The Use of Antioxidants to Delay the Onset of Anthracnose and Stem End Decay in Avocado Fruits After Harvest

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ABSTRACT

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Infiltration of avocado fruits (cultivar Fuerte) with 0.1 mM solutions of an antioxidant (butylated hydroxy toluene [BHT], α-tocopherol, *tert*-butylhydroquinone [TBHQ], epicatechin, or 0.01% gum guaiac) delayed the appearance of anthracnose symptoms for 1–2 days. A mixture of 0.1 mM BHT and either 0.5% ascorbic or 0.1% citric acid enhanced the activity of BHT alone and delayed the appearance of anthracnose symptoms for a further day without affecting fungal growth in vitro. Butylated hydroxy anisole and propyl gallate (0.1 mM) had no effect on disease expression. Treatments of fruits with 0.1 mM epicatechin or a mixture of 0.1 mM BHT and 0.5% ascorbic acid delayed the disappearance of endogenous epicatechin and the antifungal diene, and delayed disease expression; infiltration with 1 mM TBHQ, however, enhanced the disappearance and disease expression. In semicommercial trials, stem end rot in cultivars Ettinger and Fuerte was also delayed for 1–4 days as a result of dip treatment in a mixture of 0.1 mM BHT and either 0.5% ascorbic or 0.1% citric acid.

Additional keywords: antifungal compounds, quiescent infections, postharvest diseases, postharvest treatments

Anthracnose, caused by *Colletotrichum gloeosporioides* Penz., and stem end rot, caused by *Diplodia natalensis* Pole-Evans, are important fruit-rot diseases of avocado in the U.S.A., Israel, and Australia (8,12,17,18). *C. gloeosporioides* infects avocado peel (2) and *D. natalensis* infects the fruit pedicel (1) during the growing season, but the infections remain quiescent until fruit ripen after harvest. Under commercial conditions, decay development can usually be delayed either by storage at low temperature (5–6 C) (13) or by fungicide treatment (7). The latter is applied as preharvest orchard sprays or, more efficiently, as postharvest dip treatments (7).

Prusky et al (10) isolated a preformed antifungal compound from the peel of unripe avocado fruits and identified it as *cis*,*cis*-1-acetoxy-2-hydroxy-4-oxo-heneicosa-12,15 diene (Fig. 1). This compound was shown to be involved in the mechanism for quiescence of *C. gloeosporioides* infections in unripe avocado (10). The antifungal diene also inhibited elongation of germinating *D. natalensis* hyphae with an ED_{50} similar to that of *C. gloeosporioides* (480 μg/ml). It was suggested that lipoxygenase catalyzes the oxidation of the antifungal diene during fruit ripening, resulting in its decrease to subfungitoxic concentrations and, therefore, in its decrease in activation of fungal infections (Fig. 2) (9). Infiltration of fruits with α-tocopherol (BDH), an inhibitor of lipoxygenase activity, inhibited the decrease of the antifungal diene and also the development of anthracnose lesions (9). It was concluded that lipoxygenase activity in the peel of ripening fruits could be regulated by concentration of the natural antioxidant epicatechin (Fig. 2) (11). Reduction in concentration of epicatechin in the peel after harvest was always found to be associated with a decrease in diene concentration and development of anthracnose lesions (*unpublished*).

This contribution reports on exogenous postharvest treatments that inhibited lipoxygenase activity and delayed the activation of quiescent infections and development of anthracnose and stem end decay.

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MATERIALS AND METHODS

Avocado fruits (cultivars Ettinger and Fuerte) were obtained from an orchard near Rehovot, Israel. A single-spore isolate of *C. gloeosporioides* from decayed avocado fruits was used in all experiments. Cultures of this strain were maintained either on avocado fruits or on potato-dextrose agar (PDA) plates (10). Fruits were inoculated with a 10-μl drop of a spore suspension (10^6 spores/ml) placed at four different positions along both sides of the longitudinal axis of each of 20 freshly harvested fruits and then incubated at 20 C. Fruits were examined daily for disease symptoms using a binocular microscope. Peel darkening exceeding 1 mm in diameter was considered positive for disease expression. The effects of the treatments were expressed as days from inoculation to disease expression. Standard deviation was calculated for 80 infection points per treatment.

