Stunting of Soybeans by Pythium debaryanum

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ABSTRACT

Pythium debaryanum caused unusual symptoms on soybeans. The fungus incited dry, necrotic lesions on the cotyledons, death of the apical meristem, development of axillary shoots at the cotyledonary node, and a swelling of the hypocotyl. Infected plants of susceptible cultivars were severely stunted, and death of seedlings often resulted. This resulted in a 40% reduction in stand. The stand was increased when cyano-(methyl-mercuri) guanidine was applied as a seed treatment. Plant height was a reliable measure of disease resistance. Mandarin (Ottawa) was the most resistant, and Ontario and Manchu T3 were the most susceptible of 22 soybean cultivars tested. Phytopathology 60:961-963.

Pythium species, most notably *Pythium debaryanum* Hesse and *P. ultimum* Trow, have been reported to cause several disorders of soybeans including seed decay (1), wet-rot lesions on the stems and roots (1, 3, 7, 9), brown discoloration of stem lesions (3, 5), and pre- and postemergence damping-off (1, 5, 6). In 1966, soybean plants in the greenhouse and field displayed symptoms that were not typical of a *Pythium* infection. Severely stunted soybean seedlings with lesions on cotyledons were obtained from samples received from several locations in Iowa through the Plant Disease Clinic at Iowa State Univ. We investigated the cause of the disease, variation in cultivar response to the disease, and possible control measures.

MATERIALS AND METHODS.—We isolated from cotyledonary lesions, swollen hypocotyls, and roots. The *Pythium* isolate obtained was identified by means of Middleton's monograph (8) and tested in the greenhouse for pathogenicity.

A test for cultivar response to the *Pythium* isolate was made with 22 cultivars. Each cultivar treatment was replicated four times with 10 seeds used/pot. All pots and soil used were steam-sterilized twice prior to use. Inoculation procedures consisted of (i) placing a 5-day-old 9-cm disc of potato-dextrose agar plate culture in 10-cm clay pots three-fourths full of soil; or (ii) adding 10 ml of mycelial suspension to soil in 10-cm clay pots. Before planting, an additional cm of soil was added to pots after inoculum was applied.

For studies on control, Ford, Amsoy, and Corsoy cultivars of soybeans were planted in a naturally infested field near Cantril, Iowa. The seed was treated with cyano-(methyl-mercuri) guanidine, *p*-dimethylaminobenzenediazo sodium sulphonate, or chloroneb (1,4-dichloro-2,5-dimethoxybenzene) at rates of 2, 4, or 6 fluid oz/bu, respectively. The experimental design was a split plot, and all treatments were replicated four times. Each replicate consisted of a row 10 ft long with 100 seeds sown/row; 8 ft of each row was harvested for yield.

RESULTS.—*Symptoms*.—The disease first became evident in fields when large vacant areas appeared in the rows early in the spring. Individual soybean plants had small, dark, necrotic lesions on the cotyledons, swelling of the hypocotyl, and severe stunting of the epicotyl.

Cotyledonary lesions, the most prominent symptom observed, were small and dry, and occurred at random on the surface of cotyledons (Fig. 1). They appeared on either side of the cotyledon, although they were more predominant on the upper surface under field conditions. In the field, lesions were more numerous on cotyledons of seedlings that did not emerge. Lesions were small black sunken spots against a chlorotic background and sometimes coalesced, producing larger lesions. They never appeared water-soaked, and there was no correlation between lesion number and stunting.

Severe stunting of the apical meristem occurred in the field, and symptoms could be reproduced by growing plants in field soil under greenhouse conditions (Fig. 2). In severe cases, the apical meristem was killed and two axillary buds developed at the cotyledonary node 2 to 3 weeks later. Stems from these axillary buds grew more slowly; internodes were shorter; and the area of the trifoliolate leaves was approximately ¼ that of normal trifoliolate leaves of healthy plants.

