SCREENING OF THE USDA CORE COLLECTION OF COMMON BEAN FOR REACTION TO HALO BLIGHT AND IDENTIFICATION OF GENOMIC REGIONS ASSOCIATED WITH RESISTANCE

A Dissertation
Submitted to the Graduate Faculty
of the
North Dakota State University
of Agriculture and Applied Sciences

By

Kiran Ghising

In Partial Fulfillment of the Requirements
for the Degree of
DOCTOR OF PHILOSOPHY

Major Department:
Plant Sciences

July 2016

Fargo, North Dakota

North Dakota State University
Graduate School

SCREENING OF THE USDA CORE COLLECTION OF COMMON BEAN FOR REACTION TO HALO BLIGHT AND IDENTIFICATION OF GENOMIC REGIONS ASSOCIATED WITH RESISTANCE

By

Kiran Ghising

The Supervisory Committee certifies that this ***disquisition*** complies with North Dakota State University's regulations and meets the accepted standards for the degree of

DOCTOR OF PHILOSOPHY

SUPERVISORY COMMITTEE:

Dr. Juan M. Osorno
Chair

Dr. Ted Helm

Dr. Kevin McPhee

Dr. Julie Pasche

Dr. Phil McClean

Approved:

9/26/2016
Date

Dr. Richard D. Horsley
Department Chair

ABSTRACT

With only three sources of resistance currently known to race 6 of *Pseudomonas syringae* pv. *phaseolicola* (Burkholder) (*Psp*) which causes halo blight, an important bacterial disease of common bean, there is an urgent need to identify additional sources of resistance. Therefore, 283 accessions of common bean from the USDA-NPGS core collection were evaluated for resistance to race 6 of *Psp* under greenhouse conditions. Using unifoliate leaf inoculation method, a total of 13% of accessions were resistant. Five of these accessions, PI 201329, PI 309810, PI 310826, PI 319592, and PI 533259, displayed the highest levels of resistance with mean halo blight score of 1.1. Unifoliate vs trifoliate inoculation methods were also evaluated. Significantly higher mean (4.0) and range (1.0-7.0) of halo blight severity was observed at trifoliate stage compared to unifoliate stage, 2.0 and 1.0-2.4, respectively. A significant positive but weak correlation (r^2=0.17) of halo blight severity between trifoliate and pod inoculation methods within an individual plant suggests that disease resistance may be controlled by independent genes prevalent at each plant developmental stage. Halo blight severity observed in trifoliate leaves and pods under greenhouse condition was later validated under field condition. Significantly higher mean disease score and range of 4.7 and 2.3-7.1 were reported at pod stage compared to 3.6 and 2.0-6.6, respectively, at trifoliate stage. However, PI 313217 showed consistent resistant reaction across all plant development stages, i.e., unifoliate, trifoliate, and pod, under both field and greenhouse conditions. A significant but weak correlation (r^2=0.21) between halo blight severity in trifoliate leaves and pods under field condition confirmed the greenhouse results. To identify genomic regions associated with resistance to race 6 of *Psp*, genome-wide association mapping study (GWAS) was employed using 197 accessions and 4707 single nucleotide polymorphism (SNP) markers. Three significant regions were identified, of which two novel regions in Pv04

and one in Pv05 controlled for 19% of the phenotypic variation. The significant SNPs could be used in marker assisted selection (MAS) for the improvement of common bean breeding program with focus on resistance to race 6 of *Psp*.

ACKNOWLEDGEMENTS

First, I would like to thank my advisor, Dr. Juan M. Osorno, for his continuous support, encouragement, and incredible advice in my Ph.D. research. Without his guidance, this achievement would have been impossible. I also would like to express my sincere gratitude to my committee members: Drs. Julie Pasche, Kevin McPhee, Phil McClean, and Ted Helms, for their kind cooperation, constructive suggestions and guidance during my Ph.D. research.

I express sincere thanks to staff, Jody Vander Wal, and Michael Kloberdanz, and colleagues, Angela M. Linares-Ramírez, José E. Vasquez, Chiti Agrawal, and Raphael Colbert of the Dry Bean Breeding Program at NDSU for their continuous support and kindness help in all the activities during the completion of my research. I am also thankful to interns, Carlos Velasquez Villegas from CIAT, Cali, Colombia, and Edgar Escobar Romero from EARTH University, Costa Rica, for helping me in the field as well as greenhouse disease screening process. I wish to express my sincere gratitude to Drs. Samira Mafi Moghaddam, Ali Soltani, and Stephan Schroder for their kind help in association mapping analysis. I extend my sincere thanks to Drs. Jim Hammond, and Jawahar L. Jyoti and Mr. Curt Doetkott for helping me in statistical analysis. My utmost gratitude goes to Mr. Robin Lamppa from dry bean and pulse disease laboratory at NDSU, Department of Plant Pathology for providing disease inoculum for the study.

I have great appreciation for United States Department of Agriculture-National Plant Germplasm System (USDA-NPGS), National Crop Germplasm Committee and *Phaseolus* Genetics Committee for providing financial support to my Ph.D. research. I am also thankful to Northarvest Bean Growers Association for additional financial support for my research.

Last, I extend my sincere respect to my father, Mr. Kirta Ram Ghising, and mother, Mrs. Sun Maya Ghising, for their love, care and support my stay away from home. Finally, I can't forget the love, tireless support, sacrifice, and encouragement of my beloved wife, Renu Singh Maharjan, and daughter, Sheyanne Ghising.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDIX TABLES

LIST OF APPENDIX FIGURES

INTRODUCTION

Halo blight, caused by *Pseudomonas syringae* pv. *phaseolicola* (Burkholder), hereafter referred to as *Psp*, is an important endemic seed-borne bacterial disease affecting common bean production. The disease is prevalent in moderately cool and wet regions of bean growing countries in the world including Asia, Africa, Europe, Central America, South America, and North America (Saettler, 2005; Schwartz, 1989; Miklas et al., 2011). Upon favorable environmental conditions, halo blight has been reported to cause up to 45% yield loss in susceptible cultivars (Singh and Schwartz, 2010). Despite the application of traditional approaches for disease management such as chemical control, the use of pathogen-free seeds, crop rotation and crop sanitation, i.e., the selection and sowing of healthy plant parts, the use of disease resistant cultivars remain one of the important and efficient disease management approaches including for halo blight (Fry, 2012; Singh and Muñoz, 1999). Currently, nine races of *Psp* have been identified based on the series of eight differential *Phaseolus* genotypes, of which race 6 is one of the most virulent and prevalent race worldwide (Taylor et al., 1996a, 1996b).

To date, one of the common quantitative trait loci (QTL) mapping approaches, bi-parental mapping has successfully identified both race-specific (quantitative) as well as non-race specific (qualitative) resistance to all nine *Psp* races except for race 6 (Taylor et al., 1996a, 1996b; Miklas and Fourie et al., 2006; Miklas et al., 2009; 2011; 2014). Five putative major R genes named as *Pse-1*, *Pse-2, Pse-3, Pse-4* and *Pse-5* have been identified based on interaction with a set of eight differential cultivars and exhibit gene-for-gene interaction (Taylor et al., 1996b). Similarly, non-race specific resistance to all races of *Psp* have been reported in GN Nebraska # Sel. 27, PI 150414, 'Jules', CAL 143, and Wis HBR 72 (G 3954), but not for race 6

(Taylor et al., 1996b). Nonetheless, Duncan et al., (2008; 2014) recently developed US14HBR6, a pinto bean germplasm resistant to race 6 of *Psp*, where the resistance is controlled by two independently inherited recessive genes developed from the original stock of pinto US 14. In addition, the more recent report of significant QTL on Pv04 and Pv06 governing resistance to races 6 and 7 of *Psp* was analyzed in RILs derived from a bi-parental population of Xana (susceptible) and Cornell 49242 (resistant) (Trabanco et al., 2014). Porch et al. (2016), using an Andean Diversity Panel (ADP), detected a significant QTL on PV05 that controlled resistance to race 6 of *Psp*, which was later validated in a bi-parental RIL population derived from a cross of Rojo (susceptible) and CAL 143 (resistant).

Because QTL identified from bi-parental population is based upon limited number of recombination events and narrow allelic diversity, the use of genetically diverse populations using genome-wide association study (GWAS) is widely implemented as an alternative approach (Huang and Han, 2014). Ideally, GWAS takes an advantage of low linkage disequilibrium (LD) blocks in those genetically diverse panels of core-collection, which is possible due to higher frequency of recombination over the generations (Zhu et al., 2008). In common bean, the recent development of BARCBean6K_3 BeadChip with approximately 6000 SNP markers using whole genome sequence have aided in making inferences on positional candidate genes due to enhanced resolution (Hyten et al., 2010; Schmutz et al., 2014; Song et al., 2015). Using the Andean diversity panel (ADP), Kamfwa et al. (2015) provided insight into the genetic architecture of 10 important agronomic traits on common bean related to phenology, biomass, yield components, and seed yield. Shi et al. (2011) identified significant SNP markers associated with QTLs for common bacterial blight (CBB) resistance in a population of approximately 400 common bean genotypes. However, due to the limited number of SNPs (77) used in the study,

strong association of candidate genes with identified QTLs were difficult to interpret. However, no study has yet evaluated USDA-NPGS (United States Department of Agriculture-National Plant Germplasm System) common bean core collection for the identification of additional sources of resistance to race 6 of *Psp*. More important, no study has yet evaluated the core collection to report the genomic regions associated with the resistance to race 6 of *Psp* for potential use in marker-assisted selection (MAS). Therefore, this study has following objectives:

i. To identify resistant germplasm to race 6 of *Psp* from the USDA-NPGS bean core-collection in the greenhouse,
ii. To identify the most appropriate leaf stage, i.e., unifoliate versus trifoliate, for the evaluation of race 6 of *Psp*,
iii. To assess differences in reactions and levels of disease resistance, if any, upon inoculating leaves vs. pods within an individual plant,
iv. To evaluate a group of selected accessions for resistance to race 6 of *Psp* under field conditions, and
v. To identify genomic regions linked to resistance to race 6 of *Psp* using a GWAS.

References

Duncan, R.W., M. Lema, R.L. Gilbertson, and S.P. Singh. 2014. Registration of common bean pinto US14HBR6 resistant to race 6 of the halo blight pathogen, *Pseudomonas syringae* pv. *phaseolicola*. J. Plant Reg. 8:53-56.

Duncan, R.W., S.P. Singh, and R.L. Gilbertson. 2008. Identification and inheritance of a new source of halo blight resistance in common bean. Annu. Rep. Bean. Improv. Coop. 51:48-49.

Fry, W.E. 2012. Introduction to disease management. In: W.E. Fry, editor, Principles of plant disease management. Academic Press, New York/London. p. 2-9.

Huang, X., and B. Han. 2014. Natural variations and genome-wide association studies in crop plants. Annu. Rev. Plant Biol. 65:531-551.

Hyten, D.L., Q. Song, E.W. Fickus, C.V. Quigley, J.S. Lim, I.Y. Choi, E.Y. Hwang, M. Pastor-Corrales, and P.B. Cregan. 2010. High-throughput SNP discovery and assay development in common bean. BMC Genomics 11:475-483.

Kamfwa, K., K.A. Cichy, and J.D. Kelly. 2015. Genome-wide association study of agronomic traits in common bean. The Plant Genome 8:1-12.

Miklas, P.N., and D. Fourie. 2006. Halo blight resistance in host differential cultivar ZAA 12 is conditioned by three major gene loci. Annu. Rep. Bean Improv. Coop. 49:69-70.

Miklas, P.N., D. Fourie, J. Trapp, J. Davis, and J.R. Myers. 2014. New loci including conferring resistance to halo bacterial blight on chromosome Pv04 in common bean. Crop Sci. 54:2099-2108.

Miklas, P.N., D. Fourie, J. Trapp, R.C. Larsen, C. Chavarro, M.W. Blair, and P. Gepts. 2011. Genetic characterization and molecular mapping gene for resistance to halo blight in common bean. Crop Sci. 51:2439-2448.

Miklas, P.N., D. Fourie, J. Wagner, R.C. Larsen, and C.M.S. Mienie. 2009. Tagging and mapping *Pse-1* gene for resistance to halo blight in common bean differential cultivar UI-3. Crop Sci. 49:41-48.

Porch, T.G., K. Cichy, M.A. Pastor-Corrales, M.A. Grusak, J.S. Beaver, J. Hart, D. Fourie, C. Estevez de Jensen, S. Nchimbi, and P.N. Miklas. Characterization and application of the Andean Diversity Panel for the improvement of common bean productivity in Sub-Saharan Africa. Poster session presented at the meeting of the Pan-African Grain Legume & World Cowpea Conference, 2016 February 28 – March 4, Livingstone, Zambia.

Saettler, A.W. 2005. Halo Blight. In: H.F. Schwartz et al., editors, Compendium of bean diseases. APS Press, St. Paul, MN. p. 49-50.

Schmutz, J., P.E. McClean, S. Mamidi, G.A. Wu, S.B. Cannon, J. Grimwood, J. Jenkins, S. Shu, Q. Song, C. Chavarro, M. Torres-Torres, V. Geffroy, S.M. Moghaddam, D. Gao, B. Abernathy, K. Barry, M. Blair, M.A. Brick, M. Chovatia, P. Gepts, D.M. Goodstein, M. Gonzales, U. Hellsten, D.L. Hyten, G. Jia, J.D. Kelly, D. Kudrna, R. Lee, M.M.S. Richard, P.N. Miklas, J.M. Osorno, J. Rodrigues, V. Thareau, C.A. Urrea, M. Wang, Y. Yu, M. Zhang, R.A. Wing, P.B. Cregan, D.S. Rokhsar, and S.A. Jackson. 2014. A reference genome for common bean and genome-wide analysis of dual domestications. Nat. Genet. 46:707-713.

Schwartz, H.F. 1989. Halo blight. In: H.F. Schwartz and M.A. Pastor-Corrales, editors. Bean production problems in the tropics. 2nd ed. CIAT, Cali, Colombia. p. 285-301.

Shi, C., A. Navabi, and K. Yu. 2011. Association mapping of common bacterial blight resistance QTL in Ontario bean breeding populations. BMC Plant Biol. 11:52.

Singh, S.P., and C.G. Muñoz. 1999. Resistance to common bacterial blight among *Phaseolus* species and common bean improvement. Crop Sci. 39:80-89.

Singh, S.P., and H.F. Schwartz. 2010. Breeding common bean for resistance to diseases: A review. Crop Sci. 50:2199-2223.

Song, Q., G. Jia, D.L. Hyten, J. Jenkins, E.Y. Hwang, S.G. Schroeder, J.M. Osorno, J. Schmutz, S.A. Jackson, P.E. McClean, and P.B. Cregan. 2015. SNP assay development for linkage map construction, anchoring whole genome sequence, and other genetic and genomic applications in common bean. G3: Genes|Genomes|Genetics 5:2285-2290.

Taylor, J.D., D.M. Teverson, M.A. Allen, and M.A. Pastor-Corrales. 1996a. Identification and origin of races of *Pseudomonas syringae* pv. *phaseolicola* from Africa and other bean growing areas. Plant Pathol. 45:469-478.

Taylor, J.D., D.M. Teverson, and J.H.C. Davis. 1996b. Sources of resistance to *Pseudomonas syringae* pv. *phaseolicola* races in *Phaseolus vulgaris.* Plant Pathol. 45:479-485.

Trabanco, N., M.C. Asensio-Manzanera, E. Perez-Vega, A. Ibeas, A. Campa, and J.J. Ferreira. 2014. Identification of quantitative trait loci involved in the response of common bean to *Pseudomonas syringae* pv. *phaseolicola*. Mol. Breeding 33:577-588.

Zhu, C., M. Gore, E.S. Buckler, and J. Yu. 2008. Status and prospects of association mapping in plants. The Plant Genome 1:5-20.

LITERATURE REVIEW

Origin, Evolution and Domestication of *Phaselous* Species

Present-day cultivated common bean cultivars evolved from its closest relative, i.e., wild common bean, more than 165,000 years ago, and has been domesticated independently in two large geographic gene pools distributed in Mesoamerican, also known as Middle American (from Central America and Mexico) and Andean (from Andes Mountains of South America, i.e., Peru, Chile, Bolivia and Argentina to northwestern Argentina) gene pools (Gepts and Debouck, 1991; Gepts, 1998; Singh et al., 1991b; Beebe et al., 2000). Some distinguishing features among the two gene pools include seed and pod size, seeds per pod, bracteoles, internode length and flowering time (Gepts and Debouck, 1991; Singh et al., 1991b; Gepts, 1998). For example, Andean genotypes are early maturing types with bigger seed and pod size compare to Mesoamerican as late maturing with small seed and pod size (Gepts and Debouck 1991; Singh et al., 1991b; Schmutz et al., 2014).

The Mesoamerican gene pool consists of three races, i.e., Durango, Jalisco, and Mesoamerican, whereas Andean gene pool consists of three races, i.e., Nueva Granada, Peru and Chile (Singh and Debouck, 1991a, Singh et al., 1991b). Race Durango comes from central highlands of Mexico, Jalisco from coastal Mexico, and Mesoamerican from lowland tropical Central America. Market classes such as pinto, great northern (GN), small red and pink comprises of race Durango, while Mesoamerica race includes navy and black beans. Besides black (*negros*), pintos, pink (*rosas/claros*), and yellow (*amarillos*), ranked as top four bean market class in Mexico based on total consumption, Flor de Mayo and Flor de Junio are considered two most important and common varieties of pink beans belonging to race Jalisco that are predominant and native to highlands of Central Mexico (Zahniser et al., 2010). Similarly,

natural habitat of three races from Andean gene pool namely, Nueva Granada, Peru, and Chile can be traced back to Colombia and Ecuador, highlands of Peru, and northern Chile and Argentina, respectively (Singh et al., 1991a). Pre-dominant market classes such as light red kidney (LRK), dark red kidney (DRK), white kidney (WK), and cranberry beans represents race Nueva Granada. Yellow beans such as Mayacoba and Canario represents race Peru, whereas race Chile includes the vine cranberry beans and bean types distinctive to Chile (Coscorron and Tortola) (Kelly, 2010).

Common Bean Production in the United States

The global production data of dry beans by the Food and Agriculture Organization (FAO) is misrepresentative of true production statistics as category 'dry beans' includes all species of *Phaselous* and *Vigna* (FAOSTAT, 2014). Therefore, if only *P. vulgaris* is accounted for, United States is ranked as sixth largest producer after Brazil, Mexico, Tanzania, Uganda, and Kenya. In addition, the sole economic value of common bean always exceeds the combined economic value of other legumes including chickpea (*Cicer arietinum* L.), lentils (*Lens culinaris* Medikus), pea (*Pisum sativum* L.), and cowpea, and is thus considered a key grain legume (Porch et al., 2013).

The commercial production of dry bean in the United States of America (USA) started as early as in 20th century, currently growing in more than 30 states, and contributing a large portion to export market. In 2014 in USA, common beans were planted on approximately 0.6 million hectares with an annual production of about 1.4 million metric tons (MT), with an average seed yield of approximately 2000 kg ha^{-1} (USDA-NASS, 2014). If dry edible bean production from both North Dakota and Minnesota is considered collectively, they contribute about 50% of the total production in the United States followed by Michigan (16%), Nebraska

(13%), Idaho (8%), Washington (7%), and California (3%), respectively (USDA-NASS, 2014). In North Dakota, dry bean was first introduced in early 1970s, and since then, it has been one of the major commercially grown crop in the North Dakota-Minnesota region (Berglund, 1997). Since early 1990s, North Dakota has been the leading state for dry bean production. The production data in North Dakota from past eleven subsequent years, i.e., from year 2003 to 2013, estimated an average of 325,000 MT of pinto bean, and 83,000 MT of navy bean each year from an average of 250,000 ha planted per year (USDA-NASS, 2014). In 2014, North Dakota produced approximately 0.4 million MT of dry beans planted from a total of about 255,000 ha. North Dakota remains as the leading dry bean producing state with an average yield of 1795 kg ha^{-1} from the total of 250,000 ha harvested (USDA-NASS, 2014). Of the total production of 0.4 million MT of dry beans, pinto is the predominant market class with the production of about 0.3 million MT, followed by navy (82,000 MT), and black (50,000 MT) (USDA-NASS, 2014). Meanwhile in Minnesota, the total production was 0.1 million MT with an average of 2180 kg ha^{-1} where navy ranks first (44,000 MT) in terms of production followed by DRK (40,000 MT), black (24,000 MT), LRK (18,000 MT), and pinto (7,200 MT) (USDA-NASS, 2014). However, if all kidney types are considered, it is the most important market class in Minnesota, making this state the largest producer of kidney beans in the country.

Genetic Markers in Common Bean Breeding

With an advent of molecular (genetic) markers, common bean breeders have successfully able to exploit genetic variability found in different gene pools, races, and market classes of cultivated common bean for the identification of superior genes related to several biotic as well as abiotic stresses (Miklas et al., 2006; Beaver and Osorno, 2009; Singh and Schwartz, 2010). To date, molecular markers such as Restriction Fragment Length Polymorphisms (RFLPs),

Random-Amplified Polymorphic DNA (RAPD), Amplified Fragment Length Polymorphism (AFLPs), Simple Sequence Repeats (SSRs), Sequence Characterized Amplified Regions (SCAR), Expressed Sequence Tags (ESTs), insertion-deletions (InDel), KASPar (Kompetitive Allele Specific PCR) method, and more recently, Single Nucleotide Polymorphisms (SNPs) have been used extensively for linkage map construction, synteny analysis, genetic diversity study as well as for the identification of major genes/QTLs linked to several economically important traits in common bean (Adam-Blondon et al., 1994; Chen et al., 2014; Cortés et al., 2011; Freyre et al., 1998; Galeano et al., 2012; Hyten et al., 2010; Kamfwa et al., 2015; Miklas et al., 2002; Moghaddam et al., 2014; Perseguini et al., 2016; Schmutz et al., 2014; Song et al., 2015).

Importance of Landraces (Germplasm) and Genetic Diversity

One of the major drawbacks of mapping QTL in elite population is the existence of limited allelic diversity (Concibido et al., 2003; Guzman et al., 2007). This brings the need for plant breeders today to explore for genetically diverse germplasm or landraces, and its potential use in breeding programs. This approach help broadens the genetic base as well as maximizes the knowledge of novel allelic diversity present in the landraces that may be beneficial to several economically important traits.

Germplasm banks play an important role in terms of preserving the crop genetic variability especially the one with narrow genetic bases (Broughton et al., 2003). Because the study and evaluation of large number of accessions in germplasm collection is not always feasible due to insufficient funding and time, the concept of core-collection was developed where a subset of accessions, i.e., small percentage of accessions, maximum enough to represent genetic variability of the original base collection was developed (Frankel and Brown 1984; van Hintum et al., 2000). The three principal steps include to develop core-collection (a) determine

the specific size of subset of accessions, (b) divide original base collection into distinct group, and (c) assign each selected accession in each group that develops into core-collection. Therefore, a core-collection is a more comprehensive set of germplasm that represents both phenotypic as well as genotypic variation with minimum redundancy (Blair et al., 2009).

More than 29,000 domesticated and 1,300 wild accessions of *P. vulgaris* are stored in CIAT (International Center for Tropical Agriculture), while 17,610 accessions are stored in the National Plant Germplasm System (NPGS), which provides enough genetic variation to discriminate resistance versus susceptible reactions to common bean pathogens. However, unlike in CIAT collection, a limited number of studies have been evaluated the USDA-NPGS core collection for the identification of genetic variation essential for common bean improvement (Brick et al., 2006; McClean et al., 2012; Miklas et al., 1999).

Halo Blight, Disease Cycle, and Symptoms

Halo blight, a serious seed-borne bacterial disease of common bean, is caused by *Pseudomonas syringae* pv. *phaseolicola* (*Psp*) that produces a phytotoxin known as phaseolotoxin [(N^{δ}-phoaphosulphamyl) omithylalanylhomoarginine] along with minimal amount of an analogue (2-serine) -phaseolotoxin [(N^{δ}-phosphosulphamyl) - ornithylserylhomoarginine], and are associated with disease symptoms of leaf chlorosis in plant. Alternate hosts of *Psp* include adzuki bean (*Vigna angularis* (Willd). Ohwi & H. Ohashi), lima bean (*Phaseolus lunatus* L.), mung bean (*V. radiata* (L.) R. Wilczek), scarlet runner bean (*P. coccineus* L.), soybean (*Glycine max* (L.) Merr.), and tepary bean (*P. acutifolius* A. Gray). The disease is primarily seed borne, and the bacteria can survive in the contaminated seeds and infected plant tissues as well as in crop residue that serve as an inoculum for disease development. The bacteria from infected

plant residue may survive up to 1 year, and spread to healthy plants by splattering water or wind-blown rain, overhead irrigation water, and agricultural equipment (Schwartz et al., 2005).

Under favorable environment, bacterium enters the plant through natural openings like stomata and hydathodes. Initially, the bacterium causes infection on the lower leaf surface that initially appears as small water-soaked spots after several days of infection. The spots gradually develop on upper and lower leaf surfaces as necrotic spots (about 1 to 2 mm in diameter) that are surrounded by a characteristic chlorotic halo, a distinguished symptom of the disease. A chlorotic zone of yellow-green tissue resembles a halo that may appear around necrotic spots, which under severe infection may develop systemic chlorosis in the plant. A moderate to cool temperature range of 18 to 23°C aided with moist conditions trigger the production of the phaseolotoxin that develops more chlorotic symptoms and favors halo development (Hagedorn and Inglis, 1986; Mitchell, 1978; Schwartz and Pastor-Corrales, 1989), whereas with temperature above 23°C the production of the phaseolotoxin is reduced and thus inhibits halo formation. A greasy, water-soaked appearance is seen in the lesions after 7 to 10 days after infection, which results from the bacterial ooze from substomatal cavities. The disease infection is often extended to stems and pods, where a symptom in the pod is characterized by the presence of red or brown water-soaked lesions. As the pod matures, these water-soaked lesions in the pod change from yellow to tan in color exhibiting crusty bacterial ooze on the surface, and may easily result in severe loss of the marketable quality of the product (Hagedorn and Inglis, 1986; Schwartz et al., 2005).

Genetic Variability of *Psp*

The pathogen is variable, and currently nine races of the pathogen have been identified based on a set of eight differential cultivars (Taylor et al., 1996a; Schwartz et al., 2005) (Table

1). Taylor et al. (1996a) determined the race of 175 isolates of *Psp* collected from different geographical regions, and identified five out of nine races, i.e., races 1, 2, 5, 6 and 7, that were commonly distributed worldwide, with race 6 being the most frequent. A study conducted in Spain identified six different races of *Psp* such as, 1, 2, 5, 6, 7, and 9, where the race 6 was second most dominant race found after race 7 (Rico et al., 2003). Similarly, Lamppa et al. (2002), identified race 6 as most common race in the bean growing regions of North Dakota along with race 2, although the occurrence of the latter race was minimal. However, Fourie et al. (1998), reported the occurrence of seven races (races 1, 2, 4, 6, 7, 8, and 9) of *Psp* bacterium in dry bean producing areas of South Africa, with race 8 being the most prevalent one (Fourie, 1998).

Table 1. Race differentiation of halo blight (*P. syringae* pv. *phaseolicola*) using eight differential genotypes (Taylor et al., 1996a).

Differential	***Pse*-genes**	**Races**								
		1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**
Canadian Wonder	-	+	+	+	+	+	+	+	+	+
A52 (ZAA 54)	4	+	+	+	+	-	+	+	+	+
Tendergreen	3	+	+	-	-	+	+	+	+	+
Red Mexican UI 3	1,4	-	+	+	+	-	+	-	+	-
1072	2	+	-	+	-	-	+	-	+	+
A53 (ZAA 55)	3,4	+	+	-	-	-	+	+	+	+
A43 (ZAA 12)	2,3,4,5	+	-	-	-	-	+	-	-	-
Guatemala 196-B	3,4	-	+	-	-	-	+	-	+	-

+, compatible (susceptible); -, incompatible (resistant)

Genetic Resistance to Halo Blight in Common Bean

Currently, using a set of eight differential cultivars, nine differential races of *Psp* have been identified where race 6 along with races 1, 2, and 7 are predominant in all bean growing regions in the world (Taylor et al., 1996a; 1996b) (Table 1). The complex and virulent nature of

reaction of race 6 of *Psp* to all differential cultivars tested have ranked it the most virulent race of all (Taylor et al., 1996a, 1996b).

Based upon pathogen (*Psp*) and host (*P. vulgaris* L.) interaction, breeding for resistance to *Psp* in the past decades have identified both race-specific (qualitative) and non-race specific (quantitative) sources of resistance (Teverson, 1991; Taylor et al., 1996a; 1996b). Unlike qualitative resistance where major resistance genes are absent, non-race specific resistance is reportedly associated with the presence of major genes (Asensio et al., 1993; Taylor et al., 1996a, 1996b). Few examples of the accessions that showed recessively inherited quantitative resistance to different *Psp* races include great northern Nebraska #1 Sel. 27, 'Jules', PI 150414, and Wis HBR 72 (G 3954), except CAL 143, an Andean breeding line, in which resistance is governed by a single dominant gene (Patel and Walker, 1965; Taylor et al., 1996b; Chataika et al., 2011). However, five race-specific resistance genes, i.e., R genes: *R1* (*Pse-1*), *R2* (*Pse-2*), *R3* (*Pse-3*), *R4* (*Pse-4*), and *R5* (*Pse-5*), to *Psp* have been identified, which follows the gene-for-gene model with dominant mode of action (Taylor et al., 1978; Taylor et al., 1996a; Miklas et al., 2009, 2011). For example, Red Mexican cultivar #3 (also known as UI-3) conferring hypersensitive resistance to race 1 of *Psp* is controlled by a single dominant gene but showed susceptible reaction to race 2 isolates (Taylor et al., 1978). The *Pse-1* gene derived from UI-3 confers resistance to races 1, 5, 7, and 9 with a single dominant inheritance, while a second gene, *Pse-4,* discovered in UI-3 confers resistance to race 5 only (Teverson, 1991). Likewise, Teverson (1991) reported the presence of four different resistance genes namely, *Pse-2, Pse-3, Pse-4*, and *Pse-5* genes in the host differential cultivar ZAA 12 (A43), where resistance gene, *Pse-2*, showed resistance to the races 2, 5, and 7; *Pse-3* for races 3 and 4; *Pse-4* for race 5; and *Pse-5* for race 8. Later, the development of several genetic maps revealed the identification of

molecular markers closely linked to the halo blight resistance genes, and their utility for MAS. Three sequence characterized amplified regions (SCAR) markers (converted from RAPD markers) namely, SH11.800, SR13.1150, and ST8.1350, were developed that were tightly linked (0-3.3 cM) with *Pse-1* gene located in linkage group (LG) Pv10 (Teverson et al., 1991; Miklas et al., 2009). Miklas et al. (2011) also identified the SCAR (Sequence Characterized Amplified Region) marker SAE15.955 tightly linked (0 cM) with *Pse-2* in Pv10. The marker was developed from a recombinant inbred population, ZAA 12 x 'Canadian Wonder', which conferred resistance to race 2, 3, 4, 5, 7, 8 and 9 of *Psp*. Fourie et al. (2004) also identified the three major resistance genes in recombinant inbred lines (RILs) from the cross of BelNeb-RR-1/A55. These genes include *Pse-1* on Pv04, *Pse-3* on Pv02, and *Pse-4* on Pv04, and confers resistance to races 1, 5, 7 and 9, races 3 and 4, and race 5, respectively. Recently, an additional new resistance gene, *Pse-6*, for resistance to races 1, 5, 7, and 9 was reported using same population as Fourie et al., (2004), and was found tightly linked (1.4 cM) to SCAR marker SB10.550 in Pv04 (Miklas et al., 2014).

Because the identification of sources of resistance to all races of *Psp* including races 1, 2, 3, 4, 5, 7, 8, and 9, exist except to race 6, emphasis in identifying and breeding for novel resistance sources to this race is urgent. Chataika et al. (2011), however, identified a single dominant gene governing resistance to *Psp* in an Andean breeding line, CAL 143, from CIAT, which was also released as a commercial variety in different African countries. The study reported resistance to much broader pathogenic variation of *Psp* prevalent in the growing regions of Malawi including races 1, 2, 3 & 4, but lacked additional information claiming resistance to race 6 of *Psp*. However, a recent discovery of a significant QTL governing resistance to race 6 of *Psp* in Andean Diversity Panel (ADP) was later validated in a RIL population of Rojo and CAL

143 (Porch et al., 2016). Similarly, Duncan et al. (2014) recently developed a pinto line, US14HBR6 (Reg. No. GP-293, PI 666939), resistant to race 6 of *Psp.* This pinto line is developed from the US 14 (PI 549748), an original source of resistance to race 6 of *Psp* through selective breeding process under disease stressed environments (Teràn et al., 2009, Duncan et al., 2008). Resistance in US14HBR6 is controlled by two independently inherited recessive genes where each dominant allele at either locus contributed to increase in disease severity index (Duncan et al., 2014). Moreover, this US14HBR6 pinto line to date is only known Mesoamerican line governing resistance to race 6 of *Psp,* and therefore currently holds strong potential as a parent for introgression in the breeding programs.

Association Mapping

Association mapping (AM) has become one of the emerging and successful tools that identify the complex phenotypic traits and marker relationship at the population level (Nordborg and Tavare, 2002). The concept of AM particularly relies on the concept of linkage disequilibrium (LD) or LD mapping (Abdurakhmonov and Abdukarimov, 2008). Linkage disequilibrium is the non-random association of alleles at different loci in a population, and contrasts from linkage equilibrium (LE), which is the random association of alleles at different loci in a population. In other words, LE or linkage refers to the closely located genes in the chromosome due to their physical proximity and is likely to be inherited together, while LD refers to the correlation of alleles in a population (Flint-Garcia, 2003). Unlike bi-parental mapping, LD utilizes diverse populations with conserved natural variations such as germplasm collections, landraces, elite breeding lines, and synthetic populations, to identify marker-trait associations (Breseghello and Sorrells 2006; Adhikari et al., 2011; Al-Maskri et al., 2012). However, based largely on the objective of a study, AM can be conducted in two different ways

(i) candidate-gene association mapping, and (ii) genome-wide association mapping (GWAS), or genome scan, where the former relates in controlling variation for specific traits, while latter focuses in the scanning of whole genome to find the variation for several complex traits (Risch and Merikangas, 1996).

First reported in Oat (*Avena sativa* L.) (Beer et al., 1997), maize (*Zea mays* L.) (Bar-Hen et al., 1995), and rice (*Oryza sativa* L.) (Virk et al., 1996) during late 20th century, AM studies have been conducted in numerous crops such as, barley (*Hordeum vulgare* L.), soybean, wheat (*Triticum spp.*), and potato (*Solanum tuberosum* L.) (Gupta et al., 2005; Zhu et al., 2008). Currently, numerous studies have been conducted to show the association of agronomic traits and the markers linked via the concept of LD mapping. Few examples include the identification of molecular markers associated with flowering time and plant height in maize (Jafar et al., 2012), mildew and leaf rust resistance in barley (Wang et al., 2011), as well as iron chlorosis deficiency in soybean (Zuo et al., 2013). In common bean, the concept of LD based AM has been studied to map reasonable number of markers across the bean genome for several agronomic traits of economic importance, disease resistance genes, as well as to understand the population structure, diversity analysis, evolutionary history and domestication pattern (Blair et al., 2013; Cichy et al., 2015; Galeano et al., 2012; Kamfwa et al., 2015; Mamidi et al., 2011; 2013; McClean et al., 2012; Nemil et al., 2014; Shi et al., 2011).

References

Abdurakhmonov, I.Y., and A. Abdukarimov. 2008. Application of association mapping to understanding the genetic diversity of plant germplasm resources. Int. J. Plant Genomics. 1-18. doi:10.1155/2008/574927

Adam-Blondon, A., M. Dron, and M. Sevignac. 1994. A genetic map of common bean to localize specific resistance genes against anthracnose. Genome 37:915-924.

Adhikari, T., E.W. Jackson, S. Gurung, J. Hansen, and J.M. Bonman. 2011. Association mapping of quantitative resistance to *Phaeosphaeria nodorum* in spring wheat landraces from the USDA national small grains collection. Phytopathology 101:1301-1310.

Al-Maskri, A.Y., M. Sajjad, and S.H. Khan. 2012. Association mapping: A step forward to discovering new alleles for crop improvement. Int. J. Agr. Biol. 141:153-160.

Asensio, C., E. Martin, and J.L. Montoya. 1993. Inheritance of resistance to race 1 of *Pseudomonas syringae* pv. *phaseolicola* in some varieties of beans. Invest. Agrar. Prod. Prot. Veg. 8:445-456.

Bar-Hen, A., A. Charcosset, M. Bourgoin, and J. Guiard. 1995. Relationship between genetic markers and morphological traits in a maize inbred line collection. Euphytica 84:145-154.

Beaver, J.S., and J.M. Osorno. 2009. Achievements and limitations of contemporary common bean breeding using conventional and molecular approaches. Euphytica 168:145-175.

Beebe, S., P.W. Skroch, J. Tohme, M.C. Duque, F. Pedraza, and J. Nienhuis. 2000. Structure of genetic diversity among common bean landraces of Middle American origin based on correspondence analysis of RAPD. Crop Sci. 40:264-273.

Berglund, D. 1997. Dry bean production guide. NDSU Extension Service Publication A-1133.

Blair, M.W., A.J. Cortés, R.V. Penmetsa, A. Farmer, N. Carrasquilla-Garcia, and D.R. Cook. 2013. A high-throughput SNP marker system for parental polymorphism screening, and diversity analysis in common bean (*Phaseolus vulgaris* L.). Theor. Appl. Genet. 126:535-548.

Blair, M.W., L.M. Díaz, H.F. Buendía, and M.C. Duque. 2009. Genetic diversity, seed size associations and population structure of a core collection of common beans (*Phaseolus vulgaris* L.). Theor. Appl. Genet. 119:955-972.

Breseghello F., and M.E. Sorrells. 2006. Association analysis as a strategy for improvement of quantitative traits in plants. Crop Sci. 46:1323-1330.

Brick, M.A., P.F. Byrne, H.F. Schwartz, J.B. Off, K. Otto, A.L. Fall, and J. Gilbert. 2006. Reaction to three races of Fusarium wilt in the *Phaseolus vulgaris* core collection. Crop Sci. 46:1245-1252.

Broughton, W.J., G. Hernandez, M. Blair, S. Beebe, P. Gepts, and J. Vanderleyden. 2003. Beans (*Phaseolus* spp.) --model food legumes. Plant and Soil 252:55-128.

Chataika, B.Y.E., J.M. Bokosi, R.M., Chirwa, and M.B. Kwapata. 2011. Inheritance of halo blight resistance in common bean. African Crop Sci. J. 9:325-333.

Chen, M., J. Wu, L. Wang, X. Zhang, M.W. Blair, J. Jia, and S. Wang. 2014. Development of mapped simple sequence repeat markers from common bean (*Phaseolus vulgaris* L.) based on genome sequences of a Chinese landrace and diversity evaluation. Mol. Breeding 33:489-496.

Cichy, K.A., T.G. Porch, J.S. Beaver, P. Cregan, D. Fourie, R.P Glahn, M.A. Grusak, K. Kamfwa, D.N. Katuuramu, P. McClean, and E. Mndolwa. 2015. A diversity panel for Andean bean improvement. Crop Sci. 55:2149-2160.

Concibido, V.C., B. La Valle, P. Mclaird, N. Pineda, J. Meyer, L. Hummel, J. Yang, K. Wu, and X. Delannay. 2003. Introgression of a quantitative trait locus for yield from *Glycine soja* into commercial soybean cultivars. Theor. Appl. Genet. 106:575-582.

