Sodium Silicate Reduces Postharvest Decay on Hami Melons: Induced Resistance and Fungistatic Effects

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ABSTRACT

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The effect of sodium silicate (Si) for control of decay was tested in Hami melons (*Cucumis melo* L. var. *inodorus* Jacq.). Si significantly inhibited mycelial growth of *Alternaria alternata*, *Fusarium semitectum*, and *Trichothecium roseum* in vitro. Si at 100 mM was more effective than Si at 25 or 50 mM at controlling the diseases caused by the three pathogens, whereas Si at 200 mM was phytotoxic. Si treatments applied at 100 mM pre-inoculation with *T. roseum* had lower decay incidence and severity than treatments applied post-inoculation. The protection of Si was correlated with the activation of two families of defense-related enzymes, peroxidase and chitinase. Accumulation of both enzymes was induced in fruit treated with Si and challenged by *T. roseum* 24 h later, and was sustained for at least 9 days in 'New Queen' and 10 days in '8601' at room temperature. It appeared that induced resistance was an important mechanism of disease control in Hami melons treated with Si.

Alternaria, *Fusarium*, and pink rots caused by *Alternaria alternata*, *Fusarium* spp., and *Trichothecium roseum*, respectively, account for most of the postharvest losses of Hami melons (5). These pathogens are controlled primarily by chemical fungicides (1,22). However, due to potential problems related to public health associated with pesticide use, development of fungicide resistance by pathogens, and potential harmful effects on the environment, as well as the desire to reduce losses while also reducing the use of fungicides, new strategies for control have been proposed (16).

Silicon is the second most abundant atom in the Earth's crust (37). Biologically, it is a common but generally minor element in the majority of living organisms, occurring as amorphous silica ($\text{SiO}_2 \cdot n\text{H}_2\text{O}$) and soluble silicic acid ($\text{Si}(\text{OH})_4$) (29). There are several hypotheses concerning the role of silicon in plants, including a positive effect on reproduction, alleviation of metal toxicity and nutrient imbalance, provision of structural rigidity, and increased resistance to fungal diseases such as powdery mildews and root rots (4,17). Soluble silicon has shown potential for the

control of fungal diseases in cherry fruit (27), cucumber (14), muskmelon and zucchini squash (24), peach fruit (8), and pear fruit (32,33). The mechanisms responsible for the protection of plants from fungal diseases by silicon are not well understood. Silicon may act by eliciting biochemical defense reactions, including the accumulation of lignin, phenolic compounds, and pathogenesis-related proteins in the infected plants (14,18).

The objectives of this study were to (i) examine the utility of applying sodium silicate (Si) as an alternative postharvest treatment to conventional fungicides for managing decay in Hami melons, (ii) determine the effects of Si on disease development after either artificial or natural infection with the three major postharvest pathogens, and (iii) investigate whether induced resistance is involved in suppression of fungal decay in melon by Si.

MATERIALS AND METHODS

Plant material. Hami melons (*Cucumis melo* L. var. *inodorus* Jacq.) cvs. New Queen and 8601 were obtained from Huangdun State Farm, Dunhuang, Gansu. The fruit were exposed to natural infection while growing in the field. The major field disease was *Phytophthora* rot (caused by a *Phytophthora* sp.) at this farm. Latent infections in growing fruit involved *Alternaria* rot (caused by *A. alternata*) and *Fusarium* rot (caused by a *Fusarium* sp.). The fruit were harvested at the beginning of physiological maturity (42 to 43 days after full bloom for New Queen and 55 to

56 days after full bloom for 8601), sorted for uniform size and absence of obvious injuries, packed individually in 35-cm-long net bags of foam plastic, and put in the standard melon-shipping boxes (four melons/box). Fruit were transported to the laboratory and stored at room temperature ($22 \pm 2^\circ\text{C}$) with relative humidity (RH) of 55 to 60%.

