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Effect of β-aminobutyric acid on cell wall modification and senescence in sweet cherry during storage at 20 °C

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ABSTRACT

The effects of postharvest β-aminobutyric acid (BABA) treatment on fruit firmness, pectin degrading enzymes, cell wall constituents and microstructural alterations of pericarp in sweet cherry fruit were investigated. BABA significantly delayed the decline of fruit firmness and inhibited the increase of membrane permeability and the accumulation of malondialdehyde in cherries. The BABA-treated fruit exhibited significantly higher contents of water-soluble pectin, CDTA-soluble pectin, Na_2CO_3-soluble pectin, total pectin, cellulose and hemicellulose than the control during storage. Activities of pectin degrading enzymes including polygalacturonase and pectinmethylesterase were markedly reduced by BABA treatment. Observations by scanning electron microscopy showed BABA maintained smooth cuticle and integrated structure of subepidermal cell in sweet cherry. These results suggest that the delay in fruit senescence by BABA may be due to depressed membrane permeability and malondialdehyde content, reduced activities of polygalacturonase and pectinmethylesterase, enhanced cell wall polysaccharides content, and integrated subepidermal cell structure in sweet cherry.

1. Introduction

Sweet cherry (*Prunus avium*) is popular for its attractive red color, good flavor and high value of nutrients. However, sweet cheery deteriorates rapidly during storage at ambient temperature. The main causes of sweet cherry deterioration are weight loss, softening and color changes (Serrano et al., 2009). These changes directly affect the quality of fruit, as well as their shelf-life, transportability and marketing. The effect of different storage methods or postharvest treatments on reducing decay, improving quality or inhibiting softening of sweet cherry has been investigated (Choi, Wiersma, Toivonen, & Kappel, 2002; Díaz-Mula, Serrano, & Valero, 2012).

The softening and textural changes during fruit ripening and storage are characteristic of particular fruit species (Brummell, Dal Cin, Crisosto, & Labavitch, 2004). Any rapid loss of desirable texture due to excessive softening will contribute to poor quality, postharvest loss, and negative consumer buying behavior. Softening of fruit during ripening and storage is associated to a series of molecular, biochemical, and physiological changes (Li, Xu, Korban, & Chen, 2010). Solubilization and depolimerization of cell wall constituents are a consequence of the coordinated action of cell wall-modifying enzymes, such as polygalacturonase (PG), pectinmethylesterase (PME) and cellulase (Cheng et al., 2009). It has been reported that postharvest treatments with calcium and naphthalene acetic acid could individually alter the degradation of cell wall during storage in strawberry (Figueroa et al., 2012). Amnuaysin, Jones, and Seraypheap (2012) found that hot water treatment reduced enzymes activities and gene expression associated with cell wall modification in banana. In addition, the decrease in cell turgor pressure is associated with fruit softening (Thomas, Shackel, & Matthews, 2008). The decrease in membrane permeability and malondialdehyde (MDA) content is involved in the maintenance of turgor pressure. Moreover, postharvest senescence in many fruits and vegetables can be investigated by measuring lipid peroxidation as estimated by MDA production (Hodges, Delong, Forney, & Prange, 1999). The integrated cell wall

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structure can delay the senescence process of fruit (Li et al., 2010). The softening process occurs in sweet cherry influences its shelf-life, facilitates the pathogen attacks and increases susceptibility to decay. In this sense, fruit treatments aiming to maintain preferable texture and extend shelf life of sweet cherry are urgently needed.

As a non-protein amino acid, BABA has been shown to induce resistance response to protect numerous plants against various pathogens (Ton & Mauch-Mani, 2004). In addition, BABA enhanced salt and drought stress tolerance in *Arabidopsis* (Jakab et al., 2005). Zhang, Wang, Zhang, Hou, and Wang (2011) reported that BABA provided an effective control of postharvest blue mould in apple fruit, which might be associated with its direct fungitoxic property and the induction of defense-related enzymes. However, no information is available about the effect of BABA on cell wall modification and fruit-softening enzymes in postharvest fruit. The objective of this study was to investigate the effects of BABA on delaying softening process in sweet cherry fruit stored at 20 °C, as well as to evaluate the microstructural alterations in sweet cherry pericarp.

