Dynamic organization of human mitotic chromosomes

The mitotic chromosome condensation during cell division is an extraordinary self-organization of biological supramolecules. In this process, the long thin genome chromatin polymers (several centimeters) are converted into compact short chromosomes (several micrometers) together with proteins involving chromosome condensation and segregation in about one hour. ATP-driven DNA motors called condensins and topoisomerase IIα (TopoIIα), which untangles two sister chromatids, are identified as essential factors for chromosome condensation. However, the basic physical principles of condensation remain an important question in biology. Here, we analyzed the local motion of nucleosomes in the mitotic chromosomes during condensation by using intracellular single-molecule imaging techniques combined with rapid depletion of condensins by an auxin-inducible degron (AID) system. Together with computer simulations, we discuss how condensin, TopoIIα, and other factors involve the condensation process of the chromosomes from the physical aspect.

Spatio-temporal control of cellular mechanics during cell migration

Cell migration is a fundamental process in many biological events, such as development, cancer metastasis and immune responses. To migrate directionally, cells establish integrin-mediated adhesions (focal adhesions) to adhere and sense the extracellular environment. How dynamic turnover of focal adhesions is co-ordinated with the front-to-rear axis during cell migration is one of the challenging problems. Microtubules are thought to physically target and induce adhesion turnover, but its molecular mechanisms have not been fully understood.
We previously identified that KANK (Kidney ANKyrin repeat domain) proteins connect focal adhesions to microtubule ends. This ‘capture’ of microtubules by KANK traps RhoGEF GEF-H1 on microtubules, which decreases myosin IIA activity and induces disassembly of focal adhesions (Nature materials, 2019). In this seminar, I will propose the mechanism underlying the local control of KANK-to-GEF-H1 axis and discuss its contribution to directed cell migration.

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