ORIGINAL PAPER

Methyl Jasmonate Primed Defense Responses Against *Penicillium expansum* in Sweet Cherry Fruit

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Abstract The effect of methyl jasmonate (MeJA) treatment on controlling blue mold decay caused by *Penicillium expansum* in sweet cherry fruit and the possible mechanisms were investigated. The results indicated that fruit treated with MeJA had significantly lower disease incidence and smaller lesion diameter than the control fruit did. The in vitro experiment showed that MeJA transiently inhibited spore germination and germ tube elongation of *P. expansum*. It is clear that MeJA triggers a priming mechanism in sweet cherry fruit, since only in fruit that had been pretreated with MeJA and then challenged with *P. expansum* was an enhanced capacity to augment defense responses observed. These augmented responses included enhanced activities of chitinase (CHI) and β-1,3-glucanase (GLU), and increased gene expression levels of catalase (CAT), calmodulin (CaM), GLU, phenylalanine ammonia-lyase (PAL), nonexpressor of pathogenesis-related genes 1 (NPR1-like), and thaumatin-like (THAU). Moreover, MeJA inhibited the increase of activities of polygalacturonase (PG) and pectinmethylesterase (PME). These results suggest that the efficacy of MeJA on controlling blue mold decay in sweet cherry fruit may be related to the transient direct inhibitory effect against the pathogens, suppressed activities of PG and PME, and the priming of defense responses.

Keywords Sweet cherry · Methyl jasmonate · Priming · *Penicillium expansum* · Defense-related gene

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Introduction

Sweet cherries (*Prunus avium* L.) are highly perishable and susceptible to mechanical injury, physiological deterioration, and fungal decay. Blue mold decay caused by *P. expansum* is one of the most important postharvest diseases of sweet cherries (Ceponis et al. 1987). Traditionally, the control of postharvest mold decay relies mainly on the use of synthetic fungicides. However, the increasing resistance of fungal pathogens and growing concern of consumers over chemical residues make it crucial to study alternative means for control of postharvest diseases (Janisiewicz and Korsten 2002).

Induction of disease resistance in horticultural crops is an important strategy to reduce diseases' incidence. This is promising because it uses the defense mechanisms of the plant itself and has a broad-spectrum disease resistance (Walters et al. 2005; Harel et al. 2011). Various chemical compounds have been reported to induce resistance to fungi, such as salicylic acid, 2, 6-dichloroisonicotinic acid, β-aminobutyric acid (BABA), and γ-aminobutyric acid (GABA) (Conrath et al. 2002; Yu et al. 2014). The common feature of induced resistance caused by these elicitors in plants is priming, i.e., the primed plants exhibit a faster and stronger activation of defense responses only after they have been infected by a pathogen (Conrath et al. 2002; Tonelli et al. 2011). The phenomenon of priming has been investigated in various plants, fruits, and vegetables (Sha 2005; Ahn et al. 2011; Tonelli et al. 2011; Wang et al. 2013a).

As a plant signaling molecule, methyl jasmonate (MeJA) mediates diverse developmental processes and defense responses against biotic and abiotic stresses (Cheong and Choi 2003). MeJA has been reported to enhance disease resistance in various postharvest fruits. For example, application of MeJA effectively suppressed anthracnose rot caused by *Colletotrichum acutatum* in loquat fruit (Cao et al. 2008). MeJA also has been found to increase disease resistance in

guava (González-Aguilar et al. 2004), banana (Sun et al. 2013), and citrus fruit (Guo et al. 2014). In addition, postharvest brown rot of sweet cherry caused by *Monilinia fructicola* was reduced by application of MeJA (Yao and Tian 2005). These results indicate that MeJA could be a useful technique in reducing postharvest diseases in horticultural products. However, to our knowledge, there is no information concerning the effect of MeJA on blue mold decay caused by *P. expansum* in sweet cherry fruit. Thus, the objective of this study was to evaluate the effect of MeJA on controlling blue mold decay caused by *P. expansum* in cherry fruit and to explore the possible mechanisms involved.

