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MULTIPLE PLANT EXPLOITERS ON A SHARED HOST: TESTING FOR NONADDITIVE EFFECTS ON PLANT PERFORMANCE

Valérie Fournier,[1,2,3,4] Jay A. Rosenheim,[1] Jacques Brodeur,[3,5] Joselito M. Diez,[2] and Marshall W. Johnson[2,6]

[1]*Department of Entomology, University of California, Davis, California 95616 USA*
[2]*Department of Plant and Environmental Sciences, University of Hawaii at Manoa, Honolulu, Hawaii 96822 USA*
[3]*Département de Phytologie, Université Laval, Ste-Foy, Québec G1K 7P4 Canada*

Abstract. The combined impact of multiple plant parasites on plant performance can either be additive (the total damage equals the sum of the individual effects) or nonadditive (synergistic or antagonistic damage). Two statistical models are available for testing the independent (=additive) effects of two factors. Here we suggest that the natural history of the plant–parasite system should motivate the choice of a statistical model to test for additivity. Using in-field, manipulative experiments, we examined the interactions between the herbivorous mite *Calacarus flagelliseta* Fletchmann, De Moraes and Barbosa (Acari: Eriophyidae), the fungal pathogen *Oidium caricae* F. Noack (a powdery mildew), and their host plant *Carica papaya* L. in Hawaii. First, we found that herbivorous mites had a moderate negative effect on powdery mildew: when mites were absent, powdery mildew colonies were larger and more numerous. Second, we showed that each plant parasite, when evaluated alone, significantly reduced several measures of plant performance. Third, we found that the combined impact of mites and mildew on plant performance is mostly additive and, for a few variables, less than additive. Finally, we explored compensatory responses and found no evidence for nonlinearities in the relationship between plant performance and cumulative parasite impact. Plants are almost universally subject to attack by multiple herbivores and pathogens; thus a deeper understanding of how multiple plant parasites shape each other's population dynamics and plant performance is essential to understanding plant–parasite interactions.

Key words: additive effects; additivity; Calacarus flagelliseta; Carica papaya; *exploitative competition; herbivory; multispecies interactions;* Oidium caricae; *plant compensation; plant pathogen–herbivore interactions; plant performance.*

Introduction

Plants have multiple enemies, and these enemies can be taxonomically very diverse. Vertebrate, arthropod, mollusc, and nematode herbivores, as well as flagellate protozoan, fungal, bacterial, and viral pathogens can all exploit terrestrial plants (Agrios 1988). While the effects of non-disease-vectoring herbivores and phytopathogens on host plants have long been studied in isolation, the last decade has seen a growing interest in plant–herbivore–pathogen interactions (reviewed in Barbosa 1991, de Nooij et al. 1992, Hatcher 1995, Rostás et al. 2003), yielding major advances in our understanding of the ecology of these interactions (e.g., Hatcher et al. 1994*a*, *b*, *c*, 1995, Friedli and Bacher 2001, Kluth et al. 2001, Ahlholm et al. 2002, Kruess 2002) as well as their physiological bases (e.g., Maleck and Dietrich 1999, Bostock et al. 2001, Hatcher et al. 2004, Thaler and Bostock 2004).

Interactions between a herbivore, a plant pathogen, and their shared host may be examined at two levels of response: (1) the population dynamics of the herbivore and pathogen and (2) their combined effect on plant performance. At the first level, the ecological outcome of herbivore–phytopathogen interactions varies from no effect (Ahlholm et al. 2002) to facultative mutualism (e.g., Friedli and Bacher 2001, Kluth et al. 2002, Johnson et al. 2003, Mondy and Corio-Costet 2004) to different forms of competition (e.g., Karban et al. 1987, Hatcher et al. 1994*a*, *b*, *c*, 1995, 1997, Lappalainen et al. 1995, Hatcher and Ayres 1997, Moran 1998, Tinney et al. 1998, Hatcher and Paul 2000, Kruess 2002, Rostás and Hilker 2002, Simon and Hilker 2003).

Interspecific interactions between phytopathogens and herbivores may also shape their joint impact on host performance, yielding additive or nonadditive damage (Hatcher 1995). Understanding the combined effect of pathogens and herbivores on plant performance requires us to think about two transitions. First, interactions between plant parasites may change their

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[4] Present address: Department of Entomology, Rutgers University, New Brunswick, New Jersey 08901 USA.
E-mail: valfourn@rci.rutgers.edu

[5] Present address: Département de Sciences biologiques, Université de Montréal, Montréal, Québec H1X 2B2 Canada.

[6] Present address: Department of Entomology, University of California, Riverside, California 92521 USA.

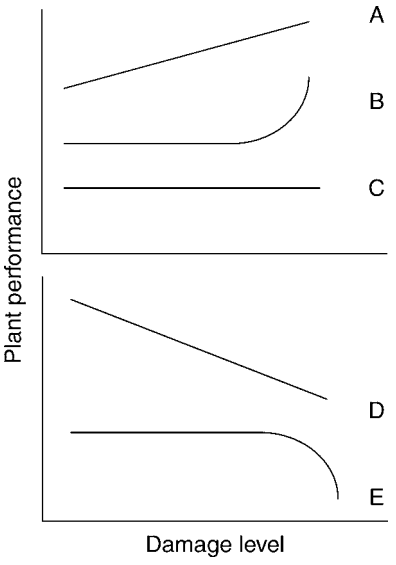

FIG. 1. The compensatory response exhibited by plants is graphically described as the relationship between plant performance (fitness) and damage level. Here we illustrate some of the basic shapes that compensatory responses can display. Functions A and B depict overcompensation (slope > 0), in which damaged individuals perform better than undamaged ones. Overcompensation can either be a linear (A) or a nonlinear function (e.g., B) of damage. Function C represents full compensation (slope $= 0$). Finally, functions D and E show groups of plants that do not fully compensate for herbivore and/or pathogen damage (=undercompensation; slope < 0). Function E exemplifies a case of "compensation breakdown" (a nonlinear relation). The figure is adapted from Strauss and Agrawal (1999) and Stowe et al. (2000).

population sizes and therefore change the total quantity of plant resources that is consumed. Second, the relationship between consumption and plant performance is shaped by compensatory responses (reviewed in Trumble et al. 1993, Stowe et al. 2000; see Fig. 1). In the simplest case, consumption of plant resources by parasites translates linearly into depressed plant performance. Thus, if two parasites do not influence one another's population dynamics, their combined consumption of plant resources is expected to be the sum of their individual consumption and plant performance is expected to decrease as the sum of their individual negative effects (additive damage). In contrast, if two plant exploiters interact in a mutualistic fashion, their combined exploitation of plant resources is expected to be greater than the sum of their individual effects and plant fitness is expected to decline by more than the sum of their individual negative effects (synergistic damage). Conversely, if two plant parasites interact in a competitive manner, their combined exploitation is expected to be less than the sum of the individual effects and plant performance is expected to decline by less than the sum of the individual effects (antagonistic or inhibitive damage).

If, however, the plant's compensation response is nonlinear or the plants exhibit overcompensation to one parasite (refer to Fig. 1), then the combined effect of two parasites on plant performance may not be a direct reflection of how the two parasites shape one another's population sizes and consumption of the plant. Plants may show little or no loss of performance in response to low or moderate levels of herbivory, but eventually plant performance may deteriorate dramatically when the intensity of plant damage passes a threshold and overwhelms a plant's mechanisms of compensation (e.g., Maschinski and Whitham 1989, Fornoni et al. 2004; function E in Fig. 1). In this case of "compensation breakdown," even if two plant exploiters do not affect one another's abundance and their combined consumption of plant resources is as expected under a model of additive effects, their joint impact on plant performance may still be more than additive. Similarly, compensation breakdown suggests that two plant parasites that interact competitively may still produce additive or synergistic impacts on plant fitness, instead of the antagonistic outcome expected under no compensation. Finally, compensation breakdown may further amplify the synergistic effects of two mutualistic parasites on plant performance. The assessment of plant compensation therefore appears to be crucial to the interpretation of additive and nonadditive effects on plant performance.

Also critical to the evaluation of joint impact of multiple attackers on plant fitness is the choice of a statistical model (i.e., multiplicative vs. additive risk models). While ecologists have carefully discussed the question of model selection in the context of higher-order interactions (Billick and Case 1994), predator–prey interactions (Sih et al. 1998), and interactions between plant competition and herbivory (Rees and Brown 1992, Hambäck and Beckerman 2003), major reviews on plant–herbivore–pathogen interactions (Barbosa 1991, Hatcher 1995, Hatcher and Ayres 1997, Rostás et al. 2003), above- and belowground herbivory (Blossey and Hunt-Joshi 2003), and plant–animal interactions (Strauss and Irwin 2004) have remained silent on this issue. Here we reemphasize Wootton's idea (1994) that the natural history and behavior of the species involved should motivate the choice of a statistical model when testing for interactions in factorial experiments. This theme is further developed in the section titled *Statistical analyses*.

