Most recent publications:

- Designing β Cells [3] (Hebrok *Cell Metabolism* 2017)
- Brg1 promotes both tumor-suppressive and oncogenic activities at distinct stages of pancreatic cancer formation [9] (Roy et al., *Genes Dev* 2015)
- Plasticity and Dedifferentiation within the Pancreas: Development, Homeostasis, and Disease [10] (Puri et al., *Cell Stem Cell* 2015)
- Aberrant innate immune activation following tissue injury impairs pancreatic regeneration [12] (Folias et al., *PLoS One* 2014)
- Numb regulates acinar cell dedifferentiation and survival during pancreatic damage and acinar-to-ductal metaplasia [18] (Greer et al., *Gastroenterology* 2013)
- Generation of Functional Thymic Epithelium from Human Embryonic Stem Cells that Supports Host T Cell Development
Nr5a2 maintains acinar cell differentiation and constrains oncogenic Kras-mediated pancreatic neoplastic initiation \cite{20} (von Figura et al., *Gut* 2013)

Factors Expressed by Murine Embryonic Pancreatic Mesenchyme Enhance Generation of Insulin-Producing Cells From hESCs \cite{21} (Guo et al., *Diabetes* 2013)

Identification of Sox9-Dependent Acinar-to-Ductal Reprogramming as the Principal Mechanism for Initiation of Pancreatic Ductal Adenocarcinoma \cite{22} (von Figura et al., *Cancer Cell* 2012)

Bmi1 is required for regeneration of the exocrine pancreas in mice \cite{23} (Fukuda et al., *Gastroenterology* 2011)

Elevated Hedgehog/Gli signaling causes beta-cell dedifferentiation in mice \cite{24} (Landsman et al., *PNAS* 2011)

Pancreatic mesenchyme regulates epithelial organogenesis throughout development \cite{25} (Landsman et al., *PLoS Biol* 2011)

Stat3 and MMP7 contribute to pancreatic ductal adenocarcinoma initiation and progression \cite{26} (Fukuda et al., *Cancer Cell* 2011)

Primary cilia regulate Gli/Hedgehog activation in pancreas \cite{27} (Cervantes et al., *PNAS* 2010)

Hedgehog signaling in pancreas epithelium regulates embryonic organ formation and adult beta-cell function \cite{28} (Lau and Hebrok, *Diabetes* 2010)

Beta-catenin blocks Kras-dependent reprogramming of acini into pancreatic cancer precursor lesions in mice \cite{29} (Morris et al., *J Clin Invest* 2010)