Strategies for management of soybean sudden death syndrome and white mold
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Introduction
Soybean sudden death syndrome (SDS) caused by Fusarium solani f. sp. glycines and soybean white mold caused Sclerotinia sclerotiorum were two of the most prevalent diseases in 2006. The two diseases have become even year production problems for some producers. This workshop will review results of the latest studies from the past two years and discuss management strategies for the two diseases.

The SDS has been recognized as a major disease of soybean (Glycine max (L.) Merrill) in the southern United States for nearly three decades. In 1995 Scherm and Yang using computer model predicted that the disease would become an important disease in North Central region. In the past 10 years SDS has become prevalent and severe in northern soybean production areas and reduces soybean yield significantly. SDS is an early to late season disease of soybean. This year SDS has been observed in most parts of Iowa towards mid September. There is concern among producers in the north central region, due to noticeable expansion in geographical array of SDS, wide spread damage in the field and the associated potential yield losses. Yield loss due to SDS in the top 10 soybean producing countries varies from $2 \times 10^3$ in Canada to $9 \times 10^5$ metric tons in the United States (Wrather, et al., 2001).

The SDS generally occur in fields where yield potentials are high and yield losses up to 50% have been observed in Arkansas (Rupe, et al 1989). The SDS was first found in Iowa, in experimental plots in 1993 (Yang and Rizvi 1994). However, SDS was first noticed in Arkansas in 1971 as disease of unknown cause. Later it was reported from Tennessee, Missouri, and Mississippi in 1984, from Illinois, Kentucky, Kansas and Indiana in 1985 (Rupe et al 1989, Wylie, T.D. 1988) and an epidemic (46% of the soybean fields) in 1993 in east-central Illinois (Hartman, et al 1995). The SDS has been reported in 2004 from Nebraska (Ziems, et al 2006), and from Argentina (Scandiani, et al 2004). The yield damage caused by SDS and white mold are comparable now. Like SDS, soybean white mold also is more prevalent in fields with high yielding potential.

What are sudden death syndrome and white mold diseases?
Both are soil borne disease, the SDS causes characteristic foliar symptoms; leaves on infected soybean plants initially show scattered yellow or white spots between leaf veins (Figure 2) at an early stage to towards flowering or pod development stages. These spots eventually coalesce to form brown streaks between the veins or interveinal necrosis and only the middle vein and major lateral veins remain green (Figure 2). In several cases symptoms at flowering are chlorotic, mottling or mosaic on the upper leaves. Within a few days, chlorotic blotches develop on leaves, which rapidly become necrotic and coalesce to form interveinal necrotic streaks (Scherm and Yang 1996). Diseased SDS plants have pigmented/deteriorated taproots and lateral roots (Figure 2). The root cortex is light-gray to brown and the discoloration may extend up into the basal stem. Sometimes bluish fungal colonies can be seen on the roots if soil moisture is high.
**Figure 2. Soybean sudden death syndrome caused by Fusarium solani f. sp. glycines**

**Disease cycle of sudden death syndrome**

The macro and micro-conidia and hyphae survive in the soil, under suitable soil conditions conidia germinate, the fungus penetrates through cortex tissues of soybean plants. The hyphae remain in xylem tissues of infected plants. Toxins produced in the infected plant pumped to foliar parts and initial symptoms produced are scattered yellow or white spots between leaf veins. As disease progresses spots eventually coalesce to form brown streaks between the veins or interveinal necrosis and only the middle vein and major lateral veins remain green. Plants with SDS symptoms do produce pigmented basal stem and tap root & the fungus remains in the cortex tissue.

**Figure 3. Soybean white mold caused by Sclerotinia sclerotiorum**

Most detectable symptoms of white mold in the field include wilting, leaves turn to pale tan to brown and they remain on the stem in the initial infestation, as infestation advances leaves drop from the stem and eventually plant dies (Figure 3). White mold is usually observed around the end
of July and into August. This fungus causing white mold is easily recognized by the white fluffy mycelium growing on the outside of infected plant stems. Diseased plants will wilt, drop their leaves, and turn white or pale tan. Fungus growing on the outside of the stem will produce black sclerotia, which are loosely attached to the stems. White mold infection is most easily identified before adjacent healthy plants have dropped their leaves. The external fungal growth quickly dries down when removed from the protection of the plant canopy, and external sclerotia will usually drop off at this time. The stems of diseased plants will still appear bleached compared to plants senescing normally, and sclerotia will be present in the stem cavities on close inspection.

