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ROOT ROT DISEASES OF SUGAR BEET*

ABSTRACT: Root rot diseases of sugar beet caused by Rhizoctonia solani (AG 2-2 IIIB and AG 2-2 IV), R. crocorum, Aphanomyces cochlioides, Phoma betae, Macrophomina phaseolina, Fusarium oxysporum f.sp. radicis-betae, Pythium aphanidermatum, Phytophthora drechsleri, Rhizopus stolonifer, R. arrhizus and Sclerotium rolfsii cause significant losses wherever sugar beets are grown. However, not all these soil-borne pathogens have been reported in all sugar beet production areas. Losses include reduced harvestable tonnage and reduced white sugar recovery. Many of these pathogens also cause post harvest losses in storage piles. Control for diseases caused by these pathogens include disease resistant cultivars, avoidance of stresses, cultural practices such as water management and the use of fungicides.

KEY WORDS: control, effects of environment, sugar beet, root rot diseases

RHIZOCOTONIA CROWN AND ROOT ROT

Rhizoctonia crown and root rot caused by the fungus, Rhizoctonia solani, AG 2-2 intraspecific groups IIIB and IV (perfect stage, Thanatephorus cucumeris) causes one of the most damaging sugar beet diseases wherever sugar beets are grown. These fungi are considered common soil inhabitants (Windle et al., 1997). In the USA more than 24% of planted acres have economic damage from this disease while in Europe only 5—10% of planted acres are considered to have economic losses, although the incidence of this disease seems to be increasing (Büttner et al., 2003). Losses can range from negligible to more than 50%. Yield reductions result from loss of harvestable roots in the field, reduced tonnage due to decay of harvestable roots and from reduced white sugar recovery. Symptoms include a sudden and permanent wilting of leaves and a dark-brown to black discoloration of petiole bases. Taproot lesions are dark-brown to black in color and typically originate near the crown. Lesions are superficial and there is a sharp distinction between diseased and

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health tissues. *Rhizoctonia* rotted roots are often cracked. In advanced stages, the entire root may rot leaving holes in the ground at harvest. While both *R. solani* intraspecific groups can also cause damping-off and crown and root rot of sugar beet and can attack both *Phaseolus* sp. and soybean, they differ in that AG 2-2 IIIB can attack wheat, maize, rice and matt rush and can grow at 35°C whereas AG 2-2 IV does not grow at 35°C and does not attack wheat or maize. Both intraspecific groups are found worldwide although AG 2-2 IIIB seems to be more common in Europe, particularly where sugar beets are rotated with maize. *Rhizoctonia solani* AG-4 causes damping-off for sugar beets, *Phaseolus* sp., alfalfa and soybeans. It is important to identify which AG group is prevalent in a production area so that appropriate rotations can be planned. Losses are highest in warm, irrigated production areas where sugar beets are cropped intensively. Occurrence is similar in dry land production, but chronically, wet areas are often most affected. Typically, damage occurs in patches where presumably populations of suppressive organisms are low relative to undamaged areas of fields. Our research and that of others clearly show that most infections occur through the crown from bulbils (sclerotia) deposited there during cultivation and by wind or water. Infection seems to be dependent on soil temperature with temperatures less than 15—20°C resulting in little disease development compared to temperatures of 24—35°C for AG 2-2 IV. Similar data is not available for AG 2-2 IIIB although observations suggest that this intraspecific group is damaging at higher temperatures than AG 2-2 IV.

While this disease occurs in all soil types, it is commonly most severe in heavy, poorly drained soils. Once soil populations of this fungus are built up, rotation is of little value and growers are dependent on relatively ineffective cultural controls such as early planting, avoiding cultivating soil into the row, maintaining adequate, balanced fertility for good crop growth and maintaining adequate soil drainage. Effects of nitrogen fertility have been reviewed by Rush and Winter (1990) and Elmer (1997). Elmer (1997) demonstrated that application of chloride salts reduce the disease on table beet. Where disease pressure is high, growers can plant specialty varieties with resistance. Available resistance is incomplete and these varieties typically have yield potentials 10—20% less than the best approved varieties, although newer varieties are only 0—10% lower yielding than the best approved varieties (Jacobsen et al., 2005). However, these varieties may not have other important disease resistant characteristics such as resistance to the *curly top* and *Rhizomania* viruses, *Fusarium yellows*, *Aphanomyces black root rot*, or *Cercospora leaf spot*. An alternative to the use of partially resistant varieties is the use of fungicides applied in furrow at planting, when plants have 4—8 leaves or when soil temperatures at the 10 cm level are greater than 20—24°C. In studies in Montana, optimal economic returns and disease control have been with susceptible varieties with fungicide application. The best partially resistant varieties have also shown yield responses to fungicide application but have never equaled the yield of susceptible varieties sprayed with fungicide.

