GENOME-WIDE ASSOCIATION MAPPING AND GENOMIC PREDICTION FOR RESISTANCE TO SCLEROTINIA STEM ROT IN RAPESEED/CANOLA (*BRASSICA NAPUS* L.) GERMPLASM COLLECTIONS

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

By

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

Major Department:
Plant Sciences

October 2021

Fargo, North Dakota

North Dakota State University
Graduate School

Title

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

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ABSTRACT

Sclerotinia stem rot (SSR), caused by *Sclerotinia sclerotiorum,* is a devastating disease of rapeseed/canola that causes significant seed yield loss, reduced oil content, and quality. Lack of complete immune genotypes and polygenic resistance between host and pathogen often impedes the development of functional molecular markers and gene identification to enable SSR resistance breeding. However, genomics-assisted breeding approaches such as genome-wide association (GWA) mapping and genomic prediction (GP) are considered most promising for the genetic improvement of complex traits over classical breeding. Therefore, the objective of this study was to perform GWA mapping and GP in a diverse rapeseed/canola panel using ~24,000 to ~28,000 single nucleotide polymorphisms (SNPs) under field and greenhouse environments. Extensive phenotyping against *S. sclerotiorum* infection revealed few lines had promising resistance at seedling and adult stages in both environments. Adult plant resistance (APR) was characterized in four field environments by recording four traits and found strong associations among them. GWA models using the four traits identified 133 SNPs and 69 putative candidate genes associated with APR. The predictive ability (PA) ranged from 0.41-0.64 depending on trait specifications. For seedling resistance (SR) under a greenhouse environment, multiple GWA models using multiple traits detected 219 SNPs. Multiple GP models resulted in 0.45-0.68 PA for these traits. Association analyses for APR under controlled environments using five traits identified 37 and 50 significant SNPs in spring (SP) and semi-winter & winter populations (SWP), respectively. GP analyses revealed 0.48-0.60 and 0.10-0.19 PA in SP and SWP, respectively. Based on the GWA results collected from all experiments, we detected previously mapped overlapping genomic regions as well as new regions on chromosome A09 (33.34-39.13 Mb), C02 (59.17-62.79 Mb), and C6 (32.24-37.67 Mb). These findings would provide exciting

opportunities to narrow the genomic regions to guide map-based cloning of SSR resistance genes to assist in future marker-assisted selection. Moreover, we have achieved a medium to high PA by implementing GP. Our study concludes that GWA mapping and GP hold promise to lead a step forward towards the genomics-assisted SSR resistance rapeseed/canola breeding that would help to achieve rapid gains from the selection.

ACKNOWLEDGMENTS

I would like to express my sincere gratitude, immense indebtedness to my advisor, Dr. Mukhlesur Rahman, for his continuous support, enthusiasm, guidance, expertise, and motivation in conducting this research and writing manuscripts. He has always been such a great inspiration and motivated me in all my endeavors. This dissertation would not have been possible without his persistent help.

I also wish to extend my heartfelt gratitude to my advisory committee members, Dr. Elias M. Elias, Dr. Phillip McClean, and Dr. Luis del Río Mendoza for their wonderful valuable suggestions, encouragement, and support throughout my Ph.D. It is such an honor and privilege to be mentored by these excellent advisors, who were incredibly kind and helpful to me. Special thanks to Dr. Luis del Río Mendoza for his support and guiding me during my phenotypic data analyses.

I am also immensely grateful and thankful to Dr. Nonoy Bandillo, Dr. Jyoti Jawahar, and Dr. Muhammad Arif- Uz-Zaman (former lab member of Oilseed breeding and genetics) for their help and suggestions at various stage of my data analyses. I also would like to extend my sincere thanks and gratitude to Dr. Venkata Chapara (Langdon research extension center), Dr. Michael Wunsch (Carrington research extension center), Andrew Ross (Former research specialist, Canola breeding and genetics group), Susan Rudd (Luis's lab), and Rian Lee (McClean's lab) for their help in conducting the research in the laboratory, greenhouse, and field. I am also thankful and grateful for the immense assistance I received from my all current lab members including T M Shaikh, Shakil Hosain, Afrina Rahman, Ahasanul Hoque, and Md Zahangir Alam; former lab member Jonathan Vollmer of Oilseed (Canola and Flax) breeding and genetics group and also the summer workers.

I am also very grateful to the funding sources of this research, USDA National Institute of Food and Agriculture (Hatch Projects No. ND01506 and ND02240) and the National Sclerotinia Initiative Non-Assistance Cooperative Agreement (NACA) No. 58-3060-6-032.

Lastly and most importantly, I would like to express my heartiest gratitude to my late father and my mother for their love, persistent affection, encouragement, and blessings. It would not be possible for me to pursue my higher study without their unconditional support, guidance, and inspirations. Special thanks to my elder sister and younger brother for always being my side whenever I need them.

DEDICATION

This dissertation is dedicated

To my Parents:

Late father **Abinash Chandra Roy** & my mother **Bina Rani Roy**

To my Sister and Brother

Lata Rani Roy & **Tanmoy Roy**

To all of you who were with me during this journey!

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CHAPTER 1: GENERAL INTRODUCTION

Rapeseed/canola (*Brassica napus* L., genomes = AACC, 2n = 4x = 38), is the second largest cultivated oilseed crop in the world after soybean (USDA Foreign Agricultural Service, 2021). It is an interspecific amphidiploid *Brassica* species, derived from hybridization between diploid *B. rapa* (AA, 2n = 2x = 20) and *B. oleracea* (CC, 2n = 2x =18). The name "Canola" stands for "**Can**adian **O**il **L**ow **A**cid" and was developed by lowering the erucic acid and glucosinolate content of traditional *B. napus*. In the United States, canola production is mainly concentrated in the state of North Dakota (ND) which accounts for approximately 83% of the area planted to canola in the US. In 2020, the estimated US canola production was 15.67×10^5 metric tons, which is about 41% higher than the 11.10×10^5 metric tons produced in 2011. Nevertheless, approximately 5.67×10^5 metric tons were imported in the year 2020-21 to meet the increasing demand of canola oil (USDA, 2021). Meeting the increasing demand for canola oil will require additional acreages devoted to its production and the use of canola cultivars with improved agronomically important traits and resistance to biotic and abiotic stresses.

Rapeseed/canola is constantly affected by several biotic and abiotic stresses. Intensified cultivation of *B. napus* to meet the increasing demands for oil and other uses, has resulted in increased incidence and disease severity caused by various pathogens (Sanogo et al., 2015; Van de Wouw et al., 2016). However, the type of ailments that become important vary from one geographic region to another. In North Dakota, sclerotinia stem rot (SSR), caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, is one of the most destructive fungal diseases affecting canola production (Del Río et al., 2007). In the US, this disease causes approximately US$ 24 million in seed yield losses per year (USDA, 2016). The estimated economic loss to ND canola growers could reach US$ 17 to 21 million every year (Lamey et al., 2000, 2001). On average, every unit

increase in SSR incidence results in a 0.5-0.7% reduction in canola yield. In addition to the major seed yield loss, seeds from infected plants have reduced oil content and inferior in oil quality (McCartney et al., 1999; Sharma et al., 2015).

Proper management of *S. sclerotiorum* is a challenging, inconsistent, and uneconomical due to the wide host range of this pathogen and the long-term survival of its melanized resting reproducing structures called sclerotium. Control measures include use of cultural practices, fungicide application, and host resistance. Cultural practices include crop rotations with non-host crop species and zero tillage to reduce the sclerotial load. However, the latter is not always effective, because sclerotia can remain active in the soil for up to 8-10 years (Adams and Ayers, 1979; Hegedus and Rimmer, 2005; Sharma et al., 2015). Biological control using mycoparasitic fungi and bacteria which antagonize sclerotia can also help to reduce the sclerotial load in the soil. Recently, the use of viral particles that perturb the growth of *S. sclerotiorum* has been evaluated (Yu et al., 2013). However, the effectiveness of SSR management using bio-control agents is population-density dependent. Moreover, the efficacy of some of the commercialized products were inconsistent and limited number of grower's have adopted this technique to control *S. sclerotiorum*. Fungicides applications during the flowering stage are routinely used for the management of SSR disease. Although the application of fungicides has some positive effects on controlling SSR infection spreading, applying fungicides at the most effective time is difficult to achieve. Failure to apply fungicides at the proper time might result in an economic cost to the growers. Moreover, use of fungicides may impose negative impact on environment and increases farming costs. Breeding and cultivation of resistant cultivars is the most efficient, economic, environment-friendly, and sustainable approach to manage *S. sclerotiorum*. Breeding for SSR-resistant cultivars in rapeseed/canola, however, is often impeded by two major

problems; the unavailability of complete immune among *B. napus* germplasm and a poor understanding of the molecular mechanisms involved in the interaction between the pathogen and resistant hosts. Over three decades of investigation of SSR resistance in *B. napus* have identified few partial SSR resistant lines (Han-zhong et al., 2004; Li et al., 2009; Gyawali et al., 2016). Breeding and genetic analyses for SSR resistance were mainly depend on the utilization of these partially resistant lines. Therefore, it is necessary to explore diverse *B. napus* germplasm collections to identify highly genetically durable resistant genotypes and to identify the corresponding loci and responsible genes associated with SSR resistance through genetic mapping and genomics-assisted studies.

Genetic improvement of breeding lines for resistance to SSR is complicated by diverse factors. For example, several screening methods such as petiole inoculation technique (PIT) (Zhao et al., 2004; Bradley et al., 2006), detached leaf inoculation (Zhao and Meng, 2003; Wu et al., 2013), detached stem inoculation (Wu et al., 2013, 2016, 2019; Wei et al., 2016), and intact plant stem inoculation (Li et al., 2004, 2006; Gyawali et al., 2016; Qasim et al., 2020; Roy et al., 2021; Shahoveisi et al., 2021) have been used to evaluate the physiological resistance of rapeseed/canola germplasm at different developmental stages. Use of diverse inoculation methods, isolates of the pathogen, and phenotyping methods, results in inconsistent performance of *B. napus* lines. In other words, some *B. napus* cultivars may be identified as partially resistant in one study and susceptible in another. The identification of genetic control of SSR resistance is further complicated by the influence of physiological traits, such as flowering time, as SSR resistant quantitative trait loci (QTL) have been mapped in the same genomic regions that control flowering time (Wei et al., 2014; Wu et al., 2019; Zhang et al., 2019). Further, inconsistencies in time and type of inoculation, differences in used isolates across studies also further hamper the

identification of resistant genes against *S. sclerotiorum* (Otto-Hanson et al., 2011; Neik et al., 2017). The use of common inoculation times and methods, phenotypic evaluation of genotypes, phenotypic measurements, and *S. sclerotiorum* isolates would assist in moving this research forward to accomplish the desired goals. Standardization of these factors would lead to the identification of materials with resistant reaction across multiple environments and to the development of durable SSR resistant cultivars.

Classical genetic analyses have shown that the nature of *S. sclerotiorum* resistance in *B. napus* is quantitatively inherited, controlled by polygenes with additive effect, and displays medium to high heritability (Zhao et al., 2006; Yin et al., 2010; Wu et al., 2013; Wei et al., 2016; Roy et al., 2021). Linkage mapping studies using bi-parental populations derived from one partially resistant and one susceptible parent, have been routinely used to identify QTLs associated with SSR resistance. Under this condition, a number of QTLs have been reported on chromosomes A01, A02, A03, A06, A07, A08, A09, A10, C01, C02, C03, C04, C05, C06, C07, C08, and C09 (Zhao and Meng, 2003; Zhao et al., 2006; Yin et al., 2010; Wu et al., 2013, 2019; Wei et al., 2014; Zhang et al., 2019; Qasim et al., 2020; Shahoveisi et al., 2021). However, these QTL were specific for each study and varied with mapping populations, environments, inoculation methods, and developmental stages. In only few instances, common QTL were mapped in different mapping populations (Behla et al., 2017; Shahoveisi et al., 2021). Moreover, bi-parental linkage analyses suffer from poor mapping resolution and often results in larger confidence intervals that could harbor too many genes. This makes it difficult to identify the putative candidate genes for SSR resistance to carry out further research (Korte and Farlow, 2013).

Genome-wide association (GWA) mapping based on linkage disequilibrium (LD) has been widely used to identify the marker-trait associations (MTAs) in complex traits, as it overcomes the limitations of traditional bi-parental linkage mapping. GWA study utilizes natural populations that have undergone extensive historical recombination events, which provides promising opportunities to achieve high mapping resolution that makes possible to map the genetic markers/QTL nearer to the gene of interest (Nordborg and Weigel, 2008). Despite its wide use, few GWA studies have been performed to identify the linked markers associated with SSR resistance in rapeseed/canola (Gyawali et al., 2016; Wei et al., 2016; Wu et al., 2016; Roy et al., 2021). Among these four GWA mapping studies, Roy et al. (2021) found few overlapping quantitative trait nucleotides (QTNs) with the detected SNPs by Wei et al. (2016) and Wu et al. (2016). Results obtained from the linkage and GWA mapping studies clearly indicates that more mapping studies are necessary to validate these QTNs/QTLs and identify more significant SNPs associated with enhanced SSR resistance in rapeseed/canola.

The main limitation of association mapping is the low power for detecting rare variants that may be associated with the trait of interests (Bernardo, 2016). The majority of detected MTAs for SSR resistance from classical linkage and GWA mapping studies explained less than 12% of the phenotypic variance by each associated SNPs/QTLs, with few exceptions. This point out that a significant amount of untapped genomic potential, which need to be captured to exploit full genetic potential for SSR resistance breeding. Genomic selection (GS) has emerged as a promising genomics-assisted based approach for the genetic improvement of complex traits (Meuwissen et al., 2001). The aim of GS is to predict breeding and/or genetic values. In GS, the genomic estimated breeding values (GEBVs) are obtained by constructing the genomic prediction models, generated using phenotypic information from a genotyped population to

estimate additive effects of alleles throughout the genome. Subsequently the estimated allelic effects are used to estimate the GEBVs in a testing population that have been genotyped but not phenotyped (Meuwissen et al., 2001; Crossa et al., 2017; Derbyshire et al., 2021). Previous genomic prediction (GP) studies for various traits including seed yield and quality-related traits (Würschum et al., 2014), oil quality (Werner et al., 2018), various agronomic traits and blackleg disease resistance (Jan et al., 2016; Fikere et al., 2020) have shown the potential of GP to accelerate the rapeseed/canola breeding. Recent GP studies in rapeseed/canola for *S. sclerotiorum* resistance by Derbyshire et al. (2021) and Roy et al. (2021) achieved medium to high predictive ability, clearly indicating the potential of GS for improving SSR resistance. This further motivated us to implement and explore its effectiveness in predicting SSR resistant genotypes. The implementation of genomics-assisted breeding approaches such as GWA mapping and GP may be especially useful in reducing the required breeding time to incorporate resistance to SSR compared with classical breeding and to achieve rapid gains from selection.

The objectives of this study were:

1. To study the differential phenotypic responses of spring, semi-winter, and winter ecotypes rapeseed/canola germplasm collections against *Sclerotinia sclerotiorum* infection at seedling and adult plant growth stages under greenhouse and field environments,
2. To identify genotypes resistant to sclerotinia stem rot disease at different growth stages using (a) petiole inoculation technique, and (b) mycelial agar plug intact stem inoculation method,
3. To identify significant markers and genomic regions associated with multiple sclerotinia stem rot resistance traits at different developmental stages in both the

greenhouse and field environments through genome-wide association mapping approach,

4. To identify putative disease resistance candidate genes, close to the physical location of the identified significant molecular markers or genomic regions associated with sclerotinia stem rot resistance,
5. To explore and evaluate the effectiveness of genomic prediction for the selection of genotypes conferring seedling and adult plant stage sclerotinia stem rot resistance in rapeseed/canola under greenhouse and field environments.

CHAPTER 2: LITERATURE REVIEW

2.1. Rapeseed/canola

2.1.1. *Brassica* species

The genus *Brassica* belongs to the Brassicaceae family and is the most economically important genus, containing 37 different species (Gómez-Campo and Prakash, 1999). Interspecific hybridization and development of stable hybrids has led to the creation of new and diverse *Brassica* species. Some of the *Brassica* species are *Brassica nigra, B. napus, B. rapa, B. oleracea, B. juncea, B. carinata*. They are flowering plants widely used in human diet as oilseed, meal, vegetables, condiments and pickles. They are also used as a supplement in animal feed rations. Biofuel production is relative a new use for some of the brassica species in different regions of the world (Abbadi and Leckband, 2011).

2.1.2. *Brassica* triangle of "U"

Six oil producing species of *Brassica* genus are cytogenetically interrelated with each other. The natural process of interspecific hybridization of three diploid species led to the evolvement of three amphidiploid *Brassica* species. The Korean-Japanese botanist Nagaharu U first described the interrelationship between these amphidiploids and his illustration is known as the "triangle of U" (Nagaharu, 1935). According to this theory, three diploid species such as *B. rapa/campestris* (AA, 2n=20), *B. nigra* (BB, 2n=16), and *B. oleracea* (CC, 2n=18), lead to the development of three new allotetraploid species by natural hybridization. The allotetraploid species *B. juncea* (AABB, 2n=36) originated from the hybridization between *B. rapa* and *B. nigra; B. napus* (AACC, 2n=38) originated from hybridization between *B. rapa* and *B. oleracea;* and *B. carinata* (BBCC, 2n=34) from the hybridization between *B. nigra* and *B. oleracea* (Figure 2.1).

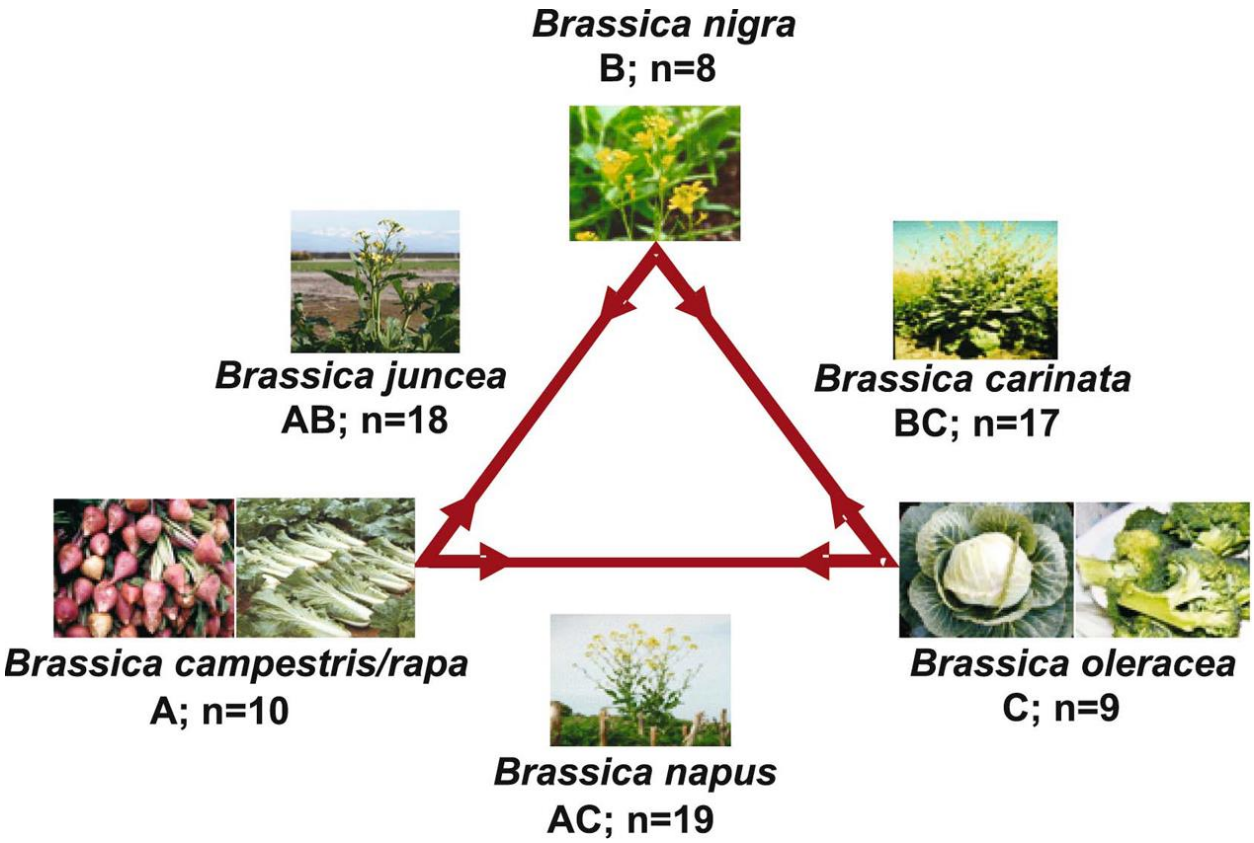

Figure 2.1. The U-triangle showing the genetic relationship among six *Brassica* species according to Nagaharu U (1935). [Figure source: Adapted from Purty et al. 2008]

2.1.3. *Brassica napus* and rapeseed

Brassica napus is an amphidiploid allotetraploid species containing A and C genomes. It is commonly known as rapeseed, swede rape, argentine rape, oil rape, and oilseed rape, among others. The term "Rape" was derived from the Latin word "Rapum" which means "Turnip" in reference to a close relative of rapeseed. Seeds from traditional rapeseed varieties, whether from *B. napus* or *B. rapa*, usually contain 22-60% of erucic acid and less than 100 µmol glucosinolates per g of air-dried oil-free meal. Oil containing high erucic fatty acid is considered undesirable for human nutrition, while high glucosinolate contents in the seed meal lead to inadequate palatability, and are generally considered nutritionally undesirable as it limits the use of this protein-rich meal in animal feed (Clandinin and Robblee, 1981; Bell, 1993).

2.1.4. Canola

The term “Canola” was derived from the description ***Can***adian ***o***il ***l***ow ***a***cid and referred to the low erucic acid rapeseed cultivars produced from *B. napus*, *B. rapa* and more recently from *B. juncea.* To use the name canola, products must need to meet internationally regulated standard, seeds of the genus *Brassica* including three species i.e. *B. napus*, *B. rapa*, *B. juncea* from which oil must contain less than 2% erucic acid in its fatty acid profile and the solid components must contain less than 30 µmol g^{-1} of glucosinolates in their seed meal. Canola oil is very popular for its nutritional qualities. Canola oil contains “good for health” Omega-6 (Alpha-linolenic acid) and Omega-3 fatty acid in a ratio of 2:1, which is considered as ideal in nutrition. These unsaturated fatty acids reduce the bad cholesterol in human body and lower the risk of heart diseases. Moreover, canola contains lowest “bad for health” saturated fatty acid among all the vegetable oils and no trans fats at all (Canola Council of Canada, 2021; Available on: https://www.canolacouncil.org/about-canola/oil/#health-benefits). In 1985, United States Food and Drug Administration certified canola oil “generally recognized as safe” (GRAS).

2.1.5. History of canola

During World War II, rapeseed oil was mainly used as a high temperature lubricant in steam ships. Due to its high content of erucic acid (20-60%), demands of oil for human consumption were very negligible. At the end of the war, the dramatic reduction in the number of steam ships led to the decline of the industrial demand for rapeseed oil. Therefore, scientists initiated an intensive program to breed rapeseed for human consumption. In 1959, a *B. napus* rapeseed line namely “Liho” containing lower levels of erucic acid was identified in nature (Eskin and Przybylski, 2003), which created the possibility of developing rapeseed lines with low erucic acid in its oil. Through repeated backcrossing and selection, low erucic acid traits

were transferred into agronomically adapted cultivars. Their continued effort led to release of the first low-erucic-acid cultivar of *B. napus*, "Oro", in 1968 and the first low-erucic-acid *B. rapa* cultivar, "Span", in 1971 (Eskin and Przybylski, 2003). The only source of low glucosinolates was identified in a Polish line "Bronowski" in late 1967, which directed into a new scope of reducing harmful glucosinolates in the rapeseed meal. After years of continued research, Dr. Stefansson developed the double low *B. napus* cultivar "Tower" in 1974 at the University of Manitoba (Stefansson and Downey, 1995). The newly developed cultivar "Tower" had less than 1% erucic acid in oil and less than 30 μmol g^{-1} aliphatic glucosinolates in air-dried oil-free meal.

2.1.6. Diseases in rapeseed/canola

The incidence and severity of disease caused by various pathogen has increased due to the intensified cultivation of *B. napus* in many countries, to meet the growing demands for oil (Sanogo et al., 2015; Van de Wouw et al., 2016). Rapeseed/canola plant could be affected by major diseases and key pathogens that cause serious damage worldwide include sclerotinia stem rot (*Sclerotinia sclerotiorum*), blackleg (*Leptosphaeria maculans*), clubroot (*Plasmodiophora brassicae*), white rust (*Albugo candida*), light leaf spot (*Pyrenopeziza brassicae*), alternaria blight (*Alternaria brassicae, A. brassicicola*, other *Alternaria spp*.) and white leaf spot (*Pseudocercosporella capsellae*); downy mildew (*Hyaloperonospora parasitica*), and blackrot pathogen (*Xanthomonas campestris* pv. *campestris*).

In North Dakota, there are two major fungal diseases affecting canola production are sclerotinia stem rot (SSR) and blackleg (Del Río et al., 2007). SSR disease is commonly more prevalent and severe in the northcentral and northeastern regions of ND. However, SSR incidence and intensity could vary depending on environmental conditions.

2.2. Sclerotinia stem rot disease in rapeseed/canola

2.2.1. The pathogen: Sclerotinia sclerotiorum

Sclerotinia sclerotiorum is a cosmopolitan, host-nonspecific, soil-borne, ascomycetous fungal plant pathogens. This pathogen attacks a broad range of host including at least 408 described species of plant from 278 genera in 75 families primarily dicotyledonous and a few monocotyledonous species (Purdy, 1979; Boland and Hall, 1994). The diseases caused *by S. sclerotiorum* in agriculture are known to have more than 60 names. For example, *S. sclerotiorum* causes sclerotinia stem rot (SSR) in rapeseed/canola and soybean (*Glycine max*), white mold in dry bean (*Phaseolus vulgaris*), chickpea (*Cicer arietinum*), lentil (*Lens culinaris*), and pea (*Pisum sativum*); *Sclerotinia* wilt, stalk rot, and head rot in sunflower (Purdy, 1979).

S. sclerotiorum (Lib.) de Bary belongs to the family Sclerotiniaceae of the order Helotiales in the phylum Ascomycota (Bolton et al., 2006). All species in the Sclerotiniaceae family produce asci on brownish apothecia that arise from a sclerotial stroma within or associated with a host plant (Whetzel, 1945; Holst-Jensen et al., 1997). *S. sclerotiorum* hyphae are multinucleate, septate, hyaline, branched and white to tan color in culture (Bolton et al., 2006).

Oxalic acid production is an important pathogenicity factor for *Sclerotinia sclerotiorum*. Oxalic acid accumulation is much higher in wilted plants than healthy plants as host colonization advances in the wilted leaves (Bateman and Beer, 1965). Noyes and Hancock (1981) observed oxalic acid accumulation in sunflower plants and they revealed that the level of oxalic acid was 15 times higher in wilted leaves than the healthy leaves.

2.2.2. Economic importance of sclerotinia stem rot disease

Sclerotinia stem rot (SSR) disease causes significant yield losses in rapeseed/canola by affecting plant structural integrity, and causing loss of photosynthetic area, wilting by blocking the transportation of water and nutrients to the developing seeds, and causing premature death of the plant. Yield losses may be 10-30% but can reach up to 80% in severely infected fields (Wu et al., 2016). In severe cases, SSR incidence can be reached up to 92% (Sharma et al., 2015). Del Río et al. (2007) estimated that on average for each 1% infected plants, there is 0.5 to 0.7% yield reduction. They also reported that economic losses caused by 17% disease incidence had the same cost of fungicide application. Therefore, 17% disease incidence should be considered as economic threshold.

According to USDA (2016), the annual economic loss of soybean, sunflowers, dry edible beans, canola, and pulses due to sclerotinia damage has been estimated at US$ 482 million, with estimated losses for canola accounting for US$ 24 million. This huge economic loss led to the development of the USDA-ARS National Sclerotinia Initiative, a program whose objectives is to reduce losses due to this disease in several crops. In North Dakota and Minnesota, average yield losses have been estimated at 13%, however, the loss can reach up to 50% in some locations. The canola growers in Minnesota and North Dakota have seen disease caused economic losses of 17.3, 20.8, and 16.8 million dollars in 1999, 2000, and 2001, respectively (Lamey et al., 2000, 2001).

The disease not only reduces seed yield, but also affects seed quality. Seeds from infected plants have reduced oil content, and contain oil of inferior quality which could threaten human health (McCartney et al., 1999; Pressete et al., 2019).

2.2.3. The sclerotinia stem rot disease symptomatology

Sclerotinia stem rot develops on rapeseed/canola plants during flowering. Spores infect flower petals and when petals are casted, it moves from there to leaves or stems. The first visible symptoms on leaves and stems appear as a mushy, light brown color lesions on and around the cast petals. Infected senescent petals are the predominant source of infection which helps to spread the infection into the adjacent plants leaves, branches and stems (Thatcher et al., 2017). On young stems, lesions appear as water-soaked which progress in all directions and causes the stem girdling which results stem to wilt and premature ripening. On leaves, irregular water-soaked lesions appear which later turn into bright like lesions (Bardin and Huang, 2001).

Infected stems become bleached, taking on a whitish appearance. On severely affected plants, they become brittle and their epidermis looks shredded. Infected plants may die early and are prone to lodging. As the disease progresses, black sclerotia can be produced inside or on the surface of the infected stems (Bardin and Huang, 2001).

2.2.4. The sclerotia

A densely mass of white mycelia known as sclerotia is the primary survival structure of *S. sclerotiorum*. It acts as a vegetative reproductive and long-term survival structures for the fungus. The pathogen can be viable in soil in the form of sclerotia for 8 to 10 years (Adams and Ayers, 1979; Hegedus and Rimmer, 2005).

A sclerotium consists of two parts, the medulla and the outer rind. The former is light colored, while the outer is black and composed of several layers of highly melanized cells. The medulla contains β-glucans, and proteins (Tourneau, 1979), whereas rind contains melanin, a compound that plays an important role in protection from adverse condition and make the sclerotia highly resistant to degradation (Bell and Wheeler, 1986; Henson et al., 1999). The size

and shape of sclerotia of *S. sclerotiorum* varies with the host. The disease cycle begins after the germination of overwintered sclerotia in soil.

Survival of sclerotia in the field depends on a number of factors. High soil temperature, high soil moisture, and reduced oxygen level in irrigated fields have an adverse effect on sclerotia survival (Wu et al., 2008). Adams and Ayers (1979) reported that soil temperatures between 10-30℃ have no adverse effect on the survival of sclerotia, but temperatures of 35℃ for 3 weeks or more cause reduced sclerotial survival. Microbial degradation is the most significant factor adversely affecting sclerotia survival in soil (Adams and Ayers, 1979). Many fungi, bacteria and other soil organisms parasitize sclerotia and use them as their carbon source including *Coniothyrium minitans, Sporidesmium sclerotivorum, Fusarium* spp., *Trichoderma* spp. *Penicillium* spp, *Aspergillus* spp., *Stachybotrys* spp. and *Verticillium* spp. (Adams and Ayers, 1979).

2.2.5. Disease cycle

The disease cycle starts with germination of sclerotia. Continuous soil moisture for a minimum period of ten days, accompanied by 60- 77° F (15-25 °C) soil temperatures are required. Depending on weather conditions, sclerotia can germinate myceliogenically or carpogenically. Myceliogenic germination results in the production of hyphae that can directly infect plant tissues (Tourneau, 1979; Bardin and Huang, 2001). On the other hand, in carpogenic germination the sclerotia germinate to produce apothecia (sexual fruiting bodies). Apothecia is capable of producing millions of ascospores (sexual spores) that are released into moving air currents. Ascospores normally cannot infect the leaves and stems directly, as they require an exogenous source of nutrients, like petals, and degrading tissues to infect above ground parts of the host (Jamaux et al., 1995; Bardin and Huang, 2001). The fungus colonizes infected petals and

eventually penetrate other host tissue. Conducive environments such as continuous moisture (free water) at 21°C for 48 h is necessary for the ascospores to germinate and move from petals to leaves for establishing the pathogen infection (Shahoveisi and del Río Mendoza, 2020). The infection process begins when ascospores land on flower petals and subsequently germinate and colonize the tissue (Turkington, 1993). Then, subsequently the fungus grows from the flower petals to leaves, leaf axils, branches, and stems. Once the *S. sclerotiorum* penetrates the plant, it will colonize and feed on the plant tissues as a source of their nutrient. Sclerotia will be formed inside or on the stem once the nutritional value of infected plant tissues has been exhausted or if environmental conditions become unfavorable for the pathogen (Figure 2.2).

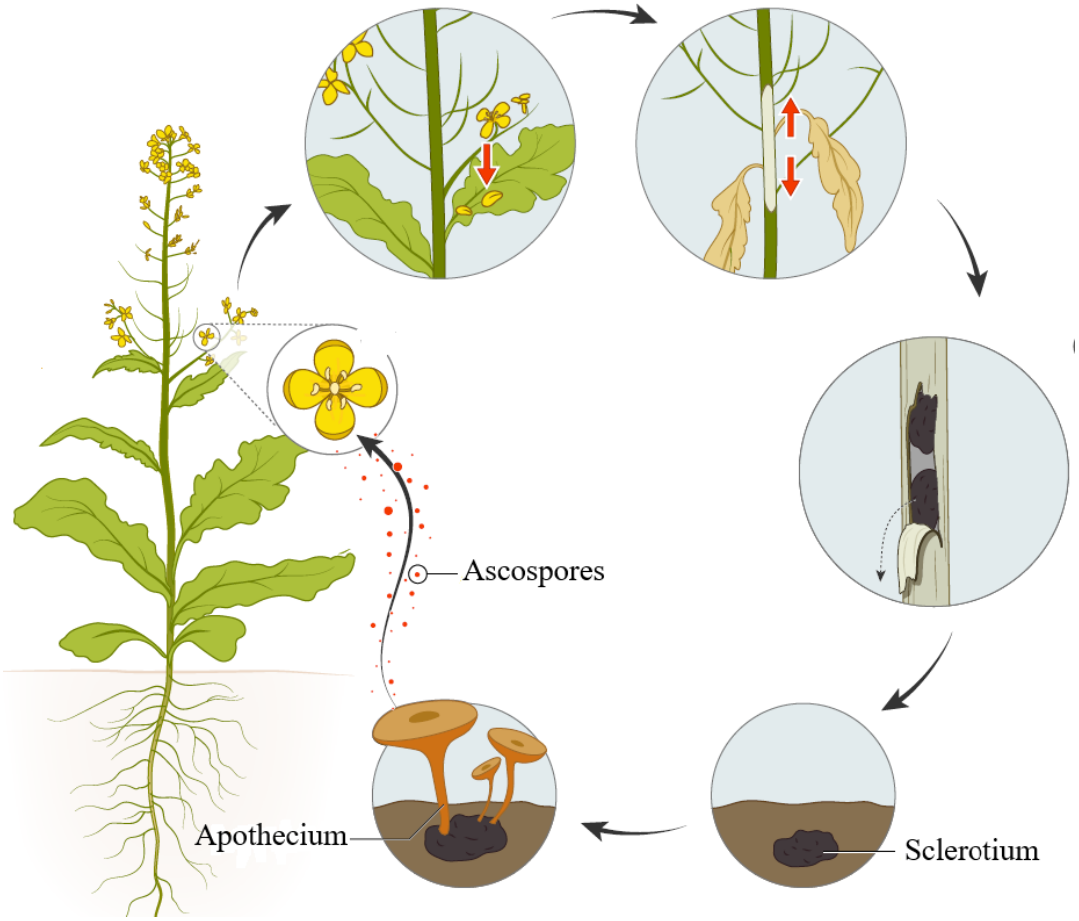

Figure 2.2. Disease cycle of Sclerotinia stem rot caused by *Sclerotinia sclerotiorum* (photo credits: https://www.canolacouncil.org/download/157/canola-encyclopedia/18611/sclerotinia-updated-final-2).

2.3. Breeding for durable resistance to sclerotinia stem rot in rapeseed/canola

2.3.1. Host resistance

Genetic resistance offers the most economically feasible, environment friendly and sustainable approach to control sclerotinia stem rot (SSR) diseases in rapeseed/canola. Therefore, development of SSR resistant varieties is the priority of breeding programs to manage this disease and to make canola production profitable to the growers. Although no highly resistant or immune accessions have been found in rapeseed germplasm resources, there is wide variation in resistance among varieties. In recent years, some progress has been made in the identification and breeding for varieties resistant to *S. sclerotiorum* infection. Partially SSR-resistant lines such as 'Zhongyou 821', 'Zhongshuang', and 'Ning RS-1' were developed (Han-zhong et al., 2004; Li et al., 2009). In North America, commercial spring hybrid canola cultivars such as Pioneer 45S51, Pioneer 45S52, Pioneer 45S56, with enhanced resistance to SSR disease were developed. Several efforts were also made to search for resistance sources in close relatives of *B. napus*. Among these, *B. oleracea* has the greatest resistance and *B. rapa* has the lowest, while *B. juncea*, *B. napus*, and *B. nigra* are intermediate (Ding et al., 2021). Screening for resistant sources is the basis of resistance breeding in crop plants. Even though some of the partial SSR resistant line have been identified, the quest for complete host resistance still encourage the plant breeders and pathologists to explore diverse germplasm collections to identify resistant genotypes.

2.3.2. Disease screening methods and phenotypic measurements

A number of methods have been used with varying results and occasional correlation with field screening. Many of the implemented methods also showed varying results across studies, populations and crops. However, there is no single, widely accepted method for

evaluating resistance in rapeseed/canola, which creates difficulty in identifying resistance source across different study and populations.

Various methods have been used to screen the germplasm accessions and to evaluate resistance to sclerotinia stem rot disease in various crops. In some cases, same screening methods were used in different crops. For example, ascospore suspension spray was used for snap bean (Abawi et al., 1978), soybean (Cline, 1982) and rapeseed (Bailey, 1987); petiole inoculation technique (PIT) was used in soybean (Hoffman et al., 2002) and rapeseed (Zhao et al., 2004; Bradley et al., 2006); stem inoculation was used in soybean (Wegulo et al., 1998), sunflower (Vuong et al., 2008) and rapeseed (Gyawali et al., 2016; Qasim et al., 2020; Roy et al., 2021; Shahoveisi et al., 2021); oxalic acid assay in soybean (Wegulo et al., 1998) and canola (Bradley et al., 2006) were used to evaluate resistance to sclerotinia stem rot with or without modifications.

Screening of resistance to *S. sclerotiorum* can be broadly divided into two categories (a) field evaluation, and (b) controlled environment evaluation. Field evaluations can be performed by growing plants on naturally infested fields (Kim and Diers, 2000; Bradley et al., 2006) or by artificially inoculating plants in the field (Bradley et al., 2006; Yin et al., 2010). Screening in controlled environments provide better evaluation of physiological resistance. In general, evaluations in controlled environments are easier and quicker to do, and they can be performed anytime during the year.

Zhao et al. (2004) screened 47 *B. napus* accessions to evaluate resistance to sclerotinia stem rot using petiole inoculation technique (PIT) in greenhouse. Petioles of the third fully expanded leaves of four weeks old plants were severed 2.5 cm from the main stem using a razor blade and loaded tips were placed into the severed petioles. Days to wilt (DW) were recorded

daily for 7 to 11 days post inoculation (dpi). In addition to DW, they also recorded phenotypic responses on a scale of 0 to 4, where 0 means no symptoms and four means expanded, sunken, water-soaked lesions resulting in wilt of the foliage. Phenotypic responses of accessions were consistent and high correlations were found between DW and lesion phenotypes. Bradley et al. (2006) compared the response of canola cultivars to SSR using oxalic acid assay, petiole inoculation technique, and detached leaf assay in both controlled environments and field conditions. Significant differences were observed for SSR and yields among cultivars in the controlled environment for all three techniques. However, in the field environments significant differences were recorded for the oxalic acid and petiole inoculation method but not for the detached leaf assay method. PIT was also used successfully by Zhao et al. (2006) and Behla et al. (2017) to identify QTL for resistance to SSR using DH populations in *B. napus*.

Cotyledon assay for SSR resistance evaluation was also used by Garg et al. (2008) by inoculating 10 days old cotyledon with mycelial suspension. Phenotypic differences were collected by measuring lesions diameter at 4 dpi. Significant differences among the genotypes were observed on the collected phenotypic data.

Stem inoculation method either in the form of detached stem assay (Wu et al., 2013, 2016, 2019; Wei et al., 2016) or intact plant stem assay (Li et al., 2004, 2006; Gyawali et al., 2016; Qasim et al., 2020; Roy et al., 2021; Shahoveisi et al., 2021) were implemented to evaluate the phenotypic performance of rapeseed/canola genotypes against *S. sclerotiorum* attack. Stem inoculation methods are usually applied when the plants are at full flowering stage, because this is the most prevalent stage for SSR infection. This method mimics natural SSR infection and has successfully been used by many researchers to evaluate the resistance performance of *B. napus* genotypes under field (Li et al., 2006; Qasim et al., 2020; Roy et al., 2021) and greenhouse

environment (Li et al., 2004; Gyawali et al., 2016; Shahoveisi et al., 2021). Various mapping studies such as bi-parental linkage (Wu et al., 2013, 2019; Qasim et al., 2020; Shahoveisi et al., 2021) and genome wide association (GWA) (Gyawali et al., 2016; Wei et al., 2016; Wu et al., 2016; Roy et al., 2021) mapping were performed to identify the marker-trait associations (MTAs) for SSR resistance in rapeseed/canola. Stem lesion length measured at 3-9 dpi were most commonly used phenotypic trait in this inoculation method to differentiate the phenotypic response of resistant and susceptible genotypes.

Besides the above-mentioned disease screening techniques, oxalic acid assay technique was also used as screening methods to evaluate SSR resistance in *B. napus*. Some methods such as the detached leaf assay, the cotyledon leaf assay and the petiole inoculation technique are suitable for large scale SSR phenotyping, while other techniques require more resources. Therefore, it is necessary to identify the resistance with differential methods over a longer period and improve the accuracy of the methods with evaluating multiple phenotypic traits. Studies are needed to further develop a universally accepted screening method which is simple, reliable and positively correlated with field screening.

2.3.3. Molecular markers and quantitative trait loci (QTL) mapping

Genetic improvement of crops resistance to biotic and abiotic stresses requires identification of useful sources of the trait variations and subsequently their introgression into the adapted elite cultivars. Significant crop improvements in terms of yield, biotic and abiotic stresses have been made through the implementation of conventional breeding and genetics. However, the required time and progress in genetic gains through classical breeding approaches alone is slow, particularly in targeting traits with complex inheritance that are influenced by pathogen population diversity, and changing climates (Araus et al., 2008; Cooper et al., 2009). In

recent years molecular marker technology has offered great potential for plant breeding by providing solutions to overcome some of the limitations faced by classical breeding. Recent advances in molecular marker technologies have allowed researchers to explore the potential of improving varieties by examining the genetic makeup of a particular genotype. Molecular markers linked with the trait of interest can facilitate pyramiding of target traits into adapted cultivars with greater precision, reduced loss of genetic gain and shorter breeding cycles (Xu and Crouch, 2008). Genetic mapping of trait of interest, plant disease diagnostics, and assessment of genetic diversity were also done using molecular markers. With the availability of more marker data and advances in statistical modeling, genomic selection approaches have also been introduced to characterize phenotypic performance of individuals based on genomic estimated breeding values (Meuwissen et al., 2001; Nakaya and Isobe, 2012).

Different types of DNA-based molecular markers have been developed. DNA molecular markers can be divided into hybridization-based markers, PCR-based markers and sequenced based markers. Hybridization based Restriction Fragment Length Polymorphisms (RFLP) was the first DNA based molecular marker used for genotyping and creating genetic linkage map (Botstein et al., 1980). Later, several PCR-based molecular markers, Random Amplified Polymorphic DNA (RAPD) (Williams et al., 1990), Simple Sequence Repeats (SSR) (Hearne et al., 1992), sequence characterized amplified region (SCAR) (Paran and Michelmore, 1993), cleaved amplified polymorphic sequences (CAPS) (Konieczny and Ausubel, 1993), Sequence Tagged Sites (STS) (Fukuoka et al., 1994), Amplified Fragment Length Polymorphism (AFLP) (Vos et al., 1995), and Sequence Related Amplified Polymorphism (SRAP) (Li and Quiros, 2001) were developed. However, genotyping of above-mentioned markers is performed for

individual markers, and are restricted in addressing high-throughput required for genotyping large number of individuals in a large population.

Single nucleotide polymorphism (SNP) results from single base changes in the DNA sequence and are the most abundant source of variation in plant and animal genome (Ganal et al., 2011). Among all the molecular markers, SNPs are now the most popular due to its high abundance in the genome and low-cost discovery (He et al., 2014). More recently, the adoption of microarrays/chips and next generation sequencing technologies for SNP detection and validation has revolutionized the development of new marker systems. This high throughput-genotyping platform are cost effective and saving time for routine use in most plant molecular research (Gupta et al., 2010). The subsequent shift to SNP markers and its rapid advancement has made excellent progress to characterize genetic diversity of major crop species, to map QTL for trait of interest, and to clone genes important for crop improvement.

2.3.4. Linkage based QTL mapping for sclerotinia stem rot resistance in rapeseed/canola

Linkage-based QTL mapping has been a key approach for genetic dissection of complex traits of agriculturally important traits in crops (Holland, 2007). In this method, mapping populations were developed through the hybridization of two parents with contrasting phenotypes i.e. one parent disease resistant and second parent is disease susceptible. Bi-parental segregating populations such as F_2, recombinant inbred lines (RIL), double haploid (DH), near-isogenic (NIL), and backcross (BC) lines could be used in QTL mapping. Genetic linkage maps were constructed based on the recombination occurred in the two parents during meiosis (Mammadov et al., 2012). It is particularly useful in tagging QTL of rare variants and small-effect alleles. However, bi-parental linkage mapping suffers from two major limitations. One major limitation is the restricted number of recombination events that occurred during the

development of bi-parental mapping population. These results in low mapping resolution, often in the range of 10-30 cM genomic interval (Korte and Farlow, 2013). The second major limitation is the low allelic diversity since only two alleles descended from two parents are used (Korte and Farlow, 2013).

QTL mapping using bi-parental mapping populations were extensively used for the identification of genomic loci associated to SSR resistance in *B. napus* (Zhao and Meng, 2003; Zhao et al., 2006; Yin et al., 2010; Wu et al., 2013, 2019; Wei et al., 2014; Zhang et al., 2019; Qasim et al., 2020; Shahoveisi et al., 2021). These studies were performed using different inoculation procedures, different plant developmental stages such as seeding and adult (Zhao and Meng, 2003; Wu et al., 2013). QTLs identified from these studies were specific to different growth stages and different inoculation methods in the rapeseed/canola genotypes. Zhao et al. (2006) used two double haploid (DH) populations (HUA and MS) to identify QTL associated to sclerotinia stem rot disease using petiole inoculation technique. Using two scoring criteria, days to wilt and stem lesion length at 4dpi, they detected eight (HUA) and one (MS) QTL and found that none of the QTL overlapped between the two studied populations. Yin et al. (2010) studied DH populations to identify QTL linked with SSR resistance using three different inoculation techniques on mature *B. napus* plants. One to 10 QTLs were mapped depending on the inoculation technique, and the explained phenotypic variation by each QTL varied between 10.2 to 36.1%. Another study by Wei et al. (2014) identified 5 and 6 QTLs under controlled and field environments, where inoculation was done at the flowering stage and identified three flowering time (FT) QTLs those were co-localized with the SSR resistance QTL. Similar trends were also observed by Wu et al. (2019), and Zhang et al. (2019). Wu et al. (2019) mapped four co-localized QTL hotspots for SSR resistance and FT. Similarly, Zhang et al. (2019) also detected

few co-localized FT QTL with SSR resistance. Both of studies results revealed a genetic link between SSR resistance and FT in *B. napus*. Similar finding was also reported by Qasim et al. (2020) who indicated that SSR resistance was negatively correlated with flowering time. They detected17 QTL associated with SSR resistance on chromosomes A02, A09, C02, C03, C04, and C06 by evaluating 181 DH for three years in field environments. The observed phenotypic variance by these QTL varied between 5.94 to 14.75%. Shahoveisi et al. (2021) evaluated two DH populations under greenhouse environments and used multiple phenotypic scoring system for QTL identification. Sixteen QTLs were detected and 12 QTL were located on chromosomes A02, and C01. Moreover, they also detected a QTL co-localized in both populations. Despite these efforts, no fine mapping or map-based cloning of the SSR resistant QTL or gene has been reported so far. This ultimately limits the utilization of the identified QTL to implement in the MAS SSR breeding. The potential reasons behind this would be highly polygenic nature of SSR resistance making difficult in pyramiding small-effect SSR resistant loci and lack of identification of common QTLs across different studies, mapping populations, different growth stages of SSR phenotyping, and different inoculation methods. Only few instances, common markers have been detected in different mapping populations (Shahoveisi et al., 2021). Therefore, more mapping studies are needed to validate these identified QTL, which will provide more confidence and could possible narrowed down the associated genomic regions.

2.3.5. Genome-wide association mapping

Identification of molecular markers and QTL associated with a trait of interest is the main prerequisite for successful implementation of MAS (Collard et al., 2005). Genome-wide association mapping is another approach, which is widely used for the genetic dissection of economically important complex traits. It is an approach that utilizes natural populations,

historical recombination and natural genetic variation within the ex situ conserved genetic resources. Association mapping identifies significant marker-trait associations by exploiting linkage disequilibrium (LD) created through ancestral and evolutionary recombination events at the population level. LD-based association studies enable the most effective utilization of conserved genetic diversity among globally distributed germplasm resources (Abdurakhmonov and Abdukarimov, 2008). Moreover, unlike linkage mapping, association mapping also leads to a higher resolution mapping because of the increased number recombination events from a large number of meiosis that occurred throughout generations that makes it possible to map the QTL near to the gene of interest (Nordborg and Weigel, 2008). It is a timesaving and cost-effective approach compared to bi-parental populations, as no mapping populations are required (Abdurakhmonov and Abdukarimov, 2008; Korte and Farlow, 2013). Association mapping has some limitations too. One of the major limitations is detection of false positives due to the confounding effects of population structure and genetic relatedness present within the populations, if unaccounted (Korte and Farlow, 2013). Fortunately, recent improvement in the statistical models for GWA mapping can handle population structure by accounting for the phenotypic covariance that is due to population structure and genetic relatedness.

Few GWA studies have been performed on *S. sclerotiorum* resistance in *B. napus* (Gyawali et al., 2016; Wei et al., 2016; Wu et al., 2016; Roy et al., 2021). Gyawali et al. (2016) conducted a GWA study using 152 *B. napus* lines and evaluated plants phenotypic performance during the full flowering stage by challenging plants against *S. sclerotiorum*. GWA analyses identified 34 significant loci associated with disease traits, out of which 21 alleles contributed to the resistance explained 6 to 25% phenotypic variance by each locus. In another study by Wu et al. (2016) mapped a total of 26 SNPs corresponding to three loci on chromosome C04, C06, and

C08 associated with adult plant SSR resistance. Moreover, they also predicted 39 putative candidate resistant genes. Wei et al. (2016) detected a total of 17 significant associations, five of which were on A8, and 12 on C6 for stem resistance from two years field data using detached stem assay. Furthermore, they also detected candidate genes specific to the resistant genotypes involved with jasmonic acid pathway, lignin biosynthesis, defense response, signal transduction and encoding transcription factors through the transcriptomic analyses of resistant and susceptible lines.

Comparison of these three GWA mapping (Gyawali et al., 2016; Wei et al., 2016; Wu et al., 2016) in *B. napus* using stem inoculation method (detached stem or intact plant stem) on SSR resistance revealed that none of the resistance loci were shared among these three GWA mapping results. In these three studies, different forms of stem inoculation methods (detached stem/intact plant stem), and the measurement of stem lesion length at 3 dpi (Wei et al., 2016), 5 dpi (Wu et al., 2016), and 7,14, and 21 dpi (Gyawali et al., 2016) were used for phenotyping of rapeseed/canola genotypes against *S. sclerotiorum* infection. The inability to detect common markers in the three GWA studies could be due to differences in phenotyping method. Also, *B. napus* germplasm collection contribute to variation of the detected SNPs/genomic regions among the various mapping studies. However, recently a GWA mapping study by Roy et al. (2021) detected few significant SNPs that were found to be overlap with the genomic regions reported by Wei et al. (2016) and Wu et al. (2016). Therefore, more GWA studies are needed to validate these genomic regions before implementing marker-assisted-selection (MAS) in breeding programs.

2.3.6. Genomic prediction

Breeding for quantitative disease resistance is a challenging task because of its complex mode of inheritance. Therefore, it is important to devise strategies for more effective evaluation and exploitation of this resistance. Recent advancements in the fields of genotyping and statistical modelling helped us to develop new selection schemes that enabled the development of low-cost efficient selection methods for complex traits (Jannink et al., 2010; Bentley et al., 2014). Genomic prediction (GP) uses dense genome wide markers and selects for large numbers of both minor and large effects QTL without prior knowledge of their genomic location (Meuwissen et al., 2001). GP is receiving considerable attention as an alternative approach to the traditional marker-assisted breeding. Instead of identifying statistically significant markers associated with single large effect QTL, GP enables simultaneous estimation of all marker effects to predict breeding values, also referred to as genomic estimated breeding values (GEBVs), of complex traits (Lorenzana and Bernardo, 2009; Lorenz et al., 2011). Genomic selection (GS) has been shown to be effective for improving quantitative traits, both in simulations (Bernardo and Yu, 2007) and in empirical studies (Crossa et al., 2010, 2014; Ornella et al., 2012; Heslot et al., 2012; Lorenz et al., 2012; Rutkoski et al., 2014; Derbyshire et al., 2021). GS uses a 'training population' comprising individuals that have been genotyped and phenotyped for the trait of interest to estimate effects of genome-wide markers or breeding values (BVs). The estimated marker effects or BVs are then used to predict the breeding values (genomic estimated breeding values (GEBVs) of any genotyped individuals that have not been phenotyped (called the selection candidates) (Goddard et al., 2010). Selection of favorable individuals among the selection candidates is then performed based on the predicted GEBVs.

The performance of GS depends primarily on the accuracy of predicting GEBs. GS can increase the frequency of favorable offspring in a population and accelerate gain from selection, when the accuracy of GEBVs are high (Pérez-Cabal et al., 2012). Prediction accuracy is defined as the correlation between GEBVs and the true breeding values. Since in the breeding program, the true breeding values of these traits are not known, we approximated the prediction accuracy by the correlation between the GEBVs and the phenotypic values divided by the square root of the phenotypic heritability ($\sqrt{H^2}$) following Lorenz et al. (2011) and Jarquín et al. (2014). Several factors could affect the GS accuracy which includes gene effects, genetic composition of the training population, level of LD, marker density, model performance, QTL number, relationship between training population and the validation population or selection candidates, training population size, and trait heritability (Zhong et al., 2009; Desta and Ortiz, 2014; Rutkoski et al., 2015).

The potential of implementing GS in rapeseed/canola were explored. Previous GS studies for various agronomic traits, including blackleg and *S. sclerotiorum* disease resistance have shown the potential of GP to accelerate the rapeseed/canola breeding (Würschum et al., 2014; Fikere et al., 2020; Roy et al., 2021; Derbyshire et al., 2021). Wei et al. (2016) investigated the potential of genomic prediction (GP) in a panel of 347 *Brassica napus* lines and they found only 0.27 predictive ability for adult plant SSR stem resistance. Recent studies by Derbyshire et al. (2021) and Roy et al. (2021) achieved 0.35-0.42, and 0.41-0.64 predictive ability for adult plant *S. sclerotiorum* resistance in rapeseed/canola clearly indicated the potential of GS for improving complex SSR resistance inheritance in rapeseed/canola.

CHAPTER 3: GENOME-WIDE ASSOCIATION MAPPING AND GENOMIC PREDICTION FOR ADULT STAGE SCLEROTINIA STEM ROT RESISTANCE IN *BRASSICA NAPUS* (L) UNDER FIELD ENVIRONMENTS[1]

3.1. Abstract

Sclerotinia stem rot (SSR) is a fungal disease of rapeseed/canola that causes significant seed yield losses and reduces its oil content and quality. In the present study, the reaction of 187 diverse canola genotypes to SSR was characterized at full flowering stage using an agar plug to inoculate stems in four environments. Genome-wide association **(**GWA) analyses using three different algorithms identified 133 significant SNPs corresponding with 123 loci for disease traits like stem lesion length (LL), lesion width (LW), and plant mortality at 14 (PM_14D) and 21 (PM_21D) days. The explained phenotypic variation of these SNPs ranged from 3.6-12.1%. Nineteen significant SNPs were detected in two or more environments and disease traits data by at least two GWAS algorithms. The strong correlations observed between LL and the other three disease traits, suggest they could be used as proxies for SSR resistance phenotyping. Sixty-nine candidate genes associated with disease resistance mechanisms were identified. Genomic prediction (GP) analysis with all four traits employing genome-wide markers resulted in 0.41-0.64 predictive ability depending on the model specifications. The highest predictive ability for PM_21D with three models was about 0.64. From our study, the identified resistant genotypes

[1]This article was published in Scientific Reports. This chapter was co-authored by Jayanta Roy, T. M. Shaikh, Luis del Río Mendoza, Shakil Hosain, Venkat Chapara, and Mukhlesur Rahman. JR carried out the experiment, collected the data, analyzed the data, interpreted the results and wrote the manuscript. LRM analyzed the data, interpreted the results, and reviewed the manuscript. TMS, SH, and VC collected the data, and reviewed the manuscript. MR principal investigator of the project, designed the project, coordinated the study, and reviewed the manuscript.

and stable significant SNP markers will serve as a valuable resource for future SSR resistance breeding. Our study also suggests that genomic selection holds promise for accelerating canola breeding progress by enabling breeders to select SSR resistance genotypes at the early stage by reducing the need to phenotype large numbers of genotypes.

Key words: Genome-wide association (GWA) study, genomic prediction (GP), canola, sclerotinia stem rot (SSR), single nucleotide polymorphism (SNP), marker-trait-associations (MTAs)

3.2. Introduction

Sclerotinia sclerotiorum (Lib.) de Bary is a devastating non-host specific, necrotrophic, and ubiquitous plant pathogenic fungus that infects at least 408 plant species including economically important dicotyledonous crops such as oilseed rape, edible dry bean, soybean, sunflower, pea, chickpea, lentils, and different types of vegetables and some monocotyledonous crops such as tulip and onion (Boland and Hall, 1994; Bolton et al., 2006). The disease caused by this pathogen in rapeseed/canola is commonly referred to as Sclerotinia Stem Rot (SSR), and it significantly limits rapeseed yield/production worldwide. This disease imposes 10-20% yield loss per year in China, but the loss could be up to 80% in severely infected fields (Oilcrop Research Institute, Chinese Academy of Sciences, 1975). In the United States, where every percentage unit of incidence reduces on average 0.5% of canola potential yields (Del Río et al., 2007), the annual seed loss due to this pathogen attack has been estimated at about $24 million (United States Department of Agriculture, 2016). Besides seed yield loss, the disease also reduces the oil content and makes changes in the fatty acid profile of affected plants that reduces oil quality (Purdy, 1979; McCartney et al., 1999; Sharma et al., 2015).

The pathogen survives in the soil up to 8-10 years by producing long-lived melanized resting structures called sclerotia (Adams and Ayers, 1979; Hegedus and Rimmer, 2005). Under favorable conditions (*i.e.*, moderate to high moisture and moderate soil temperatures) the sclerotia germinate carpogenically. Airborne ascospores released during the day (Qandah and del Río Mendoza, 2011) and start infection by colonizing on senescent petals. In the presence of free moisture, ascospores germinate and move from petals to leaves (Shahoveisi and del Río Mendoza, 2020) and main stems, where forming lesions may completely girdle the stem and cause death of plant (Rimmer et al., 2003).

Currently, rapeseed/canola growers depend primarily on use of crop rotation with non-host crop species and prophylactic fungicide applications for SSR management due to the unavailability of SSR resistant varieties (Bradley et al., 2006). However, long-term persistent survivability of the sclerotia in the soil and wider host range of the fungus makes crop rotations less effective. Moreover, properly timing fungicide applications to manage the disease is a difficult task to achieve; adds additional input cost and has a negative impact on the environment. Therefore, breeding for disease resistant rapeseed/canola varieties would be an economically feasible, more efficient, and environmentally friendly option. Hereafter, it is crucial to study different germplasm from diverse regions in order to unravel the nature of durable genetic resistance and identify responsible genes for the resistance to *S. sclerotiorum*. Subsequently, such identified SSR resistance genes can be introduced into high performing elite canola cultivars which ultimately diminish the dependence of the canola growers on cultural practices and use of fungicides and making canola production more profitable.

Unfortunately, breeding for SSR resistance is a challenging task as sources of complete resistance to this disease have not been identified in *Brassica napus* and its close relatives in

more than three decades of investigation (Zhao et al., 2006; Yin et al., 2010). Instead, few germplasms with partial resistance have been identified and utilized in the SSR resistance breeding. Several genetic studies have shown that the mode of SSR resistance is quantitatively inherited with additive effect and have medium to high heritability (Yonghong et al., 2001; Wei et al., 2016; Wu et al., 2016; Qasim et al., 2020). To date, genetic mapping studies has been carried out to identify the sclerotinia resistance loci in multiple bi-parental mapping populations developed from crosses between resistant and susceptible parents (Zhao and Meng, 2003; Zhao et al., 2006; Yin et al., 2010; Wu et al., 2013; Wei et al., 2014; Qasim et al., 2020; Shahoveisi et al., 2021). QTL mapping using bi-parental populations have detected several QTLs for SSR resistance, and the majority of them located on chromosomes A09, C02, and C06. However, in very few instances common markers have been detected in different mapping populations (Shahoveisi et al., 2021), which makes it difficult to identify the overlapping QTLs. The availability of the *B. napus* reference genome sequence (Chalhoub et al., 2014) offers an opportunity to determine the physical location of the previously identified QTLs by aligning the QTL primers with the *B. napus* genome. Integration and comparative analyses of the previously identified SSR resistance QTLs from various mapping studies with the reference genome sequence have detected conserved QTLs on chromosome A9 (22.5–27.5 Mb) and C6 (29.5–36.1 Mb) (Li et al., 2015). Despite these successes, no fine mapping or map-based cloning for sclerotinia resistance gene has been reported so far, which ultimately circumvents the utilization of identified QTL in the SSR resistance breeding strategy. To date, all identified sclerotinia resistance QTLs only explained a small portion of phenotypic variance and few QTLs could be detected in different populations, different growth stages, or different screening methods. Moreover, bi-parental QTL mapping strategy suffers from low allelic diversity since only two

allelic effects were evaluated for a single locus and limited recombination events leads to the lower mapping resolution (Korte and Farlow, 2013). Apart from bi-parental linkage mapping strategy, genome-wide association study (GWAS) uses natural population originated from non-cross derived lines which offer extensive historical recombination events and shortened linkage disequilibrium (LD) segments thus provides a promising opportunity of having high mapping resolution for the marker-trait-association (Nordborg and Weigel, 2008).

Recently, the dramatic reduction of sequencing cost and quick turn-around time has led to the development of genome-wide dense molecular markers, which further accelerated the application of GWA mapping and genomic selection (GS) towards the genetic improvement of complex traits. To highlight the effectiveness of GWA study to improve the enhanced SSR resistance and marker-assisted selection, to date, only a few genetic mapping studies through GWA for SSR on rapeseed/canola have been reported. Gyawali et al. (2016) used 84 simple sequence repeat markers to conduct a GWA study using 152 *B. napus* accessions at the flowering stage in a controlled environment and identified 34 significantly associated loci of which 21 alleles contributed to the SSR resistance. Wei et al. (2016) used 30,932 SNP markers to conduct a GWA study and detected five significant associations on A8, and twelve on C6 using detached stem inoculation method. A total of 26 SNPs associated with SSR resistance were identified on chromosome C4, C6, and C8 from a field study based on detached stem inoculation assay and the genotyping data of 25,573 SNPs by Wu et al. (2016). However, the effectiveness of GWA mapping may be limited in detecting common alleles with very small effects, as well as rare variants with small effect (Tam et al., 2019). Similar to GWA, the GS is performed by employing genome-wide markers distributed throughout the genome. GWA analysis detects significant SNP-trait associations that account only a small portion of phenotypic variance,

indicating that there is a significant amount of genetic information that could be captured with a whole genome modeling. Contrary to GWA study, GS has emerged as a promising genomics-assisted technique that uses all the molecular markers information and phenotype data of the training population to develop statistical models that predict genomic estimated breeding values (GEBVs) in testing individuals based only on the genotype information (Meuwissen et al., 2001; Crossa et al., 2017). A number of GS studies have been reported in several crops such as wheat (Heffner et al., 2011; Poland et al., 2012; Odilbekov et al., 2019), and maize (Albrecht et al., 2011; Technow et al., 2013; Liu et al., 2021) in the past ten years. The potential of GS in rapeseed/canola was investigated for various agronomic traits including resistance to blackleg disease (Würschum et al., 2014; Fikere et al., 2020) and concluded as a promising tool for rapeseed breeding. However, the application of genomic prediction for SSR resistance in rapeseed/canola has been limited to date. Recently, Derbyshire et al. (2021) implemented GP for adult plant SSR resistance in *B. napus* and reported that the GS can be used for the improvement of *S. sclerotiorum* resistance.

The objectives of the study were i) to identify SSR resistant genotypes, ii) dissect the genetic architecture of SSR resistance, iii) identify the genomic regions, marker-trait-associations (MTAs), and putative candidate genes conferring SSR resistance, iv) to explore and evaluate the effectiveness of genomic prediction (GP) for selection of genotypes for SSR resistance.

3.3. Materials and methods

3.3.1. Plant materials and experimental design

A panel of 187 diverse spring and semi-winter *B. napus* germplasm accessions and breeding lines originating from 17 countries in the world were collected from North Central Regional Plant Introduction Station (NCRPIS), Ames, Iowa, USA and North Dakota State

University (NDSU) (Table A1). Both NCRPIS and NDSU are public institutions that comply with all necessary regulations to use the seed materials for research and development purposes. The panel was planted in North Dakota State University Agricultural Experiment Station at Carrington, and Langdon in 2019; Carrington and Osnabrock (similar weather conditions and near to Langdon Research Station) in 2020. All the field experiments were carried out using a randomized complete block design with three replications. Each line was grown in six-rows plots (1.5m x 1.2 m) with 40 plants per row. Rows in each plot were distanced 25 cm apart. The field management was done essentially using regular breeding practices. Two commercially available spring canola hybrid cultivars, Pioneer 45S51 and Pioneer 45S56, as resistant checks, and publicly available cultivar Westar as a susceptible check were used in the study.

3.3.2. Disease phenotypic evaluation and plant phenotypic measurements

In this study, we used *S. sclerotiorum* isolate WM031 for all inoculations. This isolate has been used in previous studies because of its high virulence to canola (Shahoveisi et al., 2021). The isolate was cultured on autoclaved potato dextrose agar medium (24 gL^{-1} potato dextrose broth and 1.5 gL^{-1} agar) at 22-24℃ for 48h. The canola plants were inoculated at full flowering stage. The main stem of eight arbitrarily selected plants from each row were inoculated by placing a 7 mm agar plug containing actively growing hyphal tips approximately at a height of 40-50 cm above the ground (Figure 3.1a). Each plug had the hyphal side facing the epidermis of the plant and was held in place by wrapping it to the stem with parafilm to ensure close contact between the pathogen and the stem surface to maintain humidity. The lesion length (LL) on the main stem was measured at 7 days post inoculation (dpi) using a measuring scale. Stem lesion width (LW) with a visual estimation of the percentage of the main stem were girdled by the lesion was also collected at 7 dpi. The status of inoculated plants, dead/alive was recorded

14 and 21 dpi and used to calculate percentage of plant mortality, therefore designated as PM_14D and PM_21D, respectively. Eight inoculated plants per replication of each genotype were sampled for disease evaluation, which resulted in a total about 96 (8 plants x 3 replications x 4 environments) plants evaluation for each accession throughout the study.

Flowering time (FT) is an important developmental stage of the flowering plants in which they switch from the vegetative stage to the reproductive stage and it may have a role in the plant pathogen interactions (Kazan and Lyons, 2016). Therefore, data on days to flowering (DF) was recorded from days of seeding to flowering (when 50% of the plants in each replication of each genotype started flowering) during the year of 2019 and 2020 in both locations. In addition to FT, stem diameter (SD) and stem internode length (IL) may have an effect on SSR disease prevalence on *B. napus*. Therefore, to investigate its effect on SSR resistance, we collected the data on plant SD and IL of the inoculated plant keeping in mind to have the equal number of plants in each plot considering the plant development stage. Data on stem diameter (SD) in both years was measured using a Vernier caliper from the inoculated internodes slightly above where the inoculum was applied at 7 dpi. The internode length (IL) was taken using a measuring scale from the inoculated internode stems at 7 dpi from all the studied environments.

3.3.3. Statistical analyses

The four field experiments were designated as 2019 Carrington (CARR_19), 2019 Langdon (LANG_19), 2020 Carrington (CARR_20), and 2020 Osnabrock (OSN_20), were analyzed individually and as a combined set with SAS 9.4 (SAS Institute Inc., USA). The best linear unbiased estimates (BLUEs) were calculated within single environment analysis and combined analysis across environments (combENV). For the combined analysis across all environments (combENV), homogeneity of variance was determined by dividing the

environment with the highest error mean squares by the environment with the lowest error mean squares. If the calculated ratio was less than 10-fold, then the data from all environments were combined (Tabachnick and Fidell, 2000; Arifuzzaman and Rahman, 2020). For the environment-wise analyses, genotypes were considered as fixed effects and replications as random effects.

For the combENV, a mixed linear model was implemented considering genotypes as fixed effects, environment, replication within the environment, and genotype-by-environment interaction as random effects. The broad-sense heritability (H^2) for each trait was computed for the combENV set as

$$H^2 = \frac{\sigma_g^2}{\sigma_g^2 + \frac{\sigma_{ge}^2}{n} + \frac{\sigma_e^2}{nr}}$$

where σ^2_g is the genotypic variance, σ^2_{ge} is the genotype by environment variance, σ^2_e is the residual error variance, n is the number of environments, and r is the number of replications per environment. Environment-wise and combENV BLUEs for LL and LW phenotypic trait, and only combENV BLUEs for PM_14D and PM_21D were used to perform GWAS analyses. Correlations among all traits were performed by calculating Pearson correlation coefficients in SAS 9.4. To determine whether the collected data was normally distributed, the Shapiro–Wilk test was conducted for all traits in both single environment and a combined dataset. Simple linear regression analysis was performed with stem LL, LW, PM_14D, and PM_21D as dependent variables and flowering time as independent variable in R (R Core Team, 2020) (R Development Core Team). The same analysis was performed to examine the relationship between SD and IL as independent variables and stem LL, LW, PM_14D, and PM_21D as dependent variables.

3.3.4. Genotyping

Fresh young leaf tissues were harvested from each germplasm and lyophilized at -80℃ until used. The total genomic DNA was extracted from the lyophilized tissues using Qiagene DNeasy kit (Qiagen, CA, US) following the manufacturer's protocol. Then the extracted DNA was quantified using NanoDrop 2000/2000c Spectrophotometer (Thermofisher Scientific) and optimized to get the same concentration of DNA. The GBS library was prepared using the *ApekI* enzyme following the protocol described by Elshire et al. (2011). The prepared GBS library was sent to the University of Texas Southwestern Medical Center, Dallas, Texas, USA for DNA sequencing using Illumina HiSeq 2500 sequencer. Single end sequencing reads were mapped to the *B. napus* 'ZS11' reference genome (Sun et al., 2017) using Bowtie 2 (version 2.3.0) alignment tool (Langmead and Salzberg, 2012) with the default parameters. SNP calling was done using TASSEL 5 GBSv2 pipeline (Glaubitz et al., 2014) and 497,336 unfiltered SNPs were identified. High quality SNP were identified through filtering with VCFtools (Danecek et al., 2011) with the following criteria: minor allele frequency (MAF) ≥ 0.05, missing values (max-missing) $\leq 25\%$, depth (minDP) ≥ 3 and physical distance (thin) ≤ 500 bp. As canola is a self-pollinating crop, more than 25% heterozygous SNP were removed using TASSEL (Bradbury et al., 2007). The SNPs that were located outside of the chromosomes (i.e. unknown position), were removed. Thus, a total of 38,510 high quality SNPs were retained. For the present study, we utilized the polymorphic SNP markers data with minor allele frequencies greater than 0.05 on 187 genotypes.

3.3.5. Marker-trait-associations

Marker-trait-association analyses were implemented in the GAPIT R package (version 3.0) (Wang and Zhang, 2021) and GEMMA-MLM (Zhou and Stephens, 2012). In the GAPIT

analysis, we computed principal component (PC) analysis for accounting population structure using *prcomp ()* function (Price et al., 2006) in R and kinship (K) matrix by VanRaden method (VanRaden, 2008) for relationships among individual, both using molecular marker data. The first four PCs were used for model-based clustering analysis to determine the subpopulations using the Mclust package in R. The first four PCA as a population structure and kinship matrix were incorporated in the GWA models to control false-positives. The single locus mixed linear model (MLM) (Yu et al., 2006), and the multi-locus model fixed and random model circulating probability unification (FarmCPU) (Liu et al., 2016) were implemented in GAPIT. Additionally, the identified significant MTAs were verified by performing GWA analyses in another commonly used software *i.e.* GEMMA-MLM (version 0.98.1) through the execution of the command: "gemma -g [genotype file] -a [genotype annotation file] -p [phenotype] -c [first 4PCA] -k [kinship/centered relatedness matrix] -o [output file]. For this analysis, the incorporated first four PCs were the same that we used for FarmCPU and MLM. The kinship matrix was generated using the centered relatedness procedure in GEMMA, used as a random effect variable in the random model. *P-wald* test (the improved calibrated *P*-value in GEMMA) was calculated for the given model. The significant threshold of *P* value for the association between SNPs and traits were determined following the method proposed by Li and Ji (2005). The effective number of independent tests (M_{eff}) among the used 25,809 SNPs were determined by calculating the correlation matrix and eigenvalue decomposition Then the Bonferroni correction was applied based on the effective number of independent tests (loci). The effective number of independent tests was estimated as 127, thus by applying genome-wide type I error rate at $\alpha = 0.05$, the determined significant threshold of $P = 0.05/127 = 0.0004$ or $-\log_{10}(P) = 3.4$. Therefore, the significant threshold value for the association between SNP and traits were

determined by $-\log_{10}(P) \geq 3.4$, which is equivalent to $P \leq 0.0004$, for FarmCPU, MLM, and GEMMA-MLM. The SNPs detected by at least two models in at least one environment were declared as significant and considered as relatively stable significant SNPs. To identify the common significant SNP markers present in more than one environment, a threshold value of $-\log_{10}(P) \geq 3.00$ was used, only when those SNPs that had a lower association threshold ($P \leq 0.0004$) in one environment were considered common. Manhattan plot and *P value* distributions by plotting the observed *P values* against expected *P values* shown in Q-Q plots were created using the mhplot package in R language.

3.3.6. Candidate gene identification

Candidate genes were searched for those significant SNPs that were detected in more than two environments, and two or more GWA models. Genes present within 50 kb upstream and downstream of the significant markers were considered as candidate genes based on the genome and the gene models by '*ZS11*' reference genome sequence (Sun et al., 2017). Protein sequences from the gene models were blasted against TAIR 10 protein database to determine the gene annotation. Genes associated with defense response were identified based on the Gene Ontology terms (GO terms) from TAIR website and gene functions found in the previous literature, TAIR 10, and Uniport-KB.

3.3.7. Genomic prediction

The genomic prediction (GP) models were constructed with the following formula:

$$y = \mu + X\beta + \varepsilon$$

where y is the vector of the phenotypic observations, μ is the grand mean, X is the marker genotype matrix, β is the estimated random additive marker effect, and e is the residual error term. Three GS models *i.e.* ridge regression best linear unbiased prediction rrBLUP (Endelman, 2011) and two Bayesian models: Bayes C (Gianola et al., 2009), and Bayesian Ridge Regression

(BRR) (Meuwissen et al., 2001) were used for implementing genomic prediction. All models were analyzed in R language. The GP model rrBLUP and two Bayesian models were constructed using the package "rrBLUP" and BGLR (version 4.0.4) (Pérez and de los Campos, 2014) package, respectively. For all the analyses, Bayesian models were performed for 5,000 Monte Carlo Markov chain iterations with a 1,000 burn-iterations. The environment-wise BLUEs of LL, LW, and the combENV BLUEs of LL, LW, PM_14D, and PM_21D were used as phenotypic values for subsequent GP analyses. All SNP markers (25,809 SNPs) distributed in the whole genome were employed in the GP. A five-fold cross validation (150 individuals as training population and 37 individuals as validation individuals) with 100 iterations or 100 rounds of random sampling were implemented to assess the accuracy and predictive ability of the GP model for each predicting phenotypic traits with the validation populations. Predictive ability was defined as the correlation (Pearson's *r*) between genomic estimated breeding values (GEBVs) and the observed phenotypic value. The measure of prediction accuracy is the correlation between GEBV and an estimate of true breeding value. However, true breeding values for this trait are typically unknown. Therefore, the prediction accuracy was indirectly estimated by dividing the correlation between GEBV and observed phenotypic value by an estimate of square root of heritability ($\sqrt{h^2}$) (Ould Estaghvirou et al., 2013; Derbyshire et al., 2021). Thus, the prediction accuracy of each model was estimated by dividing the mean predictive ability by square root of heritability ($\sqrt{h^2}$).

