Special Issue

Pathophysiological and Therapeutic Perspectives of Type-1 Diabetes

Message from the Guest Editor

Knowledge on the pathogenesis and natural history of type 1 diabetes has substantially grown in the last few decades. It is an auto-immune condition characterized by the destruction of the pancreatic beta cells, leading to absolute insulin deficiency. The destruction of □-cells is triggered by genetic, environmental and immunologic factors that destroy the endocrine cells of the pancreas, leading to insulin deficiency. Furthermore, inflammation (e.g., interleukin-1 mediated) may play a significant role in islet □-cells loss in type-1 diabetes. Patients with type 1 diabetes may also, coincidentally, have pathophysiologic elements of type 2 diabetes. We invite scientists to contribute both original research articles and reviews that highlight the development of pathophysiological pathways of type-1 diabetes and current therapeutic perspectives. Both basic and translational research papers are welcome.

Guest Editor

Dr. Srinivas Nammi

Discipline of Medical Sciences, School of Science and NICM-Health Research Institute, Western Sydney University, Sydney, NSW 2751, Australia

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Biomedicines
Editorial Office
MDPI, Grosspeteranlage 5
4052 Basel, Switzerland
Tel: +41 61 683 77 34
biomedicines@mdpi.com

mdpi.com/journal/biomedicines

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Biomedicines (ISSN 2227-9059) is an open access journal devoted to all aspects of research on human health and disease, the discovery and characterization of new therapeutic targets, therapeutic strategies, and research of naturally driven biomedicines, pharmaceuticals, and biopharmaceutical products. Topics include pathogenesis mechanisms of diseases, translational medical research, biomaterial in biomedical research, natural bioactive molecules, biologics, vaccines, gene therapies, cell-based therapies, targeted specific antibodies, recombinant therapeutic proteins, nanobiotechnology driven products, targeted therapy, bioimaging, biosensors, biomarkers, and biosimilars. The journal is open for publication of studies conducted at the basic science and preclinical research levels. We invite you to consider submitting your work to Biomedicines, be it original research, review articles, or developing Special Issues of current key topics.

Editor-in-Chief

Prof. Dr. Felipe Fregni
1. Neuromodulation Center and Center for Clinical Research Learning, Spaulding Rehabilitation Hospital and Massachusetts General Hospital, Harvard Medical School, Boston, MA 02114, USA
2. Department of Epidemiology, Harvard T.H. Chan School of Public Health, Boston, MA 02115, USA

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