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proteins, but causes the formation of misoriented and non-polar hair bundles, indicating a role for ciliary genes in intrinsic cell polarization downstream of asymmetric PCP complexes. We further found that basal body positioning correlates with the polarity or loss of polarity of hair bundles and that basal body configuration appears to be affected in ciliary mutants. Strikingly, similar defects in the basal body and in the loss of intrinsic polarity were found in mouse mutants with defective Usher genes Usher proteins make up the machinery for the formation of polarized hair bundles. We have also detected a genetic interaction between Usher gene PCDH15 and ciliary gene IFT88 in basal body location and hair bundle morphogenesis. Together, our results suggest that ciliary genes act with Usher genes to configure the basal body to direct the formation of intrinsically polarized hair bundles.

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**Program/Abstract # 214**
**Wnt/planar cell polarity signaling controls endoderm cell rearrangements during the morphogenesis of the primitive gut tube**

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To generate normal functional anatomy in the digestive tract, the primitive gut tube (PGT) must undergo dramatic elongation and form a lumen lined by a single layer of polarized digestive epithelium. In *Xenopus* embryos, endoderm cells in the core of the PGT radially intercalate during gut elongation, but the morphogenetic mechanisms underlying these rearrangements are unknown. We previously showed that inhibition of Rho/ROCK/Myosin II activity prevents endoderm intercalation and consequently perturbs both gut elongation and digestive epithelial morphogenesis. Here we show that gut morphogenesis is governed by Wnt/PCP signaling. Gut-targeted expression of a dominant negative form of *Wnt11*, or an allele of *Disheveled* (*Dsh*) that specifically inhibits noncanonical Wnt signaling, results in shortened and malrotated gut tubes. Wnt11- or Dsh-deficient endoderm cells lose their polarized morphology and fail to properly intercalate. Moreover, exposure of late stage embryos to small molecule inhibitors of Rac or JNK perturbs the normal cell shape and adhesion patterns necessary for endoderm intercalation, and consequently induces severe defects in gut elongation and digestive epithelial morphogenesis. Our results suggest that the morphogenetic events driving tissue elongation in the PGT are mechanistically analogous to those that function during gastrulation. We propose that different Wnt/PCP signaling components control distinct endoderm cell properties and behaviors to coordinate the development of an epithelial lining with tubular tissue elongation in the PGT.

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**Program/Abstract # 215**
**Wnt5b/Ryk signaling mediates polarized cell movement in zebrafish**

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The Wnt signaling network plays an important role in patterning and morphogenesis. Wnt pathways via the seven-transmembrane receptor Frizzled (Fzd) regulate convergent extension (CE) movement in vertebrate embryos. Wnt has also been shown to signal through Ryk, an atypical receptor tyrosine kinase, to mediate axon guidance. However, the molecular mechanism of Wnt/Ryk signaling and its role outside the nervous system are less well characterized. Here we report a role of Wnt5b/Ryk signaling in zebrafish gastrulation. We combined gene knockdown and transplantation assays to show that Wnt5b/Ryk signaling is required for the CE movement during zebrafish gastrulation. We further demonstrate that Ryk internalizes into caveolin-coated endocytic vesicles upon Wnt5b stimulation and promotes polarized filapodia in migrating cells. While Wnt5b signaling through Ryk is independent of nuclear beta-catenin function, Ryk deficiency partially blocks Wnt5b-induced Disheveled (Dvl) turnover and Ryk overexpression activates intracellular calcium release, suggesting that Wnt5b/Ryk signaling regulates polarity effectors in common noncanonical Wnt pathways. In contrast to its role as a permissive cue in Wnt/Fzd signaling, Wnt5b transduces directional signals to Ryk-expressing cells. Our findings indicate that non-canonical Wnt ligands can modulate polarized cell movement in vertebrates by two mechanisms: a known mechanism by which activation of the core components of planar cell polarity (PCP) pathway through Fzd leads to establishment of polarity framework; and a novel mechanism by which Ryk signaling provides directional information for cell migration.

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**Program/Abstract # 216**
**The PCP effector Fritz governs microtubule assembly and ciliogenesis in vertebrate multi-ciliated cells**

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Cilia are microtubule-based organelles protruding from nearly all vertebrate cells. Several core components of the PCP signaling are essential for ciliogenesis. Fritz is an effector in the PCP signaling. Here, we examined its function in *Xenopus laevis* using antisense morpholino-oligonucleotides (MOs). Confocal microscopy and scanning electron microscopy revealed that axonemes on multi-ciliated cells of Fritz morphants were far shorter and fewer in number, as compared to controls. We observed that loss of Fritz results in the accumulation of apical cytoplasmic microtubules including polyglutamylated tubulins. Polyglutamylation is important for cilia assembly and function. A dramatic increase in polyglutamylated tubulin signal in the apical cytoplasm of Fritz morphant multi-ciliated cells indicates that the ectopic microtubule assembly in Fritz morphant is highly glutamylated. Next, we identified the CCT as an interacting partner of Fritz. CCT is a chaperonin and has been implicated in ciliogenesis. We generated GFP- or myc-tagged CCT subunit constructs and found that the GFP- or myc-tagged CCTa and CCTe were localized in punctate structures along the ciliary axonemes of multi-ciliated cells. We observed that loss of Fritz results in the accumulation of CCT at the apical cytoplasm in multi-ciliated cells. We suggest that by deregulating the localization or function of CCT in Fritz morphants, the turnover of microtubules may slow in the apical cytoplasm of multi-ciliated cells. As a result of reduced turnover, these microtubules may be longer-lived and thus acquire a higher concentration of polyglutamylation.

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**Program/Abstract # 217**
**Specific cellular behaviors regulate in LR asymmetric heart looping**

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