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Tradeoff between resistance and tolerance to herbivore damage in a morning glory

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MANY plant characters, including toxic secondary compounds, trichomes, spines and tough, nutrient-poor leaves have evolved at least in part as defences against pathogens and herbivores, including phytophagous insects^{1–6}. Models of the evolution of resistance^{7,9} predict that allocation to defence is determined by a tradeoff between the benefits of resistance, such as reduction in herbivore damage, and costs of resistance, generally envisaged as reduction in fitness in an environment in which herbivores are absent⁹. However, despite attempts to determine the costs of resistance, there is little convincing evidence that they exist and constrain the evolution of defences^{10,11}. Here we report the existence of such a cost in the tall morning glory, *Ipomoea purpurea*: genotypes that exhibit relatively high levels of resistance to insects that cause damage to apical meristems exhibit relatively low tolerance to this form of damage. We also show how this type of tradeoff constrains the evolution of resistance.

In a field experiment, ten inbred lines exhibited heterogeneity in damage caused by generalist herbivores, flea hoppers and insects causing damage to the apical meristem (Fig. 1). There

thus appears to be considerable genetic variation for resistance to each of these herbivores in the population from which the lines were established. Moreover, line-mean correlations between resistance to apical meristem damage and resistance to each of the other herbivores are low and non-significant, indicating that resistance to apical meristem damage involves different mechanisms, and thus is a different character, from resistance to the other herbivores (correlation coefficients $r=0.22$ and $r=-0.27$ for correlations with resistance to generalist insects and to flea hoppers, respectively). These results are consistent with previous investigations of *Ipomoea purpurea*^{9,12}, in which genetic variation was detected for resistance to generalist herbivores and other types of insects.

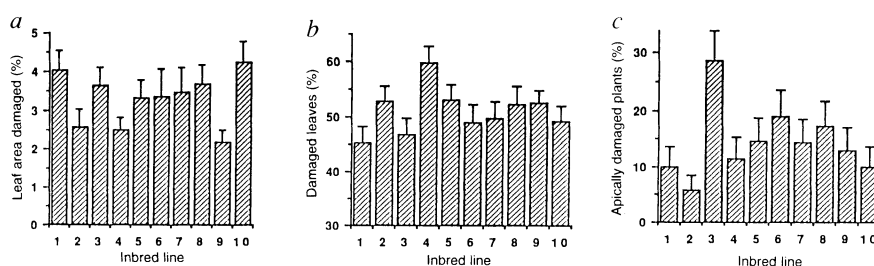
In a second experiment, we measured the tolerance to apical damage of 12 inbred lines. Tolerance differs from resistance in that it is the amount by which fitness is reduced for a given amount of damage, whereas resistance is the amount of damage a plant experiences for a given abundance of herbivores¹³. We measured tolerance to simulated herbivore feeding in a greenhouse, thus eliminating natural herbivore completely. Moreover, we investigated damage to the apical meristem because it is easy to mimic experimentally.

A multivariate analysis of variance (MANOVA) showed that lines varied overall in the five fitness components examined (main effect of line, $F=7.7$, d.f. = 55, 78, $P<0.0001$), and that damage treatment caused a significant reduction in these fitness components (main effect of damage treatment $F=10.2$, d.f. = 10, 32, $P<0.0001$). Most importantly, however, a significant treatment by line interaction ($F=1.5$, d.f. = 100, 375, $P<0.0025$) showed that inbred lines varied in their overall fitness response to the damage treatment, indicating that lines varied in their degree of tolerance to apical damage.

Univariate analyses (Table 1) of individual fitness components revealed that the MANOVA results originated largely from vari-

FIG. 1 Variation among inbred lines in percentage damage by damage type. a, Generalist insect herbivores: several grasshopper and lepidopteran species ($F_{9,538}=1.99$, $P<0.05$). b, Flea hoppers ($F_{9,538}=1.96$, $P<0.01$). c, Insects damaging apical meristem ($G=19.2$, d.f. = 9, $P<0.05$). For a and b, probabilities indicate significance of line effect in analysis of variance. For c, probability reflects significance of inbred line effect in a G-test. The effect of female parent nested within line was non-significant for generalist insects and for flea hoppers ($F_{10,38}=0.56$ and 0.99, respectively), indicating minimal maternal effects on resistance. The effect of female parent could not be tested for damage to apical meristem.

