Rhizoctonia Crown Rot and Cavity Spot of Muck-Grown Carrots

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ABSTRACT

A serious crown and root rot of muck-grown carrots in Wisconsin first appeared in the field when plants were about 5 weeks old. Isolations from crown and root lesions yielded isolates of *Fusarium* and *Rhizoctonia*. Greenhouse-grown carrots inoculated with the *Rhizoctonia* isolates developed typical crown rot and cavity spot lesions, whereas the *Fusarium* isolates were not pathogenic. Histological sections of lesions from field-grown carrots revealed abundant mycelium of *Rhizoctonia* adjacent to and within the tissue. Phytopathology 60:887-890.

Over the last 10 years there have been an increasing number of reports from several regions of Wisconsin of a crown rot and root-spotting decay on muck-grown carrots, *Daucus carota* L. Although the disorder has also been seen on carrots grown on muck soil freshly broken from its natural state, the disease was more prevalent on farms with a history of successive carrot cropping. About 1 month after seeding, carrots died in small, scattered patches. During the season, carrots on the periphery of the diseased areas became infected. The crowns of infected carrots were extensively decayed; later in the season, up to the time of harvest, carrots with dark sunken cavities at the sites of lateral root emergence were also found in the field (Fig. 1). The crown rot and root spotting were often found on the same carrots, though not necessarily so.

The crown rot resembles most closely a *Rhizoctonia* rot described by White (13). On the other hand, the dark sunken cavities on the carrot roots resemble the cavity spot disease described by Guba et al. (5), who attributed the condition to genetic and environmental factors. Maynard et al. (8, 9) also reported a cavity spot on the roots, and concluded that calcium deficiency induced either by low calcium in the nutrient solution or by a high total nutrient concentration brought on the condition. Root lesions on carrots superficially resembling the carrot rot seen in Wisconsin have been incited by *Phytomonas carotae* (1), *Stemphylium radicinum*, or *Alternaria porri* (4).

In view of the severity of the disease in Wisconsin and its importance to both farmers and processors, a study was begun to ascertain the cause of the disorder and to establish relationships with similar diseases reported in the literature. Results of greenhouse inoculations with *Rhizoctonia* isolates and histological examinations of tissues of carrots infected in the field are reported in this paper.

MATERIALS AND METHODS.—Isolation from crown rot and cavity spot lesions yielded predominantly *Fusarium* and *Rhizoctonia* spp.; however, preliminary tests showed that only *Rhizoctonia* was pathogenic to carrots. Thirty-three isolates of *Rhizoctonia* were obtained from infected carrots, and six from soil surrounding diseased carrots. Since all isolates had similar cultural characteristics and were pathogenic on carrot root discs, 2 isolates were selected for further study. Isolate R-1 was obtained by directly plating onto agar medium mycelium growing around an infected carrot in the field. Isolate R-3 was recovered from a carrot root lesion.

The incorporation of fragmented *Rhizoctonia* mycelium that had been isolated from diseased carrots into the soil prior to seeding resulted in heavy pre- and postemergence damping-off. Carrots were therefore grown for 16 days in noninfested muck soil until they were no longer susceptible to the damping-off phases of *Rhizoctonia* infection. Chantenay carrots were grown in 1-gal paper "Sealrite" containers (6.75 × 7.25 inches, Phillips Petroleum Co.) in which the bottom had been replaced by a single layer of cheesecloth. The containers were filled with muck soil collected from noninfested areas of the carrot field; approximately 40 seeds were planted in each container. Ten days after seeding, seedlings were thinned to 10/container. The greenhouse temperature was maintained at approximately 24 C, and the carrots were irrigated with Hoagland's solution at weekly intervals. Supplementary illumination to provide a 16-hr photoperiod was furnished by Sylvania 150-w light bulbs suspended 60 cm above the pots at 30-cm intervals.

