Pre-breeding for Root-rot Resistance

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ABSTRACT

A number of crown and root rot diseases reduce yield in sugarbeet. Many of these diseases are limited to small geographic areas and their incidence is often sporadic. Hence, development of resistant cultivars has not been a high priority. Rhizoctonia, Aphanomyces, and Erwinia root rots are exceptions. Commercially useful resistance to *Rhizoctonia* and *Aphanomyces* originated from only a few sources. *Erwinia* resistance is available from numerous sources and is relatively easy to select for. Selection for resistance to prevalent storage rot fungi is possible but has not received much attention from commercial sugarbeet breeders. Knowledge of the inheritance of root or storage rots is frequently incomplete and sometimes inconsistent. Results of systematic screenings of the USDA Beta collection confirm the scarcity of resistance to *Rhizoctonia* and *Aphanomyces* and the difficulty of broadening the currently narrow genetic base of sugarbeet. Genetic engineering techniques probably will not make a contribution to the development of root rot resistant germplasm in the near future. Understanding the biochemical basis of resistance will eventually improve selection efficiency and hasten the application of genetic engineering technologies to the problems of host plant resistance.

Additional Key Words: Sugarbeet; *Beta vulgaris* L.; *Sclerotium rolfsii* Sacc.; *Rhizoctonia solani* Kühn; *Aphanomyces cochlioides* Drechsler; *Erwinia carotovora* (Jones) Holland; *Phoma betae* (Oud.) Frank; *Penicillium claviforme* Bainier; *Botrytis cinerea* Pers. ex Fr.; *Fusarium oxysporum* Schlecht

A number of root and crown rot diseases cause yield losses in sugarbeet *(Beta vulgaris* L.) (Lejealle, 1982). Since many are limited to small geographic areas and disease incidence is sporadic among years and fields, the development of resistant genotypes generally has not been a high priority. Losses from the crown and/or root rot diseases Rhizoctonia, Aphanomyces, and Erwinia have been sufficient to justify development of commercially useful germplasm. Fungal pathogens also are responsible for deterioration of stored sugarbeet. Because these losses are variable from year to year and not easily measured, the development of hybrids with resistance to the major storage-rot fungi has not been an objective of commercial sugarbeet breeders.

SOUTHERN SCLEROTIUM ROOT ROT

The extreme severity of Southern Sclerotium rot *(Sclerotium rolfsii* Sacc.) has discouraged the expansion of sugarbeet into more humid areas and is a problem in some irrigated areas with relatively high growing season temperatures. Lawlor and Doxtator (1950) found differences among genotypes for survival rate and suggested that a breeding effort could successfully produce *Sclerotium* resistant cultivars. Coe and O'Neill (1983) developed a greenhouse screening procedure for *Sclerotium.* They found no significant differences in resistance among cultivars inoculated with six *Sclerotium* isolates. Progeny selected from one breeding line were more resistant than the parental line but did not have sufficient resistance to allow sugarbeet production in problem areas. Future efforts to develop *Sclerotium* resistant germplasm are unlikely unless there are economic or political reasons for expanding production into areas where the disease is a problem.

RHIZOCTONIA ROOT AND CROWN ROT

Rhizoctonia root and crown rot *(Rhizoctonia solani* Kühn) is endemic to many sugarbeet production areas. *Rhizoctonia* can survive as a saprophyte in the soil for many years, making cultural controls ineffective and host plant resistance a desirable alternative. Most of the resistance breeding efforts have utilized field screening and testing under artificially induced epiphytotics. Individual plant ratings are required to obtain the precision necessary for meaningful progress with most selection schemes (Hecker and Ruppel, 1977). Ruppel and Hecker (1988) found that timing of inoculation was a critical factor for regulating disease intensity and suggested that early

generation breeding populations be subjected to less severe disease pressure than more advanced (resistant) populations and lines. Greenhouse screening procedures may provide preliminary results in a short time (Campbell and Altman, 1976) but cannot be substituted for field testing.

