no prior genetic information is available (Mueller and Wolfenbarger, 1999). Of course, identification of QTL is not equivalent to understanding the genetic basis of a trait. Individual QTL often have large confidence intervals that may contain hundreds of genes. Even in *Arabidopsis*, which has extensive genetic markers, the confidence intervals around individual QTL are often greater than 300 kbp (Kearsey and Farquhar, 1998). Moreover, even in well studied organisms the function of many genes are unknown and can, at best, only be inferred on the basis of sequence similarity with better studied genes. Given these caveats, identification of QTL may be a powerful tool for identifying the genetic basis and thus the mechanisms of tolerance. This is especially true in *Arabidopsis* and rice where entire genome sequences will soon be available, facilitating the identification of specific genes associated with QTL. Even in systems where the identification of specific genes may not be feasible, at least in the near future, QTL analyses may be used to determine if similar chromosomal regions are involved in tolerance to different types of damage and when plants are grown in different environmental conditions. Moreover, even without the identification of specific tolerance genes, identification of QTL associated with tolerance may helpful for agricultural purposes – allowing plant breeders to use marker assisted introgression rather than having to assay for tolerance at each stage of selection in the breeding process.

Conclusions

The majority of work on the evolutionary ecology and ecological genetics of tolerance has used operational definitions of tolerance (e.g. Simms and Triplett, 1994; Mauricio *et al*., 1997; Shen and Bach, 1997; Stowe, 1998; Tiffin and Rausher, 1999; Juenger and Bergelson, 2000b). Operational definitions are useful because they allow the evolutionary and ecological dynamics of tolerance to be explored without knowledge of mechanisms. However, by ignoring mechanisms operational definitions are also limiting. The identification of mechanisms may provide insight into the selective forces that act on tolerance as well as the ecological environments that affect the expression of tolerance. Perhaps more importantly, identifying mechanisms is necessary for understanding how selective forces imposed by herbivores have altered plant physiology, morphology, and development. I have reviewed some of the empirical data related to several potential tolerance mechanisms, including

compensatory photosynthesis, compensatory growth, activation of dormant meristems, utilization of stored reserves, phenological changes, and plant architecture. All of these may serve as important mechanisms of tolerance in some systems and in some environments. However, at this time it is difficult to determine the relative importance of these mechanisms given that few empirical studies have investigated the relationship between the expression of any one of these mechanisms and the expression of tolerance, let alone examined the relationship between the expression of multiple mechanisms and the expression of tolerance. Recognizing that identification of tolerance mechanisms requires investigating the relationship between the expression of a trait and the expression of tolerance, and not just the morphological and physiological changes that occur following herbivore damage, will help to increase our understanding of tolerance mechanisms. In addition, several powerful empirical approaches, including artificial selection, phenotypic manipulations, and QTL analysis have been used either not at all or only to a limited extent in the study of tolerance. Application of these empirical approaches may prove extremely useful in identifying tolerance mechanisms and thereby extending our understanding of the evolutionary ecology of tolerance as well as our knowledge of the selective role herbivores have and continue to play in the evolution of plant phenotypes.

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References

Bazzaz, F.A., Calson, R.W. and Harper, J.W. (1979) Contribution of reproductive effort by photosynthesis of flowers and fruits. *Nature* **279**, 554–555.

Bergelson, J. and Crawley, M.J. (1992) The effects of grazers on the performance of individuals and populations of scarlet gilia, *Ipomopsis aggregata*. *Oecologia* **90**, 435–444.

Bergelson, J., Juenger, T. and Crawley, M.J. (1996) Regrowth following herbivory in *Ipomopsis aggregata* – compensation but not overcompensation. *Am. Nat.* **148**, 744–755.

Caldwell, M.M., Richards, J.H., Johnson, D.A., Nowack, R.S. and Dzurec, R.S. (1981) Coping with herbivory: photosynthetic capacity and resource allocation in two semiarid *Agropyron* bunchgrasses. *Oecologia* **50**, 14–24.

Chapin, F.S. III and McNaughton, S.J. (1989) Lack of compensatory growth under phosphorus deficiency in grazing-adapted grasses from the serengeti plains. *Oecologia* **79**, 551–557.

Chapin, F.S. III, Schulze, E.D. and Mooney, H.A. (1990) The ecology and economics of storage in plants. *Annu. Rev. Ecol. Syst.* **21**, 423–447.

Cipollini, D.F. and Schultz, J.C. (1999) Exploring cost constraints on stem elongation in plants using phenotypic manipulation. *Am. Nat.* **153**, 236–242.

Crawley, M.J. (1983) *Herbivory, the Dynamics of Animal–Plant Interactions*. University of California Press, Berkley.

Cook, C.W. and Stoddard, L.A. (1960) Physiological responses of big sagebrush to different types of herbage removal. *J. Range Mgmt.* **13**, 14–16.

Danckwerts, J.E. and Gordon, A.J. (1987) Long-term partitioning, storage, and re-mobilization of ^{14}C assimilated by *Lolium perenne* (cv. Melle). *Ann. Bot.* **59**, 55–66.

