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Enhancement of postharvest disease resistance in *Ya Li* pear (*Pyrus bretschneideri*) fruit by salicylic acid sprays on the trees during fruit growth

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Abstract

The *Ya Li* pear (*Pyrus bretschneideri*) trees were sprayed three times with 2.5 mM salicylic acid (SA) around 30, 60 and 90 days after full flowering. The fruit were harvested at commercial maturity (about 120 days after full flowering), inoculated with *Penicillium expansum*, and incubated at 20 °C, 95–100% RH. The results showed that resistance to the pathogen of the mature pear fruit was remarkably enhanced by the SA sprays. Disease incidence in the SA-treated fruit was 58.0% or 26.5%, and lesion diameter on SA-treated fruit was 58.4% or 29.0% lower than that in/on fruit without SA treatment (control) on day 12 or 17 after incubation, respectively. The SA spray applied to the trees around 30 days after full flowering notably enhanced accumulation of hydrogen peroxide in the young fruit. Meanwhile, activities of defense enzymes, including peroxidase, phenylalanine ammonia-lyase (PAL), chitinase or β-1,3-glucanase in the young fruit from SA-treated trees was 29.5%, 60.0%, 24.4% or 35.7% higher than that in the control fruit 4 days after the SA spraying. Furthermore, after harvest, activities of PAL, chitinase and β-1,3-glucanase were still significantly higher in the mature pear fruit from the trees sprayed three times with SA than those of the control fruit. Activities of the antioxidant enzymes including catalase and ascorbate peroxidase in the young fruit were significantly reduced by SA spraying. However, the activity of another antioxidant enzyme, glutathione reductase in the young fruit was significantly enhanced by SA spraying. These results suggest that enzymes exerting their functions in different ways may be coordinately regulated by SA in the pear fruit. Our study indicates that treatment of SA sprays on the trees may provide further protection against postharvest disease of *Ya Li* pear fruit in practice and could be used as an alternative and economical approach to reduce application of chemical fungicides.

Abbreviations: APX – ascorbate peroxidase; CAT – catalase; GR – glutathione reductase; H_2O_2 – hydrogen peroxide; PAL – phenylalanine ammonia-lyase; POD – peroxidase; SA – salicylic acid

Introduction

Decay caused by pathogens is responsible for most of the postharvest losses of *Ya Li* pear (*Pyrus bretschneideri*) fruit during storage but has been controlled mainly by synthetic fungicides after harvest. However, application of fungicides is increasingly limited due to the development of fungicide resistance by pathogens, public concern about fungicide residues in food and potential

harmful effects on the environment and human health. New strategies for control of postharvest diseases are therefore required. Induction of resistance to pathogen infection has been indicated as a promising approach for controlling postharvest diseases of fruit (Porat et al., 2003; Qin et al., 2003; Liu et al., 2005). It has been documented that disease resistance can be triggered by elicitors such as salicylic acid (SA) in seedlings or leaves of annual plants such as *Arabidopsis*, tobacco, cucumber, tomato, rice and bean (Malamy et al., 1990; Sticher et al., 1997). SA is a simple phenolic compound naturally produced by many plants and has been considered as a signal molecule in the signal transduction pathway in plants (Malamy et al., 1990; Sticher et al., 1997). A few studies have shown that disease resistance in fruit could also be induced by postharvest treatment with SA (Qin et al., 2003; Zainuri et al., 2001). It has been demonstrated that pre-harvest application of SA tended to suppress postharvest anthracnose disease severity caused by *Colletotrichum gloeosporioides* in mango fruit (Zainuri et al., 2001), and that application of acibenzolar-S-methyl, a functional analogue of SA, on melon plant prior to flowering could effectively inhibit infections of several postharvest fungal diseases in melon fruit (Huang et al., 2000). However, little is known about how disease resistance in harvested fruit of woody plants may be affected by SA treatment on the trees in the field during fruit growth and development.