Cortés, A.J., M.C. Chavarro, and M.W. Blair. 2011. SNP marker diversity in common bean (*Phaseolus vulgaris* L.). Theor. Appl. Genet. 123:827-845.

Duncan, R.W., M. Lema, R.L. Gilbertson, and S.P. Singh. 2014. Registration of common bean pinto US14HBR6 resistant to race 6 of the halo blight pathogen, *Pseudomonas syringae* pv. *phaseolicola*. J. Plant Reg. 8:53-56.

Duncan, R.W., S.P. Singh, and R.L. Gilbertson. 2008. Identification and inheritance of a new source of halo blight resistance in common bean. Annu. Rep. Bean. Improv. Coop. 51:48-49.

Flint-Garcia, S.A. 2003. Structure of linkage disequilibrium in plants. Annu. Rev. Plant Biol. 54:357-374.

Food and Agricultural Organization. 2014. FAOSTAT Database. http//www.faostat.fao.org (accessed 15 June 2015).

Fourie, D. 1998. Characterization of HB races on dry beans in South Africa. Plant Dis. 82:307-310.

Fourie, D., P. Miklas, and H. Ariyaranthe. 2004. Genes conditioning halo blight resistance to races 1, 7, and 9 occur in a tight cluster. Annu. Rep. Bean Improv. Coop. 47:103-104.

Frankel, O.H., and A.H.D. Brown. 1984. Plant genetic resources today: A critical appraisal. In: J.H.W. Holden and J.T. Williams, editors, Crop genetic resources: Conservation and evaluation. G. Allen and Unwin, London. p: 249-257.

Freyer, R., P.W. Skroch, V. Geffroy, A.F. Adam-Blondon, A. Shirmohamadali, W.C. Johnson, V. Llaca, R.O. Nodari, P.A. Pereira, S.M. Tsai, J. Tohme, M. Dron, J. Nienhuis, C.E. Vallejos, and P. Gepts. 1998. Towards an integrated linkage map of common bean. 4. Development of an RFLP-based linkage map. Theor. Appl. Genet. 97:847-856.

Galeano, C.H., A.J. Cortés, A.C. Fernández, A. Soler, N. Franco-Herrera, G. Makunde, J. Vanderleyden, and M.W. Blair. 2012. Gene-based single nucleotide polymorphism markers for genetic and association mapping in common bean. BMC Genet. 13:48-58.

Gepts, P. 1998. Origin and evolution of common bean: Past events and recent trends. HortScience 33:1124-1130.

Gepts, P., and D.G. Debouck. 1991. Origin, domestication and evolution of the common beans (*Phaseolus vulgaris* L.). In: A. Schoonhoven and O. Voysest, editors, Common beans: Research for crop improvement. CAB International, Wallingford. p. 7-54.

Gupta, P.K., S. Rustgi, and P.L. Kulwal. 2005. Linkage disequilibrium and association studies in higher plants: Present status and future prospects. Plant Mol. Biol. 57:461-485.

Guzman, P.S., B.W. Diers, D.J. Neece, S.K. St. Martin, A.R. LeRoy, C.R. Grau, T.J. Hughes, and R.J. Nelson. 2007. QTL associated with yield in three backcross-derived populations of soybean. Crop Sci. 47:111-122.

Hagedorn, D.J., and D.A. Inglis. 1986. Handbook of bean diseases. Madison, WI. UW Extension Publication.

Hyten, D.L., Q. Song, E.W. Fickus, C.V. Quigley, J.S. Lim, I.Y. Choi, E.Y. Hwang, M. Pastor-Corrales, and P.B. Cregan. 2010. High-throughput SNP discovery and assay development in common bean. BMC Genomics 11:475-483.

Jafar, M., R. Aggarwal, R. Buyyarapu, and S. Kumpatla. 2012. SNP markers and their impact on plant breeding. Int. J. Plant Genomics 1-11. doi:10.1155/2012/728398

Kamfwa, K., K.A. Cichy, and J.D. Kelly. 2015. Genome-wide association study of agronomic traits in common bean. The Plant Genome 8:1-12.

Kelly, J.D. 2010. The story of bean breeding. Michigan State University. http://bean.css.msu.edu/_pdf/Story_of_Bean_Breeding_in_the_US.pdf (accessed 16 June 2015).

Lamppa, R.S., P.L. Gross, and L.E. del Rio. 2002. Identification of races of *Pseudomonas syringae* pv. *phaseolicola* present in North Dakota. Phytopathology 92:S139.

Mamidi, S., M. Rossi, D. Annam, S. Moghaddam, R. Lee, R. Papa, and P. McClean. 2011. Investigation of the domestication of common bean (*Phaseolus vulgaris*) using multilocus sequence data. Funct. Plant Biol. 38:953-967.

Mamidi, S., M. Rossi, S.M. Moghaddam, D. Annam, R. Lee, R. Papa, and P.E. McClean. 2013. Demographic factors shaped diversity in the two gene pools of wild common bean *Phaseolus vulgaris* L. Heredity 110:267-276.

McClean, P.E., J. Terpstra, M. McConnell, C. White, R. Lee, S. Mamidi. 2012. Population structure and genetic differentiation among the USDA common bean (*Phaseolus vulgaris* L.) core collection. Genet. Resour. Crop Ev. 59:499-515.

Miklas, P.N., D. Fourie, J. Trapp, J. Davis, and J.R. Myers. 2014. New loci including conferring resistance to halo bacterial blight on chromosome Pv04 in common bean. Crop Sci. 54:2099-2108.

Miklas, P.N., D. Fourie, J. Trapp, R.C. Larsen, C. Chavarro, M.W. Blair, and P. Gepts. 2011. Genetic characterization and molecular mapping gene for resistance to halo blight in common bean. Crop Sci. 51:2439-2448.

Miklas, P.N., D. Fourie, J. Wagner, R.C. Larsen, and C.M.S. Mienie. 2009. Tagging and mapping *Pse-1* gene for resistance to halo blight in common bean differential cultivar UI-3. Crop Sci. 49:41-48.

Miklas, P.N., J.D. Kelly, S.E. Beebe, and M.W. Blair. 2006. Common bean breeding for resistance against biotic and abiotic stresses: From classical to MAS breeding. Euphytica 147:105-131.

Miklas, P.N., M.A. Pastor-Corrales, G. Jung, D.P. Coyne, J.D. Kelly, P.E. McClean, and P. Gepts. 2002. Comprehensive linkage map of bean rust resistance genes. Annu. Rep. Bean Improv. Coop. 45:125-129.

Miklas, P.N., R. Delorme, R. Hannan, and M. 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.

Mitchell, R.E. 1978. Halo blight of beans: Toxin production by several *Pseudomonas phaseolicola* isolates. Physiol. Plant Pathol. 13:37-49.

Moghaddam, S.M., Q. Song, S. Mamidi, J. Schmutz, R. Lee, P. Cregan, J.M. Osorno, and P.E. McClean. 2014. Developing market class specific InDel markers from next generation sequence data in *Phaseolus vulgaris* L. Front. Plant Sci. 5:1-13.

Nemli, S., T.K. Asciogul, H.B. Kaya, A. Kahraman, D. Eşiyok, and B. Tanyolac. 2014. Association mapping for five agronomic traits in the common bean (*Phaseolus vulgaris* L.). J. Sci. Food and Agric. 94:3141-3151.

Nordborg, M., and S. Tavare. 2002. Linkage disequilibrium: What history has to tell us. Trends Genet. 18:83-90.

Perseguini, J.M.K.C., P.R. Oblessuc, J.R.B.F. Rosa, K.A. Gomes, A.F. Chiorato, S.A.M. Carbonell, A.A.F. Garcia, R.P. Vianello, and L.L. Benchimol-Reis. 2016. Genome-wide association studies of anthracnose and angular leaf spot resistance in common bean (*Phaseolus vulgaris* L.). PLoS ONE 11:1-19.

Porch, T.G., J.S. Beaver, D.G. Debouck, S.A. Jackson, J.D. Kelly, and H. Dempewolf. 2013. Use of wild relatives and closely related species to adapt common bean to climate change. Agron. 3:433-461.

Porch, T.G., K. Cichy, M.A. Pastor-Corrales, M.A. Grusak, J.S. Beaver, J. Hart, D. Fourie, C. Estevez de Jensen, S. Nchimbi, and P.N. Miklas. Characterization and application of the Andean Diversity Panel for the improvement of common bean productivity in Sub-Saharan Africa. Poster session presented at the meeting of the Pan-African Grain Legume & World Cowpea Conference, 2016 February 28 – March 4, Livingstone, Zambia.

Rico, A., R. López, C. Asensio, M.T. Aizpún, M.C. Asensio, S. Manzanera, and J. Murillo. 2003. Nontoxigenic strains of *Pseudomonas syringae* pv. *phaseolicola* are main cause of halo blight of beans in Spain and escape current detection methods. Phytopathology 93:1553-1559.

Risch, N., and K. Merikangas. 1996. The future of genetic studies of complex human diseases. Sci. 273:1516-1517.

Schmutz, J., P.E. McClean, S. Mamidi, G.A. Wu, S.B. Cannon, J. Grimwood, J. Jenkins, S. Shu, Q. Song, C. Chavarro, M. Torres-Torres, V. Geffroy, S.M. Moghaddam, D. Gao, B. Abernathy, K. Barry, M. Blair, M.A. Brick, M. Chovatia, P. Gepts, D.M. Goodstein, M. Gonzales, U. Hellsten, D.L. Hyten, G. Jia, J.D. Kelly, D. Kudrna, R. Lee, M.M.S. Richard, P.N. Miklas, J.M. Osorno, J. Rodrigues, V. Thareau, C.A. Urrea, M. Wang, Y. Yu, M. Zhang, R.A. Wing, P.B. Cregan, D.S. Rokhsar, and S.A. Jackson. 2014. A reference genome for common bean and genome-wide analysis of dual domestications. Nature. Genet. 46:707-713.

Schwartz, H.F., and M. Pastor-Corrales. 1989. Bean production problems in the tropics. International Center for Tropical Agriculture (CIAT), Cali, Colombia. p. 285-293.

Schwartz, H.F., J.R. Steadman, R. Hall, and R.L. Forster. 2005. Compendium of bean diseases. 2nd ed. APS Press, St. Paul, MN. p. 30.

Shi, C., A. Navabi, and K. Yu. 2011. Association mapping of common bacterial blight resistance QTL in Ontario bean breeding populations. BMC Plant Biol. doi: 10.1186/1471-2229-11:52

Singh S.P., P. Gepts, and D.G. Debouck. 1991a. Races of common bean (*Phaseolus vulgaris* Fabaceae). Econ. Bot. 45:379-396.

Singh, S.P., J.A. Gutierrez, A. Molina, C. Urrea, and P. Gepts. 1991b. Genetic diversity in cultivated common bean II: Marker-based analysis of morphological and agronomic traits. Crop Sci. 31:23-29.

Singh, S.P., and H.F. Schwartz. 2010. Breeding common bean for resistance to diseases: A review. Crop Sci. 50:2199-2223.

Singh, S.P., H. Terán, M. Lema, D.M. Webster, C.A. Strausbaugh, P.N. Miklas, H.F. Schwartz, and M.A. Brick. 2007. Seventy-five years of breeding dry bean of the Western USA. Crop Sci. 47: 981-989.

Song, Q., G. Jia, D.L. Hyten, J. Jenkins, E.Y. Hwang, S.G. Schroeder, J.M. Osorno, J. Schmutz, S.A. Jackson, P.E. McClean, and P.B. Cregan. 2015. SNP assay development for linkage map construction, anchoring whole genome sequence, and other genetic and genomic applications in common bean. G3: Genes|Genomes|Genetics 5:2285-2290.

Taylor, J.H., N.L. Innes, C.L. Dudley, and W.A. Griffiths. 1978. Sources and inheritance of resistance to halo-blight of *Phaselous* beans. Ann. Appl. Biol. 90:101-110.

Taylor, J.D., D.M. Teverson, M.A. Allen, and M.A. Pastor-Corrales. 1996a. Identification and origin of races of *Pseudomonas syringae* pv. *phaseolicola* from Africa and other bean growing areas. Plant Pathol. 45:469-478.

Taylor, J.D., D.M. Teverson, and J.H.C. Davis. 1996b. Sources of resistance to *Pseudomonas syringae* pv. *phaseolicola* races in *Phaseolus vulgaris.* Plant Pathol. 45:479-485.

Teràn, H., M. Lema, D. Webster, and S.P. Singh. 2009. 75 years of breeding pinto bean for resistance to diseases in the United States. Euphytica 167:341-351.

Teverson, D.M. 1991. Genetics of pathogenicity and resistance in the halo-blight disease of beans in Africa. Ph.D. diss., Univ. of Birmingham, Birmingham, UK. p. 205.

Trabanco, N., M.C. Asensio-Manzanera, E. Perez-Vega, A. Ibeas, A. Campa, and J.J. Ferreira. 2014. Identification of quantitative trait loci involved in the response of common bean to *Pseudomonas syringae* pv. *phaseolicola*. Mol. Breeding 33:577-588.

USDA-NASS. 2014. Agricultural statistics database (quick stats), United States Department of Agriculture-National Agricultural Statistics Service, Washington, DC. http://www.nass.usda.gov (accessed 13 June 2015).

van Hintum, T.J.L, A.H.D. Brown, C. Spillane, and T. Hodgkin. 2000. Core collection of plant genetic resources. International Plant Genetic Resources Institute, Rome. p.51.

Virk, P., B. Ford-Lloyd, M. Jackson, H. Pooni, T. Clemeno, and H. Newbury. 1996. Predicting quantitative variation within rice germplasm using molecular markers. Heredity 76:296-304.

Wang, M., N. Jiang, T. Jia, L. Leach, J. Cockram, R. Waugh, L. Ramsay, B. Thomas, and Z. Luo. 2011. Genome-wide association mapping of agronomic and morphologic traits in highly structured populations of barley cultivars. Theor. Appl. Genet. 124:233-246.

Zahniser, S., M.V. Torres, J.A. Cuéllar Álvarez, N.F. López López, and R. Bhatta. 2010. The U.S. and Mexican dry bean sectors. VGS-341-01. USDA Economic Research Service. http://www.ers.usda.gov/media/134896/ vgs34101.pdf (accessed 20 June 2015).

Zhu, C., G. Michael, S.B. Edward, and Y. Jianming. 2008. Status and prospects of association mapping in plants. The Plant Genome 1:5-20.

Zuo, Q., J. Hou, B. Zhou, Z. Wen, S. Zhang. 2013. Identification of QTLs for growth period traits in soybean using association analysis and linkage mapping. Plant Breed. 132:317-323.

CHAPTER I. ASSESSMENT OF USDA-NPGS COMMON BEAN CORE COLLECTION FOR RESISTANCE TO RACE 6 OF *PSEUDOMONAS SYRINGAE* pv. *PHASEOLICOLA* UNDER GREENHOUSE CONDITIONS

Abstract

Halo blight, an important bacterial disease of common bean (*Phaseolus vulgaris* L.), is caused by a seed-borne bacterium *Pseudomonas syringae* pv. *phaseolicola* (Burkn.) Downs (*Psp*). However, currently only three sources of resistance are reported to race 6 of *Psp*, the most common and virulent race worldwide among nine races. Therefore, the objectives of this study were (i) to screen 283 accessions from USDA-NPGS common bean core collection for resistance to race 6 of *Psp* using primary (unifoliate) leaves inoculation method, (ii) to determine the most appropriate leaf stages, i.e., unifoliate versus trifoliate leaves, for optimal evaluation of halo blight symptoms, (iii) to differentiate the levels of disease reaction when inoculated in trifoliates versus pods within an individual plant, and (iv) to evaluate a group of selected accessions for resistance to race 6 of *Psp* under field conditions. Greenhouse experiments were conducted to address the first three objectives of the study. Five accessions with majority originating from Mexico had the highest levels of resistance to race 6 of *Psp* for unifoliate leaf inoculation with a mean disease score of 1.1. The mean of halo blight score across unifoliate inoculation (2.0) was significantly lower than trifoliate (4.0). A significant but weak correlation existed for halo blight score in trifoliate and pod inoculation in an individual plant under both greenhouse (r^2=0.17) and field conditions (r^2=0.21). The result suggests an independent mechanism of resistance at respective plant developmental stages. Resistant sources identified in the greenhouse were later confirmed under field condition where a mean halo blight score of 4.7 at pod stage was observed compared to 3.6 in trifoliate leaf stage. PI 313217 from Mexico displayed resistant reaction to

unifoliate, trifoliate, and pod stages across both greenhouse and field evaluation. Further efforts are required to introgress accessions with broad spectrum resistance to common bean breeding programs that may contribute to more durable resistance to this pathogen.

Introduction

Among several bacterial diseases, halo blight caused by *Pseudomonas syringae* pv. *phaseolicola* (Burkn.) Downs (*Psp*) is considered an important seed-borne disease of common bean (*Phaselous vulgaris* L.). The disease is prevalent within the bean growing regions of the world including in Midwestern parts of the United States, i.e., North Dakota, Minnesota, Wisconsin, Illinois, and Michigan (Duncan et al., 2014; Taylor et al., 1996b). This disease is considered one of the biggest threats to certified seed producers in the region that may sometime cause up to 45% yield losses (Hergert, 2010, Singh and Schwartz, 2010). Based on the interaction with eight differential cultivars, nine different races of *Psp* have been identified, where race 6 is most virulent and pre-dominant one (Taylor et al., 1999a; 1999b). However, besides three sources of resistance to race 6 of *Psp* reported by Duncan et al. (2014), Trabanco et al. (2014), and Porch et al. (2016), currently there is a lack of information regarding the additional sources of resistance. Most important, no previous studies have yet evaluated germplasm from USDA-NPGS core collection for resistance to race 6 of *Psp*, thus making this study one of important steps toward *Psp* resistance.

The plant-pathogen interaction for resistance to several bacterial diseases in common bean is considered complex biological relationship that greatly depends on the bacterial strain, inoculum concentration, environmental factors, methods of inoculation, and plant parts used for inoculation (Hill et al., 1972; Harper et al., 1987; Aggour et al., 1989; Mills and Silbernagel, 1992; Ariyarathne et al., 1998; Arnaud-Santana et al., 1994; Bozkurt and Soylu, 2011). A

thorough understanding of the mechanisms involved in the establishment, growth and reproduction of the pathogen at different plant growth stages also aids in the identification of age-related resistance, adult plant resistance, and ontogenic resistance (Chantret, et al., 2001; Ficke et al., 2003). For example, in common bean, Aggour et al. (1989), and Arnaud-Santana et al. (1994), confirmed the differential reactions in leaves and pods to different strains of common bacterial blight (CBB) at different concentrations, and suggested the role of independent genes controlling for resistance at respective plant growth stages. In case of halo blight, Hill et al. (1972) reported three independent genes controlling for resistance to race 1 of *Psp* when inoculated at two stages of plant development, i.e., leaves versus pods, and the systemic chlorosis development. Similarly, Mills and Silbernagel (1992) studied the effectiveness of evaluating halo blight severity at three plant growth stages using two inoculation methods. The methods included were hypodermic syringe method at 'crook neck' stem stage, multiple-needle florist pin frog method at ¾ expanded trifoliate leaves, and ½ to ¾ size of mature pod, respectively. The results displayed different halo blight severity at three stages, suggesting the occurrence of multiple genes for resistance, a finding like that reported by Hill et al., (1972).

Screening for plant disease resistance under both greenhouse and field conditions is an important and reliable step in identification of resistant genotypes to several plant diseases. Field screening not only provides the opportunity for evaluating large population at once but also considers of variable environment conditions as well as biotic interaction predominant out in the field unlike under greenhouse conditions (Zhang et al., 2004; Foolad et al., 2015). Therefore, field screening is required to identify types of resistance that may have been missed under greenhouse conditions, possibly because field resistance also may involve resistance to epiphytic colonization (Singh and Schwartz, 2010). Field evaluation provides the overall patterns of

disease progress over time or time and space that allows an opportunity to better understand host-pathogen interaction as well as crop phenology, and identify suitable techniques for disease management (Jeger and Viljanen-Robinson, 2001). For example, unlike in seedling stage, adult plants provide resistance to powdery mildew, *Blumeria graminis* f. sp. *tritici*, disease in wheat (*Triticum aestivum* L.) that delays the infection, growth, and reproduction of the pathogen (Wang et al., 2005).

Therefore, the primary objectives of this study were (i) to evaluate the USDA-NPGS common bean core collection for resistance to race 6 of *Psp* in terms of primary (unifoliate) and trifoliate leaf reactions under greenhouse conditions, (ii) to correlate the levels of halo blight symptoms in trifoliate leaves versus pods within an individual plant of selected accessions, and (iii) to evaluate a selected group of accessions for resistance to race 6 of *Psp* under field conditions.

Materials and Methods

Plant Material

During the spring of 2013, a total of 383 accessions from the core collection of *P. vulgaris* obtained from the NPGS collection were planted in a greenhouse complex at the Agriculture Experiment Station (AES) of North Dakota State University, Fargo, ND for seed increase (Appendix I). The core collection was conserved at the Western Regional Plant Introduction Station (WRPIS), USDA, Pullman, WA. Within this set, 206 accessions were originated from Mexico, 83 from Central America, 93 from South America, and 1 from Iran (Appendix I). Of the total 383 accessions, only 281 accessions flowered, and produced seeds which were harvested and cold stored. Remaining 102 accessions did not flower until 60 days

after planting and were considered as photo-period sensitive. For this reason, a set of 281 photo-period insensitive accessions were evaluated for resistance to race 6 of *Psp*.

Race 6 of *Pseudomonas syringae* pv. *phaseolicola* Isolate

Bacterial isolates of *Psp* were collected between the year 1995 and 2000, and race-typed by Robin Lamppa from the dry bean and pulse disease laboratory at North Dakota State University, Department of Plant Pathology. The highly virulent isolate of *Psp* 2000-13, collected from Oakes experiment station, Dickey county, ND, was used in this study for all the experiments under both greenhouse and field conditions. This isolate, which was derived from a single colony, was identified and later confirmed as race 6 of *Psp* using a set of eight differential cultivars under greenhouse conditions as described by Taylor et al., (1996a) (Table 1). Thus, collected isolate of race 6 was grown on King's B agar (King et al., 1954) for 48 h at 25° C and a cell suspension made in sterile distilled water. The inoculum density was adjusted to contain approximately 1 x 10^8 colony forming unit ml^{-1} (CFU/ml) using a spectrophotometer. Absorbancy was measured at an optical density (OD) 660nm, and compared with a previously established standard growth curve correlating cell number to absorbance.

Experimental Design

Three experiments were carried out to address the objectives of the study, where the first experiment was conducted under greenhouse conditions to evaluate a total of 281 core collection for resistance to unifoliate leaf inoculation method. In the second experiment, a selected group of 37 and 25 accessions displaying a range of reactions to unifoliate inoculation method was further evaluated for reaction to *Psp* using trifoliate and pod inoculation method under greenhouse conditions. A field evaluation of halo blight symptoms in 49 accessions which displayed resistant

reactions to both trifoliate and pod inoculation method under greenhouse conditions, was carried out in a third experiment. The experimental design of each three experiment is explained below.

Unifoliate Inoculation

For this experiment, 281 photo-period insensitive accessions along with two checks were evaluated for unifoliate leaf reactions under greenhouse conditions, for a total of 283 genotypes. Pinto bean US14HBR6 (Duncan et al., 2008; 2014) was used as the resistant check, and Pink Panther, light red kidney (LRK), was used as the susceptible check (Seminis Seeds). Because of uniform halo blight disease pressure observed in Pink Panther over the years across dry bean growing regions of western Minnesota and eastern North Dakota, it was chosen as a susceptible check. In addition, Pink Panther is one of the most commonly grown LRK cultivars in Minnesota (Knodel et al., 2014). Disease screening for resistance to race 6 of *Psp* was conducted in a walk-in growth chamber (Bio Cold Environmental, Inc., Ellisville, MO) in the Agricultural Experiment Station (AES) at NDSU, Fargo, ND greenhouse facilities (Figure 1). Before sowing, seeds of each accession were scarified to ensure the 100% germination.

Figure 1. Plant accessions grown in a plastic pot arranged in plastic trays in three shelves of two greenhouse metal cart inside growth chamber. Each tray contained one resistant and susceptible check.

Four seeds from each accession were planted in a plastic pot (15.2 × 15.2 ×15.2 cm diameter) containing Metro mix soil (3:1 peat moss: perlite) (Sun Gro Hort. Co.) in a randomized complete block design (RCBD) with four replications. These plastic pots (Scotts-Sierra Horticultural Product Company, Maryville, OH, USA) were transferred into a plastic tray (size 53 x 28 cm) (Figure 1). Ten days after planting, two healthy seedlings maintained in each pot were used for an experiment. The artificial light for photo-period (L: D 16:8 hours), temperature (18 to 22°C) and relative humidity (85 to 90%) were maintained automatically inside growth chamber. Two-week-old seedlings were fertilized with approximately 5 g of slow release fertilizer (20 N: 20 P: 20 K) per plastic pot.

For unifoliate experiment, each ¾ expanded primary (unifoliate) leaf of each accession at 10 d after planting (DAP), VC growth stage, were inoculated with 48 h old culture of race 6 of *Psp*. The leaf was inoculated using multiple-needle florist pin frog method (2-inch round brass

with rows of needles 0.5 inch long and 0.25 inch apart) (Figure 2; Zaiter and Coyne 1984; Mills and Silbernagel, 1992, Duncan et al., 2008, 2014).

For inoculation purposes, unifoliate from each accession was placed between multiple-needle on top and sponge underneath, where sponge was placed in a plastic petri dish (100m x 15 mm; item# T-6248) with an inoculum (Figure 2). One gentle push on top of the multiple-needle helped puncture through the leaf into the sponge, because of which the inoculum soaked up in sponge was drawn up into the wounds created by the pins as a bacterial pathway into the plant. Following inoculation, the plants were transferred into a humidity chamber maintained at 100% RH and 19°C ± 1°C for 48 h. To ensure 100% humidity inside the humidity chambers, water in form of mist was applied and regulated 1 min for every 30 mins cycle. After 48 h, inoculated plants were transferred back into a walk-in growth chamber, and were rated for halo blight disease symptoms 10 days post-inoculation (dpi) using a 1 - 9 rating scale, where 1-3 = resistant; 4-6 = intermediate; and 7-9 = susceptible (Mills and Silbernagel, 1992; Table 2; Figure 3).

Figure 2. Disease inoculation using the multiple-needle florist pin frog method. Inoculum in the petri dish is soaked in the sponge that is drawn into the leaves via the wounds.

Table 2. Halo blight disease severity ratings for evaluation of common bean (*Phaseolus vulgaris* L.) tissues following inoculation with *Pseudomonas syringae* pv. *phaseolicola* at two stages of development[1] (Mills and Silbernagel, 1992).

	Trifoliate leaf inoculation		**Pod inoculation**	
HB damage rating[1]	**Water soak at the inoculation point**	**Halo development**	**Systemic chlorosis**	**Water soak at the inoculation point**
1	None	None	None	None
2	trace (< 1 mm)	None	None	none with trace necrosis
3	slight (1-2 mm)	None	None	slight (1-2 mm) turns necrotic in 24-48 hrs
4	Slight (1-2 mm)	Slight (up to 1 mm beyond inoculation point)	Transitory	slight (1-2 mm) turns necrotic in 24-48 hrs inoculation point
5	Moderate (2-3 mm)	Slight (up to 1 mm beyond inoculation point)	Transitory	moderate (2-3 mm) turns necrotic in 48-72 hrs inoculation point
6	Moderate (2-3 mm)	Moderate (1-2 mm beyond inoculation point)	Transitory	Moderate (2-3 mm) no necrosis
7	Moderate to severe (3-4 mm)	Moderate (1-2 mm beyond inoculation point)	Slight permanent (< 1/4 leaflet affected)	Moderate (2-3 mm) no necrosis
8	Moderate to severe (3-4 mm)	Moderate (2-3 mm beyond inoculation point)	Moderate permanent (1/4-1/2 leaflet affected)	Moderate to severe (3-4 mm) no necrosis
9	Severe (> 4 mm)	Severe (> 3 mm beyond inoculation point)	Severe permanent (>1/2 leaflet affected)	Severe (> 4 mm) no necrosis

1 1 to 3 = resistant, 4 to 6 = intermediate, and 7 - 9 = susceptible.

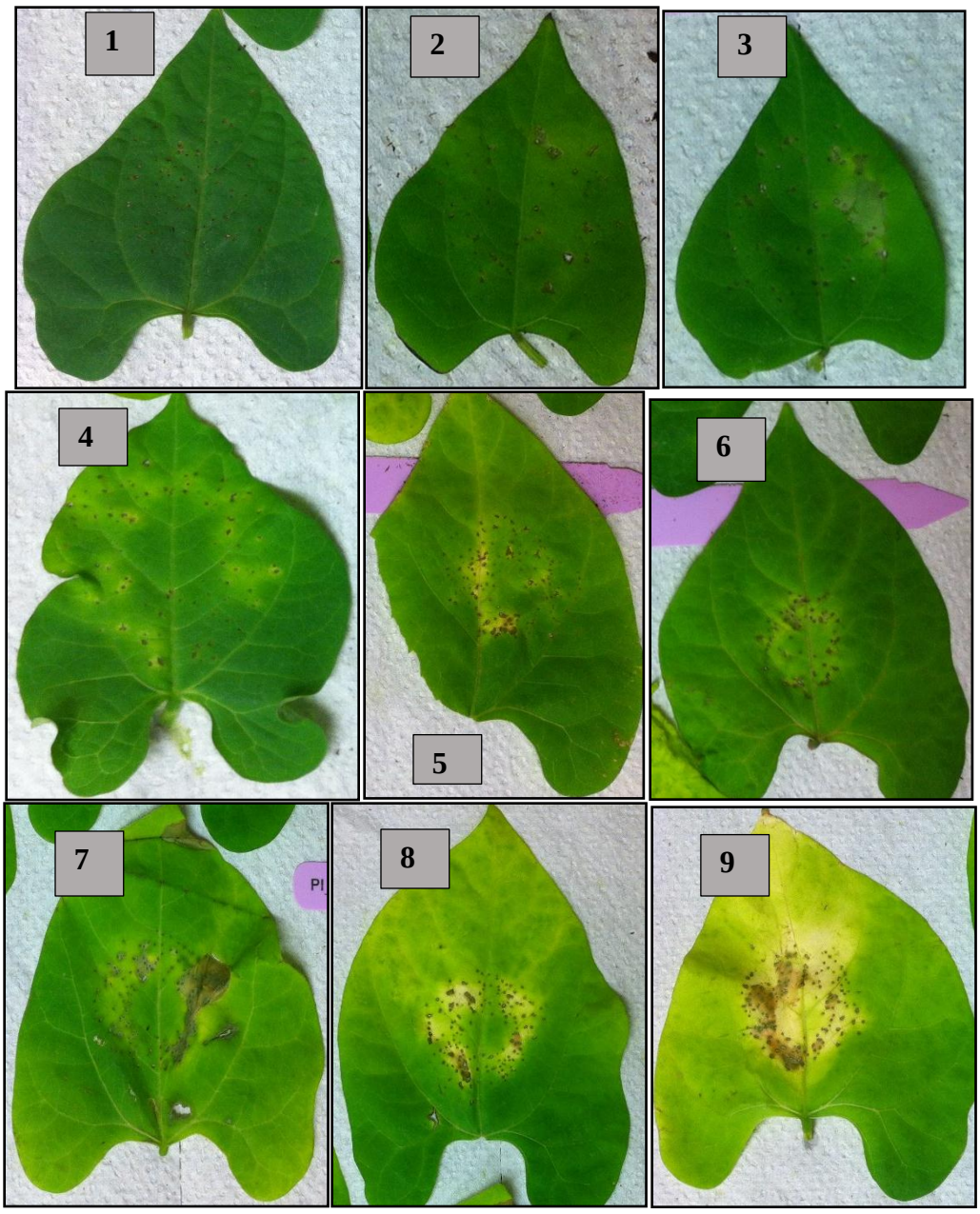

Figure 3. Halo blight severity rated in the primary (unifoliate) leaves of common bean accessions based on the disease rating scale of 1-9, where 1 being resistant and 9 being susceptible.

Trifoliate and Pod Inoculation

In this experiment, 81 selected genotypes (comprised of 62 accessions plus 19 standard cultivars as commercial checks; Table 3), were evaluated for trifoliate and pod disease reaction under greenhouse conditions. Sixty-two accessions were chosen based on the disease reactions to unifoliate inoculation method in previous experiment, and consisted of 37 resistant (score 1.0 to < 3.0) and 25 susceptible (> 7.0 to 9.0) accessions, respectively. The commercial checks represented the most commonly grown cultivars of the respective market classes in the regions of western Minnesota and eastern North Dakota (Table 3). US14HBR6 and Montcalm (DRK) were used as resistant checks (Adams and Saettler, 1974; Duncan et al., 2014; Mills and Silbernagel, 1992), while Pink Panther was used as a susceptible check. The experiment was arranged in a 9 x 9 square lattice design with four replications under greenhouse conditions.

Following seed scarification, four seeds of each accession were sown in a 6 inch x 6-inch clay/Terra Cotta pots filled with #1 Sunshine Mix Media (3:1 peat moss: perlite, SunGro Horticulture; Bellvue, WA). Each clay pot was fertilized with approximately 5 g of Osmocote (20 N: 20 P: 20 K) controlled slow release fertilizer (Scotts-Sierra Horticultural Product Company, Marysville, OH, USA) at 2-3-week-old seedling stage. Two weeks after planting, four seedlings in each pot were thinned to keep two plants that were later used for trifoliate experiment. Similar inoculation procedure as in unifoliate method was employed for trifoliate and a pod stage, except that trifoliate leaf was inoculated using the multiple-needle method at 21 days after planting (DAP), 2nd stage of trifoliate. Following trifoliate inoculation, pods from same plant were inoculated at about 1/3 to 2/4 of their respective mature sizes (R4-R5 growth stages). Three pods from each plant were arbitrarily chosen for inoculation purposes. Inoculated plants were maintained inside humidity chamber for 48 h as described in previous section, and

returned to the regular greenhouse until disease evaluation. Disease severity across both trifoliate and pod were evaluated 10 dpi using a similar disease severity scale of 1-9 (Table 2; Figure 4 & 5).

Table 3. Lists of dry bean cultivars used as check for the evaluation of trifoliate and pod reactions to race 6 of *Psp*. Included are their market classes, and sources.

Genotypes‡	**Market Class†**	**Source¶**
Eclipse	Black	NDSU
Cabernet	DRK	Seminis Seeds
Majesty	DRK	Agriculture & Agri-Food Canada, Ontario, Canada
Montcalm	DRK	USDA-ARS/Michigan Ag. Exp. Stat.
Redhawk	DRK	USDA-ARS/Michigan Ag. Exp. Stat.
Red Rover	DRK	Seminis Seeds
CELRK§	LRK	UC-Davis
Clouseau	LRK	Seminis Seeds
Foxfire	LRK	Rogers/Syngenta
Pink Panther	LRK	Seminis Seeds
Norstar	Navy	NDSU
Hime‡	Otebo	Hokkaido Pref. Tokachi Ag. Exp. Stat., Hokkaido, Japan
La Paz	Pinto	Provita
Lariat	Pinto	NDSU
Santa Cruz	Pinto	Provita
Sinaloa	Pinto	Provita
Stampede	Pinto	NDSU
US14HBR6	Pinto	UC-Davis; UI
Windbreaker	Pinto	Seminis Seeds

‡ Montcalm, and US14HBR6 used as resistant checks; Pink Panther used as susceptible checks. Remaining indicates commercial checks.
§ CELRK = California Early Light Red Kidney
† DRK = Dark Red Kidney; LRK = Light Red Kidney
¶ NDSU = North Dakota State University**;** UC-Davis = University of California-Davis; USDA-ARS = United States Department of Agriculture-Agriculture Research Station
UI = University of Idaho, Kimberly
‡ Found to be highly susceptible to halo blight disease under field conditions in 2009 in Wyoming

Figure 4. Halo blight severity evaluated in the trifoliate leaves of common bean accessions at 10 dpi (days post inoculation). The numbers in the picture denotes the disease rating scale.

Figure 5. Halo blight severity in the pods of common bean accessions 10 days post inoculation (dpi) based on the disease rating scale (1-9) under greenhouse conditions.

Field Evaluation

A total of 49 accessions comprised of 30 accessions and 19 standard cultivars as checks (Table 3) were arranged in a 7 x 7 alpha-lattice with two replications. Thirty accessions were chosen based on their resistant reactions to halo blight disease following trifoliate and pod inoculation under greenhouse conditions. However, due to the low amount of seeds available, each experimental unit consisted of one row plot where a single row was 2.7 m long with 0.3 m row-spacing.

The experiment was conducted at Perham, MN during the 2014 growing season because of the previous report on the natural presence of high disease pressure for *Psp*. According to the mechanical and chemical analysis of soil samples collected from 0 to 15 cm top layer at NDSU soil testing laboratory, the soil at experimental site was classified sandy-loamy [(name=Sandberg; family=Entic Haplydolls; order=Mollisol (USDA-NRCS, 2016)] with pH

ranged from 6.2-7.2, and contained about 1.6-2.2% organic matter, available nitrate-nitrogen (6.7 kg ha^{-1}), available phosphorus (126 kg ha^{-1}), available potassium (540 kg ha^{-1}). The climate of the region is cold and temperate with an average annual temperature of 40 °F and average annual precipitation of 656 mm, respectively (NDAWN, 2014). The experimental site is located at Lat: 46.45°N; Lon: 95.21°W; and 416 masl.

In addition, a separate greenhouse experiment was conducted to identify the predominant race at this experiment site using the differential lines as explained by Taylor et al. (1996a) (Table 1), and was later confirmed that race 6 of *Psp* as prevalent race. Therefore, the inoculum for the present experiment was prepared from the halo blight infected leaf samples collected from this site.

Four plants within each plot were randomly selected, flagged, and three trifoliates and four pods within that individual flagged plant were inoculated. However, the inoculation process was slightly different than under greenhouse conditions as trifoliates and pods of each flagged plants within a row were wounded using multiple-needle florist pin frog method (described as previous). The process was followed by spraying an inoculum to injured trifoliates and pod using a backpack sprayer (Chapin61500 4G/15.1L Pro Series Backpack Sprayer). Like the greenhouse experiment, the plants in the field were inoculated at second trifoliate stages (V2 growth stages or at 21 DAP), and same individual plant was used for pod inoculation at R4-R5 growth stages. However, to ensure enough disease pressure on experimental plants, 10 plants of CELRK (California Early Light Red Kidney) were planted along the border of each experimental plot across the two replications as infester/spreader rows. The trifoliates from the plants in infester row were also inoculated at the same time as experimental plots following similar inoculation process. Halo blight disease severity in both trifoliates and pods under field condition were

evaluated using a rating scale of 1-9 (Table 2). In addition, disease symptoms in each trifoliate and pod were repeatedly evaluated at 7, 14, 21, and 28 days after inoculation, i.e., at an interval of one week after first evaluation.

Statistical Analysis

All three experiments were analyzed using PROC MIXED PROCEDURE in SAS/STAT® 9.3 (SAS Institute Inc. 2012. SAS/STAT® 9.3 User's Guide. Cary, NC). A normality test for the data obtained from all experiments were performed using the Kolmogorov-Smirnov test with $P \leq 0.05$ used to indicate lack of fit.

