Conquering Phytophthora Root Rot with Resistant Alfalfa Cultivars

Fig. 1. Phytophthora root rot of alfalfa (left) from the field and (right) from the growth chamber. Lesions occur at various depths and turn from brownish yellow to dark brown or black.

Alfalfa, the most important forage crop in the United States, has been frequently described as not tolerant of wet soils. Almost 70 years ago, Wing (11) described a root rot of alfalfa that he attributed to wet soils or a high water table. He advised farmers to avoid wet soils or to tile-drain wet areas of fields for alfalfa production. Erwin (2,3) described this root rot disease in 1965 and identified the causal agent as a species of Phytophthora that requires free water for infection. Subsequently, Phytophthora root rot (PRR) was reported to be an important production factor in most alfalfa-growing areas of the United States, Canada, and Australia. It has also been reported from Argentina and Japan.

The Causal Agent and Its Effects

The pathogen, Phytophthora megasperma Drechs. f. sp. medicaginis (7), is a "water mold" fungus that produces oospores in infected host tissue. The oospores can remain viable in the soil for long periods and eventually germinate to form mycelia or sporangia. Zoospores liberated from the sporangia in the presence of free water are attracted to the root tips. The zoospores encyst, germinate, and penetrate the cells in the zone of cell extension, and root rot is thus initiated. The taproots are infected either by the mycelia advancing from the lateral roots or by the spongy phellem at the base of fine lateral roots (9). Root nodule infection has also been reported (6).

A thin stand is usually the first indication of PRR. If the taproots of surviving plants in an affected area have been rotted off at various depths (Fig. 1), the most likely problem is PRR.

PRR can become a serious disease problem not only in perpetually wet soils with a high water table but also in soil types with poor internal drainage because of puddled soil or an impervious layer near the surface. When soils remain excessively wet, as during extended...
periods of rainfall or after overirrigation, conditions are favorable for PRR. Established stands may be destroyed during prolonged wet periods. If rainfall is excessive after sowing, stand establishment may fail because of seed and seedling rot. Affected areas range from a few square meters to entire fields.

PRR attacks the small roots first, then the taproot. Active lesions turn from brownish yellow to dark brown or black, often with a yellow margin (Fig. 1). When the entire taproot rots, the foliage turns yellow or reddish and the plant dies. Rot stops advancing when excess soil moisture is eliminated. Branch roots may form if some of the taproot remains undamaged and the soil stays moist, and the plant can recover. Such plants usually have shallow root systems, however, and are susceptible to drought. Shallow-rooted plants can produce abundant forage when soil moisture is adequate, but production is critically reduced if rainfall is insufficient, since the plants cannot utilize deep subsoil moisture.

The Search Is Started for Resistant Plants

Adequate soil drainage of all problem areas is usually neither practical nor possible. After I identified PRR as a problem in Minnesota in 1965, the need for resistant cultivars became evident. I initiated research to determine if resistant plants could be identified and screening procedures developed.

In 1969, D. K. Barnes, a research geneticist, joined the alfalfa research program at Minnesota, and we developed a method for screening large populations of seedlings in either the greenhouse or the growth chamber (4). Steamed sand in watertight tanks was infested with laboratory cultures of *P. megasperma* f. sp. *medicaginis*. Alfalfa seed was sown in the sand and watered sparingly to initiate germination and promote normal growth for 3–4 weeks. Then the drain holes were stoppered and tap water was added daily to raise the water level to the surface. After 3–4 weeks of saturation, the plants were lifted and scored for disease severity. The method proved effective for selecting PRR-resistant plants, but greenhouse-grown plants could not be accurately evaluated for vigor and other desirable agronomic characteristics. To screen large populations in the field, we established a field plot to simulate the conditions usually associated with naturally occurring PRR.

On the Experiment Station at St. Paul, MN, an area with no surface drainage but good internal drainage was selected as a test site. The soil surface was leveled and *P. megasperma*-infested soil, retained from greenhouse experiments, was spread over the surface before seedbed preparation. A sprinkling system was used to maintain the soil moisture at or
near saturation. A PRR nursery with about 80,000 plants has been grown at this location each year since 1968 (Fig. 2) (4).

How the PRR Nursery Operates
The seed is sown in rows spaced 30 cm apart in early May. The plots are irrigated first when the plants are well established, about 4 weeks after sowing. Water is applied twice daily to maintain the soil moisture at or near saturation for about 2 weeks. Sprinkling is then withheld for about 2 weeks. During this period, the plots are clipped and weeded and field notes are taken. Two additional wet and dry periods are completed during the growing season.

