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A comprehensive test of the 'limiting resources' framework applied to plant tolerance to apical meristem damage

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Tolerance to apical meristem damage (AMD) is a form of plant defense against herbivory. Theoretical models come to different conclusions about the effects of inorganic soil nutrient levels on tolerance to AMD, and different plants have shown different relationships between these variables. To assign some order to these disparate patterns and to resolve conflicts among the models, the 'limiting resources model' (LRM) was developed. However, we believe that the LRM is actually comprised of several different models, which we describe. Our study marks the first comprehensive and simultaneous test of the entire LRM framework, treating it explicitly as separate models, which also evaluates the models' underlying assumptions. We studied tolerance to AMD in laboratory-reared natural populations of *Arabidopsis thaliana* from three different regions of Europe, spanning a wide latitudinal gradient. We show that, in different populations of this species, basic responses to nutrients and damage are best described by different models, which are based on different assumptions and make different predictions. This demonstrates the need for complexity in our explanations, and suggests that no one existing model can account for all relationships between tolerance to AMD and nutrients. Our results also demonstrate that fruit production can provide a misleading approximation of fitness in *A. thaliana*, contrary to the common assumption in the literature.

Herbivory plays a major role in plant evolution (Painter 1958, Marquis 1992, Abrahamson and Weis 1997, Juenger and Lennartsson 2000, Stowe et al. 2000). There are two ways that plants cope with herbivory: by having a phenotype that decreases the likelihood of being grazed (resistance), and by having the ability to recover from tissue loss and damage (tolerance; Rausher 1992). Although there has been extensive research on the evolution of resistance to herbivory, the evolution of tolerance has received comparatively less attention (Tiffin and Rausher 1999, Juenger and Lennartsson 2000, Stowe et al. 2000) because it has generally been assumed to be important only in perennial plants, where below-ground resources may be shunted above ground in response to tissue damage (van der Meijden et al. 1988, Belsky et al. 1993, Stowe et al. 2000), and in grasslands and other areas of exceptionally high, predictable herbivory (Juenger and Lennartsson 2000).

Despite this prevailing notion, research on the response of plants to a specific type of vertebrate herbivory, apical meristem damage (AMD), indicates that tolerance to AMD is more prevalent and can play a greater role in plant evolution than this conventional wisdom suggests. Several studies have demonstrated that monocarpic plants can recover substantially from AMD (reviewed by Stowe et al. 2000, Hawkes and Sullivan 2001, Wise and Abrahamson 2007) even if it inflicts a loss of as much as 95% of their aboveground biomass (Paige and Whitham 1987). Surprisingly, some plants seem to have higher fitness when damaged than when they remain undamaged (Paige and Whitham 1987, Paige 1994, 1999, Lennartsson et al. 1997, 1998, Juenger et al. 2000). Some researchers, however, question these results (Belsky 1986, Bergelson and Crawley 1992a, 1992b, Belsky et al. 1993, Bergelson et al. 1996), and contend that there is no unequivocal evidence that plants can benefit from herbivory in most natural settings (but see Tuomi et al. 1994, Agrawal 2000). Therefore, to clarify whether tolerance to AMD is important in natural populations, it is necessary to understand the conditions under which different levels of tolerance to AMD are expressed.

When a plant has bolted, AMD involves the consumption of inflorescence tissue, although it can also be caused by abiotic stressors such as frost (Belsky 1986, Paige and Whitham 1987, Juenger et al. 2000, Tiffin 2000). AMD releases plants from apical dominance, and thus basal inflorescences may proliferate (Paige and Whitham 1987, Benner 1988, Mopper et al. 1991, Huhta et al. 2000a, Juenger et al. 2000). This can result in different degrees of tolerance, ranging from decreased fitness (undercompensation), to no change in fitness (exact compensation), to an increase in fitness (overcompensation) when damaged as

compared to when undamaged, as indicated by the slope of the reaction norm for fitness across apically damaged and undamaged treatments (Juenger et al. 2000, Mauricio 2000, Simms 2000, Stowe et al. 2000). While tolerance to other forms of damage (e.g. the leaves or roots) is sometimes measured by biomass or tissue growth plasticity (Mauricio et al. 1997, Hochwender et al. 1999, Stowe et al. 2000), tolerance to AMD is most appropriately measured by its fitness consequences, since inflorescence development, mediated by meristems, determines reproductive fitness, and the ability to mitigate/maintain fitness despite damage is the most ecologically and evolutionarily relevant aspect to the phenomenon (Weinig et al. 2003).

Theoretical models of the growth responses to plant damage make conflicting predictions about the effects of inorganic soil nutrient availability on tolerance to AMD. Specifically, the 'compensatory continuum hypothesis' predicts tolerance to AMD will increase (Maschinski and Whitham 1989), whereas the 'growth rate model' predicts it will decrease (Hilbert et al. 1981), with increasing nutrient levels. Both models have been supported by particular studies (Hawkes and Sullivan 2001, Wise and Abrahamson 2005), suggesting that neither model by itself gives the full picture of how tolerance and nutrients are related. To rectify this problem and assign some order to the disparate patterns being observed, the 'limiting resources model' (LRM; Wise and Abrahamson 2005, 2007, 2008) was developed. Its central premise is simple – tolerance to AMD depends on whether a 'focal resource' (soil nutrients) or an 'alternate resource' (shoot meristems) limits fitness (see also Rautio et al. 2005, who hypothesized the same general framework as the LRM, although less explicitly) – although applying the model is complex (Wise and Abrahamson 2008).

