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Review

Surface sensing and signaling networks in plant pathogenic fungi

Yanjun Kou [a,*], Naweed I. Naqvi [a,b,*]

[a] Temasek Life Sciences Laboratory, and Department of Biological Sciences, 1 Research Link, National University of Singapore, 117604, Singapore
[b] School of Biological Sciences, Nanyang Technological University, 637551, Singapore

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ABSTRACT

Pathogenic fungi have evolved highly varied and remarkable strategies to invade and infect their plant hosts. Typically, such fungal pathogens utilize highly specialized infection structures, morphologies or cell types produced from conidia or ascospores on the cognate host surfaces to gain entry therein. Such diverse infection strategies require intricate coordination in cell signaling and differentiation in phytopathogenic fungi. Here, we present an overview of our current understanding of cell signaling and infection-associated development that primes host penetration in the top ten plant pathogenic fungi, which utilize specific receptors to sense and respond to different surface cues, such as topographic features, hydrophobicity, hardness, plant lipids, phytohormones, and/or secreted enzymes. Subsequently, diverse signaling components such as G proteins, cyclic AMP/Protein Kinase A and MAP kinases are activated to enable the differentiation of infection structures. Recent studies have also provided fascinating insights into the spatio-temporal dynamics and specialized sequestration and trafficking of signaling moieties required for proper development of infection structures in phytopathogenic fungi. Molecular insight in such infection-related morphogenesis and cell signaling holds promise for identifying novel strategies for intervention of fungal diseases in plants.

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* Corresponding author at: Temasek Life Sciences Laboratory 1 Research Link, 117604, Singapore.
E-mail addresses: yanjun@tll.org.sg (Y. Kou), naweed@tll.org.sg (N.I. Naqvi).

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1. Introduction

Plant pathogenic fungi attack a wide range of crops, causing devastating diseases and significant agricultural losses. Fungal diseases can be controlled through the use of fungicides. However, it is cost intensive and comes with several detrimental effects to the environment. Outbreaks of fungal diseases occur when new races of fungi emerge and spread via susceptible host plants. Highly complex incompatible or compatible interactions between specific fungal strains and host plants govern such susceptibility and resistance phenotypes. Typically, the interaction begins when fungal pathogens establish contact with the host plant surface, and this determines the final outcome following recognition of and response to specific cues. Diverse plant pathogenic fungi employ numerous strategies to perceive the plant surfaces and breach the host cuticle to establish infection. Understanding the molecular mechanisms which control signaling on the plant surface leading to the differentiation of infection-associated structures could provide new ways for fungal disease control.

In this review, we focus on the recent progress in cellular and molecular biology of infection-associated development especially the induction and subsequent spatial and temporal regulation of signaling during the host penetration phase in phytopathogenic fungi. The plant pathogenic fungi included in this review are the rice-blast fungus *Magnaporthe oryzae*, the grey mould fungus *Botrytis cinerea*, the wheat rust fungus *Puccinia* spp., the head-blight fungus *Fusarium graminearum*, vascular wilt fungus *Fusarium oxysporum*, powdery mildew fungus *Blumeria graminis*, the wheat-blotch fungus *Zymoseptoria tritici*, the anthracnose fungus *Colletotrichum* spp., the corn smut fungus *Ustilago maydis*, and the flax rust fungus *Melampsora lini*. These fungi have been rated as the top ten model pathogens in molecular plant pathology [1].

2. Infection structure formation in phytopathogenic fungi

In order to establish successful infection, fungal pathogens have evolved sophisticated and specific infection strategies. However, a number of important processes during the pre-penetration stages are common to all strategies in fungal pathogens. In most cases, these include physical contact with and adhesion to the plant surface, germination, germ tube growth (or hyphal growth) on the plant surface, induction of morphogenetic program, and differentiation of infection structures. For example, the three-celled conidia of *M. oryzae* are deposited by rain splashes onto the surfaces of host plant. Conidia adhere tightly to the plant surface via the secreted extracellular mucilage, and upon hydration send out germ tubes from the terminal cells [2]. When the conidial germ tube senses the appropriate signals, it flattens at its tip, hooks back and differentiates into the specialized infection structure called the appressorium [3]. During maturation, the appressorium develops and utilises high turgor to rupture the host cuticle, and drives a penetration peg into the underlying host cell [2].

