**Evaluation of dry bean germplasm and cultivars for partial field resistance and physiological resistance to *Sclerotinia sclerotiorum* (white mould).**

**R.L. Conner, D.L. McLaren, A. Hou and P.M. Balasubramanian.**

## Introduction and Literature review

White mould disease caused by *Sclerotinia sclerotiorum* is a highly destructive fungal disease of dry bean. Symptoms of white mould of bean include wilting, water-soaked lesions, bleached stems and pods, the presence of sclerotial bodies in infected tissue and plant death (Kolkman and Kelly, 2002). Development of white mould in dry bean is highly influenced by weather conditions and cultural practices (Schwartz et al., 2004). High plant populations, narrow row widths, vigorously vining varieties, excess fertilization and abundant irrigation/rainfall all favour the development of white mould. High humidity and a moist plant canopy and soil surface are necessary for the fungus to spread.

White mould may cause severe losses in seed yield and quality. Yield losses in irrigated bean fields in western Nebraska averaged 13% due to white mould infection (Kerr et al., 1978). Plants severely infected by *S. sclerotiorum* sustained a decrease in seed yield of 44%. Yield losses in commercial bean fields in southern Alberta range between 6 and 15% (Mr. Jim Rex, Field Representative, Viterra, Personal Communication to Dr. P.M. Balasubramanian). Few registered fungicides offer effective control of white mould in dry bean. Lance, a protectant fungicide, sprayed on plants prior to fungal infection is an expensive option for producers ($52 per acre for two applications). Identification of bean germplasm and/or cultivars with both partial field resistance and partial physiological resistance is the first step in the development of dry bean cultivars with enhanced white mould resistance.

Resistance to white mould is under complex genetic control, and includes avoidance (i.e., partial field resistance) and partial physiological resistance (Miklas et al., 2001). Complete resistance to white mould is lacking in dry bean, but lines with partial resistance to *S. sclerotiorum* isolates from the USA have been identified (Steadman et al., 1998; Miklas et al., 1998, 1999, 2007; Kolkman and Kelly, 2000). Disease severity and incidence data from inoculated disease nurseries indicate that some of the Canadian dry bean cultivars may have partial field resistance to white mould. Partial field resistance observed in bean cultivars may be attributed to disease avoidance traits such as a tall, upright growth habit and an open canopy. However, the level of partial physiological resistance of these dry bean cultivars is unknown.

A combination of partial field resistance and partial physiological resistance is essential for dry bean cultivars to withstand moderate to high white mould disease pressures encountered during cool, wet growing seasons.

## Objectives

To identify dry bean lines with partial field resistance and/or partial physiological resistance to Canadian isolates of *S. sclerotiorum* for use as parents in the dry bean breeding program, and 2) to develop hybridization strategies to pyramid white mould resistance genes into dry bean cultivars for enhanced disease resistance.

## Methodology

Disease-free seeds of 25 dry bean genotypes were increased in the greenhouse at AAFC-Lethbridge, a winter nursery in Chile and a summer nursery in Idaho. These seeds were used in the experiments to determine partial field resistance and partial physiological resistance of dry bean genotypes to white mould.

Partial Field Resistance: Prior to planting, the plot area of the disease nurseries were inoculated with sclerotia from the fungal pathogen *S. sclerotiorum*. Dry bean genotypes were planted in

disease nurseries at Lethbridge, AB, and Winkler and Brandon, MB. Each plot consisted of four rows with 23 or 30 cm row spacing, and 5 m row length depending on the equipment used for planting. The experimental design was a randomized complete block with six replications. Plots were irrigated as required especially from the onset of flowering until maturity. At maturity, each plant in the middle two rows of a plot were rated on a scale of 1 to 4, where 1 = healthy; 2 = single stem infected; 3 = multiple stems infected; and 4 = lower part of main stem infected or dead plant. White mould incidence in each plot was determined as percentage of plants infected, and disease severity (DS) was determined as the average of the disease plants ratings, using the formula $DS = \Sigma(nr)/t$, where n = number of plants, r = plant rating (1 to 4 scale), and t = total number of plants rated.

Partial Physiological Resistance: Dry bean genotypes were planted in growth chambers at AAFC-Lethbridge. Three seeds per genotype were planted in 1-gallon pot, and after emergence, the seedlings were thinned to two per pot. The experimental design was a randomized complete block with seven replications over time. Plants were grown at 23$^{o}$C day/18$^{o}$C night temperatures with a 16 h photoperiod. The *S. sclerotiorum* isolate collected from an infected sunflower field in Alberta were grown on Potato Dextrose Agar medium for 48 h prior to inoculation. At flowering (5 weeks after planting), the growing tip of the main stem was removed and an Eppendorf pipette tip containing an agar plug of actively growing mycelium of the pathogen was fitted over the cut stem. The white mould reaction was scored at 16 and 26 days after inoculation using a Modified Petzoldt and Dickson scale of 1 to 9 (Terán et al. 2006). Data from both the field and controlled environment experiments were subjected to the analysis of variance using SAS.

