Inducible Responses in Papaya: Impact on Population Growth Rates of Herbivorous Mites and Powdery Mildew Under Field Conditions

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ABSTRACT Induced plant responses to herbivores and pathogens have been found in many systems. We examined intra- and interspecific interactions among three parasites through induced responses in their shared host plant, papaya. Three key parasites attack papaya foliage in Hawaii: the carmine spider mite, *Tetranychus cinnabarinus* (Boisduval); the papaya rust mite, *Calacarus flagelliseta* Fletchmann, De Moraes, and Barbosa; and the powdery mildew causal agent, *Oidium caricae* F. Noack. Under laboratory conditions, papaya seedlings were first exposed to standardized populations of mites and mildew; the parasites were removed, and the clean, previously infested plants were transplanted into the field to be exposed to colonization by natural populations of plant parasites. Population growth of colonizers was monitored for a period of 3 mo. We found no evidence for induced plant resistance. Rather, our results suggest that papaya expresses a weak form of induced susceptibility after injury from papaya rust mites and powdery mildew. Plants exposed to rust mites as young seedlings subsequently supported larger populations of spider mites, and plants exposed early to powdery mildew subsequently supported larger populations of rust mites.

KEY WORDS herbivorous mites, induced susceptibility, papaya, powdery mildew

IN RESPONSE TO DAMAGE IMPOSED by herbivores and pathogens, host plants may exhibit dynamic responses that can significantly change their quality as a resource for subsequent attackers (Karban and Baldwin 1997, Agrawal et al. 1999). In some cases, changes in the host plant produce induced resistance in which the performance of later attackers is depressed. In other cases, however, changes in the host plant can have the reverse effect, with subsequent attackers enjoying enhanced population growth rates; this is referred to as induced susceptibility (Karban and Baldwin 1997). The potential to harness-induced resistance as a component of pest management strategies in agriculture has recently stimulated a substantial research effort into inducible plant responses (Lyon and Newton 1999, Tally et al. 1999, Zehnder et al. 1999).

Two major pathways of inducible responses have been characterized in plants (Agrawal et al. 1999). The jasmonate pathway, also called the octadecanoid pathway, produces resistance against many arthropods. For instance, in tomato plants, the elicitor jasmonic acid can induce resistance against several species of herbivores such as aphids, thrips, caterpillars, and flea beetles (Thaler et al. 2001). In contrast, the salicylate pathway, which is commonly referred to as conditioning systemic acquired resistance (SAR), produces resistance against many pathogens. For example, in cucurbits and tobacco plants, it is well documented that infection by a virus, a bacterium, or a fungus can induce systemic resistance against a variety of pathogens (Kuc 1995). Increasing evidence suggests that these two pathways may interfere with each other (Stout et al. 1999, Thaler et al. 1999, 2002, Felton and Korth 2000, Hunter 2000).

Here, we examine intra- and interspecific interactions among three plant parasites that are mediated by induced responses of their shared host plant, papaya, *Carica papaya* L. (Caricaceae). Our study was exploratory in the sense that induced responses have not been documented in papaya. We decided to conduct a large scale, in-field experiment, because our main motivation was to examine induced responses under the most realistic conditions possible.

Papaya is a short-lived perennial that is native to Central America (Storey 1976). It was introduced to Hawaii some 200 yr ago (Yee et al. 1970) and is today among the 10 most important crops in the state (Chia et al. 1990). Seedlings are grown from seed in nurseries and transplanted in commercial orchards when they are a few months old. Papaya is a fast-growing crop that starts producing fruit within the first year after planting. Trees are usually grown for 3 yr, after which

