# Latent Infection in Avocado Fruit Due to Colletotrichum gloeosporioides

N. Binyamini and Mina Schiffmann-Nadel

Plant Pathologist, Division of Mycology and Plant Pathology, Department of Botany, University of Tel Aviv, Israel; and Associate Professor in Plant Pathology, Division of Fruit Storage Research, The Volcani Institute of Agricultural Research, Bet Dagan, and Faculty of Agriculture, Hebrew University, Rehovot, Israel, respectively.

Accepted for publication 30 December 1971.

ABSTRACT

The presence of latent fungal infection in avocado fruit was demonstrated by anatomical studies of artificially inoculated fruit that revealed appressoria of the fungus on the fruit while still on the tree and after picking until softening, and by the appearance, during softening, of decay spots on fruit that had been inoculated as much as 3 months prior to harvest, but did not show any signs of rotting at harvest. The germ tube of the fungal spores penetrated the wax layer, forming appressoria. During fruit softening, the appressoria germinated and hyphae penetrated the peel and pulp of fruit. Latent infection can take place at any stage of fruit development. Wounds need not be present for infection to take place.

Phytopathology 62:592-594.

*Additional key words: Persea americana.*

Avocado fruit is picked when still hard, and softening occurs after 7 to 15 days. As the fruit softens, the peel is covered by decay lesions of *Colletotrichum gloeosporioides* Penz., the main causal agent of avocado fruit rot in Israel (7). The large number of isolated spots which develop on uninjured fruit after harvesting suggest the occurrence of latent infection.

The mode of infection of fruit in the grove by the fungus is not fully understood, but some researchers (9, 12) contend that the fungus is able to penetrate through cracks and wounds only. Wardlaw et al. (11) isolated the fungus *C. gloeosporioides* from the peel of uninjured hard fruit, suggesting that the fungus may be present in the peel in latent form. The purpose of this study was to determine whether the rot in avocado fruit during softening is caused by latent infections occurring prior to harvest, and to establish the mode of penetration of the fungus into the peel.

MATERIALS AND METHODS.–Fruits of avocado (*Persea americana* Mill.) at different stages of development, from about 2 cm in length to fully grown, were inoculated with a spore suspension on the tree or after picking. Spores were taken from inoculated fruit; the density of spore suspension was about $10^4$/ml. To avoid natural infection, fruit on the tree selected for inoculation were covered with paper bags immediately after flowering, and kept covered until inoculation and thereafter until picking.

The presence of latent mycelium was demonstrated in two ways. One lot of fruit, inoculated in the grove, was picked and examined anatomically every few hours during the first 2 days and on alternate days for the first 30-day period, and thereafter 40, 60, and 90 days after inoculation. Immediately after harvest, slides prepared by hand and stained in cotton blue were examined anatomically. The fruit inoculated after picking was also examined at intervals until it began to soften.

A second lot of inoculated fruit was covered, left on the tree, and picked during the normal harvesting season. Immediately after harvest, the fruit was washed with water or alcohol to remove any spores, and stored until softening when these fruit were examined for rot. Covered but noninoculated fruit served as controls.

