Most recent publications:

- **Brg1** promotes both tumor-suppressive and oncogenic activities at distinct stages of pancreatic cancer formation [2] (Roy et al., *Genes Dev* 2015)
- Numb regulates acinar cell dedifferentiation and survival during pancreatic damage and acinar-to-ductal metaplasia [11] (Greer et al., *Gastroenterology* 2013)
- Factors Expressed by Murine Embryonic Pancreatic Mesenchyme Enhance Generation of Insulin-Producing Cells From hESCs [14] (Guo et al., *Diabetes* 2013)
- Identification of Sox9-Dependent Acinar-to-Ductal Reprogramming as the Principal Mechanism for Initiation of Pancreatic Ductal Adenocarcinoma [15] (von Figura et al., *Cancer Cell* 2012)
- Bmi1 is required for regeneration of the exocrine pancreas in mice [16] (Fukuda et al., *Gastroenterology* 2012)
- Elevated Hedgehog/Gli signaling causes beta-cell dedifferentiation in mice [17] (Landsman et al., *PNAS* 2011)
- Stat3 and MMP7 contribute to pancreatic ductal adenocarcinoma initiation and progression
Primary cilia regulate Gli/Hedgehog activation in pancreas (Cervantes et al., PNAS 2010)

Hedgehog signaling in pancreas epithelium regulates embryonic organ formation and adult beta-cell function (Lau and Hebrok, Diabetes 2010)

Beta-catenin blocks Kras-dependent reprogramming of acini into pancreatic cancer precursor lesions in mice (Morris et al., J Clin Invest 2010)