Inocula of the two *Rhizoctonia* isolates, R-1 and R-3, were prepared by growing them on cornmeal-sand (CM-sand) medium for 26 days, following the procedure of Riker & Riker (12). Sterilized 8-inch clay pots were filled to within 5 cm of the rim with Ottawa silica sand. Sixteen days after seeding, when the tap roots were just emerging through the cheesecloth, the paper containers were placed on top of 200 g of inoculum layered on the sand in the clay pots (Fig. 2). Controls were placed on top of 200 g autoclaved CM-sand. The three treatments were replicated five times and arranged on the greenhouse bench in a randomized block design.

Counts of affected carrots were made 19, 26, 33, and 42 days after inoculation. The experiment was harvested after 43 days, and the number of dead carrots was recorded. The number of surviving carrots showing symptoms of crown rot and cavity spot was noted.

Disease development was studied by histological examination of infected and noninfected tissue of carrots collected in the field at several intervals during the growing season. Tissue samples were fixed in formalin-alcohol-acetic acid (FAA), dehydrated through the

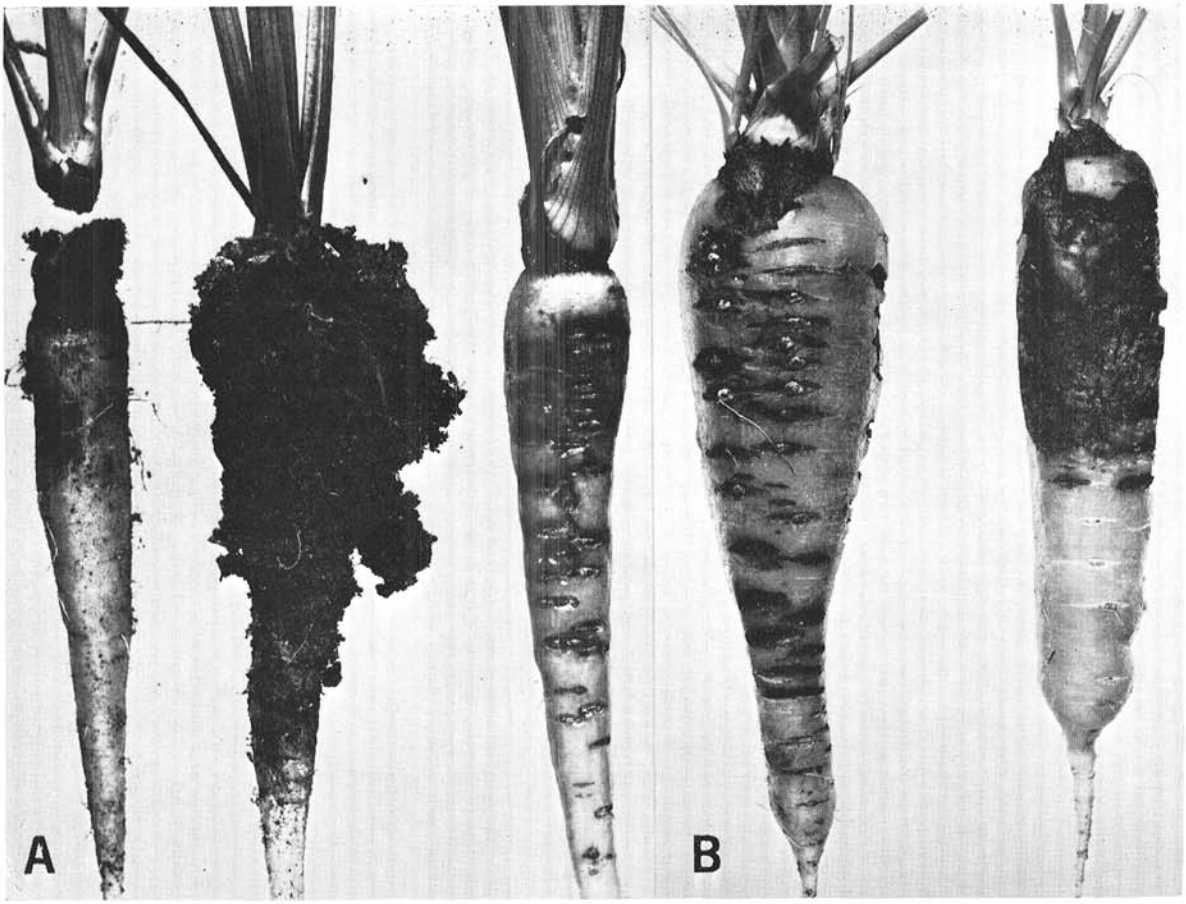

Fig. 1. A) Twelve-week-old Nantes carrots collected in the field, showing severe crown rot and cavity spot on the roots. Note the soil intertwined with mycelium adhering to the carrot. **B)** Nineteen-week-old Nantes carrots showing crown rot and cavity spot at the time of harvest.

standard tertiary butyl alcohol series, and embedded in paraffin wax for sectioning (7). Ten-μ sections were stained with Pianeze III B (6), which differentiates fungal mycelium from the host tissue.

RESULTS.—*Inoculation experiments.*—The first symptoms appeared on carrots in containers placed on R-1 inoculum about 19 days after inoculation. Initially the cotyledons wilted. This was followed by necrosis of the cotyledon commencing at the tip. Frequently *Rhizoctonia* encircled the carrots in a band about 1 mm wide below the insertion of the cotyledons. The appearance of this mycelium preceded the wilting of the cotyledons. About 1 week after the symptoms had appeared on the cotyledons, leaves of infected carrots began to wilt, and within 2 weeks many carrots had died. Carrots inoculated with isolate R-1 developed primary symptoms about 3 days earlier than those inoculated with isolate R-3. By 42 days, 52% of the carrots inoculated with isolates R-1 and R-3 had died.

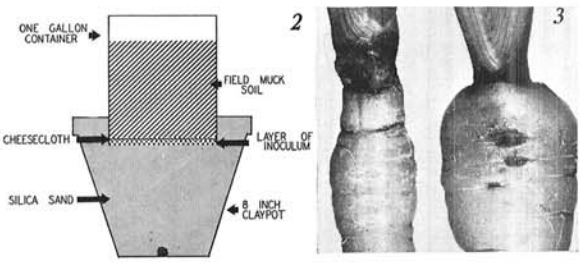

Fig. 2-3. 2) Diagram of the system used to inoculate carrots with *Rhizoctonia* sp. **3)** Eight-week-old greenhouse-grown Chantenay carrots inoculated with *Rhizoctonia* sp., showing cavity spot and crown rot.

Upon examination of the surviving carrots 43 days after inoculation, the incidence of crown rot was 100% for isolate R-1 and 87.5% for isolate R-3. Cavity spot occurred on 33.3% and 62.5% of the R-1- and R-3-inoculated carrots, respectively.

Crown rot appeared as a dark brown, dry decay extending as a band about 1 cm wide around the crown (Fig. 3). Cavity spot appeared as dry, sunken, dark-brown lesions at the sites of lateral root emergence (Fig. 1, 3), and were usually more numerous on the upper portion of the carrot root.

Field observations.—Crown rot was first located in the field about 4 weeks after seeding as small pockets of dying carrots. The outermost whorls of leaves wilted first. Carrots were often seen with a few inner turgid

leaves surrounded by whorls of wilted or dead leaves. A ring of *Rhizoctonia* mycelium 1-2 cm in width was frequently observed on the soil surface around decayed crowns.

Cavity spot was first observed in the field about 9 weeks after planting. Initially, tissue at the base of the lateral roots became discolored. The discoloration extended laterally following the horizontal groove. Degeneration of the tissue resulted in the formation of the laterally elongated lens-shaped cavities typical of the disorder. Another characteristic feature was the mass of soil, intertwined with mycelium, adhering to infected carrots (Fig. 1).

