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Review 综 述

Functions of type III effectors from multigenic family of *Ralstonia solanacearum* in plant disease development and immunity defense system

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Abstract: *Ralstonia solanacearum* causes lethal wilting disease in many economic plants, threatening food security in tropical and subtropical agriculture. It injects type III effectors (T3Es) into the host cells via the type III secretion system. T3Es act as molecular double agents that are involved in either pathogenicity in the susceptible host plants or induction of hypersensitive response in the resistant host plants. A notable feature in this T3E repertoire is the existence of several multigenic families and their various internal repeats. T3Es from multigenic family of *R. solanacearum* contribute differently to pathogenicity towards the host plants and localize on the plant cell plasma membrane or nucleus. Previous researches demonstrate that the multigenic effectors jointly contribute to the plant disease development but are barely activated individually. However, the pathogenicity mechanism on the most multigenic effectors remains unclear. This review summarizes the recent achievements on elucidating the function of T3Es from multigenic family (GALA, HLK, SKWP, AWR and PopP) in *R. solanacearum*.

Keywords: *Ralstonia solanacearum*, type III effectors, multigenic family, plant immunity and disease development

In a recent survey, *Ralstonia solanacearum* has been ranked the top two most important bacterial plant pathogen, following the first one *Pseudomonas syringae*[1]. This pathogen threatens the food safety in tropical and subtropical agriculture, especially in China, Bolivia, Bangladesh, Uganda and a number of other countries. *R. solanacearum* is a Gram-negative β-proteobacterium pathogenic to plants and responsible for the development of bacterial wilt disease on more than 200 plant species from 50 families, including economical crops such as eggplant, tomato, tobacco and banana, etc[2–3]. This pathogen infects the host plants by entering through the wounds or natural openings on the plant surface. Normally it first attaches to the host root, finds nutrients, multiplies and accumulates quickly, eventually migrates into the plant tissues and penetrates the xylem with the production of exopolysaccharides (EPS) which block xylem vassels, the water traffic routes of the plant[4].

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R. solanacearum infects plants by a type III secretion system (T3SS) to cause symptoms on their respective hosts[5]. This T3SS is a syringe needle-like structure with double-layer membrane rings and a protruding filament which delivers the repertory of bacterial proteins into the host cells. These bacterial proteins, termed as Type III effectors (T3Es), either manipulate the host defense signaling pathways and/or promote the disease development[6–7]. Studies have revealed that T3Es could betray the bacterium to the plant surveillance system. Some effectors could be recognized by a cognate resistance protein (R protein), thereby triggering the host defenses and resulting in a rapid programmed cell death (PCD). This intense host defense response is known as the hypersensitive response (HR)[8–10].

Studies over the last decade have identified more than 70 T3Es in *R. solanacearum* strains but only a few T3Es have been biochemically characterized. Some effectors are from the multigenic family with overlapping properties and may also combine with each other to orchestrate the specific responses in the host cells[11]. An array of effectors which can elicit plant resistance system are identified as the avirulence (Avr) proteins. These Avr proteins could be recognized by plant resistance (R) proteins, and then trigger the plant defense immunity system, such as popP1 and popP2. Most effectors from multigenic family synergistically contribute to the plant disease development[12].

The authors have been focused on studying T3Es from the multigenic family for many years. In this review, our current understandings that how mutants lacking of single or multiple effector genes affect disease development and plant immunity system are summarized.

1 Generals of T3Es in *R. solanacearum*

1.1 Identification methods of T3Es

Generally, T3Es have been identified through either consensus sequence screening in upstream promoters, like PIP box described in *X. camperstris* or transcriptomic studies by HrpB-deficient strains[13]. Most effector candidates have been identified by translocation analysis with a calmodulin-dependent adenylate cyclase (CyaA) reporter in Japanese *R. solanacearum* strain RS1000[14–15]. So far, more than 70 effectors of *R. solanacearum* strains, which belong to 57 families including 32 core effectors, have been found.

