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ECOLOGY AND MANAGEMENT OF DISEASES OF SOYBEAN THAT OCCURRED IN IOWA DURING THE 2004 SEASON. PART II: STEM AND ROOT DISEASES.

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The 2004 season growing season had cooler than normal temperatures associated with frequent rainfalls. The weather conditions were favorable for the occurrence of many soybean diseases. This season, we saw the worst ever outbreaks of white mold and sudden death syndrome, which resulted in considerable damage for many soybean producers. Both diseases were reported in two-to-three weeks earlier in the growing season than usual. Severe white mold outbreaks were found in eastern Iowa, especially from Interstate 80 north. Our quick-and-dirty survey indicates about 20% of soybean fields were affected with white mold in that region, and some fields were up to 80% infested with the fungus. White mold also occurred in north central Iowa, however disease severity was less when compared with eastern Iowa. Sudden death syndrome outbreaks mainly occurred in regions south of Interstate 80 but also were found in central Iowa. Numerous reports of brown stem rot and stem canker were received from central and northern Iowa. All of these diseases are caused by soil-borne fungi.

White mold

Ecology

White mold is caused by the fungus *Sclerotinia sclerotiorum*. The fungus overwinters in the soil and debris as sclerotia which are hard, dark structures that resemble mouse droppings. When the soil conditions are cool (60-70°F) and moist (half to field capacity), small light tan funnel-shaped, mushroom-like structures called apothecia germinate from the sclerotia and produce masses of spores. The spores are windblown and are carried to nearby soybean plants. Flowers are particularly susceptible to infection by the fungus. More than 90% of infected plants are colonized by spores infecting through flower petals. Plants can also be infected through wounds in the stems and petioles which come into contact with infected plants. Cool (<85°F), wet weather during and after flowering are critical for white mold development. High temperatures (greater than 90°F) that occur after infection will stop disease development.

Early symptoms of white mold are water-soaked lesions at the nodes of the stems. A mass of white, cotton-like fungal mycelium grows on the lesions of infected stems. Sclerotia form in the dense mycelium. Infected plants wilt and the upper leaves become grayish-green and then turn brown and die while remaining attached to the stem.

Management

In 2003 growing season, white was prevalent but not severe in north eastern Iowa. The disease
was also reported in east central Iowa. These fields will return to soybean in 2005 and therefore, the risk of the disease can be high if the 2005 growing season is cool and wet. There are several management options that can be used to manage white mold. It is helpful to know the history of each field since this information influences what management practices will be useful. Since sclerotia are able to survive many years in soil, it is near impossible to eradicate this pathogen from infested fields. Therefore management options that focus on creating unfavorable conditions for disease development are most successful.

**Soybean variety selection**

Variety selection is essential to successful management of white mold. Although there are no soybean varieties that are completely white mold-resistant, there are a number of partially resistant varieties available. These varieties help reduce the inoculum load (number of sclerotia) in soil because the white mold fungus has a low reproduction rate on them. Field data from several states has shown that yield loss is minimal when the incidence of white mold is less than 20%.

**Canopy management**

Cultural practices which promote rapid canopy closure favor white mold development because they result in a favorable microclimate for apothecia germination and sporulation. If soybeans are planted very early (end of April to early May), closure of the soybean canopy and flowering coincide with the formation of apothecia. White mold incidence is often higher in soybeans planted in narrow rows and at high plant populations. Therefore, avoid using row spacing of less than 15 inches in fields with a history of low to moderate white mold infection. If disease pressure is high, and environmental conditions conducive, white mold severity is similar in narrow and wide row widths.

**Rotation and Tillage**

Rotations with corn and other non-host crops help reduce the number of sclerotia in the soil and therefore decrease the incidence of white mold. The benefits of rotation can be enhanced if used in combination with no-tillage because sclerotia remain on or near the surface. This exposes them to weathering and decay, and allows them to germinate under a non-host crop. Tillage buries the sclerotia, which prevents their germination and prolongs their survival. Subsequent cultivation and tillage bring the sclerotia to the surface.

