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Original Research Article

Postharvest suppression of gray mold (*Botrytis cinerea*) on peach through application of *n*-propyl dihydrojasmonate

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ABSTRACT

Control of gray mold disease (*Botrytis cinerea*) and change of endogenous plant hormones, ethylene, jasmonic acid (JA), and abscisic acid (ABA) in peach (*Prunus persica L. Batsch 'Akatsuki'*) treated with *n*-Propyl dihydrojasmonate (PDJ) were investigated. Peaches were compared between non-PDJ dipping (control) and 400 μM-PDJ dipping before inoculation with *B. cinerea* conidial suspension stored at 25°C for 6 days. The results showed that PDJ application significantly decreased ($P \leq 0.05$) disease symptoms and lesion diameter, meanwhile, this application induced the accumulation of ethylene production, JA, and ABA concentration. The upstream expression level of *PpACS1* (1-aminocyclo-propane-1-carboxylic acid synthase) and *PpAOS* (allene oxide synthase) genes in PDJ application were significantly higher in the peaches inoculated with *B. cinerea* than the control. In contrast, PDJ application decreased ester and lactone compounds which are the major volatile compound of peach. However, alcohol and aldehyde compounds were increased by the PDJ application. These results suggested that PDJ application delayed the infection of *B. cinerea* through accumulation of ethylene, endogenous JA, and ABA in peach.

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INTRODUCTION

Botrytis cinerea is one of the most important postharvest diseases resulting in economic loss on peach. Infection by *B. cinerea*, so-called gray mold disease, is initiated at flower stage, latent during fruit stage, and eventually occurs during retail stage (Elad et al., 2004). Fungicides are widely used to control pathogens at pre and post-harvest stage, especially benzimidazoles and dicarboximides. However, pathogens have developed its resistance to these. In recent years, consumers are concerned about environmental pollution and human health risks. Alternative methods for control of postharvest diseases such as biological and cultural method. Jasmonic acid (JA) derivative and methyl jasmonate (MeJA) has been reported to be effective to enhance disease resistance in peach infected with *Penicillium expansum*, *Botrytis cinerea*, and *Rhizopus stolonifer* (Jin et.al, 2009). In Japan, *n*-Propyl dihydrojasmonate (PDJ) is JA synthetic analog which is convenient to use. PDJ application effectively delays postharvest diseases infection, for example; *Collectrotrichum gloeosporioide*s in Japanese apricot (*Prunus mume* Sieb.) and *Glomerella cingulate* in grape berry (*Vitis labrusca* × *Vitis vinifera*) respectively (Nimitkeatkai et al., 2011 and Wang et al., 2015).

JA plays an important role during plant development, ripening, pathogenesis-related (PR) protiens, and response to abiotic and biotic stress (Concha et al., 2013; Wasternack, 2007; Wei et al., 2017). Exogenous JA has been reported to enhance disease resistance, that is, activation of defense enzymes, increase phenol compound and antioxidant capacity, and accumulation of hydrogen peroxide, resulting in delaying postharvest diseases in peach, strawberry, and grape (Jiang et al., 2015; Jin et al., 2009; Saavedra et al., 2016). PDJ application also effectively delayed ripening through decreased ethylene production and expression level of *PpACO1* in peach (Ziosi et al., 2008). JA and ethylene signal transduction pathways act synergistically in a plant's immunity through accumulation of *ethylene response factor (ERF)* gene (Pieterse et al., 2009). Moreover, peach aroma volatile compounds are lactones and esters which provide fruity notes, and C6 aldehydes and alcohols contribute green sensory notes in the ripening fruit (Zhang et al. 2010).

Antagonistic interaction between the abiotic stress hormone, abscisic acid (ABA), and JA have been observed in *Arabidopsis thaliana* inoculations with *Fusarium oxysporum* (Jonathan et al., 2004). Another study found that high concentration of ABA inhibits ethylene production in tomato and strawberry (LeNoble et al., 2004). For volatile compounds, on the other hand, 2-Hexanal, C6 aldehydes, had been reported to inhibit postharvest pathogens such as *Monilinia laxa* and *B. cinerea* (Fiorella et al., 2006; Tsao and Zhou 2000). In contrast, ester and alcohol volatile compounds developed as a result of *B. cinerea* infection from the latent phase to growth phase after wounding in strawberry (Neri et al., 2015).