METHODS. Ten inbred lines were selected randomly from a set of 35 lines inbred by selfing for 7 generations, each generation being propagated by a single selfed seed. The progenitors of all lines were collected from the same population in Durham County, NC. To produce the experimental seed, two selfed offspring seed from a single seventh-generation plant were chosen as 'female parents' from each line. Each of these female parents was allowed to produce 35 experimental seeds by selfing. These experimental seeds were germinated in the greenhouse and then transplanted to a tilled field when they had produced



cotyledons. Seedlings were planted in the field in a randomized block design. Four weeks later, plants were censused for damage by several types of herbivores: damage caused by generalist herbivores was estimated as the number of 0.694 cm² transparent grid squares (to the nearest quarter square) covering the area of damage on each leaf and expressed as the proportion of total leaf area for each plant. Flea hopper damage was estimated as the presence or absence of damage on each leaf and expressed as the proportion of leaves affected. Plants were also scored for the presence or absence of damage to the apical meristem. (See ref. 25 for further details.)

TABLE 1 Univariate analysis of variance for plant performance characters

Source	df	Growth rate		First flower		First Capsule		Flower number		Capsule number	
		SS	F	SS	F	SS	F	SS	F	SS	F
Block	4	863.4	16.7***	138.3	5.4***	88.3	2.86*	74.0	1.47	1,827.6	10.2***
Line	11	924.0	6.5***	1,268.5	18.2***	1,300.3	15.3***	1,934.9	8.41***	16,425.0	18.6***
Damage	2	218.4	8.5***	371.2	29.2***	300.6	19.5***	34.5	0.83	491.6	3.1†
Block × Line	44	368.1	0.7	256.5	0.9	398.8	1.2	427.5	0.77	2,335.6	1.2
Block × Damage	8	103.0	1.0	34.6	0.7	51.8	0.8	60.9	0.61	109.9	0.3
Line × Damage	20	318.5	1.2	149.8	1.2	148.6	1.0	418.2	1.67†	1,609.2	1.8*
Error	80	1,032.3		508.0		616.7		1,004.0		3,588.1	

The other terms in the MANOVA for the 5 performance variables were: block, d.f. = 20, 253, F -ratio = 5.5, $P < 0.001$; block × line, d.f. = 220, 383, $F = 1.1$, P non-significant; and block × treatment, d.f. = 40, 334, F -ratio = 0.8, P non-significant. For all three terms, the model error was used as the error term. For the univariate analyses reported here, F -ratios were calculated using the error mean square in all cases except flower and capsule number, in which the line × damage sum of squares (ss) was used as a denominator for the main effects of line and damage. Type III sums of squares were used. The tolerance experiment used 12 inbred lines, including 6 that were also used in the field experiment. Seedlings were allocated to three treatments: (1) early damage at the seedling stage; (2) late damage at the rapid growth stage; (3) control. Three seeds from each of five females for each inbred line were grown individually in 4-inch pots and placed on greenhouse benches in a randomized block design, with three plants of each line per block. One of the plants from each line in each block was assigned to each of the treatments. Early-damage plants had the extreme tip removed at the seedling state (17 days after planting), late-damage plants had the growing tip removed during the rapid growth phase (4 weeks after planting), and control plants were not damaged. Plants were allowed to grow, flower and set seed, and the following fitness components were assessed: growth rate, date of first flowering, date of first capsule maturation, total flower production and total capsule production. Growth rate was estimated as the difference in estimated leaf area measured at 4 weeks and at 17 days, divided by that measured at 17 days. Total flower production was estimated from 11 counts performed over a six-week period. Individual flowers last for one day. Tolerance was estimated for each inbred line as the ratio of the mean number of capsules produced by plants damaged at four weeks to the mean number produced by the undamaged control plants. This ratio was based on the late-damage treatment because it best corresponded to the timing of damage measurement in the field experiment. (See ref. 25 for further details.) † $P < 0.1$, * $P < 0.05$, *** $P < 0.001$.

ation among lines in the effect of apical damage on flower and seed capsule production. Because seed production is highly correlated with capsule production ($r = 0.98$) (ref. 14), these results indicate that lines vary in tolerance, at least as measured in terms of the female component of total fitness. There was no detectable difference among inbred lines in the effect of damage on plant growth rate, or on the timing of initial flowering or capsule production.

