National Sclerotinia Initiative

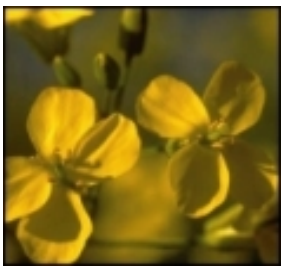
USDA-ARS

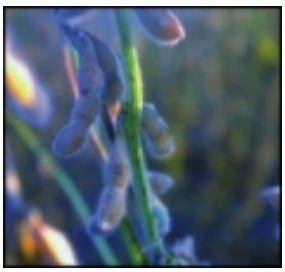
National Sclerotinia Initiative

2017 Annual Meeting

Bloomington, MN

January 18-20, 2017



15th Annual National Sclerotinia Initiative Meeting

January 18-20, 2017

Crowne Plaza Hotel & Suites
Three Appletree Square, Bloomington, MN

AGENDA

2017 Sclerotinia Initiative Annual Meeting
January 18-20, 2017

Wednesday – January 18, 2017

6-8 pm	Poster Session/Reception *(posters are displayed for the entire meeting)*	**Cortland Room**

Thursday – January 19, 2017

7:15 am	Registration/Continental Breakfast	**Cortland Room**
8:15 am	Welcome, Introductions & Meeting Charge – **Mike Foley, USDA-ARS, Fargo, ND**	
8:25 am	Welcome & Update from the Plains Area – **Bryan Kaphammer, USDA-ARS, Fort Collins, CO**	
8:35 am	ARS Office of National Programs Update – **Roy Scott, USDA-ARS, Beltsville, MD**	
8:45 am	Introduction of ***Featured Speaker*** **– Bill Underwood, USDA-ARS, Fargo, ND**	
	Quantitative genomics of resistance and virulence in the interaction of Botrytis cinerea with eudicots – **Daniel Kliebenstein, University of California-Davis, Davis, CA**	
10:00 am	Discussion Break	**Ballroom Foyer**
	Sclerotinia Research Activities – Session 1 **Fireside Room** **Moderator –Dechun Wang, Michigan State University, East Lansing, MI**	
10:30 am	Identification of *Sclerotinia sclerotiorum* virulence determinants relevant to infection of multiple host plants by association mapping – **William Underwood, USDA-ARS, Fargo, ND**	
10:45 am	Enhancing basal resistance to *Sclerotinia sclerotiorum* in Brassica – **Zhonglin Mou, University of Florida, Gainesville, FL**	
11:00 am	Characterizing resistance and pathogenicity genes associated with infection of *B. napus* by *S. sclerotiorum;* Improving resistance to *Sclerotinia sclerotiorum* in spring canola – **Luis del Rio, North Dakota State University, Fargo, ND**	
11:15 am	White mold resistance-QTL: identification, interactions, and fine mapping in common bean – **Phil Miklas, USDA-ARS, Prosser, WA; James Myers, Oregon**	

State University, Corvallis, OR; & Phil McClean, North Dakota State University, Fargo, ND

Noon — Working Lunch — **Cortland Room**

Sclerotinia Research Activities – Session 2 — **Fireside Room**
Moderator – Lili Qi, USDA-ARS, Fargo, ND

1:15 pm — Expression profiling of the pea-Sclerotinia sclerotiorum interaction for genomics assisted breeding – **Martin Chilvers, Michigan State University, East Lansing, MI**

1:30 pm — Using genomic selection to optimize prediction of Sclerotinia and agronomic phenotypes for more efficient breeding – **Brent Hulke, USDA-ARS, Fargo, ND**

1:45 pm — Sclerotinia stalk rot resistance in sunflower: QTL mapping and gene introgression from wild *Helianthus* species – **Lili Qi, USDA-ARS, Fargo, ND**

2:00 pm — Transferring Sclerotinia resistance genes from wild *Helianthus* species into cultivated sunflower –**Gerald Seiler, USDA-ARS, Fargo, ND**

2:15 pm — Improved white mold resistance in dry and snap beans through multi-site screening and pathogen characterization throughout major production areas – **James Steadman & Sydney Everhart, University of Nebraska, Lincoln, NE**

2:30 pm — Phenotypic evaluation and genetic dissection of resistance to Sclerotinia stem rot in soybean – **Dechun Wang, Michigan State University, East Lansing, MI**

2:45 pm — Improved head rot resistance screening in sunflowers and impacts and implications of Sclerotinia infection timing in dry bean, soybean, and sunflower – **Michael Wunsch, North Dakota State University, Carrington, ND**

3:00 pm — Break & Poster Session — **Cortland Room**

Sclerotinia Research Activities – Session 3 — **Fireside Room**
Moderator – Mike Foley, USDA-ARS, Urbana, IL

3:15 pm — New NACA/Proposal Format and Expectations **– Mike Foley, USDA-ARS, Fargo, ND**

4:15 pm — Wrap-up & Adjourn (Dinner on your own)

Friday – January 20, 2017

7:30 am	Steering Committee Breakfast Meeting	**Executive Conference Room**
7:30 am	Continental Breakfast	**Cortland Room**

Sclerotinia Research Activities – Session 4 **Fireside Room**
Moderator – Mike Foley, USDA-ARS, Fargo, ND

9:00 am	***Guest Speaker*** Strategic Planning & Reporting Progress – **Rich Wilson, USDA-ARS, Office of National Programs–Retired, Raleigh, NC**
9:30 am	Strategic Plan Discussion – Writing Team Input/Revisions Annual Reports & Misc. Updates
11:30 am	Working Lunch **Cortland Room**
12:00 pm	Assignment of Additional Tasks & Wrap-up of Initiative Business
1:00 pm	Adjourn (Travel Safely!)

National Sclerotinia Initiative Poster Session

January 18-20, 2017
Cortland Room
Crowne Plaza Hotel & Suites

Epidemiology & Disease Management		
Poster No.	Title	Author(s)
1	Epidemiology of Sclerotinia stem rot of soybean: a South African perspective	L. Rothmann, C. Steyn, N. McLaren
2	Methods of fungicide sensitivity for Sclerotinia sclerotiorum: determination and comparison	T. Miorini, N. Gambhir, E. Nieto, A. Pannullo, T. Hornby, J. Steadman, S. Everhart
3	Optimizing application timing of the fungicide boscalid for management of Sclerotinia in soybeans	M. Wunsch, M. Schaefer, L. Besemann, V. Chapara, P. Gautam T. Tjelde
Genomics		
Poster No.	Title	Author(s)
4	Expression profiling of the pea-Sclerotinia sclerotiorum interaction for genomics assisted breeding	P. Santos, D. Kosma, J. Trinh, X. Zhuang, K. McPhee, J. Wang, M. Chilvers
5	Fungal transcriptome analysis of the *Sclerotinia sclerotiorum-Pisum sativum* interaction	J. wang. A. Rojas, K. McPhee, M. Chilvers
6	Quantative genomics of resistance and virulence in the interaction of Botrytis cinerea with eudicots	D. Kliebenstein, W. Zhang, N. Soltis, C. Caseys, G. Shi, R. Gwinner, S. Atwell, J. Corwin
Pathogen Biology & Development		
Poster No.	Title	Author(s)
7	Characterizing resistance and pathogenicity genes associated with infection of Brassica napus by Sclerotinia sclerotiorum	K. Chittem, L. del Rio Mendoza
8	Dissecting RNA silencing pathways in Sclerotinia sclerotiorum	P. Mochama. S. Marzano
9	Variation among a large and diverse collection of S. sclerotiorum isolates for virulence on sunflower inbred lines	K. Belay, S. Solanki, B. Nelson, R. Brueggerman, M. Dufour, C. Misar, W. Underwood