Source of pathogens and inoculum production. *A. alternata* (Fr.:Fr.) Keissl., *Fusarium semitectum* Berk. et Ravenel, and *Trichothecium roseum* (Pers.:Fr.) Link were isolated from Hami melons with fruit decay and maintained on potato dextrose agar (PDA) at 4°C . Conidia of the pathogens were obtained from 10-day-old PDA cultures incubated at 25°C . To obtain conidia, the cultures were flooded with sterile water containing 0.05 % Tween 20 and rubbed gently with a glass rod. Mycelium was removed from the suspensions by filtering through two layers of cheesecloth. The spore count was determined with a hemacytometer, and the concentration was adjusted to 1×10^5 spores mL^{-1} with distilled water.

Preparation of Si and postharvest treatment. Si (Aldrich, sodium silicate solution, 27% SiO_2) was prepared as 25, 50, 100, and 200 mM solutions with distilled water. Asymptomatic fruit were dipped for 10 min in the various concentrations of Si and air dried. Control fruit were dipped in distilled water. The treated fruit were repacked individually in 35-cm-long net bags of foam plastic, put in standard melon-shipping boxes (four melons/box), and stored at room temperature ($22 \pm 2^\circ\text{C}$, RH 55 to 60%) until subjected to artificial inoculation or natural infection.

In vitro assays. The effect of Si on mycelial growth of the pathogens was assayed using the method of Aharoni et al. (2). Si treatments at 0, 12.5, 25, 50, and 100 mM were tested against *A. alternata*, *F. semitectum*, and *T. roseum*. The substances were added into PDA after the PDA was autoclaved for 15 min and cooled to 65°C . A 5-mm-diameter disc of mycelial mat from a 7-day-old culture was placed in the center of each petri plate. Colony diameter of the fungi was measured daily for a 5-day incubation period at 25°C . Plugs of mycelial mat from Si-amended plates were transferred to PDA plates without Si to

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determine whether the effect of Si was fungistatic or fungicidal. Plates were incubated for 5 days at 25°C and colony diameter was measured. Each treatment contained three replicates of six plates each and the experiment was performed twice.

In vivo assays. Artificial inoculation. The effect of Si on inoculated fruit was assayed using the method of Bi and Zhang (6). Fruit were dipped for 10 min in the various concentrations (25, 50, 100, and 200 mM) of Si and air dried. Control fruit were dipped in distilled water. Treated melons of New Queen and 8601 were sampled from ambient temperature storage (at $22 \pm 2^\circ\text{C}$) 1 day after Si treatment. Nine spots (approximately 1 cm in diameter) were disinfected with a ball of absorbent cotton containing 70% ethanol around the equator of each melon. Wounds were made on the disinfected spots with a sterile needle (3 mm deep by 3 mm in diameter). Spore suspensions ($20 \mu\text{l}$, 1×10^5 spores ml^{-1}) of each of three pathogens (*A. alternata*, *F. semitectum*, or *T. roseum*) were pipetted into each of nine wounds. After drying for approximately 1 h in air, the melons were put in boxes, covered with plastic film to increase RH to 85 to 90%, and kept at $22 \pm 2^\circ\text{C}$. Decay incidence and decay severity were determined 14 (for *A. alternata*), 11 (for *F. semitectum*), and 9 (for *T. roseum*) days after inoculation. Each treatment was applied to three replicates of 12 melons and the experiment was performed three times.

Natural infection. Fruit of cvs. New Queen and 8601, treated with distilled water (control), 50, and 100 mM of Si, were stored for 14 days respectively at $22 \pm 2^\circ\text{C}$ with RH of 85 to 90% (the time period and environment conditions correspond to those occurring during land freight delivery from northwestern to southern China and retail marketing). Fruit quality was evaluated after storage. General appearance was evaluated visually as described by Aharoni et al. (2), for freshness of the fruit, decay, and skin blemishes on a scale of 1 to 5, where 1 = poor, 3 = good, and 5 = excellent quality. Fruit with a rating of less than 2.5 were considered unfit for marketing. Total soluble solids (TSS) were determined by squeezing juice from the central section of flesh directly onto an Abbe refractometer (10481 S/N; Division of Warner-Lambert Technologies, Inc., Keene, NH). Three readings were taken from each melon. Firmness was measured using a handheld penetrometer (FT-327; UC Fruit Firmness Tester, Milano, Italy), equipped with an 8-mm plunger tip, at the equator of fruit where a section of rind (4 by 4 cm and 1 cm deep) had been removed. Three readings were taken for each melon. Fruit were considered to be decayed if there was fungal growth on the surface of the stem end and the rind. Fungi associated with rots were isolated and plated onto PDA. The isolates

were identified and tested for pathogenicity by wounding and applying a spore suspension as described above. Percentage of fruit with decay was determined. Each treatment was applied to three replicates of 20 melons and the experiment was performed twice.

