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The timing of induced resistance and induced susceptibility in the soybean-Mexican bean beetle system

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Abstract Induced plant responses to herbivory have been demonstrated in many systems. It has been suggested that the timing of these responses may influence the impact of induced resistance on herbivore populations, and may affect the evolution of induced defenses. This study used a bioassay to characterize the time course of systemic induced responses to Mexican bean beetle herbivory in four genotypes of soybeans. The results suggest that the time course of induced responses in this system is more complex than most previous studies have indicated. Herbivory provoked both rapid induced resistance and subsequent induced susceptibility to beetle feeding. All four genotypes of soybean induced significant resistance to beetle damage (beetles preferred undamaged to damaged plants) by 3 days after damage. By 15 days after damage, this resistance had decayed (beetles showed no preference for undamaged over damaged plants), and by 20 days after damage, all four genotypes exhibited significant induced susceptibility (beetles preferred previously damaged plants over undamaged plants). The magnitude of induced resistance in each genotype correlated strongly with the magnitude of induced susceptibility in that genotype.

Key words Induced resistance · Induced susceptibility · Plant-herbivore interactions · *Glycine max* · *Epilachna varivestis*

Introduction

The timing of induced plant resistance to herbivory is known to vary between plant species, from responses that are measurable within hours of damage (Green and Ryan 1972; McCloud et al. 1995) to those that take years (Haukioja 1980). One conceptual model of the time course of induced resistance prevails in the literature. Damage is expected to lead to an increase in resistance to some peak (sometimes after a lag between damage and initial induction), and in the absence of additional damage, induced resistance is expected to decay over some period of time (Fig. 1). This model has been assumed in theoretical explorations of the effect of induced resistance on herbivore population dynamics (Schultz 1988; Edelstein-Keshet and Rausher 1989; Lundberg et al. 1994; Morris and Dwyer 1997) and in verbal and mathematical theory for the evolution of induced defenses (Tuomi et al. 1991; Adler and Karban 1994; Malcolm 1995). Many studies have documented changes in plants induced by herbivore feeding or artificial damage over some period of time (e.g., Baldwin 1988; Croxford et al. 1989; Baldwin et al. 1994; McCloud et al. 1995), and the existing data generally support the model of the timing of induced resistance described above. However, most studies have looked at induced resistance over short periods of time, and many have not followed their systems through the full decay of induced responses (e.g., Edwards et al. 1985; Chiang et al. 1987; Olson and Roseland 1991; Stout et al. 1996). It is therefore not yet clear that we have a complete picture of the time course of induced resistance.

Understanding the details of the time course of induced plant responses to herbivory may be necessary for determining the role of induced resistance in ecological and evolutionary processes. For example, Edelstein-Keshet and Rausher (1989) found in an analytical model that the relative lengths of lag and decay times of induced resistance determined the size of equilibrium herbivore populations and whether regulation of herbivore populations could occur. Other population dynamics models also suggest that long lag times caused by induced resistance or other factors can promote cyclic fluctuations in herbivore populations (Turchin and Taylor 1992; Lundberg et al. 1994). The timing of induced resistance may also interact with the timing of herbivore life histories to determine the effectiveness of

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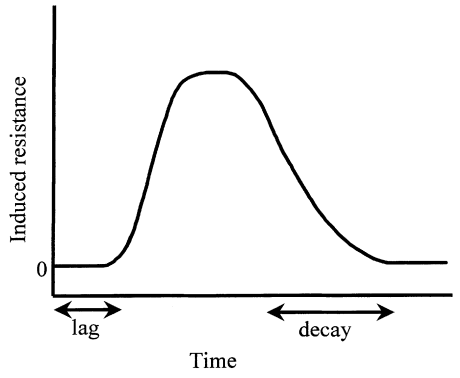

Fig. 1 Conceptual model of the time course of induced resistance assumed in theoretical studies of the effect of induced resistance on herbivore dynamics and the evolution of induced defenses. There may be some time lag between damage (occurring at time 0) and the induced response. In the absence of further damage, the response may then decay over some period of time. The degree of symmetry of the curve may also vary among systems

induced resistance in deterring herbivores, and may thus affect the potential evolution of induced defenses.[1] Knowing the time course of induced resistance in a particular system can also be important for experimental design. For example, if the time to peak induced resistance varies between species or genotypes, knowing the timing of induced resistance would be critical for comparing the magnitude of induced resistance among species or genotypes.

