PPSA

Proceedings of the 32nd Annual Meeting of the Plant Pathology Society of Alberta

November 7th to 9th, 2011
University of Alberta Conference Centre
Edmonton, Alberta

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Schedule of Events – 32nd Annual Meeting

November 7-9, 2011 **Lister Conference Centre, University of Alberta, Edmonton, AB**
116th Street and 87th Avenue, Edmonton

Date & Time	Event	Location
Monday, Nov. 7th		
6:00pm – 8:00pm	Registration	Wild Rose Room
6:00pm – 9:00pm	Informal mixer	
Tuesday, Nov. 8th		
8:00am – 8:30am	Registration	Wild Rose Room
8:00am – 8:30am	Poster set-up	Wild Rose Room
8:30am – 8:45am	Welcome and opening remarks	Wild Rose Room
8:45am – 10:00am	Paper session I	Wild Rose Room
10:00am – 10:30am	Refreshment break	
Wild Rose Room 10:30am – 12:00pm	Paper session II	Wild Rose Room
12:00pm – 1:30pm	Lunch	Wild Rose Room
1:30pm – 2:45pm	Paper session III	Wild Rose Room
2:45pm – 4:15pm	Poster session	Wild Rose Room
3:00pm – 3:15pm	Refreshment break	Wild Rose Room
6:30pm – 9:00pm	Banquet	Wild Rose Room
Wednesday, Nov. 9th		
8:00 – 8:30am	Continental Breakfast	
8:30am – 11:00am	Business meeting	Wild Rose Room
10:00am – 10:15am	Refreshment break	Wild Rose Room

Contributed Paper Session 1 (Chairperson – Bruce Gossen) **Wild Rose Room**

08:45 **Stripe rust resistance among western Canadian wheat and triticale cultivars.**
D.A. Gaudet, H. Randhawa, B.J. Puchalski, M. Frick, A. Goyal, T. Despins, R.J. Graf, A. Laroche.

09:00 **Recent shifts in the stripe rust (*Puccinia striiformis*) populations in central Alberta.** M.D. Holtz, K. Kumar, L. Langford, J. Zantinge, and K. Xi.

09:15 **Towards the genome sequencing of Alberta races of *Puccinia striiformis*, the causal agent of stripe rust.**
A. Laroche, M. Frick, B. Puchalski, R, Graf, H. Randhawa, and D. Gaudet.

09:30 **The 2011 stripe rust epidemic in western Canada.**
B.J. Puchalski, DA. Gaudet, R. Harpinder, R. Graf, and S. Wogsberg.

09:45 ***Rhynchosporium secalis* virulence and genetics in relation to barley cultivar resistance.**
K. Xi, J. Zantinge, J. Meadus, J. Nyachiro, and K. Turkington.

10:00 -10:30 **Refreshment Break** **Wild Rose Room**

Contributed Paper session 2 (Chairperson – Jie Feng)

10:30 **Transformation of the barley pathogen *Pyrenophora teres* with the *ToxB* gene confers increased virulence on wheat.**
Y.M. Kim, R. Aboukhaddour, T.K. Turkington, and S.E. Strelkov.

10:45 **Transcriptional analyses of plant defense pathway related genes in cereals.** M.M. Frick, Y. Xu, L. Robert, P. Gulick, L. Harris. C. Badea, C. Penniket, and A. Laroche.

11:00 **Deep sequencing reveals an additional small subgenomic RNA in potato leaf roll virus.**
Y.T. Hwang, M. Kalischuk, A.F. Fusaro, P. Waterhouse, and L. Kawchuk.

11:15 **A simple method to distinguish between pararetrovirus virion and pararetrovirus DNA sequence integrated into a host genome.**
M.L. Kalischuk, and L.M. Kawchuk.

11:45 **Novel treatments for control of *Sclerotinia* white mold on dry edible bean.**
M.W. Harding, M.J. Unruh, and M.E. Olson.

12:00 – 13:30 **Lunch buffet (provided)** **Wild Rose Room**

Contributed Paper session 3 (Chairperson – George Turnbull) **Wild Rose Room**

13:30 **Genetic composition of *Phytophthora infestans* in Canada reveals increased diversity.**
L.M. Kawchuk, M.L. Kalischuk, K.I. Al-Mughrabi, R.D. Peters, R. J. Howard, and H.W. (Bud) Platt.

13:45 **Initial insensitivity response of *Mycosphaerella pinodes* isolates to pyraclostobin fungicide.**
R. Bowness, K.F. Chang, B.D. Gossen, R.S. Goswami, S.F. Hwang, C.J. Willenborg, and S.E. Strelkov.

14:00 **Genetics of clubroot resistance in rutabaga (*Brassica napus* var. *napobrassica*).**
M.J. Hasan, and H. Rahman.

14:15 **Quantitative PCR as a tool to evaluate resistance to *Plasmodiophora brassicae* in *Brassica* hosts.**
T. Cao, S.F. Hwang, and S.E. Strelkov.

14:30 **Effect of soil type on assessment of biofungicide efficacy against clubroot under controlled conditions.**
B.D. Gossen, H. Kasinathan, G. Peng, and M.R. McDonald.

14:45 **Wisconsin Fast Plants as model crops for studies of clubroot**
B.D. Gossen, K. Sharma, K.K.C. Adhikari, and M.R. McDonald.

14:45 – 16:15 **Contributed Poster Session 1 (Chairperson – Denis Gaudet/Michael Harding)**
Wild Rose Room

15:00-15:15 **Refreshment Break and Group Photo** **Wild Rose Room**

18:30 –21:00 **Banquet** **Wild Rose Room**

Titles for posters presented:

P1. **Crop residues influence *Rhizoctonia solani* population dynamics and reduce canola seedling blight.**
H.U. Ahmed, S.F. Hwang, G.D. Turnbull, Q.X. Zhou, S.E. Strelkov, and B.D. Gossen

P2. **Effect of nozzle type and orientation on fungicide efficacy against mycosphaerella blight in field pea.**
R. Bowness, B.D. Gossen, R.L. Conner, T. Wolf, K.F. Chang, C. Willenborg, and S.E. Strelkov

P3. **Effect of host resistance on infection by *Plasmodiophora brassicae* in canola.**
A. Deora, B.D. Gossen, and M.R. McDonald

P4. **Characterization of the potato leafroll virus silencing suppressor and phloem confinement.**
Y.T. Hwang, M. Kalischuk, F. Leggett, and L. Kawchuk.

