SUDDEN DEATH SYNDROME IN SOYBEANS

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Sudden death syndrome (SDS) is a disease of soybean that is creeping into the vocabulary of more Midwestern farmers. It is of increasing concern because it continues to spread northward in this important soybean production area, and it is a disease that often strikes portions of fields with the highest yield potential. The disease is a root rot problem, but takes its name from the rapid progress of foliar symptoms on a susceptible cultivar under favorable environmental conditions.

Sudden death syndrome was first observed in Arkansas in 1971, but caused no concern until 1982 when the disease caused an estimated 25% yield reduction in 5 to 10% of Arkansas’ soybean acreage. In Indiana, the disease was first identified in 1985 in Posey County. Since 1985, the disease has been identified in many areas of the state. Sudden death syndrome is less damaging north of Vincennes than south in most years. The disease is now known to occur in areas of Illinois, Indiana, Iowa, Kansas, Kentucky, Mississippi, Missouri, Ohio, and Tennessee.

It is difficult to assess the total crop loss caused by this disease, but yield losses of up to 80% have been reported from affected areas of a field. During 1985, and again in 1987, 40% yield losses were reported from individual fields in southwestern Indiana. Yield losses are dependent upon cultivar, weather conditions, time of planting, time of disease onset, and possibly other factors. Yield reduction due to this disease occurs through premature plant death, pod abortion, lack of pod fill, and low test weight.

SYMPTOMS OF SUDDEN DEATH SYNDROME

The foliar symptoms of sudden death syndrome may appear any time from bloom through pod fill, although symptoms usually appear between the R3 and R6 growth stages. The time of symptom expression appears to depend on weather conditions, maturity group and other characteristics of the cultivar, and general vigor of plant growth. The disease may affect individual plants, small groups of plants, or plants in circular to oblong...
patches in a field. One to many patches may occur in a field, and patches may coalesce to form large, irregular areas. Sudden death syndrome generally occurs in fields that appear to have high yield potential. Prior to the appearance of foliar symptoms, there are no obvious reductions in plant height or other growth parameters. Sudden death syndrome frequently appears in those areas of a field that tend to have a slightly higher water holding capacity, and the disease is frequently more prevalent in irrigated than in non-irrigated fields. The disease is often more severe in the earliest-planted fields.

The first outwardly visible symptom of sudden death syndrome is the appearance of small, yellowish, interveinal blotches (chlorosis) in leaves, generally in the middle to the upper part of the canopy. The interveinal blotches quickly increase in size and number, and the tissue within the blotches becomes brown and dies. The leaf veins, petioles, and stems remain green for some time after most of the interveinal leaf tissue has died. As the disease progresses, entire plants may become affected and leaf blades drop, leaving erect, barren, somewhat green petioles attached to the stems. The foliar symptoms of sudden death syndrome may be confused with some other diseases, such as brown stem rot or stem canker, or other factors, and these foliar symptoms alone are not diagnostic for sudden death syndrome. The root and lower stem tissues of plants exhibiting these symptoms must be closely examined for a field diagnosis of sudden death syndrome.

The root systems of plants showing early stages of interveinal chlorosis may appear normal or nearly so (somewhat reduced lateral roots), but invasion and discoloration of the taproot occur at the same time as foliar symptom development. As the foliar symptoms progress, light gray to brown streaks develop in the internal taproot tissues. There is no discoloration of the vascular tissues in the stem at this time. With time, the taproot becomes progressively discolored, the lateral roots and nitrogen fixing nodules begin to deteriorate, and the vascular tissues in the lower stem become gray to brown. Stem symptoms may extend up several nodes. Discoloration of the vascular tissues of the lower stem may involve from half to three-fourths or more of the stem cross section in severely affected plants. Pith tissues of plants with sudden death syndrome remain normal and white. In addition to normal pith tissues, the epidermal tissues of affected plants also appear normal. In contrast to these symptoms, vascular tissues remain normal in appearance but pith tissues are brown in plants with brown stem rot. Plants with stem canker show definite sunken cankers on the stems.

The length of time from symptom onset to plant death appears to be from 10 days to 3 weeks, although up to 6 weeks has been reported. Dry conditions appear to reduce taproot colonization and may slow or stop symptom development. Plants growing under high temperatures or drought rarely develop symptoms of sudden death syndrome.

CAUSE OF SUDDEN DEATH SYNDROME

Greenhouse and laboratory studies have demonstrated that a specific strain of the soilborne fungus *Fusarium solani* is a causal agent of sudden death syndrome. *Fusarium solani* can be found in nearly every soybean field, and it regularly infects the lateral roots of soybean plants, but it is not an aggressive pathogen in most situations. Two distinct strains of *Fusarium solani* infect soybean
and cause root rot. The specific strain of the fungus causing sudden death syndrome, *Fusarium solani* f. sp. *glycines* (sometimes called “form A” or the “blue strain”), produces toxins that are translocated from the tap root to the foliage. All the toxins involved have not been identified, nor have the conditions under which they may be produced. Fortunately, the toxin-producing strain is not as widespread as the ordinary soybean strain (sometimes called “form B”) of *F. solani*. Research at Purdue established that lateral root infections by the blue strain occur early in the growing season, but symptoms do not appear until several weeks later when soybean is in the reproductive growth stages and taproot colonization is enhanced by abundant soil moisture.