As part of a larger study of the effect of induced resistance in soybeans on the population dynamics of the Mexican bean beetle, I characterized the time course of systemic induced responses resulting from damage by Mexican bean beetles in four genotypes of soybeans. I found that the time course of induced resistance in this system is more complicated than that envisioned by the prevailing conceptual model. Here I report that in addition to the production and subsequent decay of induced resistance, there is a period of enhanced susceptibility to herbivores following the decay of resistance in this system.

Materials and methods

Experimental organisms

I used four genotypes (cultivars) of soybeans (*Glycine max*: Fabaceae) in these experiments: Williams, Bragg, Clark, and Centennial. All four genotypes are described as being constitutively susceptible to Mexican bean beetles (National Soybean Germplasm Collection database, Urbana, Ill.), but they vary in the strength of their induced responses to Mexican bean beetle damage (in preparation). Soybean seed was obtained from North Carolina State University, Raleigh, NC. Soybeans were planted in 4-inch plastic pots filled with a mixture of soil, peat, perlite, sand, and lime, and grown in a greenhouse under a 14-h daylength with supplemental lighting (430 W high-pressure sodium lamp). This daylength was sufficient to prevent the plants from becoming reproductive, so that all plants used in these experiments were in the vegetative stage. Plants were watered daily and fertilized twice weekly with Peters soluble fertilizer (20-10-20). Plants were not deliberately inoculated with *Rhizobium*, but haphazard sampling of roots indicated that plants often had nodules which appeared to be active.

Mexican bean beetles (*Epilachna varivestis*: Coccinelidae) are specialists on legumes and are a common economic pest of soybeans in the midwestern United States. Mexican bean beetles lay eggs on the host plant and both larvae and adults feed primarily on foliage. In North Carolina, Mexican bean beetles can complete three generations in a summer. Mexican bean beetles for this study were obtained from a New Jersey Department of Agriculture laboratory colony. Beetles were reared in an environmental chamber with a 14-h daylength at 28°C, and fed on common snap bean (*Phaseolus vulgaris*) plants to prevent beetles from becoming accustomed to any particular soybean genotype.

Experimental design

The design of the experiment was to damage plants in one discrete event and then measure the level of induced resistance by bioassay at five times following damage. The five sample times were chosen based on a pilot experiment showing that induced resistance was present by day 3 and completely decayed by day 20 (unpublished data). The experiment described here was carried out in four temporal blocks from January through September 1995. I chose to use a bioassay to measure induced resistance rather than chemical analysis because the focus of my studies is the effect of induced resistance on the herbivore. The induction response of soybeans is physiologically complex and not completely understood (Felton et al. 1994). Using the response of the beetles themselves is the most direct way to obtain information on the impact of induced resistance on the beetles.

For each temporal block, I grew 30 soybean plants of each genotype in the greenhouse. When the plants had one fully expanded trifoliate leaf (their first two true leaves are simple leaves), half the plants were randomly chosen to receive damage. Four or more third- or fourth-instar Mexican bean beetle larvae were placed on the first trifoliate leaf of each damaged plant and confined there with a mesh bag. I used enough larvae to completely consume the leaf within 48 h, adding larvae to some plants after 24 h to ensure that damage was completed within 48 h. One fully eaten trifoliate leaf constitutes damage to approximately 60% of the leaf area of the plant at that stage of growth. I chose the 60% damage level because this amount of damage has been shown to induce resistance in many genotypes of soybean (personal observation). All control plants also had their first trifoliate leaves bagged, but no larvae were added.

The time course of induced resistance was measured by assessing the feeding preference of Mexican bean beetle adults for undamaged leaves from control versus previously damaged plants at five sampling times (3, 10, 15, 20, and 40 days) following the end of damage. On each sampling date, three pairs of damaged and control plants from each genotype were randomly chosen, and leaf disks were cut from their most recently expanded, undamaged, leaves for use in a bioassay. Thus, leaves from the damaged plants had not received any direct damage, so the bioassay tested for systemic induced resistance. Although no data were taken on the number of leaves on each plant at sampling, there were no obvious differences in plant size (developmental age of leaves) between damaged and control plants. Leaf disks were cut with a cork borer, which has been shown not to cause induced resistance in the disks (Kogan and Fischer 1991). For each genotype this resulted in three samples in each of four temporal blocks, yielding a total of 12

[1] I should note here that I use the term induced resistance to describe *any* change in a plant resulting from damage and having a negative effect on the damaging organism, regardless of whether or not this response has evolved due to selection imposed by the damaging organism. The term induced defense is used to refer to induced responses which have been selected for because of their protective function (Karban and Baldwin 1997).

replicates for each of five sampling times following damage. Plants were discarded after being sampled, so that no plant was used more than once in the experiment.

