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Effect of heat treatment on strawberry fruit damage and oxidative metabolism during storage

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Abstract

Strawberries (*Fragaria* × *ananassa* Duch., cv. Selva) were heat-treated in an air oven (45 °C, 3 h). After treatment, the fruit were stored at 0 °C for 0, 7 or 14 d, and then held for 2 d at 20 °C. Respiration rate, hydrogen peroxide level, potassium leakage, antioxidant capacity, ascorbic acid, superoxide dismutase (SOD), ascorbate peroxidase (APX) and pyrogallol peroxidase (POD) activities were measured. Heat-treated fruit showed lower decay and less tissue damage than control fruit as judged by changes in ion leakage, respiration rate and pyrogallol peroxidase (POD). Heat-treated fruit also presented lower levels of H_2O_2 than control fruit during storage. In addition, antioxidant capacity was higher in heat-treated fruit both after 1 d at 20 °C or 7 d at 0 °C. Ascorbate peroxidase (APX) and superoxide dismutase (SOD) activity were not affected immediately after the treatment. However, higher activity was found in heat-treated fruit during storage, indicating that the heat treatment produced changes in the oxidative metabolism of the fruit. Interestingly, the main differences were not observed immediately after the treatment but during the storage. The differential responses observed in the case of heat-treated fruit during storage could protect the fruit against reactive oxygen species generated during senescence or pathogen attack.
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Keywords: *Fragaria* × *ananassa*; Antioxidant; Peroxidase; Ascorbate; SOD; Postharvest; ROS

1. Introduction

Heat treatments have been used in fruit postharvest technology for insect disinfestation, decay control, ripening delay and modification of fruit responses to other stresses (Lurie, 1998; Paull and Chen, 2000). Heat can cause stress in fruit tissues with different levels of severity depending mainly on the temperature and time of exposure, species and even variety (Lurie, 1998). It is possible to apply a moderate treatment at non-lethal temperatures resulting in both a reversible suspension of ripening and a reduction of fungal decay without noticeable changes in fruit quality (Lurie, 1998). In the case of strawberries, the treatment at 42–48 °C for 3 h in air delayed fruit ripening and reduced fungal attack (Civello et al., 1997; Vicente et al., 2002).

Heat treatment affects several aspects of fruit ripening, such as ethylene production and cell wall degradation, probably through changes in gene expression and protein synthesis (Lurie, 1998). During application of a thermal stress, the expression of most housekeeping and ripening related genes decreases noticeably while the expression of genes corresponding to heat shock proteins (HSP) increases (Picton and Grierson, 1988; Lurie et al., 1996). It has been proposed that this diversion of protein synthesis, more than the accumulation of HSPs by itself, could be responsible for some of the physiological effects of heat stress (Saltveit, 1997). In addition, heat shock may induce reactive oxygen species (ROS), followed by the production of oxygen radical scavengers such as superoxide dismutases (SOD), peroxidases

Abbreviations: AA, ascorbic acid; APX, ascorbate peroxidase; DPPH, 2,2-diphenyl-1-picrylhydrazyl; EAU, enzyme activity unit; HSP, heat shock protein; NBT, nitro blue tetrazolium; POD, pyrogallol peroxidase; PVPP, polyvinylpolypyrrolidone; ROS, reactive oxygen species; SOD, superoxide dismutase; TCA, trichloro acetic acid

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(POD) and catalases (Holmberg and Bülow, 1998; Mittler et al., 2004).

The accumulation of ROS also occurs during senescence and fruit ripening. During fruit storage, the presence of wounds and fungal development constitutes another stress source that increases the amount of ROS (Bolwell and Wojtaszek, 1997). Although these species could act as signals and secondary messengers for the activation of stress responses and defense pathways (Desikin et al., 2001; Knight and Knight, 2001), their intracellular level has to be kept under tight control since over accumulation produces cell damage and death (Bartoli et al., 1996; del Río et al., 1998).

