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GENOMIC INSIGHTS INTO BLAST RESISTANCE AND PYRICULARIA GRISEA PATHOGENESIS IN FINGER MILLET: ADVANCES, GAPS, AND BREEDING PERSPECTIVES

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ABSTRACT
Accelerating climate change in conjunction with rapid population growth is exerting substantial pressure on global food security systems, with elevated temperatures anticipated to adversely affect the productivity of major staple cereals including rice, wheat, and maize. These emerging challenges underscore the necessity for the integration of climate-resilient crops into contemporary agricultural frameworks. Finger millet, a nutrient-dense cereal traditionally recognized as "Shree Anna", implies unsurpassed among all nutriment grains along with other millets, has significant potential to enhance sustainable food production, strengthen agroecosystem resilience, and promote dietary diversification. This study was conducted through a comprehensive analysis of scientific literature and datasets obtained from authentic platforms. Studies focusing on finger millet, blast disease, host–pathogen interactions, molecular resistance, and breeding strategies were systematically examined and critically evaluated. The study compiles available genetic and molecular information related to cereals like rice, barley, finger millet and blast fungus Pyricularia grisea. It highlights key findings on host–pathogen interactions and identifies important knowledge gaps that currently limit the efficiency of breeding programs aimed at improving resistance. Although finger millet is widely valued for its nutritional richness and adaptability to challenging environmental conditions, scientific exploration of this crop has progressed relatively slowly, especially in the area of fungal disease resistance. In particular, limited information is available regarding defense mechanisms against blast disease incited by Pyricularia grisea. Recent innovations in genomic analysis and molecular breeding methodologies have significantly expanded the understanding of trait architecture and genetic variability in finger millet. These advances are expected to accelerate the development of superior cultivars possessing enhanced resilience against both environmental stresses and pathogen-associated challenges.

KEYWORDS: Pyricularia grisea, Eleusine coracana, climate resilience, crop improvement, food security

INTRODUCTION

Climatic irregularities, increasing temperatures, and limited irrigation availability are increasingly threatening agricultural productivity and crop yields. These challenges, intensified by climate change and rapid population growth, demand higher food production from progressively shrinking arable land resources [1]. Climate projections suggest that tropical regions are likely to experience the most severe consequences of rising global temperatures, with developing nations facing particularly significant reductions in agricultural productivity and food production capacity [2].

Climatic alterations are anticipated to exert substantial impacts on agricultural production systems and global food security, with drought-affected and water-deficient regions exhibiting heightened vulnerability to these environmental stresses [3]. Water scarcity drives reductions in dietary diversity and aggregate food consumption, culminating in heightened risks of malnutrition and systemic food insecurity. These effects manifest through constrained access to nutrient-rich foods and compromised nutritional quality, particularly in vulnerable agroecosystems. Empirical observations underscore the imperative for integrated water management strategies to safeguard dietary adequacy and public health outcomes [4]. A critical global priority involves strengthening the resilience of agricultural systems to ensure the provision of sufficient, nutrient-dense, and sustainably produced food for an estimated world population approaching 9 billion by 2050. Projections from FAO indicates that global food production must increase by approximately 60–70% to satisfy future demand under conditions of constrained arable land availability, limited freshwater resources, and increasing climatic instability. Inadequate attainment of these production targets may aggravate food insecurity, malnutrition, socioeconomic vulnerability, and environmental degradation, thereby emphasizing the urgent necessity for transformative advancements in crop productivity, climate adaptation, and resource-use efficiency [5].

Climate change is expected to alter the distribution, population behavior, and virulence of agricultural pests and plant pathogens, thereby increasing risks to crop productivity. Rising temperatures and irregular precipitation patterns may facilitate the spread of invasive species and extend pathogen survival, necessitating the advancement of integrated pest management strategies [6]. Other than that adequate caloric intake alone does not ensure complete nutritional sufficiency, as deficiencies in essential micronutrients such as vitamins, minerals, and trace elements may still persist. These

deficiencies can adversely affect physiological functions and health status despite sufficient energy consumption, emphasizing the need for micronutrient-focused dietary assessment [1].

Conventional cereal production has prioritized wheat, rice, and maize, sidelining resilient millets despite their nutritional and climatic advantages. Indigenous and underutilized crops, notably millets, present viable alternatives that address multifaceted nutritional and environmental demands [7]. Millets comprise a diverse assemblage of small-seeded grasses cultivated for human consumption, animal fodder, and forage production [8-9].

Millets encompass six principal small-grained cereal species, including finger millet (Eleusine coracana), foxtail millet (Setaria italica), kodo millet (Paspalum scrobiculatum), proso millet (Panicum miliaceum), barnyard millet (Echinochloa spp.), and little millet (Panicum sumatrense). These cereal crops are characterized by distinct agronomic advantages, particularly their resilience to diverse abiotic stress conditions, as well as their substantial nutritional richness in essential macronutrients and micronutrients, thereby enhancing their significance in sustainable and nutrition-sensitive agricultural systems [10-11].

Staple cereals such as rice and wheat, despite their pivotal role in global food security, necessitate considerable production inputs and associated costs. In juxtaposition, millets confer a spectrum of agronomic and nutritional benefits—encompassing drought tolerance, short cropping cycles, and minimal resource demands—at markedly lower input levels. This cost-efficiency positions millets as a strategic complement to conventional staples in sustainable cropping systems [1]. [12] states that finger millet (Eleusine coracana), colloquially termed ragi or mandua, sustains broad cultivation throughout multiple agroecological zones in India and internationally. India predominates as the principal producer, contributing nearly 60% of global output, thereby underscoring its strategic significance in resilient cereal systems [13]. Finger millet is conventionally utilized in whole-grain form without requiring outer husk removal, enhancing its nutritional retention and processing efficiency. The crop exhibits substantial adaptability to regions receiving annual rainfall between 600 and 1,200 mm, particularly under acidic soil conditions, and typically attains physiological maturity within 100–130 days. A notable agronomic attribute of finger millet is its pronounced resilience across diverse agroclimatic environments, which contributes significantly to its superior productivity relative to other millet species [14]. Its grains have excellent nutraceutical properties, such as high dietary fiber content, amino acids (methionine, phenylalanine, tryptophan, cysteine, isoleucine, and leucine), vitamin B complex, calcium, and iron compared with maize, rice, wheat, and sorghum [1,11]. Finger millet contains methionine—an essential amino acid commonly deficient in starchy diets- at levels approximately twice as high as those found in maize and rice [15]. In India, it is widely grown in the states of Karnataka, Tamil Nadu, Andhra Pradesh and parts of North India [16]. The multifaceted advantages associated with millet cultivation, coupled with comparatively low input and production costs, render these crops integral to the reinforcement of agricultural resilience and long-term sustainability. Their adaptive capacity and resource-use efficiency support stable production systems under variable environmental and socioeconomic conditions [1]. However, blast disease is a widespread and highly destructive disease affecting these cereal crops. The pathogen responsible for the disease was identified as Pyricularia grisea [17]. Magnaporthe grisea Barr (anamorph: P. grisea (Cooke) Sacc.) is a filamentous ascomycete fungus capable of infecting more than 50 host species [18]. P. grisea is a hemibiotrophic plant pathogen that infect a wide range of grass species, including several economically important crops like rice, wheat, finger millet etc. Fungal infection of panicles in crops such as rice and finger millet, or spikes in wheat, can result in total crop failure [19]. Consequently, P. grisea is widely regarded as a key model organism for studying plant-pathogenic fungi [20-21].

