

Special Issue

Diabetes and Its Complications: Cellular and Molecular Mechanism and New Therapeutic Opportunities

Message from the Guest Editor

T2D pathogenesis is characterized by several factors including enhanced pancreatic alpha-cell deregulation, beta-cell dysfunction or death and insulin resistance. These alterations lead to chronic hyperglycemia are associated with blood vessel damage affecting both the microvasculature and macrovasculature. The knowledge of the cellular and molecular mechanisms of diabetes complications is still limited. Most of the known dysregulated pathways are strictly linked to the chronic hyperglycemic state and **reactive oxygen species** (ROS) imbalance. Hyperglycemia is also associated with atherosclerotic abnormalities leading to macrovascular complications. The modulation of these pathways could represent a treatment strategy for diabetes complications. This Special Issue aims to obtain further insights into the already known cellular and molecular mechanisms related to diabetes complications, with a particular focus on the identification of novel strategies for their modulation. Furthermore, this Issue aims to identify novel potential molecular pathways of vascular damage in diabetes that could provide novel therapeutic targets.

Guest Editor

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Deadline for manuscript submissions

closed (28 July 2023)



Cells

an Open Access Journal
by MDPI

Impact Factor 5.2
CiteScore 10.5
Indexed in PubMed



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Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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