

WATERMELON RIND NECROSIS IN IMPERIAL VALLEY

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Rind Necrosis of Watermelon, (WRN), *Citrus vulgaris*, which has been attributed to bacteria by several workers, has been reported from several parts of the country. The infected rind exhibits small (0.3 to 1.0 cm) localized and water soaked necrotic spots; or larger grayish-brown, corky, quite hard necrotic spots. In extreme cases the necrosis may extend around the entire rind and into the edible part of the fruit (photo 1). Severely infected fruit is either unmarketable or is sold at low prices.

This disorder occurs in Imperial Valley and is not uncommon in other parts of the State. As much as 25 percent of the local fruit population may be affected in the field.

Bacteria were almost consistently associated with both discolored and symptomless rind of diseased and of healthy fruit. About 70 percent of the healthy fruits examined carried the bacteria; diseased and symptomless rind from diseased fruits harbored the bacteria in 79 percent and 36 percent of the cases, respectively. Bacteria were always isolated from vines of healthy or diseased fruit. The pathogenicity of vine-isolates, however, was not tested. On culture media these vine bacterial isolates looked identical with those from rind. The bacteria were small or longer rods and motile. They lost their motility within 3 to 4 days on PDPA medium at room temperature. The

Watermelon Rind Necrosis (WRN) is a perennial problem in Imperial Valley. As much as 25 percent of the fruit of some local fields may be mildly or severely affected. Last year about \$100,000 worth of fruit was lost to this disorder. Watermelon Rind Necrosis has been reported from Hawaii, Texas, and Florida. Bacteria, *Erwinia* sp., and fluorescent pseudomonads have been implicated by several workers as possible causal agents of rind necrosis.

Even though bacteria were most often associated with necrotic rind, the conclusion that Watermelon Rind Necrosis in Imperial Valley is due to bacteria cannot be made on the basis of our findings.

local isolates were identified as *Bacillus* sp.; Florida isolates were placed in the Entero group. Bacteria isolated from diseased rind in 1973 were identified as *Erwinia* sp. (Dr. M. N. Schroth, University of California, Plant Pathology Department, Berkeley, California, personal communication). The bacterium was re-isolated from artificially inoculated rind. At the time of these observations, the air temperature was 37°C. The temperature in the rind facing the ground and that in the rind facing the sun was 32°C and higher than 49°C, respectively.

Insect Carriers. Souer beetles and other unidentified small insects were "swept out" of vines in a field with high incidence of rind necrosis. The insects were crushed in sterile distilled water and PDPA plates were streaked with the extract. When watermelon rind was inoculated with a water suspension of the bacterial growth from insects, it developed a localized brown discoloration. The insect "bacterial growth" did not rot carrot slices.

Bacterial Isolations and Pathogenicity Tests

Small pieces (about 2 cu. cm) of watermelon rind taken from the advancing margins of necrotic spots were aseptically transferred to Potato-Dextrose-Peptone-Agar (PDPA) medium. The tissue was crushed with sterile forceps and the juice was smeared over the medium. The plates were incubated at 26.6 to 28.0 C. The same procedure was used to isolate microorganisms from symptomless rind parts of diseased or healthy fruits. Vines of diseased or healthy fruit were first washed in tap water, surface sterilized in 0.5 percent sodium hypochlorite for fifteen minutes, dissected cross-wise into small pieces, and aseptically transferred to PDPA medium.

About 24 hours later, a gray-whitish, viscous bacterial growth was obtained. This was suspended in sterile distilled water and streaked on PDPA. About 24 hours after incubation at 26.6 to 28.0 C, distinct grayish, circular, glistening

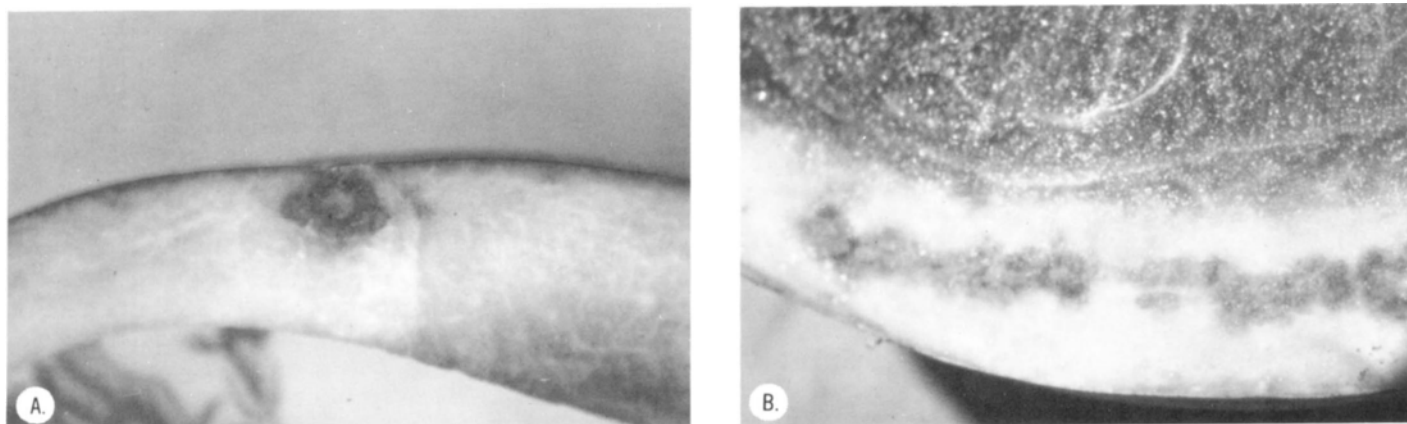


Photo 1. Watermelon Rind Necrosis in naturally infected fruit. A. Localized infection. B. Systemic infection Watermelon cultivar Peacock.

