ENLIGHTEN Agriculture

ISSN: 3049-3293
Volume: 1, Issue: 2, April 2025
Article No.: 03

Exploring genetic resistance for Blackleg disease in *Brassica napus*

Sonia Navvuru*

Department of Agricultural, Food and Nutritional Science, 4-10 Agriculture/Forestry Centre, University of Alberta, Edmonton, AB T6G 2P5, Canada.
***Email: navvuru@ualbeta.ca**

Abstract

Brassica napus is widely cultivated for its oil and seed meal. Its narrow genetic base and wide cultivation has made the crop prone to various biotic and abiotic stresses. Blackleg disease, caused by pathogen *Leptosphaeria maculans*, causes devastating losses in yield and oil quality. Integrated disease management practices although crucial, are not a sole strategy to control the devastating blackleg disease. The virulent strains of *L. maculans* continuously evolve and pose constant threat to the stability of the resistant B. napus. Resistance genes and loci in the genome are known to facilitate breeding durable resistance cultivars. Qualitative (R gene) and quantitative trait loci (QTL) in the germplasm can mediate resistance at different stages of the plant growth. In this communication, we will discuss the complex life cycle of the deadly pathogen and understand the genetic interactions between the host crop and the pathogen. Further, we will explore the genetic basis of resistance facilitated by the R genes, and QTLs against blackleg disease. In addition, we will dwell into the different resistance genes identified and emphasize their combined use in crop breeding through gene pyramiding for durability of the developed resistant cultivars.

Key words: *Brassica napus*, Blackleg disease, Genetic resistance, RMR gene, QTL, Gene pyramiding

Introduction

Brassica napus is an important oilseed crop worldwide with a global production of over 85,000 million metric tons (USDA-FAS 2025). As cultivated varieties of B. napus were bred focusing on agronomic traits they became genetically uniform; thus, resulting in their increased susceptibility to diseases, pests, and environmental stresses. In particular, blackleg fungal disease became a prominent, causing an annual yield loss of 10-15% on average and has a potential to cause up to a 90% yield

ISSN: 3049-3293
Volume: 1, Issue: 2, April 2025
Article No.: 03

loss during epidemics (Fitt et al., 2006a; Wang et al., 2020, 2023).The prominent symptom of this disease is stem canker, which affects the vascular system that facilitates water and nutrient transport.The first blackleg disease epidemic was recorded in total collapsed fields of oilseed rape in western Australia during the 1970s (Sivasithamparam et al., 2005). Subsequently, further losses were observed in early 2000s in other regions of Australia, with complete breakdown of single dominant gene-based resistance from *B. rapa supb.sylvestris*. Many other significant losses were reported from the UK, Canada, and mainland Europe in the following years (Neik et al., 2017). In addition to *L. maculans*, which mainly causes blackleg, a closely related but lesser-known species, Leptospheria biglobosa, is also often associated with this disease (Cai et al., 2014). *L. biglobosa* causes less severe symptoms, specifically on the upper stem; however, it is often out-competed by *L. maculans* when present together (Fitt et al., 2006b). In this communication, we will be discussing the disease pertaining to the main pathogen species, *L. maculans*.

Recognizing the importance of blackleg disease, scientists have explored the *Brassica* genetic base for resistance. However, eventually the research converged in one direction: mining for single major genes (qualitative resistance) from *B. rapa* due to its ease of use in breeding. Unfortunately, because of changing environments and evolving new pathotypes, this qualitative resistance rapidly breaks down. In response to this challenge, scientists have recently shifted their focus to finding novel stable resistance from other sources, specifically from *B. oleracea*. Therefore, it is crucial to delineate the underlying genetic and molecular mechanism of blackleg resistance (BR) for breeding resistant *B. napus* cultivars.

Blackleg disease pathogen, *Leptosphaeria maculans*

Leptosphaeria maculans belongs to Ascomycetes family of Fungi kingdom, which follows a hemi-biotrophic mode of nutrition, capable of infecting all stages of the crop growth. Its life cycle consists of both sexual (ascospore) and asexual (pycnidiospores) spores, facilitating high evolutionary potential and adaptivity to harsh environments. The crop debris from the previous crop cycle acts as a congenial environment for the resting spores, acting as a primary source of inoculum (Hall et al., 1992). Upon the spore germination, it first enters through the leaf stomata or wounds and causes necrotic lesions (Rouxel et al., 2003). Further, the blackleg pathogen enters and grows through the petiole to establish in the stem. However, during this initial infection phase, no significant symptoms are visible. As the plant grows and matures, pathogens turn to a necrotrophic mode of nutrition and colonize the stem crown region which restricts the flow of nutrition, weakens the plants, and reduces yield. In severe cases, infection leads to stem canker leading to plant death (Hammond et al., 1985). Primary infection during the early stages is initiated through ascospores, and subsequently, the pycnidiospores inflicts multiple cycles of infection throughout the growing season (West et al., 2001). Ascospores rapidly multiply and have the ability to travel via wind, facilitating infection in subsequent crops and the spread of the disease to distant regions (Rouxel and Balesdent et al., 2005).