The relationship of plant hormones, JA, ethylene, and ABA, and volatile compounds during infection with postharvest pathogens in peach is unclear. The aim of this study was, first, to evaluate the effect of PDJ application on controlling *B. cinerea* in peach, then to investigate relationship between PDJ application, on endogenous JA, ethylene production, ABA, and change of volatile compounds associated with defense pathogen responses.

MATERIALS AND METHODS

Peach fruit and treatment

Peach trees (*Prunus persica* L. Batsch 'Akatsuki') grown from the experimental orchard at Chiba University, Chiba prefecture, Japan (35°N latitude, 140°E longitude, and elevation of 37°m) were used for the experiment. Fruits were harvested at 96 days after full bloom (DAFB), before climacteric stage, and then transferred to laboratory. Peach fruit were uniformly selected for size and maturity, without wounds or injury, then rinsed with tap water, dipped into 150 μg•mL^{-1} of sodium hypochlorite for 3 min and rinsed with distilled water. Fruit were randomly divided into three group (90 fruit per group). Two groups were dipped into 0.1% (v/v) Approach BI® (50% polyoxyethylene hexitan fatty acid ester; Kao, Osaka, Japan) for 5 min (PDJ-), and one group was dipped into 400 μM PDJ solution containing 0.1% (v/v) Approach BI® (PDJ+). After that, fruit were air-dried and, then stored at 25°C for 24 hours.

Pathogen inoculation of peach

Botrytis cinerea (MAFF, 712208) was used and cultured on potato dextrose agar (PDA) at 25°C for 10 days. Spore suspension was prepared by removing the conidia from fungal culture on PDA and suspending in sterile distilled water. The spore suspension was adjusted to 1 × 10^6 conidia•mL-1. For pathogen inoculation, all groups were wounded with a sterilized nail (3 mm depth and 5 mm in diameter). Both groups were inoculated with a 50 μL of the spore suspension after 24 h. PDJ group and inoculation group without PDJ treatment were PDJ$^+$ Ino$^+$ and PDJ- Ino+, respectively. The untreated control group was inoculated with 50 μL of sterile distilled water with 0.5% agar.

After inoculation, fruit were placed into a container covered with polyethylene bag and then stored at 25°C with 95% relative humidity (RH) for 6 days. Peel was collected from 15 fruit (3 fruit per replication) at 0, 1, 2, 3, and 6 days after treatment. Peach peel was frozen immediately in liquid nitrogen and stored at -80°C until analysis. The frozen tissues were analyzed for 1-aminocyclo-propane-1-carboxylic acid (ACC) concentration, ethylene-related gene [*PpACS1*, *PpACO1*, *PpETR1*, and *PpCTR1* (*Constitutive triple response 1*)], endogenous abscisic acids (ABA), and JA concentration and expression levels of *PpAOS* gene. Ethylene production rate was determined from whole fruit while lesion diameter determined daily.

The lesion diameter and disease incidence in peach fruit

Fifteen fruit (5 fruit per replication) were measured daily for lesion diameter and disease incidence after inoculation. The lesion diameter of mycelium growth was determined from two equidistant wound areas in each fruit. The percentage of disease incidence was calculated as shown in the equation as show below.

$$\text{Disease incidence} = \frac{\text{number of fruit diseased}}{\text{total number of fruit}} \times 100$$

Ethylene production and ACC concentration

Ethylene production was analyzed as previously described by Kondo et al. (1991). One peach fruit was incubated in 1.67 L closed plastic box for 1 h at 25°C and then 1 mL of headspace gas was

colleded. Ethylene production was analyzed from 1 mL of headspace gas using gas chromatography (model GC-2014; Shimadzu, Kyoto, Japan) with a flame-ionisation detector (FID). The ethylene production rate was expressed as microliter of ethylene per kilogram per hour.

For ACC concentration, 2 grams of frozen peel were homogenized with 0.1 M of hydrochloric acid then centrifuged and the supernatant was used for analysis. The supernatant was mixed with 0.1 M mercuric chloride and 5% sodium hypochlorite with saturated sodium hydroxide in glass tube with lid and 2 mL headspace gas was injected into a gas chromatography (model GC-2014; Shimadzu, Kyoto, Japan) with FID and then the peak area was converted to ACC concentration.

