Postharvest Biology and Technology 64 (2012) 133–137

Contents lists available at ScienceDirect

Postharvest Biology and Technology

journal homepage: www.elsevier.com/locate/postharvbio

Effect of 1-methylcyclopropene (1-MCP) on reducing postharvest decay in tomatoes (*Solanum lycopersicum* L.)

Hai Su[1], W. Douglas Gubler*

Department of Plant Pathology, University of California, Davis, CA 95616, United States

ARTICLE INFO

Article history:
Received 15 December 2010
Accepted 12 June 2011

Keywords:
1-Methylcyclopropene
1-MCP
Alternaria alternata
Botrytis cinerea
Fusarium spp.
Tomatoes

ABSTRACT

1-Methylcyclopropene (1-MCP as SmartFresh™ technology), an ethylene antagonist, was evaluated for affecting postharvest decay caused by *Alternaria alternata*, *Botrytis cinerea*, and *Fusarium* spp. on 'Quality 23' and 'Seminis 35' tomatoes at green or pink stages. Fruit with natural or artificial infection were subjected to 1-MCP at 0.0 $\mu L L^{-1}$, 0.6 $\mu L L^{-1}$ for 12 h, and 1.0 $\mu L L^{-1}$ for 6 h. After 31–42 d storage, disease incidence and severity of individual diseases in 1-MCP treated fruit was significantly reduced compared with that of the untreated controls, except in one inoculated test for 'Quality 23' where severity of Alternaria rot in 1.0 $\mu L L^{-1}$ treated fruit were significantly higher than that of the untreated control. Fruit treated with 1-MCP at 1.0 L^{-1} for 6 h also had significantly higher incidence of Alternaria rot in the inoculated 'Quality 23' and in the non-inoculated 'Seminis 35' compared with the fruit treated with 1-MCP at 0.6 $\mu L L^{-1}$ for 12 h. The results of this study indicate that 1-MCP can reduce postharvest decay within a certain storage period.

1. Introduction

Postharvest decay, caused by various pathogens, is a great challenge for transportation and storage of tomato fruit (Barkai-Golan, 2001). Ripening stage of tomato fruit can be an important factor affecting decay after harvest. As the fruit ripen, tissue texture loosens due to disassembly of cell wall components and cells release substrates, which provide nutrients for pathogens and cause ripe fruit to be more susceptible to diseases.

Various enzymes, including polygalacturonase (PG), either from fruit or microbes, are involved in the process of tissue or cell wall dissembling in the ripening process (Vicente et al., 2007). PG is not only produced by fruit during ripening but also is secreted by *Botrytis cinerea* during infection and is considered one of the factors for pathogenicity (Leone, 1992). It has been shown that suppression of the PG gene could reduce the growth of *B. cinerea* in the fruit (Cantu et al., 2008).

The ripening process of tomatoes is induced by ethylene (Alexander and Grierson, 2002). In commercial production, tomatoes are usually harvested at the mature-green stage and later treated with ethylene for ripening before being marketed to consumers. As an elicitor of many plant genes, ethylene also regulates antimicrobial activities (Ecker and Davis, 1987; Nimchuk et al., 2003). Treatment of tomato leaves with ethylene increased resistance to infection of *B. cinerea*, while inhibition of ethylene perception with 1-methylcycloproene (1-MCP) increased the susceptibility to the pathogen (Díaz et al., 2002). Infection of *B. cinerea* also could induce the ethylene and jasmonic acid-related gene expression and signaling pathway in tomato fruit (De Martinis and Benvenuto, 2003), though its role in relation to disease resistance is complex.

