Induction of Systemic Resistance to Anthracnose in Cucumber by Phosphates

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ABSTRACT

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Solutions of K_3PO_4, K_2HPO_4, NA_3PO_4, and NA_2HPO_4 sprayed on the undersides of the first and second true leaves of cucumber induced systemic resistance in leaves 3 and 4 to anthracnose caused by *Colletotrichum lagenarium*. Solutions of KH_2PO_4, NAH_2PO_4, $CaHPO_4$, $(NH_4)_2HPO_4$, and $NH_4H_2PO_4$ were less active, and a suspension of Ca_3PO_4 was inactive. Induced resistance in leaves 3 and 4 depended on the concentration of K_3PO_4 applied to leaves 1 and 2. Spraying leaves 1 and 2 each with 1–2 ml of a solution of K_3PO_4 at concentrations of 100, 50, 10, 5, and 1 mM protected leaves 3 and 4 99, 96, 78, 54, and 15%, respectively. The level of protection was based on the total necrotic lesion area of plants sprayed on leaves 1 and 2 with water. A pH greater than 7.0 was required for high activity of potassium phosphates, and activity of di- and tripotassium phosphates was markedly reduced at lower pHs. Induction of systemic resistance, however, was not solely a result of an alkaline pH, because 50 mM potassium hydroxide (pH 11.7) was inactive. Induced systemic resistance was associated with the gradual appearance of chlorotic and necrotic stippling on leaves 1 and 2. The lack of stippling or rapid death of leaves 1 and 2 was associated with little or no induced systemic resistance. Induced systemic resistance in newly developing leaves above leaves 3 and 4 was apparent for at least 5 wk in greenhouse and outdoor tests. These data suggest that induced systemic resistance to disease caused by infection is not due to a specific component of the pathogen, but rather to the persistence of a low level of metabolic perturbation. One cause of such perturbation may be the sequestering of calcium ions.

Additional keyword: Cucumis sativus L.

Plants have evolved highly effective mechanisms for resistance to disease caused by infectious agents. Recent literature supports the contention that these mechanisms are multicomponent, layered, and coordinated (2–10). The selection for increased crop yield and quality, enhanced use of fertilizers and pesticides, irrigation, sequential cropping, the appearance of new virulent races of pathogens, and environmental stress can modify plant disease resistance.

Evidence has been reported, however, that susceptibility is not synonymous with the absence of the genetic potential for resistance mechanisms (2–10). Resistance can be systemically induced in apparently susceptible plants by inoculation with avirulent forms of pathogens, hypovirulent pathogens, and nonpathogens, or by restricted inoculation with pathogens (2–10). The induced resistance is persistent and generally nonspecific for a pathogen (2, 4–10). Systemic resistance can be induced by simple chemical substances as well as by biotic agents (4). It appears that systemic induced resistance depends on the gradual expression and persistence of a low level of metabolic perturbation. Unlike elicitors of phytoalexin accumulation, which elicit at the site of application and may be responsible for localized protection (11), inducers of systemic resistance sensitize the plant to respond rapidly after infections. These responses include phytoalexin accumulation and lignification (2,6–10) and enhanced activities of chitinase and β-glucanase (1,12). This paper reports the use of phosphate salts to induce systemic resistance against a fungal disease in cucumber.

MATERIALS AND METHODS

Pathogen and host. *Colletotrichum lagenarium* (Pass) Ell. & Halst. race 1 was maintained on green bean juice agar (215 ml of fluid from canned green beans, 285 ml of water, and 10 g of agar) at 21 C in the dark and transferred weekly. Seven-day-old cultures were used for the preparation of spore suspensions (5).

Cucumber (*Cucumis sativus* L.) cultivar Wisconsin SMR-58 was used in all experiments. Plants were grown in plastic pots (10 cm diameter) containing a 1:1 mixture of Canadian sphagnum peat moss and vermiculite supplemented with nutrients (3). A liquid fertilizer (Peter's 15-16-17, W. R. Grace and Co., Fogelsville, PA), containing 110 ppm nitrogen, was applied to the water, beginning when the first true leaf was fully open. Plants were grown at 23–33 C in a glass greenhouse receiving air filtered through activated charcoal during the summer. During the late autumn, winter, and early spring, sunlight was supplemented with high-pressure sodium lights to maintain approximately a 14-hr photoperiod.