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they become too tall to be efficiently harvested. Three key parasites attack papaya foliage in Hawaii: the carmine spider mite, *Tetranychus cinnabarinus* (Boisduval) (Acari: Tetranychidae); the papaya rust mite, *Calacarus flagelliseta* Fletchmann, De Moraes, and Barbosa (Acari: Eriophyidae); and the powdery mildew causal agent, *Oidium caricae* F. Noack (Erysiphales). The carmine spider mite is a worldwide polyphagous pest, mainly distributed in semitropical and tropical areas (Goff 1986). Usually found on the undersurface of papaya leaves, it punctures the epidermal cells of papaya leaves with its stylet-like mouthparts and sucks out the cell contents, producing a discolored area on the leaf (Jeppson et al. 1975). The papaya rust mite was first reported in Hawaii in the early 1990s (Hamasaki and Heu 1991) when it was mistakenly identified as *C. brionesae* (Fletchmann et al. 2001; J. Amrine, personal communication). It infests both surfaces of papaya leaves (Fournier et al., unpublished data), and when its population peaks, leaf rolls appear on the margin of the leaves (Fournier et al. 2003). Finally, powdery mildew is a common and economically significant disease of papaya (Yee et al. 1970). Its causal agent, *O. caricae*, is an obligate ectoparasitic fungus that is host-specific to papaya (Ooka 1994). It forms circular, white, and powdery-appearing colonies on the undersurface of the leaves. These three plant parasites infest papaya leaves year-round. However, populations of herbivorous mites usually peak in the spring and summer, whereas powdery mildew populations generally peak during winter months (Ooka 1994; Fournier et al., unpublished data).

In this study, our aim was to examine whether early damage by the carmine spider mite, the papaya rust mite, or powdery mildew could induce plant resistance or susceptibility, thereby influencing the subsequent colonization of papaya foliage by herbivores and pathogens. Our approach was to expose papaya seedlings to experimentally standardized populations of mites and mildew in the laboratory and to remove the parasites and transplant the clean papaya plants into a field setting where they were exposed to colonization by natural populations of these plant parasites.

Materials and Methods

Bioassay on Chemicals. Some pesticides have the capacity to elicit systemic plant resistance to herbivores and plant pathogens (Stout et al. 1994, Molina et al. 1998). Because we needed pesticides to kill the plant parasites used to challenge papaya seedlings (see below), it was important to determine whether these chemicals can induce plant resistance or susceptibility to herbivores. We performed a bioassay to examine the impact of a solution of the acaricide abamectin, the fungicide myclobutanil, and a spreader-sticker (phthalic/glycerol alkyl) on herbivores 10 d after their application on papaya seedlings. Seedlings were randomly assigned to one of two treatments, each replicated 10 times: seedlings dipped in water or seedlings dipped in a solution of pesticides (acaricide + fungicide + spreader-sticker, same doses as used in the main experiments). Ten days after the dipping, the seedlings had produced new leaves (not exposed to the chemicals). Five adult female spider mites were confined in a clip cage (3 cm diameter) secured on the new leaf. Seedlings were maintained in a growth chamber (25°C, L16:D8) for 7 d, after which all spider mite stages were counted using a dissecting microscope. We did not test whether the solution of pesticides could induce resistance or susceptibility to pathogens.

Field Experiments. Our research was carried out at the University of Hawaii Poamoho Experimental Station on Oahu, HI. To expose papaya plants to a wide range of colonizing plant parasite densities, we conducted an experiment on induced responses two times during different seasons: first, we performed the experiment during the fall and winter, when powdery mildew populations are usually highest (season 1), and second, we repeated the study during the spring and summer, when densities of herbivorous mites are usually highest (season 2).

Season 1. From 15 August 2001 to 10 January 2002, we conducted a manipulative experiment with five treatments: (1) control (no challenge); (2) spider mite-challenged; (3) rust mite-challenged; (4) powdery mildew-challenged; and (5) artificially damaged with an abrasive powder, carborundum ("carborundum-challenged"). The fifth treatment had been previously used to mimic feeding damage by herbivorous mites and has been shown to induce resistance to spider mites on cotton (Karban 1985). The experimental unit was a single papaya plant (mix of three varieties: 'Solo', 'Sunrise', and 'low-bearing Waimanalo'), and each treatment was replicated 42 times. The different replicates were set up in three batches that were temporally distributed (batch 1: 15 replicates, ran from 15 August to 6 December 2001; batch 2: 15 replicates, ran from 30 August 2001 to 8 January 2002; batch 3: 12 replicates, ran from 13 September 2001 to 10 January 2002). For each batch, we regrouped seedlings (groups of five seedlings) of similar height together and randomly assigned the five treatments within each block, for an overall total of 42 blocks (used for statistical purpose). Treatments were assigned randomly within each block. Seedlings were 2–3 mo old on day 0 of the experiment (11 ± 4 cm). Seedlings had never been exposed to herbivory before the beginning of the study. Within each batch, seedlings were induced under laboratory conditions on day 0. Seedlings assigned to treatment 1 (control) were unchallenged. Seedlings assigned to treatment 2 (spider mite-challenged) were inoculated with six to nine adult female spider mites. All spider mites were placed on a single leaf (midcanopy). Seedlings assigned to treatment 3 (rust mite-challenged) were inoculated with 30–70 rust mites. We used a paintbrush to brush rust mites from an infested leaf onto one leaf (midcanopy) of the experimental seedlings. Seedlings assigned to treatment 4 (powdery mildew-challenged) were inoculated with powdery mildew

