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Use of silicon oxide and sodium silicate for controlling *Trichothecium roseum* postharvest rot in Chinese cantaloupe (*Cucumis melo* L.)

Running title: Control of pink rot in cantaloupe with silicon

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Summary

Silicon oxide and sodium silicate were investigated as potential agents for the control of postharvest pink rot in Chinese cantaloupe (cultivar Yujingxing) caused by *Trichothecium roseum*. *In vitro* tests showed that sodium silicate, when added to potato dextrose agar (PDA), was effective in suppressing the radial growth of the pathogen on the medium while silicon oxide was ineffective. The effectiveness of sodium silicate increased with concentration and the growth of the fungus was completely inhibited at 100 mM. When melons were dipped in the solutions, both silicon oxide and sodium silicate significantly ($P < 0.01$) reduced the severity of pink rot of the cantaloupe with lesion diameters reduced by up to 5-fold compared with the controls. Scanning electron microscopy-energy dispersive X-ray (SEM-EDX) analysis showed that Si-treated melons had a smoother surface feature and higher Si levels in the epidermis especially at the stomata and along the junction between the exocarp and mesocarp. Enhanced peroxidase (POD) and phenylalanine ammonialyase (PAL) activities were observed in sodium silicate-treated melons but not in those treated with silicon oxide. The results indicate that different mechanisms might be involved in sodium silicate and silicon oxide-initiated reduction of postharvest pink rot in Chinese cantaloupe.

Keywords: Silicon oxide, sodium silicate, cantaloupe, pink rot, *Trichothecium*

roseum, SEM-EDX, POD, PAL

Introduction

Cantaloupe (*Cucumis melo* L.) is one of the major cash crops in northwest China, especially Ganshu province, where the hot, dry climate and large temperature difference between day and night are particularly favourable for its production (Chen *et al*., 2001). Despite its tough rind, cantaloupe has a rather short postharvest life of 7-15 days, mainly due to diseases caused by a number of pathogens (Morris & Wade, 1985). Among the major pathogens is *Trichothecium roseum*, which causes the disease known as muskmelon pink rot (Bi & Wang, 1987; Chen, 1998), which has become increasingly widespread in recent years. Infected areas appear irregular in shape without clearly defined margins and the flesh under the lesion areas tastes extremely bitter, causing large adjacent portions to become unfit for human consumption. Despite its importance, there is very little information available in the literature on the control of the disease.

In recent years, a number of studies have experimented with using silicon (Si) for the control of plant fungal diseases with promising results (Belanger *et al*., 1995; Menzies & Belanger, 1996; see also review by Epstein (1999)). Application of silicon in hydroponic growth media successfully reduced the severity of powdery mildew and Pythium root rot of cucumbers (Menzies *et al*., 1991; Cherif *et al*., 1994; Lee *et al*., 2000). Application of silicon fertilizers to potting mixes or soils reduced the incidence and severity of rice blast (Seebold *et al*., 2001) and fungal infections in peas caused by *Mycosphaerella pinodes* (Dann & Muir, 2002). When used as a foliar spray, silicon

reduced powdery mildew infections in cucumbers, muskmelons, squashes (Menzies *et al*., 1992) and grape vines (Bowen *et al*., 1992; Reynolds *et al*., 1996). Recently, Bi *et al.* (2006) showed that postharvest treatment of Hami melons (*Cucumis melo* L.var. inodorus) with sodium silicate was effective in reducing fungal decay caused by three species including *Trichothecium roseum.*

The mechanism involved in Si-induced resistance of plants to fungal diseases is not yet fully understood. Several studies have shown accumulation of silicon in cell walls around infection sites, thereby providing a greater resistance to pathogen penetration (Heath, 1981; Heath & Stumpf, 1986; Carver *et al*., 1987). On the other hand, there is also strong evidence indicating that the mechanism may be prophylactic via enhancement of the natural defence systems of the plant through the production of phenolic compounds (Fawe *et al*., 1998) or the activation of defence-related enzymes such as peroxidase, polyphenoloxidases, chitinases and beta-1,3-glucanase (Belanger *et al*., 1995; Cherif *et al*., 1994; Dann & Muir, 2002). Bi *et al*. (2006) found that sodium silicate treatment resulted in significant increases in peroxidase and chitinase activity in Hami melons while Huang et al. (2000) showed that plant activator such as acibenzolar-S-methyl could also enhance the resistance of the melon to diseases caused by several fungal species. The mechanisms by which Si imparts disease resistance to plants have recently been reviewed by Ghanmi *et al*. (2004) and Fauteux *et al*. (2005).

