Growth Inhibitors in Host Plant Resistance to Insects: Examples from a wild tomato with *Heliothis zea* (Lepidoptera: Noctuidae)[1]

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ABSTRACT Non-lethal, growth inhibiting allelochemicals have potential roles in host plant resistance to insects because they can extend the time the insects are exposed to other mortality factors. Four chemical constituents of the wild tomato, *Lycopersicon hirsutum* f. *glabratum,* PI 134417 were evaluated in artificial diet as growth inhibitors to *Heliothis zea* (Boddie): alpha-tomatine, chlorogenic acid, beta-caryophyllene and alpha-humulene. All caused small increases in developmental time, and, except for chlorogenic acid, small decreases in pupal weight. These changes may be too small to be of biological significance in host plant resistance. Our results suggest that the usual method of evaluating growth inhibitors, which is to weigh the insects after a fixed feeding period, is inadequate to assess the biological significance of any observed growth inhibitory effects. Because differences in weight after fixed feeding periods do not always translate into equivalent changes in developmental time or final weight, measurement of actual developmental time may be more appropriate.

KEY WORDS Growth inhibitor, *Heliothis zea,* tomato, alpha-tomatine, chlorogenic acid, beta-caryophyllene, alpha-humulene.

In recent years, host plant insect resistance based on non-lethal antibiotic allelochemicals has been the subject of considerable research. Such substances, or growth inhibitors, do not cause high rates of mortality directly, but rather extend developmental time and reduce the size of the insects. The pest is thus exposed to other mortality factors (e.g. natural enemies, weather factors, pesticides) for a longer period of time. The growth inhibitor may also reduce the vigor of the pest, rendering it more susceptible to these other factors. Clancy & Price (1987) review the literature on the relationship between insect developmental time and susceptibility to natural enemies. The use of growth inhibitors as mediators of plant resistance has been discussed by Duffey & Bloem (1986) who suggest that this approach may also reduce the probability of the pest evolving tolerance to the resistant plant, as compared to the probability when lethal allelochemicals are used.

The research reported herein was undertaken to evaluate the growth inhibitory potential of some allelochemicals to the tomato fruitworm, *Heliothis zea* (Boddie), and, using these results, to evaluate the usual methods of measuring growth inhibition as a predictor of the overall effect on larval growth and development. It

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should be noted that there is no intention in this paper to evaluate bioassays for all of the varied aspects of host plant resistance, only larval growth and development. The allelochemicals are all constituents of the wild tomato, *Lycopersicon hirsutum* f. *glabratum* C. H. Mull, accession PI 134417: alpha-tomatine (a glycoalkaloid), chlorogenic acid (a phenolic), beta-caryophyllene and alpha-humulene (sesquiterpenes).

PI 134417 is highly resistant to *H. zea*, as well as the tobacco hornworm, *Manduca sexta* (L.) and the Colorado potato beetle, *Leptinotarsa decemlineata* (Say) (Kennedy et al. 1985; Kennedy 1986; Kennedy et al. 1987). Resistance to *M. sexta* and *L. decemlineata* is conditioned largely by the presence of large amounts of 2-tridecanone in the tips of type VI (Luckwill 1943) glandular trichomes on the foliage (Kennedy & Dimock 1983; Kennedy & Sorenson 1985; Kennedy et al. 1985). 2-Tridecanone, however, confers only a small portion of the resistance of PI 134417 to *H. zea*. Neonate larvae placed on the foliage, or on filter paper treated with 2-tridecanone, rapidly become paralyzed, but approximately 80% recover within a few hours, and, on foliage, begin feeding, only to die within a few days (Dimock & Kennedy 1983; Kennedy & Dimock 1983; Kennedy 1984, 1986; Farrar & Kennedy 1987a).

Farrar & Kennedy (1987b, 1988) found that a second trichome constituent, 2-undecanone, is active against *H. zea*. In combination with 2-tridecanone in artificial diet, it synergistically increases mortality of young larvae. Similar synergism has also been observed for *Keiferia lycopersicella* (Walsingham) and *Spodoptera exigua* (Hübner) (Lin et al. 1987). When ingested by fifth instar *H. zea* larvae, 2-undecanone also causes mortality and deformity of pupae, both alone and in combination with 2-tridecanone (Farrar & Kennedy 1987b, 1988).