L. maculans has evolved unique strategies to overcome the resistance incorporated into canola. One of its most dangerous features is its ability to build a large population, attributed to its ability to reproduce through both sexual and asexual modes. This facilitates pathogens to undergo rapid evolution and overcome single gene resistance. In addition to these biological features, the main character that determines its efficiency is its compartmentalized genome that facilitates rapid diversification and quick adaptation to new host resistance (Rouxel et al., 2011).

ISSN: 3049-3293
Volume: 1, Issue: 2, April 2025
Article No.: 03

The variation in the avirulence (Avr) gene profile of the pathotypes is likely a result of the pressure exerted by race-specific resistance of the qualitative genes present in resistant cultivars. Consequently, emergence of new pathotype isolates carrying novel Avr genes is the result of pathogen's evolutionary dynamics (Van de Wouw et al., 2024). Therefore, updating and standardizing differential sets will be essential for effectively defining evolving pathotypes/races in the future. To mitigate this disease, several cultural, chemical and mechanical management practices are followed (West et al., 2001). However, these efforts have been quite futile for complete control. Developing canola cultivars carrying genetic resistance against the blackleg pathogen is the most environment friendly and economical approach.

Sources of genetic resistance against blackleg disease

B. napus (AABB) and its diploid progenitors, *B. rapa* (AA) and *B. oleracea* (CC), are the primary source of genetic resistance against blackleg. Winter – type *B. napus* is genetically more diverse with broad range of resistance genes in comparison to spring- type *B. napus* varieties that majorly relies on few genes e.g., Rlm1, Rlm3, Rlm4 genes for resistance (Rouxel et al., 2003; Light et al., 2011; Larkan et al., 2016). However, as a consequence of its highly inbred nature,resistance in *B. napus* is scarce. The A genome has been a major source of BR carrying several resistance genes—such as Rlm1, Rlm2, Rlm7, LepR1-4, and Rlm11—mapped on *B. rapa* genome. In contrast, resistance genes have been less frequently identified in *B. oleracea*, among the few known genes, Rlm13 and Rlm14 have been characterized in detail (Ferdous et al., 2020, Raman et al., 2021; Degrave et al., 2021). Additionally, some level of resistance is reported in *B. oleracea* var. *capitata* (Korean germplasm) and in the C genome of the allotetraploid species *B. carinata* (Robin et al., 2017; Ferdous et al., 2020).

The approach to broaden the genetic diversity in resistance to combat the blackleg disease is the most sustainable approach (Rouxel et al., 2024). Breeding effort are being carried out to identifying newer resistance species from the wide gene pool of Brassicacea family. Attempts in crossing these wild resistance species and progenitor species with cultivated *B. napus* are being carried out to introduce the resistance into the commercial lines. For instance, wild *brassica* species, *Sinapis arvensis*, is reported to demonstrate strong resistance against multiple aggressive strains of *L. maculans* (Winter et al., 2003; Snowdon et al., 2007). Further, *B. napus* has been hybridized with its related allopolyploid species such as *B. juncea* (AABB) and *B. carinata* (BBCC), which are known to carry novel genetic resistance against the disease (Katche et al., 2019; Quezada-Martinez et al., 2021, Wu et al., 2024). In the following sections, we will discuss in detail the two major categories of BR; qualitative resistance, which is mediated by major R genes and quantitative resistance which is brought about by multiple minor-effect loci.

Understanding qualitative resistance for Blackleg disease

Single gene resistance (R) that produces robust and rapid gene response is termed as qualitative resistance. These qualitative genes mediate resistance independently or in coordination with environmental factors at different stages of the plant development. Interaction between *B. napus* and *L. maculans* follows gene for gene interaction, where a response is triggered as a reaction to the pathogen, which carries corresponding Avr gene (Bent and Mackey et al., 2007). Hence the R-mediated resistance (RMR) is usually considered to be race- specific. RMR is linked with cell death and hypersensitive response (HR) reaction and can be easily accessed through cotyledon assay (Table 1.). RMR genes are mostly found to be highly heritable and hence, are tested at the cotyledon stage efficiently. The presence and absence of the Avr genes, forms the basis for the pathogen race classification.Many of the RMR genes identified do not have a published genomic location and are recognized by their characterized Avr (Rlm5, Rlm6, Rlm10,

ISSN: 3049-3293
Volume: 1, Issue: 2, April 2025
Article No.: 03

Rlm14) despite being introgressed into *B. napus* and thus are assumed to segregate as single gene (Borhan et al., 2022). In addition, LepR series of qualitative genes on the A genome of *B. rapa* ssp. *sylvestris* have been introgressed successfully into B. napus (Yu et al., 2013). Genes especially LepR1 and LepR2, play a crucial role in resistance at both cotyledonary and adult plant stages (Yu et al., 2005). LepR1 offers complete resistance at all stages of plant growth and LepR2 confers partial resistance at cotyledonary stage only (Yu et al., 2005). However, as the plant grows, it faces several diverse pathotypes, as a consequence, cultivar resistance is not retained consistently till crop reaches adult stage.

