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REVIEW

Advances in understanding and managing Alternaria leaf blight of *Brassica juncea* (Indian Mustard)

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Brassica juncea (Indian mustard) is a major oilseed crop widely cultivated across the Indian subcontinent for its high oil content and nutritional value. It contributes significantly to India's edible oil economy. Despite its importance, *B. juncea* is vulnerable to several diseases, including white rust, Alternaria blight, stem rot, powdery mildew, and *Orobanche* infestations, all of which threaten yield and oil quality. These biotic stresses, exacerbated by changing climatic conditions, underscore the need to develop disease-resistant cultivars to sustain productivity. Resistance to two notorious necrotrophic pathogens of mustard, *Alternaria brassicae* (Alternaria leaf blight) and *Sclerotinia sclerotiorum* (stem rot), is quantitative, and no complete resistance sources have yet been reported in *B. juncea* germplasm. Wild relatives offer valuable sources of resistance; however, their use in breeding is limited by incompatibility issues in interspecific hybridisation. Research using *Arabidopsis thaliana* as a model system has revealed natural variation and identified some QTLs and candidate genes associated with Alternaria blight resistance, particularly those involved in phenylpropanoid, glucosinolate, and wax biosynthesis pathways. Metabolomic and transcriptomic studies indicate the contribution of scopoletin and the phenylpropanoid pathway to defence. In parallel, pathogen genomics has identified toxins, effectors, and enzymes involved in virulence. Recent advances, including CRISPR/Cas-mediated editing of susceptibility genes, RNA interference, and nonhost resistance strategies, hold promise for developing durable Alternaria-resistant mustard varieties through molecular breeding and biotechnological approaches.

Keywords : *Orobanche,Alternaria brassicae,Sclerotinia sclerotiorum,Arabidopsis thaliana*

INTRODUCTION

Oilseed crops are the second most significant component of India's agricultural sector. *Brassicas* play a substantial role in the country's oil production. Among the *Brassica* species, Indian mustard (*Brassica juncea* (L.) Czern.) occupies the largest cultivated area in India (ICAR-IIRMR).

It is extensively grown in cooler and drier regions during winter (Oct- March). The crop is suited for semi-arid to arid climates, and mainly cultivated under rain-fed conditions. In the Indian subcontinent, it is primarily grown for oil, leafy vegetables, condiments and protein-rich feed stock. Rajasthan is a major producer of mustard, with contributions from states such as Haryana, Madhya Pradesh, Uttar Pradesh, Gujarat, Punjab, West Bengal, and the northeastern state of Assam (ICAR-IIRMR). In 2023-24, the area under rape seed and mustard cultivation in India was 91.83 lakh hectares with an output of 13.2 million tons (ICAR-IIRMR). India has achieved self-sufficiency in most agricultural products; however, we still rely on imports for edible oil (Jat *et al*. 2019). Import of edible oil in 2023-24 was 159.6 million tons. Therefore, increasing the productivity of rapeseed and mustard has become a key focus in agricultural research.

One of the many confounding factors affecting the yield of oilseed mustard is the attack by several pathogens and pests. In India, the major diseases affecting oilseed mustard productivity are Sclerotinia stem rot (*Sclerotinia sclerotiorum*), Alternaria leaf blight (*Alternaria brassicae*), White rust (*Albugo candida*), and Powdery mildew (*Erysiphe cruciferarum*). Insect pests, such as

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aphids and sawflies, as well as parasitic plants like Orobanche, also significantly contribute to reducing crop yields (Fig. 1). The changing climate has exacerbated the situation by increasing both the incidence and spread of various plant diseases. In a recent study, Meena *et al*. (2024) provide a comprehensive picture of the geographical distribution patterns of these diseases in India as well as the impact of changing climatic conditions on disease severity.

This short review summarises the research efforts, including some from our group, towards improving the understanding of the molecular mechanisms of host resistance against Alternaria leaf blight of Brassicas, and the pathogen genomics. The impact of advances in our knowledge of the host – *A. brassicae* interaction on the efforts towards improving resistance against Alternaria in *B. juncea* have also been elucidated. Additionally, the attempts at introgression of resistance identified in the wild crop relatives, as well as the use of genome editing and RNAi-based strategies for improving resistance, are discussed towards the end.