The LRM provides a broad conceptual structure that encompasses contrasting assumptions (i.e. nutrient levels vs the active shoot meristems as limiting factors affecting plant fitness in various circumstances; Box 1) and therefore makes a wide variety of predictions regarding tolerance in different environments. As a result of this flexibility, the model can be fit to most data sets (Wise and Abrahamson 2007, 2008). We believe, however, that this flexibility results from 'the' LRM actually representing a set of models, as individual parts of the LRM (represented by forks in a decision tree in Wise and Abrahamson 2005 and by the steps in a dichotomous key in Wise and Abrahamson 2007, 2008) use opposite assumptions to make different predictions of the relationship between tolerance and nutrient levels (Box 1). Because a model is defined by its assumptions and corollary predictions, this makes the LRM plural by definition. Interestingly, when the LRM is re-cast as a set of models (which we call the LRM framework), one can see that a particular model from the LRM framework (which we call LRM-IV; Box 1) is actually the aforementioned compensatory continuum hypothesis itself; this has not been pointed out before, to the best of our knowledge.

We believe that the LRM framework should be explicitly treated as a heterogeneous class made up of separate models, rather than as a single model. In this study we evaluated all of the models comprising the LRM, and the individual models' assumptions, marking the first comprehensive test of the entire LRM framework that treats all of the possible decisions about the biological/ecological assumptions (i.e. the indented levels of the dichotomous key in Box 1) as different models, and that evaluates all of the models, and all of their assumptions, on equal footing against the data. We used the plant *Arabidopsis thaliana* (Brassicaceae) for this study, because it offers an excellent opportunity to assess the fitness effects of AMD and how they relate to nutrient levels. It has the appropriate inflorescence architecture, and this form of damage has been documented in the field for this species (Weinig et al. 2003). It is small, grows readily, and has a short life cycle, allowing large experiments under controlled conditions. Furthermore, eventually the underlying molecular mechanisms for tolerance to AMD in *A. thaliana* could be studied, because the biology of this species is extremely well known and excellent genomic tools are available for its characterization (Pang and Meyerowitz 1987, Anderson and Roberts 1998, Krysan et al. 1999, Mitchell-Olds and Schmitt 2006).

Previous work using recombinant inbred lines and single lines from different natural populations (Weinig et al.

Box 1. List of the models from the 'limiting resources' framework, organized by their assumptions into a dichotomous key, adapted from Wise and Abrahamson (2008). We named the models (in parentheses) to match Fig. 2 from Wise and Abrahamson (2005).

1. Soil nutrients are limiting fitness in undamaged plants at low nutrient levels.
    2. Apical meristem damage primarily affects use/acquisition of soil nutrients.
    ..... **Higher tolerance at high nutrient levels (model LRM-I)**
    2′. Apical meristem damage primarily affects the number of active shoot meristems.
        3. The number of active shoot meristems is not limiting plant fitness at high nutrient levels.
        ..... **Same amount of tolerance at both nutrient levels (model LRM-II)**
        3′. The number of active shoot meristems is limiting fitness at high nutrient levels.
            4. Apical meristem damage exacerbates the limitation of the number active shoot meristems.
            ..... **Lower tolerance at high-nutrient levels (model LRM-III)**
            4′. Apical meristem damage ameliorates/removes the limitation of the number of active shoot meristems
            ..... **Higher tolerance at high-nutrient levels (model LRM-IV, also known as the Compensatory continuum hypothesis)**
1′. Soil nutrients are not limiting fitness in undamaged plants at low nutrient levels.
    5. Apical meristem damage primarily affects use/acquisition of soil nutrients.
        6. Apical meristem damage causes soil nutients to become limiting.
        ..... **Higher tolerance at high nutrient levels (model LRM-V)**
        6′. Apical meristem damage does not cause soil nutrients to become limiting.
        ..... **Same amount of tolerance at both nutrient levels (model LRM-VI)**
    5′. Apical meristem damage primarily affects the number of active shoot meristems.
    ..... **Same amount of tolerance at both nutrient levels (model LRM-VII)**

2003, Banta and Pigliucci 2005) has demonstrated that *A. thaliana* maternal seed families can be very tolerant to AMD. A next reasonable step to augment this information is to describe the standing genetic variation in tolerance to AMD, to determine the effects of nutrient variation on it, and to evaluate how the choice of the fitness-related trait used as a stand-in for true fitness affects the perceived patterns.

Because *A. thaliana* displays large amounts of phenotypic, even life-history, variation (Pigliucci 2002a, Griffith et al. 2004, Banta et al. 2007), we used multiple *A. thaliana* maternal seed families collected from multiple natural populations to investigate the possibility that different models from the LRM framework best explain the relationship between tolerance and nutrient levels in different accessions. If true, this could be problematic for the idea of generalizing the relationship between tolerance and resource availability. We used populations collected across a wide latitudinal gradient in western Europe – encompassing northern Spain, the Netherlands and southern Sweden – rather than laboratory strains, so as to make our results more representative of the species in the wild.

In addition to using the standard count of fruit production as an estimate of true fitness, we also used a measure arguably more closely related to fitness, the estimated total viable seed production. While Westerman and Lawrence (1970) found a strong relationship between fruit and overall seed production in *A. thaliana*, their study used laboratory lines and did not examine the relationship between fruit number and the more relevant viable seed production. We assessed whether the choice of fitness-related trait, fruit production versus estimated total viable seed production, alters conclusions as to which model(s) are best supported by the data.

Material and methods

Plant material, handling and experimental protocol

Maternal seed families of *Arabidopsis thaliana* were collected from populations in three different regions along a broad latitudinal gradient in western Europe during the spring of 2001 (Table 1). Although *A. thaliana* is a ruderal species often associated with disturbances (Napp-Zinn 1985), the populations were selected to be sufficiently far from roadsides, railroads and footpaths so that they could reasonably be considered 'natural'.