Pre- and post-inoculation effect of Si treatment. The possibilities of fruit fungistatic effect and induced resistance as mechanisms of action of Si were examined with melons which were inoculated 6 and 24 h before or 6 and 24 h after Si treatment at 100 mM. The melons (cvs. New Queen and 8601) were disinfected and inoculated with a suspension of *T. roseum* (1×10^5 spores ml^{-1}) as described above. Fruit were stored and evaluated for decay incidence and severity 9 days after inoculation as described above. Each treatment was applied to three replicate units made up of 12 melons. The experiment was done twice.

Enzyme assays. Fruit treated with Si (100 mM) or water (control) were inoculated with *T. roseum* (1×10^5 spores ml^{-1}) 24 h after treatment. Approximately 2 g of tissue were removed 0.5 to 1.5 cm below the skin and 1 cm from the edge of the wound with a stainless steel cork borer 0, 1, 3, 5, 7, and 9 days after inoculation for New Queen and 0, 2, 4, 6, 8, and 10 days after inoculation for 8601. Each sample was packed, frozen in liquid nitrogen, and kept at -80°C until crude enzyme extraction.

Crude enzyme was extracted according to the method of Venisse et al. (35) with some modifications. Frozen tissue (2 g) was homogenized with a mortar and pestle in 2 ml of cold sodium phosphate buffer (50 mM, pH 7.5) containing 1 mM polyethyleneglycol, 1 mM phenylmethylsulfonyl fluoride, 8% (wt/vol) polyvinylpyrrolidone, and 0.01% Triton X-100. Homogenates were centrifuged at $16,000 \times g$ for 20 min at 4°C , and supernatants were assayed immediately for enzyme activities.

Peroxidase (POD) activity was determined according to the methods of Chance and Maehly (11) with some modifications. Supernatants were diluted in 50 mM sodium phosphate buffer, pH 7.5 (vol/vol, 1:4 for 8601 or 1:3 for New Queen). This diluted extract (50 μl) was added to a 1-ml reaction mixture that contained 50 mM sodium acetate buffer, pH 5.5, 25 mM guaiacol, and 250 mM H_2O_2 . Oxidation of guaiacol to tetraguaiacol was monitored spectrophotometrically at 470 nm for 1 min at 30°C . POD activity was expressed as nM tetraguaiacol produced per minute and per milligram of proteins using a molar extinction coefficient of 26.6 mM/cm .

Chitinase (CHT) activity was assayed according to the method of Wirth and Wolf (36) with some modifications. Carboxymethyl-curdlan-Remazol Brilliant Violet (CM-curdlan-RBV) was used as the respective substrate (Loewe Biochemica, Otterfing, Germany). CM-curdlan-RBV

(100 μl) at 200 $\mu\text{g}/\text{ml}$ was prepared in sodium acetate buffer (200 mM, pH 5), pre-incubated at 35°C for 10 min, and then mixed with 200 μl of crude supernatant. The mixture was incubated at 35°C for 30 min and the reaction immediately stopped by addition of 2N HCl (100 μl). For a reference, the reaction was stopped immediately with HCl after the addition of the substrate. Nondigested substrate was precipitated after 10 min of incubation on ice, then separated from the digested substrate by centrifugation at $10,000 \times g$ for 10 min. CHT activity was calculated from the difference of absorbance at 550 nm between the incubated sample and the reference. Data were expressed as the increase of absorbance per minute and per milligram of protein.

Protein content in the crude enzyme extracts was determined according to the method of Bradford (9) using bovine serum albumin (BSA) as the standard protein.

