Stop SDS: Prioritizing management approaches that best fit your fields
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Introduction

Sudden death syndrome (SDS) is a mid- to late-season, soil borne disease that occurs primarily in soybean fields with high yield potential. First observed in Arkansas in 1971, SDS now causes significant losses in Midwestern states such as Illinois and Indiana and is considered the most important fungal disease in causing yield loss in the North Central Region. The disease was first noticed in Iowa in 1993. Now the disease has been found from border to border in Iowa. It occurs annually in eastern Iowa and epidemics occur frequently in recent years. Yield losses due to SDS vary widely. SDS may cause premature defoliation in the fall resulting in nearly total yield loss in all or portions of the severely affected fields, depending on the weather in the growing season. The disease causes foliar symptoms, resulting in pod abortion and yield loss up to 70% in severe cases.

This summer the SDS was wide spread in east half of Iowa, the highest prevalence ever since it was first reported in Iowa in 1993. In this paper, we discuss the infection biology of SDS disease and review when and why a control measure fails. Also, we will discuss on how to use infection biology information to select control measures that fits best for your fields. To manage the disease effectively in your fields, you will need to understand the following three aspects of this disease and check if your SDS infested fields meet any of them.

Effective time to infect soybean

Various studies have shown that the fungus attack soybean as long as seed germinated and recent studies suggest that infection in early seedling stage is critical to SDS foliar symptom production. Our study two years ago found that the fungus can infect soybean plants as early as seed germinated under greenhouse conditions. Observations were that the earlier the germinated seedling infected, the more severe the foliar symptoms observed. In a meeting late last year, researchers at the University of Illinois reported that the fungus was detected in soybean plants as early as the V1 growth stage in SDS-infested fields, consistent with results from the ISU greenhouse study. Infections early at seedling stages are effective for fungus to penetrate into vascular tissues in soybean root. Colonization by the fungus in vascular tissues (mainly xylem) is critical to the consistency of foliar symptom expression.

There is a specific relationship between the time of infection and colonization of fungus inside or outside root system. Fungus spores germinate in cool temperatures (20-22C), and penetrate through cortex tissues of the soybean radicles (root) according to an Iowa State University study. The fungus then colonizes xylem tissues of taproots in infected plants. Since the xylem is the soybean plant’s pathway for upward movement of water and nutrients, colonization in the xylem system is critical for the fungal toxin to move from the taproot to the foliar regions. When toxins produced in the infected plant are pumped to foliar parts, initial symptoms are produced as
scattered yellow or white spots between leaf veins.

Early infection at seeding stage will lead to colonization in xylem system and consequently lead to transportation of toxins to upper portion of plants foliar symptoms expression of SDS. When plants are in advance seedling stage or near reproductive stage, the vascular system is well developed and become harder for fungus to penetrate into vascular tissues. Infection after V1 growth stage should have less impact on foliar symptom development or defoliation.

The early the infection is, the higher the possibility for SDS fungus to colonize in vascular system, and the higher the chance to cause severe defoliation later in summer. If infection occurs when root formation is complete, the fungus may just stay in phloem tissues (ineffective zone See figure 1) without further entering into xylem tissue (effective zone See figure 1) which would not be able to move toxin upward, consequently, no foliar symptoms can develop.

**Figure 1.** Effective infection zone (A) and ineffective infection zone (B) for foliar symptom expression on soybean root. Attacks by soybean cyst nematode could help SDS fungus to enter the effective zone.

**Conditions for infection**

Cool and wet soil is essential for the successful infection of SDS fungus. Saturated soil moisture is needed for infection to occur. The cooler soil temperature earlier in the season is critical for larger amount infection. When soil temperature warms up in June, the fungus will not be effective to attack soybean plants. In a planting season with a lot of rain like this year, the soil temperature warms up late and soybeans planted after mid May still have high risk of being infected. When the window for disease to attach soybean is long, the disease risk increases.
Figure 2. Temperature relationship to the infection of soybean sudden death syndrome infection. The cooler soil temperature earlier in the season is critical for the fungus to infect soybean root.

**Survival of the fungus**

SDS is caused by strains of *Fusarium solani* that produce bluish pigments in culture. The fungus can produce abundant spores on roots of infected soybean plants. In the absence of soybean plants, the fungus survives in the soil as chlamydospores and in crop residue as mycelia (Rupe, 1989). SDS fungus is known so far only infects soybean plants in the North Central soybean production region; no other hosts in the absence of soybean have been reported.

Many observations suggest that corn may be a good host in the absence of soybean, and therefore, rotation with corn or continuing corn production may increase the risk of SDS. Over years, growers report severe outbreaks of SDS in fields just out of 5- or 8-years continuing corn. Such a situation may explain why after SDS was first reported in Arkansas; the disease is more severe problems in northern soybean productions states where corn-soybean is a major production system.

