3-2012

Proceedings of the 39th Annual Meeting, Southern Soybean Disease Workers (March 7-8, 2012, Pensacola Beach, Florida)

Tom Allen
Mississippi State University, tom.allen@msstate.edu

Boyd Padgett
Louisiana State University, bpadgett@agcenter.lsu.edu

Danise Beadle
Eurofins Agroscience Services, danise.beadle@gmail.com

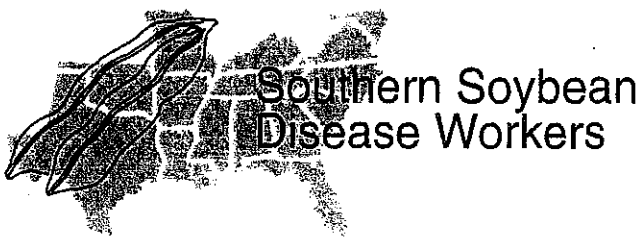
Stephen R. Koenning
North Carolina State University, srkpp@ncsu.edu

39th Annual Meeting of the Southern Soybean Disease Workers

March 7-8, 2012

Hilton Pensacola Beach Gulf Front Hotel

Pensacola Beach, FL



Sponsors of the

39th Annual Meeting of the Southern Soybean Disease Workers

Astra Life Sciences

BASF

Bayer

Cheminova

DuPont

NCSRP

Pioneer

Syngenta

US Soybean Board

We thank you for your support!

39th Annual Meeting of the Southern Soybean Disease Workers

March 7-8, 2012

Hilton Pensacola Beach Gulf Front Hotel*

Pensacola Beach, FL

Wednesday, March 7th Session

11:00 am **Registration**

1:00 pm **Welcome and Introductions**

Tom Allen, President

Symposium

Soybean Nematodes: Their Status, Impact and Management

1:15 pm **The Current Status of Nematodes of Soybean in Louisiana and Arkansas**

Charles Overstreet, Edward C. McGawley, Melea Martin, Louisiana State University Agricultural Center, and Terry Kirkpatrick, University of Arkansas

1:45 pm **A Molecular Analysis of Resistance of Soybean to the Soybean Cyst Nematode**

Vincent Klink, Mississippi State University

2:15 pm **Racism in Nematology**

Terry Niblack, The Ohio State University

2:45 pm **Break**

3:00 pm **SCN-resistant Soybeans, HG types, Yield, and SCN Reproduction – How It All Comes Together in the Field in Iowa**

Gregory Tylka, Iowa State University

3:30 pm **Soybean Lines Evaluated for Resistance to Reniform Nematode**

Sally Stetina, USDA, Stoneville, MS

Graduate Student Presentations

Moderator – Boyd Padgett, Past President

4:00 pm **Fungicide Resistance in *Cercospora kikuchii*, a Major Pathogen of Louisiana Soybean**

Trey Price, LSU AgCenter

4:20 pm **Variation in the Internal Transcribed Spacer (ITS) Region within Phakopsora pachyrhizi and Implications for the Currently Used Molecular Diagnostic Assays**

Tomas Rush, LSU AgCenter

4:40 pm **Theoretical Disease and Decision Model for Fungicide Applications for Soybean Rust**

Heather Marie Young, University of Florida

5:00 pm **Relationship Between Stink Bug Damaged Soybean Seed and Incidence of *Phomopsis longicolla* in the Mississippi Soybean Production System**

Joshua Jones, Angus Catchot, Fred Musser, Tom Allen, Maria Tomaso-Peterson, and Jeff Gore, Mississippi State University

5:20 pm **Spatial Assessment of *Rhizoctonia solani* In Fields Undergoing Rice/Soybean Rotations**

T.N. Spurlock[1], C.S. Rothrock[1], and W. S. Monfort[2]

University of Arkansas[1], Clemson University[2]

Thursday, March 8th Session

8:00 am **Registration**

Moderator – Danise Beadle

8:15 am **Sustaining the Pest Information Platform for Extension and Education with Support from Industry (I-PIPE)**

Scott Isard

9:00 am **Screening Germplasm for Resistance to Phomopsis Seed Decay: Joint Effort from USDA and University Scientists**

Shuxian Li, Gabe Sciumbato, Pengyin Chen, John Rupe, Allen Wrather, James R. Smith and Randall L. Nelson

9.25 am **Effect of Storage on Soybean Seed Quality and Emergence**

John Rupe

9:50 am **Break**

10:10 am **Foliar Fungicides to Prevent Yield Loss Attributed to Aerial Web Blight in Mississippi: 2010 and 2011**

Tom Allen, Alan Blaine, Bernie White, and Billy Moore

10:35 am **Mycovirus-induced Hypovirulence as an Alternative Means to Control Fungal Diseases of Soybean**

Said A. Ghabrial and Jiatao Xie

11:00 am **Managing Cercospora Blight in Louisiana – Facing New Challenges**

Boyd Padgett, Trey Price, Brooks Blanche, Ray Schneider, Clayton Hollier and Myra Purvis

11:25 am **Seasonal Progress of Charcoal Rot and Its Impact on Soybean Productivity**

Alemu Mengistu

11:50 am **Lunch (on your own)**

1:30 pm **Discussion Session**

Soybean Disease Resistance

Moderator - Clayton Hollier

Fungicide Resistance in *Cercospora sojina*: Chapter 2

Carl A. Bradley and Guirong R. Zhang

Fungicide Efficacy on Strobilurin Resistant *Cercospora sojina* (Frogeye Leaf Spot) in Soybean

Melvin Newman

Strobilurin Resistance in *Rhizoctonia solani* in Soybeans in Louisiana

Clayton A. Hollier

Discussion

SSDW Business Meeting (members please attend)

***The conference hotel of the 39th Annual Meeting of the Southern Soybean Disease Workers.**

Abstracts

39th Annual Meeting of the Southern Soybean Disease Workers

March 7-8, 2012

Symposium

Soybean Nematodes: Their Impact and Management

Soybean Nematodes in Louisiana and Arkansas

Charles Overstreet[1], Terry Kirkpatrick[2], Edward C. McGawley[1], and Melea Martin[1]

[1]LSU Agricultural Center: Department of Plant Pathology and Crop Physiology, Baton Rouge, LA and University of Arkansas, Southwest Research and Extension Center, Hope, AR

The nematodes that are an economic problem for soybeans in Louisiana have certainly changed during the past 30 years. Soybean cyst nematode (SCN) was a major problem for many of our producers during the 1970's and 1980's. The number of fields where this nematode was present began dropping off quickly in the 1990's and today it is extremely difficult to even find SCN in our state. During this same time period, the reniform nematode emerged from relative obscurity to become a dominant presence in many soybean fields. Reniform nematodes were found in 65% of soil samples in a survey conducted in Louisiana in 2011. The southern root-knot nematode has been fairly stable in incidence and distribution. Only about 10% of our soybean acreage has this nematode primarily because soybeans are usually planted on heavier ground. Arkansas has seen some changes in nematode problems in soybean as well. SCN is still a problem but perhaps not as much a problem as historically. Unfortunately many of the current cultivars do not cause highly visible symptoms, but still suffer 10-15 bushel losses. Races of the SCN have changed over the years, and few resistant cultivars are available for the current races. The southern root-knot nematode is the most widespread and damaging nematode in Arkansas, likely because a significant portion of the soybean acreage is either rotated to cotton or corn or has had a history of one or the other of these crops. Reniform nematode followed the same pattern in Arkansas as observed in Louisiana, moving from obscurity to become a common inhabitant in many fields throughout the Mississippi Delta. Nematode management practices in both states are primarily dependent on crop rotation and use of resistant cultivars. Crop rotation can be very effective against SCN and reniform nematodes using the crops that are available in both states. The southern root-knot is much more difficult to manage because of a lack of suitable rotation crops. A high percentage of cultivars used in both Louisiana and Arkansas have resistance to some races of SCN. Unfortunately, there is very little need for the resistance in Louisiana and the cultivars are relatively ineffective against races that are common in Arkansas. There are a few root-knot nematode resistant cultivars, but the resistance is not high in all cases. Very few reniform nematode resistant varieties are available in soybean.