The enzyme tests were performed with a minimum of three tissue sample replicates per treatment and per time point. Each experiment was repeated at least three times.

Statistical analysis. Data analyses were performed with SPSS software (SPSS Inc., Chicago). Analysis of variance was conducted on decay severity and arcsine-square root transformed data from decay incidence in various trials. Data from separate experiments were combined when statistical analyses determined that variances were homogeneous. Significant differences ($P < 0.05$) between treatments were determined with the least significant difference. Data analysis indicated that treatment effects were similar among the experimental repeats. Results presented were pooled across repeated experiments.

RESULTS

Effect of Si concentration on mycelial growth of the three pathogens in vitro.

The mycelial growth of the three pathogens on PDA significantly decreased with increasing Si concentration (Fig. 1). A concentration of Si at 100 mM completely inhibited the mycelial growth of all pathogens. *T. roseum* was more sensitive to Si than *A. alternata* and *F. semitectum*. After 5 days at 25°C , the mycelial growth of *T. roseum* was reduced by 72% at 25 mM of Si (Fig. 1C) compared with the mycelial growth of *A. alternata* and *F. semitectum* which was reduced by 62 and 40%, respectively (Fig. 1A and B). There was no difference in growth rates when compared with plugs of unamended controls after transfer from Si-amended to PDA without Si (data not shown). This indicated that Si lacked fungicidal effect.

Effect of Si treatment on decay incidence and severity of inoculated melons. Treatment with Si reduced significantly decay incidence and severity of the three

pathogens on inoculated melons (Fig. 2). A concentration of Si at 100 mM provided the most decay control; decay incidence was reduced by 72, 40, and 59% in New Queen and 67, 42, and 48% in 8601 after inoculation with *T. roseum*, *F. semitectum*, and *A. alternata*, respectively (Fig. 2 A and B). The Si treatment at 100 mM had the smallest decay severity, which was decreased by 47, 23, and 35% in New Queen and 46, 19, and 26% in 8601 after inoculation with *T. roseum*, *F. semitectum*, and *A. alternata*, respectively (Fig. 2C and D). Among the three pathogens, Si treatment was most effective against *T. roseum*. A concentration of 200 mM did not inhibit the disease further and resulted in rind injury, which consisted of sunken areas 1 to 3 mm in depth that were light to dark brown in color.

Effect of Si treatments on natural infections and quality of melons. Si treatments were effective in reducing incidence of decay in New Queen and 8601 melons after 14 days at $22 \pm 2^\circ\text{C}$. The treated fruit had a high score for general appearance and were marketable (Table 1). Si treatment at 100 mM provided effective control

of decay, with 61% fewer decayed melons for New Queen and 70% fewer for 8601 compared with nontreated melons. Infection occurred more commonly at the stem end and in cracks and nets on the rind. Approximately 70% of rot was identified as caused by *Fusarium* spp. Other pathogens were *A. alternata*, *Mucor mucedo*, *Rhizopus stolonifer*, and *T. roseum*. The fruit firmness and TSS on New Queen and 8601 melons did not differ between treatments (Table 1).

Pre- and post-inoculation effect of Si treatment. Both New Queen and 8601 melons treated with 100 mM of Si from 24 h before to 6 h after inoculation with *T. roseum* had lower decay incidence and

decay severity than control fruit. Si was less effective when fruit were inoculated before treatment. Lowest decay incidence and smallest decay severity occurred with melons inoculated 24 h after treatment with Si (Fig. 3).

Effect of Si treatment on POD and CHT activities in melons challenged by *T. roseum*. Si treatment at 100 mM caused a more progressive and significant increase in POD and CHT activities on fruit challenged by *T. roseum* (Fig. 4). The maximum POD activity was found 3 days after inoculation in New Queen and 6 days after in 8601, with the activity over 2.4 and 2 times higher in the fruit treated with Si than in the control, respectively (Fig. 4A

Fig. 1. Effect of sodium silicate on mycelial growth of **A**, *Alternaria alternata*, **B**, *Fusarium semitectum*, and **C**, *Trichothecium roseum* in vitro during 5 days of incubation at 25°C . Data were based on three replicates of six plates and the experiment was performed twice. Vertical bars represent the standard error from the means of three separate tests.