P5. **Transgenerational pathogen resistance in canola following biotic stress.**
M.L. Kalischuk, L.M. Kawchuk, and I. Kovalchuk

P6. **Rapid spread of *Apioplagiostoma populi* causing bronze leaf disease on poplar.** M.L. Kalischuk, L.M. Kawchuk, and R.J. Howard

P7. **Current status of stripe rust of wheat and barley in central Alberta.**
K. Kumar, K. Xi, M. Holtz, L. Langford, L. Vandeermaar, and M. Wilson

P8. **Effect of a wide range of pH values on resting spore germination and clubroot (*Plasmodiophora brassicae*) severity in canola.**
A. Rashid, H.U. Ahmed, S.F. Hwang, and S.E. Strelkov

P9. **Evaluation of primary and secondary infection by *Plasmodiophora brassicae* in both resistant and susceptible canola genotypes.**
Q. Xiao, J. Feng, S.F. Hwang, and S.E. Strelkov

P10. **Pathogenicity and genetic variation in *Rhizoctonia* spp. isolated from canola in central Alberta.**
Q.X. Zhou, S.F. Hwang, and S.E. Strelkov

P11. **Evaluation of a bacterium on plant growth promotion and disease control in greenhouse vegetables.** J. Yang and Y. He

P 12. **Oxidized silvers: experimental fungicides for control of white mold on dry bean.** M.J. Unruh, S. Lepp, R.J. Howard, and M.W. Harding.

Wednesday November 9th

08:00 – 08:30	**Continental breakfast**	**Wild Rose Room**
8:30 – 11:00	**Business meeting**	**Wild Rose Room**
10:00 – 10:30	**Refreshment break**	**Wild Rose Room**

32nd Annual Meeting of the Plant Pathology Society of Alberta
Abstracts

Oral Presentations:

Stripe rust resistance among western Canadian wheat and triticale cultivars. D.A. Gaudet, H. Randhawa, B.J. Puchalski, M. Frick, A. Goyal, T. Despins, R.J. Graf, A. Laroche. *Agriculture and Agri-Food Canada, Lethbridge, AB, Canada T1J 4B1.*

Stripe rust (*Puccinia striiformis* Westend.) is an important pathogen of wheat in western Canada and worldwide. One hundred and four spring wheat and triticale were evaluated for resistance to stripe rust in nurseries at Lethbridge and Creston B.C. during 2009 and 2010. Infection levels in all nurseries were high. The cultivars were also tested for the presence of the stripe rust genes *Yr10*, *Yr17*, *Yr18* and *Yr36,* using molecular markers. Among Canada Prairie Spring Red (CPSR) wheat class, newer varieties had lower severities compared to high severities on many of the older varieties. Among the white Canada Prairie Spring CPS wheats, Vista was resistant whereas Snowhite475 and Snowhite476 were susceptible. Among the hard white wheats, Karma was resistant whereas Snowstar and Snowbird were susceptible. Sixty percent of the Canadian Western Red Spring (CWRS) wheats including the currently popular varieties Lillian, Harvest and Kane were resistant. Susceptible CWRS varieties that are extensively seeded in western Canada include AC Barrie, Superb and McKenzie but also include the recently registered CDC Kernen and Vesper. Much of the stripe rust resistance particularly in the Canada Western Hard Red (CWHR), Canadian Western Extra Strong (CWES), and Canada Prairie Spring Red (CPSR) wheat classes was attributed to the presence of adult plant resistance gene *Lr34/Yr18*. However, numerous varieties were resistant but negative for markers for known genes indicating that uncharacterized genes for stripe rust are widespread among hexaploid wheats. Durum wheat and triticale varieties were universally resistant with the absence of tested markers. Therefore, there appear to be numerous sources of stripe rust resistance, both characterized and uncharacterized, among western Canadian wheat and triticale varieties.

Recent shifts in the stripe rust (*Puccinia striiformis*) populations in central Alberta. M.D. Holtz, K. Kumar, L. Langford, J. Zantinge, and K. Xi. *Field Crop Development Centre, Alberta Agriculture and Rural Development, Lacombe, AB, Canada, T4L 1W1.*

Puccinia striiformis f. sp. *tritici* (Pst) and f. sp. *hordei* (Psh), the causal agents of stripe rust of wheat and barley, respectively, have been common in Alberta for the past 10 years. Isolates collected during 2010 and 2011 were tested for virulence on a series of differential lines and genotyped with 11 simple-sequence repeat (SSR) markers. Results for both *formae speciales*

indicated that recent shifts have occurred in the pathogen populations present in central Alberta. In 2010, Pst isolates showed few differences from Pst of previous years although pathotypes virulent on the Yr10 gene increased in frequency. During 2011, a new Pst genotype, matching that of a pathotype first detected in the Pacific States during 2007, was detected. During 2010, the Psh pathotypes that were common previously were rare. Most 2010 Psh isolates belonged to two distinct groups. Isolates in the first group were similar in virulence to those previously detected. Isolates in the second group were virulent on fewer barley differentials, but were highly virulent on several wheat and triticale differentials. All of these isolates also contained SSR alleles that had previously been found only in Pst isolates. These results indicate that new races continue to migrate to central Alberta and contemporary gene flow may occur between the two *formae speciales*.

Towards the genome sequencing of Alberta races of *Puccinia striiformis*, the causal agent of stripe rust. A. Laroche, M. Frick, B. Puchalski, R, Graf, H. Randhawa, and D. Gaudet. *Agriculture and Agri-Food Canada, Lethbridge, AB, Canada T1J 4B1.*
Wheat stripe rust (*Puccinia striiformis f. sp. tritici*) is a highly aggressive and variable pathogen of wheat and triticale, quickly evolving new races that overcome existing resistance worldwide. Historically, stripe rust has been a minor, sporadic and localized problem. However this race variability has resulted in 3 major and 2 minor epidemics in 5 of the past 6 years resulting in average losses of 30% in some regions. Most Canadian wheat classes have some highly susceptible varieties that are popular and planted over substantial hectares. The goal of this study is to sequence the genome of 16 local races of *P. striiformis* with an emphasis on races exhibiting high temperature tolerance or a wide or unique virulence spectrum. A genome size of ≈68 MB has been reported of *P. striiformis* and sequencing the genome using next-generation sequencing will enable molecular distinction of Canadian races and development of race-specific DNA fingerprints. The major challenge will be assembly of the over 200-1000 million base sequences for each strain. Sequencing and subsequent analyses should permit identification of the molecular determinants conferring high temperature tolerance and virulence on previously effective resistance genes and will aid in understanding virulence shifts in the pathogen in order to design and deploy new, durable resistance genes.

The 2011 stripe rust epidemic in western Canada. B.J. Puchalski, DA. Gaudet, R. Harpinder, R. Graf, and S. Wogsberg. *Agriculture and Agri-Food Canada, Lethbridge, AB, Canada T1J 4B1.*
High infection levels of stripe rust in wheat were observed throughout Southern Alberta and in western and central Saskatchewan in 2011. Winter wheat established in the fall of 2010 became heavily infected prior to winter and over-wintered in a number of commercial fields. Over-wintering of the pathogen, attributed to the presence of a more persistent protective winter

snow cover, was observed in the counties of Warner, Lethbridge, Taber and Bow Island. In the spring, severely infected winter wheat fields were observed 40 days earlier than in previous years. A cool-wet spring favoured high infection levels in winter wheat leading to its subsequent spread to the juvenile spring wheat. By mid-July, most susceptible varieties of spring and winter wheat were highly infected. Progression of the disease was slower north of Highway 1 and west of Highway 36 where severe stripe rust levels not observed until mid-August. Juvenile infections were common in most spring and winter wheat varieties; this is consistent with the fact that the majority of resistance to stripe in varieties was imparted by the *Yr18*, adult plant resistance gene. Fungicide applications to control stripe rust were common and occurred far earlier than previous years. There was a moderate increase in the number of acres sown to susceptible varieties in 2011 compared with 2010.