A molecular analysis of resistance of soybean to the soybean cyst nematode

Vincent Klink, Mississippi State University

The defense responses of soybean to the soybean cyst nematode (SCN) occur at the site of infection, a nurse cell known as the syncytium. Two major genotype-defined defense responses exist, the *G. max*$_{[Peking]}$- and *G. max*$_{[PI\ 88788]}$-types. Resistance in *G. max*$_{[Peking]}$ is potent and rapid, accompanied by the formation of cell wall appositions (CWAs), structures known to perform important defense roles. In contrast, defense occurs by a potent but more prolonged reaction in *G. max*$_{[PI\ 88788]}$, lacking CWAs. Comparative transcriptomic analyses confirmed by Illumina® deep sequencing were performed through a custom-developed computer application, Pathway Analysis and Integrated Coloring of Experiments (PAICE) that visualizes gene expression of these cytologically and developmentally distinct defense responses. The analyses resulted in the generation of 1,643 PAICE pathways, allowing better understanding of gene activity across all chromosomes. Transgenic studies are elucidating the roles of the identified genes.

Racism in Nematology

T. L. Niblack, Department of Plant Pathology, The Ohio State University

The term "race" is time-honored in plant pathology, but unfortunately the term (along with its relatives "virulence," "aggressiveness," and even "pathogenicity," for example) has no strict definition. As Shaner et al. (1992) pointed out 20 years ago, "Our lack of precise definitions is of more than academic interest and has caused serious problems in communication." Such miscommunications have become increasingly important as our understanding of the genetic basis of host-pathogen interactions improves and the need for more and better resistant hosts increases. The most serious problem with the use of the term "race" in nematology is that a race classification is interpreted as a genotype. For "host races," that interpretation may be correct, but it is probably incorrect for "physiologic races." Dropkin (1988) long ago recommended dropping the use of "race" in nematology to avoid implications of genetic determinants.

For plant-parasitic nematodes, "race" most often relates to the ability of an isolate to reproduce on a defined *set of host species*. Host races are qualitative designations, and have been identified in such economically important species as *Ditylenchus dipsaci* (bulb and stem nematode), *Meloidogyne incognita* (southern root-knot nematode), and *Rhadopholus similis* (burrowing nematode), among others. In contrast, using a model established by mycologists for races of rust fungi, a group of scientists defined four races of *Heterodera glycines* (soybean cyst nematode [SCN]), distinguished by their ability to reproduce on four different *selections of the same host species*, soybean (Golden et al., 1970). To correct one of the problems engendered by the four-race description, the number of defined races was increased to 16 (Riggs & Schmitt, 1988), the correct number given four differentials in a dichotomous scheme. Three main problems remained, as follows: 1) one differential (Pickett) was derived from another (Peking) and thus could only differentiate SCN populations with increased, but not decreased, ability to reproduce on Pickett relative to Peking (making four of the 16 races "impossible"); 2) the addition of

new differentials would have the effect of doubling the number of race descriptions for each addition; and 3) the 10% female index threshold for determining whether to label the SCN population "+" or "-" on a differential has never been demonstrated as biologically meaningful for *H. glycines*. An attempt to address these problems was made in the proposal of the HG Type test (Niblack et al., 2002), which solved the first two problems but was unable to solve the third due to a lack of supporting data and a desire to maintain compatibility with the two race descriptions. This compatibility, however, resulted in misunderstanding of the HG Type test and allowed "racism" (i.e., the treatment of a race or HG Type designation as a genotype) to continue unchallenged.

Finally, a fourth problem remains as well, although this one is not so much a problem with identifying physiologic variation as with those who use published schemes; once published, the scheme becomes written in stone, and "racism" becomes ingrained. Labels such as "race 4" or "HG Type 1.2.3" take on a qualitative meaning. For studies of *H. glycines*, this problem impedes progress in understanding population genetics and its implications for management. At the very least, we should resist racism and report virulence profiles as the quantitative measurements that they are.

References: Dropkin, V.H. 1988. Annu. Rev. Phytopathol. 26:145-161. Golden, M.A., et al. 1970. Plant Dis. Rptr. 54:544-546. Niblack, T.L., et al. 2002. J. Nematol. 34:279-288. Riggs, R.D. & Schmitt, D.P. J. Nematol. 1988. 20:392-395. Shaner, G., et al. 1992. Annu. Rev. Phytopathol. 30:47-66.

SCN-resistant Soybeans, HG types, Yield, and SCN Reproduction – How It All Comes Together in the Field in Iowa

Gregory L. Tylka, Iowa State University

By definition, soybeans that are resistant to the soybean cyst nematode (SCN) support less than 10 percent reproduction of the nematode relative to what occurs on a standard, susceptible soybean variety. But many (or most) SCN populations in the Midwest have greater than 10 percent reproduction on PI 88788, the very common source of resistance used in breeding, presumably due to directional selection put on SCN populations through repeated and almost exclusive use of these same resistance genes. In Iowa, SCN populations with nearly 35 percent reproduction on PI 88788 have been found in several fields. The relationship among increased SCN reproduction, soybean yield, and season-long nematode control of resistant soybean varieties is not widely studied. The Iowa State University SCN-resistant Soybean Variety Trial Program annually evaluates the yield and SCN control provided by hundreds of SCN-resistant soybean varieties in field experiments throughout Iowa (see www.isuscntrials.info). The resistant varieties are grown in replicated plots at each experimental location, soil samples are collected from each individual plot at planting and at harvest to determine SCN egg population densities, and commonly grown SCN-susceptible varieties are included in each experiment as comparison (control) treatments. An HG type test is conducted on the SCN population isolated from each experimental location to determine how well the nematode population in the field can reproduce on PI 88788, Peking, and the other sources of SCN resistance. The results of these experiments

provide insight into the relationship between SCN reproduction on resistant soybean varieties and yield. Almost all SCN-resistant varieties in the experiments have the PI 88788 source of SCN resistance, and the SCN populations in many of the fields have greater than 10 percent reproduction on PI 88788. Yet soybean varieties with PI 88788 SCN resistance usually produce high yields in these experiments. Often, the PI 88788 SCN-resistant varieties yield significantly more than varieties with Peking as the source of SCN resistance in fields where the SCN populations have 25 to 35 percent reproduction on PI 88788 and very low (<2 percent) reproduction on Peking. Overall, SCN-resistant varieties keep SCN egg population densities from increasing during the season, even in fields with SCN populations that have >10 percent reproduction on PI 88788. But in-season changes in SCN population densities can vary significantly in an experiment among varieties with PI 88788 SCN resistance. Some resistant varieties allow high SCN reproduction in a single experiment, and some allow high SCN reproduction at multiple locations. Yields of resistant varieties that allow high SCN reproduction are among the lowest in some experiments, but not always. The results of these experiments show that growing soybean varieties with PI 88788 SCN resistance in fields infested with SCN populations that have greater than 10 percent reproduction on PI 88788 does not necessarily result in low yields or yields lower than varieties with Peking SCN resistance, at least not under the conditions in which these variety trial experiments were conducted. A challenge for researchers and those who advise farmers is to identify or predict specific conditions (SCN egg population density, rainfall, edaphic factors, etc.) under which soybean varieties with PI 88788 SCN resistance can produce high yields in fields infested with SCN populations with elevated reproduction on PI 88788.