3.3.8. Genomic heritability

Genomic heritability (narrow-sense heritability, h^2) was estimated for the combined datasets of all the evaluated traits for each model using all the markers. The additive variance components (*Va*) and the residual variance components (*Ve*) were estimated with *mixed.solve*

function in rrBLUP for the rrBLUP model (Endelman, 2011). Narrow-sense heritability (h^2) was assessed by dividing the additive genetic variance (*Va*) by the total variance estimate (sum of additive variance and the residual variance). For the Bayesian models, h^2 was estimated by taking the average proportion of variance explained by the regression of phenotypes on molecular markers as described by de los Campos et al. (2015) (https://github.com/gdlc/BGLR-R/blob/master/inst/md/heritability.md).

3.4. Results

3.4.1. Phenotypic evaluations

A continuous and broad range of reactions to inoculation with *S. sclerotiorum* was observed among the 187 *B. napus* accessions in this study (Table 3.1, Figure 3.1a-d). The main stems LL of the genotypes at 7 dpi varied from 2.3 to 9.0 cm and LW ranged from 19.3 to 81.4% among the studied environments (Table 3.1, Figure 3.1b-c). In the CARR_19, the main stems LL ranged from 3.0-7.3 cm with the mean of 5.1 cm, whereas LANG_19, CARR_20, and OSN_20 had a range (mean) of 2.8-8.7 cm (5.6 cm), 2.5-7.3 cm (5.0 cm), 2.3-9.0 cm (5.9 cm), respectively (Table 3.1, Figure 3.1b). In the case of stem LW, the CARR_19, LANG_19, CARR_20, and OSN_20 had a range (mean) of 26.9-70.3% (47.7%), 26.7-81.4% (55.1%), 21.9-68.0% (48.1%), and 19.3-78.6% (53.6%), respectively (Table 3.1, Figure 3.1c). The mean LL (5.9 cm) was the highest in OSN_20, and the lowest (5.0 cm) was recorded in CARR_20, whereas the highest overall mean LW (55.1%) was observed in LANG_19 followed by the lowest mean (47.7%) in both CARR_19 environments (Table 3.1, Figure 3.1b-c). The resulting BLUEs for PM_14D and PM_21D for SSR scores across all (combENV) environments ranged from 2 to 63% with an average of 33%, and from 16 to 94% with a mean of 66%, respectively (Table 3.1, Figure 3.1d). The diversity of phenotypic responses observed in this study is

consistent with observations made by other researchers (Wu et al., 2013, 2016; Wei et al., 2016; Qasim et al., 2020; Shahoveisi et al., 2021) and reinforce the notion that resistance to sclerotinia infections is quantitatively inherited and controlled by multiple genes. The combENV BLUEs of the top five promising source of resistance ranged between 2.6 to 4.2 cm for LL, from 23.7 to 40.6% for LW, had between 2 to 10% PM_14D, and between 16 to 37% PM_21D. These ranges were smaller than the respective phenotypic responses observed on the resistant checks "Pioneer 45S51" 5.2 cm, 51.9%, 32%, and 66% for LL, LW, PM_14D, and PM_21D, respectively, and "Pioneer 45S56" 5.2 cm, 49.5%, 31%, and 62% for LL, LW, PM_14D, and PM_21D, respectively, and susceptible check "Westar" 6.8 cm, 65%, 63%, and 94% for LL, LW, PM_14D, and PM_21D, respectively. Therefore, these promising genotypes will serve as a valuable resource to transfer resistance gene into the elite canola cultivars to develop SSR resistant cultivars for the growers. A two-way analysis of variance (ANOVA) indicated that genotype, interaction of genotype and environment had significant effects ($P \leq 0.001$) on both LL and LW for stem resistance. Similar results were obtained from the ANOVA analysis of the combENV sets for PM_14D and PM_21D with the exception of the interaction between genotype and environment on PM_21D, which was not-significant ($P \leq 0.05$) (Table A2). Combined across all environments, high broad-sense heritability of 0.88 and 0.86 was observed for LL and LW, respectively (Table 3.1).

Plant phenotypic variables measured on the 187 genotypes in all environments displayed a wide variation. DF values ranged from 37 to 88 days and had a coefficient of variation (CV) ranging from 3.7 to 6.4 (Table 3.1). IL varied from 7.7 to 20.4 cm with CV between 17.9 to 28.3, whereas SD ranged from 4.0 to 12.9 mm with a CV ranging from 21.9 to 31.2 (Table 3.1). The frequency distributions of phenotypic values for each trait are presented in the Figure A1.

Table 3.1. Phenotypic variation obtained through BLUEs in the response of 187 *Brassica napus* genotypes against sclerotinia stem rot

Traits[a]	Env.[b]	Unit	Min	Mean	Max	Median	CV [c]	Shapiro–Wilk test *p* value	H^2 (family mean basis)
	CARR_19	cm	3.0	5.1	7.3	4.7	24.8	0.0001	
	LANG_19	cm	2.8	5.6	8.7	5.4	26.6	0.0152	
LL	CARR_20	cm	2.5	5.0	7.3	4.7	22.7	0.0000	0.88
	OSN_20	cm	2.3	5.9	9.0	5.6	26.8	0.2339	
	CombENV	cm	2.6	5.4	7.9	5.1	25.6	0.0002	
	CARR_19	%	26.9	47.7	70.3	45.0	24.9	0.0006	
	LANG_19	%	26.7	55.1	81.4	50.0	28.8	0.6422	
LW	CARR_20	%	21.9	48.1	68.0	45.0	25.4	0.0007	0.86
	OSN_20	%	19.3	53.6	78.6	50.0	29.1	0.0694	
	CombENV	%	23.7	51.2	71.3	50.0	27.5	0.0077	
PM_14D	CombENV	%	2.4	32.6	63.1	28.6	53.3	0.7399	0.90
PM_21D	CombENV	%	15.5	66.2	71.4	78.5	26.4	0.0002	0.96
	CARR_19	days	41.3	50.7	79.3	48.0	3.7	0.0000	
	LANG_19	days	37.3	46.7	77.3	44.0	4.0	0.0000	
DF	CARR_20	days	43.0	57.6	87.0	52.0	3.8	0.0000	0.98
	OSN_20	days	42.7	54.5	88.0	50.0	6.4	0.0000	
	CombENV	days	41.1	51.9	82.3	49.0	4.7	0.0000	
	CARR_19	mm	4.4	7.0	12.0	6.7	31.2	0.0022	
	LANG_19	mm	4.5	6.9	10.2	6.7	21.9	0.1440	
SD	CARR_20	mm	4.0	6.9	11.6	6.6	23.7	0.0007	0.88
	OSN_20	mm	5.1	7.5	12.9	7.2	29.4	0.0001	
	CombENV	mm	4.9	7.1	9.9	6.8	27.1	0.0032	
	CARR_19	cm	7.8	11.6	14.5	11.4	17.9	0.1622	
	LANG_19	cm	8.1	12.0	15.4	12.0	19.1	0.0002	
IL	CARR_20	cm	8.4	10.8	18.9	10.5	28.3	0.0000	0.79
	OSN_20	cm	7.7	12.6	20.4	12.3	25.9	0.0139	
	CombENV	cm	8.6	11.7	14.9	11.5	23.1	0.0922	

Traits[a]: LL, lesion length measured at 7 days post inoculation (dpi), LW, lesion width measured at 7 dpi, PM_14D, plant mortality at 14 dpi; PM_21D, plant mortality at 21 dpi; DF, days to flowering; SD, stem diameter; IL, internode length.
Env.[b]: Environments: CARR_19, Carrington 2019, LANG_19, Langdon 2019, CARR_20, Carrington 2020, OSN_20, Osnabrock 2020, CombENV, combined across all environments
CV[c] = Co-efficient of variation.

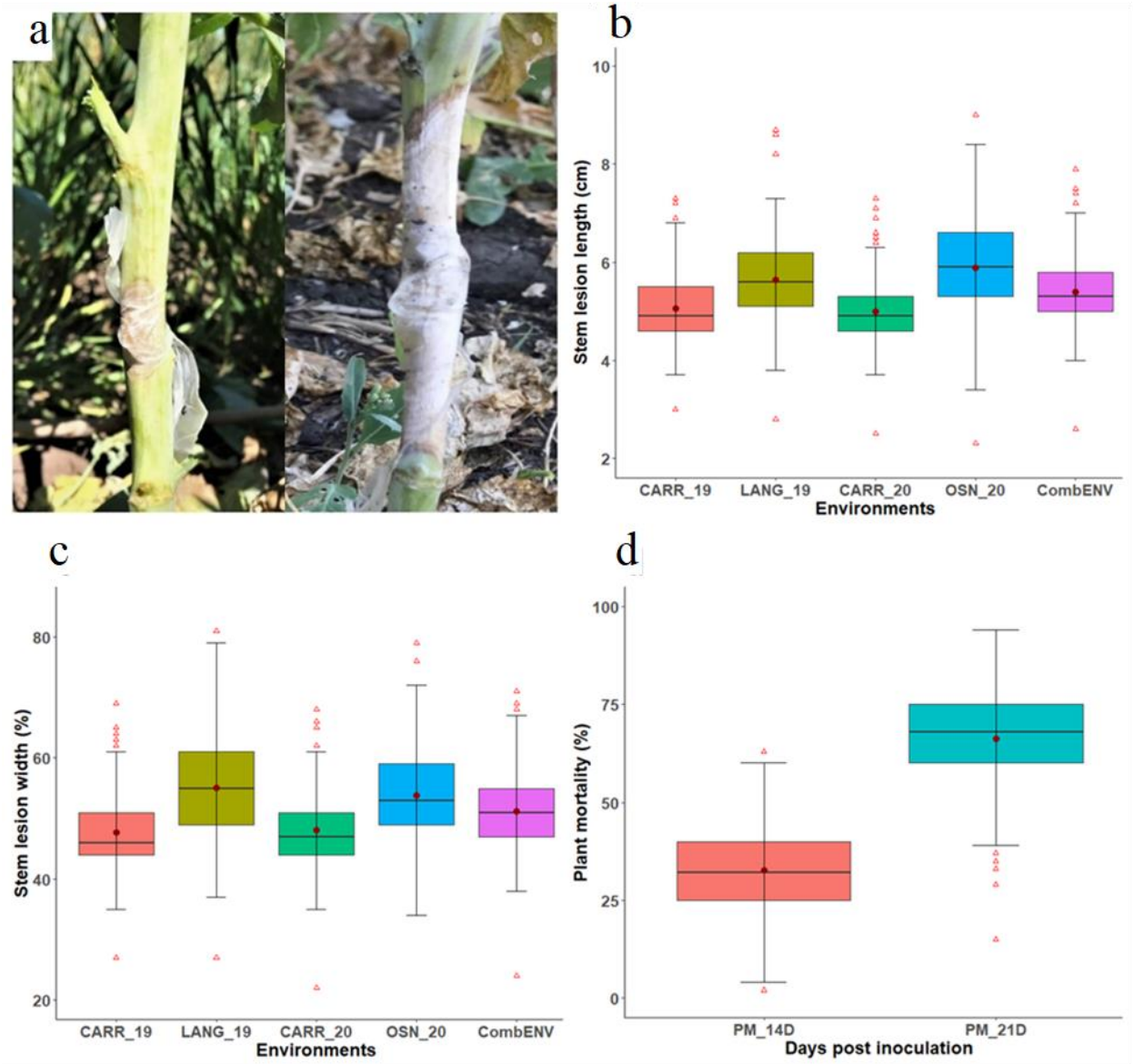

Figure 3.1. Average disease phenotypic characteristics of 187 *Brassica napus* genotypes evaluated at Carrington in 2019 (CARR_19) and 2020 (CARR_20), Langdon in 2019 (LANG_19), Osnabrock in 2020 (OSN_20), and combined across four environments (CombENV), North Dakota. Sclerotinia stem rot lesions on the most resistant (left) and susceptible (right) genotypes at 7 days post inoculation (dpi) (a). Boxplots of BLUEs values for lesion length (cm) measured at 7 dpi (b); lesion width (%) measured at 7 dpi (c); and percentages of plant mortality estimated at 14 and 21 dpi (d). BLUEs for plant mortality are averages of all environments. Box edges represent the upper and lower quartile with median value shown as a bold line in the middle of each box. Mean values are represented by red circle, and the upper and lower whiskers represent the extreme values.

3.4.2. Correlation among internode length, stem diameter, lesion length, and lesion width

To determine the effects of IL and SD on the reaction of genotypes to SSR resistance in respect to stem LL, LW, PM_14D, and PM_21D, efforts were made to maintain homogenous plant densities in every row. We found that combENV BLUEs of LL was negatively correlated with SD [r = -0.34, P= < 0.0001] (Figure 3.2). Similarly, significant negative correlations were also observed between combENV data set of LW and SD (r = -0.44, P= < 0.0001), LW and PM_14D (r = -0.45, P= < 0.0001), LW and PM_21D (r = -0.44, P= < 0.0001) (Figure 3.2). The regression analyses showed that the stem LL and LW were also significantly and negatively associated with SD (r = -0.34, R^2=0.11, P= 1.7 x 10^{-6} for LL; r = -0.44, R^2=0.19, P= 1.5 x 10^{-10} for LW) (Figure 3.3b, e). However, stem LL and IL had significant positive correlation (r =0.49, P= < 0.0001), and similarly a significant positive correlation was also found to be associated between stem LW and IL (r =0.42, P= < 0.0001), LW and PM_14D (r =0.40, P= < 0.0001), LW and PM_21D (r =0.47, P= < 0.0001) (Figure 3.2). The regression analyses between stem LL, stem LW with IL also showed positive correlation (r = 0.49, R^2=0.24, P= 6.9 x 10^{-13} for LL, and r = 0.42, R^2=0.17, P= 1.9 x 10^{-9} for LW) (Figure 3.3a, d). Regression analyses among the stem LW, PM_14D, PM_21D with IL, and SD were presented in the Figure 3.3 and Figure A2. Interestingly, a highly significant positive correlation was observed among stem LL, LW, PM_14D, and PM_21D for stem resistance across all the studied environments and combENV analyses (Figure 3.2). The correlation between stem LL and LW was strong and positive in CARR_19 (r = 0.91), LANG_19 (r = 0.90, P= < 0.0001), CARR_20 (r = 0.91, P= < 0.0001), OSN_20 (r = 0.90, P= < 0.0001), and combined (r = 0.94, P= < 0.0001) environments. Highly significant correlations were also reported between stem LL and PM_14D (r = 0.83, P= < 0.0001), LL and PM_21D (r = 0.75, P= < 0.0001) (Figure 3.2). These results suggest that stem

LW, PM_14D, and PM_21D could serve as proxies for LL during assessment of stem resistance to *S. sclerotiorum* in rapeseed/canola. Therefore, breeders could select any of the phenotypic trait out of four to evaluate the resistance performance of the genotypes in response to *S. sclerotiorum* attack, which might need further verification.

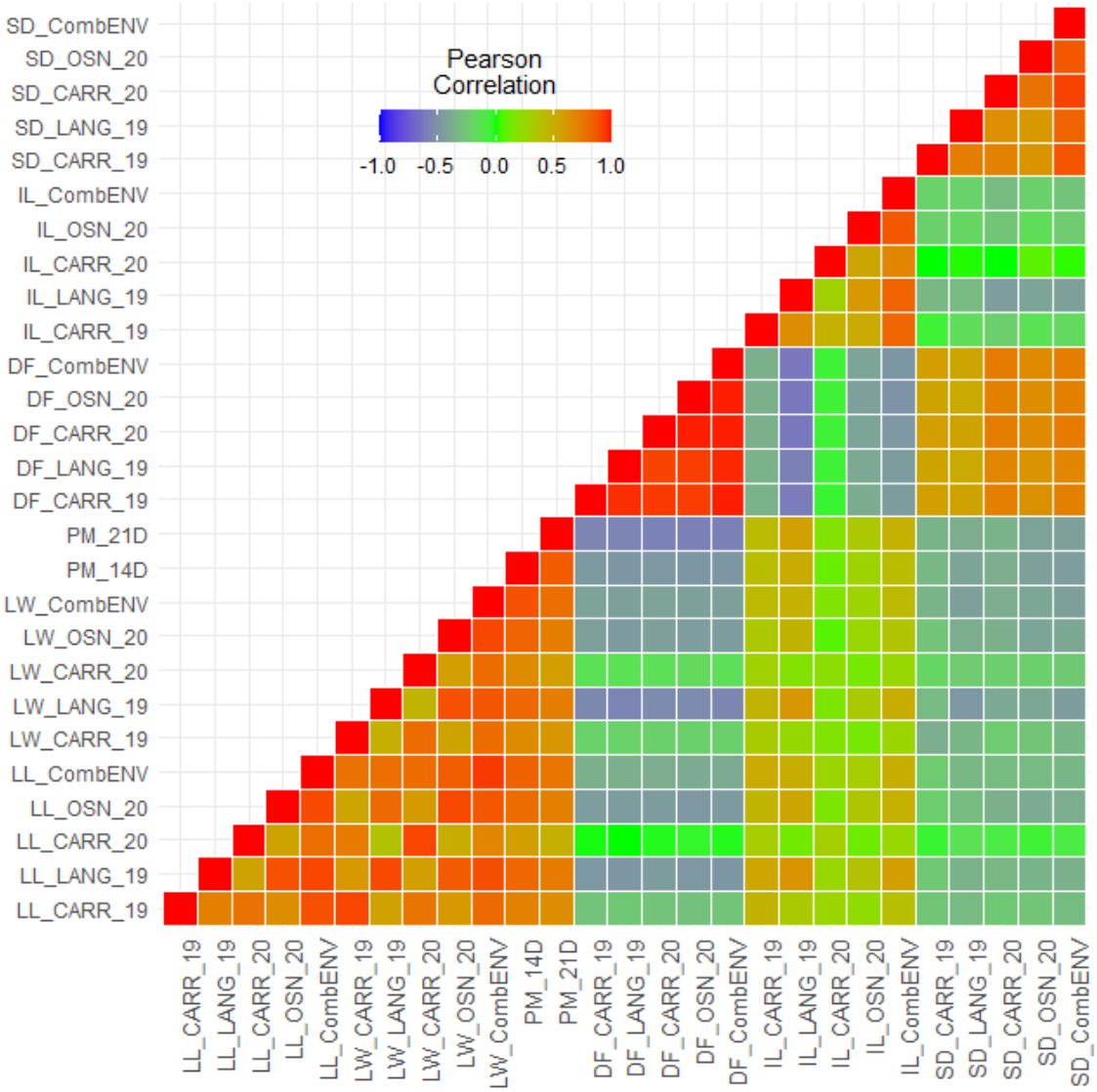

Figure 3.2. Correlation heatmap for different sclerotinia stem rot phenotypic traits in four environments and the combined dataset across environments. Traits: DF= days to flowering; IL= internode length; LL= lesion length, LW= lesion width, PM_14D= plant mortality at 14 days post inoculation (dpi); PM_21D= plant mortality at 21 dpi; SD= stem diameter. Environments: Carrington 2019 (CARR_19) and 2020 (CARR_20), Langdon 2019 (LANG_19), Osnabrock 2020 (OSN_20), and combined across all environments (CombENV). Plant mortality are averages of all environments.

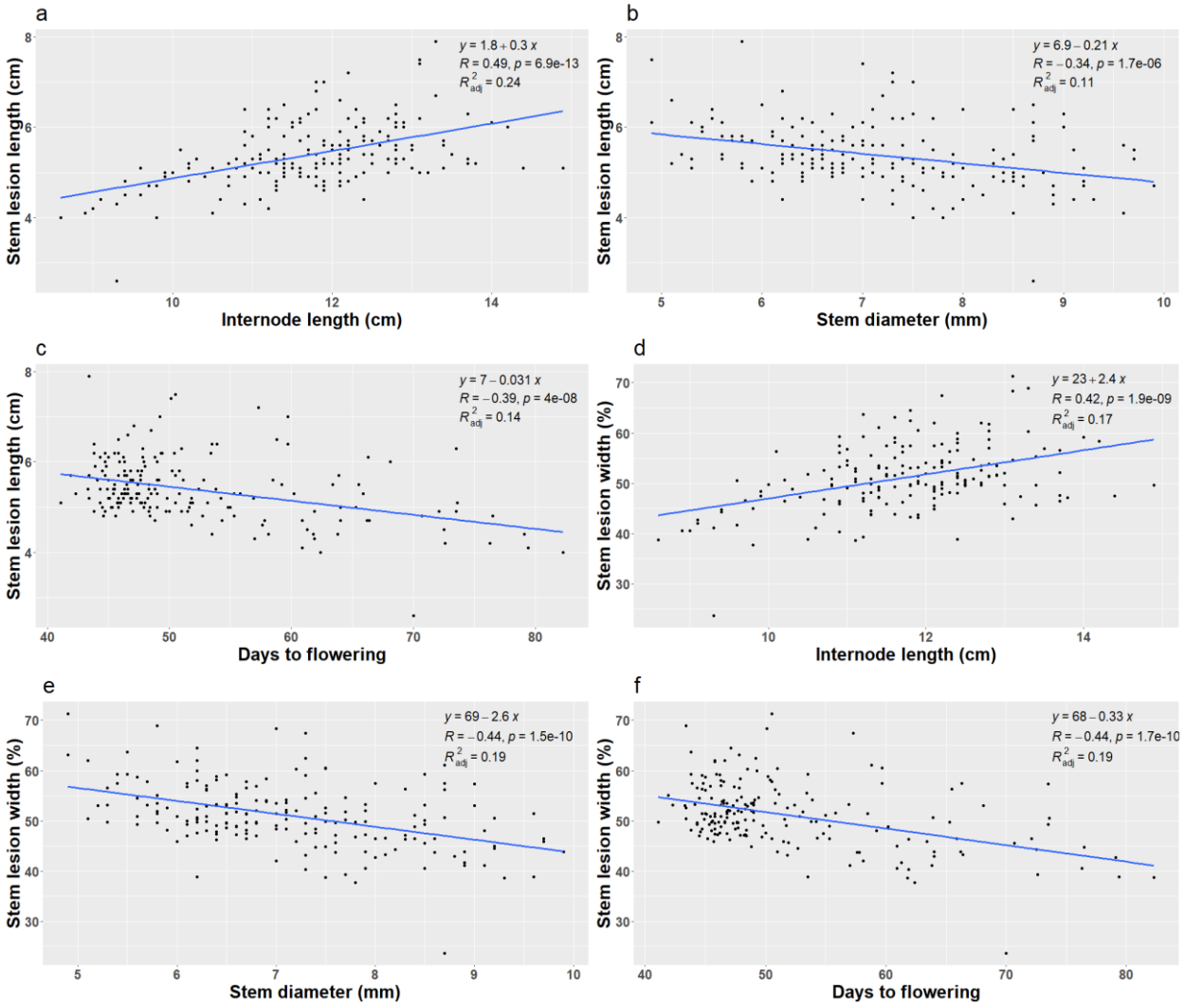

Figure 3.3. Regression analysis of sclerotinia stem rot (SSR) resistance in respect to stem lesion length, lesion width with stem internode length, stem diameter and days to flowering. *R* is Pearson's correlation coefficient between the two traits, R^2_{adj} is the coefficient of determination.

3.4.3. Correlation between days to flowering and sclerotinia stem rot resistance

The DF was significantly and negatively associated with combENV BLUEs of LL, LW, PM_14D, and PM_21D (r = - 0.39, P = < 0.0001 for LL; r = - 0.44, P= < 0.0001 for LW; r = - 0.49, P = < 0.0001 for PM_14D; and r = - 0.59, P = < 0.0001 for PM_21D) (Figure 3.2). The regression analyses showed that DF were negatively and significantly associated with stem LL, LW, PM_14D, and PM_21D (r = -0.39, R^2 = 0.14, P = 4.0 x 10^{-8} for LL; r = -0.44, R^2 = 0.19, P=1.7 x 10^{-10} for LW; r = -0.49, R^2 = 0.24, P = 5.9 x 10^{-13} for PM_14D; r = -0.59, R^2 = 0.34, P =

2.2 x 10^{-16} for PM_21D) (Figure 3.3; Figure A2). These negative correlation results further confirmed that there is a connection between the DF and SSR resistance in *B. napus*, indicating that early flowering genotypes tend to be more vulnerable to the *S. sclerotiorum* attack with increased stem LL, LW, and plant mortality than the late maturing genotypes.

3.4.4. Genotypic data and principal component analysis

After eliminating markers with missing data greater than 25%, a total of 25,809 polymorphic SNPs with minor allele frequency (MAF) greater than 5% were obtained and employed for association analysis. The highest proportions of SNPs had MAF between 0.10 and 0.15 (20%) and between 0.05 and 0.10 (20%) (Figure A3). The other seven MAF classes represent between 3 to 14% each of the total markers. To scan the population stratification of the association panel, principal component analysis and kinship matrix were performed on the genotypes based on 25,809 SNPs. The first and second PCA accounted for 9.1 and 5.8 % of the variance, respectively. The first 4 PCA accounted for 22% of the variance, and at PC4 the inflection point occurred, so we used four PCs in association mapping to avoid the confounding effect due to population structure. The model-based cluster analysis using the first four PCs suggested that there were 5 subgroups within the genotypes (Figure 3.4).

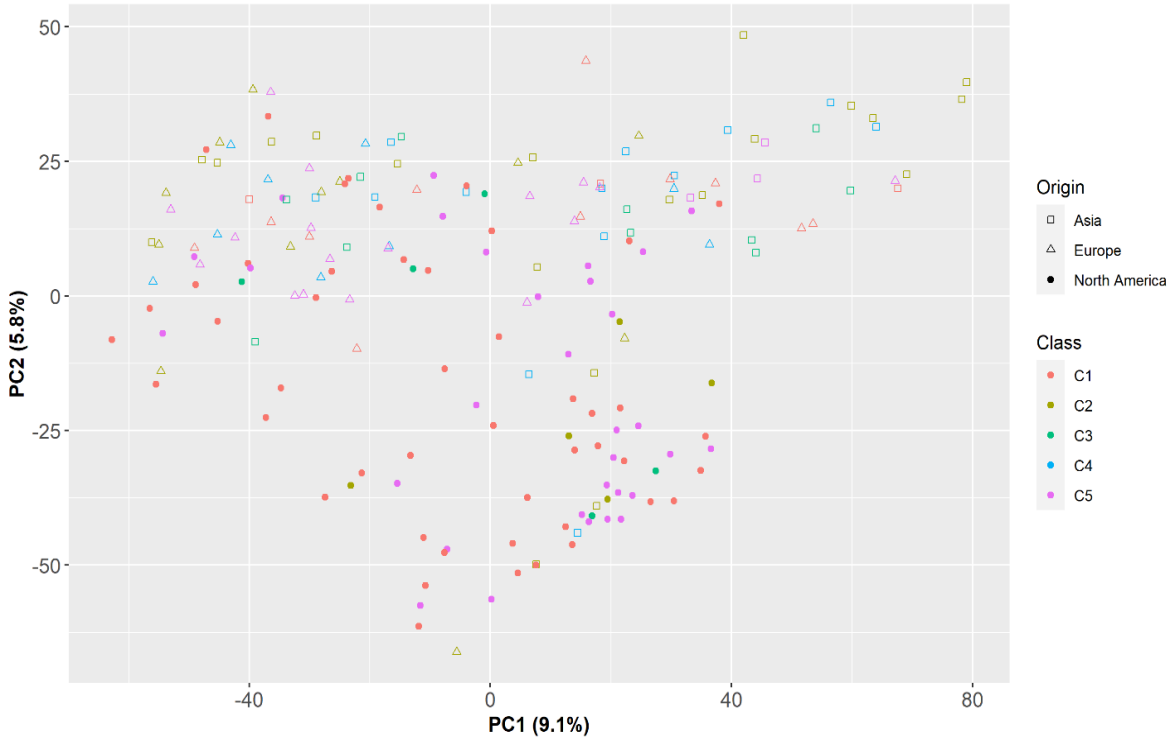

Figure 3.4. Population structure of rapeseed/canola genotypes as reflected by the scatter plot of PC1 and PC2 derived from a principal component analysis

3.4.5. Marker-trait-association (MTA) analysis

Association analyses using the phenotypic data and SNP marker data were conducted for each phenotypic trait for stem resistance (LL, LW, and PM) in each year and with the combENV BLUEs data to identify best MTAs. To reduce false positive or false negative associations *i.e.* the chance of committing Type I and Type II errors, three different GWA mapping algorithms *i.e.* FarmCPU, MLM, and GEMMA-MLM were used to identify the MTAs. The population structures using four PCA and familial relatedness with kinship matrix were incorporated in the implemented MLM and GEMMA-MLM models to control pseudo associations. Incorporation of PCA and kinship matrix in the MLM models as covariates adjusts the correction tests to control false positives, but could not solve the confounding problem between the covariates and test marker, resulting false negatives (Liu et al., 2016). FarmCPU is the model that effectively

corrects both false positives and false negatives. In FarmCPU, the Multiple Loci Linear Mixed Model (MLMM) is divided into two parts: fixed effect model (FEM) and a random effect model (REM) and uses them iteratively. The first part (FEM) contains testing maker, one at a time, and multiple associated markers fitted as covariates to control false positives. To avoid the over fitting model problem in FEM, the associated markers are estimated through maximum likelihood method in REM by using them to define kinship (Liu et al., 2016). In the current study, the SNPs detected in any two GWA models were considered reliable and declared as significant SNP for the studied trait. Significant MTAs were determined on the basis of modified Bonferroni correction by calculating the effective number of independent tests (loci) from the tested 25,809 SNPs by Li and Ji (2005). The Q-Q plots generated from all GWA analyses models of the analyzed phenotypic traits showed a sharp deviation from the expected *P* value distribution in the tail area, indicating that population structure and familial relatedness were well controlled and false positive associations were reduced (Figure 3.5a-e, Figure 3.6a-b).

3.4.5.1. Stem lesion length

Association analysis was performed using BLUEs of LL of all four environments and combENV BLUEs separately (Figure 3.5a-e, Figure A4, and A5). A total of 64 significant SNPs corresponding to 62 loci were identified at the level of [$-\log_{10}(P) \geq 3.4$; $P \leq 0.0004$] by at least two of the GWA models, and thus were regarded as more reliable. These SNPs were unevenly distributed among the *B. napus* chromosomes (Figure 3.5a-e, Figure A4, and A5; Table A3, A6). The majority of significant SNPs were detected on chromosomes A01 (5), A03 (5), A09 (6), C03 (8), and C06 (12). Significant SNPs that were present in the LD block on the same chromosome were regarded as single locus. Among these, thirty-eight significant SNPs were detected in two or more environments and in at least two of the GWA tested models and explained phenotypic

variance of the SNPs ranged from 4.5 to 9.9%. Allelic effects of these identified SNPs varied from -0.84 to 0.83 (Table A3, A6).

3.4.5.2. Stem lesion width

GWA analyses using LW BLUEs of all environments and combENV detected a total of 70 significant SNPs in 66 loci in at least one of the four environments and combENV datasets (Table A4, A6). Out of these 70 significant SNPs, a total of 30 were found in at least two or more environments of two GWA models out of three GWA models. The estimated allelic effects of those 70 significant SNPs varied from -6.83 to 7.55. The phenotypic variation accounted for by these SNP markers varied between 4.9 to 12.1% (Table A4, A6). Manhattan and Q-Q plots summarizing the analysis of stem LW for SSR resistance by FarmCPU, MLM, and GEMMA-MLM are shown in (Figure A6, A7, and A8).

3.4.5.3. Plant mortality

CombENV BLUEs value of 14 and 21 dpi plant mortality were used to perform the GWA analyses. Marker-trait-association analyses identified a total of 21 and 30 significant markers for PM_14D and PM_21D, respectively, which were commonly identified in at least two of the GWA analysis models (Figure 3.6a-b, Table A5, A6). A total of 11 significant SNP markers were commonly found in PM_14D and PM_21D (Table A5). About 3.6 to 7.7% of the phenotypic variation were explained by these significant SNP markers. The estimated allelic effects were ranged between -11.29 to 9.37 (Table A5, A6). The MTAs resulting from FarmCPU, MLM, GEMMA-MLM for PM_14D and PM_21D were presented in the Manhattan and Q-Q plots (Figure 3.6a-b, Figure A9).

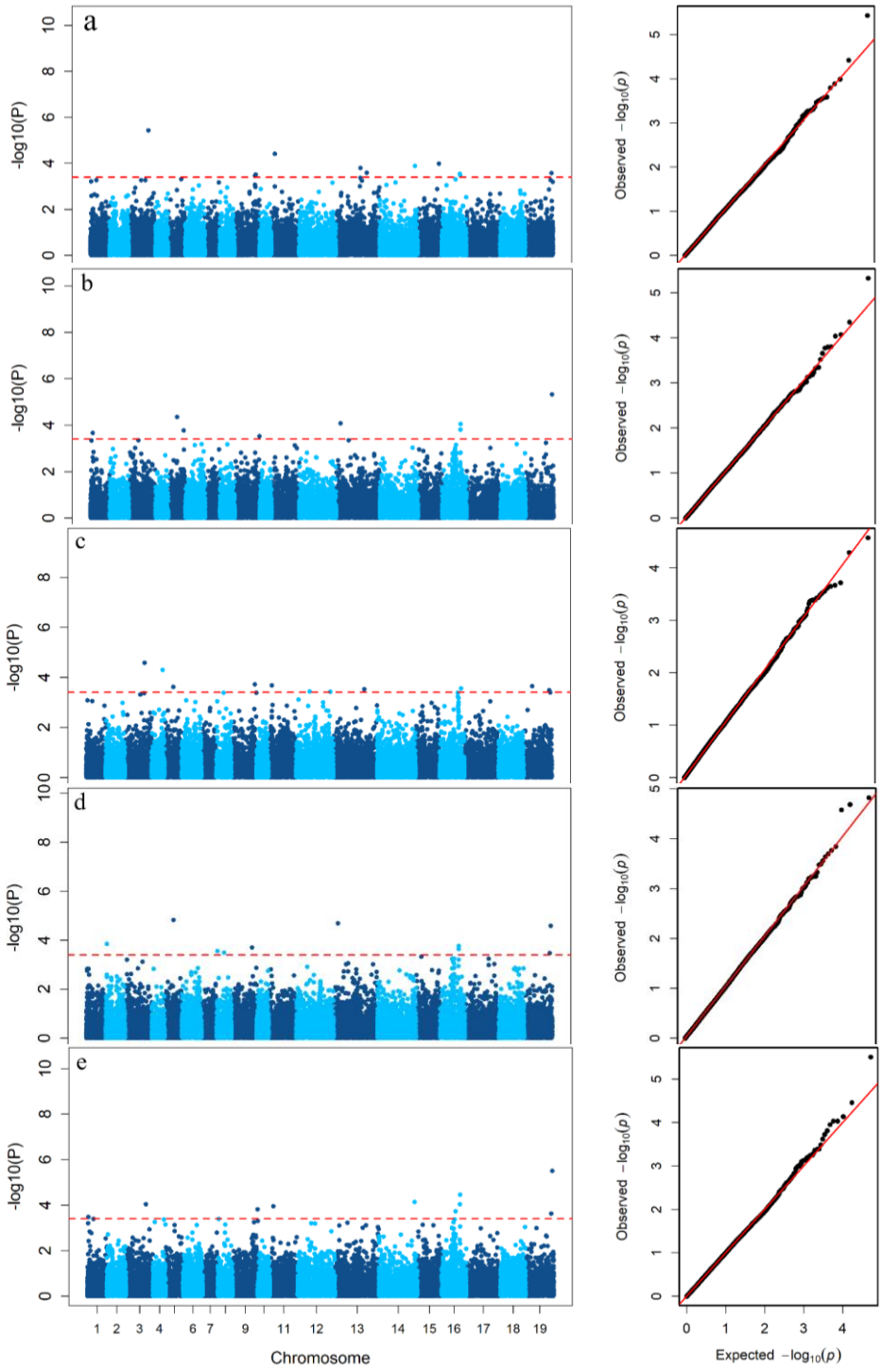

Figure 3.5. Manhattan and Q-Q plots showing the results of marker-trait association for sclerotinia stem rot resistance in 187 rapeseed/canola genotypes by the FarmCPU GWAS model. a) Lesion length, Carrington 2019; b) lesion length, Langdon, 2019; c) lesion length, Carrington 2020; d) lesion length, Osnabrock 2020; e) lesion length, combined data (CombENV). The -$\log_{10}$ (*P*) values from a genome-wide scan are plotted against positions on each of the 19 chromosomes. Discontinued horizontal lines indicate the genome-wide significance threshold.

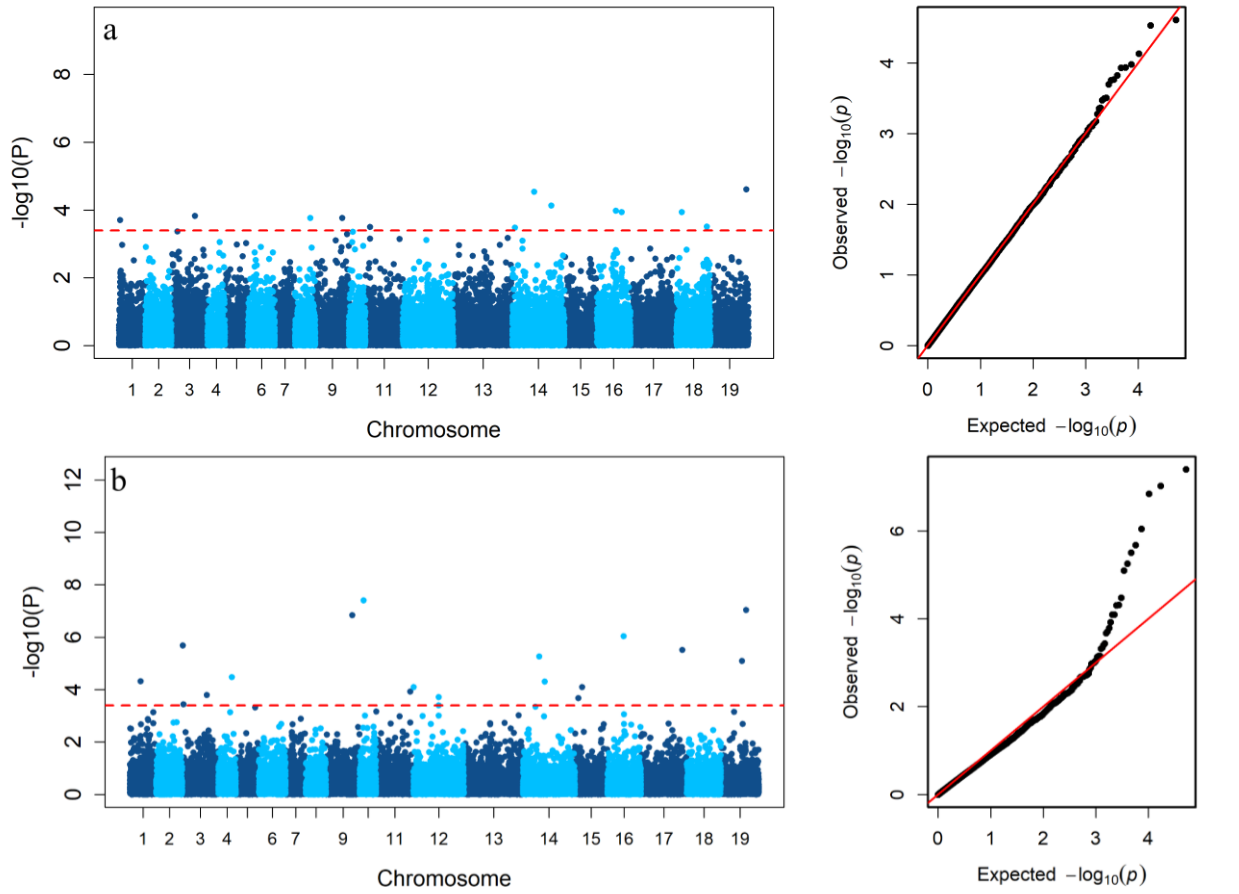

Figure 3.6. Manhattan and Q-Q plots showing the results of marker-trait association for sclerotinia stem rot resistance in 187 rapeseed/canola genotypes by the FarmCPU GWAS model. a) plant mortality at 14 days post inoculation (dpi), combined data (CombENV); and b) plant mortality at 21 dpi, combined data (CombENV). The -$\log_{10}$ (*P*) values from a genome-wide scan are plotted against positions on each of the 19 chromosomes. Discontinued horizontal lines indicate the genome-wide significance threshold.

3.4.6. Candidate genes

Significant SNPs detected in at least two environments (four environments and combENV) were used to search for the candidate genes for sclerotinia stem rot resistance using "*ZS11*" reference genome sequence (Sun et al., 2017). A total of 69 candidate genes with known functions associated with plant disease resistance mechanisms were identified within ± 50 kb of the respective significant SNPs. A list of these genes, their biological functions based on TAIR 10 and Uniport-KB, their annotations and corresponding details is provided in Table A7. The

candidate genes are involved in the biological process of defense response, defense response to fungus, programmed cell death, response to molecule of fungal origin, response to salicylic acid, indole glucosinolate biosynthetic process, induced systemic resistance, response to chitin, jasmonic acid mediated signaling pathway, ethylene-dependent systemic resistance, systemic acquired resistance, camalexin biosynthetic process, pattern recognition receptor signaling pathway, response to wounding, response to nematode, response to oxidative stress, toxin catabolic process, immune response, reactive oxygen species metabolic process, brassinosteroid mediated signaling pathway and other biological processes which might play key role in SSR resistance in rapeseed/canola (Table A7).

Table 3.2. Genomic heritability (narrow-sense heritability) of the combined analyzed phenotypic traits of *Brassica napus* genotypes for sclerotinia stem rot obtained using all SNPs

Model	Traits[a]	Narrow-sense heritability (h^2)
rrBLUP	LL	0.65
Bayes C	LL	0.68
BRR	LL	0.65
rrBLUP	LW	0.6
Bayes C	LW	0.67
BRR	LW	0.64
rrBLUP	PM_14D	0.51
Bayes C	PM_14D	0.62
BRR	PM_14D	0.62
rrBLUP	PM_21D	0.89
Bayes C	PM_21D	0.77
BRR	PM_21D	0.77

Traits[a]: LL, lesion length; LW, lesion width; PM_14D, plant mortality at 14 days post inoculation; PM_21D, plant mortality at 21 days post inoculation.

3.4.7. Genomic prediction

The three GS models used in this study showed more or less similar results across all traits that we evaluated (Figure 3.7a-b, Figure 3.8a-d). However, rrBLUP tended to generate

better results than others in most cases. Slightly differences in the predictive abilities were observed among the used models for the studied traits. The predictive abilities applying genome-wide markers for stem LL and LW traits varied from 0.06-0.51 and 0.12-0.52, respectively, for four individual environments (Figure 3.7a-b). The LANG_19 environment showed the highest predictive ability for both LL (0.51) and LW (0.52) by rrBLUP, whereas the lowest predictive ability of 0.06 and 0.12 was observed for the CARR_20 environment by Bayes C model (Figure 3.7a-b). For environment-wise LL and LW traits, 1 to 9-unit and 0 to 6-unit differences in predictive ability was observed among the models, respectively and the highest differences were found in CARR_20 environment for both traits. However, slightly differences (1 to 2-unit) in the predictive ability was observed for the combENV datasets of all traits. The average correlation between the GEBVs and the observed resistance to SSR by GP models were ranged by 0.41-0.43, 0.42-0.44, 0.47-0.49, and 0.63-0.64 for combENV LL, LW, PM_14D, and PM_21D, respectively (Figure 3.8a-d). Overall, Bayes C and BRR models perform slightly poor over the rrBLUP model for all traits with an exception for PM_21D trait, where both Bayesian models resulted 1-unit increase in predictive ability than rrBLUP model. The predictive ability of PM_21D trait was about 47-56%, 43-52%, and 28-31% higher than the LL, LW, and PM_14D traits, respectively.

Since, the true breeding value of these traits are unknown, we estimate the approximate prediction accuracy to divide the correlation between the phenotypes and the mean predictive ability obtained through cross-validation sets by the square root of heritability ($\sqrt{h^2}$). Therefore, we estimated the narrow-sense heritability based on the whole datasets using the same GS model used to fit the cross-validation sets. The genomic heritability varied, depending on the traits and used GS models as shown in Table 3.2. Thus, after using the genomic heritability, the estimated

prediction accuracy ranged from 0.49 to 0.53 for LL, 0.51 to 0.56 for LW, 0.59 to 0.68 for PM_14D, and 0.67 to 0.77 for PM_21D traits, depending on the used GS models (Figure 3.8a-d). Overall, the predictive ability and accuracy results suggested that genomic predictions were stronger when plant mortality data were used rather than the lesion length and width.

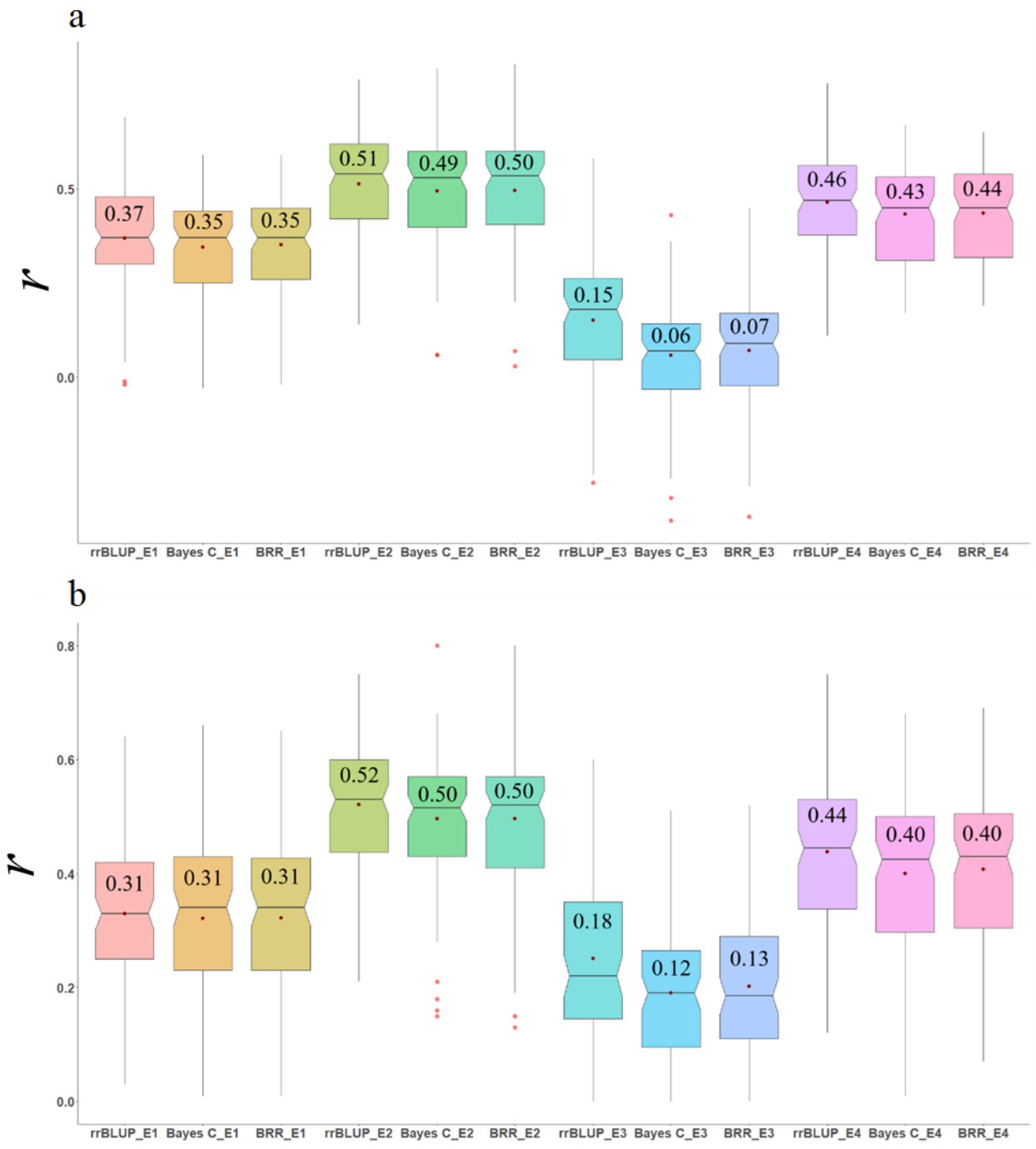

Figure 3.7 Predictive ability for sclerotinia stem rot resistance estimated from the five-fold cross-validation schemes of the association panel. Boxplot showing the result of average predictive ability (*r*) (y axis) for lesion length (a), and lesion width (b) in four environments with rrBLUP, Bayes C and Bayesisan ridge regression (BRR) models (x-axis). E1, E2, E3, and E4 represents Carrington 2019; Langdon 2019; Carrington 2020; Osnabrock 2020 environements, respectively. The boxes show second and third quartiles and wishkers show interquertile range. The red dot in each box plot represent the mean predictive ability. The number above horizontal black bars are the predictive ability (*r*).

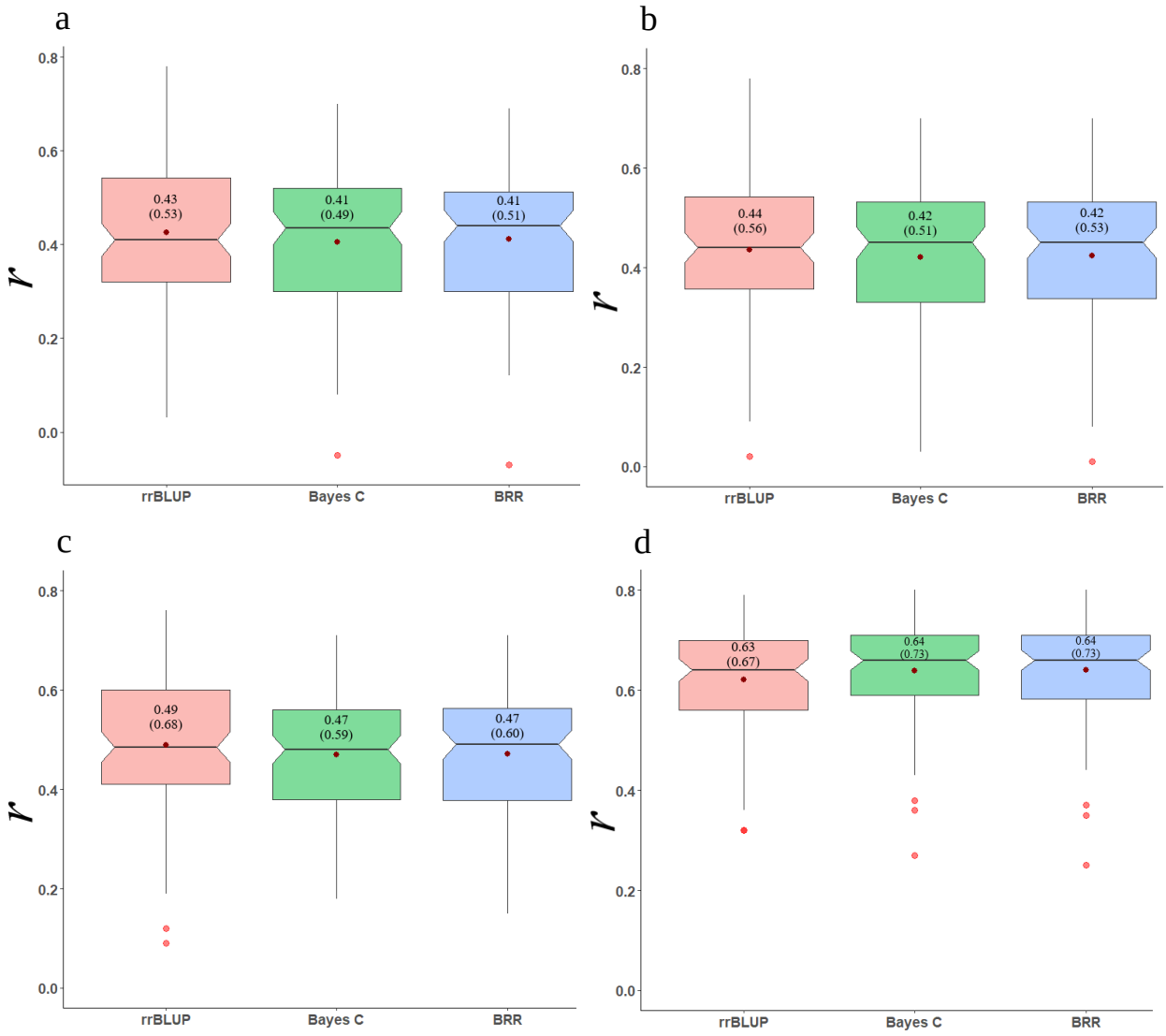

Figure 3.8. Predictive ability and accuracy for sclerotinia stem rot resistance associated phenotypic traits estimated from the five-fold cross-validation schemes of the association panel. Boxplot showing the result of average predictive ability (*r*) (y axis) and accuracy for combENV stem lesion length (a), combENV stem lesion width (b), combENV plant mortality at 14 days post inoculation (PM_14D) (c), combENV plant mortality at 21 days post inoculation (PM_21D) (d) with rrBLUP, Bayes C and Bayesisan ridge regression (BRR) models (x-axis). The boxes show second and third quartiles and wishkers show interquertile range. The red dot in each box plot represent the mean predictive ability. The number above horizontal black bars are the predictive ability (*r*) (at the top) and prediction accuracy (below in brackets).

3.5. Discussion

Sclerotinia stem rot (SSR) is one of the most economically important and devastating fungal disease of rapeseed/canola that significantly limits seed yield, oil content, and oil quality worldwide. SSR is a highly heritable complex trait, controlled by many genes with minor additive effects (Yonghong et al., 2001; Wei et al., 2016; Wu et al., 2016; Qasim et al., 2020). Since, source of completely durable genetically resistant genotypes against this disease have not been identified to date in rapeseed/canola, breeding for SSR resistance is primarily dependent to a large extent on the utilization of partially resistant source (Zhao et al., 2006; Yin et al., 2010). In this study, we explored our rapeseed/canola diversity panel, including released cultivars, advanced breeding lines, and landraces from the different geographical regions with high genetic diversity against SSR in field trials conducted in four environments by inoculating plants artificially.

This study was designed to obtain the most accurate phenotypic and genotypic data possible. *S. sclerotiorum* isolates can vary widely in their virulence, or ability to cause damage to plants and consequently, plant genotypes may respond differently to different isolates (Garg et al., 2010; Otto-Hanson et al., 2011). We used a highly virulent *S. sclerotiorum* isolate, WM031, for this study. This selection allowed us to increase the infection efficiency and effective phenotypic screening for SSR resistance. The effective screening would allow us to evaluate genotypes to produce reliable phenotypic value to identify MTAs with SSR resistance. To generate stable phenotypic values, we have conducted the study at four location-years with three replications in each location. To mimic the natural sclerotinia infections on rapeseed/canola plants, we used agar-plug stem inoculation method to inoculate the plants. This screening method has been implemented successfully by other researchers for the effective identification of

SSR resistance (Li et al., 2006; Qasim et al., 2020; Shahoveisi et al., 2021). Plants were inoculated during the flowering, because this is the most prevalent stage for SSR infection in natural field conditions (Wu et al., 2013). Moreover, the occurrence of SSR at the adult plant stage is a major cause for the seed yield loss and reduced oil content and quality. Therefore, we decided to identify the SSR resistance genotypes and significant MTAs at the mature plant stage in order to incorporate the resistance into the elite canola breeding cultivars and to facilitate MAS in rapeseed/canola breeding program. Hereafter, we analyzed 187 rapeseed/canola diverse genotypes for SSR resistance under field conditions. This germplasm primarily originates from North America, Europe, Asia and comprises of 26.7, 26.2, and 47.1%, respectively. Gyawali et al. (2016) carried out GWA analyses for resistance to SSR under controlled conditions using 152 accessions collected from 18 countries consisting of 0.7% Australia, 69.7% Asia, 20.4% Europe, 7.2% North America, 0.7% South America, and 1.3% from the unknown origin. Another GWA study for SSR resistance conducted by Wu et al. (2016) used a panel of 448 germplasm accessions comprised of 93.8% (Asia), 1.1% (Australia), 1.1% (North America), and 4.0% (Europe) geographical origins. The geographic distribution of the genotypes used in our study provides a good coverage of world-wide germplasm accessions.

The reaction of the 187 rapeseed/canola diverse genotypes was evaluated and measured in three different ways, lesion length (LL), lesion width (LW), and plant mortality for SSR resistance. In our study, wide phenotypic variability was observed in the stem LL (2.3-9.0 cm), LW (19.3-81.4%), PM_14D (2.0-63%), and PM_21D (16-94%) against the disease infection, indicating that this diversity panel was ideal for performing GWAS. Phenotypic variability together with ideal diversity panel and high number of SNPs would potentially increase the effectiveness and efficiency of significant association detection *via* marker-trait-associations

(MTAs) (Josephs et al., 2017; Karikari et al., 2020). Lesion length on the main stem is the most commonly used phenotypic parameter in studies that evaluate the resistant performance of genotypes against SSR in rapeseed/canola (Li et al., 2006; Wei et al., 2016; Wu et al., 2016; Qasim et al., 2020). The association study implemented by Gyawali et al. (2016) used lesion length and percentage of soft and collapsed lesions on the inoculated stem by noting depth of penetration for the assessment of SSR resistance. However, Wei et al. (2016), and Wu et al. (2016) used lesion length as a phenotypic trait for the evaluation of SSR resistance and GWA analyses. In addition to LL, the LW and PM were also recorded. There were significant differences among the genotypes in relation to LL, LW, and PM at 14 dpi and 21 dpi. Results of this study clearly indicated that LW, PM_14D, and PM_21D could be used as alternative phenotypic disease traits for the assessment of SSR resistance in rapeseed/canola. Since, plant mortality is directly related to yield performance, therefore it may be necessary to record plant mortality in addition to the other associated traits for sclerotinia phenotyping. Plant mortality has successfully been used by Shahoveisi et al. (2021). However, Li et al. (2006) evaluated 42 *B. napus* and 12 *B. juncea* genotypes for SSR resistance for PM at 21 dpi under field conditions and did not find any significant differences among the genotypes. Interestingly, we found strong significant correlations among the LL, LW, PM_14D, and PM_21D. Therefore, LW and PM could be used as an alternative phenotypic trait/parameter for breeders and pathologists to successfully differentiate and identify the potential SSR resistance genotypes under field conditions. To the best of our knowledge, this is the first record where LW was used as an alternative to LL, and we demonstrated a significantly high positive correlation between these two traits. The heritability of the stem resistance measured using LL was high which is consistent with previous studies (Wei et al., 2016; Wu et al., 2016; Qasim et al., 2020). Medium to high

heritability for LW, PM_14D, and PM_21D was also estimated from the replicated multiple-location trials and combENV analyses, implying that phenotypic variation is mostly derived from genetic variance and phenotypic selection is effective for improving SSR resistance and subsequent association analyses to identify favorable alleles associated with SSR resistance to utilize in MAS.

Several agronomic traits such as plant height, canopy architecture, stem diameter, and flowering time were reported as associated with the sclerotinia disease severity in different crops such as canola, soybean, dry bean etc (Kim and Diers, 2000; Kolkman and Kelly, 2002; Li et al., 2006; Wu et al., 2019; Zhang et al., 2019; Qasim et al., 2020). Therefore, the relationships between SSR disease phenotypic traits (LL, LW, PM_14D, and PM_21D) and three agronomic traits such as FT, SD, and IL were also explored to assess whether they have direct or indirect effects on the SSR resistance. Results from this study showed that FT and SD had a significant and negative correlation with LL, LW, PM_14D, and PM_21D on *B. napus*. A similar association was reported in previous studies of this pathosystem (Zhao et al., 2006; Wei et al., 2014; Wu et al., 2019; Zhang et al., 2019; Qasim et al., 2020), and implied that early flowering genotypes were more prone to SSR susceptibility with increased stem LL, LW, and plant mortality. A similar association has been reported on other pathosystems, like *Arabidopsis-Verticillium dahlia* (Veronese et al., 2003); *Arabidopsis-Fusarium oxysporum* (Lyons et al., 2015); rice-*Pyricularia oryzae* (Zhao et al., 2011). Studies carried out by Wu et al. (2019) and Zhang et al. (2019) detected few co-localized QTL for the SSR resistance and FT in *B. napus*. They suggested a possible genetic linkage between these two traits. However, the underpinning genetic and molecular mechanisms controlling these associations are still not evident (Wu et al., 2019). In addition to the effect of FT, Qasim et al. (2020) found a weak negative correlation

between LL and SD, which is in agreement with our current study. Li et al. (2006) observed lower stem LL and plant mortality when the stem diameter was around 10 mm. However, increased lesion length and plant mortality were observed when SD was smaller or larger than 10 mm. Also, significant, positive, but moderate correlations were detected between IL and stem LL, IL and stem LW, IL and PM_14D, and IL and PM_21D. Therefore, this is an indication that evaluation of IL could be another useful parameter for an indirect selection for potential SSR disease resistant genotypes to use in the rapeseed/canola breeding program. Based on the findings from our study as well as from previous studies, effect of agronomic traits *i.e.* stem IL and SD need to be taken into careful consideration for the breeders and pathologists to identify and select the accurate promising SSR resistance genotypes phenotypically from the field screening.

Bi-parental linkage mapping and association mappings have been used to dissect the complex traits such as SSR resistance in *B. napus* in order to identify the genetic loci conferring resistance. GWA mapping is a powerful genetic mapping strategy for the dissection of complex traits in plants (Rahman et al., 2016; Wei et al., 2016; Liu et al., 2021). Therefore, in this study, GWA analysis was implemented using three different models for SSR resistance associated traits *i.e.* LL, LW, PM_14D, and PM_21D with the objectives to maximizing opportunities to identify reliable and stable specific genomic regions and SNPs conferring SSR resistance in rapeseed/canola germplasm. This is the first time to use different single locus (MLM and GEMMA-MLM) and multi-locus (FarmCPU) GWA models to identify common markers associated with this disease. The purpose of using three different GWA softwares/algorithms was to reduce the chances of committing type I (false-positive association) and type II (false-negative association) errors. The identification of commonly detected SNPs simultaneously with multiple

GWA models and traits would improve the reliability of the detected MTAs associated with SSR resistance. As the SSR resistance is a quantitatively inherited complex trait and the number of SNP markers is larger than the sample size, it would be necessary to simultaneously use multiple methods for GWAS mapping to identify stable MTAs. Bonferroni-Holm correction (Holm, 1979) for multiple testing ($\alpha = 0.05$) was too conservative, since it assumes that all the tests are independent but in reality, some SNPs may not be independent and they might be in linkage disequilibrium (LD) due to their physical distance or other associated factors. Moreover, with this Bonferroni-type correction, no significant associations between markers and evaluated traits in most of the environments were detected. The use of stringent significant probability threshold reduces the risk of accepting false positives but does not necessarily reduce the risk of rejecting true MTAs. Therefore, the significant threshold value for the association between SNP and traits were estimated by the method proposed by Li and Ji (2005). In this study, most of the significant SNP markers associated with the SSR resistance traits (LL, LW, PM_14D, and PM_21D) detected in the environment-wise and combENV datasets showed small effects, explaining 3.5-12.1% of the observed phenotypic variance. These finding are in an agreement with previous genetic mapping studies (QTL and GWA) on sclerotinia resistance. This suggests and validates that SSR resistance in *B. napus* is a complex genetic trait, quantitatively inherited and determined by multiple minor QTL with small effects (Zhao and Meng, 2003; Zhao et al., 2006; Yin et al., 2010; Wu et al., 2013, 2016, 2019; Wei et al., 2014; Qasim et al., 2020). Li et al. (2015) conducted an integrated and comparative QTL analyses for SSR resistance using previously identified QTLs from various mapping studies with the *Darmor-bzh* reference genome (Chalhoub et al., 2014) and determined that chromosomes A9 (22.5–27.5 Mb) and C6 (29.5–36.1 Mb) are conserved QTL regions. The putative disease resistance nucleotide-binding-

site, leucine-rich-repeat (NBS-LRR) genes were found in this region in a cluster. GWA analyses from our study revealed a total of 14 significant SNPs located on C6 (22.3-39.4 Mb) genomic regions, and 5 of them were located on the chromosome C06 (33.2- 34.1 Mb) region that was reported by Li et al. (2015). However, the SNP markers on chromosome C06 identified in this study have shown overlapping confidence intervals with the QTLs for stem resistance detected by Zhao et al. (2006), Wu et al. (2013), Wei et al. (2016), Wu et al. (2016), and Qasim et al. (2020). Identification of the SNPs in our study which aligned with previously identified overlapping genomic regions provide strong evidence that fine-mapping using large segregating mapping population could help us to narrow down the genomic regions. This may lead us to identify the putative candidate gene conferring SSR resistance in rapeseed/canola, and therefore, guide us for the map-based cloning of the sclerotinia resistance gene in future to assist MAS.

Several QTLs reported here were localized in the vicinity of QTL identified by other researchers. GWA study conducted by Wu et al. (2016) identified five significant SNPs on chromosome A08 (15.09-15.10 Mb) region based on the alignment of *Darmor-bzh* (Chalhoub et al., 2014) reference genome. We detected six SNPs on chromosome A08 (13.7-22.9 Mb) region based on '*ZS11*' reference genome sequence (Sun et al., 2017). The '*ZS11*' reference genome sequence was aligned with '*Darmor-bzh'* reference genome (Chalhoub et al., 2014). Therefore, our finding was a close agreement with Wu et al. (2016). We also identified significant SNPs on chromosome C08 (SCM002776.2_29886188, SCM002776.2_37107013) which located near or overlapped with the identified QTL genomic regions of chromosome C8 (31.4-33.5 Mb, 38.2-38.5 Mb) reported by Wu et al. (2013). A significant SNP (SCM002777.2_46851981) for LL located on chromosome C09 repeatedly detected on multiple environments (CARR_19, CARR_20, OSN_20, CombENV) were found to be overlapped with the physical interval of

Sll19 for stem lesion length using petiole inoculation technique by Zhao et al. (2006). Moreover, another stable SNP marker (SCM002777.2_48885679) on chromosome C09 identified in almost all the environments and combENV with all the traits except PM_21D located at the physical position of 48.9 Mb in the '*ZS11*' reference genome sequence. Detection of these stable genomic regions (46.8-48.9) on chromosome C09 in our current study as well as from the previous study provide an exciting opportunity to further explore these regions to develop molecular markers for future MAS in the rapeseed/canola breeding program for SSR resistance. Shahoveisi et al. (2021) reported QTL SR54.C3.1 associated with SSR resistance in the physical region of (23.4-31.6 Mb) on chromosome C03, the two SNPs [SCM002771.2_22853068 (22.9 Mb), SCM002771.2_27877818 (27.9 Mb)] in our study was found in close proximity or within the genomic regions. Moreover, we collected information on the previously identified QTLs, SNPs and their physical positions based on marker information from the past studies and compared it with our findings. In addition to the identification of significant SNPs in the previously detected genomic regions, to best of our knowledge, hereafter we are reporting new genomic regions on chromosome A09 (35.6-45.8 Mb) consisting of ten significant SNP markers, chromosome A03 (28.2-36.2 Mb) with seven significant SNPs, and chromosome A05 (15.9-28.6 Mb) with six markers are associated with SSR resistance.

One of the key objectives of GWA study is the identification and utilization of the candidate genes. Thereby we searched for candidate genes associated with disease resistance mechanisms that were located within 50 kb upstream and downstream of significant SNPs detected in at least two environments. We choose 50 kb because LD for this population is low (< 45 kb genome wise, < 21 kb for A genome and < 93 kb for C genome (Rahman et al., 2021). Based on these criteria, sixty-nine genes associated with defense response mechanisms were

identified. A TIR-NB-LRR gene (LOC106415792) that putatively encodes *RPP1* proteins was located in the vicinity of SNP (SCM002777.2_48885679) detected in all the environments and combENV with stem LL, LW, PM_14D traits on chromosome C09. TIR-NB-LRR genes provide defense response against fungi through the activation of the salicylic acid (SA)-dependent resistance pathway (Michael Weaver et al., 2006). SA has been known to be involved in the activation of defense response against biotrophic and hemi-biotrophic pathogens. However, recent findings suggest *S. sclerotiorum* has a brief biotrophic phase followed by a necrotrophic phase (Kabbage et al., 2015; Chittem et al., 2020). Our findings are in agreement with Nováková et al. (2014). Two annotated candidate genes, *WRKY transcription factor 33* (*WRKY33*) and a peroxidase C3-like, were detected 7.1 kb upstream and 4.6 kb downstream of marker SCM002772.2_65359864 on chromosome C4. This marker was detected using stem LL and LW data sets in multiple environments. *WRKY33* is involved in defense response to fungus and the camalexin biosynthetic processes which was found to be involved in providing resistance against *S. sclerotiorum* (Stotz et al., 2011). Wang et al. (2014) demonstrated that overexpression of *BnWRKY33* markedly enhanced resistance to *S. sclerotiorum* in *B. napus*. Another candidate gene annotated peroxidase C3-like to be involved in defense response. The remaining sixty-six genes reported in this study also encode proteins involved in the disease resistance mechanisms according to TAIR 10 and Uniport-KB.

Identification of stable QTL/MTAs is a prerequisite for their use in a breeding program to facilitate MAS. In this study, thirty-three significant MTAs were found to be co-localized or in close proximity with the earlier bi-parental and GWA mapping studies reporting QTL/MTAs that could be exploited and integrated for SSR resistance into the breeding program (Table A7). To our knowledge, this is the first study that used three different GWA algorithms and four

phenotypic traits (stem LL, LW, PM_14D, and PM_21D) under field conditions to identify genomic regions associated with reaction to *S. sclerotiorum*. Further, this is the first time that comprehensive phenotypic evaluation of three physiological traits, days to flowering, stem diameter, and stem internode length, indicate these traits play an important indirect role for the selection of SSR resistance genotypes in the field. Out of one-hundred thirty-three significant SNPs, nineteen of them were detected in at least two environments by at least two GWA models and two phenotypic traits. Detection of stable and common MTAs with multiple traits, implementing multiple GWA models in multiple environments could provide more confidence and reliability on the reported MTAs, and new genomic regions for SSR resistance from our current study.

Genomic selection is an effective genomic approach for the improvement of complex traits in crops (Crossa et al., 2014, 2017; Würschum et al., 2014; Odilbekov et al., 2019). GP models with the environment-wise BLUEs of stem LL, and LW resulted 0.06-0.51 and 0.12-0.52 predictive abilities, respectively. Moreover, predictive abilities implementing three GS models *i.e.* rrBLUP, Bayes C and BRR for the combENV datasets for stem LL, LW, PM_14D, and PM_21D for SSR resistance were 0.41 to 0.3, 0.42-0.44, 0.47-0.49, and 0.63-0.64, respectively. These results clearly demonstrate that genome-wide markers are efficient in predicting SSR resistance. None of the models outperform than the others, with an exception for CARR_20 environment, consistent with results obtained by other researchers (Spindel et al., 2015; de Azevedo Peixoto et al., 2017; Derbyshire et al., 2021). The observed differences in the predictive abilities among the used models for the combENV traits were mostly 1 to 2 units, which were likely due to GS model's underlying assumptions. For example, rrBLUP model assumes that all the marker effects have identical variance and all markers effects have drawn from the same

gaussian/normal distribution (Heffner et al., 2011). Bayes C assumes a priori that markers have normally distributed effects with probability π and no effect with probability (1- π) (Meuwissen et al., 2017). BRR produces homogeneous shrinkage of all marker effects towards zero and yields a normal distribution of the marker effects (Desta and Ortiz, 2014). The consistent results were also reported by Derbyshire et al. (2021), where Bayesian models perform similar or worse than G-BLUP model to predict *S. sclerotiorum* resistance in *B. napus*. The predictive ability in this study ranging from medium to high, were comparable or higher than the estimated predictive ability of SSR resistance in two previous studies (Wei et al. 2016; Derbyshire et al., 2021). The differences in predictive ability could be attributed due to the difference in populations, diversity, linkage disequilibrium, and trait heritability (Crossa et al., 2017; Daetwyler et al., 2010; Isidro et al., 2015). However, the estimated predictive ability was more or less similar with the reported predictive ability by Derbyshire et al. (2021) when lesion length data was used as a target trait. Interestingly, in our current study, using PM_21D data we achieved 47-56% increase in predictive ability. The results suggest that use of PM_21D data rather than pathogen spreading (stem lesion length and lesion width) could be used as a useful phenotypic trait, which would potentially enable the breeders to achieve higher predictive ability and leading towards the selection of superior genotypes for SSR resistance breeding in rapeseed/canola. GWA mapping results from this study indicated that most of the significant SNPs explained only 3.6-12.1% of the phenotypic variance. However, most of the identified significant SNPs or QTL from the GWA and bi-parental linkage mapping studies also showed small effects, explaining less than 10% of the observed phenotypic variance (Zhao and Meng, 2003; Zhao et al., 2006; Yin et al., 2010; Wei et al., 2014; Wu et al., 2016). Thus, genomic selection offers promising opportunities by capturing the effects of both minor and major genes to exploit the full genetic potential over

selection based on few significant markers for the improvement of SSR resistance in rapeseed/canola. In future, this study could be further improved by including more genotypes, as well as integrating other independent biological information.

In this study, we have identified resistant genotypes from a genetically diverse resource will serve as a potential donor for improving canola cultivars with SSR resistance at North Dakota State University canola breeding program. The use of multiple phenotypic data sets and GWA models used on data collected in multiple field environments allowed for the detection of one-hundred thirty-three significant SNPs, some of them were in novel regions of the genome. Some SNPs were detected in multiple datasets and models, suggesting their association with the resistant trait may be stronger than that of others. At the same time, they validate the notion that multiple approaches, e.g., phenotypic data sets and GWA models, may yield additional information that otherwise would not be captured. The significant stable and new MTAs detected from this study could be used for future MAS of SSR resistance in rapeseed/canola breeding. Further, the strong and significant correlation detected among the phenotypic traits suggested that, stem LW, PM_14D, and PM_21D could be used as proxies for stem LL when evaluating genotypes for their reaction to SSR. This study also assessed the potential of GP using different GS models and revealed a medium to high predictive ability depending on various phenotypic traits. Our results suggest that GS holds promise for the improvement of SSR resistance, and its application would enable the breeders for early SSR resistance genotype selection to accelerate the breeding efficiency by reducing the need to phenotype large number of genotypes in the field at maturing stage.

CHAPTER 4: GENETIC MAPPING AND GENOMIC PREDICTION OF SCLEROTINIA STEM ROT RESISTANCE TO RAPESEED/CANOLA AT SEEDLING STAGE

4.1. Abstract

The complex resistance mechanisms between rapeseed/canola and *Sclerotinia sclerotiorum* limits the development of functional molecular markers and gene identification that enables sclerotinia stem rot (SSR) resistance breeding. However, genomic selection has the potential to accelerate the genetic improvement of SSR resistance. Therefore, genome-wide association (GWA) mapping and genomic prediction (GP) was performed using a diverse panel of 337 rapeseed/canola genotypes. Phenotyping was done twice by challenging 3-week old seedlings to SSR attack using petiole inoculation technique (PIT). Days to wilt (DW) upto 2 weeks and lesion phenotypes (LP) at 3, 4, and 7 days post inoculation (dpi) were recorded. Strong correlation (r = -0.94) between DW and LP_4dpi implied that single time point scoring LP_4dpi could be used as a proxy trait instead of multiple times collected DW trait for SSR evaluation. GWA analyses using single-locus (SL) and multi-locus (ML) models identified a total of 35, and 219 significantly associated SNPs, respectively. Out of these, seventy-one SNPs were identified by a combination of the SL model and any of the ML models, at least two ML models, or two traits. These SNPs explained 1.4-13.3% of the phenotypic variance, and considered as significant, could be associated with SSR resistance. Eighty-one putative candidate genes associated with disease resistance mechanisms corresponding to annotated significant SNPs were found to underlie SSR resistance. Six GP models resulted in moderate to high (0.45-0.68) predictive ability depending on SSR resistance traits. The resistant genotypes and significant SNPs identified in the present study will serve as valuable resources for future SSR

resistance rapeseed/canola breeding. Our results also highlight the potential of genomic selection towards the improvement of highly polygenic SSR resistance that could lead a step forward towards the genomics-assisted rapeseed/canola breeding.

Keywords: Sclerotinia stem rot, rapeseed/canola, genome-wide association (GWA) mapping, single nucleotide polymorphism (SNP), Quantitative trait nucleotides (QTNs), genomic prediction (GP)

4.2. Introduction

Rapeseed/canola (*Brassica napus* L., genomes=AACC, 2n=4x=38) is an amphidiploid Brassica species and the second largest cultivated oilseed crop in the world after soybean (USDA Foreign Agricultural Service, 2021). Sclerotinia stem rot (SSR), caused by the necrotrophic plant pathogenic fungus *Sclerotinia sclerotiorum* (Lib) de Bary, is one of the most economically important diseases affecting rapeseed/canola, that significantly limits worldwide rapeseed/canola production (Boland and Hall, 1994; Bolton et al., 2006). The yield losses due to this pathogen vary from 10-80% from year to year depending on the disease development environments (Del Río et al., 2007; Wu et al., 2016). However, in the United States, each unit increase in SSR incidence imposes 0.5-0.7% loss in canola seed yields (Del Río et al., 2007; Koch et al., 2007). Moreover, SSR affected plants often tend to have reduced oil content, and inferior oil quality due to the changing of oil's fatty acid profile (McCartney et al., 1999; Sharma et al., 2015).