For tests of the persistence of induced resistance, cucumber seeds were either sown in the greenhouse as described or directly into 10-gal plastic pots containing a synthetic soil mix (Metromix-360, W. R. Grace and Co., Fogelsville, PA). The 10-gal plastic pots were arranged along a wire fence on a grass lawn outside of the greenhouse area. As the plants developed, they were trained to the fence. Plants growing outdoors were fertilized as described for greenhouse-grown plants. When plants reached the appropriate stage in the greenhouse and outdoors, they were inoculated or treated as subsequently described on leaves 1 and 2. Half of the greenhouse-grown plants were transferred to 10-gal plastic pots 1 wk after inoculation or treatment. As plants developed in the greenhouse, they were trained to suspended wires.

Treatments with chemicals. Plants (18–21 days old) with the first true leaf fully expanded and the second true leaf approximately two-thirds expanded (leaves 1 and 2) were used in all experiments. An aqueous solution or suspension of the chemical was prepared and the pH determined. The lower surfaces of leaves 1 and 2 were sprayed with 1–2 ml of the solution or suspension (50 mM unless stated otherwise) or water. To avoid contamination, other parts of the plant were covered. The treated plants were grown under greenhouse or outdoor conditions until challenged 7 days after treatment (unless stated otherwise) with a conidial suspension of *C. lagenarium*. In experiments to determine the duration of induced systemic resistance in the greenhouse and outdoors, 50 mM K_2HPO_4 solutions were sprayed weekly on the two youngest fully expanded leaves of some plants beginning 3 wk after spraying leaves 1 and 2 (booster treatment).

Inoculations. Systemic resistance was induced by inoculating

the upper surfaces of leaves 1 and 2 with 30 5-μl drops of a conidial suspension (10^6 spores per milliliter) of *C. lagenarium*. At the time of inoculation, the first leaf was fully expanded and the second leaf was approximately two-thirds expanded. Plants were challenged 7 days after induction by inoculating the upper surfaces of leaves 3 and 4 with 30 5-μl drops of a conidial suspension (10^4 spores per milliliter). After inoculations, plants were placed in a dark closed humidity chamber (~100% RH) held at 22–25 C. After 24 hr, the chamber was partially opened, and after 48 hr the plants were transferred to the greenhouse. Plants receiving different treatments were randomly distributed in the humidity chamber.

In experiments to determine the duration of induced systemic resistance in the greenhouse and field, plants were induced by infiltrating 10 μl of a conidial suspension (10^6 spores per milliliter) into 30 locations on the upper surfaces of both leaves 1 and 2 with a 500-μl Eppendorf pipette. Placing infiltrated plants into humidity chambers was not necessary for the development of symptoms. In tests of the duration of induced systemic resistance, excised leaf halves with the midvein attached were challenged. The excised leaf halves were placed in plastic boxes containing moistened filter paper on the bottom of the box and were inoculated on the upper surfaces with 20 5-μl drops of a conidial suspension (0.5×10^4 spores per milliliter).

Assessment of induced resistance. Induced systemic resistance was determined by counting the number of lesions, measuring the diameter of lesions, and calculating the area of lesions. The level of induced resistance was relative to symptoms on control plants treated with water. Plants induced with *C. lagenarium* or treated with potassium oxalate often served as positive controls to confirm that conditions for inducing systemic resistance were satisfactory. Lesions appeared 3–4 days after induction with *C. lagenarium* or challenge. Seven days after challenge, the maximum lesion development was usually evident, and data obtained at this time interval are reported in this paper. Lesions first appeared chlorotic, and by 4 or 5 days they developed a necrotic center. By 7 days after inoculation, the necrotic center usually filled >90% of the lesion area. The diameter of necrosis was measured since it was sharply delineated, whereas the chlorotic ring around the necrosis was thin and difficult to determine.