1.2 Structure feature of T3Es

Genomics is a powerful tool and it helps to complete the identification of motifs and the structural features of T3Es in *R. solanacearum* strain OE1-1. The T3Es exhibit the distinguished protein structures carrying various internal repeats which can be suggestive of effector function[16]. As shown in Table 1, these effectors are classified into several families based on their related sequences. The recognized multigenic families till now include GALA (seven members), AWR (five), PopP (two), SKWP (six), and HLK (three).

GALA family possesses a group of seven genes that are homologies with plant-specific leucine-rich repeats (LRR). Members of this family contain a conserved GAxALA sequence in their LRR and an F-box domain[17].

Another multigenic family called AWR contains the genes of *RSc2139*, *RSp0099*, *RSp0846*, *RSp0847*, and *RSp1024* for the alanine-tryptophan-arginine tryad and a highly conserved region has been found in the primary sequences of the genes in this family.

The genes (*RSc3401*, *RSp1374*, *RSp0930*, *RSc1839*, *RSp0296*, *RSc2130*) in SKWP family contain 12–18 tandem repeats of a novel 42aa motif and certain designated SKWP repeats which are related to the heat/armadillo repeats from eukaryotes, a type of α-helices structure[18].

HLK (*RSc1386*, *RSp0215*, *RSp0160*) effectors are not only found in the several sequenced *R. solanacearum* strains, but also are homologues to

Table 1. Some T3Es from multigenic family of *R. solanacearum* and their functions (modified according to Deslandes[19])

Genes of T3Es	Family	Structure feature	Function	Reference
RSc1386(hlk1) *RSp0215(hlk2)* *RSp0160(hlk3)*	HLK	HLK2 contains 6 tandem repeats of a 9-nucleotide element	Collectively contribute to pathogenicity (tomato)	[20]
RSc0826(popP1) *RSc0868(popP2)*	YopJ (popP)	Serine/Threonine acetyltransferase domain	PopP1, HR-eliciting factor on some *Petunia* genotypes and tobacco species	[22–23]
			PopP2, avirulence factor on *Arabidopsis* genotypes carrying the RRS1-R resistance gene; contribution to bacterial fitness on eggplant	[26–30]
RSp0914(gala1) *RSp0672(gala2)* *RSp0028(gala3)*	GALA	Leucine Rich Repeats—F box domain	Components of ubiqutin-ligase complexes in host cells; GALA2,3,6,7 collectively contribute to pathogenicity (tomato and *Arabidopsis*);	[17] [33–34]
RSc1800(gala4) *RSc1801(gala5)*			GALA7 required for pathogenicity on *M. truncatula* and for invasion of its root cortical cells;	[35]
RSc1356(gala6)			GALA4 suppresses callose deposition (*Arabidopsis*)	[35]
RSc1357(gala7)			GALA2,7 interacted with chloroplastic proteins (*N. tabacum* and *N. benthamiana*)	[36]
RSc2139(awr1) *RSp0099(awr2)* *RSp0846(awr3)* *RSp0847(awr4)* *RSp1024(awr5)*	AWR	Alanine-tryptophan-arginine tryad	AWR2 contributes to pathogenicity (tomato) and has necrogenic activity on tobacco AWR5 acts as a HR-like eliciting factor on some tobacco species	[37–38]
RSc3401(skwp1) *RSp1374(skwp2)* *RSp0930(skwp3)* *RSc1839(skwp4)* *RSp0296(skwp5)* *RSc2130(skwp6)*	SKWP	Heat/armadillo repeats from eukaryotes, a type of α-helices structure	Contributes to bacterial proliferation in eggplant tissues	[39]

those of another plant pathogen genus *Xanthomonas*. HLK2 genes contain 6 tandem repeats of a 9-nucleotide element which are presumably involved in protein-protein interaction or in DNA/RNA binding[20]. So far, there are still a large number of T3Es having long disordered regions of which their secondary structure and functional clues have not yet been predicted.

