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Phytophthora sojae effectors orchestrate warfare with host immunity

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Phytophthora sojae is one of the most damaging plant pathogens of soybean. To aid establishment of a compatible interaction with its host, *P. sojae* deploys many secreted effectors. These effectors act either in the apoplastic space to cope with hostile conditions or inside of host cells to reprogram host physiology favoring pathogen growth. Effectors have been used as molecular probes, which revealed in *Phytophthora* that effectors execute their virulence function via manipulating host targets. In addition, recent studies have discovered 'pseudo-effectors' in *Phytophthora* that act as decoys to shield virulence effectors from host defense, a new paradigm in plant-pathogen interactions.

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Introduction

Root and stem rot caused by the oomycete pathogen *Phytophthora sojae* is one of the most destructive diseases of soybean [1]. *P. sojae*, together with other oomycete pathogens, are members of the kingdom Stramenopila that are evolutionarily distant from fungi. As one of the first sequenced oomycete pathogens [2], *P. sojae* has developed as a model species due to the rich collection of genetic and genomic toolkits. In particular, the recently developed CRISPR/Cas9-mediated genome editing technique in *P. sojae* [3•] allows high-throughput functional genomic research.

P. sojae encodes hundreds of secreted effectors which presumably act as weapons to attack its host. Studies on the action of these effectors increase our understanding of *Phytophthora* pathogenesis and would help guide the development of integrated disease control strategies. In this review, we describe the intricacies of effectors employed by *P. sojae* to suppress plant immunity with emphasis on how effectors manipulate host virulence targets (Figure 1) and discuss research topics that require further attention.

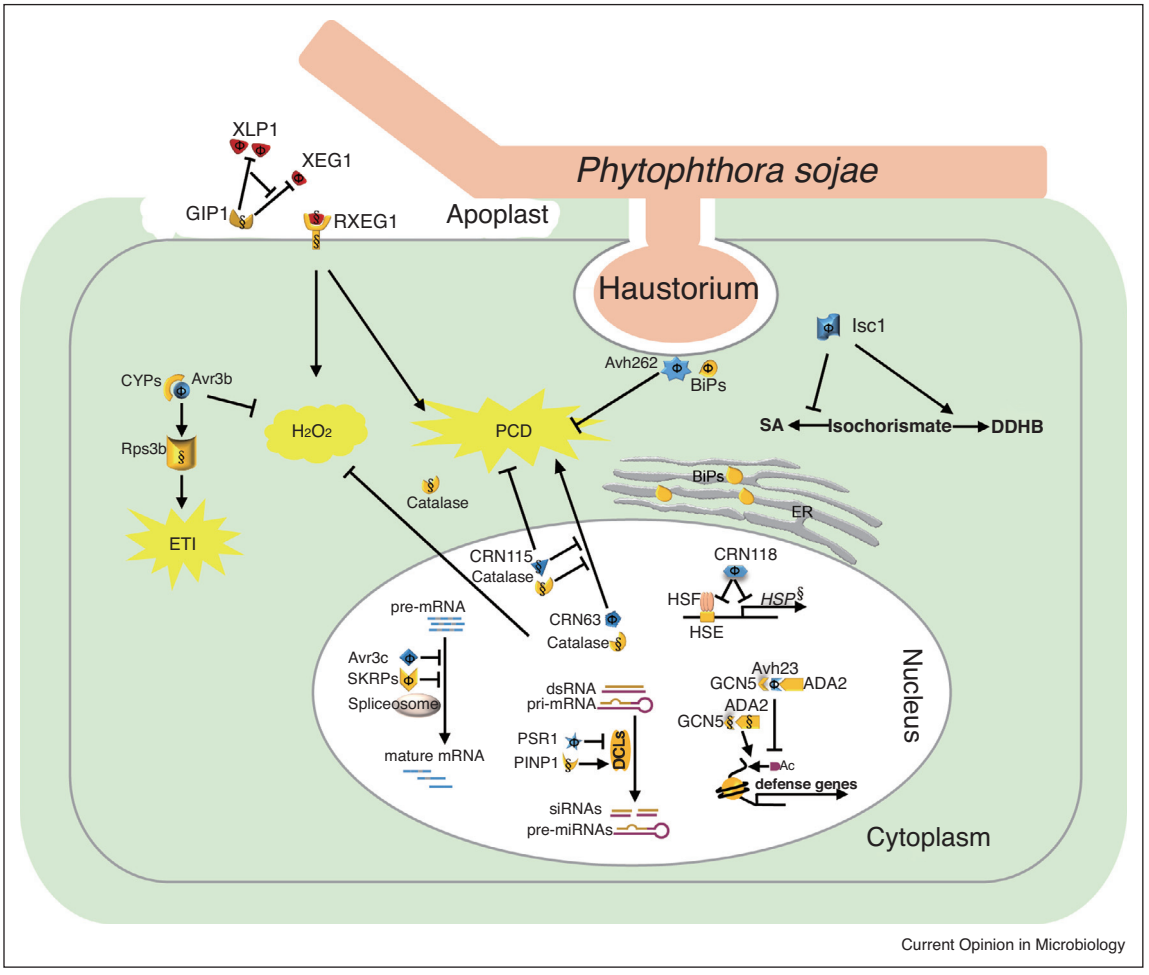
Action in the extracellular matrix: apoplastic effectors