Swollen hypocotyls were commonly observed and were usually associated with severe stunting of the apical meristem. The enlarged hypocotyl was approximately two to three times the diam of a normal hypocotyl, and was free of lesions or any discoloration at or near ground level. *Pythium debaryanum* was repeatedly isolated from the cotyledonary lesions and swollen hypocotyls. When we inoculated soybeans with a culture at the time of planting in the field or in the greenhouse, symptoms produced were the same as those originally observed in previous field and greenhouse infections. No symptoms appeared if the plants were exposed to *P. debaryanum* after emergence.

Response of cultivars to infection.—Mean plant height was an excellent measure of plant resistance to *P. debaryanum*. Cultivars differed greatly in height after 3 weeks of growth; infection of susceptible cultivars was readily expressed by stunting of the apical meristem. Mandarin (Ottawa), one of the most resistant cultivars, had no reduction in height in the presence of *P. debaryanum*. Lesions were present on the cotyledons of Mandarin (Ottawa), but none of the apical

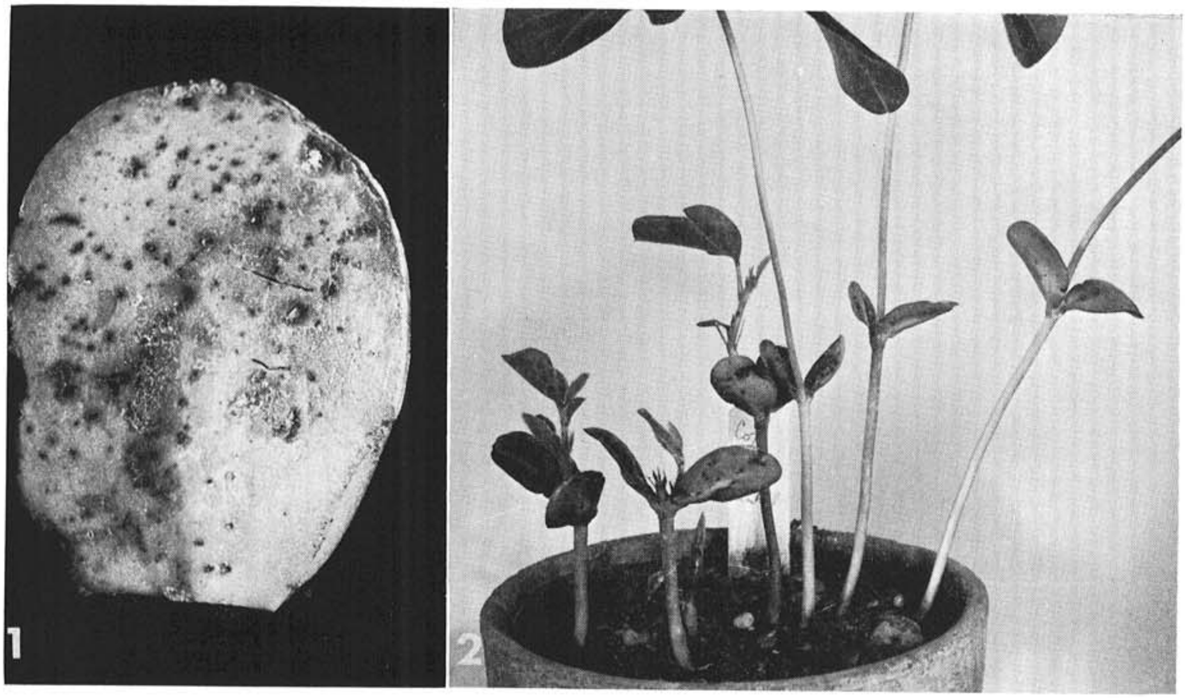

Fig. 1-2. **1)** Small, dry lesions on the lower surface of a soybean cotyledon infected by *Pythium debaryanum*. **2)** Severe stunting of soybeans in greenhouse soil containing *Pythium*. Average height of the three noninfected plants (right) was 23 cm compared to 8 cm, the height of 3 infected plants (left).

meristems was killed. Kingwa was the only resistant cultivar that did not have any lesions on the cotyledons. It was also the only other cultivar in which there were no apical meristems killed. Patoka, Kent, Perry, Kanro, Corsoy, and Adelphia cultivars were also resistant. Apical meristems were killed in a small percentage of these plants. Cultivars Hawkeye and Lindarin 63 had moderate resistance, with approximately 10% reduction in height. All susceptible cultivars, on the other hand, were short. Ontario, one of the most susceptible, had a 61% reduction in height with up to 45% of the apical meristems being killed. Other very susceptible cultivars included Mendota, Harosoy, Amsoy, and Goldsoy with height reductions of 38, 30, 40, and 29% respectively. Stands of the very susceptible cultivars Mendota, Harosoy, Amsoy, Goldsoy, and Ontario were poor, with reductions of 36, 30, 32, 30, and 31% respectively.