Materials and Methods

Pathogen

The pathogen *P. expansum* was isolated from infected sweet cherry fruit and maintained on potato dextrose agar medium (PDA, containing the extract from 200 g boiled potato, 20 g glucose, and 20 g agar in 1 L of distilled water) at 25 °C. Spores of *P. expansum* were obtained from 2-week-old cultures by flooding the cultures with sterile distilled water. The spore concentrations were determined with a hemocytometer and adjusted with sterile distilled water as required.

Fruit Material and Treatments

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. The fruits were selected for uniformity of size, ripeness, and absence of defects, and then randomly divided into two lots with 300 fruit each. Both groups were placed in 60-L airtight containers for treatment. In a preliminary experiment, we found that treatment with 10 μmol/L MeJA (Sigma-Aldrich, USA) provided greatest protection against fungal decay while no phytotoxicity occurred in sweet cherry fruit (data not shown), thus this particular concentration was used for MeJA treatment in this study. MeJA was spotted onto filter paper at final vapor concentrations of 0 (control) or 10 μmol/L within the containers, and incubated at 20 °C for 24 h. Following treatment, the containers were opened, and both groups of fruit were left at 20 °C for 6 h before challenging pathogen inoculation. Both the MeJA-treated and control fruit were sterilized with 70 % ethanol and then wounded (3 mm deep and 3 mm diameter) with a sterile nail at the equator of each fruit. The wounds were inoculated with 10 μL of a 5×10^4 spores/mL suspension of *P. expansum*. All the treatments were incubated at 20 °C with high humidity (approximately 95 %) for 5 days. Disease incidence and lesion diameter in each fruit wound were observed at 1, 3, and 5 days

postinoculation. In order to investigate the mechanisms of MeJA on reducing the blue mold decay in sweet cherry, the wounds were inoculated with *P. expansum* 6 h after the treatment with MeJA. Then, tissue samples from MeJA or sterile distilled water-treated fruit, with or without inoculation, were collected at the same intervals (0, 1, 3, and 5 days after treatment). The samples were immediately frozen in liquid N_2 and stored at −80 °C for further analysis of enzyme activity and gene expression. Each treatment was replicated three times, and the whole experiment was conducted twice.

Effect of MeJA on the In Vitro Growth of *P. expansum*

The effect of MeJA on spore germination and germ tube elongation of pathogens were assayed in potato dextrose broth (PDB) according to the method of Tian et al. (2002) with some modifications. The suspension of 1×10^5 spores/mL was prepared as described earlier. Aliquots of 100 μL of the pathogen suspension were transferred to glass tubes containing 5 mL PDB with or without 10 μmol/L MeJA. All tubes were put on a rotary shaker at 100 rpm at 28 °C. After 6, 12, and 18 h incubation, approximately 100 spores of the pathogen were observed for germination rate and germ tube length. Spores were considered germinated when germ tube length was equal to or greater than spore length. Each treatment was replicated three times, and the experiment was repeated twice.

The effects of MeJA on mycelial growth were assayed by the method of Cao et al. (2008) with some modifications. Five-mm-diameter disks were cut from PDA plates. Five-mm disks of *P. expansum* were placed in the well of each petri plate. The plates were fumigated in the sealed containers with final vapor concentrations of 0 (control) or 10 μmol/L MeJA at 20 °C for 24 h. After that, the plates were incubated at 20 °C. Colony diameter was determined 24, 48, 72, and 96 h after inoculation. Each treatment was replicated three times, and the experiment was repeated twice. Mycelial growth of *P. expansum* on PDA was expressed as growth rate, which was calculated according to the following formula: growth rate (%)=(colony diameter after inoculation−5 mm)/5 mm×100.

Assay of Enzyme Activities in Cherry Fruit

Chitinase (CHI, EC 3.2.1.14) activity was measured according to the method of Abeles et al. (1971). CHI was extracted from 1 g of frozen tissue sample with 5 mL of 50 mM sodium acetate buffer (pH 5.0). CHI activity was determined by the release of *N*-acetyl-D-glucosamine (NAG) from colloidal chitin. One unit of chitinase activity is defined as the amount of enzyme required to catalyze the production of 1 μg NAG per hour at 37 °C. The results were expressed as units per milligram protein.