Faize et al. (2003) found that the same mechanism involved in resistance against scab in leaves also operated in young pear fruit. Terry and Joyce (2004) suggested that the enhanced resistance in developing fruit triggered by pre-harvest elicitor treatment could persist in fruit during ripening and storage. So far however, little effort was focused on the enhancement and persistence of resistance in young pear fruit by SA treatment. The objective of this work was to evaluate how disease resistance in harvested fruit of *Ya Li* pear may be affected following application of SA to the trees during the growing season. In this study, we provide evidence that SA treatment of trees leads to a reduction in pathogen incidence and severity in fruit inoculated with *Penicillium expansum* after harvest and the systemic induced resistance in the SA-sprayed fruit was enhanced and persisted.

Materials and methods

Plant and field treatments

Field treatments with salicylic acid (SA) on *Ya Li* pear trees were performed in a commercial orchard in Beijing in 2003 and 2004. Other cultural management was carried out according to regular commercial practice. During the growing season, the trees were sprayed twice with 0.08% (w/v) carbendazim and once with 0.025% (w/v) iprodione to control orchard diseases. Sixteen-year-old *Ya Li* pear trees were selected for the field treatments (3 trees per treatment, 3 repeats). Based on the results of our preliminary experiment, 5 l 2.5 mM SA solution was sprayed on each tree each time. The SA sprays were applied three times around 30, 60 and 90 days after full flowering. Control trees were sprayed with water at similar intervals. The young pear fruit (10 fruits per tree per time) for the relevant assays were collected from the SA-treated and non SA-treated trees on days 2, 4, 6, 8, 10 and 12 after the first SA spray. The mature fruit was harvested at commercial maturity (about 120 days after full flowering) and sampled for uniformity, shape, size and freedom from physical damage and infections. After harvest, the fruit was stored at 20 °C, 85–95% RH for biochemical determination.

Inoculation and disease evaluation in mature fruit

Penicillium expansum was isolated from infected *Ya Li* pear fruit and maintained on potato dextrose agar (PDA) according to method of Liu et al. (2005). Conidial suspension of the pathogen was prepared by flooding the 14 day-old culture dishes incubated at 26 °C and adjusted to 1×10^5 conidia ml^{-1} with sterile distilled water containing 0.01% Tween 80 using a haemacytometer. Both the SA-treated and control fruit (15 mature fruits per treatment, three replicates) were surface-sterilized with 70% ethanol, and wounded with a sterilized nail at 3 points (3 mm deep×3 mm wide) on the equator of each fruit. Ten microliters of the conidial suspension was injected into each wounded site, and the inoculated fruit was incubated at 20 °C, 95–100% RH. Disease incidence (the percentage of fruit with visible disease development) and lesion diameter on each fruit were recorded on days 3, 7, 12 and 17 after incubation. Fruit was

classified as infected when rot extended more than 1 mm beyond the inoculation wound.

Enzyme analysis

For POD activity assay, sampled tissues (5.0 g) were homogenized on ice with 5 ml of 100 mM sodium acetate buffer, pH 5.5, containing 1 mM polyethyleneglycol (PEG-4000), 1% (v/v) Triton-100, 8% (v/v) polyvinylpolypyrrolidine (PVPP) and 1 mM phenyl-methyl-sulphonyl fluorides (PMSF). The homogenate was centrifuged at 13,000×*g* for 20 min at 4 °C. POD activity of supernatants was assayed according to Lurie et al. (1997) and expressed as ΔOD_{470} min^{-1} mg^{-1} protein.

For PAL activity assay, sampled tissues (5.0 g) were homogenized on ice with 5 ml of 100 mM sodium borate buffer, pH 8.8, containing 5 mM β-mercaptoethanol, 2 mM ethylene diaminetetra-acetic acid (EDTA) and 4% (w/v) polyvinyl pyrrolidine (PVP). The homogenate was centrifuged as described above. PAL activity was determined according to Assis et al. (2001) and expressed as nmol *trans*-cinnamic acid h^{-1} mg^{-1} protein.

For chitinase and β-1,3-glucanase assays, 5.0 g of the sampled tissue was homogenized with 5 ml of 100 mM sodium acetate buffer, pH 5.2, containing 5 mM β-mercaptoethanol and 1 mM EDTA, and centrifuged at 13,000 × *g* at 4 °C for 20 min, and the supernatant was collected for the enzymatic assay.

