Proceedings Book

Washington State University
June 28 - July 2

Ascochyta 2009
Pullman, Washington
June 28 - July 2, 2009

Proceedings

ASCOCHYTA 2009
The Second International Ascochyta Workshop
Pullman, Washington, USA
June 28 – July 2, 2009

Washington State University
Pullman, Washington

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ASCOCHYTA
2009

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Program Schedule

Sunday, June 28

6:00-9:00 **Opening Reception at Old Post Office, Downtown, Pullman**

Monday, June 29

8:00 **Welcome - *Moderator: Fred Muhelbauer***

Michael Kahn, Associate Director, ARC, CAHNRS, WSU
Tim McGreevy, Executive Director, USA Dry Pea and Lentil Council
Gail Wisler, National Program Leader, USDA-ARS
Bernard Tivoli, INRA,Co-organizer of Ascochyta 2006
Weidong Chen, USDA-ARS, Local Organizing Committee

Session I **Pathogen Biology - *Moderator: Alain Baranger***

8:30 *Didymella, Mycosphaerella, Ascochyta,* and *Phoma*: what a tangled web has been woven
J. Rogers

9:15 *Severity and distribution of Phoma koolunga on ascochyta blight-affected field peas in south eastern Australia*
J.A. Davidson, A. McKay, M. Krysinska-Kaczmarek, and E.S. Scott

9:35 *Identification of Ascochyta and Phoma species on clover: Comparison with other species from Fabaceae*
N. Ghiat, N. Boumedienne, and Z. Bouznad

9:55 *Temperature adaptation and ecological divergence of the fungal pathogen Didymella rabiei on sympatric wild and domesticated chickpea*
O. Frenkel, T.L. Peever, M.I. Chilvers, H. Ozkilinc, C. Can, D. Shtienberg, A. Sherman, S. Abbo

10:15 *Did the development of Ascochyta blight on winter and spring pea (Pisum sativum) in France depend on the same populations of Mycosphaerella pinodes?*
C. Le May, M. Guibert , A. Leclerc, and B. Tivoli

10:35 **Break**

Session II **Host Resistance I (Breeding) - *Moderator: George Vandemark***

10:50 *Breeding for ascochyta resistance in desi chickpea*
P. Gaur, S. Pande, T. Khan, S. Tripathi, M. Sharma, H. Clarke, JS Sandhu, L. Kaur, D. Basandrai, A. Basandrai, CLL Gowda1 and KHM Siddique

11:20 *Genetic enhancement of chickpea for Ascochyta blight resistance*
R. S. Malhotra, M. Imtiaz, S. Ahmed, and S. Kabbabeh

11:50 **Group Photo**

12:00 **Lunch**

Session II	**Host Resistance I (Breeding) cont.**
1:00	*Use of germplasm for Ascochyta blight resistance in pea and lentil* K.E. McPhee and A. Sarker
1:30	*Breeding for improved ascochyta blight resistance in pea* T.D. Warkentin, S. Banniza, B. Tar'an, A. Vandenberg, and K. Bett
1:50	*Control of partial resistance to Mycosphaerella pinodes in pea* A. Baranger, H. Miteul, G. Deniot, R.Lecointe, I.Lejeune-Hénaut, A.Lesné, F.Mohamadi, G.Morin, C.Onfroy, M.L.Pilet-Nayel, B.Tivoli
2:10	*Exploration of resistance to Mycosphaerella blight in wild Pisum spp. to develop resistant field pea germplasm* G. Valarmathi, S. Banniza, B. Tar'an, and T.D. Warkentin
2:30	*Enhancement of black spot resistance in field pea* K. Adhikari, T. Khan, I. Pritchard and T. Leonforte
2:50	*Mapping of Ascochyta blight resistance in chickpea* L. Buchwaldt,G.K. Kishore, A.G. Sharpe, C. Sidebottom, H. M. Booker, B. Tar'an
3:10	**Break**

Session III	**Bob Henson Award - Student Competition (Oral Component)** ***Moderator: Bernard Tivoli*** ***Evaluation Committee: Jenny Davidson (chair), Lone Buchwaldt, and Pooran Gaur.***
3:30	*A comparison of phenotypic and marker-assisted selection for Ascochyta blight resistance in chickpea* P. Castro, M.D. Fernandez, T. Millan, J. Gil and J. Rubio
3:38	*Partial cloning of two polyketide synthase genes associated with pathogenicity of Ascochyta rabiei* J. A. Delgado, S. W. Meinhardt, S. G. Markell, and R. S. Goswami
3:46	*Cloning and characterization of anonymous regions of Ascochyta lentis and A. fabae genomes and suitability of these regions for phylogenetic analysis of Ascochyta species* J.E. Stewart, R.N. Attanayake, E.N. Njambere, T. Drader, and T.L. Peever
4:00	**Poster session &Evening Reception at Emsinger Pavilion–** ***Moderator: Weidong Chen*** Poster session Bob Henson Award – Student Competition (Poster Component)
6:00	**Dinner on your own**

Tuesday, June 30

Session IV	**Disease Management - *Moderator: Fred Muehlbauer***
8:00	*Over forty years on six continents researching Ascochyta diseases of food legumes* W.J. Kaiser
9:00	*Ascochyta blight management in Australian pulse crops* J.A. Davidson
9:20	*Management of Ascochyta blight of chickpea in India* A.K.Basandrai, L.Kaur, D.Basandrai, S.Pandey, R.S.Malhotra, P.M.Gaur and A. Sarker
9:40	*Management of Ascochyta blight of chickpea in northern NSW* K.J. Moore, K.D. Lindbeck, P. Nash, G. Chiplin and E. J. Knights
10:00	**Break**
10:20	*Management of Ascochyta diseases in North America* R. Morrall (Presented by T. Warkentin)
10:35	*Fungicide trials for managing chickpea Ascochyta blight* W. Chen
10:45	*On-Farm Tests With Growers for Fungicide Comparisons* L. Smith
11:10	*Panel discussion* Jenny Davidson, Australia Mike DeVoe, USA Ashutosh Sarker, India Larry Smith, USA
11:45-6:00	**Field Tour – Moderator: Todd Scholz** WSU Spillman Agronomy Farm USDA-ARS Grain Legume Genetics and Physiology Research Unit Ascochyta blight nursery Fungicide trials Chickpea, lentil and pea breeding program Clark Farms (Chickpeas) Mader Farms (Lentils and Peas)
5:00	Kamiak Butte Donor Appreciations Mexican BBQ Dinner
9:00	**Return to Pullman**

Wednesday, July 1

Session V	**Molecular Biology – *Moderator: Tobin Peever***
8:00	Functional genomics of Dothideomycetes; applications to legume pathogens R. Oliver
9:00	*PR proteins in lentil: isolation and expression in response to Ascochyta lentis and signalling compounds* B.M. Mustafa, D.T.H. Tan, P.W.J. Taylor, and R. Ford
9:20	*Applications of suppression subtractive hybridization (SSH) in identifying differentially expressed transcripts in Ascochyta rabiei* D. White, G. Vandemark, and W. Chen
9:40	*Induced Mutations for Ascochyta blight Resistance in Chickpea* T.M. Shah, J.I. Mirza, B. Manzoor Atta, H. Ali, S.Sarwar Alam and M. Ahsanul Haq
10:00	*SCARS markers linked to Asochyta rabiei in chickpea (SCAE19336, SCM02935 and SCY17590): expression studies and homologies with EST and related sequences* M. Iruela, F. Piston, F. Barro, J. Gil, T. Millan
10:20	*Understanding Ascochyta blight resistance in chickpea using molecular genetics and genomic approaches* P.N. Rajesh, M.O'Bleness, B. Till, D. Cook, S. Henikoff, B. Roe, W. Chen, F.J.Muehlbauer
10:40	**Break**

Session VI	**Host Resistance II (Genetics) – *Moderator: Tom Warkentin***
11:00	*Pathogenesis-related genes and genetic variation in potential resistance genes of major European legumes: The LegResist project* G. Kahl, P. Winter, R. Horres, B. Rotter, R. Jüngling
11:30	*Pyramiding resistance in chickpea toAscochyta rabiei* P.W.J. Taylor, X.Y. Bian, and R. Ford
12:00	**Lunch**
1:00	*Pathotype specific seedling and adult-plant resistance sources to Ascochyta rabiei in chickpea* A.K. Basandrai, D. Basandrai, S. Pande, P.M. Gaur, S.K. Thakur, H.L. Thakur and M. Sharma
1:20	*Phenotypic and molecular characterization of chickpeas for sources of resistance to Ascochyta blight* M. Imtiaz, R.S. Malhotra, S. Ahmed, A. Khalifeh, M. van Ginkel and S. Kabbabeh
1:40	*Breeding for resistance to ascochyta blight in chickpea of India: Current status* J.S. Sandhu, S.K. Gupta, Livinder Kaur, M.M. Verma and Gurdip Singh

Session VI	**Host Resistance II (Genetics) cont.**
2:00	*Biochemical and molecular reach for disease resistance to chickpea blight caused by Ascochyta rabiei* S. S. Alam, T. M. Shah, B. M. Atta and H. Ali
2:20	*Genetics of resistance to ascochyta blight in chickpea* R. Bhardwaj, J.S. Sandhu, Livinder Kaur, S. K. Gupta and P.M. Gaur
2:40	**Break**

Session VII	**Epidemiology- *Moderator: Paul Taylor***
3:00	*Highlights of 15 years of research on Ascochyta blight on pea in France: Epidemiology and impact of the disease on yield and yield components* B. Tivoli
3:30	*Spatial distribution of Didymella pinodes Petrak and Ascochyta pinodella L.K. Jones on Austrian winter pea plants* M.I. Chilvers, D.H. Erickson, H.O. Akamatsu, and T.L. Peever
3:50	*Response of field pea varieties to the fungal components of the Ascochyta complex* H.J. Richardson, T. Leonforte and A. J. Smith
4:10	*Optimizing Ascochyta blight management in chickpea on the Canadian prairies* C. Armstrong-Cho, T. Wolf, Y. Gan, B. Tar'an, and S. Banniza
4:30	*Effect of growth stages of chickpea on the genetic resistance of Ascochyta blight* M. Sharma, S. Pande, P.M. Gaur, and C.L.L. Gowda
4:50	*Development of screening techniques and identification of new sources of resistance to Ascochyta blight disease of chickpea* S. Pande, M. Sharma, L. Kaur, A.K. Basandrai, P.M. Gaur, T. Khan, K.H.M. Siddique and C.L.L. Gowda
7:00-9:00	**Conference Banquet** Presentation of Bob Henson Awards- *Kevin McPhee and Rubella Goswami*

Thursday, July 2

8:00 Breakout session

The breakout session will be designed to identify areas of Ascochyta research that are in need of attention and provide a forum for interested parties to discuss these areas of research need and to formulate plans for going forward. These plans would include important items such as: design research plans including objectives; determine scientists (Workshop attendees and those not present) with similar research interests to foster future cooperation; and identify potential sources of funds both nationally and internationally. The areas of research to be discussed at the breakout sessions would be determined during the workshop. Examples of topics of discussion could be: (1) Pathogen biology including the infection process and the development of progressively more virulent pathotypes; (2) Plant genomics focusing on resistance genes in pea, lentil and chickpea; and (3) Exploration, collection and evaluation of germplasm for resistance to ascochyta. The plan for the breakout sessions is to identify two or three areas such as these during the first days of the workshop and then arrive at a 2-3 page concept note on how the research would be conducted, scientists with such research interests, and where the needed funds could be found.

9:30 Break

9:50 Discussion of Breakout session

This discussion session will be devoted to the presentations of the plans developed in the Breakout Session to all workshop participants. We hope to conclude this part of the Workshop program with a set of research plans that when implemented will, foster improved teamwork on Ascochyta blights and possibly other grain legume diseases, and enhance our knowledge of the important aspects of the pathogens and interactions with the host species.

11:00 Concluding session

12:00 Boxed Lunch

1:00 Lab and greenhouse tours (optional)
Wild perennial chickpea tour (optional)

Poster session

P01 - Clinostats rosea is a common inhabitant of chickpea debris in the Palouse region of the Pacific Northwest, USA
F.M. Dugan, S.L. Lupien, and W. Chen

P02 - Isolate variability and resistance to Ascochyta fabae in southern Australia
R.B.E. Kimber, S.A. Palmer, J.A. Davidson, K.J. Williams and J.G. Paull

P03 - The role of sexual reproduction of Didymella rabiei in increasing virulence on chickpea cultivars in Syria
M. M.Seid Ahmed, S. Abang, M. Kabbabeh, I. Samer, Mohammed and R. Malhotra

P04 - Identification of genes involved in resistance to Mycospherella pinodes in pea using microarray technology
S. Fondevilla, F. Krajinski, H. Küster and D. Rubiales

P05 - Genetic Resistance to Phoma medicaginis in Pea
K.E. McPhee and X. Wang

P06 - Identifying pathogenicity determinants of Ascochyta rabiei via genetic complementation
D. White, and W. Chen

P07 - Role of grain legumes as alternative hosts on the fitness of Mycosphaerella pinodes and Phoma medicaginis var. pinodella
C. Le May

P08 - Plant canopy modifications and Ascochyta blight control in chickpea
Y.T. Gan, T.D. Warkentin, R. Chandirasekaran, B.D. Gossen, T. Wolf, and S. Banniza

P09 - Sources of resistance in wild species of lentil to isolates of ascochyta blight (Ascochyta lentis)
A. Tullu, J. Fiala, S. Banniza, S. Boechler, K. Bett, B. Taran, T.Warkentin, A. Vandenberg

P10 - Breeding faba bean for resistance to Ascochyta blight
F. Maalouf, S. Ahmed, M. Kabakebji, S. Kabbabeh, K. Street and R. Malhotra

P11 - Development of the teleomorph of Ascochyta rabiei on culture media
A. Trapero-Casas and W.J. Kaiser

P12 - Preliminary investigation of the secretome of Aschochyta rabiei
S. Meinhardt, N. Mittal, and C. Tandeski

P13 - Assessment of stability in reaction to Mycosphaerella pinodes among field pea genotypes
L. Boros

P14 - Molecular detection of Ascochyta rabiei pahtotypes in infected chickpea seeds
S. Murad, N. Hassan, A. Hamwieh, M. Baum, S. Ahmed

P15 - Allelic diversity of USDA chickpea core collection at quantitative trait loci for resistance to ascochyta blight
C.J. Coyne, L. Taylor and R.K. Varshney

P16 - Severity and Distribution Of Phoma Koolunga On Ascochyta Blight-Affected Field Peas In South Eastern Australia.
J.A. Davidson, A. McKay, M. Krysinska-Kaczmarek

P17 - Identification of Ascochyta And Phoma Species On Clover: Comparison With Other Species From Fabaceae
N. Ghiat, N. Boumedienne, and Z. Bouznad

P18 - Temperature Adaptation and Ecological Divergence Of The Fungal Pathogen Didymella Rabiei On Sympatric Wild And Domesticated Chickpea.
O. Frenkel, T.L. Peever, M.I. Chilvers, H. Ozkilinc, C. Can, D. Shtienberg,A. Sherman, S. Abbo

P19 - Did The Development of Ascochyta Blight On Winter And Spring Pea (Pisum Sativum) In France Depend On The Same Populations Of Mycosphaerella Pinodes?
C. Le May, M. Guibert, A. Leclerc, and B. Tivoli

P20 - Breeding For Ascochyta Resistance In Desi Chickpea.
P. Gaur, S. Pande,T. Khan, S. Tripathi, M. Sharma, H. Clarke, JS Sandhu, L. Kaur, D. Basandrai, A. Basandrai, R. Varshney, CLL Gowda, and KHM Siddique

P21 - Genetic Enhancement of Chickpea For Ascochyta Blight Resistance
R. S. Malhotra, M. Imtiaz, S. Ahmed, and S. Kabbabeh

P22 - Control Of Partial Resistance to Mycosphaerella Pinodes In Pea
A. Baranger, H. Miteul, G. Deniot, R. Lecointe, I. Lejeune-Hénaut, A. Lesné, F. Mohamadi, G. Morin, C. Onfroy, ML. Pilet-Nayel, B. Tivoli

P23 - Enhancement of Black Spot Resistance In Field Pea
K. Adhikari, T. Khan, I. Pritchard and T. Leonforte

P24 - A Comparison of Phenotypic And Marker-Assisted Selection For Ascochyta Blight Resistance In Chickpea.
P. Castro, M.D. Fernandez, T. Millan, J. Gil and J. Rubio
Student presenter

P25 - Partial Cloning of Two Polyketide Synthase Genes Associated With Pathogenicity of Ascochyta Rabiei.
J. A. Delgado, S. W. Meinhardt, S. G. Markell, and R. S. Goswami
Student presenter.

P26 - Comparative Population Study of Didymella Rabiei In Turkey And Israel
H. Ozkilinc, O. Frenkel, C. Can, S. Abbo, D. Shtienberg, A. Sherman
Student Presenter

P27 - Cloning and Characterization Of Anonymous Regions of Ascochyta Lentis And A. Fabae Genomes and Suitability Of These Regions For Phylogenetic Analysis of Ascochyta Species.
J.E. Stewart, R.N. Attanayake, E.N. Njambere, T. Drader, and T.L. Peever
Student Presenter

P28 - A System-Based Risk Estimator of Ascochyta Blight Disease In South Australia
J.A. Davidson, M.U. Salam, J. Galloway E. S. Scott

P29 - Management of Ascochyta Blight Of Chickpea In India.
A.K.Basandrai, L.Kaur, D.Basandrai, S.Pande, R.S.Malhotra, P.M.Gaur and A.Sarker

P30 - Management of Ascochyta Blight of Chickpea In Northern Nsw.
K.J. Moore, K.D. Lindbeck, P. Nash, G. Chiplinand E. J. Knights

P31 - Applications Of Suppression Subtractive Hybridization (Ssh) In Identifying Differentially Expressed Transcripts In AscochytaRabiei.
D. White, G. Vandemark, and W. Chen

P32 - Induced Mutations for Ascochyta Blight Resistance In Chickpea (Cicer Arietinum L.)
T. Mahmud Shah, J. Iqbal Mirza, B.r Manzoor Atta, H. Ali, S. Sarwar Alam and M. Ahsanul Haq

P33 - Scars Markers Linked To Asochyta Rabiei In Chickpea (Scae19$_{336}$, Scm02$_{935}$ And Scy17$_{590)}$: Expression Studies And Homologies With Est And Related Sequences.
M. Iruela, F. Piston, F. Barro, J. Gil, T. Millan.

P34 - Understanding Ascochyta Blight Resistance in Chickpea Using Molecular Genetics And Genomic Approaches
PN Rajesh, M. O'Bleness, B. Till, D. Cook, S. Henikoff, B. Roe, W. Chen, F. Muehlbauer

P35 - Pathotype Specific Seedling And Adult-Plant Resistance Sources To Ascochyta Rabiei In Chickpea (Cicer Arietinuml.) .
A.K. Basandrai, D. Basandrai, S. Pande, PM Gaur, S.K. Thakur, H.L. Thakur and M. Sharma

P36 - Pathogenesis-Related Genes and Genetic Variation In Potential Resistance Genes of Major European Legumes: The Legresist Project.
G.Kahl, P.Winter, R. Horres, B. Rotter, R. Jüngling and the LEGRESIST Consortium.