Soybean Lines Evaluated for Resistance to Reniform Nematode

Salliana R. Stetina, James R. Smith, and Jeffery D. Ray

USDA ARS Crop Genetics Research Unit, Stoneville, MS 38776

Seventy-four wild and domestic soybean (*Glycine max* and *G. soja*) lines were evaluated for resistance to reniform nematode (*Rotylenchulus reniformis*) in growth chamber tests with a day length of 16 hours and temperature held constant at 28 C. Several entries for which reactions to reniform nematode were previously reported were included to serve as controls. Due to space limitations, entries were divided into five sets, and each line was evaluated in two separate screenings. The experimental design for each screening was a completely randomized design with 5 replications. A single plant of each soybean line was established in a container filled with 120 cm^3 of a steam-sterilized soil mixture. Upon stand establishment (approximately 5 days after planting), 500 reniform nematodes suspended in 1 ml water were added to the soil in each container. A second inoculation was conducted one week later resulting in a total inoculum level of 1,000 nematodes (mixed vermiform life stages) per container. Root infection was measured

four weeks after the second inoculation. Plant roots were separated from soil, stained with red food coloring using standard protocols, and the number of swollen females attached to the roots counted. Root fresh weights were determined and counts were expressed as females per gram of root to adjust for differences in size of the root systems. Classification of entries was based on the percentage of infection as compared to the susceptible genotypes Morsoy RTS4706N (sets 1 and 3), Delta King DK4968 (sets 1, 2, and 3), Braxton (sets 4 and 5), and PI 88788 (sets 4 and 5): nematode index <10% = resistant, 10-30% = moderately resistant, 31-60% = moderately susceptible, and >60% = susceptible. Both relative infection and consistency of phenotype across tests contributed to identification of the best materials. One accession, PI 404166, was resistant to reniform nematode, consistent with previous reports. Twenty-two entries were moderately resistant. Of these, 8 entries were previously untested, so this study is the first to document moderate resistance to reniform nematode in the breeding lines DS 97-84-1, 02011-126-1-1-2-1, and 02011-126-1-1-5-1; released germplasm lines DS-880 and DS4-SCN05; and accessions PI 417077, PI 507354, and PI 567516 C. A total of 30 entries were moderately susceptible, with 16 entries previously untested including registered germplasm line SS93-6181, the breeding lines LG01-5087-5, 02016-1-5-1-4, 02016-1-5-1-1, 02016-2-3-1-3-1, 02016-1-5-1-2, JDR-6662, and JDR-6676; the cultivars Clark and 5601T, non-nodulating 'Clark' isoline PI 547419, and accessions PI 417321, PI 467312, PI 594692, PI 561287 B, and PI 597413. Twenty-one entries were susceptible with 11 entries previously untested. Cultivars 5002T, Stafford, Lee 74 (parent cultivar Lee previously reported as susceptible), and Jackson; accessions PI 416937, PI 417274, PI 423941, PI 587982 A, and PI 603751 A; registered germplasm line D68-0099 (derived from susceptible cultivar Lee), and wild *Glycine soja* accession PI 468916 were classified as susceptible. Results from this study were inconsistent with previous reports in that 9 entries rated as moderately susceptible and 4 entries rated as susceptible in this test were reported as resistant to reniform nematode by other researchers. Identification of resistance is the first step in developing resistant soybean cultivars that will benefit growers in the Mid South, where soybean acreage on reniform nematode-infested fields has increased in recent years.

Graduate Student Competition

Fungicide resistance in *Cercospora kikuchii*, a major pathogen of Louisiana soybean.

P. Price, M. A. Purvis, C. Robertson, G. B. Padgett, and R. W. Schneider
LSU AgCenter

Purple seed stain and Cercospora leaf blight, caused by *Cercospora kikuchii*, are significant diseases of soybean worldwide. The disease has become increasingly difficult to manage in Louisiana over the past several years. Therefore, to determine if resistance to recommended fungicides exists in *C. kikuchii*, evaluations were conducted with Louisiana populations from 2000 and 2011.

Seed and leaf isolates (176 total) were collected in 2000 from three locations (East Baton Rouge, Rapides, and Franklin Parishes) in Louisiana, and were compared to foliar isolates (160 total) collected in 2011 from 21 parishes throughout the state. For these studies, isolates were transferred to PDA amended with the following fungicides and concentrations: thiophanate-methyl (0 and 5µg/ml), azoxystrobin (0, .0001, .001, .01, .1, 1, 10, and 100 µg/ml), and pyraclostrobin (0, .00001, .0001, .001, .01, .1, 1, and 10 µg/ml). Relative differences in radial growth from controls were calculated as percentages, and effective concentrations (EC_{50}) were determined using linear interpolation of these values.

A discriminatory dose of 5 µg/ml of thiophanate-methyl was used to distinguish between sensitive and resistant isolates in this study. The frequencies of resistant isolates from the years 2000 and 2011 were 23.3 and 44.8 percent, respectively, and were dependent on year (d.f.=1; X^2=15.9811; P=<.0001).

When subjected to azoxystrobin, 50 isolates selected at random from the year 2000 had EC_{50} values ranging from 0.026 to .0356 with a mean of 0.102 µg/ml. The baseline distribution of EC_{50} values was skewed with a long tail towards the less-sensitive end of the spectrum. Fifty-three isolates selected at random from 2011 had EC_{50} values ranging from 0.010 to 165.9 with a mean of 70.49 µg/ml. A significant 700-fold difference in fungicide sensitivity was observed between the two populations (d.f.=52, t=-10.42, P <.0001).

When subjected to pyraclostrobin, EC_{50} values selected at random from the year 2000 ranged from 0.0003 to 0.1034 with a mean of 0.0174 µg/ml. The baseline distribution of EC_{50} values was also skewed with a long tail towards the less-sensitive end of the spectrum. Forty-nine isolates selected at random from 2011 had EC_{50} values ranging from 0.0087 to 27.38 with a mean of 10.06 µg/ml. A significant 60-fold difference in fungicide sensitivity was observed between the two populations (d.f.=48.001, t=-12.01, P <.0001).

Based on the results from this research, *C. kikuchii* populations resistant to thiophanate-methyl have existed in Louisiana since 2000 and have increased in frequency over the past 11 years.

Furthermore, when compared to the baselines, sensitivities of *C. kikuchii* to azoxystrobin and pyraclostrobin have significantly decreased since 2000.

