# Biocontrol of Fusarium Wilt of Cucumber Resulting from Interactions Between *Pseudomonas putida* and Nonpathogenic Isolates of *Fusarium oxysporum*

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## ABSTRACT

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Fluorescent pseudomonads and nonpathogenic isolates of *Fusarium oxysporum* were effective in inducing suppressiveness to Fusarium wilt of cucumber when added to soil together (pH 6.7) but ineffective when added separately. Suppressiveness by such combination treatments was enhanced in nearly neutral (pH 6.7) to alkaline soils (pH 8.1), in comparison with acid soil (pH 5.5). Strains of fluorescent pseudomonads reduced the germination of chlamydospores of nonpathogenic and pathogenic isolates of *F. oxysporum* in the rhizospheres of cucumber plants. Population densities of fluorescent pseudomonads increased significantly in the rhizosphere of cucumber in the presence of a nonpathogenic isolate of *F. oxysporum* in soil of pH 8.1. It is hypothesized that the activity of fluorescent pseudomonads and their siderophore production are enhanced by increased root exudates induced by relatively high population densities of nonpathogenic isolates of *F. oxysporum*. This, in turn, leads to competition for iron, which is essential for successful germination of the pathogen and penetration of the host.

Researchers investigating *Fusarium*-suppressive soils have observed an abundance of *Fusarium* spp. that evidently are nonpathogenic to crops grown in the regions. Examples are found in the Salinas Valley of California (13) and in alluvial soils of the Chateaurenard region of France (15,16). Louvet et al (10) concluded that suppressiveness was induced by competition between pathogenic and saprophytic clones of *Fusarium* spp. for possession of soil niches, especially in locations supporting thalli capable of infecting the roots of hosts. Research by these French workers was done by reinfesting steamed soil with potentially antagonistic *Fusarium* spp. (1). Paulitz et al (14), however, established suppressiveness to Fusarium wilt of cucumber (*Cucumis sativus* L.), caused by *F. oxysporum* Schlecht. emend. Snyd. & Hans. f. sp. *cucumerinum*, by the addition of nonpathogenic isolates of *F. oxysporum* to raw soil. Mechanisms of biological control in raw soil could be different than those responsible for suppressiveness in previously steamed soil. Therefore, a study was undertaken to observe interactions of such nonpathogenic isolates of *F. oxysporum* with fluorescent pseudomonads, previously identified as biocontrol agents in suppressive soils (2,3,6–8,17,18). Additional studies were made to identify mechanisms associated with these interactions.

## MATERIALS AND METHODS

**Culture of fluorescent pseudomonads.** Strains of *Pseudomonas putida* A12 (18) and N1R (6), resistant to rifampicin at 100 μg/ml, were used. Bacteria were grown at 28 C in 250-ml Erlenmeyer flasks containing King's B broth medium (100 ml per flask). Cultures were incubated on a rotary shaker (100 rpm) for 48 hr. Bacterial cells were harvested by centrifugation for 10 min at 2,500*g*, washed, and resuspended twice in 0.01 M $MgSO_4$ solution. The concentration of bacterial cells was measured by absorbance at 780 nm with a spectrophotometer (Bausch and Lomb, Rochester, NY) and adjusted to specific concentrations by use of a standard absorbance curve.

**Production of chlamydospores of *F. oxysporum*.** Seven isolates of *F. oxysporum* were used. *F. o.* f. sp. *cucumerinum* (18) was pathogenic to cucumber. Nonpathogenic isolates C1, C5, C10, and C14 were isolated from surface-disinfested, symptomless cucumber roots grown in Nunn sandy loam soil (9). Sal 15 was isolated from cucumber roots grown in Metz fine sandy loam from the Salinas Valley of California (17). Isolate Fo47, isolated from the Chateaurenard region of France, was obtained from C. Alabouvette (Institut National de la Recherche Agronomique, Station de Recherches sur la Flore Pathogène dans le Sol, Dijon, France). The last two isolates were also nonpathogenic to cucumber. Isolates C1, C5, and C10 induced suppressiveness to Fusarium wilt of cucumber when introduced into previously conducive soil; however, isolates C14, Sal 15, and Fo47 did not (14). All isolates were single-spored and stored in twice-autoclaved soil. All isolates were recovered from soil storage and cultured once before being used in the experiments.