spores; mildew-infested leaves were brushed onto the undersurface of a leaf (midcanopy) using a paintbrush. All organisms used for inoculations were obtained from papaya leaves freshly collected from the field. For seedlings assigned to treatment 5 (carborundum-challenged), one leaf (midcanopy) was gently abraded with carborundum using a cotton swab. All seedlings were maintained in growth chambers at 21°C, L16:D8. On days 10 and 14, all the seedlings were dipped in a solution of the acaricide abamectin (Agri-Mek 0.15 EC, 5 oz/100 gal; Syngenta, Wilmington, DE) and the fungicide myclobutanil (Rally 40W, 2 oz/100 gal; Dow AgroSciences, Indianapolis, IN) to which we added a spreader-sticker (Latron B-1956, 8oz/100 gal, active ingredient is phthalic/glycerol alkyl; Rohm and Haas, Philadelphia, PA). These chemical treatments succeeded in completely eliminating the parasites that we used to damage the plants initially (V. F., personal observation). On day 16, seedlings were transplanted into the field. The experimental plot was surrounded by older papaya fields to facilitate colonization.

The transplanted papaya seedlings were sampled nondestructively once a month for 3 successive mo to estimate densities of spider mites, rust mites, and powdery mildew. For each plant, three leaves located in the midcrown canopy were examined (different leaves each sampling date). For each of these three leaves, we counted the number of adult spider mites and the number of discrete mildew colonies. To quantify rust mite density, we used a 2.54 by 2.54 cm-rid mounted to a hand-lens (4× collapsible magnifier; Bioquip Products, Gardena, CA) as a sampling unit within which we counted all motile stages. We randomly selected two samples on the upper leaf surface and two on the lower leaf surface (4 samples/leaf × 3 leaves/plant = 12 samples/tree, average generated 1 datum/plant).

Season 2. The design and methodology for the second experiment were as described above for season 1 with the following modifications. The study ran from 15 April to 20 August 2002. Treatments were replicated 28 times. Replicates were established in two batches temporally distributed (batch 1: 16 replicates, ran from 15 April to 19 August 2002; batch 2: 12 replicates, ran from 29 April to 20 August 2002). For all batches, we regrouped seedlings (groups of five seedlings) of similar height together and randomly assigned the five treatments within each block, for an overall total of 28 blocks (used for statistical purpose). Densities of spider mites, rust mites, and powdery mildew were monitored every other week (six sampling dates for batch 1 and five sampling dates for batch 2).

Statistical Analysis. For the bioassay, the treatment effect on spider mite densities (each individual developmental stage and all stages pooled) was analyzed using paired *t*-tests (JMP 2000; SAS Institute, Cary, NC). For the field experiments, we calculated spider mite-days, rust mite-days, and mildew colony-days using the formula: $(X_{i+1} - X_i)(Y_i + Y_{i+1})/2$, where X_i and X_{i+1} are consecutive sampling dates, and Y_i and Y_{i+1} are the corresponding estimates of parasite density (Ruppel 1983). We summed these measures across the duration of each experiment to estimate the cumulative population size for each plant parasite. Cumulative spider mite-days, rust mite-days, and mildew colony-days were analyzed without transformation using two-way analysis of variance (ANOVA) with treatments and blocks as effects (potential effects of batches were therefore included in block effects). ANOVA was followed by pairwise contrasts whenever a significant effect was detected. In addition to analyzing each season separately, we also combined the data for both years in a single ANOVA, with year as an additional factor. When treatment effects were not significant, we ran a retrospective power analysis to calculate the least significant number (LSN, i.e., the number of observations needed to achieve a significant result at $\alpha = 0.05$) and the power of the test (Cohen 1998, SAS Institute 2000).