TABLE 1. Induction of systemic resistance in cucumber plants by foliar application of potassium salts that form insoluble calcium salts

Treatment[a]	Leaf 3[b] Necrotic lesions (no.)	Leaf 4[b] Necrotic lesions (no.)
H_2O	20.8 ± 2.6[c]	22.2 ± 2.9
Colletotrichum lagenarium	2.7 ± 1.6	7.0 ± 1.5
$K_2C_2O_4$	10.2 ± 2.8	9.4 ± 2.8
K_2CO_3	18.4 ± 4.0	16.2 ± 4.6
K_2SO_4	25.2 ± 1.8	23.0 ± 3.3
K_3PO_4	7.4 ± 2.5	5.8 ± 1.3

[a] Approximately 1–2 ml of each salt (50 mM) was sprayed on the undersides of the first two true leaves (leaves 1 and 2). As positive controls, leaves 1 and 2 were each inoculated on the upper surface with 30 5-μl drops of a conidial suspension of *C. lagenarium* (10^6 spores per milliliter) or sprayed on the lower surface with potassium oxalate, as described. As an additional control treatment, water was sprayed on the undersides of leaves 1 and 2. Six or 7 days after the treatment of leaves 1 and 2, plants were challenged on leaves 3 and 4 with 30 5-μl drops of a conidial suspension of *C. lagenarium* (10^4 spores per milliliter).

[b] The data represent the means of six plants per treatment per experiment, and the experiment was repeated five times.

[c] ± Standard error.

RESULTS

Effect of different potassium salts. Spraying solutions of potassium oxalate or K_3PO_4 on leaves 1 and 2 systemically induced resistance to anthracnose in leaves 3 and 4 (Table 1). Little or no induced resistance was observed in leaves 3 and 4 after spraying leaves 1 and 2 with K_2CO_3 or K_2SO_4.

Treatment with potassium oxalate caused a chlorotic stippling on leaves 1 and 2. The stippling often became necrotic and coalesced if solutions were applied at temperatures >30 C. White, water-insoluble crystals were formed on the lower leaf surface at the site of chlorotic or necrotic symptoms. Less damage was observed on leaves sprayed with K_3PO_4 and the least on leaves sprayed with K_2SO_4. The stippling seldom became necrotic on leaves sprayed with K_3PO_4, and water-insoluble crystals were associated with symptoms. Damage and crystal formation were not evident after treatment with K_2SO_4.

Effect of concentration of K_3PO_4 on induced resistance. A solution of K_3PO_4 was sprayed on leaves 1 and 2 at concentrations ranging from 100 to 1 mM. A positive correlation was evident between the amount of salt applied and the efficacy of induced systemic resistance (Table 2). The experiment was performed with two controls, water and potassium oxalate. The latter compound was previously reported (4) as an inducer of resistance.

A significant decrease in the total necrotic lesion area was observed at 100–5 mM K_3PO_4 based on the symptoms apparent on water controls. At a concentration of 50 mM, K_3PO_4 was a more effective inducer of systemic resistance than potassium oxalate. Induced resistance was consistently associated with the appearance of chlorotic stippling or restricted necrosis on inducer leaves. Necrosis, however, was usually more severe, and occasionally coalesced, on the inducer leaves sprayed with potassium oxalate than on leaves sprayed with the other compounds.