Fig. 2. Effect of sodium silicate (Si) treatment on **A** and **B**, decay incidence and **C** and **D**, decay severity in Hami melon cvs. **A** and **C**, New Queen and **B** and **D**, 8601. Fruit were treated with Si at 25, 50, 100, and 200 mM or water (control) for 10 min and inoculated with 20 μl of spore suspensions (1×10^5 spores ml^{-1}) of *Alternaria alternata*, *Fusarium semitectum*, and *Trichothecium roseum* 1 day after treatment. Decay was evaluated 14 (for *A. alternata*), 11 (for *F. semitectum*), and 9 (for *T. roseum*) days after incubation at $22 \pm 2^\circ\text{C}$. Data are averages of three replicates each of 12 fruit for one of three experiments that produced similar results. Bars with the same letters represent values that are not significantly different according to the least significance difference test ($P < 0.05$).

Table 1. Decay incidence and quality of Hami melons 14 days after treatment with sodium silicate (Si)^y

Cultivars, treatment	Decay incidence (%)	General appearance ^z	Firmness (N)	Total soluble solids (%)
New Queen				
Before storage	0	4.5	55	11.7
Control	27 a	2.0 c	37 a	10.5 a
Si at 50 mM	19 b	3.0 b	35 a	10.8 a
Si at 100 mM	11 c	3.5 a	39 a	10.2 a
8601				
Before storage	0	4.5	62	12.4
Control	16 a	3.0 c	56 a	11.0 a
Si at 50 mM	9 b	3.5 b	54	10.6 a
Si at 100 mM	5 b	4.0 b	53 a	11.3 a

^y Data were based on three replicates of 20 fruit each. The experiment was performed twice with similar results and the treatment means were pooled. Values followed by the same letter are not significantly different according to the least significant difference test ($P < 0.05$).

^z General appearance: 1 = poor, 3 = good, and 5 = excellent.

and B). CHT activity reached maximum 5 days after inoculation in New Queen and 6 days after in 8601, which was over 2.2 and 1.6 higher than nontreated fruit in New

Queen and 8601, respectively (Fig.4C and D). The activation of POD and CHT lasted at least 9 days in New Queen and 10 days in 8601.

DISCUSSION

Postharvest treatment with Si proved effective for inhibiting pathogen growth as well as inducing disease resistance in Hami melons, thereby controlling decay and maintaining fruit quality. This is, to our knowledge, the first report linking the beneficial role of Si against decay with an induced defense mechanism in melon. Si at 100 mM was the most effective concentration in reducing decay, whereas a higher Si concentration (200 mM) was phytotoxic, which led to an unacceptable general appearance.

In our study, Si directly inhibited mycelial growth of *A. alternata*, *F. semitectum*, and *T. roseum* in vitro (Fig. 1). A concentration of Si at 100 mM completely inhibited the mycelial growth of the three pathogens. Similar results also were observed with *Penicillium expansum* and *Monilinia fructicola* (8,27). Because the three pathogens grew normally after transfer from Si-amended PDA to PDA without Si, the action of Si was fungistatic rather than fungicidal against these pathogens. A study currently underway has shown that Si caused reduction in cell turgor pressure of *A. alternata*, *F. semitectum*, and *T. roseum*, which resulted in collapse and shrinkage of hyphae and spores and consequent inability of fungi to sporulate in vitro (Y. Bi, data not shown). We suspect that these changes are related to the fungistatic effects of Si on the three pathogens of Hami melons.

Although the effects of soluble Si on diseases of crops have been reported for many years, little is known about its mode of action. Several attempts have been made to determine the possible mechanisms by which this element enhances disease resistance and interferes with fungal development. Stimulating natural defenses seemed to be the main mechanism (4). In this study, we found that disease development was significantly reduced on melons inoculated with the three pathogens 1 day after Si treatment (Fig. 2). Si treatment at 100 mM concentration before inoculation with *T. roseum* resulted in lower decay incidence and severity than treatment after inoculation (Fig. 3). This suppression clearly was correlated with induction of POD and CHT in treated fruit (Fig. 4). Si appears to induce or enhance resistance in the host.