The slow growth and excessive mortality of *H. zea* larvae that have recovered from their initial 2-tridecanone-induced intoxication on PI 134417 foliage (Dimock & Kennedy 1983) suggests the presence of growth inhibitors (Farrar & Kennedy 1987a).

Alpha-tomatine occurs in various species of solanaceous plants of the genera *Lycopersicon* and *Solanum* (Schreiber 1968; Roddick 1974). It is found in leaf lamellae and unripe fruit of tomatoes, but is degraded in ripe tomato fruit (Juvik & Stevens 1982). Elliger et al. (1981) reported that PI 134417 foliage contained 0.245% (wet wt.) alpha-tomatine, compared with 0.061% to 0.076% for susceptible *L. esculentum* cultivars. Recently, however, Barbour (1987) reported that levels of alpha-tomatine in PI 134417 were approximately the same as, or lower than, levels in the susceptible cultivar 'Walter' (2.077% vs. 2.293% (dry wt.) for PI 134417 and Walter, respectively, in the greenhouse, and 0.439% and 0.860% (dry wt.) respectively, in the field). Alpha-tomatine, incorporated into artificial diet, and has been reported to reduce the size of *H. zea* larvae after prescribed feeding periods. Elliger et al. (1981) found the concentration needed to reduce larval weight at 12 days by 50%, the "ED_{50}," to be 0.04% (wet wt.); Isman & Duffey (1982b) reported an 8 day ED_{50} of 0.09% (wet wt.). Juvik & Stevens (1983) correlated similar effects with alpha-tomatine levels in tomato fruit. These effects have not been demonstrated to occur in tomato foliage, however.

The phenolic compound, chlorogenic acid, occurs in a diverse array of higher plants (Sondeimer 1964; Harborne 1979). Elliger et al. (1981), using whole-leaf extractions, reported PI 134417 foliage to contain 0.03% (wet wt.) chlorogenic acid; P. Gregory (unpublished), analyzing only glandular trichomes, reported 0.119%.

Like alpha-tomatine, chlorogenic acid has been shown to reduce larval weight of *H. zea* on artificial diets after prescribed feeding periods. Elliger et al. (1981) found a 12 day ED_{50} of 0.25% (wet wt.); Isman & Duffey (1982b) found an 8 day ED_{50} of 0.60%. Similar results have been found for the phenolic-rich trichome exudates of *L. esculentum* (Duffey & Isman 1981; Isman & Duffey 1982a, b). By contrast, chlorogenic acid acts as a feeding stimulent for *L. decemlineata* (Hsiao & Fraenkel 1968 a,b).

Beta-caryophyllene and alpha-humulene likewise occur in a diverse array of plants. Plants from which beta-caryophyllene has been reported include corn, *Zea mays* L. (Buttery & Ling 1985), cotton, *Gossypium hirsutum* L. (Elzen et al. 1984, 1985), clove, *Eugenia caryophyllata* Thunb, (Muchalal & Crouzet 1985, Uchida et al. 1986), hops, *Humulus lupulus* L. (Chapman 1903, Uchida et al. 1986), pepper, *Piper marginatum* (Ramos et al. 1986), and lemon, *Citrus* (Lund et al. 1982). Plants in which alpha-humulene occurs include cotton (Elzen et al. 1984, 1985), hops (Chapman 1903), pepper (Ramos et al. 1986), clove (Muchalal & Crouzet 1985), and carrots, *Daucus carota* L. (Seifert & Buttery 1978). In PI 134417, beta-caryophyllene and alpha-humulene are found in the tips of type VI trichomes, where they occur in concentrations of ca. 0.035% and 0.007%, respectively, by fresh foliage weight (P. Gregory personal comm.). Biological activity involving insects has been reported for both sesquiterpenes. Beta-caryophyllene is an attractant for male boll weevils, *Anthonomus grandis* Boheman (Hedin et al. 1979), but inhibits response by *Myzus persicae* (Sulzer), to alarm pheromones (Dawson et al. 1984; Pickett et al. 1984); it also induces cytochrome P-450 and other detoxifying enzymes in tobacco budworms, *Heliothis virescens* (F.), and boll weevils (Brattsen 1987). Alpha-humulene is also an inhibitor of the alarm pheromone response in *M. persicae,* though its effects are weaker than beta-caryophyllene (Dawson et al. 1984). It is also attractive to the *Heliothis* larval parasitoid *Campoletis sonorensis* (Cameron) (Elzen et al. 1984). Gunasena et al. (1988) reported that two related compounds, caryophyllene and caryophyllene oxide, reduced larval weights of *H. virescens* after 7 d feeding periods (ED_{50} = 4 mg/ml and 1 mg/ml, respectively). They also found that relatively low doses of caryophyllene, while reducing larval weight at 7 d and increasing developmental time, did not reduce pupal weight; only higher doses reduced weight at both 7 d and pupation.

