

Changes in the Ripening Process of Avocado Fruit Infected by *Fusarium solani*

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ABSTRACT

Infection of avocado fruit by *Fusarium solani* caused an accelerated softening when compared with uninfected fruit. The basic physiological processes related to ripening were found to be similar in infected and uninfected fruit; however, an increase in respiration rate, ethylene evolution, activity of

pectolytic enzymes, and changes in pectic substances accompanying fruit softening, occurred earlier in the infected fruit. Apparently *F. solani* causes earlier ripening and softening processes in avocado.

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Additional key words: respiration pattern.

The avocado (*Persea americana* Mill.) differs from other fruit in that it does not ripen while still on the tree (5), but ripens and softens several days after harvest. At softening, various fungi which cause rot may develop on the fruit (13), among them some species of *Fusarium* (15). Previous observations showed that avocado fruit naturally infected by *Fusarium solani* (Mart.) App. and Wr. softened after harvest earlier than uninfected fruit. Softening of the infected fruit occurred during the incubation period of the fungus, before the appearance of the first sign of rot; softening did not progress from the infection point, but occurred in the whole fruit at once. With the softening of the fruit the rot started at the infection point as a brown spot, which grew larger and became covered with white mycelium. Naturally- and artificially-inoculated fruit behaved similarly.

There are reports in the literature indicating that some physiological processes change in organs infected by fungi. For example, an increase in respiration rate was found in fruit infected by different fungi (8, 12, 19). In different plant organs infected by various microorganisms, an increase in ethylene evolution (4, 6, 7, 11, 14), changes in pectic substances (9, 18) and activity of pectolytic enzymes were found (1, 2, 18).

The purpose of this study was to determine whether the nature of the early softening of the avocado fruit infected by *F. solani* was similar to that in sound fruit, and whether the earlier softening was an acceleration of the ripening processes that occur during fruit softening. The processes accompanying ripening and softening in *F. solani*-infected and uninfected fruit were studied.

MATERIALS AND METHODS.—Avocado (cultivar 'Fuerte') fruits were inoculated by placing 0.1 ml of a suspension of *Fusarium* spores (10^6 spores/ml) into a 0.5-cm wound in the fruit. The inoculation site was covered with cellotape and the fruit stored at 20 C and 90% relative humidity. Sound and wounded, but not inoculated, fruit served as controls. The experiments were repeated during 3 yr, three or four times per yr, with three to five replicates in each experiment.

Respiration and ethylene evolution determinations were made on individual fruit placed in 2-liter glass containers. The respiration rate of the fruit was

determined by Biale's method (3, 6). Ethylene evolution was measured by gas chromatography (Packard Factory). Pectin methylesterase (PME) and polygalacturonase (PG) were determined according to a method described elsewhere (20). The fractions of water-soluble pectin and protopectin were also determined (10, 16, 17).

RESULTS AND DISCUSSION.—The results obtained in this study during the 3 yr of experiments were similar. It was found that in all experiments fruit infected with *F. solani* started to soften 3-4 days earlier than the uninfected fruit. Rot development started 1 to 2 days after the beginning of softening. No basic changes in physiological processes related to ripening were found in fruit infected by *F. solani*, when compared with uninfected fruit; however, some changes in the rates of these processes were observed. The small wounds on the fruit caused during inoculation did not cause changes in the physiological processes tested or in rate of fruit softening. It was found that as long as the fruit remained firm, the fungus remained at the inoculation point.

In fruit infected by *F. solani*, the respiration rate was accelerated; the respiration rise and the climacteric peak appeared earlier than in the uninfected fruit, but the pattern and the intensity of respiration were the same. In infected fruit, the climacteric peak of respiration appeared 4 days after harvest and inoculation, and in the uninfected fruit 7 days after harvest. Softening of the infected and uninfected fruit occurred 1 to 2 days after the climacteric peak of respiration, and therefore it occurred in the infected fruit 5 days after harvest and in the uninfected fruit 8 days after harvest. The first symptoms of rot appeared with the softening of the fruit (Fig. 1).

Similarly, ethylene evolution started earlier in the infected fruit than in the uninfected ones. However, the maximal values for ethylene evolution were the same in infected and in uninfected fruit. The peak of ethylene evolution in infected fruit appeared 4 days after infection, and in uninfected fruit 9 days after infection (Fig. 2).

An effect of fungus infection on the rates of change of pectic substances was found. In the infected fruit, the fraction of water-soluble pectin increased and the fraction of protopectin decreased faster, than in the uninfected

