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# Impact of Plant Growth-Promoting Rhizobacteria and Natural Enemies on *Myzus persicae* (Hemiptera: Aphididae) Infestations in Pepper

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**ABSTRACT** Management of green peach aphid, *Myzus persicae* (Sulzer) (Hemiptera: Aphididae), in bell pepper, *Capsicum annuum* L., was explored through a combination of plant growth-promoting rhizobacteria (PGPR) and endemic biological control in New York in 2006 and 2007. We hypothesized that by using PGPR-treated peppers 1) *M. persicae* infestations would be reduced via induced resistance, 2) natural enemies would be lured to plants through the elicitation of volatile organic compounds, and 3) yield amount and quality would be improved. Pepper seed was planted in soil containing the PGPR formulation BioYield or untreated soil. Plants were transplanted to field plots and then treated with an insecticide regimen designed to remove or conserve populations of natural enemies. Apterous aphids and natural enemies were counted weekly on plants and pepper fruit were harvested, graded and weighed three times. PGPR did not directly or indirectly reduce aphid densities in either year. In 2006, there were more natural enemies in PGPR-treated plots than untreated ones, but this was probably a density-dependent response to aphid densities rather than a response of natural enemies to volatiles from PGPR-treated plants. For the first harvest date in 2006, yield of all fruit grades, especially the premium Fancy Grade, was 1.7–2.3 times greater in PGPR-treated plots than in untreated plots. However, no differences in yield were observed for the other two harvest dates or overall yield in 2006; no differences in yield among treatments were detected in 2007. Our results suggest that PGPR will not significantly impact *M. persicae* infestations or natural enemy populations but could enhance yield and quality of pepper fruit in some years.

**KEY WORDS** *Myzus persicae*, plant growth-promoting rhizobacteria, pepper, biological control

Bell pepper, *Capsicum annuum* L., is grown on nearly 120 ha (300 acres) in New York and is valued at over US\$6 million annually (NASS 2008). The principal arthropod pest of pepper in New York is the European corn borer, *Ostrinia nubilalis* (Hübner). Broad-spectrum insecticides used to control *O. nubilalis* can cause outbreaks of an important secondary pest, the green peach aphid, *Myzus persicae* (Sulzer). This common phenomenon occurs when *M. persicae* populations are resistant to the insecticide applied (Foster et al. 2000), but populations of natural enemies that often reduce *M. persicae* infestations are susceptible. *M. persicae* has developed resistance to many of the major pesticide groups, including carbamates, organophosphates, and pyrethroids (Devonshire and Moores 1982). *M. persicae* may parthenogenically produce up to ten offspring per day (Eastop 1977), which over a season can lead to 22 generations on summer hosts (Horsfall 1924). This high rate of fecundity and their propensity

for developing resistance to insecticides makes *M. persicae* an important pest to manage with tactics other than insecticides.

Plant growth-promoting rhizobacteria (PGPR) can enhance a plant's ability to defend itself from insects and pathogens by eliciting defensive responses, also known as induced resistance (Kloepper et al. 2004). Induced resistance is the production of defensive compounds in response to attack by a pathogen or herbivory. Induced resistance may be grouped into two categories, systemic acquired resistance (SAR) and induced systemic resistance (ISR), depending on how the resistance was triggered and which chemical pathways are activated. ISR is triggered in response to herbivory, drought, certain pathogens, and mechanical wounding. ISR is mediated through the activation of the jasmonate pathway (Thaler et al. 2001). SAR may be triggered by pathogens and some insects, such as aphids (for review, see van Loon et al. 1998) and is mediated through the activation of the salicylic acid pathway. Stimulation of either or both pathways can directly impact aphids by reducing fitness, fecundity, and feeding (Cooper et al. 2005, Boughton et al. 2006).

PGPR also could indirectly reduce aphid populations by attracting natural enemies to plants. The elicitation of volatile organic compounds (VOC), which

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occurs when plant defense pathways are triggered by PGPR, has been shown to attract natural enemies (De Moraes et al. 1998, McGregor and Gillespie 2004, Girling et al. 2006). For example, mycorrhizal interactions within the rhizosphere significantly increase foraging in the aphid parasitoid *Aphidius ervi* Haliday (Guerrieri et al. 2004). Because PGPR-related VOCs act by triggering a broad range of defensive reactions, both direct and indirect, the development of resistance by pests is unlikely. Perhaps PGPR applied to pepper plants could directly and indirectly regulate *M. persicae* infestations by reducing their fitness, fecundity, and feeding, as well as attracting natural enemies to increase aphid mortality.