Gaskill (1968) did not find any substantial *Rhizoctonia* resistance among 226 accessions, including 19 *Beta vulgaris* ssp. *maritima* accessions. Resistance was observed after two or three cycles of selection within 'GW674-56C' and a line derived from 'GW359' (C817), both commercial open-pollinated cultivars. After four cycles of mass selection for *Rhizoctonia* resistance, this material was released as germplasm lines FC701 and FC702 (Hecker and Gaskill, 1972). These lines provided a basis for much of the subsequent improvement in level of resistance and agronomic characteristics. FC704, a red-root *Rhizoctonia* resistant germplasm line, was the product of two cycles of selection from 'German red beet', the only unselected sugarbeet germplasm observed with a significant amount of inherent resistance to *Rhizoctonia* (Hecker and Smith, 1979). FC712 was selected from a composite cross of obsolete open-pollinated cultivars plus a small *B. maritima* contribution (Hecker and Ruppel, 1986). FC706 also was selected from a population that included some *B. maritima* genes (Hecker and Ruppel, 1979). FC711 was developed from breeding lines brought to the United States from Japan (Hecker and Ruppel, 1983), and provides a source of resistance distinct from previously available lines. FC710 also is a resistant germplasm line selected from parental material that includes lines not previously utilized in a *Rhizoctonia* breeding program (Hecker and Ruppel, 1991). *Rhizoctonia* resistant cytoplasmic male-sterile (CMS) lines (Hecker and Ruppel, 1981) and tetraploid lines (Hecker and Ruppel, 1979) are available to commercial breeders. Immunity to *R. solani* has not been observed in sugarbeet.

Gaskill et al. (1970) reported that the *Rhizoctonia* resistance found in FC702/3 was almost completely dominant. In hybrids with FC701/3 as a parent, dominance was not as strong and the F_1's exhibited a level of resistance intermediate to the parents. It was concluded the *Rhizoctonia* resistance could be transferred with relative ease and was inherited independently of resistance to Cercospora leaf spot *(Cercospora beticola* Sacc.) and Curly top (BCTV). Hecker and Ruppel (1975) reported that resistance was conditioned by two or more loci with some additive gene action. The partial dominance for resistance observed in F_1's was thought to be sufficient for the production of useful commercial hybrids. Hecker and Ruppel (1976) found no difference in resistance between diploid and tetraploid lines but observed a dosage effect in triploid hybrids. No cytoplasm by ploidy

level interactions were observed. They recommended using resistant tetraploid pollinators for the production of triploid hybrids for sugarbeet production areas were *Rhizoctonia* resistance would be beneficial.

APHANOMYCES ROOT ROT

Black root or Aphanomyces root rot *(Aphanomyces cochlioides* Drechsler) development is favored by high soil moisture and warm temperatures. Control measures include enhancing drainage, controlling weed hosts, and crop rotation. Resistant cultivars are available for some regions. Selecting for *Aphanomyces* resistance has involved both field and greenhouse evaluations. Schneider (1954) described optimum conditions for disease development in greenhouse tests. Greenhouse results corresponded closely with field results (Schneider, 1954; Henderson and Bockstahler, 1946). Schneider and Hogaboam (1983) argued that annual selection within most breeding populations should be routine for hybrid development programs in areas where Aphanomyces root rot causes frequent damage. By eliminating the most severely damaged one-fourth of the breeding lines they were able to maintain an average level of resistance only slightly below 'USH20', a moderately resistant commercial hybrid. Caution must be exercised when comparing hybrids to inbred lines as heterosis may partially compensate for lack of resistance (Coe and Schneider, 1966).

The *Aphanomyces* resistance observed in US216, a Cercospora leaf spot resistant inbred line, stimulated resistance breeding efforts that led to the release of US1177 (Coons, 1953). Bockstahler and Reece (1948) successfully selected for resistance within US216, Minnesota synthetic 1, and Minnesota synthetic 3. More recent releases of parental lines with at least moderate resistance to *Aphanomyces* include SP6322-0 (Coe and Hogaboam, 1971), SP8030 (Coe,1981a), and EL40 (Hogaboam et al., 1982). Resistant CMS lines are also available (Coe, 1974; Coe, 1981b). Immunity to *Aphanomyces* has not been observed.