Materials and Methods

All experiments were conducted using commercially obtained chemicals (alpha-tomatine and chlorogenic acid: Sigma Chemical Co., P.O. Box 14508, St. Louis, MO 63178 USA; beta-caryophyllene: Pfaltz and Bauer, Inc., Stamford, CN 06902 USA; alpha-humulene: Fluka Chemical Corp., Hauppauge, NY 11788) incorporated into a corn-soya-milk (CSM) artifical diet (Burton 1970). For all chemicals except alpha-tomatine, diet was prepared in batches of ca. 4000 ml. 500 ml portions of warm (ca. 45°C), liquid diet were poured into a 1000 ml blender, measured amounts of the chemicals were added, and blended into the diet at high speed for 1 min. Diet was then poured into 30 ml plastic cups (ca. 15 ml/cup) and allowed to cool. For alpha-tomatine, diet was prepared in small batches of ca. 240 ml. A solution was prepared of the vitamin portion of the diet with ca. 48% of the water. Alpha-tomatine, insoluble in plain water, was dissolved in this solution. This solution was then blended with the remaining diet ingredients and poured into 30 ml plastic cups. The chemicals were tested at the following rates (wet wt./wet wt.):

alpha-tomatine, 0.08% and 0.245%; chlorogenic acid, 0.119%, beta-caryophyllene, 0.035%; and alpha-humulene, 0.007%. The alpha-tomatine rates are based on Elliger et al. (1981). Barbour's recent (1987) findings suggest that these rates are not representative of PI 134417 and Walter; however, they are representative of high and low level's found in various tomato lines (Juvik et al. 1982). Rates of the other chemicals are representative of those found in PI 134417 foliage. Only one chemical, plus an unadulterated control, was tested at a time.

Neonate *H. zea* larvae were obtained from a stock culture that is maintained on CSM diet at North Carolina State University. A new colony is started annually with field-collected insects. Ten larvae were placed in each cup and held at 27°C. Mortality was recorded at 48 h; the larvae were thinned to one per cup at 72 h. Remaining insects were reared through adult eclosion at 27°C. Mortality between 72 h and pupation, pupal mortality, pupal weight, and developmental times were recorded. Pupae were classified as normal or deformed (unable to completely shed the last larval exuviae; Farrar & Kennedy 1987b). In addition, larvae on the alpha-tomatine diet were weighted at 3, 6, 10 and 13 d. 64 cups were set up in 4 groups of 16 (replicates over time) for each rate of alpha-tomatine; 54 in 2 groups of 27 for each chlorogenic acid, beta-caryophyllene, and alpha-humulene. Equal numbers of controls (untreated diet) were set up with each chemical treatment.

Percentage mortality at 48 h was calculated for each cup ((number dead/10) x 100) and transformed to arcsine $\sqrt{\%}$ prior to analysis of variance, with each cup as an observation. Data on developmental times, and larval and pupal weights were also analyzed by analysis of variance. There were insufficient numbers of insects to calculate multiple values of percentage mortality (after thinning) and thereby be able to use analysis of variance. Therefore, larval mortality (after thinning) and pupal mortality and deformity data were pooled across replications and analyzed by Chi square tests (Little & Hills 1972). All chemical treatments were compared only with their respective controls, except for alpha-tomatine, for which the two rates were compared as well as the control.

Results and Discussion

None of the chemicals tested caused high rates of mortality of any stage of *H. zea* (Tables 1 and 2). Alpha-tomatine did cause a small but statistically significant ($P \leq 0.05$) increase in mortality in both larval and pupal stages. Mortality of pupae on the control of the alpha-tomatine test was higher than on the low rate; this result is probably anomalous, however. Late larval and pupal mortality were fairly high on both the beta-caryophyllene diet and its control, though no significant treatment effects were seen ($P \geq 0.05$); the reasons for these results are not clear as values of other parameters were comparable to those in the other tests. None of these chemicals caused pupal deformity of the type seen when *H. zea* is reared on diet with 2-undecanone (Farrar & Kennedy 1987b, 1988). All of the chemicals except alpha-humulene caused significant ($P \leq 0.05$) increases in developmental time measured at both pupation and adult eclosion; alpha-humulene caused a significant ($P \leq 0.05$) increase in time to pupation, but not in time to eclosion.