RESULTS.–Anatomical examination of fruit

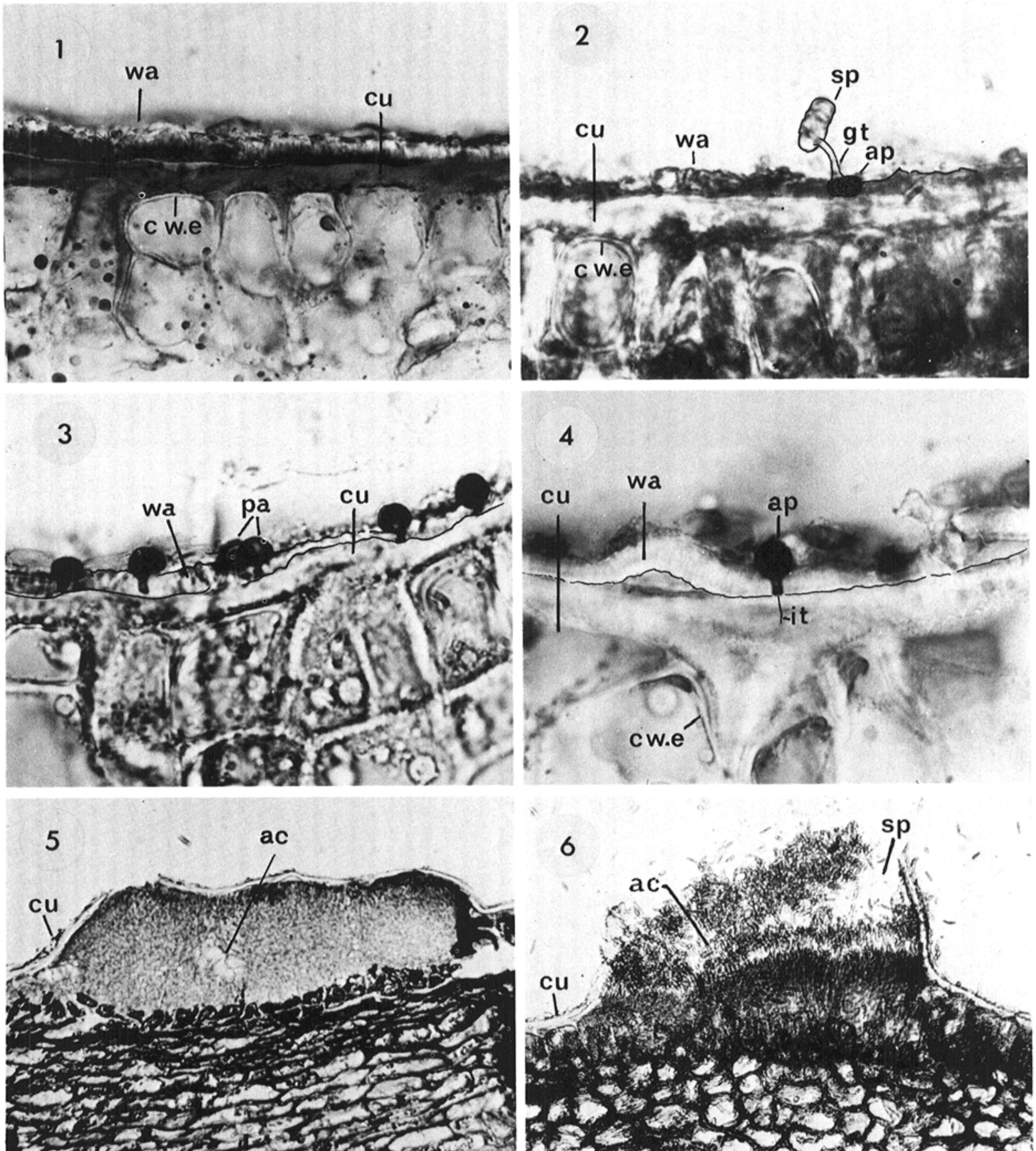


**Fig. 1-6. 1,2,3,4)** Stages of infection of *Colletotrichum gloeosporioides* in avocado fruit. **1)** Transverse section of the avocado peel (× 400). **2)** Spore germination and formation of an appressorium in the wax layer (× 630). **3)** Many appressoria in the wax layer (× 630). **4)** Germination of an appressorium (× 1,000). **5,6)** Development of the acervulus in avocado fruit. **5)** Acervulus under the cuticle (× 400). **6)** Open acervulus with spores (× 400). wa = wax; cu = cuticle; cwe = cell wall of the epidermis; sp = spore; gt = germ tube; ap = appressorium; it = infection tube; ac = acervulus.

inoculated on the tree at various stages of their development showed that spores germinated on the peel 1 day after inoculation. The germ tube entered the thick wax layer above the cuticle and formed dark appressoria (Fig. 1, 2, 3). This stage persisted throughout the time the fruit was on the tree and after picking, when the fruit remained hard. During softening, thin germ tubes developed from the appressoria and penetrated the cuticle and epidermis (Fig. 4). As softening progressed, hyphae invaded the peel and pulp, and dark spots began to appear. Acervuli appeared on the spots in the advanced stage of fruit softening (Fig. 5, 6). Appressoria seem to represent a latent stage of infection which persists until the fruit softens. A similar pattern was found in fruit inoculated after picking.

In sections cut by hand, appressoria were clearly visible in the large wax layer above the cuticle. In microtome sections, the wax layer was not present, probably because of the dehydration and embedding procedure used, and no appressoria were visible.

Fruit inoculated on the tree did not show any signs of rotting at harvest, but showed many spots of decay after softening, demonstrating that decay which develops on the soft fruit is a result of latent infection, from inoculation of fruit while still on the tree. Decay spots developed on fruit which had been inoculated at all stages of fruit development, and were similar to those that appear on naturally infected fruit. The number of spots on artificially inoculated fruit was higher than on noninoculated fruit. The control fruit did not show decay spots at softening.

