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Problems and Progress in Contro

Phytophthora root rot of soybean (PRR) caused by *Phytophthora megasperma* f. sp. *glycinea (Pmg)*, is a rather unique disease. It originated relatively recently in the United States and is the only severe *Phytophthora* disease of a major grain crop. The pathogen is notable among the species of *Phytophthora* in consisting of many races, most of which built up in response to only two genes for resistance in popular soybean cultivars. Also, soybean is unique in having many different alleles and loci for resistance to *Pmg* available in the soybean germ plasm bank, and resistance is easy to evaluate in seedlings.

Phytophthora root rot has been a popular model system for developing the phytoalexin theory of disease resistance and, more recently, for casting doubt that phytoalexins are totally responsible for resistance. Control options in addition to resistance are the use of the least susceptible (tolerant) cultivars, systemic fungicides, cultural methods, and biological control. Some of these methods can be combined into an integrated control system. Yet, despite all these options, PRR continues to ravage soybeans and the areas subject to severe damage continue to expand. Based on a phone survey of soybean pathologists, I estimate that approximately 16 million acres are now infested with *Pmg* and subject to damage under conditions conducive to root rot in the United States and Canada. PRR is also reported from Europe, Japan, and Australia. I will attempt to explain why this disease continues to cause severe damage despite 30 years of intensive research and hundreds of research papers, and I will describe future options for control and research.

History

Phytophthora root rot of soybean was first noted as a new disease of unknown etiology in Indiana in 1948. Similar root rot symptoms were found in Ohio in 1951 and in other northern midwestern states shortly thereafter. Symptoms were originally thought to be caused by *Fusarium* or *Diaporthe* (pod and stem blight). *Phytophthora* was first associated with soybeans in North Carolina and Ohio in 1954. The North Carolina pathogen, called *P. cactorum*, was isolated from damped-off seedlings; Ohio collections (from stem lesions) were not identified to species at first but were later called *P. cactorum*.

The first comprehensive report of this disease by Kaufmann and Gerdemann was published in 1958 and the pathogen was called *P. sojae*. A year later in a second definitive report, Hildebrandt changed the name to *P. megasperma* var. *sojae*, on the basis of identification of his isolates at the Commonwealth Mycological Institute in Kew, England. This name was valid until 1980, when the fungus was reclassified as *P. megasperma* f. sp. *glycinea* by Kuan and Erwin (14) after extensive studies of the host range and oospore size. They concluded that oogonial size of *P. megasperma* isolates from different hosts formed an overlapping continuum and was, therefore, unsuitable for a variety separation. However, soybean and alfalfa isolates of *P. megasperma* had sufficiently distinct host ranges to place them in two forma speciales (*P. megasperma* f. sp. *glycinea* and *P. megasperma* f. sp. *medicaginis*), separate from *P. megasperma* found on other hosts. The use of the forma speciales designation should prevent future confusion between these two important pathogens.

The Fungus

Pmg is characterized by large obpyriform sporangia (mean length 40 μm, mean width 28 μm) that have inconspicuous papillae (Fig. 1) and are proliferous (new sporangium form inside one that has germinated). Oogonia are large also (mean diameter 41 μm). Antheridia are mostly paragynous (Fig. 2) or occasionally amphigynous. *Pmg* is homothallic. Mycelium and sporangia are diploid. Meiosis occurs in antheridia and oogonia and nuclear fusion takes place in the oogonium, which forms a diploid oospore. Germination of oospores results in mycelium and sporangia. Oospores form readily in culture and in diseased tissue. Sporangia also form readily if mycelia are washed repeatedly in water or Chen-Zentmyer salt solution. Dilute V-8 juice, lima bean, and cornmeal agars are the media that are preferred for culturing *Pmg*.

Isolation and Detection

Pmg can be readily isolated from stem lesions by using selective media containing 1) pimaricin plus vancomycin or 2) PCNB, benomyl, chloramphenicol, and neomycin sulfate to inhibit other fungi and bacteria. Isolation from root tissues is more difficult because the selective media do not inhibit *Pythium* spp. that are abundant in rotted roots. Thorough washing of roots followed by gentle surface sterilization (1% NaOCl for 15 seconds) will kill most surface *Pythium* spp. without killing all *Pmg* within the roots. Alternatively, sporangia can be induced to form at room temperature by incubating thoroughly washed rotted roots in distilled water. Zoospores are released from maturing sporangia and can be pipetted onto plates of selective media for isolation.