Flowering stage is a critical period for the initiation of white fungus, as it enters soybean plants from senescing flowers. With the high level of pathogens built up in 2004, weather conditions during the growing season this year favored major disease outbreak of this disease. The white mold fungus requires cool air temperature and moist soil to produce spores. A period of cool, wet conditions during flowering will promote an outbreak.

Figure 4. Disease cycle of soybean sudden death syndrome.
**Disease cycle of white mold**

The sclerotia, which are formed both inside and outside the host plant, serve as the survival structure. During harvest the sclerotia are combined with the seed or returned to the soil surface with crop residues. Burying the sclerotia in soil appears to enhance their survival ability. In the spring and summer the sclerotia will germinate either directly by forming mycelia or by forming apothecia. Sclerotia will produce apothecia if they are within an inch of the soil surface. The apothecia produce large numbers of ascospores, which are subsequently spread in wind and splashing rain. Once the ascospores germinate, the developing fungus requires a nutrient source prior to infecting plants. On soybeans, the flowers serve as a food source, and subsequently infections occur on the stem near a node where the mycelium colonized dead flowers. Sclerotia developing on diseased soybean plants are returned to the soil during harvest. Sclerotia found in harvested seed can contaminate new fields if seed lots have not been properly cleaned to remove sclerotia prior to planting.

![Diagram of disease cycle of soybean white mold](image)

**Figure 5. Disease cycle of soybean white mold.**

**Management strategies to control soybean sudden death syndrome**

**Plant Resistance**

There are reports of various techniques of screening soybean for SDS resistance. Yang et al. (unpublished) used ground powder of FSG multiplied on steam sterilized oat, mixed with the
greenhouse mixture, and planting test varieties in plastic cups and evaluate for SDS-symptoms 3-4 weeks after planting. Other screening methods include temperature-controlled water bath method (Hashmi et al. 2005), mixing steam sterilized sorghum grains colonized by the *F. solani* f. sp. *glycinum* with greenhouse mixture and planting test varieties; (Huang and Hartman 1998; and Aoki et al. 2005), dipping seedlings in culture filtrate (Huang and Hartman 1998). It is believed that some of these techniques lacked consistency in foliar symptoms expression. Hence, during 2002-2005 at ISU we developed and refined a simple, rapid, repeatable, and quantifiable and season independent resistance screening technique to measure relative tolerance of soybean varieties to SDS. In the process during the period we attempted understanding mechanisms of (a) discoloration of basal stems and taproots of SDS infected plants (Navi and Yang 2003), (b) colonization of the fungus in phloem and or xylem tissues of taproots (Yang and Navi 2003) and (c) germination and penetration by *F. solani* f. sp. *glycinum* in relation to foliar symptoms expression of the disease (Navi and Yang 2004).

**Variety selection**

Currently, variety selection provides the best way to manage the disease, but none of the varieties tested earlier by several workers are disease free. Almost every company has resistance varieties but not all are consistent due to different variety development approaches. Pick up from companies which have longer history of SDS resistance breeding.

**Adjusting planting dates**

In general, SDS incidence is decreased by delayed planting dates and their corresponding warmer, drier soils. In Iowa, fields planted after mid-May will have much less disease than those planted before. Planting in fields previously had SDS increases risk.

**Crop rotation**

The effects of crop rotation, tillage practices, and row spacing on SDS have proven to be inconclusive based on available literature sources and experience of the authors.

**Seed treatment using Bio-fungicide**

Our recent studies using a bio-fungicide (is proposed for EPA review) proved to be effective in inhibiting FSG conidial germination and SDS-infection under controlled environments (Navi and Yang unpublished). It is too early to apply to production; however, the product appears promising.