β-1,3-glucanase (GLU, EC 3.2.1.58) activity was measured according to the method of Abeles et al. (1971). One

gram of frozen tissue sample was ground with 5 mL of 50 mM sodium acetate buffer (pH 5.0). One milliliter of enzyme preparation was incubated for 1 h at 37 °C with 1 mL of 4 % laminarin. The reaction was terminated by heating the sample in boiling water for 5 min, and the amount of reducing sugar was measured spectrophotometrically at 540 nm after reaction with 250 μL 3, 5-dinitrosalicyclic reagent. The specific activity of GLU was expressed as units per milligram protein. One unit is defined as the amount of enzyme catalyzing the formation of 1 μmol glucose equivalents per hour.

Enzymes of cell wall disassembly were extracted according to the method of Zhou et al. (2011) with some modifications. One gram of frozen tissue was homogenized at 4 °C in 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 homogenate was centrifuged at 25,000g for 20 min at 4 °C. The supernatant was removed and assayed in triplicate.

Polygalacturonase (PG, EC 3.2.1.15) activity was determined by the method of Zhou et al. (2011). The reaction mixture which consist of 100 μL of enzyme extract and 500 μL of 0.1 % (w/v) polygalacturonic acid in 40 mM sodium-acetate buffer (pH 5.2) were incubated at 37 °C for 2 h. The reaction was terminated with 2 mL 10 mM $Na_2B_4O_7$ (pH 9.0), followed by addition of 100 μL 1 % (w/v) 2-cyanoacetamide. The absorbance at 276 nm was measured. Reducing sugars were assayed using galacturonic acid as standard (Sigma Chemical Co., Ltd). One unit of activity was defined as the catalyzed release of 1 μmol of reducing groups per hour, per gram of the flesh of sweet cherries.

Pectinmethylesterase (PME, EC 3.1.1.11) activity was evaluated by the method of Lin et al. (1989). The reaction mixture consisted of 1 mL of enzyme extract and 10 mL of 1 % (w/v) citrus pectin (Sigma Chemical Co., Ltd) adjusted to pH 7.5 with 1 M NaOH. The difference between pH 7.5 and the final pH was used to calculate PME activity. One unit of enzyme activity was calculated as 1 mmol NaOH consumed per milligram protein per hour.

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

Analysis of Defense-Related Gene Expression by RT-PCR

Total RNA was extracted from tissue samples of cherry fruit according to the method of Chang et al. (1993). Reverse transcription-polymerase chain reaction (RT-PCR) was performed using the PrimeScriptTM 16 1st Strand cDNA Synthesis Kit (TaKaRa, Japan). Short and conserved segments of PaCAT (GenBank ID: EF165590), PaCaM (GenBank ID: AF292108), PaGLU (GenBank ID: EF177488), PaPAL (GenBank ID: AF036948), PaNPR1-like (GenBank ID: DQ146459), and PaTHAU-like (GenBank ID: U32440) were cloned by degenerate primers. Independent PCR with 30 cycles was performed using aliquots (1 μL) of cDNA samples, and a constitutively expressed gene Actin (GenBank ID: FJ560908) was used as a quantitative control in the RT-PCR analysis. The sequences of primers used for RT-PCR analysis were as follows: PaCAT forward 5′-CACAAGATTACAGGCACAT-3′, and reverse 5′-GAATAGTAGATACCAGGGACA-3′; PaCaM forward 5′-CCGATCAGCTCACAGACG-3′, and reverse 5′-TGCCCATCACCATCCACAT-3′; PaGLU forward 5′-GAAACGAAGTCAAGCCCTCA-3′, and reverse 5′-GCGGCATAAACAGCATCCAA-3′; PaPAL forward 5′-CTTGTCCCGCTGTCCTAC-3′, and reverse 5′-GCAATAGCCAAACGAGTA-3′; PaNPR1-like forward 5′-TTTCGTTGTGGAGTTGAT-3′, and reverse 5′-TATTCGGTCTTTGTTTGC-3′; PaTHAU-like forward 5′-CATTGCGATGATGAAGAC-3′, and reverse 5′-GCGTACCGAATTTAACAC-3′; and PaACTIN forward 5′-CAATGTGCCTGCCATGTATG-3′, and reverse 5′-CCAGCAGCTTCCATTCCAAT-3′. Relative mRNA levels of genes were analyzed based on densitometry values obtained using the Quality One software of Bio-Rad.

