CDB SEMINAR

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Wednesday, October 19, 2011

16:00~17:00 A7F Seminar Room

Breaking symmetry: polarization of the *C. elegans* zygote

Summary

A hallmark of polarized cells is the segregation of the PAR polarity regulators into asymmetric domains at the cell cortex. Antagonistic interactions involving two conserved kinases, atypical protein kinase C (aPKC) and PAR-1, have been implicated in polarity maintenance, but the mechanisms that initiate the formation of asymmetric PAR domains are not understood. Here, we describe one pathway used by the sperm-donated centrosome to polarize the PAR proteins in Caenorhabditis elegans zygotes.

Before polarization, cortical aPKC excludes PAR-1 kinase and its binding partner PAR-2 by phosphorylation. During symmetry breaking, microtubules nucleated by the centrosome locally protect PAR-2 from phosphorylation by aPKC, allowing PAR-2 and PAR-1 to access the cortex nearest the centrosome.

Cortical PAR-1 phosphorylates PAR-3, causing the PAR-3/aPKC complex to leave the cortex. Our findings illustrate how microtubules, independent of actin dynamics, stimulate the self-organization of PAR proteins by providing local protection against a global barrier imposed by aPKC.

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