The huge phenotypic effect of the R gene in a cultivar, imparts strong selection pressure which forces the pathogen to evolve higher virulence and new pathotypes. Consequently, resulting in complete breakdown of resistance within a few years of the resistant cultivar/variety release. Hence, the constant race between the host and pathogen, characterized as the boom-and-bust cycle needs to be strongly considered while breeding for resistance. The genetic basis of BR is very complex, loci of RMR genes that facilitate the resistance at cotyledonary stage are known to overlap with quantitative trait loci (QTL), which mediate resistance at adult plant stage.

Understanding quantitative resistance for Blackleg disease

Quantitative Resistance (QR) provides partial, race non-specific resistance against a broad range of pathogenic isolates imparting low selection pressure on the pathogen (Table 2.). Therefore, despite being highly influenced by environmental factors, it is very durable in comparison to RMR (Brun et al., 2010). Phenotype evaluation found that QR reduces only disease symptoms and doesn't completely eliminate the disease (Amas et al., 2021). There is a huge effect of genotype × environment interaction on the QR identified in a mapping population and hence, their effect is significantly lower than the RMR.The major reasons behind the ambiguity and ignorance towards their use in breeding resistance varieties is due to: (i) complex genetics of QR, where several QTLs control resistance at various levels during different stages of plant growth. (ii) Influence of varying environmental factors on disease progression and (iii) lack of precision in phenotyping of minor resistance showcased by the minor effect of QR genes.

Identifying characteristic quantitative gene resistance (QR) by genome-wide markers

gene The goal of QTL mapping is to sort continuous (QR), non-Mendelian variation into discrete groups of Mendelian factors (R sets) (Paterson et al., 1988). Researchers developed mapping populations including Double haploid populations from Major × stellar and resistant lines, Caiman, Canberra, ^{AV}Sapphire and Rainbow, which were then used to identify and map several QTLs (Ferreira et al., 1995, Kaur et al., 2009). Exploration of Australian *B. napus* cultivars AG-Castle and AG-Sapphiare was found to be immensely vital in identifying four prominent QTLs with constant heritability on previously identified blackleg QTLs on chromosomes A01, A09, A08 and C06 (Larkan et al., 2016).

Darmor has been the primary source for evaluating blackleg QR in canola (Pilet et al., 1998, Pilet et al., 2001, Jestin et al., 2012, 2015). DH lines from Darmor-bzh/Yudal (DYDH) population in Australian field conditions were used to identify 27 significant QTLs across 12 chromosomes explain 2.14% - 10.13% of the genotypic variance. Among these, seven QTLs on chromosomes A02, A07, A09, A10, C01 and C09 were found consistently across different experiments in multiple environments (Raman et al., 20r18). The consistent identification of QTLs on Darmor and its parent Jef Neuf reiterate their significance as vital germplasms for BR. However, identifying exact location of the QTLs in canola has been challenging due to non-uniform evaluation of different genetic mapping populations from diverse backgrounds (e.g., spring, semi-winter, or winter). With the recent publication of *B. napus* cv. Darmor-bzh sequence and knowledge on physical positions of 425 R genes (Alamery et al., 2019), it is now possible to compare different QTLs and RMR genes identified across various studies and locations

ISSN: 3049-3293
Volume: 1, Issue: 2, April 2025
Article No.: 03

(Table 1.) RMR genes identified in the Genus *Brassica*.

RMR gene (location)	Mapping population/ Genotype	References
Rlm1 on chr. A7	B. napus (DH "Maxol", and "Columbus", Cultivar "Westar," "Quinta," and "Glacier,")	Raman et al., (2012a), Ansan-Melayah et al., (1995), (1998)
Rlm2 and LepR3 (Allelic version of the same gene) on Chr. A10	B. napus (Cultivar "Westar," "Quinta," and "Glacier,") and B. rapa (cultivar "Surpass 400")	Ansan-Melayah et al., (1998), Li and Cowling (2003); Yu et al., (2008); Larkan et al.,(2015)
Rlm3 on Chr. A7	B. napus (Cultivar "Glacier, Maxol")	Li and Cowling et al., (2003); Delourme et al., (2004); Yu et al., (2008)
Rlm4, Rlm7 and Rlm9 (Allelic version of the same gene) mapped on chr. A7	B.napus (Cultivar "Jet Neuf, Caiman, Darmor- bzh")	Balesdent et al., (2001), (2002); Delourme et al., (2004); Raman et al., (2012b) Larkan et al., (2020); Haddadi et al., (2022)
Rlm5 and Rlm6 (have epistatic interaction)	B. juncea(Cultivar "Aurea" and "Picra")	Balesdent et al., (2002)
Rlm8	B. rapa (Line "156-2-1")	Balesdent et al., (2002)
Rlm 10 on Chr. B4	B. juncea (Cultivar "Junius")	Chevre et al., (1997)
Rlm11on Chr. A7	B. rapa(Accession "02-159-4-1" (R)* × DH "Z1" (S)*, and with "Darmor" and "Eurol")	Balesdent et al., (2013)
Rlm12 on chr. A1	B. napus (GWAS panel of 179 accessions from DH population SAgS)	Raman H. et al., (2016)
Rlm 13 on C03	*B. napus* (Cultivar "ATR-Cobbler")	Raman et al., (2021)