Fig. 1 Changes in disease incidence (a) and lesion diameter (b) in cherry fruit treated with MeJA and inoculated with P. expansum and incubated at 20 °C for 5 days. Each column represents the mean of three replicate samples. Vertical bars represent the standard errors of the means. Different letters above the bars indicate statistically significant differences at $p < 0.05$
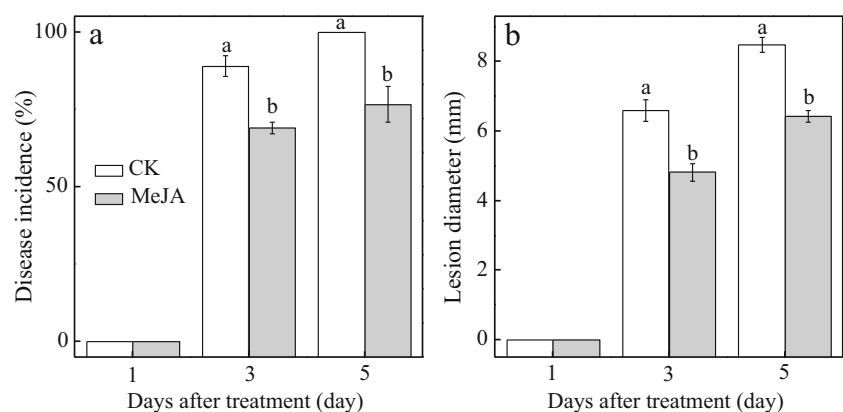
Table 1 Effect of MeJA on spore germination and germ tube elongation of *P. expansum* in vitro

Treatment	Spore germination (%)			Germ tube length (μm)		
	After 6 h	After 12 h	After 18 h	After 6 h	After 12 h	After 18 h
Control	38.2±3.11a	89.0±2.74a	98.0±1.22a	41.8±1.26a	104.3±2.48a	161.3±1.33a
MeJA	23.4±2.96b	45.8±3.27b	95.4±2.30a	37.8±0.68b	97.8±0.81b	146.3±2.48b

Germination rate and germ tube length were measured microscopically after 6, 12, and 18 h incubation at 28 °C in PDB. Data are expressed as mean± S.E. of triplicate assays. Values followed by the different letter in the same column are significantly different at *p*<0.05 according to analysis by Duncan's multiple range test

Statistical Analysis

Experiments were performed using a completely randomized design. All statistical analyses were performed using the SPSS 11.0 statistical package (SPSS, Inc., Chicago, IL) for this experiment. Analysis of variance (ANOVA) was used to compare the means. Mean separations were performed using Duncan's multiple range tests. Differences at *p*<0.05 were considered as significant.

Results

Effect of MeJA in Inhibiting Blue Mold Decay of Cherries

The disease incidence and lesion diameter of blue mold decay in cherry fruit treated with MeJA were significantly (*p*<0.05) lower than those in the control during 5 days of storage at 20 °C (Fig. 1). Compared with the control, MeJA treatment reduced disease incidence and lesion diameter by 22.4 and 26.9 %, respectively, on the 3rd day of storage (Fig. 1a, b). At the end of storage, disease incidence of MeJA-treated fruit was 76.6 %, whereas 100 % of fruit decay was shown in control fruit. The lesion diameter in MeJA-

Fig. 2 Effect of MeJA on mycelial growth of *P. expansum* in vitro. Data are expressed as the mean of triplicate assays. *Vertical bars* represent the standard errors of the means

treated fruit was 75.2 % of that in control fruit after 5 days of incubation.

Effect of MeJA on the Growth of *P. expansum* In Vitro

Spore germination and germ tube elongation of *P. expansum* in PDB were significantly (*p*<0.05) inhibited by MeJA after 6 or 12 h of incubation (Table 1). MeJA did not significantly influence the spore germination of *P. expansum* after 18 h of incubation. MeJA treatment inhibited mycelia growth of *P. expansum* on PDA. The mycelial growth rate of MeJA-treated fungi was lower than the control during 96 h of incubation; however, this difference was not significant (Fig. 2).

