

LETTER

A simultaneous test of trophic interaction models: which vegetation characteristic explains herbivore control over plant community mass?

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Abstract

Predicting herbivore control over plants (i.e. changes in plant mass due to herbivore damage) is a central goal of ecology. Progress has been limited, however, because the vegetation characteristics thought to influence herbivore control are naturally correlated and typically experimentally confounded. To address this problem, we defined eight conventional models that predict herbivore control over plant community mass, each model based on a different vegetation characteristic (i.e. host concentration, tissue nitrogen, growth rate, size, tolerance of herbivory or net primary productivity). We then used structural equation modelling to test each model against two field experiments. Our results clearly rejected all models except for a tolerance of herbivory mechanism; stems with greater access to limiting resources better tolerated herbivory, regardless of where herbivore activity was greatest. Consequently, herbivore reductions of plant community mass were greatest at low resource availability. This adds to evidence that herbivore activity poorly predicts herbivore control.

Keywords

Herbivory, host concentration, individual-based model, leaf nitrogen concentration, net primary productivity, plant apparency, plant growth rate, plant tolerance, plant vigour, top-down.

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INTRODUCTION

Over the past 50 years, models that attempt to explain variation in herbivore control over plant community mass have proliferated. These models assume that changes in herbivore control (i.e. changes in plant mass due to herbivore damage) are caused by changes in vegetation characteristics, either net primary productivity (NPP) (Power 1992; Chase *et al.* 2000), host concentration (Janzen 1970; Long *et al.* 2003), plant tissue nitrogen concentrations (White 1984; Throop & Lerdau 2004), plant growth rate (Coley *et al.* 1985; Price 1991), plant size (Feeny 1976) or plant tolerance of herbivory (Maschinski & Whitham 1989; Wise & Abrahamson 2007). Through a variety of proposed mechanisms, these vegetation characteristics are expected to determine herbivore activity (i.e. herbivore abundance and herbivore damage to plants) and/or the response of plant growth to herbivore damage. Consequently, these models assume that vegetation ultimately constrains the strength of herbivore control.

Numerous studies test these models but the empirical evidence remains equivocal. These vegetation characteristics are naturally correlated and typically confounded in experiments (Appendix S1). Moreover, models often overlap in assumptions (e.g. herbivore abundance positively influences herbivore damage). This makes it difficult to identify the mechanisms responsible for changes in herbivore control. Consequently, experimental conclusions are often questioned and meta-analyses cannot disentangle the influence of one vegetation characteristic vs. another (Moon *et al.* 1999; Schmitz *et al.* 2000; Halaj & Wise 2001; Schädler *et al.* 2003). Therefore, little progress has been made towards determining which vegetation characteristics determine herbivore control.

We addressed this problem using large-scale field experiments and structural equation modelling (SEM). SEM provides a rigorous statistical approach because it can explicitly test the entire causal structure assumed by a trophic interaction model (Grace 2006). We first employed an 'alternative' model approach where we *a priori* defined eight

causal models, each describing the mechanisms by which a vegetation characteristic determines herbivore control. Second, we employed a 'confirmatory' approach where we tested each model against two experiments that we simultaneously conducted in old-field communities. Testing each model against two experiments, of monoculture and polyculture design, confirmed whether a model's mechanisms operated in our system. Thus, we show that a resource-limited plant tolerance of herbivory model explains herbivore control over both simple and complex plant communities. Moreover, the causal mechanisms assumed by the other models cannot adequately explain herbivore control in this system.

Conventional trophic interaction models

We begin by using a review of the causal mechanisms assumed by each trophic interaction model as the basis for constructing the alternative causal models (Fig. 1). Several models make similar assumptions about how vegetation influences herbivore control. In addition, many models predict that the vegetation characteristic can have positive, negative and even neutral effects on herbivore control over individual plants. Regardless of the pattern of herbivore control over individual plants, herbivore reductions of plant community mass are expected to be greatest where herbivore reductions of per capita mass are greatest.

Resource-limited plant tolerance of herbivory

Plant tolerance models recognize two factors that determine how strongly plant growth is resource limited: a limiting resource's abundance and herbivory (Fig. 1a). Increasing the resource's abundance reduces its limitation on plant growth (Hilbert *et al.* 1981; Maschinski & Whitham 1989; Wise & Abrahamson 2007). And, while herbivory reduces plant mass by removing or damaging plant tissue, different herbivores are likely to exacerbate, cause or ameliorate limitation of different resources (Wise & Abrahamson 2007). Plausible examples are leaf chewers influencing carbon limitation; xylem feeders influencing water limitation; apical meristem feeders influencing carbon or nitrogen limitation by stimulating branching (Wise & Abrahamson 2007). As described below, this model predicts that plant tolerance either increases, remains constant or decreases as resource availability increases (Wise & Abrahamson 2007).

Increasing a limiting resource's abundance via experimental manipulation or via reduction of competitors could directly decrease herbivore reduction of per plant mass (paths 1a and 2a respectively) and therefore plant community mass (path 3). If herbivory removes tissue or exacerbates or causes resource limitation, then damaged plants have greater potential to maintain growth and replace lost tissue when they have greater access to the resource (Maschinski & Whitham 1989; Wise & Abrahamson 2007).

In contrast, damaged plants might be more tolerant at low limiting resource abundance if slow growing plants require a smaller increase in growth rate to compensate (Hilbert *et al.* 1981). Here, increasing limiting resource abundance directly increases herbivore reduction of per plant mass (paths 1b and 2b respectively). Lastly, increasing resource availability does not necessarily influence plant tolerance (paths 1c and 2c). This happens when the resource is not limiting or when herbivory does not alter resource limitation (Wise & Abrahamson 2007). Here, any relationship between herbivore control per plant and herbivore control over plant community mass (path 3) cannot be explained by an influence of resource availability on plant tolerance (paths 1c and 2c).