Variety Development/Germplasm Enhancement		
Poster No.	Title	Author(s)
9	2016 progress on transferring Sclerotinia resistance genes from wild perennial *Helianthus* species into cultivated sunflower	Z. Liu, G. Seiler, X. Cai, K. Rashid, C. Jan
10	Association mapping to identify QTL conferring white mold resistance in the snap bean association panel (SnAPP	H. Arkwazee, J. Hart, J. Myers
11	Characterization and validation of two distinct mechanisms for resistance to Sclerotinia sclerotiorum in pisum sativum	R. Ashtari Mahini, K. McPhee
12	Characterizing a new common bean recombinant inbred population (Unidor/OSU630) for while mold resistance	H. Arkwazee, J. Myers
13	Development of sunflower germplasms resistant to Sclerotinia basal stalk rot derived from bot cultivated and wild and sunflower species	Z. Talukder, G. Seiler, W. Underwood, L. Qi
14	Meta-QTL for resistance to white mold in common bean	H. Arkwazee, J. Myers, P. McClean, R. Vasconcellos, P. Miklas
15	Phenotypic evaluation and genetic dissection of resistance to Sclerotinia stem rot in soybean	Z. Wen, R. Tan, S. Zhang, P. Collins, S. Wani, C. Gu, M. Chilvers, D. Want
16	Sources of white mold resistance derived from wide crosses in common bean and progress in characterization of relevant pathogen isolates	T. Miorini, T. Hornby, A. Pannylo, R. Higgins, S. Everhart, J. Steadman
17	Understanding and improving basal resistance to Sclerotinia sclerotiorum	J. Rollins, Z. Mou
18	Using genomic selection to optimize predication of Sclerotinia and agronomic phenotypes for more efficient breeding	B. Hulke, Q. Gao, N. Kane

2016 Progress on Transferring Sclerotinia Resistance Genes from Wild Perennial *Helianthus* Species into Cultivated Sunflower

Zhao Liu[1], Gerald J. Seiler[2], Xiwen Cai[1], Khalid Y. Rashid[3], and Chao-Chien Jan[2]

[1]Department of Plant Sciences, North Dakota State University, Fargo, ND 58108
[2]USDA-ARS, Northern Crop Science Laboratory, Fargo, ND 58102
[3]Agriculture and Agri-Food Canada, Morden, Manitoba, R6M 1Y5 Canada

Funded Plan of Work: Transferring Sclerotinia Resistance Genes from Wild *Helianthus* Species into Cultivated Sunflower

ABSTRACT:
Wild *Helianthus* species are important genetic resources for the improvement of cultivated sunflower. With the aim to introgress Sclerotinia resistance genes from wild perennials to cultivated sunflower, crosses and backcrosses have been conducted between interspecific amphiploids, hexaploid, tetraploid and diploid perennials with cultivated sunflower (HA 410, HA 441, HA 451 or NMS HA 89). The backcross progenies with 2n=34 chromosomes derived from different crosses were evaluated in replicated trials in 2009-2016. In 2016, 75 families were tested for stalk rot at Carrington, ND and Staples, MN, and 45 families for head rot at Carrington, ND and Staples, MN. Over 30 newly selected early generation families of *H. salicifolius, H. divaricatus, H. occidentalis* and *H. hirsutus* were further tested in replicated stalk rot field trials in 2016. However, the stalk rot trials were not successful, only the results of the head rot trials will be presented. Several families of the first or second retests and new selections had good resistance to head rot. Families with better resistance than the recurrent parents identified in the different trials will be retested in 2017. The pollens of the BC_1F_1s derived from *H. strumosus* and *H. tuberosus* (2n=51) were crossed to NMS HA 89 in 2016 to obtain more BC_2F_1 seeds. The average backcross seed set for *H. tuberosus* (5.36%) was much better than those for *H. strumosus* (0.04%). Previously produced BC_1F_1 seeds (2n=51) of *H. smithii*, *H. atrorubens*, *H. laevigatus*, and *H. pauciflorus* (*rigidus*) with HA 89 were planted in 2016, which were crossed with HA 410 and NMS HA 89, respectively with the BC_2F_1 seeds planted in the greenhouse. These materials will provide additional potential pools of resistance genes and increase the probability of identifying useful resistance QTLs. Crosses between seven Sclerotinia resistant sources and cultivated HA 234 were made in 2016 to develop the recombinant inbred line (RIL) populations for Sclerotinia resistance QTL mapping. Five resistant and six susceptible BC_4F_4/BC_4F_5 progenies derived from *H. californicus* were planted for further analyzing the alien fragments using genomic *in situ* hybridization (GISH) and genotyping by sequencing (GBS) techniques. Thirteen amphiploids derived from eight wild perennial species will be released as germplasms.

Contact Information – Chao-Chien Jan, Sunflower and Plant Biology Research Unit, USDA-ARS-NCSL, 1605 Albrecht Blvd N, Fargo, ND 58102-2765; 701-239-1319; chaochien.jan@ars.usda.gov

Association Mapping to Identify QTL conferring White Mold Resistance in the Snap Bean Association Panel (SnAP)

Haidar Arkwazee[1], John P. Hart[2] and James Myers[1], [1]Oregon State University, Corvallis, OR [2]USDA-ARS, Mayagüez, PR

Funded Plan of Work: White mold resistance-QTL: identification, interactions, and fine mapping in common bean

ABSTRACT:
Common bean (*Phaseolus vulgaris* L.) is one of the most widely grown grain legume crops and is important as dry beans and as snap beans where pods are consumed as a vegetable. White mold, caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, is considered one of the most serious diseases in the bean field and can cause up to 100% yield loss under certain conditions. Previous research indicates that resistance to white mold is quantitative, with multiple resistance alleles each having small individual effect. Most QTL discovered to date have been in dry bean, but snap bean remains an untapped source of potential resistance. We previously conducted association mapping on the Bean CAP Snap Bean Diversity Panel but a newer and larger panel was recently assembled. We conducted a genome wide association study (GWAS) on 376 lines in the Snap Bean Association Panel (SnAP). The panel was phenotyped using the modified seedling straw test repeated three times, and genotyped with 40K SNPs generated through genotyping-by-sequencing (GBS). The straw test score (1-9 scale) across the panel showed a normal distribution where least square means for the straw test score for G122 (resistant check), and OR91G (susceptible check) were 3.33 and 6.41 respectively and 95% confident intervals were 2.23-4.43 and 5.31-7.51 respectively. SNPs with strong associations with white mold resistance QTLs were detected on Pv03, Pv05 and Pv07. Several newly identified snap bean cultivars showed relatively high levels of resistance. The results of this study may facilitate breeding for white mold resistance through the identification of new sources of resistance and in developing markers for marker assisted selection of resistance QTL.

Contact Information:– Haidar Arkwazee, PhD Candidate, Dept. of Horticulture, Oregon State University, 4017 ALS Bldg., Corvallis, ORE 97331-7304, 541-908-0882, haidarh@oregonstate.edu
Dr. James R. Myers, Dept. of Horticulture, Oregon State University, 4017 ALS Bldg., Corvallis, ORE 97331-7304, 541-737-3083, james.myers@oregonstate.edu
John P. Hart, USDA-ARS TARS, 2200 P.A. Campos Ave. Suite 201, Mayagüez, PR 00680, 607-342-7772, john.hart@ars.usda.gov

Characterization and Validation of Two Distinct Mechanisms for Resistance to *Sclerotinia sclerotiorum* in *Pisum sativum*

Rahil Ashtari Mahini, North Dakota State University & Kevin McPhee, North Dakota State University

Funded plan of work: Characterization and Validation of Two Distinct Mechanisms for Resistance to *Sclerotinia sclerotiorum* in *Pisum sativum*

ABSTRACT:
White mold caused by *sclerotinia sclerotiorum* (Lib.) de Bary is one of the main fungal diseases of pea, can causes severe yield losses. Two pea RIL populations consisting of 192 of F_8-derived lines from a cross between Lifter/PI240515 (PRIL-17) and 364 from a cross between PI169603/Medora (PRIL-19) have been established by single seed decent and used for phenotyping and genotyping. Individual RILs were screened and scored for lesion expansion inhibition (LEI) and node transmission inhibition (NTI). Genotype by sequencing (GBS) based on two 96-plex ApeKI GBS library preparations were performed on 186 individuals from each population. Single nucleotide polymorphism (SNP) markers were called using a non-reference GBS SNP calling pipeline, Network-Enabled Analysis (UNEAK), and a high density genetic linkage map created. 14,047 and 22,235 SNPs were called in PRIL-17 and PRIL-19, respectively. After filtering for minor allele frequency >0.01, and missing data per site < 50%, a total of 625 and 664 SNPs were polymorphic in PRIL-17 and PRIL-19, respectively, and were used to generate genetic maps. In PRIL-19, 597 markers were placed on 7 linkage groups spanning 702.8 cM, with an average marker distance of 1.3 cM. The genetic linkage map and the phenotypic data will be used to identify quantitative trait loci associated with LEI and NTI.

