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Pancreas-specific deletion of Bmpr1a transiently impairs islet architectural development and establishes a model of T2D of fetal origin

Fang-Xu Jiang
University of Western Australia, Australia

A subgroup of the type 2 diabetes mellitus (T2D) has its origins during fetal development, but the underlying genes and mechanisms are not defined. Microarray and real time PCR analyses demonstrated that many transforming growth factor-β superfamily genes were progressively down-regulated in the developing pancreas and undetectable in adult islets. Pancreas-specific deletion of *bone morphogenetic protein receptor type 1a* (PcKO) reduced the expression of the key cell-cell interaction molecule E-cadherin in developing endocrine cells and transiently impaired islet architectural assembly. Although many parameters were comparable to wildtype counterpart, the postnatal PcKO mice displayed glucose intolerance under a metabolic stressor and their islets had an abnormal expression profile of approximately 700 genes. Among them, striking overexpression was the gene encoding the tryptophan hydroxylase, catalyzing production of the underdescribed insulin secretion regulator, 5-hydroxytryptamine. The aging PcKO mice exhibited a reduced expression of several key regulators for β-cell function, a significantly lower fasting plasma insulin concentration and higher pancreas insulin content, and spontaneous glucose intolerance. Hence the PcKO mice exemplify how modified gene expression in the fetal pancreas can translate into T2D in adulthood.

Biography

Fang-Xu Jiang is the Head of Islet Cell Development Program, the University of Western Australia and has extensive experience in medical research both in Australia and overseas. He published 35 peer-reviewed articles, 28 of which were first author primary and review articles in the leading/world-class journals and book chapters of my field (Diabetes, J Cell Sci etc). He the senior author in 20 articles. He also an Editorial board member for ISRN Developmental Biology.

fangxu.jiang@uwa.edu.au