Pmg can be isolated from soil by baiting with seedlings or by a leaf-baiting technique (5). To prepare for leaf baiting, soil is incubated at 25 C for at least 1 week at a matric potential of -0.1 to -1.0 bars. The soil is then flooded and soybean leaf disks are placed immediately on the surface. Zoospores can be seen swimming near the surface of the water within 1 hour of flooding. The disks are removed after 2 hours and plated on selective media. After 48 hours of growth, *Pmg* can be identified and recovered.

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f Phytophthora Root Rot of Soybean

The best method to definitely identify the root rot phase of PRR is to clear and stain roots in boiling chloral hydrate–acid fuchsin or lactophenol–trypan blue, then crush them between two glass slides. Typical *Pmg* oogonia are readily visible throughout the rotted root tissue (Fig. 2).

Symptoms

Phytophthora can cause both preemergence and postemergence damping-off. Preemergence damping-off is characterized by rotted seed or light to dark brown, flaccid taproots of soybean seedlings (Fig. 3). Seedlings that are not killed before emergence germinate slowly and frequently die at the crook stage. Emerging crooks of infected seedlings appear translucent and may turn brown. Infected seedlings that do emerge stop growing and are rapidly killed. In postemergence damping-off, the lower taproot becomes brown and soft, with external discoloration extending up to the hypocotyl (Fig. 4). Internal discoloration may extend into the hypocotyl, break out to the surface just below the cotyledonary node, and form a girdling lesion that collapses the hypocotyl, killing the plant.

In older plants of very susceptible cultivars, a brown girdling rot may extend up the stem as high as 10 nodes before the plant finally wilts and dies (Fig. 5). Characteristic symptoms at this time are leaf flagging followed by wilting and collapse without the yellowing and epinasty typical of toxin-associated wilt diseases. In less susceptible (more tolerant) cultivars, symptoms are generally restricted to the roots. Occasionally, linear, nongirdling, slightly sunken lesions may extend up the stem as high as 10 nodes. Infected taproots are often covered with the orange masses of *Fusarium* macroconidia that led to early confusion of PRR with Fusarium stem rot. *Phomopsis* pycnidia may be found in stem lesions, leading to misidentification of the disease as pod and stem blight.

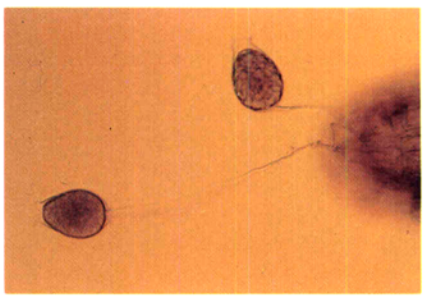

Fig. 1. Sporangia of *Phytophthora megasperma* f. sp. *glycinea* developing from the tip of an infected root incubated in distilled water at 25 C. Note new sporangia forming in empty primary sporangia, showing the proliferous sporangial characteristic.

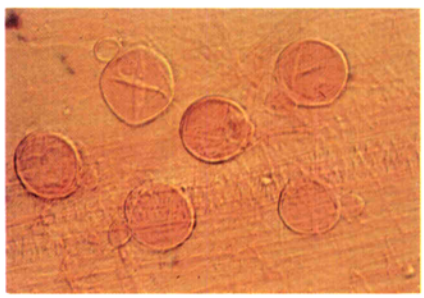

Fig. 2. Oogonia of *Phytophthora megasperma* f. sp. *glycinea* in stained and crushed roots of soybean seedlings. Note large size of the oogonia in relation to the root cells and the paragynous attachment of the antheridium (to one side of the oogonial stalk).

Ecology and Epidemiology

Pmg survives as oospores in crop residues and in soil for many years but does not grow competitively or colonize soil debris. *Phytophthora* cannot be demonstrated in soil immediately after freezing or after storage for long periods at 3 C, indicating that mycelium, sporangia, and zoospores do not survive cold temperatures. If overwintered soil is allowed to incubate for 1 week at 25 C under suitable moisture conditions, *Phytophthora* can be readily demonstrated with the leaf disk bait technique (5). Some *Pmg* can be isolated after incubation of soil at 15 C, but not 10 C. Optimum leaf-disk infection occurs after 2 weeks of incubating overwintered soil at 25 C.

