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Pyricularia oryzae: Lab star and field scourge

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Abstract

Pyricularia oryzae (syn. *Magnaporthe oryzae*), is a filamentous ascomycete that causes a major disease called blast on cereal crops, as well as on a wide variety of wild and cultivated grasses. Blast diseases have a tremendous impact worldwide particularly on rice and on wheat, where the disease emerged in South America in the 1980s, before spreading to Asia and Africa. Its economic importance, coupled with its amenability to molecular and genetic manipulation, have inspired extensive research efforts aiming at understanding its biology and evolution. In the past 40 years, this plant-pathogenic fungus has emerged as a major model in molecular plant–microbe interactions. In this review, we focus on the clarification of the taxonomy and genetic structure of the species and its host range determinants. We also discuss recent molecular studies deciphering its lifecycle.

Taxonomy: Kingdom: *Fungi*, phylum: *Ascomycota*, sub-phylum: *Pezizomycotina*, class: *Sordariomycetes*, order: *Magnaporthales*, family: *Pyriculariaceae*, genus: *Pyricularia*.

Host range: *P. oryzae* has the ability to infect a wide range of *Poaceae*. It is structured into different host-specialized lineages that are each associated with a few host plant genera. The fungus is best known to cause tremendous damage to rice crops, but it can also attack other economically important crops such as wheat, maize, barley, and finger millet.

Disease symptoms: *P. oryzae* can cause necrotic lesions or bleaching on all aerial parts of its host plants, including leaf blades, sheaths, and inflorescences (panicles, spikes, and seeds). Characteristic symptoms on leaves are diamond-shaped silver lesions that often have a brown margin and whose appearance is influenced by numerous factors such as the plant genotype and environmental conditions.

USEFUL WEBSITES:

Resources	URL
Genomic data repositories	http://genome.jouy.inra.fr/gemo/
Genomic data repositories	http://openriceblast.org/
Genomic data repositories	http://openwheatblast.net/
Genome browser for fungi (including *P. oryzae*)	http://fungi.ensembl.org/index.html

Resources	URL
Comparative genomics database	https://mycocosm.jgi.doe.gov/mycocosm/home
T-DNA mutant database	http://atmt.snu.kr/
T-DNA mutant database	http://www.phi-base.org/
SNP and expression data	https://fungidb.org/fungidb/app/

KEYWORDS
blast disease, disease cycle, fungus, genome, *Magnaporthe oryzae*, pathogen, *Pyricularia oryzae*

1 | INTRODUCTION

Blast disease caused by the fungal pathogen *Pyricularia oryzae* (syn. *Magnaporthe oryzae*) is widespread and often very destructive. *P. oryzae* is a hemibiotrophic plant pathogen that can colonize a wide range of grass plants including many economically important crops. Rice blast is the most studied disease caused by the fungus (Dean et al., 2012; Lee et al., 2006; Ou, 1985; Zhang et al., 2016). It is estimated that 5% of the annual production of rice is destroyed by this disease (Savary et al., 2019). Rice blast is characterized by small lesions on all aerial parts of the plant, with sizes and colours varying depending on rice cultivars and environmental conditions. While relatively large grey spots (about 1 cm long) can be observed on the leaves of susceptible rice cultivars under high humidity conditions, lesions on more resistant cultivars usually remain small and are surrounded by a dark brown area (Ou, 1985). Wheat blast, which emerged in South America in the 20th century and recently moved to Asia and Africa, causes similar symptoms to rice blast on leaves, but also induces massive spike bleaching (Ceresini et al., 2018; Cruz & Valent, 2017; Islam et al., 2016). The production of millets is also affected by blast disease, mainly in Africa and India (Gupta et al., 2017; Odeph et al., 2020; Poonacha et al., 2023). The infection of panicles (rice, finger millet) or spikes (wheat) by the fungus can lead to a complete loss of the harvest (Asibi et al., 2019). Moreover, the capacity of *P. oryzae* to adapt to new plant species and varieties poses a significant threat to many cereal crops (Cruz & Valent, 2017; Latorre et al., 2023). This jeopardizes global food security, as cereals are a staple food for a large part of the world's population. Various management strategies are used to control the disease, ranging from agricultural practices, fungicide treatments, quarantine and the use of resistant varieties (for comprehensive reviews see Ceresini et al., 2018 and Skamnioti & Gurr, 2009). However, given the rise in global population and the demand for ecofriendly food production, it is vital to continue developing efficient and responsible management strategies.

Extensive research on *P. oryzae* has been developed worldwide due to the relative ease of its experimental manipulation and the importance of blast diseases (Valent, 1990). As a result, *P. oryzae* is now considered a primary model organism for plant-pathogenic fungi and numerous resources for research are available (Ebbole, 2008; Perez-Nadales et al., 2014). A decade ago, *P. oryzae* was elected the number 1 fungal pathogen in terms of its economic and scientific importance (Dean et al., 2012), a statement that remains just as relevant today. In addition to the reference genome published in 2005 (Dean et al., 2005), numerous isolates of the fungus have been sequenced, and a large amount of genomic data is now publicly available (Table S1) (Gladieux, Condon, et al., 2018; Langner et al., 2021; Pordel et al., 2021; Yoshida et al., 2016; Zhong et al., 2018). *P. oryzae* can also be genetically transformed and mutants can be generated using CRISPR-Cas9 and homologous recombination techniques (Foster et al., 2018; Leung et al., 1990), including the use of Ku70 or Ku80 mutants to increase recombination frequency (Villalba et al., 2008). The use of reporter genes and fluorescent markers has allowed a thorough and precise cytological description of the development and infection cycle of the fungus (Eseola et al., 2021; Oliveira-Garcia et al., 2023; Valent & Khang, 2010). Overall, the knowledge generated on *P. oryzae* is tremendous. In this review, we compiled an elementary description of the fungus and highlighted some of the recent discoveries.

2 | TAXONOMY, HOST RANGE AND THE POPULATION STRUCTURE

P. oryzae (syn. *M. oryzae*) is an Ascomycete belonging to the *Pyriculariaceae* family in the *Magnaporthales* order of the *Sordariomycetes* (Klaubauf et al., 2014). *Pyriculariaceae* have a pathogenic lifestyle and can infect a wide range of monocot plants including the *Poales* and *Zingiberales*. Fungi from the *Pyricularia* genus specifically infect the aerial parts of their hosts, with clear host specificities that may overlap. For instance, to date *P. ctenantheicola* and *P. zingibericola* were isolated from a single host (*Ctenanthe oppenheimiana* and *Zingiber officinale*, respectively). *Pyricularia grisea* causes disease on *Digitaria* spp., but it was also isolated from *Echinochloa* species. *Pyricularia penniseticola* causes disease on *Pennisetum* spp. but it was also isolated from *Digitaria* spp. *Pyricularia pennisetigena* was isolated from *Penisetum* and *Cenchrus* spp. (Klaubauf et al., 2014).