2. Materials and methods

2.1. Fruit material and BABA treatment

Sweet cherry (*P. avium* L. cv. Hongdeng) fruit were hand-harvested at commercial maturity stage with healthy greenish stems from an orchard in Yantai, Shandong province, China. Fruit were transported to our laboratory in a refrigerated van at 2–5 °C within 6 h of harvest. The fruit were selected for uniformity of size, ripeness, and absence of physical injuries or infection. BABA was purchased from Sigma Chemical Co., Ltd.

The fruit were divided randomly into two groups with 300 fruit each. The first group was dipped into a solution of 30 mM BABA for 10 min at 20 °C based on our preliminary study. The second group was immersed into sterile deionized water for 10 min at 20 °C and served as control. All fruit were air-dried for approximately 1 h and stored at 20 °C with 95% relative humidity for 5 days. Fruit samples of 10 fruit were taken daily during storage for measurements of firmness, membrane permeability, and morphological and structural changes of sweet cherry pericarp. Another sample of 10 fruit was collected at every day intervals. Tissue samples were mixed and frozen immediately in liquid nitrogen and then stored at −80 °C for measurements of MDA content, cell wall polysaccharide composition, activities of PG (EC 3.2.1.15) and PME (EC 3.1.1.11). Each treatment was replicated three times and the experiment was conducted twice.

2.2. Determinations of fruit firmness

Fruit firmness was measured by the method described by Serrano, Guillén, Martínez-Romero, Castillo, and Valero (2005) with some modifications. For each fruit, 1 cm^2 of the skin was removed, and flesh firmness was individually recorded using a TA-XT2i texture analyzer (Stable Micro System Ltd., UK) on twenty fruit from each replicate with a 5 mm diameter probe at a speed of 1 mm S^{-1}, and the results were expressed in N.

2.3. Membrane permeability of sweet cherry pericarp and MDA content

The membrane permeability of cherry pericarp was expressed in terms of relative electrical conductivity with a conductivity meter (DDS-11A, China). Pericarp samples (2 mm thickness) of sweet cherry were excised from the equatorial zone of 10 fruit with a double edged blade. One gram sample were incubated in 40 mL double-distilled deionized water for 20 min at 20 °C and the initial electrolyte leakage (C1) was assessed. The solution was then boiled for 20 min and re-adjust to a volume of 40 mL before the final electrolyte leakage (C2) was measured. Relative electrical conductivity (%) = (C1/C2) × 100.

MDA concentration was measured according to the method of Hodges et al. (1999). Tissue samples were homogenized in 10 mL 10% trichloroacetic acid and centrifuged at 10,000*g* for 15 min. The supernatant was used to measure the absorbance at 532, 600 and 450 nm. The MDA concentration = $6.45 \times (A_{532} - A_{600}) - 0.56 \times A_{450}$. MDA content was expressed as nmol g^{-1} fresh weight (FW). Each experiment was repeated three times.