Jasmonic acid (JA) concentration

Endogenous JA concentration determination was performed as previously described by Kondo et al. (2005) with GC-MS-SIM (QP 5000; Shimadzu, Kyoto, Japan; 25 m×0.25 mm I.D. column). The frozen peels (1 g) were homogenized with ibuprofen as an internal standard in 10 mL of saturated NaCl and 20 mL of diethyl ether containing 0.005% butylated hydroxytoluene (BHT) as an antioxidant. The ether phase was removed after centrifugation at 10,000 rpm and then extracted 3 times with diethyl ether containing 0.005% BHT. The pooled ether extract was dried under warm air, after that the residue was dissolved in 200 µL of chloroform/isopropylethylamine, 1:1 (v/v), and derivatized at 50°C for 60 min with pentafluorobenzyl brominde. The ions were measured as m/z 390, 264, and 209. The concentration of JA was determined from the ratio of peak areas for m/z 209 (jasmonic acid)/264 (ibuprofen).

Abscisic acid (ABA) concentration

One gram of frozen peach peel (three replications) was homogenized in 20 mL cold 80% (v/v) methanol including 0.5 g polyvinylpyrrolidone with 0.2 µg ABA-d6 as an internal standard. Endogenous ABA was extracted and analyzed as previously described by Setha and Kondo (2009). The methyl ester of ABA was analyzed by gas chromatography-mass spectrometry-selected ion monitoring (QP 5000; Shimadzu, Kyoto, Japan; 25 m×0.25 mm I.D. column). The ions were measured as m/z 190, 194, 260, and 264. The concentrations of ABA were determined from the ratio of peak areas for m/z 190 (d0)/194 (d6).

Total RNA extraction, cDNA synthesis and quantitative RT-PCR (qRT-PCR) analysis

The frozen tissue (0.3 g fresh weight; three replications) was extracted using cetyltrimethylammonium bromide buffer and column-based extraction method (Henderson and Hammond, 2013). cDNA was synthesized from RNA using ReverTra Ace®qPCR RT Master Mix following the manufactures instructions (Code No. FSQ-201; Toyobo co., LTD., Osaka,Japan). The specific primer of each gene was used for *q*RT-PCR analysis (Table 1). Transcription level was estimated by *q*RT-PCR (model: Steo One Plus, Life Technology, Tokyo,Japan) with a KAPA SYBR FAST Master Mix (Kapa Biosystem, Boston, MA, USA) according to instruction manual. The expression level of each gene was calculated as the coefficient of variation, and normalized to the transcript level of the average of *UBIQUITIN* (*UBQ*), and *Actin* gene.

Table 1. Specific primer for real-time PCR.

Gene	Forward/reverse primer (5'- 3')	References/ Accession no.
PpACS1	(F) ACCGAGACTTGGGATGGAGA	Begheldo et al., 2008
	(R) TGATCAAGCCCTTCACGTTG	
PpACO1	(F) TGAGCTGATCAAGGGTCTCC	NCBI: AF319166
	(R) ATCCATTGGCCATCTTTGAG	
PpETR1	(F) ATGATAACGGGTCAGTGACT	Cin et al., 2006
	(R) AAATAACGTGCAAGAACTCATC	
PpCTR1	(F) GCAAGACTTTCATGCCGAAC	Cin et al., 2006
	(R) TATGGACAAGTTTGGGGGCT	
PpUBQ	(F) AAGGCTAAGATCCAAGACAAAGAG	NCBI: KJ598788.1
	(R) CCACGAAGACGAAGCACTAAG	
PpActin	(F) GATTCCGGTGCCCAGAAGT	NCBI: AB046952.1
	(R) CCAGCAGCTTCCATTCCAA	

Volatile compounds analysis

Volatile compounds were extracted and identified by gas chromatography-mass spectrometry (GC–MS) and the quantification of selected volatile compounds according to Wang et al. (2015). The frozen peel (0.5 g) was put into 4-mL vial with lid and incubated with 10 µL cyclohexanol as an internal standard at 40°C for 30 min. The headspace gas were aborted and extracted using 50/30 µm Divinylbenzene/Carboxen/Polydimethylsiloxane solid-phase micro-extraction fiber (Supelco, Bellefonte, PA) and then reabsorbed at the injection port of GC-FID (GC-4000 plus, GL Sciences, Kyoto, Japan, DB-Wax; 60 m × 0.25 mm I.D. capillary column; Agilent, Santa Clara, CA, USA), which was set at 250°C. The concentrations of individual compound were determined using the peak of the internal standard as a reference value and calculated based on standard curves of pure compounds.

