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Role of Salicylic Acid in Postharvest Physiology

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ABSTRACT

Salicylic acid (SA) belongs to a group of phenolic compounds widely distributed in plants and is now considered as a hormonal substance, playing an important role in plant physiology. SA has become more and more important in postharvest storage of fruit. Pre-harvest treatments with SA reduced lesion diameters on fruit caused by fungi, and induce β-1,3-glucanase, phenylalanine ammonia-lyase (PAL) and peroxidase (POD) activities during storage. SA shows direct fungitoxicity on fungi and significantly inhibits fungal growth and spore germination of the pathogen *in vitro*. SA can delay the ripening of fruit, probably through inhibition of ethylene biosynthesis or action. During postharvest ripening and softening of some fruit, the levels of SA in fruit tissues decline, lipoxygenase (LOX) activity increases, and this is associated with climacteric ethylene release. SA treatment maintains greater firmness, reduces chilling injury indices, and delays membrane lipid peroxidation in fruit during cold storage. The effect of SA on alleviating chilling injury of peaches during cold storage may be attributed to its ability to induce antioxidant systems and heat shock proteins or HSPs. Exogenous application of SA or methyl-salicylic acid may also induce the expression of many defense genes during fruit storage. Pre-storage or preharvest application of SA may provide a useful means of controlling post-harvest decays and extending fruit postharvest life during storage.

Keywords: chilling injury, fungal disease, fruit ripening, SA
Abbreviations: SA, salicylic acid

CONTENTS

INTRODUCTION

Salicylic acid (SA) is a ubiquitious plant phenolic compound involved in plant growth and development. When applied to plants, SA can produce a wide range of effects on many plant processes. In recent years, SA has been the focus of much attention because of its possible role in postharvest physiology and use for quality maintenance.

SA AND CHILLING INJURY

Most fruit ripen and deteriorate quickly at ambient temperature. Therefore, cold storage has been used as the main method to slow these processes as well as slow decay development, but chilling injury (CI) may limit the storage life of fruit, especially tropical and subtropical species, under low temperature. Chilling-injured fruit lose their ability to ripen normally and maintain firmness, and show increased susceptibility to rot and decay (Lurie and Crisosto 2005). Peach fruit in conventional cold storage at 0-1°C for up to 2 weeks normally suffer chilling injury, although this disorder is strongly dependent on cultivar and maturity at harvest (Morris 1982). Loquat fruit stored at 5°C for 39 days retained acceptable quality, but fruit stored at 0°C had chilling injury with symptoms including tissue browning and lignification, and a decrease in percentage juice (Cai *et al*. 2006). These chilling injury symptoms became more severe after fruit were moved to 20°C. The appearance of internal browning in the fruit flesh that occurs at low temperature may be related to tissue deterioration resulting from membrane lipid oxidation (Lurie and Crisosto 2005). Plant injury induced by chilling usually involves an imbalance between the production and elimination of active oxygen species (Wise 1995). Protection of cells from oxidative injury under stress is thought to be a major mechanism of resistance to plant stresses, and this resistance is likely to depend on the competence of the antioxidant system (Knorzer *et al*. 1999).

In recent years, studies have reported the effects of SA on chilling injury, showing that SA and MeSA treatments increased resistance to chilling in maize plants (Janda *et al*. 1999), tomato fruit (Ding *et al*. 2002), banana seedlings (Kang *et al*. 2003) and winter wheat (Tasgin *et al*. 2003). These observations suggest that SA could be linked to oxidative stress responses. Ding *et al*. (2002) reported that low concentrations of MeSA (0.1 mM) provided protection against chilling injury in tomato fruit. When MeSA concentrations higher than 0.1 mM were applied, negative effects were observed. In young maize plants, 0.5 mM SA provided protection against subsequent low temperature stress (Janda *et al*. 1999). The relative uptake from exogenous application as well as the effective concentration of SA alleviating chilling injury could vary depending on species and tissue type.

