CDB SEMINAR


# Marcos Gonzalez Gaitan

Department of Biochemistry, Sciences II

University of Geneva, Switzerland


Tuesday, June 7, 2016

16:00-17:00 C1F CDB Auditorium

## Mechanism of the asymmetry of signaling endosomes during asymmetric division


**Summary**

During asymmetric division, fate determinants at the cell cortex segregate unequally into the two daughter cells. We have recently showed that Sara signaling endosomes in the cytoplasm also segregate asymmetrically during asymmetric division. Biased dispatch of Sara endosomes mediates asymmetric Notch/Delta signaling during the asymmetric division of sensory organ precursors in *Drosophila*. In flies, this has been generalized to stem cells in the gut and the central nervous system. We also showed that, in zebrafish, Sara endosomes are implicated in asymmetric cell fate assignation during division of the neural precursors of the spinal cord. However, the mechanism of asymmetric endosome segregation is not known. We unravelled now this mechanism. The plus-end kinesin motor Klp98A targets Sara endosomes to the central spindle. At the central spindle, endosomes move bidirectionally on an antiparallel array of microtubules. The microtubule depolymerising kinesin Klp10A and its antagonist Patronin generate central spindle asymmetry. The asymmetric spindle, in turn, polarizes endosome motility, ultimately causing asymmetric endosome dispatch into one daughter cell. Spindle inversion targets the endosomes to the wrong cell. Our data uncovers the molecular and physical mechanism by which organelles localized away from the cellular cortex can be dispatched asymmetrically during asymmetric division.



**Host:**
**Fumio Matsuzaki**
Cell Asymmetry, CDB
fumio@cdb.riken.jp
Tel:078-306-3216
(ext:1632)