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Effects of Nitric Oxide on Fatty Acid Composition in Peach Fruits during Storage

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Peach (*Prunus persica* (L.) Batsch., cv. Feicheng) fruits were fumigated with 0, 5, 10, and 15 μL/L nitric oxide (NO), and the effects of NO on rot index, ion leakage, and the lipid compositions in peach fruits were studied. The results showed that the treatment with 10 μL/L NO significantly increased the contents of the palmitoleic (16:1), oleic (18:1), and linolenic (18:3) acid in phospholipid (PL), while it decreased the contents of linoleic (18:2) acid in PL. The content of monoacylglycerol (MG) in fruit fumigated with 10 μL/L NO were higher than in the control but lower in fruit fumigated with 15 μL/L NO. The treatment with 10 μL/L NO increased the content of MG, decreased the contents of diacylglycerol (DG) and triacylglycerol (TG). However, there was no significant difference in the contents of free fatty acid (FFA). The compositions of MG, DG, TG, and FFA in peach fruit were also changed by the treatment with 10 μL/L NO. It could preserve the content of 18:3, maintain the integrity of membrane, and prevent the softening and rotting of peach fruit. The content of 18:1 was detected in PL and FFA but not in MG, DG, and TG. This might be due to the different compositions of MG, DG, and TG.

KEYWORDS: Fatty acid composition, lipid peroxidation, nitric oxide, peach fruit, *Prunus persica*

1. INTRODUCTION

As the major components of biological membranes, lipids form the hydrophobic barrier that is critical to life. The physical state and composition of lipids influence both structural and functional properties of biological membranes (*1*). It has also been proven that the associations between membrane lipids and proteins, enzyme activity, transport capacity, and permeability are all affected by the composition and phase properties of the membrane lipids (*1*−*4*). Senescence is an important physiological process of plant tissues, and lipid peroxidation has been considered as a mechanism accelerating progressive deterioration of membrane integrity (*5*). Peroxidation led to increased gel-phase formation and losses in membrane functionality (*6*). It was proposed that linolenic acid (18:3) played an important role during senescence of tomato fruit (*7*). The changes of the membrane, including fatty acid composition and membrane deterioration in senescing peach, was reported (*8*). Although some metabolism of fatty acids and lipids in plants has been elucidated, the mechanism of lipid metabolism still is not fully understood because of its complexity.

There is growing evidence that nitric oxide (NO) could prolong the shelf life of fresh horticultural products (*9*−*11*), take part in the physiological and pathological processes of plants (*12*−*15*), and affect biosynthesis of ethylene in fruit (*16*). However, previous research has failed to consider the effect of NO on lipid peroxidation in fruits during ripening. In the present work, we studied the fatty acid composition and changes of the lipids in the mesocarp tissues of peach fumigated with NO with the aim of studying the effects of NO on lipid peroxidation leading to fruit senescence.

2. MATERIALS AND METHODS

2.1. Plant Materials. Peach fruit (*Prunus persica* [L.] Batsch, cv. Feicheng) was picked from trees growing in Feicheng, Shandong, China, in the summer of 2004 at a preclimacteric, but physiologically mature, stage. They were selected for uniformity of size and ground color, and freedom from defects and mechanical damage. The experiments were carried out at 5 °C. Four treatments of NO (0, 5, 10, and 15 μL/L) were assessed. There were 20 units of peaches in each treatment. One unit comprised 20 peaches. The fruits were placed in sealed 30 L containers, and the containers were put under a vacuum and flushed with nitrogen gas to displace all the oxygen. NO was then injected into the containers with nitrogen gas as a carrier to keep the concentrations of NO at 5, 10, and 15 μL/L. The control peaches were also flushed with nitrogen gas but without NO. After 3 h of exposure to NO, the controls and all of the peaches treated with concentrations of NO were placed in unsealed bags and stored immediately at 5 °C. The following measurements were taken with the newly picked peaches before treatment. These results were expressed as time point 0 day.

