

Allison Bardin

Stem Cells and Tissue Homeostasis, Institut Curie

Wednesday, July 12, 2023

14:00-15:00

1F Auditorium, DB Building C, Kobe / Broadcast online via Zoom

Zoom meeting URL will be announced on the event day by e-mail.

※Non-BDR members: Please register from the following link.

<https://krs1.riken.jp/m/bdrseminarregistration> (Registration deadline July 10)

Genomic regulation of stem cells: from lineage decisions to genome stability

Summary

Adult stem cells replenish tissues through their activities of self-renewal and lineage differentiation. These essential functions of stem cells are ensured by transcriptional and epigenetic regulation. Fidelity of gene expression, in turn, relies on an accurate genomic content, which must be safeguarded from endogenous and exogenous mutagenic processes. We aim to better understand stem cell genome expression and stability by exploring the questions: How is gene expression controlled allowing stem cell fate decisions? What are the cellular mechanisms protecting stem cell genomes from mutation and what happens when they fail? We address these questions using the *Drosophila* intestinal stem cell model system, a simplified model system with excellent genetic and genomic tools.

In the first part of my talk, I will discuss insight we have gained into epigenetic regulation of the stem cell lineage, investigating globally how chromatin states change during differentiation revealing underlying principles of chromatin changes during multipotent adult stem cell differentiation. In the second part of my talk, I will focus on understanding how the stem cell genome is altered through spontaneous mutation. Our recent work in *Drosophila* and that of others in mammalian model systems have demonstrated that adult stem cell mutation is frequent and can have significant phenotypic consequences on adult tissues. Importantly, the underlying causes driving mutational processes remain to be fully understood. In particular, I will focus on our unpublished findings (in prep) regarding tissue-specific buffering of replication stress via nucleotide pool sharing.