Bioassay

I measured induced resistance as beetle feeding preference for leaf tissue from a control plant over tissue from a damaged plant. Four 2-cm-diameter leaf disks from each pair of control and damaged plants (two control disks and two "damaged" disks) were placed in a 9-cm-diameter petri dish lined with damp filter paper. Control and "damaged" disks were arranged opposite each other at the edges of the dish and one female Mexican bean beetle, previously starved for 24 h at 28°C, was placed in the dish. Females were used because adult females have been shown to eat more tissue and to be more discriminating than males (Smith et al. 1979). The beetles were allowed to feed until approximately 25% of the total leaf area in the dish was consumed, or for 24 h, whichever came first. The area of tissue consumed was measured using the Image 1 image analysis program (Universal Imaging Corporation 1991) on a Gateway 486 computer with a BURLE black-and-white video camera (model TC7311). I ran two choice tests (two petri dishes) for each plant pair to minimize lost data due to beetles occasionally not eating. Data from the two dishes from each plant pair were averaged to form a single observation (replicate).

Analysis

I used the consumed area of the control (c) versus damaged (d) disks to calculate a preference index [PI $= 2(c/(c + d))$] (Kogan 1972) for each dish. A PI of 1 indicates no preference (no difference in feeding between damaged and undamaged plants) and PIs higher than 1 indicate a preference for the control (rejection of previously damaged plants). The mean PI of all observations from a single sample time since damage, pooled over four blocks, indicates the degree of induced resistance at that time after damage. Statistical analyses were performed on the ratio of amounts of the control and damaged disks eaten ($c/(c + d)$) rather than on the PIs, and these ratios were arcsin square-root transformed before analysis to normalize their distributions (Zar 1984).

I used analysis of variance (GLM procedure of SAS; SAS 1989) to determine whether genotypes differed significantly in the timing of their induced response, with sample time, genotype, and block as factors. In addition, I used a two-tailed t-test to determine whether the PI from each sample time since damage was significantly different from 1 (no preference). I pooled data over genotypes for these t-tests, because ANOVA indicated no difference among genotypes. The t-tests were Bonferroni corrected for multiple comparisons. Finally, to test whether strong early induced resistance was followed by strong susceptibility, I determined the correlation between the PI from the first (most resistant) and fourth (most susceptible) sample date for each genotype (CORR procedure of SAS; SAS 1989).

Results

Analysis of variance indicated that induced resistance (as measured by the preference of adult beetles for undamaged leaves) did change over time (significant effect of sample time) (Table 1). However, the time course of induced resistance did not vary among genotypes (no effect of genotype or interaction of genotype with sample time) and the timing of induced resistance did not vary among temporal blocks (no effect of block) (Table 1).

The production of induced resistance was relatively rapid, with the highest level of resistance observed 3 days after damage ceased. Induced resistance had fully decayed by 15 days after damage (Fig. 2). Between 15 and 20 days after damage there was a period of increased susceptibility to beetles (beetles preferred previously damaged plants to undamaged plants). This increased susceptibility was followed by a return to no difference between the damaged and control plants by 40 days after damage. I used two-tailed t-tests on the data pooled over genotypes to confirm the existence of both the initial induced resistance and subsequent increased susceptibility. On the first sample date (day 3), the PI was significantly greater than 1 (indicating induced resistance $t = -5.22$, $df = 46$, $P < 0.001$) and the PI on the fourth sample date (day 20 after damage) was signifi-

Table 1 Analysis of variance for induction as a function of time since damage and genotype. The acceptability to herbivores of damaged plants relative to undamaged plants changed with time since damage (significant effect of time since damage), but there was no difference among the four genotypes of soybean in timing of responses (no significant effect of genotype or genotype by timing interaction)

Source	*df*	Type III SS	*F*	*P*
Block	3	0.8649	1.79	0.1506
Genotype	3	0.5639	1.17	0.3240
Time	4	0.4664	7.23	0.0001*
Genotype * time	12	0.1602	0.83	0.6218
Error	208	33.55		
Total	230	41.27		