Hybrids with US216 as a pollinator expressed a level of *Aphanomyces* resistance comparable to US216 (Coons et al., 1946). This observation and the results of Bockstahler et al. (1950) indicated that resistance was dominant. However, Coe and Schneider (1966) obtained high levels of resistance only after many selection cycles and concluded that resistance was not simply inherited.

ERWINIA ROOT ROT

Bacterial vascular necrosis and rot or Erwinia root rot *(Erwinia carotovora* (Jones) Holland) is the only sugarbeet root rot caused by

a bacterium. Recognition of Erwinia root rot as a serious disease problem followed the introduction of two virus yellows (BYV) resistant hybrids, USH9A and USH9B, in the late 1960's in California. The pollen parents of these hybrids were more susceptible to Erwinia root rot than the parental material from which they were derived and other widely used parental lines (Whitney and Lewellen, 1977). The severity of the disease prompted efforts to screen germplasm (Whitney, 1982) and determine the inheritance of resistance. Breeding populations responded to selection rapidly with much of the improvement occurring in the first selection cycle, evidence for control by a single locus. The higher levels of resistance obtained with additional selection cycles suggested that additional genetic factors controlled the rate of disease development (Whitney and Lewellen, 1978a). Further study indicated that resistance was simply inherited, with a large dominance component. A second, primarily additive, component determined the amount of rot in susceptible individuals. This additive component may confer useful levels of resistance in the absence of a major resistance gene (Lewellen et al., 1978). Two Erwinia root rot resistant pollinator lines have been released, both were selected from C13, the susceptible pollinator of USH9A (Whitney and Lewellen, 1978b).

FUSARIUM ROOT ROT

Fusarium oxysporum Schlecht is a soil borne fungus that invades the vascular system. It frequently occurs as a stalk blight in seed production fields but also can cause losses in grower's fields. McFarlane (1981) observed responses from near immunity to dead among inbred lines screened for resistance to Fusarium stalk blight. Selection for resistance was effective and relatively easy. Resistance appeared to be dominant and was not linked to the monogerm trait. C566, a selection from C563, was released as a stalk blight resistant germplasm line.

STORAGE ROTS

Storage rots caused by three fungi are major contributors to deterioration during sugarbeet storage. *Phoma betae* (Oud.) Frank is potentially the most devastating because its disease cycle is closely associated with the life cycle of the sugarbeet. While many species of *Penicillium* can cause storage rot, *Penicillium claviforme* Bainier has been identified as the most damaging in some regions. *Botrytis cinerea* Pers. ex Fr. is more aggressive than *Phoma* or *Penicillium* and is able to rot tissue quickly over a wide temperature range. Bugbee (1979a; 1979b) described methods for evaluating individual roots for response

to storage rotting fungi and demonstrated that selection for combined resistance was possible. Comparisons of rot-resistant lines and commercial hybrids demonstrated that genetic resistance could reduce sucrose losses comparable to the application of a fungicide (Bugbee and Cole, 1979).

Selecting for resistance to storage rots was suggested by Gaskill (1952) and Nelson and Oldemeyer (1952). Commercial cultivars that sustained 1.5 to 2 times less damage from storage pathogens than nonselected cultivars were available in the former Soviet Union (Korniyenko, 1975; Popova, 1961). Five germplasm lines with resistance to storage rot fungi have been released (Bugbee 1978; Campbell and Bugbee, 1985). Three of these (F1001, F1004, and F1005) were selected from plant introductions from Russia; one (F1002) was selected from FC701/4, a *Rhizoctonia* resistant germplasm line; and one (F1006) from a polycross of storage rot resistant individuals from accessions in the USDA, National Plant Germplasm System *Beta* collection. In crosses involving F1004, F1005, and F1006 with seven CMS lines, the hybrids were intermediate to the parents. Hybrids with FC708CMS, a *Rhizoctonia* resistant line, or SP69550-01, an *Aphanomyces* resistant line, were more resistant to storage rots than hybrids involving the other CMS lines (unpublished data). Subsequent research has suggested an association between resistance to *Rhizoctonia* and resistance to *Phoma* and *Botrytis* storage rots (Bugbee and Campbell, 1990).