To manage the associated risk of SSR disease, growers primarily depend on the use of conventional rotation with non-host crop species and chemical controls which are neither completely effective nor economically and environmentally feasible (Derbyshire and Denton-Giles, 2016; Roy et al., 2021). Therefore, breeding for durable SSR resistant varieties would be a more economically feasible, environment-friendly, and sustainable strategy to manage this

disease. However, no accessions conferring high level of resistance or complete immunity to S. sclerotiorum have been identified over the last three decades of investigation (Zhao et al., 2004; Bradley et al., 2006; Yin et al., 2010). Thus, the current breeding strategy for improved SSR resistance is solely dependent on the utilization of such partially resistant germplasm. Therefore, it is crucial to screen a worldwide collection of diverse genotypes with an appropriate screening method to identify genetically resistant genotypes to improve SSR resistance in rapeseed/canola. Several disease screening methods including petiole inoculation technique (PIT) (Zhao et al., 2004; Bradley et al., 2006), detached leaf inoculation (Zhao and Meng, 2003; Wu et al., 2013), and stem inoculation (Li et al., 2006; Wu et al., 2013; Wei et al., 2016; Qasim et al., 2020; Roy et al., 2021; Shahoveisi et al., 2021) have been used to evaluate the genetic resistance of rapeseed/canola germplasm at different developmental stages under controlled and field environments. Moreover, rapeseed/canola cultivars differ in their plant architecture, growth habits, and maturity (Bradley et al., 2006; Arifuzzaman and Rahman, 2020; Rahman et al., 2021). Cultivated canola express spring (no vernalization needed to induce flowering), semi-winter (shorter period of vernalization require to induce flowering), or winter (vernalization needed over the winter to induce flowering) growth habits (Wang et al., 2011; Arifuzzaman and Rahman, 2020; Rahman et al., 2021). In North Dakota, the leading canola producing state in the USA, only spring canola is cultivated due to the shorter growing season. Poor winter hardiness prevents the cultivation of semi-winter and winter ecotypes canola. It is difficult to use the stem inoculation method to screen all ecotypes of *B. napus* because of the challenge to synchronous inoculation time and the vernalization requirement. Therefore, an early growth stage inoculation procedure that is quick, efficient, and reliable screening method that utilizes to circumvent bolting, flowering, or vernalization issues. This would ultimately accelerate the screening

process and allow the simultaneous screening of genotypes with any of the three growth habit types. Here we implemented a PIT screening at four to five leaf stage seedlings to evaluate a diverse set of rapeseed/canola germplasm for resistance to *S. sclerotiorum*.

SSR resistance is a quantitatively inherited trait controlled by polygenes with minor additive and partially dominant effects, which are affected by the environment (Wei et al., 2016; Wu et al., 2016; Qasim et al., 2020; Roy et al., 2021; Derbyshire et al., 2021). Genetic mapping studies of SSR resistance through quantitative trait loci (QTL) analysis, based on classical linkage mapping strategy with bi-parental mapping populations, were commonly used for the purpose of identifying functional genes, and to position molecular DNA markers associated with SSR resistance. A majority of the SSR resistance QTL were located on chromosomes A01, A02, A03, A06, A7, A08, A09, C01, C02, C03, C04, C06, C08, and C09 (Zhao and Meng, 2003; Zhao et al., 2006; Yin et al., 2010; Wu et al., 2013; Wei et al., 2014; Behla et al., 2017; Qasim et al., 2020; Shahoveisi et al., 2021) using a PIT, and detached leaf and/or stem inoculation technique at various developmental stages. Despite these efforts, no major QTL or gene conferring resistance to SSR were fine mapped or cloned, which seriously limits the research into genetic manipulation of SSR disease resistance breeding. Moreover, bi-parental mapping population lacks allelic diversity and have a fewer number recombination events which limits mapping resolution (Korte and Farlow, 2013). Genome-wide association (GWA) mapping has emerged as a robust approach to dissect complex traits and identify novel and superior alleles by capturing the resistance diversity in a germplasm collection to be utilized in marker-assisted breeding. GWA mapping is based on the utilization of linkage disequilibrium (LD) within a diverse population of genotypes which have undergone extensive historical and evolutionary recombination events leading to the development of shortened LD segments. Abundant genetic

allelic diversity and faster LD decay provides more promising opportunities to achieve high mapping resolution for the significant SNP/marker-trait-associations (MTAs) than the traditional linkage mapping (Nordborg and Weigel, 2008).

To date, only a few GWA studies have been carried out to identify MTAs for mapping SSR resistance in rapeseed/canola (Gyawali et al., 2016; Wei et al., 2016; Wu et al., 2016; Roy et al., 2021). The screening procedure used in these studies were based on direct inoculation of mycelium to the main intact stem of the growing plant and/or to its detached stem during the flowering stage. Several investigations on SSR resistance reported a significant interaction and negative correlation between flowering time and stem resistance (Wu et al., 2019; Zhang et al., 2019; Roy et al., 2021). Therefore, we screened our association panel using a PIT method to evaluate the performance of the genotypes for SSR resistance to eliminate the conflicts between early maturation and SSR resistance in rapeseed/canola breeding. To the best of our knowledge, this report is the first GWA analysis to identify useful SNPs associated with SSR resistance in rapeseed/canola using the PIT disease screening method at the seedling stage.

One main issue with association mapping (AM) is the low power of detecting rare variants with small effects, which might be associated with economically important traits (Bernardo, 2016). Typically, individual SNPs/QTLs identified from GWA and bi-parental linkage mapping explain less than 12% of the phenotypic variance with some exceptions, which suggests uncaptured genetic potential for SSR resistance breeding in *B. napus* remains to be discovered. To obtain the maximum genetic potential for SSR resistance traits, genomic selection (GS) has emerged as a promising genomics-assisted breeding approach that account for both major and minor QTLs effects into prediction framework. GS utilizes the full genome information regardless of its significance, for genomic-enabled prediction of the superior

genotypes as a candidate for selection (Meuwissen et al., 2001; Crossa et al., 2017). GS combines genome-wide molecular markers and phenotypic data of a training population to develop a statistical model that predicts the breeding and/or genetic values of selection individuals/candidates which are only genotyped (Meuwissen et al., 2001; Crossa et al., 2017; Derbyshire et al., 2021). Previous GS studies for various agronomic traits, including blackleg and *S. sclerotiorum* disease resistance have shown the potential of GP to accelerate the rapeseed/canola breeding (Würschum et al., 2014; Fikere et al., 2020; Roy et al., 2021; Derbyshire et al., 2021). The predictive abilities on adult plant resistance against *S. sclerotiorum* in rapeseed/canola by Derbyshire et al. (2021) and Roy et al. (2021) clearly indicating the potential of GS for improving complex SSR resistance. This further motivated us to explore the effectiveness of GP in predicting SSR resistant genotypes using *B. napus* plants at the seedling stage.

In this work, we used a diverse panel of 337 rapeseed/canola lines with the aim to i) identify new sources of SSR resistant genotypes at the seedling stage; ii) detect significant genomic regions, SNPs associated with SSR resistance at the seedling stage by performing single-locus and multi-locus GWA models; and iii) to assess the potential of GP for seedling stage SSR resistance in rapeseed/canola.

4.3. Materials and methods

4.3.1. Germplasm collection

In all, 337 diverse *B. napus* germplasm accessions, and breeding lines with worldwide geographical origin of 23 countries were collected from the North Central Regional Plant Introduction Station (NCRPIS), Ames, Iowa, USA and North Dakota State University (Table A8). The plant materials consisted of spring, semi-winter, and winter ecotypes/growth habits of rapeseed/canola. The experiments were conducted in the Agricultural Experiment Station Research Greenhouse Complex, North Dakota State University, Fargo, ND, USA during 2019 and 2020. Plants were grown in the greenhouse at 22 ± 2 ℃ temperature with a 16-h photoperiod provided by natural sunlight supplemented with 400 W HPS PL 2000 lights (P.L. Light Systems Inc.).

4.3.2. Experimental design, inoculum preparation and *Sclerotinia sclerotiorum* disease phenotyping

The experiments were conducted twice using a randomized complete block design (RCBD) with three replications in each experiment. For each replicate, six individual plants were inoculated with *S. sclerotiorum* pathogen, which resulted in a total of 36 (6 plants x 3 replications x 2 experiments) plants being evaluated for each genotype. All genotypes were screened in batches with two commercially available spring canola hybrid cultivars, “Pioneer 45S51” and “Pioneer 45S56” as resistant checks, and the publicly available Canadian cultivar “Westar” as a susceptible check. Throughout the study, a highly virulent single isolate WM031 of *S. sclerotiorum* was used for all inoculations to rapeseed/canola (Roy et al., 2021; Shahoveisi et al., 2021). The inoculum was prepared by culturing the surface sterilized sclerotia of the isolate on autoclaved potato dextrose agar (PDA) medium (24 gL-1 potato dextrose broth and 15

gL-1 agar) at 22-24℃. Then mycelium plugs from the actively growing edges were sub-cultured on another PDA plates at room temperature for 48 hrs. Three weeks old 4-5 leaf stage seedlings were inoculated using the PIT method described by Zhao et al. (2004) with a few modifications. In brief, the petiole of the second fully expanded leaf of the seedling were excised 2.5 cm from the main stem using scissors. Then two mycelium plugs of sclerotinia isolates from the actively growing edges of growing mycelium were loaded into a sterilized 200 µl pipette tips by pushing the open end of the micropipette tip into the 48 hrs old culture plates. After that, the loaded tips were carefully pushed onto the severed petioles making sure the inner side of the agar plug flush with the top of the petiole tip (Figure 4.1a). Two separate disease scoring systems were used to classify the phenotypic response. The inoculated plant was observed for two weeks and the response of individual plants of each line was determined by days to wilt (DW). A plant was considered wilted when the infected main stem girdled completely or the leaves of the infected plant became irreversibly flaccid (Figure 4.1f). DW were recorded daily for the next two weeks starting on the third day after inoculation. In addition to DW, lesion phenotypes (LP) were scored in a 1-to-5 rating scale at 3, 4, and 7 days post inoculation (dpi) denoted as LP_3dpi, LP_4dpi, LP_7dpi. The phenotypic response was categorized according to Zhao et al. (2004) with following modifications: 1 = unaffected, no symptoms on the main stem; 2 = slightly affected, small size lesions (≤ 1.0 cm) at junction of petiole and stem, no water-soaked lesion, no wilt; 3 = moderately affected, small water-soaked lesions (1.0 to ≤ 2.0 cm), no wilt; 4 = severely affected, expanded and sunken water-soaked lesion (≥ 2.0 cm), no wilt; and 5 = dead, expanded, sunken, and water-soaked lesion resulting complete wilting or topple over of the infected plant (Figure 4.1b-f).

4.3.3. Phenotypic data analyses

Collected data on DW and LP on 3 (LP_3dpi), 4 (LP_4dpi), and 7 (LP_7dpi) data were subjected to analysis of variance (ANOVA) in SAS version 9.4 (SAS Institute, Cary, NC). Data from the both experiments were combined if the ratio of the effective error variance for each trait was less than 10-fold (Tabachnick and Fidell, 2000; Rahman et al., 2019; Arifuzzaman and Rahman, 2020; Roy et al., 2021). Best linear unbiased predictions (BLUPs) for all studied traits were used as the phenotypic values for the subsequent GWA analyses. BLUP estimation was calculated considering genotypes as random effects using the MIXED procedure (PROCMIXED) of SAS. Variance components were used to compute the broad-sense heritability (H2) for each trait as

$$H^2 = \frac{\sigma_g^2}{\sigma_g^2 + \frac{\sigma_{ge}^2}{n} + \frac{\sigma_e^2}{nr}}$$

where, σ^2_g, $\sigma^2_{ge,}$ and σ^2_e represent the genotype, genotype-by-experiment interaction, and residual error variances, respectively; *n* and *r* were the number of experiments, and replicates per experiments, respectively.

Pearson's correlation was conducted to examine the relationship between all traits using R (R Core Team, Vienna, Austria). The Shapiro-Wilk test was performed to verify the normal distributions of the collected data.

4.3.4. Genotyping data and quality control

Each member of the diversity panel was genotyped as described by Roy et al. (2021) and Rahman et al. (2021). In brief, total genomic DNA was extracted from fresh young and lyophilized leaf tissues using Qiagene DNeasy kit (Qiagen, CA, USA). The extracted DNA was quantified with a NanoDrop 2000/2000c Spectrophotometer (Thermo Fisher Scientific), and

diluted to 50 ng/µl. Genomic libraries were prepared using ApekI enzyme digestion described by Elshire et al. (2011). The library was sequenced as single-end reads at the University of Texas Southwestern Medical Center, Dallas, Texas, USA, using Illumina Hi-Seq 2500 sequencer. Bowtie 2 (Langmead and Salzberg, 2012) was used to align the single end sequencing reads against the "*ZS11*" reference genome sequence (Sun et al., 2017). TASSEL 5 GBSv2 pipeline (Glaubitz et al., 2014) was used to call the bi-allelic variant, which resulted in 497,336 unfiltered SNPs. Low-quality SNP markers were filtered with an individual read depth greater than 3, missing data less than 25%, minor allele frequency (MAF) greater than 5%, and physical distance (thin) less than 500 bp with VCFtools (Danecek et al., 2011). Since canola is a self-pollinated crop, SNPs that were more than 25% heterozygous were removed using TASSEL (Bradbury et al., 2007). After applying quality filtering, a total of 38,510 high quality SNPs was obtained. SNP loci with missing values were imputed in Beagle 5.1 (Browning et al., 2018).

4.3.5. Single-locus genome-wide association analyses

SNPs with less than 5% MAF were removed from a total of 38,510 markers, leaving 27, 282 high quality SNPs for subsequent GWA analyses. GEMMA software (version 0.98.1) (Zhou and Stephens, 2012) was used for single-locus (SL) GWA analyses using a mixed linear model (MLM). The first three principal components (PCA) calculated by prcomp () function in R (R Core Team, 2020) were embedded as covariates in the GWA analyses to control the confounding effect of population structure. Model-based clustering was performed using the first three PCAs to determine the subpopulations among the association panel with Mclust package in R. The GEMMA-MLM was executed with the following command in the GEMMA (version 0.98.1) software: "gemma -g [genotype data] -p [phenotype] -a [genotype annotation data] -c [first 3PCA] -k [centered relatedness matrix/kinship matrix] -o [output]". A kinship matrix was

incorporated as a random effect. The matrix was computed using the centered relatedness procedure in GEMMA. The significance threshold was determined using the method proposed by Li and Ji (2005) to determine the significant threshold value for the identified SNPs. In this method, for the 27, 282 SNPs we calculated the effective number of independent loci (Meff) by estimating correlation matrix and eigenvalue decomposition. The test criteria were then adjusted using the Meff with the following correction by Sidak (1967):

$$\alpha_p = (1 - \alpha_e)^{1/M_{eff}}$$

where, α_p is the comparison-wise error rate and α_e is the experiment-wise error rate ($\alpha_{e=0.05}$).

4.3.6. Multi-locus genome-wide association analyses

Multi-locus (ML) GWA analyses were implemented using three multi-locus GWA algorithms that includes MLMM (Segura et al., 2012), FarmCPU (Liu et al., 2016), and mrMLM (Wang et al., 2016). For the ML models, we selected the three PCA to control population genetic stratification that we used for GEMMA-MLM. The MLMM, and FarmCPU models were carried out using the GAPIT (version 3.0) R package (Wang and Zhang, 2021). The mrMLM GWA model was implemented using the R package "mrMLM" (Wang et al., 2016) with default parameters. The critical significant threshold between a trait and SNPs for all ML models were set to $P \leq 1.0$ x 10-3 [- $\log_{10}(P) \geq 3.0$], which has been broadly adopted by other researchers in various studies (Li et al., 2018; Xu et al., 2018; Karikari et al., 2020). The GWA results were visualized with Manhattan plot and comparative quantile-quantile (Q-Q) plots by plotting the observed *P* values against expected *P* values generated using the CMplot package in R language (https://github.com/YinLiLin/R-CMplot).

4.3.7. Candidate gene search

The significant MTAs identified in at least two traits or two or more GWA models (either SL or ML) were selected for potential candidate gene search that may be associated with disease resistance. Candidate genes were searched within the LD blocks, where associated significant SNPs were located, were regarded as the candidate gene search interval. If the detected SNPs were not located in the LD block, genomic regions spanning ± 50 kbp flanking regions of the significant MTAs were used as potential candidate gene interval in B. napus "*ZS11*" reference gene models (Sun et al., 2017). LD blocks analyses on the same chromosome were computed by Haploview v4.1 with the default settings (Barrett et al., 2005).

4.3.8. Genomic prediction

Genomic prediction was conducted implementing six GS models, *i.e.* rrBLUP, and five Bayesian models: Bayes A (BA), Bayes B (BB) (Meuwissen et al., 2001), Bayes C (BC) (Gianola et al., 2009), Bayesian LASSO (BL) (de los Campos et al., 2009), and Bayesian Ridge Regression (BRR) (Meuwissen et al., 2001). All models were performed in R language. The GP model rrBLUP was constructed using the package "rrBLUP" (Endelman, 2011) and BGLR (version 4.0.4) package was used to fit the Bayesian genomic prediction models (Pérez and de los Campos, 2014). All the analyses for Bayesian models were performed for 5,000 Monte Carlo Markov chain iterations with a 1,000 burn-iterations. All of GP models were constructed using the following formula:

$$y = \mu + X\beta + \varepsilon$$

Where, y is the vector of phenotypic values, μ is the intercept/grand mean, X is the standardized marker genotype matrix, β is the estimated random additive marker effects, and ε is the residual error term.

In brief, the rrBLUP model assumes that all the markers effects are normally distributed and all these marker effects have identical variance (Meuwissen et al., 2001). Whereas, Bayesian models may utilize different prior distributions which result in different levels of effect size shrinkage with various proportions of zero effect markers (Meuwissen et al., 2001; Habier et al., 2011). In BA, the effect of each marker is estimated from a gaussian distribution and markers are assumed to have different variances. The BB model is similar to BA, but allows some of the marker effects with zero variance. The BC model assumes a priori that markers have normally distributed effects with probability π and no effect with probability (1- π) (Meuwissen et al., 2017). The BL model applies both shrinkage and variable selection. The marker effects of the BL method are estimated from a double exponential distribution (de los Campos et al., 2009). The BRR creates equal shrinkage of all the marker effects towards zero and produces a Gaussian distribution of the marker effects (Desta and Ortiz, 2014).

The predictive ability of the GP models was tested with five-fold cross validation (with 270 individuals as training set and remaining 67 individuals as validation set in each fold) and replicated 100 times to avoid biases in the estimation. Predictive ability of each trait is calculated as the Pearson correlation (*r*) between the average of the predicted genomic estimated breeding values (GEBVs) and the observed phenotypes in all the cross-validation sets. Prediction accuracy is defined as the correlation between GEBVs and the true breeding values. The true breeding values of tested traits are not known, we approximated the prediction accuracy by the correlation between the GEBVs and the observed phenotypic values divided by the square root of the phenotypic heritability ($\sqrt{H^2}$) following Lorenz et al. (2011) and Jarquín et al. (2014). Thus, the accuracy of the models was estimated by dividing the mean of Pearson's *r* between GEBVs and

phenotype values from all cross-validations with 100 cycles by square root of broad-sense heritability (*H*).

4.4. Results

4.4.1. Phenotypic variations for *Sclerotinia sclerotiorum* reactions and correlation among phenotypic traits

SSR disease reactions can be variable under field environments, therefore, phenotyping the collection of germplasm against *S. sclerotiorum* was performed in the greenhouse under a controlled environment. A continuous and broad range of phenotypic variations were observed for days to wilt (DW) and lesion phenotypes (LP) traits among the genotypes in the study (Figure 4.1a-f, Figure 4.2a-d; Table 4.1). The BLUP values for DW varied from 3.5 to 9.9 days with an overall mean of 5.4 days and standard deviation (SD) of 0.87 (coefficient of variation is 30.9%). The variations observed for LP scores at 3, 4, and 7 dpi, ranged (mean ± SD) from 2.0 to 4.3 (2.8 ± 0.50), 2.6 to 4.8 (3.8 ± 0.45), and 3.7 to 5.0 (4.8 ± 0.16), respectively. The coefficient of variation (CV) of LP scores of the association population at different days varied from 6.7 to 19.2% (Table 4.1). Based on the phenotypic data, a few genotypes, which performed better level the resistant check cultivars used in this study, were identified as promising sources of resistance to SSR at the seedling stage. The BLUP values of the top five promising resistant genotypes ranged from 7.1 to 9.9 for DW, 2.0 to 2.2 for LP_3dpi, 2.6 to 2.8 for LP_4dpi, and 3.7 to 4.4 for LP_7dpi. However, the observed phenotypic responses of the resistant checks ‘Pioneer 45S51’ were 4.7, 3.3, 4.2, and 4.9 for DW, LP_3dpi, LP_4dpi, and LP_7dpi, respectively, and ‘Pioneer 45S56’ were 5.3, 2.9, 3.8, and 4.9 for DW, LP_3dpi, LP_4dpi, and LP_7dpi, respectively. The phenotypic response of susceptible check ‘Westar’ cultivar was 3.5, 4.3, 4.8, and 5.0 for DW, LP_3dpi, LP_4dpi, and LP_7dpi, respectively (Table 4.1). Analysis of variance (ANOVA) for

SSR reaction in terms of DW and LP scores on different days revealed significant differences ($P \leq 0.001$) among the genotypes, and interaction of genotype by experiment with an exception for LP at 7 dpi (interaction non-significant) (Table 4.2). Highly significant correlations were observed among the phenotypic traits for SSR reaction. For instance, significant negative associations were found for DW with LP_3dpi (r = -0.84), LP_4dpi (r = -0.94), and LP_7dpi (r = -0.87) at $P \leq 0.001$ (Figure 4.3).

The estimated broad-sense heritability of SSR resistance on entry mean basis across the two experiments were 0.71, 0.69, 0.70, 0.62 for DW, LP_3dpi, LP_4dpi, and LP_7dpi, respectively (Table 4.1). Medium to high heritability for SSR resistance in the phenotypic traits indicated that the phenotypic data was suitable for further genetic analyses.

Table 4.1. Phenotypic variation obtained through BLUP values in the response of *Brassica napus* genotypes against sclerotinia stem rot

Traits[a]	Min	Mean	Max	CV[b]	Shapiro–Wilk test *p* value	$H^{2\,c}$	Check cultivars (mean)[c]		
							Pioneer 45S51	Pioneer 45S56	Westar
DW	3.5	5.4	9.9	30.9	$3.5e10^{-7}$	0.71	4.7	5.3	3.5
LP_3dpi	2.0	2.8	4.3	19.2	$1.1e10^{-12}$	0.69	3.3	2.9	4.3
LP_4dpi	2.6	3.8	4.8	12.2	0.003	0.70	4.2	3.8	4.8
LP_7dpi	3.7	4.8	5.0	6.7	$2.2e10^{-16}$	0.62	4.9	4.9	5.0

Traits[a]: DW, days to wilt; LP, lesion phenotypes measured in 1 to 5 categorical scale at 3 (LP_3dpi), 4 (LP_4dpi), and 7 (LP_7dpi) days post inoculation (dpi); CV[b], coefficient of variation; Check cultivars (mean)[c], mean phenotype scores of the resistant checks (Pioneer 45S51 and Pioneer 45S56) and susceptible check (Westar) cultivars of the evaluated traits; $H^{2\,c}$ (Broad sense heritability)

Figure 4.1. Differential responses of *Brassica napus* genotypes against *Sclerotinia sclerotiorum* attack. a) Inoculated plant; b) unaffected plant [Lesion phenotype (LP) score 1]; c) slightly affected plant (LP score 2); d) moderately affected plant (LP score 3); e) severely affected plant (LP score 4); f) wilted or dead plant (LP score 5).

Table 4.2. Combined analysis of variance (ANOVA) of sclerotinia stem rot resistance trait in canola/rapeseed genotypes (including check cultivars)

Source of variation	Traits[a]			
	DW	LP_3dpi	LP_4dpi	LP_7dpi
Genotype	***	***	***	***
Genotype x Experiment	***	**	***	ns

, *, and ns indicate significance at $P \leq 0.01$, $P \leq 0.001$, and not significant, respectively. Traits[a]: DW, days to wilt; LP, lesion phenotypes measured in 1 to 5 categorical scale at 3 (LP_3dpi), 4 (LP_4dpi), and 7 (LP_7dpi) days post inoculation (dpi).

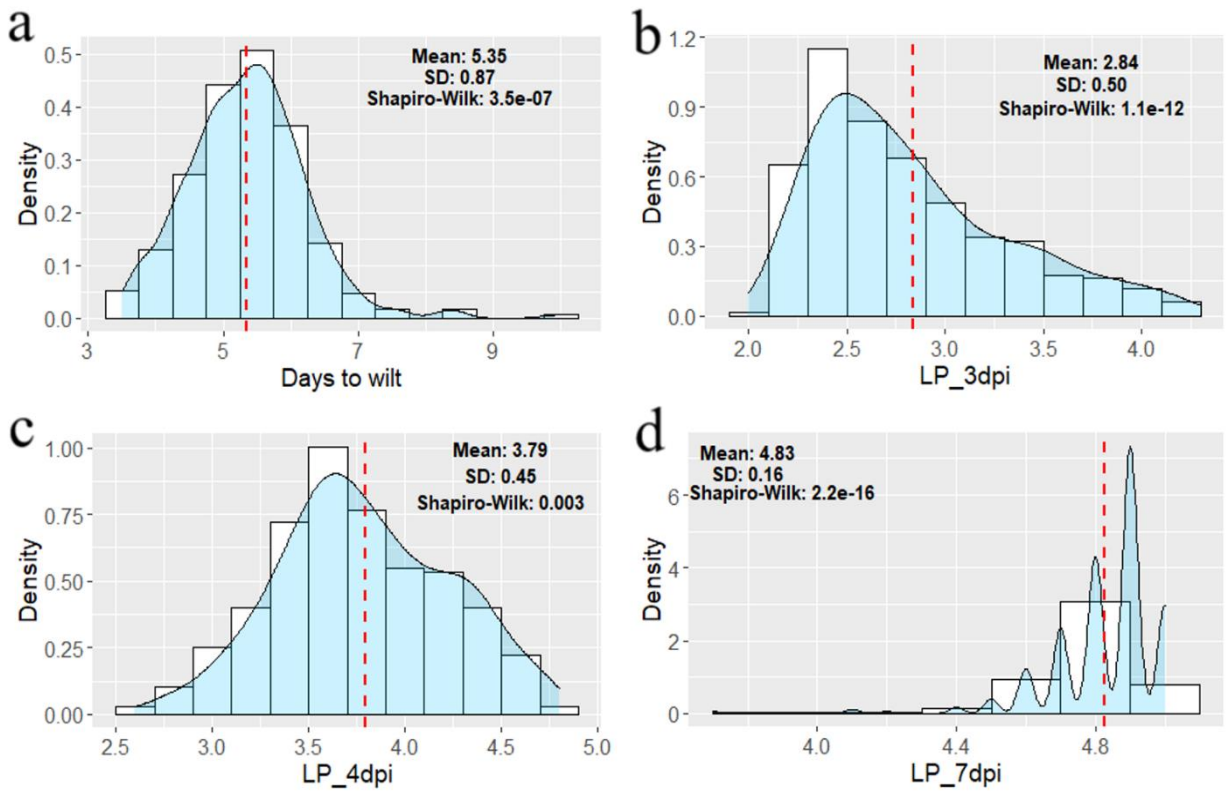

Figure 4.2. Distribution of phenotypic BLUP values of evaluated rapeseed/canola lines including check cultivars for *Sclerotinia sclerotiorum* resistance in terms of a) days to wilt b) lesion phenotype at 3 days post inoculation (LP_3dpi); c) lesion phenotype scores at 4dpi (LP_4dpi); and d) lesion phenotype scores at 7dpi (LP_7dpi). Mean values are presented by the dotted vertical lines; SD represents standard deviation; Shapiro–Wilk represents the probability values of the test for normal distribution.

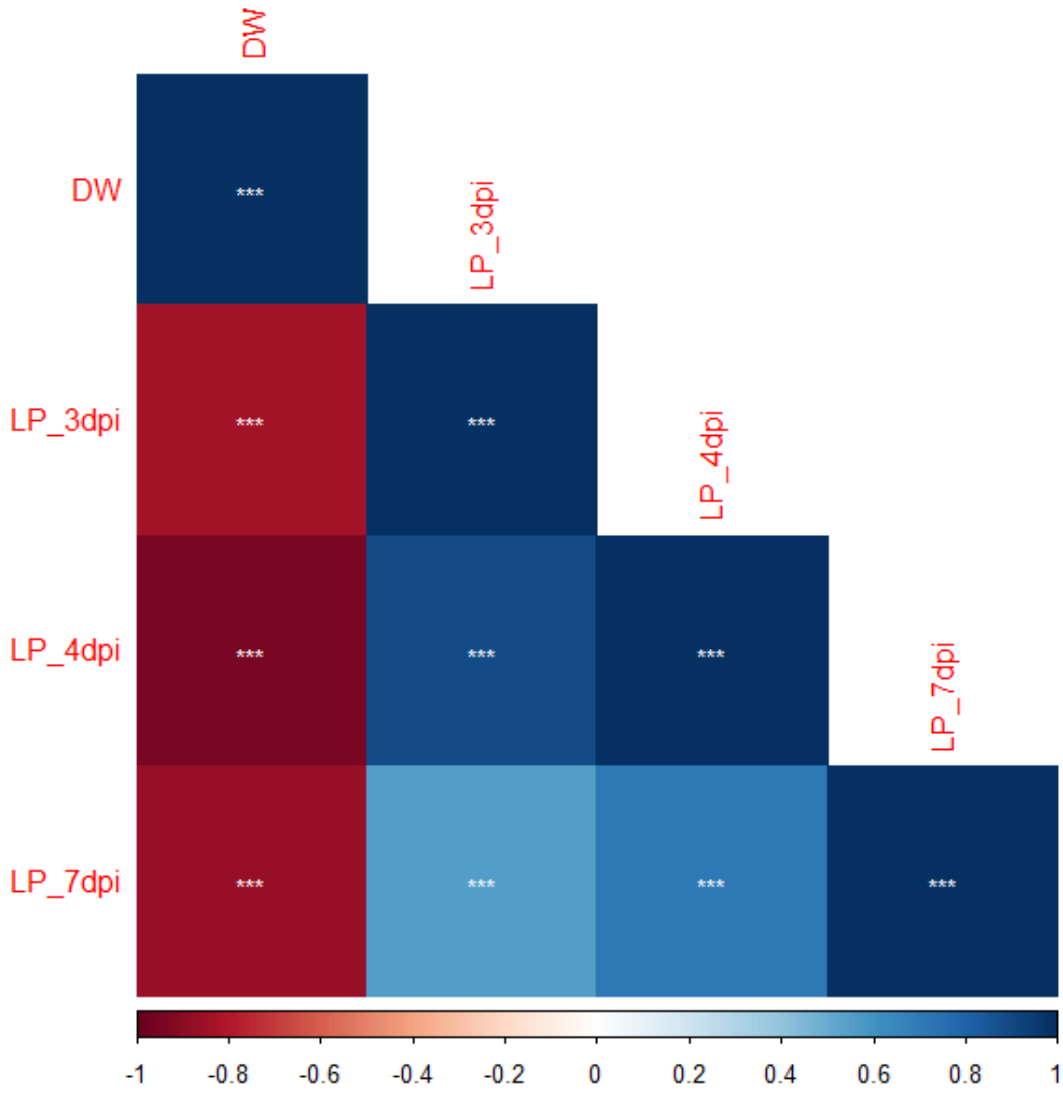

Figure 4.3. Correlation heatmap of various *Sclerotinia sclerotiorum* resistance phenotypic traits. Traits: DW= Days to wilt; LP_3dpi= lesion phenotype scores at 3 days post inoculation (dpi); LP_4dpi= lesion phenotype scores at 4dpi; LP_7dpi= lesion phenotype scores at 7dpi. *** indicate significance at $P \leq 0.001$ level.

4.4.2. SNP distribution and population structure analysis

After quality filtering and removal of markers with MAF < 5%, a total of 27, 282 high quality SNPs were used in the current study. These SNPs span a length of 854.3 Mb genome sequence representing 75.6% coverage of the *B. napus* genome (~ 1130 Mb). The number of SNPs were uneven among the 19 chromosome and ranged from 714-2386 SNPs per

chromosome with the average SNP per chromosome was 1436, where the chromosome 4 and 13 having the lowest (714 SNPs) and highest (2386 SNPs), respectively, while the average SNP per chromosome was 1436. The mean SNP density was approximately one SNP per 31.3 kb (Figure 4.4a). Based on the 27, 282 markers, principal component analysis (PCA) and kinship analyses were performed to identify the underlying genetic differences of the genotypes. The first three PCA explained 22.2% of the genotypic variation and were included in the GWA mapping model to control the confounding effect of population stratification. Furthermore, model-based clustering analysis using the first three PCA identified five subgroups within the genotypes based on three ecotypes (Figure 4.4b).

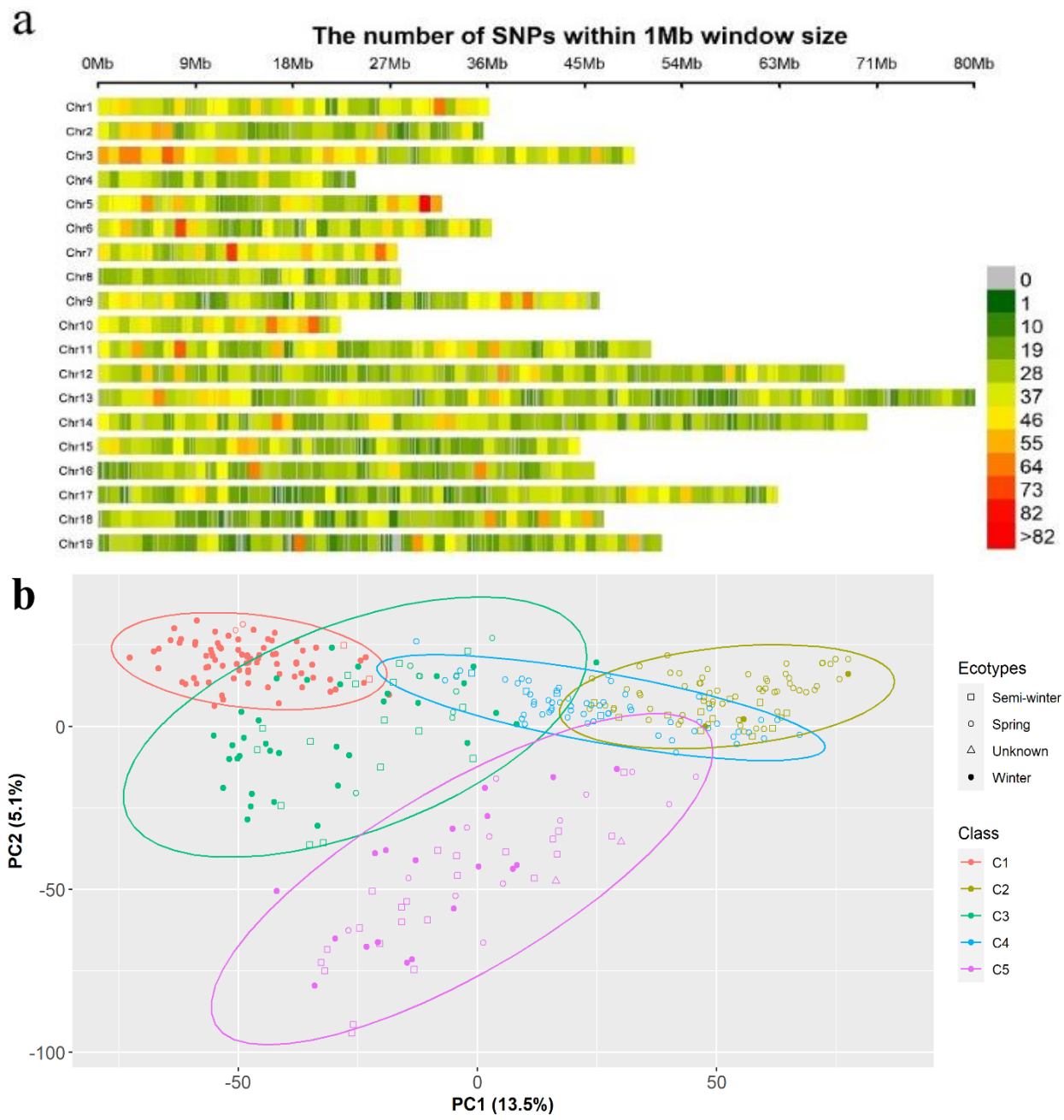

Figure 4.4. Single nucleotide polymorphism (SNP) density and distribution across the 19 chromosomes (a); and population structure as reflected by the scatter plot of PC1 and PC2 derived from a principal component analysis (b) of 337 rapeseed/canola genotypes.

4.4.3. Marker-trait-association detected for SSR resistance by single-locus GWA analyses

The single-locus (SL) GWA analyses, was performed with the GEMMA-MLM model that included the first three PCs as fixed effect and genetic relatedness matrix as random effect. The SL GWA results for DW and LP scores at 3, 4, and 7 dpi are presented in Table A9. Based

on the method developed by Li and Ji (2005), the significance threshold was $P \leq 2.40E-04$; LOD ≥ 3. A total of 35 SNPs were identified for the SSR resistance phenotypic traits. The SNPs were detected on chromosomes A01, A03, A04, A05, A06, A08, A09, C01, C02, C03, C04, C05, C06, C08, and C09. The majority of the significant SNPs were located on chromosomes C08 (5), A09 (4), A04 (3), A05 (3), A06(3), C02 (3), and C03 (3). The highest (n=15) number of significant SNPs were identified for DW while the lowest (n=11) number of SNPs were for the LP_3dpi SSR trait. Among these, 18 significant SNPs were detected for two or more of the SSR resistance traits (Table A9, A10).

4.4.4. Marker-trait-associations detected for SSR resistance by multi-locus GWA analyses

Three multi-locus (ML) GWA algorithms: MLMM, FarmCPU, and mrMLM detected a total of 219 SNPs corresponding to 216 loci across all the 19 chromosomes of *B. napus* genome [-log10 (P) =3.0-12.3] (Table A9). The number of SNPs detection by the three ML-GWA methods ranged from 10-48. The highest number of 48 SNPs were detected for DW trait by FarmCPU whereas the lowest number of 10 SNPs were found to be associated for LP_7dpi by mrMLM method. A total of 44 out of 219 SNPs were identified simultaneously in at least two phenotyped SSR resistance traits by two or more ML methods for any of the trait. The estimated allelic effects ranged between -0.54 to 0.63, -0.29 to 0.27, -0.21 to 0.19, and -0.14 to 0.12 for DW, LP_3dpi, LP_4dpi, and LP_7dpi traits, respectively. The explained phenotypic variation accounted for by the significant SNPs ranged from 2.0-9.30%, 1.60-11.90%, 1.35-13.30%, and 2.48-9.52% for DW, LP_3dpi, LP_4dpi, and LP_7dpi traits, respectively (Table A9, A10).

4.4.5. Commonly identified marker-trait-associations among the SSR resistance traits, among and between single-locus and multi-locus GWA studies methods

Of the 35 detected QTNs by SL-GWA methods, 18 were also associated with two or more SSR resistance traits. GEMMA-MLM detected a maximum of 15 SNPs for DW, 14 SNPs for both LP_4dpi and LP_7dpi respectively and 11 SNPs for LP_3dpi traits (Figure A10). Seven SNPs were mutually identified between DW and LP_4dpi, DW and LP_7dpi; followed by 5 SNPs between LP_3dpi and LP_4dpi trait, and only single SNP between DW and LP_3dpi (Table A9, A10). Moreover, only a single, SNP SCM002771.2_77997199, on chromosome C03 were co-localized by the SL methods for DW, LP_3dpi, and LP_4dpi traits. All of the QTNs detected with the SL methods were also associated with the four SSR resistance traits by any of the ML-GWA models. In addition to the 35 QTNs identified by SL, GWA analyses by ML-methods detected additional 184 SNPs associated with SSR phenotypic traits. The number of identified QTNs by all the ML models for SSR resistance traits varied between 54-88 whereas the number of QTNs for each of the ML models ranged between 10-48. The highest (48) number of QTNs were detected by FarmCPU (DW), and the lowest (10) QTNs by mrMLM model out of the three ML models for LP_7dpi (Figure A7). Comparison of the three ML models demonstrated that each model has the power to detect QTNs concurrently from each other and a few QTNs (ranged 1 to 9) were detected by all the three models for each trait. However, no common SNPs were identified by all three ML models with all SSR resistance traits. The number of commonly detected SNPs varied between and among the studied SSR resistance phenotypic traits: DW & LP_4dpi (20) > LP_3dpi & LP_4dpi (14) > DW & LP_7dpi (13) > DW & LP_3dpi (8) > DW, LP_3dpi & LP_4dpi (5) > LP_4dpi & LP_7dpi (3) > DW, LP_4dpi & LP_7dpi (2) (Table A9, A10). However, to obtain more reliable results, only the SNPs that were

simultaneously detected by both SL and any of the ML methods or at least two of the ML methods or at least two traits were considered as significant QTNs. Thus, a total of 71 QTNs controlling SSR resistance traits were obtained (Table A10). These QTNs will serve as a valuable source and could provide promising opportunities to facilitate MAS breeding for SSR resistance. Manhattan and Q-Q plots summarizing the GWA results of all the phenotypic traits for SSR resistance by SL (GEMMA-MLM) and ML (MLMM, FarmCPU, mrMLM) algorithms were present in Figure 4.5a-b, Figure 4.6a-b, and Figure A11, A12, A13, and A14. All GWA models were compared with the studied phenotypic traits to determine if the models control false positives and false negatives. The Q-Q plot depicts the expected negative $\log_{10}$ (*P*) values versus the expected negative log10 (*P*) values across all markers. Q-Q plots of models including GEMMA-MLM, and MLMM had a straight line with slightly deviated tail, which indicated that these two models reduced false positives (Figure 4.5a-b, Figure 4.6a). However, most of SNPs were close to the straight line or little bit inflates downward, indicating that they might have been reported as false negatives (Figure 4.5a-b, Figure 4.6a). In contrast, examination of Q-Q plots of FarmCPU, and mrMLM models showed a sharp upward deviation from the expected *P* value distribution in the tail area, indicating these models controlled both false positives and false negatives (Figure 4.5a-b, Figure 4.6a).

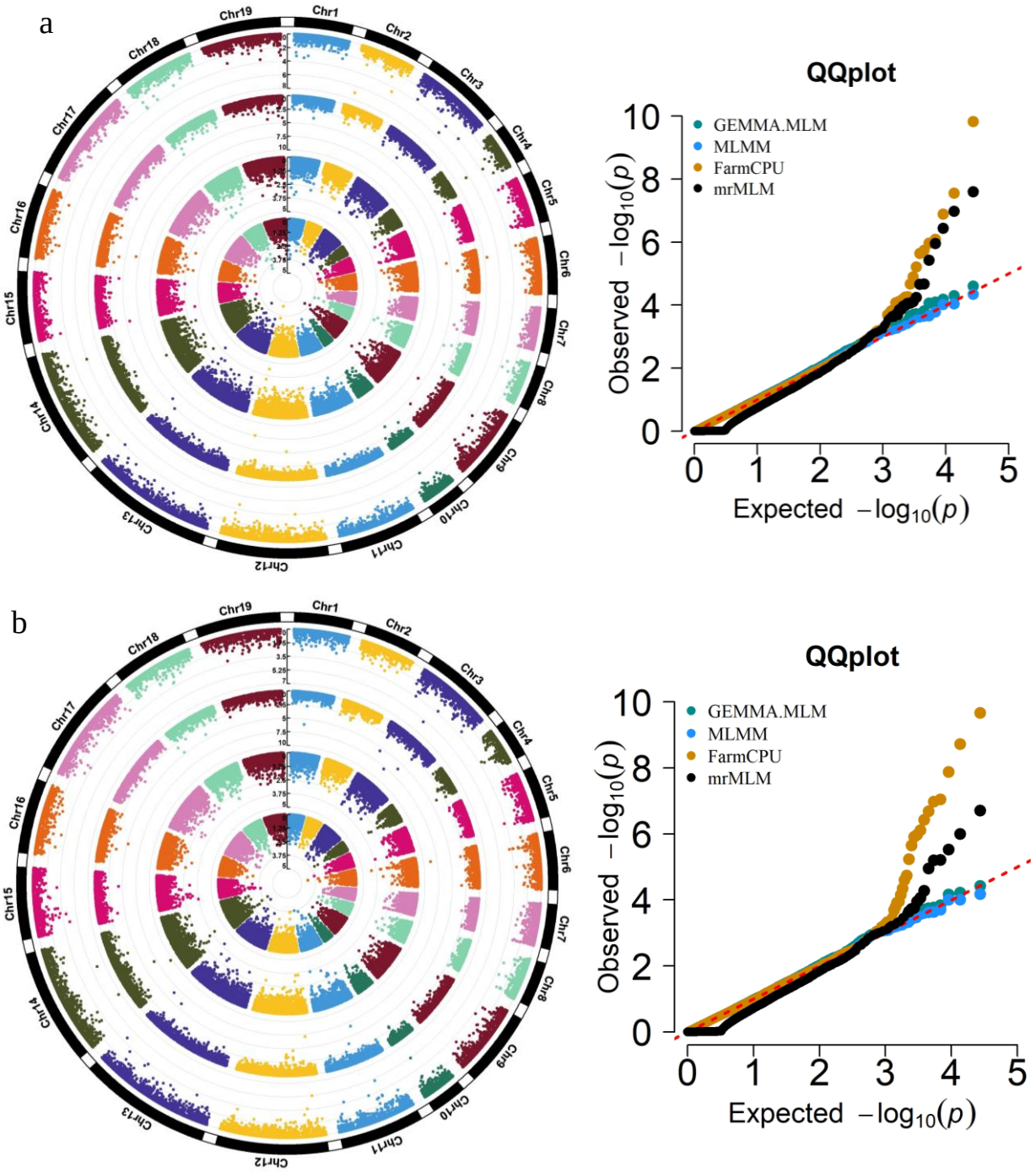

Figure 4.5. Circular Manhattan plots showing statistically significant SNPs based on single-locus (SL) GEMMA-MLM, and three multi-locus (ML) MLMM, FarmCPU, and mrMLM models located on 19 chromosomes for *Sclerotinia sclerotiorum* resistance at the seedling stage. Associations for the days to wilt (a), and lesion phenotype (LP) scores at 3 days post inoculation (dpi) (b) were shown. A multi-track Q-Q plot for each trait with all the GWA models are presented to the right of each Manhattan plot. The threshold values for SL and ML models were set up at $-\log_{10}(P) \geq 3.6$ and $-\log_{10}(P) \geq 3.0$, respectively.

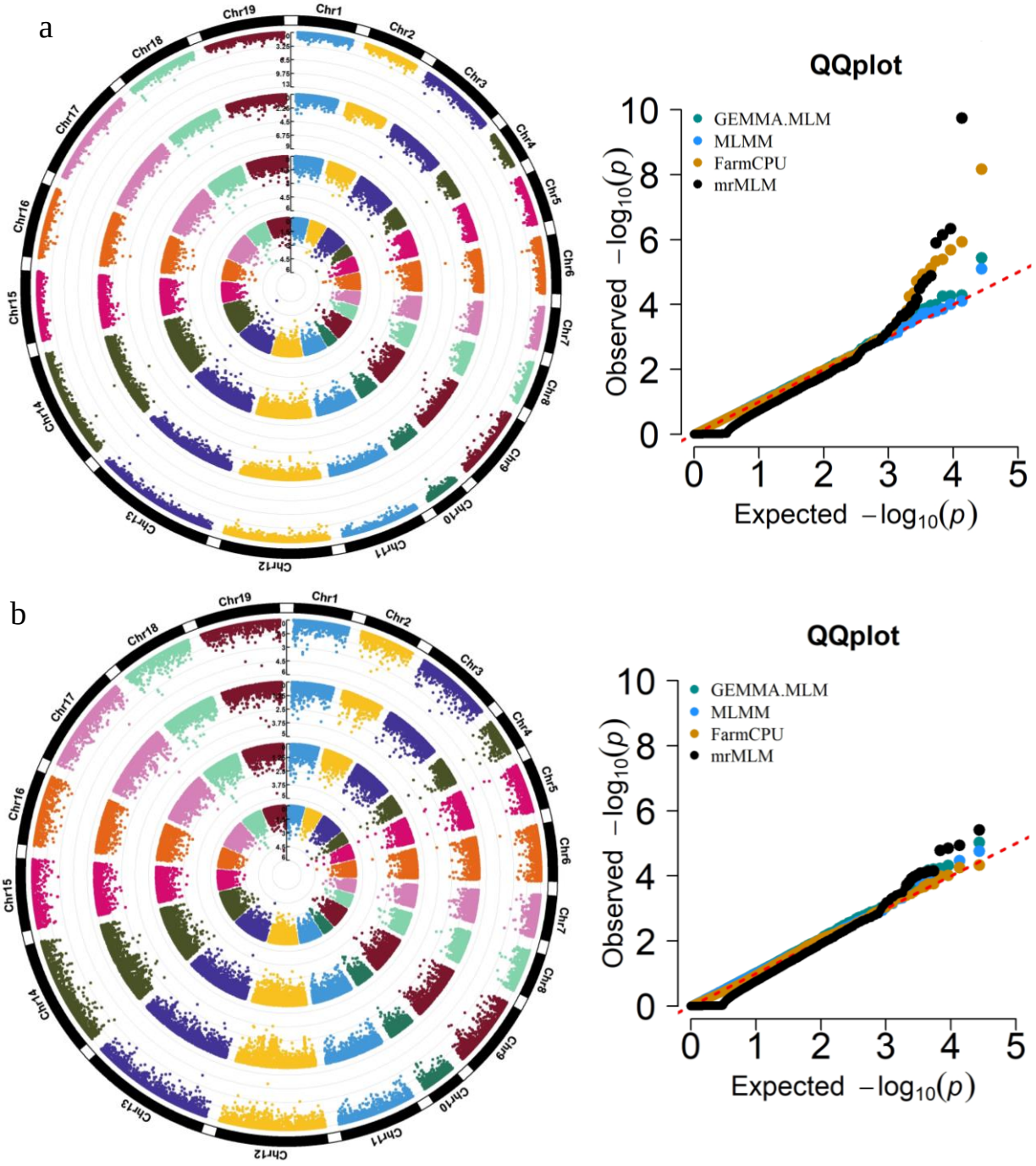

Figure 4.6. Circular Manhattan plots showing statistically significant SNPs based on single-locus (SL) GEMMA-MLM, and three multi-locus (ML) MLMM, FarmCPU, and mrMLM models located on 19 chromosomes for *Sclerotinia sclerotiorum* resistance at the seedling stage. Associations for the lesion phenotype (LP) scores at 4dpi (a), and LP scores at 7dpi (b) were shown. A multi-track Q-Q plot for each trait with all the GWA models are presented to the right of each Manhattan plot. The threshold values for SL and ML models were set up at $-\log_{10}(P) \geq 3.6$ and $-\log_{10}(P) \geq 3.0$, respectively.

4.4.6. Candidate gene prediction

To identify the potential candidate genes for the SSR resistance, the significant SNPs detected in at least two traits or with two or more GWA models were used for candidate gene mining using the "*ZS11*" reference genome sequence database (Sun et al., 2017). With this criterion, 81 putative candidate genes with known functions associated with plant disease resistance mechanisms were identified. Candidate gene protein were used as a query against the Uniport database (https://www.uniprot.org/uniprot/) to discover a putative biological function (Table A11). The biological processes of the detected candidate genes were involved in defense response, defense response to fungus, response to a molecule of fungal origin, response to chitin, programmed cell death, callose deposition in cell wall, response to salicylic acid, indole glucosinolate biosynthetic process, induced systemic resistance, jasmonic acid mediated signaling pathway, ethylene-dependent systemic resistance, systemic acquired resistance, pattern recognition receptor signaling pathway, response to wounding, protein kinase activity, response to oxidative stress, toxin catabolic process, immune response, reactive oxygen species metabolic process, brassinosteroid mediated signaling pathway and other biological processes which might play a key role in early stage SSR resistance in rapeseed/canola (Table A11).

4.4.7. Genomic prediction (GP)

The mean predictive ability and prediction accuracy of six GS models are shown in Figure 4.7a-d. There was a little difference in the predictive ability among the six GS statistical models for all the analyzed SSR resistance traits. The average predictive ability *i.e.* the correlation between observed and predicted resistance to SSR (*i.e.* GEBVs) were 0.60-0.62 for DW; 0.67-0.68 for LP_3dpi; 0.63 for LP_4dpi; and 0.45-0.48 for LP_7dpi (Figure 4.7a-d). In order to approximate the prediction accuracy, the mean predictive ability obtained from the

cross-validation sets were divided by $\sqrt{H^2}$. Therefore, the prediction accuracy estimates for DW, LP_3dpi, LP_4dpi, and LP_7dpi were, 0.71-0.73, 0.81-0.82, 0.75-0.76, and 0.57-0.60, respectively (Figure 4.7a-d). The highest genomic predictions explained ~ 67-68% of the variation was observed in LP_3dpi traits, whereas the lowest ~ 45-48% explained variation was recorded in LP_7dpi trait. Predictive abilities estimated from the various models had 0 to 3-unit differences depending on the traits. These slight differences are likely due to variations in genetic architecture and underlying model's assumptions. No model consistently resulted higher predictive ability across the traits. For example, in case of LP_3dpi, 0.68 predictive ability were recorded from rrBLUP, whereas all the Bayes models yielded a 0.67 predictive ability. However, the predictive ability for the LP_7dpi trait was 0.45 by rrBLUP and BL method, which was 3 units lower than the BB, and BRR model's estimation of 0.48. For LP_4dpi, all the implemented GS models resulted ~0.63 predictive ability. Therefore, slightly observed differences between the predictive ability could not be a criterion for selecting best GS model for SSR resistance prediction.

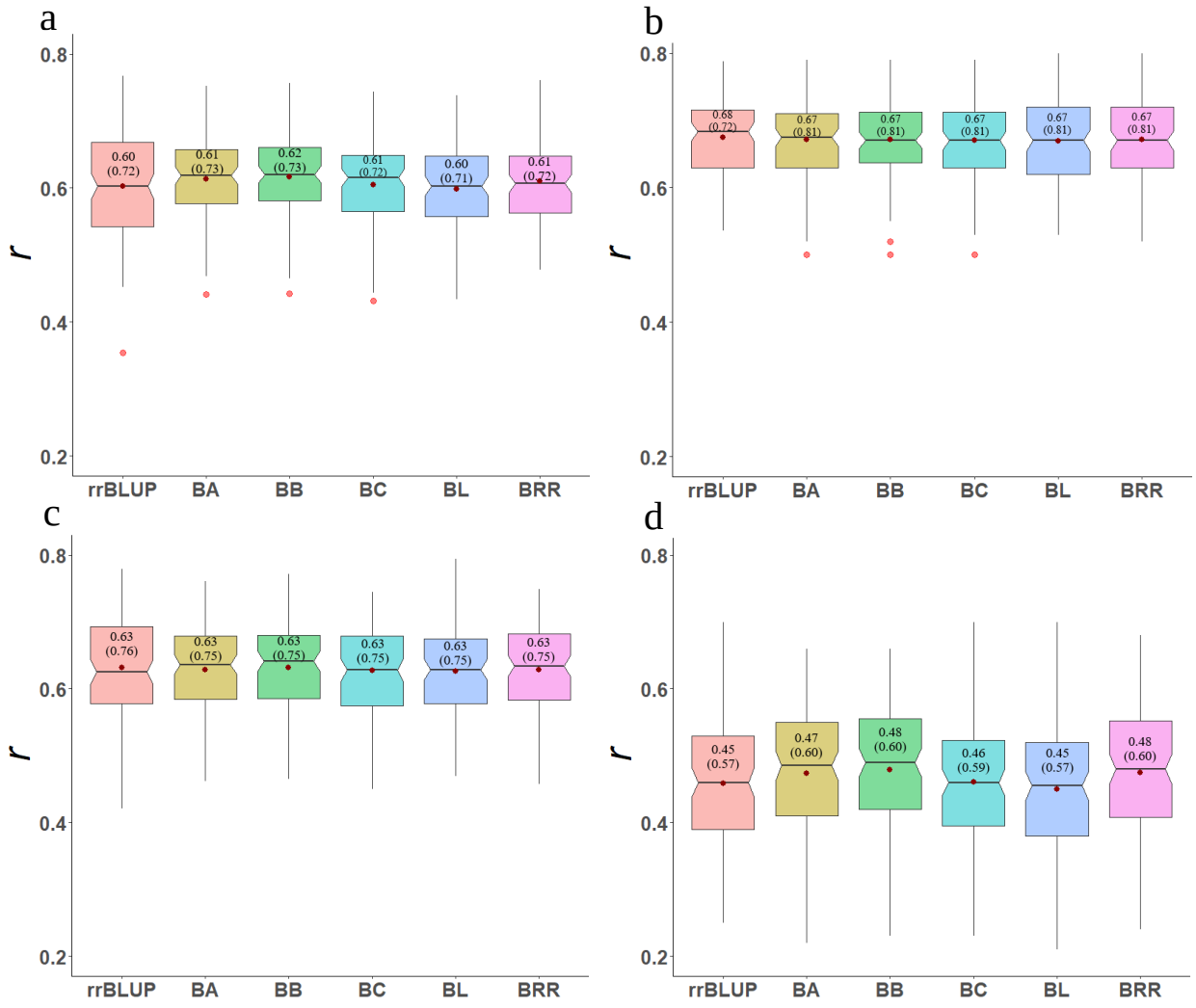

Figure 4.7. Predictive ability and accuracy of six genomic selection models, rrBLUP, Bayes A (BA), Bayes B (BB), Bayes C (BC), Bayesian Lasso (BL) and Bayesisan ridge regression (BRR) to detect sclerotinia stem rot resistant genotypes using phenotypic data of days to wilt (a), lesion phenotypes at 3 days post inoculation (b), lesion phenotypes at 4 days post inoculation (c), and lesion phenotypes at 7 days post inoculation (d) obtained from canola/rapeseed plants inoculated at the seedling stage with *S. sclerotiorum* using the petiole inoculation method. Boxplots show second and third quartiles and wishkers show interquertile range of predictive ability (*r*). The red dot in each box plot represents the mean predictive ability. The numbers above horizontal black bars represent the predictive ability (*r*) at the top and prediction accuracy in brackets.

4.5. Discussion

Sclerotinia sclerotiorum is a cosmopolitan fungal pathogen that causes significant seed yield loss, reduced oil content, and quality in the major rapeseed/canola growing regions including North America, Europe, Australia, India, and China (Barbetti et al., 2013; Wu et al.,

2016). The lack of a genotype with complete genetic resistance and useful molecular markers to select for SAR resistance limits MAS breeding towards development of durable SSR resistant cultivars for the growers to mitigate the economic loss associated with this pathogen attack. Therefore, it is necessary to identify stable resistance sources to the pathogen and to discover useful molecular markers significantly associated with SSR resistance to facilitate the SSR resistance MAS breeding.

A variety of artificial inoculation methods were used to screen *B. napus* genotypes against *S. sclerotiorum*. However, there is no single, and widely accepted method to evaluate SSR resistance. The stem inoculation method was previously used to evaluate the SSR resistance at the flowering stage. However, rapeseed/canola genotypes express spring, semi-winter, and winter growth habits while differing in plant architecture, stem diameter, stem internode length, and most importantly, flowering time (Li et al., 2006; Wu et al., 2019; Zhang et al., 2019; Qasim et al., 2020; Roy et al., 2021). Previous studies found that early flowering genotypes were prone to S. sclerotiorum infection compared to the late flowering genotypes. In addition to flowering time, plants smaller in stem diameter and longer in stem internode length were also found to be vulnerable to S. sclerotiorum infection (Li et al., 2006; Qasim et al., 2020; Roy et al., 2021). Therefore, it is necessary to implement an inoculation method which allows to evaluate all three ecotypes rapeseed/canola genotypes against *S. sclerotiorum* infection without being affected by any associated architectural or physiological traits. Thus the current study was designed to phenotype *B. napus* diversity panel at the same growth stages against *S. sclerotiorum* using petiole inoculation technique (PIT) by inoculating 3-weeks old seedlings. The PIT inoculation method was used by other researchers to successfully differentiate the resistant and susceptible lines/cultivars under the controlled and field environments. Zhao et al. (2004) successfully

implemented PIT for the evaluation of resistance of 47 *B. napus* seedlings to *S. sclerotiorum* under greenhouse conditions. Another study carried out by Bradley et al. (2006) screened 19 canola cultivars and found that PIT could differentiate resistant and susceptible canola cultivars for their reaction to *S. sclerotiorum*. The PIT inoculation method was also used by Zhao et al. (2006) and Behla et al. (2017) to identify QTL for resistance to SSR using DH populations in *B. napus*.

In the present study, 337 rapeseed/canola genotypes were evaluated for DW and LP at 3, 4, and 7dpi to identify the potential SSR resistant genotypes. The LP scores were recorded at different time points to distinguish differences in disease progress among the genotypes and to identify a single time point for phenotypic scoring that correlates with the commonly used DW data for SSR phenotyping. QTL mapping studies conducted by Zhao et al. (2006) used DW and stem lesion length data at 4 dpi, whereas Behla et al. (2017) used only DW. The use of multiple phenotyping scoring systems would provide valuable insights to accurately evaluate the resistance performance of the genotypes and detect additional QTNs associated with disease resistance (Roy et al., 2021; Shahoveisi et al., 2021). Furthermore, the evaluation of extensive phenotyping with multiple scoring systems would enable researchers to select a single time point to score disease phenotype. Based on our phenotyping screening results, a wide range of phenotypic variability were recorded in response to the *S. sclerotiorum* infection in the studied rapeseed/canola germplasm. The continuous and broad range of observed phenotypic responses reinforces the notion that SSR resistance in *B. napus* is a quantitatively inherited trait, controlled by many minor genes with small effect (Zhao et al., 2006; Wu et al., 2013, 2016; Wei et al., 2016; Qasim et al., 2020; Roy et al., 2021). Pearson correlation analyses among the phenotypic traits revealed that DW trait was significantly and strongly correlated with the different time

points LP scores data (r = -0.84 to -0.94). However, LP score at 4 dpi was found to have the strongest association (r = -0.94) with the DW. The strong association among DW and LP scores were also reported by Zhao et al. (2004, 2006). Therefore, based on these findings, LP_4dpi could be used as proxy criterion to DW when evaluating rapeseed/canola germplasm for resistance to *S. sclerotiorum*. The estimated broad sense heritability (H^2) for SSR resistance in the 337 *B. napus* germplasm (62.5 to 70.7%) implied the majority of the observed phenotypic variation was controlled by genetic factors. This level of heritability is consistent with the previous SSR resistance studies (Zhao et al., 2006; Wu et al., 2013; Wei et al., 2016; Qasim et al., 2020; Roy et al., 2021), which further indicate that phenotypic selection is effective and therefore suitable for subsequent GWA analyses to detect favorable alleles conferring SSR resistance to facilitate MAS breeding.

The power of detecting MTAs by GWA study is limited by several factors including population size, cryptic population structure, linkage disequilibrium, heritability of the trait, underlying genetic architecture of the trait of interest, and the statistical models used (Gupta et al., 2005; Yu et al., 2006; Josephs et al., 2017). The diversity panel used here consists of 337 rapeseed/canola genotypes originating from 23 countries and comprised of 0.3% Australian, 28.8% Asian, 35.0% European, and 35.9% North American origin. Therefore, the geographical distribution of the used genotypes in our study provides an ideal diverse panel with all forms of ecotypes collected from the major rapeseed/canola growing regions. The high phenotypic variability among the genotypes coupled with ideal diversity panel with good worldwide geographical coverage, high mean SNP density (~ one SNP per 31.3 kb) enhances QTN detection via GWA analyses. Hereafter, we implemented the first GWA study at the seedling stage to identify the significant SNPs, genomic regions, and putative candidate genes conferring

SSR resistance in rapeseed/canola. As the pattern of quantitatively inherited SSR resistance is complex and controlled by many genes with small effect, we simultaneously used multiple GWA models *i.e.* one SL (GEMMA-MLM) and three ML (MLMM, FarmCPU, and mrMLM) GWA models to discover SNPs associated with the traits. Although SL model (MLM) is widely used to detect the genetic variants for traits of interest in many crop species, it has several limitations to dissect complex traits. SL models perform one-dimensional genomic scan by testing one marker at a time, and also fail to simultaneously match the true overall genetic model of complex/quantitative traits controlled by multiple loci. False-negative (Type II error) could also result from the MLM-based SL models due to the model overfitting, where some potentially important associations could be missed (Liu et al., 2016). On the other hand, tremendous statistical improvement efforts were made over the few years to overcome the problems associated with SL GWA models for the dissection of complex traits. Several multi-locus (ML) GWA algorithms, such as MLMM (Segura et al., 2012), FarmCPU (Liu et al., 2016), mrMLM (Wang et al., 2016), FASTmrEMMA (Wen et al., 2018), ISIS EM-BLASSO (Tamba et al., 2017), and pLARmEB (Zhang et al., 2017), were developed. The advantages of these ML methods are that no multiple test correction is required due to the multi-locus nature of the model, and also have more statistical power and accuracy to detect associations than SL models (Wang et al., 2016; Xu et al., 2017). Similar trends were also observed in the current study, where for most of the traits, where ML GWAS models detected more QTNs compared to the SL model. Among all the GWA models implemented, FarmCPU has shown the highest power to detect MTAs and while controlling for both Type I and Type II errors. Similar results were also found in other studies where SL models generate more false negatives (Tamba et al., 2017; Wen et al., 2018; Kaler et al., 2020). Other studies were also reported, where ML models including

FarmCPU, mrMLM, FASTmrEMMA, and LASSO (ISIS EM-BLASSO), performed better than MLM-based models (Tamba et al., 2017; Wen et al., 2018; Kaler et al., 2020). Based on the Q-Q plot comparison, we also observed that ML (FarmCPU, mrMLM) models perform better in reducing false positives and false negatives; whereas even though GEMMA-MLM (SL) and MLMM (ML) models reduced the false positives, but increases false negatives in some instances. Among all the GWA models implemented, FarmCPU had the highest detection power, while controlling for both Type I and Type II errors. Similar results were observed by other researchers in other studies where SL models generated more false negatives (Tamba et al., 2017; Wen et al., 2018; Kaler et al., 2020).

GWA analyses revealed a total of 219 significant SNPs corresponding 216 loci for all the studied SSR resistance traits. All the mapped 35 QTNs by SL model were simultaneously identified by the ML models. Moreover, ML GWA analyses identified an additional 184 SNPs distributed across all the 19 chromosomes. However, to obtain more reliable results, only SNPs simultaneously detected by both SL and any of the ML methods or at least two of the ML methods or two phenotypic traits were considered as significant QTNs. Thus, a total of 71 SNPs controlling SSR resistance traits were obtained. Additionally, 44 QTNs were simultaneously mapped for at least two SSR resistance traits. Use of SL and multiple ML models for GWA analyses with four SSR resistance phenotypic traits improved the reliability of QTNs detection and were also complementary to each other in identifying common and more significant QTNs for the trait of interest. The ML models detected more significant QTNs over SL models which confirm the power and robustness of ML GWAS models compared to SL. Similar trend were also observed in cotton (Li et al., 2018), maize (Xu et al., 2018), and soybean (Kaler et al., 2020; Karikari et al. 2020), where ML models detected more significant SNPs over SL models.

Therefore, implementing GWA analyses using ML models in conjunction with SL models would enable the detection of more QTNs associated with trait of interests and provide promising opportunities to facilitate the genomics-assisted SSR resistance rapeseed/canola breeding.

Presently, several QTLs/markers associated with early (seedling) and adult stage SSR resistance either in the form of stem, leaf, and days to wilt resistance have been identified using bi-parental linkage mapping and association mapping studies (Zhao and Meng, 2003; Zhao et al., 2006; Yin et al., 2010; Wu et al., 2013, 2016; Wei et al., 2014, 2016; Gyawali et al., 2016; Zhang et al., 2019; Qasim et al., 2020; Roy et al., 2021; Shahoveisi et al., 2021). The physical position of the previously reported QTLs/MTAs were based on the '*Darmor-bzh'* reference genome (Chalhoub et al., 2014). However, in our current study, we used '*ZS11*' as reference genome sequence (Sun et al., 2017), which was aligned with '*Darmor-bzh'* reference genome. The physical location comparison of our identified 219 SNPs with the previously reported QTLs/markers revealed a total of 19 SNPs corresponded to previously reported SNPs/QTLs detected based on linkage and/or association studies. Markers that are linked with QTLs for SSR resistance located within ~500 kb of the same genomic regions, were considered as the same loci. In our current study, a total of 12 significant SNPs was detected on chromosome A09 with multiple GWA models and traits. Among them 2 SNPs (SCM002767.2_21520686, and SCM002767.2_21528172) (21.52-21.53 Mb), located ~7.5 kb apart, were in the close proximity of the mapped QTL by (Wu et al., 2013), and (Qasim et al., 2020) for stem resistance at adult stage. Another SNP (SCM002767.2_27713481) on chromosome A09 (27.71 Mb) co-localized with the *qSR11-1* QTL between the physical position of 27.13-29.36 Mb (Wei et al., 2014). Li et al. (2015) defined the genomic regions spanned 22.5–27.5 Mb on chromosome A09 as the conserved QTL regions for *S. sclerotiorum* resistance based on the integrated and comparative

QTL analyses of the previously identified QTLs. Our findings also corroborate that this physical interval is rich in QTLs conferring resistance to SSR both at the seedling and adult plant stage. Moreover, these genomic regions might be a potential hotspot of stable QTL to carry out fine mapping or map-based cloning to take the full advantage of MAS in SSR resistance breeding. Two additional SNPs identified by multiple models and traits located on C02 (4.60-6.16 Mb) were also detected by other researchers. For instance, Zhao et al. (2006) detected *Sll 12* (stem lesion length) and *Dw 12* (days to wilt) QTL within the physical interval of 0.31-6.71 Mb for SSR resistance using PIT. More QTLs such as *qSR10-3* (1.03-3.95 Mb) and *qSR11-2* (1.03-3.95 Mb) by Wei et al. (2014); *qSRC2* (0.02-4.33 Mb) by Wu et al. (2019); *SRC2a* (0.23-5.55 Mb) and *SRC2b* (0.12-5.19 Mb) by Qasim et al. (2020), were previously detected using stem inoculation method as a screening technique for adult plant resistance. A GWA study conducted by Roy et al. (2021) identified 10 significant SNPs on A09 at 35.6-45.8 Mb for SSR resistance using stem inoculation technique under field environments. Our GWA results also mapped 5 significant SNPs in this genomic regions. The simultaneous detection of common QTNs controlling seedling and adult plant stage SSR resistance in different populations and genetic mapping methods provide valuable insights that warrants further exploration of these genomic regions to develop functional molecular markers that could potentially be used in MAS of target traits at all development stages. Besides the identification of the previously detected SNPs/QTLs, our study also revealed new genomic regions [A01 (1.54-7.38 Mb); A06 (2.50-7.49 Mb); C02 (24.24-26.03 Mb) and (54.98-61.78 Mb)] that may contribute to better understanding of the architecture of *S. sclerotiorum* reaction and could provide more opportunities for SSR resistance breeding in rapeseed/canola.

The identification of stable QTNs/genomic regions is necessary to provide useful information to facilitate MAS. Therefore, the QTNs detected in two or more traits and two or more multiple GWA algorithms were selected for mining potential disease resistance candidate genes for further gene cloning and functional verifications. A total of 81 defense response candidate genes surrounding the stable QTNs were identified using the '*ZS11*' reference genome. These candidate genes were categorized on the basis of their functional characteristics for disease resistance from several databases. A TIR-NB-LRR receptor like protein (Gene ID: LOC106378095) annotated as disease resistance protein *RPP1* was located on chromosome C02 (25.75-26.03 Mb) for multiple traits with multiple GWA algorithms. This candidate was also detected as a potential candidate gene for adult plant stage SSR resistance under field environments by Roy et al. (2021). Recent findings on the lifestyle of *S. sclerotiorum* revealed that there is a brief biotrophic phase followed by a necrotrophic phase, which suggests *S. sclerotiorum* is a hemi-biotrophic pathogen rather than necrotrophic (Kabbage et al., 2015; Chittem et al., 2020). TIR-NB-LRR proteins regulate the activation of salicylic acid (SA)-dependent pathway to confers defense response against biotrophic and hemi-biotrophic pathogens. Therefore, SA may play a positive role in the defense responses against S. sclerotiorum in rapeseed/canola, which agree with Weaver et al. (2006), and Nováková et al. (2014) findings. Another two significant SNPs (SCM002770.2_25782525, SCM002770.2_25886609) identified by multiple traits and multiple models are in close genomic proximity. These SNPs are linked with a germin-like protein subfamily 3 member 1 (*GPL1*) gene (Gene ID: LOC106378104). GLPs have diverse functions including contribution to plant defense reactions against different pathogens. Expression of GLPs was found to be associated with increased resistance against *Sclerotinia* sp. pathogen (Dong et al., 2008). Enhanced resistance in

Arabidopsis thaliana plants against *Verticillium longisporum* and *Rhizoctonia solani* were also reported through the transgenic expression of *BvGLP-1* by Knecht et al. (2010), whereas increased susceptibility was reported to the rice blast fungal pathogen when GLP genes were silenced (Manosalva et al., 2009). However, in *B. napus BnGLP3,* and *BnGLP12* was found to be upregulated and generated more H_2O_2 formation in the partially resistant cultivars compared to the susceptible cultivars upon *S. sclerotiorum* infection. The tolerant lines also generate an increase in H_2O_2 leading to the oxidative burst at the early state of *S. sclerotiorum*-infected leaves and thereby restricting lesion formation compared with the susceptible cultivar (Rietz et al., 2012). Other candidate genes for SSR resistance would also provide useful insights for efforts to achieve *S. sclerotiorum* resistance rapeseed/canola germplasm.

The advantage of GS over GWA study is that it simultaneously exploits the predictive power of all the genome-wide distributed markers (Meuwissen et al., 2001). Therefore, GS is considered as an effective genomic strategy for the improvement of complex traits in crops, that could potentially capture minor-to medium effect loci (Meuwissen et al., 2001; Würschum et al., 2014; Crossa et al., 2017). Since, SSR resistance is a polygenic trait, controlled by numerous minor-effect QTL, genomic prediction would be suitable for resistance breeding purposes. This led us to assess the predictive ability and accuracy by implanting six GS models. The predictive ability of SSR resistance traits were moderate to high, depending on trait specifications. These results clearly demonstrated that genome-wide markers are efficient in predicting resistance of rapeseed/canola genotypes in response to *S. sclerotiorum* attack. All the GS models used in the present study yielded generally similar predictive ability values for all traits, and there were no models found that outperform others, which is consistent with other researchers findings (Spindel et al., 2015; Yu et al., 2016; de Azevedo Peixoto et al., 2017; Azodi et al., 2019; Roy et

al., 2021; Derbyshire et al., 2021). There were 0 to 3-unit differences in the predictive ability existed among the used different GS models across traits. Derbyshire et al. (2021) assessed the predictive ability for adult stage SSR resistance and achieved moderate predictive ability depending on GS model specification. Comparison of the models revealed that Bayesian models resulted similar or worse predictive ability than the G-BLUP model. In soybean, several GP methods including rrBLUP and Bayesian methods were compared for white mold resistance, but no differences between the GP methods were found (de Azevedo Peixoto et al., 2017). Our results agree with de Azevedo Peixoto et al. (2017) findings in common bean and those of Derbyshire et al. (2021) in *B. napus* for *S. sclerotiorum* resistance. Another study in rapeseed/canola for SSR resistance carried out by Roy et al. (2021) reported medium to high 0.43-0.68 predictive ability depending on the assessment of various phenotypic traits *i.e.* lesion length, lesion width, and plant mortality. Wei et al. (2016) used a panel of 347 *B. napus* genotypes, where a five-fold cross validation schemes resulted only 0.27 predictive ability for SSR stem resistance. The higher predictive ability (0.45-0.68) and prediction accuracy (0.57-0.82) observed from this study clearly demonstrates that GS holds promise as a potential genomics-assisted tool to predict genotypes as a potential donor/parents at the early stage (seedling) in efforts to breed SSR resistant rapeseed/canola cultivars. However, evaluation of the genotypes and testing of the GP models in the field screenings would definitely help to boost up application of GS for SSR resistance as the performance of the genotypes would be more representative in the farmer's field.

Finally, the use of PIT as an inoculation method to screen diverse 337 *B. napus* accessions consisting of three ecotypes *i.e.* spring, semi-winter, and winter, against *S. sclerotiorum* attack enabled us to identify promising resistant genotypes at the early

developmental stage. The identified resistant lines will provide a valuable source for canola breeding efforts to improve durable resistance by developing SSR resistant cultivars for the growers. Moreover, screening of genotypes at the same growth stage will help to eliminate/overcome the effects of several physiological traits, which were known to have an indirect effect for SSR resistance evaluation. However, we agree with Zhao et al. (2004) and Bradley et al. (2006), that the identified resistance source should be verified under field environments for their reaction to *S. sclerotiorum* using PIT or other inoculation methods. In addition to common use of DW as a phenotypic response, the highly correlated single time point phenotypic score LP_4dpi could be used as a potential phenotypic trait for large scale phenotyping of SSR resistant genotypes. Due to the quantitatively inherited nature of SSR resistance, it is more likely that QTNs identified here had small effects on resistance in *B. napus*. The detected significant markers identified by GWA or QTL mapping could be converted into kompetitive allele-specific PCR (KASP) markers for SNP validation (Semagn et al., 2014). Our future efforts would be directed towards validating the effects of these QTNs and the development of tightly linked markers to facilitate the cost-effective MAS resistance breeding in rapeseed/canola. Considering the potential of GS for the improvement of polygenic traits, we explored the feasibility of genomic prediction for SSR resistance. High predictive ability coupled with high prediction accuracy demonstrates that GS holds promise for the improvement of SSR resistance in rapeseed/canola. However, assessment of disease resistance and GS models under multilocation field environment might be warranted in the future for effective SSR resistance genomics-assisted rapeseed/canola breeding.