Soil-chlamydospore inoculum of each isolate was prepared as follows. Nunn sandy loam (1 kg) was amended with 1% (w/w) ground rolled oats (Quaker Oats Co., Chicago), moistened to 15% (w/w), placed in 2-L Mason jars, and autoclaved for 1 hr on two consecutive days. A microconidial suspension (5 ml) of each isolate was aseptically transferred to separate soil jars. The jars were incubated for 3 wk and shaken twice weekly. After 3 wk the soil-chlamydospore inoculum was air-dried, and the number of colony-forming units (cfu) per gram was determined by a series of 10-fold dilutions onto Nash and Snyder medium (13) amended with chloramphenicol (300 μg/ml). Examination of the inoculum with fluorescence microscopy (19) showed that the inoculum primarily consisted of chlamydospores.

**Effect of nonpathogenic isolates of *F. oxysporum* and strains of *P. putida* on Fusarium wilt of cucumber.** Soil-chlamydospore inoculum of *F. o.* f. sp. *cucumerinum* and the nonpathogenic isolates of *F. oxysporum* were mixed together with 2 kg of Nunn sandy loam for 10 min in a twin-shell blender, to give a final inoculum density of 1,000 cfu/g for *F. o.* f. sp. *cucumerinum* and 10,000 cfu/g for the nonpathogenic isolates. The soil-inoculum mixture was moistened to 15% (w/w) (−0.1 bar) with 0.01 M $MgSO_4$ or a bacterial suspension of strain A12 or N1R in 0.01 M $MgSO_4$ ($10^8$ cells per gram of soil). Soil containing *F. o.* f. sp. *cucumerinum* at 1,000 cfu/g served as the infested control, and soil

with no added *F. oxysporum* or *P. putida* served as the uninfested control. The soil treatments were placed in 10-cm-diameter pots, with four replicate pots per treatment. The pots were seeded with 10 cucumber seeds each (*C. sativus* 'Marketer Long'), and seedlings were thinned to five per pot after 1 wk. The pots were arranged on the greenhouse bench in a randomized complete block design. The plants were watered twice daily with tap water and once a week with a 1:200 solution of Peters general-purpose 20-20-20 solution (W. R. Grace & Co., Fogelsville, PA). Daytime soil temperatures ranged from 25 to 30 C, and nighttime temperatures from 20 to 25 C. The plants were observed for 40 days, and the incidence of wilt was recorded every 2 days. Disease incidence data were transformed to $\log_e 1/(1 - y)$. Linear regression lines were constructed for the transformed data in each treatment and statistically analyzed to determine statistical differences in regression coefficients. The time required to reach 50% disease in each treatment was determined from the regression equations.

**Effect of soil pH on interactions between ineffective isolates of *F. oxysporum* and *P. putida* in biocontrol of Fusarium wilt of cucumber.** Soil pH was measured by use of the $CaCl_2$ method of Schofield and Taylor (20). The pH of raw soil (pH 6.7) was lowered by mixing it with 25% acidified soil (w/w). This acidified soil (pH 2.5) was prepared by mixing 10% 1 N $H_2SO_4$ (w/w) with air-dried raw soil. The acidified soil then was air-dried and mixed with raw soil in a twin-shell blender for 10 min. The final pH of this soil was 5.5. The pH of raw soil was raised by mixing it with hydrated lime ($Ca(OH)_2$) (0.05%, w/w) in a twin-shell blender for 10 min. The final pH of this soil was 8.1. Preliminary tests showed that the pH of these adjusted soils was stable after they were moistened and incubated for 4 wk. Six subtreatments were made at each soil pH: an infested control (*F. o.* f. sp. *cucumerinum* at 3,000 cfu/g of soil), an uninfested control, strain A12 ($10^8$ cells per gram of soil), strain N1R ($10^8$ cells per gram of soil), nonpathogenic isolate Sal 15 of *F. oxysporum* (10,000 cfu/g of soil), and A12 plus Sal 15. Disease incidence was observed every other day for 30 days.

