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Letter to the Editor

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Host Specific Toxin, Suppressor Effector from Potato Late Blight Pathogen can Regulate Ca^{2+} -Dependent Protein Kinases in Host Cells

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Letter to the Editor

The interaction between plants and an incompatible pathogen leads often to rapid and localized cell death in the context of a hypersensitive response (Doke, Tomiyama et al. 1982). In the hypersensitive response in plant, hypersensitive cell death (HR), a defense mechanism, is characterized by rapid cell death at the site of infection, which restricts further growth of the infecting pathogen (Doke, Tomiyamaet al. 1982, Furuichi, Anderson et al. 1993). The mechanisms of these molecular events are presumed to be as follows: (1) initial recognition of the PAMPS (pathogen associated molecular patterns) and the suppressor and/or Host Selective Toxins of the pathogen by host plasma membrane in the infection process (Furuichi, Tomiyama et al. 1980, Langsdorf, Furuichi et al. 1990, Furuichi, Kusakari et al. 1997, Furuichi, Suzukietal. 1998); (2) increase in Ca^{2+} influx, decrease pH, and the kinase activation in the cells (Goodman and Novacky 1994, Harmon, Gribskov et al. 2000); and (3) induction of biochemical and physiological defense in the host cells (Tomiyama 1982, Levine, Tenhaken et al. 1994, Martin and Busconi 2000). A PAMP of an Oomycete, Phytophthora infestans, and the Hyphal Wall Components (HWC) elicited Hypersensitive Reaction (HR). In our long standing attempt to explore the HR suppressor and the antigenic potential of P. infestans derived surface structure to elicit cultivar-non-specific defence response in potato, we have previously identified PiPE, 38 KD, elicitor peptide for HR and the generation of Active Oxygen Species (AOS).

PiPE was almost invariably found to be associated with FBA (Fructose Bisphosphate Aldolase) from six Phytophthora species. 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 (Dou, Kaleetal. 2010). Receptor binding of PiPE evokes PAMP-specific cytoplasmic streaming, and the brownian movement in the cytosol

(Tomiyama 1982), production of AOS as well as translational activation of CDPK kinases (Furuichi, Yokokawa et al. 2008, Furuichi, Yokokawa et al. 2013), all of which are important elements for the transmission of the PiPE signal. From these evidences, we proposed that PiPE and Host Selective Toxin (HST, Alternaric acid from Alternaria solani), can regulate HR cell death by the binding with CDPK on the plasma membrane of potato as reported (Furuichi et al. 2014). Recently, we reported that f-MRI system of 7 tesra, Brain Institute of Niigata U., can detect the water channel water streaming inside the host cells of plants, and that the elicitor treatment on the host cells caused The AOS streaming from the infected host cells to the neighbouring ones through by Aquaporin 4, and 3 and 8 groups, in host cells. (N. Furuichi, Annual Meeting of Plant Physiological Society of Japan, Kanazawa, 2025). Doke, N., et al. (1982). Elicitation and suppression of hypersensitive response in host-parasite specificity. Plant Infection: The physiological and Biochemical Basis. Y. Asada, W.R. Bushnell, S. Ouchi and C.P. Vance, Japan Sci. Soc. Press, Tokyo/Springer-Verlag, Berlin: 79-96.

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