To our knowledge, no studies of plant–pathogen–herbivore interactions have simultaneously examined (1) how each plant parasite's population density is affected by the other, (2) whether the host plant compensates for parasite damage, and (3) the final combined impact on plant performance. Such holistic, ecological studies are important for at least three reasons. First, plant resistance traits, including both constitutive and inducible resistance, as well as plant compensatory abilities, have evolved in an environment in which concurrent

herbivory and pathogen infection are likely to have been the rule, and thus our understanding of the function and evolution of these traits is likely to be enhanced by considering the joint effects of multiple plant parasites (Strauss and Agrawal 1999, Strauss and Irwin 2004). Second, studying the effects of pathogens and herbivores on a shared host plant can improve our understanding of the manner in which plant populations are regulated. For instance, when examined separately, both pathogens (e.g., van der Putten and Peters 1997, Gilbert 2002) and herbivores (e.g., Crawley 1989, Carson and Root 2000, Gurevitch et al. 2000, van Ruijven et al. 2005) can play important roles in shaping competition and population dynamics in plant communities. Thus, understanding the joint effect of herbivores and pathogens may be critical to better appreciating the control of plant diversity. Finally, applied ecologists interested in weed management and biological control must also understand the joint impacts of herbivores and pathogens in order to design weed suppression programs (e.g., Johnson et al. 1986, Keller et al. 1986, Kok et al. 1996, Kruess 2002).

The present paper reports the results of manipulative field experiments that probe the interactions between a fungal pathogen and a herbivorous mite that attack the foliage of their host plant. First, we quantified the influence of each parasite on the other's abundance. Second, we assessed the individual and joint effects of the two parasites on plant performance. Third, we examined plant compensation to understand the mechanisms by which the cumulative parasite populations translated into the observed plant performance values.

In contrast to most field studies on plant–phytopathogen–herbivore interactions, in which the host plants are artificially inoculated, we used natural populations of the parasites. While each of these two methods has strengths and weaknesses and enables different aspects of the interactions to be examined, we believe that using naturally occurring densities adds realism to our study.

Materials and Methods

Study system

Papaya (*Carica papaya* L., Caricaceae) is a short-lived perennial, native to Central America (Storey 1976). It is a fast-growing, herbaceous plant that starts producing fruit within the first year after planting and fruits all year long. Trees in commercial orchards typically consist of a single erect stem up to 4 m tall with a terminal crown of ~30–60 large, palmately lobed leaves. New leaves are produced year-round, emerging from the growing point at the tip of the trunk. The life span of a papaya leaf can reach six months, and trees in commercial plantings typically live for three years.

Foliage of papaya grown in Hawaii, USA, is attacked by numerous species of herbivorous mites (Yee et al. 1970). The papaya rust mite, *Calacarus flagelliseta* Fletchmann, De Moraes and Barbosa (Acari: Eriophyidae), has become a predominant pest of papaya since it was first reported in Hawaii in the early 1990s (Hamasaki and Heu 1991, Fournier et al. 2004*a*). At the time of its original detection in Hawaii, it was mistakenly identified as *C. brionesae* (J. Amrine, *personal communication*). *Calacarus* adults are 220–246 μm long, fusiform, and grayish-brown with white longitudinal wax bands (Fletchmann et al. 2001). *Calacarus flagelliseta* is believed to be host-specific (J. Amrine, *personal communication*) and infests both surfaces of papaya leaves as well as fruit (V. Fournier, *personal observation*). The papaya rust mite 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). In Hawaii, the papaya rust mite occurs year-round, reaching densities of up to 290 000 individuals on a single leaf when populations peak in the late summer (V. Fournier, *unpublished data*). Eriophyoid mites are passively dispersed by aerial currents (Sabelis and Bruin 1996). The basic biology of *Calacarus* is poorly known, and so far no natural enemies have been found in Hawaii (V. Fournier, *personal observation*). The carmine spider mite, *Tetranychus cinnabarinus* Boisduval (Acari: Tetranychidae), is also commonly found on papaya foliage (Yee et al. 1970). This herbivorous mite attacks a broad range of host plants in Hawaii (Goff 1986). Various species of predacious arthropods are found on papaya foliage (see Fournier et al. 2003), but none of them seems to use *Calacarus* mites as a major source of food (V. Fournier, *personal observation*). However, it is possible that the spider mite specialists *Stethorus siphonulus* Kapur (Coleoptera: Coccinellidae), *Phytoseiulus macropilis* (Banks), and *Phytoseiulus persimilis* Athias-Henriot (Acari: Phytoseiidae) feed on *Calacarus* as an alternative prey. Unidentified species of tydeid mites are also present sporadically on papaya leaves, but so far there is no evidence that they prey upon *Calacarus* (V. Fournier, *personal observation*).

Powdery mildew is a common and economically significant disease of papaya (Yee et al. 1970). The causal agent, *Oidium caricae* F. Noack (Erysiphales), is an obligate ectoparasitic fungus, host-specific to papaya (Ooka 1994). It forms circular, white, and powdery-appearing colonies on the undersurface of the leaves. The mycelium and the chains of conidia constitute the visible fungal mass on the leaf surface. The fungus produces haustoria, which penetrate the epidermal cells of the host plant and act as feeding organs (Braun 1995). Conidia are dispersed by wind (Jarvis et al. 2002). *Oidium* occurs year-round in Hawaii, but infection intensity typically peaks during winter months. Disease development is enhanced by low light intensity, high humidity, moderate temperatures (18°–32°C), and moderate rainfall (Ooka 1994).

The rust mite and the powdery mildew share the same resource: both parasites feed exclusively on the first layer of epidermal cells. Stylets of eriophyid mites are typically 15–20 μm long and are inserted to 75% of

their full length in plant tissue (McCoy and Albrigo 1975; J. Amrine, *personal communication*), whereas haustoria generally penetrate to a depth of 12–16 μm (Braun 1995; R. Bélanger, *personal communication*). Moreover, papaya rust mite and powdery mildew probably share a long evolutionary history, as both parasites are highly specialized on *C. papaya*. However, in Hawaii, where papaya was introduced some 200 years ago (Yee et al. 1970), the co-occurrence of these parasites is relatively recent. The rust mite was first reported in the early 1990s (Hamasaki and Heu 1991), while the fungal disease has been present for many decades (Yee et al. 1970).

The soil-borne pathogen *Phytophthora palmivora* (Buttler), previously misidentified as *P. parasitica* Dastur, is commonly present in papaya-producing areas in Hawaii (Yee et al. 1970, Ko 1994). Root infection by this aggressive pathogen results in yellowing of the leaves, rapid defoliation, and eventual death of the tree.

Field experiments

Our research was carried out at the University of Hawaii Poamoho Experiment Station on Oahu, Hawaii. We repeated the same experiment twice (described below as year 1 and year 2).

Year 1.—From 3 May 2000 to 30 March 2001 we conducted a manipulative experiment with four treatments comprising a 2×2 factorial design: (1) clean control (−mites, −mildew); (2) presence of mites only (+mites, −mildew); (3) presence of mildew only (−mites, +mildew); and (4) presence of both mites and mildew (+mites, +mildew). The experimental unit was a single papaya tree (Solo variety, cultivar Sunrise). Treatments, each replicated 11 times, were randomly assigned to trees (44 trees distributed in 11 blocks). The experiment was set up in a young field (6 months old, no fruit borne). Powdery mildew was suppressed with the fungicide myclobutanil (Rally 40W, 0.15 g/L, Dow AgroSciences, Indianapolis, Indiana, USA), and mites were suppressed with abamectin (Agri-Mek 0.15 EC, 0.4 mL/L, Syngenta, Wilmington, Delaware, USA); the selectivity of these pesticides is addressed below (see *Bioassays*). To enhance the coverage and therefore the effectiveness of the pesticide applications, we added a spreader-sticker (Latron B-1956, 0.6 mL/L; active ingredient is modified phthalic/glycerol alkyl; Rohm and Haas, Philadelphia, Pennsylvania, USA) to the pesticide solution in treatments 1, 2, and 3; trees in treatment 4 (presence of both mites and mildew) were sprayed with the same concentration of the spreader-sticker in water. Chemicals were applied every other week using a backpack sprayer (Solo; Forestry Suppliers, Jackson, Mississippi, USA). Plants were sprayed until runoff (~500 mL/tree).