The objectives of the present study were: 1) to assess the effectiveness of silicon against postharvest cantaloupe pink rot; and 2) to investigate the mechanism/s of Si-mediated inhibition of *Trichothecium roseum* by analysing silicon accumulation in melon tissues and determining the peroxidase (POD) and phenylalanine ammonialyase (PAL) activities in Si-treated melons.

Materials and Methods

Materials

Cantaloupe cultivar 'Yujinxiang' (honeydew type) was used in this study. Melons were collected from a commercial orchard in Mingqing county, Gansu province, China. Uniform, sound melons were picked at maturity (43 days after blossom), packed in cartons and immediately transported to the laboratory. Silicon oxide ($SiO_2 \cdot nH_2O$) was purchased from Mingri Nano-materials Co. (Zhejiang, China) and was of analytical grade (fine powder). Average particle size of the chemical was 15 nm in diameter and the ratio of surface to weight was 160 $m^2 kg^{-1}$. Sodium silicate, also of analytical grade and in a powdery form, was purchased from Komeo Chemicals Co. (Tianjin, China).

Pathogen inoculum

Trichothecium roseum was isolated from infected cantaloupes, following the method

described by Fang (1998), and maintained on potato dextrose agar (PDA). Pathogen inoculum was prepared by subculturing the fungus onto PDA plates which were then incubated at 25 °C for 8 -10 days. The plates were flooded with sterile distilled water, containing 0.01% (v/v) Tween 80, with gentle stirring to remove the spores, and the spore suspension was adjusted with distilled water to 3 x10^6 conidia mL^{-1}, determined with a haemocytometer.

***In vitro* test**

In vitro tests against *T. roseum* were conducted according to the method of El Ghaouth *et al*. (1992). Silicon oxide or sodium silicate was added to autoclaved PDA medium to final concentrations of 25, 50, 100 and 200 mM, containing 0.01% (v/v) Tween 80, with the pH values of the media at 10.48, 10.93, 11.30 and 11.45, respectively. Control PDA plates contained 0.01% (v/v) Tween 80 solution only. The plates were inoculated in the centre with agar plugs (6 mm in diameter) cut from the edge of actively growing colonies of *T. roseum* on PDA, and then incubated at 25 °C. Incubation was stopped when growth of the pathogen had covered the whole Petri dishes of the controls, and the diameters of the growth in the Si-treated plates were measured. Five replicates were used for each chemical at each concentration and the whole experiment was repeated three times.

***In vivo* test**

Uniform, sound melons were selected and dipped into silicon oxide suspensions or sodium silicate solutions of 50, 100 or 200 mM, containing 0.01% (v/v) Tween 80, for five seconds. After air drying at 24±2 °C for 24 h, the melons were disinfected by wiping the surface with 75% ethanol. Six wounds (3±1mm deep and 3±1mm in diameter) were cut on the surface of each melon with a sterile knife, and each wound was inoculated with 10 µl of the spore suspension, prepared as described above. The melons were placed in plastic bags separately, which were then stored in an air-conditioned room with temperature and relative humidity set at 24±2 °C and 95±1%, respectively. Lesion diameters caused by the growth of the fungus were measured after 6 days. Three melons were used for each treatment and the whole experiment was repeated three times.

Assays of peroxidase (POD, EC 1.11.1.7) and phenylalanine ammonialyase (PAL, EC 4.3.1.5) activities

In a separate experiment, melons were treated with 0.01% (v/v) Tween 80 (control), 200 mM silicon oxide or 100 mM sodium silicate in the same way as described above and then stored at 24±2 °C and 95±1% relative humidity for 24 h. The melons were then sampled and their POD and PAL activities determined according the method described by Li (2000). Melon rind samples (5 g, 2 mm thick) were homogenized in about 8 ml 50 mM phosphate buffer (pH 6.0). The homogenate was stirred at 4 °C for 30 min, centrifuged at 5764 *g* for 15 min and the supernatant collected. The extraction

procedure was repeated twice more with the pellet, and the supernatants pooled, made up to 25 ml with the same buffer and used as enzyme preparation for POD assay. The substrate solution was prepared by mixing 2.9 ml 50 mM phosphate buffer (pH 6.0) with 1.0 ml 2% (v/v) H_2O_2 and1.0 ml 50 mM *o*-phenylenediamine. The solution was pre-warmed in a 30 °C water bath for 5min, and then 0.1ml enzyme solution was added. The mixture was incubated in a 30 °C water bath for 1 min, immediately diluted to twice its volume with the phosphate buffer and the absorbance at 470 nm measured in 1-min intervals for 5 min. POD activity was expressed as ΔA_{470} $min^{-1}g^{-1}$ fruit tissue.