2.4. Measurement of cell wall polysaccharides

Cell wall polysaccharides of sweet cherry flesh were extracted according to the method of Zhou, Li, Yan, and Xie (2011) with some modifications. Five grams of frozen tissue was homogenized in 100 mL of 80% (v/v) boiling ethanol for 20 min. The homogenate was cooled and centrifuged at 3000*g* for 10 min, and the residue was thoroughly washed three times with 15 mL of 80% (v/v) ethanol. The residue containing cell-wall material was dissolved in 15 mL of 90% (v/v) dimethylsulfoxide at 4 °C for 15 h to remove starch. The crude cell wall extract were dried at 37 °C for 12 h. The dry sample was stirred for 6 h at 20 °C with 10 mL of 50 mM sodium acetate buffer (pH 6.5), and centrifuged at 10,000*g* for 10 min, the precipitate was re-extracted twice with sodium acetate buffer (pH 6.5). The three supernatants were combined and designated as water-soluble pectin (WSP). The residue was then extracted in 10 mL of 50 mM sodium acetate buffer (pH 6.5) containing 50 mM CDTA for 6 h at 20 °C with stirring and centrifuged at 10,000*g* for 10 min. This procedure was repeated twice. The three supernatants were pooled and designated as CDTA-soluble pectin (CSP). Afterwards, the remaining residue was dispersed in 10 mL of 50 mM Na_2CO_3 containing 2 mM CDTA with constant stirring for 6 h, and centrifuged at 10,000*g* for 10 min. The sediment was re-extracted twice by the abovementioned procedure. The three supernatants were combined as Na_2CO_3-soluble pectin (NSP). The remaining residue was extracted with 10 mL of 4 M NaOH containing 100 mM $NaBH_4$, mechanically shaken for 6 h, and centrifuged at 10,000*g* for 10 min. This step was repeated twice and the three supernatants were combined as hemicellulose-containing fractions. The remaining residue was washed with double-distilled deionized water until neutralisation, and was designated as cellulose-containing fractions. The pectin contents in the fraction were determined by the m-hydroxydiphenyl method using galacturonic acid (GA) as standard, and expressed as mg g^{-1} FW. The total pectin (TP) content was obtained by adding WSP, CSP, and NSP values. The contents of hemicellulose and cellulose fractions were estimated as glucose by using the anthrone method. Results were expressed as mg g^{-1} FW.

2.5. Enzymes assays

PG and PME were extracted by homogenizing 1 g of frozen tissue sample at 4 °C with 5 mL of 40 mM sodium acetate buffer (pH 5.2) containing 100 mM NaCl, 2% (v/v) mercaptoethanol, and 5% (w/v) polyvinyl polypyrrolidone. The extracts were then centrifuged at 25,000*g* for 20 min at 4 °C. Desalting by dialysis was unnecessary because of the low salt concentration in extraction buffer. The supernatants were used to determine enzyme activity. Duplicate activity assays were performed immediately.

PG activity was determined by the method of Andrews and Li (1995). The reaction mixture contained 40 mM sodium-acetate buffer (pH 5.2), 0.1% (w/v) polygalacturonic acid, 100 μL enzyme extract. The reaction was terminated with 2 mL of 10 mM $Na_2B_4O_7$

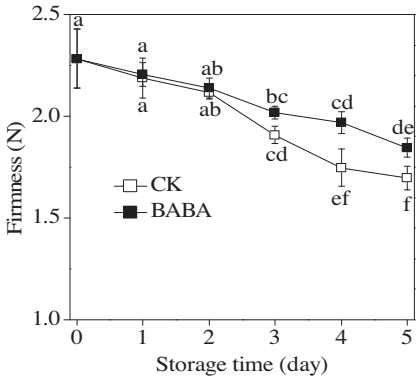

Fig. 1. Effect of BABA treatment on firmness in sweet cherry fruit during storage at 20 °C. Values are expressed as the mean ± SE of triplicate assays. Vertical bars represent the standard errors of the means. Data points carrying different letters indicate statistically significant differences ($P < 0.05$).

(pH 9.0), followed by addition of 100 μL 1% (w/v) 2-cyanoacetamide. Reducing sugars were assayed with meta-hydroxydiphenyl method using galacturonic acid as standard (Sigma Chemical Co., Ltd). Absorbance at 524 nm was measured. One unit of activity was defined as 1 μmol galacturonic acid released per mg of protein per hour.

PME activity was evaluated by an acid-base titration method described by Andrews and Li (1995). The reaction mixture consisted of 1 mL of enzyme extract and 10 mL 1% (w/v) citrus pectin (Sigma Chemical Co., Ltd). The standard curve, determined by titrating 1% (w/v) citrus pectin (Sigma Chemical Co., Ltd) with 0.5 M NaOH, was linear between pH 5–8. The difference between the initial pH and the final pH was used to calculate PME activity. One unit of enzyme activity was calculated as 1 mM NaOH consumed per mg of protein per hour.

Protein content in the enzyme extracts was determined using bovine serum albumin as a standard by the Bradford (1976) method. Specific activity of the enzymes was expressed as units per milligram of protein.