Net primary productivity

The NPP models are of three types, resource-controlled (Fig. 1b), consumer-controlled (Fig. 1c) and heterogeneous (Fig. 1d) (Chase *et al.* 2000). Each model assumes that NPP (i.e. rate of plant mass production per m² in the absence of consumers) has a bottom-up influence on standing crops (i.e. mass per m² in the presence of food web interactions) (Chase *et al.* 2000). They make different assumptions, however, about whether consumer control increases with consumer abundance and whether some plants have defences (Chase *et al.* 2000). Depending on these conditions, NPP either does not influence, increases or decreases herbivore reduction of plant mass (Chase *et al.* 2000).

The resource-controlled model assumes that NPP has a positive influence on standing crops (Fig. 1b, pathway 1–2–3). It predicts that NPP will not explain variation in herbivore control because it also assumes that herbivore damage does not vary with herbivore abundance (path 4) (Chase *et al.* 2000). This is thought to occur when intra- and inter-specific competition among herbivores increases as herbivore abundance increases (Ariditi & Ginzburg 1989) or when herbivores only attack unproductive plants (e.g. post-reproductive or diseased plants) (Pimm 1982).

The consumer-controlled model (Fig. 1c) also assumes a strong bottom-up influence of NPP on standing crops but allows herbivore damage to increase with herbivore abundance (Chase *et al.* 2000). Herbivore abundance is expected to increase if NPP does not support carnivores or when increasing NPP destabilizes carnivores (pathway 1b–2b–3b) (Oksanen *et al.* 1981; Wootton & Power 1993; Abrams & Roth 1994; Chase *et al.* 2000). In these systems, greater herbivore abundance increases herbivore damage per plant (solid-path 4), even with strong interference among herbivores (Oksanen *et al.* 1995; Chase *et al.* 2000). Herbivory reduces per capita plant mass (dashed-path 5) and therefore community level mass (solid-path 6). If herbivore reduction of plant mass is particularly strong, plant standing crops respond to NPP either weakly or not at all (dashed-path 1b)

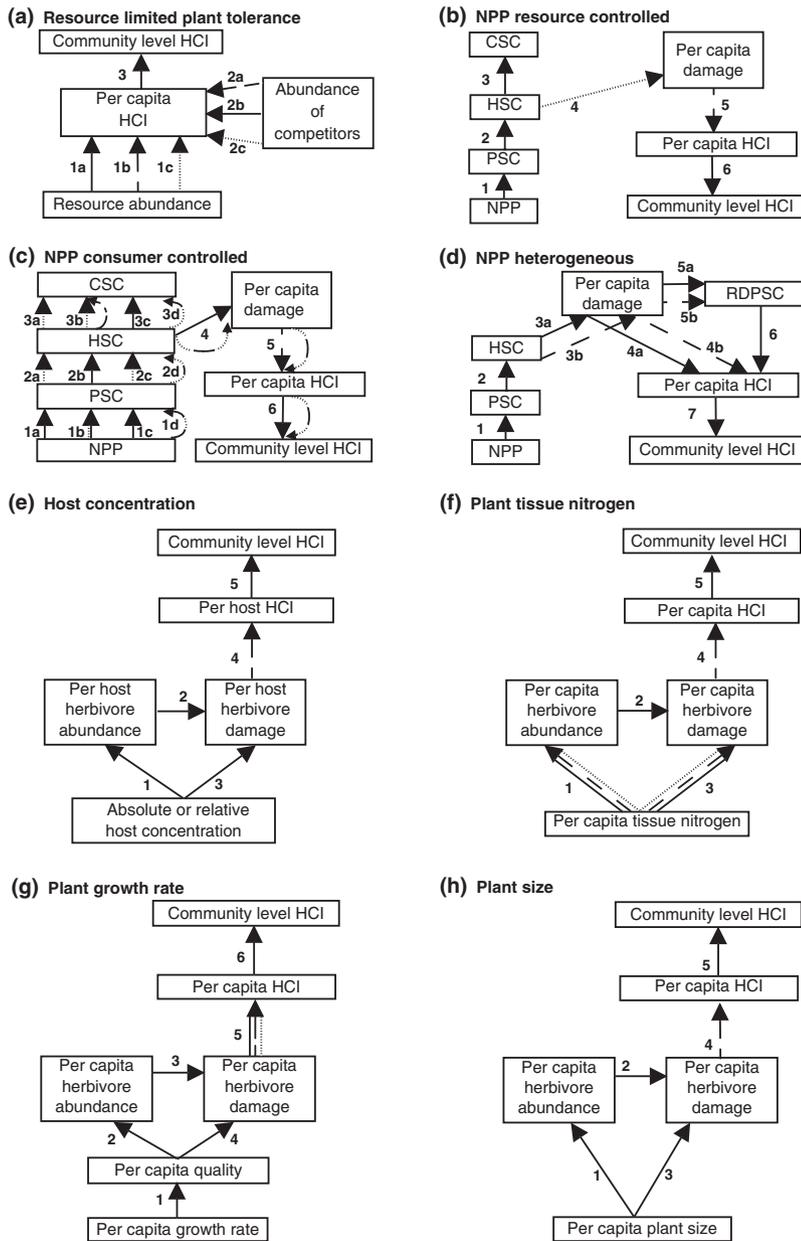


Figure 1 Conceptual diagrams that illustrate the causal relationships assumed by trophic interaction models. Solid paths indicate positive relationships; dashed paths indicate negative relationships; curved paths indicate a polynomial relationship; dotted paths indicate the model explicitly predicts no relationship. HCI, an Herbivore Control Index, is constrained between 1 and -1 (see Methods); positive values indicate herbivory increases plant mass, 0 indicates herbivory does not change plant mass and negative values indicate herbivory decreases plant mass. HCI can be calculated per host (i.e. across a single host's stems in a plot), per capita (i.e. across all species stems in a plot) or at the community level (i.e. m^{-2}). Models a, d and g allow HCI to range from negative to positive values. Models b, c, e, f and h constrain HCI to less than or equal to zero. Note that when a variable positively influences HCI, herbivores cause stronger reduction of plant mass at low values of that variable (e.g. panel a, path 1a). In contrast, when a variable negatively influences HCI, herbivores cause stronger reductions of plant mass at high values of that variable (e.g. panel a, path 1b). CSC, carnivore standing crop; HSC, herbivore standing crop; PSC, plant standing crop; RDPSC, relative defended plant standing crop.