The role of POD and CHT in defense mechanisms for host-pathogen interaction has been demonstrated previously in a number of plants (15). POD contributes to cell-wall reinforcement and is involved in the final steps of lignin biosynthesis and in the cross-linking of cell-wall protein (10,21). The enzyme often is used as a biochemical marker to predict disease resistance (7,28). In Hami melons, the present results showed a significant accumulation of POD in fruit treated with Si and challenged with *T. roseum* 24 h later

Fig. 3. Pre- and post-inoculation effect of sodium silicate (Si) on A and B, decay incidence and C and D, decay severity in Hami melon cvs. A and C, New Queen and B and D, 8601. Fruit were treated with Si at 100 mM or water (control) for 10 min and inoculated with 20 μ l of spore suspensions (1×10^5 spores ml^{-1}) of *Trichothecium roseum* either 6 and 24 h before (-24 and -6) or 6 and 24 h after (6 and 24) treatment. Decay was evaluated 9 days after incubation at $22 \pm 2^\circ\text{C}$. Data are averages of three replicates each of 12 fruit for one of three experiments that produced similar results. Bars with the same letters represent values that are not significantly different according to the least significance difference test ($P < 0.05$).

Fig. 4. Change of A and B, peroxidase (POD) and C and D, chitinase (CHT) activities in Hami melon cvs. A and C, New Queen and B and D, 8601 treated with sodium silicate (Si) at 100 mM or water (control) and challenged with 20 μ l of spore suspensions (1×10^5 spores ml^{-1}) of *Trichothecium roseum* 24 h later during storage at $22 \pm 2^\circ\text{C}$. Enzyme activities are expressed in nM tetraguaiacol produced per minute per milligram of proteins (POD) and in increase of absorbance at 550 nm per minute per milligram of proteins (CHT). Each treatment contained three replicates, and the test was performed three times. Vertical bars represent the standard error from the means of three separate tests.

(Fig. 4), which could increase the resistance against the pathogen by accelerating the rigidification of the cell wall. Fungal establishment and spread in intercellular spaces of parenchyma cells, and cell wall fortification, could limit their progression. CHT is one of the pathogenesis-related (PR) proteins in many plants (31). The enzyme hydrolyzes the cell wall of many phytopathogenic fungi (23,26,34). In this study, an increase of CHT activity also was observed in melons treated with Si, then challenged by *T. roseum*. Similar results were obtained by Chérif et al. (12), who demonstrated that several enzymes, including POD and CHT, had increased activity in cucumber plants treated with soluble silicon and challenged by *Pythium ultimum*. The present results support the hypothesis that Si is able to induce plant defense mechanisms that are expressed in response to fungal infection. These responses are multicomponent and appear to be contingent on the activation of a cascade of associated biochemical changes leading to rapid and extensive defense reactions. The multicomponent nature of these responses may explain the nonspecificity of Si-induced resistance to a wide spectrum of unrelated pathogens (3,13,25). Si present in the plant cells may mediate some defense responses that are functionally similar to systemic acquired resistance (20). However, we cannot exclude induction of other defense mechanisms which may be attributed to Si-induced resistance in melons and other systems such as accumulation of flavonoid and diterpenoid phytoalexins in cucumber and rice, respectively (19,30).

The primary findings of this work are that Si has broad-spectrum antimicrobial properties and may enhance fruit natural defenses. Considering that Si is the second most abundant atom in the Earth's crust, it is inexpensive and readily available. The cost of the Si treatment is cheaper than other new strategies. It has great commercial potential as a decay-control product. Further research is needed to test Si on additional melons types and on other host plants to confirm its broad-spectrum property and identify optimal doses.