ISSN: 3049-3293
Volume: 1, Issue: 2, April 2025
Article No.: 03

RlmI4	*B. oleracea* (Cultivar "Monaco")	Degrave et al., (2021)
BLMR1 and BLMR2, single major gene on chr. N10	*B. napus* (Mapping populations of cultivar "Surpass 400" (R) ×"Westar" (S))	Long et al., (2011)
ClmR1(same genetic interval as LmR1) chr. A7	*B. napus* (Two different mapping populations, "DH12075" from cultivar "Cresor" (R) × re-synthesized line "PSA12" (S) and "Shiralee" (R) ×"PSA12" (S))	Mayerhofer et al., (2005)
LepR1(dominant nuclear allele) on chr.A2	*B. napus* (DH population, "DHP95" and"DHP96" with resistance introgressed from *B. rapa* subsp. *Sylvestris*)	Yu et al., (2005)
LepR2 (incomplete,reduces growth) onA10	*B. rapa* (Cultivar "Surpass 400")	Van De Wouw et al., (2009); Neik et al., (2022)
LepR3 on A10	*B. rapa* (Cultivar "Surpass 400")	Li and Cowling et al., (2003); Yu et al., (2008); Larkan et al., (2013); Larkan et al., (2014); Larkan and Bohran et al., (2015)
LepR4 recessive on A genome, N6 linkage group of B. napus	*B. napus* ("DH12075" derived from cultivar"Cresor" that has R gene LmR1 × Westar (S))	Yu et al., (2013)
RlmSTEE98 on A9	*B. napus* (Cultivar "Yudal")	Jiquel et al., (2021)
LmR1 onLinkage group 6	*B. napus* (DH population from cultivar "Shiralee" and "Maluka" (R)× advanced breeding lines (S))	Mayerhofer et al., (1997), Long et al., (2011)

ISSN: 3049-3293
Volume: 1, Issue: 2, April 2025
Article No.: 03

LEM1Linkage group 6	*B. napus* (DH population from cultivar "Major" (R) × "Stellar" (S))	Ferreira et al., (1995)
LmFr1 on A7	*B. napus* (DH from cultivar "Cresor" (R) × "Westar" (S))	Dion et al., (1995), Kaur et al., (2009)
Two independent genes,onedominant (LMJR1 on J13 and one recessive (LMJR2 on J 18)	*B. juncea* (F2 population from F1progeny of Cultivar "AC Vulcan" × Inbred line "UM3132")	Christianson et al., (2006)

(R)- Resistant, (S)- Susceptible

(Table 2.) List of other QR genes identified in B. napus

QR gene (location)	Mapping population/crosses	References
aRLMc and aRLMrb (Adult stage) linked to cRLMm & cRLMrb (cotyledon stage)	*B. napus* (DH population developed from cultivar "Maluka,""Cresor," and "RB87-62" × "Westar" (S))	Rimmer et al., (1999)
Three QTL (Adult plant resistance) on chr. A1, A8, A9, and C6	*B. napus* (DH populations developed from cultivar "AG-Castle" and "AV-Sapphire"(R) × "Topas" (S))	Larkan et al., (2016)
17 QTL for adult plant resistance across 13 linkage groups	*B. napus* (DH lines from BnaDYDH mapping population, initially derived from "Darmor-bzh"(R) × "Yudal" (S))	Huang et al., (2016)
One major QTL on chr. A1	*B. napus*(Worldwide accession from Germplasm Resource Information Network)	Rahman et al., (2016)

ISSN: 3049-3293
Volume: 1, Issue: 2, April 2025
Article No.: 03

Discussion

A constant threat is posed by evolving pathotypes of *L. maculans*, making it essential to develop resistant lines by employing race-specific genes in rotation with stable quantitative genes. Major challenge in identifying QTL for BR is its sensitivity to environmental variation which necessitates evaluating the lines under uniform disease conditions. Next-generation sequencing is a useful tool to associate phenotypic observations with putative molecular mechanisms and capture the large genetic variability (Starosta et al., 2024). Today, genome sequences of all *Brassica* diploids and amphidiploids species, including *B. napus* are available for study and application (Chalhoub et al., 2014; Bayer et al., 2017). Further, utilizing advance genotyping tools like 60K *Brassica* Infinium SNP array and genotyping-by-sequencing (GBS) has swiftly accelerated identification of genes/ QTLs for BR (Rahman et al., 2016).

Earlier, to avoid the pathogen population buildup and RMR gene breakdown, rotation of resistant gene sources every year was recommended. However, as qualitative resistance started staggering, researchers have diverted their attention to the prospects of utilizing QR alongside RMR. While it's undeniable that quantitative resistance (QR) is controlled by multiple genes, exerts low pressure on pathogen evolution and reduces the risk of resistance breakdown, it's also necessary to consider that QR doesn't completely prevent allele diversification, and eventually losses occur over time. Nevertheless, QR plays a significant role in extending durability of R-genes and preventing sudden blackleg epidemics. A Five-year research study by Brun et al., (2010) evaluated Darmor MX introgression line carrying both major gene (Rlm6) and Darmor QR genes and found prolonged resistance in comparison to lines with only RMR genes. Additionally, this strategic use of both RMR and QR genes maintained the avirulence/ virulence alleles of the pathogen resulting in stable resistance (Delourme et al., 2014; Huang et al., 2018). Hence, it can be concluded that during the initial stage of plant growth, RMR restricts the pathogen's colonization and as the plant grows, QR further restricts the spread of blackleg to the petiole main crown region, thereby exhibiting effective resistance.