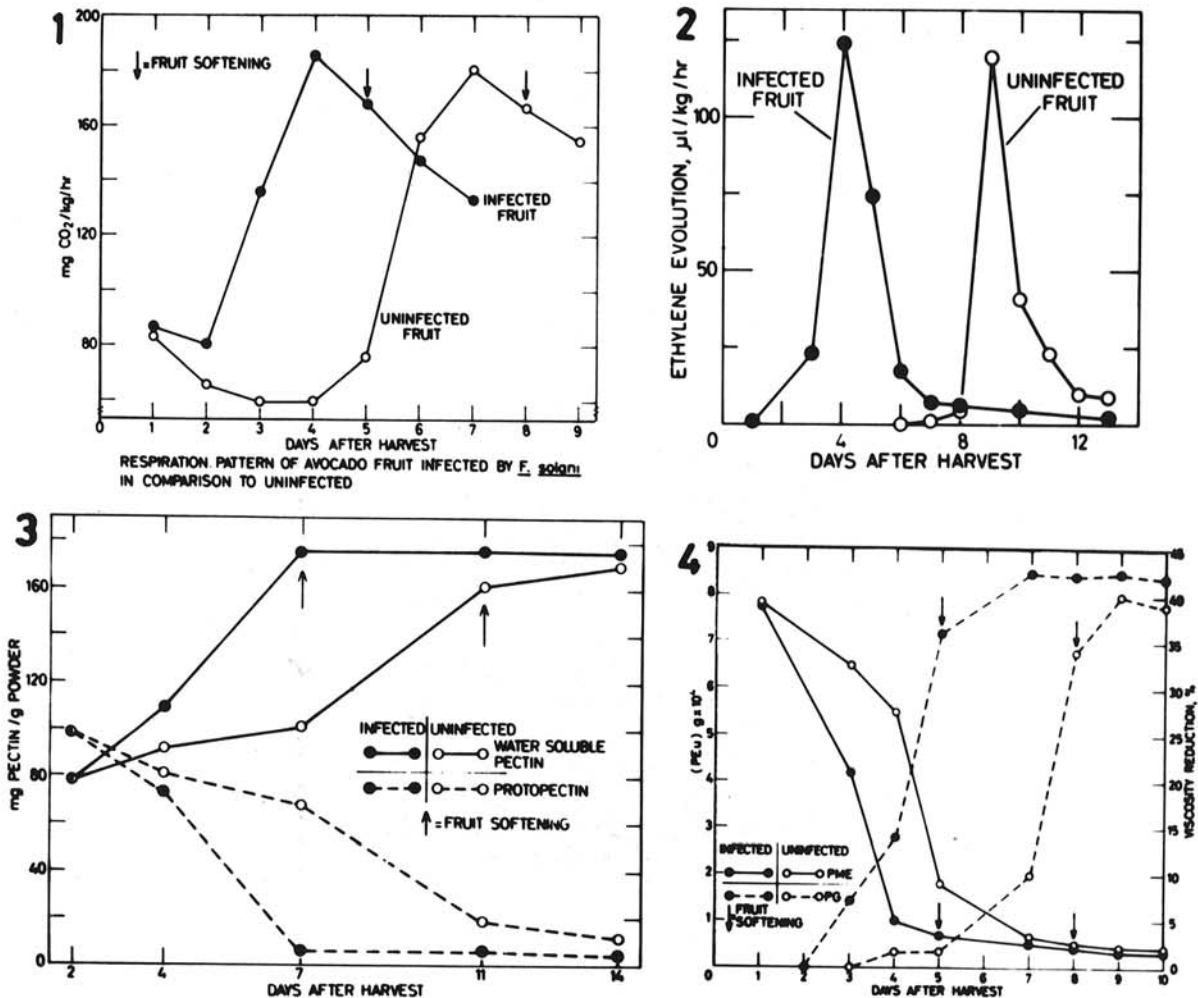


Fig. 1-4. 1) Respiration pattern of avocado fruit infected by *Fusarium solani* in comparison to uninfected fruit. 2) Ethylene evolution in avocado fruit infected by *F. solani* in comparison to uninfected fruit. 3) Changes in pectic fractions in avocado fruit infected by *F. solani* in comparison to uninfected fruit. 4) Pectin methylesterase and polygalacturonase activity in avocado fruit infected by *F. solani* in comparison to uninfected fruit.

fruit; however, the final values of these pectic substances did not differ in infected and uninfected fruit. The high values of water-soluble pectin and the low values of protopectin were reached in infected fruit 7 days after harvest and inoculation, and in uninfected fruit 11 days after. The softening of the fruit was related to the changes in pectic substances in both the infected and uninfected fruit. Since the softening of the fruit started with the increase in the amount of water-soluble pectin and decrease of protopectin fractions, it occurred earlier in the infected than uninfected fruit (Fig. 3).

The effect of fungus infection was noticeable also on the activity of pectic enzymes. PME activity reached its minimum and PG activity its maximum earlier in infected fruit than in uninfected fruit. In infected fruit the PME activity reached its low value 4 days after and the PG activity its high value 7 days after harvesting and inoculation, compared with 7 and 9 days after harvesting, respectively, in uninfected fruit. The softening of the fruit

occurred when the PME activity decreased to a low value and PG activity reached its high value (Fig. 4).

The results of this study show that the physiological processes related to fruit softening were basically similar in both infected and uninfected fruit; however, in the infected fruit these processes were accelerated. The fact that the early fruit softening caused by fungus infection does not progress gradually from the infection point but occurs evenly over the whole fruit, may mean that the early softening is not a result of the local effect of the fungus presence, but of the ability of the fungus to trigger the mechanism of fruit softening, similar to that occurring in uninfected fruit. It may be that the fungus affects fruit softening by triggering the mechanism of ethylene evolution, thus bringing about earlier ethylene evolution and earlier fruit softening. The acceleration of fruit softening caused by fungus infection is similar to that caused by the effect of exogenous ethylene treatment. The fungus infection, similar to ethylene treatment, causes

acceleration of fruit softening and of ripening processes without basic changes in their pattern, thus causing earlier senescence of the fruit.

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