Brown stem rot (BSR) is an economically important disease of soybean in the North Central United States. The disease was first observed in 1944 in Illinois and has since been reported in most soybean growing areas of North Central United States and Canada. Yield losses up to 30 percent can occur if susceptible varieties are planted and environmental conditions are favorable for disease development. Brown stem rot was the third most prevalent soybean stem disease from 2005-07 with nearly half of the counties in Iowa having BSR in 2006 and 2007 (Figure 1).

Causal organism
Brown stem rot is caused by the soilborne fungus Cadophora (Phialophora) gregata. There are two genotypes of C. gregata, and they are commonly referred to as genotype A and genotype B. The genotypes differ in their ability to cause foliar symptoms on susceptible soybeans. A disease survey completed in the mid 1990s found that genotype A was more prevalent in the eastern part of Iowa; while genotype B was more prevalent in the western part of the state.

Symptoms
Two types of disease symptoms are associated with infection by C. gregata. Internal stem (vascular tissue and pith) browning is a diagnostic symptom of BSR and is caused by both genotypes of the fungus (Figure 2). Internal stem browning starts in the root crown and progresses up the stem as the disease progresses, however it also may be discontinuous and appear only at nodes (Figure 3). Soybean stems also can be colonized without obvious browning symptoms and thus the disease can be difficult to identify.

Foliar symptoms are characterized by interveinal yellowing and browning of leaves (Figure 4). Symptomatic leaves shrivel up but remain attached to the stem. Foliar symptoms easily can be confused with those of sudden death syndrome (SDS) caused by Fusarium virguliforme (syn. F. solani f.sp. glycines) or signs of early crop maturity or drought stress. Genotype A causes stem browning and foliar symptoms on susceptible soybeans. Genotype B causes only stem browning; foliar symptoms are rare on most soybean varieties.

Other symptoms associated with BSR include stunting, leaf deformity, premature death of plants, reduced pod set, seed number and seed size, and, lodging of severely diseased plants. The distribution of brown stem rot in a field is characteristic of a soilborne disease: diseased plants often occur in clusters.

Figure 1. Prevalence of brown stem rot in Iowa in the 2005 to 2007 growing seasons.
A systematic design was used to collect 30 soybean plants from 3 to 5 fields in each county at four growth stages, V2-V3, R1-R3, R4-R5, and R6-R7, in each growing season. Each plant sample was visually assessed for brown stem rot by splitting the stem to look for pith discoloration.
Disease cycle
The BSR fungus does not form specialized survival structures but survives as mycelium in infested soybean residue. The pathogen infects the roots of young soybean plants early in the growing season. High soil moisture and low air temperatures (70-80°F) favor colonization of the stem tissues by the fungus. Therefore, BSR is usually more severe during cool, rainy growing seasons. Stem symptoms become evident at growth stage R2, while foliar disease symptoms usually only become apparent around growth stage R4. Both stem and foliar symptoms continue to develop as the season progresses. Yield losses are higher when foliar disease symptoms appear early and are severe. Yield losses can occur when foliar symptoms are absent and only vascular/pith discoloration is evident.

Management
Yield loss due to BSR can be reduced effectively if the following management practices are used.

Resistant soybean varieties
As with many diseases, growing varieties with resistance to BSR is the most effective management option. Soybean varieties with resistance to both soybean cyst nematode (SCN) and genotype A of the BSR fungus can provide adequate protection even where both SCN and C. gregata genotype A of the BSR fungus are present. Currently, researchers at Iowa State University are testing many SCN resistant varieties to determine whether these varieties also can provide adequate protection against C. gregata genotype B of the BSR fungus. No varieties with resistance to genotype B are currently available.

Rotation
Soybean is the only known natural host of C. gregata and thus rotation to other crops such as corn, alfalfa, or small grains is an effective means of decreasing population levels of the pathogen and therefore reducing BSR severity. A longer rotation, two to three years out of soybean, may be necessary in fields where the disease has been particularly severe.

Tillage
Brown stem rot is more prevalent in no-till fields compared with tilled fields. No-till fields contain higher levels of crop residue, which allows for increased fungus survival and therefore higher inoculum levels. Since tillage promotes decomposition of infested crop residue, it is an effective method for decreasing inoculum levels and thereby reducing the
risk of BSR disease. In addition, no-till fields tend to have lower soil temperatures and higher soil moisture levels, which favor the development of BSR.

**Soybean cyst nematode management**

Research at Iowa State University has demonstrated that the presence of soybean cyst nematode (SCN, Heterodera glycines) increases BSR disease in numerous soybean varieties (Table 1). In addition, high numbers of SCN result in the earlier colonization of soybeans by C. gregata and increased disease severity (Figure 5).

Therefore, SCN management should play a crucial role in BSR disease management. Many soybean varieties with PI 88788-derived SCN resistance also have BSR resistance. Therefore, planting soybean varieties with PI 88788-derived SCN and BSR resistance can reduce the adverse effect of SCN on BSR disease development. Varieties with Peking and Hartwig-derived SCN resistance may be susceptible to BSR and growers are advised to ask seed suppliers if varieties also have BSR resistance.

<table>
<thead>
<tr>
<th>Soybean variety or line</th>
<th>Resistance/ susceptibility to</th>
<th>Severity of BSR (% colonization)a</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>C.g.</td>
<td>H.g.</td>
</tr>
<tr>
<td>Sturdy</td>
<td>S</td>
<td>S</td>
</tr>
<tr>
<td>BSR101</td>
<td>R</td>
<td>S</td>
</tr>
<tr>
<td>PI 84946-2</td>
<td>R</td>
<td>S</td>
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<tr>
<td>PI 437833</td>
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<tr>
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<tr>
<td>PS2465N</td>
<td>R</td>
<td>R</td>
</tr>
<tr>
<td>Freeborn</td>
<td>R</td>
<td>R</td>
</tr>
</tbody>
</table>

a Maximum height of stem colonized by C. gregata/total stem height x 100%

Table 1. SCN increases incidence and severity of Cadophora gregata genotype A colonization of soybean genotypes ten weeks after inoculation with various combinations of C. gregata (C. g.) and Heterodera glycines (H. g.). (adapted from Tabor et al. 2003b).

![Figure 5](image.png)

**Figure 5.** Incidence of colonization of soybean stems by genotype A of Cadophora gregata (C. g.) in the absence or presence of two population densities (1,500 eggs and 10,000 eggs) of Heterodera glycines (SCN) in the greenhouse (adapted from Tabor et al. 2006). BSR101 is resistant to genotype A of C. gregata and susceptible to SCN; Jack is resistant to genotype A of C. gregata and SCN; and Sturdy is susceptible to both genotype A of C. gregata and SCN.
References

Information was collated and adapted from the following sources:


