Understanding the Powdery Mildew Pathogen and Rapeseed Mustard Interactions: Insights into Disease Resistance and Molecular Mechanisms to Enhance the Quality and Productivity of Oilseed Brassica Crops

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The quantity and quality of oilseed production in rapeseed mustard are severely affected by biotic and abiotic stresses. Among these, the biotrophic fungus *Erysiphe cruciferarum* causes powdery mildew (PM) infection in Indian mustard cultivars, potentially reducing yield by up to 50% across affected regions in India. Considering recent developments in molecular plant pathology and their impact on sustainable management of challenging plant pathogens, this article reviews the current scenario for resistance and its mechanism to *E. cruciferarum* in *Brassica* cultivars. It also covers the complex molecular signaling pathways for resistance that are regulated by phytohormones along with differential gene expression, and effectors proteins in *Brassica* spp. The recent advancements in genomics have contributed to identification of resistance/susceptibility genes as well as quantitative trait loci (QTLs) involved in PM resistance. Furthermore, this review unfolds a comprehensive understanding of the genetic as well as genomic basis of resistance that can provide the valuable insights for breeding programs focused on developing PM-resistant rapeseed-mustard varieties. This review aims to provide the background on recent discoveries and future strategies on identification of resistance genes, aiding in the development of more resilient rapeseed-mustard crops and leading to significant improvements in crop protection and yield stability.

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INTRODUCTION

Rapeseed-mustard (*Brassica* spp.) stands as a cornerstone in global oilseed cultivation, notably contributing to the edible oil market alongside soybean and oil palm. However, a gap persists between the supply and demand of oilseeds, driven by the increasing global population. Moreover, the change in global climate scenario has posed a

significant risk to *Brassica* cultivation resulting in an increased disease incidences and severity. A diverse range of plant pathogens infect *Brassica* crops. One such known fungal biotroph is *E. cruciferarum* Opiz ex L. Junell, which causes powdery mildew (PM) disease in this crop. There are about 700 species of PM pathogens capable of infecting approximately 10,000 plant species (Braun and Cook 2012). Within the last two decades, PM has become epidemic in rapeseed-mustard, affecting over 120 cruciferous host plant species in more than 25 countries (Mir *et al.* 2023). The disease is especially prevalent in regions with cool, dry conditions in leading rapeseed-mustard producing countries such as Canada, China, India, and the European Union (FAO 2022). In India, it spreads rapidly across major mustard-growing states such as Uttar Pradesh, Rajasthan, Gujarat, and Maharashtra (Dange *et al.* 2002; Mohitkar *et al.* 2012; Meena *et al.* 2018), thus posing a major bottleneck to the seed production, quality and profitability. Furthermore, the pathogen caused yield losses ranging between 20% and 40% across various regions of India, with severely affected fields experiencing losses of up to 50% (Kumar *et al.* 2016; Meena *et al.* 2018). These significant yield losses can translate into economic setbacks, affecting the livelihoods of farmers and the overall productivity of the agricultural sector.

Despite its significance, PM has received less research attention compared to other diseases such as Alternaria blight and white rust. Perhaps this is because it appears later in the crop growth stages and its damage is estimated frequently after harvest. While previous reports primarily have emphasized yield losses, PM affects overall productivity by reducing oil quality and altering the plant's physiological processes, especially decreasing photosynthetic efficiency followed by overall plant vigour and seed yield. Due to the presence of powdery mass on leaf surfaces, significant reductions were observed in leaf gas exchange parameters like net photosynthetic rate and transpiration rate in the infected leaves of susceptible genotypes compared to resistant and moderate genotype groups. Chlorophyll fluorescence analysis revealed a decrease in the maximum quantum efficiency of photosystem-II in PM-infected leaves across the genotype groups in different crop plants (Sree *et al.* 2024; Saja *et al.* 2020). The net photosynthetic efficiency goes down in susceptible genotypes, which ultimately affects the productivity of the crop plants.

Striking research efforts by plant pathologists and breeders to combat PM in *Brassica* species have been bolstered by extensive work focused on elucidating the genetic level of resistance and developing resilient cultivars. Studies leveraging model plants, such as *Arabidopsis thaliana*, have given crucial insights for molecular defense mechanism, signalling pathways underlying resistance to PM and pathogen-host interactions. Genetic studies have identified candidate resistance genes (R genes) and quantitative traits loci (QTLs) imparting with PM resistance, facilitating marker-assisted selections and breeding programs targeted for improvement and development of resistant varieties for PM disease.

Moreover, the exploration of wild relatives of *Brassica* crops has unearthed promising genetic resources for enhancing resistance. These efforts are pivotal for the sustainable agriculture, aiming not only to minimize yield losses but also to reduce dependency on chemical pesticides, thereby promoting eco-friendly crop production practices. Cultural practices, such as crop rotation and debris management, help reduce disease pressure but they are not always sufficient alone. Breeding for resistance offers a sustainable solution, though it requires time and can be challenged by pathogen evolution. An integrated approach combining these strategies (cultural, genetic resistance, minimal use of pesticides) is the most effective for long-term PM disease management in Brassica crops. However, judicious exploration of such strategies will signify mustard cultivation with enhanced yield and quality. Hence, this review comprehends current knowledge and

recent advancements in global rapeseed-mustard research, including *Brassica* genetics, the PM (*E. cruciferarum*) pathogen, associated seed yield and oil content losses, and management strategies. It further explores key aspects such as genetic diversity, host-pathogen interactions, resistance mechanisms, and breeding strategies, that are crucial for ensuring food and nutritional security, particularly in developing countries. Furthermore, it outlines future research directions aimed for elucidating the complexities of PM disease and accelerating the development of durable resistance in *Brassica* crops. By understanding host-pathogen interactions and pinpointing resistance-associated genes, the findings aid in the development of disease-resistant varieties through molecular breeding and biotechnological approaches.

Fig. 1. Image illustrating the characteristic powdery mildew disease symptoms on *Brassica* spp. leaf, stems, and pods.

ASPECTS OF THE PATHOGEN AND ITS CONTROL

Economic Significance of *E. cruciferarum* in *Brassica* Species

Powdery mildew disease is an epidemic and devastating disease in Indian mustard cultivars. Due to variable weather conditions in different agro-climatic regions, powdery mildew affects photosynthetic efficiency, leading to stunted growth, premature senescence, defoliation, and reduced biomass accumulation. This results in shrivelled seeds, reduced seed weight, and poor pod formation, ultimately lowering the oil content and collectively deteriorating overall crop productivity. Powdery mildew disrupts photosynthetic surfaces. This not only reduces the photosynthetic efficiency and yield, but it also causes a stress condition for the plant. Exposure to stress condition can alter metabolite profiling and the lipid biosynthesis pathways that ultimately change the fatty acid composition and reduce its seed nutritional quality (Baud and Lepiniec 2010). In *B. juncea*, profound yield losses were recorded due to *E. cruciferarum* infection, for example, in Gujarat, yield loss was reported at 24.1% (Dange *et al.* 2003), while in Haryana, the EC-126743 accession showed a yield loss of 17.4% and a drop in oil content of 6.5% (Saharan and Sheoran 1985). In Maharashtra, the Pusa Bold and Seeta cultivars experienced dramatic yield reduction ranging from 45% to 95% (Hare 1994). Another study from Maharashtra reported yield losses between 15% to 40% and oil content reduction up to 37.6% in cultivars Seeta, Pusa Bold, Bio-902, and TM-17 (Kohire *et al.* 2008). Kanzaria *et al*. (2013) estimated losses of

7.3%, 21.7%, 22.5%, and 21.5% in oil content, protein content, seed yield, and test weight, respectively, because of *E. cruciferarum* infection in Maharashtra and Gujarat, regions where the PM disease is highly prevalent in severe form. Powdery mildew has also been reported to cause yield losses exceeding 25% to 30% in Turkey, United States, Korea, France, Australia, and Poland where *B. napus* is grown widely (Sadowski *et al.* 2002; Mert-Turk 2008; Kaur *et al.* 2008; Khangura *et al.* 2011; Kim *et al.* 2013; Uloth *et al.* 2016; Meena *et al.* 2018). *Brassica rapa* cultivars such as Yellow Sarson in Canada and Europe have shown yield losses of 20% to 30% and oil content reductions of 10% to 20% (Smith and Johnson 2020). In *B. oleracea*, yield loss is often measured in terms of marketable quality rather than quantity, yet losses ranging from 20% to 50% in cultivars including Green Magic in California and Spain have been reported (Table 1; Saharan *et al.* 2005). These widespread and significant yield and oil content reductions around the world highlight the necessity for effective disease management programmes to mitigate the economic impact and yield losses of powdery mildew on *Brassica* crops.

Table 1. Impact of Powdery Mildew Severity on Seed Yield and Oil Quantity Losses in *Brassica* spp. under Indian and Global Perspectives

Brassica Species	Cultivars	Yield Loss (%)	Oil Quantity Loss (%)	Location/ Countries	References
B. juncea	EC126743	17.4	6.5	Haryana (India)	Saharan and
	Pusa Bold and Seeta	45 to 95	-	Maharashtra (India)	Hare 1994
	-	24.1	-	Gujarat (India)	Dange *et al.* 2003
	Seeta	40.0	37.3	Maharashtra (India)	Kohire *et al.* 2008
	Pusa Bold	34.0	37.3		
	Bio-902	36.0	37.5		
	TM-17	15.0	37.6		
B. napus	-	> 25 to 30	-	United States, Poland, Turkey,	Karakaya *et al.* 1993; Sadowski *et al.* 2002; Mert-Turk 2008
	-	25 to 30	-	Korea, Australia	Khangura *et al.* 2011; Kim *et al.* 2013; Uloth *et al.* 2016
	-	10 to 30	-	France	Penaud 1998; Meena *et al.* 2018
B. rapa	Yellow Sarson	20 to 30	10 to 20	Canada, Europe	Kumar *et al.* 2016
B. oleracea	Green Magic	20 to 50	-	California, Spain	Saharan *et al.* 2005

Genomic insights and Host Range of *E. cruciferarum*

Erysiphe cruciferarum is a fungal biotroph that is primarily responsible for causing powdery mildew (PM) in *Brassica* species including *B. juncea*, *B. napus*, *B. nigra*, *B. oleracea*, and *B. rapa* (Glawe 2008). The phenotypic and fundamental genomic information based on the conserved internal transcribed spacer (ITS) region of DNA has been made available for *E. cruciferarum*, but no whole genome assembly has been reported to date. However, the database is rich in genome of fungi causing PM disease in hosts other than Brassicaceae. The previous genome assemblies indicates that PM fungi are approximately four times larger in size compared to other ascomycetes, especially, the genome size of *Golovinomyces orontii* is approximately 160 Mb. (Saharan *et al.* 2023). Various *E. cruciferarum* isolates were identified through the amplification of ITS1 and ITS2 regions that flank the 5.8S rRNA using universal primers ITS1 (forward) and ITS4 (reverse), showing >99% nucleotide identity with GenBank sequences for *E. cruciferarum*. The entire ITS region of the DNA for PM isolate KUS-F24819 was sequenced after amplification with primers ITS5 and P3, and deposited under the accession no. KC862331 showing 100% identity with *E. cruciferarum* isolates from *Arabidopsis thaliana*, *B. oleracea* var. *acephala*, and *B. rapa* (Kim *et al.* 2013). Morphological, along with phylogenetic, analyses of various isolates were conducted using Erysiphaceae-specific primers such as PMITS1 and PMITS2 (Borges *et al.* 2023). Detection at the molecular level involved amplifying the ITS1 region with oligonucleotides EryF and EryR from infected plant tissues DNA (Attanayake *et al.* 2009; Pane *et al.* 2021). So far, the use of comparative genomic studies greatly facilitates the identification and characterization of genes linked to target traits in various species (Wan et al., 2008). However, additional research is needed to elucidate the genomics and genetic diversity of the PM pathogen in Indian mustard. In addition to cultivated *Brassica* species, the pathogen *E. cruciferarum* also infects a wide range of wild relatives across the Brassicaceae family worldwide (Table 2).

Table 2. Inventory of Different *Brassica* Crop's Wild Relatives Infected by PM Pathogen Along with the Records of Various Locations Worldwide (Saharan *et al.* 2019)

***Brassica* Crop Wild Relatives**	**Location/ Country**	**References**
Alyssum sp.	Iran	Ershad 1977
A. hirsutum	Bulgaria and Iran	Ershad 1971, 1977; Amano 1986; Negrean and Denchev 2000
A. dasycarpum, A. strigosum	Iran	Ershad 1977; Khodaparast *et al.* 2000
Alliaria petiolata	United Kingdom (UK), Ohio (USA)	Amano 1986; Ershad 1977; Ciola and Cipollini 2011
A. alyssoides, Armoracia rusticana, Antirrhinum majus, Cheiranthus cheiri, Erysimum cheiri	UK	Ellis and Ellis 1997
Argemone mexicana	India	Bappammal *et al.* 1995
A. thaliana	USA, Zurich, Germany, Korea, and Europe	Koch and Slusarenko 1990; Karakaya *et al.* 1993; Choi *et al.* 2009
Brassica tournefortii	India	Dang *et al.* 2000
Cardamine debilis	New Zealand	Cooper 2013
Coronopus didymus	Jammu	Sharma 1979

C. hirsute, *C. flexuosa*	New Zealand	Boesewinkel 1977; Cooper 2013
Camelina sativa	UK and Greece	Vellios *et al.* 2017
Capsella bursa-pastoris	UK, Massachusetts (USA), Slovenia, and Jammu (India)	Sharma 1979; Ellis and Ellis 1985; Radisek *et al.* 2018
Cleome hassleriana	France and Italy	Agha *et al.* 2008; Garibaldi *et al.* 2009,
C. spinosa	France, Italy, and New Zealand	Boesewinkel 1977; Agha *et al.* 2008; Garibaldi *et al.* 2009
Cardaria subsp. *Chalapensis, Cardaria draba*	Iran	Ghanbary 1995; Aeenfar 2006
Conringia planisiligua	Iran	Khodaparast *et al.* 2000
Crambe spp., *C. orientalis*	Iran	Kachooeian *et al.* 2006
Diplotaxis tenuifolia	Italy	Pane *et al.* 2021
Descurainia sophia	Iran	Ershad 1977
E. sativa, E. vesicaria	Australia, Haryana (India)	Gunasinghe *et al.* 2013
Eschscholzia californica	Germany and Switzerland	Schmidt and Scholler 2011
Erodium moschatum, F. officinalis, Geranium homeanum	New Zealand	Boesewinkel 1977
Iberis amara	Madhya Pradesh, Jammu (India)	Sharma and Khare 1992
Lepidium apetalum	Korea	Shin and La 1992
L. campestre, L. sativum, L. latifolium	Iran	Amano 1986; Ershad 1977; Kachooeian *et al.* 2006
L. virginicum	Himachal Pradesh (India)	Paul 1984
Malcolmia Africana, M. incana, M. maritime	Iran, Canada, France	Farr *et al.* 2009; Mirzaee *et al.* 2010
Meconopsis sp., *Papaver* sp.	Czech Republic	Pastircakova and Pastircak 2013
Orychophragmus violaceus	China	Tian *et al*. 2024
Papaver nudicaule, P. somniferum, P. rhoeas	New Zealand, Madhya Pradesh (India)	Boesewinkel 1977; Sharma and Khare 1992
R. sativus	Iran, Haryana, and Jammu (India)	Sharma 1979; Suhag and Duhan 1985
R. raphanistrum subsp. Maritimus, Wasabia japonica	New Zealand	Cooper 2013
Rapistrum rugosum	Iran and Argentina	Ershad 1977; Ghanbary 1995; Khodaparast *et al.* 2000
Sinapis arvensis	Greece and Iran	Amano 1986; Vellios *et al.* 2017
Sisymbrium officinale, S. alliaria	UK	Ellis and Ellis 1985
Sisymbrium species	Mexico	Morales *et al.* 2009
S. iirio	Iran and Argentina	Niknam and Guya 1996; Braun *et al.* 2000
S. orientale	Iran	Khodaparast *et al.* 2000
Stylophorum species	Czech Republic	Pastircakova and Pastircak 2013
S. diphyllum	Switzerland	Bolay 2005

Furthermore, different Brassica genotypes exhibit varying degrees of resistance or susceptibility to *E. cruciferarum*. This variation is largely governed by the presence or absence of resistance (R) genes, differences in the speed and intensity of immune response

activation, and hormonal regulation efficiency. For instance, certain *B. juncea* lines and wild relatives possess quantitative trait loci (QTLs) conferring partial to strong resistance, while commercial cultivars often lack these traits. Additionally, transcriptomic differences in defense gene expression further explain the differential responses, where resistant genotypes show a faster and stronger activation of PR genes, SA signaling, and ROS production compared to susceptible ones.

Mechanistic Insights Underlying Host-Pathogen Interactions in *Brassica* Species

The first line of defense: PTI response against PM infection

Inherently, plants have evolved a two-step defense mechanism that detect and deploy immune response appropriately: (1) Pattern recognition receptors or PRRs (Extracellular receptor-like kinases: RLKs and receptor-like proteins: RLPs) that detect PAMPs/DAMPs; (2) Intracellular receptors with nucleotide-binding and leucine-rich repeat domains (NLRs) that interact with pathogen specific secretory proteins called effectors (Krattinger and Keller 2016). The initial immune response to the pathogen attack is triggered when the receptors on the host cell's surface, called the PRRs, recognize pathogen/damage-associated molecular patterns (PAMPs/DAMPs) (Jones and Dangl 2006), that are present on the pathogen's surface. This recognition leads to PTI response, which then initiates further down-signaling in the form of MAPK cascade and other defense associated pathways. Previous research has shown that the chitin, in cell walls of fungal pathogen, triggered immune responses in *Arabidopsis*, as it was recognized by PRR receptors, such as LYK4/5 (lysine motif receptor-like kinases 4/5) and CERK1 (chitin elicitor receptor kinase1), which led to accumulation of defense protein and callose deposition (Cao *et al.* 2014). Moreover, mutations in CERK1 were also linked to increased susceptibility for PM, emphasizing its role in broad range disease resistance (Wan *et al.* 2008). Similarly, the recessive gene, ol-2, an analog to the MLO gene revealed in barley, facilitated histological resistance through the development of papillae. These papillae are composed of callose and other substances and form at the sites where the plant and powdery mildew interact, therefore halting fungal growth early and ensuring complete resistance (Bai *et al.* 2003, 2008). In *Brassica* species also, these mechanisms need to be worked out for the rapid screening and identification of durable resistant types.

R-gene base or vertical immunity (Effector-triggered immunity ETI)

Although PTI response provides strong defense against the infection, pathogens tend to constantly evolve novel ways to bypass PTI. One such strategy is secretion of specialized molecules called the effectors in the host cytoplasm. Effectors or the 'avirulence' factors are known to promote virulence and are highly diverse in nature (White *et al.* 2000). The ETI is marked by robust resistance responses, activated when pathogen effectors (*avr* proteins) are recognized by intracellular receptors present in the host cells, resulting in race-specific, major gene resistance. Such type of resistance follows gene-for-gene hypothesis according to which an effector gene causes avirulence in the host plant with its interactive resistance (R) gene. These R gene encode diverse intercellular receptors with two conserved domains, *i.e.,* the nucleotide-binding site (NBS) and a region of leucine-rich repeats (LRRs). Apart from LRR or LRR-like domains, the R proteins typically possess toll/interleukin-1-receptor (TIR) domains, and serine/threonine kinases (S/TK). The R genes are usually categorized into two groups: those with a TIR domain towards N terminal are termed TNLs, while those with a CC (coiled-coil) domain towards

N terminal are termed as CNLs. Additionally, R-genes with PM 8 (RPW8) domain have also been reported and are referred to as RNLs (Tirnaz *et al.* 2020). Usually, initiation of R-gene recognition system induces hypersensitive response (HR), which stops the pathogen from spreading to neighboring healthy cells by inducing localized cell death at the infection site, therefore, conferring systemic resistance (Keen *et al*. 1993). The RNL gene in *Arabidopsis* (*viz*., RPW8.1 and RPW8.2) has been reported to provide resistance by promoting accumulation of H_2O_2 and confined cell death through the SA-dependent pathway (Kim *et al.* 2014). Among this, RPW8.2 localize towards EHM (extra-haustorial membrane) to activate defense signaling, requiring specific protein interactions and transport mechanisms. The reports on mutations in RPW8.2 affect its function and resistance (Saharan and Krishnia 2001).

Role of phytohormones in PM resistance: Systemic acquired resistance (SAR)

For effectively deploying defense response against the pathogens, host plants have developed various signaling pathways during the initiation of the infection. These pathways are mostly activated in response to the changes in regulation of different phytohormones, especially SA, JA, and Et. While SA accumulates during the attack by biotrophic pathogens, JA and Et come together to stop the development of necrotrophic organisms. Previous studies have shown the role of SA in inducing systemic acquired resistance (SAR) in response to PM infection, which is caused by a biotrophic pathogen. Usually, SA accumulation leads to SAR response, resulting in a long-term resistance against multiple pathogens by activating a range of defense genes, particularly those encoding pathogenesis-related (PR) proteins (Park *et al*. 2007). Key studies have established the importance of SA signaling in the regulation of genes necessary for redox and calcium signaling, essential for activation of NPR1 gene (Chandran *et al.* 2009). This results in the increase of transcripts for genes involved in SA-production and PR genes. Mutations in SA signaling genes such as wrky18 and wrky40 can affect the plant's PM disease response. Furthermore, mutation in EDR1 protein kinase affects the accumulation of defense-related transcripts, particularly those encoding TFs such as WRKY and AP2/ERF, thus increasing SA-dependent resistance against PM (Christiansen *et al.* 2011). Moreover, infected plants with the EDR1 mutation also revealed increased genes expression related to the endomembrane system and ROS generation. The EDR1 migrates from endoplasmic reticulum to the plant-fungal interaction site during PM disease, which suggests that it is a part of secretory pathway in the defense against PM (Christiansen *et al.* 2011; Wu *et al.* 2015). These results demonstrate complex roles that the secretory system and SA signaling play in *Arabidopsis* protection against *E. cruciferarum*. The PR-1 and PR-2 genes were significantly up-regulated in *Raphanus alboglabra* compared to *B. napus*, with the elicitor stimulating these genes upon PM infection (Alkooranee *et al.* 2015). These types of studies, thus, highlight the intricate interplay between SA signaling, transcription factors, and the secretory system in orchestrating effective defense mechanisms against powdery mildew, underscoring the complexity of plant immune responses.

Inheritance and Resistance Mechanisms in *Brassica* Species Against *E. cruciferarum*

Brassica species as potential genetic resistance sources for PM

Plant genetic resources are crucial components of agricultural biodiversity, essential for the development of novel varieties to feed the world's burgeoning population. The genetic diversity within *Brassica* germplasm represents a valuable reservoir of resistance

against PM disease, which can be harnessed effectively for the development of disease resilient cultivars. Therefore, extensive research has been conducted globally to explore the inherent potential for disease resistance in rapeseed-mustard against PM pathogen (Table 3). Assessment of 71 genotypes of *B. juncea* from China, India, and Australia revealing four genotypes, 'JM06014', 'JM06015', 'JM06012', and 'JM06009', provided PM resistance in natural field conditions (Singh *et al.* 2010). However, in contrast, all tested genotypes from China and India were found to be susceptible to PM disease. In another study, 200 genotypes were screened out of which 20 resistant, 9 moderately resistant, and 71 highly susceptible genotypes were selected (Singh *et al.* 2016). Similarly, 61 germplasm were tested under natural conditions, identifying eight immune and one highly resistant, with 36 moderately resistant genotypes (Chadar *et al.* 2020). One genotype 'RDV 29' was also reported as highly resistant along with 12 moderately resistant germplasm after screening of 1,020 Indian mustard accessions (Nanjundan *et al.* 2020). Apart from cultivated *Brassica*, wild relatives are also a rich reservoir of resistance against multiple diseases including PM; however, they are less explored. At present, eleven accessions of *A. thaliana* are reported to be resistant to PM disease. Therefore, more studies to explore PM resistance in wild relatives are necessary to fully exploit their genetic potential. Furthermore, there is a need for development of more robust screening techniques to categorize germplasm more accurately from immune to susceptible. The present screening methods involve phenotyping of the genotypes based on the observation of physical symptoms post PM inoculation through staple leaf method or conidial dusting with sterile brush (Bhosle *et al*. 2021) followed by categorization under the rating scale ranging from 0 (immune) to 9 (highly susceptible), as recommended by the AICRP-RM in India. This often has led to inaccuracies and discrepancies in the observations and inconsistency during the repeated experiments. Therefore, incorporation of molecular approaches like LAMP (Loop-Mediated Isothermal Amplification) and LFA (Lateral Flow Assay) assays can offer more rapid and accurate diagnosis of the pathogen and hence better characterization of the germplasm is possible based on the quantification of disease severity (Attaluri and Dharavath 2023).

Table 3. Genetic Resources for Resistance in *Brassica* Species against Powdery Mildew Pathogen

Species/ Varieties	Genotypes/Accessions	Reaction to *E. cruciferarum*	Location	Screening Methodology	Reference
B. juncea	IJWHJ 001, PCR 9201, RK 8602, RAUD 101, DIR 621, PCR 10, RK 8615, YSPB 24	Resistance	Uttar Pradesh, India	Natural field condition	Singh and Singh 2003
	JM06009, JM06012, JM06014, JM06015	Resistance	Australia	Natural field condition	Singh *et al.* 2010
	PBC-2004-1, EC-414309, EC-399299, PBC-9221, NPC-14, NUDB-26-11, BAUSM-92-1-1, EC-339000, ONK-1, EC-338997, GSL-1, HNS-004, NRCDR-515, NRCR-837, PBC-2002-2, NPN-1, NPC-15, RGN-55, OCN-3, CAN-133,	Resistance	Ayodhya, Uttar Pradesh, India	Natural field condition	Singh 2016
	Sahara CL, Xceed X121 CL	Resistance	Australia	Artificial	Uloth *et al.*