The objective of this work is to determine if the benefits of applying a moderate short heat treatment on strawberries could be related to the modification of the fruit oxidative metabolism. Therefore, the effect of heat treatment on the accumulation of hydrogen peroxide and antioxidant protective mechanisms (antioxidant content, SOD, POD and APX) during strawberry fruit storage will be discussed.

2. Materials and methods

2.1. Plant material

Strawberries (*Fragaria* × *ananassa* Duch., cv. Selva) grown in greenhouses, were harvested with 75% superficial red color. Fruit having uniform size (corresponding to an approximate weight of 5–8 g) and color and free of external blemishes or infections were selected.

2.2. Heat treatment, storage and sampling

Five hundred and forty fruit were distributed in groups of 10, put in plastic trays as a single layer and covered with a PVC film. Then, 270 fruit were transferred to an air oven set at 45 °C for 3 h (heat-treated fruit). After treatment, the trays were placed at 0 °C for 0, 7 or 14 d and then transferred to 20 °C for 2 d. Corresponding controls were not thermally treated but directly stored at 0 °C for 0, 7 or 14 d and then transferred to 20 °C for 2 d. In both cases, heat-treated or control strawberries, 30 fruit were removed after 0, 7 or 14 d at 0 °C followed by 0, 1 or 2 d at 20 °C. The fruit were immediately analyzed or cut in four pieces, pooled, frozen in liquid nitrogen and stored at −80 °C until use. The whole experiment was repeated twice. As similar trends were found in both experiments, only the results from experiment II are shown.

2.3. Hydrogen peroxide

The method to measure H_2O_2 was adapted from Warm and Laties (1982). Frozen fruit tissue (15–20 g) was ground in liquid nitrogen and 0.5 g of the powder was extracted in 3 mL of ice-cold 5% (w/v) trichloro acetic acid (TCA). The crude extract was centrifuged at 9000 × *g* and 2 °C for 10 min. An aliquot of the supernatant (0.5 mL) was passed through a column containing 0.5 g of Dowex resin (1X1-100, chloride form, 1% cross-linked, Sigma–Aldrich, St. Louis, MO, USA) previously equilibrated with 5% (w/v) TCA. The column was washed with 3.5 mL of 5% TCA and all eluates were pooled. The content of H_2O_2 was measured by adding 0.5 mL of eluate to 0.5 mL of 0.5 mM luminol and 4.5 mL of 0.2 M NH_4OH (pH 9.0). This mixture (0.5 mL) was analyzed using a chemiluminescence meter. Chemiluminescence reaction was initiated by injecting 50 μL of 0.5 mM potassium ferricyanide in 0.2 M NH_4OH. Three extracts were prepared per each treatment and storage time analyzed, and the content of hydrogen peroxide of each extract was determined in triplicate.

2.4. Respiration rate

The CO_2 accumulation during storage was measured after 0, 7 and 14 d at 0 °C followed by 0, 1 and 2 d at 20 °C. Fruit (approximately 100 g) were enclosed in 500-mL jars and space head samples were withdrawn with a 1-mL syringe through a septum fitted in the jar lid. Gas analysis was performed using a gas chromatograph (Varian CX 3400, CA, USA) equipped with a CTR I column (Alltech, San José, CA, USA) and a thermal conductivity detector. The temperatures in the injector, column and detector were set at 120, 30 and 120 °C, respectively. Helium was used as the carrier and the gas flux was set at 0.33 mL s^{-1}. The respiration rate was expressed as the volume of CO_2 produced per kilogram of fruit in a second (mL kg^{-1} s^{-1}). Three independent replicates were prepared per each treatment and storage time analyzed, and the content of CO_2 was measured in triplicate.