Variation in the Internal Transcribed Spacer (ITS) Region within *Phakopsora pachyrhizi* and Implications for Currently Used Molecular Diagnostic Assays

T.A. Rush[1], B. Kennedy[1], A. Taggart[1], G. Heller[1], M. Toome[1], G.L. Hartman [2], R.W. Schneider[1], and M.C. Aime[1]

[1]Louisiana State University Agricultural Center, Department of Plant Pathology and Crop Physiology, Baton Rouge, LA 70803; [2]USDA-ARS, National Soybean Research Center, Department of Crop Sciences, University of Illinois, Urbana 61801

Before soybean rust (SBR) was discovered in the continental United States in 2004 conventional and quantitative real-time PCR (qPCR) assays were developed by Frederick et al. (Phytopathology 92:217-227, 2002) and, after the introduction of SBR, by Barnes et al. (Phytopathology 99:328-338, 2009). The Frederick et al. (2002) assay, which was based upon the internal transcribed spacer (ITS) rDNA locus, was used to detect the pathogen, *Phakopsora pachyrhizi*, and to differentiate between it and the other causal agent of rust on soybeans, *P. meibomiae*, in infected tissue. This assay did not need to discriminate between the SBR pathogen and additional *Phakopsora* species that did not infect soybeans. Since then a large aerobiological study was conducted by Isard et al. (Plant Dis. 95:1346-1357, 2011) to monitor the dispersal of the pathogen through rain water assays in samples that were collected at numerous sites in the U.S. Toward this end the Barnes et al. (2009) qPCR assay was developed, which could detect a single spore of *P. pachyrhizi* in rain water. In developing the primers and probe for this assay, Barnes et al. (2009) used the same infected soybean tissue samples that were used by Frederick et al. (2002) along with 30 new samples of which 27 were from Florida. However, the Barnes et al. (2009) assay, which was used in the spore dispersal study, did not always detect spores in rain water collected from areas with high disease incidences. The goal of the current project was to assess variation within the ITS region of the pathogen.

Numerous samples of infected soybean and kudzu leaves were collected throughout the southern United States. DNA was extracted, and the ITS region was amplified, cloned and sequenced. The sequences were compared to GenBank accessions that were used in the Frederick and Barnes assays. The primers and probes of both assays were in the highly variable ITS regions, however both assays detected 95% of the cloned DNA in our collection. Maximum likelihood, maximum parsimony, and neighbor-joining analyses showed substantial ITS diversity among samples that were collected in the southern United States. Moreover, ITS variation was found among sori from a single leaf. This diversity was attributable to single nucleotide polymorphisms (SNP) and indels of different lengths. Cloned asexual reproduction from a single source introduction may not be the best explanation for the level of variation observed. Other explanations include the existence of an extant sexual stage or continual re-introduction of the SBR pathogen from the Caribbean or Central and South America. A maximum likelihood tree will be presented and discussed.

Theoretical disease and decision model for fungicide applications for soybean rust

Heather Marie Young[1], D. L. Wright[1], N. S. Dufault[2], and J. J. Marois[1]

[1]University of Florida, North Florida Research and Education Center, Quincy, FL

[2]University of Florida, Plant Pathology Department, Gainesville, FL

Phakopsora pachyrhizi, the causal agent of soybean rust (SBR), is a potentially devastating disease of soybean in the U.S. Fungicides can be applied to avoid yield loss, but are expensive and to be cost efficient growers need to know when application will be most beneficial. During the winter months, in the U.S., the pathogen is limited to the southeastern states on the alternative host kudzu. This source is critical to providing the initial inoculum for the epidemic the following year. To better understand and manage the SBR epidemic in north Florida, which can potentially produce inoculum for other soybean producing areas of the country, theoretical disease models and application decision model were developed. The disease model utilized estimates of disease development and spore escape (Pivonia and Yang, 2006; Andrade et al., 2009) coupled with data from detached leaf assays assessing sporulation of SBR development on kudzu and soybean. The application decision model was developed based on SBR presence and hours of leaf wetness or amount of cumulative rain. Validation of these models in 2009, 2010, and 2011 will be discussed.

Relationship Between Stink Bug Damaged Seed and the Incidence of *Phomopsis longicolla* in the Mississippi Soybean Production System

J. Jones[1], A. Catchot[1], F. Musser[1], T. Allen[2], M. Tomaso-Peterson[1], and J. Gore[2]
[1]Department of Biochemistry, Molecular Biology, Entomology and Plant Pathology, Mississippi State University, MS;
[2]Mississippi State University, Delta Research and Extension Center, Stoneville, MS.

Every year, insect damage and disease pressure are subjects of great concern among soybean producers throughout Mississippi. Among the more important pests threatening yield and quality in soybean are stink bugs (Hemiptera: Pentatomidae) and members from the Diaporthe-Phomopsis complex, particularly *Phomopsis longicolla*. The objective of this research was to determine if a relationship exists between stink bug damage and the frequency of recoverable *Phomopsis longicolla* in the Mississippi soybean production system.

In 2010, lab tests confirmed that stink bugs are capable of moving *Phomopsis longicolla* from one point to another, but the duration of the fungus remaining in contact with the insect is still in question. In addition, field cage studies indicated that soybean exposed to stink bug feeding combined with *P. longicolla* inoculation significantly reduced yield by approximately 17% when compared to individual applications of stink bugs or *P. longicolla* and the untreated check ($F = 6.46$, $df = 3, 9$, $P = 0.0127$; $\alpha = 0.05$). Soybean sleeve cage trials in 2011 further confirmed that

stink bug damaged seed had a significantly greater incidence of fungi compared to undamaged seed ($F = 13.38$, $df = 3, 6$, $P = 0.0046$; $\alpha = 0.05$).

A two year survey was conducted to determine if a relationship exists between stink bug damaged soybean seed and the incidence of *P. longicolla* in Mississippi soybean fields. In both years soybean seed exhibiting stink bug damage were more likely to be infested with *P. longicolla* ($F = 8.59$, $df = 2, 465$, $P = 0.0002$; $\alpha = 0.05$); and other fungi ($F = 48.73$, $df = 2, 457$, $P = < 0.0001$; $\alpha = 0.05$), when compared to undamaged seed. In addition, seed damaged by means other than stink bug feeding had a greater incidence of *P. longicolla* and other fungi compared to undamaged seed. Moreover, data confirmed that soybean seed collected from the bottom third of soybean plants were more likely to be infected with *P. longicolla* in comparison to seed collected from the top third of the soybean plant ($F = 3.74$, $df = 2, 465$, $P = 0.0246$; $\alpha = 0.05$).

Spatial Assessment of *Rhizoctonia solani* In Fields Undergoing Rice/Soybean Rotations

T.N. Spurlock[1], C.S. Rothrock[1], and W. S. Monfort[2]

University of Arkansas, Clemson University

Aerial blight is a single cycle disease caused by *Rhizoctonia solani* AG1-IA. This pathogen also causes sheath blight in rice. Many soybean fields in Arkansas are rotated with rice in annual rotations ensuring a source of inoculum each season. In 2009, soil assays using a toothpick baiting method and plant sampling on GPS positions in fields undergoing rice and soybean rotations was initiated. The objective was to test the hypothesis that early-season inoculum recovery should relate to seedling colonization by *R. solani* and subsequently relate to disease development. Disease assessments also were made on a spatial scale. Methodology was evaluated and refined each year to include more GPS positions on a smaller scale and controlling of soil moisture for toothpick baiting to encourage saprophytic growth of the fungus. Soil assays and plant sampling in 2011 resulted in significant distribution of *R. solani* 1-IA in two fields in eastern Arkansas. In both instances, *R. solani* was significantly dispersed using nearest neighbor statistics about a minimum enclosing rectangle ($P<.0001$). In a field near Hazen, levy maps indicated the levy position could be influencing the artificial nature of dispersion. In a field near Stuttgart, levy maps indicated the greatest inoculum potential lied in the lower elevations compared to higher elevations. Directional distribution ellipses for both distributions of *R. solani* AG1-IA indicated agreement with drainage in both fields. This spatial distribution was not associated with all isolates of *R. solani*. The spatial distribution of the early-season inoculum potential of *R. solani* AG1-IA reiterates the idea that inoculum in the form of sclerotia and hyphae associated with crop residue when the field is planted in rice may be floating and collecting at lower points within the levys. These data point to the influence of drainage on inoculum potential of the pathogen and are an early indication of the potential for precision management of Aerial blight of soybean through targeted within field scouting or fungicide sprays.