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LITERATURE CITED

1. Aharoni, Y., Copel, A., Davidson, H., and Baikai-Golan, R. 1992. Fungicide application in water and in wax for decay control in 'Ga-

- lia' melons. N. Z. J. Crop Hortic. Sci. 20:177-179.
2. Aharoni, Y., Fallik, F., Copel, A., Gil, M., Grinberg, S., and Klein, J. D. 1997. Sodium bicarbonate reduces postharvest decay development on melons. Postharvest Biol. Technol. 10:201-206.
3. Aleshin, N. E., Avakyan, E. R., Dyakun Chak, S. A., Aleshin, E. P., Baryshok, V. P., and Vorokov, M. G. 1986. Role of silicon in resistance of rice to blast. Dokl. Akad. Nauk SSSR 291:499-502.
4. Bélanger, R. R., Bowen, P. A., Ehret, D. L., and Menzies, J. G. 1995. Soluble silicon: Its role in crop and disease management of greenhouse crops. Plant Dis. 79:329-336.
5. Bi, Y., Tian, S. P., Liu, H. X., Zhao, J., Cao, J. K., Li, Y. C., and Zhang, W. Y. 2003. Effect of temperature on chilling injury, decay and quality of Hami melon during storage. Postharvest Biol. Technol. 29:229-232.
6. Bi, Y., and Zhang, W. Y. 1991. Control of postharvest disease in Hami melon by treating inoculated fruits with fungicide and hot water. Acta Phytophylacica Sin. 18:362-363.
7. Bi, Y., and Zhang, W. Y. 1993. On changes of respiratory rate, ethylene evolution and peroxidase activity of the infected melon. Acta Phytopathol. Sin. 23:69-73.
8. Biggs, A. R., El-Kholi, M. M., El-Neshawy, S., and Nickerson, R. 1997. Effects of calcium salts on growth, polygalacturonase activity, and infection of peach fruit by *Monilinia fructicola*. Plant Dis. 81:399-403.
9. Bradford, M. M. 1976. A rapid and sensitive method for quantitation of microgram quantities of protein utilizing the principle of protein-dye binding. Anal. Biochem. 72:248-254.
10. Brisson, L. F., Tenhaken, R., and Lamb, C. J. 1994. Function of oxidative cross-linking of cell wall structural proteins in plant disease resistance. Plant Cell 6:1703-1712.
11. Chance, B., and Maehly, A. C. 1955. Assay of catalases and peroxidases. Method Enzymol. 2:764-775.
12. Chérif, M., Asselin, A., and Bélanger, R. R. 1994. Defense responses induced by soluble silicon in cucumber roots infected by *Pythium* spp. Phytopathology 84:236-242.
13. Chérif, M., and Bélanger, R. R. 1992. Use of potassium silicate amendments in recirculating nutrient solution to suppress *Pythium ultimum* on long English cucumber. Plant Dis. 76:1008-1011.
14. Chérif, M., Benhamou, N., Menzies, J. G., and Bélanger, R. R. 1992. Silicon-induced resistance in cucumber plants against *Pythium ultimum*. Physiol. Mol. Plant Pathol. 41:411-425.
15. Conrath, U., Pieterse, C. M. J., and Mauch-Mani, B. 2002. Priming in plant-pathogen interactions. Trends Plant Sci. 7:210-216.
16. El Ghaouth, A. 1994. Manipulation of defense systems with elicitors to control postharvest disease. Pages 153-167 in: Biological Control of Postharvest Diseases: Theory and Practice. C. L. Wilson and M. E. Wisniewski, eds. CRC Press, Boca Raton, FL.
17. Epstein, E. 1994. The anomaly of silicon in plant biology. Proc. Natl. Acad. Sci. USA 91:11-17.
18. Epstein, E. 1999. Silicon. Annu. Rev. Plant Physiol. Plant Mol. Biol. 50:641-664.
19. Fawe, A., Abou-Zaid, M., Menzies, J. G., and Bélanger, R. R. 1998. Silicon-mediated accumulation of flavonoid phytoalexins in cucumber. Phytopathology 88:396-401.