Statistical analysis

The statistical analysis was performed with SAS analysis (version 8.2, SAS institute, Cary, NC, USA). The data were analyzed by one-way analysis of variance (ANOVA). The treatment effect and the mean were separated by Tukey-Kramer test at $P \leq 0.05$. The data were presented the mean values of the three replications ± standard error (SE).

RESULTS AND DISCUSSION

Disease incidence and lesion diameter of *B. cinerea* on peach

PDJ^+ Ino^+ treatment prevented the infection of gray mold on peach for 5 days and lesion diameter was observed at 6 days after treatment (DAT). However, the disease incidence and lesion diameter of PDJ- Ino+ treatment was observed at 4 DAT and significantly higher than PDJ^+ Ino^+ treatment. Disease incidence and lesion diameter was not observed in the untreated group. On the other hand, the untreated group showed no infection of gray mold (Figure 1A, B). The results showed PDJ application at 400 µM concentration retarded disease incidence and suppressed *B. cinerea* infection as well as lesion diameter on peaches (Figure 1). In agreement with our results, JA derivative, methyl jasmonate (MeJA), and PDJ have been reported to suppress postharvest diseases of fruit such as strawberry (Gabriela et al., 2017), grape (Jiang et al., 2015; Wang et al., 2015), and Japanese apricot (Nimitkeatkai et al., 2011), which practicable enhanced activity of defense resistance enzyme and related genes.

Ethylene production, ACC content, Jasmonic acid concentration, and expression level of ethylene related genes and *PpAOS* gene on peaches inoculated with *B. cinerea*

Ethylene production rapidly increased in PDJ$^+$ Ino$^+$ treatment at 3 DAT. ACC concentration and expression of *PpACO1* gene were induced in PDJ$^+$ Ino$^+$ treatment and significantly higher in later stages compared with other treatment, especially *PpACO1* gene (Figure 2B, D). PDJ- Ino+ treatment showed a marked increase in ACC concentration at 6 DAT while PDJ$^+$ Ino$^+$ treatment increased ACC concentration and expression of *PpACO1* gene at 2 and 1 DAT, respectively. PDJ$^+$ Ino$^+$ treatment up-regulated expression of *PpACS1* and *PpCTR1* gene, thus, ethylene production was increased and significantly higher than PDJ- Ino+ treatment and the untreated group (Figure 2A, C, F). The highest expression of *PpETR1* gene was observed in untreated control group between 3 to 6 DAT (Figure 2E).

The increment of JA concentration and expression level of *PpAOS* gene were shown in PDJ$^+$ Ino$^+$ treatment and significantly higher than PDJ- Ino+ treatment and untreated control group, respectively (Figure 3A, B). PDJ$^+$ Ino$^+$ treatment increased JA concentration and expression level of *PpAOS* gene especially at 3 and 6 DAT and related with the increment of ethylene production. Also, JA concentration affected to decrease lesion diameter of *B. cinerea* on PDJ$^+$ Ino$^+$ treatment. On the other hand, PDJ- Ino+ treatment rapidly increased JA concentration at 2 DAT, but it was not significantly different with PDJ$^+$ Ino$^+$ treatment.

