

POSTER PRESENTATION

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PI3K class II α : a novel regulator of vesicular trafficking at the base of the primary cilium

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The class II phosphoinositide 3-kinase PI3K-C2 α is a protein of the early endocytic compartment and the trans-Golgi network. It produces PtdIns-3-P and is characterized by a clathrin-binding site which confers to this enzyme an important role in modulating clathrin distribution and activity in the cell. However, implication of PI3K-C2 α in the primary cilium biology has never been described. Through the generation of a *Pik3c2a* knock-out mouse strain, we discovered that PI3K-C2 α was fundamental during embryonic development and that its loss conferred features of ciliopathy. In particular, homozygous mutant embryos died at midgestation and displayed laterality defects and impaired Hedgehog signaling, while heterozygous adults showed renal cysts susceptibility after kidney injury. Cilia of *Pik3c2a* deficient embryos were shorter and swollen and displayed a defect in accumulating Smo and Polycystin-2. In primary mouse embryonic fibroblasts, PI3K-C2 α was highly enriched on vesicles at the basal body of primary cilia. In *Pik3c2a*-deficient cells, absence of the protein specifically caused a reduction of vesicular trafficking at the cilium base, suggesting that PI3K-C2 α , through its ability to recruit clathrin and produce PtdIns-3-P, is required for the correct exchange of structural proteins and signaling molecules between the cilium compartment and the cytoplasm. Thus, our data indicate PI3K-C2 α as a novel regulator of vesicular trafficking at the base of the primary cilium.

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