We generally followed the guidelines for germination and growth of *A. thaliana* recommended by the Arabidopsis Biological Resource Center (2008). We germinated seeds under laboratory conditions and used seeds produced by these plants in our experiment to minimize maternal effects due to collection environment. We imbibed the seeds with water on moist filter paper in 16×50 mm tissue culture dishes and exposed them to a seven-day dark stratification treatment at 4°C to facilitate germination. We then planted them in $3.25 \times 3.25 \times 5$ cm pots on two high-intensity light racks (approximately 250 $\mu E\ m^{-2}\ s^{-1}$ photon flux). While the light intensities were reasonably uniform, the experimental design was fully randomized to prevent any spatial heterogeneity from having a confounding effect. We also rotated the three shelves within each rack weekly to further homogenize the light conditions.

Table 1. *Arabidopsis thaliana* maternal seed families used in this study (third column), organized by region (first column) and population of origin (second column). The Arabidopsis Seed Stock Center numbers (<www.arabidopsis.org>) are provided, if available (fourth column).

Region	Population	Family	Stock No.
N. Spain	SP1	SP1.6	-
N. Spain	SP1	SP1.8	CS76007
N. Spain	SP1	SP1.13	CS76008
N. Spain	SP5	SP5.5	CS75807
N. Spain	SP5	SP5.6	CS75808
N. Spain	SP5	SP5.7	CS75809
N. Spain	SP6	SP6.1	CS75813
N. Spain	SP6	SP6.2	CS75815
N. Spain	SP6	SP6.7	CS75818
N. Spain	SP8	SP8.1	CS75822
N. Spain	SP8	SP8.7	CS75825
N. Spain	SP8	SP8.8	CS75826
Netherlands	NL3	NL3.3	CS75841
Netherlands	NL3	NL3.4	-
Netherlands	NL3	NL3.8	CS76078
Netherlands	NL5	NL5.6	CS75849
Netherlands	NL5	NL5.7	CS76074
S. Sweden	SW1	SW1.1	CS75860
S. Sweden	SW1	SW1.5	-
S. Sweden	SW2	SW2.4	-
S. Sweden	SW2	SW2.7	CS75862
S. Sweden	SW7	SW7.1	-
S. Sweden	SW7	SW7.2	-
S. Sweden	SW7	SW7.3	-

Rather than using standard potting soil, we employed a 50:50 mixture of river sand and vermiculite to ensure low baseline soil nutrient levels. When the seeds in a pot failed to germinate, we transplanted a seedling of the same maternal seed family into that pot. The seedling came either from another pot with extra germination or from extra seeds left over from planting, which had germinated in the tissue culture plates and had been kept moist at room temperature.

We set the photoperiodic regime to that typical of the Netherlands, roughly in the middle of the geographic range from which these populations were collected. During seed germination, we set the photoperiod to 15 September (12 h and 44 min), approximately when winter annual ecotypes of *A. thaliana* would be expected to germinate in the field, and used room temperature (around 25°C) during daytime and nighttime, which facilitates germination (*Arabidopsis* Biological Resource Center 2008). Twenty-five days later, we placed the plants into a walk-in refrigerator for rosette vernalization, with a daytime/nighttime temperature of 4°C and a photoperiod of 4 January (7 h and 47 min). After six weeks, we returned them to room temperature during daytime and nighttime and set the photoperiod to 30 April (14 h and 52 min). These conditions are a compromise between the need for as natural a setting as possible and the inevitable logistical limitations of experimental designs.

Our experiment included plants from three regions (northern Spain, the Netherlands, and southern Sweden), within which we had sampled four populations from northern Spain (three molecularly distinct maternal seed families per population; Cruzan et al. pers. com.), two populations from the Netherlands (three seed families in

one population and two in the other), and three populations from southern Sweden (two seed families in two populations and three seed families in the other). The number of replicates for each family-treatment combination averaged 6.5 and there were a total of 625 viable plants.

We added nutrients in the form of 14-14-14 time-release nitrogen-phosphorus-potassium prills applied to the sand-vermiculite surface. All plants received one prill 11 days after planting. The high-nutrient plants received another seven prills 23 days after planting, about the time the first true leaves appeared. There was some variation in prill size, which probably contributed somewhat to the residual variance in the analyses. Due to the desiccation caused by the fluorescent lights and the poor water-retention ability of the sand-vermiculite mixture, we sub-irrigated the plants twice daily. Therefore, the prills may have expunged their nutrients faster than the three-to-four month time interval indicated by the manufacturer. In spite of this variation, the nutrient levels were still quite high in the high-nutrient treatment, and were sufficient for growth in the low-nutrient treatment, approximately three months into the experiment (average low nutrients 7:18:129 ppm NPK; average high nutrients 50:46:180 ppm NPK).

Clipping for AMD was done at the time of bolting, when the inflorescences were in the unopened flower bud stage. The entire inflorescence was clipped off at the base of the rosette with scissors while we were careful not to remove or damage any rosette leaves. All plants that survived germination and/or transplanting bolted.

After the reproductive period, we measured the following traits that were most likely to have been affected by AMD and the interaction of AMD with nutrient levels: 1) number of basal inflorescences, the inflorescences growing out of the rosette; 2) number of lateral branches, the secondary and higher-order branches off of the basal inflorescences; 3) number of fruits, an estimate of lifetime reproductive fitness; 4) estimated number of total viable seeds, an integrated assessment of lifetime reproductive fitness, calculated as fruit production times the average number of seeds per fruit (determined from a sample of five fruits per plant) times the proportion of viable seeds (determined from a sample of 20–40 seeds tested for germinability per plant); when a plant did not produce fruits, produced fruits but no seeds, or produced non-viable seeds, this resulted in the number of viable seeds being zero.