Ethylene and JA play an important role in plant defense response to pathogen as induced systemic resistance (ISR). The ISR can promote mitogen-activated protein kinase (MAPK), the octadecanoic pathway (oxylipins biosynthesis), the phenylpropanoid pathway, and cell wall metabolism (Gianfranco et al., 2016; Lloyd et al., 2011; Shoresh et al., 2010). Our results showed that PDJ$^+$ Ino$^+$ treatment significantly increased ethylene production and endogenous JA at 3 and 2 DAT, respectively. Previous reports have revealed that PDJ application involved in enhancing ethylene production on pears (Kondo et al., 2007), grapes (Wang et al., 2015), and Japanese apricots (Nimitkeatkai et al., 2011). In addition, the current study indicated that PDJ$^+$ Ino$^+$ treatment induced expression of *PpACS1* and *PpCTR1* gene in peach. Tong et al. (2017) suggested JA application up-regulated both *ACS1* and *ACO1* gene pass through induction of transcription factor of the JA signaling pathway, *MYY2* gene, in apple. However, PDJ$^+$ Ino$^+$ treatment showed significantly lower ACC content and down-regulated expression of *PpACO1* gene than PDJ$^+$ Ino$^+$ treatment. This result is supported by previous study which showed that down-regulated expression of *PpACO1* by JA in peaches at harvest (Ziosi et al., 2008). On the other hand, PDJ application up-regulated expression of *PpAOS* gene resulting in increment of endogenous JA. *AOS* gene has been reported to regulate JA biosynthesis and increased at ripening stage in apple, peach, and tomato (Fan et al., 1998; Kondo et al., 2000; Torrigiani et al., 2012; Ziosi et al., 2008). Ethylene positively regulates the induction of *AOS* gene (O'Donnell et al., 1996), while JA induces expression of *ACO*, resulting in enhanced ethylene production (Hudgins and Franceschi, 2004). The defense related genes, such as *plant defensin* (*PDF1.2*) and *β chitinase* (*β-CHI*) gene, were induced by both JA and ethylene hormone against necrotrophs pathogen (Po-Wen et al., 2013; Zhu et al., 2011). Moreover, Lorenzo et al. (2003) reported *Ethylene response factor1* (*ERF1)* as a functional transcription factor which is a key gene regulating ethylene or JA or both, and responding to pathogen resistance through activate expression of PR gene. Our results suggest that PDJ application may inhibit *B. cinerea* growth through the induction of ISR by JA/ethylene signaling defense response pathways.

Abscisic acid concentration on peaches inoculated with *B. cinerea*

ABA had the highest concentration at harvest time of peach (0 DAT). PDJ$^+$ Ino$^+$ treatment slightly decreased ABA concentration when compare with PDJ- Ino+ treatment and untreated control group which was observed a profound decline at 1 DAT (Figure 4). ABA concentration increased in PDJ$^+$ Ino$^+$ treatment and was significantly higher than PDJ- Ino+ treatment at 3 DAT. However, ABA concentration was sharply decreased at 16 DAT in PDJ$^+$ Ino$^+$ and PDJ$^+$ Ino$^+$ group. ABA plays an important role in response to biotic and abiotic stress in fruit. Nevetheless, the increment of ABA are depended on time (Brigitte and Felix, 2005). ABA has been reported to regulate plant resistance to pathogens (AbuQamar et al., 2017). Our result found that *B. cinerea* infection in peach increased endogenous ABA at 3 DAT. The result was supported by previous research which reported that ABA concentrations were induced in sugar beet leaves during fungal infection, *Cercospora beticola* (Schmitd et al., 2008). Moreover, Kazan and Manners (2013) reported MYC gene, regulator of the JA signaling pathway, positively regulated ABA signaling pathway.

Volatile compound on peaches inoculated with *B. cinerea*

Twenty seven volatile compounds, i.e., 8 alcohols, 5 aldehydes, 8 esters, 2 ketones, and 4 lactones, were identified from peach inoculated with *B. cinerea* using GC-MS (data not show). Alcohol, aldehyde, and ester concentrations accumulated significantly in PDJ$^+$ Ino$^+$ treatment than in PDJ- Ino+ treatment at 2 and 3 DAT (Figure 5A, B, C). In contrast, lactone concentration was induced higher in PDJ- Ino+ than PDJ- Ino+ treatment at 3 and 6 DAT (Figure 5D). PDJ$^+$ Ino$^+$ treatment showed lower concentration of ester compound and significantly lower compared to other treatments.

In our study, alcohol and aldehyde were detected to be the most abundant compounds in peach treated with PDJ application, as they were in previous research (Nimitkeatkai et al., 2011; Wang et al., 2015). Further still, they showed that alcohol and aldehyde compounds mainly increased on Japanese apricot and grape treated with PDJ. Hexanal and (E)-2-hexanal were the main aldehyde compounds in this research on peach. C6 aldehyde (hexanal, (E)-2-hexenal, and (Z)-3-hexenal) has been shown to induce plant defense resistance to inhibit postharvest pathogen such as *C. gloeosporioides*, *B. cinerea*, and *Alternaria alternate* (Anusha et al., 2016; Gomi et al., 2003; Kishimoto et al., 2006). The lactone compounds, i.e., γ-decalactone and δ-decalactone, were enhanced in peach infected with *B. cinerea* and had been found to be associated with ripening aroma compounds (Li et al. 2015; Sánchez et al. 2013). The result showed the relationship between increment of volatile compounds and enhancement of ethylene production and JA concentration while peach was applied with PDJ application. The unsaturated fatty acids, 9- or 13-hydroperoxid, were synthesized by lipoxygenase (LOX) pathway which is a main substrate for oxylipin pathway (Hatanaka, 1993; Wasternack and Kombrink, 2010). The oxylipin pathway is responsible for biosynthesis of methyl jasmonate and volatile compounds, which is a different pathway including AOS pathway and hydroperoxidase lyase (HPL) pathway, respectively (Ismanizan et al., 2011). These results are in agreement with previous research finding that methyl jasmonate induced ethylene production although ethylene accelerated accumulation of volatile compounds (Wei et al., 2017).