P. oryzae has been separated from *P. grisea* based on the concordance of multiple gene genealogies (Couch & Kohn, 2002; Klaubauf et al., 2014). Therefore, *P. oryzae* isolates were generally referred to

as *P. grisea* or *M. grisea* in older literature. *P. oryzae* causes epidemics on a wide range of plants in the *Poaceae* family. However, individual isolates of *P. oryzae* typically have a limited host range and can only infect a few plant species, generally within the same genus. This host specificity is reflected in the genetic structure of the species (Figure 1). *P. oryzae* is subdivided into multiple, genetically differentiated lineages that are associated with a limited number of hosts. Therefore, these associations are frequently called pathotypes. The low nucleotide divergence between the lineages and the persistence of a historical signal of gene flow between them indicates that their divergence is recent compared to the divergence between different *Pyricularia* species such as *P. oryzae* and *P. grisea* (Chiapello et al., 2015; Gladieux, Condon, et al., 2018; Thierry et al., 2022). Phylogenetic relationships between lineages indicate an evolutionary history involving multiple host shifts, host range expansion and genetic exchange among lineages (Couch et al., 2005; Gladieux, Ravel, et al., 2018; Inoue et al., 2017; Pordel et al., 2021; Rahnama et al., 2023). No obvious co-divergence or co-speciation could be observed between *P. oryzae* isolates and their host plants (Figure 1). This is consistent with the divergence time of *P. oryzae* being much more recent than the one of grasses (Christin et al., 2014; Gladieux, Condon, et al., 2018; Latorre et al., 2020).

The wheat blast lineage emerged in Brazil in the 1980s, with a first description in 1985, and subsequently spread to Bolivia, Paraguay, and Argentina before being independently introduced to South Asia and Africa in the last decade (Cruz & Valent, 2017; Latorre et al., 2023). This lineage was proposed as a new species, *Pyricularia graminis-tritici* (Castroagudín et al., 2016; Castroagudín et al., 2017; Ceresini et al., 2018; Ceresini et al., 2019), which was however refuted (Valent et al., 2019). Using 2682 single-copy orthologous genes from 76 strains collected from 12 host species, it was demonstrated that wheat-infecting isolates form a host-specific lineage within *P. oryzae* (Gladieux, Condon, et al., 2018). This lineage could not be diagnosed as a distinct species based on genealogical concordance for phylogenetic sequence recognition (Gladieux, Condon, et al., 2018). The different host-specific lineages within *P. oryzae* were connected by significant and relatively recent genetic exchanges and, therefore, correspond to a single species. More recently, the narrow host range and lineage affiliation of the wheat-infecting isolates of *P. oryzae* was further confirmed (Ascari et al., 2024).

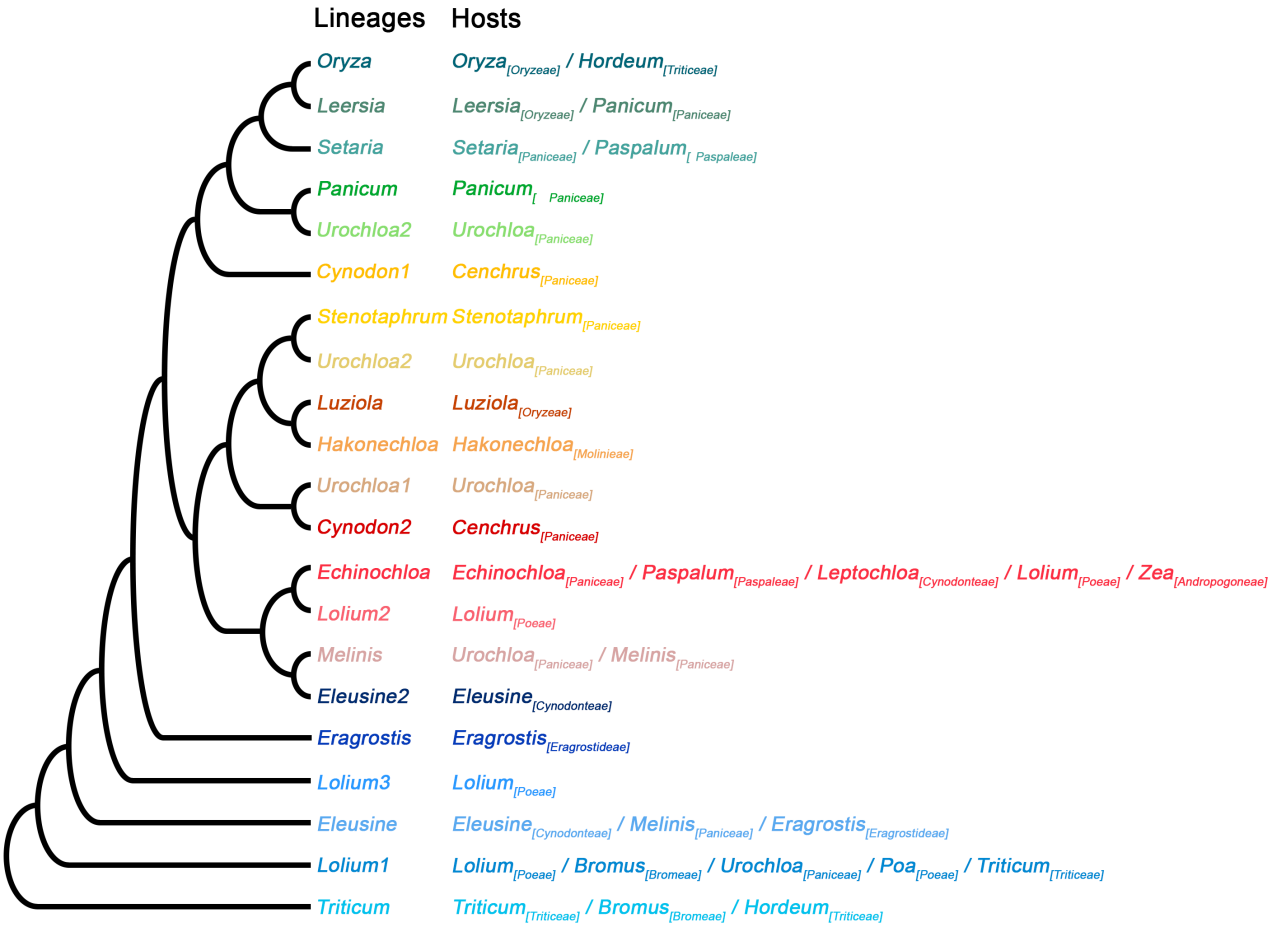

FIGURE 1 *Pyricularia oryzae* populations structure and their hosts of origin. Schematic distribution of *P. oryzae* main lineages and their hosts of origin. The host taxonomic tribe is indicated in brackets.