For the unifoliate experiment, replications and plant accessions were considered random and fixed effects, respectively. Adjusted (Lsmeans) means of halo blight severity were compared using *pdiff* to classify categories of their resistance between accessions at $\alpha = 0.05$ level of significance. In trifoliate versus pod experiment, due to missing data, the experiment was analyzed in randomized complete block design (RCBD) using PROC MIXED. Replications were considered random effects, while plant accessions and plant stages (i.e. trifoliate and pod stage), were considered as fixed effects. Lsmeans (adjusted means) of halo blight scores were compared using *pdiff* to classify categories of their resistance between accessions at $\alpha = 0.05$ level of significance. The associations between halo blight disease scores in trifoliate versus pod stage was determined in terms of Pearson's correlation coefficients using the PROC CORR procedure in SAS (SAS Institute Inc. 2012. SAS/STAT® 9.3 User's Guide. Cary, NC). Halo blight disease scores on trifoliate was measured on each of 49 accessions, but scores on pod were recorded only from 48 accessions due to one accession being photoperiod-sensitive. Halo blight severity on both trifoliate leaves and pods were analyzed as accession and time in split-plot design over time using PROC MIXED. The accessions (main plot) and weeks (main plot) were considered as

fixed effects, and replications and blocks were considered as random effects. In these experiments, successive measurements of halo blight scores on both trifoliates and pods were made on the same accession over a period of four weeks at weekly intervals. Such data are repeated measures, which are analogous to split-plot in time and therefore their analyses are performed as such (Steele and Torrie, 1980). Mean of halo blight scores were compared for accessions and weeks using F-protected LSD test ($\alpha = 0.05$). A correlation analysis of halo blight disease scores was performed to examine between trifoliate and pod using PROC CORR to generate Pearson's correlation coefficient (r).

Results

Unifoliate Inoculation

In this experiment, the comparative study of 281 accessions plus two commercial checks demonstrated the occurrence of highest levels of resistance to halo blight severity in unifoliate (primary) leaf (Appendix II). A significant variation ($P \leq 0.05$) was observed among 281 accessions evaluated for halo blight severity (Table 4).

Table 4. Analysis of Variance (ANOVA) for the reactions of 281 accessions and two checks to race 6 of *Psp* in the unifoliate leaves.

Sources of variation	**df**	**Mean Squares**
Replication	3	29.88*
Accessions	282	12.60*
Error	842	4.49
Total	6959	

* Significant at $P \leq 0.01$

The overall mean and range of halo blight disease score among 281 accessions were reported 4.3, and 1.0-7.3, respectively. Of these 281 accessions evaluated, 247 (88%) accessions are from Mesoamerican origin with mean halo blight score of 4.0 compared to 34 (12%) accessions from Andean origin with mean score of 4.5 (data not shown here). However, based on

the disease score ratings in unifoliate leaves, 37 (13%), 219 (78%), and 25 (9%) accessions exhibited resistant, intermediate, and susceptible reactions, respectively (Figure 6). Thirty-seven accessions with high levels of resistance had a mean disease score of 1.8 and range of 1.0-2.5 (Table 5). Those 37 accessions were identified as potential sources of physiological resistance to race 6 of *Psp* as halo blight scores <3 represent high level of restricted necrotic region development (1-2 mm) (Figure 3; Table 2), and were later evaluated for trifoliate and pod evaluation. The resistant check US14HBR6 had expected resistant reaction (1.9), while an intermediate reaction (6.5) was observed in susceptible check, Pink Panther.

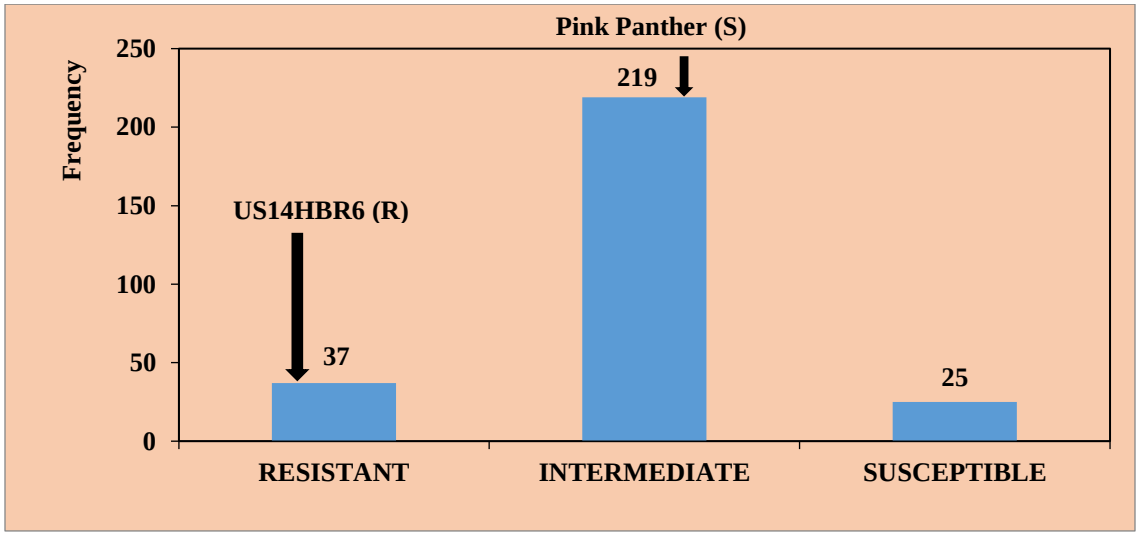

Figure 6. Distribution frequency of resistant, intermediate, and susceptible accessions to race 6 of *Pseudomonas syringae* pv. *phaseolicola* (*Psp*) bacterium following unifoliate inoculation method. Resistant (R) and susceptible (S) check values are indicated in arrows. Halo blight severity is based on the water-soaked region at the inoculation point and halo development. The 1-9 rating scale is classified into three general categories of resistant (1-3), intermediate (4-6), and susceptible (7-9).

Table 5. Mean and standard error of halo blight scores in unifoliate leaves of 37 resistant common bean accessions from the core collection to race 6 of *Psp* under greenhouse conditions.

Accession	Mean ± S.E.†	Country of Origin	Type	Seed Color‡	Accession	Mean ± S.E.†	Country of Origin	Type	Seed Color‡
533259	1.0 ± 0.5	Mexico	Landrace	BL	313328	2.0 ± 0.9	Mexico	Landrace	BL
201329	1.1 ± 0.9	Mexico	Landrace	DY	533476	2.0 ± 0.9	Mexico	Landrace	BL
309810	1.1 ± 1.0	Mexico	Landrace	LY	200956	2.1 ± 0.9	El Salvador	Landrace	BL
310826	1.1 ± 0.9	Nicaragua	Cultivated	BR	207127	2.1 ± 0.9	Colombia	Landrace	CR/DP
319592	1.1 ± 1.0	Mexico	Landrace	CR/WH	308898	2.1 ± 0.9	Costa Rica	Landrace	LT
417657	1.2 ± 0.9	Mexico	Cultivated	BL	310818	2.1 ± 0.9	Nicaragua	Cultivated	BR
290990	1.3 ± 1.0	Peru	Landrace	CR/RD	311843	2.1 ± 0.9	Guatemala	Landrace	CR/PU/LB
313343	1.3 ± 0.9	Mexico	Landrace	BL	313254	2.1 ± 0.9	Mexico	Landrace	BL
449410	1.3 ± 0.9	Mexico	Landrace	CR/DB	313490	2.1 ± 0.9	Mexico	Landrace	BL
201296	1.5 ± 0.9	Mexico	Landrace	DY	311974	2.2 ± 0.9	Mexico	Landrace	BL
313596	1.6 ± 1.0	Colombia	Cultivated	WH/BR	313572	2.2 ± 0.9	Colombia	Cultivated	BL
531862	1.6 ± 0.9	Peru	Landrace	WH	313217	2.3 ± 0.9	Mexico	Landrace	PU/RD
201343	1.7 ± 0.9	Mexico	Landrace	TN/LT	313237	2.3 ± 0.9	Mexico	Landrace	TN/BR/CR
310829	1.7 ± 0.9	Nicaragua	Cultivated	RD/DR	313394	2.3 ± 0.9	Mexico	Landrace	DE/TN
415949	1.7 ± 0.9	Peru	Landrace	WH/BR	313809	2.3 ± 0.9	Mexico	Cultivated	BL
207373	1.8 ± 0.9	Colombia	Landrace	BL	325653	2.4 ± 1.0	Mexico	Landrace	CR
209479	1.8 ± 0.9	Nicaragua	Landrace	BL	476751	2.4 ± 0.9	Guatemala	Landrace	DR/RD
325732	1.8 ± 0.9	Mexico	Cultivated	CR	US14HBR6¶	1.9 ± 0.5	UC-Davis; UI ǂ	Germplasm	PINTO
533475	1.8 ± 0.9	Mexico	Landrace	CR/DB	PINK PANTHER§	6.5 ± 0.5	Seminis Seeds	Cultivar	LRK
207322	2.0 ± 0.9	Colombia	Landrace	BL					

† Mean disease score ranged 1 to 9; 1 to 3 = resistant (R), 4 to 6 = intermediate (I), 7 - 9 = susceptible (S); SE = Standard error of mean. ‡ Seed color: BL = black; BR = Brown; CR = Cream-beige; DY = dark yellow; LRK = Light red kidney; LT = Light tan; LY = Light yellow; WH = White; TN/LT = Tan/Light tan; CR/DP = Cream-beige/Dark purple; CR/RD = Cream-beige/Red; TN/BR/CR = Tan/Brown/Cream-beige; DR/RD = Red/Dark red; CR/PU/LB = Cream-beige/Purple/Light brown; PU/RD = Purple/Red; TN/BR = Tan/Brown; DE/TN = Dark Grey/Tan; WH/BR = White/Brown; CR/WH = Cream-beige/White; CR/DB = Cream-beige/Dark brown. Susceptible check; ¶ Resistant check; ǂ University of California-Davis; University of Idaho, Kimberly, ID.

Of the 37 accessions categorized as resistant, most of the accessions were concentrated in eight countries with the majority from Mexico (56%) and remaining from Colombia (13%), Costa Rica (3%), El Salvador (3%), Guatemala (5%), Nicaragua (11%), and Peru (8%) (Table 5). Five accessions, PI 201329, PI 309810, PI 310826, PI 319592, and PI 533259 were ranked as highly resistant with a mean halo blight disease score of about 1.1 compared to the standard resistant and susceptible checks (Figure 7). Out of those five accessions, four are originated from Mexico and PI 310826 from Nicaragua.

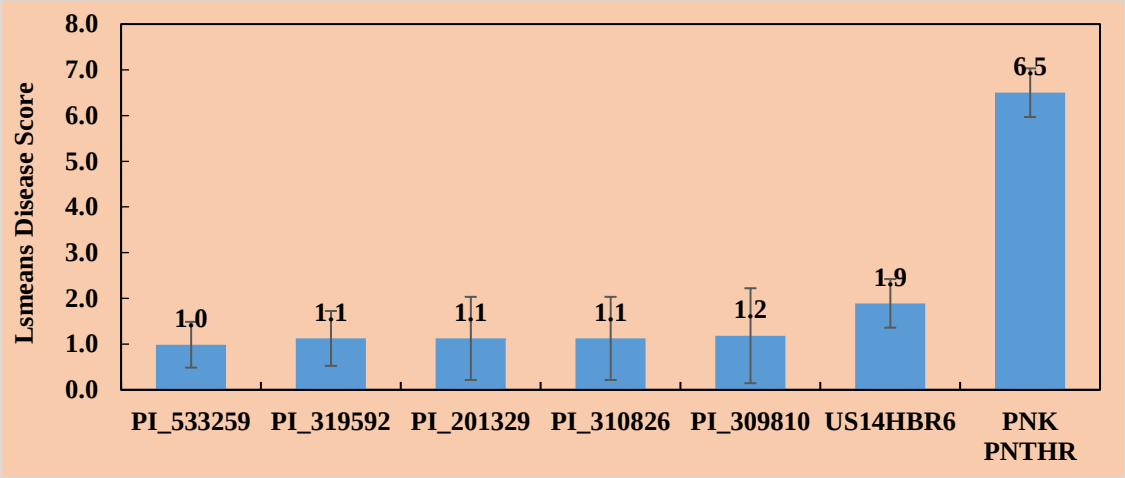

Figure 7. Mean halo blight severity of five most resistant accessions under greenhouse condition. Halo blight scores are the means of eight unifoliates per accession in each four replications.

Trifoliate and Pod Inoculation

In this study, trifoliate disease reactions to race 6 of *Psp* was investigated and compared with the results from unifoliate leaf inoculation method. Because the seeds of some accessions in this experiment had germination problem, and the trifoliate leaves of some accessions fell from the plant few days after inoculation, halo blight severity was recorded from only 77 entries out of total 81 planted. These 77 entries consisted of 58 accessions plus 19 commercial checks (Table 3). The ANOVA for disease reactions on the trifoliate leaves displayed significant variation among accessions (Table 6). The halo blight severity in trifoliate leaves was evaluated after 10-14 dpi, where the necrotic water-soaking lesions at and beyond the inoculation point were clearly visible, thus facilitating separation among different categories of disease reactions (Figure 4).

Table 6. Analysis of Variance (ANOVA) for the response of common bean accessions from the core collection in trifoliate leaves and pods reaction to race 6 of *Psp* under greenhouse conditions.

Sources of variation	**Trifoliate leaf**	
	df	**Mean Squares**
Replication	3	219.93*
Accessions	76	66.92*
Error	207	10.51
Total	1539	
	Pod	
	df	**Mean Squares**
Replication	3	4.83*
Accessions	67	15.65*
Error	175	3.46
Total	1411	

* Significant at $P \leq 0.01$

Out of the total 58 accessions, 21 (36%), 27 (46%), and 10 (17%) accessions were characterized as resistant, intermediate, and susceptible, respectively based on the mean disease score on a scale of 1-9 (Table 7). The LSmeans and range of halo blight disease score in 58

accessions used for trifoliate inoculation method was 4.3 and 1.0-7.4, respectively (Table 7). Pink Panther showed a mean halo blight disease score of 6.2 in trifoliate leaves, indicating intermediate reaction, while US14HBR6 exhibited the resistant reaction as expected with a mean disease score of 1.2. However, contrary to previous reports about the genetic tolerance to halo blight, Montcalm had susceptible reaction with a mean disease score of 7.0.

When disease scores from 35 common accessions evaluated for both unifoliate and trifoliate inoculation methods were compared, significant variation was observed (Table 8). Based on the unifoliate disease reactions, these 35 accessions were categorized as resistant (Table 5). The range and mean of halo blight disease score in 35 accessions following unifoliate inoculation method was lower with a value of 1.0-2.4 and 2.0 compared to 1.0-7.0 and 4.0 in trifoliate method, respectively (Table 8). Interestingly, Pink Panther and US14HBR6 across both inoculation methods had similar disease score of 6.5 and 1.9, respectively in unifoliate versus 6.2 and 1.2, respectively in trifoliate method.

Table 7. Mean and standard error of halo blight score in trifoliate leaves and pods of 58 common bean accessions and 19 cultivars (checks) evaluated under greenhouse conditions. Disease score in pods were collected from only 49 accessions.

Accession	Country of Origin	Type	Seed Color‡	Mean ± S.E. †		
				Trifoliate	Pod	Overall Mean
201329	Mexico	Landrace	DY	1.1 ± 0.9	1.6 ± 0.7	1.0
417657	Mexico	Cultivated	BL	1.2 ± 0.9	1.7 ± 0.7	1.0
449410	Mexico	Landrace	CR/DB	1.3 ± 0.9	2.8 ± 0.8	1.0
313343	Mexico	Landrace	BL	1.3 ± 0.9	2.1 ± 0.7	1.0
201296	Mexico	Landrace	DY	1.5 ± 0.9	3.1 ± 0.7	1.1
451917	Guatemala	Cultivated	BL	1.5 ± 0.9	3.0 ± 0.7	2.2
311942	Mexico	Landrace	CR	1.6 ± 0.9	1.5 ± 0.7	1.5
313596	Colombia	Cultivated	WH/BR	1.6 ± 1.0	1.1 ± 0.7	1.1
201343	Mexico	Landrace	TN/LT	1.7 ± 0.9	2.0 ± 0.7	1.2
313328	Mexico	Landrace	BL	2.0 ± 0.9	2.0 ± 0.7	1.3
531862	Peru	Landrace	WH	1.6 ± 0.9	2.8 ± 1.2	1.4
310818	Nicaragua	Cultivated	BR	2.1 ± 0.9	3.4 ± 0.7	1.4
311843	Guatemala	Landrace	CR/PU/LB	2.1 ± 0.9	1.9 ± 0.8	1.4
311974	Mexico	Landrace	BL	2.2 ± 0.9	3.3 ± 0.7	1.4
313572	Colombia	Cultivated	BL	2.2 ± 0.9	4.0 ± 0.8	1.5
313217	Mexico	Landrace	PU/RD	2.3 ± 0.9	1.8 ± 0.7	1.5
313237	Mexico	Landrace	TN/BR/CR	2.3 ± 0.9	3.5 ± 0.7	1.5
307788	El Salvador	Landrace	BR	1.6 ± 0.5	N/A	1.6
325653	Mexico	Landrace	CR	2.4 ± 1.0	1.3 ± 0.8	1.6
313490	Mexico	Landrace	BL	1.0 ± 0.5	3.1 ± 0.4	2.0
201388	Mexico	Landrace	BR	2.0 ± 0.5	N/A	2.0
313665	Ecuador	Cultivated	WH	3.1 ± 0.5	N/A	3.1
203936	Mexico	Landrace	BR	3.3 ± 0.5	N/A	3.3
310829	Nicaragua	Cultivated	RD/DR	4.0 ± 0.7	2.8 ± 0.4	3.4
207373	Colombia	Landrace	BL	4.3 ± 0.7	2.5 ± 0.4	3.4
200956	El Salvador	Landrace	BL	4.6 ± 0.7	2.1 ± 0.4	3.4

Table 7. Mean and standard error of halo blight score in trifoliate leaves and pods of 58 common bean accessions and 19 cultivars (checks) evaluated under greenhouse conditions. Disease score in pods were collected from only 49 accessions (continued).

Accession	Country of Origin	Type	Seed Color‡	Mean ± S.E. †		
				Trifoliate	Pod	Overall Mean
313499	Mexico	Landrace	BL	4.6 ± 0.7	2.2 ± 0.4	3.4
309810	Mexico	Landrace	LY	4.8 ± 0.7	1.9 ± 0.8	3.4
313809	Mexico	Cultivated	BL	5.6 ± 0.9	1.6 ± 0.8	3.6
319592	Mexico	Landrace	CR/WH	4.1 ± 1.4	3.3 ± 0.5	3.7
533577	Ecuador	Cultivated	WH	5.1 ± 0.7	2.2 ± 0.4	3.7
311999	Mexico	Landrace	RD	3.8 ± 0.5	N/A	3.8
415949	Peru	Landrace	WH/BR	4.2 ± 0.7	3.4 ± 0.4	3.8
304110	El Salvador	Landrace	WH	5.4 ± 0.7	2.4 ± 0.4	3.9
476751	Guatemala	Cultivated	DR/RD	4.8 ± 0.7	3.3 ± 0.4	4.1
313782	Mexico	Cultivated	BL	5.0 ± 0.7	3.1 ± 0.4	4.1
533476	Mexico	Cultivated	BL	5.2 ± 0.7	2.9 ± 0.4	4.1
310826	Nicaragua	Cultivated	BR	5.3 ± 0.7	3.0 ± 0.4	4.2
533475	Mexico	Cultivated	CR	5.8 ± 0.7	2.8 ± 0.4	4.3
313394	Mexico	Landrace	DE/TN	5.5 ± 0.7	3.3 ± 0.4	4.4
201387	Mexico	Landrace	DY	4.5 ± 0.7	N/A	4.5
313658	Ecuador	Cultivated	LT	5.5 ± 0.7	3.4 ± 0.5	4.5
201010	Guatemala	Landrace	CR	4.6 ± 0.8	N/A	4.6
313254	Mexico	Landrace	BL	6.4 ± 0.7	2.7 ± 0.4	4.6
319618	Mexico	Landrace	CR/TN	5.1 ± 0.7	4.3 ± 0.4	4.7
313609	Colombia	Cultivated	RD/CR	5.6 ± 0.8	3.8 ± 0.4	4.7
308898	Costa Rica	Landrace	LT	5.8 ± 0.8	3.8 ± 0.4	4.8
533259	Mexico	Cultivated	BL	7.0 ± 0.7	2.6 ± 0.4	4.8
207443	Colombia	Landrace	WH	4.9 ± 0.7	4.9 ± 0.4	4.9
209479	Nicaragua	Landrace	BL	6.1 ± 0.7	3.7 ± 0.4	4.9
451906	Guatemala	Cultivated	CR/RD	6.1 ± 0.7	3.8 ± 0.4	5.0
290990	Peru	Landrace	CR/RD	5.4 ± 0.7	5.0 ± 0.4	5.2

Table 7. Mean and standard error of halo blight score in trifoliate leaves and pods of 58 common bean accessions and 19 cultivars (checks) evaluated under greenhouse conditions. Disease score in pods were collected from only 49 accessions (continued).

Accession	Country of Origin	Type	Seed Color‡	Mean ± S.E. †		
				Trifoliate	Pod	Overall Mean
533332	Mexico	Cultivated	YL	6.8 ± 1.0	3.6 ± 0.4	5.2
207127	Colombia	Landrace	CR/DP	6.8 ± 0.7	4.0 ± 0.4	5.4
207148	Colombia	Landrace	LR/WH/BR	6.2 ± 0.7	5.0 ± 0.4	5.6
416468	Mexico	Cultivated	CR/WH	7.5 ± 0.7	3.0 ± 0.4	5.6
309823	Costa Rica	Landrace	CR/BL	6.2 ± 0.8	N/A	6.2
533281	Mexico	Cultivated	CR/RD	6.8 ± 0.7	5.5 ± 0.9	6.2
US14HBR6	UC-Davis; UI ǂ	Germplasm	PINTO¶	1.2 ± 0.7	1.5 ± 0.4	1.4
WINDBREAKER	Pinto	Cultivar	Seminis Seeds	2.5 ± 0.6	1.8 ± 0.2	2.1
STAMPEDE	NDSU ǂ	Cultivar	PINTO¶	2.9 ± 0.8	2.2 ± 0.4	2.6
LARIAT	NDSU	Cultivar	PINTO¶	6.0 ± 0.7	1.5 ± 0.4	3.8
LA PAZ	Provita	Cultivar	PINTO¶	6.5 ± 0.8	1.0 ± 0.4	3.8
ECLIPSE	NDSU ǂ	Cultivar	BL¶	5.7 ± 0.7	2.0 ± 0.4	3.9
SANTA_CRUZ	Provita	Cultivar	PINTO¶	4.8 ± 0.7	3.3 ± 0.4	4.1
SINALOA	Provita	Cultivar	PINTO¶	4.9 ± 0.7	3.4 ± 0.4	4.2
MAJESTY	Agriculture & Agri-Food Canada, Ontario, Canada	Cultivar	DRK¶	6.2 ± 0.8	2.2 ± 0.4	4.2
NORSTAR	NDSU	Cultivar	NAVY¶	4.5 ± 0.7	4.1 ± 0.4	4.3
FOXFIRE	Rogers/Syngenta	Cultivar	LRK¶	5.9 ± 0.7	2.7 ± 0.4	4.3

Table 7. Mean and standard error of halo blight score in trifoliate leaves and pods of 58 common bean accessions and 19 cultivars (checks) evaluated under greenhouse conditions. Disease score in pods were collected from only 49 accessions (continued).

Accession	Country of Origin	Type	Seed Color‡	Mean ± S.E. †		
				Trifoliate	Pod	Overall Mean
HIME	Hokkaido Pref. Tokachi Ag. Expt. Stat., Hokkaidao, Japan	Cultivar	OTEBO¶	6.4 ± 0.7	2.7 ± 0.6	4.6
MONTCALM	USDA-ARS/Michigan Ag. Expt. Stat.	Cultivar	DRK¶	7.0 ± 0.7	2.8 ± 0.4	4.8
CLOUSEAU	Seminis Seeds	Cultivar	LRK¶	7.0 ± 0.7	3.5 ± 0.4	5.3
CABERNET	Seminis Seeds	Cultivar	DRK¶	5.8 ± 0.8	3.9 ± 0.4	4.9
CELRK	UC-Davis ǂ	Cultivar	LRK¶	6.3 ± 0.8	3.4 ± 0.4	4.9
PINK PANTHER	Seminis Seeds	Cultivar	LRK¶	6.2 ± 0.8	4.1 ± 0.4	5.2
RED HAWK	USDA-ARS/Michigan Ag. Exp. Stat.	Cultivar	DRK¶	7.2 ± 0.7	4.9 ± 0.4	6.1
RED ROVER	Seminis Seeds	Cultivar	DRK¶	7.3 ± 0.7	3.2 ± 0.6	5.3
Mean		-	-	4.3	3.2	4.0
LSD ($P \leq 0.05$)				0.4	0.3	0.4

‡ Seed color where: BL = black; BR = Brown; CR = Cream-beige; DY = dark yellow; LRK = Light red kidney; LT = Light tan; LY = Light yellow; WH = White; TN/LT = Tan/Light tan; CR/DP = Cream-beige/Dark purple; CR/RD = Cream-beige/Red; TN/BR/CR = Tan/Brown/Cream-beige; DR/RD = Red/Dark red; CR/PU/LB = Cream-beige/Purple/Light brown; PU/RD = Purple/Red; TN/BR = Tan/Brown; DE/TN = Dark Grey/Tan; WH/BR = White/Brown; CR/WH = Cream-beige/White; CR/DB = Cream-beige/Dark brown.

† Mean disease score ranging from 1 to 9 where1 -3 = resistant (R), 4-6 = intermediate (I), 7-9 = susceptible (S); S.E. = Standard error of the mean.

ǂ North Dakota State University; University of California-Davis; University of Idaho, Kimberly

¶ Represents the market class of respective commercial checks used.

N/A = Accessions with missing pod score for halo blight.

Table 8. Mean and standard error of halo blight disease score in 35 common bean accessions following primary (unifoliate) and trifoliate leaves inoculation method. These 35 accessions displayed resistant reactions (score 1 to 3) to unifoliate leaf inoculation, and were common between both unifoliate and trifoliate experiments.

Accession	Country of Origin	Type	Seed Color‡	Mean ± S.E.†		
				Unifoliate leaves	Trifoliate leaves	Overall Mean
533259	Mexico	Landrace	BL	1.0 ± 0.5	7.0 ± 0.7	1.0
201329	Mexico	Landrace	DY	1.1 ± 0.9	1.6 ± 0.7	1.0
319592	Mexico	Landrace	CR/WH	1.1 ± 1.0	4.1 ± 1.4	1.2
309810	Mexico	Landrace	LY	1.1 ± 1.0	4.8 ± 0.7	1.0
310826	Nicaragua	Cultivated	BR	1.1 ± 0.9	5.3 ± 0.7	1.0
417657	Mexico	Cultivated	BL	1.2 ± 0.9	1.7 ± 0.7	1.0
313343	Mexico	Landrace	BL	1.3 ± 0.9	2.1 ± 0.7	1.0
449410	Mexico	Landrace	CR/DB	1.3 ± 0.9	2.8 ± 0.8	1.0
290990	Peru	Landrace	CR/RD	1.3 ± 1.0	5.4 ± 0.7	1.0
201296	Mexico	Landrace	DY	1.5 ± 0.9	3.1 ± 0.7	1.1
313596	Colombia	Cultivated	WH/BR	1.6 ± 1.0	1.1 ± 0.7	1.1
531862	Peru	Landrace	WH	1.6 ± 0.9	2.8 ± 1.2	1.4
201343	Mexico	Landrace	TN/LT	1.7 ± 0.9	2.0 ± 0.7	1.2
310829	Nicaragua	Cultivated	RD/DR	1.7 ± 0.9	4.0 ± 0.7	1.2
415949	Peru	Landrace	WH/BR	1.7 ± 0.9	4.2 ± 0.7	1.2
207373	Colombia	Landrace	BL	1.8 ± 0.9	4.3 ± 0.7	1.2
533475	Mexico	Landrace	CR/DB	1.8 ± 0.9	5.8 ± 0.7	1.2
209479	Nicaragua	Landrace	BL	1.8 ± 0.9	6.1 ± 0.7	1.2
313328	Mexico	Landrace	BL	2.0 ± 0.9	2.0 ± 0.7	1.3
533476	Mexico	Landrace	BL	2.0 ± 0.9	5.2 ± 0.7	1.3
313490	Mexico	Landrace	BL	2.1 ± 0.9	1.0 ± 0.7	1.4
311843	Guatemala	Landrace	CR/PU/LB	2.1 ± 0.9	1.9 ± 0.8	1.4
310818	Nicaragua	Cultivated	BR	2.1 ± 0.9	3.4 ± 0.7	1.4

Table 8. Mean and standard error of halo blight disease score in 35 common bean accessions following primary (unifoliate) and trifoliate leaves inoculation method. These 35 accessions displayed resistant reactions (score 1 to 3) to unifoliate leaf inoculation, and were common between both unifoliate and trifoliate experiments (continued).

Accession	Country of Origin	Type	Seed Color‡	Mean ± S.E.†		
				Unifoliate leaves	Trifoliate leaves	Overall Mean
200956	El Salvador	Landrace	BL	2.1 ± 0.9	4.6 ± 0.7	1.4
308898	Costa Rica	Landrace	LT	2.1 ± 0.9	5.8 ± 0.8	1.4
313254	Mexico	Landrace	BL	2.1 ± 0.9	6.4 ± 0.7	1.4
207127	Colombia	Landrace	CR/DP	2.1 ± 0.9	6.8 ± 0.7	1.4
311974	Mexico	Landrace	BL	2.2 ± 0.9	3.3 ± 0.7	1.4
313572	Colombia	Cultivated	BL	2.2 ± 0.9	4.0 ± 0.8	1.5
313217	Mexico	Landrace	PU/RD	2.3 ± 0.9	1.8 ± 0.7	1.5
313237	Mexico	Landrace	TN/BR/CR	2.3 ± 0.9	3.5 ± 0.7	1.5
313394	Mexico	Landrace	DE/TN	2.3 ± 0.9	5.5 ± 0.7	1.5
313809	Mexico	Cultivated	BL	2.3 ± 0.9	5.6 ± 0.9	1.6
325653	Mexico	Landrace	CR	2.4 ± 1.0	1.3 ± 0.8	1.6
476751	Guatemala	Landrace	DR/RD	2.4 ± 0.9	4.8 ± 0.7	1.5
US14HBR6¶ PNK	UC-Davis; UI ǂ	Germplasm	PINTO	1.9 ± 0.5	1.2 ± 0.7	1.3
PANTHER§	Seminis Seeds	Cultivar	LRK	6.5 ± 0.5	6.2 ± 0.8	6.3
Mean	-			2.0	4.0	1.3

† Mean disease score ranged from 1 to 9 where1 to 3 = resistant (R), 4 to 6 = intermediate (I), 7 - 9 = susceptible (S); SE = Standard error of the mean

‡ Seed color where: BL = black; BR = Brown; CR = Cream-beige; DY = dark yellow; LRK = Light red kidney; LT = Light tan; LY = Light yellow; WH = White; TN/LT = Tan/Light tan; CR/DP = Cream-beige/Dark purple; CR/RD = Cream-beige/Red; TN/BR/CR = Tan/Brown/Cream-beige; DR/RD = Red/Dark red; CR/PU/LB = Cream-beige/Purple/Light brown; PU/RD = Purple/Red; TN/BR = Tan/Brown; DE/TN = Dark Grey/Tan; WH/BR = White/Brown; CR/WH = Cream-beige/White; CR/DB = Cream-beige/Dark brown. § Susceptible check; ¶ Resistant check; ǂ University of California-Davis; University of Idaho, Kimberly, ID.

In the case of pod inoculation, a significant difference in reactions to halo blight severity was observed in the same 58 individual accessions that were used for trifoliate inoculation (Figure 5; Table 7). However, the pod score data was collected and analyzed from only 49 out of the 58 accessions because remaining accessions did not flower. Within the same plant, halo blight disease severity on average was higher in the trifoliate leaves compared to that in the pods. Most of the accessions were found to be intermediate and resistant to the pod reaction where the disease score ranged from 1.0-5.5, unlike in trifoliate leaves with a range of 1.0-7.4 (Table 7). In contrast, two accessions that exhibited susceptible disease reaction to trifoliate stage also displayed resistant reaction to pod stage, i.e., PI 416468 (7.5 in trifoliate and 3.0 in pod), and PI 533259 (7.0 in trifoliate and 2.6 in pod) (Table 7). A significant positive but weak correlation was reported for halo blight severity between the trifoliate leaves and pods stages ($r^2 = 0.1789$; $P < 0.05$) (Figure 8) in 49 accessions.

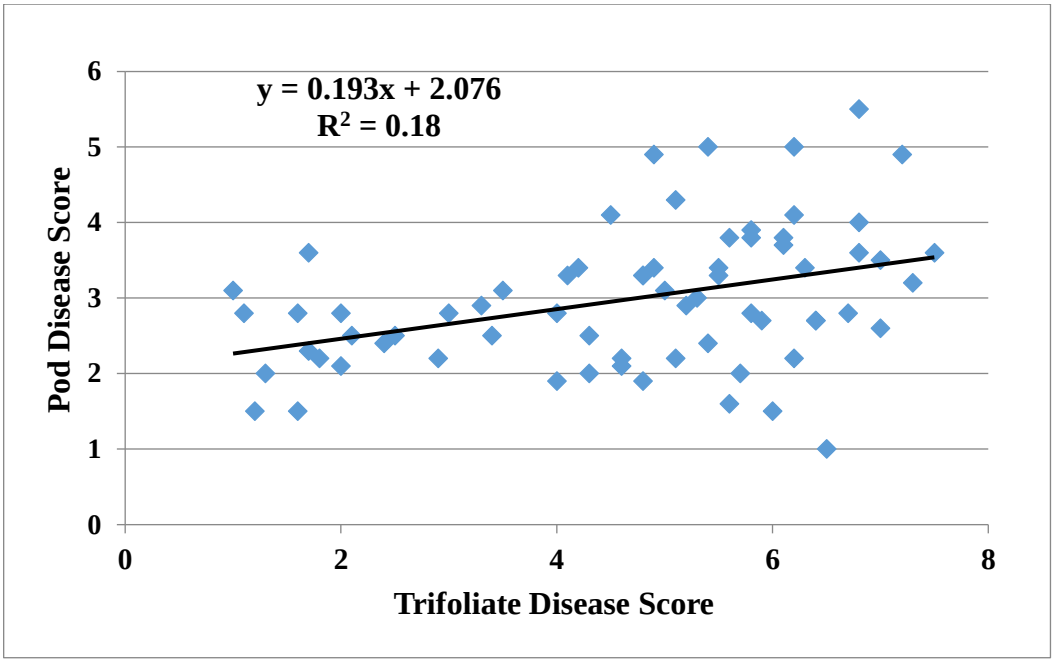

Figure 8. Linear regression for halo blight severity in trifoliate leaves and pods under greenhouse condition.

However, 12 plant accessions, i.e., PI 201343, PI 307788, PI 311942, PI 313217, PI 313328, PI 313343, PI 313490, PI 313596, PI 313665, PI 325653, PI 417657, and PI 451917 along with resistant check, US14HBR6, displayed the consistent resistant reactions across both trifoliate leaves and pod stages with a mean disease score of 1.8, and 2.0, respectively (Table 9). Of these 12 accessions, 8 accessions, PI 201343, PI 313217, PI 313328, PI 313343, PI 313490, PI 313596, PI 325653, and PI 417657 displayed the consistent resistant reactions across unifoliate, trifoliate, and pod stages (Table 9).

Table 9. Mean and standard error of halo blight score in unifoliate, trifoliate leaves, and pods of 8 common bean accessions evaluated under greenhouse conditions. Included are the disease scores of two checks, US14HBR6 and Pink Panther.

				Mean ± S.E. †			
Accession	**Type**	**Country of Origin**	**Seed Color‡**	**Trifoliate**	**Primary (unifoliate)**	**Pods**	**Overall Mean**
313596	Cultivated	Colombia	WH/BR	1.1 ± 0.7	1.7 ± 1.0	2.8 ± 0.4	1.4
417657	Cultivated	Mexico	BL	1.7 ± 0.7	1.3 ± 0.9	2.3 ± 0.4	1.5
313490	Landrace	Mexico	BL	1.0 ± 0.7	2.1 ± 0.9	3.1 ± 0.4	1.5
313343	Landrace	Mexico	BL	2.1 ± 0.7	1.4 ± 0.9	2.5 ± 0.4	1.7
325653	Landrace	Mexico	CR	1.3 ± 0.8	2.5 ± 1.0	2.0 ± 0.5	1.9
313328	Landrace	Mexico	BL	2.0 ± 0.7	2.0 ± 0.9	2.1 ± 0.8	2.0
201343	Landrace	Mexico	TN/LT	2.0 ± 0.7	1.8 ± 0.9	2.8 ± 0.5	2.0
313217	Landrace	Mexico	PU/RD	1.8 ± 0.7	2.4 ± 0.9	2.2 ± 0.4	2.1
US14HBR6	Germplasm	UC-Davis; UI ǂ	PINTO¶	1.2 ± 0.7	1.9 ± 0.5	1.5 ± 0.4	1.5
PINK PANTHER	Cultivar	Seminis Seeds	LRK¶	6.2 ± 0.8	6.5 ± 0.5	4.1 ± 0.4	5.6
Mean	-	-	-	2.0	2.4	2.5	2.1

† Mean disease score ranging from 1 to 9 where1 -3 = resistant (R), 4-6 = intermediate (I), 7-9 = susceptible (S); S.E. = Standard error of the mean.

‡ Seed color where: BL = black; CR = Cream-beige; DY = dark yellow; TN/LT = Tan/Light tan; CR/PU/LB = Cream-beige/Purple/Light brown; PU/RD = Purple/Red; WH/BR = White/Brown; CR/DB = Cream-beige/Dark brown.

Field Evaluation

The analysis of variance (ANOVA) resulted in significant interaction between the accession and time for halo blight scores (Table 10) in the trifoliate stage, but the interaction was non-significant in the pod stage. However, the results of ANOVA for accessions and time indicated significant differences on halo blight severity in both trifoliate and pod stages (Table 10). Halo blight severity in trifoliate at 28 days post inoculation (dpi) was significantly higher than earlier week, whereas halo blight severity in pod reached the highest scores at 21 dpi and remained consistent until 28 dpi (Table 11).

Table 10. Analysis of Variance (ANOVA) for the response of common bean accessions from the core collection in trifoliate leaves and pods reaction to race 6 of *Psp* under field conditions at Perham, MN in 2014.

Sources of variation	**df**	**Mean Squares**
----------Trifoliates ----------		
Replication	1	0.15
Block (Replication)	12	60.83
Accessions§	48	104.04*
Block (Replication) x Accessions	36	35.23*
Time§	3	1356.36*
Block (Replication) x Time	39	24.89*
Accessions x Time	144	12.19*
Block (Replication) x Accessions x Time	108	9.75
Total	4703	
----------Pods†----------		
Replication	1	155.0
Block (Replication)	12	61.66
Accessions§	47	140.81*
Block (Replication) x Accessions	30	50.79*
Time§	3	591.86*
Block (Replication) x Time	39	8.11
Accessions x Time	141	8.73
Block (Replication) x Accessions x Time	90	11.71
Total	5823	

* Significant at $P \leq 0.01$

† Due to photoperiod sensitivity, only 48 accessions are considered for pod analysis

§ Accessions and time are main plots

Table 11. Mean halo blight scores of common bean accessions at trifoliate leaf and pod stages evaluated at 7, 14, 21 and 28 days post inoculation (dpi). Disease score in accessions over the time is compared with disease score in susceptible (Montcalm) and resistant (US14HBR6 and Montcalm) checks.