For PAL assay, melon rind samples (5 g, 2 mm thick) were homogenized in 10 ml 0.1 M borate buffer (pH 8.8) containing 5 mM β-mercaptoethanol, 2 mM EDTA and 3% (w/v) polyvinylpolypyrrolidone (PVPP). The homogenate was stirred for 1 h at 4 °C, centrifuged at 5764 for 15 min and the supernatant was used for PAL assay. For the assay, 1 ml of the enzyme extract was mixed with 1 ml of 20 mM L-phenylalanine in 0.1 M borate buffer (pH 8.8) and 2 ml de-ionized water. The reaction mixture was incubated in a 30 °C water bath for 30 min, and the reaction was stopped by adding 0.1 ml of 6 N HCl. Absorbance at 290 nm was measured against a substrate blank without the enzyme. PAL activity was expressed as ΔA_{290} $h^{-1}g^{-1}$ fruit tissue. The experiment was repeated three times.

Scanning electron microscopy-energy dispersive X-ray (SEM-EDX) analysis

Melons stored for 24 h after treatment with 0.01% Tween 80 (control), 200 mM silicon oxide and 100 mM sodium silicate respectively, as described above, were analyzed by SEM-EDX for the silicon and calcium content in the epidermis and along the cross section of the outer layer (exocarp and outer mesocarp) using the linescan mode. The samples were cut to appropriate size and air-dried in a clean chamber. The specimens were analyzed by a Philips EOAX9100 scanning electron microscope (Philips, The Netherlands), equipped with a WD-8X energy dispersive X-ray analysis and digital image analysis system designed and manufactured by Wuhan University (Wuhan, Hubei, China). Acceleration voltage was 30kv, and the X-ray count rate 5991counts/s. The magnification was 1000 times for examination of the epidermis and 200 times for cross section.

Statistical analysis

The data obtained were analyzed by one way analysis of variance (ANOVA) and means of treatments were separated at the 5% or 1% significance level, using Duncan's multiple range test.

Results

Effect of silicon oxide and sodium silicate on the growth of *T. roseum in vitro*

Silicon oxide did not show a significant effect ($P > 0.05$) on the growth of *T. roseum* at

any of the four concentrations tested (Table 1). Growth of the pathogen on PDA plates treated with silicon oxide was similar to that on the control plates. Sodium silicate, however, significantly ($P < 0.01$) inhibited the growth of *T. roseum* at all of the concentrations tested. Higher concentrations of sodium silicate resulted in a greater inhibition of the growth and, when the concentration reached 100 mM, growth of the pathogen was completely suppressed (Table 1).

Effect of silicon oxide and sodium silicate on the growth of *T. roseum in vivo*

Both silicon oxide and sodium silicate were effective in reducing the decay of the melons caused by the pathogen (Figure 1). Compared with the controls, lesions were markedly smaller on the surfaces of melons treated with silicon oxide at all three concentrations tested. Lesions on melons treated with silicon oxide at 200 mM were significantly smaller ($P < 0.01$) than those treated with both 50 mM and 100 mM. For sodium silicate treated melons, the lesions were significantly ($P < 0.01$) smaller at all three concentrations of the chemical when compared with the controls. However, no significant differences in lesion size were observed between the three concentrations used.

Effect of silicon oxide and sodium silicate on silicon concentrations in melons

Figure 2 shows the SEM-EDX images of melon samples following different treatments. The top line in each graph indicates a line along which the silicon and

calcium contents were determined by EDX linescan analysis while the peak heights show the relative levels of Si and Ca along the line.

The silicon level of the control sample showed little variations across the epidermis and cross section of the rind (exocarp and outer mesocarp), and it was very similar to the calcium level except at stomata, where calcium levels were higher than silicon (Fig. 2a and 2b). In contrast, the silicon level in the epidermis of melons treated with either silicon oxide or sodium silicate was generally higher than calcium, especially at stomata where a silicon peak was clearly evident (Fig. 2c and 2e). On the cross section, a silicon peak was observed along the junction between the exocarp and mesocarp of melons treated with silicon oxide (Fig. 2d) and sodium silicate (Fig. 2f). Such a silicon peak was absent in the control sample (Fig. 2b).