P37 - Phenotypic and Molecular Characterization Of Chickpeas For Sources Of Resistance To Ascochyta Blight
M. Imtiaz, R.S. Malhotra, S. Ahmed, A. Khalifeh, M. van Ginkel and S. Kabbabeh

P38 - Breeding For Resistance to Ascochyta Blight In Chickpea Of India: Current Status.
J.S. Sandhu, S.K. Gupta, L. Kaur, M.M. Verma and G. Singh

P39 - Biochemical and Molecular Reach For Disease Resistance To Chickpea Blight Caused By Ascochyta Rabiei (Pass.) Labr.
S. S. Alam, T. M. Shah, B. M. Atta and H. Ali

P40 - Genetics of Resistance to Ascochyta Blight in Chickpea.
R. Bhardwaj, J.S. Sandhu,Livinder Kaur, S. K. Gupta and P.M. Gaur

P41 - Highlightsof 15 Years Of Research on Ascochyta Blight on Pea In France: Epidemiology And Impact of the Disease on Yield and Yield Components.
B. Tivoli

P42 - Spatial Distribution Of Didymella Pinodes Petrak And Ascochyta Pinodella L.K. Jones On Austrian Winter Pea Plants.
M. I. Chilvers

P43 - Response of Field Pea Varieties to the Fungal Components of The Ascochyta Complex.
H.J. Richardson, T. Leonforte and A. J. Smith

P44 - Optimizing Ascochyta Blight Management in Chickpea on The Canadian Prairies.
C. Armstrong-Cho, T. Wolf, Y. Gan, B. Tar'an, and S. Banniza

P45- Effect of Growth Stages of Chickpea on the Genetic Resistance of Ascochyta Blight
M. Sharma, S. Pande, P.M. Gaur, and C.L.L. Gowda

P46 - Development of Screening Techniques and Identification of New Sources of Resistance to Ascochyta Blight Disease of Chickpea.
S. Pande, M. Sharma, L. Kaur, A.K. Basandrai, P.M. Gaur, T. Khan, K.H.M. Siddique and C.L.L. Gowda

Session I
Pathogen Biology

O01

DIDYMELLA,MYCOSPHAERELLA, ASCOCHYTA, AND PHOMA: WHAT A TANGLED WEB HAS BEEN WOVEN

J. Rogers, *Department of Plant Pathology, Washington State University*

Didymella and *Mycosphaerella* are Loculoascomycetes, possessing stromatic ascomata and bitunicate asci. They have been separated morphologically on centrum structure: *Didymella* ascomata contain pseudoparaphyses; *Mycosphaerella* ascomata contain only pseudoparenchymatous remnants. Traditionally, *Didymella* has been accommodated in Order Pleosoporales and *Mycosphaerella* in Order Dothideales. A recent 6-gene maximum-likelihood phylogeny of the Ascomycota widely separates these genera in Class Dothidiomycetes: *Didyme*lla in Order Pleosporales and *Mycosphaerella* in Order Capnodiales. Anamorphic states of *Didymella* usually are in the sphaeropsidaceous form-genera *Ascochyta* and *Phoma*. Traditionally, *Ascochyta* is delimited on possession of hyaline bicellular conidia and *Phoma* on featuring hyaline unicellular conidia. Anamorphic states of *Mycosphaerella* are most often hyphomycetous; many form-genera are represented and synanamorphs are relatively common. Both *Didymella* and *Mycosphaerella* include many plant pathogens and these genera have been, and are, confused with one another.

O02

SEVERITY AND DISTRIBUTION OF *PHOMA KOOLUNGA* ON ASCOCHYTA BLIGHT-AFFECTED FIELD PEAS IN SOUTH EASTERN AUSTRALIA.

J.A. Davidson, A. McKay, M. Krysinska-Kaczmarek, *SARDI, South Australia, and E.S. Scott, University of Adelaide, South Australia. Email davidson.jenny@saugov.sa.gov.au*

Introduction – *Phoma koolunga* is as an important component of the ascochyta blight complex of field peas in South Australia (Davidson *et al.*, 2009). The distribution of *P. koolunga* across south eastern Australia was investigated and its incidence relative to *Mycosphaerella pinodes* and *Phoma medicaginis* var. *pinodella* compared in ascochyta blight-affected field trials.

Materials and Methods –Field experiments were conducted in 2007 and 2008 at three sites in South Australia, with high, medium and low annual rainfall and two or three times of sowing. The % severity of ascochyta blight per plot was assessed at regular intervals during the growing season, at which time 6 plants were collected from each trial x time of sowing. The plants were assessed for % disease severity and pathogens were isolated from representative lesions. DNA assays were used to quantify (a) *M. pinodes* plus *P. medicaginis* var. *pinodella* and (b) *P. koolunga* in the plant material. The assays were based on the highly conserved ITS region of the genome (Davidson *et al*., 2009). The pg of pathogen DNA per gm of plant tissue were square root transformed and regressed against % disease severity assessed in plots and on the collected plants. Soil samples from commercial fields in south eastern Australia were submitted to SARDI to test for the presence of the ascochyta blight pathogens using the DNA assays described above (Ophel-Keller *et al.,* 2008). Results of the tests were mapped to nearest town.

Results and Discussion – *M. pinodes* comprised 54% of the 697 isolates obtained, whereas 41% were *P. koolunga* and 5% *P. medicaginis*. *P. koolunga* was the most common pathogen at the medium rainfall site in both seasons. There was a significant ($P<0.001$) exponential relationship between the square root pg of DNA of the pathogens in plant material and % disease severity assessed in plots (R^2 ~0.56) and % disease on collected plants (R^2 ~0.72). There was a significant logarithmic (R^2 ~0.502, $P<0.001$) relationship between the amount of DNA of *P. koolunga* and DNA of *M. pinodes* plus *P. medicaginis* var. *pinodella* in the plant material. These results indicate that *P. koolunga* and *M. pinodes* were, in general, equally responsible for the ascochyta blight symptoms in these trials. *P. koolunga* was detected in many of the soil samples widely distributed over the pea cropping area of south eastern Australia. The extensive distribution of this pathogen across south eastern Australia and it ability to cause severe disease on field peas mean that it must be considered in any management or resistance program aimed at controlling ascochyta blight of field peas in this region.

References

Davidson, J.A., Hartley, D., Priest, M., Krysinska-Kaczmarek, M., Herdina, McKay, A. and Scott, E.S. (2009) A new species of *Phoma* causes ascochyta blight symptoms on field peas (*Pisum sativum*) in South Australia. Mycologia, 101(1): 120-128.

Ophel-Keller, K. McKay, A., Hartley, D., Herdina, Curran, J. (2008) Development of a routine DNA-based testing service for soilborne diseases in Australia. Australasian Plant Pathology, 37(3): 243-253.

Acknowledgements

This research was funded by South Australian Grains Industry Trust (SAGIT). L. McMurray, M. Lines and W. Shoobridge conducted field trials. Ina Dumitrescu, Russell Burns, Danuta Szot, Irena Dadej and Aiden Thomson for processing the samples.

O03

IDENTIFICATION OF *ASCOCHYTA* AND *PHOMA* SPECIES ON CLOVER: COMPARISON WITH OTHER SPECIES FROM FABACEAE.

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Introduction:

Fodder Fabaceae are an essential component of animal feed. They are often attacked by some *Ascochyta* and *Phoma* species, in particular those frequently isolated from berseem (*Trifolium alexandrinum*) in Algeria. These pathogens are difficult to differentiate because the confusing symptoms which they cause and their morphological and taxonomic complexity. The names of some species have been revised which leads some confusion in their identification (I.e. *P. medicaginis* var. *pinodella = A. pino*della = P. *trifolii = P. pinodella) (Boerema et al.,1993; Boerema, et al. 2004).* Hence our interest in their identification and characterization using Random Amplified Polymorphic DNA (RAPD). In this study we have taken some isolates of *Ascochyta* and *Phoma* isolated from clover to compare them with some species already identified on several fabaceae (Bouznad and al, 1996; Corbiere and Bouznad, 1998) using morphological criteria and molecular markers (RAPD) to their identification and characterization.

Materials and Methods: Our study included 4 isolates obtained from clover and 32 isolates with morphological characters *Ascochyta* and *Phoma* species isolated from different regions on other Fabaceae (Red clover, peas, chick peas, alfalfa). At first, all isolates were characterized by morphology of their conidia (measuring the length, diameter and the estimated percentage of single and bi-cellular conidia. Then inter and intra specific polymorphism was also specifically studied by analysis of mitochondrial DNA, extracted by Tris-HCL, EDTA and SDS method (White et al 1990). The amplification was performed using Operon primer:C13 (AAGCCTCGTG), according to the protocol described by Bouznad et al, (1995).

Results and discussion:

Characterization of all isolates showed a high morphological variability and was classified into four groups: those with only bi-cellular conidia with a length included between 10 and 16µm and those with both single and bi-cellular conidia (7.8 - 12µm) but are characteristics of the genus *Ascochyta* species. This last group contains an isolate obtained from Berseem (*Trifolium Alexandrinum*) with mostly bi-cellular conidia (12.5 - 17.5 µm).It seems to be close to pathogens belonging to the anamorph *Ascochyta* genera, probably the species *Ascochyta trifolii* described by Saccardo (1931). The others isolates with mostly single conidia (7.8 to 10µm) are similar to *P. pinodella* of peas and/or *P. medicaginis* of alfalfa according to Boerema (2004) but the conidia have the same morphological aspect that *Ascochyta* spp.

RAPD analysis of the same isolates also revealed a large inter and intra specific polymorphism. One of the isolate of clover with large size conidia mostly bi-cellular is distinguished by its molecular profile from *Mycosphaerella pinodes* and *Phoma pinodella.* It is located in a different cluster from them**.** These results suggest the existence of other species of *Ascochyta* and *Phoma* not described yet on some *Fabaceae*, especially on clover. They are related to *M. pinodes* and *P. pinodella* by RAPD markers but distant from *Ascochyta* species of food legumes (*A. pisi, Ascochyta lentis, A. viciae*). Some of them are known on food legumes such as *P.pinodella ,* but others isolates are different and also distant from *P. exigua* and *P. herbarum.*

In conclusion, classical morphological description and application of RAPD method allowed showing clearly the presence on Clover several species near of the genus *Phoma*. It is necessary in the future to follow up this research to clarify the identification of such isolates including pathogenic specificity of these isolates from clover.

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O04

TEMPERATURE ADAPTATION AND ECOLOGICAL DIVERGENCE OF THE FUNGAL PATHOGEN *DIDYMELLA RABIEI* ON SYMPATRIC WILD AND DOMESTICATED CHICKPEA.

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Introduction - For millennia, chickpea (*Cicer arietinum*) has been grown in the Levant sympatrically with wild *Cicer* species. Chickpea is traditionally spring-sown while its wild relatives germinate in the autumn and develop in the winter. It has been hypothesized that the human-directed shift of domesticated chickpea to summer production was an attempt to escape the devastating Ascochyta disease caused by*Didymella rabiei*. This pathogen spreads in winter and with the combination of mild temperatures and several rain events may cause total destruction of the crop, while the hot and dry conditions in late spring are less favorable for the pathogen. Recently, the pathogen was isolated from natural populations of wild *Cicer* species (*C. judaicum*), (Frenkel et al. 2007). These isolates have shown better adaptation to their original hosts (Frenkel et al. 2008). The objectives of the research were to study the genetic base of the temperature adaptation and its potential role in the pathogens' divergence.

Materials and Methods - The genetic base of temperature adaptation was tested according to the distribution of the *in vitro* hyphal growth phenotype. We used 66 *D. rabiei* isolates and 210 progeny from crosses between *D. rabiei* isolates from wild and domesticated chickpea. Selected progeny and isolates were also tested *in vivo* in controlled environment chambers which simulated the Middle Eastern winter (12-20°C) and late spring conditions (21-29°C).

Results and Discussion – Isolates from domesticated chickpea demonstrated increased adaptation to higher temperatures when grown *in vitro* compared with isolates from the wild host. The distribution of temperature response among progeny from crosses of *C. judaicum* isolates × *C. arietinum* isolates was continuous suggesting polygenic control. Some progenies had better adaptation to higher temperature than their parents (transgressive segregation). Growth chamber experiments did not pointed on association between temperature adaptation and pathogenic fitness. In addition, pathogenic fitness of the parents under favorable conditions (on their original host and in typical temperatures) was higher than their progeny. The results indicate that there is a potential for adaptation to higher temperatures, however the chances for formation of hybrids which are capable of parasitizing both hosts in broad temperature range are low. We hypothesize that this pathogenic fitness cost is due to breakdown of co adapted gene complexes controlling pathogenic fitness on each host and may be responsible for maintenance of genetic differentiation between the pathogen demes.

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O05

Did the development of Ascochyta blight on winter and spring pea (*Pisum sativum*) in France depend on the same populations of *Mycosphaerella pinodes?*

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Introduction

Plant diseases are caused by pathogen populations made up of individuals, continuously subjected to evolutionary forces. Ascochyta blight, caused by *Mycosphaerella pinodes*, is one of the most damaging necrotrophic pathogens of field pea worldwide. Some studies on time variation and according to location have shown genetic and pathogenic diversities among isolates. In France, winter and spring peas are both cultivated. These crops show an overlap period of 4 months (March to June), however, ascochyta blight does not appear at the same period and in the same conditions. Winter pea is particularly subject to the disease because of factors such as length of the growing season, conducive climatic conditions, and the high level of infection (Schoeny *et al*., 2007). Disease starts on December when temperature is generally lower than 10°C. On spring pea, disease starts at the end of May when temperature is equal to 18°C and rainfall periods are shorter than during winter period. These observations suggest that the development of ascochyta blight on winter and spring pea could depend on two different *M. pinodes* populations.

Materials and Methods

In order to specify this hypothesis, isolates of the pathogen (200) were collected in the field during the winter and the spring growing season 2005-2006 in Rennes (western France). Isolates were recovered each month from pea plants (cv Cheyenne for winter pea, and cv. Baccara for spring pea), in a plot sowed in Rennes during autumn 2005 and spring 2006. From these 200 isolates, 20 were randomly chosen for the winter period (symptoms in January-February 2006) and 20 were chosen for the May-June 2006 period. Isolates were characterised by biological and molecular methods (Zhang *et al*., 2003; Onfroy *et al*., 2007), and the structuration of the pathogen population was studied using AFLP. The aggressiveness of these isolates was studied on four pea genotypes (2 winter cv: Cheyenne and Dove, and 2 spring cv: DP and Baccara) growed either at 8-10°C or 18-20°C. Aggressiveness was studied on detached stipules among the method described by Onfroy *et al*. (2007).

Results and Discussion

Isolates from winter pea showed a higher disease level on the four genotypes than isolates from spring pea when these four pea genotypes were previously cultivated in winter conditions or the other way round. Isolates developed different disease levels according to the pea genotypes tested but these differences were not correlated with the origin of the isolate. Moreover, based on the AFLP profiles, the results showed that a genetic variability was detected in the population of *M. pinodes* isolated on winter and spring pea; however, these isolates were not differentiated by the origin of the pea culture. These observations suggested that the disease developed on winter and spring pea was initiated by a same population of *M. pinodes* which should be designed by the flow of genotypes during the overlap period or by the climatic factors.

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Session II
Host Resistance I
(Breeding)

O06

BREEDING FOR ASCOCHYTA RESISTANCE IN DESI CHICKPEA.

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Introduction – Ascochyta blight (AB), caused by *Ascochyta rabiei* is a major foliar disease of chickpea (*Cicer arietinum* L.) in areas where the crop season is cool and wet (Pande et al. 2005). It affects both desi (colored seed, mostly brown) and kabuli (white seed) chickpeas equally. Over 80% of the world's chickpea area is under desi type and the countries/regions where desi chickpea is widely grown and affected by AB include north-western India, Pakistan, Australia, Canada and Ethiopia. This paper deals with the progress made in AB resistance breeding in desi chickpea in recent years through collaborative efforts between ICRISAT and Universities/ Research Institutions in Western Australia and Northern India.

Materials and Methods – The sources of AB resistance used included several germplasm accessions (ICC 3996, ICC 12004, ICC 12965, ICC 14917) and cultivars/breeding lines (ICCV 98502, ICCV 98503, ICCV 04512, ICCV 04516, ICCV 04538, ICCV 05529, ICCV 05530, PBG 5, GL 90135). These were crossed with selected cultivars from Western Australia and India and segregating generations (F_4 or F_5) were screened for AB resistance at seedling stage under artificial epiphytotic conditions in controlled environment chamber. The progenies developed were further screened for AB resistance in field conditions in Northern India and Western Australia. These lines were also screened for resistance to botrytis grey mould (BGM) and fusarium wilt (FW) and evaluated for phenology, agronomic traits, seed quality and yield.

Results and Discussion – Desi chickpea breeding lines have been developed that have shown high level of AB resistance both at seedling (under controlled environment screening at ICRISAT-Patancheru) and at adult-plant (under field screening in northern India and Western Australia) stages. Breeding lines have also been developed that have combined resistance to AB (score 3 to 4), BGM (score 4) and FW (< 10% plant mortality). The new AB resistant breeding lines offer a wide choice of maturity duration (early to late) and many of these lines have acceptable seed quality. Selected lines are further being evaluated for agronomic performance and adaptation in target environments.

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O07

GENETIC ENHANCEMENT OF CHICKPEA FOR ASCOCHYTA BLIGHT RESISTANCE

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Introduction – Ascochyta blight (AB) caused by *Ascochyta rabiei,* occasionally in epidemic form, causes heavy yield losses, sometimes leading to complete crop failures. Although various chemical and cultural practices have been identified to combat this disease, their usage is not eco-friendly and with the presently cultivated varieties having low level of resistance, is also uneconomical. Thus host resistance seems to be the only alternative. The present paper on genetic enhancement of chickpea for ascochyta blight resistance for international environments is therefore presented.

Materials and Methods – Every year we make about 100 crosses (single as well as three-way) for AB resistance at ICARDA and their seed is advanced to F_2. Each F_2 is advanced using single pod descent (SPD) method and F_3 bulks are grown in the off-season and carried further to the next generation using the SPD method. These F_4 bulks are grown in an artificially created ascochyta blight disease nursery (ABDN) for screening for resistance. The disease nursery is developed using debris, a repeated susceptible check, and artificial spore suspension under a mist irrigation system. Seeds from each selected plant are grown as F5 in a single progeny row in a disease nursery, and the AB resistance progenies are selected and their seed is increased for evaluation for other agronomic traits.

Results and Discussion – Every year about 17-19,000 of breeding materials including segregating populations and progeny rows in different generations, elite genetic stocks, and new germplasm as well as mapping populations, are grown in the ABDN. The evaluation for AB resistance is done two times, first when the repeated susceptible check reaches full susceptibility to reveal AB and the second at the podding stage. Following this technique at ICARDA, we have been successful in pyramiding the genes for AB resistance in elite lines with good agronomic background. These elite lines are shared with National Agricultural Research Systems (NARS) through a well established Legume International Testing Program, every year. NARS have identified and released lines with AB resistance and good agronomic background for general cultivation in several countries. Because of the complexity of the AB causing pathogen population, work on identification of molecular markers linked to resistance to different AB pathotypes is in progress, which will complement the conventional breeding program in the near future.

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O08

USE OF GERMPLASM FOR ASCOCHYTA BLIGHT RESISTANCE IN PEA AND LENTIL.