Contact Information – Kevin McPhee, Dept. 7670, P.O. Box 6050, 370G Loftsgard Hall, Fargo, ND, 58108, (701) 231-8156, kevin.mcphee@ndsu.edu

Characterizing a New Common Bean Recombinant Inbred Population (Unidor/OSU5630) for White Mold Resistance

Haidar Arkwazee and James Myers, Oregon State University, Corvallis, OR

Funded Plan of Work: White mold resistance-QTL: identification, interactions, and fine mapping in common bean

ABSTRACT:
Common bean (*Phaseolus vulgaris* L.) is an important grain legume crop grown for its dry edible seeds and green pods consumed as a vegetable. White mold, caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, is considered one of the most serious diseases in the bean field that can cause up to 100% yield loss under certain conditions. Unidor, a wax snap bean, showed outstanding white mold resistance in field tests in 2012 and 2013. A recombinant Inbred (RI) population that included 190 F_5 lines was developed by crossing Unidor with OR5630 (a susceptible bush blue lake cultivar) to dissect the genetic of resistance for this new promising line. The population was screened using a modified seedling straw test (scale of 1-9, 1 = resistant) in the greenhouse with three replications. The population showed a normal distribution for the disease score. The disease score least square mean for both parents (Unidor and OSU5630) were 2.78 and 6.65 and 95% confident interval 1.45 - 4.10 and 5.32 -7.97 respectively; while disease score mean for G122, partial resistance check, and OSU91G, susceptible check, were 4.55 and 5.72 and 95% confident interval 3.23 - 5.88 and4.39 - 7.05 respectively.

Contact Information – Haidar Arkwazee, PhD Candidate, Dept. of Horticulture, Oregon State University, 4017 ALS Bldg., Corvallis, ORE 97331-7304, 541-908-0882, haidarh@oregonstate.edu and Dr. James R. Myers, Dept. of Horticulture, Oregon State University, 4017 ALS Bldg., Corvallis, ORE 97331-7304, 541-737-3083, james.myers@oregonstate.edu

Characterizing resistance and pathogenicity genes associated with infection of *B. napus* by *S. sclerotiorum*

Kishore Chittem, and Luis del Río Mendoza, North Dakota State University, Fargo, ND 58102.

Funded Plan of Work: Characterizing resistance and pathogenicity genes associated with infection of *B. napus* by *S. sclerotiorum*

ABSTRACT:
The objective of this project has been to characterize putative resistance and pathogenicity genes associated with infection of *B. napus* by *S. sclerotiorum.* Several potential *S. sclerotiorum* candidate pathogenicity genes were identified from an RNASeq project conducted to study canola – *S. sclerotiorum* interaction funded by the Initiative. Gene knock-out mutants were generated for six genes (SS1G_03845 (thiol methyltransferase), SS1G_05491 (cytochrome P450), SS1G_09997 (Glycosyltransferase), SS1G_14184 (S/T kinase) and metallothionein (SS1G_04760)). Correct replacement of the genes of interest (GOI) by hygromycin phosphotransferase (*hph*) gene in the putative mutants were confirmed by PCR. The effect of deletion of these genes on growth characteristics and pathogenicity/ virulence on canola is currently being evaluated. Cleaved Amplified Polymorphic Sequence markers were developed for four SNPs linked to QTL associated with SSR resistance. High resolution meltcurve (HRM) assays Further, qPCR based high resolution meltcurve (HRM) assays were also developed to facilitate rapid genotyping of these SNPs. These HRM assays are quick and doesn't require the use of restriction digestion to identify the SNPs. Validation of the CAPS and HRM assays on F_2 progenies is underway.

Contact Information – Dr. Luis del Rio, Dept. of Plant Pathology, North Dakota State University, Dept. 7660 P.O. Box 6050, Fargo ND 58108-6050; (701) 231-7073; luis.delrio-mendoza@ndsu.edu

Development of sunflower germplasms resistant to Sclerotinia basal stalk rot derived from both cultivated and wild sunflower species

Zahirul Talukder[1], Gerald Seiler[2], William Underwood[2], and Lili Qi[2]
[1]Department of Plant Sciences, North Dakota State University, Fargo, ND
[2]USDA-ARS, Northern Crop Science Laboratory, Fargo, ND

Funded Plan of Work: Identification of major genes-QTL for Sclerotinia resistance in cultivated sunflower and wild *Helianthus*

ABSTRACT:
Sclerotinia basal stalk rot (BSR) caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, is a devastating disease of sunflower worldwide. Although, no complete resistance has found in sunflower, considerable genetic variation for BSR resistance has been observed among the inbred lines. High levels of BSR resistance have also been identified in wild *Helianthus* species. Here we report the development of highly BSR tolerant germplasms by incorporating genetic factors from partially tolerant breeding lines and wild species. In 2016, we released eight Sclerotinia BSR resistant sunflower germplasms, HA-BSR1 to HA-BSR8. HA-BSR1 is an F_7-derived oilseed maintainer line developed from the cross of HA 441/RHA 439, two cultivated sunflower inbred lines moderately tolerant to Sclerotinia BSR. A four-year mean disease incidence (DI) of HA-BSR1 was 1.6%, significantly lower than that of the parents HA 441 (20.0%) and RHA 439 (12.6%), as well as the susceptible hybrid check Cargill 270 (32.8%) and the resistant hybrid check Croplan 305 (10.4%). Genetic analysis revealed that HA-BSR1 possessed six alleles associated with quantitative trait loci (QTL) conferring resistance to BSR from both parents. The BSR resistant germplasms HA-BSR2, HA-BSR3 to HA-BSR5, and HA-BSR6 to HA-BSR8 were BC_2F_6 derived introgression lines developed from crosses of wild annual *Helianthus* species, *H. petiolaris*, *H. argophyllus*, and *H. praecox*, respectively, with inbred lines HA 89 and HA 458. A four-year mean DI of HA-BSR2 to HA-BSR8 was 2.5, 3.0, 0.8, 1.9, 4.8, 1.2, and 2.2%, respectively compared to 31.0% for HA 89 (recurrent parent), 19.5% for HA 441 (resistant inbred), 36.1% for Cargill 270, and 11.6% for Croplan 305. A whole genome scan of these introgression lines using genotype-by-sequencing (GBS) revealed the presence of *H. petiolaris* segments in linkage group (LG) 8 of HA-BSR2, *H. argophyllus* segments in LGs 8, 9, 10, and 11 of HA-BSR3 and HA-BSR4, in LGs 3 and 10 of HA-BSR5, and *H. praecox* segments in LGs 6, 8, 10, and 11 of HA-BSR6, and in LGs 1, 6, 8, 10, and 11 of HA-BSR7 and HA-BSR8. The introgressed segments in the cultivated sunflower background are assumed to be associated with Sclerotinia BSR resistance. Except for HA-BSR5, the remaining six introgression lines are also homozygously resistant to downy mildew conferred by the Pl_{17} gene derived from HA 458, one of the parents. These lines represent some of the first sunflower germplasms to combine resistance for both Sclerotinia BSR and downy mildew.

