have distinctly different morphologies but all three execute the epiboly movement. *poky* is a maternal effect mutant that displays delayed epiboly and breakdown of the blastoderm associated with defective EVL differentiation. The yolk cell cytoskeleton is disrupted with large voids observable in the cortical cytoskeleton. In strongly affected embryos the blastoderm becomes clear and disperses. In these strongly affected embryos, the EVL cells do not slow down their cell division rate, they do not adopt a normal flattened morphology and they fail to express the intermediate filament genes *krt4* and *cyt1*. However *poky* mutant EVL cells do express the tight junction marker ZO1 but it does not display robust localization to the cell junctions. *poky* mutant cells display some aspects of EVL identity but fail to differentiate fully. We have identified the gene encoding *poky*, the zebrafish homolog of Conserved Helixloophelix Ubiquitous Kinase (CHUK/IKK1) a component of the NFkB regulatory pathway. CHUK is a kinase with numerous identified targets including the IkB proteins leading to NFkB activation. As in *poky* EVL cells, the CHUK mutant mouse keratinocytes fail to express specific intermediate filament genes and fail to exit the cell cycle. We hypothesize that the differentiation of both the zebrafish EVL and mouse skin is dependent on CHUK and that similar pathways regulate differentiation and function of these essential organs.

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**Program/Abstract # 222**
**Cadherin function: Right place, right time**
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Transmembrane cadherins are calcium-dependent intercellular adhesion molecules. Recent studies from our lab using the *Xenopus* blastula identified that C-cadherin plays a central role in regulating the actin cytoskeleton during early stages of development. We demonstrated that membrane presentation of C-cadherin is the rate-limiting step to organize actin and the cadherin juxta membrane domain binding protein p120 catenin is also necessary for this activity (Nandadasa, Tao et al., 2007). However, the roles of actin assembly on transmembrane cadherins during development are not fully understood. Using the developing ectoderm of the *Xenopus* embryo as a model, we show that F-actin assembly is a primary function of both N-cadherin in the neural ectoderm, and E-cadherin in the non-neural (epidermal) ectoderm, and each cadherin is essential for the characteristic morphogenetic movements of these two tissues. However, depletion of N-cadherin and E-cadherin did not cause dissociation in these tissues at the neurula stage, probably due to the expression of C-cadherin in each tissue. Depletion of each of these cadherins is not rescued by the other, nor by the expression of C-cadherin, which is expressed in both tissues (Nandadasa et al., 2009). One possible reason for this is that each cadherin is expressed in a different domain of the cell membrane. These data indicate the combinatorial nature of cadherin function, the fact that N- and E-cadherin play primary roles in F-actin assembly, in addition to roles in cell adhesion, and that this function is specific to individual cadherins.

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**Program/Abstract # 223**
**Cell adhesion and the cell biology of gastrulation in the cnidarian, *Nematostella vectensis***
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Gastrulation is a central event in metazoan development and the first morphogenetic process in the embryo, resulting in the formation of a multilayered embryo from a monolayered blastula. Adhesive mechanisms, both cellcell and cellextracellular matrix, are intimately involved in this process. Modulation of adhesive complexes could therefore be seen as a central component in the molecular control of morphogenesis, the translation of information encoded within the genome into organismal form. Understanding how morphogenesis is controlled in early-branching metazoans will help clarify the evolution of morphogenetic mechanisms. To this end we have examined the cell biology underlying gastrulation in the cnidarian, *Nematostella vectensis*, a valuable context in which to study morphogenesis in an early-branching taxon. Gastrulation in *Nematostella* occurs through invagination. The cells adjacent to the blastopore adopt extreme bottle-like morphologies as they constrict their apical surfaces, but retain projections that extend to the archenteron as they zip up against the basal surface of the ectodermal cells. In silico screening of the *Nematostella* genome has revealed a number of cell junction components that may be involved in this process, as well as other genes potentially involved in cellular behaviors required for gastrulation. Discovery of the molecular nature of morphogenesis in early-branching groups such as cnidarians, coupled with comparisons across the metazoa, promises to reveal the ways evolution has generated the myriad forms seen in the animal kingdom.

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**Program/Abstract # 224**
**Cells use different cell polarity and fate regulators to control the same cytoskeletal mechanism in *C. elegans* gastrulation**
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One of the most important processes in morphogenesis is gastrulation, the process by which germ layers are positioned in an embryo. *C. elegans* is unusual among model animal systems in that gastrulation involves sequential internalizations of small numbers of cells. The first cells to internalize are the endodermal (E) cells, which must be specified properly in order to internalize at the right time. In addition, these cells are polarized by the PAR proteins, required for myosin to accumulate at the apical surface. E cells then undergo a myosin-driven apical constriction, activated by myosin regulatory light chain phosphorylation, which is dependent on Wnt signaling. The mesodermal (MS) cells begin internalizing when there are 16 MS cells, followed by the germline cells; the mechanisms behind these internalizations are not well characterized. We show that active myosin accumulates at the apical surface of MS cells during their internalization and that myosin accumulates at the apical surface of internalizing germline cells. While the basic mechanism behind the E cells, MS cells, and germline internalizations may be similar, it requires their distinct cell fate specifications. In addition, we found that the polarity regulators that are required for E cells to internalize do not appear to be required for MS cells to internalize. We are currently exploring which cell fate transcription factors and polarity regulators are acting upstream of gastrulation in these other lineages.

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**Program/Abstract # 225**
**MAGI-1 modulates the function of the *C. elegans* cadherin-catenin complex during morphogenesis**
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