However, it has been found that tomato plants that are insensitive to ethylene are less susceptible to infection of *Xanthomonas campestris* pv. *vesicatoria*, *Psudomonas syringae* pv. *tomato* and *Fusarium oxysporum* f. sp. *lycopersici* (Lund et al., 1998). Exogenous ethylene caused more necrosis, while inhibition of ethylene limited disease development on plant tissues, including tomato, infected with *B. cinerea* (Elad, 1990). It has been shown that infection by *B. cinerea* increases ethylene production and accelerates ripening processes, but does not affect susceptibility of pears (Akagi and Stotz, 2007). Ethylene also stimulates spore germination and mycelium growth of certain pathogens including *B. cinerea* (El-Kazzaz et al., 1983; Barkai-Golan and Lavy-Meir, 1989). Since ethylene acts as an elicitor of both ripening and plant defense (Alexander and Grierson, 2002), the eventual outcome of exogenous ethylene treatments can vary depending on multiple factors.

Antagonists of ethylene have been well studied and in recent years 1-MCP has emerged as an excellent ethylene antagonist for commercial application (Blankenship and Dole, 2003; Sisler, 2006;

* Corresponding author. Tel.: +1 530 752 0304; fax: +1 530 752 5674.
E-mail address: wdgubler@ucdavis.edu (W.D. Gubler).
[1] Current address: Marrone Bio Innovations, Inc., Davis, CA 95618, United States.

0925-5214/$ – see front matter © 2011 Published by Elsevier B.V.
doi:10.1016/j.postharvbio.2011.06.005

Watkins, 2006). 1-MCP has been proven to be very effective in delaying ripening and in extending the shelf life of tomatoes (Wills and Ku, 2002; Mostofi et al., 2003; Mir et al., 2004; Guillén et al., 2007). Tomato fruit treated with 1-MCP were firmer and PG activity in tomato fruit was suppressed (Choi and Huber, 2008).

Though a plethora of studies have been conducted on the effect of 1-MCP on improving physiological characters and extending shelf life of fruits and vegetables, including tomatoes, direct evidence of its effect on postharvest decay caused by pathogens is relatively sparse. It has been shown that inhibition of ethylene production by 1-MCP does not affect the expansion of *B. cinerea* in pear fruit (Akagi and Stotz, 2007) and may reduce disease resistance in strawberries (Jiang et al., 2001). The objective of this study was to evaluate if 1-MCP treatment could affect postharvest decay caused by *A. alternata*, *B. cinerea*, and *Fusarium* spp. in tomato fruit.

2. Materials and methods

2.1. Preparation of tomato fruit

Tomato fruit used in the experiments were purchased from San Joaquin Tomato Growers, Inc. (Crows Landing, CA). Cultivar 'Quality 23' fruit were treated with ethylene for 72 h and 'Seminis 35' for 12 h before purchase. Pink to light red stage fruit is more responsive to 1-MCP treatment (Hurr et al., 2005). Thus the fruit were placed at room temperature (≈25 °C) until the majority of fruit reached pink stage and were sorted for uniformity according to their ripening stage. The fruit that remained at green stage after 30-d storage at room temperature (≈25 °C) were also used for one repeated test.

There were four replicates per treatment. The number of fruit per replicate was determined based on the availability of the fruit with the same ripening stage. For each cultivar, the same number of fruit was used and the fruit were stored in both crisper boxes and original commercial cartoons in either inoculated or non-inoculated tests (Tables 1–4).

For 'Quality 23', there were 20 green stage fruit per replicate, which were individually placed in plastic crisper boxes (18 cm × 24 cm area) with lids open to allow air movement. There were 30 pink stage fruit per replicate, which were packed in original commercial cartoons with lids on but with side openings in repeated experiments. All the fruit were stored for 42 d.

For 'Seminis 35', there were 10 pink stage fruit per replicate, which were stored in crisper boxes for 33 d. There were 15 pink stage fruit per replicate, which stored in commercial cartoons for 31 d in repeated experiments.

All the fruit were stored in a controlled storage room at 15 °C and 90% relative humidity (RH) after being treated with 1-MCP.