2.5. Potassium leakage

Five discs (3 mm thick each, 3 g total weight) from the equatorial zone of the fruit were incubated in 25 mL of a solution containing 0.6 M sorbitol and 10 mM sodium phosphate (pH 6.0). The potassium content of the solution was measured by flame photometry (Metron Lab Instruments, London, England) at the beginning of the incubation and after 3 h. To evaluate the residual amount of potassium, the discs were frozen, thawed and ground with an Omnimixer (Sorvall Inc., Norwalk, CT, USA) in 25 mL of the solution described above. The obtained suspension was filtered through cheesecloth and centrifuged at 10,000 × *g* for 10 min and the potassium content of the supernatant was measured. The results were expressed as the percentage of potassium that leaked out of the tissues after 3 h of incubation. Three replicates were prepared per each treatment and storage time analyzed, and the potassium content was measured in triplicate.

2.6. Antioxidant capacity and ascorbic acid

The free radical scavenging capacity of strawberry fruit was tested according to the procedure described by Brand

Williams et al. (1995). Frozen fruit tissue (15–20 g) was ground and 1 g of the resultant powder was suspended in 6 mL of ethanol; the mixture was centrifuged at 9000 × *g* at 4 °C for 10 min. Three milliliters of the resulting supernatant were brought to 100 mL with water. Aliquots of this ethanolic extract (5, 10, 20, 40, 80 and 120 μL) were added to test tubes containing 3.9 mL of 0.025 g L^{-1} 2,2-diphenyl-1-picrylhydrazyl (DPPH) in methanol prepared daily. The absorbance at 515 nm was measured at different times with a spectrophotometer (DU650 Model, Beckman Instruments Inc., Berkeley, CA, USA) until the reaction reached a plateau. The percentage of remaining DPPH against the extract volume was then plotted to obtain the amount of sample necessary to decrease the initial DPPH concentration by 50%, which was defined as EC_{50}. The antioxidant capacity was expressed as $EC_{50}{}^{-1}$.

The extraction and measure of ascorbic acid was adapted from Doner and Hicks (1986). About 5 g of fruit was ground with 20 mL of 3% (w/v) citric acid in an Omnimixer (Sorvall Inc., Norwalk, Conn, USA). Immediately, the extract was centrifuged at 10,000 × *g* at 4 °C for 10 min. Aliquots of the supernatant were filtered through a cellulose membrane and used for ascorbic acid determination. The measurements were done by HPLC in a Waters chromatograph (Waters Associates Inc., Mildford, MA, USA), using a reverse phase column (Ultrasphere ODS of 250 mm × 4.6 mm; Beckman Instruments Inc.). Acetonitrile–water (30:70; 0.01 M K_2HPO_4 pH 4.3) was used isocratically as the mobile phase at a flux of 0.033 mL s^{-1}. The detection was performed spectrophotometrically at 210 nm, and the results were expressed as grams of ascorbic acid per kilogram of fruit (g kg^{-1}).

Three replicates were prepared per each treatment and storage time analyzed, and the antioxidant capacity and content of ascorbic acid were measured in triplicate.

2.7. Pyrogallol peroxidase activity (POD)

The POD activity measurement was adapted from Jiménez et al. (1997). Approximately 5 g of fresh fruit was homogenized for 2 min in an Omnimixer with 20 mL of buffer (0.02 M Na_2HPO_4, 0.03 M NaH_2PO_4, 10 g L^{-1} polyvinylpolypyrrolidone (PVPP), 1 mM PMSF, 0.1% (v/v) Triton X-100, 1 M NaCl, pH 7.0). The suspension was centrifuged at 10,000 × *g* for 10 min; the supernatant was saved and used to measure peroxidase activity with pyrogallol as substrate at 430 nm. The reaction mixture contained 50 mM phosphate buffer pH 6.5, 8 mM H_2O_2, 45 mM pyrogallol and 300 μL of extract, in a total volume of 3.0 mL. The enzyme activity was measured at 30 °C; one enzyme activity unit (EAU) was defined as the amount of enzyme that produced an OD increase of 1 min^{-1}. The results were expressed as EAU per kilogram of fresh fruit (EAU kg^{-1}). Two extracts were prepared for each treatment and storage time analyzed and each extract was measured twice.