Herman et al. (2008) recently reported that the PGPR formulation, BioYield, had no statistically significant impact on *M. persicae* densities in pepper in New York. However, the season total numbers of *M. persicae* in BioYield-treated plots were 49 and 41% lower than in untreated plots in 2003 and 2004, respectively. In that study, natural enemies were reduced from test plots by using multiple applications of the broad-spectrum pyrethroid esfenvalerate. The purpose of those applications was to manipulate the system to promote increases in *M. persicae* populations in the field. *M. persicae* populations were considered high in 2003 but low in 2004 and virtually nonexistent in 2005. We expanded upon this previous study by examining the direct impact of BioYield on infestations of *M. persicae* as well as the impact of naturally occurring predators and parasitoids on *M. persicae* infestations.

The goal of this study was to examine the utility of PGPR, in combination with endemic natural enemies for controlling *M. persicae* and for improving pepper yield and quality. We hypothesized that by using PGPR-treated peppers 1) *M. persicae* infestations would be reduced via induced resistance, 2) natural enemies would be lured to plants through the elicitation of volatile organic compounds, and (3) yield amount and quality would be improved.

### Materials and Methods

**Planting and PGPR Inoculation.** Bell pepper seeds 'Camelot' were planted in 128-cell flats in Cornell Mix soil-less planting mix, prepared with a 4-1-1 ratio with perlite and vermiculite. In all studies, the PGPR formulation BioYield (Bayer CropScience, Research Triangle Park, NC) consisted of  $1.0 \times 10^9$  colony-forming units (CFUs) of *Paenobacillus macerans* GB122 and *Bacillus amyloliquefaciens* GB99 per liter of planting mix. For each flat, 1.2 g of BioYield was incorporated into the planting mix by using a soil mixer before seeds were planted. After seeding, the pepper flats were placed in a greenhouse (photoperiod of 16:8 [L:D] h) at the New York State Agricultural Experiment Station in Geneva, NY, until plants were  $\approx 8$  wk old. The peppers were moved into outdoor cold frames for a minimum of a week before transplanting.

Peppers were transplanted during the weeks of 12 June and 4 July 2006 and during the same week of 11

**Table 1.** Treatments examined for their impact on naturally occurring populations of green peach aphid and natural enemies in bell pepper in New York in 2006 and 2007

Factor 1 (PGPR)	Factor 2 (insecticide)	Intended impact on aphids	Intended impact on natural enemies
No PGPR	No insecticide	None	None
No PGPR	Pymetrozine (Fulfill)	Reduce	None
No PGPR	Esfenvalerate (Asana)	Increase	Reduce
PGPR	No insecticide	None	Increase
PGPR	Pymetrozine (Fulfill)	Reduce	Increase
PGPR	Esfenvalerate (Asana)	Slight increase	Reduce

June 2007. Field plots consisted of six rows and four rows in 2006 and 2007, respectively. Rows were 7.6 m in length and spaced 0.9 m apart. Plants were spaced at 0.3-m intervals within rows. In 2006 and 2007, plots were distributed across three fields that were separated by 0.5–2 km. Within each field, plots were separated from each other by a minimum of 15.2 m in 2006 and 7.6 m in 2007. All plots within a replication were located in the same field.

Drip irrigation was used to hydrate all plots in 2007, but no irrigation was necessary in 2006. Supplemental fertilization was also applied following standard recommendations (Reiners and Petzoldt 2008).

**Insecticides.** Insecticides were selected to manipulate populations of *M. persicae* and natural enemies to test our hypotheses (Table 1). The broad-spectrum pyrethroid esfenvalerate (Asana XL, DuPont, Wilmington, DE) was used at a rate of 0.3 liters/ha to reduce natural enemies in one third of the treatments. Esfenvalerate has a moderate toxicity to predators and high toxicity to parasitoids (University of California 2005). The other product was the aphicide pymetrozine (Fulfill, Syngenta Crop Protection, Greensboro, NC), which was used at a rate of 0.2 kg/ha to reduce aphids without affecting natural enemy populations in the plots. Finally, there were plots to which no insecticides were applied. All treatments were applied using a backpack sprayer and a three-nozzle boom delivering a spray volume of 271 liters/ha at 276 kPa. All insecticides were mixed with 0.5% (vol:vol) of Silwet surfactant.

Treatments were initially applied when the *M. persicae* population reached a threshold of one aphid per leaf. Additional applications were made 3–4 wk after the first spray. In 2006, treatment plots were sprayed accordingly with esfenvalerate and pymetrozine twice during the season, on 21 July and 17 August. In 2007, plots were sprayed with the insecticide treatments three times: 11 July, 9 August, and 9 September.

**Effect of PGPR on Aphids and Natural Enemy Densities.** We hypothesized that if PGPR triggered a direct negative effect on aphid populations, our best opportunity to observe this response in the field would be in plots treated with esfenvalerate, which would remove natural enemies that often regulate

aphid populations. Thus, we expected to observe lower aphid densities in PGPR plots treated with esfenvalerate than in plots without PGPR treated with esfenvalerate (Table 1).