Presentation Abstracts

Sustaining the Pest Information Platform for Extension and Education with Support from Industry

(I-PIPE)

Scott Isard, The Pennsylvania State University

The I-PIPE is the Industry Pest Information Platform for Extension and Education. It is an information technology (IT) platform which provides industry participants with tools and models for managing and analyzing data in order to generate products and commentary in support of commercial agricultural decision making and food security.

I-PIPE was designed with the following goals: (i) Integration with government and university IT platforms (e.g., SBR-PIPE, CRIP, NAPFAST, SCOPE), (ii) Promote data sharing among agricultural stakeholders, (iii) Support compliance with phytosanitary regulations and (iv) Facilitate the monitoring of endemic and exotic pests.

SBR-PIPE will integrate seamlessly into the I-PIPE for the 2012 season. We do not anticipate that there will be any changes that are apparent to Extension Specialists and other users. We have also expanded the system to include observations and commentary on frogeye leaf spot caused by *Cercospora sojina*.

Screening Germplasm for Resistance to Phomopsis Seed Decay:

Joint Effort from USDA and University Scientists

Shuxian Li[1], Gabe Sciumbato[2], Pengyin Chen[3], John Rupe[3], Allen Wrather[4],

James R. Smith[1], and Randall L. Nelson[5]

[1]USDA-ARS, Crop Genetics Research Unit Stoneville, MS; [2]Mississippi State University, Stoneville; MS, [3]University of Arkansas, Fayetteville, AR; [4]University of Missouri, Portageville, MO; [5]USDA-ARS, Soybean/Maize Germplasm, Pathology, Genetics Research Unit, Urbana, IL

Soybean Phomopsis seed decay (PSD) causes poor seed quality and suppresses yield in most soybean-growing states in the United States. In 2009, PSD caused yield loss of over 12 million bushels in 16 southern states. The disease is primarily caused by *Phomopsis longicolla* along with other *Phomopsis* and *Diaporthe* spp. Few soybean cultivars currently available for planting in the U. S. have resistance to PSD. To identify new sources of resistance to PSD, 135 soybean germplasm lines (maturity groups III, IV, and V), including PSD resistant and susceptible checks from 28 countries, were field screened by natural

infection in 2009 at Kibler, AR, Stoneville, MS and Portageville, MO. Based on seed assay in 2009, 42 lines along with six resistant and susceptible checks were selected and field-tested with inoculated and non-inoculated treatments in these states in 2010. In addition, seed of 208 representative maturity group V soybean lines, obtained from the USDA Soybean Germplasm Collection in 2006, were plated and assayed for the percentage of Phomopsis seed infection. Based on the disease data in 2006, 122 lines without Phomopsis seed infection were selected and field-screened naturally in Stoneville, MS in 2007. Based on the results of seed assays from 2006 and 2007, 14 lines were selected for further evaluation with inoculated and non-inoculated treatments in 2008 and 2009. In 2009, frequent rainfall during seed maturation led to high levels of seed infection by Phomopsis (up to 80%) and other fungal pathogens for most soybean lines but several lines were identified that had low percentage of seed infection, good visual quality, and high germination rates. These resistant sources will be used to develop cultivars resistant to PSD.

Effect of Storage on Soybean Seed Quality and Emergence

J.C. Rupe[1], R.E. Cartwright[2], J. Robinson[3], K. Cochran[1], R. Holland[1], S. Goeke[1], A. Palmer[4], S. Steger[1]

[1]Department of Plant Pathology, University of Arkansas, Fayetteville, AR; [2]Cooperative Extension Service, University of Arkansas, Little Rock, AR; [3]Department of Human and Community Resource Development, Ohio State University, Columbus, OH; [4]Arkansas State Plant Board, Little Rock, AR

Stand establishment in late planted soybean is a common problem in Arkansas and may be due to loss of vigor in storage. Soybean seed can easily loose vigor in storage especially with elevated temperatures and humidities. Since the seed for late plantings remains in the warehouse into the early part of the summer when temperatures are rising, there was concern that these seed were losing vigor in storage. To determine if soybean seed was losing vigor in storage, two cultivars, UA 4910 and Osage were stored in a commercial warehouse in DeWitt, AR, and at the Arkansas State Plant Board (ASPB), Little Rock, AR, in a controlled environment. In the warehouse, seed was placed in an areas deemed by the seedsman to be a good, bad, or like on-farm storage site. At all sites, seed was stored in 25 kg seed bags, each bag represented a replication and there were four replications. Temperature and relative humidity was measured inside and outside the bags ( ). Seed was sampled every two weeks from 1 April until 15 August. The seed was assayed for standard germination (SG), accelerated aging (AA), and field emergence. For SG and AA, standard protocols were used. For field emergence, 200 seed from each replication was planted at the Vegetable Research Station, Kibler, AR. Adequate soil moisture was maintained with overhead irrigation. Stands were counted at two and four weeks after planting.

At the beginning of the experiment, SG and AA for UA 4910 were 87 and 78%, respectively, and for Osage were 81 and 76%, respectively. At the ASPB, within the bag temperatures were approximately 20 C and relative humidity was 60% and there was little effect on SG and only a slight reduction (10%) in AA for the duration of the experiment. At the seed warehouse in the good site, temperatures rose from 20 C to 30 to 35 C beginning in June. RH held steady at 60%.

There was little effect on SG, but AA began to decline in June reaching 42 and 43% for UA4910 and Osage, respectively. At the bad site, temperature patterns were similar to the good site, but RH dropped in June to around 50% for the rest of the test. There was no effect on SG, but AA declined to 54 and 55% for U4910 and Osage, respectively. At the on-farm site, temperatures rose to about 30C in June. Relative humidity rose throughout the season reaching over 70% in August. There was little effect on SG, but AA fell to 5 and 14 % for UA 4910 and Osage, respectively. Emergence was high with all treatments until 30 June when there was almost no emergence. This corresponded to extremely high air temperatures, but once temperatures cooled, emergence improved and reflected AA results.

Both losses in seed vigor and extreme soil temperatures may be involved in stand failures in late planted soybean. Growers and seedsmen should avoid storing soybean seed where temperatures and humidites are likely to be high such as on-farm storage and avoid periods of extreme heat at planting.

Foliar Fungicides to Prevent Yield Loss Attributed to Aerial Web Blight in Mississippi: 2010 and 2011

T.W. Allen[1], M.A. Blaine[2], B. White[2], and W.F. Moore[1,2]
[1]Mississippi State University, Delta Research and Extension Center, Stoneville, MS
[2]Southern Ag Consulting, Incorporated, Starkville, MS

During the hot and generally dry 2010 and 2011 seasons, aerial web blight (*Rhizoctonia solani* Kuhn) was one of the most prominent soybean diseases in east and northeast Mississippi. Typically, aerial web blight has been of greatest concern in the Delta, where a rice soybean rotation is common. However, given a conducive environment and the overall size and general type of soybean fields encountered throughout eastern MS (generally less than 100 acres in size with trees surrounding three sides) aerial web blight reached epidemic levels.

