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Effects of pre- and post-harvest application of salicylic acid or methyl jasmonate on inducing disease resistance of sweet cherry fruit in storage

Hongjie Yao[a,b], Shiping Tian[a,*]

[a] *Key Laboratory of Photosynthesis and Environmental Molecular Physiology, Institute of Botany, The Chinese Academy of Sciences, Xiangshan Nanxincun 20, Haidian District, Beijing 100093, China*
[b] *Graduate School of the Chinese Academy of Sciences, Yuquanroad 19, Haidian District, Beijing 100039, China*

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Abstract

Pre-harvest treatments with 2 mM salicylic acid (SA) and 0.2 mM methyl jasmonate (MeJA) significantly reduced lesion diameters on sweet cherry fruit caused by *Monilinia fructicola* compared with control post-harvest treatments. Pre-harvest treatment of sweet cherry with SA or MeJA induced β-1,3-glucanase, phenylalanine ammonia-lyase (PAL) and peroxidase (POD) activities during the early storage time. The efficacy of inducing resistance in sweet cherry fruit pre-harvest-treated with SA or MeJA to *M. fructicola* was better than that for fruit with post-harvest treatments, especially, at 25 °C. Activities of β-1,3-glucanase and PAL in SA- or MeJA-treated cherry fruit stored at 25 °C for both pre- and post-harvest treatments were significantly higher than those in fruit stored at 0 °C. SA with a concentration of 2 mM showed direct fungitoxicity on *M. fructicola* and significantly inhibited mycelial growth and spore germination of the pathogen in vitro. MeJA at 0.2 mM had little inhibitory effect on mycelial growth and spore germination of *M. fructicola*. The fruit treated with MeJA pre-harvest expressed higher activity of β-1,3-glucanase and PAL than fruit treated with SA and the control during the early storage time.

Keywords: Salicylic acid; Methyl jasmonate; Sweet cherry fruit; Inducing resistance

1. Introduction

Fungal diseases result in major losses of fruits and vegetables and can be controlled effectively by synthetic chemical fungicides (Snowdon, 1992; Eckert and Ogawa, 1998). However, the development of fungicide resistance by pathogens and increasing environmental concern over fungicidal residues in fruit and vegetables have stimulated a search for alternative measures for disease control (Holmes and Eckert, 1999; Castoria et al., 2001; Fan and Tian, 2001; Zhang et al., 2002). Recently, many reports have shown that induced disease resistance in plants by biotic and abiotic

* Corresponding author. Tel.: +86 10 82594675; fax: +86 10 82594675.
E-mail addresses: tsp@ibcas.ac.cn, shipingt@vip.sina.com (S. Tian).

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elicitors is a very effective method for restricting the spread of fungal infection (Rodov et al., 1994; Droby et al., 2000; Soylu et al., 2003). Resistance of plants to pathogens is based on both constitutive defense mechanisms such as pre-existing antimicrobial compounds and inducible defense mechanisms. Induced disease resistance in plants by biotic or abiotic treatments is a very attractive strategy for controlling diseases (Droby et al., 2002; Qin et al., 2003).

The signal molecules salicylic acid (SA), jasmonic acid (JA) and methyl jasmonate (MeJA) are endogenous plant growth substances that play key roles in plant growth and development, and responses to environmental stresses. These signal molecules are involved in some signal transduction systems, which induce particular enzymes catalyzing biosynthetic reactions to form defense compounds such as polyphenols, alkaloids or pathogenesis-related (PR) proteins (Hahlbrock and Scheel, 1989; Creelman and Mullet, 1995; Tamari et al., 1995; Van Loon, 1995). This can result in induction of defense responses and provide protection for plants from pathogen-attack (Delaney et al., 1994; Kozlowski et al., 1999). Such signaling molecules, when exogenously applied, have been shown to move systemically through plants, resulting in the expression of a set of defense genes that are activated by pathogen infection, thus inducing resistance against pathogens (Epple et al., 1997; Kozlowski et al., 1999).

Sweet cherry (*Prunus avivum* L.) fruit is very susceptible to post-harvest decay caused by some pathogenic fungi, brown rot caused by *Monilinia fructicola* being a major fungal disease of sweet cherry in China (Tian et al., 2001). Effective control of brown rot disease of sweet cherry fruit requires novel strategies. The objectives of this study were to investigate post-harvest diseases of cherry fruit treated pre- and post-harvest with SA or MeJA, as well as to determine changes in activities of β-1,3-glucanase, phenylalanine ammonia-lyase (PAL) and peroxidase (POD) during storage in fruit treated with or without SA and MeJA.

2. Materials and methods

2.1. Plant material

Sweet cherry (*P. avivum* L. cv.) fruit were harvested from an orchard of the Institute of Forest and Fruits, Beijing Academy of Agricultural Sciences. After harvest, the fruit were pre-cooled and immediately transported to our laboratory (Institute of Botany, Chinese Academy of Sciences). The fruit were sorted for uniform size and maturity, without wounds or rots.

2.2. Pathogen

M. fructicola was isolated from decayed sweet cherry fruit. The fungus was maintained on potato-dextrose agar (PDA) at 4 °C. Spores of *M. fructicola* were obtained from 2-week-old PDA cultures incubated at 25 °C by flooding the cultures with sterile distilled water containing 0.05% (v/v) Tween-80, and filtered through four layers of sterile cheesecloth. The concentration of spores was adjusted to 1×10^3 conidia ml^{-1} with the aid of a haemocytometer.

2.3. Treatments

2.3.1. Pre-harvest treatment

In order to choose suitable concentrations of SA and MeJA, preliminary experiments were carried out. Two micromoles of SA and 2 mM MeJA showed no phytotoxicity on the fruit and induced the greatest responses (data not shown). Before preparing the MeJA solution, MeJA was dissolved in 1% (v/v) Tween-80. Sweet cherry trees were sprayed with SA (2 mM) or MeJA (0.2 mM), using a hand-held sprayer (each spray volume consisted of 15 L per tree). Fruit sprayed with sterile distilled water were used as controls. Three days after the treatments, the fruit were harvested. A sample of fruit was not wounded, and was analyzed for the percentage of infected fruit. Disease incidence was determined 15 days after treatment at 25 °C or 60 days after treatment at 0 °C. Another sample of fruit from these three treatments was wounded (a uniform hole 3 mm deep and 3 mm wide) with a sterile borer. After 4 h, the fruit were challenge-inoculated with 15 μl of conidial suspension of *M. fructicola*. The treated fruit were put in 200 mm × 130 mm × 50 mm plastic boxes at high humidity (about 95%) and stored at 25 or 0 °C, respectively. Disease incidence and lesion diameter were determined 3 days after treatment at 25 °C or 50 days after treatment at 0 °C, respectively. There were three replicates (boxes) for each treatment, each replicate (box) containing 10 fruit.