CHAPTER 5: ASSOCIATION MAPPING AND GENOMIC PREDICTION FOR SCLEROTINIA STEM ROT DISEASE RESISTANCE IN *BRASSICA NAPUS* (L.) USING STEM INOCULATION METHOD

5.1. Abstract

Sclerotinia stem rot (SSR), caused by *Sclerotinia sclerotiorum*, is a destructive disease that seriously reduces seed yield and oil quality in rapeseed/canola. In this study, we screened a panel of 144 spring (Study_1), and 152 semi-winter and winter (Study_2) populations two times by inoculating plants at the full flowering stage. Extensive phenotypic evaluation with five traits identified few lines as the potential resistant sources, which showed better resistance over the resistant check cultivars. Genome-wide association (GWA) study with two models identified 37 and 50 SNPs in Study_1 and Study_2 populations, respectively with two or more traits, were regarded as significant. Ten and twelve significant SNPs from Study_1 and Study_2 populations respectively, were found to be co-localized with the previously reported SNPs/QTLs by linkage/GWA mapping studies. In the Study_1 and Study_2 populations, a total of 35 and 46 putative candidate genes associated with plant disease resistance respectively, were annotated within or adjacent to the significant SNPs markers. The genomic prediction (GP) revealed higher predictive ability for Study_1 population (0.48-0.60) over Study_2 population (0.10-0.19), depending on trait specifications. Overall, our study revealed that favorable alleles from significant SNPs from GWA mapping could be utilized as an important resource to transfer and pyramiding resistance genes to improve SSR resistance. Genomic selection could also be used as a potential genomics-based approach for the improvement of SSR resistance in rapeseed/canola.

Key words: Sclerotinia stem rot, genome-wide association study, single nucleotide polymorphism (SNP), genomic prediction, predictive ability.

5.2. Introduction

The fungus *Sclerotinia sclerotiorum* (Lib.) de Bary is a cosmopolitan plant pathogen which infects a wide range of dicotyledonous plant species, including soybean, dry bean, pulses, lettuce, and sunflower (Purdy, 1979; Boland and Hall, 1994). This pathogen also infects one of the most economically important oilseed crop rapeseed/canola (*Brassica napus* L.), in which the disease is commonly referred to as sclerotinia stem rot (SSR) (Barbetti et al., 2013; Wu et al., 2013). SSR disease can impose serious seed yield losses in the major rapeseed/canola growing regions, reaching up to 80% in the severely infected fields (Barbetti et al., 2013; Wu et al., 2016). However, in the United States, for each unit increase in SSR incidence imposes 0.5-0.7% reduction in seed yields (Del Río et al., 2007). In addition to yield loss, *S. sclerotiorum* infected plants also have shown to result in reduced oil content, and oil quality by changing oil's fatty acid composition (McCartney et al., 1999; Sharma et al., 2015).

The pathogen produces sclerotia, a long-lived melanized resting structure, which can survive in soil up to 8-10 years (Adams and Ayers, 1979; Hegedus and Rimmer, 2005), which sometimes makes SSR management using crop rotation ineffective. Although, management of SSR disease through fungicide application has some positive effects, but pinpointing the optimum time in fungicide application is difficult to achieve. Furthermore, use of fungicides may impose a negative impact on the environment and increase additional input costs, which makes rapeseed/canola production less profitable for the growers (Bradley et al., 2006). Therefore, the use of durable disease resistant cultivars is the most effective, environment friendly, and economically feasible to control the disease. Although some partially resistant varieties are available, complete resistance to SSR still has not been identified. Therefore, the identification of

resistant genotypes in diverse germplasm accessions is one of the best options to develop SSR resistant cultivars.

There are three ecotypes of cultivated rapeseed/canola cultivars based on their growth habit differences in terms of flowering time and vernalization requirements (Wang et al., 2011; Rahman, 2013; Arifuzzaman and Rahman, 2020). The winter type requires a long period of vernalization, mainly grown in Europe, whereas semi-winter types grown in China, which needs a shorter period of vernalization. No vernalization is required for spring ecotypes rapeseed/canola to induce flowering and primarily cultivated in North America (USA and Canada) and Australia. In the USA, approximately 83% of the US canola production comes from North Dakota (USDA-NASS, 2021). However, only spring canola is cultivated in North Dakota. Semi-winter and winter types canola are superior in yield than the spring types (Rahman and Mcclean, 2013; Arifuzzaman and Rahman, 2020), but shorter growing season and poor winter hardiness prevents their cultivation in ND. Nevertheless, the identification of resistant sources among semi-winter and winter types is important since their resistance could be introgressed into spring type canola to develop SSR resistant spring cultivars with high yield potential. Phenotyping of *B. napus* genotypes against *S. sclerotiorum* with commonly used stem inoculation method usually applied when the plants are in the flowering stage, because SSR infection is more prevalent in this stage. This further complicates the screening of all growing types of rapeseed/canola against *S. sclerotiorum* under field environments. Therefore, in addition to screening of spring types rapeseed/canola against *S. sclerotiorum*, the present study was also designed to phenotype semi-winter and winter ecotypes by providing 6-weeks vernalization to induce flowering in the controlled environments.

Genetic studies have demonstrated that SSR resistance to rapeseed/canola is a heritable quantitative trait, which is determined by multiple genes with minor or major effects (Wu et al., 2013, 2016; Wei et al., 2016; Qasim et al., 2020). Genetic mapping studies such as classical bi-parental linkage mapping (Zhao and Meng, 2003; Zhao et al., 2006; Yin et al., 2010; Mei et al., 2013; Wu et al., 2013; Wei et al., 2014; Behla et al., 2017; Qasim et al., 2020) and genome-wide association (GWA) study (Gyawali et al., 2016; Wei et al., 2016; Wu et al., 2016; Roy et al., 2021) are the two different strategies that have been applied for the identification of quantitative trait nucleotides (QTNs)/QTL, and putative candidate resistant genes against SSR in rapeseed/canola. The majority of QTL detected by linkage mapping studies were mapped mainly on chromosomes A01, A02, A03, A06, A07, A08, A09, A10, C01, C02, C03, C04, C05, C06, C07, C08, and C09 (Zhao and Meng, 2003; Zhao et al., 2006; Yin et al., 2010; Wu et al., 2013, 2019; Wei et al., 2014; Zhang et al., 2019; Qasim et al., 2020; Shahoveisi et al., 2021). However, QTL detected from different linkage mapping studies were inconsistent and varied in different environments, different mapping populations, different inoculation methods, and various developmental stages. Moreover, in very few instances common QTLs were detected in different mapping populations (Behla et al., 2017; Shahoveisi et al., 2021). Therefore, more mapping studies are necessary to validate these QTLs and fine mapped or cloned the SSR resistant QTLs or genes to take the full advantage of the genetic manipulation through implementing marker-assisted selection (MAS). However, the mapping resolution and accuracy of QTL detection by linkage mapping is often limited by the higher confidence intervals, low allelic diversity, and limited number of recombination events, since only two alleles descended from the two parental lines were analyzed (Korte and Farlow, 2013). In contrast to linkage mapping, genome-wide association (GWA) study has emerged as a promising strategy to dissect quantitative traits

because it overcomes the limitations of the bi-parental populations. GWA mapping exploits natural populations genetic diversity and ancestral historical meiotic recombination events present in germplasm collections. The aim of the GWA mapping is to identify the genetic markers strongly associated with the traits of interest by using linkage disequilibrium (LD) between genes and molecular markers. Extensive historical recombination events led to the development of shorter LD fragments in natural populations and high-density genetic maps allowing an increased mapping resolution that makes possible to map the genetic markers/QTLs near to the gene of interest (Nordborg and Weigel, 2008).

To date, several GWA analyses have been performed for genetic mapping of SSR resistance in rapeseed/canola (Gyawali et al., 2016; Wei et al., 2016; Wu et al., 2016; Roy et al., 2021). A GWA study by Gyawali et al. (2016) identified 34 significant associated loci, out of which 21 alleles contributed to the resistance. Twenty-six SNPs, located on chromosome C04, C06, and C08, were detected by Wu et al. (2016) using detached stem inoculation assay. Wei et al. (2016) mapped 17 significant associations distributed on chromosome A08, and C06, from two years field evaluations using detached stem inoculation method. Another study by Roy et al. (2021) mapped a total of 133 significant SNPs using four SSR resistance traits from the four environments field evaluations by directly inoculating the plant into the main stem. Of these four GWA studies, no overlapping or common significant SNPs were detected from the study carried out by Gyawali et al. (2016); Wei et al. (2016); and Wu et al. (2016). However, Roy et al. (2021) found few overlapping QTNs with the SNPs detected by Wei et al. (2016) and Wu et al. (2016). These outcomes clearly highlight that more GWA analyses are needed to identify more significant SNPs associated with enhanced SSR resistance to identify all the variable nature of SSR disease interaction. Moreover, most of the detected QTNs only explained a small portion of

phenotypic variance, and were highly influenced by the genetic backgrounds of the studied populations. These results limit in pyramiding the small-effect favorable alleles to take the advantage of MAS for improving SSR resistance in rapeseed/canola.

Genomic selection (GS) has emerged as an alternative to MAS, offering promising potential for the genetic improvement of complex traits (Meuwissen et al., 2001). In GS, genome-wide markers were used to predict the genomic estimated breeding values (GEBVs) of the individuals by capturing both large and small-effect loci (Meuwissen et al., 2001; Poland and Rutkoski, 2016). The GEBVs are estimated from a trained genomic prediction (GP) model, developed using the phenotypic information of a genotyped population called the training population. Then the trained GP model is used to predict the genetic values (GEBVs) in a validating population that have been only genotyped but not phenotyped. Relatively few GP studies have been reported in rapeseed/canola for various traits including seed yield and quality-related traits (Würschum et al., 2014), oil quality (Werner et al., 2018), various agronomic traits and blackleg disease resistance (Jan et al., 2016; Fikere et al., 2020). GS studies for SSR resistance, a complex trait in rapeseed/canola, suggested that GS is promising to enhance genetic improvement of SSR resistance (Roy et al., 2021; Derbyshire et al., 2021).

In the present study, we evaluated spring, semi-winter, and winter types *B. napus* lines in the greenhouse with aimed to i) identify the SSR resistant genotypes at the adult plant stage; ii) reveal SNPs or genomic regions significantly associated with SSR resistance; iii) search for SSR resistance candidate genes in the identified SNP regions for further study; iv) assess the potential of GS for predicting SSR resistance genotypes.

5.3. Materials and methods

5.3.1. Plant materials and experimental design

In this study, we used a panel of 144 spring types, and 152 semi-winter and winter types *B. napus* germplasm accessions and advanced breeding lines, collected from North Central Regional Plant Introduction Station (NCRPIS), Ames, Iowa, USA and North Dakota State University (Table A12, Table A13). The experiments were carried out in the Agricultural Experiment Station Research Greenhouse Complex, North Dakota State University, Fargo, ND, USA. Each genotype was planted in a randomized complete block design (RCBD) with three replicates. In each replicate, five plants were sampled for inoculation. The temperature of the greenhouse was maintained at 22 ± 2 ℃ provided with 16-hours of photoperiod supplemented with 400 W HPS PL 2000 lights (P.L. Light Systems Inc.). The whole experiments were divided into different batches by taking a subset of genotypes in each batch with respective used check cultivars. For the phenotyping of spring types canola, two commercially available spring canola cultivars, “Pioneer 45S51” and “Pioneer 45S56”, were used as resistant checks, and publicly available Canadian spring type canola cultivar “Westar” were included as a susceptible check. SSR resistant semi-winter types genotype “NEP63” (Chittem et al., 2020; Shahoveisi et al., 2021), was used as a resistant check for the screening of semi-winter and winter types of rapeseed/canola.

5.3.2. Vernalization of semi-winter and winter type genotypes to induce flowering

The *S. sclerotiorum* pathogen mainly infects the plant during the flowering stage. Therefore, we decided to evaluate the reaction of the respective genotypes by inoculating the plants at the full flowering stage. To induce flowering in semi-winter and winter type rapeseed/canola lines, three weeks old seedlings were transferred into a plant growth chamber for

six weeks of vernalization at 4℃ temperature with 16-h/8-h day/night cycle with high-pressure sodium light provided. After vernalization, the plants were moved into the greenhouse room and allowed them to induce flowering.

5.3.3. Assessment of resistance to *Sclerotinia sclerotiorum*

The screening of three ecotypes rapeseed/canola were divided into two separate experiments. Since, no vernalization is required to induce flowering in spring canola, therefore, the spring type was grouped into the first study designated as Study_1. Vernalization was required to induce flowering in semi-winter and winter types, and was grouped into the second study designated as Study_2. Phenotyping of each study population was conducted twice to evaluate the performance of rapeseed/canola genotypes against *S. sclerotiorum* reaction. A single high virulence isolates of *S. sclerotiorum* (WM031) was used for inoculating plants (Roy et al., 2021; Shahoveisi et al., 2021). Inoculum was prepared by culturing the surface sterilized sclerotia on autoclaved potato dextrose agar (PDA) medium (24 gL^{-1} potato dextrose broth and 15 gL^{-1} agar) containing in a petri dish and incubated at 22-24 ℃ temperature. When the plants reached at full flowering stage, the main stem was inoculated following a protocol described by Li et al. (2004). Briefly, mycelium agar plugs (5-7 mm) were cut from the actively growing margin of the 2 days old mycelium with the help of 1000 µl pipette tips before hyphae reached at the edge of the petri dish. Each mycelium plug was placed on a 3×6 cm piece of parafilm by making a small pole with the help of finger and attached to the main stem with the mycelium facing the stem epidermis. For each accession, lesion lengths on the main stem and lesion width (visual estimation of the percentage of the main stem that is girdled by the lesion) were recorded 3, 5, 7, 9, 11, 13, and 15 days post inoculation (dpi). The status of inoculated plant was recorded as dead or alive at 15 dpi. Plants were considered as dead when infected main stem girdled

completely and irreversible wilting symptoms were observed on plant parts above the inoculated stem.

5.3.4. Phenotypic measurements and statistical analyses

5.3.4.1. Stem lesion length (LL), lesion width (LW), and plant mortality (PM)

We have collected data on lesion length (LL) and lesion width (LW) at 7 dpi. Data collection on LL and LW was stopped once the inoculated plant was considered dead. We also calculated % of plant mortality (PM) of each genotype at 15 dpi.

5.3.4.2. Area under disease progress curve (AUDPC)

Area under disease progress curve (AUDPC) on LL (LL_AUPDC) and LW (LW_AUDPC) for each evaluated accession were calculated using the following formula:

$$\text{AUDPC} = \sum_{i}^{n-1} \left(\frac{y_i + y_{i+1}}{2} \right) (t_{i+1} - t_i)$$

where y_i is symptom lesion length and lesion width on the i^{th} day, t_i is time in days on the i^{th} day, and n is the number of measurements (seven in this study).

Since, we did not record data once the inoculated plant was dead, therefore we adjusted the LL and LW data by replacing the dead plant value with the recorded maximum lesion length and lesion width value of the respective replication within the same batches following Shahoveisi et al. (2021). However, we ensured that the calculated AUDPC value of susceptible lines would be larger than the resistant lines to reflect the true phenotypic performance of the evaluated genotypes.

Statistical analyses of all phenotypic traits were carried out using SAS 9.4 software (SAS Institute 2012). Common check cultivars were used in every batches within each experiment. To determine whether the data within the batches in an experiment could be combined or not,

homogeneity of variance tests was performed by Levene's test using common check cultivars phenotypic performance. Hence after, the data from two experiments were combined if the ratio of the effective error variance of each trait is less than 10-fold (Tabachnick and Fidell, 2000; Arifuzzaman and Rahman, 2020). For each study, best linear unbiased estimates (BLUEs) of five traits (LL, LW, PM, LL_AUDPC, and LW_AUPDC) were calculated for each genotype by considering genotypes as a fixed effect, whereas replication and experiments were considered as random effects.

5.3.5. Genotyping

Fresh, young leaves of 12 days old seedlings were lyophilized, and total genomic DNA was extracted using Qiagen DNeasy kit (Qiagen, CA, USA) according to manufacturer's protocol. Extracted DNA was quantified and diluted to 50 ng/µl. The DNA Libraries were prepared using *ApekI* enzyme digestion described by Elshire et al. (2011). Single-end reads were sequenced from the developed libraries at the University of Texas Southwestern Medical Center, Dallas, Texas, USA, using Illumina Hi-Seq 2500 sequencer. The sequencing reads were aligned with the "*ZS11*" reference genome sequence of *B. napus* (Sun et al., 2017) with Bowtie 2.0 (Langmead and Salzberg, 2012). The SNP discovery and genotype calling were performed using the TASSEL 5 GBSv2 pipeline (Glaubitz et al., 2014). SNP markers were filtered with an individual read depth > 3, minor allele frequency (MAF) > 0.05, and missing data < 25% with VCFtools (Danecek et al., 2011). As canola is a self-pollinated crop, more than 25% heterozygous SNPs were removed with TASSEL 5.0 (Bradbury et al., 2007). Moreover, we also removed the SNP markers which were located outside of the chromosomes (i.e. unknown physical position). After applying all these quality filtering, 38,150 SNPs markers were obtained.

5.3.6. Genome-wide association studies

After filtering for MAF at 5% level, a total of 23,768 SNPs and 27,580 SNPs was used for subsequent association analyses for Study_1 (Spring) and Study_2 (Semi-winter and winter) populations, respectively. Two GWAS algorithms *i.e.* GEMMA-MLM (Zhou and Stephens, 2012) and FarmCPU (Liu et al., 2016) were used to identify the marker-trait-associations (MTAs) in both studies. The principal component analysis (PCA) was calculated by *prcomp ()* function in R language (R Core Team, 2020). Mclust R package was used to perform model-based clustering using first three PCA to determine the number of subpopulations among the association panel from each study. To account for confounding effect of population structure and genetic relatedness, the first three PCA and a kinship matrix were incorporated in both GWA models as covariates to control the detection of spurious associations. The GEMMA-MLM was performed through the execution of following commands in the GEMMA (version 0.98.1) software: "gemma -g [genotype data] -p [phenotype] -a [genotype annotation data] -c [first 3PCA] -k [centered relatedness matrix] -o [output]. The kinship matrix was generated using from the centered relatedness procedure in GEMMA, used as a random effect variable in the random model. The PCA used in the GMMA-MLM model was obtained from the *prcomp ()* function. *P-wald* test (the improved calibrated *P*-value in GEMMA) was calculated for the given model. FarmCPU is a multi-locus R based GWA mapping model that was developed to control false positives without comprising false negatives (Liu et al., 2016). In FarmCPU, Multiple Loci Linear Mixed Model (MLMM) is divided into two parts, a Fixed Effect Model (FEM) and a Random Effect Model (REM), which were used iteratively. False positives are controlled by FEM that contains testing markers, one at a time, and associated multiple markers as a covariate. To avoid model overfitting problem in FEM, REM uses estimated associated markers to define

the kinship. FarmCPU was run using the following command line in R: FarmCPU (Y = myY, GD = myGD, GM = myGM, CV = myCV). Y, GD and GM represent phenotype, genotype and genotypic map data respectively. CV represents the first three principal components files that were also used in GEMMA-MLM. The automatically estimated kinship matrix by FarmCPU was used. The purpose of using two different models was to reduce the chances of committing type 1 and type 2 errors.

The GWA significant threshold determined by Bonferroni correction was too stringent, which results in elimination of many important loci associated with trait of interest. Therefore, we used a method proposed by Li and Ji (2005) to determine the significant threshold value for the identified QTNs in both studied populations. In this method, we calculated the effective number of independent loci (M_{eff}) by estimating correlation matrix and eigenvalue decomposition. Then the test criteria were adjusted using the M_{eff} with the following correction by Sidak (1967):

$$\alpha_p = (1 - \alpha_e)^{1/M_{eff}}$$

where, α_p is the comparison-wise error rate and α_e is the experiment-wise error rate ($\alpha_{e=0.05}$).

5.3.7. Candidate gene predictions

Candidate genes, located within ± 50-kb genomic regions of each significant SNP, were searched using *B. napus* "*ZS11*" reference genome sequence (Sun et al., 2017). The obtained gene models found within the genomic interval regions of the genome sequence were subjected to search on Uniport website (https://www.uniprot.org/uniprot/) to validate their putative functions. The genes associated with defense response mechanisms were identified based on TAIR 10, Uniport-KB database, and gene functions found in previous literature and considered as putative candidate genes for *S. sclerotiorum* resistance in rapeseed/canola.

5.3.8. Genomic prediction

For both studies, the rrBLUP GS model was implemented in the R package for estimating the predictive ability of all the phenotypic traits for SSR resistance (Endelman, 2011). The rrBLUP model is described as follows:

$$y = \mu + X\beta + \varepsilon$$

where y is the vector of phenotypic values, μ is the intercept/grand mean, X is the standardized marker genotype matrix, β is the estimated random additive marker effects, and ε is the residual error term.

The predictive ability of the GP model was tested with five-fold cross validation sets. For Study_1, out of 144 lines 115 individuals were used as training set and remaining 29 individuals as validation sets. In the case of Study_2 (Semi-winter and winter), 122 genotypes were considered as training individuals and 30 genotypes as validation individuals from a total of 152 semi-winter and winter type genotypes. The cross-validation procedures were replicated 100 times to avoid biases in the estimation of predictive ability. Prediction ability of each trait was defined as the estimation of Pearson correlation between the average of the predicted Genomic Estimated Breeding Values (GEBVs) and the observed phenotypes in all the cross-validation sets.

5.4. Results

5.4.1. Phenotypic responses of *Brassica napus* cultivars to *Sclerotinia sclerotiorum* infection

5.4.1.1. Study_1 population (Spring)

None of the phenotypic datasets i.e. LL, LW, PM, LL_AUDPC, LW_AUDPC were normally distributed (Figure A15a-e). A broad range and distinct phenotypic variations existed for the recorded phenotypic traits upon the challenge of *S. sclerotiorum* infection on spring types genotypes (Table 5.1, Figure 5.1a-b). Among the recorded five SSR traits, LL ranged from 2.8-

13.3 cm with the mean ± standard deviation (SD) of 7.6 (mean) ± 2.7 cm (SD), whereas LW, PM, LL_AUDPC, and LW_AUDPC had a range (mean ± SD) of 21.2-97.3% (63.4 ± 18.2 %), 6.7-100.0 % (64.1 ± 24.9%), 38.6-143.1 (96.7 ± 26.9), and 308.3-1054.8 (790.0 ± 173.0) respectively. The coefficient of variation (CV) were 31.4, 26.7, 38.8, 24.7, and 19.6 for LL, LW, PM, LL_AUDPC, and LW_AUDPC, respectively (Table 5.1). Among the evaluated 144 spring types genotypes, only 4 lines had less than or equal to 20% plant mortality at 15 dpi. These genotypes were considered as the promising SSR resistant genotypes. Moreover, they showed better level of resistant performance compared to the used commercial resistant check cultivars in the current study. The phenotypic responses of the top four promising spring ecotypes were ranged 2.8-4.3 cm for LL, 21.2-38.5 % for LW, 6.7-20.0% for PM, 38.6-57.4 for LL_AUDPC, and 308.3-503.7 for LW_AUDPC. However, phenotypic responses in terms of LL, LW, PM, LL_AUDPC, and LW_AUDPC traits for resistant check "Pioneer 45S51" were 5.6 cm, 49.4%, 46.9%, 78.5, and 649.1, respectively, and "Pioneer 45S56" were 5.1 cm, 41.1%, 31.3%, 70.9, and 556.9, respectively. The recorded performance of the susceptible spring cultivar "Westar" was 9.1 cm, 79.9%, 100.0%, 108.4, and 925.5, for LL, LW, PM, LL_AUDPC, and LW_AUDPC traits, respectively (Table 5.1). Analysis of variance (ANOVA) for all SSR traits revealed significant differences ($P \leq 0.05$) among the genotypes, interaction of genotypes by experiment with an exception for LW_AUDPC trait (interaction non-significant) (Table A14). The estimated broad-sense heritability (H^2) was high, varying from 0.68 to 0.92 (Table 5.1).

5.4.1.2. Study_2 population (Semi-winter and winter)

Continuous and distinct phenotypic difference was observed in terms of resistance responses among the 152 semi-winter and winter lines after inoculation with *S. sclerotiorum* (Table 5.2, Figure 5.1c-d). Among the five collected SSR resistance traits, four traits such as LL,

LW, LL_AUDPC, LW_AUDPC were normally distributed based on the Shapiro Wilk normality test *P*-value ($P > 0.05$) (Table 5.2, Figure A15f-j). However, plant mortality data was found not to be distributed normally ($P < 0.05$) (Figure A15h). The average LL, LW, PM, LL_AUDPC, and LW_AUDPC were 7.8 cm, 58.2%, 57.3%, 100.3, and 725.1, respectively. The coefficient of variation (CV) of the association population for different SSR traits varying from 21.7 to 48.8% (Table 5.2). The percentage of plant mortality among the genotypes varied from 16.7 to 100 %. Nine genotypes had less than or equal to 20% plant mortality at 15 dpi. However, few genotypes showed promising resistance performance by producing smaller lesions on the main stem and the lowest percentage of plant mortality compared to the susceptible genotypes. The phenotypic response of the top four resistance genotypes in terms of LL, LW, PM, LL_AUDPC, and LW_AUDPC ranged from 4.4 to 5.6 cm, 32.2 to 42.0 %, 16.7 to 20.0 %, 57.1 to 71.3, and 427.7 to 533.0, respectively. However, performance of the used resistant check "NEP63" was 7.1 cm, 54.7%, 45.6%, 95.4, and 705.3 for LL, LW, PM, LL_AUDPC, and LW_AUDPC traits, respectively. ANOVA of all SSR traits revealed significant differences ($P \leq 0.05$) among the genotypes, interaction of genotypes by experiment (Table A15). The broad sense heritability (H^2) estimate for these phenotypic traits were low to medium, ranging from 0.25 to 0.44 (Table 5.2).

Figure 5.1. Differential phenotypic responses of *Brassica napus* genotypes against *Sclerotinia sclerotiorum* attack. The top promising spring resistant line at 7 days post inoculation (7dpi) (a); susceptible check spring cultivar (Westar) at 5 dpi (b); top semi-winter and winter ecotypes resistant line at 7 dpi (c); susceptible semi-winter and winter line at 11 dpi (d)

Table 5.1. Phenotypic variation obtained through BLUE values in the response of *Brassica napus* spring genotypes against sclerotinia stem rot

Traits[a]	Min	Mean	Max	Median	SD[b]	CV[c]	Shapiro–Wilk test *p* value	H^2	Check cultivars (mean)[d]		
									Pioneer 45S51	Pioneer 45S56	Westar
LL	2.8	7.6	13.3	6.4	2.7	31.4	$1.0e^{-8}$	0.85	5.6	5.1	9.1
LW	21.2	63.4	97.3	60.0	18.2	26.7	$8.6e^{-5}$	0.92	49.4	41.1	79.9
PM	6.7	64.1	100.0	80.0	24.9	38.8	$1.1e^{-5}$	0.68	46.9	31.3	100.0
LL_AUDPC	38.6	96.7	143.1	93.6	26.9	24.7	$4.7e^{-7}$	0.83	78.5	70.9	108.4
LW_AUDPC	308.3	790.0	1054.8	818.0	173.0	19.6	0.001	0.89	649.1	556.9	925.5

Traits[a]: LL, Lesion length measured at 7 days post inoculation (dpi); LW, lesion width at 7 dpi; PM, plant mortality at 15 dpi; LL_AUDPC, lesion lengths area under disease progress curve (AUDPC) calculated using 7 time points reading; LW_AUDPC, lesion widths AUDPC calculated using 7 time points reading; SD[b]: standard deviation; CV[c]: co-efficient of variation; Check cultivars (mean)[d]: mean phenotype scores of the resistant checks (Pioneer 45S51 and Pioneer 45S56) and susceptible check (Westar) cultivars of the evaluated traits

Table 5.2. Phenotypic variation obtained through BLUE values in the response of *Brassica napus* semi-winter and winter ecotypes genotypes against sclerotinia stem rot

Traits[a]	Min	Mean	Max	Median	SD[b]	CV[c]	Shapiro–Wilk test *p* value	H^2	NEP-63 (Resistant check)
LL	4.4	7.8	12.3	6.7	1.6	41.1	0.18	0.25	7.1
LW	32.2	58.2	86.5	55.0	10.8	36.3	0.79	0.40	54.7
PM	16.7	57.3	100.0	60.0	18.1	48.8	0.001	0.35	45.6
LL_AUDPC	57.1	100.3	143.3	97.7	16.9	28.7	0.34	0.25	95.4
LW_AUDPC	427.7	725.1	986.8	723.5	110.0	21.7	0.72	0.44	705.3

Traits[a]: LL, Lesion length measured at 7 days post inoculation (dpi); LW, lesion width at 7 dpi; PM, plant mortality at 15 dpi; LL_AUDPC, lesion lengths area under disease progress curve (AUDPC) calculated using 7 time points reading; LW_AUDPC, lesion widths AUDPC calculated using 7 time points reading; SD[b]: standard deviation; CV[c]: co-efficient of variation.

5.4.2. Relationships/correlations among the sclerotinia stem rot resistance traits

Highly significant correlations were found among the studied phenotypic traits in the spring population for SSR reaction. For instance, the significant positive associations were found for LL with LW (r = 0.96), PM (r = 0.88), AUDPC_LL (r = 0.98), and AUDPC_LW (r = 0.94) at $P \leq 0.001$ (Figure 5.2a). Similarly, strong positive associations (r = 0.78-0.93, $P \leq 0.001$) were found to be associated among the five phenotypic traits in the studied 152 semi-winter and winter ecotypes genotypes (Figure 5.2b).

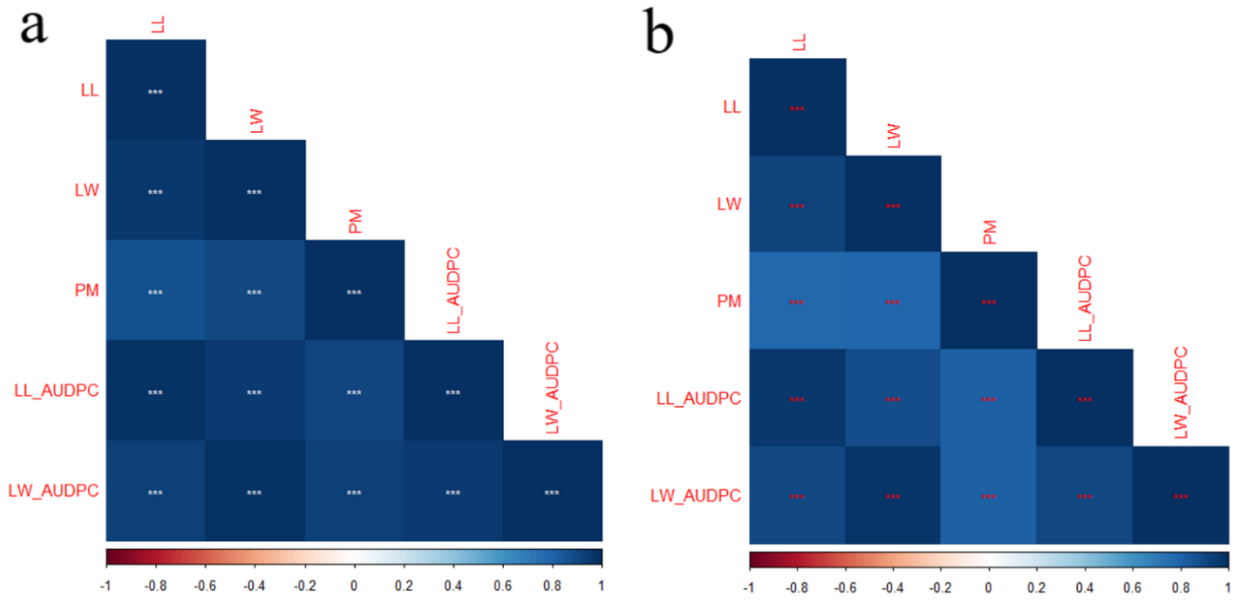

Figure 5.2. Correlation heatmap of various *Sclerotinia sclerotiorum* resistance phenotypic traits in spring ecotypes (a), and semi-winter and winter populations (b). Traits: LL = Lesion length measured at 7 days post inoculation (dpi); LW = lesion width at 7 dpi; PM = plant mortality at 15 dpi; LL_AUDPC = lesion lengths area under disease progress curve (AUDPC) calculated using 7 time points reading; LW_AUDPC = lesion widths AUDPC calculated using 7 time points reading.

5.4.3. Genotypic data and principal component analysis

In the spring type population, the first and second PCA explained 8.9% and 5.9% of the variance, respectively (Figure 5.3a), whereas the first three PCA total accounted for 19.6% of the variance (Figure 5.3b). In the Study_2 (Semi-winter and winter), the first three PCA accounted

for a total of 19.0% of the variance, and at PC3 the inflection point occurred. Therefore, we selected the first three PCA for both studied populations to incorporate as a covariate in the GWA analyses to correct the problems associated with population structure. Furthermore, model-based clustering analysis using the first three PCA in both populations suggested that there were seven and six subgroups within the genotypes (Figure 5.3a-b).

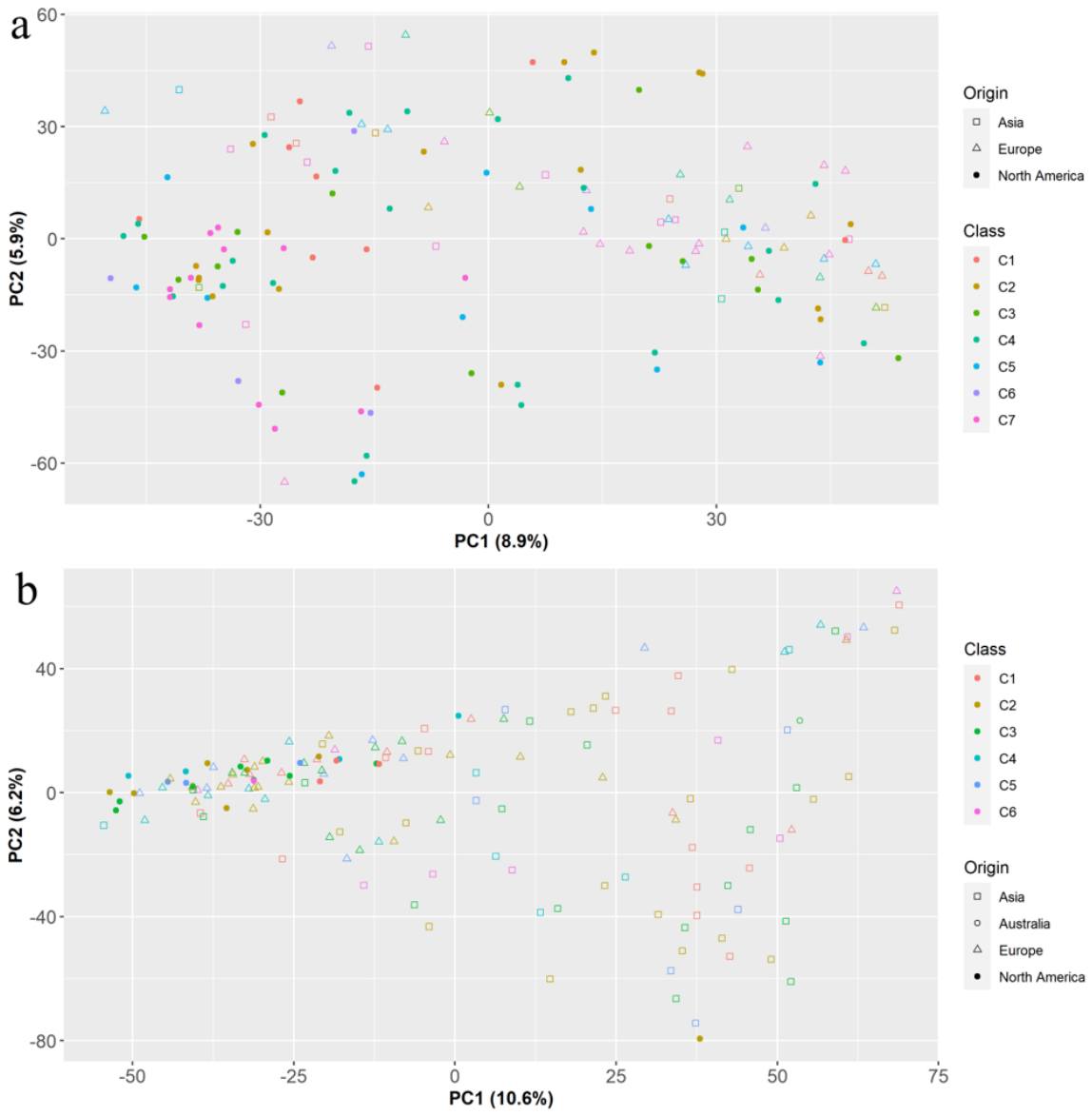

Figure 5.3. Population structure as reflected by the scatter plot of PC1 and PC2 derived from a principal component analysis of a) 144 spring ecotypes rapeseed/canola genotypes using 23,768 SNP markers, and b) 152 semi-winter and winter ecotypes rapeseed/canola genotypes using 26,768 SNP markers.

5.4.4. Marker-trait association analyses

Combined analyses BLUEs for each trait were used for the subsequent GWA analyses in both populations. The *P*-value threshold to declare significant SNPs were determined on the basis of modified Bonferroni correction by calculating the effective number of independent loci from the tested SNP markers for both populations according to the proposed method by Li and Ji (2005). Using this method, a total of 100 and 111 independent markers were identified for Study_1 and Study_2 populations, respectively. Therefore, the significant threshold for spring population was ($P \leq$ 5.12E-04; LOD $\geq$ 3.29 ~3.3), and ($P \leq$ 4.62E-04; LOD $\geq$ 3.34 ~3.3) for semi-winter and winter ecotypes. Moreover, to identify the common significant SNPs present in more than one trait, a threshold value of $P \leq 0.001$ (LOD =3.0) was used, but only if the representative SNP had an association in the determined threshold *P*- value in a second trait by any of the GWA models.

5.4.4.1. Study_1 (Spring population)

GWA analyses with five associated traits for SSR disease have varied from 10-17 QTNs depending on trait specifications and used GWA mapping algorithms at the threshold value of ($P \leq$ 5.12E-04; LOD $\geq$ 3.3) (Figure 5.4a-b; Table A16). The highest 17 SNPs were detected for PM traits by the GEMMA-MLM model followed by the lowest 10 SNPs for LL (GEMMA-MLM) and PM (FarmCPU) traits. A total of 46 SNPs was detected among the five traits for SSR resistance. The detected SNPs were distributed unevenly in the 17 chromosomes of *B. napus* genome. Seven SNPs were found to be commonly mapped among all the phenotypic traits. Out of the 46 SNPs, 37 SNPs were commonly identified by at least two or more traits by any of the GWA models and declared as significant, which could be associated with SSR resistance in rapeseed/canola (Table A17). The estimated allelic effects of the identified SNPs ranged between

-2.65 to 1.28 for LL, -25.19 to 11.83 for LW, -35.55 to 22.26 for PM, -36.83 to 17.35 for LL_AUDCP, and -267.28 to 120.17 for LW_AUDPC, respectively (Table A16, A17).

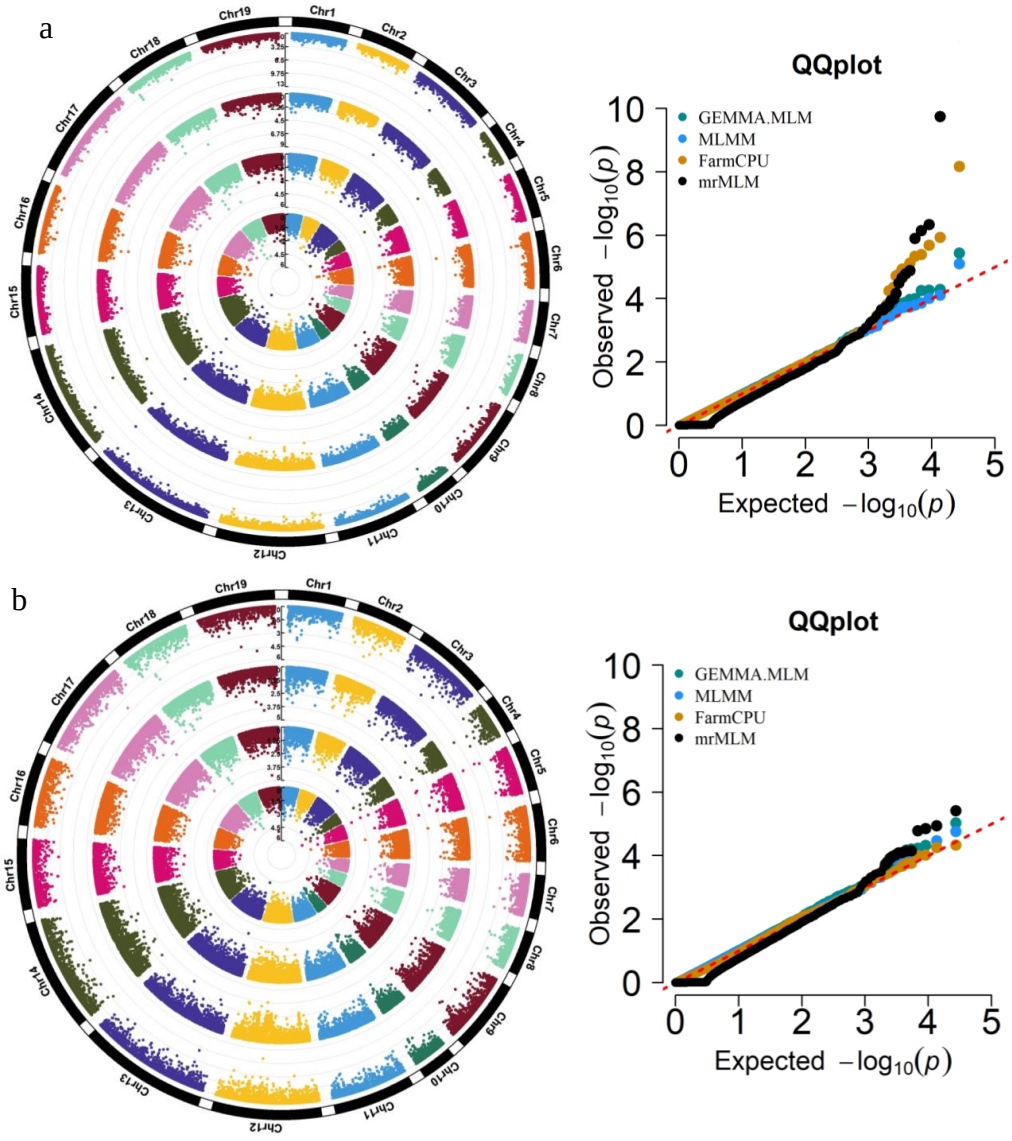

Figure 5.4. A circular Manhattan plot showing the significantly detected SNPs for five phenotypic traits in spring ecotype populations based on GEMMA-MLM (a), and FarmCPU (b) models located on 19 chromosomes for the Sclerotinia sclerotiorum resistance at adult plant stage. Traits: LL = lesion length measured at 7 days post inoculation (dpi); LW = lesion width at 7 dpi; PM = plant mortality at 15 dpi; LL_AUDPC = lesion lengths area under disease progress curve (AUDPC) calculated using 7 time points reading; LW_AUDPC = lesion widths AUDPC calculated using 7 time points reading. A multi-track Q-Q plot for each trait with the two GWAS models is presented at the upper right corner of the circular Manhattan plot. The threshold values for both models were set up at $-\log_{10}(P) \geq 3.3$ ($P \leq 5.12 \times 10^{-4}$).

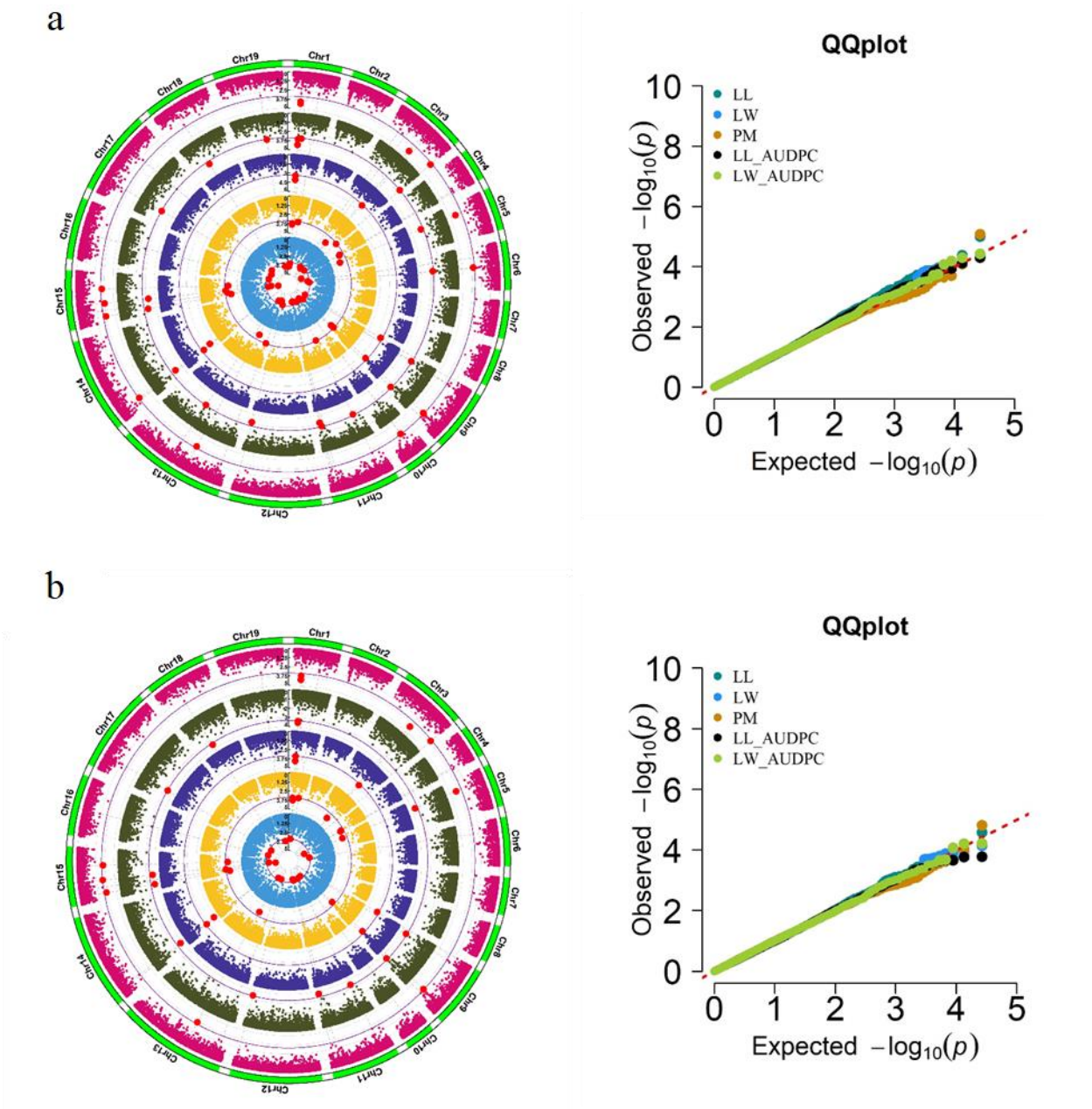

Figure 5.5. A circular Manhattan plot showing the significantly detected SNPs for five phenotypic traits in semi-winter and winter ecotype populations based on a) GEMMA-MLM, and b) FarmCPU models located on 19 chromosomes for the Sclerotinia sclerotiorum resistance at adult plant stage. Traits: LL = Lesion length measured at 7 days post inoculation (dpi); LW = lesion width at 7 dpi; PM = plant mortality at 15 dpi; LL_AUDPC = lesion lengths area under disease progress curve (AUDPC) calculated using 7 time points reading; LW_AUDPC = lesion widths AUDPC calculated using 7 time points reading. A multi-track Q-Q plot for each trait with the two GWAS models is presented at the upper right corner of the circular Manhattan plot. The threshold values for both models were set up at $-\log_{10}(P) \geq 3.3$ ($P \leq 4.62 \times 10^{-4}$).

5.4.4.2. Study_2 (Semi-winter and winter population)

The results of GWA analyses for SSR resistance on five traits using GEMMA-MLM, and FarmCPU methods were shown in Figure 5.5a-b, and Table A18, A19. In total, 34, 18, 8, 23, and 19 SNPs were significantly associated with disease resistance at the *P*- value threshold of 4.62 x 10^{-4} were identified in the LL, LW, PM, LL_AUDPC, and LW_AUDPC traits, respectively. The GWA analyses for all traits detected a total of 59 SNPs (Table A18). Among these 59 SNPs, 50 SNPs were commonly identified in two or more phenotypic traits that are distributed on chromosome A01, A02, A03, A04, A05, A06, A09, A10, C01, C02, C03, C04, C05, C06, C07, C08 and C09 (Table A19). These 50 SNPs were considered as significant SNPs, and could be associated with adult plant stage resistance to SSR reaction. Five SNPs, including two SNPs SCM002759.2_7025054, SCM002759.2_7052802 on chromosome A01, two SNPs SCM002767.2_38082926, SCM002767.2_38774352 on chromosome A09, and one SNP SCM002772.2_7708586 on chromosome C04 were simultaneously detected in all the five phenotypic traits. The allelic effect of the detected SNPs for LL, LW, PM, LL_AUDPC, and LW_AUDPC traits varying from -1.75 to 1.92, -10.72 to 9.16, -19.61 to 11.14, -17.63 to 18.16, and -109.46 to 116.08, respectively (Table A18, A19).

5.4.5. Candidate genes

To identify the putative disease resistance candidate genes, only the significant SNPs detected in at least two or more traits in both populations were used. Candidate gene mining was done using "*ZS11*" reference genome sequence within ± 50 kb of the respective SNPs on genomic regions. We selected 50 kb genomic interval because LD for this population is low (< 45 kb genome wise, < 21 kb for A genome and < 93 kb for C genome) (Rahman et al., 2021). A total of 35 and 46 potential candidate genes in the spring population, and semi-

winter and winter populations, respectively, with known functions associated with plant disease resistance mechanisms were identified. The detailed list of the genes of these populations, their biological functions based on TAIR 10, Uniport-KB database, and corresponding details are provided in Table A20 and Table A21. The detected candidate genes are involved in the biological process of defense response, defense response to fungus, innate immune response, programmed cell death, response to molecule of fungal origin, response to salicylic acid, indole glucosinolate biosynthetic process, induced systemic resistance, response to chitin, jasmonic acid mediated signaling pathway, systemic acquired resistance, pattern recognition receptor signaling pathway, response to wounding, response to nematode, response to oxidative stress, toxin catabolic process, immune response, cell wall organization, reactive oxygen species metabolic process, protein serine kinase activity, signal transduction, calcium-mediated signaling, ethylene-activated signaling pathway, brassinosteroid mediated signaling pathway and other biological processes which might play key role in SSR resistance in rapeseed/canola (Table A20, A21).

5.4.6. Predictive ability of different traits for *Sclerotinia sclerotiorum* resistance

The implementation of rrBLUP GP model with five-fold cross validations resulted in different predictive ability across different SSR resistance traits and across two studied populations. In the case of spring type population, using all 23,768 genome-wide SNPs the average predictive ability values were high (r = 0.48-0.60) depending on the used phenotypic traits (Figure 5.6a). The highest (r = 0.60) value was obtained for 7 days main stem lesion length (LL), and the lowest (r = 0.48) for 15 dpi plant mortality traits. There are 1 to 4-unit differences in predictive ability observed among the four SSR traits such as LL (0.60), LW (0.59),

LL_AUDPC (0.57), and LW_AUDPC (0.56). However, the observed predictive ability was lower in the semi-winter and winter type population than the spring type population. The estimated mean predictive ability for five SSR traits in semi-winter and winter type populations employing genome-wide markers (26,768 SNPs) ranged from 0.10 to 0.19 (Figure 5.6b). The predictive ability was highest (r = 0.19) for LL_AUDPC trait, whereas the lowest predictive ability (r = 0.10) was observed in PM trait. The observed higher and lower predictive ability between the two populations were partially related to trait heritability. For example, LL had a high heritability of 0.85 and high predictive ability of 0.60 in the spring population, whereas low heritability (0.25) and predictive ability (r = 0.17) was observed for LL in semi-winter and winter type populations.

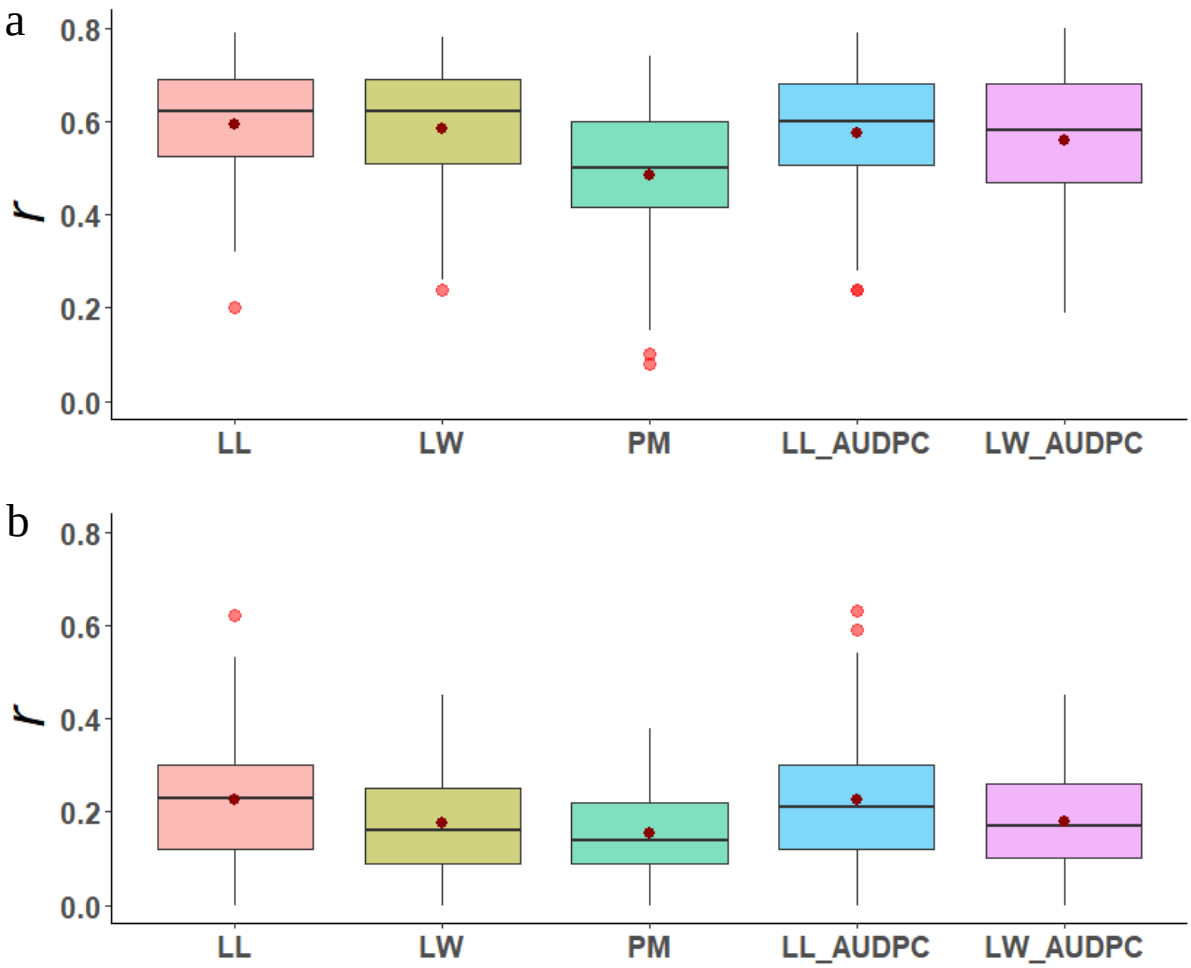

Figure 5.6. Average predictive ability for sclerotinia stem rot resistance associated five phenotypic traits estimated from the five-fold cross-validation schemes of the a) spring ecotype, and b) semi-winter and winter ecotypes association panel. The boxes show second and third quartiles and wishkers show interquertile range. The red dot in each box plot represent the mean predictive ability. The studied five traits were: LL = Lesion length measured at 7 days post inoculation (dpi); LW = lesion width at 7 dpi; PM = plant mortality at 15 dpi; LL_AUDPC = lesion lengths area under disease progress curve (AUDPC) calculated using 7 time points reading; LW_AUDPC = lesion widths AUDPC calculated using 7 time points reading.

5.5. Discussion

Sclerotinia stem rot is one of the destructive fungal diseases in rapeseed/canola. Lack of complete immune genotypes against this pathogen makes it difficult to improve SSR resistance using only classical breeding methods. Genomics-assisted breeding approaches such as GWA mapping and GP may be especially useful for the genetic improvement of complex traits by

reducing the required breeding time compared with classical breeding (Varshney et al., 2005). Therefore, we implemented GWA and GP approaches to study the complexly inherited *S. sclerotiorum* resistance in rapeseed/canola to facilitate the future genomics-enabled SSR resistance breeding.

In GWA study, the power of QTL detection is not only dependent on the sample size, but also on the underlying genetic architecture of the trait, cryptic population structure, heritability of the trait and statistical models (Yu et al., 2006; Josephs et al., 2017). Therefore, precise phenotypic evaluation with appropriate inoculation method for the trait of interest is crucial. To obtain reliable phenotypic data, we have evaluated our studied *B. napus* populations under optimized agar plug stem inoculation procedures in controlled environments. Throughout the study, we used a single highly virulent *S. sclerotiorum* isolate WM031 (Roy et al., 2021; Shahoveisi et al., 2021), because the isolates of this pathogen vary in aggressiveness (Garg et al., 2010; Taylor et al., 2015). The inoculation was done using the stem inoculation method during the full flowering stage which is the most prevalent stage of SSR infection in rapeseed/canola (Wu et al., 2013; Roy et al., 2021). Moreover, the used agar-plug stem inoculation technique mimics the natural SSR infections in *B. napus* plants and used successfully by many researchers to distinguish responses of rapeseed/canola lines inoculated by *S. sclerotiorum* (Li et al., 2006; Qasim et al., 2020; Roy et al., 2021; Shahoveisi et al., 2021; Derbyshire et al., 2021). In the present study, resistance performance of the inoculated spring, semi-winter, and winter types genotypes were evaluated by taking the measurement of main stem lesion length, lesion width starting at 3 dpi and continued on each alternate day up to 15 dpi. As a day advanced after plant infection by the pathogen, susceptible genotypes started to die, which resulted in loss of data points as we stopped data taking on LL and LW once the plants were considered dead. However,

LL and LW data at 7 dpi were selected for both populations because at this single time point the prominent phenotypic differences were observed among the genotypes without sacrificing data points due to the increase of plant mortality. The use of single reading of lesion length developed on the main stem, usually recorded between 3-9 dpi, is commonly used for bi-parental linkage mapping and GWA analyses for SSR resistance in rapeseed/canola (Yin et al., 2010; Wu et al., 2013, 2016; Gyawali et al., 2016; Wei et al., 2016; Qasim et al., 2020; Roy et al., 2021; Shahoveisi et al., 2021). In addition to the commonly used stem LL data, we also recorded lesion width (LW) at 7dpi, and plant mortality (PM) at 15 dpi. Data on LW (Roy et al., 2021) and PM (Roy et al., 2021; Shahoveisi et al., 2021) were successfully used to differentiate the performance of the susceptible and resistant genotypes upon *S. sclerotiorum* attack. Moreover, we also calculated AUDPC considering 7 readings data points for stem LL (LL_AUDPC) and LW (LW_AUDPC). Pearson correlation analyses among the five traits in both populations revealed strong associations among the traits. The strong associations among the SSR resistance traits clearly demonstrated that each phenotypic trait complement each other and use of any of the traits out of five could be used for large scale phenotyping of *B. napus* genotypes against *S. sclerotiorum* reaction. Moreover, instead of data on different time points (7 time points in this study), a single time point *i.e.*7 dpi LL and LW scoring would be enough for SSR phenotyping. This would ultimately reduce the excessive workloads related to data collection, and would facilitate the large-scale screening of *B. napus* germplasm against *S. sclerotiorum* infection.

In the current study, two approaches such as (i) multiple phenotypic scoring systems and (ii) two GWA algorithms (GEMMA-MLM and FarmCPU) were applied to improve the detection power and robustness of GWA mapping to identify the MTAs associated with SSR resistance. In GWA study, the control of false positives is very crucial, but the effect of false negatives also

should not be ignored. To reduce the chance of committing Type I (false positives) and Type II errors (false negatives), two GWA algorithms *i.e.* GEMMA-MLM, and FarmCPU were implemented by incorporating PCA and kinship matrix to control the confounding effect of population structure and familial relatedness. Moreover, the use of stringent Bonferroni-Holm correction (Holm 1979) as a threshold value to determine significant MTAs could lead to detection of spurious associations due to false negatives (Type II error). Therefore, the *P*-value threshold to declare association between SNPs and trait of interests were determined on the basis of modified Bonferroni correction by calculating the effective number of independent loci from the tested SNP markers for both populations according to the proposed method by Li and Ji (2005). Thirty-seven (37) SNPs were commonly mapped by at least two or more traits by any of the two GWA models were considered stable and significantly associated with SSR resistance. Likewise to the spring population, SNPs detected in two or more traits were regarded as significant and the total 50 SNPs as significant were determined. The use of multiple phenotypic traits, one single locus (GEMMA-MLM), and one multi-locus (FarmCPU) GWA model in the current study contributed to the detection of additional significant SNPs compared to the use of single trait. Similar trends were also demonstrated by Shahoveisi et al. (2021) for linkage mapping study in controlled environment and Roy et al. (2021) for association mapping study under field environment for SSR resistance in rapeseed/canola. Moreover, detection of common or overlapping significant SNPs across multiple scoring systems and multiple methods for the trait of interest could provide more confidence and reliability of the detected MTAs to facilitate future MAS.

Comparison of identified SNPs revealed that no SNPs were found exactly in the same physical location in both populations. The discrepancy could be explained by different mapping

populations, different *B. napus* ecotypes, and application of vernalization to induce flowering. Zhao et al. (2006) evaluated two double haploid (DH) populations using petiole inoculation technique (PIT) to identify QTL for SSR resistance. They detected a total of 9 QTLs, however no common QTL were identified between two populations. Three GWA mapping (Gyawali et al., 2016; Wei et al., 2016; Wu et al., 2016) in *B. napus* using stem inoculation method (detached stem or intact plant stem) for SSR resistance identified numerous resistance loci located on different chromosomes. However, none of the resistance loci were shared among the three GWA results. In these three studies, different forms of stem inoculation methods (detached stem/intact plant stem), and the measurement of stem lesion length at 3 dpi (Wei et al., 2016), 5 dpi (Wu et al., 2016), and 7,14, and 21 dpi (Gyawali et al., 2016) were used for phenotyping of rapeseed/canola genotypes against *S. sclerotiorum* infection. This further explained that differences in phenotyping method, and *B. napus* germplasm collection contribute to variation of the detected SNPs/genomic regions among the various mapping studies. Moreover, the effect of vernalization on flowering induction in the semi-winter and winter populations could lead to the variation in the detected MTAs. Several studies have reported that vernalization of overwintering crops can enhance the disease resistance to several pathogens. Associations between vernalization and increased disease resistance has been reported in the various pathosystems, like winter wheat and triticale- *Puccinia striiformis* (Rodriguez-Algaba et al., 2020), winter cereals- *Microdochium nivale* and *Typhula ishikariensis* (Tronsmo, 1986; Hofgaard et al., 2006; Kuwabara and Imai, 2009), winter barley-*Blumeris graminis* f.sp. *hordei* (White and Jenkyn, 1995). Application of vernalization treatment in semi-winter and winter type populations may contribute to the variation of SSR phenotyping which might ultimately affect the detection of MTAs. Therefore, differences in the identified SNPs in two populations could be due to the

different ecotypes of *B. napus* germplasm collection, and effect of vernalization. Therefore, future research is necessary to elucidate whether vernalization in semi-winter and winter genotypes might have an effect in contributing resistance to *S. sclerotiorum* attack.

Few SNPs mapped in multiple traits in both populations were found to be associated in the close proximity in the *B. napus* genome. In spring population, four SNPs (SCM002771.2_1951273, SCM002771.2_2495279, SCM002771.2_4764427, SCM002771.2_5119497) mapped in multiple traits and methods were positioned between 1.95-5.12 Mb genomic regions on chromosome C03, were in close proximity of the SNP SCM002771.2_5548449 (C03, 5.55 Mb) found to be associated with multiple traits in semi-winter and winter populations. The simultaneously the SNP SCM002774.2_32559985 by multiple phenotypic traits and GWA methods on C06 (32.56 Mb) in semi-winter and winter population was located ~779 kb apart from the SNP SCM002774.2_33338934 with multiple traits in spring population. Mapping of these two SNPs on C06 (32.56-33.34 Mb) in the current studied two populations overlapped with the defined conserved QTL regions (29.5-36.1 Mb) by Li et al. (2015) and other detected QTNs or QTLs genomic regions for SSR resistance (Zhao et al., 2006; Wu et al., 2013; Qasim et al., 2020; Roy et al., 2021). Another two SNPs (SCM002775.2_9203423, SCM002775.2_11411683) detected in spring population, located on C07 in the physical genomic regions of (9.20-11.41 Mb) were found near to the SNP SCM002775.2_12215584 (12.22 Mb) in semi-winter and winter type populations.

Classical linkage and GWA mapping for SSR resistance have identified a number of QTLs/SNPs located in the different chromosomes in *B. napus* genome (Zhao and Meng, 2003; Zhao et al., 2006; Yin et al., 2010; Wu et al., 2013, 2019; Wei et al., 2014; Zhang et al., 2019; Qasim et al., 2020; Shahoveisi et al., 2021). The previously reported QTNs/QTLs physical

position was determined based on '*Darmor-bzh*' reference genome sequence (Chalhoub et al., 2014). In the present study, we map the physical location of the SNPs using '*ZS11*' as *B. napus* reference genome sequence (Sun et al., 2017), which was aligned with '*Darmor-bzh'* reference genome sequence. Therefore, the identified SNPs in the current study found within ~500 kb of the previously mapped QTNs/QTLs were considered as the same loci that could be associated with SSR resistance. Thus 10 and 12 QTNs detected in our GWA analyses in Study_1 and Study_2 populations, respectively corresponded to previously reported SNPs and QTLs detected based on linkage and/or association mapping. These QTNs/QTLs simultaneously detected in different populations with different genetic backgrounds, different phenotypic and GWA methods, could potentially be exploited and integrated in the MAS for target traits of interest. Moreover, additionally detected novel QTNs with multiple SSR resistance traits, and GWA methods in the current study, may contribute to better understanding of the architecture of *S. sclerotiorum* reaction and could provide more opportunities conferring SSR resistance breeding in rapeseed/canola. These detected putative genes from the current study would provide opportunities for future gene cloning and functional verifications as a candidate for controlling *S. sclerotiorum* resistance in rapeseed/canola.

The results from this study provide insight into the potential of genomic prediction of adult plant SSR resistance in rapeseed/canola. The potential of GS has been assessed for simple and complex traits in rapeseed/canola (Würschum et al., 2014; Jan et al., 2016; Werner et al., 2018; Fikere et al., 2020; Roy et al., 2021; Derbyshire et al., 2021). GS allows to capture the contribution of small and large effect loci distributed throughout the genome. The resulted predictive ability of spring populations in the current study is in accordance with the previous study by Roy et al. (2021), and higher than that of mean predictive ability resulted from

Derbyshire et al. (2021), and Wu et al. (2016) findings. However, the predictive ability was low in semi-winter and winter ecotypes populations. The differences in predictive ability could be attributed to their low heritability, impact of vernalization on SSR phenotyping. Therefore, it is necessary to implement an inoculation method which would allow to evaluate different ecotypes of *B. napus* germplasm during *S. sclerotiorum* infection at the same developmental stage without being affected by any other physiological effects such as vernalization requirement. However, future investigations are warranted to determine whether vernalization of *B. napus* genotypes have any effect on physiological resistance to SSR disease.

The extensive phenotypic evaluation of diverse *B. napus* germplasm in response to *S. sclerotiorum* attack revealed a few promising genotypes of different ecotypes with moderate levels of resistance against this pathogen. These resistance sources would serve as a valuable resource to transfer and pyramid the SSR resistant genes/QTLs into the elite cultivars leading towards the development of durable SSR resistant canola cultivars for the growers. GWA scan in two studied populations identified few overlapping/co-localized SNPs with the previously reported SNPs/QTLs as well novel significant SNPs associated with SSR resistance, would provide exciting opportunities to explore their potential in integrating into the MAS resistance breeding in rapeseed/canola. However, further research is warranted on validating the effects of identified SNPs, candidate genes that these QTNs and genes engender resistance to *S. sclerotiorum* infection. Impact of vernalization on SSR resistance phenotyping also needs further verification to be used in future for screening. GS results revealed higher (spring population) and low to medium (semi-winter and winter population) predictive ability could be implemented to accelerate the breeding cycle by selecting SSR resistant genotypes at early growth stages.

CHAPTER 6: SUMMARY

In this research, we investigated the phenotypic performance of a diverse panel of *Brassica napus* germplasm collections consisting of spring, semi-winter, and winter ecotypes against *Sclerotinia sclerotiorum* disease infection. Extensive screening of the studied genotypes under field and greenhouse environments were carried out using agar plug intact plant stem inoculation method (greenhouse and field environments) for adult plant resistance, and petiole inoculation technique (PIT) (greenhouse) for seedling resistance. Both inoculation methods showed statistically significant differences among the genotypes for all phenotypic traits in all experiments. This clearly demonstrates that these methods are reliable, repeatable, and can be used successfully to distinguish the response of *B. napus* accessions to *S. sclerotiorum* infection. In addition, we also assessed the effectiveness of multiple sclerotinia stem rot (SSR) phenotypic traits to evaluate the phenotypic response of genotypes in response to the *S. sclerotiorum* infection. Finally, using the multiple phenotypic traits from each experiment, we expedited two strategies, genome-wide association (GWA) mapping to identify significant markers associated with trait of interests, and genomic prediction (GP) to utilize all marker effects to predict the performance of SSR resistant genotypes at seedling and adult plant stage.

In each experiment, we recorded multiple phenotypic traits for the assessment of rapeseed/canola lines against *S. sclerotiorum* infection. In field study, a total of 187 *B. napus* lines were characterized for adult plant resistance using intact stem inoculation method in four environments. Phenotypic measurements were done by taking the commonly used stem lesion length at 7 days post inoculation (dpi) with three additional phenotypic traits such as lesion width at 7 dpi, plant mortality at 14 and 21 dpi, for the evaluation of corresponding genotypes against SSR resistance. Strong correlations observed among the traits which suggest that they could be

used as proxies for SSR phenotyping. Moreover, we also investigated the relationships between SSR disease phenotypic traits with three physiological traits such as flowering time (FT), stem diameter (SD), and stem internode length (IL) under field environments. The FT and SD were negatively, and IL was positively correlated with the SSR phenotypic traits. We also characterized our *B. napus* association panel consisting of 337 lines for seedling resistance using PIT two times in the greenhouse. Phenotypic response of the infected plants was determined by days to wilt (DW) and scored on each day up to two weeks. The DW was strongly correlated with lesion phenotypes (LP) (scored using a categorical scale based on developed lesions on the main stem) recorded on 3, 4, and 7 dpi. DW had the highest associations with LP scores at 4dpi. This implied that single time point scoring data (LP_4dpi) could be used as a potential proxy trait instead of DW for large scale phenotyping of SSR resistant genotypes, which would eliminate the excessive workload associated with SSR phenotyping. Moreover, adult plant resistance against *S. sclerotiorum* were also characterized under greenhouse environments including 144 spring and 152 semi-winter and winter types in two separate experiments. Screening of each population was done two times. Multiple traits such as lesion length, lesion width, and plant mortality were collected as phenotype response for the evaluation of genotypes. Similar to field study, highly significant correlations existed among the phenotypic traits in spring, and semi-winter and winter populations. Extensive phenotypic evaluation in the field and greenhouse environments helped us to identify few promising lines conferring resistance to SSR at both seedling and adult developmental stages. However, developmental stage specific disease resistance genotypes were also identified. In the present study, the promising resistant lines showed better resistance performance than the used commercial resistant check cultivars.

In field study, genome-wide association (GWA) analyses using three algorithms identified 133 significant SNPs for four traits associated with SSR resistance. The explained phenotypic variation of these SNPs varied from 3.6-12.1%. Among these, 19 SNPs were mapped in two or more environments, disease traits and at least by two GWA algorithms. Candidate gene search based on disease resistance annotated 69 putative genes found within ± 50 kb genomic interval of the significant SNPs. In case of seedling resistance using PIT inoculation method, one single-locus (GEMMA-MLM) and three multi-locus (MLMM, FarmCPU, mrMLM) GWA mapping models with 27, 282 SNP markers mapped a total of 79 significant SNPs detected by at least two traits or two models, explaining 1.35-13.30% of the phenotypic variance with multiple phenotypic traits. Eighty-one putative annotated candidate genes associated with disease resistance mechanisms corresponding to the significant SNPs were found to underlie SSR resistance. In the greenhouse, we phenotyped 144 spring types population (SP), 152 semi-winter and winter types populations (SWP) using stem inoculation method at the full flowering stage. GWA mapping with two models and five phenotypic traits identified 37 and 50 significant SNPs in SP, and SWP, respectively. Candidate gene search using ± 50 kb flanking genomic regions around the significant SNPs revealed a total of 35, and 46 for SP and SWP respectively. The putative annotated candidate genes known to have plant disease resistance mechanism, which could be associated with SSR resistance. The defined conserved QTL genomic regions for SSR resistance were positioned on chromosomes A09 (22.5-27.5 Mb) and C06 (29.5-36.1 Mb). In this study, we mapped 3 significant SNPs on chromosome A09 (21.5-27-7 Mb), which is located near to the defined conserved QTL regions. A total of 9 significant SNPs detected from all experiments were positioned on chromosome C06 (31.7-34.1 Mb) genomic regions, which overlapped with the conserved SSR resistance QTL genomic regions. We simultaneously

detected few significant SNPs, which are overlapped in the certain genomic regions on chromosomes from different experiments, different environments and different inoculation methods. These narrowed down genomic regions were located on chromosome A09 (33.3-39.1 Mb) consisting of 20 significant SNPs, C02 (59.2-62.8 Mb) with 7 significant SNPs, and C6 (31.7-37.7 Mb) with 10 significant SNPs (Figure 6.1). These genomic regions may contribute to better understanding of the genetic architecture of *S. sclerotiorum* and could provide more opportunities for SSR resistance breeding in rapeseed/canola. A TIR-NB-LRR gene encodes *RPP1* proteins, detected in both field (adult plant resistance) and greenhouse (seedling resistance) study were found as a potential candidate gene could be associated with SSR resistance. Moreover, we also identified *WRKY* transcription factor 33 (*WRKY33*) as a potential candidate gene, which is known to be involved in providing resistance against *S. sclerotiorum* infection.

We also explored and evaluated the effectiveness of genomic prediction (GP) for predicting SSR resistant genotypes. Under field environments, the GP models resulted in moderate to high predictive ability in predicting adult plant stage SSR resistance. Plant mortality trait yielded the highest predictive ability compared to the stem lesion length and lesion width phenotypic traits. In terms of seedling stage SSR resistance, medium to high predictive ability resulted from the four phenotypic traits predicted by six genomic selection models. All the six GP models gave similar results and none of the models outperform others in terms of achieving high predictive ability. Single time point scoring data LP_3dpi and LP_4dpi resulted in higher predictive ability compared to DW. In the case of adult plant resistance under greenhouse environment, moderate to high, and low to moderate predictive ability in SP and SWP, respectively, were observed. Our study concludes that the genomics-assisted approach i.e. GWA

mapping and genomic prediction hold promise and could be implemented for the genetic improvement of complexly inherited SSR resistance in rapeseed/canola to achieve higher predictive ability and rapid gains from selection, thereby reducing the required time compared with conventional breeding.

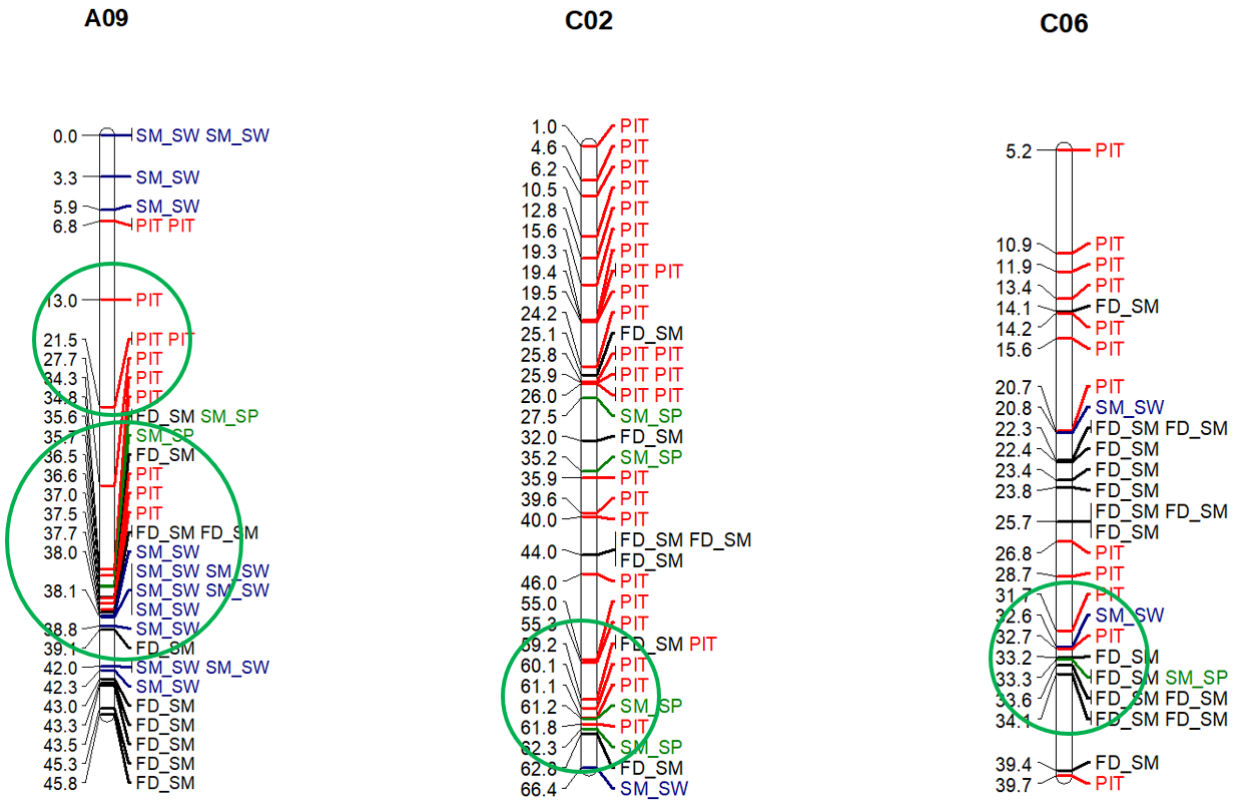

Figure 6.1. Significant markers detected from the field and greenhouse study at seedling and adult plant stage neighboring on the same chromosome. FD_SM, field study using stem inoculation method at adult plant stage; PIT, greenhouse study at the seedling stage using petiole inoculation technique; SM_SP, greenhouse study at the adult plant stage using stem inoculation method for spring growth habit genotypes; SM_SW, greenhouse study at the adult plant stage using stem inoculation method for semi-winter and winter growth habit genotypes. Map were constructed using MapChart software (Voorrips, 2002).