Several fungicides have been shown to be useful in reducing disease incidence including TPTH, chlorothalonil, pencycuron, PCNB, tebuconazole, azo-
Azoxystrobin, trifloxystrobin and pyraclastrobin. Of these, azoxystrobin has provided the most consistent level of control in both inoculated and natural infection trials (Kiewick et al., 2001; Jacobsen et al., 2005). Timing of application is critical, with fungicide deposition in the crown needed before infection occurs. Recent studies in Montana and in North Dakota have shown that application of azoxystrobin when the soil temperature at the 10 cm depth is in the 19—22°C range will provide good to excellent control while applications when the 10 cm soil temperature exceeds 24°C result in poor or no control.

VIOLET ROOT ROT

Violet root rot is caused by Rhizoctonia crocorum (Pers.: Fr.) De Candolle (perfect stage, Helicobasidium brebissonii) and has been observed sporadically throughout the western U.S. and Europe where sugar beet is grown. It is considered to be the most important disease of sugar beet in Spain. The fungus has been observed on other hosts nearly worldwide. Unlike R. solani AG 2-2, this fungus seems to be intimately associated with the roots of weed hosts such as white cockle (Lynchnis alba Mill.), annual sowthistle (Sonchus oleraceous L.), common yarrow (Achillea millefolium L.), shepherds purse (Capsella bursa-pastoris L.), medic and common groundsel (Senecio vulgaris L.) (Schneider and Whitney, 1986).

Affected plants generally occur in localized, often circular patches. While wilting sometimes occurs, root symptoms are most characteristic. Roots initially have purple, reddish-purple spots with a similar felt-like fungal growth that advances from the root tip to the crown. Roots shrivel and will have large amounts of soil associated with the root surface. Sclerotia are often found around secondary roots.

Specific control have not been developed and management involves rotations that avoid susceptible crops such as potato, sweet potato, carrot, asparagus, bean, cabbage, turnip, rape, oil seed radish, pea, clover, vetch and alfalfa and control of weed hosts. There are more than 160 plant species that are known hosts. Improved soil aeration is said to reduce disease severity. Early harvest may reduce losses.

APHANOMYCES BLACK ROOT

Aphanomyces black root has been reported from the sugar beet growing areas of the North Central and High Plains regions of the U.S., Canada, England, Europe, Chile and Japan. Losses can be 0—100% depending on environmental factors and the degree of soil infestation (Windels, 2000). In all areas, the disease occurs in two phases, acute seeding blight and chronic root rot. Infection and disease development requires warm (22—28°C), wet soils. Seedling damping-off is typically characterized initially by grayish, water-soaked hypocotyls lesions that eventually become black, constricting lesions near
the soil line. These constricting lesions often extend up the hypocotyls causing the hypocotyls be black and threadlike. Plants often break off at the site of these lesions. Chronic root rot symptoms initially appear as yellowish-brown lesions that extend into the root interior. These lesions become dark-brown or black with time. The root may rot entirely, have only tip rot, or will have bark like scabby superficial lesions. Tip rots will often become dry and constrict the root. Above ground portions of the plant will exhibit wilt on hot, bright days, often with recovery at night. Leaves may show scorch type symptoms. While the disease can develop in soils of all textures, it is most common in heavy-textured soils that have a propensity to remain wet for extended periods.

This disease is caused by the oomycete, *Aphanomyces cochlioides* D r e s c h and survives as oospores (16—24 μm) in the soil. Large numbers of oospores are produced in rooted root tissues. The fungus is homothallic and a relatively small percentage of oospores will form sporangia at any given time. Recent studies have shown that some oospores are relatively short lived, however rotations are relatively ineffective because of the large numbers of oospores produced (D y e r et al., 2004). While oospores can directly infect roots, infection is more commonly the result of sporangia formation and release of zoospores (biflagellate, 7—11 μm). Sporangia formation and zoospore release is favored by temperatures in the 22—28°C range, by presence root exudates and by presence of free water. Secondary infections can occur from zoospores released by sporangia produced on the surface of diseased roots. *Aphanomyces cochlioides* can also cause disease on table beet, mangel, chard, spinach, *Chenopodium*, *Amaranthus* and *Beta* species. Weed species appear to contribute to the survival and increase of inoculum levels in soils where rotations are used. The pathogen is spread by anything that moves infested soil. Growers should avoid pacing tare soil back on production fields.