MATERIALS AND METHODS

This paper is based on study of various literature related to the blast fungus, its associated genes, mechanisms of action, and management strategies. The collected information was organized and the findings were summarized and presented in the form of text and tables, along with a concluding section. Additionally, data related to other crops were also examined to explore potential approaches for implementing effective genetic improvements in plants.

TAXONOMY, HOST RANGE, AND POPULATION DYNAMICS OF P. grisea

Table 1. Taxonomy

Kingdom	Fungi
Phylum	Ascomycota
Sub-phylum	Pezizomycotina
Class	Sordariomycetes
Order	Magnaporthales
Family	Pyriculariaceae (anamorph) / Magnaporthaceae (telemorph)
Genus	Pyricularia (anamorph) / Magnaporthe (telemorph)
Species	P. grisea (anamorph) / M. grisea (telemorph)

Host range

P. grisea possesses a broad host range encompassing multiple members of the Poaceae family and is differentiated into distinct host-adapted lineages that exhibit specificity toward particular cereal genera. Table 1 represents taxonomy, host range and population dynamics of P. grisea. The pathogen is regarded as one of the most destructive fungal agents affecting

economically important crops such as rice, wheat, maize, barley, sorghum, and finger millet, where it causes significant yield losses and substantial agricultural damage across diverse agroecological regions.

Disease symptoms

The pathogen is capable of colonizing multiple aerial tissues, including leaves, stems, sheaths, and panicles, across all growth stages of the host plant. Disease onset is characterized by the appearance of small pale or grayish-green lesions surrounded by darker margins. With disease progression, these lesions enlarge into characteristic elliptical or spindle-shaped necrotic spots possessing ash-gray centers and pronounced necrotic boundaries. Under severe infection conditions, lesion coalescence may occur, resulting in extensive tissue necrosis and eventual destruction of the affected leaf area.

The differentiation of P. oryzae from P.grisea has been supported by comprehensive phylogenetic investigations and molecular characterization studies, which demonstrate clear genetic divergence between the two fungal species [22-23]. Therefore, P. oryzae isolates were generally referred to as P. grisea or M. grisea in older literature.

Pathogen isolates of P. grisea from a given crop are typically pathogenic to plant species belonging to the same genus as the original host. For example, isolates collected from finger millet (E. coracana) can infect other species within the genus Eleusine, including E. indica and E. africana, as well as E. coracana itself. [24] designated those host genus-specific groups as pathotypes. Several pathotypes have been recognised in P.grisea e.g., the Eleusine pathotype pathogenic on finger millet, Oryza pathotype pathogenic on rice, Setaria pathotype pathogenic on foxtail millet, Triticum pathotype pathogenic on wheat [25] and Lolium pathotype pathogenic on ryegrass [26-27].

MOLECULAR MECHANISM UNDERLYING THE LIFE CYCLE OF P. grisea

Numerous investigative methodologies have been established to examine fungal developmental processes and to characterize the molecular events governing host invasion by P. grisea. Current understanding of these infection-associated mechanisms has largely been derived from studies involving the rice–P. grisea pathosystem (Figure 1). However, the conserved nature of several pathogenicity-related processes suggests that these findings may also be broadly relevant to interactions between P. grisea and other susceptible host species [28].

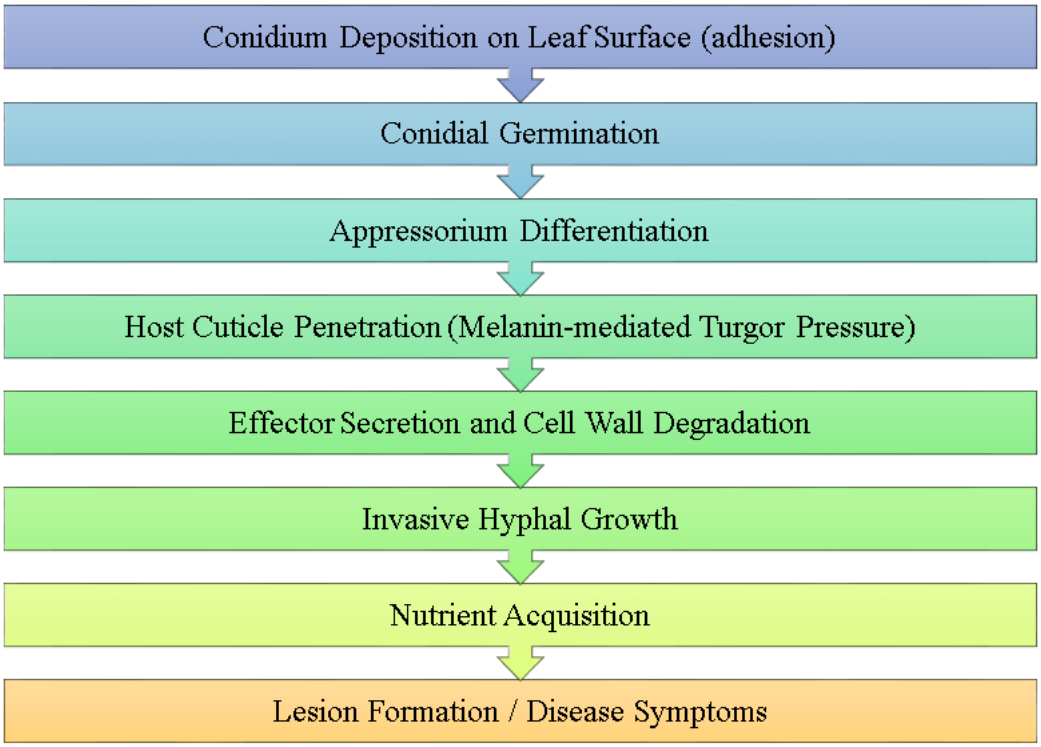

Figure 1. The flow chart of P. grisea Infection Process

1. Adhesion

P. grisea is capable of infecting diverse aerial organs of rice, including leaves, stems, sheaths, and panicles, across all developmental stages of the host plant. The infection cycle is initiated by deposition of an asexual three-celled conidium onto the hydrophobic surface of the rice leaf. Successful establishment of infection requires firm adhesion of the conidium, a process mediated by the secretion of an extracellular adhesive matrix commonly referred to as spore tip mucilage, which enables stable attachment to the host cuticle prior to fungal penetration [29-30]. Recent investigations have demonstrated that the spermine synthase-encoding gene SPS1 plays a crucial role in mediating appressorial adhesion, indicating its functional importance during the early stages of fungal host attachment and infection establishment [30]. Although spermine is unlikely to function as a component of the extracellular adhesive matrix, it contributes significantly to the detoxification of reactive oxygen species within the endoplasmic reticulum. Furthermore,

mutations in several genes, including the O-mannosyltransferase Pmt2, the transcription factor Tra1p, the fasciclin-like protein Flp1, and the ubiquitin ligases Rad6 and Bre1, have been shown to disrupt various developmental and pathogenicity-related processes, particularly impairing conidial adhesion and fungal infection efficiency [31-35,28].

