Ripening of Kiwifruit Exposed to Ethanol and Acetaldehyde Vapors

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Abstract. Exposure to acetaldehyde (M) vapors that produced tissue levels of 0.02% AA (w/w) stimulated ripening of 'Hayward' kiwifruit (*Actinidia deliciosa* L.) much more effectively than exposure to ethanol vapors that produced tissue levels of 0.18% ethanol. Tissue levels of 0.02% and 0.18% ethanol stimulated ripening, while the ripening rate of tissue with 0.04% was similar to the controls. Ethylene and CO₂ production from M-treated tissues were, respectively, 23 times and 60% higher than from control tissues. AA induced a rapid softening that was localized in the core tissue and rendered the fruit unmarketable for 7 days after treatment.

A brief postharvest exposure to ethanol vapors reduced the rate of tomato fruit ripening (Saltveit and Mencarelli, 1988), possibly through its effect on membranes associated with ethylene action (Saltveit, 1989). Treatment with ethanol also reduces the astringency of Japanese persimmon (Pesis and Ben-Arie, 1984) while stimulating respiration and ethylene synthesis (Itamura, 1986; Ito, 1971). Exposure to acetaldehyde (AA) vapors promotes ripening and ethylene synthesis in pears (Janes and Frenkel, 1978) as well as color formation and a climacteric-like respiratory rise in blueberry and straw-

berry fruit (Janes et al., 1978).

Kiwifruit are very sensitive to ethylene and their storability is inversely correlated with the concentration of ethylene in the storage atmosphere (Arpaia et al., 1980; Ito and Hashinaga, 1985). Kiwifruit are also very susceptible to mechanical injury at harvest and during shipping due to flesh softening (Gatti, 1984). Results reported in this paper show the effect of exposure to ethanol and AA vapors on the ripening and softening of kiwifruit.

'Hayward' kiwifruit were purchased from a commercial source during the harvest season; late October to the end of November 1988. Within a few hours of harvest, the fruit were sorted on the basis of fruit size and firmness. Fruit were subjectively judged to be hard when they did not deform under slight hand pressure. Fruit were separated

Table 1. Soluble solids content and titratable acidity of kiwifruit treated with ethanol and acetaldehyde vapors for 7 h and held at 17C in ethylene-free air for 13 days.

Tissue levels (w/w)	Time after treatment (days)	
	1	13
Soluble solids content (%) ^a		
Ethanol		
0.18%	6.2	10.4
0.04%	6.1	8.7
0.02%	5.9	9.6
Acetaldehyde		
0.04%	5.8	12.9
0.02%	5.9	12.8
Control	6.2	9.3
LSD 5%	0.3	---
LSD 1%	---	0.8
Titratable acidity (% citric acid) ^b		
Ethanol		
0.18%	2.0	1.3
0.04%	2.1	1.5
0.02%	1.9	1.2
Acetaldehyde		
0.04%	2.0	1.6
0.02%	1.9	0.9
Control	1.9	1.0
LSD 5%	NS	---
LSD 1%	---	0.18

^aData are the mean of three readings from each fruit, for three fruits for each replicate, and two replicates per each of two experiments. Mean separation by LSD at *P* = 0.05 or 0.01.

^bData are the mean of readings from 10 g of tissue from each fruit, for three fruits for each replicate, and two replicates per each of two experiments. Mean separation by LSD at *P* = 0.05 or 0.01.

into 12 groups of 13 fruit each (≈ 1.5 kg/group); two replicates for each of the six treatments (three ethanol levels, two AA levels, and a water control). Each of the 12 groups of fruit was placed in a 5-liter jar, the jar capped with a large two-hole rubber stopper, and reagent-grade absolute ethanol, AA, or distilled water then was injected into the jar through serum stoppers in the holes in the rubber stopper as described by Saltveit

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Fig. 1. Core tissue firmness of kiwifruit exposed to ethanol and acetaldehyde vapors for 7 h at 17C and held in ethylene-free air at 17C for 1, 7, or 13 days. Mean separation for each variable by Duncan's multiple range test, $P = 0.01$.

