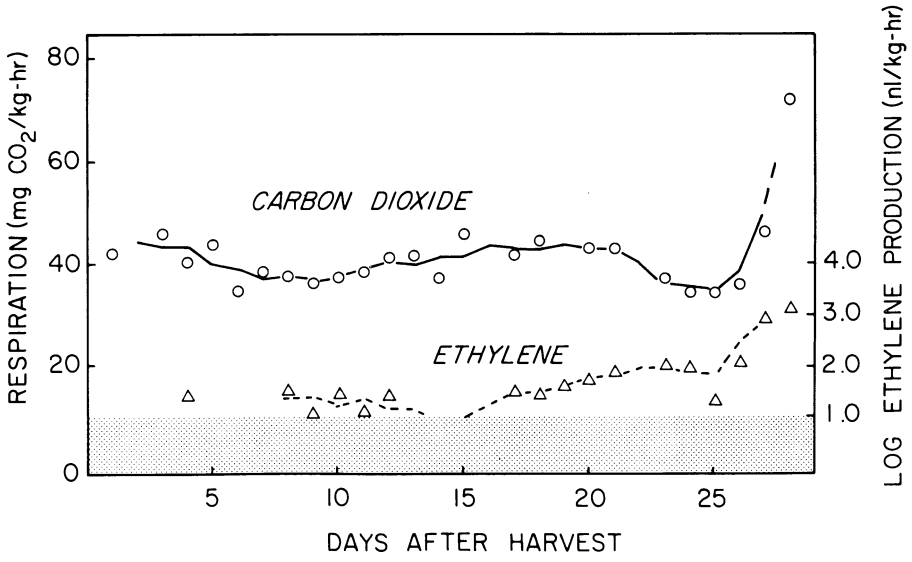

Fig. 2. Respiration and ethylene production from an individual fruit. For carbon dioxide production, the solid line is a 3-day moving average. For ethylene production, the dashed line is the logarithm of the 3-day moving average. The stippled area represents trace concentrations of ethylene – an identifiable gas chromatography peak too small for accurate measurement. This specimen was terminated with *Pestalotia* stem and decay.

morning the respiration and ethylene production rates were measured and the gas treatment was resumed. Some fruits were treated with a 10 min dip in 1000 ppm of (2-chloroethyl) phosphonic acid (ethephon). None of these treatments, even repeated gassing for 3 days, induced an appreciable rise in respiration or in ethylene production by the fruit.

From our evidence we conclude that the star apple is a nonclimacteric fruit. This may explain the apparent confusion in the meager literature. Popenoe (4) says, "The fruits are not good unless allowed to remain on the tree until fully ripe; if picked when immature they are astringent and contain a sticky white latex." On the other hand, Kennard and Winters (2) say, "The fruits do not normally drop and, therefore, must be picked and allowed to ripen off the tree." Wilcox and Hunn (6) imply that the fruit should be picked half-ripe and ripened off the tree. The best indication of horticultural fruit maturity, especially in the green type, is attainment of a highly glossy skin.

The principal research problem we encountered was the lack of a firm basis for actual individual fruit age. Stem end decay, leading to serious internal decay, was very common. With the help of Prof. Lina L. Ilag the most frequent pathogens were identified as species of *Pestalotia* and *Diplodia*. Occasionally fruits were infested with small larvae, usually only 1 per fruit, which was almost indetectable until frass appeared in the fruit jar.

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Guava Fruit Firm Rot Induced by Bruising[1]

W.H. Ko and R.K. Kunimoto[2]

Beaumont Agricultural Research Center, University of Hawaii, Hilo HI 96720

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Abstract. **Firm rot of guava (*Psidium guajava* L.) fruit usually started at the blossom end where affected tissue became water soaked but remained firm. The disorder, which advanced 7 to 40 mm in 24 hours, enhanced the development of fruit spot associated with *Guignardia musae* Racib. Guava fruit inoculated with diseased tissues did not develop firm rot, nor did hot water treatment prevent further development of the disorder. Healthy fruit struck against a bench in the laboratory or guava branches in the field developed firm rot at the blossom end but not at the stem end. Guava fruit firm rot, which is not caused by an infectious agent, appears to be triggered by wounding resulting from striking against hard objects.**

Guava fruit are used mainly for processing into puree and beverage juice in the state of Hawaii. The guava industry has expanded rapidly in recent years due to the greater market demand for these products. A new guava fruit rot was noticed on the island of Hawaii in 1974. Subsequently, the disorder was found in guava orchards every year, although the severity varied from year to year and seemed to be more severe during the dry seasons. The present study was initiated to determine the nature of this new disorder.

The disease usually started at the blossom end of the mature guava fruit on trees or on the ground. The affected area became water-soaked and expanded rapidly but tissue remained firm. Flesh of affected tissue also appeared water-soaked. This firm rot was different from mucor rot of guava fruit incited by *Mucor hiemalis* Wehmer (3) in that it did not give off a yeasty odor and diseased fruit were not covered with a yellowish fuzzy mass of fungal fruiting bodies and mycelia. The blackish fruit spot associated with *Guignardia musae* always developed within the affected area in the later stage of firm rot development.

