occurred. Overexpression of *TBX22* caused a striking decrease in proliferation but did not change the level of apoptosis. Furthermore we identified two targets of *TBX22* that could be mediating the phenotype, *DLX5* and *MSX2*. We have therefore demonstrated novel functions for *TBX22*, a gene that causes some forms of human orofacial clefting.

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Program/Abstract # 494
A point mutation in Arid1a reveals an essential role for this SWI/SNF subunit in extraembryonic blood vessel and trophoblast development

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Arid1a is a signature subunit of the mammalian SWI/SNF ATP-dependent chromatin remodeling complex. We performed an ENU mutagenesis screen for Arid1a coding mutations and generated mice carrying a valine-to-glycine point mutation in the ARID DNA binding domain of Arid1a. Although mutant protein is expressed near wild-type levels and capable of interacting with its catalytic subunit, Brg1, it appears to display reduced DNA binding capacities in vitro. Homozygous mutant embryos undergo development arrest by E10.5 and exhibit defects in the trophoblast placenta and extraembryonic vasculature, including a compacted labyrinth layer and reduced vascular branching. These data suggest the ARID domain of Arid1a is essential for development.

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Program/Abstract # 495
The role of Friend of GATA in primitive red blood cell development

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The transcription factor GATA-1 and its cofactor Friend of GATA (FOG) are required to promote embryonic red blood cell (RBC) development in mice. In contrast, the current model in *Xenopus*, based on overexpression studies, predicts that FOG inhibits RBC development by recruiting the transcriptional co-repressor C-terminal Binding Protein (CtBP). To resolve these seemingly contradictory findings, we have used morpholinos to perform a loss-of-function study in frogs. We find that in *Xenopus*, as in mice, FOG is in fact required for RBC development. Specifically, targeted injection of FOG morpholinos into the ventral blood-forming mesoderm of 8-cell *Xenopus* embryos results in a dose-dependent loss of *globin* expression at the tailbud stage. In addition, we find that overexpression of both wildtype FOG and mutant FOG isoforms that either lack known repressor binding domains, or harbor mutations at key GATA-interaction residues, also result in loss of blood. Together, these studies suggest that FOG is required for RBC development and that loss of blood seen with FOG overexpression is likely due to a dominant-interfering effect by which excess FOG sequesters other co-factors required for RBC development away from endogenous target promoters. Specific domains of FOG required for RBC development *in vivo* have not yet been elucidated. We are currently asking whether various FOG mutant constructs can rescue RBC formation in FOG morphants. This will allow us to determine which functional domains of FOG are required for normal erythropoiesis, and may suggest novel binding partners that are important for FOG's role during RBC development.

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Program/Abstract # 496
Stacked expression of Hand2 and Dlx mediates signaling from Edn1 to produce discrete pharyngeal arch patterning domains

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Recent studies have suggested that Endothelin1 (Edn1) acts in a dose dependent fashion to pattern skeleton from the first two pharyngeal arches into dorsal, intermediate, and ventral domains via its targets *hand2* and Dlx. We hypothesized that *hand2* expression defines the ventral domain, in part by repressing intermediate domain genes. We further hypothesized that Dlx genes pattern the intermediate domain. Third, we propose that the combined patterning from *hand2* and Dlx delineates ventral/intermediate domains from dorsal. Here we demonstrate that *hand2* is expressed next to *edn1*, and the expression of all Dlx genes extends dorsal to *hand2*. We provide evidence that *dlx3b*, *dlx4b*, and *dlx5a* are redundantly required for intermediate domain patterning. Furthermore we show that by 36 hpf, *dlx3b*, *dlx4a*, and *dlx4b* are specifically expressed in intermediate arch mesenchyme. Previous work demonstrated that *hand2* is required for ventral cartilage formation. We confirmed this with two alleles of *hand2*. We further show that in *hand2* mutants, *dlx3b*, *dlx4a*, and *dlx4b* expression expands into the ventral domain at 36hpf. Finally, when Dlx-MO is injected into *hand2* mutants, both ventral and intermediate defects are seen, and the ventral-most structures may acquire dorsal shape. Collectively our work suggests that the stacked expression of *hand2* and Dlx mediates signaling from Edn1 to generate ventral and intermediate domains with distinct identities separate from dorsal.

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Program/Abstract # 497
A follistatin-BMP7 feedback circuit controls taste papillae development and patterning in mouse tongue

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Interactions between epithelium and mesenchyme are thought to drive development and patterning of taste papillae, but the identities of the mesenchymal signals are unknown. Using mouse genetics, we show that *Fst*, which is expressed in tongue mesenchyme during development, controls these processes in both anterior (normally gustatory) and posterior (normally non-gustatory) lingual epithelium. In anterior $Fst^{-/-}$ tongue there are increased numbers of *Sox2*+ taste progenitors, with fungiform papillae of abnormal size and spacing. In posterior $Fst^{-/-}$ tongue, ectopic *Sox2*+ epithelial domains develop and non-gustatory filiform papillae are absent. Increased *Bmp7* expression is evident in regions of ectopic *Sox2*+ progenitors, and