The Relationship Between the Causal Agent of SDS and SCN in Wisconsin

David Marburger, Shawn Conley and John Gaska
Department of Agronomy, UW-Madison;
Ann MacGuidwin and Damon Smith, Department of Plant Pathology, UW-Madison

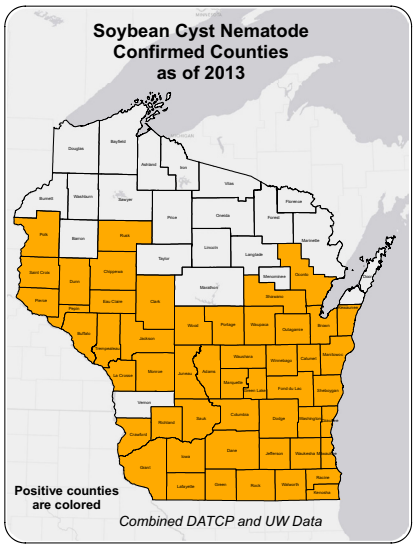

Figure 1. WI counties where SCN is confirmed as of 2013. DATCP and UW data.

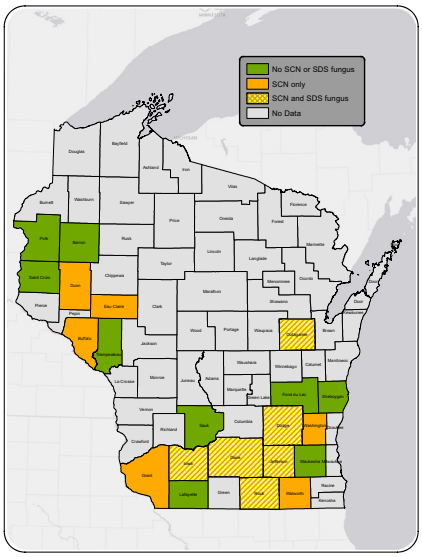

Figure 2. Wisconsin counties where soil samples were submitted from soybean fields that tested positive for SCN (orange shading), both SCN and the SDS fungus (orange and yellow shading), did not test positive for either pathogen (green shading), or were not sampled (light gray) in 2011.

Introduction

Soybean Cyst Nematode (SCN) is an economically important pest of soybean in Wisconsin. It was first discovered in the southeastern part of the state in 1981 and now is found in over 90% of the state's soybean acres (Figure 1). It is caused by the soybean cyst nematode, a non-segmented roundworm that inhabits the soil. More recently, another economically important disease of soybean, Sudden Death Syndrome (SDS), was first found in southeastern WI in 2006. A fungus found in the soil called *Fusarium virguliforme* is the causal agent of SDS.

Soybean Cyst Nematode (SCN): In high-yielding fields or during years when soil moisture is plentiful, profoundly visible symptoms of SCN are rarely seen. Subtle symptoms include uneven plant height, a delay in canopy closure, or early maturity. Severely infected plants appear stunted with yellow foliage, and canopy closure may be delayed or not occur in affected areas. Management of SCN should begin by sampling soil to confirm the presence of the nematode. For a detailed description about sampling for SCN in WI, see the pamphlet titled *Soybean Cyst Nematode Sampling and Testing in Wisconsin*. Additional management should also include an integrated plan where crop rotation and resistant cultivars are be used. Rotating to non-hosts of SCN can help reduce SCN populations in soil. Cultivars resistant to SCN should be planted when numbers of SCN are above suggested thresholds, and sources of resistance (e.g. Peking vs. PI 88788) should be alternated in fields with high populations. When SCN numbers are below threshold, rotating with resistant and susceptible varieties can slow the increase in populations of SCN that can overcome common types of resistance available in commercial soybean cultivars. Cultural practices such as managing weeds, providing adequate fertility, amending soil pH to at least 6.5, and improving soil moisture through tillage and supplemental irrigation can reduce plant stress and help plants deal with SCN populations.