2.2. Rot Index of Fruits. In each treatment, five fruits were selected for investigating the number of rotten fruits. All fruits were classified in four ranks by the extent of rot. 0, fruits were not rotten; 1, the rotten surface was less than 1/3; 2, the rotten surface was between 1/3 and 2/3; 3, the rotten surface was more than 2/3. The rot index was expressed by the following equation.

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$$\text{rot index} = \frac{\sum(\text{rank} \times \text{quantity})}{4 \times 5} \times 100\%$$

2.3. Measurement of Firmness. Firmness was determined using a Hunter-Spring penetrometer that was fitted with an 8-mm-long probe with a diameter of 11 mm. Two measurements were taken from the mesocarps without the skin on the opposite face of each fruit; the results were the mean ± SE of the determinations made from 10 replications. The unit of measurement was N cm^{-2}.

2.4. Ion leakage. The effect of NO on membrane permeability was assessed in ion leakage studies using methods modified from Knowles et al. (2001) (*17*). Leakage measurements were done on tissue from all treatments of Feicheng peaches. Tissue discs (2 mm thick, 6 mm diameter) for leakage studies were prepared from a 5-cm-long cross-section from the middle portion of each fruit. The discs were cut from cores (6 mm diameter, 5 cm long) of mesocarp tissue from this section with a Braun food processor. Fifteen discs were randomly selected and placed in 10 mL of distilled deionized H_2O. Conductivity of the surrounding solution was monitored with a PXS-350 conductivity meter (Dapu, Shanghai, China) at 1-min intervals over 30 min. The tissue was then frozen with liquid N_2 and boiled for 10 min, and the total conductivity was recorded. Ion leakage was expressed as a percentage of total ions in the tissue.

2.5. Lipid Extraction. Fruits were peeled and the mesocarp tissue (10 g fresh weight), cut into small pieces, was boiled in *iso*PrOH for 5 min. Lipids were extracted with $CHCl_3$/MeOH (2:1, v/v) containing 50 μg/mL butylhydroxytoluene (BHT) at 4 °C for 24 h. The combined extracts were centrifuged at 30000*g* for 25 min. The supernatants were collected in a separatory funnel and mixed with 5 mL of 0.76% (w/v) NaCl. The mixture was incubated at 4 °C for 1 h. The upper phase was collected and washed three times with $CHCl_3$/MeOH/H_2O (3:48:47, v/v/v) containing 0.76% (w/v) NaCl (*18*). The lower phase of the washed solution was collected and washed again with $CHCl_3$/MeOH/H_2O (86:14:1, v/v/v), and the organic phase was collected as lipid extracts.

2.6. Thin-Layer Chromatography. The lipid extracts from mesocarp were separated by thin-layer chromatography (TLC) on activated silica gel G-60 developed in a solvent mixture of *n*-hexane/Et_2O/HOAc (70:30:1) with 50 μg/mL BHT and fractionated into TPL, monoacylglycerol (MG), diacylglycerol (DG), free fatty acid (FFA), and triacylglycerol (TG) (1). The band containing the TPL was fractionated by two-dimensional TLC, using $CHCl_3$/Me_2CO/MeOH/HOAc/H_2O (10:4:2:2:1) and $CHCl_3$/Me_2CO/MeOH/HOAc/H_2O (6:8:2:2:1) as developing solvent, respectively. Each component was identified by cochromatography with authentic standards under UV light after spraying with 0.1% rhodamine 6G in EtOH. Known aliquots from TPL were used to determine PL through the phosphorus contents (*19*). MG, DG, TG, and FFA were analyzed for their fatty acid content (*20*). 17:0 was used as an internal standard, and the methyl esters, obtained by transmethylation in 2 mol/L MeOH, were analyzed by GC (*1*).