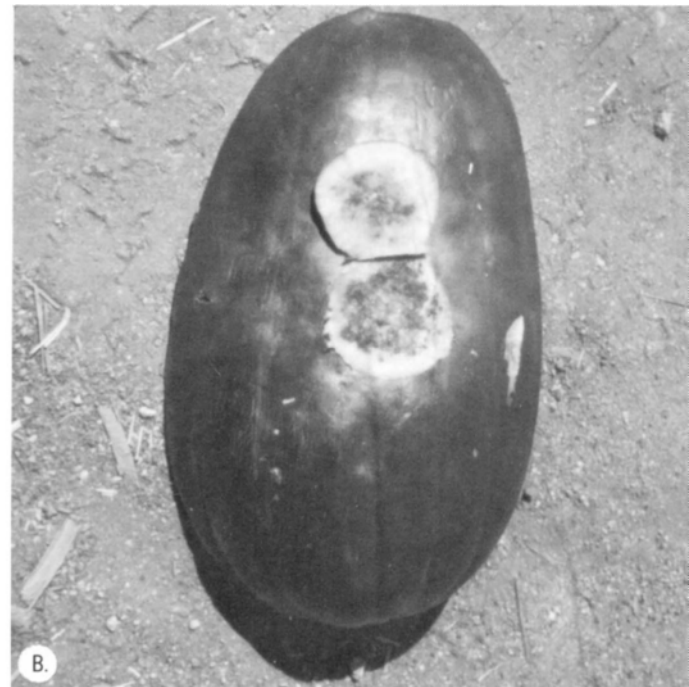


Photo 2. Watermelon Rind Necrosis. A. Artificial inoculation in the field. Variety Smokylee; bacterial isolate 10H. Note extensive rind discoloration. B. Natural infection. Variety Peacock.

colonies were formed on the medium. Growth from a single colony was then transferred to PDPA slants where it was maintained and used in pathogenicity tests. A bacterium from Florida, isolated from watermelon, designated 73-1, was similarly maintained and it was tested along with local bacterial isolates.

Twenty-four to thirty-six hour old bacterial growth on PDPA medium was suspended in sterile distilled water. This inoculum was immediately used to inoculate watermelon fruit at various developmental stages in the laboratory or in the field. The fruit to be inoculated was surface sterilized at the points of inoculation with either 70 percent methyl alcohol or 0.5 percent sodium hypochlorite. About 0.25 ml bacterial inoculum or sterile distilled water (ck) was introduced into the rind with a sterile hypodermic syringe. Carrot and non-cooked potato slices in petri dishes were also smeared with bacteria and kept at 26.6 to 28.0 C. The results of these tests were recorded 5 or 10 days after inoculation.

Results

Several isolates obtained from the necrotic area of locally raised watermelons incited a deep brown

discoloration in the inoculated rind. In field inoculations isolate 10H (*Bacillus* sp.) caused very extensive symptoms in watermelon cultivars, Peacock, Blue Ribbon, and Smokylee (photo 2). The same isolate caused moderate carrot rotting, and moderate gas production in both carrot and watermelon. The Florida isolate 73-1, on the other hand, induced only watersoaked or light brown and localized necrosis. In all cases, the symptoms were similar but not identical with those observed in natural infections. This was the case regardless of fruit age, variety, or inoculation site. The symptoms under field environment appeared to be more extensive than those in the laboratory. In similar laboratory tests in 1973, with *Erwinia* sp. which was isolated from diseased rind, similar results were obtained.

Discussion

Tomato, pineapple and cucumber fruit, stems of healthy pinto bean plants, bean seed, potato tubers, and many other plant tissues harbor bacteria and yet they may remain healthy or symptomless for life. The significance of this internal bacterial microflora in healthy tissues is largely unknown. The rind of local watermelon usually harbors bacteria. These bacteria,

when used in artificial inoculations, induced localized or extensive rind discoloration and necrosis. The necrosis which was induced artificially, however, was brown or brown-gray and soft; the necrosis observed in natural infections, though of the same discoloration, was hard and dry. Other workers have made similar observations. It was interesting that inoculations of *Bacillus* sp. and *Erwinia* sp. produced similar symptoms in the rind. Our failure to induce typical symptoms in artificial inoculations may be due to our inability to duplicate the environmental conditions required for typical symptom induction in the field.

As long as typical symptoms are not reproduced by artificial inoculations of bacteria, the real cause of Watermelon Rind Necrosis in Imperial Valley is an open question. We do feel, however, that the isolated bacteria play a role in this disorder, either initiating infection or accelerating the breakdown of rind tissue.

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