Effect of antioxidants on *C. gloeosporioides* decay development. Twenty-four hours after inoculation, opposite halves of the same fruits were infiltrated with either antioxidant or solvent solutions (9). Infiltration was carried out by immersing the appropriate half of the fruit and reducing the pressure to 100 mm Hg for 60 sec. The amount of solution absorbed by each half fruit was 0.20 ± 0.08 g per 100 g of fruit. Fruits were infiltrated with α-tocopherol, butylated hydroxy toluene (BHT), butylated hydroxy anisole (BHA), gum guaiac, *tert*-butylhydroquinone (TBHQ), and epicatechin in 0.1% ethanol, 0.1% dimethyl sulfoxide (DMSO), and 0.01% Triton X-100. The same detergent-ethanol mixture, without antioxidant, served as a control. In some experiments, ascorbic or citric acid at 0.5% and 0.1%, respectively, were added to BHT as

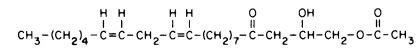

Fig. 1. Structural formula of the antifungal diene from avocado.

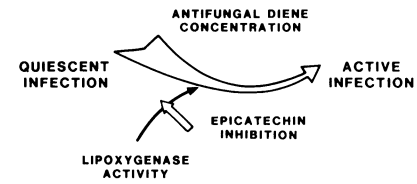

Fig. 2. Sequence of reactions leading to active infections of *Colletotrichum gloeosporioides* in ripening avocado: lipoxygenase-catalyzed decrease in diene concentration facilitates activation of quiescent infection. Epicatechin inhibits lipoxygenase activity.

synergists of antioxidant activity (14). Experiments were repeated at least twice.

Effect of antioxidants on *C. gloeosporioides* growth in vitro. Antioxidants dissolved in 95% ethanol were amended into PDA. Antioxidant-amended and unamended plates were inoculated with 5-mm-diameter plugs of fungal mycelium sampled from a 7-day-old culture. The diameter of growth developed at 20 C and was checked daily. Presence of ethanol at a concentration of 1% in the media did not affect *C. gloeosporioides* growth.

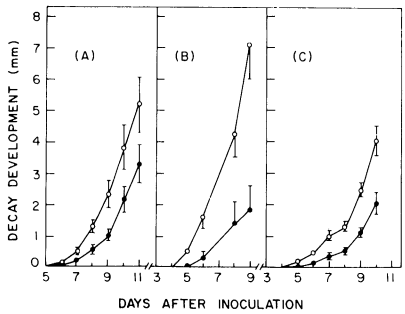

Fig. 3. Effect of antioxidants on decay development of *Colletotrichum gloeosporioides* in avocado fruits stored at 20 C. One-half of the whole fruit was infiltrated 24 hr after inoculation of freshly harvested fruits with **(A)** butylated hydroxy toluene, **(B)** α-tocopherol at 0.1 mM, and **(C)** gum guaiac at 0.01% in 0.01% Triton X-100, 0.1% DMSO, and 0.1% ethanol (●). Control halves (o) were infiltrated with the solvent alone. Decay development is expressed as the diameter of peel darkening at the inoculated site. Darkening of the peel exceeding an area of 1 mm in diameter was considered as symptom expression of disease. Standard deviation is provided in bracket.

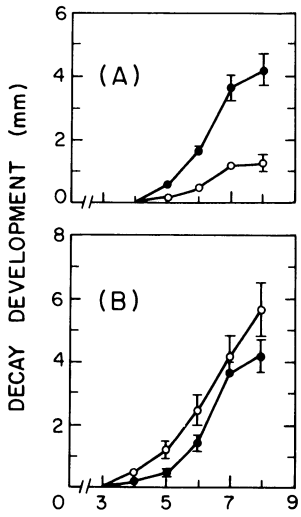

Fig. 4. Effect of *tert*-butylhydroquinone on decay development of *Colletotrichum gloeosporioides* in avocado fruits stored at 20 C. Freshly harvested fruits were inoculated. Twenty-four hours later, one-half of the whole fruit was infiltrated either with the solvent (controls) consisting of 0.01% Triton X-100, 0.1% DMSO, and 0.1% ethanol (o), or with solvent also containing TBHQ (●) at concentrations of **(A)** 1 mM or **(B)** 0.1 mM. Decay development is expressed as the diameter of peel darkening at the inoculated site. Darkening of the peel exceeding an area of 1 mm in diameter was considered symptom expression of disease. Standard deviation is provided in bracket.