2. Germination of conidium and appressorium formation

Upon attachment of the three-celled conidium to the leaf surface and exposure to water, a germ tube emerges from the apical cell. Within 4–6 hours, the germ tube stops elongating and differentiates into an appressorium [29,36]. This melanized, dome-shaped infection structure enables the fungus to breach the leaf cuticle and penetrate the underlying epidermal cells. Appressorium development is orchestrated by complex signalling networks. Two pathways have been particularly well characterized: one involving cyclic adenosine monophosphate (cAMP) and the other the mitogen-activated protein (MAP) kinase Pmk1 [35,37-40]. The cAMP pathway is initiated by the receptor Pth11, which perceives surface hydrophobicity and controls the initiation of appressorium development following recognition of the hydrophobic leaf cuticle by germinating hyphae [41]. The signals triggering the MAP kinase cascade are not fully understood but are downstream of the cAMP signalling pathway. Other than that, MPG1 expression is strongly induced during host infection, particularly during appressorium formation and disease development. Loss of MPG1 function reduces fungal virulence and impairs infection establishment. The gene encodes a hydrophobin-like cysteine-rich secreted protein involved in maintaining fungal surface hydrophobicity, with mutant strains displaying increased surface wettability. These findings indicate that MPG1-associated hydrophobins are important for infection structure development and fungal pathogenicity [42]. Also, analyses of the pmk1 deletion mutant revealed that Pmk1 is required for the control of appressorium development and cell-to-cell movement during invasive growth [43-45].

In P. grisea, chitin deacetylation by chitin deacetylases generates chitosan. Chitosan is a polymer of β1,4-glucosamine and it accumulates within germ tubes and appressoria during infection-related development. Functional disruption of CDA genes reduced chitosan formation, leading to impaired germling adhesion and defective appressorium differentiation on hydrophobic artificial surfaces. However, supplementation with exogenous chitosan restored these developmental processes. Despite these defects, mutant strains retained pathogenicity on host plants, indicating that plant cuticular waxes can compensate for the requirement of chitosan during infection. These findings demonstrate that chitosan primarily facilitates surface adhesion and perception of physical cues necessary for appressorium development rather than functioning as a stealth factor in fungal pathogenicity [46].

Cell cycle regulation also plays a prominent role in controlling appressorium development and maturation [47-48]. Autophagy is tightly correlated with the cell cycle progression and is another checkpoint for appressorium maturation [28,48-49]. Autophagic activity is controlled by a highly conserved network of more than thirty autophagy-associated genes (ATGs), which coordinate the sequential events of the pathway. In M. oryzae, several ATG proteins, including ATG1, ATG2, ATG3, ATG17, and ATG18, exhibit enhanced phosphorylation during the development of the appressorium, while phosphorylation of ATG13 is reduced at a specific site. These phosphorylation dynamics suggest that regulation of ATG proteins through post-translational modifications is closely linked to host penetration and pathogenic differentiation. In addition, autophagy can be activated to facilitate the transport and recycling of host-derived nutrients and may additionally function as an alternative pathway for secretion [50].

A putative ortholog of the yeast SNT2 gene was identified and functionally characterized in M. oryzae and designated as MoSNT2 [52]. The fungal genome contains only a single copy of this gene. It has been known already that disruption of genes involved in autophagic cell death impairs plant infection in M. oryzae [51]. Functional analyses demonstrated that MoSNT2 is essential for maintaining autophagic balance, and its expression is positively regulated by the MoTor signaling pathway. MoSnt2 localizes to the nucleus, where its PHD1 domain recognizes acetylated histone H3, while its ELM2 domain interacts with the histone deacetylase MoHos2 to recruit HDAC complexes to specific chromatin regions. This recruitment reduces histone H3 acetylation levels and consequently modulates the expression of numerous genes associated with developmental regulation, including those involved in autophagy [52].

3. Appresorium maturation and penetration by P. grisea

Maturation of the appressorium is a critical process required for strengthening its structural framework and generating the elevated turgor pressure necessary for successful host tissue penetration [39]. The initial stage of this maturation process is characterized by melanin deposition within the appressorial cell wall, which facilitates intracellular glycerol accumulation and osmotic water uptake. These events collectively contribute to the development of substantial internal turgor pressure, reaching approximately 8.0 MPa (80 bars), thereby enabling mechanical penetration of the host surface [53-55]. The second phase involves the rearrangement of F-actin by septin proteins [56-59]. Multiple cytoskeletal and membrane-trafficking proteins, including septins (Sep3, Sep4, Sep5, and Sep6), exocyst complex subunits (Sec3, Sec5, Sec6, Sec8, Sec15, Exo70, and Exo84), together with Rvs167 and Tea1, spatially associate with the F-actin network at the basal region of the appressorium. This coordinated localization is essential for cytoskeletal organization and membrane remodeling processes that drive differentiation of the penetration peg during host invasion [56,58]. This process is tightly regulated by the turgor- sensing kinase Sln1 ensuring that the right pressure is reached in the appressorium [28,59-60]. Sln1 is a histidine–aspartate kinase that act during appressorium-mediated host invasion. Localization of Sln1 at the appressorium pore occurs in a pressure-dependent manner, facilitating recognition of the turgor threshold necessary for successful plant penetration. Deletion of Sln1 results in excessive intracellular turgor, abnormal melanization, and impaired organization of septins and polarity-associated proteins. Sln1 functions alongside the protein kinase C-mediated cell integrity pathway to regulate cAMP-dependent signaling through protein kinase A. In this regulatory network, Pkc1 targets the NADPH oxidase regulator NoxR through phosphorylation, and together

these interconnected signaling pathways control appressorial turgor generation [61].
Expression of cutinase gene CUT2 is also strongly induced during appressorium maturation and host penetration in blast fungus. The cut2 mutant shows impaired conidiation, and abnormal germ tube and appressorium development, ultimately leading to diminished pathogenicity in rice and barley. Although CUT2 does not influence adhesion or appressorial turgor generation, it is essential for penetration peg differentiation and effective host invasion [62].