(Oksanen *et al.* 1981, 1995; Wootton & Power 1993). In other systems, however, NPP might not support herbivores and predators (pathway 1a-2a-3a) or NPP might positively and stably influence carnivores, preventing herbivores from increasing with NPP (pathway 1c-2c-3c) (Oksanen *et al.* 1981; Chase *et al.* 2000). Here, herbivore reduction of plant mass is expected to remain constant as NPP increases. Finally, herbivore reduction of plant mass might increase nonlinearly if NPP sequentially increases the number of trophic levels (curved pathway 1d-2d-3d, 4-6) (Oksanen *et al.* 1981). We find, however, that the consumer-controlled model poorly translates to a causal framework for some

systems. The SEM cannot detect an indirect effect of NPP on higher trophic levels when herbivores or predators prevent their resource from increasing (i.e. dashed-paths 1b and 2c).

The heterogeneous model (Fig. 1d) predicts that herbivore reduction of plant mass declines as NPP increases (Chase *et al.* 2000). This model assumes a positive influence of NPP on plant and herbivore standing crops (pathway 1-2). Herbivores, however, positively influence plant defences, namely resistance and tolerance (Holt *et al.* 1994; Leibold 1996; Chase *et al.* 2000). Resistance reduces herbivory (path 3b; e.g. trichomes or secondary chemicals) but herbivory still negatively influences plant mass (path 4b)

(Chase *et al.* 2000). Lowered damage on resistant plants also positively influences their relative abundance in the community (path 5b), which further reduces the negative impact of herbivory (path 6) (Holt *et al.* 1994; Leibold 1996; Chase *et al.* 2000). While tolerance does not reduce herbivory (path 3a), it reduces herbivory's negative impact on plant fitness (path 4a; e.g. herbivory stimulates photosynthesis, branching or reallocation of stored reserves) (Chase *et al.* 2000; Stowe *et al.* 2000). Here, greater herbivore damage favours tolerant plants, increasing their relative abundance (path 5a), further reducing herbivory's negative impact (path 6) (Chase *et al.* 2000).

Host concentration

This model assumes that increasing host species' absolute or relative stem density positively influences herbivore damage per host and reduces host mass (Fig. 1e) (Janzen 1970; Connell 1971; Carson & Root 2000; Long *et al.* 2003). This increases herbivore reduction of plant community mass as the host increasingly dominates the community (Carson & Root 2000; Long *et al.* 2003; Carson *et al.* 2004; see also Feeny 1976).

Hosts at high stem density might (1) be less chemically or visually masked by non-hosts; (2) create a favourable microclimate for herbivores; and/or (3) reduce predation risk (Root 1973; Hambäck & Beckerman 2003). Increasing host abundance therefore directly increases herbivore immigration, residence time, reproduction and survivorship (path 1), which increases herbivore damage per host (path 2) (Carson & Root 2000; Long *et al.* 2003). As host abundance increases, herbivores also have longer residence time per host and more easily locate the next host (Hambäck & Beckerman 2003). Therefore, increasing host abundance directly increases damage per host by increasing herbivore attack rates (path 3) (Hambäck & Beckerman 2003). Damage reduces per host mass (path 4) and, as the host's abundance in the community increases, increasingly determines community level mass (path 5) (Carson & Root 2000; Long *et al.* 2003).

Tissue nitrogen concentrations

This model assumes that increasing tissue nitrogen concentration increases or decreases herbivore damage and that herbivory reduces plant mass (Fig. 1f) (Mattson 1980; White 1984; Throop & Lerdau 2004). Thus, the indirect influence of tissue nitrogen concentration on herbivore control can range from positive to negative (Mattson 1980; White 1984; Throop & Lerdau 2004).

Nitrogen is a limiting resource for herbivores and high nitrogen concentrations could decrease allocation to carbon-based defences (Mattson 1980; White 1984; Throop & Lerdau 2004). Thus, nitrogen concentrations might increase herbivore damage by positively influencing herbivore preference, reproduction, and survivorship (solid-paths 1–2) and herbi-

vore attack rate (solid path 3) (Mattson 1980; White 1984; Awmack & Leather 2002; Throop & Lerdau 2004). In contrast, high nitrogen concentration could allow herbivores to meet their nitrogen requirements in fewer bites, cause plants to become toxic to herbivores or increase nitrogen-based defences (Mattson 1980; Throop & Lerdau 2004). These decrease herbivore damage both indirectly by decreasing herbivore abundance (dashed-path 1–2) and directly by decreasing attack rate (dashed-path 3) (Mattson 1980; Awmack & Leather 2002; Throop & Lerdau 2004). Regardless of whether per plant damage increases or decreases with tissue nitrogen concentration, herbivore reduction of per plant mass increases as per capita damage increases (path 4), reducing plant community mass (path 5) (Throop & Lerdau 2004). Finally, herbivore species certainly respond differently to nitrogen concentration, which could result in no direct (dotted-path 3) or indirect effect (dotted-path 1) of nitrogen concentrations on per plant damage (Throop & Lerdau 2004).

Plant growth rate

Here, herbivore damage increases with plant growth rate but herbivory has positive and negative affects on plant mass (Fig. 1g) (Coley *et al.* 1985; Price 1991; Cebrian & Duarte 1994; Stowe *et al.* 2000). Consequently, the indirect influence of plant growth rate on herbivore control ranges from negative to positive (Price 1991; Cebrian & Duarte 1994).