2.7. Fatty Acid Methylation. Fatty acids from TLC lipid fractions were converted into their methyl esters (FAMEs) by dissolving in a mixed solution consisting of 1 mL of 0.4 mol/L KOH methanol solution and 1 mL of benzene/petroleum ether (60−90 °C) (1:1, v/v). Eight milliliters of distilled water was added, and the mixture was oscillated and incubated for 15 min. The organic phase was collected and analyzed.

2.8. GC Analysis. FAMEs were analyzed by gas chromatography (GC-9A, Shimadzu, Japan) equipped with a 2 m × 3 mm GDX-104 column and a flame ionization detector (FID). The column temperature was 190 °C. The detector and injection temperatures were 300 °C. N_2 at 100 mL/min was used as carrier gas. The time needed for one GC run was 17 min.

2.9. Statistics. Data were processed by analysis of variance (ANOVA), comparing treatments at $P = 0.05$ according to Fisher's least significant difference (LSD) test, indicating the LSD value in each case.

3. RESULTS

3.1. Changes of Rot Index of Peach. The effect of NO on the rot index of peaches was shown in **Figure 1**. It was found that the control fruits and the peach fruits treated with 15 μL/L NO started to rot at day 20 during storage. However, the fruits treated with 5 and 10 μL/L NO started to rot at day 30. That was to say, 5 and 10 μL/L NO delayed the rot of peaches for 10 days. Rot indices of fruits treated with 5 and 10 μL/L NO were significantly lower ($P = 0.017$ at day 30) and increased more slowly than the control. The rot index of 5 μL/L NO-treated fruit was higher than that of 10 μL/L NO fruits at day 40 ($P = 0.038$).

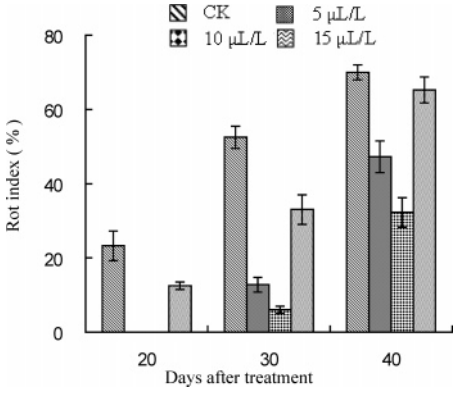

Figure 1. Changes of rot index of peach fruit.

3.2. Changes of Firmness and Ion Leakage. Firmness and ion leakage was determined at timed intervals during storage at 5 °C. All results were tabulated (**Table 1**). **Table 1** shows that firmness decreased and the ion leakage increased during the storage period in all the peaches. Furthermore, the changes were more obvious in the control fruit. The fruit firmness in the control and 15-μL/L NO treatment changed rapidly during the first 20 days ($P = 0.021$ and $P = 0.026$, respectively) in storage at 5 °C. However, the decrease in firmness was slow, and there were significant differences in the 5 and 10 μL/L NO-treated peach fruits during day 40 ($P = 0.0054$) at 5 °C. Changes in membrane integrity that affect increased permeability can readily be demonstrated by measuring leakage of ions from tissue. Tissue from 5 and 10 μL/L NO-treated fruit leaked ions at a slower rate initially, and lost a lower percentage of total ions over a 20-day interval, than that from the control and 15 μL/L NO-treated fruit. After 40 day, tissue from 10 μL/L NO-treated fruit had leaked 22.8% less total ions than tissue from the controlled fruit.

3.3. Changes of Lipid Composition of Peach Fruits. Lipid compositions of the mesocarp tissue from peach fruits fumigated with NO were listed in **Table 2**. Increased contents of phospholipids (PLs) were observed in fruit fumigated with 5 and 10 μL/L NO, while phospholipids decreased in fruit fumigated with 15 μL/L NO. The contents of MG in fruit fumigated with 10 μL/L NO were higher ($P = 0.031$) than the control but lower ($P = 0.044$) than in fruit fumigated with 15 μL/L NO. Opposite effects of NO were found in the contents of DG and TG. There were no significant differences in the contents of FFA.

