SOYBEAN STEM HEALTH

Nancy C. Koval and Craig R. Grau[1/]

Importance of Soybean Stem Diseases

Soybean stem health is an understudied area of soybean pathology. With the exception of white mold (Sclerotinia stem rot), symptoms of brown stem rot and stem canker are often overlooked or confused with stress related to climatic conditions or with seasonal changes in the growth and development of soybean. Brown stem rot occurs each year, but severity and yield loss is favored by conditions ideal for soybean growth. Stem canker has increased in incidence and severity throughout the north central U.S. and Ontario, Canada. The recent resurgence of stem canker in the north central region has not been explained. However, likely factors are associated with reduced tillage, shortened rotation systems and changes in soybean germplasm. Additional considerations are that the stem canker pathogen has undergone genetic changes or that related fungi have emerged and are capable of causing symptoms along with the original causal pathogen. If considered as a complex, brown stem rot, white mold and stem canker occur across a range of climatic conditions that essentially ensure a high probability that one of them will be yield-limiting in a given year. Thus, the ideal soybean variety would have resistance to each disease.

Brown Stem Rot

The incidence and impact of brown stem rot on yield is commonly underestimated because symptoms are often not readily observed, or if foliar symptoms occur, are mistaken for premature, but natural senescence late in the season. These situations lead to an underestimation of the occurrence and yield robbing ability of brown stem rot. Brown stem rot is caused by a fungus that infects roots early in the season, but in time moves into the vascular system of the soybean plant. The pathogen causes a gradual disruption of the vascular system resulting in symptoms appearing during reproductive growth stages. Yield loss is generally greatest when foliar symptoms develop compared to only when symptoms are evident inside stems. The soybean cyst nematode (SCN) is also a widespread and destructive pathogen of soybean. In many areas, both brown stem rot and SCN diseases occur together in the same fields and both cause ‘hidden’ yield losses in the absence of clear symptoms. A brown stem rot management plan involves matching soybean varieties to crop rotation sequences, soil pH, tillage practices and planting date.

[1/] Research Specialist and Professor, Department of Plant Pathology, Univ. of Wisconsin-Madison, Madison, WI 53706

Crop Rotation

The brown stem rot pathogen survives in soybean residue and does not form structures that enable it to survive for long periods in the absence of a host. Thus, crop management systems that favor the decay of soybean residue can reduce the survival of the pathogen. Since the host range of brown stem rot pathogen is limited to soybean, extended periods of cropping to nonhosts effectively lowers inoculum of the pathogen. The rate of inoculum decline is directly related to rate of soybean residue decomposition. The incidence and severity of brown stem rot is modified by ambient and soil environments, and crop management systems. The brown stem rot pathogen is not carried with seed and minimal inoculum is carried with soil adhering to equipment. Incidence and severity of brown stem rot is suppressed effectively by 2 or more years of corn or another nonhost crop. The popular rotation of alternating soybean and corn on annual basis is better than continuous soybean culture, but still maintains a risk of yield loss caused by brown stem rot. Other management practices, soybean varieties planted and climatic conditions modify the risk involved with the annual alternating rotation of soybean and corn.

Tillage Systems and Brown Stem Rot

The brown stem rot pathogen survives in soybean residue and does not form long-term survival structures. This situation places importance on how soybean residue is managed. The severity of brown stem rot is frequently, but not always, greater for soybean grown in no-till systems. Greater severity in no-till or minimal tillage systems is likely related to higher population densities of the brown stem rot pathogen. However, cool soil conditions may be a contributing factor to why brown stem rot is favored by none or less tillage.

Table 1. Effect of tillage and crop rotation on the severity of brown stem rot and soybean yield.

	Disease severity of variety		Yield bu/a	
Tillage	**Susceptible**	**Resistant**	**Susceptible**	**Resistant**
Conventional	21	1	51	59
No tillage	67	2	44	55

Disease severity rated on a scale of 0-11; 0 equals no foliar symptoms of brown stem rot and 11 equals 100% of the foliage expressing symptoms. Disease severity and yield values are the means of eight location years of data from 1989-1992 at the Arlington Agricultural Research Station.