Alternaria brassicae -The causal pathogen

*Alternaria*spp. viz *A. brassicae*, *A. brassicicola*, *A. raphani* and *A. alternata,* are known to pose a global challenge affecting the Brassicaceae crops worldwide. However, *A. brassicae* is considered most destructive on oilseed mustard (Meena *et al*. 2010; Saharan *et al*. 2016a).

The infection generally starts in the lower leaves and then spreads to the younger leaves, the stem, and the siliques (Fig. 2).*A. brassicae* produces large club-shaped- broader at the base and tapering at the top to form a long beak. The multicellular conidia have both longitudinal and transverse septa and contain melanin, which makes them appear dark-coloured(Fig. 3b). The increase in humidity resulting from winter rain can significantly increase the severity of infection during the growing season. The Alternaria blight disease symptoms appear as distinct concentric necrotic lesions surrounded by a yellow halo. As the infection progresses, these lesions enlarge, sometimes coalesce to form large necrotic areas. Severe infections, especially on the silique, significantly reduce seed production and oil content (Meena *et al* 2010; Saharan *et al.*2016a).

Control of A. brassicae by conventional agricultural practices

Managing Alternaria leaf blight in Brassicas remains a complex challenge. Developing resistant cultivars has long been recognised as the most economical and environmentally sustainable solution for disease management. However, despite decades of conventional breeding efforts, durable resistance remains elusive. This limitation arises from the complex, quantitative nature of resistance to *A. brassicae*, as well as the limited availability of well-characterised resistance donors. Additionally, the necrotrophic lifestyle of the pathogen adds another layer of complexity to host–pathogen interactions. Unlike biotrophic pathogens, against which gene-for-gene resistance can be effective, necrotrophs often bypass these mechanisms, making it more challenging to achieve durable resistance.

Fungicides such as azoxystrobin, propiconazole, mancozeb, and carbendazim are effective, especially when applied before the disease onset. Azoxystrobin and propiconazole are among the most effective fungicides for controlling Alternaria blight in brassicas (Purohit *et al.* 2024). Moreover, overuse of these chemical fungicides over time can result in the development of resistant pathogen strains (Rajvanshi *et al,* 2020). Concerns about cost, the timing of application, and environmental impact are compelling agricultural scientists to explore alternative management strategies. Bio-agents such as *Trichoderma viride*, *Trichoderma harzianum*, and *Pseudomonas fluorescens* are widely evaluated for controlling *A*. *brassicae* and *A. brassicicola*. Seed treatment and foliar sprays with these agents significantly reduce disease intensity and improve yield. *Trichoderma viride* and *T. harzianum* are especially effective in inhibiting pathogen growth and reducing disease severity in both laboratory and field conditions (Sanchez-Gomez *et al*. 2025).

Alternaria brassicae – Brassica juncea pathosystem

Large-scale screening of *B. juncea* germplasm, cultivated lines, and landraces has identified

some moderately tolerant lines. The non-availability of a usable source of robust resistance against *A. brassicae* is a major bottleneck limiting the success of the breeding approach to develop Alternaria blight-resistant varieties. Several research groups have explored the wild relatives within the Brassica cenospecies to identify robust sources of *A. brassicae* resistance.The wild relatives of crop plants are generally potential resources for disease resistance genes that can be introgressed into popular varieties to develop biotic and abiotic stress-tolerant crops. Several sources of complete resistance to *A. brassicae*, identified in *Sinapis alba*, *Camelina sativa*, and *Diplotaxiserucoides*, are being explored for introgression into *B. juncea* (Sharma *et al*. 2002; Al-Lami *et al.* 2023). Choudhury *et al.* (2023) screened 94 accessions from 26 wild species for *resistance to A. brassica*e. The team identified several accessions of *Diplotaxis tenuifolia*, *D. erucoides*, *D. muralis*, and *Sinapis arvensis* exhibiting high or complete resistance. The hybrid and amphiploid plants synthesised by crossing these wild relatives with *B. rapa* also exhibit high resistance against Alternaria blight. However, the vast difference in ploidy level, cross incompatibility, and loss of female fertility in interspecific hybrids pose challenges in introgressing the Alternaria blight resistance trait in cultivated B. *juncea*. Vasupalli *et al.* (2017) developed an introgression population by advancing the generations of an interspecific hybridisation between *B. juncea* and D. *erucoides*. To overcome the bottlenecks in wide hybridisations, Kumari *et al*. (2020) used a somatic hybridisation approach to introgress disease resistance and high-temperature tolerance from *Sinapis alba*. The group reported the identification of three QTLs (Abr-01, Abr-02, Abr-03) responsible for blight resistance using a BC, F*ƒ* -„ population derived from *S. alba* × *B. juncea* somatic hybrids (Singh *et al*. 2021).