1. *Population dynamics*.—The densities of rust mites and powdery mildew were monitored on the following dates: 3 May, 22 May, 5 June, 19 June, 17 July, 1 August, 25 August, 26 October 2000, and 30 March 2001. Non-destructive field counts were performed. For each tree, four random leaves located in the mid-crown canopy were examined (different leaves each sampling date). We estimated the density of powdery mildew by counting the number of discrete colonies on one randomly selected half of each leaf (Nicot et al. 2002). At the end of the experiment, we measured the diameter of three randomly selected colonies on each of the four sampled leaves. To assess rust mite density, we developed a subsampling technique (Fournier et al. 2004*a*). Using a 2.5×2.5 cm grid mounted to a hand-lens (4× collapsible magnifier; Bioquip Products Gardena, California, USA) as a sampling unit, we randomly selected three samples on the upper leaf surface and three on the lower leaf surface (6 samples/leaf × 4 leaves/tree = 24 samples per tree). We counted all motile individuals within the 2.5×2.5 cm grid. However, when densities were high (≥100 individuals within the grid), we counted mites in only one randomly selected quarter of the grid and multiplied by four to estimate the population within the full grid. For each papaya leaf sampled, we also counted the total number of spider mites (adults only).

2. *Plant performance*.—The impacts of rust mites and powdery mildew on papaya performance were assessed for the following parameters: leaf longevity, number of leaves per tree, trunk circumference, tree height, and fruit yield. Leaf longevity was measured by tagging the newest unfurling leaf on each tree and monitoring its status on a regular basis until senescence. Five leaves per tree were tagged successively over a period of two months (leaf 1 was tagged on 8 May; leaf 5 was tagged on 3 July 2000) and checked until they died. At the end of the study, we counted the total number of leaves on each tree and measured the trunk circumference at 1 m above the ground. Tree height was measured at the beginning and at the end of the experiment. When the trees started bearing mature fruit (September 2000), we harvested papayas once per week at color break (when fruit color changes from green to yellow) and recorded their mass. At the end of the experiment (30 March 2001), all immature fruit were harvested and weighed. Finally, we recorded any tree mortality that occurred over the course of the experiment.

Year 2.—The design and methodology for the second experiment were as described above for year 1 with the following modifications. The study ran from 25 April 2001 to 26 August 2002. We set up 10 replicates of each of the four treatments in a 4-month-old planting (Solo variety, cultivar X77). Each treated tree was surrounded by four "buffer" trees (nonexperimental trees) to minimize the potential for pesticide drift among treatments. Due to difficulties we experienced during year 1 with suppressing powdery mildew, we sprayed the fungicide every week instead of every other week. Trees assigned to the +mites, +mildew treatment were therefore also sprayed every week with the spreader-sticker, while trees assigned to the −mites, +mildew treatment

were sprayed with the spreader-sticker alone or with the acaricide and the spreader-sticker on alternate weeks. Because of the lower risk of powdery mildew outbreaks in the spring and summer, from April to August 2002 we switched back to the spraying regimen instituted the first year (every other week for all chemicals).

1. *Population dynamics*.—Mite and mildew densities were monitored monthly (16 sampling dates between April 2001 and August 2002). Mildew colonies have a tendency to merge when the fungus reaches high densities during the winter. Therefore, we complemented colony counts with estimates of the percentage of the leaf area covered by mildew. We used a 2.5×2.5 cm grid on a transparency divided into 25 squares to estimate coverage. We randomly affixed the grid to five locations on the undersurface of each of the four sampled leaves (total of 20 samples per tree). To compare mildew densities in years 1 and 2 of the experiments, we needed to relate colony counts to percentage of leaf coverage. Applying linear regression to the total data set available from year 2 (439 observations made over 12 sample dates), we obtained: (percentage leaf coverage) = 0.12 (the number of colonies/half leaf) + 0.8 ($r^2 = 0.62$, $F_{1,437} = 703.8$, $P < 0.0001$). We did not measure the diameter of colonies at the end of the experiment.

2. *Plant performance*.—As in year 1, the impacts of rust mites and powdery mildew were assessed on leaf longevity, total number of leaves, trunk circumference, tree height, and fruit yield. To estimate leaf longevity, 10 leaves per tree were tagged over five months (18 June to 15 November 2001).

Bioassays

Under our experimental design it was critical that the acaricide used to manipulate rust mite density did not have any effects on powdery mildew in the absence of rust mites (i.e., no direct effects of the acaricide on powdery mildew). Likewise, it was critical that the fungicide used to manipulate the powdery mildew did not have any effects on rust mites in the absence of mildew (i.e., no direct effects of the fungicide on rust mites). To determine whether these conditions were met, we performed controlled bioassays.

Test for direct effects of the acaricide on the fungus.—We performed a bioassay of the impact of abamectin on powdery mildew in the greenhouse, where we could exclude rust mites. Papaya seedlings (Solo variety, cultivar Sunrise) were inoculated with mildew spores. Using a paint brush, we "swept" spores from leaves freshly collected from the field onto the lower leaf surface of two leaves per seedling. To prevent any contamination of the seedlings with rust mites, leaves were taken from trees previously sprayed with abamectin. All inoculated seedlings were placed in a growth chamber for two weeks at 20°C and photoperiod 16 h: 8 h (light:dark) to allow fungal spores to germinate and initiate colonies. On 13 August 2002, we randomly assigned one of two treatments, each replicated 20 times, to each experimental unit (a seedling); the treatments were (1) control (seedlings sprayed with the spreader-sticker) and (2) acaricide (seedlings sprayed with abamectin and spreader-sticker). The bioassay ran for 16 d. Chemicals were sprayed on days 0, 7, and 14, using the same doses as were used in the field experiment run in year 1. Nondestructive sampling was done on days 0, 6, 9, and 16. For each seedling, we estimated the percentage of leaf area covered with powdery mildew by affixing one 2.5×2.5 cm transparent grid onto each of the infested leaves, as described in *Year 1: 1. Population dynamics*.

Test for direct effects of the fungicide on the mite.—We performed a field experiment to determine whether the fungicide myclobutanil affected the population dynamics of the rust mite in the absence of powdery mildew. To exclude the potential effect of the fungus on rust mites, we selected a very young papaya planting (Solo variety, cultivar Sun-Up) in which powdery mildew was naturally absent. On 26 October 2002 we set up 10 replicates of two treatments: (1) control (leaves sprayed with the spreader-sticker only); and (2) fungicide (leaves sprayed with myclobutanil and spreader-sticker). The doses applied were the same as used in the main experiments. The experimental unit was a single leaf; one replicate of each treatment was established on each of 10 trees, which were used as blocks. The bioassay ran 20 d. Chemicals were sprayed on days 0 and 13, and in-field sampling of mite populations was done on days 0, 13 (prior to pesticide applications), and 20. Rust mite densities were estimated by counting motile instars within five subsampling units on the lower surface of the leaves, as described in *Year 1: 1. Population dynamics*.

Statistical analyses

Population dynamics.—For each year and each parasite, we performed repeated-measures MANOVA on densities, with main effects for mites, mildew, and the mites × mildew interaction. For each year, we also calculated cumulative mite-days and mildew-days (colony-days for year 1 and coverage-days for year 2) across the duration of the experiments by summing for all dates $(X_{i+1} - X_i)(Y_i + Y_{i+1})/2$, 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 analyzed cumulative mite-days and cumulative mildew-days (data untransformed) using two-way ANOVA, with main effects for mites, mildew, and mites × mildew. Pairwise contrasts were performed on cumulative densities to determine the effect of the fungus on the population dynamics of the mite (+mites, +mildew vs. +mites, −mildew) and the effect of the mite on the population dynamics of the fungus (+mites, +mildew vs. −mites, +mildew) (JMP; SAS Institute 2000). Block effects were not significant, therefore we did not include them in the final analyses.

Plant performance.—The individual and combined impacts of rust mites and powdery mildew on leaf

longevity, total number of leaves, trunk circumference, tree height, and fruit yield were analyzed using two-way ANOVA, with main effects for mites, mildew, and a mites × mildew interaction. Block terms were only retained when their effects were significant. ANOVA was followed by pairwise contrasts whenever a significant effect was detected. The following comparisons were made: (1) clean control vs. presence of mildew only, to determine the effect of the fungus alone; (2) clean control vs. presence of mites only, to determine the effect of mites alone; (3) clean control vs. presence of both mites and mildew, to determine the combined impact of both parasites; (4) presence of both mites and mildew vs. pooled data from presence of mildew only and presence of mites only, to determine whether the combined impact of both parasites differed from the mean impact of a single parasite. Corrections for multiple comparisons were done with the sequential Bonferroni test ($\alpha = 0.05$, $k = 4$) (Rice 1989). Additionally, for each year we simultaneously analyzed all the plant performance variables using MANOVA (identity response design) with main effects for mites, mildew, and the mites × mildew interaction. To examine treatment effects on tree survival, we used (1) Fisher's exact test and (2) logistic regression with main effects for mites (mean density), mildew (mean density), and the mites × mildew interaction. For the analyses of tree death, we excluded five trees that died at the end of July 2000. These early-dying trees were the first five trees at the end of one row of our experimental plot (all four trees from the same block, plus the first tree in the next block), and they died within the same week from root infection by *Phytophthora palmivora*. A week after their death, the fungicide mefenoxam (Ridomil Gold EC, Syngenta) was applied as a soil drench to suppress *P. palmivora*. The remaining trees that died during year 1 were not adjacent to these early-dying trees, were scattered across the plot, and started dying after September 2000.