There were six treatments in this 2-yr project. The experiment was designed as a 2 by 3 factorial with PGPR as the first factor (PGPR versus no PGPR) and insecticide as the second factor (no insecticide, pymetrozine and esfenvalerate). All treatments were arranged in a randomized complete block design and replicated five times in 2006 and eight times in 2007.

*M. persicae* and natural enemies were surveyed in plots nearly every week from 17 July to 27 September in 2006 and from 3 July to 11 October in 2007. In 2006, aphids and natural enemies were counted from 30 plants randomly chosen from each of the two middle rows. Because no significant difference in aphid numbers per plant was found between counting aphids on 30 and 15 plants, sample size was reduced to 15 plants in 2007. Three leaves were randomly chosen from the middle canopy of each plant, and the number of apterous adults and immatures was recorded.

We also hypothesized that natural enemies would be higher in PGPR-treated plots than in those not treated with PGPR because they would be attracted to VOCs. Testing this idea required the use of a selective insecticide such as pymetrozine that would 1) reduce aphid populations to eliminate the possibility of a positive density-dependent response by natural enemy populations and 2) reduce aphid populations in a manner that would not negatively impact natural enemies. Natural enemies that were observed in our study were recorded to the family level and included Anthocoridae, Chrysopidae, Coccinellidae, Hemero-biidae, Cecidomyiidae, and Syrphidae. Parasitized aphids (mummies) were recorded as an indicator of the relative abundance of hymenopteran parasitoids. No fungal epizootics were observed during this study.

**Yield.** We hypothesized that fruit yield or total fruit grades (e.g., Fancy grade) or both would be greater in PGPR-treated plots than in non-PGPR-treated plots. Peppers were harvested and graded at three points during each season. In 2006, peppers were harvested on 24 August, 21 September, and 3 October. In 2007, peppers were harvested on 29 August, 14 September, and 10 October. Peppers were divided into the three grades as defined by the USDA and used for national market orders (USDA 2005). Fancy grade peppers are defined as a fruit with >7.62-cm diameter and >8.90-cm length; U.S. no. 1 grade fruit must have a diameter between 6.35 and 7.62 cm, and a length >6.35 cm; and U.S. no. 2 grade fruit was considered unmarketable or as culls in our study. Fancy grade, no. 2 grade and total yield across all grades were analyzed. The no. 2 grade peppers were analyzed separately to ensure that any differences in yield between the treatments were due to increased marketable yields. An increase in no. 2 grade peppers, which would need to be culled, would increase picking costs, reducing profit. Yield differences solely attributable to our treatments were of primary interest. Therefore, we did not exclude fruit with physical flaws, European corn

borer injury or disease, all of which were uncommon in our study.

**Statistical Analyses.** The effects of BioYield, insecticide, sampling date, and their interactions with insect abundance were examined by a repeated measures analysis of variance (ANOVA) using the Proc Mixed procedure in SAS 9.1 (SAS Institute 2003). The variables PGPR, insecticide, and sampling date were considered fixed and unstructured in our models. First-order autoregressive covariance structures were used to model covariance between treatments and sampling dates by choosing a combination of Akaike's information criterion, which evaluates whether the model used best explains the data, while minimizing free parameters in the model. The expectation was that correlations would be greater for counts taken on dates that were closer together. For natural enemy data, the model was run as described above, then rerun with aphid data included as a covariate to account for any numerical effect. Replication was considered a random effect in the model. Independent statistical analyses were performed on each natural enemy group, but because trends were similar for each group, data were pooled into a single category called total natural enemies.

Total marketable fruit yield and yield separated by grade were both analyzed as a two-way ANOVA. Fruit harvested from each plot row was averaged to provide the average yield per row for the plot. In 2006, late planting due to weather conditions affected plant growth in two of the five replicates, which then were excluded from the analysis ( $n = 3$ ). All eight replicates were included in the 2007 analysis. Means were compared using the Proc Mixed procedure in SAS 9.1. Some data were transformed using a  $\log_{10}(x + 1)$  transformation to normalize data before analysis, but only untransformed data are presented. Natural enemy, parasitoid, and predator densities were compared with aphid densities using Pearson's product-moment correlation ( $P < 0.05$ ).

## Results

**Weather Conditions.** Weather conditions varied substantially between 2006 and 2007, particularly in June, when most peppers were transplanted into the field. The 2006 average temperature for June was 18.9°C, approximately equal to the 10-yr average. Rainfall in June 2006 was 12.7 cm, >2.9 cm above average. Precipitation in July and September was also above average. Extended periods of rainfall are stressful for peppers and may reduce growth (Jett 2006).