Slow growing plants often have traits that resist herbivore damage (e.g. tough leaves with low nitrogen content). In contrast, rapidly growing plants often have traits that encourage herbivore attack (e.g. soft leaves with high nitrogen content) (Coley *et al.* 1985; Wright *et al.* 2004; Blumenthal 2006; Poorter & Bongers 2006). Thus, plant quality increases with plant growth rate (path 1). Greater plant quality (i.e. lower defences and greater tissue nitrogen) increases herbivore damage both by increasing herbivore colonization, reproduction and survivorship (pathway 2–3) and by increasing herbivore attack rate (path 4) (Price 1991; Kursar & Coley 2003).

Because herbivory removes and damages plant tissue, herbivore reduction of per plant mass might increase with herbivore damage (dashed-path 5) (Cebrian & Duarte 1994). Alternatively, herbivore reduction of per plant mass might decrease with herbivore damage (solid-path 5). The low levels of herbivory experienced by a slow growing plant could be more costly than high levels of herbivory experienced by a rapidly growing plant (Coley *et al.* 1985; Price 1991). While slow growing plants are more resistant to herbivore damage, their traits can also confer lower tolerance per unit of herbivore damage (e.g. low photosynthetic capacity, large investment of resources in tissue). Similarly, rapidly growing plants are less resistant but have traits that might confer high tolerance of herbivore damage (e.g. high photosynthetic capacity, low investment in the tissue) (Coley

et al. 1985; Stowe *et al.* 2000; Wright *et al.* 2004; Blumenthal 2006; Poorter & Bongers 2006). Moreover, herbivory can increase plant growth by stimulating photosynthesis, branching and reallocation of stored resources (Price 1991; Stowe *et al.* 2000). The pattern of herbivore control will certainly depend on the relative importance of resistance vs. tolerance defence mechanisms and could result in no net influence of damage on herbivore control (dotted-path 5).

Plant size

Here, plant size positively affects herbivore damage, which decreases plant mass (Fig. 1h) (Feeny 1976; Hambäck & Beckerman 2003). Consequently, increasing plant size might increase herbivore reduction of plant mass.

Larger plants might be more apparent because neighbouring plants do not mask them (Feeny 1976; Hambäck & Beckerman 2003). Thus, herbivores might have increased discovery and residence time on larger plants, which increases herbivory by increasing herbivore attack rate (path 3) and by increasing herbivore abundance (pathway 1–2). Because herbivory removes or damages tissue, greater plant damage results in strong herbivore reduction of per plant mass (path 4) and therefore plant community mass (path 5).

Summary

The review above highlights two key points. First, most models assume that herbivore responses to vegetation characteristics explain herbivore control. The exception is the plant tolerance model. Second, these models rely on vegetation characteristics that are confounded (e.g. Appendix S1) and overlap in some assumptions (e.g. herbivores respond to tissue quality), making them difficult to test. Yet, these models embody distinct causal structures. We therefore view them as diagnostic tools that, when simultaneously tested, might guide further research. Our approach, outlined below, was to use experimental treatments to directly and indirectly vary the vegetation characteristics, quantify herbivore activity and control, and then use SEM to test whether these causal models explain the observed data.

METHODS

Study system and experimental design

We simultaneously conducted a monoculture experiment and a polyculture experiment from 2003 to 2004 in 4 m² mid-successional old-field plots at the University of Pittsburgh's Pymatuning Laboratory of Ecology (Linesville, PA, USA). In both experiments, we factorially manipulated total plant stem density (by thinning naturally occurring vegetation to 8, 16, 24 or 32 stems per m²), soil fertility (by

adding fertilizer at 0, 8 or 16 g N m⁻² year⁻¹), and herbivory (by pairing neighbouring plots and treating one with insecticide) (Appendix S2). Monocultures consisted of naturally extant *Solidago canadensis*, a native herbaceous perennial that dominates north-eastern North American old-fields (Carson & Root 2000). Polycultures consisted of naturally extant old-field herbaceous perennials, mostly *Solidago* and *Aster* species (Appendix S2). In polycultures, *S. canadensis* stem density was held constant at eight stems per m² as total plant species density increased. Thus, we tested host concentration mechanisms by manipulating stem density: in polyculture, we decreased relative host concentration while holding absolute host concentration constant. In monoculture, we increased absolute host concentration while holding relative host concentration constant (Appendix S2). We tested the tolerance model by increasing resource abundance via fertility and decreasing resource abundance via stem density's influence on resource competition (Appendix S2). We tested the plant size, plant growth rate, tissue nitrogen concentration and NPP models using variation in these vegetation characteristics that was generated by fertility and stem density (Appendix S2). Finally, the spray treatment quantified herbivore control by allowing comparisons of plant biomass in plots with herbivores to plots without herbivores.

We established the experiments by randomly assigning treatments to two hundred and ninety-four 4 m² plots (Appendix S2). Plots were selected from a matrix of six hundred 4 m² plots that were separated by 3–5 m buffers. Plot selection was based on pre-treatment plant density and heights, ensuring the experimental treatments were not confounded with pre-existing vegetation characteristics (Appendix S2). The entire site was fenced to exclude large herbivores, mainly white-tailed deer (*Odocoileus virginianus*).

Response variables

Vegetation

We quantified stem densities, per capita size, per capita mass, NPP, tissue nitrogen concentrations and herbivore control. First, we recorded the size of every stem in the centre 1 m² of each plot and estimated each stem's mass using species-specific allometric regressions (Appendix S2). Mean stem mass provided an estimate of growth rate. Aboveground mass per m² in spray plots provided an estimate of NPP. We calculated a Herbivore Control Index (HCI), which compares measures of either per capita mass or plant mass per m² in the presence of herbivores to the same mass measure in plots without herbivores (Appendix S2). HCI ranges from 1 to -1, where positive values indicate herbivory increased plant mass, 0 indicates herbivores did not change plant mass (i.e. compensation), and negative values indicate herbivores decreased plant mass.