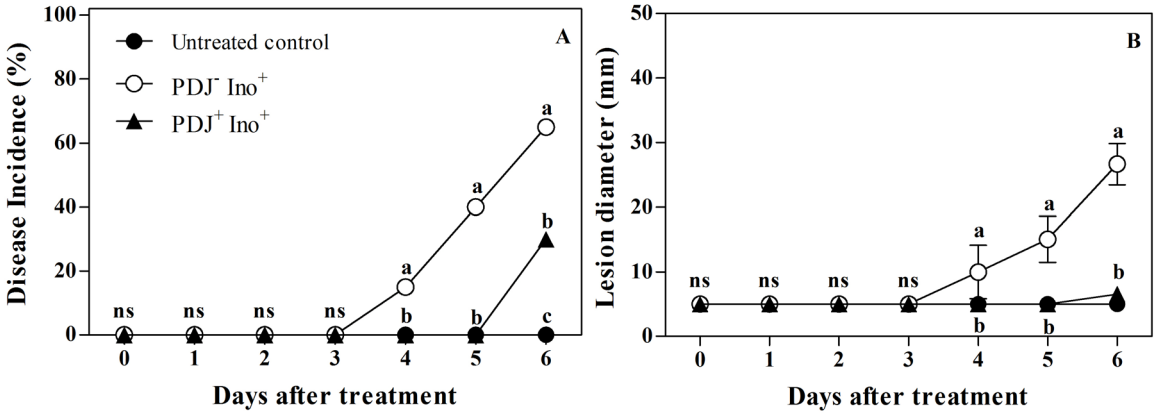

Figure 1. Disease incident (A) and lesion diameter (B) on peaches treated with PDJ or without (untreated) and inoculated with *Botrytis cinerea* during storage at 25°C for 6 day.