In 2007, Geneva experienced the second warmest June in the past 30 yr (National Weather Service, www.nws.noaa.gov). The average daily temperature was 19.9°C, which was 1°C above the 10-yr average. Rainfall in June 2007 was 5.33 cm, which was 5.08 cm less than average. Due to the dry conditions that persisted throughout the 2007 summer, drip irrigation was used for the entire field season. Irrigation significantly increases the vigor and yield of bell pepper (Costa and Gianquinto 2002).

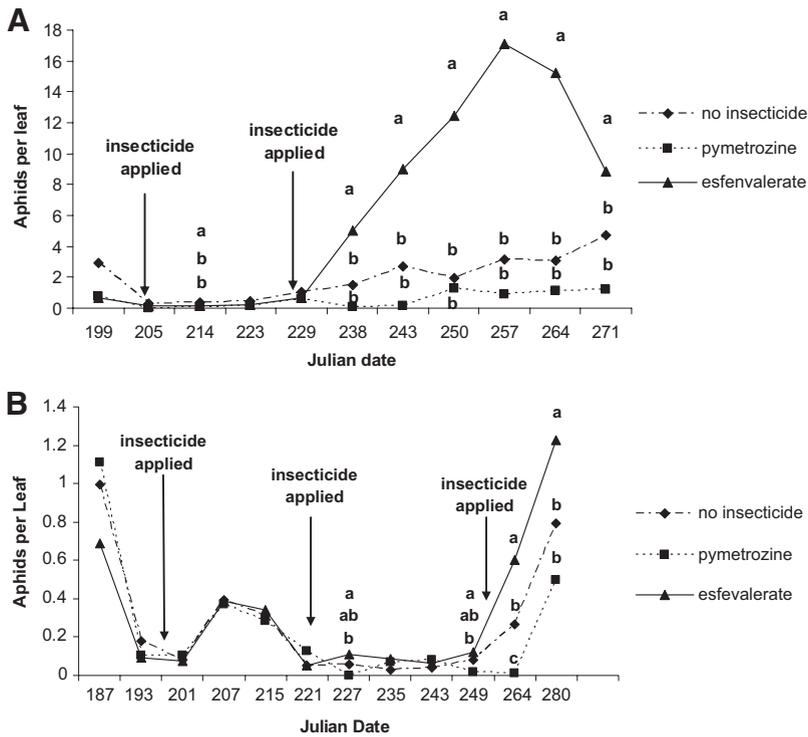


Fig. 1. Effect of insecticide on *M. persicae* densities on bell pepper through the season in Geneva, NY, in 2006 (A) and 2007 (B). Dates on which significant differences occurred among treatments are marked with letters. Treatment means sharing the same letter are not significantly different ( $P > 0.05$ ).

**Aphid Density.** Aphid densities differed substantially between the 2 yr (Fig. 1). In 2006, there was an overall average of 2.7 aphids per leaf, whereas the average was 0.3 aphids per leaf in 2007. PGPR had no effect on aphid density in either 2006 or 2007 (Table 2). Aphid density was significantly affected by insecticide as well as by an interaction between insecticide and date in both 2006 and 2007 (Table 2). Plots treated with esfenvalerate had significantly more aphids than those in untreated plots and those treated with

pymetrozine, especially after Julian date 229 in 2006 (Fig. 1A) and after Julian date 249 in 2007 (Fig. 1B). This difference was most dramatic in 2006 when there was a season average of  $6.9 \pm 1.0$  aphids per leaf in esfenvalerate-treated plots and only  $0.7 \pm 0.2$  aphids per leaf in the untreated control (Fig. 1A). The average number of aphids in plots treated with pymetrozine did not differ from the number in untreated plots in 2006 (Fig. 1A) and 2007 (Fig. 1B).

Aphid densities were not impacted by an interaction between PGPR and insecticide in either 2006 or 2007 (Table 2). We predicted that if PGPR had a negative effect on aphid densities, we would observe lower aphid densities in PGPR plots treated with esfenvalerate than in plots without PGPR treated with esfenvalerate (i.e., a significant PGPR  $\times$  insecticide interaction). However, the interaction did not exist and the opposite was observed. Plots treated with both PGPR and esfenvalerate had a season average of  $8.1 \pm 1.2$  aphids per leaf, whereas plots treated with esfenvalerate alone had an average of  $5.7 \pm 1.5$  aphids per leaf.

**Natural Enemies.** More natural enemies were observed in plots in 2006 than in 2007 (Fig. 2). Yet, the proportion of each group of natural enemies relative to the total number of natural enemies encountered was relatively similar between years (Fig. 2). Coccinellids and parasitoids were the most dominant groups observed.