Han *et al*. (2002) reported tomato (*Lycopersicum esculentum* Mill.) and cucumber (*Cucumis sativus* L.) fruit im-

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mersed in 0, 0.001, 0.01 and 0.1 g/L SA solutions for 15 min, respectively, influenced chilling injury during cold storage (2 ±1°C). Cell membrane electrolyte leakage, Malondialdehyde (MDA) content and free proline content in tomato with 0.01 g/L and 0.1 g/L SA were lower than those of control. Immersion of cucumber in 0.001 g/L SA significantly decreased cell membrane electrolyte leakage and thiobarbituric acid-reactive-substance (TBARS) content when stored at chilling injury temperature as well as decreasing free proline content. A postharvest application of 1 mmol/L aqueous acetylsalicylic acid (ASA, a derivative of SA) to loquat fruit significantly alleviated chilling injury symptoms, inhibited accumulation of superoxide free radical, and reduced phenylalanine amonia lyase (PAL), catalase (CAT), and peroxide (POD) activities (Cai *et al*. 2006). Ascorbate acid (AsA) treatment impaired the accumulation of superoxide free radicals preventing chilling injury and lignification in loquat (Cai *et al*. 2006).

Wang *et al*. (2006) reported that treatment with SA was effective in alleviating chilling injury of peach fruit. Peach fruit were immersed in 0, 0.35, 0.7 and 1 mM SA solution for 5 min, respectively, stored at 0°C for 28 days, then moved to 20°C for 3 days to simulate shelf life. Chilling injury index, decay index (DI), firmness and TBARS content of fruit were measured during cold storage and at the end of shelf life. The results showed that only 1 mM SA significantly (P<0.05) maintained higher firmness and lower CI, DI, and TBARS content of fruit compared with the control. Reduced-to-oxidized ascorbate ratio (AsA/DHAsA) in 1 mM SA-treated fruit was 39%, 61%, and 55% higher than that in controls at the midpoint and end of storage, and after 3 days of shelf life, respectively. The reduced-to-oxidized glutathione ratio (GSH/GSSG) in SA treated-fruit was 68% higher than that in controls at the midpoint of storage. Ascorbate peroxidase and glutathione reductase activities of SA-treated fruit were significantly (P<0.05) greater than those of controls during cold storage.

Heat shock proteins (HSPs) are a group of conserved proteins induced or increasingly expressed in prokaryotes and eukaryotes in response to many environmental factors including heat and cold stresses and a variety of physical and chemical stimuli, including oxidative stress (Lindquist 1986). HSPs play a central role in the maintenance of cellular homeostasis through their chaperonin functions in folding and assembly of immature and damaged polypeptides or proteins (Boston *et al*. 1996). Wang *et al*. (2006) reported that before SA treatment heat shock protein 101 (HSP101) was not found in peach fruit, nor was it found in control fruit during or after cold storage. In contrast, at the midpoint of cold storage, HSP101 was expressed strongly in SA-treated fruit. At the end of storage and after 3 days of shelf life, expression of HSP101 was weak. In both SA-treated and control fruit, expression of HSP73 was found before, during, and after cold storage, but expression in SA-treated fruit was stronger than that in control fruit. Thus the effect of SA on alleviating chilling injury of peaches during cold storage may be attributed to its ability to induce antioxidant systems and HSPs.

The HSP70 family has essential functions in preventing aggregation and in assisting refolding of non-native proteins under both normal and stress conditions (Hartl 1996). Some family members of HSP70 including HSP73 are constitutively expressed. It was shown that HSP73 was constitutively expressed because HSP73 existed in peach fruit before cold storage (Wang *et al*. 2006). However, HSP73 was expressed more strongly in SA-treated fruit. These results suggest that SA can induce HSP101 expression and increase HSP73 accumulation in peach fruit to enhance chilling tolerance. Acquired stress tolerance in plants is often a result of various stress-response mechanisms that act coordinately or synergistically to prevent cellular damage and maintain cellular homeostasis. An increasing number of studies suggest that the HSP/chaperones interact with other stress-response mechanisms. The redox status of thiol-containing molecules is important to cellular functions such as the synthesis and folding of proteins (Wang *et al*. 2004). Therefore, at the middle of cold storage, HSP101 presence and enhanced HSP73 accumulation could be correlated to the increase in GSH content in SA-treated peach fruit. Several HSP genes were up-regulated in *Arabidopsis* under high light stress, implicating HSP in the antioxidative response in addition to their chaperonin function (Rossel *et al*. 2002). However, the involvement of HSP as regulators of cellular redox states and in other stress-response mechanisms in fruit, or plants generally, remains to be established.