				inoculated condition	2016
	DIR-1507, DIR-1522	Resistance	Hisar, India	Natural field condition	Dang *et al.* 2000
	TM 18, RM 505, NPJ-143, PRD 2013-3, DRMR 1-5	Resistance	India	Natural field condition	Meena *et al.* 2019
	RDV29, RDV 21 (IC0589658, ICGR20041)	Resistance	Tamil Nadu, India	Natural field condition	Nanjundan *et al.* 2020, 2021
	GM-3 and Swarna Jyoti	Resistance	Polasa, Jagtial, Telangana (India)	Natural field condition	Lavanya *et al.* 2023
B. napus	Hyola 650TT, Bravo TT, Tumby, Narendra and GS-7027, Midas, Tower Trooper, Summit,	Resistance	Australia, Hisar (India)	Artificial inoculated condition	Dang *et al.* 2000; Uloth *et al.* 2016
B. napus* ssp. *rapifera	UG4 and UG3	Resistance	Ontario, Canada	Artificial inoculated condition	Shattuck 1993
B. rapa	PPBR-2, EC-414299	Resistance	India	Natural field condition	Saharan *et al*. 2019
***B. rapa* Toria**	PT-2006-4, RMT-10-7, and PT-303	Resistance	India	Natural field condition	Saharan *et al*. 2019
***B. rapa* Yellow sarson**	YSPb-24, TH-68	Resistance	India	Natural field condition	Mehta *et al.* 2008
***B. rapa* Brown sarson**	BSH-1	Moderate resistance	India	Natural field condition	Mehta *et al.* 2008
B. carinata	DLSC 1, HC-9603, HC 1, HC-2 PBC-9221, PI 360883, PBC-2002, DRMR 243, DRMR 261, NPC-16, NPC-21, DRMR-316, DRMR-100	Resistance	India	Natural field condition	Tonquc and Griffiths 2004; Mehta *et al.* 2008
B. juncea, B. carinata, B. napus, Eruca sativa* and *B. rapa)	UDN-11-32	Resistance	Kanpur, India	Natural field condition	Kumar *et al.* 2017
	UDN-11-03, UDN-11-28, and UDN-11-26	Moderate Resistance	Kanpur, India	Natural field condition	Kumar *et al.* 2017
	UDN-18- 25	High Resistance	Chhattisgarh, India	Natural field condition	Chadar *et al.* 2020
	UDN18.1, UDN18.5, UDN18.6, UDN18.8, UDN18.12, UDN18.21, UDN18.25 UDN18.31, UDN18.36, UDN18.42, UDN18.43, UDN18.47, UDN18.54, UDN18.56, UDN18.58 and UDN18.59	Resistance	Chhattisgarh, India	Natural field condition	Chadar *et al.* 2020
	UDN18.24, UDN18.34, UDN18.40, UDN18.44, UDN18.48, UDN18.50, UDN18.56 and UDN18.61	Immune	Chhattisgarh, India	Natural field condition	Chadar *et al.* 2020
Arabidopsis thaliana	La-0, Se-0, C24, Stw-0, Te-0, Co-1, Wa-1, Su-0Su Kas-1, SI-0	Resistance		Natural field condition	Adam *et al.* 1999

Inheritance pattern of PM resistance in Brassica species

Introgression or crossings have been developed widely to study the inheritance pattern of resistance in *Brassica* species for PM disease resistance (Alkooranee *et al.* 2015). Early breeding experiments crossing a susceptible parent from *B. juncea* with the resistant parent belonging to *B. carinata* (Varuna × PCC2; RH 30 × HC-1) demonstrated that one dominant gene provides wide resistance against multiple pathogens (PM, white rust and Alternaria blight disease) in *B. carinata* (Kumar *et al.* 2002). In a recent study, accessions of *B. napus* and other species were screened, and a *B. carinata* cv. 'white flower' was identified immune for PM under natural and *in-vitro* conditions (Gong *et al.* 2020). Hybridization of this *B. carinata* with the elite cultivar of *B. napus* 'Zhongshuang11' generated F_1 hybrids that inherited cytoplasm from the resistant parent (*B. carinata*). The progenies were backcrossed to yield five and a single line from BC_1F_3 and BC_2F_2 generation, respectively, that showed highly resistance to moderate resistance against the PM disease. Moreover, these lines exhibited similar seed quality and morphological traits to 'Zhongshuang11', indicating successful introgression of resistance genes into *B. napus*. In India *B. juncea*, one accession, namely RDV29, was found to be completely resistant to the PM disease. The genetic analysis of populations obtained by crossing the resistant parent 'RDV29' with susceptible parent 'RSEJ775' was made. The screening and evaluation of F1 generation showed that the PM resistance in RDV29 is semi-dominant in nature and determined by two unlinked loci. The segregation ratios in F2 (9:6:1; resistant: susceptible: highly susceptible), susceptible backcross (1:2:1; partially resistant: susceptible: highly susceptible), and resistant backcross (all resistant) further confirmed this statement. Furthermore, it was also inferred from the data that the expression of the resistance depends on the gene dosage (Nanjundan *et al.* 2020). Similar findings were previously reported in *A. thaliana*, where resistance to PM was governed by a locus in 5 genotypes and by two distant loci in single germplasm. In some cases, resistance was encoded by semi-dominant alleles, while in others, susceptibility by dominant alleles (Adam and Somerville 1996). The locus RPW8 in *A. thaliana* consist of two resistance genes (RPW8.1 and RPW8.2) that are of dominant nature and confers broad range resistance to PM (Xiao *et al.* 2001). These reports collectively shed light on the complex and variable nature of resistance against the PM disease in *Brassica* crops, demonstrating that effective resistance can arise from single dominant genes, semi-dominant alleles, or multiple loci, thus offering insightful strategies for future breeding plans targeted at improving *Brassica* crops.

Mechanism of resistance against PM in Brassica spp.

Exposure of *Brassica* plants to *E. cruciferarum* triggers the initial defense mechanisms against pathogen infection and proliferation. These include physical barriers such as wax, cuticle, epidermal cell wall, stomata, leaf hairs, and thick-walled tissues, that prevent pathogen entry. In cruciferous plants, resistance to *E. cruciferarum* before penetration is mainly provided by the cuticle and waxes (Malinovsky *et al.* 2014). Microscopic and transcriptome analyses were performed for PM resistance in the progenies of inter-specific crosses between resistant *B. carinata* and susceptible parent *B. napus* and found formation of needle-like and few flaky particles on the leaf wax of progenies that showed resistance. Furthermore, the study also found elevated expression of genes involved in biosynthesis of wax (*CER*, *KCS6*, *MAH1*, and *LACS2*), which are necessary for modulating certain wax components in resistant genotypes (Zhang *et al.* 2022). Additionally, several cell wall integrity genes, such as PGIP1, PMR5, RWA2,

PDCB1, C/VIF2, and PMEI9, were also observed to be upregulated. In contrast, low callose deposition in resistant progenies as compared to in susceptible ones was observed in the study, perhaps due to initiation of other structural responses deterring the pathogen entry in resistant genotypes. The histopathological and phenotypic studies revealed that necrosis as a result of PM disease was observed higher after infection in *Camelina sativa* and *B. juncea* as compared to *Sinapis alba* (Mir *et al.* 2023). This cell death was apprehended due to antioxidant enzyme activities in these species following infection by *E. cruciferarum* as corresponding to the cell death, the enzyme activity was observed to be relatively higher in *C. sativa* and *B. juncea* in comparison with *S. alba.* However, if the pathogen overcomes these initial defensive layers, it encounters a more systematic defense response through proven mechanisms *viz*., ETI and PTI. Similar mechanism of resistance was documented in other species of PM pathogen (Muthamilarasan and Prasad 2013). A form of cell death as HR (hypersensitive reactions) was reported in tomato powdery mildew interactions. This HR reaction is due to the interaction among pathogen avirulence (*avr*) factors and plant proteins (Nimchuk *et al.* 2003). There are two forms of HR responses in response to *Oidium neolycopersici* on tomato. The single cell HR reactions and fast HR reactions occur in the presence of two different genes, *i.e*., Ol-4 and Ol-6, respectively, which could happen if the epidermal cells are infected by the haustoria (Huang *et al.* 1998; Bai *et al.* 2005). Tomato plants carrying three Ol-genes (Ol-1,3, and 5) are associated with three accessions of *Solanum habrochaites* (Li *et al.* 2007). In *Arabidopsis thaliana* CPR5 mutants, hypersensitive response is accompanied by spatial and temporal overexpression of CEP1 in a specific manner, coinciding with the spontaneous cell death. However, it is noteworthy that while CEP1's involvement in programmed cell death (PCD) is regulated by CPR5 and induced by *E. cruciferarum*, it is not essential for CPR5 mutants to show elevated amounts of resistance to PM (Misas-Villamil *et al.* 2016; Howing *et al.* 2017). The inherent powdery mildew resistance genes with crop plants encode different physiological responses in host cells and result in different levels of resistance. Similarly, Indian mustard cultivars also comprise various species and accessions from *Brassica.* Therefore, there are possibilities of different genes that could respond to such HR reactions, which need thorough investigations.