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Ascochyta blight of legumes is caused by a group of largely host specific pathogens that cause necrotic spotting on above-ground plant parts including leaves, stems and pods . *Ascochyta lentis* is the causal organism for Ascochyta blight on lentil (*Lens culinaris* Medik.) and three pathogens, *Ascochyta pisi*, *Phoma medicaginis* var. *pinodella* and *Mycosphaerella pinodes*, contribute to Ascochyta blight on pea (*Pisum sativum* L.). Genetic resistance to these plant pathogens is the most efficient and cost-effective means to control disease (Erskine et al. 1994). World collections of both pea and lentil germplasm held at USDA-ARS, ICRISAT, ICARDA and other repositories are available and have been used to identify useful resistance genes. Resistance genes identified in individual accessions of *Pisum* and *Lens* have been deployed in improved cultivars (Vandenberg et al. 2005; 2006). Resistance is partial and controlled by multiple genetic factors making it difficult to breed improved cultivars (Ye et al. 2004; Tar'an et al. 2003; Prioul et al. 2004). Further characterization of *Pisum* and *Lens* germplasm is needed to identify additional resistance genes for both crops. Complications based on inheritance and availability of resistance genes can limit progress in developing resistant cultivars. Molecular markers linked to genes (QTL) for resistance have been used in the development of cultivars with improved resistance. Continued application of molecular marker technology to characterize germplasm collections offers an opportunity to mine the collections for useful and rare alleles for resistance. These same markers can then be used to deliver and maintain desired alleles in selected progeny, thereby pyramiding resistance genes and improving overall levels of resistance.

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O09

BREEDING FOR IMPROVED ASCOCHYTA BLIGHT RESISTANCE IN PEA

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Ascochyta blight is the most important disease complex of pea in western Canada; thus breeding for improved resistance is a key goal of the Crop Development Centre (CDC), University of Saskatchewan pea breeding program. The CDC pea breeding program is heavily field based, with early generation nurseries and yield trials typically following a cereal-based rotation whereby pea plots are included one year in four. Since the ascochyta blight complex develops naturally in each season, this arrangement ensures that the breeding program is continuously screened for resistance. Prior to bulking sub-lines of a new cultivar, the sub-lines are screened in *Mycosphaerella pinodes* inoculated micro-plots. Any sub-lines with greater than average disease ratings are excluded from the bulk. QTL mapping studies (for example, Timmerman-Vaughan et al., 2002 and Tar'an et al., 2003) have resulted in the identification of numerous genomic regions involved in the control of the resistance, confirming the polygenic nature of resistance to ascochyta blight in the germplasm studied. At the CDC we are characterizing a new recombinant inbred line (RIL) population for mycosphaerella blight resistance based on cultivar CDC Striker. We have initiated a study to characterize pea germplasm from Eastern Europe for mycosphaerella blight resistance and other traits. For the most promising accessions, we plan to assess allele diversity at loci for mycosphaerella blight resistance (Prioul-Gervais et al 2007). Wild pea (*Pisum fulvum, Pisum sativum* ssp. *elatius*) accessions may also provide new alleles for improved mycosphaerella blight resistance (Fondevilla et al. 2005), and thus we have initiated a study with this goal (see V. Gurusamy abstract). Incidence of seed-borne *Ascochyta pisi* in pea has increased in Saskatchewan in recent years, and variation in resistance levels among varieties was detected in a greenhouse study (Banniza et al. 2007). Field studies are in progress to characterize the extent of yield losses. In addition, a RIL population has been developed from a cross between cultivars with contrasting reaction in the greenhouse. Field pea production in western Canada has declined in regions in which mycosphaerella blight has been most prevalent, typically the wetter regions, and has risen in regions in which the disease has been less prevalent. We plan to investigate the foliar and root diseases of pea most prevalent in the Black soil zone of Saskatchewan to develop a region-specific control strategy.

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O10

CONTROL OF PARTIAL RESISTANCE TO *MYCOSPHAERELLA PINODES* IN PEA

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Introduction

Ascochyta blight, caused by Mycosphaerella pinodes, is the most destructive foliar disease in field peas worldwide. Resistance in breeding lines is partial, controlled by minor genes (Prioul et al., 2004). Our purpose was to identify, in three RIL populations generated from three different resistance sources, QTL associated with partial resistance, using isolates showing different aggressiveness levels.

Material and methods

Three RIL populations derived from the crosses Térèse x Champagne, JI296 x DP and JI296 x FP (partially resistant genotypes underlined), and RIL parental lines as controls, were assayed in disease resistance tests. Plants were grown in a growth chamber under hardening conditions until the 5-6 leaf stage. Inoculation was carried out either by spraying on whole plants or by depositing a drop on detached stipules (Onfroy et al., 2007) of pycnidiospore suspensions prepared from three monosporic strains varying in agressiveness (Onfroy et al., 1999). Disease severity and components of resistance were assessed using semi quantitative scales. Adjusted means were used for QTL detection on genetic maps developed from the three RIL populations and related to a SSR based pea reference map (Loridon et al., 2005).

Results and discussion

Four, five and four QTL were detected on Térèse x Champagne, JI296 x DP and JI296 x FP populations respectively, for plantlet resistance to M.pinodes under controlled conditions, which were consistent across organs (stem and stipule) and across at least two of the strains assayed. QTL projection on the pea reference map allowed to infer three QTL common to all three resistance sources, and one specific to a single resistance source. Involvement of these QTL into the control of symptom appearance or lesion diameter extension gives insights into the identification of choice QTL for marker-assister selection.

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O11

EXPLORATION OF RESISTANCE TO *MYCOSPHAERELLA* BLIGHT IN WILD *PISUM* SPP. TO DEVELOP RESISTANT FIELD PEA GERMPLASM

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Field pea production in western Canada has increased steadily since the early 1980's, but ascochyta blight mainly caused by *Mycosphaerellapinodes* can substantially reduce yields in the region (Chang *et al*, 1999). Available genetic resistance to ascochyta blight accumulated through two decades of breeding reduces the damage from disease, however, under cool, wet conditions, the resistance is not sufficient to prevent economic losses. Several studies by various researchers indicate that complete or high level resistance for ascochyta is not available in the accessions of *Pisumsativum,* the cultivated species (for example, Zhang et al, 2006). Fondevilla *et al*. (2007) demonstrated that a few accessions of *Pisum fulvum*, a wild relative of field pea, possess a high level of resistance to ascochyta blight. We have initiated the research necessary to implement a breeding strategy for improving and maintaining resistance to ascochyta blight by systematically screening for new resistance genes to ascochyta blight in selected *P. fulvum* and other wild pea accessions. A total of 67 wild accessions of *Pisum spp*. were collected from USDA, Pullman and IFAPA, Spain and the seeds were multiplied under controlled conditions. Fifty three wild accessions (based on the seed availability) along with two check cultivars (CDC Striker and Alfetta) were raised under greenhouse conditions. Plants were inoculated 2 weeks after seeding with spore suspensions of isolate MP25 at a concentration of 5 x 10^5 spores mL^{-1}. The experiment was conducted in a RCBD with 5 replicates and was conducted twice. Plants were scored for disease on 10 days after inoculation using a 0 -5 scale as defined by Tivoli (1994). Data were analysed using the mixed procedure in SAS and means were speárated based on Fisher's least significance. The wild accessions of *Pisum* sp. differed significantly in their resistance level to the ascochyta blight (p=≤0.0001). Among the 53 accessions tested, 8 accessions including *P. fulvum* and *P. sativum* ssp. *elatius* were found to be moderately resistant with scores from 2.2 to 3.4, while both cultivar checks scored 5. The results on the varying levels of resistance and susceptibility for ascochyta blight among the wild pea accessions, and the future plans for the exploitation of this variability will be discussed.

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O12

ENHANCEMENT OF BLACK SPOT RESISTANCE IN FIELD PEA

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Introduction

Black spot, also known as ascochyta blight, is the most important disease of field pea across southern Australia. *Mycosphaerella pinodes* is the major causal organism in Australia. Black spot is one of the major factors for yield decline in field peas in South Australia (Davidson & Ramsey, 2000) and yield losses are generally in the order of 20-30% (Bretag *et al*., 1995). A hot spot for natural epidemic of black spot has been identified at Medina, Western Australia (WA). Consequently, WA has been regarded as a primary site for enhancing black spot resistance in the Australian Field Pea Improvement Program (AFPIP).

Materials and methods

In 2008, more than 350 breeding lines from the AFPIP and nearly 300 lines from the Australian Temperate Field Crops Collection (ATFCC), Horsham, Victoria were screened at Medina. About 40 crosses are made every year. They include crosses between best resistant lines and elite agronomic lines to develop varieties, and also amongst resistant lines. The early generations are advanced using the single seed descent (SSD) method in glasshouses. Single plants are selected at F_4-F_5 stage under natural epidemic. Genetically stable lines are screened for resistance as well as for agronomic desirability over two years before being advanced to yield trials.

Results and discussion

The disease in 2008 developed unusually early. All the susceptible lines, such as Dundale and Helena were heavily infected with almost the whole plant covered with disease. Fifteen lines from the ATFCC showed some resistance. Accession ATC 6296 from Moldova was early flowering, but all others were landraces collected mainly from Henan Province in China with some wild characteristics and were very late flowering. None of the lines in the above germplasm were more resistant than the breeding line WAPEA2211. WAPE2211 is the first germplasm developed in an agronomically suitable background in WA with a moderate level of resistance and is used as a benchmark. More than 50 F_5/F_6 lines showed some resistance and among them a dozen lines had higher resistance than WAPEA2211. All the latter lines had good agronomic features and yielded greater than cv. Kaspa in a small plot trial. At least six lines in yield trials showed a significantly higher resistance than WAPEA2211. It appears that late flowering lines have better resistance than early flowering lines. More promising resistance has been seen at earlier stages of the breeding cycle showing a good promise for the breeding strategies applied.

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Acknowledgement

This program is supported by the Australian Grain Research and Development Corporation.

O13

MAPPING OF ASCOCHYTA BLIGHT RESISTANCE IN CHICKPEA.

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Our objective was to map quantitative trait loci (QTLs) conferring resistance to *Ascochyta rabiei* in cultivated chickpea *Cicer arietinum*, kabuli lines ILC72, ILC3279, ILC195 and Amit, desi lines, ICC3996, ICC4200, ICC4475 and ICC6328, and one wild relative *C. reticulatum* line PI489777. Progenies in populations of recombinant lines (RIL) and F_2 were phenotyped for ascochyta resistance in a detached-leaf assay. The lines were genotyped with microsatellite markers using 220 primers from the literature (Winter et al. 1999 and Sethy et al. 2006) and 70 designed from our data base of 30.000 chickpea EST. QTL analysis (Cartographer) confirmed the presence of five QTL previously published in various chickpea lines, on linkage groups LG2 (ILC3279, FLIP84-92C, ICCV04516 and PI48977), LG3 (ILC72, CDC Frontier, ICC4475 and ICC3996), two QTL on LG4 (a: ILC72, ILC3279, Hadas, CDC Frontier, ICC12004, ICCV04516 and PI489777; b: ILC3279 and ICC3996) and LG6 (FLIP84-92C, CDC Frontier and PI489777) (Cho et al, 2004 and Iruela et al. 2006). Four other QTL were new on LG2 (ILC72), LG3 (ICC4200), LG4 (ILC3279) and LG6 (ILC72). Using the chickpea EST sequences, new primers were designed in intron-exon regions inferred by *Medicago* sequence information. DNA from sixteen *C. arietinum*, three *C. reticulatum* and one each of *C. bijugum, C. judaicum, C. anatolicum, C. canariensis and C. songaricum* were screened with 596 of these primers and amplicons examined for single nucleotide polymorphisms (SNP). Only 3 -5% of the SNP markers were polymorphic between any two *C. arietinum* lines which precluded mapping. In contrast, 270 SNP were mapped in an inter-specific population of RIL, *C. arietinum* ILC72 x *C. reticulatum* Cr 5-10 (developed by J. Gill and T. Millán UCO, and J. Rubio CIFA, Córdoba, Spain), resulting in identification of eight SNP markers on LG3 and LG6 closely linked to ascochyta blight resistance. To avoid re-sequencing of DNA harboring these SNP, we successfully tested the KASPar assay (KBioscience, UK), which is based on competitive allele-specific PCR amplification of the DNA followed by fluorescent endpoint genotype detection. The low level of SNP among the *C. arietinum* genotypes, the high number of QTL shared among resistant lines, and the fact that these lines are not highly resistant under field conditions, indicates a need to identify different sources of ascochyta blight in germplasm from diverse parts of the world, and combine QTL, with the help of marker-assisted selection, in ways that increases their effectiveness.

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Session III
Bob Henson Award
Student Competition

O14

A COMPARISON OF PHENOTYPIC AND MARKER-ASSISTED SELECTION FOR ASCOCHYTA BLIGHT RESISTANCE IN CHICKPEA.

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Student presenter

Introduction

Ascochyta blight caused by *Ascochyta rabiei* (teleomorph: *Didymella rabiei*) is one of the most serious diseases of chickpea. QTL for resistance to blight have been located on linkage group 4 (LG4) (QTL_{AR1} and QTL_{AR2}) and LG2 (QTL_{AR3}) of the chickpea map (Iruela et al., 2007). Molecular markers have been associated with these QTLs for resistance to blight. Marker assisted selection (MAS) for Ascochyta blight resistance would greatly accelerate the development of new chickpea cultivars. In this study, MAS and phenotypic selection were employed to select blight resistant chickpea genotypes comparing the effectiveness of both methods.

Materials and Methods

600 F_2 plants derived from the cross ILC3279 x WR315, resistant and susceptible to blight, were used to MAS and phenotypic selection. The genotyped of F_2 plants was performed in a multiplex PCR. STMS GAA47 linked to QTL_{AR1}, STMS TA72 and the SCAR SCY17 to QTL_{AR2} and the STMS TA194, TS82 and TR58 to QTL_{AR3} were analyzed. Early generation testing method of F2-derived lines (Fehr, 1987) was used for resistance selection. Ascochyta blight was evaluated in the field using a 1 to 9 rating scale as proposed by Singh et al (1981). $F_{2:5}$ resistant lines were also genotyped to confirm the presence of the alleles associated with the resistance.

Results and Discussion

The AUDPC data distribution in the $F_{2:3}$ was skewed toward the susceptible parental. Only 58 out of the 558 $F_{2:3}$ families evaluated were resistant. This data suggest that resistance to Ascochyta blight is recessive. Resistance was also confirmed in selected $F_{2:4}$ lines. However, the markers TA72 and SCY17 (QTL_{AR2}) exhibited a strong distorted segregation toward the susceptible parental with respect to the expected Mendelian inheritance (1:2:1) in F2 plants, GAA47 marker (QTL_{AR1}) showed also distorted segregation although in less extension. All of these markers are located in LG4. Most of resistant $F_{2:3}$ lines selected in the field were derived from heterozygous F2 plants according to the mentioned markers. These results suggest that resistance may be dominant. Markers linked to QTL_{AR3} were not associated to resistance. The GAA47 allele associated to resistance is being fixed in the $F_{2:5}$ resistant lines. Markers linked to QTL_{AR2} were not clearly associated to resistance in the selected $F_{2:5}$ lines probably due to the strong distorted segregation. In this study STMS GAA47 linked to QTL_{AR1} was the most reliable marker to predict resistant phenotype and it would be an useful marker in MAS for Ascochyta blight.

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O15

PARTIAL CLONING OF TWO POLYKETIDE SYNTHASE GENES ASSOCIATED WITH PATHOGENICITY OF *ASCOCHYTA RABIEI*.

***J. A. Delgado*, *S. W. Meinhardt, S. G. Markell, and R. S. Goswami*.** *Department of Plant Pathology, North Dakota State University, Fargo, ND, USA. Email: rubella.goswami@ndsu.edu.*
Student presenter.

Introduction

Ascochyta rabiei (Pass.) Labr., is the most important chickpea fungal pathogen in North Dakota. According to previous studies, solanapyrones (mycotoxin) and melanin have been associated with virulence in this pathogen. Solanapyrone crude extracts produce blight symptoms on chickpea leaflets (Hölh et al. 1991) and melanin deficient mutants are non pathogenic (Chen et al. 2004). Both metabolites are synthesized via the polyketide synthase pathway. Polyketide synthases (PKSs) are multidomain proteins and their minimum functional domain structure consists of beta-ketoacyl synthase (KS), acyltransferase (AT), and acyl carrier protein (ACP) domains (Hopwood and Sherman 1990). PKSs have been studied in other fungi, however, to date, little is known about the genetics of these pathogenicity determinants in *A. rabiei.* The work to be presented is part of a larger study that focuses on cloning PKS genes involved in *A. rabiei* pathogenicity.

Materials and Methods

A pathogenic pathotype II, *A. rabiei* isolate from chickpea fields in North Dakota was used for this study. An *A. rabiei* PKS nucleotide sequence available in Genbank was used to initiate the melanin PKS (PKS-Mel) cloning. However, no information of the solanapyrone PKS (PKS-Sol) nucleotide sequence was available. Therefore, initial portions of the PKS-Sol sequences were amplified using degenerate primers designed by alignment of reducing type I PKSs following which, both PKS nucleotide sequences were extended towards the N-terminus using new degenerate primers. Subsequently, a genome walking approach involving construction and screening of four genomic libraries with adaptor specific and gene specific primers was used to extend both PKS sequences towards the N- and C- terminus. The significant PCR products were then cloned and sequenced.

Results and Discussion

We have cloned a 3098 bp region likely to be related to PKS-Sol using degenerate primers and genome walking. The sequence matched several reducing type I PKSs involved in secondary metabolite biosynthesis. The most significant BLASTX match was to a PKS from *Cochliobolus heterotrophus* involved in the biosynthesis of T-toxin. Similarly, a 2466 bp region of a related PKS-Mel was amplified. It had a BLASTX match to several non-reducing type I PKSs involved in melanin biosynthesis with the most significant match being a PKS from *C. heterotrophus*. The translated amino acid sequences of both PKS-Sol and PKS-Mel matched the KS and AT domains using the Conserved Domain Search at the NCBI website. Thus, to date we have partially cloned two different PKS genes involved in the biosynthesis of secondary metabolites and melanin respectively from *A. rabiei.* These are believed to be associated with pathogenicity and further genome walking to obtain the complete gene sequences is in progress.

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O16

COMPARATIVE POPULATION STUDY OF *DIDYMELLA RABIEI* IN TURKEY AND ISRAEL

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Student Presenter

Introduction

To properly infer the evolutionary history of an agricultural pathogen, it is necessary to sample pathogen populations from both domesticated and the wild hosts, especially from the host/pathogen's center of origin.*D. rabiei* isolates were sampled from domesticated chickpea and wild *Cicer* spp. in Israel and southeastern Turkey where chickpea was first domesticated. Different seasonality of the hosts and conditions in natural and agro-ecosystems may affect their pathogens. The main objectives of this study was to compare *D. rabiei* populations from wild and domesticated *Cicer* spp. in Israel and Turkey in view of population genetic structure and *in-vitro* temperature growth response of the pathogen. The results were evaluated using integrated genetic, ecological and evolutionary approaches.

Materials and Methods

A total of 128 *D. rabiei* isolates from naturally infected Turkish and Israeli domesticated chickpea and wild *Cicer* species (*C. pinnatifidum* and *C. judaicum*, respectively) were screened for variation at six STMS loci (1). The *in vitro* hyphal growth response of the 80 *D. rabiei* isolates was determined at 15°C and 25°C (2).

Results

The majority of the microsatellite variation occured within populations. The highest genetic diversity was detected within the Turkish *D. rabiei* populations from domesticated chickpea (H_T=0.68). Genetic distance analysis based on pooled allele frequencies within populations presented two main clusters of isolates from wild and domesticated *Cicer* spp. The model based Bayesian algorithm demonstratedthe highest posterior probability for three populations among all isolates: while Turkish and Israeli isolates from domesticated chickpea took part in population 1 or population 2, most of the isolates from the wild *Cicer* spp. were strongly assigned to population 3.

Isolates from domesticated chickpea were significantly better adapted to 25°C and many isolates from wild host *C. judaicum* were adapted to both 15°C and 25°C.