2.6. The structure of sweet cherry pericarp observed with scanning electron microscopy (SEM)

The sweet cherry pericarps were excised from the equatorial zone of the fruit with a double edged blade. The pieces of pericarp (3 mm length × 2 mm width × 1 mm thickness) were fixed in 2.5% glutaraldehyde (prepared in 0.1 M sodium phosphate buffer pH 6.8) at 4 °C for 24 h, rinsed three times with phosphate buffer and subsequently fixed with 2% osmium tetraoxide for 2 h at 20 °C. The samples were subsequently dehydrated in a graded series of ethanol solutions and then critical point dried with liquid CO_2 in a desiccator. The specimens were mounted onto aluminum specimen stubs using conductive silver paint, sputter coated with gold palladium, and observed with a scanning electron microscope (S-3000N, Hitachi High-Technologies Corporation, Japan) at 7 kV. Samples were taken from ten fruit respectively from each treatment.

2.7. Statistical analysis

All statistical analyses were performed with SPSS 11.0 (SPSS Inc., Chicago, Illinois, US). The data were analyzed by two-way analysis of variance (ANOVA) with treatment and storage time as factors. Mean separations were performed using Duncan's multiple range test, and differences at $P < 0.05$ were considered to be significant. In each figure, data points carrying different letters indicate statistically significant differences.

3. Results

3.1. Effect of BABA on sweet cherry fruit firmness

The firmness of cherry fruit decreased gradually during the storage (Fig. 1). The decrease of firmness was significantly inhibited by BABA treatment from the 2nd day of storage. The treatment of BABA was effective in maintaining the firmness of cherry fruit. At the end of storage, the firmness of BABA-treated fruit was 8.8% higher than that in control fruit.

3.2. Effect of BABA on membrane permeability of sweet cherry pericarp and MDA content

As shown in Fig. 2, the membrane permeability and MDA content in both control and BABA-treated cherries increased during storage. BABA-treatment significantly inhibited the increase of membrane permeability and MDA content during storage at 20 °C. At the end of storage, the membrane permeability and MDA content in BABA-treated fruit were 10.2% and 7.65%, respectively, lower than those in control fruit.

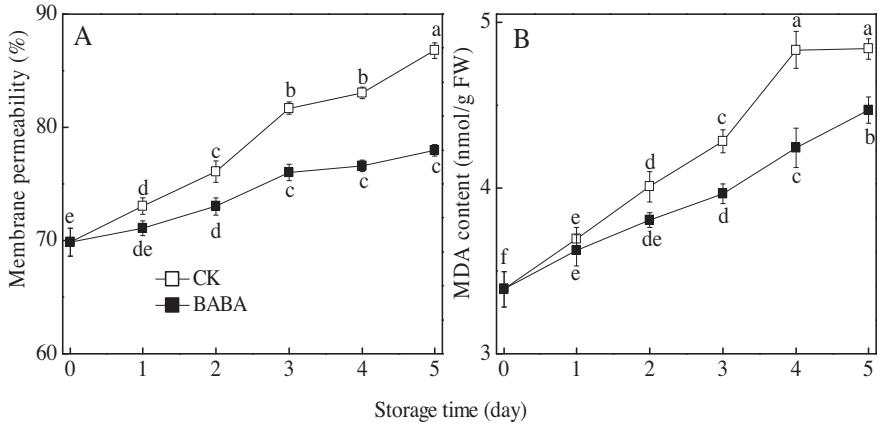

Fig. 2. Effect of BABA treatment on membrane permeability (A) and MDA content (B) in sweet cherry fruit during storage at 20 °C. Values are expressed as the mean ± SE of triplicate assays. Vertical bars represent the standard errors of the means. Data points carrying different letters indicate statistically significant differences ($P < 0.05$).