Data analysis

Analyses of variance

We analyzed the data with a mixed model analysis of variance (ANOVA) for the following traits: number of basal inflorescences, number of lateral stems, bolting to fruit ripening time, number of fruits, and total viable seed production. We improved normality, homoscedasticity and kurtosis (Sokal and Rohlf 1995) by performing the following data transformations: $\log_{10}$ transformation of number of basal inflorescences, number of lateral branches, fruit ripening time and estimated total viable seed production. Analyses were performed with JMP IN ver. 5.1 using the method of moments approach (SAS Inst. 2003). For each trait, the full model included: region (fixed effect), population nested within region (random effect), maternal seed family nested within population within region (random effect), nutrient levels (fixed effect), AMD treatment (damaged vs undamaged; fixed effect), all possible interaction effects among those factors, light rack (overall effects of one light rack vs the other; fixed effect), and transplant status (non-transplants, pot-to-pot transplants, or petri dish to-pot transplants; fixed effect). For the model for fruit ripening time we excluded the family by treatment interactions because some plants failed to reach maturity after bolting, and therefore not all maternal seed families were represented by every nutrient level-AMD treatment combination for this trait.

We eschewed Bonferroni correction, which is often applied to maintain the overall probability of committing a type I error, because it unacceptably increases the probability of type II errors. Following Moran (2003), we instead report the probability of finding a particular number of significant test results (what we call Moran's p) using the equation:

$$p = \left(\frac{\mathrm{N!}}{\mathrm{N - K}}\mathrm{K!}\right)\alpha^{\mathrm{K}}(1-\alpha)^{\mathrm{N-K}}$$

where K refers to the α value (0.05), and N is the number of tests performed under the null hypothesis of no true effect (for examples see Bossdorf et al. 2004, Muth and Pigliucci 2007).

In order to account for the possibility that our results might not reflect natural patterns and instead derive from growing the plants in non-native photoperiodic regimes, we determined whether rosette size affects the response to AMD. An *A. thaliana* rosette responding to novel vernalization or photoperiodic regimes might grow more, or fewer, rosette leaves than it would in its native environment, leading to a concomitant increase, or decrease, in the number of quiescent shoot meristems able to respond to AMD. By determining whether rosette size affects the multivariate response to AMD, we tested whether our findings were sensitive to rosette size, which might be altered by the experimental conditions as compared to native conditions. We re-ran the models for the number of basal inflorescences, fruit production and total viable seed production in an analysis of covariance (ANCOVA; Sokal and Rohlf 1995) using rosette size as the covariate. To best represent rosette size, we performed principal components analysis on the covariance matrix of standardized values (Dillon and Goldstein 1984, p. 36) of rosette diameter and the number of rosette leaves at bolting, and used the first principal component (Somers 1989), which accounted for 83% of the variation, as a compound measure of rosette size.

Comparisons of the LRM models

To discriminate among the models making identical predictions and thereby to determine whether our data support any of the models, we evaluated their assumptions (Box 1). For instance, consider a scenario where the observed outcome is higher tolerance at high nutrients. LRM-I and LRM-IV both predict this outcome, and both assume that soil nutrients are limiting fitness in undamaged plants (Box 1, assumption 1). To test that assumption, we would examine the reaction norm across nutrient levels for fitness (according to our presumably best estimate, total

viable seed production); if fruit production increased at high nutrient levels, then we would consider this assumption accurate. The assumptions of LRM-I and LRM-IV part ways thereafter: LRM-I assumes that soil that AMD primarily affects the use/acquisition of soil nutrients (Box 1, assumption 2), whereas LRM-IV assumes that AMD affects the number of active shoot meristems rather than the use/acquisition of soil nutrients (Box 1, assumption 2'). Because AMD damages the apical meristem, and does not affect the amount of nutrients in the soil or, presumably, the plant's vascular system for acquiring/using those nutrients, LRM-I can be ruled out a priori.

Although LRM-I can be ruled out, LRM-IV does not win by default; rather, it has further assumptions that would need to be investigated to judge it congruent with the data. Specifically, we would investigate the assumptions that AMD primarily affects the number of active shoot meristems (Box 1 assumption 2') that the number of active shoot meristems is limiting fitness at high nutrient levels (Box 1, assumption 3'), and that AMD ameliorates shoot meristem limitation (Box 1, assumption 4'). To test whether AMD primarily affects the number of active shoot meristems (Box 1 assumption 2'), we would examine the reaction norm across AMD treatments (no AMD or AMD) for basal inflorescence number; if the number of basal inflorescences (which are produced by activated shoot meristems) changed depending on AMD, then this assumption would be satisfied. To test if the number of active shoot meristems is limiting fitness at high nutrient levels (Box 1 assumption 3'), we would regress the number of basal inflorescences on fitness (as estimated by total viable seed production); if the slope of the regression were positive, then this assumption would be correct. To test whether AMD ameliorates shoot meristem limitation (Box 1, assumption 4'), we would further examine the reaction norm across AMD treatments for basal inflorescence number; if the number of basal inflorescences increased with AMD, then this assumption would be deemed valid. Only if all of these assumptions were correct would we conclude that LRM-IV is an appropriate candidate model for explaining the data; otherwise, we would conclude that the data did not fit any of the LRM models.

Results

Phenotypic variation

Nutrient levels, apical meristem damage (AMD) treatment, light rack, transplant status and the region of origin by nutrient by AMD interaction all had influence on at least some of the traits, when controlling for the number of simultaneous tests performed (Moran's *p*, Table 2). Analysis of covariance models including rosette size did not differ from analysis of variance (ANOVA) models without it, in terms of model R^2, other significant model factors and interactions. Therefore, we only present the results from the ANOVAs.