Discussion

The six STMS markers and *in vitro* temperature responses of colony hyphal growth exposed a distinction between *D. rabiei* from the domesticated and wild *Cicer* spp. hosts. *D. rabiei* populations of domesticated chickpea may have diverged from its ancestral population on wild *Cicer* spp. and the pathogen populations evolved on their wild and domesticated hosts seperately in accord with ecological divergence.Turkish areas of the pathogen that exhibit high genetic diversity are likely to be important sources of host resistance genes, both among wild and domesticated *Cicer* spp. Studying the effect of the temperature on hyphal growth under controlled conditons provided useful information supporting the hypothesis regarding the evolutionary and ecologically effect of the cropping shift of chickpea on its pathogen *D. rabiei.*

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O17

CLONING AND CHARACTERIZATION OF ANONYMOUS REGIONS OF *ASCOCHYTA LENTIS* AND *A. FABAE* GENOMES AND SUITABILITY OF THESE REGIONS FOR PHYLOGENETIC ANALYSIS OF *ASCOCHYTA* SPECIES.

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Student Presenter

Introduction

Ascochyta species cause blights on a number of wild and cultivated cool-season legume hosts, including chickpea (*Cicer arietinum* L.), faba bean (*Vicia faba* L.), lentil (*Lens culinaris* Medik.), pea (*Pisum sativum* L.), and vetches (*Vicia spp*.). Ascochyta blight of faba bean and lentil are caused by the host-specific fungi *A. fabae* Speg. and *A. lentis*Vassiljevsky, respectively. Identification of these species has been primarily based on host specificity because they are morphologically indistinguishable (Gossen et al.1986). Previous studies have demonstrated that each species have distinct RAPD-PCR banding patterns (Kaiser et al. 1997), and each form a monophyletic group in a combined phylogeny estimated from glyceraldehyde-3-phospate-dehydrogenease (*G3PD*), translation elongation factor alpha (*EF*), and chitin synthase (*CHS*) genes (Peever et al. 2007). No additional fast-evolving markers have been identified for these fungi that would facilitate research at the population/species interface. Therefore, the objective of this research was to develop sequence characterized anonymous region (SCAR) markers for identification of *A. fabae* and *A. lentis,* for estimating genetic variation within and among species, and for inferring phylogenetic relationships.

Materials and Methods

Two isolates of each species were used. Random Amplified Polymorphic DNA (RAPD)-PCR was performed using 40 decamer primers. Twenty clones were sequenced from each species. Sequence contigs were assembled, annotated, and sequence specific primers were designed. Sequences were analyzed for SNPs, insertion/deletions and restriction sites between *A. fabae* and *A. lentis*. A BLASTn search was conducted to determine sequence similarity to known genes. Phylogenetic signal was assessed for each developed SCAR locus.

Results and Discussion- Five primers were polymorphic between the species, resulting in 38 polymorphic amplicons. Direct cloning of RAPD-PCR amplicons resulted in 80 and 95 clones from *A. fabae* and *A. lentis*, respectively. A total of 7 intra- and inter-specific polymorphic SCAR markers were developed and characterized demonstrating the usefulness of the technique. BLASTn results of SCAR marker T1 revealed homology to *SirH*, a trichothecene acetyl transferase gene involved in sirodesmin phytotoxin pathway of *Leptosphaeria maculans.* These loci will prove useful for diagnostics and population genetics, phylogeographic, and phylogenetic studies to facilitate studies of speciation of these and related taxa.

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Session IV
Disease Management

O18

***Over forty years on six continents researching Ascochyta* diseases of food legumes.**

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Introduction –I began studying the Ascochyta diseases of food legumes over 40 years ago, and they continue to cause important diseases of these crops in many different countries. In my talk, I will discuss research conducted over the last forty plus years in which I participated on the etiology, biology, epidemiology, life cycles, sexual stages, heterothallism, survival, alternative hosts, and control of these Ascochyta diseases.

Background – Over the years, I have initiated research projects with colleagues in different countries on the African, Asian, Australian, European, North American, and South American continents. The first time I saw an Ascochyta disease was on chickpea in Iran in the mid-1960's when I worked as a plant pathologist on the USDA Regional Pulse Improvement Project. During six years in Iran, I traveled extensively in the Middle East, India, and Pakistan where Ascochyta blight of chickpea was widespread and, at times, a very devastating disease. While in Iran, I began studying the biology, epidemiology, and control of this important disease (Kaiser, 1973).

From 1978 until my retirement from the USDA in 1998, I was associated with the USDA, Western Regional Plant Introduction Station at Washington State University (WSU) in Pullman, WA. While at WSU, I worked cooperatively with scientists, students, agribusiness personnel, and growers on Ascochyta diseases of different food legumes in Africa (Algeria, Morocco, Tunisia), Australia, Asia (India, Iran, Pakistan, Syria, and Turkey), Europe (Bulgaria, France, Germany, Italy, and Spain), and North America (United States and Canada) (Kaiser 1997; Kaiser and Hannan 1986; Kaiser et al. 1997, 2000).

After more than 40 years of conducting research on Ascochyta diseases of food legumes in the laboratory, greenhouse and field, I still maintain a keen interest in these diseases, especially those affecting chickpea, faba bean, lentil, and pea. During retirement, I have continued to investigate the Ascochyta diseases of food and forage legumes in Europe (Armenia, Bulgaria, Republic of Georgia, and Spain) (Kaiser et al. 2008) and in South America (Bolivia where I served as a U.S. Peace Corps Volunteer) (Kaiser et al. 2000).

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O19

A SYSTEM-BASED RISK ESTIMATOR OF ASCOCHYTA BLIGHT DISEASE IN SOUTH AUSTRALIA

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Introduction

'Blackspot Manager' (Salam *et al*., 2006) predicts % ascospore release from ascochyta blight-affected pea stubble for a given time of sowing. This model is used in Western Australia and South Australia to determine optimum sowing dates for field peas to reduce ascochyta blight risk. The current research incorporates regional conditions into 'Blackspot Manager' to develop a disease risk model associated with the ascospore predictions.

Materials and Methods

Severity of ascochyta blight was measured in 3 naturally infected field experiments in 2007 and 2008. Treatments included time of sowing. The experimental sites were in different rainfall regions *viz*. 450mm (high), 350mm (medium) and 325mm (low) per annum. Immediately after harvest in 2006 and 2007, one batch of infested stubble collected from the high rainfall region and three batches, of different disease severity, collected from the medium rainfall region were incubated in nylon mesh bags on the soil surface in their region of origin. Each fortnight, one bag of stubble per batch was wetted and ascospores from the stubble were captured on slides in a wind tunnel and counted microscopically. A disease risk model was developed incorporating (a) absolute ascospore numbers per region calculated from the actual number of ascospores released from the stubble, (b) % ascospores remaining to be released at sowing for each site x time of sowing predicted by 'Blackspot Manager', (c) distance from infested stubble, (d) field pea cropping intensity in the region, (e) infectivity factor measured as low, medium or high winter rain, and (f) pea rotation factor. Commercial pea crops in the medium rainfall zone were surveyed in October (spring) of 2008 to validate the model. The calculated disease risk and absolute ascospore numbers at sowing were regressed against actual disease severity in the trials and the commercial crops.

Results and Discussion

There was a linear relationship (R^2 ~0.87) between effective number of ascospores up to 400per g infested stubbleand observed severity in trials and surveys; thereafter the relationship was flat. In medium and high rainfall regions there was a high disease risk associated with 20% ascospores remaining on stubble at sowing, while in the low rainfall region there was little disease irrespective of ascospore numbers. In the medium rainfall area, crops distant (>400m) from infested stubble had reduced risk so that 50% ascospores needed to be remaining on stubble at sowing for disease risk to be high. This model may be used in conjunction with 'Blackspot Manager' to optimise management strategies that reduce ascochyta blight on field peas in different rainfall and cropping regimes.

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Acknowledgements

Technical assistance by P. Payne, T. Humphreys (DAFWA), C. Wilmshurst, M. Krysinska-Kaczmarek (SARDI), P. Hooper, S. Sherrif. GRDC funded development of 'Blackspot Manager'. L. McMurray, M. Lines, W. Shoobridge (SARDI) conducted field trials, funded by SAGIT.

O20

MANAGEMENT OF ASCOCHYTA BLIGHT OF CHICKPEA IN INDIA.

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Introduction

Ascochyta blight caused by the fungus *Ascochyta rabiei* (perfect stage *Didymella rabiei*) Kovachevski) is the most devastating disease of chickpea worldwide, and yield losses up to 100% have been reported (Acikgoz et al 1994). In the Indian subcontinent, the disease is predominant in the North-West Plain Zone of India and in the northern regions of Pakistan, causing severe yield loss and instability in yield. This is one of the major reasons that chickpea cultivation in northern Indian states has gone down drastically. To bring back the crop in the northern traditional chickpea growing areas of India, chickpea researchers have developed integrated disease management option. This includes use of resistant or partially resistant cultivars, minimizing initial onset of epidemics through cultural practices and seed treatment with appropriate chemicals, and suppressing disease development by foliar fungicides.

Materials and Methods

More than 5000 germplasm and breeding lines have been screened at Dhaulakuan and at Ludhiana, hot-spots for Ascochyta blight disease in India. The indicator-cum infector lines (L 550, ILC 1912, ICCV 96029, Pb 7) were planted after every 2-4 test lines. Infected plant stubbles were added during land preparation. At flowering, the materials were artificially inoculated by spore suspension of *A.rabiei* @ 1x $10^{5- 1ml}$. Relative humidity above 85% was maintained by perfo-spray system from 10-16 h daily. Screening in controlled conditions is being carried out using cloth chamber, cut twig and detached leaf techniques. Cultural practices like disease-free seed, crop rotation, manipulation in sowing time and plant spacing, inter-cropping, etc. are being carried out. Seed treatment with Captan, Hexacap, Bavistin + Thiram (1:1), Rovral @ 3 g/kg of seed and based on disease forecast, 1-2 sprays of Thiabendazole/Carbendazim/ Triozoles were applied.

Results and Discussions

Sources of resistance to Ascochyta blight have been identified in local germplasm, breeding lines developed by Indian programs and at ICARDA and ICRISAT. Sources of resistance to Ascochyta blight in world germplasm collection have been reported (Basandrai et al. 2008). Recently 30 genotypes comprising of Desi and Kabuli types have been identified at Dhalakuan with a disease reaction of ≥3 on a 1-9 scale. A number of lines with durable resistance have been identified at PAU, Ludhiana. Resistant varieties, Himachal Channa 1, Himachal Channa 2, GPF 2, HPG-17, PBG1, PBG2 proved promise among the farmers of northern India following seed and crop hygiene with seed treatment and foliar fungicide like difenconalzole

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O21

MANAGEMENT OF ASCOCHYTA BLIGHT OF CHICKPEA IN NORTHERN NSW.

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Introduction

Chickpea is the main pulse crop of north-eastern Australia which covers the region of northern New South Wales and southern Queensland and represents the largest chickpea growing region in Australia. In 2008, favourable conditions for disease resulted in multiple infection periods and the highest levels of ascochyta blight development in commercial chickpea crops since the first disease major epidemic in eastern Australia in 1998. Ascochyta blight was found widespread with almost every commercial crop inspected having some disease: pod and seed lesions were common. The severity of disease ranged from no impact on yield and grain quality to 100% loss. In 2008, an ascochyta blight management trial was conducted at Tamworth to evaluate the currently recommended Variety Management Packages (VMPs) for five desi varieties (Jimbour - highly susceptible, Kyabra - highly susceptible, Yorker - intermediate, Flipper - moderately resistant and CICA512 - resistant and the small seeded kabuli Genesis425 - resistant), under high disease pressure conditions.

Materials and Methods

The experiment was sown on the 27th June 2008 and inoculated on 30th August just prior to a rain event. The site received 430 mm rain between inoculation and harvest and was exposed to 25 potential infection periods (leaf wetness >3 hr). Two control treatments were used; one to maximise disease (nil fungicide application) and one to minimise disease (a pre-inoculation application and then regular applications with 1.0 L/ha fungicide containing 720 g/L chlorothalonil). A third treatment was also applied to all varieties comprising a pre-inoculation application and then regular applications of 1.0 kg/ha fungicide containing 750 g/kg mancozeb. The VMP treatments varied according to variety: Jimbour and Kyabra – 500 mL/ha fungicide containing 720g/L chlorothalonil: Yorker - an initial 1 kg/ha mancozeb product then 500 mL/ha chlorothalonil product; Flipper, CICA512 and Genesis425 - first spray not until after 93 mm rainfall and 4 infection events (5 weeks) after inoculation, by which time ascochyta had established in the lower half of the canopy.

Results and Discussion

The trial demonstrated the relative susceptibility/resistance to *Ascochyta rabiei* of the varieties and showed that as susceptibility increases, spraying for ascochyta blight becomes critical. Not protecting Jimbour and Kyabra resulted in virtually 100% yield loss. Conversely, as resistance increases the need to spray for *A. rabiei* becomes less important. For example, results suggest that a farmer should be able to grow CICA512 with no in-crop fungicides and get the same yield as if they had sprayed six times with 1 L/ha chlorothalonil. The experiment also showed that 1 kg/ha mancozeb is cost effective on varieties whose resistance to *A. rabiei* is as good or better than Yorker. However 1 kg/ha mancozeb was less effective than 500 mL/ha chlorothalonil on very susceptible varieties (i.e. Jimbour, Kyabra). Whilst 1 L/ha chlorothalonil gave the least disease and the cleanest plots, it gave the highest gross margins ($/ha) only on the most susceptible varieties. The most profitable ascochyta blight strategy on the other varieties was either the VMP i.e. 500 mL/ha chlorothalonil or 1 kg/ha mancozeb.

Session V
Molecular Biology

O22

FUNCTIONAL GENOMICS OF DOTHIDEOMYCETES; APPLICATIONS TO LEGUME PATHOGENS

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The dothideomycetes is a large group of filamentous ascomycetes many of which are significant crop pathogens. It includes the Ascochyta pathogens; neighbours within the Pleosporales include the cereal pathogens Stagonospora, Pyrenophora and Cochliobolus and the crucifer pathogen Leptosphaeria spp. and the generalist Alternaria. More distant relatives include Venturia, and Mycosphaerella. The diseases caused by the pathogens in this class have striking similarities. Most diseases are clearly necrotrophic, causing rapid host-cell necrosis. Others are best described as temporal hemibiotrophs – defined as an extended (days to months) asymptomatic phase followed by a necrotic phase accompanied by sporulation and spread. It is not clear whether the pathogen feeds from host cell sources during the asymptomatic phase. Most of these pathogens seem to be favoured by agricultural practices that retain stubble from season to season. Host resistance is typically partial at best. Genetic analysis of resistance usually reveals multiple, weak quantitative trait loci. Marker-assisted crop breeding has had a limited impact so far because host genetic resources are inadequate and validated markers are rare. Finally, fungicidal control is generally regarded as inefficient, uneconomic and environmentally damaging.

This bleak picture is exemplified by the wheat disease Stagonospora nodorum blotch in Western Australia. It has been consistently amongst the top two or three pathogens of all crops in Australia. Early work by Chris Caten and colleagues had demonstrated that it was amenable to many critical laboratory procedures. Our laboratory therefore set out to study this pathogen and to use it as a model to investigate the related pathogens of the grain legumes.

S. nodorum was the first dothideomycete to be publicly sequenced (2005) and the preliminary annotation was released in 2007. Analysis of the sequence and use of functional genomics tools has had a significant impact on our understanding of the disease and opened up a clear pathway for the substantial control of losses. Whereas we had thought of *S. nodorum* as a relatively unsophisticated pathogen that interacted with wheat via a series of non-specific toxins and cell-wall degrading enzymes, we know recognise that the disease plays out via the interaction of numerous highly specific secreted fungal effectors and corresponding wheat receptors. Interaction of an effector with a receptor leads to host cell necrosis and an open feeding site for the pathogen. We postulate that the disease is integral to all effector/receptor interactions. Disease control can therefore be achieved by identifying host receptors and removing their genes from current cultivars. We have found that the easiest route to identifying host receptors is to isolate the fungal effectors.

Preliminary evidence suggests that this model applies to the legume-infecting Pleosporales. Therefore we have set out to establish functional genomics tools for pea and chickpea diseases. Comparison with the other dothideomycetes has indicated that significant similarities between them will assist the analysis of the legume pathogens and the identification of their effectors. Furthermore, intriguing evolutionary relationships have been identified which may explain the current status of these diseases.

O23

PR PROTEINS IN LENTIL: ISOLATION AND EXPRESSION IN RESPONSE TO *ASCOCHYTA LENTIS* AND SIGNALLING COMPOUNDS.

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Abstract is withheld for a peer-reviewed publication.

O24

APPLICATIONS OF SUPPRESSION SUBTRACTIVE HYBRIDIZATION (SSH) IN IDENTIFYING DIFFERENTIALLY EXPRESSED TRANSCRIPTS IN *ASCOCHYTA RABIEI*.

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Introduction

Ascochyta rabiei, casual agents of chickpea ascochyta blight, is divided into two pathotypes based on virulence levels (Chen et al., 2004). Genetic mechanisms of this phenotypic differentiation are poorly understood. This research is directed toward understanding molecular differences between the two pathotypes during early stages of conidial germination by employing SSH.

Materials and Methods

cDNA pools were synthesized from mRNAs from germinating conidia of strains AR19 (pathotype I) and AR628 (pathotype II). Two distinct cDNA subtractions were performed (Diatchenko et al., 1996) to generate pools of pathotype I and pathotype II-enriched transcripts. Pools of enriched transcript from each pathotype were cloned into the pCR2.1 TOPO vector to create libraries of enriched transcript. In addition, differential display (virtual Northerns) was performed on each pathotype-specific library using probes generated from total or subtracted RNA from each pathotype. Clones from each library that exhibited differential expression were sequenced. Southern hybridizations were performed to determine if each pathotype carried the genetic material for each enriched transcript. Temporal expression of three pathotype II transcripts, a ubiquitin, an ADP-ribosylation factor, and a translation initiation factor, were monitored using RT-PCR over 36 h of early infection on chickpea material. Expression of each transcript was compared between four pathotype I and four pathotype II isolates.

Results and Discussion

Three up-regulated transcripts in pathotype I were identified, which are highly homologous to a hypothetical protein, a transaldolase, and an alcohol oxidase of other fungi. Five up-regulated transcripts in pathotype II were identified with homology to fungal gene products of a ubiquitin, an ADP-ribosylation factor, a translation initiation factor, and a gene involved in nitrate assimilation. The genomic DNA pathotype I isolates carries the genetic component of each up-regulated pathotype II transcript. Expression over the first 36 h of germination on plant material using RT-PCR showed that pathotype I isolates exhibited high expression of the ubiquitin, ADP-ribosylation factor, and translation initiation factor transcripts during the first 12 hours followed by a rapid decrease between 12 and 24 h. Conversely, pathotype II isolates exhibited low initial expression of the 3 transcripts during the first 12 h and increased dramatically between 12 and 24 h. Results show that the differences between the two pathotypes are not only in levels of expression of certain transcripts during conidial germination, but also in temporal expression during the first 36 hours of early stages of germination. Exploring the roles of the transcripts will help us understand the different pathogenic mechanisms of *A. rabiei* pathotypes.

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O25

INDUCED MUTATIONS FOR *ASCOCHYTA* BLIGHT RESISTANCE IN CHICKPEA (*CICER ARIETINUM* L.)

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Introduction

Chickpea is the most important rabi legume crop of Pakistan. The average yield of the country is quite low which is due to the occurrence of *Ascochyta* blight and *Fusarium wilt* diseases. Chickpea blight caused by *Ascochytarabiei* has been a major limiting factor of chickpea production in the country. The average yield of chickpea was dropped as low as 228 Kg/ha in the epidemics of 1979-80 to 1981-82 (Akhtar 2008). Since most of the commercial high yielding cultivars of chickpea in the country have been found to be susceptible, there is great need for the improvement of disease resistance in these high yielding varieties using induced mutations. It was designed to conduct the screening of M_2 segregating population and promising morphological mutants in M_3 and M_4 generation for the identification of mutant(s) having improved level of resistance to *Ascochyta* blight in four chickpea genotypes.

