a necessary step in the formation of a mature typically vertebrate frog pancreas.

doi:10.1016/j.ydbio.2008.05.457

Program/Abstract # 477
Roles of Bmp, Fgf and Wnt signaling in liver formation and recovery in zebrafish embryos
Shin Donghun, Didier Y. Stainier
Department of Biochemistry and Biophysics, UCSF, San Francisco, CA, USA

Bmp, Fgf and Wnt have been implicated in liver specification, differentiation, and proliferation in several systems including zebrafish. Liver specification and subsequent differentiation were blocked in embryos following a block in Bmp or Fgf signaling and in *wnt2bb* mutant embryos. However and surprisingly, the liver eventually recovered in most of these embryos, suggesting that endodermal cells remain competent to give rise to the liver. To understand the process of liver recovery, we blocked Bmp, Fgf or Wnt signaling in the *wnt2bb* mutant background. The inhibition of Wnt signaling using a transgenic line overexpressing Dkk1, an inhibitor of the canonical Wnt signaling pathway, under the heat-shock promoter, completely blocked liver recovery in *wnt2bb* mutant embryos. In addition, the inhibition of Bmp signaling using a transgenic line overexpressing a dominant-negative Bmp receptor also blocked liver recovery. In contrast, the inhibition of Fgf signaling using SU5402, an Fgf inhibitor, promoted liver recovery in *wnt2bb* mutant embryos. Furthermore, this inhibition also promoted liver recovery in *wnt2bb* mutant embryos with Bmp or Wnt signaling repressed. The knockdown of *fgf10* using morpholinos also promoted liver recovery in these embryos. Since it was recently reported that *fgf10* mutant embryos have defects in the extrahepatic duct and contain ectopic hepatocytes, we hypothesize that extrahepatic ductal progenitor cells can give rise to duct and liver, and that Fgf signaling mediated by Fgf10 represses these cells to become liver, thereby allowing them to become duct.

doi:10.1016/j.ydbio.2008.05.458

Program/Abstract # 478
Zebrafish homologue of FKBP65 plays a role in intestinal smooth muscle differentiation
Tanveer Akhtar, Rich Yerry, Kenneth N. Wallace
Department of Biology, Clarkson University, Potsdam, NY USA

Zebrafish intestinal smooth muscle differentiates from a thin layer of mesenchymal cells surrounding the developing epithelial layer. As with other vertebrates, circular is first to differentiate early on the third day of embryogenesis followed by longitudinal smooth muscle late on the third day. Few regulatory pathways have been identified which control intestinal smooth muscle differentiation. FKBP65 has previously been shown to regulate differentiation of avian intestinal smooth muscle. We have begun to characterize the role of the zebrafish homologue of *fkbp65* gene in intestinal smooth muscle development. We find the zebrafish *fkbp65* is expressed in the intestinal mesenchyme during the third day of embryogenesis at a time when smooth muscle is developing. We find that zebrafish *fkbp65* expression is present at the correct time and place to play a role in intestinal smooth muscle development. To address the role of FKBP65 in intestinal smooth muscle differentiation we used both FK506, a general inhibitor of the class of FKBPs, and a 5′ morpholino to the gene. We find that injection of either theFK506 or morpholino inhibits differentiation of smooth muscle primarily in the anterior intestine. These results suggest that the zebrafish homologue of FKBP65 plays a similar role in smooth muscle differentiation to the avian system. Inhibition of smooth muscle differentiation in only the anterior intestine suggests that there may be another homologue that plays a role in posterior smooth muscle differentiation.

doi:10.1016/j.ydbio.2008.05.459

Program/Abstract # 479
Effect of thyroid hormone on gut development in a direct developing frog
Srikanth Singamsetty, Richard P. Elinson
Department of Biological Sciences, Duquesne University, Pittsburgh, PA, USA

During metamorphosis of *Xenopus laevis*, thyroid hormone (TH) induces apoptosis of the tadpole gut and development of the adult gut. *Eleutherodactylus coqui*, a direct developing frog, lacks a tadpole. Its embryonic gut is a miniature adult form with a mass of large yolk-rich cells attached to the intestine. The yolk-rich cells provide nutrition but do not contribute to the adult gut (Buchholz et al., 2007 Dev Dyn 236:1259–1272). We asked whether TH is involved in *E. coqui* gut development as it is in *X. laevis*. The expression of TH receptors, *EcTRα* and *EcTRβ*, was detected in the developing gut by RT-PCR, indicating a TH role. To test a TH requirement, endogenous TH synthesis was inhibited with methimazole. The diameter of the methimazole treated gut was reduced, and the submucosa and muscularis layers were significantly smaller than those of the untreated embryos. Despite these gross histological differences, RT-PCR indicated no obvious differences in expression of *EcSox17*, *EcShh*, *EcBMP4* and *EcCad* between methimazole treated and untreated embryos. Embryos treated with methimazole failed to utilize their yolky tissue, but survived for weeks without any further development. When T_3, the active form of TH was added along with methimazole, the gut resembled that of controls. There were, however, many more cells in guts from T_3-treated embryos compared to untreated ones and from untreated embryos compared to methimazole treated ones. These results suggest that a major role of TH in the development of the *E. coqui* gut is to stimulate cell proliferation of gut tissue and utilization of yolk.

doi:10.1016/j.ydbio.2008.05.460

Program/Abstract # 480
Alpha 2-macroglobulin regulation of axial and gut morphogenesis in *Xenopus laevis*
Liliam L. Pineda Salgado, Daniel Kessler
Department of Cell and Develomental Biology. UPenn, Philadelphia, PA, USA

α2-macroglobulin is a major serum protein which inhibits protease activity. In *Xenopus laevis*, two α2M genes, Endodermin (Edd) and Panza, have been isolated. Edd is expressed in endoderm and dorsal mesoderm cells and with the onset of gut coiling expression is restricted to the liver. In contrast, Panza is expressed in the dorsal domain of the gut endoderm. During gut coiling Panza expression is initiated and maintained in the liver. The overlapping expression of Edd and