CHAPTER 7: FUTURE DIRECTION

These resistance genotypes will serve as a valuable resource for NDSU canola breeding program to transfer the resistant genes into elite cultivars leading towards the development of high yielding canola cultivars with improved SSR resistance for the canola growers. Multi-parent advanced generation inter-cross (MAGIC) lines could be generated using these SSR resistant lines, together with other parents carrying economically important traits, with an aim to incorporate all the useful traits into a single background. These MAGIC lines could be used for various genetic mapping studies and as a potential parent to extract canola cultivars with multiple useful traits. The development of MAGIC lines is in progress including identified SSR resistant genotypes from the current study.

We have simultaneously detected new genomic regions across different experiments, different inoculation methods and different environments. These narrowed down genomic regions were located on chromosome A09 (33.3-39.1 Mb), C02 (59.2-62.8 Mb), and C6 (31.7-37.7 Mb). These genomic regions may contribute to better understanding of the genetic architecture of *S. sclerotiorum* reaction and could provide more opportunities for SSR resistance breeding in rapeseed/canola. Therefore, future efforts would be directed to validate the identified SNPs and develop tightly linked markers to facilitate the marker-assisted-selection (MAS) resistance breeding in rapeseed/canola. SNP validation could be done by converting the identified SNPs into kompetitive allele-specific PCR (KASP) markers. Semi-thermal asymmetric reverse PCR (STARP) also provides a new flexible, scalable, and cost-effective approach for using SNP markers in MAS. Another approach would be to perform gene expression analyses by collecting RNA samples from the identified highly resistant and susceptible lines by infecting with *S. sclerotiorum*. The repeatedly detected putative disease resistance candidate genes could

be selected as potential candidate genes to observe their differential gene expression in the contrasting phenotyped rapeseed/canola lines upon *S. sclerotiorum* infection. In addition to that, gene editing with CRISPR could be used to validate the putative function of the detected candidate genes. This could be done by knocking-out the disease resistant candidate genes, and by observing the phenotype of the corresponding lines.

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APPENDIX

Table A.1. Name, origin, and growth habits of the 189 germplasm accessions (including check cultivars) used in this study

Name of the accessions	Country of origin/obtained	Growth habit
Pioneer 45S51[a]	Pioneer (Check)	Spring
Pioneer 45S56[a]	Pioneer (Check)	Spring
Galant	USA	Spring
Galaxy	Sweden	Spring
Gido	Germany	Spring
Girita	Germany	Semi-winter
Gisora	Germany	Spring
Global	Sweden	Spring
Golden	Canada	Spring
Gora	Germany	Spring
Gulle	Sweden	Spring
Gullivar	Sweden	Spring
Gylle	South Korea	Semi-winter
Helga	Germany	Semi-winter
Hi-Q	Canada	Spring
INRA-R-2000	France	Spring
IR-2	Hungary	Spring
Janetzkis	South Korea	Spring
Jasna	Serbia	Spring
Kanada	Poland	Spring
Klinki	South Korea	Spring
Kosa	Germany	Spring
Kovalevskjj	Ukraine	Spring
Kraphhauser	South Korea	Spring
Kritmar rape	South Korea	Spring
Laura	Germany	Spring
Legend	Sweden	Spring
Lieikoposki	South Korea	Semi-winter
Aviso	Canada	Spring
Lifura	South Korea	Spring
Azuma	South Korea	Semi-winter

Table A.1. Name, origin, and growth habits of the 189 germplasm accessions (including check cultivars) used in this study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Lisora	Germany	Semi-winter
Major	France	Semi-winter
Mali	South Korea	Semi-winter
Azumasho	South Korea	Semi-winter
Mar'janovskij	Ukraine	Spring
Matador	South Korea	Semi-winter
Mazowiecki	Poland	Spring
Midas	Canada	Spring
Miekuro Dane	South Korea	Spring
Miochowski	France	Semi-winter
Mlochowski	Poland	Semi-winter
Mura yamasho	South Korea	Spring
Murame nadame	South Korea	Semi-winter
N001-28-246-5-4	South Korea	Semi-winter
NDC-A14026	USA	Spring
NDC-A14032	USA	Spring
NDC-A14033	USA	Spring
NDC-A14035	USA	Spring
NDC-A14036	USA	Spring
NDC-A14045	USA	Spring
NDC-A14046	USA	Spring
NDC-A14050	USA	Spring
NDC-A14055	USA	Spring
NDC-A14056	USA	Spring
NDC-E12009	USA	Spring
NDC-E12023	USA	Spring
NDC-E12025	USA	Spring
NDC-E12027	USA	Spring
NDC-E12044	USA	Spring
NDC-E12079	USA	Spring
NDC-E12081	USA	Spring

Table A.1. Name, origin, and growth habits of the 189 germplasm accessions (including check cultivars) used in this study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
NDC-E12086	USA	Spring
NDC-E12119	USA	Spring
NDC-E12120	USA	Spring
NDC-E12121	USA	Spring
NDC-E12131	USA	Spring
NDC-E12133	USA	Spring
NDC-E13193	USA	Spring
NDC-E13279	USA	Spring
NDC-E13285	USA	Spring
NDC-E15031	USA	Spring
NDC-E15146	USA	Spring
NDC-E15174	USA	Spring
NDC-E15200	USA	Spring
NDC-E15234	USA	Spring
NDC-E15294	USA	Spring
NDC-E16015	USA	Spring
NDC-E16053	USA	Spring
NDC-E16152	USA	Spring
NDC-E16169	USA	Spring
NDC-E16198	USA	Spring
NDC-E17132	USA	Spring
NDSU01104	USA	Spring
NDSU0417	USA	Spring
NDSU0472	USA	Spring
NDSU0473	USA	Spring
NDSU0474	USA	Spring
NDSU0475	USA	Spring
NDSU0521	USA	Spring
NDSU0522	USA	Spring
NDSU0619	USA	Spring
NDSU0620	USA	Spring
NDSU0726	USA	Spring

Table A.1. Name, origin, and growth habits of the 189 germplasm accessions (including check cultivars) used in this study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
NDSU0728	USA	Spring
NDSU0729	USA	Spring
NDSU10999	USA	Spring
NDSU12989	USA	Spring
NDSU151000	USA	Spring
NDSU15989	USA	Spring
NDSU161013	USA	Spring
NDSU31001	USA	Spring
NDSU31011	USA	Spring
Bingo	USA	Spring
NDSU41000	USA	Spring
NDSU7997	USA	Spring
NDSU81000	USA	Spring
NDSU91013	USA	Spring
Nilla 1022	South Korea	Semi-winter
Nilla glossy	South Korea	Semi-winter
NU 41737	Turkey	Spring
NU 51084	Sweden	Spring
Nugget	South Korea	Semi-winter
NY-12	China	Semi-winter
NY-20	China	Semi-winter
NY-7	China	Semi-winter
NY-8	China	Semi-winter
O 84	China	Semi-winter
BO-63	Canada	Spring
Oro	Canada	Spring
Orpal	France	Spring
Peace	Canada	Spring
Petanova-lihonova	South Korea	Semi-winter
Polo canola	USA	Spring
Premier	USA	Spring
Printol	USA	Spring

Table A.1. Name, origin, and growth habits of the 189 germplasm accessions (including check cultivars) used in this study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Prota	Germany	Spring
Q2	Canada	Spring
Rang	South Korea	Semi-winter
Ratnik	Serbia	Spring
Rebel	USA	Semi-winter
Regent	Canada	Spring
Regina II	Canada	Spring
Reston	USA	Spring
Rico	Germany	Spring
Romeo	France	Spring
Russia 5	Russian Federation	Spring
S.V. Gulle	South Korea	Spring
Seoul	South Korea	Spring
Sera	Germany	Semi-winter
Silex	Canada	Spring
Brio	France	Spring
Su weon chag	South Korea	Semi-winter
Sunrise	USA	Spring
Sval of Gullen	South Korea	Spring
Taichang	South Korea	Semi-winter
Bronowski	Poland	Spring
Taiwan	Taiwan	Spring
Tanka	South Korea	Semi-winter
Tanto	France	Spring
Target	Sweden	Spring
Todane	South Korea	Semi-winter
Buk Wuk 3	South Korea	Spring
Tokiwa	South Korea	Semi-winter
Tonus	South Korea	Spring
Topas	Sweden	Spring
Tower	Canada	Spring
Turret	Canada	Spring

Table A.1. Name, origin, and growth habits of the 189 germplasm accessions (including check cultivars) used in this study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Vostochno-sibirskii	Russian Federation	Spring
Wasefuji	South Korea	Spring
Weal dong cho	South Korea	Semi-winter
Westar[b]	**Canada**	**Spring**
Willa	South Korea	Spring
Wipol	Norway	Semi-winter
Yong dang	South Korea	Semi-winter
Zhoungyou-821	China	Semi-winter
Cescaljarni repka	South Korea	Semi-winter
Ceskia Tabor	Czech Republic	Spring
Chisaya natane	Japan	Semi-winter
Colt	USA	Spring
Colza	South Korea	Spring
Colza 18 Miroc	South Korea	Semi-winter
Comet	Sweden	Spring
Conquest	Canada	Spring
Cougar	Canada	Spring
Cresor	France	Spring
Cresus	France	Spring
Czyzowski	Poland	Spring
Czyzowskich	Poland	Semi-winter
Dae cho sen	South Korea	Semi-winter
Delta	Sweden	Spring
Drakkar	France	Spring
Eckendorfer Mali	South Korea	Semi-winter
Evvin	Russian Federation	Spring
Flint	USA	Spring
Fonto	South Korea	Spring
France 1	France	Spring
Fu 58 Drakkar	France	Spring
Fuji	South Korea	Spring/ semi

[a] Accessions used as resistant check
[b] Accession used as susceptible check

Table A.2. Results for ANOVA of sclerotinia stem rot resistance traits and other traits in each environment and combined across environments.

SOV[a]	Environment[b]	LL	LW	PM_14D[c]	PM_21D[c]	SD	IL	DF
Genotype	CARR_19	***	***	----	----	***	***	***
Genotype	LANG_19	***	***	----	----	***	***	***
Genotype	CARR_20	***	***	----	----	***	***	***
Genotype	OSN_20	***	***	----	----	***	***	***
Genotype	CombENV	***	***	***	***	***	***	***
Genotype x Env.	CombENV	***	***	***	ns	***	***	***

*** and ns indicate differences were significant at $P \leq 0.0001$ levels of significance, and not significant

SOV[a], source of variation; Env., environment; LL, lesion length; LW, lesion width; PM_14D, plant mortality at 14 days post inoculation (dpi); PM_21D, plant mortality at 21 dpi; SD, stem diameter; IL, internode length; DF, days to flowering.

Environment[b]: CARR_19, carrington 2019; CARR_20, carrington 2020; LANG_19, langdon 2019, OSN_20, osnabrock 2020, CombENV, combined across all environments

PM_14D[c] and PM_21D[c], combined analyses across all environments

Table A.3. Marker-trait-associations (MTAs) with stem lesion length (cm) for sclerotinia stem rot resistance in 187 canola/rapeseed genotypes analyzed by the FarmCPU, MLM, and GEMMA-MLM models

Locus	Chr[a]	SNP ID	Position (bp)	Alleles[b]	-log$_{10}$ (*P*)[c]	Env & Model[d]	Allelic effect[e]	R^2 [f]
1	1	SCM002759.2_1471693	1471693	A/T	4.3-3.1	E1^{123}, E2^{23}, E4^{23}, CombENV^{123}	-0.30, -0.22	4.8-7.0
2	1	SCM002759.2_1766199	1766199	A/G	3.6-3.0	E2^{123}, CombENV^{123}	0.47, 0.37	4.6-5.6
3	1	SCM002759.2_4239970	4239970	A/C	4.8-3.3	E2^{123}, E4^{23}, CombENV^{23}	-0.34; -0.25	5.4-7.3
4	1	SCM002759.2_5478454	5478454	C/T	3.5-3.3	E2^{23}	0.38	5.1
5	1	SCM002759.2_11482241	11482241	C/T	3.1-3.4	E1^{123}, E3^{3}, CombENV^{13}	-0.28; -0.27	5.4
6	2	SCM002760.2_1865667	1865667	A/C	4.8-3.0	E2^{23}, E4^{123}, CombENV^{23}	-0.70, -0.43	4.5-7.7
7	2	SCM002760.2_10378033	10378033	G/A	3.5-3.3	E4^{23}	-0.28	5.3
8	2	SCM002760.2_30827371	30827371	C/G	3.6-3.1	E3^{123}	-0.22; -0.21	6.2
9	3	SCM002761.2_17320071	17320071	T/A	3.8-3.1	E2^{123}	-0.56; -0.51	4.8
10	3	SCM002761.2_28225133	28225133	T/C	3.7-3.4	E3^{123}	-0.36	7.2
11	3	SCM002761.2_30742237	30742237	T/C	3.4-3.0	E1^{123}, CombENV^{3}	0.41	5.4
12	3	SCM002761.2_35236913	35236913	C/A	3.5-3.2	E3^{123}	-0.20	6.7
13	3	SCM002761.2_36170515	36170515	G/A	5.4-4.0	E1^{123}, E3^{123}, CombENV^{123}	0.26, 0.23	7.0-9.9
14	4	SCM002762.2_3551539	3551539	C/T	3.4-3.0	E4^{123}, CombENV^{123}	0.61, 0.45	4.8-5.2
15	4	SCM002762.2_20212672	20212672	T/A	4.5-3.0	E3^{123}, CombENV^{13}	-0.56; -0.44	5.0-8.6
16	5	SCM002763.2_15914063	15914063	G/A	5.2-3.0	E2^{123}, E4^{123}, CombENV^{123}	-0.59; -0.33	5.0-8.0
17	5	SCM002763.2_16279322	16279322	A/C	3.7-3.1	E3^{123}, CombENV^{23}	-0.39; -0.34	5.1-7.2
18	5	SCM002763.2_28142332	28142332	A/G	3.9-3.1	E2^{123}	0.24; 0.27	4.7
19	5	SCM002763.2_28608175	28608175	A/G	3.5-3.3	E2^{123}	0.42; 0.38	5.1
20	6	SCM002764.2_25241979	25241979	C/A	3.3-3.0	E1^{13}, E3^{23}, CombENV^{123}	-0.47; -0.45	5.0-5.9
21	7	SCM002765.2_25991221	25991221	G/C	3.7-3.1	E1^{123}, E2^{123}, CombENV^{3}	-0.63; -0.51	5.4-5.5
22	8	SCM002766.2_1982656	1982656	G/C	4.6-3.1	E3^{123}, E4^{123}, CombENV^{123}	-0.57; -0.38	7.0-7.5
23	8	SCM002766.2_13657808	13657808	T/G	3.5-3.0	E3^{123}	-0.41	6.0
24	8	SCM002766.2_13864870	13864870	A/G	3.7-3.1	E2^{123}, E4^{123}, CombENV^{123}	0.67, 0.47	5.2-5.7
25	9	SCM002767.2_37664281	37664281	A/G	3.9-3.7	E4^{123}	-0.34	6.2

Table A.3. Marker-trait-associations (MTAs) with stem lesion length (cm) for sclerotinia stem rot resistance in 187 canola/rapeseed genotypes analyzed by the FarmCPU, MLM, and GEMMA-MLM models (continued)

Locus	Chr[a]	SNP ID	Position (bp)	Alleles[b]	-log$_{10}$ (P)[c]	Env & Model[d]	Allelic effect[e]	R^2 [f]
26	9	SCM002767.2_37671479	37671479	G/A	3.6-3.1	E1^{123}, E2^{1}	-0.31; -0.27	5.9
27	9	SCM002767.2_39128782	39128782	T/C	3.6-3.2	E1^{123}, CombENV^{123}	-0.60; -0.56	5.4-6.0
28	9	SCM002767.2_43041855	43041855	C/T	3.8-3.5	E3^{123}	0.65	7.1
29	9	SCM002767.2_45297735	45297735	T/C	4.0-3.3	E2^{123}, E4^{13}, CombENV^{2}	-0.47; -0.33	5.1-6.1
30	9	SCM002767.2_45841268	45841268	A/T	3.5-3.0	E3^{123}, CombENV^{13}	0.44, 0.43	6.0
31	11	SCM002769.2_6223969	6223969	T/C	4.4-3.3	E1^{123}, E3^{123}, CombENV^{123}	-0.45; -0.38	6.0-7.3
32	11	SCM002769.2_46192098	46192098	T/C	4.2-3.0	E2^{123}, E4^{23}, CombENV^{23}	-0.74; -0.52	4.9-6.4
33	12	SCM002770.2_25096010	25096010	A/T	3.6-3.0	E3^{123}, CombENV^{1}	0.50, 0.49	6.1
34	12	SCM002770.2_32020747	32020747	A/G	4.0-3.0	E2^{123}, E4^{13}, CombENV^{123}	0.46, 0.34	5.1-6.0
35	12	SCM002770.2_62791159	62791159	T/C	3.6-3.3	E3^{123}	-0.35; -0.34	7.0
36	13	SCM002771.2_7893201	7893201	C/G	5.0-3.1	E2^{123}, E4^{123}, CombENV^{13}	0.78, 0.47	5.7-7.9
37	13	SCM002771.2_22853068	22853068	G/A	3.7-3.0	E2^{123}, E4^{123}, CombENV^{123}	-0.50; -0.38	4.9-5.3
38	13	SCM002771.2_45250459	45250459	T/G	3.8-3.5	E1^{123}	0.26	6.2
39	13	SCM002771.2_56900783	56900783	G/A	3.8-3.0	E1^{123}, E3^{123}, CombENV^{13}	0.40, 0.34	4.9-7.4
40	13	SCM002771.2_69833085	69833085	C/T	3.7-3.2	E2^{23}	0.40	5.6
	13	SCM002771.2_69833922	69833922	C/G	3.4-3.2	E2^{23}	0.37	5.0
41	13	SCM002771.2_78761785	78761785	G/T	3.5-3.0	E4^{3}, CombENV^{123}	0.68, 0.63	5.3
42	13	SCM002771.2_80035604	80035604	C/T	3.4-3.0	E1^{3}; E3^{3}; CombENV^{23}	0.64	5.3
43	14	SCM002772.2_65359864	65359864	C/T	4.5-3.1	E1^{123}, E2^{13}, CombENV^{123}	0.82; 0.72	6.6-7.6
44	15	SCM002773.2_29580386	29580386	A/T	3.7-3.0	E3^{3}, E4^{23}, CombENV^{23}	0.78, 0.60	5.1-6.0
45	15	SCM002773.2_38993115	38993115	A/C	3.9-3.0	E1^{123}, CombENV^{23}	0.51, 0.44	5.0-6.6
46	16	SCM002774.2_22279509	22279509	A/G	3.8-3.0	E4^{123}, CombENV^{123}	-0.45, -0.28	4.9-5.7
47	16	SCM002774.2_22321613	22321613	C/A	3.5-3.1	E4^{13}, CombENV^{1}	0.39, 0.26	
48	16	SCM002774.2_22403960	22403960	A/C	4.0-3.0	E4^{123}, CombENV^{1}	-0.42, -0.27	5.0
49	16	SCM002774.2_23837210	23837210	A/T	3.4-3.1	E4^{13}, CombENV^{13}	-0.36, -0.26	
50	16	SCM002774.2_25678898	25678898	T/C	3.8-3.0	E2^{23}, E4^{123}	0.35, 0.23	4.7-6.0

Table A.3. Marker-trait-associations (MTAs) with stem lesion length (cm) for sclerotinia stem rot resistance in 187 canola/rapeseed genotypes analyzed by the FarmCPU, MLM, and GEMMA-MLM models (continued)

Locus	Chr[a]	SNP ID	Position (bp)	Alleles[b]	-log$_{10}$ (*P*)[c]	Env & Model[d]	Allelic effect[e]	R^{2} [f]
51	16	SCM002774.2_25739024	25739024	T/C	3.7-3.0	E1^{123}, E3^{13}, E4^{1}, CombENV^{123}	0.29, 0.21	5.0-5.4
52	16	SCM002774.2_33238118	33238118	G/T	3.4-3.0	E3^{123}	-0.32	5.9
	16	SCM002774.2_33265642	33265642	G/C	3.4-3.1	E3^{123}	0.31	6.2
53	16	SCM002774.2_33563646	33563646	T/A	4.0-3.0	E1^{123}, E2^{1}, E3^{123}, E4^{1}, CombENV^{123}	0.32, 0.25	5.4-6.0
54	16	SCM002774.2_34137593	34137593	C/T	4.5-3.0	E1^{123}, E2^{123}, E4^{123}, CombENV^{123}	-0.36, -0.25	4.6-6.8
55	16	SCM002774.2_34149554	34149554	T/C	3.8-3.0	E2^{1}, E4^{123}, CombENV^{1}	0.37, 0.24	4.7
56	16	SCM002774.2_39405155	39405155	A/G	4.0-3.4	E3^{123}	-0.25	6.9
57	17	SCM002775.2_43529605	43529605	C/G	4.0-3.1	E4^{123}; CombENV^{12}	0.51; 0.34	5.1-6.3
58	17	SCM002775.2_49740638	49740638	C/A	3.5-3.1	E4^{23}; CombENV^{23}	-0.53; -0.39	5.1-5.2
59	18	SCM002776.2_29886188	29886188	G/C	3.4-3.0	E2^{123}, E4^{23}	-0.45, -0.41	4.8-5.0
60	19	SCM002777.2_15808490	15808490	G/A	3.8-3.5	E3^{123}	0.38	7.2
61	19	SCM002777.2_46851981	46851981	G/A	4.0-3.1	E1^{123}, E3^{123}, E4^{123}, CombENV^{123}	-0.84, -0.59	5.3-6.8
62	19	SCM002777.2_48885679	48885679	C/T	5.8-3.3	E1^{123}, E2^{123}, E3^{123}, E4^{123}, CombENV^{123}	0.83, 0.52	5.8-9.2

Chr[a], *Brassica napus* chromosome

Alleles[b], (_/_) Major allele/minor allele

-log$_{10}$ (*P*)[c], The highest and lowest -log$_{10}$ (*P*) value resulted from the studied environments with different GWAS models where the identified SNPs -log$_{10}$ (*P*) value ≥3.4 by at least of the GWAS models

Env & Model[d], Environments: E1, Carrington 2019; E2, Landon 2019; E3, Carrington 2020; E4, Osnabrock 2020; CombENV, combined analysis across four environments; Model: superscript 1, 2 & 3 represents FarmCPU, MLM and GEMMA-MLM GWAS models respectively

Allelic effect[e], difference in mean stem lesion length (cm) between genotypes with major allele and minor allele. Positive sign indicates major allele is associated with increased lesion length (cm). Negative sign indicates that the major allele is associated with reduced lesion length (cm)

R^{2} [f], Percentage of phenotypic variation explained by the identified significant SNP derived from the results of MLM method

Table A.4. Marker-trait associations (MTAs) with stem lesion width (%) for sclerotinia stem rot resistance in 187 canola/rapeseed genotypes analyzed by the FarmCPU, MLM, and GEMMA-MLM models

Locus	Chr[a]	SNP ID	Position (bp)	Alleles[b]	-log$_{10}$ (*P*)[c]	Env & Model[d]	Allelic effect[e]	R^{2} [f]
1	1	SCM002759.2_1471693	1471693	A/T	6.9-3.3	CombENV^{123}	-1.97; -1.56	5.50
2	1	SCM002759.2_2561773	2561773	C/T	6.6-3.0	E3^{123}, CombENV^{23}	4.42, 1.90	4.9-12.1
3	1	SCM002759.2_4235780	4235780	A/C	3.5-3.2	E2^{123}	2.42, 2.32	5.10
	1	SCM002759.2_4239970	4239970	A/C	4.0-3.3	E2^{123}, E4^{123}, CombENV^{23}	-3.26, -2.26	5.7-6.0
4	1	SCM002759.2_5478454	5478454	C/T	3.4-3.0	E4^{3}, CombENV^{23}	2.78	5.20
5	1	SCM002759.2_12025515	12025515	A/G	3.5-3.3	E1^{123}	2.35; 2.33	6.10
6	1	SCM002759.2_31973754	31973754	A/C	3.5-3.2	E4^{123}	-2.48; -2.50	5.40
7	2	SCM002760.2_1865667	1865667	A/C	4.5-3.6	E4^{123}, CombENV^{23}	-4.27, -5.98	6.2-7.3
8	3	SCM002761.2_6322149	6322149	C/G	3.8-3.3	E2^{123}	-2.70; -2.78	5.70
9	3	SCM002761.2_11084205	11084205	G/A	3.7-3.1	E2^{23}; CombENV^{1}	5.2; 2.4	4.90
10	3	SCM002761.2_11191427	11191427	T/A	3.4-3.3	E2^{13}	-6.47	
11	3	SCM002761.2_28225133	28225133	T/C	3.7-3.4	E3^{23}	-3.34	6.70
12	3	SCM002761.2_28954312	28954312	T/C	3.4-3.2	E4^{123}	4.91; 4.76	5.30
13	3	SCM002761.2_35236913	35236913	C/A	8.7-3.6	E1^{123}, E3^{123}	-2.07, -1.84	6.7-8.2
14	3	SCM002761.2_36170515	36170515	G/A	7.0-3.3	E1^{123}, E3^{23}, CombENV^{123}	2.24, 1.52	5.6-8.7
15	5	SCM002763.2_1779846	1779846	G/A	4.4-3.1	E1^{3}; E3^{23}; CombENV^{23}	-2.47; -2.15	5.2-7.1
16	5	SCM002763.2_15914063	15914063	G/A	5.1-3.3	E2^{123}, E4^{123}, CombENV^{23}	-5.49, -3.18	5.5-7.8
	5	SCM002763.2_15924240	15924240	T/C	3.5-3.2	E2^{13}	2.73	
17	5	SCM002763.2_16279322	16279322	A/C	5.8-3.0	E1^{13}, E3^{123}, CombENV^{123}	-3.89, -2.66	5.4-8.0
18	5	SCM002763.2_24009625	24009625	C/G	3.5-3.2	E1^{123}	2.69; 2.58	6.00
19	5	SCM002763.2_28608175	28608175	A/G	3.5-3.0	E2^{123}, CombENV^{3}	4.16	5.20
20	6	SCM002764.2_25241979	25241979	C/A	4.5-3.2	E1^{123}, E3^{123}, CombENV^{123}	-5.10, -3.15	5.2-8.0
21	6	SCM002764.2_29348402	29348402	A/C	5.3-3.1	E2^{1}, E4^{13}, CombENV^{123}	-2.60, -1.43	5.50
22	6	SCM002764.2_33502340	33502340	C/A	3.6-3.2	E1^{13}; E3^{3}	-3.20	
23	6	SCM002764.2_33622715	33622715	T/C	3.4-3.0	E2^{3}; E4^{3}; CombENV^{23}	1.77	5.30
24	8	SCM002766.2_13657808	13657808	T/G	3.6-3.1	E3^{23}	-3.76	5.90

Table A.4. Marker-trait associations (MTAs) with stem lesion width (%) for sclerotinia stem rot resistance in 187 canola/rapeseed genotypes analyzed by the FarmCPU, MLM, and GEMMA-MLM models (continued)

Locus	Chr[a]	SNP ID	Position (bp)	Alleles[b]	-log$_{10}$ (*P*)[c]	Env & Model[d]	Allelic effect[e]	R^{2} [f]
25	8	SCM002766.2_20621551	20621551	A/T	3.6-3.4	E2^{23}	2.64	5.40
26	8	SCM002766.2_20760271	20760271	G/C	3.9-3.2	E3^{23}; CombENV^{23}	2.35; 2.06	5.4-7.2
27	9	SCM002767.2_37671479	37671479	G/A	4.0-3.2	E1^{123}, CombENV^{3}	-2.60; -2.64	6.90
28	9	SCM002767.2_45297735	45297735	T/C	3.4-3.1	CombENV^{23}	-2.95	5.10
29	9	SCM002767.2_45841268	45841268	A/T	5.9-3.8	E3^{123}	4.58; 3.56	7.60
30	10	SCM002768.2_2190513	2190513	A/C	3.5-3.1	E1^{123}, E3^{23}	3.57; 3.41	6.00
31	10	SCM002768.2_12144744	12144744	T/C	3.4-3.2	E3^{23}	2.62	6.30
32	10	SCM002768.2_21099405	21099405	G/C	3.5-3.0	E2^{13}; E4^{13}	2.89; 2.47	
33	11	SCM002769.2_6223969	6223969	T/C	3.4-3.1	E1^{123}; CombENV^{1}	-3.43	5.70
34	11	SCM002769.2_46192098	46192098	T/C	3.9-3.0	E2^{123}, CombENV^{23}	-7.37, -4.75	5.0-5.8
35	12	SCM002770.2_25096010	25096010	A/T	3.8-3.1	E1^{123}, E3^{23}; CombENV^{3}	4.70, 4.50	5.9-6.2
36	12	SCM002770.2_32020747	32020747	A/G	4.3-3.0	E1^{123}, E2^{1}, E4^{123}, CombENV^{123}	4.04, 1.80	4.9-6.2
37	12	SCM002770.2_43963414	43963414	A/T	3.6-3.0	E2^{13}, E4^{123}	-3.02; -2.71	5.10
	12	SCM002770.2_44006308	44006308	G/A	3.5-3.1	E2^{13}; E4^{13}	2.88; 2.51	
	12	SCM002770.2_44012322	44012322	C/T	3.6-3.0	E4^{123}, CombENV^{1}	-2.78, -2.68	5.50
38	12	SCM002770.2_59170961	59170961	A/C	3.7-3.4	E3^{23}	-5.15	6.80
39	12	SCM002770.2_62791159	62791159	T/C	3.7-3.4	E3^{23}	-3.20	6.70
40	13	SCM002771.2_7893201	7893201	C/G	5.0-3.5	E2^{123}, E4^{123}, CombENV^{23}	7.51, 4.54	6.0-7.1
41	13	SCM002771.2_22853068	22853068	G/A	3.4-3.0	E4^{13}, CombENV^{23}	-4.24, -3.34	4.90
42	13	SCM002771.2_27877818	27877818	G/C	3.4-3.2	E4^{123}	-5.67; -5.77	5.40
43	13	SCM002771.2_45250459	45250459	T/G	3.4-3.2	E1^{123}	2.22; 2.20	5.90
44	13	SCM002771.2_48097242	48097242	G/A	3.9-3.1	E1^{123}, CombENV^{23}	-4.19, -3.81	5.2-6.9
45	13	SCM002771.2_56900783	56900783	G/A	3.5-3.1	E3^{23}	3.52	6.50
46	14	SCM002772.2_8693165	8693165	A/G	3.4-3.2	E1^{123}, E3^{23}	5.90, 5.49	5.8-6.2
47	14	SCM002772.2_10285794	10285794	T/G	3.5-3.3	E4^{123}	4.12, 4.05	5.60

Table A.4. Marker-trait associations (MTAs) with stem lesion width (%) for sclerotinia stem rot resistance in 187 canola/rapeseed genotypes analyzed by the FarmCPU, MLM, and GEMMA-MLM models (continued)

Locus	Chr[a]	SNP ID	Position (bp)	Alleles[b]	-log$_{10}$ (*P*)[c]	Env & Model[d]	Allelic effect[e]	R^2 [f]
48	14	SCM002772.2_12898850	12898850	G/A	3.9-3.6	$E1^{123}$	-4.07, -4.02	6.80
49	14	SCM002772.2_13679559	13679559	C/T	3.9-3.6	$E3^{23}$	3.90	7.20
50	14	SCM002772.2_65359864	65359864	C/T	3.8-3.2	$E1^{123}$, $E3^{123}$, $CombENV^{23}$	6.07, 3.28	5.5-6.8
51	14	SCM002772.2_70479506	70479506	A/G	4.0-3.0	$E2^{3}$, $E4^{123}$	-6.83, -6.75	6.40
52	15	SCM002773.2_9748366	9748366	T/C	3.6-3.2	$E4^{123}$, $CombENV^{23}$	4.15, 3.08	5.2-5.7
53	15	SCM002773.2_10201221	10201221	C/T	7.2-3.9	$E3^{123}$	7.75, 6.43	7.80
54	15	SCM002773.2_35983051	35983051	G/A	3.9-3.1	$E1^{23}$	2.69; 1.52	5.60
55	16	SCM002774.2_25673001	25673001	G/A	3.4-3.1	$E2^{123}$, $E4^{123}$	3.33, 3.05	5.0-5.2
56	16	SCM002774.2_25678898	25678898	T/C	5.0-3.0	$E2^{123}$, $E4^{123}$, $CombENV^{23}$	3.81, 2.15	4.9-7.6
57	16	SCM002774.2_25739024	25739024	T/C	10.9-3.4	$E3^{23}$, $E4^{123}$, $CombENV^{123}$	2.78, 2.17	6.1-6.9
58	16	SCM002774.2_33238118	33238118	G/T	3.4-3.0	$E3^{23}$	-2.91	5.80
59	16	SCM002774.2_33563646	33563646	T/A	3.4-3.0	$E1^{123}$; $CombENV^{23}$	2.16; 2.13	5.0-5.5
60	16	SCM002774.2_39405155	39405155	A/G	3.7-3.2	$E3^{23}$	-2.23	6.30
61	17	SCM002775.2_41131780	41131780	A/G	3.5-3.2	$E2^{23}$	-2.58	5.00
62	17	SCM002775.2_49740638	49740638	C/A	3.6-3.0	$E4^{123}$, $CombENV^{3}$	-5.08, -4.98	5.70
63	17	SCM002775.2_56829232	56829232	C/G	3.7-3.0	$E1^{3}$, $E4^{3}$, $CombENV^{23}$	-2.84	5.80
64	18	SCM002776.2_37107013	37107013	C/T	3.8-3.5	$CombENV^{23}$	3.33	5.90
65	19	SCM002777.2_28925630	28925630	C/A	3.4-3.1	$E1^{123}$	4.64	5.70
66	19	SCM002777.2_48885679	48885679	C/T	8.2-3.8	$E1^{123}$, $E2^{123}$, $E3^{123}$, $E4^{123}$, $CombENV^{123}$	7.60, 3.37	7.0-10.3

Chr[a], *Brassica napus* chromosome

Alleles[b] (_/_), Major allele/minor allele

-log10 (*P*)[c], The highest and lowest -log10 (*P*) value resulted from the studied environments with different GWAS models where the identified SNPs -log10 (*P*) value ≥3.4 by at least of the GWAS models

Env & Model[d], Environments: E1, Carrington 2019; E2, Landon 2019; E3, Carrington 2020; E4, Osnabrock 2020; CombENV, combined analysis across four environments; Model: superscript 1, 2 & 3 represents FarmCPU, MLM and GEMMA-MLM GWAS models respectively

Allelic effect[e], difference in mean stem lesion width (%) between genotypes with major allele and minor allele. Positive sign indicates major allele is associated with increased lesion width (%). Negative sign indicates that the major allele is associated with reduced lesion width (%)

R^2 [f], Percentage of phenotypic variation explained by the identified significant SNP derived from the results of MLM method

Table A.5. Marker-trait-associations (MTAs) with combENV plant mortality (14D, 21D) for sclerotinia stem rot resistance in 187 canola/rapeseed genotypes analyzed by the FarmCPU, MLM, and GEMMA-MLM models

Locus	Chr[a]	SNP ID	Position (bp)	Alleles[b]	-log$_{10}$ (*P*)[c]	Model & Model[d]	Allelic effect[e]	R^{2} [f]
1	1	SCM002759.2_1471693	1471693	A/T	4.0-3.2	PM_14D^{123}; PM_21D^{23}	-3.51; -3.34	3.9-5.3
2	1	SCM002759.2_5322870	5322870	T/A	3.5-3.3	PM_21D^{23}	-6.28	3.9
	1	SCM002759.2_5323805	5323805	G/A	3.4-3.2	PM_21D^{23}	-6.12	3.8
3	3	SCM002761.2_660936	660936	T/A	5.7-3.5	PM_21D^{123}	6.49, 4.85	4.3
4	3	SCM002761.2_8296124	8296124	G/A	3.4-3.2	PM_14D^{123}	5.41	4.8
5	3	SCM002761.2_31300829	31300829	G/A	3.4-3.2	PM_21D^{23}	7.94	3.8
6	3	SCM002761.2_31986941	31986941	G/A	3.9-3.0	PM_14D^{123}, PM_21D^{23}	4.43, 4.15	3.6-5.5
7	4	SCM002762.2_16168004	16168004	G/C	3.5-3.1	PM_14D^{13}, PM_21D^{23}	4.07, 3.79	4.0
8	4	SCM002762.2_20789292	20789292	G/A	3.7-3.1	PM_21D^{23}	-3.32	3.7
9	4	SCM002762.2_20860192	20860192	A/T	4.0-3.7	PM_21D^{23}	4.11	4.6
10	6	SCM002764.2_8266872	8266872	G/C	3.5-3.0	PM_21D^{23}	5.36	3.6
11	6	SCM002764.2_24985572	24985572	G/A	3.4-3.2	PM_21D^{23}	3.20	3.8
12	8	SCM002766.2_17407234	17407234	G/T	3.5-3.3	PM_21D^{23}	-10.66	4.0
13	8	SCM002766.2_20760271	20760271	G/C	4.3-3.6	PM_14D^{123}, PM_21D^{23}	4.01, 3.82	5.0-5.4
14	8	SCM002766.2_22920266	22920266	T/C	3.8-3.3	PM_14D^{3}, PM_21D^{23}	7.24	4.4
15	9	SCM002767.2_35588232	35588232	T/C	6.8-3.9	PM_21D^{123}	-7.47, -5.64	4.8
16	9	SCM002767.2_36527400	36527400	T/C	3.8-3.1	PM_14D^{123}, PM_21D^{3}	-5.83	5.4
17	9	SCM002767.2_43331392	43331392	C/A	3.4-3.0	PM_14D^{123}, PM_21D^{23}	-3.43, -3.30	3.6-4.6
	9	SCM002767.2_43506803	43506803	G/T	3.4-3.1	PM_21D^{23}	3.26	3.7
18	10	SCM002768.2_4273597	4273597	G/A	4.0-3.1	PM_14D^{13}, PM_21D^{23}	-11.29, -9.48	4.5
19	10	SCM002768.2_5144576	5144576	T/C	7.4-3.2	PM_14D^{123}, PM_21D^{123}	5.74, 4.04	4.7-7.7
20	11	SCM002769.2_6223969	6223969	T/C	3.5-3.3	PM_14D^{123}	-6.05	5.0
21	11	SCM002769.2_46192098	46192098	T/C	4.1-3.0	PM_14D^{123}, PM_21D^{123}	-9.69, -5.60	4.4-4.8
22	13	SCM002771.2_7740873	7740873	C/A	3.5-3.0	PM_14D^{13}, PM_21D^{23}	4.19, 3.97	3.7
23	14	SCM002772.2_3422830	3422830	G/A	3.5-3.3	PM_14D^{123}	7.66	4.9
24	14	SCM002772.2_29581828	29581828	C/A	4.6-3.1	PM_14D^{123}, PM_21D^{123}	-9.74, -5.74	3.7-6.6
25	14	SCM002772.2_53077839	53077839	A/G	4.2-3.9	PM_14D^{123}	4.22	6.0
26	15	SCM002773.2_9748366	9748366	T/C	4.1-3.5	PM_21D^{123}	5.77, 3.23	4.3
27	15	SCM002773.2_36238886	36238886	G/T	3.6-3.4	PM_21D^{23}	-3.94	4.2
28	15	SCM002773.2_36273958	36273958	A/G	3.7-3.4	PM_21D^{23}	-3.89	4.2

Table A.5. Marker-trait-associations (MTAs) with combENV plant mortality (14D, 21D) for sclerotinia stem rot resistance in 187 canola/rapeseed genotypes analyzed by the FarmCPU, MLM, and GEMMA-MLM models (continued)

Locus	Chr[a]	SNP ID	Position (bp)	Alleles[b]	-log$_{10}$ (*P*)[c]	Env & Model[d]	Allelic effect[e]	R^{2} [f]
29	16	SCM002774.2_14148427	14148427	G/A	3.6-3.4	PM_21D^{23}	6.91	4.10
30	16	SCM002774.2_23440605	23440605	G/T	4.0-3.6	PM_21D^{23}	-8.68	4.5
31	16	SCM002774.2_25739024	25739024	T/C	4.0-3.8	PM_14D^{123}	3.64	5.7
32	16	SCM002774.2_33563646	33563646	T/A	4.0-3.7	PM_14D^{123}	4.06	5.7
33	18	SCM002776.2_7320194	7320194	G/T	4.0-3.7	PM_14D^{123}	6.27	5.7
34	18	SCM002776.2_12692146	12692146	T/G	3.6-3.3	PM_21D^{23}	-4.71	4.0
35	18	SCM002776.2_41681055	41681055	C/T	3.5-3.3	PM_14D^{123}	7.14	5.0
36	19	SCM002777.2_34413846	34413846	C/T	7.0-3.2	PM_21D^{123}	-10.25, -9.21	3.8
37	19	SCM002777.2_48885679	48885679	C/T	4.6-4.3	PM_14D^{123}	9.37	6.7

Chr[a], *Brassica napus* chromosome

Alleles[b], (/) Major allele/minor allele

-log10 (*P*)[c], The highest and lowest -log10 (P) value resulted from the studied environments with different GWAS models where the identified SNPs -log10 (P) value ≥3.4 by at least of the GWAS models

Trait & Model[d], Trait: PM_14D, combENV average plant mortality at 14 days post inoculation across four environments; PM_21D, combENV average plant mortality at 21 days post inoculation; Model: superscript 1, 2 & 3 represents FarmCPU, MLM and GEMMA-MLM GWAS models respectively

Allelic effect[e], difference in mean stem plant mortality (%) between genotypes with major allele and minor allele. Positive sign indicates major allele is associated with increased plant mortality (%). Negative sign indicates that the major allele is associated with reduced plant mortality (%)

R^{2} [f], Percentage of phenotypic variation explained by the identified significant SNP derived from the results of MLM method

Table A.6. List of significant single-nucleotide polymorphisms (SNPs) associated with stem lesion length (LL), stem lesion width (LW), and combENV plant mortality (PM_14D, PM_21D) traits for sclerotinia stem rot resistance identified by the FarmCPU, MLM, and GEMMA-MLM models

Locus	Chr[a]	SNP ID	Position (bp)	-log$_{10}$ (*P*)[c]	Traits, Env & Model[d]	Allelic effect[e]	R^2 [f]	Previously reported MTAs[g]
1	1	SCM002759.2_1471693	1471693	4.3-3.1	LL_E1^{123}, LL_E2^{23}, LL_E4^{23}, LL_CombENV^{123}	-0.30, -0.22	4.8-7.0	
	1	SCM002759.2_1471693	1471693	6.9-3.3	LW_CombENV^{123}	-1.97; -1.56	5.5	
	1	SCM002759.2_1471693	1471693	4.0-3.2	PM_14D^{123}; PM_21D^{23}	-3.51; -3.34	3.9-5.3	
2	1	SCM002759.2_1766199	1766199	3.6-3.0	LL_E2^{123}, LL_CombENV^{123}	0.47, 0.37	4.6-5.6	
3	1	SCM002759.2_2561773	2561773	6.6-3.0	LW_E3^{123}, LW_CombENV^{23}	4.42, 1.90	4.9-12.1	
4	1	SCM002759.2_4235780	4235780	3.5-3.2	LW_E2^{123}	2.42, 2.32	5.1	
	1	SCM002759.2_4239970	4239970	4.8-3.3	LL_E2^{123}, LL_E4^{23}, LL_CombENV^{23}	-0.34; -0.25	5.4-7.3	
	1	SCM002759.2_4239970	4239970	4.0-3.3	LW_E2^{123}, LW_E4^{123}, LW_CombENV^{23}	-3.26, -2.26	5.7-6.0	
5	1	SCM002759.2_5322870	5322870	3.5-3.3	PM_21D^{23}	-6.28	3.9	
	1	SCM002759.2_5323805	5323805	3.4-3.2	PM_21D^{23}	-6.12	3.8	
6	1	SCM002759.2_5478454	5478454	3.4-3.0	LW_E4^{3}, LW_CombENV^{23}	2.78	5.2	
	1	SCM002759.2_5478454	5478454	3.5-3.3	LL_E2^{23}	0.38	5.1	
7	1	SCM002759.2_11482241	11482241	3.1-3.4	LL_E1^{123}, LL_E3^{3}, LL_CombENV^{13}	-0.28; -0.27	5.4	Wu *et al*. 2013 (12.44-19.86 Mb)
8	1	SCM002759.2_12025515	12025515	3.5-3.3	LW_E1^{123}	2.35; 2.33	6.1	Wu et al. 2013 (12.44-19.86 Mb)
9	1	SCM002759.2_31973754	31973754	3.5-3.2	LW_E4^{123}	-2.48; -2.50	5.4	
10	2	SCM002760.2_1865667	1865667	4.8-3.0	LL_E2^{23}, LL_E4^{123}, LL_CombENV^{23}	-0.70, -0.43	4.5-7.7	
	2	SCM002760.2_1865667	1865667	4.5-3.6	LW_E4^{123}, LW_CombENV^{23}	-4.27, -5.98	6.2-7.3	

Table A.6. List of significant single-nucleotide polymorphisms (SNPs) associated with stem lesion length (LL), stem lesion width (LW), and combENV plant mortality (PM_14D, PM_21D) traits for sclerotinia stem rot resistance identified by the FarmCPU, MLM, and GEMMA-MLM models (continued)

Locus	Chr[a]	SNP ID	Position (bp)	-log$_{10}$ (*P*)[c]	Traits, Env & Model[d]	Allelic effect[e]	R^2 [f]	Previously reported MTAs[g]
11	2	SCM002760.2_10378033	10378033	3.5-3.3	LL_E4^{23}	-0.28	5.3	Shahoveisi *et al*. 2021 (7.32-10.93 Mb); (7.59-10.93 Mb)
12	2	SCM002760.2_30827371	30827371	3.6-3.1	LL_E3^{123}	-0.22; -0.21	6.2	
13	3	SCM002761.2_660936	660936	5.7-3.5	PM_21D^{123}	6.49, 4.85	4.3	
14	3	SCM002761.2_6322149	6322149	3.8-3.3	LW_E2^{123}	-2.70; -2.78	5.7	
15	3	SCM002761.2_8296124	8296124	3.4-3.2	PM_14D^{123}	5.41	4.8	
16	3	SCM002761.2_11084205	11084205	3.7-3.1	LW_E2^{23}; CombENV^{1}	5.2; 2.4	4.9	
17	3	SCM002761.2_11191427	11191427	3.4-3.3	LW_E2^{13}	-6.47		
18	3	SCM002761.2_17320071	17320071	3.8-3.1	LL_E2^{123}	-0.56; -0.51	4.8	
19	3	SCM002761.2_28225133	28225133	3.7-3.4	LL_E3^{123}	-0.36	7.2	
	3	SCM002761.2_28225133	28225133	3.7-3.4	LW_E3^{23}	-3.34	6.7	
20	3	SCM002761.2_28954312	28954312	3.4-3.2	LW_E4^{123}	4.91; 4.76	5.3	
21	3	SCM002761.2_30742237	30742237	3.4-3.0	LL_E1^{123}, LL_CombENV^{3}	0.41	5.4	
22	3	SCM002761.2_31300829	31300829	3.4-3.2	PM_21D^{23}	7.94	3.8	
23	3	SCM002761.2_31986941	31986941	3.9-3.0	PM_14D^{123}, PM_21D^{23}	4.43, 4.15	3.6-5.5	
24	3	SCM002761.2_35236913	35236913	3.5-3.2	LL_E3^{123}	-0.20	6.7	
	3	SCM002761.2_35236913	35236913	8.7-3.6	LW_E1^{123}, LW_E3^{123}	-2.07, -1.84	6.7-8.2	
25	3	SCM002761.2_36170515	36170515	5.4-4.0	LL_E1^{123}, LL_E3^{123}, LL_CombENV^{123}	0.26, 0.23	7.0-9.9	
	3	SCM002761.2_36170515	36170515	7.0-3.3	LW_E1^{123}, LW_E3^{23}, LW_CombENV^{123}	2.24, 1.52	5.6-8.7	

Table A.6. List of significant single-nucleotide polymorphisms (SNPs) associated with stem lesion length (LL), stem lesion width (LW), and combENV plant mortality (PM_14D, PM_21D) traits for sclerotinia stem rot resistance identified by the FarmCPU, MLM, and GEMMA-MLM models (continued)

Locus	Chr[a]	SNP ID	Position (bp)	-log$_{10}$ (P)[c]	Traits, Env & Model[d]	Allelic effect[e]	R^2 [f]	Previously reported MTAs[g]
26	4	SCM002762.2_3551539	3551539	3.4-3.0	LL_E4^{123}, LL_CombENV^{123}	0.61, 0.45	4.8-5.2	
27	4	SCM002762.2_16168004	16168004	3.5-3.1	PM_14D^{13}, PM_21D^{23}	4.07, 3.79	4.0	
28	4	SCM002762.2_20212672	20212672	4.5-3.0	LL_E3^{123}, LL_CombENV^{13}	-0.56; -0.44	5.0-8.6	
29	4	SCM002762.2_20789292	20789292	3.7-3.1	PM_21D^{23}	-3.32	3.7	
	4	SCM002762.2_20860192	20860192	4.0-3.7	PM_21D^{23}	4.11	4.6	
30	5	SCM002763.2_1779846	1779846	4.4-3.1	LW_E1^{3}; LW_E3^{23}; LW_CombENV^{23}	-2.47; -2.15	5.2-7.1	
31	5	SCM002763.2_15914063	15914063	5.2-3.0	LL_E2^{123}, LL_E4^{123}, LL_CombENV^{123}	-0.59; -0.33	5.0-8.0	
	5	SCM002763.2_15914063	15914063	5.1-3.3	LW_E2^{123}, LW_E4^{123}, LW_CombENV^{23}	-5.49, -3.18	5.5-7.8	
	5	SCM002763.2_15924240	15924240	3.5-3.2	LW_E2^{13}	2.73		
32	5	SCM002763.2_16279322	16279322	3.7-3.1	LL_E3^{123}, LL_CombENV^{23}	-0.39; -0.34	5.1-7.2	
	5	SCM002763.2_16279322	16279322	5.8-3.0	LW_E1^{13}, LW_E3^{123}, LW_CombENV^{123}	-3.89, -2.66	5.4-8.0	
33	5	SCM002763.2_24009625	24009625	3.5-3.2	LW_E1^{123}	2.69; 2.58	6.0	
34	5	SCM002763.2_28142332	28142332	3.9-3.1	LL_E2^{123}	0.24; 0.27	4.7	
35	5	SCM002763.2_28608175	28608175	3.5-3.3	LL_E2^{123}	0.42; 0.38	5.1	
	5	SCM002763.2_28608175	28608175	3.5-3.0	LW_E2^{123}, LW_CombENV^{3}	4.16	5.2	
36	6	SCM002764.2_8266872	8266872	3.5-3.0	PM_21D^{23}	5.36	3.6	
37	6	SCM002764.2_24985572	24985572	3.4-3.2	PM_21D^{23}	3.20	3.8	
38	6	SCM002764.2_25241979	25241979	3.3-3.0	LL_E1^{13}, LL_E3^{23}, LL_CombENV^{123}	-0.47; -0.45	5.0-5.9	Wu *et al.* 2019 (21.63-23.50 Mb)
	6	SCM002764.2_25241979	25241979	4.5-3.2	LW_E1^{123}, LW_E3^{123}, LW_CombENV^{123}	-5.10, -3.15	5.2-8.0	

Table A.6. List of significant single-nucleotide polymorphisms (SNPs) associated with stem lesion length (LL), stem lesion width (LW), and combENV plant mortality (PM_14D, PM_21D) traits for sclerotinia stem rot resistance identified by the FarmCPU, MLM, and GEMMA-MLM models (continued)

Locus	Chr[a]	SNP ID	Position (bp)	-log$_{10}$ (P)[c]	Traits, Env & Model[d]	Allelic effect[e]	R^2 [f]	Previously reported MTAs[g]
39	6	SCM002764.2_29348402	29348402	5.3-3.1	LW_E2[1], LW_E4[13], LW_CombENV[123]	-2.60, -1.43	5.5	
40	6	SCM002764.2_33502340	33502340	3.6-3.2	LW_E1[13]; LW_E3[3]	-3.20		
41	6	SCM002764.2_33622715	33622715	3.4-3.0	LW_E2[3]; LW_E4[3]; LW_CombENV[23]	1.77	5.3	
42	7	SCM002765.2_25991221	25991221	3.7-3.1	LL_E1[123], LL_E2[123], LL_CombENV[3]	-0.63; -0.51	5.4-5.5	
43	8	SCM002766.2_1982656	1982656	4.6-3.1	LL_E3[123], LL_E4[123], LL_CombENV[123]	-0.57; -0.38	7.0-7.5	
44	8	SCM002766.2_13657808	13657808	3.5-3.0	LL_E3[123]	-0.41	6.0	
	8	SCM002766.2_13657808	13657808	3.6-3.1	LW_E3[23]	-3.76	5.9	Wei *et al*. 2016 (15.01-15.43 Mb)
45	8	SCM002766.2_13864870	13864870	3.7-3.1	LL_E2[123], LL_E4[123], LL_CombENV[123]	0.67, 0.47	5.2-5.7	
46	8	SCM002766.2_17407234	17407234	3.5-3.3	PM_21D[23]	-10.66	4.0	
47	8	SCM002766.2_20621551	20621551	3.6-3.4	LW_E2[23]	2.64	5.4	
48	8	SCM002766.2_20760271	20760271	3.9-3.2	LW_E3[23]; LW_CombENV[23]	2.35; 2.06	5.4-7.2	
49	8	SCM002766.2_20760271	20760271	4.3-3.6	PM_14D[123], PM_21D[23]	4.01, 3.82	5.0-5.4	
50	8	SCM002766.2_22920266	22920266	3.8-3.3	PM_14D[3], PM_21D[23]	7.24	4.4	
51	9	SCM002767.2_35588232	35588232	6.8-3.9	PM_21D[123]	-7.47, -5.64	4.8	
52	9	SCM002767.2_36527400	36527400	3.8-3.1	PM_14D[123], PM_21D[3]	-5.83	5.4	
53	9	SCM002767.2_37664281	37664281	3.9-3.7	LL_E4[123]	-0.34	6.2	
54	9	SCM002767.2_37671479	37671479	3.6-3.1	LL_E1[123], LL_E2[1]	-0.31; -0.27	5.9	
	9	SCM002767.2_37671479	37671479	4.0-3.2	LW_E1[123], LW_CombENV[3]	-2.60; -2.64	6.9	

Table A.6. List of significant single-nucleotide polymorphisms (SNPs) associated with stem lesion length (LL), stem lesion width (LW), and combENV plant mortality (PM_14D, PM_21D) traits for sclerotinia stem rot resistance identified by the FarmCPU, MLM, and GEMMA-MLM models (continued)

Locus	Chr[a]	SNP ID	Position (bp)	-log$_{10}$ (*P*)[c]	Traits, Env & Model[d]	Allelic effect[e]	R^2 [f]	Previously reported MTAs[g]
55	9	SCM002767.2_39128782	39128782	3.6-3.2	LL_E1[123], LL_CombENV[123]	-0.60; -0.56	5.4-6.0	
56	9	SCM002767.2_43041855	43041855	3.8-3.5	LL_E3[123]	0.65	7.1	
57	9	SCM002767.2_43331392	43331392	3.4-3.0	PM_14D[123], PM_21D[23]	-3.43, -3.30	3.6-4.6	
	9	SCM002767.2_43506803	43506803	3.4-3.1	PM_21D[23]	3.26	3.7	
58	9	SCM002767.2_45297735	45297735	4.0-3.3	LL_E2[123], LL_E4[13], LL_CombENV[2]	-0.47; -0.33	5.1-6.1	
	9	SCM002767.2_45297735	45297735	3.4-3.1	LW_CombENV[23]	-2.95	5.1	
59	9	SCM002767.2_45841268	45841268	3.5-3.0	LL_E3[123], LL_CombENV[13]	0.44, 0.43	6.0	
	9	SCM002767.2_45841268	45841268	5.9-3.8	LW_E3[123]	4.58; 3.56	7.6	
60	10	SCM002768.2_2190513	2190513	3.5-3.1	LW_E1[123], LW_E3[23]	3.57; 3.41	6.0	
61	10	SCM002768.2_4273597	4273597	4.0-3.1	PM_14D[13], PM_21D[23]	-11.29, -9.48	4.5	
62	10	SCM002768.2_5144576	5144576	7.4-3.2	PM_14D[123], PM_21D[123]	5.74, 4.04	4.7-7.7	
63	10	SCM002768.2_12144744	12144744	3.4-3.2	LW_E3[23]	2.62	6.3	
64	10	SCM002768.2_21099405	21099405	3.5-3.0	LW_E2[13]; LW_E4[13]	2.89; 2.47		
65	11	SCM002769.2_6223969	6223969	4.4-3.3	LL_E1[123], LL_E3[123], LL_CombENV[123]	-0.45; -0.38	6.0-7.3	
	11	SCM002769.2_6223969	6223969	3.4-3.1	LW_E1[123], LW_CombENV[1]	-3.43	5.7	
	11	SCM002769.2_6223969	6223969	3.5-3.3	PM_14D[123]	-6.05	5.0	
66	11	SCM002769.2_46192098	46192098	4.2-3.0	LL_E2[123], LL_E4[23], LL_CombENV[23]	-0.74; -0.52	4.9-6.4	
	11	SCM002769.2_46192098	46192098	3.9-3.0	LW_E2[123], LW_CombENV[23]	-7.37, -4.75	5.0-5.8	
	11	SCM002769.2_46192098	46192098	4.1-3.0	PM_14D[123], PM_21D[123]	-9.69, -5.60	4.4-4.8	

Table A.6. List of significant single-nucleotide polymorphisms (SNPs) associated with stem lesion length (LL), stem lesion width (LW), and combENV plant mortality (PM_14D, PM_21D) traits for sclerotinia stem rot resistance identified by the FarmCPU, MLM, and GEMMA-MLM models (continued)

Locus	Chr[a]	SNP ID	Position (bp)	-log$_{10}$ (*P*)[c]	Traits, Env & Model[d]	Allelic effect[e]	R^2 [f]	Previously reported MTAs[g]
67	12	SCM002770.2_25096010	25096010	3.6-3.0	LL_E3^{123}, LL_CombENV^{1}	0.50, 0.49	6.1	
	12	SCM002770.2_25096010	25096010	3.8-3.1	LW_E1^{123}, LW_E3^{23}; LW_CombENV^{3}	4.70, 4.50	5.9-6.2	
68	12	SCM002770.2_32020747	32020747	4.0-3.0	LL_E2^{123}, LL_E4^{13}, LL_CombENV^{123}	0.46, 0.34	5.1-6.0	
	12	SCM002770.2_32020747	32020747	4.3-3.0	LW_E1^{123}, LW_E2^{1}, LW_E4^{123}, LW_CombENV^{123}	4.04, 1.80	4.9-6.2	
69	12	SCM002770.2_43963414	43963414	3.6-3.0	LW_E2^{13}, LW_E4^{123}	-3.02; -2.71	5.1	
	12	SCM002770.2_44006308	44006308	3.5-3.1	LW_E2^{13}; LW_E4^{13}	2.88; 2.51		
	12	SCM002770.2_44012322	44012322	3.6-3.0	LW_E4^{123}, LW_CombENV^{1}	-2.78, -2.68	5.5	
70	12	SCM002770.2_59170961	59170961	3.7-3.4	LW_E3^{23}	-5.15	6.8	
71	12	SCM002770.2_62791159	62791159	3.6-3.3	LL_E3^{123}	-0.35; -0.34	7.0	
	12	SCM002770.2_62791159	62791159	3.7-3.4	LW_E3^{23}	-3.20	6.7	
72	13	SCM002771.2_7740873	7740873	3.5-3.0	PM_14D^{13}, PM_21D^{23}	4.19, 3.97	3.7	
	13	SCM002771.2_7893201	7893201	5.0-3.1	LL_E2^{123}, LL_E4^{123}, LL_CombENV^{13}	0.78, 0.47	5.7-7.9	
	13	SCM002771.2_7893201	7893201	5.0-3.5	LW_E2^{123}, LW_E4^{123}, LW_CombENV^{23}	7.51, 4.54	6.0-7.1	
73	13	SCM002771.2_22853068	22853068	3.7-3.0	LL_E2^{123}, LL_E4^{123}, LL_CombENV^{123}	-0.50; -0.38	4.9-5.3	Qasim *et al*. 2020 (22.21-30.60 Mb)
	13	SCM002771.2_22853068	22853068	3.4-3.0	LW_E4^{13}, LW_CombENV^{23}	-4.24, -3.34	4.9	

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Locus	Chr[a]	SNP ID	Position (bp)	-log$_{10}$ (*P*)[c]	Traits, Env & Model[d]	Allelic effect[e]	R^2 [f]	Previously reported MTAs[g]
74	13	SCM002771.2_27877818	27877818	3.4-3.2	LW_E4^{123}	-5.67; -5.77	5.4	Qasim *et al*. 2020 (22.21-30.60 Mb); Shahoveisi *et al*. 2021 (23.5-31.7 Mb)
75	13	SCM002771.2_45250459	45250459	3.8-3.5	LL_E1^{123}	0.26	6.2	Qasim *et al*. 2020 (30.60-47.86 Mb) and (47.86-50.63 Mb)
	13	SCM002771.2_45250459	45250459	3.4-3.2	LW_E1^{123}	2.22; 2.20	5.9	
76	13	SCM002771.2_48097242	48097242	3.9-3.1	LW_E1^{123}, LW_CombENV^{23}	-4.19, -3.81	5.2-6.9	
77	13	SCM002771.2_56900783	56900783	3.8-3.0	LL_E1^{123}, LL_E3^{123}, LL_CombENV^{13}	0.40, 0.34	4.9-7.4	
	13	SCM002771.2_56900783	56900783	3.5-3.1	LW_E3^{23}	3.52	6.5	
78	13	SCM002771.2_69833085	69833085	3.7-3.2	LL_E2^{23}	0.40	5.6	
	13	SCM002771.2_69833922	69833922	3.4-3.2	LL_E2^{23}	0.37	5.0	
79	13	SCM002771.2_78761785	78761785	3.5-3.0	LL_E4^{3}, LL_CombENV^{123}	0.68, 0.63	5.3	
80	13	SCM002771.2_80035604	80035604	3.4-3.0	LL_E1^{3}; E3^{3}; CombENV^{23}	0.64	5.3	
81	14	SCM002772.2_3422830	3422830	3.5-3.3	PM_14D^{123}	7.66	4.9	Wu *et al*. 2013 (3.06-7.93 Mb)
82	14	SCM002772.2_8693165	8693165	3.4-3.2	LW_E1^{123}, E3^{23}	5.90, 5.49	5.8-6.2	
83	14	SCM002772.2_10285794	10285794	3.5-3.3	LW_E4^{123}	4.12, 4.05	5.6	Zhao *et al*. 2006 (11.7-28.7 Mb)
84	14	SCM002772.2_12898850	12898850	3.9-3.6	LW_E1^{123}	-4.07, -4.02	6.8	
85	14	SCM002772.2_13679559	13679559	3.9-3.6	LW_E3^{23}	3.90	7.2	

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Locus	Chr[a]	SNP ID	Position (bp)	-log$_{10}$ (*P*)[c]	Traits, Env & Model[d]	Allelic effect[e]	R^2 [f]	Previously reported MTAs[g]
86	14	SCM002772.2_29581828	29581828	4.6-3.1	PM_14D^{123}, PM_21D^{123}	-9.74, -5.74	3.7-6.6	Zhao *et al*. 2006 (11.7-28.7 Mb); Qasim *et al*. 2020 (28.37-37.43 Mb)
87	14	SCM002772.2_53077839	53077839	4.2-3.9	PM_14D^{123}	4.22	6.0	
88	14	SCM002772.2_65359864	65359864	4.5-3.1	LL_E1^{123}, LL_E2^{13}, LL_CombENV^{123}	0.82; 0.72	6.6-7.6	
	14	SCM002772.2_65359864	65359864	3.8-3.2	LW_E1^{123}, LW_E3^{123}, LW_CombENV^{23}	6.07, 3.28	5.5-6.8	
89	14	SCM002772.2_70479506	70479506	4.0-3.0	LW_E2^{3}, LW_E4^{123}	-6.83, -6.75	6.4	
90	15	SCM002773.2_9748366	9748366	3.6-3.2	LW_E4^{123}, LW_CombENV^{23}	4.15, 3.08	5.2-5.7	
	15	SCM002773.2_9748366	9748366	4.1-3.5	PM_21D^{123}	5.77, 3.23	4.3	
91	15	SCM002773.2_10201221	10201221	7.2-3.9	LW_E3^{123}	7.75, 6.43	7.8	
92	15	SCM002773.2_29580386	29580386	3.7-3.0	LL_E3^{3}, LL_E4^{23}, LL_CombENV^{23}	0.78, 0.60	5.1-6.0	
93	15	SCM002773.2_35983051	35983051	3.9-3.1	LW_E1^{23}	2.69; 1.52	5.6	
94	15	SCM002773.2_36238886	36238886	3.6-3.4	PM_21D^{23}	-3.94	4.2	
	15	SCM002773.2_36273958	36273958	3.7-3.4	PM_21D^{23}	-3.89	4.2	
95	15	SCM002773.2_38993115	38993115	3.9-3.0	LL_E1^{123}, LL_CombENV^{23}	0.51, 0.44	5.0-6.6	
96	16	SCM002774.2_14148427	14148427	3.6-3.4	PM_21D^{23}	6.91	4.1	
97	16	SCM002774.2_22279509	22279509	3.8-3.0	LL_E4^{123}, LL_CombENV^{123}	-0.45, -0.28	4.9-5.7	
98	16	SCM002774.2_22321613	22321613	3.5-3.1	LL_E4^{13}, LL_CombENV^{1}	0.39, 0.26		Zhao *et al*. 2006 (23.18-35.47 Mb)
99	16	SCM002774.2_22403960	22403960	4.0-3.0	LL_E4^{123}, LL_CombENV^{1}	-0.42, -0.27	5.0	
100	16	SCM002774.2_23440605	23440605	4.0-3.6	PM_21D^{23}	-8.68	4.5	
101	16	SCM002774.2_23837210	23837210	3.4-3.1	LL_E4^{13}, LL_CombENV^{13}	-0.36, -0.26		

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Locus	Chr[a]	SNP ID	Position (bp)	-log$_{10}$ (P)[c]	Traits, Env & Model[d]	Allelic effect[e]	R^2 [f]	Previously reported MTAs[g]
102	16	SCM002774.2_25673001	25673001	3.4-3.1	LW_E2^{123}, LW_E4^{123}	3.33, 3.05	5.0-5.2	
103	16	SCM002774.2_25678898	25678898	3.8-3.0	LL_E2^{23}, LL_E4^{123}	0.35, 0.23	4.7-6.0	Zhao *et al*. 2006 (23.18-35.47 Mb); Wu *et al*. 2016 (25.8-26.47 Mb)
	16	SCM002774.2_25678898	25678898	5.0-3.0	LW_E2^{123}, LW_E4^{123}, LW_CombENV^{23}	3.81, 2.15	4.9-7.6	
104	16	SCM002774.2_25739024	25739024	3.7-3.0	LL_E1^{123}, LL_E3^{13}, LL_E4^{1}, LL_CombENV^{123}	0.29, 0.21	5.0-5.4	
	16	SCM002774.2_25739024	25739024	10.9-3.4	LW_E3^{23}, LW_E4^{123}, LW_CombENV^{123}	2.78, 2.17	6.1-6.9	
	16	SCM002774.2_25739024	25739024	4.0-3.8	PM_14D^{123}	3.64	5.7	
105	16	SCM002774.2_33238118	33238118	3.4-3.0	LL_E3^{123}	-0.32	5.9	Zhao *et al*. 2006 (23.18-35.47 Mb) and (28.55-35.47 Mb); Wu *et al*. 2013 (30.28-34.59 Mb) and (34.59-36.61 Mb); Wei *et al*. 2016 (30.78-31.34 Mb); Qasim *et al*. 2020 (32.95-34.44 Mb)
	16	SCM002774.2_33238118	33238118	3.4-3.0	LW_E3^{23}	-2.91	5.8	
	16	SCM002774.2_33265642	33265642	3.4-3.1	LL_E3^{123}	0.31	6.2	
106	16	SCM002774.2_33563646	33563646	4.0-3.0	LL_E1^{123}, LL_E2^{1}, LL_E3^{123}, LL_E4^{1}, LL_CombENV^{123}	0.32, 0.25	5.4-6.0	
	16	SCM002774.2_33563646	33563646	3.4-3.0	LW_E1^{123}; LW_CombENV^{23}	2.16; 2.13	5.0-5.5	
	16	SCM002774.2_33563646	33563646	4.0-3.7	PM_14D^{123}	4.06	5.7	

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Locus	Chr[a]	SNP ID	Position (bp)	-log$_{10}$ (*P*)[c]	Traits, Env & Model[d]	Allelic effect[e]	R^2 [f]	Previously reported MTAs[g]
107	16	SCM002774.2_34137593	34137593	4.5-3.0	LL_E1^{123}, LL_E2^{123}, LL_E4^{123}, LL_CombENV^{123}	-0.36, -0.25	4.6-6.8	Zhao *et al*. 2006 (23.18-35.47 Mb) and (28.55-35.47 Mb); Wu *et al*. 2013 (30.28-34.59 Mb) and (34.59-36.61 Mb); Wei *et al*. 2016 (30.78-31.34 Mb); Qasim *et al*. 2020 (32.95-34.44 Mb)
108	16	SCM002774.2_34149554	34149554	3.8-3.0	LL_E2^{1}, LL_E4^{123}, LL_CombENV^{1}	0.37, 0.24	4.7	
109	16	SCM002774.2_39405155	39405155	4.0-3.4	LL_E3^{123}	-0.25	6.9	
	16	SCM002774.2_39405155	39405155	3.7-3.2	LW_E3^{23}	-2.23	6.3	
110	17	SCM002775.2_41131780	41131780	3.5-3.2	LW_E2^{23}	-2.58	5.0	
111	17	SCM002775.2_43529605	43529605	4.0-3.1	LL_E4^{123}; LL_CombENV^{12}	0.51; 0.34	5.1-6.3	
112	17	SCM002775.2_49740638	49740638	3.5-3.1	LL_E4^{23}; LL_CombENV^{23}	-0.53; -0.39	5.1-5.2	
	17	SCM002775.2_49740638	49740638	3.6-3.0	LW_E4^{123}, LW_CombENV^{3}	-5.08, -4.98	5.7	
113	17	SCM002775.2_56829232	56829232	3.7-3.0	LW_E1^{3}, LW_E4^{3}, LW_CombENV^{23}	-2.84	5.8	
114	18	SCM002776.2_7320194	7320194	4.0-3.7	PM_14D^{123}	6.27	5.7	
115	18	SCM002776.2_12692146	12692146	3.6-3.3	PM_21D^{23}	-4.71	4.0	
116	18	SCM002776.2_29886188	29886188	3.4-3.0	LL_E2^{123}, LL_E4^{23}	-0.45, -0.41	4.8-5.0	Wu *et al*. 2013 (31.40-32.04 Mb); (31.40-33.50 Mb)

Table A.6. List of significant single-nucleotide polymorphisms (SNPs) associated with stem lesion length (LL), stem lesion width (LW), and combENV plant mortality (PM_14D, PM_21D) traits for sclerotinia stem rot resistance identified by the FarmCPU, MLM, and GEMMA-MLM models (continued)

Locus	Chr[a]	SNP ID	Position (bp)	-log$_{10}$ (*P*)[c]	Traits, Env & Model[d]	Allelic effect[e]	R^{2} [f]	Previously reported MTAs[g]
117	18	SCM002776.2_37107013	37107013	3.8-3.5	LW_CombENV^{23}	3.33	5.9	Wu *et al.* 2013 (38.19-38.47 Mb)
118	18	SCM002776.2_41681055	41681055	3.5-3.3	PM_14D^{123}	7.14	5.0	
119	19	SCM002777.2_15808490	15808490	3.8-3.5	LL_E3^{123}	0.38	7.2	
120	19	SCM002777.2_28925630	28925630	3.4-3.1	LW_E1^{123}	4.64	5.7	
121	19	SCM002777.2_34413846	34413846	7.0-3.2	PM_21D^{123}	-10.25, -9.21	3.8	
122	19	SCM002777.2_46851981	46851981	4.0-3.1	LL_E1^{123}, LL_E3^{123}, LL_E4^{123}, LL_CombENV^{123}	-0.84, -0.59	5.3-6.8	Zhao *et al.* 2006 (46.58-46.75 Mb)
123	19	SCM002777.2_48885679	48885679	5.8-3.3	LL_E1^{123}, LL_E2^{123}, LL_E3^{123}, LL_E4^{123}, LL_CombENV^{123}	0.83, 0.52	5.8-9.2	
	19	SCM002777.2_48885679	48885679	8.2-3.8	LW_E1^{123}, LW_E2^{123}, LW_E3^{123}, LW_E4^{123}, LW_CombENV^{123}	7.60, 3.37	7.0-10.3	
	19	SCM002777.2_48885679	48885679	4.6-4.3	PM_14D^{123}	9.37	6.7	

Chr[a], *Brassica napus* chromosome

Alleles[b], (_/_) Major allele/minor allele

-log10 (*P*)[c], The highest and lowest -log10 (*P*) value resulted from the studied environments with different GWAS models where the identified SNPs -log10 (*P*) value ≥ 3.4 by at least of the GWAS models

Traits, Env & Model[d], Traits: LL, Lesion length; LW, Lesion width; PM_14D, Combined environment (CombENV) plant mortality at 14 days post inoculation (dpi); PM_21D, Combined environment (CombENV) plant mortality at 21 dpi; Environments: E1, Carrington 2019; E2, Landon 2019; E3, Carrington 2020; E4, Osnabrock 2020; CombENV, combined analysis across four environments; Model: superscript 1, 2 & 3 represents FarmCPU, MLM and GEMMA-MLM GWAS models respectively

Allelic effect[e], difference in mean stem lesion length (cm) between genotypes with major allele and minor allele. Positive sign indicates major allele is associated with increased lesion length (cm). Negative sign indicates that the major allele is associated with reduced lesion length (cm)

R^{2} [f], Percentage of phenotypic variation explained by the identified significant SNP derived from the results of MLM method

Previously reported MTAs g, previously reported marker-trait-associations (MTAs) along with their physical positions from various QTL and GWAS-related studies which overlapped or in close proximity with the genomic regions in this study.