2.2. Preparation of inoculum and fruit inoculation

A *B. cinerea* isolate was cultured on 5-cm petri dishes with King's medium B for 10 d and an *A. alternata* isolate on 10-mm petri dishes with potato dextrose agar (PDA) for 20 d at room temperature (≈25 °C) to produce conidia.

The inoculation was dispersed over tomato fruit using a customized inoculation tower which measured 1.2 m high, 0.6 m long, and 0.6 m wide. The fruit were placed in the base of the tower and each individual petri dish with *B. cinerea* and *A. alternata* was placed on the petri dish holder, which was hung on the ceiling of the tower. A new culture plate was used for each inoculation to reduce variation. A vacuum was applied for about 1 min to reach 3.4 kPa inside the tower and then the lid was opened suddenly to allow air to push into the tower and generate air current. The conidia were released by air current and were allowed to settle for 5 min to reach the tower base where fruit were placed. Inoculum level was determined by placing three 7.6 cm × 2.5 cm glass slides at the tower base. The number of conidia for each pathogen was determined by counting vertical crosses three times under 10× magnification with a microscope. The average counts were 965 spores cm^{-2} for *B. cinerea* and 100 spores cm^{-2} for *A. alternata*. The fruit were moved back to original carton boxes or crisper boxes for later 1-MCP treatments or were used as untreated controls.

2.3. Treatment with 1-MCP

There were three treatments, including controls, which were not treated with 1-MCP (SmartFresh™ Technology) for both non-inoculated fruit (natural infection) and inoculated fruit: (1) untreated control (0.0 μL L^{-1} 1-MCP); (2) treated with 0.6 μL L^{-1} 1-MCP for 12 h (commercial rate) and (3) treated with 1.0 μL L^{-1} 1-MCP for 6 h.

Table 1
Incidence and severity of individual diseases in 'Quality 23' fruit treated with 1-MCP at green stage (20 fruit per replicate) after 42 d storage (turning to pink stage) in crisper boxes at 15 °C and 90% RH.

Disease	Inoculation	1-MCP (μL L^{-1})	Incidence (%)	Severity (%)
Alternaria rot (*Alternaria alternata*)	No	0.0	4.5 ± 3.0	0.2 ± 0.1
		0.6	4.1 ± 2.5	0.3 ± 0.2
		1.0	0.0 ± 0.0	0.0 ± 0.0
	Yes	0.0	13.1 ± 6.1	1.2 ± 0.6
		0.6	2.7 ± 1.5	0.5 ± 0.4
		1.0	1.5 ± 1.5	0.5 ± 0.5
Botrytis rot (*Botrytis cinerea*)	No	0.0	3.1 ± 1.8	1.3 ± 0.8
		0.6	2.7 ± 1.6	0.4 ± 0.3
		1.0	1.4 ± 1.4	0.7 ± 0.7
	Yes	0.0	16.9 ± 6.8	5.6 ± 2.3
		0.6	5.2 ± 0.1	1.7 ± 0.9
		1.0	11.6 ± 2.6	6.3 ± 1.3
Fusarium rot (*Fusarium* spp.)	No	0.0	13.4 ± 3.7	2.1 ± 0.6
		0.6	7.0 ± 1.4	2.0 ± 0.9
		1.0	4.2 ± 1.4	0.6 ± 0.4
	No	0.0	12.9 ± 1.6 a***	3.5 ± 1.2 A*
		0.6	1.3 ± 1.3 b	0.3 ± 0.3 B
		1.0	0.0 ± 0.0 b	0.0 ± 0.0 B

Data with different letters in a column for the inoculated or non-inoculated treatments of each disease are significantly different according to Fisher's least significant difference (LSD) at $\alpha = 0.05$ level.
Data are means ± S.E. ($n = 4$). Data of different treatments not labeled with letters are not significantly different for each disease with or without inoculation.
* $P \leq 0.05$.
*** $P \leq 0.001$