thought to involve secondary lumen formation in deep epithelial layers as well as epithelial rearrangement by radial intercalation. However, we now demonstrate that the intestinal epithelium between E12.5 and E14.5 is pseudostratified and that epithelial girth is generated by apicobasal elongation through actinomyosin- and microtubule-dependent mechanisms. We also find that, while epithelial cells do not undergo radial intercalation, their nuclei move in the plane of the epithelium by interkinetic nuclear migration. Furthermore, using three-dimensional reconstruction, we find that luminal expansion occurs by extension from the primary lumen rather than by secondary lumen formation and we identify specialized cell mitotic events as drivers of luminal extension. In these events, the cytokinetic furrow is co-opted to form the extending apical luminal surface. Finally, we show that proper epithelial organization requires Shroom3, an actin binding protein. This revised model of intestinal organization has enormous implications for the interpretation of previous and future functional studies of the developing intestine.

doi:10.1016/j.ydbio.2011.05.162

Program/Abstract # 139
Rho1 GTPase controls *Drosophila* salivary gland lumen size by regulating the distribution of cortical F-actin and phosphorylated Moesin
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Generation and maintenance of proper lumen size are important for tubular organ function. We report on a novel role for the Rho1 small GTPase in control of lumen size in the *Drosophila* embryonic salivary gland. We demonstrate that Rho1 GTPase and Rho kinase are required for novel and distinct cellular processes that define salivary gland lumen size, such as cell flattening, apical domain elongation and cell rearrangement. We show that in salivary gland cells, Rho1-dependent distribution of cortical F-actin and phosphorylated Moesin is important for control of gland lumen size. In Rho1 mutant gland cells apical localization of F-actin and phosphorylated Moesin is enhanced. Rho1 functions with Ribbon, a BTB-domain containing transcription factor, in limiting apical phosphorylated Moesin during apical domain elongation and cell flattening. A role for the actin cytoskeleton in lumen size control is further supported by our findings that reducing polymerized actin levels with mutations in the *Drosophila* profilin homolog, chickadee, phenocopied the Rho1 lumen defect and promoting actin polymerization with mutations in twinstar, the *Drosophila* cofilin homolog, partly suppressed the lumen defect of Rho1 mutant embryos. We also demonstrate that the Formin protein, DAAM, regulates cortical actin distribution with Rho1 to control gland lumen size. Thus, our studies reveal a novel mechanism for controlling salivary gland lumen size, namely through Rho1-mediated distribution of the cortical F-actin cytoskeleton and phosphorylated Moesin and identify Ribbon and DAAM as novel co-regulators with Rho1 in control of gland lumen size.

doi:10.1016/j.ydbio.2011.05.163

Program/Abstract # 140
Patched1 is essential for nasal pit invagination in mouse
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Hedgehog (Hh) signalling plays a critical role in vertebrate face formation. To understand the role of the receptor Patched1 (PTCH1) in facial development, we are using the Wnt1–Cre to conditionally inactivate Ptch1 in neural crest cells. This results in constitutive, ligand-independent upregulation of the Hh pathway in the majority of mesenchymal cells in the face. We have shown for the first time that Ptch1 deletion drastically alters the shape of the nasal pit and disrupts its ability to invaginate. While neural crest cells are able to migrate and populate the developing nasal processes, they appear to form part of a loosely organised mesenchyme, unable to respond to FGF signals from the overlying epithelium and consequently fail to specify the lateral nasal process. We find that the structure of the overlying nasal pit epithelium is perturbed, evidenced by changes in cell–cell contacts and actin organisation. In summary, our analysis of Ptch1 conditional mutants has revealed unexpected changes in the shape and structure of cells of the nasal pit epithelium, which may explain why invagination is perturbed. These findings identify novel roles mediated by PTCH1 that are essential for correct morphogenesis of the nasal pit. Furthermore, they indicate that strict, spatio-temporal control of Hh signalling helps to define face shape in the embryo.

doi:10.1016/j.ydbio.2011.05.164

Program/Abstract # 141
V-ATPase-dependent ectodermal voltage and pH regionalization are required for Xenopus craniofacial morphogenesis
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Despite solid evidence for the importance of ion-based signaling during development, only Ca 2+-dependent signals have received extensive attention. Regulated ion flux is a fundamental aspect of physiology, and new reporting dyes promise to reveal the roles of other ions the way Fura and Fluo dyes have done for Ca 2+. Using voltage and pH reporters we have discovered a never-before-seen regionalization of the ectoderm, with ectodermal-cell sub-populations delimited by different membrane voltage and pH. Following a wave of hyperpolarization during gastrulation, patterns that match shape changes and gene expression domains of the developing face appear and evolve in the developing face; hyperpolarization marks folding epithelium including the neural tube, stomodeum and sensory placodes; both hyperpolarized and depolarized regions overlap domains of important head patterning genes. Around stage 20, localized regions of hyperpolarization form at various positions, expand, and disappear. Inhibiting H+-transport by the H+-V-ATPase causes abnormalities in: (a) the morphology of tadpole facial structures, especially the branchial arches and eyes; (b) voltage patterns; and (c) the ISH patterns of the genes sox9, pax8, slug, mitf, xfz3, otx2, and pax6. We conclude that this bioelectric signal has a role in the development of the face, thus it exemplifies an important, little-studied mechanism of developmental regulation. Understanding how bioelectrical signals intertwine with developmental signaling pathways will provide important insight into both differentiation and morphogenesis.

doi:10.1016/j.ydbio.2011.05.165

Program/Abstract # 142
Serotonin 2B receptor signaling is required for craniofacial and ocular morphogenesis in *Xenopus*
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