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The role of Rabconnectin3a in cilia length regulation

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Background

Using the zebrafish mutant for the *deltaD* gene (*dld*$^{-/-}$), it was shown the involvement of Notch signaling in the control of cilia length in the cells of the fish laterality organ (Kupffer's Vesicle, KV) [1]. Further research based on KV specific microarray screening allowed the discovery of several genes with differential expression. Of these, 23% were associated with ciliogenesis and upon analysis, many proved to be involved in cellular trafficking.

Rabconnectin3a or *rbcn3a* was strongly downregulated in *dld*$^{-/-}$ KV cells. Homologs of this gene have been associated with Notch signaling in Drosophila and mammalian cells through the regulation of the V-ATPase activity [2,3]. *Rbcn3a* had also been associated with vesicular acidification in zebrafish hair cells [4] and with vesicular endocytosis and maturation in zebrafish neural crest migration [5].

Objective

We investigated the role of Rbcn3a in cilia length regulation.

Methods

We used a Morpholino against *rbcn3a* and fluorescent confocal imaging to explore cilia length. Furthermore we observed the consequences of reduced Rbcn3a in organ *situs* by ISH. We also performed rescue experiments by injecting *rbcn3a* full length mRNA at 1-cell stage *dld*$^{-/-}$ KO mutants.

Results

We showed that the downregulation of *rbcn3a* negatively regulates cilia length and that this can be rescued by *rbcn3a* overexpression in *dld*$^{-/-}$ embryos.

Conclusion:

The ciliary phenotype in *dld*$^{-/-}$ mutants is partially due to the downregulation of *rbcn3a*. Our hypothesis is that a generalized decrease in endocytic acidification, by deregulating the V-ATPase activity, results in shorter cilia.

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