Conclusion and Future perspectives

Blackleg disease remains a persistent challenge in *B. napus* (canola) cultivation, significantly impacting crop yields and economic sustainability. Integrating genetic resistance with sustainable cultural management practices is vital for a comprehensive disease management strategy. The continuous evolution of *L. maculans* poses a formidable barrier to breeding durable, resistant canola cultivars. To combat this pathogen, plants employ two primary resistance mechanisms: qualitative (major gene) resistance and quantitative resistance (QR). It is mandatory to pyramid both qualitative and quantitative genes for developing stable *B. napus*. Where major gene resistance provides strong but often short-lived protection, quantitative resistance—due to its polygenic nature—offers more durable and sustainable defense.

Given the dynamic nature of the pathogen, the demand for novel resistance genes will remain ongoing. A collaborative effort targeting in compositing a universally accessible database encompassing resistant germplasm is essential. Further, advancements in genome sequencing,multi-omics and gene technologies need to be employed for identifying novel resistant genes and understanding the complex genetic and molecular basis of resistance. Rapid expansion in use of computational biology, machine learning, data mining, and artificial intelligence and high-throughput phenotyping, has revolutionized resistance breeding by facilitating the development of prediction models for precise selection in breeding programs. Therefore, integrated approach combining modern molecular tools with agronomic practices will enhance the durability of resistance, ensuring stable *B. napus* yields.

ISSN: 3049-3293
Volume: 1, Issue: 2, April 2025
Article No.: 03

References

Anastasiou, E., Koutsiaras, M., Fountas, S., Kriezi, O., Voulgaraki, M., Lazarou, E., Psiroukis, V., Vatsanidou, A., Bartolo, L. F. F. D., Hurle, J. B., & Barbero, M. G. (2023). Precision farming technologies for crop protection: A meta-analysis. Smart Agricultural Technology, 5, 1–20.

Alamery S, Tirnaz S, Bayer P, Tollenaere R, Chaloub B, Edwards D & Batley J (2018) Genome-wide identification and comparative analysis of NBS-LRR resistance genes in Brassica napus. Crop Pasture Sci 69(1):79–93.

Amas J, Anderson R, Edwards D, Cowling W, Batley J (2021) Status and advances in mining for blackleg (Leptosphaeria maculans) quantitative resistance (QR) in oilseed rape (Brassica napus). Theor Appl Genet 134(10):3123–3145.

Ansan- Melayah D, Balesdent M H, Buée M, Rouxel T (1995) Genetic characterization of AvrLm1, the first avirulence gene of Leptosphaeria maculans. Phytopathology 85:1525–1529.

Ansan-Melayah D, Balesdent M-H, Delourme R et al (1998) Genes for race-specifc resistance against blackleg disease in Brassica napus L. Plant Breed 117:373–378.

Balesdent M H, Attard A, Ansan-Melayah D, Delourme R, Renard M, Rouxel T (2001) Genetic control and host range of avirulence toward Brassica napus cultivars Quinta and Jet Neuf in Leptosphaeria maculans. Phytopathology 91:70–76.

Balesdent M H, Attard A, Kuhn M L, Rouxel T (2002) New avirulence genes in the phytopathogenic fungus Leptosphaeria maculans. Phytopathology. 92:1122–1133.

Bent A F & Mackey D (2007) Elicitors, effectors, and R genes: the new paradigm and a lifetime supply of questions. Annu Rev Phytopathol 45:399–436.

Borhan M H, Van de Wouw A P & Larkan N J (2022) Molecular interactions between Leptosphaeria maculans and Brassica species. Annu Rev Phytopathol 60:237–257.

Brun H, Chèvre A M, Fitt B D, Powers S, Besnard A L, Ermel M, Huteau V, Marquer B, Eber F, Renard M, Andrivon D (2010) Quantitative resistance increases the durability of qualitative resistance to Leptosphaeria maculans in Brassica napus. New Phytol 185(1):285–299.

Cai X, Yang L, Zhang J, Li GQ (2014) First report of Leptosphaeria biglobosa causing blackleg on Rhapanus sativus in Central China. Plant Dis 98:993.

Chalhoub B, Denoeud F, Liu S, Parkin I A, Tang H, Wang X, Chiquet J, Belcram H, Tong C, Samans B, Corréa M (2014) Early allopolyploid evolution in the post-Neolithic Brassica napus oilseed genome. Science 345(6199):950–953.

Chèvre A, Barret P, Eber F. et al. Selection of stable Brassica napus-B. juncea recombinant lines resistant to blackleg (Leptosphaeria maculans). 1. Identification of molecular markers, chromosomal and genomic origin of the introgression. Theor Appl Genet 95, 1104–1111 (1997).

Christianson J A, Rimmer S R, Good A G, Lydiate D J (2006) Mapping genes for resistance to Leptosphaeria maculans in Brassica juncea Genome 49:30–41.