The plants are dug in September (Fig. 3). The roots on each plant are examined and classified according to disease severity. Plants having the least disease are selected and transplanted into pots in the greenhouse. The plants are grouped into populations of at least 150 according to genetic origin, relative winterhardiness, and other agronomic traits. The plants in each population are intercrossed by hand pollination without emasculation. The seed produced on all plants within a population is bulked. Seed from the intercrossed populations is sown in the PRR nursery the next year and evaluated for average disease severity and percentage of resistant plants. Usually two or three cycles of phenotypic recurrent selection are needed for a population to reach the desired level of resistance.

The Resistant Populations
Essentially all alfalfa cultivars from North America and many cultivars from other parts of the world have been evaluated for resistance in the PRR nursery. Most cultivars contain only a few resistant plants. The percentage of resistant plants in alfalfa cultivars not intentionally selected for resistance to *Phytophthora* has ranged from 0 to about 16. The one exception in North America has been the cultivar Lahonton, developed in Reno, NV, for resistance to the stem nematode (*Ditylenchus dipsaci*) and bacterial wilt (*Corynebacterium insidiosum*); about 30% of Lahonton plants are resistant to PRR.

The first experimental alfalfas developed for resistance to PRR were two nonhardy germ plasm lines, UC 38 and UC 47, released by the University of California in 1969 (8). According to the Minnesota evaluation, about 23% of the plants were resistant to PRR.

Four populations were developed simultaneously for resistance to PRR in Minnesota. MnP-A2 was developed from Vernal-type hardy alfalfas and released as the cultivar Agate in 1973 (1). MnP-B1 was developed from plants selected from 29 moderately hardy and hardy alfalfa cultivars, and MnP-D1 was developed from plants from 14 nonhardy cultivars. These two populations were released as PRR-resistant germ plasm lines in 1972 (5). The fourth population, MnP-C2, was developed with selections from Lahonton plants. The relative percentages of resistant plants for Agate, MnP-B1, MnP-D1, and MnP-C2 were 43, 34, 37, and 35, respectively.

The four selected populations and four nonselected cultivars were compared for forage and root yields when grown under conditions favorable for PRR (Fig. 4). The yields of the PRR-resistant selections were nearly double those of the nonselected cultivars. Lahonton is moderately resistant to PRR. A level of resistance similar to that in Agate (Fig. 5) appears satisfactory for most situations under Minnesota conditions.

Methods for selecting and screening for resistance to PRR were rapidly adopted by other plant breeding programs. Plant breeders in private seed companies have used the selection methods together with released resistant germ plasm and resistant plants from adapted cultivars to develop PRR-resistant alfalfas. By December 1979, the National Certified Alfalfa Variety Review Board had approved for certifica-

![Fig. 4. Forage and root yields of four selected lines (Agate, MnP-B1, MnP-D1, MnP-C2) and four cultivars not selected for resistance (Lahonton, Ranger, Saranac, Vernal) grown under conditions favorable for Phytophthora root rot. Yields are the total of three harvests in the seeding year.](image-url)
tion 12 proprietary cultivars with moderate (30%) to high (60%) levels of resistance to PRR.

Resistant But Not Immune

Roots of even the most resistant alfalfa plants are not immune to PRR infection. According to Marks and Mitchell (10), the young cortical cells in the root tips of resistant plants have a hypersensitive reaction. Cells in the resistant plant roots become infected, but the rot advances much slower than in susceptible roots. Older plants with large-diameter steles are more resistant, recover faster, and are able to produce more branch roots than those with small steles.

The lack of immunity explains why even resistant plants sometimes can be severely injured or killed when growing in waterlogged soil for extended periods. Because of slower rotting and greater ability to recover, however, resistant plants are usually able to survive temporary drainage problems. Growers with soils subject to temporary drainage problems will have greater success in establishing stands and prolonging the life of the stands by planting Phytophthora-resistant cultivars.

Literature Cited


Fig. 5. In an experimental field of alfalfa at Waseca, MN, natural infection by Phytophthora killed many Vernal plants (lower right) but not Agate plants (upper left and right).

F. I. Frosheiser

Dr. Frosheiser is a research plant pathologist with USDA/SEA-AR and adjunct professor in the Department of Plant Pathology at the University of Minnesota, St. Paul. He received an M.S. degree in agronomy at the University of Wyoming in 1949 and a Ph.D. in plant pathology at the University of Minnesota in 1955. He has been with the USDA investigating alfalfa diseases since 1955.