Effect of cation sources on induced resistance by phosphates. Solutions or suspensions of KH_2PO_4, K_2HPO_4, K_3PO_4, NaH_2PO_4, Na_2HPO_4, Na_3PO_4, $NH_4H_2PO_4$, $(NH_4)_2HPO_4$, $CaHPO_4$, $Ca_3(PO_4)_2$, and potassium oxalate were sprayed on leaves 1 and 2 and induced resistance was determined in leaves 3 and 4 as described earlier. Potassium oxalate, K_2HPO_4, K_3PO_4, Na_2HPO_4, Na_3PO_4, and $CaHPO_4$ were highly effective inducers of systemic resistance; KH_2PO_4 and NaH_2PO_4 were less effective, and $NH_4H_2PO_4$ and $(NH_4)_2HPO_4$ had little or no effect (Table 3). Solutions of K_2HPO_4, K_3PO_4, Na_2HPO_4, and Na_3PO_4 were more

TABLE 2. Relationship between amount of phosphate applied to foliage and induced systemic resistance to anthracnose in cucumber

Treatment[a]	Concentration (mM)	pH	Leaf 3[b] Necrotic lesions (no.)	Leaf 3[b] Diameter of necrotic lesions (mm)	Leaf 4[b] Necrotic lesions (no.)	Leaf 4[b] Diameter of necrotic lesions (mm)
H_2O		6.2	27.6 ± 0.7[c]	2.6 ± 0.3	21.3 ± 0.5	4.0 ± 0.7
Potassium oxalate	50	7.3	10.2 ± 2.9	1.0 ± 0.5	9.4 ± 2.9	1.6 ± 0.6
K_3PO_4	100	12.3	4.7 ± 1.5	0.4 ± 0.2	4.0 ± 2.0	0.8 ± 0.2
	50	11.7	6.3 ± 2.1	0.8 ± 0.2	5.8 ± 1.7	1.4 ± 0.3
	10	11.3	16.5 ± 2.5	1.4 ± 0.2	16.1 ± 1.0	2.0 ± 0.4
	5	10.3	25.0 ± 1.6	1.6 ± 0.2	19.6 ± 0.9	3.1 ± 0.3
	1	9.4	28.1 ± 0.9	2.4 ± 0.3	20.7 ± 0.6	3.9 ± 0.8

[a] See footnote a, Table 1.

[b] Data are the mean from three experiments with four plants per treatment per experiment.

[c] ± Standard error.

effective than potassium oxalate. Of all the treatments, inoculation with *C. lagenarium* was the most effective inducer of resistance.

Effect of pH on induced resistance. In one series of experiments, the effect of pH of the solution applied on induced systemic resistance was studied with 50 mM dipotassium phosphate at pH values ranging from 3 to 9, along with water, 50 mM potassium chloride, and 50 mM KOH at pH values of 6.1, 6.5, and 11.7, respectively (Table 4). The pH values of K_2HPO_4 solutions were adjusted with HCl to 3.0, 5.0, and 7.0. Induced systemic resistance in leaves 3 and 4 and chlorotic stippling of leaves 1 and 2 were evident with K_2HPO_4 when the pH was at or greater than 7.0. A solution of K_2HPO_4 at pH 3 and 5 caused little or no damage on the inducer leaves and induced little or no resistance. Solutions of KCl and KOH produced little or no stippling on the inducer leaves at the concentrations applied and did not induce systemic resistance.

In a second series of experiments, aqueous solutions of 50 mM KH_2PO_4, K_2HPO_4, K_3PO_4, or potassium oxalate was adjusted to pH 6.0 with either potassium hydroxide or hydrochloric acid. A decrease of the pH for K_2HPO_4 and K_3PO_4 (from 9.0 and 11.7, respectively) resulted in a decrease in their ability to induce systemic resistance (Table 5). The highest level of induced resistance was observed for K_2HPO_4 and K_3PO_4 without adjustment to pH 6.0. A solution of KH_2PO_4 alone or adjusted to pH 6.0 induced little or no resistance. Potassium oxalate at a pH of 6.0 induced resistance. The water sprayed on leaves for the controls had a pH of 6.1.

Duration of induced systemic resistance. Lesions on leaves infiltrated with conidia were indistinguishable from those formed on leaves inoculated with drops on the upper leaf surface, and lesions appeared in 3 or 4 days. Systemic resistance induced by *C. lagenarium* and K_2HPO_4 was evident in newly developed leaves for at least 5 wk after induction of resistance in greenhouse and outdoor tests (Table 6). Plants induced in the greenhouse and grown in the greenhouse were generally more effectively protected than those induced in the greenhouse and transplanted outdoors. Applying a booster inoculation of phosphate during this period did not significantly enhance induced resistance (data not shown).