Analysis of the interaction of mites × mildew on plant performance.—Two mathematical models can be used to test for independent effects of two factors: the additive risk model and the multiplicative risk model (Sih et al. 1998). Both models test the same null hypothesis: parasite A and parasite B act independently. In the additive risk model, the predicted proportional decline in plant performance in the face of combined parasites, p_{ab}, is calculated as: $p_{ab} = p_a + p_b$, where p_a is the proportional loss generated by parasite A, and p_b the proportional loss generated by parasite B. For example, under this model if parasite A alone consumes 50% of the foliage and parasite B alone consumes 50% of the foliage, the prediction for their combined action would be 100% of the foliage consumed. The additive risk model thus must be used with the additional stipulation that the maximum possible value for $p_{ab} = 1.0$. In the multiplicative risk model, the expected proportional decline in plant performance in the face of combined parasites is $p_{ab} = p_a + p_b - p_a p_b$. Thus, if parasite A alone consumes 50% of the foliage, and parasite B alone consumes 50% of the foliage, the multiplicative risk model predicts that 75% of the foliage will be consumed when parasites act together (i.e., each unit of foliage has a probability of 50% of escaping attack by each parasite; thus, if the parasites act independently, the probability of escaping attack by both parasites is $[0.5] \times [0.5] = 0.25$). The multiplicative risk model offers the clear advantage of avoiding predictions that plant performance can be suppressed by >100% (for instance, if each parasite taken alone depletes 60% of the plant material, their combined impacts cannot deplete available plant material by 120%). When individual effects of parasites on plant performance variables are minor (i.e., both p_a and p_b are small), the two models generate similar predictions. The multiplicative risk model is implemented with a two-way ANOVA on log-transformed data, whereas the additive risk model is implemented with a two-way ANOVA performed on untransformed data. In each case, nonadditive, or nonindependent, effects of plant parasites are identified by a significant interaction term (Underwood 1997).

We suggest that the natural history and behavior of the plant–parasite system should motivate the choice of a statistical model to test for nonadditive interactions. In the present study, we are examining one mobile parasite with flexible foraging (the rust mite) and another parasite with very limited mobility following its landing on its host plant (the mildew). As an obligate parasite, powdery mildew will not grow on cells that are damaged or dead (R. Bélanger, *personal communication*). If a spore lands at a leaf location where the epidermal cells have been exploited by mites, the haustoria will fail to develop. On the other hand, if a mite lands on a portion of the leaf where the cells have been killed by powdery mildew, it has the ability to walk to a less exploited area, unless all or nearly all of the leaf has been infested with the fungus. Thus, from the fungus' perspective, the multiplicative risk model may be more applicable, because the spores cannot move after landing on the leaf surface, and thus their impact on the plant is proportionally reduced by the prior action of the rust mites. However, from the mite's perspective, the additive risk model may be a more appropriate choice, because their ability to exploit the leaf may be minimally influenced by the prior action of the mildew, at least until the leaf approaches total exploitation. Thus, for this system, the ideal model may be one that is intermediate between the predictions of the multiplicative and the additive risk models. As an example, in the hypothetical case of each plant parasite consuming 50% of the foliage when alone, this hybrid model would predict 87.5% of the foliage consumed when the parasites co-occur.

Seasonal synchrony between plant parasites may also influence the choice of a statistical model to test for nonadditive interactions. If parasites are temporally segregated, the extent to which each parasite interferes

with the ability of the other to exploit the host may be reduced. Such a scenario would favor the choice of the additive model over the multiplicative model. In the present system, the rust mite and the mildew co-occur year round, but their seasonal prevalence differs: the mite population reaches high densities during the spring, summer, and fall, while the mildew population peaks during the winter.

In the absence of a clear argument for the superiority of one model, we decided to test for interactions using both models. The two models should define the upper limit (multiplicative risk model) and lower limit (additive risk model) for the range of plant performance values expected under the hypothesis of independent action of mites and mildew. We tested whether the ANOVA assumption of homogeneity of variance was met for both untransformed and log-transformed plant performance data. We found that all data exhibited homoscedasticity (data not shown). Untransformed and log-transformed data were also examined for normality using the Shapiro-Wilk test. The assumption of normality was met for all log-transformed data (data not shown). For untransformed data, we found that the following data sets were normally distributed: year 1, trunk circumference, and yield; year 2, leaf longevity, trunk circumference, and yield.

Plant compensation.—We examined the functional relationship between the intensity of plant exploitation by each parasite (cumulative mite-days when mildew is suppressed, cumulative mildew-days when mites are suppressed) and select plant performance variables (leaf longevity, number of leaves, trunk circumference, and fruit yield) using multiple regression analyses. A negative linear relationship between plant performance and cumulative parasite population density was our expectation under the simplest model of plant compensation. To test for deviations from linearity, and in particular a concave-down plant response curve that is expected under plant compensation that breaks down with increasingly intense plant exploitation, we included both linear terms (e.g., cumulative mite-days) and quadratic terms (e.g., [cumulative mite-days]^2) in the multiple regression analyses. To explore plant response to pressure from both parasites, we performed a multiple regression for each plant performance variable using cumulative mite-days and cumulative mildew-days as the independent variables (we used the full data set in contrast to the truncated data set we used to estimate plant compensation for each individual parasite). To illustrate the plant's response to combined pressure from mites and mildew in a way that was comparable to the single-parasite plant compensation graphs, we plotted observed plant performance (y-axis) against predicted loss in performance (x-axis), where the predicted loss in performance was obtained from the coefficients estimated from regressing plant performance on cumulative mite-days and cumulative mildew-days. Because we did not establish a series of experimental treatments with different cumulative parasite-day levels, our results are partially correlative and thus should be interpreted cautiously.

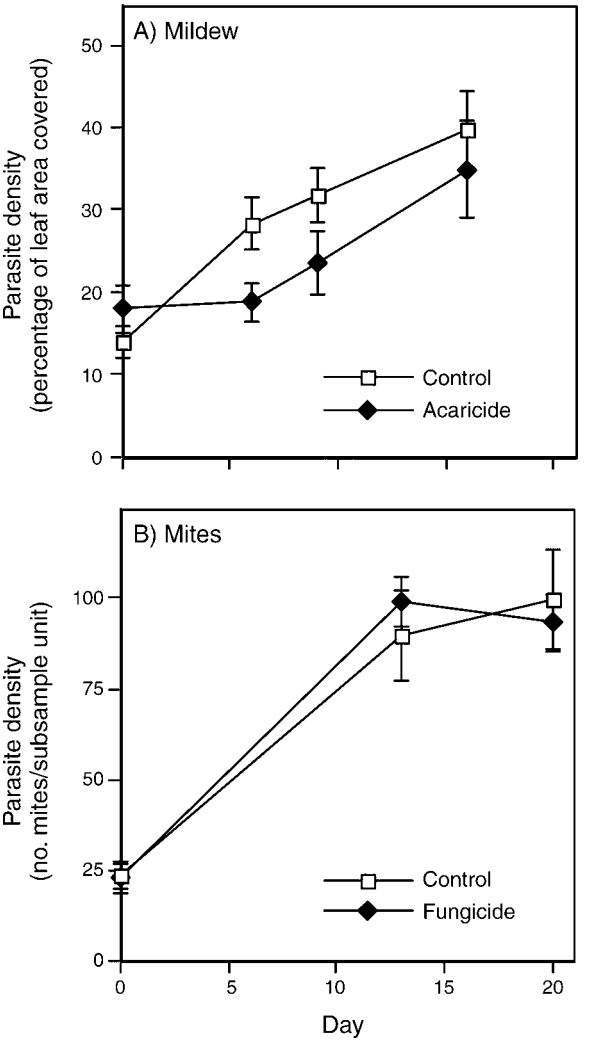

FIG. 2. Direct effects of (A) the acaricide abamectin on density of the powdery mildew *Oidium caricae* and (B) the fungicide myclobutanil on density of the rust mite *Calacarus flagelliseta* (no. mites/subsample unit on the lower leaf surface). All values are means ± SE. Our research was carried out at the University of Hawaii Poamoho Experiment Station on Oahu, Hawaii, USA.

Bioassays.—Population dynamics of the focal parasite were analyzed using a repeated-measures MANOVA with main effect for the pesticide treatments.