3.4. Fatty Acid Composition in Lipid Fraction. The fatty acids, palmitic (16:0), palmitoleic (16:1), stearic (18:0), linoleic (18:2), and linolenic (18:3) were found in all lipid fractions. Oleic acid (18:1) was detected only in PL and FFA in fruit treated with NO.

In the PL class (**Figure 2**), 16:1 and 18:1 were the predominant fatty acids. The content of 16:1 in fruits treated with 10 μL/L NO was higher than that of other treatments during storage. In all treatments, 18:1 in PL increased rapidly during storage. The content of 18:1 in fruits treated with 10 μL/L NO

Table 1. Changes of Firmness and Ion Leakage in Feicheng Peach Fruit, as Affected by NO Fumigation[a]

	firmness (N cm^{-2})			ion leakage (%)		
treatments	0	20	40	0	20	40
CK	272.4 ± 1.2a	141.7 ± 2.0c	34.3 ± 1.0c	20.8 ± 0.9a	37.8 ± 1.6a	52.5 ± 2.9a
5 μL/L	272.4 ± 1.2a	213.8 ± 1.7b	108.4 ± 2.0b	20.8 ± 0.9a	26.8 ± 1.3b	36.6 ± 1.7b
10 μL/L	272.4 ± 1.2a	248.6 ± 2.3a	139.9 ± 1.9a	20.8 ± 0.9a	23.1 ± 1.5b	29.7 ± 2.4c
15 μL/L	272.4 ± 1.2a	152.5 ± 1.4c	59.8 ± 0.9c	20.8 ± 0.9a	33.1 ± 2.1a	45.3 ± 2.6a

[a] Data are the average of five fruits. Different letters in each column mean the significance at $P = 0.05$.

Table 2. Lipid Composition of the Mesocarp Tissue from Peach Fruit Fumigated with Nitric Oxide (represented as %)[a]

lipid fraction	phospholipids	monoacyl-glycerols	diacyl-glycerols	triacyl-glycerols	free fatty acid	other lipids
CK	18.80 ± 1.78a	14.61 ± 0.77a	26.33 ± 0.63a	15.78 ± 0.75a	16.85 ± 1.24a	7.62 ± 0.81a
5 μL/L	22.03 ± 2.15b	18.87 ± 0.98b	24.05 ± 1.17a	13.02 ± 0.71a	17.22 ± 1.63a	4.81 ± 0.59b
10 μL/L	26.30 ± 1.37c	20.92 ± 0.86b	21.44 ± 1.03b	8.84 ± 0.65b	18.20 ± 1.39a	4.30 ± 0.74b
15 μL/L	14.22 ± 0.96d	12.82 ± 0.54c	29.05 ± 1.12c	19.33 ± 1.12c	15.81 ± 1.47a	8.77 ± 0.56a

[a] Values represent the mean of percentages ± SE of five replications. Different letters in each column mean the significance at $P = 0.05$.

