NEW SOYBEAN DISEASES FOR WISCONSIN: SUDDEN DEATH SYNDROME AND STEM CANKER

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The pathogens and insect pests of soybean have steadily changed in the past 30 years. Many pathogens are first detected in the southern USA and steadily make their way north. Soybean cyst nematode and the bean leaf beetle/*Bean pod mottle virus* complex are classic examples of this south to north migration of pathogens and pests. Sudden death syndrome (SDS) and stem canker are diseases that have been active to the south of Wisconsin, but appear to have become established in Wisconsin. Symptoms of both diseases were observed in 2003 and causal pathogens were isolated confirming the diagnosis based on symptoms. The following text is a general review of both diseases.

Sudden Death Syndrome

Sudden death syndrome (SDS) was observed in commercial soybean fields for the first time in Wisconsin in 2003. Sudden death syndrome is caused by the soilborne fungus *Fusarium solani* f. sp. *glycines*. The SDS pathogen is a specialized strain of *F. solani*, a common soil inhabitant. The SDS pathogen also has a narrow host range consisting of soybean, green bean, and lima bean. The disease has first observed in Arkansas in 1970 and has steadily progressed into the upper Midwest.

Sudden death syndrome is more severe in fields with high yield potential. Symptoms of SDS generally appear during the reproductive stages, but onset varies with geographic location. Symptoms can appear as early as R_4-R_6 in the northern part of its range. Initial symptoms are interveinal chlorotic spots on leaves that expand into interveinal chlorotic streaks and become necrotic. In severely affected foliage, only the leaf veins remain green. There is no obvious wilting of foliage, but symptomatic leaflets can abscise and leave only petioles attached to stems. Root systems of diseased plants are smaller and exhibit varying degrees of necrosis. Vascular tissue shows light brown discoloration that can extend several nodes up the stem, but pith tissue remains white. In infested fields, symptoms appear in somewhat circular or elongated patches, which can coalesce into large, irregular areas of diseased plants.

Yield losses can be sporadic because SDS is not a severe problem every year or in every part of an affected field. Sudden death syndrome affects yield by reducing both seed size and seed number. Smaller seed size results from leaf area reductions due to foliar necrosis and premature defoliation, whereas lower seed number results from flower and pod abortion.

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Both soil moisture and temperature can affect disease severity, although the exact nature of these relationships remains unclear. Early reports indicated that symptoms appeared following passage of a weather front that brought cooler temperatures and rain. Symptoms are associated cool, wet weather near the time of flowering with symptom development. Field observations suggested that symptoms generally are less severe in dry years and more severe in wet years or irrigated fields. Differences in SDS severity vary among years and are correlated strongly with early season (June) rainfall and less so with late season (August) rainfall. Although wet soils favor infection, once infected, plants express greater severity of foliar symptoms as soil moisture decreases.

Several reports addressed the role of soil fertility in development of SDS. Greater severity of SDS is associated with increased levels of available soil P, soluble salts, organic matter, and exchangeable Na, Ca, and Mg and greater available K. Soil compaction also is associated with increase disease severity. In areas where compaction is problematic, subsoiling can increase porosity, decrease water-holding capacity, and reduce disease severity substantially. Severity of SDS may decrease as soil pH is lowered from 7.7 to 5.5.

The soybean cyst nematode (SCN) and SDS frequently occur together in the same field. Greenhouse and field studies have shown that foliar symptoms of SDS developed more rapidly and were more severe when SCN also parasitized soybean roots. Soybean varieties resistant to SCN frequently show mild symptoms of SDS compared to SCN susceptible varieties. Collectively, most pathologists agree that SCN may increase severity of SDS but is not required for disease development. The SDS pathogen is reported to colonize cysts of SCN and movement of cysts coincides with movement of the SDS pathogen.

The most effective approach to SDS control is use of resistant cultivars. Reaction of cultivars in the field is expressed as partial resistance. Because cultivar reactions to SDS can be mitigated by SCN, resistance to both pathogens is desirable. Most major seed companies are actively breeding for resistance to SDS and partially resistant varieties should be available in the near future.

Several cultural practices provide reasonable control options. Symptoms often are less severe in delayed plantings and in early-maturing cultivars. Both can delay SDS development until later reproductive stages, which may reduce yield loss. Severity of SDS may be greater under no-till than conventional tillage. Control of SDS in various crop rotation studies has been inconsistent.

Stem Canker

Stem canker has been widely recognized as an important soybean disease, but recently has been divided into northern stem canker and southern stem canker based on two causal agents. Northern stem canker was first reported in the late 1940s in Iowa, and by the 1950s, the disease was observed throughout the Midwestern USA and Ontario, Canada. Southern stem canker was reported in the south in 1973, and by 1984, had been

detected in all southern states. Southern stem canker has recently been reported in the North Central States.

Northern stem canker and southern stem canker are caused by *Diaporthe phaseolorum* var. *caulivora* and *Diaporthe phaseolorum* var. *meridionalis*, respectively. The host range of both pathogens has not been study extensively, however, over 16 weed species are known to harbor *D. phaseolorum* var. *meridionalis.*

Initial expression of symptoms occurs during the early reproductive stages, with the development of a small, reddish-brown superficial stem lesion at the base of branches or petioles. The lesion is first observable in the leaf scar after the petiole has fallen from one of eight nodes. The lesion elongates and becomes dark brown or black, sunken in appearance and often girdles the stem. Interveinal chlorosis and necrosis are expressed in the leaves and is soon followed by plant death. Above and below the canker, green tissue is present and the leaves on the dead plant wither, but remain attached. A top dieback can occur and results in a characteristic shepherd's crook curling of the terminal bud.

Yield losses have been reported to be as high as 50 to 80%. Yield reductions resulting from stem canker have increased dramatically over the past 2 yr. Estimated yield losses to stem canker were 89.2 and 59.5 thousand metric tons in 1999 and 2000, respectively.

Both pathogens overwinter in colonized stems and infected seed. Stem canker is effectively managed by the combination of planting resistant cultivars and reducing infested soybean residue on the soil surface. Deep plowing can reduce crop residue prior to planting a soybean crop. The benefits of crop rotation to reduce stem canker have not been demonstrated in production fields. Also, a double-cropping system of wheat and soybean may result in greater disease than monocropping soybean. Delayed planting can reduce the incidence and severity of stem canker. Seed that are to be used for planting should not be harvested from fields with a history of stem canker. Seed infection by *D. phaseolorum* var. *caulivora* can be as high as 10 to 20%; however, seed transmission of *D. phaseolorum* var. *meridionalis* generally does not exceed 1%. Fungicides applied to seed greatly reduce stem canker but will not completely eliminate the pathogen. Foliar fungicides can be effective when applied during vegetative stages. However, results are inconsistent, and in most cases foliar fungicides would not be an economical management strategy.

Resources

Plant Health Initiative; North Central Soybean Research Program.
http://www.planthealth.info/

Soybean Plant Health website, University of Wisconsin-Madison.
http://www.plantpath.wisc.edu/soyhealth/