Trends in Microbiology | Microbe of the Month

Magnaporthe oryzae

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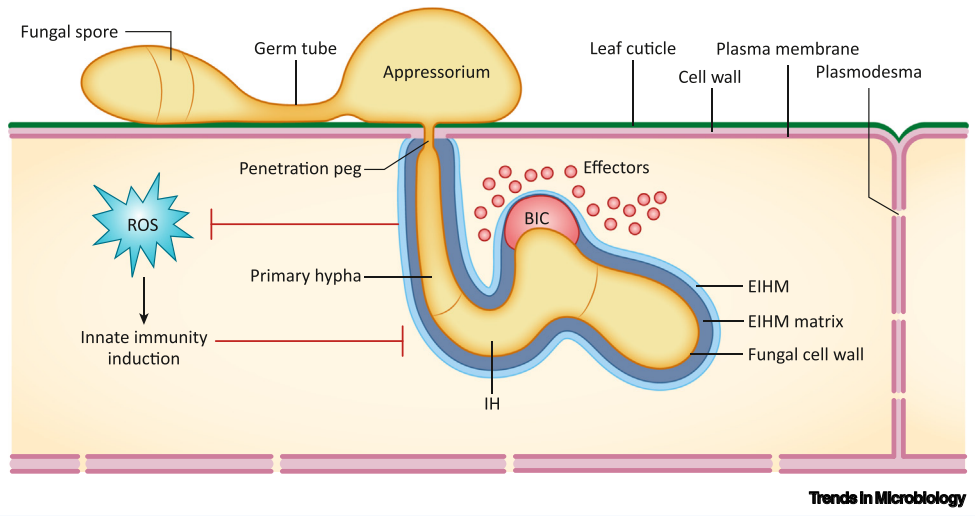

Figure 1. Snapshot of Early Host Invasion. This schematic shows events at 32–36 h postinoculation (hpi) that characterize the early biotrophic colonization of living host rice cells by *Magnaporthe oryzae*. Infection began when a three-celled spore adhered to the rice leaf surface and germinated. By 4–6 hpi, the germ tube tip started to swell and form a melanized appressorium. Appressorium maturation was completed by 24 hpi and was accompanied by collapse of the spore, the recycling of spore contents into the nascent appressorium, and the build-up of hydrostatic turgor resulting from glycerol accumulation. By 28 hpi, the penetration peg punctured the leaf cuticle to gain access to the underlying epidermal cells. Inside, the peg differentiated into a filamentous primary hypha that then elaborated bulbous invasive hyphae (IH). IH are separated from rice cytoplasm by the host-derived extra-invasive hyphal membrane (EIHM). The EIHM matrix forms between the EIHM and the fungal cell wall. During this early biotrophic growth phase, *M. oryzae* deploys host reactive oxygen species (ROS)-scavenging systems and secreted effectors in order to suppress plant host innate immunity and avoid detection. Effector secretion occurs via two mechanisms: (i) apoplastic effectors are secreted by the conventional endoplasmic reticulum (ER)–Golgi pathway into the EIHM matrix, and (ii) the plant membrane-rich biotrophic interfacial complex (BIC) receives cytoplasmic effectors by an unconventional secretion route before translocation into the host. At 44–48 hpi, IH will constrict and move through plasmodesmata to adjacent living cells. New BICs form at IH tips in newly colonized cells while the EIHM loses integrity in the first infected host cell, which subsequently dies. This process continues until extensive host cell death and the switch to necrotrophy manifests by 120–144 hpi as visible necrotic lesions on the leaf surface. T-bars represent inhibition of host ROS accumulation or fungal biotrophic growth, as indicated.

The filamentous fungus *Magnaporthe oryzae* (anamorph: *Pyricularia oryzae*) causes blast, the most devastating disease of cultivated rice. *M. oryzae* is a haploid ascomycete that grows as branching hyphae and disperses via three-celled, tear-shaped asexual conidiospores produced sympodially on aerial hyphae. Sexual reproduction occurs in the laboratory and is also evident in population studies but has not been observed in the field. *M. oryzae* has a sequenced genome, can be cultured away from the host, and is readily transformable. This facilitates detailed functional genomic studies involving targeted gene deletion by homologous recombination, genome-wide '-omics' investigations, and the use of fluorescent reporters for live-cell imaging. Recent studies – focused on understanding infection-related development, host invasion, and fungal growth in rice cells – have improved our understanding of rice blast fungus biology and the molecular underpinnings of host infection. Such findings may aid the search for durable disease mitigation strategies. For example, knowledge of the proteins involved in host penetration recently led to the discovery of broad-spectrum fungicides targeting this crucial step.

Acknowledgments

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Declaration of Interests

There are no interests to declare.