In addition, under SEM examination, the epidermis of the control sample appeared to be relatively rough (Fig. 2a), whereas that of silicon treated melons, especially those treated with silicon oxide, was much smoother in comparison (Fig. 2c and 2e).

Effect of silicon treatment on POD and PAL activity

The effects of silicon oxide and sodium silicate on the activity of the POD and PAL were quite different (Table 2). The activity of POD and PAL increased by more than 100 % and 26 %, respectively, over the control ($P < 0.01$) as a result of the treatment

with sodium silicate. In contrast, treatment with silicon oxide did not cause a significant ($P > 0.05$) change in PAL activity while the activity of POD actually decreased compared with that of the control.

Discussion

Results presented in this paper show that both silicon oxide and sodium silicate were effective in reducing postharvest *T. roseum* rot in Chinese cantaloupes. Bi *et al*. (2006) reported that application of sodium silicate to Hami melons (a different cultivar to the one used in the current study) postharvest significantly reduced the incidence and severity of fungal decay caused by three species including *T. roseum*, which concurs with the results of the current study. Previous studies in this area have focused on the reduction of fungal infections in pre-harvest crops through fertilizer, soil or foliar-applied silicon. Two hypotheses have been put forward to explain the mode of action of fertilizer and soil-applied silicon: 1) silicon deposits and polymerizes in cell walls thereby enhancing the resistance of host tissues to fungal attack; 2) silicon acts by stimulating the natural defence systems of the plant through the production of phenolic compounds and the activation of enzymes such as POD, PAL and PPO. The mode of action of foliar-applied silicon is not clear. One study has observed specific accumulation of exogenous silicon near the powdery mildew infected sites of Si-sprayed grape berries (Reynolds *et al*., 1996) suggesting a mechanism similar to the first hypothesis. This hypothesis also seems to apply in the silicon oxide-induced

reduction of pink rot in cantaloupes observed in the present study. Melons treated with silicon oxide showed an increased level of Si in the epidermis tissues, especially at the stomata and along the junction between the exocarp and mesocarp where Si peaks were observed. Although silicon oxide is not a water-soluble substance, the particle sizes of the material used in the present study were in the nanometre range (15 nm in average), which could have made it possible for the chemical to penetrate into the exocarp and form deposits. Such accumulation of exogenous Si could conceivably form a physical barrier to the invasion of fungal pathogens. When tested *in vitro*, silicon oxide did not show any effect on the growth of *T. roseum* on PDA, nor did treatment of melons with the chemical stimulate POD and PAL activity. Although this does not preclude the activation of other plant defence mechanisms such as production of phenolic compounds, formation of a physical barrier to fungal penetration as a result of accumulation of Si in the rind tissues seems to play a key role in the enhanced resistance of the melons to pink rot. Furthermore, the surface of silicon oxide-treated melons were much smoother compared with the control, which could make the attachment of fungal hyphae more difficult.

The mechanism of sodium silicate-induced resistance to pink rot of cantaloupes, on the other hand, appears to be more complex. Compared with the control, melons treated with sodium silicate showed not only a smoother surface feature and an increased level of Si in the rind tissues, similar to those observed in silicon

oxide-treated melons, but also a significant increase in both the POD and PAL activities. It is well known that both POD and PAL play a significant role in the defence systems of plants against fungal infections (Vidyasekaran, 1988; Taiz & Zeiger, 2002) Furthermore, when tested *in vitro*, sodium silicate inhibited the growth of *T. roseum* on PDA. Total inhibition was achieved when the concentration of the chemical reached 100 mM. A previous study has reported that sodium silicate at the concentration range of 0-17 mM mildly promoted the conidia germination and germ tube development of *Uncinula necator* (Bowen *et al*., 1992). However, it is not unusual for a chemical to support fungal growth at lower concentrations yet to be fungistatic at higher concentrations. Thus, the observed inhibition of pink rot in cantaloupe as a result of treatment with sodium silicate could be due to the accumulation of Si in the epidermal tissues forming a physical barrier to fungal attack, its ability to induce defence responses in the melons, its fungistatic properties, or a combination of the three modes of action. It should be pointed out that sodium silicate solutions are highly alkaline and it is therefore possible (and even probable) that their fungistatic property is due to the strong alkalinity. It is also possible that the apparently enhanced POD and PAL activities are a stress response of the melon to exposure to the strongly alkaline conditions rather than a specific response to Si treatment. Bi *et al*. (2006) reported that treatment of Hami melons with sodium silicate resulted in increases in the activity of both POD and chitinase, a finding similar to that of the current study. They further found that sodium silicate at 200 mM was phytotoxic to the

epidermal tissues of the melon. However, such phytotoxicity was not found in the current study, probably due to the fact that the melon cultivar used in the current study has a much tougher rind compared to the Hami melon used in the study of Bi *et al*. (2006).