3.3. Effect of BABA on cell-wall polysaccharides in sweet cherry

As shown in Fig. 3, the contents of WSP, CSP, NSP, TP, cellulose, and hemicellulose showed varying degrees of reduction during the storage time. Fruit treated with BABA had higher levels of WSP, CSP, and NSP compared with the control during storage. The TP content in BABA-treated fruit was 36.9% higher than that in control fruit after 5 days of storage at 20 °C (Fig. 3D). In terms of hemicellulose and cellulose, dramatic reductions occurred in both control and BABA-treated fruit during storage time. However, BABA treatment delayed the decrease in their contents. The contents of hemicellulose and cellulose in BABA-treated fruit were 17.3% and 33.2%, respectively, higher than those in control fruit on the 3rd day of storage (Fig. 3E and F), which may contribute to delayed softening in sweet cherry fruit.

3.4. Effect of BABA on PG and PME activities

As shown in Fig. 4, PG activity increased gradually during initial 4 days of storage and decreased slightly afterwards. BABA treatment inhibited the increase of PG activity significantly after 3 days of storage (Fig. 4A). PME activity increased gradually with storage time. BABA treatment maintained lower PME activity in comparison with the control fruit after 2 days during storage. The PME activity was 17.73% and 24.63%, respectively, lower in BABA-treated fruit than those in control fruit on the 4th and 5th day (Fig. 4B).

3.5. Effect of BABA on morphological and structural changes in sweet cherry pericarp with scanning electron microscopy

The effects of BABA on the morphological and structural changes in sweet cherry pericarp are shown in Fig. 5. The epidermal tissue of the cherry is made up of small polygonal cells covered by a thin hydrophobic cuticle. The cuticle in BABA-treated fruit retained smooth, while the cuticle of control fruit had a lumpy appearance after 5 days of storage at 20 °C (Fig. 5A and B). The subepidermal tissue consisted of 2 to 3 layers of small cells with wide cell walls and without intercellular spaces. Under SEM observation, the cell wall of subepidermal tissue in control fruit had vague contour and irreversible deformation in cell wall was observed (Fig. 5C). In contrast, the subepidermal cells in BABA-treated fruit had distinct boundary and plump stereoscopic structure (Fig. 5D).

4. Discussion

Fruit softening, primarily due to cell wall degradation, is a common phenomenon that occurs during fruit ripening and storage. Increasing studies have shown that postharvest treatment,