**Germination of chlamydospores of *F. oxysporum* in the rhizosphere of cucumber treated with *P. putida*.** Soil-chlamydospore inoculum ($5 \times 10^5$ to $1 \times 10^6$ cfu/g) of isolates *F. o.* f. sp. *cucumerinum*, C1, C5, C10, C14, Sal 15, and Fo47 was placed into wells (3 g of inoculum mix per well) of multiwell tissue culture plates (Becton Dickinson & Co., Oxnard, CA) and moistened to −0.1 bar. The percent moisture corresponding to −0.1 bar was determined from a moisture-retention curve (wetting) previously constructed for Nunn sandy loam. Cucumber seeds of the cultivar Marketer Long were incubated on moist filter paper for 24 hr. When the radicle had emerged 3–5 mm, they were dipped in a bacterial suspension ($10^8$ cells per milliliter) of either strain A12 or strain N1R. The germinated seeds were placed on top of the soil-chlamydospore mixture in the tissue-culture wells (one seed per well). After 24 hr, the roots were carefully removed, and the excess soil was shaken off. Each root was placed in 1 ml of sterile distilled water and agitated in a vortex mixer for 30 sec to remove the rhizosphere soil. The supernatant and root were decanted off, and 1 ml of 1% calcofluor white M2R solution (Polysciences, Inc., Warrington, PA) was added to the tube. After 2 min the calcofluor was decanted and replaced with sterile distilled water. Soil smears were made from each sample and viewed with a fluorescence ultraviolet microscope (19). The number of germinated and ungerminated chlamydospores on each slide was recorded, and the percent germination determined. From each sample, 100 chlamydospores were counted, and each treatment was replicated five times.

**Effect of soil pH and pathogenic and nonpathogenic isolates of *F. oxysporum* on population densities of *P. putida* in the rhizosphere of cucumber root tips.** Cucumber seeds were treated with either strain N1R or strain A12 by being dipped in a bacterial suspension of $10^9$ cells per milliliter in 1% methyl cellulose for 5 min; they were then placed on a paper towel and air-dried for 1 hr. Three soil treatments were prepared: raw soil, raw soil plus *F. o.* f. sp. *cucumerinum* (3,000 cfu/g), and raw soil plus *F. o.* f. sp. *cucumerinum* (3,000 cfu/g) plus Sal 15 (10,000 cfu/g). Each of these treatments was prepared in soils of pH 5.5, 6.7, and 8.1 (pH was adjusted as previously described). Soil-chlamydospore inoculum of the two isolates of *F. oxysporum* was mixed with the pH-adjusted soils for 10 min in a twin-shell blender, and the infested soil was moistened to −0.1 bar. Polypropylene centrifuge tubes (28.6 × 103.6 mm) were cut in half longitudinally, and each half was filled with the moistened soil. The inoculated cucumber seed was placed on one half-tube, 2 cm below the rim. The unseeded half was then placed on the seeded half, and the two halves were bound together with rubber bands. Six tubes were placed vertically in 105 × 105 × 95 mm plastic pots, and the space surrounding the tubes was filled with soil of the same moisture content and pH. The pots were covered with plastic bags, to maintain constant matric potential, and placed under constant illumination supplied by 10 white, 40-W, 120-cm-long fluorescent lamps. After 7 days, the tubes were removed from the pots. The unseeded half of the tube was lifted, and the root in the seeded half was cut into 1-cm segments, from the crown to the tip, with a sterile scalpel. After loosely adhering soil was shaken off, the last centimeter of root (the root tip) with the adhering rhizosphere soil was weighed and transferred to a test tube containing 1 ml of sterile distilled water. The test tubes were agitated vigorously with a vortex mixer. The number of colony-forming units of each bacterial strain in the rhizosphere soil at the root tip was determined by plating a series of 10-fold dilutions on King's B medium containing rifampicin (100 μg/ml). The root tip was removed from the dilution test tube, blotted on a paper towel, and weighed to determine the weight of the rhizosphere soil removed by washing. This experiment was performed twice, and the results pooled for analysis.