Black root is best controlled by implementation of an integrated management program that may include partially resistant varieties, seed treatment with hymexazole, early planting, enhanced soil drainage, control of *Chenopodium* and *Amaranthus* weed species, soil indexing (long rotations with non-host crops, use of oat green manures (W i n d e l s and B r a n t n e r, 2004) and application of industrial waste lime (W i n d e l s et al., 2004). While soil indexing to predict disease severity has limitations, it is useful in predicting the need for partially resistant varieties or avoiding planting fields with high root rot potential (W i n d e l s and N a b b e n - S c h i n d l e r, 1996). It is important to understand that in general the partially resistant varieties have a lower yield potential in the absence of disease than susceptible varieties and that severely infested fields can have non-economic yields if conditions favor disease despite implementation of the best integrated management program. The use of hymexazole plus bacterial biological seed treatments has shown promise in reducing *Aphanomyces black root* damage under moderate disease pressure. Because *Aphanomyces* infected roots have higher respiration and increased invert sugar levels, they deteriorate more rapidly in storage than healthy roots thus heavily infected lots should be processed as soon as possible (C a m p b e l l and K l o t z, 2005).
PHOMA ROOT ROT

Phoma root rot caused by Phoma betae (perfect stage, Pleospora bjoerlingii, Byford) has been reported in wherever sugar beets are grown in Asia, Australia, Europe and North America. The causal fungus can cause seedling damping-off, root rot and leaf spot in the field and decay in storage piles. Unless plants are stressed in the field, root rot is generally of little economic importance. Seedling damping-off results from planting infected seed. Root rot symptoms in the field include wilting of foliage and the occurrence of small, dark-brown, sunken lesions with a watery rot near the crown. Decay spreads inward with the decayed area typically becoming dark-brown to black with prominent black lines. Later, rotted areas become coal-black, dry and shriveled. Bolting beets are most likely to be killed. Phoma rot in storage often does not appear until after approximately 80 days of storage and is considered to be one of the most important reasons for storage pile deterioration. Rot typically begins in the center of the crown and extends downward developing a cone shaped decayed area. Decayed tissues often have pockets lined with white mycelium of the causal fungus.

Infected seed is considered to be the primary source of inoculum, although ascospores have also been shown to initiate infection of seed plants after overwintering in overwintering infected root debris (Bugbee and Cole, 1981). Damping-off is favored by cool (5—20°C) weather and conditions that slow seed germination. Plants that survive seedling infection may continue to harbor the fungus systemically (Edson, 1915). Under moist conditions, conidia exude out of pycnidia on hypocotyls and may also cause leaf spots and lesions on seed stalks when spread by splashing water. The percentage decay in storage is highly correlated with the percentage of infected seed planted.

This disease is best controlled by planting seed free of infection. Seed should be produced in dry areas under surface irrigation. Seed treatments including hot water, thiram, prochloraz and benzimidazole fungicides will reduce or eliminate seed infection. Field root rot is best reduced by growing the crop with minimal stress due to water and nutrient availability. Decayed roots associated with tare or old storage piles should not be placed in production fields. The fungus has been shown to be able to survive in rotted root tissues for up to 26 months.

CHARCOAL ROT

Charcoal rot caused by Macrophomina phaseolina (Tassi) Goid. (syn. M. phaseoli (Maubl.) Ashby) is common in the hot interior valleys of California and it has also been reported in Greece, Egypt, Iran, Hungary, India and several countries in the former USSR (Schneider and Whitney, 1986). Like other root rots, Charcoal rot reduces root yield, sugar percentage and purity and makes roots less storable. Losses range from 0—30%, with high losses only occurring where beets are produced under high temperatures (31°C optimum) and significant moisture stress. Initial signs of this disease are pronounced wilting of the foliage, which subsequently turns brown and dies. Root
lesions are brownish-black and are irregular in shape, typically occurring in the crown region. Old lesions rupture to expose masses of charcoal-colored microsclerotia. Decayed areas of the internal root are initially mustard yellow and subsequently change in color to buffy citrine. In advanced stages of decay, root tissue is brownish-black, with masses of microsclerotia commonly found in cavities. Such roots may ultimately shrink and become mummified.