Arabidopsis as a surrogate host

Arabidopsis thaliana is a premier model for unravelling the molecular basis of plant–pathogen interactions. Its fully sequenced genome, extensive collections of T-DNA insertion mutants, and the comprehensive resources maintained by the Arabidopsis Biological Resource Centre (ABRC; Ohio, USA) make it uniquely suited for investigating the genetic and molecular determinants of host and nonhost responses. To assess its utility as a surrogate host for *A. brassicae*, Mandal *et al*. (2018) conducted a comparative histopathological analysis of *A. brassicae* infection in *A. thaliana* and *B. juncea*. Notably, both compatible interactions exhibited similar disease progression patterns, with pathogen development closely correlated with the accumulation of reactive oxygen species (ROS), extensive host cell death, and callose deposition. These observations demonstrate that *A. thaliana* effectively recapitulates the key features of *A. brassicae* pathogenesis in Brassica crops, thereby providing a powerful experimental system for dissecting host–pathogen crosstalk at the molecular level (Fig.3).

Resistance against Alternaria is quantitative

Like other necrotrophic fungi, monogenic resistance has not been found for Alternaria leaf blight. Resistance in Brassicas is reported to be multilayered, involving both structural and biochemical components. Epicuticular waxes form a physical barrier forming the first layer of defense. The hydrophobic surfaces deters spore adherence and germination. The structural and compositional dissection of the epicuticular waxes in connection with resistance has been studied by several early researchers (Conn and Tewari, 1989). Epicuticular waxes are found to be more abundant in the resistant genotypes than in the susceptible ones. Some of the resistant lines in *B. carinata*, *B. napus*, and *S. alba* have high epicuticular waxes, as opposed to *B. juncea* (Saharan, 2016b, Tomasi and Abdel-Haleem 2023). *A. brassicae,* unlike *A. brassicicola*, predominantly gains access into the host through stomatal opening (Mandal *et al*. 2018) (Fig. 3). and it has been reported that stomatal frequency, distribution and the aperture show a significant correlation with resistance against *A. brassicae* (Saharan *et al*. 2016b). The resistance against *Alternaria* sps is also reported to be associated with increased levels of antioxidant enzymes, including polyphenol oxidases, peroxidases, and catalases (Gupta and Kaushik 2002). In crop wild relatives such as *S. alba*, *C. sativa*, and *Capsella bursa-pastoris*, resistance is linked to the plant's