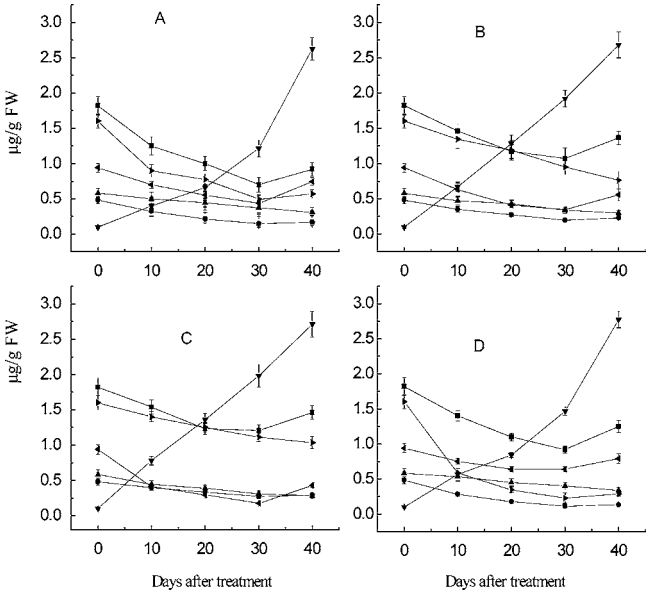

Figure 2. Fatty acid composition of PL in the mesocarp of peach fumigated with NO. (A) CK; (B) 5 μL/L; (C) 10 μL/L; (D) 15 μL/L. FW stands for fresh weight. 16:0 (-●-), 16.1 (-■-), 18.0 (-▲-), 18.1 (-▼-), 18.2 (left facing arrows), 18.3 (right-facing arrows).

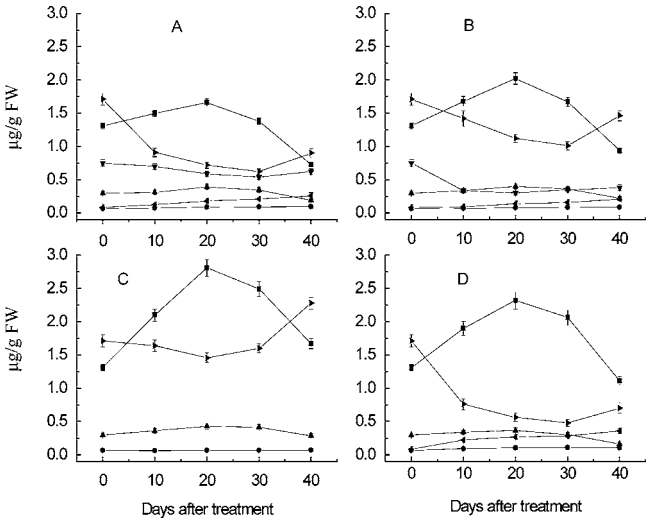

Figure 3. Fatty acid composition of MG in the mesocarp of peach fumigated with NO. (A) CK; (B) 5 μL/L; (C) 10 μL/L; (D) 15 μL/L, FW stands for fresh weight. 16:0 (-●-), 16.1 (-■-), 18.0 (-▲-), 18.1 (-▼-), 18.2 (left facing arrows), 18.3 (right-facing arrows).

was 1.7 times that of the control at day 30. The content of 18:2 in fruit fumigated with 10 μL/L NO was lower ($P = 0.042$) than that of the control at 30 days, while the content of 18:3 was significant higher ($P = 0.043$). There were no significant effects of NO on 16:0, 16:1, and 18:0 in PL.

In the MG (**Figure 3**), 16:1 was the most abundant, followed by 18:3. After day 20, the content of 16:1 decreased, while 18:3 decreased before day 30 and then increased concomitantly. The 16:1 in 10 μL/L NO treatment was 2.3 times higher than the control at day 40. It was notable that no 18:1 in peaches fumigated with 10 and 15 μL/L NO was detected. The content of 18:2 in 10 μL/L NO-fumigated fruit was lower ($P = 0.041$) than that of the control fruit. 5 μL/L NO did not significantly reduce the content of 18:2. At day 30, the content of 18:2 in 15 μL/L NO-fumigated peaches was higher ($P = 0.043$) than that of the control fruit. The content of 18:3 decreased rapidly during 30 days. However, the content of 18:3 was higher ($P = 0.047$) in 10 μL/L NO-fumigated fruit and lower ($P = 0.043$) in 15 μL/L NO-fumigated fruit than that of the control fruit, and no significant difference was observed between 5 μL/L NO and the control.