We provide a detailed justification of these measures in Appendix S2.

Resource abundance

We measured light abundance in addition to imposing our soil fertility treatment. We quantified mean diffuse non-interceptance, which estimated the probability (0–1) that diffuse canopy light reached the soil surface in the centre of the plot (Appendix S2).

Herbivore and predator activity

Using techniques designed for our study system, we visually sampled herbivore abundance, herbivore damage to leaves (e.g. leaf galls, leaf chewers), herbivore damage to stems (e.g. stem galls, stem borers) and predator abundance (Appendix S2). Plot means were calculated as the number of herbivores or predators per plot or per stem, the average number of leaves damaged per stem and the percentage of stems with stem damage.

Statistical analyses

We separately analysed the monoculture and polyculture experiments. Analyses are based on year 2, when the experiments were fully established. To accept a causal model, we first required the SEM to be a good fit to the observed data. SEM is considered a statistically good fit when $\chi^2 P > 0.05$, standardized residual covariance < 1.96 and modification indices < 4 (Grace 2006). We then examined the significance (i.e. b 's P -value < 0.05) and direction of the regression coefficients for each pathway (e.g. does herbivory significantly increase as host concentration increases?). Finally, in search of generality, we only accepted the model if its mechanisms were supported in both monoculture and polyculture.

We removed stem density and fertility treatment variables from an SEM when they prevented it from being a good fit to the data (i.e. SEM $\chi^2 P > 0.05$ due to treatment's standardized residual covariance > 1.96). This provided rigorous tests of each model because model predictions are ultimately based on the vegetation characteristics, not always on the underlying experimental manipulations. We did not remove treatment variables from the plant tolerance and host concentration SEMs because testing these models directly relied on the experimental treatments. Prior to analyses we normalized distributions using transformations and we inspected bivariate correlations for nonlinearity. Although all the models, except Fig. 1c, assume linearity, we analysed each SEM including any observed nonlinear relationships. Linear terms were always significant, nonlinear relationships were rare and marginally significant, and comparing linear to nonlinear SEMs did not change the conclusions.

RESULTS

Resource-limited plant tolerance of herbivory

Our plant tolerance SEM tested for the influence of two resources on plant tolerance, soil fertility and light availability (Fig. 2a,b). We also included a direct effect of stem density to further account for the negative influence of resource competition on soil resources (Fig. 2a,b). In both experiments, light availability decreased as stem density increased (Fig. 2a,b). And, in both experiments, herbivore reduction of per plant mass was greatest under low light conditions reduction of per plant mass (Fig. 2a–d). Moreover, strong reduction of per capita biomass resulted in strong reduction of plant community level biomass in both monoculture (Fig. 2a,e) and polyculture (Fig. 1b,f). Light availability did limit per capita plant mass in both experiments (Appendix S2). Thus the cumulative impact of herbivory was to limit the ability of plants to acquire or use carbon and this has a stronger negative influence on plant growth when light becomes limiting. Soil fertility did not directly influence herbivore control nor did stem density directly influence herbivore control (Fig. 1a,b). This suggests that although fertility limited per capita plant growth (Appendix S2), herbivory did not limit the ability of plants to acquire or use soil resources (Wise & Abrahamson 2007).

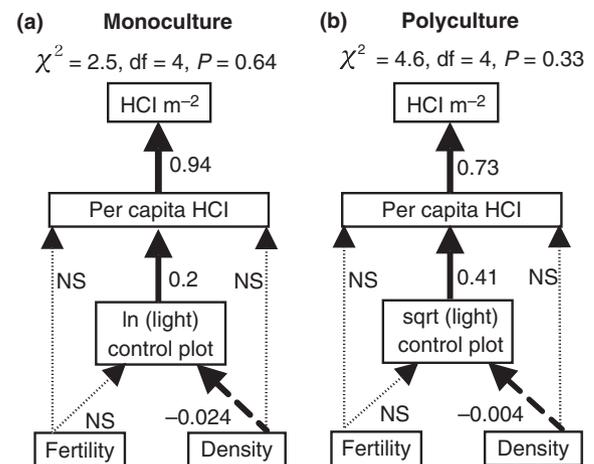


Figure 2 Host tolerance structural equation modelling results for monoculture (a) and polyculture (b). Solid paths indicate a significant positive relationship; dashed paths indicate a significant negative relationship; dotted paths indicate a non-significant relationship (n.s. = $P > 0.05$). Path values are structural equation modelling-generated b 's [e.g. if monoculture light increases by 1.0, per capita Herbivore Control Index (HCI) increases by 0.2]. Path size illustrates R^2 . Recall from Fig. 1 that positive correlations (i.e. solid path) between light and per capita HCI indicate that herbivores cause greater reductions of plant mass at low light availability. HCI values ranged from negative to positive (see Appendix S1, S3).

Net primary productivity

Relationships between NPP and plant standing crops or between herbivore and carnivore abundances were not nonlinear (Appendix S3). We therefore rejected nonlinear consumer-controlled mechanisms and tested the linear SEM structure (Fig. 3a,b). This SEM was a poor fit in both experiments (Fig. 3a,b). The poor fit occurred because NPP left significant unexplained variation in herbivore control and damage (Appendix S3). This does not necessarily reject either model: NPP might inadequately explain variation in herbivore damage or control if NPP does not support herbivores (i.e. consumer-controlled), if herbivore damage does not increase despite NPP indirectly increasing herbivore abundance (i.e. resource-controlled), or if NPP indirectly and stably increases carnivores, which prevent herbivores from increasing (i.e. consumer-controlled). Both herbivore and carnivore trophic levels were present across the entire NPP gradient (Appendix S3). Therefore, in accordance with these models, we reanalysed the SEMs but excluded herbivore control and damage to leaves and/or stems.