2.8. Superoxide dismutase activity (SOD)

The activity of SOD was measured according to Giannopolitis and Ries (1977), with slight modifications. Five grams of frozen tissue were ground with a mortar and pestle in 16 mL of buffer (100 mM sodium phosphate pH 7.8, 1 M NaCl, 1 mM PMSF, 0.1 mM EDTA, 0.1% (v/v) Triton-X 100, 10 g L^{-1} PVPP) and the obtained suspension was stirred for 1 h. The homogenate was centrifuged at 10,000 × *g* for 10 min and the supernatant was used for assaying the enzyme activity. All the steps during extract preparation were carried out at 0–4 °C. The enzyme assay mixture contained 50 mM sodium phosphate buffer pH 7.8, 13 mM methionine, 75 μM nitro blue tetrazolium (NBT), 2 μM riboflavin, 0.1 mM EDTA and 300 μL of enzyme extract, in a total volume of 3.0 mL. The mixture, prepared into uniformly transparent test tubes, was then homogenized and placed 50 cm below four 15 W fluorescent lamps at room temperature for 15 min. The reaction was started or stopped by switching on or off the lamps, respectively. Then, the absorbance of the assay mixture was measured at 560 nm. Non-irradiated assay mixtures did not develop color and were used as control. The mixture lacking enzyme extract developed maximum color, which decreased as increasing amounts of the extract were added. One enzyme activity unit (EAU) was defined as the amount of enzyme that inhibited the photoreduction of NBT by 10% under the assay conditions. The results were expressed as EAU per kilogram of fresh fruit (EAU kg^{-1}). Two extracts were prepared for each treatment and storage time analyzed and each extract was measured twice.

2.9. Ascorbate peroxidase activity (APX)

APX activity measurement was adapted from Jiménez et al. (1997). Approximately 5 g of frozen fruit were ground with 16 mL of extraction buffer (100 mM sodium phosphate pH 7.0, 0.1% (v/v) Triton X-100, 1 M NaCl, 10 g L^{-1} PVPP) using an Omnimixer. The suspension obtained was stirred for 1 h and then centrifuged at 10,000 × *g* for 10 min, and the supernatant was used for assaying the enzyme activity. All the steps during the extract preparation were carried out at 0–4 °C. The activity was assayed at 30 °C in a mixture containing 100 mM sodium phosphate buffer pH 7.0, 2 mM ascorbic acid, 4 mM H_2O_2 and 500 μL of enzymatic extract in a final volume of 3.0 mL. The reduction of absorbance at 290 nm was followed; one APX enzyme activity unit (EAU) was defined as the amount of enzyme that produced an OD reduction of 1 min^{-1} in the assay conditions. The results were expressed as EAU per kilogram of fresh fruit (EAU kg^{-1}). Two extracts were prepared for each treatment and storage time analyzed, and each extract was measured twice.

2.10. Statistical analysis

Experiments were performed according to a factorial design. Data were analyzed by ANOVA and the means

were compared by a Fisher test at a significance level of 0.05.

3. Results and discussion

3.1. Potassium leakage and respiration rate

Both potassium leakage and respiration rate depend on fruit tissue integrity, and an increase of these parameters should be expected at the end of ripening or when the fruit is exposed to severe stress conditions (e.g. high or low temperatures) (Kahl, 1974). Immediately after the treatment, no differences in potassium leakage between control and heat-treated fruit were found (Fig. 1A). During incubation at 20 °C for 1 or 2 d, an increase of potassium leakage was found in both control and heat-treated fruit. However, the leakage was considerably higher in the case of control fruit. A similar trend was observed when the fruit were stored at 0 °C for 7 d and then transferred to 20 °C. After 14 d at 0 °C, heat-treated fruit also showed lower potassium leakage, and the difference remained for 1 d at 20 °C.

In the absence of refrigerated storage, the respiration rate increased after 2 d at 20 °C and there were no significant differences between control and heat-treated fruit (Fig. 1B). After 7 d at 0 °C and 2 d at 20 °C, control fruit had higher respiration rate than heat-treated fruit. A similar result was obtained after 14 d at 0 °C followed by 1 or 2 d at 20 °C. The respiration of the fruit held at 0 °C for 14 d was higher than at harvest or after 7 d at 0 °C.