DISCUSSION

The data reported here support the hypothesis that induced systemic resistance to disease induced by restricted infection is not due to a specific component of the pathogen, but rather to the gradual appearance and persistence of a low level of metabolic perturbation leading to stress in the host (2,4,6–10). The common feature of all microorganisms that induce systemic resistance is their ability to cause gradual and restricted damage to the host. Rapid and extensive damage caused by mechanical injury or treatment with 50 mM hydrochloric or sulfuric acid does not elicit systemic resistance (data not shown). Sufficient living tissue appears necessary to respond to the trauma and produce an "alarm" signal, which is translocated throughout the plant and elicits systemic resistance. Only the inducer leaf is the factory for this signal; the receiver leaves that are uninjured do not produce the signal, although they have had resistance induced (3).

Recently, Doubrava et al (4) demonstrated that induced systemic resistance in cucumber caused by extracts of spinach and rhubarb leaves was caused by oxalates. They suggested that oxalate released an alarm signal by sequestering Ca^{2+} from host

TABLE 3. Effect of cation source on induction by phosphates of systemic resistance to anthracnose in cucumber

Treatment[a]	pH	Leaf 3[b] Necrotic lesions (no.)	Leaf 3[b] Diameter of necrotic lesions (mm)	Leaf 4[b] Necrotic lesions (no.)	Leaf 4[b] Diameter of necrotic lesions (mm)
H_2O	6.2	23.5 ± 1.4[c]	2.3 ± 0.2	21.2 ± 1.6	2.5 ± 0.3
Potassium oxalate	7.3	9.2 ± 2.1	1.1 ± 0.2	11.4 ± 2.6	1.3 ± 0.3
KH_2PO_4	4.3	13.7 ± 2.3	2.6 ± 0.4	17.5 ± 3.7	2.3 ± 0.4
K_2HPO_4	9.0	3.9 ± 1.0	0.6 ± 0.1	3.7 ± 1.1	0.4 ± 0.2
K_3PO_4	11.7	8.5 ± 1.1	1.1 ± 0.4	6.2 ± 1.7	1.0 ± 0.5
NaH_2PO_4	4.8	19.8 ± 1.8	1.9 ± 0.4	14.8 ± 1.8	1.0 ± 0.2
Na_2HPO_4	9.0	1.8 ± 0.6	0.5 ± 0.2	1.0 ± 0.5	0.3 ± 0.2
Na_3PO_4	11.7	1.8 ± 0.7	0.5 ± 0.2	1.5 ± 0.6	0.2 ± 0.1
$NH_4H_2PO_4$	4.4	20.8 ± 2.1	1.5 ± 0.3	26.3 ± 0.5	1.8 ± 0.3
$(NH_4)_2HPO_4$	7.8	22.5 ± 1.8	1.9 ± 0.5	25.5 ± 2.8	1.8 ± 0.2
$CaHPO_4$	7.2	10.4 ± 0.9	1.0 ± 0.1	10.2 ± 1.1	1.0 ± 0.1
$Ca_3(PO_4)_2$	7.3	23.1 ± 1.1	2.2 ± 0.2	22.0 ± 1.7	2.4 ± 0.3
C. lagenarium		2.7 ± 1.4[c]	0[d]	1.7 ± 1.0[d]	0[d]

[a] See footnote a, Table 1.
[b] The data are the mean of five experiments, six plants per treatment per experiment.
[c] ± Standard error.
[d] Only chlorotic lesions 0.5 mm or less in diameter were observed.

