論文の内容の要旨

Molecular mechanism of stress induced cellular reprograming

（ストレスによる細胞リプログラミング誘導機構の解析）

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Plants readily regenerate organs and repair tissue after injury by inducing cellular reprograming near the wound site. This process is governed by both stress-activated reprograming regulators and developmental regulators induced by phytohormones such as auxin and cytokinin. Exogenous application of these hormones to explants can induce organ regeneration, however, wounding often provides the trigger necessary for cellular reprogramming and subsequent shoot regeneration. While previous reports have demonstrated the requirement of important stress-induced transcriptional regulators such as WOUND INDUCED DEDIFFERENTIATION 1 (WIND1) in these reprogramming processes, further identification of novel regulators is needed to gain more comprehensive understanding of this interplay at the molecular level. In this thesis I first demonstrate that the master regulators of the heat stress response HEAT SHOCK FACTOR A1s (HSFA1s) positively regulate *in vitro* shoot regeneration. I also show that overexpression of a gain-of-function *HSFA1d*, which lacks a key regulatory repressive domain (*HSFA1dΔ1*), dramatically promotes shoot regeneration. RNA sequencing analysis of *HSFA1dΔ1* overexpressor undergoing shoot regeneration revealed that HSFA1 can influence expression of shoot meristem

regulators but not callus and pluripotency regulators. Furthermore, I demonstrate that the SUMO E3 ligase SIZ1, which catalyzes the attachment of SUMO to proteins, negatively regulates *in vitro* shoot regeneration. I revealed that *siz1* mutant exhibits enhanced transcriptional responses to wound stress and that upregulation of *WIND1* and *WIND2* in *siz1* mutant, contributes to the observed phenotype. I found that the expression of shoot meristem regulators like *WUSCHEL* (*WUS*) is also enhanced in *siz1* explants, while expression of callus and pluripotency regulators is largely unchanged. In addition, I demonstrate that one of the HSFA1 paralogs, HSFA1d, can be modified by SMALL UBIQUITIN-LIKE MODIFIER (SUMO) *in vivo* in response to stress, raising the possibility that HSFA1 may be one of the targets of SUMOylation that has impact on the stress-induced cellular reprogramming and subsequently shoot regeneration. Together, I revealed two novel mechanisms that regulate shoot regeneration in Arabidopsis.