Journal of Diabetes Research

Special Issue on
Compensatory Mechanisms of Pancreatic Beta Cells: Insights into the Therapeutic Perspectives for Diabetes

Call for Papers

Diabetes prevalence has risen dramatically during the last 20 years in parallel to the pandemic of obesity. The disease develops when insulin production is insufficient to compensate for the increased insulin requirements elicited by insulin resistance. Defective circulating insulin levels are the consequence of reduced beta cells number and decreased secretory capacity of the remaining beta cells.

Despite being exposed to environmental risk factors including excess of food and sedentary lifestyles, many obese and insulin resistant people do not suffer from diabetes. This situation is thought to result from the capacity of beta cells to resist against diabetogenic factors and to adapt their mass and function in order to produce enough insulin to compensate for the increased demand from target tissues. Such plastic ability is a physiological process that occurs in efficiently from birth to early childhood periods and during pregnancy. Protection and adaptation of beta cells has been shown to result from humoral mediators, which activate several intracellular adaptive pathways. So far, the interplay between these signaling cascades remains poorly understood. An integrative understanding of the events underlying beta cell adaptation will pave the way for the development of innovative strategies and new therapeutic approaches aiming to preserve beta cell activity in diabetes. We invite authors to submit original research and review papers investigating the contribution and the mode of action of a broad range of molecules including nutrients, hormones and plasma signals involved in beta cell adaptation. Potential topics include, but are not limited to:

- Signals from gestational and obesogenic environment fostering beta cell mass and function
- Role of microRNAs and long noncoding RNA in beta cells compensation and protection
- Entero-humoral signals eliciting beta cell function in metabolic surgery
- Identification of signaling and intracellular key targets that can be triggered for preserving beta cell function in diabetes

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First Round of Reviews	Friday, 9 May 2014
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