TABLE 4. Effect of pH on the ability of solutions of dipotassium phosphate to induce systemic resistance to anthracnose in cucumber

Treatment[a]	pH	Leaf 3[b] Necrotic lesions (no.)	Leaf 3[b] Diameter of necrotic lesions (mm)	Leaf 4[b] Necrotic lesions (no.)	Leaf 4[b] Diameter of necrotic lesions (mm)
H_2O	6.1	26.2 ± 1.4[d]	2.1 ± 0.2	22.7 ± 1.6	1.7 ± 0.2
K_2HPO_4	3.0[c]	25.3 ± 1.6	1.9 ± 0.2	21.5 ± 2.0	1.5 ± 0.2
	5.0[c]	25.7 ± 0.8	1.8 ± 0.3	20.5 ± 1.8	1.1 ± 0.2
	7.0[c]	21.2 ± 0.9	1.3 ± 0.3	15.5 ± 1.8	0.8 ± 0.2
	9.0	6.5 ± 1.1	0.8 ± 0.1	3.7 ± 1.1	0.5 ± 0.1
KCl	6.5	26.2 ± 1.1	2.0 ± 0.3	21.7 ± 1.3	1.4 ± 0.2
KOH	11.7	27.2 ± 1.2	2.8 ± 0.4	20.5 ± 1.6	1.8 ± 0.3

[a] See footnote a, Table 1.
[b] Data are the mean for three experiments, six plants per treatment per experiment.
[c] The pH was adjusted with HCl.
[d] ± Standard error.

TABLE 5. Effect of potassium phosphates adjusted to pH 6.0 on induced systemic resistance to anthracnose in cucumber

Treatment[a]	pH	Leaf 3[b] Necrotic lesions (no.)	Leaf 3[b] Diameter of necrotic lesions (mm)	Leaf 4[b] Necrotic lesions (no.)	Leaf 4[b] Diameter of necrotic lesions (mm)
H_2O	6.1	20.2 ± 2.8[c]	2.1 ± 0.2	19.2 ± 1.6	1.5 ± 0.1
KH_2PO_4	4.3	18.2 ± 1.8	1.8 ± 0.1	18.0 ± 1.4	1.3 ± 0.1
K_2HPO_4	9.0	3.5 ± 0.8	0.5 ± 0.1	3.1 ± 1.0	0.4 ± 0.2
K_3PO_4	11.7	5.2 ± 0.9	0.8 ± 0.3	4.1 ± 0.9	0.9 ± 0.2
Potassium oxalate[d]	6.0	10.0 ± 2.9	1.4 ± 0.2	10.5 ± 1.7	1.1 ± 0.1
KH_2PO_4[e]	6.0	18.8 ± 2.3	2.3 ± 0.5	16.3 ± 3.9	1.1 ± 0.3
K_2HPO_4[d]	6.0	21.2 ± 3.5	1.8 ± 0.1	20.7 ± 4.3	1.1 ± 0.3
K_3PO_4[d]	6.0	18.0 ± 2.1	1.9 ± 0.2	19.8 ± 3.5	1.1 ± 0.2

[a] See footnote a, Table 1.
[b] Data are the mean for three experiments, six plants per treatment per experiment.
[c] ± Standard error.
[d] Adjusted to pH 6.0 with HCl.
[e] Adjusted to pH 6.0 with KOH.

TABLE 6. Duration of systemic resistance to anthracnose in cucumber induced by inoculation with *Colletotrichum lagenarium* or treatment with dipotassium phosphate in greenhouse and outdoor tests

Treatment[a]	Time after induction of resistance (wk)	Percent Protection Greenhouse[b]	Greenhouse-Outdoors[b]	Outdoors[b]
C. lagenarium	1	98	93	90
	3	95	91	89
	4	98	80	86
	5	96	89	85
K_2HPO_4	1	98	74	70
	3	78	69	74
	4	97	90	87
	5	96	89	85

[a] See Materials and Methods section of text for description of method of induction and challenge.
[b] Data reported are for a single experiment, six plants per treatment. The percent protection is based on the area of necrosis of plants treated with water. Data are from plants induced and grown in the greenhouse, induced in the greenhouse and grown outdoors, and induced and grown outdoors.