Materials and Methods

The seed material used for this study consists of four chickpea genotypes, i.e. two desi (Pb2000 and C44), one kabuli (Pb-1) and one recombinant of desi x kabuli introgression (CH41/91).The M_2 and M_3 material resulting from mutagenic seed treatment was screened in the *Ascochyta* blight screening nurseries at NIAB, Faisalabad during 2001-2002, 2002-2003 and 2003-2004, respectively. Chickpea plants grown in the screening nursery were initially sprinkled by plain water for creating artificial humidity followed by spray of spore suspension of virulent isolate of *Ascochyta rabiei* in the first week of February, when the plants were about eight to ten-leaf stage.The data was recorded on 1-9-rating scale.Difference between mean disease scores of cv. K-850 (susceptible control) and mean disease scores of individual mutants were calculated using *t*-test.

Results and Discussion.

A wide range of disease resistance was observed in the segregating populations. Out of total plant population of 11013, thirty mutants were found highly resistant, 23 were resistant and 87 showed tolerant reaction to *Ascochyta* blight. Maximum number of highly resistant mutants was observed in kabuli genotype Pb-1 followed by desi C44 (9), Pb2000 and CH40/91 (two mutants in each genotype). The trend of resistance was Pb-1>C44>CH40/91>Pb2000. The higher number of highly resistant mutants (20) was induced through EMS treatment followed by gamma irradiation (10). In case of M_3 morphological mutants, out of 167 mutants of four genotypes, 26 mutants were found resistant, 76 tolerant and 65 highly susceptible. By the induced mutagenesis, promising disease resistant mutants and useful morphological mutations were produced which could be used in hybridization program to transfer resistant genes into high yielding elite cultivars/ producing better recombinants. The direct release of these mutants as a variety (s) could also be explored.

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O26

SCARS MARKERS LINKED TO *ASOCHYTA RABIEI*IN CHICKPEA ($SCAE19_{336}$, $SCM02_{935}$ AND $SCY17_{590)}$: EXPRESSION STUDIES AND HOMOLOGIES WITH EST AND RELATED SEQUENCES.

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Introduction

Three SCARs markers ($SCAE19_{336}$, $SCM02_{935}$ and $SCY17_{590}$) linked to QTL_{AR2} for resistance to *Ascochyta rabiei* were located on LG4 being $SCY17_{590}$ a codominantmarker (Iruela et al. 2006). In this work the expression of these SCARs, their associations with the resistance to blight, and homologies of the SCARs sequences with EST or related sequences are presented.

Material and Methods

LinesILC3279 (resistant kabuli line) and WR315 (susceptible desi landrace) and a RIL population derived from a cross between them were used. RILs with extreme values for resistance and susceptibility to blight were chosen. RNA was isolated from plants with TRIZOL reagent (Invitrogen). First strand cDNA was generated with SuperScript III Reverse Transcriptase (Invitrogen). PCR and electrophoresis were performed as described (Iruela et al. 2006). Sequence identity searches for SCARswere analyzed at NCBI (http://www.ncbi.nlm.nih.gov) using BLASTN, BLASTN employing only EST and BLATX.

Results and Discussion.

The bands for the three SCARs amplified using cDNA or genomic DNA showed the same length. The majority of resistant RILs displayed the same genotype of the resistant parental for each marker and the same occurred with most susceptible RILs but with regards to the susceptible parental. $SCAE19_{336}$ showed significant similarity with the FE672658.1 EST of chickpea expressed in drought conditions. $SCM02_{935}$ provided homologies with pentatricopeptide (PPR) repeat-containing proteins, domains involved in RNA processing which can modify the expression of genes and hold features in common with disease resistance genes. $SCY17_{590}$ matched well with aldo/keto reductases from *Medicago truncatula* and *Arabidopsis thaliana,* these enzymes detoxify fungal toxins in plants. The homology of the three SCARs presented here was checked against ESTs from chickpea or other legumes previously reported as related with blight resistance (Cho and Muehlbauer 2004, Coram and Pang 2005, Biam et al. 2007). However, a high degree of similarity was not found. It would be interesting to perform RACE-PCR using these SCARs as anchored sequences towards identification of candidate genes for this disease resistance.

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O27

UNDERSTANDING ASCOCHYTA BLIGHT RESISTANCE IN CHICKPEA USING MOLECULAR GENETICS AND GENOMIC APPROACHES

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Introduction

Ascochyta blight, caused by *Ascochyta rabiei*(Pass.) Lab.*,* is a devastating fungal disease worldwide that hampers chickpea production. A better understanding of the genetics of host resistance will facilitate development of resistant cultivars. Hence, our research focuses on QTL cloning and gene discovery targeting a genomic region associated with ascochyta blight resistance (ABR).

Materials and Methods

Mapping population: FLIP84-92C and PI599072 and 142 $F_{7:8}$ recombinant inbred lines (RILs); **Marker development**: SNP markers were developed as described in Rajesh and Muehlbauer 2008; **Phenotyping**: Disease screening at greenhouse and at blight nursery was done using methods described by Chen et al. 2004; Tekeoglu 2002; **Mapping software**: JoinMap and Q gene were used for linkage analysis and QTL analysis respectively; **BAC sequencing**: Sequencing reactions were performed as described by Roe et al. 1996

Results and discussion

We targeted ABR- *QTL*1 for QTL cloning that accounted for 35% of variation and was identified using both interspecific and intraspecific crosses. Genetic characterization of this ABR-*QTL*1 by enriching the marker density identified tightly linked markers with increased contribution (56%) to the ABR that will have direct application in marker assisted breeding. We discovered 15 candidate genes at ABR-*QTL*1 and none of them resembled NBS-LRR type genes. The novel discovery is the presence of EIN4-receptor (Ethylene insensitive) gene at this agronomically important genomic region. Our scientific quest to understand ABR resulted in development of several tools such as SNP markers, a reference linkage map, Heterogeneous Inbred Family (HIF) lines, a BAC library, genome sequences Agrobacterium mediated transformation and TILLING mutants for the chickpea research community. Functional correlation of candidate genes with the blight resistance using TILLING mutants is underway. The work described here should increase the understanding of ABR and provide a strong foundation for advanced research in chickpea.

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Session VI
Host Resistance II
(Genetics)

O28

PATHOGENESIS-RELATED GENES AND GENETIC VARIATION IN POTENTIAL RESISTANCE GENES OF MAJOR EUROPEAN LEGUMES: THE LEGRESIST PROJECT.

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Introduction

In Europe, agronomical, economical and ecological benefits of legumes are notoriously under-exploited, since an unstable yield caused by a wide range of pathogens is not attractive for growers. Despite considerable investment and progress in the use of molecular tools for resistance breeding, any application of marker technologies fordurable resistance is still hampered by insufficient knowledge of allelic diversity in resistance genes and our only limited insight into plant-pathogen interactions. Moreover, cost-effective, modern tools for advanced resistance breeding are missing. Therefore, the ERA-PG-project LEGRESIST (LEGRESIST grant0313997D), involving a consortium of 10 leading European legume breeding centers and 2 leading technology providers, aims at i) exploiting genetic diversity of resistance genes on the level of single nucleotide polymorphisms (SNPs) for genetic mapping of all expressed resistance genes in major crop legumes, ii) understanding quantitative resistance through characterization of the interacting transcriptomes of plants and their pathogens from the genus *Ascochyta* spp. by SuperSAGE analysis and iii) mapping of expression (e)QTLs underlying quantitative resistance.

Material and Methods

SuperSAGE technology was employed to resolve the stress transcriptomes of different legume host plants infected by their corresponding *Ascochyta* pathogens(*A. rabiei, A. lentis, A. fabae*, *A. lathyri* and *A. pisi*) simultaneously during their interaction(s). A catalogue of up- and down-regulated transcripts from infected host legumes(*Cicer arietinum, Lens culinaris, Vicia faba*, *Lathyrus sativus* and *Pisum sativum*) and resulting GO categories were established, and 3'- and 5'-RACE sequences screened for SNPs and small indels.

Results and Discussion

So far, the project produced transcription profiles comprising more than 4.4 millions of SuperTag cDNA fragments from *Ascochyta*-infected legumes, detected more than 7000 pathogenesis-related genes, and identified more than 50 potential resistance genes and their allelic variants. Assignment of differentially expressed SuperTags to Gene Ontology (GO) terms revealed differential as well as similar responses of the different legumes to pathogen stress, suggesting overlapping as well as species-specific defense strategies. The project delivers polydimensional SNP-arrays for rapid and cost-efficient mapping of resistance genes, legume-biotic-stress-arrays (LBSAs), and qRT-PCR assays for eQTL mapping. Applying these modern tools, LEGRESIST will produce the most advanced expression maps for all major crop legumes.

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O29

PYRAMIDING RESISTANCE IN CHICKPEA TO *ASCOCHYTA RABIEI*.

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Introduction

Ascochyta blight caused by *Ascochyta rabiei* is a major pathogen of chickpea. Resistance is thought to be inherited in a complex and quantitative manner (Flandez-Galvez et al. 2003). The recessive nature of the resistance genes has most probably hindered the development of highly resistant and superior varieties. The QTL 4-6 on linkage group III appeared to be a critical region for chickpea resistance to *A. rabiei*, as this QTL accounted for the majority of the estimated phenotypic variation. This paper reports on studies to identify molecular markers closely associated with major QTL for Ascochyta blight resistance from a cross between Lasseter and ICC12004 and marker assisted selection of resistant progeny from a F_5 RIL population.

Materials and Methods

Linkage groups containing the six QTL regions from an intra-specific cross between ICC12004 (resistant) and Lasseter (susceptible) by Flandez-Galvez et al. (2003) were aligned with the syntenic regions from the published *Medicago truncatula* map and the chickpea integrated genetic and chromosome maps. *Medicago truncatula* BAC clones orthologous to the QTL 4-6 were identified using the SCAR marker SCY17 (GenBank accession:AY860670) sequence to search homologous sequences in NCBI non-redundant databases by BLASTn. Thirteen chickpea disease resistance related ESTs and three gDNA sequences were selected and mapped *in silico* ontothe *M. truncatula* pseudochromosomes. Six RIL lines containing QTL 1-6 were progressed to an F_5 generation using marker assisted selection. These lines were assessed for Ascochyta blight resistance in glasshouse bioassays.

Results and Discussion

Through co-linearity analyses, four new markers were added to linkage group III using 25 F_2 individuals from the cross ICC12004 × Lasseter. All four of these markers fell between and cosegregated (linked) with the original markers associated with QTL 4-6. The presence of the same associated markers in the same region among different chickpea linkage maps created from different germplasm worldwide strongly indicated conservation of this region of the genome involved in *A. rabiei* resistance. Using the chickpea integrated genetic and chromosome maps as a bridge, *M. truncatula* chromosome regions orthologous to the six chickpea QTL regions were identified. The region in *M. truncatula* pseudochromosome 1 was orthologous to the chickpea QTL 4-6. One *M. truncatula* BAC clones, AC142096.25, was identified that contained sequences homologous to the marker sequences most closely related to and flanking the chickpea QTL 4-6 region. RIL lines containing combination of markers associated with the QTL showed enhanced resistance. Three selected lines were backcrossed to Lasseter and seed of 10 BCF5:F2 families were collected for further assessment.

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PATHOTYPE SPECIFIC SEEDLING AND ADULT-PLANT RESISTANCE SOURCES TO *ASCOCHYTA RABIEI* IN CHICKPEA (*CICER ARIETINUM*L.) .

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Introduction

The average global yield of chickpea (0.8 t ha^{-1}) is less than potential yield (5t ha^{-1}) of the present day improved varieties. Diseases, pests and poor management are the major factors for this gap. Among diseases, Ascochyta blight (AB) caused by *A. rabiei* is the most destructive disease (Pande et al., 2005). It has resulted in drastic reduction in area of chickpea in north western plain zone of India, Australia and Canada. AB can be managed by cultivation of resistant varieties. Identification of stable resistant donors is a pre-requisite to develop AB resistant varieties. This paper embodies information on identification of stable sources of resistance against the prevalent Indian pathotypes of *A. rabiei*. .

Materials and method

Fourteen pathotypes of *A. rabiei* representing diverse agro-climatic areas of India and 180 genotypes of chickpea were used. Pure cultures of each isolate were mass multiplied and spore suspension was inoculated on the 12 days old seedlings of the test genotypes. The inoculated plants were incubated in a growth chamber at 20°± 1^0 C, artificial day light and 100% RH. The data were recorded using modified 1-9 scale (Nene et al., 1981), 14 days after inoculation. The field evaluations were done at Dhaulakuan during 2003-04 to 2006-07 under artificially epidemic conditions. The data were recorded on terminal DR on 1-9 scale.

Results and Discussion

Out of 180 genotypes, 60 genotypes showed differential resistance to the pathotypes. Genotypes ICCX 910028-32 ABR-BP-5PABR-BP showed resistance to 12 pathotypes. Genotypes ICCX 910028-39 ABR-BP-10PABR-BP, ICCX 910028-42 ABR-BP- 21PABR-BP and ICCX 910028-42 ABR-BP-2PABR-BP showed resistance to 11 pathotypes. Additionally, 3, 3, 7, 11, 5, 6 and 6 genotypes were resistant to 10, 9, 8, 7, 6 and 5 pathotypes respectively. These genotypes showed field resistance to AB at Dhaulakuan. Most of the resistant genotypes involved known source of resistance viz. ILC 202, ICC 1069, NEC 138-2, ILC 3279, ILC 4421, E 100 Y (M) and FLIP 87-4C in their pedigree. The genotype with high level of resistance may be directly exploited as varieties or used in breeding programme as resistant donors to develop agronomically superior AB resistant varieties. Genotypes with pathotype specific resistance may be used to pyramid AB resistance genes for different pathotypes Genotypes with differential resistance may be used to identify and monitor pathogenic variability in *A. rabiei*.

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PHENOTYPIC AND MOLECULAR CHARACTERIZATION OF CHICKPEAS FOR SOURCES OF RESISTANCE TO ASCOCHYTA BLIGHT

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Introduction

Ascochyta blight (AB), caused by *Ascochyta rabiei* is a major biotic constraint to chickpea production in the cool and humid environments. The causal organism is highly variable most likely due to the presence of sexual phase (*Didymella rabiei*).Until now four pathotypes *D. rabiei* have been documented with pathotype IV being more virulent in Syria. This nature of the pathogen therefore, demands continuous efforts to search for new and diverse sources of resistance and their deployment for the chickpea improvement. This present study was therefore designed to clarify the relationship among different sources of AB resistance, to search for new sources of resistance, and generate information that will assist in choosing parental materials for crossing to improve chickpea cultivars with desirable level of AB resistance.

Materials and Methods

A set of 15 global AB resistant and susceptible genotypes were grown in pots and leaves were collected from 3–4-week-old seedlings from each genotype for DNA extraction (Imtiaz *et al*. 2008). The genotypes were tested against four pathotypes under plastic house conditions, maintained at 18-20^0 C with 80-90% humidity. Forty six mircrosatellite markers (Winter *et al*. 1999) including those markers associated with AB resistant quantitative trait loci (QTLs), particularly on linkage group 4 were used. Additionally, 170 recombinant inbred lines (RILs) derived from a cross ('FLIP97-1065C' x 'ILC1929') were genotyped with 100 SSR markers. RILs were tested under both field and plastic house conditions against ascochyta blight using a 1-9 scale. Estimates of pair-wise-genotypic genetic similarity (GS) between individuals were calculated using Gower General Similarity Coefficient. A principal coordinate (PCO) analysis was performed on the GS matrix and the first two axes were plotted using MVSP ver3.13. Analysis of variance was done using Genstat while Map Manager QTX Version b20 QTL was used to detect QTLs associated with AB.

Results and Discussion -The resistant genotypes ILC191, ILC200, and ILC202 shared a common haplotype across SCY17, TA2, TAa72, and TA146 marker loci while ILC482, ILC605 and ILC1929 carried common alleles at these marker loci. The Pathotype III resistant genotype ICC12004 possessed similar allele at SCY17, however, carried a unique allele at TA72 marker locus. Genotypes ILC72, ILC182 and FLIP98-1065C hold alike alleles at SCY17 and TA2 marker loci. The same set of genotypes has also been evaluated against Pathotypes I, II, III and IV where none of the lines showed resistance against Pathotype IV. SSR markers TA2, TA146 and TA72 clearly differentiated the resistant and susceptible bulks derived from RIL population. QTL analyses confirm the involvement of linkage group 4 in conferring resistance to AB.

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O32

BREEDING FOR RESISTANCE TO ASCOCHYTA BLIGHT IN CHICKPEA OF INDIA: CURRENT STATUS.

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Introduction

Ascochyta blight (AB) caused by *Ascochyta rabiei* (Pass.) Lab. is a serious foliar disease of chickpea (*Cicer arietinum* L.). Its occurrence was first reported in North-West Frontier of India (now partly in Pakistan) by Butler (1911). The environmental conditions of the region are quite conducive for development of disease and thus, near to complete crop loss were observed several times. Consequently, chickpea production area was reduced drastically in the Indo-gangetic plains of India. Keeping in view of seriousness of the disease, breeding for resistance was initiated to provide the most reliable and practical solution to chickpea cultivation. The detail account of varietal and improved resistant genetic stocks development is presented.

Materials and methods

Breeding for resistance to AB was taken up with the introduction of genetic stock F_8 from France (Luthra *et al.* 1938). Resistance derived from F_8 was used first to develop C 12/34, then C 235 and G 543 with variable base parent through hybridization in 1940, 1960 and 1977, respectively. In the epidemic years 1981 to 1983, these cultivars were completely damaged which led to 100% crop loss. Massive screening and breeding programmes for AB resistance were initiated in 1981 at national level. These programmes resulted in identification of stable genetic stocks, efficient screening techniques and development of resistant cultivars at different Institutes in the region. Simultaneously, stable genetic stocks namely E 100 Y, E 100 Y(m), P 1528-1-1, GG 578, NEC 206, ICC 76, F 8, ICC 1069 and Negro from germplasm lines were identified. These stocks were used extensively in hybridization programme to develop new resistant cultivars, improved resistant genetic stocks and other breeding material.

Results and discussion

The resistant cultivars C 12/34, C 235 and G 543 were developed using resistance derived from F_8 and in genetic base of Pb 7, IP 58 and C 168, respectively. Among them, cultivar C 235 had wide adaptability besides having high yield potential and resistance to ascochyta blight. All the three cultivars lost their resistance in a short period after their release as their genetic base of resistance was same and pathogen become more virulent and variable over the time. That probably led to epidemics of disease in the region. Thereafter, intensive breeding programme resulted in development of diverse new cultivars H 75-35, BG 261 and GNG 146 in 1985. Two highly resistant cultivars PBG 1 and PBG 5 (Sandhu *et al*. 2004) were developed at Punjab Agricultural University, Ludhiana in 1989 and 2004, respectively. These cultivars are more diverse in resistance. As many as 14 improved genetic stocks were developed by combining resistance from 2-4 genetic stocks and these stocks have maintained high degree of resistance for the last 10-15 years under simulated epidemic of disease. Large number of other improved lines was also developed and possessed multiple disease resistance.

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O33

BIOCHEMICAL AND MOLECULAR REACH FOR DISEASE RESISTANCE TO CHICKPEA BLIGHT CAUSED BY *ASCOCHYTA RABIEI* (PASS.) LABR.