Effect of MeJA and *P. expansum* on Defense-Related Enzymes Activities in Fruit Wounds

CHI activity in control fruit increased only slightly before declining. The greatest increase was in MeJA+P. exp, which reached a plateau after 3 days. Application of *P. expansum* or MeJA alone also induced higher activity than control on day 3 and 5, but lower than in MeJA+P. exp (Fig. 3a). The fruit that were both treated with MeJA and inoculated with *P. expansum* showed 7.11 and 21.9 % higher activity of CHI than those only treated with MeJA or inoculated with *P. expansum* after 5 days of incubation, respectively. When fruit were inoculated with *P. expansum*, GLU activity increased slightly in control fruit over 5 days at 20 °C. Applying P. exp or MeJA alone notably increased GLU activity at day 1 and 3, respectively. Again, the highest increase in GLU activity was observed in MeJA+P. exp (Fig. 3b).

Effect of MeJA and *P. expansum* on the Activities of PG and PME in Fruit Wounds

PG activity in control fruit increased during 5 days at 20 °C. *P. expansum* inoculation caused a greater increase, and MeJA resulted in less increase in activity compared with the control fruit. PG activity in the combined treatment was similar to that of using MeJA alone for the first 3 days and then increased.
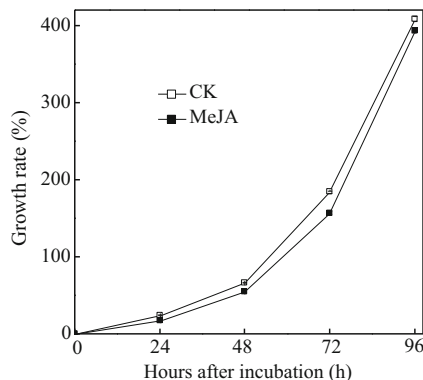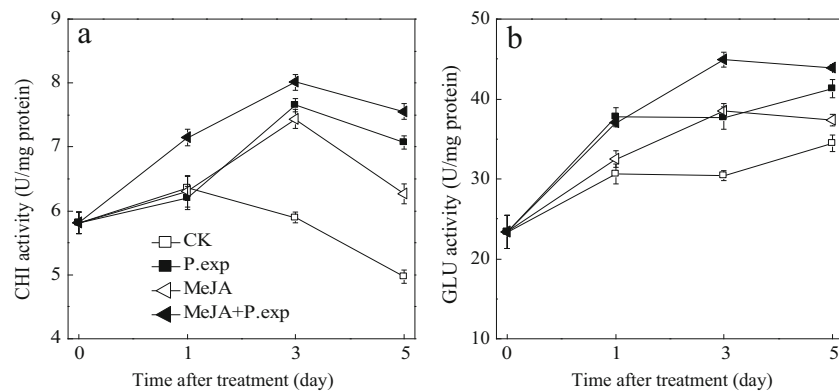
Fig. 3 Effect of MeJA and *P. expansum* on the activities of CHI (**a**), and GLU (**b**) in cherry fruit wounds incubated at 20 °C for 5 days. The wounds were inoculated with *P. expansum* 6 h after the treatment with MeJA. Then, tissue samples from MeJA or sterile distilled water-treated fruit, with or without inoculation, were taken for enzyme extractions. Data are expressed as the mean of triplicate samples. Standard errors of three replications are given as the *short bars*

PME activity was rather flat for control and MeJA-treated fruit for first 3 days and then increased, while *P. expansum* alone or together with MeJA had higher activities after 3 and 5 days. The activities of PG and PME in fruit treated with MeJA and followed by inoculation with *P. expansum* were 16.2 and 19.1 %, respectively, higher than those in fruit treated with MeJA alone after 5 days of inoculation (Fig. 4a, b).

Effect of MeJA and *P. expansum* on Defense-Related Genes Expression in Sweet Cherry Fruit