The reduced SEMs suggest that increasing NPP in this system increases plant abundance (Fig. 3a,b), which either does not or negatively influences herbivore abundance (Fig. 3a,b). The reduced models also suggest that herbivore abundance can positively influence herbivore damage per plant (Fig. 3b) and carnivore abundance (Fig. 3a,b). These results strongly reject the resource-controlled model, where NPP positively influences all standing crops and consumer damage does not increase with consumer abundance. Despite an inability to detect a potential indirect effect of NPP on carnivores in polyculture (Fig. 3b), these results also reject a consumer-controlled model. NPP had an indirect negative effect on herbivore abundance (Fig. 3a). No NPP model, however, assumes that NPP will indirectly decrease herbivore abundance. We therefore note that herbivore abundance even tended to decline in polyculture though this was marginally non-significant (Fig. 3b).

We could not test part of the heterogeneous model. Our weeding protocol maintained specific plant species compositions, eliminating the possibility that herbivores might shift plant species composition. However, shifts in plant defence can only occur if herbivore abundance increases as plant abundance increases (Fig. 1d). We did not observe this: increasing plant abundance either decreased or tended to decrease herbivore abundance (Fig. 3a,b). This strongly rejects the heterogeneous model's key causal mechanism. These conclusions are further supported by conventional, non-causal tests of NPP models (Appendix S3).

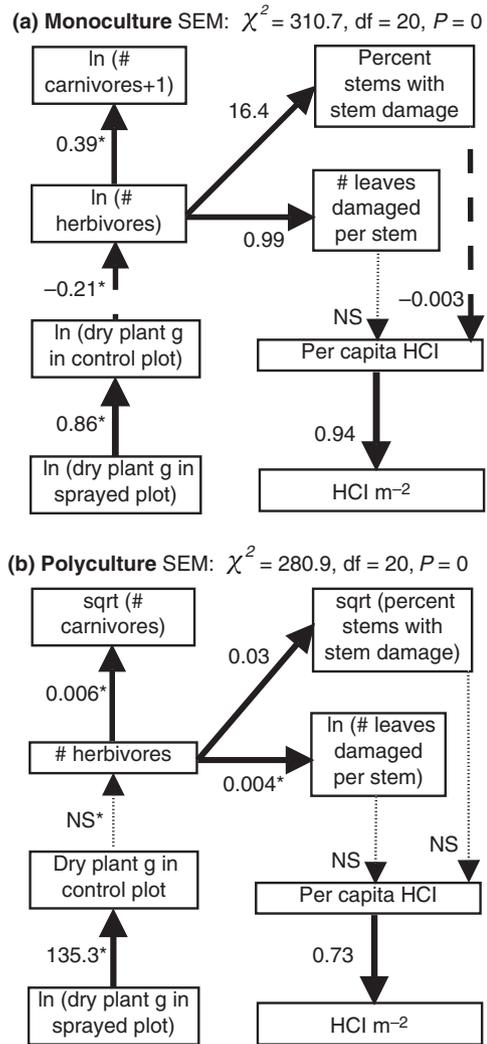


Figure 3 Net primary productivity (NPP) structural equation modelling (SEM) results for monoculture (a) and polyculture (b). NPP mechanisms were rejected: nonlinear mechanisms were rejected because there were no nonlinear relationships between NPP and plant standing crop or between herbivore and predator abundance (Appendix S3). Linear SEMs did not fit because NPP left significant unexplained variation in herbivore damage and control (Appendix S3). In accordance with resource- and consumer-controlled models, we reanalysed the SEMs without these variables. *Indicates the path was included in the reduced model and that its significance and *b* did not differ between the full model and the reduced model. The reduced models fit the observed data (monoculture: $\chi^2 = 2.4$, $d.f. = 3$, $P = 0.49$; polyculture: $\chi^2 = 5.5$, $d.f. = 6$, $P = 0.49$). In both experiments, NPP did not indirectly increase all standing crops, which rejects resource-controlled mechanisms. NPP indirectly reduced herbivore abundance in monoculture (NPP Indirect Effect: -0.18) and tended to indirectly reduce herbivore abundance in polyculture (herbivore abundance on plant abundance: $b = -0.08$, $P = 0.07$; NPP Indirect Effect = -10.8), rejecting consumer-controlled and heterogeneous mechanisms. Legend same as in Fig. 2.

Host concentration

To test host concentration mechanisms, we focused on herbivore responses to and control over the dominant species in our study system, *S. canadensis*. We found that herbivore abundance per *S. canadensis* increased as relative *S. canadensis* stem density increased in polyculture (Fig. 4b). As herbivore abundance per *S. canadensis* increased, both leaf and stem damage per *S. canadensis* increased (Fig. 4b). While leaf damage did not have a significant effect on herbivore control over *S. canadensis* (Fig. 4b), herbivore reduction of per *S. canadensis* mass increased as stem damage increased (Fig. 4b). Further, herbivore reduction of per *S. canadensis* mass was a good predictor of herbivore control at the community level (Fig. 4b). Thus, stem damage in polyculture, but not leaf damage, seems to support a relative host concentration mechanism.

In monoculture, as in polyculture, leaf damage did not have a significant effect on per *S. canadensis* stem mass (Fig. 4a). Thus, a leaf damage pathway does not support an absolute host concentration mechanism. The only other significant pathway linking absolute host concentration to herbivore damage was negative; herbivore abundance per *S. canadensis* stem actually declined as absolute *S. canadensis* stem density increased (Fig. 4a). Thus, we did not find any evidence in monoculture to support the absolute host concentration mechanism.

