



Title	Molecular genetic study of lateral root regeneration on environmental stresses [an abstract of dissertation and a summary of dissertation review]
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学位論文内容の要旨

博士の専攻分野の名称 博士（生命科学） 氏名 徐冬ヤン

学位論文題名

Molecular genetic study of lateral root regeneration on environmental stresses
(環境ストレスによる側根再生の分子遺伝学的研究)

Plants have a powerful ability of organ regeneration in response to adverse environmental conditions such as physical damage or exposure to toxic chemicals. A root system with the primary root (PR) injured is able to undergo regeneration by increases in lateral root (LR) number and acceleration of LR growth. This regeneration ability has been extensively used in agricultural or horticultural techniques such as "root pruning", where part of the root system was removed and a new root system with more branches and smaller size built, to control plant growth and fruit quality. However, how the damage of PR leads to the regeneration of LRs remains elusive. In this study, I try to answer this question by revealing the underlying mechanisms of LR regeneration in response to environmental stresses, particularly mechanical wounding such as root pruning or wound related peptide hormones, like *AtPeps*.

In the first chapter of this thesis, genetic and molecular mechanism of root regeneration following root pruning was investigated. I found 1) After removal of the PR tip by root pruning, LR defects in wild-type plants treated with polar auxin transport (PAT) inhibitors or in the auxin-signaling mutant *auxin/indole-3-acetic acid19massugu2* were recovered. 2) Induction of *IAA19* following root pruning indicates an enhancement of auxin signaling by root pruning. 3) Endogenous levels of IAA increased after root pruning and *YUCCA9* was identified as the primary gene responsible. 4) PAT-related genes were induced after root pruning and the YUCCA inhibitor yucasin suppressed root regeneration in PAT-related mutants. These results indicate the crucial role of *YUCCA9*, along with other redundant *YUCCA* family genes, in the enhancement of auxin biosynthesis following root pruning. This further enhances auxin transport and activates downstream auxin signaling genes, thus increases LR number.

In the second chapter of this thesis, the plant elicitor peptides, *AtPeps*, which has been extensively studied for their function on the innate immune response, however less documented in their role on plant growth and development, were investigated for their effect on root formation. I found 1) *AtPep1* and *AtPep2* inhibited PR growth, meanwhile increased LR density. 2) The PEPR2 receptor was responsible for the *AtPeps*-induced root system morphology changes. 3) *AtPep1* and *AtPep2* inhibit PR growth through the disturbance of cell cycle in root tip, thus promote LR formation, which is dependent on the auxin signaling pathway. 4) This process is independent of *AtPeps*' role on pattern-triggered immunity, as *flg22* and *elf18* were unable to trigger the same response in the root. 5) *AtPep1* treatment induced the ectopic expression of *PEPR1* and *PEPR2* in the root tip, which also occurred with mechanical damages on root tip, suggesting the potential role of *AtPep1* in response to environmental stimuli, though the specific biological meaning is yet to be revealed. These results indicate that *AtPep1* and *AtPep2* modify the root system architecture through the inhibition of PR growth and promotion of LR formation, which is dependent on the auxin signaling pathway.

Inhibition of PR growth through removal of PR tip or treatment of *AtPeps* on root tip led to the increase of LR number and acceleration of LR growth. This reflects the plants' high plasticity to maintain a balanced growth of the root system architecture when part of the root is injured. These two ways of PR damage triggered the shared downstream pathways of auxin signaling and LR formation, however, the perception of the damage signals is differed. In this study, I showed how LR regeneration was induced by the restriction of PR growth, elucidated different mechanisms underlying this process with root pruning or root tip inhibition by *AtPeps*, and discussed how different environmental stresses differentially induce LR regeneration through the common integrator of auxin.