The fungus survives as microsclerotia in soil or host tissue for at least two years. Microsclerotia are formed in beet and other hosts such as common bean, cotton, maize, potato, sorghum, soybean, strawberry, sunflower and sweet potato (Collins et al., 1991, Su et al., 2001). Because of the longevity of microsclerotia and the broad host range crop rotation is not a practical control. Control measures should focus on preventing moisture stress by using proper irrigation methods or using cultural practices that conserve soil moisture.

FUSARIAUM ROOT ROT

_Fusarium root rot_, caused by _Fusarium oxysporum_ f.sp. _radis-betae_, has been observed in Texas, Colorado and Montana and likely occurs in other sugar beet producing areas of the U.S. (Franc et al., 2001). It has likely been overlooked because it often occurs as part of a complex with other root diseases such as _Rhizoctonia root_ and _crown rot_, _Aphanomyces root rot_ and _rhizomania_ and with the wilt disease _Fusarium yellows_. Other _Fusarium_ species such as _Fusarium culmorum_ and _Fusarium solani_ have been described as causal agents of root rot diseases in sugar beets in the U.K.

Foliar symptoms associated with _Fusarium root rot_ are similar to those of _Fusarium yellows_ caused by _Fusarium oxysporum Schlecht f.sp. betae Snyd & Hans_. These include intervenal yellowing, general chlorosis, wilting and brown scorched leaves. Initially wilted plants may regain turgor at night, but plants severely affected by _Fusarium root rot_ seldom recover completely. Root symptoms are characterized by a distinctive black external rot on the distal tip of the primary taproot and necrosis of vascular elements.

_Fusarium oxysporum_ f.sp. _radis-betae_ is morphologically similar to the _Fusarium oxysporum_ f. sp. _betae_. Microconidia (3—5 x 8—10 μm) are produced in false heads and globose to ovoid chlamydospores (4—7.5 x 20—30 μm) are common. Macroconidia are produced only sparsely on common media. These species can be differentiated by analyses utilizing RAPD PCR, isozymes from at least 3 enzymes and vegetative compatibility groupings (VCGs) (Harverson and Rush, 1997).

The pathogen most likely survives in the soil between host crops as chlamydospores and observations suggest that it can survive for long periods without a host. Disease development is favored by temperatures in excess of 27°C (Harverson and Rush, 1998). Some isolates are specific only to sugar beet while others can cause disease in other plants in the family Chenopodiaceae, such as spinach (_Spinacia oleracea_) and red root pigweed (_Amaranthus retroflexus_).
This disease is best controlled by the use of resistant germplasm and practices that minimize moisture stress. Early planting that allows plant development before conditions favor infection, crop rotation with non-host crops and control of weeds within the family Chenopodiaceae are also thought to be effective management practices.

**PYTHIUM ROOT ROT**

*Pythium root rot* caused by *Pythium aphanidermatum* (Edson) Fitzp has been reported in the states of Arizona, California, Colorado and Texas in the U.S. and in Canada, Austria and Iran. *P. deliense* Meurs, has been reported to cause a root rot with different symptoms in Arizona and Texas (R u s h, 1987). Root rot caused by *P. aphanidermatum* is characterized by wilting and a watery dark-brown to black rot of petioles and the interior portion of the taproot. On the exterior of the root lesions are dark colored and irregular in shape and maybe similar to those caused by *Rhizoctonia crown and root rot*. Infected roots often have a “rubbery feel”. Root rot caused by *P. deliense* is characterized by a marbled brown to black root rot that progresses upward in the taproot from infections of secondary roots.

These *Pythium* species survive in the soil as oospores (17—19 μm). When conditions are favorable, the oospores germinate directly or produce zoospores. Favorable conditions for infection and disease development are soil temperatures of 27°C or greater for at least 12 hours and wet soil conditions (0 to –0.1 bar). Epiphytotics have been associated with the fore-mentioned environmental conditions and alkaline soils with high levels of soluble salts and exchangeable sodium (H i n e and R u p p l e, 1969; v o n B r e t z e l et al., 1988).

Management of this disease is best accomplished by avoiding practices that promote prolonged periods of high soil moisture. The use of hymexazole and metalaxyl seed treatments or metalaxyl soil treatments may be helpful.