Apoplastic effectors have emerged as important players in plant-pathogen interactions. These effectors bind either host derived apoplastic proteins or membrane-localized pattern recognition receptors (PRRs), acting as either virulence factors to suppress plant defense or as molecular patterns to provoke plant immunity [4]. The necrosis- and ethylene-inducing-like proteins (NLPs) constitute a group of conserved apoplastic effectors [5,6]. The *P. sojae* genome contains 70 potential NLP genes with 33 being predicted to encode authentic NLPs [7]. An assay of 19 representative NLP genes showed that only eight triggered cell death in *Nicotiana benthamiana*, but how these NLPs contribute to *P. sojae* infection remains unclear. NLPs of *Phytophthora parasitica* and *Pythium aphanidermatum* were recently found binding to series A glycosylinositol phosphorylceramide (GIPC) sphingolipids to disrupt membrane integrity [8••]. This binding determines NLP cytotoxicity in plants but has not yet been demonstrated responsible for NLP-mediated virulence in pathogen infection. Although no virulence target of *P. sojae* NLPs has been discovered thus far, NLPs contain a conserved peptide of 20 amino acids designated nlp20 which elicits immune responses in Arabidopsis [5] by binding to the leucine-rich repeat (LRR) receptor-like protein RLP23 [9•]. In this case, recognition of NLPs by RLP23 may partially account for the non-host resistance of Arabidopsis against *P. sojae*.

Orchestrating an evolutionary struggle in the host apoplast

The constant arms race between plants and pathogens has driven the evolution of novel virulence strategies. The xyloglucan-specific endoglucanase 1 (PsXEG1) is the best characterized apoplastic effector in *P. sojae* [10•,11••]. PsXEG1 belongs to the glycoside hydrolase 12 (GH12) family which is common across microbial kingdoms [10•]. PsXEG1 is highly expressed in early infection stages and promotes *P. sojae* infection dependent on its hydrolytic activity toward xyloglucans [11••]. An assay for PsXEG1 host targets captured a

Figure 1



Working models of several characterized *P. sojae* effectors and their corresponding host targets summarized based on the current knowledge. *P. sojae* secretes effectors that act in different cellular compartments. In the apoplast, XEG1 plays dual roles in plant-pathogen interactions. XEG1 acts as a virulence effector under the protection of its close ortholog XLP1 which competes for GIP1 binding site to counter the inhibition of GIP1 on XEG1 [10•,11••]. XEG1 can also be recognized by membrane-localized receptor complex [12•], which triggers plant immunity and cell death [9•]. Of the intracellular effectors delivered into the soybean cytoplasm, Avr3b is processed by cyclophilin proteins to activate its avirulence and virulence functions [27••]; Avh262 co-localizes with host BiPs around the haustoria upon infection to regulate ER stress and plant susceptibility [44•]; Isc1 encodes a potential isochorismatase that suppresses SA accumulation and related defenses by hydrolyzing the SA precursor isochorismate into DDHB [53]. In the nucleus, both CRN115 and CRN63 interact with host catalases that are essential for CRN115 to suppress CRN63-induced cell death [46•]; Avr3c targets the plant splicesomal complex regulators SKRPs to reprogram host pre-mRNA splicing [34••]; CRN118 suppresses *HSP* gene expression by competing with transcription factors (HSFs) for binding to *HSP* promoters [33••]; PSR1 interferes with sRNA biogenesis and hijacks PINP1, a component in RNA silencing machinery [32••]; and Avh52 interacts with ADA2 to reduce GCN5-ADA2 complex-mediated histone acetylation which affects defense-related gene expression [30••]. §: positively regulates plant immunity; Φ: suppresses plant immunity.

xyloglucan-specific endoglucanase inhibitor protein called soybean glucanase inhibitor protein 1 (GmGIP1), which associates with PsXEG1 both *in vivo* and *in vitro*. GmGIP1 strongly inhibits the hydrolase activity of PsXEG1; this inhibition is intimately linked to the association between GmGIP1 and PsXEG1, illustrating that GmGIP1 blocks PsXEG1 virulence by directly deactivating the hydrolase activity. Besides PsXEG1, *P. sojae* genome encodes 10 other GH12 proteins. Of these, only *P. sojae* XEG1-like protein 1 (PsXLP1) associates with GmGIP1. PsXLP1 shares ~67% protein sequence identity with PsXEG1, but lacks detectable hydrolase activity due to a truncation in the enzyme activation site. PsXLP1 shows an expression pattern reminiscent of PsXEG1 throughout infection and is essential for *Phytophthora* virulence. The execution of PsXLP1 virulence relies strictly on the binding affinity to GmGIP1 but not on the theoretical enzymatic site. In terms of GmGIP1 binding, PsXLP1 exhibits a much higher binding affinity and effectively competes with PsXEG1 for GmGIP1