***Rhynchosporium secalis* virulence and genetics in relation to barley cultivar resistance.** K. Xi, J. Zantinge, J. Meadus, J. Nyachiro, and K. Turkington. *(K.X., J.Z., J.N.) Field Crop Development Centre, Alberta Agriculture and Rural Development, Lacombe, AB, Canada, T4L 1W1; and (J.M., K.T.) Agriculture and Agri-Food Canada, Lacombe, AB, Canada, T4L 1W1.*
The use of resistant barley cultivars is a primary strategy for the management of scald caused by *Rhynchosporium secalis* in central Alberta. The objective of the present study was to determine *R. secalis* pathotype virulence in relation to cultivar resistance. Field plots consisting of barley scald differentials with resistance genes and local commercial cultivars were set up across central Alberta during 2007 to 2011 to monitor scald development in relation to changes in pathotype virulence. The scald reactions of a few barley differentials and commercial resistant cultivars were found to change substantially during the test period. Inoculation at the seedling stage demonstrated that virulent pathotype E#2 and weakly virulent pathotype H#2 were generally separated based on virulence spectra. DNA microsatellites showing polymorphism did not reveal a clear pattern in grouping of isolates collected from central Alberta. Analysis of molecular variance showed a small but significant genetic difference accounting for 12% of total variation between the early (1997-2004) and the more recently (2009-2010) collected isolate groups. A higher degree of genetic similarity was found between other chronological groups. Sequence comparison of a 1.4 kb DNA band designated ‘OPX7’, previously found to be polymorphic between E#2 and H#2, was able to separate the pathotypes according to virulence in relation to barley cultivar resistance.

Transformation of *Pyrenophora teres* with the *ToxB* gene causes altered infection phenotypes on wheat and barley. Y.M. Kim, R. Aboukhaddour, T.K. Turkington, and S.E. Strelkov. *(Y.M.K., R.A., S.E.S.) Department of Agricultural, Food and Nutritional Science, University of Alberta, Edmonton, AB, Canada, T6G 2P5; and (T.K.T.) Agriculture and Agri-Food Canada, Lacombe, AB, Canada, T4L 1W1.*
Ptr ToxB, a 6.6 kDa proteinaceous molecule, is a host-specific toxin (necrotrophic effector) produced by some races of *Pyrenophora tritici-repentis*, the causal agent of tan spot of wheat. The toxin is encoded by the *ToxB* gene and induces extensive chlorosis on Ptr ToxB-sensitive wheat genotypes. A sister species of *P. tritici-repentis*, *Pyrenophora teres*, causes net blotch of barley, an economically important foliar disease of cultivated barley worldwide. In order to gain further insights into the function of Ptr ToxB, we transformed the ‘wild-type’ *ToxB* gene from *P. tritici-repentis* into *P. teres* by PEG-mediated protoplast transformation. Integration of the *ToxB* gene in the transformed *P. teres* strains was evaluated by PCR and Southern blotting analyses, while production of Ptr ToxB was assessed via Western blotting. The transformed *P. teres* strains were then tested for virulence on their original barley host as well as on Ptr ToxB-sensitive and insensitive

wheat genotypes. Transformants expressing functional Ptr ToxB caused significantly increased levels of disease on a net blotch-resistant barley cultivar and an altered infection phenotype on a Ptr ToxB-sensitive wheat genotype, compared with the un-transformed strain. These results suggest that Ptr ToxB or ToxB-like proteins may contribute to virulence in pathosystems other than tan spot.

Transcriptional analyses of plant defense pathway related genes in cereals. M.M. Frick, Y. Xu, L. Robert, P. Gulick, L. Harris. C. Badea, C. Penniket, and A. Laroche. *(MMF, Y.X., A.L.) Agriculture and Agri-Foods Canada, Lethbridge, AB, Canada T1J 4B1, (L.R., L.H.) ECORC Research Centre, Ottawa, ON, Canada K1A 0C6, (P.G.) Concordia University, Montreal, QC, Canada H4B 1R6, (C.P.) University of Lethbridge, Lethbridge, AB, Canada T1K 3M4, (C.B.) University of Alberta, Edmonton, AB, Canada T6G 2G6.*
Salicylic acid (SA) functions as a key signal in regulating disease resistance in response to biotrophic pathogen attack. It induces pathogenesis-related gene expression and the synthesis of defensive compounds associated with both local and systemic acquired resistance resulting in a hypersensitive response and cell death. Alternately, for necrotrophic pathogens, jasmonic acid (JA) and ethylene (ET) signalling pathways regulate the plant defense response through induced systemic response. The JA and ET pathways are also involved in response to abiotic stresses. The SA and JA signalling pathways operate antagonistically in plant defense as cell death would be advantageous to necrotrophic pathogens. Using 454 and Illumina sequencing, we have produced 12.3 million 454 reads and 214.9 million Illumina reads from different tissues at various development stages and exposed to different stress treatments from both rye and triticale. Analyses yielded a reference transcriptome of more than 49,000 rye contigs covering more than 90% of the hypothetical gene loci. Analysis of the triticale transcriptome is ongoing. From our sequencing data, we have identified the various genes involved in salicylic acid, jasmonic acid and ethylene synthesis in rye and triticale and determined their expression levels amongst the various tissues sequenced to gain a better understanding of their role in plant responses to biotic and abiotic stress.

Deep sequencing reveals an additional small subgenomic RNA in potato leaf roll virus. Y.T. Hwang, M. Kalischuk, A.F. Fusaro, P. Waterhouse, and L. Kawchuk. *(Y.T.H., L.K) Agriculture and Agri-Food Canada, Lethbridge, AB, Canada T1J 4B1, (M.K.) University of Lethbridge, AB, Canada T1K 3M4, (A.F.F., P.W.) CSIRO Plant Industry, Canberra ACT 2601, Australia.*
Potato leafroll virus (PLRV) causes significant economic losses worldwide in infected potato (*Solanum tuberosum* L.). PLRV is a single-stranded, positive-sense RNA virus (genus: *Poleroviruses*, family *Luteoviridae*) transmitted by aphids in a persistent manner and remains restricted to phloem tissues of the infected plants. PLRV produces isometric particles that encapsidate a 5.9 kb genomic RNA. Previous studies have shown the 3’ proximal viral open reading frames (ORFs) were translated from the ~2.3 kb subgenomic RNA1 (sgRNA1; ORF3, ORF3/ORF5 and ORF4) and the ~800 bp sgRNA2 (ORF6 and ORF7). Here we report that deep sequencing analysis of viral small RNAs profile for PLRV-infected plants allowed the detection of a third ~0.5 kb sgRNA3 that has not been previously described. Both quantitative real-time polymerase chain reaction (qRT-PCR) and northern blots analyses confirmed the existence of this small sgRNA3 in PLRV-infected plants. The 5’ rapid amplification of

cDNA ends analyses and subsequent southern blot analyses mapped the 5’ initiation site of sgRNA3 to the 3’ end of PLRV RNA genome (position 5347). A regulatory role has been postulated for PLRV sgRNA3 with similar characteristics to sgRNA3 identified in other Luteoviruses.

