



ON-SITE BIOLOGY COLLOQUIUM

Friday, 20 Feb 2026 | 4 pm | S3 05-02 Conference Room 1

Hosted by Associate Prof Liou Yih-Cherng

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Ensuring faithful chromosome segregation by controlling the stability and dynamics of spindle microtubules



By Patrick Meraldi

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About the Speaker

Patrick Meraldi studied Biochemistry at the ETH Zurich (Switzerland), before carrying out his PhD and a first post-doc with Erich Nigg at University of Geneva (Switzerland) and the Max-Planck Institute for Biochemistry in Munich (Germany) working on centrosome duplication, and the function of mitotic kinases. After a post-doc with Peter Sorger at the MIT (USA), where he studied kinetochores and the spindle assembly checkpoint, he started his own research group as a Swiss National Science Assistant professor at the ETH Zurich in 2005, working on mitosis and chromosome segregation. In 2012 he was nominated Associate Professor at the Medical Faculty of the University of Geneva, and then promoted to Full Professor in 2018. His group continues to study the fundamental mechanisms controlling mitosis and chromosome segregation in human cells, and to investigate how a deregulation of these mechanisms can be targeted in cancer cells.

During mitosis, cells must achieve two fundamental steps: aligning the chromosomes on the metaphase plate and synchronously segregate the sister chromatids into the two daughter cells. This is achieved by the mitotic spindle, a dynamic microtubule-based structure. How the different elements of the mitotic spindle orchestrate chromosome alignment and segregation is still not well understood. Here I will highlight two key contributions to this process. First, I will show in human cells, how five targets of the Ran-GTP gradient jointly regulate the nucleation and stabilization of bridging fibers, a subset of spindle microtubules that connect sister-kinetochore microtubules. While depletion of single Ran-GTP targets leads to mild defects, double depletions severely disrupt chromosome alignment and segregation, illustrating how cells bring robustness to the spindle. Second, I will show how the loss of a set of microcephaly-linked genes deregulates microtubule dynamics at spindle poles, causing lagging chromosomes in anaphase, that activate 53BP1/p53/p21 and impair cell proliferation. Restoring normal microtubule dynamics at spindle poles suppresses these lagging chromosomes and the cell proliferation defects in both human cells and in fly neuroblasts. Most importantly, it also rescues the overall microcephaly phenotype in flies, illustrating how a deregulation of mitosis is linked to severe pathologies.