Sudden Death Syndrome (SDS): Symptoms of sudden death syndrome are expressed as yellowing and necrosis between the veins of leaflets. Veins of symptomatic leaves will remain green. Leaflets will eventually curl or shrivel and drop off with only the petiole remaining. Management of SDS includes a combination of strategies. Most importantly, SDS-resistant cultivars should be chosen whenever possible. If SDS and SCN are both problems in a field, choosing a variety with the best resistance/tolerance to both will be beneficial. Planting into cool, wet soils typically seen early in the season can favor infection by the SDS fungus. Delaying planting can reduce the risk of infection, but remember that yield loss will occur from delaying planting too long. Improving soil drainage and reducing compaction can help reduce levels of SDS. Crop rotation can be useful to manage other diseases of soybean; however, research has demonstrated that crop rotation does not significantly reduce levels of the SDS fungus in the soil. Even after several years of planting corn in a field, the SDS fungus can survive on corn plants and corn kernels can harbor the pathogen. In addition, the SDS fungus can survive in soil for long periods of time as specialized, thick-walled spores. For more information about SDS, consult the Wisconsin Farm Fact Sheet XGT1015 titled *Sudden Death Syndrome of Soybean*.

Cross Relationships: The relationship between these two diseases has been studied for almost 30 years and has yielded inconsistent results. Studies have shown positive associations between SCN and SDS foliar symptom development, where more severe SDS symptoms occur when SCN is present (McLean and Lawrence, 1993; Melgar et al., 1994; Roy et al., 1989; Sherm et al., 1998; Xing and Westphal, 2006). However, other studies report weak or no association between SDS symptom development in the presence of SCN (Gao et al., 2006; Roy et al., 1993; Sherm et al., 1998). The relationship between the actual presence of the SDS fungus in the soil as it relates the presence of SCN has been under-studied.

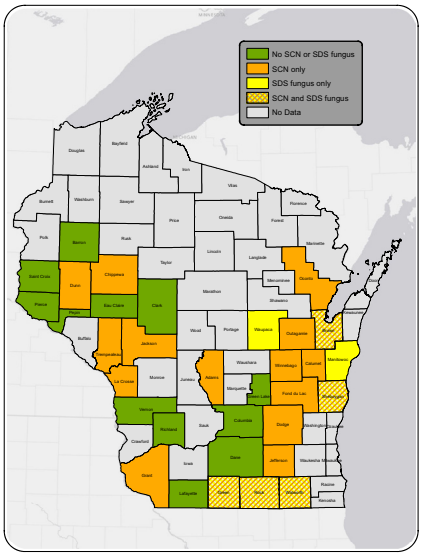

Figure 3. Wisconsin counties where soil samples were submitted from soybean fields that tested positive for SCN (orange shading), the SDS fungus (yellow shading), both SCN and SDS fungus (orange and yellow shading), did not test positive for either pathogen (green shading) or were not sampled (light gray) in 2012.

References

Bernstein, E.R., Atallah, Z.K., Koval, N.C., Hudelson, B.D., and Grau, C.R. 2007. *First report of sudden death syndrome of soybean in Wisconsin*. Plant Dis. 91:9, 1201-1201.

Gao, X., Jackson, T.A., Hartman, G.L., and Niblack, T.L. 2006. *Interactions between the soybean cyst nematode and Fusarium solani f. sp. glycines based on greenhouse factorial experiments*. Phytopathology 96: 1409-1415.

McLean, K.S. and Lawrence, G.W. 1993. *Interrelationship of Heterodera glycines and Fusarium solani in sudden death syndrome of soybean*. J. Nematology 25(3): 434-439.

Melgar, J., Roy, K.W., and Abney, T.S. 1994. *Sudden death syndrome of soybean: etiology, symptomology, and effects of irrigation and Heterodera glycines on incidence and severity under field conditions*. Can. J. Bot. 72: 1647-1653.

