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., García 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.

**References**


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 phylogenetic 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\textsubscript{1}, F\textsubscript{2} and F\textsubscript{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\textsubscript{3} progeny from 969-3*970 found resistance was controlled by one dominant gene, while F\textsubscript{2} and F\textsubscript{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 ascochytia blight in faba beans will require constant monitoring of pathogen variability and development of lines with distinct sources of durable resistance.

References


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.

References
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 introgression 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 10⁴ 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 ≥ 0.8 or M ≤ -0.8, p≤ 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.

References
P05
GENETIC RESISTANCE TO PHOMA MEDICAGINIS IN PEA.
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Introduction
The Ascochyta blight complex affecting pea is comprised of three pathogens, Ascochyta 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 mm² 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 mm², 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.

References
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 ArWS19 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.

References
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; Akinsanmi *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.

References
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.

References
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.

References


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.

References
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 x 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 40μE/m²/s) 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.

References
P12
PRELIMINARY INVESTIGATION OF THE SECRETOME OF ASCOCHYTA RABIEI.
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Introduction
Ascochyta rabiei is one of the most destructive diseases of chickpeas worldwide but information about the biochemical interaction of 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 dependent 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.

References
P13
ASSESSMENT OF STABILITY IN REACTION TO MYCOSPHERELLA PINODES AMONG FIELD PEA GENOTYPES.
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Introduction
Mycosphaerella blight is an important yield 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 is substantially 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 (2×10⁶ 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 group cv. 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.

References
Madry W., Kang M. 2005. Sheffé-Calinski and Shukla models; Their interpretation and usefulness in stability and adaptation analyses. Journal of Crop Improvement. 13(1/2) 325-369
P14
MOLECULAR DETECTION OF ASCOCHYTA RABIEI PATHOTYPES 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 ArH05T (CTT)\textsubscript{18}. Mating type specific markers have also been developed (Barve et al. 2003, Rhami 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 Rhami 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 ArH05T (CTT)\textsubscript{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.

References
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 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.

References
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SEVERITY AND DISTRIBUTION OF PHOMA KOOLUMGA 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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P19
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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P20
BREEDING FOR ASCOCHYTA RESISTANCE IN DESI CHICKPEA.
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P21
GENETIC ENHANCEMENT OF CHICKPEA FOR ASCOCHYTA BLIGHT RESISTANCE
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P22
CONTROL OF PARTIAL RESISTANCE TO MYCOSPHAERELLA PINODES IN PEA
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P23
ENHANCEMENT OF BLACK SPOT RESISTANCE IN FIELD PEA
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P24
A COMPARISON OF PHENOTYPIC AND MARKER-ASSISTED SELECTION FOR ASCOCHYTA BLIGHT RESISTANCE IN CHICKPEA.
P. Castro(1), M.D. Fernandez(2), T. Millan(2), J. Gil(2) and J. Rubio(1), 1IFAPA, Córdoba, Spain, 2Córdoba University, Córdoba, Spain. Email: patriciar.castro@juntadeandalucia.es
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P25
PARTIAL CLONING OF TWO POLYKETIDE SYNTHASE GENES ASSOCIATED WITH PATHOGENICITY OF ASCOCHYTA RABIEI.
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P26
COMPARATIVE POPULATION STUDY OF DIDYMELLA RABIEI IN TURKEY AND ISRAEL
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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.
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P28
A SYSTEM-BASED RISK ESTIMATOR OF ASCOCHYTA BLIGHT DISEASE IN SOUTH AUSTRALIA
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P29
MANAGEMENT OF ASCOCHYTA BLIGHT OF CHICKPEA IN INDIA.
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MANAGEMENT OF ASCOCHYTA BLIGHT OF CHICKPEA IN NORTHERN NSW.
K.J. Moore, K.D. Lindbeck, P. Nash, G. Chiplin and E. J. Knights, New South Wales – Department of Primary Industries. 4 Tamworth Agricultural Institute, Tamworth, NSW, Australia. 8 Wagga Wagga Agricultural Institute, Wagga Wagga, NSW, Australia. Email: kevin.moore@dpi.nsw.gov.au
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APPLICATIONS OF SUPPRESSION SUBTRACTIVE HYBRIDIZATION (SSH) IN IDENTIFYING DIFFERENTIALLY EXPRESSED TRANSCRIPTS IN ASCOCHYTARABIEI.
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P32
INDUCED MUTATIONS FOR ASCOCHYTA BLIGHT RESISTANCE IN CHICKPEA (CICER ARIETINUM L.)
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P33
SCARS MARKERS LINKED TO ASCOCHYTA RABIEI IN CHICKPEA (SCAE19336, SCM02335 AND SCY17590): EXPRESSION STUDIES AND HOMOLOGIES WITH EST AND RELATED SEQUENCES.
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P34
UNDERSTANDING ASCOCHYTA BLIGHT RESISTANCE IN CHICKPEA USING MOLECULAR GENETICS AND GENOMIC APPROACHES
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P35
PATHOGENESIS-RELATED GENES AND GENETIC VARIATION IN POTENTIAL RESISTANCE GENES OF MAJOR EUROPEAN LEGUMES: THE LEGRESIST PROJECT.
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P36
PATHOTYPE SPECIFIC SEEDLING AND ADULT-PLANT RESISTANCE SOURCES TO ASCOCHYTA RABIEI IN CHICKPEA (CICER ARIETINUM L.).
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P37
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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P39
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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P42
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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P44
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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P46
DEVELOPMENT OF SCREENING TECHNIQUES AND IDENTIFICATION OF NEW SOURCES OF RESISTANCE TO ASCOCHYTA BLIGHT DISEASE OF CHICKPEA.
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