In conclusion, this study has demonstrated the potential of silicon oxide and sodium silicate for controlling postharvest *T. roseum* rot in cantaloupes. This potential is enhanced by the fact that silicon is one of the most abundant materials on the surface of the earth. Further studies could examine the effect of silicon on the host-pathogen interactions to gain a better understanding of the modes of actions, and to explore the possibility of extending the application of the chemicals to the control of other postharvest fungal diseases in fruits.

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Table 1 Effect of silicon oxide and sodium silicate on the radial growth of *Trichothecium roseum* on PDA

Silicon agent	Concentration (mM)	Lesion diameter (mm)
Control	-	80.3 a
Silicon oxide	25	81.3 a
	50	82.3 a
	100	80.3 a
	200	80.6 a
Sodium silicate	25	16.7 b
	50	12.3 c
	100	0.0 d
	200	0.0 d

[a-d] Data were means of five replicate tests and values followed by a different letter differ significantly ($P < 0.01$) according to Duncan's multiple range test.

Table 2 Peroxidase (POD) and phenylalanine ammonia-lyase (PAL) activities in Chinese cantaloupe treated with 200 mM silicon oxide and 100 mM sodium silicate

Treatment	POD activity (ΔA_{470} $min^{-1}g^{-1}$ fruit tissue)	PAL activity (ΔA_{290} $h^{-1}g^{-1}$ fruit tissue)
Control	4.03 a	5.25 a
Silicon oxide	2.46 b	5.54 a
Sodium silicate	8.86 c	6.63 b

Data were means of triplicate tests. Values within the same column followed by a different letter differ significantly ($P < 0.01$) according to Duncan's multiple range test.

Figure captions

Figure 1 Effect of silicon oxide and sodium silicate on the growth of *Trichothecium roseum* in Chinese cantaloupe. C1 and C2 represent control 1 (no treatment) and control 2 (treated with 0.01% tween 80) respectively; Si stands for silicon oxide and Na for sodium silicate while the suffixal numbers 1, 2 and 3 represents the concentration of the two chemicals at 50, 100 and 200 mM, respectively. Columns with different letters differ significantly ($P < 0.01$) in values according to Duncan's multiple range test.

Figure 2 Scanning electron microscope (SEM) images of epidermis (left) and cross section (right) of the outer layer of Chinese cantaloupe melons with the Si (black) and Ca (white) distribution obtained by energy dispersive X-ray (EDX) linescan analysis. A and B, control (treated with 0.01% tween 80 only); C and D, melons treated with 200 mM silicon oxide; E and F, melons treated with 100 mM sodium silicate. Arrows with the number 1 indicate stomata while those with the number 2 indicate the junction between exocarp and mesocarp.

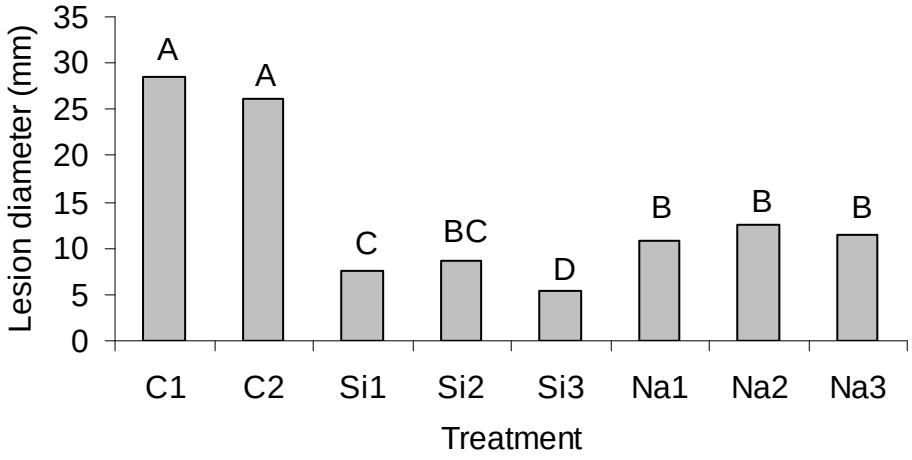
Lesion diameter (mm)
35
30
25
20
15
10
5
0
A
A
C
BC
D
B
B
B
C1
C2
Si1
Si2
Si3
Na1
Na2
Na3
Treatment