2 Function of T3Es as avirulence factors in plant-*R. solanacearum* interaction

2.1 Avirulence of T3Es and effector-triggered immunity (ETI)

Some T3Es act as avirulence factors or trigger defense responses in plants. They possess corresponding *R* genes and recognize individual effector proteins. This interaction is now termed as effector-triggered immunity (ETI), which leads to a strong disease resistance response that is often associated with HR. Loss or inactivation of an avirulence gene often extends the host range of a pathogen to induce plants previously found to be resistant. For *R. solanacearum*, only *Gala4* and *PopS* have been described as being able to suppress the plants defense responses[19]. Awr5 acts as a HR-like eliciting factor on some tobacco species. AvrA and Rip36 are HR-eliciting factor on tobacco species and eggplant *S. torvum* respectively. PopP members, from YopJ family of *R. solanacearum*, are found to encode avirulence determinants[21].

2.2 PopP1 and PopP2 functional analysis

PopP1 protein controls the host specificity towards some petunia lines but is not essential for pathogenicity on tomato or *Arabidopsis*. Furthermore, *popP1* is a canonical avirulence gene in controlling host specificity at the plant species level. It acts as an avirulence gene toward *Petunia St40* line but not toward sensitive *Petunia Tr66* line[22]. In *R. solanacearum* strain GMI1000, *avrA* and *popP1* are both responsible in restricting the host range on different *Nicotiana* species, although they differ in their respective contribution to the HR elicitation[23]. *PopP1* is the major avirulence factor on *N. glutinosa*, whereas *avrA* is the major determinant on *N. tabacum* and *N. benthamiana*. Most of the Japanese virulent strains do not contain *popP1*, although a previous report mentions that *popP1* is present in most Asiatic and African isolates (phylotypes I and III)[24]. In our study, when *popP1* of HR-eliciting strain 8107 was transferred into the virulent strain OE1-1, the transconjugant strain had significantly reduced virulence and showed a HR-like phenotype[25].

PopP2 is pinpointed as the GMI1000 avirulence protein recognized by the *Arabidopsis thaliana* resistant-to-*Ralstonia solanacearum* 1-R (RRS1-R) resistance protein and targets RD19 which is an *Arabidopsis* lytic vacuole-targeted cysteine protease[19, 26]. RRS1 is strongly suspected to act as a negative transcriptional regulator of disease resistance signaling through its WRKY DNA binding -domain[27–29]. PopP2-mediated stabilization of RRS1 might reflect a bacterial strategy aimed at the down-regulation of plant defense-related genes. Further study shows that the C-terminal extension of RRS1-R is essential for popP2-dependent defense activation and RRS1-RSLH1 auto-immunity[30]. PopP2 recognition triggers a conformational switch of the immune receptor that initiates resistance signaling.

Generally, the plasma membrane and the plant nucleus are major sites of action for the phytopathogen effectors[31]. PopP family members might not be functionally equivalent because of their different subcellular localization. PopP2 is addressed to the plant nucleus because of its predicted nuclear localization signal[32]. PopP3 has a potential myristoylation site at the N-terminus, suggesting that this protein could be addressed to the plant cell membrane[32]. PopP1 is predicted to remain in the cytoplasm after translocation into the plant cell.

So far, *popP1* and *popP2* have been well characterized but the study on *popP3* is scarce due to its limited distribution.

3 Function of T3Es as virulence factors in pathogenicity or in suppression of plant immune responses

Several T3Es have been proved to be involved in *R. solanacearum* disease development and identified as virulence factors. These T3Es are classified into several families based on their related sequence features. So far, there are four T3Es families have been well characterized to contribute to pathogenicity, such as GALA, AWR, SKWP and HLK. The virulence function of these four T3Es families has been well summarized in this review.

3.1 GALA family collectively contributes to pathogenicity

Effectors from GALA family possess an F-box domain and Leu-rich repeat (LRR) which is component of E3-ubiquitin ligase complexes in eukaryotes and interacts with *Arabidopsis* ASK proteins (Table 1). GALA effectors collectively contribute to pathogenicity but are individually dispensable on *Arabidopsis* and tomato plants[33–34]. GALA7 is required for pathogenicity on *M. truncatula* and for invasion of its root cortical cells[35]. Our study indicated that GALA effectors, especially for GALA2 and GALA7, interacted with chloroplastic proteins of *N. tabacum* and *N.*

benthamiana[36]. It is speculated that SCF-GALA complex interacting Skp1 with GALA effectors would target the plant chloroplastic proteins for ubiquitination and subsequent degradation which indicated that GALA effectors might target the chloroplastic proteins of the host plants to inhibit photosynthesis and impair plant immunity for the disease development.