Chitinase activity was measured according to Boller et al. (1983) with some modifications. Two hundred μl of the enzymatic extract and 0.5 mg washed chitin (Sigma) plus 1.5 ml of 100 mM sodium acetate buffer (pH 5.2), was incubated at 37 °C for 1 h, then was centrifuged at 12,000×*g* for 5 min. Afterwards, 1 ml of the supernatant plus 0.1 ml of 3% (w/v) desalted snailase (snail acetone powder, Sigma, No. S9764) was incubated at 37 °C for 1 h and stopped by immediately adding 0.2 ml of 0.6 M potassium tetraborate and heating for 5 min in a boiling water bath. After cooling, 2 ml of reagent of 10% (w/v) 4-(dimethylamino)-benzaldehyde (DMAB) diluted 1:5 with glacial acetic acid was added to the mixture and incubated at 37 °C for 20 min. Absorbance of the solution at 585 nm was measured. The chitinase activity was expressed as unit mg^{-1} protein, where one unit was defined as 10^{-9} mol of *N*-acetyl-D-glucosamine (Glc-NAc) produced per second under the assay conditions.

β-1,3-glucanase activity was assayed according to Abeles and Forrence (1979) with modifications. Fifty microliters of the enzymatic extract plus 50 μl of 4% (w/v) laminarin (Sigma) was incubated at 37 °C for 30 min. The reacted mixture was added to 400 μl of dinitrosalicylate (DNS) and boiled for 5 min. After cooling, absorbance of the solution at 500 nm was measured. The β-1,3-glucanase activity was expressed as unit mg^{-1} protein, where one unit was defined as the reducing sugar equivalent to 10^{-9} mol of glucose produced per second under the assay conditions.

For CAT assay, 5.0 g sampled tissue was homogenized with 8 ml of 100 mM sodium phosphate buffer, pH 7.8, containing 2% (w/v) PVP and 5 mM DTT. The homogenate was centrifuged as described above. CAT activity was determined by the method of Milosevic and Slusarenko (1996) and expressed as ΔOD_{240} min^{-1} mg^{-1} protein.

For APX and GR assays, 5.0 g sampled tissue was homogenized with 5 ml of potassium phosphate buffer, pH 7.5, containing 1 mM EDTA. The homogenate was centrifuged as described above and the supernatant was used as crude enzyme. The APX activity was determined as described by Nakano and Asada (1981), and expressed as ΔOD_{290} min^{-1} mg^{-1} protein. The GR activity was assayed according to Foyer and Halliwell (1976), and expressed as ΔOD_{340} min^{-1} mg^{-1} protein.

Soluble protein content was assayed according to the method of Bradford (1976) with bovine serum albumin as standard.

Determination of H_2O_2

Tissue samples (3.0 g) were homogenized with 5 ml of cooled acetone at 4 °C. The homogenate was centrifuged at 12,000×*g* at 4 °C for 10 min. H_2O_2 of the supernatant was estimated by forming titanium-hydroperoxide complex according to Prochazkova et al. (2001). The H_2O_2 content was expressed as nmol g^{-1} FW.

Statistical analysis

All data were collected and analyzed by one-way analysis of variance (ANOVA) with statistical software of the SPSS 11.0 for windows (SPSS Inc., Chicago, IL, USA). Mean separations were performed

by employing Duncan's multiple comparison procedure, and differences at the 5% level were considered significant. Each experiment had three replicates and all experiments were run three times with similar results in both years of 2003 and 2004. Measurements from all the replicates were combined and treatment effects analyzed.

Results

Effects of SA treatment on disease resistance of Ya Li *pear fruit*

Disease incidence and development in the pear fruit inoculated with *P. expansum* were significantly reduced ($P < 0.05$) by SA sprays applied to the trees during fruit development. As shown in Figure 1a, disease incidence of the SA-treated fruit was 48.7%, 58.0% or 26.5% lower than that on the fruit without SA treatment (control) on days 7, 12 or 17 after inoculation with *P. expansum*, respectively. Lesion diameter (Figure 1b) on the SA-treated fruit was 58.4% or 29.0% lower than that in the control fruit on days 12 or 17 of the incubation period, respectively.