**Experimental design and statistical analyses.** All experiments were arranged in a randomized block design. There were four replications in the greenhouse experiments and at least five replications in all other experiments. Appropriate statistical analyses were applied with separations at $P = 0.05$. All experiments were performed twice. Data from repeated experiments were pooled in cases where variances between the trials were homogeneous.

## RESULTS

**Effect of nonpathogenic isolates of *F. oxysporum* and strains of *P. putida* on Fusarium wilt of cucumber.** In the absence of *P. putida*, only isolates C1, C5, and C10 of *F. oxysporum* significantly reduced Fusarium wilt of cucumber in soil of pH 6.7 (Fig. 1). These isolates increased the time required to reach 50% disease incidence by 2.9–6.1 days, compared with the time required in the infested control. Isolates C14, Sal 15, and Fo47 did not significantly reduce disease. However, when these ineffective isolates of *F. oxysporum* were combined individually with *P. putida* strain A12, disease development was significantly delayed. In these treatments, the time required to reach 50% disease incidence was delayed by 3.1–18.6 days, compared with the time required in the infested control. *P. putida* strain A12 alone did not reduce disease. *P. putida* strain N1R alone and in combination with nonpathogenic isolates of *F. oxysporum* induced significant suppressiveness, compared with the infested control. *P. putida* strain N1R delayed the time required to reach 50% disease incidence by 10 days. Even so, the combination of strain N1R with isolate C10 or C14 resulted in significant increases in disease suppression, compared with N1R alone.

**Effect of soil pH on interactions between ineffective isolates of *F. oxysporum* and *P. putida* in biocontrol of Fusarium wilt of cucumber.** In soil of pH 5.5, the addition of *P. putida* strain A12 or N1R, nonpathogenic isolate Sal 15, or A12 plus Sal 15 did not result in significant suppression of Fusarium wilt of cucumber, in comparison with the infested control (Fig. 2A). The onset of symptoms was delayed, in comparison with other treatments, by the addition of strain N1R or strain A12 plus Sal 15 to soil of pH 6.7. After 30 days, disease incidence in the treatment with A12 plus Sal 15 was significantly different from that in the infested control. At pH 8.1, a significant increase in incubation period, compared with that of the infested control, was observed when N1R or A12

alone or combined with Sal 15 was added to soil. At 30 days, however, disease incidence was reduced significantly only by the addition of N1R or A12 plus Sal 15, in comparison with the other treatments.

**Germination of chlamydospores of *F. oxysporum* in the rhizosphere of cucumber treated with *P. putida*.** Strains A12 and N1R of *P. putida* significantly reduced the germination of chlamydospores of all isolates of *F. oxysporum* in rhizosphere soil of pH 6.7, except for C14 (Table 1). The germination of this isolate was low (13%) in the untreated control. Even though A12 and N1R reduced the germination of isolate C14 by 38 and 85%, respectively, in comparison with the germination in the control, the differences were not significant.

**Effect of soil pH and pathogenic and nonpathogenic isolates of *F. oxysporum* on population densities of *P. putida* in the rhizosphere of cucumber root tips.** When *P. putida* strain N1R or A12 was applied to seeds, the addition of *F. o.* f. sp. *cucumerinum* did not increase the population densities of these strains in soil of pH 6.7 or 8.1, in comparison with untreated controls, although there was a slight tendency toward higher population densities in these treatments (Fig. 3). At pH 5.5, the population density of strain A 12 was significantly lower at the root tip in the presence of *F. o.* f. sp. *cucumerinum*, in comparison with the control in which the pathogen was not added. The addition of *F. o.* f. sp. *cucumerinum* with Sal 15 tended to increase the population densities of both N1R and A12 at the root tips. The differences were greatest and were statistically significant only at pH 8.1. In these cases, the population densities of the strains were 5.3 to 17 times greater than in other treatments.

