Colloquium

Asymmetric signaling endosomes in asymmetric division

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During asymmetric division, fate determinants at the cell cortex segregate unequally into the two daughter cells. It has recently been shown 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 and, in zebrafish, to 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.

Monday, November 27, 2017 04:00pm - 05:00pm
Raiffeisen Lecture Hall, Central Building

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