In the alpha-tomatine test, significant ($P \leq 0.05$) effects of alpha-tomatine concentration on insect weight were found on each day the insects were weighted (Fig. 1). These results were consistent with previous reports of 50% reduction in larval size at 12 days by 0.04% alpha-tomatine (Elliger et al. 1981) and at 8 days by 0.09%

Table 1. Effects of alpha-tomatine in artificial diet on survival and growth of *H. zea*.

Parameter	Mean on rate, %			F	X2	d.f.	Probability
	0.0	0.08	0.245				
Larval Mortality 0-48 h, %*	4.7	5.0	12.2	24.01	—	1	<0.0001
Larval Mortality 72 h - pupa, %†	0.0	2.0	6.0	—	2.32	1	0.1277
Pupal Mortality, %†	17.0	5.0	30.0	—	13.66	1	0.0022
Days to Pupa*	13.8	15.9	18.9	224.50	—	1	<0.0001
Pupal wt., mg. *	446.0	425.5	381.7	30.91	—	1	<0.0001
Pupal Deformity, %†	2.0	2.0	7.0	—	1.95	1	0.1626
Days to adult*	24.0	26.5	29.1	151.83	—	1	<0.0001

* Analysis of variance for effects of alpha-tomatine rate.
† Chi-square tests are for differences between highest and lowest values.

alpha-tomatine (Isman & Duffey 1982b). In our case, 0.08% alpha-tomatine caused a 47% reduction in size at 10 days compared to the control. Increasing the rate to 0.245% reduced weight at 10 days by another 52%. However, pupal weights of those insects on the low rate were only 4.6% less than those on the control, while weights of those pupae on the high rate were 10.3% less than the ones on the low rate. Similarly, larval developmental time was increased only 15.2% by the low rate, and were increased by the high rate, an additional 18.9% over the low rate.

The other three chemicals also showed growth-inhibiting activity. Chlorogenic acid, beta-caryophyllene and alpha-humulene increased larval developmental time by 3.9%, 9.2%, and 8.3% respectively, and decreased pupal weight by 1.2%, 6.5% and 7.1%, respectively. All of these differences except pupal weight for chlorogenic acid were statistically significant ($P \leq 0.05$). Though not directly comparable, our results for chlorogenic acid seem consistent with those of Elliger et al. (1981) and Isman & Duffey (1982b). Our sesquiterpene results were similar to those of Gunasena et al. (1988) for caryophyllene and caryophyllene oxide.

None of the chemicals in these tests seems to provide an adequate explanation for the effects of PI 134417 foliage on *H. zea*, or appears promising for use in host plant resistance. Unlike PI 134417 foliage (Farrar & Kennedy 1987a), none caused much larval mortality, or drastically reduced growth. It is possible, however, that the chemicals do not act in diet as they do in intact plants. The activity of alpha-tomatine can be nullified by dietary sterols at levels comparable to those in tomato foliage (Duffey & Bloem 1986). This may be why its activity in intact foliage has not been demonstrated. Duffey & Bloem (1986) also report that the toxicity of a phenolic compound, rutin (which is equitoxic to chlorogenic acid in *H. zea* (Elliger et al. 1981)) is dependent on quantity and quality of dietary protein.

Even if they act in intact plants as they do in artificial diet, these chemicals, and any others that act similarly, are not likely to be of great value as mediators of insect resistance appropriate for use in breeding resistant tomato cultivars. A high level of alpha-tomatine could be of some value as a mortality agent, although, at least in this test, increases in mortality were small, and occurred mostly in the pupal stage. As discussed above, the usual rationale for using non-lethal growth

Table 2. Effects of chlorogenic acid, beta-caryophyllene and alpha-humulene in artificial diet on survival and growth of *H. zea*.