Because six inbred lines were common to both the field and greenhouse experiments, we could determine whether resistance to apical damage and tolerance of apical damage are related. Line means for these two traits are strongly negatively correlated (Fig. 2; $r_s = -0.943$, $P < 0.02$), indicating a strong negative genetic correlation between these two traits. Such a correlation is the embodiment of a tradeoff^{15,17}, as it indicates that selection for increased resistance should lead to decreased tolerance. By using an analysis based on inbred lines we were not able to separate the additive and non-additive components of covariance between resistance and tolerance, but previous investigations of several types of resistance in *I. purpurea*^{9,12} have indicated that most variation for resistance is additive, suggesting that the covariance observed involves primarily additive variance for resistance to meristem damage. Because additive and non-additive variance are uncorrelated¹⁸, this suggests that the measured covariance is primarily due to additive genetic effects.

Several theoretical treatments have examined the effects of tradeoffs between the benefits (reduced herbivore damage) and costs (reduced fitness in the absence of herbivores) of resistance on the evolution of resistance^{7,9}. In general these models, which assume that fitness costs of resistance are either 'self-toxicity', 'allocation' or 'ecological' costs¹¹, yield three important predictions for plausible cost and benefit functions: (1) there is a single peak in the adaptive landscape, so either net selection on resistance is directional (meaning that either no resistance or complete resistance is favoured) or it is stabilizing (meaning that an intermediate level of resistance is favoured); (2) an increase in herbivore abundance causes an evolutionary increase in the mean level of resistance; and (3) reduction in the efficacy of defence by herbivore counteradaptation causes an evolutionary change in the level of resistance. Tradeoffs between resistance and tolerance, however, may have different implications for the evolution of resistance, as can be seen from the following model.

Previous models of the evolution of resistance infer the evolutionary equilibrium for allocation to resistance from the position of the peak(s) in an adaptive landscape, in which plant fitness, W , is represented by the relationship

$$W(R) = W_0 - C(R) - D(R)T, \quad (1)$$

where W_0 is the fitness of a plant with no resistance in the absence of herbivores, R is the proportion of the maximum possible allocation of resources to resistance, $C(R)$ is the cost of resistance, $D(R)$ is amount of damage experienced by a plant with allocation R , and T is the reduction in fitness per unit damage. T is essentially a measure of tolerance, which is assumed to be the same for all values of R .

To incorporate the observed inverse relationship between resistance and tolerance in *I. purpurea*, this equation can be modified by allowing T to be a function of R . If, as suggested by Fig. 2, tolerance is inversely related to resistance, this may be represented by

$$T(R) = K_1 + cR. \quad (2)$$

Large values of $T(R)$ indicate low tolerance (damage causes a

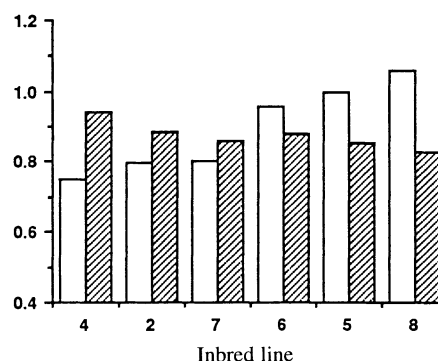


FIG. 2 Line means for resistance to and tolerance of apical meristem damage for the six inbred lines common to field and greenhouse. The y axis is proportion damage (for resistance bars shaded) or proportional reduction in fitness (for tolerance bars white). Line numbers correspond to lines portrayed in Fig. 1. Spearman's rank correlation, $r_s = -0.943$, $P < 0.02$.

large reduction in fitness), and small values indicate high tolerance (damage causes little reduction in fitness).