Time (dpi)	Halo blight mean disease score (1-9)							
	Accessions		US14HBR6		Montcalm		Pink Panther	
	Trifoliate stage‡	Pod stage§	Trifoliate stage	Pod stage	Trifoliate stage	Pod stage	Trifoliate stage	Pod stage
7	3.0a†	3.8a	1.2a	4.2a	3.4a	4.4a	4.2a	5.1a
14	3.0a	4.7b	2.3b	5.1b	1.7b	6.2b	4.5b	7.4b
21	3.4b	5.2c	2.0b	4.5c	4.4c	6.1b	4.4b	7.5b
28	5.2c	5.2c	4.8c	4.5c	6.4d	6.0b	7.7c	6.5c

† Values with the same letter are not significantly different (LSD, $\alpha = 0.05$)

‡ Halo blight disease score at trifoliate stage was collected from 49 accessions. Trifoliate leaf was inoculated 21 DAP

§ Halo blight disease score at pod stage was recorded from 48 accessions. Pod was inoculated at about 1/3 to 2/4 of their respective mature sizes (R4-R5 growth stages)

Of the total 49 accessions evaluated under field condition, 16, 30, and 3 accessions had resistant, intermediate, and susceptible halo blight severity, respectively (Table 12). These 49 accessions were also evaluated for trifoliate and pod reactions under greenhouse condition. Under field conditions, the mean and range of halo blight disease score in the pod was significantly higher, i.e., 4.7 and 2.3-7.1, than at trifoliate stage, i.e., 3.6 and 2.0-6.6, respectively (Table 12). However, contrary to the field condition, the results from greenhouse conditions had significantly higher mean disease score and range at trifoliate stage 5.3 and 1.2-7.3, compared to pod stage 3.0 and 1.0-5.0 (Table 12).

Table 12. Overall mean of halo blight score in trifoliate and pod stages of 30 common bean accessions and 19 checks under greenhouse and field conditions. Origin, type, and seed color of each accession are also presented.

Accession	Country of Origin	Type	Seed Color‡	Halo blight score (Mean ± S.E. †)				Overall Mean
				Field condition		Greenhouse condition		
				Trifoliate	Pod	Trifoliate	Pod	
313217	Mexico	Landrace	PU/RD	2.5 ± 0.6	2.6 ± 0.6	2.3 ± 0.9	1.8 ± 0.7	2.3
311942	Mexico	Landrace	CR	3.3 ± 0.6	2.4 ± 0.6	1.6 ± 0.5	N/A	2.4
313596	Colombia	Cultivated	WH/BR	3.4 ± 0.6	3.5 ± 0.6	1.6 ± 1.0	1.1 ± 0.7	2.4
451917	Guatemala	Cultivated	BL	3.9 ± 0.6	2.9 ± 0.6	1.4 ± 0.5	N/A	2.7
203936	Mexico	Landrace	BR	2.3 ± 0.6	3.2 ± 0.6	3.3 ± 0.5	N/A	2.9
313237	Mexico	Landrace	TN/BR/CR	2.9 ± 0.6	3.4 ± 0.6	2.3 ± 0.9	3.5 ± 0.7	3.0
313499	Mexico	Landrace	BL	2.3 ± 0.6	3.5 ± 0.6	4.6 ± 0.7	2.2 ± 0.4	3.1
313343	Mexico	Landrace	BL	3.1 ± 0.6	6.1 ± 0.6	1.3 ± 0.9	2.1 ± 0.7	3.1
311974	Mexico	Landrace	BL	2.8 ± 0.6	4.5 ± 0.6	2.2 ± 0.9	3.3 ± 0.7	3.2
309810	Mexico	Landrace	LY	4.3 ± 0.6	2.6 ± 0.6	4.8 ± 0.7	1.9 ± 0.8	3.4
207373	Colombia	Landrace	BL	2.7 ± 0.6	4.8 ± 0.6	4.3 ± 0.7	2.5 ± 0.4	3.6
310829	Nicaragua	Cultivated	RD/DR	3.3 ± 0.6	4.3 ± 0.6	4.0 ± 0.7	2.8 ± 0.4	3.6
313394	Mexico	Landrace	DE/TN	3.0 ± 0.6	2.4 ± 0.6	5.5 ± 0.7	3.3 ± 0.4	3.7
415949	Peru	Landrace	WH/BR	3.7 ± 0.6	4.0 ± 0.6	4.2 ± 0.7	3.4 ± 0.4	3.7
310826	Nicaragua	Cultivated	BR	3.2 ± 0.6	4.0 ± 0.6	5.3 ± 0.7	3.0 ± 0.4	3.8
313665	Ecuador	Cultivated	WH	3.9 ± 0.6	4.4 ± 0.6	3.1 ± 0.5	N/A	3.8
201010	Guatemala	Landrace	CR	3.3 ± 0.6	N/A	4.6 ± 0.8	N/A	3.9
319618	Mexico	Landrace	CR/TN	2.8 ± 0.6	3.0 ± 0.6	5.1 ± 0.7	4.3 ± 0.4	4.1
533476	Mexico	Cultivated	BL	4.3 ± 0.6	4.1 ± 0.6	5.2 ± 0.7	2.9 ± 0.4	4.1
311999	Mexico	Landrace	RD	3.4 ± 0.6	5.3 ± 0.6	3.8 ± 0.5	N/A	4.2
533577	Ecuador	Cultivated	WH	4.3 ± 0.6	5.1 ± 0.6	5.1 ± 0.7	2.2 ± 0.4	4.2

Table 12. Overall mean of halo blight score in trifoliate and pod stages of 30 common bean accessions and 19 checks under greenhouse and field conditions. Origin, type, and seed color of each accession are also presented (continued).

Accession	Country of Origin	Type	Seed Color‡	Halo blight score (Mean ± S.E.†)				Overall Mean
				Field condition		Greenhouse condition		
				Trifoliate	Pod	Trifoliate	Pod	
200956	El Salvador	Landrace	BL	4.5 ± 0.6	5.8 ± 0.6	4.6 ± 0.7	2.1 ± 0.4	4.2
304110	El Salvador	Landrace	WH	4.7 ± 0.6	4.2 ± 0.6	5.4 ± 0.7	2.4 ± 0.4	4.2
209479	Nicaragua	Landrace	BL	2.1 ± 0.6	5.3 ± 0.6	6.1 ± 0.7	3.7 ± 0.4	4.3
308898	Costa Rica	Landrace	LT	3.1 ± 0.6	5.1 ± 0.6	5.8 ± 0.8	3.8 ± 0.4	4.4
476751	Guatemala	Cultivated	DR/RD	3.6 ± 0.6	6.5 ± 0.6	4.8 ± 0.7	3.3 ± 0.4	4.5
533475	Mexico	Cultivated	CR	4.1 ± 0.6	5.5 ± 0.6	5.8 ± 0.7	2.8 ± 0.4	4.5
207443	Colombia	Landrace	WH	4.3 ± 0.6	3.9 ± 0.6	4.9 ± 0.7	4.9 ± 0.4	4.5
313782	Mexico	Cultivated	BL	4.4 ± 0.6	5.7 ± 0.6	5.0 ± 0.7	3.1 ± 0.4	4.5
313254	Mexico	Landrace	BL	4.5 ± 0.6	4.4 ± 0.6	6.4 ± 0.7	2.7 ± 0.4	4.5
US14HBR6	UC-Davis; UI ǂ	Germplasm	PINTO¶	1.8 ± 0.6	4.2 ± 0.6	1.2 ± 0.7	1.5 ± 0.4	2.2
STAMPEDE	NDSU ǂ	Cultivar	PINTO¶	2.9 ± 0.6	5.2 ± 0.6	2.9 ± 0.8	2.2 ± 0.4	3.3
LARIAT	NDSU	Cultivar	PINTO¶	2.8 ± 0.6	5.0 ± 0.6	6.0 ± 0.7	1.5 ± 0.4	3.4
WINDBREAKER	Pinto	Cultivar	Seminis Seeds	3.2 ± 0.6	6.3 ± 0.6	2.5 ± 0.6	1.8 ± 0.2	3.4
ECLIPSE	NDSU ǂ	Cultivar	BL¶	2.2 ± 0.6	4.7 ± 0.6	5.7 ± 0.7	2.0 ± 0.4	3.6
LAPAZ	Provita	Cultivar	PINTO¶	2.0 ± 0.6	5.0 ± 0.6	6.5 ± 0.8	1.0 ± 0.4	3.7
SANTA CRUZ	Provita	Cultivar	PINTO¶	2.7 ± 0.6	4.7 ± 0.6	4.8 ± 0.7	3.3 ± 0.4	3.8
NORSTAR	NDSU	Cultivar	NAVY¶	2.2 ± 0.6	5.4 ± 0.6	4.5 ± 0.7	4.1 ± 0.4	4.0
SINALOA	Provita	Cultivar	PINTO¶	3.7 ± 0.6	4.4 ± 0.6	4.9 ± 0.7	3.4 ± 0.4	4.1
HIME	Hokkaido Pref. Tokachi Ag. Expt. Stat., Hokkaidao, Japan	Cultivar	OTEBO¶	3.2 ± 0.6	5.2 ± 0.6	6.4 ± 0.7	2.7 ± 0.6	4.4

Table 12. Overall mean of halo blight score in trifoliate and pod stages of 30 common bean accessions and 19 checks under greenhouse and field conditions. Origin, type, and seed color of each accession are also presented (continued).

Accession	Country of Origin	Type	Seed Color‡	Halo blight score (Mean ± S.E.†)				Overall Mean
				Field condition		Greenhouse condition		
				Trifoliate	Pod	Trifoliate	Pod	
MONTCALM	USDA-ARS/Michigan Ag. Expt. Stat.	Cultivar	DRK¶	4.0 ± 0.6	5.7 ± 0.6	7.0 ± 0.7	2.8 ± 0.4	5.2
FOXFIRE	Rogers/Syngenta	Cultivar	LRK¶	4.1 ± 0.6	6.1 ± 0.6	5.9 ± 0.7	2.7 ± 0.4	4.7
CLOUSEAU	Seminis Seeds	Cultivar	LRK¶	4.3 ± 0.6	6.0 ± 0.6	7.0 ± 0.7	3.5 ± 0.4	4.9
MAJESTY	Agriculture & Agri-Food Canada, Ontario, Canada	Cultivar	DRK¶	4.4 ± 0.6	5.3 ± 0.6	6.2 ± 0.8	2.2 ± 0.4	4.5
PINK PANTHER	Seminis Seeds	Cultivar	LRK¶	5.2 ± 0.6	6.6 ± 0.6	6.2 ± 0.8	4.1 ± 0.4	5.5
CABERNET	Seminis Seeds	Cultivar	DRK¶	5.3 ± 0.6	6.8 ± 0.6	5.8 ± 0.8	3.9 ± 0.4	5.4
RED HAWK	USDA-ARS/Michigan Ag. Exp. Stat.	Cultivar	DRK¶	6.3 ± 0.6	5.9 ± 0.6	7.2 ± 0.7	4.9 ± 0.4	6.1
CELRK	UC-Davis ǂ	Cultivar	LRK¶	6.6 ± 0.6	6.6 ± 0.6	6.3 ± 0.8	3.4 ± 0.4	5.7
RED ROVER	Seminis Seeds	Cultivar	DRK¶	6.6 ± 0.6	7.1 ± 0.6	7.3 ± 0.7	3.2 ± 0.6	6.0
Mean				3.6	5.1	4.3	3.2	3.9

‡ Seed color where: BL = black; BR = Brown; CR = Cream-beige; DY = dark yellow; LRK = Light red kidney; LT = Light tan; LY = Light yellow; WH = White; TN/LT = Tan/Light tan; CR/DP = Cream-beige/Dark purple; CR/RD = Cream-beige/Red; TN/BR/CR = Tan/Brown/Cream-beige; DR/RD = Red/Dark red; CR/PU/LB = Cream-beige/Purple/Light brown; PU/RD = Purple/Red; TN/BR = Tan/Brown; DE/TN = Dark Grey/Tan; WH/BR = White/Brown; CR/WH = Cream-beige/White; CR/DB = Cream-beige/Dark brown.

† Mean disease score ranging from 1 to 9 where1 -3 = resistant (R), 4-6 = intermediate (I), 7-9 = susceptible (S); S.E. = Standard error of the mean.

ǂ North Dakota State University; University of California-Davis; University of Idaho, Kimberly

¶ Represents the market class of respective commercial checks used. **N/A =** Accessions with missing pod score for halo blight.

Nine plant accessions, PI 203936, PI 207373, PI 209479, PI 311974, PI 313217, PI 313237, PI 313394, PI 313499, and PI 319618, had resistant reaction to trifoliate leaves with a mean disease score range of 1 to <3 (Table 12). Of these 9 accessions, 3 accessions PI 313217, PI 313237, and PI 313596, displayed resistant halo blight reaction with an overall mean disease score of 2.6 across unifoliate (primary), trifoliate, and pod stages, under both field and greenhouse conditions. The agronomic performance of 30 accessions and 19 commercial checks are presented in Appendix V. The standard resistant check, US14HBR6, had resistant reactions to both plant stages with a mean disease score of 2.2, a result consistent to that reported under greenhouse conditions. However, other resistant check, Montcalm, had an intermediate (5.0) reaction across both trifoliate and pod stages unlike under greenhouse conditions where the reaction was susceptible with a mean disease score of 7.0. Pink panther, a susceptible check, had an intermediate (6.0) disease reaction, a reaction like that observed under greenhouse conditions. The mean disease score of accessions over time across both growth stages and conditions is presented in figure 9.

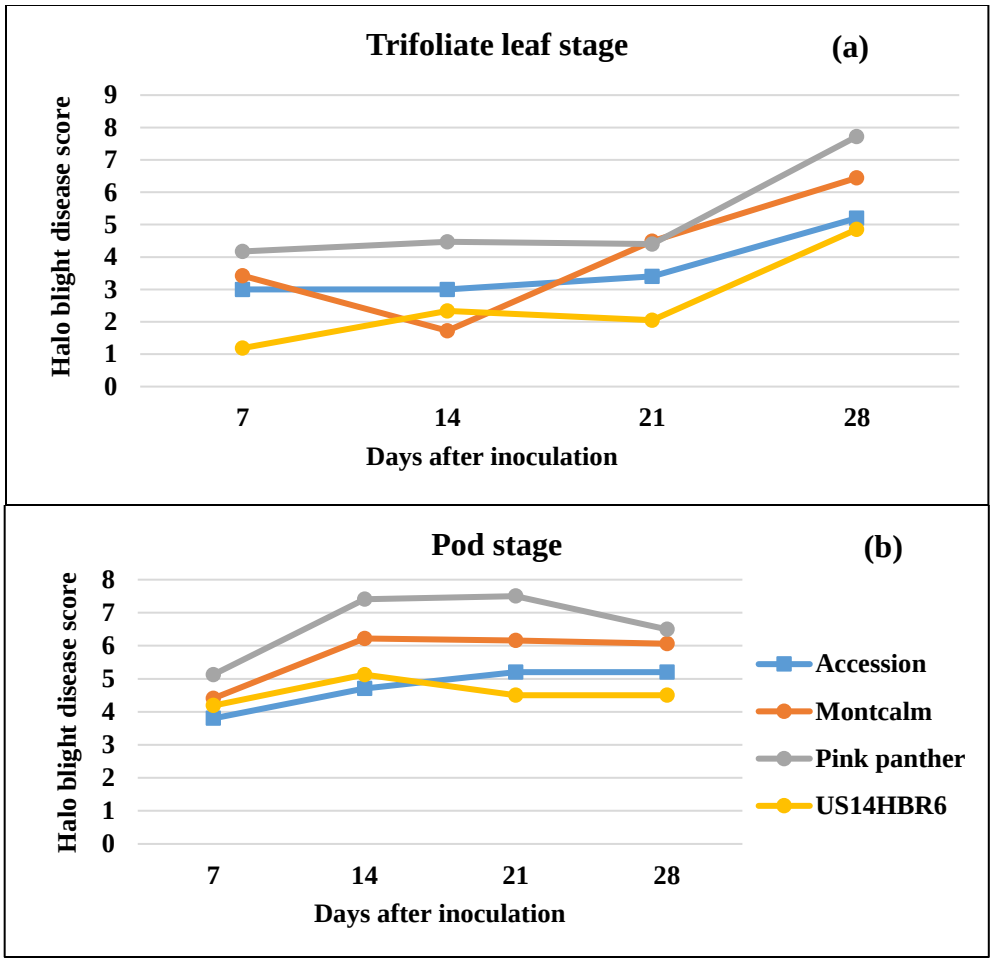

Figure 9. Mean halo blight severity of 49 common bean accessions and three commercial checks evaluated at 7, 14, 21 and 28 days post inoculation (dpi) under field condition. (a) Halo blight severity in trifoliate stage; (b) Halo blight severity in pod stage.

Following trifoliate inoculation, 49 accessions displayed resistant reaction until 14 days post inoculation (dpi) with a mean disease score of 3.0, where disease reaction was intermediate beginning at 21 dpi (3.4) until 28 dpi (5.2) (Figure 9a). In contrast, susceptible check, Pink Panther, had intermediate (4.4) reaction until 21 dpi, whereas susceptible (7.7) reaction was observed beginning at 21 dpi until 28 dpi (Figure 9a). Meanwhile, in the case of pod infection an intermediate disease reaction with a mean disease score of 4.7 was observed beginning 7 dpi until 28 dpi (Figure 9b). Despite a slight increase in halo blight severity in pods of commercial checks Montcalm and Pink Panther at 14 dpi, the disease severity remained consistent at 21 until

28 dpi. A significant and positive, but weak correlation was observed between the trifoliate and pod disease severity under field conditions, a result like that under greenhouse condition (Figure 10).

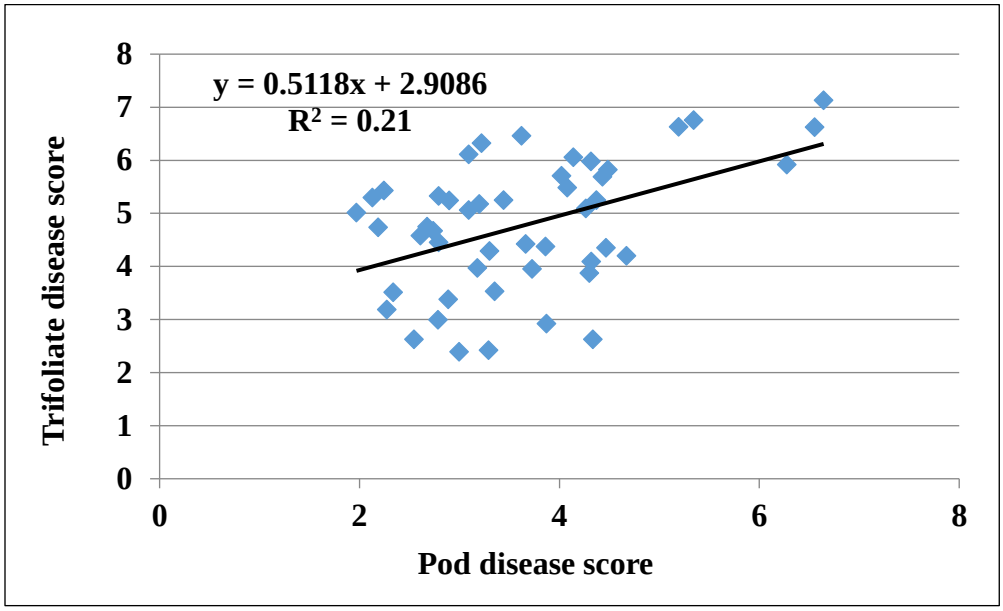

Figure 10. Linear regression of halo blight severity in trifoliate leaves and pods under field condition.

In summary, the halo blight severity following primary (unifoliate) leaf inoculation method under greenhouse conditions successfully categorized 281 accessions into resistant (37), intermediate (219) and susceptible (25). The mean and range of halo blight disease score in unifoliate leaves was 4.3 and 1.0-7.3. A selected group of 37 resistant and 25 susceptible accessions were further evaluated for trifoliate and pod disease symptoms under greenhouse conditions. The mean and range of halo blight disease score of 35 common accessions used for both unifoliate and trifoliate inoculation methods was compared. Significantly higher mean (2.0) disease score and range (1.0-2.4) was observed in trifoliate compared to 4.0 and 1.0-7.0, respectively in unifoliate stage. The significant positive but weak correlation ($r^2 = 0.1789$; $P <$

0.05) was observed between the halo blight severity in trifoliate leaves and pods within an individual plant. A total of twelve accessions had resistant reaction across both trifoliate and pod stages with a mean disease score of 1.8 and 2.0, respectively. Out of 12, 8 accessions exhibited consistent resistant reaction across all unifoliate, trifoliate and pod stages with an overall mean score of 2.1. Under field condition, the evaluation of halo blight severity in 49 accessions confirmed higher mean and range of disease score in pods compared to trifoliate leaves stage.

The mean and range disease score in pod and trifoliate was 4.7 and 2.3-7.1, and 3.6 and 2.0-6.6, respectively. The mean and range of disease score in trifoliate leaves of 30 common accessions evaluated under both greenhouse and field conditions were assessed.

A higher mean and range of disease score was observed in trifoliate stage under greenhouse condition, 5.3 and 1.2-7.3, respectively, in contrast to 3.0 and 1.0 -5.0, under field conditions. Field evaluation identified 3 accessions displaying consistent resistant reactions to halo blight severity across all unifoliate, trifoliate, and pod stages under both field and greenhouse conditions. Severity of halo blight over time showed accessions displaying resistant reaction until 14 dpi, whereupon the disease severity was intermediate from 21 until 28 dpi. In contrast, the halo blight severity in pod was intermediate from 7 to 28 dpi. Like that under greenhouse condition, a significant positive but weak correlation was observed between the disease severities in trifoliate versus pod stages.

Discussion

Among the evaluation of 281 *P. vulgaris* accessions from USDA-NPGS core collection, 37 accessions displayed resistant reactions to race 6 of *Psp* following primary (unifoliate) leaf inoculation method under greenhouse conditions. Commonly used for evaluating several bacterial (*P syringae* pv. *phaseolicola*), fungal (e.g. *Colletotrichum lindemuthianum* (Sacc. &

Magnus) Lams.-Scrib., *Uromyces appendiculatus* (Pers.: Pers.) Unger), and viral (e.g. Bean common mosaic virus, an aphid-vectored potyvirus) diseases of common bean, unifoliate leaf inoculation method in the past have successfully identified the resistant and susceptible genotypes at early plant stages saving costs, time, and effort (Mills and Silbernagel 1992; Strausbaugh et al., 2003; Terán et al., 2013). In this study, the successful categorization of disease reaction also revealed the effectiveness of multiple-needle florists pin frog inoculation method (Aggour et al., 1989; Mills and Silbernagel, 1992; Manzanera et al., 2006; Duncan et al., 2014). However, Singh and Muñoz (1999) reported the multiple-needle method as more labor intensive and time consuming especially when screening for large set of germplasm, and, thus emphasized the use of aspersion as preliminary method instead.

With approximately 56% of the total 37 resistant accessions originating from Mexico, it reflects the concentration of greater genetic diversity in the Mesoamerican gene pool (Beebe et al., 2000; Bitocchi et al., 2012; Singh et al., 1991). Based on the unifoliate inoculation method, five accessions were identified as resistant to halo blight with a mean score of 1.1. Plant breeders are continuously interested in these best performing accessions in terms of disease resistance, and exploit them as parents in the breeding program. One resistant accession, PI 207373 from Colombia, was also reported to be highly resistant to multiple races of *F. oxysporum* f. sp. *phaseoli*, i.e., race 1, 4, & 5 (Brick et al., 2006). Likewise, PI 290990 from Peru, was also reported to show intermediate reactions to white mold with score <5 in a rating scale of 1-9, where 1 = no disease symptoms, and 9 = total plant collapse (Miklas et al., 1999). Analysis and exploitation of such accessions with multiple-disease resistance to several important diseases should be of interest to breeders as it is considered an important and logical step towards gene pyramiding.

In the present study, the incidence of higher mean halo blight disease score using trifoliate inoculation method (4.0) compared to unifoliate method (2.0) suggests the importance of former method for the evaluation of diseases that appear late during plant development stages. The general findings of our trifoliate inoculation study also agree with the results reported by Lema et al., (2007), and Viteri and Singh, (2014) where higher common bacterial blight (*X. campestris* pv. *phaseoli* Smith (Dye) disease symptoms were found in trifoliate compared to primary (unifoliate) leaves inoculation method. However, the nature of the pathogen and their isolates used in both study may have different disease epidemiology, thus making it difficult to compare the results to the results from current experiment. This high incidence of disease in trifoliate can be explained by the complexion of modification in host-pathogen interactions with increasing plant age and microclimatic environment within host populations (Kora et al., 2005). Coyne and Schuster (1974) argued the possible weaker plant immune system for defense with increasing plant age may be due to extensive accumulation of phaseolotoxin, a secondary plant metabolite, in trifoliate leaf tissues. In addition, many pathogens also secrete enzymes that enhance virulence by detoxifying secondary metabolites or that can interfere with plant cell signaling (Duca et al., 2014; Fan et al., 2011). Significant differences in halo blight severity observed in the current study following trifoliate inoculation method explain its power for facilitating the separation among resistant, intermediate and susceptible accessions. Nonetheless, the results also suggest the practical application of identifying and eliminating susceptible genotypes following unifoliate inoculation method in early stages of plant development, and then confirm resistance of the selected group using trifoliate method. However, this approach is particularly feasible when dealing with small number of genotypes, thus saving considerable amount of time and expenses. The resistant reaction in US14HBR6 was as expected, i.e., 1.2,

however intermediate reaction in Pink Panther across both unifoliate (6.5) and trifoliate (6.2) inoculation method may suggest an evidence of disease tolerance mechanism in the cultivar. This could be explained as one of the reasons why Pink Panther persists as a popular cultivar widely grown in Minnesota regions without apparent significant reductions in seed yield.

The findings from this study displayed the differential halo blight severity in trifoliate versus pod stages when evaluated within an individual plant, and suggest the role of potential independent genes governing for resistance at respective plant growth stages. Acevedo-Román et al. (2004) and Velez et al. (1998) found similar results that reported two specific genes at two plant stages controlling for resistance to bean golden yellow mosaic virus (BGYMV) in common bean, i.e., *bgm-1*, a recessive gene, conferred resistance to leaf chlorosis, while *Bgp-1*, a dominant gene, prevented pod deformation. Similarly, Osorno et al., (2007) also identified additional genes for BGYMV, namely *bgm-3* and *Bgp-2* that conferred resistance to leaf chlorosis and prevented pod deformation, respectively. The present study also identified a group of accessions, i.e., PI 201343, PI 313217, PI 313328, PI 313343, PI 313490, PI 313596, PI 325653, and PI 417657, with high levels of resistance across all plant developmental stages including unifoliate, trifoliate leaf, and pod stages. Therefore, further efforts on the evaluation and validation of these genotypes to other races of *Psp* would provide practical implications on broad-spectrum resistance as well as strain-specific resistance.

Despite the limitations in disease assessment especially when large number of cultivars are involved across locations, field disease evaluation correlates true to environmental variation as well as host phenology (Hernandez et al., 1993). For this purpose, the field evaluation was conducted to validate the performance of accessions observed under greenhouse conditions for halo blight severity. The pod stage had higher mean disease score (4.7) compared to trifoliate

stage (3.6) suggesting the prevalence of higher disease pressure in later plant development stage. This finding could be informative indicator for farmers and plant breeders in regards to careful inspection of plants during later pod filling stages. However, the results were inconsistent when compared with greenhouse conditions where higher mean disease score of 5.3 was reported in trifoliate stage than in pod stage (3.0). This inconsistency in halo blight in terms of halo blight severity may be due to major impacts of several environmental factors, such as light, temperature, rainfall, humidity, and other pathogens predominant under field conditions. Our results were also like that reported by Baggett and Frazier, (1967), and Coyne et al. (1967), which reported inconsistency in halo blight severity when evaluated under greenhouse versus field conditions. Nine accessions, PI 203936, PI 207373, PI 209479, 311974, PI 313217, PI 313237, PI 313394, PI 313499, and PI 319618, displayed resistance to trifoliate leaves under field conditions. Among these accessions, Brick et al. (2006), in a separate study, reported PI 207373 resistant to several races of *F. oxysporum* f. sp. *phaseoli* including race 1, 4 & 5. Therefore, this accession should be considered for future breeding for multiple-disease resistance. Nonetheless, field evaluation also successfully identified three accessions, i.e., PI 313217, PI 313237, and PI 313596, that displayed resistant disease reactions across all unifoliate, trifoliate, and pod stages under both greenhouse and field conditions. Resistance from this group of accessions could be introgressed into common bean breeding program aimed for developing resistance to race 6 of *Psp*.

In an attempt to evaluate the halo blight severity over the time under field condition, the disease scores were evaluated at 7, 14, 21, and 28 days post inoculation (dpi) at weekly intervals. However, it is important to note that the results presented here is based on one-year field evaluation across one location only. The accessions showed resistant reactions to trifoliate

inoculation from 7 dpi until 14 dpi after which the reaction was intermediate until 28 dpi. The incidence of resistant trifoliate reaction at early developmental stages may provide enough time for plant to overcome disease pressure especially in a very sensitive stage such as in young seedlings. This results in trifoliate reaction was consistent to findings by Durham et al., (2013) who reported the higher CBB symptoms around 20 dpi and clearly facilitated the separation in the levels of disease reaction. However, unlike to our findings, Mazzola et al., (1994) concluded that resistance genes, *Xa21*, to bacterial blight in rice (*Xanthomonas oryzae* pv. *oryzae*) was expressed at 21 dpi instead of early development stages such as V0 (germination) and V1 (emergence).

To differentiate or correlate the levels of halo blight severity in trifoliate leaves versus pods within an individual plant, a simple linear regression calculated under greenhouse condition was compared with that under field environment. Similar results observed under greenhouse condition was detected under field condition, and hold true for the hypothesis that different genes and/or mechanisms at different plant growth stages controls for halo blight resistance despite of environmental conditions under evaluation. However, as the findings from this study are based on only one year field evaluation, the additional screening of resistant accessions across several locations would help to estimate genotype by environment (G x E) interaction.

In summary, the identification of very few sources of resistance to race 6 of *Psp* indicates that there is an immediate need to explore for resistance in more diverse germplasm including exotic lines, landraces, and wild common bean. The results from both greenhouse and field experiments following unifoliate, trifoliate and pod inoculation method demonstrated the effectiveness of identifying common bean accessions with novel sources of resistance to race 6 of *Psp*. Three accessions, PI 313217, PI 313237, and PI 313596, carrying broad-spectrum

resistance across all plant developmental stages, and environments could be of excellent choice as parental lines in breeding program aimed for resistance to race 6 of *Psp*.

References

Acevedo-Román, M., A.M. Molina-Castańeda, J.C. Angel Sánchez, C.G. Muñoz, and J.S. Beaver. 2004. Inheritance of normal pod development in bean golden yellow mosaic resistant common bean. J. Amer. Soc. Hort. Sci. 129:549-552.

Aggour, A.R., D.P. Coyne, and A.K. Vidaver. 1989. Comparison of leaf and pod disease reactions of beans (*Phaseolus vulgaris* L.) inoculated by different methods with strains of *Xanthomonas campestris* pv. *phaseoli* (Smith) Dye. Euphytica 43:143-152.

Ariyarathne, H.M., D.P. Coyne, A.K. Vidaver, and K.M. Eskridge. 1998. Selection for common bacterial blight resistance in common bean: Effects of multiple leaf inoculation and detached pod inoculation test. J. Amer. Soc. Hort. Sci. 123:864-867.

Arnaud-Santana, E., D.P. Coyne, K.M. Eskridge, and A.K. Vidaver. 1994. Inheritance, low correlations of leaf, pod, and seed reactions to common bacterial disease in common beans, and implications for selection. J. Am. Soc. Hortic. Sci. 119:116-121.

Baggett, J.R., and W.A. Frazier. 1967. Sources of resistance to halo blight in *Phaseolus vulgaris*. Plant Dis. Rep. 51:661-665.

Beebe, S., P.W. Skroch, J. Tohme, M.C. Duque, F. Pedraza, and J. Nienhuis. 2000. Structure of genetic diversity among common bean landraces of Middle American origin based on correspondence analysis of RAPD. Crop Sci. 40:264-273.

Bitocchi, E., L. Nanni, E. Bellucci, M. Rossi, A. Giardini, P.S. Zeuli, and R. Papa. 2012. Mesoamerican origin of the common bean (*Phaseolus vulgaris* L.) is revealed by sequence data. PNAS 109: E788-E796.

Bozkurt, I.A., and S. Soylu. 2011. Determination of responses of different bean cultivars against races of *Pseudomonas syringae* pv *phaseolicola*, causal agent of halo blight of bean. Euphytica 179:417-425.

Brick, M.A., P.F. Byrne, H.F. Schwartz, J.B. Off, K. Otto, A.L. Fall, and J. Gilbert. 2006. Reaction to three races of Fusarium wilt in the *Phaseolus vulgaris* core collection. Crop Sci. 46:1245-1252.

Chantret, N., D. Mingeot, P. Sourdille, M. Bernard, J.M. Jacquemin, and G. Doussinault. 2001. A major QTL for powdery mildew resistance is stable over time and at two development stages in winter wheat. Theor. Appl. Genet. 103:962-971.

Coyne, D.P., and M.L. Schuster. 1974. Inheritance and linkage relations of reaction to *Xanthomonas phaseoli* (E.F. Smith) Dowson (common blight), stage of plant development and plant habit in *Phaseolus vulgaris* L. Euphytica 23:195-204.

Coyne, D.P., M.L. Schuster, and R. Fast. 1967. Sources of tolerance and reaction of beans to races and strains of halo blight bacteria. Plant Dis. Rep. 51:20-24.

Duca, D., D.R. Rose, and B.R. Glick. 2014. Characterization of a nitrilase and a nitrile hydratase from *Pseudomonas* sp. strain UW4 that converts indole-3-acetonitrile to indole-3-acetic acid. Appl. Environ. Microbiol. 80:4640-4649.

Duncan, R.W., M. Lema, R.L. Gilbertson, and S.P. Singh. 2014. Registration of common bean pinto US14HBR6 resistant to race 6 of the halo blight pathogen, *Pseudomonas syringae* pv. *phaseolicola*. J. Plant Reg. 8:53-56.

Durham, K.M., W. Xie, K. Yu, K.P. Pauls, E. Lee, and A. Navabi. 2013. Interaction of common bacterial blight quantitative trait loci in a resistant inter-cross population of common bean. Plant Breeding. 132:658-666.

Fan, J., C. Crooks, G. Creissen, L. Hill, S. Fairhurst, P. Doerner, and C. Lamb. 2011. *Pseudomonas* sax genes overcome aliphatic isothiocyanate-mediated nonhost resistance in *Arabidopsis*. Science 331:1185-1188.

Ficke, A., D.M. Gadoury, R.C. Seem, and I.B. Dry. 2003. Effects of ontogenic resistance upon establishment and growth of *Uncinula necator* on grape berries. Phytopathology 93:556-563.

Foolad, M.R., M.T. Sullenberger, and H. Ashrafi. 2015. Detached-leaflet evaluation of tomato germplasm for late blight resistance and its correspondence to field and greenhouse screenings. Plant Disease 99:718-722.

Harper, S., N. Zewdie, I.R. Brown, and J.W. Mansfield. 1987. Histological, physiological and genetical studies of the responses of leaves and pods of *Phaseolus vulgaris* to three races of *Pseudomonas syringae* pv. *phaseolicola* and to *Pseudomonas syringae* pv. *coronafaciens*. Physiol. Mol. Plant P. 31:153-172.

Hernandez, C.M., J. Crossa, and A. Castillo. 1993. The area under the function: an index for selecting desirable genotypes. Theor. Appl. Genet. 87:409-415.

Hergert, M. 2010. Halo blight reduces Wyoming seed production. Northarvest Beangrower Mag. 16:31-32.

Hill, K., D.P. Coyne, and M.L. Schuster. 1972. Leaf, pod, and systemic chlorosis reactions in *Phaseolus vulgaris* to halo blight controlled by different genes. J. Amer. Soc. Hort.Sci. 97:494-498.

Jeger, M.J., and S.L.H. Viljanen-Rollinson. 2001. The use of the area under the disease-progress curve (AUDPC) to assess quantitative disease resistance in crop cultivars. Theor. Appl. Genet. 102:32-40.

King, E.O., M.K. Ward, and D.E. Raney. 1954. Two simple media for demonstration of pyocyanin and fluorescein. J. Lab. Clin. Med. 44:301–307.

Knodel, J.J., P.B. Beauzay, D.W. Franzen, H.J. Kandel, S.G. Markell, J.M. Osorno, and R.K. Zollinger. 2015. 2014 Dry bean grower survey of pest problems and pesticide use in Minnesota and North Dakota. NDSU Ext. Serv. Doc. E-1522, Fargo, ND.

Kora, C., M.R. McDonald, and G.J. Boland. 2005. Epidemiology of sclerotinia rot of carrot caused by *Sclerotinia sclerotiorum*. Can. J. Plant Pathol. 27:245-258.

Lema, M., H. Teràn, and S.P. Singh. 2007. Selecting common bean with genes of different evolutionary origins for resistance to *Xanthomonas campestris* pv. *phaseoli*. Crop Sci. 47:1367-1374.

Manzanera, A.S., M. Carmen, C. Asensio, and S.P. Singh. 2006. Gamete selection for resistance to common and halo bacterial blights in dry bean intergene pool populations. Crop Sci. 46:131-135.

Mazzola, M., J.E. Leach, R. Nelson, and F.F. White. 1994. Analysis of the interaction between *Xanthomonas oryzae* pv. *oryzae* and the rice cultivars IR24 and IRBB21. Phytopathology 84:392-397.

Miklas, P.N., R. Delorme, R. Hannan, and M. 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.

Mills, L.J., and M.J. Silbernagel. 1992. A rapid screening technique to combine resistance to halo blight and bean common mosaic virus in *Phaseolus vulgaris* L. Euphytica 58:201-208.

Osorno, J.M., C.G. Muñoz, J.S. Beaver, F.H. Ferwerda, M.J. Bassett, P.N. Miklas, T. Olczyk, and B. Bussey. 2007. Two genes from *Phaseolus coccineus* confer resistance to bean golden yellow mosaic virus in common bean. J. Am. Soc. Hort. Sci. 4:530-533.

Porch, T.G., K. Cichy, M.A. Pastor-Corrales, M.A. Grusak, J.S. Beaver, J. Hart, D. Fourie, C. Estevez de Jensen, S. Nchimbi, and P.N. Miklas. Characterization and application of the Andean Diversity Panel for the improvement of common bean productivity in Sub-Saharan Africa. Poster session presented at the meeting of the Pan-African Grain Legume & World Cowpea Conference, 2016 February 28 – March 4, Livingstone, Zambia.

Singh S.P., P. Gepts, and D.G. Debouck. 1991. Races of common bean (*Phaseolus vulgaris* Fabaceae). Econ. Bot. 45:379-396.

Singh, S.P., and C.G. Muñoz. 1999. Resistance to common bacterial blight among *Phaseolus* species and common bean improvement. Crop Sci. 39:80-89.

Singh, S.P., and H.F. Schwartz. 2010. Breeding common bean for resistance to diseases: A review. Crop Sci. 50:2199-2223.