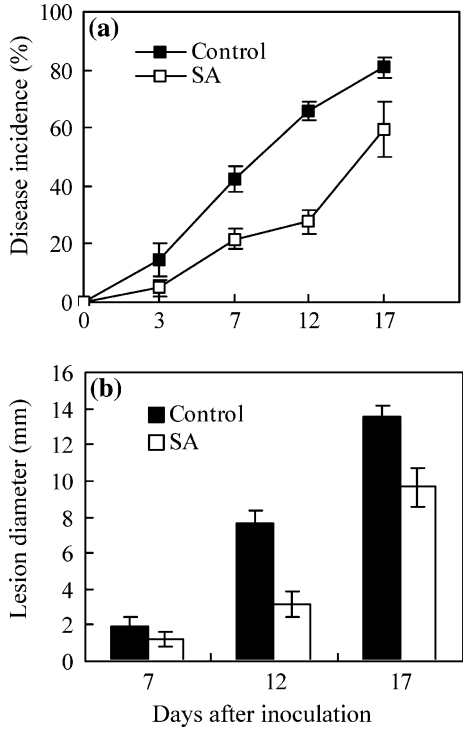

Figure 1. Effects of the SA sprays on disease incidence (a) and lesion diameter (b) in the pear fruit inoculated with *P. expansum* after harvest. *Ya Li* pear trees were sprayed three times with 2.5 mM salicylic acid (SA) or water (control) around 30, 60 and 90 days after full flowering. The fruit harvested about 120 days after full flowering from the sprayed trees were inoculated with 10 μl of the conidial suspension of *P. expansum* (1×10^5 conidia ml^{-1}), and incubated at 20 °C, 95–100% RH for disease development. Each point or column represents the mean of three replicates (15 fruits of each) and bars represent standard errors.

Effects of SA treatment on the accumulation of H_2O_2 in pears

SA sprays on the trees effectively enhanced accumulation of H_2O_2 in the young fruit. As shown in Figure 2, the H_2O_2 level in fruit of SA-treated trees was 18.0% or 13.3% higher than that in control fruit 2 or 4 days after the SA sprays, respectively.

Effects of SA treatment on activities of defense enzymes in pears

As shown in Figure 3, activities of defense enzymes of POD, PAL, chitinase and β-1,3-glucanase in the young fruit were notably enhanced by SA sprays on the trees. POD and PAL activities in SA-treated fruit were 12.8% and 51.4%, or 29.5% and 60.0%, respectively, higher than those in control 2 or 4 days after the SA spray (Figure 3a, b). Meanwhile, chitinase or β-1,3-glucanase activity in SA-treated fruit was 24.4% or 35.7% higher than that in control 4 days after the SA spray (Figure 3c, d). Activities of all the enzymes in the SA-treated pear fruit were still significantly

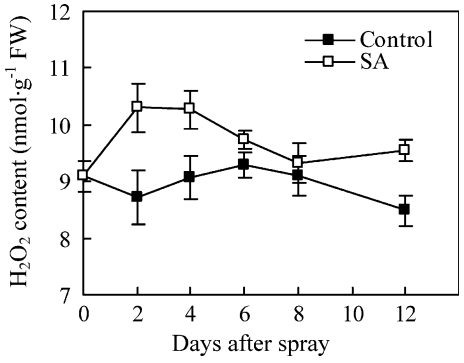

Figure 2. Effects of the SA spray on the H_2O_2 level in the young pear fruit. *Ya Li* pear trees were sprayed with 2.5 mM salicylic acid (SA) or water (control) 30 days after full flowering and the young fruit were harvested after the spray. Bars represent standard errors for the means of three replications.