Contact Information – Dr. Lili Qi, Sunflower and Plant Biology Research Unit, USDA-ARS Northern Crop Science Laboratory, 1605 Albrecht Blvd., Fargo, ND USA 58102-2765; (701) 239-1351, lili.qi@ars.usda.gov

Dissecting RNA Silencing Pathways in *Sclerotinia sclerotiorum*

Pauline K. Mochama[1], Shin-Yi L. Marzano[1,2]
[1]Biology & Microbiology, [2]Plant Science, South Dakota State University, Brookings, SD

ABSTRACT:
RNA silencing functions as an antiviral defense in fungi. Recent studies have also demonstrated that plant pathogenic fungi use RNA silencing to silence host immunity genes through the delivery of small RNAs that target these genes. Furthermore, this cross-kingdom RNA silencing is bidirectional, with plant hosts capable of delivering small RNAs into fungal cells to silence fungal genes. This host-induced gene silencing, targeting other fungal RNA silencing genes, reduces growth and pathogenicity of the fungal pathogen. These findings demonstrate the importance of RNA silencing mechanisms in white mold growth, spread, and virulence. Innovative fungal control strategies in the near future will likely involve targeting and disrupting RNA silencing pathways in fungal pathogens using RNAs delivered to fungal cells. Therefore, elucidating RNA silencing pathways in *S. sclerotiorum* is an important step in the development of such strategies. This study aims to disrupt key silencing-related genes in *S. sclerotiorum* in order to dissect the role RNA silencing pathways play in this widely distributed fungal pathogen. Homologs of the core eukaryotic RNA silencing genes have been identified in *S. sclerotiorum* including two DCL homologs, two AGO homologs, and three predicted RdRp homologs. Genome editing techniques such as split-marker recombination and CRISPR-Cas9 are being used to generate disruption mutants of these RNA silencing genes. Disruption mutants will then be studied for changes in phenotype, strains. The findings of these studies will broaden our understanding of RNA silencing pathways in *S. sclerotiorum* and shed light on how these pathways can be exploited in the development of robust techniques to manage the spread and virulence of this fungal plant pathogen.

Contact Information – Dr. Shin-Yi Lee Marzano, Biology and Microbiology Department, South Dakota State University, 2140 North Campus Drive, SNP 252 Box 2140D, Brookings, SD 57007-2142; Shinyi.Marzano@sdstate.edu

Epidemiology of Sclerotinia Stem Rot of soybean: A South African perspective

Lisa A. Rothmann, Chrisna Steyn & Neal W. McLaren, University of the Free State, Bloemfontein, South Africa

Funded Plan of Work: Not Applicable

ABSTRACT:
Data provided by the local Crop Estimates Committee (CEC) from 2006-2015, over 43 soybean production localities indicated that locality accounted for 32.98% variation in disease incidence and seasonal variation for 4.75%. Average incidences exceeding 20% over this period were recorded in a number of key production areas. Molecular characterisation of 77 isolates by means of AFLP analysis indicated a relatively uniform geographic distribution, with evidence of genetic diversity within and amongst isolates. The low recombination frequency suggests that the population in South Africa is young and developing. Minimal spanning network confirmed these findings and the dendogram showed high similarity between isolates from within and across provinces. The significance of the genetic diversity in host response and locality interactions still requires elucidation. Resistance in soybean cultivars in inoculated, sequentially planted field experiments showed that ranking according to disease incidence was not correlated and highly significant genotype x environment interactions were recorded. Nonlinear regression analysis identified cultivar responses linearly related to disease potential, highly susceptible at low disease potentials and various degrees of resistance despite increasing disease potentials. Regression parameters indicated disease potential required to initiate disease and subsequent responses. Area under the disease potential curve quantified genotype x environment responses but it could not differentiate between the time of disease onset and the rate of disease response. This methodology proved useful to differentiate between levels of resistance and genotype x environment interactions. Data provided by the ARC-Institute for Soil Water and Climate's Meteorology were used for weather x Sclerotinia incidence analyses of CEC historic data. Respective season's weather data were bulked into periodic running means and multivariate analyses were used to estimate critical growth stages and the length of infection and colonization periods. Analyses identified mid- to late January, 21 day periods associated with R3-R5 critical growth stages. Since details of agronomic practices associated with each survey point were not provided, an initial model was developed and used to identify and realign the most probable R3 growth stages with critical weather conditions for each locality/season data point. The realigned data yielded an improved model, which subsequent to stepwise variable selection was reduced to $Y=3.348*RF-0.3798*RHmn+1.005*Tmn-6.598$ ($R^2=0.82$). An intervention threshold model will be built on the above and the final validation will be done during field trials. It is anticipated that the study will enable an integration of genetic resistance with the optimization of fungicide applications.

Contact Information – Lisa A. Rothmann, Dept. of Plant Sciences, Univ. of the Free State, P.O. Box 339, Bloemfontein, 9300, South Africa; +27-51-401 9681; coetzeeLA@ufs.ac.za

Expression profiling of the pea-*Sclerotinia sclerotiorum* interaction for genomics-assisted breeding

Patrícia Santos, Dylan Kosma, Jasmine Trinh, Dept. of Biochemistry and Molecular Biology, Univ. of Nevada Reno, Reno, ND, Xiaofeng Zhuang, Dept. of Horticulture and Crop Science, Ohio State Univ., Wooster, OH, Kevin McPhee, Dept. of Plant Sciences, North Dakota State Univ., Fargo, ND, and Jie Wang, Martin Chilvers, Dept. of Plant, Soil and Microbial Sciences, Michigan State Univ.

Funded Plan of Work: Expression profiling of the pea-*Sclerotinia sclerotiorum* interaction for genomics assisted breeding

ABSTRACT:
The overall project goal is to utilize genomic resources and techniques to study the host-pathogen interaction of *Pisum sativum* and *Sclerotinia sclerotiorum*, at the genetic level to ultimately improve white mold control. Gene expression profiling was conducted via RNAseq, on a susceptible (Lifter) and a partially resistant (PI240515) line inoculated with *S. sclerotiorum*, during the early stages of infection (i.e. 12, 24 and 48 hpi). Briefly, and based on the expression profile comparison between these cultivars, we found that some of the most highly differentially expressed genes (*PsPOX3-2*, *PsPOX3-3*, *PsCHS-1*, *PsF5H* and *PsEGase*) in PI240515, encoded proteins involved in the reinforcement of cell walls and production of phytoaleaxins, or were pathogenesis-related (PR). Also included in this list of most highly differentially expressed genes, were genes encoding proteins (putatively) involved in jasmonic acid (JA) and flavonoid/anthocyanins biosynthetic processes, oxygen peroxide (H_2O_2) catabolism process and negative regulation of transcription, defense and abscisic acid (ABA) signaling pathway, alongside with several transcription factors (TFs).

We were able to correlate predicted gene expression, specifically the *PsF5H* gene, with the biology of infected plants, i.e., increases of the syringyl (S) monolignol in the inoculated PI240515 cultivar, and begin to unveil how pea plants use the phenylpropanoid pathway to resist infection by *S. sclerotiorum*. Via hormone analysis it was seen that the JA pathway was highly induced in the susceptible Lifter 12 hpi. A signal of JA was also present in the resistant PI240515 plants, however this was much lower than in Lifter, and appeared to peak at 24 hpi. Staining with 3,3'-diaminobenzidine (DAB) was also conducted to identify reactive oxygen species (ROS) accumulation during the interaction. Significantly higher ROS production was found at early time points in the inoculated susceptible Lifter plants. This result led to another interesting finding about the strategies employed by pea plants to fight this specific disease: several genes (*PsHxk-1*, *PsPLD* and *PsBAK-1*), previously linked to cell death-related events, have higher levels of transcripts in infected Lifter than in infected PI240515, and thus may contribute to a rapid development of disease and susceptibility in Lifter.

Contact Information – Dr. Martin Chilvers, Dept. of Plant Soil and Microbial Sciences, Michigan State University, 578 Wilson Rd, CIPS 104, East Lansing, MI 48823; 517-353-9967; chilvers@msu.edu

Fungal transcriptome analysis of the *Sclerotinia sclerotiorum*-*Pisum sativum* interaction

Jie Wang, Alejandro J. Rojas, Patrícia Santos, Michigan State Univ.; Kevin McPhee, Dept. of Plant Sciences, North Dakota State Univ., Fargo, ND, and Martin Chilvers, Dept. of Plant, Soil and Microbial Sciences, Michigan State Univ.