Degrave A, Wagner M, George P, Coudard L, Pinochet X, Ermel M, Gay EJ, Fudal I, Moreno-Rico O, Rouxel T and Balesdent M H (2021) A new avirulence gene of Leptosphaeria maculans, AvrLm14, identifies a resistance source in American broccoli (Brassica oleracea) genotypes. Molecular Plant Pathology, 22(12), pp.1599-1612.

Delourme R, Bousset L, Ermel M, Duffe P, Besnard A L, Marquer B, Fudal I, Linglin J, Chadoeuf J, Brun H (2014) Quantitative resistance affects the speed of frequency increase but not the diversity of the virulence alleles overcoming a major resistance gene to Leptosphaeria maculans in oilseed rape. Infect genet evol 27:490–499.

Delourme R, Pilet-Nayel M L, Archipiano M, Horvais R, Tanguy X, Rouxel T, Brun H, Renard M, Balesdent M H (2004) A cluster of major specific resistance genes to Leptosphaeria maculans in Brassica napus. Phytopathology 94:578–583.

Dion Y, Gugel R K, Rakow G F, Seguin-Swartz G, Landry B S (1995) RFLP mapping of resistance to the blackleg disease [causal agent, Leptosphaeria maculans (Desm.) Ces. et de Not.] in canola (Brassica napus L.) Theor Appl Genet 91:1190–1194.

Ferdous, M.J., Hossain, M.R., Park, J.I., Robin, A.H.K., Natarajan, S., Jesse, D.M.I., Jung, H.J., Kim, H.T. and Nou, I.S., 2020. In-silico identification and differential expressions of LepR4-syntenic disease resistance related domain containing genes against blackleg causal fungus Leptosphaeria maculans in Brassica oleracea. Gene Reports, 19, p.100598.

Ferreira M E, Rimmer S R, Williams P H, Osborn T C (1995) Mapping loci controlling Brassica napus resistance to Leptosphaeria maculans under different screening conditions. Phytopathology 85(2):213–217.

Fitt B D L, Brun H, Barbetti M J, Rimmer S R (2006a) World-wide importance of phoma stem canker (Leptosphaeria maculans and L. biglobosa) on oilseed rape (Brassica napus). Eur J Plant Pathol 114:3–15.

Fitt B D L, Huang Y J, van den Bosch F, West J S (2006b) Coexistence of related pathogen species on arable crops in space and time. Annu Rev Phytopathol 44:163–168.

Haddadi P, Larkan N J, Van de Wouw A, Zhang Y, Neik T X, Beynon E. et al. (2022) Brassica napus genes Rlm4 and Rlm7, conferring resistance to Leptosphaeria maculans, are alleles of the Rlm9 wall-associated kinase-like resistance locus. Plant Biotechnology Journal, 20, 1229–1231.

Hall R (1992) Epidemiology of blackleg of oilseed rape, Canadian Journal of Plant Pathology, 14:1, 46-55.

Hammond K E, Lewis B G, Musa T M (1985) A systemic pathway in the infection of oilseed rape plants by Leptosphaeria maculans. 34(4):557–65.

Huang Y J, Jestin C, Welham S J, King G J, Manzanares-Dauleux M J, Fitt B D, Delourme R (2016) Identification of environmentally stable QTL for resistance against Leptosphaeria maculans in oilseed rape (Brassica napus). Theor Appl Genet 129:169–180.

Huang Y J, Mitrousia G K, Sidique S N M, Qi A, Fitt B D (2018) Combining R-gene and quantitative resistance increases effectiveness of cultivar resistance against Leptosphaeria maculans in Brassica napus in different environments. PLoS ONE13:e0197752.

ISSN: 3049-3293
Volume: 1, Issue: 2, April 2025
Article No.: 03

Iestin C, Bardol N, Lode M, Duffe P, Domin C, Vallée P, Mangin B, Manzanares-Dauleux M J, Delourme R (2015) Connected populations for detecting quantitative resistance factors to phoma stem canker in oilseed rape (Brassica napus L.). Mol Breed 35:1–16 .

Jestin C, Vallée P, Domin C, Manzanares-Dauleux M J, Delourme R (2012) Assessment of a new strategy for selective phenotyping applied to complex traits in Brassica napus. Open J Genet, 2: 190-201.

Jiquel A, Gervais J, Geistodt-Kiener A, Delourme R, Gay E J, Ollivier B, Fudal I, Faure S, Balesdent M H and Rouxel T (2021) A gene-for-gene interaction involving a 'late' effector contributes to quantitative resistance to the stem canker disease in Brassica napus. New phytologist, 231(4):1510-1524.

Katche E, Quezada-Martinez D, Katche E et al (2019) Interspecific hybridization for Brassica crop improvement. Crop Breed Genet Genom 1:190007.

Kaur S, Cogan NOI, Ye G, et al. (2009)Genetic map construction and QTL mapping of resistance to blackleg (Leptosphaeria maculans) disease in Australian canola (Brassica napus L.) cultivars. Theor Appl Genet 120:71–83.

Larkan N J, Lydiate D J, Parkin I A P, Nelson M N, Epp D J, Cowling W A, Rimmer S R and Borhan M H (2013) The B rassica napus blackleg resistance gene LepR3 encodes a receptor-like protein triggered by the L eptosphaeria maculans effector AVRLM 1. New Phytologist 197(2): 595-605.