4. Biotrophic stage of infection

During the initial stage of appressorium-mediated penetration, a narrow penetration peg forms and breaches the host surface and enters the very first host plant cell, where it differentiates into a thin primary invasive hypha [63-65]. Primary hyphae turns into secondary hyphae. These are infection hyphae which invade the cytoplasm of the first infected plant cell. Infection hyphae are surrounded by an extra- invasive hyphal matrix (EIHMx) [64]. Hundreds of proteins are secreted in the EIHMx by the fungus, including apoplastic effectors, plant polymer-degrading enzymes and proteins protecting the fungal cell wall [43,66,67-68]. Apoplastic effectors, such as the Biotrophy-associated secreted proteins 4 and 113 (Bas4, Bas113), the Secreted LysM protein 1 (Slp1) and the Magnaporthe effector protein 1 (Mep1) are secreted by a conventional Golgi- dependent secretory pathway and accumulate in the EIHM around the infection hyphae [43,67,69]. The secretion of some apoplastic effectors, such as Slp1, requires the coat protein complex II (COPII) cargo receptor ER-derived vesicle protein MoErv29 and involves the recognition of amino-terminal tripeptide motifs [70]. Slp1 accumulates at the interface between the infection hyphae and the host cell wall, where it binds to chitin preventing recognition of these fungal pathogen-associated molecular patterns (PAMPs) by the rice chitin elicitor binding protein OsCEBiP [67,71]. Other effectors are translocated into the plant cells (cytoplasmic effectors) through an extracellular structure called the biotrophic interfacial complex (BIC), which is localized at the tip of the primary invasive hypha and composed of plant-derived membrane vesicles [65,68-69]. Cytoplasmic effectors including AVR- Pik [43], PWL2, Rbf1 [72], MoHTR3 [73] and Bas1 [65] accumulate at high levels in the BIC. A role for the Bas83 effector in recruiting plant membrane fragments for endocytosis at the BIC has been suggested [68]. A few cytoplasmic effectors, like Bas170, also localize, before BIC formation, to punctate membranous compartments in the rice cytoplasm underneath appressoria and in surrounding rice nuclei. These observations suggest that effector uptake could take place either before or at early stages of host penetration [68]. The effectors Avr-Pita, AvrPiz-t and Avr-Pii play a role in ROS burst inhibition [74-79]. The effectors HTR1, HTR2, HTR3 and lug4 target host transcriptional reprogramming [73,75,80]. The effectors HTR3, lug4, lug6 and lug9 modulate plant hormonal pathway [73,81-82]. AvrPiz- t has also been shown to interfere with host protein degradation [77-78]. Finally, Avr-Pii targets vesicle trafficking [83]. A subset of cytoplasmic biotrophy-associated effectors is recognized, either directly or indirectly, by intracellular immune receptors and these are therefore named avirulence proteins (AVRs). They include AvrPiz-t [74], AvrPib [75], AVR-Pii [86], AVR-Pia [86-87], AVR1-CO39 [88-89], AVR-Pi9 [90] and AVR-Pik [86], which are respectively recognized in rice by Piz-t [91], Pib [92], Pii [86], Pi-a [93-94], Pi-CO39 [93,95], Pi9 [96] and Pi-k [97-98]. Because of their critical role in the virulence of the pathogen, the precise regulation of effector expression is key and involves presumably numerous complex regulatory networks that remain, however, largely unexplored. Relatively few transcription factors regulating the infection programme of the blast fungus and, specifically, effector expression have been identified. Examples are the bZIP transcription factors BIP1 and MoEITF2, and the zinc finger transcription factor MoEITF1 that control distinct infection-related gene networks [99-100], as well as the WOPR box transcription factor MoWOR1 (syn. MoGTI1) that acts in P. grisea like in numerous other pathogenic fungi as a central regulator of pathogenicity [28, 101-102].
A particularly important virulence function of P. grisea is coping with the oxidative burst that the host plant produces upon pathogen detection. This oxidative burst is mostly composed of ROS and reactive nitrogen species (RNS) and has a dual function in immune signalling and antimicrobial defence. The fungus interferes with the oxidative burst by the effector- mediated suppression of host immunity and ROS production and by various fungal oxidative stress responses that are critical for attenuating the ROS burst or limiting its toxicity for the fungus. Critical elements of these oxidative stress responses that are necessary for successful infection are, for instance, superoxide degradation by the superoxide dismutase Sod1 [103], nitro-oxidative damage responses involving the nitronate monooxygenases Nmo2 [49], and ROS scavenging by glutathione involving the glutathione peroxidase Hyr1 [104] and the glutathione reductase Gtr1 [105]. Another important mechanism is the preservation of glucose for the synthesis of the antioxidant NADPH through the pentose phosphate pathway (PPP), and the use of other compounds such as glutamate as a primary carbon source [106].
In the late stage of the invasion of the first infected cell, the EIHM integrity is lost, leading to the spill of the EIHMx content into the cytoplasm of the host cell. This is accompanied by shrinking and rupture of the host cell vacuole, marking the death of the first invaded cell. The invasive hypha continues to grow in the dead cell, becoming more filamentous and getting closer to the cell wall [28,66,107-108].
The subsequent movement of the fungus from cell to cell occurs at pit fields, characterized by clusters of plasmodesmata. It involves the formation of a specialized hyphal structure called the transpressorium that develops from the more filamentous invasive hyphae by swelling and subsequent constriction when they pass the cell wall at the pit field. Such morphological transition and cell junction crossing requires fungal septin and actin reorganization regulated by the Pmk1 MAP kinase pathway [43-44,109]. The biotrophic invasion of plant cells lasts up to 4 days after the start of the infection. Subsequently, P. grisea undergoes a switch in development and adopts a necrotrophic development [28].

5. Necrotrophic stage of Infection

The molecular and developmental mechanisms governing the transition of blast fungi from biotrophic to necrotrophic growth phases remain inadequately characterized. This developmental shift is generally accompanied by the appearance

of conspicuous disease symptoms approximately five days following host infection, reflecting significant changes in fungal pathogenic behavior. The necrotrophic stage is associated with elevated expression of genes encoding cell wall-degrading enzymes, including hydrolases, glucosidases, and glycosyl hydrolases, as well as necrosis-inducing factors such as NLP1, which collectively contribute to host tissue degradation and disease progression [108,110]. Recent findings suggest that suppression of MIF1 expression, a factor involved in inhibiting host cell death, is closely associated with the developmental transition from the biotrophic to the necrotrophic phase of fungal infection. This regulatory shift may contribute to enhanced host tissue necrosis and facilitate disease progression during later stages of pathogen colonization [111]. Despite advances in the study of fungal pathogenicity, comprehensive functional characterization of genes associated with the necrotrophic phase in P. grisea remains insufficient. Consequently, the necrotrophic stage continues to represent one of the most poorly understood phases within the pathogen's life cycle and infection strategy [28].