Table A.7. Candidate genes for different sclerotinia stem rot resistance traits within 50 kb region at either side of the significant markers

Significant SNP	Chr[a]	Gene symbol and ID	Distance (kb)[b]	AT Equivalent	Gene Description	GO biological Function (TAIR)
SCM002759.2_1471693	1	LOC106353725	27.6	AT4G36690.1	splicing factor U2af large subunit A	defense response to bacterium
SCM002759.2_1766199	1	LOC106349418	-46.8	AT5G17880.1	disease resistance-like protein CSA1	signal transduction
SCM002759.2_1766199	1	LOC106349225	-38.0	AT5G45260.1	disease resistance protein RRS1-like	cell death, defense response
SCM002759.2_1766199	1	LOC106348260	15.0	AT4G36010.1	thaumatin-like protein 1b	defense response
SCM002759.2_1766199	1	LOC106347663	42.8	……..	la-related protein 1C-like	response to jasmonic acid; response to salicylic acid Source: UniProtKB
SCM002759.2_11482241	1	LOC106364078	-43.7	AT4G15400.1	BAHD acyltransferase BIA1-like	brassinosteroid mediated signaling pathway
SCM002759.2_11482241	1	LOC106364076	-28.1	AT5G39580.1	probable peroxidase 61	defense response to fungus
SCM002759.2_11482241	1	LOC106364075	-22.9	AT3G15356.1	Acidic endochitinase	cellular response to chitin, cellular response to jasmonic acid stimulus
SCM002759.2_11482241	1	LOC106364071	-6.4	AT5G24110.1	probable WRKY transcription factor 30	response to hydrogen peroxide, response to salicylic acid
SCM002759.2_11482241	1	LOC106364076	-28.1	AT5G39580.1	probable peroxidase 61	defense response to fungus
SCM002759.2_11482241	1	LOC106364075	-22.9	AT3G15356.1	acidic endochitinase	cellular response to chitin, cellular response to jasmonic acid stimulus

Table A.7. Candidate genes for different sclerotinia stem rot resistance traits within 50 kb region at either side of the significant markers (continued)

Significant SNP	Chr[a]	Gene symbol and ID	Distance (kb)[b]	AT Equivalent	Gene Description	GO biological Function (TAIR)
SCM002759.2_11482241	1	LOC106364071	-6.4	AT5G24110.1	probable WRKY transcription factor 30	response to hydrogen peroxide, response to salicylic acid
SCM002759.2_11482241	1	LOC106366011	45.2	……..	glucan endo-1,3-beta-glucosidase-like	defense response Source: UniProtKB-KW
SCM002760.2_1865667	2	LOC106428331	27.6	AT1G18870.1	isochorismate synthase 2, chloroplastic-like	salicylic acid biosynthetic process, phylloquinone biosynthetic process
SCM002760.2_1865667	2	LOC106431790	49.5	AT5G01900.1	probable WRKY transcription factor 62	salicylic acid mediated signaling pathway; defense response to bacterium
SCM002761.2_36170515	3	LOC106440304	-42	AT3G05800.1	transcription factor bHLH147-like	brassinosteroid mediated signaling pathway
SCM002762.2_16168004	4	LOC106450030	-11.9	AT2G19190.1	probable leucine-rich repeat receptor-like protein kinase At2g28990	defense response to bacterium
SCM002762.2_20212672	4	LOC106447450	-1.5	AT2G39940.1	coronatine-insensitive protein 1-like	defense response to fungus, jasmonic acid mediated signaling pathway, response to wounding
SCM002763.2_15914063	5	BNAA05G15190D	-15.6	…….	stress-response A/B barrel domain-containing protein DABB1	defense response to fungus, incompatible interaction Source: TAIR
SCM002763.2_28608175	5	LOC106415470	4.1	AT4G27320.1	universal stress protein PHOS34-like	response to molecule of fungal origin

Table A.7. Candidate genes for different sclerotinia stem rot resistance traits within 50 kb region at either side of the significant markers (continued)

Significant SNP	Chr[a]	Gene symbol and ID	Distance (kb)[b]	AT Equivalent	Gene Description	GO biological Function (TAIR)
SCM002764.2_25241979	6	LOC106351996	41.1	AT5G24400.1	probable 6-phosphogluconolactonase 3	cellular response to redox state, defense response to oomycetes, defense response to bacterium
SCM002764.2_25241979	6	LOC106348257	48.3	AT5G24400.1	probable 6-phosphogluconolactonase 3	cellular response to redox state, defense response to oomycetes, defense response to bacterium
SCM002764.2_29348402	6	LOC106408214	15	AT5G47910.1	respiratory burst oxidase homolog protein D-like	defense response to fungus, negative regulation of programmed cell death, response to wounding
SCM002767.2_36527400	9	LOC106411656	-45.4	AT4G28110.1	transcription factor MYB41	response to chitin
SCM002767.2_36527400	9	LOC106415606	23.3	AT3G61440.1	bifunctional L-3-cyanoalanine synthase/cysteine synthase C1, mitochondrial-like	immune response
SCM002767.2_36527400	9	LOC106416331	34	AT3G61460.1	E3 ubiquitin-protein ligase RHA1B	response to brassinosteroid, response to chitin
SCM002767.2_37671479	9	LOC106368555	33	AT2G25620.1	probable protein phosphatase 2C 22	regulation of defense response to virus

Table A.7. Candidate genes for different sclerotinia stem rot resistance traits within 50 kb region at either side of the significant markers (continued)

Significant SNP	Chr[a]	Gene symbol and ID	Distance (kb)[b]	AT Equivalent	Gene Description	GO biological Function (TAIR)
SCM002767.2_37671479	9	LOC106365129	40.3	AT5G59270.1	putative L-type lectin-domain containing receptor kinase II.2	defense response to oomycetes; defense response to bacterium
SCM002767.2_37671479	9	LOC106363673	48.4	AT5G27420.1	NEP1-interacting protein-like 2	defense response to other organism, response to chitin
SCM002767.2_43331392	9	LOC106399775	15.3	AT1G02930.1	glutathione S-transferase F6-like	response to oxidative stress, toxin catabolic process
SCM002767.2_45841268	9	LOC106369308	42.5	AT1G09340.1	chloroplast stem-loop binding protein of 41 kDa a, chloroplastic	response to wounding
SCM002768.2_2190513	10	LOC106371170	-3	AT1G05630.1	type I inositol polyphosphate 5-phosphatase 13-like	response to wounding
SCM002768.2_5144576	10	LOC106370828	-33.3	AT1G02120.1	protein VASCULAR ASSOCIATED DEATH 1, chloroplastic	negative regulation of programmed cell death, response to ethylene, response to salicylic acid
SCM002769.2_6223969	11	LOC106418377	-31	AT1G61850.1	patatin-like protein 8	jasmonic acid biosynthetic process
SCM002769.2_6223969	11	LOC106416990	-28.8	AT1G61850.1	patatin-like protein 8	jasmonic acid biosynthetic process

Table A.7. Candidate genes for different sclerotinia stem rot resistance traits within 50 kb region at either side of the significant markers (continued)

Significant SNP	Chr[a]	Gene symbol and ID	Distance (kb)[b]	AT Equivalent	Gene Description	GO biological Function (TAIR)
SCM002769.2_46192098	11	LOC106435207	73.7	AT3G17860.1	protein TIFY 6B-like	regulation of defense response, regulation of jasmonic acid mediated signaling pathway
SCM002771.2_7740873	13	LOC106418114	-26.4	AT2G01980.1	sodium/hydrogen exchanger 7-like	response to oxidative stress, response to reactive oxygen species
SCM002771.2_7740873	13	LOC111198287	-17.7	AT2G01980.1	sodium/hydrogen exchanger 7-like	response to oxidative stress, response to reactive oxygen species
SCM002771.2_22853068	13	LOC106420603	-4.7	AT2G39660.1	serine/threonine-protein kinase BIK1-like	defense response to fungus, pattern recognition receptor signaling pathway
SCM002771.2_22853068	13	LOC106420698	20.6	AT2G39730.1	ribulose bisphosphate carboxylase/oxygenase activase, chloroplastic-like	response to jasmonic acid
SCM002771.2_48097242	13	LOC111203738	15.5	AT2G18060.1	NAC domain-containing protein 37-like	response to chitin
SCM002771.2_78761785	13	LOC106427438	-39.7	AT1G51700.1	dof zinc finger protein DOF1.7	response to chitin
SCM002772.2_29581828	14	LOC106429730	38	AT1G61120.1	(E, E)-geranyllinalool synthase-like	response to jasmonic acid, response to wounding

Table A.7. Candidate genes for different sclerotinia stem rot resistance traits within 50 kb region at either side of the significant markers (continued)

Significant SNP	Chr[a]	Gene symbol and ID	Distance (kb)[b]	AT Equivalent	Gene Description	GO biological Function (TAIR)
SCM002772.2_29581828	14	LOC106429715	9.5	AT1G61120.1	(E,E)-geranyllinalool synthase-like	response to jasmonic acid, response to wounding
SCM002772.2_65359864	14	LOC106403148	-21.9	AT3G49120.1	peroxidase C3	defense response to fungus, pattern recognition receptor signaling pathway
SCM002772.2_65359864	14	LOC111205825	-15.8	AT3G49120.1	peroxidase C3-like	defense response to fungus, pattern recognition receptor signaling pathway
SCM002772.2_65359864	14	LOC106402880	-8.6	AT3G49120.1	peroxidase C3	defense response to fungus, pattern recognition receptor signaling pathway
SCM002772.2_65359864	14	LOC106402879	-4.6	AT3G49120.1	peroxidase C3-like	defense response to fungus, pattern recognition receptor signaling pathway,
SCM002772.2_65359864	14	LOC106405881	7.1	AT2G38470.1	probable WRKY transcription factor 33	defense response to fungus, response to chitin, systemic acquired resistance, camalexin biosynthetic process
SCM002772.2_65359864	14	LOC106406388	40.6	AT2G38530.1	non-specific lipid-transfer protein A	programmed cell death
SCM002772.2_70479506	14	LOC106392319	-5.1	AT4G27320.1	universal stress protein PHOS34-like	response to molecule of fungal origin
SCM002773.2_38993115	15	LOC106435386	44.8	……..	protein EMSY-LIKE 1-like	defense response to fungus
SCM002773.2_38993115	15	LOC106435387	-38	AT3G12500.1	endochitinase CH25	defense response to fungus, jasmonic acid and ethylene-dependent systemic resistance

Table A.7. Candidate genes for different sclerotinia stem rot resistance traits within 50 kb region at either side of the significant markers (continued)

Significant SNP	Chr[a]	Gene symbol and ID	Distance (kb)[b]	AT Equivalent	Gene Description	GO biological Function (TAIR)
SCM002773.2_38993115	15	LOC106435413	-35.7	AT3G12490.2	cysteine proteinase inhibitor 6-like	response to oxidative stress
SCM002773.2_38993115	15	LOC106435414	-22.9	……..	nascent polypeptide-associated complex subunit alpha-like protein 1	wound healing Source: BHF-UCL
SCM002774.2_22279509	16	LOC106411532	21.4	……..	aldehyde oxidase GLOX-like	defense response to fungus Source: UniProtKB
SCM002774.2_22403960	16	LOC106410769	-5.8	AT5G54430.1	universal stress protein PHOS34	response to molecule of fungal origin
SCM002774.2_23837210	16	LOC106406682	-33	AT3G56400.1	probable WRKY transcription factor 70	indole glucosinolate biosynthetic process, induced systemic resistance, jasmonic acid mediated signaling pathway
SCM002774.2_25678898	16	LOC106428228	43.3	……..	F-box/LRR-repeat protein At3g59200-like	defense response to oomycetes
SCM002774.2_33563646	16	LOC106426248	6.8	AT2G37710.1	L-type lectin-domain containing receptor kinase IV.2-like	response to salicylic acid, defense response to oomycetes
SCM002774.2_34137593	16	LOC106352125	33.9	AT1G66700.1	paraxanthine methyltransferase 1	response to molecule of fungal origin, response to salicylic acid
SCM002775.2_56829232	17	LOC106409984	-18.6	AT2G14610.1	pathogenesis-related protein PR-1	systemic acquired resistance

Table A.7. Candidate genes for different sclerotinia stem rot resistance traits within 50 kb region at either side of the significant markers (continued)

Significant SNP	Chr[a]	Gene symbol and ID	Distance (kb)[b]	AT Equivalent	Gene Description	GO biological Function (TAIR)
SCM002776.2_29886188	18	LOC106359636	-40	AT3G55970.1	probable 2-oxoglutarate-dependent dioxygenase JRG21	regulation of jasmonic acid mediated signaling pathway, regulation of defense response to fungus
SCM002776.2_29886188	18	LOC106360811	-18.1	AT3G55980.1	zinc finger CCCH domain-containing protein 47	response to chitin
SCM002777.2_46851981	19	LOC106372283	-42.5	AT5G13580.1	ABC transporter G family member 6	response to nematode
SCM002777.2_48885679	19	LOC106415792	34.22	……..	probable disease resistance protein RPP1	TIR-NB-LRR receptor-like protein that confers resistance to the pathogen, defense response to fungus
SCM002777.2_48885679	19	LOC106416705	-21.9	…….	transcriptional corepressor SEUSS	response to fungus, response to oxidative stress

Chr[a]: *Brassica napus* chromosome

Distance (kb)[a]: negative values= genes are downstream of the marker, positive values = genes are upstream of the marker

Table A.8. Name, origin, and growth habits of the 339 germplasm accessions (including check cultivars) used in the seedling stage sclerotinia stem rot resistance study

Name of the accessions	Country of origin/obtained	Growth habit
Pioneer 45S51[a]	**Pioneer (Check)**	**Spring**
Pioneer 45S56[a]	**Pioneer (Check)**	**Spring**
Abilene	USA	Winter
ARC 97019	USA	Winter
Galant	USA	Spring
Galaxy	Sweden	Spring
Galileo	Canada	Winter
Gebr Dippes	South Korea	Winter
Gido	Germany	Spring
Girita	Germany	Semi-winter
Gisora	Germany	Spring
GK Savaria	Hungary	Winter
Glacier	Sweden	Winter
Global	Sweden	Spring
ARC-2180-1	USA	Winter
Golden	Canada	Spring
Gora	Germany	Spring
Goya	Canada	Winter
Gulle	Sweden	Spring
Gullivar	Sweden	Spring
Gylle	South Korea	Semi-winter
Helga	Germany	Semi-winter
Hi-Q	Canada	Spring
Armander	South Korea	
HOBSON	UK	Winter
Host rape regel	South Korea	Winter
Ibiza	Canada	Winter
INRA-R-2000	France	Spring
IR-2	Hungary	Spring
Isek urodane	South Korea	Semi-winter
Iwao natane	South Korea	Winter
Iwashiro-natane	South Korea	Winter

Table A.8. Name, origin, and growth habits of the 339 germplasm accessions (including check cultivars) used in the seedling stage sclerotinia stem rot resistance study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Iwawoochi	South Korea	Winter
Janetzkis	South Korea	Spring
Janpol	Poland	Winter
Jantar	Poland	Winter
Jasna	Serbia	Spring
Jet Neuf	Canada	Winter
Jupiter	USA	Winter
Kanada	Poland	Spring
Kasuya	South Korea	Winter
Kasuyashu	South Korea	Winter
Arwin	Germany	Winter
Klinki	South Korea	Spring
Korina	Germany	Winter
Kosa	Germany	Spring
Koubun	South Korea	Spring
Kovalevskjj	Ukraine	Spring
Kraphhauser	South Korea	Spring
Krasnodarskii	Russian Federation	Winter
Kritmar rape	South Korea	Spring
KS3579	USA	Winter
Kuju	South Korea	Winter
Kutkowski	South Korea	Winter
Ladoga	Canada	Winter
Laura	Germany	Spring
Legend	Sweden	Spring
Lembkes	South Korea	Winter
Lesira	Germany	Winter
Lester	Germany	Winter
Librador	Germany	Winter
Lieikoposki	South Korea	Semi-winter
Aviso	Canada	Spring
Lifura	South Korea	Spring

Table A.8. Name, origin, and growth habits of the 339 germplasm accessions (including check cultivars) used in the seedling stage sclerotinia stem rot resistance study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Lindora-00	Germany	Winter
Lindore	Germany	Winter
Linglandor	Germany	Winter
Linus	South Korea	Winter
Lirabon-00	Germany	Winter
Lirama	Germany	Winter
Azuma	South Korea	Semi-winter
Liratrop	Germany	Winter
Liropa	Germany	Winter
Lisora	Germany	Semi-winter
Lorenze	Canada	Winter
Luna	Germany	Winter
Major	France	Semi-winter
Mali	South Korea	Semi-winter
Azumasho	South Korea	Semi-winter
Mar 160059	Poland	Winter
Marinus	Germany	Winter
Mar'janovskij	Ukraine	Spring
Matador	South Korea	Semi-winter
Mazowiecki	Poland	Spring
Mendel	Germany	Winter
Midas	Canada	Spring
Miekuro Dane	South Korea	Spring
Mihonatane	South Korea	Winter
Baraska	Germany	Winter
Miochowski	France	Semi-winter
Mirander	Germany	Winter
Mlochowski	Poland	Semi-winter
MR 1	South Korea	Winter
Mu.che!	South Korea	Winter
Mulchower	South Korea	Winter
Mura yamasho	South Korea	Spring

Table A.8. Name, origin, and growth habits of the 339 germplasm accessions (including check cultivars) used in the seedling stage sclerotinia stem rot resistance study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Murame nadame	South Korea	Semi-winter
Barkant	Netherlands	Winter
N001-28-246-5-4	South Korea	Semi-winter
Nabo	South Korea	Semi-winter
NDC-A14026	USA	Spring
NDC-A14032	USA	Spring
NDC-A14033	USA	Spring
NDC-A14035	USA	Spring
NDC-A14036	USA	Spring
NDC-A14045	USA	Spring
NDC-A14046	USA	Spring
NDC-A14050	USA	Spring
Barnapoli	Germany	Winter
NDC-A14055	USA	Spring
NDC-A14056	USA	Spring
NDC-E12009	USA	Spring
NDC-E12023	USA	Spring
NDC-E12025	USA	Spring
NDC-E12027	USA	Spring
NDC-E12044	USA	Spring
NDC-E12079	USA	Spring
NDC-E12081	USA	Spring
NDC-E12086	USA	Spring
Barplina	South Korea	Winter
NDC-E12119	USA	Spring
NDC-E12120	USA	Spring
NDC-E12121	USA	Spring
NDC-E12131	USA	Spring
NDC-E12133	USA	Spring
NDC-E13193	USA	Spring
NDC-E13279	USA	Spring
NDC-E13285	USA	Spring

Table A.8. Name, origin, and growth habits of the 339 germplasm accessions (including check cultivars) used in the seedling stage sclerotinia stem rot resistance study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
NDC-E15031	USA	Spring
NDC-E15146	USA	Spring
Beryl	Poland	Winter
NDC-E15174	USA	Spring
NDC-E15200	USA	Spring
NDC-E15234	USA	Spring
NDC-E15294	USA	Spring
NDC-E16015	USA	Spring
NDC-E16053	USA	Spring
NDC-E16152	USA	Spring
NDC-E16169	USA	Spring
NDC-E16198	USA	Spring
NDC-E17132	USA	Spring
Bienvenu	USA	Winter
NDSU01104	USA	Spring
NDSU0417	USA	Spring
NDSU0472	USA	Spring
NDSU0473	USA	Spring
NDSU0474	USA	Spring
NDSU0475	USA	Spring
NDSU0521	USA	Spring
NDSU0522	USA	Spring
NDSU0619	USA	Spring
NDSU0620	USA	Spring
Billy	Sweden	Winter
NDSU0726	USA	Spring
NDSU0728	USA	Spring
NDSU0729	USA	Spring
NDSU10999	USA	Spring
NDSU12989	USA	Spring
NDSU151000	USA	Spring
NDSU15989	USA	Spring

Table A.8. Name, origin, and growth habits of the 339 germplasm accessions (including check cultivars) used in the seedling stage sclerotinia stem rot resistance study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
NDSU161013	USA	Spring
NDSU31001	USA	Spring
NDSU31011	USA	Spring
Bingo	USA	Spring
NDSU41000	USA	Spring
NDSU7997	USA	Spring
NDSU81000	USA	Spring
NDSU91013	USA	Spring
Nemercanskjj 2268	Ukraine	Winter
Niedera-rubacher	South Korea	Winter
Nilla 1022	South Korea	Semi-winter
Nilla glossy	South Korea	Semi-winter
NU 41737	Turkey	Spring
NU 51084	Sweden	Spring
Nugget	South Korea	Semi-winter
NY-10	China	Semi-winter
NY-12	China	Semi-winter
NY-20	China	Semi-winter
NY-7	China	Semi-winter
NY-8	China	Semi-winter
O 84	China	Semi-winter
Oleifera	South Korea	Semi-winter
BO-63	Canada	Spring
Oro	Canada	Spring
Orpal	France	Spring
Panter	Germany	Winter
Peace	Canada	Spring
Petanova-lihonova	South Korea	Semi-winter
Polo canola	USA	Spring
Premier	USA	Spring
Bolko	Poland	Winter
Primer	France	Semi-winter

Table A.8. Name, origin, and growth habits of the 339 germplasm accessions (including check cultivars) used in the seedling stage sclerotinia stem rot resistance study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Printol	USA	Spring
Prota	Germany	Spring
Q2	Canada	Spring
Quinta	Germany	Winter
R. Creaus	South Korea	Winter
Rafal	France	Winter
Ramses	South Korea	Winter
Rang	South Korea	Semi-winter
BRA 1168/85	Italy	Winter
Rapifera	South Korea	Winter
Ratnik	Serbia	Spring
Rebel	USA	Semi-winter
Regal	South Korea	Winter
Regent	Canada	Spring
Regina II	Canada	Spring
Reston	USA	Spring
Rico	Germany	Spring
Ridana	Germany	Winter
Bridger	USA	Winter
Riley	USA	Winter
Romeo	France	Spring
Rubin	Germany	Winter
Ruby	USA	Rutabaga
S.V. Gulle	South Korea	Spring
Santana	Germany	Winter
Scherwitz	South Korea	Winter
Sei yoshu	South Korea	Semi-winter
Seoul	South Korea	Spring
Sera	Germany	Semi-winter
Siberian	USA	Winter
Silesia	Czech Republic	Winter
Silex	Canada	Spring

Table A.8. Name, origin, and growth habits of the 339 germplasm accessions (including check cultivars) used in the seedling stage sclerotinia stem rot resistance study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Skrzeszowicki	Poland	Winter
Start	Poland	Winter
Brio	France	Spring
Status	Sweden	Winter
Su weon chag	South Korea	Semi-winter
Sumner	USA	Winter
Sunrise	USA	Spring
Sval of Gullen	South Korea	Spring
Svaloefs Karab	Sweden	Winter
Svalof Victoria	South Korea	Winter
Synra	South Korea	Winter
Taichang	South Korea	Semi-winter
Bronowski	Poland	Spring
Taiwan	Taiwan	Spring
Takagi MS	South Korea	Semi-winter
Tamara	Germany	Winter
Tanka	South Korea	Semi-winter
TANTAL	France	Semi-winter
Tanto	France	Spring
Target	Sweden	Spring
Titus	South Korea	Winter
Todane	South Korea	Semi-winter
Buk Wuk 3	South Korea	Spring
Tokiwa	South Korea	Semi-winter
Tonus	South Korea	Spring
Topas	Sweden	Spring
Tosharshu	South Korea	Winter
Tower	Canada	Spring
Trebicska	Czech Republic	Winter
Tsukushishu	South Korea	Winter
Turret	Canada	Spring
Capricorn	UK	Winter

Table A.8. Name, origin, and growth habits of the 339 germplasm accessions (including check cultivars) used in the seedling stage sclerotinia stem rot resistance study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Ujfertodi	Hungary	Winter
Valdor	France	Winter
Vanda	Germany	Winter
Viking	Denmark	Winter
Vinnickij 15/59	Ukraine	Winter
Vision	Canada	Winter
Vostochno-sibirskii	Russian Federation	Spring
Wasefuji	South Korea	Spring
Weal dong cho	South Korea	Semi-winter
Weibulls margo	South Korea	Semi-winter
Cascade	USA	Winter
Westar[b]	**Canada**	**Spring**
Wichita	USA	Winter
Wielkopolski	South Korea	Winter
Willa	South Korea	Spring
Winfield	USA	Spring
Wipol	Norway	Semi-winter
Wira	Germany	Winter
Yong dang	South Korea	Semi-winter
Yonkkaichi kwo	South Korea	Semi-winter
Cathy	USA	Winter
Yonkokuban	South Korea	Winter
Zhoungyou-584	China	Semi-winter
Zhoungyou-821	China	Semi-winter
Zhoungyou-9	China	Semi-winter
NEP-63	USA	Winter
Aspen	USA	Winter
LB2125	Canada	
Celebra	Sweden	Spring
Norin 16	Japan	
Licantara	Germany	Winter
Merrick	USA	Winter

Table A.8. Name, origin, and growth habits of the 339 germplasm accessions (including check cultivars) used in the seedling stage sclerotinia stem rot resistance study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Cescaljarni repka	South Korea	Semi-winter
Ceskia Tabor	Czech Republic	Spring
Chisaya natane	Japan	Semi-winter
Chon nam	South Korea	Semi-winter
CHUN-NUNG 1	China	Winter
Cobra	Germany	Winter
Colza 18 Miroc	South Korea	Semi-winter
Comet	Sweden	Spring
AR-256	Russian Federation	Winter
Conquest	Canada	Spring
Corvette	UK	Winter
Cougar	Canada	Spring
Cresor	France	Spring
Cresus	France	Spring
Crop	France	Spring
Crystal	Sweden	Winter
AR91004	USA	Winter
Cult	Canada	Winter
Czyzowski	Poland	Spring
Czyzowskich	Poland	Semi-winter
Da vinci	Canada	Winter
Dae cho sen	South Korea	Semi-winter
Delta	Sweden	Spring
DKW-46-5	USA	Winter
Dong Buk	South Korea	Winter
AR91017	USA	Winter
Doon Major Swede	New Zealand	Winter
Drakkar	France	Spring
Dramor	Poland	Winter
Drawft	South Korea	Winter
ECD06	UK	Winter
ECD07	UK	Winter

Table A.8. Name, origin, and growth habits of the 339 germplasm accessions (including check cultivars) used in the seedling stage sclerotinia stem rot resistance study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
ECD08	UK	Winter
ECD09	UK	Winter
ARC 90016	USA	Winter
Eckendorfer Mali	South Korea	Semi-winter
Elena	Germany	Winter
Englu	South Korea	Winter
Eragi	Germany	Winter
Erra	Germany	Winter
Evvin	Russian Federation	Spring
Expander	Germany	Winter
Fashion	Canada	Winter
ARC 97018	USA	Winter
Fertodi	South Korea	Winter
Flint	USA	Spring
Fonto	South Korea	Spring
France 1	France	Spring
France 12	France	Winter
Fuji	South Korea	Spring/ semi
G 32327	Albania	Winter

[a] Accessions used as resistant check
[b] Accession used as susceptible check

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
1	LP_4dpi	SCM002759.2_1538720	1	1538720			FarmCPU	0.110	3.1	
2	LP_7dpi	SCM002759.2_1842985	1	1842985			FarmCPU	0.068	3.0	
3	DW	SCM002759.2_1985056	1	1985056			mrMLM	-0.198	3.2	2.70
4	LP_4dpi	SCM002759.2_5225099	1	5225099			FarmCPU	-0.065	3.1	
5	DW	SCM002759.2_7316181	1	7316181	GEMMA-MLM	3.6	MLMM	-0.220	3.4	2.44
	LP_7dpi	SCM002759.2_7316181	1	7316181	GEMMA-MLM	3.6	MLMM, FarmCPU	0.044-0.046	3.3-3.5	3
	LP_7dpi	SCM002759.2_7376806	1	7376806			mrMLM	-0.035	3.7	2.90
6	LP_7dpi	SCM002759.2_10122721	1	10122721			MLMM	0.080	3.1	2.63
7	DW	SCM002759.2_10302868	1	10302868			MLMM	-0.179	3.1	2.20
8	LP_3dpi	SCM002759.2_12777356	1	12777356			FarmCPU	-0.089	6.0	
9	DW	SCM002759.2_20706751	1	20706751			FarmCPU	0.135	3.8	
10	LP_7dpi	SCM002759.2_31473630	1	31473630			MLMM, FarmCPU	0.059, 0.066	3.0-3.3	2.48
11	DW	SCM002760.2_5112867	2	5112867			FarmCPU	0.150	3.2	
12	DW	SCM002760.2_5283289	2	5283289			mrMLM	-0.204	7.0	4.39
	LP_7dpi	SCM002760.2_5283289	2	5283289			FarmCPU	-0.038	3.0	
13	DW	SCM002760.2_9186001	2	9186001			FarmCPU	-0.336	3.3	
14	LP_3dpi	SCM002760.2_11237590	2	11237590			FarmCPU	0.182	3.2	
15	LP_7dpi	SCM002760.2_14854978	2	14854978			mrMLM	0.041	3.8	4.05
16	LP_7dpi	SCM002760.2_18393359	2	18393359			MLMM	-0.074	3.3	2.77
17	LP_7dpi	SCM002760.2_20228240	2	20228240			FarmCPU	0.088	3.3	
18	LP_4dpi	SCM002760.2_20844443	2	20844443			MLMM, mrMLM	-0.174, 0.128	3.1-3.3	2.3-4.3
19	DW	SCM002760.2_30564102	2	30564102			mrMLM	-0.196	4.7	5.03
	LP_4dpi	SCM002760.2_30564102	2	30564102			MLMM	0.094	3.0	2.06

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
20	DW	SCM002761.2_1762853	3	1762853			FarmCPU	-0.123	3.2	
21	DW	SCM002761.2_3034487	3	3034487			FarmCPU	0.217	3.7	
22	LP_7dpi	SCM002761.2_4059294	3	4059294			FarmCPU	0.077	3.0	
23	LP_3dpi	SCM002761.2_5046706	3	5046706			MLMM, mrMLM	0.119, 0.109	3.2-3.5	2.08-2.40
24	DW	SCM002761.2_5607745	3	5607745	GEMMA-MLM	3.7	MLMM	-0.282	3.6	2.56
	LP_4dpi	SCM002761.2_5607745	3	5607745	GEMMA-MLM	3.5	MLMM, FarmCPU, mrMLM	-0.117, 0.139	3.4-5.7	2.4-2.7
25	LP_3dpi	SCM002761.2_6227578	3	6227578			FarmCPU	0.098	5.6	NA
26	DW	SCM002761.2_20918043	3	20918043			MLMM	-0.175	3.0	2.06
	LP_3dpi	SCM002761.2_20918043	3	20918043			MLMM, FarmCPU, mrMLM	-0.100, 0.110	3.0-7.9	1.99-3.07
	LP_4dpi	SCM002761.2_20918043	3	20918043			MLMM, mrMLM	-0.083, 0.093	3.2-5.9	2.23-2.59
	DW	SCM002761.2_20921893	3	20921893			MLMM, FarmCPU	0.107, 0.145	3.0-4.1	2.00
27	DW	SCM002761.2_23584078	3	23584078			MLMM	0.226	3.0	2.10
28	LP_7dpi	SCM002761.2_23664593	3	23664593	GEMMA-MLM	4.2	MLMM, FarmCPU	0.035,0.038	3.3-4.0	3.60
29	LP_3dpi	SCM002761.2_24009249	3	24009249			MLMM	0.212	3.2	2.12
30	LP_7dpi	SCM002761.2_31481512	3	31481512			mrMLM	0.030	3.4	3.66
31	LP_4dpi	SCM002761.2_31766146	3	31766146			FarmCPU	0.103	3.4	
32	LP_3dpi	SCM002761.2_31948050	3	31948050			FarmCPU	-0.099	6.1	
33	DW	SCM002761.2_32875227	3	32875227			mrMLM	0.196	5.4	3.36

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
33	DW	SCM002761.2_32875227	3	32875227			mrMLM	0.196	5.4	3.36
34	DW	SCM002761.2_33164427	3	33164427			mrMLM	-0.143	4.0	2.19
35	LP_3dpi	SCM002761.2_34924695	3	34924695			FarmCPU	0.096	3.1	
36	DW	SCM002761.2_45757845	3	45757845			mrMLM	-0.269	4.0	9.30
	LP_4dpi	SCM002761.2_45757845	3	45757845			MLMM	0.156	3.1	2.11
37	LP_3dpi	SCM002761.2_45882841	3	45882841			MLMM	0.228	3.0	2.00
38	LP_7dpi	SCM002762.2_263464	4	263464			FarmCPU	-0.077	3.1	
39	LP_7dpi	SCM002762.2_10716343	4	10716343	GEMMA-MLM	4.3	MLMM, FarmCPU, mrMLM	0.072, 0.084	3.6-4.8	3.5-6.8
40	LP_7dpi	SCM002762.2_11910128	4	11910128			MLMM	0.064	3.3	2.78
41	LP_3dpi	SCM002762.2_13620028	4	13620028	GEMMA-MLM	3.4	MLMM	0.108	3.2	2.10
	LP_4dpi	SCM002762.2_13620028	4	13620028	GEMMA-MLM	4.3	MLMM, FarmCPU, mrMLM	0.058, 0.111	3.3-5.3	1.50-2.76
42	DW	SCM002762.2_14955165	4	14955165			mrMLM	'0.265	6.4	5.21
43	DW	SCM002762.2_15836859	4	15836859			mrMLM	0.185	3.7	4.50
44	LP_7dpi	SCM002762.2_19685094	4	19685094	GEMMA-MLM	3.8	MLMM, FarmCPU	0.052, 0.055	3.5	3.00
45	LP_3dpi	SCM002762.2_21806203	4	21806203			FarmCPU	0.098	3.9	
46	DW	SCM002763.2_659551	5	659551			FarmCPU	0.392	3.4	
47	LP_7dpi	SCM002763.2_746381	5	746381	GEMMA-MLM	4.0	MLMM, FarmCPU	-0.115, -0.108	3.6-3.8	3.30
48	DW	SCM002763.2_1914189	5	1914189	GEMMA-MLM	3.3	MLMM	0.298	3.1	2.20
	LP_7dpi	SCM002763.2_1914189	5	1914189			MLMM, FarmCPU, mrMLM	-0.075, 0.066	3.8-5.4	4.33-5.30
49	LP_7dpi	SCM002763.2_2070843	5	2070843			MLMM, mrMLM	-0.033, 0.028	3.0-3.4	2.47-2.55

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
50	LP_4dpi	SCM002763.2_12372445	5	12372445			MLMM	-0.129	3.0	2.06
51	LP_7dpi	SCM002763.2_19842238	5	19842238			FarmCPU	0.047	3.0	
52	LP_7dpi	SCM002763.2_26512878	5	26512878			FarmCPU	-0.082	4.2	
53	LP_3dpi	SCM002763.2_28498462	5	28498462	GEMMA-MLM	3.8	MLMM	0.207	3.6	2.50
	LP_4dpi	SCM002763.2_28498462	5	28498462			MLMM	0.169	3.0	2.05
54	DW	SCM002763.2_28503051	5	28503051			MLMM	-0.244	3.3	2.30
55	DW	SCM002763.2_29583961	5	29583961			MLMM	-0.271	3.1	2.20
56	DW	SCM002764.2_2497618	6	2497618			MLMM	-0.190	3.2	2.24
57	LP_7dpi	SCM002764.2_3011988	6	3011988	GEMMA-MLM	4.2	MLMM, FarmCPU	-0.064, -0.063	3.7-4.0	3.50
58	LP_3dpi	SCM002764.2_7193358	6	7193358	GEMMA-MLM	3.5	MLMM, FarmCPU	-0.095, -0.064	3.1-3.4	2.24
	LP_4dpi	SCM002764.2_7193358	6	7193358	GEMMA-MLM	3.7	MLMM	-0.090	3.6	2.54
59	LP_3dpi	SCM002764.2_7381974	6	7381974			MLMM	0.103	3.3	2.20
	LP_4dpi	SCM002764.2_7381974	6	7381974			MLMM	0.089	3.0	2.08
60	DW	SCM002764.2_7471454	6	7471454			MLMM, FarmCPU, mrMLM	-0.192, 0.174	3.0-7.6	2.17-4.74
	LP_3dpi	SCM002764.2_7471454	6	7471454	GEMMA-MLM	4.4	MLMM, FarmCPU, mrMLM	-0.117, 0.104	3.8-8.7	2.9-4.2
	LP_4dpi	SCM002764.2_7471454	6	7471454	GEMMA-MLM	4.0	MLMM, FarmCPU, mrMLM	-0.100, 0.086	3.7-4.8	2.66-3.56
61	LP_4dpi	SCM002764.2_10061186	6	10061186			FarmCPU	-0.116	3.1	
62	DW	SCM002764.2_15841609	6	15841609			FarmCPU	0.198	3.8	
63	LP_3dpi	SCM002764.2_20413466	6	20413466			MLMM	0.144	3.2	2.10
64	LP_3dpi	SCM002764.2_33897878	6	33897878			FarmCPU	-0.163	3.3	
65	DW	SCM002765.2_3858871	7	3858871			FarmCPU	0.163	3.0	
66	LP_7dpi	SCM002765.2_11787278	7	11787278			MLMM	0.054	3.1	2.63

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
67	LP_4dpi	SCM002765.2_15407314	7	15407314			FarmCPU	-0.086	3.3	
68	DW	SCM002765.2_22697279	7	22697279			FarmCPU	0.438	3.9	
69	LP_7dpi	SCM002765.2_26044299	7	26044299			MLMM	-0.050	3.4	2.88
70	DW	SCM002765.2_26141436	7	26141436			FarmCPU	-0.131	3.2	
	LP_4dpi	SCM002765.2_26141436	7	26141436			FarmCPU	0.076	3.6	
71	DW	SCM002766.2_3646103	8	3646103			MLMM	-0.237	3.2	2.26
72	DW	SCM002766.2_6170383	8	6170383			FarmCPU	0.351	3.3	
73	LP_4dpi	SCM002766.2_6576920	8	6576920			FarmCPU	-0.053	3.2	
74	DW	SCM002766.2_8308223	8	8308223	GEMMA-MLM	3.7	MLMM, FarmCPU	-0.516, -0.348	3.5-4.7	2.49
	LP_7dpi	SCM002766.2_8308223	8	8308223			MLMM	0.093	3.0	2.50
75	LP_3dpi	SCM002766.2_8408776	8	8408776			mrMLM	0.101	3.0	1.60
76	LP_7dpi	SCM002766.2_9991617	8	9991617			FarmCPU	-0.070	3.0	
77	LP_4dpi	SCM002766.2_11253257	8	11253257			FarmCPU	0.059	3.2	
78	LP_3dpi	SCM002766.2_17142324	8	17142324			MLMM	-0.154	3.1	2.04
79	LP_4dpi	SCM002766.2_18839525	8	18839525			FarmCPU	0.056	3.0	
80	DW	SCM002766.2_25236628	8	25236628			FarmCPU	-0.235	3.2	
81	LP_3dpi	SCM002767.2_6778855	9	6778855			MLMM	-0.097	3.0	1.93
82	DW	SCM002767.2_6830741	9	6830741			MLMM	-0.182	3.0	2.10
	LP_4dpi	SCM002767.2_6830741	9	6830741	GEMMA-MLM	4.0	MLMM, FarmCPU, mrMLM	0.084, 0.106	3.8-5.4	2.35-2.7

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
83	DW	SCM002767.2_12982760	9	12982760			FarmCPU, mrMLM	-0.179,0.173	4.2-6.1	1.5-2.34
	LP_3dpi	SCM002767.2_12982760	9	12982760			MLMM, mrMLM	-0.091	3.2-5.7	1.96-2.3
	LP_4dpi	SCM002767.2_12982760	9	12982760			MLMM, FarmCPU, mrMLM	-0.110, 0.104	3.0-6.3	2.0-3.54
84	DW	SCM002767.2_21520686	9	21520686	GEMMA-MLM	3.7	MLMM	0.188	3.6	2.60
	LP_4dpi	SCM002767.2_21520686	9	21520686			MLMM, FarmCPU, mrMLM	-0.071, 0.064	3.0-4.2	1.47-2.07
	LP_7dpi	SCM002767.2_21520686	9	21520686	GEMMA-MLM	3.4	MLMM	-0.035	3.3	2.80
85	DW	SCM002767.2_21528172	9	21528172			MLMM	0.268	3.0	2.10
	LP_4dpi	SCM002767.2_21528172	9	21528172			FarmCPU	-0.109	3.5	
86	DW	SCM002767.2_27713481	9	27713481			mrMLM	0.214	3.6	2.20
	LP_4dpi	SCM002767.2_27713481	9	27713481			mrMLM	-0.086	3.6	1.35
87	DW	SCM002767.2_34295849	9	34295849	GEMMA-MLM	4.2	MLMM	0.340	4.0	2.90
	LP_4dpi	SCM002767.2_34295849	9	34295849	GEMMA-MLM	3.8	MLMM	-0.167	3.7	2.64
88	DW	SCM002767.2_34792904	9	34792904			MLMM	-0.540	3.1	2.20
	LP_7dpi	SCM002767.2_34792904	9	34792904			MLMM	0.111	3.3	2.87
89	DW	SCM002767.2_36616904	9	36616904	GEMMA-MLM	3.4	MLMM	-0.375	3.3	2.30
	LP_7dpi	SCM002767.2_36616904	9	36616904	GEMMA-MLM	3.7	MLMM	0.077	3.5	3.07
90	LP_3dpi	SCM002767.2_36951909	9	36951909			MLMM, FarmCPU	0.077, 0.101	3.0-3.5	1.94
	LP_4dpi	SCM002767.2_36951909	9	36951909			FarmCPU	0.068	3.0	
91	DW	SCM002767.2_37453827	9	37453827			FarmCPU	-0.122	4.2	NA

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
92	DW	SCM002768.2_3376886	10	3376886			FarmCPU	-0.211	3.3	
93	DW	SCM002768.2_14496342	10	14496342			mrMLM	-0.144	3.8	2.69
94	LP_3dpi	SCM002768.2_15292534	10	15292534			FarmCPU	0.076	3.1	
95	LP_3dpi	SCM002768.2_15438080	10	15438080			mrMLM	0.074	5.2	2.16
	LP_4dpi	SCM002768.2_15438080	10	15438080			mrMLM	0.063	4.7	1.94
96	DW	SCM002768.2_16001293	10	16001293			FarmCPU	-0.122	4.1	
97	LP_4dpi	SCM002769.2_15972249	11	15972249			MLMM	0.102	3.1	2.11
98	LP_3dpi	SCM002769.2_16506702	11	16506702			MLMM, FarmCPU, mrMLM	-0.129, 0.104	3.0-6.7	2.0-2.7
99	LP_3dpi	SCM002769.2_24927831	11	24927831	GEMMA-MLM	3.6	MLMM, FarmCPU	0.181, 0.231	3.4-3.8	2.30
100	DW	SCM002769.2_30433458	11	30433458			FarmCPU	-0.314	3.0	
101	LP_3dpi	SCM002769.2_43323649	11	43323649	GEMMA-MLM	3.8	MLMM	0.193	3.6	2.45
102	LP_4dpi	SCM002770.2_959722	12	959722			MLMM	-0.208	3.1	2.13
103	LP_4dpi	SCM002770.2_4598582	12	4598582			mrMLM	0.113	3.2	6.15
104	LP_3dpi	SCM002770.2_6162074	12	6162074	GEMMA-MLM	3.7	MLMM, FarmCPU, mrMLM	0.088, 0.103	3.6-7.0	2.4-4.21
	LP_4dpi	SCM002770.2_6162074	12	6162074	GEMMA-MLM	3.9	MLMM, FarmCPU	0.058, 0.089	3.7-4.4	2.70
105	LP_3dpi	SCM002770.2_10543365	12	10543365	GEMMA-MLM	3.7	MLMM	0.241	3.5	2.37
106	LP_3dpi	SCM002770.2_12757488	12	12757488			MLMM	0.096	3.0	1.93
107	LP_7dpi	SCM002770.2_15588436	12	15588436			MLMM	-0.035	3.1	2.64
108	LP_3dpi	SCM002770.2_19302314	12	19302314			FarmCPU	0.056	3.0	
	LP_3dpi	SCM002770.2_19391550	12	19391550			FarmCPU	0.067	3.3	
109	DW	SCM002770.2_19427637	12	19427637			mrMLM	0.158	4.0	3.23
	LP_3dpi	SCM002770.2_19427637	12	19427637			FarmCPU	-0.082	4.6	
110	LP_3dpi	SCM002770.2_19481389	12	19481389			FarmCPU	-0.066	3.4	

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
111	LP_3dpi	SCM002770.2_24237417	12	24237417			FarmCPU	0.070	3.4	
112	LP_3dpi	SCM002770.2_25745655	12	25745655			FarmCPU	-0.099	3.9	
	LP_4dpi	SCM002770.2_25782525	12	25782525			FarmCPU	0.062	3.2	
113	DW	SCM002770.2_25886609	12	25886609			FarmCPU	-0.139	5.2	
	LP_4dpi	SCM002770.2_25886609	12	25886609			FarmCPU, mrMLM	-0.073, 0.071	3.3-3.8	2.51
114	LP_3dpi	SCM002770.2_25911411	12	25911411			FarmCPU	-0.067	3.2	
115	LP_4dpi	SCM002770.2_25988684	12	25988684			FarmCPU	-0.064	3.5	
116	LP_3dpi	SCM002770.2_26027936	12	26027936			FarmCPU	0.070	3.5	
117	LP_4dpi	SCM002770.2_35869952	12	35869952			FarmCPU	-0.132	3.0	
118	LP_7dpi	SCM002770.2_39644852	12	39644852			mrMLM	0.051	4.9	9.52
119	LP_7dpi	SCM002770.2_40023010	12	40023010			FarmCPU	0.050	3.7	NA
120	LP_7dpi	SCM002770.2_46013498	12	46013498			FarmCPU	-0.088	3.3	
121	DW	SCM002770.2_54978115	12	54978115			FarmCPU	0.094	3.1	
122	DW	SCM002770.2_55268968	12	55268968			FarmCPU	-0.104	3.0	
123	LP_7dpi	SCM002770.2_59170961	12	59170961			FarmCPU	-0.104	3.4	
124	DW	SCM002770.2_60099459	12	60099459	GEMMA-MLM	4.6	MLMM, FarmCPU	0.547, 0.625	4.3-7.6	3.20
	LP_7dpi	SCM002770.2_60099459	12	60099459	GEMMA-MLM	3.3	MLMM, FarmCPU	-0.121, -0.101	3.1-3.5	2.60
125	LP_4dpi	SCM002770.2_61095811	12	61095811			FarmCPU, mrMLM	-0.061, 0.064	3.8-4.8	1.83
126	DW	SCM002770.2_61783724	12	61783724			MLMM	0.352	3.4	2.40
127	LP_3dpi	SCM002771.2_7949135	13	7949135			FarmCPU	-0.115	3.1	
128	DW	SCM002771.2_9976695	13	9976695			MLMM	0.191	3.1	2.17
129	LP_7dpi	SCM002771.2_12476994	13	12476994	GEMMA-MLM	4.2	MLMM	0.088	4.0	3.55
130	DW	SCM002771.2_12892646	13	12892646			FarmCPU	0.208	3.3	
131	LP_3dpi	SCM002771.2_13382147	13	13382147			MLMM	-0.134	3.2	2.08

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
132	DW	SCM002771.2_32935711	13	32935711			FarmCPU	0.187	3.0	
	LP_4dpi	SCM002771.2_32935711	13	32935711			MLMM	-0.152	3.4	2.35
133	DW	SCM002771.2_41249711	13	41249711			FarmCPU	0.174	3.3	
134	LP_7dpi	SCM002771.2_46004351	13	46004351			FarmCPU	-0.138	3.2	
135	LP_3dpi	SCM002771.2_54279046	13	54279046			MLMM	-0.229	3.3	2.17
136	LP_3dpi	SCM002771.2_55583051	13	55583051			MLMM	0.147	3.0	1.96
137	DW	SCM002771.2_62907726	13	62907726			MLMM	0.227	3.2	2.21
138	LP_3dpi	SCM002771.2_64722553	13	64722553			FarmCPU	-0.100	5.9	
139	LP_4dpi	SCM002771.2_68049798	13	68049798	GEMMA-MLM	3.8	MLMM	0.107	3.6	2.55
140	DW	SCM002771.2_69685367	13	69685367			MLMM, FarmCPU	0.151, 0.218	3.1-4.9	2.10
141	LP_3dpi	SCM002771.2_69898422	13	69898422			MLMM	-0.124	3.1	2.06
142	DW	SCM002771.2_77997199	13	77997199	GEMMA-MLM	4.3	MLMM, FarmCPU	0.268, 0.337	4.0-6.0	3.00
	LP_3dpi	SCM002771.2_77997199		77997199	GEMMA-MLM	4.2	MLMM, FarmCPU	-0.189, -0.163	4.0-6.4	2.75-5.78
	LP_4dpi	SCM002771.2_77997199	13	77997199	GEMMA-MLM	5.4	MLMM, FarmCPU, mrMLM	-0.199, -0.187	5.1-9.7	3.8-13.3
143	LP_7dpi	SCM002772.2_396291	14	396291			MLMM, FarmCPU	0.062, 0.065	3.0-3.3	2.80
144	DW	SCM002772.2_2193400	14	2193400			MLMM	-0.338	4.9	2.20
145	LP_3dpi	SCM002772.2_7462457	14	7462457			FarmCPU	0.091	3.1	
146	LP_3dpi	SCM002772.2_10363577	14	10363577			MLMM	-0.180	3.0	1.98
147	LP_7dpi	SCM002772.2_12876714	14	12876714			MLMM	0.064	3.0	2.51
148	LP_3dpi	SCM002772.2_16918673	14	16918673			MLMM, mrMLM	-0.203, 0.180	3.2-4.1	2.3-8.15

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
149	LP_7dpi	SCM002772.2_17256004	14	17256004			FarmCPU	-0.089	3.2	
150	LP_7dpi	SCM002772.2_18471902	14	18471902			FarmCPU	-0.092	3.4	
151	LP_3dpi	SCM002772.2_18961270	14	18961270	GEMMA-MLM	4.2	MLMM, FarmCPU, mrMLM	-0.169, -0.184	4.0-9.7	2.75-11.9
	LP_4dpi	SCM002772.2_18961270	14	18961270			mrMLM	-0.088	4.9	3.62
152	LP_3dpi	SCM002772.2_27563465	14	27563465			FarmCPU	0.131	7.0	
153	DW	SCM002772.2_28114395	14	28114395			FarmCPU	0.201	3.1	
154	DW	SCM002772.2_31266664	14	31266664			MLMM, FarmCPU	-0.295, -0.241	3.2-9.8	2.20
	LP_4dpi	SCM002772.2_31266664	14	31266664			FarmCPU, mrMLM	0.082, 0.122	4.9-12.3	4.28
155	DW	SCM002772.2_32008911	14	32008911			FarmCPU	0.146	3.2	
	LP_7dpi	SCM002772.2_32008911	14	32008911			MLMM, mrMLM	-0.040, 0.037	3.1-3.3	2.62-5.68
156	LP_3dpi	SCM002772.2_46691195	14	46691195			FarmCPU, mrMLM	-0.131, -0.083	3.4-6.0	5.50
157	LP_7dpi	SCM002772.2_50481857	14	50481857			FarmCPU	0.108	3.1	
158	DW	SCM002772.2_57995332	14	57995332			MLMM	0.164	3.0	2.10
159	DW	SCM002772.2_58049994	14	58049994			mrMLM	0.264	5.9	5.77
160	DW	SCM002773.2_610542	15	610542			FarmCPU	0.151	5.6	
161	DW	SCM002773.2_2102213	15	2102213			MLMM	0.293	3.3	2.30
162	DW	SCM002773.2_3543525	15	3543525			FarmCPU	0.140	3.2	
163	LP_3dpi	SCM002773.2_6001138	15	6001138	GEMMA-MLM	3.8	MLMM, FarmCPU, mrMLM	-0.178, 0.165	3.7-4.3	2.51-8.56
164	LP_4dpi	SCM002773.2_8136668	15	8136668			FarmCPU	-0.123	3.7	
165	LP_3dpi	SCM002773.2_10198222	15	10198222			MLMM	-0.289	3.0	1.98
166	LP_3dpi	SCM002773.2_12105076	15	12105076			mrMLM	-0.076	3.9	1.61

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
167	LP_3dpi	SCM002773.2_12868611	15	12868611			MLMM	-0.207	3.2	2.12
168	LP_3dpi	SCM002773.2_16562786	15	16562786			MLMM	0.097	3.0	1.98
169	LP_4dpi	SCM002773.2_19013399	15	19013399			FarmCPU	-0.159	3.0	
170	LP_3dpi	SCM002773.2_31319827	15	31319827			MLMM, FarmCPU	0.172, 0.205	3.0-4.3	2.00
171	DW	SCM002773.2_42992177	15	42992177			FarmCPU	-0.164	4.2	
172	DW	SCM002774.2_5170460	16	5170460			FarmCPU	0.102	3.0	
173	DW	SCM002774.2_10918047	16	10918047	GEMMA-MLM	3.3	MLMM, FarmCPU	0.110, 0.173	3.2-3.8	2.20
	LP_4dpi	SCM002774.2_10918047	16	10918047	GEMMA-MLM	3.5	MLMM, FarmCPU	-0.091, -0.058	3.4-3.5	2.40
174	LP_4dpi	SCM002774.2_11948447	16	11948447			FarmCPU	-0.059	4.7	
175	LP_3dpi	SCM002774.2_13414096	16	13414096			FarmCPU	-0.168	3.3	
176	DW	SCM002774.2_14241506	16	14241506			FarmCPU	0.339	6.9	
177	LP_3dpi	SCM002774.2_15647651	16	15647651			MLMM	-0.128	3.0	1.98
	LP_4dpi	SCM002774.2_15647651	16	15647651	GEMMA-MLM	3.6	MLMM	-0.127	3.5	2.44
178	LP_3dpi	SCM002774.2_20726433	16	20726433			FarmCPU	-0.106	5.2	
179	LP_4dpi	SCM002774.2_26766858	16	26766858			FarmCPU	0.086	3.1	
180	DW	SCM002774.2_28747745	16	28747745			FarmCPU	0.183	3.3	
181	LP_4dpi	SCM002774.2_31650615	16	31650615			MLMM	-0.108	3.1	2.10
182	LP_3dpi	SCM002774.2_32688891	16	32688891			FarmCPU	-0.069	3.1	
183	DW	SCM002774.2_39728849	16	39728849			MLMM	0.454	3.0	2.10
184	LP_3dpi	SCM002775.2_8016185	17	8016185			MLMM	-0.112	3.0	1.98
185	DW	SCM002775.2_13344420	17	13344420			FarmCPU	-0.291	3.0	
	LP_3dpi	SCM002775.2_13344420	17	13344420			MLMM	0.267	3.3	2.15
186	LP_3dpi	SCM002775.2_27882458	17	27882458			FarmCPU	-0.093	3.2	
187	LP_4dpi	SCM002775.2_40154705	17	40154705			MLMM, mrMLM	0.065, 0.088	3.3-3.5	1.93-2.32

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
188	LP_7dpi	SCM002775.2_40831558	17	40831558			FarmCPU	0.041	3.1	
189	LP_3dpi	SCM002775.2_42751463	17	42751463			MLMM	-0.116	3.0	1.98
190	LP_4dpi	SCM002775.2_44433020	17	44433020			FarmCPU	0.106	3.0	
191	LP_7dpi	SCM002775.2_50645830	17	50645830			MLMM, FarmCPU, mrMLM	-0.057, 0.046	3.3-4.0	2.8-4.5
192	LP_7dpi	SCM002775.2_55718256	17	55718256			FarmCPU	0.067	3.0	
193	LP_3dpi	SCM002776.2_1006878	18	1006878			FarmCPU	0.132	3.4	
194	LP_3dpi	SCM002776.2_2935480	18	2935480			FarmCPU	0.082	4.7	
195	DW	SCM002776.2_3485443	18	3485443	GEMMA-MLM	4.1	MLMM	0.227	3.8	2.73
	LP_4dpi	SCM002776.2_3485443	18	3485443	GEMMA-MLM	4.2	MLMM	-0.120	4.0	2.89
196	DW	SCM002776.2_3494019	18	3494019	GEMMA-MLM	4.1	MLMM, FarmCPU	-0.228, -0.150	3.6-4.3	2.55
	LP_4dpi	SCM002776.2_3494019	18	3494019	GEMMA-MLM	3.7	MLMM, FarmCPU	0.080, 0.112	3.4-4.2	2.40
	LP_7dpi	SCM002776.2_3494019	18	3494019			FarmCPU	0.043	3.0	
197	LP_4dpi	SCM002776.2_5249316	18	5249316			FarmCPU	0.102	3.0	
	DW	SCM002776.2_5358231	18	5358231			FarmCPU	0.311	3.2	
198	DW	SCM002776.2_11945389	18	11945389			MLMM	-0.360	3.0	2.06
	LP_7dpi	SCM002776.2_11945389	18	11945389	GEMMA-MLM	3.6	MLMM, FarmCPU	0.077, 0.085	3.3-3.4	2.90
199	LP_7dpi	SCM002776.2_11998032	18	11998032			MLMM	-0.095	3.2	2.71
200	DW	SCM002776.2_12692146	18	12692146	GEMMA-MLM	3.8	MLMM	0.277	3.7	2.60
	LP_7dpi	SCM002776.2_12692146	18	12692146	GEMMA-MLM	4.0	MLMM, FarmCPU	-0.057, -0.049	3.0-3.9	3.40
201	LP_3dpi	SCM002776.2_27101496	18	27101496			MLMM	0.270	3.0	1.96
202	DW	SCM002776.2_27672332	18	27672332	GEMMA-MLM	3.3	MLMM	-0.316	3.2	2.30
	LP_3dpi	SCM002776.2_27672332	18	27672332			MLMM	0.171	3.1	2.02

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

					Single-locus GWAS		Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (P)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
202	DW	SCM002776.2_27672332	18	27672332	GEMMA-MLM	3.3	MLMM	-0.316	3.2	2.30
	LP_3dpi	SCM002776.2_27672332	18	27672332			MLMM	0.171	3.1	2.02
	LP_4dpi	SCM002776.2_27672332	18	27672332	GEMMA-MLM	4.3	MLMM, FarmCPU, mrMLM	0.152, 0.185	4.0-5.1	2.97-8.66
203	LP_3dpi	SCM002776.2_29554663	18	29554663			FarmCPU	0.138	4.2	
204	DW	SCM002776.2_34159337	18	34159337			FarmCPU	0.123	3.0	
205	DW	SCM002776.2_42850291	18	42850291			FarmCPU	0.137	3.0	
206	LP_3dpi	SCM002777.2_2395391	19	2395391			FarmCPU	0.092	3.1	
207	DW	SCM002777.2_13355921	19	13355921			MLMM	0.247	3.2	2.30
	LP_3dpi	SCM002777.2_13355921	19	13355921			mrMLM	-0.107	3.1	4.35
208	LP_4dpi	SCM002777.2_24890194	19	24890194			FarmCPU	0.082	3.6	
	LP_7dpi	SCM002777.2_24890194	19	24890194			mrMLM	-0.037	4.2	4.69
209	LP_7dpi	SCM002777.2_29273334	19	29273334			FarmCPU	-0.094	3.1	
210	LP_4dpi	SCM002777.2_29633297	19	29633297			FarmCPU	-0.107	3.1	
211	LP_7dpi	SCM002777.2_34107430	19	34107430			FarmCPU	-0.073	3.4	
212	DW	SCM002777.2_36581746	19	36581746	GEMMA-MLM	3.7	MLMM, FarmCPU	-0.330, -0.522	3.4-4.1	2.40
	LP_7dpi	SCM002777.2_36581746	19	36581746	GEMMA-MLM	4.9	MLMM, FarmCPU	0.116, 0.122	4.0-4.5	4.00
213	DW	SCM002777.2_42617889	19	42617889			FarmCPU	0.223	3.0	
214	DW	SCM002777.2_47487736	19	47487736			FarmCPU	0.244	3.7	

Table A.9. List of significant single-nucleotide polymorphisms (SNPs) associated with days to wilt, lesion phenotypes at 3, 4, and 7 days post inoculation traits for sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

				Single-locus GWAS			Multi-locus GWAS			
Locus	Traits[a]	SNP_ID	Chr[b]	Position (bp)	Method	-log$_{10}$ (*P*)[d]	Methods[e]	QTN effect[f]	-log$_{10}$ (*P*)[g]	R^2 *(%)*[h]
215	LP_4dpi	SCM002777.2_50176743	19	50176743			MLMM, FarmCPU, mrMLM	-0.154, 0.166	3.1-6.1	2.1-7.8
216	DW	SCM002777.2_51133509	19	51133509			MLMM, FarmCPU	-0.218, -0.139	3.2-4.1	2.20

Traits[a]: Days to wilt (DW); LP_3dpi, lesion phenotype scores at 3 days post inoculation; LP_4dpi, lesion phenotype scores at 4 days post inoculation; LP_7dpi, lesion phenotype scores at 7 days post inoculation
Chr[b]: *Brassica napus* chromosome
-log$_{10}$ (*P*)[d], The -log10 (*P*) value resulted from the GEMMA-MLM GWA model
Methods[e]: Three multi-locus *i.e.,* MLMM, FarmCPU, and mrMLM GWA models
QTN effect[f]: difference in mean phenotypic values between genotypes with major allele and minor allele. Positive sign indicates major allele is associated with increased phenotypic values. Negative sign indicates that the major allele is associated with reduced
-log$_{10}$ (*P*)[g], The highest and lowest -log10 (*P*) value resulted from the various studied traits with different multi-locus GWA models
R^2 (%) [h]; The range of the percentage of phenotypic variation explained by the identified significant SNP derived from the results of MLMM, and mrMLM GWA models

Table A.10. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected by at least two GWAS models or at least two traits associated with sclerotinia stem rot resistance by single-locus and multi-locus GWA studies

				Single-locus GWAS		Multi-locus GWAS		
Locus	SNP_ID	Chrm[a]	Position (bp)	Method and Traits[c]	-$\log_{10}$ (P)[d]	Methods and Traits[f]	-$\log_{10}$ (P)[g]	R^2 (%)[h]
1	SCM002759.2_7316181	1	7316181	GEMMA-MLM[14]	3.6	MLMM[14], FarmCPU[4]	3.3-3.5	2.4-3.0
2	SCM002759.2_31473630	1	31473630			MLMM[4], FarmCPU[4]	3.0-3.3	2.5
3	SCM002760.2_5283289	2	5283289			FarmCPU[4], mrMLM[1]	3.0-7.0	4.4
4	SCM002760.2_20844443	2	20844443			MLMM[3], mrMLM[3]	3.1-3.3	2.3-4.3
5	SCM002760.2_30564102	2	30564102			MLMM[3], mrMLM[1]	3.0-4.7	2.1-5.0
6	SCM002761.2_5046706	3	5046706			MLMM[2], mrMLM[2]	3.2-3.5	2.1-2.4
7	SCM002761.2_5607745	3	5607745	GEMMA-MLM[13]	3.5-3.7	MLMM[13], FarmCPU[3], mrMLM[3]	3.4-5.7	2.4-2.7
8	SCM002761.2_20918043	3	20918043			MLMM[123], FarmCPU[2], mrMLM[23]	3.0-7.9	2.0-3.07
	SCM002761.2_20921893	3	20921893			MLMM[1], FarmCPU[1]	3.0-4.1	2.0
9	SCM002761.2_23664593	3	23664593	GEMMA-MLM[4]	4.2	MLMM[4], FarmCPU[4]	3.3-4.0	3.6
10	SCM002761.2_45757845	3	45757845			MLMM[3], mrMLM[1]	3.1-4.0	2.1-9.3
11	SCM002762.2_10716343	4	10716343	GEMMA-MLM[4]	4.3	MLMM[4], FarmCPU[4], mrMLM[4]	3.6.-4.8	3.5-6.8
12	SCM002762.2_13620028	4	13620028	GEMMA-MLM[23]	3.4	MLMM[23], FarmCPU[3], mrMLM[3]	3.2-5.3	1.5-2.8
13	SCM002762.2_19685094	4	19685094	GEMMA-MLM[4]	3.8	MLMM[4], FarmCPU[4]	3.50	3.0
14	SCM002763.2_746381	5	746381	GEMMA-MLM[4]	4.0	MLMM[4], FarmCPU[4]	3.6-3.8	3.3
15	SCM002763.2_1914189	5	1914189	GEMMA-MLM[1]	3.3	MLMM[14], FarmCPU[4], mrMLM[4]	3.1-5.4	2.2-5.3
16	SCM002763.2_2070843	5	2070843			MLMM[4], mrMLM[4]	3.0-3.4	2.5-2.6
17	SCM002763.2_28498462	5	28498462	GEMMA-MLM[2]	3.8	MLMM[23]	3.0-3.6	2.1-2.5
18	SCM002764.2_3011988	6	3011988	GEMMA-MLM[4]	4.2	MLMM[4], FarmCPU[4]	3.7-4.0	3.5
19	SCM002764.2_7193358	6	7193358	GEMMA-MLM[23]	3.5-3.7	MLMM[23], FarmCPU[2]	3.1-3.6	2.2-2.5
20	SCM002764.2_7381974	6	7381974			MLMM[23]	3.0-3.3	2.1-2.2
21	SCM002764.2_7471454	6	7471454	GEMMA-MLM[23]	4.0-4.4	MLMM[123], FarmCPU[123], mrMLM[123]	3.0-8.7	2.2-4.7
22	SCM002765.2_26141436	7	26141436			FarmCPU[13]	3.2-3.6	NA
23	SCM002766.2_8308223	8	8308223	GEMMA-MLM[1]	3.7	MLMM[14], FarmCPU[1]	3.0-4.7	2.50
24	SCM002767.2_6830741	9	6830741	GEMMA-MLM[3]	4.0	MLMM[13], FarmCPU[3], mrMLM[3]	3.0-5.4	2.0-2.7

Table A.10. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected by at least two GWAS models or at least two traits associated with sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

				Single-locus GWAS		Multi-locus GWAS		
Locus	SNP_ID	Chrm[a]	Position (bp)	Method and Traits[c]	$-\log_{10}(P)$[d]	Methods and Traits[f]	$-\log_{10}(P)$[g]	R^2 (%)[h]
25	SCM002767.2_12982760	9	12982760			FarmCPU[13], mrMLM[123], MLMM[23]	3.0-6.3	1.5-3.5
26	SCM002767.2_21520686	9	21520686	GEMMA-MLM[14]	3.4-3.7	MLMM[134], FarmCPU[3], mrMLM[3]	3.0-4.2	1.5-2.8
27	SCM002767.2_21528172	9	21528172			FarmCPU[3], MLMM[1]	3.0-3.5	2.10
28	SCM002767.2_27713481	9	27713481			mrMLM[13]	3.60	1.4-2.2
29	SCM002767.2_34295849	9	34295849	GEMMA-MLM[13]	3.8-4.2	MLMM[13]	3.7-4.0	2.6-2.9
30	SCM002767.2_34792904	9	34792904			MLMM[14]	3.1-3.4	2.2-2.9
31	SCM002767.2_36616904	9	36616904	GEMMA-MLM[14]	3.4-3.7	MLMM[14]	3.3-3.5	2.3-3.1
32	SCM002767.2_36951909	9	36951909			MLMM[2], FarmCPU[23]	3.0-3.5	1.9
33	SCM002768.2_15438080	10	15438080			mrMLM[23]	4.7-5.2	1.9-2.2
34	SCM002769.2_16506702	11	16506702			MLMM[2], FarmCPU[2], mrMLM[2]	3.0-6.7	2.0-2.7
35	SCM002769.2_24927831	11	24927831	GEMMA-MLM[2]	3.6	MLMM[2], FarmCPU[2]	3.4-3.8	2.30
36	SCM002769.2_43323649	11	43323649	GEMMA-MLM[2]	3.8	MLMM[2]	3.6	2.45
37	SCM002770.2_6162074	12	6162074	GEMMA-MLM[23]	3.7-3.9	MLMM[23], FarmCPU[23], mrMLM[2]	3.6-7.0	2.4-4.21
38	SCM002770.2_10543365	12	10543365	GEMMA-MLM[2]	3.7	MLMM[2]	3.50	2.37
39	SCM002770.2_19427637	12	19427637			FarmCPU[2], mrMLM[1]	4.0-4.6	3.23
40	SCM002770.2_25886609	12	25886609			FarmCPU[13], mrMLM[3]	3.3-5.2	2.51
41	SCM002770.2_60099459	12	60099459	GEMMA-MLM[14]	3.3-4.6	MLMM[14], FarmCPU[14]	3.1-7.6	2.6-3.2
42	SCM002770.2_61095811	12	61095811			FarmCPU[3], mrMLM[3]	3.8-4.8	1.83
43	SCM002771.2_12476994	13	12476994	GEMMA-MLM[4]	4.2	MLMM[4]	4.01	3.55
44	SCM002771.2_32935711	13	32935711			MLMM[3], FarmCPU[1]	3.0-3.4	2.35

Table A.10. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected by at least two GWAS models or at least two traits associated with sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

				Single-locus GWAS		Multi-locus GWAS		
Locus	SNP_ID	Chrm[a]	Position (bp)	Method and Traits[c]	-log$_{10}$ (P)[d]	Methods and Traits[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
45	SCM002771.2_68049798	13	68049798	GEMMA-MLM[3]	3.8	MLMM[3]	3.6	2.55
46	SCM002771.2_69685367	13	69685367			MLMM[1], FarmCPU[1]	3.1-4.9	2.10
47	SCM002771.2_77997199	13	77997199	GEMMA-MLM[123]	4.2-5.4	MLMM[123], FarmCPU[123], mrMLM[3]	4.0-9.7	2.8-13.3
48	SCM002772.2_396291	14	396291			MLMM[4], FarmCPU[4]	3.0-3.3	2.8
49	SCM002772.2_16918673	14	16918673			MLMM[2], mrMLM[2]	3.2-4.1	2.3-8.2
50	SCM002772.2_18961270	14	18961270	GEMMA-MLM[2]	4.2	MLMM[2], FarmCPU[2], mrMLM[23]	4.0-9.7	2.8-11.9
51	SCM002772.2_31266664	14	31266664			FarmCPU[13], mrMLM[3], MLMM[1]	3.2-12.3	2.2-4.3
52	SCM002772.2_32008911	14	32008911			MLMM[4], mrMLM[4], FarmCPU[1]	3.1-3.3	2.6-5.7
53	SCM002772.2_46691195	14	46691195			FarmCPU[2], mrMLM[2]	3.4-6.0	5.50
54	SCM002773.2_6001138	15	6001138	GEMMA-MLM[2]	3.8	MLMM[2], FarmCPU[2], mrMLM[2]	3.7-4.3	2.5-8.6
55	SCM002773.2_31319827	15	31319827			MLMM[2], FarmCPU[2]	3.0-4.3	2.00
56	SCM002774.2_10918047	16	10918047	GEMMA-MLM[13]	3.3-3.5	MLMM[13], FarmCPU[13]	3.2-3.8	2.2-2.4
57	SCM002774.2_15647651	16	15647651	GEMMA-MLM[3]		MLMM[23]	3.0-3.5	2.0-2.4
58	SCM002775.2_13344420	17	13344420			MLMM[2], FarmCPU[1]	3.0-3.3	2.15
59	SCM002775.2_40154705	17	40154705			MLMM[2], mrMLM[2]	3.3-5.0	1.9-2.3
60	SCM002775.2_50645830	17	50645830			MLMM[4], FarmCPU[4], mrMLM[4]	3.3-4.0	2.8-4.5
61	SCM002776.2_3485443	18	3485443	GEMMA-MLM[13]	4.1-4.2	MLMM[13]	3.8-4.0	2.7-2.9
62	SCM002776.2_3494019	18	3494019	GEMMA-MLM[13]	3.7-41	MLMM[13], FarmCPU[134]	3.0-4.2	2.4-2.6
63	SCM002776.2_11945389	18	11945389	GEMMA-MLM[4]	3.6	MLMM[14], FarmCPU[4]	3.0-3.4	2.1-2.9
64	SCM002776.2_12692146	18	12692146	GEMMA-MLM[14]	3.8-4.0	MLMM[14], FarmCPU[4]	3.0-3.9	2.6-3.4
65	SCM002776.2_27672332	18	27672332	GEMMA-MLM[13]	3.3-4.3	MLMM[123], FarmCPU[3], mrMLM[3]	3.1-5.1	2.0-8.7

Table A.10. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected by at least two GWAS models or at least two traits associated with sclerotinia stem rot resistance by single-locus and multi-locus GWA studies (continued)

				Single-locus GWAS		Multi-locus GWAS		
Locus	SNP_ID	Chrm[a]	Position (bp)	Method and Traits[c]	-log$_{10}$ (P)[d]	Methods and Traits[f]	-log$_{10}$ (P)[g]	R^2 (%)[h]
66	SCM002777.2_13355921	19	13355921			mrMLM[2], MLMM[1]	3.1-3.2	2.3-4.4
67	SCM002777.2_24890194	19	24890194			FarmCPU[3], mrMLM[4]	3.6-4.2	4.69
68	SCM002777.2_36581746	19	36581746	GEMMA-MLM[14]	3.7-4.9	MLMM[14], FarmCPU[14]	3.4-4.5	2.4-4.0
69	SCM002777.2_50176743	19	50176743			MLMM[3], FarmCPU[3], mrMLM[3]	3.1-6.1	2.1-7.8
70	SCM002777.2_51133509	19	51133509			MLMM[1], FarmCPU[1]	3.2-4.1	2.20

Chrm[a]: *Brassica napus* chromosome

Method and Traits[c]: Method, sinlge-locus GEMMA-MLM model; Traits, Days to wilt (DW); LP_3dpi, lesion phenotype scores at 3 days post inoculation (dpi); LP_4dpi, lesion phenotype scores at 4 dpi; LP_7dpi, lesion phenotype scores at 7 dpi

-log10 (P)[d], The highest and lowest -log10 (P) values resulted from different traits obtained by the GEMMA-MLM GWA model

Methods and Traits[f]: Methods, multi-locus GWA models i.e. MLMM, FarmCPU, mrMLM; Traits, Days to wilt (DW); LP_3dpi, lesion phenotype scores at 3 days post inoculation (dpi); LP_4dpi, lesion phenotype scores at 4dpi; LP_7dpi, lesion phenotype scores at 7 dpi

-log10 (P)[g], The highest and lowest -log10 (P) value resulted from the various studied traits with different multi-locus GWA models

R^2 (%)[h]; Percentage of phenotypic variation explained by identified significant SNP resulted from MLMM, and mrMLM GWA models

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002759.2_7316181	7316181	1	LOC106360741	-57.42	receptor-like cytosolic serine/threonine-protein kinase RBK1	defense response to fungus Source: UniProtKB; defense response to other organism Source: TAIR
SCM002759.2_31473630	31473630	1	LOC106394796	-2.25	ethylene-responsive transcription factor ERF071-like	ethylene-activated signaling pathway Source: UniProtKB-KW
SCM002760.2_5283289	5283289	2	LOC106354437	-19.88	NADP-dependent alkenal double bond reductase P2-like	response to oxidative stress Source: TAIR
SCM002760.2_5283289	5283289	2	LOC106355981	44.00	probably inactive leucine-rich repeat receptor-like protein kinase At5g48380	negative regulation of defense response Source: TAIR
SCM002760.2_5283289	5283289	2	LOC106354440	7.41	binding partner of ACD11 1-like	defense response to fungus Source: TAIR; negative regulation of plant-type hypersensitive response Source: TAIR
SCM002760.2_30564102	30564102	2	LOC106403204	45.32	sm-like protein LSM5	response to bacterium Source: Ensembl
SCM002760.2_30564102	30564102	2	LOC106414702	-10.43	universal stress protein PHOS34-like	response to molecule of fungal origin Source: TAIR

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits (continued)

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002761.2_5607745	5607745	2	LOC106452384	1.50	endochitinase CH25-like	chitin catabolic process Source: UniProtKB-KW
SCM002761.2_5607745	5607745	2	LOC106452385	-7.98	cysteine proteinase inhibitor 6	defense response Source: UniProtKB-KW
SCM002761.2_5607745	5607745	2	LOC106345435	-11.03	cysteine proteinase inhibitor 6-like	defense response Source: UniProtKB-KW
SCM002761.2_23664593	23664593	3	LOC106353180	13.49	E3 ubiquitin-protein ligase RDUF2-like	response to chitin Source: TAIR
SCM002761.2_23664593	23664593	3	LOC106353179	12.56	probable serine/threonine-protein kinase PBL7	positive regulation of brassinosteroid mediated signaling pathway Source: TAIR
SCM002761.2_23664593	23664593	3	LOC106353181	1.04	serine/threonine-protein kinase EDR1-like	response to fungus Source: TAIR, regulation of salicylic acid mediated signaling pathway Source: UniProtKB
SCM002761.2_23664593	23664593	3	LOC106353190	-26.80	serine/threonine-protein phosphatase PP2A-4 catalytic subunit-like	defense response Source: UniProtKB-KW

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits (continued)

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002762.2_10716343	10716343	4	LOC106448335	21.39	senescence-specific cysteine protease SAG12-like	defense response to fungus Source: TAIR, programmed cell death involved in cell development
SCM002762.2_19685094	19685094	4	LOC106447416	39.45	phosphatidylinositol:ceramide inositolphosphotransferase 2	defense response Source: TAIR
SCM002762.2_19685094	19685094	4	LOC106447426	-2.33	steroid 5-alpha-reductase DET2-like	brassinosteroid biosynthetic process Source: TAIR
SCM002763.2_1914189	1914189	5	LOC106454482	14.05	transcription factor PAR1-like	brassinosteroid mediated signaling pathway Source: UniProtKB-KW
SCM002763.2_1914189	1914189	5	LOC106377403	-8.62	serine/threonine-protein phosphatase 5-like	cellular response to hydrogen peroxide Source: Ensembl
SCM002763.2_1914189	1914189	5	LOC106377404	-15.30	protein TOO MANY MOUTHS-like	defense response to fungus, regulation of antifungal innate immune response Source: UniProtKB
SCM002763.2_2070843	2070843	5	LOC106421025	3.08	transcription factor IBH1	brassinosteroid mediated signaling pathway Source: UniProtKB-KW
SCM002763.2_2070843	2070843	5	LOC106421020	-23.18	pirin-like protein 2	defense response to bacterium Source: TAIR

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits (continued)

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002764.2_7193358	7193358	6	LOC106346585	2.69	probable LRR receptor-like serine/threonine-protein kinase At1g14390	defense response to nematode Source: TAIR
SCM002764.2_7193358	7193358	6	LOC106351297	-6.56	aldehyde oxidase GLOX-like	defense response to fungus Source: UniProtKB
SCM002766.2_8308223	8308223	8	LOC106360646	52.64	RING-H2 finger protein ATL17	defense response Source: UniProtKB-KW
SCM002766.2_8308223	8308223	8	LOC106359593	46.16	putative pectinesterase/pectinesterase inhibitor 43	cell wall modification Source: InterPro
SCM002767.2_12982760	12982760	9	LOC106433772	-16.27	putative UDP-glucuronate:xylan alpha-glucuronosyltransferase 4	cell wall organization Source: UniProtKB-KW
SCM002767.2_21520686	21520686	9	LOC111201004	33.64	protein ENHANCED DISEASE RESISTANCE 2-like	regulation of defense response to fungus Source: UniProtKB; response to salicylic acid Source: UniProtKB
SCM002767.2_21520686	21520686		LOC106366744	23.88	MOB kinase activator-like 1A	regulation of jasmonic acid biosynthetic process Source: TAIR

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits (continued)

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002767.2_34295849	34295849	9	LOC106368101	22.81	staphylococcal-like nuclease CAN1	May be involved in genomic DNA degradation during programmed cell death.
SCM002767.2_34792904	34792904	9	LOC106363516	74.68	transcription factor MYB56-like	cellular response to brassinosteroid stimulus Source: TAIR
SCM002767.2_34792904	34792904	9	LOC106363514	22.99	lysM domain receptor-like kinase 3	defense response Source: TAIR, defense response to fungus Source: TAIR
SCM002767.2_34792904	34792904	9	LOC106364931	7.07	regulatory protein NPR6	induced systemic resistance, jasmonic acid mediated signaling pathway Source: TAIR
SCM002767.2_36616904	36616904	9	LOC106368478	-31.59	ethylene-responsive transcription factor CRF6	ethylene-activated signaling pathway Source: UniProtKB-KW
SCM002767.2_36616904	36616904	9	LOC106368479	-34.13	tubulin gamma-1 chain	response to nematode Source: UniProtKB
SCM002767.2_36951909	36951909	9	LOC106368528	-24.09	dnaJ protein ERDJ3B	pattern recognition receptor signaling pathway Source: TAIR
SCM002767.2_36951909	36951909	9	LOC111200369	-44.98	dnaJ protein ERDJ3B-like	pattern recognition receptor signaling pathway Source: TAIR

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits (continued)

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002768.2_15438080	15438080	10	LOC106371937	47.76	NADP-dependent alkenal double bond reductase P2-like	response to oxidative stress Source: TAIR
SCM002768.2_15438080	15438080	10	LOC106371944	19.58	binding partner of ACD11 1-like	defense response to fungus Source: TAIR; negative regulation of plant-type hypersensitive response Source: TAIR
SCM002768.2_15438080	15438080	10	LOC106370502	-3.30	transcription factor MYB41-like	response to chitin Source: TAIR
SCM002769.2_24927831	24927831	11	LOC106373937	17.77	protein SUPPRESSOR OF npr1-1, CONSTITUTIVE 1-like	systemic acquired resistance, salicylic acid mediated signaling pathway Source: T
SCM002769.2_24927831	24927831	11	BNAC01G22450D	5.72	senescence/dehydration-associated protein At4g35985, chloroplastic	response to oomycetes Source: UniProtKB
SCM002770.2_6162074	6162074	12	LOC106377774	26.33	la-related protein 1C-like	response to jasmonic acid Source: UniProtKB, response to salicylic acid Source: UniProtKB

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits (continued)

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002770.2_6162074	6162074	12	LOC106379206	16.50	peroxidase 58-like	hydrogen peroxide catabolic process Source: UniProtKB-KW, response to oxidative stress
SCM002770.2_6162074	6162074	12	LOC106379205	12.48	peroxidase N-like	hydrogen peroxide catabolic process Source: UniProtKB-KW, response to oxidative stress
SCM002770.2_10543365	10543365	12	LOC106388390	-5.07	glucan endo-1,3-beta-glucosidase 6-like	defense response Source: UniProtKB-KW
SCM002770.2_10543365	10543365	12	LOC106388425	-26.11	temperature-induced lipocalin-1-like	response to reactive oxygen species Source: GO_Central, positive regulation of response to oxidative stress Source: UniProtKB
SCM002770.2_19427637	19427637	12	LOC106381160	10.67	WAT1-related protein At1g70260	negative regulation of defense response to oomycetes Source: TAIR, regulation of defense response to fungus Source: TAIR
SCM002770.2_25745655	25745655	12	LOC106378095		probable disease resistance protein RPP1	defense response Source: TAIR, defense response to fungus Source: TAIR, response to oomycetes Source: TAIR

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits (continued)

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002770.2_25886609	25886609	12	LOC106379438		TSK-associating protein 1-like	defense response to fungus Source: TAIR
SCM002770.2_25886609	25886609	12	LOC106378104		germin-like protein subfamily 3 member 1	May play a role in plant defense.
SCM002770.2_61095811	61095811	12	LOC106379309	-23.80	ethylene-responsive transcription factor ERF025-like	defense response to fungus Source: TAIR, glucosinolate metabolic process Source: TAIR,
SCM002771.2_12476994	12476994	13	LOC111204360	50.58	heat shock protein 90-2-like	innate immune response Source: UniProtKB-KW
SCM002771.2_12476994	12476994	13	LOC106387440	45.48	heat shock protein 90-2-like	innate immune response Source: UniProtKB-KW
SCM002771.2_32935711	32935711	13	LOC106388602	-3.56	cullin-1	jasmonic acid mediated signaling pathway Source: TAIR, response to jasmonic acid Source: TAIR
SCM002771.2_32935711	32935711	13	LOC106388601	-26.78	NADH dehydrogenase [ubiquinone] flavoprotein 2, mitochondrial	response to oxidative stress

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits (continued)

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002771.2_32935711	32935711	13	LOC106388600	-29.26	transcription factor UNE12-like	regulation of defense response Source: TAIR
SCM002771.2_77997199	77997199	13	LOC106358708	48.73	protein ETHYLENE INSENSITIVE 3-like	defense response to bacterium Source: TAIR
SCM002771.2_77997199	77997199	13	LOC106358707	43.91	AP2/ERF and B3 domain-containing transcription factor At1g51120-like	defense response Source: UniProtKB-KW
SCM002771.2_77997199	77997199	13	LOC106358706	42.23	protein ETHYLENE INSENSITIVE 3-	defense response to bacterium Source: TAIR
SCM002771.2_77997199	77997199	13	LOC106358705	37.25	AP2/ERF and B3 domain-containing transcription factor At1g51120-like	defense response Source: UniProtKB-KW
SCM002772.2_18961270	18961270	14	LOC106378157	0.87	branched-chain-amino-acid aminotransferase 6-like	glucosinolate biosynthetic process from homomethionine Source: TAIR
SCM002772.2_31266664	31266664	14	LOC106394890	-11.18	peroxidase 64-like	response to oxidative stress Source: InterPro, response to pathogen attack and oxidative stress.