Date of Planting and Row Width

Brown stem rot is a disease of greater importance in high yield potential environments. The severity of brown stem rot is generally not greater in management

systems employing early planting date or narrow row systems. However, the yield advantage of resistant soybean varieties to susceptible varieties will increase with increasing yield potential. For example, there is a significant chance that yield of brown stem rot resistant variety will not respond to narrow row widths and an early planting date in fields with high potential for brown stem rot. However, in the presence of brown stem rot, a brown stem rot resistant variety can have a decisive yield advantage if planted in 7-inch row width versus 30-inch rows. The impact of brown stem rot is greatest when the environmental conditions and management are favorable for high yield potential of soybean.

Soil pH and Brown Stem Rot

The severity of brown stem rot is greatest if soil pH approaches 6.0 and is less severe as soil pH approaches 7.0 or greater. This conclusion is based on several years of small plot and large-scale on-farm trials. Furthermore, the effect of soil pH is regarded as significant and modifies effects of crop rotation and tillage on brown stem rot potential.

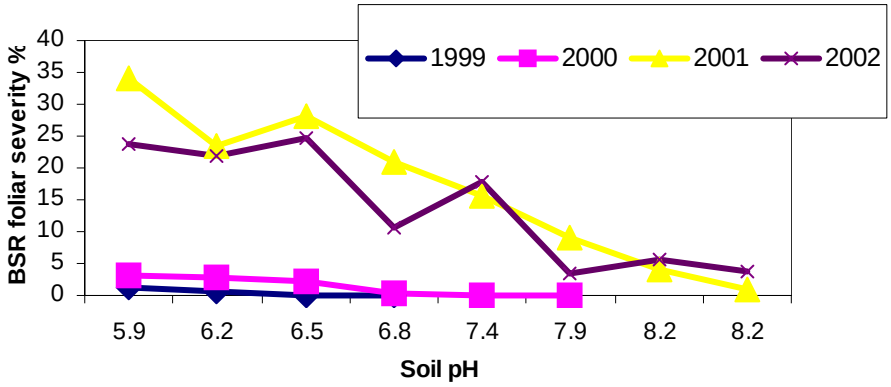

Figure 1. The severity of brown stem rot increases with rising soil pH. Data is from 4 years of field studies in Wisconsin.

Brown Stem Rot Resistant Varieties

The genetic yield potential of soybean varieties has increased through the efforts of soybean breeders. Much of the increased yield potential is linked to increased pod set and seed numbers. There is evidence that improved disease resistance is contributing to yield components correlated to increased yield potential. Resistance to brown stem rot is believed to be a contributing factor of greater yield potential of modern soybean varieties. The majority of yield loss due to brown stem rot is related to a reduction in seed number. Severely diseased plants lodge, which results in greater mechanical harvest loss in addition to physiological yield loss. The majority of newly released soybean varieties do not express a high level of susceptibility that was common a decade ago. Variety selection to manage brown stem rot relates the degree of resistance needed to match disease potential. The release of brown stem rot resistant varieties has opened new

avenues of flexibility for farmers experiencing problems with brown stem rot. Varieties vary in degrees of resistance. Complete resistance to brown stem rot has not been reported, but many several sources of partial resistance are available to soybean breeders. However, many soybean varieties on the market today express good to excellent resistance that minimizes yield loss even in high brown stem rot potential fields. Partial resistance is defined as a plant reaction of mild to no foliar symptoms, and a lesser severity of internal stem symptoms. Complete resistance, which prevents infection, has not been identified at this time against the BSR pathogen. Soybean accessions rated as partially resistant support varying degrees of reproduction of the Brown stem rot pathogen.

Soybean varieties have improved greatly for resistance to both brown stem rot and the SCN. Resistance to both brown stem rot and SCN comes from several different and independent sources. Many SCN resistant varieties also expressed resistance to brown stem rot. In most cases, SCN resistant varieties with resistance derived from PI 88788 are also resistant to brown stem rot but varieties with SCN resistance derived from Peking are susceptible to brown stem rot.

