and xanthophores. Genetics will now add to the strengths of the *Xenopus* model system for understanding vertebrate development.

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A G-protein-coupled receptor (Xflop) controls cortical actin assembly via EP-cadherin complex in early *Xenopus* embryos

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Actin cytoskeleton is indispensible for many aspects of cellular functions. The mechanisms that control actin polymerization and reorganization have been intensively studied in tissue cultures. However, little is known how cortical actin is assembled in early vertebrate embryos. We have shown previously that cortical actin is essential for maintaining the cytoarchitecture of early *Xenopus* embryos. We identified a G-protein-coupled receptor Xflop, related to mammalian GPR4 is both necessary and sufficient for cortical actin assembly, suggesting the intercellular signaling plays a critical role in regulating cortical actin assembly. However the mechanism by which Xflop controls cortical actin assembly remains unclear. Here we present evidence that EP-cadherin, a maternally expressed classical Cadherin is required for Xflop function. Antisense oligo DNA mediated maternal mRNA knockdown showed that EP-cadherin is necessary for dense cortical actin assembly. Both gain of function and loss of function analyses demonstrate that Xflop regulates the level of EP-cadherin on cell membrane. We propose that Xflop controls cortical actin assembly through regulating the cadherin complex formation on cell membrane in early *Xenopus* embryos.

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An exploration of the nuclear localization of intersectin in *Xenopus laevis*

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Intersectin (ITSN) is a protein with two isoforms generated by an alternative splicing event. The shorter isoform (ITSN-S) contains two Eps15 homology (EH) domains, a central coiled-coil domain, and five Src homology 3 (SH3) domains. The longer isoform (ITSN-L) contains an additional Dbl homology (DH) domain, a Pleckstrin homology (PH) domain, and a calcium-binding (C2) domain. ITSN appears to act as an intermediary between endocytosis, exocytosis and mitogenic processes. Software analysis of the ITSN amino acid sequence using PSORT II and PredictNLS algorithms suggests that the protein may have as many as 7 nuclear localization sequences (NLSs). Confocal immunofluorescent microscopy revealed that during normal *X. laevis* development, ITSN shifts from the cytoplasm to the nucleus at stage 8. ITSN then returns to the cytoplasm at stage 20. Semi-quantitative real-time RT-PCR showed a dramatic increase at stage 15 in expression of both isoforms, followed by a decrease in expression at stage 19.

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Lunatic fringe plays multiple, distinct roles during vertebrate segmentation

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Somites are the most obvious example of segmentation in developing vertebrate embryos. The regulation of somitogenesis is complex, and the Notch signaling pathway plays multiple roles during segmentation. Notch signaling is involved in both the segmentation clock that regulates somitogenesis and in rostro-caudal (R/C) patterning of the somites. In vivo analysis of Notch in the segmentation clock has been complicated by these multiple roles. Lunatic fringe (*Lfng*) encodes a glycosyltransferase that modulates Notch signaling and which appears to play roles in both clock function and R/C somite patterning. The enhancer FCE1 (fringe clock element 1) is required for cyclic expression of *Lfng* transgenes. To assess the role of cyclic *Lfng* expression in the clock and in somitogenesis FCE1 was removed from the endogenous *Lfng* locus. This established an allele ($Lfng^{\Delta FCE1}$) that lacks cyclic *Lfng* expression within the segmentation clock, but that still expresses *Lfng* during R/C patterning of the somites. Homozygous $Lfng^{\Delta FCE1}$ mice exhibit segmentation phenotypes that are significantly less severe than those of *Lfng* null animals. The $Lfng^{\Delta FCEl}$ mice exhibit altered expression of Hes7 and other oscillating genes, suggesting that loss of *Lfng* interferes with clock function. In contrast, $Lfng^{\Delta FCEl}$ mice demonstrate relatively normal R/C somite patterning compared to *Lfng* null animals. This strongly supports our hypothesis that *Lfng* plays multiple, critical roles in somitogenesis, and that *Lfng* null mice have a complex phenotype resulting from altered Notch signaling in both the clock and R/C patterning.

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