学 位 論 文 内 容 の 要 旨

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Study on epigenetic-independent control of Tam3 transposition in *Antirrhinum*
(キンギョソウのトランスポゾン Tam3 における非エピジェネティックな転移制御機構に関する研究)

Transposable elements (TEs) are widespread in the plant genomes. As characterized as mobile elements, TEs can move and insert into new positions within a genome, resulting in gene disruption, chromosome breakage, illegitimate recombination, and genome rearrangement. To maintain the stability and integrity of plant genomes, TE activities are under rigorous control of the hosts. Although hosts adopt a series of mechanisms in eliminating TE activity, a variety of conditions can trigger transposon activation, which are necessary for both TEs and their hosts. TEs need to maintain a certain level of activity to ensure their propagation and survival, while sometimes hosts need to activate TEs to serve for their growth and development, especially under stress. Because TEs are able to benefit the host organisms through their functions in genome organization, gene expression regulation and new gene creation. In this study, we performed detailed analyses on how cold stress affect the activities of TEs in plants and the mechanisms involved in regulating the TEs activation.

In *Antirrhinum*, DNA transposon Tam3 exhibits an unusual and remarkable feature of low-temperature dependent transposition (LTDT): activation at low growth temperatures (15℃) and inhibition at high temperatures above 25℃. Most especially, we found that LTDT of Tam3 is epigenetic-independent. LTDT of Tam3 was associated with temperature-dependent nuclear transfer of Tam3 transposase (TPase). The low temperature allows the TPase to transfer into nuclei resulting in the transposition of Tam3, but high temperature inhibits this nuclear import, causing the silencing of Tam3. Our previous study found that Tam3 TPase was equipped with a functional domian in the N-terminal region in confining nuclear transport of Tam3 TPase. In this study, this functional domain was identified to be comparable to BED-zinc finger domain that detained Tam3 TPase on the plasma membrane. This BED-zinc finger domain could also direct the other proteins with the different subcellular localizations to the plasma membrane in *Antirrhinum* cells. Some particular point mutations in the BED-zinc finger domain abolished detainment of the TPase on the plasma membrane. Functional amino acids in the BED-zinc finger domain were also determined by these mutation analysis. BED-zinc finger domains in transposable elements have been generally known to be capable of binding ability to their own element DNAs to initiate the transposition. Our study reveals that the BED-zinc finger domain of Tam3 TPase has bidirectional functions in regulating the activity of Tam3, which depends on the subcellular localization of TPase. Besides DNA binding ability in targeting transposon sequences in the nucleus, the BED-zinc finger domain in Tam3 facilitates to interact with certain host factor(s) causing inhibition of TPase nuclear transport, resulting in inactivation of Tam3. In addition, this post-translational control, in an epigenetic-independent manner, is a first finding regarding silencing of transposon that the BED-zinc finger domain targeted by host factor(s) resulted in detainment of transposase at plasma membrane.