2.1 | The molecular genetics of host shifts and host-range expansion

Genetic analysis of crosses between isolates from different host-specific lineages have demonstrated that the incompatibility of *P. oryzae* on nonhost species is in most cases controlled by relatively few genes, generally one to three (Cruz & Valent, 2017; Murakami et al., 2003; Takabayashi et al., 2002; Tosa et al., 2006; Valent & Chumley, 1991). Several of these genes code for effector proteins such as PWL1 and PWL2 that control pathogenicity towards *Eragrostis curvula* (weeping lovegrass) or PWT3, PWT4 or PWT7 that control pathogenicity on wheat (Asuke et al., 2023; Inoue et al., 2017; Sweigard et al., 1995). In all studied cases, it was the loss-of-function alleles that conferred compatibility on the new host, suggesting that the host specificity of the blast fungus is frequently determined by immune receptor-mediated detection of virulence effectors in nonhost plants. Such mechanisms are supported for PWT3 and PWT4, which are detected in wheat by the products of typical resistance genes, the nucleotide-binding and leucine-rich repeat (NLR) domain immune receptor RWT3 (Rmg1) and the tandem-protein kinase RWT4 (Rmg6), which is allelic to the race-specific powdery mildew resistance protein Pm24 (Arora et al., 2023). Pathogenicity genes required specifically for the infection of some but not all host species of *P. oryzae* have not yet been identified but may also act as host range determinants in the blast fungus.

Consequently, in the host shifts of the blast fungus, the loss of avirulence effectors appears as the critical step in the establishment of basal compatibility on new host species (Inoue et al., 2017). These founding events are believed to be followed by thorough adaptation to the new plant involving adaptive changes at numerous loci and resulting in a truly adapted pathogen able to cause epidemics on its new host (Le Naour-Vernet et al., 2023). An example of a potentially ongoing adaptation to a new host of the blast fungus is the locally restricted emergence of maize-specific genotypes within populations of the barnyard grass pathotype in northern Iran (Pordel et al., 2021).

2.2 | Population structure of the rice-attacking lineage

The rice-infecting lineage of *P. oryzae* is composed of one genetically diverse recombinant lineage and three clonal lineages with broad and largely overlapping geographic distributions (Gladieux, Ravel, et al., 2018; Latorre et al., 2020; Thierry et al., 2022; Zhong et al., 2018). The coexistence of clonal pandemic lineages despite compatible mating types is caused by reproductive isolation and niche separation. Indeed, the three clonal lineages of *P. oryzae* have lost fertility, are adapted to different subspecies of rice, and occur in areas with different prevailing environmental conditions (Thierry et al., 2022). The recombining lineage can be further subdivided into one widely distributed group, which has lost female fertility, and three fertile groups that are restricted to South-east Asia (Thierry et al., 2022).

3 | GENOMICS

Analysis of the genomic sequences of 120 *P. oryzae* isolates from various host-specific lineages revealed genome sizes of 37–43 Mb with a mean of 40 Mb and standard deviation of 2 Mb (Le Naour-Vernet et al., 2023) (Figure 2a). Coding sequences make up roughly half of the genome and encode 11,732 genes on average ($SD = 148$) resulting in a mean gene density of one gene per 3.25 kb (Figure 2b). Similar characteristics of *P. oryzae* genomes were found in other genome studies (Table S1). Orthology analysis in the above-mentioned 120 *P. oryzae* genomes identified 8374 core orthogroups present in all isolates, 840 softcore genes present in >99% isolates, 874 shell genes present in 1%–99% isolates, and 4679 cloud genes present in <1% isolates (Le Naour-Vernet et al., 2023).

About 15% of the genes encode secreted proteins, among which 684 or 849 are effector-like in the Guy11 reference isolate according to the effector prediction algorithms EffectorP or DeepRedEff (Kristianingsih & MacLean, 2021; Le Naour-Vernet et al., 2023; Sperschneider & Dodds, 2022; Yan et al., 2023) (Figure 2b). Among these candidate effectors, 550 are expressed in planta (Yan et al., 2023).

Repeated genetic elements contribute to genome plasticity in *P. oryzae*. The content in transposable elements and repeat sequences in *P. oryzae* genomes varies between 5% and 11%, which is moderate for phytopathogenic ascomycetes (Chiapello et al., 2015; Dean et al., 2005; Nakamoto et al., 2023). Genes that show presence/absence polymorphism between lineages are frequently associated with transposable elements, suggesting that transposons are implicated in their acquisition and/or loss (Yoshida et al., 2016). The underlying mechanisms have been addressed in the case of LTR retrotransposons that tend to form extrachromosomal circular (ecc) DNAs, which presumably promote gene loss or gene duplications (Joubert & Krasileva, 2022). Such genomic dynamics are frequent for effector-encoding genes. For instance, the effector gene *AVR-Pita* is highly variable in its genome localization and frequently flanked by a retrotransposon that has presumably fostered multiple translocations, duplications, and losses (Chuma et al., 2011). In addition, retrotransposons presumably cause the high instability of chromosome ends in *Lolium*-infecting isolates whose telomeres rearrange at high frequency (Rahnama et al., 2020). Minichromosomes, that is, chromosomes smaller than 3 Mb that occur in addition to the seven core chromosomes (Orbach et al., 1996; Talbot et al., 1993), have also been associated with dynamic structural rearrangements, including interchromosomal translocations and segmental duplications (Chuma et al., 2011; Kusaba et al., 2014). Minichromosomes can carry virulence-related genes, but they are generally enriched