All ANOVAs were highly statistically significant, and explained between 33% (for basal inflorescence number) and 54% (for fruit ripening time) of the total phenotypic

Table 2. Analyses of variance for all traits. For each factor (rows) we report the mean squares and the p-value associated with the F-ratio test. Instead of a multiple-test correction, we report for each factor (row) Moran's p, the probability of finding by chance the observed number of p-values below $\alpha = 0.05$ given the number of tests performed (Moran 2003).

R^2	Basal inflorescence number	Lateral branch number	Fruit ripening time	Fruit production	Estimated total viable seed production	*p*
	0.33	0.39	54.00	0.52	0.44	
Region; DF = 2	0.095 (0.446)	0.072 (0.915)	0.50 (0.059)	223.05 (0.065)	20.35 (0.172)	0.773
Population [region]; DF = 6	0.11 (0.630)	0.83 (0.426)	0.16 (0.040)	55.13 (0.102)	9.31 (0.144)	0.203
Family [population, region]; DF = 15	0.10 (0.362)	0.63 (0.110)	0.025 (0.0003)	6.68 (0.731)	2.10 (0.635)	0.204
Nutrient levels (NTL); DF = 1	**5.06 (0.00019)**	**32.15 (<0.0001)**	**0.20 (0.012)**	**948.29 (<0.0001)**	**46.80 (0.004)**	**<0.0001**
Apical meristem damage (AMD); DF = 1	**1.53 (0.0019)**	0.051 (0.624)	**1.97 (<0.0001)**	**45.86 (0.011)**	**9.60 (0.032)**	**<0.0001**
NTL × AMD; DF = 1	0.22 (0.074)	0.79 (0.106)	0.032 (0.106)	16.48 (0.050)	0.018 (0.881)	0.204
Region × NTL; DF = 2	0.085 (0.443)	0.23 (0.568)	0.065 (0.106)	25.85 (0.205)	0.33 (0.886)	0.774
Region × AMD; DF = 2	0.17 (0.157)	0.41 (0.184)	0.089 (0.031)	0.108 (0.976)	0.44 (0.737)	0.204
Region × NTL × AMD; DF = 2	0.016 (0.736)	0.10 (0.669)	**0.054 (0.026)**	**23.51 (0.013)**	**4.06 (0.036)**	**0.001**
Population × NTL; DF = 6	0.096 (0.287)	0.39 (0.301)	0.028 (0.149)	13.43 (0.191)	2.84 (0.261)	0.774
Population × AMD; DF = 6	0.071 (0.499)	0.19 (0.648)	0.021 (0.241)	4.13 (0.560)	1.41 (0.418)	0.774
Population × NTL × AMD; DF = 6	0.048 (0.532)	0.24 (0.357)	0.012 (0.365)	2.88 (0.825)	0.76 (0.436)	0.774
Family × NTL; DF = 15	0.054 (0.496)	0.13 (0.765)	–	7.82 (0.309)	1.99 (0.029)	0.171
Family × AMD; DF = 15	0.078 (0.234)	0.22 (0.394)	–	7.61 (0.327)	1.25 (0.149)	0.815
Family × NTL × AMD; DF = 15	0.054 (0.710)	0.20 (0.648)	–	6.01 (0.647)	0.72 (0.668)	0.815
Light rack; DF = 1	**0.63 (0.0028)**	**2.19 (0.0024)**	0.000049 (0.830)	**484.42 (<0.0001)**	**11.67 (0.0003)**	**<0.0001**
Transplant status; DF = 2	0.013 (0.834)	0.22 (0.403)	0.0073 (0.934)	**21.88 (0.050)**	**3.54 (0.019)**	**0.021**
Residual	0.069; DF = 524	0.24; DF = 526	0.011; DF = 408	7.27; DF = 526	0.89; DF = 509	

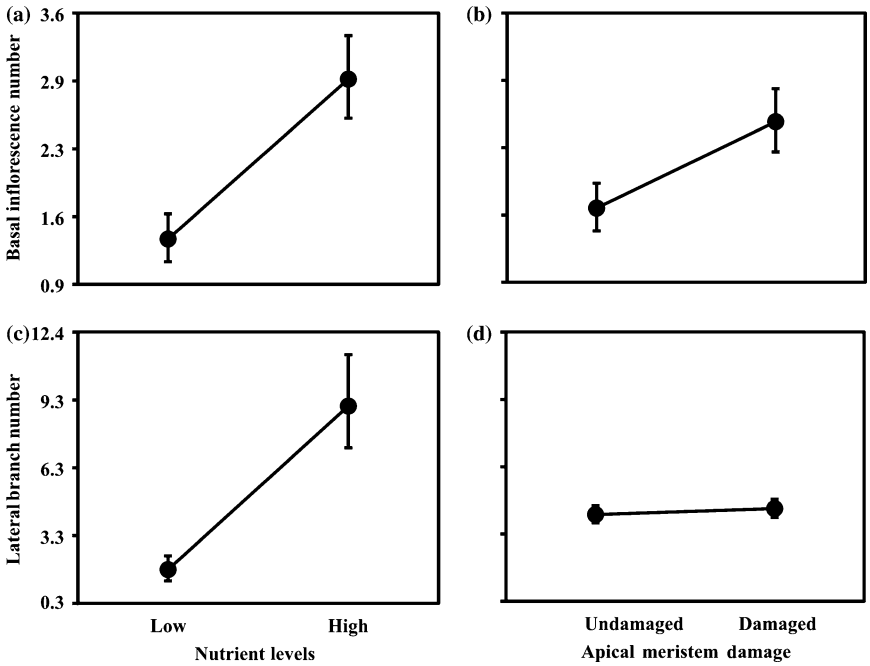

Figure 1. Least squares mean values of basal inflorescence number (top) and lateral branch number (bottom) according to the nutrient levels (left) or apical meristem damage (right) treatment. The error bars are the 95% confidence intervals.

variance in our samples. Effects of light rack and transplant status were significant in several models (Table 2).