Biochemical indices associated with resistance against powdery mildew infections

Cruciferous plants generally synthesize biochemical molecules upon PM pathogen's infection as defense response. In response to the pathogen penetration, it has been observed that the infected cells of *Arabidopsis* and other crucifer develop cell wall appositions (CWAs) that not only provide physical reinforcements but also act as a chemical antimicrobial barrier against *E. cruciferarum* (Hardham *et al.* 2007; Huckelhoven 2007). Post-penetration, the changes in biochemistry of the host plant come as part of SAR and ISR response, where the ISR pathway is arbitrated by NPR1 gene and is ultimately crucial for disease resistance and responds to changes in ethylene and jasmonic acid, while the SAR pathway is regulated by salicylic acid (SA) accumulation (Choudhary *et al.* 2007). This pathway usually regulates the PR proteins, such as PR1, PR2, and PR5, while the JA-ET pathway controls different defense genes, including PDF1.2 and PR3 in *A. thaliana.* Additionally, the plant cytochrome P450 gene also responds to the powdery mildew infection by encoding defense-related biochemical enzymes. In *Arabidopsis*, the mutants lacking the CYP83A1 gene were found to be involved in synthesizing plant chemicals that exhibit resistance to PM (Weis *et al.* 2013). It was then found that this gene (CYP83A1) aided in producing glucosinolates that are key molecules involved in host defense

mechanisms (Bak *et al.* 2001). Glucosinolates have a key role in both deterring or attracting insects and combatting diseases such as powdery mildew (Bednarek and Osbourn 2009). Without sufficient glucosinolates, the tested pathogen struggles to infect *Brassicaceae* plants, reliant on these compounds for host recognition and penetration (Weis *et al.* 2014). Another tryptophan-derived compound called camalexin is also believed to contribute to enhanced PM resistance. Its production relies on several cytochrome P450 enzymes, *viz*. CYP71A13, CYP79B2, and CYP71B15 (associated with the camalexin and phytoalexin deficiency) (Nafisi *et al.* 2007; Schuhegger *et al.* 2007a, 2007b). Mutations within these genes usually hamper the camalexin production, therefore leading to reduced resistance (Saharan *et al.* 2019). These insights underscore the multifaceted nature of powdery mildew resistance in cruciferous plants, where diverse biochemical pathways and genetic components, including glucosinolates, camalexin, and PMR genes, collectively contribute to complex and dynamic defense responses against pathogen invasion.

Molecular events involved in resistance in Brassica species in response to E. cruciferarum

Although the histological or cellular reactions and the biochemical molecules are associated with some of the defense responses to PM infections, the genomic or molecular interactions are the ultimate end process for ascertaining the host resistance types. The evolutionary mechanism that helps maintain homologous R genes in *B. napus*, which provide broad spectrum resistance for PM pathogen, have been previously studied. In *B. rapa*, one gene and three genes in *B. oleracea*, have been identified as being similar to RPW8 (Li *et al.* 2016). Additionally, two loci RPW6 and RPW7 were located on chromosome 5 and 3, respectively, in *A. thaliana* genome that showed independent dominant inheritance against PM disease (Xiao *et al.* 1997). Most of the characterized R genes in *A. thaliana* for powdery mildew are C-terminal NB-LRRs, while there is a smaller number of N-terminal coiled-coil motif superfamily and transmembrane domains. Key genes include EDS1, EDR1, PAD4, EDS5, NPR1, EIN2, RAR1, COI1, SGT1b, PBS3, NDR1, and RPW8 (Xiao *et al.* 2005). In *Arabidopsis*, genetic screenings have also led to the identification of six recessive loci of PM resistant mutants (PMR1 to PMR6), with genes PMR2, PMR4/GSL5, PMR5, and PMR6 having been partially characterized in *Arabidopsis* (Vogel and Somerville 2000; Jacobs *et al*. 2003; Nishimura *et al.* 2003; Consonni *et al*. 2006). These mutants have shown increased resistance against PM disease. Furthermore, the susceptibility gene containing MLO locus was also screened and characterized in *Brassica* with respect to its sequence similarity with barley MLO and identified as a susceptibility gene (Consonni *et al*. 2006). Chandran *et al*. (2009) elucidated 67 transcription factors with altered expression at the PM infection site, revealing that MYB3R4 acts as a regulator for transcription, thus influencing host endo-reduplication at the infection site. This combination of homologous R genes, transcription factors, and resistance-related loci work together to provide robust defense against *E. cruciferarum* and other PM pathogens.

Non-host Resistance (NHR)

The most prevalent and important plant immunity, known as NHR, offers a broad-spectrum resistance to multiple pathogens (Mysore and Ryu 2004; Thordal-Christensen 2003). Such resistance prevents infections from pathogens that did not co-evolve alongside the host, mainly due to the lack of adapted fungal effectors or the presence of numerous resistance genes (Jones and Dangl 2006).

The NHR functions against fungal pathogens that are non-adapted like PM. The NHR has an independent, multi-component defense system, *i.e.*, pre- and post-invasion immunities (Lipka *et al.* 2005; Wiermer *et al.* 2005; Stein *et al.* 2006; Meena *et al.* 2018). *Arabidopsis* NPR1 genes, which are important for systemic acquired resistance, are essential for the regulation of NHR (Chen *et al.* 2013; Zhong *et al.* 2015).

Previous studies on *Arabidopsis* have shown that it exhibits resistance to PMs, such as *Blumeria graminis* and *E. pisi*, that are not adapted to *A. thaliana*. These function through strong pre-invasion defenses that are mediated by PEN1 to PEN4 genes (Collins *et al.* 2003; Stein *et al.* 2006). The gene PEN1 codes a protein called syntaxin that prevents fungal penetration by forming ternary SNARE complexes (Assaad *et al.* 2004; Bhat *et al.* 2005; Kwon *et al.* 2008). PEN 2 and PEN 3 proteins also play roles in penetration resistance, with PEN 2 demonstrating myrosinase activity that can impede fungal infection (Lipka *et al.* 2005; Stein *et al.* 2006).

While the NHR response in pre-invasion stage is mediated by PEN (penetration gene), in the post-invasion stage, it is regulated by PM pathogen genes with increased disease susceptibility, phytoalexin-deficient, and genes associated with senescence (Saharan *et al.* 2019). The dual role of non-host resistance, where pre- and post-invasion immunity, regulated by various pathogen-related genes, collectively offer robust protection against non-adapted powdery mildew pathogens.

Genes Governing Intrinsic Resistance in *Brassica* Species and their Mechanisms Against *E. cruciferarum*

The resistance often has been controlled by Mendelian genes (Biffen 1905). Many research outcomes have accumulated the knowledge on different basis of plant resistance (Lucas 2011; Russel 2013). However, limited efforts have been made to explore this basis content for developing durable resistant genotypes. Recent advancements in genomic and transcript analysis techniques have significantly contributed to understanding the molecular pathway that contributes toward resistance against PM. Identified genes represent both SAR and NHR pathways and belong to R- gene (RPW8), SA signaling pathways (NPR1 and NPR2), calmodulin binding protein (MLO), transcription factors (WRKY70), *etc*. (Table 4).

A recent transcriptome analysis using RNA-seq data on two *B. napus* cultivars, displaying a contrasting range of resistance against powdery mildew disease, found that gene expressions involved during pectin modification and degradation were at elevated levels in resistant cultivar in comparison to susceptible. These genes included PM resistant5 (PMR5), polygalacturonase inhibitor1 (PGIP1), pectin methyl esterase inhibitor 9 (PMEI9), and reduced wall acetylation2 (RWA2) (Zhang *et al.* 2022). Studies conducted in *Arabidopsis* and other crops have recently confirmed that members of these gene families, such as PMR, PGIP, PMEI, and RWA, are strongly linked with susceptibility or resistance to pathogen (Engelsdorf *et al.* 2017; Lionetti *et al.* 2017). These studies highlight specific gene families and molecular pathways that play a key role in developing durable resistance against powdery mildew, emphasizing the potential for utilizing advanced genomic techniques to enhance resistance breeding strategies in *Brassica* species and other crops.

Table 4. Genes Along with their Functions Conferring Resistance in Crucifer's Species Against Powdery Mildews