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits (continued)

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002773.2_31319827	31319827	15	LOC106408757	-235.19	cysteine protease XCP2-like	defense response to bacterium Source: UniProtKB
SCM002774.2_10918047	10918047	16	LOC106402791	28.55	jacalin-related lectin 15-like	Confers broad resistance to potexviruses.
SCM002774.2_10918047	10918047	16	LOC106402792	26.66	jacalin-related lectin 15-like	Confers broad resistance to potexviruses.
SCM002774.2_10918047	10918047	16	LOC106402793	21.23	jacalin-related lectin 15	Confers broad resistance to potexviruses.
SCM002775.2_13344420	13344420	17	LOC106379304	-28.49	RPM1-interacting protein 4-like	innate immune response-activating signal transduction Source: TAIR
SCM002775.2_40154705	40154705	17	LOC106348985	27.41	probably inactive leucine-rich repeat receptor-like protein kinase At5g48380	negative regulation of defense response Source: TAIR
SCM002775.2_50645830	50645830	17	LOC106421137	-8.11	protein LSD1-like	negative regulation of programmed cell death Source: UniProtKB, Negative regulator pathogen-induced hypersensitive response (HR), basal disease resistance

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits (continued)

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002775.2_50645830	50645830	17	LOC106421159	-32.88	berberine bridge enzyme-like 19	defense response to fungus Source: TAIR
SCM002775.2_50645830	50645830	17	LOC106421160	-40.75	berberine bridge enzyme-like 19	defense response to fungus Source: TAIR
SCM002776.2_12692146	12692146	18	LOC106412847	-29.94	protein NAR1-like	response to oxygen levels Source: TAIR
SCM002776.2_27672332	27672332	18	LOC106411008	-5.17	cellulose synthase A catalytic subunit 4 [UDP-forming]	defense response to fungus Source: TAIR
SCM002777.2_13355921	13355921	19	LOC106391263	10.91	G-type lectin S-receptor-like serine/threonine-protein kinase At1g61500	innate immune response Source: GO_Central
SCM002777.2_50176743	50176743	19	LOC106449880	68.43	probable L-type lectin-domain containing receptor kinase II.1	defense response to oomycetes Source: GO_Central
SCM002777.2_50176743	50176743	19	LOC106446564	203.16	probable serine/threonine-protein kinase BSK3	positive regulation of brassinosteroid mediated signaling pathway Source: UniProtKB
SCM002777.2_50176743	50176743	19	LOC111209304	-85.05	defensin-like protein 22	defense response to fungus

Table A.11. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits (continued)

SNP_Markers	Marker Position	Chr[a]	Gene symbol and ID	Distance (kb)[b]	Gene Description	GO biological Function
SCM002777.2_50176743	50176743	19	LOC106406147	-94.88	porphobilinogen deaminase, chloroplastic	defense response to bacterium Source: TAIR
SCM002777.2_50176743	50176743	19	LOC106387307	-164.79	poly [ADP-ribose] polymerase 1-like	cellular response to oxidative stress Source: MGI
SCM002777.2_51133509	51133509	19	LOC111209972	1.24	putative pentatricopeptide repeat-containing protein At1g56570	defense response to fungus Source: TAIR

Chr[a]: *Brassica napus* chromosome

Distance (kb)[b]: negative values= genes are downstream of the marker, positive values = genes are upstream of the marker

Table A.12. Name, origin, and growth habits of the 146 spring ecotype germplasm accessions (including check cultivars) used in the study

Name of the accessions	Country of origin/obtained	Growth habit
Pioneer 45S51[a]	**Pioneer (Check)**	**Spring**
Pioneer 45S56[a]	**Pioneer (Check)**	**Spring**
Galant	USA	Spring
Galaxy	Sweden	Spring
Gido	Germany	Spring
Gisora	Germany	Spring
Global	Sweden	Spring
Golden	Canada	Spring
Gora	Germany	Spring
Gulle	Sweden	Spring
Gullivar	Sweden	Spring
Hi-Q	Canada	Spring
INRA-R-2000	France	Spring
IR-2	Hungary	Spring
Janetzkis	South Korea	Spring
Jasna	Serbia	Spring
Kanada	Poland	Spring
Klinki	South Korea	Spring
Kosa	Germany	Spring
Koubun	South Korea	Spring
Kraphhauser	South Korea	Spring
Kritmar rape	South Korea	Spring
Legend	Sweden	Spring
Aviso	Canada	Spring
Lifura	South Korea	Spring
Mar'janovskij	Ukraine	Spring
Mazowiecki	Poland	Spring
Midas	Canada	Spring
Miekuro Dane	South Korea	Spring
Mura yamasho	South Korea	Spring
NDC-A14026	USA	Spring
NDC-A14032	USA	Spring

Table A.12. Name, origin, and growth habits of the 146 spring ecotype germplasm accessions (including check cultivars) used in the study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
NDC-A14033	USA	Spring
NDC-A14035	USA	Spring
NDC-A14036	USA	Spring
NDC-A14045	USA	Spring
NDC-A14046	USA	Spring
NDC-A14050	USA	Spring
NDC-A14055	USA	Spring
NDC-A14056	USA	Spring
NDC-E12009	USA	Spring
NDC-E12023	USA	Spring
NDC-E12025	USA	Spring
NDC-E12027	USA	Spring
NDC-E12044	USA	Spring
NDC-E12079	USA	Spring
NDC-E12081	USA	Spring
NDC-E12086	USA	Spring
NDC-E12119	USA	Spring
NDC-E12120	USA	Spring
NDC-E12121	USA	Spring
NDC-E12131	USA	Spring
NDC-E12133	USA	Spring
NDC-E13193	USA	Spring
NDC-E13279	USA	Spring
NDC-E13285	USA	Spring
NDC-E15031	USA	Spring
NDC-E15146	USA	Spring
NDC-E15174	USA	Spring
NDC-E15200	USA	Spring
NDC-E15234	USA	Spring
NDC-E15294	USA	Spring
NDC-E16015	USA	Spring
NDC-E16053	USA	Spring

Table A.12. Name, origin, and growth habits of the 146 spring ecotype germplasm accessions (including check cultivars) used in the study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
NDC-E16152	USA	Spring
NDC-E16169	USA	Spring
NDC-E16198	USA	Spring
NDC-E17132	USA	Spring
NDSU01104	USA	Spring
NDSU0417	USA	Spring
NDSU0472	USA	Spring
NDSU0473	USA	Spring
NDSU0474	USA	Spring
NDSU0475	USA	Spring
NDSU0521	USA	Spring
NDSU0522	USA	Spring
NDSU0619	USA	Spring
NDSU0620	USA	Spring
NDSU0726	USA	Spring
NDSU0728	USA	Spring
NDSU0729	USA	Spring
NDSU10999	USA	Spring
NDSU12989	USA	Spring
NDSU151000	USA	Spring
NDSU15989	USA	Spring
NDSU161013	USA	Spring
NDSU31001	USA	Spring
NDSU31011	USA	Spring
Bingo	USA	Spring
NDSU41000	USA	Spring
NDSU7997	USA	Spring
NDSU81000	USA	Spring
NDSU91013	USA	Spring
NU 41737	Turkey	Spring
NU 51084	Sweden	Spring
BO-63	Canada	Spring

Table A.12. Name, origin, and growth habits of the 146 spring ecotype germplasm accessions (including check cultivars) used in the study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Oro	Canada	Spring
Orpal	France	Spring
Peace	Canada	Spring
Polo canola	USA	Spring
Premier	USA	Spring
Printol	USA	Spring
Prota	Germany	Spring
Q2	Canada	Spring
Ratnik	Serbia	Spring
Regent	Canada	Spring
Regina II	Canada	Spring
Reston	USA	Spring
Rico	Germany	Spring
Romeo	France	Spring
Russia 5	Russian Federation	Spring
S.V. Gulle	South Korea	Spring
Seoul	South Korea	Spring
Silex	Canada	Spring
Brio	France	Spring
Sunrise	USA	Spring
Sval of Gullen	South Korea	Spring
Bronowski	Poland	Spring
Taiwan	Taiwan	Spring
Tanto	France	Spring
Tobin	USA	Spring
Buk Wuk 3	South Korea	Spring
Tonus	South Korea	Spring
Topas	Sweden	Spring
Tower	Canada	Spring
Turret	Canada	Spring
Vostochno-sibirskii	Russian Federation	Spring
Wasefuji	South Korea	Spring

Table A.12. Name, origin, and growth habits of the 146 spring ecotype germplasm accessions (including check cultivars) used in the study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Westar[b]	**Canada**	**Spring**
Willa	South Korea	Spring
Zhoungyou-821	China	Spring
Celebra	Sweden	Spring
Ceskia Tabor	Czech Republic	Spring
Colt	USA	Spring
Comet	Sweden	Spring
Conquest	Canada	Spring
Cougar	Canada	Spring
Cresor	France	Spring
Crop	France	Spring
Czyzowski	Poland	Spring
Delta	Sweden	Spring
Drakkar	France	Spring
Evvin	Russian Federation	Spring
Flint	USA	Spring
Fonto	South Korea	Spring
France 1	France	Spring

[a] Accessions used as resistant check
[b] Accession used as susceptible check

Table A.13. Name, origin, and growth habits of the 152 spring ecotype germplasm accessions (including check cultivars) used in the study

Name of the accessions	Country of origin/obtained	Growth habit
AR91004	USA	Winter
AR91017	USA	Winter
ARC 97018	USA	Winter
ARC 97019	USA	Winter
ARC-2180-1	USA	Winter
Azuma	South Korea	Semi-winter
Azumasho	South Korea	Semi-winter
Baraska	Germany	Winter
Barkant	Netherlands	Winter
Barnapoli	Germany	Winter
Barplina	South Korea	Winter
Beryl	Poland	Winter
Bienvenu	USA	Winter
Billy	Sweden	Winter
Bolko	Poland	Winter
BRA 1168/85	Italy	Winter
Bridger	USA	Winter
Capricorn	UK	Winter
Cascade	USA	Winter
Cathy	USA	Winter
Cescaljarni repka	South Korea	Semi-winter
Chisaya natane	Japan	Semi-winter
Chon nam	South Korea	Semi-winter
Cobra	Germany	Winter
Colza	South Korea	Spring
Colza 18 Miroc	South Korea	Semi-winter
Corvette	UK	Winter
Crystal	Sweden	Winter
Cult	Canada	Winter
Da vinci	Canada	Winter
Dae cho sen	South Korea	**Semi-winter**
DKW-46-5	USA	Winter

Table A.13. Name, origin, and growth habits of the 152 spring ecotype germplasm accessions (including check cultivars) used in the study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Dong Buk	South Korea	Winter
Doon Major Swede	New Zealand	Winter
Drawft	South Korea	Winter
Eckendorfer Mali	South Korea	Semi-winter
Elena	Germany	Winter
Eragi	Germany	Winter
Erra	Germany	Winter
Expander	Germany	Winter
Fashion	Canada	Winter
Fertodi	South Korea	Winter
Fuji	South Korea	Spring/ semi
G 32327	Albania	Winter
Galileo	Canada	Winter
Gebr Dippes	South Korea	Winter
Girita	Germany	Semi-winter
Glacier	Sweden	Winter
Goya	Canada	Winter
Gylle	South Korea	Semi-winter
Helga	Germany	Semi-winter
HOBSON	UK	Winter
Host rape regel	South Korea	Winter
Ibiza	Canada	Winter
Iwao natane	South Korea	Winter
Iwashiro-natane	South Korea	Winter
Iwawoochi	South Korea	Winter
Janpol	Poland	Winter
Jupiter	USA	Winter
Kasuya	South Korea	Winter
Kasuyashu	South Korea	Winter
Korina	Germany	Winter
Krasnodarskii	Russian Federation	Winter
KS3579	USA	Winter

Table A.13. Name, origin, and growth habits of the 152 spring ecotype germplasm accessions (including check cultivars) used in the study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Kuju	South Korea	Winter
Kutkowski	South Korea	Winter
Ladoga	Canada	Winter
Lembkes	South Korea	Winter
Lesira	Germany	Winter
Lester	Germany	Winter
Librador	Germany	Winter
Lieikoposki	South Korea	Semi-winter
Lindora-00	Germany	Winter
Lindore	Germany	Winter
Linglandor	Germany	Winter
Linus	South Korea	Winter
Lirama	Germany	Winter
Liratrop	Germany	Winter
Liropa	Germany	Winter
Lisora	Germany	Semi-winter
Luna	Germany	Winter
Major	France	Semi-winter
Mali	South Korea	Semi-winter
Marinus	Germany	Winter
Mihonatane	South Korea	Winter
Miochowski	France	Semi-winter
Mlochowski	Poland	Semi-winter
MR 1	South Korea	Winter
Mu.che!	South Korea	Winter
Murame nadame	South Korea	Semi-winter
Mutsumi	Japan	Semi-winter
N001-28-246-5-4	South Korea	Semi-winter
Nabo	South Korea	Semi-winter
Niedera-rubacher	South Korea	Winter
Nilla 1022	South Korea	Semi-winter
Nilla glossy	South Korea	Semi-winter

Table A.13. Name, origin, and growth habits of the 152 spring ecotype germplasm accessions (including check cultivars) used in the study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Nugget	South Korea	Semi-winter
NY-10	China	Semi-winter
NY-20	China	Semi-winter
NY-7	China	Semi-winter
NY-8	China	Semi-winter
Oleifera	South Korea	Semi-winter
Panter	Germany	Winter
Per	Sweden	Winter
Petanova-lihonova	South Korea	Semi-winter
Quinta	Germany	Winter
R. Creaus	South Korea	Winter
Rafal	France	Winter
Ramses	South Korea	Winter
Rang	South Korea	Semi-winter
Rapifera	South Korea	Winter
Rebel	USA	Semi-winter
Regal	South Korea	Winter
Ridana	Germany	Winter
Riley	USA	Winter
Rubin	Germany	Winter
Scherwitz	South Korea	Winter
Sera	Germany	Semi-winter
Silesia	Czech Republic	Winter
Skrzeszowicki	Poland	Winter
Status	Sweden	Winter
Su weon chag	South Korea	Semi-winter
Sumner	USA	Winter
Svaloefs Karab	Sweden	Winter
Synra	South Korea	Winter
Taichang	South Korea	Semi-winter
Takagi MS	South Korea	Semi-winter
Tamara	Germany	Winter

Table A.13. Name, origin, and growth habits of the 152 spring ecotype germplasm accessions (including check cultivars) used in the study (continued)

Name of the accessions	Country of origin/obtained	Growth habit
Tanka	South Korea	Semi-winter
Titus	South Korea	Winter
Todane	South Korea	Semi-winter
Tosharshu	South Korea	Winter
Trebicska	Czech Republic	Winter
Tri-Bridger	USA	Winter
Tsukushishu	South Korea	Winter
Ujfertodi	Hungary	Winter
Valdor	France	Winter
Vanda	Germany	Winter
Viking	Denmark	Winter
Vision	Canada	Winter
Weal dong cho	South Korea	Semi-winter
Weibulls margo	South Korea	Semi-winter
Wichita	USA	Winter
Wielkopolski	South Korea	Winter
Wipol	Norway	Semi-winter
Wira	Germany	Winter
Yong dang	South Korea	Semi-winter
Yonkokuban	South Korea	Winter
Zhoungyou-584	China	Semi-winter
Zhoungyou-9	China	Semi-winter
NEP63[a]	**USA**	**Semi-winter**
Licantara	Germany	Winter

[a] Accession used as resistant check

Table A.14. Combined analysis of variance (ANOVA) of sclerotinia stem rot resistance traits in 146 spring canola/rapeseed genotypes (including check cultivars)

Source of variation	Traits[a]				
	LL	LW	PM	LL_AUDPC	LW_AUDPC
Genotype	***	***	***	***	***
Genotype x Experiment	***	***	***	*	ns

*, ***, and ns indicate differences were significant at $P \leq 0.05$, $P \leq 0.0001$ levels of significance, and not significant

Traits[a]: LL, Lesion length measured at 7 days post inoculation (dpi); LW, lesion width at 7 dpi; PM, plant mortality at 15 dpi; LL_AUDPC, lesion lengths area under disease progress curve (AUDPC) calculated using 7 time points reading; LW_AUDPC, lesion widths AUDPC calculated using 7 time points reading.

Table A.15. Combined analysis of variance (ANOVA) of sclerotinia stem rot resistance traits in 152 semi-winter and winter canola/rapeseed genotypes (including check cultivars)

Source of variation	Traits[a]				
	LL	LW	PM	LL_AUDPC	LW_AUDPC
Genotype	*	**	**	*	***
Genotype x Experiment	***	***	*	*	*

*, **, and *** indicate differences were significant at $P \leq 0.05$, $P \leq 0.01$, $P \leq 0.0001$ levels of significance, and not significant

Traits[a]: LL, Lesion length measured at 7 days post inoculation (dpi); LW, lesion width at 7 dpi; PM, plant mortality at 15 dpi; LL_AUDPC, lesion lengths area under disease progress curve (AUDPC) calculated using 7 time points reading; LW_AUDPC, lesion widths AUDPC calculated using 7 time points reading.

Table A.16. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in spring ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies

Traits[a]	SNP	Chr[b]	Position	GWA models[c]	LOD	MAF	Allelic effect
LW_AUDPC	SCM002760.2_20267096	2	20267096	GEMMA-MLM	3.3	0.18	-87.651
PM	SCM002760.2_20267096	2	20267096	GEMMA-MLM	3.4	0.18	-13.616
PM	SCM002760.2_20267096	2	20267096	FarmCPU	3.0	0.18	14.827
LL	SCM002761.2_1259602	3	1259602	FarmCPU	4.0	0.21	0.646
LL	SCM002761.2_1259602	3	1259602	GEMMA-MLM	3.2	0.21	-0.954
LL_AUDPC	SCM002761.2_1259602	3	1259602	FarmCPU	3.7	0.21	12.169
LL_AUDPC	SCM002761.2_1259602	3	1259602	GEMMA-MLM	3.1	0.21	-9.460
LW	SCM002761.2_1259602	3	1259602	FarmCPU	3.2	0.21	7.558
LW_AUDPC	SCM002761.2_1259602	3	1259602	FarmCPU	3.3	0.21	75.804
PM	SCM002761.2_1259602	3	1259602	FarmCPU	3.3	0.21	10.729
LL	SCM002761.2_33351214	3	33351214	GEMMA-MLM	3.0	0.20	-0.889
LL_AUDPC	SCM002761.2_33351214	3	33351214	GEMMA-MLM	3.3	0.20	-9.547
LL_AUDPC	SCM002761.2_33351214	3	33351214	FarmCPU	3.2	0.20	-11.308
PM	SCM002761.2_33351214	3	33351214	GEMMA-MLM	3.0	0.20	-8.825
LL	SCM002761.2_44267358	3	44267358	GEMMA-MLM	3.0	0.18	1.017
LL_AUDPC	SCM002761.2_44267358	3	44267358	GEMMA-MLM	3.3	0.18	10.850
PM	SCM002761.2_44267358	3	44267358	GEMMA-MLM	3.0	0.18	10.159
LL	SCM002761.2_44424417	3	44424417	FarmCPU	3.3	0.06	-0.901
PM	SCM002762.2_11094674	4	11094674	GEMMA-MLM	3.8	0.06	22.258
LL	SCM002763.2_7201665	5	7201665	GEMMA-MLM	3.4	0.15	-1.045
LL_AUDPC	SCM002763.2_7201665	5	7201665	GEMMA-MLM	4.2	0.15	-11.858
LL_AUDPC	SCM002763.2_7201665	5	7201665	FarmCPU	4.0	0.15	-13.102
LW_AUDPC	SCM002763.2_7201665	5	7201665	GEMMA-MLM	3.2	0.15	-65.850
LW_AUDPC	SCM002763.2_7201665	5	7201665	FarmCPU	3.1	0.15	-75.897
PM	SCM002763.2_7201665	5	7201665	FarmCPU	3.7	0.15	-11.927
PM	SCM002763.2_7201665	5	7201665	GEMMA-MLM	3.6	0.15	-10.644

Table A.16. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in spring ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA models[c]	LOD	MAF	Allelic effect
LL	SCM002763.2_10442725	5	10442725	GEMMA-MLM	3.5	0.04	-2.646
LL_AUDPC	SCM002763.2_10442725	5	10442725	GEMMA-MLM	3.4	0.04	-26.240
LL_AUDPC	SCM002763.2_10442725	5	10442725	FarmCPU	3.4	0.04	-32.046
LW	SCM002763.2_10442725	5	10442725	GEMMA-MLM	4.0	0.04	-19.014
LW	SCM002763.2_10442725	5	10442725	FarmCPU	3.6	0.04	-22.376
LW_AUDPC	SCM002763.2_10442725	5	10442725	GEMMA-MLM	3.6	0.04	-176.372
LW_AUDPC	SCM002763.2_10442725	5	10442725	FarmCPU	3.2	0.04	-207.932
PM	SCM002763.2_10442725	5	10442725	GEMMA-MLM	3.7	0.04	-27.241
PM	SCM002763.2_10442725	5	10442725	FarmCPU	3.6	0.04	-31.608
LW_AUDPC	SCM002763.2_23237025	5	23237025	FarmCPU	3.3	0.03	-260.277
LL	SCM002763.2_25221820	5	25221820	GEMMA-MLM	3.8	0.21	0.920
LL_AUDPC	SCM002763.2_25221820	5	25221820	GEMMA-MLM	3.4	0.21	8.678
LW	SCM002763.2_25221820	5	25221820	GEMMA-MLM	3.0	0.21	5.392
LL	SCM002763.2_31228663	5	31228663	FarmCPU	4.7	0.11	-0.790
LL	SCM002763.2_31228663	5	31228663	GEMMA-MLM	3.7	0.11	-1.419
LL_AUDPC	SCM002763.2_31228663	5	31228663	FarmCPU	3.9	0.11	-16.589
LL_AUDPC	SCM002763.2_31228663	5	31228663	GEMMA-MLM	3.7	0.11	-14.453
LW	SCM002763.2_31228663	5	31228663	GEMMA-MLM	4.3	0.11	-10.482
LW	SCM002763.2_31228663	5	31228663	FarmCPU	4.0	0.11	-11.328
LW_AUDPC	SCM002763.2_31228663	5	31228663	FarmCPU	4.2	0.11	-114.020
LW_AUDPC	SCM002763.2_31228663	5	31228663	GEMMA-MLM	4.2	0.11	-101.048
PM	SCM002763.2_31228663	5	31228663	FarmCPU	3.8	0.11	-15.365
PM	SCM002763.2_31228663	5	31228663	GEMMA-MLM	3.8	0.11	-13.997
LL_AUDPC	SCM002764.2_27014783	6	27014783	FarmCPU	3.1	0.03	-36.833
LL_AUDPC	SCM002764.2_27014783	6	27014783	GEMMA-MLM	3.0	0.03	-29.709
LW	SCM002764.2_27014783	6	27014783	FarmCPU	3.2	0.03	-25.188

Table A.16. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in spring ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA models[c]	LOD	MAF	Allelic effect
LW	SCM002764.2_27014783	6	27014783	GEMMA-MLM	3.2	0.03	-20.525
LW_AUDPC	SCM002764.2_27014783	6	27014783	FarmCPU	3.7	0.03	-267.275
LW_AUDPC	SCM002764.2_27014783	6	27014783	GEMMA-MLM	3.6	0.03	-214.376
PM	SCM002764.2_27014783	6	27014783	FarmCPU	3.2	0.03	-35.552
PM	SCM002764.2_27014783	6	27014783	GEMMA-MLM	3.1	0.03	-29.604
LL	SCM002764.2_33628221	6	33628221	FarmCPU	3.7	0.32	-0.516
LL	SCM002765.2_8200081	7	8200081	GEMMA-MLM	3.1	0.18	1.017
LL_AUDPC	SCM002765.2_8200081	7	8200081	FarmCPU	3.3	0.18	-12.383
LW	SCM002765.2_8200081	7	8200081	FarmCPU	3.5	0.18	-8.576
LW_AUDPC	SCM002765.2_8200081	7	8200081	FarmCPU	3.2	0.18	-81.221
PM	SCM002765.2_8200081	7	8200081	FarmCPU	3.0	0.18	-11.146
LW	SCM002765.2_8273774	7	8273774	FarmCPU	3.4	0.22	-7.566
LW	SCM002765.2_8273774	7	8273774	GEMMA-MLM	3.3	0.22	6.204
LW_AUDPC	SCM002765.2_8273774	7	8273774	FarmCPU	3.2	0.22	-72.490
LW_AUDPC	SCM002765.2_8273774	7	8273774	GEMMA-MLM	3.1	0.22	59.008
LW	SCM002765.2_8339128	7	8339128	GEMMA-MLM	3.4	0.42	-6.292
LW	SCM002765.2_8339128	7	8339128	FarmCPU	3.4	0.42	-7.329
LW_AUDPC	SCM002765.2_8339128	7	8339128	GEMMA-MLM	3.0	0.42	-57.198
LL	SCM002766.2_24223849	8	24223849	FarmCPU	3.8	0.06	1.059
PM	SCM002767.2_35641015	9	35641015	GEMMA-MLM	3.1	0.21	-14.873
LL_AUDPC	SCM002767.2_35737007	9	35737007	GEMMA-MLM	3.5	0.17	-11.354
LL_AUDPC	SCM002767.2_35737007	9	35737007	FarmCPU	3.3	0.17	-13.095
PM	SCM002767.2_35737007	9	35737007	GEMMA-MLM	3.0	0.17	-10.224
LL_AUDPC	SCM002768.2_1532243	10	1532243	FarmCPU	3.4	0.08	16.523
LL_AUDPC	SCM002768.2_1532243	10	1532243	GEMMA-MLM	3.1	0.08	-13.390
LW_AUDPC	SCM002768.2_1532243	10	1532243	FarmCPU	3.2	0.08	105.877

Table A.16. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in spring ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA models[c]	LOD	MAF	Allelic effect
LW_AUDPC	SCM002768.2_1532243	10	1532243	GEMMA-MLM	3.1	0.08	-88.014
PM	SCM002768.2_1532243	10	1532243	FarmCPU	3.4	0.08	15.649
PM	SCM002768.2_1532243	10	1532243	GEMMA-MLM	3.2	0.08	-13.467
LL	SCM002769.2_2668073	11	2668073	GEMMA-MLM	3.0	0.30	0.855
LW	SCM002769.2_2668073	11	2668073	GEMMA-MLM	3.4	0.30	6.206
LW	SCM002769.2_5529604	11	5529604	GEMMA-MLM	3.5	0.33	6.063
LW_AUDPC	SCM002769.2_5529604	11	5529604	GEMMA-MLM	3.2	0.33	56.162
LL	SCM002769.2_13591509	11	13591509	GEMMA-MLM	3.4	0.29	-0.855
LL_AUDPC	SCM002769.2_13591509	11	13591509	FarmCPU	3.5	0.29	10.676
LL_AUDPC	SCM002769.2_13591509	11	13591509	GEMMA-MLM	3.0	0.29	-8.132
LW	SCM002769.2_13591509	11	13591509	FarmCPU	3.4	0.29	7.059
LW_AUDPC	SCM002769.2_13591509	11	13591509	FarmCPU	3.3	0.29	68.805
PM	SCM002769.2_13591509	11	13591509	FarmCPU	3.1	0.29	9.548
LW_AUDPC	SCM002769.2_13914840	11	13914840	GEMMA-MLM	3.4	0.04	-151.178
PM	SCM002769.2_13914840	11	13914840	GEMMA-MLM	3.1	0.04	-21.939
LL	SCM002769.2_29417842	11	29417842	GEMMA-MLM	3.8	0.43	-0.958
LL	SCM002769.2_29417842	11	29417842	FarmCPU	3.2	0.43	-0.438
LL_AUDPC	SCM002769.2_29417842	11	29417842	FarmCPU	3.2	0.43	-10.817
LL_AUDPC	SCM002769.2_29417842	11	29417842	GEMMA-MLM	3.1	0.43	-8.589
LW	SCM002769.2_29417842	11	29417842	FarmCPU	4.0	0.43	-8.141
LW	SCM002769.2_29417842	11	29417842	GEMMA-MLM	3.8	0.43	-6.421
LW_AUDPC	SCM002769.2_29417842	11	29417842	FarmCPU	3.4	0.43	-74.084
LW_AUDPC	SCM002769.2_29417842	11	29417842	GEMMA-MLM	3.1	0.43	-56.235
LL_AUDPC	SCM002769.2_31445688	11	31445688	FarmCPU	3.0	0.32	-14.928
LW	SCM002769.2_31445688	11	31445688	FarmCPU	3.4	0.32	-10.646
LW_AUDPC	SCM002769.2_31445688	11	31445688	FarmCPU	3.3	0.32	-103.165

Table A.16. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in spring ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA models[c]	LOD	MAF	Allelic effect
PM	SCM002769.2_31445688	11	31445688	FarmCPU	3.1	0.32	-14.157
LW_AUDPC	SCM002770.2_27505977	12	27505977	GEMMA-MLM	3.4	0.09	-108.919
PM	SCM002770.2_27505977	12	27505977	GEMMA-MLM	3.8	0.09	-17.828
LL_AUDPC	SCM002770.2_35193244	12	35193244	FarmCPU	3.7	0.22	-20.592
LL_AUDPC	SCM002770.2_35193244	12	35193244	GEMMA-MLM	3.3	0.22	-15.767
LW_AUDPC	SCM002770.2_35193244	12	35193244	FarmCPU	3.0	0.22	-120.926
PM	SCM002770.2_35193244	12	35193244	FarmCPU	3.8	0.22	-19.722
PM	SCM002770.2_35193244	12	35193244	GEMMA-MLM	3.7	0.22	-16.637
LL_AUDPC	SCM002770.2_61242936	12	61242936	GEMMA-MLM	3.4	0.09	-13.618
LL_AUDPC	SCM002770.2_61242936	12	61242936	FarmCPU	3.1	0.09	15.245
LW	SCM002770.2_61242936	12	61242936	GEMMA-MLM	3.7	0.09	-9.592
LW	SCM002770.2_61242936	12	61242936	FarmCPU	3.1	0.09	10.228
LW_AUDPC	SCM002770.2_61242936	12	61242936	GEMMA-MLM	4.2	0.09	-100.099
LW_AUDPC	SCM002770.2_61242936	12	61242936	FarmCPU	3.6	0.09	110.361
PM	SCM002770.2_61242936	12	61242936	GEMMA-MLM	3.2	0.09	-12.962
PM	SCM002770.2_61242936	12	61242936	FarmCPU	3.0	0.09	14.316
LL	SCM002770.2_62330224	12	62330224	GEMMA-MLM	3.0	0.43	0.749
LL_AUDPC	SCM002770.2_62330224	12	62330224	FarmCPU	3.3	0.43	9.079
LW	SCM002770.2_62330224	12	62330224	FarmCPU	3.1	0.43	5.964
LW_AUDPC	SCM002770.2_62330224	12	62330224	FarmCPU	3.0	0.43	57.655
LL	SCM002771.2_1951273	13	1951273	GEMMA-MLM	4.4	0.18	-1.139
LL_AUDPC	SCM002771.2_1951273	13	1951273	GEMMA-MLM	4.8	0.18	-12.112
LL_AUDPC	SCM002771.2_1951273	13	1951273	FarmCPU	4.2	0.18	-13.149
LW	SCM002771.2_1951273	13	1951273	GEMMA-MLM	3.4	0.18	-6.648
LW_AUDPC	SCM002771.2_1951273	13	1951273	GEMMA-MLM	3.5	0.18	-66.636
LW_AUDPC	SCM002771.2_1951273	13	1951273	FarmCPU	3.3	0.18	-76.777

Table A.16. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in spring ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA models[c]	LOD	MAF	Allelic effect
PM	SCM002771.2_1951273	13	1951273	GEMMA-MLM	3.7	0.18	-10.231
PM	SCM002771.2_1951273	13	1951273	FarmCPU	3.6	0.18	-11.499
PM	SCM002771.2_2495279	13	2495279	GEMMA-MLM	3.4	0.16	-14.301
LL	SCM002771.2_4764427	13	4764427	GEMMA-MLM	3.2	0.13	-1.257
LL_AUDPC	SCM002771.2_4764427	13	4764427	GEMMA-MLM	3.9	0.13	-14.191
LW	SCM002771.2_4764427	13	4764427	GEMMA-MLM	3.3	0.13	-8.558
LW_AUDPC	SCM002771.2_4764427	13	4764427	GEMMA-MLM	3.8	0.13	-90.827
PM	SCM002771.2_4764427	13	4764427	GEMMA-MLM	4.2	0.13	-14.710
LL_AUDPC	SCM002771.2_5119497	13	5119497	FarmCPU	3.0	0.17	-11.649
LL_AUDPC	SCM002772.2_1456731	14	1456731	FarmCPU	3.0	0.17	10.832
PM	SCM002772.2_1456731	14	1456731	FarmCPU	3.4	0.17	11.064
PM	SCM002772.2_1456731	14	1456731	GEMMA-MLM	3.3	0.17	9.652
LL	SCM002772.2_30952459	14	30952459	FarmCPU	6.2	0.11	0.994
LW	SCM002772.2_31459241	14	31459241	GEMMA-MLM	3.0	0.18	8.895
LL	SCM002772.2_44203541	14	44203541	FarmCPU	3.8	0.04	-1.481
LL	SCM002772.2_54108357	14	54108357	GEMMA-MLM	3.6	0.17	1.284
LL_AUDPC	SCM002772.2_54108357	14	54108357	GEMMA-MLM	4.3	0.17	14.242
LL_AUDPC	SCM002772.2_54108357	14	54108357	FarmCPU	3.2	0.17	-15.208
LW_AUDPC	SCM002772.2_54108357	14	54108357	GEMMA-MLM	3.1	0.17	77.182
PM	SCM002772.2_54108357	14	54108357	GEMMA-MLM	3.0	0.17	11.595
LL	SCM002772.2_69471400	14	69471400	GEMMA-MLM	5.1	0.25	-1.208
LL	SCM002772.2_69471400	14	69471400	FarmCPU	4.0	0.25	0.590
LL_AUDPC	SCM002772.2_69471400	14	69471400	GEMMA-MLM	4.7	0.25	-11.797
LL_AUDPC	SCM002772.2_69471400	14	69471400	FarmCPU	4.6	0.25	14.054
LW	SCM002772.2_69471400	14	69471400	GEMMA-MLM	4.7	0.25	-7.833
LW	SCM002772.2_69471400	14	69471400	FarmCPU	4.4	0.25	9.284

Table A.16. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in spring ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA models[c]	LOD	MAF	Allelic effect
LW_AUDPC	SCM002772.2_69471400	14	69471400	GEMMA-MLM	4.1	0.25	-71.445
LW_AUDPC	SCM002772.2_69471400	14	69471400	FarmCPU	4.0	0.25	87.063
PM	SCM002772.2_69471400	14	69471400	GEMMA-MLM	4.0	0.25	-10.719
PM	SCM002772.2_69471400	14	69471400	FarmCPU	4.0	0.25	12.387
LL	SCM002773.2_22943594	15	22943594	FarmCPU	5.5	0.33	0.822
LL_AUDPC	SCM002773.2_22943594	15	22943594	FarmCPU	4.1	0.33	17.350
LL_AUDPC	SCM002773.2_22943594	15	22943594	GEMMA-MLM	3.2	0.33	-12.457
LW	SCM002773.2_22943594	15	22943594	FarmCPU	4.2	0.33	11.833
LW	SCM002773.2_22943594	15	22943594	GEMMA-MLM	3.2	0.33	-8.387
LW_AUDPC	SCM002773.2_22943594	15	22943594	FarmCPU	4.4	0.33	120.174
LW_AUDPC	SCM002773.2_22943594	15	22943594	GEMMA-MLM	3.6	0.33	-86.771
PM	SCM002773.2_22943594	15	22943594	FarmCPU	4.2	0.33	16.664
PM	SCM002773.2_22943594	15	22943594	GEMMA-MLM	3.9	0.33	-13.705
LL	SCM002773.2_28072565	15	28072565	FarmCPU	4.1	0.32	-0.600
LL	SCM002774.2_33338934	16	33338934	GEMMA-MLM	3.1	0.18	-1.005
LW	SCM002774.2_33338934	16	33338934	GEMMA-MLM	3.3	0.18	-7.106
LW_AUDPC	SCM002774.2_33338934	16	33338934	GEMMA-MLM	3.1	0.18	-67.328
PM	SCM002774.2_33338934	16	33338934	GEMMA-MLM	3.3	0.18	-10.324
LL	SCM002775.2_9203423	17	9203423	FarmCPU	6.2	0.09	-1.392
PM	SCM002775.2_9203423	17	9203423	GEMMA-MLM	3.4	0.09	-16.690
LL	SCM002775.2_11411683	17	11411683	GEMMA-MLM	3.3	0.18	0.999
LL_AUDPC	SCM002775.2_11411683	17	11411683	GEMMA-MLM	3.7	0.18	10.808
LW	SCM002775.2_11411683	17	11411683	GEMMA-MLM	3.7	0.18	7.158
LW_AUDPC	SCM002775.2_11411683	17	11411683	GEMMA-MLM	3.9	0.18	71.423
LL_AUDPC	SCM002776.2_8341541	18	8341541	FarmCPU	3.6	0.36	-10.659
LW	SCM002776.2_8341541	18	8341541	FarmCPU	3.3	0.36	-6.899

Table A.16. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in spring ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA models[c]	LOD	MAF	Allelic effect
LW_AUDPC	SCM002776.2_8341541	18	8341541	FarmCPU	3.4	0.36	-68.812
PM	SCM002776.2_9877851	18	9877851	GEMMA-MLM	3.4	0.10	-13.349
LL_AUDPC	SCM002776.2_12072563	18	12072563	GEMMA-MLM	3.1	0.15	-10.761
LW	SCM002776.2_12072563	18	12072563	GEMMA-MLM	3.1	0.15	-7.306
LW_AUDPC	SCM002776.2_12072563	18	12072563	GEMMA-MLM	3.0	0.15	-69.408
PM	SCM002776.2_12072563	18	12072563	GEMMA-MLM	3.4	0.15	-11.024
LL	SCM002776.2_45864688	18	45864688	FarmCPU	3.5	0.32	0.644

Traits[a]: LL = Lesion length measured at 7 days post inoculation (dpi); LW = lesion width at 7 dpi; PM = plant mortality at 15 dpi; LL_AUDPC = lesion lengths area under disease progress curve (AUDPC) calculated using 7 time points reading; LW_AUDPC = lesion widths AUDPC calculated using 7 time points reading
Chr[b]: *Brassica napus* chromosome
GWA Models[c]: Two GWA mapping models *i.e.,* GEMMA-MLM (Single-locus), FarmCPU (multi-locus)

Table A.17. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected in 144 spring populations by at least two or more traits associated with sclerotinia stem rot resistance by genome-wide association studies

SNP	Chr[a]	Position	GWA models and Traits[b]	LOD[c]	MAF	Previously Detected MTAs[d]
SCM002760.2_20267096	2	20267096	GEMMA-MLM[34]; FarmCPU[3]	3.0-3.4	0.18	Wu et al. (2013) [QTL: SRA2-1: Position: (16.67-20.47 Mb)]
SCM002760.2_31650036	2	31650036	GEMMA-MLM[145]	3.0-3.2	0.08	
SCM002761.2_1259602	3	1259602	GEMMA-MLM[14]; FarmCPU[12345]	3.1-4.0	0.21	
SCM002761.2_33351214	3	33351214	GEMMA-MLM[134]; FarmCPU[1]	3.0-3.3	0.20	
SCM002761.2_44267358	3	44267358	GEMMA-MLM[134]	3.0-3.3	0.18	
SCM002763.2_7201665	5	7201665	GEMMA-MLM[1345]; FarmCPU[345]	3.1-4.2	0.15	
SCM002763.2_10442725	5	10442725	GEMMA-MLM[12345]; FarmCPU[2345]	3.2-4.0	0.04	
SCM002763.2_25221820	5	25221820	GEMMA-MLM[124]	3.0-3.8	0.21	
SCM002763.2_31228663	5	31228663	GEMMA-MLM[12345]; FarmCPU[12345]	3.7-4.7	0.11	
SCM002764.2_27014783	6	27014783	GEMMA-MLM[2345]; FarmCPU[2345]	3.0-3.7	0.03	
SCM002765.2_8200081	7	8200081	GEMMA-MLM[2345]; FarmCPU[1]	3.0-3.5	0.18	
SCM002765.2_8273774	7	8273774	GEMMA-MLM[25]; FarmCPU[25]	3.0-3.4	0.22	
SCM002765.2_8339128	7	8339128	GEMMA-MLM[25]; FarmCPU[2]	3.0-3.4	0.42	
SCM002767.2_35737007	9	35737007	GEMMA-MLM[34]; FarmCPU[4]	3.0-3.5	0.17	Roy et al. (2021) [SCM002767.2_35588232 (35.59 Mb), SCM002767.2_36527400 (36.53 Mb)]

Table A.17. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected in 144 spring populations by at least two or more traits associated with sclerotinia stem rot resistance by genome-wide association studies (continued)

SNP	Chr[a]	Position	GWA models and Traits[b]	LOD[c]	MAF	Previously Detected MTAs[d]
SCM002768.2_1532243	10	1532243	GEMMA-MLM^{345}; FarmCPU^{345}	3.1-3.4	0.08	
SCM002769.2_2578220	11	2578220	GEMMA-MLM^{125}	3.0-3.2	0.40	
SCM002769.2_13591509	11	13591509	GEMMA-MLM^{14}; FarmCPU^{2345}	3.0-3.5	0.29	
SCM002769.2_13914840	11	13914840	GEMMA-MLM^{35}	3.1-3.4	0.04	
SCM002769.2_29417842	11	29417842	GEMMA-MLM^{1245}; FarmCPU^{1245}	3.1-4.0	0.43	Shahoveisi et al. (2021) [SR63.C1.3 (21.85-31.35 Mb)]
SCM002769.2_31445688	11	31445688	FarmCPU^{2345}	3.0-3.4	0.32	Shahoveisi et al. (2021) [SR63.C1.3 (21.85-31.35 Mb)]
SCM002770.2_18672852	12	18672852	GEMMA-MLM^{1245}; FarmCPU^{4}	3.0-3.2	0.15	
SCM002770.2_35193244	12	35193244	GEMMA-MLM^{34}; FarmCPU^{345}	3.0-3.8	0.22	
SCM002770.2_61242936	12	61242936	GEMMA-MLM^{2345}; FarmCPU^{2345}	3.0-4.2	0.09	
SCM002770.2_62330224	12	62330224	GEMMA-MLM^{1}; FarmCPU^{245}	3.0-3.2	0.43	Roy et al. (2021) [SCM002770.2_62791159 (62.79 Mb)]
SCM002770.2_63268428	12	63268428	GEMMA-MLM^{12}; FarmCPU^{2}	3.0-3.2	0.43	
SCM002771.2_1951273	13	1951273	GEMMA-MLM^{12345}; FarmCPU^{345}	3.3-4.8	0.18	Wu et al. (2019) [qSRC3: 0.19-3.39 Mb]; Qasim et al. (2020) [SRC3a: 2.26-3.47 Mb]
SCM002771.2_4764427	13	4764427	GEMMA-MLM^{12345}	3.2-4.2	0.13	

Table A.17. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected in 144 spring populations by at least two or more traits associated with sclerotinia stem rot resistance by genome-wide association studies (continued)

SNP	Chr[a]	Position	GWA models and Traits[b]	LOD[c]	MAF	Previously Detected MTAs[d]
SCM002772.2_1456731	14	1456731	GEMMA-MLM[3]; FarmCPU[34]	3.0-3.4	0.17	Zhao et al. (2006) [*Sll14a* (0.40-9.42 Mb)]
SCM002772.2_54108357	14	54108357	GEMMA-MLM[1345]; FarmCPU[4]	3.0-4.3	0.17	
SCM002772.2_69471400	14	69471400	GEMMA-MLM[12345]; FarmCPU[12345]	4.0-5.1	0.25	
SCM002773.2_22943594	15	22943594	GEMMA-MLM[2345]; FarmCPU[12345]	3.2-5.5	0.33	
SCM002773.2_36045830	15	36045830	GEMMA-MLM[1234]	3.0-3.2	0.08	
SCM002774.2_11393878	16	11393878	GEMMA-MLM[3]; FarmCPU[3]	3.0	0.10	
SCM002774.2_33338934	16	33338934	GEMMA-MLM[1245]	3.1-3.3	0.18	Zhao et al. (2006) [Sll16 (23.18-35.47 Mb); Dw16 (28.55-35.47 Mb)]; Wu et al. (2013) (SRC6-1, SRC6-2: 30.28-34.59 Mb; Qasim et al. (2020) [SRC6 (32.95-34.45 Mb)]; Roy et al. (2021) [SCM002774.2_33238118 (33.24 Mb); SCM002774.2_33265642 (33.27 Mb); SCM002774.2_33563646 (33.56 Mb)

Table A.17. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected in 144 spring populations by at least two or more traits associated with sclerotinia stem rot resistance by genome-wide association studies (continued)

SNP	Chr[a]	Position	GWA models and Traits[b]	LOD[c]	MAF	Previously Detected MTAs[d]
SCM002775.2_11411683	17	11411683	GEMMA-MLM[1245]	3.3-3.9	0.18	
SCM002776.2_8341541	18	8341541	FarmCPU[245]	3.3-3.6	0.36	Wu et al. (2019) [qSRC8 (1.62-12.09 Mb)]
SCM002776.2_12072563	18	12072563	GEMMA-MLM[2345]	3.0-3.4	0.15	Wu et al. (2019) [qSRC8 (1.62-12.09 Mb)]

Chr[a]: *Brassica napus* chromosome

GWA models and Traits[b]: Methods, GEMMA-MLM and FarmCPU; Traits: Superscript 1, 2, 3, 4, and 5 represents lesion length (LL) measured at 7 days post inoculation (dpi), lesion width (LW) at 7 dpi, plant mortality (PM) at 15 dpi, lesion lengths area under disease progress curve (LL_AUDPC) calculated using 7 time points reading, and lesion widths AUDPC (LW_AUDPC) calculated using 7 time points reading, respectively.

LOD[c]: The highest and lowest $-\log_{10}$ (P) value resulted from the various studied traits with different GWA models

Previously reported MTAs[d]: previously reported marker-trait-associations (MTAs) along with their physical positions from various QTL and GWA-related studies which overlapped or in close proximity with the genomic regions in this study

Table A.18. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in semi-winter and winter ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies

Traits[a]	SNP	Chr[b]	Position	GWA Models[c]	LOD	MAF	Allelic effect
LL	SCM002759.2_7025054	1	7025054	FarmCPU	3.3	0.47	0.52
LL_AUDPC	SCM002759.2_7025054	1	7025054	FarmCPU	3.6	0.47	3.56
LW	SCM002759.2_7025054	1	7025054	FarmCPU	3.4	0.47	6.23
LW_AUDPC	SCM002759.2_7025054	1	7025054	FarmCPU	3.7	0.47	5.81
PM	SCM002759.2_7025054	1	7025054	FarmCPU	4.0	0.47	38.36
LL	SCM002759.2_7025054	1	7025054	GEMMA-MLM	3.9	0.47	
LL_AUDPC	SCM002759.2_7025054	1	7025054	GEMMA-MLM	4.3	0.47	
LW	SCM002759.2_7025054	1	7025054	GEMMA-MLM	3.7	0.47	
LW_AUDPC	SCM002759.2_7025054	1	7025054	GEMMA-MLM	4.1	0.47	
PM	SCM002759.2_7025054	1	7025054	GEMMA-MLM	4.1	0.47	
LL	SCM002759.2_7052802	1	7052802	FarmCPU	3.1	0.41	0.51
LL_AUDPC	SCM002759.2_7052802	1	7052802	FarmCPU	3.4	0.41	3.80
LW	SCM002759.2_7052802	1	7052802	FarmCPU	3.7	0.41	5.74
LW_AUDPC	SCM002759.2_7052802	1	7052802	FarmCPU	4.2	0.41	5.72
PM	SCM002759.2_7052802	1	7052802	FarmCPU	3.4	0.41	41.86
LL	SCM002759.2_7052802	1	7052802	GEMMA-MLM	3.3	0.41	
LL_AUDPC	SCM002759.2_7052802	1	7052802	GEMMA-MLM	3.5	0.41	
LW	SCM002759.2_7052802	1	7052802	GEMMA-MLM	3.8	0.41	
LW_AUDPC	SCM002759.2_7052802	1	7052802	GEMMA-MLM	4.3	0.41	
PM	SCM002759.2_7052802	1	7052802	GEMMA-MLM	3.4	0.41	
LL	SCM002759.2_10710048	1	10710048	FarmCPU	3.4	0.07	-0.97
LL_AUDPC	SCM002759.2_10710048	1	10710048	FarmCPU	3.3	0.07	-10.12
LL	SCM002759.2_10710048	1	10710048	GEMMA-MLM	3.5	0.07	
LL_AUDPC	SCM002759.2_10710048	1	10710048	GEMMA-MLM	3.4	0.07	

Table A.18. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in semi-winter and winter ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA Models[c]	LOD	MAF	Allelic effect
LW	SCM002759.2_22108854	1	22108854	FarmCPU	3.4	0.15	-4.74
LW	SCM002759.2_22108854	1	22108854	GEMMA-MLM	3.4	0.16	
LW	SCM002760.2_3833184	2	3833184	GEMMA-MLM	3.3	0.10	
LL_AUDPC	SCM002760.2_24777699	2	24777699	GEMMA-MLM	3.3	0.07	
LL	SCM002761.2_9437414	3	9437414	GEMMA-MLM	3.4	0.04	
LL	SCM002761.2_16323070	3	16323070	GEMMA-MLM	3.6	0.33	6.59
LW	SCM002761.2_27270949	3	27270949	FarmCPU	4.1	0.20	6.59
LW_AUDPC	SCM002761.2_27270949	3	27270949	FarmCPU	4.2	0.20	68.89
LW	SCM002761.2_27270949	3	27270949	GEMMA-MLM	4.4	0.20	-6.37
LW_AUDPC	SCM002761.2_27270949	3	27270949	GEMMA-MLM	4.4	0.20	-10.11
PM	SCM002761.2_29897262	3	29897262	FarmCPU	3.5	0.20	-11.89
PM	SCM002761.2_29897262	3	29897262	GEMMA-MLM	3.5	0.20	
LW_AUDPC	SCM002761.2_49022900	3	49022900	FarmCPU	3.5	0.08	-68.94
LL	SCM002761.2_49022900	3	49022900	GEMMA-MLM	3.4	0.08	
LL_AUDPC	SCM002761.2_49022900	3	49022900	GEMMA-MLM	3.4	0.08	
LW	SCM002761.2_49022900	3	49022900	GEMMA-MLM	3.4	0.08	
LW_AUDPC	SCM002761.2_49022900	3	49022900	GEMMA-MLM	3.5	0.08	
LW	SCM002762.2_20793967	4	20793967	FarmCPU	3.7	0.47	-3.92
LW	SCM002762.2_20793967	4	20793967	GEMMA-MLM	3.9	0.47	
LW	SCM002762.2_22210032	4	22210032	FarmCPU	3.8	0.11	-7.52
LW	SCM002762.2_22210032	4	22210032	GEMMA-MLM	4.0	0.11	
LL	SCM002763.2_8993185	5	8993185	FarmCPU	4.0	0.13	0.92
LL_AUDPC	SCM002763.2_8993185	5	8993185	FarmCPU	3.6	0.13	6.18
LW	SCM002763.2_8993185	5	8993185	FarmCPU	4.0	0.13	9.36

Table A.18. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in semi-winter and winter ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA Models[c]	LOD	MAF	Allelic effect
LW_AUDPC	SCM002763.2_8993185	5	8993185	FarmCPU	4.1	0.13	64.89
LL	SCM002763.2_8993185	5	8993185	GEMMA-MLM	4.4	0.14	
LL_AUDPC	SCM002763.2_8993185	5	8993185	GEMMA-MLM	3.6	0.14	
LW	SCM002763.2_8993185	5	8993185	GEMMA-MLM	4.3	0.14	
LW_AUDPC	SCM002763.2_8993185	5	8993185	GEMMA-MLM	4.2	0.14	
PM	SCM002763.2_18946206	5	18946206	FarmCPU	3.3	0.04	-16.65
PM	SCM002763.2_18946206	5	18946206	GEMMA-MLM	3.3	0.04	42.14
LL	SCM002764.2_9194211	6	9194211	FarmCPU	3.4	0.04	1.92
LL	SCM002764.2_9194211	6	9194211	GEMMA-MLM	3.6	0.04	42.14
LL_AUDPC	SCM002764.2_17606602	6	17606602	GEMMA-MLM	3.4	0.20	
LW_AUDPC	SCM002764.2_17606602	6	17606602	GEMMA-MLM	3.5	0.20	
LL	SCM002764.2_17885307	6	17885307	GEMMA-MLM	3.4	0.05	
LW	SCM002764.2_17885307	6	17885307	GEMMA-MLM	3.3	0.05	
PM	SCM002767.2_29730	9	29730	FarmCPU	3.6	0.19	8.19
PM	SCM002767.2_29730	9	29730	GEMMA-MLM	3.6	0.19	
LL	SCM002767.2_3253388	9	3253388	GEMMA-MLM	3.3	0.09	
LL_AUDPC	SCM002767.2_5277679	9	5277679	FarmCPU	3.4	0.07	12.06
LL	SCM002767.2_5277679	9	5277679	GEMMA-MLM	3.3	0.07	
LL_AUDPC	SCM002767.2_5277679	9	5277679	GEMMA-MLM	3.4	0.07	
LL	SCM002767.2_5862687	9	5862687	FarmCPU	3.3	0.08	1.05
LL	SCM002767.2_5862687	9	5862687	GEMMA-MLM	3.5	0.08	
LL	SCM002767.2_38044090	9	38044090	GEMMA-MLM	3.3	0.11	
LW_AUDPC	SCM002767.2_38052182	9	38052182	FarmCPU	3.3	0.10	67.47
LW_AUDPC	SCM002767.2_38052182	9	38052182	GEMMA-MLM	3.4	0.10	

Table A.18. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in semi-winter and winter ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA Models[c]	LOD	MAF	Allelic effect
LW	SCM002767.2_38061266	9	38061266	FarmCPU	3.3	0.15	6.19
LW	SCM002767.2_38061266	9	38061266	GEMMA-MLM	3.4	0.15	
LW	SCM002767.2_38082926	9	38082926	FarmCPU	3.7	0.13	7.08
LW_AUDPC	SCM002767.2_38082926	9	38082926	FarmCPU	3.4	0.13	11.14
PM	SCM002767.2_38082926	9	38082926	FarmCPU	3.6	0.13	69.68
LW	SCM002767.2_38082926	9	38082926	GEMMA-MLM	3.8	0.14	
LW_AUDPC	SCM002767.2_38082926	9	38082926	GEMMA-MLM	3.4	0.14	
PM	SCM002767.2_38082926	9	38082926	GEMMA-MLM	3.6	0.14	
LW_AUDPC	SCM002767.2_38083442	9	38083442	FarmCPU	3.4	0.11	-65.33
LW_AUDPC	SCM002767.2_38083442	9	38083442	GEMMA-MLM	3.3	0.11	
LL	SCM002767.2_38119743	9	38119743	GEMMA-MLM	3.3	0.09	-10.83
LW_AUDPC	SCM002767.2_38774352	9	38774352	GEMMA-MLM	3.3	0.10	
LL_AUDPC	SCM002767.2_41958710	9	41958710	GEMMA-MLM	3.3	0.10	
LL_AUDPC	SCM002767.2_41990070	9	41990070	FarmCPU	3.4	0.08	10.51
LW_AUDPC	SCM002767.2_41990070	9	41990070	FarmCPU	3.3	0.08	66.83
LL_AUDPC	SCM002767.2_41990070	9	41990070	GEMMA-MLM	3.4	0.08	
LW_AUDPC	SCM002767.2_41990070	9	41990070	GEMMA-MLM	3.3	0.08	
LL	SCM002767.2_42258678	9	42258678	GEMMA-MLM	3.4	0.14	
LL	SCM002768.2_9952251	10	9952251	FarmCPU	3.6	0.12	-0.87
LL	SCM002768.2_9952251	10	9952251	GEMMA-MLM	3.9	0.12	
LW_AUDPC	SCM002768.2_10253247	10	10253247	GEMMA-MLM	3.4	0.22	
LL	SCM002768.2_14218999	10	14218999	GEMMA-MLM	3.3	0.50	
LL	SCM002769.2_3794970	11	3794970	GEMMA-MLM	3.3	0.08	
LL	SCM002769.2_8989138	11	8989138	FarmCPU	3.5	0.16	-0.94

Table A.18. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in semi-winter and winter ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA Models[c]	LOD	MAF	Allelic effect
LL_AUDPC	SCM002769.2_8989138	11	8989138	FarmCPU	3.4	0.16	-6.04
LW_AUDPC	SCM002769.2_8989138	11	8989138	FarmCPU	3.3	0.16	-62.58
LL	SCM002769.2_8989138	11	8989138	GEMMA-MLM	3.6	0.16	
LL_AUDPC	SCM002769.2_8989138	11	8989138	GEMMA-MLM	3.5	0.16	
LW	SCM002769.2_8989138	11	8989138	GEMMA-MLM	3.4	0.16	
LL	SCM002769.2_43565693	11	43565693	FarmCPU	3.5	0.05	-1.36
LL	SCM002769.2_43565693	11	43565693	GEMMA-MLM	3.9	0.05	
LL_AUDPC	SCM002769.2_46229694	11	46229694	GEMMA-MLM	3.4	0.04	
LL	SCM002769.2_47724348	11	47724348	FarmCPU	3.7	0.37	-0.54
LL_AUDPC	SCM002769.2_47724348	11	47724348	FarmCPU	3.7	0.37	-5.81
LL	SCM002769.2_47724348	11	47724348	GEMMA-MLM	3.8	0.37	
LL_AUDPC	SCM002769.2_47724348	11	47724348	GEMMA-MLM	3.9	0.37	
LL_AUDPC	SCM002770.2_66428152	12	66428152	FarmCPU	3.4	0.13	9.89
LL	SCM002770.2_66428152	12	66428152	GEMMA-MLM	3.4	0.13	
LL_AUDPC	SCM002770.2_66428152	12	66428152	GEMMA-MLM	3.8	0.13	
LL	SCM002771.2_5548449	13	5548449	FarmCPU	3.6	0.05	1.47
LL	SCM002771.2_5548449	13	5548449	GEMMA-MLM	3.9	0.05	
LW	SCM002771.2_5548449	13	5548449	GEMMA-MLM	3.4	0.05	
LL	SCM002771.2_30920466	13	30920466	FarmCPU	3.6	0.35	-0.71
LW	SCM002771.2_30920466	13	30920466	FarmCPU	3.9	0.35	-6.89
LW_AUDPC	SCM002771.2_30920466	13	30920466	FarmCPU	3.6	0.35	-49.01
LL	SCM002771.2_30920466	13	30920466	GEMMA-MLM	3.7	0.35	
LW	SCM002771.2_30920466	13	30920466	GEMMA-MLM	4.0	0.35	
LW_AUDPC	SCM002771.2_30920466	13	30920466	GEMMA-MLM	3.6	0.35	

Table A.18. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in semi-winter and winter ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA Models[c]	LOD	MAF	Allelic effect
LL	SCM002771.2_38591806	13	38591806	GEMMA-MLM	3.3	0.04	
LL_AUDPC	SCM002771.2_44393183	13	44393183	GEMMA-MLM	3.4	0.14	
PM	SCM002772.2_5924485	14	5924485	FarmCPU	3.5	0.45	-5.63
PM	SCM002772.2_5924485	14	5924485	GEMMA-MLM	3.7	0.45	-1.03
LL_AUDPC	SCM002772.2_7708586	14	7708586	FarmCPU	3.8	0.09	-14.11
PM	SCM002772.2_7708586	14	7708586	FarmCPU	4.8	0.09	-73.52
LL_AUDPC	SCM002772.2_7708586	14	7708586	GEMMA-MLM	3.9	0.09	
LW_AUDPC	SCM002772.2_7708586	14	7708586	GEMMA-MLM	3.4	0.09	
PM	SCM002772.2_7708586	14	7708586	GEMMA-MLM	5.1	0.09	
LL_AUDPC	SCM002772.2_67533692	14	67533692	GEMMA-MLM	3.3	0.05	
LL	SCM002773.2_5859014	15	5859014	FarmCPU	4.6	0.09	-1.75
LL_AUDPC	SCM002773.2_5859014	15	5859014	FarmCPU	3.8	0.09	-10.72
LW	SCM002773.2_5859014	15	5859014	FarmCPU	3.9	0.09	-16.86
LW_AUDPC	SCM002773.2_5859014	15	5859014	FarmCPU	3.7	0.09	-107.59
LL	SCM002773.2_5859014	15	5859014	GEMMA-MLM	5.0	0.09	
LL_AUDPC	SCM002773.2_5859014	15	5859014	GEMMA-MLM	3.9	0.09	
LW	SCM002773.2_5859014	15	5859014	GEMMA-MLM	4.1	0.09	
LW_AUDPC	SCM002773.2_5859014	15	5859014	GEMMA-MLM	3.7	0.09	
LW	SCM002773.2_11017178	15	11017178	FarmCPU	3.4	0.05	-9.73
LL	SCM002773.2_11017178	15	11017178	GEMMA-MLM	3.6	0.05	
LW	SCM002773.2_11017178	15	11017178	GEMMA-MLM	3.6	0.05	
LW_AUDPC	SCM002773.2_11017178	15	11017178	GEMMA-MLM	3.3	0.05	
LL	SCM002773.2_17211900	15	17211900	FarmCPU	3.6	0.04	-1.64
LL_AUDPC	SCM002773.2_17211900	15	17211900	FarmCPU	3.7	0.04	-10.16

Table A.18. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in semi-winter and winter ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA Models[c]	LOD	MAF	Allelic effect
LW_AUDPC	SCM002773.2_17211900	15	17211900	FarmCPU	3.4	0.04	-109.46
LL	SCM002773.2_17211900	15	17211900	GEMMA-MLM	4.0	0.04	
LL_AUDPC	SCM002773.2_17211900	15	17211900	GEMMA-MLM	4.1	0.04	
LW	SCM002773.2_17211900	15	17211900	GEMMA-MLM	3.4	0.04	
LW_AUDPC	SCM002773.2_17211900	15	17211900	GEMMA-MLM	3.7	0.04	
LW	SCM002773.2_29962917	15	29962917	FarmCPU	3.7	0.15	6.38
LW_AUDPC	SCM002773.2_29962917	15	29962917	FarmCPU	3.5	0.15	64.39
LW	SCM002773.2_29962917	15	29962917	GEMMA-MLM	3.8	0.16	
LW_AUDPC	SCM002773.2_29962917	15	29962917	GEMMA-MLM	3.5	0.16	
LL	SCM002773.2_43256138	15	43256138	GEMMA-MLM	3.3	0.06	
LL	SCM002774.2_20774649	16	20774649	FarmCPU	3.5	0.10	-1.28
LL	SCM002774.2_20774649	16	20774649	GEMMA-MLM	3.7	0.10	
LL	SCM002774.2_32559985	16	32559985	FarmCPU	3.3	0.04	1.48
LL	SCM002775.2_12215584	17	12215584	FarmCPU	3.6	0.10	-0.97
LL_AUDPC	SCM002775.2_12215584	17	12215584	FarmCPU	3.4	0.10	-9.93
LL	SCM002775.2_12215584	17	12215584	GEMMA-MLM	3.7	0.10	
LL_AUDPC	SCM002775.2_12215584	17	12215584	GEMMA-MLM	3.4	0.10	
LL	SCM002775.2_41663703	17	41663703	GEMMA-MLM	3.4	0.40	
LL_AUDPC	SCM002775.2_41663703	17	41663703	GEMMA-MLM	3.3	0.40	
LL_AUDPC	SCM002776.2_16868604	18	16868604	FarmCPU	3.5	0.08	-10.88
LL_AUDPC	SCM002776.2_16868604	18	16868604	GEMMA-MLM	3.7	0.08	

Table A.18. List of significant single-nucleotide polymorphisms (SNPs) associated with five traits for sclerotinia stem rot resistance in semi-winter and winter ecotype rapeseed/canola genotypes detected by single-locus (GEMMA-MLM) and multi-locus (FarmCPU) genome-wide association studies (continued)

Traits[a]	SNP	Chr[b]	Position	GWA Models[c]	LOD	MAF	Allelic effect
LL	SCM002777.2_32367681	19	32367681	FarmCPU	3.7	0.07	1.39
LL_AUDPC	SCM002777.2_32367681	19	32367681	FarmCPU	3.3	0.07	8.47
LL	SCM002777.2_32367681	19	32367681	GEMMA-MLM	3.9	0.07	
LL_AUDPC	SCM002777.2_32367681	19	32367681	GEMMA-MLM	3.4	0.07	

Traits[a]: LL = Lesion length measured at 7 days post inoculation (dpi); LW = lesion width at 7 dpi; PM = plant mortality at 15 dpi; LL_AUDPC = lesion lengths area under disease progress curve (AUDPC) calculated using 7 time points reading; LW_AUDPC = lesion widths AUDPC calculated using 7-time points readings

Chr[b]: *Brassica napus* chromosome

GWA Models[c]: Two GWA models i.e. GEMMA-MLM (Single-locus), FarmCPU (multi-locus)

Table A.19. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected in 152 semi-winter and winter populations by at least two or more traits associated with sclerotinia stem rot resistance by GWA studies

SNP	Chr[a]	Position	GWA methods and Traits[b]	LOD[c]	MAF	Previously Detected MTAs[d]
SCM002759.2_7025054	1	7025054	GEMMA-MLM[12345]; FarmCPU[12345]	3.3-4.3	0.47	
SCM002759.2_7052802	1	7052802	GEMMA-MLM[12345]; FarmCPU[12345]	3.1-4.3	0.41	
SCM002759.2_10710048	1	10710048	GEMMA-MLM[14]; FarmCPU[14]	3.3-3.5	0.07	
SCM002759.2_22108854	1	22108854	GEMMA-MLM[12]; FarmCPU[12]	3.0-3.4	0.15	
SCM002760.2_3833184	2	3833184	GEMMA-MLM[12345]; FarmCPU[12345]	3.0-3.3	0.10	Zhao et al. (2006) [Sll2: 0.03-3.45 Mb)]; Wei et al. (2014) [qSR10-1 (1.61-7.71 Mb)]
SCM002761.2_9437414	3	9437414	GEMMA-MLM[1]; FarmCPU[15]	3.0-3.4	0.04	
SCM002761.2_27270949	3	27270949	GEMMA-MLM[125]; FarmCPU[25]	3.0-4.4	0.20	
SCM002761.2_49022900	3	49022900	GEMMA-MLM[1245]; FarmCPU[1245]	3.1-3.5	0.08	
SCM002762.2_20793967	4	20793967	GEMMA-MLM[25]; FarmCPU[25]	3.0-3.9	0.47	Roy et al. (2021) [SCM002762.2_20212672 (20.21 Mb); SCM002762.2_20789292 (20.79 Mb); SCM002762.2_20860192 (20.86 Mb)]
SCM002762.2_22210032	4	22210032	GEMMA-MLM[125]; FarmCPU[125]	3.1-4.0	0.11	
SCM002763.2_8993185	5	8993185	GEMMA-MLM[1245]; FarmCPU[1245]	3.6-4.4	0.13	

Table A.19. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected in 152 semi-winter and winter populations by at least two or more traits associated with sclerotinia stem rot resistance by GWA studies (continued)

SNP	Chr[a]	Position	GWA methods and Traits[b]	LOD[c]	MAF	Previously Detected MTAs[d]
SCM002763.2_18946206	5	18946206	GEMMA-MLM[34]; FarmCPU[34]	3.1-3.3	0.04	
SCM002764.2_17606602	6	17606602	GEMMA-MLM[1245]; FarmCPU[5]	3.0-3.5	0.20	
SCM002764.2_17885307	6	17885307	GEMMA-MLM[125]; FarmCPU[12]	3.0-3.4	0.05	
SCM002767.2_3253388	9	3253388	GEMMA-MLM[12]	3.1-3.3	0.09	
SCM002767.2_5277679	9	5277679	GEMMA-MLM[145]; FarmCPU[145]	3.1-3.4	0.07	
SCM002767.2_5862687	9	5862687	GEMMA-MLM[14]; FarmCPU[1]	3.0-3.5	0.08	
SCM002767.2_38044090	9	38044090	GEMMA-MLM[125]; FarmCPU[125]	3.0-5.3	0.11	Roy et al. (2021) [SCM002767.2_37664281 (37.66 Mb); SCM002767.2_37671479 (37.67 Mb)]
SCM002767.2_38052182	9	38052182	GEMMA-MLM[25]; FarmCPU[25]	3.0-3.4	0.10	Roy et al. (2021) [SCM002767.2_37664281 (37.66 Mb); SCM002767.2_37671479 (37.67 Mb)]
SCM002767.2_38061266	9	38061266	GEMMA-MLM[125]; FarmCPU[125]	3.0-3.4	0.15	Roy et al. (2021) [SCM002767.2_37664281 (37.66 Mb); SCM002767.2_37671479 (37.67 Mb)]

Table A.19. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected in 152 semi-winter and winter populations by at least two or more traits associated with sclerotinia stem rot resistance by GWA studies (continued)

SNP	Chr[a]	Position	GWA methods and Traits[b]	LOD[c]	MAF	Previously Detected MTAs[d]
SCM002767.2_38082926	9	38082926	GEMMA-MLM[1235]; FarmCPU[1235]	3.1-3.7	0.13	Roy et al. (2021) [SCM002767.2_37664281 (37.66 Mb); SCM002767.2_37671479 (37.67 Mb)]
SCM002767.2_38119743	9	38119743	GEMMA-MLM[12]; FarmCPU[1]	3.0-3.3	0.09	Roy et al. (2021) [SCM002767.2_37664281 (37.66 Mb); SCM002767.2_37671479 (37.67 Mb)]
SCM002767.2_38774352	9	38774352	GEMMA-MLM[12345]; FarmCPU[235]	3.0-3.3	0.10	Roy et al. (2021) [SCM002767.2_39128782 (39.13 Mb)]
SCM002767.2_41958710	9	41958710	GEMMA-MLM[14]	3.1-3.3	0.10	
SCM002767.2_41990070	9	41990070	GEMMA-MLM[145]; FarmCPU[145]	3.0-3.4	0.08	
SCM002768.2_9952251	10	9952251	GEMMA-MLM[14]; FarmCPU[14]	3.0-3.9	0.12	
SCM002768.2_10253247	10	10253247	GEMMA-MLM[235]	3.1-3.4	0.22	
SCM002768.2_14218999	10	14218999	GEMMA-MLM[14]	3.1-3.3	0.50	
SCM002769.2_8989138	11	8989138	GEMMA-MLM[1245]; FarmCPU[1245]	3.2-3.6	0.16	
SCM002769.2_43565693	11	43565693	GEMMA-MLM[124]; FarmCPU[124]	3.0-3.9	0.05	

Table A.19. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected in 152 semi-winter and winter populations by at least two or more traits associated with sclerotinia stem rot resistance by GWA studies (continued)

SNP	Chr[a]	Position	GWA methods and Traits[b]	LOD[c]	MAF	Previously Detected MTAs[d]
SCM002769.2_46229694	11	46229694	GEMMA-MLM[145]; FarmCPU[45]	3.0-3.4	0.04	Roy et al. (2021) [SCM002769.2_46192098 (46.19 Mb)]
SCM002769.2_47724348	11	47724348	GEMMA-MLM[124]; FarmCPU[14]	3.0-3.9	0.37	
SCM002770.2_66428152	12	66428152	GEMMA-MLM[14]; FarmCPU[14]	3.0-3.8	0.13	
SCM002771.2_5548449	13	5548449	GEMMA-MLM[12]; FarmCPU[12]	3.1-3.9	0.05	
SCM002771.2_30920466	13	30920466	GEMMA-MLM[1245]; FarmCPU[1245]	3.0-4.0	0.35	Qasim et al. (2020) [SRC3b (22.22-30.60 Mb), SRC3c (30.60-47.86 Mb)]
SCM002771.2_38591806	13	38591806	GEMMA-MLM[14]; FarmCPU[14]	3.1-3.3	0.04	
SCM002771.2_44393183	13	44393183	GEMMA-MLM[34]; FarmCPU[34]	3.1-3.4	0.13	
SCM002772.2_7708586	14	7708586	GEMMA-MLM[1345]; FarmCPU[1345]	3.0-5.1	0.09	
SCM002772.2_67533692	14	67533692	GEMMA-MLM[145]	3.0-3.3	0.05	
SCM002773.2_5859014	15	5859014	GEMMA-MLM[1245]; FarmCPU[1245]	3.7-5.0	0.09	
SCM002773.2_11017178	15	11017178	GEMMA-MLM[125]; FarmCPU[125]	3.2-3.6	0.05	
SCM002773.2_17211900	15	17211900	GEMMA-MLM[1245]; FarmCPU[1245]	3.1-4.1	0.04	
SCM002773.2_29962917	15	29962917	GEMMA-MLM[125]; FarmCPU[125]	3.1-3.8	0.15	Roy et al. (2021) [SCM002773.2_29580386 (29.58 Mb)]
SCM002773.2_43256138	15	43256138	GEMMA-MLM[14]; FarmCPU[14]	3.0-3.3	0.06	

Table A.19. List of significant single-nucleotide polymorphisms (SNPs) simultaneously detected in 152 semi-winter and winter populations by at least two or more traits associated with sclerotinia stem rot resistance by GWA studies (continued)

SNP	Chr[a]	Position	GWA methods and Traits[b]	LOD[c]	MAF	Previously Detected MTAs[d]
SCM002774.2_20774649	16	20774649	GEMMA-MLM[14]; FarmCPU[14]	3.2-3.7	0.10	
SCM002774.2_32559985	16	32559985	GEMMA-MLM[14]; FarmCPU[145]	3.0-3.3	0.04	Wu et al. (2013) [SRC6-1 (30.28-34.59 Mb)]; Qasim et al. (2020) [SRC6 (32.95-34.45 Mb)]; Roy et al. (20210) [SCM002774.2_33238118 (33.24 Mb); SCM002774.2_33265642 (33.27 Mb)]
SCM002775.2_12215584	17	12215584	GEMMA-MLM[14]; FarmCPU[14]	3.4-3.7	0.10	SCM002775.2_12215584
SCM002775.2_41663703	17	41663703	GEMMA-MLM[124]; FarmCPU[14]	3.0-3.4	0.40	SCM002775.2_41663703
SCM002776.2_16868604	18	16868604	GEMMA-MLM[45]; FarmCPU[4]	3.0-3.7	0.08	SCM002776.2_16868604
SCM002777.2_32367681	19	32367681	GEMMA-MLM[1245]; FarmCPU[1245]	3.0-3.9	0.07	SCM002777.2_32367681

Chr[a]: *Brassica napus* chromosome

GWA methods and Traits[b]: Methods, GEMMA-MLM and FarmCPU; Traits: Superscript 1, 2, 3, 4, and 5 represents lesion length (LL) measured at 7 days post inoculation (dpi), lesion width (LW) at 7 dpi, plant mortality (PM) at 15 dpi, lesion lengths area under disease progress curve (LL_AUDPC) calculated using 7 time points reading, and lesion widths AUDPC (LW_AUDPC) calculated using 7 time points reading, respectively.

