

植物ゲノム・遺伝子源解析センター

月例セミナー

- とき 平成22年5月27日(木)
16時～17時15分
- ところ 農学部 BW106講義室(大講義室)
- 題目 「Multiple routes to activation of salicylic acid signaling - A key role of CBP60 proteins」
- 講師 米国ミネソタ大学准教授 Jane Glazebrook 博士

概略

Salicylic acid (SA) signaling is important for resistance to biotrophic and hemi-biotrophic pathogens such as the bacterium *Pseudomonas syringae* pv. *maculicola* strain Psm ES4326. It can be activated by recognition of Microbe Associated Molecular Patterns (MAMPs) such as flg22, a fragment of bacterial flagellin (Pattern Triggered Immunity, PTI), by recognition of pathogen effectors (Effector Triggered Immunity, ETI), or other responses to pathogen attack. In published work, we showed that Calmodulin Binding Protein 60g (CBP60g) contributes to activation of SA production during PTI, is required for wild-type levels of resistance to Psm ES4326, and requires the ability to bind calmodulin (CaM) in order to function (Wang et al., *PLoS Pathogens* 5(12): e1000772 2009). Another family member, CBP60h, is also required for resistance to Psm ES4326, but does not bind CaM. SA levels in *cbp60h* mutants are normal early during PTI, but reduced at later times and during infection by Psm ES4326. Double *cbp60g,h* mutants have severely reduced SA levels under all conditions tested and extreme susceptibility to Psm ES4326. Thus, these two proteins perform a critical and partially-redundant function in activation of SA production during defense responses. Expression profiling revealed that the CBP60g,h node lies between the EDS1/PAD4 and SA nodes in the defense signaling network, and suggests that none of *eds1*, *pad4*, or *cbp60g,h* result in complete loss of function of their respective nodes. Our results suggest a model in which CBP60g responds to Ca²⁺ flux early during PTI and activates SA signaling. As the Ca²⁺ flux wanes, CBP60h assumes primary responsibility for promotion of SA production.