A simple method to distinguish between pararetrovirus virion and pararetrovirus DNA sequence integrated into a host genome. M.L. Kalischuk, and L.M. Kawchuk. *(M.L.K.) University of Lethbridge, Lethbridge, AB, Canada, (M.L.K., L.M.K.) Agriculture and Agri-Food Canada, Lethbridge, AB, Canada T1K 4B1.*

Rubus yellow net virus (RYNV) is a pararetrovirus belonging to the genus *Badnavirus* in the family of *Caulimoviridae* and is a serious component of mosaic disease in red raspberry (*Rubus idaeus* L). The genome of RYNV is composed of 8001 bp of circular dsDNA encapsidated in a bacilliform virus particle. The RYNV genome is typically in a relaxed state produced by one discontinuity in each DNA strand. Immediately before transcription within a cell nucleus, the relaxed state of the RYNV genome is filled in by host DNA dependent DNA polymerases to produce a supercoiled minichromosome. Under some conditions, the viral minichromosome can become inserted into the host genome through homologous recombination. Interestingly, both infection and innate immunity can arise from pararetrovirus DNA sequence integrated into a host genome. It is therefore important for disease forecasting and prevention strategies to differentiate between an infection of actively multiplying virions and integrated genomic sequence. Polyclonal antibodies were developed against the coat protein of *Sugarcane bacilliform virus* and RYNV and used in immunological-polymerase chain reaction (IC-PCR) for detecting RYNV. Transmission electron microscopy (TEM) confirmed that the IC-PCR was a highly sensitive assay for detecting encapsidated virus particles. Serological relationships amongst badnavirus members were also obtained. IC-PCR is a technically simple, accurate, rapid and cost-effective method for distinguishing between encapsidated virus particles and pararetroviral DNA sequences that have been integrated into a host genome.

Fruit infection and postharvest decay of greenhouse tomatoes caused by *Penicillium* species in British Columbia. S. Chatterton, A. Wylie, and Z.K. Punja. *(S.C.) Agriculture and Agri-Food Canada, Lethbridge, AB, Canada T1J 4B1, (A.W., Z.K.P.) Department of Biological Sciences, Simon Fraser University, Burnaby, BC, Canada V5A 1S6.*

In 2009 to 2011, a previously undescribed spotting of tomato fruits was observed in the greenhouse and packinghouse of a commercial organic greenhouse producer in the Fraser Valley of British Columbia. Symptoms initially appeared as small black spots on the tomato surface that were barely visible at harvest. The spots expanded after 5-10 days of storage to form larger gray and yellow lesions with evidence of mycelial growth in the center. Isolations from symptomatic skin and pericarp tissues from early and expanded lesions yielded three species of *Penicillium*. Healthy tomato fruits inoculated with isolates identified as *P. olsonii* developed black spots which progressed to form larger gray lesions, typical of symptoms observed in the commercial greenhouse. Isolates identified as *P. solitum* and *P. polonicum* only caused soft rot and decay. Swabs of tomato fruit surfaces and calyx tissues taken from fruit developing on the vine, followed by plating onto PDA,

revealed that high populations of *P. olsonii* were present (> 40 CFU/ fruit). Similar populations were recovered from calyx tissues plated directly onto agar media. Wounding did not significantly enhance disease severity compared to unwounded treatments, suggesting that the mode of entry of *P. olsonii* is likely by entry of resident populations through naturally occurring cracks in the fruit cuticle. Studies undertaken to determine conditions favouring infection indicated that lesion development was greatest at low (12^{o}C) temperatures, and when *P. olsonii* conidia were inoculated onto the shoulder of ripe tomato fruits. To our knowledge, this is the first report of a fruit spotting and postharvest decay of tomato fruits caused by *P. olsonii*.

Novel treatments for control of *Sclerotinia* white mold on dry edible bean. M.W. Harding, M.J. Unruh, and M.E. Olson. *Innovotech Inc., Edmonton, AB Canada T6N 1H1.*
White mold on dry bean, caused by ***Sclerotinia sclerotiorum*** (Lib.) de Bary is a major production problem for dry and succulent bean producers in Canada. In years where environmental conditions are conducive to disease development, the disease can lead to devastating yield reduction and economic loss. Genetic resistance to white mold in dry bean germplasm is limited or unavailable. As a result, foliar fungicides are an important tool in the integrated approach required to manage white mold. Fungicides currently registered are preventative and, in some years, are often unable to turn the tide of disease epidemics. New and improved fungicides are needed to help manage white mold. In this study, methods of improving white mold control in dry beans using micronutrient fertilizers, or a plant defense activator, were analyzed. A replicated, small-plot study (ca. 750m^{2}) was established in a randomized complete block design at Brooks, Alberta. Industry standard fungicides, and water as an untreated check, were compared to experimental treatments. Treatments were applied to seed prior to planting, or as foliar treatments at flowering. The results clearly indicated that white mold control could be significantly improved by one micronutrient (manganese) when applied at 20% to 50% bloom in a tank mix with an industry standard fungicide (boscalid). Additionally, the plant defense activator (saponin) demonstrated significant potential as a seed treatment for improvement of white mold management in dry bean production systems.

Genetic composition of *Phytophthora infestans* in Canada reveals increased diversity. L.M. Kawchuk, M.L. Kalischuk, K.I. Al-Mughrabi, R.D. Peters, R. J. Howard, and H.W. (Bud) Platt. *(L.M.K.) Agriculture and Agri-Food Canada, Lethbridge, AB, Canada T1J 4B1; (M.L.K.) Department of Biological Sciences, University of Lethbridge, Lethbridge, AB, Canada T1K 3M4; (K.I.A.) NB Department of Agriculture and Aquaculture, Wicklow, NB; (R.D.P. H.W.P.) Agriculture and Agri-Food Canada, Charlottetown, PE; (R.J.H.) Crop Diversification Centre South, Alberta Agriculture and Rural Development, Brooks, AB, Canada T1R 1E6.*

A dramatic increase in the incidence of late blight and changes within populations of *Phytophthora infestans* were observed recently in Canada. The occurrence of several new genotypes with associated phenotypes that dominated pathogen populations in various regions was documented. Genotype US-23 that was previously detected only among isolates from the United States, dominated in the western Canadian provinces of British Columbia (BC), Alberta (AB), Saskatchewan (SK), and Manitoba (MB). Although US-23 infects both potato and tomato, it was more aggressive on tomato and was the only genotype recovered from commercial garden centers. Genotype US-8, previously dominant throughout Canada, was the only genotype detected in isolates from the eastern Canadian provinces of New Brunswick (NB) and Prince Edward Island (PE). Other genotypes detected in Canada included US-11 in AB, US-24 in MB, and US-22 in Ontario (ON). An additional genotype was detected in ON, US-22a, which appears to be a derivative of US-22 that may have arisen through sexual reproduction. However, clonal reproduction dominated and opportunities for sexual reproduction were probably limited because of a surprising separation of the A1 and A2 mating types geographically. Long-distance movement in seed tubers and garden center transplants contributed to the rapid spread of the genotypes across Canada.

