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INTERACTIONS BETWEEN PLANTS AND PATHOGEN MICROORGANISMS: FROM STRESS PERCEPTION TO DEFENSE RESPONSES

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INTRODUCTION

Phytopathogenic microorganisms (fungi, oomycetes and bacteria) cause each year a yield loss of 10 - 15 % in the world. Around 70 % of plant diseases are related to fungi, these microorganisms representing with oomycetes more than 100 000 phytopathogenic species whereas there are approximately 100 species of phytopathogenic bacteria.Phytopathogenic species can have three modes of trophy depending on the species[1]:

- they can grow in living plant tissues (biotrophy) such as *Ustilago*, *Puccinia* and *Agrobacterium* species,

- they can lyse plant cells to obtain nutrients and thus grow in dead tissues such as *Botrytis* or *Fusarium* (necrotrophy),

- they can be first biotrophic and then switch to a necrotrophic mode (hemibiotrophy) such as *Phytophtora* and most of phytopathogenic bacteria.

Phytopathogens can infect plant organs after penetration by stomata for leaves (that is the most frequent mode for bacteria), after wounding or after adhesion on an aboveground or an underground organ. The adhesion requires the secretion of lytic enzymes (lipases, cutinases) by phytopathogen to remove cuticle and the secretion of adhesive substances containing polysaccharides, lipids and proteins such as hydrophobins [2].

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1. Pathogen recognition

Plants can develop defense mechanisms against most of these pathogens. The induction of defenses begins at the first step of infection when plant recognizes the pathogen in a specific or non-specific manner. Non-specific recognition corresponds to the binding of well conserved domains of pathogenic proteins to transmembrane of intracellular plant receptors. The pathogenic proteins are common in many microorganisms and are called PAMP proteins for pathogen-associated molecular patterns[3-6]. They are secreted or not by pathogens at the beginning of infection. In bacteria, the main PAMP proteins are harpins (secreted proteins leading to a K^+ efflux from plant cells) and flagellins (structural proteins of flagellate bacteria). In fungi and oomycetes, main PAMP proteins are transglutaminases (enzymes catalyzing the binding of amino acids with glutamine) and elicitins (proteins secreted to form a complex with plant sterols and use them as nutrients). In addition, other PAMP proteins called endopolygalacturonases (PGAses) are produced by fungi as well as oomycetes and bacteria and are secreted for cell wall degradation (Fig. 1).

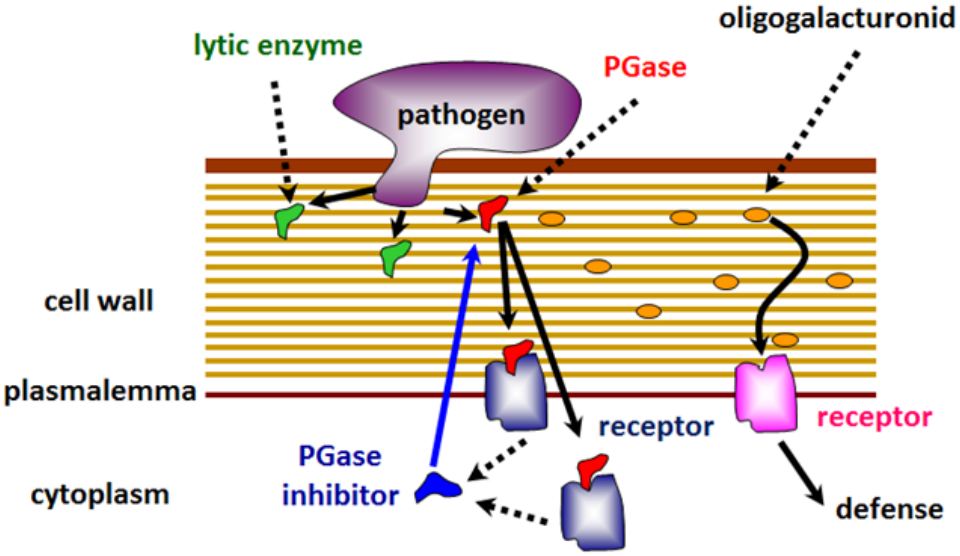

Figure 1. Non-specific recognition of phytopathogenic PGases by plant cell. When a pathogen attacks a plant cell, it liberates many lytic enzymes including PGAses. PGases are recognized by transmembrane or cytosolic plant receptors that lead to a secretion of PGAse inhibitors to the cell wall. Inhibitors slow down PGase activity and oligogalacturonids generated by PGAses are then recognized by other plant receptors as an attack by a pathogen.