As shown in Fig. 5, of the gene expression examined, *PaCAT*, *PaPAL*, and *PaTHAU-like* all increased greatly in MeJA-treated fruit compared with the control. The expression of the genes for *PaCaM*, *PaGLU*, and *PaNPR1-like* were increased by *P. expansum* and by the combined treatment, but not by MeJA alone. Transcript levels of all six genes in fruit treated with MeJA and inoculated with *P. expansum* were significantly enhanced and maintained at high level in all the sampling time points compared with the other three treatments
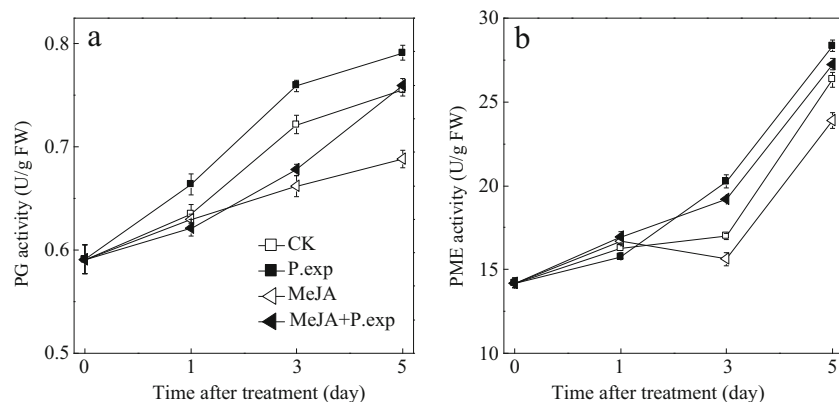
(Fig. 5). These results indicated that MeJA primed stronger expression of the six defense-related genes in cherry fruit upon challenged with the pathogen of *P. expansum*.

Discussion

Induced disease resistance in harvested horticultural crops using physical, biological, and/or chemical elicitors is considered a preferred strategy for disease management (Durrant and Dong 2004). It has been reported that MeJA treatment could effectively inhibit postharvest diseases of various fruits including loquat (Cao et al. 2008), sweet cherry (Yao and Tian 2005), and peach (Jin et al. 2009), possibly by directly suppressing pathogen growth, and/or indirectly inducing disease resistance. In this study, we found that MeJA treatment could effectively reduce blue mold decay in sweet cherry fruit wounds inoculated with *P. expansum* during 5 days of storage at 20 °C (Fig. 1). These results suggest that the disease

Fig. 4 Effect of MeJA and *P. expansum* on the activities of PG (**a**) and PME (**b**) in cherry fruit wounds incubated at 20 °C for 5 days. The wounds were inoculated with *P. expansum* 6 h after the treatment with MeJA. Then, tissue samples from MeJA or sterile distilled water-treated fruit, with or without inoculation, were taken for enzyme extractions. Data are expressed as the mean of triplicate samples. Standard errors of three replications are given as the *short bars*

resistance of sweet cherry fruit was enhanced by postharvest treatment with MeJA.

To further understand the mechanisms by which MeJA reduced blue mold decay caused by *P. expansum* infection, we investigated the direct effect of MeJA on *P. expansum* in vitro. Our results showed that MeJA had a transient effect on delaying spore germination and inhibiting germ tube elongation of *P. expansum*, which may function in defense mechanisms against blue mold decay in MeJA-treated cherry fruit. It has been reported that MeJA had an inhibitory effect on several fungal pathogens including *C. acutatum* (Cao et al. 2008) and *Botrytis cinerea* (Zhu and Tian 2012). However, other researchers observed that MeJA could not directly inhibit the growth of some pathogens such as *Penicillium digitatum* and *M. fructicola* (Droby et al. 1999; Yao and Tian 2005). These different phenomena might be related to different sensitivities of fungal species to MeJA.