The moderate heat treatment used (45 °C for 3 h in air) did not cause damage to the fruit (data not shown). On the contrary, the treatment delayed ripening, and the progress of decay that is characteristic at the end of ripening and the beginning of fruit senescence, in agreement with previous reports (Civello et al., 1997; Vicente et al., 2002). In absence of cold storage, 25% of control fruit had fungal infections after 2 d at 20 °C, while only 5% of heat-treated fruit decayed. After 7 d at 0 °C and 1 d at 20 °C, 25% of control fruit showed infection while no treated fruit decayed (data not shown). Potassium leakage from excised tissue into an isotonic aqueous solution is a measure of increased membrane permeability (King and Ludford, 1983). Both, the lower potassium leakage and the reduced respiration rate found in heat-treated strawberries during storage at 20 °C indicated a reduction in tissue damage. Therefore, the integrity of fruit membrane components (plasmalemma and/or tonoplast) was protected by heat treatment. A similar protective effect was observed in tomato; heat-treated fruit showed less ion movement across the membranes and an enhanced resistance against chilling-induced damage (Salveit, 2005).

3.2. Hydrogen peroxide

The hydrogen peroxide level increased during both storage at 0 °C and the holding period at 20 °C. Storage of fruit at 20 °C for 1 d caused a large increase in hydrogen peroxide compared to the level at harvest (0 d at 0 °C) (Fig. 2). The increase was almost 2.5-fold in control fruit and somewhat less than 2-fold in heated fruit. Storage at 0 °C for 7 d did not change the hydrogen peroxide level from that at har-

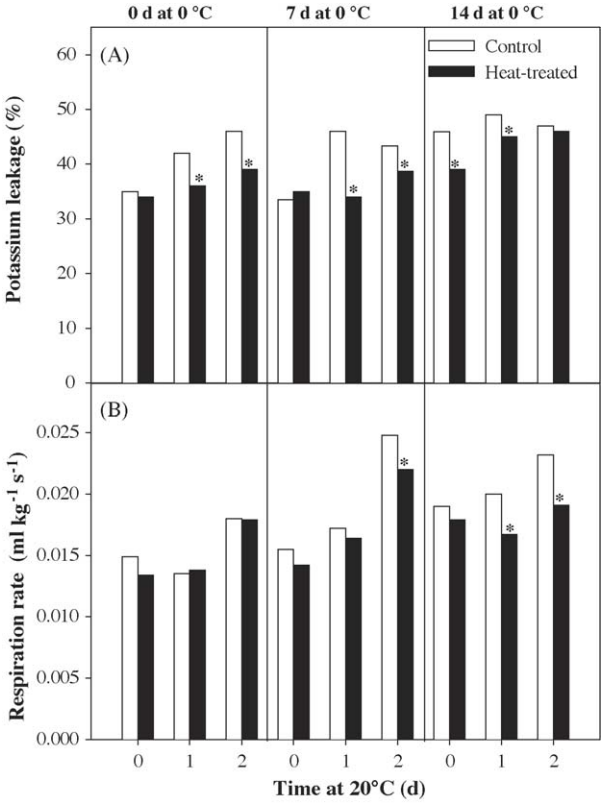

Fig. 1. Potassium leakage (A) and respiration rates (B) in control and heat-treated strawberries (45 °C, 3 h) during storage at 0 and 20 °C. Bars with symbol (*) are significantly different from the corresponding control ($P = 0.05$).

