

## Over the Suppressor from *Phytophthora infestans* Single Molecule Signaling in Hypersensitive Cell Death of Host Cell against Ca<sup>2+</sup>-Dependent Protein Kinases

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### Editorial

The interaction between plants and a suppressor or a host-selective toxin (HST) producing pathogen leads often to suppression of rapid and localized cell death in the context of a hypersensitive response [1]. The suppressor for hypersensitive response (HR) was reported from *Phytophthora infestans* [1]. The elicitor of hyphal wall of *P. infestans* was also reported from Tomiyama group [2]. Recently, we have reported the isolation of PiPE effector gene from *P. infestans*, of which peptide induce the generation of active oxygen species (AOS) and HR cell death in potato and tomato [3]. In these reported physiological events for the induction of HR and AOS generation, and the inhibition of these resistance responses, Ca<sup>2+</sup>-dependent protein kinase (CDPK) was involved in for the signal cascades in host cell [4,5]. It was suggested that CDPK molecule is a switch for the induction or the inhibition of HR response in host cell (2008) [4,6].

The mechanisms of these molecular infection events are presumed to be as follows: (1) initial recognition of the PAMPS (pathogen associated molecular patterns) and the suppressor of the pathogen by host plasma membrane in the infection process [2,7-10]; (2) increase in Ca<sup>2+</sup> influx and the kinase activation in the cells [11,12]; and (3) induction of biochemical defense in the host cells [5]. A PAMP of *Phytophthora infestans* and the hyphal wall components (HWC) elicited HR. In our attempt to explore the HR suppressor and the antigenic potential of *P. infestans* derived surface structure to elicit non-specific defense response in potato, we have previously identified PiPE, an elicitor peptide for HR and the generation of AOS [3]. The PiPE was shown to serve as a recognition PAMP for the activation of HR.

However, from the reported RXLR-genes of *Phytophthora* species, what is the real product is not yet known. Receptor binding of PiPE evokes a PAMP-specific cytoplasmic streaming, and the brownian movement and gelation in the cytosol, production of AOS as well as translational activation of CDPK kinases, all of which are important elements for the transmission of the PiPE signal. From these evidences, we proposed that PiPE and a HST (host-selective-toxin) from *Alternaria solani*, can regulate HR by the binding with CDPK on the plasma membrane of potato as reported [4,5].

So far, the RXLR genes from *Phytophthora* spp. interacted with the resistance genes in gene-for-gene level in the host cell [13]. However, how did a secreted protein from Avr4/6 with an RXLR-dEER protein translocation motif work with the receptors in host cells, is not yet resolved.

In the future works, we need the single molecule detection and observation in situ in host cells. For the new frontier of plant infection mechanisms of the host-pathogens interaction, we will explain the

infection process by using the single molecule interaction in situ analysis in host cell with regard to the effector-receptor interaction and the signal transduction in a specific host-parasite interaction for the explanation of hypersensitive response in host cells (Specific Issue, 2015, J Plant Pathology and Microbiology, in preparation).

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