BDR SEMINAR (Kobe/Online hybrid)

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Wednesday, January 12, 2022

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. <u>https://krs1.riken.jp/m/bdrseminarregistration</u>

Molecular steps to break the genome during meiosis

Summary

Meiotic cells have developed a sophisticated molecular pathway allowing homologous chromosome to interact, pair and be connected. This pathway relies on homologous recombination events which are initiated by the programmed induction of DNA double strands breaks (DSBs). The absence of meiotic recombination leads to sterility. In mammals, about 300 DSBs are induced in each oocyte or spermatocyte at the onset of the first meiotic prophase. These molecular events are therefore at the same time a major challenge for genome stability and a driving force for generating genetic diversity at each generation, a unique feature of sexual reproduction.

Our lab has been aiming to understand the molecular mechanism and regulation of meiotic DSB formation.

We discovered a few years ago that the distribution of meiotic recombination is determined by PRDM9 in humans and mice. PRDM9 is a sequence-specific DNA binding protein with a methyltransferase activity, catalysing H3K4me3 and H3K36me3. We have recently identified HELLS, a chromatin remodeler as a partner of PRDM9. We have also identified the long-sought missing subunit for DNA break formation, TOPOVIBL, a partner of SPO11 carrying the catalytic activity, and also highlighting the evolutionary link with Type II DNA Topoisomerases. Our current view of how these molecular steps are coordinated to ensure the proper formation and repair of DSBs will be presented.

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