Fungal Effectors and Plant Susceptibility

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Abstract

Plant fungal pathogens are primary causal agents of many crop diseases of economic significance and pose serious threats to crop production and yield worldwide. Effector proteins are defined as small secreted proteins containing ≤300 amino acids. Many of these proteins are cysteine rich, and their tertiary structures are stabilized by disulfide bridges. The effectors are important virulence determinants of pathogenic fungi and play important role in successful pathogenesis, predominantly by avoiding host-surveillance system. As plant-pathogen interactions evolve, plants are selected for an incompatible (resistant) interaction and parasites are selected for a compatible (susceptible) interaction. The underlying principle for this antagonistic coevolution is based on the gene-for-gene model, arms race model, trench warfare model, zig-zag model. Effector evolution is a trade-off between escaping from detection and optimizing the virulence function. Compared with the avirulence function of effectors, a molecular understanding of the virulence and symbiosis-promoting activities of fungal effectors is still in its infancy. It is conceivable that fungal effectors may also be used to combat other microbes rather than being exclusively addressed toward plant targets.

Introduction

Plant fungal pathogens are primary causal agents of many crop diseases of economic significance and pose serious threats to crop production and yield worldwide. Effector proteins are defined as small secreted proteins containing ≤ 300 amino acids. Many of these proteins are cysteine rich, and their tertiary structures are stabilized by disulfide bridges. The effectors are important virulence determinants of pathogenic fungi and play important role in successful pathogenesis, predominantly by avoiding host-surveillance system.

Fungal plant pathogens are of huge economic importance because they threaten the production of crops already growing in the field and can cause post-harvest diseases. Estimates suggest that approximately 10 per cent of agricultural production is lost annually owing to fungal infection. With the growing consequences of climate change, these losses are expected to increase. To combat fungal infections, farmers rely on resistant crop varieties or multiple fungicide treatments, which can have negative effects on the environment. In addition, current agricultural practices that rely largely on planting one crop genotype on huge areas of land promote the selection of fungal strains that overcome genetic resistance quickly, necessitating the constant development and introduction of new resistance traits into crops by breeding approaches.

Interestingly, several targeted TFs are connected to plant growth and development. For instance, *P. infestans* AVR2 upregulates potato TF StCHL1, a positive regulator of the brassinosteroid (BR) signalling pathway. AVR2 expression in potato plants alters growth morphology, induces BR-marker gene expression and enhances susceptibility to *P. infestans* (Turnbull *et al*., 2017).

Depending upon the localization within the host, fungal effectors are broadly categorized into apoplastic and cytoplasmic effectors. Apoplastic effectors are secreted into the intercellular spaces (apoplast) of host tissue, while cytoplasmic effectors get directly translocated into the host cytoplasm (Rocafort *et al.,* 2020).

Knowledge about functions and double-edged role of fungal effectors of biotrophic, hemibiotrophic and necrotrophic pathogens, are crucial for understanding their mode of colonization and role in virulence/avirulence functions. This helps in effective management of fungal diseases in crop plants (Pradhan *et al.,* 2020).

Intracellular effectors of oomycete pathogens, mostly proteins but also small ribonucleic acids, are delivered by the pathogens into the host cell cytoplasm where they interfere with normal plant physiology. The diverse host processes emerging as 'victims' of these 'specialised bullets' include gene transcription and RNA-mediated silencing, cell death, protein stability, protein secretion and autophagy. Susceptibility factors are mostly negative regulators of immunity, but some seem necessary to sustain or promote pathogen colonization (Fabro, 2022).

Plant immune system: zig zag model

All fungi that colonize plants are recognized by the plant immune system and elicit host defenses. These initial defense responses are triggered by invariant molecular patterns exposed by the microbe, referred to as pathogen-associated molecular patterns (PAMPs) and microbe-associated molecular patterns (MAMPs). In fungi, the cell wall component chitin functions as such a PAMP: After fungal contact, chitin oligomers are released from the fungal cell wall through plant chitinases. PAMPs are recognized through membrane localized pattern recognition receptors (PRRs), which trigger a first line of defense reactions called PAMP-triggered immunity (PTI) (Presti *et al.*, 2015). PRR signaling can also be triggered by host-derived damage-associated molecular patterns (DAMPs).

Double facet functions of AVR4 effector

Knowledge about functions and double-edged role of fungal effectors of biotrophic, hemibiotrophic and necrotrophic pathogens, are crucial for understanding their mode of colonization and role in virulence/avirulence functions. This helps in effective management of fungal diseases in crop plants. Phyto-pathogenic fungi can cause huge damage to crop production. During millions of years of coexistence, fungi have evolved diverse life-style to obtain nutrients from the host and to colonize upon them. They deploy various proteinaceous as well as non-proteinaceous secreted molecules commonly referred as effectors to sabotage host machinery during the infection process.

Conclusion

Fungal effectors have evolved in variety and capability to target multiple plant proteins and mRNAs located in different compartments of the plant cell. Effector targets can influence plant defence either directly or indirectly, as well as positively or negatively. While some effectors alter main defence components (*e.g.,* ACS, NPR1), others disturb the activity of proteins that appear to be collaterally connected to defence execution, such as those participating in transcriptional regulation of growth/development or in protein degradation /secretion. A more subtle activity of effectors involves exploiting susceptibility factors (SFs), which are mostly negative regulators of immunity. SFs also include a subset of host proteins participating in processes (autophagy, RNAi, cell death, specific transcription) whose selective activation is required by the pathogen to maintain compatibility.

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