Results

Bioassay on Chemicals. We found no evidence that the pesticide solution that we used to remove parasites for the field experiments was capable of inducing resistance or susceptibility in the papaya host plant. Spider mite density on the newly grown leaf of the seedlings treated with pesticides was similar to that observed on seedlings treated with water (paired *t*-tests, df = 18; adults [means ± SE], water: 0.6 ± 0.16; pesticides: 0.6 ± 0.22, $t = 0.0$, $P = 1.0$; immatures, water: 4.1 ± 1.6, pesticides: 7.8 ± 1.8, $t = 1.53$, $P = 0.14$; eggs, water: 3.0 ± 1.0, pesticides: 2.8 ± 0.9, $t = -0.14$; $P = 0.89$; all instars combined, water: 7.7 ± 2.3; pesticides: 11.2 ± 1.5, $t = 1.3$, $P = 0.21$).

Field Experiments. The results obtained in the two field experiments were concordant: we detected no evidence of induced resistance in response to any of our challenging parasites and only weak and inconsistent evidence for induced susceptibility (Fig. 1; Table 1). In season 1, we observed no significant treatments effects (Table 1), but a weak trend toward induction of susceptibility to spider mites after challenge with spider mites and rust mites (Fig. 1A). In Season 2, we observed the same pattern of induced susceptibility to spider mites, this time with marginal lack of statistical support ($P = 0.08$, Table 1). In season 2, we also found that seedlings exposed to powdery mildew infection exhibited significantly higher densities of rust mites than the control plants (pairwise contrast, control versus mildew-challenged, $F_{1,95} = 7.18$, $P = 0.009$; Fig. 1E), another instance of induced susceptibility; this treatment effect was expressed most strongly at the end of our sampling period, ≈90 d after the seedlings were transplanted in the field (Fig. 2). Moreover, when seasons 1 and 2 were analyzed together, the same pattern was observed: plants challenged with powdery mildew harbored more rust mites than the control plants (Table 1; pairwise contrast, control versus mildew-challenged, $F_{1,317} = 9.19$, $P = 0.002$).

For season 1, the power analysis revealed that 11, 115, and 145 more replicates would have been re-