RESULTS

Bioassays

Test for direct effects of the acaricide on the fungus.—Overall, the acaricide abamectin had no direct effect on powdery mildew (MANOVA, $F_{1,18} = 0.83$, $P = 0.37$; Fig. 2A). However, for reasons that we do not understand, mildew colonies appeared to grow more slowly during

TABLE 1. Results of ANOVA tests (cumulative densities) and MANOVA tests (repeated measures) performed on the population dynamics of the herbivorous mite and powdery mildew for year 1 and year 2 of the study carried out at the University of Hawaii Poamoho Experiment Station on Oahu, Hawaii, USA.

Population dynamics	Test	Year 1			Year 2		
		F	df	*P*	*F*	df	*P*
Herbivorous mite							
Mite effect†	ANOVA	9.57	1, 30	0.004	959.54	1, 35	0.0001
Mite effect†	MANOVA	37.10	1, 30	0.0001	802.63	1, 35	0.0001
Mite × mildew	MANOVA	0.68	1, 30	0.41	14.06	1, 35	0.0006
Effect of mildew on mite dynamics‡	ANOVA	0.03	1, 30	0.86	22.80	1, 35	0.0001
Powdery mildew							
Mildew effect§	ANOVA	22.98	1, 30	0.001	1534.95	1, 35	0.0001
Mildew effect§	MANOVA	13.18	1, 30	0.001	1443.74	1, 35	0.0001
Mite × mildew	MANOVA	7.48	1, 30	0.01	6.46	1, 35	0.016
Effect of mites on mildew dynamics¶	ANOVA	15.50	1, 30	0.0004	7.80	1, 35	0.008

† Significant mite effect indicates that we were successful in establishing the +mite and −mites treatments.
‡ Pairwise contrast: +mite, +mildew vs. +mite, −mildew.
§ Significant mildew effect indicates that we were successful in establishing the +mildew and −mildew treatments.
¶ Pairwise contrast: +mite, +mildew vs. −mite, +mildew.

days 0–7 in the acaricide treatment, but thereafter grew very comparably with colonies in the control treatment. The time × treatment interaction was therefore significant (MANOVA, $F_{3,18} = 4.81$, $P = 0.01$).

Test for direct effects of the fungicide on the mite.—The fungicide myclobutanil had no direct effect on rust mites (MANOVA, $F_{1,18} = 0.01$, $P = 0.91$; Fig. 2B). Rust mite populations grew rapidly in both the control and fungicide treatments.

Field experiments: years 1 and 2

The results obtained in years 1 and 2 were highly concordant.

Population dynamics.—In both years, we were successful in establishing and maintaining our experimental treatments by selectively suppressing mites and powdery mildew (Table 1, Figs. 3, 4). The acaricide produced very strong suppression of the spider mites (data not shown) and the rust mites in year 1 (Fig. 3A) and year 2 (Fig. 4A). For both years, densities of spider mites remained very low (0–5 adults/leaf; V. Fournier, *unpublished data*) even on trees that were not sprayed with abamectin; therefore, we only report rust mite counts. The fungicide produced only partial suppression of the mildew (Fig. 3B) in year 1, and thus we likely underestimated the magnitude of powdery mildew effects on the host plant that year. In year 2, however, we achieved nearly complete suppression of mildew infection in the −mildew treatments by applying the fungicide weekly instead of every other week as we had done in year 1 (Fig. 4B).

In year 1, the density of rust mites peaked in August 2000 at 75.7 ± 22.3 motile individuals/subsampling unit on trees not treated with acaricide (mean ± SE; Fig. 3A). The density of powdery mildew colonies also peaked in August 2000 at 158.0 ± 14.2 colonies/half leaf (equivalent to coverage of ~20%/leaf) on trees not treated with fungicide, but that year trees were not sampled over the winter when powdery mildew usually reaches its highest densities (Fig. 3B). In year 2, the density of rust mites reached 146 ± 6.2 motile individuals/subsampling unit in June 2002 on trees not sprayed with the acaricide (Fig. 4A). The mean densities observed over the course of this experiment were similar to those observed in year 1. Powdery mildew prevalence peaked at 35.5% ± 0.9% leaf coverage (approximately equivalent to 300 colonies/half leaf) in January 2002 on trees not treated with fungicide (Fig. 4B), and mean densities observed over the course of the experiment were similar to those observed in year 1.

In year 1, rust mite populations were not influenced by the presence of powdery mildew (Table 1, Fig. 3A). Moreover, the mite × mildew interaction term was not significant (Table 1), suggesting that the population dynamics of rust mites were similar in the presence and absence of mildew. In year 2, however, the rust mite populations were significantly lower in the treatment in which mildew was suppressed (Table 1, Fig. 4A). The mite × mildew interaction term was also significant (Table 1). Given that mite densities were suppressed in the +mites, −mildew treatment during the first summer of the experiment when powdery mildew densities were near zero, we suspect that weekly applications of the fungicide myclobutanil had a subtle, direct, negative effect on rust mites that was not expressed during our bioassay or during year 1, when the fungicide was applied only every two weeks.

For both years, the density of powdery mildew colonies was significantly higher in the absence of rust mites (Table 1, Fig. 4B). The mite × mildew interaction terms were also significant (Table 1), supporting the view that the presence of mites altered the seasonal dynamics of the mildew population. Moreover, the mean diameter of fungal colonies, which we measured only at the end of year 1, was significantly smaller in the presence of rust mites (0.6 ± 0.3 cm) than in their

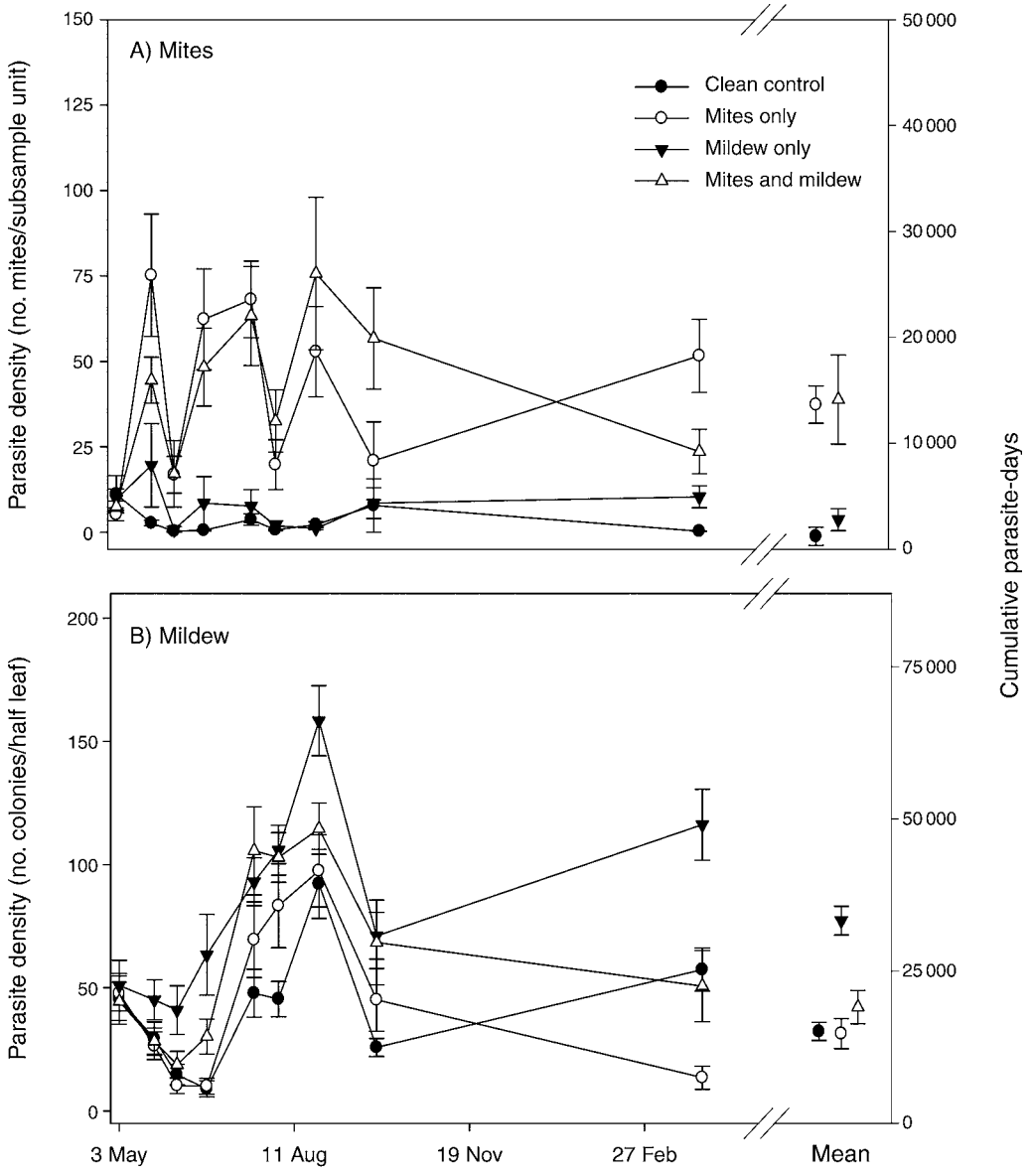

FIG. 3. Year 1 population dynamics (density, mean ± SE) and cumulative parasite-days (mean ± SE) of (A) rust mites and (B) powdery mildew from May 2000 to March 2001. Refer to Table 1 for ANOVA and MANOVA results.

absence (1.3 ± 0.2 cm; pairwise contrast, $F_{1,29} = 6.1$, $P = 0.02$). Thus, powdery mildew established a larger number of colonies and the colonies grew to larger sizes when rust mite populations were suppressed.

