

**Specific regulation on
diverse regenerative responses
in plants****Momoko Ikeuchi**
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Nara Institute of Science and Technology)**Replication dynamics identifies
the folding principles of
the inactive X chromosome****Rawin Poonperm**
(Laboratory for Developmental Epigenetics,
RIKEN BDR)**2023.2.20 (Mon)****13:00-14:00 JST**

← via Zoom Register here

[https://riken-jp.zoom.us/meeting/register/tJcucOuuqzqvE9wXfy2HEjEHd-vsTutZGJ3r](https://riken-jp.zoom.us/join/91234567890)**Specific regulation on diverse regenerative responses in plants**

Life is full of damaging stresses. Upon injury, plants display various wounding responses including callus formation, activation of defense responses and the establishment of stem cells de novo. Previous studies identified general regulators that orchestrate various physiological responses provoked by tissue injury (Iwase et al., 2011; Iwase et al., 2021). Plants display specific regenerative response in a context-dependent manner, yet molecular and developmental mechanisms that specifically regulate different regenerative responses remain unknown. We recently identified WUSCHEL-RELATED HOMEODOMAIN 13 (WOX13) as a key regulator of tissue repair and organ adhesion in *Arabidopsis thaliana* (Ikeuchi et al., 2022; Tanaka et al., 2023). Strikingly, wox13 mutant is totally deficient in organ reconnection in petiole grafting, suggesting that WOX13 is pivotal for the establishment of organ reconnection. We recently found that WOX13 represses other aspects of organ regeneration, implicating that WOX13 may be a specific regulator of diverse regenerative responses.

Replication dynamics identifies the folding principles of the inactive X chromosome

Chromosome-wide late replication is an enigmatic hallmark of the inactive X chromosome (Xi). How it is established and what it represents remains obscure. By single-cell DNA replication sequencing, here we show that the entire Xi is reorganized to replicate rapidly and uniformly in late S-phase upon X-chromosome inactivation (XCI), reflecting its relatively uniform structure revealed by 4C-seq. Despite this uniformity, only a subset of the Xi became earlier replicating in SmcHD1-mutant cells. In the mutant, these domains protruded out of the Xi core, contacted each other, and became transcriptionally reactivated. 4C-seq suggested that they constituted the outermost layer of the Xi even before XCI and were rich in escape genes. We propose that this default positioning forms the basis for their inherent heterochromatin instability in cells lacking the Xi-binding protein SmcHD1 or exhibiting XCI escape. These observations underscore the importance of 3D genome organization on heterochromatin stability and gene regulation.