DISCUSSION

Most of the current commercial sugarbeet germplasm was originally developed in northern temperate climates and traces back to 'Silesian fodder beet.' The relatively low incidence of disease in these regions did not provide sufficient stress for maintenance of high levels of host plant resistance (Lewellen, 1992). In most breeding programs, breeders have capitalized on a few sources of disease resistance and devoted much of their effort to improving agronomic characteristics of resistant lines. Although U. S. breeders have a large *Beta* collection available, it has not been used extensively as a source of resistance genes (Lewellen, 1992). The U. S. Sugarbeet Crop Advisory Committee has attempted to remedy this, in part, by coordinating a systematic screening program for some economically important diseases. The evaluation data are made available on the Germplasm Resources Information Network (GRIN). These screenings confirm the scarcity of high levels of resistance to *Rhizoctonia* and *Aphanomyces* (Table 1) and the difficulty of expanding the currently narrow genetic base for sugarbeet, if resistance to these diseases

Table 1. Response of accessions from the USDA Beta collection screened for reaction to three root-rot organisms, 1987-1991 (Source: USDA/ARS GRIN database).

Disease agent	*Beta* species	Resistant (1 - 3)*	Intermediate (4 - 6)	Susceptible (7 - 9)	Total
			no.		
Rhizoctonia					
	B. vulgaris	12	14	265	291
	B. maritima	1	32	30	63
	B. cicla	—	1	1	2
	B. macrocarpa	—	1	5	6
	Total	13	48	301	362
Aphanomyces					
	B. vulgaris	—	162	50	212
	B. maritima	9	20	2	31
	B. macrocarpa	—	3	1	4
	Total	9	185	53	247
Erwinia					
	B. vulgaris	100	11	1	112
	B. maritima	31	5	1	37
	B. cicla	1	—	—	1
	B. macrocarpa	7	1	—	8
	B. patellaris	1	—	—	1
	Total	140	17	2	159

*Rating scale of 0 = immune to 9 = severe; no 0's were recorded.

are required for commercial production. *Erwinia* resistance is available in many breeding populations and parental lines and relatively high levels of resistance frequently occur among the accessions in the collection; thus breeding for *Erwinia* resistance should not require a serious restriction of the genetic base of the commercial crop.

The development of root rot resistant germplasm has been achieved by utilizing long established greenhouse and/or field screening techniques and traditional breeding methods. For most root rots only a few sources of resistance have been utilized and knowledge of the inheritance of resistance is limited. Germplasm collections have contributed very little, in part because of the difficulty of eliminating

undesirable traits (Lewellen, 1992; VanGeyt et al., 1990). Genetic engineering techniques might overcome these difficulties in some instances; however, the present technology is best adapted to the transfer of simply inherited traits, a major constraint in transferring resistance to many sugarbeet diseases. Bugbee (1993) identified a pectin lyase inhibitor protein in sugarbeet cell walls that is active against pectin lyase produced by *R. solani.* The concentration and activity was greater in FC712, a Rhizoctonia resistant line, than in a susceptible line. This inhibitor was also active against a pectin lyase produced by *P. betae,* providing a feasible explanation for combined resistance to Rhizoctonia root rot and Phoma storage rot (Bugbee and Campbell, 1990). Knowledge of the biochemical basis of disease resistance could provide more efficient screening techniques, clarify the inheritance of resistance, facilitate the utilization of new technologies for transferring genes, and allow germplasm collections to make a significant contribution to the genetic base of commercial sugarbeet.

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