Singapore Developmental Biology Club

SEMINAR ANNOUNCEMENT

23 May 2012, Wednesday
Aspiration Theatrette, Level 2M, Matrix, Biopolis
5:45PM – 6:45PM

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Seminar Title: Piecing together the squint puzzle.

The earliest steps of axis formation in zebrafish are thought to be regulated by maternal factors that activate Wnt/β-catenin signaling to specify the dorsal axis. We previously showed that asymmetric localization of maternal transcripts of the conserved zebrafish TGF-β factor, Squint (Sqt/Ndr1), in 4-cell stage embryos predicts dorsal, preceding nuclear accumulation of β-catenin. Cell ablations and antisense oligonucleotides targetting Sqt lead to dorsal deficiencies, suggesting that localized maternal sqt functions in dorsal specification. However, due to discrepancies between the genetic *nodal* mutants and the ablations/antisense knock-down results, maternal sqt function was debated. We find that sqt RNA has activity independent of Sqt protein in dorsal specification. Surprisingly, over-expression of mutant/non-coding sqt RNA and the sqt 3' UTR, leads to ectopic nuclear b-Catenin accumulation and expands dorsal gene expression. Dorsal activity of sqt RNA requires Wnt/β-catenin but not Oep-dependent Nodal signaling, explaining the discrepancy between the *nodal* mutants and morpholino phenotypes. Finally, we revisited the activity of the sqt morpholinos, and show that in addition to blocking Sqt translation/splicing, they also disrupt sqt RNA localization. Depletion of maternal sqt RNA abolishes nuclear β-Catenin, providing the mechanism for the loss of dorsal in the morphants, and places maternal sqt RNA activity upstream of β-Catenin. Remarkably, loss of early dorsal gene expression can be rescued by sqt 3' UTR sequences. Our findings identify new non-coding functions for the *nodal* genes, and support a model wherein sqt RNA acts as a scaffold to bind and deliver/sequester maternal factors to future embryonic dorsal.

No registration required.
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