Variety selection, canopy management and crop rotation are highly effective successfully reducing white mold incidence in soybean. These three practices should be the cornerstone of a white mold disease management program. Other tactics which can be considered include chemical and weed control.

**Chemical control**

Under low disease incidence, foliar sprays that penetrate the canopy can reduce the incidence of white mold. However if disease incidence is high, consistent control is not guaranteed. Thiophanate-methyl (Topsin-M®) is registered for white mold control in Iowa and is highly effective against S. sclerotiorum, if it is applied properly. The fungicide must be applied to the
soybeans at 50 percent of full bloom. The herbicide lactofen (Cobra ®) also is registered for use on white mold in Iowa. There are conflicting opinions on the use of Cobra ® in the midwest. Various research results show that the activity of this chemical is influenced by soybean variety, its use with or without crop oil, and the time of application. In addition, the application of Cobra ® does not necessarily lead to improved yields, in some cases, yields were lower than the untreated control. In Iowa, if disease pressure is moderate to high, we have had good results when the herbicide is applied to white mold susceptible varieties before R1 (early flowering) without crop oil. Use of white-mold-tolerant cultivars can achieve equal or superior effects to that of chemical strategies.

**Weed control**

*S. sclerotiorum* has a wide host range. A number of weed species are hosts of the fungus, for example, lambsquarters, velvet leaf, ragweed, nightshade, Canadian thistle and mustard. Therefore good weed control is necessary to reduce the build up of white mold inoculum.

**Sudden Death Syndrome**

**Ecology**

Sudden death syndrome (SDS) is caused by the fungus *Fusarium solani* f. sp. *glycines*. In the absence of soybean, the fungus survives as a thick-walled survival structure (chlamydospores) in the soil, soybean debris and on the cysts of the soybean cyst nematode (SCN). Infection occurs during early vegetative growth and is favored by high soil moisture and cool temperatures (55-65°F). Fungal colonization is limited to the roots and crowns of infected plants. SDS symptoms do not occur until the pod developing stage (R2-R5) in Iowa (late July and early August). Cool and rainy periods favor symptom development. Leaves on infected plants first have scattered yellow or white spots between leaf veins. These spots start to die and enlarge to form brown streaks between the veins (interveinal necrosis). Only mid-vein and major lateral veins remain green. Eventually, infected leaves drop but petioles remain on the stem. Diseased plants have rotted taproots and lateral roots and the root cortex is discolored a light gray-brown. Severe disease development early during reproductive development of the soybean plant can result in lower and pod abortion. Symptoms result from a toxin which is produced by the fungus and translocated from the roots to the leaves.

This season, SDS foliar symptoms may have been mistaken for brown stem rot which has similar leaf symptoms. However, no root rot is associated with brown stem rot.

**Management**

It is difficult to manage SDS but an integrated management plan which includes the following options can help to reduce the incidence and severity of this disease. Variety selection and planting date are particularly important and experience has shown that these alone can help reduce the incidence and severity of SDS.

**Variety selection**

Variety selection is crucial in fields with a history of SDS. There are several varieties available with partial resistance to SDS. In addition, varieties with resistance to SCN tend to be less
susceptible to SDS.

**Planting date**

Design a planting route so that any field with a high risk of SDS is planted last. Planting in mid-May versus early May reduces the risk of SDS because the soils have had time to warm up and dry out.

**Manage soybean cyst nematode**

There are reports showing a high correlation of SDS severity with high soybean cyst nematode populations. Soil sampling, rotation and crop rotation all help to reduce SCN levels.

**Improve drainage and reduce soil compaction**

Since SDS is favored by excess soil moisture, improved drainage will help reduce disease incidence. Soil compaction is directly related to foliar symptoms of SDS, therefore, if feasible, subsoiling can be done to decrease SDS severity.