Funded Plan of Work: Expression profiling of the pea-*Sclerotinia sclerotiorum* interaction for genomics assisted breeding

ABSTRACT:
The fungus *Sclerotinia sclerotiorum* is a broad host range necrotrophic pathogen that causes white mold on pea (*Pisum sativum*). Gene expression profiling during infection has been characterized for *Sclerotinia sclerotiorum* on several model plants. However, the *S. sclerotiorum-P. sativum* interaction is still poorly understood. In this study, fungal transcriptomic data was collected *at* three time points: 12 h, 24 h, and 48 h post inoculation (hpi), from susceptible and partially resistant pea cultivars inoculated with *S. sclerotiorum*. As a control, transcriptomic data was also collected from mycelia grown on PDA medium used in the inoculations. Four gene clusters were identified using expression profile analysis, one of the clusters was exclusively up-regulated on PDA, while the remaining three clusters were either associated with the host or sampling time. Gene ontology enrichment analysis demonstrated that 83% of the up-regulated cluster 1 genes were categorized as oxidoreductases at 12 hpi. Whereas, 43% of the up-regulated cluster 2 genes were involved in hydrolysis and transport functions at 24 and 48 hpi. The temporal gene expression profile change may indicate the lifestyle transition of the pathogen from biotroph to necrotroph. In addition, three out of 486 predicted effector candidate genes were up-regulate *in planta* at 24 and 48 hpi. The identification of key genes involved in the *S. sclerotiorum-P. sativum* interaction will facilitate effector assisted breeding for disease management.

Contact Information – Dr. Martin Chilvers, Dept. of Plant Soil and Microbial Sciences, Michigan State University, 578 Wilson Rd, CIPS 104, East Lansing, MI 48823; 517-353-9967; chilvers@msu.edu

Improved head rot resistance screening in sunflowers and impacts & implications of timing of Sclerotinia infection in dry bean, soybean & sunflower

Michael Wunsch, Michael Schaefer, Billy Kraft and Suanne Kallis, NDSU Carrington Research Extension Center; Leonard Besemann, Heidi Eslinger and Kelly Cooper, NDSU Robert Titus Research Farm, Oakes.

Funded Plan of Work:

ABSTRACT:
This project characterized the impact of early versus late Sclerotinia disease development on seed yield and quality in sunflowers and the associated implications for fungicide usage, and it evaluated whether screening for resistance to Sclerotinia head rot of sunflowers could be improved by including shattering of diseased head tissue in disease assessments.

Sunflowers exhibited maximum susceptibility to Sclerotinia head rot when inoculated at R5.6 to R5.9 (60 to 90% of the disk flowers flowering or completed flowering) and developed less disease when inoculated at earlier stages of bloom or when inoculated after heads began reaching R6.0 (ray flowers wilted, bloom complete).

Fungicides applied to the front of sunflower heads through drop nozzles facilitated reductions in Sclerotinia head rot and/or increases in sunflower yield when fungicides were applied prior to inoculation. However, results suggest that residual activity from fungicide applications could be relatively short. In Carrington, Sclerotinia incidence and severity index were reduced nearly 50% when fungicides were applied 1 to 2 days before most sunflower heads were inoculated but were reduced less than 20% when fungicides were applied 4 days before most sunflower heads were inoculated.

Sunflower head tissue exhibiting Sclerotinia head rot often shatters before harvest, dropping seed and sclerotia to the ground, and the susceptibility of sunflowers to shattering of diseased head tissue differed significantly across hybrids at screening nurseries conducted in Carrington and in Oakes. Sclerotinia head rot incidence and severity were strongly correlated between the Oakes and Carrington trials, but susceptibility to shattering was not. Weighting or adjusting Sclerotinia incidence and severity index assessments to include differences in susceptibility to shattering did not improve the ability to predict sunflower yield or contamination of harvested seeds with sclerotia in either the Carrington or Oakes field trials.

The impact of the timing of Sclerotinia disease development on agronomic performance of dry edible beans and soybeans could not be assessed due to a severe hail storm that occurred on July 9. The hail storm completely defoliated the dry beans and soybeans at bloom initiation, and Sclerotinia did not develop.

Results from this project suggest that the application of fungicides through drop nozzles may permit management of Sclerotinia head rot of sunflowers when fungicides are applied within a few days of pathogen infection. Results from the project also suggest that current methodologies for screening sunflowers for resistance to Sclerotinia head rot are unlikely to be improved by quantifying differences in shattering of diseased head tissue across breeding lines or hybrids.

Contact Information – Dr. Michael Wunsch, North Dakota State University Carrington Research Extension Center, PO Box 219, 663 Hwy. 281 N., Carrington, ND 58421-0219; 701-652-2951; michael.wunsch@ndsu.edu

Meta-QTL for Resistance to White Mold in Common Bean

Haidar Arkwazee and James Myers, OSU, Corvallis OR; Phil McClean, NDSU, Fargo, ND; Renato Vasconcellos and Phil Miklas, USDA-ARS, Prosser, WA

Funded Plan of Work: White mold resistance-QTL: identification, interactions, and fine mapping in common bean

ABSTRACT:
White mold, caused by the fungus *Sclerotinia sclerotiorum* (Lib.) de Bary, is a major disease that limits common bean production and quality worldwide. The host-pathogen interaction is complex, with partial resistance in the host inherited as a quantitative trait with low to moderate heritability. A number of QTL have been identified but have not been systematically organized into a framework for resistance. Our objective was to identify meta-QTL conditioning partial resistance to white mold from individual QTL identified across multiple populations and environments. The physical positions for 37 individual QTL were identified across 14 recombinant inbred bi-parental populations (six new, three re-genotyped, and five from the literature). A meta-QTL analysis of the 37 QTL was conducted using the genetic linkage map of Stampede x Red Hawk population as the reference. The 37 QTL condensed into 17 named loci (12 previously named and five new) of which nine were defined as meta-QTL: WM1.1, WM2.2, WM3.1, WM5.4, WM6.2, WM7.1, WM7.4, WM7.5, and WM8.3. The nine meta-QTL had confidence intervals ranging from 0.42 to 5.89 Mb. Candidate genes shown to express under *S. sclerotiorum* infection in other studies, including cell wall receptor kinase, COI1, ethylene responsive transcription factor, peroxidase, and a MYB transcription factor were found within the confidence intervals for five of the meta-QTL. The nine meta-QTL are recommended as potential targets for MAS for partial resistance to white mold in common bean.

Contact Information – Phillip Miklas, USDA-ARS, Grain Legume Genetics and Physiology Research Unit, 24106 N. Bunn Road, Prosser, WA 99350; 509 786-9258; phil.miklas@ars.usda.gov

Methods of fungicide sensitivity for *Sclerotinia sclerotiorum*: determination and comparison

T.J.J. Miorini, N. Gambhir, E. Nieto, A. Pannullo, T. Hornby, J. Steadman, and S. Everhart, University of Nebraska - Lincoln

Funded Plan of Work: Improved white mold resistance in dry and snap beans through multi-site screening and pathogen characterization throughout major production areas