Host Species	Genes Controlling Host Resistance	Mechanism of Action/ Function	References
Arabidopsis thaliana	MLO genes (mlo2, mlo6, mlo12) Triple mutant	Confer wide range R by altered cell wall composition	Consonni *et al.* 2006
A. thaliana	NPR1, NPR2	Regulators of SAR	Zhang *et al.* 2003
B. juncea	NPR1	Activates SAR	Ali *et al.* 2017
B. rapa and *B. oleracea*	BnHR	Resistance to PM	Lie *et al.* 2016
A. thaliana	RPW8.1, RPW8.2	R through SHL or HR	Xiao *et al.* 2001, 2003
A. thaliana	CPR5	Control R through PCD	Misas-Villamil *et al.* 2016; Howing *et al.* 2017
A. thaliana	PAD3, WRKY33d, and Cyp 83 a-1-3 mutant	Elevated amounts of camalexin for R	Qiu *et al.* 2008; Pandey *et al.* 2010; Mao *et al.* 2011; Weis *et al.* 2013
B. juncea	AtMLO6, AtMLO2, AtMLO12, AtROP-regulated AtRLCK VIA3	Provide basal R	Saharan *et al.* 2019
A. thaliana	PEN genes, EDS1, PAD4, SAG101	NHR at pre and post--invasion stage	Wiermer *et al.* 2005; Stein *et al.* 2006; Lipka *et al.* 2008
Crucifers	MYB 51 TF	Regulator of glucosinolate biosynthesis genes	Saharan *et al.* 2019
Crucifers	Overexpression of r genes, such as PAD3, PAD4, MLO, PEN, EDR, NPR1, MAPK, MAPK 65-3, PMR, SNARE, RLCKs, ED5, KDLd, and WRKY70	Confer R to PM	Saharan *et al.* 2019
Crucifers	Plant UBX domain-containing protein (PUX) 2	Fungal reproduction and growth	Rancour *et al.* 2004
Crucifers	Bax inhibitor 1s	Support PM penetration and development	Huckelhoven *et al.* 2004; Babaeizad *et al.* 2009; Eichmann *et al.* 2010
Crucifers	Lifeguard (LEG) proteins	Support PM penetration and development	Reimers *et al.* 2006, 2008; Hu *et al.* 2009
Crucifers	NPR1, PD4	Exhibit enhanced S	Xiao *et al.* 2005
Crucifers	nah G (transgenic)	Exhibit enhanced S	Reuber *et al.* 1998
Crucifers	Receptor-like cytoplasmic kinases) VIA3 mutant	Enhanced fungal growth	Saharan *et al.* 2009
A. thaliana	Penetration gene 1, VAMP	Increase PM penetration	Kim *et al.* 2014
A. thaliana	wrky40 mutants, wrky18	Enhanced camalexin	Affolter *et al.* 2008; Beers *et al.* 2004
A. thaliana	MLO2, MLO6, and MLO12	Suppress basal defense and modulate infection process	Panstruga 2005
A. thaliana	Soluble carbohydrate elicitor	Decreases fungal growth	Schweizer *et al.* 2000
A. thaliana	Chito-octamer	Elicitor of plant defenses	Ramonell *et al.* 2002
A. thaliana	Chitonases, glucanases,	Important role in PM resistance	Makandar *et al.* 2006

	thaumatins, defensins (PR proteins)		
A. thaliana	PAMPs, PRRs	Activate immune responses	Niks and Marcel 2009; Boller and Felix 2009
A. thaliana	CERK1	Contributes to basal R	Wan *et al.* 2008
A. thaliana	KDEL, Cys EP, CEP1	Provide R during post-penetration	Howing *et al.* 2017
A. thaliana	SR 1	Regulates NDR1 expression and EIN 3	Nie *et al.* 2012
A. thaliana	PMR4/GSL5	Callose synthesis (a physical barrier)	Ellinger *et al.* 2013
A. thaliana	AtL31, overexpression, RABA4,	Enhanced penetration	Maekawa *et al.* 2014; Ellinger *et al.* 2014

***Abbreviations:** KDEL-CysEPs- C-terminal KDL endoplasmic reticulum retention signal with cysteine endopeptidases from Castor bean; CEP- Constitutive expression of protein; PMR PM-resistant; EDR-Enhanced disease resistance; CERK- Chitin elicitor receptor kinase gene; SR-Signal-responsive; PAD- Phytoalexin-deficient; VAMPs-Vesicle-associated membrane protein; RLCKs- Receptor-like cytoplasmic kinases and EIN-Ethylene-insensitive, and SNAREs- Soluble N-ethylmaleimide–sensitive factor attachment protein receptors

Major R Genes Identified for *Brassica* Powdery Mildew Resistance

Status of RPW8 gene in cultivated Brassica species

The major concern to ascertain durable resistance is whether resistance genes (R) after undergoing diploidization remain present or are lost in *Brassica* species. For instance, it was discovered that *B. rapa* and *B. oleracea* each have four homologs of the RPW8 gene. However, in *B. napus* (Bn), which is a cross between *B. oleracea* (Bo) and *B. rapa* (Br), seven homologs (RPW8) were observed. It was unclear whether these genes were lost in *B. napus* and through what evolutionary process (Li *et al.* 2016). The findings suggested that, although, BoHR homolog originating from *B. oleracea* remained largely unchanged, the BrHR homolog introduced from *B. rapa* displayed comparatively more variability within the genome of *B. napus* due to evolutionary processes such as gene loss, deletion, insertion, substitution, mutation, and intragenic recombination. Furthermore, the BnHR genes, although shared high sequence similarity with BoHR genes, the absence of homologs in *B. napus* accessions was explained through intragenic recombination involving two paralogs and two orthologs. Additionally, subcellular localization studies were done by fusing truncated BnHRa and BnHRb at the C-terminus, as well as full length BnHR with Yellow Fluorescent Protein (YFP). It was found that the BnHRb-YFP and BnHRa-YFP predominantly present at the extra-haustorial membrane encompassing the haustorium of PM pathogen. The study also revealed the role of these genes in induction of cell death leading to improved resistance against PM disease in *Arabidopsis* by ectopic expression studies (Li *et al.* 2016).

BjNPR1 gene

The non-expressor of PR genes family, having NPR1, NPR3, and NPR4, is extremely important in signaling pathway of the salicylic acid (SA) for plant defense. NPR1 acts as a positive regulator by activating (PR) genes upon SA accumulation during pathogen infection. Both NPR3 and NPR4 serve as negative regulators and SA receptors, controlling NPR1 activity by facilitating its degradation, ensuring a balanced immune response. The overexpressed BjNPR1 in transgenic *B. juncea* presented increased resistance to *E. cruciferarum* and *A. brassicae*, its involvement in broad-spectrum disease resistance is shown by its delayed symptom development and decreased disease severity (Ali *et al.* 2017).

Mildew Locus O (MLO) proteins

The MLO gene family plays a pivotal role in pathogen-host interactions and offering resistance to PM diseases. A transmembrane protein that the wild-type MLO gene produces is implicated in increasing powdery mildew susceptibility by aiding fungal entry and establishment within the host plant. This susceptibility pathway is disrupted by the loss-of-function mutations in MLO genes, which strengthens the plant defense against the pathogen. The significance of the MLO gene in improving plant resistance to PM has been highlighted by recent developments in gene editing technology (Shi *et al.* 2022). In *Arabidopsis*, studies have identified PMR2, which encodes MLO2, as a crucial regulator promoting compatibility between PM species (Consonni *et al.* 2006). MLO-2 aids in PM pathogen entry into plant cells, along with the close paralogs MLO6 and MLO12 (Panstruga 2005). These MLO genes are part of membrane proteins having a conserved role in plant defense (Devoto *et al.* 2003). *Arabidopsis* possesses a group of 15 MLO gene, among which MLO2, MLO6, and MLO12 are particularly influential in PM compatibility (Collins *et al.* 2003). The MLO proteins interact with calmodulin, influencing defense responses independently of signaling pathways such as SA or JA/ET (Bhat *et al.* 2005). The presence of MLO is crucial for *E. cruciferarum* infection, with pathogens utilizing MLO functions to inhibit host defense responses (Panstruga 2005). In rapeseed , mutating the BnMLO6 gene resulted in significant resistance to *E. cruciferarum* and *Sclerotinia sclerotiorum* (Shi *et al.* 2022). Moreover, resistant plants showed lower expression levels of the susceptibility genes MLO6 and MLO12, demonstrating their critical function in PM resistance (Zhang *et al.* 2022). Triple mutant devoid of AtMLO2, AtMLO6, and AtMLO12 exhibited nearly total resistance against *E. cruciferarum* infections (Consonni *et al.* 2006). Above-mentioned studies demonstrate the critical role of MLO genes in facilitating PM susceptibility and highlight the potential of gene-editing technologies, like CRISPR/Cas9, to disrupt MLO pathways and significantly enhance resistance to powdery mildew in various crops.

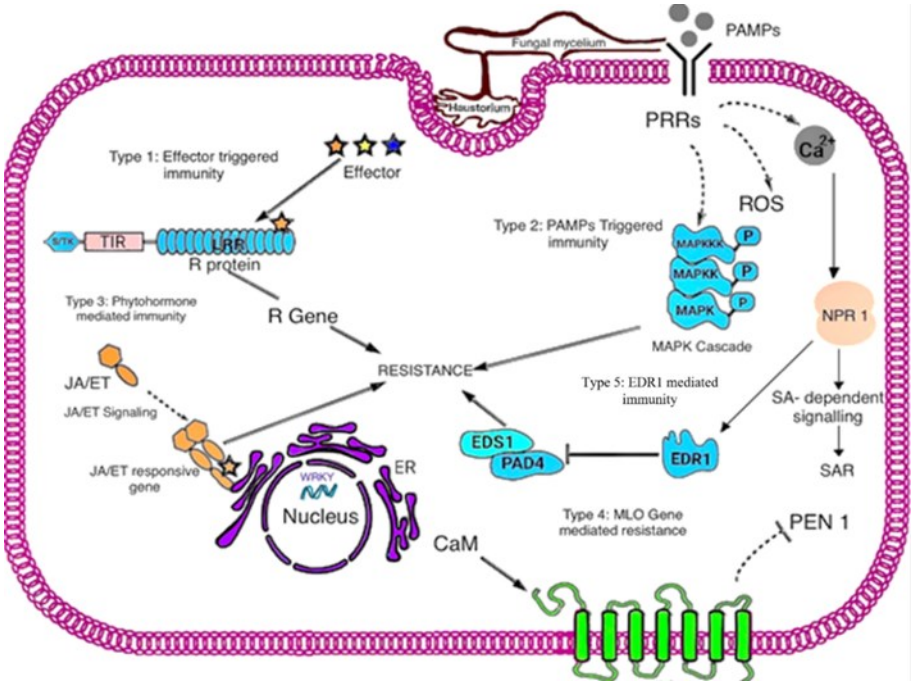

Fig. 2. Image describing different pathways for resistance against powdery mildew pathogen