The protection of fruit from invasion of fungal pathogens is largely due to activation of a highly coordinated biochemical and structural defense system that helps ward off the spread of pathogens (Yao and Tian 2005). Pathogenesis-related (PR) proteins are a class of defense molecules that are induced by pathogen attack, wounding, or environmental stressors (Van Loon 1985). Among PR proteins, CHI and GLU hydrolyze polymers of fungal cell walls and are thought to be involved in plant defense mechanisms against fungal infection (Vilanova et al. 2014). Induction of these enzymes by MeJA has been observed in postharvest peaches, which was correlated to the increased disease resistance and reduced disease severity (Yao and Tian 2005). Catalase (CAT) is an important enzyme that protects plants against oxidative damage (Gill and Tuteja 2010). CaM is involved in plant defense responses. The previous studies provided evidence that CaM might be one of the key players in transducing the pathogen-induced Ca^{2+}
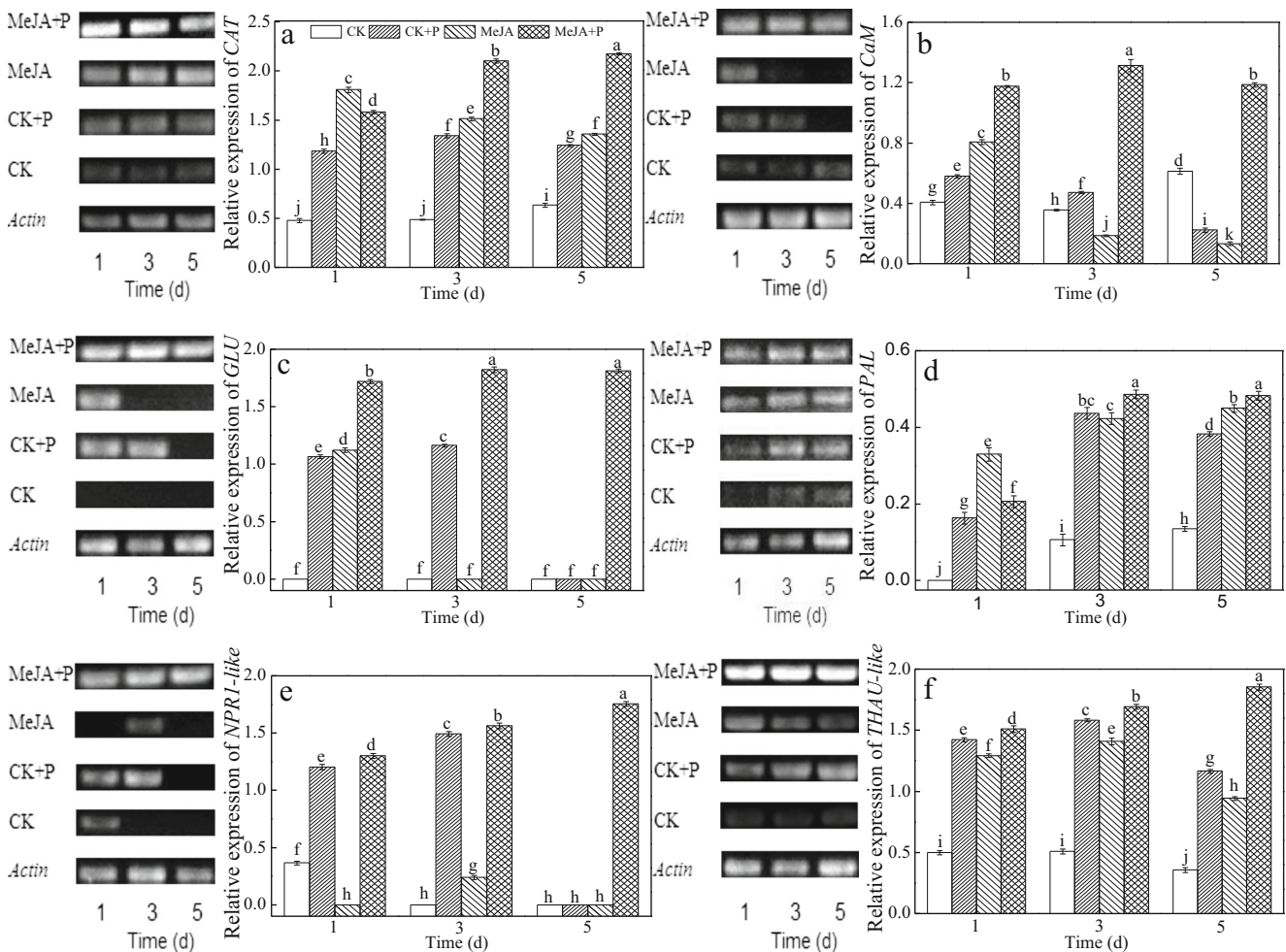