These data indicate that *Pmg* is typical of most soilborne *Phytophthora* spp. (9); it survives as a resistant oospore that germinates when dormancy is broken and when temperature and moisture are suitable. Sporangia are formed and accumulate in soil. Zoospores are quickly released when soil is flooded and are attracted to and aggregate around germinating seeds, young roots, or exudates from older roots. Phytophthora root rot is not a problem unless soil is saturated

Fig. 3. Typical preemergence damping-off of soybeans caused by *Phytophthora megasperma* f. sp. *glycinea*.

for a significant length of time, but the minimum saturation time at various temperatures has not been established.

The relative importance of primary and secondary inoculum for infection has not been evaluated. Sporangia form very rapidly on rotted, flooded seedling roots (Fig. 1), so inoculum is abundant in flooded fields with infected plants. Many soybean cultivars quickly develop field resistance (tolerance), however, and may not be significantly damaged after the seedling stage.

Another unsolved problem is the length of time that oospores survive in the

field. Massive numbers of oospores are produced in soybean roots during epidemics. Severe root rot can occur on double-cropped soybeans planted after wheat in July, so, apparently, residual oospores germinate and new sporangia accumulate throughout the summer. Yet, extended rotations (4 years) with a nonhost do not eliminate the pathogen. There appears to be some internal timing mechanism that prevents synchronous oospore germination and prolongs dormancy for indefinite periods in a portion of the population. Oospores are known to be endogenously dormant and do not require exogenous nutrients for germination. While no studies have been made on germination of oospores produced in the field, germination of oospores produced in the laboratory is unpredictable (11). We do not yet know what factors break dormancy of *Pmg* oospores.

Resistance and Races

Resistance to *Pmg* was found the same year (1954) that the fungus was first associated with PRR in two group I maturity cultivars, Blackhawk and Monroe, already in production. This resistance gene (*Rps*$_1$) was soon released in three additional cultivars in Ohio, and the same gene was incorporated into the most popular Upper Midwest cultivar, Harosoy, released in 1964 as Harosoy 63. Other resistant cultivars followed. Much of the acreage in the *Phytophthora* area was cropped to resistant cultivars with the *Rps*$_1$ gene by 1972. In the South, resistant cultivars were widely grown also. The *Phytophthora* problem appeared to be on its way out.

Pmg race 2 was identified in the southern United States in 1965 but was found only on breeding material. In the North, many other new races were found: race 3 in 1972, race 4 in 1974, races 5 and 6 in 1976, and races 7, 8, and 9 in 1978. Severe *Phytophthora* damage again appeared throughout the original infested areas in the North. *Phytophthora* damage became more widespread with proliferation of new, highly susceptible (low-tolerant) public and private cultivars. Races 10 through 16 and races 17 through 20 were reported from the South in 1981 and 1982, respectively. Races 21, 22, and 23 were reported from the northern Midwest in 1983, and race 24 was reported from Mississippi in 1984. These races increased as a result of widespread planting of cultivars with only *Rps*$_1$, *Rps*$_1^c$, or *Rps*$_1^b$ *Rps*$_3$ genes for resistance. *Pmg* races 1, 3, 4, and 7 are the most common in the northern Midwest (15,20). The race picture is becoming so complicated that a new nomenclature system may be necessary. A formula method has been suggested to describe each race by its host gene interaction (20). Only a few of the theoretically possible *Pmg* races have been described, based on the number of known resistance genes.

Genetic analysis has revealed that the first gene for resistance found was one of an allelic series consisting of *Rps*$_1$, *Rps*$_1^b$, and *Rps*$_1^c$. A root resistance gene, *Rps*$_2$, was next found, followed by *Rps*$_3$, *Rps*$_4$, *Rps*$_5$, *Rps*$_6$, *Rps*$_1^k$, *Rps*$_7$, and more alleles at the *Rps*$_3$ locus (2). Other yet unidentified genes may be present in soybean germ plasm.