Steel, R.G.D., and J.H. Torrie. 1980. Principles and procedures of statistics: a biometrical approach. New York, Toronto, London: McGraw-Hill Book Company.

Strausbaugh, C.A., J.R. Myers, R.L. Forster, and P.E. McClean. 2003. A quantitative method to screen common bean plants for resistance to bean common mosaic necrosis virus. Phytopathology 93:1430-1436.

Taylor, J.D., D.M. Teverson, M.A. Allen, and M.A. Pastor-Corrales. 1996a. Identification and origin of races of *Pseudomonas syringae* pv. *phaseolicola* from Africa and other bean growing areas. Plant Pathol. 45:469-478.

Taylor, J.D., D.M. Teverson, and J.H.C. Davis. 1996b. Sources of resistance to *Pseudomonas syringae* pv. *phaseolicola* races in *Phaseolus vulgaris.* Plant Pathol. 45:479-485.

Terán, H, C. Jara, G. Mahuku, S. Beebe, and S.P. Singh. 2013. Simultaneous selection for resistance to five bacterial, fungal, and viral diseases in three Andean 9 Middle American inter-gene pool common bean populations. Euphytica 189:283-292.

Trabanco, N., M.C. Asensio-Manzanera, E. Perez-Vega, A. Ibeas, A. Campa, and J.J. Ferreira. 2014. Identification of quantitative trait loci involved in the response of common bean to *Pseudomonas syringae* pv. *phaseolicola*. Mol. Breeding 33:577-588.

USDA-NRCS. 2016. Web soil survey. National resources conservation service. http://websoilsurvey.nrcs.usda.gov (accessed June 6, 2016).

Velez, J.J., M.J. Bassett, J.S. Beaver, and A. Molina. 1998. Inheritance of resistance to bean golden mosaic virus in common bean. J. Am. Soc. Hort. Sci. 4:628-631.

Viteri, D.M., H. Teran, A.S. Manzanera, M. Carmen, C. Asensio, T. Porch, P.N. Miklas, and S.P. Singh. 2014. Progress in breeding Andean common bean for resistance to common bacterial blight. Crop Sci. 54:2084-2092.

Wang, Z.L., L.H. Li, Z.H. He, X.Y. Duan, Y.L. Zhou, X.M. Chen, M. Lillemo, R.P. Singh, H. Wang, and X.C. Xia. 2005. Seedling and adult plant resistance to powdery mildew in Chinese bread wheat cultivars and lines. Plant Disease. 89:457-463.

Zaiter, H.Z., and D.P. Coyne. 1984. Testing inoculation methods and sources of resistance to the halo blight bacterium (*Pesudomonas syringae* pv. *phaseolicola*) in *Phaseolus vulgaris*. Euphytica 33:133-141.

Zhang, J.Z., R.A. Creelman, and J.K. Zhu. 2004. From laboratory to field. Using information from Arabidopsis to engineer salt, cold, and drought tolerance in crops. Plant Physiol. 135:615-621.

CHAPTER II. GENOME-WIDE ASSOCIATION STUDY FOR HALO BLIGHT DISEASE RESISTANCE TO RACE 6 OF *PSP* IN USDA-NPGS COMMON BEAN CORE COLLECTION

Abstract

Halo blight, caused by *Pseudomonas syringae* pv. *phaseolicola* (*Psp*), is an important seed-borne bacterial disease of common bean (*Phaselous vulgaris* L.) causing up to 45% yield losses. Both quantitative and qualitative resistance to different races of *Psp* including race 6, the most important and predominant race worldwide, have been reported in bi-parental mapping populations. However, no study has been conducted to explore novel sources of resistance to race 6 of *Psp* in the USDA-NPGS core collection of common bean. Therefore, this study aims to identify the genomic regions associated with halo blight disease resistance using genome-wide association study (GWAS). A total of 197 accessions and 4707 SNP markers were used to map resistance to race 6 of *Psp*. Three significant regions, two in Pv04, and one in Pv05, controlling for 19% phenotypic variation were found associated with resistance to race 6 of *Psp*. Unique SNPs, sc00112ln569344_270381_C_T_ and sc00835ln140787_67166_T_C_, identified in genomic region of Pv04 may be effective for marker-assisted selection (MAS) in common bean breeding program aimed for resistance to race 6 of *Psp*. Most accessions carrying the favorable allele contributing to halo blight disease resistance were from Andean origin. The results from the current study also identified candidate genes involved in biochemical defense pathway and its potential role in resistance to race 6 of *Psp*. This is the first report localizing the genomic regions against halo blight in USDA-NPGS core collection and the further validation of these markers in MAS to race 6 of *Psp* is warranted more research.

Introduction

Common bean (*Phaseolus vulgaris* L.) is an important annual grain legume, mainly in Asia, Africa, Caribbean, and Latin America (Beebe et al., 2012). Because it is a major source of quality protein and contains high concentrations of carbohydrates, fiber, mineral, and vitamins, common bean contributes a significant part to human diets (Broughton et al., 2003). However, among several bacterial diseases of common bean, halo blight, caused by *Pseudomonas syringae* pv. *phaseolicola* (*Psp*), is an important limitation factor for achieving high seed yield throughout the world, including the US (Taylor et al., 1996a, 1996b). In Wyoming in 2009, halo blight caused a significant loss of common bean growing fields dedicated to certified seed production showing its importance as an endemic disease (Hergert, 2010). Nine different races of *Psp* identified based on a set of eight differential bean cultivars represents the prevalent genetic variability of the disease (Taylor et al., 1996a). Because chemical control method is not the most cost-effective and environmentally friendly approach for disease management, the development and use of resistant cultivars is crucial for any breeding programs. Using bi-parental mapping (linkage mapping), five race-specific resistant genes referred as *Pse-1*, *Pse-2*, *Pse-3*, *Pse-4*, and *Pse-5* genes have been identified from a set of eight differential cultivars (Taylor et al., 1996b; Ariyarathne et al., 1999; Fourie et al., 2004; Miklas et al., 2009; 2011). These genes confer resistance to several races of *Psp* except to race 6, one of the most important and predominant races of *Psp* worldwide. However, until recently, limited studies have reported the sources of resistance to race 6 of *Psp*, thus realizing the need for investigating additional sources of resistance (Duncan et al., 2011; 2014; Porch et al., 2016; Trabanco et al., 2014).

Because QTLs identified from bi-parental mapping population may contain narrow genetic information due to limited allelic diversity, genome-wide association mapping (GWAS)

or association mapping (AM) in recent years have become an alternative approach to detect genomic regions associated with target traits (Abdurakhmonov and Abdukarimov, 2008; Jannink et al., 2001; Nordborg and Tavare, 2002). In common bean, AM has been successfully used to identify genomic regions associated to various agronomically important traits through genome-wide scans (Kamfwa et al., 2015; Nemli et al., 2014; Perseguini et al., 2016; Shi et al., 2011). Additionally, AM has also been widely employed to understand population structure and genetic diversity existing among two gene pools, Andean and Middle American, in common bean (Beebe et al., 2000; Bitocchi et al., 2012; Blair et al., 2009; Mamidi et al., 2011; 2013; McClean et al., 2012). Nonetheless, the importance of GWAS lies in the efficient assessment of genetically diverse germplasm for identifying the novel sources of resistance for several economically important traits. Germplasm maintained in the gene banks represent and preserve valuable sources of crop genetic variation essential for genetic improvement of any crop species especially during genetic erosion (Blair et al., 2010; Broughton et al., 2003). Despite few studies have focused on the evaluation of the USDA-NPGS common bean core collection for resistance to several common bean diseases (Brick et al., 2006; Miklas et al., 1999), no study has attempted to develop genetic resistance to race 6 of *Psp* using core collection. Therefore, the main objective of the study was to identify the genomic regions controlling for resistance to race 6 of *Psp* using GWAS approach in the USDA-NPGS common bean core collection.

Materials and Methods

Plant Materials

A total of 383 accessions from USDA-NPGS/ARS Western Regional Plant Introduction Station, Pullman, WA were evaluated for trifoliate reactions to race 6 of *Psp* under greenhouse

conditions. These 383 accessions were comprised of 281 accessions used for unifoliate leaves inoculation method (Chapter I) and remaining 102 photoperiod-sensitive accessions.

For this experiment, the entire set (383) of core collection was screened, and its phenotypic data were used for GWAS analyses because of the following reasons; (i) in preliminary study, the GWAS results obtained using the phenotypic data from 281 accessions (following unifoliate leaf inoculation; Chapter I) did not yield strong genomic regions associated to disease resistance (data not shown), and (ii) the GWAS analyses using the phenotypic data following trifoliate inoculation method was not feasible as data were collected from only 81 accessions (Chapter I) and thus were not used for GWAS analyses.

The core collection used for this study included 206 accessions from Mexico, 42 from Colombia, 31 from Guatemala, 24 from Peru, 22 from Ecuador, 14 from Costa Rica, 15 from Nicaragua, 14 from El Salvador, 9 from Honduras, 5 from Bolivia, and 1 from Iran.

Experimental Design

An experiment was arranged at the Agricultural Experiment Station (AES) at NDSU, Fargo, ND greenhouse facilities during the spring of 2015. The experimental design was conducted using a randomized complete block design (RCBD) with four replications. Pinto germplasm US14HBR6, and Montcalm DRK, were considered resistant checks, and Pink Panther LRK as susceptible (Duncan et al., 2008; 2014).

One seed of each accession and check was scarified with a needle to ensure 100% germination before planting. Plastic trays (15.2 × 15.2 ×15.2 cm diameter) each containing 50 circled compartments filled with #1 Sunshine Mix Media (3:1 peat moss: perlite) (SunGro Horticulture; Bellvue, WA) were used. To facilitate the disease scoring process due to dense planting, seeds were sown in 25 compartments leaving every other compartment. Slow release

fertilizer (Osmocote 20 – 20 – 20 N – P – K, 2.5 g per compartment) was applied to 2-weeks-old seedlings (Scotts-Sierra Horticultural Product Company, Maryville, OH, USA).

Source of Inoculum

The procedure for inoculum preparation was like that explained in previous chapter I.

Bacterial Inoculation

Due to the observed higher disease symptoms at trifoliate leaf stages from previous experiment (see Chapter I), accessions in this experiment were evaluated using trifoliate inoculation. Trifoliate at the second trifoliate leaf stage, i.e., at 21 DAP, were inoculated along with checks using multiple-needle pin frog method as described in previous chapter I.

Inoculated plants were rated for halo blight infection 10 days post-inoculation (dpi) using an alternative rating scale of 1 – 5 (Innes et al., 1984; Taylor et al., 1996a), where 1 = red-brown necrotic reaction in the area of maximum inoculation (highly resistant); 2 = red-brown necrotic reaction with a trace of water soaking (resistant); 3 = some necrosis but extensive water-soaking in the area of maximum inoculation (slightly susceptible); 4 = small water-soaked lesion of <1 mm diameter distributed at random across the leaf underside (susceptible); and 5 = larger water-soaked lesions of about 1-3 mm diameter distributed at random across the leaf underside (highly susceptible) (Figure 11). Unlike previous experiment (Chapter I), disease symptoms in this experiment were evaluated using a rating scale of 1-5 because of the following reasons: As the preliminary GWAS results obtained using phenotypic data with disease rating scale of 1-9 (Mills and Silbernagel, 1992) did not show significant genomic regions associated with disease resistance. This was caused in part because the phenotypic data on the disease score using 1-9 rating scale did not follow a normal distribution and therefore, it was decided to use the alternative disease score scale of 1-5 that did follow a normal distribution.

Statistical Analysis

PROC MIXED (SAS Institute Inc. 2012. SAS/STAT® 9.3 User's Guide. Cary, NC) was used to analyze halo blight disease scores on trifoliate. Replications and plant accessions were considered as random and fixed effects, respectively. Lsmeans or adjusted means were separated using *pdiff* to classify categories of resistance between accessions at ($\alpha = 0.05$) level of significance. A normality test for the data was performed using the Kolmogorov-Smirnov test with $p < 0.05$ used to indicate lack of fit.

Proc t-test was used to determine significance of two alleles from each significant SNP markers. Mean halo blight scores were compared for two alleles of significant SNPs using LSD test ($\alpha = 0.05$).

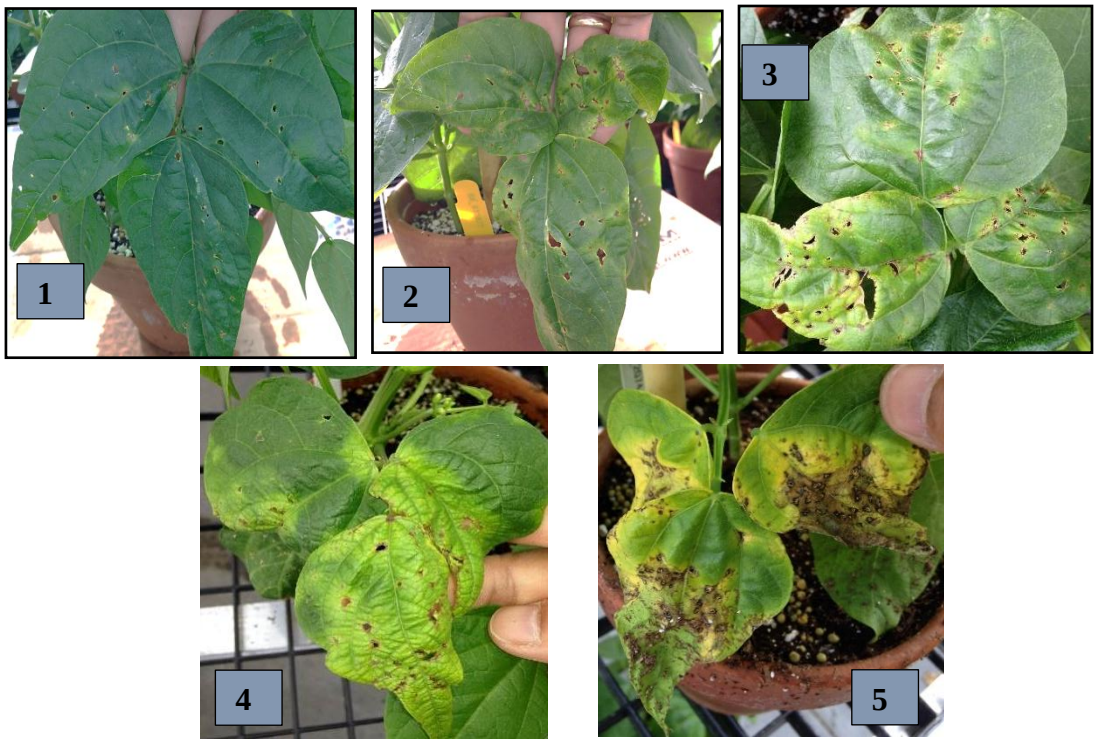

Figure 11. Halo blight severity evaluated in trifoliate leaves of common bean accessions 10 days post inoculation (dpi). The number inside the picture denotes the disease rating scale of 1-5, where 1= highly resistant, 2= resistant, 3= slightly susceptible, 4= susceptible, and 5= highly susceptible.

Genotyping and Marker Selection

For SNP genotyping, a total of 422 accessions were grown and DNA samples were extracted in the laboratory of Dr. Paul Gepts at University of California, Davis, CA. Extracted DNA samples were then sent to Dr. Perry Cregan laboratory in USDA-ARS, Beltsville, MD for genotyping. The lines were genotyped using a beanCAP 6K SNP chip (BARCBean6k_3 BeadChip; designed using the Illumina Infinium BeadChips system) with 5398 SNPs that were evenly distributed across the 11 pairs of common bean chromosome (Hyten et al., 2010; Song et al., 2015). However, the genotypic file obtained from Dr. Cregan laboratory contained SNP information of only 357 accessions because there was missing genotypic information for 65 accessions. These 65 accessions were not genotyped because of mixed reasons such as (i) poor germination rate, (ii) low DNA quality, and (iii) low genotyping quality (P. Gepts, personal communication).

However, of these remaining 357 accessions, 160 accessions were not included for GWAS analyses because they either lacked genotypic information or phenotypic data. Therefore, only 197 accessions were used for GWAS analyses as both genotypic and phenotypic information of these accessions were available.

Association Mapping Procedures

Data Imputation and Minor Allele Frequency

Data imputation for missing data was performed using the software fastPHASE v. 1.2, using maximum likelihood-based imputation (Scheet and Stephens, 2006). Out of total 5398 SNP markers, a set of 4988 markers remained after removing markers with more than 50% missing data. The 4988 markers were then filtered to remove SNP markers with a minor allele frequency (MAF) <0.05 and remaining 4707 markers were used for the GWAS (genome-wide

association study) analysis. GWAS was conducted with remaining polymorphic markers using GAPIT (Genome Association and Prediction Integrated Tool) in the R software (Zhang et al., 2010; Lipka et al., 2012).

Principal Component Analysis and Marker-Trait Association Tests

To control number of false positives generated due to population structure, family relatedness and structured association, principal component analysis (PCA) was employed as covariates for association analysis, where smaller set of correlated variables are converted into smaller set of uncorrelated variables known as principal components (PCs) (Larsson et al., 2013). Three PCs (PCA3) and top ten PCs (PCA10), which explained more than 50% of total variation were used as covariate in the association analysis. To identify marker-trait associations, six different models were assessed: Naïve, Efficient Mixed Model Association (EMMA), PCA + EMMA (Mixed Model), PCA3 + EMMA (Mixed Model 3), Principal Component Analysis 3 (PCA3), and Principal Component Analysis 10 (PCA10) were tested using GAPIT (Genome Association and Prediction Integrated Tool) in the R software (R Development Core Team 2015). These approaches helped address the spurious or false positive marker-trait association due to population structure. The statistical description of the models is presented in Table 13, where naïve model does not consider for population structure and family relatedness, and is based upon the Bayesian model (Mamidi et al., 2011). EMMA corrects for both population structure and genetic relatedness (Kang et al., 2008), whereas PCA + EMMA accounts for both population structure and family relatedness (K) correcting for cryptic relatedness in the panel. The final number of principal components (PCs) that adequately explain population structure was determined through spree plot generated by GAPIT.

For the presentation of results, based on the QQ (quantile-quantile) – plot, the models that visually showed less deviation of observed *p*-value from the expected *p*-value were considered as the best model. This model is likely to sufficiently account for spurious association due to population structure and familial relatedness and consider of false positive rates (Stich et al., 2008). In addition, mean square difference (MSD) was also calculated to select for best model for GWAS using formula described in Mamidi et al., (2011). The model that showed lowest MSD (i.e., approaching zero) value represented the best model, and significant markers from that respective model were selected. From the best model selected, graphical representation of the genomic position of each SNP markers are presented in the Manhattan plot. Genomic regions that have strong correlation with the trait of interest are identified via large peaks in the Manhattan plot. The Manhattan plots were constructed using negative logarithm (-log10) of the *p*-value in which large peaks in a chromosome(s) suggest significant marker-trait associations with strong correlation.

Table 13. The statistical description of the association mapping model components used in the study (Adapted from Mamidi et al., 2011).

Model	Statistical model‡	Description
Naïve	$y = X\alpha + e$	Model does not account for population structure
EMMA	$y = X\alpha + Kv + e$	Model corrects for both population structure and genetic relatedness
PCA + EMMA	$y = X\alpha + p\beta + Kv + e$	Model controls for both population structure and familial relatedness
PCA3	$y = X\alpha + p\beta + e$	Model accounts only for population structure using the first three dimensions of multidimensional scaling
PCA10	$y = X\alpha + p\beta + e$	Model accounts only for population structure using the first ten dimensions of multidimensional scaling
PCA3 + EMMA	$y = X\alpha + p\beta + Kv + e$	Model controls for both population structure and familial relatedness using the first three dimensions of multidimensional scaling

Where, ‡ y is the response vector of observed phenotypes, X denotes genotypes at the marker, α is a vector for the fixed effects related to SNP marker effects, β is a vector for the fixed effects related to population structure, ν is a vector of random effects related to familial relatedness, and e is a vector for the residual effects, p denotes the four dimensions from the multidimensional scaling, and K is the relationship matrix.

In addition, the significance of these SNP markers and their chromosomal positions identified in Manhattan plots are validated from the GWAS result file (excel sheet). The Bonferonni corrected $p = 1.1 \times 10^{-5}$ (for $\alpha = 0.05$ and 4707 SNPs) was used as a cutoff for determining the significance threshold for SNPs. The SNP with the lowest *p*-value was reported for each significant locus, and was considered a significant SNP marker. Surrounding genes to each significant SNP were checked for their physiological function using the *Phaseolus vulgaris* genome version 1.0 (Schmutz et al., 2014) and functional annotation on Phytozome v10 (http://www.phytozome.net) (Goodstein et al., 2012). Later, the phenotypic variation (R^2) of each significant marker(s) was identified using stepwise regression in SAS 9.3 (SAS Institute, Inc. 2011). Information on significant SNP marker(s) and the corresponding genes near, i.e., up

to 100 kbp upstream and downstream to that specific marker, potentially causing the phenotypic effect, were considered putative candidate genes. These candidate gene(s), if any in previous study have been reported to be responsible for halo blight disease resistance in common beans or any other crop species was further used to confirm the significance of that gene associated with trait of interest, i.e., resistance to race 6 of *Psp*. The genomic sequence data of the gene (from Phytozome v10) with inadequate functional annotation data was localized using marker's sequences available on NCBI (http://www.ncbi.nlm.nih.gov/) databases using BLASTn (basic local alignment search tool) (Zhang et al., 2010). The criteria to consider homologous sequences (genes) between query and database sequences included E-value <1 x 10^{-40} and a minimum identity of 70%.

Results and Discussion

Phenotypic Analysis

Of the 383 accessions used for trifoliate evaluation, 197 accessions were used for GWAS analysis as halo blight severity data from these accessions were successfully collected from all four replications. Highly significant ($P < 0.0001$) differences existed among the 197 accessions of common bean for halo blight severity with a mean disease score of 2.0 and range of 1-4, respectively (Appendix III). At ten days post inoculation, inoculated plants displayed a wide range of foliar symptoms (Figure 11). Based on the mean halo blight score, of total 197 accessions, 17 (9%), 105 (53%), 37 (19%), 36 (18%) and 2 (1%) accessions were categorized as highly resistant, resistant, slightly susceptible and susceptible, respectively (Figure 12). Most accessions from highly resistant category were from Mexico (41%), followed by Colombia (12%), Guatemala (12%), Peru (12%), Ecuador (12%), and Costa Rica (6%), respectively. Among 17 highly resistant accessions from current study, 8 accessions, i.e., PI 201343, PI

201388, PI 313217, PI 313237, PI 313490, PI 313572, PI 417657, and PI 449410, also displayed resistant reactions to trifoliate inoculation in previous experiment (see chapter I). The high incidence of accessions resistant to trifoliate inoculation to halo blight in the current study is from Mexico, an example of predominance of greater genetic variability in the Middle American gene pool than in Andean (Bitocchi et al., 2012). Similar results were obtained from previous unifoliate and trifoliate experiments (Chapter I). However, the proportion of Mesoamerican versus Andean accessions in the set used for this experiment may have greatly influenced the findings as most accessions evaluated represents Mesoamerican gene pool, (167 Mesoamerican vs. 30 Andean). The susceptible check, Pink Panther, had a score of 3.0, whereas the resistant checks, US14HBR6 and Montcalm, had a score of 1.2 and 2.0, respectively.

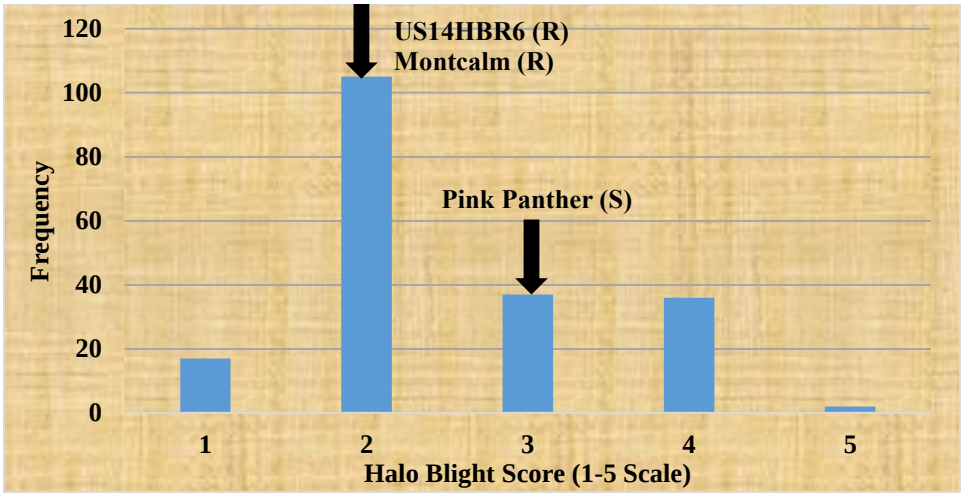

Figure 12. Frequency distribution of 197 common bean accessions based on the halo blight severity evaluated across four replications under greenhouse condition. Halo blight disease score in resistant (R) (US14HBR6 and Montcalm) and susceptible (S) (Pink Panther) checks are indicated in arrows.

Population Structure

Since in association studies, population structure lead to spurious association between markers and trait due to difference in allele frequencies and genetic relatedness in mapping

population, PCA can be used in minimizing number of false positives and maximize the power to detect true associations (Price et al., 2006; Zhao et al., 2007). Analysis of population structure with PCA revealed that first, second, and third PCs accounted for 39.9%, 10.7%, and 2.8% of the genotypic variation, respectively. These first three PCs that explained 53.4% of the genotypic variation in the core collection were used as covariates in the analysis. The principal component plot of PC1 against PC2 (Figure 14) clearly identified two distinct clusters corresponding to the Mesoamerican and the Andean gene pools. Most of the accessions, i.e., 141 accessions, clustered within the Mesoamerican genepool, while 47 accessions clustered within the Andean genepool. The first PC, i.e., PC1, also separated the small cluster of accessions from the Mesoamerican and Andean groups that comprised of 9 accessions as admixture, of which 6 accessions were from Mexico, 2 from Guatemala, and 2 from Peru. Figure 13 shows the scree plot generated through GAPIT recommending the first three components as informative.

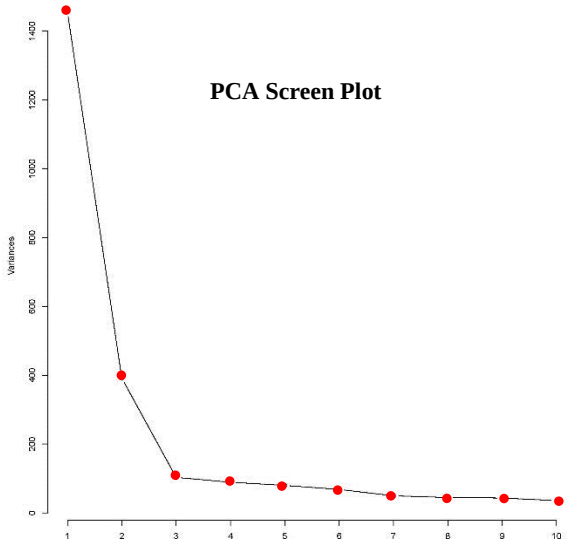

Figure 13. Screen plot from GAPIT showing the selection of PCs for GWAS study.

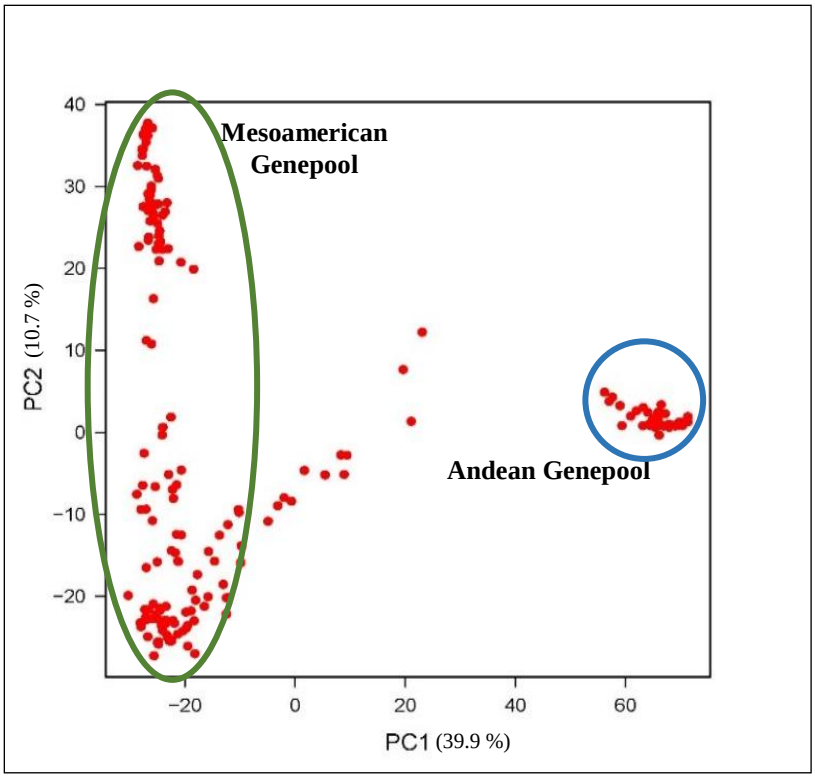

Figure 14. Principal component analysis (PCA) plot of PC1 versus PC2 of 197 core collection of common bean determined using 4707 SNP markers.

Marker-trait Association

In the current study, to minimize the confounding effect due to population structure and family relatedness to get non-spurious association between markers and trait, six different statistical models, i.e., Naïve, EMMA, Mixed Model (MM/EMMA + PCA1), PCA3, PCA10, and Mixed Model 3 (MM3/EMMA + PCA3) were employed (Table 13). Out of tested models, both approaches, i.e., QQ-plot (Figure 15) and MSD values, indicated MM3 as best model with lowest MSD value of 0.00002, followed by PCA10, PCA3, EMMA, MM, and, Naïve with MSD values of 0.0002, 0.0003, 0.0003, 0.0003, and 0.06, respectively suggesting that control for both population structure and familial relatedness reduced the likelihood of identifying false positives for the trait. However, the discussion on significant markers and its association with trait of interest are based on the results of the analysis using the best model selected, i.e., MM3.

Manhattan plot from best model representing the chromosomal position of significant markers is presented in Figure 16, where different chromosomes are represented by different colors.

The Manhattan plot indicated that a total of 3 loci reach the genome-wide significance threshold of $p < 1.1 \times 10^{-5}$ (Table 14 and Figure 16). The number of significant loci varied from chromosome to chromosome, where 2 significant loci, i.e., sc00112ln569344_270381_C_T_97989381, and sc00835ln140787_67166_T_C_289648473 was observed on chromosome Pv04, and one locus, sc00004ln1947458_1678611_C_T_8133337, on chromosome Pv05. These three significant loci explained total of 19% phenotypic variation, where the most significant locus ($p = 1.8 \times 10^{-4}$), sc00112ln569344 on Pv 04, alone accounted for about 7% of the phenotypic variation (Table 14).

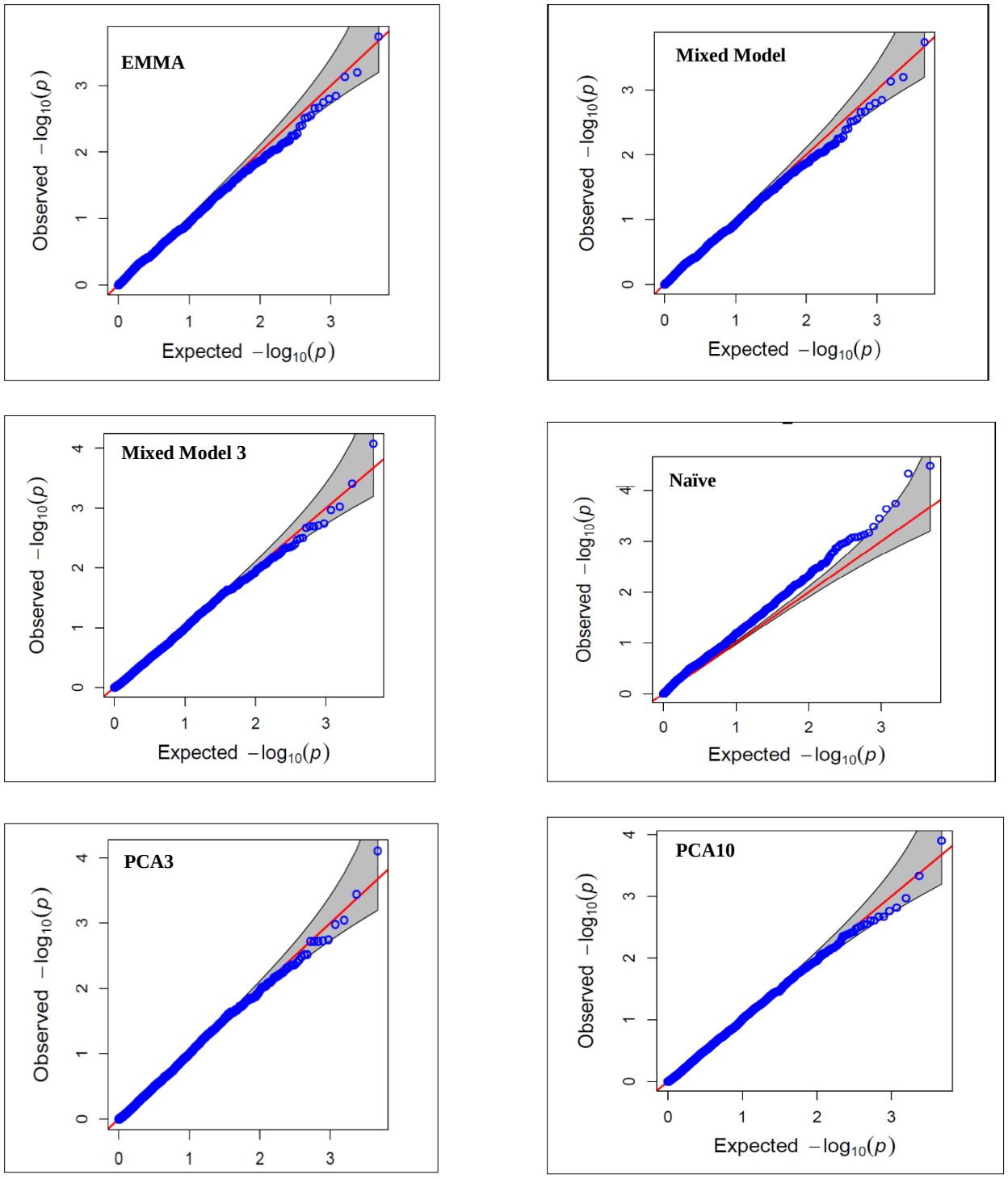

Figure 15. Graphical representation of QQ-plot showing the distribution of *p*-values for six different models tested.

Table 14. Chromosome, position, *P*-values, proportion of phenotypic variation explained (R^2), and minor allele frequency of three significant SNPs controlling for resistance to race 6 of *Psp* measured on 197 accessions of common bean from USDA-NPGS core collection.

SNP[§]	**Chromosome**	**SNP Position (Mbp)**	***P*-value**[‡]	$R^{2\dagger}$	**Favorable allele frequency**
sc00835ln140787_67166_T_C_	Pv04	547510	7.3×10^{-4}	0.06	0.06
sc00112ln569344_270381_C_T_	Pv04	1248990	1.8×10^{-4}	0.07	0.05
sc00004ln1947458_1678611_C_T_	Pv05	39442503	6.3×10^{-4}	0.06	0.47

[§] SNP, single nucleotide polymorphic code in million base pair
‡ *P*, Significance level
† R^2, phenotypic variation explained by respective SNP

Favored allelic frequencies of all three significant markers were estimated in different origins of bean to gain insight into the allelic diversity present in each of those markers located on Pv04/5.4 Mbp, Pv04/12.4 Mbp, and Pv05/39.4 Mbp. The allelic distribution for SNP sc00004ln1947458_1678611_C_T_ on Pv05 was more diverse compared to allelic frequency for remaining two SNPs in Pv04. The frequency of favorable allele 'T' for significant SNP sc00004ln1947458_1678611_C_T_ on Pv05 was 0.47 (Table 14). Therefore, the breeders may have to increase the frequency of this allele to incorporate their significance in the breeding program aimed for resistance to race 6 of *Psp*.

The geographic distribution of accessions that carried favorable alleles contributing to lesser halo blight disease score are presented in Table 15. Twenty-three accessions with favorable allele 'T' were landraces from Andean origin, where four accessions were from Bolivia, 8 from Ecuador, and 11 from Peru. The results from the current study agrees with the findings from Porch et al., (2016) and Vasquez et al., (2016) where the significant markers to race 6 of *Psp* was also reported on Pv05 using Andean Diversity Panel (ADP). Therefore, the selection and introduction of genotypes from Andean origin with this favorable allele could bring

new genetic variability for breeding resistance to race 6 of *Psp* and could be of interest to breeders. In contrast, two other significant SNPs, sc00112ln569344_270381_C_T_ and sc00835ln140787_67166_T_C_, on Pv04 carrying favorable allele 'T' contributing to disease resistance were fixed across the accessions from both Andean and Mesoamerican origin (Table 15). Therefore, the allelic effects for both SNPs on Pv04 were also informative; however, they may not be useful since most of the accessions already have the favorable allele fixed across both gene pools. This could also be reflected in non-significant mean halo blight disease score of accessions from Andean origin (1.5) in contrast to Mesoamerican origin (2.0) (data not shown). This could be explained due to the lower MAF of 0.05 and 0.06 of favorable allele 'T' for both SNP sc00112ln569344_270381_C_T_ and sc00835ln140787_67166_T_C_ on Pv04, respectively (Table 14). This even distribution of favorable allele in Pv04 across both origins could be due to a highly skewed population used in the study with majority of accessions from Mesoamerican (85%) genepool in contrast to Andean (15%) (Table 15).

The mean halo blight disease score contributed by the alleles in each of three significant SNPs are presented in table 16. Data shows the higher effect of favorable allele 'T' in Pv05 with lesser disease score, thus contributing to disease resistance to race 6 of *Psp*. However, there were no significant differences between the mean halo blight disease score of alleles of two SNPs in Pv04 categorizing it as non-informative markers unlike SNP in Pv05.

Table 15. Geographic distribution of the favorable allele of three significant SNPs measured on 197 accessions from USDA-NPGS common bean core collection.

Country‡	Favorable Allele Percentage (%) for each significant SNP		
	Pv05 (sc00004ln19477458 _1678611_C_T) †	**Pv04 (sc00112ln569344_ 270381_C_T)** †	**Pv04 (sc00835ln140787_67166 _T_C)** †
Andean			
Bolivia	100	100	100
Ecuador	80	80	80
Peru	68	87	87
Mean§	82	89	89
Mesoamerican			
Costa Rica	25	87	75
Colombia	30	94	88
El Salvador	30	100	100
Guatemala	23	100	100
Honduras	0	100	100
Nicaragua	20	100	100
Mexico	24	95	95
Mean§	22	96	94

‡ Respective countries are categorized per two gene pools in common beans, i.e., Andean and Middle American (Mesoamerican).
† T is the favorable allele with a frequency of 0.47, 0.05, and 0.06 for SNPs sc00004ln19477458_1678611_C_T (Pv05), sc00112ln569344_270381_C_T (Pv04) and sc00835ln140787_67166_T_C (Pv04), respectively.
§ Average percentage of genotypes with favorable allele of each three significant SNPs.

Table 16. Alleles of three significant SNPs with mean and standard deviation of halo blight scores measured on 197 accessions from USDA-NPGS common bean core collection.