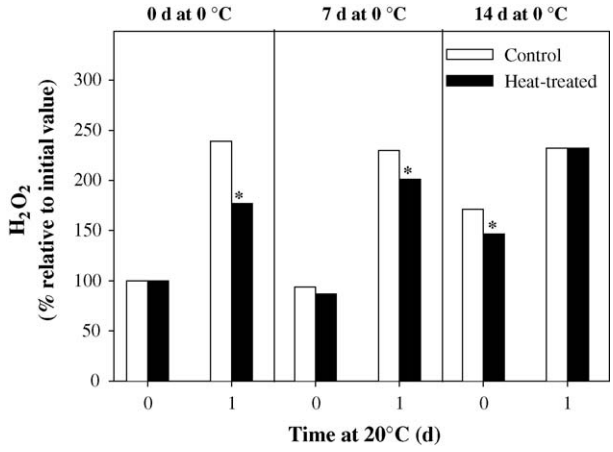

Fig. 2. Effect of heat treatment (45 °C, 3 h) on accumulation of hydrogen peroxide in strawberries during storage at 0 and 20 °C. The asterisk (*) indicates a significant difference in H_2O_2 content between the heat-treated fruit and the corresponding control ($P = 0.05$).

vest, while after 14 d at 0 °C the levels of both control and heated fruit were elevated, with control fruit levels rising more than heated fruit. One day of holding at 20 °C after both 7 and 14 d at 0 °C caused an increase in hydrogen peroxide, though generally heated fruit accumulated less than the control.

The increase of ROS has been frequently observed during senescence, pathogen attack or under stress conditions, and these compounds have been involved in the destruction of plant cells through peroxidation of lipids in the plasma membrane or the initiation of programmed cell death (Thompson et al., 1987; Greenberg, 1996). The accumulation of ROS is one of the processes that contribute to loss of membrane integrity and membrane-bound enzyme activities (Bartoli et al., 1996). The increase of H_2O_2 and lipid hydroperoxides has been reported during fruit ripening and senescence (Thompson et al., 1987; Lacan and Baccou, 1998). In this sense, the reduction of fruit damage observed in this work in heat-treated strawberry fruit could be partly due to the lower increase of hydrogen peroxide content during storage at 0 or 20 °C.

3.3. Antioxidant capacity and ascorbic acid

No significant differences were observed immediately after the treatment, but after 1 d of storage at 20 °C, heat-treated fruit showed higher antioxidant capacity than the control (Table 1). After 7 d of refrigerated storage, heat-treated fruit had higher antioxidant capacity than control fruit, and this difference remained for 1 d after the fruit was transferred to 20 °C. After 14 d at 0 °C, there were no differences between control and heated fruit. Therefore, the antioxidant capacity was, in general, higher in heat-treated fruit, which would help the fruit to cope with the overproduction of ROS species produced at the end of ripening.

Ascorbic acid is one of the most abundant antioxidants present in strawberry fruit. Just after treatment, heat-treated fruit showed higher levels of ascorbic acid than control fruit (Table 1); however, no difference was detected during the holding at 20 °C. After 7 d at 0 °C, the content of ascorbic acid was higher in heat-treated than in control fruit, and the difference was also detectable after 1 d at 20 °C. After 14 d at 0 °C, the content of ascorbic acid was similar in control and treated fruit, and only slight differences were found after 1 d of storage at 20 °C. An increase of ascorbic acid has been reported in some strawberry cultivars (e.g. Oso Grande) during cold storage (Cordenunsi et al., 2005). In this case, no ascorbic acid accumulation was observed in control or treated fruit during storage at 0 °C. Though, a higher level of ascorbic acid was found in response to heat treatment, which contributed to the higher total antioxidant capacity found in treated fruit.

3.4. Activity of pyrogallol peroxidase (POD), superoxide dismutase (SOD) and ascorbate peroxidase (APX)

The activity of these enzymes, related to oxidative metabolism, was measured in heat-treated and control fruit. After treatment, no difference in POD activity was found (Fig. 3A). During the holding time at 20 °C, POD activity increased in all the fruit, though heat-treated fruit showed lower enzyme activity than control fruit. After 7 or 14 d at 0 °C plus 1 or 2 d at 20 °C, heat-treated fruit had lower POD activity than control fruit. Peroxidases are widely distributed in the plant kingdom and have been shown to participate in different physiological processes like lignification and wound healing (Mäder and Fussi, 1982; Wakamatsu and Takahama, 1993). Biles and Martin (1993) reported a role for POD in damaged tissues, describing its participation in the cross-linking of the cell wall and in tissues infected with pathogens. Therefore, the increasing POD activity found in both control and treated strawberry fruit would reflect the progress of tissue damage during storage, and the lower POD activity found in heat-treated fruit could indicate that these fruit experienced less damage than the control. A similar reduction of POD activity, along with the reduction of PPO, has been found in lettuce after heat treatment application (Loaiza-Velarde et al., 1997).

