

POSTER PRESENTATION

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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*<sup>-/-</sup>), 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*<sup>-/-</sup> 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*<sup>-/-</sup> 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*<sup>-/-</sup> embryos.

## Conclusion:

The ciliary phenotype in *dld*<sup>-/-</sup> 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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## References

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