thought to provide the motive force for epiboly, are defective in MZ*eomesa* mutant embryos. Prior to doming, the yolk microtubules are abnormally bundled, leaving large regions entirely devoid of microtubules. Importantly, both the doming delay and the yolk microtubule distribution are rescued by injection of *eomesa* mRNA into embryos at the 1-cell stage. In addition to the yolk defects, the deep cells of the blastoderm display abnormal morphologies. The deep cells are more tightly packed and exhibit more bleb-like protrusions than cells in control embryos. Transplantation studies are being conducted to determine if the deep cell defects are cell autonomous or non-cell autonomous. Our continued investigation of the basis of the defects in MZ*eomesa* mutant embryos should provide new insights into the molecular control of epiboly. Eomesodermin has also been implicated in gastrulation movements in both *Xenopus* and mice, pointing to a conserved role in regulating morphogenesis.

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Program/Abstract # 114
The cytoplasmic tyrosine kinase Arg regulates Xenopus gastrulation via the adaptor protein CrkII
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Coordinated cell movements during vertebrate gastrulation are crucial for correct placement of embryonic tissues along body axes and are controlled by multiple signals. While non-canonical Wnt pathway is shown to regulate cell polarity and directional cell behaviors via the cytoplasmic protein Dishevelled, the mechanisms used by receptor tyrosine kinases, such as PDGFR, FGFR and ErbBs, to modulate gastrulation are less understood. Here, we show that the actin-binding cytoplasmic tyrosine kinase Arg modulates cell movements during Xenopus gastrulation. Arg was expressed in dorsal tissues at the onset of gastrulation, and both gain- and loss-of-function of Arg disrupted gastrulation movements and led to defective frog tadpoles. Overexpression of Arg inhibited head mesoderm migration effectively, while reduction of Arg by specific antisense morpholino oligos caused aberrant head mesoderm migration, resulting in reduced migratory distance and increased cell dissociation. Both overexpression and depletion of Arg also affected convergent extension movements. The regulation of Xenopus gastrulation by Arg required an intact kinase domain, but the actin-binding motif could be dispensed. Arg controlled phosphorylation of endogenous CrkII, an adaptor protein involved in activation of Rho family GTPases and actin reorganization. Our data thus imply that Arg may be an essential mediator of receptor tyrosine kinases during gastrulation and can modulate cell movements via phosphorylation of an important effector CrkII.

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Program/Abstract # 115
Fritz regulates the membrane stability mediated by septins dynamics during Convergent Extension in Xenopus embryo
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The Planar Cell Polarity (PCP) pathway is acritical regulator for cell behaviors during development. Although there isaccumulating data showing that core PCP proteins are necessary for cellpolarity, much less is known about how individual cells respond to PCP signaland change their behavior. Fritz is one of the PCP effector proteins, whichacts downstream of the core PCP proteins to control specific processes in *Drosophila*. We investigated the function of Fritz to unveil the processbetween core PCP and the changing of cell behavior during Convergent Extensionin *Xenopus* embryos. We found that Fritz wasexpressed in the dorsal mesoderm, and GFP fused Fritz localized at the cellmembrane. Inhibition of Fritz function using antisensemorpolino-oligonucleotides (MO) lead to the gastrulation defects and abnormalcell membrane dynamics (undulation and appearance of blebs). We found thatFritz physically interacted with septins, cytoskeletal elements that providecortical rigidity. Septins–MOs caused blastopore closure and cell behaviordefects similar to Fritz-MO. Also, GFP-fused septins localized in or near thecell membrane depending on Fritz. Importantly, the cell elongation wasattenuated in all these morphants, but the medio-lateral polarity wasmaintained as in wild type embryos. From these results, we conclude that Fritzregulates septins, as the executants of the PCP pathway to control the membranestability and cell elongation during Convergent Extension.

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Program/Abstract # 116
Serotonin and Wnt signaling are required for morphogenesis of the gastrocoel roof plate epithelium, the site of symmetry breakage in the frog embryo
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Organ laterality in vertebrates results from asymmetric signaling in the embryo. Symmetry breakage in fish, amphibian and mammalian embryos depends on cilia-driven flow of extracellular fluid during neurulation. In *Xenopus* a functionally relevant asymmetry of serotonin localization was postulated already at the 16-cell stage. We report the role of serotonin signaling in the context of flow. Flow, and consequently asymmetry, were lost in embryos in which serotonin signaling was downregulated, either in receptor morphants or by sequestration of extracellular serotonin upon expression of a secreted serotonin-binding domain. Serotonin signaling was required for the specification of the ciliated gastrocoel roof plate (GRP) epithelium during gastrulation, the site of leftward flow. A second pathway involved in this process is canonical Wnt signaling, as shown by flow and laterality defects in receptor (fz8) morphants. Our data suggest that serotonin acts as a permissive and Wnt as an instructive signal to specify the GRP.

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Program/Abstract # 117
Development of swimming regulation systems in sea urchin: From blastulae to larvae
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In sea urchin embryos, motile cilia are evident from the blastula stage, and rotatory movement by embryos can be observed in the fertilization envelope. Serotonin plays a role in the regulation of the beating of larval cilia. The serotonergic nervous system is yet to appear in blastulae. Thus, the regulation system of cilia of blastulae is unknown. Nevertheless, the swimming behavior of blastulae is organized to a considerable degree. The beating of cilia is regulated also by dopamine (DA) in invertebrates and