Upon pathogenic invasion, powdery mildew pathogens release PAMPs and DAMPs. These molecules are recognized by PRRs present on the host plant cell surface, triggering a MAPK cascade that leads to a plant defense response. Simultaneously, the pathogen releases effector molecules that interact with resistance (R) proteins, typically containing LRR, (TIR), and S/TK domains, activating R genes to halt pathogen spread. Another defense pathway involves calcium signaling, which activates NPR1 protein, leading to the accumulation of salicylic acid (SA)-responsive transcription factors such as WRKY and AP2/ERF. Plants with the EDR1 mutation show increased expression of genes linked to the endomembrane system and reactive oxygen species (ROS) generation, indicating that EDR1-mediated immunity against powdery mildew may rely heavily on the secretory pathway. Furthermore, the MLO (Mildew Locus O) gene family plays an essential part in pathogen-plant interactions; MLO genes of the wild type increase vulnerability by enabling fungal entry. This susceptibility pathway is disrupted by loss-of-function mutations in MLO genes, strengthening the plant's defense against the pathogen.

Arabidopsis RPW8

Crop breeding has made considerable use of disease resistance genes (R-genes) to create durable disease-resistant cultivars to reduce crop losses by diseases. Resistance to PM fungus can be introgressed in many plant species, and R-genes inherited dominantly or semi-dominantly provide unique protection as a supplementary defense mechanism (Bai *et al.* 2005; Marone *et al.* 2013). Plant populations naturally contain numerous allelic variants of R-genes. Previous studies indicate PM resistance in the model plant *Arabidopsis* is often polygenic, with the RPW8 gene being a significant QTL. Subsequent studies identified additional RPW genes ranging from RPW6 to RPW13 and also including semi-dominant resistance loci, RPW1, RPW2, RPW4, and RPW5, mapped on chromosomes 2, 3, 4, and 5, respectively (Adam *et al.* 1996; Wilson *et al.* 2001). However, RPW8 emerged as a vital contributor to natural resistance and is present in accessions such as Shahdara, Co3, Do00, Ei4, 5, Kas1, Ms0, Nok3, and Wa11 (Wilson *et al.* 2001; Xiao *et al.* 2001; Gollner *et al.* 2008). The RPW8 locus in the accession Ma0 consists of two genes, RPW8.1 and RPW8.2. The combination provides resistance to PM pathogens (Xiao *et al.* 2001). Unlike typical R genes that offer isolate-specific resistance (Martin *et al*. 2003), RPW8-mediated resistance activates PR genes, leading to a hypersensitive response (HR) marked by H_2O_2 accumulation, cell death, and callose deposition upon pathogen attack (Xiao *et al.* 2001; Xiao *et al.* 2003; Xiao *et al.* 2005; Gollner *et al.* 2008). Additionally, enhanced gene expression of these has been linked to formation of necrotic spots associated with hypersensitive response-like activity induced by elevated SA levels, in the transgenic line S24 (Saharan *et al.* 2019). RPW8.1 is a broad-spectrum resistance gene that balances plant immunity by feedback regulation of WRKY51 transcription factor (Yang *et al.* 2024). Furthermore, another study has also shown that the promoters of RPW8.1 and RPW8.2 genes are essential for resistance (Xiao *et al.* 2003). These insights into the role of RPW8 and its associated allelic variants underscore the potential for harnessing these genes in breeding programs to develop durable, broad-spectrum resistance against powdery mildew in various crops, ultimately improving agricultural resilience and productivity.

Transcription Factors (TFs) Regulating Resistance in *Brassica* spp. against *E. cruciferarum*

Transcriptional regulators in plants can be activated either directly through pathogen receptors or *via* signal transduction by MAP kinases (Shen *et al.* 2007). In

Arabidopsis, WRKY TFs, such as WRKY18, WRKY33, and WRKY40, are crucial for camalexin biosynthesis, contributing to powdery mildew (PM) resistance (Qiu *et al.* 2008; Pandey *et al.* 2010; Mao *et al.* 2011). WRKY70 is necessary for activating SA pathway and provides resistance against multiple pathogens. Interestingly, WRKY-18 and 40 also plays an important role by negatively regulating defense genes against *Golovinomyces orontii*, with double mutants showing complete resistance to infection (Shen *et al.* 2007). In barley, WRKY1 and WRKY2, which are homologous to WRKY18 and WRKY40, interact with MLA immune receptors (Shen *et al.* 2007). Under normal conditions, they suppress defense genes, but upon pathogen detection, MLA displaces them, enhancing defense against *Blumeria graminis* f. sp. *hordei* (Bgh). NAC TFs, such as the barley gene NAC6 and its homolog ATAF1 in *Arabidopsis*, are also key regulators of early defense responses to PM (Jensen *et al.* 2007). Silencing these genes reduces resistance, while over-expression enhances it, indicating their role in pre-haustorial defense mechanisms. In cruciferous plants, WRKY transcription factors and the overexpression of various resistance genes (*e.g.*, MLO, PEN, PMR, MAPK, EDR, PAD3, MPK3, MPR1, EDS5, SNARE, PAD4, RLCKs, and KDL) are critical for effective defense against PM. These studies highlight the pivotal role of transcription factors, particularly WRKY and NAC, along with various resistance genes, in regulating complex defense pathways, highlighting their potential for enhancing powdery mildew resistance through targeted genetic interventions in cruciferous plants (Saharan *et al.* 2019).

Future Strategies on Genetics and Genomic Approaches for Augmenting Resistance in Rapeseed-Mustard Against *E. cruciferarum*

Molecular breeding is of utmost importance for enhancing resistance to pests and diseases in Brassicaceae crops. It involves precise breeding, enrichment of genetic diversity, broad-spectrum resistance to multiple pathogens, and development of climate-resilient genotypes, *etc*. Interspecific hybridization within the *Brassica* genus shows promising results for crop enhancement due to the close genetic relationships between species. For instance, *B. napus* originates from the wide hybridization among *B. oleracea* and *B. rapa* resulting in a distinct species. In contrast, other species such as *B. juncea*, *B. carinata*, and *B. nigra* share common genomes. The PM resistance was introduced in *B. oleracea*, their BC_1 progeny and interspecific hybrid plants were produced using embryo culture and sexual crosses procedures by involving accession PI 360883 (*B. carinata*) and *B. oleracea* cultivars Cecile and Titleist. Plant morphology and RAPD evaluation confirmed the origin of *B. carinata*, used as maternal parent to obtain hybrids *via* embryo rescue culture. Amid these populations, eight BC_1 plants and all interspecific hybrids displayed resistance to PM (Tonguc and Griffiths 2004). *B. carinata cv*., 'White flower' was identified as immune under field and greenhouse conditions after the evaluation of 102 germplasm of *B. napus* as well as other Brassicas for resistance to PM disease. Inclusion of *B. carinata* cytoplasm, yellow petals, and male sterility, true F_1 hybrids were produced without embryo rescue. Morphological characteristics, seed quality, and molecular marker analysis confirms the hybrids and their progenies. Breeding lines such as W3PS.1, W7.6, W7.4, W7.1, W8.1, and W8.3 resulted in resistance or moderate resistance reaction to PM (Gong *et al.* 2020). Previous studies identified two semi-dominant genes governing PM resistance in Indian mustard (Kapadia *et al.* 2019). Using molecular markers OI10-B12 and OI10-C01, they distinguished between susceptible and resistant bulks in the cross GM-3 × Pusa Swarnim. These markers are valuable for identifying disease resistance across various *Brassica* species, especially because the 'C' genome might have naturally

introgressed into different genotypes through outcrossing. Molecular markers closely associated with various resistance genes are crucial for enhancing the selection of trait, especially for combining multiple distinct genes within the same genetic background (Tanksley *et al*. 1989). The development of resistant *Brassica* species against diseases can be accelerated by utilizing genetic and molecular marker-based techniques, thereby improving sustainable agriculture and world food security. Transgenic approaches have proven to be effective in enhancing disease resistance in rapeseed mustard (*B. juncea*), a crucial oilseed crop. Through incorporating specific genes known for their defensive properties, genetically modified plants with improved resistance to various pathogens have been developed. An example includes the chitinase gene, which has been demonstrated to provide resistance against fungal infections. Chitin is degraded by chitinase enzymes, which are essential to plant defense. Plants that have their chitinase genes overexpressed are considerably more resistant to fungus-related diseases. In a previous study, the introduction of chitinase gene was done from rice (*Oryza sativa*) into *B. juncea via* Agrobacterium mediated transformation. Transgenic plants of *B. juncea* evaluated for resistance against two predominant fungal pathogens, *i.e*., *A. brassicae* and *Sclerotinia sclerotiorum.* A significant decrease in disease severity was recorded in transgenic lines compared to control plants (Grover *et al.* 2015). Chitinase enzyme effectively degrades the cell wall of fungi and reduces the growth and development of the pathogens. So far advancements in molecular breeding, transgenic, and genome editing approaches can play a crucial role in generating broad-spectrum resistance to PM pathogen.