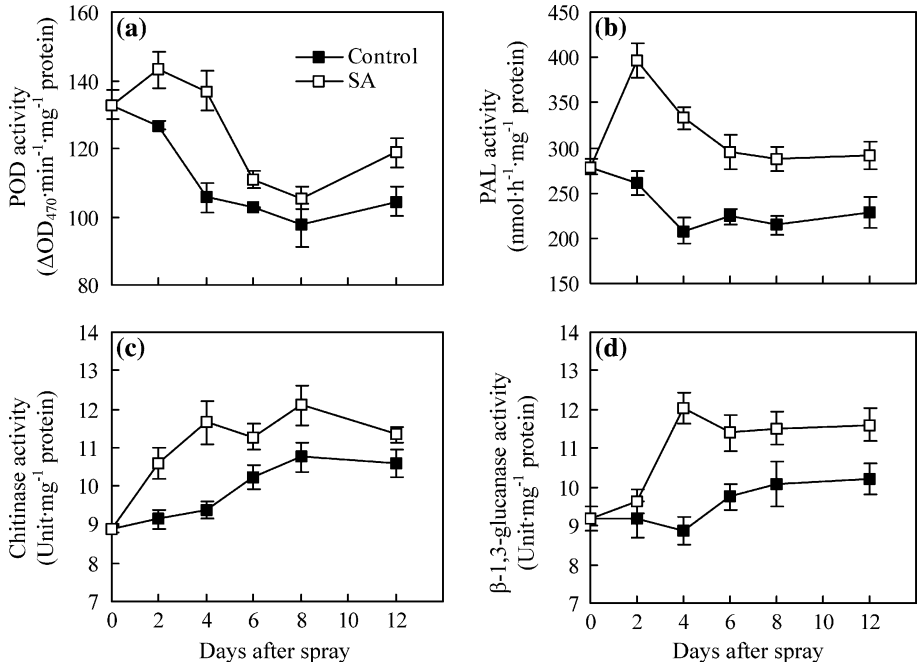

Figure 3. Effects of the SA spray on activities of peroxidase (POD) (a), phenylalanine ammonia-lyase (PAL) (b), chitinase (c) and β-1,3-glucanase (d) in the young pear fruit. *Ya Li* pear trees were sprayed with 2.5 mM salicylic acid (SA) or water (control) 30 days after full flowering and the young fruit were harvested after the spray. Bars represent standard errors for the means of three replications.

($P < 0.05$) higher than that in control fruit 12 days after SA application.

Effect of SA treatment on activities of antioxidant enzymes in pears

As shown in Figure 4, activities of antioxidant enzymes including CAT and APX in young pear fruit were significantly reduced by the SA spray. The CAT activity in fruit of SA-treated trees was 24.5% or 15.3% lower than that in the control on days 4 or 6 after the SA spray (Figure 4a), respectively. APX activities in the young fruit were also reduced by the SA spray (Figure 4b). Unlike CAT and APX, GR activity in the fruit was enhanced by the SA sprays. As shown in Figure 4c, GR activity in fruit of SA-treated trees was 20.4%, 26.1% or 24.3% higher that in the control on days 6, 8 or 12 after the SA sprays, respectively.

Effects of SA treatment on activities of defense enzymes in mature pears after harvest

As shown in Figure 5a–c, activities of PAL, chitinase and β-1,3-glucanase in the mature *Ya Li* pear fruit harvested from trees sprayed three times with 2.5 mM salicylic acid (SA) were all significantly enhanced by the pre-harvest foliar SA sprays. Activities of PAL, chitinase and β-1,3-glucanase in SA-treated fruit were, respectively, 54.6%, 41.9% and 20.6% higher than those in non SA-treated control fruit. During storage, activities of PAL and β-1,3-glucanase in SA-treated fruit were still significantly higher than those in the control fruit except chitinase. No significant difference of POD activity was detected at harvest in the SA-treated fruit and the control fruit (data not presented).