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Introduction

Immunity to blight disease has not been reported in commercial cultivars but in some wild species of chickpea. *Ascochyta rabiei* has been identified to produce toxins involved in blight disease (Alam et.al.1989) of chickpea. Uptill now many blight QTLs have been tagged with molecular markers and mapped on the chickpea linkage groups 2, 4 & 6 (Tekeoglu *et al*., 2002; Rakshit *etal*., 2003; Udupa & Baum 2003). Marker assisted selection (MAS) for blight resistance would greatly accelerate the development of new chickpea cultivars at NIAB.

Materials Methods

Culture filtrates of *A. rabiei* were passed through ODS silica and the adsorbed compound was eluted into Acetronitrile to calculate concentration by taking absorbance. Isolated cells of chickpea varieties were incubated in two fold dilutions of toxins in holding buffer and dead cells were counted under microscope by staining it with phensafranine. LD_{50} values were extracted from graphs of probit percent cell death plotted against $\log_2$ dilution factor. Chickpea seedlings were cut from collar region and the stem was dipped into purified Solanapyrone (70µl/ml) in water in test tubes. Molecular markers used to detect resistant loci in twenty one mutants/local genotypes. These genotypes have been screened in *Ascochyta* blight screening nursery for three years at NIAB. Nine RAPD decamer primers, one ISSR sequence primers (prefix UBC), three SCAR primers and six STMS markers were selected for the analysis of these genotypes as they had been associated with QTLs for blight resistance previously reported by several authors.

Results and Discussion

Chickpea lines CM611/03, CH30/03, CH 23/04, CH83/04, and CH98/04 were found resistant to blight. The highly virulent isolate (AB-6) was found to synthesize more solanapyrone A (32.57µl) and Solanapyrone C (28.69) as compared to least virulent isolate (A-11) producing solanapyrone A (7.82µl) and Solanapyrone C (15.32µl). Variety C-727 and 6153 were highly sensitive to toxins and were also susceptible to disease as compared to CM72 and CM88. Isolated cells of susceptible variety C-727 was highly sensitive to toxins as compared to resistant cultivar. QTL located on Linkage group 4b saturated with SCAR, RAPD & STMS markers is also found to be involved in conferring resistance against blight in local genotypes of chickpea. The result achieved will further be continued to establish these findings for strengthening the breeding program in development of chickpea resistant cultivar(s).

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O34

GENETICS OF RESISTANCE TO ASCOCHYTA BLIGHT IN CHICKPEA.

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Introduction

Ascochyta blight (*Ascochyta rabiei* (Pass.) Lab.) is a major production constraint in most of the chickpea (*Cicer arietinum* L.) growing areas world wide. In India, the environmental conditions of north India are quite conducive for the development of disease and 100% crop losses have been noticed in the epidemic year 1981-83 (Singh *et al*. 1984). It is imperative to develop the resistant cultivars and as well as to understand the genetics of resistance to the pathogen.

Materials and methods

Two susceptible parents GL 769, C 214 and four resistant lines GG 1267, GL 90168, GL 96010 and GL 98010 were used to develop F_1s and F_2s of 15 crosses. Some of the back crosses and F_3 generations were also developed. These generations were evaluated using field screening technique of Gurha *et al*. (2003) by inoculating with mixture of ten prevalent isolates of pathogen. Individual plants were scored for disease reaction on 1-9 scale (Singh and Sharma, 1998). The plants with disease rating ≤ 5 were considered as resistant and above 5 as susceptible. The F_3 progenies were scored as segregating, true breeding resistant and susceptible. Chi-square ($\chi 2$) test was applied to fit the appropriate genetic ratio for the estimation of number of gene (s) governing resistance and also to find out allelic relationship among resistance genes.

Results and discussion

Genetics studies showed digenic recessive control of resistance in the cross GL 769 x C 214 whereas monogenic recessive control of resistance was found in the crosses GL 769 x GL 98010 and C 214 x GL 98010. Digenic dominant and recessive control of resistance was found in the crosses GL 769 x GG 1267 and C 214 x GG 1267 while the crosses GL 769 x GL 90168 and C 214 x GL 96010 showed the monogenic dominant control of resistance. Trigenic dominant and recessive control of resistance was observed in the crosses GL 769 x GL 96010 and C 214 x GL 90168. Allelic relationship studies showed that three resistant parents *viz.,* GG 1267, GL 96010 and GL 90168 possessed allelic single dominant gene for resistance. Besides, GG 1267 possessed two minor recessive genes which were allelic to the minor recessive gene of GL 90168 and other with GL 96010. The resistant parents GL 90168 and GL 96010 possessed non-allelic minor gene for resistance. The resistant parent GL 98010 possessed two minor recessive genes for resistance which were allelic to respective single recessive gene for resistance possessed by the susceptible parents GL 769 and C 214. The susceptible parents GL 769 and C 214 also possessed single independent inhibitory dominant susceptibility gene.

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Session VII
Epidemiology

O35

Highlights of 15 years of research on ascochyta blight on pea in France: Epidemiology and impact of the disease on yield and yield components.

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Ascochyta blight is a serious disease affecting field peas and since the early '90s, we have conducted studies on the pathosystem *Pisum sativum*/*Mycosphaerella pinodes*. The plant, the pathogen and the environment were considered as essential to understand the epidemic development of the disease and the impact of the disease on plant functioning and yield.

Epidemiology Schoeny *et al.* (2007) developed a model to predict ascochyta blight onset in field peas based on calculations of weather-dependent daily infection values. Moreover, Moussart *et al.* (1998) had shown the role of seed infection in disease transmission. We have established the epidemiological conditions for disease epidemiology development (Roger *et al.*, 1999a; 1999b; Roger and Tivoli, 1996; Tivoli and Banniza, 2007). Disease severity on pods and stems was substantially reduced in a pea-cereal intercrop treatment compared to the pure pea crop treatment when the epidemic was moderate to severe (Schoeny *et al.*, 2009). Disease development depends on characteristics of the cultivars (Onfroy *et al.*, 1999, 2007). Moreover, Le May *et al.* (2009a, 2009b) compared the reaction of several winter and spring pea types. Schoeny *et al.* (2008) had investigated the effect of canopy architecture on splash dispersal of the asexual spores of the fungus in controlled conditions, using a rainfall simulator. All this knowledge in epidemiology was used in several programs of genetics for ascochyta blight resistance on pea (Prioul *et al.*, 2003; Tivoli *et al.*, 2006a; Onfroy *et al.*, 2007) or on the model plant *Medicago truncatula* (Tivoli *et al.*, 2006b; Moussart *et al.*, 2007).

Overall effects of ascochyta blight on yield and yield components The effect of the disease on yield and yield components was investigated by Tivoli et al. (1996). Moreover, disease affected the photosynthetic activity (Garry et al., 1998a) and the remobilisation of carbon and nitrogen (Garry et al., 1996; Garry et al., 1998b; Béasse et al., 1999); the number of seeds per stem and mean seed weight were significantly decreased.

Effect of plant growth stage and plant organs infected on yield The impact of the disease on yield is not fixed and depends on growth stage at the onset of disease (Garry et al., 1996) and the location of the disease on the plant (Béasse et al., 1999, 2000). Using and building upon a disease-coupled crop growth model published by Béasse et al. (2000), Le May et al. (2005) developed an improved model to predict the impact of ascochyta blight in pea on yield components by incorporating a combination of disease progression in the canopy (number of nodes affected by the disease) and the structure of the canopy (leaf area index profile).

The approach which takes account of disease severity, plant growth stage, the risks of epidemic development, and the impact of the disease on yield losses, should lead to a better characterization of cultivars in terms of the role of their architecture in the development of disease epidemics.

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O36

SPATIAL DISTRIBUTION OF *DIDYMELLA PINODES* PETRAK AND *ASCOCHYTA PINODELLA* L.K. JONES ON AUSTRIAN WINTER PEA PLANTS.

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Introduction

Although little empirical evidence exists it has been reported that the distribution of Ascochyta blight disease symptoms on pea plants can often be used to discern between *D. pinodes* and *A. pinodella* (Bretag et al. 2006; Kraft and Pfleger 2001). The purpose of this study was twofold i) to develop and demonstrate a rapid PCR based assay to assist in the identification of *Ascochyta* spp. of the Ascochyta complex of pea, and ii) investigate the spatial distribution of *D. pinodes* and *A. pinodella* on pea plants collected from the field.

Materials and Methods

A severe outbreak of Ascochyta blight on Austrian Winter peas (*Pisum sativum* L.) was detected in northern Idaho, USA. Whole plant samples were collected randomly from the field on May 26 2007. Spatial sampling of *Ascochyta* spp. isolates from disease lesions from ten plants was performed in order to characterize the position of each isolate collected from the plant. The plants were divided by branch, node, stipule and tendril. Plant parts were coded for reference and isolations were performed on symptomatic plant parts. Plant parts were surface sterilized and single spore isolates were derived following standard procedures. Isolates were identified with a rapid PCR assay designed on the intergenic spacer region of ribosomal RNA gene cluster (Chilvers and Peever, unpublished).

Results and Discussion

The molecular assay expedited the identification of 151 isolates from 10 plants. 61 isolates (40%) were identified as *A. pinodella* and 90 isolates (60%) as *D. pinodes*. Accuracy of the molecular assay was confirmed by identifying a subset of isolates by cultural and conidial morphology after Onfroy et al. (1999). All plants were infected with both species of Ascochyta. *A. pinodella* and *D. pinodes* were found to be randomly distributed over the diseased plant parts.

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RESPONSE OF FIELD PEA VARIETIES TO THE FUNGAL COMPONENTS OF THE ASCOCHYTA COMPLEX.

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Introduction

Ascochyta blight of field pea (*Pisum sativum*) is caused by a complex of four fungal species: *Ascochyta pisi*, *Mycosphaerella pinodes*, *Phoma medicaginis* var. *pinodella* and *Phomakoolunga* sp. nov. (Davidson *et al*., 2009). To date, studies on resistance to the ascochyta blight complex in pea have described it as a partial resistance that is difficult to evaluate in the field. A controlled environment screening method is, therefore, a practical solution. A method using drop spore suspensions on detached leaves has been used previously to identify partial field pea resistance sources and to study the epidemiological components of resistance in field pea (Onfroy *et al*., 2007).

Within this current study a detached leaf assay was used to assess how field pea varieties respond to the fungal species found in the Australian ascochyta blight complex. This information will then be used to identify varieties to be used as controls for future screening of Australian field pea breeding material for resistance to each component of the ascochyta blight complex.

Materials and Methods

A set of thirty one field pea varieties with putative resistance to ascochyta blight, and of various origins, was used to identify potential controls to the individual fungal components of the ascochyta complex. Six different isolates were used; three single isolates (*Mycosphaerella pinodes*, *Phoma medicaginis* var. *pinodella* and *Phomakoolunga* sp. nov.) and three ascochyta blight complexes collected from Australian field pea crops. Plants were grown in a glasshouse until the 5 to 6 leaf stage, with 4 replicates of each variety. Using a method developed from Onfroy *et al*. (2007), detached pea leaves on 0.5% water agar were inoculated with a 10 µl droplet of a 5×10^4 spores/ml spore suspension. The detached leaves were assessed each day after inoculation using a 0-3 scale as described by Onfroy *et al.* (2007). Once lesions began extending beyond the borders of the droplet the lesion diameter was measured daily until the lesions grew beyond the leaf edges and onto the agar.

Results and Discussion

Significant interactions between the varieties and fungal isolates were observed for both disease appearance and disease extension. This work has identified field pea varieties that will act as useful controls for the screening of breeding lines against ascochyta blight. However, different standard control varieties will be required for each separate fungal species due to the different response of varieties to the fungal species. Because of the small space required this method will allow for screening of a large number of field pea varieties or fungal isolates. Whilst acting as a methodology for screening breeding material, it can also give an insight into the components of resistance of field pea to specific species of the ascochyta blight complex.

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O38

OPTIMIZING ASCOCHYTA BLIGHT MANAGEMENT IN CHICKPEA ON THE CANADIAN PRAIRIES.

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Introduction

Despite improvements in disease resistance in chickpea (*Cicer arietinum*) cultivars adapted to our semi-arid climate and short growing season, ascochyta blight (*Didymella rabiei,* anamorph *Ascochyta rabiei*) continues to be a serious issue. Fungicides are an important tool in an integrated management strategy, and our research has focused on optimizing fungicide use. Western Canadian producers tend to use lower than recommended carrier volumes, due to water supply issues and time constraints. In response to this situation, studies were conducted to examine the effect of carrier volume on fungicide efficacy in the field, and on spray deposition and penetration in the laboratory. The potential for spray quality to influence fungicide coverage, and in turn, fungicide efficacy, was also investigated in field and laboratory trials. Fungicide application timing and product sequences have been studied previously, and ongoing field research is being conducted in this area using cultivars with improved resistance.

Materials and Methods

Field studies on the effect of carrier volume on ascochyta blight were conducted using three carrier volumes: 100, 200 and 300 L ha^{-1}. The impact of spray quality on ascochyta blight was also investigated in field trials using three nozzle types. Spray retention and canopy penetration were studied in two concurrent indoor trials, one comparing the three carrier volumes and the other comparing the three nozzle types. Simulated chickpea canopies of cvs. Myles and Sanford were sprayed with fluorescent tracer dye using an indoor track sprayer. Various fungicide product sequences and application timings have been assessed in two ongoing field trials to determine the impact of these factors on ascochyta blight management using improved cultivars (cvs. CDC Frontier, CDC Luna, and CDC Vanguard).

Results and Discussion

Under low to moderate disease pressure, increasing carrier volume for fungicide applications was not critical for ascochyta blight control. When disease pressure was high (>49%), however, higher carrier volumes significantly reduced ascochyta blight development in seven out of nine trials. The use of higher carrier volumes increased the penetration of spray into the fern-type canopy of cv. Myles in laboratory studies, but had a less pronounced effect on penetration of the unifoliate canopy of cv. Sanford. Nozzle type had no effect on disease development or yield in all seven site-years, and had no effect on spray coverage or spray penetration. Results of application timing trials are preliminary, but seem to support prior work in which the importance of early application(s) were demonstrated. Preliminary results of fungicide sequencing trials suggest that sequences of a pyraclostrobin/boscalid mix and prothioconazole or chlorothalonil provide effective control.

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O39

EFFECT OF GROWTH STAGES OF CHICKPEA ON THE GENETIC RESISTANCE OF ASCOCHYTA BLIGHT

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Introduction

Ascochyta blight (AB, Ascochyta *rabiei* (Pass.) Lab.) is one of the most important foliar disease of chickpea (*Cicer arietinum* L.), globally (Pande et al. 2005). Chickpea is attacked by AB at any growth stage in cool and humid weather depending on the inoculum availability. However, the disease epidemics are most prominent during the flowering and podding growth stages. Higher susceptibility of chickpea to AB at reproductive growth stage may be due to senescing of the plant tissue and or to favorable environmental conditions. The main objective of this study was to determine the effect of growth stages of chickpea on the genetic resistance of AB and use this information in resistance breeding program.

Materials and Methods

Following staggered intervals, two susceptible (ICC 4991 and ICCV 10) and two moderately resistant (ICCV 05562 and ICCV 04512) chickpea cultivars to AB were sown in pots in the green house. Plants in seedling (GS1), post-seedling (GS2), vegetative (GS3), flowering (GS4) and podding (GS5) growth stages were spray inoculated with *A. rabiei* conidial suspension (5 x 10^4 conidia/ml). The experiment was arranged in a randomized complete block design with four replications in controlled environment facility at ICRISAT, Patancheru, India. Four pots (five-seedlings/pot) constitute a replication. Data on incubation period (IP) in days after inoculation and disease severity on 1-9 rating scale was recorded.

Results and Discussion

Irrespective of crop cultivars the IP was shorter in GS1, GS4 and GS5 and significantly extended than IP of GS2 and GS3. This is attributed to the development gene expression, as resistance genes reported to be highly expressive during the vegetative growth stages than at maturity (Trapero-Casas and Kaiser 1992). However, symptom development was delayed by 2-3 days in moderately resistant cultivars. The AB severity 10 days after inoculation ranged between 7 and 9 in susceptible cultivars and 3 and 5 in moderately resistant cultivars. Further the correlation coefficient between GS1, GS4 and GS5 was highly significant (r = 0.95) indicating that, evaluation for resistance to AB can be done at GS 1 (10 days old seedling stage), and or GS4 (flowering stage) to GS5 (podding stage) growth stages of chickpea. This supports the evaluation for AB resistance using 10-day-old-seedlings in controlled environment at ICRISAT and adult plant field screening at hot-spot locations in Dhaulakuan and Ludhiana in India (Pande et al. 2009 unpublished).

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O40

DEVELOPMENT OF SCREENING TECHNIQUES AND IDENTIFICATION OF NEW SOURCES OF RESISTANCE TO ASCOCHYTA BLIGHT DISEASE OF CHICKPEA.

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Introduction

Ascochyta blight (AB), caused by *Ascochyta rabiei* (Pass) Lab. (anamorph), is a devasting disease of chickpea (*Cicer arietinum* L.) in areas where cool and humid weather prevails during the cropping season. Fungicidal management of AB is unsustainable and hazardous to environment. Host plant resistance (HPR) is most effective either alone or as a major component of integrated AB management. The preliminary step for exploiting HPR is the development of reliable and repeatable techniques for large-scale screening of germplasm. A number of such techniques under field and green house conditions have been developed (Sharma et al. 1995; Bretag and Meredith 2002; Pande et al. 2005). The objective of this study was to develop better screening techniques and/or improve the existing techniques developed earlier, and examine correlation between these techniques. Sources of stable host resistance were also identified.

Materials and Methods

Components of resistance screening techniques using detached - leaf (cut-twig) and whole plants (10-day-old seedlings) were standardized and a controlled environment facility (CEF) developed to facilitate reliable large scale screening for AB resistance. Similarly, components of field screening (FS) were standardized at Dhaulakuan and Ludhiana in India, where environmental conditions were found to be more favorable for AB development. A large number of breeding (F_2 to F_7) lines were screened. Correlation coefficient between disease severity rating in CEF and FS was calculated. Stable sources of AB resistance in breeding lines with good agronomic background were identified

Results and Discussion

The results of the CEF using cut-twig (detached-leaf) were found to be highly correlated with the 10-day-old seedlings ($r = 0.94$) and FS ($r = 0.88$). Similarly results between 10-day-old seedlings and FS were also highly correlated ($r = 0.89$). The detached-leaf method is quick and reliable, and thus useful in screening segregating breeding lines and wild *Cicer* species. The remaining plants can then be used to screen for other target traits and seed production. Using these techniques high levels of stable resistance in new breeding lines were identified.

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Poster Session

P01

CLONOSTACHYS IN CHICKPEA DEBRIS IN THE PALOUSE REGION OF THE PACIFIC NORTHWEST, USA.

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Introduction

There are few examples of biocontrol of Ascochyta blight of chickpea (causal agent *Didymella rabiei*, anam. = *Ascochyta rabiei*). Isolates identified by morphology as *Clonostachys rosea* have been used to suppress *D. rabiei* as well as *Botrytis cinerea*, agent of gray mold in chickpea (Burgess et al. 1997; Dugan et al. 2005). *C. rosea* has an extensive history in biocontrol, and *C. rhizophaga* and other *Clonostachys* species have also been used for experimental biocontrol (e.g., Garćia et al. 2003), but *C. rhizophaga* is recently documented as inducing wilt in chickpea (Abang et al. 2009). When chickpea debris was plated to growth media, isolates then assigned to *C. rosea* comprised 0-2% of isolates (Dugan et al. 2005). We resolved to assess recovery of *Clonostachys* with an alternative sampling protocol, to identify isolates on the basis of sequence analysis, and to test our isolates for pathogenicity to chickpea.