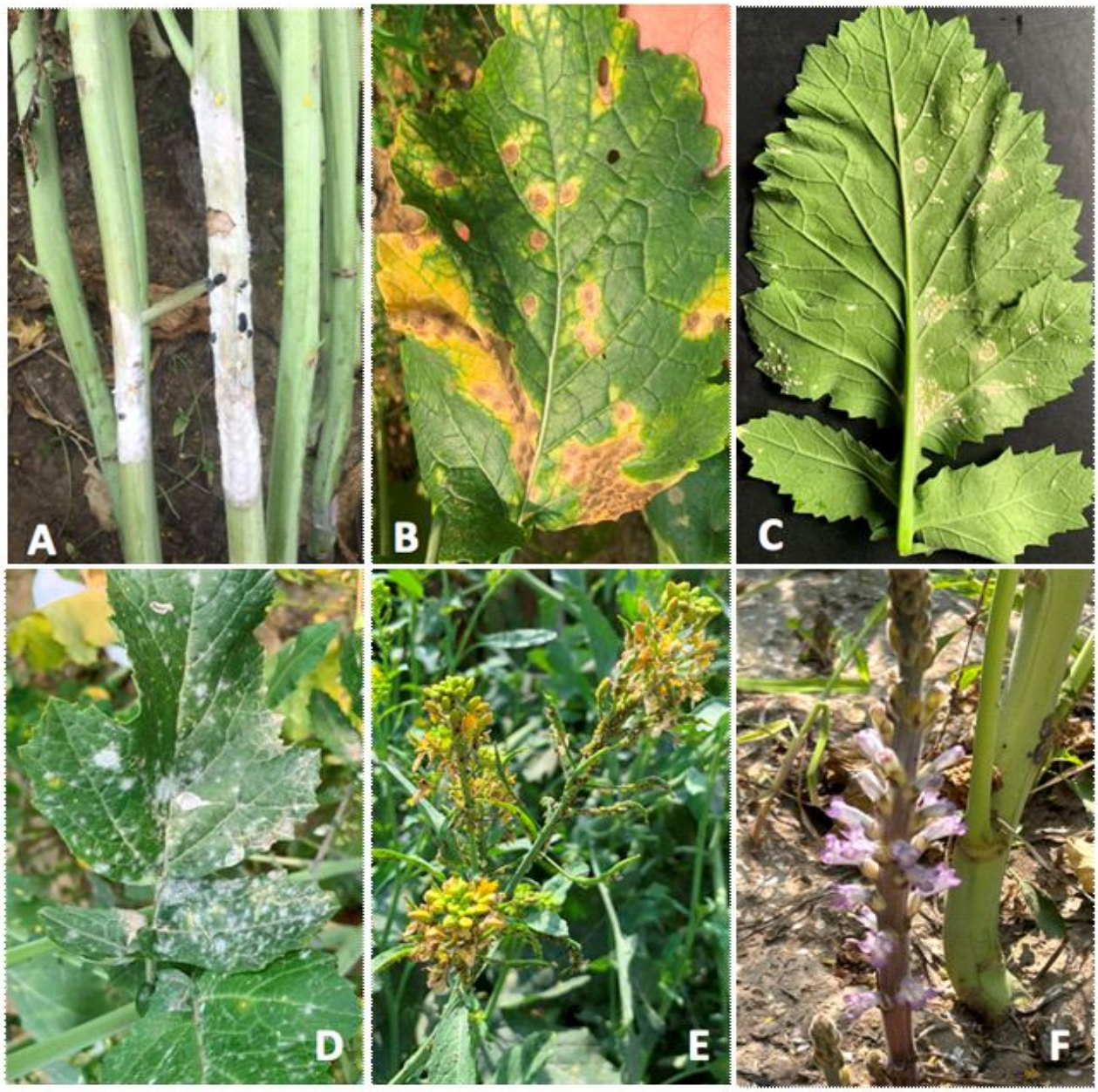

Fig.1: Diseases of *Brassiaca juncea* prevelant in Indian subcontinent with causal organism.(A) Stem rot (*Sclerotinia sclerotiorum*) ,(B) Alternaria leaf blight (*Alternaria brassicae* and *A. brassicicola*) (C) Whiterust (*Albugo candida*) (D) Powdery mildew (*Erysiphe cruciferarum*) (E) Aphid infestation (*Myzuspersicae* S.) (F) Broomrape (*Orobanche*)

ability to degrade/ detoxify the fungal toxin Destruxin B primarily through hydroxylation and glycosylation, coupled with the production of phytoalexin camalexin (Pedras *et al.* 2001, 2023). Due to the large genome size, limited information on genomics, and the tools available for genetic dissection, the molecular players involved in resistance in the wild relatives have yet to be unraveled.

Arabidopsis, a member of the Cruciferae family, has been extensively used to investigate the molecular crosstalk between the host and the pathogen esp. *A. brassiccicola* and to a certain extent *A. brassicae*. Mutants have been used to study the early responses of *A. thaliana* to*A. brassicicola*. Mutants deficient in camalexin production, such as *pad 2*, *pad3*, *pad5*, and JA-signalling compromised mutant- *coi1*, showed enhanced susceptibility to *A. brassicicola* (Van Wees *et al*. 2003). Likewise, the mutants defective in auxin biosynthesis *asa1-1* and *cyp79b2 cyp79b3*, were reported to be more susceptible to *A. brassicicola* infection than WT (Qi *et al.* 2012).