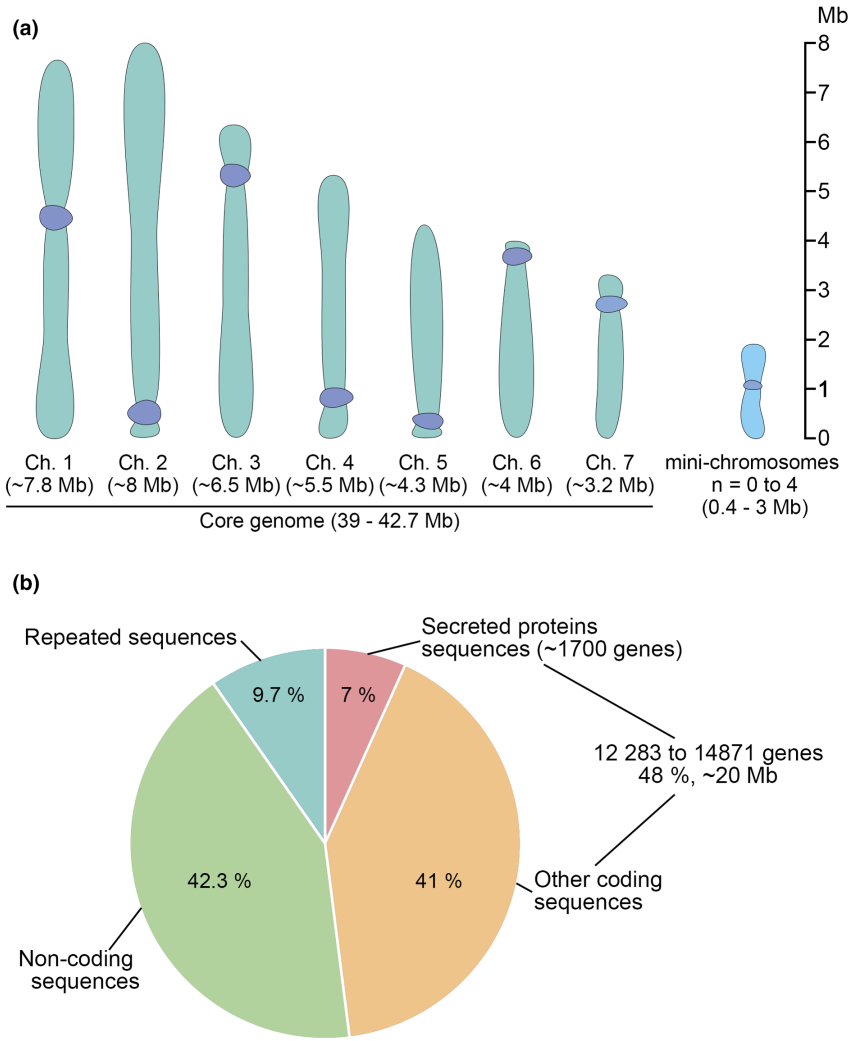

FIGURE 2 *Pyricularia oryzae* genome data. (a) Schematic representation of *P. oryzae* chromosomes (Ch.) and their estimated size in megabases (Mb) The approximate position of centromere according to Yadav et al. (2019) is represented by the dark blue shape. (b) Relative genome content of *P. oryzae* reference isolate 70–15 (Dean et al., 2005).

in repetitive elements and have lower gene density than core-chromosomes (Langner et al., 2021; Peng et al., 2019).

4 | MOLECULAR MECHANISMS GOVERNING THE LIFE CYCLE

Numerous tools have been developed to study the development of the fungus during its infectious cycle and to analyse the molecular processes allowing *P. oryzae* to infect the host plants. In this section, we describe the molecular mechanisms involved at different stages of *P. oryzae* rice infection. These steps are summarized in Figure 3, which provides a detailed cytological description of the infection cycle of *P. oryzae*. Although most of the mechanisms described hereafter were discovered in the rice–*P. oryzae* pathosystem, it is likely that these observations can be extended to the interaction of *P. oryzae* with other host plants. A description of large-scale mutagenesis studies in the blast fungus is provided in the review article from Motaung et al. (2017). For a comprehensive listing and description of the about 400 *P. oryzae* genes that have been studied by mutant analysis, we recommend the very exhaustive review article of Tan et al. (2023).

4.1 | Adhesion

P. oryzae can infect the aerial parts of rice (i.e., leaves, stems, sheaths and panicles) at all stages of its development. The life cycle usually begins with the landing and adhesion of an asexual three-celled conidium on the hydrophobic cuticle of a host leaf. Adhesion is critical for infection and depends on the secretion at the conidial apex of an extracellular matrix, also called spore tip mucilage (Howard & Valent, 1996; Rocha et al., 2020). According to chemical, enzymatic and immunological studies, it is constituted by a fibrous mixture of polysaccharides and proteins similar to what has been described in other plant-pathogenic fungi (Apoga & Jansson, 2000; Ebata et al., 1998; Hamer et al., 1988; Inoue et al., 2007; Inoue et al., 2011; Ohtake et al., 1999). The precise composition and biosynthetic pathways of the spore tip mucilage is still unknown. However, it was recently shown that the spermine synthase-encoding gene *SPS1* is essential for appressorial adhesion (Rocha et al., 2020). Although spermine is probably not a component of the extracellular matrix, it is involved in scavenging reactive oxygen species (ROS) in the endoplasmic reticulum (ER). This property allows the correct folding of glycoproteins from the extracellular matrix before their secretion (Rocha et al., 2020). Several other genes such as the O-mannosyltransferase *Pmt2*, the transcription factor *Tra1p*, the