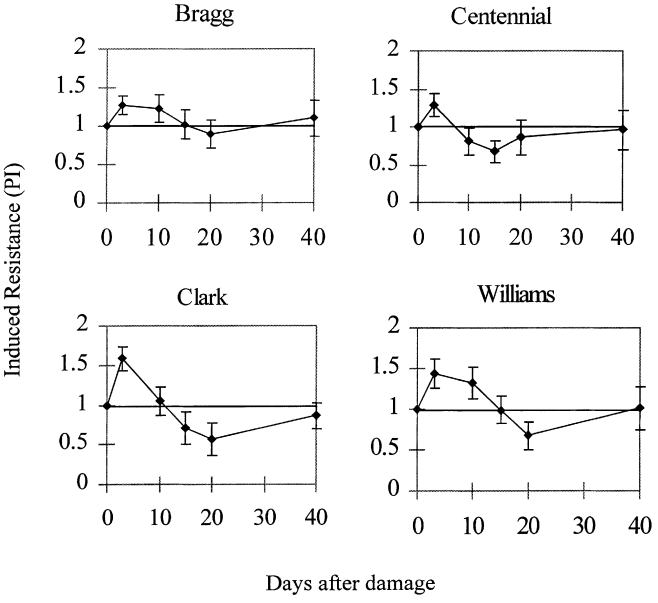

Fig. 2 Induction (measured by beetle preference for undamaged versus damaged plants) as a function of time since damage for four genotypes of soybean. A preference index (*PI*) of 1 indicates no preference (no induced response). PI values >1 indicate induced resistance and PI values <1 indicate induced susceptibility. The sample size for each point = 12, *error bars* indicate SEs

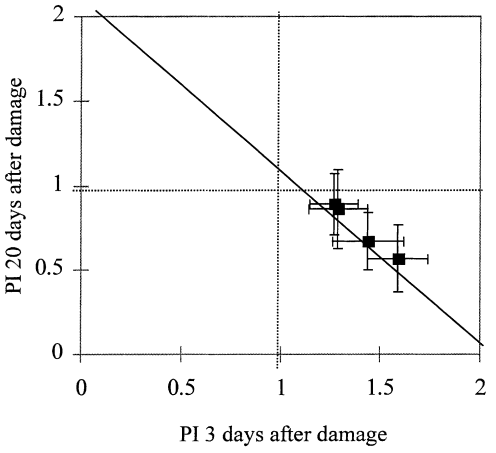

Fig. 3 Correlation between strength of induced resistance (PI on day 3) and induced susceptibility (PI on day 20) for four genotypes of soybean (PI day 20 = −0.71 * PI day 3 + 1.69, $R^2 = 0.98$, $P < 0.01$). Induced responses are measured as beetle preference for damaged versus undamaged plants. A preference index (*PI*) of 1 indicates no preference (no induction). PIs > 1 indicate induced resistance, PIs < 1 indicate induced susceptibility. *Each point* represents the mean of four blocks for a single genotype. *Error bars* indicate SEs

cantly less than 1 (indicating increased susceptibility $t = 2.75$, $df = 47$, $P < 0.01$). All other dates were not significantly different from 1 (Fig. 2).

Although the genotypes did not differ significantly in PI when pooled over all sample times, the magnitude of resistance and susceptibility did appear to differ among genotypes (Fig. 2). Interestingly, the magnitude of induced resistance for all cultivars (PI 3 days after damage) was strongly correlated with the magnitude of induced susceptibility (PI 20 days after damage) ($R^2 = 0.98$, $P < 0.01$) (Fig. 3). This resulted in the soybean genotype with the lowest peak induced resistance (Bragg) having the highest level of induced resistance over time [area between the line indicating no induced resistance (PI = 1) and the curve above the line, minus the area of the curve below the line] and vice versa.

Discussion

The most surprising result of this study is the discovery of a period of enhanced susceptibility following induced resistance. Although induced susceptibility without induced resistance has been observed in a variety of systems (e.g., Kielkiewicz 1988; English-Loeb and Karban 1991; Strauss 1991; Karban and Baldwin 1997), most previous experimental studies of the time course of induced resistance stopped when resistance returned to constitutive levels (e.g., Green and Ryan 1972; Baldwin 1988; McCloud et al. 1995; Zangerl and Berenbaum 1995) – too soon to know whether induced susceptibility would have followed resistance. Only one other study to date has reported susceptibility *following* resistance (Roland and Myers 1987; and see Seldal et al. 1994). Susceptibility that follows induced resistance may affect herbivore populations quite differently from induced susceptibility or induced resistance alone.

The induced susceptibility found in this study is not likely to be an experimental artifact. Samples on each date were taken from independent plants, preventing any effect of damage done by sampling on the induced responses. Further, since each data point is a comparison of damaged and control plants of the same age, aging of the plants alone could not be responsible for enhanced susceptibility. Most importantly, this experiment was carried out in four temporal blocks, so that induced susceptibility could not be caused by some external event in time (such as seasonality). Indeed, the fact that there was no significant effect of block in this experiment is interesting in its own right, contrasting with previous evidence that induced resistance varies a great deal among different environments (Karban 1987). Finally, it is striking to note that the same pattern of induced resistance and susceptibility was observed in four different genotypes, suggesting that enhanced susceptibility following induced resistance may be characteristic of induced resistance in soybeans in general.

