

CDB SEMINAR

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Cell polarity regulator Pard6b is essential for trophectoderm formation in the preimplantation mouse embryo

In mouse preimplantation development, the first cell lineages to be established are the trophectoderm (TE) and inner cell mass. TE possesses epithelial features, including apical-basal cell polarity and intercellular junctions, which are crucial to generate a fluid-filled cavity in the blastocyst. Homologs of the partitioning defective (Par) genes are critical regulators of cell polarity. However, their roles in regulating TE differentiation and blastocyst formation are not known. Here, the role of Pard6b, a homolog of Par6 gene and a component of the PAR-aPKC complex, was investigated. Pard6b expression was down-regulated by microinjecting RNA interference construct into zygotes. Pard6b-knockdown embryos cleaved and compacted normally but failed to form the blastocyst cavity. The cavitation failure is likely due to defective intercellular junctions, as Pard6b knockdown caused abnormal distribution of ZO-1 tight junction (TJ) protein and interfered with cavitation in chimeras containing cells from normal embryo. Defective TJ formation may be due to abnormal cell polarization, as the apical localization of aPKC was absent in Pard6b-knockdown embryos. Pard6b knockdown also diminished the expression of Cdx2, a TE-lineage transcription factor, in the outer cells. Tead4, a transcriptional activator that is required for Cdx2 expression and cavity formation, was not essential for the transcription of *Pard6b*. Taken together, Pard6b is necessary for blastocyst morphogenesis, particularly the development of TE-specific features, namely the apical-basal cell polarity, formation of TJ, and up-regulated expression of Cdx2.

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