Table 2. Collective description of genes, proteins, transcription factors of P. grisea with their function.

Genes/ Factors	Type	Function/ Role	Infection Stage	References
SPS1	Spermine synthase gene	Required for appressorial adhesion, involved in ROS scavenging in ER via spermine metabolism	Adhesion	[30]
Pmt2	O-mannosyltransferase	Protein glycosylation, mutation causes defects in conidial adhesion	Adhesion	[32]
Tra1p	Transcription factor	Regulates gene expression, mutation results in pleiotropic defects including adhesion	Adhesion	[31]
Flp1	Fasciclin like protein	Cell surface adhesion protein contributing to conidial adhesion	Adhesion	[33]
Rad6	Ubiquitin ligase	Protein ubiquitinylation, mutation causes adhesion defects and other pleiotropic phenotypes	Adhesion	[34]
Bre1	Ubiquitin ligase	Histone ubiquitination and mutation affects conidial adhesion	Adhesion	[35]
Pth11	GPCR-like receptor	Senses hydrophobic surface of leaf and triggers cAMP signalling for appressorium development	Germination / Appressorium development	[41]
MPG1	Gene encoding hydrophobin-like cysteine-rich protein	Important for fungal pathogenicity	Appressorium development	[42]
cAMP pathway	Signalling pathway	Controls initiation of appressorium development	Germination	[37]
Pmk1	MAP kinase	Essential for appressorium development, penetration and cell-to-cell invasive growth	Germination & Invasion	[45]
DA (Chitin deacetylase)	Chitosan generating enzyme	Disruption leads to defective appressoria	Germination and Invasion	[46]
ATG (1,2,3,13,17,18)	Autophagy related genes	Phosphorylated during host penetration	Appressorial penetration	[50]
MoSNT2	Gene	Essential for proper autophagy	Appressorium maturation	[52]
Sep3, Sep4, Sep5, Sep6	Septin proteins	Organize F-actin cytoskeleton at appressorium base for penetration peg formation	Appressorium maturation	[56]
Sec3, Sec5, Sec6, Sec8, Sec15, Exo70, Exo84	Exocyst complex proteins	Mediate polarity during penetration peg formation	Appressorium maturation	[58]

Rvs167	Inverse-bin-amphiphysin-RVS-domain protein	Co-localizes with actin network during penetration peg differentiation	Appressorium maturation	[56]
Tea1	Ezrin-radixin-moesin protein	Coordinates actin organization at appressorium base required for penetration	Appressorium maturation	[56]
Sln1	Turgor-sensing kinase	Monitors turgor pressure in appressorium before penetration	Appressorium maturation	[59-60]
CUT2	Cutinase gene	Mutation in it leads to impaired conidiation and abnormal germ tube formation	Appressorium maturation and host penetration	[62]
Bas4	Apoplastic effector	Secreted into EIHM; functions in biotrophic infection interface	Biotrophic stage	[43,69]
Bas113	Apoplastic effector	Secreted protein accumulating in EIHM during infection	Biotrophic stage	[69]
Slp1 (Secreted LysM protein 1)	Apoplastic effector	binds to chitin preventing recognition of (PAMPs) by the rice chitin elicitor binding protein OsCEBiP	Biotrophic stage	[67, 69]
Mep1 (Magnaporthe effector protein 1)	Effector protein	Secreted into EIHM contributing to virulence	Biotrophic stage	[43, 67, 69, 108]
MoErv29	COPII cargo receptor	Required for secretion of certain effectors (e.g., Slp1)	Effector secretion	[70]
AVR-Pik	Cytoplasmic effector	Accumulates at high levels in the BIC	Biotrophic stage	[43]
PWL2	Cytoplasmic effector	Accumulates in biotrophic interfacial complex (BIC) for host targeting	Biotrophic stage	[72]
Rbf1	Cytoplasmic effector	Required for biotrophic interfacial complex formation and invasive growth	Biotrophic stage	[72]
MoHTR3	Cytoplasmic effector	Accumulates at high levels in BIC, help in invasion	Biotrophic stage	[73]
Bas1	Cytoplasmic effector	Accumulates at BIC and enters plant cells	Biotrophic stage	[65]
Bas83	Effector	May recruit plant membrane fragments for endocytosis at BIC	Biotrophic stage	[68]
Bas170	Effector	Localizes in rice cytoplasm and nuclei before BIC formation to punctate membranous compartment	Biotrophic stage	[68]
Avr-Pita AvrPiz-t Avr-Pii	Effector and avirulence Proteins	Suppresses ROS burst in host; AvrPiz-t, AVR-Pii is also recognised in rice by Piz-t, Pii respectively.	Host immune suppression	[74-79]
HTR1 HTR2	Effectors	Manipulate host transcriptional regulation	Biotrophic stage	[80]
HTR3 lug4	Effector	Regulates host transcription and hormonal pathways	Biotrophic stage	[73, 81]
lug6 lug9	Effectors	Modulate plant hormone signalling pathways	Biotrophic stage	[82]
AvrPib	AVR effector	Recognized by Pib resistance gene	Host-pathogen recognition	[85]

AVR-Pia	AVR effector	Recognized by Pi-a resistance gene	Host immunity	[86-87]
AVR1-CO39	AVR effector	Recognized by Pi-CO39 resistance gene	Host immunity	[88-89]
AVR-Pi9	AVR effector	Recognized by Pi9 resistance gene	Host immunity	[90]
BIP1	bZIP transcription factor	Regulates effector expression	Infection program	[100]
MoEITF2	bZIP transcription factor	Controls infection gene networks	Infection regulation	[99]
MoEITF1	Zinc finger transcription factor	Regulates effector expression and infection genes	Infection regulation	[99]
MoWOR1 (MoGTI1)	WOPR box transcription factor	Central regulator of fungal pathogenicity	Infection regulation	[101-102]
Sod1	Superoxide dismutase	Detoxifies superoxide radicals during host ROS burst	Oxidative stress response	[103]
Nmo2	Nitronate monooxygenase	Protects fungus from nitro-oxidative damage	Oxidative stress response	[49]
Hyr1	Glutathione peroxidase	Does ROS scavenging by glutathione	Oxidative stress response	[104]
Gtr1	Glutathione reductase	Maintains glutathione redox balance for ROS detoxification	Oxidative stress response	[105]
NLP1	Necrosis-inducing protein	Promotes host cell death during necrotrophic stage	Necrotrophy	[108, 110]
MIF1	Effector-like regulator	Inhibits plant cell death; its downregulation triggers necrotrophic switch	Biotrophy to Necrotrophy transition	[111]
Hydrolases, Glucosidases, Glycosyl hydrolases	Cell-wall degrading enzymes	Degradation of host cell wall	Necrotrophy	[108, 110]

Host Plants of P. grisea

Plants and plant pathogens share an antagonistic relationship and have been engaged in a continuous evolutionary struggle for survival since ancient times. Both have developed diverse strategies to outcompete each other. Table 2 represents collective description of genes, proteins, transcription factors of P. grisea with their function.