The overproduction of ROS is an intrinsic feature of senescence and fruit ripening (Buchanan-Wollaston et al., 2003; Jimenez et al., 2002). The enzyme SOD is involved in the elimination of superoxide radicals, and then it contributes to protect the cell from damage due to an excessive radical accumulation (Sen Gupta et al., 1993; Van Breusegem et al., 1999). In this work, an increase of SOD activity was found in all the fruit during holding at 20 °C (Fig. 3B). In

Table 1
Change in antioxidant capacity and ascorbic acid concentration in control (C) and heat-treated fruit (T; 45 °C, 3 h) during storage at 0 and 20 °C

		0[a]			7[a]			14[a]		
		0[b]	1[b]	2[b]	0[b]	1[b]	2[b]	0[b]	1[b]	2[b]
Antioxidant capacity (EC_{50}^{-1})	C	0.117	0.105	0.122	0.104	0.100	0.122	0.109	0.112	0.136
	T	0.124	0.133*	0.129	0.132*	0.139*	0.123	0.127	0.125	0.144
Ascorbic acid ($g\,kg^{-1}$)	C	1.170	1.045	1.110	1.056	1.123	0.974	1.083	0.771	0.888
	T	1.425*	1.007	1.090	1.287*	1.211*	0.971	1.118	0.884*	0.936

The asterisk (*) indicates that the value is significantly different from the corresponding control ($P = 0.05$).
[a] Time at 0 °C (d).
[b] Time at 20 °C (d).