The induced resistance observed in this experiment is likely due at least in part to induction of chemical resistance factors. Insect herbivore damage is known to provoke a variety of chemical responses in soybeans (Kogan and Fischer 1991; Felton et al. 1994), and these responses are known to affect Mexican bean beetle feeding and performance (Kogan and Fischer 1991). Other changes that may be provoked by herbivore damage and which might contribute to induced changes in beetle preference for plants include changes in leaf nutrient content and changes in plant growth rates (Schultz 1988). Regardless of the mechanism, the pattern of effect on beetle feeding preference remains. Mexican bean beetle feeding preference and performance on soybean plants seem to be closely correlated (unpublished data), suggesting that the induced resistance and susceptibility observed in this experiment are likely to affect beetle performance, and thus might affect beetle population dynamics.

The observation that induced susceptibility follows induced resistance in soybeans suggests that in this system induced resistance might cause induced susceptibility. For example, if the induced resistance observed in this experiment is due to changes in plant chemistry, and assuming that the resources a plant is able to invest in resistance are limited (Simms 1992), the production of induced resistance might require reductions in constitutive resistance levels, resulting in subsequent enhanced susceptibility to herbivores. If resistance and susceptibility are causally related, we would expect that the greater the induced resistance response in a genotype, the greater the increase in susceptibility. In fact, in this experiment, the magnitudes of induced resistance and susceptibility in individual genotypes were strongly

positively correlated (Fig. 3). This result is consistent with the hypothesis that induced susceptibility is the result of induced resistance, and could represent two kinds of costs of resistance to the plant. The production of induced resistance might impose physiological costs (Zangerl et al. 1997) that result in induced susceptibility, as described above. In addition, the period of induced susceptibility itself could constitute a second cost of the production of induced resistance measured in increased herbivore damage. It is also possible that induced resistance, and induced susceptibility are not causally related, but are independent events both triggered in the same magnitude by damage. Damage by herbivores can cause many changes in plant tissues (Karban and Baldwin 1997). Some of these changes may have negative effects on herbivores while others may have positive effects. If negative effects are more short-lived than positive ones, induced susceptibility would only be measurable once induced resistance subsides. For instance, damage might trigger an immediate increase in resistance due to changes in plant chemistry, but might also trigger overcompensating regrowth, resulting in damaged plants being more favorable for insects than less actively growing plants (Price 1991). In this case, susceptibility would not represent a direct cost of resistance, but would constitute a consistent force opposing the effects of resistance on herbivore populations.

Implications

Many authors have suggested that induced resistance may have important effects on herbivore population dynamics, such as regulating populations or driving fluctuations in population size (e.g., Haukioja 1980; Rhoades 1985; Edelstein-Keshet and Rausher 1989). But the occurrence of enhanced susceptibility following induced resistance could substantially alter the predicted impact of induced resistance on herbivore populations. It seems likely, for instance, that a period of increased susceptibility following resistance would lessen the negative effect of induced resistance on herbivores, resulting in increased growth of herbivore populations. Alternation between induced resistance and induced susceptibility that occurs on a long time scale relative to herbivore generation time might influence the degree of fluctuations in herbivore population size. Induced susceptibility might also alter herbivore movement on a large scale, perhaps slowing herbivore spread relative to induced resistance alone.

Induced susceptibility following induced resistance might also change the predictions of existing theory for the conditions under which induced defenses might evolve (Karban and Baldwin 1997). If resistance and susceptibility are genetically or physiologically linked in a particular system, such that increased resistance always entails subsequent increased susceptibility, susceptibility could counteract any benefits of resistance and prevent selection for induced defenses. If resistance and susceptibility are not linked, strong selection could act to decouple the two events, leading to genotypes with high induced defense and no induced susceptibility.

Conclusion

The observation in this study of a time course of induced responses to herbivory that is more complex than the great majority of previous studies have indicated suggests that we may not yet fully understand the time course of induced responses to herbivory. It is obviously important to determine the generality of the pattern observed in this study – whether it holds up in this system in the field, and whether other systems behave in similar ways. The predictions of existing conceptual and mathematical models for how induced resistance affects herbivore dynamics and for the evolution of induced resistance may need to be re-examined if this pattern proves to be a general one.

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