Nutrient addition increased the number of basal inflorescences and lateral (i.e. higher order) branches (Fig. 1). Removal of the main inflorescence (AMD) also increased the number of basal inflorescences, although it had no effect on the number of lateral branches (Fig. 1). There was no significant variation among plants from different regions, populations, maternal seed families, or nutrient level treatments in the effect of AMD on basal inflorescence number or lateral branch number (Table 2).

AMD delayed fruit ripening in all plants (Fig. 2), but especially the Dutch plants grown under low nutrients (region by nutrients by AMD $p < 0.05$, Table 2, Fig. 2). Nutrients boosted fruit production in all plants, while AMD reduced fruit production in most (Fig. 2, Table 2). The only exception was that added nutrients seemed to allow for greater fruit production in the damaged than in the undamaged Dutch plants (region by nutrients by AMD $p < 0.05$, Table 2, Fig. 2). Nutrients appeared to enhance total viable seed production, but any positive effect was highly dependent on the region of origin of the plants and on whether AMD was imposed (region by nutrients by AMD $p < 0.05$, Table 2, Fig. 2).

Comparison of the LRM models

After filtering out candidate models characterized by inaccurate assumptions, we found that: (1) Spanish plants appeared to behave consistently with LRM-II when fruit production was used to estimate fitness, and appeared to behave according to LRM-V when the measure more closely linked to fitness (estimated total viable seed production) was used instead. (2) Dutch plants did not behave in accordance with any of the LRM family when fruit production was considered, but appeared to behave according to LRM-V when estimated total viable seed production was used instead. (3) Swedish plants appeared to behave according to LRM-II when fruit production was used, but did not behave in accordance with any of the models when estimated total viable seed production was used (Table 3).

Discussion

General patterns

We found that apical meristem damage (AMD) delayed the time to fruit ripening substantially, and Dutch plants at low nutrient levels were more affected than the others. Considering that *A. thaliana* is an opportunistic ruderal species (Napp-Zinn 1985), and that changes in its timing of germination can have dramatic effects on fitness in the field (Donohue 2002), AMD-induced delay to mature seed set could translate into reduced fitness in natural situations. Delayed flowering and fruiting as a cost of tolerance to damage has been recognized in other studies (Bergelson and Crawley 1992b, Lennartsson et al. 1998, Huhta et al. 2000b, Juenger and Bergelson 2000, Hanley and Fegan 2007; but see Paige and Whitham 1987).

This study demonstrates for the first time the existence of genetic variation in tolerance in natural populations of *A. thaliana*, although it had been demonstrated previously in recombinant inbred lines (Weinig et al. 2003). This highlights the ecological relevance of studying tolerance to AMD using *A. thaliana*. The genetic variation in