Extraction and quantitative analysis of the antifungal diene and epicatechin. The antifungal diene was extracted from avocado peels and quantified as described by Prusky et al (10). The lipoxygenase inhibitor, epicatechin, was extracted in 5-mM sodium phosphate buffer (pH 7.2), partially purified by flash chromatography, and quantified by HPLC as described by Prusky et al (9).

Effect of antioxidant and fungicide dip treatment on development of *D. natalensis* decay in semicommercial trials. In these experiments, fruits were not artificially inoculated. To assure a high incidence of stem end rot, avocado cultivars Fuerte and Ettinger were picked early during the harvesting season from orchards known for high incidence of the disease (12,13). The fungicide imidazole (Prochloraz, 45 EC) 1-*N*-propyl-*N*-[2-(2,4,6-trichlorophenoxy)ethyl]carbamoylimidazole (7,12) was compared with antioxidant treatments for control of stem end rot. Twenty-four hours after harvest, fruits were dipped for 30 sec in 900 μg/ml prochloraz or in aqueous antioxidant solutions at 0.1 mM BHT and 0.5% ascorbic acid, and 0.1 mM BHT and 0.1% citric acid. After treatment, fruits were air-dried and stored at 20 C or stored at 2 C for 10 days and then transferred to 20 C. Symptoms of Diplodia stem end rot exceeding 1 cm deep in the fruit flesh were considered positive for disease expression. Each treatment was replicated six times with 20 fruits in each replicate. Experiments were conducted twice in each of two harvesting seasons.

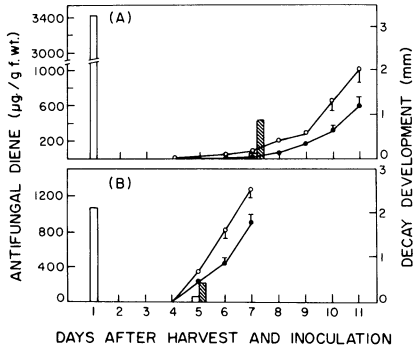

Fig. 5. Effect of epicatechin on the antifungal diene concentration and decay development by *Colletotrichum gloeosporioides* in avocado fruits stored at 20 C. Epicatechin (0.1 mM), in 0.01% Triton X-100, 0.1% DMSO, and 0.1% ethanol (●), was infiltrated into fruit halves 24 hr after inoculation of freshly harvested fruits. Control halves were infiltrated with the solvent alone (o). Concentration of the antifungal diene in crude extracts of peel treated with epicatechin (hatched bar) or the solvent alone (open bar). Two different experiments were carried out **(A)** in the beginning of the harvest season and **(B)** 3 mo later. Darkening of the peel exceeding an area of 1 mm in diameter was considered symptom expression of disease. Standard deviation is provided in bracket.

RESULTS

Effect of antioxidants on *C. gloeosporioides* decay development. Infiltration of avocado fruits with 0.1 mM BHT, 0.1 mM α-tocopherol, or 0.01% gum guaiac delayed decay development by *C. gloeosporioides* on the treated side of the fruits (Fig. 3) compared with the control side. Butylated hydroxy toluene delayed disease expression by 1 day, and α-tocopherol and gum guaiac delayed it by 2 days. Inhibition of lesion expansion was observed until 9–11 days after harvest in the antioxidant-treated versus control halves of the fruits. Treatments with 0.1 mM BHA or 0.1 mM propyl gallate were ineffective (results not presented). The effect of TBHQ differed with concentration. *Tert*-butylhydroquinone at 0.1 mM delayed disease expression by 1 day (Fig. 4B), but treatment with 1 mM TBHQ significantly hastened disease expression by 1 day (Fig. 4A). Effects of treatments lasted for at least 8 days after harvest.