Significant markers	**Chromosome**	**Alleles**†	**Number of genotypes (N)**	**Mean ± S.D.**§
sc00004ln19477458_1678611_C_T	Pv05	C	105	2.3 ± 0.9a
		T	92	1.9 ± 0.8b
sc00112ln569344_270381_C_T	Pv04	C	11	2.4 ± 0.9a
		T	186	2.1 ± 0.8a
sc00835ln140787_67166_T_C	Pv04	C	13	2.3 ± 0.9a
		T	184	2.1 ± 0.8a

† 'T' and 'C' are favorable and unfavorable alleles on each respective marker with lesser and larger effects on halo blight disease score, respectively.
§ Values bearing the same letter are not significantly different (LSD, $\alpha = 0.05$).

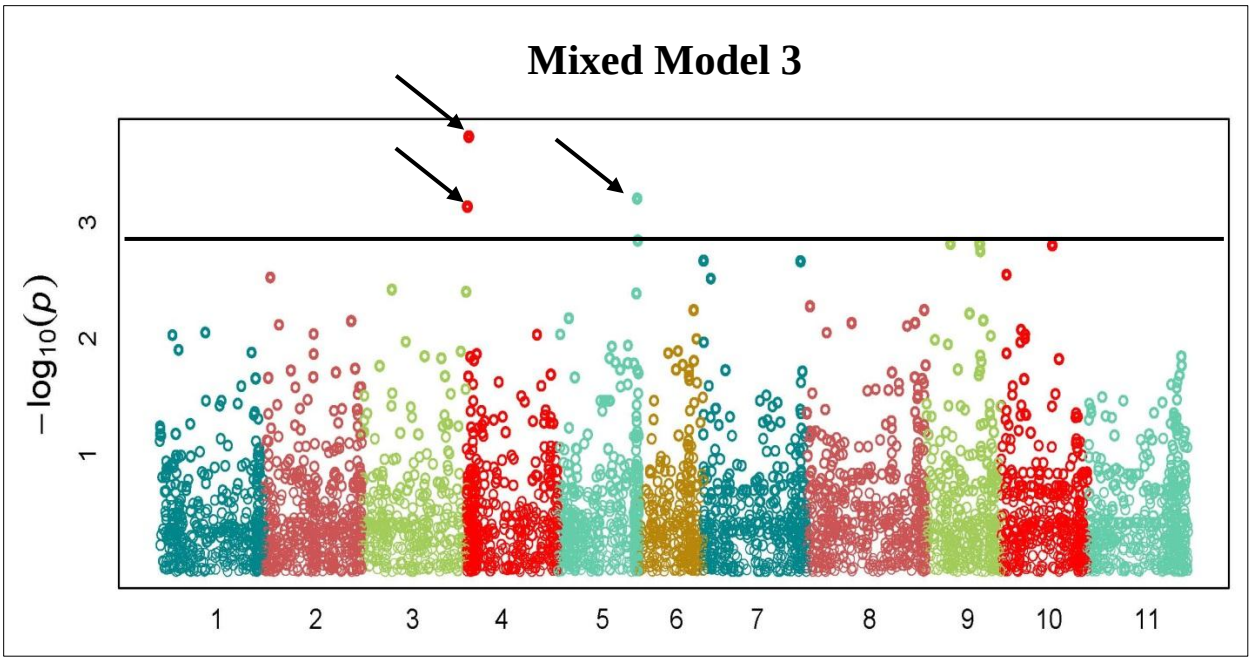

Figure 16. Manhattan plots generated in Mixed Model 3 (EMMA + PCA3) showing the SNP markers associated with resistance to race 6 of *Psp*. Each different color represents eleven different bean chromosomes. Horizontal black line shows the significance threshold of $P = 1.1 \times 10^{-5}$ after Bonferonni correction of $\alpha = 0.05$. Black arrows point out the most significant markers after Bonferroni correction tests.

Using the functional annotation on Phytozome (http://www.phytozome.net), the potential candidate genes co-localizing with significant SNPs was examined to understand the causes of variation in resistance to race 6 of *Psp*. The functional annotation indicated two candidate genes, *Phvul.004G007600* and *Phvul.004G007700*, about 58 Kbp and 75 Kbp, respectively downstream of significant SNP marker sc00835ln140787_67166_T_C_ in chromosome P04. Similarly, two candidate genes, *Phvul.004G012600* and *Phvul.004G012800*, were found co-localized about 26 Kbp and 67 Kbp, respectively downstream of another significant SNP marker SNP sc00112ln569344_270381_C_T_ in Pv04. While, in chromosome Pv05, three different candidate genes, *Phvul.005G170900*, *Phvul.005G16990*, and *Phvul.005G169600*, were found approximately at 11 Kbp downstream, 58 Kbp upstream, and 69 Kbp, respectively downstream of significant SNP marker SNP sc00004ln1947458_1678611_C_T_.

The candidate gene *Phvul.004G012800* located approximately 67 Kbp downstream of SNP sc00112ln569344_270381_C_T_ on Pv04 codes for a NB-ARC (Nucleotide-binding ARC) domain containing disease resistance protein that determine the regulatory function under stress conditions. Similarly, the other significant marker on Pv04, sc00835ln140787_67166_T_C_, was co-localized approximately 75 Kbp downstream of candidate gene *Phvul.004G007700*, which also codes for a NB-ARC domain containing disease resistance protein. Nucleotide-binding ARC domain containing disease resistance protein is among one of the five classes of proteins encoded by disease resistance (*R*) genes (Dangl and Jones, 2001). Plant resistance to several bacterial, fungal and viral pathogens is governed by specific interactions between plant disease resistance (*R*) genes, also known as R proteins, and corresponding pathogen avirulence (*Avr*) gene following gene-for-gene interactions (Flor, 1971). Upon recognition of specific *avr*-dependent signals in pathogen, *R* gene in the host triggers the activation of plant defense mechanism to restrict pathogen proliferation via programmed cell death known as hypersensitive response (HR) (Ooijen et al., 2008). Most of the *R* genes encode a central nucleotide-binding (NB) domain and a leucine-rich repeat (NB-LRR) domain that is associated with protein-protein interactions, and activates downstream signal pathways leading to disease resistance responses (Dangl and Jones, 2001; Kobe and Deisenhofer, 1994). A homology region is shared between NB and LRR domains and is known as ARC (Apaf-1, R-protein, and Ced-4 genes) domain, which is consists of three subdomains, i.e., NB, ARC1, and ARC2 (Riedl et al., 2005). Rairdan and Moffett (2006) showed the importance of ARC1 and ARC2 domain for binding of Rx protein to LRR domain, which conferred resistance to Potato Virus X (PVX) in potato (*Solanum tuberosum* L.). Similarly, Rx2 and Gpa2 protein in potato conferring resistance to PVX virus and nematode *Globodera pallida*, respectively, and Bs2 protein in pepper (*Capsicum annuum* L.) conferring

resistance to bacterium *Xanthomonas campestris* pv. *vesicatoria* were consisted of CC-NB-ARC plus LRR or CC plus NB-ARC-LRR protein (Bendahmane et al., 2000; Moffett et al., 2002; Leister et al., 2005; van der Vossen et al., 2000). Genes *RPP1* and *RPP5* are the members of one of the largest *R* genes class in plants, that encode NB sites and LRR domains (NB-LRR proteins), where the central region of NB-LRR proteins is shared with Apaf-1 protein of NB-ARC domain (Staskawicz et al., 1995; Hammond-Kosack and Jones, 1997; Van der Biezen and Jones, 1998). Parker et al. (1997) reported the role of *RPP5* gene on chromosome 4 of *Arabidopsis* conferring resistance to strain Noco2 of the pathogen *Peronospora parasitica* (Pers.) Tul. In contrast, Botella et al. (1998) showed the role of *RPP1* gene controlling resistance to same strain on *P. parasitica* in chromosome 3 of *Arabidopsis* ecotype Wassilewskija. Similarly, the role of *RPP5* gene in *Arabidopsis* was emphasized in terms of recognizing the novel pathogen variants through gene duplication, diversification, and subsequent selection (Noël et al., 1999). In common bean, the investigation of disease resistance (*R*) gene cluster situated at the subtelomeric region of the short arm of chromosome Pv04 was found to encode 29 B4-CC nucleotides-binding-site–leucine-rich-repeat (B4-CNL) genes, and provided resistance to bean anthracnose, caused by the fungus *Colletotrichum lindemuthianum* (Sacc. & Magnus) Briosi & Cavara (Geffroy et al., 2009). Of 24 B4-CNL genes, 12 were found in genotype, JaloEEP558, from Andean origin, while remaining 17 were found in genotype, BAT93, of Mesoamerican origin.

The association on chromosome Pv04 contained the candidate gene *Phvul.004G012600* that encode protein kinase superfamily protein, and was located at about 26 Kbp downstream from SNP marker sc00112ln569344_270381_C_T_. Tomato (*Solanum lycopersicum* L.) *Pto* gene, a plant protein kinase, has been one of the first reported plant gene to be cloned, and

mediates resistance to bacterial speck disease caused by *Pseudomonas syringae* pv. *tomato* exhibiting gene-for-gene interaction (Martin et al., 1993). This gene encodes for serine/threonine protein kinases, but without LRRs, that plays a vital role in a signal transduction pathway, a biochemical phenomenon that involves in change in cell enzymatic activity, and gene expression in response to environment. In tomato, the effective activation of host disease resistance because of interaction of *Pto* and *AvrPto* protein has been widely studied (Abramovitch et al., 2003; Kim et al., 2002; Martin et al., 1993; Ronald et al., 1992).

Approximately 58 Kbp downstream of significant marker, sc00835ln140787_67166_T_C_, in chromosome Pv04 is located other candidate gene *Phvul.004G007600* that code for zinc finger (CCCH-type) family protein associated with plant growth, development and stress response. CCCH-zinc finger proteins contain a typical motif consisted of three cysteines and one histidine residue. A cDNA clone known as *Gossypium hirsutum* zinc finger protein 1 (*GhZFP1*) isolated from *G. hirsutum* was studied in transgenic tobacco (*Nicotiana tabacum* L.) cultivar NC89, for its central role in stress signaling (Guo et al., 2009). The study concluded the interaction of *GhZFP1* protein with GZIRD21A and GZIPR5 enhanced drought, and salt tolerance and resistance to the fungus *Rhizoctonia solani* Kühn in transgenic tobacco. The role of *C3H12,* CCH-type zinc finger family protein, in controlling bacterial blight in rice (*Oryzae sativa* L.) caused by *Xanthomonas oryzae* pv *oryzae* (*Xoo*) was emphasized in the study conducted by Deng et al., (2012). *C3H12* enhanced the resistance by triggering the jasmonic acid (JA) accumulation and inducing the expression of JA signaling genes in rice. Among several zinc finger family proteins, NFX1-type expresses MHC II gene, and is highly associated with growth and development of many crops via regulation of salicylic

acid, reactive oxygen species, and abscisic acid under biotic and abiotic stress conditions (Ciftci-Yilmaz et al., 2007).

Likewise, significant marker, sc00004ln1947458_1678611_C_T_ on Pv05, was approximately 69 Kbp upstream of gene *Phvul.005G169600* that code for IAA8 (Indole Acetic Acid protein 8) protein. A BLASTn search against of *Phvul.005G169600* genomic sequence in NCBI (National Center for Biotechnology Information) data resulted in a best hit to a gene GLYMA_13G356600 in soybean that code for auxin-responsive protein IAA. Indole Acetic Acid is a plant hormone that regulates the plant growth and development, and can induce the expression of three phylogenetic categories of auxin-induced genes, *Aux/IAA* family, *GH3* family and small auxin-up RNA (*SAUR*) family (Woodward and Bartel, 2005). Of these genes, overexpression of *GH3* gene have been reported to play an important role in plant defense responses against bacterial blight disease in rice caused by *Xoo* (Ding et al., 2008). In *Arabidopsis thaliana*, when plant is under pathogen attack, salicylic acid (SA) is found to be in higher concentration that help trigger the activity of NPR1 protein, which ultimately trigger the defense response genes such as pathogenesis-related (PR) genes (An and Mou, 2011; Wang et al., 2007).

Similarly, at 58 Kbp upstream of the SNP sc00004ln1947458_1678611_C_T_ on Pv05 lies gene *Phvul.005G16990* that codes for transducin family protein/ WD-40 repeat protein. Predominant in eukaryotes, but rarely present in prokaryotes, WD40 repeat proteins are involved in many biological functions including cell division and cytokinesis, apoptosis, light signaling and vision, cell motility, flowering, floral development, meristem organization, protein trafficking, and transcriptional mechanism (Stirnimann et al., 2010). Gachomo et al. (2014), emphasized the importance of GIGANTUS1 (GTS1), a new member of WD-40 protein in *A.*

thaliana, for regulating the plant growth development such as seed germination, faster growth, flowering time, and biomass accumulation. In *Arabidopsis*, WD-40 protein domain such as Transparent Testa2 (TT2), a Myb transcription factor, TT8, and Transparent Testa Glabrous1 (TTG1) help regulate the flavonoid biosynthesis, a plant secondary metabolite controlling flower pigmentation and flower color (Nesi et al., 2001; Baudry et al., 2004). Galeotti et al. (2008) reported the antifungal activity of flavonoid glycoside analogues via mycelial growth against different *Fusarium oxysporum* f. sp. *dianthi* pathotypes in carnation, *Dianthus caryophyllus*. Expression of several WD-40 protein such as HOS15, TaWD-40 in wheat and *Arabidopsis*, have been associated with several abiotic stresses such as cold sensitivity, enhanced tolerance to ABA, salt and osmotic stress in plants (Kong et al., 2015; Zhu et al., 2008). CYCLOPHILIN71 (CYP71), a WD-40 repeat protein, in *Arabidopsis* have been reported in meristem development, reduced lateral organ development, and reduction in root elongation (Li et al., 2007).

At 11 Kbp downstream of significant SNP sc00004ln1947458_1678611_C_T_ on Pv05 lies the gene *Phvul.005G170900* that encodes Ras-related small GTP-binding family protein. Programmed cell death is one of a major characteristic feature of plant self-defense mechanisms during resistance reaction to pathogens that is possible due to rapid production of reactive oxygen species (ROS) (Vaux and Korsmeyer, 1999). The rapid production of ROS because of infection by avirulent strains of pathogens triggers hypersensitive response (HR) thus killing pathogens in the infected cells (Lamb and Dixon, 1997; Neill et al., 2002). Rac/ROP small GTPases are plant-specific members that participate as key regulators of diverse processes including the production of ROS as well as pollen tube growth, root hair development and hormone responses in plants (Chen et al., 2010). In rice, a key role of *OsRac1* in the production of ROS and cell death causing HR responses against a virulent race of rice blast fungus

(*Magnaporthe grisae* Hebert and Barr, race 007), and bacterial blight (*Xanthomonas oryzae* Swings pv. *oryzae*, race 1) has been emphasized by Kawasaki et al., (1999) and Ono et al., (2001).

Chromosome Pv04 harbors the largest known cluster of *R* genes in bean governing resistance to several bacterial, and fungal diseases (David et al., 2009; Geffroy et al., 1999; Keller et al., 2015; Meziadi et al., 2015; Perseguini et al., 2016; Perez-Vega et al., 2013). Additionally, the recent common bean genome sequence also reported the presence of majority of clusters of putative disease resistance gene encoding NBS-LRR at the end of chromosome Pv04 including Pv10, and PV11 (Schmutz et al., 2014). In common bean, a major QTL on chromosome Pv04 for resistance to different races of *Psp* has been reported repeatedly across different bi-parental mapping populations (Ariyaranthe et al., 1999; Fourie et al., 2004; Miklas et al., 2014; Trabanco et a., 2014). Ariyaranthe et al. (1999) mapped the first known QTL resistant to two different strains of *Psp*, i.e., HB 16 and HB 83-Sc2A (classified as race 7) using RI lines derived from a bi-parental population of Great Northern Belneb RR-1 x A-55. The study also reported the four QTLs on Pv02, 04, 05 and 09 significantly associated with resistance to race 7 of *Psp* that collectively accounted for 32% of total phenotypic variation. However, in this study the author(s) found the gene controlling for hypersensitive resistance (HR) to the race in same QTL region of Pv04 at an interval of 21.6 cM from RAPD marker B10.520. Later Fourie et al., (2004) using same bi-parental population, reported the three major resistance gene, i.e., *Pse-1* on Pv04 conditioning resistance to race 1, 5, 7, and 9, *Pse-3* on Pv02 conditioning resistance to race 3 and 4, and *Pse-4* on Pv04 conditioning resistance to race 5. Likewise, towards the end of Pv04 a tight cluster of new gene known as *Pse-6* was reported at about 1.4 cM from SCAR marker, SB10.550, which governed resistance to race 1, 5, 7, and 9 of *Psp* (Miklas et al., 2014). Similar

bi-parental population used by Fourie et al., (2004) was used for mapping genes in this experiment. A recent study by Trabanco et al. (2014), identified two QTLs in Pv04 and Pv06, where QTL on Pv04 was flanked by two markers Pvag004 and BMd15 at about 44 Mbp. Similarly, the study also reported two additional QTLs on Pv06 that controlled for resistance to race 7 of *Psp* (Trabanco et al., 2014). Contrastingly, when the relative position of both significant SNPs in Pv04, sc00835ln140787_67166_T_C_ mapped at 547510 Mbp, and sc00112ln569344_270381_C_T_ at 1248990 Mbp, reported from this study was compared with the QTL position in Pv04 reported by Trabanco et al. (2014), i.e., 44 Mbp, they appear to be in different position. This finding suggests the identification of two potentially novel genomic regions in Pv04 (547510 and 1248990 Mbp; Table 14) that is associated with resistance to race 6 of *Psp*. Therefore, future work is required to validate the resistance loci with novel resistance to develop user friendly, tightly linked markers for incorporation into common bean breeding programs. Nonetheless, in the current study, across all the models tested for association analysis including naïve showed the Manhattan plot with consistency of large peaks in Pv04 suggesting its strong correlation with resistance to race 6 of *Psp* (Appendix IV).

Reports on the genomic regions underlying halo blight disease resistance mapped in linkage group Pv05 is limited. Ariyaranthe et al. (1999) reported a QTL governing resistance to halo blight disease in Pv05 that alone accounted for 20% phenotypic variation. Recently, a cluster of significant SNPs involved in the resistance to race 6 of *Psp* were identified in the Andean Diversity Panel in approximately same relative positions as reported in current study (Porch et al., 2016; Vasquez et al., 2016). Porch et al. (2016) found four significant SNPs controlling for about 33% phenotypic variation at approximately 38 Mbp, as well as Vasquez et al. (2016) also found three significant markers in exactly similar position. The findings from this

current study confirms the relative position of SNPs in Pv05 (39442503 Mbp; Table 14) governing resistance to race 6 of *Psp* to be consistent with the position identified in previous studies.

In summary, to the best of our knowledge, this study is the first attempt to diagnose the common bean genomic regions conferring resistance to race 6 of *Psp* using the USDA-NPGS core collection of common bean. Results from the present study provide an insight into the genetic architecture of QTLs identified in two genomic regions and their potential for developing resistance to race 6 of *Psp* in bean breeding programs. However, the small phenotypic variation explained by the QTL identified in the current study can be validated in different genetic background and environments that may prove beneficial to marker-assisted selection (MAS). The physical position and the candidate genes identified in the current study could be of clear utility to genotype-by-sequencing (GBS) for genomics-assisted common bean improvement.

References

Abdurakhmonov, I.Y., and A. Abdukarimov. 2008. Application of association mapping to understanding the genetic diversity of plant germplasm resources. Int. J. Plant Genomics. 1-18. doi:10.1155/2008/574927

Abramovitch, R.B., Y.J. Kim, S. Chen, M.B. Dickman, and G.B. Martin. 2003. *Pseudomonas* type III effector *AvrPtoB* induces plant disease susceptibility by inhibition of host programmed cell death. EMBO. J. 22:60-69.

An, C., and Z. Mou. 2011. Salicylic acid and its function in plant immunity. J. Integr. Plant Biol. 53:412-428.

Ariyarathne, H.M., D.P. Coyne, G. Jung, P.W. Skroch, A.K. Vidaver, J.R. Steadman, P.N. Miklas, and M.J. Bassett. 1999. Molecular mapping of disease resistance genes for halo blight, common bacterial blight, and bean common mosaic virus in a segregating population of common bean. J. Am. Soc. Hort. Sci. 124:654-662.

Baudry, A., M.A. Heim, B. Dubreucq, M. Caboche, B. Weisshaar, and L. Lepiniec. 2004. TT2, TT8, and TTG1 synergistically specify the expression of BANYULS and proanthocyanidin biosynthesis in *Arabidopsis thaliana*. Plant J. 39:366–380.

Beebe, S., I.M. Rao, C. Mukankusi, and R. Buruchara. 2012. Improving resource use efficiency and reducing risk of common bean production in Africa, Latin America and the Caribbean. Eco-efficiency: From vision to reality. CIAT, Cali, Colombia. p. 1-18.

Beebe, S., P.W. Skroch, J. Tohme, M.C. Duque, F. Pedraza, and J. Nienhuis. 2000. Structure of genetic diversity among common bean landraces of Middle American origin based on correspondence analysis of RAPD. Crop Sci. 40:264-273.

Bendahmane, A., M. Querci, K. Kanyuka, and D.C. Baulcombe. 2000. Agrobacterium transient expression system as a tool for the isolation of disease resistance genes: Application to the Rx2 locus in potato. Plant J. 21:73-81.

Bitocchi, E., L. Nanni, E. Bellucci, M. Rossi, A. Giardini, P.S. Zeuli, and R. Papa. 2012. Mesoamerican origin of the common bean (*Phaseolus vulgaris* L.) is revealed by sequence data. PNAS 109:E788-E796.

Blair, M.W., L.M. Díaz, H.F. Buendía, and M.C. Duque. 2009. Genetic diversity, seed size associations and population structure of a core collection of common beans (*Phaseolus vulgaris* L.). Theor. Appl. Genet. 119:955-972.

Blair, M.W., L.F. Gonzalez, P.M. Kimani, and L. Butare. 2010. Genetic diversity, inter-gene pool introgression and nutritional quality of common beans (*Phaseolus vulgaris* L.) from central Africa. Theor. Appl. Genet. 121:237-248.

Botella, M.A., J.E. Parker, L.N. Frost, P.D. Bittner-Eddy, J.L. Beynon, M.J. Daniels, E.B. Holub, and J.D.G. Jones. 1998. Three genes of the *Arabidopsis RPP1* complex resistance locus recognize distinct *Peronospora parasitica* avirulence determinants. Plant Cell 10:1847-1860.

Brick, M.A., P.F. Byrne, H.F. Schwartz, J.B. Off, K. Otto, A.L. Fall, and J. Gilbert. 2006. Reaction to three races of Fusarium wilt in the *Phaseolus vulgaris* core collection. Crop Sci. 46:1245-1252.

Broughton, W.J., G. Hernandez, M. Blair, S. Beebe, P. Gepts, and J. Vanderleyden. 2003. Beans (*Phaseolus* spp.)--model food legumes. Plant and Soil. 252:55-128.

Chen, L., K. Shiotani, T. Togashi, D. Miki, M. Aoyama, H.L. Wong, T. Kawasaki, and K. Shimamoto. 2010. Analysis of the Rac/Rop small GTPase family in rice: Expression, subcellular localization and role in disease resistance. Plant Cell Physiol. 51:585-595.

Ciftci-Yilmaz, S., M.R. Morsy, L. Song, A. Coutu, B.A. Krizek, M.W. Lewis, D. Warren, J. Cushman, E.L. Connolly, and R. Mittler. 2007. The EAR-motif of the Cys2/His2-type zinc finger protein Zat7 plays a key role in the defense response of Arabidopsis to salinity stress. J. Biol. Chem. 282:9260-9268.

Dangl, J.L., and J.D. Jones. 2001. Plant pathogens and integrated defense responses to infection. Nature 411:826-833.

David, P., N.W. Chen, A. Pedrosa-Harand, V. Thareau, M. Sévignac, S.B. Cannon, D. Debouck, T. Langin, and V. Geffroy. 2009. A nomadic subtelomeric disease resistance gene cluster in common bean. Plant Physiol. 151:1048-1065.

Deng, H., H. Liu, X. Li, J. Xiao, and S. Wang. 2012. A CCCH-type zinc finger nucleic acid-binding protein quantitatively confers resistance against rice bacterial blight disease. Plant Physiol. 158:876-889.

Ding, X., Y. Cao, L. Huang, J. Zhao, C. Xu, X. Li, and S. Wang. 2008. Activation of the indole-3-acetic acid-amido synthetase GH3-8 suppresses expansin expression and promotes salicylate- and jasmonate-independent basal immunity in rice. Plant Cell 20:228-240.

Duncan, R.W., M. Lema, R.L. Gilbertson, and S.P. Singh. 2014. Registration of common bean pinto US14HBR6 resistant to race 6 of the halo blight pathogen, *Pseudomonas syringae* pv. *phaseolicola*. J. Plant Reg. 8:53-56.

Duncan, R.W., S.P. Singh, and R.L. Gilbertson. 2008. Identification and inheritance of a new source of halo blight resistance in common bean. Annu. Rep. Bean. Improv. Coop. 51:48-49.

Duncan, R.W., S.P Singh, and R.L. Gilbertson. 2011. Interaction of common bacterial blight bacteria with disease resistance quantitative trait loci in common bean. Phytopathology 101:425-435.

Flor, H.H. 1971. Current status of the gene-for-gene concept. Annu. Rev. Phytopathol. 9:275-296.

Fourie, D., P. Miklas, and H. Ariyaranthe. 2004. Genes conditioning halo blight resistance to races 1, 7, and 9 occur in a tight cluster. Annu. Rep. Bean Improv. Coop. 47:103-104.

Gachomo, E.W., J.C. Jimenez-Lopez, L.J. Baptiste, and S.O. Kotchoni. 2014. GIGANTUS1 (GTS1), a member of Transducin/WD40 protein superfamily, controls seed germination, growth and biomass accumulation through ribosome-biogenesis protein interactions in *Arabidopsis thaliana*. BMC Plant Biol. 14:1-17.

Galeotti, F., E. Barile, P. Curir, M. Dolci, and V. Lanzotti. 2008. Flavonoids from carnation (*Dianthus caryophyllus*) and their antifungal activity. Phytochemistry Letters 1:44-48.

Geffroy, V., C. Macadré, P. David, A. Pedrosa-Harand, M. Sévignac, C. Dauga, and T. Langin. 2009. Molecular analysis of a large subtelomeric nucleotide-binding-site–leucine-rich-repeat family in two representative genotypes of the major gene pools of *Phaseolus vulgaris*. Genetics 181:405-419.

Geffroy, V., D. Sicard, J.C. de Oliveira, M. Sévignac, S. Cohen, P. Gepts, C. Neema, T. Langin, and M. Dron. 1999. Identification of an ancestral resistance gene cluster involved in the coevolution process between *Phaseolus vulgaris* and its fungal pathogen *Colletotrichum lindemuthianum*. Mol. Plant-Microbe Interact. 12:774-784.

Goodstein, D.M., S. Shu, R. Howson, R. Neupane, R.D. Hayes, J. Fazo, T. Mitros, W. Dirks, U. Hellsten, N. Putnam, and D.S. Rokhsar. 2012. Phytozome: A comparative platform for green plant genomics. Nucleic Acids Res. 40: D1178-D1186.

Guo, Y.H., Y.P. Yu, D. Wang, C.A. Wu, G.D. Yang, J.G. Huang, and C.C. Zheng. 2009. GhZFP1, a novel CCCH-type zinc finger protein from cotton, enhances salt stress tolerance and fungal disease resistance in transgenic tobacco by interacting with GZIRD21A and GZIPR5. New Phytol. 183:62-75.

Hagedorn, D.J., and D.A. Inglis. 1986. Handbook of bean diseases. Madison, WI. UW Extension Publication.

Hammond-Kosack, K.E., and J.D.G. Jones. 1997. Plant disease resistance genes. Annu. Rev. Plant Physiol. Plant Mol. Biol. 48:575-607.

Hergert, M. 2010. Halo blight reduces Wyoming seed production. Northarvest Beangrower Mag. 16:31-32.

Hyten, D.L., Q. Song, E.W. Fickus, C.V. Quigley, J.S. Lim, I.Y. Choi, E.Y. Hwang, M. Pastor-Corrales, and P.B. Cregan. 2010. High-throughput SNP discovery and assay development in common bean. BMC Genomics 11:475-483.

Innes, N.L., J. Conway, and J.D. Taylor. 1984. Resistance to halo-blight in the Cambridge accessions V4604 and V4058 of *Phaseolus* beans. Ann. Appl. Biol. 104:307-314.

Jannink, J.L., M.C.A.M. Bink, and R.C. Jansen. 2001. Using complex plant pedigrees to map valuable genes. Trends Plant Sci. 6:337-342.

Kamfwa, K., K.A. Cichy, and J.D. Kelly. 2015. Genome-wide association study of agronomic traits in common bean. The Plant Genome 8:1-12.

Kang, H.M., N.A. Zaitlen, C.M. Wade, A. Kirby, D. Heckerman, M.J. Daly, and E. Eskin. 2008. Efficient control of population structure in model organism association mapping. Genetics 178:1709-1723.

Kawasaki, T., K. Henmi, E. Ono, S. Hatakeyama, M. Iwano, H. Satoh, and K. Shimamoto. 1999. The small GTP-binding protein Rac is a regulator of cell death in plants. Proc. Natl. Acad. Sci. 96:10922-10926.

Keller, B., C. Manzanares C. Jara, J.D. Lobaton, B. Studer, and B. Raatz. 2015. Fine-mapping of a major QTL controlling angular leaf spot resistance in common bean (*Phaseolus vulgaris* L.). Theor. Appl. Genet. 128:813-826.

Kim, Y., N. Lin, and G. Martin. 2002. Two distinct *Pseudomonas* effector proteins interact with the *Pto* kinase and activate plant immunity. Cell 109:589-598.

Kobe, B., and J. Deisenhofer. 1994. The leucine-rich repeat: A versatile binding motif. Trends Biochem. Sci. 19:415-421.

Kong, D., M. Li, Z. Dong, H. Ji, and X. Li. 2015. Identification of TaWD40D, a wheat WD40 repeat-containing protein that is associated with plant tolerance to abiotic stresses. Plant Cell Rep. 34:395-410.

Lamb, C., and R.A. Dixon. 1997. The oxidative burst in plant disease resistance. Annu. Rev. Plant Biol. 48:251-275.

Larsson, S.J., A.E. Lipka, and E.S. Buckler. 2013. Lessons from *Dwarf8* on the strengths and weaknesses of structured association mapping. PLoS Genetics 9:e1003246.

Leister, R.T., D. Dahlbeck, B. Day, Y. Li, O. Chesnokova, and B.J. Staskawicz. 2005. Molecular genetic evidence for the role of SGT1 in the intramolecular complementation of Bs2 protein activity in *Nicotiana benthamiana*. Plant Cell 17:1268-1278.

Li, H., Z. He, G. Lu, S.C. Lee, J. Alonso, J.R. Ecker, and S. Luan. 2007. A WD40 domain cyclophilin interacts with histone H3 and functions in gene repression and organogenesis in *Arabidopsis*. Plant Cell 19:2403-2416.

Lipka, A.E., F. Tian, Q. Wang, J. Peiffer, M. Li, P.J. Bradbury, M.A. Gore, E.S. Buckler, and Z. Zhang. 2012. GAPIT: Genome association and prediction integrated tool. Bioinformatics 28:2397-2399.

Mamidi, S., S. Chikara, R.J. Goos, D.L. Hyten, D. Annam, S.M. Moghaddam, R.K. Lee, P.B. Cregan, and P.E. McClean. 2011. Genome-wide association analysis identifies candidate genes associated with iron deficiency chlorosis in soybean. The Plant Genome 4:154-164.

Mamidi, S., M. Rossi, S.M. Moghaddam, D. Annam, R. Lee, R. Papa, and P.E. McClean. 2013. Demographic factors shaped diversity in the two gene pools of wild common bean *Phaseolus vulgaris* L. Heredity 110:267-276.

Martin, G.B., S.H. Brommonschenkel, J. Chunwongse, A. Frary, M.W. Ganal, R. Spivey, T. Wu, E.D. Earle, and S.D. Tanksley. 1993. Map-based cloning of a protein kinase gene conferring disease resistance in tomato. Science 262:1432-1436.

McClean, P.E., J. Terpstra, M. McConnell, C. White, R. Lee, and S. Mamidi. 2012. Population structure and genetic differentiation among the USDA common bean (*Phaseolus vulgaris* L.) core collection. Genetic resources and crop evolution. 59:499-515.

Meziadi, C., M.M. Richard, A. Derquennes, V. Thareau, S. Blanchet, A. Gratias, S. Pflieger, and V. Geffroy. 2016. Development of molecular markers linked to disease resistance genes in common bean based on whole genome sequence. Plant Sci. 242:351-357.

Miklas, P.N., R. Delorme, R. Hannan, and M. 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., D. Fourie, J. Trapp, R.C. Larsen, C. Chavarro, M.W. Blair, and P. Gepts. 2011. Genetic characterization and molecular mapping gene for resistance to halo blight in common bean. Crop Sci. 51:2439-2448.

Miklas, P.N., D. Fourie, J. Trapp, J. Davis, and J.R. Myers. 2014. New loci including conferring resistance to halo bacterial blight on chromosome Pv04 in common bean. Crop Sci. 54:2099-2108.

Miklas, P.N., D. Fourie, J. Wagner, R.C. Larsen, and C.M.S. Mienie. 2009. Tagging and mapping *Pse-1* gene for resistance to halo blight in common bean differential cultivar UI-3. Crop Sci. 49:41-48.

Mills, L.J., and M.J. Silbernagel. 1992. A rapid screening technique to combine resistance to halo blight and bean common mosaic virus in *Phaseolus vulgaris* L. Euphytica 58:201-208.

Moffett, P., G. Farnham, J. Peart, and D.C. Baulcombe. 2002. Interaction between domains of a plant NBS-LRR protein in disease resistance-related cell death. EMBO J. 21:4511-4519.

Neill, S., R. Desikan, and J. Hancock. 2002. Hydrogen peroxide signalling. Curr. Opin. Plant Biol. 5:388-395.

Nemli, S., T.K. Asciogul, H.B. Kaya, A. Kahraman, D. Eşiyok, and B. Tanyolac. 2014. Association mapping for five agronomic traits in the common bean (*Phaseolus vulgaris* L.). J. Sci. Food and Agric. 94:3141-3151.

Nesi, N., C. Jond, I. Debeaujon, M. Caboche, and L. Lepiniec. 2001. The *Arabidopsis* TT2 gene encodes an R2R3 MYB domain protein that acts as a key determinant for proanthocyanidin accummulation in developing seed. Plant Cell 13:2099–2114.

Noël, L., T.L. Moores, E.A. Van Der Biezen, M. Parniske, M.J. Daniels, J.E. Parker, and J.D. Jones. 1999. Pronounced intraspecific haplotype divergence at the *RPP5* complex disease resistance locus of *Arabidopsis*. The Plant Cell 11:2099-2111.

Nordborg, M., and S. Tavare. 2002. Linkage disequilibrium: What history has to tell us. Trends Genet. 18:83-90.

Ono, E., H.L. Wong, T. Kawasaki, M. Hasegawa, O. Kodama, and K. Shimamoto. 2001. Essential role of the small GTPase Rac in disease resistance of rice. Proc. Natl. Acad. Sci. 98:759-764.

Parker, J.E., M.J. Coleman, V. Szabò, L.N. Frost, R. Schmidt, E.A. Van der Biezen, T. Moores, C. Dean, M.J. Daniels, and J.D.G. Jones. 1997. The *Arabidopsis* downy mildew resistance

gene *RPP5* shares similarity to the Toll and interleukin-1 receptors with N and L6. Plant Cell 9:879-894.

Pérez-Vega, E., N. Trabanco, A. Campa, and J.J. Ferreira. 2013. Genetic mapping of two genes conferring resistance to powdery mildew in common bean (*Phaseolus vulgaris* L.). Theor. Appl. Genet. 126:1503-1512.

Perseguini, J.M.K.C., P.R. Oblessuc, J.R.B.F. Rosa, K.A. Gomes, A.F. Chiorato, S.A.M. Carbonell, A.A.F. Garcia, R.P. Vianello, and L.L. Benchimol-Reis. 2016. Genome-wide association studies of anthracnose and angular leaf spot resistance in common bean (*Phaseolus vulgaris* L.). PLoS ONE 11:1-19.

Porch, T.G., K. Cichy, M.A. Pastor-Corrales, M.A. Grusak, J.S. Beaver, J. Hart, D. Fourie, C. Estevez de Jensen, S. Nchimbi, and P.N. Miklas. Characterization and application of the Andean Diversity Panel for the improvement of common bean productivity in Sub-Saharan Africa. Poster session presented at the meeting of the Pan-African Grain Legume & World Cowpea Conference, 2016 February 28 – March 4, Livingstone, Zambia.

Price, A.L., N.J. Pattterson, R.M. Plenge, M.E. Weinblatt, N.A. Shadick, and D. Reich. 2006. Principal components analysis corrects for stratification in genome-wide association studies. Nat. Genet. 38:904-909.

Rairdan, G.J., and P. Moffett. 2006. Distinct domains in the ARC region of the potato resistance protein Rx mediate LRR binding and inhibition of activation. The Plant Cell 8:2082-2093.

Riedl, S.J., W. Li, Y. Chao, R. Schwarzenbacher, and Y. Shi. 2005. Structure of the apoptotic protease-activating factor 1 bound to ADP. Nature 434:926-933.

Ronald, P.C., J.M. Salmeron, F.M. Carland, and B.J. Staskawicz. 1992. The cloned avirulence gene *AvrPto* induces disease resistance in tomato cultivars containing the *Pto* resistance gene. J. Bacteriol. 174:1604-1611.

Scheet, P., and M. Stephens. 2006. A fast and flexible statistical model for large-scale population genotype data: Applications to inferring missing genotypes and haplotypic phase. Am. J. Hum. Genet. 78:629-644.

Schmutz, J., P.E. McClean, S. Mamidi, G.A. Wu, S.B. Cannon, J. Grimwood, J. Jenkins, S. Shu, Q. Song, C. Chavarro, M. Torres-Torres, V. Geffroy, S.M. Moghaddam, D. Gao, B. Abernathy, K. Barry, M. Blair, M.A. Brick, M. Chovatia, P. Gepts, D.M. Goodstein, M. Gonzales, U. Hellsten, D.L. Hyten, G. Jia, J.D. Kelly, D. Kudrna, R. Lee, M.M.S. Richard, P.N. Miklas, J.M. Osorno, J. Rodrigues, V. Thareau, C.A. Urrea, M. Wang, Y. Yu, M. Zhang, R.A. Wing, P.B. Cregan, D.S. Rokhsar, and S.A. Jackson. 2014. A reference genome for common bean and genome-wide analysis of dual domestications. Nat. Genet. 46:707-713. doi: 10.1038/ng.3008

Shi, C., A. Navabi, and K. Yu. 2011. Association mapping of common bacterial blight resistance QTL in Ontario bean breeding populations. BMC Plant Biol. 52:1-11.

Song, Q., G. Jia, D.L. Hyten, J. Jenkins, E.Y. Hwang, S.G. Schroeder, J.M. Osorno, J. Schmutz, S.A. Jackson, P.E. McClean, and P.B. Cregan. 2015. SNP assay development for linkage map construction, anchoring whole genome sequence, and other genetic and genomic applications in common bean. G3: Genes|Genomes|Genetics 5:2285-2290.

Staskawicz, B.J., F.M. Ausubel, B.J. Baker, J.G. Ellis, and J.D.G. Jones. 1995. Molecular genetics of plant disease resistance. Science 268:661-667.