The long-term usefulness of single genes for control of *Phytophthora* is debatable. Races capable of attacking *Rps*$_1$ resistance increased significantly throughout the Midwest in about 8 years. *Rps*$_1^c$ resistance (controlling races 1, 2, 3, 6 through 11, 13, 15, 17, 21, and 23) was introduced in 1980 (Fig. 6) and already is ineffective in many fields where race 4 has increased. Cultivars with *Rps*$_1^k$ (controlling all races but 12, 16, 19, and 20) or *Rps*$_1^c$ *Rps*$_3$ (controlling all races but 12, 19, 20, and 22) or *Rps*$_1^b$ *Rps*$_3$ (controlling all races but 10, 12, 19, and 20) are now available. However, nonselective isolation of *Pmg* from soil in Ohio has revealed new races not yet found in soybean, some of which were virulent to *Rps*$_1^k$ and one to the *Rps*$_1^b$ *Rps*$_3$ combination (Hobe and Schmitthenner, *unpublished*). In the South, two-gene resistance is prevalent, yet many new races have been reported. It will be very interesting to see how long *Rps*$_1^k$ (Williams 82), *Rps*$_1^c$ *Rps*$_3$ (Keller and Miami), and *Rps*$_1^b$ *Rps*$_3$ (Winchester) resistance remains effective.

Testing for single-gene resistance is very easy. Most commonly, mycelium is placed in a hypocotyl slit 1 cm below the cotyledonary node. Inoculated plants are kept in a moist chamber overnight or the slit is covered with petrolatum. Susceptible plants die in 2–4 days (Fig. 7). Single-gene resistance is also very easy to incorporate and therefore will continue to be popular among soybean breeders. Resistance has two advantages: 1) complete disease control and 2) ease of incorporation. Resistance has three disadvantages: 1) Backcrossing takes time and therefore only old cultivars have resistance, 2) there may be a slight yield loss for incorporation of each gene for resistance, and 3) resistance promotes buildup of new races that render resistance ineffective. Resistance could be made more durable by pyramiding many different single genes within a cultivar, provided incorporation of all these genes does not significantly reduce the yield potential of the cultivars. An alternative approach would be to incorporate resistance into the least susceptible (most tolerant) cultivars.

The *Pmg*-soybean hypocotyl model has been very useful and popular in research to elucidate the mechanism of resistance. There now is general concurrence (17) that accumulation of phytoalexins (several isomers of glyceollin) is correlated with cessation of growth of *Pmg* in resistant plants (incompatible interaction). Glyceollins accumulate in a narrow band of cells adjacent to the infection site. They are stress metabolites produced in response to biotic (fungi and bacteria) or abiotic (heavy metal and detergent) elicitors. Specific biotic elicitors are probably low molecular weight carbohydrates released after host-pathogen contact. All elicitors induce de novo production of enzymes involved in phenylpropanoid metabolism. Inhibition of phenylalanine ammonia-lyase suppresses glyceollin accumulation, and resistance is lost. In susceptible plants (compatible interaction), glyceollin is formed initially but the rate of synthesis declines significantly after 14 hours, compared with a resistant interaction. Apparently, glyceollin synthesis in the compatible interaction is somehow inhibited after 14 hours and this inhibition is race-specific (24). There is still disagreement among researchers as to whether race specificity involves induction or suppression of glyceollin accumulation. Evidence has been presented that accumulation of glyceollin may be incidental to resistance (23) and that *Pmg* is inhibited in resistant plants by some unrelated mechanism.

Field Resistance and Tolerance

Some susceptible cultivars are not severely damaged by *Pmg* in the field but are killed when hypocotyls are inoculated with a compatible race. Such cultivars have been described as having field resistance, field tolerance, tolerance, rate-reducing resistance (18), or root resistance (2). Yield of field-infected plants of such cultivars may not be adversely affected compared with that of healthy or resistant plants. This type of disease reaction—root rot with little yield loss—should be called tolerance, following the concepts of Mussell (16).

Whereas resistance is qualitative and race-specific, PRR tolerance is quantitative and race-nonspecific. Tolerance has a relatively high heritability, indicating that a limited number of genes is involved (22). In general, late cultivars have a higher tolerance than early ones. It is not clear if maturity is involved or if the genetic background of early cultivars is sufficiently different to account for this phenomenon. Glyceollin accumulation has been implicated in race-specific resistance to *Pmg*, but not tolerance (Olah and Schmitthenner, *unpublished*). It is likely that tolerance involves different mechanisms from race-specific resistance and that concepts of elicitation, enzyme induction, and glyceollin accumulation associated with resistance may not be applicable. Also, there is no evidence that the gene-for-gene theory is applicable to tolerance or that the reaction is the result of accumulation of defeated resistance genes. More information on the mechanism of PRR tolerance is needed before its full potential can be realized and commercially manipulated.