Fig. 2. Flesh firmness of kiwifruit exposed to ethanol and acetaldehyde vapors for 7 h at 17C and held in ethylene-free air at 17C for 1, 7, and 13 days. Mean separation for each variable by Duncan's multiple range test, $P = 0.01$.

and Mencarelli (1988). Calculated amounts of ethanol and AA were introduced into the jars to produce concentrations of 0.02%, 0.04%, or 0.18% (w/w) ethanol and 0.02% or 0.04% (w/w) AA in the kiwifruit tissue. After holding the jars for 7 h at 17C, the jars were flushed with air and the fruit were kept for 13 days in an ethylene-free atmosphere at 17C. Less than 0.7% CO_2 had accumulated in the jars at the end of the 7-h treatment.

Fruit ripening was followed by measuring fruit firmness, soluble solids content (SSC),

titratable acidity (TA), and CO_2 and C_2H_4 production. Samples of three fruit from each replica were taken for firmness, SSC, and TA measurements after 1, 7, and 13 days. Firmness was measured with an Instron Universal Testing Machine model 4301 (Instron Ltd., High Wycombe, England) fitted with a flat, 8-mm-diameter probe by compressing flesh and core tissue of a 1-cm-thick slice cut from the equatorial portion of each fruit. The full scale range was 49 N for the flesh and 196 N for the core. TA was determined by titrating expressed juice from 10 g of tis-

Table 2. ACC content ($\text{nmol}\cdot\text{g}^{-1}$ fresh weight) of kiwifruit treated with ethanol and acetaldehyde vapors for 7 h and held at 17C in ethylene-free air for 13 days. The ACC content was 0.023 $\text{nmol}\cdot\text{g}^{-1}$ before treatment.

Tissue levels (w/w)	ACC content ($\text{nmol}\cdot\text{g}^{-1}$)	
	Time after treatment (days)	
	1	13
Ethanol 0.04%	0.052 b ^z	0.061 b
Acetaldehyde 0.04%	0.42 a	3.2 a
Control	0.026 c	0.051 b

Data are the mean readings from each fruit, for three fruits for each replicate, and two replicates per each of two experiments. Mean separation for each variable within columns by Duncan's multiple range test, $P = 0.01$.

sue to pH 8.2 with 0.1 N NaOH and is presented as percent citric acid. The percent SSC of the expressed juice was determined with a Galileo Abbe refractometer (Galileo SpA., Florence, Italy). Carbon dioxide concentrations were measured with an Isolcell Infrared Analyzer and C_2H_4 concentrations were measured with a Carlo Erba model Fractovap gas chromatography (Carlo Erba SpA., Milan, Italy) equipped with an 80 to 100 mesh alumina column kept at 100C and a flame ionization detector as described by Mencarelli and Saltveit (1988). Content of l-amino cyclopropane-1-carboxylic acid (ACC) was evaluated according to Lizada and Yang (1979). The experiment was conducted twice with similar results.

Subjective and objective measurements failed to detect any changes in firmness between the initial and day 1 observations. After 7 days, there were no differences in core tissue firmness among the ethanol treatments and the control. After 13 days of storage, 0.02% and 0.18% ethanol had reduced core firmness, while fruit with 0.04% ethanol were as firm as the control (Fig. 1). In contrast, ethanol had no significant effect on flesh firmness after any storage period (Fig. 2). AA-treated fruit behaved differently. Within 7 days of treatment, core tissue firmness was reduced by > 90% by 0.04% and 0.02% AA (Fig. 1). These treatments also reduced flesh firmness by > 80% to 90% (Fig. 2). The rapid softening produced by both AA concentrations rendered the fruit unmarketable because of excessive softness.