SA AND FUNGAL DISEASE

Fungal diseases result in major losses of postharvest fruits and vegetables and may be controlled effectively by synthetic chemical fungicides. However, there is increasing public concern about health risks and environmental hazards associated with use of the fungicides, stimulating a search for alternative measures for disease control (Castoria *et al*. 2001; Fan and Tian 2001). In addition, the increasing possibility of pathogens developing resistance to specific chemicals is another good reason for seeking alternative control measures (Agrios 1997). Inherent resistance of plants to pathogens is based on both constitutive defense mechanisms such as pre-existing antimicrobial compounds and on inducible defense mechanisms. Induced disease resistance in plants by biotic or abiotic treatments can be a very effective, safe and economic strategy for controlling diseases (Droby *et al*. 2002). However, a thorough understanding of induced mechanisms, as well as the means to induce them, is lacking.

The signal molecule SA has a role in the endogenous signal transduction pathway that may lead to expression of resistance factors (Gaffney *et al*. 1993). In tomato fruit, an increase in endogenous salicylic acid concentration is related to defense responses (van Kan *et al*. 1995). Exogenous salicylic acid treatment may enhance host defense responses (Gaffney *et al*. 1993; van Kan *et al*. 1995) in fruit and vegetables, such as has been shown for kiwifruit (Poole and McLeod 1994), sweet cherry (Yao and Tian 2005), mango (Zeng *et al*. 2006), and pear (Cao *et al*. 2006).

SA is involved in some signal transduction systems which result in induction of particular enzymes catalyzing biosynthetic reactions forming defense compounds such as polyphenols, alkaloids or pathogenesis-related (PR) proteins (van Loon 1999). This can result in induction of defense responses and provide protection for plants from pathogen-attack (Kozlowski *et al*. 1999). SA, when exogenously applied, has been shown to move systemically through plants, resulting in the expression of a set of defense genes that are also activated by pathogen infection, thus inducing resistance against pathogens (Kozlowski *et al*. 1999).

The protection of fruit from invasion by fungal pathogens is largely due to activation of a highly coordinated biochemical and structural defense system that helps ward off the spread of the pathogens (Lawton *et al*. 1996). Chitinase and β-1,3-glucanase hydrolyze polymers that are thought to be involved in plant defense mechanisms against fungal infection (Collinge *et al*. 1993). PAL is a key enzyme in the first step of the phenylpropanoid pathway, which is related to the plant defense system (Dixon and Paiva 1995). POD activity produces the oxidative power for cross-linking of proteins and phenylpropanoid radicals resulting in reinforcement of cell walls against attempted fungal penetration (Huckelhoven *et al*. 1999). Thus SA treatment of fruit might have two effects. One, SA showed direct fungitoxicity and inhibited growth and spore germination of the fungus *in vitro*. Two, SA treatment increased activities of defense-related enzymes such as β-1,3-glucanase, PAL and POD during storage. These two aspects could be useful in the integrated control of postharvest diseases of fruit.

Wen *et al*. (2005), incubating the grape berry in SA-containing medium *in vivo*, showed that SA induced the accumulation of PAL mRNA and the synthesis of new PAL protein, and increased PAL activity. The activation of PAL

by SA was blocked by pretreatment with the protein synthesis inhibitor cycloheximide, the mRNA transcription inhibitor actinomycin D and the PAL inhibitor 2-amino-2-indanophonic acid (AIP). These results suggested that the development of resistance by SA reported by others may be attributed, at least partly, to SA-induced PAL gene expression and activation.

Yao and Tian (2005) demonstrated that pre-harvest treatment with SA significantly reduced disease incidence of sweet cherry fruit stored at 25°C. However, postharvest treatment with SA did not reduce disease incidence of fruit following inoculation with the brown rot fungus, *Monilinia fructicola*. This induced resistance and systemic protection gradually developed with time and was highly dependent on the stage of fruit ripeness. Rasmussen *et al*. (1991) also found that resistance induced by SA in younger cucumbers leaves was greater than that in older ones.