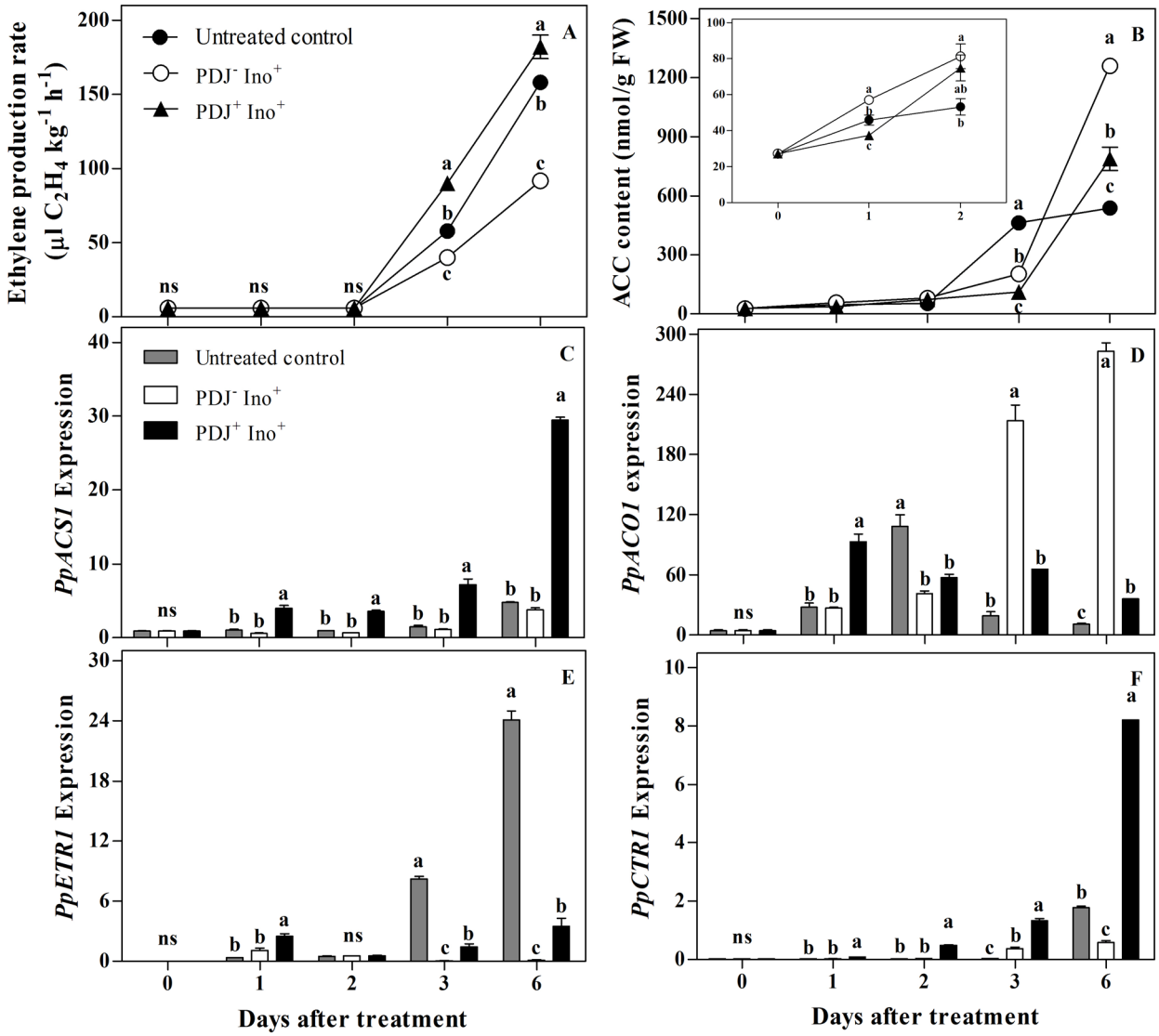

Figure 2. Changes of ethylene production rate (A), ACC content (B), expression of *PpACS1* (C), *PpACO1* (D), *PpETR1* (E), and *PpCTR1* (F) gene on peaches treated with PDJ or without (untreated) and inoculated with *Botrytis cinerea* during storage at 25°C for 6 days.

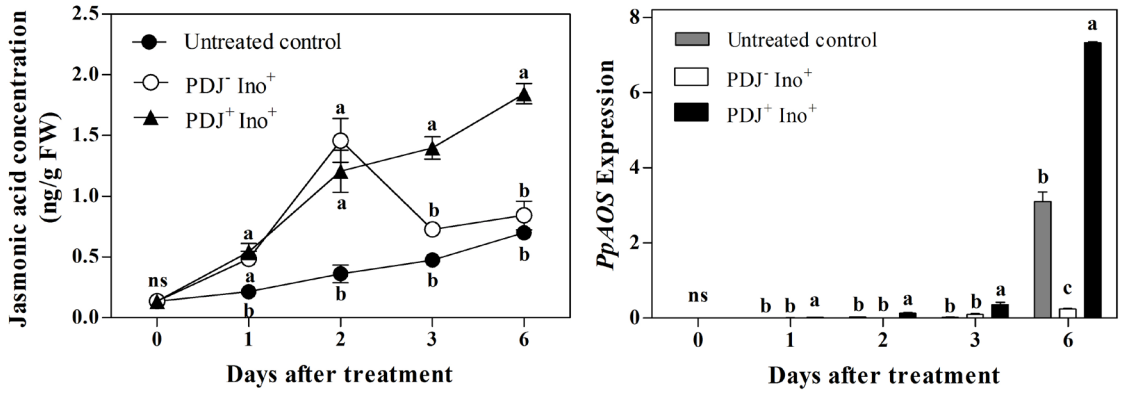

Figure 3. Changes of jasmonic acid concentration (A) and expression of *PpAOS* gene (B) on peaches treated with PDJ or without (untreated) and inoculated with *Botrytis cinerea* during storage at 25°C for 6 days.