Davidson, J.L. and Milthorpe, F.L. (1966) The effect of defoliation on the carbon balance in *Dactylis glomerata*. *Ann. Bot.* **30**, 185–198.

Doak, D. (1991) The consequences of herbivory for dwarf fireweed: different time scales, different morphological scales. *Ecology* **72**, 1397–1407.

English-Loeb, G.M. and Karban, R. (1992) Consequences of variation in flowering phenology for seed head herbivory and reproductive success in *Erigeron glaucus* (Compositae). *Oecologia* **89**, 588–595.

Fineblum, W.L. and Rausher, M.D. (1995) Evidence for a trade-off between resistance and tolerance to herbivory damage in a morning glory. *Nature* **377**, 517–520.

Harnett, D.C. and Abrahamson, W.G. (1979) Effects of stem gall insects on life-history patterns in *Solidago canadensis*. *Ecology* **60**, 910–917.

Houle, G. and Genevieve, S. (1996) Additive effects of genotype, nutrient availability and type of tissue damage on the compensatory response of *Salix planifolia* spp. *planifolia* to stimulated herbivory. *Oecologia* **107**, 373–378.

Inouye, D.W. (1982) The consequences of herbivory: a mixed blessing for *Jurinea mollis* (Asteraceae). *Oikos* **39**, 269–290.

Islam, Z. and Crawley, M.J. (1983) Compensation and regrowth in ragwort (*Senecio jacobaea*) attacked by cinnabar moth (*Tyria jacobaeae*). *J. Ecol.* **71**, 829–843.

Juenger, T. and Bergelson, J. (1997) Pollen and resource limitation of compensation to herbivory in scarlet gilia, *Ipomopsis aggregata*. *Ecology* **78**, 1684–1695.

Juenger, T. and Bergelson, J. (2000a) Does early season browsing influence the effect of self-pollination in scarlet gilia? *Ecology* **81**, 41–48.

Juenger, T. and Bergelson, J. (2000b) 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.

Karban, R. and Baldwin, I.T. (1997) *Induced Responses to Herbivory*. The University of Chicago Press, Chicago.

Kingsolver, J.G. and Schemske, D.W. (1991) Path analyses of selection. *Trends Ecol. Evol.* **6**, 246–280.

Kearsey, M.J. and Farquhar, A.G.L. (1998) QTL analysis in plants; where are we now? *Heredity* **80**, 137–142.

Lennartsson, T.J., Nilsson, P. and Tuomi, J. (1998) Induction of overcompensation in the field gentian, *Gentianella campestris*. *Ecology* **79**, 1061–1072.

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

Marquis, R.J. (1988) Phenological variation in the neotropical understory shrub *Piper arieianum* – causes and consequences. *Ecology* **69**, 1552–1565.

Mauricio, R.D., Bowers, M.D. and Bazzaz, F.A. (1993) Pattern of leaf damage affects fitness of the annual plant *Raphanus sativus* (Brassicaceae). *Ecology* **74**, 2066–2071.

Mauricio, R. and Rausher, M.D. (1997) Experimental manipulation of putative selective agents provides evidence for the role of natural enemies in the evolution of plant defense. *Evolution* **51**, 1435–1444.

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.