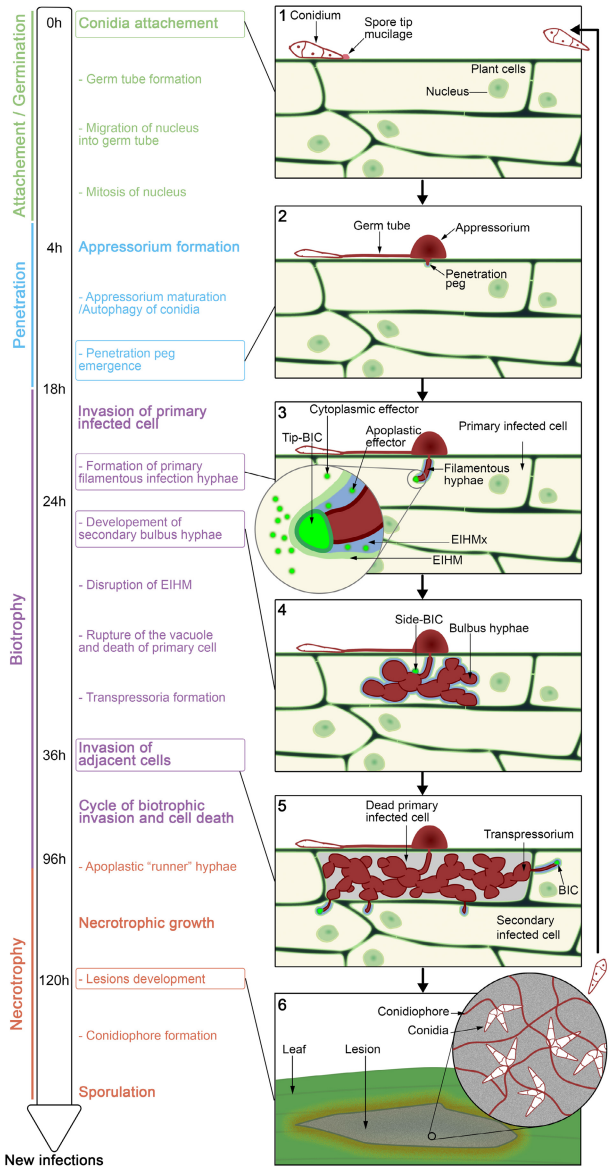

FIGURE 3 The cellular biology of *Pyricularia oryzae* life cycle. When an asexual spore (called a conidium) lands on the surface of a plant leaf it secretes mucilage to adhere to the leaf surface within minutes (Hamer et al., 1988) (panel 1). Following adhesion, the three-celled conidium forms a germ tube from its apex. Shortly after, the tip of the hypha hooks and swells to form a specialized structure called an appressorium. During maturation, the appressorium is wrapped in a thick layer of melanin that retains glycerol, which translates into water uptake and a massive increase of turgor pressure (up to 8 MPa) (panel 2). Rearrangements of the cytoskeleton target the pressure towards the base of the appressorium, which breaks the leaf cuticle and penetrates the host epidermis cell about 20 h after adhesion of the conidium (Ryder et al., 2013). The penetration peg then differentiates into a primary invasive hypha that progresses in the host cytoplasm surrounded by the extra-invasive hyphal matrix (EIHMx) and the extra-invasive hyphal membrane (EIHM) (panel 3) (Oliveira-Garcia et al., 2023). At the tip of the filamentous invasive hypha a specific dome-shaped structure called the biotrophic interfacial complex (BIC) is formed (Khang et al., 2010; Valent & Khang, 2010). The BIC is a mainly plant-derived structure that seems to be critical for the secretion of effector proteins in the host cytoplasm while apoplastic effectors localized in the EIHMx are secreted via the classic secretory pathway (Giraldo et al., 2013). The development of intracellular hyphae then switches to form bulbus hyphae that invade the host cell while still being enclosed by the EIHM (panel 4). The tip-BIC is moved to the side of the first bulbus hypha, which correlates with a switch in vesicle content (Oliveira-Garcia et al., 2023). Around 30 h post-infection, the EIHM ruptures leading to the spill of the EIHMx content in the cytoplasm, and the vacuole of the host cell shrinks (Jones et al., 2021). The vacuole then disrupts, which correlates with the death of the primary infected cell. The invasive hyphae continue to grow in the dead cell, becoming more filamentous and getting closer to the cell wall (Jones et al., 2021). Movement from cell to cell is mediated by the localization of hyphae to pit fields, the hyphae then undergo a constriction to go through the pit fields and invade the next cell; these structures are called transpressoria (Cruz-Mireles et al., 2021; Sakulkoo et al., 2018) (panel 5). The invasion of adjacent cells follows a similar pattern to the infection of the primary cell with first a filamentous hypha emerging from the pit field, capped by a BIC and surrounded by the EIHM. The cycle of biotrophic invasion and cell death continues until around 4 days after the start of the infection, when the fungus undergoes another switch in development with necrotrophic growth characterized by the development of apoplastic filamentous hyphae. This coincides with the appearance of symptoms in the form of diamond-shaped silver lesions. Asexual reproduction is initiated with the formation of conidiophores outside the leaf tissues initiating a new cycle of infection (Lau & Hamer, 1998) (panel 6).

fasciclin-like protein *Flp1*, and the ubiquitin ligases *Rad6* and *Bre1* showed pleiotropic phenotypes when mutated, including defects in conidial adhesion (Breth et al., 2013; Guo et al., 2016; Liu et al., 2009; Shi et al., 2016; Tan et al., 2023).

4.2 | Germination and appressorium formation

Following the attachment of the three-celled conidium on a leaf surface and its contact with water, a germ tube emerges from its apical cell. The germ tube stops its apical growth when differentiating into an appressorium within 4 to 6 h (Howard & Valent, 1996; Jelitto et al., 1994). The appressorium is a melanized, dome-shaped cell that allows the fungus to break through the leaf cuticle and penetrate epidermal cells. The differentiation of germinating hyphae into appressoria is dependent on environmental signals such as surface hardness and hydrophobicity, cell cycle progress, as well as induced autophagy (Ryder & Talbot, 2015). 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 (Fernandez & Orth, 2018; Li et al., 2012; Ryder & Talbot, 2015; Tan et al., 2023; Wilson & Talbot, 2009). 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 (DeZwaan

et al., 1999). The signals triggering the MAP kinase cascade are not fully understood but are downstream of the cAMP signalling pathway (Xu & Hamer, 1996). Multiple components regulating the activation of Pmk1 and downstream responses have been identified (Fernandez & Orth, 2018; Li et al., 2012; Osés-Ruiz et al., 2021; Turrà et al., 2014). Analyses of the *pmk1* deletion mutant or the analogue-sensitive mutant *pmk1*^{AS} revealed that Pmk1 is required for the control of appressorium development and cell-to-cell movement during invasive growth (Eseola et al., 2021; Sakulkoo et al., 2018; Xu & Hamer, 1996).

