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Building the interaction interfaces: host responses upon infection with microorganisms

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Research fields of plant symbiosis and plant immunity were relatively ignorant with each other until a little while ago. Recently, however, increasing intercommunications between those two fields have begun to provide novel aspects and knowledge for understanding relationships between plants and microorganisms. Here, we review recent reports on plant–microbe interactions, focusing on the infection processes, in order to elucidate plant cellular responses that are triggered by both symbionts and pathogens. Highlighting the core elements of host responses over biotic interactions will provide insights into general mechanisms of plant–microbe interactions.

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Introduction

In the natural environment, plants are consistently exposed to a range of microorganisms. In order to dissect and investigate interactions among them, microorganisms are often categorized into several groups such as pathogens, commensal microbes or symbionts, based on consequences of their interactions. In the long evolution history, microorganisms have obtained 'keys' to open 'gates' of host plants. For example, by producing and secreting lipochitooligosaccharides (LCOs), some microorganisms let host plants being ready for starting symbiotic interactions: preparing 'guides' for the internalization of symbionts and, in the case of root nodule symbiosis (RNS), initiating active proliferation of cortical cells [1,2]. Finally, symbionts reach to the place where mutual symbiosis occurs and become so-called symbiosomes in order to exchange nutrition [1,2]. On the other hand, pathogens invade and sneak into hosts, or physically breach host 'gates' [3,4]. After breaking into host tissues, biotrophic fungi form feeding structures [3]. Although several features are unique to each interaction, principles of some host processes triggered by microorganisms seem to be conserved among interactions. Besides, both symbiotic and biotrophic interactions form interaction interfaces where they exchange signals and nutrition, indicating the involvement of microorganisms in the growth of host plants, while outcomes depend on the degree and balance of the 'exchange'.

Although the pathogenicity of necrotrophs is obviously different from other interactions (they kill their host cells and proliferate on nutrients from dead tissues) [5], as just mentioned, host responses triggered by biotrophs and symbionts seem to, at least partially, be shared. This opens up a question; are there any central mechanisms by which plants respond to microorganisms regardless of whether the interaction is beneficial or detrimental? Here, we review recent progresses in the plant–microbe interactions in the context of infection processes of microorganisms (Box 1) to elucidate host processes underlying symbiotic and biotrophic interactions.

Interaction signals and attachment

Before the physical contact between plants and microorganisms, they secrete a number of signal molecules to their environments, which can be perceived by either plants or microorganisms and trigger certain cellular programs as they 'talk' to each other. Arbuscular mycorrhizal fungi (AMF) and rhizobia secrete LCOs (Myc-LCOs and NFs, respectively) that act as 'keys' for mutual symbioses (Figure 1a) [6]. Besides, some signals flitting among plants and microbes are involved in host growth, development and innate immunity. For instance, strigolactones, initiators of hyphal branching of AMF, modulate shoot branching and root architecture [6,7]. Rhizobial NFs whose production is induced by host-derived flavonoids exhibit immunosuppressive effects [8••] (see Modulation of host immunity). Bacterial *N*-acetyl homoserine lactones (AHLs) that have been found as bacterial quorum sensing (QS) signals, contribute to alteration of root length and to priming plant cell-wall strengthening [9•,10]. On one hand, several plant-derived signals have been known to initiate developmental transitions of microbes. Plant epicuticle waxes and cutin monomers act as cues for differentiating hyphae into specialized structures for invasion [11,12,13•,14•,15–17]. A mutation in *RAM2* (*Reduced Arbuscular Mycorrhization2*), whose product is involved in the production of cutin monomers, exhibits hyphopodium impairment, defects in oomycete

Box 1 Infection processes of plant symbionts and pathogens

The infection of microorganisms to their hosts generally consists of following processes: signal perception/exchange, physical contact/attachment, internalization and establishment of infection.

Arbuscular mycorrhizal fungi (AMF) start hyphal branching upon perception of strigolactones, and AMF-derived Myc-LCOs activate common symbiotic pathway and initiate arbuscular mycorrhiza (AM) (Figure 1a) [2,6]. A branched hypha is then differentiated into hyphopodium upon plant-derived cutin signals [12,13,14•]. AMF internalize into host tissue by the guidance of prepenetration apparatus (PPA), and form arbuscules in inner cortical cells (Figure 1d) [2].

In RNS, host plants and cognate rhizobia exchange signals: flavonoids and flavonoid-induced Nod factors (NFs) [1]. Rhizobia attach to the tip of root hairs and internalize through infection threads (ITs) (Figure 1c) following root hair curling and pre-infection thread (PIT) formation [1]. Simultaneously, NFs activate downstream signaling pathways and initiate the formation of nodule primordium where rhizobia are released, which eventually becomes the nodule [1,6].

Filamentous pathogens are also able to respond to plant-derived signals [11,15,16]. Some of biotrophic fungi form appressorium on the cuticle layer of host plant for breaching plant surface layers, and some enter from stomata [3,15]. Then, they form feeding structures, invasive hyphae or haustorium, in host cells [3].