The monoculture results cast doubt on the relative host concentration mechanism as a cause of the polyculture results. If herbivore activity on *S. canadensis* is determined by the extent to which it is surrounded by non-hosts, then herbivore activity on *S. canadensis* should not vary in monoculture because non-hosts are not present. Yet herbivore abundance per *S. canadensis* stem in monoculture still declined as absolute stem density increased (Fig. 4a). This clearly requires a causal explanation beyond relative host concentration mechanisms. It suggests that herbivores responded to something positively correlated with relative host concentration in polyculture and negatively correlated with absolute host density in monoculture. Our results show this variable was tissue nitrogen concentration (see section Plant tissue nitrogen concentrations). Moreover, the plant tolerance model explains herbivore control in both experiments. Thus, the causal mechanisms assumed by the host concentration model do not provide a robust, general explanation for herbivore control in this system.

Plant tissue nitrogen concentrations

In both experiments, herbivore abundance was positively correlated with leaf nitrogen (Fig. 5a,b) and more leaves were damaged per stem as herbivore abundance increased (Fig. 5a,b). The number of leaves damaged per stem,

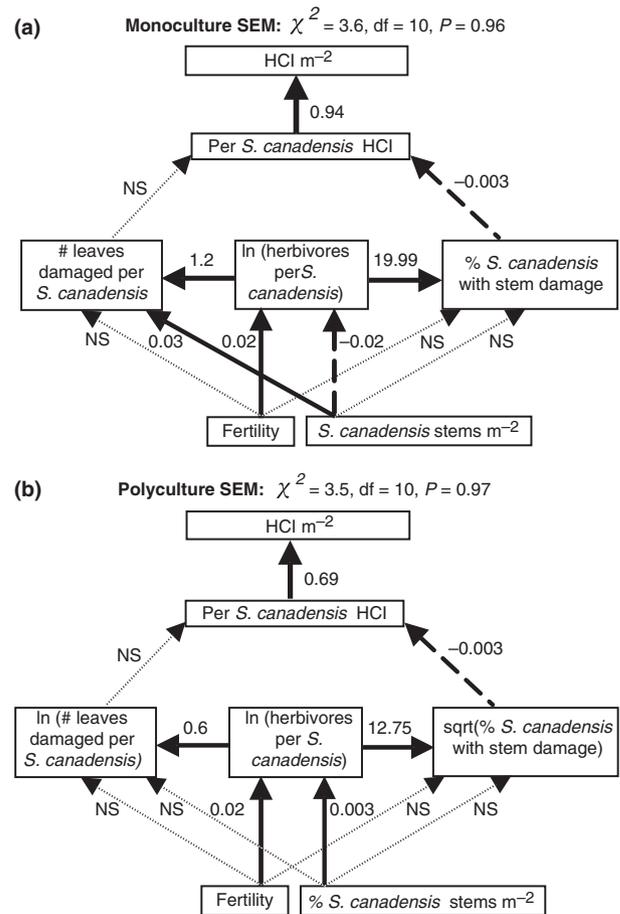


Figure 4 Host concentration structural equation modelling results for monoculture (a) and polyculture (b). Host concentration mechanisms were rejected. In monoculture, herbivore abundance should have either increased with absolute host stem density (predicted by the absolute host concentration mechanism) or remained constant (predicted by the relative host concentration mechanism). Herbivore abundance, however, declined as absolute host stem density increased and even though relative host concentration remained constant (b). Thus, even though herbivore abundance per *Solidago canadensis* increased with relative *S. canadensis* density in polyculture (c) (predicted by the relative host concentration mechanism), the monoculture result clearly rejects a relative host concentration mechanism for our system. Legend same as in Fig. 2.

however, did not influence herbivore control in either experiment (Fig. 5a,b).

Leaf nitrogen positively influenced stem damage in polyculture, but stem damage did not explain herbivore control (Fig. 5b). In monoculture, two stem damage pathways link leaf nitrogen to herbivore control (Fig. 5a: leaf nitrogen → stem damage → per capita herbivore control; leaf nitrogen → herbivore abundance → stem damage → per capita herbivore control). The ‘combined’ or ‘total’ effect of leaf nitrogen on stem damage was

