Plant pathogen defense: Signalling, resistance and cell death

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Akademisk avhandling för filosofie doktorsexamen i Naturvetenskap med inriktning Biologi, som med tillstånd från Naturvetenskapliga fakulteten kommer att offentligt försvaras fredagen den 24:e april 2015 kl. 10.00 i Hörsalen, Institutionen för biologi och miljövetenskap, Carl Skottsbergs gata 22B, Göteborg.

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ISBN: 978-91-85529-78-0
Tryck: Kompendiet
Digital source of thesis: http://hdl.handle.net/2077/38369

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ABSTRACT

Pathogenic microorganisms are present everywhere in nature and infect both animals and plants. Phytopathogenic microorganisms cause diseases on plants, and are responsible for crop loss amounting in the order billions of dollars annually. Plants have however co-evolved with these organisms and have consecutively been forced to develop mechanisms that prevent disease. The plant immune system unlike that of animals lack adaptive cells and rely on the innate immunity of each plant cell. There is however no doubt in the effectiveness of the plant immune system as most plants are healthy most of the time.

The plant immune system consists of two main tiers of defense responses; the MAMP triggered immunity (MTI) and the Effector triggered immunity (ETI). MTI is triggered by recognition of microbe associated molecular patterns MAMPs. MTI strengthens the cell by producing antimicrobial substances, proteins and by fortifying the cell wall. This stops the majority of non-adapted microbes. A subset of microbes have adapted to these measures and evolved effector proteins that subdue the MTI responses. Again, plants have responded, by evolving resistance (R) proteins that recognize effector activity and mount the swift responses that are ETI. The plant responses during ETI are commonly termed the hypersensitive response (HR) and culminate in programmed cell death of the infected and sometimes surrounding cells.

The thesis has approached the plant disease resistance response in four ways. The first focused on improving methods for quantifying the programmed cell death response during ETI (**Paper I**) and lipid analysis by chromatography (**Paper II**). These methods are then used in the following pappers. The second part focused on signalling during the HR. Signalling on gene regulation level (**Paper III**) and various parts of lipid metabolism (**Paper IV, V** and **VI**) during the HR was pursued. The main results from these studies include the high redundancy identified among *Arabidopsis thaliana* phospholipase D isoforms in producing the lipid phosphatidic acid, the identification and initial characterization of the enzyme (AGAP1) that is responsible for producing head group acylation of lipids in *A. thaliana* and the reported involvement of a chloroplast localized 13-lipoxygenase in initiating the HR related programmed cell death in *A. thaliana.*

The third part of the thesis proposes a role in the HR in *A.thaliana* for two reactive molecules; indole acetonitrile (**Paper VII**) and sulforaphane (**Paper VIII**). Both compounds induce cell death when infiltrated into leaves and studies using mutants suggest that absence of these compounds result in a reduced cell death response. A redox related mechanism for these compounds is suggested. The fourth and final part of the thesis aimed to investigate if novel components could be identified in post penetration response against powdery mildew funguses. Much less is known on the relative dependence of MTI and ETI of this system, the results from **Paper IX** suggest that besides the known involvement of the protein EDS1, additional components are present.

In conclusion, this thesis contributes with insight into different aspects of how lipid-, redox- and hormone signalling contributes to resistance and cell death in plants.

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