Stich, B., J. Möhring, H.P. Piepho, M. Heckenberger, E.S. Buckler, and A.E. Melchinger. 2008. Comparison of mixed-model approaches for association mapping. Genetics 178:1745-1754.

Stirnimann, C.U., E. Petsalaki, R.B. Russell, and C.W. Müller. 2010. WD40 proteins propel cellular networks. Trends Biochem Sci. 35:565-574.

Taylor, J.D., D.M. Teverson, M.A. Allen, and M.A. Pastor-Corrales. 1996a. Identification and origin of races of *Pseudomonas syringae* pv. *phaseolicola* from Africa and other bean growing areas. Plant Pathol. 45:469-478.

Taylor, J.D., D.M. Teverson, and J.H.C. Davis. 1996b. Sources of resistance to *Pseudomonas syringae* pv. *phaseolicola* races in *Phaseolus vulgaris.* Plant Pathol. 45:479-485.

Trabanco, N., M.C. Asensio-Manzanera, E. Perez-Vega, A. Ibeas, A. Campa, and J.J. Ferreira. 2014. Identification of quantitative trait loci involved in the response of common bean to *Pseudomonas syringae* pv. *phaseolicola*. Mol. Breeding. 33:577-588.

Van der Biezen, E.A., and J.D.G. Jones. 1998. The NB-ARC domain: A novel signaling motif shared by plant resistance gene products and regulators of cell death in animals. Curr. Biol. 8:R226-R227.

van der Vossen, E.A., J.N. van der Voort, K. Kanyuka, A. Bendahmane, H. Sandbrink, D.C. Baulcombe, J. Bakker, W.J. Stiekema, and R.M. Klein-Lankhorst. 2000. Homologues of a single resistance-gene cluster in potato confer resistance to distinct pathogens: A virus and a nematode. Plant J. 23:567-576.

Van Ooijen, G., G. Mayr, M.M. Kasiem, M. Albrecht, B.J. Cornelissen, and F.L. Takken. 2008. Structure-function analysis of the NB-ARC domain of plant disease resistance proteins. J. Exp. Bot. 59:1383-1397.

Vasquez Guzman, J.E. 2016. Identifying dry bean genotypes and genomic regions associated with root rot resistance with emphasis on *Fusarium solani* f.sp. *phaseoli*. Ph.D. Thesis, North Dakota State University, Fargo, ND.

Vaux, D. L., and S.J. Korsmeyer. 1999. Cell death in development. Cell 96:245-254.

Wang, D., K. Pajerowska-Mukhtar, A.H. Culler, and X. Dong. 2007. Salicylic acid inhibits pathogen growth in plants through repression of the auxin signaling pathway. Curr. Biol. 17:1784-1790.

Woodward, A.W., and B. Bartel. 2005. Auxin: Regulation, action, and interaction. Ann. Bot. 95:707-735.

Zhang, Z., E. Ersoz, C.Q. Lai, R.J. Todhunter, H.K. Tiwari, M.A. Gore, P.J. Bradbury, J. Yu, D.K. Arnett, J.M. Ordovas, and E.S. Buckler. 2010. Mixed linear model approach adapted for genome-wide association studies. Nat. Genet. 42:355-360.

Zhao, H.H., R.L. Fernando, and J.C.M. Dekkers. 2007. Power and precision of alternate methods for linkage disequilibrium mapping of quantitative trait loci. Genetics 175:1975-1986.

Zhu, J., J.C. Jeong, Y. Zhu, I. Sokolchik, S. Miyazaki, J.K. Zhu, P.M. Hasegawa, H.J. Bohnert, H. Shi, D.J. Yun, and R.A. Bressan. 2008. Involvement of *Arabidopsis* HOS15 in histone deacetylation and cold tolerance. PNAS. 12:4945-4950.

GENERAL SUMMARY AND CONCLUSIONS

Knowledge on the phenotypic diversity and its relationship with genetic diversity in the crop species offer opportunities for discovering unexploited traits that is crucial for crop improvement. In that respect, core collection represents plant resources with adequate genetic variation prerequisite for improvement of any crop species for several biotic and abiotic stresses. However, unlike in CIAT collection, exploration of the phenotypic diversity existed in the USDA-NPGS core collection of common bean is scarce, especially in terms of resistance to race 6 of *Pseudomonas syringae* pv. *phaseolicola* (*Psp*). Therefore, the current study was conducted with the following objectives (i) to identify resistant germplasm to race 6 of *Psp* from the USDA-NPGS bean core-collection in the greenhouse following unifoliate (primary) leaf inoculation, (ii) to identify most appropriate leaf stages, i.e., unifoliate versus trifoliate, for the evaluation of race 6 of *Psp*, (iii) to evaluate the levels of resistance in leaves vs. pods inoculation within an individual plant, (iv) to evaluate a group of selected accessions for resistance to race 6 of *Psp* under the field conditions, and (v) to identify genomic regions linked to resistance to race 6 of *Psp* using a Genome-Wide Association Mapping Study (GWAS) approach.

Inoculation of unifoliate (primary) leaves following multiple-needle pin frog method successfully categorized the different levels of halo blight severity to race 6 of *Psp.* Based on the evaluation of disease symptoms 10 days post inoculation (dpi) in unifoliate leaves, 37 accessions from a total of 281 displayed resistant reaction to the disease, where the majority of accessions were from Mesoamerican origin, i.e., Mexico. Two accessions from this resistant category, PI 207373 and PI 290990 from Colombia and Peru, were also found to be highly resistant to other common bean diseases such as white mold and Fusarium wilt, and categorize itself as potential accessions with broad-spectrum disease resistance.

Under greenhouse conditions, the incidence of higher halo blight severity in trifoliate stage compared to unifoliate distinctly categorized the different levels of halo blight reaction. Similarly, the weak correlation ($r^2 = 0.1789$; $P < 0.05$) for halo blight severity observed between the trifoliate and pod within an individual plant accession suggested the role of independent genes controlling for disease resistance at specific plant growth stages. Eight accession accessions, PI 201343, PI 313217, PI 313328, PI 313343, PI 313490, PI 313596, PI 325653, and PI 417657 exhibited consistent resistant disease reactions across all development stages, i.e., unifoliate, trifoliate, and pods.

Contrary to greenhouse results, significant differences in halo blight severity in trifoliate and pod stage were observed under field conditions. Like that, under greenhouse conditions, a weak correlation (r^2=0.2131; $P < 0.05$) between halo blight severity in trifoliate to pod stage was observed. One accession, PI 313217, exhibited resistant reaction to both trifoliate and pod stages when evaluated under greenhouse conditions.

Using genome-wide association study (GWAS), three significant SNPs, two previously identified in Pv04 and one novel in Pv05, explaining a total of 19% phenotypic variation were detected in this study. Based on the PV annotation data, GWAS identified three candidate genes on Pv05, *Phvul.005G170900, Phvul.005G16990,* and *Phvul.005G169600,* that code for Ras-related small GTP-binding family protein, WD-40 repeat protein, and Indole Acetic Acid Protein, respectively, and are involved in plant self-defense mechanisms during pathogen attack. In addition, the current study also detected the new QTL in Pv04 that governed for resistance to this disease and may be of interest to common bean improvement. Four candidate genes, *Phvul.004G007600, Phvul.004G007700, Phvul.004G012600* and *Phvul.004G012800*, were found co-localizing with significant SNPs on Pv04. Two SNPs *Phvul.004G007700* and

Phvul.004G012800 code for a NB-ARC (Nucleotide-binding ARC) domain containing disease resistance protein, while SNP *Phvul.004G012600* code for a first reported cloned plant gene, Tomato *Pto* gene, that encode serine/threonine protein kinases responsible for disease resistance.

From a practical standpoint, the ability to detect novel sources of resistance to plant disease using GWAS will be of considerable utility. Since this is the first attempt to evaluate and identify the genomic regions related to resistance to race 6 of *Psp* using USDA-NPGS common bean core collection, the three significant SNPs could be potential candidates for marker-assisted selection (MAS). The future validation of current significant SNPs in other segregating populations and environments could be of importance to develop resistance to race 6 of *Psp* for marker-assisted selection (MAS). Nonetheless, the effectiveness of more molecular markers governing for enhanced resolution should be practiced to identify strong significant marker-QTL association controlling for trait of interests.

APPENDIX

Table A.1. Country of origin, type, and seed color of 383 common bean accessions from USDA-NPGS core collection.

Accession	Country of origin	Type	Seed Color ‡	Accession	Country of origin	Type	Seed Color ‡
145886	Iran	Cultivated	BL/DR	201324	Mexico	Landrace	DT/GE
150957	Mexico	Landrace	BL	201329	Mexico	Landrace	TN/LT
151407	Colombia	Landrace	WH	201343	Mexico	Landrace	DY
152208	Bolivia	Landrace	DR	201360	Mexico	Landrace	LB
152311	Ecuador	Landrace	BL	201369	Mexico	Landrace	WH
165422	Mexico	Landrace	BR	201370	Mexico	Landrace	BL
165423	Mexico	Landrace	RD/LR	201387	Mexico	Landrace	DY
165455	Mexico	Landrace	RD	201388	Mexico	Landrace	DY/BR
165462	Mexico	Landrace	BL	201480	Mexico	Landrace	DB
165466	Mexico	Landrace	BL	202834	Mexico	Landrace	LY
182000	Guatemala	Landrace	WH	202835	Mexico	Landrace	LT
182004	Guatemala	Landrace	BR	203920	Mexico	Landrace	RD
189407	Guatemala	Landrace	WH	203921	Mexico	Landrace	BL
189408	Guatemala	Landrace	CR/DR/RD	203924	Mexico	Landrace	BL
194574	Guatemala	Landrace	WH	203934	Mexico	Landrace	CR/LT
195402	Guatemala	Landrace	WH	203936	Mexico	Landrace	BR
196463	Nicaragua	Landrace	BR	203958	Mexico	Landrace	BL
197031	El Salvador	Landrace	BL	206223	Honduras	Landrace	BL/RD
198026	Peru	Landrace	WH	207127	Colombia	Landrace	CR/DP
198037	Peru	Landrace	BL	207136	Colombia	Landrace	BL/RD
200956	El Salvador	Landrace	BL	207148	Colombia	Landrace	LR/WH/BR
200967	Guatemala	Landrace	BL	207154	Colombia	Landrace	DY
201004	Guatemala	Landrace	BL	207165	Colombia	Landrace	RD
201010	Guatemala	Landrace	CR	207180	Colombia	Landrace	LT
201296	Mexico	Landrace	DY	207182	Colombia	Landrace	DB
207186	Colombia	Landrace	LG	260418	Bolivia	Landrace	WH/TN
207193	Colombia	Landrace	TN	263593	Mexico	Landrace	CR
207203	Colombia	Landrace	LB/BR	263596	Mexico	Landrace	LB/WH/GE
207207	Colombia	Landrace	DB	269209	Peru	Landrace	RD/DR
207216	Colombia	Landrace	DR	269210	Peru	Landrace	LB
207253	Colombia	Landrace	CR/LT	288016	Nicaragua	Cultivated	BL
207279	Colombia	Landrace	LB	290990	Peru	Landrace	CR/RD
207300	Colombia	Landrace	CR/LT	290995	Peru	Landrace	CR/RD
207322	Colombia	Landrace	BL	293353	Peru	Cultivated	DP/PU
207336	Colombia	Landrace	LY	293355	Peru	Cultivated	PI/LR
207373	Colombia	Landrace	BL	297295	El Salvador	Cultivated	RD/DR
207389	Colombia	Landrace	CR/LR	299019	Ecuador	Landrace	YL
207420	Colombia	Landrace	RD	304110	El Salvador	Landrace	WH
207428	Colombia	Landrace	PI	304113	El Salvador	Landrace	BL
207443	Colombia	Landrace	WH	306200	Peru	Cultivated	CR/CR
208774	Nicaragua	Cultivated	RD	307788	El Salvador	Landrace	BR
209479	Nicaragua	Landrace	BL	307790	El Salvador	Landrace	LR
209482	Costa Rica	Landrace	WH	307791	El Salvador	Landrace	BL
209486	Costa Rica	Landrace	CR/RD	307806	El Salvador	Landrace	DR
209491	Costa Rica	Landrace	BL	307808	El Salvador	Landrace	DB/RD
209498	Costa Rica	Landrace	BL	307810	El Salvador	Landrace	BL

Table A.1. Country of origin, type, and seed color of 383 common bean accessions from USDA-NPGS core collection (continued).

Accession	Country of origin	Type	Seed Color ‡	Accession	Country of origin	Type	Seed Color ‡
224715	Mexico	Landrace	BL	307816	El Salvador	Landrace	BL
224718	Mexico	Landrace	BL	307820	El Salvador	Landrace	BL
224728	Mexico	Landrace	RD	308894	Costa Rica	Landrace	LR/PI/BR
241794	Ecuador	Landrace	CR	308898	Costa Rica	Landrace	LT
309698	Mexico	Landrace	DT	310751	Guatemala	Landrace	WH
309700	Mexico	Landrace	CR/LT	310761	Guatemala	Landrace	WH
309701	Mexico	Landrace	CR	310778	Guatemala	Landrace	BL
309715	Mexico	Landrace	DP/PI	310786	Guatemala	Landrace	RD
309759	Mexico	Landrace	LB/BR	310814	Nicaragua	Cultivated	PI/DT
309787	Mexico	Landrace	YL	310818	Nicaragua	Cultivated	BR
309810	Mexico	Landrace	LY	310826	Nicaragua	Cultivated	BR
309823	Costa Rica	Landrace	CR/BL	310829	Nicaragua	Cultivated	RD/DR
309825	Costa Rica	Landrace	CR/BL	310836	Nicaragua	Cultivated	PI
309827	Costa Rica	Landrace	CR/WH/LT	310842	Nicaragua	Cultivated	BL
309830	Costa Rica	Landrace	RD/LR	310850	Nicaragua	Cultivated	PI
309837	Costa Rica	Landrace	RD/LR	310886	Nicaragua	Cultivated	LB
309844	Costa Rica	Landrace	DY	310891	Nicaragua	Cultivated	DU/WH
310511	Honduras	Landrace	WH	310915	Nicaragua	Cultivated	DR
310515	Honduras	Cultivated	RD/DR	311794	El Salvador	Landrace	DR
310546	Honduras	Cultivated	LR/RD	311807	Guatemala	Landrace	DR/RD
310556	Honduras	Cultivated	BL/DR/RD	311843	Guatemala	Landrace	CR/PU/LB
310586	Honduras	Cultivated	RD/DR	311853	Guatemala	Landrace	BL
310611	Mexico	Landrace	BL	311900	Mexico	Landrace	CR
310660	Guatemala	Landrace	WH	311940	Mexico	Landrace	BL
310663	Guatemala	Landrace	BL	311942	Mexico	Landrace	CR
310668	Guatemala	Landrace	BL	311944	Mexico	Landrace	BL
310674	Guatemala	Landrace	RD/CR	311947	Mexico	Landrace	BL
310718	Guatemala	Landrace	WH	311956	Mexico	Landrace	BL
310739	Guatemala	Landrace	BL	311962	Mexico	Landrace	RD/CR
311967	Mexico	Landrace	BL	313394	Mexico	Landrace	DE/TN
311974	Mexico	Landrace	BL	313397	Mexico	Landrace	CR
311982	Mexico	Landrace	PI/LT	313408	Mexico	Landrace	LB
311999	Mexico	Landrace	RD	313425	Mexico	Landrace	CR/LT
312016	Mexico	Landrace	BL	313429	Mexico	Landrace	CR/LT
312018	Mexico	Landrace	BL	313440	Mexico	Landrace	YL/DY
312031	Mexico	Landrace	PU	313444	Mexico	Landrace	BL
312064	Mexico	Landrace	BL	313445	Mexico	Landrace	BL
312083	Mexico	Landrace	CR	313458	Mexico	Landrace	BL
312090	Mexico	Landrace	LY/CR	313459	Mexico	Landrace	CR
312098	Mexico	Landrace	YL	313470	Mexico	Landrace	BL
313217	Mexico	Landrace	PU/RD	313473	Mexico	Landrace	TN/DT
313237	Mexico	Landrace	TN/BR/CR	313483	Mexico	Landrace	WH
313254	Mexico	Landrace	BL	313486	Mexico	Landrace	BL
313270	Mexico	Landrace	CR/DP	313487	Mexico	Landrace	BL

Table A.1. Country of origin, type, and seed color of 383 common bean accessions from USDA-NPGS core collection (continued).

Accession	Country of origin	Type	Seed Color ‡	Accession	Country of origin	Type	Seed Color ‡
313297	Mexico	Landrace	CR	313499	Mexico	Landrace	BL
313322	Mexico	Landrace	CR/LT	313501	Mexico	Landrace	CR
313328	Mexico	Landrace	BL	313512	Mexico	Landrace	DY
313333	Mexico	Landrace	BL	313531	Mexico	Landrace	CR/PI
313343	Mexico	Landrace	BL	313532	Mexico	Landrace	YL/DY
313348	Mexico	Landrace	BR/DT	313535	Mexico	Landrace	CR/LY
313357	Mexico	Landrace	CR	313537	Mexico	Landrace	CR/GE
313366	Mexico	Landrace	CR/LT	313571	Colombia	Cultivated	DR
313386	Mexico	Landrace	YL/LY	313572	Colombia	Cultivated	BL
313583	Colombia	Cultivated	CR/DT	313749	Mexico	Cultivated	BL
313592	Colombia	Cultivated	LT	313782	Mexico	Cultivated	BL
313596	Colombia	Cultivated	WH/BR	313809	Mexico	Cultivated	BL
313597	Colombia	Cultivated	RD/CR/WH	313820	Mexico	Cultivated	BL
313598	Colombia	Cultivated	YL	313830	Mexico	Cultivated	PU
313608	Colombia	Cultivated	GE	313833	Mexico	Cultivated	BL
313609	Colombia	Landrace	RD/CR	313835	Mexico	Cultivated	LR
313613	Colombia	Cultivated	CR	313837	Mexico	Cultivated	RD
313615	Colombia	Cultivated	RD	313839	Mexico	Cultivated	DR/RD
313630	Colombia	Cultivated	RD/LR	313842	Peru	Cultivated	DY
313634	Colombia	Cultivated	CR	313843	Peru	Cultivated	RP/CR/PI
313636	Colombia	Cultivated	BL	313847	Peru	Cultivated	CR/DR
313639	Colombia	Cultivated	YL/CR/TN	313850	Peru	Cultivated	DI/LR
313658	Ecuador	Cultivated	LT	316016	Peru	Cultivated	BR/DB
313664	Ecuador	Cultivated	DP/DR	316023	Peru	Cultivated	DY
313665	Ecuador	Cultivated	WH	316030	Peru	Cultivated	BL/WH
313667	Ecuador	Cultivated	CR/LT	316031	Peru	Cultivated	WH
313671	Ecuador	Cultivated	DY	317350	Mexico	Wild	GE
313674	Ecuador	Cultivated	BL	318691	Mexico	Wild	TN
313685	Ecuador	Cultivated	LT	318694	Mexico	Wild	CR
313693	Ecuador	Cultivated	YL/DY	318695	Mexico	Wild	GE
313701	Mexico	Cultivated	BL	318703	Mexico	Wild	LT
313720	Mexico	Cultivated	LB/LT/BR	319554	Mexico	Landrace	CR/LT
313727	Mexico	Cultivated	CR	319573	Mexico	Landrace	CR
313733	Mexico	Cultivated	CR/TN	319587	Mexico	Landrace	BL
319592	Mexico	Landrace	CR/WH	325750	Mexico	Cultivated	DY
319595	Mexico	Landrace	CR	326106	Honduras	Landrace	WH
319607	Mexico	Landrace	CR/LB	326110	Honduras	Landrace	BL
319618	Mexico	Landrace	CR/TN	343950	Guatemala	Wild	CR
319619	Mexico	Landrace	YL/TN	345576	Costa Rica	Landrace	WH
319636	Mexico	N/A	DR	345581	Costa Rica	Landrace	BL
319640	Mexico	Landrace	CR	346955	Mexico	Wild	LB
319674	Mexico	Landrace	CR	346960	Mexico	Cultivated	BL
319677	Mexico	Landrace	CR	355419	Ecuador	Cultivated	DR/RD
319683	Mexico	Landrace	BL/RD	387862	Bolivia	Cultivated	DY
319684	Mexico	Landrace	CR	387865	Bolivia	Cultivated	CR
325614	Mexico	Landrace	BL	387866	Bolivia	Cultivated	WH/CR
325618	Mexico	Landrace	PU	399169	Nicaragua	N/A	N/A
313272	Mexico	Landrace	PU/LP/WH	313490	Mexico	Landrace	BL

Table A.1. Country of origin, type, and seed color of 383 common bean accessions from USDA-NPGS core collection (continued).

Accession	Country of origin	Type	Seed Color ‡	Accession	Country of origin	Type	Seed Color ‡
325626	Mexico	Landrace	BL	406940	Honduras	Cultivated	CR
325630	Mexico	Landrace	BL/LB	415887	Ecuador	N/A	CR
325653	Mexico	Landrace	CR	415906	Ecuador	N/A	LB/DT/TN
325664	Mexico	Landrace	BL	415909	Ecuador	Landrace	WH
325676	Mexico	Landrace	PU	415913	Ecuador	N/A	LR
325684	Mexico	Wild	DT	415936	Ecuador	N/A	CR/PU
325685	Mexico	Wild	BL	415949	Peru	Landrace	WH/BR
325687	Mexico	Wild	BR	415950	Peru	N/A	LR/PI/RD
325691	Mexico	Wild	CR	415954	Peru	Landrace	WH/CR/WH
325731	Mexico	Cultivated	CR/LR	415955	Peru	Landrace	WH
325732	Mexico	Cultivated	CR	415975	Colombia	N/A	CR
415986	Colombia	N/A	RD/CR	417731	Mexico	Cultivated	BL
415987	Colombia	N/A	CR/GE	417739	Mexico	Landrace	BL
416468	Mexico	Cultivated	CR/WH	417742	Mexico	Landrace	BL
416713	Mexico	Cultivated	CR	417754	Mexico	Landrace	BL
417616	Mexico	Landrace	CR/LT	417778	Mexico	Wild	DT
417621	Mexico	Wild	BL	417780	Mexico	Wild	CR/TN
417622	Mexico	Wild	BL	417782	Mexico	Wild	TN/TN
417627	Mexico	Cultivated	LT	417784	Mexico	Wild	YL
417628	Mexico	Cultivated	LB	417786	Mexico	Cultivated	LB
417630	Mexico	Cultivated	CR	417790	Mexico	Landrace	DY/LY
417633	Mexico	Cultivated	PI	430167	Colombia	N/A	RD
417634	Mexico	Cultivated	PI	430201	Mexico	Wild	YL
417641	Mexico	Cultivated	DY	430204	Mexico	Cultivated	BL
417645	Mexico	Cultivated	DY	430206	Mexico	Cultivated	CR
417647	Mexico	Cultivated	BR	449389	Mexico	Cultivated	BL/PI
417653	Mexico	Cultivated	CR/YL	449410	Mexico	Cultivated	CR/DB
417654	Mexico	Cultivated	BL	449412	Mexico	Cultivated	BL
417657	Mexico	Cultivated	BL	449422	Mexico	Cultivated	BL
417667	Mexico	Cultivated	BR	451885	Guatemala	Cultivated	LR
417679	Mexico	Cultivated	CR	451889	Guatemala	Cultivated	GE
417697	Mexico	Landrace	BL	451906	Guatemala	Cultivated	CR/RD
417707	Mexico	Cultivated	BL	451917	Guatemala	Cultivated	BL
417708	Mexico	Cultivated	N/A	451921	Guatemala	Cultivated	BL
417721	Mexico	Landrace	BL	476693	Mexico	Cultivated	BL
417725	Mexico	Landrace	BL	476751	Guatemala	Cultivated	DR/RD
510574	Peru	Cultivated	LR/PU/RD	533432	Mexico	Cultivated	CR/TN
511767	Peru	Cultivated	DY	533437	Mexico	Cultivated	BL
512003	Mexico	Landrace	GE/CR	533475	Mexico	Cultivated	CR/DB
531862	Peru	Cultivated	WH	533476	Mexico	Cultivated	BL
533259	Mexico	Cultivated	BL	533484	Mexico	Cultivated	BL
533277	Mexico	Cultivated	CR/LY	533498	Mexico	Cultivated	WH
533281	Mexico	Cultivated	CR/RD	533502	Mexico	Cultivated	PU/PI
533286	Mexico	Cultivated	CR/YL	533428	Mexico	Cultivated	DP/PU/DR
533299	Mexico	Cultivated	LB	533510	Mexico	Cultivated	CR
533311	Mexico	Cultivated	LP/PU	533528	Mexico	Cultivated	CR/WH
533312	Mexico	Cultivated	BL	533545	Guatemala	Cultivated	RD/LR
533313	Mexico	Cultivated	BL	533561	Guatemala	Cultivated	LR
533316	Mexico	Cultivated	BL	533577	Ecuador	Cultivated	WH

Table A.1. Country of origin, type, and seed color of 383 common bean accessions from USDA-NPGS core collection (continued).

Accession	Country of origin	Type	Seed Color‡	Accession	Country of origin	Type	Seed Color‡
533332	Mexico	Cultivated	YL	533584	Ecuador	Cultivated	BL/LB
533363	Ecuador	Cultivated	LB/DT	535395	Mexico	Cultivated	CR
533373	Mexico	Cultivated	BL	557483	Ecuador	Cultivated	RD/DR
533420	Mexico	Cultivated	DY				

‡ Seed color where: BL = black; BR = Brown; CR = Cream-beige; DY = dark yellow; LRK = Light red kidney; LT = Light tan; LY = Light yellow; WH = White; TN/LT = Tan/Light tan; CR/DP = Cream-beige/Dark purple; CR/RD = Cream-beige/Red; TN/BR/CR = Tan/Brown/Cream-beige; DR/RD = Red/Dark red; CR/PU/LB = Cream-beige/Purple/Light brown; PU/RD = Purple/Red; TN/BR = Tan/Brown; DE/TN = Dark Grey/Tan; WH/BR = White/Brown; CR/WH = Cream-beige/White; CR/DB = Cream-beige/Dark brown; DR = Dark Tan; N/A = Not Available.

Table A.2. Country of origin, type, seed color and mean halo blight scores in unifoliate leaves of 281 common bean accessions from USDA-NPGS core collection evaluated under greenhouse condition at North Dakota State University, Fargo, ND.

Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡	Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡
533259	1.0 ± 0.5	Mexico	Landrace	BL	308898	2.1 ± 0.9	Costa Rica	Landrace	LT
201329	1.1 ± 0.9	Mexico	Landrace	DY	310818	2.1 ± 0.9	Nicaragua	Landrace	BR
309810	1.1 ± 1.0	Mexico	Landrace	LY	311843	2.1 ± 0.9	Guatemala	Landrace	CR/PU/LB
310826	1.1 ± 0.9	Nicaragua	Landrace	BR	313254	2.1 ± 0.9	Mexico	Landrace	BL
319592	1.1 ± 1.0	Mexico	Landrace	CR/WH	313490	2.1 ± 0.9	Mexico	Landrace	BL
417657	1.2 ± 0.9	Mexico	Landrace	BL	311974	2.2 ± 0.9	Mexico	Landrace	BL
290990	1.3 ± 1.0	Peru	Landrace	CR/RD	313572	2.2 ± 0.9	Colombia	Landrace	BL
313343	1.3 ± 0.9	Mexico	Landrace	BL	313217	2.3 ± 0.9	Mexico	Landrace	PU/RD
449410	1.3 ± 0.9	Mexico	Landrace	CR/DB	313237	2.3 ± 0.9	Mexico	Landrace	TN/BR/CR
201296	1.5 ± 0.9	Mexico	Landrace	DY	313394	2.3 ± 0.9	Mexico	Landrace	DE/TN
313596	1.6 ± 1.0	Colombia	Landrace	WH/BR	313809	2.3 ± 0.9	Mexico	Landrace	BL
531862	1.6 ± 0.9	Peru	Landrace	WH	325653	2.4 ± 1.0	Mexico	Landrace	CR
201343	1.7 ± 0.9	Mexico	Landrace	TN/LT	476751	2.4 ± 0.9	Guatemala	Landrace	DR/RD
310829	1.7 ± 0.9	Nicaragua	Landrace	RD/DR	203934	2.5 ± 0.9	Mexico	Landrace	CR/LT
415949	1.7 ± 0.9	Peru	Landrace	WH/BR	288016	2.5 ± 0.9	Nicaragua	Landrace	BL
207373	1.8 ± 0.9	Colombia	Landrace	BL	313357	2.5 ± 0.9	Mexico	Landrace	CR
209479	1.8 ± 0.9	Nicaragua	Landrace	BL	313598	2.5 ± 0.9	Colombia	Landrace	YL
325732	1.8 ± 0.9	Mexico	Landrace	CR	196463	2.6 ± 0.9	Nicaragua	Landrace	BR
533475	1.8 ± 0.9	Mexico	Landrace	CR/DB	310786	2.6 ± 0.9	Guatemala	Landrace	RD
207322	2.0 ± 0.9	Colombia	Landrace	BL	311967	2.6 ± 0.9	Mexico	Landrace	BL
313328	2.0 ± 0.9	Mexico	Landrace	BL	417725	2.6 ± 0.9	Mexico	Landrace	BL
533476	2.0 ± 0.9	Mexico	Landrace	BL	182004	2.7 ± 0.9	Guatemala	Landrace	BR
200956	2.1 ± 0.9	El Salvador	Landrace	BL	203958	2.7 ± 0.9	Mexico	Landrace	BL
207127	2.1 ± 0.9	Colombia	Landrace	CR/DP	325618	3.1 ± 0.9	Mexico	Landrace	CR
307806	2.7 ± 0.9	El Salvador	Landrace	DR	207216	2.7 ± 0.9	Colombia	Landrace	DR
313408	2.7 ± 0.9	Mexico	Landrace	LB	533437	3.1 ± 1.0	Mexico	Landrace	BL
313664	2.7 ± 0.9	Ecuador	Landrace	DP/DR	533528	3.1 ± 0.9	Mexico	Landrace	CR/WH
533299	2.7 ± 0.9	Mexico	Landrace	LB	207182	3.2 ± 0.9	Colombia	Landrace	DB
189407	2.8 ± 0.9	Guatemala	Landrace	WH	307790	3.2 ± 0.9	El Salvador	Landrace	LR
195402	2.8 ± 0.9	Guatemala	Landrace	WH	310718	3.2 ± 0.9	Guatemala	Landrace	WH
313636	2.8 ± 0.9	Colombia	Landrace	BL	313348	3.2 ± 0.9	Mexico	Landrace	BR/DT
325630	2.8 ± 0.9	Mexico	Landrace	BL/LB	533584	3.2 ± 0.9	Ecuador	Landrace	BL/LB
476693	2.8 ± 0.9	Mexico	Landrace	BL	200967	3.3 ± 0.9	Guatemala	Landrace	BL
319618	2.8 ± 0.9	Mexico	Landrace	CR/TN	309701	3.3 ± 0.9	Mexico	Landrace	CR
311942	3.0 ± 0.9	Mexico	Landrace	CR	310814	3.3 ± 0.9	Nicaragua	Landrace	PI/DT

Table A.2. Country of origin, type, seed color and mean halo blight scores in unifoliate leaves of 281 common bean accessions from USDA-NPGS core collection evaluated under greenhouse condition at North Dakota State University, Fargo, ND (continued).

Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡	Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡
307810	3.0 ± 0.9	El Salvador	Landrace	BL	312090	3.3 ± 0.9	Mexico	Landrace	LY/CR
310751	3.0 ± 0.9	Guatemala	Landrace	WH	430167	3.3 ± 0.9	Colombia	N/A	RD
310761	3.0 ± 0.9	Guatemala	Landrace	WH	313727	3.4 ± 1.0	Mexico	Landrace	CR
313608	3.0 ± 1.0	Colombia	Landrace	/GE	201480	3.5 ± 0.9	Mexico	Landrace	DB
319607	3.0 ± 0.9	Mexico	Landrace	CR/LB	207389	3.5 ± 0.9	Colombia	Landrace	CR/LR
417754	3.0 ± 0.9	Mexico	Landrace	BL	207428	3.5 ± 0.9	Colombia	Landrace	LB/BR
430204	3.0 ± 0.9	Mexico	Landrace	BL	310668	3.5 ± 0.9	Guatemala	Landrace	BL
165422	3.1 ± 0.9	Mexico	Landrace	BR	311853	3.5 ± 0.9	Guatemala	Landrace	RD
299019	3.1 ± 0.9	Ecuador	Landrace	WH	313458	3.5 ± 1.0	Mexico	Landrace	BL
307791	3.1 ± 0.9	El Salvador	Landrace	BL	313501	3.5 ± 0.9	Mexico	Landrace	CR
307820	3.1 ± 0.9	El Salvador	Landrace	BL	313615	3.5 ± 0.9	Colombia	Landrace	RD
310511	3.1 ± 0.9	Honduras	Landrace	WH	313674	3.5 ± 0.9	Ecuador	Landrace	BL
415954	3.1 ± 0.9	Peru	Landrace	WH/CR/WH	319619	3.7 ± 0.9	Mexico	Landrace	YL/TN
417739	3.1 ± 0.9	Mexico	Landrace	BL	415955	3.7 ± 0.9	Peru	Landrace	WH
201010	3.2 ± 0.9	Guatemala	Landrace	CR	201324	3.8 ± 0.9	Mexico	Landrace	DT/GE
207154	3.2 ± 0.9	Colombia	Landrace	DY	325664	3.5 ± 0.9	Mexico	Landrace	BL
415975	3.5 ± 0.9	Colombia	N/A	CR	345576	3.5 ± 0.9	Costa Rica	Landrace	WH
430206	3.5 ± 0.9	Mexico	Landrace	CR	313333	3.8 ± 1.0	Mexico	Landrace	BL
533312	3.5 ± 0.9	Mexico	Landrace	BL	313733	3.8 ± 0.9	Mexico	Landrace	CR/TN
533313	3.5 ± 0.9	Mexico	Landrace	BL	346960	3.8 ± 1.0	Mexico	Landrace	BL
533428	3.5 ± 0.9	Mexico	Landrace	DP/PU/DR	449422	3.8 ± 0.9	Mexico	Landrace	BL
207279	3.6 ± 0.9	Colombia	Landrace	LB	533363	3.8 ± 0.9	Ecuador	Landrace	LB/DT
241794	3.6 ± 0.9	Ecuador	Landrace	CR	533561	3.8 ± 0.9	Guatemala	Landrace	LR
309837	3.6 ± 0.9	Costa Rica	Landrace	RD/LR	313842	3.9 ± 1.0	Peru	Landrace	DY
310915	3.6 ± 1.0	Nicaragua	Landrace	DR	151407	4.0 ± 0.9	Colombia	Landrace	WH
313272	3.6 ± 0.9	Mexico	Landrace	PU/LP/WH	309827	4.0 ± 0.9	Costa Rica	Landrace	CR/WH/LT
313444	3.6 ± 1.0	Mexico	Landrace	BL	311900	4.0 ± 0.9	Mexico	Landrace	CR
415950	3.6 ± 0.9	Peru	N/A	LR/PI/RD	312031	4.0 ± 1.0	Mexico	Landrace	BL
415987	3.6 ± 0.9	Colombia	N/A	DU/WH	312098	4.0 ± 1.0	Mexico	Landrace	YL
417616	3.6 ± 0.9	Mexico	Landrace	CR/LT	326110	4.0 ± 0.9	Honduras	Landrace	BL
449389	3.6 ± 0.9	Mexico	Landrace	BL	345581	4.0 ± 0.9	Costa Rica	Landrace	BL
201004	3.7 ± 0.9	Guatemala	Landrace	BL	399169	4.0 ± 0.9	Nicaragua	N/A	N/A
311807	3.7 ± 0.9	Guatemala	Landrace	DR/RD	533277	4.0 ± 0.9	Mexico	Landrace	CR/LY
313429	3.7 ± 1.0	Mexico	Landrace	CR/LT	150957	4.1 ± 0.9	Mexico	Landrace	BL
207148	3.4 ± 1.0	Colombia	Landrace	LR/WH/BR	203920	4.1 ± 0.9	Mexico	Landrace	RD
308894	3.4 ± 0.9	Costa Rica	Landrace	LR/PI/BR	207186	4.1 ± 0.9	Colombia	Landrace	LG
311956	3.4 ± 1.0	Mexico	Landrace	BL	311794	4.1 ± 1.0	El Salvador	Landrace	DR
309823	3.6 ± 0.8	Costa Rica	Landrace	CR/BL	313701	4.1 ± 1.0	Mexico	Landrace	BL
533281	3.7 ± 0.4	Mexico	Cultivated	CR/RD	208774	4.5 ± 0.9	Nicaragua	Landrace	RD
313665	3.7 ± 0.4	Ecuador	Cultivated	WH	313843	4.1 ± 0.9	Peru	Landrace	RP/CR/PI
207165	3.8 ± 0.9	Colombia	Landrace	RD	310611	4.2 ± 0.4	Mexico	Landrace	BL
297295	3.8 ± 0.9	El Salvador	Landrace	YL	165455	4.2 ± 0.9	Mexico	Landrace	BL
309844	3.8 ± 0.9	Costa Rica	Landrace	DY	165466	4.2 ± 0.9	Mexico	Landrace	BL
198026	4.2 ± 0.9	Peru	Landrace	WH	313322	4.5 ± 0.9	Mexico	Landrace	CR/LT
310674	4.2 ± 0.9	Guatemala	Landrace	RD/CR	533316	4.5 ± 0.9	Mexico	Landrace	BL
310836	4.2 ± 0.9	Nicaragua	Landrace	PI	310586	4.6 ± 0.9	Honduras	Landrace	RD/DR
313583	4.2 ± 0.9	Colombia	Landrace	CR/DT	310739	4.6 ± 0.9	Guatemala	Landrace	BL
313720	4.2 ± 0.9	Mexico	Landrace	BL	313634	4.6 ± 0.9	Colombia	Landrace	CR
417742	4.2 ± 0.9	Mexico	Landrace	BL	313667	4.6 ± 1.0	Ecuador	Landrace	CR/LT

Table A.2. Country of origin, type, seed color and mean halo blight scores in unifoliate leaves of 281 common bean accessions from USDA-NPGS core collection evaluated under greenhouse condition at North Dakota State University, Fargo, ND (continued).

Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡	Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡
533484	4.2 ± 0.9	Mexico	Landrace	BL	416713	4.6 ± 0.9	Mexico	Landrace	CR
201360	4.3 ± 0.9	Mexico	Landrace	LB	182000	4.7 ± 0.9	Guatemala	Landrace	WH
207420	4.3 ± 0.9	Colombia	Landrace	RD	189408	4.7 ± 0.9	Guatemala	Landrace	CR/DR/RD
209482	4.3 ± 0.9	Costa Rica	Landrace	WH	201369	4.7 ± 0.9	Mexico	Landrace	WH
304113	4.3 ± 1.0	El Salvador	Landrace	PI/LR	207207	4.7 ± 1.0	Colombia	Landrace	DB
313613	4.3 ± 0.9	Colombia	Landrace	CR	310660	4.7 ± 0.9	Guatemala	Landrace	WH
313685	4.3 ± 0.9	Ecuador	Landrace	LT	313483	4.7 ± 0.9	Mexico	Landrace	WH
313830	4.3 ± 0.9	Mexico	Landrace	BL	313486	4.7 ± 1.0	Mexico	Landrace	BL
417654	4.3 ± 0.9	Mexico	Landrace	BL	313571	4.7 ± 0.9	Colombia	Landrace	DR
194574	4.5 ± 1.0	Guatemala	Landrace	WH	313592	4.7 ± 0.9	Colombia	Landrace	LT
198037	4.5 ± 0.9	Peru	Landrace	BL	417731	4.7 ± 0.9	Mexico	Landrace	BL
201370	4.5 ± 0.9	Mexico	Landrace	CR/GE	557483	4.7 ± 0.9	Ecuador	Landrace	BL/PI
202834	4.5 ± 0.9	Mexico	Landrace	LY	197031	4.8 ± 0.9	El Salvador	Landrace	BL
207180	4.8 ± 0.9	Colombia	Landrace	LT	203921	5.2 ± 0.9	Mexico	Landrace	BL
309787	4.5 ± 0.9	Mexico	Landrace	YL	209491	4.8 ± 0.9	Costa Rica	Landrace	BL
310546	4.5 ± 0.9	Honduras	Landrace	LR/RD	290995	4.8 ± 0.9	Peru	Landrace	PI/LR
310886	4.5 ± 0.9	Nicaragua	Landrace	LB	307816	4.8 ± 0.9	El Salvador	Landrace	BL
312064	4.5 ± 0.9	Mexico	Landrace	BL	310663	4.8 ± 0.9	Guatemala	Landrace	BL
310891	4.8 ± 0.9	Nicaragua	Landrace	RD/DR	449412	5.2 ± 0.9	Mexico	Landrace	BL
313366	4.8 ± 0.9	Mexico	Landrace	CR/LT	207253	5.3 ± 0.9	Colombia	Landrace	CR/LT
313597	4.8 ± 0.9	Colombia	Landrace	RD/CR/WH	269210	5.3 ± 0.9	Peru	Landrace	LB
451921	4.8 ± 1.0	Guatemala	Landrace	BL	310850	5.3 ± 0.9	Nicaragua	Landrace	PI
189016	5.0 ± 0.9	Guatemala	Landrace	DR/BL	313270	5.3 ± 0.9	Mexico	Landrace	CR/DP
207136	5.0 ± 0.9	Colombia	Landrace	BL/RD	313639	5.3 ± 0.9	Colombia	Landrace	YL/CR/TN
207336	5.0 ± 0.9	Colombia	Landrace	LY	313847	5.3 ± 0.9	Peru	Landrace	CR/DR
209498	5.0 ± 0.9	Costa Rica	Landrace	BL	325750	5.3 ± 0.9	Mexico	Landrace	DY
310515	5.0 ± 0.9	Honduras	Landrace	RD/DR	326106	5.3 ± 0.9	Honduras	Landrace	WH
310556	5.0 ± 0.9	Honduras	Landrace	BL/DR/RD	415986	5.3 ± 0.9	Colombia	N/A	RD/CR
310842	5.0 ± 0.9	Nicaragua	Landrace	BL	152208	5.5 ± 0.9	Bolivia	Landrace	DR
311940	5.0 ± 0.9	Mexico	Landrace	BL	203924	5.5 ± 0.9	Mexico	Landrace	BL
190078	5.1 ± 0.9	Guatemala	Landrace	BL	209486	5.5 ± 0.9	Costa Rica	Landrace	CR/RD
293355	5.1 ± 0.9	Peru	Landrace	RD/DR	224728	5.5 ± 0.9	Mexico	Landrace	RD
311962	5.1 ± 0.9	Mexico	Landrace	CR/YL	263593	5.5 ± 0.9	Mexico	Landrace	CR
313440	5.1 ± 0.9	Mexico	Landrace	YL/DY	165462	5.6 ± 0.9	Mexico	Landrace	BL
533420	5.1 ± 0.9	Mexico	Landrace	BL/CR	207193	5.6 ± 1.0	Colombia	Landrace	TN
202835	5.2 ± 0.9	Mexico	Landrace	LT	312083	5.6 ± 0.9	Mexico	Landrace	CR
318703	5.6 ± 0.9	Mexico	Wild	LT	207203	6.5 ± 0.9	Colombia	Landrace	LB/BR
207300	5.2 ± 0.9	Colombia	Landrace	CR/LT	313499	6.5 ± 0.9	Mexico	Landrace	BL
311944	5.2 ± 0.9	Mexico	Landrace	BL	313609	6.5 ± 0.9	Colombia	Landrace	RD/CR
313671	5.2 ± 0.9	Ecuador	Landrace	DY	416468	6.5 ± 0.9	Mexico	Landrace	CR/WH
313693	5.2 ± 1.0	Ecuador	Landrace	YL/DY	207443	6.7 ± 0.9	Colombia	Landrace	WH
415913	5.2 ± 0.9	Ecuador	N/A	LR	304110	6.8 ± 0.9	El Salvador	Landrace	BL
313487	5.8 ± 0.9	Mexico	Landrace	BL	311999	6.8 ± 0.9	Mexico	Landrace	RD
313833	5.8 ± 0.9	Mexico	Landrace	BL	533332	6.8 ± 0.9	Mexico	Landrace	YL
313835	5.8 ± 0.9	Mexico	Landrace	LR	451906	7.0 ± 0.9	Guatemala	Landrace	CR/RD
313850	5.8 ± 0.9	Peru	Landrace	DI/LR	451917	7.0 ± 0.9	Guatemala	Landrace	BL
319595	5.8 ± 0.9	Mexico	Landrace	CR	533577	7.0 ± 0.9	Ecuador	Landrace	WH
533373	5.8 ± 0.9	Mexico	Landrace	BL	201388	7.1 ± 1.0	Mexico	Landrace	DY/BR

Table A.2. Country of origin, type, seed color and mean halo blight scores in unifoliate leaves of 281 common bean accessions from USDA-NPGS core collection evaluated under greenhouse condition at North Dakota State University, Fargo, ND (continued).

Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡	Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡
533510	5.8 ± 0.9	Mexico	Landrace	CR	307788	7.1 ± 0.9	El Salvador	Landrace	BR
165423	6.0 ± 0.9	Mexico	Landrace	RD/LR	533286	7.1 ± 0.9	Mexico	Landrace	CR/YL
309825	6.0 ± 0.9	Costa Rica	Landrace	CR/BL	203936	7.2 ± 0.9	Mexico	Landrace	BR
309830	6.0 ± 0.9	Costa Rica	Landrace	RD/LR	313658	6.3 ± 0.9	Ecuador	Landrace	LT
313837	6.0 ± 0.9	Mexico	Landrace	RD	313782	6.3 ± 0.9	Mexico	Landrace	BL
325731	6.0 ± 0.9	Mexico	Landrace	CR/LR	201387	7.3 ± 1.0	Mexico	Landrace	DY
206223	6.1 ± 0.9	Honduras	Landrace	BL/RD	US14HBR6¶	1.9 ± 0.5	UC-Davis; UI ǂ	Germplasm	PINTO
307808	6.1 ± 0.9	El Salvador	Landrace	DB/RD	PNK PANTHER§	6.5 ± 0.5	Seminis Seeds	Cultivar	LRK
269209	6.3 ± 0.9	Peru	Landrace	RD					

† Disease reaction scored from 1 to 9 where1 -3 = resistant (R), 4-6 = intermediate (I), 7-9 = susceptible (S); SE = Standard error of the mean

‡ Seed color where: BL = black; BR = Brown; CR = Cream-beige; DY = dark yellow; LRK = Light red kidney; LT = Light tan; LY = Light yellow; WH = White; TN/LT = Tan/Light tan; CR/DP = Cream-beige/Dark purple; CR/RD = Cream-beige/Red; TN/BR/CR = Tan/Brown/Cream-beige; DR/RD = Red/Dark red; CR/PU/LB = Cream-beige/Purple/Light brown; PU/RD = Purple/Red; TN/BR = Tan/Brown; DE/TN = Dark Grey/Tan; WH/BR = White/Brown; CR/WH = Cream-beige/White; CR/DB = Cream-beige/Dark brown; DR = Dark Tan; N/A = Not Available.

Table A.3. Mean halo blight scores, country of origin, type, and seed color in trifoliate leaves of 197 common bean accessions from USDA-NPGS core collection and 19 checks evaluated for GWAS under greenhouse condition at North Dakota State University, Fargo, ND.

Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡	Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡
182004	1.0 ± 0.3	Guatemala	Landrace	BR	307820	1.3 ± 0.3	El Salvador	Landrace	BL
207193	1.0 ± 0.3	Colombia	Landrace	TN	310586	1.3 ± 0.3	Honduras	Cultivated	RD/DR
293353	1.0 ± 0.3	Peru	Cultivated	DP/PU	310674	1.3 ± 0.3	Guatemala	Landrace	RD/CR
293355	1.0 ± 0.3	Peru	Cultivated	RD/DR	312083	1.3 ± 0.3	Mexico	Landrace	CR
299019	1.0 ± 0.3	Ecuador	Landrace	WH	312090	1.3 ± 0.3	Mexico	Landrace	LY/CR
309715	1.0 ± 0.3	Mexico	Landrace	DP/PI	313217	1.3 ± 0.3	Mexico	Landrace	PU/RD
310663	1.0 ± 0.3	Guatemala	Landrace	BL	313366	1.3 ± 0.3	Mexico	Landrace	CR/LT
313333	1.0 ± 0.3	Mexico	Landrace	BL	313490	1.3 ± 0.3	Mexico	Landrace	BL
313429	1.0 ± 0.3	Mexico	Landrace	CR/LT	313847	1.3 ± 0.3	Peru	Cultivated	CR/DR
313445	1.0 ± 0.3	Mexico	Landrace	BL	319592	1.3 ± 0.3	Mexico	Landrace	CR/WH
319684	1.0 ± 0.3	Mexico	Landrace	CR	319607	1.3 ± 0.3	Mexico	Landrace	CR/LB
325635	1.0 ± 0.3	Mexico	Landrace	BL	319683	1.3 ± 0.3	Mexico	Landrace	BL/RD
345581	1.0 ± 0.3	Costa Rica	Landrace	BL	325626	1.3 ± 0.3	Mexico	Landrace	BL
415936	1.0 ± 0.3	Ecuador	N/A	CR/PU	326110	1.3 ± 0.3	Honduras	Landrace	BL
415975	1.0 ± 0.3	Colombia	N/A	CR	346960	1.3 ± 0.3	Mexico	Cultivated	BL
533312	1.0 ± 0.3	Mexico	Cultivated	BL	415950	1.3 ± 0.3	Peru	N/A	LR/PI
533373	1.0 ± 0.3	Mexico	Cultivated	BL	415954	1.3 ± 0.3	Peru	Landrace	WH/CR
189408	1.3 ± 0.3	Guatemala	Landrace	CR/DR	415955	1.3 ± 0.3	Peru	Landrace	WH
201343	1.3 ± 0.3	Mexico	Landrace	TN/LT	417633	1.3 ± 0.3	Mexico	Cultivated	PI
203934	1.3 ± 0.3	Mexico	Landrace	CR/LT	417657	1.3 ± 0.3	Mexico	Cultivated	BL
207389	1.3 ± 0.3	Colombia	Landrace	CR/LR	417780	1.3 ± 0.3	Mexico	Wild	CR/TN
209482	1.3 ± 0.3	Costa Rica	Landrace	WH	533363	1.3 ± 0.3	Ecuador	Cultivated	LB/DT
304113	1.3 ± 0.3	El Salvador	Landrace	BL	196463	1.5 ± 0.3	Nicaragua	Landrace	BR
307791	1.3 ± 0.3	El Salvador	Landrace	BL	198037	1.5 ± 0.3	Peru	Landrace	BL
207127	1.5 ± 0.3	Colombia	Landrace	CR/DP	387862	1.5 ± 0.3	Bolivia	Cultivated	DY
207136	1.5 ± 0.3	Colombia	Landrace	BL/RD	387866	1.5 ± 0.3	Bolivia	Cultivated	WH/CR
207148	1.5 ± 0.3	Colombia	Landrace	LR/WH /BR	415909	1.5 ± 0.3	Ecuador	Landrace	WH
209498	1.5 ± 0.3	Costa Rica	Landrace	BL	415913	1.5 ± 0.3	Ecuador	N/A	LR
307808	1.5 ± 0.3	El Salvador	Landrace	DB/RD	417708	1.5 ± 0.3	Mexico	Cultivated	N/A
307810	1.5 ± 0.3	El Salvador	Landrace	BL	417754	1.5 ± 0.3	Mexico	Landrace	BL
308894	1.5 ± 0.3	Costa Rica	Landrace	LR/PI/ BR	476751	1.5 ± 0.3	Guatemala	Cultivated	DR/RD
309759	1.5 ± 0.3	Mexico	Landrace	LB/BR	533259	1.5 ± 0.3	Mexico	Cultivated	BL
310850	1.5 ± 0.3	Nicaragua	Cultivated	PI	533311	1.5 ± 0.3	Mexico	Cultivated	LP/PU
313270	1.5 ± 0.3	Mexico	Landrace	CR/DP	533475	1.5 ± 0.3	Mexico	Cultivated	CR/DB
313348	1.5 ± 0.3	Mexico	Landrace	BR/DT	533510	1.5 ± 0.3	Mexico	Cultivated	CR
313473	1.5 ± 0.3	Mexico	Landrace	TN/DT	150957	1.8 ± 0.3	Mexico	Landrace	BL
313486	1.5 ± 0.3	Mexico	Landrace	BL	200967	1.8 ± 0.3	Guatemala	Landrace	BL
313572	1.5 ± 0.3	Colombia	Cultivated	BL	201004	1.8 ± 0.3	Guatemala	Landrace	BL
313664	1.5 ± 0.3	Ecuador	Cultivated	DP/DR	203920	1.8 ± 0.3	Mexico	Landrace	RD
313693	1.5 ± 0.3	Ecuador	Cultivated	YL/DY	203924	1.8 ± 0.3	Mexico	Landrace	BL
313835	1.5 ± 0.3	Mexico	Cultivated	LR	209491	1.8 ± 0.3	Costa Rica	Landrace	BL
313839	1.5 ± 0.3	Mexico	Cultivated	DR/RD	288016	1.8 ± 0.3	Nicaragua	Cultivated	BL
313842	1.5 ± 0.3	Peru	Cultivated	DY	313237	1.8 ± 0.3	Mexico	Landrace	TN/BR/ CR
316030	1.5 ± 0.3	Peru	Cultivated	BL/WH	313483	1.8 ± 0.3	Mexico	Landrace	WH
318691	1.5 ± 0.3	Mexico	Wild	TN	313837	1.8 ± 0.3	Mexico	Cultivated	RD
318703	1.5 ± 0.3	Mexico	Wild	LT	417628	1.8 ± 0.3	Mexico	Cultivated	LB
319587	1.5 ± 0.3	Mexico	Landrace	BL	417641	1.8 ± 0.3	Mexico	Cultivated	DY
326106	1.5 ± 0.3	Honduras	Landrace	WH	510574	1.8 ± 0.3	Peru	Cultivated	LR/PU/ RD

Table A.3. Mean halo blight scores, country of origin, type, and seed color in trifoliate leaves of 197 common bean accessions from USDA-NPGS core collection and 19 checks evaluated for GWAS under greenhouse condition at North Dakota State University, Fargo, ND (continued).

Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡	Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡
533299	1.8 ± 0.3	Mexico	Cultivated	LB	260418	2.0 ± 0.3	Bolivia	Cultivated	LB
533502	1.8 ± 0.3	Mexico	Cultivated	PU/PI	263596	2.0 ± 0.3	Mexico	Cultivated	LB
533561	1.8 ± 0.3	Guatemala	Cultivated	LR	198026	2.3 ± 0.3	Peru	Landrace	WH
165462	2.0 ± 0.3	Mexico	Landrace	BL	207182	2.3 ± 0.3	Colombia	Landrace	DB
201388	2.0 ± 0.3	Mexico	Landrace	DY/BR	207428	2.3 ± 0.3	Colombia	Landrace	LB/BR
207216	2.0 ± 0.3	Colombia	Landrace	DR	312016	2.3 ± 0.3	Mexico	Landrace	BL
290995	2.0 ± 0.3	Peru	Landrace	PI/LR	313512	2.3 ± 0.3	Mexico	Landrace	DY
307790	2.0 ± 0.3	El Salvador	Landrace	LR	430204	2.3 ± 0.3	Mexico	Cultivated	BL
309787	2.0 ± 0.3	Mexico	Landrace	YL	203958	2.5 ± 0.3	Mexico	Landrace	BL
309810	2.0 ± 0.3	Mexico	Landrace	LY	269209	2.5 ± 0.3	Peru	Landrace	RD/DR
310515	2.0 ± 0.3	Honduras	Cultivated	RD/DR	307816	2.5 ± 0.3	El Salvador	Landrace	BL
310829	2.0 ± 0.3	Nicaragua	Cultivated	RD/DR	309698	2.5 ± 0.3	Mexico	Landrace	DT
311900	2.0 ± 0.3	Mexico	Landrace	CR	313583	2.5 ± 0.3	Colombia	Cultivated	CR/DT
311967	2.0 ± 0.3	Mexico	Landrace	BL	313701	2.5 ± 0.3	Mexico	Cultivated	BL
313394	2.0 ± 0.3	Mexico	Landrace	DE/TN	343950	2.5 ± 0.3	Guatemala	Wild	CR
313458	2.0 ± 0.3	Mexico	Landrace	BL	417630	2.5 ± 0.3	Mexico	Cultivated	CR
313535	2.0 ± 0.3	Mexico	Landrace	CR/LY	417742	2.5 ± 0.3	Mexico	Landrace	BL
316023	2.0 ± 0.3	Peru	Cultivated	DY	417778	2.5 ± 0.3	Mexico	Wild	DT
319674	2.0 ± 0.3	Mexico	Landrace	CR	533313	2.5 ± 0.3	Mexico	Cultivated	BL
325731	2.0 ± 0.3	Mexico	Cultivated	CR/LR	201480	2.8 ± 0.3	Mexico	Landrace	DB
417721	2.0 ± 0.3	Mexico	Landrace	LA	207154	2.8 ± 0.3	Colombia	Landrace	DY
430206	2.0 ± 0.3	Mexico	Cultivated	CR	313254	2.8 ± 0.3	Mexico	Landrace	BL
512003	2.0 ± 0.3	Mexico	Landrace	GE/CR	313720	2.8 ± 0.3	Mexico	Cultivated	BL
533528	2.0 ± 0.3	Mexico	Cultivated	CR/WH	417645	2.8 ± 0.3	Mexico	Cultivated	DY
194574	3.0 ± 0.3	Guatemala	Landrace	WH	313592	3.3 ± 0.3	Colombia	Cultivated	LT
201370	3.0 ± 0.3	Mexico	Landrace	CR/GE	313667	3.3 ± 0.3	Ecuador	Cultivated	CR/LT
201387	3.0 ± 0.3	Mexico	Landrace	DY	417621	3.3 ± 0.3	Mexico	Wild	BL
207186	3.0 ± 0.3	Colombia	Landrace	LG	417653	3.3 ± 0.3	Mexico	Cultivated	LB/LT/BR
241794	3.0 ± 0.3	Ecuador	Landrace	CR	417679	3.3 ± 0.3	Mexico	Cultivated	CR
309825	3.0 ± 0.3	Costa Rica	Landrace	CR/BL	430167	3.3 ± 0.3	Colombia	N/A	RD
309827	3.0 ± 0.3	Costa Rica	Landrace	CR/WH/LT	449422	3.3 ± 0.3	Mexico	Cultivated	BL
311962	3.0 ± 0.3	Mexico	Landrace	CR/YL	451885	3.3 ± 0.3	Guatemala	Cultivated	LR
313608	3.0 ± 0.3	Colombia	Cultivated	/GE	451917	3.3 ± 0.3	Guatemala	Cultivated	BL
313850	3.0 ± 0.3	Peru	Cultivated	DI/LR	533332	3.3 ± 0.3	Mexico	Cultivated	YL
319595	3.0 ± 0.3	Mexico	Landrace	CR	533428	3.3 ± 0.3	Mexico	Cultivated	DP/PU/DR
325614	3.0 ± 0.3	Mexico	Landrace	BL	533476	3.5 ± 0.3	Mexico	Cultivated	BL
325653	3.0 ± 0.3	Mexico	Landrace	CR	533577	3.5 ± 0.3	Ecuador	Cultivated	WH
417654	3.0 ± 0.3	Mexico	Cultivated	BL	309844	3.5 ± 0.3	Costa Rica	Landrace	DY
449412	3.0 ± 0.3	Mexico	Cultivated	BL	311999	3.5 ± 0.3	Mexico	Landrace	RD
189407	3.3 ± 0.3	Guatemala	Landrace	WH	313830	3.5 ± 0.3	Mexico	Cultivated	BL
201369	3.3 ± 0.3	Mexico	Landrace	WH	451906	3.3 ± 0.3	Guatemala	Cultivated	CR/RD
297295	3.3 ± 0.3	El Salvador	Cultivated	YL	208774	4.0 ± 0.3	Nicaragua	Cultivated	RD
310778	3.3 ± 0.3	Guatemala	Landrace	DY	290990	4.0 ± 0.3	Peru	Landrace	CR/RD
311794	3.3 ± 0.3	El Salvador	Landrace	DR	533286	4.0 ± 0.3	Mexico	Cultivated	CR/YL

Table A.3. Mean halo blight scores, country of origin, type, and seed color in trifoliate leaves of 197 common bean accessions from USDA-NPGS core collection and 19 checks evaluated for GWAS under greenhouse condition at North Dakota State University, Fargo, ND (continued).

Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡	Accession	Mean ± S.E.†	Country of origin	Type	Seed Color ‡
311853	3.3 ± 0.3	Guatemala	Landrace	RD	313501	4.0 ± 0.3	Mexico	Landrace	CR
312031	3.3 ± 0.3	Mexico	Landrace	BL	309700	4.0 ± 0.3	Mexico	Landrace	CR/LT
313386	3.3 ± 0.3	Mexico	Landrace	YL/LY	417731	4.0 ± 0.3	Mexico	Cultivated	BL
313459	3.3 ± 0.3	Mexico	Landrace	CR	417634	4.3 ± 0.3	Mexico	Cultivated	PI
313487	3.3 ± 0.3	Mexico	Landrace	BL	307788	5.0 ± 0.3	El Salvador	Landrace	BR
152208	3.3 ± 0.3	Bolivia	Landrace	DR	SANTA CRUZ	1.0 ± 0.3	AmeriSeed	Cultivar	PINTO¶
182000	3.3 ± 0.3	Guatemala	Landrace	WH	Sinaloa	1.0 ± 0.4	Provita	Cultivar	PINTO¶
449389	3.5 ± 0.3	Mexico	Cultivated	BL	Stampede	1.0 ± 0.4	AmeriSeed	Cultivar	PINTO¶
RED ROVER	2.7 ± 0.3	Seminis Seeds	Cultivar	DRK¶	Montcalm	2.0 ± 0.3	USDA-ARS/Michigan Ag. Expt.	Cultivar	DRK¶
Cabernet	1.3 ± 0.3	Seminis Seeds	Cultivar	DRK¶	RED HAWK	2.0 ± 0.3	USDA-ARS/Michigan Ag. Exp. Stat.	Cultivar	DRK¶
Foxfire	1.0 ± 0.3	Kelly Bean Co.	Cultivar	LRK¶	Lapaz	2.2 ± 0.3	SeedWest Inc.	Cultivar	PINTO¶
9212-4	1.1 ± 0.3	Colorado State University	Breeding Line	PINTO	Long's Peak	2.2 ± 0.3	Colorado Agricultural Experiment Station, CO, USA	Cultivar	PINTO¶
VAX3[β]	1.0 ± 0.4	CIAT	Breeding Line	Shiny Red	Windbreaker	2.3 ± 0.4	Seminis Seeds	Cultivar	PINTO¶
US14HBR6[β]	1.2 ± 0.4	UC-Davis; UI	Germplasm	PINTO¶	Croissant	2.6 ± 0.3	Colorado Agricultural Experiment Station, CO, USA	Cultivar	PINTO¶
Norstar[β]	1.2 ± 0.3	NDSU	Cultivar	NAVY¶	Majesty[β]	1.3 ± 0.3	Agri-Food Canada	Cultivar	DRK¶
Lariat[β]	1.7 ± 0.4	NDSU	Cultivar	PINTO¶	Clouseau PNK	1.8 ± 0.3	Seminis Seeds	Cultivar	LRK¶
Eclipse[β]	1.8 ± 0.3	NDSU	Cultivar	BL¶	PANTHER	3.0 ± 0.3	Seminis Seeds	Cultivar	LRK¶
CELRK[β]	2.2 ± 0.3	UC-Davis	Cultivar	LRK¶	HIME[β]	1.3 ± 0.3	H.P.T.A.E.S.	Cultivar	OTEBO¶

† Disease reaction scored from 1 to 9 where1 -3 = resistant (R), 4-6 = intermediate (I), 7-9 = susceptible (S); SE = Standard error of the mean

‡ Seed color where: BL = black; BR = Brown; CR = Cream-beige; DY = dark yellow; LRK = Light red kidney; LT = Light tan; LY = Light yellow; WH = White; TN/LT = Tan/Light tan; CR/DP = Cream-beige/Dark purple; CR/RD = Cream-beige/Red; TN/BR/CR = Tan/Brown/Cream-beige; DR/RD = Red/Dark red; CR/PU/LB = Cream-beige/Purple/Light brown; PU/RD = Purple/Red; TN/BR = Tan/Brown; DE/TN = Dark Grey/Tan; WH/BR = White/Brown; CR/WH = Cream-beige/White; CR/DB = Cream-beige/Dark brown; DR = Dark Tan; N/A = Not Available.

¶ Represents the market class of respective commercial checks used.

[β] CIAT = International Center for Tropical Agriculture, Cali Colombia; UC-DAVIS= University of California, Davis; UI = University of Idaho; NDSU = North Dakota State University; H.P.T.A.E.S. = Hokkaido Pref. Tokachi Ag. Expt. Stat., Hokkaidao, Japan

Table A.4. Plant stand, days to flowering (DTF), agronomic value, days to maturity, 100 seed weight and seed yield of 49 common bean accessions from USDA-NPGS core collection grown under field conditions.

Accession	Plant stand†	Days to flowering	Agronomic value‡	Maturity	100 seed weight (gram)	Seed yield (gram per acre)
200956	54	49	6	96	24.8	706.1
201010§	34	N/A	7	N/A	N/A	N/A
203936	41	45	7	98	27.23	457.7
207373	57	43	6.5	91	17.5	585.1
207443	44	53	7	100	21.28	571.3
209479	52	44	5.5	90	17.93	180.7
304110	53	51	6.5	98	23.5	625.8
308898	49	50	3.5	89	20.41	678.5
309810	49	66	7	97	16.38	509.4
310826	55	53	6.5	98	23.2	760.4
310829	57	50	4	87	20.715	628.6
311942	48	62	7	96	15.86	259.4
311974	56	47	7	101	15.85	397.2
311999	54	48	5.5	94	22.64	518.8
313217	38	57	7	99	22.67	514.9
313237	54	53	7	97	38.09	695.3
313254	57	51	6	98	24.01	719.8
313343	51	60	6.5	92	26.06	494.7
313394	42	54	7	97	24.72	810.5
313499	49	49	6	89	17.29	424.6
313596	59	51	7	91	23.9	705.5
313665	54	54	7	102	16.35	536.2
313782	48	48	5.5	88	19.54	565.1
319618	47	62	7	96	25.8	513.9
415949	59	54	7	99	19	512.8
451917	51	76	8	99	20.5	522.6
476751	58	47	5.5	91	23.19	833.4
533475	47	48	5.5	93	19.15	593.3
533476	51	48	5	92	19.67	508.9
533577	55	53	5.5	96	27.58	632.6
CABERNET	51	41	7	79	46.14	270.8
CELRK	30	36	7.5	74	50.15	121.3
CLOUSEAU	55	37	6	74	62.15	524.3
ECLIPSE	58	48	4	89	19.36	735.8
FOXFIRE	51	38	5.5	76	48.88	415.8
HIME	36	49	6	93	27.56	398.8

Table A.4. Plant stand, days to flowering (DTF), agronomic value, days to maturity, 100 seed weight and seed yield of 49 common bean accessions from USDA-NPGS core collection grown under field conditions (continued).

Accession	Plant stand†	Days to flowering	Agronomic value‡	Maturity	100 seed weight (gram)	Seed yield (gram per acre)
LAPAZ	55	49	4	88	35.97	568.8
LARAIT	50	47	3	87	41.37	661.4
MAJESTY	44	45	6	81	57.82	323.7
MONTCALM	48	42	4.5	85	54.57	531.4
NORSTAR	30	48	4.5	84	16.28	245.6
PINK PANTHER	32	37	6	75	54.62	239.7
RED HAWK	51	41	6.5	81	46.5	331.2
RED ROVER	57	41	6.5	84	50.14	399.2
SANTA CRUZ	57	48	4	88	35.91	579.2
SINALOA	61	49	5	88	31.7	311.9
US14HBR6	48	45	6	90	28.55	271.1
WINDBREAKER	47	47	5	87	38.19	402.4
Mean	49.5	49.0	5.9	90.0	29.8	513.2

‡ Agronomic value range from 1 to 7, where 1 = good, and 7 = worst. § Denotes accessions that did not flower. † Plant stand was counted 10 days after plant germination

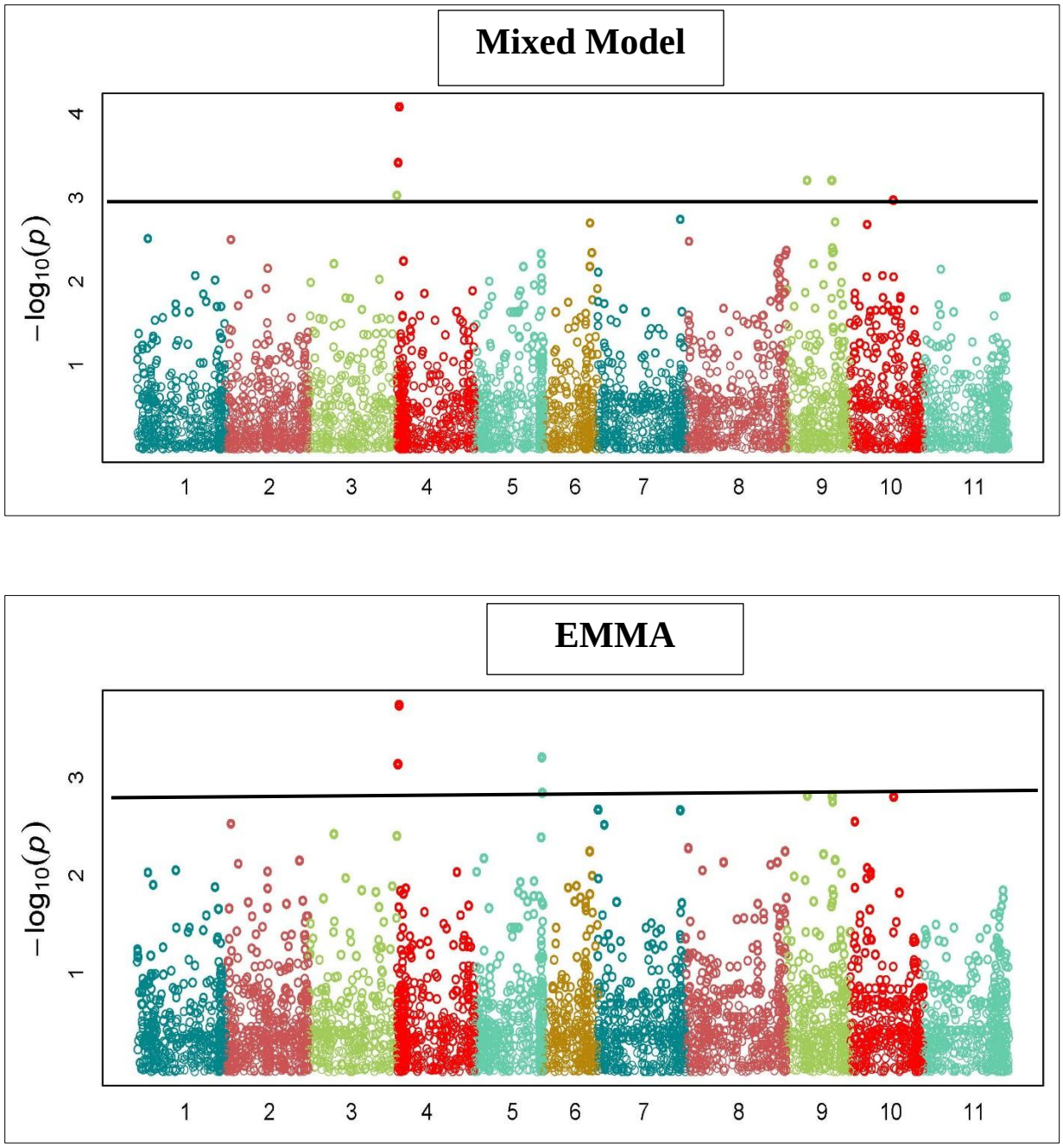

Figure A.1. Statistical models showing Manhattan plot resulting from GWAS for halo blight severity index to race 6 of *Psp*. The black horizontal line depicts the Bonferroni-adjusted significance threshold (1.1×10^{-5}).

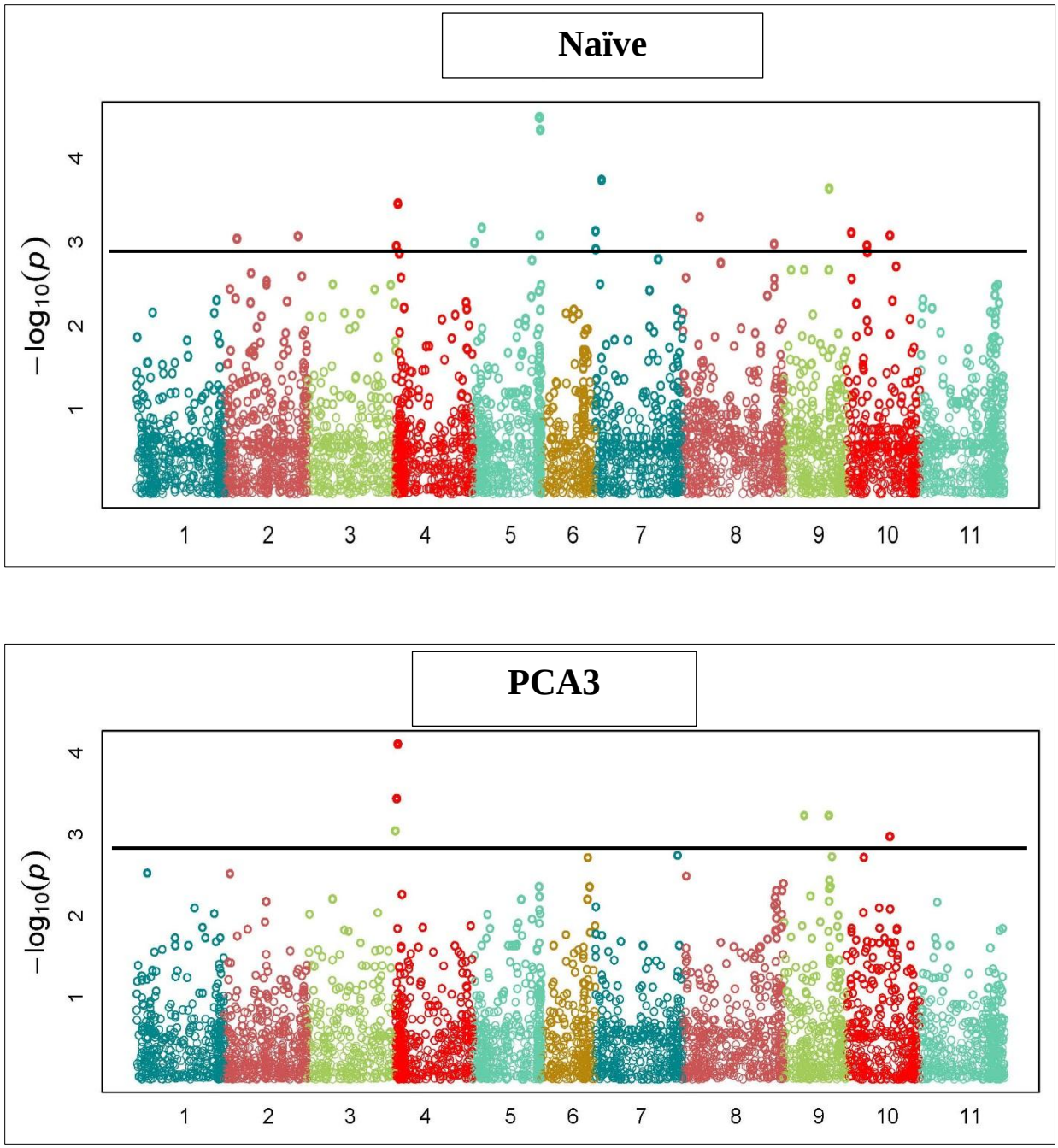

Figure A.1. Statistical models showing Manhattan plot resulting from GWAS for halo blight severity index to race 6 of *Psp*. The black horizontal line depicts the Bonferroni-adjusted significance threshold (1.1×10^{-5}) (continued).

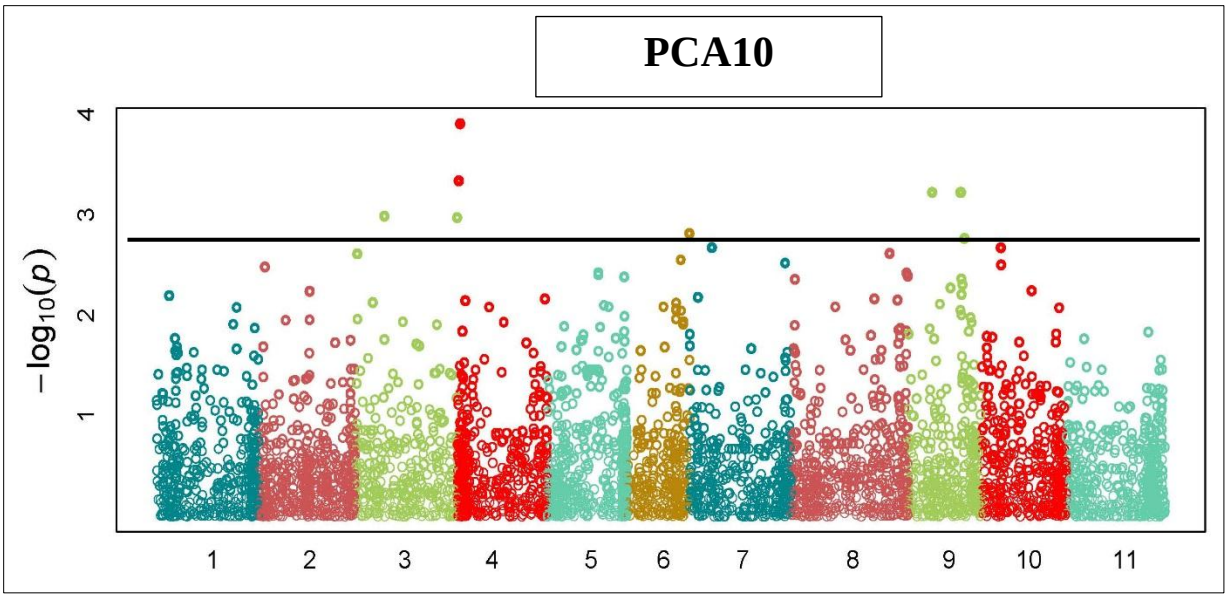

Figure A.1. Statistical models showing Manhattan plot resulting from GWAS for halo blight severity index to race 6 of *Psp*. The black horizontal line depicts the Bonferroni-adjusted significance threshold (1.1×10^{-5}) (continued).