These findings suggest that rust mites and powdery mildew interact asymmetrically: rust mites have a negative effect on mildew, but mildew has no detectable effect on rust mites.

Plant performance.—In both years we detected significant treatment effects for leaf longevity, total number of leaves, trunk circumference, and fruit yield, but not for tree height (year 1, $F_{3,30} = 0.98$, $P = 0.41$; year 2, $F_{3,36} = 1.21$, $P = 0.32$; Fig. 5). The mites × mildew interaction term was never significant under the multiplicative risk model (Table 2). However, for year 1 and under the additive risk model, the interaction term was significant for leaf longevity, the total number of leaves per tree, and when all variables were analyzed simultaneously (Table 2); in each of these cases, the combined impact of mites and mildew generated a significantly smaller drop in performance than expected for the independent action of the parasites (antagonistic or less-than-additive damage).

In year 1, five trees assigned to the +mites, +mildew treatment died after beginning to produce mature fruit, whereas no trees in the other treatments died. This

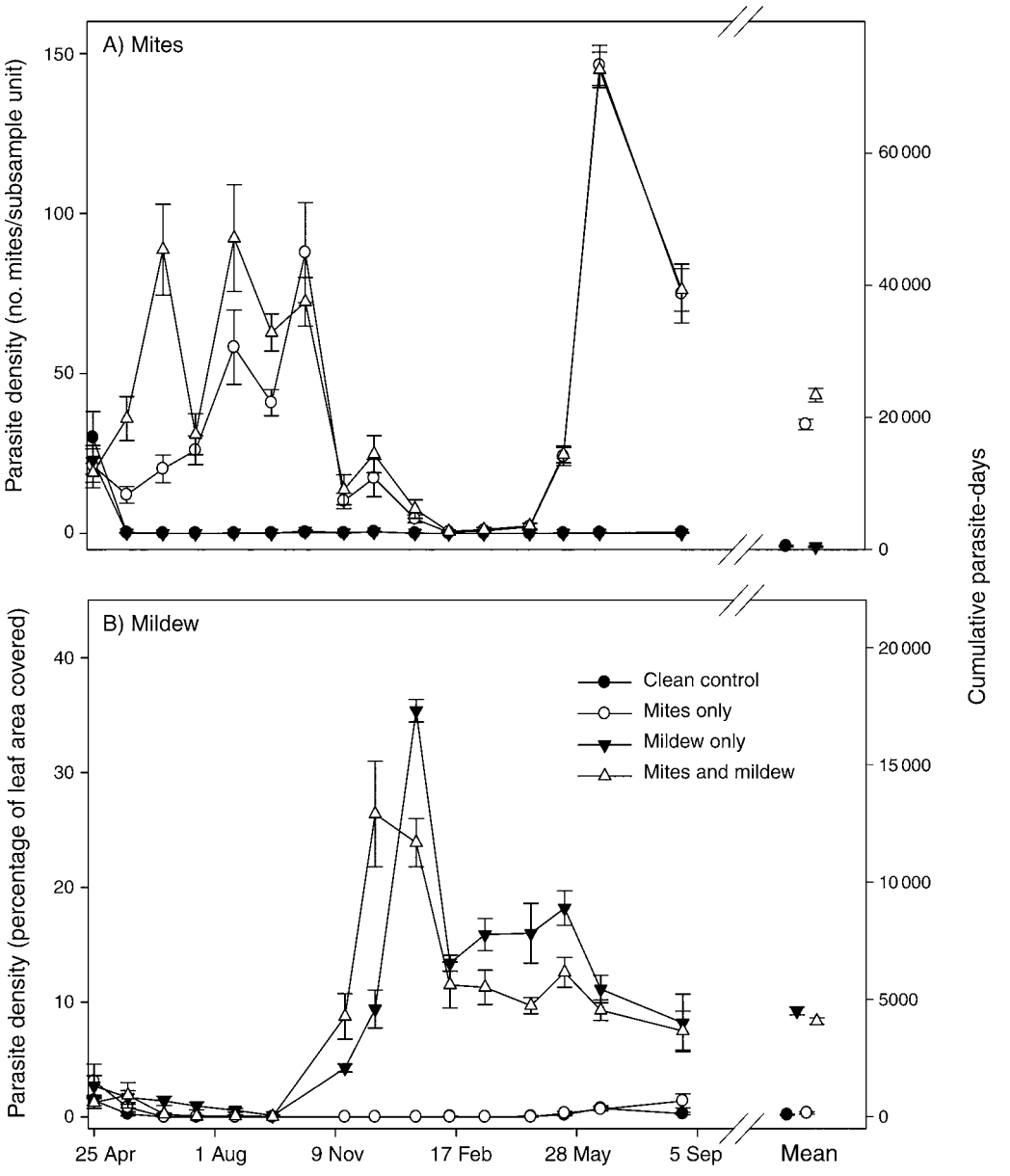

FIG. 4. Year 2 population dynamics (density, mean ± SE) and cumulative parasite-days (mean ± SE) of (A) rust mites and (B) powdery mildew from April 2001 to August 2002. In some cases the error bars are too small to be shown. Refer to Table 1 for ANOVA and MANOVA results.

pattern of mortality suggests a strong effect of treatment on tree survival (Fisher's exact test, $P = 0.00029$). Logistic regression of treatment effects on tree survival revealed a significant mites × mildew interaction term (Wald test, $\chi^2 = 4.17$, $P = 0.04$); in this case, the combined impact of mites and mildew generated a significantly larger drop in tree survival than expected for the independent action of the parasites (synergism). No tree mortality occurred in year 2.

Plant compensation.—In year 1, the relationships between cumulative mite and mildew population size and plant performance were well described by linear regressions with significant negative slopes for all variables measured (Fig. 6). Moreover, for all variables but one (number of leaves per tree, Fig. 6J), we found nonsignificant quadratic terms (parasites^2; $P > 0.05$). Thus, we saw no evidence for nonlinearities in the compensation response of papaya to either mite herbivory, mildew infection, or their combined attack.

The plant performance data from year 2 could not be used effectively to assess the shape of the plant compensation function. The experimental treatments were so effective during the second year that they produced consistently near-zero levels of mites and

mildew where we tried to suppress them. Moreover, there was very little natural variation across replicate trees in mite and mildew densities in the treatments in which we did not suppress them (data not shown). Thus, we did not obtain the continuous variation in parasite density levels across replicate trees that is needed to test for nonlinear plant performance responses to increasing parasite density.

Discussion

We used a holistic approach to investigate interactions among a herbivorous mite, a fungal pathogen, and their shared host plant, papaya. To our knowledge, this is the first report on plant–herbivore–pathogen interactions to address simultaneously (1) the reciprocal effects of the plant parasites on one another's population size, (2) the compensatory responses for parasite damage, and (3) the joint impact of parasites on plant performance. Our two in-field, manipulative experiments produced congruent results: (1) herbivorous mites had a moderate, negative effect on powdery mildew, but mildew had no effect on mite populations; (2) when evaluated alone, each parasite significantly reduced several measures of papaya performance (leaf life span, number of leaves per tree, trunk circumference, and fruit yield); (3) mites and powdery mildew had mostly additive or slightly less-than-additive effects on plant performance; and (4) plant performance declined linearly with increasing populations of either rust mites or powdery mildew.

Negative effect of herbivorous mites on powdery mildew

Powdery mildew colonies were smaller and fewer in number when mites were present. This antagonistic effect was observed despite the fact that the populations of these two parasites are, to some extent, seasonally segregated, with rust mites reaching their highest densities during the spring, summer, and fall and the mildew population peaking during the winter (e.g., Fig. 4; Yee et al. 1970, Fournier et al. 2004*a*, *b*). Such seasonal variation in the intensity of competition among species has been observed in many systems (Connell 1983, Schoener 1983).