The addition of either 0.1% citric or 0.5% ascorbic acid enhanced the activity of BHT. In both treatments disease expression was delayed by 1 day. Eight days after inoculation, the lesion diameter on the BHT-ascorbic acid- and BHT-citric acid-treated sides was 1.0 ± 0.2 mm and 0.7 ± 0.3 mm, respectively; in the BHT controls, the lesion averaged 1.8 ± 0.3 mm. Neither 0.1% citric nor 0.5% ascorbic acid affected decay development significantly. Eight days after inoculation, lesion diameter averaged 1.5 ± 0.3 mm on the control side and 1.7 ± 0.5 mm on the ascorbic acid-infiltrated side. On fruits treated with citric acid, the lesion averaged 2.2 ± 0.3 mm on the control side compared with 1.8 ± 0.3 mm on the treated side.

C. gloeosporioides growth was not affected by mixtures of 0.1 mM BHT with either 0.1% citric or 0.5% ascorbic acid added to PDA. After 7 days at 20 C, the diameter of 5-mm fungus circles had grown to 5.8 ± 0.1 mm in the antioxidant-amended and unamended plates.

Effect of antioxidants on epicatechin and diene concentration and on decay development of *C. gloeosporioides*. *Effect of epicatechin treatments on diene concentration and decay development.* Two experiments were carried out during one harvesting season. At the beginning of the season, the concentration of the antifungal diene in the fruit peel was 3,450 μg/g fresh wt 1 day after harvest (Fig. 5A). Infiltration with 0.1 mM

epicatechin delayed the decrease of the antifungal diene. Seven days after harvest, diene concentration was 460 μg/g fresh wt on the epicatechin-treated side and 60 μg/g fresh wt on the control side. Concentrations of endogenous epicatechin on the epicatechin-treated and control sides were 674 and 451 μg/g fresh wt, respectively, 2 days after treatment. Four days after treatment, epicatechin concentrations were similar in both sides. In the second experiment, 3 mo later, the initial concentration of the diene was 1,100 μg/g fresh wt. Five days later, concentrations of the diene on the epicatechin-treated and control sides of the fruit were 250 and 90 μg/g fresh wt, respectively (Fig. 5B). Epicatechin treatment delayed expression of disease in both experiments by 1 day.

Effect of BHT-ascorbic acid treatment on epicatechin and diene concentration. Infiltration of avocado fruits with a mixture of 0.1 mM BHT and 0.5% ascorbic acid delayed the decrease of endogenous epicatechin concentration that occurs after harvest (Table 1). In the peel on the antioxidant-mixture-treated side, the concentration of epicatechin was higher throughout the experiment. The antioxidant treatment also delayed the diene decrease that occurred in the peel on the control side following the fifth day after harvest. On the seventh day, diene concentration on the control side was about 25% of that on the treated side. Nine days after harvest, disease was expressed on the control side, whereas on the treated side it was expressed on the tenth day.

Effect of TBHQ treatment on epicatechin and diene concentration. Infiltration with 1 mM TBHQ enhanced disease expression by 1 day (Fig. 4) and it also enhanced the decrease of endogenous epicatechin and antifungal diene concentration of the fruit (Table 2). Concentration of epicatechin on the control side had decreased to 288 μg/g fresh wt on the eighth day after harvest, whereas on the treated side it was already 62 μg/g fresh wt. Diene concentration in the TBHQ-treated side of the fruit was lower throughout the experiment. On the eighth day after harvest, it was 520 μg/g fresh wt on the treated side and 2,050 μg/g fresh wt on the control side. On the twelfth day after harvest, disease expression occurred on the TBHQ-treated side, whereas on the control side it occurred on the thirteenth day.