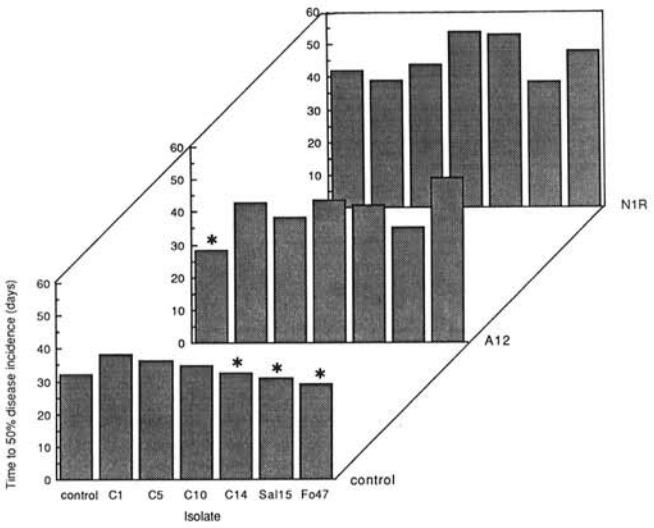


**Fig. 1.** Biological control of Fusarium wilt of cucumber with nonpathogenic isolates C1, C5, C10, C14, Sal 15, and Fo47 of *Fusarium oxysporum* alone and in combination with *Pseudomonas putida* strains A12 and N1R in soil of pH 6.7. *F. o.* f. sp. *cucumerinum* was added at 1,000 colony-forming units (cfu) per gram of soil, and the nonpathogenic isolates of *F. oxysporum* added at 10,000 cfu/g of soil. The bacteria were added at $10^8$ cfu/g of soil. All treatments are significantly different from the infested control ($P = 0.05$) except for those marked with an asterisk.

TABLE 1. Germination of chlamydospores (%) of isolates of *Fusarium oxysporum* in rhizospheres of cucumber infested with strains of *Pseudomonas putida*

| Treatment | Isolates of *F. oxysporum* | | | | | | |
|---|---|---|---|---|---|---|---|
| | C1 | C5 | C10 | C14 | Sal 15 | Fo47 | *Foc*[y] |
| Control | 66 a[z] | 28 a | 55 a | 13 a | 52 a | 54 a | 75 a |
| *P. putida* strain A12 | 22 b | 12 b | 16 b | 8 a | 10 b | 16 b | 10 b |
| *P. putida* strain N1R | 36 ab | 8 b | 10 b | 2 a | 15 b | 12 b | 13 b |

[y] *Foc* = *F. o.* f. sp. *cucumerinum*.
[z] Numbers within each column followed by the same letter are not significantly different ($P = 0.05$).

## DISCUSSION

Some nonpathogenic isolates of *F. oxysporum* induced suppressiveness to Fusarium wilt of cucumber when added to soil, whereas others did not (Fig. 1). The addition of strain N1R of *P. putida* induced suppressiveness in soil of pH 6.7, but strain A12 did not, which confirmed trends observed previously (C.-S. Park, *unpublished*). When three isolates of *F. oxysporum* (C14, Sal 15, and Fo47), ineffective in inducing suppressiveness themselves, were added together with A12, significant suppression of Fusarium wilt resulted. Thus, bacteria and *F. oxysporum*, ineffective by themselves, induced suppressiveness in combinations.

The profound effects of soil pH on the ability of fluorescent pseudomonads to induce suppressiveness to Fusarium wilt