The mechanisms underlying the negative impact of mites on mildew are unknown. However, as both parasites obtain their nutrients from the first layer of epidermal cells (McCoy and Albrigo 1975, Braun 1995, Oldfield 1996; J. Amrine, *personal communication*; R. Bélanger, *personal communication*), we suggest that exploitative competition is a likely candidate. With their cheliceral stylets, eriophyoid mites generally puncture the same cell many times, gradually causing mechanical injury, emission of ethylene, lignification, and finally cell death (McCoy and Albrigo 1975). Powdery mildews cannot develop on mechanically injured cells (Bushnell 2002). In contrast with other pathogens that require wounds to infect plant tissue (e.g., some viruses and bacteria), infection rates of powdery mildews are greatly reduced by host wounding

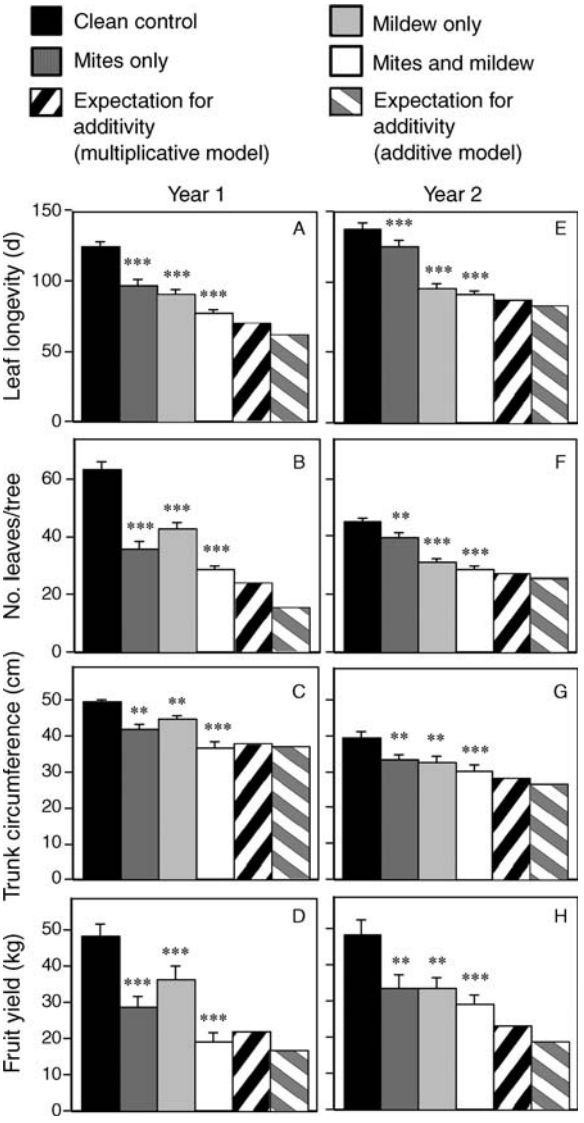

FIG. 5. Impact of powdery mildew and rust mites on (A, E) leaf longevity, (B, F) number of leaves per tree, (C, G) trunk circumference, and (D, H) fruit yield for each treatment (mean + SE). In each graph of the panel, the cross-hatched bars represent the predicted expectations for additivity under the multiplicative risk model and the additive risk model. Asterisks above bars report the results of pairwise contrasts of that treatment with the clean control treatment; $^{**}P < 0.01$; $^{***}P < 0.001$. The combined action of mites and mildew generated reductions that were generally greater than that observed for the mean of the parasites' individual effects (pairwise contrast of presence of both mites and mildew vs. pooled presence of mites only and presence of mildew only; year 1, leaf longevity, $F_{1,36} = 16.8$, $P = 0.0002$; total number of leaves per tree, $F_{1,30} = 9.3$, $P = 0.004$; trunk circumference, $F_{1,30} = 16.8$, $P = 0.003$; fruit yield, $F_{1,34} = 9.3$, $P = 0.004$; year 2, leaf longevity, $F_{1,36} = 20.5$, $P < 0.0001$; total number of leaves, $F_{1,35} = 21.2$, $P < 0.0001$; trunk circumference, $F_{1,36} = 2.5$, $P = 0.12$; fruit yield, $F_{1,36} = 1.2$, $P = 0.27$).

TABLE 2. Results of ANOVA tests performed on the plant performance parameters for the interaction term mites × mildew under both the multiplicative and the additive risk models.

		Multiplicative risk model, mites × mildew		Additive risk model, mites × mildew	
Plant performance variables	df	*F*	*P*	*F*	*P*
Year 1					
All variables combined†	4, 27	1.33	0.28	3.30	0.02
Leaf longevity	1, 36	2.57	0.12	5.59	0.02
No. leaves/tree	1, 36	2.51	0.12	7.10	0.01
Trunk circumference	1, 30	0.29	0.59	0.04	0.83
Fruit yield	1, 34	0.51	0.48	1.08	0.31
Year 2					
All variables combined†	4, 32	0.37	0.83	0.65	0.62
Leaf longevity	1, 36	0.48	0.49	1.14	0.29
No. leaves/tree	1, 35	0.50	0.49	1.42	0.24
Trunk circumference	1, 36	0.99	0.32	1.47	0.23
Fruit yield	1, 36	1.27	0.27	2.43	0.12

† The analyses for "All variables combined" were performed using MANOVA (identity response design) with main effects for mites, mildew, and the mites × mildew interaction (SAS 2000).

(Bushnell 2002). Informal observations of papaya leaves suggest that rust mites can live in mildew colonies when the density of mycelia is low to moderate (V. Fournier, *personal observation*). It is possible that the fungal pathogen invades some cells, but not others, and that the mites are feeding on the cells not yet exploited by the fungus. Therefore, the mites may be slowing mildew colony growth, both at the periphery of the colony and within the colony, by utilizing the host resource before the fungus. We do not know whether or not rust mites can exploit cells already invaded by the mildew.

We believe that other mechanisms, such as direct consumption of fungal material by mites, direct effects of the acaricide on the fungus, and interference competition via induced plant resistance, are unlikely to be involved in our system. First, while some families of mites are known to feed on fungi (Krantz 1978, English-Loeb et al. 1999, 2005), mycophagy seems very unlikely in the Eriophyoidea, which are thought to be strictly phytophagous (Lindquist et al. 1996).

Second, we conducted bioassays to evaluate the impact of the acaricide on the fungus in the absence of the rust mites and found no significant main effect after three applications over 16 days but a significant time × acaricide interaction (Fig. 2A). The significant interaction suggests that if there is an effect of the acaricide on the fungus, this effect is negative rather than positive (see Fig. 2A). Thus, if anything, our study might have underestimated the negative effect of mites on mildew, because the acaricide that was used to remove the mites may itself have slowed mildew colony growth somewhat. However, other sources of information about the mode of action of ivermectins cast doubt on any significant effect of these compounds on powdery mildew growth. Ivermectins are poisons that act at the neuromuscular junction (gamma-amino-butyric acid [GABA]-receptor agonists; Turner and Schaeffer 1989, Lasota and Dybas 1991), target sites that are highly unlikely to be found in fungi. Experimental studies have also consistently demonstrated that ivermectins have no significant impact on fungal activity (e.g., Wang and Pong 1982, Halley et al. 1990, de Oliveira and Neves 2004, Kollmann et al. 2004).

Finally, we previously reported on field experiments in which we assessed the possibility that rust mites might induce systemic host plant resistance in papaya towards mildew; these experiments produced no evidence for induced resistance (Fournier et al. 2004*b*). Several studies have found induced plant resistance to be responsible for negative interactions observed between herbivores and pathogens exploiting the same host plant (e.g., Karban et al. 1987, Hatcher et al. 1994*a*, 2004, Stout et al. 1999, Rostás and Hilker 2002, Simon and Hilker 2003, reviewed in Stout et al. 2006).

Additional work is needed to assess further the mechanisms underlying the negative impact of rust mites on powdery mildew. However, our work to date is consistent with a primary role for exploitative competition.