Figure 1

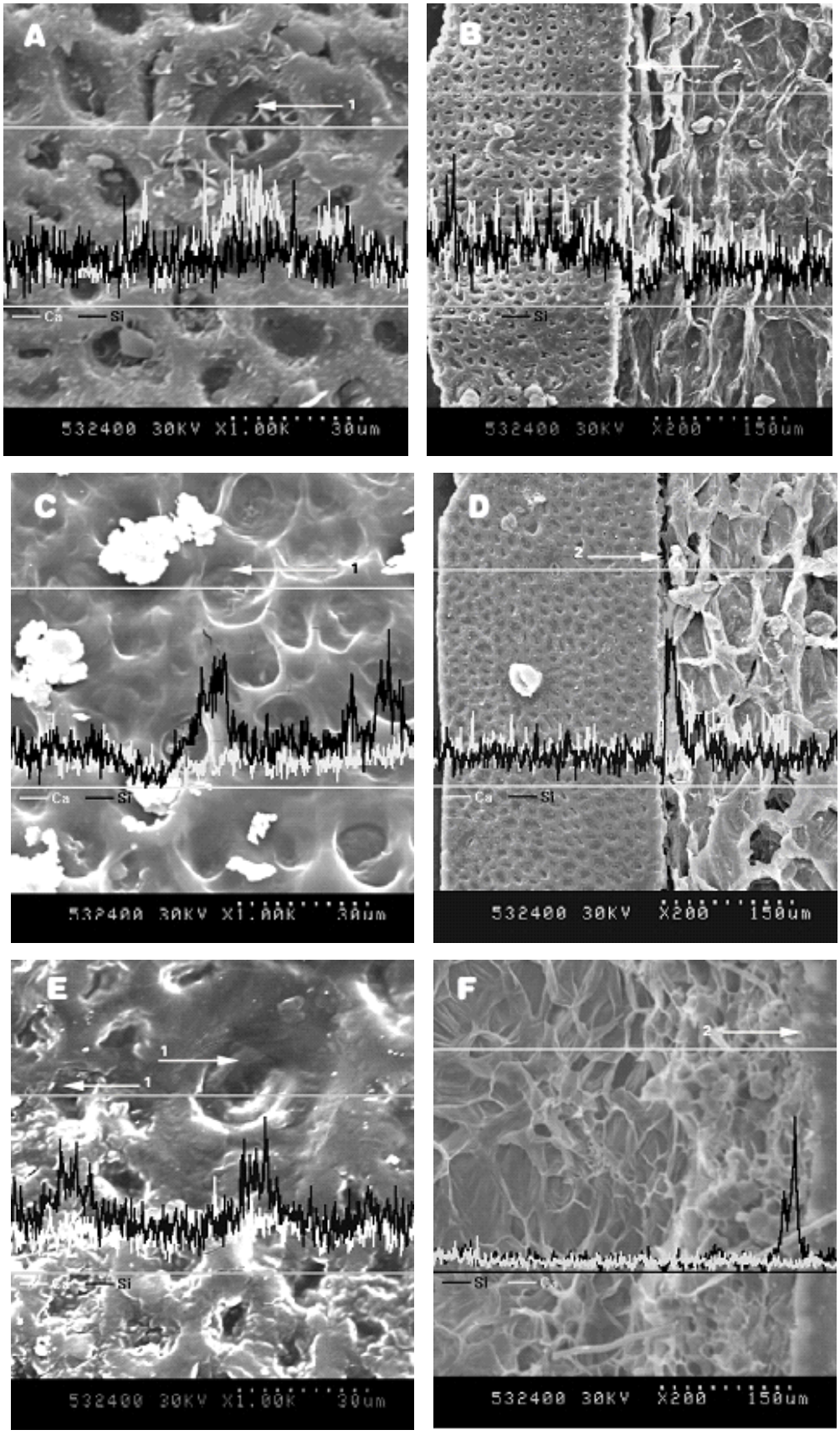
A
1
Ca
Si
532400 30KV X1.00K 30um
B
2
Ca
Si
532400 30KV X200 150um
C
1
Ca
Si
532400 30KV X1.00K 30um
D
2
Ca
Si
532400 30KV X200 150um
E
1
1
Si
532400 30KV X1.00K 30um
F
2
Si
532400 30KV X200 150um

Figure 2