PRR tolerance can be evaluated in the field by comparing test lines with cultivars having a known PRR reaction. It is important that consistent, severe disease occurs every year in the test field. The field should be poorly drained, and fall plowing or no-till is better than spring plowing. Planting after the soil has warmed to 20 C and flooding the seedlings at the crook stage will help ensure uniform seedling disease. A comparison of the vigor of the surviving plants with the vigor of cultivars of known tolerance at growth stage R7 (physiological maturity) provides a good prediction of the performance under PRR conditions (Fig. 8). Tolerance has been evaluated by differences in plant loss (dead plants) among cultivars. This method may not be sufficiently sensitive for separating the best from the moderately good lines. A number of greenhouse tests have been developed to evaluate PRR tolerance of cultivars in the seedling stage (21). Most evaluate seedling root rot, but one method involves the rate of cotyledon and cotyledonary node infection. Generally, seedling tests correlate well with field reactions and yield in the presence of PRR.

The ultimate test for PRR tolerance is a yield comparison of cultivars under severe disease conditions either with a resistant cultivar or in soil treated with the fungicide metalaxyl as a reference. Unfortunately, near-isogenic resistance is not available in enough different agronomically useful cultivars to provide a good range of references. Also, metalaxyl, the best *Phytophthora*-specific soil fungicide, may reduce yield of some cultivars. Often, one has to settle for a yield rank comparison of cultivars under severe disease and disease-free conditions.

PRR tolerance was first used extensively in Ohio in the early 1970s when race 3 became so prevalent that race 1 resistance was no longer effective. Growers found that they could obtain acceptable yields even though PRR was present. The first tolerant cultivars identified have not lost their yield potential in *Pmg*-infested soil in 11 years, so there does not appear to be any loss of tolerance or increase of super races. With the release of Rps_1^c resistance (controlling all common races except 4 and 5), it was possible to evaluate how effective tolerance was for PRR control. In tests in Ohio under severe disease pressure, the best tolerance available yielded 76% as much as a resistant cultivar with approximately the same yield potential. Under mild disease pressure, the high-tolerant cultivar yielded as much as the resistant cultivar. In concurrent experiments, treatment of a high-tolerant cultivar with metalaxyl significantly increased yield compared with an untreated high-tolerant control. Since that time, even higher levels of tolerance have been identified. Approximately one-third of the cultivars available in the northern Midwest now have acceptable tolerance. However, none yield as well as resistant cultivars under severe disease pressure. It is apparent that tolerance by itself will not produce acceptable PRR control. Recent work in Ohio (Olah and Schmitthenner, *unpublished*) has indicated that not all individuals in a cultivar are equally tolerant, and it may be possible to clone for tolerance within cultivars. The yield potential and other characteristics of high-tolerant clones need to be determined, however.

Control Strategies

Fungicides. There have been some recent exciting developments in fungicide control of PRR. Pyroxyfur (Grandstand), developed by Dow Chemical, was the first *Phytophthora*-selective fungicide effective against PRR. Pyroxyfur controlled damping-off well but not root rot of low-tolerant cultivars. It was very effective when used with high-tolerant cultivars. Over a 3-year test, a pyroxyfur-treated high-tolerant cultivar yielded as much as a resistant cultivar under severe disease stress. Pyroxyfur was never released because the soybean seed treatment market was considered too small.

This is unfortunate because pyroxyfur has the unique property of accumulating in roots when used as a seed treatment.

Concurrently, a second *Phytophthora*-selective fungicide, metalaxyl, developed by Ciba-Geigy, was tested on soybean. Metalaxyl is slightly more effective than pyroxyfur as a seed treatment and is even more effective as a soil treatment (1). It can be used as a seed treatment (Apron) of high-tolerant cultivars at 0.5 oz a.i./100 lb of seed (already labeled) or as an in-furrow granular treatment (Ridomil) at 4 oz a.i./acre (not yet labeled). High rates (1 lb a.i./acre as a 7-in. band) effectively controlled PRR on low-tolerant cultivars except under the most severe root rot conditions.