Materials and Methods

In 2008, post-harvest chickpea stems were collected and placed into 30 incubation chambers for recovery of fungi. We followed Schroers (2001) and Schroers et al. (1999) for morphological identification, and Schroers (2001) for identification on the basis of beta-tubulin sequences, for isolates CP98B (Dugan et al. 2005) and CP08C6 (2008 isolate). Pathogenicity trials were conducted with these isolates and chickpea line ICC 12004 following Burgess et al. (1997).

Results and Discussion

Clonostachys was recovered from 10 of 30 chambers. Isolates identified as *C. rosea* on the basis of conidial L/W ratios, secondary conidiophore frequency and morphology, and color of conidial masses, were identified as *C. rhizophaga* on the basis of beta-tubulin sequences, which for both isolates had 93% similarity with the type (CBS 710.86) of *C. rosea*, and 99% with the type (CBS 202.37) of *C. rhizophaga*. No plants wilted, but emergence in treatments was usually significantly less than in controls. Schroers (2001) and Schroers et al. (1999) provided descriptions and illustrations whereby *C. rosea* and *C. rhizophaga* could be distinguished, but Schroers (2001) refrained from incorporating these distinctions into his key. Assuming species assignment on the basis of beta-tubulin sequence, our results indicate that variation in morphological and colony characters of *C. rhizophaga* is greater than heretofore described, and confirm the validity of Schroers' (2001) decision to refrain from separating the two species in his morphological key.

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P02

ISOLATE VARIABILITY AND RESISTANCE TO *ASCOCHYTA FABAE* IN SOUTHERN AUSTRALIA.

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Introduction

Didymella fabae and its anamorph (*A. fabae*) are both present in Australia, increasing the likelihood of genetic variability in the population (Kaiser *et al*., 1997). The evolution of virulence compromises durable resistance and so it is important to monitor changes in isolate pathogenicity to *V. faba* genotypes. This study examined diversity in *A. fabae* isolates using molecular and phenotypic screening techniques and defined the genetics of resistance in two faba bean accessions frequently used as parents in the Australian breeding program.

Materials and Methods

Forty isolates of *A. fabae* from southern Australia were examined *viz.* 9 from long-term culture storage (1999-2002), 18 collected from commercial crops in 2001-2002, and 13 collected from a 150 m field transect of one cultivar in 2001. DNA was extracted from each isolate and AFLP analysis performed, amplified with six PCR primer combinations. The data was analysed to assess chronological or geographical influence on variation. Seven isolates, representing different phylogenic groups, were selected for pathogenicity testing on six *V. faba* genotypes in a glasshouse trial. Plants were artificially inoculated with each isolate, maintained in high humidity, and assessed 21 days later. In a plant genetics study, University of Adelaide resistant accessions 622 and 970 were each used as male homozygous resistant parent crossed with susceptible accession 969-3. Parents, F_1, F_2 and F_3 progeny were artificially inoculated with one isolate of *A. fabae* (median isolate in pathogenicity test) in a screen-house trial and the ratio of resistant to susceptible plants evaluated 28 days later.

Results and Discussion

The AFLP analysis and phenogram found significant variation amongst the 40 isolates of *A. fabae.* Variation was not attributed to collection date, geographical origin or *V. faba* genotypes from which the pathogen was cultured. There was as much variation observed amongst 13 isolates collected from one field as was observed amongst 27 isolates collected across southern Australia over 10 years. The pathogenicity studies on seven isolates showed no ($P>0.05$) differentiation into races or pathotypes. This contrasts studies by Kophina *et al.* (1999) on Australian *A. fabae* populations, where 2 isolates were in common with this study. Segregation patterns in F_3 progeny from 969-3*970 found resistance was controlled by one dominant gene, while F_2 and F_3 progeny of 969-3*622 found inheritance was controlled by 2 or possibly 3 recessive genes, previously considered a single incomplete dominant or recessive gene (Ramsey *et al*., 1995). Sustainable management of resistance to ascochyta blight in faba beans will require constant monitoring of pathogen variability and development of lines with distinct sources of durable resistance.

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P03

The role of sexual reproduction in increasing the virulence of *Didymella rabiei* on chickpea cultivars

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Introduction

Ascochyta blight (*Didymella rabiei*) is one of the most important biotic factors affecting the production and quality of chickpea. Many of the released chickpea cultivars or farmer land races have been reported to be susceptible to Ascochyta blight in different countries, due to changes in the pathogen population structure, caused mainly by sexual reproduction and gene flow through infected seeds. The two mating types of *D. rabiei* are reported from different countries including Syria, and there are reports that ascospores play a role in initiating primary disease foci, as well as shaping the population structure in the pathogen. The *D. rabiei* population in Syria develops highly virulent populations that attack the existing sources of resistance in ICARDA's breeding materials (Udupa *et al*. 1998). This study was designed to investigate the role of sexual reproduction in increasing pathogen virulence on chickpea genotypes, and the association of mating type with different virulence groups of the pathogen.

Materials and methods

In 2007/08, the virulence spectrum of 88 single spore isolates from ascospore infected plants were studied in five separate experiments, using five genotypes with varying levels of resistance to Ascochyta blight (Ghab-1, Ghab-2, ICC-12004, ICC-3996 and ILC-1929). The mating types of 33 isolates were also determined using the primers Com1, SP21 and Tail 5 (Barve *et al*., 2003).

Results and discussions

Significant ($P<0.001$) differences among genotypes and isolates, as well as their interactions, were observed in four of the experiments. Some 76% of the isolates (exhibiting a rating of 5-9 based on a 1-9 rating scale) were virulent on the highly resistant small-seeded 'desi' genotypes (ICC-12004 and ICC-3996), and 10% of the isolates were weakly virulent on the susceptible genotypes ILC-1929 and Ghab-1, with ratings between 1 and 4..The mating type analysis showed that 67% of the isolates were Mating type 1 (MAT1-1) and the remaining isolates being Mating type 2 (MAT1-2). The emergence of new virulent isolates through sexual reproduction could threaten the expansion of winter chickpea production in Syria; though these results indicate that mating type is not associated with virulence of this pathogen.

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P04

IDENTIFICATION OF GENES INVOLVED IN RESISTANCE TO *MYCOSPHERELLA PINODES* IN PEA USING MICROARRAY TECHNOLOGY.

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Introduction

Mycosphaerella pinodes(Berk & Blox) Vesterg is one of the most devastating pea diseases. Genetic resistance is the most efficient, economical and ecologically sound strategy to control this disease. Highest levels of resistance to M. pinodes have been identified in wild accessions of pea (Fondevilla et al. 2005). Little is known on M. pinodes – pea interaction at molecular level. Identification of genes controlling resistance in these resistant wild peas would facilitate the introgession of these genes into cultivars. The goal of the present study was to identify genes underlying phenotypic variation in resistance to ascochyta blight in pea by using microarray technology.

Materials and methods

Plants of the resistant *Pisum sativum* subsp. *syriacum* accession P665 and the susceptible pea cultivar Messire were inoculated with *M. pinodes* by spraying a conidia suspension containing 35 x 10^4 spores/ ml. The experiment was conducted in three replicates, each containing five plants per line and time point. Sixteen, 24 and 48 hours after inoculation RNA was isolated from leaves of infected plants and transcribed into cDNA. For each time point and replicate, Cy-labelled cDNA samples from resistant and susceptible plants were mixed and hybridized to Mt16kOLI1Plus microarray. This microarray contains 16.509 different 70mer oligonucleotides from *Medicago truncatula* as well as different controls (Hohnjec et al. 2005). Resulting data were normalized and analysed using the EMMA software.

Results and discussion

Of the 16.509 sequences analysed, 348 were up or down regulated in P665 comparing to Messire in at least one time point ($M \geq 0.8$ or $M \leq -0.8$, $p \leq 0.05$). 9 % of them corresponded to genes involved in defence. Candidates showing interesting sequence similarities and expression profiles will be selected and their differential expression during *M. pinodes*- pea interaction will be validated in control and inoculated plants by Northern hybridisation.

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P05

GENETIC RESISTANCE TO *PHOMA MEDICAGINIS* IN PEA.

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Introduction

The Ascochyta blight complex affecting pea is comprised of three pathogens, *Ascochtya pisi*, *Mycosphaerella pinodes* and *Phoma medicaginis* var. *pinodella* (Kraft and Pfleger 2001). *P. medicaginis* var. *pinodella* causes a blackening on stems near the soil surface and has been referred to as black stem rot or Phoma foot rot. Yield loss can be substantial and reduced crop quality can reduce crop value to the producer. Genetic resistance to *P. medicaginis* is available in Pisum germplasm; however, resistance has been incorporated into few cultivars.

Materials and Methods

A detached leaf assay was used to identify variation for resistance to *P. medicaginis* var. *pinodella*. Thirty-five registered cultivars and breeding lines including parents of several recombinant inbred line (RIL) mapping populations were evaluated for disease development. One hundred eighty-seven RILs from PRIL12 (Shawnee/Bohatyr) were screened in replicate using the detached leaf assay. Area under the disease progress curve (AUDPC) was also calculated and used for QTL analysis. A genetic map of PRIL12 (previously developed) comprised 8 linkage groups (LG) and aligns with the consensus Pisum map (Loridon et al. 2005). QTL analysis was conducted based on lesion size 10d after inoculation and AUDPC values for 178 RILS using QTL Cartographer v. 2.5 (Wang et al. 2003).

Results and Discussion

Lesion expansion among the thirty-five germplasm lines and cultivars ranged from 2.6 to 173.1 mm^2 9d after inoculation. These results indicated that genetic resistance is present in available germplasm. Parents of PRIL12, Bohatyr and Shawnee, had a mean lesion size of 0.4 and 14.1 mm^2, respectively, in trials where individual RILs were evaluated. QTL Cartographer analysis detected one QTL on LGVI based on data for lesion size 10d post inoculation and AUDPC with LOD scores of 9.0 and 7.8, respectively. Two smaller QTL each with a LOD score of 2.2 were detected on LGIII. Two additional minor QTL were detected on LGVII with LOD scores of 1.7 and 2.1. Results from this QTL analysis require cross-validation in additional mapping populations; however, the presence of a single strong QTL indicates that resistance should be heritable and genetic gain from selection is possible.

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P06

IDENTIFYING PATHOGENICITY DETERMINANTS OF *ASCOCHYTARABIEI* VIA GENETIC COMPLEMENTATION.

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Introduction

The necrotrophic pathogen *Ascochyta rabiei* causes chickpea ascochyta blight, an economically important disease worldwide. Despite extensive investigations into the biology, epidemiology of the disease, very little is known about the molecular mechanisms of the pathogen. The objective of this research is to identify pathogenicity determinants of *A. rabiei* using complementation tests.

Materials and Methods

The mutant strain ArW519 was non-pathogenic on chickpea generated from wild-type strain AR628 as a result of a single T-DNA insertion event (White and Chen, 2007). Genomic DNA flanking the T-DNA insertion was used as a probe to isolate genomic DNA clones from a phage library of the strain AR628 genome (White and Chen 2006). These genomic fragments were moved separately into a T-DNA shuttle vector with geneticin resistance and re-transformed into the ArW519 genome. The T-DNA insertion was verified by resistance to both hygromycin and geneticin and by PCR. Both double recombination events, resulting in replacement of the original T-DNA with new T-DNA as well as novel integration sites of the new T-DNA were recovered. The pathogenicity of eight complemented ArW519 mutants recovered from independent T-DNA integration events were compared to that of the parent ArW519 mutant and of wild-type AR628 on chickpea cultivars Spanish White and Dwelley using a minidome bioassay (Chen el al., 2005).

Results and Discussion

Six clones were isolated from the AR628 library using the ArW519 probe, ranging from 4016 bp to 5529 bp. Each clone was independently re-introduced into the ArW519 genome and tested for the restoration of pathogenicity. Only one clone was able to functionally restore pathogenicity to the ArW519 mutant. For the re-integration of each region, the identical T-DNA molecule, conferring resistance to geneticin, was utilized. For each re-integration, nearly three quarters of the recovered geneticin-resistant *A. rabiei* transformants were no longer resistant to hygromycin, suggesting that the newly introduced T-DNA region had simply replaced the original T-DNA integrant. DNA isolated from transformants that were resistant to both hygromycin and geneticin was used as template for primers specific for each of the antibiotic cassettes to verify that both genes were intact. The genomic fragment that restores pathogenicity to the ArW519 mutant contains about 3000 bp of DNA upstream of the T-DNA insertion and about 1000 bp of DNA downstream. When compared to sequence databases this *A. rabiei* genomic fragment carries regions that are similar to retrotransposon Molly from *Stagonospora nodorum* and the AvrLM1 avirulence gene from *Leptosphaeria maculans*. The ability to successfully complement non-pathogenic *A. rabiei* mutants is an important step to better understand the underlying pathogenic mechanisms of the pathogen.

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P07

Role of grain legumes as alternative hosts on the fitness of *Mycosphaerella pinodes* and *Phoma medicaginis* var. *pinodella* C. Le May, INRA, AGROCAMPUS Ouest, Laboratoire Ecologie et Sciences Phytosanitaires, 65 rue de Saint Brieuc, 35042 Rennes, France (lemay@agrocampus-ouest.fr)

Introduction

Many studies have been undertaken to know how the fungi responsible for plant diseases can survive year after year. Fungi display different strategies to survive and colonise the crops. These strategies seem to have different importance, depending on the fungi species, cropping practices and climatic factors. A passage through alternative hosts can modify the pathogenic fitness of fungi (Abbo *et al*., 2007; Akinsamni *et al*., 2007). The knowledge of ecology of alternative hosts and the way they affect the pathogen fitness would be precious to allow a management over many years and on a larger scale than the field itself. Ascochyta blight is a disease complex involving two main separate pathogens, *Mycosphaerella pinodes* and *Phoma medicaginis* var *pinodella* (Bretag *et al*., 2006). Four main primary sources of inoculum have been described: seeds, stubble, soil and volunteer plants (Tivoli and Banniza, 2007). The purpose of this study is to evaluate the evolution of the fitness components of *M. pinodes* and *P. pinodella* after being grown for four generations on a pea cultivar or on an alternative host.

Materials and Methods

Plants (pea cultivar: Baccara, alternative hosts: 2 cultivars of common vetch: Bingo and Pepite; 1 cultivar of horse bean: Diana) were maintained in growth chambers at 18-20°C and 12h photoperiod for three weeks before the inoculation of the two fungi. Seven days after the inoculation, the fungi were isolated. Necrosis area was measured to estimate the aggressiveness of these isolates on pea plants by using the ASSESS software (7 days after inoculation), and the production of pycnidiospores was estimated with a Malassez cell (10 days after inoculation) (Schoeny *et al.,* 2008).

Results and Discussion

The results showed differences in the effect of the passage according to the alternative host-plant and the fungi. *M. pinodes* and *P. pinodella* displayed an opposite behaviour. *P. pinodella* isolates cultivated on Bingo and Pepite cultivars were more aggressive on pea than the control (70, 65 and 14% of necrosis area respectively), while *M. pinodes* isolates cultivated on identical cultivars were less aggressive than the control (28, 59, and 95% of necrosis area respectively). Concerning the reproductive fitness, no difference was observed between the control isolates of the two fungi and the other isolates. For *M. pinodes*, spore production ranged between 4.400 and 10.000 spores. For *P pinodella*, spore production ranged between 6.000 and 12.000 spores. If the effect of the fungal aggressiveness and the reproductive behaviour due to the passage through a host-plant is a reduction, this would allow a better control of the disease by managing these alternative hosts. This study showed that some behaviour modifications could occur between pathogen agents and their hosts. The possible application of such studies could help to estimate the risk of cropping pea according to cultivated and wild potential host-plant.

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P08

PLANT CANOPY MODIFICATIONS AND ASCOCHYTA BLIGHT CONTROL IN CHICKPEA.

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Introduction

The area seeded to chickpea (*Cicer arietinum* L) crops reached a high of 420,000 ha in Saskatchewan in 2001, but the area has since declined substantially and dropped to <50,000 ha in 2007, mainly due to ascochyta blight. In susceptible cultivars, yield loss due to the disease was up to 100%. Cultivars currently available are only partially resistant (Chandirasekaran et al. 2009), and thus foliar fungicides were used heavily for ascochyta blight control. It is believed that severity of ascochyta blight (ABS) in chickpea can be minimized with improved cultural practices. The objectives of this study were to determine (i) the effect of varying planting patterns on ascochyta blight severity and seed yield of chickpea, and (ii) reduction of foliar fungicides and plant density on the maintenance of ascochyta control and yield.

Materials and Methods

Field experiments were conducted in southwest Saskatchewan, 2004-2005. The cultivars Amit and CDC Xena were tested under solid- and paired-row planting patterns under two plant densities (44 vs 31 plants m^{-2}) and four fungicide intensities (1X and 0.67X rates, each at 1 and 4 applications). For each cultivar, the eight treatments were arranged in an incomplete factorial, randomized complete block design with four replicates. Ascochyta blight rating on leaves and stems was carried out using the Horsfall-Barratt scale (0-11) (Horsfall and Barratt 1945). Disease rating was initiated after the first appearance of disease symptoms, and the consecutive ratings were carried out at 15-d intervals.

Results and Discussion

Overall, CDC Xena exhibited greater responses to applied treatments than Amit. Chickpea receiving four fungicide applications always had significantly lower ABS than chickpea with one application. In 2004, CDC Xena receiving four fungicide applications yielded 1054 kg ha^{-1}, whereas the yield was <40 kg ha^{-1} with one application. In 2005, CDC Xena at four fungicide applications yielded 1074 kg ha^{-1}, compared to 332 kg ha^{-1} at one application. Paired-row planting patterns had a lower ABS rating than solid-planting in most cases, but seed yield was not affected. With the use of paired-row planting, the ABS rating was similar between 1X and 0.67X rates or between the two plant densities (44 vs 31 plants m^{-2}). With paired-row planting, fungicide use was reduced by as much as 30% (0.67X rate) and plant density was reduced from 44 to 31 plants m^{-2}; this did not decrease disease control efficacy or seed yield in chickpea. Paired-row planting allowed fungicide drops penetrated to the lower part of the plant canopy, and thus improved control efficacy. We conclude that minimizing ascochyta blight and optimizing economical return in chickpea can be achieved through the integration of genetic resistance, improved planting patterns, and fungicide applications.

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P09

SOURCES OF RESISTANCE IN WILD SPECIES OF LENTIL TO ISOLATES OF ASCOCHYTA BLIGHT (*ASCOCHYTALENTIS*).

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Introduction

Ascochyta blight caused by *Ascochyta lentis* Vassilievsky (Kaiser et al. 1997) is a serious fungal disease of lentil in Canada and worldwide. The disease can severely reduce yield and grain quality. Isolates of *A. lentis* are highly variable in their pathogenicity and virulence (Kemal and Morrall, 1995) and exploitation of resistance in the wild germplasm becomes important. The objectives of this research were to identify sources of resistance in accessions of wild species to a mixture of Canadian *A. lentis* isolates, to make interspecific crosses with the cultivated species, and to identify the hybrid progeny that carry the resistance gene(s).

Materials and Methods

Evaluation of world collection of wild species were carried out in replicated experiments both in the field and greenhouse using artificial inoculation with mixtures of isolates collected in commercial lentil fields in Saskatchewan, Canada. Resistant (R) and susceptible (S) controls were included in the experiments.