1.1 Genes in rice (Oryza Sativa) against blast pathogen

Rice blast disease is characterized by the formation of lesions across all aerial tissues of the rice plant, with lesion morphology and pigmentation varying according to cultivar susceptibility and prevailing environmental conditions. In highly susceptible cultivars exposed to elevated humidity levels, expansive grayish lesions measuring nearly 1 cm in length commonly develop on leaf surfaces. Conversely, resistant cultivars generally exhibit comparatively smaller lesions delineated by distinct dark brown margins, reflecting restricted pathogen progression and enhanced host defense responses [28,112].

1. OsRING113- The rice really interesting new gene (RING)-type E3 ubiquitin ligase OsRING113 plays a pivotal role in plant defense regulation by mediating the 26S proteosomal degradation of APIP5 which is a negative regulator associated with immune signalling and programmed cell death pathways. The osring113 mutants in rice exhibited decreased BSR (Broad-spectrum disease resistance), whereas the elevated expression of OsRING113 showed significantly enhanced BSR against fungal pathogen M. oryzae and the bacterial pathogen Xanthomonas oyzae pv. oryzae [113].

2. DEP2 / OsRING80- The DENSE AND ERECT PANICLE 2 (DEP2) gene mutation in rice induces distinct morphological modifications, including erect panicles, reduced plant stature, altered grain morphology, and changes in leaf orientation. In addition to these developmental effects, impairment of DEP2 function confers enhanced resistance against the blast pathogen M. oryzae. Molecular analyses demonstrated that DEP2 associates with the E3 ubiquitin ligase OsRING80, which facilitates DEP2 ubiquitination and subsequent degradation through the 26S proteasome system. The observed reduction in OsRING80 transcript accumulation within dep2 mutant backgrounds indicates the existence of a potential feedback regulatory mechanism. Furthermore, targeted disruption of OsRING80 markedly improved blast resistance while maintaining normal plant growth and development, highlighting its potential utility in disease-resilient

rice breeding programs [114].

3. OsSEC3A- OsSEC3A functions as a crucial constituent of the rice exocyst complex and demonstrates widespread expression across diverse plant tissues. Protein interaction studies suggest its involvement in exocyst assembly through association with multiple exocyst subunits. Functional impairment of OsSEC3A leads to pronounced developmental abnormalities, including reduced plant height and lesion-mimic manifestations. Moreover, mutant lines display enhanced immune activation characterized by elevated transcription of defense-associated and salicylic acid biosynthetic genes, indicating a regulatory role of OsSEC3A in coordinating plant growth and immune signaling pathways [115].

4. OsPIE3 / PID2- The rice resistance gene PID2 plays a central role in defense against the blast pathogen M. oryzae. The U-box E3 ubiquitin ligase OsPIE3 (PID2-interacting E3) has been identified as a functional interactor of PID2-mediated rice blast resistance, with the armadillo repeat domain being essential for this protein–protein association. Functional characterization revealed that loss-of-function of OsPIE3 enhances resistance to the M. oryzae isolate ZB15, whereas constitutive overexpression of the gene compromises disease resistance. Additionally, the blast response observed in OsPIE3/PID2-double mutants closely resembled that of the PID2 single mutant, indicating that OsPIE3 functions as a negative regulator within the PID2-associated defense pathway. These findings emphasize the significance of ubiquitin-mediated regulation in rice immune signaling against blast disease [116].

5. OsBIERF3 (ERF transcription factor)- The rice ERF transcription factor gene OsBIERF3 plays an important regulatory role in plant immune responses and tolerance to abiotic stresses. Its transcriptional activation capability is dependent on the C-terminal region. Enhanced expression of OsBIERF3 significantly improves resistance against both M. oryzae and X. oryzae, whereas suppression of the gene results in increased disease susceptibility. Furthermore, OsBIERF3 positively regulates the expression of genes involved in cell wall biosynthesis, leading to increased cell wall thickening in overexpression lines and reduced wall integrity in OsBIERF3-suppressed plants. These observations suggest that OsBIERF3-mediated modulation of structural defense components contributes substantially to rice disease resistance mechanisms [117].
6. OsFBX156 / OsHSP71.1-The F-box protein OsFBX156 functions as a positive regulator of innate immune responses in rice through its interaction with the heat shock protein OsHSP71.1. Enhanced expression of OsFBX156, or knockout of OsHSP71.1, stimulates defense-associated mechanisms characterized by elevated transcription of pathogenesis-related genes and increased reactive oxygen species accumulation. These molecular responses collectively contribute to strengthened resistance against the rice blast pathogen M. oryzae, highlighting the significance of ubiquitin-mediated regulatory pathways in rice immunity [118].
7. OsMLO gene family- The rice genome encodes twelve MLO (Mildew resistance locus O) genes that display diverse spatial and physiological expression profiles, suggesting substantial functional specialization within the gene family. Distinct OsMLO members demonstrate preferential expression in specific tissues such as leaves, roots, root tips, mature pollen, and trinucleate pollen, reflecting potential roles in developmental regulation and tissue-specific physiological processes. Certain genes, including OsMLO1, OsMLO3, and OsMLO8, exhibit diurnal expression patterns, indicating their involvement in environmental response mechanisms. Furthermore, altered expression of these OsMLO genes in light-response-defective osdxr mutants, together with associated calmodulin gene suppression, suggests interactions with light-mediated signaling pathways. Additionally, OsMLO4 and OsMLO5 participate in heat- and cold-stress responses, respectively. Notably, pathogen-induced upregulation of OsMLO3 following M. oryzae infection implies a contributory role in rice defense and stress-responsive signaling networks [119].

8. Pi9/AvrPi9- The AvrPi9 locus of M. oryzae encodes a secreted avirulence effector associated with the biotrophic interfacial complex during host–pathogen interaction. Specific recognition of this effector by the rice resistance gene Pi9 initiates effector-triggered immune responses, thereby activating host defense mechanisms that confer resistance against rice blast disease. This interaction exemplifies the molecular basis of gene-for-gene resistance in rice–pathogen system [90].

1.2 WHEAT (Triticum aestivum)
Wheat blast, which originated in South America during the 20th century and has recently spread to Asia and Africa, produces symptoms on leaves similar to those of rice blast, but it also causes severe bleaching of wheat spikes [25, 28, 120-121].