Metalaxyl is an important addition to our control strategies for *Phytophthora* but by itself is not the magic answer to PRR control. Metalaxyl seed treatment has three limitations. There is evidence that labeled seed-treatment rates reduce yield in the absence of PRR that can be demonstrated using resistant cultivars. This yield reduction may be enhanced in the presence of other seed-treatment fungicides. More information is needed to determine if this response is cultivar-specific or environment-specific. Second, metalaxyl accumulates in the top of the plant and does not protect seedling roots from PRR. Thus, low-tolerant cultivars will not be protected after emergence. Third, there appears to be some reluctance in the seed industry to treat seed, since the cost passed on to the grower will make treated seed less competitive.

Metalaxyl soil treatment solves most of these objections but also has some limitations. The cost to the grower will be about five times as much as seed treatment. Second, there are application problems with drilled beans. Broadcast sprays of metalaxyl combined with preemergence herbicides would be the logical method, but there are no data indicating that surface broadcast sprays of metalaxyl are effective. Preplant incorporation would be best but is not commonly used in Ohio. Also, rates for broadcasting have not been established and may be too expensive. Third, rates required for effective PRR control in reduced or no-till cropping systems have not been established. Several analogues of metalaxyl are being tested that may be more effective as seed or broadcast treatments with fewer negative effects. More active materials would be useful for specialized drilling and no-till cropping systems.

Dependence on metalaxyl seed or soil application for PRR control may pose the danger of buildup of metalaxyl-resistant strains of *Pmg*. Metalaxyl acts on *Phytophthora* in two ways. It is fungistatic, inhibiting growth and sporulation, and, as a systemic, it induces accumulation of glyceollin in challenged hypocotyls (3) and probably in roots. Resistance of the fungus to direct toxicity

Fig. 4. Typical postemergence damping-off symptoms in soybeans caused by *Phytophthora megasperma* f. sp. *glycinea*. Note brown discoloration extending up the hypocotyl to the cotyledonary node.

Fig. 5. Typical stem lesion (left plant) produced by *Phytophthora megasperma* f. sp. *glycinea* in a low-tolerant soybean cultivar. Note brown, girdling stem lesion extending up the stem for several nodes without killing the plant.

of metalaxyl has been induced by exposure to metalaxyl, but the resistant isolates were not virulent in metalaxyl-treated hypocotyls (6). As yet, there is no indication of resistance to metalaxyl in field isolates of root-infecting *Phytophthora* spp., although differences in sensitivity occur among isolates of *P. megasperma* (10). Development of resistance, should it occur, will probably take many years because of the limited number of generations of sporangia produced during a season. Use of metalaxyl with only high-tolerant cultivars would further delay development of resistance because even fewer generations of inoculum would occur with these cultivars. Isolates resistant to metalaxyl would not necessarily be able to colonize root tissue in which resistance was induced by systemic metalaxyl. A virulence change would also have to occur. There is evidence (Schmitthenner, *unpublished*) that low rates of metalaxyl are more effective in high-tolerant than in low-tolerant cultivars. Again, the use of metalaxyl with only high-tolerant cultivars may lengthen the usefulness of this fungicide.

Cultural factors. Soil moisture is one of the most critical factors affecting PRR severity (7,13). As indicated earlier, saturated soil is essential for indirect germination (zoospore formation) and dispersal of inoculum of *Pmg*. Generally, disease is not a problem in well-drained soils unless a perched water table prevents rapid drainage through large soil pores. In heavy soils, tiling can increase the rate of drainage. As little as 2 hours of soil flooding may result in significant infection by released zoospores, as demonstrated with the leaf disk assay. Under normal rainfall conditions, good drainage may control disease by itself. In low areas or floodplains or after heavy rains, however, good drainage is not possible if ditches and natural waterways fill and water has no place to drain. This is a common occurrence in the lake bed area of Ohio and in river bottoms. Cyclic drought stress also may increase severity of PRR as it does with other Phytophthora root rots. There is no control over moisture distribution in most of the soybean-growing region, but in irrigated areas, avoidance of drought stress might significantly reduce the impact of PRR.

Tillage is the second most critical factor for PRR control. Early work in Ontario indicated a positive correlation between bulk density of soil and disease severity. Spring plowing reduces bulk density and decreases disease. Disease is always more severe in tractor tracks and where equipment turns at the end of a field. Excessive spring tillage (multiple disking) may compact soil and increase disease severity. The importance of bulk density has been confirmed in Illinois (13).