Results and Discussion

Results indicated that resistance was evident in *L. ervoides* (Brign.) Grande, *L. orientalis* (Boiss.), *L. nigricans* (M. Bieb.) Gordon, *L. lamottei* Czefr. and in cultivated control lines, but not in *L. tomentosus* L. The level of resistance in some wild accessions was higher than the resistant control, cv. 'Indianhead' in both the field and greenhouse environments. A few wild accessions previously reported to be resistant to the *A. lentis* isolates of Syrian origin were also resistant to Canadian isolates. Interspecific recombinant inbred lines (RILs) from a cross between cv. 'Eston' (S), and *L. ervoides* accession, PI 72815 (R) and LR59-81 (R) were successfully developed for genetic studies including resistance for ascochyta blight and anthracnose (Fiala et al., 2009). Deployment of resistance from different species would help manage ascochyta blight in lentil.

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P10

BREEDING FABA BEAN FOR RESISTANCE TO ASCOCHYTA BLIGHT

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Introduction

Faba bean is adversely affected by numerous fungal diseases and parasitic weeds in different regions of the world (Lopez-Bellido et al. 2005). Ascochyta blight (*Ascochyta fabae*) is one of the major diseases that affect both the quantity and quality of faba bean production in many countries (Hanounik and Robertson, 1989). The International Center for Agricultural Research in the Dry Areas (ICARDA) has the global mandate for improving the productivity of faba bean through developing resistance gene pools to Ascochyta blight that are suitable for different countries. However, the available resistant gene pools cannot fit to agro-ecological zones where ascochyta blight is a problem. Hence, this paper summarizes recent efforts to develop new sources of resistance to ascochyta blight that can be used by national agricultural research systems (NARS) in different eco-regions.

Material and Methods

Two hundred fifty genotypes obtained from Genetic Resource Section (GRS) at ICARDA were planted in two replications with two repetitive checks (Giza 4 as susceptible and Ascot as resistant checks). Those were evaluated under natural and artificial infections at Lattakia Research Station, Syria 2005. The entries were scored for ascochyta blight reactions using 1-9 rating scale where 1 is highly resistant and 9 is highly susceptible. Single resistant plants were selected with a rating scale of 3 and below. The progenies of these single plants showing resistance reaction were further evaluated for three successive generations from 2006 to 2008 under Ascochyta blight nursery.

Results and discussions

Among the tested genotypes, 18 were found resistance (scoring of 1 to 3) and 12 of them showed similar reaction with the resistant check Ascot (scoring of 1). The selected materials were originated from Spain, Ethiopia, Canada, Turkey, Netherlands, Lebanon, Morocco, Greece, Syria and Australia. All the identified genotypes showed uniformity and are being used in the faba bean breeding program as parents to incorporate resistance genes to high yielding and adapted genotypes in targeted environments

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P11

DEVELOPMENT OF THE TELEOMORPH OF *ASCOCHYTA RABIEI* ON CULTURE MEDIA.

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Introduction

Development of fertile pseudothecia of *Didymella rabiei*, the teleomorph of *Ascochyta rabiei*, requires the pairing of two compatible mating types which are referred to as MAT1-1 and MAT1-2 (Trapero-Casas and Kaiser 1992). Under field conditions, the teleomorph only develops on infested chickpea debris that overwinters on the soil surface in the presence of both mating types of the pathogen. In the laboratory, fertile pseudothecia develop on sterile chickpea stem pieces that are inoculated with a conidial suspension of two compatible isolates of the fungus and incubated on moist filter paper or in a humid atmosphere at 10 C for 40-45 d. Preliminary attempts by the authors to produce pseudothecia of *D. rabiei* on agar media failed (Trapero-Casas and Kaiser 1987).

Materials and Methods

Natural and synthetic agar media were dispensed in 9-cm-diameter plastic Petri dishes. These media included: chickpea stem powder (2.5, 5, 10, 20 and 40 g/L), chickpea stem extracts in hot water (10, 20, 40, 60 and 80 g/L), chickpea seed meal agar (with or without dextrose), carrot agar, carrot slices, corn meal agar, Czapek-Dox agar, malt extract agar, potato-carrot agar, potato dextrose agar (natural and Difco), V-8 juice agar, 2% water agar (WA), and four synthetic media containing different inorganic salts and trace elements. All media were seeded with 0.5 mL of a conidial suspension (1×10^5 spores/mL) of compatible isolates of *A. rabiei*. Sterile chickpea stems inoculated with the two compatible pairs of isolates and incubated in Petri dishes with WA were included in all tests as a control treatment. Dishes, half of which were sealed with Parafilm, were incubated at 10 C in continuous dark or light and dark (12-h photoperiod at 40$\mu E/m^2s$) for 50 d. Experiments were conducted three times.

Results and Discussion

Fertile pseudothecia developed among the pycnidia of compatible isolates of the fungus from Spain and the United States only on 2% water agar (WA) amended with powdered chickpea stems (10, 20 and 40 g/L) or hot water extracts of chickpea stems (20, 40, 60 and 80 g/L). Within 2-3 wk pseudothecia began to develop and reached maturity after 5-6 wk. Development of pseudothecia was best on WA amended with the highest concentrations of chickpea stem powder (20 and 40 g/L), followed by the highest concentration of chickpea stem extracts (80 g/L). The size and shape of pseudothecia, asci and ascospores that formed on the highest concentrations of these two media were not significantly different from those that developed on inoculated chickpea stem pieces in the control treatment, although density of pseudothecia was lower than that on chickpea stem pieces. Pseudothecia did not develop on any of the other media. Development of the teleomorph on culture media was not affected by light conditions, aeration, or compatible isolates of the pathogen. Ascospores discharged from the powdered chickpea stem medium onto young chickpea seedlings in a moist chamber were pathogenic and induced symptoms identical to those developing on plants inoculated with conidia. This is the first report of the development of mature pseudothecia of *D. rabiei* on agar media.

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P12

PRELIMINARY INVESTIGATION OF THE SECRETOME OF *ASCHOCHYTA RABIEI*.

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Introduction

Ascochyta rabiei is one of the most destructive diseases of chickpeas worldwide but information about the biochemistry of the host-pathogen interactions is very limited. Studies of proteins secreted by this pathogen have indicated the presence of enzymes involved in cell wall and membrane degradation such as pectin methyl esterase, cutinase and acid phosphatases (1,2), the presence of pathogen protection enzymes such as NADPH dependendent reductases of phytoalexins (3) and a possible repressor of phytoalexin production (4). We are investigating the secretome of *A. rabiei* when grown in modified Fries and Czapek Dox media, which induces solanapyrone production.

Materials and Methods

A. rabiei isolates, collected from North Dakota from 2005-2007, were grown in modified Fries or Czapek Dox media for 8-12 days. Culture solutions were filtered through Whatman No. 2 filters, centrifuged at 2200g, the supernatant dialyzed against distilled water and concentrated 10 fold by distillation at 37C. For 1D gels, proteins were acetone precipitated and dissolved in SDS sample buffer. The proteins were separated by SDS-PAGE using the Tris-Tricine buffer system. For 2D gels, the concentrate obtained above was dialyzed against distilled water and concentrated an additional 5 fold. Fifty to sixty g of protein was precipitated with the 2D clean up kit (GE Healthcare), dissolved in destreak rehydration solution (GE) and separated on a 7 cm 3-11 non-linear immobilized pH gradient IEF gel (GE) and focused for a total of 35000 kV-h. The proteins were then separated by SDS-PAGE as described above. Gels were silver stained with the BioRad Silver Stain Plus kit. All protein concentrations were determined using the BioRad protein assay.

Results and Discussion

Analysis of over 50 isolates by 1D gel electrophoresis showed very similar patterns indicating limited variability in the types of proteins expressed between isolates. Three isolates were chosen for further study by 2D gel electrophoresis. When grown on modified Fries media, up to 50 different protein spots were observed with 33 being found in all three isolates. When grown on Czapek Dox media there is an overall reduction in the number of protein spots. The most dramatic changes occur in the basic pH ranges where there is a reduction in the total number of spots.

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P13

ASSESSMENT OF STABILITY IN REACTION TO *MYCOSPHAERELLA PINODES* AMONG FIELD PEA GENOTYPES.

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Introduction

Mycosphareալla blight is an importantyield constrains of pea worldwide and using host resistance is the most economical means in managing the disease. No complete resistance to *M. pinodes* has been identified in peas; however good sources of partial resistance have been identified and are being used in breeding programme (Tivoli et al. 2006). In field pea, Bretag et al. (2000) found that environmental conditions are critical determining disease severity. Since the resistance to *M. pinodes* in pea is a quantitative trait, and expression of resistance issubstantially influenced by environment (Zhang & Gossen 2008) we decided to assess stability of reaction to *M. pinodes* of some partial resistant accessions in comparison with some commercial pea cultivars under field conditions with vary epidemic pressure.

Materials and Methods

Field studies were conducted in 2005-2008. Twenty one field pea genotypes, among them commercial cultivars, partially resistant accessions from USDA-ARS collection (PI 142441, PI 142442, PI 381132, PI 404241 and PI 413691) and cv. Radley were used for these tests. Peas were grown on two–row 20 cm spaced plots, 1,5 m long with 100 plants per plot and 50 cm between plot spacing with three replications. Prior to flowering plants were inoculated with *M. pinodes* ($2x10^6 ml^{-1}$).Control plots were sprayed with fungicide Bravo. Disease severity was assessed with 0-9 scale (Xue et al. 1996) where increasing scores represent higher disease severity and disease development higher in the plant canopy. The Sheffé-Calinski mixed model for genotype-environment interaction analysis was applied (Madry & Kang 2005).

Results and Discussion

Analyses of variance of cultivars across environments for disease severity revealed significant differences among cultivars, environments, and their interactions (C x E). Significant differences among environments indicate that the cultivars were exposed to and evaluated at significantly different disease levels. Cultivars were divided in two groups one with strong reaction of disease severity to changed environmental conditions classified as unstable and second stable genotypes with lack of significance of C x E interaction. Genotypes of first group showed high disease severity scores with small differences among them.Within the second groupcv.Agra and Rubin had high mean values for disease severity and nonsignificant C x E indicating that they are stable susceptible cultivars while PI142441, PI 142442, PI 381132, PI 404241, cv. Radley and Bohun had low mean values for disease severity and stable in response to *M.pinodes* infection. In seedling test and detached leaf assessment they were also the most resistant genotypes tested.

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P14

MOLECULAR DETECTION OF *ASCOCHYTA RABIEI* PAHTOTYPES IN INFECTED CHICKPEA SEEDS

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Introduction

Ascochyta blight, caused by *Ascochyta rabiei,* is one of the most damaging diseases of chickpea, and can cause total yield loss in years of severe epidemics (Saxena and Singh 1987). Effective disease management depends on rapid and precise identification of the pathogen. Four pathotypes of *A. rabiei* have been identified. Three of them (P1, P2, and P3) were identified using RAPD, and one (P4) using the Simple Sequence Repeat (SSR) primer *Ar*H05T $(CTT)_{18}$. Mating type specific markers have also been developed (Barve et al. 2003, Rhaiem et al. 2008) that can be used to differentiate between the two mating types necessary for sexual reproduction. The objective of our study was to identify markers that could be used as diagnostic tools, to identify the presence of different *A. rabiei* pathotypes in chickpea seed, as well as the presence of different mating types.

Materials and methods

Seeds of four chickpea varieties (Ghab1, Ghab2, Ghab3 and Ghab4) infected by *A. rabiei* from were collected from ICARDA's research fields in Tel Hadya, Aleppo, in 2007. Total genomic DNA of the infected seeds was extracted using a modified CTAB method. Four DNA samples of each pathotype (P1, P2, P3 and P4) were used as a positive control for A*. rabiei*. PCR was conducted as recommended by Rhaiem et al. (2008) for SSR primers, and Barve et al. (2003) for multiplex MAT-specific PCR. The amplified products of mating types were separated on 1.5% agarose gel, and the SSR products were separated by 6% polyacrylamide gel.

Results and discussion

PCR amplification with *Ar*H05T $(CTT)_{18}$ primer with the four pathotypes (positive control) produced a clear banding pattern that allowed differentiation of pathotypes. When the primer was then used with DNA from infected seed, again the four different pathotypes could be clearly identified. The multiplex MAT-specific primers produced clear amplification products that allowed differentiation between the two mating types in infected seed samples. A combination of both primer sets could be used to identify the presence of *A. rabiei* in infected seed samples. The test could be further developed to quantify the amount of *A. rabiei* DNA using quantitative PCR. The test could be used by regulatory and quarantine authorities to ensure safe and clean plant introduction into countries.

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P15

ALLELIC DIVERSITY OF USDA CHICKPEA CORE COLLECTION OF QUANTITATIVE TRAIT LOCI FOR RESISTANCE TO ASCOCHYTA BLIGHT.

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Introduction

Knowledge of the molecular genetic variation of the accessions of the core collection will be important for their efficient use in marker-assisted breeding programs. If the allelic state at a locus associated with a trait of interest, i.e. ascochyta blight resistance in chickpea, of a given accession is known, its usefulness to a breeding and research program is greatly enhanced. As part of a larger experiment to determine the genetic structure of the USDA chickpea core collection for association studies (Varshney et al. 2007), we examined the allelic diversity of the collection for nine flanking markers of ascochyta blight resistance QTLs in chickpea.

Materials and Methods

Two genotyping experiments were used to assess the allelic diversity of the USDA chickpea core collection (www.ars-grin.gov/npgs), including nine SSR markers flanking QTL associated with genetic resistance. DNA was isolated form a bulk of ten plants to represent each accession and used in both experiments. The first experiment genotyped 376 accessions using five QTL flanking markers including TA130, TA14, TA22, TA 72 and TR29 (Tar'an et al. 2007; Collard et al 2003) at ICRISAT. The second experiment, conducted in Pullman, genotyped 504 accessions with an additional six QTL flanking markers: TA80, TA146, TR20, TS12, TS45 and TS19 (Tar'an et al. 2007a, 2007b).

Results and Discussion

In the first experiment, the five SSRs revealed 125 alleles from the bulk genotyping of 376 core accessions. Two completely inbred ICRISAT accessions were used as controls, Annigeri and ICCV2. The allelic diversity discovered is encouraging from a conservation perspective, but further reveals an inherent problem in germplasm collections of landrace genetic stocks. Our mission statement includes a mandate to preserve the maximum amount of genetic diversity within and between accessions, as demonstrated by the 25 allele average discovered per SSR. But to provide the breeding and research community with a more utilitarian resource, a further step of selection and inbreeding of the core collection is required. As the result of this study, construction of a single plant descent chickpea core is underway.

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SEVERITY AND DISTRIBUTION OF *PHOMA KOOLUNGA* ON ASCOCHYTA BLIGHT-AFFECTED FIELD PEAS IN SOUTH EASTERN AUSTRALIA.

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IDENTIFICATION OF *ASCOCHYTA* AND *PHOMA* SPECIES ON CLOVER: COMPARISON WITH OTHER SPECIES FROM FABACEAE.

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TEMPERATURE ADAPTATION AND ECOLOGICAL DIVERGENCE OF THE FUNGAL PATHOGEN *DIDYMELLA RABIEI* ON SYMPATRIC WILD AND DOMESTICATED CHICKPEA.

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DID THE DEVELOPMENT OF ASCOCHYTA BLIGHT ON WINTER AND SPRING PEA (*PISUM SATIVUM*) IN FRANCE DEPEND ON THE SAME POPULATIONS OF *MYCOSPHAERELLA PINODES?*

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BREEDING FOR ASCOCHYTA RESISTANCE IN DESI CHICKPEA.

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GENETIC ENHANCEMENT OF CHICKPEA FOR ASCOCHYTA BLIGHT RESISTANCE

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CONTROL OF PARTIAL RESISTANCE TO *MYCOSPHAERELLA PINODES* IN PEA

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ENHANCEMENT OF BLACK SPOT RESISTANCE IN FIELD PEA

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A COMPARISON OF PHENOTYPIC AND MARKER-ASSISTED SELECTION FOR ASCOCHYTA BLIGHT RESISTANCE IN CHICKPEA.

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PARTIAL CLONING OF TWO POLYKETIDE SYNTHASE GENES ASSOCIATED WITH PATHOGENICITY OF *ASCOCHYTA RABIEI*.

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COMPARATIVE POPULATION STUDY OF *DIDYMELLA RABIEI* IN TURKEY AND ISRAEL

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CLONING AND CHARACTERIZATION OF ANONYMOUS REGIONS OF *ASCOCHYTA LENTIS* AND *A. FABAE* GENOMES AND SUITABILITY OF THESE REGIONS FOR PHYLOGENETIC ANALYSIS OF *ASCOCHYTA* SPECIES.

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A SYSTEM-BASED RISK ESTIMATOR OF ASCOCHYTA BLIGHT DISEASE IN SOUTH AUSTRALIA

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MANAGEMENT OF ASCOCHYTA BLIGHT OF CHICKPEA IN INDIA.

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MANAGEMENT OF ASCOCHYTA BLIGHT OF CHICKPEA IN NORTHERN NSW.

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APPLICATIONS OF SUPPRESSION SUBTRACTIVE HYBRIDIZATION (SSH) IN IDENTIFYING DIFFERENTIALLY EXPRESSED TRANSCRIPTS IN *ASCOCHYTARABIEI*.

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INDUCED MUTATIONS FOR *ASCOCHYTA* BLIGHT RESISTANCE IN CHICKPEA (*CICER ARIETINUM* L.)

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SCARS MARKERS LINKED TO *ASOCHYTA RABIEI*IN CHICKPEA (SCAE19$_{336}$, SCM02$_{935}$ AND SCY17$_{590)}$: EXPRESSION STUDIES AND HOMOLOGIES WITH EST AND RELATED SEQUENCES.

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UNDERSTANDING ASCOCHYTA BLIGHT RESISTANCE IN CHICKPEA USING MOLECULAR GENETICS AND GENOMIC APPROACHES

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PATHOGENESIS-RELATED GENES AND GENETIC VARIATION IN POTENTIAL RESISTANCE GENES OF MAJOR EUROPEAN LEGUMES: THE LEGRESIST PROJECT.

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PATHOTYPE SPECIFIC SEEDLING AND ADULT-PLANT RESISTANCE SOURCES TO *ASCOCHYTA RABIEI* IN CHICKPEA (*CICER ARIETINUM*L.) .

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PHENOTYPIC AND MOLECULAR CHARACTERIZATION OF CHICKPEAS FOR SOURCES OF RESISTANCE TO ASCOCHYTA BLIGHT

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BREEDING FOR RESISTANCE TO ASCOCHYTA BLIGHT IN CHICKPEA OF INDIA: CURRENT STATUS.

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BIOCHEMICAL AND MOLECULAR REACH FOR DISEASE RESISTANCE TO CHICKPEA BLIGHT CAUSED BY *ASCOCHYTA RABIEI* (PASS.) LABR.

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GENETICS OF RESISTANCE TO ASCOCHYTA BLIGHT IN CHICKPEA.

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HIGHLIGHTS OF 15 YEARS OF RESEARCH ON ASCOCHYTA BLIGHT ON PEA IN FRANCE: EPIDEMIOLOGY AND IMPACT OF THE DISEASE ON YIELD AND YIELD COMPONENTS.

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SPATIAL DISTRIBUTION OF *DIDYMELLA PINODES* PETRAK AND *ASCOCHYTA PINODELLA* L.K. JONES ON AUSTRIAN WINTER PEA PLANTS.

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RESPONSE OF FIELD PEA VARIETIES TO THE FUNGAL COMPONENTS OF THE ASCOCHYTA COMPLEX.

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OPTIMIZING ASCOCHYTA BLIGHT MANAGEMENT IN CHICKPEA ON THE CANADIAN PRAIRIES.

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EFFECT OF GROWTH STAGES OF CHICKPEA ON THE GENETIC RESISTANCE OF ASCOCHYTA BLIGHT

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DEVELOPMENT OF SCREENING TECHNIQUES AND IDENTIFICATION OF NEW SOURCES OF RESISTANCE TO ASCOCHYTA BLIGHT DISEASE OF CHICKPEA.

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