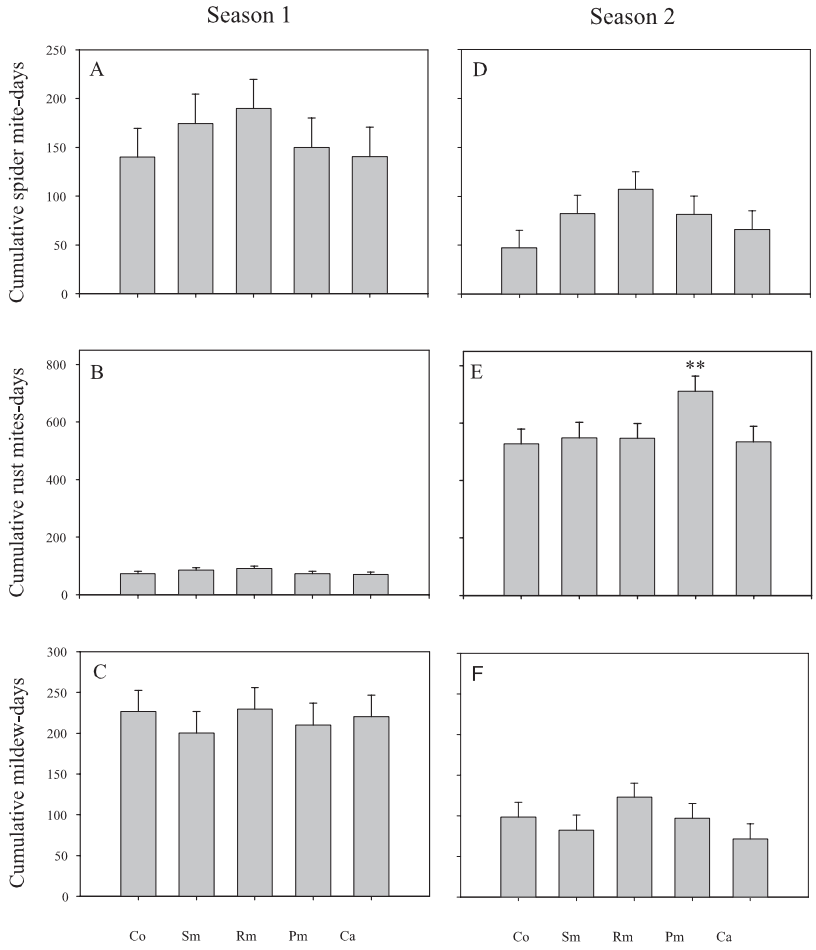

Fig. 1. Influence of early challenge of papaya plants on the cumulative densities (cumulative parasite days ± SE) of (A and D) spider mite (*T. cinnabarinus*), (B and E) papaya rust mite (*C. flagelliseta*), and (C and F) powdery mildew (*O. caricae*) across 29 August 2001–10 January 2002 (season 1) and 6 May–20 August 2002 (season 2). Treatments included (1) control (no challenge, Co); (2) spider mite-challenged (Sm); (3) rust mite-challenged (Rm); (4) powdery mildew-challenged (Pm); and (5) carborundum-challenged (Ca). $^{**}P < 0.01$, pairwise contrast between that treatment and the control.

quired to achieve significance ($\alpha = 0.05$) for spider mite, rust mite, and powdery mildew densities, respectively. For season 2, the power analysis indicated that 4 and 20 more replicates would be necessary to reach significance ($\alpha = 0.05$) for spider mite and powdery mildew densities, respectively. The power calculated for all tests was below the level of 0.8, which is often considered adequate (Cohen 1998) (season 1, spider mite: 0.2, rust mite: 0.5, powdery mildew: 0.2; season 2, spider mite: 0.7, rust mite: 0.6, powdery mildew: 0.4). The need for larger sample size and the low power suggest that our field studies were characterized by high variation.

Discussion

We examined intra- and interspecific interactions among two herbivorous mites and one plant pathogen through induced responses in their shared host plant, papaya. We found no evidence for induced resistance. Conversely, our results suggest that papaya expresses a weak form of induced susceptibility after injury from rust mites and powdery mildew. Plants exposed as young seedlings to rust mites subsequently supported larger population of spider mites, and plants exposed early to powdery mildew subsequently supported larger populations of rust mites.

Induced plant susceptibility after attacks by herbivores has been reported in many systems (Karban and Baldwin 1997). For instance, some perennial, woody host plants harbor increased densities of folivores in the year after a defoliation event (Williams and Myers 1984, Roland and Myers 1987, Kaitaniemi et al. 1997, Karban and Kittelson 1999). In herbaceous host plants, early-season defoliation can also improve the late-season performance of herbivores (Pullin 1987,

Table 1. Results of two-way ANOVA tests performed on the cumulative densities of spider mites, rust mites, and powdery mildew for season 1, season 2, and seasons 1 and 2 combined

Source	df	*F*	*P*
Season 1			
Cumulative spider mite-days			
Treatment	4	0.62	0.65
Block	41	1.80	<0.01
Error	155		
Cumulative rust mite-days			
Treatment	4	1.81	0.13
Block	41	3.81	<0.01
Error	155		
Cumulative powdery mildew-days			
Treatment	4	0.50	0.73
Block	41	4.97	<0.01
Error	155		
Season 2			
Cumulative spider mite-days			
Treatment	4	2.12	0.08
Block	26	2.43	<0.01
Error	95		
Cumulative rust mite-days			
Treatment	4	2.65	0.03
Block	26	1.84	<0.01
Error	95		
Cumulative powdery mildew-days			
Treatment	4	1.33	0.26
Block	26	2.35	<0.01
Error	95		
Seasons 1 and 2 combined			
Cumulative spider mite-days			
Treatment	4	1.16	0.33
Year	1	20.41	<0.0001
Year × treatment	4	0.07	0.99
Error	317		
Cumulative rust mite-days			
Treatment	4	3.03	0.02
Year	1	658.30	<0.01
Year × treatment	4	3.38	0.01
Error	317		
Cumulative powdery mildew-days			
Treatment	4	0.60	0.66
Year	1	57.07	<0.0001
Year × treatment	4	0.20	0.94
Error	317		

Messina et al. 1993). Induced susceptibility has also been shown in response to herbivory by mites. Spider mites were more strongly attracted to previously challenged plants than unchallenged plants (Kielkiewicz 1988), and spider mite fecundity and survivorship can be greater on previously damaged plants than on undamaged plants (English-Loeb and Karban 1991). Westphal et al. (1992) also found that feeding injury by the eriophyid mite *Acalus cladophthirus* on *Solanum dulcamara* L. triggered higher fecundity in the spider mite *T. urticae.* Many of these examples of induced susceptibility may reflect an increase in the nutritional quality of the host plant after damage (Rodriguez and Rodriguez 1987, Karban and English-Loeb 1988). For example, it has been shown that the increased susceptibility of woody host plants after defoliation may be explained by the higher water and nitrogen contents of leaves produced during regrowth (Pullin 1987, Messina et al. 1993). In our study, we did not examine the water and nutrient content of papaya seedlings.