Tillage intensity may be negatively correlated with PRR severity (18). Evidence is accumulating that PRR is more severe in reduced tillage and no-till planting systems. In Ohio, the standard tillage system of fall plowing, followed by spring seedbed preparation with a field cultivator, appears to result in less disease than no-till or reduced tillage systems. Reduced tillage enhancement of PRR is not clearly understood. Complete tillage may decrease soil bulk density compared with reduced tillage. The rate of water percolation might be slower in reduced than in complete tillage, resulting in longer periods of soil saturation. Tillage mixes and dilutes inoculum, whereas in a no-till system, inoculum may be concentrated in permanent soil cracks where roots accumulate. Additional research is needed to clarify the mechanism by which reduced tillage enhances PRR.

Other factors having a minor effect on PRR severity are rotation, fertilization, and ridging. Rotation of soybean with nonsuscepts of *Pmg* may decrease severity of root rot slightly. In more recent work (Schmitthenner and VanDoren, *unpublished*), rotation reduced the yield loss from PRR by 50% as much as did improved drainage and complete tillage. This is not surprising in light of the well-known longevity of *Pmg* oospores. High fertility may increase severity of PRR in soybean slightly (7), about as much as rotation decreases severity. In preliminary laboratory work, chloride salts increased PRR more than

sulfates or phosphates did (4). Chloride salts generally are added as muriate of potash and would be present if potash were added immediately before planting or if heavy rates of manure or sludge are used. The mechanism of chloride enhancement of PRR is not known. Planting soybeans on ridges or beds has been attempted in Ohio to compensate for poor drainage, but results have been generally disappointing. Ridged planting did not eliminate PRR in wet years and was not as effective as tile drainage. Some herbicides, such as 2,4-DB, trifluralin (8), or sublethal rates of glyphosate (12), may increase severity of PRR and should be used with care.

Biological control. There is increased interest in biological control of *Pmg*. In Michigan, an attempt has been made to utilize various fungal and actinomycete parasites of oospores for reducing inoculum and disease. Promising results have been obtained in controlled tests (19), but the system has not been evaluated in the field. There is research in progress elsewhere on the use of various antagonistic fungi and bacteria as seed treatments for control of PRR. Ultimately, the system that is developed must be effective under saturated conditions and either prevent aggregation of zoospores on roots or interfere with their germination and penetration.

Integrated control. It is now apparent that no single strategy will be effective for completely eliminating PRR. Resistance will not last because of the demonstrated high pathogenic variability in *Pmg*, and PRR tolerance does not offer a high enough level of control. Fungicides, such as metalaxyl, are very effective with certain—but not all—cultivars. Various cultural factors can be utilized to reduce PRR damage but do not offer complete control. I propose two options for integrated control. Neither will completely eliminate disease, as does resistance, but both will result in satisfactory yields in the presence of disease.

The first option is to combine the use of metalaxyl soil treatment with high-tolerant cultivars. In Ohio, metalaxyl has been evaluated on high-tolerant and low-tolerant cultivars in nine experiments over 3 years in nontiled, poorly drained fields (18). The high-tolerant cultivar yielded 86% more, the high-tolerant cultivar with metalaxyl seed treatment yielded 116% more, and the high-tolerant cultivar with metalaxyl soil treatment yielded 136% more than the low-tolerant cultivar. Yield of the low-tolerant cultivar was improved 53% by metalaxyl seed treatment and 113% by metalaxyl soil treatment. The resistant cultivar yielded only 100% more than the low-tolerant cultivar without fungicide. Thus, the best control was obtained with high tolerance combined with metalaxyl soil treatment. This combination has three disadvantages mentioned earlier: 1) The cost may be too high (about $15/acre), 2) in-furrow application of metalaxyl is not practical with drilled soybeans and broadcast applications have not yet been adequately evaluated, and 3) soil use of metalaxyl for soybeans has not been labeled.

The second option consists of combining high-tolerant cultivars, complete tillage, tile drainage, rotation, and metalaxyl seed treatment. Preliminary information from experiments in Ohio (Schmitthenner and VanDoren, *unpublished*) indicates the following yield increases: 25% with high tolerance compared with low tolerance, 19% with complete tillage compared with no tillage, 10% with tile drainage compared with no drainage, 9% with rotation compared with continuous soybeans, and 6% with metalaxyl seed treatment compared with no seed treatment. Yield increase for integrated control was 48% compared with 24% for resistance. Metalaxyl seed treatment had a small impact on yield in this system but may be important if uncontrolled flooding conditions occur soon after planting. The disadvantages of this system are the cost of tiling and its unacceptability to growers who prefer to plant no-till soybeans. The only option these growers have is to use resistant cultivars or, possibly, soil-applied metalaxyl when it becomes labeled.