**Results and Discussion**

Partial Field Resistance: The white mould disease incidence data from the three nurseries each in 2009, 2011 and 2012, and from the Winkler nursery in 2010 (Lethbridge and Brandon nurseries were lost due to waterlogging in 2010) indicate the differences between dry bean genotypes for disease incidence and severity were significant and fairly consistent.

Partial Physiological Resistance: This study was completed in 2010. The differences in disease severity between dry bean genotypes were significant at 16 and 26 days after inoculation. White mould disease severity rating for all of the genotypes increased over time.

The dry bean cultivars developed in Canada lacked both partial field and partial physiological resistances when grown under high disease pressure. This study determined that the germplasm lines A195, G122, I9365-31, and L 192 expressed both partial field and physiological resistance.

**Next Steps**

White mould resistant genotypes are currently being used as parents in the AAFC dry bean breeding program for the development of future cultivars with enhanced resistance to white mould. In 2012, $F_3$ lines were grown in the field at Lethbridge and Vauxhall, AB and single plant selections were made in the fall based on lodging resistance, yield potential (number and distribution of pods) and early maturity. The plants were threshed and seed quality was assessed on $F_4$ seeds in the winter months. Each year, high yielding, early maturing lines will be advanced in the dry bean breeding program for the development of future cultivars. Partial field resistance and partial physiological resistance will be assessed starting in $F_7$ and subsequent generations. A scientific manuscript describing this study is currently being prepared.

**Acknowledgements**

We thank Waldo Penner and Dennis Stoesz at AAFC-Morden, Danny Hausermann at AAFC-Brandon and Cathy Daniels, Cheryl Chelle-Anderson, John Braun and Todd Reid at AAFC-Lethbridge for their technical assistance, and the Alberta Pulse Growers Commission, Manitoba Pulse Growers Association, the Growing Forward I program of AAFC and Alberta Crop Industry Development Fund for research funding. We also thank the dry bean researchers in Canada and the USA for providing seeds of some of the bean genotypes used in this study.

**References**

Kerr, E.D., J.R. Steadman and L.A. Nelson. 1978. Estimation of white mold disease reduction of yield and yield components of dry edible beans. Crop Sci. 18:275-279.

Kolkman, J.M. and J.D. Kelly. 2000. An indirect test using oxalate to determine physiological resistance to white mold in common bean. Crop Sci. 40:281-285.

Kolkman, J.M. and J.D. Kelly. 2002. Agronomic traits affecting resistance to white mold in common bean. Crop Sci. 42:693-699.

Miklas, P.N., R. Delorme, R. Hannan and M.H. Dickson. 1999. Using a subsample of the core collection to identify new sources of resistance to white mold in common bean. Crop Sci. 39:569-573.

Miklas, P.N., K.F. Grafton, J.D. Kelly, J.R. Steadman and M.J. Silbernagel. 1998. Registration of four white mold resistant dry bean germplasm lines: I9365-3, I9365-5, I9365-31 and 92BG-7. Crop Sci. 38:1728.

Miklas, P.N., W.C. Johnson, R. Delorme and P. Gepts. 2001. QTL conditioning physiological resistance and avoidance to white mold in dry bean. Crop Sci. 41:309-315.

Miklas, P.N., K.L. Larsen, K.A. Terpstra, D.C. Hauf, K.F. Grafton and J.D. Kelly. 2007. QTL Analysis of ICA Bunsi-derived resistance to white mold in a pinto x navy bean cross. Crop Sci. 47:174-179.

Schwartz, H.F., M.A. Brick, R.M. Harveson and G.D. Franc. 2004. Disease Management, pp. 109-143. In: Dry bean production and pest management. APS Press, Minneapolis, MN.

Steadman, J.R., K.F. Grafton, K. Kmiecik, J.M. Kolkman, M.K. Jahn and R. Mainz. 1998. Bean white mould nursery 1997. Annu. Rep. Bean Improv. Coop. 41:173-174.

Terán, H., M. Lema, H.F. Schwartz, R. Duncan, R. Gilbertson and S.P. Singh. 2006 Modified Petzoldt and Dickson scale for white mold rating of common bean. Annu. Rep. Bean Improve. Coop. 49:115-116.