**PHYTOPHTHORA ROOT ROT**

*Phytophthora root rot* caused by *Phytophthora drechsleri* Tucker has been observed in the states of California, Colorado, Idaho, Montana, Oregon and Utah in the U.S and in Iran. In England, *P. megasperma* has been reported to cause as similar root rot. In nearly every report beets are exposed to excessive soil moisture when temperatures are high (28—32°C). This member of the *Oomycota* survives in the soil as chlamydospores (7—15 μm) and oospores (24—36 μm). Oospores germinate to form sporangia which release zoospores that infect plants. Symptoms include wilting and a wet rot at the base of the taproot that eventually extends upward toward the crown. There is a sharp margin between rotted and healthy tissues. Wilted plants may recover at night in the early stages of this disease. This disease often occurs when wilted
beets are irrigated during hot weather. Disease progress will slow markedly when soil temperatures cool.

Management of this disease is best accomplished by avoiding practices that promote prolonged periods of high soil moisture (Schneider and Whitney, 1986).

RHIZOPUS ROOT ROT

*Rhizopus root rot* caused by *Rhizopus stolonifer* and *R. arrhizus* has been reported as occurring in Arizona, California, Wyoming and Colorado in the U. S. and in Canada (Alberta and Ontario) and Italy, Iran, France and many countries that made up the former USSR. Like other root rot diseases, the first symptom is wilting of the foliage. In this case the foliage rapidly wilt and becomes dry and brittle, collapsing on the crown similar in appearance to plants affected by *Rhizoctonia crown and root rot*. Initially, gray to brown lesions are seen on the taproot. These lesions gradually spread downward from the crown and eventually are covered with a white mycelial growth that later takes on a dark appearance as sporangia develop. Internal decay is brown and spongy in appearance. In advanced stages there are cavities in the decayed area filled with a fluid with the smell of vinegar. Secondary roots are generally unaffected.

Both *Rhizopus* species are considered to be common saprophytes on organic matter and only weak parasites on sugar beet. Sporangia are airborne and infect sugar beet only when the beet is stressed by excessive soil moisture, mechanical damage to the crown or insect damage. High temperatures (30—40°C) are required for infection and disease development by *R. arrhizus* while low temperatures (14—16°C) favor *R. stolonifer*.

This disease is best controlled by avoiding excessive moisture and reducing mechanical damage or insect damage to the crown. Implementation of techniques that favor healthy, rapid plant growth will be helpful in reducing damage (Schneider and Whitney, 1986).

SOUTHERN SCLEROTIUM ROOT ROT

*Southern Sclerotium Root Rot* caused by the fungus, *Sclerotium rolfsii*, which has been reported as occurring in the southern and southwestern U. S. and in Argentina, Bangladesh, Brazil, Czechoslovakia, Egypt, Italy, India, Israel, Japan, Korea, Morocco, Pakistan, Spain, Taiwan and Uruguay. This fungus is found worldwide and will like cause root rot of sugar beet wherever frozen soil does not occur in the winter. The fungus survives in unfrozen soils as sclerotia and anything that moves infested soil will spread the pathogen. The fungus has a host range of more than 200 species. Disease development is favored by moist soils and temperatures in the 25—30°C range (Schneider and Whitney, 1986).
Symptoms initially include unthrifty top growth and wilting. Infected roots are covered by white cottony mycelium and masses of white tan-orange to brown sclerotia. Internal decay is a watery soft rot.

This is a difficult disease to manage because of the wide host range and pervasive presence of sclerotia in tropical to subtropical areas. Rotations that include less susceptible hosts such as wheat, barley, corn, alfalfa or asparagus will reduce inoculum potential. Application of adequate nitrogen and other nutrients will provide vigorous growth that will reduce losses (Leach and Davey, 1942). While resistance is known in breeding lines no resistant cultivars are known to exist. Use of soil applications of carboxin, chloroneb, PCNB and some triazole class fungicides have provided control in sugar beets and other crops such as peanut, although there are no registered uses for these fungicides on sugar beet in the U.S.

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производној области, у великој мери зависи од температуре, земљишних фактора као што су влага и соли у земљишту, производне прaksi и плодореда. Многи од ових патогена проузрокују губитке и после вађења репе, у складиштима. Рад ће се усмерити на распрострањеност ових патогена, идентификацију обољења и циклус развоја болести, епидемиологију и интегралне мере сузбијања. Интегралне мере сузбијања ових патогена укључују сорте орнаментације на болести, незарађено семе, избегавање стреса, агрохемијске мере као што су наводњавање и примена фунгицида. Биће размотрена примена азоксистробина за сузбијање ризоктониозне тругаче главе и корена репе у односу на методе примене и дозе, времена примене и у поређењу са другим фунгицидима.