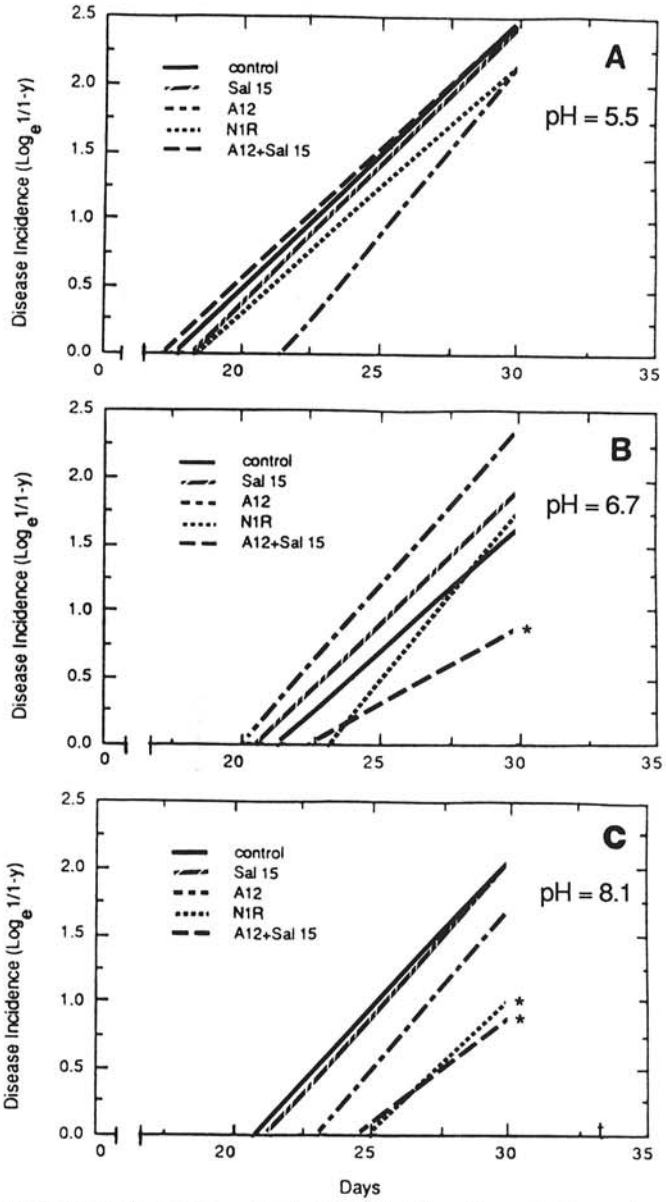


**Fig. 2.** Effect of soil pH on biological control of Fusarium wilt of cucumber with two strains of *Pseudomonas putida* (A12 and N1R) and a nonpathogenic isolate of *Fusarium oxysporum* (Sal 15). **A,** pH 5.5; **B,** pH 6.7; **C,** pH 8.1. *F. o.* f. sp. *cucumerinum* was added at 3,000 colony-forming units (cfu) per gram of soil, and Sal 15 added at 10,000 cfu/g of soil. The bacteria were added at $10^8$ cfu/g of soil. The *Fusarium* isolates and the bacteria were added at the beginning of the experiment. Treatments marked with an asterisk are significantly different from the infested control at $P = 0.05$.