In the DG (**Figure 4**), 16:1 was abundant, while 18:1 was not detected in any of the treatments. The content of 16:1 in the control fruit decreased sharply during storage, while the treatment with 10 μL/L NO effectively alleviated the decrease, and maintained the high content of 18:1 at day 40. 18:3, which declined during storage in 10 μL/L NO-fumigated fruit, was 1.66 to 2.42 times higher than that of the control. The 18:3 in 15 μL/L NO-fumigated fruit was only 58−72% of the control fruit during ripening.

In the FFA (**Figure 5**), the 16:1 decreased before day 30 and increased at day 40. The content of 16:1 in 10 μL/L NO was higher than that of the control and other treatments and changed gently during storage. The content of 18:1 in 10 μL/L NO-fumigated fruit decreased over time during storage and was lower ($P = 0.044$) than that of the control at 30 days. The 18:3 acid in fruit fumigated with 10 μL/L NO was higher ($P = 0.011$) than that of the control during storage. A similar change was found in fruit fumigated with 5 μL/L NO ($P = 0.039$). The 18:3 in 15 μL/L NO-fumigated fruit was lower ($P = 0.045$) than that of the control at day 30 after treatment. The contents

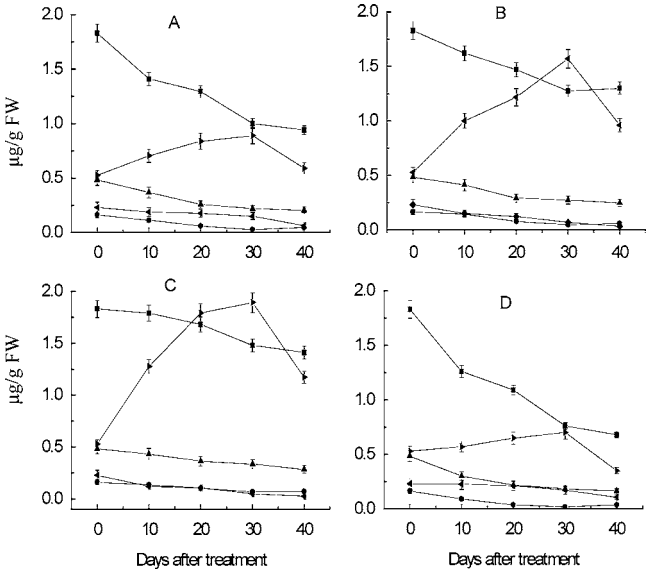

Figure 4. Fatty acid composition of DG in the mesocarp of peach fumigated with NO. (A) CK; (B) 5 μL/L; (C) 10 μL/L; (D) 15 μL/L. FW stands for fresh weight. 16:0 (-●-), 16.1 (-■-), 18.0 (-▲-), 18.1 (-▼-), 18.2 (left facing arrows), 18.3 (right-facing arrows).

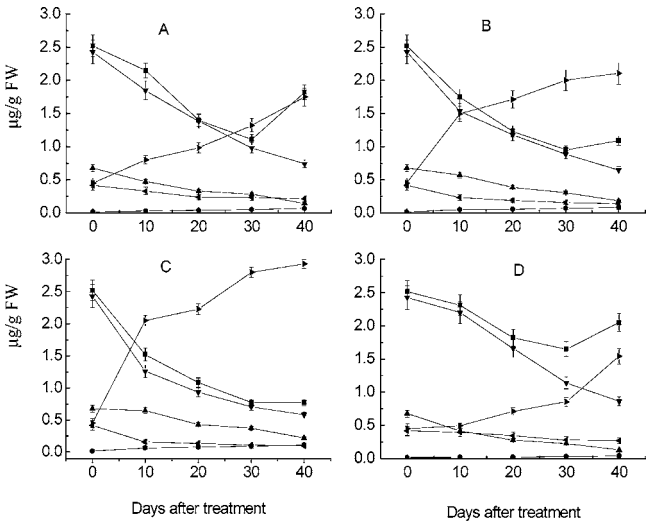

Figure 5. Fatty acid composition of FFA in the mesocarp of peach fumigated with NO. (A) CK; (B) 5 μL/L; (C) 10 μL/L; (D) 15 μL/L. FW stands for fresh weight. 16:0 (-●-), 16.1 (-■-), 18.0 (-▲-), 18.1 (-▼-), 18.2 (left facing arrows), 18.3 (right-facing arrows).