Brown Stem Rot and Soybean Cyst Nematode

There are significant yield benefits for soybean varieties that are resistant to predominant genetic types of the brown stem rot pathogen and to SCN. Many SCN resistant varieties are derived from the PI 88788 form of SCN resistance. Researchers have observed that soybean varieties with resistance to SCN derived from PI 88788 express low symptom severity of BSR. Conversely, varieties with SCN resistance derived from Peking, expressed symptom severity similar to BSR-susceptible varieties. Development and release of new SCN resistant varieties pose the risk of increasing problems with SCN. For example, the variety Hartwig, derives its resistance from PI 437.654 and is being used extensively in public and private soybean breeding programs as a source of SCN resistance. Although Hartwig offers an advantage of broad spectrum activity to SCN control, studies in Wisconsin have found Hartwig and its parent, PI 437.654, to be susceptible to BSR in greenhouse experiments. These findings raise concern that the BSR control achieved with PI 88788-derived SCN resistance will be lost if Hartwig-derived SCN-resistant varieties displace the former varieties. Efforts will continue to select breeding lines with both the PI 88788 and Hartwig form of SCN resistance. Crosses will be made with Hartwig SCN sources and Wisconsin BSR resistant lines to develop breeding lines with both BSR resistance and Hartwig form of SCN resistance. This action does not involve the Cyst-X technology thus derived lines can be used without tech fees.

White Mold

Resistance to white mold takes on greater importance with the rise in soybean aphid and rust activity in Wisconsin. Early planting is a management recommendation for

soybean aphid and virus control, and will likely emerge as a recommendation for rust management. However, early planted fields are more prone to have white mold problems. Thus, higher and more stable forms of white mold resistance are needed in order to effectively implement management programs for aphids and rust. Again, managing fields for maximum yield potential will assist with potential costs related to rust control.

The ultimate goal of our research project is to develop soybean lines that express complete and environmentally stable resistance to white mold. Soybean lines were identified that express superior resistance to the white mold pathogen. Sources of resistance will be studied to determine if different mechanisms of resistance operate in soybean. It is hypothesized that complete resistance can be achieved by combining genes regulating different mechanisms into an elite soybean line. These results have been achieved by an improved inoculation method for white mold pathogen. A greater chance of success will be increased by knowledge of genetic inheritance of resistance to white mold and how specific lines resist the white mold pathogen. Many soybean lines have been studied at several universities to determine genetics of white mold resistance in soybean. The general result is that many gene groups (QTL) have been identified but no one group contributes a high degree of resistance. A comprehensive understanding of partial resistance could lead to combining field relevant resistance genes into one breeding lines resulting in a more complete resistance than we have in the current commercial germplasm.

Summary of Recommendations to Manage White Mold

Factors that modify the effectiveness of recommendations: Weather and excessive soil fertility may promote excessive vegetative growth, or soybean varieties that lodge or produce dense crop canopies may modify the benefits of suggested tactics presented in the table. A summary of factors that may modify recommendations is presented in Table 2. A core set of recommendations is presented in Table 3 and could be modified after further research.

Table 2. Factors affecting the severity of white mold in soybean.

Seasonal risk factors	**Long-term risk factors**
Weather: cool temperatures (<85 F), normal or above normal precipitation, field capacity or above soil moisture; and prolonged morning fog and leaf wetness (high canopy humidity) at and following flowering into early pod development.	Field/cropping history: inoculum of pathogen will gradually increase if: other host crops are grown in rotation with soybean; interval between soybean crops is shortened; and white mold susceptible varieties grown.
Early canopy closure: due to early planting, high plant population, narrow rows, excessive plant nutrition and optimal climatic conditions. Dense canopy increases apothecia density.	Weed management systems: degree of broadleaf weed control; herbicides used in rotation systems may e suppressive to white mold.
History of white mold: population density of white mold pathogen; apothecia present on soil surface at flowering; distribution of pathogen/disease in field.	Topography of field: pockets of poor air drainage; tree lines and other natural barriers to impede air movement.
Soybean variety planted: physiological functions and plant structure govern reaction.	Pathogen introduction: contaminated and infected seed; movement of infested soil with equipment; wind-borne spores from apothecia from area outside fields.

Table 3. Core recommendations for management of soybean white mold.