Transverse sections of decayed crowns from 5-week-old Nantes field carrots showed mycelium penetrating the cortex extensively, but invasion did not proceed beyond the cambial layer. Longitudinal sections revealed mycelium penetrating the cortex just below the insertion of the cotyledons and also penetrating the base of the cotyledon. Cortical invasion by the fungus was extensive to a distance of about 2 mm below the attachment of the cotyledons.

Transverse sections through cavity spot lesions on 12-week-old Nantes field carrots revealed that mycelium had penetrated the horizontal groove in V-shaped fashion, tapering inward to a depth of 1-2 mm (Fig. 4). Hyphae penetrated the vascular tissue of the lateral root. Collapse of the tissue adjacent to the xylem vessels of the lateral root occurred closely behind the invading hyphae (Fig. 4). Masses of hyphae were adjacent to the outermost layer of cells.

DISCUSSION.—These studies have confirmed that *Rhizoctonia* sp. induces the crown rot and cavity spot of carrots grown on Wisconsin muck farms. The data from greenhouse studies, however, indicate that upon inoculation the occurrence of cavity spot is relatively infrequent and erratic, whereas crown rot occurs consistently.

The development of cavity spot is associated with the ontogeny of the horizontal groove. Studies on the ontogeny of the storage organ of *Daucus carota* L. (3) show that enlargement of the stele through secondary growth results in sloughing of the cortex. This occurs after the 5th week from seeding. Deterioration of the cortex proceeds towards the root apex. A periderm bearing horizontal grooves at the base of each lateral root covers the surface of the older storage organ (3). The site of invasion appears to be the horizontal grooves, since these areas were often discolored while the lateral roots showed no signs of infection.

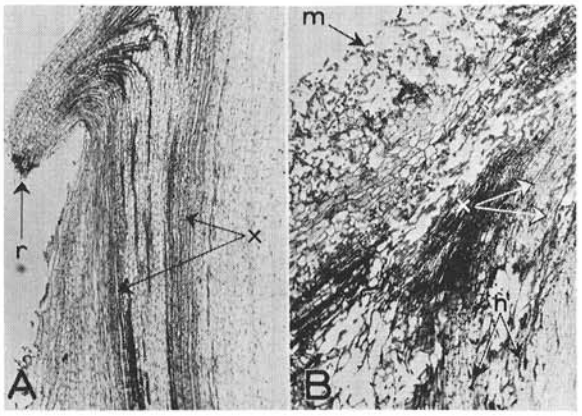

Fig. 4. Transverse sections through the horizontal groove of **A)** healthy and **B)** cavity-spotted 12-week-old Nantes carrots collected in the field (×80); h = hyphae penetrating vascular tissue; m = mass of mycelium adjacent to horizontal groove; r = lateral root; x = xylem vessels of lateral root.

Rader (10) described a new species of *Rhizoctonia, R. carotae,* which incited what he termed "crater rot" of stored carrots. No visible symptoms were evident at the time of harvest, however, and the symptoms appeared only after 1-2 months in cold storage. Rader postulated that incipient infections occurred in the field, and that "the spotted appearance of the lesions on the roots is suggestive of penetration through areas left by dead secondary rootlets". The same organism was reported by Ramsey & Smith (11) on stored carrots in Illinois. In common with our studies, Rader (10) reported that *R. carotae* induced a firm rot, and that "the characteristic pitting found commonly associated with the disease in storages has been produced in the laboratory with difficulty". Clamp connections and a mean hyphal diameter of 4.1 μ were key characters for *R. carotae,* whereas clamp connections were rare in our isolates, which had a mean hyphal diam of 8.2 μ.

During the crown rot phase, the restriction of the fungus in the hypocotyl to the cortical tissues is similar to the *Rhizoctonia* infection of bean described by Christou (2). Hyphal growth in the cortex of bean stems ceased in a zone one to four layers outside the endodermis.

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