Initial insensitivity response of *Mycosphaerella pinodes* isolates to pyraclostobin fungicide. R. Bowness, K.F. Chang, B.D. Gossen, R.S. Goswami, S.F. Hwang, C.J. Willenborg, and S.E. Strelkov. (*R.B.) Alberta Agriculture and Rural Development, Lacombe, AB, (K.F.C., S.F.H.) Alberta Agriculture and Rural Development, Edmonton, AB, (B.D.G.) Agriculture and Agri-Food Canada, Saskatoon, SK, (R.S.G.) North Dakota State University, Fargo, ND, (C.J.W.) University of Saskatchewan, Saskatoon, SK, (S.E.S.) University of Alberta, Edmonton, AB.*
Mycospharella pinodes (Berk. and Blox.) Vestergren (anamorph *Ascochyta pinodes* L.K. Jones) is the dominant pathogen in the Ascochyta blight disease complex on field pea in western Canada. Symptoms include necrotic lesions on leaves, stems and pods that reduce straw strength, seed quality and yield. The most effective management strategy is the repeated application of foliar fungicides. Pyraclostrobin, a strobilurin fungicide, is frequently applied across western Canada and is highly effective. However, strobilurins have a site-specific mode of action and there is a high risk that this pathogen population will develop fungicide-insensitivity. Seventy-five isolates, never exposed to this chemistry, were collected from Saskatchewan, Alberta and North Dakota. These isolates were assessed for baseline insensitivity to pyraclostrobin using radial growth measurements on potato dextrose agar amended with fungicide concentrations ranging from 0 µg/mL to 50 µg/mL a.i. The fungicide concentration that effectively inhibited 50% of growth (EC_{50}) was 0.12 µg/mL. Using this discriminatory dose, isolates collected from the same geographical area in 2010 and 2011were tested. Of 60 isolates analyzed, 10 were very sensitive to pyraclostrobin, 7 were insensitive, while the majority (43)

responded in a similar manner to the unexposed isolates showing intermediate sensitivity. The identification of pyraclostrobin-insensitive isolates indicates that fungicide resistance may be developing in this pathosystem.

Quantitative PCR as a tool to evaluate resistance to *Plasmodiophora brassicae* in *Brassica* hosts. T. Cao, S.F. Hwang, and S.E. Strelkov. *(T.C., S.E.S.) Department of Agricultural, Food and Nutritional Science, University of Alberta, Edmonton, AB, Canada, T6G 2P5; and (S.F.H.) Crop Diversification Centre North, Alberta Agriculture and Rural Development, Edmonton, AB, Canada, T5Y 6H3.*
Clubroot, caused by *Plasmodiophora brassicae* (Pb), is an emerging canola disease in Alberta. In an attempt to rapidly identify canola lines carrying clubroot resistance, quantitative PCR (qPCR) analysis was employed to measure the amount of Pb DNA in the roots of susceptible, moderately susceptible, moderately resistant and resistant hosts, as well as in a non-host genotype (wheat), at 5, 10, 15, 20, and 42 days post-inoculation (dpi). Results indicated that Pb biomass, as estimated by Pb DNA, showed an overall upward trend in clubroot susceptible hosts from 5 to 42 dpi, in contrast to an overall downward trend in clubroot resistant hosts and wheat. Disease severity was positively correlated with the amount of Pb DNA in roots sampled at 42, 20, 15, and 5 dpi. The amount of Pb DNA in the roots sampled at 20, 15, and 5 dpi was also positively correlated with the amount of Pb DNA in the roots sampled at 42 dpi. These results suggest that the *in planta* quantification of Pb DNA at 20, 15, and 5 dpi could be used as an early indicator of the final disease reaction of the host to the pathogen.

Effect of soil type on assessment of biofungicide efficacy against clubroot under controlled conditions. B.D. Gossen, H. Kasinathan, G. Peng, and M.R. McDonald. *(B.D.G., G.P.) Agriculture and Agri-Food Canada, Saskatoon, SK, Canada, S7N 0X2; and (H.K., M.R.M.) Department of Plant Agriculture, University of Guelph, Guelph, ON, Canada, N1G 2W1.*
Three studies were conducted under controlled conditions to examine the effect of soil type on the efficacy of two biofungicides, Serenade (*Bacillus subtilis*) and Prestop (*Gliocladium catenulatum*), against clubroot caused by *Plasmodiophora brassicae* (Woronin). Combinations of three factors were examined: soil type (muck soil, pH 6.2; mineral soil, pH 6.8; non-calcareous sand, pH 6.5; soil-less mix, pH 6.0), pathotype (P3, P6), and biofungicide. Seedlings of canola (*Brassica napus* L.'46A76') and Shanghai pak choy (*B. rapa* L. subsp. Chinensis (Rupr.) var. communis Tsen and Lee 'Mei Quing Choi') were treated with Serenade (5% v/v) and Prestop (7.5 g L-1 of water) at 5 days after germination inoculated with 5×10^5 resting spores of *P. brassicae* per seedling 3 days later. Clubroot severity was assessed at 6 weeks after inoculation. Each experiment consisted of four replicates and 12 plants per experimental unit, and each was repeated. Clubroot levels (incidence and severity) in Shanghai pak choy were consistently higher than in canola, and inoculation with P3 resulted in slightly higher clubroot levels than P6. Clubroot levels in soilless mix were substantially lower than in the other soil types, but there was little or no difference among the other soil types. Application of the biofungicides often reduced clubroot incidence and severity, but the reduction was generally small. Also, the relative impact of the two biofungicides was not consistent; Prestop was the more effective agent in two trials, but Serenade was more effective in the third trial. There was also a small interaction of soil type with biofungicide, associated primarily with low levels of clubroot in the soilless mix treatment. These results indicate that growing medium is an important factor in evaluation of clubroot under controlled conditions, and that soil type will likely influence the efficacy of biofungicide in field situations.

Wisconsin Fast Plants as model crops for studies of clubroot. B.D. Gossen, K. Sharma, K.K.C. Adhikari, and M.R. McDonald. *(B.D.G.) Agriculture and Agri-Food Canada, Saskatoon, SK, Canada, S7N 0X2; and (K.S., K.K.C.A., M.R.M.) Department of Plant Agriculture, University of Guelph, Guelph, ON, Canada, N1G 2W1.*
The acreage affected by clubroot (*Plasmodiophora brassicae* Woronin) on canola (*Brassica napus* L., *B. rapa* L) in western Canada is expanding rapidly. Arabidopsis is widely used as a model crop for studies of clubroot, but a model crop with a plant architecture more similar to susceptible crop species would be useful for many kinds of studies. Wisconsin Fast Plants are small, short-generation selections that represent a wide range of *Brassica* species, which might be very useful as model crops for such studies. Field assessments of the reaction of selected lines were conducted from 2008–2010 on naturally infested soil at the Muck Crops Research Station in Ontario,

where pathotype 6 is predominant. *B. carinata* and *B. juncea* were highly susceptible, several lines of *B. napus* were moderately susceptible, and *B. napus* and *R. sativus* were resistant, and their response was consistent over years. Also, these selected lines were tested against pathotypes 2, 3, 5 and 6 under controlled conditions. The response to pathotype 6 under controlled conditions was strongly correlated with those from the field. In addition, a strong interaction in response to several of the pathotypes was observed for several of the lines. We conclude that Wisconsin Fast Plants have potential for use as model crops for clubroot research, e.g., under controlled conditions and other situations where space is limited.

