BACTERIAL DISEASES OF TOMATO AND PEPPER

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Bacterial speck of tomato caused by *Pseudomonas tomato* and bacterial scab of pepper caused by *Xanthomonas vesicatoria* are widespread in commercial vegetable fields in Israel. Bacterial speck of young seedlings caused losses up to 75% of the total yield; the losses in plants infected later were at most 25%. Bacterial scab of plants with 4-6 true leaves caused losses up to 30%, but was minimal in fully mature plants. Infection was monitored by scanning electron and light microscopy. During incubation, pathogenic cells became located inside the stomata, in the sub-stomatal chamber and in the intercellular spaces between mesophyll cells. The primary infection sites were the vein areas in pepper and the stomata and trichomes in tomato. Extensive multiplication (10^6-10^7 cells/g leaf) was observed on the leaf surfaces, and masses of *P. tomato* cells were found in abraded leaves. Necrosis was first observed microscopically (in both diseases) 100-120 h after inoculation near the leaf veins (pepper) and at broken trichome bases (tomato). There was a sharp delineation between necrotic areas (specks, scabs) and apparently healthy tissue. Infection of sterilized or natural soil with *P. tomato* or *X. vesicatoria* at an inoculum concentration of 10^2 to 10^9 cells/ml inhibited germination of tomato and pepper seeds, respectively. Infested tomato plants which were symptomless during the growing season had 20-30% less foliage than uninoculated plants. Infested symptomless pepper plants had a 20-30% lower yield than uninoculated plants, but had a high endogenic population of *X. vesicatoria* (10^6 cells/g leaf). Necrotic activity of *P. tomato in vivo* was accompanied by the production of large quantities of ammonia in the diseased plants.

CELL INTERACTIONS BETWEEN *TRICHODERMA* SPP. AND *RHIZOCTONIA SOLANI* OR *SCLEROTIUM ROLFSII*

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Treatment of iris bulbs with conidia of *Trichoderma hamatum* in a gum arabic slurry protected the bulbs and seedlings from soilborne plant pathogens. Diseases caused by *Rhizoctonia solani* and *Sclerotium rolfsii* were reduced by 73% and 89%, respectively. Significant biological control of *R. solani* disease was also obtained by coating cotton seeds. The cell-cell interactions between the antagonist and the pathogenic fungi were studied by scanning and transmission electron microscopy, which revealed that *Trichoderma* hyphae attack their host by formation of appressoria-like bodies or by coiling around the pathogens. Host-parasite interaction sites were detected by photomicrographs using infrared film; infrared irradiation from these sites demonstrated high activity. The findings indiated that *Rhizoctonia* cell walls may have a lectin which binds type "O". Inhibition of this binding was obtained by flooding the mycelium with L-fucose. The possibility that *Rhizoctonia* is "recognized" by *Trichoderma* through lectins was considered.