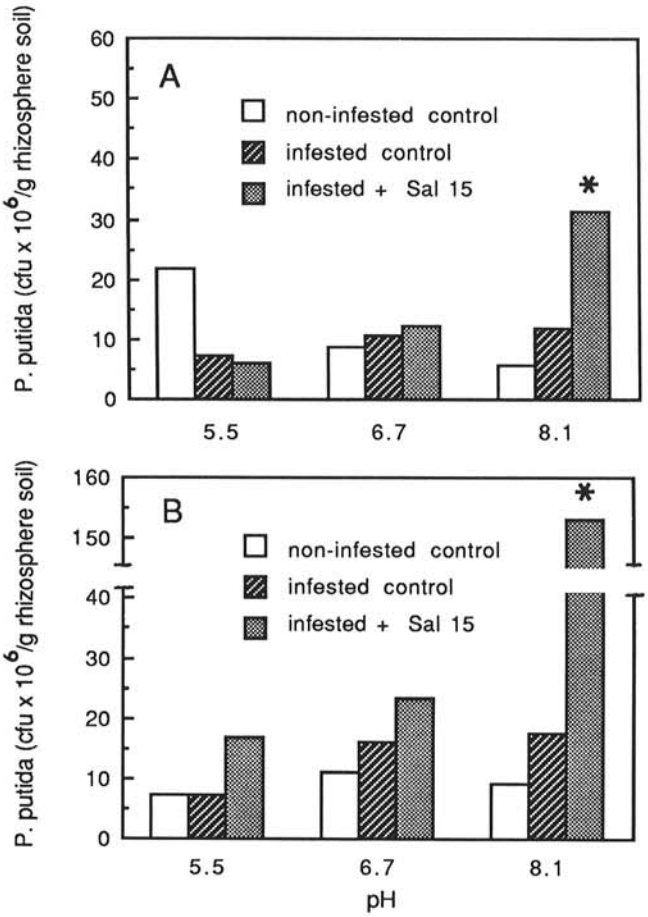


**Fig. 3.** Effect of soil pH and isolates of *Fusarium oxysporum* on the population density of *Pseudomonas putida* strains A12 (**A**) and N1R (**B**) in the rhizosphere of root tips of cucumber. Three treatments were used: raw soil (uninfested control), raw soil infested with *F. o.* f. sp. *cucumerinum* at 3,000 colony-forming units per gram of soil (infested control), and soil infested with *F. o.* f. sp. *cucumerinum* plus a nonpathogenic isolate of *F. oxysporum* (Sal 15), added at 10,000 colony-forming units per gram. Cucumber seeds were treated with a bacterial suspension ($10^9$ cells per milliliter) of *P. putida* strain A12 or strain N1R. Treatments marked with an asterisk are significantly different from the uninfested control at $P = 0.05$.

diseases is well documented (2,3,7,17,18). Thus, in this study, no significant biocontrol occurred when either strain N1R or strain A12 was added to soil of pH 5.5 (Fig. 2). At pH 6.7, strain N1R significantly delayed the onset of symptoms of Fusarium wilt of cucumber, but there was no difference in disease incidence, compared with that of the infested control, after 30 days. Neither the *P. putida* strain A12 nor the nonpathogenic isolate Sal 15, added alone, induced suppressiveness, but when added together, they effectively reduced disease incidence. At pH 8.1, *P. putida* strain N1R and A12 plus Sal 15 induced suppressiveness. Therefore, as the pH of soils was increased, suppressiveness induced by *P. putida* strain N1R or strain A12 with Sal 15 became more pronounced. Such biocontrol phenomena associated with soil pH have been explained by the mechanism of iron competition (2,3,7,18,22).

The reason or reasons why two relatively inefficient biocontrol organisms (*P. putida* A12 and *F. oxysporum* Sal 15) have enhanced biocontrol activity when combined are not completely understood. However, several possibilities exist.

*P. putida* strains N1R and A12 reduced the germination of chlamydospores of pathogenic and nonpathogenic isolates of *F. oxysporum* in rhizospheres of cucumber (Table 1). Such reductions in chlamydospore germination have been directly correlated with the level of nutrients in root exudates of various plants and, in turn, related to enhanced production of siderophores by pseudomonads at higher carbon levels (7). Thus, one hypothesis to explain the phenomena reported here is that the addition of nonpathogenic strains of *F. oxysporum* increased root exudation, thereby enhancing the activity of added fluorescent pseudomonads in the rhizosphere.

Parasitic microorganisms and viruses induce increases in root exudations (5,11,21). The soil microbiota induce similar increases (4), and such responses may be due, at least in part, to alterations in the permeability of membranes (23) in root cells. This may explain why nonpathogenic isolates of *F. oxysporum* added to alkaline soil at a relatively high population density (10,000 cfu/g of soil) induced significantly higher population densities of strains A12 and N1R in the rhizosphere than when the fungus was not added (Fig. 3).

"The impact of interaction of microbial populations in the root-soil interface on prepenetration activity, infection and resulting disease is a critical area that has received relatively little attention . . . [and] the interactions that occur are of major significance" (12). Appropriate systems to study such interactions are difficult to identify. Further, technical difficulties connected with methodology complicate quantitative measurements. The biocontrol system described in this paper may be an appropriate tool for definitive description of such interactions in microbial populations.

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