Table 2. Significance of PGPR, insecticide, and their interaction on *M. persicae* on bell pepper through time in Geneva, NY, in 2006 and 2007

Source	df	F	P
2006			
PGPR	1	1.06	0.3039
Insecticide	2	28.79	<0.0001
PGPR $\times$ insecticide	2	0.33	0.7208
Date	10	11.72	<0.0001
PGPR $\times$ date	10	0.26	0.9886
Insecticide $\times$ date	20	6.22	<0.0001
PGPR $\times$ insecticide $\times$ date	20	0.39	0.9920
2007			
PGPR	1	0.01	0.922
Insecticide	2	10.06	<0.0001
Date	12	61.84	<0.0001
PGPR $\times$ insecticide	2	1.26	0.2851
PGPR $\times$ date	12	0.92	0.5239
Insecticide $\times$ date	24	3.19	<0.0001
PGPR $\times$ insecticide $\times$ date	24	1.27	0.1784

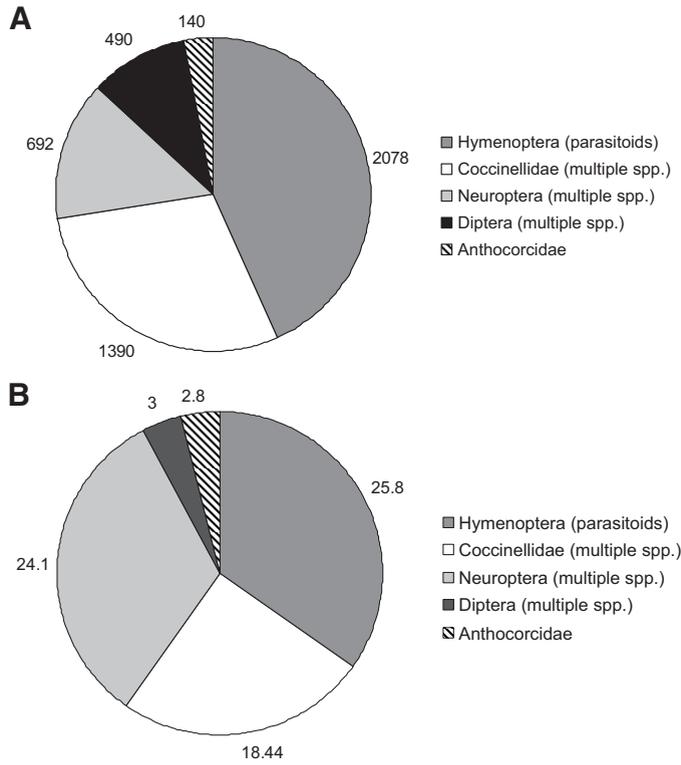


Fig. 2. Natural enemies of *M. persicae* sampled on plants across all treatments in 2006 (A) and 2007 (B) and their relative abundance.

PGPR had a significant effect on natural enemies in 2006, but not in 2007 (Table 3). Overall, 17% more natural enemies were observed in PGPR-treated plots than in those not treated with PGPR. Insecticide also

Table 3. Significance of PGPR, insecticide, and their interactions on total natural enemy density through time, in Geneva, NY, in 2006 and 2007

Source	df	Normal		Including aphid density as a covariate	
		F	P	F	P
<b>2006</b>					
PGPR	1	4.47	0.0354	0.69	0.0989
Insecticide	2	42.73	<0.0001	9.91	<0.0001
PGPR × insecticide	2	4.19	0.0162	4.19	0.0704
Date	10	9.57	<0.0001	9.57	<0.0001
PGPR × date	10	1.85	0.0526	1.85	0.0532
Insecticide × date	20	5.97	<0.0001	5.97	<0.0001
PGPR × insecticide × date	20	1.11	0.3423	1.11	0.3330
Aphid density				66.95	<0.0001
<b>2007</b>					
PGPR	1	1.79	0.1813		
Insecticide	2	1.20	0.3010		
PGPR × insecticide	2	1.84	0.1597		
Date	12	10.50	<0.0001		
PGPR × date	12	0.83	0.6244		
Insecticide × date	24	1.05	0.4029		
PGPR × insecticide × date	24	0.95	0.5305		

impacted natural enemy densities in 2006, but not in 2007 (Table 3). Over the entire 2006 season, the average number of natural enemies in esfenvalerate-treated plots ( $174 \pm 24$ ) was significantly greater than the average number in pymetrozine-treated ( $27 \pm 4$ ) or untreated plots ( $51 \pm 6.8$ ). In addition, more natural enemies were observed in esfenvalerate-treated PGPR plots than in esfenvalerate-treated plots that did not receive PGPR (i.e., interaction between PGPR and insecticide) (Table 3; Fig. 3).