Roy, K.W., Lawrence, G.W., Hodges, H.H., McLean, K.S., and Killebrew, J.F. 1989. *Sudden death syndrome of soybean: Fusarium solani as incitant and relation of Heterodera glycines to disease severity*. Phytopathology 79:191-197.

Roy, K.W., Abney, T.S., and Patel, M.V. 1993. *Soybean SDS in the Midwest and South: Disease incidence and association of Fusarium solani with roots and with cysts of Heterodera glycines*. (Abstr.) Phytopathology 83: 467.

Scherm, H., Yang, X.B., and Lundeen, P. 1998. *Soil variables associated with sudden death syndrome in soybean fields in Iowa*. Plant Dis. 82: 1152-1157.

Xing, L.J. and Westphal, A. 2006. *Interaction of Fusarium solani f. sp. glycines and Heterodera glycines in sudden death syndrome of soybean*. Phytopathology 96: 763-770.

More research is needed to not only understand the relationship between SDS and SCN but also specifically between the two causal agents of these diseases. In WI, we have the unique opportunity to address this relationship. Until recently, the distribution of the SDS fungus throughout WI was not well understood, while the range of SCN was well documented in the state. We conducted a study to determine the presence of both causal agents, SCN and SDS fungus, from commercial soybean fields in WI and to determine if establishment of these two causal agents in production soybean fields is correlated.

Sample Collection Program: This study was possible through the checkoff-funded Wisconsin Soybean Marketing Board (WSMB) SCN soil testing program which offers free testing to WI growers. Soil samples that were voluntarily submitted during the 2011 and 2012 growing seasons were tested for SCN by wet-seiving methods and for the SDS fungus using DNA based detection methods.

WI Results: Sample submission totals are presented in table below for 2011 and 2012. In 2011, 56 of 135 (41.5%) samples were positive for SCN while 10 of 135 (7.4%) samples were positive for the SDS fungus. In 2012, 64 of 318 (20.1%) samples tested positive for SCN while 13 of 318 (4.1%) tested positive for the SDS fungus.

	SCN			*F. virguliforme* (SDS fungus)	
	# Samples	# Detected	Population range (Eggs/100 cc soil)	# Detected	Population range[a] (Spores/g soil)
2011	135	56	0-10,050	10	0-401,252
2012	318	64	0-37,200	13	0-11,226

Wisconsin counties testing positive for SCN in 2011 and 2012 were representative of the confirmed SCN-positive region of the state (Figures 1,2, and 3). Soil samples where both SCN and the SDS fungus were found in the same sample occurred infrequently (data not shown), and counties where both SCN and the SDS fungus were found were not common. Our results also show the SDS fungus was found in counties farther west and north of the area where Bernstein and colleagues first found the pathogen.

Conclusions and Recommendations

Our study found a negative correlation between SCN and the SDS fungus, indicating that as the probability of finding the SDS fungus in a soil sample increases, the probability of finding SCN in the same soil sample decreases. As the odds of detecting the SDS fungus in soil approach 100%, the likelihood of finding SCN in Wisconsin soybean fields is estimated at just 60%. This negative correlation suggests that SCN and the SDS fungus do not rely on each other to colonize fields. Therefore, fields with heavy SCN pressure are not at greater risk for colonization by the SDS fungus. However, in the infrequent case where SCN and the SDS fungus do occur together, symptoms of disease and damage by both pathogens can be synergistic. Therefore, disease management practices for both pathogens should be implemented in these fields.

Growers should continue to test their soils for SCN. If SCN is found, management practices discussed above should be implemented to reduce the effect of SCN and to ensure continued profitable soybean production.

The WSMB offers a free testing program for SCN. Sample kits can be requested by emailing to freescntest@mailplus.wisc.edu. More information is available at www.coolbean.info and http://fyi.uwex.edu/fieldcroppathology/

Data from: Marburger, D., Conley, S., Esker, P., MacGuidwin, A., and Smith, *D. Relationship between Fusarium virguliforme and Heterodera glycines in commercial soybean (Glycine max) Fields in Wisconsin*. Plant Health Progress. In press.