Terminally differentiated cells in the pancreas have the plasticity to adopt alternative cell fates through dedifferentiation and reprogramming. This cellular plasticity, although considered a physiological process of tissue homeostasis, renders the tissue susceptible to diseases, such as cancer and diabetes. Cell-type specific gene regulatory programs maintain the identity and function of mature cell types, and although we have a clear picture of the steady-state transcriptional and epigenetic landscape in development and disease, the molecular regulators of cell fate transitions are not well understood.

In previous and ongoing work in our laboratory, we have shown that this process is dynamic and regulated at least in part by cell-type specific conserved long non-coding RNAs. Research in our lab focuses in understanding the molecular events, with emphasis in non-coding regulatory elements, leading to cell fate transition in development and cancer. These studies may provide molecular regulators of cellular and pathological associated plasticity leading to novel treatments in regenerative therapies and pancreatic cancer.