Larkan N J, Lydiate D J, Yu F, Rimmer S R & Borhan M H (2014). Co-localisation of the blackleg resistance genes Rlm2 and LepR3 on Brassica napus chromosome A10. BMC Plant Biology 14:1-9.

Larkan N J, Yu F, Lydiate D J, Rimmer S R, Borhan M H (2016) Single R gene introgression lines for accurate dissection of the Brassica–Leptosphaeria pathosystem. Front Plant Sci 7:1771.

Larkan N J, Ma L and Borhan M H (2015) The B rassica napus receptor-like protein RLM 2 is encoded by a second allele of the LepR3/Rlm2 blackleg resistance locus. Plant Biotechnology Journal, 13(7), pp.983-992.

Larkan N J, Ma L, Haddadi P, Buchwaldt M, Parkin I A, Djavaheri M and Borhan M H (2020) The Brassica napus wall-associated kinase-like (WAKL) gene Rlm9 provides race-specific blackleg resistance. The Plant Journal, 104(4):892–900.

Li C X, Cowling W A (2003) Identification of a single dominant allele for resistance to blackleg in Brassica napus 'Surpass 400'. Plant Breed 122:485–488.

Light K A, Gororo N N, Salisbury P A (2011) Usefulness of winter canola (Brassica napus) race-specific resistance genes against blackleg (causal agent Leptosphaeria maculans) in southern Australian growing conditions. Crop Pasture Sci 62:162–168.

Long Y, Wang Z, Sun Z, Fernando D W, Mcvetty P B, Li G (2011) Identification of two blackleg resistance genes and fine mapping of one of these two genes in a Brassica napus canola cultivar 'Surpass 400.'Theor Appl Genet 122:1223–1231.

Mayerhofer R, Good A G, Bansal V K, Thiagarajah M R, Stringam G R (1997) Molecular mapping of resistance to Leptosphaeria maculans in Australian cultivars of Brassica napus. Genome 40:294–301.

Mayerhofer R, Wilde K, Mayerhofer M, Lydiate D, Bansal V K, Good A G, Parkin I A (2005) Complexities of chromosome landing in a highly duplicated genome: toward map-based cloning of a gene controlling blackleg resistance in Brassica napus. Genetics 171:1977–1988

Neik T X, Barbetti M J, Batley J (2017) Current Status and Challenges in Identifying Disease Resistance Genes in Brassica napus. Front Plant Sci 6(8):1788.

Neik T X, Ghanbarnia K, Ollivier B, Scheben A, Severn-Ellis A, Larkan N J, ... & Balesdent M H (2022). Two independent approaches converge to the cloning of a new Leptosphaeria maculans avirulence effector gene, AvrLmS-Lep2. Molecular Plant Pathology, 23(5), 733.

Paterson A H, Lander E S, Hewitt J D, Peterson S, Lincoln S E, Tanksley S D (1988) Resolution of quantitative traits into Mendelian factors by using a complete linkage map of restriction fragment length polymorphisms. Nature 335:721–26.

Pilet M L, Delourme R, Foisset N, Renard M (1998) Identification of loci contributing to quantitative field resistance to blackleg disease, causal agent Leptosphaeria maculans (Desm.) Ces. et de Not., in winter rapeseed (Brassica napus L.). Theor Appl Genet 96:23–30.

Pilet M L, Duplan G, Archipiano M, Barret P, Baron C, Horvais R, Tanguy X, Lucas M O, Renard M and Delourme R (2001) Stability of QTL for field resistance to blackleg across two genetic backgrounds in oilseed rape. Crop Sci. 41:197–205.

Quezada-Martinez D, Addo Nyarko CP, Schiessl SV, Mason AS (2021) Using wild relatives and related species to build climate resilience in Brassica crops. Theor Appl Genet 134:1711–1728.

Raman H, Raman R, Diffey S, Qiu Y, McVittie B, Barbulescu D M, Salisbury P A, Marcroft S, Delourme R (2018) Stable quantitative resistance loci to blackleg disease in canola (Brassica napus L.) over continents. Front Plant Sci 9:1622.

Raman R, Diffey S, Carling J, Cowley R B, Kilian A, Luckett D J, Raman H (2016) Quantitative genetic analysis of grain yield in an Australian Brassica napus doubled-haploid population. Crop Pasture Sci. 67, 298–307.

Raman H, Raman R, Qiu Y, Zhang Y, Batley J and Liu S (2021) The Rlm13 gene, a new player of Brassica napus–Leptosphaeria maculans interaction maps on chromosome C03 in canola. Frontiers in Plant Science, 12, p.654604.

Raman R, Taylor B, Lindbeck K, Coombes N, Barbulescu D, Salisbury P, Raman H (2012a). Molecular mapping and validation of Rlm1 gene for resistance to Leptosphaeria maculans in canola (Brassica napus L.). Crop Pasture Sci. 63, 1007–1017.

ISSN: 3049-3293
Volume: 1, Issue: 2, April 2025
Article No.: 03

Raman R, Taylor B, Marcroft S, Stiller J, Eckermann P, Coombes N, Rehman A, Lindbeck K, Luckett D, Wratten N, Batley J (2012b). Molecular mapping of qualitative and quantitative loci for resistance to Leptosphaeria maculans causing blackleg disease in canola (Brassica napus L.). Theor Appl Genet 125, 405–418.