Field History	Variety Selection	Canopy Modification	Crop Rotation	Agricultural Chemicals	
				Grain Fields	Seed Fields
No white mold; Monitor fields closely	Variety of choice; plant pathogen free seed	Maintain current row width; plant population	Avoid crops susceptible to white mold	Adjustments to herbicide program not needed	Adjustments to herbicide program not needed
<5% diseased plants aggregated in field	Avoid suscept-ible varieties	Maintain current row width; plant population	Minimum of 1 year out of soybean	Adjustments to herbicide program not needed	Adjustments to herbicide program not needed
<5% diseased plants uniformly distributed in field	Consider partially resistant; avoid susceptible varieties	Maintain current row width; lower population for less resistant varieties	Minimum of 1 year out of soybean	Adjustments to herbicide program not needed	Adjustments to herbicide program not needed
5-25% diseased plants	Partially resistant varieties	Maintain current row width; plant population	Minimum of 1 year out of soybean	Adjustments to herbicide program not needed	Adjustments to herbicide program not needed
5-25% diseased plants	Moder-ately susceptible varieties	Widen row width; lower plant population	Minimum of 1 year out of soybean	Consider white mold suppressive herbicides	Consider white mold suppressive herbicides or fungicides
25-50% diseased plants	Partially resistant varieties	Maintain current row width; lower plant population	1 to 2 years out of soybean	Consider white mold suppressive herbicides	Consider white mold suppressive herbicides or fungicides
>50% diseased plants	Partially resistant varieties	May consider narrow row spacing for most partially resistant varieties; Wide row; 150,000 plants/acre	2 to 3 years out of soybean	Consider white mold suppressive herbicides	Consider white mold suppressive herbicides or fungicides

Stem Canker

Stem canker has been widely recognized as an important soybean disease, but recently has been divided into northern stem canker and southern stem canker based on two causal agents. Northern stem canker was first reported in the late 1940s in Iowa, and by the 1950s, the disease had spread into the upper Midwest and Canada. Southern stem canker was reported in the south in 1973, and by 1984, had been detected in all southern states. Northern stem canker and southern stem canker are caused by *Diaporthe phaseolorum* var. *caulivora* and *Diaporthe phaseolorum* var. *meridionalis*, respectively. The host range of both pathogens has not been study extensively, however, over 16 weed species are known to harbor *D. phaseolorum*.

Symptoms and Losses

Initial expression of symptoms occurs during the early reproductive stages, with the development of a small, reddish-brown superficial lesion at the base of branches or petioles. The lesion is first observable in the leaf scar after the petiole has fallen. The lesion elongates and becomes dark brown or black, sunken in appearance and often girdles the stem. As a result of a phytotoxin produced by the fungus, interveinal chlorosis and necrosis are expressed in the leaves and is soon followed by plant death. Above and below the canker, green tissue is present and the leaves on the dead plant wither but remain attached. A top dieback can occur and results in a characteristic shepherd's crook curling of the terminal bud.

Yield losses have been reported to be as high as 50 to 80% in naturally infested fields. Yield reductions resulting from stem canker have increased dramatically over the past 2 years. Estimated yield losses to stem canker were 3.3 and 2.2 million bushels in 1999 and 2000, respectively.

Stem canker over winters in colonized stems and infected seed. Long distance dissemination of the pathogen is made possible by the movement of infested soybean residue and to a lesser extent by infected seed. Seed infection by northern stem canker can be as high as 10 to 20%. In late winter, pycnidia (fruiting bodies) begin to develop and conidia (spores) are released beginning in late April continuing into June and serve as the primary inoculum. Splashing, blowing rain, and wind disperse spores up to 6 feet from the point source to petioles, petiole bases, stems, and leaves. The growth stage of the plant at the time of exposure to the inoculum heavily influences the incidence and severity of disease. Exposure to inoculum at V3 corresponds to the highest severity of disease. Disease severity is progressively reduced when first contact is delayed from V3 to V10 growth stages. Secondary inoculum is released from pycnidia present in stem cankers. Conidia produced at this time could be responsible for late season infections and thereby increase the inoculum potential for the next growing season.

Environmental conditions during the vegetative stages govern disease development. Temperature greatly influences infection, with the highest levels of infection occurring when the air temperature is between 82 and 93° F, with and optimal temperature of 83.5°F. Temperature and period of wetness are significantly related.

Rainfall during plant vegetative growth is critical for the development of stem canker epidemics. Cumulative rainfall, not the number of rainy days, is related to higher disease severity.

Management

Stem canker is effectively managed by the combination of planting resistant cultivars and reducing crop infested on the soil surface. Deep plowing can reduce crop residue prior to planting a soybean crop. Seed that are to be used for planting should not be harvested from fields with a history of stem canker. Fungicides applied to seed greatly reduce stem canker but will not completely eliminate the pathogen. Foliar fungicides can be effective when applied during vegetative stages, however, results are inconsistent, and in most cases, foliar fungicides would not be an economical management strategy. The benefits of crop rotation to reduce stem canker have not been demonstrated in production fields. Delayed planting can reduce the incidence and severity of stem canker; however, loss of yield potential that accompanies delayed planting makes this a questionable control strategy.