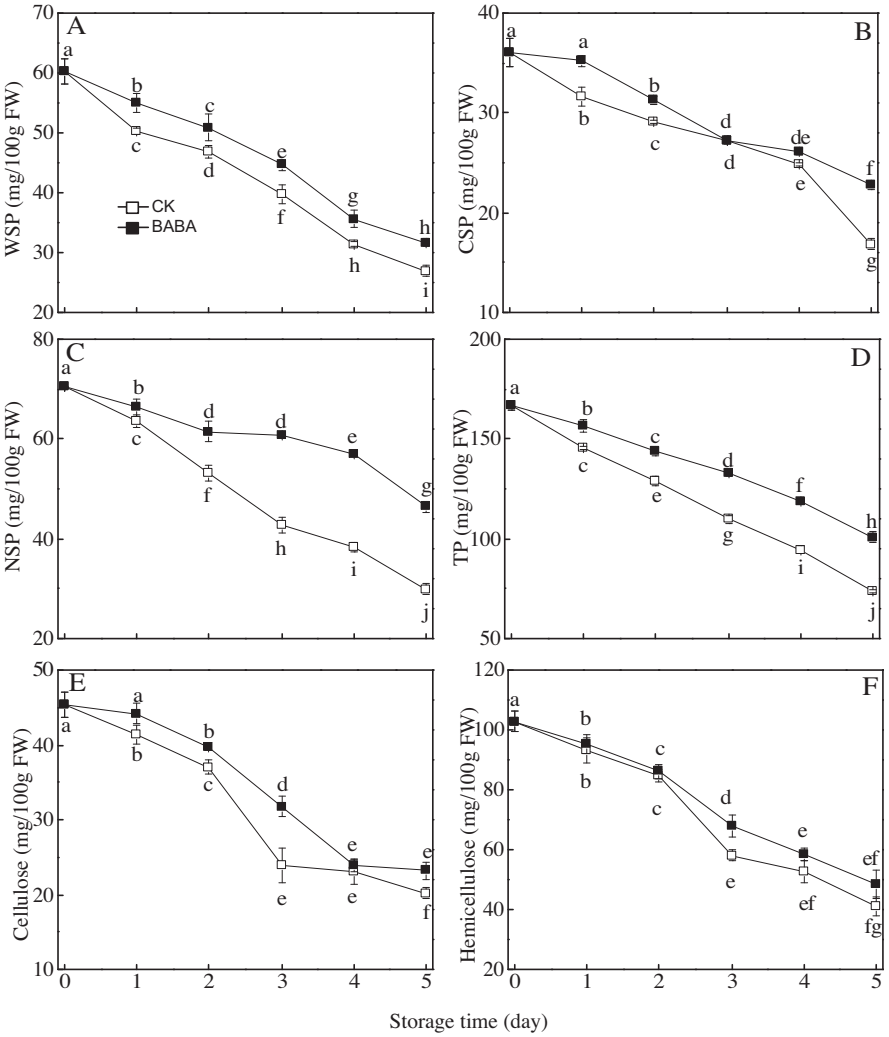

Fig. 3. Effect of BABA treatment on WSP (A), CSP (B), NSP (C), TP (D), cellulose (E), and hemicellulose (F) contents in sweet cherry fruit during storage at 20 °C. Values are expressed as the mean ± SE of triplicate assays. Vertical bars represent the standard errors of the means. Data points carrying different letters indicate statistically significant differences ($P < 0.05$).

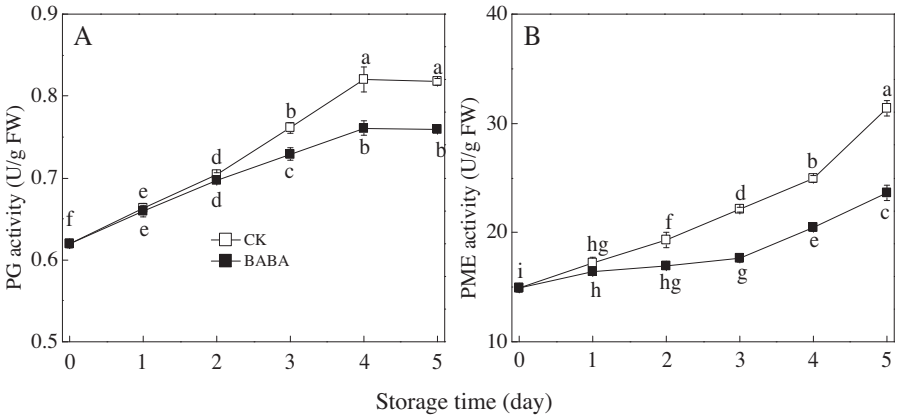

Fig. 4. Effect of BABA treatment on PG (A), PME (B) activities in sweet cherry fruit during storage at 20 °C. Values are expressed as the mean ± SE of triplicate assays. Vertical bars represent the standard errors of the means. Data points carrying different letters indicate statistically significant differences ($P < 0.05$).