ABSTRACT:
Sclerotinia sclerotiorum causes disease on over 400 species of plants worldwide including important crops and numerous weeds. One method of disease control is well-timed fungicide applications. The intensive use of fungicides, especially site-specific products, can trigger resistance in isolates that may reduce fungicide efficacy. Fungicide sensitivity surveys are used to determine the baseline sensitivity of pathogen populations and for monitoring changes over time. Although various methods exist for determining fungicide sensitivity, no studies have compared these methods to determine if they yield similar or accurate results for *S. sclerotiorum*. In the present study, we compared the plate-dilution, spiral gradient, spore germination, and germ tube elongation methods to calculate the fungicide concentration that causes 50% reduction (EC_{50}) in growth/germination. To compare plate-dilution and spiral gradient methods, we obtained the EC_{50} using both methods for 34 *S. sclerotiorum* isolates to seven fungicides with different modes of action: iprodione, procymidone (dicarboximides), thiophanate methyl (MBC), fluazinam (phenyl-pyridinamine), boscalid (SDHI), prothioconazole (DMI), and pyraclostrobin (QoI). Preliminary results of linear regression showed strength of correlation varied by fungicide, with most showing weak correlations ($R^2 \leq 0.41$; $P > 0.05$). Ascospores of a single, self-fertile isolate were used to estimate the EC_{50} of six fungicides with different modes of action (dicarboximide, MBC, QoI, and DMI) using three methods: germination rate after 5h, germ tube length after 20h, and mycelial growth using the plate dilution method assessed after 48h. EC_{50} estimation with each method varied, but results were similar for fungicides with the same mode of action. Comparisons within each mode of action showed ascospore germination and germ tube growth were significantly more inhibited with QoI and dicarboximides, and ascospore germination least inhibited with DMI and MBC treatment. This is the first study to evaluate the effect of fungicides on ascospores of *S. sclerotiorum*, which is relevant since fungicide applications to prevent disease target primary infection by ascospores. A more complete analysis is underway and results of this study will be used for further work to characterize fungicide sensitivity of hundreds of isolates.

Contact Information – Dr. Sydney E. Everhart, Department of Plant Pathology, University of Nebraska-Lincoln, 406 Plant Science Hall, Lincoln, NE 68583-0722, 402-472-2879, everhart@unl.edu

Phenotypic evaluation and genetic dissection of resistance to Sclerotinia stem rot in soybean

Zixiang Wen, Ruijuan Tan, Shichen Zhang, Paul J. Collins, Shabir Wani, Cuihua Gu, Martin Chilvers, and Dechun Wang, Department of Plant, Soil and Microbial Sciences, Michigan State University

Funded Plan of Work: Enhancing soybean for resistance to Sclerotinia stem rot

ABSTRACT:
Sclerotinia stem rot (SSR), caused by *Sclerotinia sclerotiorium*, is an important soybean disease in northern soybean growing regions of United States. To perform resistance evaluation of SSR in soybean germplasm, 110 advanced breeding lines were evaluated for SSR in a naturally infected field at our research farm. Among those lines, 20 lines showed high level of resistance with disease severity index (DSI) less than 10. Two cultivars, E10151 and E12076-T, with partial SSR resistance were released in the past year. Sixty-four soybean plant introductions (PIs) that showed SSR resistance in both 2014 and 2015 field evaluations were re-evaluated. Sixty-one accessions of these PIs showed high level of resistance to white mold (DSI < 10) and confirmed as new sources of resistance to SSR. Moreover, two recombinant-inbred-line (RIL) populations were evaluated for SSR resistance again in the disease nursery. One QTL, located at chromosome (Chr.) 10, was repeatedly identified as being associated with SSR resistance in the population derived from E09088 × E12901 in both years. Two QTLs, located at Chr. 6 and 10, were confirmed in the population derived from E07048 × E06186 based on the past year's test. Molecular markers tightly linked to the resistance alleles on Chr. 6 and 10 will be useful for marker-assisted selection to develop new varieties with enhanced resistance to SSR.

Contact Information – Dr. Dechun Wang, 1066 Bogue St., Rm. A384E, East Lansing, MI 48824-1325; 1-517-355-0271 Ext. 1188; wangdech@msu.edu

Quantitative Genomics of Resistance and Virulence in the interaction of Botrytis cinerea with eudicots

Daniel J Kliebenstein, Wei Zhang, Nicole Soltis, Celine Caseys, Gongjun Shi, Raoni Gwinner, Suzi Atwell and Jason A. Corwin. Department of Plant Sciences, University of California, Davis, One Shields Ave, Davis, CA 95616

Molecular and/or Genomic studies of host/pathogen interaction studies rely on the use of large effect models with pathogens that cause epidemic disease outbreaks. This has developed a general molecular model where the interaction of proteins or metabolites from the host and pathogen trigger an "immune" response to create qualitative resistance. It is not presently known how this triggered immunity model applies to other systems such as broad host range endemic quantitative pathogens. To test how the innate immunity model applies to quantitative endemic pathogens, we are studying the molecular genomic basis of host/pathogen interactions using the necrotrophic fungal pathogen *Botrytis cinerea* and an array of eudicot hosts and more recently previously unrecognized monocot hosts.

Sequencing the genome of 96 diverse *Botrytis cinerea* isolates showed that high levels of genetic. Diversifying selection occurs at virulence loci such as toxin metabolite clusters and cell wall degrading genes and unknown loci that could be candidates for new virulence mechanisms. Extensive recombination in the genome shows that the species is not clonal and we can conduct GWA studies in the pathogen. Infecting all 96 isolates on *Arabidopsis thaliana* defense mutants with RNAseq showed that the pathogens genetic variation greatly alters the host's transcriptional responses. The salicylate and jasmonate signaling pathways functioned as amplifiers of the response. Genome wide association mapping in the pathogen showed that there were no major effect loci in the pathogen and instead quantitative virulence on the pathogens side was as polygenic as quantitative resistance in the host.

Extending this analysis to other dicots including Tomato, Lettuce, Chicory, Sunflower, Soybean and Brassica shows that *Botrytis cinerea* also displays a polygenic genomic architecture in virulence on these diverse hosts. Intriguingly there was minimal effect of domestication on the host/pathogen interaction with most plant hosts showing increased resistance in the domesticated germplasm and a similar range of variation. Using this, we were able to identify pathogen loci that control virulence across wild and domestic germplasm and these loci seem to focus on toxin production and detoxification with no identifiable MAMP/PAMP involved gene such as chitin or mannitol.

Flipping the genome to use genome wide association in *Arabidopsis thaliana* showed that plant resistance is highly polygenic and dependent on the genetics of the specific isolates. There was a slight but significant enrichment for R genes in this list, yet 95% of the causal genes were in other functional classes. All together, these results indicate that the ETI/PTI – MAMP/PAMP model may not be the best model for quantitative endemic pathogens to drive studies focused on increasing host resistance. These results will be presented.

Contact Information – Dr. Daniel J Kliebenstein, Univ. of California at Davis, Dept. of Plant Biology, Mail Stop 3, One Shields Ave, Davis, CA, 95616; kliebenstein@ucdavis.edu

Sources of white mold resistance derived from wide crosses in common bean and progress in characterization of relevant pathogen isolates

T.J.J. Miorini, T. Hornby, A. Pannullo, R. Higgins, S.E. Everhart, and J.R. Steadman
University of Nebraska-Lincoln

Collaborators: J. Kelly (MI), H. Rietman (Bel), M. Wunsch (ND), J. Myers (OR), P. Miklas (WA), M. Brick (CO), C. Urrea (NE), E. Berghauer (WI), and S. Singh (ID)

Funded Plan of Work: Improved white mold resistance in dry and snap beans through multi-site screening and pathogen characterization throughout major production areas

ABSTRACT:
One of our goals is to test putative sources of resistance in adapted backgrounds at multiple sites located in most of the major bean-production areas of the northern states. Multi-site testing assures annual bean evaluation data when some sites have no data due to weather. A straw test that consistently identifies sources of resistance in adapted and unadapted bean germplasm is used for greenhouse tests. In 2016 field testing four locations plus Belgium provided evidence of white mold resistance or escape in pinto, red, black, navy and snap bean seed types. The five 2016 greenhouse tests were surprising because only three out of 25 lines yielded data supporting resistance. One explanation for so few resistant lines is that many of the submitted lines used field performance data to select them; most lines only had escape mechanisms. To characterize *S. sclerotiorum* obtained from production regions, we previously phenotyped and genotyped 366 *S. sclerotiorum* isolates mostly from dry bean in the USA. For out-group comparison and due to low disease incidence across locations, we performed similar characterizations for 69 isolates from soybean collected in the USA (15), Brazil (49), and Argentina (5). Mycelial compatibility group (MCG) was determined by pairing isolates on DS medium and evaluating after 10 days. A total of 23 MCGs were identified, with 39% represented by a single isolate. Some MCGs were found in more than one region, for example one MCG was identified in the USA (one isolate; Bellwood, NE) and Brazil (two isolates; Chapadão do Sul, MS). Isolates were genotyped using 8 microsatellite primers and identified 51 multilocus genotypes. To assess aggressiveness, leaves of partially resistant soybean cultivar 'Dassel' were each inoculated with mycelium on an agar plug. The three youngest and fully expanded leaves were collected at 21, 28, and 35 days after emergence and lesion evaluations made after 48 hours. Preliminary analysis showed leaf age influenced aggressiveness assays, with aggressiveness higher in younger plants (21 days) and lower in older plants (35 days). Aggressiveness differences and genetic variation using molecular markers have identified *S. sclerotiorum* isolate variation that could impact resistance evaluation studies. The overall research approach we are using is also applicable to facilitate identification of white mold resistance in other susceptible crops such as canola, pulses, soybean, and sunflower.