Effect of antioxidants and fungicide dip treatment on development of *D. natalensis* decay in semicommercial trials. Dip treatment of Ettinger fruits in a mixture of BHT and either 0.1% citric or 0.5% ascorbic acid delayed symptoms of stem end rot by about 4 days (Fig. 6). After the decay appeared, it increased more rapidly in fruits dipped in BHT and ascorbic acid than in the mixture of BHT and citric acid. Dip treatments of Fuerte fruits (Fig. 7) on a mixture of BHT and citric acid delayed disease expression by 2 days compared with controls. The mixture of BHT and ascorbic acid and the commercial fungicide prochloraz at 900 μg/ml delayed disease expression by only 1 day.

DISCUSSION

The resistance of unripe avocado fruits to attack by postharvest pathogens was associated with the preformed antifungal compound called diene (10). Previous results (9,11) led to the belief that diene decrease was a result of increased lipoxygenase activity. Activity of this enzyme was regulated by the decrease in concentration of the flavan 3-ol inhibitor, epicatechin (11). In the present work, compounds that inhibited lipid peroxidation activity of lipoxygenase (4,16) were tested as possible inhibitors of diene peroxidation. They delayed the development of anthracnose and stem end rot. Infiltration treatments of avocado with α-tocopherol, BHT, TBHQ at 0.1 mM, and 0.01% gum guaiac delayed the expression of anthracnose symptoms by 1–2 days (Figs. 1 and 2).

Treatments with mixtures of 0.1 mM BHT and either 0.5% ascorbic or 0.1% citric acid delayed the expression of disease symptoms by 1 day more than BHT alone. These mixtures did not inhibit the fungus in culture, and their effect must have been on plant metabolism. Plant lipoxygenases are strongly inhibited by some polyphenols

Table 1. Effect of infiltration of avocado fruits (cultivar Fuerte) with a mixture of 0.1 mM butylated hydroxy toluene (BHT) and 0.5% ascorbic acid on the concentration of epicatechin and antifungal diene, and on decay development of *Colletotrichum gloeosporioides* at 20 C

Days after harvest	Epicatechin[a] (μg/g fresh wt)		Diene[a] (μg/g fresh wt)		Decay development (mm)	
	Control	BHT +[b] ascorbic acid	Control	BHT + ascorbic acid	Control	BHT + ascorbic acid
5	913	1,173	1,490	1,500		
6	1,431	1,817	500	1,480		
7	516	786	120	500		
9	12	47			1.9[c]	1.0 *
10					3.3	1.8 *

[a] Samples of partially purified extracts were chromatographed on reverse-phase HPLC columns and peak heights were compared with those of standards.
[b] Fruits were infiltrated with an aqueous solution of 0.01% Triton X-100, 0.1% dimethyl sulfoxide, and 0.1% ethanol. The same solution, without the antioxidant, was infiltrated into control fruits.
[c] Fruits were inoculated by placing 10-μl drops of a spore suspension (10^6 spores/ml) at four different positions along both sides on each of 20 freshly harvested fruits. Decay development was measured as darkening of the peel. Expression of disease was recorded when peel darkening exceeded an area of 1 mm in diameter. * = Means for treated versus control were different at $P = 0.05$.

Table 2. Effect of infiltration of avocado fruits (cultivar Fuerte) with 1 mM *tert*-butylhydroquinone (TBHQ) on the concentration of epicatechin and diene, and on decay development by *Colletotrichum gloeosporioides* at 20 C

Days after harvest	Epicatechin[a] (μg/g fresh wt)		Diene[a] (μg/g fresh wt)		Decay development (mm)	
	Control	TBHQ[b]	Control	TBHQ	Control	TBHQ
1	1,235		720			
4	572	233	910	560		
6	639	273	1,940	510		
8	288	62	2,050	520		
11	28	13	830	440		
12					0.7[c]	1.0 *
13					1.0	2.0 *
14					1.6	3.6 *

[a] Samples of partially purified extracts were chromatographed on reverse-phase HPLC columns and peak heights were compared with those of standards.
[b] Fruits were infiltrated with an aqueous solution of TBHQ in 0.01% Triton X-100, 0.1% dimethyl sulfoxide, and 0.1% ethanol. The same solution, without the antioxidant, was infiltrated into control fruits.
[c] Fruits were inoculated by placing 10-μl drops of a spore suspension (10^6 spores/ml) at four different positions along both sides on each of 20 freshly harvested fruits. Decay development was measured as darkening of the peel. Expression of disease was recorded when peel darkening exceeded an area of 1 mm in diameter. * = Means for treated versus control were different at $P = 0.05$.