Treatment	Rate, % (wt./wt.)	Mean % Mortality in Stage: larva, 0-48 h*	larva, 72 h-pupa†	Pupa†	Mean Days to pupa*	Mean Pupal wt., mg.*	Mean % Pupal Deformity†	Mean Days to Adult*
Control	-	4.5 A	3.9 A	6.3 A	15.2 A	479.7 A	0.0 A	26.5 A
Chlorogenic acid	0.119	4.8 A	3.9 A	6.1 A	15.8 B	473.8 A	2.0 A	27.5 B
Control	-	1.2 A	18.7 A	43.2 A	16.3 A	406.0 A	2.6 A	26.7 A
Beta-caryophyllene	0.035	1.2 A	34.6 A	40.0 A	17.8 B	379.6 B	8.3 A	27.8 B
Control	-	6.0 A	17.4 A	15.0 A	13.3 A	416.0 A	14.0 A	23.4 A
alpha-Humulene	0.007	5.2 A	6.2 A	13.6 A	14.4 B	386.3 B	7.7 A	23.8 A

* Mean separation vertical for each chemical and its control $P \leq 0.05$, F-test.
† Mean separation vertical for each chemical and its control $P \leq 0.05$, Chi-square.

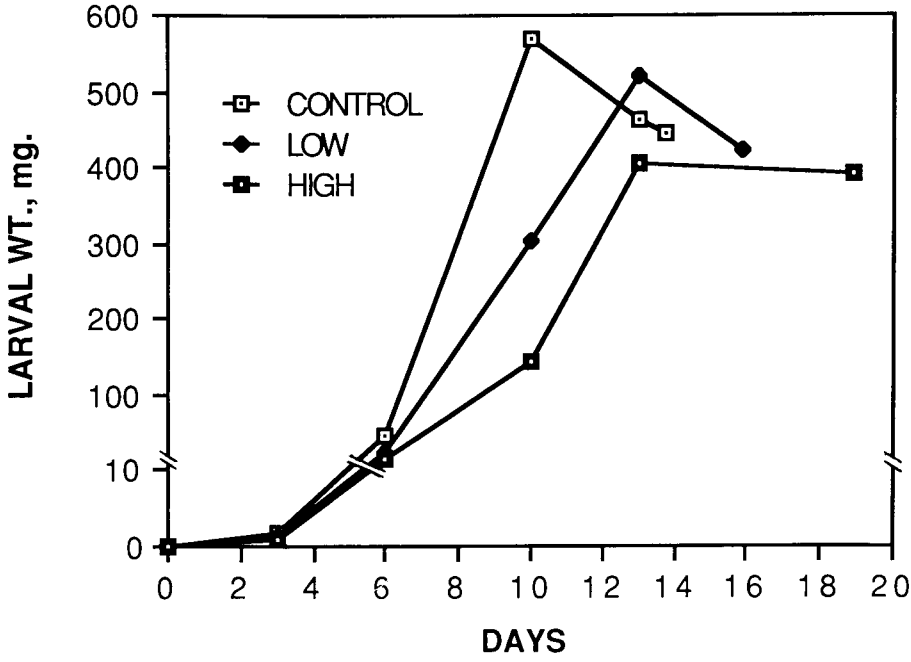

Fig. 1. Changes in weight of *H. zea* from hatching to pupation on artificial diet containing 0.08% alpha-tomatine, 0.245% alpha-tomatine or no alpha-tomatine (control). Larval weights on each day (3, 6, 10, 13), pupal weights, and developmental times were all significantly ($P \leq 0.05$) affected by alpha-tomatine concentrations.

inhibitors in host plant resistance is that they increase the length of time that the pest is vulnerable to other mortality factors (Duffey & Bloem 1986, Clancy & Price 1987). On tomato *H. zea* larvae usually feed on foliage for the first few instars where they are vulnerable to natural enemies and other mortality factors, then enter the fruit, where they are largely protected. A growth inhibitor that acts as those in our tests did, would thus only extend the vulnerable stage by a day or two. Subsequent damage by each surviving larva would likely be nearly as severe as without the growth inhibitor, since the larvae are protected within the fruit and eventually attain nearly the same size.

One commonly used method of evaluating plant allelochemicals for activity as insect growth inhibitors involves rearing insects on diets containing various concentrations of the test chemical, and weighing them at one given time (e.g. 10 days). The concentration that reduces size by 50% at that time, the ED_{50}, is then calculated. Evaluation of, and comparisons between, chemicals are then made based on their respective ED_{50}'s.