During 2010 and 2011, two fields were selected for fungicide efficacy trials. In the past, identifying producer fields for fungicide trials was made difficult by the sporadic nature of the disease. However, two fields were identified (one each in 2010 and 2011) whereby aerial web blight was occurring in a cosmopolitan fashion. Two trials were conducted in each field. In 2010 (Noxubee County, MS) trials considered several experimental products from BASF compared with some "standard" fungicide products for aerial blight management (e.g. azoxystrobin, azoxystrobin + propiconazole) and pyraclostrobin, which in the past had not proven to be effective at preventing yield loss from *R. solani*. One of the experimental fungicides resulted in a significant increase in yield, ranging from 19 to 22% compared to the untreated check, regardless of application rate. The second trial compared different rates and timings (R5 alone and R5 fb R5.5) of pyraclostrobin, trifloxystrobin, and trifloxystrobin + propiconazole. Only application with trifloxystrobin applied at R5 resulted in a significant yield increase (22%) compared to the untreated check. However, from a producer standpoint, all fungicide applications compared in the two tests resulted in an economical return-on-investment by preventing yield loss.

In 2011 (Clay County, MS), two trials were conducted to determine the outcome of fungicide application on yield loss as a result of aerial web blight. In the first trial, comparisons were made between two different rates of pyraclostrobin (4 or 6 oz), an experimental product from BASF, azoxystrobin + propiconazole (Quilt Xcel) and a new product from BASF, Priaxor (pyraclostrobin + Xemium). In the first trial, when comparing pyraclostrobin, BAS 700, azoxystrobin + propiconazole, and Priaxor, yield as a result of fungicide application was significantly greater than the untreated check and ranged from a 20 to 27% increase. In the second test, treatment with azoxystrobin resulted in a significant, 11% increase in yield compared to the untreated check. Similar to 2010, a fungicide application, regardless of product, reduced the risk of yield loss and was an economical management decision.

Mycovirus-induced hypovirulence as an alternative means to control fungal diseases of soybean

Said A. Ghabrial and Jiatao Xie

Department of Plant Pathology, University of Kentucky

Mycoviruses (or fungal viruses) are prevalent in all major groups of plant pathogenic fungi. Although many of dsRNA mycoviruses have been reported to be associated with symptomless infections of their hosts, there are well-documented cases for mycoviruses that induce hypovirulence in their plant pathogenic fungal hosts and are currently being exploited as means of biological control for combating plant diseases. Mycovirus-hypovirulent fungal systems present an opportunity to dissect the molecular basis of virulence in phytopathogenic fungi.

Sclerotinia sclerotiorum, the white mold pathogen, is an economically important pathogenic fungus worldwide and is emerging as an excellent fungal host model for studying virus/host interactions. This is due to the fact an increasing number of diverse viruses have been identified from various hypovirulent strains of *S. sclerotiorum*. The list of these diverse viruses includes those with dsRNA as well as single-stranded RNA genomes and also includes the only known geminivirus-related mycovirus with ssDNA genome. Methods for transfection of fungal protoplasts with purified virions or transcripts from cloned viral cDNA are established for some of these viruses.

Two examples of mycoviruses that induce transmissible hypovirulence in their *S. sclerotinia* host isolates will be discussed. The first is a partitivirus (family *Partitivirus*, designated Sclerotinia scleoritiorum partitivirus 1 (SsPV1). SsPV1 represents the first known partitivirus that induces hypovirulence in its host. Hypovirulence and SsPV1 could be co-transmitted from a hypovirulent strain to a virulent one via hyphal anastomosis converting the latter to hypovirulence. Furthermore, purified SsPV1 virions can transfect protoplasts from virus-free virulent fungal isolate and thereby the colonies regenerated protoplast exhibit hypovirulent phenotypes.

We have recently reported the molecular characterization of two novel mitoviruses, Sclerotinia sclerotiorum mitovirus 1 (SsMV1) and SsMV2 and described the biological properties of their hypovirulent host, strain KL-1 of *S. sclerotiorum*. The full-length nucleotide sequences of the two mitoviruses were determined and their genome organizations were characterized.

Mitochondrial codon usage revealed that the genome of each of the two mitoviruses comprises a single unique ORF. BLAST searches with the deduced aa sequences showed that SsMV1 and SsMV2 each encodes a putative RdRp with six conserved RdRp motifs (I-VI). The RdRps of SsMV1 and SsMV2 share only 30-40% amino acid sequence identity with some other known mitoviruses. Based on these results as well as those of phylogenetic analysis and genome organization, SsMV1 and SsMV2 represent two new members of the genus *Mitovirus* in the family *Narnaviridae*.

Managing Cercospora Blight in Louisiana – Facing New Challenges

G.B. Padgett[1], P. Price [1], B. Blanche[2], R.A. Schneider[1], C.A. Hollier[1], and M.A. Purvis[1]. [1]LSU AgCenter, Baton Rouge, LA 70803; and [2]Dow AgroSciences.

Cercospora leaf blight (CLB) caused by *Cercospora kikuchii* is a major disease impacting soybean produced in Louisiana. Loss estimates for 2007 to 2010 range from 0.5 to 2%. Currently, individual practices for managing this disease have limited impact on incidence and severity. The efficacy of fungicides that were once highly effective has diminished over the past several years. Genetic resistance to this pathogen exists, but has not been fully characterized in commercial varieties. Based on preliminary disease ratings taken on LSU AgCenter Official Variety Tests' (OVT), certain varieties have some resistance to *C. kikuchii.* However, the genetic variability in this pathogen population is very diverse and it is not known if this resistance will be stable over time. Therefore, LSU AgCenter scientists continue to evaluate varieties in the OVTs. Additional efforts have been initiated to evaluate the combined impact of genetic resistance and fungicides on CFB development. Beginning in 2010, the varieties entered in the OVT located at the Dean Lee Research Station near Alexandria, LA were evaluated with or without a fungicide application. This is intended to provide producers with information on the response of each variety to a fungicide relative to disease incidence and severity. Disease ratings in 2010 and 2011 varied considerably, and results for individual varieties are inconclusive. However, when treated with a fungicide and compared to non-treated, varieties in the late group IVs yielded an average 2.4 bu/A more, but disease incidence and severity did not differ. This research was prompted by results from studies conducted at the Macon Ridge, Rice, and Central Research Stations in 2003 and 2004. Selected varieties were not sprayed and another set of the same varieties received multiple fungicide applications beginning at pod development and continuing through R7. Fungicides were effective for limiting disease incidence and severity; however, this did not always correlate to higher yields. When averaged across all varieties, disease severity (CLB, frogeye, and pod diseases) was less in fungicide-treated varieties. The average yield of treated varieties was higher in 2003, but no differences were observed in 2004. Ongoing research evaluates the impact of new fungicides and application timings on CLB. Based on results, early applications at R1 and triazole fungicides may have utility for managing CLB. Hopefully, this research can be used to develop disease management strategies utilizing genetic resistance and fungicides to reduce the impact of *Cercospora* leaf blight in soybean.

Seasonal Progress of Charcoal Rot and Its Impact on Soybean Productivity

Alemu Mengistu

USDA-ARS, Crop Genetics Research Unit, Jackson, TN

The seasonal progress of charcoal rot (*Macrophomina phaseolina*) was measured for two growing seasons in four separate experiments: irrigated inoculated, irrigated non-inoculated, non-irrigated inoculated and non-irrigated non-inoculated. Disease was assessed at V5, R1, R3, R5, R6 and R7 growth stages based on colony forming unit (CFU) of *M. phaseolina* and the area under the disease progress curve (AUDPC) recovered from the lower stem and root tissues. The CFU and AUDPC were related to yield loss, which was estimated as the percentage difference in yield between moderately resistant and susceptible cultivars. The population density of *M. phaseolina* increased slowly from the V5 to R6 growth stages and then rapidly from the R6 to R7 growth stages for all genotypes in all the four experiments. Yield loss due to charcoal rot was estimated be upto 33 % in irrigated environments. The extent of yield loss however, was affected by severity of charcoal rot, which in turn was affected by irrigation. Disease severity using CFU accounted for more yield loss variation (42 %) than did the AUDPC (36 %) when used to assess disease. Colony forming unit measured at growth stage R7 was the optimum period for estimating yield loss, and yield loss was consistently measured under irrigation than in non-irrigated environments (Plant Dis. 95: 1159-1166. 2011).