Cell cycle regulation also plays a prominent role in controlling appressorium development and maturation (Oses-Ruiz et al., 2017; Veneault-Fourrey et al., 2006). Indeed, the germ tube must undergo two independent DNA replications (S-phase) (Oses-Ruiz et al., 2017). Entry in the first S-phase is required to initiate appressorium development and the separation of the germ tube from the appressorium by a septum (Saunders et al., 2010). The second S-phase is a checkpoint for the build-up of turgor pressure in the appressorium (Oses-Ruiz et al., 2017). Nonselective autophagy of the conidium occurs after the initiation of appressorium development and is required to recycle nutrients from the conidium to the appressorium (Kershaw & Talbot, 2009). Autophagy is tightly correlated with the cell cycle progression and is another checkpoint for appressorium maturation (Marroquin-Guzman et al., 2017; Veneault-Fourrey et al., 2006). Disruption of macroautophagy by mutating *ATG* (autophagy-related) genes results in the loss of pathogenicity for all tested *ATG* genes except *ATG13* (Kershaw & Talbot, 2009; Zhu et al., 2019). The acetylation of Atg3 and Atg9 by the histone acetyltransferase Hat1 is particularly important to promote autophagosome formation (Yin et al., 2019). During prepenetration events, the metabolic state of the fungus is monitored by the conserved target of rapamycin (TOR) kinase that negatively regulates autophagy (Marroquin-Guzman & Wilson, 2015). Upon conidial germination and in response to glucose deficiency, TOR is inactivated, thereby alleviating the repression of cAMP/Pmk1 signalling, autophagy, and cell cycle progression required for appressorium morphogenesis (Fernandez et al., 2014; Marroquin-Guzman et al., 2017; Marroquin-Guzman & Wilson, 2015; Sun et al., 2019).

4.3 | Appressorium maturation and penetration into host plant cells

Appressoria are formed by many fungi but the shape and maturation of appressoria can differ between species (Demoor et al., 2019; Ryder et al., 2022; Shi et al., 2023; Thilini Chethana et al., 2021). This structure has been particularly well studied in *P. oryzae* and multiple excellent reviews are available (Cruz-Mireles et al., 2021; Eseola et al., 2021; Foster et al., 2017; Ryder et al., 2022; Ryder & Talbot, 2015). The maturation of the appressorium is critical to strengthening the structure and to sustain a high turgor pressure (Ryder & Talbot, 2015). The appressorium cell wall is reinforced by the deposition of a melanin layer, except at its base where the pore is later formed. This creates a tight cellular compartment in which glycerol accumulates rapidly, leading to the uptake of water and the build-up of an internal turgor of 8.0 MPa (80 bars) (de Jong et al., 1997; Foster et al., 2017; Howard et al., 1991). The second phase of appressorium maturation is the repolarization of the cytoskeleton at the base of the appressorium near the pore (for reviews, see Eseola et al., 2021; Ryder et al., 2022). The F-actin network is rearranged in a ring-shape fashion by septin proteins (Dagdas et al., 2012; Dulal et al., 2021; Gupta et al., 2015; Ryder et al., 2013). In addition to the septin proteins (Sep3, Sep4, Sep5, Sep6), the exocyst components (Sec3, Sec5, Sec6, Sec8 Sec15, Exo70, Exo84) as well as Rvs167 and Tea1 colocalize with the F-actin network at the base of the appressorium to initiate the penetration peg differentiation (Dagdas et al., 2012; Gupta et al., 2015). This process is tightly regulated by the turgor-sensing kinase Sln1 ensuring that the right pressure is reached in the appressorium and mediating the recruitment of the NADPH oxidase required for the assembly of septin proteins (Ryder et al., 2013, 2019). As the penetration of the cuticle is essential for infection success, the components regulating the maturation of the appressorium are ideal targets for fungicides, including melanin biosynthesis inhibitors that have been widely used to control rice blast (He et al., 2020; Lopez-Moya et al., 2021).

4.4 | Biotrophic stage of infection

During the first step of appressorium-mediated penetration, a narrow penetration peg breaches the host surface and enters the first host plant cell, where it differentiates into a thin primary invasive hypha (Bourett & Howard, 1992; Kankanala et al., 2009; Khang et al., 2010). The primary invasive hypha differentiates into secondary infection hyphae. These secondary infection hyphae are bulbous, ramified and invade the cytoplasm of the first infected plant cell. Infection hyphae are surrounded by an extra-invasive hyphal matrix (EIHMx) in close contact with the plant extra-invasive hyphal membrane (EIHM), creating an extracellular compartment that seems sealed from the apoplast (Kankanala et al., 2009). The TOR signalling pathway, and in particular the Imp1 protein, play important roles in maintaining the EIHM integrity (Sun et al., 2018).

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 (Eseola et al., 2021; Kankanala et al., 2007; Mentlak et al., 2012; Oliveira-Garcia et al., 2023). 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 (Eseola et al., 2021; Giraldo et al., 2013; Mentlak et al., 2012). 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 (Qian et al., 2022). 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 (Chen, Shi, et al., 2014; Mentlak et al., 2012). Importantly, during biotrophic invasion, *P. oryzae* secretes other types of molecules in the apoplast such as secondary metabolites or enzymes that can target various host processes such as metabolism or mediate cell wall modification (Jung et al., 2012; Yan et al., 2023).

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 (Giraldo et al., 2013; Khang et al., 2010; Oliveira-Garcia et al., 2023). Cytoplasmic effectors including AVR-Pik (Eseola et al., 2021), PWL2, Rbf1 (Nishimura et al., 2016), MoHTR3 (Lee et al., 2023) and Bas1 (Khang et al., 2010) accumulate at high levels in the BIC. These cytoplasmic effectors are secreted by a Golgi-independent and brefeldin A-insensitive secretion pathway that involves the exocyst components Exo70 and Sec5, and the t-SNARE Sso1 protein (Giraldo et al., 2013; Yan et al., 2023). BIC formation requires the cytoplasmic effector Rbf1 (Nishimura et al., 2016). A recent study showed that cytoplasmic effectors are packaged into dynamic vesicle-like membranous compartments within the BIC and the host cytoplasm (Nishimura et al., 2016; Nishizawa et al., 2016; Oliveira-Garcia et al., 2023; Oliveira-Garcia et al., 2024). These puncta colocalize with the plant plasma membrane and with the Clathrin Light Chain 1 protein (CLC1), implicating clathrin-mediated endocytosis in effector translocation. A role for the Bas83 effector in recruiting plant membrane fragments for endocytosis at the BIC has been suggested (Oliveira-Garcia et al., 2023). Some cytoplasmic effectors translocated into the plant cell move to uninvaded neighbouring cells, presumably through plasmodesmata (Khang et al., 2010). 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 (Oliveira-Garcia et al., 2023). It has been recently shown that effectors addressed to the BIC undergo a fine-tuned regulation at the translational level (Li, Dulal, et al., 2023). The E1-like Urm1-activating enzyme UBA4 mediates the tRNA anticodon wobble uridine 2-thiolation, a modification associated with AA-ending codons particularly present in cytoplasmic effector mRNAs (Li, Dulal, et al., 2023). Analysis of codon usage in effector mRNA could therefore be a new way to predict whether the protein will be translocated in the host cell. A significant number of cytoplasmic effectors have been detected by their localization in the BIC and/or their translocation into plant cells (Dong et al., 2015; Jung et al., 2012; Le Naour-Vernet et al., 2023; Mosquera et al., 2009; Yan et al., 2023), but their roles in infection remain mostly uncharacterized. The effectors Avr-Pita, AvrPiz-t and Avr-Pii play a role in ROS burst inhibition (Han et al., 2021; Liu & Zhang, 2022; Oliveira-Garcia et al., 2024; Park et al., 2012; Park et al., 2016; Singh et al., 2016). The effectors HTR1, HTR2, HTR3 and lug4 target host transcriptional reprogramming (Kim et al., 2020; Lee et al., 2023; Liu et al., 2022). The effectors HTR3, lug4, lug6 and lug9 modulate plant hormonal pathway (Dong et al., 2015; Lee et al., 2023). AvrPiz-t has also been shown to interfere with host protein degradation (Park et al., 2012; Park et al., 2016). Finally, Avr-Pii targets vesicle trafficking (Fujisaki et al., 2015).