The ratios of core to flesh firmness after 1 day of treatment were between 5 and 6 for all samples. After 7 days, the ratios had dropped to < 1 for both AA treatments, while the ratio increased to between 6 and 8 for the ethanol treatments and the water control. After 13 days, the ratios for all AA treatments and for the 0.18% ethanol treatment were similar and significantly lower than for the other treatments and the control, which were between 5 and 7. These lower ratios at 13 days indicate that both AA levels and 0.18% ethanol hastened ripening. This conclusion is confirmed by the higher SSC for these treated fruit in comparison to the 0.02% and 0.04% ethanol treatments and the water control (Table 1).

Fig. 3. Production of CO_2 in $\text{ml}(\text{kg}\cdot\text{h})^{-1}$ from kiwifruit exposed to ethanol and acetaldehyde vapors for 7 h at 17°C and subsequently held in ethylene-free air at 17°C .

Fig. 4. Production of C_2H_4 in $\mu\text{l}(\text{kg}\cdot\text{h})^{-1}$ from kiwifruit exposed to ethanol and acetaldehyde vapors for 7 h at 17°C and subsequently held in ethylene-free air at 17°C .

Titrateable acidity declined with storage for all treatments (Table 1). The decline from day 1 to day 13 ranged from an average of 50% for the control and 0.02% AA-treated tissue, to an average of 34% for the ethanol-treated tissue, to 20% for the 0.04% AA treatment. The 0.04% ethanol and the 0.04% AA treatments retained the most titrateable acidity, 71% and 80%, respectively. These data suggest that the metabolism of organic acids was most inhibited by the 0.04% ethanol and AA treatments, less by the remaining ethanol treatments, and not at all by 0.02% AA.

One day after treatment, CO_2 production was higher from tissue exposed to ethanol or AA than from the controls, and then gradually declined (Fig. 3). AA-treated tissue had a small peak in CO_2 production 2 days after treatment, and had significantly higher rates

of CO_2 production than fruit from the other treatments for the duration of the experiment. In contrast, there was no peak in CO_2 production from ethanol-treated tissue, and the initial stimulation gradually declined until the rates of CO_2 production were similar to the controls 10 days after treatment.

AA-treated fruit exhibited a large increase in the rate of ethylene evolution that peaked 10 days after treatment, and the rate of evolution was proportional to the applied concentration of AA (Fig. 4). Carbon dioxide production did not parallel the large increases in C_2H_4 production from AA-treated fruit. Ethylene production from ethanol-treated fruit was similar to that of the control, but 20 to 25 times less than that from the AA-treated fruit. A small increase in C_2H_4 production peaked around the 10th day for fruit with tissue levels of 0.02% and 0.18%

ethanol, while C_2H_4 production from tissue with 0.04% ethanol was lower than for the control.

The ACC content was measured in control fruit and fruit with tissue levels of 0.04% ethanol and 0.04% AA because they showed very different ripening behavior. Seven days after treatment, fruit with 0.04% AA had 16 times more ACC than air control fruit, while fruit with 0.04% ethanol had only twice the control level of ACC (Table 2). After 13 days of storage, the ACC content of the AA-treated tissue had increased to 63 times the air control, while the increase in ACC content of the ethanol-treated tissue was now similar to that in the controls. The ACC content of control fruit had almost doubled between 7 and 13 days of storage, but it still was much lower than in the AA-treated tissues.

An untrained, informal taste panel was unable to detect any flavor differences among the treated and control fruit after 13 days, except for the 0.02% and 0.04% AA and 0.18% ethanol treated fruit, which had a flat, insipid flavor and were too soft.

Exogenous application of AA stimulated the ripening of kiwifruit, as described with other fruits (Janes and Frenkel, 1978; Janes et al., 1978). Fruit with tissue levels of 0.04% ethanol ripened more slowly than fruit with tissue levels of 0.02% or 0.18% ethanol. The differential effect of ethanol on inhibiting ethylene action (Saltveit, 1989) and of its close metabolite, AA, on promoting ripening (Janes and Frenkel, 1978) may account for the anomalous response of ripening kiwifruit to ethanol.

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