Both these integrated control options are independent of *Pmg* races. Both will allow the grower to produce high yields with *Pmg*-susceptible cultivars provided they have high tolerance to PRR. High-tolerant cultivars are the key to integrated control.

Fig. 6. Portion of a soybean *Phytophthora* nursery in Ohio in 1976 showing failure of race 1 resistance. Rows one, three, and five are lines with Rps_1 resistance and rows two and four are lines with Rps_1^c resistance. Cultivars with Rps_1^c resistance no longer control *Phytophthora* in this field and elsewhere in Ohio where races 4 and 5 have built up.

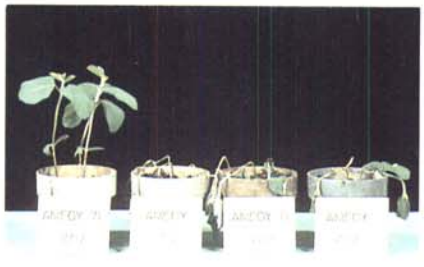

Fig. 7. Amsoy 71 (with Rps_1 for resistance to race 1) and Amsoy (no resistance) inoculated in the hypocotyl with race 1 (isolate 507) and race 3 (isolate 627). Note race 3 kills plants with race 1 resistance.

Fig. 8. Portion of a hill plot in a *Phytophthora* screening nursery (hills, each planted with 12 seeds, are 25-cm rows spaced 75 cm apart). All lines are susceptible to race 1, 3, 4, or 7 present in the field. The line in the center is considered high-tolerant and the line at the bottom, low-tolerant; all other lines have intermediate levels of tolerance.

Conclusions and Future Direction of Research

Phytophthora root rot of soybeans continues to be a problem and appears to be spreading for several reasons. There has been too much reliance on single-gene resistance. New races of *Pmg* can readily build up in the presence of single-gene resistance. During the past 10 years the market has been flooded with many new cultivars, many of which have little tolerance (are very susceptible) to PRR. These cultivars have been used widely in areas not thought to have PRR. Early-season flooding, conducive to Phytophthora damping-off and inoculum increase, appears to be occurring more frequently. Damage from this disease can be eliminated by using cultivars resistant to all the common races of *Pmg* or by either of two systems of integrated control. These are combining high tolerance with metalaxyl soil treatment or combining high tolerance with good drainage, maximum tillage, rotation, and metalaxyl seed treatment.

In developing controls for Phytophthora root rot during the past 30 years, we have just scratched the surface of our knowledge of this disease. Numerous intriguing questions remain to be answered. What is the mechanism of virulence in *Pmg*? How do compatible races suppress glyceollin production or whatever mechanism is responsible for cessation of mycelial growth in incompatible interactions? Once this process is known, it might be possible to find chemicals that would trigger induction of resistance irrespective of pathogen virulence genes. Is the mechanism of PRR tolerance (partial suppression of

mycelial growth) similar to resistance (complete suppression of mycelial growth)? Cultivars need to be cloned for specific tolerance reactions before such mechanisms can be effectively studied. Is tolerance the result of accumulation of defeated resistance genes, as suggested by Nelson? Pyramiding as many resistance genes and combinations of resistant genes as possible into low-tolerant and high-tolerant cultivars will provide material necessary for examining this theory. Adding multiple genes through backcrossing and progeny testing is very time-consuming. Production of multiple, clonal test units through somatic embryogenesis would help speed up development of multigenic resistant cultivars.

How are virulence genes inherited in *Pmg*? Is there a gene-for-gene relationship between *Pmg* and soybean? Methods need to be developed to cross homothallic *Phytophthora* spp. and consistently germinate oospores before such questions can be researched. What factors trigger oospore germination? Are dormant oospores inert and unaffected by their biotic and abiotic environment? The answers to these questions would be useful in developing biocontrol for *Pmg*. What are the mechanisms of fertilizer and salt enhancement of Phytophthora root rots? Are there fertilizer regimes that can reduce severity of PRR? Suppressive fertilizer applications and biocontrol both would be useful components to add to integrated control. As answers to some of these questions are forthcoming, additional, novel control methods for this important disease might be suggested.

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