Fungicide Resistance in *Cercospora sojina*: Chapter 2

C. A. Bradley[1], G. R. Zhang[1], R. Ming[1], F. Zeng[1], S. Ebelhar[1], K. Ames[1], G. Olaya[2], H. Sierotzki[2], J. Wullschleger[2], D. Hershman[3], B. Kennedy[3], B. Padgett[4], and M. Newman[5].

[1]University of Illinois; [2]Syngenta Crop Protection; [3]University of Kentucky; [4]LSU AgCenter; and [5]University of Tennessee.

In 2010, isolates of *Cercospora sojina* (the causal fungus of frogeye leaf spot of soybean) were found in areas of southern Illinois, western Kentucky, and western Tennessee that were resistant to quinone outside inhibitor (QoI) fungicides. Additional research was conducted in this area in 2011, which included continued efforts in monitoring for QoI resistant strains of *C. sojina*, field management studies, and characterization of QoI resistant *C. sojina* isolates.

In 2011, newly identified areas with QoI resistant *C. sojina* isolates were five new counties in Kentucky, one county in Missouri, two new counties in Tennessee, and two parishes in Louisiana. These isolates were similar to those identified in 2010, in that they were highly resistant to QoI fungicides in laboratory assays.

A field study was conducted at the University of Illinois – Dixon Springs Agricultural Center (DSAC) to evaluate the efficacy of different fungicides for control of frogeye leaf spot caused by

a QoI fungicide resistant isolate. In this trial, fourteen different fungicide treatments were applied and compared to a non-treated control. Treatments included fungicides from the demethylation inhibitor (DMI) class, the methyl benzimidazole carbamate (MBC) class, and the QoI class. Products that contained only a QoI fungicide did not reduce frogeye leaf spot severity compared to the non-treated control. Fungicides that contained a fungicide from the DMI or MBC class had significantly less frogeye leaf spot severity than the non-treated control. Additional field studies were conducted at the DSAC and at the University of Tennessee Research and Education Center at Milan, TN designed to evaluate the effect of different ratios of QoI fungicide resistant and sensitive *C. sojina* isolates (1:3; 1:1; and 3:1) on frogeye leaf spot severity when different fungicide active ingredients were applied (either pyraclostrobin or flutriafol). At both locations, flutriafol was the most effective in reducing frogeye leaf spot severity under all three QoI fungicide resistant:sensitive ratios of *C. sojina* isolates.

Isolates of QoI fungicide resistant *C. sojina* were characterized and compared to QoI fungicide sensitive *C. sojina* isolates in laboratory and greenhouse studies. The cytochrome b gene was sequenced in both QoI resistant and sensitive isolates in order to identify the mutation responsible for QoI resistance. When compared to QoI sensitive isolates, QoI resistant *C. sojina* isolates had glycine to alanine substitutions at codon 143 in the cytochrome b gene, which is referred to as the G143A mutation. In a greenhouse study, 5 QoI resistant and 5 QoI sensitive *C. sojina* isolates were compared in their ability to cause disease on a susceptible (cv. Blackhawk) and a resistant (cv. Davis) soybean cultivar. On cv. Blackhawk, QoI resistant isolates caused significantly greater disease severity than sensitive isolates 7 to 8 days after inoculation, but no differences in severity were observed after 9 days. On cv. Davis, QoI resistant isolates caused significantly greater disease severity than sensitive isolates 8 to 14 days after inoculation.

Fungicide Efficacy on Strobilurin Resistant *Cercospora sojina* in Soybean

M.A. Newman (1), G.R.Zhang and C.A. Bradley (2)

(1) University of Tennessee Extension, Jackson, TN, USA;

(2) University of Illinois, Urbana, IL, USA

Frogeye leaf spot (FLS) of soybean, caused by the fungus *Cercospora sojina* was found to be highly resistant to strobilurin fungicides in leaf samples collected from a production soybean field in Lauderdale County, Tennessee in 2010. In 2011, soybean samples collected from four or five additional states mainly in the Mid-South have been confirmed to have strobilurin resistant Frogeye leaf spot (SR-FLS). This disease is the number one foliar disease in Tennessee, causing a five-year average yield loss of 3.2% state wide. In laboratory tests conducted at the University of Illinois, spores from the Tennessee isolates of *C. sojina* were found to germinate in the presence of high concentrations of selected strobilurin fungicides.

Two test locations (Milan, TN and Lauderdale Co. TN) were treated with the same 10 fungicides. The RCB plots were planted no-till to the FLS susceptible cultivar Asgrow 4703 in fields with previously high FLS disease. At the Research and Education Center at Milan, TN there was only a very slight indication of SR-FLS, but fairly severe ratings of the "normal" non-resistant FLS. Fungicides with significantly higher yields over the untreated check were: pyraclostrobin + fluxapyroxad (Priaxor) with a 13.3 bu/a increase, tetraconazole (Domark) 11.5 bu/a increase, flutriafol (Topguard) a 11.3 bu/a increase, azoxystrobin + propiconizole (Quilt Xcel) a 10.6 bu/a increase, and picoxystrobin (Aproach) a 9.7 bu/a increase.

At the Lauderdale Co. location where SR-FLS had been severe in 2010, strobilurins alone which were not mixed or premixed with a triazole fungicide gave no control.

Fungicides with significantly higher yields above the untreated control were: tetraconazole with a 6.0 bu/a yield increase, prothioconazole + trifloxystrobin (Stratego YLD) a 5.9 bu/a increase, azoxystrobin + propiconizole a 5.2 bu/a increase, flutriafol a 5.1 bu/a increase, propiconizole a 3.0 bu/a increase and fluoxastrobin (Evito) a 2.5 bu/a increase.

Although this is only one year's testing in the field, it seems fairly clear that strobilurins alone will not adequately control the strobilurin resistant frogeye leaf spot.

Soybean growers are urged to manage FLS through the use of resistant or tolerant varieties, crop rotation and use of effective triazole or triazole-strobilurin fungicides when susceptible varieties are used. Research recently conducted in Tennessee has shown that a number of varieties are resistant or tolerant to FLS. More field tests will be conducted in 2012 to determine which triazoles are most effective and if premixes with strobilurins will be an option for producers.

Strobilurin Resistance in *Rhizoctonia solani* in Soybeans in Louisiana

Clayton A. Hollier

Louisiana State University Agricultural Center

Several soybean and rice fields in Acadia Parish, Louisiana experienced high levels of sheath blight (rating of 9 on a 0-9 SB rating scale) after applying the strobilurin containing products, Quadris and Quilt Xcel. As the non-performance did not appear to be due to misapplication, incorrect timing or incorrect rate, tests were conducted to determine the sensitivity of *Rhizoctonia solani* to azoxystrobin and propiconazole. A shift in azoxystrobin sensitivity was identified and has been classified as "resistance". Guidelines on disease management in both crops have been suggested as well as actions to prevent spread of the pathogen.