Our results suggest that this method may exaggerate the effects of some chemicals, however. Because of the sigmoidal shape of many insect growth curves (e.g. Fig. 1; see also Kogan [1986]), a 50% reduction in growth after a fixed time

interval does not always translate into a 50% change in either developmental time or final weight. In our alpha-tomatine test, a treatment which caused a 47% reduction in size at 10 days caused only a 4.6% reduction in pupal weight and a 15% increase in developmental time. Similar results were obtained by Gunasena et al. (1988) for caryophyllene and caryophyllene oxide. In cases such as these, measurements of weight at a given time will often exaggerate the overall effect of the growth inhibitors.

There are at least three ways in which host plant resistance mediated by insect growth inhibitors can be used to enhance crop protection. In some crops, simply slowing down the development and growth of a pest insect may facilitate management of the pest by extending the periods of time during which it can be readily controlled with insecticides or by allowing the crop to pass its sensitive growth stages before the insect pest reaches its most damaging stages. In these situations a knowledge of the effects of the resistance (growth inhibitor?) on developmental time may be adequate to fully predict potential benefits.

In other crop/pest situations, the effects of host plant resistance (growth inhibitors?) on the fecundity of the insect pest may be of particular importance to management of that pest. This would often be the case for species which cycle through multiple generations per year within a crop. There are, however, many crop/pest situations in which, from a pest management perspective, effects of resistance on pest fecundity are of little consequence. For example, most of the *H. zea* population in North Carolina feeds on corn for two generations per year, before moving to an array of late season hosts including tomato where a single generation is passed. *H. zea* on tomato represent only a very small fraction of the total population. Therefore, any effects of tomato allelochemicals on fecundity would confer no pest management benefit for the tomato crop and would have a negligible effect on the overall population of *H. zea.*

In those instances where effects on fecundity are of interest, it may be necessary to measure fecundity directly, or to measure final size, if the latter is highly correlated with fecundity. In such cases, reductions in insect size after a fixed interval on a resistant plant or a diet containing a suspected growth inhibitor could not be assumed to translate into comparable reductions in final size, or fecundity in those situations where final size and fecundity are correlated.

Perhaps the most commonly cited potential benefit of sublethal plant defenses involves their functioning to increase pest mortality by increasing the amount of time the target insect is vulnerable to biotic and abiotic mortality factors. The generality of this effect as it applies to enhanced mortality from parasitoids and predators is controversial and there are few field studies documenting the phenomenon (Clancy & Price 1987). There are, however, a number of cases in which the hypothesized effect is reasonable since many species of parasitoids and predators are more effective when attacking the small, early instars of their hosts/prey. The efficiency of *Cardiochiles nigriceps* Viereck (Hymenoptera: Braconidae), for example, is the highest when attacking early instars of its host, *Heliothis virescens* (F.), and decreases as the larvae grow (Neunzig 1969). This is not the case for all species, however; Clancy & Price (1987) documented higher rates of parasitism among the most rapidly developing individuals of a leaf galling sawfly. They attributed their results to the larger final size of fast growing individuals, which makes them a superior resource for the parasitoid they studied. Thus, any effect of altered developmental time on predation and parasitism rates will likely depend on the

species involved. The shape of the herbivore's growth curve is important in such cases since it indicates how long the herbivore remains in a vulnerable stage. Any influence of growth inhibitors on predation and parasitism rates will likely depend on how the growth curve is altered; provided the growth inhibitor is not toxic to the natural enemy. Bioassays measuring insect growth reductions after a fixed interval are useful preliminary indicators of potential effects, but determination of how the complete growth curve is affected and the population consequences of altered growth rates are needed to better assess the potential effects of growth inhibitors on the effectiveness of natural enemies.

In conclusion, our data demonstrate that measurement of the growth reducing effects of allelochemicals or resistant plants at a single point in time does not provide an unbiased indicator of the final effects on insect growth or of the ecological or pest management consequences of altered growth. To more fully assess the potential impact of growth inhibiting allelochemicals or resistant plants on a particular pest species, it is important to document the full nature of alterations in the insect's growth curve. To assess the actual ecological or pest management consequences of any alterations in insect growth, it is necessary to study the pest's population dynamics on plants differing in levels of the growth inhibiting resistance.

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