As in the previous models, as allocation to resistance increases, the amount of damage experienced by a plant must asymptote towards zero⁹, which can be represented by

$$D(R) = D_0 e^{-bR}, \quad (3)$$

where D_0 is the amount of damage to plants with no resistance ($R=0$) and b is a constant representing the efficacy of resistance at reducing damage. Combining equations (1)–(3) yields

$$W(R) = W_0 - C(R) - (K_1 + cR)D_0 e^{-bR}. \quad (4)$$

Analysis of this model indicates that a cost of resistance in the form of a tradeoff between resistance and tolerance may constrain the evolution of resistance differently from standard allocation or ecological costs in at least three ways.

(1) There may be two peaks in the adaptive landscape, corresponding to maximal resistance and no tolerance, and to no resistance and maximal tolerance (Fig. 3a). For a given abundance of herbivores, increased resistance is favoured only if mean allocation to resistance is initially greater than a threshold corresponding to the minimum in Fig. 3a; otherwise, decreased resistance, accompanied by increased tolerance, will be favoured. Different levels of resistance and tolerance may thus evolve in different populations, even though the environmental conditions are similar.

(2) When two such peaks exist, an increase in herbivore abundance is generally not expected to cause an evolutionary increase in resistance. This expectation arises because, unless the increase in herbivore abundance is very large, there will be no change in the position of the peaks. The adaptive landscape remains concave upwards, and only the position of the fitness minimum may change (Fig. 3a). In the previous models, although the cost of resistance is fixed, when herbivore abundance increases the benefit of resistance also increases because more herbivores are deterred. The benefit thus increases relative to the cost, and it is profitable to increase allocation to resistance. By contrast, in equation (4) the benefits of tolerance also increase with herbivore abundance. The 'cost' of resistance, in this case absence of tolerance, also increases, tending to maintain both maximal tolerance and maximal resistance as alternative evolutionary equilibria.

(3) Counteradaptation to plant defenses by a herbivore, which in effect decreases the efficacy of resistance, b , tends not to influence allocation to resistance (Fig. 3b). Changing b often alters only the fitness minimum, but does not affect the alternative equilibria, as it does in the previous models.

These considerations indicate that the type of tradeoff we have identified is likely to constrain the evolution of resistance in ways very different from those previously envisaged. Although we have considered in detail here a model in which the relationship between resistance and tolerance is linear, similar results are obtainable from models in which this relationship is not linear (for example, quadratic). In particular, analysis of such models indicates that results (1)–(3) above can easily occur (see Fig. 3a legend for criteria for multiple peaks; a more detailed analysis will be presented elsewhere). We emphasize, however, that in both the linear and nonlinear cases, some parameter combinations yield results similar to those of the previous models (for example, when the slope of the regression of tolerance on resistance, c_1 , is low, or when ecological/allocation costs of resistance, K_2 , are high). The general evolutionary implication of a tradeoff between resistance and tolerance is that modifying the previous models by simply incorporating a tradeoff of the type reported here yields a more diverse array of possible evolutionary outcomes.

The observation that most inbred lines tested exhibited intermediate levels of resistance to apical damage (Fig. 1) may appear to contradict the prediction of our model that resistance should evolve to be minimal or maximal. However, the values of the

model's parameters are currently unknown for *I. purpurea*, and they may lie in the region of parameter space that corresponds to stabilizing selection. Alternatively, there may be additional constraints acting on the evolution of resistance in *I. purpurea* that are not accounted for in our model.