KEY FACTS:

The *Oryza sativa* (rice)-infecting lineage of *M. oryzae* diversified from the *Setaria italica* (foxtail millet)-infecting lineage around 2500–7500 years ago, during or shortly after rice domestication in East Asia. The fungus was first described by Cavara in 1891.

Infection begins when spores germinate on the leaf surface and, in response to surface cues, form dome-shaped infection cells, called appressoria, at germ tube tips.

Appressoria become pressurized and melanized. When tightly annealed, hydrostatic turgor acts on a penetration peg at the appressorial base, forcing it through the cuticle.

M. oryzae is a hemibiotroph: bulbous, branching invasive hyphae (IH) elaborate from the peg and grow undetected in living host cells for the first 3–4 days of infection (biotrophy), spreading cell-to-cell via plasmodesmata before host cell death (necrotrophy) and the onset of disease symptoms.

To remain undetected during the biotrophic growth phase, *M. oryzae* suppresses host innate immunity using reactive oxygen species (ROS)-scavenging systems and secreted effector proteins.

Sporulation from disease lesions completes the life cycle.

DISEASE FACTS:

Rice blast disease destroys up to 30% of world rice harvests each year, eliminating enough food to feed 60 million people and thus straining global food security. No effective long-term solution exists.

M. oryzae infects leaves, stems, nodes, and panicles of rice.

M. oryzae causes blast on other important grass species, including barley and millets.

A host jump by *M. oryzae* underlies heavy wheat losses in Brazil and neighboring countries, and in Asia.

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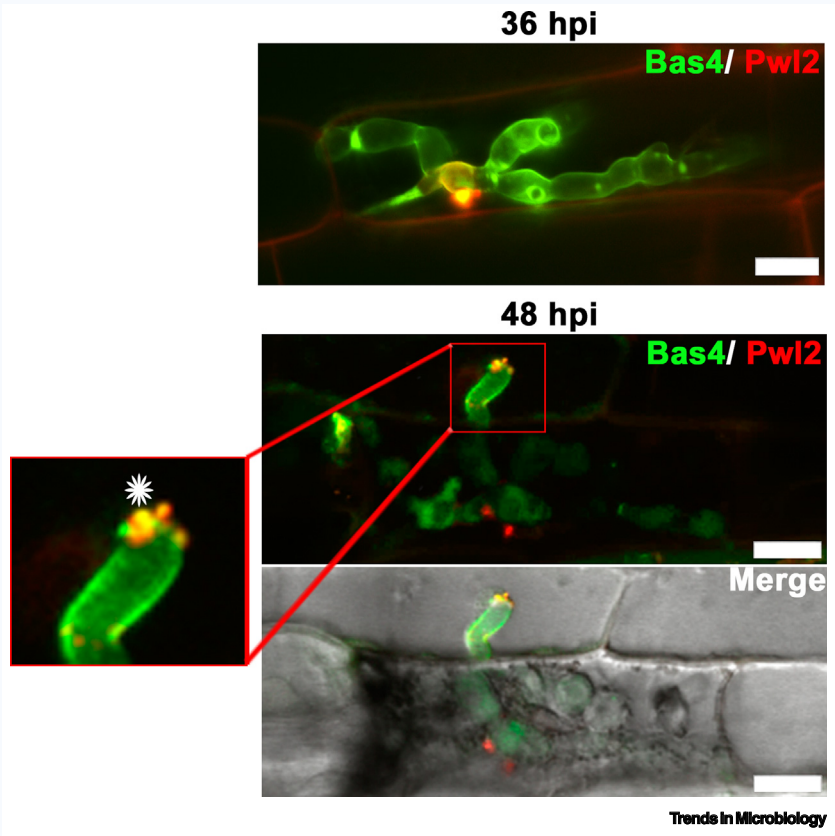

TAXONOMY AND CLASSIFICATION:
KINGDOM: Fungi
PHYLUM: Ascomycota
CLASS: Sordariomycetes
ORDER: Magnaporthales
FAMILY: Pyriculariaceae
GENUS: *Pyricularia*
SPECIES: *Pyricularia oryzae* (syn. *Magnaporthe oryzae*)

Figure 2. Effector Secretion during Biotrophic Growth in Living Rice Cells. Live-cell imaging of the wild-type Guy11 isolate expressing the apoplastic effector Bas4 fused to GFP, which is secreted into the extra-invasive hyphal membrane (EIHM) matrix and outlines invasive hyphae (IH), and the cytoplasmic effector Pwl2 fused to mCherry and a nuclear localization sequence (NLS), which is secreted into the plant membrane-rich biotrophic interfacial complex (BIC) before translocation into rice cells. BICs are composed of vesicles and form outside fungal IH. Abbreviation: hpi, hours postinoculation. Scale bar: 10 μm. Source: Li, Sun, and Wilson, unpublished.

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