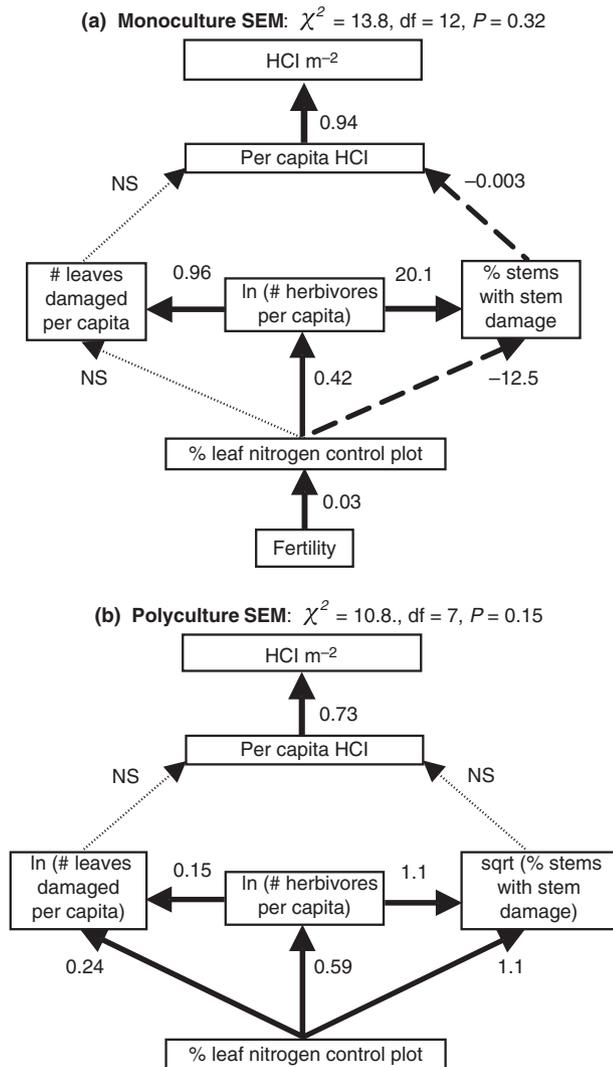


Figure 5 Plant tissue nitrogen structural equation modelling results for monoculture (a) and polyculture (b). The tissue nitrogen model was rejected. Tissue nitrogen concentration may explain patterns of herbivore control over a single species (a) and may even explain herbivore activity in complex communities (b). It did not, however, explain herbivore control over complex plant communities (b). The total effect of leaf nitrogen on stem damage was negative in monoculture (Total Effect = -4.057). Therefore, herbivore reduction of plant mass in monoculture was greatest at low leaf nitrogen. Legend same as in Fig. 2.

negative (Total Effect = -4.057) and its total effect on herbivore control over monocultures was very weak (Total Effect = 0.005). Thus, the evidence that leaf nitrogen explains herbivore control is not strong; we found either no relationship (polyculture) or a weak relationship (monoculture) between leaf nitrogen and herbivore control. Tissue nitrogen however, can explain herbivore activity (Fig. 5). Tissue nitrogen is also negatively correlated with absolute host concentration in monoculture and positively correlated

with relative host concentration in polyculture (Appendix S1). Thus, tissue nitrogen likely explains the relationships between host concentration and herbivore activity.

Plant growth rate

The growth rate SEM was not a statistically good fit in monoculture ($\chi^2 = 32$, d.f. = 12, $P = 0.001$) or polyculture ($\chi^2 = 57.6$, d.f. = 12, $P = 0$) (Appendix S4). Again, the poor fit occurs because of significantly large unexplained variation in herbivore activity and/or herbivore control (Appendix S4). Resistance and tolerance mechanisms, however, might equally reduce herbivore control, preventing herbivore control from varying with plant growth rate. Thus, we reanalysed the SEMs but excluded herbivore control indices. The reduced SEM fit the observed data in monoculture but, in contrast to the growth rate model, growth had a net negative effect on stem damage (Appendix S4). In addition, growth still provided an inadequate explanation for herbivore activity in polyculture (Appendix S4). In total, the plant growth rate model does not adequately explain herbivore control.

Plant size

The plant size SEM also was not a statistically good fit in either monoculture ($\chi^2 = 19.2$, d.f. = 7, $P = 0.007$) or polyculture (polyculture: $\chi^2 = 28.7$, d.f. = 7, $P = 0$) (Appendix S5). Once again, the model structure resulted in significantly large unexplained variation in herbivore control (Appendix S5). Thus, plant size models also provide an inadequate explanation.

DISCUSSION

Our results show that plants with greater access to limiting resources better tolerated herbivory. Consequently, herbivore reduction of plant community mass was greatest under low limiting resource availability. Moreover, our results clearly reject all other prominent models. While we focused on insect herbivores, which can strongly impact plant communities, explanations for vertebrate and pathogen control invoke similar mechanisms (McNaughton 1979; Augustine & McNaughton 1998; Chase *et al.* 2000; Mitchell 2003; Jones *et al.* 2008).

Ecologists have been concerned that community-level patterns might not be predicted from patterns at the individual level due to emergent properties (i.e. higher order interactions) (Fowler 1981; Wilbur & Fauth 1990). There is, however, a growing body of research indicating that patterns in complex communities can be understood from the processes operating at the individual level (Fowler 2002; Poorter & Bongers 2006). Here, we demonstrate that the

effect of resource availability on the tolerance of individuals explained herbivore control at the community level.

The vast majority of trophic interaction models assume correlations between herbivore demography, damage to plants and herbivore control. Our results strongly suggest that herbivore activity is best explained by host-level mechanisms, namely tissue nitrogen concentrations (Fig. 5) and not community-level mechanisms such as NPP (Fig. 3) or host concentration (Fig. 4). We found, however, that models that include herbivore activity did not explain variation in herbivore control. Meta-analyses have also shown that herbivore activity poorly predicts herbivore control (Schmitz *et al.* 2000; Halaj & Wise 2001). These results suggest that the key 'herbivore' question is not about herbivore activity *per se*, but whether the herbivore community's cumulative impact is to prevent plants from acquiring or using a limiting resource.

Empirical evidence suggests that plant tolerance should play a more prominent role in community and ecosystem theory. The tolerance model accurately predicts herbivore control across a diversity of plant traits (Wise & Abrahamson 2007). Even in our experimental communities, which differed in species' relative abundances, resource availability increased tolerance despite trait differences amongst species. Furthermore, plant traits thought to influence tolerance generally covary according to the growth rate model (Coley *et al.* 1985; Wright *et al.* 2004; Poorter & Bongers 2006). Thus, plant tolerance could influence the pattern of herbivore control when the species pool's trait composition is drastically different at the opposite ends of resource availability gradients (e.g. light gradients in forest communities). Undoubtedly, other mechanisms (e.g. predation, abiotic factors, outbreaks) can be important in this and other systems (Carson & Root 2000; Ritchie 2000; Schmitz *et al.* 2000; Halaj & Wise 2001; Oviada & Schmitz 2004). We encourage future theory to consider that these processes operate on a fundamental relationship between per-capita resource availability and plant tolerance to enemies.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Appendix S1 Data range for each vegetation characteristic and bivariate correlations among the key vegetation characteristics in our experimental monoculture and polyculture 4 m² field plots.

Appendix S2 Expanded methods.

Appendix S3 Conventional NPP tests.

Appendix S4 Plant growth rate SEM results for monoculture (a) and polyculture (b).

Appendix S5 Plant size SEM results for monoculture (a) and polyculture (b).

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