**Management strategies to control white mold**

White mold management measures are preventative and include the application of chemicals. This means that correctly assessing the risk of this disease helps guide our decisions on chemical controls. The first major outbreak of white mold in Iowa occurred in 2004. Since then, the disease has been severe every other year. There were severe outbreaks in 1996, 1998, 2004, and 2006. Due to a period of dry weather, the disease was not a problem in 2000 and 2002. It appeared that if the weather condition in a growing season has moisture above normal, one could expect a return of this disease.

Keep in mind that the risk of white mold varies from field to field. In general, narrow row spacing soybean fields have a higher risk than wide row soybean fields. Soybean fields planted
early have a higher risk than those planted later. In narrow row or early-planted soybean fields, the canopy closes earlier, which helps the soil retain moisture, a factor favoring white mold mushroom production. River-bottom fields, fields with long hours of shade in the morning, fields applied with manure, and fields with a dense canopy all carry a higher risk of white mold due to the higher moisture content in the soil as well. If the soil is dry, though, the disease should not be a problem.

No single method can entirely eliminate the disease problem; therefore, an integrated management approach might help reduce the disease.

**Row Spacing**

Studies on row spacing consistently show that white mold incidence is higher in soybeans with narrow row width than wide row soybeans. Soil moisture and humidity are higher and temperatures are lower in narrow rows soybean - good conditions for apothecia production and spore infection. However, when weather conditions are favorable for white mold, wide rows (30 inches) can have similar levels of disease as narrow rows, especially with highly susceptible varieties. Avoid using row spacing of less than 15 inches in fields where white mold has caused significant yield reduction in the past.

**Variety selection**

Varietals selection should be the first line of protection to combat white mold. Cultivars that are short and do not tend to lodge. Use of tolerant varieties has been proven effective in managing white mold. In variety tests, the level of disease may vary by location and year because of environmental conditions. Choose varieties that have consistent tolerance rankings across locations and years. Often, tolerance is measured by the percent of plants killed. Studies show that with about 10 percent of plants killed, yield losses are not measurable. Yield loss becomes significant when the percentage of diseased plants reaches 20 percent or more.

**Chemicals**

Benlate (Benomyl) and Topsin (Thiophanate-methyl) provide control for soybean white mold if applied properly. The fungicides must be applied during flowering stage, 25-50 percent of full bloom for Benlate and 50 percent for Topsin followed by a second spray two weeks later. A total of two sprays 10 to 14 days apart are necessary for chemical control of white mold in Iowa. Topsin is registered for the control of soybean white mold. Benlate is not specifically registered for soybean white mold. However, it is labeled for control of other soybean diseases and dry bean white mold caused by the same fungus. Because of the cost of chemicals, selected application to areas where white mold has been observed in previous crops can prevent disease spread while reducing costs. Post emergence application of cobra has been reported to suppress white mold by growers for years. Cobra’s label has recently been amended to include white mold suppression along with weed control.

**Tillage**

The unique features of the biology of *Sclerotinia* complicate the situation in managing the disease. In a growing season, sclerotia within two inches of the soil surface germinate and release ascospores. Burying infested residues into deep soils by mold-board plow or deep chiseling can prevent the germination of the sclerotia the next year. Therefore, tillage preceding the soybean crop can reduce sclerotial germination. The sclerotia, however, can survive in the soil for up
to seven years. Tillage/cultivation operations in subsequent seasons could uncover sclerotia. Since an apothecium can produce three million airborne spores, it does not take many sclerotia per square foot to cause damage. Use of no-till for non-host crops after a white mold year is recommended. This will promote the germination of sclerotia in non-host crops. Consequently, fewer sclerotia will survive for the following soybean season.

**Crop rotation**

White mold or sclerotinia stem rot also can affect a wide range of field crops, such as edible beans and sunflowers, as well as vegetables, fruits, and ornamentals. Rotation with corn or other non-host crops with no-tillage may result in lower S. sclerotiorum populations as the sclerotia germinate under the non-host crop.

**References**


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