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学 位 論 文 要 旨
Dissertation Abstract

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学位論文題目：
Title of Dissertation　Analysis of the pathogenic function of *Cauliflower mosaic virus* multifunctional protein Tav (Transactivator/viroplasmin) in transgenic tobacco（形質転換タバコを用いたカリフラワーモザイクウイルスTavタンパク質の病原性機能の解析）

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Cauliflower mosaic virus (CaMV), the type member of plant pararetroviruses (Caulimoviridae), encodes a specific multifunctional protein, the transactivator/viroplasmin (Tav). A striking feature of CaMV Tav as a model to study virus pathogenesis is that it induces virus-symptom-like phenotype in transgenic plants expressing Tav alone. Expression of defense-related genes was reported in the transgenic tobacco plants expressing Tav. However, it remains unknown what of tobacco cell perceives Tav, which signaling pathway(s) is activated, and what causes the chloroplast damage. To establish an experimental system which allows us to pursue the molecular changes during, especially in the early phase of chlorosis, we generated transgenic tobacco plants expressing CaMV Transactivator/viroplasmin (Tav) under the control of chemically inducible promoter. Induction of Tav resulted in visible chlorosis in seven days, statistically significant decrease in chlorophyll content in two days, decreased expression of chloroplast protein genes, and abnormal thylakoid stacks, indicating that this system reproduces the common features of chlorosis in virus-infected plants. Further, to identify pathogenesis or stress related genes up-regulated/ down-regulated during the expression of symptom-like phenotype, a common transcriptome analysis was done in transgenic tobacco expressing the

symptom-like phenotype. Shortly after the induction of Tav expression, pathogenesis-related protein (PR) 1a gene expression was up-regulated in the transgenic tobacco lines. Then it was shown that the expression of Tav also induce some salicylic acid (SA)-and ethylene-responsive PR genes. In contrast to transiently expressed Tav, which suppressed the Agrobacterium-induced and SA-induced PR1a expression, the artificial induction of Tav from the transgene did not affect SA-induced PR1a expression and rather induced the *PR1a* expression by itself. In deletion analysis, chlorosis and PR1a induction function in transgenic tobacco was mapped to a region in Tav, which had been shown to have a role in pathogenesis in susceptible host, elicitation of hypersensitive response in resistant host, RNA-silencing suppression, and the suppression of Tomato bushy stunt virus P19-mediated cell death in tobacco. Transient expression of nahG gene coding SA hydrolyzing enzyme barely affected the Tav-induced PR1a expression and didn’t affected Tav-induced chlorosis. The results suggest that Tav-induced chlorosis is resulted from a SA-independent host response to pathogenic function of Tav.

Characterization of the transgenic plants supported the idea that they would provide a good system for analyzing early events as well as temporal changes in the process of virally induced chlorosis. In next study, we observed that Tav-induced PR1a expression is SA-independent, because tobacco may have a kind of broken immune receptor that inefficiently recognize Tav and drive inefficient ETI response. Further studies examining the effect of knock-down of ETI components on Tav-induced chlorosis would help us understand the relationship between chlorosis, which has been regarded as a susceptible symptom, and the cell death, which is accepted to be a major component of plant disease resistance.