DISCUSSION.—The existence of latent fungus infection in avocado fruit was demonstrated by anatomical studies of artificially inoculated fruit which revealed that latent infection persists in the form of appressoria, and by the appearance of large numbers of decay spots during softening of fruit artificially inoculated on the tree. Our findings show that there is no need for wounds to be present for infection to take place, as previously suggested (9, 12).

Latent fungal infection on immature fruit has been demonstrated by others. Edney (4) found latent infection in the form of appressoria in apples infected by *Gloeosporium perennans.* Latent infection of *Colletotrichum gloeosporioides* has been found in oranges (1). Evidence that, after a latent period in unripe fruit, fungi will develop in ripe fruit, has been presented by Wade (10) for *Sclerotinia fructicola* in apricot and by Meredith (6) for *Pyricularia grisea* in banana.

The reason for lack of development of *C. gloeosporioides* in the fruit while it is still on the tree may be that the hard fruit contain some inhibitors of fungal development. Wade (10) found some evidence that unripe apricot fruit contained substances fungistatic or fungicidal to *Sclerotinia fructicola.* Barnell & Barnell (2) and Chakravarty (3) noted that unripe banana fruit contained compounds, such as tannins, which inhibit the development of fungi. Similarly, Simmonds (8) suggested that the green peel of banana fruit contained substances toxic to *Colletotrichum* sp. Meredith (5) found that *Gloeosporium musarum* was able to infect unripe banana fruit only when inoculated through wounds. He stressed that nutrients available at wounds nullify the inhibition of fungal growth caused by substances present in unripe skins.

Hard avocado fruit may contain some inhibiting substances, as indicated by the findings that when fruit was inoculated even into deep wounds, the fungus did not develop in the fruit while still on the tree nor after picking, when still hard (M. Schiffmann-Nadel, *unpublished data*). Decay appeared only when softening took place, as in the case of fruit inoculated on the tree without wounds or in naturally infected fruit.

## LITERATURE CITED


1. ADAM, D. B., J. MC NEIL, B. M. HANSON-MERZ, D. F. MC CARTHY, & J. STOKES. 1949. The estimation of latent infection of oranges. Australian J. Sci. Res. B2:1-18.
2. BARNELL, H. R., & E. BARNELL. 1945. Studies in tropical fruits. Ann. Bot. Lond. N.S. 9:77-99.
3. CHAKRAVARTY, T. 1957. Anthracnose of banana (Gloeosporium musarum Cke and Massee) with special reference to latent infection in storage. Brit. Mycol. Soc. Trans. 40:337-345.
4. EDNEY, K. L. 1958. Observations on the infection of Cox's orange Peppin apples by Gloeosporium perennans Zeller and Childs. Ann. Appl. Biol. 46:622-629.
5. MEREDITH, D. S. 1960. Studies on Gloeosporium musarum Cke and Massee causing storage rots in Jamaican bananas. I. Anthracnose and its chemical control. Ann. Appl. Biol. 48:279-290.
6. MEREDITH, D. S. 1963. Pyricularia grisea (Cooke) Sacc. causing pitting disease of banana in Central America. Ann. Appl. Biol. 52:453-463.
7. SCHIFFMANN-NADEL, MINA, & F. S. LATTAR. 1960. Investigations on storage of avocado fruit, p. 1-16. Nat. Univ. Inst. Agr. Rep. No. 313 [Hebrew, with English summary].
8. SIMMONDS, J. H. 1963. Studies in the latent phase of Colletotrichum species causing ripe rots of tropical fruits. Queensland J. Agr. Sci. 20:373-424.
9. STEVENS, H. E., & R. B. PIPER. 1941. Avocado diseases in Florida. U.S. Dep. Agr. Circ. 582:1-46.
10. WADE, G. C. 1956. Investigations on brown rot of apricots caused by Sclerotinia fructicola (Wint.) Rehm. Australian J. Agr. Res. 7:516-524.
11. WARDLAW, C. W., R. E. D. BAKER, & S. H. CROWDY. 1939. Latent infection in tropical fruits. Trop. Agr. 16:275-276.
12. WOLFE, H. S., L. R. TOY, & A. L. STAHL. 1934. Avocado production in Florida. Fla. Agr. Exp. Sta. Bull. 272:1-96.