Plant-derived volatiles can act as attractants for soil microorganisms including pathogenic bacteria [70]. They attach to host surface predominantly by forming biofilms and internalize via natural openings and wounds [4]. Symptom varies depending on the pathogenicity of bacteria.

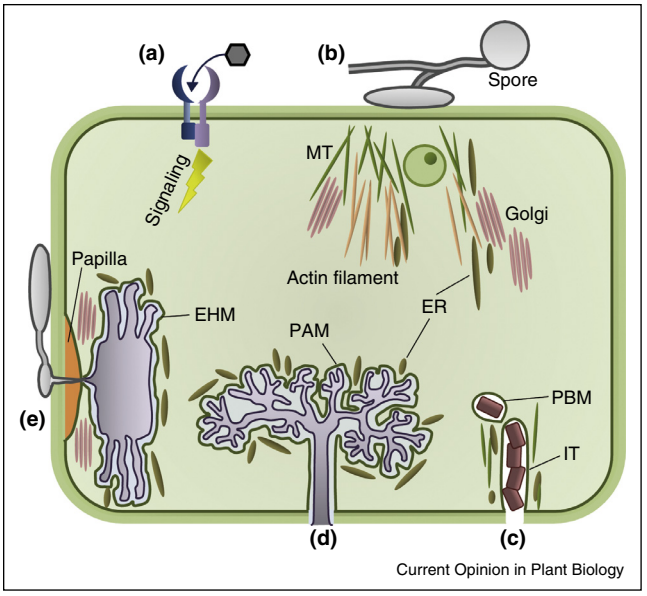
colonization and seed coat-defect phenotypes in *Medicago truncatula* [13•,14•].

Instead of differentiation, bacteria form specialized surface structure called biofilm for attachment to host surface. Biofilm is mainly composed by bacterial surface appendages and extracellular compounds [18–22]. Plant associated bacteria including pathogens and symbionts regulate the formation and dispersal of biofilm by secreting QS signals, and plants hijack this system and confuse bacteria by releasing QS mimics to their environments [23].

Modulation of host immunity

To cope with potential pathogens, plants have evolved reception systems by which they sense signs of danger. Several substances that are derived from microorganisms or from plants upon microbe association can be recognized by plants as microbe associated molecular patterns (MAMPs) via a variety of pattern recognition receptors (PRRs), resulting in activation of MAMPs triggered immunity (MTI) (Figure 1a). Following the perception of MAMPs and the subsequent rapid calcium influx into cytoplasm, plants activate immune processes such as generation of reactive oxygen species (ROS), salicylic acid production and stomatal closure [24]. Cell-wall strengthening is initiated by the flg22 (a highly conserved N-terminal epitope of bacterial flagellins)-triggered ROS generation via a callose accumulation [24].

Figure 1



The schematic diagram of plant cellular responses observed in both symbiotic and pathogenic interactions. **(a)** Microorganism-derived signals (a hexagon in gray) are perceived by their cognate plant receptors (structures on the plasma membrane), which in turn activate downstream signal pathways. **(b)** Plant cuticle monomers induce differentiation of a fungal hypha into attachment structure (appressorium or hyphopodium). Upon contact with microorganisms, cytoplasmic aggregation consisting of focusing of MTs (filaments in green), rearrangement of actin microfilaments (filaments in orange), rapid accumulation of ER (thick filaments in brown) and aggregations of Golgi bodies at the contact site occurs. In symbiotic interactions, cytoplasmic bridge-like structures called PIT (in the case of RNS) or PPA (in the case of AM) are formed before internalization of symbionts. Similar cytoplasmic rearrangement occurs during EHM and EIHM formation in plant–pathogen interactions. **(c)** Rhizobial cells (rectangles in red) propagated in ITs are released into nodule primordium cell as infection droplets engulfed by PBM. **(d)** AMF form arbuscules in root cortical cells, which are engulfed by PAM. **(e)** An obligate parasite, *B. graminis* f. sp. *hordei*, forms haustorium as a terminal feeding structure. The haustorium is engulfed by EHM. Golgi stacks are observed around the neck. These membranes that enwrap microorganisms are all derived from host plants. Accumulation of ER around the membrane seems to be due to the extensive elongation and generation of membrane structures. MTs: microtubules, ER: endoplasmic reticulum, PIT: pre-infection thread, PPA: pre-penetration apparatus, EHM: extrahaustrial membrane, EIHM: extrainvasive hyphal membrane, PBM: peribacteroid membrane, PAM: periarbuscular membrane.

To circumvent these host immune responses, microbes modulate host immunity by secreting effectors. Secreted proteins SP7 and MiSSP7 (Mycorrhiza-induced Small Secreted Protein7) of *Rhizophagus irregularis* and *Laccaria bicolor*, respectively, reduce host defense responses, and for the latter, by protecting JAZ6 (Jasmonate-Zim-domain protein6) that acts as a negative regulator of jasmonic acid-induced gene regulation [25,26••].