Contact Information – Dr. James R. Steadman and Dr. Sydney Everhart, Department of Plant Pathology, University of Nebraska-Lincoln, 406 Plant Science Hall, Lincoln, NE 68583-0722, 402-472-3163, jsteadman1@unl.edu.

Understanding and Improving Basal Resistance to *Sclerotinia sclerotiorum*

Jeffrey A. Rollins & Zhonglin Mou, University of Florida, Gainesville, FL

Funded Plan of Work: Enhancing basal resistance to *Sclerotinia sclerotiorum* in Brassica

ABSTRACT:
The goal of the project is to use the newly identified *Arabidopsis thaliana* gene hypersusceptible to *S. sclerotiorum* (*HSS1*) for engineering high levels of disease resistance in canola. Through map-based cloning, we have determined that *HSS1* encodes the Mediator complex subunit MED16, indicating that MED16 is a key component regulating basal resistance to *S. sclerotiorum*. We found that MED16 is highly conserved in *Brassica napus*, since the *BnHSS1* homolog complemented the *A. thaliana hss1* mutant. Unfortunately, overexpression of either the *Arabidopsis HSS1* gene or the *BnHSS1* homolog did not provide any resistance to *S. sclerotiorum*. To better understand these results, we analyzed the fate of the HSS1/MED16 protein during infection and discovered that *S. sclerotiorum* infection induces complete degradation of HSS1/MED16. We are screening other novel forms of HSS1/MED16 from fungi and non-host plants for their ability to confer resistance to *S. sclerotiorum*. We found that the rice *HSS1* homolog complemented the *A. thaliana hss1* mutant and the *S. sclerotiorum HSS1* homolog did not. We are testing if the rice HSS1/MED16 protein is more resistant to *S. sclerotiorum*-mediated degradation. The Mediator complex is emerging as a master regulator of plant immunity against pathogens, especially necrotrophic fungal pathogens, which underlines our discovery of the critical role of *HSS1* in basal resistance against *S. sclerotiorum*. We are now focusing our efforts on identifying and developing stable forms of HSS1/MED16 for overexpression and evaluation of resistance.

Contact Information – Dr. Zhonglin Mou, Department of Microbiology and Cell Science, University of Florida, Gainesville, FL 32611—0700; 352-392-0285; zhlmou@ufl.edu

Using genomic selection to optimize prediction of *Sclerotinia* and agronomic phenotypes for more efficient breeding

Brent S. Hulke[1], Qing-Ming Gao[1] and Nolan C. Kane[2]
[1] USDA-ARS, Northern Crop Science Laboratory, Fargo, ND
[2] University of Colorado, Ecology and Evolutionary Biology Dept., Boulder, CO
Funded Plan of Work: Using GS to optimize prediction of Sclerotinia and agronomic phenotypes for more efficient breeding.

ABSTRACT:
Sunflower breeding has made huge gains in disease resistance and quality traits that are simply inherited, but lacks efficiency to adequately deal with Sclerotinia resistance, because of the complex genetic architecture. Many genes of small effect must work in concert to facilitate partial resistance. Lines exist with high levels of resistance in the field, as demonstrated by recent germplasm evaluations. They do not, however, bring the most favorable yield and agronomic characteristics to sunflower hybrids. The primary goal of this work is to better balance the intensity and efficiency of selection for Sclerotinia resistance and other agronomic traits, to make more breeding progress per generation on all traits proportional to their actual value to the producer. Genomic Selection (GS) is a new statistical technology we would like to investigate for this purpose. In the past year, we have conducted 5x to 10x whole genome shotgun resequencing of all of the remaining parental stocks for each of our breeding populations since 2008. The 2015 breeding lines from the USDA sunflower breeding program that have both yield and Sclerotinia resistance data from specialized nurseries were also analyzed with GBS and added to the dataset. We have called SNPs in each of these lines and are currently completing imputation and deposit into our relational database system that will allow us easy access to nursery, field phenotyping trial, and genomic datasets to conduct model fitting. In the next year, we will begin analyzing the phenotypic data for each line with respect to its genotype to determine whether random effect predictors in the form of molecular markers could improve accuracy of selection for Sclerotinia and yield in early generations of inbred lines. This could potentially replace unreplicated testing in early generations or, in other words, allow for preliminary line performance prediction in the absence of field data.

Contact Information – Dr. Brent S. Hulke, Sunflower and Plant Biology Research Unit, USDA-ARS Northern Crop Science Laboratory, 1605 Albrecht Blvd. N., Fargo, ND 58102-2765; (701) 239-1321, Brent.Hulke@ars.usda.gov

Variation among a large and diverse collection of *S. sclerotiorum* isolates for virulence on sunflower inbred lines

Kassaye Belay[1], Christopher Misar[2], Mitchell DuFour[1], Shyam Solanki[1], Michael Wunsch[3], Hannah Barrett[4], Ron Nelson[4], Keith Olander[4], Berlin Nelson[1], Robert Brueggeman[1], and William Underwood[2]

[1]Department of Plant Pathology, North Dakota State University, Fargo, ND
[2]USDA-ARS, Northern Crop Science Laboratory, Fargo, ND
[3]Carrington Research Extension Center, North Dakota State University, Carrington, ND
[4]Central Lakes College, Ag and Energy Center, Staples, MN

Research Project: Identification of *Sclerotinia sclerotiorum* virulence determinants relevant to infection of multiple host plants by association mapping.

ABSTRACT:
Sclerotinia sclerotiorum is one of the most destructive pathogens of sunflower in the United States, causing several distinctive diseases including basal stalk rot initiated by root infection and head rot resulting from infection of the sunflower head. The pathogen has a remarkably broad host range, yet little is currently known about the virulence strategies that allow *S. sclerotiorum* to successfully infect a wide range of plant hosts. The goals of our project are to build on a previous effort toward genotyping a large collection of *S. sclerotiorum* field isolates collected from diverse hosts, to phenotype these isolates for virulence on multiple sunflower genotypes, and to identify and validate candidate genes contributing to the virulence of this fungal pathogen. To this end, we have supplemented a previous collection of 140 *S. sclerotiorum* field isolates with an additional 62 isolates from our laboratory and 50 isolates generously provided by Dr. Jim Steadman (University of Nebraska) to assemble a collection of 252 diverse isolates. We are currently phenotyping this isolate collection for virulence (aggressiveness) on stems of USDA sunflower inbred line HA 207. We will conduct genotyping-by-sequencing to genotype isolates for which no genotypic data are available and improve marker density for previously genotyped isolates, and we will use these data for association mapping to identify candidate virulence factors. We are also currently evaluating six candidate *S. sclerotiorum* genes potentially associated with virulence on bean by direct gene replacement in the pathogen as well as host-induced gene silencing in the plant model *Arabidopsis thaliana*. In a parallel effort, we have determined the virulence of multiple *S. sclerotiorum* isolates on a panel of USDA sunflower inbred lines. Our preliminary results indicate that at least some isolates exhibit differential interactions with specific sunflower genotypes.