of 18:2 in 5 and 10 μL/L NO-fumigated fruit were 1.20−1.87 and 1.63−2.57 times higher than that of the control, respectively. However, one in 15 μL/L NO-fumigated fruit was not significantly different than the control fruit.

In the TG (**Figure 6**), no significant differences were found in the contents of 16:0 and 18:0 between each treatment. The 18:1 acid was also not detected in any of the treatments. The content of 16:1 in 10μL/L NO treatment was lower than that of the control after day 20. The contents of 18:3 in 5 and 10 μL/L NO-fumigated fruit were 1.2−1.4 fold and 1.5−1.6 fold of the control during storage, respectively. In contrast, the 18:3 in 15 μL/L NO-fumigated fruit was 73−80% of the control.

4. DISCUSSION

The results described above showed that NO could prevent the gradual decline of the peach fruit toward softening and rot. The impact of 10 μL/L NO was obvious. This effect was similar

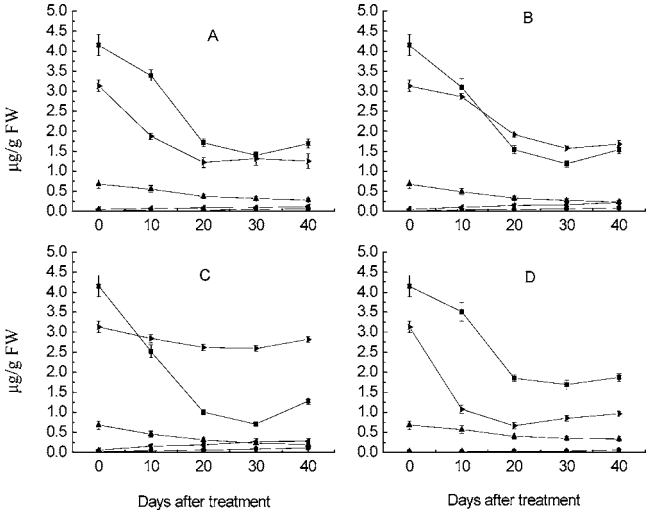

Figure 6. Fatty acid composition of TG in the mesocarp of peach fumigated with NO. (A) CK; (B) 5 μL/L; (C) 10 μL/L; (D) 15 μL/L. FW stands for fresh weight. 16:0 (-●-), 16.1 (-■-), 18.0 (-▲-), 18.1 (-▼-), 18.2 (left facing arrows), 18.3 (right-facing arrows).

to Leshem's observation that NO could delay the ripening and senescence of fruit (*21*). A common feature accompanying senescence was the increase of membrane permeability, expressed as increasing leakage of ions. In apples, this leakage correlated with increased membrane viscosity and decreased degree of fatty acid unsaturation (*22*). In the present experiment, the ion leakage in 10 μL/L NO fumigated peach fruit was slow, indicating that 10 μL/L NO could maintain the membrane integrity in peach fruits, as was consistent with the changes of rot index.

Membrane fatty acid composition and phospholipids composition could be modified. The modification was extensive enough to alter membrane fluidity and affect a number of cellular functions (*23*). The results in this experiment showed that fumigation with NO could have profound influences on the composition of lipids in peaches. The effects of treatment with 10 μL/L NO were more visible than those of treatment with 5 μL/L NO, but treatment with 15 μL/L NO induced some opposite effects. Results also indicated that the effects of NO on lipid peroxidation in peach fruits depended on time and the concentrations of NO. NO changed the lipid composition of peach fruits. In the compositions of PL, MG, DG, TG, and FFA, the unsaturated C18 fatty acids (18:1, 18:2 and 18:3) were prone to be affected by NO. The content of 18:3 decreased in 5 and 10 μL/L NO-fumigated fruits but increased in fruit fumigated with 15 μL/L NO. As 18:3 plays important roles in membrane leakage and lipid composition (*24*), these results also confirmed the idea that lipids might play an important role in ripening of peach fruit (*25*).