We did not anticipate encountering more natural enemies in esfenvalerate-treated plots than in the other treatments because the purpose of using esfenvalerate was to eliminate natural enemies. This was not successful later in the season and we suspected that the high number of natural enemies in esfenvalerate-treated plots was a positive density-dependent population response to the high number of aphids in these plots. Thus, when the ANOVA model included aphid density as a covariate in the analysis, neither the PGPR main effect nor the interaction between PGPR and insecticide were significant. Natural enemy densities in both PGPR-treated ( $r^2 = 0.7$ ,  $df = 9$ ,  $P < 0.001$ ) and untreated plots ( $r^2 = 0.4$ ,  $df = 9$ ,  $P < 0.001$ ) were positively correlated with the number of aphids per leaf. These results indicated that the higher number of natural enemies in the plots treated with PGPR and esfenvalerate was a density-dependent numerical re-

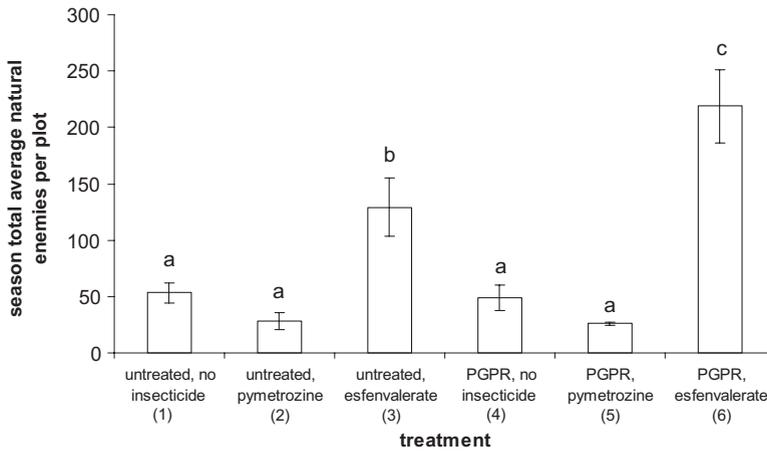


Fig. 3. Effect of PGPR and insecticide on natural enemy densities in 2006. Means  $\pm$  SEM sharing the same letter are not significantly different ( $P > 0.05$ ).

sponse to aphid populations within the plots, rather than a response of natural enemies to VOCs.

The impact of insecticide on natural enemy densities through time was similar to the impact that insecticide had on aphid densities through time in 2006 (Table 3; Fig. 4). More natural enemies were observed in esfenvalerate-treated plots than in the other treatments after Julian date 229 (Fig. 4), and no differences existed between pymetrozine and untreated plots.

**Yield.** In 2006, PGPR had a significant positive effect on both fancy grade (Table 4) and yield of all-grades in the first harvest (Table 5). The per-row average yields from the first harvest of Fancy grade peppers were  $1.6 \pm 0.4$  and  $3.6 \pm 0.6$  kg in untreated and PGPR-treated plots, respectively (Fig. 5). For all grades, PGPR-treated plots averaged  $5.2 \pm 0.6$  kg per row, whereas the average was only  $2.4 \pm 0.3$  kg per row in the untreated plots. Neither insecticide nor PGPR insecticide interaction had an effect on first harvest yield (Tables 4 and 5). For the second, third, and overall harvest yields, no significant differences were seen between PGPR-treated plots and untreated plots

in fancy grade (Table 4) and all grade categories (Table 5).

In 2007, there were no significant interactions between the variables PGPR, insecticide and yield (Tables 4 and 5). Similarly, there was no significant effect of insecticide or significant PGR by insecticide interaction that was biologically relevant.

**Discussion**

The PGPR formulation, BioYield, had no impact on *M. persicae* populations in pepper. This result was consistent across years that varied substantially in both aphid pressure and environmental conditions. We hypothesized that if PGPR triggered a direct negative effect on aphid populations, our best opportunity to observe this response would be in plots treated with esfenvalerate, which would remove natural enemies and allow aphid populations to reach higher levels. This approach successfully increased aphid populations, but natural enemy populations responded to the aphid increases in a positive density-dependent man-

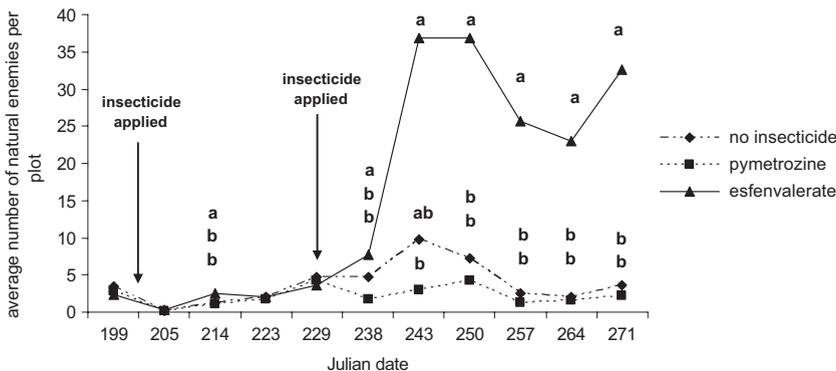


Fig. 4. Effect of insecticide on natural enemy densities of *M. persicae* over the 2006 season. Dates on which significant differences occurred between treatments are marked with a letter. Means sharing the same letter between treatment within a date are not significant ( $P \geq 0.05$ ).