Discussion

Studies have shown that SA treatment can enhance disease resistance of a few growing plants or detached plant organs (Sticher et al., 1997; Meena et al., 2001; Zainuri et al., 2001; Qin et al., 2003). Our study indicated that postharvest disease resistance of *Ya Li* pear fruit could also be notably enhanced by foliar SA sprays on the trees during the growth season. Both disease incidence and

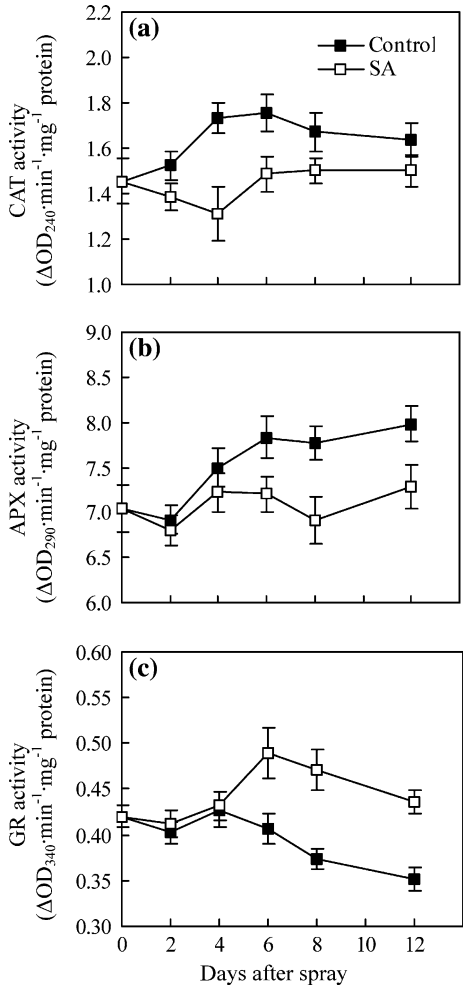

Figure 4. Effect of the SA spray on activities of catalase (CAT) (a), ascorbate peroxidase (APX) (b) and glutathione (GR) (c) in the young pear fruit. *Ya Li* pear trees were sprayed with 2.5 mM salicylic acid (SA) or water (control) 30 days after full flowering and the young fruit were harvested after the spray. Bars represent standard errors for the means of three replications.

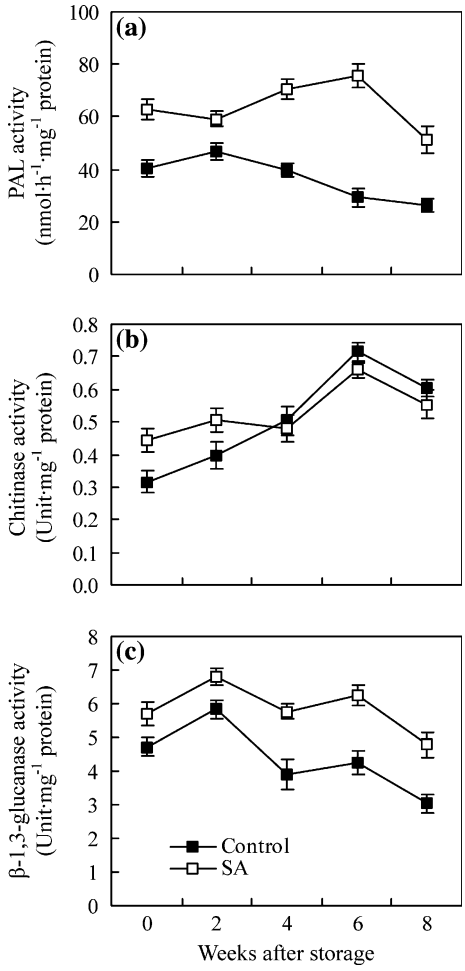

Figure 5. Effects of the SA sprays on activities of phenylalanine ammonia-lyase (PAL) (a), chitinase (b) and β-1,3-glucanase (c) in the mature pear fruit after harvest. *Ya Li* pear trees were sprayed three times with 2.5 mM salicylic acid (SA) or water (control) around 30, 60 and 90 days after full flowering. The mature fruit were harvested about 120 days after full flowering and stored at 20 °C, 85–95% RH. Bars represent standard errors for the means of three replications.

lesion diameter in the fruit inoculated with *P. expansum* were significantly reduced by treating the trees with SA. These results may suggest that the enhanced resistance system in *Ya Li* pears was established in the fruit with SA sprays, and lasted for a long period from the beginning of growth and development until postharvest. Indeed, it has been demonstrated that application of SA on mango fruit before harvest (Zainuri et al., 2001), or acibenzolar-S-methyl, a functional analogue of SA, on melon plant prior to flowering (Huang et al., 2000) could effectively reduce postharvest diseases in fruit.