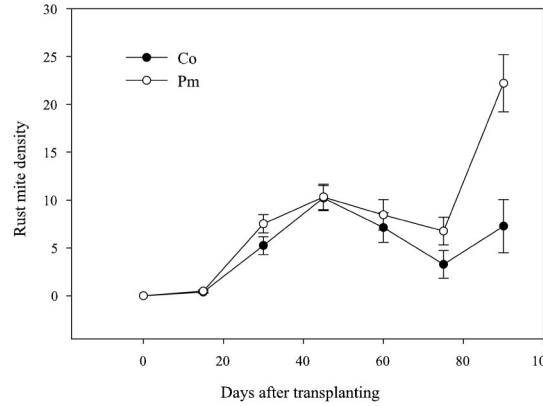

Fig. 2. Density of papaya rust mite (*C. flagelliseta*; (no. of motile instars/subsampling unit ± SE) over time (season 2, May–August 2002) on plants initially unchallenged (control, Co) and plants challenged with powdery mildew (Pm).

Increased plant susceptibility to phytophagous arthropods after attack by a pathogen has been documented less frequently than induced susceptibility in systems involving herbivores only. In season 2 of our study, we found that papaya rust mites exhibited higher population growth on plants that had been challenged previously with powdery mildew than on control plants (Table 1; Figs. 1E and 2). Recent research supports the hypothesis that inhibitory crosstalk between the jasmonate and salicylate pathways may influence herbivore–phytopathogen interactions (Thaler et al. 2002). For instance, Thaler et al. (2002) showed that the growth rate of the cabbage looper, *Trichoplusia ni*, increased on tomato plants treated with benzothiadiazole, an elicitor of the salicylate pathways that are naturally induced by pathogen attack, compared with untreated plants. Antagonism between the jasmonate and the salicylate pathways in papaya nevertheless seems unlikely to explain the induction of susceptibility to rust mites by powdery mildew because we found no evidence for induced resistance playing a role in the plant's response to either the mites or mildew.

In another study, we investigated the interaction of powdery mildew and rust mites when they co-occurred on papaya leaves, as opposed to this study, in which the two plant parasites were initially segregated in time. Rust mites were found to have a moderately negative effect on the mildew's population density, with no reverse effect of the mildew on the mites (asymmetrical competition or amensalism; unpublished data). The mechanisms responsible for this negative interaction are unclear. However, the results obtained in this study argue against the involvement of induced plant resistance and suggest that simple exploitative competition should be explored as a causal basis for the observed negative effect of mites on mildew populations.

Although we chose to perform our experiments under natural field conditions, our decision to rely on natural populations of colonizing mites and mildew to

detect the induction of resistance or susceptibility in papaya does have some drawbacks. Mite and mildew population densities were relatively light in both seasons. It is therefore possible that a transient period of induced resistance might have occurred in our system but was not detected by our sampling. It has been shown that induced resistance can decay quickly and be followed by a period of enhanced susceptibility to herbivory. For example, Underwood (1998) showed that induced resistance to beetle damage in soybean had totally disappeared 20 d after the induction, with the host plant exhibiting induced susceptibility thereafter. Nevertheless, we believe that our monthly sampling was appropriate. If induced resistance peaked in expression during the first month the plants were in the field and then gradually waned, we would still expect that the population densities present at the t = 1 mo sample should have revealed the earlier expression of resistance. That is, if during the first month, a putative resistance trait was slowing the colonization or growth of papaya's parasite populations, even if the resistance itself were entirely gone by 1 mo, we would still expect smaller parasite populations to persist on those plants for some time. In contrast, our samples at time = 1 mo revealed no evidence for plant resistance (time = 1 mo, season 1, $P > 0.05$; season 2, $P > 0.05$; data not shown). Moreover, based on other studies on perennial plants, it is reasonable to expect that induced responses would affect herbivore populations many months after the induction (Tables 4.1 and 4.2 of Karban and Baldwin 1997).

In this study, using an experimental protocol that was intended to reflect as much as possible natural events occurring in the field during colonization of young papaya seedlings by a group of plant parasites, we found evidence for a form of induced susceptibility.

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