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 (Li et al., 2009), AvrPib (Zhang et al., 2015), AVR-Pii (Yoshida et al., 2009), AVR-Pia (Miki et al., 2009; Yoshida et al., 2009), AVR1-CO39 (Farman & Leong, 1998; Ribot et al., 2013), AVR-Pi9 (Wu et al., 2015) and AVR-Pik (Yoshida et al., 2009), which are respectively recognized in rice by Piz-t (Zhou et al., 2006), Pib (Wang et al., 1999), Pii (Yoshida et al., 2009), Pi-a (Cesari et al., 2013; Okuyama et al., 2011), Pi-CO39 (Cesari et al., 2013; Chauhan et al., 2002), Pi9 (Qu et al., 2006) and Pi-k (Ashikawa et al., 2008; Maqbool et al., 2015).

Many cytoplasmic effectors belong to structural families, such as the MAX (*Magnaporthe* Avrs and ToxB like) effectors expanded in *P. oryzae* (de Guillen et al., 2015). MAX effectors are massively and specifically expressed during early biotrophy (de Guillen et al., 2015; Le Naour-Vernet et al., 2023; Yan et al., 2023). They include many AVR proteins recognized by intracellular NLR immune receptors (Lahfa et al., 2023). Additional structural families of *P. oryzae* effectors have been identified by genome-wide modelling of protein structures (Derbyshire & Raffaele, 2023; Seong & Krasileva, 2021; Seong & Krasileva, 2023).

Recently, Yan et al. (2023) reported the global pattern of fungal gene expression during rice blast infection. Their analysis described the temporal expression dynamics of the candidate effector repertoire and identified 10 modules of temporally co-expressed genes, often corresponding to key phases of the life cycle. Interestingly, effectors belonging to the same structural classes such as the MAX, ART, hydrolase or glucosidase families are co-expressed during biotrophic invasion (Yan et al., 2023). 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 (Cao et al., 2022; Lambou et al., 2024), as well as the WOPR box transcription factor MoWOR1 (syn. MoGTI1) that acts in *P. oryzae* like in numerous other pathogenic fungi as a central regulator of pathogenicity (Chen, Zhai, et al., 2014; Li et al., 2016). Rgs1 has been shown to be required for the repression of a large group of effectors and to prevent their

expression before the infection of plant tissues (Tang et al., 2023). Histone modifications also play a central role in keeping in planta-induced genes silenced in axenic growth (Zhang et al., 2021).

A particularly important virulence function of *P. oryzae* 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 (Fernandez et al., 2014), nitro-oxidative damage responses involving the nitronate monooxygenases Nmo2 (Marroquin-Guzman et al., 2017), and ROS scavenging by glutathione involving the glutathione peroxidase Hyr1 (Huang et al., 2011) and the glutathione reductase Gtr1 (Fernandez & Wilson, 2014). 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 (Li, Gong, et al., 2023).

Regulation of these oxidative stress response systems is highly complex and involves numerous signalling proteins of the blast fungus. Examples are the bZIP transcription factor MoAP1 that is a conserved master regulator of the oxidative stress response in fungi, and is, therefore, critical for infection (Guo et al., 2011) and the E3 ubiquitin ligase Upl3 controlling the accumulation of the antioxidation response regulator Sir2 (Li et al., 2020). Other elements are the fungal-specific protein Des1 (Chi et al., 2009) that probably acts via the regulation of peroxidase gene expression, and the nucleoside diphosphate kinase Ndk1 that regulates the intracellular nucleotide pools to maintain redox balance via metabolic homeostasis (Rocha et al., 2020).

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 (Jones et al., 2021; Kankanala et al., 2007; Yan et al., 2023).

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 (Cruz-Mireles et al., 2021; Eseola et al., 2021; Sakulkoo et al., 2018). Invasion of neighbouring cells, although more rapid, follows a similar pattern to the infection of the primary cell. The infection starts first with a primary invasive hypha emerging from the pit field, capped by a BIC and surrounded by the EIHM, and is then followed by the differentiation of secondary infection hyphae that are bulbous and ramified (Eseola et al., 2021; Kankanala et al., 2007). The biotrophic invasion of plant cells lasts up to 4 days after the start of the infection. Subsequently, *P. oryzae* undergoes a switch in development and adopts a necrotrophic development.

4.5 | Necrotrophic stage of development

The switch from biotrophic to necrotrophic growth is not well understood and poorly documented in *P. oryzae*. This transition is characterized by the synchronous appearance of macroscopic symptoms 5 days post-infection and a change in the development of the fungus. *P. oryzae* begins to produce thin filamentous hyphae that develop in the apoplast. At the molecular level, a drastic switch in the gene expression patterns occurs, characterized by the downregulation of biotrophy genes (including biotrophic cytoplasmic effectors) and the induction of genes specifically associated with the later stages of infection (Yan et al., 2023). Prominent necrotrophy-associated genes are cell-wall degrading enzymes (e.g., hydrolases, glucosidases and glycosyl hydrolases) and necrosis inducing proteins such as NLP1 (Mogga et al., 2016; Yan et al., 2023). Recently, it was shown that the downregulation of *MIF1* that inhibits plant cell death is associated with the switch from biotrophy to necrotrophy (Galli et al., 2023). However, to this day, we are lacking thorough functional analyses of the necrotrophy-associated genes, and this stage therefore remains one of the last largely uncharacterized steps of the *P. oryzae* life cycle.