Although other constraints may be operating, it is clear that the evolution of resistance to insects attacking the apical meristems of *I. purpurea* cannot be completely understood without considering the simultaneous evolution of tolerance to these insects. Although tolerance is often assessed in forage and agricultural plants^{19–23}, evolutionary biologists have seldom consid-

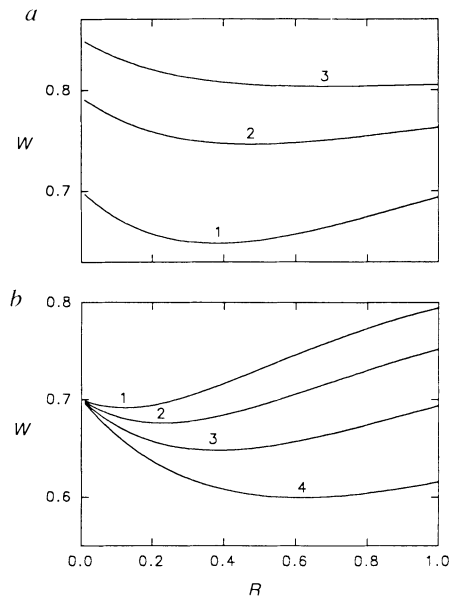


FIG. 3 Examples of two-peaked fitness landscapes for resistance. Lines are plots of relative fitness, $W(R)$, versus proportional allocation to resistance, R , using equation (4). Fitness values are all made relative to a fitness of 1 for genotypes with no resistance ($R=0$) in the absence of herbivores (that is, to $W_0=1$). a, Changing herbivore abundance, as reflected in the parameter D_0 , alters the position of the fitness minimum but does not alter the position of the fitness peaks (equilibria). In this example, for all plots, $K_2=0.083$, $c=0.7$, and $b=1.5$. Lines labelled 1, 2 and 3 correspond to D_0 values of 1.0, 0.69 and 0.5, respectively. Labels are positioned along the x axis at the minimum of the curve. In general, the variables in equation (4) may be rescaled so that $T(R=1)=1$. Then $K_1=1-c$. Let the cost, C , of resistance be linearly related to resistance, so that $C(R)=K_2R$. Then equation (4) becomes $W(R) = W_0 - K_2R - ((1-c) + cR)D_0 e^{-bR}$. The conditions for two peaks are $(dW/dR)|_{R=0} < 0$ and $(dW/dR)|_{R=1} > 0$, which is equivalent to the condition $(b-\alpha)/(1+b) < c < b - \alpha e^b$, where $\alpha = K_2/D_0$. This condition can be satisfied as long as $(b-\alpha)/(1+b) < b - \alpha e^b$, which is possible as long as α is sufficiently small. For example, for b values of 0.5, 1.0 and 2.0, this condition will be satisfied as long as α is less than 0.17, 0.225 and 0.188, respectively. Thus, in general, the existence two peaks is possible as long as the cost of resistance is small compared to the amount of damage incurred by plants with no resistance. By contrast, when there is no cost of resistance (that is, $K_2=0$), $d^2W/dR^2 = bD_0 e^{-bR}(1+c+cR)$, which is always ≥ 0 . Consequently, stabilizing selection is not possible without a cost of resistance, and will be unlikely when the cost of resistance is small. When $K_2=0$, differentiating equation (4) with respect to R and solving for the equilibrium, R , gives $\hat{R} = (1/b) - (1-c)/c$. Because this relationship does not involve D_0 , changes in herbivore abundance, which affect D_0 , will not change the position of the fitness minimum. When K_2 is small, that is, when physiological costs of resistance are small, this result should hold approximately, as shown in a. A nonlinear relationship between tolerance and resistance can be modelled by allowing equation (2) to be $T(R) = K_1 + c_1R + c_2R^2$. Then the conditions for two fitness peaks are $[(1-c_1-c_2)b] < \alpha < e^{-b}(b-c_1-2c_2)$. b, Changing efficacy of resistance, b , alters the position of the fitness minimum, but does not alter the position of the fitness peaks. In this example, for all plots, $\alpha = 0.083$ and $c = 0.7$. Lines 1, 2, 3 and 4 correspond to $b = 2.1, 1.8, 1.5$ and 1.2 , respectively.