Presence of natural variation in resistance to *A. brassicae* was evident across a panel of *Arabidopsis thaliana* accessions (124 accessions), with phenotypes spanning from strong resistance to high susceptibility (Rajarammohan *et al.* 2018). The continuous nature of this variation further supports the observation that resistance to Alternaria is polygenic and under additive genetic control. Three biparental mapping populations, derived from three resistant accessions crossed with two susceptible accessions, identified six QTLs. Five QTLs were genotype-specific, and one QTL was shared across all populations. The presence of shared and specific/unique QTLs underscores the quantitative nature of resistance. Notably, one QTL showed moderate to large effects explaining nearly 50% of the phenotypic variation, suggesting the presence of a major resistance gene. Identification of a major QTL/gene opens up the possibility of its use in heterologous systems (Rajarammohan *et al*. 2017). The occurrence of distinct major QTLs across populations suggests that different accessions deploy distinct resistance mechanisms. Even the QTL common to all three populations displayed variable effect sizes depending on the genetic background, highlighting the context-dependent contribution of individual alleles. These findings underscore the complexity of the genetic architecture underlying resistance and emphasise the need for fine-mapping and functional validation to identify causal genes and clarify their mechanistic roles (Rajarammohan *et al.* 2017).

A complementary genome-wide association study (GWAS) in Arabidopsis has further refined the genetic basis of resistance by capturing allelic variation at higher resolution. A GWAS of 124 natural accessions revealed several candidate defence-related genes. CYP82G1, a cytochrome P450 monooxygenase involved in volatile homoterpene biosynthesis, was consistently upregulated in resistant accessions, whereas susceptible lines frequently carried alleles with reduced expression or compromised function. Similarly, allelic variants of GORK, a guard cell outward-rectifying K^+ channel, influenced stomatal behaviour and pathogen entry. In addition, a GDSL-motif lipase (At1g06990), previously implicated in defence against necrotrophs, also showed allelic differentiation between resistant and susceptible lines. These candidates exhibit diverse resistance mechanisms, including metabolic defences, structural reinforcement, and stomatal regulation (Rajarammohan *et al.* 2018).

Together, QTL mapping and GWAS highlight the polygenic and quantitative nature of A. brassicae resistance in Arabidopsis. The integration of these approaches, followed by fine-mapping and functional validation, will be crucial in identifying causal genes and unravelling the molecular basis of resistance. Beyond advancing our understanding of necrotrophic defence in Arabidopsis, these findings provide valuable genetic targets for breeding Alternaria blight resistance in Brassica crops.

Insights from a comparative omics and functional genomics

In the absence of Arabidopsis genotypes with contrasting responses to *A. brassicicola* infection, several groups have studied the changes in transcriptional dynamics in response to infection in wild-type Arabidopsis accession Col-0 and the susceptible mutant *pad3*, which is defective in the camalexin biosynthesis pathway (Narusaka *et al*. 2003). Metabolomic profiling of *Arabidopsis* infected with *A. brassicicola* revealed a significant downregulation of ascorbate upon *A. brassicicola* challenge and the ascorbate mutants were more susceptible. Additionally, exogenous application of ascorbate reduced infection symptoms (Botanga *et al.* 2012).

To understand the molecular mechanisms underlying resistance versus susceptibility*, Arabidopsis* accessions exhibiting contrasting reactions to *A. brassicae* infection were used in a comparative transcriptomic study conducted in a time-dependent manner (Hamsa *et al.* 2024). Differentially expressed genes were identified as being prominently enriched in pathways related to primary metabolism, secondary metabolite biosynthesis, and the glucosinolate pathway. This indicates that both basal metabolic re-programming and specialized defence-associated metabolites contribute to resistance. Weighted Gene Co-expression Network Analysis (WGCNA) highlighted the phenylpropanoid