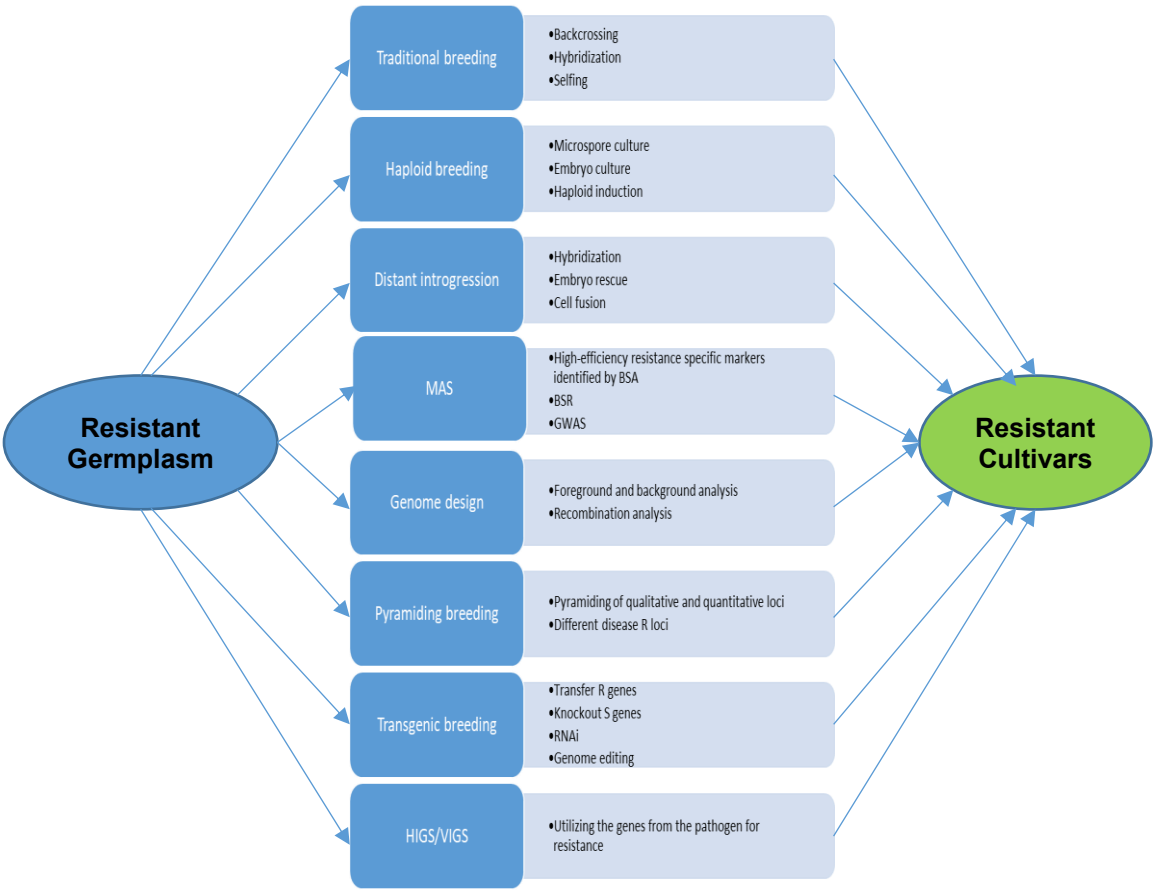

Fig. 3. Proposed genomic era high-efficiency integrated breeding (HIB) model

Genome Editing and CRISPR/Cas9 Technology in *Brassica* Powdery Mildew Resistance

Globally, systematic, strategic, and practical research approaches focusing on the genetic development of cruciferous crop plants draw on a diverse range of associated subjects. The control of many prevailing plant pathogens has been achieved since the development of molecular tools and techniques in the field of molecular plant pathology. Among contemporary methods, an efficient and desirable genetic approach for managing increasingly problematic plant diseases is the modification of intrinsic genetic contents through genome editing technologies. This technology has created more interest within the scientific community due to the effectiveness, versatility, and simplicity associated with the genome editing tool CRISPR (Clustered Interspaced Palindromic Repeats) (Banerjee *et al*. 2023). It offers rapid selection and modification of target genomic areas by deletion or addition of specific base pairs, facilitating the development of desired traits, with special reference to disease resistance genes.

The genome editing (CRISPR) method has shown potential in altering numerous desirable traits in a various agricultural commodity, including rice, wheat, coffee, bananas, cassava, soybeans, and sweet oranges (Prado *et al*. 2024). This approach has also contributed to the introduction of genes conferring resistance to powdery mildew in some crops. For instance, the CRISPR/Cas9 system has been used to generate elite tomato (*Solanum lycopersicum*) lines, with the SlMLO1 knockout line targeting pathogen *Oidium neolycopersici* (Pramanik *et al*. 2021). There are sixteen MLO genes (MLO1 through MLO16) identified as the primary causes of powdery mildew susceptibility. Furthermore, Agrobacterium-mediated transformation is used to insert the DMR1 gene for PM resistance into the aromatic sweet basil plant (*Ocimum basilicum)* (Navet and Tian 2020). The target gene MLO-7 introduced in grapevines to combat the powdery mildew-causing *Uncinula necator* using CRISPR/Cas9 ribonucleoproteins (RNPs) led to enhanced resistance against the powdery mildew disease (Malnoy *et al.* 2016). Although a disease resistance mechanism in *Brassica* crops has not yet been established, a candidate gene, MYB28, has been introduced in broccoli (*B. oleracea*) to increase glucoraphanin content through protoplast transfection using RNPs (Kim *et al*. 2022). By replacing the RGEN RNP gene in place of the BolMYB28 gene, researchers successfully developed a broccoli cultivar with increased glucoraphanin contents. A recently published technique for editing the genome of the mustard crop (*B. juncea*) utilized cotyledon explants to introduce CRISPR components into the plant genome *via* an Agrobacterium-mediated transformation (Ahmad *et al*. 2024). The research output involves an expanded workflow and various steps for recovering genome-edited knockouts, further verification of the edits, and accurate recovery of the transgene-free genome edited plants, providing a robust foundation for future studies on CRISPR/cas9 technology. The emphasis is on developing multiple of disease-resistant mustard cultivars worldwide.

CONCLUSIONS

This comprehensive review has summarized published information about rapeseed-mustard, which is a significant edible oil crop, ranking closer to soybean and palm oil in context of its contribution in the edible oil market, in India. However, this crop faces a lot of challenges from both biotic and abiotic stresses, resulting in substantial losses in quality and production. Among the biotic stress, PM incited by *E. cruciferarum* poses a significant

economic threat in mustard cultivation in India. Severe incidences encompass 17% to 29.5% yield loss in various Indian states. The Indian mustard cultivars grown across different regions of India are highly vulnerable to the infection of powdery mildew pathogen. Researchers worldwide have extensively investigated various aspects of PM in *Brassica* species. Many *Brassica* species and their wild relatives are rich sources of important candidate genes, especially those related to resistance against various stresses including biotrophic diseases. Studies focusing on varietal screening have disclosed the resistance levels of *B. juncea* to *E. cruciferarum*. Additionally, research has explored the genetic and genomic elements contributing to PM resistance in different *B. juncea* genotypes, aiming to offer empirical data and reliable markers for detecting resistant plant materials. The absence of clear scientific information regarding the resistance source for powdery mildew disease in *B. juncea* and its genetic traits has impeded comprehensive research efforts to combat this disease. Therefore, in this paper the authors explicitly reviewed different aspects about *Brassica* powdery mildew, its economic significance, and the cellular and molecular aspects of resistance mechanisms for sustainable management practices. Furthermore, molecular studies using a model plant like *A. thaliana* are crucial to analyze host-parasite interactions with the PM pathogen, particularly in economically significant hosts, such as rapeseed-mustard, Chinese cabbage, broccoli, cauliflower, radish, turnip, horseradish, turnips, kohlrabi, kale, and rape. This review further contributes with a comprehensive understanding of plant defense mechanisms that offer self-sustained crop protection and reduce stress factors, promoting eco-friendly and sustainable crop production.

In future investigations on *B. juncea*, emphasis should be placed on diverse aspects of PM, encompassing host-pathogen interactions, variability, racial profiling, virulence patterns, QTL mapping, and molecular mechanisms underlying pathogenesis. Identifying effector molecules and their corresponding R-genes will further enhance our understanding about resistance mechanisms for future programmes on resistance breeding. Creating innovative pre-breeding materials by utilizing resistant crop wild relatives, whether closely or distantly related, and employing marker-assisted selection methods can streamline *Brassica* improvement initiatives. Implementing strategies to manipulate host factors targeted by the PM pathogen will greatly contribute to the advancement of PM management techniques within *Brassica* crop improvement programs.

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