Perimeters of advancing margins on recently diseased fruit on trees were marked with a felt marker to determine the expansion rate of firm rot. Distances between the marked margin and the new advancing marign were measured at 5 random points on each of 6 fruit after 24 hr. The expansion of guava firm rot was unusually fast at the beginning, about 23 mm in the first 24 hr, with

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[2]Professor and Research Associate, respectively, Department of Plant Pathology.

about half the initial rate thereafter.

Diseased fruit were surface-sterilized with 0.8% sodium hypochlorite solution for 5 min and rinsed with sterile distilled water. Pieces (ca. 6 x 4 x 2 mm) of rind and flesh (2 mm below the surface) tissues were removed with a sterile scalpel from the water-soaked areas, streaked separately on agar media, and placed on the edge of the plates. Media used were Bacto potato dextrose agar, Bacto nutrient agar and V-8 juice agar (20% V-8 juice, 0.02% $CaCO_3$); three and 12 plates were used for rind and flesh tissues, respectively. Plates were incubated at 24°C for 5 days. No microorganism grew from flesh of fruit with firm rot but a fungus closely resembling *Guignardia musae* (4) was consistently isolated from rind tissue. Twenty pieces of rind tissues from firm rot lesions of 10 guava fruit were plated on V-8 juice agar in another experiment, *G. musae* was isolated from all of the pieces.

Two holes (6 mm in diameter and 4 mm deep) were made at each of the polar ends of healthy mature green fruit to determine if firm rot of guava was caused by an infectious agent. Mycelia and spores of *G. musae* which was isolated in previous experiments, were obtained from a 10-day-old culture grown on Mycophil agar and about 0.04 ml of the fungal suspension was applied to each inoculation site. Inoculated fruit were placed in a large inflated polyethylene bag with 6 pin-size vent holes. Moist air was continuously pumped into the bag to prevent desiccation. The plastic bag was removed after incubation overnight at 24°C and fruit were further incubated for 5 days. Control fruit were treated similarly. Five fruit were used for each treatment and experiments were repeated twice. None of the inoculated fruit developed firm rot. Diseased tissues (about 4 x 4 mm) were also used to inoculate healthy guava fruit as described above, but again none of the inoculated fruit developed firm rot. Juice was extracted from diseased tissues, sterilized by passage through a polycarbonate membrance (CPR, 0.2 μm, 25 mm diameter; Nuclepore Corporation, Pleasanton, California 94566), and used to inoculate healthy guava fruit, as previously described, to determine if sterile liquid from diseased fruit could induce disease symptoms. All of the inoculated fruit remained healthy after 6 days of incubation.

Table. 1. Induction of guava firm rot by wounding.

	No. sites with firm rot[z]			
	Experiment I		Experiment II	
Position of wound	Wounded[y]	Non-wounded	Wounded[y]	Non-wounded
Stem end	0	0	0	0
Blossom end	16	0	10	0

[z]Total sites were 16 and 40 in Experiment I and II, respectively.
[y]Guava fruit were struck once on opposite polar ends against a laboratory bench.

Microorganisms were isolated only from rind tissues, hence a band (about 4 mm wide, 2 mm deep) of rind tissue about 2 mm beyond the advancing margin of firm rot was removed with a sterile scalpel from a diseased fruit. The furrow did not prevent the expansion of firm rot on any of 3 guava fruit tested, the advancing margin extending beyond the furrows within 24 hours.

Hot water treatment at 47.8 to 48.9°C for 20 min is effective in controlling early stage of papaya fruit diseases caused by *Colletotrichum gloeosporiodes* Penz., *Botryodiplodia theobromae* Patou., and *Fusarium solani* (Mart.) Sacc. (1, 2). Diseased guava fruit were submerged in water at 47.8 to 48.9°C for 30 min to determine if such treatment could stop the expansion of firm rot. Firm rot on all 6 guava fruit tested continued to expand after hot water treatment.

We observed that firm rot in guava fruit on the orchard floor always started from lesions caused by lava rocks when fruit fell. Apparently healthy mature green fruit were struck once against a laboratory bench close to the blossom or stem end and wounded areas marked with a felt marker. Striking force determined with a triple beam balance was about 400 to 600 g/cm^2. Non-wounded fruit marked at similar locations were used as controls. Fruit were observed after 24 hr at 24°C. All 8 wounded fruit in one experiment developed firm rot at the stylar end only (Table 1). One fourth of the wounded sites at the blossom end developed firm rot in another experiment. Neither lesions at the stem end nor non-wounded fruit developed firm rot. Guava fruit on trees were also similarly struck against branches and observed the next day. Five of 20 wounded fruit on trees developed firm rot at the stylar end but not at the stem end. All 20 non-wounded fruit used as controls remained healthy. It is, therefore, suggested that firm rot of guava fruit is induced by wounding resulting from striking against hard objects. Why certain guava fruit develop firm rot after being wounded is still unknown.

Guignardia musae was isolated consistently from blackish spots of guava fruit. However, the causal relationship between *G. musae* and fruit spot still has not been established. This type of fruit spot always developed within the perimeter of the affected areas in the later stage of firm rot development. No spots were observed on control fruit which did not develop firm rot. This indicates that firm rot also enhances development of fruit spot associated with *G. musae*.

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