Genetics of clubroot resistance in rutabaga (*Brassica napus* var. *napobrassica*). M.J. Hasan, and H. Rahman. *Department of Agricultural, Food and Nutritional Science, University of Alberta, Edmonton, AB, Canada, T6G 2P5.*

Development of canola (*Brassica napus* L.) cultivars resistant to clubroot disease, caused by *Plasmodiophora brassicae*, is an important objective for breeders and researchers. Several resistant canola cultivars have been developed and marketed by private companies in Canada, in which resistance is conferred by single gene. However, there are reports of the breakdown of monogenic resistance in Europe. Therefore, it is important to have multiple resistance genes in canola for durability in resistance to clubroot disease in this crop. The objective of this research is to introgress clubroot resistance from rutabaga into canola and to develop molecular marker(s) for use in marker-assisted breeding. Two rutabaga genotypes, Rutabaga-BF and Rutabaga-PL, inbred for resistance to multiple *P. brassicae* pathotypes including pathotype 3, were crossed with two clubroot susceptible spring canola lines, A07-29NI and A05-17NI. Double haploid (DH) populations were produced from the F_1 plants through the application of microspore culture technique, and were evaluated for resistance to pathotype 3. Segregation in the DH population of Rutabaga-BF × A07-29NI followed a simple Mendelian 1:1 segregation for resistant and susceptible phenotypes; while, segregation in the DH population of Rutabaga-PL × A05-17NI cross deviated significantly from a 1:1 ratio, suggesting a more complex genetic control of this trait in this population. Molecular mapping of resistance in these two populations is in progress.

Poster Presentations:

Crop residues influence *Rhizoctonia solani* population dynamics and reduce canola seedling blight. H.U. Ahmed, S.F. Hwang, G.D. Turnbull, Q.X. Zhou, S.E. Strelkov, and B.D. Gossen. *(H.U.A., G.D.T., Q.X.Z., S.F.H.) Crop Diversification Centre North, Alberta Agriculture and Rural Development, Edmonton, AB, Canada, T5Y 6H3; (S.E.S.) Department of Agricultural, Food and Nutritional Science,University of Alberta, Edmonton, AB, Canada, T6G 2P5; and (B.D.G.) Agriculture and Agri-Food Canada, Saskatoon, SK, Canada, S7N 0X2.*

Seedling blight of canola (*Brassica napus* L. and *B. rapa* L.), caused by *Rhizoctonia solani* Kühn, is an important disease that reduces crop stand establishment. Seed treatment chemicals are used to control seedling blight, but can have a negative impact on the environment. The effects of previous crop residues on soil *R. solani* populations and canola seedling blight severity were examined. Field plots were established in 2010 following a split-plot design with inoculum (*Rhizoctonia* and no *Rhizoctonia*) as the main plots and crop residue (barley, canola, oat and pea) as the sub-plots. Following harvest, the crop residues were ploughed down in the fall of 2010. In the spring of 2011, soil samples were collected from each experimental unit. The effect of crop residue on *Rhizoctonia* populations was assessed by growing canola in these soils in a replicated greenhouse trial following the same design as was used in the field. Populations of *Rhizoctonia* were estimated by soil dilution plating onto selective medium. Canola seedling emergence was greater, while damping off and disease severity were less severe, in soil with barley or oat residues compared to soil with canola or pea residues. Similar results were obtained in both inoculated and non-inoculated plots. A plate assay also revealed reduced *Rhizoctonia* populations in plots with barley or oat residues. Crop rotation and residue incorporation with barley or oat between canola crops may be a useful strategy to manage seedling blight of canola.

Effect of nozzle type and orientation on fungicide efficacy against mycosphaerella blight in field pea. R. Bowness, B.D. Gossen, R.L. Conner, T. Wolf, K.F. Chang, C. Willenborg, and S.E. Strelkov. *(R.B.) Alberta Agriculture and Rural Development (AARD), Lacombe, AB, Canada; (B.D.G., T.W.) Agriculture and Agri-Food Canada (AAFC), Saskatoon, SK, Canada, (R.L.C.) AAFC, Morden, MB, Canada, (K.F.C.) AARD, Edmonton, AB, Canada, (C.W.) University of Saskatchewan, Saskatoon, SK, Canada, (R.B., S.E.S.) University of Alberta, Edmonton, AB, Canada.*

Mycospharella pinodes (Berk. and Blox.) Vestergren causes substantial yield loss in field pea (*Pisum sativum* L.) across western Canada. Symptoms include necrotic lesions on leaves, stems and pods. Epidemics initiate at the base of the plant canopy, but quickly spread when conditions are favorable. The only effective strategy to manage mycosphaerella blight is the application of foliar fungicide. The effect of spray application options on severity of mycosphaerella blight were assessed at Morden, Man. in 2008–2010, Saskatoon, Sask. in 2008 and 2009 and Lacombe, Alta. in 2009. The

nozzles used included fine and coarse spray quality, single and double nozzle arrangements, a reduced application rate and a non treated control. Pyraclostrobin fungicide was applied in 250 L/ha of water at flowering. Across all years and sites, blight severity was lowest in the double nozzle system with either two fine droplet nozzles or a fine and coarse nozzle combination. Similarly, the highest yields were obtained using a double nozzle system, but differences were small. Mean yield was increased by only 4% over the single nozzle system and 13% over the control. It is likely that double nozzle systems improve canopy penetration and droplet retention, especially on stems, where lesions increase lodging and have the largest impact on yield.

Effect of host resistance on infection by *Plasmodiophora brassicae* in canola. A. Deora, B.D. Gossen, and M.R. McDonald. *(A.D., M.R.M.) Department of Plant Agriculture, University of Guelph, Guelph, ON, Canada, N1G 2W1; (B.D.G.) Agriculture and Agri-Food Canada, Saskatoon, SK, Canada, S7N 0X2.*
Little is known about how and when resistance to clubroot (*Plasmodiophora brassicae* Woronin) is expressed in canola (*Brassica napus* L.). Time series assessments of root hair infection and cortical infection were made in inoculated seedlings of four commercial cultivars that differed in reaction to two pathotypes (P3 and P6) of *P. brassicae*: cvs. ‘45H29’ (resistant), ‘Invigor 5030’ (partially resistant), ‘46A76’ (susceptible), and ‘45H21’ (susceptible to P3, resistant to P6). For assessment of root hair infection (RHI), seedlings were harvested at 4, 8, and 12 days after inoculation (DAI). For assessment of cortical infection (CI), plants were harvested at 16, 22, and 28 DAI. RHI occurred quickly in compatible (susceptible cultivar × pathotype) combinations and more slowly in incompatible combinations. The maximum RHI for both reactions was about 65%, except on ‘Invigor 5030’ where RHI was >60%. At 28 DAI, CI was high in the susceptible cv. ‘46A76’ (P3 = 45%, P6 = 35%), intermediate in ‘Invigor 5030’ (P3 = 23%, P6 = 16%), and no CI (0%) was observed for either pathotype in the resistant ‘45H29’. In ‘45H21’, CI caused by P3 was high (35%), but P6 resulted in no CI (0%). CI caused by P3 was consistently higher than for P6 in compatible reactions. Although there were small differences in the pattern of RHI associated with resistance, the largest impact of resistance was on CI.