1. Lr34- The wheat resistance gene Lr34 is recognized for conferring durable and broad-spectrum partial resistance against several biotrophic fungal pathogens, including rust fungi and powdery mildew, particularly in mature wheat plants. The resistant Lr34 allele originated through two gain-of-function modifications within an ATP-binding cassette (ABC) transporter gene during post-domestication evolution. When introduced into rice, the transgenic rice plants expressing Lr34 showed increased resistance against multiple isolates of the hemibiotrophic pathogen M. oryzae. The presence of Lr34 delayed pathogen colonization during the biotrophic phase of infection, thereby restricting disease progression and resulting in the formation of comparatively smaller necrotic lesions on rice foliage [122].

1.3 BARLEY (Hordeum vulgare)
1. MLA3 (NLR resistance gene)- The barley (H. vulgare) resistance gene Mildew locus a (Mla), belonging to the

nucleotide-binding leucine-rich repeat (NLR) class, has undergone extensive functional diversification, enabling distinct allelic variants to recognize specific isolates of the powdery mildew pathogen Blumeria graminis. Among these variants, MLA3 has been shown to detect the M. oryzae effector Pwl2, thereby activating effector-triggered immune responses that confer resistance to blast infection. Furthermore, homologous resistance genes of Mla including Sr33 in wheat (T. aestivum) and Sr50 in rye (Secale cereale) provide effective resistance against multiple races of the stem rust pathogen Puccinia graminis, illustrating the evolutionary conservation of disease-resistance mechanisms among cereal crops [123].

2. 2. Rmoq1 (blast resistance QTL)- The quantitative trait locus Rmoq1, mapped to chromosome 7H in barley, has been identified as a significant genetic determinant of resistance against M. oryzae pathotypes capable of infecting wheat and rice. This locus accounts for a considerable proportion of the observed phenotypic variability associated with blast resistance. Additionally, Rmoq1 is linked with genomic regions implicated in host–nonhost resistance responses, indicating its potential involvement in broad-spectrum defense mechanisms against fungal pathogens in cereal crops [124].

1.4 FINGER MILLET (E. coracana)

The production of millets is also affected by blast disease, mainly in Africa and India [1, 28, 125-126].

1. Prolamins are the grain storage proteins of finger millet and are structured into protein bodies that serve as a physical and a nutritional barrier due to their resistance against digestion by the insect and fungal proteases [127].

2. chi11 (rice chitinase gene)- The chi11 gene is a rice gene, encoding a chitinase enzyme. It was introduced into finger millet (E. coracana) through Agrobacterium-mediated transformation, where it hydrolyzes fungal cell wall chitin and inhibits pathogen growth. Transgenic plants showed significantly increased chitinase activity and enhanced resistance to leaf blast caused by P. grisea compared with non-transgenic plants [128].

3. EcRGHs (finger millet resistance gene homologs)- Multiple nucleotide-binding site leucine-rich repeat (NBS-LRR) resistance gene homologs were characterized in finger millet (E. coracana) through PCR-based amplification employing degenerate oligonucleotide primers designed from conserved regions of previously identified plant resistance genes (R-genes). Sequence analyses revealed the presence of highly conserved motifs, including kinase-2 and kinase-3a domains, which are functionally associated with pathogen detection and downstream activation of host defense signaling pathways. These findings suggest a potential role of the identified EcRGHs in mediating resistance against fungal diseases such as blast [12].

1.5 FOXTAIL MILLET (Setaria italica)

1. Chitinase gene family (GH18/GH19)- Comprehensive genome-wide investigations in foxtail millet (S. italica) have identified forty chitinase-encoding genes predominantly classified within the GH18 and GH19 families. These genes encode enzymes that play critical role in plant defense by catalyzing the hydrolysis of chitin, a major structural component of fungal cell walls, thereby restricting pathogen proliferation. Functional analyses further indicate that these chitinase genes participate in pathogen-responsive defense mechanisms and stress-associated signaling networks, contributing to resistance against fungal pathogens including M. grisea [130].

2. CNL (NBS-LRR) resistance genes- A comprehensive genome-wide survey in foxtail millet (S. italica) revealed the presence of 242 Coiled-coil, Nucleotide-binding site, Leucine-rich repeat (CNL) R-genes associated with pathogen effector recognition and activation of defense responses against pathogens such as M. grisea. A substantial proportion of these genes are organized in clustered genomic arrangements and exhibit significant homology with established cereal resistance genes such as Pib and Pi9. The observed structural organization and evolutionary conservation suggest an important role for these CNL genes in the diversification and evolution of disease resistance mechanisms within cereal crops [131]. Table 2 summarizes the major blast resistance genes identified in cereal crops, along with their classification and functional roles in disease resistance. These genes contribute to pathogen recognition, activation of plant defense responses, and the development of durable resistance against blast disease. Blast disease, caused by Magnaporthe oryzae (formerly Pyricularia grisea), affects several cereal crops, including rice, wheat, foxtail millet, and finger millet. Among these, finger millet experiences substantial yield losses due to severe pathogen infection, highlighting the importance of developing durable disease resistance (Figure 2).

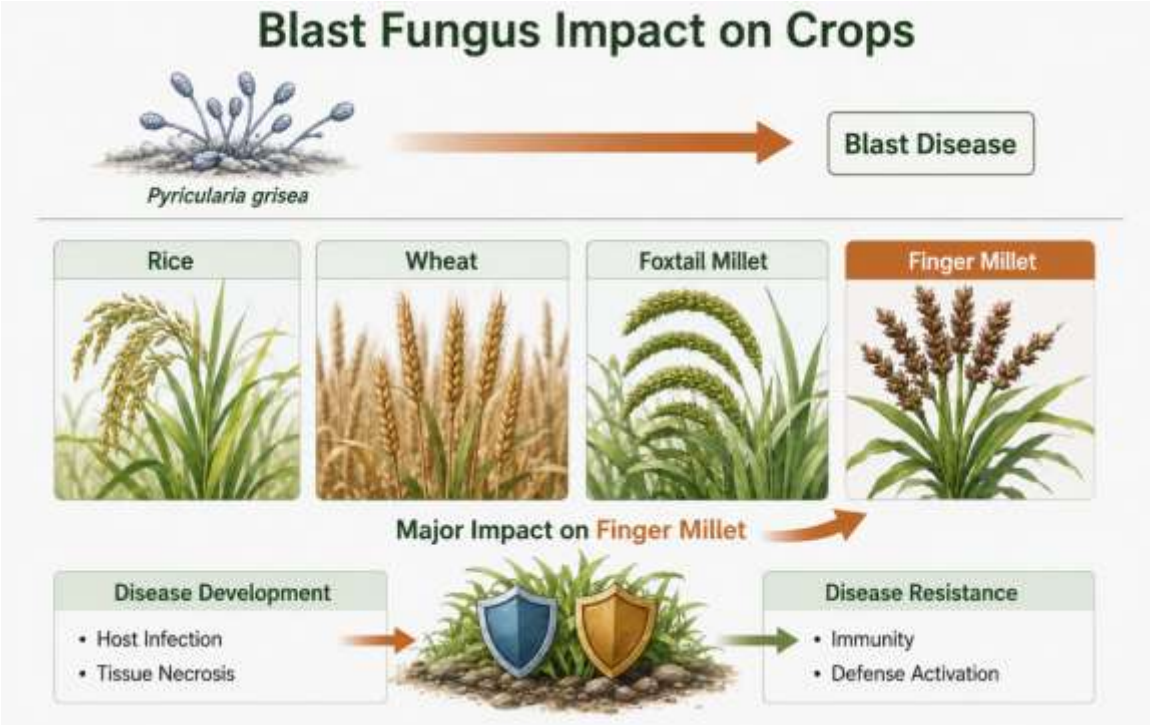

Figure 2. Impact of Pyricularia grisea Infection on Major Cereal Crops with Emphasis on Finger Millet

Table 3. Genes in cereal crops against blast disease.