Contact Information – Dr. William Underwood, Sunflower and Plant Biology Research Unit, USDA-ARS Northern Crop Science Laboratory, 1605 Albrecht Blvd., Fargo, ND USA 58102-2765; (701) 239-1316, william.underwood@ars.usda.gov

2017 Sclerotinia Initiative Meeting Participants

Last Name	First Name	Company	Address	City	State	Zip	Phone	Email
Arkwazee	Haidar	Oregon State University	4017 Ag. & Life Sci. Bldg.	Corvallis	OR	97331	541-908-0882	haidarh@oregonstate.edu
AshtariMahini	Rahil	North Dakota State University	166 Loftsgard Hall	Fargo	ND	58108		rahil.ashtarimahini@ndsu.edu
Belay	Kassaye	North Dakota State University	1 Niskanen Expansion	Fargo	ND	58102-5910	701-793-4470	kassaye.belay@ndsu.edu
Berghauer	Erika	Monsanto Vegetabl Seeds/Seminis	7202 Portage Road	DeForest	WI	53532	608-842-1435	emberg@monsanto.com
Bruce	Myron	Montana State Univeristy	130 Plant Biosciences Bldg	Bozeman	MT	59717-3150	406-994-5572	myron.bruce@montana.edu
Chen	Weidong	USDA-ARS	303 Johnson Hall, WSU	Pullman	WA	99164	509-335-9178	w-chen@wsu.edu
Chilvers	Martin	Michigan State University	578 Wilson Rd., CIPS104	East Lansing	MI	48823	517-353-9967	chilvers@msu.edu
Chittem	Kishore	North Dakota State University	306 Walster Hall	Fargo	ND	58102	701-429-8381	kishore.chittem@ndsu.edu
Coetzee	Lisa Ann	University of the Free State	PO Box 339	Bloemfontein	Free State	9300	27514019681	coetzeeLA@ufs.ac.za
Coleman	Barry	Northern Canola Growers Association	125 Slate Drive, Ste. #4	Bismarck	ND	58503	701-223-4124	coleman@ndpci.com
del Rio	Luis	North Dakota State University	NDSU Dept. 7660	Fargo	ND	58108	701-231-7073	luis.delrio-mendoza@ndsu.edu
Everhart	Sydney	University of Nebraska	406 Plant Science Hall	Lincoln	NE	68583	402-472-2879	everhart@unl.edu
Foley	Michael	USDA-ARS	1605 Albrecht Boulevard N	Fargo	ND	58102-2765	701-239-1322	michael.foley@ars.usda.gov
Grusak	Mike	USDA-ARS	1605 Albrecht Boulevard N	Fargo	ND	58102	701-239-1371	mike.grusak@ars.usda.gov
Hulke	Brent	USDA-ARS	1605 Albrecht Boulevard N	Fargo	ND	58102-2765	701-239-1321	brent.hulke@ars.usda.gov
Kaphammer	Bryan	USDA-ARS	2150 Centre Ave., Bldg D, Suite 300	Fort Collins	CO	80526	970-492-7054	bryan.kaphammer@ars.usda.gov
Kliebenstein	Daniel J.	University of California, Davis	One Shields Avenue	Davis	CA	95616	530-754-7775	kliebenstein@ucdavis.edu
McClean	Phillip	North Dakota State University	Loftsgard 270B	Fargo	ND	58102	701-371-3250	phillip.mcclean@ndsu.edu
McLaren	Neal	University of the Free State	PO Box 339	Bloemfontein	Free State	9300	27-51-401-366	mclarenN@ufs.ac.za
McPhee	Kevin	North Dakota State University	PO Box 6050, Dept. 7670	Fargo	ND	58108	701-231-8156	kevin.mcphee@ndsu.edu
Miklas	Phillip	USDA-ARS	24106 N Bunn Road	Prosser	WA	99350	509-786-8492	phil.miklas@ars.usda.gov
Miorini	Thomas	University of Nebraska-Lincoln	406 Plant Sciences Hall	Lincoln	NE	68583	402-314-9470	tmiorini3@unl.edu
Misar	Chris	USDA-ARS	819 10th Ave. N. Apt # Basement	Fargo	ND	58102	605-661-2530	christopher.misar@ars.usda.gov
Mochama	Pauline	South Dakota State University	1047 Main Avenue, Apt. 7B	Brookings	SD	57006	612-801-8742	pauline.mochama@sdstate.edu
Mou	Zhonglin	University of Florida	Microbiology and Cell Science	Gainsville	FL	32611	352-392-0285	zhlmou@ufl.edu
Myers	James	Oregon State University	Department of Horticulture, ALS 4017	Corvallis	OR	97331	541-737-3083	james.myers@oregonstate.edu
Ortiz	Vivana	Michigan State University	578 Wilson Rd, CIPS 104	East Lansing	MI	48824	517-3538913	ortizviv@msu.edu
Plouy	Alexis	Seminis	21220 Hwy 30	Twin Falls	ID	83301	208-329-0335	alexis.plouy@monsanto.com
Qi	Lili	USDA-ARS	1605 Albrecht Boulevard N	Fargo	ND	58102-2765	701-239-1351	lili.qi@ars.usda.gov
Rahman	Mukhlesur	North Dakota State University	1350 Albrecht Boulevard	Fargo	ND	58108-6050	701-231-5768	md.m.rahman@ndsu.edu
Rashid	Khalid	Agricultur & Agri-Food Canada	Morden Research & Development Ctr	Mordn	Manitoba, Canad	R6M 1Y5	204-822-7520	khalid.rashid@agr.gc.ca
Sandbakken	John	National Sunflower Association	2401 46th Avenue SE	Mandan	ND	58554	701-328-5100	johns@sunflowernsa.com
Scholz	Todd	American Pulse Assoc./USA Dry Pea & Lentil	2780 W Pullman Road	Moscow	ID	83843	208-882-3023	tscholz@usapulses.org
Scott	Roy	USDA-ARS	5601 Sunnyside Avenue	Beltsville	MD	20705	301-504-4670	roy.scott@ars.usda.gov
Seiler	Gerald	USDA-ARS	1605 Albrecht Boulevard N	Fargo	ND	58102-2765	701-239-1380	gerald.seiler@ars.usda.gov
Shahoveisi	Fereshteh	North Dakota State University		Fargo	ND	58102	224-856-0353	fereshteh.shahoveisi@ndsu.edu
Steadman	James R.	University of Nebraska	406 PSH - UN-L East Campus	Lincoln	NE	68583-0722	402-472-3163	jsteadman1@unl.edu
Swanson	Kim	USDA-ARS	1605 Albrecht Boulevard N	Fargo	ND	58102-2765	701-239-1370	kimberly.swanson@ars.usda.gov
Talukder	Zahirul	North Dakota State University	1605 Albrecht Boulevard N	Fargo	ND	58102-2765	701-239-1324	zahirul.talukder@ars.usda.gov
Underwood	William	USDA-ARS	1307 18th Street N	Fargo	ND	58102	701-239-1316	william.underwood@ars.usda.gov
Varner	Greg	Michigan Dry Bean Research Bd.	8439 North Blair Road	Breckenridge	MI	48615-9725	989-751-8415	varnerbean@hotmail.com
Wang	Dechun	Michigan State University	1066 Bogue St.,Rm. A384E	East Lansing	MI	48824-1325	517-353-0219	wangdech@msu.edu
Wen	Zixiang	Michigan State University	1066 Bogue St., Rm A366	East Lansing	MI	48824-1325	517-353-0167	wzxsoy@msu.edu
Whiting	Kelly	United Soybean Board	16305 Swingley Ridge Road	Chesterfield	MO	63017	314-579-1598	kwhiting@smithbucklin.com
Wilson	Richard	Oilseeds & Biosciences Consulting	5517 Hickory Leaf Drive	Raleigh	NC	27606-9502	919-906-6937	rfwilson@mindspring.com
Wunsch	Michael	NDSU - Carrington Research Ext. Ctr	PO Box 219	Carrington	ND	58421-0219	701-652-2951	michael.wunsch@ndsu.edu