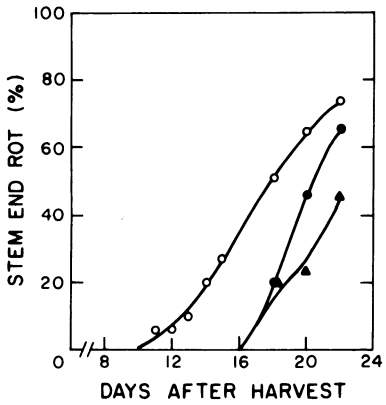

Fig. 6. Effect of postharvest dips in antioxidants on the incidence of *Diplodia natalensis* stem end rot on stored avocado fruit (cultivar Ettinger). Fruits were dipped in a mixture of 0.1 mM BHT and 0.5% ascorbic acid (o) or 0.1 mM BHT and 0.1% citric acid (▲). Controls (●) were dipped in water only. Dipped fruits were stored at 2 C for 10 days and then transferred to 20 C. *D. natalensis* penetration into fruit flesh to a depth of 1 cm was considered rot development.

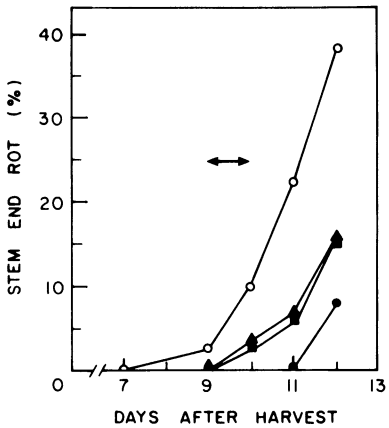

Fig. 7. Effect of postharvest dips in antioxidants and fungicide on the natural incidence of *Diplodia natalensis* stem end rot on stored avocado fruits (cultivar Fuerte). Fruits were dipped in a mixture of 0.1 mM BHT and 0.5% ascorbic acid (■), 0.1 mM BHT and 0.1% citric acid (•), or 900 $\mu g/ml^{-1}$ prochloraz (▲). Control fruits (o) were dipped in water. Fruits were stored at 20 C throughout the experiment. *Diplodia* penetration into fruit flesh to a depth of 1 cm was considered disease expression. Horizontal arrow indicates fruit were completely softened.

(6,15). The inhibition, which is often competitive, is the result of the removal of free radicals required for the reaction (3). When functioning as antioxidants, phenols constitute a trap for free radicals, resulting in their own oxidation to a very stable product (3), preventing epicatechin oxidation and inhibiting lipoxygenase increase and diene decrease. A similar effect was observed with an exogenous treatment of epicatechin, which prevented endogenous epicatechin decrease and delayed the diene decrease.

Tert-butylhydroquinone (0.1 mM) delayed disease expression by 1 day but at 1 mM, epicatechin and antifungal diene decrease and decay development were enhanced (Table 2). Antioxidants at certain concentrations act as prooxidants (5), indicating the importance of optimization of treatments because different concentrations may have opposite effects. This indicates that the regulation of decay development, initiated by the decrease of epicatechin and then of the diene, is a process that might be affected in either way.

The mixture of BHT and ascorbic or citric acid delayed the beginning of stem end rot development in semicommercial experiments by 1–4 days. A mixture of BHT and citric acid was more effective than the commercial fungicide prochloraz used in Israel for this purpose (11).

Treatments of avocado fruits with antioxidants delay the conversion of quiescent infections of *C. gloeosporioides* and *D. natalensis* into active infections and disease. This delay seems to depend on prevention of the decline of the natural mechanisms of resistance.

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