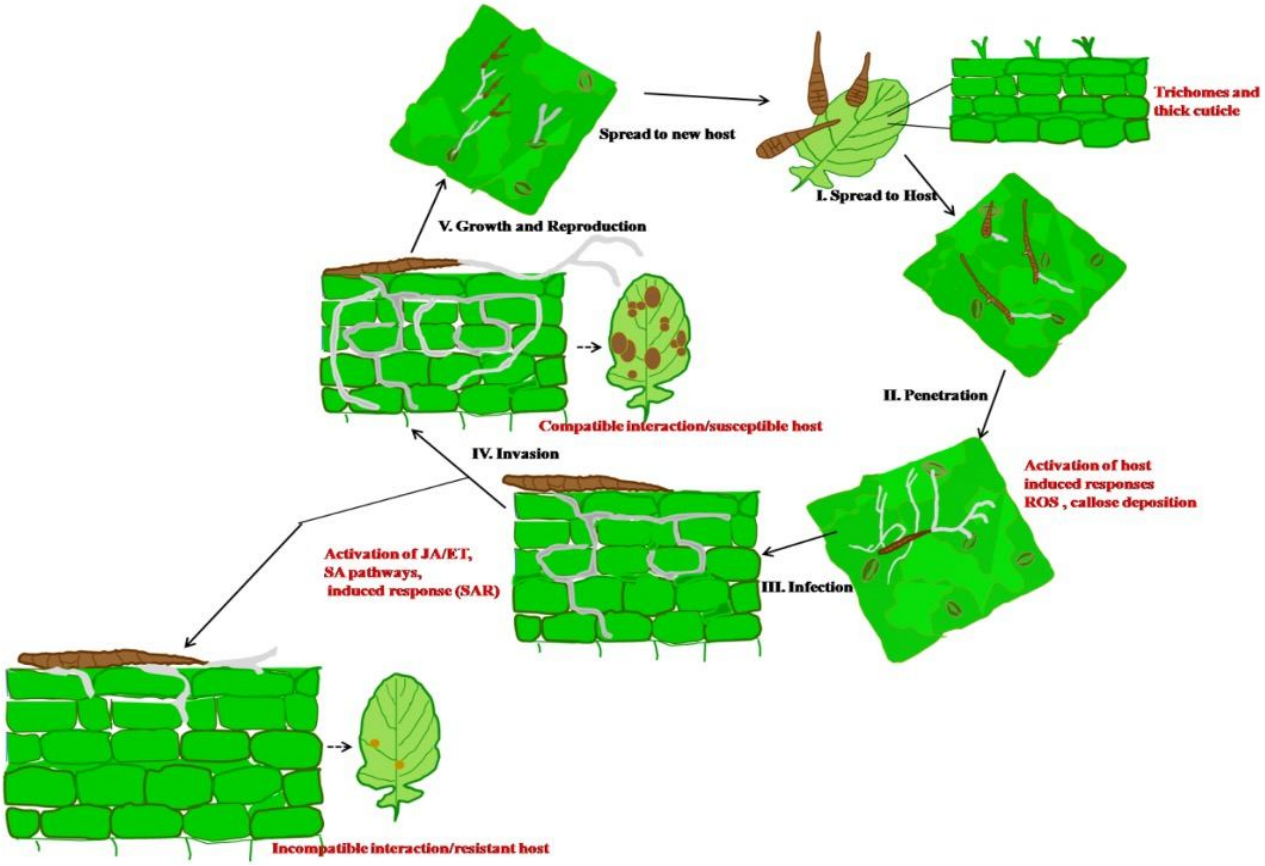

Fig. 2: Different stages of infection and host responses**.** Disease cycle includes inoculation, penetration, infection, invasion, growth, and reproduction (Sayanti Mandal PhD thesis 2015)

Fig. 3. (a) *Alternaria brassicae* isolate J3 grown on artificial growth media, (b) typical club shaped spores of *A. brassicae* with clear horizontal septe and tapering tail,(c) infection symptoms on mustard leaf under controlled conducive conditions, (d) Symptoms on susceptible Arabidopsis accession (Zdr),(e) leaves of resistant accession 7 days post infection with *A. brassicae,* (f & g) *A. brassicae* hyphae entering the host (*B. juncea*) and *A. thaliana* through the stomata (experiment conducted under controlled conditions).

pathway as a central hub in *A. brassicae* defence. Genes associated with lignin biosynthesis, flavonoid metabolism, and coumarin derivatives were co-expressed with resistance-associated modules, suggesting that the structural fortification of cell walls and accumulation of antimicrobial phenolics form an integral part of the resistance response in Arabidopsis.