Yu and Zheng (2006) showed SA significantly inhibited spore germination of blue mold (*Penicillium expansum)* on apple *in vitro* when its concentration was increased to 1000 μg ml^{-1}, but it was not effective in controlling the disease *in vivo*. Simultaneous application of SA and *Cryptococcus laurentii* to wounds on the apple fruit surface showed that SA could improve the efficacy of *C. laurentii* against *P. expansum* in a concentration-dependent manner, being most effective at 10 μg ml^{-1} but less effective at higher or lower concentrations. Besides reducing blue mold incidence in the local wound sites, the combination of *C. laurentii* with SA at 10 μg ml^{-1} also had a synergistic effect on the induction of fruit resistance to the disease. However, SA at 100 μg ml^{-1} or above showed an adverse effect on the growth of *C. laurentii in vitro* and *in vivo*, suggesting that SA could enhance the biological activity of *C. laurentii* in apple fruit by inducing resistance to pathogens based on the antagonistic activity of *C. laurentii.*

'Ya Li' pear (*Pyrus bretschneideri*) trees sprayed three times with 2.5 mM SA around 30, 60 and 90 days after full bloom were harvested at commercial maturity (about 120 days after full bloom), inoculated with *Penicilium expansum*, and incubated at 20°C and 95-100% RH (Cao *et al*. 2006). The results showed that resistance to the pathogen was remarkably enhanced by the SA sprays. The SA spray applied around 30 days after full bloom notably enhanced accumulation of hydrogen peroxide in the young fruit. Meanwhile, activities of defense enzymes, including POD, PAL, chitinase and β-1,3-glucanase in the young fruit of SA-treated trees was 29.5%, 60.0%, 24.4% or 35.7% higher, respectively, than that in control fruit 4 days after SA application. Furthermore, after harvest, activities of PAL, chitinase and β-1,3-glucanase were still significantly higher from trees sprayed three times with SA than from control trees. Activities of 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 with diverse functions may be coordinately regulated by SA in pear fruit. Our study indicated that SA sprays on pear trees may provide protection against postharvest disease in practice and could be used as an alternative and economical approach to reduce application of chemical fungicides. Babalar (2007) studied the effect of SA treatment on fungal decay of fruit at growth stages of strawberry. The results showed treatment of plants at vegetative stage and fruit development stage followed by postharvest treatment of fruits with 1 and 2 mmol L^{-1} was the most effective strategy. Postharvest treatment with 4 mmol L^{-1} SA slightly damaged the fruits and was less effective than 2 mmol L^{-1} in retaining fruit quality.

SA AND FRUIT RIPENING

Pre- and postharvest exogenous SA or ASA application delayed the ripening of fruit, including banana, kiwifruit, apple (Srivastava and Dwivedi 2000; Zhang *et al*. 2003). Fruit softening, pulp:peel ratio, reducing sugar content, invertase and respiration rate decreased in SA-treated fruit. The activities of major cell wall degrading enzymes, such as cellulase, polygalacturonase and xylanase decreased in response to SA. The major enzymatic antioxidants, catalase and peroxidase, also decreased in response to SA during banana fruit ripening (Srivastava and Dwivedi 2000).

Ethylene is an important factor in the ripening of fruit. SA can interfere with the biosynthesis and action of ethylene in plants (Raskin 1992), so SA-induced ripening delay is probably through such inhibition. Both SA and ASA inhibited ethylene production of mung bean hypocotyls, apple and pear fruit tissue discs (Romani *et al*. 1989), and carrot cell suspension cultures (Roustan *et al*. 1990). Leslie and Romani (1986, 1988) showed that SA inhibited ethylene biosynthesis, suppressing ethylene production in suspension-cultured pear cells 1 to 3 h after addition of SA to the medium. This suppression was believed to be due to its inhibitory effect on the conversion of 1-aminocyclopropane-1-carboxylic acid (ACC) into ethylene. Production of ethylene from discs of apple flesh and peel and from apple seeds was also substantially suppressed after treatment with 0.1-20.0 mM SA for 6 h, the effect being concentration dependent (Fan and He 1998). SA has also been shown to inhibit both wound induced transcription and activity of ACC synthase (Li *et al*. 1992). Xu *et al*. (2000) observed inhibition of ACC oxidase activity in tissue sections of SA-treated kiwifruit, and similar results occurred with apple fruit tissue receiving ASA treatment (Fan *et al*. 1996). Zhang *et al* (2003) reported that ASA treatment suppressed ACC synthase and ACC oxidase activities and biosynthesis of ethylene, and hence retarded the climacteric rise of ethylene production in banana fruit.

