# Guava Fruit Rot Caused by Rhizopus stolonifer in Hawaii

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#### ABSTRACT

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A fruit rot that was serious on guava on Kauai island was characterized by rapidly expanding water-soaked lesions, which were slightly sunken at the margins of the soft rot. Fruit was diseased in all maturity stages from mature green to fully ripe. *Rhizopus stonolifer* was the pathogen.

For years, guava (Psidium guajava L.) fruit for processing in Hawaii was collected almost entirely from wild plants. Guava gatherers did not put resources into maintaining the plants they harvested, so fruit rot losses were not considered in the cost of producing guava products. With expansion of the market for guava products and changing land use patterns, commercial plantings are rapidly replacing wild plants as a major fruit source, and fruit disease losses are an important factor in determining profitability. A soft rot affecting mature green to fully ripe fruit caused severe losses in one orchard on Kauai during the 1978 season (Fig. 1).

In early disease development the lesions appear oily and water-soaked. The lesion margin is distinct and the lesion is slightly sunken at the margin. The rapidly expanding lesion reduces the fruit flesh to a semisolid state in a few days. The epidermis generally remains intact. Aerial hyphae develop at the point of infection. Although development is not extensive, it rapidly extends over the lesion and covers it with a sparse white to gray mycelium. White sporangiophores and sporangia, which later turn black, develop where the aerial hyphae contact the surface. Sporangiophores and sporangia develop more densely at breaks that may occur in the epidermis of the affected area and at the point of infection. The presumed pathogen was invariably associated with this soft rot. Infected fruit remain attached to the tree until they are manually dislodged or fall in the course of natural maturation. As a result of this disease, yield of marketable fruit was significantly reduced in the field and in storage before processing. The cause of this disease is reported.

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## MATERIALS AND METHODS

Diseased fruits for isolation and healthy fruits for inoculation were surface-disinfected in 0.5% sodium hypochlorite for 1 min and blotted dry with sterile paper towels. To isolate the fungus tissue from near the advanced margin, lesions was placed on 2% water agar and incubated at 27 C overnight. Hyphal tips were transferred to 10% V-8 juice agar (VJA; 100 ml of V-8 juice, 0.5 g of CaCO<sub>3</sub>, and 20 g of agar per liter) for maintenance and propagation.

To determine if diseased tissue contained infectious agents, two 4-mm diameter wells about 1 cm deep were made in mature green test fruit, one near the stem end and the other near the blossom end. Disks of diseased tissue approximately the same diameter as the well and half as long as the well was deep were inserted into the wells. After being trimmed to accommodate the diseased tissue, the healthy fruit disk was reinserted into the fruit and the wound sealed with adhesive tape. Control fruit were treated similarly except that healthy rather than diseased tissue was inserted into the wells. Each treatment consisted of incubating five fruits for 24 hr in a sealed moist chamber at 27 C and then for 72 hr with the moist chamber lid ajar.



Fig. 1. Guava infected by Rhizopus stolonifer.

Subsequent inoculation tests used pure cultures of *Rhizopus stolonifer* (Fr.) Lind. isolated from diseased guavas and grown on VJA. Agar disks 4 mm in diameter were taken from the edges of the *R. stolonifer* colonies after 5 days' growth, and 30 test fruits were inoculated and incubated as described. The same number of control fruit were similarly inoculated with sterile VJA disks and incubated.

In addition, VJA disks, with and without R. stolonifer, were placed on the uninjured epidermis of five fruits each and incubated.

## RESULTS

All fruits inoculated with diseased tissue developed typical water-soaked lesions in 24-48 hr at both stem and blossom end. In 72-96 hr the lesions were considerably enlarged, involving 20-100% of the fruit. All control fruit remained sound. R. stolonifer was consistently isolated from the rotted fruit.

Of the healthy fruit inoculated with mycelia and sporangia of *R. stolonifer* on agar disks, 76% developed soft rot identical to that occurring naturally at both inoculation sites, but none of the control fruit rotted from the inoculation site. The fungus was reisolated from the advancing margin of lesions on inoculated fruit

All control and test fruit inoculated without wounding did not rot at the inoculation site.

#### **DISCUSSION**

This is the first report of R. stolonifer fruit rot of guava in Hawaii. Srivastava and Tandon (2) reported R. nigricans as the cause of a postharvest guava rot in India. They gave no indication that the disease occurred in the field. Kunimoto et al (1) reported on a soft rot in Hawaii caused by Mucor hiemalis Wehmer. Rhizopus rot is easily distinguished from Mucor rot in the field. Mucor-rotted fruits are covered with abundant yellow mycelia and sporangia, and Rhizopusrotted fruit show comparatively sparse aerial mycelium with dark gray to black sporangiophores and sporangia

Very favorable conditions are apparently required for an epidemic of Rhizopus rot. For example, the severely affected orchard is situated in a small valley with a rapidly flowing stream running through it. Air drainage is poor, resulting in high humidity and persistent dew well into the day. In orchards on

open sites where air can move freely, Rhizopus fruit rot is rarely or never found. Infection courts were probably provided by insects, because what appeared to be oviposition sites were almost always

associated with the disease. Fruit flies

inoculum on fallen fruit. These fruits were visited by a variety of insects, including fruit flies. The conditions for disease development and involvement of insects in the disease are being investigated.

were abundant in the orchard, as was

#### LITERATURE CITED

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