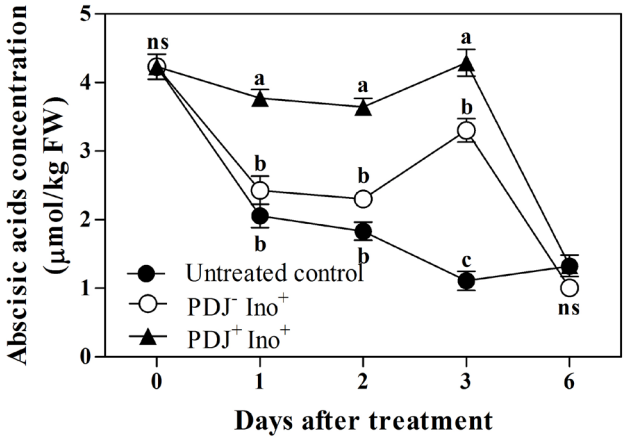

Figure 4. Change of abscisic acid concentrations on peaches treated with PDJ or without (untreated) and inoculated with *Botrytis cinerea* during storage at 25°C for 6 days.

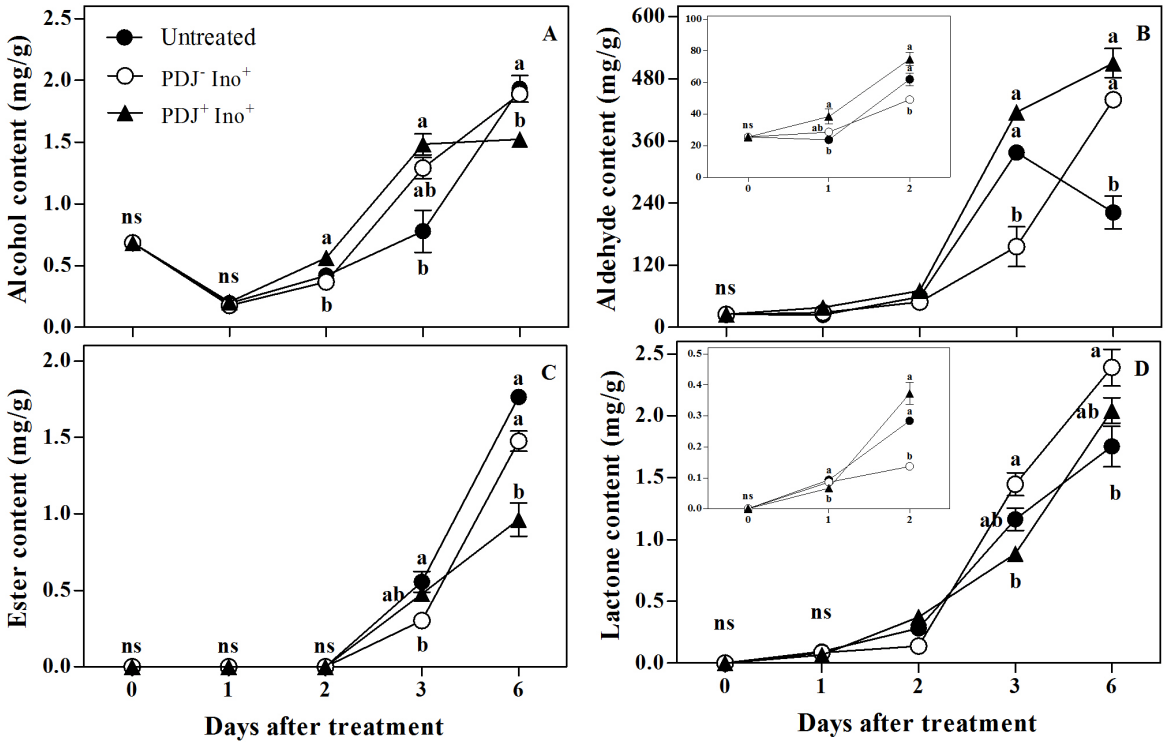

Figure 5. Changes of volatile compounds, alcohol (A), aldehyde (B), ester (C), and lactone (C) concentration, on peach fruits treated with PDJ or without (control) and inoculated with *Botrytis cinerea* during storage at 25°C for 6 days.

CONCLUSIONS

The increment of endogenous JA and ethylene production as well as the expression levels of *PpACS1, PpCTR1*, and *PpAOS* gene inhibit the growth of *B. cinerea* in peach. The defense resistance system in peach against *B. cinerea* depends on the synergistic relationship between JA and ethylene. In addition, alcohol and aldehyde enhance by PDJ application also inhibit fungal development.

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