Within the phenylpropanoid-derived metabolites, scopoletin has emerged as a promising candidate. Scopoletin has known antimicrobial activity and may function by limiting pathogen colonization *in planta* (Stringlis *et al*. 2019). The transcriptome evidence suggests that enhancing scopoletin biosynthesis could serve as a practical strategy for improving disease resistance in oilseed rape (*B. napus*) and other vegetable brassicas. These findings open up avenues for metabolic engineering and marker-assisted breeding to exploit scopoletin-associated defence (Hamsa *et al.* 2024).

Expanding beyond Arabidopsis, transcriptomic analysis of broccoli (*Brassica oleracea*) infected with *A. brassicicola* revealed differential expression of cuticular wax (QAX) biosynthesis genes, including 3-ketoacyl-CoA synthase and alkane hydroxylase (Gangurde *et al.* 2025). These reports highlight that cuticular traits, in addition to internal metabolic pathways, contribute to resistance against Alternaria leaf blight. Transcriptome profiling of susceptible *B. oleracea* and *B. juncea* during *A. brassicae* or *A. brassicicola* infection shows a marked suppression of photosynthesis-related genes. This is generally accompanied by visible chloroplast damage and reduced photosynthetic efficiency (ref). This downregulation may represent a host defence strategy aimed at limiting pathogen access to resources, or it could be a direct effect of fungal virulence factors targeting chloroplasts. Additionally, the role of WRKY transcription factors has also become evident, especially in the *Brassica -A. brassicicola* interaction. WRKY 33, a positive regulator of resistance against nectotrphic pathogen, contributes to *A. brassicicola* resistance through the activation of JA-regulated defense genes and indole glucosinolate pathway.

Alternaria brassicae's pathogenicity mechanisms

It is essential to understand the pathogenesis strategies employed by the fungus to develop novel counter-strategies. The pathogenic success of a typical necrotrophy depends on the production of CWDE and toxins to remove the first physical barriers, including the cuticle, waxes, and cell wall. The aggressiveness of the fungus *Alternaria* sp correlates with its ability to induce hypersensitive reaction and programmed cell death at the point of host invasion. Upon initial recognition of the compatible host, *A. brassicae* is known to secrete host-specific (HST)-Destruxin-B, and ABR. Destruxin-B, a cyclic depsipeptide, is a pathogenicity factor that acts as a cytotoxin, initiating cell death at the site of penetration and providing nutrients for the pathogen. The germinating spores also secrete ABR, a proteinaceous, host-specific toxin (roughly 27.5 kDa) (Parada *et al*. 2008). Besides toxins, the secreted cell wall-degrading enzymes (CWDE), namely cutinases, lipases, pectinases, and proteases, are upregulated *in planta* during early stages and facilitate fungal ingress within the host (Rajarammohan, 2023). *Alternaria* sp. produces a large number of secondary metabolites. Zhao *etal.* (2023) identified the presence of various new meroterpenoids (tricycloalternarenes, alternarins) in *A.brassicicola* and *A. alternata*; however, their presence and function in *A. brassicae* are still emerging areas of research. The actual functions of the majority of small, secreted secondary metabolites remain unknown; ongoing studies use genomics and functional approaches to assign roles to these compounds in disease. Continued exploration and characterisation of these unique natural products can advance crop protection strategies towards sustainable agriculture.

Transcriptome analysis of a pathogen like *A. brassicae* during host interaction provides comprehensive insights into gene expression changes that occur during infection, allowing researchers to pinpoint critical pathways, effectors, and secondary metabolite genes required for disease establishment and progression. Transcriptome analyses by Rajarammohan (2023) have revealed the

upregulation of secondary metabolite clusters and approximately 80 candidate effectors during infection, although many remain uncharacterised and functionally unexplored. At least two NLPs (AbrNLP1 and AbrNLP2) have been characterised. AbrNLP2 induces necrosis in both host and non-host plants, triggering pathogen-associated molecular pattern (PAMP)-triggered immunity, whereas AbrNLP1 does not induce necrosis but may have other roles in pathogenesis (Duhan *et al*. 2021). This combination of secreted toxins and diverse effectors underpins the aggressive necrotrophic lifestyle of *A. brassicae* in Brassica hosts.

