CDB SEMINAR

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16:00~17:00 A7F Seminar Room

Microtubules break symmetry in *C. elegans* zygotes by protecting PAR-2 from cortical exclusion by PKC-3 kinase

Summary

Polarity is a fundamental property of many cell types. To become polarized, cells use asymmetric cue(s) to trigger local, self-reinforcing changes in the distribution of polarity regulators. The sperm-donated centrosome functions as such an asymmetric cue in *C. elegans* zygotes, but the mechanisms involved are poorly understood. Here we show that one mechanism employed by the centrosome involves a direct interaction between microtubules and the polarity regulator PAR-2. PAR-2 is a RING domain protein that competes with the conserved polarity regulators PAR-3, PAR-6 and PKC-3 for access to the cell periphery. Before polarization, PKC-3 phosphorylates PAR-2, keeping PAR-2 in the cytoplasm. We have found that PAR-2 binds microtubules directly. When bound to microtubules, PAR-2 is protected from phosphorylation by PKC-3. Microtubule-binding is essential for PAR-2 to overcome cortical exclusion by PKC-3, localize on the cortex nearest the sperm centrosome, and polarize the zygote. PKC-3 homologs have been implicated in cortical exclusion of polarity regulators in several cell types. Our findings provide a simple model for how centrosome-nucleated microtubules can break symmetry by providing local protection against a global barrier imposed by PKC-3.

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