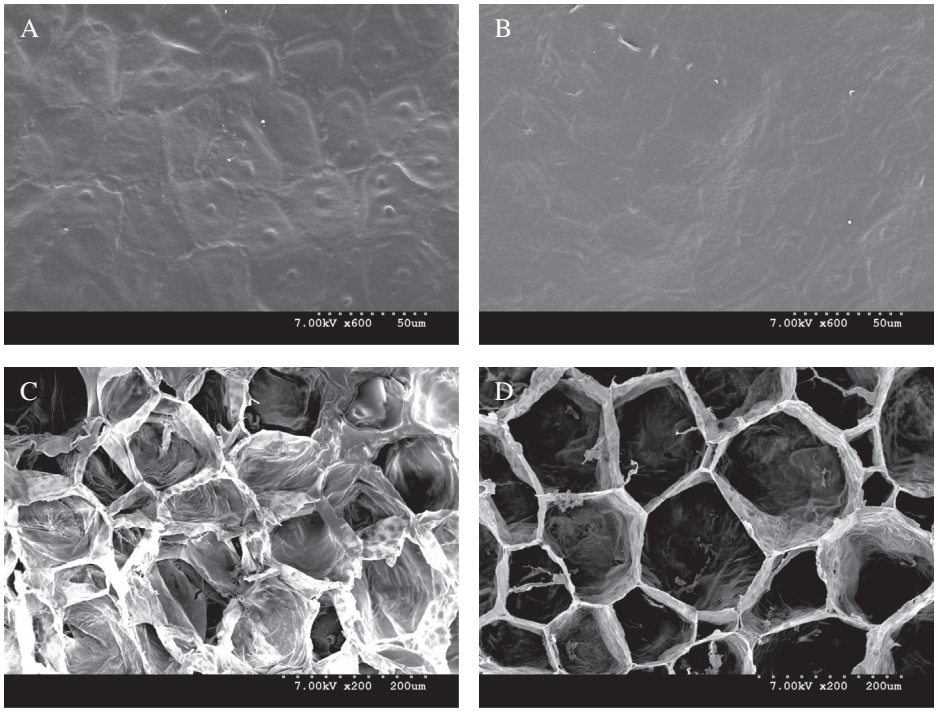

Fig. 5. Effect of BABA treatment on morphological and structural changes in sweet cherry pericarp observed with SEM. General view of epidermal and parenchymal tissue in control fruit (A and C) and BABA-treated fruit (B and D). Samples from ten fruit of each treatment were examined.

including $CaCl_2$, ozone or hot water, can delay softening in various fruits (Amnuaysin et al., 2012; Figueroa et al., 2012; Rodoni, Casadei, Concellón, Alicia, & Vicente, 2010). BABA has been shown to induce disease resistance to protect numerous plants against various pathogens (Ton & Mauch-Mani, 2004). Furthermore, in our study, we found that BABA was effective on delaying softening and retaining the texture in sweet cherry fruit.

As indicators of membrane damage, membrane permeability and MDA content are considered to be indirect measurements of membrane integrity and can reflect the loss of membrane integrity (Hodges et al., 1999). The reduction in the plasma-membrane integrity of fruit cells contributed to the loss of cell turgor, which was related to the decrease in the firmness of the fruit during storage (Khin, Zhou, & Yeo, 2007). MDA is one of the intermediate products of lipid peroxidation that is associated with senescence, and is often used as an index of cell oxidative damage (Li & Yu, 2001). Moreover, the accumulation of MDA causes the damage of plasma membrane and organelle of fruit cells. Zhou et al. (2011) found that the softening of pear fruit was accompanied by loss of membrane integrity. In the present work, the membrane permeability and MDA content increased with storage time, and the increase was effectively inhibited by BABA treatment (Fig. 2). This indicates that reduced membrane permeability and decreased MDA content may play positive roles in delaying fruit softening. Thus, the effect of BABA treatment on delaying the softening process of sweet cherry fruit could be attributed to reduced membrane permeability and MDA content.

Fruit softening is an irreversible process associated with increased depolymerisation of cell wall polysaccharides. Softening-related changes in cell wall polysaccharides including pectins, cellulose and hemicelluloses have been well documented in avocadoes (Wakabayashi, Chun, & Huber, 2000), peaches (Brummell et al., 2004) and strawberries (Figueroa et al., 2012). Generally, during fruit ripening, depolymerisation of cell wall polysaccharides