Characterization of the potato leafroll virus silencing suppressor and phloem confinement. Y.T. Hwang, M. Kalischuk, F. Leggett, and L. Kawchuk. *(Y.T.H., F.L. L.K.) Agriculture and Agri-Food Canada, Lethbridge, AB, Canada T1J 4B1, (MK) University of Lethbridge, AB, Canada T1K 3M4.*
Potato leafroll virus (PLRV) is a single-stranded positive-sense RNA virus (genus: *Poleroviruses*, family *Luteoviridae*) transmitted by aphids in a persistent manner and causes significant losses in infected potato (*Solanum tuberosum* L.). The isometric particles of PLRV encapsidate a 5.9 kb genomic RNA and are largely confined to the phloem of infected plants. The phloem limitation of PLRV might be attributed to a combination of inability to

counteract the silencing-based plant defence mechanism outside of phloem tissue or protein-mediated mechanism that limits virus to phloem tissue to improve vector acquisition. Recently it was reported that Polerovirus P0 prevents the assembly of small RNA-containing RISC complexes and leads to degradation of ARGONAUT1. To further characterize the potential role of the different PLRV ORFs in phloem confinement and RNA silencing suppression, fusion proteins of were developed in frame with green fluorescent protein (GFP) within the full length infectious clone of PLRV. Results provide an excellent platform for the dissection of virus infection and RNA silencing mechanisms through reverse genetics.

Transgenerational pathogen resistance in canola following biotic stress. M.L. Kalischuk, L.M. Kawchuk, and I. Kovalchuk. *(M.L.K., I.K.) University of Lethbridge, Lethbridge, AB, Canada T1K 3M4, (M.L.K., L.M.K.) Agriculture and Agri-Food Canada, Lethbridge, AB, Canada T1K 4B1.*
Pathogen infection often causes instability in the host genome as a result of increased rearrangements in resistance gene loci and transgene sequences. In previous work, we demonstrated that in typical laboratory plants (e.g. *Nicotiana tabacum* and *Arabidopsis thaliana*) exposed to either virus, bacteria or fungi in the parent generation developed resistance to the same pathogen and exhibited cross-protection against other biotic and abiotic stresses. Here we investigated whether similar transgenerational response to infection can also be observed for economically important crops such as canola. Canola (*Brassica napus*) is an economically important crop in Canada, producing 10 billion tonnes of seed and contributing $15.4 billion to the Canadian economy annually. As a first step in this exploration process, *Brassica rapa* plants were infected with three different types of pathogens, *cauliflower mosaic virus*, *Pseudomonas syringae* pathovar *tomato* DC3000 or *Phytophthora brassicae.* The progeny (F1) of parent *B. rapa* (P0) plants were challenged with the same pathogen and the level of pathogen infection was quantified. We discovered F1 stemming from P0 exposed to the pathogen had lower pathogen titres than F1 deriving from P0 not exposed to the pathogen. This demonstrated the potential for developing pathogen resistance in canola through transgenerational inheritance. Although the mechanisms behind the pathogen resistance remain to be fully explored reversible alterations to the parent epigenome are known to occur through epigenetic marks such as DNA methylation, histone modifications, chromatin remodelling and/or RNA silencing and these marks are inherited by the offspring.

Rapid spread of *Apioplagiostoma populi* causing bronze leaf disease on poplar. M.L. Kalischuk, L.M. Kawchuk, and R.J. Howard. *(M.L.K.) Department of Plant Pathology, Washington State University, Pullman, USA 99164, (L.M.K.) Agriculture and Agri-Food Canada, Lethbridge, AB, Canada T1J 4B1; (R.J.H.) Crop Diversification Centre South, Alberta Agriculture and Rural Development, Brooks, AB, Canada T1R 1E6.*
Poplar (*Populus* species) is an important ornamental, windbreak, and pulp and wood product tree in Alberta and across western Canada because of its rapid growth, architecture, and hardiness. Symptoms resembling bronze leaf disease were observed in Alberta as early as 2003 and have been seen each subsequent year on an increasing number of *Populus x canescens* Smith, *Populus tremula* L., and *Populus tremuloides* Michx. trees from urban areas, shelterbelts and nurseries. Disease symptoms became pronounced in mid- to late summer with bronze to dark reddish brown leaves, while the petiole and the midrib remained green. Nucleic acid was extracted from isolated perithecia and amplified with the polymerase chain reaction and oligonucleotides 5’GCATCGATGAAGAACGCAGC3’ and 5’TCCTCCGCTTATTGATATGC3’ specific for rDNA ITS sequence. The cloned

amplified sequence of the *A. populi* rDNA ITS region (GenBank Accession No. GU205341) showed considerable homology (>90% identity) to other *Apioplagiostoma* species. In total, 117 independent leaf samples from 32 trees exhibiting disease symptoms were positive for *A. populi*, producing an approximately 300 bp sequence not observed in any of the symptomless samples. Continued spread of the pathogen threatens the viability of the shelterbelt, nursery and processed wood industries. The developed diagnostic is helping to determine the distribution and epidemiology of the pathogen.

Current status of stripe rust of wheat and barley in central Alberta. K. Kumar, K. Xi, M. Holtz, L. Langford, L. Vandeermaar, and M. Wilson. *Field Crop Development Centre, Alberta Agriculture and Rural Development, Lacombe, AB, Canada T4L 1W1.*

Stripe rust of cereals caused by *Puccinia striiformis* (Schm.) Eriks & Henn has been widespread in central Alberta since the late 90's, coinciding with the epidemics in the US. Surveys from 2007 to 2010 revealed that stripe rust was widespread in cereal crops in central Alberta, with disease incidence and severity in winter wheat being more severe than spring wheat, and wheat generally having higher severity than barley and triticale. Higher levels of stripe rust in winter wheat may have been caused by overwintering inoculum in this crop. The 2011 epidemics appeared to be the most extensive and severe in the last 10 years in central Alberta. Stripe rust was observed in all 43 commercial wheat fields with 11 winter wheat fields and 7 spring wheat fields being moderately or severely infected in the end of the 2011 season. Severe stripe rust on barley lines, especially 6-row, have also been observed in breeding nurseries at Lacombe and Morrin, indicating that barley is not immune to this disease although stripe rust in commercial barley fields was less severe than in wheat fields in central Alberta.

