Soybean Sudden Death Syndrome

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Soybean 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 and is considered the most important fungal disease in reducing yield losses in the North Central Region. The disease was first noticed in Iowa in 1993. Yield losses due to SDS vary widely. SDS may cause premature defoliation in the fall while resulting in almost total yield loss in all or portions of severely affected fields, depending on weather in the growing season.

Causal Agent

SDS is caused by strains of soil borne root pathogen Fusarium virguliforme (formerly F. solani f. sp. glycines) that produces bluish pigments in culture. This 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 on crop residue as mycelia. Fusarium virguliforme differs from Fusarium oxysporum, a closely related species that causes soybean root rot and wilt.

Symptoms

Although SDS has been observed in seedlings, symptoms at the seedling stage are rare and resemble those of herbicide injury. Furthermore, the disease symptoms often disappear with the growth of new leaves. In most cases, initial symptom development is associated with the reproductive growth stages of the soybean: the foliar symptoms appear during or after the flowering stage, and severe symptoms develop after pod set. In Iowa, the disease may be noticeable in late July to early August, depending on the planting date.

The first disease symptoms to appear are scattered, interveinal, chlorotic spots or blotches on the leaves (Figure 1), especially on new leaves. The chlorotic areas become necrotic in about three days, forming interveinal necroses on the soybean leaves (Figure 2). After one to two weeks, the necrotic spots may enlarge and coalesce, forming interveinal chlorotic streaks. These streaks eventually
become necrotic, leaving only the mid veins and major lateral veins green (Figure 3). Leaflets of severely affected plants either curl upward and remain attached to the plant (Figure 4) or drop off, leaving the petioles attached to the plant (Figure 5). This spreading necrosis gives areas of the field affected by the disease a characteristic brown to tan cast.

Although the brown to tan cast given to the affected areas by the foliar symptoms may be the first evidence of the disease, roots actually are infected before the foliar symptoms appear. Infected root systems are characterized by deterioration of the taproots, lateral roots, and nitrogen fixing nodules. In moist soil, bluish fungal colonies may be visible on the taproots (Figure 6). The cortex of affected taproots is a light gray-brown, and this discoloration may extend up the basal stem a few inches above the ground or into several nodes in the vascular tissue. Comparing a suspected SDS plant with a healthy plant (Figure 7) will help judge whether the suspected plant is actually infected with the SDS pathogen.

SDS may occur in scattered areas of a field or may pervade an entire field. In fields where SDS is less severe, the disease occurs in small, circular patches or on scattered plants (Figure 8). In fields where the disease is severe, premature defoliation may occur in large areas (Figure 8). The level of damage caused by SDS depends on the growth stage at which the disease appears and on the severity of foliar symptom development. Severe symptoms in the early reproductive stages may lead to flower and pod abortion (Figure 5). Symptom development during the pod fill stage may result in smaller seeds. If the disease occurs after the pod-fill stage, little or no yield loss occurs.

The earlier the infection occurs, the more likely the severe foliar symptoms will be late in a season. Early seedling infection enables the fungus to colonize in xylem vessels of soybean root, which translocates the fungal toxin to leaves. The fungus starts to infect plants as soon as seed germinates.

Infections in early seedling stage are critical for xylem colonization, therefore, are very effective to production of foliar symptoms. Management of infections early in a season is a key to prevent the disease.

Confusion with Other Diseases
Stem canker and brown stem rot (BSR) exhibit foliar symptoms similar to those of SDS and often are misidentified as SDS. Stem canker is
distinguished from SDS by the presence of a canker on the surface of the lower stem and the absence of root rot.

Brown stem rot, most commonly mistaken for SDS in the north central United States, is distinguished from SDS by an internal brown discoloration of the pith and the absence of root rot. In a plant infected by the SDS pathogen, the vascular tissue turns brown and the pith remains white; root rot also is present. While examining a plant for SDS, one should dissect the stems to the base because BSR starts from the plant crown. Some plants may have both BSR and SDS, with symptoms of both diseases, which make identification difficult. In such cases, plants may be sent to the Plant Disease Clinic at Iowa State University for identification.

Phytophthora root rot, another soybean disease common in Iowa, is distinguished from SDS by the absence of interveinal necrosis. (See Table 1 for a list of symptomatic differences between SDS and other soybean diseases.) The time when a disease occurs within the growing season is also useful for separating SDS from other diseases (Table 1). In Iowa stem canker and Phytophthora root rot may appear before flowering, but SDS symptoms occur just before flowering to pod formation stages depending on weather. Foliar symptoms of BSR occur no earlier than reproductive stage R5.

Another disease with SDS-like symptoms that may be misidentified as SDS is caused by different strains of F. solani. This disease produces interveinal chlorosis and light defoliation but no necrosis. These Fusarium strains produce less toxin than the strains that cause SDS.

**Risk factors**

SDS is more prevalent during cool, wet growing seasons. Both early planting in cool and wet soils and flooding during the early vegetative stage increases disease risk. In contrast, later planting after mid-May significantly reduces the disease. Hot and dry weather appears to slow or arrest the disease.

Tillage practices also affect disease occurrence: studies show that the disease is more severe in no-till fields but lightest in ridge till fields because tillage practices increase soil temperatures and reduce soil moisture. Generally, the disease occurs in fields where soybean growing conditions are optimal and yield potentials are high.

Fields with a history of the disease are likely to develop the disease again in subsequent years. Corn-soybean rotation will not reduce the disease.

Presence of soybean cyst nematode (SCN) (*Heterodera glycines*) enhances the disease. It is not clear how SCN enhance the SDS. However, SCN populations are usually, but not always, associated with SDS.
Management

In Iowa, SDS is more prevalent in fields east of Interstate 35. Fields in western Iowa are less infested. Prevention of the spread of the SDS pathogen is the most effective means for managing the disease in areas where disease incidence is low. In areas where SDS is not found, one should avoid introducing the pathogen. The pathogen can be disseminated with soil, which is similar to the spread of SCN. Controlling the movement of soil with farming equipment, which also is suggested for SCN management, is effective.

If SDS is already present, use integrated approach to manage the disease.

- Use of resistant varieties. Find one with consistent evaluation scores.
- Avoid planting before the first week of May. Fields planted after mid-May often have less risk as a rule of thumb. If a field had severe SDS in the past, design a planting route in which the infested field is planted last.
- Improve soil drainage and use tillage to warm up soil and reduce soil moisture.
- Manage to keep SCN population in a very low level.
- Corn and soybean rotation does not reduce the disease. Numerous observations show that the disease will return after corn has been planted for years.

Table 1. Symptomatic differences between SDS and other soybean diseases

<table>
<thead>
<tr>
<th>Diseases</th>
<th>Time of first occurrence</th>
<th>Intervenial chlorotic spots</th>
<th>Root rot</th>
<th>Pith browning</th>
<th>Canker on stem</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sudden death syndrome</td>
<td>R 1</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Stem canker</td>
<td>V10</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Brown stem rot</td>
<td>R5</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Phytophthora root rot</td>
<td>V1</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

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