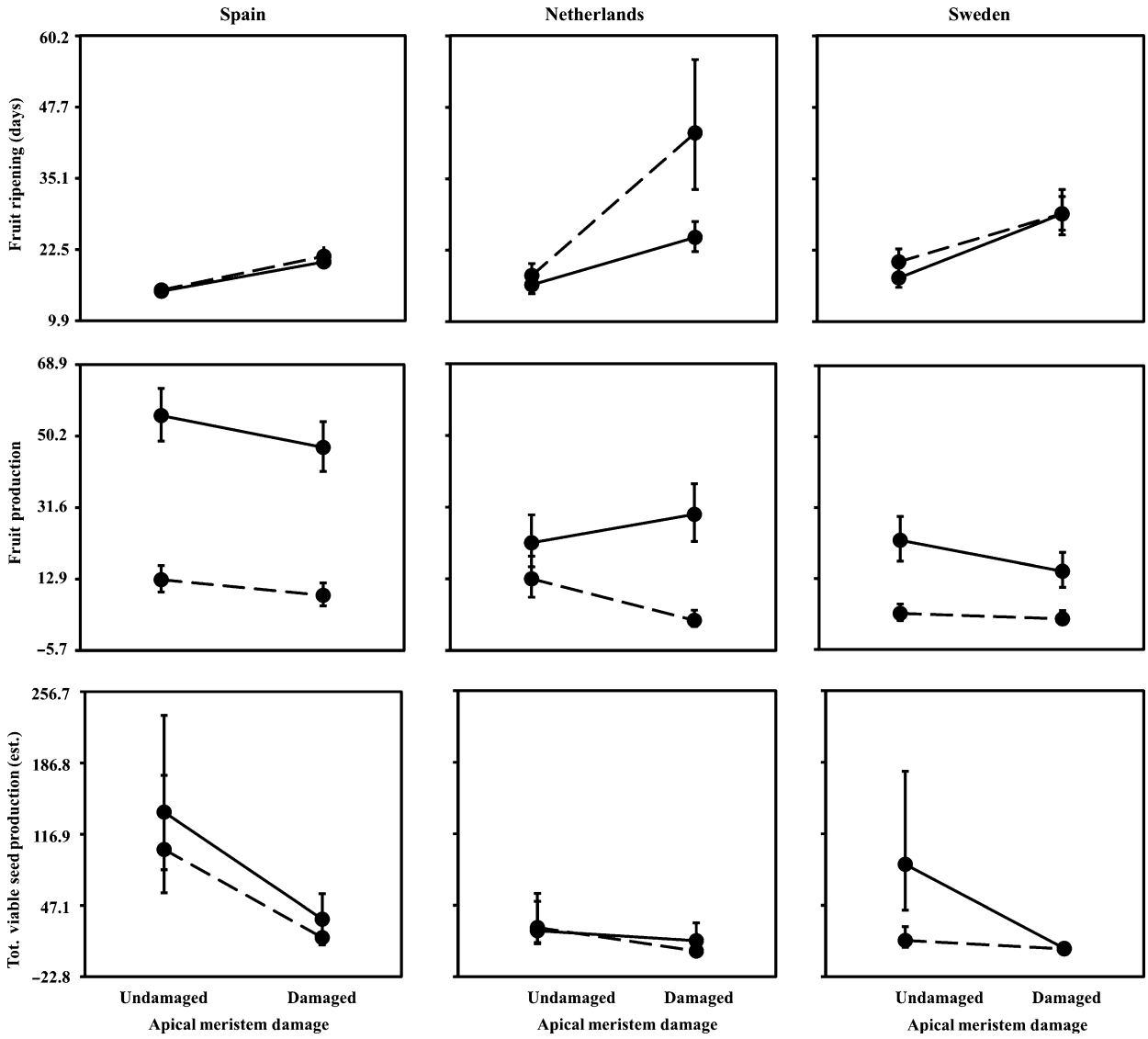

Figure 2. Least squares mean values of fruit ripening time (top row) fruit production (middle row), and estimated total viable seed production (bottom row) for plants from northern Spain (left column), the Netherlands (middle column), and southern Sweden (right column). Solid lines are high nutrient levels and dotted lines are low nutrient levels. The error bars are the 95% confidence intervals, although they are sometimes too small to be visible.

tolerance we observed was only detected among plants from different regions, and not among plants from different populations within regions or among different maternal seed families within the same population. The reason for this spatially coarse-grained differentiation in tolerance remains unaddressed and is worthy of further inverstigation. Even though Banta et al. (2007) found that neutral molecular differentiation is not associated with either overall phenotypic differentiation or geographic distance in *A. thaliana*, differentiation in specific traits may be due to drift, and specific alternative selective hypotheses need to be tested based on ecological data.

Our results show that it is not always safe to assume that fruit production will yield the same results as estimates more directly linked to fitness, contrary to Westerman and Lawrence (1970) and to common practice in *Arabidopsis* research. We found that 'tolerance' sometimes changed appreciably depending on whether it was estimated using total viable seed production or fruit production. In fact, basing our conclusions on fruit production would have altered which models were supported. In particular, an apparent instance of overcompensation, Dutch plants at high nutrients, disappeared when estimated total viable seed production was used. An important caveat is that our growth conditions may have caused the incongruity between the two fitness estimates; we grew the plants in a sand mixture under high-intensity artificial lighting, which may have stressed the plants and perturbed the correlation between these traits. We suggest, though, that our growing conditions were ecologically realistic, since *A. thaliana* is often observed to grow in sandy, stressful conditions (J. Banta unpubl.). We believe our study illustrates the need to estimate fitness using traits that are as closely linked to fitness as possible (Hanley and Fegan 2007). We also believe, however, that further study of the correlation between fruit number and estimated viable seed production,

Table 3. Comparisons of the results to the various models within the LRM framework, broken down by region of origin of the plants (first column) and the fitness proxy being considered (second column). The observed outcomes are presented (third column), as well as the models that predicted those outcomes (fourth column). These candidate models are then filtered (fifth column, grey) by examining the applicability of their underlying assumptions to finally find the models that are consistent with the data both in their predictions and assumptions (sixth column, bold).

Region	Fitness proxy	Results	Predicted by	Incorrect assumptions	Consistent with
Spain	Fruits	The same level of tolerance at both nutrient levels	II, V, VII	V, VII	**II**
	Total viable seeds	The same level of tolerance at both nutrient levels	II, V, VII	II, VII	**V**
Netherlands	Fruits	Greater tolerance at higher nutrient levels	IV, VI	IV, VI	**None**
	Total viable seeds	The same level of tolerance at both nutrient levels	II, V, VII	II, VII	**V**
Sweden	Fruits	The same level of tolerance at both nutrient levels	II, V, VII	V, VII	**II**
	Total viable seeds	Lower tolerance at higher nutrient levels	III	III	**None**

directly in the natural environment, is necessary before any firm conclusions are formed as to how well (or poorly) fruit production estimates fitness.

Although AMD proliferated basal inflorescences as expected, this did not translate into an increase in fitness with damage (except for Dutch plants, when estimating fitness using fruit production). This is because damaged plants did not proliferate lateral branches (i.e. secondary and higher-order branches off of the basal inflorescences) where most of the fruits develop. An increase in basal inflorescences in damaged plants, without a concomitant increase in lateral branches, was also observed in Banta and Pigliucci (2005) for different *A. thaliana* accessions. It seems that, in *A. thaliana*, AMD causes a proliferation of the modules on which fruits and seeds are grown (i.e. basal inflorescences), but these modules contain less surface area for fruits and seeds (i.e. fewer lateral branches) as compared to undamaged plants.

Comparisons to model expectations

We suggest that the limiting resources model (LRM) of tolerance is more appropriately treated as a broad conceptual framework rather than as a single model, due to the flexibility of its assumptions. Therefore, we believe that Wise and Abrahamson's studies (2007, 2008), which surveyed the tolerance literature and tested all available datasets against 'the' LRM, were actually testing the datasets against a group of models making different assumptions, what we call the LRM framework. This is important, because Wise and Abrahamson (2007, 2008) found that 'the' LRM fits empirical data better than either the compensatory continuum hypothesis or the growth rate model. This is not surprising, however, when one views the LRM as a plurality of models, wherein the compensatory continuum hypothesis is actually one of the LRM framework's nested models (LRM-IV; Box 1). Rather than supporting any one model, we believe their findings show that no one model of tolerance consistently fit their data appreciably more than any other (although the entire group of LRM models in aggregate proved to be good at explaining many of the patterns).