It has been argued that the death of the first infected cell (about 36 h post-infection) coincides with a transient necrotrophic phase (Figure 3). This phase is initiated after the collapse of the vacuole and lasts a few hours, until penetration into neighbouring cells (Jones et al., 2021). However, there is no marked expression of necrotrophy-associated genes at this stage and the fungus continues to colonize adjacent cells with a biotrophic lifestyle (Cruz-Mireles et al., 2021; Jones et al., 2021). This phase has therefore only limited resemblance to true necrotrophy.

4.6 | Reproduction

Like most ascomycetes, *P. oryzae* can reproduce both asexually and sexually. While sexual reproduction can easily be performed in the laboratory, it has never been observed in the field and most populations are clonal (Saleh et al., 2012; Thierry et al., 2022). *P. oryzae* has a haplontic life cycle. The multicellular state is haploid. The breeding system is heterothallic, with mating occurring only between haploid individuals of opposite mating types. Mating-type genes are localized by the *MAT* loci, which are annotated as *MAT1-1* and *MAT1-2* depending on the mating type (Notteghem & Silué, 1992). Several genes essential for the development of sexual organs and

the determination of sex type are encoded at the *MAT* loci (Wang et al., 2021). Successful mating also requires that at least one of the two partners is producing fertile trichogynes, which represent the female organs (Billiard et al., 2012). These female organs are fertilized by male gametes called microconidia (Chuma et al., 2009; Kato et al., 1994; Lassagne et al., 2022; Zhang et al., 2014). While the mating type is critical for sexual reproduction, it does not play a role in the virulence and asexual reproduction of the fungus (Wang et al., 2021). Interestingly, a few cycles of asexual reproduction in axenic culture have been shown to be sufficient to select female sterility mutants (Saleh et al., 2012). These findings could explain why most populations are clonal. Recently, the transcriptional regulator Pro1 was shown to be required for female fertility in *P. oryzae* (Uchida et al., 2023). In the field, the production of asexual spores called conidia is critical for propagating the disease. Several transcription factors have been identified as master regulators of conidiogenesis (Dong et al., 2016; Lau & Hamer, 1998; Lu et al., 2014). Even if no clear signalling pathway has been characterized so far, numerous *P. oryzae* mutants display conidiation phenotypes, including mutants from the MPS1/STL2 MAP kinase signalling pathway (Aliyu et al., 2019; Batool et al., 2021; Dubey et al., 2019; Fu et al., 2019; Guo et al., 2016; Kwon et al., 2018; Liu et al., 2009; Norvienyeku et al., 2017; Patkar et al., 2010; Ramanujam & Naqvi, 2010; Sangappillai & Nadarajah, 2020; Shi et al., 2016; Shi et al., 2019; Yan et al., 2011; Zhou et al., 2021).

5 | CONCLUSION

Despite having been at the forefront of molecular plant pathology studies for decades, and recent innovations in its control (Cadiou et al., 2023), *P. oryzae* remains a major obstacle to agricultural productivity. The fungus continues to impact rice production, and wheat blast is now damaging crops in multiple continents (Latorre et al., 2023). *P. oryzae* has also been detected on maize (Pordel et al., 2021). In addition, the *Lolium*-infecting lineage, which is closely related to the wheat-infecting lineage, is widespread in Europe, North America and probably elsewhere (Milazzo et al., 2018). These threats illustrate not only the increased need for epidemiological surveillance in this system, but also the lack of knowledge on the prevalence of the fungus on wild hosts and outside intensive agrosystems (Ali et al., 2023; Barragan et al., 2022).

Recent progress in solving and modelling protein folding opened a new means to study the biology of effectors and their cognate immune receptors. Structural biology allows us to define families of effectors by tertiary structure, which sheds a new light in the conservation of effectors across isolates and species (Le Naour-Vernet et al., 2023; Seong & Krasileva, 2021). The study of interactions between effectors and receptors, both at a structural and molecular level, has allowed molecular engineering of immune receptors. The *P. oryzae*–rice pathosystem leads the way with the model NLRs Pikp and RGA5 from rice that have been modified to extend their effector recognition spectrum (Cadiou et al., 2023; Cesari et al., 2022; Kourelis et al., 2023; Liu et al., 2021; Maidment et al., 2023; Zdrzałek et al., 2024). We can expect this field of research to be more prominent in the near future with potential applications in the breeding of resistant rice cultivars.

P. oryzae remains a premier model system with abundant biological resources and a highly active global scientific community. Among the aspects of the fungal life cycle that remain largely uncharacterized, we identified the regulation of virulence gene networks and the transition from biotrophy to necrotrophy. We can expect that the development of single-cell omics will allow a finer description of the fungal molecular mechanisms during infection. These approaches will be essential to understand how the fungus perceives its environment and switches from undercover biotrophic growth to destructive necrotrophic development. The thorough cytological studies that have been performed for the early stages of infection could also be extended to the later stages, leading to a better description of these events. Another area in which further studies are required is the characterization of fungal effectors virulence functions, which still remains very challenging in most pathosystems. As fungi secrete several hundreds of effectors, the individual contribution of each protein to the virulence as well as their eventual cooperative action is challenging to appreciate. Identification of host target proteins by proteomic and functional studies of polymutants that are both readily available in *P. oryzae*–rice pathosystem could be the way to make progress on this question. Another emerging topic is the integration of the abiotic and biotic environment for a more holistic understanding of plant–pathogen interactions. In this perspective, it will be particularly exciting to learn how *P. oryzae* interacts with the leaf microbiome in the different phases of its life cycle to efficiently exploit its host plants and to compete with other leaf-associated microorganisms.

In conclusion, we envision that research on *P. oryzae* will continue to play a pivotal role in addressing the threat of devastating crop diseases. This enduring prominence is owed to the methodological strengths inherent in studying the blast fungus, the profound insights gleaned from half a century of meticulous and rigorous scientific exploration into its biology, and the vitality of a dynamic research community. As the blast fungus continues to serve as a leading model in plant pathology, the collective efforts of researchers will ensure that it remains at the forefront in the ongoing battle to safeguard global agricultural productivity.

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DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analysed.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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