**Brown Stem Rot**

**Ecology**

Brown stem rot (BSR) is caused by the fungus *Phialophora gregata*. The fungus survives as mycelium in soybean residue. It does not form survival structures like *S. sclerotiorum* (sclerotia) and *F. solani* (chlamydospores). In early spring, spores produced by the fungus, germinate, infect soybean roots and colonize the vascular systems of soybean seedlings. The fungus remains in the lower stem until pod development starts and then it advances up the stem. Wet and cool (60-75°F) conditions accelerate the movement of the pathogen.

There are two pathotypes of *P. gregata*, A and B (M). Both cause internal stem discoloration. However, only pathotype A causes external foliar symptoms of interveinal chlorosis and necrosis, wilting and defoliation. Foliar symptoms become evident in late July and early August. Dry conditions decrease the severity of leaf symptoms, but above normal air temperatures suppress leaf symptoms.

**Management**

Effective control of BSR can be achieved using combinations of the following practices: resistant varieties, crop rotation, tillage and soybean cyst nematode management.

**Variety selection**

Brown stem rot resistant varieties are available to significantly reduce the incidence of BSR and protect yield potential. Research done at the University of Wisconsin-Madison has shown that resistant varieties can provide a "rotation effect" by reducing BSR severity and improving yield potential in the following soybean crop.

**Rotation**

Soybean is the only host of *P. gregata*. Rotations of two or more years of corn or another crop
will greatly reduce the population of the fungus and the risk of BSR.

**Tillage**

Since the fungus only survives in infected soybean residue, decomposition of residue is important for managing this pathogen. Tilling soil increases the decomposition of residue and therefore decreases population levels of *P. gregata*. In no-till systems, high BSR disease severity is correlated with a high inoculum density of *P. gregata* within soybean residue. Therefore, longer crop rotations and shredding soybean straw are recommended for no-till fields.

**Soybean cyst nematode management**

Research at Iowa State University has shown that the incidence and severity of BSR can be enhanced with SCN infection. Therefore, monitor SCN levels in fields with a history of BSR and grow SCN-resistant varieties and follow good crop rotations to decrease SCN populations.

**Stem canker**

**Ecology**

Stem canker is caused by two fungi. *Diaporthe phaseolorum* var. *caulivora* causes northern stem canker and *D. phaseolorum* var. *meridionalis* causes southern stem canker. Southern stem canker usually is confined to the southern United States but its presence has now been confirmed in Illinois (1998) and Wisconsin (2004). Its presence in Iowa will be determined during the 2005 soybean growing season. *D. pahseolorum* is seed-borne and probably is introduced into fields by infected seed. The fungus survives as mycelium in infected soybean debris. In spring, fruiting structures called perithecia form and release ascospores which are splash-dispersed by rain to healthy soybeans. Young soybean seedlings (V3) are infected but symptoms do not develop until flowering (usually mid-July onwards in Iowa). Excessive rainfall, high humidity and cool temperatures (70°F) are optimal for canker infection. Hail damage also may predispose soybean to canker infection. Disease symptom expression is increased by water stress during the reproductive stages of soybean growth. Initial symptoms of stem canker are small reddish-brown lesions, usually near leaf node three or four. The lesions darken and become sunken. The leaves above the canker turn yellow, die but remain attached to the plants. The canker can girdle the stem killing branches. Plants become very brittle and easily break at te infection site even if it remains green.

Stem canker can be confused with Phytophthora root rot but stem canker does not cause root rot. Stem canker is closely related to pod and stem blight, caused by *Diaporthe phaseolorum* var. *sojae*, which does not cause stem cankers.

**Management**

**Variety selection**

Stem canker was a significant problem in the North Central region during the 1950s but the use of resistant varieties significantly reduced the incidence of stem canker. Resistant varieties are the primary decision for canker management.
Rotation or tillage

Since the fungus survives in infected soybean residue, decomposition of residue is important for managing this pathogen. Tilling soil increases the decomposition of residue. As an alternative to tillage, longer crop rotations and shredding soybean straw are recommended.

Seed source

Avoid saving seed for planting purposes from fields where stem canker has been identified. Infected seed may introduce the fungus into previously disease-free fields.