**Table 4. Significance of PGPR, insecticide, and their interactions on fancy grade pepper yield across all three harvests and total yield in Geneva, NY**

Sources of variation	df	Harvest 1		Harvest 2		Harvest 3		Total	
		F	P	F	P	F	P	F	P
2006									
PGPR	1	18.50	0.0016	2.15	0.1737	4.92	0.0509	0.19	0.6755
Insecticide	2	0.34	0.7218	0.48	0.6304	1.21	0.3381	0.33	0.7247
PGPR × insecticide	2	0.85	0.4544	1.24	0.3298	0.09	0.9142	0.34	0.7217
2007									
PGPR	1	2.21	0.1459	0.00	0.9989	0.94	0.3402	0.02	0.8840
Insecticide	2	1.31	0.2835	1.33	0.2763	0.81	0.4528	1.88	0.1671
PGPR × insecticide	2	1.14	0.3328	1.67	0.2022	0.82	0.4484	2.18	0.1286

ner. Consequently, we were not able to compare high aphid population densities in PGPR-treated and non-treated plots in the absence of natural enemies. Nonetheless, our results are identical to those reported by Herman et al. (2008), who failed to detect a significant effect of BioYield on *M. persicae* densities in bell pepper in New York over 2 yr. In their study, test plots also were treated with multiple applications of esfenvalerate to increase aphid populations. Combining our results with those presented in Herman et al. (2008), there were four of 4 yr in which PGPR was not shown to reduce aphid populations. Thus, our first hypothesis—fewer aphids would establish in PGPR plots compared with untreated plots—was not substantiated.

Direct defenses triggered by PGPR or other elicitors have been shown to cause direct antixenotic and antibiotic defenses (Zehnder et al. 2001). Potato aphids, *Macrosiphum euphorbiae* (Thomas), that were fed tomato in the laboratory in which the jasmonic acid pathway had been artificially triggered, produced fewer offspring and had fewer offspring survive compared with the control (Cooper and Goggin 2005). If such a response occurred in pepper in our study, the difference was too small to detect under field conditions and likely not of practical value for aphid management.

Overall aphid population levels in our study were opposite of what would be expected given the weather conditions. Previous research has found that *M. persicae* migration and densities are positively correlated with temperature and have a negative correlation with rainfall (Cocu et al. 2005, Kuroli and Lantos 2006). Yet, we observed higher *M. persicae* population levels in 2006, which was cool and wet, than in 2007, which was

hot and dry. An exception to the 2007 weather pattern occurred in June, when >3 cm of rain fell in 24 h. Perhaps this single rain event reduced initial *M. persicae* populations and they never recovered.

Natural enemy densities were not impacted by PGPR. Although densities of natural enemies were significantly higher in plots treated with PGPR than in those not treated with PGPR in 2006, we demonstrated that these results were caused by a positive density-dependent response to high aphid densities. In 2007, when aphid pressure was similar across all treatments, no significant differences in natural enemy densities were seen between PGPR and untreated plots. Thus, there was no evidence supporting our hypothesis that PGPR triggered the plants' indirect defenses to attract higher levels of natural enemies.

The rapid colonization of natural enemies into pepper plots treated with esfenvalerate late in the season may have been exacerbated by the relatively small plot size. The large area of fallow land surrounding test plots supported natural enemies that apparently established quickly in plots after the residual activity of the esfenvalerate had dissipated. Perhaps, if this study was conducted in larger field plots, we would not have observed this phenomenon.

Endemic natural enemies were highly effective in managing *M. persicae* infestations in pepper in both years. Natural enemies reduced *M. persicae* populations to subeconomic threshold levels in all insecticide-free plots. Moreover, natural enemies provided an equivalent level of *M. persicae* reduction as that provided by multiple applications of pymetrozine.