LOD[c]: The highest and lowest $-\log_{10}$ (P) value resulted from the various studied traits with different GWA models

Previously reported MTAs[d]: previously reported marker-trait-associations (MTAs) along with their physical positions from various QTL and GWA-related studies which overlapped or in close proximity with the genomic regions in this study

Table A.20. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for spring ecotypes rapeseed/canola genotypes

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002760.2_20267096	2	20267096	8.04	LOC106381520	YTH domain-containing family protein 2-like	innate immune response Source: UniProtKB-KW
SCM002760.2_20267096	2	20267096	-16.45	LOC106381519	metacaspase-5	positive regulation of programmed cell death response to fungus Source: UniProtKB; regulation of systemic acquired resistance Source: TAIR; regulation of salicylic acid biosynthetic process Source: UniProtKB
SCM002760.2_31650036	2	31650036	39.64	LOC111207571	calmodulin-binding protein 60 G-like	
SCM002760.2_31650036	2	31650036	13.95	LOC111207576	lon protease homolog 1, mitochondrial	cellular response to oxidative stress Source: UniProtKB
SCM002760.2_31650036	2	31650036	-10.43	LOC111197937	BON1-associated protein 2-like	defense response Source: UniProtKB-KW
SCM002761.2_1259602	3	1259602	-30.57	LOC106429356	protein BONZAI 2	Negative regulator of cell death and defense responses; defense response Source: UniProtKB-KW
SCM002761.2_1259602	3	1259602	-39.37	LOC106441369	ethylene-responsive transcription factor ERF115	ethylene-activated signaling pathway Source: UniProtKB-KW

Table A.20. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for spring ecotypes rapeseed/canola genotypes (continued)

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002761.2_33351214	3	33351214	-49.66	LOC111214229	ethylene-responsive transcription factor ERF104-like	defense response to fungus Source: TAIR; ethylene-activated signaling pathway Source: UniProtKB-KW
SCM002761.2_44267358	3	44267358	-36.65	LOC106345590	putative Peroxidase 48	response to wounding, pathogen attack and oxidative stress Source: UniProtKB-KW
SCM002761.2_44267358	3	44267358	24.18	LOC106441054	UDP-glucuronate 4-epimerase 1-like	defense response to fungus Source: TAIR
SCM002763.2_7201665	5	7201665	-19.21	LOC106451045	transcription factor AS1-like	defense response to fungus Source: TAIR
SCM002763.2_10442725	5	10442725	-20.00	LOC106454882	glycine-rich protein-like	defense response Source: UniProtKB-KW; response to biotic stimulus
SCM002763.2_10442725	5	10442725	-20.00	LOC106454882	glycine-rich protein-like	defense response Source: UniProtKB-KW; response to biotic stimulus
SCM002763.2_10442725	5	10442725	-32.54	LOC106451406	oxygen-evolving enhancer protein 2-1, chloroplastic	defense response to bacterium Source: TAIR
SCM002763.2_31228663	5	31228663	8.83	LOC106454400	aspartic proteinase CDR1-like	defense response to bacterium Source: TAIR; regulation of salicylic acid metabolic process Source: TAIR

Table A.20. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for spring ecotypes rapeseed/canola genotypes (continued)

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002763.2_31228663	5	31228663	-11.21	LOC111197868	probable WRKY transcription factor 58	defense response to bacterium
SCM002764.2_27014783	6	27014783	33.36	LOC106407392	F-box/LRR-repeat/kelch-repeat protein At1g09650-like	response to chitin
SCM002767.2_18969125	9	18969125	25.37	LOC106450125	pathogenesis-related protein 1	defense response Source: TAIR; systemic acquired resistance Source: TAIR programmed cell death Source: TAIR
SCM002767.2_18969125	9	18969125	10.25	LOC106447109	hexokinase-1	
SCM002767.2_18969125	9	18969125	-31.40	LOC106447113	S-alkyl-thiohydroximate lyase SUR1-like	defense response to fungus Source: TAIR
SCM002767.2_18969125	9	18969125	-46.15	LOC106447116	profilin-1-like	defense response Source: UniProtKB-KW
SCM002768.2_1532243	10	1532243	12.47	LOC106427016	1-aminocyclopropane-1-carboxylate oxidase homolog 2-like	defense response Source: UniProtKB-KW
SCM002768.2_1532243	10	1532243	9.37	LOC106427011	1-aminocyclopropane-1-carboxylate oxidase homolog 2-like	defense response Source: UniProtKB-KW

Table A.20. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for spring ecotypes rapeseed/canola genotypes (continued)

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002769.2_13914840	11	13914840	12.40	LOC106423878	subtilisin-like protease SBT1.3	serine-type endopeptidase activity Source: InterPro
SCM002769.2_2578220	11	2578220	28.92	LOC106354285	phospholipase D delta-like	programmed cell death Source: TAIR
SCM002769.2_2578220	11	2578220	21.20	LOC106351615	phospholipase D delta	programmed cell death Source: TAIR
SCM002769.2_29417842	11	29417842	-48.50	LOC106400149	protein ACCELERATED CELL DEATH 6	regulation of defense response to fungus Source: UniProtKB; cell death Source: TAIR
SCM002770.2_18672852	12	18672852	-34.75	LOC111202568	1-aminocyclopropane-1-carboxylate oxidase 5-like	defense response Source: UniProtKB-KW
SCM002771.2_1951273	13	1951273	9.49	LOC106448897	probable serine/threonine-protein kinase PBL11	May be involved in plant defense signaling Source: UniProtKB-KW
SCM002771.2_1951273	13	1951273	-24.51	LOC106448766	E3 ubiquitin-protein ligase PRT6-like	defense response to fungus Source: TAIR
SCM002772.2_69471400	14	69471400	24.53	LOC106395637	probable L-type lectin-domain containing receptor kinase V.3	defense response Source: GO_Central; defense response to oomycetes Source: GO_Central

Table A.20. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for spring ecotypes rapeseed/canola genotypes (continued)

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002772.2_69471400	14	69471400	-1.64	LOC106394184	probable polygalacturonase At2g43860	cell wall organization Source: UniProtKB-KW
SCM002772.2_69471400	14	69471400	-29.05	LOC106391815	probable thiocyanate methyltransferase 2	Involved in glucosinolate metabolism and defense against phytopathogens Source: UniProtKB-KW
SCM002776.2_8341541	18	8341541	-36.09	LOC106414500	peroxidase 7-like	response to oxidative stress Source: InterPro
SCM002776.2_12072563	18	12072563	16.23	LOC106363590	cystine lyase CORI3-like	response to jasmonic acid, response to wounding Source: TAIR

Chr[a]: *Brassica napus* chromosome

Marker distance from gene (kb)[b], Negative values = genes are downstream of the markers, positive values= genes are upstream of the marker

Table A.21. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for semi-winter and winter ecotypes rapeseed/canola genotypes

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002759.2_7025054	1	7025054	49.94	LOC106437839	disease resistance protein RRS1-like	defense response Source: UniProtKB-KW
SCM002759.2_7025054	1	7025054	13.99	LOC106358002	heat stress transcription factor A-4a-like	response to chitin Source: TAIR; response to reactive oxygen species Source: TAIR
SCM002759.2_7025054	1	7025054	3.98	LOC106358009	heat stress transcription factor A-4a-like	response to chitin Source: TAIR; response to reactive oxygen species Source: TAIR
SCM002759.2_7025054	1	7025054	-25.96	LOC106437694	regulatory protein NPR4	defense response to fungus; systemic acquired resistance, salicylic acid mediated signaling pathway; regulation of jasmonic acid mediated signaling pathway Source: UniProtKB
SCM002759.2_7025054	1	7025054	-46.89	LOC106373483	fe(2+) transport protein 1-like	response to bacterium Source: TAIR
SCM002759.2_22108854	1	22108854	7.12	BNAANNG08350D	senescence/dehydration-associated protein At4g35985, chloroplastic	response to oomycetes Source: UniProtKB

Table A.21. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for semi-winter and winter ecotypes rapeseed/canola genotypes (continued)

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002759.2_22108854	1	22108854	-5.34	LOC106451503	protein GIGAS CELL1-like	defense response Source: UniProtKB-KW
SCM002759.2_22108854	1	22108854	-22.44	LOC106451458	la-related protein 1C	response to jasmonic acid Source: UniProtKB; response to salicylic acid Source: UniProtKB
SCM002760.2_3833184	2	3833184	45.53	LOC106416109	L-type lectin-domain containing receptor kinase IX.1-like	defense response to oomycetes Source: UniProtKB; positive regulation of cell death Source: UniProtKB
SCM002760.2_3833184	2	3833184	53.81	LOC106383204	AP2-like ethylene-responsive transcription factor AIL6	defense response to fungus Source: UniProtKB
SCM002760.2_3833184	2	3833184	49.52	LOC106383209	receptor-like cytosolic serine/threonine-protein kinase RBK1	defense response to fungus Source: UniProtKB
SCM002760.2_3833184	2	3833184	31.16	LOC106383222	HBS1-like protein	signal transduction
SCM002760.2_3833184	2	3833184	10.20	LOC106383257	histidine kinase 5	defense response Source: UniProtKB-KW; ethylene-activated signaling pathway Source: UniProtKB-KW

Table A.21. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for semi-winter and winter ecotypes rapeseed/canola genotypes (continued)

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002760.2_3833184	2	3833184	-23.19	LOC111208285	NDR1/HIN1-like protein 3	response to salicylic acid; response to wounding Source: UniProtKB
SCM002761.2_27270949	3	27270949	45.11	LOC106424437	pathogenesis-related protein 1	defense response Source: UniProtKB-KW
SCM002761.2_27270949	3	27270949	40.89	LOC106424421	methionine aminotransferase BCAT4	glucosinolate biosynthetic process Source: TAIR
SCM002762.2_20793967	4	20793967	39.93	LOC106447554	calcium-transporting ATPase 4, plasma membrane-type	negative regulation of programmed cell death Source: TAIR
SCM002762.2_20793967	4	20793967	-33.42	LOC106447562	cytochrome P450 83A1-like	glucosinolate biosynthetic process Source: TAIR
SCM002762.2_20793967	4	20793967	-42.09	LOC106450307	dihomomethionine N-hydroxylase-like	glucosinolate biosynthetic process Source: TAIR
SCM002762.2_22210032	4	22210032	24.60	LOC111215168	putative defensin-like protein 27	defense response to fungus Source: UniProtKB-KW
SCM002762.2_22210032	4	22210032	-6.31	LOC111215172	eukaryotic translation initiation factor 4E-1-like	response to virus Source: TAIR

Table A.21. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for semi-winter and winter ecotypes rapeseed/canola genotypes (continued)

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002763.2_8993185	5	8993185	25.93	LOC106454802	UV-B-induced protein At3g17800, chloroplastic-like	response to wounding Source: UniProtKB
SCM002763.2_8993185	5	8993185	-21.30	LOC106451268	ninja-family protein AFP3-like	signal transduction
SCM002764.2_17606602	6	17606602	15.09	LOC106351681	putative respiratory burst oxidase homolog protein J	peroxidase activity Source: UniProtKB-KW
SCM002764.2_17606602	6	17606602	-49.25	LOC106347512	mitogen-activated protein kinase 3	response to chitin Source: TAIR
SCM002764.2_17885307	6	17885307	38.24	LOC106347498	classical arabinogalactan protein 9	Programmed cell death source: UniProtKB-KW
SCM002767.2_5277679	9	5277679	-19.10	LOC106432965	myrosinase-like	glucosinolate catabolic process Source: TAIR; response to insect Source: TAIR
SCM002767.2_5277679	9	5277679	-45.40	LOC106432973	myrosinase-like	glucosinolate catabolic process Source: TAIR; response to insect Source: TAIR
SCM002767.2_38044090	9	38044090	39.81	LOC106365141	peroxidase 18	response to oxidative stress Source: InterPro
SCM002767.2_41990070	9	41990070	30.29	LOC106367041	5'-3' exonuclease	innate immune response Source: UniProtKB-KW

Table A.21. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for semi-winter and winter ecotypes rapeseed/canola genotypes (continued)

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002769.2_43565693	11	43565693	-29.28	LOC106374862	mitogen-activated protein kinase kinase 5-like	innate immune response Source: UniProtKB-KW
SCM002769.2_46229694	11	46229694	-36.12	LOC106435207	protein TIFY 6B-like	regulation of defense response; jasmonic acid mediated signaling pathway Source: TAIR
SCM002769.2_47724348	11	47724348	-6.87	LOC111201803	probable leucine-rich repeat receptor-like serine/threonine-protein kinase At3g14840	jasmonic acid and ethylene-dependent systemic resistance Source: TAIR; regulation of innate immune response Source: TAIR
SCM002769.2_47724348	11	47724348	-22.16	LOC111202397	probable leucine-rich repeat receptor-like serine/threonine-protein kinase At3g14840	jasmonic acid and ethylene-dependent systemic resistance Source: TAIR
SCM002772.2_7708586	14	7708586	16.70	LOC106450940	rop guanine nucleotide exchange factor 4	defense response to fungus Source: TAIR

Table A.21. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for semi-winter and winter ecotypes rapeseed/canola genotypes (continued)

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002772.2_67533692	14	67533692	32.74	LOC106402216	protein MICRORCHIDIA 6-like	defense response Source: UniProtKB-KW; positive regulation of defense response to oomycetes Source: UniProtKB
SCM002772.2_67533692	14	67533692	5.64	LOC106401000	two-component response regulator ARR7	response to chitin Source: TAIR
SCM002772.2_67533692	14	67533692	-36.94	LOC106400858	germin-like protein subfamily T member 2	May play a role in plant defense. Source: UniProtKB
SCM002773.2_29962917	15	29962917	-47.29	LOC106401967	wall-associated receptor kinase 2-like	response to salicylic acid Source: TAIR
SCM002773.2_29962917	15	29962917	-10.61	LOC106397552	wall-associated receptor kinase 2	response to salicylic acid Source: TAIR
SCM002773.2_43256138	15	43256138	-3.73	LOC106345645	protein RALF-like 22	calcium-mediated signaling Source: UniProtKB; cell-cell signaling Source: TAIR
SCM002773.2_43256138	15	43256138	-28.26	LOC106453048	acyl-CoA-binding domain-containing protein 4	response to jasmonic acid Source: UniProtKB

Table A.21. The significantly associated loci and candidate genes for different sclerotinia stem rot resistance traits for semi-winter and winter ecotypes rapeseed/canola genotypes (continued)

Significant markers	Chr[a]	Marker position	Distance (kb)[b]	Gene symbol and ID	Gene description	GO biological Function
SCM002774.2_20774649	16	20774649	-49.29	LOC106454140	probable LRR receptor-like serine/threonine-protein kinase At4g36180	protein serine kinase activity Source: UniProtKB-EC
SCM002774.2_20774649	16	20774649	-17.35	LOC106345455	transcription factor MYB27	response to chitin Source: TAIR
SCM002776.2_16868604	18	16868604	6.44	LOC106453139	universal stress protein PHOS34	response to molecule of fungal origin Source: TAIR
SCM002776.2_16868604	18	16868604	-0.50	LOC106453135	aspartyl protease AED3	regulation of programmed cell death; systemic acquired resistance Source: TAIR

Chr[a]: *Brassica napus* chromosome

Marker distance from gene (kb)[b], Negative values = genes are downstream of the markers, positive values= genes are upstream of the marker.

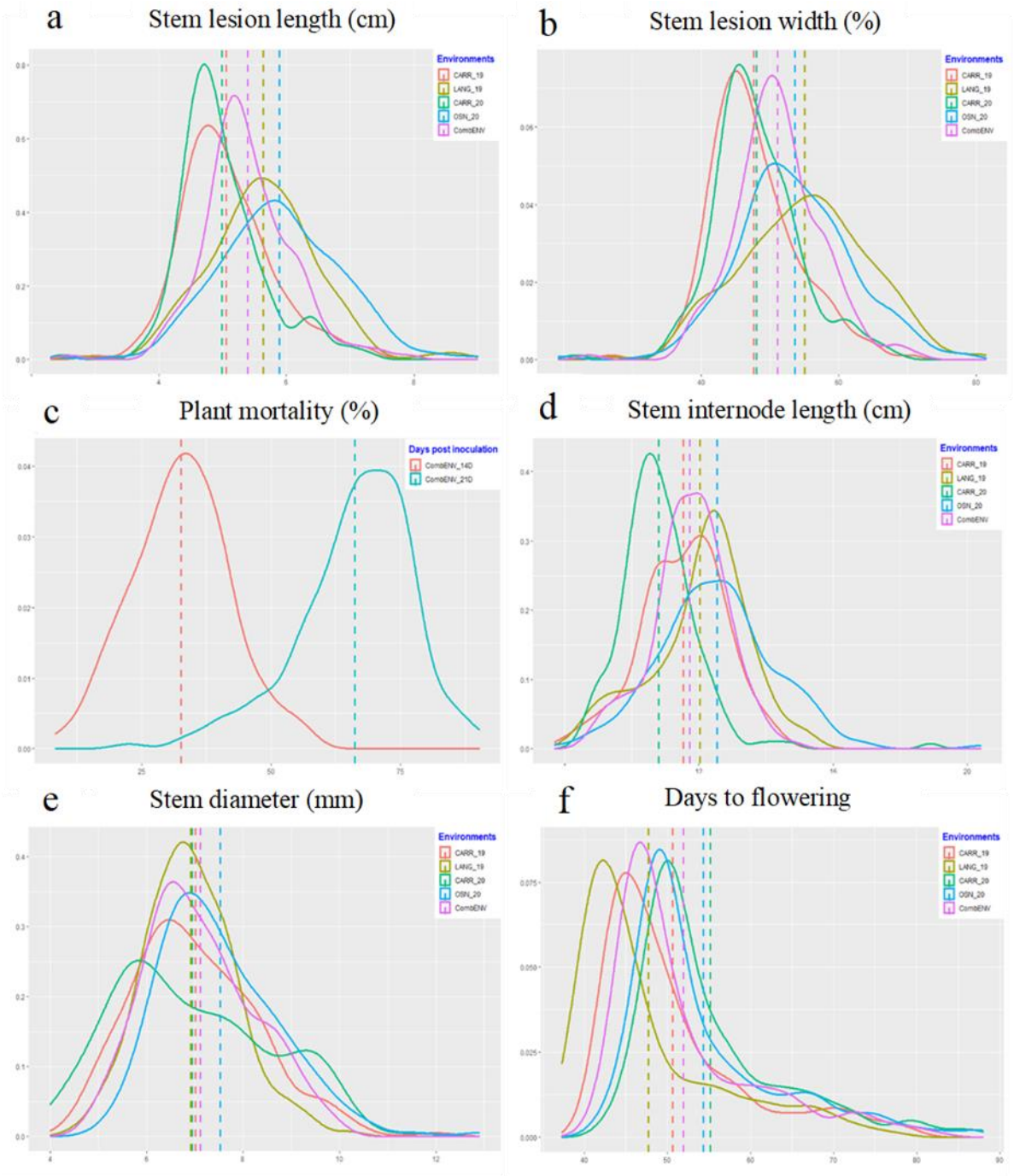

Figure A.1. Phenotypic distribution of different phenotypic traits BLUE values for sclerotinia stem rot disease. Traits are stem lesion length (a), stem lesion width (b), plant mortality (c), stem internode length (d), stem diameter (e), and days to flowering (f). Four environments: Carrington in 2019 (CARR_19), 2020 (CARR_20), Langdon in 2019 (LANG_19), Osnabrock in 2020 (OSN_20), and combined best linear unbiased estimates (CombENV_BLUEs) across all four environments. Mean values are represented by the vertical dashed line in the density distribution plot.

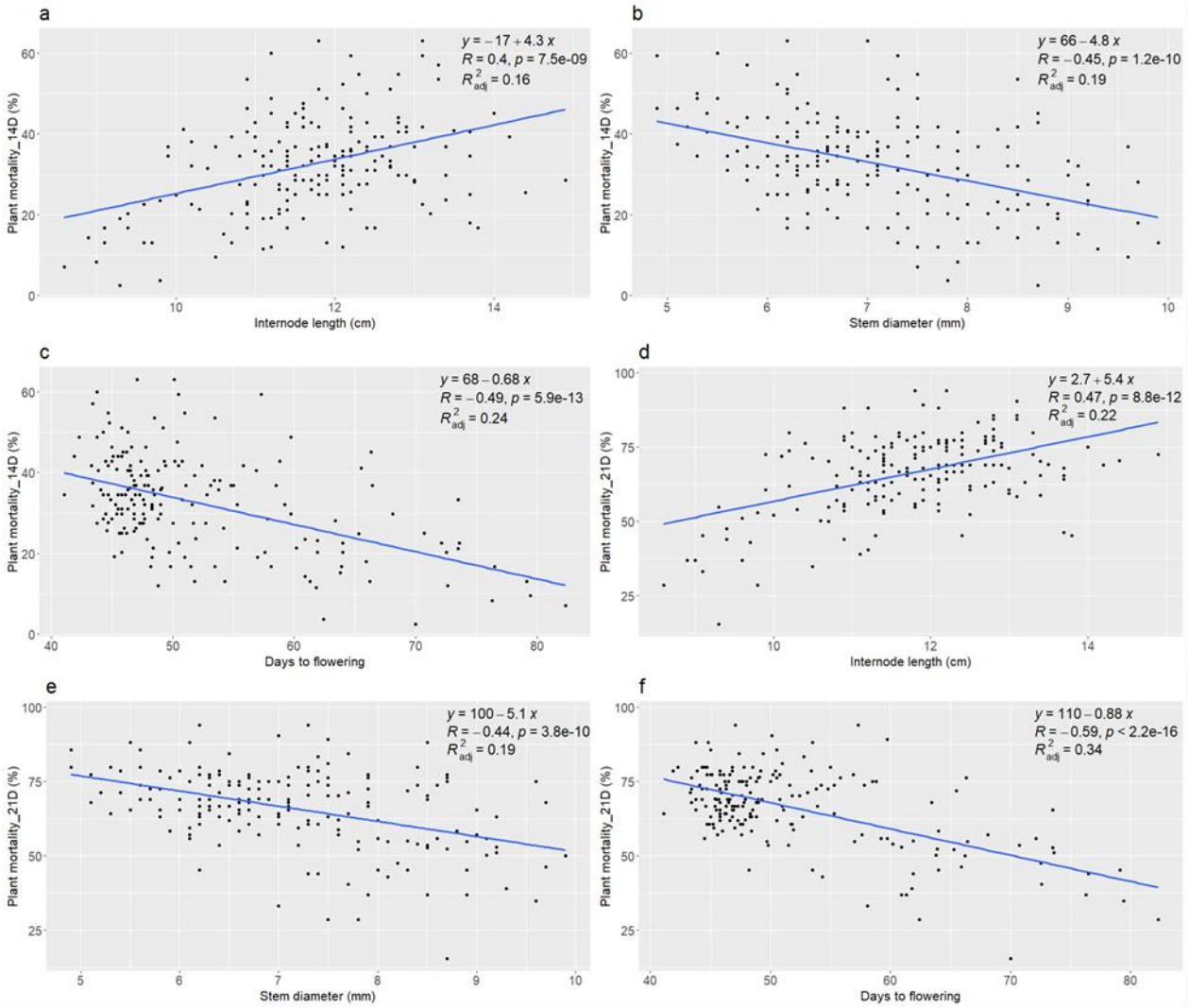

Figure A.2. Regression analysis of sclerotinia stem rot resistance in respect to plant mortality at 14 days post inoculation (dpi), plant mortality at 21 dpi with stem internode length, stem diameter and days to flowering. *R* is Pearson's correlation coefficient between the two traits, R^2_{adj} is the coefficient of determination.

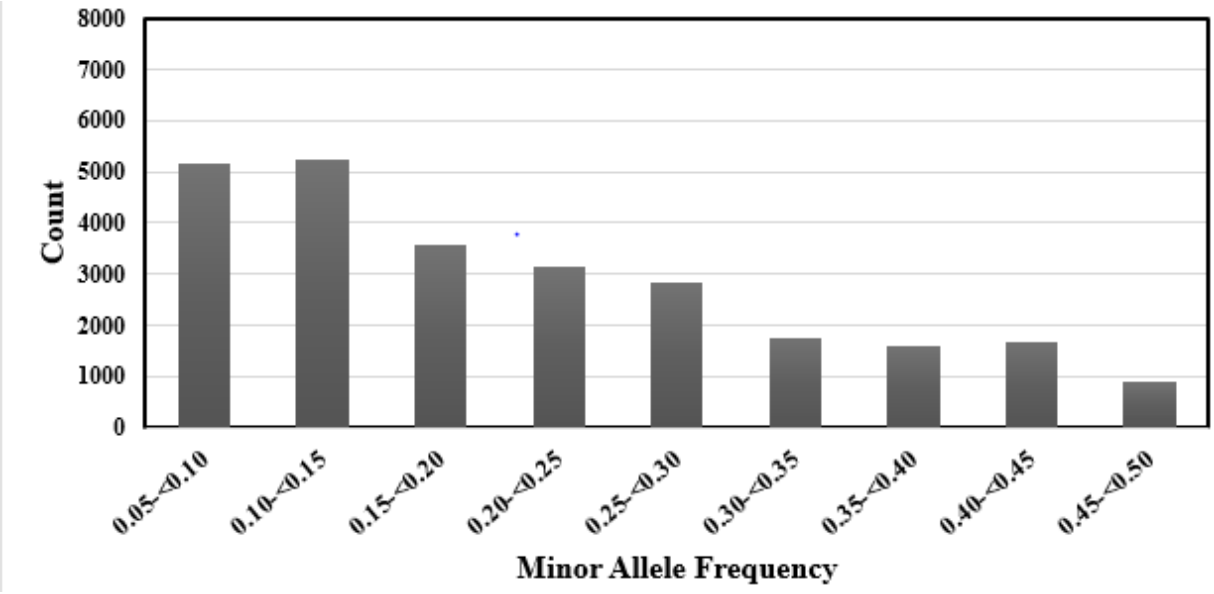

Figure A.3. Single-nucleotide polymorphism distribution with minor allele frequency in this population panel

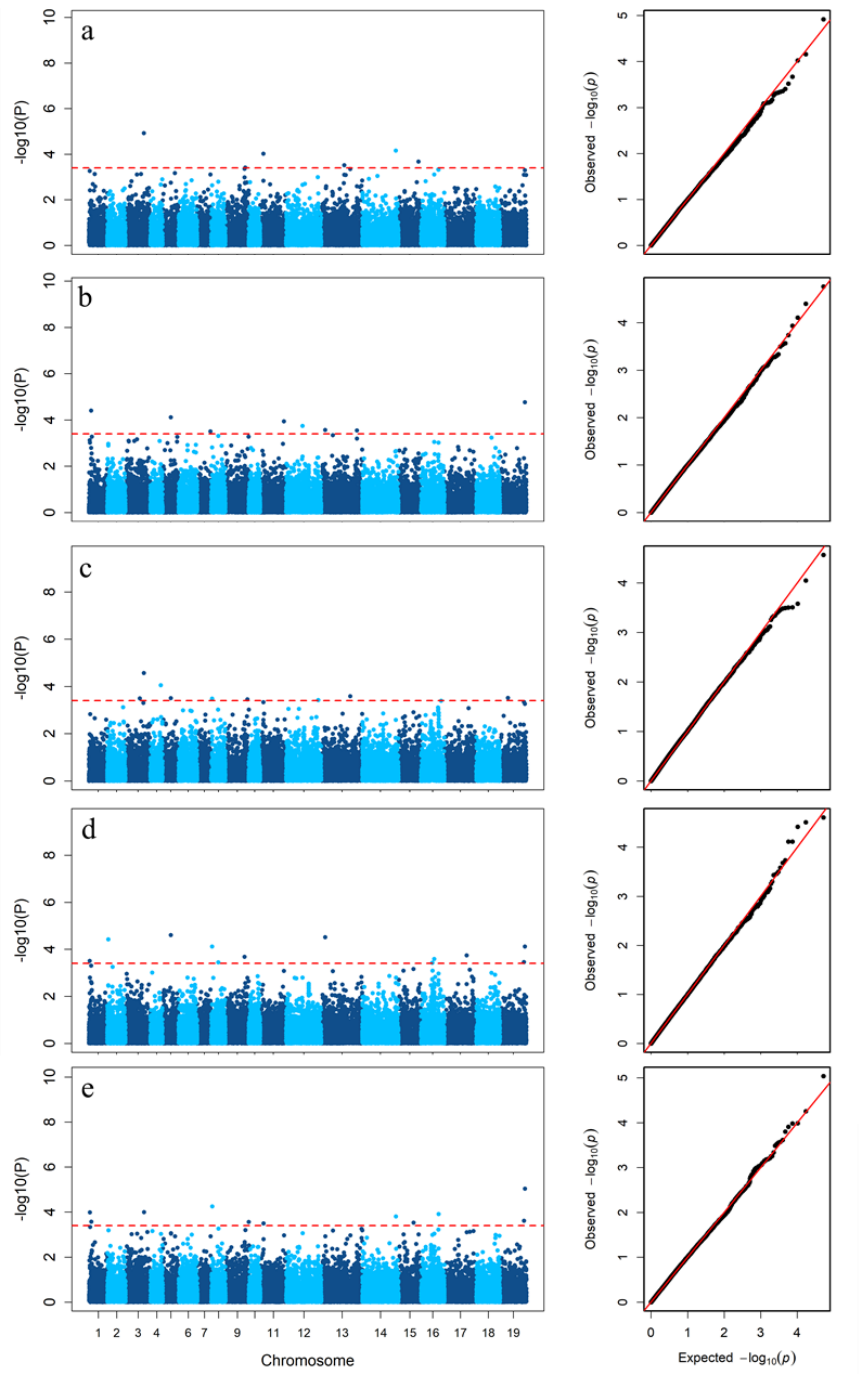

Figure A.4. Manhattan and Q-Q plots showing the results of marker-trait-associations for stem lesion length associated with sclerotinia stem rot resistance in 187 canola/rapeseed genotypes by the MLM GWA model. a) MLM, Carrington 2019; b) MLM, Langdon, 2019; c) MLM, Carrington 2020; d) MLM, Osnabrock 2020; e) MLM, combined data (CombENV), f) GEMMA-MLM, Carrington 2019. The -log10 (P) values from a genome-wide scan are plotted against positions on each of the 19 chromosomes. Discontinued horizontal lines indicate the genome-wide significance threshold.

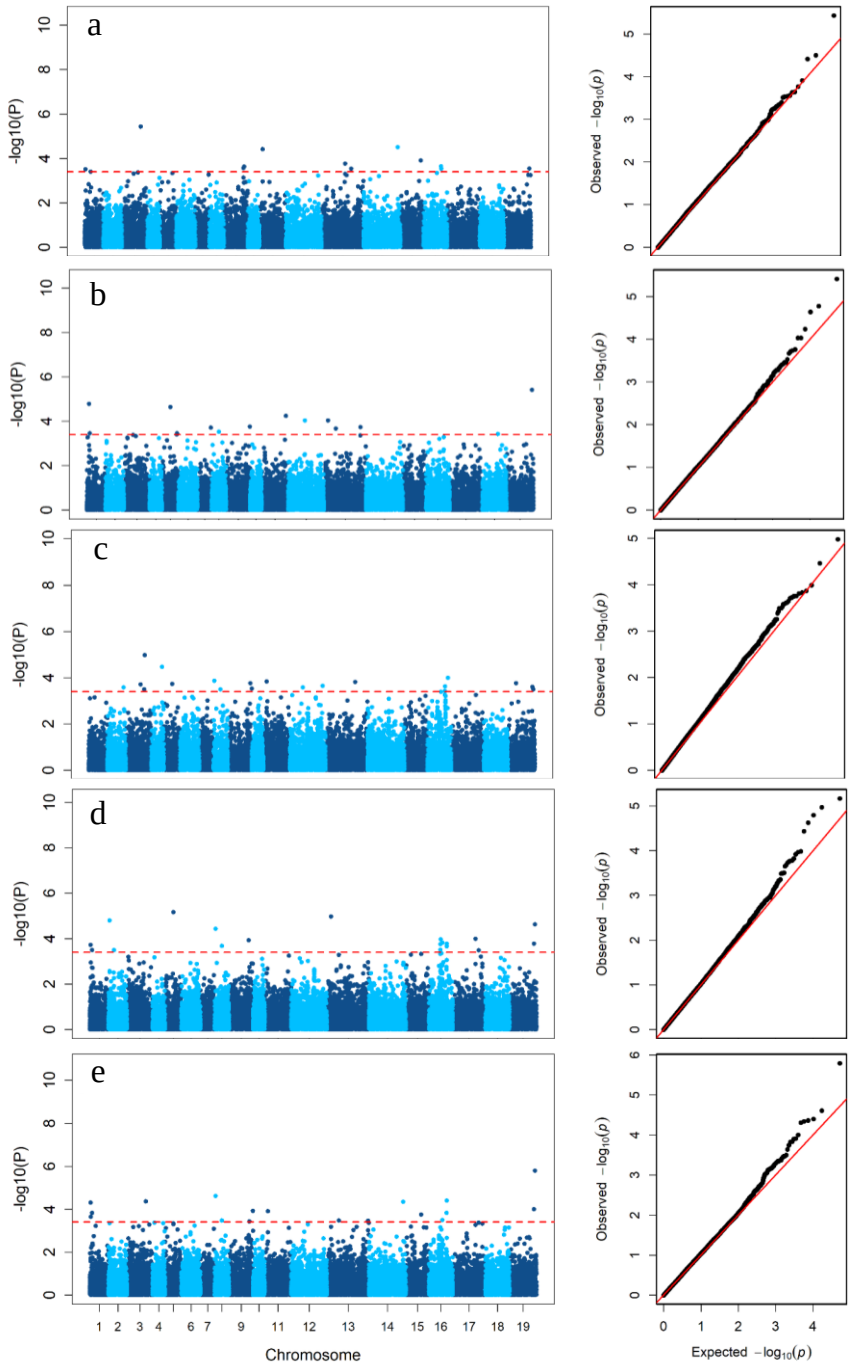

Figure A.5. Manhattan and Q-Q plots showing the results of marker-trait-associations for stem lesion length associated with sclerotinia stem rot resistance in 187 canola/rapeseed genotypes by the GEMMA-MLM GWA models. a) GEMMA-MLM, Carrington 2019; b) GEMMA-MLM, Langdon, 2019; c) GEMMA-MLM, Carrington 2020; d) GEMMA-MLM, Osnabrock 2020; e) GEMMA-MLM, combined data (CombENV). The -$\log_{10}$ (*P*) values from a genome-wide scan are plotted against positions on each of the 19 chromosomes. Discontinued horizontal lines indicate the genome-wide significance threshold.

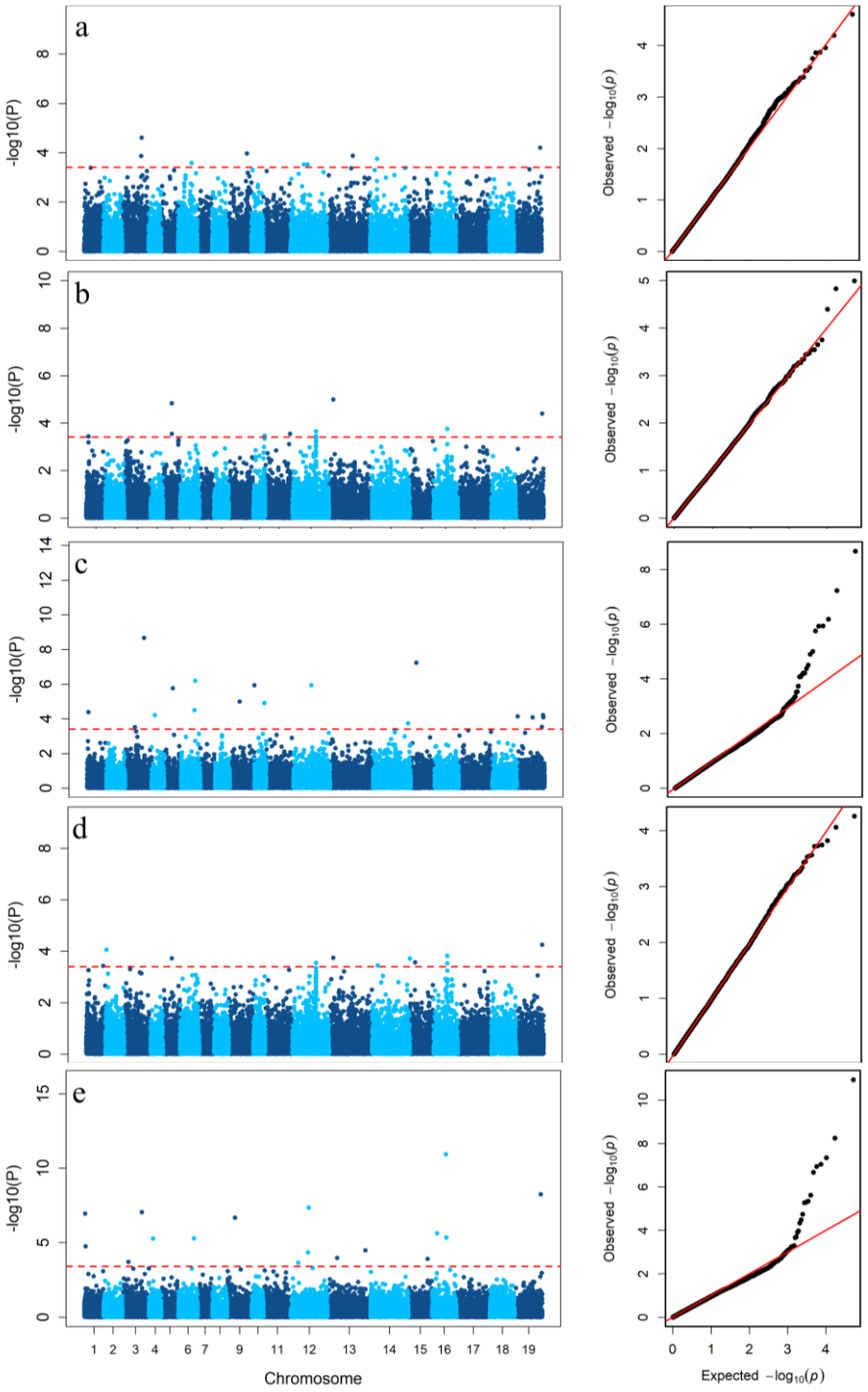

Figure A.6. Manhattan and Q-Q plots showing the results of MTAs for stem lesion width associated with SSR resistance in 187 canola/rapeseed genotypes by the FarmCPU GWA model. a) FarmCPU, Carrington 2019; b) FarmCPU, Langdon, 2019; c) FarmCPU, Carrington 2020; d) FarmCPU, Osnabrock 2020; e) FarmCPU, combined data (CombENV). The -$\log_{10}$ (*P*) values from a genome-wide scan are plotted against positions on each of the 19 chromosomes. Discontinued horizontal lines indicate the genome-wide significance threshold.

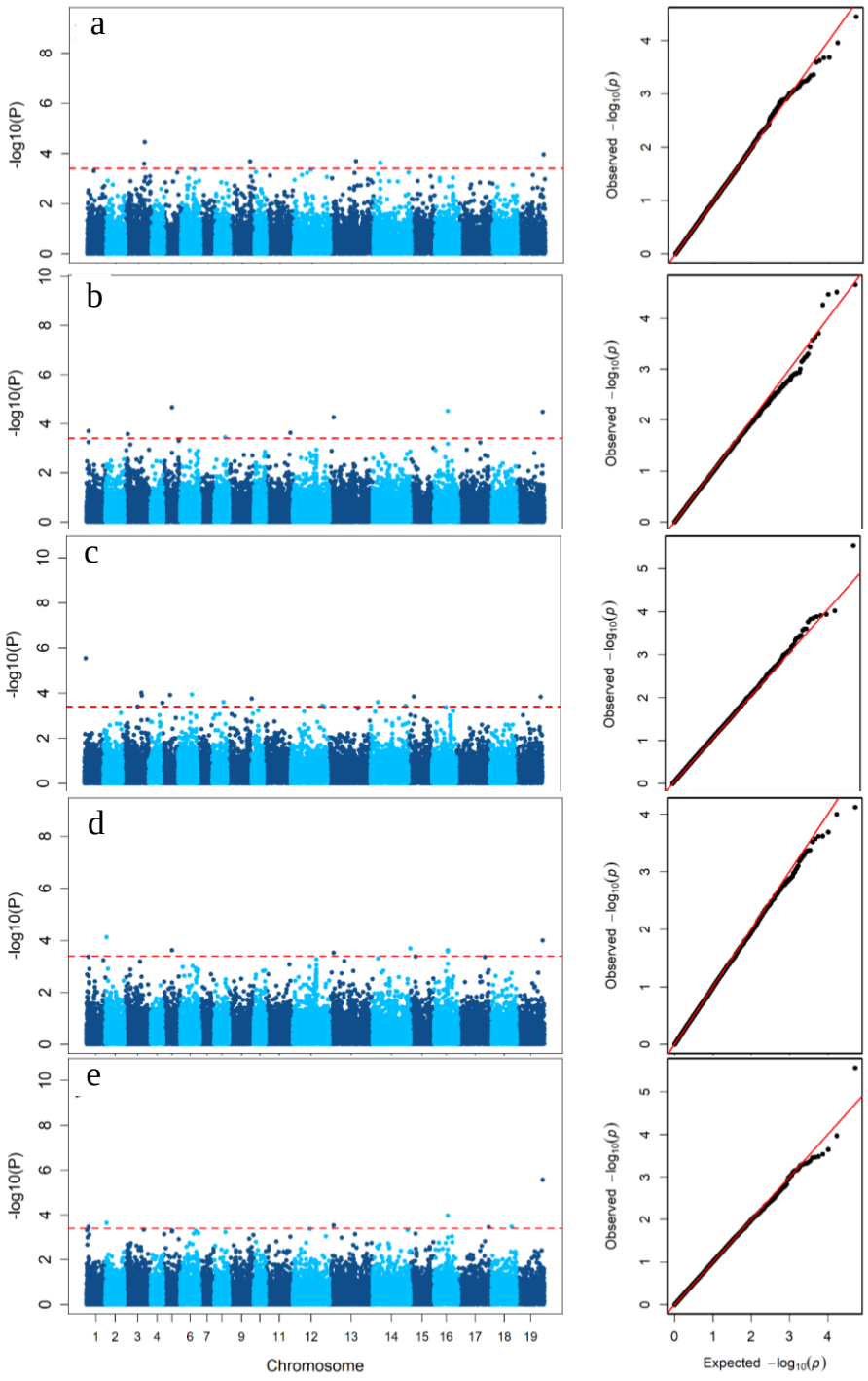

Figure A.7. Manhattan and Q-Q plots showing the results of MTAs for stem lesion width associated with SSR resistance in 187 canola/rapeseed genotypes by the MLM GWA model. a) MLM, Carrington 2019; b) MLM, Langdon, 2019; c) MLM, Carrington 2020; d) MLM, Osnabrock 2020; e) MLM, combined data (CombENV). The -$\log_{10}$ (*P*) values from a genome-wide scan are plotted against positions on each of the 19 chromosomes. Discontinued horizontal lines indicate the genome-wide significance threshold.

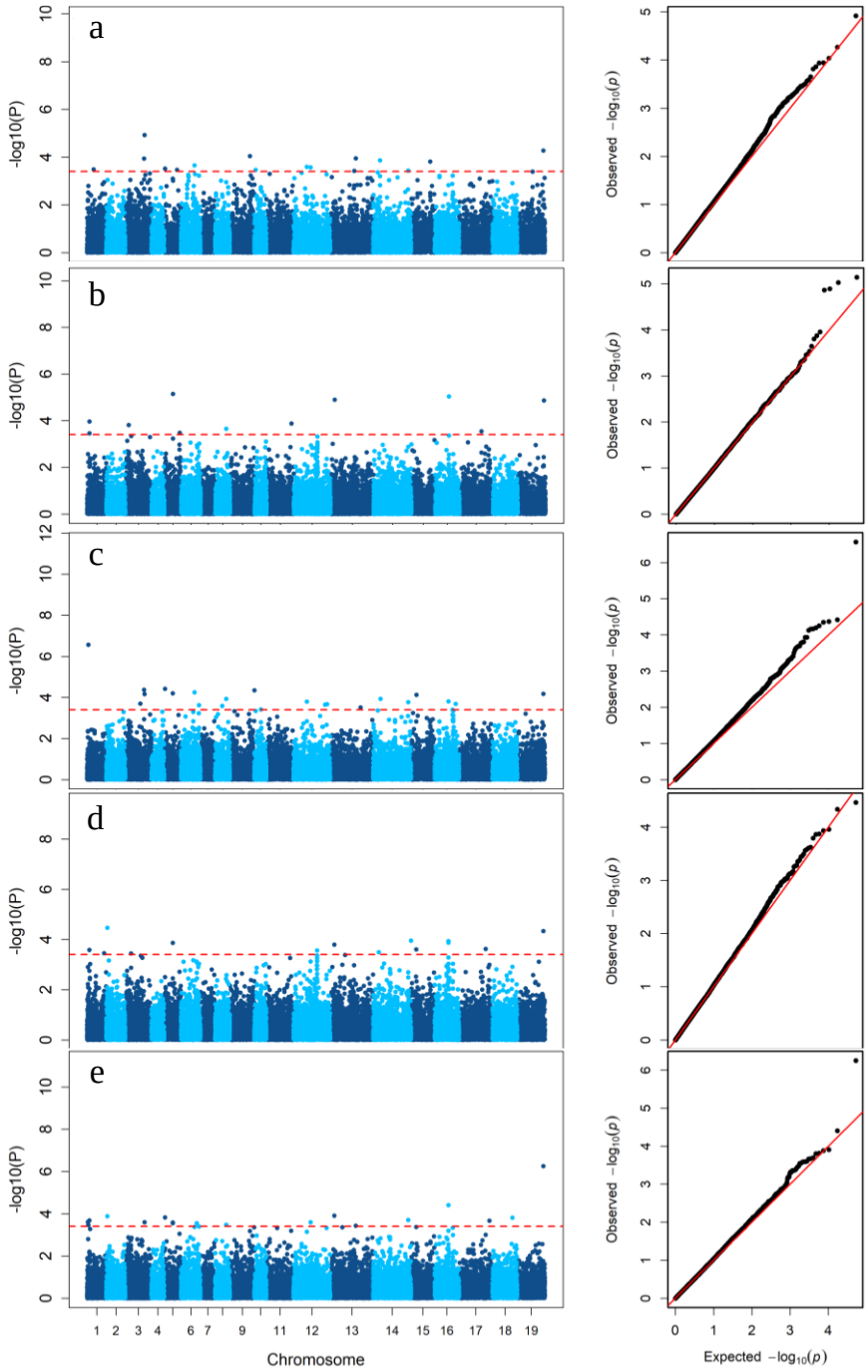

Figure A.8. Manhattan and Q-Q plots showing the results of MTAs for stem lesion width associated with SSR resistance in 187 canola/rapeseed genotypes by the GEMMA-MLM GWA model. a) GEMMA-MLM, Carrington 2019; b) GEMMA-MLM, Langdon, 2019; c) GEMMA-MLM, Carrington 2020; d) GEMMA-MLM, Osnabrock 2020; e) GEMMA-MLM, combined data (CombENV). The -$\log_{10}$ (*P*) values from a genome-wide scan are plotted against positions on each of the 19 chromosomes. Discontinued horizontal lines indicate the genome-wide significance threshold.

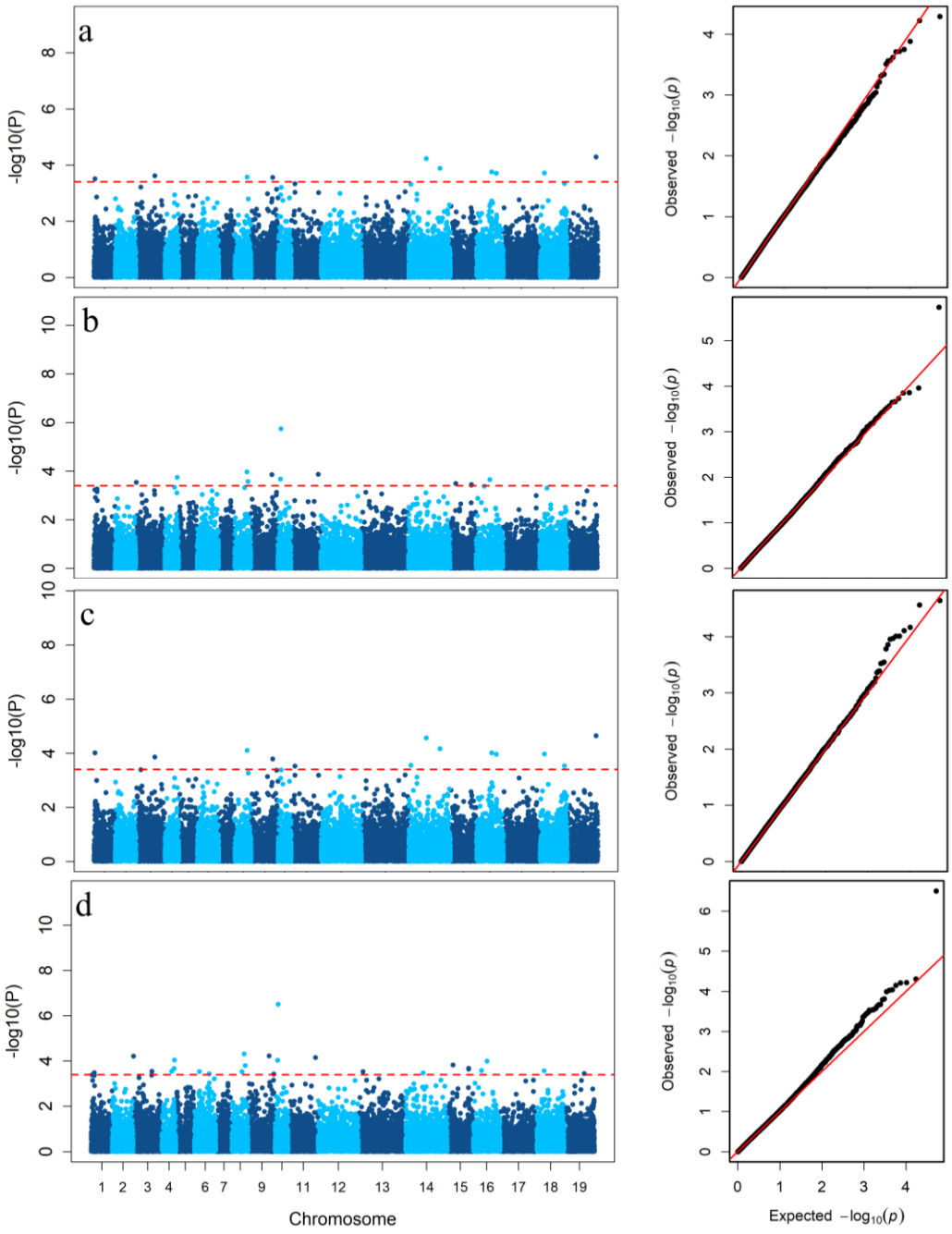

Figure A.9. Manhattan and Q-Q plots showing the results of marker-trait association for plant mortality associated with sclerotinia stem rot resistance in 187 canola/rapeseed genotypes by the MLM, and GEMMA-MLM GWA models. a) MLM, combined (CombENV) plant mortality at 14 days post inoculation (dpi); b) MLM, combined (CombENV) plant mortality at 21 dpi; c) GEMMA-MLM, combined (CombENV) plant mortality at 14 dpi; d) GEMMA-MLM, combined (CombENV) plant mortality at 21 dpi. The -$\log_{10}$ (*P*) values from a genome-wide scan are plotted against positions on each of the 19 chromosomes. Discontinued horizontal lines indicate the genome-wide significance threshold.

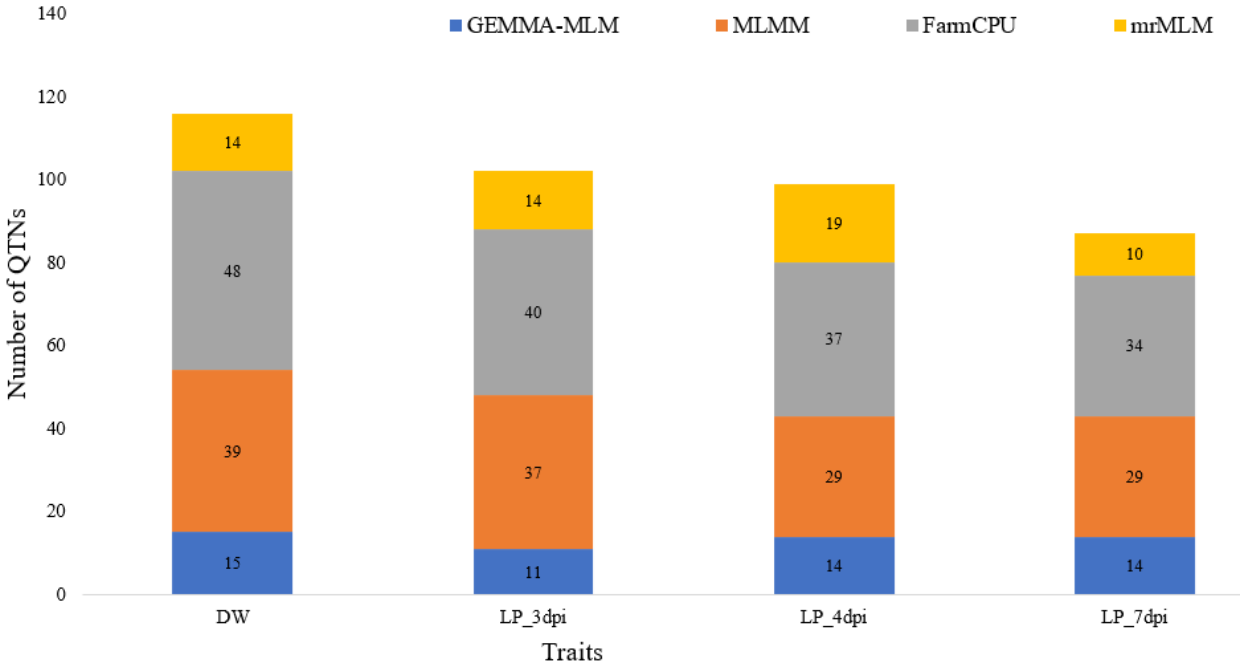

Figure A.10. Total number of quantitative trait nucleotides (QTNs) detected by the 4 models in four sclerotinia stem rot resistance traits. The X axis represent the traits (DW, days to wilt; LP_3dpi, lesion phenotypes at 3 days post inoculation; LP_4dpi, lesion phenotypes at 4 days post inoculation; LP_7dpi, lesion phenotypes at 7 days post inoculation) and Y axis represent the number of detected QTNs by each of GWA models.

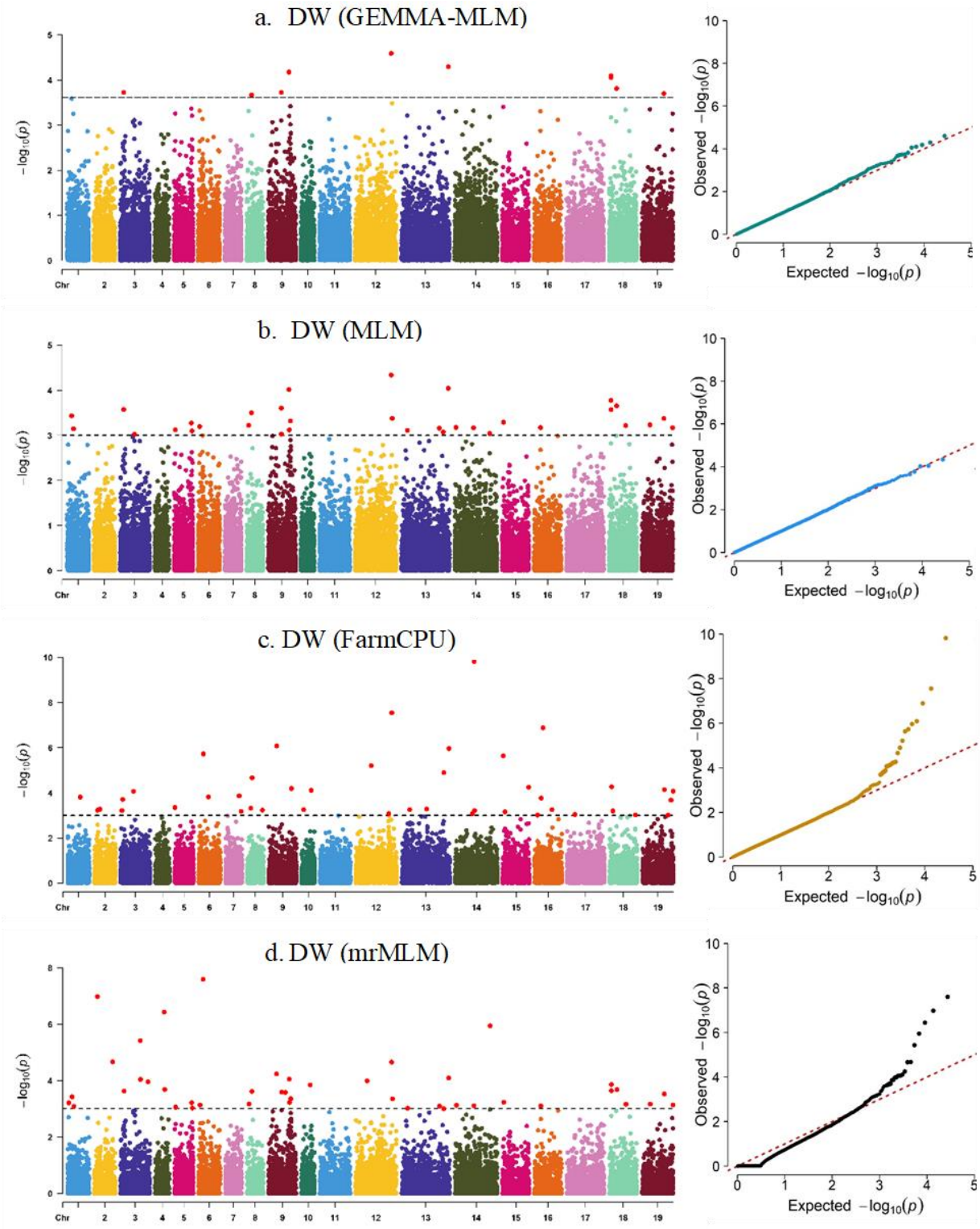

Figure A.11. Manhattan and quantile-quantile (Q-Q) plot of single-locus and multi-locus GWA models for Sclerotinia sclerotiorum resistance trait, days to wilt (DW) (a-d) in rapeseed/canola.

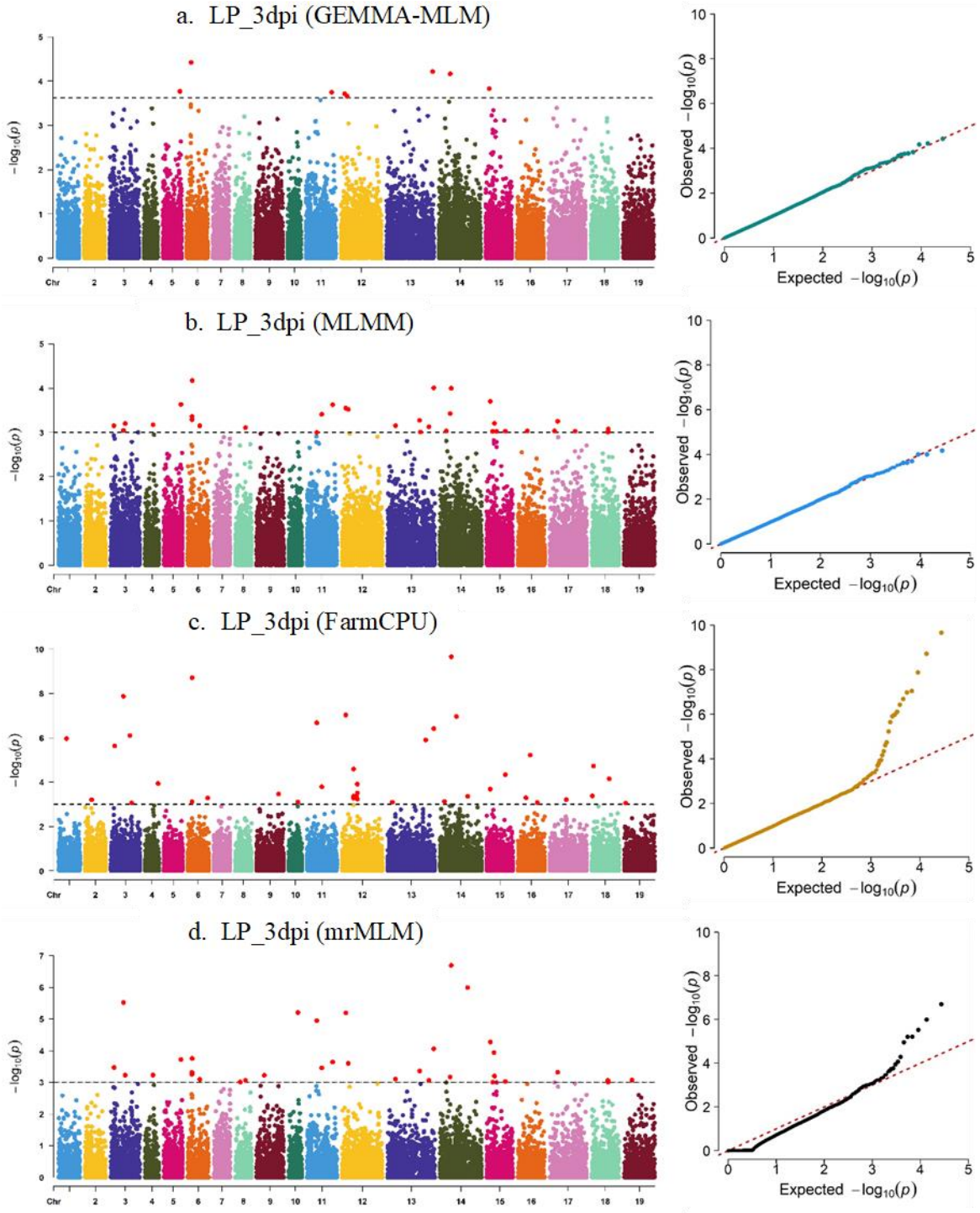

Figure A.12. Manhattan and quantile-quantile (Q-Q) plot of single-locus and multi-locus GWA models for Sclerotinia sclerotiorum resistance trait, lesion phenotype scores at 3 days post inoculation (LP_3dpi) (a-d) in rapeseed/canola.

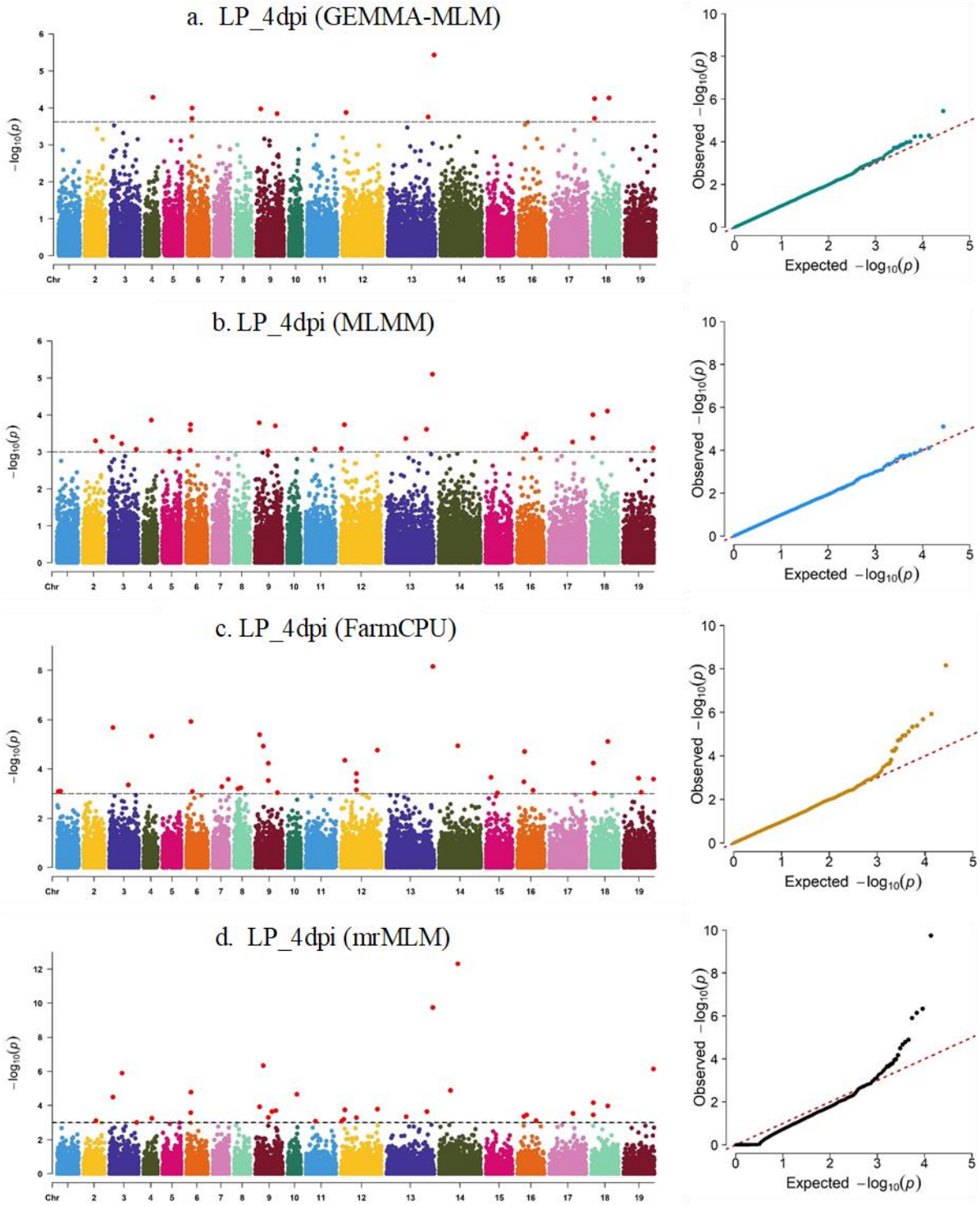

Figure A.13. Manhattan and quantile-quantile (Q-Q) plot of single-locus and multi-locus GWA models for *Sclerotinia sclerotiorum* resistance trait, lesion phenotype scores at 4 days post inoculation (LP_4dpi) (a-d) in rapeseed/canola.

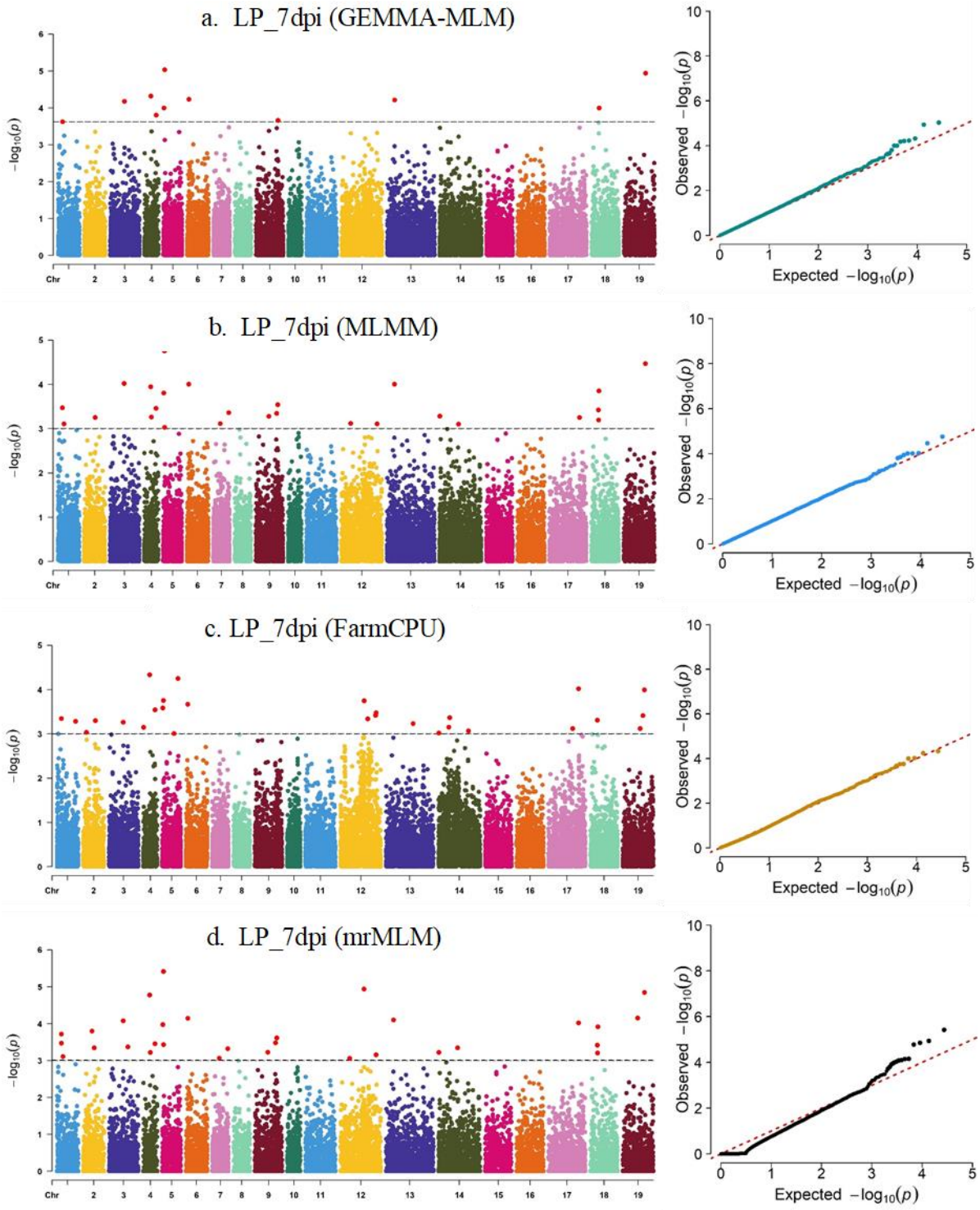

Figure A.14. Manhattan and quantile-quantile (Q-Q) plot of single-locus and multi-locus GWA models for *Sclerotinia sclerotiorum* resistance trait, lesion phenotype scores at 7 days post inoculation (LP_7dpi) (a-d) in rapeseed/canola.

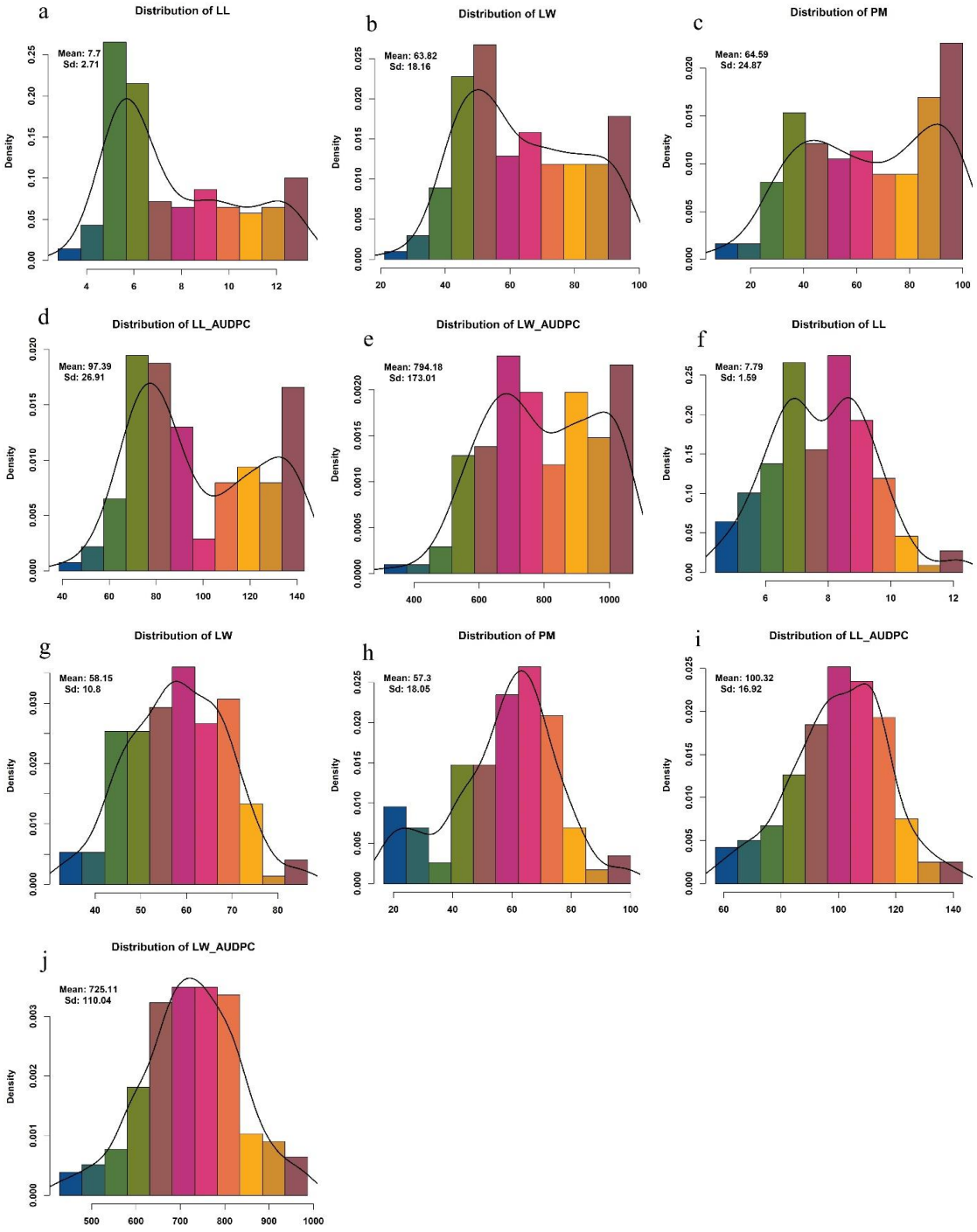

Figure A.15. Distribution of five phenotypic traits BLUE values of evaluated rapeseed/canola lines for *Sclerotinia sclerotiorum* resistance. Five traits are: LL, lesion length; LW, lesion width; PM, plant mortality; LL_AUDPC, lesion lengths area under disease progress curve (AUDPC) calculated using 7 time points reading, LW_AUDPC, lesion widths AUDPC calculated using 7 time points reading. (a-e) represents distribution of five traits for spring ecotypes genotypes; whereas (f-j) represents distribution of five traits for semi-winter and winter ecotypes genotypes.