No effect of powdery mildew on herbivorous mites

We observed different patterns of rust mite densities in the +mites, +mildew and +mites, −mildew treatments in the two field experiments (Figs. 3A, 4A). We suspect that this discrepancy is attributable to a small, direct, negative effect of the fungicide on rust mites, which was only expressed during year 2 when we doubled the frequency of fungicide application. We suggest a direct effect of the fungicide, rather than an indirect effect mediated by the mildew, for two reasons. First, rust mites were clearly suppressed on trees treated with myclobutanil during the first five months of the year 2 trial, when powdery mildew densities were near zero (Fig. 4A). And second, the suppression of rust mite densities on the fungicide-treated trees disappeared during the last four months of the year 2 trial, after

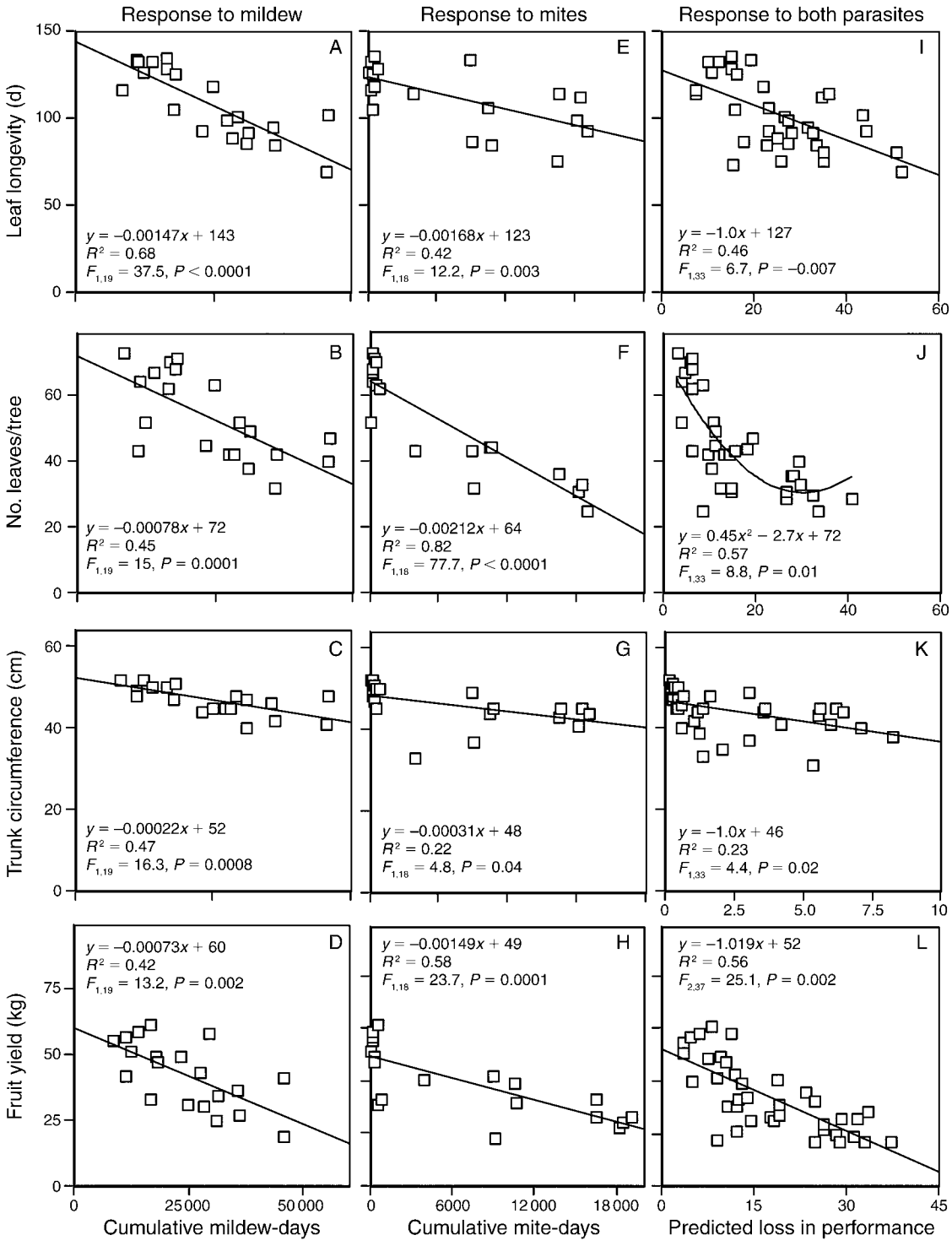

FIG. 6. Year 1 papaya performance in response to different cumulative population densities of rust mites and powdery mildew (panels A–H) or compared to predicted loss in performance due to the combined presence of mites and mildew (panels I–L). Shown are the responses of (A, E, I) leaf longevity, (B, F, J) number of leaves per tree, (C, G, K) trunk circumference, and (D, H, L) fruit yield to different intensities of powdery mildew infestation and mite herbivory (cumulative parasite-days).

we reverted to treating the trees only every other week (Fig. 4A). On the other hand, our bioassay testing the direct effect of the fungicide on the mites did not show any detrimental impact over a 20-day period (Fig. 2B). Whether a longer trial would have produced different results remains an open question.

The small negative effect of myclobutanil on mite populations observed in year 2 may have resulted in an underestimation of the impact of mites on plant performance. However, because our results are consistent across years, the magnitude of the underestimation is likely to be small.

Joint impact on plant performance

The natural history of the system may be crucial in choosing an appropriate model to test for independent effects on plant performance (Wootton 1994). In our system, an evaluation of the foraging behavior of rust mites and mildew suggested that the ideal null hypothesis for independent action included elements from both the multiplicative and additive risk models. Despite the differences between these two models (Rees and Brown 1992, Billick and Case 1994, Sih et al. 1998, Hambäck and Beckerman 2003), their predictions were quantitatively similar for most of our performance variables (Fig. 5). It appears that the negative impact of rust mites on powdery mildew populations was not strong enough to generate statistically significant antagonistic effects for all the performance variables (Table 2). The two variables for which we did find a significant interaction term using the additive risk model were not normally distributed, but did exhibit homoscedasticity (data not shown). The robustness and power of ANOVA rely more strongly on the assumption of homoscedasticity than on the assumption of normality (Ramsey and Schafer 2002). Furthermore, when outliers were omitted from the data sets to satisfy the normality condition, the interaction terms were still statistically significant (data not shown). For both of these reasons, we suggest that the statistical support for our interaction terms is real.

Few studies on plant–pathogen–herbivore interactions have reported antagonistic effects on plant performance (reviewed in Hatcher 1995). For instance, in a two-year field study, Johnson et al. (1986) found a less-than-additive effect on potato yield but only at times when infestation levels of the early blight fungus, *Alternaria solani*, and the potato leafhopper, *Empoasca fabae*, were high. Hatcher and coworkers showed that the negative interaction between the rust fungus *Uromyces rumicis* and the beetle *Gastrophysa viridula* was reciprocal and important under both laboratory and natural conditions (Hatcher et al. 1994*a*, *b*, *c*, 1995, Hatcher and Ayres 1997). However, when the combined impact of herbivory and infection was measured on *Rumex*, Hatcher did not find less-than-additive effect, which would be expected when both plant parasites affect one another in a negative fashion, but rather found an additive effect (Hatcher 1996). While this discrepancy can be explained as being the result of spatial separation of the rust fungus and the beetle (see Hatcher and Paul 2001), an alternative explanation may be that *Rumex* exhibited compensation breakdown under the pressure of both parasites. Compensatory growth of *Rumex* in response to *U. rumicis* and *G. viridula* was not formally examined (but see Hatcher and Paul 2001).

Compensatory growth response

The possibility that compensatory responses of papaya might be overwhelmed by summing the insults generated by multiple parasites is not generally supported in this system (Fig. 6). The sole exception to this conclusion was the concentration of tree mortality in the +mites, + mildew treatment during year 1. The combined impact of rust mites and powdery mildew appeared to render papaya plants vulnerable to the effects of the soilborne pathogen *Phytophthora palmivora*, which produced 50% tree death in the +mites, +mildew treatment, whereas no trees in the other treatments died after they began fruiting. Although 50% tree death is a dramatic example of compensation breakdown, implications for nonadditive impacts on longer-term host plant performance are unclear, because tree mortality might have occurred in other treatments had the duration of the experiment been extended. This possibility and the possible longer-term joint effects of mites and mildew on papaya performance would be valuable topics for further exploration.

Papaya exhibited simple, linear compensation in a wide suite of performance variables, including fruit yield, suggesting that any deviations from additive effects on plant performance should be due to one parasite interfering with the other, rather than a plant-based effect. The presence of linear compensation also means that we should expect plant performance variables to directly reflect cumulative parasite populations, both when they are present individually and when they are present together. This is consistent with our results: we have demonstrated that mites reduce the ability of mildew to exploit the papaya foliage. Therefore, the somewhat less-than-additive effects of rust mites and mildew on papaya that we observed is entirely consistent with predictions based on the asymmetrical competitive interactions between these two parasites. Thus, in our study system, an understanding of the natural history of two interacting plant parasites appears to explain their asymmetrical competition, which in turn appears to explain their joint impact on plant performance.

Implications for applied ecology

Our results have important implications for applied ecologists working in the fields of pest management and weed biological control. If the goal of weed suppression programs is to maximize the overall impact of herbivory and parasitism on target plant populations, then

combinations of plant exploiters that interact additively or synergistically will be especially valuable and thus important to identify. Here we have shown that even two agents that prefer to attack precisely the same plant structures (the epidermal cells on the underside of leaves) may still produce nearly additive effects on overall plant performance and perhaps synergistic effects when combined with a soil-borne pathogen. This result suggests that ample opportunities should exist to augment the overall impact of plant antagonists by combining the impacts of multiple exploiting species (cf. Denoth et al. 2002).

Acknowledgments

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