PGPR did not consistently increase pepper fruit yield or grade in either year of our study. The only increase in fruit yield and grade in PGPR-treated plots

**Table 5. Significance of PGPR, insecticide, and their interactions on all grades of pepper yield across all three harvests and total yield in Geneva, NY**

Sources of variation	df	Harvest 1		Harvest 2		Harvest 3		Total	
		F	P	F	P	F	P	F	P
2006									
PGPR	1	12.68	0.0051	0.4	0.5372	2.45	0.1482	2.02	0.1857
Insecticide	2	0.06	0.9421	0.76	0.4869	1.19	0.3449	0.23	0.8011
PGPR × insecticide	2	0.22	0.8093	4.06	0.0450	1.75	0.2235	1.48	0.2739
2007									
PGPR	1	0.85	0.3622	0.00	0.9989	0.94	0.3402	0.15	0.7006
Insecticide	2	0.32	0.7314	1.33	0.2763	0.81	0.4528	2.20	0.1232
PGPR × insecticide	2	0.23	0.7963	1.67	0.2022	0.82	0.4484	0.43	0.6510

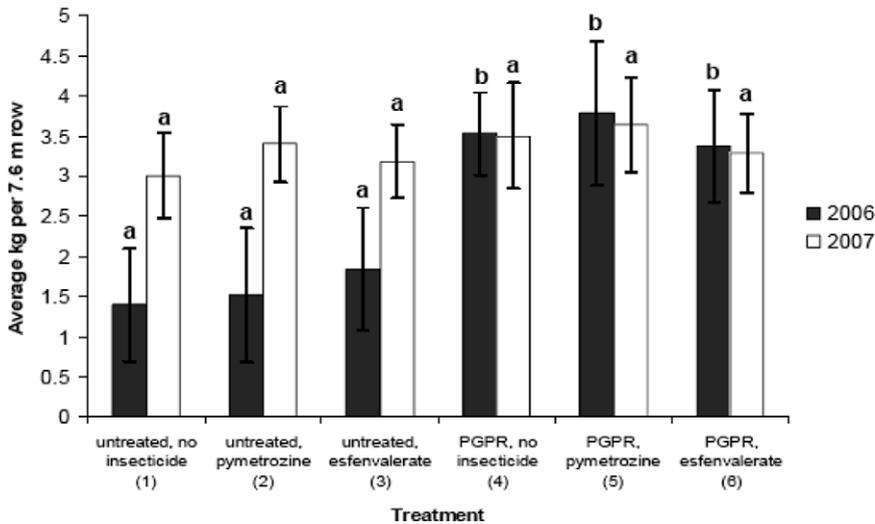


Fig. 5. Effect of PGPR on first harvest fancy grade pepper yield for 2006 and 2007. Means  $\pm$  SEM sharing the same letter between treatments and within years are not significantly different ( $P \geq 0.05$ ).

attributable to PGPR occurred during the first harvest in 2006. In this case, fruit yield was nearly doubled in plots treated with PGPR as compared with plots not treated with PGPR. Because environmental conditions were stressful for pepper growth early in 2006, the use of PGPR may have been responsible for the increase in yield. The absence of fruit yield increases in our study during later harvests in 2006 and all harvests in 2007 indicate that a yield benefit provided by PGPR will not occur consistently. Thus, we cannot conclude that PGPR will always enhance yield or grade of pepper.

During the period preceding the first harvest in 2006, aphid pressure was low (less than two aphids per leaf) and similar between all treatments. Thus, it is doubtful that the greater yields in PGPR plots were due to increased aphid tolerance at the time of fruit initiation and development. Murphy et al. (2003) reported that tomato plants treated with PGPR were  $\approx 10$  d ahead in physiological maturity than control plants of the same age. They also reported that PGPR-treated plants had a significantly greater number of early season buds. Boutard-Hunt (2008) recently showed that pepper plants treated with PGPR had more buds early in the plant's development compared with those not treated with PGPR. Greater fruit yield and percentage of fancy grade peppers produced in PGPR-treated plots early in our 2006 study could have been caused by the production of more early-season buds. Further investigation into the effect of PGPR on the initiation of bud development would be useful in elucidating the mechanism behind early season yield increases observed in this and other studies.

High *M. persicae* pressure can reduce pepper yield (Reiners and Petzoldt 2008), but we did not observe this in either year of our study. In 2006, aphid densities were much greater in esfenvalerate-treated plots and exceeded the action threshold more often than in

those plots sprayed with either pymetrozine or were not treated. Yet, fruit yield did not differ among these insecticide treatments. These results are consistent with those reported by Herman et al. (2008). The absence of pepper fruit yield loss attributed to *M. persicae* infestations suggests that this pest may not need to be managed with insecticides in small plantings of pepper in most years.

Our results indicate that the primary benefit of using PGPR in pepper production in New York may be as a soil amendment to buffer plants against early season stress, rather than as a management tactic for *M. persicae* or a lure for natural enemies. The use of PGPR in this regard may assure growers that they will have early-season marketable pepper yield even if growing conditions are not ideal.

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