Maschinski, J. and Whitham, T.G. (1989) The continuum of plant responses to herbivory: the influence of plant association, nutrient availability, and timing. *Am. Nat.* **134**, 1–19.
McNaughton, S.J. (1979) Grazing as an optimization process – grass ungulate relationships in the Serengeti. *Am. Nat.* **113**, 691–703.
Meyer, G.A. (1998a) Mechanisms promoting recovery from defoliation in goldenrod (*Solidago altissima*). *Can. J. Bot.* **76**, 450–459.
Meyer, G.A. (1998b) Pattern of defoliation and its effect on photosynthesis and growth of goldenrod. *Func. Ecol.* **12**, 270–279.
Mitchell-Olds, T. and Shaw, R.G. (1987) Regression analysis of natural selection: statistical inference and biological interpretation. *Evolution* **41**, 1149–1161.
Mueller, U.G. and Wolfenbarger, L.R. (1999) AFLP genotyping and fingerprinting. *Trends Ecol. Evol.* **14**, 389–394.
Nowak, R.S. and Caldwell, M.M. (1984) A test of compensatory photosynthesis in the field: implications for herbivory tolerance. *Oecologia* **61**, 311–318.
Paige, K.N. and Whitham, T.G. (1987) Overcompensation in response to mammalian herbivory: the advantage of being eaten. *Am Nat.* **129**, 407–416.
Pilson, D. (2000) Herbivory and natural selection on flowering phenology in wild sunflower, *Helianthus annuus*. *Oecologia* **122**, 72–82.
Prins, A.H. and Verkaar, H.J. (1989) Responses of *Cynoglossum officinale* and *Senecio jacobaea* to various degrees of defoliation. *New Phytol.* **111**, 725–731.
Purrington, C.B. and Bergelson, J. (1999) Exploring the physiological basis of costs of herbicide resistance in *Arabidopsis thaliana*. *Am. Nat.* **154**, S82–S91.
Rausher, M.D. (1984) Tradeoffs in performance on different hosts: evidence from within and between-site variation in the beetle *Deloyala guttata*. *Evol.* **38**, 582–595.
Rausher, M.D. (1992a) Natural selection and the evolution of plant–insect interactions. In B.D. Roitberg and M.S. Isman, (eds) *Insect Chemical Ecology: and Evolutionary Approach*. Routledge, Chapman and Hall, New York, pp. 20–88.
Rausher, M.D. (1992b) The measurement of selection on quantitative traits: biases due to environmental covariances between traits and fitness. *Evolution* **46**, 616–626.
Richards, J.H. (1984) Root growth responses to defoliation in two *Agropyron* bunchgrasses: field observations with an improved root periscope. *Oecologia* **64**, 21–25.
Richards, J.H. and Caldwell, M.M. (1985) Soluble carbohydrates, concurrent photosynthesis and efficiency in regrowth following defoliation: a field study with *Agropyron* species. *J. App. Ecology* **22**, 907–920.
Rosenthal, J.P. and Kotanen, P.M. (1994) Terrestrial plant tolerance to herbivory. *Trends Ecol. Evol.* **9**, 145–148.
Rosenthal, J.P. and Welter, S.C. (1995) Tolerance to herbivory by a stemboring caterpillar in architecturally distinct maizes and wild relatives. *Oecologia* **102**, 146–155.
Ryle, G.J.A. and Powell, C.E. (1975) Defoliation and regrowth in the graminaceous plant: the role of current assimilate. *Ann. Bot.* **39**, 297–310.
Scheiner, S.M. and Callahan, H.S. (1999) Measuring natural selection on phenotypic plasticity. *Evolution* **53**, 1704–1713.
Schmitt, J. (1999) Introduction: experimental approaches to testing adaptation. *Am. Nat.* **154**, S1–S3.
Schmitt, J.A., McCormac, C. and Smith, H. (1995) A test of the adaptive plasticity hypothesis using transgenic and mutant plants disabled in phytochrome-mediated elongation responses to neighbors. *Am. Nat.* **146**, 937–953.
Shen, C.S. and Bach, C.E. (1997) Genetic variation in resistance and tolerance to insect herbivory in *Salix cordata*. *Ecol. Entomol.* **22**, 335–342.
Simms, E. and Triplett, J. (1994) Costs and benefits of plant responses to disease: resistance and tolerance. *Evolution* **48**, 1973–1985.
Simoes, M. and Baruch, Z. (1991) Responses to simulated herbivory and water-stress in two tropical C-4 grasses. *Oecologia* **88**, 173–180.

Stowe, K.A. (1998) Experimental evolution of resistance in *Brassica rapa*: correlated response of tolerance in lines selected for glucosinolate content. *Evol.* **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. *Annu. Rev. Ecol. Syst.* **31**, 565–595.

Strauss, S.Y. and Agrawal, A.A. (1999) The ecology and evolution of plant tolerance to herbivory. *Trends Ecol. Evol.* **14**, 179–185.

Tatar, M. (2000) Transgenic organisms in evolutionary ecology. *Trends Ecol. Evol.* **15**, 207–211.

Tiffin, 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.

Trlica, M.J. Jr. and Cook, C.W. (1971) Defoliation effects on carbohydrate reserves of desert species. *J. Range Mgmt.* **24**, 418–425.

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

Van der Heyden, F. and Stock, W.D. (1996) Regrowth of a semiarid shrub following simulated browsing: the role of reserve carbon. *Funct. Ecol.* **10**, 647–653.

Van der Meijden, E., Wijn, M. and Verkaar, H.J. (1988) Defense and regrowth, alternative plant strategies in the struggle against herbivores. *Oikos* **51**, 355–363.

Via, S. (1990) Ecological genetics and host adaptation in herbivorous insects: the experimental study of evolution in natural and agricultural systems. *Annu. Rev. Entomol.* **35**, 421–446.

Wade, M.J. and Kalisz, S. (1990) The causes of natural selection. *Evolution* **44**, 1947–1955.

Welter, S.C. (1989) Arthropod impact on plant gas exchange. In E.A. Bernays (ed.) *Insect–Plant Interactions*, vol. 1. CRC Press, Boca Raton, FL, pp. 135–150.

Whitham, T.G., Maschinski, J., Larson, K.C. and Paige, K.N. (1991) Plant responses to herbivory: the continuum from negative to positive and underlying physiological mechanisms. In P.W. Price, T.M. Lewisohn, G.W. Fernandes and W.W. Bensons (eds) *Plant–Animal Interactions: Evolutionary Ecology in Tropical and Temperate Regions*. John Wiley and Sons, New York.

Zangerl, A.R., Arntz, A.M. and Berenbaum, M.R. (1997) Physiological price of an induced chemical defense: photosynthesis, respiration, biosynthesis, and growth. *Oecologia* **109**, 433–441.