Specific recognition ("gene for gene theory") corresponds to the recognition of a specific pathogenic protein (Avr protein) by a specific plant receptor (R protein)[7]. Each plant species has several hundreds of *R* genes: more than 400 in *Arabidopsis thaliana* and even 2-3 more in crop species. This helps plants to identify several hundreds of pathogens and to rapidly induce defenses. In contrast, phytopathogens have only a few dozen *Avr*genes (up to 40 for several bacteria). Recognition can be direct between Avr and R proteins or can be indirect when an Avr protein is recognized by a plant guard protein (GP) associated with a R protein (figure 2)[8].

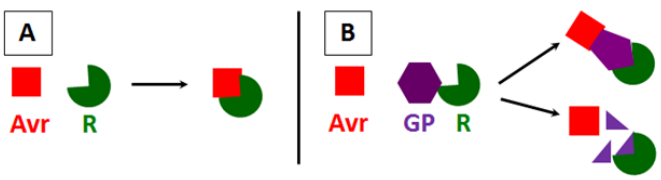

Figure 2. Specific recognition of phytopathogenic Avr protein by plant R receptor. A) The R receptor binds directly a domain of the Avr protein that induces a signaling pathway. B) The GP binds a domain of the Avr protein and is modified (modification of conformation or by degradation). The modification of GP is then recognized by the R receptor that induces a signaling pathway.

2. Hypersensitive response (HR)

After recognition, plant response comprises a succession of three steps: the hypersensitive response (HR), the local acquired resistance (LAR), and then the systemic acquired resistance (SAR) (Fig. 3).

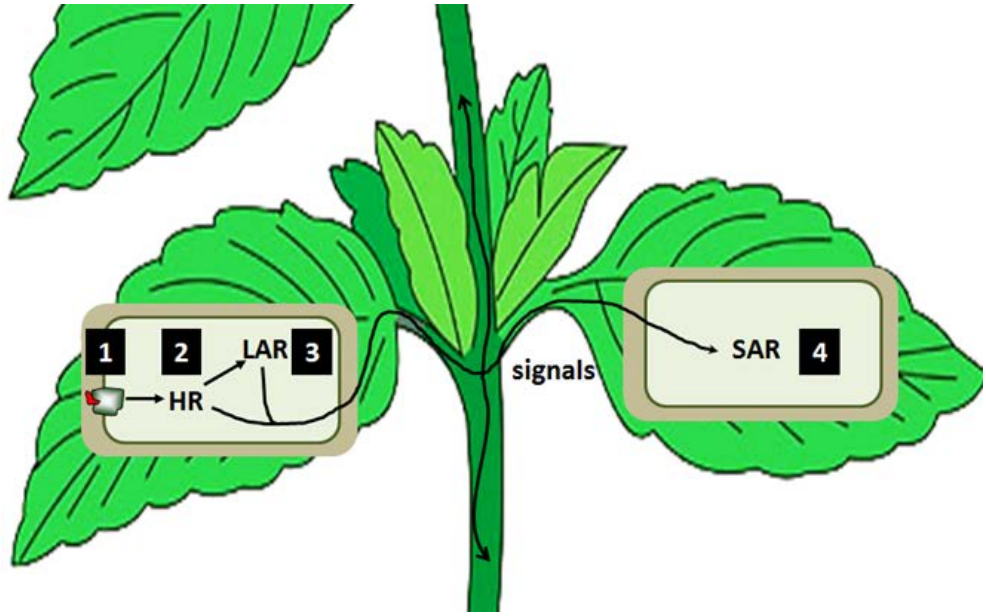

Figure 3. Chronology of plant infection and defenses. After recognition of a pathogenic PAMP or Avr protein by a plant receptor (1), plant develops HR (2) characterized by a necrosis around the infection site. Signals are sentaround the HR to induce LAR around the necrosis zone (3). Then, HR and LAR produce messengers transported to the other organs where the SAR can take place (4).

HR corresponds to a plant cell death around the infection site to stop the infection or at least to delay it. This necrosis takes place after specific or non-specific recognition: plant receptors activate G proteins that activate or inhibit ion canals and channels [2]. Therefore, calcium strongly accumulates in the cytosol (with a concomitant H^+ and K^+ efflux) and activates a phospholipase C [9]. This enzyme converts phosphatidylinositol 4,5-bisphosphate into inositol 1,4,5-trisphosphate (IP3) and diacyl glycerol that is then converted into phosphatidic acid (AP)[2]. IP3 stimulates the release of vacuolar calcium and the subsequent calcium accumulation in cytosol activates several kinases. AP activates other kinases and at the end of these pathways, kinases activate transcription factors to induce the expression of genes [5, 10].

Expressed genes encode enzymes involved in callose synthesis to form a papilla under the site of infection. This physical barrier delays pathogen propagation and allows plant cell to increase the production of reactive oxygen species (ROS) and nitric oxide (NO) *via* the stimulation of NADPH oxidaseand nitric oxide synthase [11, 12]. Consequently, the production of this oxidative burst causes a death of plant cell as well as pathogen. This necrosis is reinforced by the accumulation of terpene- and phyenylpropanoid-derived molecules called phytoalexins which synthesis is stimulated by ROS and NO [5]. Each plant species produces a panel of phytoalexins species- (or family) specific with one or two main phytoalexin(s) per species such as resveratrol in grapevine, pisatin in pea, and rishitin in potato [13]. In addition, during HR constitutive phytoanticipins stored in the vacuole as glycosylated inactive precursors are cleaved and can thus exhibit an anti-pathogenic activity [5].