Lipid peroxidation is a major mechanism for membrane deterioration in plant tissue. Nitric oxide can inhibit this process by acting as a potent inhibitor of the lipid peroxidation chain reaction via scavenging propagatory lipid peroxyl radicals or by inhibiting many potential initiators of lipid peroxidation, such as enzymes (*26*). Free radicals play important roles in the ripening and senescencing fruits. The lipid peroxyl radical ($LOO^{\bullet}$) is the central species of the lipid peroxidation chain reaction. The reaction between $LOO^{\bullet}$ and NO led to immediate termination of oxidation (*27*). The hydroxyl radical ($^{\bullet}OH$) is also a highly oxidizing free radical and will initiate lipid peroxidation by hydrogen abstraction. Hydroxyl radicals are thought to be generated in vivo from the combination of iron

ions (or copper ions), hydrogen peroxide, and a reducing agent (e.g., ascorbate) by the Fenton reaction (*26*). This mixture is an aggressive oxidant and will oxidize many biological molecules, including unsaturated fatty acids. Nitric oxide has been shown to inhibit the Fenton reaction by binding to ferrous iron and thus preventing the formation of hydroxyl radical (*28*). It has also been demonstrated that linolenic acid peroxidized by $O_2^{\bullet -}$, H_2O_2, and $^{\bullet}OH$ showed dose-dependent regulation of lipid peroxidation by NO (*29*). LOX participates in the onset of the process (*30*). It has been shown that NO can inhibit LOX activity in peach fruits (*31*), by reacting with the nonheme iron at the active site (*32*) or hydroxyl radicals (*33*). By inhibiting the activity of LOX, 10 μL/L NO can preserve the unsaturated fatty acids (such as 18:1, 18:2, and 18:3) against peroxidation. In addition, the 18:2 can also be converted to 18:3 by the catalysis of fatty acid desaturase. These would explain the decrease of 18:2 and the increase of 18:1 and 18:3 in PL of peaches treated with 10 μL/L NO. It has been confirmed that ion leakage could be correlated to losses in 18:3 (*6*). We thought it might be that the treatments with 5 and 10 μL/L NO restrained the lipid peroxidation in peach fruits and consequently maintained the high content of 18:3. Thereby, the ion leakage was prevented and the integrity of the membrane in peach fruits was maintained by 5 and 10 μL/L NO.

MG and DG are the major lipids associated with plastid structure. These variations of compositions of MG and DG can in part be the result of changes that occur at the plastid membrane level during the ripening of peach fruits. The marked decreases of 18:3 in MG and DG might be due to the degradation of the lipid composition, as a result of plastid transformations in the mesocarp of peach fruits. In this experiment, the decrease of 18:3 from 30 to 50 days was restrained by treatments with 5 and 10 μL/L NO, indicating that these concentrations of NO could protect the plastid structure against deterioration.

Previous researchers (*34*) also proposed that plant cells responded to different stress by producing various uncommon PLs, which play key roles in cell signaling. Some specific lipid-binding domains, which act as docking sites, have been found in *Arabidopsis* proteins. When such proteins are recruited to the membrane locations where the PLs are synthesized, the PLs activate them directly by inducing a conformational change, or by juxtaposing them with an activator protein (*34*). In this experiment, the 18:1 was detected in PL and FFA but not in MG, DG, and TG. These results may be due to the different compositions and structures of MG, DG, and TG in the mesocarp of peach fruits. Further work, however, needs to be done to confirm them.

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