Rahman M, Mamidi S, del Rio L, Ross A, Kadir M M, Rahaman M M & Arifuzzaman M (2016). Association mapping in Brassica napus (L.) accessions identifies a major QTL for blackleg disease resistance on chromosome A01. Molecular Breeding, 36, 1-15.

Rimmer S R, Borhan M H, Zhu B, Somers D (1999). Mapping resistance genes in Brassica napus to Leptophaeria maculans, in 10th International Rapeseed Congress (Canberra, ACT: The Regional Institute;).

Robin A H K, Larkan N J, Laila R, Park J I, Ahmed N U, Borhan H, Parkin I A and Nou I S (2017) Korean Brassica oleracea germplasm offers a novel source of qualitative resistance to blackleg disease. European Journal of Plant Pathology 149:611–623.

Rouxel T, Balesdent MH (2005) The stem canker (blackleg) fungus, Leptosphaeria maculans, enters the genomic era. Mol Plant Pathol 6:225–241.

Rouxel T, Grandaubert J, Hane J K, Hoede C, Van De Wouw A P, Couloux A, Dominguez V, Anthouard V, Bally P, Bourras S, Cozijnsen A J (2011) Effector diversification within compartments of the Leptosphaeria maculans genome affected by Repeat-Induced Point mutations. Nat. Commun. 2:202.

Rouxel T, Willner E, Coudard L, Balesdent M H (2003) Screening and identification of resistance to Leptosphaeria maculans (stem canker) in Brassica napus accessions. Euphytica 133: 219–231.

Rouxel T, Peng G, Van de Wouw A, Larkan N J, Borhan H and Fernando W D (2024) Strategic genetic insights and integrated approaches for successful management of blackleg in canola/rapeseed farming. Plant Pathology, 73(9), pp.2260-2280.

Sivasithamparam K, Barbetti M J, Li H U A (2005) Recurring challenges from a necrotrophic fungal plant pathogen: a case study with Leptosphaeria maculans (causal agent of Blackleg disease in Brassicas) in Western Australia Ann Bot 96:363–377.

Snowdon R, LüHs W, Friedt W (2007) Oilseed rape, in Genome Mapping and Molecular Breeding in Plants Vol 2: Oilseeds, ed Kole C. (Berlin, Heidelberg; New York, NY: Springer;), 55–114.

Starosta, E., Jamruszka, T., Szwarc, J., Bocianowski, J., Jędryczka, M., Grynia, M. and Niemann, J., 2024. DArTseq-based, high-throughput identification of novel molecular markers for the detection of blackleg (Leptosphaeria Spp.) resistance in rapeseed. International Journal of Molecular Sciences, 25(15), p.8415.

USDA-FAS (2025) Oilseeds: World Markets and Trade. Retrieved from: https://apps.fas.usda.gov/psdonline/circulars/oilseeds.pdf.

Van de Wouw AP, Marcroft SJ, Barbetti MJ, Hua L, Salisbury PA, Gout L, Rouxel T, Howlett BJ, Balesdent MH. 2009. Dual control of avirulence in Leptosphaeria maculans towards a Brassica napus cultivar with 'sylvestris-derived' resistance suggests involvement of two resistance genes. Plant Pathology 58: 305–313.

Van de Wouw AP, Scanlan J L, Al-Mamun H A, Balesdent M H, Bousset L, Burketová L, del Rio Mendoza L, Fernando W D, Franke C, Howlett B J and Huang Y J (2024) A new set of international Leptosphaeria maculans isolates as a resource for elucidation of the basis and evolution of blackleg disease on Brassica napus. Plant Pathology 73(1):170-185.

Wang Y, Strelkov S E & Hwang S-F (2023). Blackleg Yield Losses and Interactions with Verticillium Stripe in Canola (Brassica napus) in Canada. Plants, 12(3), p434.

Wang, Y., Strelkov, S.E. and Hwang, S.F., 2020. Yield losses in canola in response to blackleg disease. Canadian Journal of Plant Science, 100(5):488-494.

West J S, Kharbanda P D, Barbetti M J, Fitt B D L (2001) Epidemiology and management of Leptosphaeria maculans (phoma stem canker) on oilseed rape in Australia, Canada and Europe. Plant Pathology 50, 10–27.

Winter H, Snowdon R J, Diestel A, Gartig S, Sacristan M D (2003) Transfer of new blackleg resistances into oilseed rape, in 11th Int Rapeseed Congress (Copenhagen:) 19–21.

Wu T (2024). Exploring resistance genes to blackleg (Leptosphaeria maculans) in Brassica napus, Brassica juncea and Hirschfldia incana.

Yu F, Gugel R K, Kutcher H R, Peng G, Rimmer S R (2013) Identification and mapping of a novel blackleg resistance locus LepR4 in the progenies from Brassica napus × B. rapa subsp. sylvestris. Theor Appl Genet 126:307–315.

Yu F, Lydiate D J, Rimmer S R (2005) Identification of two novel genes for blackleg resistance in Brassica napus. Theor Appl Genet 110:969–979.