SOUTHERN UNITED STATES SOYBEAN DISEASE LOSS ESTIMATE FOR 2011

Compiled by S. R. Koenning Extension Specialist, Department of Plant Pathology, Campus Box 7616, North Carolina State University, Raleigh, NC 27695-7616

Since 1974, soybean disease loss estimates for the Southern United States have been published in the Southern Soybean Disease Workers Proceedings. Summaries of the results from 1977 (8), 1985 and 1986 (4), 1987 (5), 1988 to 1991 (7), 1992 to 1993 (8), 1994 to 1996 (6) have been published. A summary of the results from 1974 to 1994 for the Southern United States was published (9) in 1995, and soybean losses from disease for the top ten producing countries of 1994 was published in 1997(11). An estimate of soybean losses to disease in the US from 1996-1998 was published in 2001(12), and a summary of losses from 1999-2002 was published online in 2003 (10, 11). In 2005, a summary of disease losses for the US from 1996-2004 was published electronically (14) and in 2006 a summary of 2003 to 2005 was published in the Journal of Nematology (15), a 2009 summary of losses from 1996-2007 (16), a 2010 summary focusing on soybean rust was published on line in Plant Health Progress (2). The 2010 disease loss estimates were published in the SSDW proceeding in 2011(1).
\

The loss estimates for 2011 published here were solicited from: Edward Sikora in Alabama, Clifford Coker in Arkansas, Robert Mulrooney in Delaware, Nicholas Dualt in Florida, Bob Kemerait in Georgia, Don Hershman in Kentucky, Clayton Hollier and Boyd Padgett in Louisiana, Arvydas Grybauskas in Maryland, Tom Allen in Mississippi, Allen Wrather in Missouri, Steve Koenning in North Carolina, John Damicone in Oklahoma, John Mueller in South Carolina, Melvin Newman in Tennessee, Tom Isakeit in Texas, and Patrick Phipps in Virginia. Various methods were used to obtain the disease losses, and most individuals used more than one. The methods used were: field surveys, plant disease diagnostic clinic samples, variety trials, and questionnaires to Cooperative Extension staff, research plots, grower demonstrations, private crop consultant reports, foliar fungicide trials, and "pure guess". The production figures for each state were taken from the USDA/NASS website in mid-January of 2011. Production losses were based on estimates of yield in the absence of disease. The formula was: potential production without disease loss = actual production ÷ (1-percent loss) (decimal fraction).

Soybean acreage in the sixteen southern states covered in this report in 2011 was almost 1,000,000 acres less than in 2010 (1). The 2011 average per acre soybean yield increased from that reported 2010 due in large part to late season rain. In 2011, 630 million bushels were harvested from almost 18 million acres in 16 Southern states. The overall average (weighted for acreage) for the 16 reporting states was 36.0 bushels/acre in 2011 while the overall average reported in 2010 was 35.0 bushels/acre (Table 1). The 2011 total acres harvested, average yield in bushels per acre, and total production in each state are presented in Table 1. Percentage loss estimates from each state are specific as to causal organism or the common name of the disease (Table 2). The total average percent disease loss for 2011 was 6.54` % or million bushels in potential production. In 2011, Tennessee reported the greatest percent loss at 14.33 %, followed by Mississippi at 9.05 %.

The estimated reduction of soybean yields is specific as to the causal organism or the common name of the disease (Table 3). The total reduction in soybean yield due to diseases in the 16 southern states was 47.2 million bushels in 2011. The highest average estimated percent loss was caused by charcoal rot at 1.48%. Diseases continued to cause significant loss in soybean production throughout the 16 southern states that participated in this disease loss estimate in 2011. This was a significant improvement over 2010, possibly do the reduced soybean acreage which tends to indicate a greater amount of rotation. It is essential that Extension and University research continue their efforts to discover methods to control these diseases and to educate soybean producers concerning the best methods to prevent yield loss due to soybean diseases.

Table1. Soybean production for 16 Southern states in 2011.

State	acres (1000's)	Production in bu	Bu/Acre
Alabama	295	9,735,000	33
Arkansas	3,270	12,4260,000	38
Delaware	168	6,552,000	39
Florida	16	432,000	27
Georgia	135	2,970,000	22
Kentucky	1,480	57,720,000	39
Louisiana	980	34,300,000	35
Maryland	465	17,903,000	38.5
Mississippi	1,800	70,200,000	39
Missouri	5,200	189,800,000	36.5
North Carolina	1,360	40,800,000	30
Oklahoma	265	3,445,000	13
South Carolina	360	9,000,000	25
Tennessee	1,250	40,000,000	32
Texas	90	1,710,000	19
Virginia	550	21,450,000	39
			31.6 bu/a wt. avg 36 bu/a
	17,684,000	630,277,000	

Literature Cited

1. Koenning, S.R. 2011. Southern United States soybean disease loss estimate for 2010. Proceedings of the Southern Soybean Disease Workers, Thirty Seventh Annual Meeting, Pennsacola, FL. Pp. 1-5.

2. Koenning, S. R., and Wrather, J. A. 2010. Suppression of soybean yield potential in the Continental United States by plant diseases from 2006 to 2009. Plant Health Progress doi:10.1094-2010-1122-01-RS.

3. Koenning, S.R. 2010. Southern United States soybean disease loss estimate for 2009. Proceedings of the Southern Soybean Disease Workers, Thirty Sixth Annual Meeting, Pennsacola, FL. Pp. 1-5.

4. Mulrooney, R. P. 1988. Soybean disease loss estimate for Southern United States in 1985 and 1986. Plant Disease 72:364-365.

5. Mulrooney, R. P. 1988. Soybean disease loss estimate for Southern United States in 1987. Plant Disease 72:915.

6. Pratt, P.W., and A.J. Wrather. 1998. Soybean disease loss estimates for the Southern United States during 1994-1996. Plant Disease 82:114-116.

7. Sciumbato, G. L. 1993. Soybean disease loss estimate for the Southern United States during 1988-1991. Plant Disease 77:954-956.

8. Whitney, G. 1978. Southern states soybean disease loss estimates-1977. Plant Disease Reporter 62:1078-1079.

9. Wrather, J. Allen, A. Y. Chambers, J. A. Fox, W. F. Moore, and G. L. Sciumbato. 1995. Soybean disease loss estimates for the southern United States, 1974 to 1994. Plant Disease 79:1076-1079.

10. Wrather, J. Allen and G. L. Sciumbato. 1995. Soybean disease loss estimates for the Southern Untied States during 1992-1993. Plant Disease 79:84-85.

11. Wrather, J. Allen, T.R. Anderson, D.M. Arsyad, J. Gai, L.D. Ploper, A. Porta-Puglia, H.H. Ram, J.T. Yorinori. 1997. Soybean Disease Loss Estimates for the Top 10 Soybean Producing Countries in 1994. Plant Disease 81:107-110.

12. Wrather, J. A., W.C. Stienstra, and S.R. Koenning. 2001. Soybean disease loss estimates for the United States from 1996-1998. Canadian Journal of Plant Pathology 23:122-131.

13. Wrather, J. A., S. R. Koenning, and T. Anderson. 2003. Effect of diseases on soybean yields in the United States and Ontario 1999-2002. Online. Plant Health Progress doi: 10.1094/PHP-2003-0325-01-RV.

14. Wrather, J. A. and Koenning, S. R. 2005. Soybean disease loss estimates for the United States 1996-2004. http://aes.missouri.edu/delta/research/soyloss.stm

15. Wrather, J. A., and S. R. Koenning. 2006. Estimates of disease effects on soybean yields in the United States 2003-2005. Journal of Nematology 38:173-180.

16. Wrather, J.A., and S. R. Koenning. 2009. Effects of diseases on soybean yields in the United States 1996 to 2007. Plant Health Progress doi10:1094/PHP-2009-0401-01-RS.