The patterns of lipoxygenase (LOX) activity in fruit have been found to be closely related to fruit ripening and senescence. In apple, it was suggested that LOX might induce autocatalytic ethylene production through its involvement in lipid catabolism (de Pooter and Schamp 1989), and McRae *et al*. (1982) have provided evidence for involvement of superoxide free radicals in ethylene production. The key step attacked by superoxide free radicals was thought to be the conversion of ACC to ethylene. SA has also been shown to reduce superoxide free radical levels through its effect on SOD activity (Palva *et al*. 1994). Superoxide free radicals and hydroperoxides produced via the LOX pathway may not only directly take part in peroxidation of cell membrane lipids, but may also play a regulatory role in biosynthesis of ethylene (Lynch *et al*. 1985; Xu *et al*. 2000). SA has also been shown to suppress LOX activity in disks of kiwifruit, with a consequent reduction in the production of free radicals and ethylene biosynthesis (Xu *et al*. 2000). There is evidence for a positive correlation between LOX activity and ethylene biosynthesis in apple fruit tissue (Marcelle 1991), and free radicals produced by LOX activity have been shown to play a role in regulating the biosynthesis of ethylene (Kacperska and Kubacka-Zebalska 1989), and in postharvest ripening and softening of climacteric fruit such as apple (de Pooter and Schamp 1989) and tomato (Todd *et al*. 1990). SA may take part in regulation of ethylene formation also by restraining the increase in superoxide free radical production and cell membrane deterioration and tissue senescence induced by superoxide free radicals, rather than, or in addition to, its action on enzymes directly participating in ethylene synthesis.

Research on tobacco (Hennig *et al*. 1993) suggested that once the free SA content of plant tissue exceeds certain levels, dependent on growing and environmental conditions, excess amounts are converted into conjugated SA, considered to be the storage form of this compound in plants. Exogenous SA is reportedly able to induce salicylic acid glucosyltransferase (SA-Gtase) activity, and consequently promote the conjugation of SA into salicylic acid glucoside (SAG) (Yalpani *et al*. 1992). Zhang *et al*. (2003) demonstrated that ASA treatment resulted in an increase in total SA content of banana fruit. Accumulation of conjugated SA

was accompanied by a decrease in free SA content during the course of ripening in ASA-treated fruit. During postharvest ripening and softening of kiwifruit at 20°C, the levels of SA in fruit tissues declined. In fruit stored at 0°C with a reduced rate of softening, SA concentrations remained at relatively high levels. Externally-applied ASA helped maintain relatively high SA levels in the tissue, thus delaying the degradative activities of LOX and associated superoxide free radical production, and delaying the rise in ethylene production. If maintenance of endogenous levels of SA, and its conjugates, is important in fruit ripening and tissue senescence, then its effects in delaying ripening may be a result of a balance between growth promoting and catabolic effects. SA may have a general homeostatic role in plant development. A close relationship was found to exist between change in SA levels in the fruit tissues and the extent of fruit ripening and softening.

CONCLUSION AND FUTURE PROSPECTS

Application of SA at pre-harvest and post-harvest of fruit can alleviate chill injury and fungal disease during fruit storage, moreover, promote fruit ripening. However, the effect mechanism of SA remains unknown. Signal transduction and molecular biology of SA effect need to clarify, especially, effect of SA to respiration be worth to further studying. For widely application of SA and its derivatives, different fruit during pre-harvest or post-harvest should be studied.

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