Gene / Locus	Crop	Type	Function or Role in disease resistance	References
OsRING113	Rice	RING-type E3 ubiquitin ligase	Regulates broad-spectrum disease resistance by ubiquitinating and degrading APIP5	[113]
DEP2 / OsRING80	Rice	E3 ubiquitin ligase	Does proteosoal degradation of DEP2, Loss of OsRING80 enhances blast resistance	[114]
OsSEC3A	Rice	Exocyst complex component	Exocyst complex subunit in rice, disruption increases salicylic acid accumulation and resistance to M. oryzae	[115]
OsPIE3 / PID2	Rice	E3 ubiquitin ligase	Loss of OsPIE3 enhances blast resistance, functions along with PID2	[116]
OsBIERF3	Rice	Transcription factor	Functions in defense and abiotic stress tolerance, overexpression increases resistance against blast	[117]
OsFBX156 / OsHSP71.1	Rice	F-box protein	Targets OsHSP71.1, positively regulates rice innate immunity	[118]
OsMLO gene family	Rice	Transmembrane proteins	Involved in stress responses and pathogen interaction	[119]
Pi9	Rice	Resistance gene	Recognizes Avr-Pi9 of fungus and triggers effector-triggered immunity (ETI)	[90]
Lr34	Wheat	ABC transporter protein	Provides durable broad-spectrum resistance to multiple pathogens in wheat; also confers resistance to rice blast when expressed in rice	[122]
MLA3	Barley	Resistance gene	Recognizes fungal effector Pwl2 and activates ETI	[123]
Rmoq1	Barley	Quantitative trait locus	Contributes to blast resistance and host-nonhost resistance	[124]
Prolamins	Finger millet	Storage proteins	Provide physical and mechanical barrier due to resistance to insect and fungal proteases.	[127]
chi11	Finger millet (transgenic)	Chitinase enzyme	Hydrolyzes fungal cell wall chitin and enhances resistance to P. grisea	[128]

EcRGHs	Finger millet	NBS-LRR resistance gene homolog	Contain conserved motifs associated with pathogen recognition and activation of plant defense responses	[129]
Chitinase gene family (GH18 / GH19)	Foxtail millet	Chitinase	Hydrolyse fungal cell wall	[130]
CNL resistance genes	Foxtail millet	NBS-LRR immune receptor	Recognize pathogen effectors and activate defense responses	[131]

CONCLUSION

Blast disease caused by P.grisea remains one of the most destructive fungal diseases affecting major cereal crops worldwide, particularly rice, wheat, barley, and millets. The pathogen has gained considerable scientific attention not only because of its severe yield losses but also due to its role as a model organism for studying plant–fungal interactions. The infection process of P.grisea is highly specialized and begins with the deposition of conidia on the host surface. Upon germination, the fungus forms an appressorium, a melanized infection structure capable of generating extremely high turgor pressure that allows the pathogen to mechanically penetrate the plant cuticle. After penetration, invasive hyphae colonize host cells, suppress plant immune responses, and lead to characteristic necrotic lesions on leaves, nodes, and panicles. There are many genes governing the steps but the virulence of the pathogen largely depends on secreted effector proteins that manipulate host cellular processes and suppress defense signaling pathways. These effectors interact with host resistance genes and this interaction decides whether infection will proceed or not. For infection, the fungus initially establishes a biotrophic phase in which it penetrates the host cell and forms invasive hyphae that grow within living plant cells without immediately killing them. These hyphae are surrounded by a specialized interface that facilitates nutrient uptake and secretion of fungal proteins that suppress host defenses and allow the pathogen to spread from cell to cell. As the infection progresses, the fungus transitions to a necrotrophic phase, where the integrity of the host cell structure is disrupted, leading to vacuole rupture, cell death, and extensive tissue damage. In this stage, the fungus grows more aggressively in the dead host tissue and spreads further through the plant, contributing to disease symptoms and lesion development. This shift from a biotrophic to a necrotrophic lifestyle enables the pathogen to first colonize living cells and later exploit dead tissue for continued growth and disease progression. Furthermore, during the course of the investigation aimed at identifying candidate genes associated with stress resistance in finger millet (E. coracana), a comparative genomic exploration was conducted across several related cereal and millet species, including foxtail millet (S. italica), barley (H. vulgare), wheat (T. aestivum), and rice (O.sativa). This analysis revealed a diverse set of homologous and functionally annotated genes distributed across these species that are known or predicted to participate in stress response pathways, defense signaling, and adaptive physiological regulation. The identification of these genes highlights the evolutionary conservation of resistance-related mechanisms among cereal crops and underscores the potential value of cross-species genomic resources. The homologs and candidate loci identified in these species may serve as important references for future functional validation and molecular characterization in finger millet. Consequently, these findings provide a useful foundation for the discovery and deployment of suitable resistance-associated genes that could be leveraged to enhance stress tolerance and improve the resilience of finger millet through molecular breeding or genetic engineering approaches.

Future study should emphasize integrated genomic and molecular approaches, detailed characterization of host–pathogen interactions, and the identification of stable resistance sources across diverse agro-climatic conditions. Strengthening genomic resources and advancing functional studies will be crucial for enhancing blast resistance and ensuring sustainable productivity of finger millet as a nutritionally important and climate-resilient crop.

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Authorship & Contributorship Details

Authors' Contributions

Conceptualization and design of the study were carried out by Amit Prakash Raghuwanshi. Literature collection, analysis and manuscript drafting were performed by Prerna Upadhyay. Critical revision and final approval of the manuscript were completed by Noopur Singh. Other co-authors reviewed the manuscript and rewritten the manuscript through various versions.

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Research Content

The manuscript is an original research article related to blast resistance in finger millet and other cereals. The content has not been published previously and is not under consideration for publication elsewhere.

Conflict of Interest
The authors declare that there are no conflicts of interest regarding the publication of this manuscript.
Data Availability

No new data were generated or analyzed in this study. All information discussed in this research is derived from previously published and cited sources.

Consent to Publish
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