Effect of a wide range of pH values on resting spore germination and clubroot (*Plasmodiophora brassicae*) severity in canola. A. Rashid, H.U. Ahmed, S.F. Hwang, and S.E. Strelkov. *(A.R., H.U.A., S.F.H.) Crop Diversification Centre North, Alberta Agriculture and Rural Development, Edmonton, AB, Canada, T5Y 6H3; and (S.E.S.) Department of Agricultural, Food, and Nutritional Sciences, University of Alberta, Edmonton, AB, Canada, T6G 2P5.*

Clubroot, caused by *Plasmodiophora brassicae*, is an important soilborne disease of cruciferous crops. Disease development is associated with the formation of large root galls, which reduce water and nutrient uptake by infected plants. Liming of the soil to increase its pH is often recommended as an effective strategy to reduce clubroot severity. In order to evaluate the effectiveness of this approach to manage clubroot in canola (*Brassica napus*), root hair infection and clubroot severity over a range of soil pH values (pH 5.0 to 9.0) were assessed. Significant levels of root hair infection and gall formation were observed at neutral and alkaline pH (6.5 to 9.0), although these declined at pH <6.0. The germination rates of *P. brassicae* resting spores were also assessed after treatment with host root exudates, the pH of which also had been adjusted from pH 5.0 to 9.0. Germination of the spores was significantly higher in all treatments that contained root exudates, relative to a control in which the spores were treated with a nutrient solution alone. However, the highest spore germination rates were observed at pH 5.0 to 7.0 and gradually declined at pH >7.5 in both the presence and absence of root exudates. The implications of these results are discussed.

Evaluation of primary and secondary infection by *Plasmodiophora brassicae* in both resistant and susceptible canola genotypes. Q. Xiao, J. Feng, S.F. Hwang, and S.E. Strelkov. (Q.X., S.E.S.) *Department of Agricultural, Food and Nutritional Science, University of Alberta, Edmonton, AB, Canada, T6G 2P5; and (J.F., S.F.H.) Crop Diversification Center North, Alberta Agriculture and Rural Development, AB, Canada, T5Y 6H3.*
Clubroot, caused by *Plasmodiophora brassicae* Woronin, has emerged as a major disease of canola (*Brassica napus* L.) in Alberta, Canada. The deployment of resistant cultivars is an important aspect of an integrated control strategy. To better understand resistance mechanisms, primary and secondary infection of resistant ('73-77RR') and susceptible ('45H26') canola genotypes was investigated in two experiments, by inoculation with resting spore (primary zoospore) and secondary zoospore suspensions. Over an 8-day time course, the level of primary infection was lower in the resistant genotype than in the susceptible genotype, following inoculation with either resting spores or secondary zoospores. However, a greater number of secondary plasmodia (indicative of secondary infection) were observed in the resistant genotype. These results suggest that resistance in '73-77RR' is expressed during primary infection.

Pathogenicity and genetic variation in *Rhizoctonia* spp. isolated from canola in central Alberta. Q.X. Zhou, S.F. Hwang, and S.E. Strelkov. *(Q.X.Z., S.F.H.) Crop Diversification Centre North, Alberta Agriculture and Rural Development, Edmonton, AB, Canada, T5Y 6H3; and (S.E.S.) Department of Agricultural, Food and Nutritional Science, University of Alberta, Edmonton, AB Canada, T6G 2P5.*
Canola is an important oilseed crop grown worldwide. In western Canada, *Rhizoctonia* spp. represents one of the major pathogens causing canola seedling blight and damping off, which result in significant yield losses. In 2009, 2010 and 2011, 98 isolates of *Rhizoctonia* spp. were isolated from fields in central Alberta. The pathogenicity of these isolates on canola was tested, with 58% causing high disease severity, 26% causing low disease severity, and 5% causing no disease. Genetic variability among the isolates was assessed by analysis of the internal transcribed spacer (ITS) 1, 5.8S, and ITS 2 regions of the rDNA repeat, from which a neighbor-joining tree was constructed using the PAUP software. Isolates of *Rhizoctonia* were separated into four groups (Groups I – IV) with strong bootstrap support. Isolates in Groups I and IV tended to be weakly virulent, while those in Group II caused the highest disease severity.

Evaluation of a bacterium on plant growth promotion and disease control in greenhouse vegetables. J. Yang (J. Y.) and Y. He (Y. H.). *(J. Y.) Alberta Innovates - Technology Futures, Vegreville, AB. T9C 1T4; (Y.H.) Yunnan Agricultural University, Kunming, Yunnan, China, 650201.*
A bacterium of *Bacillus* sp. strain P3 was evaluated for its disease control and plant growth promoting properties *in vitro* and in the greenhouse trials. In the

in vitro bioassay, the bacterium could inhibit the growth of *Botrytis* sp., *Didymella* sp., *Fusarium* spp., and *Sclerotinia* sp., but less effect on *Pythium* spp. The cell-free filtrate also inhibited the growth of these fungi, the mycelial growth reduction ranged from 3.52% (*Pythium*) to 35.8% (*Sclerotinia*) compared with the control. Cucumber and lettuce plants treated with this bacterial cell suspension had higher fresh shoot weight, higher dry root weight in a hydroponic system with recirculated nutrient solution. Lettuce plants treated with the bacterium also showed less Botrytis infection. In a greenhouse trial with open hydroponic system, cucumber plants treated with the bacterium and inoculated with *Fusarium oxysporum* had significantly less root rot disease compared to the untreated Fusarium-inoculated plants. The results demonstrated that this bacterium could promote root growth and development of plants, and could be a good PGPR candidate and a potential biocontrol agent for greenhouse vegetable production in Canada.

Oxidized silvers: experimental fungicides for control of white mold on dry bean. M.J. Unruh, S. Lepp, R.J. Howard, and M.W. Harding. *(M.J.U., S.L., M.W.H.) Innovotech, Edmonton, AB, T6N 1H1, Canada; and (R.J.H) Crop Diversification Centre South, Alberta Agriculture and Rural Development, Brooks, AB, Canada, T1R 1E6.*
White mold, caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, routinely causes destructive disease outbreaks in dry bean fields in Southern Alberta. Oxidized silver compounds are fungicidal, and have been demonstrated to control diseases such as Ascochyta blight (*Ascochyta rabiei* (Pass.) Labr.) on chickpea (*Cicer arietinum* L.). In this study, we evaluated the potential of oxidized silver compounds to control, or reduce, white mold symptoms on dry edible bean (*Phaseolus vulgaris* L.). We report reductions in white mold disease incidence and severity on bean after foliar applications of silver-based compounds, such as oxysilver nitrate and sodium diperiodatoargentate (III), when applied independently, and as tank mixes with registered, commercially formulated fungicides. Taken together with previous findings, these data suggest that oxidized silver compounds are effective, broad-spectrum fungicides that are frequently compatible with existing, commercially available, fungicide formulations. The use of oxidized silvers in fungicide tank mixes may provide more versatile disease management tools, particularly in areas such as fungicide resistance management, broad-spectrum activity and efficacy enhancement.