tissues. Inducer leaves sprayed with extracts of spinach or rhubarb or with potassium oxalate developed chlorotic and necrotic stippling, and this damage was associated with the formation of crystals on the sprayed leaf surface that were water-insoluble but soluble in 2 N hydrochloric acid. It is possible that phosphates act as oxalate in sequestering Ca^{2+} from host tissues. As reported by Doubrava et al (4), we observed the stippling of inducer leaves and appearance of water-insoluble crystals associated with the stippling when systemic resistance was induced by phosphates. The sequestering may affect membranes, destroy cell compartmentalization, and cause the release or synthesis of hydrolytic enzymes. These enzymes may, in turn, act on plant cell walls, which include pectic substances that have been rendered more susceptible to hydrolytic plant enzymes by the removal of Ca^{2+}. The oligosaccharides or oligogalacturonates formed may function as the alarm signal or cause its release. The sequestering of Ca^{2+}, however, may be just one of many ways to elicit production of the alarm signal.

A pH above 7 markedly enhanced the activity of phosphates, and this may be due to the ability of phosphates to form highly water-insoluble complexes with Ca^{2+} at alkaline pH values or the enhanced activity of HPO_4^{2-} and PO_4^{3-} per se as compared with that of KH_2PO_4. An alkaline pH alone does not induce systemic resistance, since 50 mM potassium hydroxide or sodium hydroxide (data not shown) were not active and elicited little damage on inducer leaves. The lack of damage caused by KOH and NaOH came as a surprise, since acids caused extensive damage.

The duration and effectiveness of systemic resistance induced by phosphates, their low cost, low animal toxicity, nutrient value, and comparative safety for the environment suggest that phosphates may find application in the field for disease control.

LITERATURE CITED

1. Boller, T. 1985. Induction of hydrolases as a defense reaction against pathogens. Pages 247-262 in: Cellular and Molecular Biology of Plant Stress. J. L. Key and T. Kosuge, eds. UCLA Symposium on Molecular and Cellular Biology, New Series, Vol. 22. Alan R. Liss, New York.
2. Dean, R. A., and Kuć, J. 1985. Induced systemic protection in plants. Trends Biotechnol. 3:125-128.
3. Dean, R. A., and Kuć, J. 1986. Induced systemic protection in cucumbers: The source of the "signal." Physiol. Mol. Plant Pathol. 28:227-233.
4. Doubrava, N., Dean, R., and Kuć, J. 1988. Induction of systemic resistance to anthracnose caused by *Colletotrichum lagenarium* in cucumber by oxalates and extracts from spinach and rhubarb leaves. Physiol. Mol. Plant Pathol. 33:69-79.
5. Hammerschmidt, R., Acres, S., and Kuć, J. 1976. Protection of cucumber against *Colletotrichum lagenarium* and *Cladosporium cucumerinum*. Phytopathology 66:790-794.
6. Kuć, J. 1982. Induced immunity to plant disease. BioScience 32:854-860.
7. Kuć, J. 1985. Expression of latent genetic information for disease resistance in plants. Pages 100-118 in: Cellular and Molecular Biology and Development of Adaption. Ciba Foundation 102. Pitman, London.
8. Kuć, J. 1987. Plant immunization and its applicability for disease control. Pages 255-274 in: Innovative Approaches to Plant Disease Control. I. Chet, ed. J. Wiley & Sons, Inc., New York.
9. Kuć, J., and Preisig, C. 1984. Fungal regulation of disease resistance mechanisms in plants. Mycologia 76:767-784.
10. Kuć, J., and Rush, J. 1985. Phytoalexins. Arch. Biochem. Biophys. 236:379-389.
11. Lancake, P. 1981. Alternative chemical agents for controlling plant disease. Phil. Trans. R. Soc. London B295:83-101.
12. Metraux, J. P., and Boller, T. 1986. Local and systemic induction of chitinase in cucumber plants in response to fungal, bacterial and viral infections. Physiol. Mol. Plant Pathol. 28:161-169.