New avenues for controlling Alternaria blight disease Targeting Susceptibility (S) Genes

Susceptibility genes contribute to the plant's vulnerability during pathogen attack. Modifying or knocking out these genes can reduce the success of pathogen infection, increasing tolerance or resistance to diseases. Targeting susceptibility genes with genome editing tools such as CRISPR/Cas9 can facilitate the precise knockouts, knockdowns, or modifications of host susceptibility genes, thereby creating durable and broad-spectrum resistance in Brassicas (Ton *et al.* 2025). In *Brassica rapa*, CRISPR/Cas9-mediated editing of the eIF(iso)4E gene was reported to confer resistance against turnip mosaic virus (TuMV), a major viral disease, demonstrating the practical power of this approach (Lee *et al*. 2023). Similarly, editing the host target (BnQCR8) of Sclerotinia effector SsSSVP1 through CRISPR/Cas9 improved resistance against *Sclerotinia* and Botrytis (Zhang *et al*. 2021). Identification of susceptibility genes for *A. brassicae* through transcriptomics, proteomics or genetics can open new avenues for improving resistance using genome editing.

Exploring Nonhost resistance against Alternaria infections

In nature, a whole plant species can be naturally protected against all forms or variants of a particular pathogen and this kind of resistance is called Non-host resistance (NHR). This form of resistance is generally very robust and durable, making it a valuable approach for improving resistance against notorious pathogens. Despite several attempts, the molecular basis underlying NHR is poorly comprehended. Fatima *et al.* (2019) attempted to explore the molecular basis of NHR in Chickpea against *A.brassicae* with the aim of identifying NHR-relatedgenesfor transfer to develop durable resistance against *A. brassicae.*

Exploring the RNAi technology

To minimise dependency on chemical pesticides to control diseases, farmers are encouraged to integrate multiple strategies, such as using resistant varieties, biological control, and crop rotation. However, over-reliance on agrochemicals has led to the evolution of pathogens with pesticide resistance, including the emergence of new biotypes and the expansion of their host range in a changing climate scenario. This scenario underscores the urgent need for innovative and sustainable approaches to curtail the damage caused by pathogens.

RNA interference-based crop protection has emerged as a promising tool, effective against a wide range of pathogens. The strategy has been deployed either as HIGS, where transgenic plants express dsRNA targeting fungal genes, or SIGS, where *in vitro*/ artificially synthesised dsRNA against fungal genes is applied exogenously to restrict pathogen progression. Since the latter does not require the development of transgenics, it poses fewer regulatory concerns; therefore, it has been explored as an effective solution for managing diseases under real/ field conditions. The success of the RNAi-based approach depends on several factors, including the selection of target genes and optimising the effectiveness of the dsRNA selected, as well as the stability of the dsRNA and its targeting/ delivery.

The availability of genomic data for *A. brassicae*, *A. brassicicola, and A.alternata* (Dang *et al.* 2015; Rajarammohan *et. al*. 2019), along with an in-depth understanding of the molecular, biochemical and physiological players involved in Alternaria pathogenicity and growth (Rajarammohan, 2023) provides a rich resource for identifying candidate target genes. Potential targets include genes involved in Cell wall

degradation (e.g., cutinases, polygalacturonases), Toxin biosynthesis pathways, signal transduction and effector secretion, and essential housekeeping functions required for survival and colonisation. Simultaneously targeting multiple essential genes through RNAi can be explored as an alternate strategy to control Alternaria blight. With further advances in delivery methods, formulation stability, and multiplex gene targeting, topical application or SIGS (Spray Induced Gene Silencing), in particular,holds promise as a field-deployable, non-GMO strategy for managing diseases, including Alternaria blight in Brassica crops. RNAi-based crop protection could become a cornerstone of next-generation integrated disease management.

DECLARATION

Conflict of Interest. Author declares no conflict of interest.

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