Fig. 5 Effect of MeJA and *P. expansum* on the expression of defense-related genes in cherry fruit wounds incubated at 20 °C for 5 days. **a–f** represent the gene expression of *CAT*, *CaM*, *GLU*, *PAL*, *NPR1-like*, and *THAU-like*, where the *left* is RT-PCR, and the *right* is analysis of relative expression of genes. The wounds were inoculated with *P. expansum* 6 h after the treatment with MeJA. Then, tissue samples from MeJA or sterile distilled water-treated fruit, with or without inoculation, were taken for gene expression. Data are expressed as the mean of triplicate samples. Standard errors of three replications are given as the *short bars*

increase to downstream components of defense signaling (Chiasson et al. 2005; Takabatake et al. 2007; Zhu et al. 2010). Phenylalanine ammonia-lyase (PAL) is a key enzyme in the phenylpropanoid pathway leading to the biosynthesis of phenolics, phytoalexins, and many other compounds related to disease resistance (Dixon and Paiva 1995). NPR1 is a key regulator in the signal transduction pathway that leads to induced resistance and increased the expression of PR genes (Kinkema et al. 2000). Grenier et al. (1999) reported that the THAU-like proteins hydrolyzed polymeric β-1, 3-glucans by using an in-gel glucanase assay. Wang et al. (2013b) reported that *Bacillus subtilis* induced the expression of CAT and NPR1-like and elevated the resistance of peach fruit against *Rhizopus* rot caused by *Rhizopus stolonifer*. Yu et al. (2014) found that GABA strongly enhanced the activities or gene expression of GLU and PAL and reduced the disease incidence caused by *P. expansum* in pear fruit. Several studies clearly showed that CaM played a critical role in plant defense (Harding et al. 1997; Takabatake et al. 2007; Zhu et al. 2010). Our data showed that MeJA significantly enhanced the activities of CHI and GLU, together with increased expression of defense-related genes in cherry fruit, especially when inoculated with *P. expansum*, which might contribute to the fruit resistance against *P. expansum* by the MeJA treatment.

The mechanisms of induced resistance appear to involve the priming of defenses that are expressed following a challenge inoculation with a virulent strain of a pathogen (Hammerschmidt 2008). Priming is a process of induced resistance, which is often associated with an enhanced capacity to mobilize infection-induced defense responses (Conrath et al. 2002). Our observation is in agreement with the definition of priming. Therefore, we infer that the induced disease resistance by MeJA in sweet cherry might be associated with mechanisms of priming. The application of MeJA triggered the defense response at enzymatic and transcript level. As indicators of defense expression, the antimicrobial enzymes and transcription of defense-related genes measured in this study showed that only in fruit that had been primed with MeJA and subsequently challenged with *P. expansum*, significant increase in enzymes and defense-related genes expression were observed. The phenomenon of priming has also been found in *Arabidopsis* treated with BABA, peaches treated with *B. subtilis* AR156, and grapevine treated with *Pseudomonas fluorescens* (van Hulten et al. 2006; Verhagen et al. 2010; Wang et al. 2013b). Priming seems to be the strategy followed by several beneficial inducers to enhance resistance, avoiding a direct activation of defenses which would be too expensive for the host in the absence of challenging attackers (van Hulten et al. 2006). Therefore, induced resistance by means of priming offers an efficient form of plant protection with significant benefits under conditions of disease occurrence.

Furthermore, the softening of fruit will facilitate the infection of pathogen and increase postharvest decay (Wei et al. 2010). This complex biochemical process is correlated with the action of a number of cell wall-modifying enzymes. PG and PME are two major enzymes that act on the pectin fraction of the cell wall (Li et al. 2010). Cantu et al. (2008) reported that suppression of tomato fruit softening reduced susceptibility to *B. cinerea* infection. Roper et al. (2007) reported that PG is required for *Xylella fastidiosa* to successfully infect grapevines. In the present study, the relatively lower activities of PG and PME in MeJA-treated cherries may contribute to the enhanced disease resistance.

In conclusion, our results demonstrated that MeJA treatment could effectively delay the development of decay caused by *P. expansum* in harvested sweet cherry fruit. It is postulated that the control of the disease is directly due to the transient inhibitory effect of MeJA on pathogen growth, and indirectly due to the induced disease resistance primed by MeJA. Moreover, the enhanced disease resistance may be also due to lower activities of PG and PME in fruit treated with MeJA. Future research is needed to further elucidate the mechanisms of MeJA in inhibiting postharvest decay in fruit, particularly the molecular mechanisms underlying defense priming.

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