Dynamics, mechanisms, and evolution of a highly resilient plant immune signaling network

講師：片桐　文章　教授

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第５回 ERATO 学術セミナー

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Microbial pathogens can evolve much faster than plants and compromise the plant immune signaling network. Thus, the immune signaling network needs to be highly resilient against perturbations to its internal components, so that its underlying mechanisms are effectively concealed from pathogen evolution. To overcome the identifiability problem associated with a highly resilient network, we reduced the network to a network of four signaling sectors, the jasmonate (JA), ethylene (ET), PAD4, and salicylate (SA) sectors, in the model plant Arabidopsis and simultaneously impaired the four sectors by quadruple mutations (*quad*). Pattern-triggered immunity (PTI) triggered by the bacterial molecular pattern flg22 and effector-triggered immunity (ETI) triggered by the bacterial effector AvrRpt2 were largely abolished in *quad* [1]. Then the network functions and the signal flows among the four sectors were analyzed in comprehensive combinatorial states of the sectors. This analysis enabled a conceptual reconstitution of the network at the sector scale, which allowed simpler interpretations of mechanistic relationships among the sectors underlying network resilience[1,2]. Using network reconstitution, we also demonstrated that transcriptome response during flg22-PTI is highly resilient against loss of some of the sectors[3]. Furthermore, studying the network with highly impaired resilience in *quad*, we discovered another signaling sector that mediates strong inhibition of ETI signaling by PTI signaling (ETI-Mediating PTI-Inhibited Sector, EMPIS). We speculate that the role of this inhibition is to limit ETI when PTI is not compromised: such a mechanism would limit a negative impact of unnecessary immunity on plant fitness. I will also discuss how such a resilient network may have evolved.

日時：2017年５月25日（木）

時間：16:00 ～ 17:30

会場：筑波大学総合研究棟 A110 室

参加費無料・事前申し込み不要

[1] Tsuda *et al.* 2009 PLOS Genet 5, e1000772.
[2] Kim *et al.* 2014 Cell Host Microbe 15, 84.
[3] Hillmer *et al.* PLOS Genet, in press

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