Research is underway to determine the effect of corn on the survival of SDS fungus. It is yet to learn the mechanisms of SDS fungus survival in the corn residues. However, it is common practice for plant pathologists to use grain kernels such as corn grain or sorghum grain are good medium to increase SDS fungus in laboratory setting to infest research plots for SDS experiments. Unharvested corn kernels are likely a source of foot for the increase of SDS fungus in crop fields and the amount of un harvested corn kernels are much greater than the amount of inoculated grains that plant pathologists in field inoculation. Further, numerous studies in corn diseases have shown that corn stalks have been reported as good residues for many Fusarium fungi. It is likely that corn residue is an equal or better harbor than soybean for SDS survival over a long term. Therefore, management of corn residue may provide a solution to manage the risk of SDS during soybean production season.
**Prioritize your SDS management**

Currently most effective management options for soybean producers in Iowa are: 1) the use of resistant varieties, mostly for central and southern Iowa where MG3 or later are planted, 2), delayed planting, 3) plant with non soybean crops. The first two practices have been used for growers effectively with limited success because 1) resistance is mostly available for varieties in later maturity groups and 2) delayed planting is conflict with agronomic recommendation that is to promote early planting to achieve maximum yield potential. Delayed planting in northern Iowa could result in yield penalties. In a season with prolonged rain during planting, soil temperatures are wet and cool even in later May, in which later planting will not work. Like this season, many later planted soybeans still have high level of SDS infection. For the third option, research from Indian indicates rotation with wheat may help to reduce SDS. Rotation with corn seems not working in Iowa. There is no information on how long the pathogen can maintain a high population in the absence of a soybean crop.

If you continue to have significant SDS problems in the same field and production management measures have been made, consider to reprioritize and put SDS management in top priority. A change in production measure, such as tillage or variety selection, may reduce the disease risk. If you have taken SDS management measure but did not work, the measure may not fit to your field condition. You should prioritize the management measures according to the conditions in your fields. Following SDS management options should be taken after finding out the cause why previous measurement dose not work.

**Plant resistance varieties**

There are not many choices for varieties in maturity groups 2 and 1 because the disease used to be a southern disease and breeding efforts started with later maturity groups. For group 3 varieties, one can find good one with consistent results. There are two types of resistance, resistance to toxins produced by fungus and resistance to infection/penetration by the fungus. Theoretically, the second type of disease resistance could break down if SCN population is high in a field when resistance to SDS is limited to resistance to root infection.

**Delay planting to minimize the risk**

As discussed above, an early planting in cool soil increases xylem colonization by SDS fungus. The younger the soybean plants, the more likely the pathogen will penetrate into xylem tissues of taproots. If the fungus fails to penetrate the xylem during the infection, no foliar symptoms would occur. Therefore, the earlier the SDS fungus attacks soybean plants, the higher the disease risk is. Since the fungus attacks soybean effectively in cool soil and warm soil reduces the fungal activities, early-planted soybeans in cool soils are more likely to be colonized by the SDS fungus inside its tap root. Later in the fall, more foliar symptoms follow. Knowing this, one should avoid planting soybeans in cool soil if SDS has been a production problem in his fields.

**Control SCN population**

The information of xylem colonization also reminds us to consider soybean cyst nematodes (SCN) in your SDS management because the nematodes may help the SDS fungus penetrate into xylem tissue. It has been shown consistently that SCN can enhance SDS infestation. If SCN population is high, soybeans which are not planted early would also have high risk to SDS because SCN can support SDS fungus to enter vascular system of soybean. The higher the SCN
population is, the greater the chance for SDS pathogen to enter xylem system, or higher risk of producing defoliation. One therefore should prioritize your management between the two pests as management measure for the two pests may be different in terms of rotation and tillage practice.

Management of SCN can be achieved by rotation with non-host crops to SCN or by planting soybean cultivar resistant to both SCN and SDS. Most varieties do not have good resistance to both crops. Therefore, in season, not favorable to SDS, one can plant SCN resistant variety to low SCN population.

**Avoid no-till**

Three things in no till increase SDS. Cooler and wet soil temperature in no-till field compared with conventional fields favor SDS infections. If your no-till field does not have wet soil conditions, this measure is not your option and the SDS problem may be a results of other factors, such as early planting or high SCN population. The second aspect of no-till has higher disease is the soil vertical profile where SDS survive remains unchanged after rotation. SDS fungus from infected roots of previous soybean crop will not be touched in a no till field. When soybean seeds are planted in the same root zone loaded with SDS fungus, the contact probability for the fungus to soybean root increased compared with previous season. When deep tillage is applied, the soybean seeds are physically separated from SDS fungal population and therefore reduce the disease risk. Therefore, if SDS has been severe at least two crops in a row, one should consider to change no-till to till to minimize disease risk.

**References**