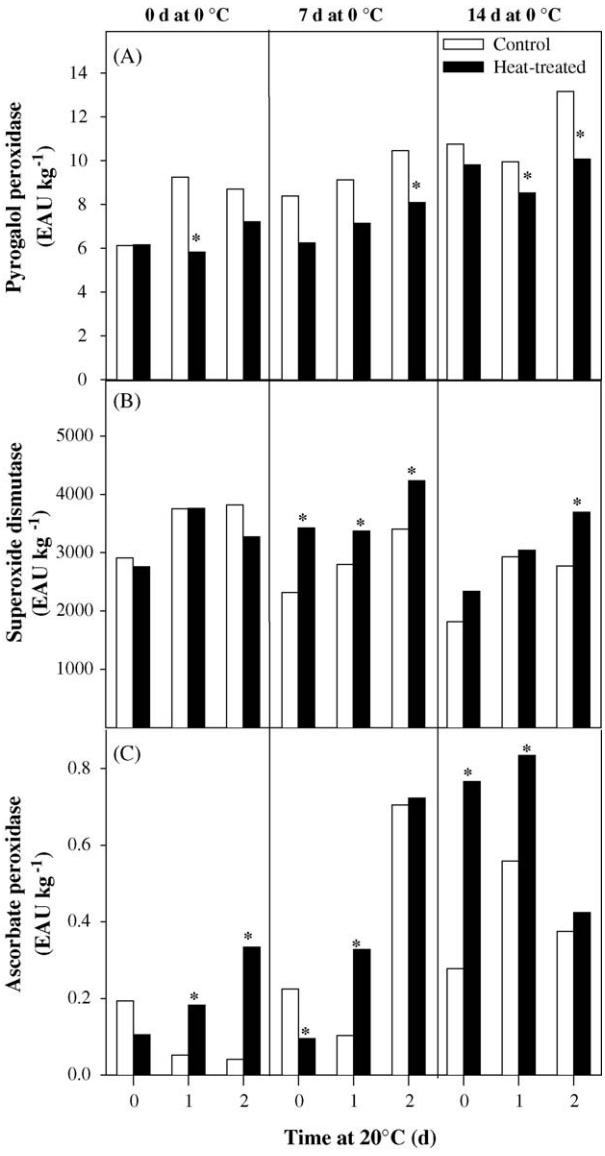

Fig. 3. Pyrogallol peroxidase (A), superoxide dismutase (B) and ascorbate peroxidase (C) activity in control and heat-treated strawberries (45 °C, 3 h) during storage at 0 and 20 °C. Bars with symbol (*) are significantly different from the corresponding control ($P = 0.05$).

addition, SOD activity of control fruit decreased during storage at 0 °C (0, 7 and 14 d at 0 °C). Once the heat treatment was finished, control and heat-treated fruit had similar SOD activity (Fig. 3B). When the fruit were held at 20 °C, the SOD activity increased in both control and heat-treated fruit, but no significant differences between them were observed. However, after 7 d at 0 °C the heat-treated fruit had higher SOD activity and the difference remained after 2 d at 20 °C. After 14 d at 0 °C plus 2 d at 20 °C, heat-treated fruit showed higher SOD activity than the respective control. Though no differences were observed immediately after the treatment, higher SOD activity was found in heat-treated fruit during storage. The increased SOD activity could contribute to avoiding or delaying the accumulation of superoxide radicals during storage and then to decrease tissue damage in heat-treated fruit.

The accumulation of ROS can be partly counteracted by the ascorbate–gluthatione cycle, where APX has an important role (Asada, 1994). After the treatment, no significant differences in ascorbate peroxidase activity were found (Fig. 3C). However, the heat-treated fruit showed higher APX activity after 1 or 2 d at 20 °C. After 7 d at 0 °C, heat-treated fruit presented lower APX activity than control fruit, but this reverted after 1 d at 20 °C. Instead, after 14 d at 0 °C the APX activity was higher in heat-treated fruit and the difference remained after being transferred to 20 °C for 1 d. Lower activity was found after 2 d at 20 °C in both heat-treated and control fruit, and no difference between them was detected. In those cases where the content of hydrogen peroxide was lower in heat-treated fruit (Fig. 2), a higher APX was found (Fig. 3C). The induction of APX in response to heat treatments has been reported by Sato et al. (2001), who found an increase of APX activity in heat-treated rice seedlings. In *Arabidopsis*, the expression of *apx1* gene is induced by heat stress as well as oxidative stress, and a putative heat-shock cis element was found in its promoter (Storozhenko et al., 1998). Since heat-treated fruit showed higher level of APX activity, this could lead to a higher protection against ROS.

4. Conclusion

Postharvest metabolism of strawberry fruit is deeply modified by the application of a moderate heat treatment, which delayed fruit ripening and decreased fruit decay. The heat-treated strawberry fruit had a better general appearance, and showed a lower increase in potassium leakage, respiration rate and POD activity indicating lower tissue damage and disruption. The effects could result from the partial decay control achieved in heat-treated fruit. These fruit also displayed higher levels of both enzymatic and non-enzymatic protecting mechanisms against reactive oxygen species, which are known to rise during aging and pathogen attack.

The non-damaging heating conditions caused a moderate stress in the fruit that mobilized antioxidant responses. Fruit antioxidant defenses such as ascorbic acid and total antioxidant capacity increased or remained high in treated fruit during 1 week of refrigerated storage. In addition, the activity of APX and SOD, two of the enzymes involved in ROS detoxification, was higher in heat-treated fruit. Interestingly, the main differences between control and heat-treated fruit were not observed immediately after the treatment, but later during storage probably when the fruit was exposed to secondary stresses.

The results found in this work suggest that the benefits of applying a short heat treatment on strawberry fruit quality and shelf-life could be partly due to the higher protection against oxidative molecules generated during fruit senescence or pathogen attack.

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