**SOYBEAN CYST NEMATODE AND SUDDEN DEATH SYNDROME**

The role of the soybean cyst nematode in the development of sudden death syndrome is not clearly defined. Infection by the nematode is not necessary for the development of sudden death syndrome, but it exacerbates the disease. There is no correlation between the size of cyst nematode populations in the soil and the development of sudden death syndrome in a given field. Sudden death syndrome does occur in soybean fields where the cyst nematode does not occur, but almost all fields in areas where sudden death syndrome is a major problem also support a high population of soybean cyst nematodes. Research by Ken Roy in Mississippi and Scott Abney in Indiana indicates that the blue strain of *F. solani* can colonize and overwinter in the cysts of the soybean cyst nematode. While the nematode is not necessary for the fungus to infect soybean roots, the cysts may provide an important means of survival for the fungus in the soil. This may partially explain why the development of sudden death syndrome paralleled the earlier spread of soybean cyst nematode in the Midwest.

**FACTORS AFFECTING SUDDEN DEATH SYNDROME**

Cultivars differ in symptom expression and yield when affected by sudden death syndrome. Cultivar comparisons frequently differ from location to location within a state, between states, and from year to year, making it difficult to identify cultivars with reliable resistance. Resistance found in Ripley, Callahan 3484, and P.I. 520733 has been stable in Indiana during the 1990s. Most researchers feel that the identification of sources of resistance, which can then be bred into adapted cultivars, holds great promise for the control of this disease.

Early reports indicated that symptoms of sudden death syndrome appeared shortly after the passage of a major weather front that would bring rainfall and cooler temperatures at or near the flowering stage. More recent observations suggest that adequate to surplus soil moisture during the pre-bloom to bloom stage is the most important predisposing factor, especially when there is heavy rain subsequently sometime during the early reproductive stages of development. Rainfall patterns may explain why sudden death syndrome is generally more severe in early-planted fields than in later-planted fields. While sudden death syndrome has been observed in double-crop soybean, it is generally less prevalent and less severe compared to what is seen in crops planted earlier in the season.

The effects of crop rotation on sudden death syndrome have not been clearly identified. Research suggests that sudden death syndrome is a greater problem in continuous soybean production than in a corn-soybean rotation. However, Don Hershman of the University of Kentucky has observed severe sudden death syndrome in a soybean crop that followed 5 years of corn. The disease is less severe in a wheat-corn-soybean rotation than in a corn-soybean rotation. Premature dying due to sudden death syndrome has consistently been more severe in continuous soybean production with no-till planting than with conventional or chisel tillage.

**MANAGEMENT OF SUDDEN DEATH SYNDROME**

The following practices should be beneficial in reducing the effects of sudden death syndrome.

1. **Cultivar selection.** Cultivars differ in their response to sudden death syndrome. In those areas where the disease occurs or is suspected, select cultivars that performed well for the local area in those years when sudden death syndrome was severe. Two of the original sources of resistance to the soybean cyst nematode are very susceptible to sudden death syndrome, but most adapted cultivars with resistance to the cyst nematode are more resistant to sudden death syndrome than are cultivars that are susceptible to the nematode.

2. **Crop rotation.** While the effect of crop rotation on sudden death syndrome is not fully known, this practice aids in the control of the soybean cyst nematode and other soil-borne diseases. In Indiana, sudden death syndrome is usually more severe when soybean is grown continuously in a field compared to corn-soybean or corn-soybean-wheat rotations.
3. Tillage practices. Indiana research suggests that sudden death syndrome is more prevalent in no-till than in chisel or conventional tillage. This may be an indirect effect of soil moisture at the time of planting.

4. Planting date. Fields with a history of severe sudden death syndrome should be planted toward the end of the soybean planting season. The disease frequently is more severe in the earliest-planted fields.

5. Nematicides do not control sudden death syndrome. Soil fumigation with methyl bromide-chloropicrin controls sudden death syndrome, but is not economically feasible. Soil fumigation with Vapam has reduced the severity of sudden death syndrome in Indiana, but this treatment is not economical.

6. Maintain plant health through management practices that reduce competition from weeds, and damage from insects or other diseases, and stress from soil compaction or other soil factors. Research has shown that both foliar fungicides and foliar nutrient treatments during pre-bloom enhanced plant health and were associated with reduced severity of sudden death syndrome. This research indicates that general plant health is important in reducing yield losses from sudden death syndrome.

7. The blue strain of *F. solani* is not seed-borne, and seed treatment fungicides are not a control measure for sudden death syndrome.