was accompanied by increase in water-soluble pectins, whilst the levels of water-insoluble pectins, hemicellulose and cellulose decreased (Li et al., 2010). In our study, for the purpose to better characterize the alteration of pectic substances during fruit senescence of harvested sweet cherry, we fractionated pectic polymers on the basis of their solubility in water (WSP), CDTA (CSP), or Na_2CO_3 (NSP), which represents the polyuronides loosely, ionically or covalently associated to the cell wall, respectively (Vicente, Ortugno, Powell, Greve, & Labavitch, 2007). We found all the three pectic fractions WSP, CSP and NSP decreased gradually during the storage (Fig. 3). It is because sweet cherry is a non-climacteric fruit that is commercially harvested at ripe stage, most of the solubilization and depolimerization of cell wall polysaccharides and significant fruit softening have occurred during pre-harvest on tree ripening (Andrews and Li (1995)). After harvest the fruit enters senescence process and softens progressively, the transition of water-soluble pectin substance to water-insoluble pectic acid due to the action of PE could account for the continuous decrease in WSP content during the storage. Cellulose-hemicellulose network in fruit cell wall are formed by hydrogen-bond and crosslinks between cellulose microfibrils associated to hemicellulose. The rigidity of cellular walls can be probably attributed to this network structure (Wakabayashi et al., 2000). Increasing evidences have shown that the breakdown of pectin and hemicellulose resulted in the disassembly of the cellulose-hemicellulose network and fruit softening (Cheng et al., 2009). In contrast to hemicellulose, moderate changes in the quantity of cellulose content occurred, which was ascribed to the fact that the crystalline nature of cellulose endows a high resistance to enzymatic degradation (Zhou et al., 2011). Taken together, our results suggest that the continuous degradation of WSP, NSP and hemicellulose played an important role in enhancing fruit softening of sweet cherry during storage, BABA treatment suppressed the depolymerization and degradation of the cell wall polysaccharides, thus delaying fruit softening.

Fruit softening is an irreversible process associated with cell wall disassembly due to the concerted action of suites of cell wall modifying enzymes. Pectin is one of the main constituents of primary cell walls and middle lamella of higher plant cells. The activities of the pectin degrading enzymes PG and PME have been found to be closely related to the degradation of pectins, which play a significant role in the softening changes in fruit and vegetable tissues (Rodoni et al., 2010). PME, the enzyme involved in pectin metabolism, catalyzes the demethylesterification of cell wall polygalacturonans to generate demethylated pectins that can be more easily hydrolyzed by PG (Micheli, 2001). PG hydrolyzes pectin acid along with the main chain of polygalacturonic acid, causing cell wall dissolution, and ultimately, fruit softening (Brummell et al., 2004). Furthermore, PG activity influences the integrity of the middle lamella, which modulates cell-to-cell adhesion and thus influences fruit texture. In this study, BABA treatment remarkably suppressed the increase of PG and PME activity (Fig. 4) and maintained higher levels of WSP, CSP, and NSP (Fig. 3) compared with the control during storage, which suggested that BABA suppressed degradation of pectins by inhibiting their degrading enzymes. Supapvanich and Tucker (2013) also found that softening process was delayed via the inhibition of cell wall degrading enzymes including PG and PME in muskmelon fruit treated with 1-methylcyclopropene, which confirmed that cell wall degrading enzymes play an important role in fruit softening. Therefore, our results indicate that the effect of BABA on delaying softening in sweet cherry fruit may be partially due to relatively lower activities of PG and PME in the treated fruit.

Fruit textural change during ripening and storage influences consumer preference, fruit storability, transportability, shelf-life, and response to pathogen attack. The microstructure of fruit pericarp in sweet cherry fruit plays a primary role in water regulation and protection from external stresses (Alonso, Tortosa, Canet, & Rodríguez, 2005; Yeats et al., 2012). In order to better understand the textural changes of sweet cherry fruit during storage, the microstructure of fruit pericarp in both BABA-treated and control cherries was observed with SEM, which enabled us to examine, over a wide range of magnifications, the morphological and structural alterations in sweet cherry fruit. Our results showed that fruit treated with BABA had a more smooth cuticle and integrated subepidermal cell structure than the control (Fig. 5). Alonso et al. (2005) also reported that calcium treatment maintained the microstructure of subepidermal tissue in sweet cherry fruit. Thus, our results suggest that BABA may delay fruit softening of sweet cherry by maintaining the microstructure of cells.

In conclusion, our results indicated that BABA can effectively delay fruit senescence and softening in postharvest sweet cherry fruit, possibly by depressing increases in membrane permeability and malondialdehyde content, inhibiting activities of polygalacturonase and pectinmethylesterase, enhancing cell wall polysaccharides content, and maintaining subepidermal cell structure in sweet cherry.

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