ered it explicitly as a trait that may be involved in plant-herbivore coevolution. Moreover, a tradeoff between resistance and tolerance is not likely to be detected by standard assays used for assessing costs of resistance, which involve growing genotypes differing in resistance in a common environment lacking herbivores and determining whether there is a negative genetic correlation between resistance level and fitness^{9,11,17,24}. However, the cost of resistance engendered by the negative correlation with tolerance arises because plants that are slightly resistant experienced damage, and the negative effect of that damage on fitness is large. Because the cost in this case (reduced tolerance) is expressed only in the presence of herbivory, eliminating herbivores in the standard assay would preclude the detection of that cost. Therefore, previous experiments that have failed to detect allocation costs associated with resistance^{9,11,12,14} cannot rule out the existence of tradeoffs between resistance and tolerance. Thus we do not yet know how common such tradeoffs are in natural populations. □

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Mutual policing and repression of competition in the evolution of cooperative groups

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EVOLUTIONARY theory has not explained how competition among lower level units is suppressed in the formation of higher-level evolutionary units^{1,2}. For example, the key problem of early evolution is how small, individual replicators formed cooperative groups of sufficient complexity to allow accurate copying of the genetic material³. The puzzle is why parasites did not subvert the formation of cells by obtaining benefits from the group without contributing to shared traits that enhance reproduction⁴. These parasites would outcompete other replicators within the cell, disrupting reproductive fairness among subunits and destroying the functional coherence of the group. A similar problem arose at a later evolutionary stage with the orderly mendelian segregation of subunits (chromosomes) within cells, and reproductive fairness continued to be a problem in the evolution of insect⁵ and human societies⁶. Here I present a simple model to show how reproductive fairness evolves among subunits to create functional coherence and higher-level units. Self-restraint, which evolves according to the kin-selection coefficient of relatedness, is not sufficient: mutual policing and enforcement of reproductive fairness are also required for the evolution of increasing social complexity.

Competition within groups can have both benefits and costs for an individual. If resources are limited within the group, the most competitive individuals will gain a disproportionate share of the local benefits. However, competition often reduces the group's overall efficiency in using local resources, thus lowering the average success of the group members. A simple model describing these costs and benefits of group competition⁷ is:

$$w_{ij} = (z_{ij}/z_i) (1 - z_i) \quad (1)$$

where w_{ij} and z_{ij} are the fitness and competitive intensity, respectively, for the j th individual in the i th group, and z_i is the average

competitive intensity for members of the i th group. Each individual gains a share z_{ij}/z_i of the local resources, but higher levels of competition reduce the average group productivity, $1 - z_i$.

The model captures the essential tension between individual and group success. For example, the individuals may be parasites and the local resource may be food obtained from the host. Parasites compete within a host by increasing the rate at which host tissues are exploited and consequently damaged. If z_{ij} is the rate of exploitation by an individual parasite, then greater exploitation leads to greater relative success within the host, z_{ij}/z_i . However, rapid exploitation may damage the host, thus reducing the total food available to the parasites by an amount $1 - z_i$.

The equilibrium for the model in equation (1) can be found by maximizing w_{ij} with respect to variants in z_{ij} (see the legend of Fig. 1 for details). The equilibrium is $z_i^* = 1 - r$, where r is the kin-selection coefficient of relatedness among group members. Self-restraint evolves when relatedness is high, reducing competition among group members and increasing group success. By contrast, low relatedness leads to intense competition and low group productivity. In the parasite example, decreasing relatedness causes greater damage (virulence) to the host⁸.

Self-restraint favoured by kin selection promotes improved efficiency of resource utilization. However, there remains a gap between selectively favoured behaviour and complete functional coherence. The gap occurs because within-group competition is increasingly favoured as r declines. In many cases, the genetic relatedness among subunits is low because of mixing among groups and mutation. In addition, many of the important transitions required cooperation between different kinds of units. For example, cooperative symbiosis among different 'quasispecies' of replicators is believed to be essential for the early evolution of genetic systems and the first protocells³. The orderly patterns of mendelian segregation are spectacularly rigid controls on the relative success of the different kinds of subunits (chromosomes) within the cell. In many social insects the relatedness among workers is low, and the workers can subvert the cooperative coherence of the colony by laying their own eggs rather than raising those produced by the queen. Thus kin selection alone is unlikely to explain transitions to new evolutionary units or more complex cooperative groups.

If competition within the group can be repressed then the success of each group member would be increased. Reduced