Considering that the LRM was developed to improve prediction in tolerance studies (Wise and Abrahamson 2008), we believe it is important that the LRM framework be recognized as a plurality of models. The LRM framework encompasses every logically possible outcome – i.e. greater tolerance with increasing nutrient levels (the models we have named LRM-1, LRM-IV and LRM-VI), decreased tolerance with increasing nutrient levels (LRM-III), and the same level of tolerance across all nutrient levels (LRM-II, LRM-V and LRM-VII; Box 1) – and so finding that 'the' LRM fit the data very well does not, by itself, inform future tolerance studies or improve their predictive abilities. Only by breaking the LRM down into its constituent models does it become clear which particular models (and corollary assumptions) were supported in a given study, thereby informing future work.

When the LRM is decomposed into separate models, it is apparent that several of them predict the same outcome. Therefore, in order to judge a particular empirical result as being consistent with a model from the LRM family, or with any model for that matter, we believe two considerations are paramount. First, one must ask: were the model's assumptions, not just its predicted outcome, congruent with the observed results? This is crucial, because multiple models can predict the same outcome, making it impossible to distinguish among them without testing their assumptions as well, and because even models that make unique predictions can predict the right outcome for the wrong reasons. The second question one must ask is: are the individual assumptions of the model tested independently, i.e. using measurements that are independent from the results? Specifically, in the case of the LRM models, 'alternate resource limitation' and 'tolerance' should be estimated from separate variables. In Wise and Abrahamson (2007), however, they were both defined as the slope of the fitness–proxy reaction norm (across damage treatments). This inflates the apparent agreement between data and model; although one may find that empirical data are consistent with a particular model, and that the assumptions of the model are valid, this is logically (as opposed to biologically) inevitable, since assumptions and predictions amount to the same thing, and the approach therefore does not yield a fair evaluation of model-data congruity. For instance, LRM-IV (Box 1) predicts greater tolerance to AMD at high nutrient levels, and assumes as one of its predicates that AMD alleviates shoot meristem limitation. When a positive slope of the fitness reaction norm implies that the assumption that AMD alleviated shoot meristem limitation is correct, and also simultaneously predicts increased tolerance, the assumption and prediction are confounded, and the mechanism in the model becomes unfalsifiable.

After evaluating the assumptions of the LRM models against our data, and filtering out models that predicted the right outcomes for the wrong reasons, we were left with a

situation where different models fit the data in different circumstances; no one model consistently fit our results across the wide area of origin of our samples. This is in line with studies by Marshall and Avila-Sakar (2008) and Suwa and Maherali (2008), who also noted that any one set of assumptions made using the LRM framework do not seem to hold species-wide. When 'the' LRM is treated as unitary, it might be considered to fit species-wide; however, when the LRM is broken down into separate entities, it becomes clear that no one model accounts for all instances of tolerance, even within one species.

Our heterogeneous results indicate that the relationship between tolerance and nutrient levels is complex and not readily predictable. In other words, a researcher will probably not know a priori which model, from the LRM framework or elsewhere, should apply to their system, and thereby what outcome to expect, without detailed prior information about the idiosyncrasies of the particular genetic stocks. We could not have anticipated, for example, which models to test against our data unless we knew which populations were nutrient limited and under what circumstances, and whether active shoot meristems were limiting for those populations and under what circumstances, facts that changed depending on the population being considered. This sort of problem is common in biology, due to the contingent evolutionary histories of living systems (Pigliucci 2002b), and illustrates that there is probably no one set of assumptions able to account for the tolerance of all plants, even for different populations of a single species (discussed by Hawkes and Sullivan 2001). We suggest that efforts at unifying all instances of tolerance under a single explanatory model will not be fruitful and that tolerance is best studied on a more local (genetically and geographically) level, where a given set of assumptions are more likely to be homogeneously valid and where particular models (such as individual ones from the LRM framework, for instance) can be applied after careful consideration and prior study.

It is interesting to note that, one third of the time, the data was not consistent with any LRM models, whether using fruit production or estimated total viable seed production. Furthermore, none of our results were consistent with LRM-IV (also known as the compensatory continuum hypothesis) despite the fact that it would seem, on its face, to be the most applicable to plants with *A. thaliana*'s architecture (Maschinski and Whitham 1989, Wise and Abrahamson 2005, 2007, 2008). It therefore seems that more models to account for the relationship between nutrient levels and tolerance are needed. The only model we did not test, that we are aware of, is the growth rate model (Hilbert et al. 1981). This model may explain the patterns observed in the Swedish plants (as measured by estimated total viable seed production). To evaluate the growth rate model, one would need to test its central assumption that plants are growing at their biologically maximal rate at high nutrient levels. This would entail growing the plants across a broad range of nutrient levels, with periodic measurements, to test whether the growth rate levels off at high nutrients.

We found that, in contrast to Westerman and Lawrence (1970), our results were highly sensitive to the choice of fitness estimate. It may be important in evolutionary ecological studies, especially those measuring primarily fitness and fitness plasticity (as in this case), to use traits that are linked to fitness as closely as possible, such as those that are based on several fitness components (like estimated total viable seed production). When this sort of estimate is not possible and phenotypic measurements farther removed from fitness must be used, either because of the mating system or because of logistical considerations, it is at least important to recognize the problem and the uncertainty it creates.

Concluding remarks

Our results show that tolerance to apical meristem damage, and the effect of nutrient levels on it, are not uniform across populatins in *Arabidopsis thaliana*, and that no one model is able to account for this non-uniformity. The basic problem is that the assumptions of any one model (at least from the LRM framework) do not apply species-wide. To test specific models of tolerance appears to require extensive ecological information about the individual populations of study to ensure that the models, and their underlying assumptions, are appropriate for those populations. Sometimes, the addage that 'all politics is local' might well apply to ecology.

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