

Research

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The primary interest of my laboratory is to understand the mechanisms that regulate cell differentiation during embryogenesis as well as the basis for cell fate reprogramming in the adult. Our central focus is to study the formation and regeneration of pancreatic beta cells.

Type I diabetes is characterized by the immune destruction of pancreatic beta cells, and there is enormous interest in developing therapies to replace destroyed beta cells.

Therapeutic strategies involve restoring functional beta cell mass by exogenous replacement or transplantation of a source of beta cells such as embryonic stem cells derived beta cells or activation of endogenous regeneration of beta cells. We approach this by investigating the epigenetic mechanism of beta cells differentiation during organogenesis, the changes in the epigenetic landscape in differentiation of stem cells as well as understanding how other pancreatic cell types can be reprogrammed to beta cell fate. Strategies for tackling these complex problems include the generation and analysis of null mouse mutants, development of cell-type-specific inducible transgenic mice, as well as developing methods to drive embryonic stem cells to a beta cell fate.

Elucidation of the epigenetic network that regulates transcription during pancreatic developmental and regeneration will provide the blueprint by which the extra-cellular cues are interpreted at the nuclear level by the transcription machinery to select the repertoire of beta cell-specific genes. Such a blueprint will inform us on how to direct stem cells to beta cell fate in vitro and allow us to devise pharmacological interventions toward selective manipulation of gene expression to promote regeneration of beta cell mass. Furthermore, generation of animal models of diabetes and strategies to develop novel cellular therapies for diabetes is enabling us to test outcomes for candidate therapeutic approaches.

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