Seed treatments.—In an infested field, stand was reduced 40% in susceptible varieties where no control measures were applied. The cyano-(methyl-mercuri) guanidine treatment significantly increased stand of Ford (susceptible), Amsoy (very susceptible), and Corsoy (resistant) by 12% (at the 5% level of significance) in this experiment. Chloroneb (1,4-dichloro-2, 5-dimethoxybenzene) had no effect on stand, and *p*-dimethyl-aminobenzenediazo sodium sulphonate decreased the stand by 10%, apparently due to phytotoxicity. There was no significant difference at the 5% level of significance in yield among treatments.

Discussion.—Wet-rot symptoms and postemergence damping-off, previously described by Lehman (5) and McLaughlin (6) for *P. debaryanum* on soybeans, were not observed in our studies. Marked differences were also evident when we compared our culture of *P. debaryanum* with a culture of *P. ultimum* described from Minnesota (1). Both caused infection of soybean cotyledons; however, *P. ultimum* caused a complete and rapid rot of the seedling, whereas wet-rot symptoms were absent with our *P. debaryanum* isolate. The difference in activity could possibly be explained by enzyme capacity, as Damle (2) observed that there was a parallelism between the capacity of a *Pythium* species to produce pectinase enzyme and ability to parasitize potato and lettuce tissue.

The stand in several field plots was reduced by the *P. debaryanum* attack, but a decrease in yield was not observed. This may be explained on the basis that field plots were hand-weeded. Also, soybean seedlings have the ability to produce additional stem branches that compensate in part for the removal or loss of adjacent plants. Kalton et al. (4) found that removal of 25 or 50% of the stand in the seedling stage did not cause an appreciable decrease in yield. Yield loss and compensation or filling in of vacant areas might be expected in fields containing a poor stand, especially where weeds, providing competition, fill vacant areas where soybeans fail to grow.

LITERATURE CITED

1. Brown, G. E., & B. W. Kennedy. 1965. Pythium preemergence damping-off of soybean in Minnesota. Plant Dis. Reptr. 49:646-647.
2. Damle, V. P. 1952. Enzymatic study of certain parasitic fungi. J. Indian Bot. Soc. 31:13-55.
3. Hildebrand, A. A., & L. W. Koch. 1952. Observations

on a root and stem rot of soybeans new to Ontario, caused by *Pythium ultimum* Trow. Sci. Agr. 32:574-580.
4. Kalton, R. R., C. R. Weber, & J. C. Eldredge. 1949. The effect of injury stimulating hail damage to soybeans. Iowa State Agr. Exp. Sta. Res. Bull. 359:775-780.
5. Lehman, S. G., & F. A. Wolf. 1926. Pythium root rot of soybean. J. of Agr. Res. 33:375-380.
6. McLaughlin, J. H. 1946. Vegetable seed treatment for Oklahoma. Okla. Agr. Exp. Sta. Bull. 293.
7. Melhus, I. E., J. N. Martin, & H. C. Murphy. 1944. The influence of pythiaceous and other fungi on seedling stands of legumes and other crops. Iowa Agr. Exp. Sta. Rep. Agr. Res. year ending June 30, 1944, Part 1: p. 157.
8. Middleton, J. T. 1943. The taxonomy, host range and geographic distribution of the genus Pythium. Torrey Bot. Club Mem. 20. 171 p.
9. Schmitthenner, A. F. 1964. Fungi associated with root necrosis of *Phytophthora*-resistant soybeans. Phytopathology 54:906 (Abstr.).