Infection structures, which are produced by fungal pathogens on/outside the plant surface, show varied forms and structures (Fig. 1). *M. oryzae and Colletotrichum* initiate invasion of host plants using dome-shaped appressoria [3,4]. The appressorial cell walls are thick, multi-layered and highly melanized. Although the inducing signals differ, the appressoria in *Puccinia* are similar to those in *M. oryzae* and *Colletotrichum* [5–7]. *B. cinerea* forms melanized and aseptate infection structures that lack high turgor and are thus distinct from the appressoria produced by *M. oryzae* or *Colletotrichum* [8]. *F. graminearum* forms foot structures and compound lobate appressoria and infection cushions on plant surfaces [9]. *B. graminis* conidium forms primary germ tube with a short peg to attach to the leaf surface. The secondary germ tube differentiates into an elongated hooked appressorium that penetrates the plant cuticle and epidermal cell [10]. Appressoria of *U. maydis* are slightly swollen tips of the germ tubes formed during the dikaryon phase [11]. Likewise, the urediniospores of *M. lini* generate appressoria with slight swelling of the germ tube apices [12]. Unlike many other plant pathogenic fungi, *F. oxysporum* and *Z. tritici* invade host plants without the use of an appressorium-like structure [1,13].

3. Sensing and responding to the physico-chemical cues from the host surface

3.1. Topographic signals

Some phytopathogenic fungi, such as *Puccinia*, *Z. tritici*, and *M. lini*, invade leaves through stomata [1,5,6,12]. The germ tubes of *Puccinia triticina* orient along the long axis of the epidermal cells to search for stomata. Such germ tubes initiate pathogenic growth and form appressoria upon contact with stomata [5]. *Z. tritici* penetrates wheat leaves through stomata by hyphal extension [1]. *M. lini* generates swollen appressoria on the stomata and penetrates between the guard cells [12,14]. The germ tubes or hyphae of these fungi distinguish the ridges of the guard cells and recognize the specific surface topology of stomata to induce the infection structures. However, such topographic signals may not be sufficient to induce infection structures in some fungal pathogens. The germ tubes of *P. triticina* are unable to recognize the stomata from non-host plants [15].

3.2. Surface rigidity or hardness

Surface hardness is required for initiation of appressoria in *M. oryzae* and *Colletotrichum* [16]. Conidial germ tubes of *M. oryzae* fail to form appressoria on soft substrates such as petroleum jelly or polymerized agar. However, the dried agar surface induces appressoria which is directly related to the hardness of agar surface. The hardness signal is sensed within 2 h post germination, and the critical hardness necessary for appressorium initiation has been estimated in *M. oryzae* [16]. *Colletotrichum trifolii* is able to develop appressoria on hard surfaces such as the plant cuticle, glass, and plastic, but not on soft surfaces such as mineral oil [17]. Contact with hard surfaces is required for appressoria formation in *Colletotrichum gloeosporioides* [18].

3.3. Hydrophobicity

Several reports suggest that surface hydrophobicity is an important stimulus for induction of infection structures in plant pathogenic fungi including *M. oryzae* [16,19]. Germination success and differentiation in *B. graminis* conidia is related to surface hydrophobicity [20]. Hydrophobicity induces filaments and appressoria in *U. maydis*. However, hydrophobicity combined with other chemical signals is comparatively more efficient [11,21]. Hydrophobicity breaks dormancy and promotes spore attachment and germination in *C. graminicola* [22]. The response to hydrophobicity of the plant surface is a prerequisite for adhesion of conidia in *B. cinerea* [23].

3.4. Plant surface waxes and cutin

Plant-derived lipids such as wax and cutin are known to induce appressorial differentiation in pathogenic fungi. The wax deposits represent the first barrier *per se* during fungal attachment to plant cuticle. Fungal spores secrete cutinases (and/or other cell wall degrading enzymes) during germination and release cutin/cell wall monomers [24]. Cutin monomers and cuticular waxes are critical inducers of appressorium formation in *M. oryzae* and several

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