Rapid generation of reactive oxygen species has been considered as one of the earliest events correlated with plant resistance to pathogens (Milosevic and Slusarenko, 1996). The elevated H_2O_2 levels have been proved to be associated with resistance mechanisms in a few plants and fruits (Sticher et al., 1997; Torres et al., 2003). In the presence of POD, H_2O_2 can be involved in the oxidation of phenolic compounds in the plant

against infection by pathogens (Chittoor et al., 1999). Our results showed that the H_2O_2 level in *Ya Li* pear fruit was significantly enhanced by the SA treatment. Therefore, enhancing H_2O_2 generation may be one part of the mechanisms of the SA-enhanced resistance in *Ya Li* pears.

PAL is associated with the biosynthesis of secondary metabolites, such as phytoalexins and phenolic compounds, which may directly inhibit the growth of pathogens (Milosevic and Slusarenko, 1996); POD participates in the wall-building process such as oxidation of phenols, suberization, and lignification of host plant cells during infection (Chittoor et al., 1999; Lurie et al., 1997). Chitinase and β-1,3-glucanase have been considered as key enzymes directly against pathogens in plant-disease interactions (Ji and Kuć, 1996; Schneider and Ullrich, 1994). The present study showed that activities of POD, PAL, chitinase and β-1,3-glucanase in the young fruit were all enhanced by SA spray on the trees (Figure 3). However, at harvest and during storage, activities of PAL, chitinase and β-1,3-glucanase in mature fruit with SA sprays during growth were still significantly higher than those in the control fruit. Similar phenomena were also observed in relevant studies on other plants (Schneider and Ullrich, 1994; Sticher et al., 1997; Meena et al., 2001; Qin et al., 2003). These results may imply that these defense enzymes exerting their functions in different ways in the defense system may be coordinately regulated by SA in *Ya Li* pear fruit and other plants.

Since PAL and POD can catalyze the biosynthesis of secondary metabolites and lignification in plant tissue, it is of some concern whether the increased activities of PAL and POD would reduce edible quality of the fruit by the SA treatment. Nevertheless, no remarkable change in edible quality of the fruit was observed after the SA treatment (data not shown). However, studies by Zhang et al. (2003) and Zainuri et al. (2001) showed that SA treatment could delay ripening and senescence of fruit after harvest.

Although H_2O_2 could contribute to enhancement of disease resistance in the plant, it could be scavenged off by antioxidant enzymes such as CAT and APX against harmful effects of excess H_2O_2 on the membrane system in plant tissues. Our studies showed that activities of CAT and APX in the young fruit were temporally reduced by the SA sprays, which may partly account for the increase in H_2O_2 level in the fruit. Similar results were also reported in studies of SA treatments on tobacco (Conrath et al., 1995) and other plant species (Sánchez-Casas and Klessig, 1994). Unlike CAT and APX, activity of GR, another type of antioxidant enzyme in the young fruit significantly increased on day 6 after the SA sprays. The fact that increase of GR activity occurred much later than increase of H_2O_2 level in fruit by the SA treatment agreed with previous studies in various SA-treated plant tissues/organs (Knörzera et al., 1999; Ganesan and Thomas, 2001). However, the role of GR in the induced response in the plant is still not very clear. These results may suggest that the activity of GR could be induced by the elevated level of H_2O_2, as a response of the cell's protective mechanisms to oxidative stress (Milosevic and Slusarenko, 1996). Therefore, H_2O_2 increased by pathogens or elicitors in plant tissues could serve as a second messenger acting downstream of a range of genes encoding the defense enzymes, such as PAL, chitinase and β-1,3-glucanase (Sticher et al., 1997), as well as the genes encoding some antioxidant enzymes such as GR (Ganesan and Thomas, 2001).

In conclusion, results from our studies indicate that foliar SA sprays may provide protection against postharvest diseases of *Ya Li* pear fruit in practice. Depending on its own efficacy, SA can be used in the orchard in combination with fungicides to obtain further protection against postharvest diseases of the fruit.

Acknowledgement

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