20. Fawe, A., Menzies, J. G., Chérif, M., and Bélanger, R. R. 2001. Silicon and disease resistance in dicotyledons. Pages 159-169 in: Silicon in Agriculture. L. E. Datnoff, G. H. Snyder, and G. H. Kornröder, eds. Elsevier Science, New York.
21. Kombrink, E., and Somssich, I. E. 1995. Defense responses of plants to pathogens. Adv. Bot. Res. 21:1-34.
22. Ma, L. Y., Bi, Y., Zhang, Z. K., Zhao, L., An, L., and Ma, K. Q. 2004. Control of pre- and postharvest main diseases on melon variety Yindi by preharvest azoxystrobin spraying. J. Gansu Agric. Univ. 39:14-17.
23. Mauch, F., Hadwiger, L. A., and Boller, T. 1988. Antifungal hydrolases in pea tissue. II. Inhibition of fungal growth by combination of chitinase and β -1,3-glucanase. Plant Physiol. 88:936-942.
24. Menzies, J. G., Bowen, P. A., and Ehret, D. L. 1992. Foliar application of potassium silicate reduce severity of powdery mildew on cucumber, muskmelon and zucchini squash. J. Am. Soc. Hortic. Sci. 117:902-905.
25. Menzies, J. G., Bowen, P. A., Ehret, D. L., Glass, A. D. M., Helmer, T., Koch, C., and Seywerd, F. 1991. Effects of soluble silicon on the parasitic fitness of *Sphaerotheca fuliginea* on *Cucumis sativus*. Phytopathology 81:84-88.
26. Pan, S. Q., Ye, X. S., and Kuc, J. 1992. Induction of chitinases in tobacco plants systemically protected against blue mold by *Peronospora tabacina* or tobacco mosaic virus. Phytopathology 82:119-123.
27. Qin, G. Z., and Tian, S. P. 2005. Enhancement of biocontrol activity of *Cryptococcus laurentii* by silicon and the possible mechanisms involved. Phytopathology 95:69-75.
28. Reuveni, R., Shimoni, Z., and Kuc, J. 1992. Peroxidase activity as a biochemical marker for resistance of muskmelon (*Cucumis melo*) to *Pseudoperonospora cubensis*. Phytopathology 82:749-753.
29. Rochow, E. G. 1973. The element. Pages 1323-1348 in: The Chemistry of Silicon. J. C. Bailar, H. J. Emeleus, R. Nyholm, and A. F. Trotman-Dickenson, eds. Pergamon Press, Oxford.
30. Rodrigues, F. A., McNally, D. J., Datnoff, L. E., Jones, J. B., Labbé, C., Benhamou, N., Menzies, J. G., and Bélanger, R. R. 2004. Silicon enhances the accumulation of diterpenoid phytoalexins in rice: a potential mechanism for blast resistance. Phytopathology 94:177-183.
31. Shewry, P. R., and Lucas, J. A. 1997. Plant proteins that confer resistance to pests and pathogens. Adv. Bot. Res. 26:135-192.
32. Spotts, R. A., and Cervantes, L. A. 1989. Evaluation of disinfectant-flotation salt-surfactant combinations on decay fungi of pear in a model dump tank. Phytopathology 79:121-126.
33. Sugar, D., and Spotts, R. A. 1986. Effect of flotation salt solutions on spore germination of four decay fungi and on side rot of pear. Plant Dis. 70:1110-1112.
34. VanLoon, L. C. 1997. Induced resistance in plants and the role of pathogenesis-related proteins. Eur. J. Plant Pathol. 103:753-765.
35. Venisse, J. S., Gullner, G., and Brisset, M. N. 2001. Evidence for the involvement of an oxidative stress in the initiation of infection of pear by *Erwinia amylovora*. Plant Physiol. 125:2164-2172.
36. Wirth, S. J., and Wolf, G. A. 1992. Microplate colorimetric assay for endo-acting cellulase, xylanase, chitinase, 1,3- β -glucanase and amylase extracted from forest soil horizons. Soil Biol. Biochem. 24:511-519.
37. Werner, D., and Roth, R. 1983. Silica metabolism. Pages 682-694 in: Encyclopedia of Plant Physiology, New Series. Vol. 15B. A. Läuchli and R. L. Bicleski, eds. Springer-Verlag, New York.