3.2 AWR family synergistically contributes to pathogenicity

It has been demonstrated that effectors of AWR family synergistically contributed to bacterial virulence, although AWR2 is the major contributor to virulence (Table 1). Additionally, AWR4 and AWR5 restrict the bacterial growth in *Arabidopsis*, and the later one shows characteristics of a typical hypersensitive response on *N. benthamiana*[37]. AWR can specify either virulence or avirulence in the interaction of *R. solanacearum* with its plant hosts. AWR2 is the major contributor to virulence on tomato and eggplant while AWR5 exhibited a typical HR phenotype on tobacco. Furthermore, AWR5 has been proved to be an inhibitor of the TOR signaling pathway[38]. Solé shows that all AWR effectors are evenly localized in the cytoplasm, with some association to membranes[37].

3.3 SKWP function hypothesis

SKWP family distributes in all sequenced *R. solanacearum* strains and also appears in other bacterial pathogens. We used Competitive Index (CI) assay to evaluate the contribution of single SKWP effectors to bacterial fitness towards the host plants[39–40]. The SKWP effectors were important for bacterial proliferation in eggplant tissues while SKWP4 appeared to be most important (Table 1). Like the leucine-rich repeat and an F-box domain of GALA family, it is predicted that the SKWP repeats somehow participate in the interaction between bacteria and plant, but the mechanism and function remains unknown and requires further experiments to verify.

3.4 HLK family jointly contributes to pathogenicity

The inactivation of the individual T3E genes has no detectable impact on virulence on the susceptible host, except in two cases (*awr2* and *gala7*) for which the corresponding mutants showed slightly delayed symptom development on plant. Our study showed that single HLK mutants did not affect virulence on tomato but the triple HLK mutant did[20]. The HLK double deletion mutants caused the wilting death on tomato but the one bearing only *hlk2* exhibited more aggressive than two others indicating that *hlk2* played an important role in bacterial fitness in planta. The existence of 6 tandem repeats of a 9-nucleotide element of HLK2 might be vital to protein-protein interaction or in DNA/RNA binding.

In addition to a functional overlap among effectors, it is also likely that such functional groups of T3Es are required to establish host susceptibility by suppressing immune responses that may vary from one plant to another[41]. GALA effectors contribute to pathogenicity much more on *Arabidopsis* than on tomato. AWR effectors are capable of restricting the bacterial progression on both eggplant and tomato but not on *Arabidopsis*. HLK effectors were jointly important for the virulence of *R. solanacearum* on tomato, and each HLK effectors compensated each other.

4 Perspectives

Current studies have proven that T3Es of multigenic family collectively contribute to the bacterial virulence development and individually suppress the plant immunity defense. This review summarizes the functions of the known T3Es from multigenic families to date. However, the most studies focus on the disease development, but the mechanism and signaling cascade of these T3Es remain unknown[42–43]. More investigations of biochemical function of multigenic families (SKWP, HLK and AWR) are required in future. Consecutive

approaches including target protein screening, subcellular localization, effector expression and translocation during infection, and enzymatic activity assay are necessary for us to better understand the T3Es functions in the interaction between *R. solanacearum* OE1-1 and its host plants.

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青枯劳尔氏菌多基因家族 III 型效应蛋白在植物病害发展及防御系统中的作用

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摘要：青枯劳尔氏菌是导致多种重要经济作物毁灭性枯萎(bacterial wilt)的一种土传病害，严重危害热带和亚热带地区食品安全。该细菌通过 III 型分泌系统(T3SS)向寄主细胞注射大量效应蛋白(T3Es)。效应蛋白是把双刃剑，既可诱导植物感病，又能激活植物防御系统。具有特殊重复结构的效应蛋白被归类成多基因家族，各家族成员协同致病，但其分子机制尚不清楚。本文围绕近年来有关多基因家族效应蛋白结构、功能和致病性等方面最新进展进行综述，为青枯菌致病机理和病害防治提供新思路。

关键词：青枯菌，III 型效应蛋白，多基因家族，植物免疫和致病性

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