Phytoanticipins are saponins (derived from steroids or triterpenes), cyanogenic glycosides (releasing HCN after cleavage) and glucosinolates (giving thiocyanate, isothiocyanate or nitrile depending on the pH after cleavage).

3. Local acquired resistance (LAR)

When HR takes place, ROS and NO diffuse around the necrotic zone but their concentration is lower and in this case, ROS and NO become messengers to activate other transcription factors. Expressed genes are involved in several pathways including the trans-cinnamic acid pathway leading to LAR. The main characteristics of LAR are [14, 15]:

1) A cell wall strengthening: cell walls accumulate various compounds to limit a subsequent attack by the pathogen. These compounds are callose, lignins, coumarins (that bind hemicelluloses) and structural proteins such as hydroxyproline-rich glycoproteins.

2) An increase in anti-oxidative enzyme activity: plants stimulate superoxide dismutases, catalases, glutathione-S-transferases or peroxidases to decrease the concentration of ROS and NO up to a non-toxic level in plant cells.

3) An accumulation of phenolic compounds: plants synthesize secondary metabolites (phytoalexins, anthocyanins, condensed tannins) stored at vacuolar level. If the pathogen counteracts the HR or if another pathogen attacks, plant cells will thus already have defense compounds to limit the infection. These compounds are not specific to pathogens as various kinases activated during HR induce pathways common with other stresses. For example, anthocyanins are more often involved as a response to oxidative stress (such as UV excess) and tannins to phytophagous animals.

4) The synthesis of pathogenesis related proteins (PR proteins): these proteins are produced during LAR and SAR and will be described in the next part.

4. Systemic acquired resistance (SAR)

Kinases as well as ROS and NO activate genes encoding enzymes involved in the synthesis of messengers to induce LAR and SAR [16]. These messengers are salicylic acid (SA) generally against biotrophic pathogens and jasmonic acid (JA) and ethylene (ET) more often synthesized as a response to necrotrophic pathogens [5, 17].

Chaperone proteins of the “lipid transfer protein” family transport SA and JA *via* phloem sap to the living tissues of the infected organ and to the other organs of the plant [18]. The transport of ET is not completely clarified but ET is probably transported *via* the apoplastic pathway.

The messengers stimulate the synthesis of PR proteins for LAR and SAR [17, 18]. These proteins allow plant to resist to a subsequent attack by the pathogen or by another pathogen as PR proteins are not specific to one pathogenic species. These proteins are divided into 17 classes but a plant species doesn’t produce all PR proteins and PR proteins can be different between LAR and SAR in the same plant species [19]. PR proteins can be divided in four main functions:

1) weakening of the membrane by blocking calcium channel or by creating pore in the membrane of the pathogen.

2) alteration of the cell wall of pathogens as some PR proteins are endo β-(1,3)-glucanases and endochitinases.

3) modification at protein level: several PR proteins are inhibitors of pathogenic proteases or ribosomal RNA (28 S) whereas other PR proteins are proteases against pathogenic proteins.

4) protection of plant cell wall: some PR proteins are cell wall peroxidases helping to increase bounds between lignins in the cell wall. Other PR proteins are oxalate oxidases with a function of oxalate hydrolysis. Indeed, pathogens can release oxalate during infection, a molecule known to remove calcium, leading to a reduction of bounds between cell wall homogalacturonans and thus leading to a weakening of the cell wall.

CONCLUSION

As each plant species can recognize (specifically or not specifically) many phytopathogens, the development of a disease is an exception compared to the number of potential phytopathogens. However, plant defenses are not definitive as pathogens adapt and can counteract several steps during plant - pathogen interactions. Indeed, it was shown for example that several pathogens can:

- modify their Avr proteins to be no longer recognized by the plant,
- increase their anti-oxidative enzymes to reduce the concentration of ROS and NO during the HR (and thus to limit plant and pathogen cell death),
- -synthesize inhibitors of phytoalexins to reduce the intensity of plant defenses during HR and/or LAR,
- synthesize inhibitors of PR proteins.

Nevertheless, plants can adapt in return their metabolism and sometimes counteract the new strategies of phytopathogens. For example, several plant species modified R proteins or guard proteins (associated to R proteins) to recognize the new modifications of Avr proteins. Moreover, some plant species synthesize inhibitors of phytoalexin inhibitors to maintain the synthesis of these secondary metabolites. The interactions between plants and phytopathogenic microorganisms are thus a constant competition for life.

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