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Pathogenicity of *Pseudomonas gladioli* pv. *gladioli* on Rhizomatous Iris and Its Possible Role in Iris Scorch

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Scorch of rhizomatous iris, *Iris* spp., occurs throughout much of the United States (Black, 1984). It is characterized as a rust-colored browning that starts on the tips of the youngest leaves in the center of the fan. Leaf necrosis progresses downward on the leaves and outward on the fan until the entire fan is affected. Roots shrivel and decay. Root cortical tissue completely deteriorates, but the epidermis and stele remain intact. The rhizome appears unaffected (Wadkamper, 1972).

Because of the sporadic occurrence of scorch and the difficulty of artificially reproducing scorch symptoms, little is known about the etiology, epidemiology, or the pathogenicity of organism(s) involved in its development (Black, 1984). Attempts to identify a predominant causal organism or transmit scorch through inoculation with various fungi and bacteria recovered from scorched iris have failed to reproduce the disease (Wadkamper, 1972).

Bald (1971) implicated the bacterium *Pseudomonas gladioli* pv. *gladioli* (Severini) as the probable cause of iris scorch. He obtained evidence of pathogenicity by inoculating wounded iris tissue with cell suspensions of the *Pseudomonas* bacterium. However, symptoms were less severe than

those that occurred in the field under natural disease development. Rainio (1936) reported that *P. marginata*, synonymous with *P. g. pv. gladioli*, entered the iris leaf through wounds, where it caused a localized water-soaked wet rot but not the characteristic scorch symptoms.

Since the pathogenicity of *P. g. pv. gladioli* to rhizomatous iris is not clear, the present study was undertaken to determine if *P. g. pv. gladioli* could be recovered from scorched iris and to characterize the symptoms produced by inoculation of rhizomatous iris with *P. g. pv. gladioli*.

To determine if *P. g. pv.* was present in scorched iris, isolations were made from symptomatic root and leaf tissues. Excised tissue sections were surface sterilized 2 min in 0.5% sodium hypochlorite and rinsed with sterile distilled water. These were placed in a 125-ml Erlenmeyer flask with 10 ml sterile 12.5 mM PO₄ buffer, pH 7.1, containing 10 mM MgSO₄, and incubated overnight on a rotary shaker. Resulting suspensions were serially diluted in PO₄ buffer and plated onto nutrient broth yeast (NBY) agar medium (Vidaver, 1967). Plates were incubated 2 days at 25 to 27°C. We selected 26 bacterial colonies, based on colony morphology, for SDS-polyacrylamide gel electrophoresis (PAGE) comparison of whole cell polypeptide profiles with those of two reference strains of *P. g. pv. gladioli* (New Zealand Plant Disease Division Cultured Collection Strains 3950 and 3951) (Gwen and Jackman, 1982).

To examine pathogenicity of *P. g. pv. gladioli*, roots, rhizomes, crowns, and leaves of separate plants of 'Victoria Falls' were inoculated with suspensions of *P. g. pv. gladioli* Strains 3950 and 3951 at a concentration of 1×10^8 cfu/ml using a sterile 1.0-cm³ tuberculin syringe. Inoculated plants were placed in a humidity chamber, maintained at 95% to 100% relative humidity and 21 to 30°C for 1 week, after which they were moved to a greenhouse bench where symptom development was monitored. After 8 weeks,

P. g. pv. gladioli was reisolated from lesions and identified using SDS PAGE.

The attempts to recover and identify *P. g. pv. gladioli* from leaf and root tissues of scorched iris were unsuccessful. A comparison of the polypeptide band patterns of the 26 bacterial colonies with those of the reference *P. g. pv. gladioli* strains showed no evidence of *P. g. pv. gladioli* in the tissues examined. Furthermore, inoculation of healthy iris with *P. g. pv. gladioli* Strains 3950 and 3951 failed to produce scorching of inoculated plants. Inoculated roots and rhizomes were asymptomatic. Inoculated crowns sometimes showed signs of localized water soaking at the point of inoculation when some leaf tissue remained; but when leaf tissue was totally removed, inoculated crowns remained symptomless. Leaf inoculations produced irregular, translucent, water-soaked lesions that ranged from 2.4 to 5.6 cm long. The two *P. g. pv. gladioli* strains differed in their virulence with Strain 3950 producing significantly longer lesions than Strain 3951. These data were consistent with those reported by Rainio (1936) but did not confirm the findings of Bald (1971).

Under the conditions of this study, *P. g. pv. gladioli* was not identified among bacteria isolated from scorched iris nor were scorch symptoms induced when healthy iris were inoculated with two strains of *P. g. pv. gladioli*. The pathogenicity of *P. g. pv. gladioli*, previously reported to cause scorch (Bald, 1971), in scorch development remains unclear, as does the etiology of this iris disease.

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